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Parental body shape in mid-life and its association with adult offspring obesity, body shape and self-perception of weight status

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ABSTRACT

Obesity and its health-related risks of increased morbidity and premature mortality are global concerns, with previous studies examining associations between obesity and mortality mainly focussing on Body Mass Index (BMI), without central adiposity. Intervening in the intergenerational transfer of obesity is recognised as a significant opportunity to impact the obesity epidemic. For individuals, recognition of parental obesity and determining accurate self-perception of body weight may result in healthier behaviours and ageing. There are few studies on the Australian population regarding the association between parental body shape and adult offspring body shape, and no literature was located using an Australian or international population regarding parental body shape and offspring weight self-perception.

The aim of this research was to examine the effect of excess weight and waist circumference on mortality, and then explore the influence of mid-life parental body shape, using recall from pictograms by their adult offspring, with measured and self-perceived weight of adult offspring.

All three studies in this thesis used data collected on adults from the North West Adelaide Health Study (NWAHS), a South Australian longitudinal cohort study established in 1999 (baseline n=4060).

The first study presents findings on the association between obesity and all-cause, cardiovascular disease and cancer-related mortality, using a recently developed measure of mortality risk (A Body Shape Index (ABSI)) incorporating both waist circumference (WC) and BMI. Results suggest that people with the highest BMI and

WC combined, as calculated by the ABSI, had the highest mortality risk; more than two and a half times those with the lowest ABSI.

The second study presents the association between midlife parental body shape (using pictograms for offspring recall) and four weight measures of obesity and fat distribution (BMI; WC; waist-hip ratio, WHR; and waist-height ratio, WHtR) of their adult children. Having two obese parents resulted in an increased likelihood of their adult offspring also being overweight or obese. This association tended to be stronger for daughters than sons across BMI, WC and WHtR.

The third study examined self-perception of weight and demonstrated that only 27% of obese men and 39% of obese women perceived themselves to be "very overweight". This study also examined the association between midlife parental body shape and self-perception of weight among adult offspring, finding that obese men and overweight or obese women who had a heavier mother were more likely to correctly estimate or underestimate their own weight. Obese women who also had an obese father were more likely to correctly estimate or underestimate their own weight than women whose father was not obese. This association did not hold for obese men. Among normal weight men, those who had a heavier mother were more likely to overestimate their weight.

Through the use of pictograms of parental body shape as screening devices, in combination with a person's current body shape measures and weight self-perception, primary care physicians may be able to identify those with an increased

risk of developing obesity related co-morbidities and premature mortality, for

targeted monitoring, intervention and treatment.

DECLARATION

I certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name, in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name, for any other degree or diploma in any university or other tertiary institution without the prior approval of the University of Adelaide and where applicable, any partner institution responsible for the joint-award of this degree.

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            (Janet Grant)

ACKNOWLEDGEMENTS

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ACRONYMS/ABBREVIATIONS

ABSI	A Body Shape Index
AIHW	Australian Institute of Health and Welfare
BAI	Body Adiposity Index
BMI	Body Mass Index
BRFSS	Behavioral Risk Factor Surveillance System
CATI	Computer assisted telephone interview
CI	Confidence interval
CrI	Credible interval
CVD	Cardiovascular disease
DXA	Dual-energy X-ray Absorptiometry
EWP	Electronic White Pages
FEV1	Forced expiratory volume in one second
FTO	Fat mass and obesity-associated (gene)
HALS	Health and Lifestyle Study (UK)
HPA	Hypothalamic-Pituitary-Adrenal (axis)
HR	Hazard ratio
ICD	International Classification of Diseases
MHO	Metabolically healthy obese
mmHg	millimetre of mercury
mmol/L	millimoles per litre
MR	Mortality risk
NAAFA	National Association to Advance Fat Acceptance (US)
NCD	Non-communicable diseases
NCIS	National Coronial Information Service (Australia)
NDI	National Death Index (Australia)
NHANES	National Health and Nutrition Examination Survey (US)
NHMRC	National Health and Medical Research Council
NMD	National Mortality Database (Australia)
NWAHS	North West Adelaide Health Study
OR	Odds ratio

PPV	Positive predictive value
RR	Relative risk
SD	Standard deviation
SES	Socio-economic status
SNPs	Single nucleotide polymorphisms
TFU	Telephone follow-up
UK	United Kingdom (England, Scotland, Wales & Northern Ireland)
US	United States (of America)
WC	Waist circumference
WHO	World Health Organization
WHR	Waist-hip ratio
WHtR	Waist-height ratio
WTR	Waist-to-thigh ratio

PUBLICATIONS CONTRIBUTING TO THIS THESIS

> Grant JF, Chittleborough CR, Shi, Z, Taylor AW. **The association between A Body Shape Index and mortality: results from an Australian cohort.** *PLoS ONE.* 2017;12(7): e0181244; https://doi.org/10.1371/journal.pone.0181244 *(see Chapter 4)*

> Grant JF, Chittleborough CR, Taylor AW. **Parental midlife body shape and association with multiple adult offspring obesity measures: North West Adelaide Health Study.** *PLoS ONE.* 2015;10(9):e0137534; https://doi.org/10.1371/journal.pone.0181244 *(see Chapter 5)*

> Grant JF, Chittleborough CR, Taylor AW. **Parental midlife body shape influences offspring self-perception of weight in a cohort of Australian adults.** *Journal of Obesity and Overweight.* 2016;2(3), 303-1-303-11; http://dx.doi.org/10.15744/2455-7633.2.303 *(see Chapter 6)*

CONFERENCE PRESENTATIONS ARISING FROM THIS THESIS

Parental body shape at midlife and its association with adult offspring weight measures

- Janet Grant, Catherine Chittleborough, Anne Taylor - The University of Adelaide

(poster presentation) | Australian & New Zealand Obesity Society Annual Scientific

Meeting, Brisbane - Australia, 19-21 Oct 2016.

1.1 Introduction

Obesity continues to be a major public health issue, imposing a significant health, care and economic burden on the individual, their families and the community in general, and involving a considerable contribution by health professionals, including those involved in clinical care and preventive health. Obesity has been recognised as the world's most recent major epidemic, with acknowledgement in 1997 by the World Health Organization (WHO) that it had rarely appeared as a health issue before the 20th century and that global rates of obesity have doubled since 1980 [1]. Obesity has traditionally been considered an epidemiological risk factor for such health conditions as cardiovascular disease (CVD), diabetes, some cancers and mental ill-health [2], but was also deemed to be a disease in its own right, earning a classification in 1948 from the International Statistical Classification of Diseases (ICD) [3].

There has been growing recognition of the importance of fat distribution as well as overall body weight as calculated by BMI [4] on overall and cause-specific mortality [5,6]. Recent development of new measures such as A Body Shape Index (ABSI) that incorporates height, weight and waist circumference (WC) have been shown to accurately predict mortality risk, while providing a means to lower one's risk through subsequent reduction in weight and/or waist measurements [7].

A wealth of information has been published regarding probable contributors to the obesity epidemic. One such contributor is the influence of the body shape of biological and adoptive parents on the subsequent body shape of their adult children which has been examined since the 1980s [8-11] to the present [12,13], providing

strong evidence that both familial genetic and environmental factors can impact on offspring weight and fat distribution. A number of reliable and validated calculation tools have been developed to measure weight and central adiposity at both an individual and population level including body mass index (BMI), WC, waist-hip ratio (WHR) and waist-height ratio (WHtR).

A potential barrier to achieving weight loss for obese individuals is the failure to perceive that they have a problem in the first place that is affecting their health, particularly if they have grown up in an obesogenic environment with parents and/or other family members and a social network of friends who are also obese, where they perceive that they are of 'normal' size [14]. Misperception of body weight among people who are obese can lead to continued unhealthy lifestyle choices and the development of multiple morbidities, possibly leading to premature mortality. Accurate self-perception of being overweight/obese is an important factor in the process of weight loss and control, and associated health risks [15]. Perception of parental body shape could also be important in shaping offspring's perception as to what is 'normal'.

Confronting misperception of obesity involves measurement of weight including central adiposity (waist and hip circumference) by health professionals so that a discussion of actual weight and fat distribution can be compared with healthy weight ranges. The misperception of most concern is that the person considers themselves to be normal or only a little overweight when they are actually obese. Discussions with the health professional can then include the risks associated with obesity for chronic disease, leading to premature mortality.

1.2 Overview of thesis

This thesis provides background literature regarding obesity (Chapter 2): as a public health issue; its prevalence, incidence and trends; as an economic burden; its association with mortality; and its aetiology using a framework of contributing factors. This chapter also includes information about parental body shape as a determinant of adult offspring obesity and self-perception of weight. Chapter 3 provides information about the methodology (based on the North West Adelaide Health Study (NWAHS) as the data source), including the study design (sampling, recruitment, ethical approvals and response rates), and how obesity and parental body shape were measured.

The aim of this research is to firstly determine the existence and strength of the association between overall/central obesity and premature mortality in an Australian population (see Chapter 4). It then aims to examine the strength of the association between parental body shape and offspring body shape, firstly through various measures of obesity (BMI, WC, WHR and WHtR) (Chapter 5), as well as the association between parental body shape and self-perception of weight (Chapter 6). Chapter 7 provides a summary of the research findings, a discussion regarding implications of the research findings, future research directions and a conclusion.

2.1 Literature search

While a systematic review was not conducted for the purposes of this thesis, a number of search terms relating to measurement of parental body shape and obesity were entered into a range of literature databases. From an initial pool of over 2000 publications, a review was undertaken to create a bibliography of 576 references, from which a final reference list of papers (including methodology information, measurement, aetiology etc of obesity, ABSI, parental body shape and misperception) totalled n=310).

2.2 Definition of obesity

Obesity (and overweight) have been defined by the WHO as *"... abnormal or excessive fat accumulation that may impair health." [16].* Obesity has traditionally been considered an epidemiological risk factor, but was also deemed to be a disease in its own right, earning a classification from the ICD in 1948 [3].

2.3 Prevalence and trends of obesity

The WHO have estimated that in 2014, more than 1.9 billion adults aged 18 years and over were overweight (39%), with 600 million (13%) of these being obese [16]. Obesity rarely appeared as a health issue before the 20th century. It has been identified as the most recent major worldwide epidemic/pandemic, with the WHO reporting global rates of obesity doubling between 1980 and 2008 [1].   In this time period, the age-standardised mean global BMI increased between 0.4 to 0.5 BMI units

each decade. The Oceania[1] subregion saw the largest increase in this time, at 1.4 kg/m^2 for men and 1.9 kg/m^2 for women.   In contrast, there was minimal increase, or even a potential decrease, in obesity trends observed for women in central and eastern Europe, and in central Africa and south Asia for men [17].

In a pooled analysis of data from 19.2 million participants (9.9 million men and 9.3 million women) in 1698 population-based measurement studies across 186 countries, the Non-Communicable Diseases (NCD) Risk Factor Collaboration [18] reported for men, that the global age-standardised mean BMI rose from 21.7 kg/m^2 (95% credible interval 21.3-22.1) in 1975 to 24.2 kg/m^2 (95% CrI 24.0-24.4) in 2014. Overall, regional mean BMIs ranged from 21.4 kg/m^2 in central Africa to 29.2 kg/m^2 in Polynesia and Micronesia [18]. From 1975 to 2014, obesity increased for men from 3.2% (95% CrI 2.4-4.1) to 10.8% (95% CrI 9.7-12.0); with 2.3% (95% CrI 2.0-2.7) of men being severely obese (BMI ≥35) and 0.64% (95% CrI 0.46-0.86) being morbidly obese (BMI ≥40).

Similar results were reported for women: from 1975 to 2014, the global age-standardised mean BMI rose from 22.1 kg/m² (21.7–22.5) to 24.4 kg/m² (24.2–24.6); and the regional mean BMIs ranged from 21.8 kg/m² (21.4–22.3) in south Asia to 32.2 kg/m² (31.5–32.8) in Polynesia and Micronesia [18]. Women have a higher prevalence of obesity than men: from 1975 to 2014, this increased from 6.4%

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[1] *Oceania comprises the Cook Islands, Fiji, French Polynesia, Kiribati, Marshall Islands, Micronesia (Federated States of), Nauru, Palau, Papua New Guinea, Samoa, Solomon Islands, Tonga and Vanuatu.*

(5.1–7.8) to 14.9% (13.6–16.1), with 5.0% (4.4-5.6) being severely obese and 1.6% (1.3-1.9) being morbidly obese [18].

Obesity is a worldwide problem. In 2014, the top five countries with the greatest obesity burden are provided below (in millions M/% of global obesity), in descending order of the number of obese men using BMI ≥30:

o   1st - China (n=43.2M/16.3%);

o   2nd - United States of America (US) (n=41.7M/15.7%);

o   3rd – Brazil (n=11.9M/4.5%);

o   4th - Russia (n=10.7M/4.0%); and

o   5th – India (n=9.8M/3.7%) [18].
For obese women using BMI ≥30, in descending order, the top five countries with the greatest obesity burden are:

o   1st - China (n=46.4M/12.4%);

o   2nd - United States of America (US) (n=46.1M/12.3%);

o   3rd –India (n=20.0M/5.3%);

o   4th - Russia (n=46.1M/12.3%); and

o   5th – Brazil (n=18.0M/4.8%) [18].

These five countries contain 44% of the male obesity cases in the world, and 47% of the female obesity cases. Rounding out the top ten countries for obesity in the world are (in order): for men, Mexico, Germany, UK, Italy and France; for women, Mexico, Egypt, Turkey, Germany and Iran [18].

The US has the highest prevalence of severe obesity (BMI ≥*35)* for both men

(n=16.2M/27.8%) and women (n=23.1M/18.3%), followed by China (men

4.3M/7.4%; women 7.6M/6.1%). For men, the next three countries with the greatest

burden of severe obesity were Mexico (n=2.4M/4.1%), Russia and Brazil (both

n=2.2M/3.8%). For women, the next three countries were Russia (n=7.3M/5.8%),

Brazil (n=6.7M/5.3%) and then Mexico (n=5.5M/4.4%) [18]. An investigation of

severe obesity in the NWAHS population found Classes II and III measured obesity

(BMI≥35) had increased from 2.4% in 1991 to 8.1% in 2006, with women more than

two and half times more likely than men to be severely obese [19].

While Australia was not listed within the top ten countries of the world with the

greatest obesity burden, due to its smaller population, the Australian Institute of

Health and Welfare (AIHW) nevertheless reported increases in obesity (based on

measured BMI) from 1995 to 2007-08 in adults aged 18 years and over: for men,

from 19% to 26%; for women, from 19% to 25% [20]. In Australia in 2011-12, 63%

of adults were overweight or obese. It is estimated that this will increase to 72% by

2025 (approximately 7.2 million obese Australians, equating to a 1.5% increase each

year) and will include 33% of people who are obese [21,22].

2.4 Economic burden of obesity

The global obesity problem is increasing associated health-care costs and reducing

productivity. In 2011, the trends by 2030 for obesity were simulated for the US and

the UK, resulting in a projection of an additional 65 million and 11 million obese

adults for each respective country [23]. Combining the two countries, these largely

preventable figures translate to an extra 5.7-7.3 million cases of heart disease and

stroke, 6-8.5 million cases of diabetes, 492,000-669,000 cases of cancer and 26-55 million quality-adjusted life years forgone [23]. The costs for providing medical treatment range from USD$48-66 billion and GBP£1.9-2 billion [23].

These trends are similar to earlier projections in 2005 regarding the obesity burden of disease in Australia of AUD$14.5 billion [24]. This was increased in a more recent 2008 estimation by Obesity Australia of AUD$6.3 billion (for obesity alone), together with another AUD$49.9 billion for lost wellbeing, resulting in a total cost of AUD$58.2 billion [25].

In an examination of the financial impact of meeting WHO targets by 2025 by either a reduction in the number or percent of people by obesity class, Obesity Australia – in conjunction with Pricewaterhouse Coopers – provided the following predictions of benefits in 2015 present value terms (Table 2.1) [22]. For example, looking at Obesity Class I, if there is no evidence of change in the growth of obesity from 2011-12, the authors predict that there will be 4,186,450 people in Obesity Class I by 2025, representing a 33% increase from 2011-12 and resulting in an additional cost of $42.1 billion [22]. If Australia were able to meet the WHO target of halting the rise in obesity, and return to and maintain the 2010 obesity prevalence of 26%, the authors predict that the number of obese people could be reduced by 605,800 people, representing a 14.5% decrease and saving $2,550 million [22].

Table 2.1 Numbers and percent of WHO BMI obesity class I, II and III by 2025, with associated additional costs and predicted benefits (AUD$ million)

For the year 2025	Obesity Class I (BMI 30.00-34.99)	Obesity Class 2 (BMI 35.00-39.99)	Obesity Class III (BMI ≥40.00)	TOTAL
Number of obese				
Predicted *(from 2011-12) (n)*	4 186 450	1 722 900	1 337 100	**7 246 450**
WHO target *(from 2015-16) (n)*	-605 800	-388 850	-567 350	**-1 562 000**
Percent of obese				
Increase predicted *(from 2011-12) (%)*	33%	52%	147%	**50%**
WHO target *(from 2015-16) (%)*	-14.5%	-22.6%	-42.4%	**-21.6%**
Costs *(from 2015-16 to 2024-25 FY)*				
Additional predicted *($ billion)*	$42.1	$21.5	$24.1	**$87.7**
Benefits from reductions *($ million)*	**$2 550**	**$2 090**	**$5 690**	**$10 330**

Source: Obesity Australia, van Smeerdijk, Jovic, Babbage, Hockings, Schlesinger et al, 2015 [22]

2.5 Obesity and mortality

The global epidemic of obesity is strongly linked to an increased risk for major chronic diseases [26] and is also associated with impaired quality of life and mental health [27]. Obesity is associated with an increased risk of myocardial infarction, heart failure and premature atherosclerosis [28,29], as well as multiple types of cancer including prostate, breast, endometrial, colon, oesophageal, kidney and thyroid [30,31]. It is also associated with asthma [32], type 2 diabetes [33], liver disease, and gastro-oesophageal reflux disease and its complications [26].

The WHO report that 65% of the global population live in countries where overweight and obesity kills more people than underweight [16]. For about one-fifth of the world's population who live in industrialised countries, overweight is one of five leading risk factors for mortality, along with high alcohol consumption, high blood pressure, high cholesterol and tobacco use [34].

Many studies of the association between overweight/obesity and mortality have used self-reported or measured BMI [35-38]. For example, a large study of 900,000 adults predominantly from western Europe and the US reported that of the 66,552 deaths of known cause over an average of eight years with a mean age of 67 years at death, 30,416 were vascular-related; 2070 diabetic, renal or hepatic; 22,592 neoplastic; 3770 respiratory and 7704 other causes. Mortality was found to be lowest at a BMI of 22.5-25; above this range, the progressive excess mortality was generally due to vascular causes. The authors reported that for every five unit increase in BMI, there was an association of approximately 30% higher overall morality. Median survival was reported to be reduced between two and four years for those with low obesity (BMI 30.0-34.9), and between eight to ten years for those in the severely obese range (BMI 40-45) [4]. Likewise, a study in the US population found that those in Classes II/III (BMI ≥35) had an increased mortality risk of 62% for men and 40% for women compared with normal weight people (BMI 18.5-24.9), but that overweight (BMI 25.0-29.9) and low obesity did not increase mortality risk [39]. Similar results were reported for a German population, in a study that determined that overweight people did not have a higher mortality risk (MR) than normal weight people but that their mortality from individual diseases either increased, decreased or was unchanged, according to the disease. The authors also concurred that obesity (BMI ≥30) conferred a higher risk than overweight [40].

Regarding the influence of age, Kuk et al [41] examined the risk of all-cause mortality for a US population of 4437 men and 5166 women before or after the age of 65 years using 1988-1994 NHANES data. The authors examined BMI, WC, WHR, hip circumference (HC), sum of skinfolds and bioelectrical impedance measures [41].

They found that both overall and central adiposity was associated with higher premature mortality, but for older people the association was either null or inverse [41]. In contrast, a review article examined the impact of obesity on mortality among elderly people and found that obese older adults have an increased risk of premature mortality, whilst highlighting the limitations of BMI and the usefulness of WC as an indication of central adiposity and as a risk factor for mortality [42]. The previous finding of a null or inverse association of obesity with mortality for older people was examined by Masters et al [43], who argued that the results were biased due to confounding regarding cohort participant age at survey and/or cohort membership. In their investigation, they found that the relationship of obesity and premature mortality grew stronger with age [43].

A growing number of studies have highlighted the usefulness of incorporating central adiposity measures (eg WC, WHR, WHtR etc) when investigating risk factors for premature mortality [5,44-50]. An examination of 5799 men and 6429 women using the third National Health and Nutrition Examination Survey (NHANES) study (1988-1994) found that waist-to-thigh ratio (WTR) in both sexes and WHR in women showed a positive linear association with mortality in adults aged 30 to 64 years; BMI and WC showed U- or J-shaped associations. Those with a normal BMI but a higher WTR and/or WHR had increased premature mortality risk, as well as those who were obese according to their BMI [51].

2.6 Aetiology of obesity

2.6.1 Aetiological framework

The aetiological factors discussed in this section are illustrated in the framework below (Figure 2.1), which was adapted from a diagram showing the modified stream of causation metaphor applied to the study of obesity [52].

Glass & McAtee's original version included cultural norms, local food and built environments, area deprivation, psychosocial hazards and commercial messaging as risk regulators. The framework has been updated for this thesis to also include parental body shape and self-perception as risk regulators which are major subjects in this thesis. The focus of this section, is to describe the relevance of the adapted risk regulators in detail, whilst providing an overview of the remaining regulators. This section will also provide background about genetic and biologic influences, birth weight and early exposures (in pregnancy and infancy; and in childhood and adolescence), and health behaviours.

Within the framework, the hierarchical axis provides an indication of the weight of importance from genetic influence (low) to risk regulators (high). The time axis begins with birth weight and early exposures, moving through health behaviours consisting of 'energy input' and 'energy expenditure' to body weight change. Excessive weight gain can lead to chronic disease which may lead to premature mortality, the latter outcome is also examined in this thesis.
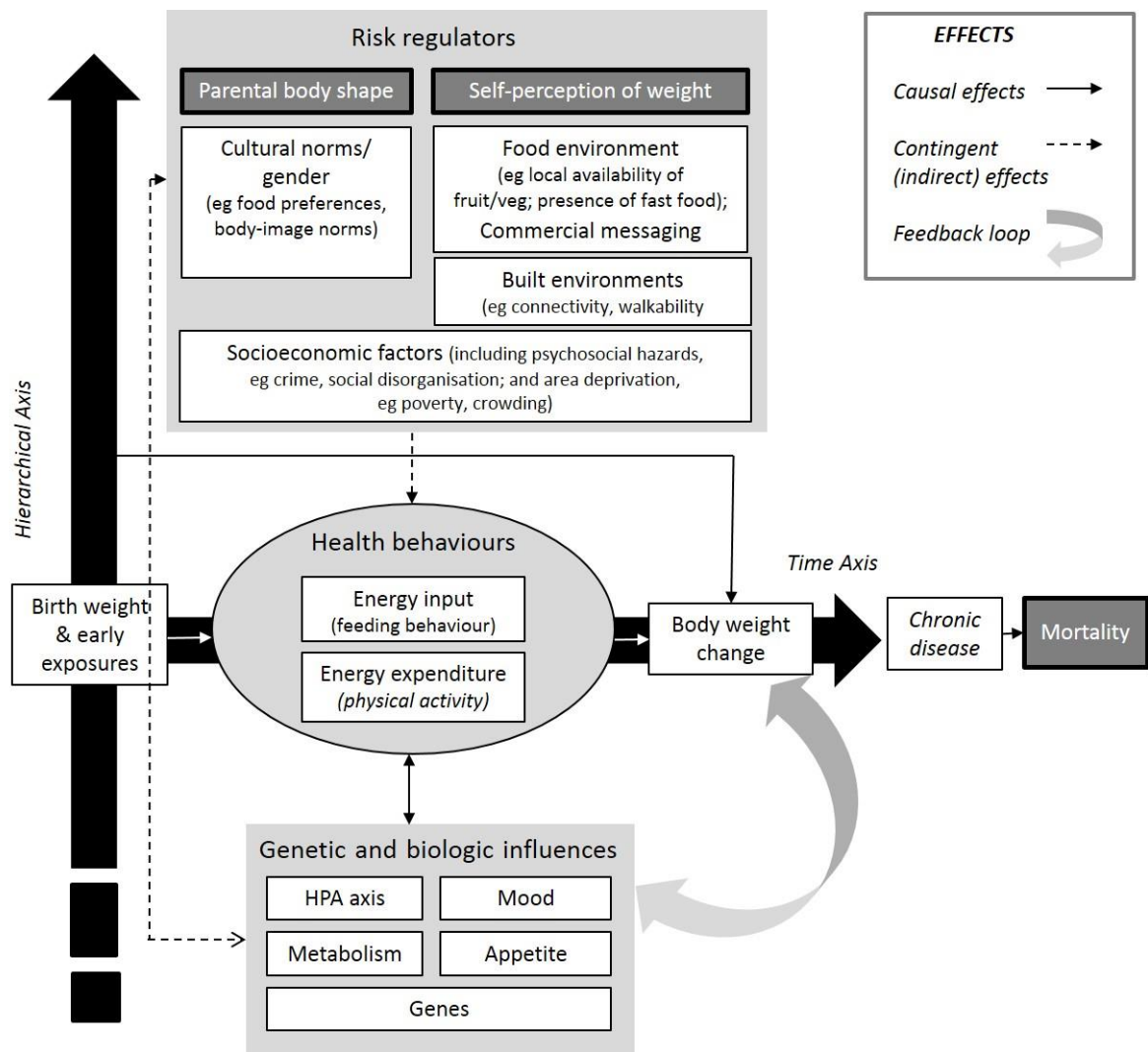
Figure 2.1 Framework detailing the association between parental body shape, self-perception of weight and (premature) mortality for adult offspring, with health behaviours, risk regulators and genetic and biologic influences

Adapted from: Glass & McAtee, 2006 [52]

Overlaying both genetic and environmental determinants is the significant contribution of a myriad of factors during a person's life-course. Overall, it is argued that genes contribute in a small way to obesity risk but that food intake and activity levels more determine the number of people who are obese [53-55]. However, whether the association weakens over the lifespan as influences from the home background diminish and others come to the fore, has been little researched [56].

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This research used this framework to show the development of obesity, as a basis for exploring the association of parental body shape, weight self-perception and obesity-related premature mortality with that of their adult offspring.

2.6.2 Genetic and biologic influences

Researchers have long been intrigued with the influence of genes on the human condition, although there is ongoing debate regarding how much they each contribute to weight status and the clustering of obesity within a family. Summarising the genetic and biological influences as shown in the framework above, Glass and McEntee hypothesise that risk regulators modify biological factors such as mood, appetite and metabolism, as well as the HPA (hypothalamic-pituitary-adrenal) axis [52], Vicennati et al [57] reported a close relationship between the HPA and adipose tissue. They consider that obesity, and in particular the visceral phenotype, may result in a deregulation of HPA axis activity as a maladaptation to chronic stress, similar to the metabolic syndrome and its alterations to metabolic and cardiovascular function [57].

Regarding parental genetic influence, Sorenson et al [58] observed that adoptees' weight was strongly associated with their biological parents' weights, but not their adoptive parents' weights. Schousboe et al [59] surmised that heritability estimates are typically strong (45% to 85%) in family studies. Stunkard et al [60] found that the intra-pair correlation of BMI between twins reared apart was similar to that of twins reared together, that is sharing the same environment as children did not contribute to a similar BMI in later life; therefore the influence of genetics was substantial.

In recent genome-wide association studies, the variation in BMI that can be explained by more than 30 common BMI loci identified is low (1.5%), highlighting the degree of influence from other genetic and environmental factors [61]. While single gene mutations are responsible for rare forms of monogenic obesity [62], there is growing evidence that common genetic variants or single nucleotide polymorphisms (SNPs) can have a large effect due to their high frequency at the population level, whilst having only a modest effect on susceptibility to common forms of obesity at an individual level [63].

In the first genome-wide association study of 13 cohorts (38,759 participants), Frayling et al [64] found a genetic variation which was associated with an increased risk for type 2 diabetes (the fat mass and obesity-associated (FTO) gene) mediated through an effect on BMI. They found that 16% of adults who were homozygous[2] for the risk allele[3] of FTO weighed approximately three kilograms more and had a 1.67-fold increased odds of obesity, compared with those who did not have the risk allele. This resulted from longitudinal changes in fat mass (both increased WC and subcutaneous fat) from the age of seven years. In their 2008 study of the FTO obesity risk genotype in more than 3000 children, Wardle et al [65] found that the most common known risk allele for obesity was more likely to influence appetite through impaired satiety responsiveness, in turn affecting adiposity.

_____

[2] *homozygous: having identical pairs of genes for any given pair of hereditary characteristics*
[3] *allele: any of several forms of a gene, usually arising through mutation, that are responsible for hereditary variation*

Other obesity-related risk factors from genetic influences have also been researched. In one genetic study, paternal overweight was associated with an increased risk of breast cancer in daughters, highlighting the need for fathers to also be aware of excess body weight when considering parenthood [66].

Approximately fifty years ago, Neel [67] described a hypothesis regarding the existence of a "thrifty gene" that historically influenced the natural selection of survivors in certain populations during "feast and famine" cycles but which in today's abundance of food to populations in developed countries, has increased people's susceptibility to obesity and related diseases such as type 2 diabetes. Wells [68] argues that the transmission of obesity over generations is due to exposure to maternal, familial and environmental "niches" during development, incorporating response to ecological changes that affect height and lean mass.

2.6.3   Birth weight and early exposures

Barker and Hales [69] challenged the notion of the 'thrifty gene' and highlighted the importance of developmental influences during fetal life and infancy that may cause an individual to be more susceptible to an obesogenic environment during later life. There are also factors that adversely influence young children and adolescents and contribute to the development of obesity during these stages, as well as adulthood. These will be discussed here in more detail.

2.6.3.1   Obesity in pregnancy and infancy

It is well recognised that maternal obesity is a major factor in the health and wellbeing of both mother and baby [70]. In a review of early markers of adult

obesity, Brisbois et al [71] identified potential early markers including two factors

relating to maternal obesity: maternal weight gain during pregnancy (possible

marker) and maternal BMI (probable marker). For expectant mothers, obesity

increases the risk of caesarean section, medical complications such as hypertension

and pre-eclampsia, and depression at the time of birth [72-74] and in the long term,

of increased obesity [75]. In Australia, the prevalence of obesity among women of a

reproductive age is 28%, with 20% of those giving birth being obese [76]. In the US,

morbidly obese women (BMI≥40, Class III) comprise 8% of women aged in their

reproductive years.

For the baby, maternal obesity increases the risk of miscarriage, pre-term birth, still-

birth and fetal anomalies, hypoglycaemia, jaundice, respiratory distress and neonatal

intensive care [73,74,77] as well as obesity, increased insulin resistance,

hypertension, dyslipidaemia, behavioural problems and risk of asthma in the long-

term [78,79]. Pre-pregnancy obesity has also been linked to gestational diabetes

mellitus, increasing the risk for maternal type 2 diabetes, as well as offspring obesity

[80]. In a review of outcomes for women in BMI Classes I to III obesity, the authors

found that compared to Class I and/or Class II, morbidly obese women were 31%

more likely to give birth before 37 weeks than women of normal weight; and to have

large-for-gestational age babies [81]. A review of cohort studies found that maternal

obesity increased the risk of high birthweight of >4000g two-fold (OR 2.00, 95% CI

1.84-2.18) and of the baby being large for gestational age overall (OR 2.08, 95% CI

1.95-2.23) when compared to mothers who were normal weight [82].

An Australian study reported increases from 1998 to 2009 in maternal BMI Class III from 1.2% to 2%, and from 2.5% to 3.2% for maternal BMI Class II (35 to <40) [73]. Of concern is the small but growing rates of super-obesity (BMI≥50 or the weight equivalent of ~140 kg) among prospective mothers, ranging from an estimated 8.7 women per 10,000 (0.1%) in the UK to 2.2% in the US, with 0.21% in Australia [76]. The effects of a child's early life environmental exposures on BMI was shown in a study of twin brothers versus non-twin siblings, highlighting possible modifiable influences [83].

Umberson et al [84] also examined how parenthood affects a person's weight gain (the opposite of how parents affect their offspring's weight), using data from a 15 year period. The transition to parenthood has been shown to increase weight for a year after birth (about 1.7 kg) for about 50% of women [84]. The timing of parenthood is also a potential risk, with the very young, as well as those approaching middle age at first birth, showing increased risk for weight gain [84]. Becoming a parent results in weight gain for different reasons: for men, the risk increases if they are a teenage father; for women, the number of pregnancies carried to viable gestational age [84].

Although the vast majority of research in this area has been focused on the effect of maternal obesity, recent research has also been undertaken regarding the possible effect of paternal obesity on offspring body size. In a study of the influence of the body shape of both parents, the authors reported that there were distinct differences in the BMI growth curves of infants born to obese fathers, although these were not as pronounced as those relating to obese mothers [85].

2.6.3.2 Obesity in childhood and adolescence

Evidence shows that infants who are overweight or obese are more likely to maintain their increased weight into childhood and adolescence [86,87]. One study found that those in the highest quartiles at both birth and at six months of age have a 40% risk of obesity at three years of age, compared to a 1% risk for those infants in the lowest quartiles [88]. Similar results were found in a review that examined the impact of overweight in infancy and weight gain in babies and young children up to the age of two years, resulting in high body size at thirteen years of age [89]. Li et al [90] followed up approximately 1620 adults (aged 20-51 years) who had been examined for BMI change at least four times from the age of four to nineteen years. They found that, independent of the mean BMI during childhood, ORs for adult obesity increased progressively from the lowest to the highest BMI quartiles during childhood, where there was either a rapid increase in weight or greater fluctuations in BMI during childhood [90].

The problem of childhood obesity is getting worse in many countries. In their review paper, Karnik et al [91] describe childhood obesity as a global public health crisis that has increased over recent years in both developing and developed countries and acknowledge that like other stages across the lifecourse, obesity during childhood is due to a wide range of genetic, behavioural and environmental causes. They echo the WHO which see it as one of the most serious challenges for this century, and estimate that the number of infants and young children aged 0-5 years who are obese has increased globally from 32 million in 1990 to 42 million in 2013 [92]. The WHO highlight that the increase has been predominantly in developing countries; 30% higher than developed countries as shown in the WHO African region alone, where in

2013, 9 million children were estimated to be obese compared to 4 million in 1990 [92].

In Australia in 2011-12, the overall prevalence of measured overweight and obesity for children aged 5-14 years was estimated at 26% (19% overweight, 7% obese) with no differences observed between boys and girls or between the age groups (5-9/10-14 years) [93]. Sanders et al [94] state that Australia is predicted to outrank the US and the UK by 2022 with its prevalence of overweight and obesity. In their review article, the authors predict that every third Australian child aged between 5 and 19 years will be overweight or obese by this time, with an expected associated burden of non-fatty liver disease and cardio-metabolic risk factors, as well as mental health conditions and reduced quality of life [94].

There are a number of health implications for children who are overweight or obese which may track into adulthood, in addition to the probability that they will most likely contribute to the risk of their own children being overweight or obese as infants, young children, adolescents and/or adults. Physical health effects in childhood include reduced tolerance for exercise and/or breathlessness on exertion, sleep apnoea, and a number of orthopaedic and gastrointestinal problems, including non-alcoholic fatty liver disease [95]. Mental health effects include impaired psychological wellbeing from experiencing bullying, teasing and discrimination at school and in social groups, and the resulting impact on relationships with peers and educational progress [95]. In the long term, being overweight or obese in childhood has been shown to increase the risk for obesity-related diseases such as cardiovascular disease, diabetes and some cancers, as well as musculoskeletal

conditions, such as those described in Chapter 5, and for premature mortality as described in Chapter 4. Therefore, it is not only that obese parents produce obese children who are likely to go on to become obese adults, there is an added burden that obesity in childhood increases the risk of developing chronic conditions later on.

The economic burden of childhood obesity was explored in a review by Finkelstein et al, who estimated the incremental lifetime medical cost of a ten year old obese child to be approximately $USD19,000 more than a normal weight child of the same age [96]. They also provide an alternative estimate of an additional $USD12,600 for young people of normal weight who then have eventual weight gain [96].

2.6.4   Health behaviours (energy input and expenditure)

Complex interactions between human behaviour, genetic disposition and the environment can contribute towards obesity, which the WHO have stated is fundamentally an imbalance between excessive energy intake and decreased energy expenditure [16]. Hill et al [97] argue that each of these factors require action, and suggest that having a high level of energy expenditure with a matching level of energy intake would be more feasible for most people, rather than restricting food intake to match a low level of physical activity. Further, they point to the easier goal of preventing excessive weight gain rather than reducing obesity [97]. Romieu et al [98] contend that the difference between energy intake and expenditure is the main driver of weight gain for populations in high income countries, and that diet quality impacts energy balance through complex neurological and hormonal pathways that affect satiety and other mechanisms.

2.6.5 Risk regulators

Risk regulators have been defined by Glass and McPhee [52] as "a class for variables that capture aspects of social structure that influence individual action". There is an abundance of literature discussing the impact of various factors on obesity, both in children and adults. A search in all fields in PubMed of 'environment and obesity' found over 6000 articles; using 'socioeconomic and obesity' found over 5500 articles. Epidemiological and medical publications have promulgated the effects of, as well as interactions between, socioeconomic and physical factors such as income, race, education, gender, work status, marital status, neighbourhood crime and violence and their link to feelings of safety and resulting levels of physical activity, housing, alcohol intake, immigration and access to good food. The aetological framework for obesity above (Figure 2.1) highlights a number of risk regulators. The focus of this thesis includes two of these risk regulators - parental body shape and self-perception of weight. Others include cultural/gender norms, the food environment and commercial messaging, the built environment including area deprivation, and socioeconomic factors including psychosocial hazards. These will be summarised in turn.

2.6.5.1 *Parental body shape*

Parental body shape can be viewed as influencing offspring body shape through both genetic and environment influences. The idea that parental body shape influences the weight of their offspring has been explored since the mid-1970s, with Albert Stunkard (US) and Thorkild Sorensen (Denmark) emerging as prominent early researchers who sought to determine the influence of inherited obesity through examination of adopted and twin children [9,10,58,60,99-101]. In 1986, Stunkard et al [10] reported regarding adoptees "... that genetic influences have an important role

in determining human fatness in adults, whereas the family environment alone has no apparent effect" (p193); and in 1990 regarding twins, "... that genetic influences on body-mass index are substantial, whereas the childhood environment has little or no influence" (p1483) [60]. Parental BMI is considered to be the most powerful determinant of their offspring's BMI [102-104].

There is ongoing debate regarding the relative importance of genetics versus environmental influences. In their 2012 study of three generations, Murrin et al [105] examined the relative maternal and paternal associations and claim to be the first study to report an enduring association between mother and offspring BMI, corroborating previous animal studies.   This is supported by other studies [99,106,107] suggesting intrauterine effects and a stronger influence on household dietary patterns, although both parents may contribute to the development of the household environment and behaviours [71,108,109]. Johnson et al [110] found that while both parents' BMI influenced adult male offspring BMI equally, maternal BMI was a considerably stronger influence on adult female offspring BMI.   Mirmiran et al [111] found that the BMI for daughters aged 10 to 17 years was correlated with their parents' BMI, but not for sons. However, this is countered by Davey Smith et al [112] who found in their investigation of intrauterine influence, that there was no difference in the relative contributions of maternal or paternal BMI to offspring BMI.

It is widely recognised that obesity has a tendency to run in families and that logically there is greater risk of having an unhealthy weight among families of obese individuals than the wider population [14]. Lee et al reported that the risk of obesity in the study participants was approximately linearly associated with their relatives.

Relatives of extremely obese women (BMI>40) were five times more likely than the general population to also be extremely obese [113]. Conversely, the risk of thinness in relatives of obese study participants was considerably lower than the general population [113]. In the Danish Nurses Cohort Study, Overgaard et al [114] concurred with earlier studies, finding that participants who had obese parents gained more weight: if both parents were obese, a gain of 5.2 kg; if only one parent was obese, a gain of 3.2 kg. In a study from the 1980s, Garn et al [11] described a curvilinear relationship between obese parents and their offspring: lower when the offspring were young, then peaking in teenage years and declining thereafter, perhaps indicating recognition of lifestyle factors as the children became young adults.

Abu-Rmeileh et al [115] and Power et al [56] found a correlation between parental and adult offspring obesity. The former found that the addition of midparental BMI (at age 40) to their regression model more than doubled the explained variation of their children's BMI from 7.7 to 17.0%; each increment of 1 kg/m^2 of the parents was associated with an increased BMI in their offspring of 0.51 kg/m^2. Power et al [56] found only a small effect when associations were adjusted for lifestyle and socioeconomic factors, but that lower social classes had a higher average BMI gain. The associations were stronger for the study participants and their offspring (younger generation) than for their parents and themselves (older generation), which the authors suggests demonstrates that "intergenerational transmission" of adiposity can be modified. This was in contrast to a study of over 40,000 participants in the Nurses' Health Study, which found that those who were overweight had a greater

intake of dietary fat and were more susceptible to weight gain without clear evidence

of a stronger association among those who had overweight parents [116].

2.6.5.2   *Self-perception of weight*

Self-perception of weight is another important risk regulator, related to cultural

norms. It has been said that *"The eye sees only what the mind is prepared to*

comprehend." Henri Bergson (1859-1941) [117]. Body image has been studied in

considerable detail during the latter half of the last century, often linked to the

growing problem of obesity within western societies. A review of the literature from

the 1960s to the 1980s highlighted increasing negative attitudes towards obesity that

produced body image disturbance and an inability to accurately assess one's own

body size, sometimes in those who had a normal weight and particularly in women

[118].

The influence of the media in promoting progressively thinner images of people led to

a global cultural/societal change in body dissatisfaction, a subsequent increase in the

use of diets, an escalation in the prevalence of eating disorders [119] and over-

estimation of weight status [120]. This change has been recognised in both women

[121,122] and in men [123]. However, some populations appear to respond to media

images of severe obesity with under-estimation of weight status, believing

themselves to be smaller/lighter than they actually are.

In their study of self-reported normal weight people and their weight perception,

Cash et al [124] found that self-classification was strongly related to psychosocial

wellbeing as well as weight concerns, attitudinal body image and reported dietary

behaviours. A large cross-sectional study among approximately 15,000 people in the European Union regarding weight perception based on self-reported BMI and using drawings of nine body shape silhouettes, found that those who were underweight had the most accurate perception, and that women generally perceived their weight status more accurately than men (57.6% compared to 32.7% respectively) [125].

An examination of sociodemographic factors, self-reported BMI and weight perception was undertaken among approximately 5400 adults in the US in the 1990s and found that those who perceived themselves as overweight, regardless of whether they were actually normal weight, overweight or obese, were more likely to be women than men, to be Caucasian than Hispanic or African-American, and to have higher levels of income and education [126,127]. These associations were strengthened by a study using the NHANES that found just over a quarter of adult respondents misperceived their measured weight status (BMI), including 38.3% of normal weight women who considered themselves to be overweight and 32.8% of overweight men who thought that they were about the 'right' weight (ie, normal weight) or underweight [128]. A further examination of the same NHANES population reported that those more likely to under-assess their weight status were aged 65 years and over, and had low levels of education and income. Those people who over-assessed their weight status were more likely to be women aged under 65 years of age (for men, aged between 35 and 64 years), and to have higher levels of education and income [129]. These findings were similar to a Dutch study [130]. Howard et al [131] examined self-reported misperception of weight status among obese participants (measured BMI ≥30) of the NWAHS, compared to their baseline biomedical measurement, and found that 59.6% considered themselves to be 'a little

overweight'. Overall, 41.5% of males and 32.2% of females under-estimated their weight, 53.6% of males and 57.3% of females were in agreement between their perception and their actual weight, while 4.9% of males and 10.5% of females over-estimated their weight [131]. Visscher et al [132] highlight that misperception of obesity among individuals results in a lack of action such as seeking help for weight management, and an acceptance at the population level of obesity as 'normal'.

Additional factors associated with weight misperception have been identified. In a study of Greek people, of those who were obese, 38% considered themselves to be overweight and of those who had a BMI<25, 21% considered themselves to be overweight [133].   The authors also found an association of under-reporting of weight with the presence of hypertension and diabetes [133]. They suggested that this may be a defensive mechanism in response to pressure from their doctor to lose weight [133]. A study that investigated stroke risk and weight perception found that overweight and obese people who were physically active were more likely to misperceive their weight as normal, and therefore consider themselves to be less likely to suffer from a stroke [134]. Powell et al [135] concurred with their findings from the Dallas Heart Study; that people who misperceived their weight believed they had a lower lifetime risk of hypertension, diabetes and myocardial infarction, with two thirds of those who were already obese believing they also had a lower lifetime risk of developing obesity.

An examination of the effect of the stress associated with the difference between actual and ideal body weight upon a person's physical and mental health was undertaken using the US Behavioral Risk Factor Surveillance System (BRFSS). The

study found those people who reported the greatest difference between what they actually weighed and what they would like to weigh, had more 'unhealthy days' (ie physical and/or mental health 'not good') than those who were satisfied with their current weight [136]. Further, obese people who under-estimated their weight status were found to undertake less binge-eating and had less eating disorder psychopathology, leading to the authors surmising that perception of obesity is related to greater psychological distress [137].

Misperception of weight does not only apply to individuals regarding their own body shapes. In a study of parents and children in the UK, Jeffery et al [138] found parents were poor at correctly identifying themselves as being overweight and also their children, particularly their sons. They hypothesised that this may be due to a reluctance to admit to a weight problem and denial of the issue, as well a general desensitisation of awareness of overweight due to increased body size becoming the 'norm' in the population. This was recognised by Lorenc et al [139] who had difficulty recruiting obese children into a study of healthy cooking programme, due to issues regarding stigma and denial of overweight. Family history of obesity and related comorbidities has also been shown to be a factor in weight misperception [140].

Accurate perception of weight has been shown to contribute to adoption of a healthier lifestyle and resulting weight loss, at least in young adults. Lynch et al [141] reported in their study of weight perception and weight change over 13 years of US adults aged in their thirties using measured BMI and the Stunkard pictograms, that women who recognised themselves as obese had lost 0.09 BMI units annually

compared to women who considered themselves as normal who gained 0.31 BMI units annually. However of concern is a finding by Burke et al [142] using NHANES data (1988-1994 and 1999-2004) that people were less likely to classify themselves as overweight in the latter survey: for women, within the 17-35 year age group; for men, across all age groups. They reported that compared to normal weight men, overweight men had a sharper decline in feeling overweight but that weight misperception did not increase markedly for men between surveys, while there was a notable decrease for women. While highlighting the positive trend towards improved body image, the authors pointed out that their findings may mean that less people may therefore seek to lose weight [142].

Similar findings of increased rates of overweight and obesity but no or little change in levels of body misperception have been reported in Switzerland [143]. The reduced imperative to change unhealthy lifestyle behaviours is borne out by Duncan et al [144], who reported that obese people who under-estimate their weight are less likely to want to lose weight or to undertake physical activity. DeBoy and Monsilovich [145] examined the growing trend to accept obesity, and argued that people are seeing obesity as another addition to the American ideal of having the "right to choose", and the increase in the number of food manufacturers producing more unhealthy food choices and in larger portions. They maintain that denial of obesity creates an escape from feelings of anxiety and guilt, as well as the need for obese people to take action to change their eating habits. They point to a US National Association to Advance Fat Acceptance (NAAFA) that aims to reduce discrimination against people who are overweight or obese, but also advocates for the condition of obesity [145].

Information from a health care professional about the need to reduce weight to improve health has been shown to increase efforts to adopt healthy choices regarding diet and exercise. Based on NHANES data from 2003-2008, Yaemsiri et al [146] reported that 74% of overweight and 29% of obese adults had not received a diagnosis of unhealthy weight from their health care professional. Further, those with a health care professional diagnosis were more likely than those without this diagnosis to diet (74% compared to 52%), exercise (44% compared to 34%) or undertake both activities (41% compared to 30%) [146]. Indeed, a review of evidence-based management strategies for treating obesity in men found being defined as obese by a health care professional to be one of the main motivators for weight loss [147].

2.6.5.3 Cultural norms/gender

In their systematic review of dietary and physical activity behaviours between childhood and adulthood, Craigie et al [148] highlight the effects of human behaviour in the development of obesity over time. A Canadian study found that for Class II (BMI 35+) obese individuals, the risk of obesity for their spouses was more than two and a half times that of healthy weight individuals, and more than seven times for their first degree relatives [149]. In the Christakis et al [14] 2007 study of over 12,000 Framingham Heart Study participants, the authors found that having a friend, sibling or spouse who became obese in a given interval, increased a person's risk of being obese by 57% (95% CI 6-123), 40% (95% CI 21-60) and 37% (95% CI 7-73) respectively. They argue that obesity spreads via social networks in a discernible and quantifiable pattern, and that people may be more influenced by people whom they resemble. Of interest was the claim by Christakis that "... the increase in obesity

cannot be explained by genetics ..." (p371), citing research by Stunkard et al who actually stated the opposite [10,60].

The information in Section 2.2 regarding the global prevalance and trends in obesity showed that in some countries, women have higher rates of obesity than men (eg Mexico, Russia, Brazil). Garawi et al [150] highlight that this may be due to differences in feminine identity across the many cultures, which is closely tied to both the production and consumption of food within the household and family unit, as well as differences in body image to men. There may also be differences between men and women regarding the physiology of eating, from the effects of genetics and reproductive hormones, resulting in both permanent early developmental effects and later effects from hormone levels [151].

In Australia, a study of morbid obesity among a population of women found an increase from 2.5% in 1993-1997 to 4.2% in 2004-2008, and there was an increase in mean BMI and prevalence of morbid obesity observed for all ages and across the socioeconomic spectrum [152]. Greater prevalence of obesity for women may not necessarily translate into poorer health outcomes, depending on fat distribution. Karastergiou et al [153] point out that for women, having a pear-shaped body can relate to lower cardiometabolic risk. Risk can also be mediated through healthier behaviours. An Australian study examined fruit and vegetable intake among ~247,000 adults aged 45 years and over, and found that overweight and obese women were more likely than men to consume the recommended levels of five serves of vegetables and two serves of fruit per day [154].

There are strong arguments to consider both men's and women's health issues in recognition of the significant biological differences that affect health and well-being [155,156]. A review of social determinants of health conducted by the WHO European Region and chaired by Sir Michael Marmot, considers that men have poorer survival because of greater occupational hazards (exposure to physical and chemical hazards), more risk-taking behaviours relating to masculinity paradigms, and their reluctance to visit health professionals or to discuss health concerns [157]. In contrast, women may be more prone to physical and emotional changes. An example of this is a study by Goosby et al [156] in which they explore how 'chains of risks' relating to birth/childhood body size and fetal origins, adolescent body size and sensitive periods (rapid physiological development and/or environmental change), and early adulthood body size and social factors (such as becoming independent and making adult decisions) lead to adult obesity and inflammation. From a gender perspective, they found that a greater sensitivity to weight gain in the early years was implicated in increased levels of inflammation for adult women compared to men [156]. They argue that for women, rapid weight gain is more harmful and that unhealthy body size, as a chain of risks across infancy to adulthood, may result in poorer health [156].

2.6.5.4 Food environment and commercial messaging

Wilding [158], in his rebuttal to Frayling [64], acknowledges that 40-70% of body fatness is inherited, but argues that the rapid increase of obesity worldwide cannot be due to genetics alone. He points to the rapid underlying changes from environmental factors such as the reduced cost and increased marketing of energy dense foods (whilst healthier options have increased in price), resulting in higher rates of obesity

among lower income households. A review of economic factors that have impacted

on increased obesity in the US found three main forces: (1) an increase in the number

of full-service and fast-food restaurants per capita; (2) decreased prices for food; and

(3) an increase in the costs of cigarettes which often results in reduced smoking and

subsequent weight gain [159].

In her review article, Gordon-Larsen [160] contends that processed foods have

become more accessible and are often cheaper, compared to relative costs of fruit and

vegetables which have become more expensive. In particular, she highlights that low

income individuals/families may be more affected by these circumstances [160]. She

also posits that low income individuals/families may be more sensitive to the

prescence of fast-food restaurants, while recognising that consumer buying patterns,

choice of store and perceived quality of produce may also be important factors in

deprived neighbourhoods [160].

In recognition of the growing problem of child obesity in developed countries,

Hawkes et al [161] undertook a review of the worldwide regulation of the

commercial promotion of food to children. They highlight the widespread cultural

phenomenon of promotion of energy-dense food and beverages, directed primarily at

children by commerical organisations. The *Process for a Global Strategy on Diet,

Physical Activity and Health* produced by the WHO in 2003 underscored the effect of

food advertising on dietary habits and food choices, and sought action by member

states to address unsuccessful attempts at self-regulation by the food industry [162].

2.6.5.5 Built environment

Changes to the urban environment such as increased crime (or perception of crime) in neighbourhoods, more dense housing and less open space, increased working hours and commitments to children and elderly parents, have also contributed to people's reduced ability or desire to be physically active, contributing to the obesity problem [26]. At the same time, many occupations and busy lives have led to more sedentary lifestyles through the increased use of motorised transport, with less time available for recreational or incidental physical activity [158].

One important factor in addressing increased physical activity is the improvement of walkability in neighbourhoods. In a 2016 study using the NWAHS, Sugiyama et al [163] reported that the cohort population had in general increased their waist circumference by 1.8 cm over a four year period. They also found that having an increased WC was associated with living a greater distance from the city centre and also in the vicinity of a suburban centre [163]. Surprisingly, in this cohort, they concluded that walkability was not markedly associated with WC [163].

Street connectivity (or permeability) can be defined as "... *the directness of links and the density of connections in a transport network. A highly permeable network has many short links, numerous intersections, and minimal dead-ends.*" [164] One US study assessed the association between electronic health record data regarding BMI from a public health centre and local environment walkability using street connectivity [165]. It confirmed the effect of good street connectivity and the weight status of the local population, finding an inverse association between intersection density and

BMI, observed in multilevel models that controlled for age, gender, race, and marital status [165].

2.6.5.6 *Socioeconomic factors*

Socioeconomic factors such as increasing car ownership and television viewing have decreased the amount of physical activity undertaken by the population, together with an increased availability of relatively low cost but high fat and energy-dense processed foods and beverages [166]. In particular, the adverse impact of sugar-sweetened beverages has increasingly gained prominence with a shift in focus from dietary fat, as has the deleterious effects of shift work and associated lack of sleep [166]. Other studies have identified a range of factors that contribute to childhood obesity such as living in a single-parent household [167], full-time maternal employment [168], and regular consumption of away-from-home meals rather than meals prepared and cooked in the home [169].

Regarding socieconomic determinants of obesity, in an Australian study that examined the socioeconomic gradient of overweight and obese adults by BMI, typical results were found – increasing income was associated with a decrease in obesity prevalence [170]. However among overweight males, a reverse socioecomic gradient was observed [170]. This was similar to results found by Hajizadeh et al [171] in a study of Canadian adults, who also reported that obese men were more likely to be economically well-off, compared to women who were more likely to be obese if they were economically disadvantaged and that for men, this trend was increasing over time. The authors reported that key factors associated with this included income,

education and immigration, as well as health-related risk factors such as alcohol consumption and physical activity [171].

Early-life predictors of obesity include socioecomic factors. In a systematic review, Cameron et al [172] report that children who have a lower socioeconomic position experience a steeper weight gain from birth, and also a strong socioeconomic gradient in child and adult obesity prevalance. They point to strong evidence of the link between socioeconomic position and risk factors in the pre-pregnancy period (such as maternal BMI, diabetes and diet), the ante-/peri-natal period (such as maternal smoking and infant low birthweight) and in early life (such as breastfeeding, introduction of solids, the home food environment and children's television viewing) [172] .

A study of socioeconomic status (SES) over the life course on genetic variants showed that the genetic influence on BMI has become stronger in more recent times, and suggested that persistently low SES, or a transition from high SES in childhood to low SES in adulthood, increased the genetic influence on BMI. Those with high SES during childhood or who transition from low SES in childhood to high SES in adulthood, were able to compensate for this genetic influence [173].

2.7 Measurement of obesity

Measurement of obesity is an integral part of determing correlations of various factors with obesity. A number of anthropometric measures have been used by

researchers to identify the weight status of individuals, including BMI and central

adiposity, including WC, WHR, WHtR and more recently, ABSI.

2.7.1 Body mass index

BMI is a calculation of a person's overall obesity by dividing their weight (in

kilograms) by their height (in metres^2). It was invented by Adolphe Quetelet between

1830 and 1850 [174], and was initially known as the Quetelet Index before being

re-titled "body mass index" by Keys in 1972 [175]. Its limitations were discussed as

early as 1989: the measurement may be affected by stature, relative leg length or

relative sitting height and may reflect both lean and fat tissue [11]. BMI may also

have a small degree of bias when compared to measured height and weight [176-

179]. However, it can be based on either self-reported or measured factors and has

been an accepted inexpensive and relatively simple measure for use in population

studies.

Based on this measure, the WHO defined the following weight classifications for

adults: underweight <18.50; normal range 18.50 to 24.99; overweight ≥25.00; obese

≥30.00. Within the obese classification, there is a further set of cut-offs for obese

Class I (30.00 to 34.99), Class II (35.00 to 39.99) and Class III (≥40.00) [180]. The

WHO recommend that the cut-off points of 23, 27.5, 32.5 and 37.5 kg/m^2 be added as

points for public health action; and that all countries use the cut-off points of 18.5, 23,

25, 27.5, 30, 32.5 kg/m^2 and where relevant, cut-off points of 35, 37.5, and 40 kg/m^2,

for reporting purposes so that international comparisons can be undertaken [180].

BMI has been strongly associated with CVD, diabetes and hypertension [4,181,182].

2.7.2 Central adiposity – waist and hip circumference

Since the mid 1950s, attention has been focused on central adiposity and its relation to obesity-related chronic disease. It is recognised that android or "apple" shaped bodies have a stronger association with obesity-related health risks that gynoid or "pear" shaped bodies [183]. WC and HC allow the calculation of WHR (WC divided by HC) providing another measure of centralised fat distribution.

A number of cut-off points for central adiposity have been recommended. O'Dea et al [184] state that android obesity is indicated for a WHR of greater than 1.0 for men or 0.85 for women, while Han et al and Lean et al [185,186] recommend that if the WC is greater than or equal to 95 cm for men or 80 cm for women, that no further weight be gained and further that weight reduction is advised for those men with a WC of ≥100 cm and for those women with a WC of ≥90 cm. Overall, it has been found that some people can be classified as having a low or normal BMI whilst having a WC within the range where it is recommended no further weight be gained and indeed, weight should be lost [186]. This was found to be more likely among females [186]. However it is considered that both measurements are useful in assessing obesity in populations [187].

2.7.3 Waist-height ratio

A third measure incorporating anthropometric measures is the WtHR which was first suggested in the mid 1990s. It has a cut-off score of 0.5, translatable as a need to keep one's WC to less than half one's height. WHtR when compared to BMI was found to improve discrimination of adult cardiometabolic risk factors by 4-5%, compared to WC (3%) [188]. Further, WHtR was shown to be significantly better than BMI in

screening for diabetes, CVD, dyslipidaemia and hypertension (and the metabolic

syndrome overall) [188].

2.7.4   A Body Shape Index (ABSI)

A more recent formula for predicting premature mortality from body shape

measurements is the ABSI, created by Nir and Jesse Krakauer [7] which accounts for

the additional risk of having a high WC while adjusting for height and weight.

The ABSI was developed in 2012 using a population of 14,105 US adults from the

1999-2004 NHANES, with an average five year follow-up for mortality (828 deaths).

Krakauer and Krakauer [7] found that death rates increased approximately

exponentially when the baseline ABSI was above the average. They reported that

overall, 22% (95% CI 8-41%) of the population mortality hazard was associated with

high ABSI – in contrast to 15% for both BMI and WC (95% CI 3-30% and 4-29%

respectively). They found the association held even when adjusted for known

confounders such as diabetes, blood pressure, cholesterol and smoking, and across

age, sex, BMI, and Caucasian and African American ethnicities [7].

In 2014, the same authors evaluated the ABSI using data from the British Health and

Lifestyle Study (HALS) collected in 1984/85 and 1991/92 to 2009 (7011 individuals;

2203 deaths) [189]. They reported that the ABSI was found to be a strong indicator

of all-cause mortality hazard in this United Kingdom (UK) population; there was an

increase in death rates by a factor of 1.13 (95% CI 1.09-1.16) per standard deviation

increase in ABSI. Compared to those with an ABSI score in the lowest 20% of the

population (with a corresponding lowest BMI and/or WC), those in the highest 20%

had a hazard ratio (HR) of 1.61 (95% CI 1.40-1.86). The authors asserted that ABSI outclassed traditional central adiposity measures and was a consistent predictor of mortality hazard over a minimum follow-up period of 20 years. Another important finding was that those individuals who had a lower ABSI at the second examination (ie, those who had lost weight/had a smaller WC) had a lower mortality hazard risk than those who had a subsequent higher ABSI [189].

Among 77,505 US postmenopausal women, ABSI was shown to be a more useful measure for mortality risk than BMI or Body Adiposity Index *(BAI = ((hip circumference)/((height)$^{1.5}$) − 18))* [190,191]. ABSI had a linear association and HR 1.37 (95% CI 1.28-1.47) at the highest quintile, compared to U-shaped associations and HRs of 1.06 for BAI (95% CI 0.99-1.13), 1.21 (95% CI 1.13-1.29) for the highest quintile of WC, and 1.30 (95% CI 1.20-1.40) for BMI Classes II/III [191].

ABSI has been explored using different populations. In 2015, the association of ABSI was explored with all-cause, cardiovascular- and cancer mortality (22 years of follow-up - 3675, 1195 and 873 deaths respectively), using a middle-aged and elderly population of 2626 men and 3740 women from the Netherlands. Compared to BMI, WC, WHtR and WHR, the study found that ABSI had a stronger association with mortality with a HR per 1 SD increase in ABSI of 1.15 for men and 1.10 for women (95% CIs 1.08-1.29 and 0.99-1.22 respectively). The authors found ABSI to be more informative as a predictor of total mortality, and it also improved risk stratification [6]. In a Middle Eastern population of 9242 people followed for approximately 10 years, ABSI was found to be the strongest predictor of all-cause mortality for Iranian men, however WHR was identified as a better mortality predictor for Iranian women

[192]. He et al [193] showed in their study of a Chinese population of 780 middle-aged men over 15 years of follow-up, that ABSI was not associated with mortality. Similarly, Tian et al [194] found ABSI to be the weakest predictor of cardiometabolic abnormalities in a Chinese population of 8126 people [194]. Both studies suggest that use of ABSI is limited with Asian populations. However, ABSI was shown to be a useful marker of arterial stiffening in a Japanese population with type 2 diabetes [195]. Arterial stiffness can be one manifestation of atherosclerosis, and has been strongly linked with cardiovascular events and mortality among people with type 2 diabetes.

ABSI has also been highlighted as a potentially useful tool in a range of other applications, including in the identification of sarcopenic obesity in men (decreased fat free mass and increased fat mass), following investigation within an elderly Netherlands population [196]; as an easily determined and reliable means of predicting outcomes in laparoscopic liver resections with a high degree of difficulty [197]; and also surgical complications in gastric cancer patients [198].

2.7.5 Pictograms

Pictograms of body shapes are a tool that can be easily utilised in a primary care setting by general practitioners and other physicians to ascertain the overall size of their patients' family members (including siblings, parents and grandparents, including those who are deceased) at a time-point, providing an opportunity to discuss changes in lifestyle towards a more healthy weight.

Pictograms generally contain five to twelve figures. One set of pictograms frequently utilised in research studies is the ordinal series of nine silhouettes of both men and women (ranging from very thin to obese) devised by Stunkard et al in 1983, originally formulated to determine the body build of the parents of both adoptees and biological parents where self-reported and/or measured information was not available. They were initially validated using self-reported recall of parental body shape compared to measured parental weight in a sample of 1000 people from a different research study, and found to be "surprisingly accurate" (p119) [9]. Sorensen et al [101] further examined the accuracy of pictograms based on research conducted on 251 children and their recall of their parents' weight some 15 years earlier using both recalled height and weight as well as a selected figure. The correlation between measured and reported was 0.82 for mothers and 0.56 for fathers, with some under-estimation of measured values in the high range and over-estimation of those in the low range [101]. Further, the authors found that neither the age, sex, BMI, height, skinfold thickness nor confidence of the subject's ability to recall this information had any major influence on the accuracy of their report. Pictograms have since also been validated by other studies, with one European study of self-reported BMI versus silhouette selection found females were more accurate then males (57.6% versus 32.7%), and underweight people more accurate (79.3% of females, 92.9% of males in this BMI classification) [125]. Within their age categories, older men (38.3%) and younger women (60%) were also found to be more accurate [125]. Similarly, Bulik et al [199] used receiver-operating characteristic (ROC) with self-reported BMI and silhouette data on 3347 individual US twins, and found the figural stimuli to be effective in classifying individuals as thin or obese. Further, Keshtkar et al [200] compared measured BMI to pictograms in an Iranian population, also using ROCs, and

found sensitivity values of 77% for obese females and males (discrimination from normal/overweight subjects), and 77-82% for obese/overweight (discrimination from normal subjects) and corresponding specificity values of 75-79% and 73-76%.

Some limitations have been identified with the Stunkard pictograms such as the coarseness of the scale; the lack of the same exact interval between each of the silhouettes; that the figures may not accurately depict the changes that occur during weight gain; that the figures do not take into account differences between respondents regarding where on the silhouette they fixate their gaze to make their judgement [201]; and that the figures are not consistent with social norms of desirable body weight and shape, which may result in mis-identification [202]. Pictograms can be used in field studies instead of taking anthropometric measures where cost and practicalities make this difficult, particularly in developing countries, and also in questionnaire-based studies [114,203]. They have been used in studies for population norms [199]; to determine people's perception of themselves currently, and also as an indication of how they would like to look (ideal body size) in research examining populations of different ethnicity and gender [202,204,205] and weight change [141].

2.8 Research questions

The preceding information has provided a basis for exploring the following research questions in the Australian population:

1)   Is there a dose-response association between obesity, as measured by central adiposity, BMI and ABSI, and mortality risk? *(Chapter 4)*;

2)   Is there an association between parental body shape in mid-life and adult offspring BMI and central adiposity? *(Chapter 5);*

3)   What is the degree of misperception of body weight within an Australian adult population? *(Chapter 6);* and

4)   Is there an association between parental body shape in mid-life and adult offspring's self-perception of their own body shape? *(Chapter 6).*

Although there have been a number of studies since 2012 based on the ABSI, there was no research that specifically explored cause of death across ABSI quartiles, nor were there any Australian studies as yet using this measure. This led to the first research question. Data from the NWAHS included the necessary anthropometric measurements to calculate ABSI quartiles and a continuous score, and to analyse this using information from those participants who had died, including hazard ratios and cause of death analysis.

The majority of studies that have examined the link between parental and adult offspring obesity, have used studies outside Australia and have primarily used BMI as their weight measure. The aim of the study in Chapter 5 was to contribute to the literature on this topic by answering the second research question using anthropometric measures including both WC and HC, from the NWAHS of South Australian adults. It was therefore the first Australian study to address this question.

An examination of factors contributing to obesity uncovered a lack of studies that looked at the degree of obesity misperception within an Australian population, and whether mid-life parental body shape had an association with self-perception of

weight status. This led to the third and fourth research questions examined in a study comprising Chapter 6. These questions were examined using data from the NWAHS that included a question in a telephone survey asking adult participants about their body weight, which was compared to subsequent anthropometric measurements to classify people as either pessimistic, optimistic or realistic about their weight, and then matched with information about their parents' body shape.

Methodology of the NWAHS is detailed in Chapter 3, followed by the results of the studies addressing the research questions in Chapters 4 to 6.

3.1   Introduction

All three studies in this research used data from the NWAHS [206,207], a longitudinal cohort study that was established in 1999 to provide sought-after information about South Australians. It was well suited for the aims of this thesis due to its wealth of sociodemographic, health-related risk factors (including parental body shape at midlife and self-perception of weight) and chronic disease information (including obesity), as well as its anthropometric measurements (height, weight, WC and HC), and linked mortality data.

The candidate was the Study Co-ordinator of the NWAHS for over 13 years and as such, assisted with the first telephone follow-up in 2002 and then facilitated much of the study from Phase 1B in 2002 (used to supplement the original sample of 2523 with an additional n=1537), through Stage 2 (2004-06), the second telephone follow-up in 2007, Stage 3 (2008-2010) and assisted in the most recent 15 year online/postal follow-up in 2015. The candidate was instrumental in its day-to-day operation of the study, including the setting up, implementation and maintenance of the recruitment telephone survey, appointment information (brochures, maps and reminder sheets) and paper-based questionnaire as part of daily mail-out to new participants, as well as assistance with the biomedical clinic examination. Her tasks included the creation of many documents and forms; liaison with study recruitment, office and clinic staff, as well as participants; creation of analysis and cohort detail statistical and study-detail databases; management of data collection; development of multiple submissions to ethics committees; analyses of a wide range of socio-demographic, health-related risk factor and chronic disease variables; the reporting of study results and study information in publications, reports and conference 76

presentations, and the organisation of participant events, as well as ongoing maintenance of requests for study data, co-ordination of a weekly mail-out of birthday cards, following up of 'lost' participants; an annual newsletter and involvement with the media and the management of two websites (one for participants and one for health professionals and researchers). The candidate was also the executive officer for the study management committee.

For this thesis, the candidate initiated theme topics, and researched the literature for each, and undertook deep analysis of a wide range of associated factors relating to participants' weight measures including premature mortality, parental body shape and perception of weight. Part of the requirements for PhD included a detailed written and presented proposal of the themes. The candidate drafted and updated all three publications and undertook a range of statistical analyses to present results and their discussion, following consultation with supervisors.

In this chapter, the data source and methods for measuring obesity and parental body shape are described. The methodology for each study has been described in each paper (Chapters 4, 5 and 6), however the NWAHS, as the data source, has also been summarised below.

3.2 Overview of the North West Adelaide Health Study

The NWAHS was initially formulated in 1997, due to a lack of longitudinal biomedical data on chronic conditions in South Australia. Trend analyses of 1990s data from a Statewide face-to-face health survey showed increases in the prevalence of diabetes and asthma [206] and these prevalence rates formed the basis for the initial sample

size calculation. Overall, 4060 participants were randomly recruited from the north-west suburbs of Adelaide, with the sample region representing approximately half of the metropolitan area. Adelaide is the capital city of South Australia with a population of ~1.1 million people, which represents 78% of the total State population of ~1.7 million people [208]. Baseline recruitment was undertaken in Stage 1 (1999-2003) in two stages: Phase 1A from 1999-2000 and Phase 1B from 2002-2003. Cohort status was achieved in 2004-2006 with Stage 2 of the study, and Stage 3 was carried out in 2008-2010. The study was a collaboration of the State Department of Health (SA Health), two major public teaching hospitals (The Queen Elizabeth Hospital and the Lyell McEwin Hospital), two State universities (The University of Adelaide and the University of South Australia), and a major pathology organisation (Institute of Medical and Veterinary Science, now SA Pathology). The research team used quantitative and qualitative methodologies via a wide range of disciplines, including academic and clinical medicine, public health, epidemiology, social science and nursing.

3.3 Sampling

All households in the northern and western areas of Adelaide with a telephone connected and a telephone number listed in the Electronic White Pages (EWP) were eligible for selection in the study. The sample was stratified into two health regions: northern Adelaide and western Adelaide. These regions reflect the demographic profile of the State's population.

3.4 Recruitment

Participants were recruited using the telephone to conduct the interviews and the EWP as the sampling frame. Within each household, the person who had their birthday last and was aged 18 years and over, was selected for interview and invited to attend the clinic for a biomedical examination. This method of randomly selecting within the household avoids bias towards unemployed and retired people or homemakers (often women) as those most likely to be home at the time that the interviews are conducted [16].

Exclusion criteria were applied by recruiting staff at the initial telephone contact. Interviewers were responsible for determining if the selected respondent had sufficient intellectual ability to understand the implications and requirements of participating and if not, to thank the respondent for their time and terminate the interview. Those people who indicated that they were too ill to participate were similarly questioned by the interviewer to ascertain if they had sufficient physical abilities to attend the clinic, either using their own transport means or a taxi provided by the study. Both categories were coded by interviewers as "too sick" to participate in the study (n = 77). There were 215 people who were eligible but uncontactable. Of those who were eligible to participate in the study and could be contacted, 2148 respondents (26.9%) refused any participation in the study due to a number of factors including being too busy, not wanting to participate, or considering themselves to be too old. This cohort study did not recruit people residing in institutions, such as nursing homes (the majority of whom are elderly women) because of the inability to randomly select one individual from the group living

arrangement. However people who had their own telephone number and who were living in individual units attached to a nursing home were eligible to participate.

The study did not include those people from a non-English speaking background who could not communicate sufficiently well with the telephone interviewer and who could not answer questions at the initial recruitment stage, although every effort was made to encourage family members to assist in translating. A short trial of having an interpreter present if required for the clinic examination, was found to be neither time nor cost-effective. A review of non-participants found that only four people had refused to participate in the study due to the perceived language barrier. In addition, the small number of Aboriginal and/or Torres Strait Islander people (n = 20) recruited in this cohort means that no association or causality inferences can be made on their data because of the potential for misrepresentation, as highlighted by the Aboriginal Health Research Ethics Committee (South Australia).

To maximise the response rate, the telephone interview was restricted to approximately 15 minutes and a letter and information brochure, including endorsements by prominent South Australian sports people, were sent to the household of each selected telephone number, informing the household of the purpose of the study and indicating that they would be contacted by telephone about participating. Within two weeks of the letter and brochure mail-out, telephone calls were made to householders at various times of the day and evening, both during the week and on the weekend. The interviews were conducted in English by professional interviewers. Up to ten calls were made to each household in an effort to speak to someone. Repeated unsuccessful attempts that resulted in either no answer, the busy signal or a message being left on an answering machine were considered to be "non-

contacts" and regarded in the same light as refusals. Contact details supplied in the approach letter and brochure did however enable those participants who had not been contacted and who did want to take part to call the study co-ordinator to arrange a mutually convenient time for the telephone recruitment interview.

3.5 Response rates

Stage 1 of the study (January 1999 to June 2003) began with an initial sample of 10,096 people. A number of people were ineligible to take part (n=1883), due to non-connected or non-residential telephone numbers, or fax/modem connections. Of the remaining 8213 who were eligible to take part, 2363 people were uncontactable or did not complete the initial telephone interview. Of the 5850 people contacted, 4060 completed all three aspects of the study (telephone survey, questionnaire and clinic examination) representing 49.4% of the eligible population. A telephone follow-up survey of 3622 participants (91.7% response rate) was undertaken in March 2002. Stage 2 of the study (May 2002 to February 2006) began with an eligible sample of n=3957, with 3564 participants providing information (90.1% participation rate); of these, 3206 participants also attended the study clinic, resulting in a 81.0% response rate. A second telephone follow-up survey of 2996 cohort participants was carried out from July to November 2007 (response rate 79.7%). Stage 3 of the study (June 2008 to May 2010) resulted in 2710 participants providing information (66.8% participation rate based on the original eligible sample); of these, 2487 participants also attended the study clinic, resulting in a 61.5% response rate (eligible sample) [207,209].

3.6 Obesity measures

3.6.1 Offspring obesity measures

Four anthropometric measures of study participants were undertaken. Height without shoes was measured to the nearest 0.5 centimetres using a wall-mounted stadiometer. Weight was measured to the nearest 0.1 kilogram in light clothing and without shoes using standard digital scales. WC was measured to the nearest 0.1 centimetre using an inelastic tape maintained in a horizontal plane, with the subject standing comfortably with weight distributed evenly on both feet. The measurement was taken at the level of the narrowest part of the waist. HC was also measured using an inelastic tape, at the level of the maximum posterior extension of the buttocks.

BMI was calculated by dividing the participant's weight in kilograms by the square of their height in metres (kg/m^2). BMI values were initially grouped according to the WHO BMI classifications [180].

Three measurements of the waist and hip were taken and the mean for each was calculated. The cut-off points for recommended weight reduction to reduce major cardiovascular risk factors using WC were ≥102 cm for men and ≥88 cm for women [186], and a WHR of >1.0 for men and >0.85 for women [210]. The WHR is a proxy for central (visceral) adipose tissue [211]. The cut-off point for WHtR indicating a reduction in cardiometabolic outcomes was 0.5 [212].

The ABSI uses height, BMI and WC in its formula:

$$\frac{WC}{BMI^{2/3} \times Height^{1/2}}$$

A z-score can also be calculated from the ABSI mean and standard deviation (SD) within a population, dependent upon adjustment for age and sex:

$$\frac{ABSI - ABSI_{mean}}{ABSI_{SD}}$$

A high ABSI z-score indicates that a person's WC is more than expected, given their height and weight, corresponding to a higher concentration of body volume centrally [7].

3.6.2 Parental obesity measure

In Stage 3, participants were asked in the self-completed questionnaire to provide the body shape of their parents at age 40, using the pictogram in Figure 3.1 [9,203]:

Which body type did your biological MOTHER have, when she was 40 years old?

(tick the drawing that best applies)

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
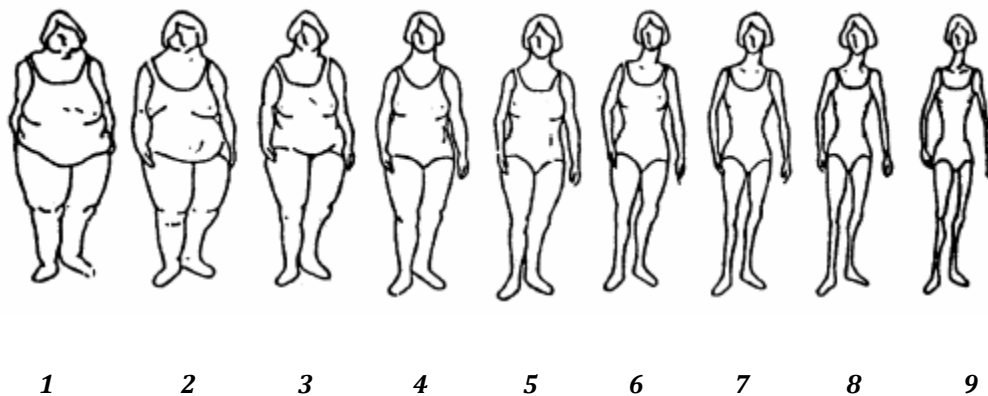
OR ☐ Not applicable/Don't know

Which body type did your biological FATHER have, when he was 40 years old?

(tick the drawing that best applies)

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
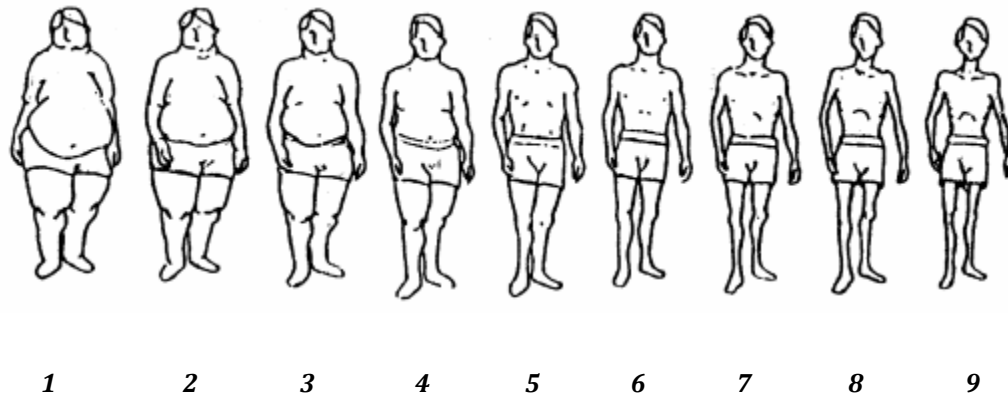
OR ☐ Not applicable/Don't know

Figure 3.1 Pictogram of parental body shapes
Source: Stunkard, Sorensen & Schulsinger, 1983; Hundrup, Simonsen, Jorgensen & Obel, 2011 [9,203]

For scoring purposes, Silhouettes 1-2 were classified as very overweight; Silhouettes 3-4 as moderately overweight; Silhouette 5 as slightly overweight, Silhouettes 6-7 as appropriate (healthy) weight; and Silhouettes 8-9 as underweight [204]. Permission for use of the figure for publication relating to this thesis was granted by the Nature Publishing Group in September 2014.

3.7 Mortality

The NWAHS maintain follow-up of their participants. At the time when the first research question was examined, 581 participants (14.3% of the original cohort) died between January 2000 and September 2015. More detail is provided in Chapter 4 regarding the notification of deaths. Confirmation of death and date is provided in South Australia through the State Births, Deaths & Marriages Office and nationally, through the National Death Index which is maintained by the Australian Institute of Health and Welfare with the National Coronial Information Service (NCIS). Cause of

death information is provided by NCIS after two years of date of death. The 10th revision of the ICD was used to classify causes of death.

3.8 Analytical approaches

A range of analytical approaches were used in each study (see Chapters 4, 5 and 6 for more detail) including univariable analyses of demographic, health-related risk factors including weight measures and mortality outcomes. A Cox proportional hazards survival model was used to answer the first research question. Odds ratios and a sensitivity analysis were reported for the second research question. Multinomial regression, including the calculation of relative risk ratios, was undertaken for the third research question. In a number of instances, analyses compared males and females to distinguish gender differences across socio-demographic, lifestyle and biomedical variables. Supplementary tables have been provided at the end of each paper, together with additional tables with added ABSI information for previously published papers.

3.9 Ethical approvals

3.9.1 North West Adelaide Health Study

Ethical approval was sought and granted for each stage of the NWAHS, through the Human Research Ethics Committee that oversees the two participating hospitals in the study: The Queen Elizabeth and Lyell McEwin Hospitals.

3.9.2 Thesis

Ethical approval for this research was granted by the Low Risk Human Research Ethics Review Group (Faculty of Health Sciences), The University of Adelaide, in October 2013 (Approval No. HS-2013-055) and extended in October 2016 for a further three years.

Chapter 4 The association between A Body Shape Index and mortality: results from an Australian cohort (Publication)

4.1 Statement of Authorship

Title of Paper	The association between A Body Shape Index and mortality: results from an Australian cohort
Publication Status	☑ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmited work written in manuscript style
Publication Details	PLoS ONE. 2017;12(7): e0181244. https://doi.org/10.1371/journal.pone.0181244

Name of Principal Author (Candidate)	Janet F Grant
Contribution to the Paper	Participated in the design and co-ordination of the study, performed statistical analyses, and drafted and revised the manuscript.
Overall percentage (%)	80%
Certification	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date 7/9/17

Name of Co-Author	Catherine R Chittleborough
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

Name of Co-Author	Zumin Shi
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

Name of Co-Author	Anne W Taylor
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

4.2 Chapter 4 Contextual Statement

In the initial stages of this research, it was planned to examine whether parental midlife body shape was associated with mortality, however the questions accompanying the set of pictograms of body shapes of both mothers and fathers were not asked until Stage 3 of the NWAHS (conducted from 2008 to 2010). Therefore there was not a sufficient number of deaths to undertake statistical analysis for this thesis.

Instead, a relatively new prediction measure of mortality (ABSI) was utilised with anthropometric participant data from the NWAHS, in association with all deaths recorded and where available, the cause of death. The ABSI incorporates WC as a measure of central adiposity, together with BMI, and provides a more accurate prediction of premature mortality from overweight and obesity. Additional ABSI analyses were conducted following publication of the second and third papers and have been included under the heading *Extra analyses not included in the published paper* (Sections 5.10 and 6.10 respectively)*.*

The aim of this study was to answer the first research question *"Is there a dose-response association between obesity, as measured by central adiposity, BMI and ABSI, and mortality risk?"*. It was the first study, to our knowledge, to examine this question in Australia, finding that those people who had the combination of the highest BMI and WC, as calculated by the ABSI, had the highest risk of premature mortality; more than two and a half times those with the lowest mortality risk. The risk of dying prematurely increased steadily across the ABSI, being one and a half times higher for those in the second quartile, to almost two times higher for those in the third quartile.

The following chapter presents the results of this investigation of incorporating WC

as a measure of central adiposity, with height and BMI, as a predictor of premature

mortality across quartiles of an Australian population. This manuscript was

published by PLoS One on 31 July 2017, and has been re-formatted to meet the

requirements of this thesis. The manuscript in its published format is included in this

thesis as Appendix 2.

4.3 Abstract

It is well recognised that obesity increases the risk of premature death. ABSI is a formula that uses WC, BMI and height to predict risk of premature mortality, where a high score (Quartile 4) indicates that a person's WC is more than expected given their height and weight. Our study examines the association between ABSI quartiles and all-cause-, cardiovascular- and cancer-related mortality, and primary cause of death. Self-reported demographic and biomedically measured health-related risk factor and weight data was from the baseline stage of the NWAHS (1999-2003, n=4056), a longitudinal cohort of Australian adults. Death-related information was obtained from the National Death Index. Primary cause of death across ABSI quartiles was examined. The association between mortality and ABSI (quartile and continuous scores) was investigated using a Cox proportional hazards survival model and adjusting for socioeconomic, and self-reported and biomedical risk factors. The proportion of all three types of mortality steadily increased from ABSI Quartile 1 through to Quartile 4. After adjusting for demographic and health-related risk factors, the risk of all-cause mortality was higher for people in ABSI Quartile 4 (HR 2.64, 95% CI 1.56-4.47), and ABSI Quartile 3 (HR 1.95, 95% CI 1.15-3.33), with a moderate association for the continuous ABSI score (HR 1.32, 95% CI 1.18-1.48). ABSI is therefore positively associated with mortality in Australian adults. Different combined measures of obesity such as the ABSI are useful in examining mortality risk.

4.4 Introduction

It is well recognised that obesity increases the risk of premature death. A number of studies from the US, but also from studies based in Europe, Asia and Australia, have examined the link between obesity and mortality based on a range of individual anthropometric measures including BMI [4,37,38,213,214], WC [47], waist hip ratio (WHR) [50], together with studies that have combined all or some of these measures [40,46,49] with the addition of skinfold measures [48] and WTR [51].

Regardless of which obesity measure was used, there was a general consensus that being overweight and particularly obese, increased the risk for premature death from all-causes, and for CVD conditions and cancer. Wang et al [23] report that for each additional 5 kg/m^2 in BMI, there is a subsequent increased risk of developing oesophageal (52%) and colon (24%) cancer for men, and for endometrial (59%), gall bladder (59%) and postmenopausal breast cancer (12%) for women. In their study of the projected health and economic burden of obesity for 2030 in the US and the UK, they estimate that 2.1-2.4 million (US)/179,000-230,000 (UK) incident cases of diabetes, 1.4-1.7 million (US)/122,000 (UK) incident cases of cardiovascular disease and 73,000-127,000 (US)/32,000-33,000 (UK) incident cases of cancer may be preventable, if adults were to reduce their BMI by 1% [23].

Similar results may be possible in Australia, due to the similarity of primary causes of death. Nine of the twenty leading causes of death for 2015 for Australians were obesity-related, namely (in order) ischaemic heart disease (1), cerebrovascular diseases (3), diabetes (6), colon and associated cancers (7), heart failure (9), breast

cancer (14), pancreatic cancer (15), cardiac arrhythmias (17), and hypertensive diseases (18) [215].

Studies based on BMI highlighted the 'obesity paradox', finding concave associations of people with both lower and higher BMIs having lower survival rates than those in the normal-overweight BMI range [37,39,216]. These findings have been countered by other studies that have highlighted the limitations of using BMI in prediguish mortality due to its inability to measure central adiposity or to distinguish between fat mass and lean body mass. BMI has undetermined validity for use as a measure of fatness in older people (as aging is generally associated with a considerable loss in lean body mass and some increase in fat mass). Further, a low BMI may result from one or more underlying issues, possibly distorting the association between body shape and premature mortality. An additional element is the generally lower body weight of smokers who consequently have higher rates of premature mortality [47,217,218]. The importance of including measures of central adiposity were emphasised in a recent study of all-cause and CVD-related mortality risk among people who had normal weight but with central obesity, which found twice the mortality risk of those who were overweight or obese based on BMI alone [5].

In recognition of the need to incorporate a measure of central adiposity in a formula that could more accurately predict mortality risk than using BMI alone, ABSI was developed [7]. This index is based on a person's WC whilst adjusting for their height and weight [6]. The ABSI was developed to examine the mortality hazard and characteristics of populations, and its predictive power was proven to be consistent over a minimum period of 20 years [189].

The ABSI is relatively new and therefore studies are still emerging regarding different aspects of this measure, including differences between males and females. A recent study validated ABSI for its predictive power for total and cause-specific mortality in comparison with BMI, WC, WHtR and WHR in a middle-aged population of 2626 men and 3740 women from the Netherlands, and found ABSI to have a stronger association than the other weight measures but with limited added predictive value. It also found a stronger association with mortality with a HR per 1 SD increase in ABSI of 1.15 for men and 1.10 for women (95% CIs 1.08-1.29 and 0.99-1.22 respectively) [6].

During a review of literature regarding the ABSI, it was observed that no study had yet explored the cause of death for those within each ABSI quartile. Our study provides these characteristics, as well as assessing the predictive power for mortality of ABSI using both quartiles and the continuous score for all-cause, CVD-and cancer-related mortality in an Australian population.

4.5 Material and methods

4.5.1 Sample

The NWAHS is a longitudinal study of 4056 randomly selected adults aged 18 years and over recruited from the north-west region of Adelaide, the capital of South Australia. All households in the northern and western areas of Adelaide with a telephone connected and a telephone number listed in the EWP were eligible for selection in the study. Participants were recruited from 1999 to 2003 through an initial Computer Assisted Telephone Interview (CATI), and the eligible adult to have had the most recent birthday in the household was invited to participate.

Respondents were excluded if they did not have the capacity to participate due to illness or intellectual limitations, together with those who were unable to communicate in English and those living in a residential institution. Data collection incorporated questions in the CATI survey and a self-completed questionnaire, as well as a biomedical examination that included anthropometric measures. The study methodology has been previously described in detail [206,219]. Our study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the Human Research Ethics Committee of The University of Adelaide and of the Central Northern Adelaide Health Service (The Queen Elizabeth and Lyell McEwin Hospitals). Written informed consent was obtained from all participants.

Data used in this paper are drawn from baseline recruitment (Stage 1, n=4056, response rate 49.1%). Characteristics of the study participants at baseline, as well as a comparison with Australian Census and local data, have been published elsewhere [220]. The main analysis sample (n=3311) comprised those participants with complete data available for those variables included in the model, including those participants who died between 2000 and 2015. Missing values were not imputed due to very few differences being observed between the baseline and analysis samples.

4.5.2 Mortality

Overall, 581 participants (14.3% of the original cohort) died between January 2000 and September 2015. There are four administrative levels regarding notification and confirmation of cohort participant deaths. Of the 581 deaths, 207 deaths had minimal information available for analysis purposes.

The first level related to the most recent deaths (n=32), which had been communicated by family or friends to the cohort study co-ordinator, but were yet to be confirmed by the South Australian Births, Deaths & Marriages Office. The second level related to a number of deaths of which the study had been notified within the past year, which were subsequently confirmed by this authority (n=104).

 The third level involved a process where demographic data of both those participants who were being tracked as well as notified deaths (from family and friends, as well as notifications from the registry of South Australian Births, Deaths and Marriages) are submitted on an annual basis to the National Death Index (NDI), and matched using a probabilistic record-linking software with the National Mortality Database (NMD), facilitated by the NDI which is maintained by the Australian Institute of Health and Welfare, and the National Coronial Information Service (NCIS). The NDI uses multiple passes that incorporates full names, sex, dates of birth, last contact and death, with the Australian State at their last known address in a weighted algorithm that identifies matches between NDI records and study participants.

Date of death was only available from the NDI for the more recent deaths (from 2013 onwards, n=71). For our study, the date of last contact was used if the date of death was unable to be determined. There is at least a two year time lag following preparation and submission of cohort participant details to the NDI regarding cause of death information; this was subsequently provided on 374 participants at the fourth and final level (from January 2000 to December 2012).

One primary and up to seven secondary causes of death were supplied using the 10th revision of the *International classification of diseases* (ICD10). Cardiovascular disease (CVD)-related deaths were classified as ICD10 codes of I00 to I99 (diseases of the circulatory system), while cancer-related deaths were classified as ICD10 codes of C00 to D49 (neoplasms).

4.5.3 A Body Shape Index

The ABSI was developed as an indication of risk that incorporates the excess risk of WC while adjusting for BMI and height [7]. The ABSI was based on baseline data on non-pregnant adults aged 18 years and over (n=14,105) from the 1999-2004 US NHANES, and was evaluated for prediction of mortality using the US National Death Index data through to December 2006 (2-8 years of follow-up; average 5 years (828 deaths) [7]. Krakauer and Krakauer [7] performed linear least-squares regression on log(WC) as a function of log (height) and log (weight) for the sample, and then approximated the obtained regression coefficients with ratios of small integers (WC ∞ weight$^{2/3}$ height$^{-5/6}$) to produce the final formula (WC / BMI$^{2/3}$x height$^{1/2}$). A z score can also be calculated from the ABSI mean and standard deviation (SD) within a population, dependent upon adjustment for age and sex (ABSI-ABSI$_{mean}$/ABSI$_{SC}$). Online calculators are available that calculate an ABSI value and the z score, and then provide information for comparison purposes for a person of the same sex and age, as well as an indication of individual mortality risk via quintiles (also see <https://nirkrakauer.net/sw/absi-calculator.html>that provides relative risk values for BMI and ABSI). For the purposes of this paper, the ABSI continuous score was classified into quartiles; Quartile 1 being the lowest and Quartile 4 being the highest.

A high ABSI indicates that a person's WC is more than expected, given their height and weight, corresponding to a higher concentration of body volume centrally [7].

4.5.4 Body shape measures

Height without shoes was measured to the nearest 0.5 centimetres using a wall-mounted stadiometer and weight to the nearest 0.1 kilogram in light clothing and without shoes using standard digital scales. BMI was calculated by dividing the participant's weight in kilograms by the square of their height in metres (kg/m^2). WC was measured to the nearest 0.1 centimetre using an inelastic tape maintained in a horizontal plane, with the subject standing comfortably with their weight distributed evenly on both feet. The measurement was taken at the level of the narrowest part of the waist, and the mean calculated from three measurements of the waist. A high WC was defined as being at least 102 cm for males and 88 cm for females [185].

4.5.5 Risk factors

A number of risk factors were included in this research. Participants were asked in a questionnaire if they currently smoked or if they had ever smoked regularly (at least once a day) and their responses were categorised into non-smokers, ex-smokers and current smokers. Alcohol risk was based on the amount and frequency of alcohol usually consumed, and categorised into non-drinkers and no-, low-, intermediate-, high- and very high risk drinkers [221]. Participants were also asked nine questions that comprise the physical activity component of the National Health Survey [222] and their results were calculated on the formula "e × t × i" where *e* was number of times walking, moderate and/or vigorous exercise was undertaken during the past two weeks, *t* was the average amount of time spent on each exercise session and *i* was

the intensity (walking scored at 3.5, moderate exercise scored at 5.0 or vigorous exercise scored at 7.5). Participants were classified as sedentary (score less than 100, including no exercise), or as having low (score of at least 100 but less than 1600), moderate (score of at least 1600 to 3200, or more than 3200 but less than 2 hours of vigorous exercise) or high (score of at least 3200 and 2 hours or more of vigorous exercise) levels of physical activity. This risk factor was further reduced to a dichotomous variable – sedentary or undertaking some level of physical activity [29].

Clinic attendees had their blood pressure measured using a standard, calibrated blood pressure sphygmomanometer. Two blood pressure measurements were taken five to ten minutes apart while the participant was relaxed and seated, and the average calculated. From these, a variable was derived classifying high blood pressure as at least 140/90 mmHg (systolic and/or diastolic) [223]. A fasting blood sample of approximately 10 ml was taken for a number of blood-related measures, and the results were dichotomised according to recognised cut-off values, including total blood cholesterol [224](<or ≥5.5 mmol/L), triglycerides [225] (<or ≥1.7 mmol/L), glycated haemoglobin [226] (HbA1c) (<or >7%). Study participants were also asked if they had a parental history of diabetes, heart disease and/or stroke.

4.5.6 Demographics

Demographic variables at Stage 1 included age, sex, marital status, work status, gross annual household income (before tax deducted), highest educational qualification achieved and country of birth.

4.6 Statistical analysis

The unweighted data were initially analysed using SPSS Version 20.0 (IBM, Armonk, NY) and the final analysis used Cox regression using Stata Version 13 (StataCorp, College Station, TX). Univariable analyses were undertaken on weight measures, mortality-related variables, together with demographic and health-related risk factors at baseline for the overall cohort and the analysis sample. The mean, standard deviations and p values were calculated for age, BMI and WC, together with the proportions, number and p values for all-cause-, CVD- and cancer-related mortality across ABSI quartiles; analyses are provided for overall, as well as male and female. Person years were calculated from the date of the baseline biomedical clinic appointment, and either the date of death or date of last contact for use in a Cox regression. The overall association between all-cause mortality and ABSI quartiles, BMI categories and high WC categories were examined using a Cox proportional hazards survival model to examine these measures as predictors of all-cause mortality (CVD- and cancer-related mortality and differences between males and females could not be explored due to small numbers in the lower quartiles). ABSI Quartile 1 (lowest BMI/WC), BMI<18.5 (underweight) and WC of <94 cm (males) and <80 cm (females) were the reference categories and the hazard ratio (HR), the relative risk (RR), 95% confidence intervals and p value for each is provided. The first model adjusted for age and sex; while the second model adjusted for age, sex, demographic characteristics (marital status, work status, annual gross household income, highest educational qualification achieved, country of birth) and health-related risk factors (smoking, alcohol risk, physical activity level, high blood pressure, high total blood cholesterol, high triglycerides, high glycated haemoglobin, and

parental history of disease-diabetes, heart disease and stroke). A Kaplan-Meier survival graph was used to show the differences in survival by ABSI quartiles.

4.7 Results

An overview of selected demographic, socioeconomic and health-related risk factor characteristics for study participants at baseline for the original response sample (n=4056) versus the analysis sample for the hazard ratios (n=3311) is shown in Table 4.1. There were minimal differences between the characteristics of each sample. Table 4.1 shows that within the analysis sample, 12.9% of participants had died (from all-causes), including 4.3% from cardiovascular-related causes and 3.6% from cancer-related causes.

Table 4.2 shows the mean and standard deviation for overall and then by sex for age, BMI and WC for each ABSI quartile at baseline, as well as the proportion and number of those participants with a high WC, and those who have subsequently died from all-cause, CVD-related and cancer-related mortality. P values are provided for males and females within each quartile, and across all quartiles. Males were more likely to be in Quartiles 3 and 4; females were more likely to be in Quartiles 1 and 2. Overall, mean age increased across the quartiles from 39.6 years in Quartile 1 (33.8/41.4 males/females) to 63.4 years in Quartile 4 (63.7/63.0 males/females). Mean BMI remained in the overweight range (25-29) for all ABSI quartiles overall, however WC steadily increased and the proportion of people with a high WC increased from 21.7% in Quartile 1 to 58.5% in Quartile 4, with females more likely than males to have a high WC. The proportion of all three types of mortality examined steadily increased from ABSI Quartile 1 through to Quartile 4. Those in Quartile 4 had a higher

proportion of all-cause, CVD- and cancer-related mortality than the other three quartiles combined, with a higher proportion of females than males for all mortality causes across all quartiles, except for cancer-related mortality. The mean time between the baseline biomedical clinic appointment date and date of death was 7.5 years (range 2 weeks to 14 years).

Table 4.3 shows that the association between the ABSI (quartile and continuous scores) and mortality was attenuated after adjusting for selected demographics and health-related risk factors, but was still present. Stratification by sex was not possible due to small numbers. This analyses shows that after adjusting for sociodemographic and health-related risk factors, the risk of all-cause mortality for ABSI Quartile 3 showed a strong association (HR 1.97, 95% CI 1.16-3.34, p=0.014), increasing for ABSI Quartile 4 (HR 2.90, 95% CI 1.72-4.88, p<0.001), with a moderate association for the continuous ABSI score (HR 1.32, 95% CI 1.18-1.48, p<0.001). The risk of all-cause mortality with being obese as measured by BMI and WC was lower (ORs of 1.06 and 1.26 respectively).

Table 4.1 Descriptive variables for baseline original response sample and analysis sample (unweighted)

PARTICIPANT CHARACTERISTICS	Original sample (n=4056)			Analysis sample (n=3311)			
	n	%	Mean (SD)	n	%	Mean (SD)	p
WEIGHT MEASURES							
BMI (n, mean, SD)	**4054**		*27.8 (5.5)*	3311		*27.8 (5.4)*	0.712
BMI (n, %)							0.899
Underweight <18.50	44	1.1		33	1.0		
Normal 18.50-24.99	1289	31.8		1047	31.6		
Overweight 25.00-29.99	1562	38.5		1301	39.3		
Obese 30.00+	1159	28.6		930	28.1		
WC (cm) (n, mean, SD)	**4053**		*92.5 (14.7)*	**3311**		*92.3 (14.5)*	0.600
WC (cm) (n, %)							0.753
Normal WC (M<94 cm, F<80 cm)	1427	35.2		1175	35.5		
Overweight WC (M94-101 cm, F80-87 cm)	1005	24.8		839	25.3		
Obese WC (M>=102 cm, F>=88 cm)	1621	40.0		1297	39.2		
ABSI							0.862
Quartile 1	1013	25.0		840	25.4		
Quartile 2	1013	25.0		835	25.2		
Quartile 3	1013	25.0		837	25.3		
Quartile 4	1013	25.0		799	24.1		
MORTALITY							
All-cause	581	14.3		427	12.9		0.076
CVD-related*	209	5.2		143	4.3		0.095
Cancer-related *	157	3.9		120	3.6		0.580
DEMOGRAPHICS							
Age							0.277
20 to 34 years	755	18.6		626	18.9		
35 to 54 years	1670	41.2		1414	42.7		
55 to 74 years	1275	31.4		1015	30.7		
75 years and over	356	8.8		256	7.7		
Sex							0.839
Male	1932	47.6		1585	47.9		
Female	2124	52.4		1726	52.1		
Marital status							0.338
Married/defacto	2461	60.7		2068	62.5		
Separated/divorced	579	14.3		471	14.2		
Widowed	375	9.2		270	8.2		
Never married	618	15.2		502	15.2		
Work status							0.370
Full time employed	1431	35.3		1246	37.6		
Part time/casual employed	690	17.0		582	17.6		
Unemployed	146	3.6		114	3.4		
Home duties/retired	1520	37.5		1191	36.0		
Student/other	226	5.6		178	5.4		

Table 4.1 cont'd ...

PARTICIPANT CHARACTERISTICS contd	Original sample (n=4056)			Analysis sample (n=3311)			
	n	%	Mean (SD)	n	%	Mean (SD)	p
Annual gross household Income							0.447
Up to $20,000	1193	29.4		977	29.5		
$20,001 to $40,000	1029	25.4		896	27.1		
$40,001 to $60,000	799	19.7		708	21.4		
$60,001 and over	806	19.9		730	22.0		
Highest education level							0.302
Secondary	1749	43.1		1432	43.2		
Trade/Apprentice/Certificate/ Diploma	1686	41.6		1446	43.7		
Bachelor degree or higher	473	11.7		433	13.1		
Country of birth							0.864
Australia	2777	68.5		2291	69.2		
United Kingdom/Ireland	700	17.3		584	17.6		
Europe	394	9.7		310	9.4		
Asia/other	164	4.0		126	3.8		
RISK FACTORS							
Smoking							0.782
Non-smoker	1819	45.1		1487	44.9		
Ex-smoker	1321	32.8		1108	33.5		
Current smoker	892	22.1		716	21.6		
Alcohol							0.607
Non-drinker/no risk	2152	53.5		1733	52.3		
Low risk	1648	41.0		1393	42.1		
Intermediate to very high risk	223	5.5		185	5.6		
Physical activity							0.555
Sedentary	1035	28.2		912	27.5		
Undertakes some form of exercise	2638	71.8		2399	72.5		
High blood pressure (≥140/90 mmHg)	1253	30.9		986	29.8		0.302
High total blood cholesterol (≥5.5 mmol/L)	1580	39.4		1298	39.2		0.842
High triglycerides (≥1.7 mmol/L)	1128	28.2		927	28.0		0.885
High HbA1c (>7%)	140	3.5		101	3.1		0.287
PARENTAL HISTORY OF DISEASE							
Diabetes	745	18.4		612	18.5		0.898
Heart disease	1507	37.2		1228	37.1		0.953
Stroke	798	19.7		650	19.6		0.963

Note: Not stated not shown

* Either a primary or subsequent cause of death

Table 4.2 Baseline mean, standard deviation (SD) and p value for age and weight measures; proportion, n and p value for all-cause, CVD-related and cancer-related mortality across ABSI quartiles for overall, males and females

ABSI QUARTILES	WEIGHT MEASURES								MORTALITY					
	Age (yrs)		BMI		WC (cm)		High WC*		All cause		CVD- related**		Cancer- related**	
	Mean (SD)		Mean (SD)		Mean (SD)		% (n)		% (n)		% (n)		% (n)	
OVERALL (n=4052)	*50.3*	*(16.3)*	*27.8*	*(5.5)*	*92.5*	*(14.7)*	*40.0*	*(1621)*	**14.3**	(581)	**5.2**	(209)	**3.9**	(157)
Males (n=1932)	50.6	(16.8)	27.9	(4.8)	98.4	(13.0)	36.3	(701)	17.7	(341)	6.7	(129)	5.2	(101)
Females (n=2124)	50.1	(16.1)	27.7	(6.0)	87.1	(14.1)	43.4	(920)	11.3	(240)	3.8	(80)	2.6	(56)
p (ABSI quartiles)	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	
QUARTILE 1 (n=1013)	*42.0*	*(14.2)*	*27.0*	*(5.9)*	*80.8*	*(11.8)*	*21.7*	*(220)*	**3.3**	(33)	**1.1**	(11)	**1.2**	(12)
Males (n=103)	31.1	(11.2)	25.4	(3.5)	82.5	(8.4)	1.9	(2)	-	(n<5)	-	(n<5)	-	(n<5)
Females (n=910)	43.2	(14.0)	27.2	(6.1)	80.6	(12.1)	24.0	(218)	3.3	(30)	1.1	(10)	1.2	(11)
p (males/females)	<0.001		0.002		0.126		<0.001		-		-		-	
QUARTILE 2 (n=1013)	*47.1*	*(15.4)*	*27.6*	*(5.6)*	*89.2*	*(12.0)*	*33.9*	*(343)*	**7.9**	(80)	**2.1**	(21)	**1.8**	(18)
Males (n=372)	39.6	(13.2)	27.3	(4.7)	91.5	(10.5)	15.9	(59)	5.1	(19)	-	(n<5)	-	(n<5)
Females (n=641)	51.4	(15.0)	27.7	(6.0)	87.9	(12.6)	44.3	(284)	9.5	(61)	3.0	(19)	2.5	(16)
p (males/females)	<0.001		0.228		<0.001		<0.001		0.012		0.009		0.38	
QUARTILE 3 (n=1013)	*51.9*	*(15.2)*	*28.3*	*(5.0)*	*96.7*	*(11.5)*	*45.8*	*(464)*	**14.6**	(148)	**5.0**	(51)	**3.6**	(36)
Males (n=656)	48.9	(14.4)	28.1	(4.5)	97.8	(10.6)	32.8	(215)	11.1	(73)	3.8	(25)	2.7	(18)
Females (n=357)	57.6	(15.1)	28.8	(5.8)	94.7	(12.8)	69.7	(249)	21.1	(75)	7.3	(26)	5.0	(18)
p (males/females)	<0.001		0.041		<0.001		<0.001		<0.001		0.016		0.059	
QUARTILE 4 (n=1013)	*60.4*	*(15.1)*	*28.2*	*(5.2)*	*103.0*	*(13.3)*	*58.5*	*(593)*	**31.5**	(319)	**12.3**	(125)	**9.0**	(91)
Males (n=800)	59.6	(15.1)	28.3	(5.0)	104.1	(13.0)	53.1	(425)	30.6	(245)	12.5	(100)	10.0	(80)
Females (n=213)	63.1	(14.9)	28.1	(5.9)	99.2	(13.7)	78.9	(168)	34.7	(74)	11.7	(25)	5.2	(11)
p (males/females)	0.003		0.797		<0.001		<0.001		0.250		0.764		0.028	

* Males ≥102 cm; Females ≥88 cm   ** Either a primary or subsequent cause of death

Table 4.3 Hazard ratios (95% confidence intervals) for all-cause mortality by measures of adiposity

WEIGHT MEASURES	MODEL 1			MODEL 2		
	HR	(95% CI)	p	HR	(95% CI)	p
BMI						
Underweight/Normal (<24.99) *(Reference)*	**1.00**			**1.00**		
Overweight (25.00-29.99)	**0.75**	(0.59-0.95)	0.015	**0.74**	(0.58-0.94)	0.013
Obese (≥30.00)	**1.06**	(0.82-1.36)	0.662	**0.92**	(0.70-1.20)	0.543
Continuous	*1.00*	*(0.98-1.03)*	*0.662*	*0.99*	*(0.97-1.01)*	*0.333*
WC						
Normal WC M<94 cm, F<80 cm (Reference)	**1.00**			**1.00**		
Overweight WC M94-101 cm, F80-87 cm	**0.95**	(0.72-1.25)	0.712	**0.87**	(0.66-1.15)	0.337
Obese WC M>=102 cm, F>=88 cm	**1.26**	(0.99-1.60)	0.066	**1.06**	(0.82-1.38)	0.639
Continuous	*1.01*	*(1.00-1.02)*	*0.007*	*1.00*	*(1.00-1.01)*	*0.337*
ABSI						
Quartile 1 (Reference)	**1.00**			**1.00**		
Quartile 2	**1.43**	(0.81-2.50)	0.215	**1.50**	(0.85-2.64)	0.158
Quartile 3	**1.97**	(1.16-3.34)	0.012	**1.95**	(1.15-3.33)	0.014
Quartile 4	**2.90**	(1.72-4.88)	<0.001	**2.64**	(1.56-4.47)	<0.001
Continuous	*1.41*	*(1.26-1.57)*	*<0.001*	*1.32*	*(1.18-1.48)*	*<0.001*

Model 1 - adjusted for age and sex

Model 2 - adjusted for Model 1 variables, plus demographics (marital status, work status, annual gross household income, highest educational qualification achieved, country of birth) and health-related risk factors (smoking, alcohol risk, physical activity level, high blood pressure, high total blood cholesterol, high triglycerides, high glycated haemoglobin, and parental history of disease-diabetes, heart disease and stroke)

A Kaplan-Meier estimate graph (Figure 4.1) provides the proportion of survival and years of follow-up by ABSI quartile. In particular, it shows an increasingly steep gradient for ABSI quartiles 3 and 4 at approximately 0.88 and 0.65 respectively after more than 14 years of follow-up.
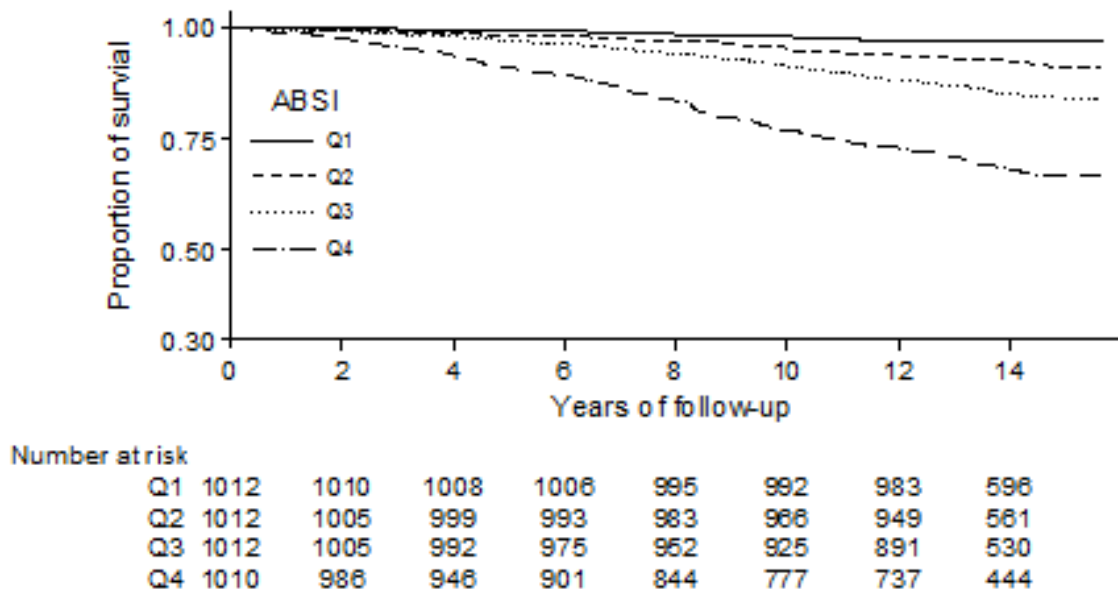
Figure 4.1 Kaplan-Meier estimate graph - ABSI Quartiles

A review of ICD10 codes for the primary cause of death across ABSI quartiles was undertaken (n=374) (Table 4.4). There was a higher proportion of deaths in Quartile 4 in the majority of cases. Supplementary information provided in Table 4.5 (Section 4.9) shows that this was particularly the case for malignant neoplasm of the colon (C18.9) and the bronchus or lung (C41.0), diabetes mellitus (E14.9), acute myocardial infarction (I24.9), chronic ischaemic heart disease (I25.9), stroke (I64) and chronic obstructive pulmonary disease (J44.9).

Table 4.4 Primary cause of death by ICD10 chapters across ABSI quartiles

ICD10 CHAPTER	ABSI QUARTILES				Total
	Q1	Q2	Q3	Q4	
Chapter I - Certain infectious and parasitic diseases (A00-B99)	1	0	2	5	8
Chapter II Neoplasms (C00-D48)	11	17	32	80	140
Chapter III Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (D50-D89)	0	0	0	2	2
Chapter IV Endocrine, nutritional and metabolic diseases (E00-E90)	0	1	3	10	14
Chapter V Mental and behavioural disorders (F00-F99)	0	0	1	5	6
Chapter VI Diseases of the nervous system (G00-G99)	1	1	1	3	6
Chapter IX Diseases of the circulatory system (I00-I99)	4	15	30	71	120
Chapter X Diseases of the respiratory system (J00-J99)	2	2	8	22	34
Chapter XI Diseases of the digestive system (K00-K93)	1	2	1	5	9
Chapter XIII Diseases of the musculoskeletal system and connective tissue (M00-M99)	0	0	0	2	2
Chapter XIV Diseases of the genitourinary system (N00-N99)	0	1	3	7	11
Chapter XVIII Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (R00-R99)	0	0	0	1	1
Chapter XX External causes of morbidity and mortality (V01-Y98)	2	4	3	12	21
TOTAL	**22**	**43**	**84**	**225**	**374**

Supplementary information has also been provided (Table 4.6, Section 4.9) regarding the primary and secondary/subsequent cause of death for males and females for each chapter (n=374).

4.8 Discussion

Our study found that those people who had the combination of the highest BMI and WC, as calculated by the ABSI, had the highest risk of premature mortality; more than two and a half times those with the lowest mortality risk. The risk of dying prematurely increased steadily across the ABSI, being one and a half times higher for those in the second quartile, to almost two times higher for those in the third quartile. Cause of death was more likely to be from CVD- and cancer-related causes, both also related to obesity.

Compared with those who had a similar BMI across the quartiles, the incorporation of the central adiposity measure found that people who had a high WC were more likely to die prematurely than those of normal WC. Females were more likely than males to have a high WC across all ABSI quartiles. Despite this, females were less likely than men to be in the highest risk quartile (Quartile 4) across all-cause and cancer-related mortality. The importance of including central adiposity in quantifying the risk was highlighted by those who developed the ABSI measure, who found that it outperformed former standard measures of abdominal obesity including WC, WHR and WHtR in predicting mortality risk. Our study also was consistent with these results regarding the greater predictability of the joint obesity measure (ABSI) compared to the single measures of obesity of BMI and WC. The developers of the ABSI found that among a British population, the risk prediction held over time (follow-up of at least 20 years) and that those whose ABSI score increased over approximately seven years subsequently had a greater mortality risk compared to those whose ABSI which had decreased [189]. In an examination of measures of body shape and their association with mortality, ABSI was also found to be the strongest predictor of all-cause mortality among an Iranian population, except for WHR in women [6,192]. Sahakyan et al [5] found that those with a normal BMI of 22 but who had central adiposity (high WHR) had a higher total mortality risk than those who had a similar BMI but no central adiposity (males HR 1.87, 95% CI 1.53 to 2.29; females HR 1.48, 95% CI 1.35-1.62). Further, these same people had higher mortality risks than those considered overweight or obesity by BMI only, particularly for males (HR 2.24, 95% CI 1.52 to 3.32; females HR 1.32, 95% CI 1.15-1.51) [5].

Our study found that males were more likely than females (32.3%) to die from CVD-related diseases. This concurred with an AusDiab study [45] that found that obese Australian males had a higher risk of myocardial infarction (HR 2.75; 95% CI 1.08-7.03) than females (HR 1.43; 95% CI 0.37-5.50). In the AusDiab study, the adverse influence of abdominal obesity was found to be associated with type 2 diabetes, dyslipidaemia, hypertension and the metabolic syndrome, with odds ratios ranging from 2 to 5, and population attributable fractions ranging from 13% to 47% with the highest proportion being for type 2 diabetes [45]. It has been reported that men who are overweight or obese at midlife were found to have an increased risk of coronary heart disease of 25% and 60% respectively [29,43,227-230]. The degree of obesity is significant in predicting mortality with a study of 1248 Spanish study participants reporting a HR of 1.94 (95% CI 1.11-3.42) for all-cause mortality for those people with a BMI of ≥35 compared to non-obese people (BMI <30) [231].

It has been hypothesised that a developing phenotype of a metabolically healthy obese (MHO) population would have a lower risk of complications associated with obesity such as metabolic syndrome and insulin sensitivity. A review found instead that MHO was significantly associated with all-cause mortality (with an increased risk of mortality of 30% from two studies), CVD mortality (with an increased risk of mortality of 14% from one study) and incident CVD (with an increased risk of mortality of 33% from three studies) [232]. The association with CVD mortality was further supported by four of six subclinical studies showing associations of MHO with CVD disease, such as increased carotid artery thickness and coronary artery calcium within the MHO population, compared to a metabolically healthy, normal weight population [232].

Our study used baseline weight measures and the length of time that the cohort had been overweight or obese at that time was not determined, however the time that a person lives with obesity has been shown to increase their mortality risk. The authors of a study of Framingham cohort participants reported that as the number of years living with obesity increased, so did their adjusted HR for mortality. Compared to those who were never obese, the HR ranged from 1.51 (95% CI 1.27-1.79) for those obese between 1 and 4.9 years to 2.52 (95% CI 2.08-3.06) for those living with obesity for 25 years or more. They found a dose-response relationship for all-cause, CVD- and cancer-related, as well as other-cause mortality, with each additional two years of obesity providing a HR of 1.06, 1.07, 1.03 and 1.07 respectively [233]. Further, it has been reported that a dynamic measure rather than a static measure of weight status was found to be more predictive of mortality [227]. Supporting evidence from Zheng et al [227] found that approximately 7% of deaths after 51 years of age through to age 77 were due to obese Class I (BMI 30-34.9) and Class II/III (BMI ≥35) upward trajectories (increasing weight gain), with increases in mortality risk of 25% and 128% respectively when compared to those who were stable overweight. It has been reported by previous studies that age weakens the association between obesity and mortality risk – that obesity is an important risk factor for mortality for those aged 40 to 65 years, but that this risk decreases for those aged 65 years and over and may indeed provide a survival advantage [228-230]. These assertions have been challenged by Masters et al who accounted for both the 2-way interactions between obesity and age at survey as well as cohort variation in mortality, and found a strengthening with age of the association between mortality risk and obesity [43]. It would be worthwhile to assess both the length of time lived with obesity and

dynamic measures of obesity in additional studies using repeated measures collected of this cohort.

For those cases where cause of death information was available, our study found the leading cause of death was cancer, and diseases of the circulatory, respiratory and endocrine systems. Similar results were found in the overall Australian population in 2015 [215]. Comparable but slightly different results can be seen in the US population in 2014: as proportions of all deaths, heart disease (males 24.5%, females 22.3%) and cancer (males 23.4%, females 22.6% of all deaths) ranked as the first and second leading causes of death respectively [234]. In our study, of the total 374 deaths, 244 (65.2%) of participants were in the ABSI Quartile 4 and another 79 (21.1%) were in Quartile 3, highlighting the association of obesity with mortality.

A major strength of our study is its use of biomedical rather than self-reported measures of obesity; the latter being shown to provide an under-estimation of weight but an over-estimation in height [235]. The inclusion of WC has been acknowledged as providing useful clinical information, particularly regarding CVD risk factors [185,236]. Android or "apple" shaped bodies have been recognised as having a stronger association with obesity-related health risks than gynoid or "pear" shaped bodies [183].

A further strength is the cohort study design which allows for investigation of important outcomes such as cause of death, as well as observations over time, pertaining to the same group of individuals. Our study was also able to provide

information on biomedically measured health-related risk factors such as blood pressure, cholesterol, triglycerides and glycated haemoglobin.

There are a number of limitations in our study including the use of arbitrary cut-off points in analyses, and responder bias due to response rates and anthropometric measurement bias during the clinic visit. Anthropometric bias has been defined as the difference between measurements taken by an expert and those taken by an observer or observers of the same subject [237]. An examination of representativeness of the cohort was undertaken following baseline recruitment. It found that there were no significant differences between those people who had participated in the NWAHS and the comparison South Australian population with regard to BMI, physical activity, current smoking status, proportions of current high blood cholesterol and high blood pressure, and overall health status [220]. While there was a decrease between the original baseline study group (n=4056) and the analysis group for our study (n=3311), and some resulting missing values, the proportions across the weight measures, mortality, demographics, risk factors and parental history of disease remained similar (see Table 4.1).

Finally, the developers of the ABSI highlighted the need for further studies to investigate whether ABSI could be used as an indicator of the effectiveness of lifestyle modification [189]. Focusing on reducing one's WC, if possible, would lead to a lowering of the ABSI score and a subsequent reduction in mortality risk. Overall weight loss to reduce risk of developing multiple morbidities is a useful and worthwhile endeavour. From a public health perspective, it is encouraging to see evidence that adopting and more importantly maintaining healthy lifestyle choices

such as not smoking, consuming a moderate amount of alcohol, undertaking regular exercising and eating the recommended levels of fruit and vegetables, can lead to a reduced HR for all-cause mortality as shown from NHANES data. Over a six-year period, those respondents who adhered to all four healthy lifestyle habits had a HR of 1.29 (95% CI 1.09-1.53) compared to 3.27 (95% CI 2.36-4.54) for those who did not undertake any of them [238].

In conclusion, ABSI is positively associated with mortality in Australian adults. Our study highlights the importance of using different measure of obesity to examine mortality risk and contributes to the growing use of ABSI as a useful predictor of mortality hazard in populations.

4.9 Supplementary Tables

Table 4.5 (Supplementary Table S1) Primary cause of death by and within ICD10 chapters across ABSI quartiles

ICD10 CHAPTER	ABSI QUARTILES				Total
	Q1	Q2	Q3	Q4	
Chapter I - Certain infectious and parasitic diseases (A00-B99)					
A047 Enterocolitis due to Clostridium difficile	0	0	0	1	1
A410 Sepsis due to Staphylococcus aureus	0	0	1	0	1
A419 Sepsis, unspecified organism	1	0	1	3	5
B909 Sequelae of respiratory and unspecified tuberculosis	0	0	0	1	1
SUB-TOTAL	**1**	**0**	**2**	**5**	**8**
Chapter II Neoplasms (C00-D48)					
C07 Malignant neoplasm of parotid gland	0	0	0	1	1
C159 Malignant neoplasm of esophagus, unspecified	0	0	2	3	5
C169 Malignant neoplasm of stomach, unspecified	0	1	1	3	5
C180 Malignant neoplasm of cecum	0	0	0	2	2
C189 Malignant neoplasm of colon, unspecified	0	1	1	5	7
C19 Malignant neoplasm of rectosigmoid junction	1	0	0	0	1
C20 Malignant neoplasm of rectum	0	0	0	1	1
C220 Liver cell carcinoma	0	0	0	2	2
C221 Intrahepatic bile duct carcinoma	0	0	0	3	3
C229 Malignant neoplasm of liver, not specified as primary or secondary	0	0	1	1	2
C259 Malignant neoplasm of pancreas, unspecified	1	2	1	0	4
C260 Malignant neoplasm of intestinal tract, part unspecified	0	0	0	2	2
C269 Malignant neoplasm of ill-defined sites within the digestive system	0	0	0	1	1
C329 Malignant neoplasm of larynx, unspecified	0	0	0	1	1
C349 Malignant neoplasm of unspecified part of bronchus or lung	2	2	5	17	26
C410 Malignant neoplasm of bone and articular cartilage of other and unspecified sites	0	0	0	1	1
C412 Malignant neoplasm of vertebral column	0	0	0	1	1
C439 Malignant melanoma of skin, unspecified	0	0	1	2	3
C459 Mesothelioma, unspecified	0	0	1	3	4
C509 Malignant neoplasm of breast of unspecified site	2	4	1	0	7
C539 Malignant neoplasm of cervix uteri, unspecified	1	0	0	0	1
C549 Malignant neoplasm of corpus uteri, unspecified	0	1	0	0	1
C56 Malignant neoplasm of ovary	0	0	1	0	1
C61 Malignant neoplasm of prostate	0	1	7	8	16
C64 Malignant neoplasm of kidney, except renal pelvis	0	1	0	0	1
C679 Malignant neoplasm of bladder, unspecified	0	0	0	3	3
C689 Malignant neoplasm of urinary organ, unspecified	0	0	0	1	1
C711 Malignant neoplasm of frontal lobe	0	0	1	0	1

Table 4.5 cont'd …

ICD10 CHAPTER contd	ABSI QUARTILES				
	Q1	**Q2**	**Q3**	**Q4**	**Total**
C719 Malignant neoplasm of brain, unspecified	2	0	1	4	7
C762 Malignant neoplasm of abdomen	0	0	1	0	1
C787 Secondary malignant neoplasm of liver and intrahepatic bile duct	0	1	0	0	1
C80 Malignant neoplasm without specification of site	1	1	2	3	7
C819 Hodgkin lymphoma, unspecified	0	0	0	1	1
C859 Non-Hodgkin lymphoma, unspecified	0	0	2	1	3
C900 Multiple myeloma	0	0	1	1	2
C911 Chronic lymphocytic leukaemia of B-cell type	0	1	0	0	1
C920 Acute myeloblastic leukemia	0	0	0	1	1
C939 Monocytic leukemia, unspecified	0	0	1	0	1
C97 Malignant neoplasms of independent (primary) multiple sites	0	1	0	3	4
D181 Lymphangioma, any site	0	0	1	0	1
D329 Benign neoplasm of meninges, unspecified	0	0	0	1	1
D361 Benign neoplasm of peripheral nerves and autonomic nervous system	0	0	0	1	1
D432 Neoplasm of uncertain behavior of brain, unspecified	0	0	0	1	1
D469 Myelodysplastic syndrome, unspecified	0	0	0	2	2
D471 Chronic myeloproliferative disease	1	0	0	0	1
SUB-TOTAL	**11**	**17**	**32**	**80**	**140**
Chapter III Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (D50-D89)					
D649 Anemia, unspecified	0	0	0	1	1
D689 Coagulation defect, unspecified	0	0	0	1	1
SUB-TOTAL	**0**	**0**	**0**	**2**	**2**
Chapter IV Endocrine, nutritional and metabolic diseases (E00-E90)					
E105 Type 1 diabetes mellitus with circulatory complications	0	0	0	1	1
E115 Type 2 diabetes mellitus with circulatory complications	0	0	0	1	1
E119 Type 2 diabetes mellitus without complications	0	0	2	0	2
E149 Unspecified diabetes mellitus without complications	0	1	1	3	5
E669 Obesity, unspecified	0	0	0	2	2
E780 Pure hypercholesterolemia	0	0	0	1	1
E875 Hyperkalemia	0	0	0	1	1
E86 Volume depletion	0	0	0	1	1
SUB-TOTAL	**0**	**1**	**3**	**10**	**14**
Chapter V Mental and behavioural disorders (F00-F99)	0	0	0	1	1
F019 Vascular dementia, unspecified	0	0	1	0	1
F03 Unspecified dementia	0	0	0	3	3
F448 Other dissociative and conversion disorders	0	0	0	1	1
F102 Mental and behavioural disorders due to use of alcohol; Dependence syndrome	0	0	0	1	1
SUB-TOTAL	**0**	**0**	**1**	**5**	**6**

Table 4.5 cont'd …

ICD10 CHAPTER contd	ABSI QUARTILES				
	Q1	Q2	Q3	Q4	Total
Chapter VI Diseases of the nervous system (G00-G99)					
G20 Parkinson's disease	1	0	0	1	2
G309 Alzheimer's disease, unspecified	0	0	1	2	3
G409 Epilepsy, unspecified	0	1	0	0	1
SUB-TOTAL	**1**	**1**	**1**	**3**	**6**
Chapter IX Diseases of the circulatory system (I00-I99)					
I10 Essential (primary) hypertension	0	0	1	0	1
I120 Hypertensive chronic kidney disease with stage 5 chronic kidney disease or end stage renal disease	0	0	0	1	1
I219 Acute myocardial infarction, unspecified	0	5	6	23	34
I229 Subsequent myocardial infarction of unspecified site	0	0	0	1	1
I249 Acute ischaemic heart disease, unspecified	0	0	0	1	1
I251 Atherosclerotic heart disease	1	3	3	8	15
I255 Ischaemic cardiomyopathy	0	0	0	3	3
I258 Other forms of chronic ischaemic heart disease	1	0	0	0	1
I259 Chronic ischaemic heart disease, unspecified	1	0	7	11	19
I272 Other secondary pulmonary hypertension	0	0	0	1	1
I340 Mitral (valve) insufficiency	0	0	0	1	1
I350 Aortic (valve) stenosis	0	0	4	0	4
I359 Aortic valve disorder, unspecified	0	0	0	1	1
I420 Dilated cardiomyopathy	0	2	1	0	3
I48 Atrial fibrillation and flutter	0	0	2	1	3
I500 Congestive heart failure	0	0	0	2	2
I509 Heart failure, unspecified	0	1	0	4	5
I615 Intracerebral haemorrhage, intraventricular	0	1	0	0	1
I619 Intracerebral haemorrhage, unspecified	0	0	0	2	2
I629 Intracranial haemorrhage (non-traumatic), unspecified	0	0	1	0	1
I635 Cerebral infarction due to unspecified occlusion or stenosis of cerebral arteries	0	0	0	1	1
I639 Cerebral infarction, unspecified	0	0	0	1	1
I64 Stroke, not specified as haemorrhage or infarction	1	2	3	3	9
I671 Cerebral aneurysm, non-ruptured	0	0	1	0	1
I679 Cerebrovascular disease, unspecified	0	1	0	1	2
I724 Aneurysm and dissection of artery of lower extremity	0	0	1	0	1
I739 Peripheral vascular disease, unspecified	0	0	0	2	2
I38 Endocarditis, valve unspecified	0	0	0	1	1
I609 Subarachnoid haemorrhage, unspecified	0	0	0	1	1
I678 Other specified cerebrovascular diseases	0	0	0	1	1
SUB-TOTAL	**4**	**15**	**30**	**71**	**120**

Table 4.5 cont'd …

ICD10 CHAPTER contd	ABSI QUARTILES				
	Q1	Q2	Q3	Q4	Total
Chapter X Diseases of the respiratory system (J00-J99)					
J110 Influenza with pneumonia, virus not identified	0	0	0	1	1
J189 Pneumonia, unspecified	1	0	0	2	3
J439 Emphysema, unspecified	0	0	2	2	4
J448 Other specified chronic obstructive pulmonary disease	0	0	0	2	2
J449 Chronic obstructive pulmonary disease, unspecified	1	1	1	8	11
J690 Pneumonitis due to food and vomit	0	0	2	1	3
J841 Other interstitial pulmonary diseases with fibrosis	0	0	1	2	3
J849 Interstitial pulmonary disease, unspecified	0	1	2	2	5
J939 Pneumothorax, unspecified	0	0	0	1	1
J984 Other disorders of lung	0	0	0	1	1
SUB-TOTAL	**2**	**2**	**8**	**22**	**34**
Chapter XI Diseases of the digestive system (K00-K93)					
K559 Vascular disorder of intestine, unspecified	1	0	1	0	2
K650 Acute peritonitis	0	1	0	0	1
K729 Hepatic failure, unspecified	0	0	0	1	1
K746 Other and unspecified cirrhosis of liver	0	0	0	2	2
K859 Acute pancreatitis, unspecified	0	1	0	0	1
K250 Gastric ulcer; Acute with haemorrhage	0	0	0	1	1
K460 Unspecified abdominal hernia with obstruction, without gangrene	0	0	0	1	1
SUB-TOTAL	**1**	**2**	**1**	**5**	**9**
Chapter XIII Diseases of the musculoskeletal system and connective tissue (M00-M99)					
M313 Wegener granulomatosis	0	0	0	1	1
M353 Polymyalgia rheumatica	0	0	0	1	1
SUB-TOTAL	**0**	**0**	**0**	**2**	**2**
Chapter XIV Diseases of the genitourinary system (N00-N99)					
N179 Acute renal failure, unspecified	0	0	2	3	5
N180 Chronic kidney disease	0	0	0	1	1
N189 Chronic kidney disease, unspecified	0	0	1	1	2
N19 Unspecified kidney failure	0	1	0	1	2
N390 Urinary tract infection, site not specified	0	0	0	1	1
SUB-TOTAL	**0**	**1**	**3**	**7**	**11**
Chapter XVIII Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (R00-R99)					
R99 Other ill-defined and unspecified causes of mortality	0	0	0	1	**1**
SUB-TOTAL	**0**	**0**	**0**	**1**	**1**

Table 4.5 cont'd …

ICD10 CHAPTER contd	ABSI QUARTILES				Total
	Q1	Q2	Q3	Q4	
Chapter XX External causes of morbidity and mortality (V01-Y98)					
V031 Pedestrian injured in collision with car, pick-up truck or van; Traffic accident	0	0	1	0	1
V436 Car occupant injured in collision with car, pick-up truck or van; Passenger injured in traffic accident	1	1	0	0	2
V446 Car occupant injured in collision with heavy transport vehicle or bus; Passenger injured in traffic accident	0	1	0	0	1
V475 Car occupant injured in collision with fixed or stationary object; Driver injured in traffic accident	0	0	0	1	1
V476 Car occupant injured in collision with fixed or stationary object; Passenger injured in traffic accident	0	1	0	0	1
V685 Occupant of heavy transport vehicle injured in non-collision transport accident; Driver injured in traffic accident	0	0	1	0	1
W19 Unspecified fall	0	0	0	1	1
W199 Unspecified fall	0	0	0	2	2
X599 Exposure to unspecified factor causing other and unspecified injury	0	0	0	1	1
X640 Intentional self-poisoning by and exposure to other and unspecified drugs, medicaments and biological substances	1	0	0	0	1
X670 Intentional self-poisoning by and exposure to other gases and vapours	0	0	0	1	1
X70 Intentional self-harm by hanging, strangulation and suffocation	0	1	0	1	2
Y442 Agents primarily affecting blood constituents - Anticoagulants	0	0	0	1	1
V204 Motorcycle rider injured in collision with pedestrian or animal; Driver injured in traffic accident	0	0	1	0	1
W014 Fall on same level from slipping, tripping and stumbling	0	0	0	1	1
W190 Unspecified fall	0	0	0	1	1
W011 Fall on same level from slipping, tripping and stumbling; Residential institution	0	0	0	1	1
Y260 Exposure to smoke, fire and flames, undetermined intent; Home	0	0	0	1	1
SUB-TOTAL	**2**	**4**	**3**	**12**	**21**
TOTAL	**22**	**43**	**84**	**225**	**374**

Table 4.6 (Supplementary Table S2) Primary and secondary/subsequent causes of death ICD10 chapters for males and females

ICD10 MAJOR CHAPTER	PRIMARY CAUSE OF DEATH				SECONDARY/SUBSEQUENT CAUSE OF DEATH*			
	Male		Female		Male (n=232)		Female (n=142)	
	n	%	n	%	n	%	n	%
Neoplasms (C00-D49)	90	38.8	50	35.9	23	9.9	15	10.6
Diseases of the circulatory system (I00-I99)	74	32.3	46	32.4	92	39.7	57	40.1
Diseases of the respiratory system (J00-J99)	18	7.8	16	11.3	62	26.7	18	12.7
External causes of morbidity (V00-Y99)	12	4.7	9	4.9	56	24.1	32	22.5
Endocrine, nutritional and metabolic diseases (E00-E89)	10	4.3	4	2.8	20	8.6	10	7.0
Diseases of the genitourinary system (N00-N99)	4	1.7	7	4.9	35	15.1	11	7.7
Diseases of the digestive system (K00-K95)	5	2.2	4	2.8	10	4.3	8	5.6
Certain infectious and parasitic diseases (A00-B99)	5	2.2	3	2.1	11	4.7	9	6.3
Diseases of the nervous system (G00-G99)	5	2.2	1	0.7	9	3.9	5	3.5
Mental, behavioural and neurodevelopmental disorders (F01-F99)	4	1.7	2	2.1	10	4.3	8	5.6
Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism (D50-D89)	2	0.9	0	0.0	4	1.7	3	2.1
Diseases of the musculoskeletal system and connective tissue (M00-M99)	2	0.9	0	0.0	2	0.9	7	4.9
Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified (R00-R99)	1	0.4	0	0.0	16	6.9	5	3.5
Injury, poisoning and certain other consequences of external causes (S00-T88)	-	-	-	-	14	6.0	7	4.9
Diseases of the skin and subcutaneous tissue (L00-L99)	-	-	-	-	2	0.9	2	1.4
Diseases of the ear and mastoid process (H60-H95)	-	-	-	-	1	0.4	-	-
Total	**232**	**100.0**	**142**	**100.0**	-	-	-	-

Chapter 5 Parental body shape at midlife and multiple adult offspring obesity measures (Publication)

5.1 Statement of Authorship

Title of Paper	Parental midlife body shape and association with multiple adult offspring obesity measures: NWAHS
Publication Status	☑ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmited work written in manuscript style
Publication Details	PLoS ONE. 2015;10(9):e0137534 https://doi.org/10.1371/journal.pone.0181244

Name of Principal Author (Candidate)	Janet F Grant
Contribution to the Paper	Participated in the design and co-ordination of the study, performed statistical analyses, and drafted and revised the manuscript.
Overall percentage (%)	85%
Certification	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date 7/9/17

Name of Co-Author	Catherine R Chittleborough
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

Name of Co-Author	Anne W Taylor
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

5.2 Chapter 5 Contextual Statement

When considering factors that may contribute to excessive weight gain, it is important to examine the genetic and environment influences involved. Obese parents provide both of these through the transition of DNA at the time of conception which affect metabolism, appetite and acceptance of physical activity, as well as household level factors such as the type and amount of food purchased and amount of physical activity. There is compelling evidence that parental weight is a strong determinant of offspring weight, particularly from mothers for their daughters.

The aim of this study was to answer the second research question, *"Is there an association between parental body shape in mid-life and adult offspring BMI and central adiposity?"*. This study concluded that if a person had two obese parents, it resulted in an increased likelihood of them also being overweight or obese and that this association tended to be stronger for daughters than sons across BMI, WC and WHtR. It recommended the use of pictograms of parental body shape as screening tools for use in primary care settings to start conversations between doctors and their patients about the health implications of having one or more obese parents.

The following chapter presents the results of an exploration of parental midlife body shape and its association with multiple adult offspring obesity measures. This manuscript was published by PLOS One on 10 September 2015, and has been reformatted to meet the requirements of this thesis. The manuscript in its published format is included in this thesis as Appendix 2.

5.3 Abstract

There is compelling evidence that parental weight is a strong determinant of offspring weight status. The study used cross-sectional self-reported and measured data from a longitudinal cohort of Australian adults (n=2128) from Stage 3 (2008-10) of the NWAHS (1999-2003, baseline n=4056) to investigate the association between midlife parental body shape and four indicators of obesity and fat distribution. The analysis used measured BMI, WC, WHR and WHtR of adult offspring, together with pictograms for recall of parental body shape.

Compared to both parents being a healthy weight, offspring were more likely to be overweight or obese if both parents were an unhealthy weight at age 40 (OR 2.14, 95% CI 1.67-2.76) and further, those participants whose mother was an unhealthy weight were more likely to be overweight or obese themselves (OR 1.50, 95% CI 1.14-1.98). There were similar but lower results for those with an overweight/obese father (OR 1.44, 95% CI 1.08-1.93). The effect of one or both parents being overweight or obese tended to be stronger for daughters than for sons across BMI, WC and WHtR. BMI showed the strongest association with parental body shape (OR 2.14), followed by WC (OR 1.78), WHtR (OR 1.71) and WHR (OR 1.45). WHtR (42-45%) and BMI (35-36%) provided the highest positive predictive values for overweight/obesity from parental body shape.

Parental obesity increases the risk of obesity for adult offspring, both for overall body shape and central adiposity, particularly for daughters. Pictograms could potentially be used as a screening tool in primary care settings to promote healthy weight among young adults.

5.4 Introduction

Research suggests that the location of excess body fat within individuals is associated with morbidity and mortality [239]. Furthermore, cardiometabolic complications are more likely to occur when visceral fat storage is present in excess [240]. Obesity is the most recent major global epidemic, rarely appearing as a health issue before the 20th century but doubling in prevalence since 1980 [1]: it is also a major problem in Australia with 35.3% of the population being overweight and 27.5% being obese in 2011-12 [241].

Accurate assessment of body fat distribution on a large-scale population basis can be problematic due to increased costs and portability of valid medical technologies. Population-level proxy measures can therefore be used to determine health risk through the categorisation of obesity [180] by indices such as BMI and central adiposity measures including WC, WHR [242] and WHtR [188]. Existing literature propone pictograms, representing body size and shape, as a valid approach to estimating personal BMI [9,200] and recalling parental weight [101].

There is compelling evidence that parental weight is a strong determinant of offspring weight status [56,60,100]. A 2012 study of three generations examined the relative maternal and paternal associations and reported an enduring association between mother and offspring BMI [105]. Recent research has explored the relative influence of both maternal and paternal factors such as parental smoking, poor diet, low rates of physical activity and lower social class, together with mother's older age and weight gain during pregnancy, may negatively impact on offspring health [13,56,243]. Findings from another recent study support the conclusion that

maternal BMI has a significantly stronger influence on adult female offspring BMI despite the fact that both parents' BMI influence adult male offspring BMI equally [110].

Currently, available data relating to the association between parental body shape and adult offspring weight status predominantly use BMI. Fewer studies incorporate measures of central adiposity.

This study aimed to assess if there was an association between midlife parental body shape and four measures of obesity and fat distribution among Australian adults. Combining an indication of parental body shape as a screening device, together with a person's current body shape measure, may be useful in primary care to assist in the early identification of those who may be at an increased risk of developing obesity and related co-morbidities, for targeting purposes for regular monitoring, intervention and treatment.

5.5 Methods

5.5.1 Sample

The NWAHS is a representative longitudinal study of 4056 randomly selected adults aged 18 years and over, recruited from 1999 to 2003 from the north-west region of Adelaide, the capital of South Australia. Participants were recruited using the Electronic White Pages and during the initial CATI, the eligible adult who had the most recent birthday in the household was invited to participate. People were excluded if they did not have the capacity to participate due to illness or intellectual limitations, if they were unable to communicate in English or if they lived in a

residential institution. The study methodology has previously been described in detail [206,244]. Written informed consent was gained from study participants. Ethical approval for this research was granted by the Human Research Ethics Committee of The University of Adelaide.

NWAHS participants have been followed up several times since initial recruitment. Measured anthropometric data used in this paper are from Stage 1 (baseline 1999-2003, response rate 49.1%) and Stage 3 (second follow-up 2008-2010, overall n=2871 (questionnaire n=2483, clinic n=2487), response rate 76.0%). Self-reported information was also collected by CATI and self-completed questionnaire at both stages, as well as via a telephone follow-up (TFU) survey in 2007 (TFU2, n=2996, response rate 90.2%).

Participants who attended all three major stages of the study and who provided information about their parents' occupation and country of birth in TFU2, as well as their parents' body shape in the Stage 3 questionnaire, were included in the study. This reduced the overall sample from 4056 to 2128, after excluding those without biomedical information at each major stage or related information about at least one of their parents. There were 176 participants who provided information on only parent (mother only n=119; father only n=57), resulting in a multinomial regression analysis sample of 1952 who provided body shape information on both parents.

5.5.2   Offspring body shape

Four anthropometric measures of adult offspring were undertaken. Height without shoes was measured to the nearest 0.5 centimetres using a wall-mounted

stadiometer (height measurement), and weight to the nearest 0.1 kilogram in light clothing and without shoes using standard digital scales. BMI was calculated by dividing the participant's weight in kilograms by the square of their height in metres (kg/m^2). BMI values were initially grouped according to the WHO BMI classifications [180] and then reduced to three categories for analysis: underweight/healthy weight (BMI <25), overweight (BMI 25-29) and obese (BMI ≥30).

WC was measured to the nearest 0.1 centimetre using an inelastic tape maintained in a horizontal plane, with the subject standing comfortably with weight distributed evenly on both feet. The measurement was taken at the level of the narrowest part of the waist. HC was also measured using an inelastic tape, at the level of the maximum posterior extension of the buttocks. Three measurements of the waist and hip were taken and the mean for each was calculated. The cut-off points for recommended weight reduction to reduce major cardiovascular risk factors using WC were ≥102 cm for men and ≥88 cm for women [186], and a WHR of >1.0 for men and >0.85 for women [210]. The cut-off points for WHtR for a reduction in cardiometabolic outcomes was 0.5 [212].

After their clinic examination, participants were provided with selected results with an indication of where these results were outside desirable levels (including BMI <18.5 or >24.9, blood pressure >140/90mmHg, total cholesterol >7.0mmol/L, glucose >7.0mmol/L and lung function >80% predicted for age and sex of forced expiratory volume in one second (FEV1)), while their general practitioner was provided with all results, including blood and urine pathology, blood pressure and lung function, BMI and WHR.

5.5.3 Parental body shape

Parental body shape was asked in the Stage 3 self-completed questionnaire, and operationalised through the use of a set of nine figures from a set of validated pictograms. The pictograms ask respondents to identify the body type of their biological mother and father at age 40 (Figure 5.1). For analysis purposes, the set of silhouettes were each derived into a dichotomous variable for mothers and fathers: silhouettes 1 through 5 were classified as unhealthy weight, and silhouettes 6 through 9 were classified as healthy weight/underweight [204].
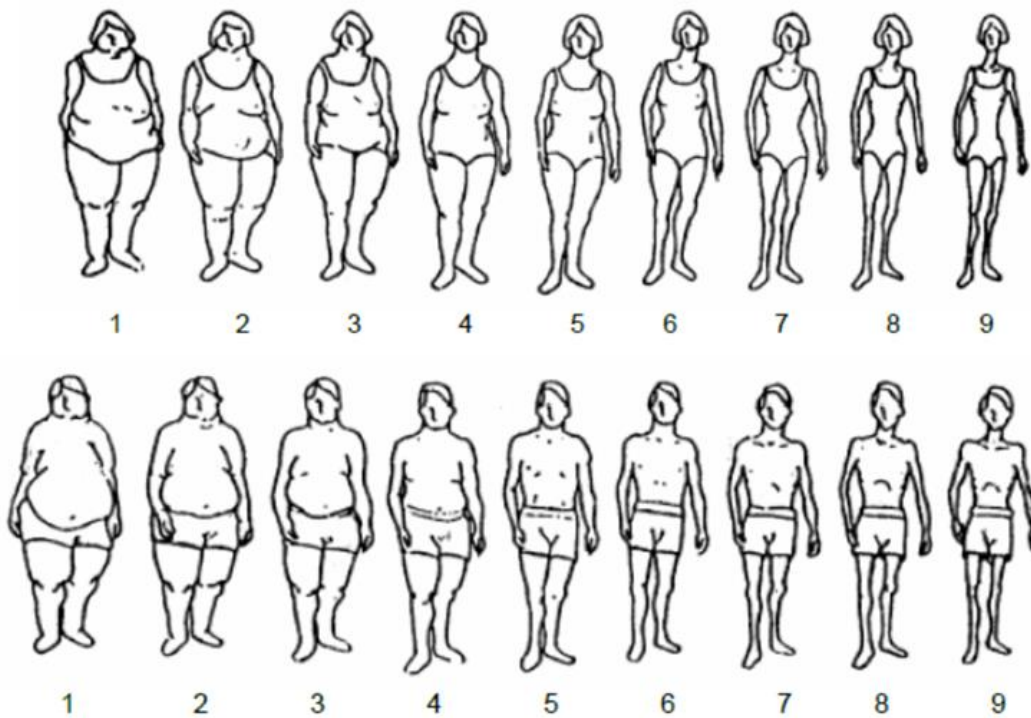
Figure 5.1 Images for perceived body shape of parents from the Figure Rating Scale

Legend: Silhouettes 1-2 = Very overweight; Silhouettes 3-4 = Moderately overweight; Silhouette 5 = Slightly overweight; Silhouettes 6-7 = Appropriate (healthy) weight; Silhouettes 8-9 = Underweight. Reprinted with permission.

Source: Adaption - Sorensen et al [9]), for use in the NWAHS

5.5.4 Demographics

Demographic variables at both Stage 1 (baseline) and Stage 3 included marital status, work status, highest level of education achieved and gross annual household income. Household tenure was asked only in Stage 3. Country of birth was asked at baseline for participants and in TFU2 for their parents. Occupation data regarding participants and their parents was asked in TFU2 and was coded into eight major groups based on the Australian and New Zealand Standard Classification of Occupations [245].

5.5.5 Statistical analysis

The data were analysed using SPSS Version 20.0 (IBM, Armonk, NY). Univariable analyses using chi-square tests compared demographic and body shape proportions of daughters and sons at baseline and at follow-up, as well as the reported body shape of their parents at midlife at follow-up. Baseline anthropometric measures were used in the univariate analyses to reduce the effect of possible bias from participation in a longitudinal study and action from feedback of clinical information.

Parental body shape via pictograms was used in the absence of measurements. The silhouettes were further classified into four categories for use as the independent variable: both parents healthy weight, only father unhealthy weight, only mother unhealthy weight and both parents unhealthy weight. Statistical analysis regarding the association of offspring body shape with parental body shape was cross-sectional. Using both parents having a healthy weight as the reference category, unadjusted odds ratios (OR), together with proportions, 95% confidence intervals and p values, were calculated across the four weight measures on those participants who had

provided information about parental body shape for both parents (n=1952).

Sensitivity (true positives), specificity (true negatives), and positive and negative

predictive values of parental body shape forecasting offspring obesity were calculated

for those adult offspring who had a measured BMI <25, using dichotomous variables

for both the recognised cut-offs of each weight measure and the pictogram

silhouettes.

5.6 Results

5.6.1 Demographics

Table 5.1 provides an overview of selected demographic and life-course variables for

participants and their parents from baseline and/or follow-up (Stage 3 or TFU2

where indicated). A comparison of selected demographic variables between baseline

and the analysis sample is show in Table 5.5 (S3).

5.6.2 Body shape of offspring and parents

Table 5.2 examines the proportion of female and male offspring participants within

each category of four measures of body shape at baseline and second follow-up, with

parental weight status.

Table 5.1 Socio-demographics for study participants for baseline and follow-up

SOCIO-DEMOGRAPHICS (self-reported)	DAUGHTERS				SONS			
	Baseline		Follow-Up		Baseline		Follow-Up	
	n	%	n	%	n	%	n	%
Age								
18 to 29 years	84	7.5	17	1.5	87	8.7	24	2.4
30 to 39 year	183	16.3	91	8.1	164	16.4	86	8.6
40 to 49 years	300	26.6	223	19.8	239	23.9	199	19.9
50 to 59 years	275	24.4	294	26.1	249	24.9	230	23.0
60 to 69 years	186	16.5	258	22.9	165	16.5	249	24.9
70 years and over	98	8.7	243	21.6	98	9.8	214	21.4
Marital status								
Married/defacto	737	65.5	719	63.8	685	68.4	714	71.3
Separated/divorced	158	14.0	156	13.9	135	13.5	123	12.3
Widowed	116	10.3	157	13.9	40	4.0	59	5.9
Never married	110	9.8	80	7.1	140	14.0	85	8.5
Not stated	5	0.4	14	1.3	2	0.2	21	2.1
Work status								
Full time employed	294	26.1	297	26.4	599	59.8	523	52.2
Part time / casual employment	307	27.3	265	23.5	83	8.3	74	7.4
Unemployed	24	2.1	18	1.6	29	2.9	16	1.6
Home duties	264	23.4	67	6.0	8	0.8	3	0.3
Retired	180	16.0	393	34.9	228	22.8	326	32.5
Student	18	1.6	6	0.5	22	2.2	4	0.4
Other	25	2.2	65	5.8	29	2.9	34	3.4
Not stated	14	1.2	15	1.3	4	0.4	22	2.2
Highest educational qualification								
Up to & including secondary	677	60.1	624	55.4	358	35.7	307	30.6
Trade/Apprenticeship/Certificate/ Diploma	276	24.5	250	22.2	503	50.2	467	46.6
Bachelor degree or higher	160	14.2	239	21.2	134	13.4	207	20.7
Other/Don't know/Not stated	13	1.2	13	1.2	7	0.7	21	2.1
Income (gross annual household)								
Up to $12,000	147	13.1	30	2.7	58	5.8	16	1.6
$12,001 - $20,000	174	15.5	157	13.9	104	10.4	103	10.3
$20,001 to $40,000	263	23.4	280	24.9	295	29.4	213	21.3
$40,001 to $60,000	251	22.3	157	13.9	252	25.1	178	17.8
$60,001 to $80,000	131	11.6	133	11.8	120	12.0	141	14.1
More than $80,000	111	9.9	251	22.3	141	14.1	290	28.9
Not stated	49	4.4	118	10.5	32	3.2	61	6.1
Country of birth								
Australia	785	69.7			701	70.0		
United Kingdom/Ireland	217	19.3			172	17.2		
Europe	86	7.6			95	9.5		
Asia/ Oceania/ Americas/ Africa	31	2.8			33	3.3		
Other/Not stated	7	0.6			1	0.1		

Table 5.1 cont'd …

SOCIO-DEMOGRAPHICS	DAUGHTERS				SONS			
(self-reported) contd	Baseline		Follow-Up		Baseline		Follow-Up	
	n	%	n	%	n	%	n	%
*Occupation								
Manager	27	2.4			67	6.7		
Professional	190	16.9			144	14.4		
Technician or trade worker	58	5.2			289	28.8		
Community or personal service worker	90	8.0			37	3.7		
Clerical or admin worker	320	28.4			145	14.5		
Sales worker	122	10.8			67	6.7		
Machinery operator or driver	28	2.5			87	8.7		
Labourer	98	8.7			132	13.2		
Unable to classify/economically inactive/NS	193	17.1			34	3.4		
Housing tenure								
Owned/being purchased by the occupants			959	85.2			858	85.6
Renting/board			127	11.3			98	9.8
Retirement unit, nursing home, life tenure			24	2.1			21	2.1
Other/Not stated			16	1.4			25	2.5
*Mother's country of birth								
Australia			667	59.2			569	56.8
United Kingdom/Ireland			263	23.4			225	22.5
Europe			164	14.6			169	16.9
Asia/Oceania/Americas/Africa			32	2.8			38	3.8
Not stated			-	-			1	0.1
*Father's country of birth								
Australia			612	54.4			550	54.9
United Kingdom/Ireland			273	24.2			221	22.1
Europe			188	16.7			187	18.7
Asia/Oceania/Americas/Africa			42	3.7			34	3.4
Not stated			11	1.0			10	1.0
*Mother's occupation								
Manager			57	5.1			34	3.4
Professional			81	7.2			65	6.5
Technician or trade worker			71	6.3			49	4.9
Community or personal service worker			56	5.0			52	5.2
Clerical or admin worker			89	7.9			60	6.0
Sales worker			67	6.0			59	5.9
Machinery operator or driver			2	0.2			5	0.5
Labourer			138	12.3			108	10.8
Unable to classify/economically inactive/NS			565	50.2			570	56.9
*Father's occupation								
Manager			166	14.7			132	13.2
Professional			108	9.6			105	10.5
Technician or trade worker			253	22.5			245	24.5
Community or personal service worker			53	4.7			41	4.1
Clerical or admin worker			85	7.5			78	7.8
Sales worker			56	5.0			62	6.2
Machinery operator or driver			105	9.3			72	7.2
Labourer			270	24.0			247	24.7
Unable to classify/economically inactive/NS			30	2.7			20	2.0
TOTAL	**1126**	**100.0**	**1126**	**100.0**	**1002**	**100.0**	**1002**	**100.0**

Asked in the Telephone Follow-Up survey, 2007

Table 5.2 Body shape of study participants for baseline and second follow-up, and body shape of the parent(s)

WEIGHT MEASURES	DAUGHTERS				SONS			
	Baseline		Follow-Up		Baseline		Follow-Up	
	n	%	n	%	n	%	n	%
OFFSPRING (measured)								
BMI								
Underweight/Healthy weight (<25)	429	38.1	349	31.0	241	24.1	191	19.1
Overweight (25-29)	367	32.6	387	34.4	493	49.2	477	47.6
Obese (≥30)	330	29.3	390	34.6	268	26.7	333	33.2
Central adiposity								
Android obesity (WHR>1.0 males; >0.85 females)	284	25.2	430	38.2	110	11.0	246	24.6
High WC (≥102 cm males; ≥88 cm females)	469	41.7	571	50.7	367	36.6	460	45.9
High WHtR (≥0.5)	686	60.9	777	69.0	824	82.2	853	85.1
PARENTS' BODY SHAPE AT MID-LIFE (pictograms)								
Mother								
Underweight/Healthy weight			444	39.4			406	40.5
Overweight			614	54.5			537	53.6
Obese			41	3.6			29	2.9
Not stated			27	2.4			30	3.0
Father								
Underweight/Healthy weight			492	43.7			419	41.8
Overweight			535	47.5			521	52.0
Obese			26	2.3			16	1.6
Not stated			73	6.5			46	4.6
TOTAL	**1126**	**100.0**	**1126**	**100.0**	**1002**	**100.0**	**1002**	**100.0**

Overall, using WHO BMI classifications at baseline (unweighted data), 0.8% (n=17) of the 2128 participants were underweight (BMI <20); 30.7% (n=653) were normal weight (BMI 20-24); 40.4% (n=860) were overweight (BMI 25-29); and 28.1% (n=598) were obese. Of those who were obese, 65.9% (n=394) were in Obese Class I (BMI 30-34), 25.6% (n=153) were in Obese Class II (BMI 35.00 to 39.99) and 8.5% (n=51) were in Obese Class III (BMI ≥40), with daughters more likely than sons to be in the latter (heavier) two obese classes (not shown).

Regarding central adiposity overall, 18.5% of participants had a high WHR (men >1.0; women >0.85); 39.3% had a high WC (men ≥102 cm; women ≥88 cm) and 71.0% had a high WHtR (≥0.5). There were also 798 participants (37.5%) who lost weight (mean 4.7 kg, 95% CI 4.36-5.10%) during the same timeframe. Of those, more daughters lost slightly more weight (n=421, mean 5.2 kg, 95% CI 4.61-5.71, range 0.05 to 46.0 kg) than sons (n=377, mean 4.3 kg, 95% CI 3.76-4.74, range 0.05 to 41.2 kg).

The BMI of study participants increased from a mean of 27.80 (SD 5.21) at Stage 1 over approximately seven years to 28.66 (SD 5.48) at Stage 3, with a corresponding increase in the mean waist circumference from 92.23 cm (SD 14.31) to 95.0 cm (SD 14.97) (not shown). Overall, 1322 participants (62.1%) gained weight (mean 6.0 kg, 95% CI 5.67-6.30) between Stage 1 and Stage 3. Of those, daughters gained slightly more weight (n=699, mean 6.1 kg, 95% CI 5.73-6.55, range 0.05 to 34.0 kg) than sons (n=623, mean 5.8 kg, 95% CI 5.34-6.29, range 0.05 to 60.7 kg). Those participants who gained weight were more likely to be younger (aged 18 to 49 years) and male.

There were also 798 participants (37.5%) who lost weight (mean 4.7 kg, 95% CI 4.36-5.10) during the same timeframe. Of those, daughters lost slightly more weight (n=421, mean 5.2 kg, 95% CI 4.61-5.71, range 0.05 to 46.0 kg) than sons (n=377, mean 4.3 kg, 95% CI 3.76-4.74, range 0.05 to 41.2 kg). There were no differences between daughters and sons regarding their responses to the question about their parents' body shape at midlife.

Table 5.3 provides a comparison of the four measures of obesity and central

adiposity, with four combinations of parental overall body shape, as well as for

daughters and sons. Regardless of which body shape measure was used, there was

strong evidence that offspring were more likely to be overweight or obese if both

parents were an unhealthy weight at age 40 when compared to those whose parents

were a healthy weight. For example, using BMI and the reference category as both

parents being a healthy weight, the overall OR when both parents have an unhealthy

weight was 2.14 (95% CI 1.67-2.76). There was moderate evidence that an unhealthy

maternal body shape influenced their offspring's adult body shape when compared to

both parents being a healthy weight (OR 1.50, 95% CI 1.14-1.98), with a slightly lower

result for unhealthy paternal body shape (OR 1.44, 95% CI 1.08-1.93). The effect of

one or both parents being overweight or obese tended to be stronger for daughters

than for sons regardless of whether one or both parents were an unhealthy weight for

BMI, WC and WHtR (eg BMI daughters/sons - OR both parents 2.36, 1.92; mother

only 1.87, 1.17; father only 1.54; 1.28 respectively). BMI showed the strongest

association with parental body shape (OR 2.14), followed by WC (OR 1.78), WHtR (OR

1.71) and WHR (OR 1.45).

Table 5.3 Unadjusted odds ratios (proportions, 95% confidence intervals and p values) for overweight/obese offspring measures of parental body shape/weight

Overweight/obese (Stage 1)* (measured)	Both parents healthy weight Overall n=453 *Daughters n=242* *Sons n=211* Ref 1.0 n (%)	Father UNHEALTHY weight Overall n=353 *Daughters n=176* *Sons n=177* n (%)	OR	95% CI	p	Mother UNHEALTHY weight Overall n=431 *Daughters n=239* *Sons n=192* n (%)	OR	95% CI	p	Both parents UNHEALTHY weight Overall n=715 *Daughters n=369* *Sons n=346* n (%)	OR	95% CI	p
BMI overall	264 (58.3%)	236 (66.9%)	1.44	(1.08 - 1.93)	0.013	292 (67.7%)	1.50	(1.14 - 1.98)	0.004	536 (75.0%)	2.14	(1.67 - 2.76)	<0.001
Daughters	117 (48.3%)	104 (59.1%)	1.54	(1.04 - 2.28)	0.030	152 (63.6%)	1.87	(1.30 - 2.69)	0.001	254 (68.8%)	2.36	(1.69 - 3.30)	<0.001
Sons	147 (69.7%)	132 (74.6%)	1.28	(0.82 - 2.00)	0.284	140 (72.9%)	1.17	(0.76 - 1.81)	0.472	282 (81.5%)	1.92	(1.29 - 2.86)	0.001
WC overall	140 (30.9%)	127 (36.0%)	1.26	(0.94 - 1.69)	0.129	176 (40.8%)	1.54	(1.17 - 2.04)	0.002	317 (44.3%)	1.78	(1.39 - 2.28)	<0.001
Daughters	74 (30.6%)	69 (39.2%)	1.46	(0.97 - 2.20)	0.067	99 (41.4%)	1.61	(1.10 - 2.34)	0.013	175 (47.4%)	2.05	(1.46 - 2.88)	<0.001
Sons	66 (31.3%)	58 (32.9%)	1.07	(0.70 - 1.64)	0.754	77 (40.1%)	1.47	(0.98 - 2.22)	0.065	142 (41.0%)	1.53	(1.07 - 2.19)	0.021
WHtR overall	292 (64.5%)	236 (66.9%)	1.11	(0.83 - 1.49)	0.478	303 (70.3%)	1.31	(0.98 - 1.73)	0.064	541 (75.7%)	1.71	(1.33 - 2.22)	<0.001
Daughters	126 (52.1%)	104 (59.1%)	1.33	(0.90 - 1.97)	0.154	140 (58.6%)	1.30	(0.91 - 1.97)	0.151	245 (66.4%)	1.82	(1.31 - 2.54)	<0.001
Sons	166 (78.7%)	132 (74.6%)	0.80	(0.50 - 1.28)	0.341	163 (84.9%)	1.52	(0.91 - 1.28)	0.109	296 (85.5%)	1.60	(1.03 - 2.51)	0.037
WHR overall	69 (15.2%)	54 (15.3%)	1.01	(0.68 - 1.48)	0.979	84 (19.5%)	1.35	(0.95 - 1.91)	0.095	148 (20.7%)	1.45	(1.06 - 1.99)	0.020
Daughters	50 (20.7%)	44 (25.0%)	1.28	(0.81 - 2.03)	0.295	60 (25.1%)	1.29	(0.84 - 1.97)	0.247	101 (27.4%)	1.45	(0.98 - 2.13)	0.061
Sons	19 (9.0%)	10 (5.6%)	0.61	(0.27 - 1.34)	0.215	24 (12.5%)	1.44	(0.76 - 2.73)	0.258	47 (13.6%)	1.59	(0.90 - 2.79)	0.107

Note: n=1952 (176 participants provided parental body shape about only one parent)

* Defined as: BMI >25; high WHR (1.00 males, 0.85 females); high WC (≥102 cm males, ≥88 cm females); high WHtR >0.05)

Table 5.4 shows the sensitivity, specificity, positive and negative predictive values of parental body shape predicting offspring obesity, for those participants who were underweight or normal weight as measured by BMI at baseline (n=670; male 241, female 429), using four measures of weight status at Stage 3. The highest positive predictive values (PPV) were for both WHtR (overall mothers-fathers 41.8-45.1%; daughters 35.4-36.0%; sons 51.9-62.0% respectively) and BMI (overall mothers-fathers 35.4-36.4%; daughters 31.1-33.5; sons 41.9-42.3% respectively). Sensitivity of parental overweight/obesity in pictograms in predicting overweight/obesity in offspring ranged from 45.2% to 61.3% across all four offspring body shape measures.

5.7 Discussion

This study found that having two obese parents resulted in an increased likelihood of their adult offspring also being overweight or obese. This association tended to be stronger for daughters than sons across BMI, WC and WHtR. Compared to offspring who had both healthy weight parents, those with one parent or both parents who had an unhealthy weight had an increased odds of obesity based on BMI ranging from 44% to 114%. These results were slightly lower based on WC (26 to 78%), WHtR (11 to 71%) and WHR (1 to 45%).

These results support previous findings [56,246] from predominantly Western societies suggesting that adults with one obese parent during their childhood are more likely to also be obese, with a stronger association if both parents are obese. Overall, when compared with adults who had healthy weight parents, one study observed that adult offspring with obese parents were up to four times more likely to be obese themselves [115].

Table 5.4 Sensitivity, specificity, and positive and negative predictive values of weight measures based on parental overweight/obesity status for previously underweight or normal weight adult offspring

Weight measures above cut-off by gender and parental weight status		Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
WHtR					
Daughters	Mothers	63.3%	46.0%	64.1%	45.0%
	Fathers	56.5%	51.7%	64.2%	43.7%
Sons	Mothers	60.9%	54.1%	86.0%	22.9%
	Fathers	56.3%	44.6%	81.9%	18.6%
Both	**Mothers**	**62.0%**	**48.3%**	**74.3%**	**34.5%**
	Fathers	**56.4%**	**49.6%**	**72.9%**	**32.2%**
BMI					
Daughters	Mothers	65.2%	49.4%	67.3%	47.1%
	Fathers	57.0%	52.7%	65.8%	43.5%
Sons	Mothers	60.6%	49.4%	79.2%	28.3%
	Fathers	58.7%	51.7%	79.0%	28.9%
Both	**Mothers**	**62.8%**	**49.4%**	**72.8%**	**38.1%**
	Fathers	**57.9%**	**52.3%**	**72.2%**	**36.8%**
WC					
Daughters	Mothers	66.2%	45.0%	46.0%	65.3%
	Fathers	58.5%	50.3%	44.9%	63.6%
Sons	Mothers	64.1%	45.2%	40.6%	68.2%
	Fathers	57.8%	44.8%	37.8%	64.7%
Both	**Mothers**	**65.2%**	**45.1%**	**43.5%**	**66.7%**
	Fathers	**58.2%**	**47.6%**	**41.4%**	**64.1%**
WHR					
Daughters	Mothers	65.0%	42.2%	27.2%	78.4%
	Fathers	56.2%	47.7%	26.6%	76.4%
Sons	Mothers	71.0%	43.4%	13.4%	92.4%
	Fathers	56.3%	43.8%	10.8%	89.3%
Both	**Mothers**	**66.7%**	**42.8%**	**20.8%**	**85.1%**
	Fathers	**56.3%**	**45.7%**	**18.9%**	**82.3%**

The proportion of obese South Australians in this study was similar to the national figure (28.1% compared to 26.8%). Our study found that in this population, offspring were more likely to be obese across three of the four measures (BMI, WC and WHtR but not WHR) if their parents were also obese, and the association was stronger for daughters than for sons. Like our study, an earlier study of American families using skinfold thickness measurements reported that mothers of the adult offspring were no more obese than fathers, which may be age-related. In contrast to our study, this

study reported no difference in the size of parents of obese sons when compared to obese daughters, which may be due to the different measure used [11]. A study among Canadian families examining obesity risk reported a higher risk ratio for first degree relatives than spouses using BMI, however this was the opposite when using skinfold measurements [246].

Our results also support recent findings from British [56] and Irish [105] studies examining multiple generations suggesting that there is a stronger maternal influence for BMI. The comparable studies used measured data of offspring participants and their children, and reported data for parents. Findings from the British study included that increased maternal BMI was associated with offspring who had a higher consumption of fried foods, a higher level of television watching and smoking, and a lower consumption of fruit. Paternal BMI was considered to have fewer associations with their offspring's lifestyles in a separate study [247].

There is an ongoing debate regarding the relative contributions of genetic and environmental factors [64,158]. Repeated early research by one group in Denmark reported a strong association of weight status between adoptees and their biological parents [58]. However, it is argued that the global increase over the past 30 years cannot be explained by biological factors alone and that complex environmental changes, including changes to type and amount of foods consumed, physical activity and socioeconomic factors, play a key role [13,248].

The majority of earlier studies were based on results from BMI and/or skinfold measurements. A main strength of our study was the ability to compare the

association of parental body shape using four clinically measured weight indices. BMI

is a composite measure of height and weight, endorsed by the WHO as the most useful

population-level measure [249], as well as being inexpensive and relatively simple to

determine by self-report or by clinical measure. WC, WHR and WHtR are indices of

abdominal obesity. It is recognised that android or "apple" shaped bodies have a

stronger association with obesity-related health risks than gynoid or "pear" shaped

bodies [183]. WC alone is useful in predicting this risk [185,236] and together with

BMI, has been shown to have stronger correlations with systolic and diastolic blood

pressure than WHR. WC together with HC allows the calculation of WHR, providing

another measure of centralised fat distribution. WHR is purported to be a more

powerful predictor of CVD related deaths than WC and in turn, more powerful than

BMI in both sexes [250]. In a study of adult cardiometabolic risk in different

nationalities, WHtR was observed to improve discrimination by 4-5% (compared

with BMI) and 3% (compared with WC). WHtR has been shown to be significantly

better than WC in screening for diabetes, CVD, hypertension and the metabolic

syndrome overall [188,212]. It is acknowledged that each of these measures have

limitations when used in isolation. An examination of BMI, WC and WHR within the

NWAHS cohort at baseline was undertaken to explore the limitations of each

measure, and to determine if participants would be classified as obese using different

criteria. It reported that of those women with a normal BMI, 19.0% had a high WC

(≥80 cm), while 8.5% had a high WHR (>0.85). There were corresponding lower

proportions for men - 3.4% for WC (≥90 cm) and 0.1% for WHR (1.0). Conversely it

found that 10.9% of those with a high WHR and 7.8% of those with a high WC were

classified as being underweight or normal weight using BMI [187]. Therefore, each

measure has a role in identifying people who are overweight or obese with their associated cut-offs being useful as a means to predict risk of chronic disease.

Another strength of the study was the use of clinical rather than self-reported anthropometric measurements, as the latter have been shown to provide an over-estimation of people's height and an under-estimation of their weight compared to biomedical measures [176].

It was found that identified changes over time in the height-related measures (BMI and WHtR) were not due to any significant variation in participant height. There was minimal loss in height between Stages 1 and 3 mainly due to the effect of age, with the mean height for women being 161.9 cm (SD 6.56) and 161.2 cm (SD 6.74) respectively; and for men 175.5 cm (SD 7.06) and 175.1 cm (SD 7.12) respectively.

Fair to moderate PPVs of between 35 to 45% were observed for both WHtR and BMI. This suggests that overall, among those offspring who were underweight or normal weight at baseline and who identified their mother or father as overweight/obese in pictograms, almost half were overweight/obese according to WHtR and one-third were overweight/obese according to BMI at Stage 3. Higher PPVs were seen for sons (52 to 62%) than daughters (~36%). In terms of sensitivity, rates varied from 45% to 61% across all four offspring body shape measures. This suggests that approximately half of overweight/obese offspring could be identified from parental overweight/obese pictograms. The rates of specificity were generally about 52%.

There are limitations in this study that need to be highlighted. These include the use of cross-sectional and self-reported data and some responder bias due to response rates. There was some loss to follow-up in two surveys incorporated in the analysis sample. Regarding TFU2, of the initial cohort of 4056, 8.4% (n=341) were unable to take part due to death, illness or incapacity or loss, and a further 17.7% (n=719) withdrew from the cohort study, were unable to be contacted or declined to take part. Regarding Stage 3, the corresponding figures for loss to follow-up were 8.5% (n=346) and 20.7% (n=839). An examination was undertaken of the representativeness of cohort participants compared to Australian Bureau of Statistics Estimated Residential Population age and sex data, and to demographic and risk factor information from a statewide health and wellbeing surveillance telephone survey (South Australian Monitoring and Surveillance System) [251]. It showed that by Stage 3, NWAHS had a higher proportion of females and older people, and that study participants were more likely to be employed, have a certificate or trade level of education, and to have a higher level of gross annual household income [251]. They were also more likely to report better overall health, to be ex- or non-smokers and to be obese (based on self-report) [251].

Parental obesity has been suggested as one factor in a complex interaction between human behaviour, genetic disposition and the environment which can contribute to obesity. Ideally biomedical measures of the participants' parents would be used, however the focus of our cohort study is the epidemiology of chronic disease and health-related risk factors among participants. Only limited information has been collected about participants' parents, including their midlife body shape, occupation for most of their life and country of birth for initial exploration of life-course factors.

Pictograms were originally formulated to determine the body build of the parents of both adoptees and biological parents where reported and/or measured information was not available, for example when parents have died [9], and were considered to be accurate representations [101]. These pictograms were also used in the Danish Nurse Cohort Study to determine familial predisposition to obesity [252]. Sorensen et al [101] argue that while reports of body weight are less accurate than measurements, they are also less costly and enable epidemiological studies of obesity to be undertaken. They further highlight their value in separating extremes of the distribution, as well as allowing associations between relative weights of people to be investigated, particularly where absolute values are not available [101]. In their study, participants were asked in 1979 to recall parental body shape during the early 1960s, some 15 or so years earlier, which was deemed to be sufficiently accurate [101]. This is similar to the approximate 17 year recall period asked of our study participants, whose mean age at Stage 3 was 57.6 years. Body shape at age 40 allows for consistency of recall across study participants, while avoiding earlier ages when parents are predominantly growing their families, as well as later middle age when people's metabolism slows and weight gain is often experienced. It is also argued that while midlife parental height may be reported quite accurately, midlife parental weight would be less easily recounted. There have been some criticisms of the use of pictograms as representations of body shape, relating to coarseness of the scale with loss of information through the need to reduce the response to fit one of the options. Secondly, the restriction of the range of responses and the limited number of options available may lead to an inability to provide a standard deviation around the response. In addition, concern has been expressed regarding the method of presentation such as silhouettes being presented in ascending or descending order in

one figure, rather than randomly presented as separate figures. There is also criticism regarding the scale of measurement in that silhouettes are inconsistent in size across the scale and all figures are the same height [201]. However, a number of studies have regarded pictograms to be a valid measure for the discrimination of overweight or obese compared to normal individuals, which can be reliably used for the estimation of BMI [199,200].

The use of quick and easy to use pictograms to highlight a person's risk of becoming obese like their parents may assist general practitioners with obesity management of their patients. A recent study reported that national guidelines regarding the documentation of height, weight and waist circumference were only being partially met, with 22.2% of patients having a recorded BMI score and 3.4% having a recorded waist circumference in their medical record [253]. Incorporating these measures may assist with improved health outcomes for people at risk of developing obesity-related diseases such as diabetes and hypertension.

5.8 Conclusion

In conclusion, this is the first study, to our knowledge, to examine the influence of parental and adult offspring body shape in an Australian population. It provides further evidence that parental obesity increases the risk of obesity for adult offspring, both for overall body shape (as measured by BMI) as well as central adiposity (as measured by WC, WHR and WHtR). It also highlights the differences across four weight measures; two of which (BMI and WC) are used routinely to provide an indication of a person's weight status, while providing evidence of the usefulness of another two measures (WHR and WHtR) in estimating the risk status regarding CVD

and related factors such as hypertension. Using the adage "like mother, like daughter"(and similarly, father and son), pictograms could be used as a screening tool among young and early middle-aged adults in primary care settings to promote discussion regarding possible future risk of obesity, who may not recognise that this may be a problem in their family and for them in particular. This may lead to lifestyle changes to reduce weight, which may impact on the health-related consequences of obesity, particularly cardio-metabolic disease.

5.9 Supplementary Table

The following supplementary table (Table 5.5) provides a comparison of

demographic variables between the baseline cohort (n=4056) and the analysis

sample (n=2097).

Table 5.5 (Supplementary Table S3) Comparison of demographic variables for Stage 1 overall and Analysis Sample for adult sons and daughters (unweighted)

DEMOGRAPHICS (self-reported)	STAGE 1 (baseline) (1999-2000 - n=4056)				ANALYSIS SAMPLE (n=2124)			
	Males		Females		Males		Females	
	n	%	n	%	n	%	n	%
Age								
18 to 29 years	238	12.3	228	10.7	40	4.1	39	3.5
30 to 39 years	306	15.8	370	17.4	128	13.1	137	12.3
40 to 49 years	391	20.2	484	22.8	214	21.9	270	24.2
50 to 59 years	380	19.7	415	19.5	242	24.7	314	28.1
60 to 69 years	304	15.7	318	15.0	212	21.7	199	17.8
70 years and over	313	16.2	309	14.5	143	14.6	159	14.2
Marital status								
Married or living with partner	1208	62.5	1253	59.0	701	71.8	714	64.0
Separated/ divorced	262	13.6	317	14.9	134	13.7	164	14.7
Widowed	110	5.7	265	12.5	46	4.7	138	12.4
Never married	344	17.8	274	12.9	93	9.5	97	8.7
Work status								
Full time employed	942	48.8	489	23.0	533	54.6	294	26.4
Part time/casual employed	173	9.0	517	24.3	98	10.0	288	25.8
Unemployed	91	4.7	55	2.6	25	2.6	25	2.2
Home duties	17	0.9	538	25.3	6	0.6	240	21.5
Retired	570	29.5	395	18.6	277	28.4	239	21.4
Student	51	2.6	51	2.4	11	1.1	7	0.6
Other	72	3.7	52	2.4	22	2.3	19	1.7
Highest educational qualification obtained								
Up to secondary	642	33.2	1107	52.1	331	33.9	645	57.8
Trade/apprenticeship	601	31.1	83	3.9	261	26.7	29	2.6
Certificate/diploma	416	21.5	586	27.6	228	23.4	265	23.8
Bachelor degree or higher	210	10.9	263	12.4	146	15.0	165	14.8
Other	11	0.6	11	0.5	7	0.7	6	0.5
Gross annual household income								
Up to $12,000	214	11.1	363	17.1	58	5.9	116	10.4
$12,001 to $20,000	283	14.6	333	15.7	105	10.8	167	15.0
$20,001 to $40,000	531	27.5	498	23.4	249	25.5	289	25.9
$40,001 to $60,000	416	21.5	383	18.0	225	23.1	206	18.5
$60,001 to $80,000	185	9.6	223	10.5	144	14.8	146	13.1
More than $80,000	205	10.6	193	9.1	174	17.8	148	13.3

Note: Not stated not shown

5.10 Extra analyses not included in the published paper

The following original published tables regarding the body shape of study participants (designated as adult offspring) have been updated to include ABSI quartiles. Table 5.6 shows that daughters were more likely than sons to be categorised in Quartiles 1 and 2, and less likely to be categorised in Quartiles 3 and 4, at both baseline and second follow-up.

Table 5.6 Addition of ABSI at baseline to original Table 5.2 - Body shape of study participants for baseline and second follow-up

WEIGHT MEASURES	DAUGHTERS				SONS			
	Baseline		Follow-Up		Baseline		Follow-Up	
	n	%	n	%	n	%	n	%
OFFSPRING (measured)								
BMI								
Underweight/Healthy weight (<25)	429	38.1	349	31.0	241	24.1	191	19.1
Overweight (25-29)	367	32.6	387	34.4	493	49.2	477	47.6
Obese (≥30)	330	29.3	390	34.6	268	26.7	333	33.2
Central adiposity								
Android obesity (WHR>1.0 males; >0.85 females)	284	25.2	430	38.2	110	11.0	246	24.6
High WC (≥102 cm males; ≥88 cm females)	469	41.7	571	50.7	367	36.6	460	45.9
High WHtR (≥0.5)	686	60.9	777	69.0	824	82.2	853	85.1
ABSI quartiles								
Quartile 1	*503*	*44.7*	*465*	*41.3*	*46*	*4.6*	*81*	*8.1*
Quartile 2	*360*	*32.0*	*347*	*30.8*	*195*	*19.5*	*181*	*18.1*
Quartile 3	*174*	*15.5*	*200*	*17.8*	*374*	*37.3*	*330*	*32.9*
Quartile 4	*89*	*7.9*	*114*	*10.1*	*387*	*38.6*	*410*	*40.9*
TOTAL	**1126**	**100.0**	**1126**	**100.0**	**1002**	**100.0**	**1002**	**100.0**

Table 5.7 shows that proportions for daughters and sons are similar for the two heavier quartiles (Quartiles 3 and 4) for one or both parents being either a healthy or unhealthy weight, with the highest proportions (daughters 38.5%/sons 37.3%) for those whose parents were both an unhealthy weight. It is worthwhile noting that there is no association between ABSI and adult offspring-reported midlife parental

body shape. To be able to show this association, the silhouettes would need to be

updated to show the type of obesity, ie 'apple-' or 'pear-' body shapes.

Table 5.7 Addition of ABSI at baseline to original Table 5.3 - Unadjusted odds ratios (proportions, 95% confidence intervals and p values) for overweight/obese offspring measures of parental body shape/weight

Overweight/ obese (Stage 1)*	Both parents healthy weight	Father UNHEALTHY weight				Mother UNHEALTHY weight				Both parents UNHEALTHY weight			
	Overall n=453 *Daughters n=242* *Sons n=211* Ref 1.0	Overall n=353 *Daughters n=176* *Sons n=177*				Overall n=431 *Daughters n=239* *Sons n=192*				Overall n=715 *Daughters n=369* *Sons n=346*			
(measured)	n (%)	n (%)	OR	95% CI	p	n (%)	OR	95% CI	p	n (%)	OR	95% CI	p
BMI overall	264 (58.3%)	236 (66.9%)	1.44	(1.08 - 1.93)	0.013	292 (67.7%)	1.50	(1.14 - 1.98)	0.004	536 (75.0%)	2.14	(1.67 - 2.76)	<0.001
Daughters	117 (48.3%)	104 (59.1%)	1.54	(1.04 - 2.28)	0.030	152 (63.6%)	1.87	(1.30 - 2.69)	0.001	254 (68.8%)	2.36	(1.69 - 3.30)	<0.001
Sons	147 (69.7%)	132 (74.6%)	1.28	(0.82 - 2.00)	0.284	140 (72.9%)	1.17	(0.76 - 1.81)	0.472	282 (81.5%)	1.92	(1.29 - 2.86)	0.001
WC overall	140 (30.9%)	127 (36.0%)	1.26	(0.94 - 1.69)	0.129	176 (40.8%)	1.54	(1.17 - 2.04)	0.002	317 (44.3%)	1.78	(1.39 - 2.28)	<0.001
Daughters	74 (30.6%)	69 (39.2%)	1.46	(0.97 - 2.20)	0.067	99 (41.4%)	1.61	(1.10 - 2.34)	0.013	175 (47.4%)	2.05	(1.46 - 2.88)	<0.001
Sons	66 (31.3%)	58 (32.9%)	1.07	(0.70 - 1.64)	0.754	77 (40.1%)	1.47	(0.98 - 2.22)	0.065	142 (41.0%)	1.53	(1.07 - 2.19)	0.021
WHtR overall	292 (64.5%)	236 (66.9%)	1.11	(0.83 - 1.49)	0.478	303 (70.3%)	1.31	(0.98 - 1.73)	0.064	541 (75.7%)	1.71	(1.33 - 2.22)	<0.001
Daughters	126 (52.1%)	104 (59.1%)	1.33	(0.90 - 1.97)	0.154	140 (58.6%)	1.30	(0.91 - 1.97)	0.151	245 (66.4%)	1.82	(1.31 - 2.54)	<0.001
Sons	166 (78.7%)	132 (74.6%)	0.80	(0.50 - 1.28)	0.341	163 (84.9%)	1.52	(0.91 - 1.28)	0.109	296 (85.5%)	1.60	(1.03 - 2.51)	0.037
WHR overall	69 (15.2%)	54 (15.3%)	1.01	(0.68 - 1.48)	0.979	84 (19.5%)	1.35	(0.95 - 1.91)	0.095	148 (20.7%)	1.45	(1.06 - 1.99)	0.020
Daughters	50 (20.7%)	44 (25.0%)	1.28	(0.81 - 2.03)	0.295	60 (25.1%)	1.29	(0.84 - 1.97)	0.247	101 (27.4%)	1.45	(0.98 - 2.13)	0.061
Sons	19 (9.0%)	10 (5.6%)	0.61	(0.27 - 1.34)	0.215	24 (12.5%)	1.44	(0.76 - 2.73)	0.258	47 (13.6%)	1.59	(0.90 - 2.79)	0.107
ABSI overall	*217 (23.2%)*	*158 (16.9%)*	*0.88*	*(0.67 - 1.17)*	*0.658*	*208 (22.3%)*	*1.01*	*(0.78 - 1.32)*	*0.915*	*351 (37.6%)*	*1.05*	*(0.83 - 1.33)*	*0.692*
Daughters	*56 (23.9%)*	*33 (14.1%)*	*0.77*	*(0.47 - 1.24)*	*0.280*	*55 (23.5%)*	*0.99*	*(0.65 - 1.52)*	*0.973*	*90 (38.5%)*	*1.07*	*(0.73 - 1.57)*	*0.723*
Sons	*161 (23.0%)*	*125 (17.9%)*	*0.75*	*(0.47 - 1.17)*	*0.574*	*153 (21.4%)*	*1.22*	*(0.76 – 1.96)*	*0.414*	*261 (37.3%)*	*0.95*	*(0.64 - 1.42)*	*0.816*

Note: n=1952 (176 participants provided parental body shape about only one parent)

* Defined as: BMI >25; high WHR (1.00 males, 0.85 females); high WC (≥102 cm males, ≥88 cm females); high WHtR >0.05; ABSI Quartiles 3-4 (two highest quartiles/heaviest)

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Table 5.8 shows that the ABSI values were similar to those for the other four

measures.

Table 5.8 Addition of ABSI at baseline to original Table 5.4 - Sensitivity, specificity, and positive and negative predictive values of weight measures based on parental overweight/obesity status for previously underweight or normal weight adult offspring

Weight measures above cut-off by gender and parental weight status		Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value
WHtR					
Daughters	Mothers	63.3%	46.0%	64.1%	45.0%
	Fathers	56.5%	51.7%	64.2%	43.7%
Sons	Mothers	60.9%	54.1%	86.0%	22.9%
	Fathers	56.3%	44.6%	81.9%	18.6%
Both	**Mothers**	**62.0%**	**48.3%**	**74.3%**	**34.5%**
	Fathers	**56.4%**	**49.6%**	**72.9%**	**32.2%**
BMI					
Daughters	Mothers	65.2%	49.4%	67.3%	47.1%
	Fathers	57.0%	52.7%	65.8%	43.5%
Sons	Mothers	60.6%	49.4%	79.2%	28.3%
	Fathers	58.7%	51.7%	79.0%	28.9%
Both	**Mothers**	**62.8%**	**49.4%**	**72.8%**	**38.1%**
	Fathers	**57.9%**	**52.3%**	**72.2%**	**36.8%**
WC					
Daughters	Mothers	66.2%	45.0%	46.0%	65.3%
	Fathers	58.5%	50.3%	44.9%	63.6%
Sons	Mothers	64.1%	45.2%	40.6%	68.2%
	Fathers	57.8%	44.8%	37.8%	64.7%
Both	**Mothers**	**65.2%**	**45.1%**	**43.5%**	**66.7%**
	Fathers	**58.2%**	**47.6%**	**41.4%**	**64.1%**
WHR					
Daughters	Mothers	65.0%	42.2%	27.2%	78.4%
	Fathers	56.2%	47.7%	26.6%	76.4%
Sons	Mothers	71.0%	43.4%	13.4%	92.4%
	Fathers	56.3%	43.8%	10.8%	89.3%
Both	**Mothers**	**66.7%**	**42.8%**	**20.8%**	**85.1%**
	Fathers	**56.3%**	**45.7%**	**18.9%**	**82.3%**
ABSI					
Daughters	*Mothers*	*62.7%*	*41.3%*	*24.1%*	*78.8%*
	Fathers	*51.8%*	*46.3%*	*22.6%*	*76.0%*
Sons	*Mothers*	*59.5%*	*45.6%*	*77.2%*	*26.6%*
	Fathers	*54.7%*	*39.1%*	*73.9%*	*21.5%*
Both	*Mothers*	*60.3%*	*42.3%*	*48.7%*	*53.9%*
	Fathers	*54.0%*	*44.7%*	*47.7%*	*50.9%*

Chapter 6 Parental midlife body shape influences offspring self-perception of weight in a cohort of Australian adults (Publication)

6.1 Statement of Authorship

Title of Paper	Parental midlife body shape influences offspring self-perception of weight in a cohort of Australian adults
Publication Status	☑ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmited work written in manuscript style
Publication Details	Journal of Obesity and Overweight. 2016;2(3), 303-1-303-11; http://dx.doi.org/10.15744/2455-7633.2.303

Name of Principal Author (Candidate)	Janet F Grant
Contribution to the Paper	Participated in the design and co-ordination of the study, performed statistical analyses, and drafted and revised the manuscript.
Overall percentage (%)	85%
Certification	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date 7/9/17

Name of Co-Author	Catherine R Chittleborough
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

Name of Co-Author	Anne W Taylor
Contribution to the Paper	Participated in the design and co-ordination of the study, provided advice on analyses, and was involved in the drafting and revising of the manuscript. I give permission for Janet Grant to present the paper for examination towards the Doctor of Philosophy.
Signature	Date 7/9/17

6.2 Chapter 6 Contextual Statement

Together with inherited genetic and external environmental factors that influence obesity, internal aspects such as self-perception have been shown to influence weight status. Self-perception of weight and actual weight do not always match, and those people who are obese who under-estimate their weight are likely to maintain or gain their weight, leading to obesity-related health conditions and premature mortality.

The aim of this study was to answer the third and fourth research questions, "*What is the degree of misperception of body weight within an Australian adult population?*" and "*Is there an association between parental body shape in mid-life and adult offspring's self-perception of their own body shape?*". While the degree of misperception had been answered in an earlier study of the NWAHS using baseline anthropometric measures followed by the weight perception question in a later study, this was the first study, to our knowledge, to examine these questions in association with parental body shape. Our findings included that obese men with an obese mother and overweight/obese women with either parent obese were more likely to either correctly estimate or under-estimate their own weight. Those who under-estimate their weight may be more inclined to indulge in unhealthy behaviours such as excessive calorie intake and inadequate physical activity, and to have a lack of motivation to improve their health. These people are an important target for interventions, including highlighting how their parents' excessive weight may have contributed to poorer health outcomes and how this may affect their short and long term health, as well as their risk of dying earlier, particularly from cardiovascular- and cancer-related illnesses.

The following chapter presents the results of this investigation of parental midlife

body shape and its influence on offspring self-perception of weight. This manuscript

was published by the Journal of Obesity and Overweight on 1 December 2016, and

has been re-formatted to meet the requirements of this thesis. The manuscript in its

published format is included in this thesis as Appendix 2.

6.3 Abstract

Self-perception and measures of body weight and central adiposity are key indicators of a population's attitude and level of concern regarding obesity. Parental weight has been shown to be a strong determinant of the adult offspring weight. This study initially investigates the association between self-perception, and measured BMI and WC, and characterised this association by perception type (pessimist, optimist and realist). We then examined the link between (mis)perception with midlife parental body shape, which may assist with targeted interventions for those misperceiving their weight. Data were from a telephone survey (2007) and two biomedical stages (2004-06 and 2008-10) of the NWAHS (n=2710), a longitudinal cohort of Australian adults. The study included offspring measured BMI and WC and midlife parental body shape recalled from pictograms. Over half of participants misperceived their weight status, with heavier males more likely than females to underestimate their weight and females more likely to correctly estimate or overestimate their weight. Among males, higher maternal weight was associated with a greater risk of offspring being pessimistic about their weight than being an underweight/normal weight realist (BMI RR 2.03, 95% CI 1.01-4.07, p=0.046). Having an overweight mother was also associated with increased risk of both male and female offspring being obese optimists (BMI males RR 1.80, 95% CI 1.14-2.84, p=0.011/females RR 1.77, 95% CI 1.16-2.68, p=0.008; WC males RR 1.95, 95% CI 1.30-2.92, p=0.001/females RR 1.58, 95% CI 1.09-2.28, p=0.015). Higher paternal weight was also associated with being an obese realist for females (BMI RR 1.80, 95% CI 1.15-2.83, p=0.011; WC RR 1.95, 95% CI 1.23-3.09, p=0.004) but not males. Our findings suggest that having an obese parent, particularly an obese mother, may contribute to adult offspring's misperception of their weight.

6.4 Introduction

Perception of body weight and its association with actual body weight has been widely researched [15,131,141-143,254,255]. Perceived rather than actual weight has been associated with dieting and eating behaviours, and body image [124]. Social/cultural norms about what constitutes an appropriate or "normal" weight for adults often differ between public health professionals and the wider community [128]. Obesity is increasingly being accepted as normal by society [130,145]. It has been reported that when comparing themselves with 'normal' people in the general population, overweight and obese people may under-estimate their weight and subsequently lack motivation to improve their health status [141,256].

Misperception can be defined as discordance between perceived and actual weight status [144] and is also known as body-image distortion [257]. Misperception can be either an over-estimation of weight which may lead to an eating disorder [258] or more commonly, an under-estimation of weight that may be associated with a failure to address overweight or obesity and the risk of consequent health-related conditions and risk factors [126,137,144]. Misperception is multidimensional and can change across a person's lifetime due to social, biological, psychological and physiological factors [259].

Misperception of weight is increasingly common across a number of countries such as the US, where NHANES found only 28.5% of women and 61.1% of men considered themselves to be their "right" weight as opposed to being underweight or overweight [146]. A study of young adult overweight and obese women in the US (n=42) found that they perceived themselves to be larger than they actually were, particularly for

those who were overweight [260]. Similarly, a study of two household surveys in Great Britain reported an increase in underestimation of weight from 19% in 1999 to 25% in 2007, despite an associated rise in overweight/obese from 43% to 53% respectively [261]. Comparable results of adults misperceiving being overweight or obese were identified in Switzerland (women 33%; men 53%) [143], Greece (38% overall) [133], Japan (women 46%, men 39%) [262] and Sri Lanka (75% overall) [255]. Overall, men are more likely to underestimate their weight status [125,263], while women tend to overestimate [128,129,137,254]. Misperception generally increases with age [130].

Within misperception, underestimation of weight was found to be more likely among older people (aged 65 years and over), those with lower levels of income and education[129], and women with higher levels of depressive symptoms [254]. Over-estimation of weight was more common among women aged under 65 years (particularly those aged 17-35 years) and men aged 35 to 64 years [129]. Most studies utilised either self-reported or measured BMI; fewer include WC as a measure of central adiposity [142,146,255].

Parental weight has been shown to be a strong determinant of the adult offspring weight through pre-natal programming, genetic predisposition and shared environmental factors such as diet and physical activity [56,60,100,105]. A series of studies examining parent-offspring BMI associations within a 1958 British birth cohort reported that they (1) were maintained from child to mid-adulthood; (2) had strengthened over two generations; and (3) varied by socioeconomic origins rather than the lifestyle and socioeconomic position of adult offspring. There is concern that

the cycle of increasing weight within families will continue to affect subsequent generations [56].

Although there is a growing body of evidence regarding a parent-offspring association with obesity, no studies have been found that examine the association of mid-life parental body shape with self-perception of weight among adult offspring. While anthropometric measurements of parents would be ideal, pictograms that represent body size and shape have been used in a number of body image studies [119,141,205]. They have been shown to have validity as an instrument to recall parental weight where actual measurements are unavailable, including one study that explored the association between parental measured BMI and offspring's recall (15 years) based on pictograms and found correlations of 0.74 for mothers and 0.63 for fathers [101].

The aim of this study was to firstly investigate misperception of weight status among males and females, and to characterise misperception as pessimist, optimist or realist. Secondly, we examined the association between weight misperception and offspring's recall of parental body shape at age 40 using pictograms.

6.5 Methods

6.5.1 Sample

The NWAHS is a longitudinal study of 4056 randomly selected adults aged 18 years and over recruited between 1999 and 2003 from the north-west region of Adelaide, the capital of South Australia. Participants were recruited using the Electronic White Pages during an initial CATI, and the adult who had the most recent birthday in the

household was invited to participate. Exclusions included those who did not have the capacity to participate due to illness or intellectual limitations, those who were unable to communicate in English and those living in a residential institution. The study methodology has been previously described in detail [206,207].

NWAHS participants have been followed up several times since initial recruitment (Stage 1 (baseline), n=4056, response rate 49.1%). Data in this paper were from self-reported (CATI and questionnaire) and/or measured clinic assessment, primarily from the Stage 2 first follow-up clinic examination (2004-2006, n=3205, response rate 81.5%); a TFU survey in 2007 (TFU2, n=2996, response rate 90.2%); and Stage 3 (second follow-up, 2008-2010, n=2487, response rate 76.0%) (see Supplementary Table S4) for a demographic comparison of participants at Stage 2, TFU and Stage 3).

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the Human Research Ethics Committee of The University of Adelaide and of the Central Northern Adelaide Health Service (The Queen Elizabeth and Lyell McEwin Hospitals). Written informed consent was obtained from all subjects/patients.

The overall sample comprised those participants who underwent biomedical examination at Stage 2 and approximately one to three years later (mean 2 years, SD 0.5) provided information on perception of their weight status at TFU2 (n=2710 BMI, n=2691 WC). The analysis sample included participants who also provided information about parental body shape at age 40 in the Stage 3 questionnaire (BMI mother n=2055, father n=2000; WC mother n=2038, father n=1984). Table 6.5

(Section 6.9) provides a demographic comparison of participants at baseline and for the analysis sample.

6.5.2   Offspring

6.5.2.1   Body shape

At Stage 2, height without shoes was measured to the nearest 0.5 centimetres using a wall-mounted stadiometer and weight to the nearest 0.1 kilogram in light clothing and without shoes using standard digital scales. BMI values were calculated (kg/m^2) and classified according to the WHO [180]. Three measurements of WC was made to the nearest 0.1 centimetre using an inelastic tape maintained in a horizontal plane at the level of the narrowest part of the waist, with the subject standing comfortably with weight distributed evenly on both feet and the mean calculated. The definitions for WC for men and women respectively were as follows: normal <94 cm (men) and <80 cm (women); overweight 94-101 cm (men) and 80-87 cm (women); obesity, ≥102 cm (men) and ≥88 cm (women) [186]. To reduce the effect of possible bias, Stage 2 anthropometric measures were used as this was prior to participants reporting self-perception of weight status.

6.5.2.2   Self-perception of weight status

In TFU2, participants were asked "In terms of your weight, do you consider yourself to be … too thin, a little thin, normal weight, a little overweight or very overweight?". Six perception types were created based on measured BMI and WC: (1) underweight or normal weight realists correctly classified themselves as too thin, a little thin or normal when they were actually underweight or normal weight (BMI <25; WC male <94 cm, female <80 cm); (2) overweight realists correctly considered themselves to

be a little overweight when they were indeed overweight (BMI 25-29; WC male 94-101 cm, female 80-87 cm); (3) obese realists correctly considered themselves to be very overweight (BMI ≥30; WC male ≥102 cm, female ≥88 cm); (4) pessimists incorrectly classified themselves as a little overweight when they were normal weight, or very overweight when they were actually normal weight or slightly overweight; (5) overweight optimists incorrectly classified themselves as too thin or a little thin when they were actually normal weight, or normal weight when they were actually overweight; and (6) obese optimists incorrectly classified themselves as a little thin, normal or a little overweight when they were actually obese. Table 6.6 (Section 6.9) provides a cross-tabulation of self-reported perception of weight and measured BMI and WC.

6.5.3   Parents' body shape

Parental body shape was determined at Stage 3 based on a set of validated pictograms (Figure 6.1) [9]. The mean age of the study population at the time they were asked about their parents' body shape at age 40, was 58.6 years (SD 13.97). These diagrams were originally formulated to determine the body build of the parents of both adoptees and biological parents where self-reported and/or measured information was not available [9]. Pictograms have been shown to be accurate representations [101], and have been used with self-reported height and weight to determine familial predisposition to obesity [252]. Based on the Danish Nurse Cohort Study [203], study respondents were asked to identify the body shape of their biological mother and father at age 40 from nine silhouettes of each sex (Figure 6.1). Each set of silhouettes were derived into a dichotomous variable for

mothers and fathers: silhouettes 1 through 5 were classified as obese/overweight, and silhouettes 6 through 9 were classified as healthy/underweight [204].
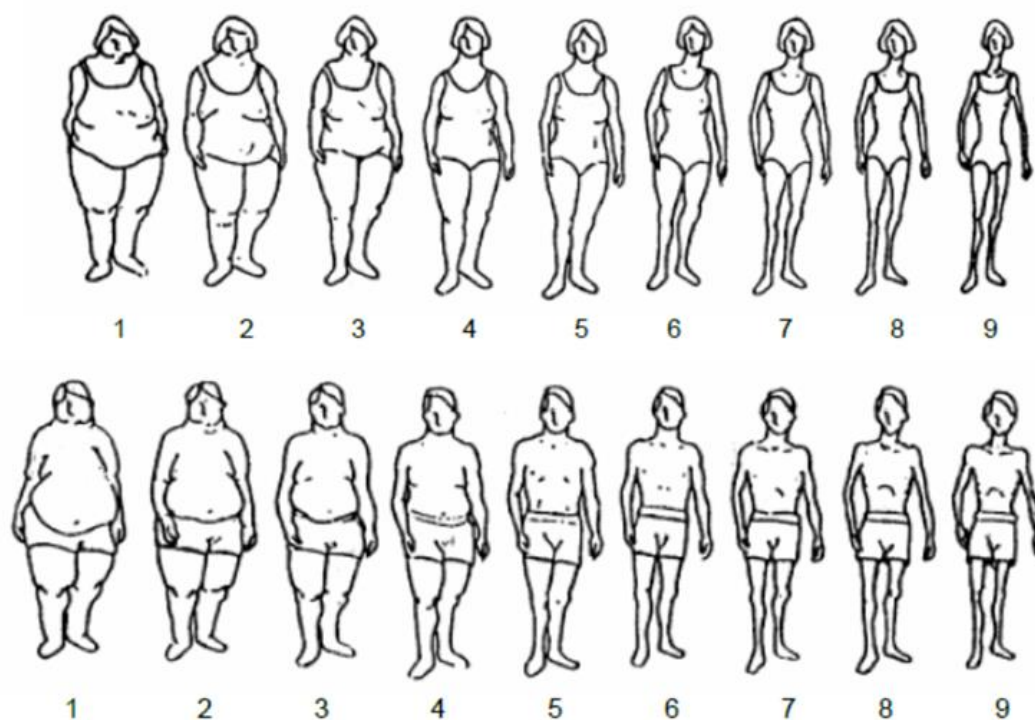
Figure 6.1 Images for perceived body shape of parents from the Figure Rating Scale

Legend: Silhouettes 1-2 = Very overweight; Silhouettes 3-4 = Moderately overweight; Silhouette 5 = Slightly overweight; Silhouettes 6-7 = Appropriate (healthy) weight; Silhouettes 8-9 = Underweight. Reprinted with permission.

Source: Adaption - Sorensen et al [9]), for use in the NWAHS

6.5.4 Statistical analysis

The unweighted data were analysed using SPSS Version 20.0 (IBM, Armonk, NY) and Stata Version 13 (StataCorp, College Station, TX). Univariable analyses using chi-square tests were undertaken on demographic characteristics and midlife parental body shape, and means for BMI and WC calculated for each category. Cross tabulations were undertaken of perceived versus measured weight status for BMI and WC, and a pictorial representation of the misperception according to offspring BMI

was produced. The number, proportion, 95% confidence intervals (CIs) and the mean BMI and WC were calculated for the six perception types. Analyses were stratified by sex due to observed differences between males and females on measured BMI and WC and self-perception of weight (p<0.001) [130].

The association between parental body shape at midlife and weight perception type (combined perception and actual own body weight using both BMI and WC) was examined for males and females using multinomial regression. Underweight/normal weight realists were the reference category, and the RR, 95% confidence intervals and p value for each is provided. The first model adjusted for age. The second model adjusted for age, parental country of birth and occupation, and number of siblings and family structure (type of relationship between adults and children living in the household, eg child/ren living with one or both biological or adopted parents or step/blended/shared care families; adults living alone, only with partner, or with related/unrelated adults) for the participant. The third model adjusted for the characteristics in model 2 and the following offspring demographics: marital status, highest educational qualification achieved, work status and gross annual household income.

6.6 Results

6.6.1 Body shape

Table 1 provides the distribution of offspring measured BMI and WC, and reported parental body shape at midlife, with the mean and SD across BMI and WC within each category.

Based on BMI, females were more likely than males to be normal weight but less likely to be overweight (overweight). Based on both BMI and WC, males were more likely than females to be overweight but less likely to be obese. Regarding parental body shape, there was a higher proportion of missing paternal (11.2%) than maternal (8.2%) information. More than half of mothers and fathers were classified as overweight or obese by their offspring.

6.6.2 Self-perception

Figure 6.2 shows four male figures and four female figures representing measured weight status (BMI), together with self-perception of weight status as a pie chart for each. The proportions for self-perception based on WC are similar. Table 6.6 (Section 6.9) provides a cross-tabulation of BMI and WC and self-perception of weight categories. Overall, 51.2% of males and 57.4% of females misperceived their weight status according to BMI. Males were more likely than females to under-estimate their actual weight (optimists) in most categories. For example, among overweight males, 35.8% perceived themselves as normal weight compared to 26.5% of females; among obese men, 64.4% perceived themselves as a little overweight compared to 55.5% of females. Females were more likely to over-estimate their actual weight (pessimists); for example, 21.7% of normal weight females considered themselves to be a little overweight compared to 16.4% of normal weight men.

Table 6.1 Distribution of offspring BMI & WC and parental body shape at midlife, with mean and standard deviation (SD) of measured BMI & WC within each category

WEIGHT MEASURES (measured)	MALES						FEMALES						
			BMI		WC* (cm)				BMI		WC* (cm)		
	n	%	Mean	(SD)	Mean	(SD)	n	%	Mean	(SD)	Mean	(SD)	p
OFFSPRING BODY SHAPE (Stage 2 – measured)													
Body Mass Index (n=2710)													<0.001
Underweight (<18.50)	4	0.3	17.7	(0.4)	72.3	(6.0)	23	1.6	17.3	(1.1)	68.7	(6.5)	
Normal (18.50-24.99)	280	22.2	23.1	(1.5)	87.5	(7.5)	475	32.8	22.4	(1.7)	76.4	(6.6)	
Overweight (25.0-29.99)	601	47.5	27.4	(1.4)	98.3	(6.4)	494	34.2	27.3	(1.4)	88.2	(6.8)	
Obese1 (30.00-34.99)	267	21.1	31.8	(1.3)	109.5	(6.6)	272	18.8	32.2	(1.5)	98.7	(6.5)	
Obese2 (35.00-39.99)	81	6.4	37.0	(1.3)	121.6	(7.1)	115	8.0	36.9	(1.3)	106.6	(6.3)	
Obese3 (40.00+)	31	2.5	43.1	(3.4)	135.4	(9.1)	67	4.6	44.5	(4.3)	118.6	(10.7)	
Waist circumference (n=2691)													0.001
Normal (M<94 cm/ F<80 cm)	383	30.5	24.2	(2.3)	87.0	(5.6)	410	28.6	22.2	(2.3)	73.4	(4.4)	
Overweight (M94-101 cm/ F80-87 cm)	339	27.0	27.2	(2.0)	97.7	(2.3)	315	21.9	25.8	(2.3)	83.9	(2.3)	
Obese (M>=102 cm/ F>=88 cm)	533	42.5	32.0	(4.3)	112.1	(9.4)	711	49.5	32.3	(5.5)	100.0	(9.6)	
Total	**1264**	**100.0**	**28.3**	**(4.7)**	**100.1**	**(12.8)**	**1446**	**100.0**	**28.0**	**(6.1)**	**88.9**	**(13.7)**	

Table 6.1 cont'd …

PARENTAL BODY SHAPE (Stage 3 – pictograms)	MALES						FEMALES						p
		BMI			WC* (cm)			BMI			WC* (cm)		
	n	%	Mean	(SD)	Mean	(SD)	n	%	Mean	(SD)	Mean	(SD)	
Mother **(overall BMI n=2055, WC n=2038)**					(n=947)						(n=1091)		0.720
Underweight/Normal weight	400	41.8	27.5	(4.2)	98.4	(11.7)	446	40.6	26.8	(5.4)	86.3	(12.5)	
Overweight	527	55.1	28.7	(4.6)	101.5	(12.5)	614	55.9	28.6	(6.5)	90.1	(14.4)	
Obese	29	3.0	32.2	(6.2)	108.3	(15.1)	39	3.5	30.6	(7.1)	92.8	(12.9)	
Total	**956**	**100.0**	**28.3**	**(4.6)**	**100.4**	**(12.4)**	**1099**	**100.0**	**27.9**	**(6.2)**	**88.6**	**(13.7)**	
Father **(overall BMI n=2000, WC n=1984)**					(n=934)						(n=1050)		0.066
Underweight/ Normal weight	415	44.0	27.9	(4.5)	99.7	(11.9)	504	47.7	27.1	(5.6)	87.1	(13.1)	
Overweight	514	54.5	28.3	(4.5)	100.6	(12.7)	528	50.0	28.4	(6.2)	89.3	(13.7)	
Obese	14	1.5	31.8	(6.4)	107.9	(14.0)	25	2.4	35.5	(9.8)	103.4	(18.3)	
Total	**943**	**100.0**	**28.2**	**(4.6)**	**100.3**	**(12.4)**	**1057**	**100.0**	**28.0**	**(6.2)**	**88.6**	**(13.8)**	

*WC males n=1255; females n=1436
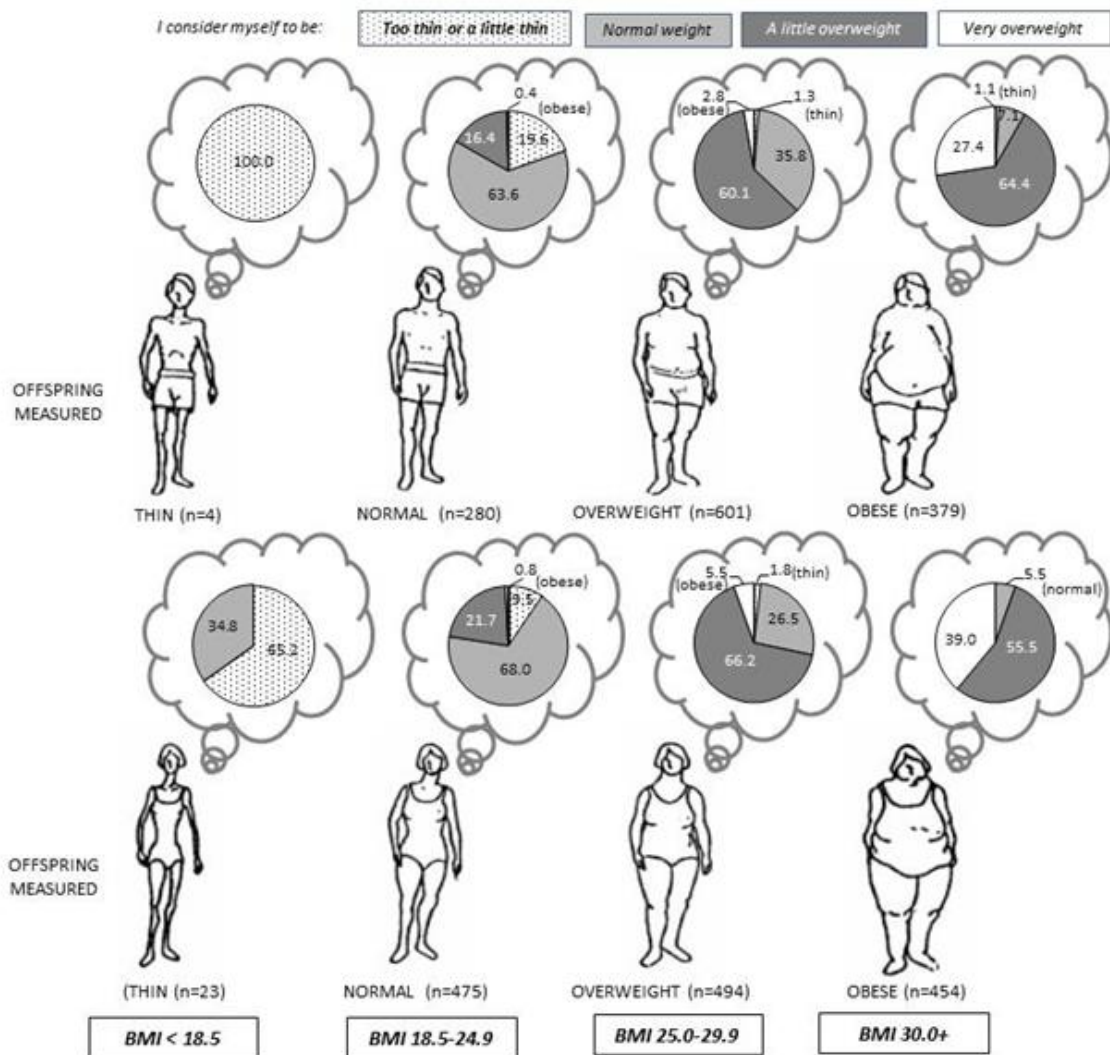
Figure 6.2 Distribution of self-perceived weight among measured BMI categories of participants

Table 6.2 provides the distribution and mean BMI and WC of males and females within the six perception types. Based on perception type by BMI, males were more likely than females to be optimists (overweight or obese) while females were more likely than males to be realists (under/normal weight or obese) or pessimists. Based on perception type by WC, females were more likely than males to be obese realists.

Table 6.2 Perception type and mean of weight measurement by BMI and WC

PERCEPTION TYPE	MALES					FEMALES					p
	n	%	95% CI	Mean	(SD)	n	%	95% CI	Mean	(SD)	
BMI*											<0.001
Underweight/Normal weight REALISTS	237	18.8	(16.7-21.0)	22.9	(1.7)	391	27.0	(24.8-29.4)	21.9	(2.0)	
PESSIMISTS	64	5.1	(4.0-6.4)	25.0	(2.2)	134	9.3	(7.9-10.9)	24.2	(2.3)	
Overweight OPTIMISTS	223	17.6	(15.6-19.8)	26.8	(1.3)	140	9.7	(8.3-11.3)	26.9	(1.4)	
Overweight REALISTS	275	21.8	(19.6-24.1)	27.7	(1.3)	277	19.2	(17.2-21.3)	27.4	(1.4)	
Obese OPTIMISTS	361	28.6	(26.1-31.1)	33.0	(2.9)	327	22.6	(20.5-24.8)	33.4	(3.1)	
Obese REALISTS	104	8.2	(6.8-9.9)	36.3	(4.8)	177	12.2	(10.7-14.0)	38.1	(5.7)	
Total	**1264**	**100.0**		**28.3**	**(4.7)**	1446	**100.0**		**28.0**	**(6.1)**	
	n	%		Mean (cm)	(SD)	n	%		Mean (cm)	(SD)	
WC* *											<0.001
Underweight/Normal weight REALISTS	283	22.5	(20.3-24.9)	86.1	(5.8)	324	22.6	(20.5-24.8)	72.7	(4.4)	
PESSIMISTS	110	8.8	(7.3-10.5)	90.5	(4.8)	97	6.8	(5.6-8.2)	76.8	(4.0)	
Overweight OPTIMISTS	138	11.0	(9.4-12.8)	97.4	(2.3)	122	8.5	(7.2-10.1)	83.4	(2.3)	
Obese OPTIMISTS	423	33.7	(31.1-36.4)	97.8	(2.3)	520	36.2	(33.8-38.7)	84.2	(2.3)	
Overweight REALISTS	191	15.2	(13.3-17.3)	110.0	(7.3)	182	12.7	(11.1-14.5)	97.3	(7.2)	
Obese REALISTS	110	8.8	(7.3-10.5)	120.4	(11.8)	191	13.3	(11.6-15.2)	107.5	(11.4)	
Total	**1255**	**100.0**		**100.6**	**(12.8)**	1436	**100.0**		**88.9**	**(13.7)**	

*BMI Underweight/Normal <25; Overweight 25-29; Obese30+

** WC Normal M<94 cm, F<80 cm; Overweight M 94-101 cm, F 80-87 cm; Obese M>=102 cm, F>=88 cm (n=18 incomplete not shown)

6.6.3 Misperception

An examination of the association between having an overweight/obese mother and/or father at age 40 and their offspring's self-perception of their own weight status was undertaken using multinominal logistic regression (Table 6.3) with underweight/normal weight realists used as the reference category. Model 3 shows that the risk of being a pessimist was twice as high for males (BMI RR 2.03, 95% CI 1.01-4.07) with an overweight/obese mother as males without an overweight/obese mother. The risk of being an obese optimist, compared to being an underweight/normal realist, was also higher for people with an overweight/obese mother (BMI males RR 1.80, 95% CI 1.14-2.84, females RR 1.77, 95% CI 1.16-2.68; WC males RR 1.95, 95% CI 1.30-2.92, females RR 1.58, 95% CI 1.09-2.28).

Compared to being an underweight/normal weight realist, the risk of being an overweight realist was higher for males and females who had an overweight/obese mother (BMI males RR 2.62, 95% CI 1.39-4.93/females RR 2.04, 95% CI 1.29-3.22; WC males RR 2.69, 95% CI 1.45-4.98/females RR 1.99, 95% CI 1.26-3.16). Having an overweight/obese father was associated with being an obese realist for females (BMI RR 1.80, 95% CI 1.15-2.83; WC RR 1.95, 95% CI 1.23-3.09) but not for males.

Regardless of which body shape measure was used, there was generally a stronger association with having an overweight/obese mother rather than father. The three models did not vary greatly after adjusting for parental or offspring demographics.

Table 6.3 Relative risks of perception type by parental body shape and offspring BMI and WC (measured)

PERCEPTION TYPE	BMI (n=2710)				WC (n=2691)			
REFERENCE CATEGORY	MALES (n=1264)		FEMALES (n=1446)		MALES (n=1255)		FEMALES (n=1436)	
Realists - Underweight/ Normal Weight	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p
PESSIMISTS								
*MOTHER Overweight/obese								
Model 1	2.09 (1.06-4.11)	0.033	1.43 (0.90-2.27)	0.129	1.36 (0.81-2.28)	0.249	1.34 (0.78-2.28)	0.285
Model 2	2.11 (1.06-4.20)	0.033	1.39 (0.87-2.22)	0.169	1.48 (0.87-2.53)	0.146	1.34 (0.78-2.31)	0.293
Model 3	2.03 (1.01-4.07)	0.046	1.40 (0.87-2.26)	0.165	1.45 (0.84-2.50)	0.181	1.36 (0.78-2.37)	0.271
**FATHER Overweight/obese								
Model 1	0.61 (0.32-1.18)	0.144	0.96 (0.60-1.52)	0.852	0.85 (0.50-1.42)	0.528	0.61 (0.35-1.06)	0.078
Model 2	0.57 (0.29-1.11)	0.100	1.00 (0.62-1.60)	1.000	0.78 (0.46-1.33)	0.366	0.60 (0.34-1.06)	0.080
Model 3	0.59 (0.30-1.16)	0.128	1.00 (0.62-1.61)	0.997	0.78 (0.46-1.35)	0.381	0.59 (0.33-1.05)	0.071
OPTIMISTS - OVERWEIGHT								
*MOTHER Overweight/obese								
Model 1	1.06 (0.68-1.67)	0.788	1.48 (0.90-2.41)	0.120	1.01 (0.61-1.67)	0.963	1.28 (0.76-2.16)	0.347
Model 2	0.96 (0.60-1.52)	0.858	1.44 (0.87-2.37)	0.152	1.06 (0.63-1.78)	0.821	1.27 (0.75-2.15)	0.375
Model 3	1.02 (0.64-1.63)	0.923	1.38 (0.83-2.30)	0.216	1.04 (0.62-1.76)	0.878	1.22 (0.71-2.11)	0.465
**FATHER Overweight/obese								
Model 1	1.19 (0.76-1.87)	0.448	1.30 (0.79-2.11)	0.299	1.00 (0.60-1.66)	0.996	1.31 (0.77-2.20)	0.316
Model 2	1.15 (0.73-1.83)	0.549	1.43 (0.87-2.36)	0.157	0.94 (0.56-1.58)	0.811	1.41 (0.83-2.39)	0.206
Model 3	1.14 (0.72-1.82)	0.570	1.56 (0.93-2.60)	0.090	0.92 (0.55-1.56)	0.766	1.42 (0.83-2.46)	0.204

Table 6.3 cont'd …

PERCEPTION TYPE contd	BMI (n=2710)				WC (n=2691)			
REFERENCE CATEGORY	FEMALES (n=1446)		MALES (n=1255)		FEMALES (n=1446)		MALES (n=1255)	
Realists - Underweight/ Normal Weight	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p
OPTIMISTS - OBESE								
*MOTHER Overweight/obese								
Model 1	1.82 (1.17-2.82)	0.008	1.86 (1.25-2.77)	0.002	1.78 (1.21-2.61)	0.003	1.69 (1.19-2.41)	0.004
Model 2	1.84 (1.17-2.88)	0.008	1.81 (1.21-2.72)	0.004	1.97 (1.32-2.93)	0.001	1.64 (1.15-2.36)	0.007
Model 3	1.80 (1.14-2.84)	0.011	1.77 (1.16-2.68)	0.008	1.95 (1.30-2.92)	0.001	1.58 (1.09-2.28)	0.015
**FATHER Overweight/obese								
Model 1	1.29 (0.83-1.99)	0.258	1.57 (1.06-2.34)	0.024	1.27 (0.86-1.87)	0.223	1.49 (1.05-2.12)	0.028
Model 2	1.25 (0.81-1.95)	0.317	1.61 (1.07-2.41)	0.021	1.24 (0.84-1.83)	0.288	1.52 (1.06-2.19)	0.022
Model 3	1.23 (0.79-1.93)	0.358	1.61 (1.07-2.44)	0.023	1.19 (0.80-1.78)	0.390	1.49 (1.03-2.15)	0.034
REALISTS - OVERWEIGHT								
*MOTHER Overweight/obese								
Model 1	1.29 (0.87-1.92)	0.207	2.15 (1.49-3.08)	0.000	1.17 (0.75-1.82)	0.478	1.98 (1.28-3.06)	0.002
Model 2	1.32 (0.88-1.98)	0.176	2.12 (1.47-3.06)	0.000	1.25 (0.79-1.95)	0.340	1.97 (1.27-3.08)	0.003
Model 3	1.32 (0.87-1.99)	0.186	2.04 (1.40-2.97)	0.000	1.23 (0.78-1.95)	0.372	1.95 (1.25-3.06)	0.004
**FATHER Overweight/obese								
Model 1	1.19 (0.80-1.78)	0.383	1.21 (0.85-1.73)	0.295	1.00 (0.64-1.55)	0.985	1.33 (0.86-2.05)	0.195
Model 2	1.15 (0.77-1.72)	0.504	1.26 (0.87-1.81)	0.215	0.98 (0.63-1.54)	0.930	1.41 (0.91-2.19)	0.127
Model 3	1.14 (0.76-1.72)	0.522	1.25 (0.87-1.81)	0.231	0.93 (0.59-1.47)	0.754	1.40 (0.90-2.19)	0.135

Table 6.3 cont'd ...

PERCEPTION TYPE contd	BMI (n=2710)				WC (n=2691)			
REFERENCE CATEGORY	FEMALES (n=1446)		MALES (n=1255)		FEMALES (n=1446)		MALES (n=1255)	
Realists - Underweight/ Normal Weight	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p	RR (95% CI)	p
REALISTS - OBESE								
*MOTHER Overweight/obese								
Model 1	2.47 (1.37-4.48)	0.003	2.11 (1.36-3.26)	0.001	2.46 (1.39-4.35)	0.002	2.07 (1.33-3.22)	0.001
Model 2	2.62 (1.41-4.84)	0.002	2.08 (1.33-3.25)	0.001	2.75 (1.52-4.99)	0.001	2.03 (1.29-3.18)	0.002
Model 3	2.62 (1.39-4.93)	0.003	2.04 (1.29-3.22)	0.002	2.69 (1.45-4.98)	0.002	1.99 (1.26-3.16)	0.003
**FATHER Overweight/obese								
Model 1	1.08 (0.61-1.90)	0.790	1.80 (1.17-2.78)	0.007	1.06 (0.61-1.83)	0.837	1.95 (1.26-3.02)	0.003
Model 2	1.03 (0.58-1.84)	0.907	1.77 (1.14-2.75)	0.011	1.01 (0.58-1.77)	0.968	1.95 (1.24-3.05)	0.004
Model 3	0.98 (0.54-1.77)	0.949	1.80 (1.15-2.83)	0.011	0.94 (0.53-1.67)	0.828	1.95 (1.23-3.09)	0.004

*Mother, n=2055; **Father, n=2000

Model 1 *adjusted for age | ***Model 2*** *adjusted for age, parents' country of birth, parents' occupation; and in offspring's early childhood - family structure, having siblings and parental unemployment |* **Model 3** *adjusted for age, parents' country of birth, parents' occupation; in offspring's early childhood - family structure, having siblings and parental unemployment; and offspring's marital, education, work and income status*

6.7 Discussion

This study found that obese men and overweight or obese women who had a heavier mother were more likely to correctly estimate or underestimate their own weight. Obese women who also had an obese father were more likely than men to correctly estimate or underestimate their own weight. Normal weight men who had a heavier mother were more likely than women to overestimate their actual weight.

The strength of these associations suggests that parents contribute to their offspring's frame of reference and this persists into adulthood. Research argues that obesity is "socially contagious" with individuals more likely to become obese themselves if people in their family, social network or neighbourhood are obese [14,141]. While Frayling argues that the causes of obesity are genetic, through the influences of appetite, metabolism and physical activity tolerance [64], Wilding acknowledges that although genetics plays a part, it is primarily environmental factors such as the lower cost of energy dense foods and the rising rates of sedentary activities that have contributed to the predominantly global increase in obesity prevalence over the last 30 years [158]. Studies of self-perception explain that because of shifts in what is considered a normal body size, increases in perceptions of desired and ideal weights, a decrease in body dissatisfaction as people age, and multiple family histories of obesity, people are more accepting of overweight and obesity in general, and less motivated to control or reduce their body size [126,140,141,143].

The association with underestimation of weight has been linked to increased hours of television viewing for males and depressive symptoms for females [254]. Those who are depressed and/or feel badly about their body image may be in a fragile

psychological state and therefore less able to make lifestyle changes to improve their health [263]. Conversely, those who overestimate their weight may be sensitive to the risk of resembling their overweight/obese parents and may be drawn to unhealthy eating patterns including unnecessary and/or excessive dieting, or binge eating and purging [126].

In this study, almost half of the participants misperceived their weight status, somewhat higher than recent findings within similar Western societies [146,261]. Almost two-thirds of participants thought that they were only a little overweight when they were actually obese. Males were more likely than females to be optimistic about their weight in line with other self-perception studies [128,129,254]; of those who considered themselves to be normal weight, more males than females were actually overweight, similar to a 2010 study [137]. It has been suggested that this may be due to heavier males seeing themselves as big and strong rather than overweight or obese [263].

It has been hypothesized that different methods of measurement (clinical versus community), different samples (including people at a stable weight or losing weight) and weight extremes may explain how overweight/obese people can be either realistic or optimistic about their weight [263]. Optimistic weight perceptions may lead to risky health behaviours; one study reported that obese women who underestimated their weight actually gained weight [141]. It is widely held that theories such as the Health Belief Model require a perceived susceptibility in order for a change in health behaviour, which may result in a desire to lose weight through improved diet or increased physical activity [144]. Chang et al estimated that

increasing one's weight by approximately three kilograms (equalling a 1 unit increase in BMI) increased the odds of being in a heavier/higher weight perception category by over 60% for both males and females, while a two-unit BMI increase tripled the odds of this [128]. Jones et al found that those whose BMI was 35 and over had less distress about emotional overeating, less weight concern and less disinhibition about overeating, as well as less time for dieting [137]. It has been suggested that those who misjudge their own weight may also misjudge their children's weight which has implications for the next generation continuing down the obesity pathway [255].

A previous study by the authors examined the association between parental body shape and the overall body shape as well as the central adiposity of their adult offspring, and found that compared to both parents being a healthy weight at age 40, those whose parents were overweight or obese were themselves more likely to be also overweight or obese, particularly if their mother was an unhealthy weight, and that this effect was stronger for daughters than for sons [264]. Guidelines for obesity management in general practice developed by the National Health and Medical Research Council recommend that doctors document BMI and WC during practice visits, in efforts to prevent and manage obesity. A study of 78 Australian general practices between July 2011 and December 2013, found that only 22.2% and 4.3% of patients had a documented BMI and WC respectively [253]. The use of pictograms in primary care settings to identify the risk from parental overweight and obesity, together with identification of the patient's own body weight status and their perception of their body shape, may prove beneficial as a screening tool for young adults in efforts to reach or maintain a healthy weight.

It is also recognised that effective treatment of patients with chronic conditions incorporates successful self-management and adherence to programs which also take into account individual's health beliefs; fear regarding their future health; frustrations with restrictions, the use of medications and the need for lifestyle changes; and possible depression [265].

Denial or inability to acknowledge one's obesity may impact on the uptake of warnings and possible strategies to achieve a healthy weight. People so affected may retreat from medical interventions and find solace and encouragement of current unhealthy lifestyle choices elsewhere, such as in obesity acceptance groups and websites [145]. However it is encouraging that weight misperception can be modified, and advice from physicians may increase successful weight loss [253].

The strengths of this study include anthropometric versus self-reported measurements and the additional information regarding severe obesity, compared with a single category of overweight/obese (BMI 25+) used in US surveys such as NHANES. This study also incorporated WC as a measure of body fat distribution, which may provide a better indicator of health risk due to the identification of location of body fat, and it has been suggested that a higher WC is associated with a higher risk of feeling overweight [142].

This study has some limitations including a single measure of weight perception of weight, approximately one to three years (mean 2 years, SD 0.5) following anthropometric measurements of height, weight, and WC and HC. An examination of weight loss/gain between baseline and Stage 3 (n=2128) found relatively stable

weight change over the approximate eight years between these studies, with approximately 37% losing weight (mean 4.7 kg, SD 5.36) and 62% gaining weight (mean 6.0 kg, SD 5.76). Other limitations of the study include that perception type was characterised based on WHO BMI cut-offs, which have been acknowledged as possibly differing regarding the degree of fatness for different ethnic groups, particularly for the Asian and Pacific populations [180]. However, as approximately 70% of the participants were born in Australia, with a further 18% born in the United Kingdom/Ireland and 9% in Europe, it is considered that the WHO BMI cut-offs were appropriate for use in this population. Parental country of birth was similar, with approximately 56% born in Australia, 23% born in the UK/Ireland, and 17% born in Europe. Another limitation is that participants may have incorporated medical standards from their general physician, or personal standards based on cultural norms or the weight of family or friends. It is acknowledged that there is divergence between clinical and public health standards of what constitutes "normal" weight and its healthy distribution within the body, and the perception among the general community [138].

Another limitation was some loss to follow-up of the initial cohort. In Stage 2, 3.1% (n=126) were unable to take part due to death, illness or incapacity or loss, while a further 14.2% (n=575) withdrew from the study, were unable to be contacted or declined to take part; the figures were 8.5% (n=345) and 14.2% (n=577) respectively for TFU2. An examination was undertaken of the representativeness of cohort participants which showed that by Stage 3, the NWAHS had a higher proportion of females and older people, and that study participants were more likely to be employed, have a certificate or trade level of education, and to have a higher level of

gross annual household income. They were also more likely to report better overall

health, to be ex- or non-smokers and to be obese (based on self-report) [209].

The focus of our cohort study is the epidemiology of chronic disease and health-

related risk factors among adult participants which limited the exploration of familial

factors such as midlife parental obesity. Sorensen et al argue that while reports of

body weight are less accurate than measurements, they are also less costly and

enable epidemiological studies of obesity to be undertaken [101]. They further

highlight their value in allowing associations between relative weights of people to be

investigated particularly where absolute values are not available, while separating

extremes of the distribution. Further, they reported recall to be sufficiently accurate

after comparing reported parental body shape in 1979 with measured values in the

early 1960s[101], similar to the approximate 17 year recall period between TFU2 and

Stage 3.

Setting the age at 40 regarding parental body shape allows for consistency of recall

while avoiding younger ages when parents are raising children, as well as later

middle and older age. It is also argued that midlife parental weight would be less

easily recounted while midlife parental height may be reported quite accurately. The

use of pictograms allows for recall of body shape when actual measurements are not

available such as when parents have died. Some criticisms of the use of pictograms as

representations of body shape relating to restricted range of responses and limited

number of available options may lead to an inability to provide a standard deviation

around the response; the coarseness of the scale with loss of information through the

need to reduce the response to fit a single option; method of presentation with

silhouettes presented in ascending or descending order in one figure, rather than randomly presented as separate figures; and scale of measurement with silhouettes inconsistent in size across the scale and all figures the same height [201]. However, a number of studies consider pictograms to be a valid estimation of BMI measure to be used in the discrimination of overweight or obese compared to normal individuals [199,200].

6.8 Conclusions

This is the first study, to our knowledge, to examine the association between parental body shape and self-perception of weight among their adult offspring. Obese people who think they are normal weight or just a little overweight are an important group to engage in discussion about healthy lifestyle choices. While balance is required to maintain healthy weight and not encourage eating disorders such as anorexia and bulimia among vulnerable people, action is required to counter ongoing denial of health risk due to obesity which may result in comorbidities and possible early mortality. Public health prevention programs which provide information to people about how their parents' overweight or obesity may have affected their health as well as providing a 'norm' regarding unhealthy weight status, together with information about their own overall weight and their central adiposity, and the possibility of associated health risks, may provide impetus for change in people's lifestyle choices, such as diet and physical activity.

6.9 Supplementary Tables

Table 6.4 (Supplementary Table S4) provides a comparison of demographic variables for male and female participants for overall Stage 2 (first follow-up biomedical examination), TFU2 (second follow-up survey) and Stage 3 (second follow-up biomedical examination), NWAHS.

Table 6.4 (Supplementary Table S4) Demographics of participants

DEMOGRAPHICS (self-reported)	STAGE 2 (2004-06) (overall n=3564)				TEL FOLLOW-UP 2 (2007) (overall n=2996)				STAGE 3 (2008-10) (overall n=2871)			
	Males		Females		Males		Females		Males		Females	
	n	%	n	%	n	%	n	%	n	%	n	%
Age												
20 to 29 years	99	5.9	90	4.8	58	4.2	46	2.9	34	2.5	28	1.8
30 to 39 years	240	14.3	263	13.9	166	12.0	188	11.7	130	9.7	146	9.5
40 to 49 years	339	20.3	437	23.1	277	20.0	351	21.8	251	18.8	311	20.2
50 to 59 years	348	20.8	431	22.8	289	20.9	372	23.1	298	22.3	364	23.7
60 to 69 years	312	18.6	311	16.5	304	22.0	311	19.3	305	22.9	322	20.9
70 years and over	336	20.1	358	18.9	289	20.9	345	21.4	316	23.7	366	23.8
Marital status												
Married/defacto	1048	67.7	1030	60.2	983	71.1	1004	62.2	904	71.9	920	63.3
Separated/ divorced	211	13.6	261	15.3	179	12.9	229	14.2	157	12.5	208	14.3
Widowed	97	6.3	256	15.0	83	6.0	246	15.3	76	6.0	205	14.1
Never married	184	11.9	158	9.2	138	10.0	134	8.3	120	9.5	117	8.1
Work status												
Full time employed	754	48.7	419	24.5	708	51.2	423	26.2	647	51.6	401	27.7
Part time/casual employed	140	9.0	403	23.6	110	8.0	388	24.1	93	7.4	324	22.4
Unemployed	42	2.7	32	1.9	35	2.5	21	1.3	19	1.5	21	1.5
Home duties	14	0.9	381	22.3	6	0.4	145	9.0	5	0.4	96	6.6
Retired	532	34.4	418	24.4	473	34.2	548	34.0	431	34.4	509	35.2
Student	16	1.0	13	0.8	3	0.2	12	0.7	4	0.3	6	0.4
Other	41	2.6	35	2.0	48	3.5	74	4.6	55	4.4	91	6.3
Highest educational qualification obtained												
Up to secondary	586	37.9	1009	59.0					526	41.8	895	61.6
Trade/apprentice-ship	402	26.0	52	3.0					100	8.0	107	7.4
Certificate/ diploma	337	21.8	394	23.0		*not asked*			411	32.7	215	14.8
Bachelor degree or higher	201	13.0	227	13.3					218	17.3	231	15.9
Don't know	12	0.8	18	1.1					-	-	-	-

Table 6.4 cont'd …

DEMOGRAPHICS (self-reported) contd	STAGE 2 (2004-06) (overall n=3564)				TEL FOLLOW-UP 2 (2007) (overall n=2996)				STAGE 3 (2008-10) (overall n=2871)			
	Males		**Females**		**Males**		**Females**		**Males**		**Females**	
	n	%	n	%	n	%	n	%	n	%	n	%
Gross annual household income												
Up to $12,000	150	9.7	224	13.1					20	1.6	39	2.7
$12,001 to $20,000	199	12.9	263	15.4					148	11.8	207	14.2
$20,001 to $40,000	385	24.9	424	24.8		*not asked*			287	22.8	363	25.0
$40,001 to $60,000	329	21.3	298	17.4					221	17.6	198	13.6
$60,001 to $80,000	189	12.2	197	11.5					170	13.5	169	11.6
More than $80,000	234	15.1	210	12.3					352	28.0	331	22.8

Note: Not stated not shown

Table 6.5 (Supplementary Table S5) provides a comparison of demographic and

weight variables for baseline (Stage 1) participants (n=4056) and for the Analysis

Sample (n=2710) for adult offspring (unweighted), NWAHS.

Table 6.5 (Supplementary Table S5) Demographics and weight measures of participants at baseline and the analysis sample

PARTICIPANT CHARACTERISTICS	MALES				FEMALES			
	Baseline		Analysis Sample		Baseline		Analysis Sample	
	n	%	n	%	n	%	n	%
DEMOGRAPHICS (self-reported)								
Age								
18 to 29 years	238	12.3	111	8.8	228	10.7	97	6.7
30 to 39 years	306	15.8	203	16.1	370	17.4	241	16.7
40 to 49 years	391	20.2	281	22.2	484	22.8	352	24.3
50 to 59 years	380	19.7	286	22.6	415	19.5	331	22.9
60 to 69 years	304	15.7	217	17.2	318	15.0	244	16.9
70 years and over	313	16.2	166	13.1	309	14.5	181	12.5
Marital status								
Married or living with partner	1208	62.5	863	68.3	1253	59.0	908	62.8
Separated/ divorced	262	13.6	169	13.4	317	14.9	208	14.4
Widowed	110	5.7	53	4.2	265	12.5	178	12.3
Never married	344	17.8	176	13.9	274	12.9	144	10.0
Work status								
Full time employed	942	48.8	695	55.0	489	23.0	349	24.1
Part time/casual employed	173	9.0	97	7.7	517	24.3	371	25.7
Unemployed	91	4.7	47	3.7	55	2.6	29	2.0
Home duties	17	0.9	12	0.9	538	25.3	357	24.7
Retired	570	29.5	342	27.1	395	18.6	271	18.7
Student	51	2.6	25	2.0	51	2.4	23	1.6
Other	72	3.7	40	3.2	52	2.4	30	2.1
Highest educational qualification obtained								
Up to secondary	642	33.2	382	30.2	1107	52.1	744	51.5
Trade/apprenticeship	601	31.1	401	31.7	83	3.9	50	3.5
Certificate/diploma	416	21.5	288	22.8	586	27.6	412	28.5
Bachelor degree or higher	210	10.9	162	12.8	263	12.4	184	12.7
Other	11	0.6	10	0.8	11	0.5	9	0.6
Gross annual household income								
Up to $12,000	214	11.1	93	7.4	363	17.1	215	14.9
$12,001 to $20,000	283	14.6	164	13.0	333	15.7	231	16.0
$20,001 to $40,000	531	27.5	351	27.8	498	23.4	343	23.7
$40,001 to $60,000	416	21.5	304	24.1	383	18.0	284	19.6
$60,001 to $80,000	185	9.6	136	10.8	223	10.5	167	11.5
More than $80,000	205	10.6	164	13.0	193	9.1	130	9.0

Table 6.5 cont'd …

PARTICIPANT CHARACTERISTICS contd	MALES				FEMALES			
	Baseline		Analysis Sample		Baseline		Analysis Sample	
	n	%	n	%	n	%	n	%
WEIGHT MEASURES (measured)								
BMI								
Underweight <18.50	12	0.6	4	0.3	32	1.5	21	1.5
Normal 18.50-24.99	515	26.7	280	22.2	776	36.5	519	35.9
Pre-obese 25.00-29.99	886	45.9	601	47.5	674	31.7	474	32.8
Obese1 30.00-34.99	377	19.5	267	21.1	368	17.3	258	17.8
Obese2 35.00-39.99	102	5.3	81	6.4	185	8.7	121	8.4
Obese3 40.00+	40	2.1	31	2.5	87	4.1	51	3.5
WC								
Normal WC M<94 cm, F<80 cm	707	36.6	429	33.9	720	33.9	486	33.6
Overweight WC M94-101 cm, F80-87 cm	523	27.1	356	28.2	482	22.7	329	22.8
Obese WC M>=102 cm, F>=88 cm	701	36.3	479	37.9	920	43.3	630	43.6
Total	**1932**	**100.0**	**1264**	**100.0**	**2124**	**100.0**	**1446**	**100.0**

Note: Not stated not shown

Table 6.6 (Supplementary Table S6) provides the self-reported perception of weight

(asked in TFU2, 2007) versus the measured BMI and WC of male and female

participants from Stage 2 (2004-2006) in the NWAHS.

Table 6.6 (Supplementary Table S6) Self-perception of weight and measured BMI & WC of participants (Telephone Follow-Up 2 and Stage 2)

WEIGHT MEASURES	SELF-PERCEPTION								Total (n=1264, 100%)
	Too thin/ a little thin		Normal weight		A little overweight		Very overweight		
	n	%	n	%	n	%	n	%	n
BMI									
Males									
Underweight <18.50	4	100.0	0	0.0	0	0.0	0	0.0	**4**
Normal 18.50-24.99	55	19.6	178	63.6	46	16.4	1	0.4	**280**
Overweight 25.00-29.99	8	1.3	215	35.8	361	60.1	17	2.8	**601**
Obese 30.00+	4	1.1	27	7.1	244	64.4	104	27.4	**379**
Females									
Underweight <18.50	15	65.2	8	34.8	0	0.0	0	0.0	**23**
Normal 18.50-24.99	45	9.5	323	68.0	103	21.7	4	0.8	**475**
Overweight 25.00-29.99	9	1.8	131	26.5	327	66.2	27	5.5	**494**
Obese 30.00+	0	0.0	25	5.5	252	55.5	177	39.0	**454**
WC									
Males									
Normal WC <94 cm	55	14.4	228	59.5	99	25.8	1	0.3	**383**
Overweight WC 94-101 cm	8	2.4	129	38.1	191	56.3	11	3.2	**339**
Obese WC >=102 cm	8	1.5	60	11.3	355	66.6	110	20.6	**533**
Females									
Normal WC <80 cm	53	12.9	271	66.1	83	20.2	3	0.7	**410**
Overweight WC 80-87 cm	13	4.1	109	34.6	182	57.8	11	3.5	**315**
Obese WC >=88 cm	3	0.4	103	14.5	414	58.2	191	26.9	**711**

6.10 Extra analyses not included in the published paper

The following original published tables regarding self-perception of weight of study participants (designated as adult offspring) have been updated to include ABSI quartiles.

Table 6.7 provides the proportion, mean and SD for BMI and WC, including for the ABSI Quartiles. It shows similar results for BMI (overweight category) for both males and females.   However for WC, males were categorised as being 'normal' for Quartiles 1 and 2, overweight for Quartile 3 and obese for Quartile 4, while females were categorised as being overweight for Quartile 1 and obese for Quartiles 2, 3 and 4. Higher proportions of males than females were in the highest quartile (Quartile 4).

Table 6.7 Addition of ABSI at baseline to original Table 6.1 - Distribution of offspring BMI & WC, with mean and standard deviation (SD) of measured BMI, WC and ABSI within each category

WEIGHT MEASURES (measured)	MALES						FEMALES						
			BMI		**WC*** (cm)				**BMI**		**WC*** (cm)		
	n	%	Mean	(SD)	Mean	(SD)	n	%	Mean	(SD)	Mean	(SD)	p
OFFSPRING BODY SHAPE (Stage 2 – measured)													
Body Mass Index (n=2710)													<0.001
Underweight (<18.50)	4	0.3	17.7	(0.4)	72.3	(6.0)	23	1.6	17.3	(1.1)	68.7	(6.5)	
Normal (18.50-24.99)	280	22.2	23.1	(1.5)	87.5	(7.5)	475	32.8	22.4	(1.7)	76.4	(6.6)	
Overweight (25.0-29.99)	601	47.5	27.4	(1.4)	98.3	(6.4)	494	34.2	27.3	(1.4)	88.2	(6.8)	
Obese1 (30.00-34.99)	267	21.1	31.8	(1.3)	109.5	(6.6)	272	18.8	32.2	(1.5)	98.7	(6.5)	
Obese2 (35.00-39.99)	81	6.4	37.0	(1.3)	121.6	(7.1)	115	8.0	36.9	(1.3)	106.6	(6.3)	
Obese3 (40.00+)	31	2.5	43.1	(3.4)	135.4	(9.1)	67	4.6	44.5	(4.3)	118.6	(10.7)	
Waist circumference (n=2691)*													0.001
Normal (M<94 cm/ F<80 cm)	383	30.5	24.2	(2.3)	87.0	(5.6)	410	28.6	22.2	(2.3)	73.4	(4.4)	
Overweight (M94-101 cm/ F80-87 cm)	339	27.0	27.2	(2.0)	97.7	(2.3)	315	21.9	25.8	(2.3)	83.9	(2.3)	
Obese (M>=102 cm/ F>=88 cm)	533	42.5	32.0	(4.3)	112.1	(9.4)	711	49.5	32.3	(5.5)	100.0	(9.6)	
ABSI (n=2690)													*<0.001*
Quartile 1	68	5.4	25.9	(3.8)	85.0	(8.9)	622	43.3	27.8	(6.6)	83.3	(13.0)	
Quartile 2	263	21.0	27.5	(4.1)	93.2	(9.6)	433	30.2	28.4	(5.9)	90.5	(12.4)	
Quartile 3	431	34.3	28.7	(4.7)	100.4	(11.2)	247	17.2	28.7	(5.6)	95.7	(12.4)	
Quartile 4	493	39.3	28.8	(4.9)	106.8	(12.2)	133	9.3	26.8	(5.3)	97.3	(12.1)	
Total	**1264**	**100.0**	**28.3**	**(4.7)**	**100.1**	**(12.8)**	**1446**	**100.0**	**28.0**	**(6.1)**	**88.9**	**(13.7)**	

*WC males n=1255, females n=1436; **ABSI males n=1255, females n=1435*

Table 6.8 provides the proportions of males and females across the ABSI quartiles for

the baseline sample (Stage 1) and the analysis sample (Stage 2), which are similar.

Table 6.8 Addition of ABSI at baseline to original Table 6.5 - Demographics and weight measures of participants at baseline and the analysis sample

PARTICIPANT CHARACTERISTICS contd	MALES				FEMALES			
	Baseline		Analysis Sample		Baseline		Analysis Sample	
	n	%	n	%	n	%	n	%
WEIGHT MEASURES (measured)								
BMI								
Underweight <18.50	12	0.6	4	0.3	32	1.5	21	1.5
Normal 18.50-24.99	515	26.7	280	22.2	776	36.5	519	35.9
Pre-obese 25.00-29.99	886	45.9	601	47.5	674	31.7	474	32.8
Obese1 30.00-34.99	377	19.5	267	21.1	368	17.3	258	17.8
Obese2 35.00-39.99	102	5.3	81	6.4	185	8.7	121	8.4
Obese3 40.00+	40	2.1	31	2.5	87	4.1	51	3.5
WC								
Normal WC M<94 cm, F<80 cm	707	36.6	429	33.9	720	33.9	486	33.6
Overweight WC M94-101 cm, F80-87 cm	523	27.1	356	28.2	482	22.7	329	22.8
Obese WC M>=102 cm, F>=88 cm	701	36.3	479	37.9	920	43.3	630	43.6
ABSI								
Quartile 1	*103*	*5.3*	*68*	*5.4*	*910*	*42.9*	*622*	*43.3*
Quartile 2	*372*	*19.3*	*263*	*21.0*	*641*	*30.2*	*433*	*30.2*
Quartile 3	*656*	*34.0*	*431*	*34.3*	*357*	*16.8*	*247*	*17.2*
Quartile 4	*800*	*41.4*	*493*	*39.3*	*213*	*10.0*	*133*	*9.3*
Total	**1932**	**100.0**	**1264**	**100.0**	**2124**	**100.0**	**1446**	**100.0**

Table 6.9 provides a comparison of self-perception of weight from TFU2, with

measured ABSI quartiles at Stage 2. It shows that there was a higher proportion of

males than females across all categories of self-perception within Quartile 4.

Table 6.9 Addition of ABSI at baseline to original Table 6.6 - Self-perception of weight and measured BMI & WC of participants

WEIGHT MEASURES	SELF-PERCEPTION								Total (n=1264, 100%)
	Too thin/ a little thin		Normal weight		A little overweight		Very overweight		
	n	%	n	%	n	%	n	%	n
BMI									
Males									
Underweight <18.50	4	100.0	0	0.0	0	0.0	0	0.0	**4**
Normal 18.50-24.99	55	19.6	178	63.6	46	16.4	1	0.4	**280**
Overweight 25.00-29.99	8	1.3	215	35.8	361	60.1	17	2.8	**601**
Obese 30.00+	4	1.1	27	7.1	244	64.4	104	27.4	**379**
Females									
Underweight <18.50	15	65.2	8	34.8	0	0.0	0	0.0	**23**
Normal 18.50-24.99	45	9.5	323	68.0	103	21.7	4	0.8	**475**
Overweight 25.00-29.99	9	1.8	131	26.5	327	66.2	27	5.5	**494**
Obese 30.00+	0	0.0	25	5.5	252	55.5	177	39.0	**454**
WC									
Males									
Normal WC <94 cm	55	14.4	228	59.5	99	25.8	1	0.3	**383**
Overweight WC 94-101 cm	8	2.4	129	38.1	191	56.3	11	3.2	**339**
Obese WC >=102 cm	8	1.5	60	11.3	355	66.6	110	20.6	**533**
Females									
Normal WC <80 cm	53	12.9	271	66.1	83	20.2	3	0.7	**410**
Overweight WC 80-87 cm	13	4.1	109	34.6	182	57.8	11	3.5	**315**
Obese WC >=88 cm	3	0.4	103	14.5	414	58.2	191	26.9	**711**
ABSI									
Males									
Quartile 1	*8*	*11.3*	*35*	*8.4*	*25*	*3.9*	*0*	*0.0*	***68***
Quartile 2	*17*	*23.9*	*92*	*22.1*	*144*	*22.3*	*10*	*8.2*	***263***
Quartile 3	*15*	*21.1*	*148*	*35.5*	*221*	*34.3*	*47*	*38.5*	***431***
Quartile 4	*31*	*43.7*	*142*	*34.1*	*255*	*39.5*	*65*	*53.3*	***493***
Females									
Quartile 1	*27*	*39.7*	*236*	*48.9*	*272*	*40.1*	*87*	*42.4*	***622***
Quartile 2	*24*	*35.3*	*131*	*27.1*	*216*	*31.8*	*62*	*30.2*	***433***
Quartile 3	*10*	*14.7*	*65*	*13.5*	*131*	*19.3*	*41*	*20.0*	***247***
Quartile 4	*7*	*10.3*	*51*	*10.6*	*60*	*8.8*	*15*	*7.3*	***133***

Perception type (realist, pessimist and optimist) could not be created for ABSI as BMI

and more specifically WC for ABSI quartiles at Stage 2 do not match appropriately,

particularly for females (see Table 6.10). As indicated in Table 6.7, while BMI was

overweight across the quartiles for both males and females, the categories for WC

differed between males and females.

Table 6.10 Mean BMI and WC and categories for ABSI quartiles by sex at Stage 2 (n=2690; males n=1255/females n=1435)

ABSI	Mean BMI *(category - all overweight)*		Mean WC (cm) *(category)**	
	Males	Females	Males	Females
Quartile 1	*25.9*	*27.8*	*85.0 (normal)*	*83.3 (overweight)*
Quartile 2	*27.5*	*28.4*	*93.2 (normal)*	*90.5 (obese)*
Quartile 3	*28.7*	*28.7*	*100.4 (overweight)*	*95.7 (obese)*
Quartile 4	*28.8*	*26.8*	*106.8 (obese)*	*97.3 (obese)*

* WC categories: Normal (M<94 cm/ F<80 cm); Overweight (M94-101 cm/ F80-87 cm); Obese (M>=102 cm/ F>=88 cm) [186]

Chapter 7 Discussion, future directions and conclusions

7.1 Introduction

This chapter comprises a summary of the findings from each study undertaken for this thesis in answer to the four original research questions. It then reiterates the strengths and limitations of the thesis (although it will not revisit the discussion contained in each of the published/submitted papers). It finally discusses implications of the research undertaken in this thesis and suggests future directions for action.

7.2 Summary of findings

This thesis examined a recently developed predictor of mortality risk (the ABSI) that incorporated WC as well as height and weight, and to investigate the extent to which this prediction measure was associated with obesity-related causes of death. It then considered parental body shape, as both a genetic and environmental risk factor for obesity, and examined its association with the body shape of adult offspring across four weight measures. Lastly, it determined the level of weight misperception within an Australian population, and investigated the association of parental body shape with how adult offspring perceived their own weight status.

The first research question was "*Is there a dose-response relationship between obesity, as measured by central adiposity and body mass index, and a person's mortality risk?*". The joint influence of genetic factors and environmental factors experienced during childhood that contribute to a person being obese, together with an unrealistic perception that they have a healthy weight status in adulthood, may contribute to an unhealthy weight and ongoing weight gain which ultimately may lead to premature mortality. Chapter 4 used the ABSI to predict the mortality risk of carrying excess

central fat in addition to being heavier overall, within a cohort of Australian adults. Quartiles calculated within this measure were used to determine that having a higher WC was particularly predictive for all-cause mortality and for CVD- and cancer-related mortality. For those in Quartile 4, with the highest WC/BMI, the mortality risk was more than two and a half times those with the lowest mortality risk. This study found there was a dose-response to the risk of dying prematurely compared to those in the first quartile, with those in the second quartile being one and a half times higher, and those in the third quartile having a risk almost two times higher. The novelty of this study was that it was the first Australian study using ABSI and also the first to include primary cause of death information at the ICD10 code level and across the ABSI quartiles, which allowed for investigation of cause of death at a more detailed level.

The second research question was "*Is there a relationship between parental body shape in mid-life and adult offspring's BMI and central adiposity among Australian adults?*". In consideration of risk factors for the development of obesity in adults, Chapter 5 explored the association between midlife parental body shape (using pictograms regarding each parent at age 40 for recall by adult offspring) and four biomedically measured indicators of the adult children's obesity and fat distribution (namely BMI, WC, WHR and WHtR), as well as ABSI in the extra analyses. The novelty of this study included measures of central adiposity; most other studies investigating possible similarities between the body shape of parents and their adult children have focused on BMI alone. Pictograms of the body shape for each parent at age 40 years were used as a recall measure of parental body shape by their adult offspring. This study found that people who had two obese parents were more likely to be

overweight or obese themselves, compared to people who had both parents who were a healthy weight. The study also found that those with only one parent who was obese were more likely to be overweight or obese, more so if they had an obese mother. There were differences by gender, with daughters of one or both obese parents more likely to be overweight or obese than sons for three of the four measures examined (BMI, WC and WHtR). In the extra analyses not included in the original publication, daughters and sons had similar ORs, except for those who had an obese mother where daughters were slightly more likely than sons to be in the highest two ABSI quartiles. In an investigation of which measure provided the strongest association with parental body shape, BMI came first, followed by (in order) WC, WHtR, WHC and ABSI. WHtR provided the highest positive predictive values for overweight and obesity from parental body shape, followed by (in order) BMI, ABSI, WC and WHR.

The third and fourth research questions were "*What is the degree of misperception of body weight within an Australian adult population?*" and "*Is there an association between parental body shape in mid-life and adult offspring's self-perception of their own body shape?*". Following confirmation of an association between parental body shape and adult offspring body shape, Chapter 6 then considered these as yet unexplored questions. The novelty of this study is that it was the first, to our knowledge, to examine misperception using anthropometric measures in association with parental body shape. The study used a question in a telephone survey to determine participants' perception of their weight status and then compared this self-perception with their biomedically measured weight status (BMI). Pictograms of the body shape for each parent at age 40 years were used as a proxy for parental weight

status. It was found that for men, having a heavier mother was associated with being pessimistic about their weight, that is they thought that they were heavier than they actually were, compared to males who were realistic (that is accurate) about being underweight or normal weight. Conversely, having a heavier mother also was associated with another group, namely obese adult males and females who were optimistic about their weight status; that is, they thought that they were not as heavy as they actually were. Having a heavier father was only associated with having a realistic view about their weight status for obese females; that is, their perception matched their measured weight status.

7.3 Strengths

There are three major strengths of these three studies, which are based primarily on the use of a cohort study of South Australian adults from the northern and western suburbs of Adelaide (the NWAHS), who participated in surveys and clinic examinations for more than a decade. The collection of core data elements across time from cohort study participants allowed intergenerational aspects to be explored, as well as changes over time in their health and social circumstances. Strengths regarding use of a cohort design for this research have also been provided within each publication.

The first major strength in using the NWAHS was its use of biomedical measures of obesity, instead of self-reported. Self-reported measures have been shown to provide an over-estimation in height and an under-estimation of weight [235]. Waist and hip circumference were also measured, which allowed for the calculation of WHR, providing another measure of centralised fat distribution. WHR is considered to be a

more powerful predictor of CVD related deaths than WC and in turn, more powerful than BMI in both sexes [250]. Using height and WC allows for WHtR to be easily determined both for research and in clinical practice. WHtR was observed to improve discrimination of adult cardiometabolic risk by 4-5% (compared with BMI) and 3% (compared with WC) in a study in different nationalities. It has been shown to be significantly better than WC in screening for diabetes, CVD, hypertension and the metabolic syndrome overall [188,212]. Valuable clinical information was provided through the inclusion of WC, in particular CVD risk factors [185,236]. Research on body shapes has found that gynoid or "pear" shaped bodies have less of an association with obesity-related health risks than android or "apple" shaped bodies [183].

The second major strength is that other health-related risk factors were biomedically measured in the NWAHS including blood pressure, cholesterol, triglycerides and glycated haemoglobin which were used as confounders.

The third major strength is the cohort study design which allows for observations over time, providing different data points to be used when examining each research question. The study also provided a wealth of demographic and socioeconomic factors to be considered in the analyses. Strong strategies for retention of cohort participants allowed for healthy response rates for each of the three major biomedical stages and the two minor telephone follow-up surveys in between them, all providing valuable information to allow for data mining to address research interests. Ongoing associations with various research-related organisations has

allowed useful outcome data such as date and cause of death, regarding the same group of individuals.

7.4 Limitations

There were a number of factors which would have contributed to a more rigorous examination of the research questions within this thesis. These limitations are detailed in the preceding chapters and have been summarised here.

Limitations include a more accurate measure of the parental body shape, ideally through the data collection of anthropometrically measured height, weight and WC. However, due to limitations of budget and time, this was not possible – nor was it identified as a research priority as the focus of the NWAHS was primarily on the epidemiology of chronic conditions. The pictograms were also used in the Danish Nurse Cohort Study to determine familial predisposition to obesity [252]. Pictograms were originally formulated to determine the body shape of the parents of both biological and adoptee parents when measured and/or reported information was not available. for example when parents have died [9]. Based on their 1979 study, Sorensen et al [101] argue that reports of body weight are less costly and so enable epidemiological studies of obesity to be undertaken, whilst acknowledging that they are less accurate than measurements. Body shape at age 40 allows for study participants to recall consistently across their parents' life span, acknowledging that while midlife parental height may be reported quite accurately, actual midlife parental weight would be less easily recounted. A number of studies [9,101,200,266,267] have shown pictograms to be valid measure of recall regarding parental body shape.

7.5 Implications

Obesity tends to run in families – together with its associated implications for poorer health for children, adolescents and adults, as well as premature mortality. This section discusses the implications of the research undertaken for this thesis.

7.5.1 Mortality and obesity

As discussed in the published paper regarding mortality and obesity (Chapter 4) and addressing the first research question, there is an association between obesity and premature mortality, particularly if the excess weight is carried centrally in the body. The research in this thesis contributed further to the literature on this type of association which is still at an early stage, due to the ABSI only being developed and then published in 2012 [7] followed by validation within a British population in 2014 [189]. The study undertaken in Chapter 4 shows that those with the highest BMI and WC, using the ABSI, had more than two and a half times the mortality risk compared to those with the lowest BMI and WC. The proportion of all-cause, and CVD- and cancer-related mortality steadily increased from Quartile 1 through to Quartile 4 of the ABSI. This confirmed results of an overall study (not divided by degree of obesity) of all-cause, and CVD- and cancer-related mortality in a Netherlands population [6]. This study provided similar results to a study of ABSI quintiles of only all-cause mortality of an Iranian population [268]. This information about the ABSI is the first to be published using an Australian population. It was also the first to provide valuable primary and secondary (subsequent) cause of death information, as well as the differences for men and women.

Mortality is implicit as the end-point of the time axis in the framework of obesity aetiology (Figure 2.1). Also within this framework and discussed in Section 2.5, the development of obesity at different stages across the lifecourse from pre-birth, through to early childhood and adolescence to adulthood and the association with mortality, has been examined since the 1970s [269]. To begin with the earliest contributing factor for childhood obesity as highlighted in Section 2.5.3.1 and its effect on later morbidity, one study examined the effect of maternal obesity during pregnancy and later development of CVD in their adult children, using a Scottish cohort study established in 1950 and following participants to 2013 [270]. The authors reported that adult offspring whose mother was obese (BMI>30) had an increased risk of all-cause mortality (HR 1.35, 95% CI 1.17-1.55) when compared to normal weight mothers. In addition, these adult children also had an increased risk for a cardiovascular event (HR 1.29, 95% CI 1.06 to 1.57) and a higher risk of adverse outcomes such as other cerebrovascular disease and peripheral artery disease [270].

Similar implications can be found with people who were obese as children, as highlighted in Section 2.5.3.2, with Llewellyn et al [271] reporting that this risk factor moderately increased the risk for both diabetes and coronary heart disease (CHD) developing in adulthood (OR 1.70, 95% CI 1.30-2.22 and OR 1.20, 95% CI 1.10-1.31 respectively) and also for a range of cancers, but not for breast cancer or stroke. However, the authors stated that childhood BMI could not be considered a good predictor of adult health conditions, highlighting that children aged 12 years or over who were overweight or obese only accounted for 31% of diabetes and 22% of CHD and hypertension in the future in their study [90]. Although these only account for approximately a quarter of the population with these conditions, with the remainder

only developing these obesity-related conditions in adulthood, it would seem that there are still implications for public health action targeting overweight or obese children to prevent the later development of these chronic diseases.

Further, as emphasised in Section 2.5.3.2, the adverse influence of obesity in young adulthood has been reinforced by Hirko et al [272] in their follow-up of over 75,000 people from the south-east US, who were enrolled in the study in 2002 and 2009 at ages 40–79 years and followed through December, 2011. The authors reported higher mortality rates of 19% (95% CI 12-27) and 64% (95% CI 52-78) for people who reported overweight and obesity (BMI) at age 21, respectively [272]. Similarly, a British cohort study followed 2547 women and 2815 men who were recruited in 1946 – mortality follow-up of 26 to 60 years started in 1972 [273]. The authors reported a consistent U-shaped relationship for mortality with both underweight and overweight/obese people aged 20 years and over [273]. There was a similar relationship also for adolescent females (aged 15 years): compared to the mean BMI, underweight females who had a low BMI (2 SD below the mean) had a HR of 2.96 (95% CI 1.26 to 6.97), while overweight/obese females with a high BMI (2 SD above the mean) had a HR of 1.97 (95% CI 0.85-3.28) [273].

Research on obesity and mortality in this thesis used height, weight and WC (Chapter 4). The majority of studies reviewed examining obesity and its association with mortality were based on BMI only. There has been much discussion regarding reported findings of a protective influence of overweight and mild obesity from Flegal et al [37], much of it concerning inadequate adjustment for weight loss and higher mortality from ageing, smoking and chronic illness [274]. The limitations of BMI to

provide an accurate measure of body shape prompted the interest in the growing trend for measures that incorporated central adiposity, such as WC, leading to further exploration of the ABSI as a predictor of mortality (see Chapter 4). Ahima and Lazar [274] highlight the importance of such incorporated measures, arguing that many factors are involved in the prediction of optimal weights for health and mortality, including age, sex, cardio-metabolic fitness, genetics, pre-existing disease and other health-related risk factors. In their review paper of the associations of BMI, WC, WHR and WHtR with mortality within cohort studies, Carmienke et al [46] stated that both measures of general obesity and abdominal obesity were strongly associated: BMI and WC showing U- or J-shaped associations predominantly, and WHR and WHtR showing positive liner associations – all measures showed that upper quantiles were related to increased risk of premature mortality.

While ultimately mortality cannot be avoided, the enhancement in quality of life through healthy ageing is an important goal. As part of this public health/medical objective, a reduction in premature mortality through the prevention of multiple obesity-related chronic conditions is warranted and one place to tackle this is via a discussion between health practitioner and patient.

7.5.2 Individual level implications: the "elephant" in the room - discussing overweight and obesity in a clinical/ health promotion setting

Much of the challenge to reduce obesity is placed in the hands of the health sector; primarily doctors and allied health professionals, who use a range of options from weight loss counselling, management of obesity-related conditions and the use of pharmaceuticals to control appetite, through to the most extreme cases who may benefit from bariatric surgery to reduce food consumption. Opportunities for weight

counselling may be more serendipitous than planned and can become the 'elephant in the room'; ie, something obvious that needs discussing [275]. In November 2016, the Council of Presidents of Medical Colleges (CPMC), representing over 100,000 Australian doctors, convened a National Health Summit of stakeholders to discuss "the health crisis of obesity" and the growing trend of obese people to mistakenly perceive themselves as being a healthy weight. Its aim was to develop potential approaches to halting obesity trends in Australia; and if possible even, to reverse them. Its six point plan was aimed at both the individual and population level (see Figure 7.1) [276]:

(i) **A chronic disease, not a lifestyle choice***: recognise that obesity is a chronic disease with multiple causes, and remove stigma, focus on prevention (especially in children) and maximise access to optimal disease management.*

(ii) **Education and upskilling***: build health professional capability in the prevention and management of obesity by upskilling through education and training, provide disease management toolboxes, and fund clinical research to identify new evidence-based prevention and treatment strategies.*

(iii) **Health professionals leading by example***: encourage health professionals to lead by example with initiatives across universities, hospitals and health services, including reducing access to sugar-sweetened beverages and processed foods on site, and promoting a greater variety of fresh foods and water as healthier choices for staff, students and visitors.*

(iv) **Pre-conception planning:** *focus on prevention before and early after birth; provide obesity prevention and care for all women as part of routine perinatal care (and women and men before conception), and provide support services after birth via a nationally funded strategy.*

(v) **National obesity prevention strategy***: develop and adopt a new comprehensive evidence-based strategy including a focus on diet, exercise and healthy cities (bringing health expertise to the table to maximise the benefits of new urban planning).*

(vi) **Stronger voluntary regulation and new legislation:** *incentivise voluntary food reformulation and support food ratings; reduce unhealthy food marketing to children; reduce the consumption of unhealthy high sugar beverages and foods by implementing a sugar-sweetened beverage tax, and use the funding to support the entire plan.*

Figure 7.1 Six-point plan for action on obesity

Source: The Council of Presidents of Medical Colleges; adapted from the National Health Summit on Obesity summary [276,277]

As summarised in Item ii above (Figure 7.1), primary care providers are in a prime position to assist with health-promoting behaviour changes. In a review of publications regarding this subject, Rose et al [278] reported that most of the studies demonstrated a positive effect of provider weight loss advice on patient weight loss behaviour. The overall mean weighted effect size for patient weight loss efforts in a random effects meta-analysis had an OR of 3.85 (95% CI 2.71-5.49; p=0.01) but no difference for obese health care consumers alone versus mixed samples [278].

Arising from the four research questions and the resulting publications undertaken for this thesis, there are three potential ways to support the message from health professionals that excessive weight is unhealthy and that action should be taken to reduce weight: (1) asking the patient about their perception of their weight (before actually taking measurements to eliminate responder bias) to determine if there is misperception, particularly if there is an under-estimation of their weight; (2) asking the patient about the body shape of their parents (using pictograms regarding their mother and father); and (3) highlighting the link between obesity and mortality, as already discussed, using inexpensive equipment to obtain measures (a stadiometer for height, scales for weight and a tape measure for WC) to calculate their ABSI z-score through the use of online calculators (http://elsenaju.eu/Calculator/Body-Shape-Index-ABSI.html). A value of 0.0 indicates that this score is the same, on average, for all people aged the same and of the same gender; higher ABSI scores indicate a higher relative risk of death [279]. ABSI z-scores have been classified into five risk categories: very low (<-0.868); low (-0.868 to -0.272); average (-0.272 to 0.229); high (0.229 to 0.798) and very high (>0.798) [279].

7.5.3 Summary of findings and implications regarding perception of body shape

As highlighted in the third published paper (see Chapter 6) and addressing the third and fourth research questions, self-perception about body shape and weight is pivotal in initiating and maintaining healthy dietary and physical activity behaviours, including body weight management. This research is the first study to date that examines the association between parental body shape and weight perception [280].

This study found similar but slightly higher results to those recently reported in the US (using measured height and weight) and Great Britain (using self-reported height and weight) where over half of participants misperceived their weight status [146,261]. The present study also found similar results regarding misperception of weight status (males 51.2%, females 57.4%). Further, the present study also found that men are more likely than women to be optimistic about their weight; that is, to under-estimate their weight and consider themselves to be lighter than they actually were. This finding is in line with other self-perception studies [129,146]. Of those who considered themselves to be 'normal' weight, men were more likely than women to be overweight; again, a similar finding to an earlier US study that also only used self-reported measures [137].

Misperception of weight has implications for future generations because not only do adult offspring misperceive their weight, but as parents they also often misperceive the weight of their children. This has implications for a lack of action to address their obesity which then affects health behaviours, risk regulators such as cultural and general norms, and the influence of the household and wider community, as highlighted in the obesity aetiology framework (Figure 2.1). As women are generally

the primary caregiver within a family, maternal perception of weight status of their children is important to avoid unhealthy weight gain in their children. However misperception has been shown to be a concern, particularly where children are considered to be normal weight when they are in fact overweight or obese. A 2013 review found that approximately 60% of parents misperceived their children as normal weight, and that parents of younger children (aged between 2-6 years) were more likely than parents of older children to under-estimate weight [281]. A more recent review found the rate of misperception to be 50.7% (95% CI 31.1-70.2%), dependent upon the child's age and BMI [282].

Factors contributing to this misperception have been examined. In a study of eight European countries (Belgium, Greece, Hungary, the Netherlands, Norway, Slovenia, Spain and Switzerland), measurements of the weight of children aged 10-12 years were compared to their own assessment and that of their parents: 42.9% of children and 27.9% of their parents misperceived the children's weight as normal when they were actually overweight/obese [283]. In addition, children and parents from Eastern and Southern region countries were more likely than Central and Northern countries to under-estimate their children's weight, as were parents who were also overweight/obese themselves (OR 1.81/1.78, 95% CI 1.39-2.35/1.22-2.60 respectively), parents of boys (OR 1.32, 95% CI 1.05-1.67) and unemployed parents (OR 1.53, 95% CI 1.22-1.92) [283]. A 2012 study among mothers of Portuguese children, who have some of the highest rates of overweight and obesity in Europe, found similar rates of misperception (62%); those who correctly classified their children as overweight/obese were more likely to be the most-educated [284].

The time axis in the obesity aetiology framework (Figure 2.1) starts at birth and moves through childhood, and into adulthood to mortality. Self-perception of body shape/weight starts at a relatively young age, and a recent study showed that health messages regarding the need to maintain a healthy weight are being heard [285]. A study of perceptions about overweight and obesity among US children aged 8-18 years showed that almost half (47.1%) over-estimated the rate of overweight and obesity, with the vast majority (91.1%) reporting that they felt it was important not to be overweight – an encouraging sign noted by the authors [285]. In a study of overweight/obese English teenagers, 60% (males 53%, females 68%) correctly identified themselves as such, while 39% (males 47%, females 32%) considered themselves to be 'about the right weight' or 'too light' [286].

Misperception can also be in the direction of over-estimation – a concern in itself, as reported by Sutin et al [287], who found in their US study of 16 year old adolescents between 1996 and 2008, that those who over-estimated their weight were more likely to be obese in later years and more likely to be male (OR 1.41, 95% CI 1.22-1.64; males OR 1.89, females 1.29). However, in a 2005-2012 year study of adolescents aged 13 to 15 in England, it was encouraging that over-estimation of weight was uncommon (7% of normal weight teenagers – males 4%, females 11%) [286].

Misperception of overweight or obese among children and adolescents is a concern because of the lack of recognition of the potential health issues of being too large or the misapprehension of obesity which can lead to unhealthy dietary and physical activity behaviours, such as discussed in Section 2.5.3 (*Aetiology of obesity*). Obesity-

related conditions for young people include hypertension, sleep apnoea, poor glucose tolerance, hyperinsulinaemia and a raised risk of type 2 diabetes [288], musculoskeletal conditions [289] and depression [290]. These problems can start in childhood/adolescence and persist in adulthood, and that there is a cycle of obesity contributing to these conditions as well as the condition(s) contributing to increasing obesity. The ongoing succession of health problems with unhealthy weight sets up a potential lifetime of management of chronic diabetes, cardiovascular, cancer, musculoskeletal and mental health conditions involving medications, loss of productivity and quality of life for young adults as they age [288].

As discussed in Section 2.2 (*Prevalence and trends of obesity*), the prevalence of obesity is increasing and particularly so for women. There is a risk that this will continue to increase the numbers of people who view themselves as 'normal' weight because of the acceptance of heavier weights and bigger body shapes as highlighted in Chapter 6. While there are issues with more women than men suffering from eating disorders such as anorexia nervosa and bulimia nervosa (64% of those with an eating disorder in Australia are females [291]), the number of people who are underweight are low (1.7%) when compared to those who are overweight or obese (35.3% and 27.5% respectively) [241].

These results suggest that for some people, their perception of what constitutes an unhealthy body shape requires some adjustment, away from what they may consider 'normal' based on the size of their overweight/obese parents and others around them. Results from this study may assist with primary care-based and health promotion programs aimed at these people who are unrealistic about their unhealthy

weight, who may not be taking any steps to improve their diet or to exercise more to reach a healthy weight.

7.5.4 Summary of findings and implications regarding parental body shape

As discussed throughout this thesis and already explored with the second research question, there is a strong association between parental obesity and offspring obesity, due to both genetic and environmental factors, and that unhealthy weight and behaviours are more likely to be passed to the next generation. The implications of the research undertaken for this thesis underline a need for a break in the cycle of familial obesity so that future generations can progress from pre-birth to adulthood without the burden of obesity-related conditions that impact on themselves, their families and the wider society in terms of health care, quality of life and premature mortality.

The research contained in Chapter 5 contributed to the literature on the association between parental body shape and adult offspring body shape by supporting findings of a stronger maternal influence from recent British and Irish studies [56,105]. This study was the first in Australia to examine the association between parental body shape and adult offspring body shape. Further, it incorporated three measures of central adiposity (WC, WHR and WHtR) where the majority of studies only use BMI [56,59,115] and WC [246,292]. It also looked at adult offspring rather than younger offspring [293]. Research on parent and adult offspring body shape from 2012 in a Scottish population reported that maternal BMI is the significantly stronger influence on their daughters' adult BMI whereas both parents influence sons' adult BMI equally [110]. The research provided in Chapter 5 found different outcomes of adult

offspring weight for daughters and sons when examining multiple measures of body weight/shape. In this Australian population, of parents who were an unhealthy weight at age 40, mothers were the stronger influence on daughters than fathers (using BMI and WC), while fathers were the stronger influence on their sons than mothers (using BMI). Mothers of an unhealthy weight at age 40 were also stronger influences than fathers on their sons using WHR and WHtR.

The intergenerational association of obesity between parent and child at earlier ages has also been investigated in recent years, including a study of mothers from the 1958 British birth cohort and their young children which found a 50% increase in overweight/obesity prevalence in the next generation over time (1965 to 1991) [168]. In a longitudinal study of obese Swedish children at ages 7 and 15 and their parents, Svensson et al [294] reported that maternal obesity was strongly correlated with obesity at age 7. For adolescents, a stronger correlation was reported with obesity in both parents at age 15 (higher for boys) [294]. Similar to results reported in Chapter 5 for adult offspring, a study of English children found that having two overweight parents increased the risk for childhood obesity (OR 12.0, 95% CI 7.2-20.1, p=0.01), while having two obese parents was associated with an even higher risk (OR 22.3, 95% CI 10.3-48.4, p=.01), and this was independent of age, sex, ethnicity and socioeconomic status [295]. Once again, maternal BMI showed stronger associations with their child's body size than paternal BMI; the associations were the same for sons and daughters but increased with age [295]. Similar to the results reported in Chapter 5, a study of the association between BMI, DXA-(Dual-energy x-ray absorptiometry) derived fat mass (total as well as trunk and leg fat mass) and serum leptin between young adult daughters and their parents reported a stronger

association with their mothers [296]. Maintaining or increasing overweight and

obesity in mothers has also been shown to be a strong factor in adolescent children's

unhealthy weight for both sons and daughters [297].

A Finnish study of adolescents and their parents was undertaken both at the time

before the child was born and then when they reached 16 years of age [298].

Jaaskelainen et al [298] reported that both parents being overweight at both times

posed a major risk for their children also being overweight/obese, with a higher OR

for the father-daughter relationship of 5.58 (95%CI 3.09-10.07), followed by the

mother-son relationship (OR 4.36, 95%CI 2.50-7.59), then the relationships for

mother-daughter (OR 3.95, 95%CI 2.34-6.68) and finally father-son (OR 3.17, 95%

1.70-5.92). This is in contrast to results reported in Chapter 5 of a stronger mother-

daughter association regarding body shape, and which may be due to differences of

age and ethnicity. Han et al [299] also found stronger associations in BMI between

mothers and their daughters, and fathers and their sons, reporting that mothers

whose BMI was either <25 or ≥30 provided the greatest heritability estimates for

BMI. The authors also reported increased ORs of 10.25 for a high WC (≥102 cm for

men, ≥88 cm women; 95% CI 6.56-13.93), 3.03 for angina and/or myocardial

infarction (95% CI 1.55-5.91) and 2.46 for metabolic syndrome (95% CI 1.22-4.57)

for those offspring who had two obese parents, compared with those without obese

parents [299].

It can be seen that the cultural environment, lifestyle preferences, together with

genetic and behavioural factors within the shared family and/or household

environment such as excessive intake of sugar both in food and drinks, larger portion

sizes and decreased levels of physical activity due to increases in the use of technological devices [300,301], can explain much of the similarities between parental and offspring body shape. However, a study of participants from the three cohorts – Original (1948), Offspring (1971) and Third Generation (2002) - of the Framingham Heart Study found no change in the effect size of a BMI genetic risk score over these 54 years, despite also reporting a stronger association with parental obesity and a BMI increase of 1.49 kg/m^2 and 2.09 kg/m^2 among Offspring and Third Generation participants respectively [302]. This suggests that the increases in weight in later generations and more recent times may be due to environmental factors rather than genetic factors which remained stable.

The results of the study provided in Chapter 5 suggest that general practitioners and physicians in primary health care, as well as health professionals in health promotion settings, could utilise pictograms to identify parental obesity as a possible risk factor for obesity among health care consumers attending their practices. This information could then be used to initiate a conversation about pursuing healthy weight targets, in an effort to help address the public health concerns regarding our increasing rates of obesity and related co-morbidities.

7.5.5 Summary of findings and implications regarding measurement of obesity in health care

Following discussions with health professionals regarding self-perception of weight and norms based on long term exposure of parents with an unhealthy weight as role models, the next step is to accurately measure body shape to see where the person is along the continuum of weight and fat distribution.

A recent study refers to the term 'overfat' as the "presence of excess body fat that can impair health, even for normal weight non-obese individuals" (p1) [303]. The authors highlight that the prevalence of abdominal fat in 30 of the world's most developed countries is higher than global estimations, which is a concerning increase [303]. In recognition of the importance of monitoring central adiposity, guidelines for obesity management in general practice developed by the National Health and Medical Research Council (NHMRC) recommend that doctors document BMI and WC during practice visits, in efforts to prevent and manage obesity [ref].   A study of 78 Australian general practices between July 2011 and December 2013, found that only 22.2% and 4.3% of health care consumers had a documented BMI and WC respectively [253]. In efforts to encourage such weight loss, the NHMRC, in their 2013 report *Clinical Practice Guidelines for the Management of Overweight and Obesity for Adults, Adolescents and Children in Australia,* recommended that "If patients wish to be measured, a combination of BMI and waist circumference or weight and waist circumference should be used." (p48) [2]. This report was commissioned by the Australian Government Department of Health in 2010, in recognition of the health-related consequences of overweight and obesity. Its strategies were framed in the '5As' of the GP's role: Ask and Assess, Advise, Assist, Arrange. These approaches are

just one part of the continuum of efforts that seek to arrest the ongoing trend of people developing obesity.

Krakauer and Krakauer [304], who developed the ABSI in 2012, undertook a further study in 2014 and showed that its values have an almost equal distribution across BMI values, whereas cut-offs for WC were generally correlated strongly with BMI thresholds. They suggest the ABSI may be a better biometric measure in the assessment of obesity and body composition, as it may have wide applicability in clinical settings [304].

7.5.6 Policy level implications - potential solutions to tackle obesity

Of even more importance than individual efforts to halt the obesity epidemic are the efforts of the health community and other stakeholders. In efforts to influence health behaviours as outlined in the framework used as a basis for this thesis (Figure 2.1), the "Measure Up" health promotion campaign was employed in Australia in 2008, across television, radio and print media, to link WC and risk of chronic disease [305]. From a telephone survey of approximately 1000 people living in New South Wales, it was reported that the campaign achieved 38% unprompted awareness and 89% prompted awareness [305]. It was judged to be a success as knowledge of the correct waist measurement increased five-fold, when adjusted for demographic characteristics, despite no significant changes in reported levels of physical activity or fruit and vegetable consumption [305]. It is difficult to determine if knowledge of the importance of central adiposity, as a health-related risk factor, translated into actual waist measurements being made. Generally people have limited knowledge about their own waist measurement, even though they recognise it could be useful to know

with regard to their health [306]. As a comparison, in an examination of the validity

of self-reported height and weight of participants of the NWAHS, about half

accurately reported their height (49.6%) and their weight (51.8%) [235].

As discussed in Section 2.5.5.1 (*Aetiology of obesity – Risk Regulators*) and in the

aetiology framework (Figure 2.1), it is recognised that there are many factors

impacting on obesity. Governments often run health promotion campaigns

encourage healthy behaviours, as part of a multilevel approach required to tackle the

obesity epidemic, as well as strategies aimed at reducing obesity and promoting

healthy eating and physical activity using the built environment, the local food

environment and commercial messaging. Through recognition of distorted

perceptions of 'norms' of overweight and obesity provided by parents who are an

unhealthy weight (Chapter 5) and also within the wider community, together with

weight misperceptions that may be corrected through simple anthropometric

measurements in a health care setting as discussed in Section 7.5.2.3 (*Measurement of*

obesity in health care) or indeed at home, individual and whole-of-population

strategies are needed to address the issues that are linked to decision-making by

individuals. One initiative is the 2013 *Health in all Policies (HiAP)* approach from the

World Health Organization which has been defined as:

> *"... an approach to public policies across sectors that systematically takes into account*
> *the health implications of decisions, seeks synergies, and avoids harmful health*
> *impacts, in order to improve population health and health equity."* [307]

The policy seeks collaboration across multiple government sectors such as housing,

nutrition, education, transport and water/sanitation, and focuses on health equity

rather than public efficiency, coherence and coordination which is the focus of a

whole of government approach. This WHO initiative has been adopted by a number of governments, including South Australia whose projects include the Premier's Healthy Kids Menu Taskforce to help reduce child obesity in the State [308].

As highlighted in Section 2.5.5 (*Risk regulators*) and based on the framework provided (Figure 2.1), strategies from policy/government organisations and other stakeholders at both a State and national level could include health professionals leading by example across health centres and academic institutions, a greater focus on pre and post birth for women and their families, greater emphasis on healthy cities and towns through urban/town planning as part of a national obesity prevention strategy, and stronger voluntary regulation and new legislation such as updated food and beverage labelling and marketing (see Figure 7.1). Murrin et al [105] argue that modifiable risk factors, such as physical activity, may be more successful if targeted at a family level rather than an individual level.

In summary, weight reduction strategies are being developed at all levels of society; at the primary care level, they are directed towards the need for individual change. The use of a simple prediction tool for premature mortality risk such as the ABSI incorporating relatively easily measures as height, weight and WC (as discussed in the publication encompassing Chapter 4) by a general practitioner may further assist with the uptake of healthier dietary and physical activity behaviours. The publication comprising Chapter 5 highlighted the usefulness of using pictograms of parental obesity by primary care physicians as tools to start conversations with health care consumers who have an unhealthy weight, about the need for adopting healthier lifestyles. This may lead to valuable discussions about possible misperceptions of

their weight among health care consumers, particularly of interest for those who under-estimate their excess weight and who do not realise that there is a need for change, as discussed in the publication comprising Chapter 6. There is global consensus that obesity prevention requires efforts that are multifactorial that are aimed at multiple sectors. However, it is considered that improving self-perception of weight among the population, through individual and clinical approaches, and through health promotion programs, is likely to be a useful contribution.

7.6   Future research

There is a great deal of ongoing research being undertaken in the field of obesity, both for adults and children. The National Institute for Health and Care Excellence (NICE) recommend, from an epidemiological viewpoint, that obesity-related data, with anthropometric measures if possible, continue to be collected, particularly among the most vulnerable groups within the population including children. Carmienke et al [46] similarly suggest that measures of central adiposity be used in association with measures of general obesity, to provide independent information regarding risk.   The importance of this has been highlighted throughout this thesis. Analysis of these data allows for trends to be identified and the magnitude of the issue to be monitored [309]. In recognition of this, Krakauer et al [310] recently built on their mortality predictor tool (the ABSI) by adding hip circumference and found that it demonstrated a U-shaped relationship to mortality. They then developed the Anthropometric Risk Index which outperformed height, BMI, ABSI and a hip index as a predictor of mortality in a US population, but which requires additional testing in other populations [310]. Indeed, as a result of the paper comprising Chapter 4 being published, a request was received in early August 2017 from Dr Jesse Krakauer, an

endocrinologist from the Metro Detroit Diabetes and Endocrinology Center, and Assistant Professor Nir Krakauer from the Department of Civil Engineering, City College of New York, to collaborate on further research regarding the incorporation of central measures of adiposity.

It is suggested that future research could include a study to investigate how weight perception can be improved so that people recognise the imperative to improve their health and lead a more active and longer life. This could be conducted on an individual level, through consultation with health professionals on a one to one basis with tailored advice and support; or at a population level through messages such as "Measure up". Of equal benefit would be a qualitative investigation investigating the reasons people (both adults and children) misperceive their weight.

There is also growing recognition of the importance of generation studies which may, in coming years, help to identify successful interventions that diverted the journey of obesity inheritance for later generations. Genetic studies are being increasingly used – not only to identify the genes that are involved in the development of obesity, but also to determine the mechanisms by which they accomplish this. Haire-Joshu and Tabak [12] argue that the pathway for halting the intergenerational obesity epidemic requires the additional discovery and development of evidence-based interventions that can perform across multiple dimensions of early life influences. There is indeed a large network of roads leading to the goal of reducing the global epidemic of obesity, both in developed and developing countries, with some evidence that suggests that the trends are slowing – an encouraging note.

7.7   Conclusion

To summarise, measurements that include central adiposity provide better information regarding risk of premature mortality from obese-related morbidities. A significant risk factor for obesity is intergenerational transmission via parental obesity, through a combination of genetic, biologic, cultural, social and environmental influences. This thesis has investigated a recently developed obesity-related measure of premature mortality that incorporates WC with height and weight, using an Australian population for the first time, and found that those people who had the heaviest weight and greatest WC had almost two and a half times the risk of all-cause mortality compared to those with a healthy weight. It has contributed to existing evidence regarding the association of parental midlife body shape through recall via pictograms by their adult offspring, with the body shape of the adult offspring using four measures (BMI, WC, WHR and WHtR) in the first study of Australians. This study found that parental body shape increased the risk of obesity, particularly for daughters. It has provided the first study, to our knowledge, of evidence relating to the association of parental body shape at midlife with self-perception of weight by adult offspring, finding that over half of participants misperceived their weight status, and that having an overweight or obese mother increased the risk of their obese adult offspring underestimating their weight status.

Claims that obesity is not a communicable disease have not been borne out by recent cohort studies of increasing obesity within families, and across generations. There is a need to break the cycle of unhealthy weight from maternal and paternal obesity, and the adverse influences that increase the risk of their children being overweight or obese from infancy, through early childhood, adolescence and adulthood.

Regarding the nature and impact of Australia's obesity problem, an emphasis on the complexities involved in undertaking a long-term and multi-generational shift in culture around obesity, and the need for a collaborative, co-ordinated and multi-level approach by a number of levels in society, including governments and industry, together with communities and individuals, would be beneficial.

This thesis has underscored the importance of accurate self-perception of overweight and obese, as the first step in making lifestyle changes to reduce individual mortality risk and hopefully reduce the risk of future generations being obese by acting as role models for behaviour change. The use of pictograms of parents in primary care settings, together with measures such as the ABSI that incorporate central adiposity, predict mortality risk and which may challenge inaccurate perceptions of weight, may initiate discussions between doctors and health care consumers regarding the risk of carrying excess weight, particularly around the waist. It is hoped that this, together with a wide range of targeted population-based strategies and policies to raise awareness about accurate perception of weight, may help contribute to a reduction in obesity in Australia and internationally.

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APPENDIX 1: Conference Presentation

Poster: Parental body shape at midlife and its association with adult offspring weight measures

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Parental weight has been shown to be a strong determinant of offspring weight status. This study used cross-sectional self-reported and measured data from Stage 3 (2008-10) of the NWAHS (baseline 1999-2003, n=4056), a longitudinal cohort of Australian adults, to investigate the association between midlife parental body shape and four indicators of obesity and fat distribution. The analysis used pictograms for recall of parental body shape, and measured BMI, WC, waist hip ratio (WHR) and waist height ratio (WHtR) of adult offspring (n=2128). Compared to both parents being a healthy weight, offspring were more likely to be overweight or obese if both parents were an unhealthy weight at age 40 (OR 2.14, 95% CI 1.67-2.76). Furthermore, those participants whose mother was an unhealthy weight were more likely to be overweight or obese themselves (OR 1.50, 95% CI 1.14-1.98). There were similar but lower results for those with an overweight/obese father (OR 1.44, 95% CI 1.08-1.93). The effect of one or both parents being overweight or obese tended to be stronger for daughters than for sons across BMI, WC and WHtR. BMI showed the strongest association with parental body shape (OR 2.14), followed by WC (OR 1.78), WHtR (OR 1.71) and WHR (OR 1.45). WHtR (42-45%) and BMI (35-36%) provided the highest positive predictive values for overweight/obesity from parental body shape. This study showed that in this population, parental obesity increased the risk of overall obesity and central adiposity for adult offspring, particularly for daughters. Pictograms could potentially be used as a screening tool in primary care settings to promote healthy weight among young adults.

Keywords: parental body shape, adult offspring, BMI, central adiposity, longitudinal cohort

APPENDIX 2: Reprints of published papers

> Grant JF, Chittleborough CR, Shi, Z, Taylor AW. **The association between A Body Shape Index and mortality: results from an Australian cohort.** *PLoS ONE.* 2017;12(7): e0181244; doi.org/10.1371/journal.pone.0181244 *(see Chapter 4)*

> Grant JF, Chittleborough CR, Taylor AW. **Parental midlife body shape and association with multiple adult offspring obesity measures: North West Adelaide Health Study.** *PLoS ONE.* 2015;10(9):e0137534; doi: 10.1371/journal.pone.0137534 *(see Chapter 5)*

> Grant JF, Chittleborough CR, Taylor AW. **Parental midlife body shape influences offspring self-perception of weight in a cohort of Australian adults.** *Journal of Obesity and Overweight.* 2016;2(3), 303 *(see Chapter 6)*