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**Individual, behavioural and environmental pathways to  
adolescent obesity.**

**Research Thesis  
submitted for the degree  
Doctor of Philosophy**

**Paola Teresa Chivers**

**School of Health Sciences**

**The University of Notre Dame, Australia**

**June 2010**



*Dedicated to the memory of my  
Zia Grazia*

## **Declaration of Authorship**

This thesis is my own work and contains no material which has been accepted for the award of any degree or diploma in any other institution.

To the best of my knowledge, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

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Paola Teresa Chivers

## Abstract

The longitudinal investigations of the contributions of obesogenic variables to developmental pathways of adolescent obesity were examined. Key obesogenic variables were examined from the extensive database of the Western Australian Pregnancy Cohort (Raine) Study. The data set included variables collected in utero (18-20 weeks), at birth, and at ages 1, 2, 3, 6, 8, 10, and 14 years.

The key research question was: How do individual, behavioural and environmental factors in childhood contribute to weight status at early adolescence? Investigation of this key question examined how factors change over time and their respective influence on obesity, identified critical points in the timing of change, and gender differences.

The conceptual model was framed from an individual, behavioural and environmental perspective, based on Bandura's Social Cognitive Theory. Cross-sectional statistics described the sample and variables of interest. A longitudinal model of BMI from birth to 14 years using linear mixed modelling examined the influence of obesogenic variables on BMI over time and differences across weight status groups. Interrelationships between key variables and BMI at each follow-up and over time were investigated using exploratory structural equation modelling.

Some key findings were that adolescents who were overweight or obese at 14 years followed different BMI trajectories from birth, compared to those of normal weight. There was a difference between weight status groups in the timing of adiposity rebound ( $p < .001$ ) and BMI at nadir ( $p < .001$ ), as well as differences in influence of obesogenic factors. The obese group had the fastest increase of BMI over time ( $p < .005$ ), while the rate of change was faster for females compared to males ( $p < .001$ ). Interrelationships between physical activity and sedentary behaviours were shown and changed across models at age 6 years ( $\chi^2$  (df=22) = 25.036  $p = .295$ ), age 8 years ( $\chi^2$  (df=32) = 33.326  $p = .403$ ), age 10 years ( $\chi^2$  (df=40) = 47.820  $p = .185$ ) and age 14 years ( $\chi^2$  (df=57) = 59.487  $p = .385$ ).

This study showed, within the constraints of available obesogenic variables, the complex interrelationships between individual, behavioural and environmental factors, and their relative importance to obesity from birth through to early adolescence. Weight status is a complex balancing act between positive and negative influences, and an individual's ability (genetic, psychological and environmental) to be resilient to the impact of negative influences. Early childhood was identified as a critical time point for establishing key behaviours that influence later obesity.

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This amazing journey would never have started without the enthusiasm and encouragement of my supervisors Professor Beth Hands and Professor Helen Parker. En route ... Professor Max Bulsara for everything statistical!



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## Abbreviations

ABS	Australian Bureau of Statistics
AFEA	Australian Fitness Education Award
AFL	Australian Football League
BMI	body mass index
cm	centimetre
KEMH	King Edward Memorial Hospital for Women
kg	kilogram
kg/m <sup>2</sup>	kilogram per metre squared
IMQ	Infant Monitoring Questionnaire
IOTF	International Obesity Task Force
LMM	linear mixed model
MAND	McCarron Assessment of Neuromuscular Development
mths	months
NW	normal weight (according to IOTF weight status categories)
OB	obese (according to IOTF weight status categories)
OW	overweight (according to IOTF weight status categories)
PWC170	Physical Work Capacity at heart rate of 170 beats per minute
SEIFA	Socioeconomic Index For Areas
SEM	structural equation model
SES	socioeconomic status
TV	television
VO <sub>2</sub> Max	maximum oxygen consumption
WHR	waist-hip ratio
WHtR	waist-height ratio
yrs	years



## Statistical Notations

AIC	Akaike's Information Criterion
CFI	Comparative Fit Index
CI	confidence interval
$M$	mean
MAR	missing at random
$n$	number of cases (sub sample)
$N$	total number of cases (full sample)
$p$	probability
RMSEA	Root Mean Square Error of Approximation
$SD$	standard deviation
$t$	t-test
TLI	Tucker Lewis Index
$\chi^2$	chi-square
%	percentage

## Glossary

*Absolute aerobic fitness* is the raw score for PWC 170 (American College of Sports Medicine, 2009)

*Adiposity* is a term used to describe the amount of associated fatness and is an excessive accumulation of lipids (*Stedman's Medical Dictionary*, 2006).

*Adiposity rebound* is the age at which BMI increases continuously from its lowest BMI value (nadir) (Rolland-Cachera, Deheeger, Bellisle, Sempe, Guilloud-Bataille, & Patois, 1984).

*Body Mass Index (BMI)* is a proxy indicator of fatness. It is a measure calculated by dividing a person's weight in kilograms by their height in metres squared (Cole, Bellizzi, Flegal, & Dietz, 2000).

$$BMI = \frac{X \text{ kg}}{X \text{ m}^2}$$

*Epigenetic influence* is any heritable influence on genes (Walley, Blakemore, & Froguel, 2006).

*Height* is a vertical measure of how tall a person is, measured in centimetres or metres (*Stedman's Medical Dictionary*, 2006).

*Inactivity* describes the non-participation in a given activity or activities (Hands, Parker, Glasson, Brinkman, & Read, 2004).

*Intensity (activity)* is the ranking of the intensity of activity (light, moderate or vigorous) based on self-report or a compendium of activity type classification (Hands et al., 2004).

*Listwise* is a method for handling missing data. Listwise deletion involves the removal of an entire case (individual) from the analysis if any single value is missing (SPSS for Windows, Rel.17.0.0. 2008; SPSS Inc., Chicago, IL)

*Nadir* (in respect to adiposity rebound) is the lowest BMI value (Rolland-Cachera et al., 1984).

*Obese* is the term ascribed to a person whose BMI is above a certain cut off point using the IOTF standards. These cut-offs are age adjusted for children up to age 18, but for adults obese refers to a BMI  $\geq 30$  kg/m<sup>2</sup> (Cole et al., 2000).

*Obesogenic* is the term used to describe factors that are associated causes with overweight or obese (Quinion, 2002).

*Overweight* is the term ascribed to a person whose BMI is above a certain cut off point, but below the obese cut off point using the IOTF standards. These cut-offs are age adjusted for children up to age 18, but for adults overweight refers to a BMI between 25-29.9 kg/m<sup>2</sup> (Cole et al., 2000).

*Physical activity* is any bodily movement produced by skeletal muscles that result in energy expenditure (Caspersen, Powell, & Christenson, 1985).

*Physical Education* describes school curriculum based physical activity sessions (Hands et al., 2004).

*Physical fitness* is a set of health or skill related attributes that can be measured by prescribed tests and relates to the ability to perform physical activity (Caspersen et al., 1985)

*PWC 170* is a physical fitness test for cardiorespiratory fitness. It predicts the required workload for a heart-rate of 170 beats per minute (Caspersen et al., 1985)

*Relative aerobic fitness* is the raw score for PWC 170 adjusted for body weight (American College of Sports Medicine, 2009)

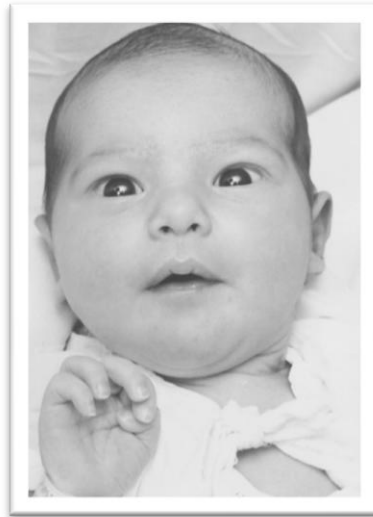
*Sedentary activity* is used to describe activities that require minimal energy expenditure such as screen based activities (Hands et al., 2004).

*School Sport* describes school curriculum based sport sessions (Hands et al., 2004).

*Waist girth* is a measure in centimetres of the circumference of a person's waist (measured in The Raine Study at the level of the umbilicus) and is an indicator of central adiposity (Hands et al., 2004).

*Weight* is a measure in kilograms of the mass of a person (*Stedman's Medical Dictionary*, 2006).

*Weight status* refers to the categories normal weight, overweight and obese which are based on the IOTF cut-points, age and gender adjusted equivalent of  $18 \text{ kg/m}^2$  and  $25 \text{ kg/m}^2$  at 18 years of age (Cole et al., 2000).



## Birth

"Birth is the sudden opening of a window, through which you  
look out upon a stupendous prospect.  
For what has happened? A miracle!  
You have exchanged nothing for the possibility of everything."

Author unknown

## Chapter One

### Introduction

Obesity is a condition of excess body fat accumulation which has serious physical, psychological, health, behavioural, social and economic consequences (WHO, 2005). Over the past decades there has been an explosion in the number of children and adults who are overweight or obese worldwide (WHO, 2006), with similar trends in Australia (Australian Bureau of Statistics, 2009). Most alarmingly, overweight and obese children are presenting with adult-related health disease and once reaching adulthood have an increased risk of premature death and disability (WHO, 2006).

Despite the high level of interest in the *obesity epidemic*, researchers have been unable to determine strong causal factors of obesity. Research teams are considering obesity from numerous perspectives such as prevalence, causation, health consequences and intervention programs from birth to adulthood. Biological, metabolic, behavioural and psychosocial perspectives are used in an effort to identify key factors linked to obesity, with little consensus. Findings are often conflicting due to variation in population groups, measurement methods, population sampling and research designs. Unfortunately evidence is accumulating on the impact of obesity on individual quality of life, increased disease risks, the growing drain on health services, and the mounting associated costs to the individual, community and country.

The causes of obesity are complex and dynamic and research aims to identify its determinants and consequences (Hu, 2008e). Obesity is considered to have developmental origins, whereby foundations for this chronic disease begin early in life (Gillman, 2008). The intra-uterine and early childhood periods are thought to influence later obesity via various inter-related pathways which include maternal behaviours and characteristics during pregnancy, early infant feeding, post-natal

growth, and early childhood behaviours such as diet and physical activity (Hu, 2008e).

While many factors have been implicated in the aetiology of obesity, the identification of causation or consequence is still unclear (Jebb & Lambert, 2000). The more commonly reported factors include adiposity rebound, birth weight, ethnicity, genetics, sedentary behaviour, physical activity, diet, socioeconomic status, parental weight status, sleep behaviour (Skinner, Bounds, Carruth, Morris, & Ziegler, 2004), early infant feeding, puberty (Watkins, Clark, Foster, Welch, & Kasavubu, 2007), rapid weight gain, maternal age, prenatal environment (Blair et al., 2007), birth order, income, parent education (Hallal, Wells, Reichert, Anselmi, & Victoria, 2006), and smoking during pregnancy (Toschke, Ruckinger, Bohler, & Von Kries, 2007).

In seeking to determinate pathways to obesity, researchers have used different underlying models, such as the epidemiological triad (Hu, 2008e) and the ecological model of predictors of childhood overweight (Davison & Birch, 2001). This study is based on Social Cognitive Theory (Bandura, 2001) which provides a theoretical framework for understanding, predicting and altering human behaviour, both at an individual and population group level. Briefly, Social Cognitive Theory describes the complex inter-play of individual, environment and behaviour with reciprocal causality (Bandura, 2001; Davis, 2006; Lindzey, Hall, & Thompson, 1978). It suggests that individuals interact, rather than react with their environment throughout their lives (Bandura, 2001) through complex thoughts and actions, beliefs and competencies, and among social influences and structures. These interactions build an individual's attention, memory, modelling and motivation (Davis, 2006). These life course approaches to investigating obesity, provide for an emphasis on developmental origins of disease, as well as the identification of risk factors at particular life stages (Hu, 2008e).

More recently, research has focussed on multi-factorial approaches to identify pathways to obesity, with a heightened call for longitudinal based research. In

particular, descriptive epidemiology enables an unfolding of patterns and trends over time (Hu, 2008b), particularly in prospective cohort studies. The cohort design is less affected by selection and differential recall bias, provides for periodic collection of data, and is considered the strongest non-randomised study design (Hu, 2008a), although considerations must be made for confounding and reverse causation (Hu, 2008d). Overall, evidence from prospective cohort studies is considered stronger than analytic epidemiological studies (Hu, 2008a).

This study utilised the data from the Western Australian Pregnancy Cohort (Raine) Study. This prospective cohort study began in 1989 recruiting pregnant women from antenatal clinics at Perth's primary specialist obstetric clinic to measure the effects of repeated ultrasounds during pregnancy (Newnham, Evans, Michael, Stanley, & Landau, 1993). It has evolved into an ongoing health research project, collecting environmental, developmental and health information. It is one of the few cohort studies where information has been collected on children from in utero, and now with over 18 years of data, provides a unique source for investigating complex causal pathways to health outcomes (The Raine Study, 2010).

## **Purpose**

Most studies of obesity have used a cross sectional design perspective. Few longitudinal studies have investigated weight status pathways, nor a multi-factorial investigation of the interplay between individual, behavioural and environmental obesogenic factors. This study aimed to address both these gaps in research.

This investigation examined multi-factorial pathways to healthy weight from an individual, behavioural and environmental perspective via a statistical process using cross-sectional and longitudinal analysis. This research involved a longitudinal investigation of the relationship between individual variables with weight status, the phase shift over time of these relationships, interrelationships between variables, and gender differences.



This study aimed to answer questions regarding individual, behavioural and environmental triggers to differences in weight status. It looked at protective behaviours; identified differences between sexes in the interplay of obesogenic influences; and provided an insight into direct and indirect parental, family and community influences, with a particular focus on the effects of physical activity, sedentary activity, physical fitness and motor ability on weight status.

## **Significance**

This study contributed to the knowledge on pathways to obesity using a multi-factorial approach. The focus on individual, behavioural and environmental factors may provide effective mechanisms for the future development of comprehensive rather than simplistic initiatives towards facilitating healthy weight in children, particularly from a health and education policy perspective and program development.

Behavioural and environmental factors related to obesity, such as attitudes to physical activity and the school environment, have received minimal attention, or have been adjuncts of studies. More specifically, parenting styles, which provide early foundations for children's behaviours and the environment they grow within, have rarely been studied (Gibson, Byrne, Davis, Blair, Jacoby, & Zubrick, 2007). However, most significantly, Okely and colleagues (2004) suggest that behavioural determinants may present the most successful way of combating obesity. If this is so, then more emphasis on behaviour and environmental factors is required in research on children's development of obesity.

Many cross-sectional, but few longitudinal studies of weight status exist, and there is a call for longitudinal data to investigate the obesogenic trends over time. "Untangling the time course of these events will require long-term prospective studies with repeated measurements of fatness, diet and physical activity" (Jebb & Lambert, 2000, p.53). Longitudinal studies can help to unravel the

interrelationships and identify factors with similar causal pathways (Parsons, Powers, Logan, & Summerbell, 1999).

The Raine Study's longitudinal database provided a rich array of individual, behavioural and environmental variables and offered a unique opportunity to track the time course of obesogenic factors, investigate the intricate interrelationships of these variables, following a cohort from birth to early adolescence. The characteristics of the Raine Study address the Australian research priorities of regular population monitoring and identifying influences involved in the development of unhealthy weight identified by Baur (2000). In addition, the cohort size, although variable across time points, is large ( $N=2868$  (The Raine Study, 2010)) and provides good statistical power for analysis.

### **Major Research Questions**

The key question this research addressed was:

To what extent do individual, behavioural and environmental factors during childhood contribute to weight status at early adolescence?

In answering this over-arching question, this research aimed to answer the following sub questions with respect to the Raine Study cohort.

What is the relative contribution of individual, behavioural and environmental factors to weight status at adolescence?

What is the relative contribution of individual, behavioural and environmental factors in a longitudinal model of body mass index (BMI)?

Are there any critical phase shifts in the relative contribution of these factors during this age range?

Are there any gender differences over time and across factors?

How do the interrelationships between individual, behavioural and environmental factors affect BMI?

## **Delimitations**

This study involved the use of data collected from birth to age 14 years as part of the Western Australian Pregnancy Cohort (Raine) Study. As a result, this research inherited the limitations of the initial and ongoing data collection processes implemented at the time the data were collected, and for each subsequent follow-up. More specifically related to this research study were the following delimitations.

1. Obesogenic variables available for selection were restricted to those collected in the Raine Study.
2. Time points of data collection limited by the Raine Study, i.e. birth and follow-ups at mean age 1, 2, 3, 6, 8, 10, and 14 years.
3. Selection of variables within the Raine database will be delimited to those classified as individual, behavioural and environmental.
4. Proxy measures will be used as variables e.g. skin folds and BMI for adiposity (waist girth at age 14 years or waist-height ratio); Denver and McCarron Assessment of Neuromuscular Development results for motor competence; pedometer step counts for physical activity levels; television, computer, or screen time for sedentary behaviour (parental report); and parenting scale for parental influence.

## **Limitations**

Additional limitations associated with this study included:

1. Raine Study sample may not be representative of the metropolitan Western Australian child population, due to the nature of recruitment.

2. Tracking of some variables changed across follow-ups (e.g. income brackets changed to reflect inflation, physical activity question wording changed).
3. Reliability and validity (psychometric properties) of self-reported measures cannot be established e.g. parent height and weight.
4. Parent reported observational data (i.e. non-standardized surveys or questionnaires) have no reliability or validity measures (psychometric properties).
5. Data were gathered by parent report prior to the age 14 year follow-up, and then changed to a mix of parent and self-report from 14 years.
6. Sample size limited to the original 2,868 infants at birth, and variations in numbers of participants at each consecutive time point.
7. For the following variables the question format and responses collected were not deemed to be interpretable for use in this study:
  - Childcare attendance from birth to age 6 years.
  - Playgroup attendance from birth to age 6 years.
  - Parent planned family activities at 14 years.
  - Pets at home.
8. Lack of diversity in ethnicity variables, small samples within different ethnic groups, with a high skew toward Caucasian. Two groups were created, Caucasian and non-Caucasian, with the latter a composite group of many nationalities, which may mask true effects between ethnic groups.
9. Diet indicator variables chosen were based on intake of fat (10 question composite score), vegetables and fruit. These variables do not provide an accurate indication of complete dietary patterns in this cohort and may account for weak and null findings, particularly in respect to positive associations between diet and obesity in the literature.
10. Adiposity Rebound. Data collection at follow-ups is in years, with the time point being the average age at that assessment. In some follow-ups there was a large range of ages for collection of data. This is particularly significant in respect to the mean BMI trajectories presented, and the calculation of adiposity rebound, although the clear distinction in time

points still provide a valid assessment of the difference between the weight status categories. The Raine Study did not collect data in years four and five and these are important years for adiposity rebound, although this seemed relevant to the overweight and obese group distinctions only. Notably, these results may underestimate the occurrence of adiposity rebound because of this limitation (Chivers et al., 2010).

11. Structural equation modelling (SEM). Although a model was proposed during the concept stage of this current research with SEM in mind, the model was specified after the Raine Study data collection. Therefore there is the likelihood that some key variables (to the current research) have been omitted from the original Raine Study design (Kline, 2005).
12. SEM. An exploratory approach was taken and hence findings reported are only a representation of what was found in this cohort sample, and not necessarily of population behaviour. For this, confirmation with either a hold-out sample (internal replication) or another similar cohort group (external replication) would be necessary (Kline, 2005). However the purpose of SEM in this study was to use these models to diagrammatically articulate the interrelationships occurring in concurrence with other statistical findings.
13. SEM. Only model building was performed using a data set with mean replacement of missing data. This enabled the use of theoretically sound single step model building (adding pathways) using modification indices.

### **Publications Resulting from this Thesis**

Over the course of this research program, aspects from this thesis have been accepted for publication. They are listed below, with full PDF articles attached at Appendix A.

**Chivers, P. T.,** Hands, B., Parker, H., Beilin, L. J., Kendall, G. E., & Bulsara, M. (2009). Longitudinal modelling of body mass index from birth to 14 years. . *Obesity Facts*, 2, 302-310.

**Chivers, P.,** Hands, B., & Parker, H. (2009) The role of physical activity is different for normal weight, overweight and obese 14-year-old adolescents. *Journal of Science and Medicine in Sport, 12(6)*, e137-138. Seventh National Physical Activity Conference, Brisbane QLD. October. doi:10.1016/j.jsams.2009.10.286

**Chivers, P. T.,** Hands, B., Parker, H. E., Bulsara, M., Beilin, L. J., Kendall, G. E., & Oddy, W. H. (2010). Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study). *International Journal of Obesity, 34*, e1-8. doi:10.1038/ijo.2010.61

**Chivers, P. T.,** Hands, B., Parker, H. E., Beilin, L. J., Kendall, G. E., & Bulsara, M. (2010). A comparison of field measures of adiposity among Australian adolescents from the Raine Study. *Malaysian Journal of Sports Science and Recreation, 6(1)*, 33-45.



## Toddlerhood

*"The toddler craves independence, but fears desertion."*

By Dorothy Briggs

## Chapter Two

### Literature Review

This chapter provides a summary of key findings related to individual, behavioural and environmental influences on obesity. The prevalence, aetiology, and health consequences of obesity are reported and current knowledge on obesogenic factors related to this study are described. As the sample for this research is drawn from the state of Western Australia, specific Australian and Western Australian studies are reviewed.

#### Obesity

Obesity is a condition of excess body fat accumulation (WHO, 2005). The prevalence of obesity is increasing at profound rates, in both developed and developing countries, with a more recent and disturbing increase in the incidence of obesity in children. Sadly, this epidemic has serious physical, psychological, health, behavioural, social and economic consequences (WHO, 2006).

Some argue that obesity is simply an energy balance problem (Reilly, Ness, & Sherriff, 2007; te Velde et al., 2007), that is,  $\text{energy balance} = \text{energy intake} - \text{energy requirement}$ . Therefore if a person's energy intake is greater than their energy requirement, then there is a positive energy balance and weight is gained. If the opposite occurs, there is a negative energy balance and weight is lost. However, in reality this model is far more complex with diverse interplays of factors in how the intake and expenditure are determined at an individual level (Reilly et al., 2007).

Dietz (1998) makes an interesting point when he says that "it is not clear whether obesity alone or the behaviours that generate obesity are more important determinants of obesity and its complications" (p. 523). Increasingly it seems that genetics, physiology, environmental, lifestyle and cultural factors all play important



and interrelated roles in the energy equation (Dehghan, Akhtar-Danesh, & Merchant, 2005). The interactions between these factors may be different for each individual, and may also affect them in many different ways.

### **Prevalence of Obesity**

Traditionally obesity has been a problem of western countries, but in recent times the prevalence of obesity is rising in developing countries (Lasserre, Chiolero, Paccaud, & Bovet, 2007). The World Health Organization (2006) report that in 2005, globally, there were about 1.6 billion overweight adults (based on BMI  $\geq 25$ ), with at least 400 million adults considered obese (BMI  $\geq 30$ ). In 2007, Dalton reported at least 20 million children under five years of age overweight in the world. Projections for 2015 indicate a 1.5 fold increase in overweight adults and almost a doubling of obese adults (2.3 billion and 700 million respectively). Worldwide about one child in four is overweight or obese with the highest prevalence in ethnic minority and low income groups (Dalton, 2007).

In Australia, in 1995, 45% (5.4 million) of the adult population were overweight or obese, rising almost 10% in 2005 to 54% (7.4 million) of the adult population (Australian Bureau of Statistics, 2007b). More recently, Australia's National Health Survey in years 2007-2008 reported that the adult overweight and obesity rate had increased further to 62% (Australian Bureau of Statistics, 2009). In all national surveys conducted since 1995 the obesity rates have been higher for males at all ages (Gill et al., 2009). Most recently, in 5-17 year old children 17% were overweight and 7.8% obese (Australian Bureau of Statistics, 2009). Nationally, childhood obesity remains a widespread health concern (Gill et al., 2009).

Booth and colleagues (2003) conducted a review of Australian childhood data (aged from 5-17 years) collected independently in 1969, 1985 and 1997. They found a dramatic 60-70% increase in overweight (BMI) from 1985 to 1997. Their comparison of the 1969 and 1985 data showed no increase in prevalence among females, but in males, the prevalence of overweight and obese had risen 60%. They concluded that

significant increases in obesity levels have occurred in the Australian population from the mid 1980s and the increase is accelerating (Booth et al., 2003). This trend is supported by international data which also identifies the 1980s as the key turning point (International Association for the Study of Obesity, n.d.). Other Australian researchers, Olds and colleagues (2004), have until recently, also held this view. Now, based on a review of Australian obesity prevalence studies between 1985 and 2007, they believe that obesity levels in Australia may have plateaued (Olds, Ferrar, Tomkinson, & Maher, 2009), although this viewpoint is contrary to Australian Bureau of Statistics based on the National Health Surveys (Australian Bureau of Statistics, 2009) and Access Economics data comparison between 2005 and 2008 (Access Economics, 2008).

A review of Western Australian data by Hands and colleagues (2001) showed over a sixty year period (1940 - 2000), at three time points (1940, 1974 and 2000) a significant upward trend in BMI, particularly in the 10-12 year old children. Most notably, the largest increase was in the 25 years between 1974 and 2000. The report also notes an overall shift in weight range, with the minimum weights in 2000 (10<sup>th</sup> percentile) higher than the same group in 1974, which the authors suggested corresponded with earlier puberty, a period of major lifestyle changes, and the impact of technology on leisure time activity (Hands, Parker, Blanksby, & Larkin, 2001).

The Western Australian Child and Adolescent Physical Activity and Nutrition Survey reported an increase in overweight and obese prevalence in 7-15 year olds from 1985 to 2003. For boys the prevalence of overweight and obese rose from 9.3% to 21.7%, while in females it rose from 10.6% to 27.8% (Hands et al., 2004).<sup>1</sup>

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<sup>1</sup> Obesity prevalence rates from the 2008 WA Health Survey were yet to be released at the time of finalising this thesis.

## **Health Consequences Relating to Obesity**

In Australia, obesity has overtaken smoking and is the major cause of preventable disease (Australian Institute of Health and Welfare, 2006), supporting a similar trend in the USA (Haslam & James, 2005). Childhood obesity is associated with higher risk of premature death and disability in adulthood (WHO, 2006). With the increasing incidence of obesity in children, there is an increase in health consequences for these children, with typically adult health concerns now being diagnosed in children.

A systematic review from 1997 to 2001 identified the main health consequences of childhood obesity as psychological morbidity, asthma, chronic systemic inflammation, cardiovascular risk, diabetes, and orthopaedic problems. The long term consequences of obesity in childhood included social and economic effects, persistence of obesity into adulthood, increased morbidity and premature mortality, and continued cardiovascular risk (Reilly et al., 2003).

Health consequences are widely reported across the literature and are summarised in Table 1. Those listed are typically seen in the adult population, although they are now also being seen in children.

Table 1

*Summary of Health Consequences Related to Obesity*

Factor	Source
Arthritis	Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; WHO, 2006; Australian Bureau of Statistics, 2007; Olds et al., 2004
Asthma	Krebs, et al., 2003
Bullying and social exclusion	Centre for Community Child Health, 2007; Krebs, et al., 2003; Gillman, Rifas-Shiman, Camargo Jr, & Berkey, 2001
Cancers (colorectal, breast, uterine, kidney)	Access Economics, 2006; Australian Bureau of Statistics, 2007
Cardiovascular disease	Guo & Chumlea, 1999; Buchan, IE., Bundred, PE., Kitchiner, DJ. and Cole, TJ., 2007; Toschke et al., 2007; WHO, 2006; Katzmarzyk, Srinivasan, Chen, Malina, Bouchard, & Berenson, 2004; Centre for Community Child Health, 2007; Access Economics, 2006; Krebs, et al., 2003; Baur, 2002; Gillman et al., 2001; Denney-Wilson, Booth, & Baur, 2003; Australian Bureau of Statistics, 2007; Olds et al., 2004
Diabetes	Buchan, IE., Bundred, PE., Kitchiner, DJ. and Cole, TJ., 2007; Guo & Chumlea, 1999; Dietz, 1998; WHO, 2006; Huus et al., 2007; Centre for Community Child Health, 2007; Access Economics, 2006; Krebs, et al., 2003; Baur, 2002; Denney-Wilson, Booth, & Baur, 2003; Australian Bureau of Statistics, 2007; Olds et al., 2004
Gastro-intestinal disorders	Dehghan, Akhtar-Danesh, & Merchant, 2005; Krebs, et al., 2003; Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Baur, 2002; Olds et al., 2004

Factor	Source
Hepatic disorders	Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Dietz, 1998; Centre for Community Child Health, 2007; Krebs, et al., 2003; Baur, 2002; Olds et al., 2004
High blood pressure / hypertension	Dietz, 1998; Guo & Chumlea, 1999; Access Economics, 2006; Dehghan, Akhtar-Danesh, & Merchant, 2005; Krebs, et al., 2003; Baur, 2002; Olds et al., 2004
Hyperinsulinemia	Dietz, 1998; Olds et al., 2004
Infertility	Dehghan, Akhtar-Danesh, & Merchant, 2005
Lower growth hormone secretion	Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Olds et al., 2004
Mental health disorders (depression, self-image, self-esteem)	Krebs, et al., 2003; Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Buchan, IE., Bundred, PE., Kitchiner, DJ. and Cole, TJ., 2007; Centre for Community Child Health, 2007; Baur, 2002; Gillman, Rifas-Shiman, Camargo Jr, & Berkey, 2001; Dehghan, Akhtar-Danesh, & Merchant, 2005
Metabolic syndrome	(Ford, Kohl, Mokdad, & Ajani, 2005; Huang et al., 2009; Vanhala, Vanhala, Kumpusalo, Halonen, & Takala, 1998)
Orthopaedic disorders	Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Dietz, 1998; WHO, 2006; Krebs, et al., 2003; Baur, 2002; Gillman et al., 2001
Osteoarthritis	Access Economics, 2006
Polycystic Ovary Disease	Dietz, 1998
Respiratory disorders	Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007
Sleep disorders	Dietz, 1998; Centre for Community Child Health, 2007; Krebs, et al., 2003; Baur, 2002

## **Economic cost of obesity**

Obesity within the community has a significant economic cost. From an Australian perspective these include direct costs to the health system (medical services, pharmaceuticals, allied health, research and health administration), indirect costs to the economy (productivity losses, carer costs, *deadweight loss from transfers*<sup>2</sup>, other support services and infrastructure), and non-financial costs (burden of disease). Encompassing these, it was reported that in 2005 the estimated costs to the Australian economy was just over \$3.7 billion (Access Economics, 2006). Unfortunately this is modest compared to the \$21 billion for 2005 reported by the Australian Bureau of Statistics (2007b), which is still well above 2008 cost projections of \$8.3 billion (Access Economics, 2008). If one considers the costs in relation to those who bear them, including lost well-being, then according to Access Economics (2008) the great majority of financial burden, approximately 90% lies with the individual.

## **Aetiology of Obesity**

The aetiology of obesity is multi-factorial and not well understood (Reilly et al., 2007). There are many complex interactions among and between factors, some yet to be defined. There is a complex interplay of family, community, environment, social demographics, genetics, epigenetics, biology, behaviour, diet and psychology, all intricately interwoven in cause, effects and feedback loops.

Global shifts in diet, along with decreased physical activity and increased sedentary activity are seen as the primary attributable factors (Dehghan et al., 2005; Huus, Ludvigsson, Enskar, & Ludvigsson, 2007; WHO, 2006). Obesity has been explained as “a natural biological response to a changed environment and that innate body-weight regulatory mechanisms have been overwhelmed by energy-dense diets and sedentary lifestyles” (Prentice, 2007 p.89). A different perspective observed from

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<sup>2</sup> “Deadweight loss (DWL) from transfers’ – taxation revenue foregone, welfare and other government payments” (Access Economics, 2006 p. iv).

rat animal models suggests that environmental factors and diet are accelerators of obesity, but are not causative (Vickers, Breier, Cufield, Hofman, & Gluckman, 2000).

Years of research have been unable to find clear causes of the increased prevalence of obesity, although in a small number of cases obesity is a genetic medical condition. One of the major difficulties in obesity research is the problem of causation or consequence (Jebb & Lambert, 2000). A summary of the most commonly reported factors identified to be involved in the complex process of obesity is depicted in Table 2.

Table 2  
*Aetiology of Obesity*

Factors reported in the aetiology of obesity	
Adiposity rebound <sup>3</sup> (timing and BMI) (4)	Parental feeding style (4)
Birth weight (4,16)	Parental weight status (1,2,3,4,5,6)
Breastfeeding duration / formula feeding (5,11,16)	Physical activity (4,10,11,12)
Diet / nutrition (4,5,6,10,11)	Prenatal environment (1,15)
Early maturation (5,7)	Rapid weight gain (1)
Ethnicity (4)	Sedentary activity (1,3,4,5,9,10,11,12,17)
Genetics (4,5,13,14)	SES (4,5, 8)
Income (9)	Sleep duration (4)
Maternal age (1)	Smoking during pregnancy (11)
Parent's education (9)	

*Note.* Sources: 1. Blair, et al., 2007; 2. Burke, Beilin, & Dunbar, 2001; 3. Ochoa, et al., 2007; 4. Skinner, Bounds, Carruth, Morris, & Ziegler, 2004; 5. Krebs, et al., 2003; 6. Hediger, Overpeck, Kucumarski, & Ruan, 2001; 7. Dietz, 1998; 8. Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; 9. Hallal, Wells, Reichert, Anselmi, & Victora, 2006; 10. Magarey, Daniels, & Boulton, 2001; 11. Toschke, Ruckinger, Bohler, & Von Kries, 2007; 12. Baur, 2002; 13. Prentice, 2007; 14 Olds, Dollman, Ridley, Boshoff, Hartshorne, & Kennaugh, 2004; 15. Vickers, Breier, McCarthy, & Gluckman, 2003; 16. Watkins, Clark, Foster, Welch, & Kasa-Vubu, 2007; 17 Must & Tybor, 2005.

<sup>3</sup> Adiposity rebound is “the age at which BMI increased following the lowest BMI” p. 478 (Skinner et al., 2004)

Although not the focus of this study, there has been a shift toward epigenetic mechanisms in respect to obesity. There appears to be a sub-group within the population that has a predisposition to increased adiposity, with evidence of heritability (Silventoinen, Pietiläinen, Tynelius, Sorensen, Kaprio, & Rasmussen, 2007; Walley et al., 2006), and that this genetic predisposition is strongly inter-related with environmental influences (Silventoinen et al., 2007; Sorensen & Echwald, 2001). These biological mechanisms are being studied in mice showing a genetic tendency for obesity where effects of obesity accumulate over generations (Waterland, Travisano, Tahiliani, Rached, & Mirza, 2008). Others have shown in rats that fat accumulation is accelerated, but not caused by environmental and behavioural factors (Vickers et al., 2000).

### **Contributing factors of childhood obesity.**

In light of increasing obesity prevalence in children, research focus has swung to this population group, both from a deterministic and preventive perspective. Ochoa and colleagues (2007) identified some predictive variables for childhood obesity in 6-18 year-olds. From most to least influence were family history of obesity; time spent watching TV (indicator of sedentary activity); consumption of high sugar drink; energy intake (diet); and time spent in physical activity (leisure time). In contrast, they reported that breastfeeding, birth weight, and time asleep did not seem to play a significant role. A longitudinal study on children born in 1992, found that, over time, dietary factors (energy intake) did not appear to be a significant factor in children's weight by the age of eight years (Skinner et al., 2004).

It is a common perception that decreased physical activity and increased sedentary behaviour are contributors to obesity. In obese adolescents, physical activity levels have been shown to be lower compared to their age and gender matched counterparts (Ekeland, Sarnblad, Brage, Ryberg, Wareham, & Aman, 2007). It has also been reported that time spent involved in sedentary type activities is inversely associated with physical activity in adolescents (te Velde et al., 2007).



Familial factors are also important. High parental BMI was strongly associated with high BMI in their children (Bell et al., 2007; Huus et al., 2007). The presence of family history of Type II diabetes also increased the risk of obesity (Huus et al., 2007; Watkins et al., 2007). Interestingly, Watkins and colleagues (2007) point out in their study (which is supportive of others), that parents did not recognise that their children were overweight, nor realise the impact over-eating and being sedentary had on their child's obesity. Another interesting aspect was that parents believed excess weight, in their case, was genetic (but not medically the case), and the authors explain this as being more indicative of the perception that parents believed obesity ran in the family.

Further, "families influence food and activity habits" (Baur, 2002 p. S526) and behaviour change, rather than simply diet, is an important step in prevention. Certainly a family based approach has been shown to be more effective in a weight intervention program, than a child focussed program (Golan, Weizman, Apter, & Fainaru, 1998). Considering these intervention effects, Dollman and colleagues (2007) call for family focussed research identifying physical and social aspects of the home environment that may impact on physical activity and diet in children, and therefore relate to obesity.

### **Theoretical Framework – Social Cognitive Theory**

Given the complex array of factors influencing weight status and behaviours, a theory which can explain multiple influences is central to our understanding of obesity, and the method of research employed. Social Cognitive Theory is a theoretical framework for understanding, predicting and altering human behaviour, both individual and group (Davis, 2006). Bandura developed the Social Cognitive Framework from the perspective that human learning occurs through modelling processes and observing others (Lindzey et al., 1978).

Social Cognitive Theory explains human behaviour as a continuous reciprocal interaction between cognitive, emotional and behavioural aspects. It incorporates

the acquisition and maintenance of behaviours through the complexity of sensory, motor and cerebral systems (Bandura, 2001). Figure 1 depicts this relationship of reciprocal causality with respect to this study.

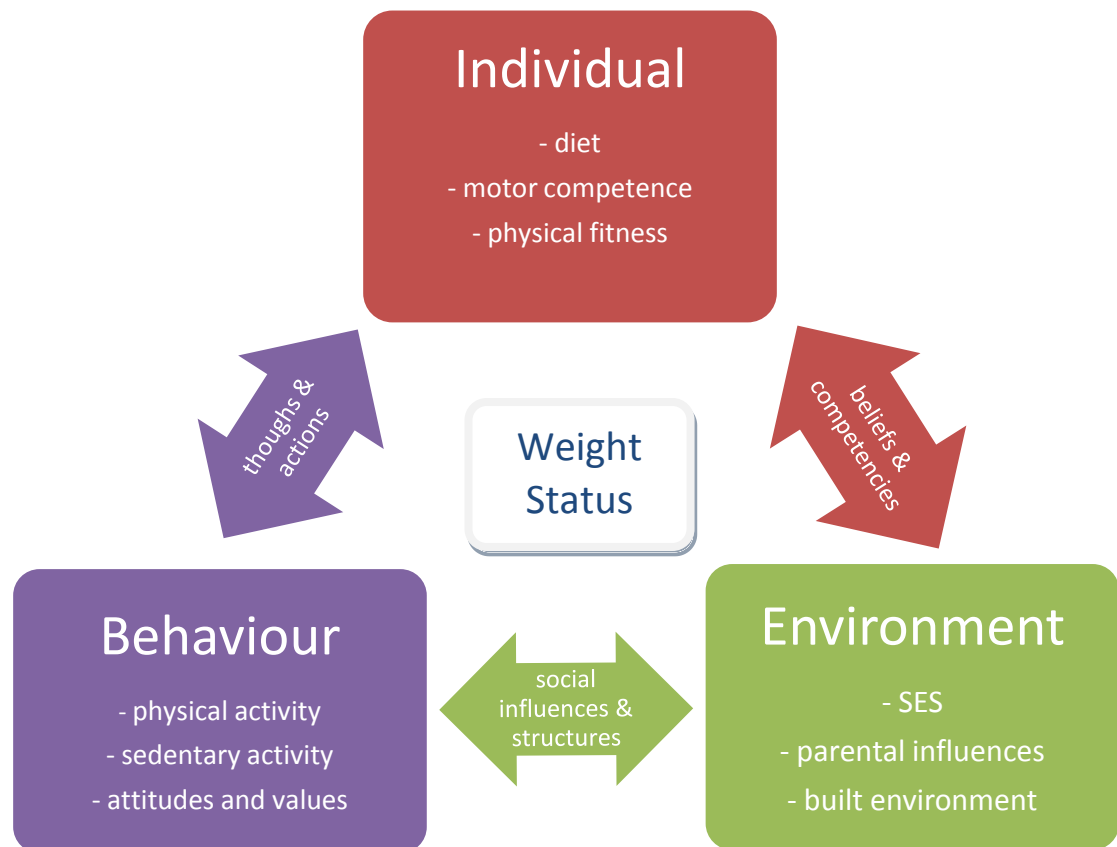


Figure 1. Social Cognitive Model: An obesity research perspective. Adapted from “Social cognitive theory.” by A. Davis, 2006, <http://www.istheory.yorku.ca/socialcognitivetheory.htm>.

Bandura (2001) stresses that it is not the individual passively reacting with his or her environment that explains behaviour, but rather an interaction with their environment through exploration, manipulation and influence. Davis (2006) adds that people will react differently to the same situation, and that the same individual may react differently to the same situation under varying circumstances. This complex interaction of human behaviour involves individual thoughts and actions,

beliefs and competencies, among social influences and structures building upon individual attention, memory, modelling and motivation.

The environment factor incorporates both social and physical environments which are imposed, selected and constructed. It may include factors related to socioeconomic status (SES), education, parenting style, family structure, social networks, social norms and facilities (such as homes and schools). The behavioural factors are those actions of the individual, such as physical activity, sedentary activity, or attitudes and values. The individual factor describes personal attributes of that person (cognitive, affective and biological), such as gender, age, ethnicity, knowledge and skills, motor competence, physical fitness (Bandura, 2001). Even with this overview, it becomes apparent that there are direct and indirect effects among and between the key factors that are constantly changing and evolving over time, and with age.

### **Obesogenic Factors**

“Obesogenic” is used to describe factors that lead people to becoming overweight or obese (Quinion, 2002). In this study, factors that may be associated with or put an individual at risk of becoming obese are considered obesogenic. This section reviews factors of interest to this study under the Social Cognitive Theory categories of individual, behaviour and environment. Individual factors include intrauterine and perinatal factors, timing of and BMI at adiposity rebound, early infant feeding, diet, child development and motor abilities, and physical fitness. Behavioural factors include physical activity, screen time, self-perceptions, and attitudes and values. Environmental factors include socioeconomic status, parental influences, and the built environment.

Of key importance, are differences between males and females, which will be considered across individual, behavioural and environmental factors. Gender differences have already been discussed in obesity prevalence rates, both in adults and children. Specific to this study, gender differences are known for BMI (Cole et

al., 2000), physical activity and related skills, sedentary behaviours, dietary behaviours (Hands et al., 2004; Martin et al., 2009), and pubertal development (Tanner, 1962). Evidence about gender differences is provided for relevant factors throughout this chapter.

### **Individual obesogenic factors.**

Factors affecting obesity related to the individual include one's innate abilities, knowledge and skills to interact in their environment (Bandura, 2001). Those of interest to this study include intrauterine and perinatal factors; timing of, and BMI at, adiposity rebound; early infant feeding; diet; developmental milestones and motor competence; physical fitness; and timing of puberty.

### ***Intrauterine and perinatal factors.***

Several potential intrauterine and perinatal factors have been implicated in the later development of obesity in the child. These include birth weight, gender, maternal parity, smoking during pregnancy, season of birth, gestational age and multiple birth (Reilly et al., 2005). This study focuses on birth weight and gestational age.

Infant birth weight is seen as a marker of intra-uterine health and well being with influence on the child's developmental outcome. Blair and colleagues (2007) conducted a longitudinal study of children from birth until age 7 years. They found a relationship between high birth weight and later high body fatness. They demonstrated that at each stage, birth, early childhood (age 3.5 years) and middle childhood (age 7 years), the development of obesity was critical. This supports earlier evidence by Reilly and colleagues (2005).

Infant birth weight is related to maternal smoking during pregnancy, which is also associated with restricted fetal growth (Gillman, 2008), lower birth weight, and increased odds of the development of obesity in the offspring (Dubois & Girard, 2006), although residual confounding by socio-cultural factors may still be possible

(Gillman, 2008). Previously, Hallal and colleagues (2006) have reported no association between birth weight and later sedentary lifestyle at 10-12 years of age, nor did they find an association between early growth acceleration (up to age 4 years) and later obesity. However, recent evidence has identified a positive association between increased gestational weight gain and BMI during childhood, with a heightened risk of later adult obesity. This may be a marker for permanent change in susceptibility to obesogenic environments (Schack-Nielsen, Michaelsen, Gamborg, Mortensen, & Sorensen, 2010). These recent studies are reflective of the conflicting evidence regarding the role of infant birth weight and its relationship to later weight status, and other obesogenic factors. This again reflects the lack of understanding of relationships between different contributing factors to weight status.

### ***Adiposity rebound.***

Adiposity rebound is an important marker for identifying the development of later obesity (Dietz, 2000; Dorosty, Emmett, Cowin, & Reilly, 2000; Rolland-Cachera et al., 1984; Rolland-Cachera, Deheeger, Maillot, & Bellisle, 2006). Adiposity rebound refers to the second rise in BMI curve that usually occurs between the ages of 5-7 years, or more specifically the upward trend in BMI after its nadir (Dietz, 2000; Dorosty et al., 2000; Rolland-Cachera, Deheeger, Guillaud-Bataille, Avons, Patois, & Sempe, 1987; Rolland-Cachera et al., 2006; Small, Anderson, & Melnyk, 2007; Williams & Dickson, 2002). This adiposity rebound has been argued to reflect upward BMI centile crossing (across BMI growth curves), which at any age can predict later obesity (Cole, 2004). In infancy, the most rapid height and weight growth rate occurs, slowing to a relatively constant growth rate during early to middle childhood (Botton, Heude, Maccario, Ducimetiere, Charles, & FLVS Study group, 2008; Sun, 2006). Some argue that the timing of adiposity rebound in early childhood can accurately predict up to 30% of later obesity (Dietz, 2000; Dorosty et al., 2000; Rolland-Cachera et al., 1987; Rolland-Cachera et al., 2006; Whitaker, Pepe, Wright, Seidel, & Dietz, 1998). Rolland-Cachera and colleagues (Rolland-Cachera et al., 1987; Rolland-Cachera et al., 2006) found that adiposity rebound at

age 3 years corresponded to obese individuals, while a later adiposity rebound at age 6 years corresponded to normal weight individuals.

In the last decade, several studies have investigated the biological and environmental factors that influence the timing of adiposity rebound (Dorosty et al., 2000; Williams & Dickson, 2002). While parental obesity was strongly associated with early adiposity rebound (Dorosty et al., 2000; Whitaker et al., 1998; Williams & Dickson, 2002), dietary variables such as high protein intake were not (Dorosty et al., 2000). It may be that early adiposity rebound is the result of factors yet to be identified (Dubois & Girard, 2006; Rolland-Cachera, Deheeger, & Bellisle, 1999; Small et al., 2007), which may program later weight status (Hallal et al., 2006; Skinner et al., 2004; Small et al., 2007).

### ***Early infant feeding.***

In the first year of life infants are breastfed or bottle fed and then transition to solid foods around 4 to 6 months of age. Although there is relative consensus to the health benefits of breastfeeding, the association and protective benefit in relation to obesity is as yet still unclear.

Infant feeding patterns may play an important role in the development of biological and behavioural processes, affecting subsequent growth and health (Oddy, Scott, & Binns, 2006b; Savage, Fisher, & Birch, 2007). However, debate continues as to whether breastfeeding is protective against, or predictive of childhood obesity, or rather uncontrolled bias (Arenz, Ruckerl, Koletzko, & von Kries, 2004; Horta, Bahl, Martines, & Victora, 2007; Kramer et al., 2009; Michels et al., 2007; Owen, Martin, Whincup, Smith, & Cook, 2005). According to Horta and colleagues (2007) biological, hormonal and behavioural mechanisms are implicated. Recent reviews and meta-analyses suggest longer duration of exclusive breastfeeding may be protective against later obesity (Arenz et al., 2004; Horta et al., 2007).

Increasingly, breast and formula feeding are being co-investigated, particularly in relation to later weight status (Burke et al., 2005; Dubois & Girard, 2006; Hediger, Overpeck, Kucamarski, & Ruan, 2001; Robinson et al., 2007). Hediger and colleagues (2001) reported that low birth weight infants (1,500 - 2,499g) were less likely to be breastfed compared to their heavier counterparts. There appears to be a link between breast feeding and timing of transition to solids. Infants who were introduced to solids at the recommended age (4-6 months) were most likely to have been breastfed, and those infants whose introduction to solids was delayed beyond this time tended to be breastfed for longer. In respect to mother's weight status, normal weight mothers breastfed for longer compared to underweight mothers, while overweight mothers tended to not breastfeed at all. However in relation to BMI and duration of breastfeeding, there was no difference between breastfeeding for 3 to 6 months versus more than 6 months, although the strongest effect was between never breastfed and breastfed. They found that there was an association between the timing of introduction of solids and weight status, with a 0.1% reduction in risk for each month of delay in the transition to solids (Hediger et al., 2001).

In contrast, Gillman and colleagues (2001) reported that the timing of transition to solids, infant formula or cow's milk was not associated with risk of obesity. They found breastfeeding for a longer time conferred a greater protective effect. One model estimated that for each 3 months of breastfeeding increment, there was an 8% reduction in risk of overweight. However, in relation to non-milk diet, they did report that 6- to 9-month-old breastfed infants consumed less non-milk foods than the formula-fed infants. They proposed a behavioural mechanism as a possible explanation, with breastfeeding being associated with less parental control. In other words, bottle feeding has less child self-regulation, with parent's behaviour able to override satiety (Gillman et al., 2001). From this perspective, breastfeeding could also be considered as a behavioural factor in the Social Cognitive Theory model.

## ***Diet.***

The increasing prevalence of obesity may be the result of the cumulative effects of excess daily energy intake which is both directly and indirectly related to dietary intake (Hu, 2008c; Rennie, Johnson, & Jebb, 2005). However dietary reviews have not found secular increases in energy intake (Rennie et al., 2005), nor consistent associations with obesity (Togo, Osler, Sorensen, & Heitmann, 2001), although increasing obesity rates may be associated with under-reporting of food intake in this group (Rennie et al., 2005). Studies over the years have focussed on intakes of fruit and vegetables, fat, energy, sugar snacking, fibre, fast food, and drinks, as well as looking at dietary patterns (McClain, Chappuis, Nguyen-Rodriguez, Yaroch, & Spruijt-Metz, 2009). These investigations have ranged from epidemiologic to clinical trials, yet the specific role of dietary factors remains unclear (Hu, 2008c).

Previously, energy intakes in Australian children have been reported to be increasing (Magarey, Daniels, & Boulton, 2001), with male energy intake typically higher than females (2-8 yrs) (Skinner et al., 2004). However, even within Australian cohort studies, results are mixed. Among girls, path analysis of variables dietary intake, physical activity and screen time did not explain the SES gradients in their adiposity. In boys however, fat intake seemed to play a mediatory role (Dollman et al., 2007).

Investigation of dietary patterns in the Raine cohort at age 14 years did not reveal a clear association with BMI, although longer television viewing was associated with a poorer quality, energy dense diet (*Western pattern*). They concluded that dietary patterns depended upon familial factors, in particular the psychosocial environment (Ambrosini et al., 2009a). A differently focused study on the same cohort found the *Western pattern* was associated with a greater risk of metabolic syndrome in females. It also found an inverse relationship between the healthier quality diet (*Healthy pattern*) and serum glucose levels across gender (Ambrosini et al., 2009b).



A link between diet and familial factors (Ambrosini et al., 2009a) has been shown. Permissive parental feeding styles (*indulgent*) or (*uninvolved*) had a negative influence on children's dietary intake. These children had lower intakes of fruits, juice and vegetables, and dairy foods (Hoerr, Hughes, Fisher, Nicklas, Liu, & Shewchuk, 2009). An association between perceived parent modelling and adolescent (aged 12 - 16 years) fruit and vegetable intake has also been shown previously (Young, Fors, & Hayes, 2004).

These findings support previous research. A diet high in fruits, vegetables, reduced fat dairy, wholegrains, along with low intakes of red and processed meat, fast food and soda was associated with smaller gains in BMI and waist girth (Newby, Muller, Hallfrisch, Qiao, Andres, & Tucker, 2003). Also, in adult middle aged women, an inverse relationship was shown between increased fruit and vegetable intake and risk of obesity or weight gain (He, Hu, Colditz, Manson, Willett, & Liu, 2004). Recently, a Western Australian survey found that only 40% of primary school children and 25% of secondary students consumed the recommended vegetable intake. While only 60% of primary school children and 25% of secondary students consumed the recommended fruit intake (Martin et al., 2009).

### ***Developmental milestones and motor competence.***

Childhood is an important period of development where biological, motor, psychosocial and cognitive changes are occurring. The process of change is both dynamic and interactive. Of particular relevance to the study of obesity is the role of physical activity, which itself is related with motor skill development (Dwyer, Baur, & Hardy, 2009).

Motor competence provides the critical building blocks for physical ability and the development of fundamental movement skills (FMS). These in turn provide for behavioural competencies in physical activity participation, including activities, games and team sports (Dwyer et al., 2009; Okely, Booth, & Chey, 2004; Stodden et al., 2008). There is a dynamic and developmental relationship between physical

activity, motor skill (perceived and actual), physical fitness and obesity. It has even been suggested that motor competence may be a “critically important, yet underestimated, causal mechanism partially responsible for the health-risk behaviour of physical activity” (Stodden et al., 2008 p.302). Certainly in 4-year-olds, poor motor skill was associated with less moderate to vigorous activity (Williams et al., 2008); confirmed in an older cohort of children aged 6-9 years, with actual motor competence positively associated with physical activity level (McIntyre, 2009).

Locomotor and object control (ball) skill proficiency has been investigated with respect to BMI. Among New South Wales children (grades 2, 4, 6, 8 and 10), proficiency in locomotion skills was inversely associated with BMI, but that this relationship was confounded around puberty. Based on these results, the authors speculated that motor competence and weight status are reciprocally related in the younger years (Okely et al., 2004).

Graf and colleagues (2004) compared fatness in 7-year-olds, with leisure behaviour (questionnaire) and motor development (Koperkoordinationstest fur Kinder KTK<sup>4</sup>). They found that children who were overweight or obese had poorer gross motor ability and endurance performance. Also, behaviour rated as an active lifestyle was positively associated with better motor ability and less TV watching. They supported this finding in a later study (Graf et al., 2005), which also found that obese children performed less well than non-obese children in coordination and endurance tasks.

Southall and colleagues (2004) suggest a correlation between motor competence and obesity in children. They found the healthy weight versus unhealthy weight group performed better in locomotor skills (run, gallop, hop, leap, horizontal jump,

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<sup>4</sup> KTK includes four items: balancing backwards, one-legged jumping obstacle, jumping from side to side, and sideways movements. Tasks are assigned points, with an overall Motor Quotient (MQ) resulting. Based on the MQ gross motor development is categorised ‘not possible’, ‘severe motor disorder’, ‘moderate motor disorder’, ‘normal’, ‘good’, and ‘high’.

slide), but not object-control skills (striking, stationary dribbling, catch, kick, overhand throw, underhand roll). They theorised this was related to the ability to move body mass in the locomotor tasks, and that actual or perceived lack of competence in motor skills for obese children may decrease participation in physical activity (Southall et al., 2004). Certainly evidence in 4-years-olds has shown that proficiency in fundamental movement skills have a weak but significant association with physical activity behaviours (Fisher et al., 2005), the latter a possible obesogenic factor. More recently, an association has been shown between motor competence and physical fitness in 14-year-olds of the Raine cohort, but not physical activity level (Hands, Larkin, Parker, Straker, & Perry, 2008).

### ***Physical fitness.***

Health related physical fitness encompasses cardiovascular performance, muscle strength, muscle endurance, flexibility and body composition (Caspersen et al., 1985) and is considered a stronger predictor of health outcomes than physical activity (Blair & Church, 2004). However there is a positive relationship between fitness and physical activity (Rowlands, Eston, & Ingledew, 1999). In the Raine Study cohort, aerobic fitness in 14-year-olds was associated with BMI, physical activity and motor competence (Hands et al., 2008). In children aged between 4 and 11 years, aerobic fitness was an independent predictor of adiposity, and also independent of ethnicity (Johnson et al., 2000), a finding consistent with others (Rowlands et al., 1999).

Physical fitness is considered a core component of successful and sustained weight loss (Blair & Church, 2004). Cardiovascular fitness has an independent protective role in cardiovascular disease and all cause mortality for adults (Blair, 1993; Carnethon, Gidding, Nehgme, Sidney, Jacobs, & Liu, 2003; Sui et al., 2007; Wei et al., 1999), adolescents (Carnethon, Gulati, & Greenland, 2005; Eisenmann, Katzmarzyk, Perusse, Tremblay, Despres, & Bouchard, 2005) and children (Wedderkopp, Froberg, Hansen, & Andersen, 2004). However, for individuals who

became obese during childhood, this protection may not be conferred (Carnethon et al., 2003).

There is also an association between increasing prevalence of obesity and decreasing fitness. Among Australian children, there is a general decline in fitness, which is most evident in the least fit children, or children who performed the worst (Olds, Dollman, Ridley, Boshoff, Hartshorne, & Kennaugh, 2004). In European youth, a similar decline across fitness tests has been found, but in addition, they report a widening in the differences between the least fit and most fit, as well as a widening in the differences between the healthy and unhealthy weight 9-year-old Danish children (Wedderkopp et al., 2004). This decline is reported for a number of different types of tests, such as shuttle run, and endurance. Olds and colleagues' (2004) review of international data did not support the Australian trend, although they did find an international decline in the time for distance runs (i.e. cardiovascular fitness) (US, Italy, Poland, Asia, Japanese, South Korea). By way of explaining these differing findings, the authors stress methodological inconsistencies in fitness measures that make comparison difficult (Olds et al., 2004). However, comparisons of fitness tests in tri-athletes (Basset & Boulay, 2003) and severely overweight youth (Loftin, Sothorn, Warren, & Udall, 2004) suggests that differences may lie between population or ethnic groups, rather than between fitness tests.

Overall though, aerobic fitness is not necessarily reduced in individuals with fatness and excess body weight (Goran, Fields, Hunter, Herd, & Weinsier, 2000). Those who were overweight or obese and less aerobically fit had a higher risk of cardiovascular disease and diabetes, compared to those with higher fitness (Sui et al., 2007; Wei et al., 1999), which suggests that obesity and fitness may operate independently in respect to health outcomes. It should also be noted, that in adolescents the relationship between fitness, BMI and cardiovascular disease risk is complex and dependent upon risk factor assessed (Eisenmann et al., 2005).

### ***Puberty.***

Sexual maturation correlates better than chronological age with physical, physiological and behavioural measures during adolescence (Duke, Litt, & Gross, 1980). In girls, a minimum amount of body fat is required in childhood to achieve menarche (Sloboda, Hart, Doherty, Pennell, & Hickey, 2007). Evidence is mounting that greater body fat and obesity is associated with earlier menarche and puberty (Aksglaede, Juul, Olsen, & Sorensen, 2009; Chen, Mi, Chen, Hou, & Zhao, 2007; Davison, Susman, & Birch, 2003; Sloboda et al., 2007). Overall though, puberty is occurring at an earlier age, regardless of weight (Aksglaede et al., 2009; Davison et al., 2003).

### **Behavioural factors.**

Behavioural factors relate to an individual's interactions with their environment. Behaviour is influenced by an individual's thoughts and actions, peers, and social structures (Davis, 2006). Factors specific to this study include physical activity, sedentary behaviour, self concept, and attitudes and values.

### ***Physical Activity.***

The level of participation in physical activity is considered to be an important factor in the prevention of obesity (Flynn et al., 2006). It has been consistently shown that boys are generally more active than girls, and children more active than adolescents (Dollman & Lewis, 2009; Hands et al., 2008; Hands et al., 2004; Martin et al., 2009; Thompson, Campagna, Durant, Murphy, Rehman, & Wadsworth, 2009). Systematic review of physical activity levels though suggest that only about 54% of preschool children were moderately or vigorously active for at least 60 minutes a day (Tucker, 2008). Of concern, a 2008 Western Australian survey found that less than half of all primary and secondary students were meeting this recommendation, with girls less active than boys (Martin et al., 2009). Physical activity levels themselves are said to be the result of a "complex causal web of influences" (Olds et al., 2004 p.38), and physical activity cause and effect in respect to BMI is still debated.

Over the years there has been conflicting evidence regarding the relationship between adiposity and physical activity, indicating a lack of understanding of the relationships between different contributing factors, and/or measurement issues. In 2005, a 20 month school based health and physical activity program showed no effect on the incidence of overweight and obese, suggesting other factors, or an interplay of factors were at work (Graf et al., 2005). Dragan and Akhtar-Danesh (2007) reported an inverse relationship between physical activity and BMI, that is, with increasing levels of physical activity there is decreasing obesity. This concurs with Ness and colleagues (2007), but they go further to suggest that the association is stronger for boys, and intensity of activity is more important than total activity. Hallal and colleagues (2006) found that neither weight gain, nor overweight at age 1 or 4 years predicted physical activity levels in early adolescence. A study using the Raine Study cohort found no relationship between adiposity and physical activity (Hands et al., 2008). More recently a “weak and inconsistent” (p.32) association was found in children and adolescents (Thompson et al., 2009). Regardless of cause and effect, few health professionals would question the broader positive health benefits that accrue from being physically active.

While the extent to which physical activity is implicated in the development of obesity is debatable, it is important to continue to consider factors related to physical activity participation such as its role in energy expenditure in the energy balance equation. Evidence supports the decline in physical activity levels, greatest in adolescence (13-18 years), and that in children this may be due to the decline in active free play (Olds et al., 2004; Thompson et al., 2009). This decline is probably a result of many environmental factors such as perceived safety, parental commitments, the urban in-fill, and lack of neighbourhood social networks (Olds et al., 2004). If indeed the decline in physical activity is related to the increases in childhood obesity, then these possible causes must also be considered.

Predictors of adolescent physical activity include maternal BMI, birth order and earlier physical activity behaviours (Hallal et al., 2006), gender (Hallal et al., 2006; Olds et al., 2004), SES (e.g. family income, maternal education) (Cleland, Ball,

Magnussen, Dwyer, & Venn, 2009; Hallal et al., 2006), time spent outdoors (Centre for Community Child Health, 2007; Olds et al., 2004; Sallis, 2000), and using physical activity for transport (Fisk, 2007). Physical activity levels have also been shown to predict other obesogenic variables, such as sedentary behaviour (Hallal et al., 2006).

Overall, Fogelholm & Kukkonen-Harjula's (2000) review of a number of studies regarding physical activity and weight, summarises the possible relationship between physical activity and obesity. They hypothesised that physical activity could play a role in three different ways, one in respect to preventing weight gain. Secondly that less weight gain resulted in better physical activity adherence. Their third hypothesis follows the line of thinking for this thesis, in that physical activity may be a "proxy for a generally healthier lifestyle or psychological profile" (p. 105). That is, those who tend to be more physically active, also tend to have healthier diets, normal weight parents, spend less time in sedentary activity, have positive parenting, and so forth.

### ***Sedentary behaviour.***

Traditionally obesity research has focussed on physical activity (energy output) and diet (energy input). Over time however, the role of physical inactivity or sedentary activities has been highlighted, with screen time a common modern proxy for sedentary behaviour. Physical inactivity generally describes sedentary activities such as sitting, screen time and reading (Zderic & Hamilton, 2006). It may play a more significant role than diet and exercise as a primary and independent risk factor for obesity (Chaput & Tremblay, 2009; Olds et al., 2004; Struber, 2004) insulin sensitivity (Alberti, Zimmet, & Shaw, 2006) and metabolic syndrome (Zderic & Hamilton, 2006) and generally chronic disease, disability and premature death (Haskell, Blair, & Hill, 2009). Recent work found that physical inactivity in adolescence was predictive of obesity (waist girth) into young adulthood (Pietiläinen et al., 2008), with sleep time and knowledge-based work also thought to be important (Chaput & Tremblay, 2009). Others have suggested motor

competence as a causal mechanism of physical inactivity (Stodden et al., 2008). Interestingly, body weight has been shown to be strong predictor of later physical inactivity (Bak, Petersen, & Sorensen, 2004).

It has been speculated that sedentary behaviours displace the time available to be spent on physical activity, commonly referred to as the *displacement hypothesis*. Evidence varies, some studies confirming the hypothesis (Hohepa, Scragg, Schofield, Kolt, & Schaaf, 2009), and with others finding weak or no associations (Biddle, Gorely, & Stensel, 2004b; Marshall, Biddle, Gorely, Cameron, & Murdey, 2004; Smith, Rhodes, Naylor, & McKay, 2008; te Velde et al., 2007). Noteworthy, Biddle and colleagues (2004a) argue that although sedentary behaviours may preclude physical activity at that time, it is not necessarily indicative of an inverse relationship over a longer period (e.g. day or week).

Currently, the lifestyles led in western society, both as adults and children, are increasingly sedentary. This applies for transport, work, play, entertainment and general living (Struber, 2004; Wood, 2009). Both Blair et al. (2007) and Mitchell et al. (2009) have found that for every hour of sedentary activity, the risk of obesity is increased. Olds and colleagues' (2004) earlier review of literature suggest such a correlation is significant, but small, and it may be the holistic environmental effect that is of significance. For example the displacement of active play, lowering of resting metabolism, snacking behaviours and choices of snacks, whilst involved in sedentary activity may together contribute to obesity.

Not surprisingly, sedentary behaviour among children may also reflect parental attitudes and behaviours. Dehghan and colleagues (2005) report that parents prefer their children at home (watching TV or playing on the computer) so that they can continue to perform other activities (e.g. chores) and keep watch on their children. Active outside play is usually perceived as unsafe, especially when unsupervised, which exemplifies the parental value of outside play and physical activity in their hierarchy of child rearing (Dehghan et al., 2005).



Lastly, interventions that target sedentary behaviours are more effective than exercise and diet programs in encouraging free play in children (Dehghan et al., 2005) and increased leisure-time activity in adults (Sugiyama, Healy, Dunstan, Salmon, & Owen, 2008). Even small reductions in physical inactivity reap substantial health benefits (Struber, 2004). Evidence suggests physical inactivity may be independent of SES factors (maternal education) (Ball, Cleland, Timperio, Salmon, & Crawford, 2009), and that targeting individual and environmental barriers to being active (Struber, 2004) may be more effective in reducing inactivity levels. Certainly evidence suggests that inactivity during adolescence may trigger “a self-perpetuating vicious circle of less activity, low energy expenditure and increasing adiposity” (Pietiläinen et al., 2008 p.412).

### *Screen Time.*

In respect to obesity, a panel review report recommended the importance of investigating sedentary activities such as TV watching, as separate to the absence of activity (Jebb & Lambert, 2000). Increasingly, researchers are using the proxy measures of screen based activity such as watching television or videos and playing video or computer games for sedentary activity. Generally, research to date identifies strong causal links between screen time and obesity (Boone, Gordon-Larsen, Adair, & Popkin, 2007; Hume, Singh, Brug, van Mechelen, & Chinapaw, 2009), with some evidence suggestive of a greater importance for boys (Aucote & Cooper, 2009; Olds, Ridley, & Dollman, 2006).

Also, high screen time users tend to have lower moderate-vigorous physical activity (Olds et al., 2006), which suggests a possible operational mechanism for obesity. Previously, Olds and colleagues (2004) found that the main competitor to physical activity participation is screen time, that is, TV, video games, movies / DVDs and phone texting (something not commonly reported in the literature), a view supported by Viner and Cole (2005). During what Olds and colleagues (2004) deemed the critical window (after school and before dinner time), children were more likely to be involved in screen based activity than physical activity by three to

four times. The authors summarised studies looking at youth attitudes and their rank order of leisure pursuits. In 1974, girls ranked TV 10<sup>th</sup>, but by 1994 it is ranked second, retaining this rank in 2000. For boys, TV was ranked 2<sup>nd</sup> in 1994, but was 4<sup>th</sup> by the year 2000.

Screen time has also been associated with food snacking behaviours. The effects of these snacking behaviours are worse among unhealthy weight families (Francis, Lee, & Birch, 2003). In addition, screen time, particularly television viewing, reduces metabolic rate (i.e. resting energy expenditure) and perhaps is another operational mechanism for obesity (Klesges, Shelton, & Klesges, 1993). By adulthood screen time is associated with increased risk of all-cause and cardiovascular disease mortality (Dunstan, Barr, Healy, Salmon, Shaw, & Balkau, 2010). Overall, screen time may operate via several mechanisms, both direct (e.g. by reducing metabolic rate) and indirect (e.g. by reducing physical activity levels), to affect risk of obesity.

### ***Self Concept.***

Self concept may play a role in respect to obesity, although study results are mixed (Field, 2008). Self concept is a prominent construct in investigations of human behaviour (Hagger, Biddle, & John Wang, 2005; Harter, 1990), providing an avenue to articulate characteristics of self and their role in specific behaviours. It impacts on an individual's functioning processes, specifically emotion, motivation and ability to cope (Harter, 1990). One's self concept determines how one perceives their ability to perform tasks, and includes perceptions about self, the latter commonly referred to as self-esteem (Wilgenbusch & Merrell, 1999). It has been shown that by adolescence, children are able to make peer related judgements about popularity, friendship and acceptance (Harter, 1990). The reliability of physical self-perceptions in adolescence is relatively consistent at a population level, although at an individual level, some inconsistencies have been observed (Raudsepp, Kais, & Hannus, 2004).

Waters and Baur (2003) found that children who are obese have lower self-esteem and have psycho-social issues related to social acceptance, athletic competence and physical appearance. A positive self image is important for adaptive functioning and general happiness (Harter, 1990). Like other obesogenic variables, self concept may operate in respect to obesity via both direct and indirect mechanisms. For example, self concept is an important construct in respect to physical activity level (Sallis, Proschaska, & Taylor, 2000; Weiss, 1987). Gender specific perceived competencies and motivations may affect physical activity participation (Murcia, Gimeno, Lacarcel, & Perez, 2007; Savage, DiNallo, & Downs, 2009; Weiss, 1987). Increased participation (sport practice) was related to increased physical self-perceptions, but not for strength or physical appearance domains (Murcia et al., 2007). Certainly providing opportunities for participation in a diverse range of physical activities and sports is positive intrinsic motivation for continued participation, with this motivation built on experiences where an individual can feel confident, realise benefits of effort and succeed (Li, Lee, & Solmon, 2005). Conversely, adolescent perceptions of poor fitness are associated with increased risk of adult obesity (Pietiläinen et al., 2008).

Participation in regular physical activity is thought to be driven by dynamic and complex behavioural processes related to self concept (Dishman, Sallis, & Orenstein, 1985; Sallis et al., 2000). These involve interactions between individual's attitudes and beliefs, perceived needs and skill abilities, outcome expectations, personality, feelings, lifestyle and the environment. Being active or sedentary may not be reasoned decisions, but rather the outcome from critical behavioural determinants that act in direct and indirect ways (Dishman et al., 1985). The role of self concept in the determination of the perceived importance, benefits and barriers to being physically active may be one mechanism for understanding physical activity participation levels and its role with obesity.

### **Environmental obesogenic factors.**

Factors related to environment include the social and physical environments imposed, selected and constructed for the individual (Bandura, 2001). Variables specific to this study include socioeconomic status (SES), parental influences, and the built environment.

### ***Socioeconomic status.***

Socioeconomic status (SES) comprises a number of characteristics that influence behaviour and energy balance. These include wealth (material), environment (community), educational level, knowledge (experience) and stress (Sanigorski, Bell, Kremer, & Swinburn, 2007). Typically SES is measured by occupation, education and income elements (Kendall, 2003; Sanigorski et al., 2007). According to Social Cognitive Theory, SES could be working at each level, that is, income could determine one's ability to participate in organised sport (behavioural), or parental culture (e.g. participation in sport by girls) and education (e.g. importance and valuing of out of school sport and activity for health and well being) could impact one's ability to build physical fitness (individual).

The Australian Bureau of Statistics (2007b) identified that areas of greatest relative disadvantage had the highest prevalence of adults classified obese, although those classified overweight were similar across SES. The total number of overweight and obese individuals were similar in low and high income groups, although low income populations had higher proportions of obese, while high income populations had higher proportions of overweight. These statistics concur with Australian studies of children (Dollman & Lewis, 2009; Dollman et al., 2007; Sanigorski et al., 2007), and similar to the inverse social gradients reported for children in high-income countries (Due et al., 2009).

In 2003 and 2004, Sanigorski and colleagues (2007) looked at the association between BMI indicators and SES in 2,184 Victorian children aged 4-12 years. Their study confirmed that low SES (measured by Socioeconomic Index for Areas – SEIFA,

low parental education and low family income) were associated with increased rates of obesity. Certainly in adult men, intelligence and educational attainment were inversely related to changes in BMI and risk of obesity (Halkjoer, Holst, & Sorensen, 2003). Sanigorski and colleagues (2007) also found that female children were more likely to become overweight or obese. This adds further support to the gender effect reported earlier by Olds and colleagues (2004).

Interestingly, Sanigorski and colleagues (2007) found no association between obesity and food security assessed by reported “days the previous month when families did not have enough food to eat or enough money to buy food” (p. 1909). Bua, Olsen and Sorensen (2007) reviewed height and weight measures of Danish 7- to 13-year-olds from 1930-1983 in respect to economic growth. They determined that the increased prevalence in obesity in their population was not associated with the corresponding trends in economic growth. Another observation was that the largest increase in BMI occurred in the upper percentiles ( $\geq 95$ ), across ages and gender.

SES influences on obesity may also contribute indirectly through other environmental factors. Dalton (2007) identified that children from low incomes (compared to high income children) have a greater exposure to fast food advertising (TV), access to more fast food shops (less healthy options), live within neighbourhoods with more unsafe streets, have unmaintained parks and playgrounds, with the local school environment reflecting the community environment. Therefore it appears that SES influences obesity at both an individual and environmental level.

### ***Parental influences.***

Parents play an integral role in the prevention of obesity in their children and this role changes with child development and age (Lindsay, Sussner, Kim, & Gortmaker, 2006). Broadly, parental influences such as intrauterine experiences, parent BMI, parenting styles, and parents as role models have been investigated.

In particular, maternal behaviours during pregnancy have been a focus of developmental origins of obesity research. As previously discussed (Intrauterine and prenatal factors), negative maternal behaviours during pregnancy have been associated with restricted fetal growth, lower birth weights, and increased odds of the development of obesity in the offspring (Dubois & Girard, 2006; Gillman, 2008; Lindsay et al., 2006).

### *Parent BMI.*

Increasingly it has been shown that overweight and obese parents tend to have overweight and obese children (Dubois & Girard, 2006), with high parental BMI strongly associated with high BMI in their children (Bell et al., 2007; Huus et al., 2007; Schack-Nielsen et al., 2010). In most cases this pathway to obesity is not genetic, but rather a genetic predisposition which increases susceptibility to obesogenic factors (Silventoinen et al., 2007; Sorensen & Echwald, 2001). A genetic predisposition may mean that particular families are influenced to a greater extent by factors such as diet and lack of exercise (Campión, Milagro, & Martínez, 2009).

A study by Davey Smith and colleagues (2007) found no difference in the strength of association between maternal, paternal or non-biological paternal BMI (self-report) and their child's BMI at age 7.5 years. In contrast, a Western Australian study found that maternal overweight and single mother parenting increased the chance of the child being overweight or obese (Gibson et al., 2007). The overwhelming evidence suggests a strong association between parent BMI and child BMI (Bell et al., 2007; Dubois & Girard, 2006; Gibson et al., 2007; Huus et al., 2007).

### *Parenting styles.*

Parental styles may influence their child's weight (Gibson et al., 2007; Wake, Nicholson, Hardy, & Smith, 2007). Authoritarian parenting has been shown to be

associated with higher risk of obesity in 7-year-olds, with a lack of development of independent self-regulation of diet and exercise postulated as a possible mechanism. Further, children of permissive and neglectful parents are also at increased risk, however the mechanism is suggested to be related to lack of appropriate guidelines for acceptable behaviours (Rhee, Lumeng, Appugliese, Kaciroti, & Bradley, 2006).

Parenting styles are rarely studied, but may play a key role in maintaining healthy weight (Gibson et al., 2007). Cultural and SES factors help shape parenting styles. Together parental, cultural and SES are associated with issues of access, physical resources such as space and equipment, sedentary options, physical activity behaviours, and diet, all factors implicated in a causal role with obesity. Family structure and number of siblings influence activity behaviours, with more siblings resulting in more sport played as well as facilitating social networks. In contrast, parent's concern for children's safety, both from traffic and disagreeable people, has seen a decline in free play within communities (Olds et al., 2004).

### *Parents as role models.*

Parents serve as role models to their children in all aspects of their life. Parents influence patterns of behaviour both directly and indirectly. Food choices, activity levels, leisure time choices, appropriate social behaviours, and support, all play an important part in moulding the child and their own behaviour patterns (Gibson et al., 2007; Olds et al., 2004).

Generally, parents play a direct role in controlling young children's food intake (Birch & Davison, 2001; Lindsay et al., 2006). Among older children, parent dietary modelling and using food as rewards were associated with obesity, the latter was also associated with higher unhealthy food consumption (Kroller & Warschburger, 2009).

In respect to physical activity, Olds and colleagues (2004) reported that indirect influences affect a child's attraction to physical activity and also their perceived competence. Direct modelling appears to be least influential, especially as the child gets older, however there was an association between father's involvement. Parents also influence through their level of support for sport and leisure pursuits, such as payment of fees, providing choices, transportation, verbal recognition, attitudes and values (Olds et al., 2004).

In today's society parents are less likely to be home during the day, which may limit their ability to supervise their child's play. Consequently, there has been a one-third decrease in free play in neighbourhoods. Most parents (80%) in their childhoods played unsupervised in their neighbourhood, but this has dropped to 25% a generation later (Olds et al., 2004). The rationale for this decline is research that found parental health concerns were neighbourhood safety, alcohol, drugs and sex, rather than their children's weight (Dalton, 2007).

### ***Built environment.***

Built environmental factors can have both positive and negative influences on obesity related behaviours, particularly physical activity. Increases in physical activity occur with greater access to recreational facilities, opportunities to exercise, and spending more time outdoors (Maddison et al., 2009; Sallis & Glanz, 2006; Veitch, Salmon, & Ball, 2010; Wood, 2009), although these seem to be dependent upon physical activity context and gender (Page, Cooper, Griew, & Jago, 2010). Physical activity levels tend to decrease in areas of high crime, where there are personal safety concerns, or transport infrastructure barriers (traffic, speed and density) (Maddison et al., 2009; Veitch et al., 2010; Wood, 2009).

Parks and open space environments are often underutilised opportunities for maintaining and improving physical activity levels. For children and adolescents, these environments can be places for socialisation, physical activity, exploring, and free play. There is evidence to suggest close proximity of the family home to parks



and play spaces increase their use (Wood, 2009) and hence should be associated with decreased obesity levels. However, a recent Canadian study, controlling for SES confounders, found that the presence of community level parks and green space was not associated with obesity levels (Potestio, Patel, Powell, McNeil, Jacobson, & McLaren, 2009). The latter supports results from an earlier American study of urban low-income 3- to 4-year-olds. They also found no associations between obesity and proximity to fast food outlets or neighbourhood safety (crime) (Burdette & Whitaker, 2004).

The school environment may be important given the time spent each day at school by children and adolescents. A Canadian study identified that the design of the school environment may determine the difference between a child being moderately or highly active (Leatherdale, Manske, Faulkner, Arbour-Nicitopoulos, & Bredin, 2010). However another study found only a weak association between the school physical activity environment and adolescent obesity prevalence (O'Malley, Johnston, Delva, & Terry-McElrath, 2009). In that school, participation rates in physical education classes were low and declined across the adolescent school grades. In Australia no association was found between low, medium and high compulsory physical activity schools and total level of physical activity, fitness or obesity (Cleland, Dwyer, Blizzard, & Venn, 2008b).

Despite current evidence, a recent systematic review of the built and biophysical correlates of obesity in children and adolescents suggested that strong empirical evidence is lacking. Inconsistencies in results across studies, lack of repetitions, and confounding by other variables such as SES and gender were reported (Dunton, Kaplan, Wolch, Jerrett, & Reynolds, 2009).

### **Critical Risk Period for the Development of Obesity**

Pathways to obesity are dynamic and influences change over time. If research can identify periods of increased risk, along with the factors implicated at these times, then appropriate and timely prevention measures could be introduced (Hilbert et

al., 2008). Probable periods of risk for development of obesity are infancy, early childhood and adolescence (Blair et al., 2007; Dietz, 1994; Lawlor & Chaturvedi, 2006; Steinbeck, 2001). In summary, low birth weight is associated with risk of later obesity (Dietz, 1997). Early adiposity rebound is a marker for greater adiposity in adolescence and adulthood (Lawlor & Chaturvedi, 2006; Rolland-Cachera et al., 1984). While adolescence presents additional risk with normal fat deposition related to puberty, especially in females (Steinbeck, 2001), as well as declines in physical activity and increases in sedentary behaviour (Olds et al., 2004).

The influence of other factors may be important and vary over time. These include early feeding choices and dietary behavioural mechanisms (Gillman et al., 2001; Oddy et al., 2006b); physical activity and movement related development of muscle strength, kinaesthetic awareness and motor skills which peak between age 7 and 9 years (Eaton, McKeen, & Campbell, 2001); early patterns of sedentary behaviour such as screen time (Blair et al., 2007; Centre for Community Child Health, 2009); and possibly early socioeconomic influences (Sanigorski et al., 2007).

### **Measurement Issues<sup>5</sup>**

Obesity is considered an excess of body fat that predisposes an individual to adverse health consequences. When tracking and investigating the prevalence of obesity in large populations, valid and accurate field measures of body composition are required. However, it has been questioned how body fat can be accurately measured, and which proxy measure is a better indicator for adverse health consequences (Marshall, Hazlett, Spady, Conger, & Quinney, 1991)? The selection of a measure in children and adolescents is constrained by time, cost, and reproducibility. Additional considerations must be made, especially for those with excess fat, as it is more difficult to consistently measure obese individuals

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<sup>5</sup> Adapted with permission: Chivers, P., Hands, B., Parker, P., Beilin, L., Kendall, G. & Bulsara, M. (in press). *A comparison of field measures of adiposity among Australian adolescents from the Raine Study*. Malaysian Journal of Sports Science and Recreation.

compared to their lean counterparts, for most anthropometric measures (Heyward, 2001).

At the moment, one simple, direct field measure is not available. Instead there are many indirect measures of adiposity, each with their own limitations, assumptions, and criticisms (Goran, 1998; Marshall et al., 1991). Many factors including those related to gender, age, and puberty impact on the value of these measures. What constitutes classification of obesity itself is arbitrary, as each measure is more or less continuously related to the risk of adult disease. Not surprisingly, with no common percentile point for obesity across measures, there is the likelihood of discrepancies in classification of continuous variables into categories of adiposity (Marshall et al., 1991).

Commonly used anthropometric measures involve body size and proportion such as height, weight, and circumference measures (Heyward, 2001; Hills, Lyell, & Byrne, 2001). Most anthropometric indices are a ratio between two different body measurements and include Body Mass Index (BMI), Waist-Hip Ratio (WHR), and Waist-Height Ratio (WHtR) (Hills et al., 2001). Anthropometric measures generally make for good field tests as they are relatively easy to administer, inexpensive, and require less technical skill compared to laboratory constrained tests such as the Dual energy X-ray absorptiometry (DXA) (Heyward, 2001).

The problem lies with which field measure to choose for studies using adiposity proxy measures. Is there a difference between these measures in weight status categorisation? For longitudinal studies, can we interchange or make comparisons between these proxy measures dependent upon which have been collected across time points?

### **Adiposity measures.**

A brief summary of commonly used measures for adiposity studies in children and adults is presented at Table 3. A description along with advantages and disadvantages are given for each measure.

Table 3

*Brief Summary: Description, Advantages and Disadvantages of Common Proxy Measures of Adiposity*

	BMI	Skinfold	Waist Girth	Hip Girth	WHtR	WHR
Description	Ratio of height over weight.	Indirect measure of of subcutaneous adipose tissue, usually over a number of body sites.	Measurement of waist circumference.	Measurement of hip circumference.	Ratio of waist girth over height.	Ratio of waist girth over hip girth.
Advantages	<ul style="list-style-type: none"> <li>• Most common indicator for adiposity.</li> <li>• Reliable</li> <li>• Easy to measure.</li> <li>• Cheap to measure.</li> <li>• International recognised cut-points (Cole et al., 2000).</li> <li>• BMI-for-age z-score option.</li> <li>• High correlation with fat mass.</li> <li>• High correlation with DXA.</li> <li>• High correlation with skinfold measures.</li> <li>• Similar to waist girth.</li> </ul>	<ul style="list-style-type: none"> <li>• 12 site measurement provides similar results to magnetic resonance imaging.</li> <li>• Good predictor of percentage fat.</li> <li>• Triceps, subscapular, chest and abdominal are best predictors.</li> <li>• Sum of skinfolds correlated with BMI.</li> </ul>	<ul style="list-style-type: none"> <li>• Simple and better estimation of central fatness than BMI.</li> <li>• Correlates with DXA fat distribution.</li> <li>• Associated with metabolic risk.</li> <li>• Measures central subcutaneous and visceral fat.</li> <li>• Associated with a number of adverse health outcomes.</li> <li>• Better predictor of cardiovascular risk compared to BMI.</li> <li>• Less gender specific.</li> </ul>	<ul style="list-style-type: none"> <li>• Measures overall fat distribution.</li> <li>• Associated with a number of adverse health outcomes.</li> </ul>	<ul style="list-style-type: none"> <li>• Similar predictive ability to BMI.</li> <li>• High correlation with BMI.</li> <li>• Equivalent cut-point to BMI cut-points.</li> <li>• Simpler and better associations with metabolic risk.</li> <li>• Does not require age and gender centiles.</li> <li>• A better measure of weight status than BMI.</li> </ul>	<ul style="list-style-type: none"> <li>• Cut-points exist.</li> </ul>

	BMI	Skinfold	Waist Girth	Hip Girth	WHtR	WHR
Disadvantages	<ul style="list-style-type: none"> <li>• Limitations in children (growth spurts, gender, maturity).</li> <li>• Underestimates obesity prevalence.</li> <li>• Less accurate in normal weight.</li> <li>• No age and gender cut-points before age two years.</li> <li>• Problems with self report measures, typically underestimated.</li> </ul>	<ul style="list-style-type: none"> <li>• Measurements are highly investigator dependent.</li> <li>• Not usually recommended for obese.</li> <li>• Skinfold equations tend to underestimate body fat.</li> <li>• Prediction equations not suitable in children.</li> <li>• Misclassification problematic.</li> </ul>	<ul style="list-style-type: none"> <li>• Does not have accepted cut-points.</li> </ul>	<ul style="list-style-type: none"> <li>• Reproducibility good but lower than BMI.</li> <li>• Different measurement protocols used internationally.</li> </ul>	<ul style="list-style-type: none"> <li>• Similar limitations to BMI.</li> </ul>	<ul style="list-style-type: none"> <li>• Prognostic value in children low.</li> <li>• Does not appear to accurately reflect intra-abdominal fat mass.</li> <li>• Value as health risk predictor unclear.</li> <li>• Does not have accepted cut-points.</li> </ul>

(Abbott et al., 2002; Ashwell & Lejeune, 1996; Australian Institute of Health and Welfare, 2005; Blair et al., 2007; Bua et al., 2007; Chatterjee, Chatterjee, & Bandyopadhyay, 2006; Claessens, Delbroek, & Lefevre, 2001; Cole et al., 2000; Cole, Faith, Pietrobelli, & Heo, 2005; Croft, Keenan, Sheridan, Wheeler, & Speers, 1995; Daniels, Khoury, & Morrison, 2000; Dehghan et al., 2005; Denney-Wilson, Booth, & Baur, 2003; Dezenberg, Nagy, Gower, Johnson, & Goran, 1999; Eisenmann, 2005; Fredriks, van Buuren, Fekkes, Verloove-Vanhorick, & Wit, 2005; Freedman, Khan, Serdula, Dietz, Srinivasan, & Berenson, 2005; Garnett, Baur, Srinivasan, Lee, & Cowell, 2007; Guo & Chumlea, 1999; Heyward, 2001; Heyward, 1998; Hills et al., 2001; Himes, Bouchard, & Pheley, 1991; Hsieh, Yoshinaga, & Muto, 2003; Huus et al., 2007; Kahn, Imperatore, & Cheng, 2005; Katzmarzyk, Srinivasan, Chen, Malina, Bouchard, & Berenson, 2004; Krebs et al., 2003; Li, Ford, Mokdad, & Cook, 2006; Lindsay, Hanson, Roumain, Ravussin, Knowler, & Tataranni, 2001; Marshall et al., 1991; McCarthy & Ashwell, 2006; McCarthy, Ellis, & Cole, 2003; McCarthy, Jarrett, Emmett, Rogers, & ALSPAC Study Team, 2005; Mei, Grummer-Strawn, Pietrobelli, Goulding, Goran, & Dietz, 2002; Must & Tybor, 2005; National Health and Medical Research Council, 2003; Peterson, Czerwinski, & Siervogel, 2003; Steinbeck, 2001; Steinberger, Jacobs, Ratz, Moran, Hong, & Sinaiko, 2005; Taylor, Jones, Williams, & Goulding, 2002; te Velde et al., 2007; Tremblay, Kastzmarzyk, & Willms, 2002; Yarnell, Patterson, Thomas, & Sweetnam, 2001)

Population studies commonly use body mass index (BMI), a weight for height ratio ( $\text{wt}/\text{ht}^2$ ) to determine weight status. The most common cut-points used are based on the International Obesity Task Force (Cole et al., 2000) and the Centre for Disease Control (CDC) (Division of Nutrition Physical Activity and Obesity, 2009). The International Obesity Task Force (Cole et al., 2000) developed BMI cut-points for normal weight, overweight and obese, which are now well established as benchmarks, with age and gender adjusted cut-offs for children (age 2-18 years). For adults, a BMI between 25 and 30 is categorised as *overweight*, while a BMI  $>30$  is categorised as *obese*. For children and adolescents, cut-off points have been derived to equate to a body mass index of 25 and 30  $\text{kg}/\text{m}^2$  at age 18 years (Cole et al., 2000). More recently, an underweight category has been added (Cole, Flegal, Nicholls, & Jackson, 2007). An alternative is the Centre for Disease Control (CDC) BMI-for-age growth charts which use percentile rankings of underweight  $<5^{\text{th}}$  percentile, healthy weight  $5^{\text{th}} - 85^{\text{th}}$  percentile, overweight  $85^{\text{th}} - 95^{\text{th}}$  percentile, and obese  $\geq 95^{\text{th}}$  percentile (Division of Nutrition Physical Activity and Obesity, 2009).

In summary, BMI is considered a reasonable marker of fatness or adiposity (Bua et al., 2007; Cole et al., 2000; Dehghan et al., 2005; Guo & Chumlea, 1999; Katzmarzyk et al., 2004; Krebs et al., 2003; Taylor et al., 2002; Tremblay et al., 2002; WHO, 2006), and is considered, at this stage, the measure of choice in children for adiposity tracking and association with health risk (Garnett et al., 2007), but is not considered a diagnostic tool (Division of Nutrition Physical Activity and Obesity, 2009). It is a ratio based on the measurements of height and weight which are considered to be highly reliable (Bua et al., 2007; Guo & Chumlea, 1999; Lindsay et al., 2001). Its other advantage is that height and weight is relatively easy and cheap to obtain, and BMI simple to derive (Lindsay et al., 2001; Mei et al., 2002; Steinberger et al., 2005), with weight status cut-points age and gender specific, although its value in the child population has been questioned (Hills et al., 2001). In the research and epidemiological settings, BMI is a practical, convenient and useful tool for assessment of weight status, including for children and adolescents (Denney-Wilson et al., 2003; Steinbeck, 2001).

## **Longitudinal Studies**

Longitudinal studies are an important research design for investigating how humans develop over time. Understanding the processes and structures involved in human development over time is fundamental to understanding illnesses and diseases (Magnusson, 1993) such as obesity. A longitudinal research design is valuable when the object(s) of investigation are dynamic, there are individual differences in growth rate, or critical windows for the timing of specific events, and effects may be lagged (Magnusson, 1993).

Longitudinal studies provide advantages over cross sectional research designs (Magnusson, 1993; Singer & Willet, 2003), and repeated cross-section surveys (Yee & Niemeier, 1996) by measuring change over time (Singer & Willet, 2003; Thomas & Nelson, 1990; Yee & Niemeier, 1996). Statistical processes are able to separate cohort (differences between individuals at baseline) and age (changes over time within individuals) effects. Repeated measures on the same cohort also minimise large standard errors of between subject variation that are often found in repeated cross-sectional analyses (Yee & Niemeier, 1996).

Some disadvantages include individual inter-correlations, unequal time points, and most critically, missing data (Pahwa & Blair, 2002). Also, once the original cohort is recruited, subsequent data waves are restricted to this original sample and there is invariably attrition over time (Thomas & Nelson, 1990; Yee & Niemeier, 1996). In addition, the cohort is limited to within the region of study with inferences about the population not accurately accounting for regional population changes (e.g. influx of migrants) (Yee & Niemeier, 1996). Measurements used for variables sometimes change over time, making tracking comparisons problematic. There is also a risk that participants become familiar with test items. Data collection over a long period can be expensive and time consuming (Thomas & Nelson, 1990).

Internationally, there are numerous major longitudinal studies, many with an interest in obesity (Centre for Longitudinal Studies, 2008). Some studies include the

Raine Study (The Raine Study, 2010), from which this research is based; The Longitudinal Study of Australian Children (LSAC) (Nicholson, Sanson, & LSAC Research Consortium, 2003); Danish National Birth cohort (Olsen et al., 2001); The Young Finns Study (The Cardiovascular Risk in Young Finns Study, 2008), and the Avon Longitudinal Study of Parents and Children (ALSPAC). The diversity and extensive list of cohort studies demonstrates their importance and value in investigating complex research questions, at individual, family and community levels, as well as across generations.

## **Summary**

The review of current literature paints a somewhat conflicting body of evidence regarding causation of obesity, although many factors have been implicated. It is apparent though that obesity is complex, multi-faceted and individualist. Consequences are clear and deleterious with adverse health, psychological, physical, behavioural, social and economic outcomes. Against this backdrop, this research aimed to investigate this complex problem by using a longitudinal cohort, with multi-factorial design, to examine the interplay between individual, behavioural and environmental obesogenic factors.





## Six Years

"Play is often talked about as if it were a relief from serious learning.  
But for children play is serious learning.  
Play is really the work of childhood."

By Fred Rogers

## **Chapter Three**

### **Methods**

This study is based on data gathered as part of a longitudinal research project, The Western Australian Pregnancy Cohort (Raine) Study. This study design aims to track patterns of behaviour from birth and determine their relative influences on weight status at age 14 years. The study design was determined retrospectively to the data collection.

The methodology for this research is focussed on the statistical analysis of the data, investigating the interrelationships between key variables at each survey point and over time, and weight status at 14 years. This chapter will describe the database from which data are drawn, the sample, measures and their treatment, and the data analysis processes undertaken.

#### **Data**

The data for each variable was cleaned by authorised research officers of the Institute for Child Health Research using standardised protocols. Permission to use the data were provided by the Raine Executive Committee, with selected data provided to research teams through the Raine Study de-identified database.

Selection of obesogenic variables from the Raine database involved identifying individual, behavioural and environmental variables linked to obesity from the literature, as well as other potential factors such as motor competence, physical fitness, and timing of transition to solids. Variables selected required review, and if necessary, transformation to an appropriate form for statistical analysis (e.g. height and weight converted to BMI scores, continuous or categorical variables).

## Sample

The Raine Study enrolled mothers of 2,979 children *in utero* from antenatal clinics at King Edward Memorial Hospital for Women (KEMH), Perth's primary specialist obstetric health care facility, and has followed 2,868 live birth children. Women were enrolled into the project over 30 months from May 1989 to November 1991. Enrolment criteria included gestational age of 16-20 weeks, basic proficiency in English for informed consent, expectation of delivery at KEMH, and intention to remain in the state of Western Australia. All mothers gave written informed consent and the study was approved by the institutional ethics committees. The protocol for the original study has been previously reported describing the antenatal (Newnham et al., 1993) and postnatal periods (Joseph-Bowen, de Klerk, Firth, Kendall, Holt, & Sly, 2004; Oddy et al., 1999).

The Raine Study children have been assessed at birth, and ages 1, 2, 3, 6, 8, 10 and 14 years, with testing of 17 years olds completed early in 2010. The Raine Study families are broadly representative of the Western Australian population<sup>6</sup>: 10.7% of parents being never married (versus 9.8%), 7.5% children were born <37 weeks (versus 6.9%), with a slight overrepresentation children born <2,500g, 8.6 (versus 6.5%). The Raine cohort is well established and there is frequent contact between enrolled families and study organisers (Li, Kendall, Henderson, Downie, Landsborough, & Oddy, 2008). There has been attrition over time in sample size from each survey wave and among variables (refer to Table 4). Overall retention rates are high for each survey wave (92% at 1-year, 74% at 2-years, 85% at 6-years, 82% at 8- and 10-years, and 79% at 14-years), with enthusiasm amongst participants to provide high quality information. At follow-up age 2 years, funding limitations restricted the number of individuals assessed across physical measures, which impacted upon the sample size for BMI.

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<sup>6</sup> Western Australian population was drawn from all births 1989-1992 utilising the Western Australian Maternal and Child Health Research Database at Telethon Institute for Child Health Research (Kendall, 2003).

Table 4

*Raine Study Sample (n) of Participants who Completed All or Part of Each Follow-up*

Mean age at Follow-up	Completed	Deferred	Lost	Withdrawn	Deceased	Total
1	2,441	204	174	21	28	2,868
2	1,988	381	418	51	30	2,868
3	2,280	321	158	79	30	2,868
6	2,237	339	135	127	30	2,868
8	2,140	376	124	198	30	2,868
10	2,047	281	162	348	30	2,868
14	1,860	357	207	412	32	2,868

*Note.* Completed = participant completed all or part of the follow-up; Deferred = participant did not participate in this follow-up but remained part of the cohort; Lost = participant was unable to be contacted for this follow-up, but remained in the cohort for future contact; Withdrawn = participant has withdrawn from the cohort and no further contact was made. (The Raine Study, 2010)

## Measures and Treatment

This research draws on the extensive data base of the longitudinal Raine Study cohort. From a background perspective the following summarises the data collection methods used in the Raine Study (Newnham et al., 1993; The Raine Study, 2010).

The Raine Study began in concept in 1988 by Professor John Newnham, receiving funding through the Raine Foundation in 1989. The original goal for the study was to determine the effects of growth before birth and other factors during pregnancy on outcomes through childhood and into adult life (The Raine Study, 2010). Table 5 provides an overview of the data collected at each follow-up.

Table 5

*Summary Table of Raine Study Data Collection (The Raine Study, 2010)*

Average Age (Time line)	Data collection - key variables
18-20 weeks gestation	Maternal 108 item questionnaire (SES, lifestyle, medical history, environmental exposures) Male partner 30 item questionnaire (physical size, education, occupation, environmental exposures)
34 weeks gestation	Maternal questionnaire (exposures during 18-34 weeks)
Birth (1989-1991)	Hospital notes (pregnancy, deliveries, neonatal outcomes) N=2801
1-year-olds (1990 -)	Caregiver questionnaire (child's health, growth and development since birth; parental questions) Physical examination (height, weight, blood pressure, and physical health and development assessment)
2-year-olds	Caregiver questionnaire (child's health, growth and development since age one; parental questions) Physical examination (height, weight, blood pressure, and physical health and development assessment)
3-year-olds (-1994)	Caregiver questionnaire (child's health, growth and development since age two; parental questions) Physical examination (height, weight, blood pressure, and physical health and development assessment)
6-year-olds (1994-1996)	Caregiver questionnaire (child's health, growth and development since age three; parental questions, family history of asthma, eczema and hay fever) Physical examination (height, weight, blood pressure, physical health and development assessment, spirometry and methacholine challenge or bronchodilator response test, skin prick tests for atopy and blood sample tests)

Average Age (Time line)	Data collection - key variables
8-year-olds (1996-1997)	Caregiver questionnaire (child's health, growth and development since age five; parental questions, family history of cardiovascular disease, fat intake and physical activity), short fat questionnaire / physical activity. Physical examination (height, weight, blood pressure and blood sample tests for both parent and child), spirometry and blood pressure assessment during a sub-maximal bicycle.
10-year-olds (1997-2000)	Caregiver questionnaire (child's home life, leisure activities, schooling, language development, behaviour and general health since age eight); parental questions (health and happiness), parent-teacher questionnaire (child's mental health, cognitive, language and neuromuscular development) and school report on academic achievement. Family history of cardiovascular disease, fat intake and physical activity), short fat questionnaire / physical activity. Physical examination (height, weight, blood pressure)
14-year-olds (2003-2006)	Caregiver questionnaire (similar to previous, since age ten); food frequency questionnaire. Child questionnaire (self-perceptions, risk-taking behaviour). Principal – teacher questionnaire (school environment, child participation, academic achievement). Physical examination (height, weight, blood pressure – child and caregiver). Stress test (PWC170), lung function test, bronchial responsiveness test, skin prick test for allergies, urine sample, 7-day pedometer assessment and diary of physical activity, blood tests and postural body images)

A methodical review of all variables collected as part of the Raine Study was conducted. Based on the literature, a subset of individual, behavioural and environmental variables was selected as obesogenic variables of interest.

Variables collected as part of the Raine study, have been acquired using standard protocols, by specialist trained research staff at the Institute for Child Health Research, under the guidance of specific academic research teams and institutions. This study involved the use of variables that were already cleaned, scored or categorised and available via a de-identified data base. The variables used in this study are discussed individually below, with any additional transformation to variables detailed. Where possible, protocols used by the Raine Study team for physical assessments are provided.

### ***Age and time.***

Age and time variables were used for this study as recommended by Singer and Willet (2003). Age was reported in the smallest meaningful unit, in this study, months. Age in months was calculated at each follow-up by computing the difference between the date of physical assessment for that follow-up and the child's date of birth.

Time was coded according to the average age in years of the cohort at each measurement wave i.e. 0, 1, 2, 3, 6, 8, 10.5, and 14. In this longitudinal sample, "occasional creep – over time" (Singer & Willet, 2003 p.141) was found where the actual ages exceeded that originally planned. In this study the year five follow-up occurred when individuals were an average of 6 years of age, the 10 year follow-up occurred when individuals were an average of 10.5 years of age, and 13 year follow-up occurred when individuals were an average of 14 years of age. This variable forms a time-structured predictor that records time on a scale comparable numerically to the individual's age, that is, the values indicate the individual's expected age on each follow-up assessment (Singer & Willet, 2003).

### **Individual factors.**

Broadly, individual factors considered for this study included gestational age, anthropometric measures, adiposity rebound, diet, developmental milestones and motor competence, physical fitness, and puberty. Some factors had multiple indicators.

### ***Gestational age.***

Gestational age is the duration of time, measured in weeks, that the foetus has spent developing in utero. This duration is estimated using the date of the mother's last menstrual period. At birth, the gestational age (gweeks) was used as a marker for pre-term birth exclusion from the sample, with pre term births determined to be a gestational age of less than 37 weeks.

### ***Anthropometric measures.***

Anthropometric measures were taken by a small group of extensively trained staff of the Telethon Institute of Child Health Research using scientific protocols, with intra and inter-rater reliability established at the beginning of each follow-up survey. Depending upon anthropometric measurements assessed at follow-up, the sequence of measures taken were Biacromial<sup>7</sup> measurement, waist measurement, hip measurement, height, weight, and arm circumference<sup>7</sup>.

The neonatal examination was conducted between 24 and 72 hours following birth. Length was measured by two people using the Harpenden Neonatometer (Holtain Ltd. Crosswell, United Kingdom) to the nearest 0.1cm. Newborns and infants (follow-up one year) were laid in supine position, with their head held by one person against a curved head plate in mid-line. The other person stretched the legs straight, knees held together, ankle flexed at right angles to the lower leg, moving the mobile plate to rest against the baby's feet. Weight was measured to the

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<sup>7</sup> This measure was not used in this research study.



nearest 100g, using calibrated hospital scales at birth and a Wedderburn digital chair scales in follow-up one year.

In follow-ups year two and on, where appropriate, children were standing in the anatomical position, palms facing forward. Each area was measured at least twice in sequence with measures within one centimetre. Unless stated, measures were taken at expiration. Height was measured using a Holtain stadiometer (Holtain Ltd. Crosswell, United Kingdom) with shoes off, and heels, bottom and head against the board. The chin was positioned to straighten the neck and the measure taken with a breath intake. Weight was measured with children wearing light clothing (running shorts and singlet top), to the nearest 100g, using a Wedderburn digital chair scales (Wedderburn, Australia).

#### *Skin-fold measurement.*

Skin-fold measurements were assessed at follow-ups 1, 2, 3, 6 and 10 years. The protocol involved children standing in the anatomical position, palms facing forward. All measurements were taken on the individual's left side. A skin fold was obtained by pinching, using a slight rolling action of the left thumb and index finger. The skin fold was raised at the marked site with firm pressure and held throughout the measurement. A Holtain skin fold calliper was applied 1cm from finger/thumb, released and after a two second wait, the measurement read. Measurements were taken in sequence (not consecutively), with a minimum of two measures with less than 10% difference. Additional measures were taken as necessary. The sequence for measurement was subscapular, triceps, suprailiac, and mid abdominal with details of protocol detailed at Table 6 (Telethon Institute for Child Health Research, n.d.).

Table 6

*Protocol for Skin-fold Site Identification and Procedure*

Skin fold site	Site identification	Procedure
Subscapular	Site is 1cm below the lower angle of the left scapular. The skin fold runs downward at an angle of about 45 degrees from the horizontal.	Pinch left fold immediately beneath the apex of the left scapula, with the arm hanging at the subject's side.
Triceps	The skin fold is measured on the back of the left upper arm.	Pinch a vertical skin fold on the posterior aspect of the upper arm, half way between the olecranon and acromium, with the arm hanging by the subject's side.
Suprailiac	Leading edge of the iliac crest ... or ...Feel laterally for the iliac crest and follow along anteriorly.	Pinch skin fold at a 45 degree angle immediately above the left anterior superior iliac spine.
Abdominal	5cms to the left of the mid point of the navel.	Pinch vertical skin fold and raise.

*Note.* Adapted from "Skin folds measurement sites," by Telethon Institute for Child Health Research. (n.d.), *The Western Australian Pregnancy Cohort (Raine) Study* [Protocol]. Perth.

*Waist girth.*

At age 14 years, waist girth measures were taken. For waist girth, the individual held the measuring tape in place over the umbilicus on the skin in two places. The tape was kept horizontal and firm to the skin. The research assistant moved the tape around the body ensuring the tape was horizontal and in the same plane at the back before recording the measurement in centimetres.

Waist-height Ratio (WHtR) was calculated using the formula waist girth (cm)/height (cm). WHtR cut-offs were selected to represent the cut-offs for BMI as proposed by Kahn and colleagues (2005), namely  $\geq 0.539$  obese (BMI  $\geq 30$ ), 0.490-0.539 at risk or overweight (BMI 25-30) and  $< 0.490$  (normal) (BMI  $< 25$ ), with the McCarthy and

Ashwell (2006) 0.5 cut-off used for the two category comparison normal weight and overweight.

### *Hip girth.*

Hip girth was recorded at age 14 years. Individuals were dressed in light clothing. The research assistant stood to the side of the individual and placed the tape measure around the body, over the widest part of the buttocks. The individual held the tape measure firm and horizontal in two places at the front of their body, while a measurement was taken.

Waist-hip Ratio (WHR) was calculated using the formula waist girth (cm) / hip girth (cm). Age and gender adjusted waist girth cut-offs between normal and overweight were calculated according to those reported in Taylor and colleagues (Taylor, Jones, Williams, & Goulding, 2000).

### *Body mass index (BMI).*

At each follow-up BMI was calculated from measured height and weight using the formula weight (kg)/height (m)<sup>2</sup>. At birth and year one, a proxy for BMI was used (weight/length<sup>2</sup>) (Rolland-Cachera, Deheeger, Akrouit, & Bellisle, 1995). At age 14 years, BMI cut-off points equivalent to 25 and 30, adjusted for age and gender, classified the participants as normal weight, overweight or obese as defined by the IOTF criteria (Cole et al., 2000).

### *Adiposity rebound.*

Adiposity rebound, defined as the last minimum (nadir) BMI before the continuous increase with age (Rolland-Cachera et al., 1984), was determined in a subset of individuals ( $n=173$ ) for whom a complete set of BMI data were available. This small sample was due to limited anthropometric measurements taken at the two year follow-up (insufficient funding), but is similar in size to another study (Rolland-Cachera et al., 1995). Adiposity rebound was based on the child's age in months.

Given the wide range of participant ages in the three and six year follow-ups, data were available for age 3-4 years, and age 5 ½-6 years leaving a small gap between ages 4 and 5½ years without data. This is pertinent to the calculation of adiposity rebound. BMI, and age at nadir were calculated for both the raw BMI and predicted BMI (based on the longitudinal LMM which adjusts for age, gender, weight status and gestational age and is discussed later).

### ***Puberty.***

Pubertal data were collected at age 14 years and included the Tanner Stages of pubic hair development for males and females, and for females, Tanner Stages of breast development (Tanner, 1962) and the date of first menstruation. The self-assessment of sexual maturation (Tanner, 1962) has been shown to have excellent agreement with physical assessment by medical professionals (Duke et al., 1980).

For girls, menstruation was determined as the age at which they experienced their first menstruation. This was calculated by subtracting their birth date (day/month/year) from the reported date of their first menstruation (day/month/year), with a reported measure in years. Female breast development was reported according to the five stages of development (Tanner, 1962).

During the data collection for the Tanner Stages an error was detected where there were incorrect codes indicated on the Tanner image from which the adolescents made a selection of their pubertal developmental stage. In effect, codes of 1, 2, 3, and 4 that corresponded with images 1, 2, 3, and 4, were instead coded 2, 3, 4 and 5. It was believed that less than a third of the sample was affected by this error ( $n=334$ ). All affected individual's data for this variable that were labelled *doubtful* were re-coded for this study as missing. Pubic hair development was reported according to five stages of development (Tanner, 1962).

## ***Diet.***

Diet variables available for analysis for this study included early infant feeding, self-reported adolescent fruit and vegetable intake at age 14 years, and fat intake at ages 8 years (parent report) and 14 years (self-report).

### ***Early infant feeding.***

Parent recall to specific questions in follow-ups conducted at one, two and three years collected information pertaining to early infant feeding. Mothers reported the age breast feeding stopped (in months), age milk other than breast milk was introduced (in months), and age they first gave their baby solids (weaning) (in months). Milk other than breast milk typically included infant formula (67.7%), cow (87.7%) or soy milk (20.8%). The age breastfeeding stopped, the age at which milk other than breast milk was introduced, and weaning were determined from the mother's diary of early feeding milestones, an interview with the study nurse, and survey questions at later follow-ups.

Like Burke and colleagues (2005) who also analysed early feeding data for this cohort, no distinction between exclusive or partial breastfeeding was made. The variables for age breastfeeding stopped, age other milk introduced, and weaning were categorized using a 4-month cut-point (categories  $\leq 4$  months and  $>4$  months). This cut-point split the cohort almost equally, was the World Health Organisation recommendation for duration of exclusive breastfeeding at the time of that data being collected (1990-1993), and has been reported by others for this cohort (Burke et al., 2005; Oddy, Li, Landsborough, Kendall, Henderson, & Downie, 2006a).

### ***Fruit and vegetable intake.***

Adolescents responded to a survey item asking them to report how often they ate fruit (fresh or canned, but excluding dried fruit, juices, fruit bars or frozen desserts), and vegetables (fresh, frozen, canned or salads). Response categories included

*rarely / never, 1-2 times a month, 1-2 times a week, 3-5 times a week, and 6 or more times a week.*

### ***Fat intake.***

At follow-up age 8 years, parents reported their child intake on the following identified fat intake items: *food with batter coating, gravy or sauces, add butter to food, fried or roasted veg, eat sausages, bacon; chips/french fries; pastries and cakes; chocolate intake; crisps intake; cream intake; icecream intake; cheese intake; skin on chicken; and fat on meat.* Responses were categorised *never (0), less than once a week (1), 1-2 times a week (2), 3-5 times a week (3), and 6 or more times a week (4).* A derived fat intake score was computed, as the listwise sum score of these categories (numbers in brackets to categories above), with a score range from 0- 56.

At follow-up 14 years, adolescents reported their fat intake. Most items were similar to follow-up eight years, except for ***fried food with batter coating, hard cheese intake,*** and a rewording and response categories for chicken and meat fat. The question was reworded to: *how much fat on meat do you usually eat, and how much skin on chicken do you usually eat,* with response categories *none (0), some (1), and most or all (2).* A derived fat intake score was computed as the listwise sum score of these categories (as for follow-up 8 years), with a score range from 0-52.

### ***Developmental milestones and motor competence.***

Developmental milestones and motor competence was assessed in this study by parent reported developmental milestones (1-3 years), Denver Developmental Screening Test, 2<sup>nd</sup> Edition (1-3 years) (Frankenburg, Dodds, Archer, Shapiro, & Bresnick, 1992), Infant Monitoring Questionnaire (1-3 years) (Bricker & Squires, 1989), parent perceived motor skills (10 years), and the McCarron Assessment of Neuromuscular Development (10 and 14 years) (McCarron, 1997).

### *Parent reported developmental milestones.*

Questionnaire items at follow-up age 1 year evaluated the timing of developmental milestones (months) based on parent recall. Items included when the child first smiled (weeks), sat up, babbled, crawled, stood, tottered, walked, spoke their first word, showed handedness, and lost their first tooth. Questions on when the child first walked, spoke their first word and showed handedness were repeated at follow-up age 2 years. Data were merged for these items across the 2 years. If data were present at the 1 year follow-up, the year 2 data were ignored; otherwise, if data were missing at the 1 year follow-up, the year 2 data were used.

### *Denver Developmental Screening Test, 2<sup>nd</sup> edition (Denver II).*

The Denver II assessment is used to screen for possible developmental problems in pre-school children (Frankenburg et al., 1992). In this study it was conducted at follow-up ages 1, 2 and 3 years (Newnham, Doherty, Kendall, Zubrick, Landau, & Stanley, 2004). It reported on gross motor, fine motor, personal social, language, and overall development (age 3 years only). Categories included *uncooperative*, *fail*, *suspect*, *adequate*, and *normal*. The validity and reliability of the measure is unclear, although the revised version (Denver II) was re-standardized and is considered to be a quick screening tool (Glascoe & Frankenburg, 2002). The test-retest reliability (.90) and inter-rater reliability (.99) is excellent. Validity of the tool though is weak, and tends to result in a high rate of false positives (Naar-King, Ellis, & Frey, 2004).

### *Infant Monitoring Questionnaire (IMQ).*

The IMQ is a series of parent completed questionnaire about motor, communication, social/personal and adaptive development (Bricker & Squires, 1989) and screens for developmental competence (Houck, 1999). Validity and reliability of the IMQ is good. It has good agreement with standardized tests (86% - 91%), low under- (6%) and over- (6%) screening rates, high inter-observer reliability (87%) and test-retest agreement (91%) (Bricker & Squires, 1989), and more recently

was found as an accurate screening measure for identifying Australian infants (aged 0-18 months ) at risk of developmental delay (Dixon, Badawi, French, & Kurinczuk, 2009).

Parent responses were obtained at follow-ups 1, 2 and 3 years (Newnham et al., 2004). For this study, only items related to gross motor (1, 2 and 3 years), fine motor (1, 2 and 3 years), and overall development (2 and 3 years) were analysed. Response categories for gross and fine motor development were *not yet*, *sometimes* and *yes*. Overall development questions were a *no* or *yes* response.

### *Parent perceived child motor skills.*

At follow-up age 10 years, parents rated their child's motor skills in locomotion (running, jumping, hopping, skipping, dodging); object control (throwing, catching, kicking, striking); balance (biking, balancing); and overall coordination and development progress. Categories for responses were *poor*, *below average*, *average*, *above average* and *excellent*. For most items, sample size for response *poor* was less than 10, hence category *poor* was merged with *below average* for analyses to create four categories.

### *McCarron Assessment of Neuromuscular Development (MAND).*

The MAND (McCarron, 1997) was used to assess motor competence at age 10 and 14 years. The test involved five fine motor tasks (beads in box, beads on rod, finger tapping, nut and bolt and rod slide) and five gross motor tasks (hand strength, finger-nose-finger, jumping, heel-toe walk, standing on one foot). The test has been shown to have good validity and reliability (McCarron, 1997). Evidence of the content, construct, predictive and concurrent validity of the test is provided by McCarron (1997). Tan, Parker and Larkin (2001) provided further evidence of the concurrent validity of the MAND. Performance rankings for the MAND were highly correlated with the Bruininks-Oseretsky Test of Motor Proficiency-Short Form ( $r =$



.86) and Movement ABC (.86) in a sample of 69 children aged between 4 and 10 years.

Based on absolute quantitative and qualitative test performance, raw scores were awarded. These raw scores were adjusted for age, then summed to yield an overall scaled score for fine motor competence, gross motor competence, and total motor competence. The scaled total score was converted to a Neuromuscular Development Index (NDI) score, which is normally distributed with a mean of 100 and a standard deviation of 15 (Hands et al., 2008) . In addition, a three category variable was derived, low ( $\leq 85$ ), medium (86-114) and high ( $\geq 115$ ) motor competence based on the McCarron (1997) NDI cut-points of  $\leq 85$  below normal performance, and 85-115 normal performance.

### ***Physical fitness.***

Objective measures of physical fitness included aerobic fitness, muscle endurance, muscle strength, and flexibility at age 14 years.

### ***Aerobic fitness.***

Aerobic fitness was represented by the physical working capacity 170 (PWC 170) Monark cycle ergometer test at follow-up 14 years (Hands et al., 2008). Although this test was also conducted at the 8-year-old follow-up, essential information for the calculation of the PWC 170 was missing, and unavailable for this study.

Aerobic fitness is an index of oxygen delivery and oxidative mechanisms of muscles during exercise (Armstrong, Welsman, Nevill, & Kirby, 1999), with PWC 170 providing an estimate of maximum oxygen consumption ( $VO_2$  Max). At present, within the literature, both absolute and relative PWC 170 (Physical Working Capacity at an extrapolated heart rate of 170 beats per minute) measures are reported to be suitable in adolescents (American College of Sports Medicine, 2009;

Armstrong et al., 1999; Cleland et al., 2009; Hands et al., 2008), with either methodology used in respect to studies of obesity (Cleland et al., 2009; Hands et al., 2008). In adolescents the predictive ability of VO<sub>2</sub> Max from PWC 170 is good ( $r=.71$  and  $.70$  for females and males respectively (Rowland, Rambusch, Staab, Unnithan, & Siconolfi, 1993); and  $r=.84$  (Boreham, Paliczka, & Nichols, 1990), although there is wide variability (Rowland et al., 1993). An evaluation by Armstrong and colleagues (1999) demonstrated that it was an inappropriate assumption that scaling using body mass or other mass components controls adequately for body size differences in children and adolescents, with the relationship between VO<sub>2</sub>max and PWC 170 weakened ( $r=.65$  and  $r=.48$  for females and males respectively) when adjusted for body weight (Rowland et al., 1993). Age, gender and pubertal development are instead strong confounders to the measure of aerobic fitness (Armstrong et al., 1999). Further it is argued that excess body weight does not necessarily mean maximum oxygen consumption is reduced, and that VO<sub>2</sub> max should be considered separately (Goran et al., 2000).

Adjustment of PWC 170 for body mass was not made, with this raw measure considered an absolute measure. This decision was based on considerations of mixed adjustment usage within the literature, evidence to suggest that adjustment for body mass does not adequately control for body size differences in children and adolescents (Armstrong et al., 1999), and that the outcome measure for this study is BMI, with an adjustment for body mass considered to be made twice.

### *Muscle endurance.*

Abdominal muscle endurance was evaluated using a curl-ups test. Participants lay with their back and head on a mat, knees bent to 90 degrees and feet flat on the floor. The arms were outstretched, grasping a pencil in the fists and resting on the thighs. The examiner placed a ruler over the peak of the knees to act as a target. No pressure was placed on participants' feet or knees. At a cadence of 20 curls per minute, set by a metronome, participants repeatedly curled up to touch their fingers on the ruler and returned to the starting position. The test ended after 60

curls or if the following occurred for two consecutive curls: curls were not in time with the beat, the grip on the pencil was released, one or both soles of the feet left the floor, one or both fists did not touch the ruler, the head did not make contact with the mat, or the arms/elbows were bent. The number of completed curls was counted (Hands et al., 2008).

### *Muscle strength.*

Upper body strength was measured by a chest pass test. Participants sat on the floor with their back, buttocks, shoulder and head flat against a wall, and their legs extended with the feet together. The examiner placed a hoop on top of participants' toes. Participants held a basketball with elbows touching the wall, and the ball touching their chest, and then used a two-handed chest to pass the ball through the top of the hoop. Distance was measured from the wall to the landing point and the better of two trials was recorded (Hands et al., 2008).

### *Flexibility.*

Two tests of flexibility were conducted: hip flexibility (sit and reach), and shoulder flexibility (shoulder stretch). The Figure-Finder Flex tester was used to measure hip flexibility. To measure the right leg, with shoes removed, the participants sat on the floor and extended their right leg so that their right foot was placed flat against a 30cm high box placed against a wall. The opposite knee was bent, with the sole against the medial border of the extended knee. With palms prone and touching, participants reached as far forward as possible, holding for 2–3 seconds, with no jerkiness, unevenness of hands or bending of knees. The meter rule used for measurement projected from the box toward participants, with the 0 cm mark nearest to the participant and the 50cm mark at the front edge of the box. The 23cm mark was located above the toes. The better of two trials was recorded. This was then repeated on the left leg and finally with both feet placed against the box together (Hands et al., 2008).

The shoulder stretch test involved participants reaching their right hand over and behind their right shoulder, and their left hand behind their back toward the right shoulder, attempting to touch their hands together. The stretch was graded as *able* if participants could touch at least fingertips together. This was repeated on the opposite side (Hands et al., 2008).

### **Behavioural factors.**

Broadly, behavioural factors considered for this study included physical activity behaviours, sedentary behaviour, self concept, and parent reported progress. For some factors there were multiple indicators.

#### ***Physical activity behaviours.***

Physical activity behaviours were represented by frequency of visits to the park in early childhood (1-3 years), parent reported activity levels in late childhood (6, 8 and 10 years), physical activity level (14 years), and attitudes and values to physical activity.

#### ***Frequency of visits to a park or playground.***

At follow-ups one, two and three years, parents reported the frequency of visits to the park or playground with their children. Category response frequencies included *never*, *seldom* (<1/month), *occasionally* (<1/week), *often* (>1/week) and *every day*. This type of survey item has been shown to have moderate reliability and validity (Veitch, Salmon, & Ball, 2009).

#### ***Parent reported activity levels at ages 6, 8 and 10 years.***

Organised sport participation was assessed at follow-up ages 6 and 8 years with a *yes* or *no* response to participation. Organised sport was defined in the survey as “at school or with a club”. Similarly organised activity at age 6 years was assessed with a *yes* or *no* response to participation and was defined in the survey as “music, dancing, kindy gym, other clubs”. At age 8 years, activity level responses were

categorised as *sedentary or little*, *slightly active* and *active*. Activity was defined in the survey as:

sedentary or little	gets very little exercise, ... spends most of (their) time sitting, watching TV, or reading; ...
slightly active	gets some exercise, ... spends more time in active play than reading or watching TV; ...
active	is involved in an organised activity 2 or 3 times per week or walks/runs 2km or more per day”.

At age 10 years, questionnaire items asked to state the activity involved in at each week day, the time spent in minutes, and whether the activity was in the local community (*yes or no*). Activities reported were then coded into activity levels *no*, *light*, *moderate* or *vigorous* based on the Raine Protocol for Physical Activity Categories (Appendix B). These protocols were based on those used in the WA CAPANS study (Hands et al., 2004).

From this several variables were computed:

- Vigorous activity during the week – count of all reported vigorous activity during the week Monday to Friday.
- Moderate activity during the week – count of all reported moderate activity during the week Monday to Friday.
- Light activity during the week – count of all reported light activity during the week Monday to Friday.
- No activity during the week – count of all reported no activity during the week Monday to Friday.

The same four variables were computed for weekend activity levels. That is, for Saturday and Sunday a total count was computed for vigorous, moderate, light and no activity. A total activity count was computed, total vigorous, total moderate and total light activity for the whole week, that is, Monday to Sunday. Activity in the local community was calculated for weekday (Monday to Friday), weekend

(Saturday and Sunday), and total (Monday to Sunday) activity levels. In addition, a total activity time was computed, a sum of activity time in minutes from Monday to Sunday.

### *Physical activity level.*

Actual physical activity level was measured by pedometer step count at age 14 years. The step count measure demonstrates convergent (Tudor-Locke, Williams, Reis, & Pluto, 2002) and construct (Tudor-Locke, Williams, Reis, & Pluto, 2004b) validity, with the protocol established as reliable (Hands et al., 2008). Physical activity levels determined by pedometer have good intra-individual variability (Tudor-Locke et al., 2004a) and do not appear to be affected by participant reactivity (Rowe, Mahar, Raedeke & Lore, 2004).

Pedometer steps were recorded over a seven day period, with adolescents wearing the Yamax Digiwalker SW200 pedometers (Yamasa Tokei Keiki Co. Ltd., Tokyo, Japan) on the right hip. Step counts were recorded daily via an electronic or paper diary. Daily step counts below 1,000 or above 44,000 were removed (Hands et al., 2008).

Continuous forms of the variables investigated included a weekday average step count (mean daily step count per weekday) and weekend average step count (mean daily step count per weekend). However the mean daily step count determined for those participants recording feasible step counts for a minimum of 4 days, including at least one weekend day was the primary measure of interest for modelling analyses (mean daily step count per week). This method has been shown to be strongly correlated with 7-day averages (Ridley, Olds, Hands, Larkin, & Parker, 2009). Gender specific categories determined by tertiles, mean step count cut-point, median step count cut-point, and a 12,000 step count cut-point were also investigated.

### *Physical activity attitudes and values.*

At age 14 years the questionnaire included multi-item questions in areas of encouragement and support to exercise (11 items); physical activity and physical education (13 items); effects of physical activity (15 items); importance of physical activity (15 items); excuses for not increasing physical activity in the future (15 items); and other physical activity related perceptions (6 items). Responses were according to a Likert scale, differing dependent upon question. For effects and importance of physical activity the Likert categories *extremely unlikely* and *very unlikely* were combined into one category, as well as *extremely likely* and *very likely*, due to small sample sizes, resulting in a five category variable. Likewise, excuses for not increasing physical activity, the Likert categories', *applies strongly* and *applies very strongly* were combined into one category, resulting in a four category variable.

A derived score was calculated for variables asking about encouragement and support to exercise (11 items, score 0-4), physical activity and physical education (13 items, score 0-3); effects of physical activity (15 items, score 0-6); importance of physical activity (15 items, score 0-6); excuses for not increasing physical activity in the future (15 items, score 0-4). The derived score was based on the mean of the sum of each variable's item. The mean was computed for each using listwise selection (that is, no missing data points). Listwise assessment found the number of cases with missing data points for these variables were very few, with a paired sample t-test reporting no significant differences between the listwise derived mean score versus the mean score . The listwise derived score was used for further analysis.

### *Sedentary behaviour.*

Screen time was used as a proxy for sedentary behaviour. Questionnaire responses have moderate reliability with validity results suggesting under-reporting (Clark, Sugiyama, Healy, Salmon, Dunstan, & Owen, 2009). Measures included usual time spent watching television, categorised as *none*, *<3 hrs/wk*, *up to 1 hr/day (3-7*

*hrs/wk*), *1-2 hrs/day (7-14 hrs/wk)*, *2-3 hrs/day (14-21 hrs/wk)*, and *>3 hrs/day (>21 hrs/wk)* at ages 6, 8 and 10 years. At 10 years the question changed slightly to include “TV and/or playing computer games”. At follow-up 14 years, the question changed further. Categories were *none, up to 1 hr/day (3-6 hrs/wk)*, *1-2 hrs/day (7-13 hrs/wk)*, *2-3 hrs/day (14-21 hrs/wk)*, and *>4 hrs/day (>21 hrs/wk)*. The wording changed to watching “TV or videos”. An additional question on usual time spent “playing video games or computer games, use the internet or chat online” was added with the same categories for responses.

A dichotomous television variable was created using the Australian National Television Watching guidelines of no more than 2 hours per day (Australian Government Department of Health and Ageing, 2004), with a 2 hours cut-point (i.e. the first three categories at ages 6, 8 and 10 years and the first two categories at 14 years, were collapsed into one category). At 14 years, a total screen time variable was created combining time spent in both television and computer use with categories *none, up to 1 hr/day, 1-2 hrs/day, 3-4 hrs/day (14-21 hrs/wk)*, and *>4hrs/day*.

### ***Self concept.***

Self concept was represented by the Self Perception Profile for adolescents (SPPA) (Harter, 1988) and was used to help evaluate the relationship between the individual’s personal beliefs and subsequent behaviour, particularly related to physical activity behaviour. All nine domains were included for analysis. These were global self worth, and eight specific domains: physical appearance, athletic competence, scholastic competence, social acceptance, behavioural conduct, romantic appeal, close friendship and job competence. Each domain was scored on a continuous scale of one to four. SPPA has been shown to have good reliability and factor validity (Harter, 1988). Recently SPPA has been shown to be representative of the Australian population, reporting good factor validity and reliability in the Raine Cohort sample (Rose, Larkin, & Hands, 2010).



### ***Parent reported academic, social and behavioural progress.***

Parent questionnaire responses at age 10 years evaluated parent perceived academic performance categorised as *poor, below average, average, very good, or excellent*. A 5-point Likert scale (*very satisfied, satisfied, neither, dissatisfied, and very dissatisfied*) was reported for parent satisfaction with child's progress in learning skills, social and behaviour.

### **Environmental factors.**

Broadly, environmental factors considered for this study included socioeconomic status, parental influences, and the built environment. For all factors there were multiple indicators.

### ***Socioeconomic status (SES).***

SES data were collected via questionnaire at birth and each follow-up. SES data included Socioeconomic Index For Area, ethnicity, income, parent education, father's occupation, employment and life stress.

### ***Socioeconomic Index For Area (SEIFA).***

The Australian Bureau of Statistics (ABS) (Australian Bureau of Statistics, 2007a) reported four indexes in its 2001 census data which related to socioeconomic aspects of geographic areas. The index of advantage/disadvantage is a new index that reports the advantage (high scores) to disadvantage (low scores) continuum. This index was considered the most appropriate for use with weight status in this study, used as an indicator of community SES.

The Index of Relative Socioeconomic Advantage / Disadvantage is an Australian wide index encompassing both rural and metropolitan areas. Areas with a relatively high proportion of people on high income and involved in skilled work will report high scores. While areas with a high proportion of people on low income

and involved in unskilled work will report low scores. This index is intended as a summary of area characteristics only (Australian Bureau of Statistics, 2007a).

The child's postcode at the 14 year follow-up was used to determine the corresponding SEIFA score from the ABS tables (Australian Bureau of Statistics, 2007a). The SEIFA index was investigated as a continuous variable, and also categorised into low ( $\leq 25$  centile), medium ( $>25$ - $<75$  centile) and high ( $\geq 75$  centile).

### *Parent ethnicity.*

Parent ethnicity included survey response categories *other, Aboriginal, Polynesian, Vietnamese, Chinese, Indian* and *Caucasian*. For both males and females, non-Caucasians represented only 9% of the sample. Hence a dichotomous ethnicity variable Caucasian and non-Caucasian was used.

### *Income.*

Family income was used as an indicator of individual family wealth, but did not consider levels of debt, and was collected at birth and at each follow-up. Parents reported their family income bracket in survey response. Income brackets differed over the eight follow-ups and are summarised in Table 7. However, a low income cut-point was used as previously reported (Kozyrskyj, Kendall, Jacoby, Sly, & Zubrick, 2009). Cut-points for low income at each follow-up were: Birth and age 1 year -  $< \$24,000$ , age 2 years -  $< \$27,000$ , age 3 and 6 years -  $< \$26,000$ , and age 8, 10 and 14 years -  $< \$30,000$ .

### *Parent education.*

Mother's and father's highest level of education responses were categorised as: *none, trade certificate or apprenticeship, professional registration (non-degree), college diploma or degree, University degree*, and *other*. The reported education level at the birth survey was used for this study.

### *Father's occupation.*

Father's occupation response were categorised as: *managerial, professional, par-professional, trade, clerical, sales, plant operator, and labourer*. The reported father's occupation at the birth survey was used for this study.

Table 7

#### *Family Income Brackets across Follow-ups*

Income (\$AUS)	Birth	Ages 1,2,3 & 6	Ages 8, 10 &14	Study Category
Less 7,000	1			2
Less 8,000		0	0	2
7K-11,999	2			2
8K-13,999		1		2
8K-15,999			1	2
12k-23,999	3			3
14k-26,999		2		3
16k-24,999			2	3
24K-35,999	4			4
27K-40,999		3		4
25K-29,999			3	4
>36K	5			5
>41K		4		5
30K-34,999			4	4
35K-39,999			5	4
40K-49,999			6	5
50K-59,999			7	6 (or 5)
60K-69,999			8	7 (or 5)
>70K			9	8 (or 5)

*Note.* K=1,000. Less than \$7,000 and less than \$8,000 had very small sample sizes so were merged with the next category.

### *Employment.*

Family minimum work hours were investigated in terms of whether having a non-working or part-time parent at home was related with the child's weight status. The minimum hours were calculated using the minimum hours of either the mother or partner. At ages 1, 2 and 3 years there were no partner hours recorded, so those data represent the mother only. When the father's data were missing, then mother's data were used. When mother's data were missing, then data were coded missing. Minimum work hours were categorised as zero hours, 1-29 hours and 30 or more hours.

Family employment assessed separately whether the mother and partner were working using a *yes* or *no* response at birth and ages 1, 2, 3, 6 and 8 years. At 10 years the question changed to a *no* and multi-option *yes* (work for payment, unpaid work or other unpaid work) response. For this analysis the multi-*yes* response was converted to a simple *yes* response. The variable was converted into three categories which investigated differences between both mother and partner not working, either mother or partner working, and both mother and partner working. Mother's work was a *yes* or *no* response. Mother's work hours was the nominated hours of work per week reported.

### *Life stress.*

Life stress item responses (*yes* or *no*) were collected at 18 and 34 weeks gestation, and all follow-up years. Parents were asked whether any of the following stress events had happened to them in the past year. The stress items included pregnancy problems, death of a close relative, death of a close friend, separation or divorce, marital problems, problems with children, own job loss, partner's job loss, money problems, residential move, and other. A count of life stress events was computed for individuals with all 11 items answered, along with a dichotomous variable, <3 stress events and ≥3 stress events.

### ***Parental influences.***

Parental influences were reflected through parent weight and BMI prior to and across follow-ups, maternal smoking during pregnancy, as well as parenting styles in later childhood.

### ***Parent weight.***

Mother's and father's birth weight were reported by parent recall at the initial survey. Self reported height and weight was collected by survey for the parents, Mother's pre-pregnancy and at their child's age 6, 8 and 14 year follow-up; and father's at child's birth and child's age 8 year follow-up. BMI was calculated for each using self-reported height and weight and the formula  $\text{weight (kg)}/\text{height (m)}^2$  (Cole et al., 2000).

### ***Maternal smoking during pregnancy.***

Mother's smoking behaviour during pregnancy was assessed by a *yes* or *no* response.

### ***Parenting styles.***

At the 10 year follow-up parents completed the Parent Scale (O'Leary, 2003). The parenting scale score was derived from the individual item scores reported according to the following scoring instructions (O'Leary, 2007).

- Each item receives a 1-7 score, where seven is the "ineffective" end of the item. (i.e. items 2, 3, 6, 9, 10, 13, 14, 17, 19, 20, 23, 26, 27, 30 score seven on the left, the others on the right).
- Total score is the average of all items (listwise).
- Factor score, average the responses on the items on that factor (listwise):
- Laxness: 7, 8, 12, 15, 16, 19, 20, 21, 24, 26, 30 (11 items).
- Over-reactivity: 3, 6, 9, 10, 14, 17, 18, 22, 25, 28 (10 items).

- Verbosity: 2, 4, 7, 9, 11, 23, 29 (7 items).
- Items not on a factor: 1, 5, 13, 27 (4 items).

At age 14 years, adolescents rated their own parents' parenting, during the last 6 months, selecting categories *never*, *sometimes*, *often* and *very often* for 11 items. Each item was analysed separately and included *parents smile at me*, *parents forget rules*, *parent praise me*, *parents nag me about little things*, *parents use rules only when it suits them*, *parents tell me they appreciate me*, *parents threaten more than do*, *parents speak of good things I do*, *parents enforce or don't depending upon mood*, *parents threaten or hit me*, and *parents proud of me*.

### ***Built environment.***

Proxies for the built environment related indirectly to obesity through opportunities to be physically active. They included home environment factors in the early years, and adolescent school environment factors.

### ***Home environment.***

The influence of the home environment on opportunities to be active was investigated by factors home swimming pool, outdoor play space, and proximity to the park or playground. At ages 1, 2, and 3 years, parents reported via a *yes* or *no* response whether they had a backyard swimming pool, home garden (outdoor play space), and whether they lived closed to a park or playground.

### ***School facilities.***

At age 14 years, school principals responded (*yes* or *no*) to whether they had the following facilities: playing fields or ovals, gymnasium, swimming pool and or playing courts (tennis/basketball). This questionnaire was issued to principals on an individual basis (i.e. linked to individual cases), and hence there were duplicated responses across a period of 4 years. Duplicated entries were deleted, with the most recent date of assessment entry retained. Combinations of the various

facilities at the schools are presented in Table 8. Each combination reflects the number of schools with only that combination.

Table 8

*Descriptive Statistics for School Facilities Combination*

Facility Combination	Present	
	Frequency	Percent (%)
Oval only	1	0.5
Gymnasium only	0	0
Swimming pool only	0	0
Court (Tennis, Basketball) only	6	2.7
Oval & Gymnasium	5	2.3
Oval & Court	41	18.5
Gymnasium & Court	1	0.5
Oval, Gymnasium & Court	96	43.2
Oval, Gymnasium & Pool	1	0.5
Oval, Court & Pool	8	3.6
All 4 Facilities	63	28.4

Based on this information, a positive school environment for sports facilities was determined to be high (three or more facility combinations), medium (two facility combinations) and low (one facility). The rationale was that the more physical activity choices available, the higher the likelihood for participation in physical activity by students at and around school times.

Total school facilities included the number of school ovals, council or shire facilities, and other facilities used for physical activity. For the same school, the number of facilities reported may be different from each date the questionnaire was completed. Some possible explanations may be that the school building expansion may reduce the number of ovals, sporting activities requiring council or other facilities may change from term to term. For this variable, duplicate school cases were treated as separate cases and not removed. Percentiles depicted for the total

number of school facilities were used to create three categories low ( $\leq 2$  or  $\leq 25^{\text{th}}$  percentile), medium (2-4 or  $25^{\text{th}}$ - $75^{\text{th}}$  percentile) and high ( $> 4$  or  $> 75^{\text{th}}$  percentile).

### *School representative sports.*

Question 11 and 12 of the school questionnaire evaluated if schools participated in the representative sports program<sup>8</sup>, and if so, which sports. All responses were *yes* or *no*. The categories for representative sport included netball, athletics, swimming, soccer, tennis, rugby, AFL (Australian Football League) and other. All but six school respondents participated in representative sports. A total score for the number of representative sports offered was calculated for each school with a minimum of one and a maximum of eight.

Representative sport data were skewed towards maximum influence with many schools offering six or more options. However the range in the number of representative sports offered was varied enough to warrant further investigation of any influences. Based on data percentiles (low  $< 5^{\text{th}}$  percentile, high  $> 50^{\text{th}}$  percentile), representative sport was categorised according to a positive environment for fostering after school physical activity. The rationale was that representative sport typically entails participation by students outside of their normal school hours. It was also assumed that within a metropolitan area, most schools would have the opportunity to participate in representative athletics and swimming. Therefore, representative sport was categorised as minimal influence:  $\leq 3$ , moderate influence: 4-6, and maximum influence:  $> 6$  sports offered.

### *Physical education classes.*

The variable *minutes allocated to physical education per week* was not used in this study due to the lack of variance between schools, averaged across years 8-10. On average, schools ranged from category two (46-60minutes) to category five (more

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<sup>8</sup> Representative sports program – sports in which children can represent their school in organised weekly competitions.



than 90 minutes). A few schools reported category one (31-45minutes) for an individual year, but this was countered by extra hours in the other 2 years. In summary, all schools with data on physical education per week provided a positive environment for physical education.

### **Data Analysis**

Generally, the number of variables used was dependent on sample size power, missing data, time points available, relevance to the statistical test, and requirements of the statistical technique being used. As mentioned previously sample size varies across follow-ups (Table 4), as well as for each individual variable.

This research study involved basic descriptive statistics and two key data analysis techniques, longitudinal data analysis using linear mixed modelling (LMM) and relationship modelling using structural equation modelling (SEM). All statistical processes were conducted using statistical software SPSS for Windows (Release 17.0.0. 2008).

#### **Descriptive statistics.**

Continuous variables were described statistically according to sample size ( $n$ ), mean ( $M$ ) and standard deviations ( $SD$ ). Gender differences between males and females were assessed using independent t-tests ( $t$ ) with a reported probability ( $p$ ). Weight status group comparisons were assessed using ANOVA ( $F$ ) and Dunnett T3 *post hoc* tests for IOTF weight status groups (normal weight, overweight and obese), determined at 14 years.

Categorical variables were described statistically according to sample size ( $n$ ). Gender differences between males and females, and weight status group differences were assessed using Chi-square ( $\chi^2$ ) and reported probability ( $p$ ).

### ***Comparison of adiposity field measures<sup>9</sup>.***

Several proxies for adiposity were available at age 14 years. The inter-changeability of these proxies were examined to guide a decision on the most appropriate measure to use considering the longitudinal structure of this research. Similarities among adiposity measures were investigated using the Bland-Altman method (Bland & Altman, 1986) using z-scores, an alternative regression approach (Bland, 2004; Bland & Altman, 2003), and the traditional Pearson Product Moment Correlation. The latter has been criticised as an inappropriate statistical analysis of similarity (Bland, 2004; Bland & Altman, 2003). Differences in weight status categorisation were examined using the International Obesity Task Force (IOTF) BMI cut-offs (Cole et al., 2000), WHtR cut-offs (Kahn et al., 2005; McCarthy & Ashwell, 2006), and waist girth cut-offs (Taylor et al., 2000).

Pearson Product Moment Correlation was calculated between each of the adiposity measures taken at follow-up age 14 years. The categorisations of weight status using BMI, WHtR, and waist girth were compared using a Kappa Chi-square test. Correlations are good at measuring the strength of a relationship, but not the amount of agreement (Bland & Altman, 1986). The Bland-Altman method (Bland & Altman, 1986) was used to assess the limits of agreement, with raw scores converted to z-scores prior to analysis and the resulting confidence intervals converted back into associated raw score units. The technique allows two methods to be compared when neither provides an unequivocally correct measure, and the degree of agreement can be assessed. The Bland regression method (Bland, 2004) was used to determine 95% prediction limits at the mean.

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<sup>9</sup> From "A comparison of field measures of adiposity among Australian adolescents from the Raine Study," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2010, *Malaysian Journal of Sports Science and Recreation*. Copyright 2010 by MJSSR. Adapted and reprinted with permission.

## Linear mixed modelling (LMM)<sup>10</sup>.

Multilevel models are becoming popular in health and social sciences for the analysis of longitudinal data and provide an opportunity to investigate within-person and between-person change over time. The multilevel model for change was used to separate differences between individuals at baseline and changes over time within individuals. The model is a mathematical representation of population behaviour (Garson, 2008; Singer & Willet, 2003), which enables correct modelling of correlated errors for repeated, continuous and correlated observations (Garson, 2008), and has an underlying assumption that data are missing at random (West, Welch, & Galecki, 2007). LMM has the advantage over repeated measures ANOVA in that they are more flexible in fitting and testing covariance structures, permit individuals to have missing data points, and allow the inclusion of time-varying factors as well as the time measurement (West et al., 2007).

Model selection is performed using information criteria. Here the *goodness of fit* is based on a log-likelihood statistic. It allows the selection of a subset of interrelated predictors that best captures the effect of a single underlying construct (Singer & Willet, 2003; West et al., 2007). Predominantly the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) are used. The AIC parameter is based on the number of model parameters, while the BIC includes the number of model parameters plus sample size (Singer & Willet, 2003).

Investigations using LMM required the dataset to be transformed into person-period data in which each individual had multiple data entries corresponding to each measurement occasion (Singer & Willet, 2003). A LMM was used to model the trajectory of BMI over time from birth to 14 years. Fixed and random effects, interactions and covariance structure were all investigated in the determination of the final model, with residual diagnostics performed. Time was used as a repeated measure. Best model fit was determined based on Akaike's Information Criterion

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<sup>10</sup> From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S.Karger AG, Basel. Adapted and reprinted with permission.

(AIC), with a lower result indicating a better fit. BMI was treated as the dependent variable with effects of age, gender, gestational age and weight status (determined at age 14 years) investigated. There was no treatment for missing data, which were assessed as missing at random (MAR) (Chivers, Hands, Parker, Beilin, Kendall, & Bulsara, 2009).

The final LMM was used to test each obesogenic variable. Covariate only, full interactions and subsets were investigated consecutively, with model fit comparisons based on AIC. There was no treatment of missing data (MAR). However, for many variables, measurement occasions were limited to one or less than the complete eight measurement occasions, hence complete missingness for particular measurement occasions was present. For variables not collected at each of the eight measurement occasions, the one measurement occasion value was replicated for each of the other measurement occasions for each individual (that is, no change over time for that covariate).

### **Structural equation modelling (SEM).**

SEM is a statistically powerful and flexible tool which enables the exploration of interrelationships among variables (Gao, Thompson, Xiong, & Miller, 2006; Garson, 2007; Thomas & Nelson, 1990). It is able to assess both directional and non-directional relationships (Gao et al., 2006). It allows for overall model testing, use of multiple dependents, investigation of mediating variables, testing across multiple between subject groups. It is also able to handle auto correlated errors across longitudinal data points, non-normal data, and incomplete data. Modelling also accounts for interactions, nonlinearities, correlations, and measurement errors. SEM has been explained as an extension of general linear modelling and incorporates and integrates path and factor analysis (Garson, 2007). SEM is becoming a popular choice in multivariate methods as it allows for mixed model analysis, especially where, traditionally, different levels of measurement have been problematic (Schumacker & Lomax, 2004).

The goal of SEM is to determine how well the theoretical model is supported by the data. It is a way of testing hypotheses or can be confirmatory in approach, although there is some element of exploration in the process (Dragan & Akhtar-Danesh, 2007; Garson, 2007; Schumacker & Lomax, 2004). It takes into account group level (people within groupings) as well as individual processes (Rasbash, 2006). For causation-effect relationships to be established, there must be logical preceding order with preceding variables affecting following variables in direct or indirect ways; existence of covariance or correlation between variables; control for other causes or influences; and measurement on at least an interval level. Structural models in longitudinal data may be able to show changes in the latent variables over time, while modelling both individual and group changes (Schumacker & Lomax, 2004).

In this study however, a purely exploratory approach was taken, using add on SPSS module AMOS version 17 (SPSS for Windows, Release 17.0.0. 2008). At each follow-up, possible interrelationships were explored. The basic hypothesised model at each follow-up had all variables having pathways to BMI only. Normality was examined for each variable and below the recommended limits (skewness <3; kurtosis <10) required for SEM (Kline, 2005). Due to the presence of missing data, a series mean replacement of missing values was conducted in SPSS to create a complete dataset for single-step model building (adding pathways) (Boomsma, 2000; Garson, 2007). This permitted model building to be explored by the addition of theoretically sound pathways based on modification indices (Byrne, 2001; Garson, 2007; Kline, 2005). The original dataset (with missing values) was then used for single step model trimming (removing pathways) (Boomsma, 2000; Garson, 2007), with a maximum likelihood estimation procedure (Byrne, 2001). Each model was assessed separately for males and females and additional significant pathways included. Each final model included all significant pathways for both sexes with gender differences assessed using multi-group analysis (Byrne, 2001).

These procedural steps taken are summarised below:

- Model specification. All variables have a direct pathway to BMI.
- Single-step model building, estimation and testing. Modification indices used to add pathways.
  - Data set contains series mean replacement of missing values.
- Single-step model trimming, estimation and testing. Removal of non-significant pathways.
  - Original data set with missing values.
- Estimation and testing for each gender. Model modification.
  - Original data set with missing values.
- Final model - multi-group analysis for gender.
  - Original data set with missing values.

## **Ethics**

Initial data collection and all follow-ups of the Western Australian Pregnancy Cohort (Raine Study) have been approved by the Human Ethics Committee at King Edward Memorial Hospital and Princess Margaret Hospital for Children, Perth, Western Australia. Parents had been informed, prior to agreeing to participate, that the study would be ongoing over a number of years and would involve the investigation of a number of undisclosed child health outcomes. Consent to participate in the study was obtained from the mother of each child at enrolment and a parent at each subsequent follow-up (Kendall, 2003; Newnham et al., 1993).

Data collection has been conducted by approved research personnel of the Telethon Institute of Child Health, under the approved ethical guidelines and procedures established for the Raine Study.

All personal information pertaining to this study is kept secure and confidentially as part of the Telethon Institute of Child Health Research processes and procedures. Copies of completed questionnaires and examination data collected are identified only by study number and are stored separately from personal information in a

restricted access storage facility at the Telethon Institute of Child Health Research. Access to this information was not required for this study. Electronic stored data is also subject de-identified with restricted access by approved personnel with password access. Electronic personal information is stored separately from the data, again with restricted access and password security (Kendall, 2003). This research only accessed subject de-identified data for statistical analysis.

Research study approval and permission to access de-identified data were granted by the Raine Study Executive Committee. An expedited application for ethics approval was made to the University of Notre Dame Australia Human Research Ethics Committee and approved by both the School of Health Science Research Committee and the University of Notre Dame Human Research Ethics Committee. Copies of relevant ethic approval documents are provided at Appendix C.

## **Summary**

In summary, the methodology for this study describes a longitudinal research approach. The research design identified key obesogenic variables of interest, examined their relationship with weight status at 14 years, and identified differences between males and females. Tests of similarity for adiposity measures were used to guide the decision on best proxy for adiposity for use in this study (Chivers, Hands, Parker, Beilin, Kendall, & Bulsara, in press). A model of BMI trajectory was developed using LMM, to investigate the influences of obesogenic variables on adolescent weight status (Chivers et al., 2009) and SEM procedures were designed to explore the interrelationships among identified obesogenic variables at each follow-up.



Eight years

"... That all children are created whole, endowed with innate intelligence, with dignity and wonder, worthy of respect. The embodiment of life, liberty and happiness, children are original blessings, here to learn their own song ...

... To recognize the early years as the foundation of life, and to cherish the contribution of young children to human evolution ... "

By Raffi



## Chapter Four

### Results

Results are presented in three main parts, the descriptive analysis, the linear mixed modelling (LMM) and the structural equation modelling (SEM). Both descriptive and LMM results are structured according to individual, behavioural and environmental factors. The SEM results are presented according to follow-up years. Due to the number of variables and volume of results, supplementary results are available in the Technical Report (Appendix D CD Rom) with cross-references provided throughout this chapter.

#### Part One: Descriptive Statistics

This section presents results that describe the sample and the variables used in this study. Generally sample size, distribution, gender and weight status group differences are presented. As appropriate, results are summarised with more detailed tables provided in the Technical Report.

#### Sample.<sup>11</sup>

In this study, multiple birth ( $n=126$ ), congenital abnormality ( $n=13$ ) and preterm birth (gestational age <37 weeks,  $n=327$ ) cases were excluded, consequently 383 were removed from the sample, resulting in a sample of 2,485. Participants without a BMI at the survey year 14 were excluded, with a total of 1,403 participants available for analysis from birth to 14 years, 674 (48%) females and 729 (52%) males. ANOVA tests were used to compare the weight status of individuals at ages 2, 3, 6, 8 and 10 years selected for this study, to those excluded, with no statistical difference at each follow-up year in the prevalence of weight categories normal, overweight and obese. At follow-up age 2 years funding limitations

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<sup>11</sup> From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S.Karger AG, Basel. Adapted and reprinted with permission.

restricted the collection of physical measures to 449 individuals with valid BMI data. There was a slight selection bias in this sub-sample; they had a higher proportion of professional fathers, high income families, fathers living at home, and older mothers, but overall were similar to the retention trends seen across the survey waves in the Raine Study. There were no statistical differences in gestational age or gender between the sample selected and not selected (Technical Report Table 1).

### **Adiposity proxy measure.**

This study required a proxy measure for adiposity. Data for several outcome measures at age 14 years were available, although not all were available at each previous follow-up. Hip girth, waist girth, waist height ratio (WHtR), body mass index (BMI) and waist hip ratio (WHR) in addition to weight categories based on international cut-points were considered. A summary follows, with full details of results of the study provided in Appendix A [Chivers, P. T., Hands, B., Parker, H. E., Beilin, L. J., Kendall, G. E., & Bulsara, M. (2010). A comparison of field measures of adiposity among Australian adolescents from the Raine Study. *Malaysian Journal of Sports Science and Recreation*, 6(1), 33-45.]

Apart from WHtR, all adiposity measures were significantly different across gender ( $p < .05$ ). Pearson Product Moment Correlations found waist girth had the strongest significant correlations with all adiposity measures except height ( $r = 0.281$ ). The next strongest relationship was between BMI and WHtR, whereas WHR was only weakly correlated to the other measures.

The Kappa Chi-square test for reliability showed a significant association between the BMI (Cole et al., 2000) and WHtR (Kahn et al., 2005) categories ( $n = 1581$ , Kappa = 0.620,  $p < 0.01$ ). For comparative purposes, BMI was then categorised into two groups by collapsing the IOTF overweight and obese into one group (group one included underweight and normal, group two included overweight and obese), and a WHtR cut-off of 0.5 was used (McCarthy & Ashwell, 2006). A stronger association was found between the two-category BMI and WHtR measures ( $n = 1581$ ,

Kappa=0.701,  $p<0.01$ ). The associations of waist girth categories with BMI ( $n=1581$ , Kappa=0.671) and WHtR ( $n=1,581$ , Kappa=0.615) were also significant ( $p<0.01$ ).

The regression of the Bland-Altman calculated bias and mean was no different from zero (that is, not significant), indicating no systematic relationship between bias and mean, and hence a high level of agreement between measures. The small confidence intervals indicated small variation in differences between measures, with an acceptable degree of agreement.

The Bland regression analysis (Bland, 2004) found a significant relationship between all comparisons ( $p<.005$ ), suggesting that one variable could predict the other. Calculation of the 95% prediction interval at the mean found that these prediction intervals had a large variation, and prediction was not within acceptable limits for the clinical setting. For example a comparison of BMI versus waist girth reported a confidence interval of between 17.7 kg/m<sup>2</sup> and 24.9 kg/m<sup>2</sup>, which would classify a 14-year-old male as normal weight or overweight if using the IOTF cut-offs (Cole et al., 2000).

Based on these results, the availability of BMI at each follow-up, and the internationally used BMI IOTF cut-points (Cole et al., 2000) for weight status, the decision was made to use BMI as the proxy measure for adiposity, and as the primary outcome measure.

### **Individual factors.**

Individual factors included birth weight, gestational age, age, weight, height, BMI, adiposity rebound, puberty, early infant feeding, diet, developmental milestones and motor competence, and physical fitness.

#### *Birth weight.*

Babies were born with an average weight of 3,439 grams ( $SD=468g$ ), with males ( $M=3,501g$   $SD=467g$ ) heavier than females ( $M=3,371g$   $SD=460g$ ) ( $p<.001$ ). Overall

there were no weight status group differences ( $p=.083$ ). A gender separated ANOVA comparison found significant weight status group difference for females ( $p=.037$ ), with Post hoc analysis indicating this difference was between the normal weight ( $M=3,345g$   $SD=450g$ ) and overweight ( $M=3,457g$   $SD=450g$ ) ( $p=.042$ ) groups. Detailed summary is provided in Technical Report Tables 2 and 3.

### *Gestational age, age, weight, height and BMI.*

A summary of descriptive statistics for gestational age; age; anthropometric measures of weight, height; and the BMI are presented in Table 9. Notably, gender differences were found for BMI at follow-ups 1, 2, 3, and 14 years.

Table 9

*Cohort characteristics of the study sample, males and females at birth and each follow-up (age 1, 2, 3, 6, 8, 10 and 14 years) for gestational age, age, height, weight and BMI.*

Variable	Total		Male		Female		Gender Difference	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
<b>BIRTH</b>								
Gestational age (weeks)	1,403	39.3(1.3)	729	39.3(1.3)	674	39.3(1.3)	0.80	.421
Weight (kg)	1,402	3.4(0.5)	728	3.5(0.5)	674	3.4(0.5)	<b>5.26</b>	<b>&lt;.001</b>
Height (cm)	1,388	49.4(2.1)	722	49.8(2.1)	666	49.0(2.0)	<b>7.64</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	1,388	14.0(1.4)	722	14.1(1.3)	666	14.0(1.4)	0.48	.631
<b>Follow-up age 1 year</b>								
Age (mths)	1,317	13.3(1.2)	679	13.3(1.2)	638	13.3(1.2)	-0.82	.412
Weight (kg)	1,312	10.3(1.2)	677	10.7(1.2)	635	10.0(1.1)	<b>10.7</b>	<b>&lt;.001</b>
Height (cm)	1,305	77.6(3.1)	673	78.3(3.1)	632	76.9(2.9)	<b>8.28</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	1,305	17.1(1.4)	673	17.3(1.4)	632	16.8(1.4)	<b>7.23</b>	<b>&lt;.001</b>
<b>Follow-up age 2 years</b>								
Age (mths)	449	25.6(1.7)	238	25.7(1.6)	211	25.5(1.8)	1.14	.255
Weight (kg)	438	13.0(1.5)	234	13.3(1.5)	204	12.6(1.4)	<b>4.97</b>	<b>&lt;.001</b>

Variable	Total		Male		Female		Gender Difference	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
Height (cm)	401	90.1(3.7)	208	90.6(3.6)	193	89.5(3.7)	<b>2.97</b>	<b>.003</b>
BMI (kg/m <sup>2</sup> )	400	16.0(1.3)	207	16.2(1.3)	193	15.7(1.21)	<b>3.82</b>	<b>&lt;.001</b>
Follow-up age 3 years								
Age (mths)	981	36.9(1.5)	505	36.9(1.6)	476	36.8(1.5)	0.73	.467
Weight (kg)	969	15.1(1.8)	498	15.4(1.8)	471	14.7(1.7)	<b>6.27</b>	<b>&lt;.001</b>
Height (cm)	964	96.4(3.8)	494	97.0(3.9)	470	95.6(3.5)	<b>5.91</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	956	16.2(1.3)	488	16.3(1.3)	468	16.0(1.3)	<b>3.39</b>	<b>.001</b>
Follow-up age 6 years								
Age (mths)	1,327	70.5(2.3)	690	70.6(2.4)	637	70.5(2.2)	0.33	.741
Weight (kg)	1,261	21.4(3.3)	656	21.7(3.4)	605	21.1(3.2)	<b>3.24</b>	<b>.001</b>
Height (cm)	1,260	116.0(4.8)	657	116.6(5.1)	603	115.3(4.5)	<b>4.66</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	1,259	15.8(1.8)	656	15.9(1.8)	603	15.8(1.8)	1.02	.308
Follow-up age 8 years								
Age (mths)	1,272	96.7(4.1)	651	96.8(4.1)	621	96.6(4.1)	0.98	.327
Weight (kg)	1,271	28.2(5.6)	652	28.4(5.7)	619	27.9(5.5)	1.67	.095
Height (cm)	1,272	129.1(6.0)	652	129.8(6.1)	620	128.4(5.7)	<b>4.10</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	1,271	16.8(2.5)	652	16.8(2.5)	619	16.8(2.5)	-0.34	.738
Follow-up age 10 years								
Age (mths)	1,247	126.6(2.2)	651	126.6(2.3)	596	126.5(2.0)	1.10	.270
Weight (kg)	1,246	38.7(8.8)	650	38.6(9.0)	596	38.8(8.6)	-0.48	.633
Height (cm)	1,247	143.7(6.6)	651	143.7(6.7)	596	143.7(6.4)	-0.12	.904
BMI (kg/m <sup>2</sup> )	1,329	18.1(3.8)	692	18.0(3.9)	637	18.1(3.8)	-0.37	.708
Follow-up age 14 years								
Age (mths)	1,403	168.3(2.3)	729	168.2(2.4)	674	168.3(2.3)	-0.54	.592
Weight (kg)	1,403	58.0(13.3)	729	58.8(14.3)	674	57.1(12.1)	2.40	.017
Height (cm)	1,403	164.3(8.1)	729	166.3(8.9)	674	162.1(6.3)	<b>10.23</b>	<b>&lt;.001</b>
BMI (kg/m <sup>2</sup> )	1,403	21.4(4.2)	729	21.1(4.2)	674	21.7(4.2)	<b>-2.51</b>	<b>.012</b>

Note. . *t* = *t*-test, mths = months, kg = kilogram, cm = centimetre, kg/m<sup>2</sup> = kilogram per metre squared. **Bolded** indicates significant gender differences. From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S.Karger AG, Basel. Adapted and reprinted with permission.

BMI cut-off points of 25 and 30, age and gender adjusted for children, were used to classify the participants as normal weight, overweight or obese at age 14 years (Cole et al., 2000). At age 14 years, 73% (1,031) of adolescents were classified as normal weight (501 female, 530 male), 19% (263) as overweight (127 female, 136 male), and 8% (109) as obese (46 female, 63 male). An underweight category was not used in this study, although within this sample, 5.8% (81) were classified underweight according to the Cole and colleagues criteria (2007). Descriptive statistics for BMI, grouped according to IOTF weight status at age 14 years, for each follow-up are presented in Table 10.

ANOVA comparisons with post hoc tests found that there were significant differences in mean BMI for weight status groups for both males and females from age 1 year until age 14 years ( $p < .001$ ). These differences strengthened over time with increasing  $F$ -values. At age 1 and 2 years, there were significant differences between normal weight and overweight ( $p < .05$ ) and normal weight and obese ( $p < .05$ ), but not between overweight and obese groups. At age 3 years, significant differences were reported between all group comparisons ( $p < .01$ ), except between overweight and obese females. From age 6 years, all group differences were between normal weight and overweight ( $p < .001$ ), normal weight and obese ( $p < .001$ ) and overweight and obese ( $p < .001$ ) (Technical Report Table 4).

Table 10

*BMI Descriptive Statistics at each Follow-up based on BMI Weight Status at 14 Years*

Age at Follow-up	BMI Weight Status determined at 14 years	Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Birth	Normal Weight	526	14.0(1.3)	494	14.0(1.4)
	Overweight	133	14.1(1.5)	127	14.3(1.3)
	Obese	63	14.3(1.5)	45	14.1(1.6)
1 year	Normal Weight	490	17.2(1.3)	472	16.6(1.3)
	Overweight	124	17.6(1.4)	117	17.2(1.4)
	Obese	59	18.0(1.4)	43	17.5(1.1)
2 years	Normal Weight	153	15.9(1.1)	151	15.5(1.1)
	Overweight	40	16.9(1.8)	29	16.3(1.2)
	Obese	14	16.8(1.1)	13	16.8(1.0)
3 years	Normal Weight	357	16.1(1.1)	347	15.8(1.2)
	Overweight	88	16.7(1.3)	93	16.5(1.2)
	Obese	43	17.6(1.5)	28	17.2(1.8)
6 years	Normal Weight	476	15.3(1.2)	449	15.3(1.2)
	Overweight	122	16.7(1.7)	114	16.7(1.6)
	Obese	58	18.7(2.6)	40	18.9(2.9)
8 years	Normal Weight	475	15.8(1.4)	459	15.9(1.6)
	Overweight	119	18.2(2.1)	119	18.5(1.9)
	Obese	58	21.7(3.3)	41	21.8(3.9)
10 years	Normal Weight	506	16.8(2.5)	473	16.9(2.6)
	Overweight	127	19.9(3.8)	121	20.3(3.6)
	Obese	59	24.7(5.0)	43	25.0(5.8)
14 years	Normal Weight	530	19.1(1.8)	501	19.8(1.9)
	Overweight	136	24.5(1.4)	127	25.2(1.4)
	Obese	63	31.3(3.3)	46	32.8(3.7)

*Note.* From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310.

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### *Adiposity rebound.*<sup>12</sup>

BMI at nadir and age at nadir were calculated for a subset of individuals with actual BMI at each follow-up ( $n=173$ ). There were no gender differences in the adiposity rebound nadirs (Technical Report Table 5). There were significant weight status group differences for BMI at nadir and age at nadir ( $p<.001$ ), but no significant gender differences (Table 11). *Post hoc* analysis of weight status groups found a significant difference for females between BMI and age at nadir of the normal weight compared to overweight group, and normal weight compared to obese groups. For males, there was only a significant difference in BMI at nadir between the normal weight and overweight and obese groups. There were no statistically significant differences between the overweight and obese groups. (For more detailed results refer to the Technical Report Table 6.)

Table 11

*BMI and Age at Nadir for a Subset of Individuals (n=173) with BMI Scores at Every Follow-up, Birth to Age 14 years*

	BMI Weight Status	n	Total	Male		Female	
			M (SD)	n	M (SD)	n	M (SD)
	determined at 14 years						
BMI Nadir <sup>a</sup> (kg/m <sup>2</sup> )	Normal Weight	132	15.0(1.0) <sup>b</sup>	71	15.1(0.9) <sup>b</sup>	61	14.9(1.0) <sup>b</sup>
	Overweight	32	16.4(1.3) <sup>c</sup>	19	16.7(1.3)	13	16.1(1.2) <sup>c</sup>
	Obese	9	16.4(0.9)	5	16.3(1.2)	4	16.6(0.3)
Age Nadir <sup>a</sup> (years)	Normal Weight	132	5.3(2.2) <sup>b</sup>	71	5.4(2.0)	61	5.1(2.3) <sup>b</sup>
	Overweight	32	3.8(2.2) <sup>c</sup>	19	4.2(2.4)	13	3.2(1.9) <sup>c</sup>
	Obese		2.6(1.4)	5	3.1(1.7)	4	2.0(0.7)

Note.<sup>11</sup>

<sup>a</sup> Significant difference in ANOVA between groups test  $p<0.005$ .

<sup>b</sup> Significant difference between ANOVA Post hoc test groups normal weight and overweight  $p<0.05$ .

<sup>c</sup> Significant difference between ANOVA Post hoc test groups normal weight and obese  $p<0.005$ .

<sup>12</sup> From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S. Karger AG, Basel. Adapted and reprinted with permission.



Adiposity rebound occurred for the normal weight group at 5.3 years, for the overweight group at 3.8 years, and for the obese group at 2.6 years. Adiposity rebound results were similar for the subsample ( $n=173$ ) and full sample ( $n=1,403$ ) and is shown pictorially for the full sample in Figure 2. The BMI trajectory paths for each weight status are distinct in their profiles, particularly in the timing of the peaks and troughs (Figure 2) and are based on the mean BMI for each weight status group, and mean age at each survey wave.

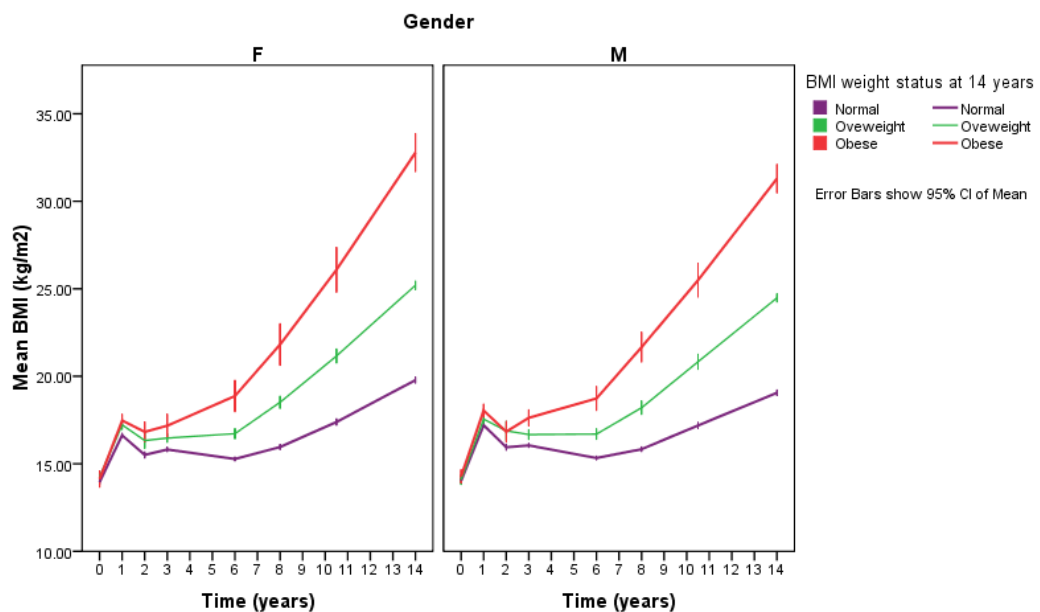


Figure 2. Mean BMI over time based on BMI IOTF weight categories normal weight, overweight and obese, as determined at follow-up 14 years ( $n=1,403$ ). M=males ( $n=95$ ), F=females ( $n=78$ ). From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S. Karger AG, Basel. Adapted and reprinted with permission.

### ***Early infant feeding.***<sup>13</sup>

Early infant feeding was investigated by the age at which breastfeeding stopped, the age milk other than breast milk was introduced, and weaning. On average, breastfeeding stopped at 7.9 months ( $SD=6.9$ ,  $n=1330$ ), other milk started at 5.0 months ( $SD=4.1$   $n=1,320$ ), and weaning started at 4.3 months ( $SD=1.3$   $n=1,326$ ). No gender differences were found (Technical Report Table 7).

Investigations using continuous data found weight status group differences for when breastfeeding was stopped ( $F=3.126$ ,  $p=.044$ ) with Post hoc tests indicating that this difference was between the normal weight and obese only ( $p=.032$ ). Gender separated analysis found that weight status groups differences were only significant for males ( $F=3.171$ ,  $p=.043$ ), with Post hoc tests indicating that this difference was between the normal weight ( $M=8.3$  months  $SD=6.5$  months) and obese ( $M=6.0$  months  $SD=6.7$  months) group only ( $p=.037$ ). No weight status group differences were found for when other milk started, nor time of weaning (Technical Report Tables 8 and 9).

The age breastfeeding stopped and age other milk was introduced using the 4-month cut-point and weight status at age 14 are compared in Table 12. Chi-square analysis identified a significant difference between the age breastfeeding stopped ( $p<.001$ ) and the age other milk was introduced ( $p=.011$ ) for weight status groups at 14 years. The groups who had been breastfed for  $\leq 4$  months or introduced to other milk  $\leq 4$  months contained a significantly higher proportion of overweight and obese adolescents (32.3% and 30.1% respectively) compared with those breastfed for longer or started other milk later (22.9% and 22.8% respectively).

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<sup>13</sup> From "Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study)," by P. T. Chivers, B. Hands, H. E. Parker, M. Bulsara, L.J. Beilin, G.E. Kendall, and W.H. Oddy, 2010, 34, e1-8, *International Journal of Obesity*. Copyright 2010 Nature Publishing Group. Adapted and reprinted with permission.

Table 12

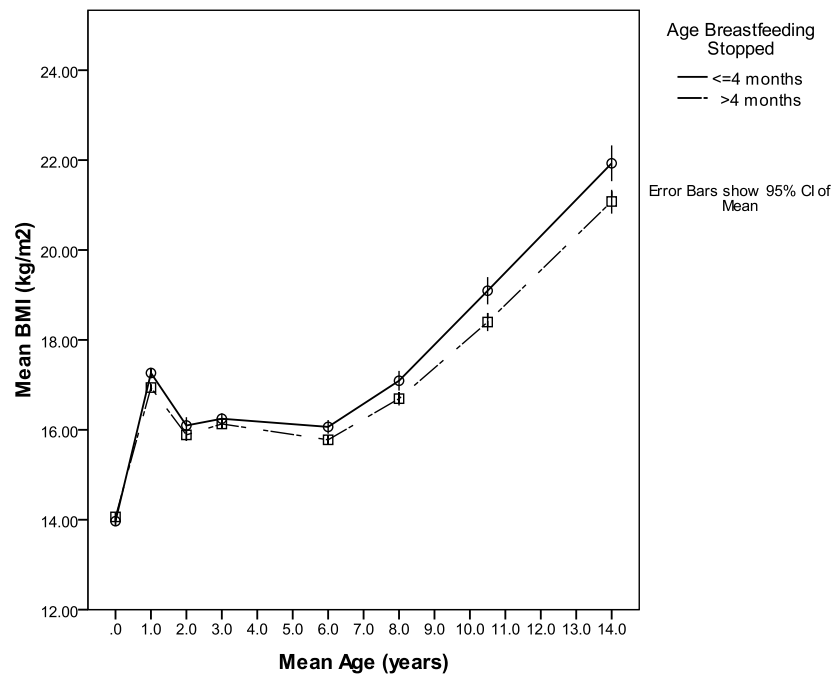
*Age breastfeeding was stopped (Stop Breast) and when other milk introduced (Start Milk) for weight status groups at 14 years*

	BMI Weight Status determined at age 14 years				$\chi^2$	<i>p</i>
	Total	Normal Weight	Overweight	Obese		
Stop Breast	1,330	977	248	105	<b>15.218</b>	<b>&lt;.001</b>
≤ 4 months	521	353 (67.8%)	114 (21.9%)	54 (10.4%)		
>4 months	809	624 (77.1%)	134 (16.6%)	51 (6.3%)		
Start Milk	1,320	969	246	105	<b>9.062</b>	<b>.011</b>
≤ 4 months	688	481 (69.9%)	144 (20.9%)	63 (9.2%)		
>4 months	632	488 (77.2%)	102 (16.2%)	42 (6.6%)		

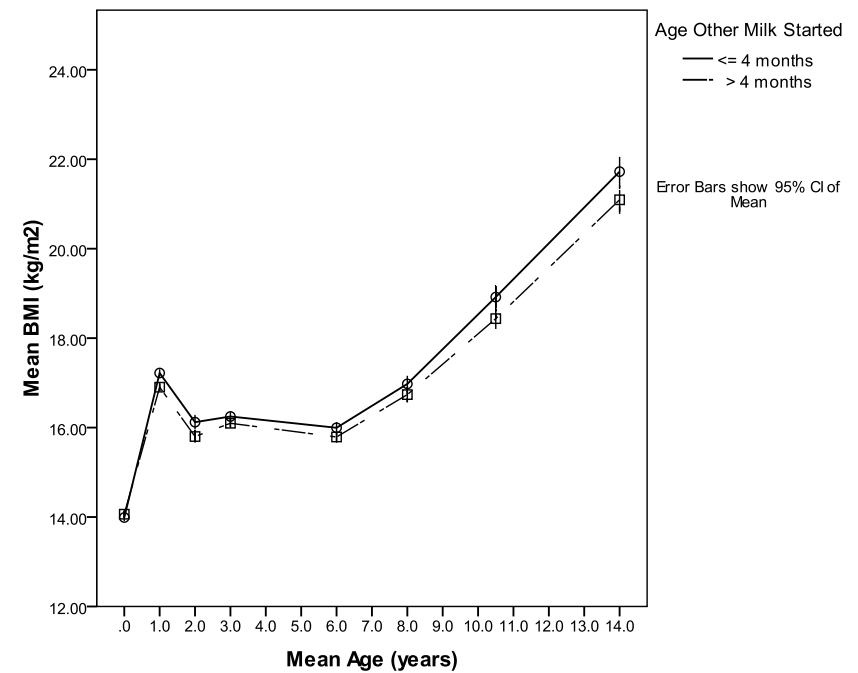
*Note.* **Bolded** indicate significant group differences  $p < .05$ . From "Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study)," by P. T. Chivers, B. Hands, H. E. Parker, M. Bulsara, L.J. Beilin, G.E. Kendall, and W.H. Oddy, 2010, 34, e1-8, *International Journal of Obesity*. Copyright 2010 Nature Publishing Group. Adapted and reprinted with permission.

The influence of early infant feeding on adiposity rebound was also investigated in a small sub-sample ( $n=171$ ). There were no significant differences between age breastfeeding stopped, age other milk introduced or weaning groups for BMI and age at nadir. Distinct differences are seen in Figure 3, which is based on the full sample means for both age breastfeeding stopped and age other milk was introduced.

This influence was also investigated using predicted BMI. Briefly, predicted BMI was based on the LMM, adjusted for age, gestational age, gender and weight status and is discussed in detail as part of the LMM section. In summary, when predicted BMI was used there were significant group differences in both BMI and age at nadir for when breastfeeding stopped and other milk was introduced (Chivers et al., 2010).



A. Age breastfeeding stopped.



B. Age other milk started.

Figure 3. Mean BMI over mean age based on Age Breastfeeding Stopped (A) ( $n=1,330$ ) and Age Other Milk Started (B) ( $n=1,320$ ) groups  $\leq$  4 months and  $>$  4 months. From “Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study),” by P. T. Chivers, B. Hands, H. E. Parker, M. Bulsara, L.J. Beilin, G.E. Kendall, and W.H. Oddy, 2010, 34, e1-8, *International Journal of Obesity*. Copyright 2010 Nature Publishing Group. Adapted and reprinted with permission.

### ***Diet.***

Diet variables included a derived score of fat intake at ages 8 (caregiver report) and 14 years (self-report), and frequency of fruit and vegetable intake at age 14 years (self-report).

### ***Fat intake.***

At age 8 years, 1,032 respondents had a mean derived score of 18.7 ( $SD=4.7$ , range 0-56) with no gender differences ( $p=.413$ ). At 14 years, there were fewer respondents ( $n=783$ ) with a mean derived score of 21.4 ( $SD=6.2$ ), with males reporting a higher fat intake ( $M=21.1$   $SD=5.8$ ) than females ( $M=20.7$   $SD=6.5$ ) ( $p=.002$ ) (Table 13). No weight status group differences were found at either 8 or 14 years (Table 14).

Table 13

*Descriptive Statistics for Fat Intake (derived score) at 8 and 14 years*

Age Follow-up	BMI Weight Status determined at 14 years	Male		Female		Total	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
8 Years	Normal Weight	389	18.7(4.7)	368	18.8(4.6)	757	18.7(4.7)
	Overweight	94	18.1(4.4)	105	19.0(4.7)	199	18.6(4.6)
	Obese	36	17.9(5.0)	40	18.4(5.3)	76	18.1(5.1)
14 Years	Normal Weight	290	22.1(5.8)	274	20.7(6.5)	564	21.4(6.2)
	Overweight	76	21.9(5.8)	82	21.0(6.0)	158	21.4(5.9)
	Obese	27	22.6(5.9)	34	20.4(7.6)	61	21.3(6.9)

Table 14

*Weight Status Group Differences for Fat Intake (derived score) at 8 and 14 years*

Follow-up	Gender	ANOVA		Post hoc Tests p-value		
		<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
8 Years	Male	1.120	.327	.475	.725	.998
	Female	.335	.716	.931	.952	.852
	Total	.612	.542	.954	.698	.891
14 Years	Male	.140	.869	.992	.966	.937
	Female	.140	.870	.962	.994	.959
	Total	.005	.995	1.000	1.000	1.000

#### *Fruit and vegetable intake.*

Self-reported frequency of fruit intake at age 14 years was similar for males and females (Table 15) with no differences between weight status groups (Table 16). Adolescent female's frequency of vegetable intake was higher than males ( $p=.001$ ) (Table 15). Weight status group differences were significant for frequency of vegetable intake ( $p=.020$ ) (Table 16). Fewer obese adolescents (36.1%) reported eating vegetables *more than six times per week*, than those overweight (53.5%) or normal weight (48.5%). When data were gender separated, this weight status difference was only significant for females ( $\chi^2=21.45$   $p=.006$ ), but not males ( $\chi^2=6.914$   $p=.546$ ).

Table 15

*Gender Differences for Fruit and Vegetable Intake at 14 years*

Variable	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
Adolescent fruit frequency					
Rarely or never	25	14	11	6.962	.138
1-2 times a month	64	41	23		
1-2 times a week	265	147	118		
3-5 times a week	560	282	278		
6+ times a week	479	237	242		
Adolescent vegetable frequency					
Rarely or never	21	16	5	<b>18.556</b>	<b>.001</b>
1-2 times a month	25	18	7		
1-2 times a week	164	101	63		
3-5 times a week	508	259	249		
6+ times a week	675	327	348		

Note. **Bolded** indicate significant group differences  $p < .05$ .

Table 16

*Weight Status Group Differences for Fruit and Vegetable Intake at 14 years*

Frequency	Total	Obese	overweight	Normal weight	$\chi^2$	$p$
Adolescent fruit frequency					11.011	.201
Rarely or never	25	1	6	18		
1-2 times a month	64	9	14	41		
1-2 times a week	265	25	51	189		
3-5 times a week	560	46	94	420		
6+ times a week	479	27	95	357		
Adolescent vegetable frequency					<b>18.140</b>	<b>.020</b>
Rarely or never	21	0	7	14		
1-2 times a month	25	2	5	18		
1-2 times a week	164	18	33	113		
3-5 times a week	508	49	76	383		
6+ times a week	675	39	139	497		

Note. **Bolded** indicate significant group differences  $p < .05$ .

***Developmental milestones and motor competence.***

Measurements of developmental milestones and motor competence from birth to 14 years differed between follow-ups, but included parent reported milestones; Denver II assessments at ages 1, 2 and 3 years; Infant Monitoring Questionnaire fine and gross motor skills at ages 1, 2 and 3 years; parent perceived motor skills at 10 years, and the MAND at 10 and 14 years. These are each discussed separately below.

***Parent reported developmental milestones.***

Developmental milestones included when, in months, the child first smiled, sat up, babbled, crawled, stood, tottered, walked, spoke their first word, showed hand preference and their first tooth erupted.



Gender differences were found for *first sat up* ( $t=3.198$   $p=.001$ ) with males ( $M=5.9$  months  $SD=1.4$ ) sitting later than females ( $M=5.6$  months  $SD=1.3$ ); *spoke their first word* ( $t=4.949$   $p<.001$ ) with males ( $M=12.2$  months  $SD=3.1$ ) talking later than females ( $M=11.3$  months  $SD=2.7$ ); and their *first tooth erupted* ( $t=-2.988$   $p=.003$ ) with males' ( $M=6.6$  months  $SD=2.0$ ) first tooth erupting earlier than females ( $M=6.9$  months  $SD=2.2$ ). There were no weight status group differences across milestones. Full details are presented in the Technical Report Tables 10-12.

### *Denver II assessment.*

The Denver II assessment was conducted at 1, 2 and 3 years and evaluated gross motor, fine motor, personal social, language, and overall development on a scale of *uncooperative, fail, adequate* and *normal*. Compared to males, more girls were assessed to have *normal* development at age 1 year for fine motor ( $\chi^2=11.355$   $p=.033$ ) and language ( $\chi^2=10.954$   $p=.004$ ); and at 3 years for gross motor ( $\chi^2=17.804$   $p<.001$ ), personal social ( $\chi^2=24.940$   $p<.001$ ), language ( $\chi^2=16.809$   $p<.001$ ) and overall development ( $\chi^2=30.492$   $p<.001$ ). Full details are provided in the Technical Report Table 13.

Weight status group differences were found for two year fine motor ( $\chi^2=13.767$   $p=.032$ , fewer obese assessed *normal*), personal social ( $\chi^2=24.529$   $p<.001$ , fewer obese assessed as *normal*), language ( $\chi^2=30.993$   $p<.001$ , fewer overweight and obese assessed as *normal*); and three year fine motor ( $\chi^2=10.426$   $p=.034$ , fewer overweight assessed as *normal*), language ( $\chi^2=11.680$   $p=.020$ , fewer overweight and obese assessed as *normal*), and overall development ( $\chi^2=27.069$   $p<.001$ , fewer obese assessed as *normal*). Full details are provided in the Technical Report Table 14.

### *Infant Monitoring Questionnaire.*

Gross and fine motor skills at ages 1, 2 and 3 years, along with overall development items at ages 2 and 3 years, were evaluated with the Infant Monitoring

Questionnaire (IMQ). Sample sizes varied across the years, although response rates remained above 1,000. A summary is provided below, with details provided in the Technical Report Tables 15-30.

Overall, males and females were similar in their gross motor IMQ ratings except at age 1 year for *using stairs* ( $n=1,289$   $\chi^2=6.601$   $p=.037$ ), *standing and walking* ( $n=1,287$   $\chi^2=16.436$   $p<.001$ ); and at 3 years for *balance on one leg* ( $n=1,227$   $\chi^2=9.500$   $p=.009$ ). There were more gender differences for IMQ ratings of fine motor skills and included at 1 year for *turn pages* ( $n=1,307$   $\chi^2=7.005$   $p=.030$ ), *pincer tips* ( $n=1,300$   $\chi^2=7.895$   $p=.019$ ), *pincer without support* ( $n=1,292$   $\chi^2=5.440$   $p=.006$ ), *put down object and release* ( $n=1,288$   $\chi^2=8.212$   $p=.016$ ); at 2 years for *thread* ( $n=1,018$   $\chi^2=7.310$   $p=.026$ ), *pencil grip* ( $n=1,084$   $\chi^2=20.836$   $p<.001$ ), *flip switch* ( $n=1,112$   $\chi^2=8.836$   $p=.012$ ); and all fine motor tasks at age 3 years. The IMQ overall ratings found gender differences at 2 years for *talking* ( $n=1,109$   $\chi^2=23.256$   $p<.001$ ), *understanding* ( $n=1,120$   $\chi^2=11.925$   $p=.001$ ), *worries* ( $n=1,114$   $\chi^2=4.331$   $p=.037$ ); and all items at 3 years, except locomotion. Across gross, fine and overall IMQ motor skills, females had a higher proportion of achievement of task, compared to males, except for *flipping a switch* and *worries* at 2 years; and *worries* and *medical* at 3 years.

Generally no weight status group differences were found. Where a significant difference was observed, proportionally more normal weight children were likely to have achieved the skill, followed by those overweight, then those children obese. Among the gross motor skills, only *balance on one leg* at 3 years revealed a significant weight status group difference ( $n=1,289$   $\chi^2=10.083$   $p=.039$ ). For fine motor skills, significant weight status differences were found at 1 year for *pick up pincer* ( $n=1,298$   $\chi^2=13.709$   $p=.008$ ), at 2 years for *thread* ( $n=1,295$   $\chi^2=11.554$   $p=.021$ ), and at 3 years for *copy circle* ( $n=1,296$   $\chi^2=13.075$   $p=.011$ ). For overall development, only at 2 years *talking* revealed significant weight status group differences ( $n=1,109$   $\chi^2=7.188$   $p=.027$ ).

*Parent perceived child motor skills at 10 years.*

Significant gender differences were found for the locomotor skills of *hop*, *skip*, and *dodge*; all object control skills; and the body management skill of *bike riding* (Table 17). Parents reported that boys were better at *throwing* ( $p < .001$ ), *catching* ( $p < .001$ ), *kicking* ( $p < .001$ ), *striking* ( $p < .001$ ), and *riding a bike* ( $p < .001$ ), whereas girls were better at *hopping* ( $p = .005$ ) and *skipping* ( $p < .001$ ). Parents were more satisfied with girls' *academic performance* ( $p < .001$ ) and *learning skills progress* ( $p < .001$ ). No gender differences were found for *running*, *jumping* and *coordination progress*.

Table 17

*Gender Differences for Parent Perceived Motor Skills at 10 Years*

Motor Skill	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference $\chi^2$	<i>p</i>
<b>Locomotion</b>					
<b>Run (<i>n</i>=1,311)</b>					
poor	12	7	5	4.404	.354
below average	91	46	45		
average	749	375	374		
above average	296	164	132		
excellent	163	93	70		
<b>Jump (<i>n</i>=1,307)</b>					
poor	7	6	1	4.775	.311
below average	57	31	26		
average	890	459	431		
above average	236	120	116		
excellent	117	67	50		

Motor Skill	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
<b>Hop (<i>n</i>=1,306)</b>					
poor	9	7	2	<b>14.852</b>	<b>.005</b>
below average	52	37	15		
average	960	496	464		
above average	190	86	104		
excellent	95	55	40		
<b>Skip (<i>n</i>=1,308)</b>					
poor	12	11	1	<b>39.775</b>	<b>&lt;.001</b>
below average	70	56	14		
average	908	475	433		
above average	218	90	128		
excellent	100	50	50		
<b>Dodge (<i>n</i>=1,301)</b>					
poor	6	5	1	<b>27.864</b>	<b>&lt;.001</b>
below average	52	26	26		
average	819	390	429		
above average	290	171	119		
excellent	134	91	43		
<b>Object Control</b>					
<b>Throw (<i>n</i>=1,310)</b>					
poor	5	4	1	<b>47.191</b>	<b>&lt;.001</b>
below average	61	30	31		
average	754	340	414		
above average	327	194	133		
excellent	163	116	47		

Motor Skill	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
<b>Catch (<i>n</i>=1,309)</b>					
poor	7	6	1	<b>35.246</b>	<b>&lt;.001</b>
below average	73	41	32		
average	745	340	405		
above average	321	187	134		
excellent	163	109	54		
<b>Kick (<i>n</i>=1,306)</b>					
poor	4	2	2	<b>81.159</b>	<b>&lt;.001</b>
below average	82	34	48		
average	786	346	440		
above average	274	181	93		
excellent	160	121	39		
<b>Strike (<i>n</i>=1,306)</b>					
poor	5	3	2	<b>55.917</b>	<b>&lt;.001</b>
below average	88	40	48		
average	810	369	441		
above average	265	173	92		
excellent	131	95	36		
<b>Body Management</b>					
<b>Ride Bike (<i>n</i>=1,312)</b>					
poor	18	10	8	<b>29.632</b>	<b>&lt;.001</b>
below average	58	29	29		
average	745	344	401		
above average	326	194	132		
excellent	165	108	57		

Motor Skill	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
Balance ( <i>n</i> =1,308)					
poor	6	5	1	5.356	.253
below average	38	22	16		
average	812	433	379		
above average	290	139	151		
excellent	162	85	77		
Overall					
Coordination development progress ( <i>n</i> =1,312)					
poor	7	5	2	2.968	.563
dissatisfied	34	20	14		
neither	74	43	31		
satisfied	661	338	323		
very satisfied	536	280	256		

Note. **Bolded** indicate significant group differences  $p < .05$ .

There were significant weight status group differences for all locomotor skills, balance, and overall coordination. There was a trend of scores along a continuum across weight status groups (Table 18). A higher proportion of obese followed by overweight individuals were rated below average for locomotive abilities *running* ( $p < .001$ ), *jumping* ( $p < .001$ ), *hopping* ( $p < .001$ ), *skipping* ( $p < .001$ ), *dodging* ( $p = .001$ ); body management *balancing* ( $p < .001$ ), and for overall a dissatisfaction with *coordination developmental progress* ( $p < .001$ ). On the other hand, normal weight individuals typically rated higher proportions of *above average*, *excellent* or *satisfied*. No weight status group differences were found for object control abilities, or body management *ride bike*.

Table 18

*Weight Status Group Differences for Parent Perceived Motor Skills at 10 Years*

Motor Skills	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
<b>Locomotion</b>						
Run					<b>93.833</b>	<b>&lt;.001</b>
poor	12	2	3	7		
below average	91	45	25	21		
average	749	71	161	517		
above average	296	6	37	253		
excellent	163	2	16	145		
<b>Jump</b>						
Jump					<b>58.986</b>	<b>&lt;.001</b>
poor	7	2	1	4		
below average	57	14	15	28		
average	890	80	173	637		
above average	236	3	35	198		
excellent	117	3	16	98		
<b>Hop</b>						
Hop					<b>59.862</b>	<b>&lt;.001</b>
poor	9	3	2	4		
below average	52	14	12	26		
average	960	78	192	690		
above average	190	5	24	161		
excellent	95	2	12	81		
<b>Skip</b>						
Skip					<b>60.634</b>	<b>&lt;.001</b>
poor	12	1	2	9		
below average	70	19	11	40		
average	908	75	183	650		
above average	218	3	35	180		
excellent	100	3	12	85		

Motor Skills	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
Dodge					<b>27.209</b>	<b>.001</b>
poor	6	1	0	5		
below average	52	7	14	31		
average	819	77	162	580		
above average	290	12	42	236		
excellent	134	4	21	109		
Object Control						
Throw					11.307	.185
poor	5	0	0	5		
below average	61	2	12	47		
average	754	73	141	540		
above average	327	19	61	247		
excellent	163	8	29	126		
Catch					13.705	.090
poor	7	0	0	7		
below average	73	7	16	50		
average	745	70	146	529		
above average	321	17	55	249		
excellent	163	8	26	129		
Kick					10.321	.243
poor	4	0	0	4		
below average	82	3	17	62		
average	786	73	141	572		
above average	274	16	59	199		
excellent	160	10	25	125		



Motor Skills	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
<b>Strike</b>					<b>6.369</b>	<b>.606</b>
poor	5	0	0	5		
below average	88	5	17	66		
average	810	69	143	598		
above average	265	19	58	188		
excellent	131	7	24	100		
<b>Body Management</b>						
<b>Ride Bike</b>					<b>12.270</b>	<b>.140</b>
poor	18	3	1	14		
below average	58	4	13	41		
average	745	68	144	533		
above average	326	18	52	256		
excellent	165	9	33	123		
<b>Balance</b>					<b>35.997</b>	<b>&lt;.001</b>
poor	6	0	0	6		
below average	38	8	11	19		
average	812	78	158	576		
above average	290	10	45	235		
excellent	162	6	26	130		
<b>Overall</b>						
<b>Coordination development progress</b>					<b>40.582</b>	<b>&lt;.001</b>
poor	7	0	1	6		
dissatisfied	34	8	4	22		
neither	74	8	21	45		
satisfied	661	66	130	465		
very satisfied	536	20	87	429		

Note. **Bolded** indicate significant group differences  $p < .05$ .

*McCarron Assessment of Neuromuscular Development (MAND).*

Results include individual sum of scaled scores for gross and fine motor skills, and the Neuromuscular Development Index (NDI  $M=100$   $SD=15$ ), at ages 10 and 14 years. At 10 years, except for total gross motor skills, females scored higher than males in motor competence. By 14 years, these differences existed only for sum of scales scores for gross and fine motor sub scales, with females having higher scores in the fine motor sub scale, and males having higher scores in the gross motor sub scale. Gender differences are detailed in Table 19.

Table 19

*Gender Differences for the MAND at 10 and 14 Years*

Variable	Total		Male		Female		Gender Difference	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
10 Years								
Fine Motor	1,193	50.6(10.8)	621	48.3(10.8)	572	53.1(10.2)	<b>-7.758</b>	<b>&lt;.001</b>
Gross Motor	1,188	41.3(10.1)	618	41.4(10.7)	570	41.1(9.4)	.577	.564
NDI Score	1,186	94.3(14.4)	617	92.7(15.1)	569	96.1(13.5)	<b>-4.027</b>	<b>&lt;.001</b>
14 Years								
Fine Motor	1,384	47.0(10.6)	721	45.4(10.8)	663	48.7(10.0)	<b>-5.960</b>	<b>&lt;.001</b>
Gross Motor	1,384	44.3(11.1)	721	45.9(11.6)	663	42.5(10.2)	<b>5.758</b>	<b>&lt;.001</b>
NDI Score	1,384	97.3(17.3)	721	97.6(18.2)	663	97.0(16.4)	.662	.508

*Note.* **Bolded** indicate significant group differences  $p<.05$ .

Weight status group differences are detailed in Table 20 with summaries below.

Analysis of NDI scores using categories of low ( $NDI \leq 85$ ), average ( $NDI > 85$  to  $< 115$ ) and high ( $NDI \geq 115$ ) are detailed in the Technical Report, Tables 31-32.

Table 20

*Weight Status Group Differences for MAND at 10 and 14 Years*

Variable	Gender	ANOVA		Post hoc Tests p-value		
		<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
10y Fine	Male	2.453	.087	.961	.083	.279
Motor	Female	<b>4.905</b>	<b>.008</b>	.058	.085	.915
	Total	<b>6.740</b>	<b>.001</b>	.184	<b>.003</b>	.184
10y Gross	Male	<b>5.997</b>	<b>.003</b>	.894	<b>.009</b>	.067
Motor	Female	2.375	.094	.852	.209	.491
	Total	<b>8.322</b>	<b>&lt;.001</b>	.707	<b>.002</b>	<b>.028</b>
10y NDI	Male	<b>5.025</b>	<b>.007</b>	.994	<b>.008</b>	<b>.047</b>
Score	Female	<b>4.378</b>	<b>.013</b>	.167	.096	.709
	Total	<b>9.108</b>	<b>&lt;.001</b>	.434	<b>&lt;.001</b>	<b>.029</b>
14y Fine	Male	<b>4.958</b>	<b>.007</b>	.074	.077	.937
Motor	Female	<b>3.657</b>	<b>.026</b>	.396	.137	.533
	Total	<b>8.779</b>	<b>&lt;.001</b>	<b>.026</b>	<b>.005</b>	.435
14y Gross	Male	<b>11.724</b>	<b>&lt;.001</b>	.085	<b>&lt;.001</b>	.075
Motor	Female	<b>10.564</b>	<b>&lt;.001</b>	.138	<b>&lt;.001</b>	<b>.024</b>
	Total	<b>20.569</b>	<b>&lt;.001</b>	<b>.012</b>	<b>&lt;.001</b>	<b>.004</b>
14y NDI	Male	<b>8.827</b>	<b>&lt;.001</b>	.066	<b>.001</b>	.260
Score	Female	<b>8.101</b>	<b>&lt;.001</b>	.082	<b>.003</b>	.138
	Total	<b>16.762</b>	<b>&lt;.001</b>	<b>.005</b>	<b>&lt;.001</b>	<b>.032</b>

Note. y=years. Bolded indicate significant group differences  $p < .05$ .

At 10 years, weight status group differences were present for fine and gross motor sub scales, and NDI score (Table 20). Group differences were only between the normal weight and obese group for the fine motor sub scale, with the obese group having a lower fine motor score (Table 21). In respect to the gross motor skills, the differences were between the normal weight and overweight groups, compared to obese (Table 20) again the obese group had a lower gross motor score (Table 21).

By 14 years, except for fine motor, differences were found between all groups (Table 20). Motor competence scores showed a trend with the normal weight group scoring the highest, overweight in comparison slightly lower, and the obese group had the lowest scores (Table 21). Differences in fine motor scores were only significant between the normal weight group compared to both the overweight and obese groups.

Table 21

*Descriptive Statistics for MAND at 10 and 14 Years*

Variable	BMI Weight Status	Total		Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
	determined at 14 Years						
10y Fine Motor	Normal Weight	877	51.2(10.7)	454	48.7(10.8)	423	53.8(10.0)
	Overweight	220	49.7(10.8)	110	48.2(10.6)	110	51.2(10.8)
	Obese	96	47.3(10.6)	57	45.4(10.6)	39	50.1(10.1)
10y Gross Motor	Normal Weight	874	41.8(9.7)	452	42.0(10.3)	420	41.5(9.1)
	Overweight	219	41.0(10.5)	110	41.3(11.2)	109	40.7(9.8)
	Obese	95	37.4(11.7)	56	36.8(12.1)	39	38.1(11.2)
10y NDI Score	Normal Weight	872	95.2(14.0)	451	93.4(14.5)	421	97.0(13.1)
	Overweight	219	93.6(15.4)	110	93.1(16.6)	109	94.2(14.1)
	Obese	95	88.7(15.3)	56	86.7(15.3)	39	91.5(15.2)
14y Fine Motor	Normal Weight	1,017	47.7(10.2)	524	46.2(10.4)	493	49.3(9.8)
	Overweight	261	45.7(11.0)	136	43.7(11.8)	125	47.9(9.5)
	Obese	106	43.9(11.8)	61	42.7(11.3)	45	45.4(12.5)
14y Gross Motor	Normal Weight	1,017	45.2(10.7)	524	47.0(11.0)	493	43.3(10.1)
	Overweight	261	42.9(11.4)	136	44.4(12.6)	125	41.4(9.8)
	Obese	106	38.5(12.0)	61	39.9(12.8)	45	36.4(10.6)
14y NDI Score	Normal Weight	1,017	98.7(16.9)	524	99.2(17.5)	493	98.3(16.3)
	Overweight	261	94.9(17.7)	136	94.9(19.8)	125	94.8(15.2)
	Obese	106	89.6(17.6)	61	90.1(17.9)	45	89.0(17.3)

Note. y=years.

### **Physical Fitness.**

Objective measures of physical fitness included aerobic fitness, muscle endurance, muscle strength, and flexibility at age 14 years.

#### *Aerobic fitness.*

Aerobic fitness was measured by the PWC 170 at 14 years for 1,322 individuals. There was a significant gender difference ( $t=19.39$   $p<.001$ ), with males ( $n=689$   $M=123.9$   $SD=30.5$ ) having a higher predicted workload score than females ( $n=633$   $M=96.9$   $SD=19.2$ ) (Figure 4). The normal weight group had a lower score (Table 22) with weight status group differences present (Table 23, Figure 4). When PWC 170 was adjusted for body weight, the obese group had the lowest relative PWC 170 score, with results similar to the absolute PWC 170 results (Technical Report Tables 33 and 34).

Table 22

#### *Descriptive Statistics for PWC 170 Score (Watts) at 14 Years*

BMI Weight Status determined at 14 years	Total		Male		Female	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Normal Weight	971	109.0(28.8)	501	122.5(30.3)	470	94.5(18.1)
Overweight	251	113.9(27.2)	130	124.6(29.2)	121	102.3(19.1)
Obese	100	123.2(32.1)	58	133.9(33.0)	42	108.3(24.2)

Table 23

#### *Weight Status Group Differences for PWC 170 Score (Watts) at 14 years*

Gender	ANOVA	<i>Post hoc</i> Tests p-value			
	<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Male	<b>3.756</b>	<b>.024</b>	.842	<b>.041</b>	.187
Female	<b>16.582</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>.002</b>	.387
Total	<b>12.675</b>	<b>&lt;.001</b>	<b>.036</b>	<b>&lt;.001</b>	<b>.034</b>

Note. **Bolded** indicate significant group differences  $p<.05$ .

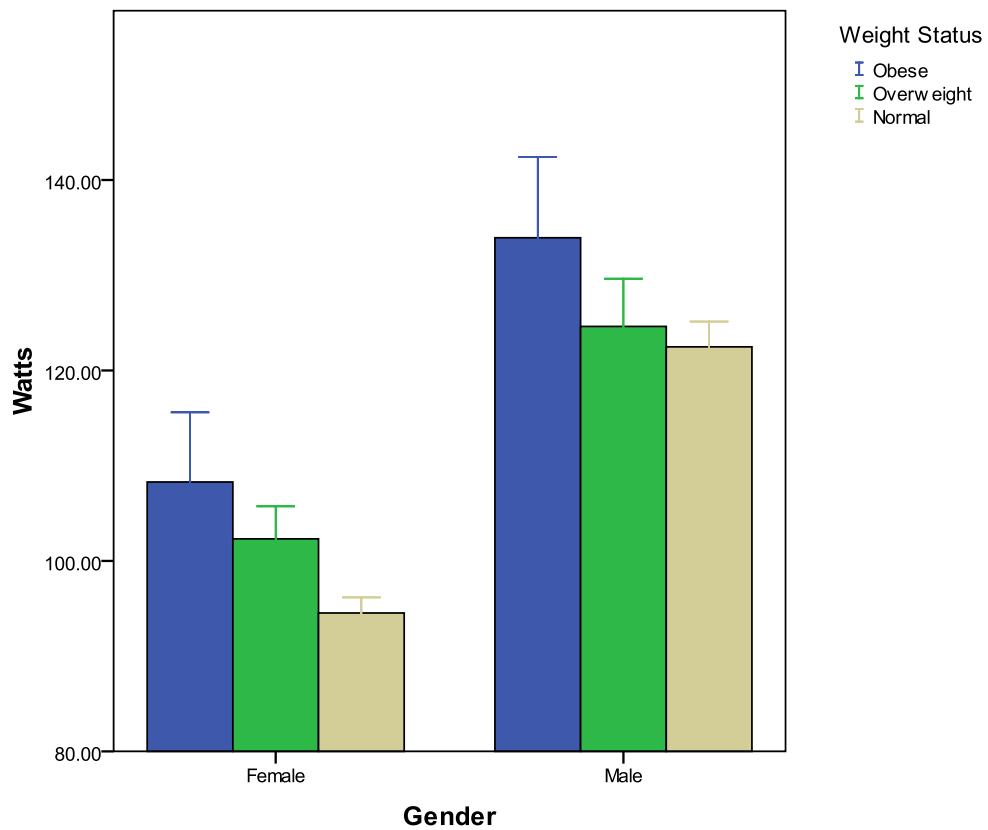


Figure 4. Weight status group differences for absolute aerobic fitness (PWC 170) at 14 years, for females and males.

### *Fitness tests.*

Gender differences were present for all fitness test items, with males scoring higher in muscle endurance (curl-ups) and muscle strength (basketball throw), while females were more flexible scoring higher in sit and reach and shoulder stretch items (Table 24). There were no weight status group differences for any of the fitness test items (Table 25, Table 26 and Table 27).

Table 24

*Gender Differences for Fitness Tests Muscle Endurance, Muscle Strength and Flexibility at age 14 Years*

Fitness Test Item	Total		Male		Female		Gender Difference	
	<i>n</i>	Mean(SD)	<i>n</i>	Mean(SD)	<i>n</i>	Mean(SD)	<i>t</i>	<i>p</i>
Curl-ups	824	22.5(17.7)	436	26.7(19.0)	388	17.7(14.7)	<b>7.646</b>	<b>&lt;.001</b>
Basketball throw	828	5.3(1.0)	437	5.8(1.0)	391	4.8(.7)	<b>17.408</b>	<b>&lt;.001</b>
Sit & Reach L	828	24.4(8.8)	437	20.9(7.7)	391	28.4(8.3)	<b>-13.470</b>	<b>&lt;.001</b>
Sit & Reach R	828	25.2(8.8)	437	21.6(7.8)	391	29.2(8.0)	<b>-13.901</b>	<b>&lt;.001</b>
Sit & Reach	828	23.7(9.1)	437	20.2(8.0)	391	27.6(8.6)	<b>-12.842</b>	<b>&lt;.001</b>
	<i>n</i>		<i>n</i>		<i>n</i>		$\chi^2$	<i>p</i>
Shoulder Stretch Left								
No	94		63		31		<b>8.557</b>	<b>.003</b>
Yes	733		374		359			
Shoulder Stretch Right								
No	38		28		10		<b>6.947</b>	<b>.008</b>
Yes	791		410		381			

Note. **Bolded** indicate significant group differences  $p < .05$ .

Table 25

*Weight Status Group Differences for Fitness Tests Muscle Endurance, Muscle Strength and Flexibility at age 14 Years*

Fitness Test Item	Gender	ANOVA		Post hoc Tests p-value		
		F	p	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Curl-ups	Male	1.782	.169	.221	.988	.533
	Female	.481	.618	.999	.707	.820
	Total	1.924	.147	.357	.706	.232
Basketball throw	Male	1.090	.337	.715	.548	.268
	Female	.731	.482	.999	.525	.680
	Total	2.307	.100	.697	.158	.059
Sit and reach left leg	Male	.145	.865	.941	.991	1.000
	Female	.503	.605	.906	.794	.969
	Total	.625	.535	.967	.674	.866
Sit and Reach right leg	Male	.047	.954	.995	.997	.989
	Female	.244	.783	.999	.901	.944
	Total	.238	.789	1.000	.908	.908
Sit and reach both legs	Male	.005	.995	1.000	1.000	1.000
	Female	.152	.859	1.000	.940	.962
	Total	.263	.769	.997	.899	.875



Table 26

*Descriptive Statistics for Fitness Tests Muscle Endurance, Muscle Strength and Flexibility at age 14 Years*

Fitness Test Item	BMI Weight Status determined at 14 years	Male		Female		Total	
		N	M (SD)	N	M (SD)	N	M (SD)
Curl-ups	Normal Weight	315	26.0(18.7)	282	18.0(14.9)	597	22.2(17.4)
	Overweight	90	30.0(19.2)	72	17.8(14.2)	162	24.6(18.2)
	Obese	31	24.8(20.8)	34	15.4(15.0)	65	19.9(18.5)
Basketball throw	Normal Weight	315	5.8(1.0)	284	4.8(.7)	599	5.3(1.0)
	Overweight	91	5.9(.8)	72	4.8(.6)	163	5.4(.9)
	Obese	31	5.6(.9)	35	4.7(.6)	66	5.1(.9)
Sit and reach left leg	Normal Weight	316	20.8(7.8)	285	28.2(8.4)	601	24.3(8.9)
	Overweight	90	21.2(7.6)	72	28.8(7.7)	162	24.6(8.5)
	Obese	31	21.2(8.2)	34	29.5(9.1)	65	25.5(9.6)
Sit and Reach right leg	Normal Weight	316	21.6(7.9)	285	29.1(8.0)	601	25.2(8.8)
	Overweight	90	21.8(7.3)	72	29.2(7.4)	162	25.1(8.2)
	Obese	31	21.3(8.4)	34	30.1(9.2)	65	25.9(9.8)
Sit and reach both legs	Normal Weight	316	20.2(8.1)	285	27.5(8.7)	601	23.7(9.2)
	Overweight	90	20.3(7.5)	72	27.6(8.1)	162	23.5(8.5)
	Obese	31	20.2(8.6)	34	28.4(9.4)	65	24.5(9.9)

Table 27

*Weight Status Group Differences for Fitness Test Flexibility (Shoulder Stretch) at age 14 Years*

Fitness Test Item	Total	Obese	overweight	Normal weight	$\chi^2$	p
Shoulder Stretch Left					.055	.973
No	94	7	19	68		
Yes	733	58	142	533		
Shoulder Stretch Right					1.500	.472
No	38	1	8	29		
Yes	791	64	154	573		

### ***Puberty.***

Puberty was assessed by questionnaire at age 14 years. For girls, puberty was assessed in three ways: menstruation age, Tanner scale for pubic hair (five stages), and Tanner scale for breast development (five stages). For boys, only the Tanner scale for pubic hair was applicable.

#### *Menstruation age for girls.*

In this sample of females ( $n=676$ ) the mean age at menstruation was 14.1 years ( $SD=0.19$ ) with a range from 13.0 – 14.9 years. Weight status groups each had the same mean age (14.1 years) for each category, with only the obese group having a smaller standard deviation (0.16 compared to 0.19). ANOVA group tests were not significant ( $F=.563$ ,  $p=.570$ ), with non-significant *post hoc* tests for all group comparisons.

#### *Tanner stages of pubertal development.*

Most females identified themselves at breast development stages three (34.2%) and four (51.2%). Significant differences were found between weight status groups ( $\chi^2=26.345$   $p=.001$ ) with proportionally more obese females identifying themselves at stage five (23.5% versus overweight 17.5%, normal weight 6.8%), and more overweight identifying themselves at stage four (58.3% versus obese 44.1%, normal weight 50.1%).

Within this sample no participants were in the fifth stage of pubic hair development. Compared to boys, more females were in the later stages of pubic hair development ( $\chi^2=18.461$   $p<.001$ ). No weight status group differences were found for the total sample ( $\chi^2=11.683$   $p=.069$ ), males ( $\chi^2=8.124$   $p=.229$ ) nor females ( $\chi^2=7.975$   $p=.240$ ). Please refer to Technical Report Tables 35 and 36 for full details.

The influence of puberty was also investigated in respect to absolute aerobic fitness (PWC 170) with a descriptive summary provided in Table 28. There was a significant group difference for stage of pubic hair development and aerobic fitness ( $F=10.585$   $p<.001$ ) with a positive relationship between stage of development and PWC 170 score. Post hoc comparisons were significant for all groups ( $p<.05$ ) except between the last two stages of development (stages 3 and 4,  $p=.667$ ). When a gender separated analysis was conducted, these group differences were only applicable to males ( $F=24.745$   $p<.001$ ) with significant differences between all groups ( $p<.05$ ), but not for females ( $F=1.614$   $p=.185$ ).

Table 28

*Comparison of Tanner Stages of Pubic Hair Development and Absolute Aerobic Fitness (PWC 170)*

Tanner Stages of Pubic Hair Development	Absolute Aerobic Fitness (PWC 170 Watts)					
	Total	Male		Female		
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Stage 1	43	94.3(21.3)	33	97.0(21.7)	10	85.3(18.3)
Stage 2	217	105.0(26.0)	124	112.7(27.1)	93	94.7(20.6)
Stage 3	572	112.7(30.0)	282	128.3(31.1)	290	97.7(18.9)
Stage 4	203	116.4(32.7)	98	137.7(28.2)	105	96.4(22.5)

### **Behavioural factors.**

Broadly, behavioural factors included physical activity; sedentary behaviour; self-perception; and parent reported academic, social and behavioural progress.

### ***Physical activity behaviours.***

Physical activity behaviours included frequency of visits to the local park or playground, parent reported activity levels, physical activity level, and attitudes and values to physical activity.

*Frequency of visits to a park or playground.*

Gender differences for frequency of visits to a park or playground were reported by parents at ages 1 and 2 years, but not 3 years. At 1 and 2 years more females *never* visited the park or playground, while more males visited the park or playground *everyday* (Table 29).

Table 29

*Gender Differences for Frequency of Visits to a Park or Playground at ages 1, 2 and 3 Years*

Mean age at Follow-up	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference	
				$\chi^2$	<i>p</i>
<b>1 Year (<i>n</i>=1,204)</b>					
Never	107	49	58	<b>12.459</b>	<b>.014</b>
Seldom <1mth	256	140	116		
Occasionally <1wk	454	248	206		
Often >1wk	322	153	169		
Everyday	65	44	21		
<b>2 Years (<i>n</i>=366)</b>					
Never	18	3	15	<b>11.626</b>	<b>.020</b>
Seldom <1mth	62	34	28		
Occasionally <1wk	140	62	78		
Often >1wk	131	67	54		
Everyday	15	10	5		
<b>3 Years (<i>n</i>=386)</b>					
Never	17	5	12	4.829	.305
Seldom <1mth	67	32	35		
Occasionally <1wk	164	88	76		
Often >1wk	116	63	53		
Everyday	22	13	9		

Note. **Bolded** indicate significant group differences  $p < .05$ .

Weight status group differences were found at ages 1 and 3 years, but not 2 years. Generally, compared to the normal weight group, more obese, followed by overweight visited the park *infrequently* (Table 30).

Table 30

*Weight Status Group Differences for Outdoor Play Space at ages 1, 2 and 3 Years*

Follow-up	Total	Obese	Overweight	Normal weight	$\chi^2$	<i>p</i>
1 Year					<b>17.046</b>	<b>.030</b>
Never	107	15	24	68		
Seldom <1mth	256	30	44	182		
Occasionally <1wk	454	34	92	328		
Often >1wk	322	16	57	249		
Everyday	65	4	13	48		
2 Years					6.634	.577
Never	18	3	2	13		
Seldom <1mth	62	4	10	48		
Occasionally <1wk	140	13	30	97		
Often >1wk	131	7	25	99		
Everyday	15	0	3	12		
3 Years					<b>22.735</b>	<b>.004</b>
Never	17	3	7	7		
Seldom <1mth	67	11	18	38		
Occasionally <1wk	164	9	33	122		
Often >1wk	116	8	17	91		
Everyday	22	3	2	17		

Note. **Bolded** indicate significant group differences  $p < .05$ .

*Parent reported activity levels at ages 6, 8 and 10 years.*

At age 6 years, more males were involved in organised sport (at school or with a club), while more females were involved in organised activity (music, dancing, kindy gym, other clubs). By age 8 years, there were no gender differences in

organised sport participation, although males had higher activity levels (Table 31). By 10 years, parents reported that males participated in more total activity over the week and weekend, as well as higher levels of moderate and vigorous intensity activity (Table 32).

Table 31

*Gender Differences for Parent Reported Organised Sport, Organised Activity and Level of Activity at ages 6 and 8 Years*

Variable	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
6 Years Organised Sport ( <i>n</i> =1,299)					
No	873	430	443	<b>7.818</b>	<b>.005</b>
Yes	426	245	181		
6 Years Organised Activity ( <i>n</i> =1,299)					
No	863	534	329	<b>96.620</b>	<b>&lt;.001</b>
Yes	436	144	292		
8 Years Organised Sport ( <i>n</i> =1,303)					
No	259	125	134	1.485	.223
Yes	1044	548	496		
8 Years Level of activity ( <i>n</i> =1,308)					
Sedentary or little	148	64	84	<b>16.221</b>	<b>.001</b>
Slightly active	655	317	338		
Active	505	295	210		

Note. **Bolded** indicate significant group differences  $p < .05$ .

Table 32

*Gender Differences for Parent Reported Activity Level (None, Light, Moderate, and Vigorous) and Activity Time at age 10 Years*

Activity Level	Total		Male		Female		Gender difference	
	<i>N</i>	<i>M (SD)</i>	<i>N</i>	<i>M (SD)</i>	<i>N</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
Vigorous /wk	1,403	0.9(1.2)	729	1.0(1.2)	674	0.9(1.2)	<b>2.287</b>	<b>.022</b>
Moderate /wk	1,403	2.1(2.1)	729	2.4(2.1)	674	1.8(2.0)	<b>5.902</b>	<b>&lt;.001</b>
Light /wk	1,403	1.5(2.0)	729	1.4(1.9)	674	1.7(2.1)	<b>-2.749</b>	<b>.006</b>
None /wk	1,403	1.1(1.6)	729	1.0(1.6)	674	1.3(1.7)	<b>-3.319</b>	<b>.001</b>
Vigorous /wkend	1,403	0.4(0.6)	729	0.5(0.6)	674	0.3(0.5)	<b>3.812</b>	<b>&lt;.001</b>
Moderate /wkend	1,403	1.0(0.9)	729	1.1(0.9)	674	0.9(0.9)	<b>4.206</b>	<b>&lt;.001</b>
Light /wkend	1,403	0.4(0.7)	729	0.4(0.7)	674	0.5(0.7)	-1.404	.161
None /wkend	1,403	0.5(0.7)	729	0.4(0.6)	674	0.6(0.8)	<b>-4.546</b>	<b>&lt;.001</b>
Total Vigorous	1,403	1.3(1.6)	729	1.5(1.6)	674	1.2(1.5)	<b>3.154</b>	<b>.002</b>
Total Moderate	1,403	3.1(2.7)	729	3.5(2.8)	674	2.6(2.6)	<b>5.960</b>	<b>&lt;.001</b>
Total Light	1,403	2.0(2.4)	729	1.8(2.4)	674	2.1(2.5)	<b>-2.674</b>	<b>.008</b>
Total None	1,403	1.6(2.1)	729	1.4(2.0)	674	1.8(2.2)	<b>-4.115</b>	<b>&lt;.001</b>
Suburb /wk	1,403	4.0(3.0)	729	4.3(3.0)	674	3.8(3.0)	<b>3.013</b>	<b>.003</b>
Suburb wkend	1,403	1.4(1.2)	729	1.5(1.2)	674	1.3(1.2)	<b>2.804</b>	<b>.005</b>
Total suburb	1,403	5.4(3.9)	729	5.7(3.8)	674	5.1(3.9)	<b>3.196</b>	<b>.001</b>
Total activity	1,403	6.4(3.9)	729	6.7(3.8)	674	6.0(4.0)	<b>3.633</b>	<b>&lt;.001</b>
Total time (mins)	59	847(303)	34	852(348)	25	840(236)	0.155	.878

*Note.* wk=week, wkend=weekend, mins=minutes, Suburb = activity in suburb. **Bolded** indicate significant group differences  $p<.05$ .

Weight status group differences were found for organised activity at age 6 and 8 years only. Here, fewer obese were involved in organised activity at 6 years and they were less active at 8 years, when compared to overweight and normal weight groups (Table 33). At 10 years a number of trends were found (Table 34). The amount of vigorous activity during the week and on weekends was lower between the obese, compared to normal weight and overweight groups, and this was also the case for moderate and no activity on the weekends. There were no weight status group differences in light activity during the week or on weekends, or

moderate and low activity levels during the week. When total activity levels (week and weekends) were analysed, only vigorous activity and no activity were significant. Here the obese group, compared to normal weight and overweight, had lower levels of vigorous activity, and higher levels of no activity. Comparison of weekday versus weekend activity found a significant weight status group difference for weekend activity. Overall, the obese group were less *vigorously active* and more *inactive*, compared to the normal weight and overweight groups (Table 35).

Table 33

*Weight Status Group Differences for Parent Reported Organised Sport, Organised Activity and Level of Activity at ages 6 and 8 Years*

Activity	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
6 Years Organised Sport ( $n=1,299$ )					1.383	.501
No	873	71	158	644		
Yes	426	27	81	318		
6 Years Organised Activity ( $n=1,299$ )					<b>7.714</b>	<b>.021</b>
No	863	79	157	627		
Yes	436	21	81	334		
8 Years Organised Sport ( $n=1,303$ )					4.522	.104
No	259	28	46	185		
Yes	1044	72	199	773		
8 Years Level of activity ( $n=1,308$ )					<b>24.076</b>	<b>&lt;.001</b>
Sedentary or little	148	23	30	95		
Slightly active	655	56	128	471		
Active	505	22	88	395		

Note. **Bolded** indicate significant group differences  $p<.05$ .



Table 34

*Weight Status Group Differences for Parent Reported Activity Level (None, Light, Moderate, and Vigorous) and Activity Time at age 10 Years*

Activity Level	Gender	ANOVA		Post hoc Tests p-value		
		F	p	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Vigorous/wk	Male	<b>4.075</b>	<b>.017</b>	.939	<b>.005</b>	<b>.007</b>
	Female	2.363	.095	.950	.051	.202
	Total	<b>5.919</b>	<b>.003</b>	1.000	<b>&lt;.001</b>	<b>.002</b>
Moderate/wk	Male	1.867	.155	.156	.934	.846
	Female	1.452	.235	.316	.898	.988
	Total	0.093	.911	.985	.993	.971
Light/wk	Male	1.202	.301	.446	.628	1.000
	Female	1.771	.171	.935	.181	.147
	Total	1.989	.137	.935	.098	.340
None/wk	Male	<b>7.683</b>	<b>&lt;.001</b>	<b>.031</b>	<b>.025</b>	.684
	Female	1.304	.272	.303	.987	.634
	Total	2.883	.056	.875	.118	.342
Vigorous/wkend	Male	<b>6.584</b>	<b>.001</b>	.987	<b>&lt;.001</b>	<b>&lt;.001</b>
	Female	0.131	.877	.993	.955	.989
	Total	<b>4.609</b>	<b>.010</b>	1.000	<b>.002</b>	<b>.009</b>
Moderate/wkend	Male	2.883	.057	.455	.112	.706
	Female	<b>4.016</b>	<b>.018</b>	.438	.057	<b>.012</b>
	Total	<b>4.361</b>	<b>.013</b>	1.000	<b>.012</b>	<b>.033</b>
Light/wkend	Male	1.236	.291	.254	.991	.813
	Female	2.406	.091	.817	<b>.029</b>	.201
	Total	2.391	.092	.228	.197	.935
None/wkend	Male	<b>10.494</b>	<b>&lt;.001</b>	.483	<b>.002</b>	<b>.029</b>
	Female	<b>5.175</b>	<b>.006</b>	.629	.063	<b>.025</b>
	Total	<b>12.488</b>	<b>&lt;.001</b>	1.000	<b>&lt;.001</b>	<b>.001</b>

Activity Level	Gender	ANOVA		Post hoc Tests p-value		
		<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Total Vigorous	Male	<b>6.071</b>	<b>.002</b>	.945	<b>&lt;.001</b>	<b>.001</b>
	Female	7.787	.168	.954	.164	.393
	Total	<b>7.019</b>	<b>.001</b>	1.000	<b>&lt;.001</b>	<b>.001</b>
Total Moderate	Male	2.188	.113	.167	.618	.993
	Female	1.624	.198	.258	.992	.578
	Total	0.320	.726	.992	.831	.936
Total Light	Male	1.432	.240	.304	.690	.997
	Female	2.188	.113	.994	.092	.113
	Total	2.430	.088	.731	.068	.402
Total None	Male	<b>9.687</b>	<b>&lt;.001</b>	.053	<b>.005</b>	.289
	Female	2.304	.101	.280	.651	.226
	Total	<b>6.136</b>	<b>.002</b>	.932	<b>.011</b>	<b>.048</b>
Total Suburb wk	Male	<b>3.289</b>	<b>.038</b>	.102	.323	1.000
	Female	2.247	.106	.486	.424	.155
	Total	2.227	.108	.880	.150	.439
Total Suburb wkend	Male	<b>3.912</b>	<b>.020</b>	.257	.064	.660
	Female	<b>4.457</b>	<b>.012</b>	.530	<b>.040</b>	<b>.011</b>
	Total	<b>5.707</b>	<b>.003</b>	.981	<b>.004</b>	<b>.021</b>
Total Suburb	Male	<b>3.952</b>	<b>.020</b>	.095	.161	.980
	Female	<b>3.259</b>	<b>.039</b>	.420	.216	.060
	Total	<b>3.553</b>	<b>.029</b>	.900	<b>.042</b>	.178
Total Activity	Male	<b>5.376</b>	<b>.005</b>	.128	<b>.026</b>	.650
	Female	<b>3.296</b>	<b>.038</b>	.605	.145	.054
	Total	<b>5.587</b>	<b>.004</b>	.859	<b>.006</b>	.050
Total Activity time	Male	1.630	.212	.697	<b>.015</b>	.266
	Female	1.301	.292	.416	.296	.755
	Total	1.776	.179	.857	<b>.046</b>	.230

Note. wk=week, wkend=weekend, Suburb = activity in suburb. **Bolded** indicate significant group differences  $p<.05$ .

Table 35

*Descriptive Statistics for Parent Reported Activity Level (None, Light, Moderate, and Vigorous) and Activity Time at age 10 Years*

Activity Level	BMI Weight Status determined at 14 years	Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Vigorous/wk	Normal Weight	530	1.0(1.2)	501	0.9(1.2)
	Overweight	136	1.1(1.2)	127	0.9(1.2)
	Obese	63	0.6(1.0)	46	0.5(1.0)
Moderate/wk	Normal Weight	530	2.5(2.1)	501	1.7(1.9)
	Overweight	136	2.1(2.1)	127	2.0(2.1)
	Obese	63	2.3(2.1)	46	1.9(2.2)
Light/wk	Normal Weight	530	1.5(2.0)	501	1.7(2.1)
	Overweight	136	1.2(1.9)	127	1.8(2.1)
	Obese	63	1.2(1.8)	46	1.2(1.9)
None/wk	Normal Weight	530	0.9(1.5)	501	1.3(1.7)
	Overweight	136	1.3(1.6)	127	1.1(1.6)
	Obese	63	1.5(1.9)	46	1.4(2.0)
Vigorous/wkend	Normal Weight	530	0.5(0.6)	501	0.3(0.5)
	Overweight	136	0.5(0.6)	127	0.3(0.5)
	Obese	63	0.2(0.4)	46	0.3(0.6)
Moderate/wkend	Normal Weight	530	1.1(0.9)	501	0.9(0.9)
	Overweight	136	1.0(0.9)	127	1.0(0.9)
	Obese	63	0.9(0.9)	46	0.5(0.9)
Light/wkend	Normal Weight	530	0.4(0.7)	501	0.5(0.8)
	Overweight	136	0.3(0.6)	127	0.4(0.7)
	Obese	63	0.4(0.7)	46	0.2(0.6)
None/wkend	Normal Weight	530	0.3(0.6)	501	0.6(0.7)
	Overweight	136	0.4(0.6)	127	0.5(0.7)
	Obese	63	0.7(0.8)	46	0.9(0.9)
Total Vigorous	Normal Weight	530	1.5(1.7)	501	1.2(1.5)
	Overweight	136	1.6(1.6)	127	1.2(1.5)
	Obese	63	0.8(1.3)	46	0.8(1.4)

Activity Level	BMI Weight Status determined at 14 years	Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Total Moderate	Normal Weight	530	3.6(2.7)	501	2.5(2.5)
	Overweight	136	3.1(2.8)	127	3.0(2.7)
	Obese	63	3.2(2.8)	46	2.4(2.8)
Total Light	Normal Weight	530	1.9(2.4)	501	2.2(2.5)
	Overweight	136	1.5(2.3)	127	2.2(2.5)
	Obese	63	1.6(2.2)	46	1.4(2.3)
Total None	Normal Weight	530	1.2(1.9)	501	1.9(2.2)
	Overweight	136	1.7(2.1)	127	1.5(2.1)
	Obese	63	2.3(2.4)	46	2.3(2.6)
Total Suburb wk	Normal Weight	530	4.4(2.9)	501	3.8(3.0)
	Overweight	136	3.8(3.1)	127	4.1(2.9)
	Obese	63	3.8(3.1)	46	3.0(3.4)
Total Suburb wkend	Normal Weight	530	1.5(1.2)	501	1.3(1.2)
	Overweight	136	1.3(1.2)	127	1.4(1.2)
	Obese	63	1.1(1.3)	46	0.8(1.2)
Total Suburb	Normal Weight	530	6.0(3.7)	501	5.0(3.9)
	Overweight	136	5.1(4.0)	127	5.6(3.8)
	Obese	63	4.9(4.0)	46	3.8(4.4)
Total Activity	Normal Weight	530	7.0(3.7)	501	6.0(4.0)
	Overweight	136	6.2(4.1)	127	6.4(3.8)
	Obese	63	5.6(4.0)	46	4.7(4.3)
Total Activity time	Normal Weight	25	817(259)	19	880(249)
	Overweight	7	1,041(578)#	3	764(93)
	Obese	2	629(35)	3	663(166)

*Note.* wk=week, wkend=weekend, Suburb = activity in suburb. #checked data and this does contain 2 cases with high activity times, but they are not outliers to the sample, hence are not excluded.

Activity level - count of the number of times per week activity level reported.

Activity time – measured in minutes.

*Physical activity level.*

Physical activity level is represented by pedometer step counts at 14 years, and includes a mean daily step count -per weekday, -per weekend and -per week. For all measures, males recorded more steps than females (Table 36).

Table 36

*Gender Differences for Mean Daily Step Count at 14 Years*

Mean daily step count	Total (n=604)	Male (n=288)	Female (n=316)	Gender Difference	
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
Weekday	11,017 (4,110)	12,075 (4,542)	10,052 (3,403)	<b>6.145</b>	<b>&lt;.001</b>
Weekend	9,849 (4,865)	10,910 (5,437)	8,882 (4,051)	<b>5.158</b>	<b>&lt;.001</b>
Week	10,683 (3,902)	11,742 (4,348)	9,718 (3,159)	<b>6.491</b>	<b>&lt;.001</b>

*Note.* **Bolded** indicate significant group differences  $p<.05$ .

Table 37

*Weight Status Group Differences for Mean Daily Step Count at 14 Years*

Mean daily step count	Gender	ANOVA	Post hoc Tests p-value			
		<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Weekday	Male	1.474	.231	.416	.534	.986
	Female	2.992	.052	.570	.052	<b>.018</b>
	Total	1.498	.224	.990	.220	.390
Weekend	Male	.680	.507	.998	.186	.424
	Female	2.976	.052	.976	<b>.002</b>	<b>.017</b>
	Total	2.210	.111	.964	<b>.010</b>	.074
Week	Male	1.286	.278	.582	.359	.887
	Female	<b>3.412</b>	<b>.034</b>	.807	<b>.005</b>	<b>.004</b>
	Total	2.104	.123	.977	.070	.186

*Note.* **Bolded** indicate significant group differences  $p<.05$ .

Overall there were no weight status group differences in physical activity level measures. Gender separated analysis found weight status group differences for females only across variables, and that this difference was between the obese group, compared to normal weight and overweight (Table 37) with the obese group consistently taking less steps (Table 38). Although a similar trend was noted for the males, there were no significant differences.

Table 38

*Descriptive Statistics for Mean Daily Steps Gender at 14 Years*

Mean daily step count	BMI Weight Status determined at 14 Years	Male		Female		Total	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Weekday	Normal Weight	210	12,350 (4,587)	232	10,001 (3,187)	442	11,117 (4,083)
	Overweight	56	11,431 (4,309)	67	10,642 (4,162)	123	11,001 (4,230)
	Obese	22	11,087 (4,584)	17	8,434 (2,362)	39	9,930 (3,967)
Weekend	Normal Weight	210	11,041 (5,706)	232	9,060 (4,075)	442	10,001 (5,011)
	Overweight	56	10,926 (5,128)	67	8,847 (4,164)	123	9,793 (4,724)
	Obese	22	9,618 (3,028)	17	6,593 (2,413)	39	8,300 (3,135)
Week	Normal Weight	210	11,976 (4,517)	232	9,732 (3,022)	442	10,798 (3,964)
	Overweight	56	11,286 (3,842)	67	10,129 (3,705)	123	10,656 (3,797)
	Obese	22	10,668 (3,779)	17	7,908 (1,949)	39	9,465 (3,378)

Physical activity was also investigated as a tertile variable (Figure 5), and dichotomous mean cut-point, median cut-point and 12,000 step count cut-point. For details refer to Technical Report Table 37 and Table 38. Overall, Figure 5 shows that for both males and females, fewer obese were classified in the highest tertile (i.e. high active). For females, more obese were low active, while for boys more obese were moderately active. Overweight males and females were similar to the normal weight group in their mean step count levels.

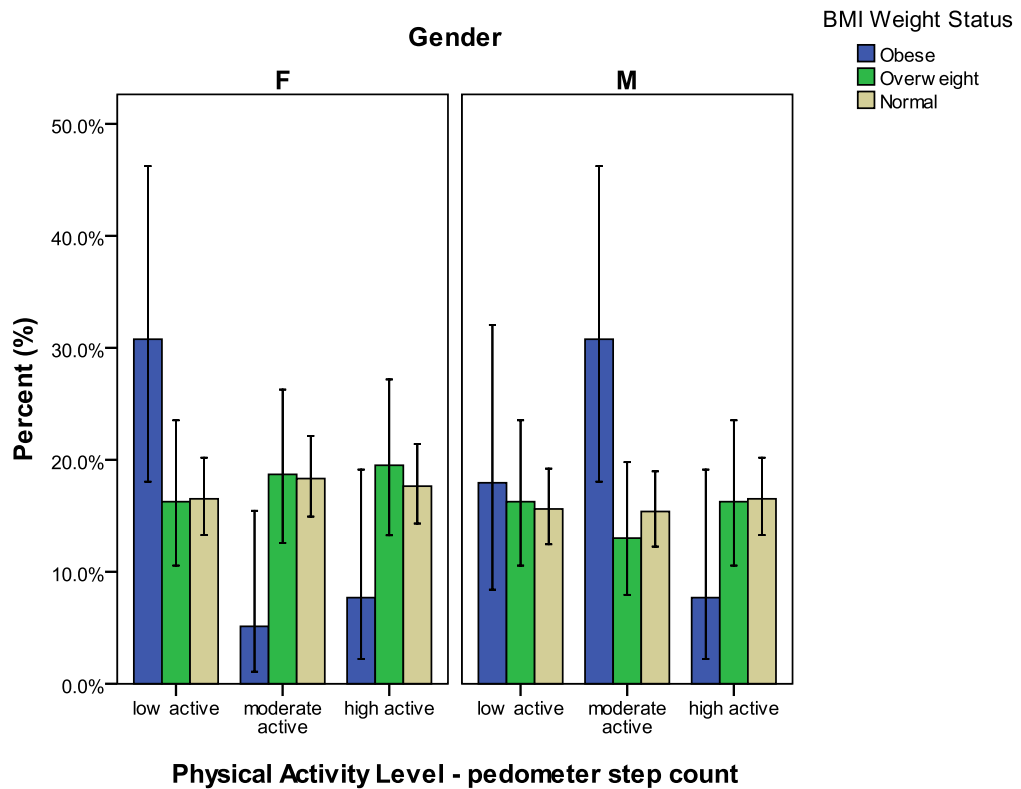


Figure 5. Comparison of weight status group differences (determined at 14 years), and mean step count per week tertiles, at 14 years for females (F) ( $p < .05$ ) and males (M).

### *Physical activity attitudes and values.*

At follow-up 14 years, survey items were used to assess physical activity attitudes and values. These included the broad areas of encouragement and support to exercise, physical education and exercise, effects of physical activity, importance of physical activity, excuses for not increasing physical activity in the future, and other physical activity questions. A summary is provided here for each broad area, with specific tables provided in the Technical Report. Specific details on the derived scores are presented last.

### *Encouragement and support to exercise.*

Gender differences were found only for *friends' frequency of exercise*, *dad encourages exercise*, *dad helps exercise*, and *friends help exercise*. For each item, males reported a higher frequency of exercise, compared to girls (Technical Report Table 39).

Significant weight status group differences were found for dad and mum's frequency of exercise: *dad helps exercise* (for females  $\chi^2=20.01$   $p=.003$ , but not males  $\chi^2=9.351$   $p=.155$ ), and *mum helps exercise* (for males  $\chi^2=21.65$   $p=.001$ , but not females  $\chi^2=7.78$   $p=.254$ ) (Technical Report Table 40). Children of normal weight tended to have mothers ( $p=.012$ ) and fathers ( $p<.001$ ) who exercised *two or more times a week* and also parents who *helped them to exercise* ( $p<.05$ ).

### *Physical education and exercise.*

Overall males felt more positively than females about physical education and exercise survey items (Technical Report Table 41). Males were more positive in how they felt about physical education ( $p<.001$ ), had higher reported duration of exercise out of school ( $p <.001$ ), were different in intentions for future exercise ( $p<.001$ ), were more confident others would not make fun of them ( $p <.001$ ), were more likely to participate without peers ( $p <.001$ ), were more likely to be active if low on energy ( $p =.014$ ) not good at it ( $p =.046$ ) had no help ( $p <.001$ ) and friends don't participate ( $p =.015$ ), compared to females.

There were some weight status group differences which differed for males and females (Technical Report Table 42). Fewer obese females enjoyed physical education ( $\chi^2=25.821$   $p=.004$ ), with this group *not confident* to participate, and reporting barriers *no-one to exercise with* ( $\chi^2=23.153$   $p=.004$ ) and *no friends* ( $\chi^2=20.652$   $p=.008$ ), and found *duration of hard exercise* ( $\chi^2=23.765$   $p=.008$ ). More obese males were unsure about increasing their *exercise in the future* (for males  $\chi^2=17.577$   $p=.007$ ). Overall more obese individuals were *not confident* to exercise with *no energy* ( $\chi^2=15.708$   $p=.047$ ).



### *Effects of physical activity.*

Individuals were surveyed on perceived positive (e.g. *makes me healthy*) and negative (e.g. *people laugh at me*) effects of physical activity. More females responded *unlikely* and *neither*, while males were more likely to report positive effects such as *have lots of fun, make parents happy, spend time with friends, compete* and *win something* (Technical Report Table 43).

There were significant differences across weight status groups on the likelihood of positive effects of physical activity (Technical Report Table 44). Normal weight individuals were more likely to report that exercise makes them healthy (76.7% vs overweight 67.4% and obese 56.9%  $p<.001$ ); study better ( $p=.045$ ); indifferent or a little likely to improve appearance ( $p<.001$ ); and reported as more unlikely that people would laugh at them ( $p<.001$ ). Compared to overweight and normal weight individuals, obese individuals were less likely to: report that exercise made them feel good about them self ( $p=.047$ ); made them fit ( $p<.001$ ); and reported *very likely* to make new friends ( $p=.038$ ), compete ( $p=.001$ ) and win something ( $p<.001$ ). More overweight individuals reported that exercise was *very likely* to help them lose or control weight (78.1% vs. obese 70.6% and normal weight 60.6%), and a higher proportion of normal weight individuals were indifferent ( $p<.001$ ).

### *Importance of physical activity.*

Compared to males, more females rated as *very likely* the importance of physical activity for improving appearance ( $p<.001$ ), feeling good about self ( $p=.001$ ), losing or controlling weight ( $p<.001$ ), spending time with and making new friends ( $p<.001$ ); and rated as *unlikely* that physical activity prevented them from doing other things ( $p=.040$ ). Compared to females, more males rated as *very likely* the importance of physical activity for competition ( $p<.001$ ) and winning something ( $p<.001$ ). (Full details provided at Technical Report Table 45).

There were significant weight status group differences across groups for the likelihood of positive effects of physical activity (Technical Report Table 46).

Compared to normal weight and overweight, fewer obese individuals rated as *very likely* that exercise makes them healthy ( $p=.008$ ), makes them study better ( $p=.003$ ), and rated as more *unlikely* that exercise made them feel good about self ( $p<.001$ ) (Figure 6), made them fit ( $p<.001$ ) have fun ( $p<.001$ ) spend time with friends ( $p=.002$ ) make new friends ( $p<.001$ ), compete ( $p=.040$ ), and were more indifferent to making their parents happy ( $p=.003$ ). Compared to normal weight, obese and overweight individuals rated exercise as important for weight control or loss ( $p<.001$ ) (Figure 7).

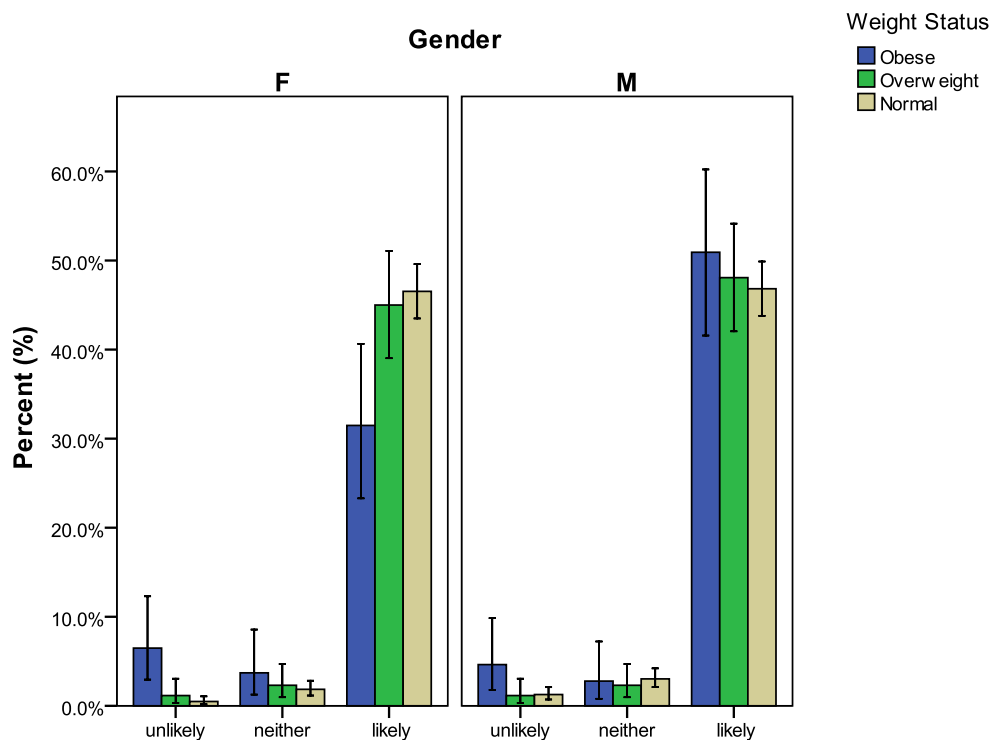


Figure 6. Comparison of weights status group differences (determined at 14 years) for importance of being physically active to feel good about self, at 14 years, for females (F) and males (M) with 95% CI intervals ( I ).

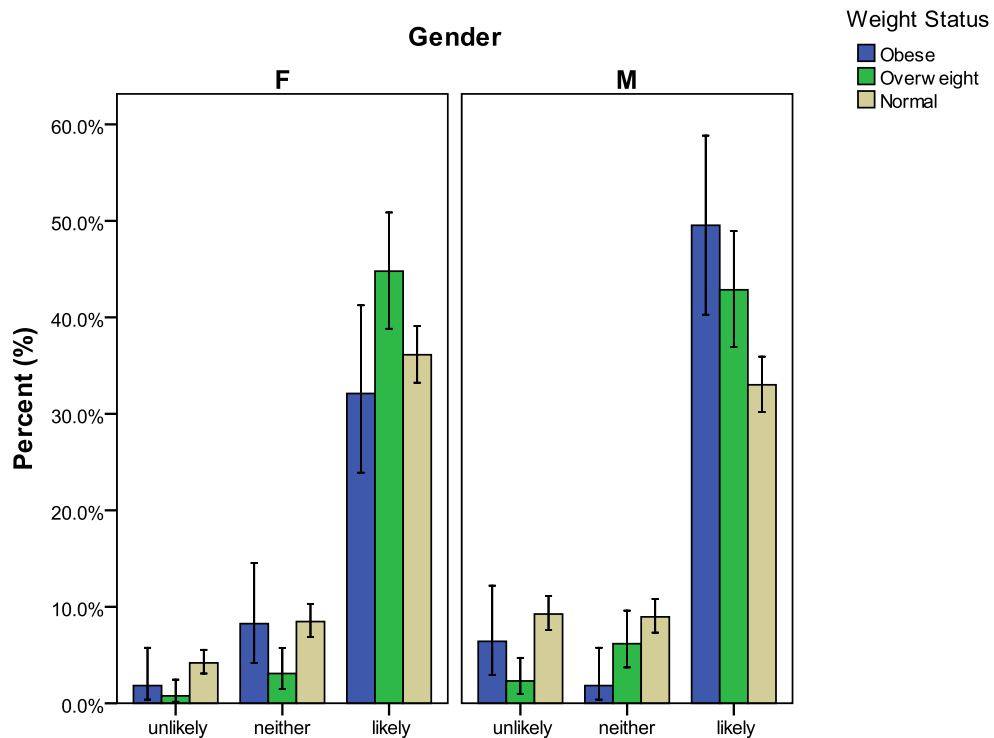


Figure 7. Comparison of weights status group differences (determined at 14 years) for importance of being physically active to lose or control weight, at 14 years, for females (F) and males (M) with 95% CI intervals ( I ).

Item responses to the effect of physical activity compared to, the importance of physical activity, demonstrated different gender and weight status group differences across constructs. For example, the perceived effects of physical activity improving appearance were different across weight status groups, but there was no difference between weight status groups for the importance of physical activity for improving appearance. This was similar for the item winning something. For other items (such as *makes me healthy* and *study better*) the differences were similar, but a higher proportion rated as *very likely* the importance, compared to the effects of physical activity. Therefore it seemed that across weight status groups, the relative importance of physical activity was recognised. For those individuals overweight or obese, physical activity was seen as important, but they did not think that physical activity would be effective.

### *Excuses for not increasing physical activity in the future.*

Overall, females were more likely than males to make excuses for not increasing their physical activity in the future (Technical Report Table 47). Gender differences were significant for *already do lots* ( $p<.001$ ), *insufficient time* ( $p<.001$ ), *other likes* ( $p=.003$ ), *don't enjoy* ( $p<.001$ ), *no skills* ( $p<.001$ ), and *not good at sport* ( $p<.001$ ).

Gender separated weight status group analysis found that in addition to full sample differences discussed below, weight status group differences were only significant for females for *self conscious* ( $\chi^2=32.328$   $p<.001$ ) and *parents don't help* ( $\chi^2=13.905$   $p=.031$ ), and for males *not good at sport* ( $\chi^2=17.386$   $p=.008$ )

Unlike previous attitudes and values to physical activity survey items where the overweight group tended to respond in a similar manner to the normal weight group, the overweight group responded in a similar way to the obese group for excuses for not increasing physical activity in the future (Technical Report Table 48). More obese individuals reported *applies a little* for *already do lots* ( $p<.001$ ) (Figure 8), more likely to be self-conscious ( $p=.002$ ), have other likes ( $p<.001$ ), didn't enjoy physical activity ( $p<.001$ ), not good at sports ( $p=.003$ ), people laughed at them ( $p<.001$ ), poor health ( $p<.001$ ) (Figure 9). Overweight and obese were more likely to report they had no company ( $p=.002$ ), had no skills ( $p<.001$ ) (Figure 10).

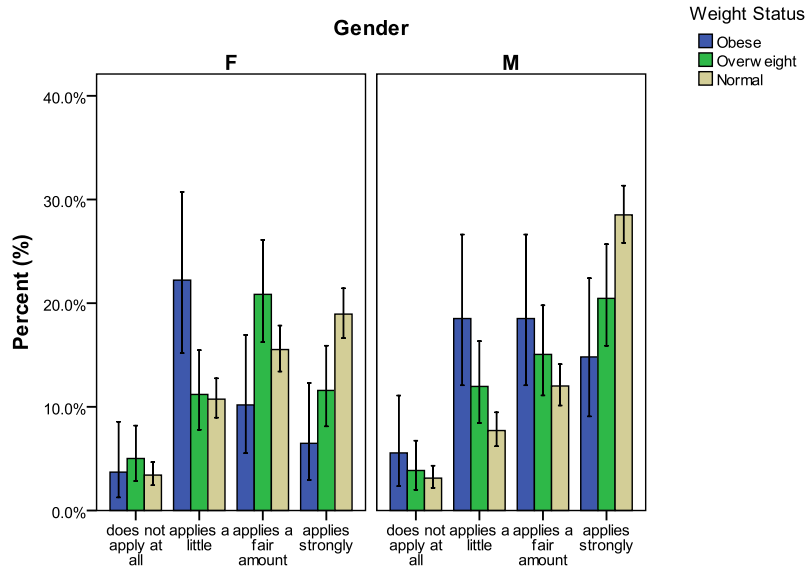


Figure 8. Comparison of weight status group differences (determined at 14 years) for excuse I already do lots, at 14 years, for females (F) and males (M) with 95% CI intervals ( I ).

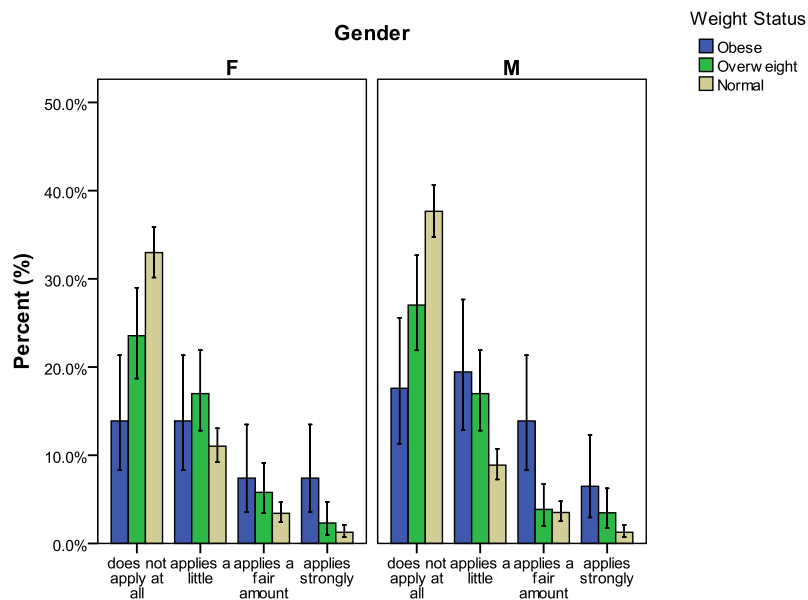


Figure 9. Comparison of weight status group differences (determined at 14 years) for excuse poor health, at 14 years, for females (F) and males (M) with 95% CI intervals ( I ).

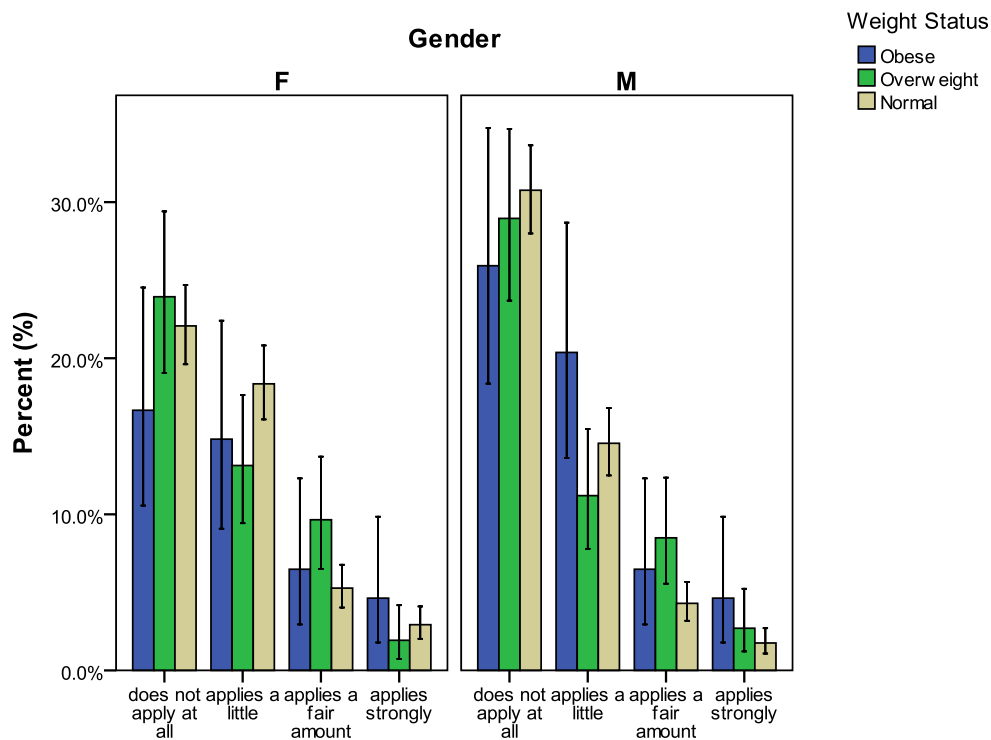


Figure 10. Comparison of weight status group differences (determined at 14 years) for excuse I have no skills, at 14 years, for females (F) and males (M) with 95% CI intervals ( I ).

### *Derived attitudes and values score.*

A composite score was derived for encouragement, participation, effects, importance and excuses from each of the previously described physical activity attitudes and values variables. Participation scores were higher for males than females, and females made more excuses than males (Table 39). Weight status differences were found for the derived mean scores of encouraged to exercise ( $p=.015$ ), effects of physical activity ( $p<.001$ ), importance of physical activity ( $p=.018$ ) and excuses for not being active in the future ( $p<.001$ ). The biggest difference was between the normal weight group compared to the overweight ( $p=.010$ ) and obese ( $p<.001$ ) groups, with girls making more excuses than boys ( $p=.024$ ) (Table 40 and Table 41).

Table 39

*Gender Differences for Derived Attitude and Value Scores at 14 Years*

Derived Total Score	Total		Male		Female		Gender Difference	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>
Encouragement	891	18.8(6.2)	455	19.1(6.2)	436	18.5(6.1)	1.389	.165
Participation	1391	15.1(5.4)	722	15.9(5.4)	669	14.3(5.3)	<b>5.577</b>	<b>&lt;.001</b>
Effects	1371	66.8(9.9)	709	67.2(10.0)	662	66.4(9.7)	1.524	.128
Importance	1344	61.7(7.9)	688	61.7(8.2)	656	61.8(7.5)	-.190	.849
Excuses	1357	12.8(7.0)	704	12.4(6.8)	653	13.2(7.1)	<b>-2.256</b>	<b>.024</b>

*Note.* **Bolded** indicate significant group differences  $p < .05$ . Derived scores were calculated based on scale score range and number of items. Therefore maximum scores for each derived score were: Encouragement (44), Participation (39); Effects (90); Importance (90); and Excuses (60).

Table 40

*Weight Status Group Differences for Derived Attitude and Value Scores at 14 Years*

Derived Total Score	Gender	ANOVA <i>F</i>	<i>p</i>	Post hoc Tests p-value		
				Normal versus Overweight	Normal versus Obese	Overweight versus obese
Encouragement	Male	3.014	.050	.124	.314	.981
	Female	1.525	.219	.686	.299	.728
	Total	<b>4.232</b>	<b>.015</b>	.095	.091	.791
Participation	Male	.548	.578	1.000	.717	.809
	Female	<b>4.167</b>	<b>.016</b>	.791	<b>.028</b>	.141
	Total	2.920	.054	.890	.092	.289
Effects	Male	<b>5.123</b>	<b>.006</b>	.997	<b>.020</b>	<b>.030</b>
	Female	<b>3.312</b>	<b>.037</b>	.612	.184	.514
	Total	<b>7.777</b>	<b>&lt;.001</b>	.887	<b>.004</b>	<b>.024</b>
Importance	Male	1.728	.178	.993	.359	.378
	Female	2.399	.092	1.000	.419	.464
	Total	<b>4.019</b>	<b>.018</b>	.997	.118	.139
Excuses	Male	<b>6.687</b>	<b>.001</b>	<b>.034</b>	.071	.934
	Female	<b>10.181</b>	<b>&lt;.001</b>	.281	<b>.003</b>	.067
	Total	<b>14.953</b>	<b>&lt;.001</b>	<b>.010</b>	<b>&lt;.001</b>	.164

*Note.* **Bolded** indicate significant group differences  $p < .05$ .

Table 41

*Descriptive Statistics for Derived Attitude and Value Scores at 14 Years*

Derived Total Score	BMI Weight Status determined at year 14	Male		Female		Total	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Encouragement	Normal Weight	351	19.5(6.2)	339	18.8(6.1)	690	19.2(6.2)
	Overweight	75	18.0(5.9)	77	18.0(6.3)	152	18.0(6.1)
	Obese	29	17.5(6.6)	20	16.7(5.6)	49	17.1(6.2)
Participation	Normal Weight	526	15.9(5.4)	498	14.5(5.2)	1,024	15.2(5.4)
	Overweight	134	15.9(5.2)	127	14.1(5.3)	261	15.0(5.3)
	Obese	62	15.2(6.0)	44	12.1(5.6)	106	13.9(6.0)
Effects	Normal Weight	514	67.4(9.9)	490	66.8(9.4)	1,004	67.2(9.6)
	Overweight	132	67.7(9.4)	126	65.8(9.6)	258	66.8(9.5)
	Obese	63	63.4(11.3)	46	63.2(13.0)	109	63.3(12.0)
Importance	Normal Weight	504	61.8(7.8)	487	61.9(7.0)	991	61.9(7.4)
	Overweight	127	62.0(9.0)	124	61.9(7.4)	251	62.0(8.3)
	Obese	57	59.8(10.0)	45	59.4(11.9)	102	59.6(10.8)
Excuses	Normal Weight	514	11.8(6.3)	486	12.7(6.6)	1,000	12.2(6.5)
	Overweight	129	13.7(7.6)	123	13.9(7.8)	252	13.8(7.7)
	Obese	61	14.3(8.2)	44	17.5(9.0)	105	15.7(8.7)

*Note.* Derived scores were calculated based on scale score range and number of items. Therefore maximum scores for each derived score were Encouragement (44), Participation (39); Effects (90); Importance (90); and Excuses (60).

*Other physical activity related perceptions.*

Overall males were more positive in their responses than females (Table 42). Compared to females, more males were able to *participate in their favourite activity* ( $p=.021$ ), did not *exercise hard to control weight* ( $p<.001$ ), were not good at *gymnastics, dancing or sport* ( $p=.042$ ), and were not *clumsy or uncoordinated* ( $p<.001$ ).



Table 42

*Gender Differences for Other Physical Activity Related Survey Items at age 14 Years*

Variable	Total	Male	Female	Gender Difference	
	n	n	n	$\chi^2$	<i>p</i>
<b>Favourite Activity</b>					
None	3	1	2	.580	.748
Yes	1,380	716	664		
Don't know	13	6	7		
<b>Able to do Favourite Activity</b>					
No	135	67	68	<b>7.729</b>	<b>.021</b>
Yes	1,098	587	511		
Don't know	162	68	94		
<b>Exercise hard to control weight</b>					
Not at all	629	364	265	<b>19.426</b>	<b>&lt;.001</b>
Some of the time	551	250	301		
A lot of the time	171	83	88		
Most of the time	41	23	18		
<b>Teased about weight or shape</b>					
No	1,118	591	527	2.249	.134
Yes	276	132	144		
<b>Good at gymnastics, dancing or sport</b>					
No	198	118	80	<b>6.356</b>	<b>.042</b>
Yes	1,034	526	508		
Don't know	162	77	85		
<b>Clumsy uncoordinated</b>					
No	1,143	628	515	<b>26.388</b>	<b>&lt;.001</b>
Yes	160	59	101		
Don't know	91	34	57		

Note. **Bolded** indicate significant group differences  $p < .05$ .

More obese reported being unable to *participate in their favourite activity* (OB 17.6% OW 8.8% NW 9.1%;  $p=.004$ )<sup>14</sup>; more obese and overweight *exercised to control weight* (not at all OB 16.7%, OW 23.9% NW 53.6%;  $p<.001$ ) (Figure 11), and *were teased about their weight or shape* (OB 60.7% OW 36.0 NW 11.4%,  $p<.001$ ) (Figure 12); while fewer obese reported being *good at gymnastics, dancing or sport* (OB 51.9% OW 72.0% NW 77.1%;  $p<.001$ ) (Table 43).

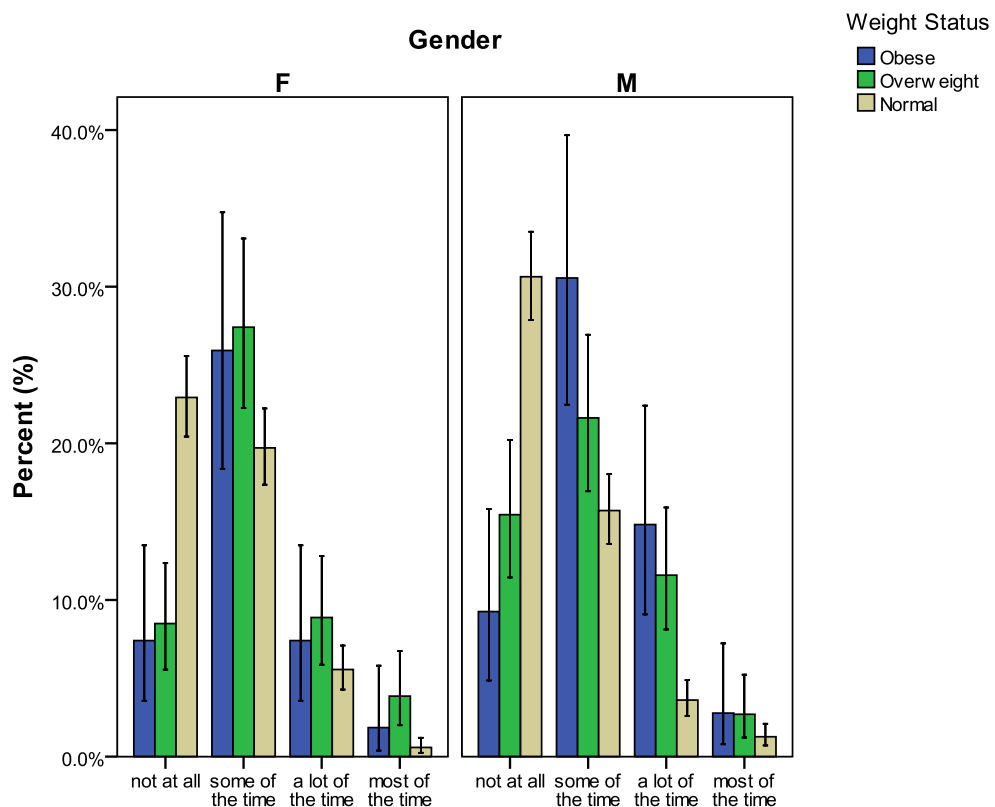


Figure 11. Comparison of weight status groups (determined at 14 years) and exercised hard to control weight, for females (F) and males (M) with 95% CI intervals ( I ).

<sup>14</sup> Note. OB=obese, OW=overweight, NW=normal weight.

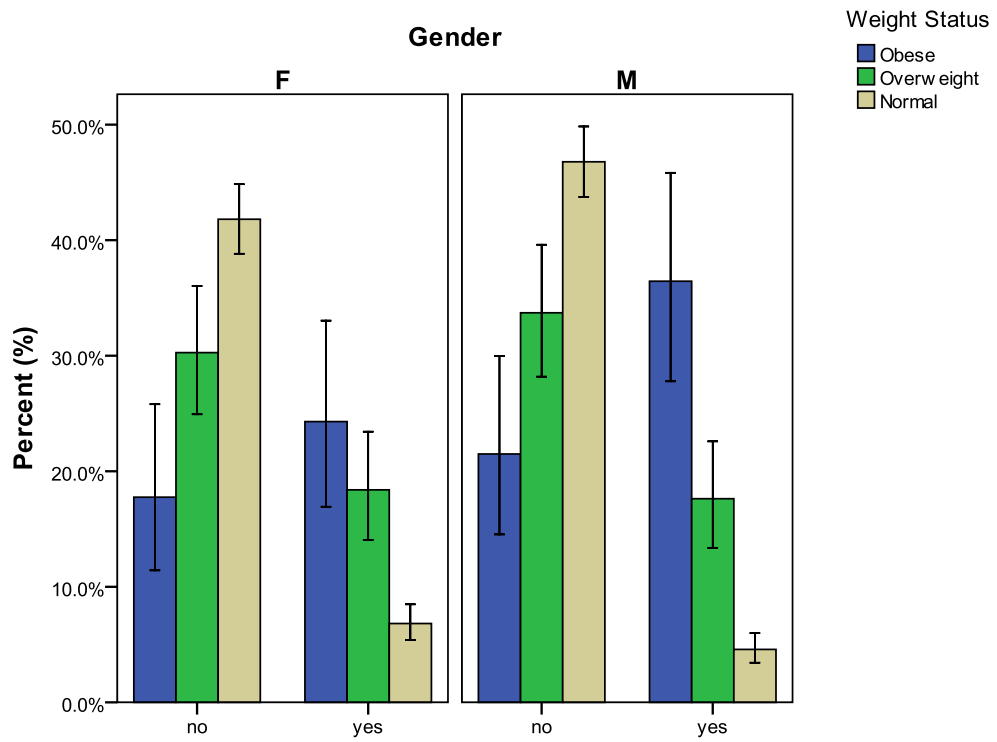


Figure 12. Comparison of weight status groups (determined at 14 years) and teased about weight or shape, for females (F) and males (M) with 95% CI intervals ( I ).

Table 43

*Weight Status Group Differences for Other Physical Activity Related Survey Items at age 14 Years*

Survey year	Total	Obese	Overweight	Normal weight	$\chi^2$	<i>p</i>
Favourite Activity					3.227	.521
None	3	1	0	2		
Yes	1,380	106	259	1,015		
Don't know	13	1	2	10		
Able to do favourite activity					<b>15.527</b>	<b>.004</b>
No	135	19	23	93		
Yes	1,098	73	198	827		
Don't know	162	16	40	106		
Exercise hard to control weight					<b>126.939</b>	<b>&lt;.001</b>
Not at all	629	18	62	549		
Some of the time	551	61	127	363		
A lot of the time	171	24	53	94		
Most of the time	41	5	17	19		
Teased about weight or shape					<b>201.755</b>	<b>&lt;.001</b>
No	1,118	42	167	909		
Yes	276	65	94	117		
Good at gymnastics, dancing or sport					<b>37.879</b>	<b>&lt;.001</b>
No	198	34	38	126		
Yes	1,034	56	188	790		
Don't know	162	18	35	109		
Clumsy uncoordinated					3.488	.480
No	1,143	86	208	849		
Yes	160	14	30	116		
Don't know	91	8	23	60		

Note. **Bolded** indicate significant group differences  $p < .05$ .

### ***Sedentary behaviour.***

Screen time was a proxy for sedentary behaviour and represented at ages 6 and 8 years by television viewing time, at age 10 years by television and computer time, and at age 14 years by separate items television time and computer time, which were combined to create a total screen time variable. Males and females were similar in the television viewing time at ages 6, 8 and 10 years. However, by age 14 years, males were watching more television and spending more time on the computer than girls (Table 44).

Table 44

#### *Gender Differences for Screen Time at ages 6, 8, 10 and 14 years*

Screen Time	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
6 Years ( <i>n</i> =1,303) TV time per day					
0 Never	11	3	8	6.405	.269
1 <3 hrs per wk	108	51	57		
2 up to 1 hr per day	330	165	165		
3 1-2 hrs per day	533	279	254		
4 3-4 hrs per day	270	152	118		
5 >3 hrs per day	51	28	23		
8 Years ( <i>n</i> =1,309) TV time per day					
0 Never	10	4	6	7.156	.209
1 <3 hrs per wk	100	50	50		
2 up to 1 hr per day	359	168	191		
3 1-2 hrs per day	549	304	245		
4 3-4 hrs per day	246	126	120		
5 >3 hrs per day	45	24	21		
10 Years ( <i>n</i> =1,312) TV and computer time per day					
0 Never	3	0	3	7.233	.204
1 <3 hrs per wk	86	41	45		
2 up to 1 hr per day	285	144	141		
3 1-2 hrs per day	538	288	250		
4 3-4 hrs per day	316	162	154		
5 >3 hrs per day	84	51	33		

Screen Time	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference	
				$\chi^2$	<i>p</i>
14 Years ( <i>n</i> =1,398) TV and video time per day					
0 Never	21	9	12	7.688	.104
1 up to 1 hr per day	213	94	119		
2 1-2 hrs per day	459	239	220		
3 3-4 hrs per day	510	280	230		
4 >3 hrs per day	195	102	93		
14 Years ( <i>n</i> =1,397) TV and video time per week					
0 Never	14	8	6	<b>11.723</b>	<b>.020</b>
1 up to 7 hrs/wk	357	159	198		
2 7-14 hrs /wk	483	253	230		
3 14-21 hrs /wk	420	231	189		
4 >21 hrs /wk	123	72	51		
14 Years ( <i>n</i> =1,398) Computer time per day					
0 Never	143	43	100	<b>63.053</b>	<b>&lt;.001</b>
1 up to 1 hr per day	511	230	281		
2 1-2 hrs per day	419	251	168		
3 3-4 hrs per day	206	119	87		
4 >3 hrs per day	119	81	38		
14 Years ( <i>n</i> =1,398) Computer time per week					
0 Never	142	40	102	<b>62.467</b>	<b>&lt;.001</b>
1 up to 7 hrs/wk	682	327	355		
2 7-14 hrs /wk	333	200	133		
3 14-21 hrs /wk	163	104	59		
4 >21 hrs /wk	78	53	25		
14 Years ( <i>n</i> =1,397) Screen time per day					
0 Never	6	1	5	<b>41.966</b>	<b>&lt;.001</b>
1 up to 1 hr per day	27	12	15		
2 1-2 hrs per day	171	63	108		
3 3-4 hrs per day	275	120	155		
4 4-5 hrs per day	352	187	165		
5 > 5 hrs per day	566	340	226		

Screen Time	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference $\chi^2$	<i>p</i>
14 Years ( <i>n</i> =1,397) Screen time per week					
0 Never	3	1	2	<b>45.306</b>	<b>&lt;.001</b>
1 up to 7 hrs/wk	54	17	37		
2 7-14 hrs /wk	265	117	148		
3 14-21 hrs /wk	334	147	187		
4 21-28 hrs /wk	342	190	152		
5 > 28 hrs/wk	399	251	148		

Note. hrs/wk = hours per week. **Bolded** indicate significant group differences  $p < .05$ .

Weight status group differences were found for television viewing time, but not computer use (Table 45). A higher percentage of obese participants watched more than 2 hours spent watching television at 6 years (OB 47.0% OW 26.3% NW 21.9%)<sup>15</sup>; 8 years (OB 41.6% OW 20.7% NW 20.7%); and 10 years (OB 49.0% OW 30.2% NW 28.6%). At 14 years, almost half of each weight status group were watching more than 2 hours of television a day. A significant weight status group difference remained, although similar proportions of the overweight and obese groups were now watching more than 2 hours of television a day (OB 55.1% OW 56.0% NW 48.5%).

<sup>15</sup> Note. OB=obese, OW=overweight, NW=normal weight.

Table 45

*Weight Status Group Differences for Screen Time at ages 6, 8, 10 and 14 years*

Survey year	Total	Obese	overweight	Normal weight	$\chi^2$	$p$
6 Years TV time per day					<b>42.264</b>	<b>&lt;.001</b>
0 Never	11	0	2	9		
<3 hrs per wk	108	6	20	82		
up to 1 hr per day	330	16	58	256		
1-2 hrs per day	533	31	96	406		
3-4 hrs per day	270	34	55	181		
>4 hrs per day	51	13	8	30		
8 Years TV time per day					<b>38.502</b>	<b>&lt;.001</b>
Never	10	0	3	7		
<3 hrs per wk	100	6	15	79		
up to 1 hr per day	359	9	73	277		
1-2 hrs per day	549	45	104	400		
3-4 hrs per day	246	31	44	171		
5 >4 hrs per day	45	10	7	28		
10 Years TV and computer time per day					<b>26.930</b>	<b>.003</b>
Never	3	0	1	2		
<3 hrs per wk	86	6	16	64		
up to 1 hr per day	285	13	48	224		
1-2 hrs per day	538	33	104	401		
3-4 hrs per day	316	34	58	224		
>4 hrs per day	84	16	15	53		
14 Years TV time per day					<b>16.669</b>	<b>.034</b>
Never	21	2	2	17		
up to 1 hr per day	213	11	36	166		
1-2 hrs per day	459	36	77	346		
3-4 hrs per day	510	34	109	367		
>4 hrs per day	195	26	37	132		



Survey year	Total	Obese	overweight	Normal weight	$\chi^2$	<i>p</i>
14 Years TV time per week					<b>24.968</b>	<b>.002</b>
Never	14	1	3	10		
up to 7 hrs/wk	357	19	65	273		
7-14 hrs /wk	483	35	86	362		
14-21 hrs /wk	420	31	83	306		
>21 hrs /wk	123	23	23	77		
14 Years Computer time per day					12.707	.122
Never	143	16	23	104		
up to 1 hr per day	511	30	99	382		
1-2 hrs per day	419	27	76	316		
3-4 hrs per day	206	22	43	141		
>4 hrs per day	119	14	20	85		
14 Years Computer time per week					10.031	.263
Never	142	14	26	102		
up to 7 hrs/wk	682	46	131	505		
7-14 hrs /wk	333	22	55	256		
14-21 hrs /wk	163	16	34	113		
>21 hrs /wk	78	11	15	52		
14 Years Screen time per day					7.693	.659
Never	6	1	0	5		
up to 1 hr per day	27	2	3	22		
1-2 hrs per day	171	12	28	131		
3-4 hrs per day	275	17	51	207		
4-5 hrs per day	352	24	70	258		
> 5 hrs per day	566	53	109	404		

Survey year	Total	Obese	overweight	Normal weight	$\chi^2$	$p$
14 Years Screen time per week					<b>18.616</b>	<b>.045</b>
Never	3	1	0	2		
up to 7 hrs/wk	54	2	11	41		
7-14 hrs /wk	265	18	56	191		
14-21 hrs /wk	334	18	50	266		
21-28 hrs /wk	342	27	62	253		
> 28 hrs/wk	399	43	81	275		

Note. hrs=hours, hrs/wk= hours per week. **Bolded** indicate significant group differences  $p < .05$ .

### ***Self concept.***

Self concept was examined using all nine domains of the Harter Self-Perception Profile for Adolescents (SPPA). Males had higher scores for Athletic Competence, Physical Appearance, Global Self Worth, and Romantic Appeal domains, while females had higher scores for Behavioural Conduct and Close Friendship domains. Males and females had similar scores in Job Competence, Scholastic Competence and Social Acceptance domains (Table 46).

Table 46

*Gender Differences for Self Concept (SPPA) at age 14 Years*

SPAA Domain	Total		Male		Female		Gender Difference	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>P</i>
Athletic	1,368	2.8(0.7)	707	3.0(0.6)	661	2.7(0.7)	<b>8.488</b>	<b>&lt;.001</b>
Appearance	1,370	2.7(0.6)	709	2.8(0.6)	661	2.5(0.7)	<b>9.526</b>	<b>&lt;.001</b>
Behaviour	1,370	2.9(0.5)	709	2.8(0.5)	661	3.0(0.5)	<b>-5.288</b>	<b>&lt;.001</b>
Friend	1,370	3.3(0.6)	709	3.2(0.6)	661	3.5(0.6)	<b>-8.922</b>	<b>&lt;.001</b>
GSW	1,370	3.1(0.5)	709	3.2(0.5)	661	3.1(0.6)	<b>3.830</b>	<b>&lt;.001</b>
Job	1,368	2.8(0.6)	707	2.8(0.5)	661	2.9(0.6)	-1.695	.090
Romantic	1,272	2.7(0.5)	647	2.7(0.5)	625	2.6(0.5)	<b>3.503</b>	<b>&lt;.001</b>
Scholastic	1,370	2.9(0.6)	709	2.9(0.6)	661	2.8(0.6)	1.111	.267
Social	1,369	3.2(0.5)	708	3.2(0.5)	661	3.2(0.5)	-1.311	.190

*Note.* Athletic = athletic competence, Appearance= physical appearance, Behaviour = behavioural conduct, Friend = close friendship, GSW=Global Self Worth, Job = job competence, Romantic = romantic appeal, Scholastic = scholastic competence, Social = social acceptance. Score range 1-4.

**Bolded** indicate significant group differences  $p < .05$ .

In general, weight status groups differed in scores for Athletic Competence, Global Self Worth and Physical Appearance domains (Table 47) and in all cases the obese group scored lower (Table 48). Post hoc analysis indicated these differences were mainly between the normal weight group and overweight and obese groups, and consistent for males and females. Significant weight status group differences were found for Social Acceptance, Romantic Appeal and Close Friendship domains. However gender analysis identified that this was prevalent for females only, with differences between the normal weight group and overweight and obese groups. No weight status differences were found for Job Competence, Scholastic Competence, or Behavioural Conduct domains.

Table 47

*Weight Status Group Differences for Self Concept (SPPA) at age 14 Years*

SPPA Domain	Gender	ANOVA		Post hoc Tests p-value		
		F	p	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Athletic	Male	<b>12.743</b>	<b>&lt;.001</b>	<b>.014</b>	<b>&lt;.001</b>	.061
	Female	<b>12.837</b>	<b>&lt;.001</b>	<b>.002</b>	<b>&lt;.001</b>	.132
	Total	<b>22.622</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>.021</b>
Appearance	Male	<b>52.636</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>.001</b>
	Female	<b>28.993</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	.337
	Total	<b>68.065</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>.001</b>
Behaviour	Male	1.414	.244	.938	.470	.370
	Female	1.164	.313	.606	.352	.938
	Total	2.152	.117	.955	.112	.310
Friend	Male	.552	.576	1.000	.671	.723
	Female	<b>8.881</b>	<b>&lt;.001</b>	<b>.018</b>	<b>.006</b>	.621
	Total	<b>6.244</b>	<b>.002</b>	.142	<b>.005</b>	.332
GSW	Male	<b>4.173</b>	<b>.016</b>	.940	<b>.009</b>	.076
	Female	<b>13.446</b>	<b>&lt;.001</b>	<b>.001</b>	<b>.003</b>	.522
	Total	<b>14.476</b>	<b>&lt;.001</b>	<b>.005</b>	<b>&lt;.001</b>	.103
Job	Male	1.020	.361	.484	.991	.685
	Female	.118	.889	.978	.979	1.000
	Total	.760	.468	.578	1.000	.848
Romantic	Male	1.711	.181	.900	.156	.509
	Female	<b>3.698</b>	<b>.025</b>	.662	<b>.037</b>	.219
	Total	<b>4.869</b>	<b>.008</b>	.556	<b>.007</b>	.129
Scholastic	Male	1.360	.257	.499	.621	.993
	Female	1.104	.332	.387	1.000	.766
	Total	2.073	.126	.161	.786	.977
Social	Male	1.264	.283	.606	.575	.991
	Female	<b>10.545</b>	<b>&lt;.001</b>	<b>.035</b>	<b>.002</b>	.177
	Total	<b>9.143</b>	<b>&lt;.001</b>	<b>.032</b>	<b>.003</b>	.387

Athletic = athletic competence, Appearance= physical appearance, Behaviour = behavioural conduct, Friend = close friend, GSW=Global Self Worth, Job = job competence, Scholastic = scholastic competence, Social = peer social acceptance. **Bolded** indicate significant group differences  $p<.05$ .

Table 48

*Descriptive Statistics for Self Concept (SPPA) at age 14 Years*

SPPA Domain	BMI Weight Status determined at 14 Years	Total		Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Athletic	Normal Weight	1,007	2.9(0.7)	515	3.0(0.6)	492	2.7(0.7)
	Overweight	259	2.7(0.7)	134	2.9(0.6)	125	2.5(0.6)
	Obese	102	2.5(0.6)	58	2.7(0.5)	44	2.3(0.6)
Appearance	Normal Weight	1,008	2.8(0.6)	516	2.9(0.5)	492	2.6(0.7)
	Overweight	259	2.4(0.6)	134	2.6(0.6)	125	2.2(0.6)
	Obese	103	2.2(0.7)	59	2.3(0.6)	44	2.0(0.7)
Behaviour	Normal Weight	1,008	2.9(0.5)	516	2.8(0.5)	492	3.0(0.6)
	Overweight	259	2.9(0.5)	134	2.8(0.5)	125	2.9(0.5)
	Obese	103	2.8(0.5)	59	2.7(0.6)	44	2.9(0.4)
Friend	Normal Weight	1,008	3.3(0.6)	516	3.2(0.6)	492	3.5(0.6)
	Overweight	259	3.3(0.6)	134	3.2(0.6)	125	3.3(0.7)
	Obese	103	3.2(0.6)	59	3.1(0.6)	44	3.2(0.6)
GSW	Normal Weight	1,008	3.2(0.5)	516	3.2(0.4)	492	3.1(0.6)
	Overweight	259	3.1(0.5)	134	3.2(0.5)	125	2.9(0.6)
	Obese	103	2.9(0.5)	59	3.0(0.4)	44	2.8(0.6)
Job	Normal Weight	1,007	2.8(0.5)	515	2.8(0.5)	492	2.9(0.6)
	Overweight	258	2.9(0.6)	133	2.9(0.6)	125	2.9(0.6)
	Obese	103	2.8(0.6)	59	2.8(0.6)	44	2.9(0.6)
Romantic	Normal Weight	943	2.7(0.5)	479	2.7(0.5)	464	2.6(0.5)
	Overweight	238	2.6(0.5)	118	2.7(0.5)	120	2.6(0.5)
	Obese	91	2.5(0.5)	50	2.6(0.5)	41	2.4(0.5)
Scholastic	Normal Weight	1,008	2.9(0.6)	516	2.9(0.6)	492	2.9(0.6)
	Overweight	259	2.8(0.6)	134	2.8(0.6)	125	2.8(0.6)
	Obese	103	2.8(0.6)	59	2.8(0.7)	44	2.9(0.6)
Social	Normal Weight	1,007	3.2(0.5)	515	3.2(0.5)	492	3.2(0.5)
	Overweight	259	3.1(0.6)	134	3.1(0.6)	125	3.1(0.6)
	Obese	103	3.0(0.6)	59	3.1(0.5)	44	2.9(0.6)

*Note.* Athletic = athletic competence, Appearance= physical appearance, Behaviour = behavioural conduct, Friend = close friendship, GSW=Global Self Worth, Job = job competence, Romantic = romantic appeal, Scholastic = scholastic competence, Social = social acceptance. Score range 1-4.

**Parent reported academic, social and behavioural progress.**

Gender differences were found for parent reported academic performance, learning skills progress, and behaviour progress, but not social progress, with a higher percentage of females rated as *excellent* compared to males (Table 49).

Table 49

*Gender Differences for Parent Reported Academic, Social and Behavioural Progress at age 10 Years*

Variable	Total	Male	Female	Gender Difference	
	<i>n</i>	<i>n</i>	<i>n</i>	$\chi^2$	<i>p</i>
<b>Academic Performance</b>					
Poor	10	7	3	<b>44.396</b>	<b>&lt;.001</b>
Below Average	100	68	32		
Average	418	251	167		
Very Good	521	260	261		
Excellent	264	100	164		
<b>Learning Skills Progress</b>					
Very Dissatisfied	9	7	2	<b>20.089</b>	<b>&lt;.001</b>
Dissatisfied	73	43	30		
Neither	67	44	23		
Satisfied	666	367	299		
Very Satisfied	496	225	271		
<b>Social Progress</b>					
Very Dissatisfied	5	2	3	7.348	.119
Dissatisfied	54	32	22		
Neither	71	45	26		
Satisfied	590	315	275		
Very Satisfied	593	292	301		
<b>Behaviour Progress</b>					
Very Dissatisfied	6	5	1	<b>62.930</b>	<b>&lt;.001</b>
Dissatisfied	48	42	6		
Neither	53	39	14		
Satisfied	560	321	239		
Very Satisfied	646	279	367		

Note. **Bolded** indicate significant group differences  $p < .05$ .

Weight status group differences were found for social and behaviour progress (Table 50). Fewer parents were *very satisfied* with social progress if their child was obese or overweight (OB 34.3%, OW 36.2%, NW 48.6%). This was similar for behaviour progress, with fewer parents *very satisfied* if their child was obese or overweight (OB 40.2%, OW 44.9%, NW 51.2%).

Table 50

*Weight Status Group Differences for Parent Reported Academic, Social and Behavioural Progress at 10 Years*

Survey year	Total	Obese	overweight	Normal weight	$\chi^2$	<i>p</i>
Academic Performance					12.226	.141
Poor	10	2	3	5		
Below Average	100	13	22	65		
Average	418	34	82	302		
Very Good	521	36	95	390		
Excellent	264	17	41	206		
Learning Skills Progress					10.376	.240
Very Dissatisfied	9	2	3	4		
Dissatisfied	73	8	15	50		
Neither	67	5	16	46		
Satisfied	666	55	125	486		
Very Satisfied	496	32	82	382		
Social Progress					<b>34.510</b>	<b>&lt;.001</b>
Very Dissatisfied	5	0	4	1		
Dissatisfied	54	9	9	36		
Neither	71	4	16	51		
Satisfied	590	54	126	410		
Very Satisfied	593	35	88	470		
Behaviour Progress					<b>28.071</b>	<b>&lt;.001</b>
Very Dissatisfied	6	2	3	1		
Dissatisfied	48	7	8	33		
Neither	53	9	5	39		
Satisfied	560	43	118	399		
Very Satisfied	646	41	109	496		

## Environmental factors.

Environmental factors included socioeconomic status, parental influences, and proxies for the built environment.

### *Socioeconomic status (SES).*

Socioeconomic status influences on obesity were assessed at both a community (SEIFA and ethnicity) and individual (family income, employment, and life stress) level.

### *Socioeconomic Index for Areas (SEIFA).*

SEIFA advantage/disadvantage was not different between males ( $n=650$ ) and females ( $n=600$ ) ( $p=.935$ ), with a mean index of 1,030 ( $SD=74$ ). Weight status group differences were found ( $F=12.173$   $p<.001$ ) between the normal weight and overweight ( $p=.002$ ) and normal weight and obese ( $p<.001$ ), but not the overweight and obese ( $p=.318$ ) groups. These differences were consistent when analysed separately for males and females (Technical Report Table 49). Weight status group mean index scores for the total sample, males and females are summarised in Table 51.

Table 51

#### *Descriptive Statistics for SEIFA Advantage/Disadvantage at age 14 Years*

BMI Weight Status determined at 14 years	Total		Male		Female	
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Normal Weight	916	1,036.1(74.2)	478	1,035.9(76.0)	438	1,036.5(72.3)
Overweight	234	1,017.0(70.5)	115	1,018.7(71.8)	119	1,017.3(69.5)
Obese	100	1,005.2(67.3)	57	1,005.0(61.3)	43	1,005.6(75.2)



### *Parent ethnicity.*

Ethnic groups, other than Caucasian, accounted for only 9% of the sample. Hence ethnicity investigations were limited to a comparison of Caucasian to non-Caucasian (other). There were no gender differences for mother's or father's ethnicity, nor weight status group differences for mother's ethnicity. There was a significant weight status group difference for father's ethnicity ( $\chi^2=7.378$   $p=.025$ ) with the non-Caucasian group less obese (3% versus 8%), less overweight (14% versus 19%) and more normal weight (83% versus 73%) compared to the Caucasian group. (Refer to Technical Report Tables 50 and 51 for full details.)

### *Income.*

Due to inflation, income brackets (hence cut-points) used in each follow-up year varied. Different category groupings were analysed, however results were similar. For simplicity and replication with other Raine Study publications (Kozyrskyj et al., 2009) the two category income variable is reported in full here, while the more detailed three, four and seven category income variable results are presented in the Technical Report Tables 52-54.

More females came from a low income family than males at follow-up ages 2 years (46.2% versus 37.7% respectively  $p=.004$ ) and 3 years (41.9% versus 34.8%  $p=.009$ ), with no gender differences at birth nor for the other follow-ups (Table 52). Except for follow-up ages 2 and 3 years, all follow-up years had a significant weight status group difference. The more obese adolescents from came low income families and this outcome was particularly strong from six years (Table 53).

Table 52

*Gender Differences for Family Income at each Follow-up*

Follow-up	Total		Male		Female		Gender Difference	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	$\chi^2$	<i>p</i>
<b>BIRTH (<i>n</i>=1,343)</b>								
Low <\$24,000	496	36.9	258	36.7	238	37.2	0.034	.853
Not Low	847	63.1	445	63.3	402	62.8		
<b>1 Year (<i>n</i>=1,319)</b>								
Low <\$27,000	476	36.1	230	33.7	246	38.6	3.421	.064
Not Low	843	63.9	452	66.3	391	61.4		
<b>2 Years (<i>n</i>=1,092)</b>								
Low <\$27,000	457	41.8	209	37.7	248	46.2	<b>8.150</b>	<b>.004</b>
Not Low	635	58.2	346	62.3	289	53.8		
<b>3 Years (<i>n</i>=1,265)</b>								
Low <\$27,000	484	38.3	227	34.8	257	41.9	<b>6.760</b>	<b>.009</b>
Not Low	781	61.7	425	65.2	356	58.1		
<b>6 Years (1,282)</b>								
Low <\$27,000	339	26.4	177	26.6	162	26.3	0.021	.884
Not Low	943	73.6	488	73.4	455	73.7		
<b>8 Years (<i>n</i>=1,279)</b>								
Low <\$30,000	343	26.8	173	26.1	170	27.6	0.368	.544
Not Low	936	73.2	490	73.9	446	72.4		
<b>10 Years (<i>n</i>=1,290)</b>								
Low <\$30,000	341	26.4	169	25.0	172	28.0	1.421	.233
Not Low	949	73.6	506	75.0	443	72.0		
<b>14 Years (<i>n</i>=1,338)</b>								
Low <\$30,000	258	19.3	128	18.6	130	20.1	0.490	.484
Not Low	1,080	80.7	562	81.4	518	79.9		

Note. **Bolded** indicate significant group differences  $p < .05$ .

Table 53

*Weight Status Group Differences for Family Income at each Follow-up*

Follow-up	Total	Obese	Overweight	Normal weight	$\chi^2$	<i>p</i>
Birth					<b>6.872</b>	<b>.032</b>
Low <\$24,000	496	46	102	348		
Not Low	847	55	145	647		
1 Year					5.561	.062
Low <\$27,000	476	47	94	335		
Not Low	843	56	152	635		
2 Years					2.960	.228
Low <\$27,000	457	39	90	328		
Not Low	635	42	109	484		
3 Years					<b>7.989</b>	<b>.018</b>
Low <\$27,000	484	339	101	44		
Not Low	781	600	134	47		
6 Years					<b>16.871</b>	<b>&lt;.001</b>
Low <\$27,000	339	41	71	227		
Not Low	943	56	167	720		
8 Years					<b>13.250</b>	<b>.001</b>
Low <\$30,000	343	40	70	233		
Not Low	936	56	173	707		
10 Years					<b>15.412</b>	<b>&lt;.001</b>
Low <\$30,000	341	228	73	40		
Not Low	949	726	164	59		
14 Years					<b>19.948</b>	<b>&lt;.001</b>
Low <\$30,000	258	33	61	164		
Not Low	1,080	68	191	821		

Note. **Bolded** indicate significant group differences  $p < .05$ .

*Parent education and father's occupation.*

There were no gender differences for mother's education ( $\chi^2=5.631$   $p=.344$ ), father's education ( $\chi^2=4.263$   $p=.512$ ), nor father's occupation ( $\chi^2=7.688$   $p=.361$ ) (Technical Report Table 55). Weight status group differences and descriptive statistics are detailed in Table 54.

Table 54

*Weight Status Group Differences for Mother's Education, Father's Education and Father's Occupation*

Variable	Total	Obese	overweight	Normal weight	$\chi^2$	$p$
Mother's Education ( $n=1,403$ )					<b>21.751</b>	<b>.016</b>
Trade/apprenticeship	115	14	20	81		
Professional non-degree	154	9	30	115		
College diploma/degree	249	10	38	201		
University degree	164	8	26	130		
Other	67	4	13	50		
None	654	64	136	454		
Father's Education ( $n=1,202$ )					<b>21.191</b>	<b>.020</b>
Trade/apprenticeship	327	30	52	245		
Professional non-degree	45	2	11	32		
College diploma/degree	171	9	31	131		
University degree	224	9	32	183		
Other	54	3	8	43		
None	381	38	84	259		
Father's Occupation ( $n=1,403$ )					<b>37.344</b>	<b>.001</b>
Managerial	67	3	12	52		
Professional	242	7	37	198		
Para-professional	78	4	19	55		
Trade	303	20	45	238		
Clerical	86	6	19	61		
Sales	104	15	16	73		
Plant Operator	88	11	23	54		
Labourer	142	17	29	96		

Note. **Bolded** indicate significant group differences  $p<.05$ .

Educational attainment for both mothers ( $p=.016$ ) and fathers ( $p=.020$ ) were related to weight status. Parents with low educational attainment had a higher proportion of obese children. Father's occupation was also related to weight status ( $p=.001$ ). Those involved in sales, plant operations and labouring had a higher proportion of obese children.

### *Employment.*

The impact of employment was investigated for both, either or no parent working; family minimum work hours; and mother's working hours; at birth and each follow-up. In general, results for employment variables did not differ between weight status groups or gender. A summary follows, with detailed tables provided in the Technical Report Tables 56 -65.

There were no significant differences for gender across all follow-ups for the family employment setting. Only at age 2 years was a significant weight status group difference ( $p=.003$ ) found. At that age slightly more obese (13.0%) and overweight (13.5%) children had neither parent employed compared to normal weight (10.3%); with slightly more normal weight (37.6%) children having both parents employed compared to obese (26.0%) and overweight (28.1%).

Minimum work hours in the family were not different for males and females, or weight status, at birth and every follow-up. Whether a mother worked or not, was not different for males and females at birth and across all follow-up years. Weight status group differences were significant at birth ( $\chi^2=6.138$   $p=.046$ ) and at age 2 years ( $\chi^2=8.445$ ,  $p=.015$ ), 6 years ( $\chi^2=6.673$   $p=.036$ ), 10 years ( $\chi^2=9.223$   $p=.010$ ) and 14 years ( $\chi^2=7.652$   $p=.022$ ). In each significant follow-up, more obese and overweight children had mothers who did not work.

The number of hours a mother worked was not significantly different for males or females at birth and across all follow-ups. Weight status group differences were not found except at age 2 years for females only ( $F=3.149$   $p=.044$ ), with no group

differences; and overall at age 10 years ( $F=3.197$   $p=.041$ ), with no group differences, and specifically for males ( $F=3.081$   $p=.047$ ) with a significant difference between normal weight and obese groups ( $p=.028$ ).

### *Family Life stress.*

Pregnancy problems, death of a relative, death of a friend, separation or divorce, marital problems, problems with children, job loss, partner job loss, money problems, residential move, and other stress were investigated individually at 18 and 34 weeks gestation and each follow-up. In addition, a dichotomous variable comprising less than three stress events or three or more stress events was investigated.

Generally, across individual family life stress variables, there were few significant gender differences. More females came from homes with separation or divorce at age 10 years, partner job loss at age 10 years, and money problems at age 6 and 14 years. More males came from homes with marital problems at age 1 year, other stress at age 2 years, problems with children at age 10 years, and job loss at age 14 years (Table 66 of the Technical Report). No significant gender differences were found for the dichotomous stress variable at 18 and 34 weeks gestation nor any follow-up (Technical Report Table 67).

Weight status group differences showed a consistent trend across follow-ups and stress variables, with six years an important time period (Technical Report Table 68). Weight status differences were found for pregnancy problems at age 6 years ( $\chi^2=10.066$   $p=.007$ , response yes: OB 7.1%, OW 12.7%, NW 6.5%) and age 14 years ( $\chi^2=9.096$   $p=.011$ , OB 4.8%, OW 2.4%, NW 1.1%); separation or divorce at age 6 years ( $\chi^2=9.948$   $p=.007$ , OB 13.0%, OW 13.9%, NW 7.8%); marital problems at age 6 years ( $\chi^2=10.003$   $p=.007$ , OB 23.2%, OW 19.2%, NW 13.5%); problems with children at age 6 years ( $\chi^2=21.406$   $p<.001$ , OB 38.9%, OW 22.6%, NW 18.9%) and age 14 years ( $\chi^2=12.092$   $p=.002$ , OB 52.9%, OW 42.5%, NW 36.6%); partner job loss at age 2 years ( $\chi^2=16.063$   $p<.001$ , OB 23.7%, OW 9.3%, NW 9.1%); money problems at

every time point ( $p < .01$ ) with higher reporting of *yes* for obese and overweight; and residential move at age 8 years ( $\chi^2 = 8.219$   $p = .016$ , OB 14.9%, OW 26.7%, NW 19.6%). Significant weight status group differences were found for three or more life stress events at 18 weeks gestation, and ages 1, 6 and 14 years (Table 55).

Table 55

*Weight Status Group Differences for Family Life Stress at 18 and 34 weeks Gestation, and each Follow-up*

Age at Follow-up	Number of Stress Items	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
18 Weeks	<3	1,227	88	224	915	<b>7.312</b>	<b>.026</b>
	$\geq 3$	176	21	39	116		
34 Weeks	<3	1,204	95	219	890	4.920	.085
	$\geq 3$	139	11	36	92		
1 Year	<3	763	53	131	579	<b>8.241</b>	<b>.016</b>
	$\geq 3$	225	25	50	150		
2 Years	<3	539	37	96	406	1.585	.453
	$\geq 3$	134	12	28	94		
3 Years	<3	643	41	122	480	2.529	.282
	$\geq 3$	145	13	21	111		
6 Years	<3	794	47	132	615	<b>13.957</b>	<b>.001</b>
	$\geq 3$	199	24	44	131		
8 Years	<3	891	65	154	672	5.463	.065
	$\geq 3$	179	18	41	120		
10 Years	<3	827	53	132	642	4.752	.093
	$\geq 3$	207	14	46	147		
14 Years	<3	1,007	68	187	752	<b>6.173</b>	<b>.046</b>
	$\geq 3$	334	36	65	233		

Note. **Bolded** indicate significant group differences  $p < .05$ .

### ***Parental influences.***

The influence of parents on their children's obesity was investigated by parent BMI, parenting scales, and maternal smoking during pregnancy.

#### ***Parent BMI.***

There were no gender differences for parent birth weight, parent BMI at child's birth, mother's BMI at follow-ups 6, 8 and 14 years, or father's BMI at follow-up 8 years (Table 56). Parent birth weight was not related to the child's 14 year weight status, however significant weight status group differences were found for all variables (Table 57). Typically, children who were categorised as obese, had parents with higher BMI (Table 58).

Table 56

*Gender Differences for Parent Birth Weight, Parent BMI at Child's Birth, Mother's BMI at Follow-ups 6, 8 and 14 Years, and Father's BMI at 8 Years*

	Male		Female		Gender difference			
	<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>t</i>	<i>p</i>		
Mother's Birth Weight (g)	1,082	3,204(589)	567	3,192(583)	515	3,217(596)	-0.697	.486
Father's Birth Weight (g)	771	3,499(628)	406	3,470(625)	365	3,532(631)	-1.368	.172
Mother's pre-pregnancy BMI	1,409	22.3(4.2)	731	22.3(4.3)	678	22.3(4.2)	0.083	.934
Birth Year	1,172	24.5(3.4)	598	24.4(3.4)	574	24.6(3.4)	-0.845	.398
Father's BMI								
Mother's BMI 6 Years	284	26.2(6.0)	141	26.5(6.7)	143	25.9(5.3)	0.865	.388
Mother's BMI 8 Years	1,164	25.9(5.6)	598	25.9(5.5)	566	25.9(5.8)	-0.207	.836
Father's BMI 8 Years	389	26.8(4.4)	206	26.5(4.0)	183	27.2(4.7)	-1.601	.110
Mother's BMI 14 Years	1,079	26.4(6.1)	548	26.4(5.7)	531	26.4(6.6)	0.043	.966



Table 57

*Weight Status Group Differences for Parent Birth Weight, Parent BMI at Child's Birth, Mother's BMI at Follow-ups 6, 8 and 14 Years, and Father's BMI at 8 Years*

Variable	Gender	ANOVA		Post hoc Tests p-value		
		F	p	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Mother's Birth Weight	Male	0.667	.514	.744	.728	.988
	Female	0.213	.809	.877	.998	.957
	Total	0.581	.559	.619	.943	.999
Father's Birth Weight	Male	0.811	.445	.765	.773	.973
	Female	0.513	.599	.974	.812	.741
	Total	0.914	.401	.990	.589	.755
Mother's Pre-Pregnancy BMI	Male	<b>44.926</b>	<b>&lt;.001</b>	<b>.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
	Female	<b>36.167</b>	<b>&lt;.001</b>	<b>.021</b>	<b>&lt;.001</b>	<b>.002</b>
	Total	<b>80.919</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
Father's BMI at child's Birth	Male	<b>11.468</b>	<b>&lt;.001</b>	.282	<b>&lt;.001</b>	<b>.019</b>
	Female	<b>10.040</b>	<b>&lt;.001</b>	<b>.001</b>	.120	.991
	Total	<b>19.492</b>	<b>&lt;.001</b>	<b>.001</b>	<b>&lt;.001</b>	.126
Mother's BMI 6 Years	Male	<b>9.061</b>	<b>&lt;.001</b>	.925	<b>.004</b>	<b>.030</b>
	Female	<b>4.046</b>	<b>.020</b>	.111	.524	.835
	Total	<b>10.967</b>	<b>&lt;.001</b>	.134	<b>.002</b>	.190
Mother's BMI 8 Years	Male	<b>42.864</b>	<b>&lt;.001</b>	<b>.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
	Female	<b>46.582</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
	Total	<b>88.797</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
Father's BMI 8 Years	Male	<b>7.714</b>	<b>.001</b>	.975	<b>.012</b>	<b>.040</b>
	Female	<b>5.011</b>	<b>.008</b>	<b>.015</b>	.579	.995
	Total	<b>10.230</b>	<b>&lt;.001</b>	<b>.037</b>	<b>.011</b>	.278
Mother's BMI 14 Years	Male	<b>40.950</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>
	Female	<b>18.906</b>	<b>&lt;.001</b>	<b>.027</b>	<b>.003</b>	.064
	Total	<b>55.781</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>	<b>&lt;.001</b>

Note. **Bolded** indicate significant group differences  $p < .05$ .

Table 58

*Descriptive Statistics for Parent Birth Weight, Parent BMI at Child's Birth, Mother's BMI at Follow-ups 6, 8 and 14 Years, and Father's BMI at 8 Years*

Variable	BMI Weight Status determined at 14 Years	Male		Female	
		<i>n</i>	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>
Mother's Birth Weight (g)	Normal Weight	403	3174(600)	386	3,210(603)
	Overweight	116	3227(535)	94	3,252(515)
	Obese	48	3254(560)	35	3,192(717)
Father's Birth Weight (g)	Normal Weight	288	3446(612)	285	3,529(620)
	Overweight	84	3511(589)	52	3,492(656)
	Obese	34	3569(805)	28	3,639(708)
Mother's Pre-Pregnancy BMI	Normal Weight	531	21.6(3.9)	505	21.7(3.5)
	Overweight	137	23.1(4.3)	127	22.9(4.2)
	Obese	63	26.5(4.9)	46	26.8(7.2)
Father's BMI at child's Birth	Normal Weight	440	24.0(3.3)	430	24.2(3.1)
	Overweight	108	24.7(3.5)	105	25.6(3.6)
	Obese	50	26.4(3.6)	39	25.8(4.7)
Mother's BMI Follow-up 6 Years	Normal Weight	104	25.5(6.2)	108	25.2(4.7)
	Overweight	19	26.4(6.0)	26	28.3(7.0)
	Obese	18	32.4(7.4)	9	26.9(3.8)
Mother's BMI Follow-up 8 Years	Normal Weight	439	24.9(4.8)	417	24.9(4.6)
	Overweight	106	27.1(5.7)	110	27.5(5.9)
	Obese	53	31.6(6.7)	39	32.9(9.3)
Father's BMI Follow-up 8 Years	Normal Weight	161	26.1(3.8)	140	26.6(4.5)
	Overweight	28	26.4(3.9)	35	29.0(4.4)
	Obese	17	30.0(4.8)	8	29.6(7.0)
Mother's BMI Follow-up 14 Years	Normal Weight	396	25.3(5.0)	400	25.6(5.8)
	Overweight	107	28.0(6.0)	95	27.5(6.5)
	Obese	45	32.2(6.2)	36	32.0(10.6)

### *Parenting styles.*

Parents completed a parenting scale at follow-up age 10 years, while at age 14 years adolescents rated their parent's parenting. Details of results are presented in Tables 69-73 in the Technical Report with a summary below.

At 10 years there were no gender differences in the parenting scale, nor differences between weight status groups. At 14 years, more females reported that their parents smiled at them ( $p<.001$ ), praised them ( $p=.012$ ), and told them they were appreciated ( $p=.013$ ). More males reported being nagged about little things ( $p=.022$ ).

A weight status group difference was found for the response that parents appreciated them ( $\chi^2=13.620$ ,  $p=.034$ ), with fewer obese individuals told they were appreciated *very often* (36.2%) compared to the overweight (40.6%) and normal weight (42.4%) groups. More parents of the obese group threatened or hit their child ( $\chi^2=14.829$ ,  $p=.022$ ; 7.6%), compared to overweight (4.7%) and normal weight (4.9%), however more overweight reported *sometimes* being threatened or hit (22.3%) compared to normal weight (16.7%) and obese (12.4%).

### *Maternal smoking during pregnancy.*

Smoking data were available for 1,092 individuals, with 366 (33.5%) mothers smoking during pregnancy. More female children (195 smokers versus 331 non-smokers) had mothers who smoked, compared to males (171 smokers versus 395 non-smokers) ( $\chi^2=5.758$   $p=.016$ ). Weight status group differences were also found ( $\chi^2=12.602$   $p=.002$ ), with the overweight group having the highest proportion of mothers who smoked during pregnancy (42.9%) compared to obese (39.5%) and normal weight (30.5%) groups. Gender separated weight status group differences were not significant for females ( $\chi^2=5.530$   $p=.063$ ), but significant for males ( $\chi^2=7.172$   $p=.028$ ).

### ***Built environment.***

The built environment variables included home swimming pool; outdoor play space; community park or playground; and school influences. They are associated indirectly with obesity via the opportunity to be physically active.

### ***Home swimming pool.***

The presence of a family home swimming pool at 1, 2 and 3 years, was not different between males and females (Table 59), nor related to weight status at 14 years (Table 60).

Table 59

#### *Gender Differences for Home Swimming Pool at ages 1, 2 and 3 Years*

Mean age at Follow-up	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference $\chi^2$	<i>p</i>
<b>1 Year (<i>n</i>=1,326)</b>					
No	1147	600	547	0.852	.356
Yes	179	87	92		
<b>2 Years (<i>n</i>=404)</b>					
No	357	168	189	0.626	.429
Yes	47	25	22		
<b>3 Years (<i>n</i>=441)</b>					
No	395	200	195	1.072	.300
Yes	46	27	19		

Table 60

*Weight Status Group Differences for Home Swimming Pool at ages 1, 2 and 3 Years*

Mean Age at Follow-up	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
1 Year					2.490	.288
No	1147	93	206	848		
Yes	179	11	40	128		
2 Years					2.579	.275
No	357	27	71	259		
Yes	47	3	5	39		
3 Years					5.562	.062
No	395	38	81	276		
Yes	46	0	8	38		

*Outdoor play space.*

Outdoor play space determined by the presence of a family garden, was reported at ages 1, 2 and 3 years. There was no difference between males and females (Table 61), or any weight status group differences (Table 62).

Table 61

*Gender Differences for Outdoor Play Space at ages 1, 2 and 3 Years*

Mean Age at Follow-up	Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference $\chi^2$	$p$
1 Year ( <i>n</i> =1, 328)					
No	55	29	26	0.023	.880
Yes	1,273	658	615		
2 Years ( <i>n</i> =405)					
No	18	8	10	0.078	.780
Yes	387	185	202		
3 Years ( <i>n</i> =439)					
No	13	7	6	0.025	.876
Yes	426	220	206		

Table 62

*Weight Status Group Differences for Outdoor Play Space at ages 1, 2 and 3 Years*

Mean Age at Follow-up		Total	Obese	Overweight	Normal weight	$\chi^2$	<i>p</i>
1 Year							
	No	55	8	11	36	3.877	.144
	Yes	1,273	96	236	941		
2 Years							
	No	18	2	4	12	0.600	.741
	Yes	387	28	72	287		
3 Years							
	No	13	0	5	8	3.494	.174
	Yes	426	37	84	305		

*Live near a park or playground.*

There were no gender differences in the family's home proximity to a park or playground (Table 63). Weight status group differences were only significant at one year, with more obese children (8.2%) living near a park or playground, compared to not living near a park or playground (3.5%), and similarly for the overweight group (Table 64).

Table 63

*Gender Differences for Live Near a Park of Playground at ages 1, 2 and 3 Years*

Mean Age at Follow-up		Total <i>n</i>	Male <i>n</i>	Female <i>n</i>	Gender Difference $\chi^2$	<i>p</i>
1 Year ( <i>n</i> =1,326)						
	No	115	51	64	2.808	.094
	Yes	1,211	636	575		
2 Years ( <i>n</i> =405)						
	No	38	17	21	0.168	.682
	Yes	367	177	190		
3 Years ( <i>n</i> =439)						
	No	48	22	26	0.634	.426
	Yes	391	203	188		

Table 64

*Weight Status Group Differences for Live Near a Park or Playground at ages 1, 2 and 3 Years*

Mean Age at Follow-up		Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
1 Year						<b>6.6786</b>	<b>.034</b>
	No	115	4	15	96		
	Yes	1,211	100	231	880		
2 Years						0.247	.884
	No	38	3	6	29		
	Yes	367	27	70	270		
3 Years						0.233	.890
	No	48	4	11	33		
	Yes	391	34	78	279		

Note. **Bolded** indicate significant group differences  $p < .05$ .

*School influences.*

School characteristics investigated at age 14 years included type ( $n=958$ , single gender= $160$ , coeducational  $n=798$ ); affiliation ( $n=958$ , government  $n=563$ , independent  $n=181$ , or Catholic  $n=214$ ); number of school facilities promoting physical activity ( $n=957$ ) and number of representative sport opportunities ( $n=142$ ).

There were no gender differences for school type ( $\chi^2=2.806$   $p=.094$ ), school affiliation ( $\chi^2=.665$   $p=.717$ ), government versus independent and Catholic ( $\chi^2=.652$   $p=.420$ ), nor the number of representative sport opportunities ( $\chi^2=.664$   $p=.882$ ).

The number of school facilities promoting physical activity was different for males and females ( $t= -2.319$   $p=.021$ ) with males attending schools with slightly more facilities ( $n=482$   $M=6.9$   $SD=3.2$ ) than females ( $n=475$   $M=6.5$   $SD=2.3$ ).

Significant weight status group differences were found for school type, school affiliation, and representative sport (Table 65), but not the number of school facilities promoting physical activity (Table 66). More individuals who were obese

attended coeducational, government (public) schools, and had fewer representative sport opportunities. Notably, Catholic schools had a higher proportion of overweight adolescents compared to both government and independent schools. Although the sample size was small for the number of school facilities, a significant weight status difference was found for males only, with the significant difference between the obese ( $n=46$   $M=5.8$   $SD=1.9$ ) compared to normal weight ( $n=348$   $M=6.9$   $SD=3.2$ ) and overweight ( $n=88$   $M=7.2$   $SD=3.4$ ) groups.

Table 65

*Weight Status Group Differences for School Characteristics at age 14 years*

School	Total	Obese	Overweight	Normal weight	$\chi^2$	$p$
Characteristic	$n$	$n$	$n$	$n$		
Type					<b>6.885</b>	<b>.032</b>
Coeducational	798	77	152	569		
Single gender	160	6	27	127		
Affiliation					<b>17.451</b>	<b>.002</b>
Government	563	63	101	399		
Catholic	214	13	51	150		
Independent	181	7	27	147		
Private versus Government					<b>11.059</b>	<b>.004</b>
Public	563	63	101	399		
Private	395	20	78	297		
Representative Sport					<b>13.804</b>	<b>.032</b>
$\leq 5$	39	1	11	27		
6	37	7	4	26		
7	38	0	10	28		
$\geq 8$	28	3	6	19		

Note. **Bolded** indicate significant group differences  $p < .05$ .



Table 66

*Weight Status Group Differences for the Number of School Facilities Promoting Physical Activity at age 14 Years*

Gender	ANOVA		<i>Post hoc</i> Tests p-value		
	<i>F</i>	<i>p</i>	Normal versus Overweight	Normal versus Obese	Overweight versus obese
Male	<b>3.119</b>	<b>0.045</b>	0.900	<b>0.003</b>	<b>0.010</b>
Female	0.093	0.912	0.995	0.972	0.994
Total	1.729	0.178	0.901	0.131	0.105

*Note.* **Bolded** indicate significant group differences  $p < .05$ .

## **Part Two. Linear Mixed Modelling**

This section reports two main components:

- The BMI longitudinal model development and results.
- Results from testing of covariates in the final longitudinal model of BMI.

### **BMI longitudinal model.**

The longitudinal model tracks BMI from birth to age 14 years. Details of results from the model development process along with the final model are provided.

#### ***Model development.***

The derivation of the basic model began with the dependent variable BMI and initial covariates. The repeated measure was time (0, 1, 2, 3, 6, 8, 10.5 and 14 years) to correspond to the average age in years at birth and each follow-up. Other covariates included age (actual age at assessment in months), gender (male or female), and IOTF BMI weight status calculated at 14 years (obese, overweight, normal weight).

A non-linear relationship between BMI and age was found. Non-linear transformations were applied to the variable age to determine the best model fit (based on Akaike's Information Criterion (AIC)). Polynomial fits were investigated to the fifth order, but the quadratic (age squared), compared to higher form transformations, provided the best model fit (AIC) (Appendix E, Table 1).

The linear term in the model did not account for variation in the early years, nor describe the functional form, particularly the initial peak around age one year, nor the nadir (adiposity rebound). Further transformations of age investigated included natural log of age, square root of age, age centred with adiposity rebound adjustment (Appendix E, Table 2). The final model included age, age squared and the natural log of age and was the best model overall based on simplicity, model fit and model diagnostic results.

Although data for preterm births <37 weeks were excluded from the sample, a model review with gestational age as a fixed effect found that gestational age continued to be an important contributor ( $p<.001$ ) and also improved model fit (AIC).

Fixed and random effects, interactions, covariance structure and model diagnostics were all investigated in the determination of the final model. The final linear mixed model found no significant gender-weight category; age/age<sup>2</sup>/log(age)-gestational age; gender-gestational age; nor weight category-gestational age interactions, and these were removed from the final model. The best covariance structure type was unstructured (Appendix E, Table 3). Final model diagnostics found that the residuals followed an approximate normal distribution, although there was a slight deviation at the positive tail (Figure 13). Individual residual plots were also examined by gender and weight status, with approximate normal distributions still observed, although the obese followed by overweight group demonstrated greater deviations, compared to normal weight group (Appendix E, Figure 1).

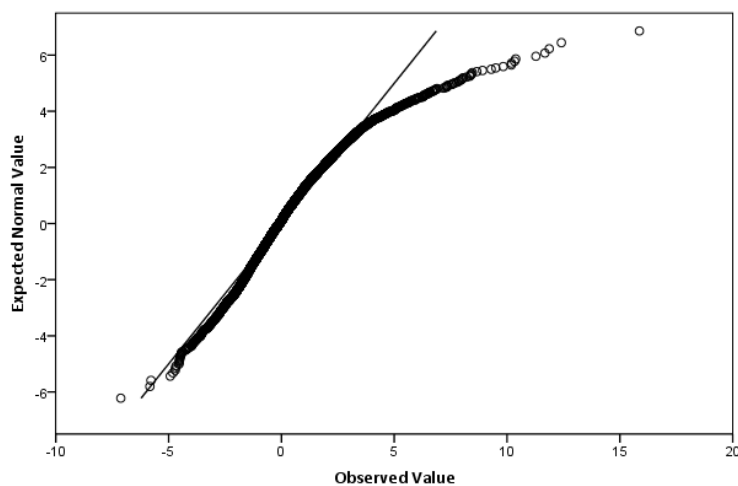


Figure 13. Normal Q-Q plot of residuals for the final BMI linear mixed model.

### ***Final Model<sup>16</sup>.***

The final model can be described by the following equation:

$$\begin{aligned} \text{BMI (predicted)} = & \text{intercept} + \text{weight status} + \text{gender} + \text{age(actual age)} + \text{age(actual} \\ & \text{age)}^2 + \text{logAge (actual age +1)} + \text{gestational age (actual gestational age)} + \\ & [\text{age(actual age)*gender}] + [\text{age(actual age)}^2 \times \text{gender}] + [\text{logAge (actual age +1)} \times \\ & \text{gender}] + [\text{age(actual age)} \times \text{weight status}] + [\text{age(actual age)}^2 \times \text{weight status}] + \\ & [\text{logAge (actual age +1)} \times \text{weight status}]. \end{aligned}$$

The model estimates and statistic results are shown in Table 67. By interchanging estimates into the equation, predicted BMI can be determined for any individual dependent upon their gender, weight status, age and gestational age.

Even with preterm children (<37 weeks) removed from the sample, there was a significant gestational age effect ( $p < .001$ ), with every additional week in gestation resulting in an increase in BMI at birth. At birth (model intercept) there was a significant difference between BMI of normal weight children compared to overweight ( $p = .029$ ) and obese ( $p = .019$ ), while the difference between overweight and obese was not significant. For example, for a female child born at 40 weeks gestation, their BMI at birth would be, if normal weight at age 14 years, 14.2 kg/m<sup>2</sup> compared to 14.4 kg/m<sup>2</sup> if overweight at age 14 years, or 14.5 kg/m<sup>2</sup> if obese at age 14 years. There was no significant gender effect between male and female BMI at birth. However, females increased their BMI at a faster rate than males ( $p < .001$ ), which accounts for the overlap seen in the trajectory at about eight years. The increase in BMI over time was statistically different for each weight status group, with the obese cohort having the largest rate of increase ( $p < .001$ ) as shown in Figure 14. The modelled BMI trajectories, with an overlay of mean age-adjusted BMI at each survey wave is depicted in Figure 14 indicating a relative good fit,

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<sup>16</sup> From "Longitudinal Modelling of Body Mass Index from Birth to 14 Years." By Chivers P, Hands B, Parker H, Beilin L, Kendall G, Bulsara M, 2009, *Obesity Facts*, 2, p.308. Copyright 2009 S. Karger AG, Basel. Adapted with permission.

supported by the residual diagnostics which followed an approximate normal distribution, although there was a slight deviation at the positive tail.

Table 67

*Final BMI Linear Mixed Model: Estimates of Fixed Effects for Parameters*

Parameter	Estimate	S.E.	p
Intercept	8.334684	0.762569	<.001
Weight status – Obese group	0.314924	0.134407	.019
Weight status – Overweight group	0.201214	0.092172	.029
Weight status – Normal weight group	0	0	
Gender – female	-0.038381	0.071336	.591
Gender – male	0	0	
Age	-0.121952	0.002623	<.001
Age <sup>2</sup>	0.000614	0.000011	<.001
LogAge	1.546032	0.033056	<.001
Gestational Age	0.146582	0.019332	<.001
<b>Interactions</b>			
Age*gender – female	0.015887	0.003451	<.001
Age*gender – male	0	0	
Age <sup>2</sup> *gender – female	-0.000027	0.000015	.076
Age <sup>2</sup> *gender – male	0	0	
LogAge*gender – female	-0.217211	0.043482	<.001
LogAge*gender – male	0	0	
Age*Weight status – Obese	0.028256	0.006479	<.001
Age*Weight status – Overweight	0.005560	0.004472	.214
Age*Weight status – Normal weight	0	0	
Age <sup>2</sup> *Weight status – Obese	0.000269	0.000028	<.001
Age <sup>2</sup> *Weight status – Overweight	0.000144	0.000019	<.001
Age <sup>2</sup> *Weight status – Normal weight	0	0	
LogAge*Weight status – Obese	-0.036766	0.081960	.654
LogAge*Weight status – Overweight	0.030509	0.056258	.588
LogAge*Weight status – Normal weight	0	0	

*Note.* From “Longitudinal Modelling of Body Mass Index from Birth to 14 Years.” By Chivers P, Hands B, Parker H, Beilin L, Kendall G, Bulsara M, 2009, *Obesity Facts*, 2, p.308. Copyright 2009 S. Karger AG, Basel. Adapted with permission.

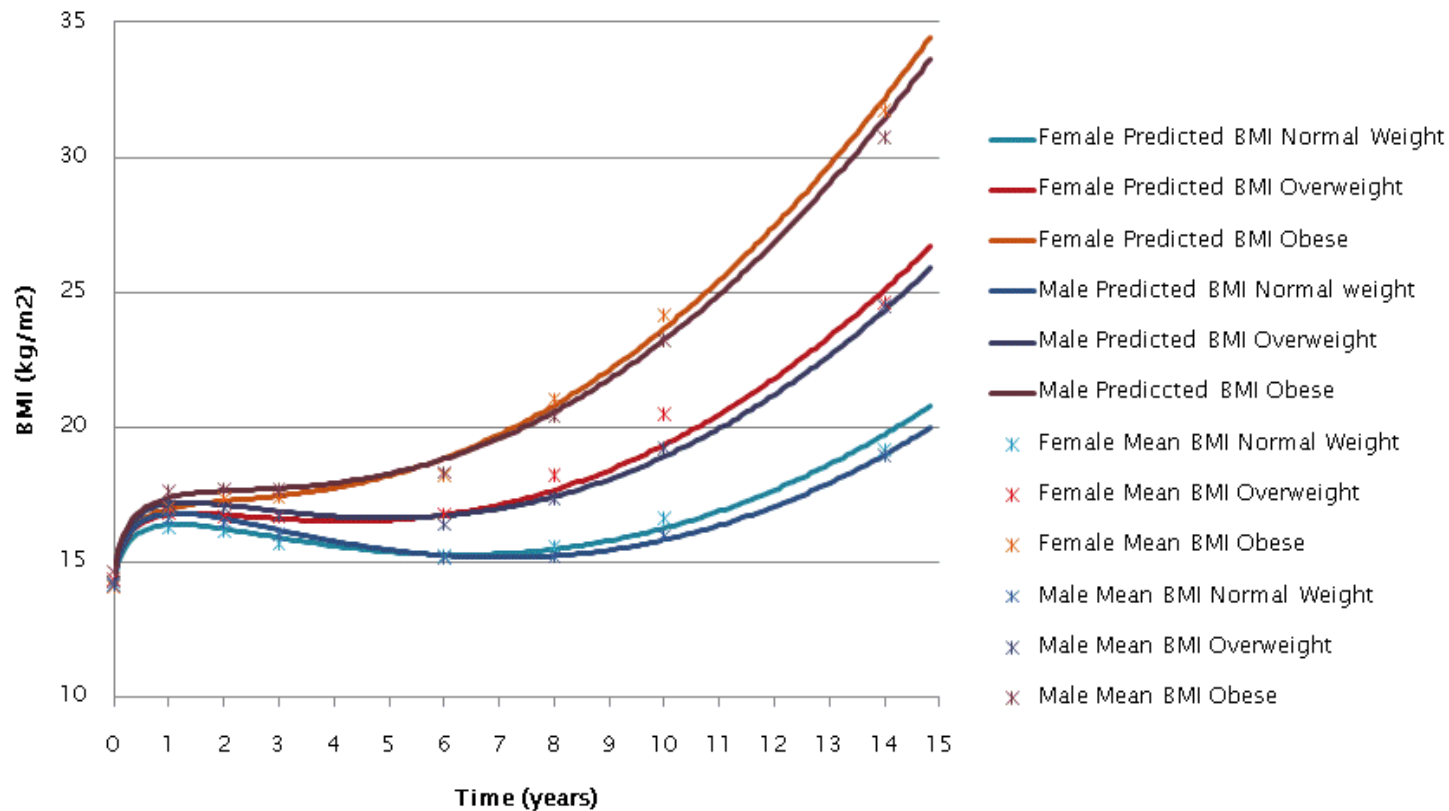


Figure 14. Predicted BMI trajectories from birth to 14 years, separated by weight categories normal weight, overweight and obese (determined at 14 years using IOTF cut-offs) and gender. Mean age-adjusted BMI calculated for each survey wave is overlaid for each weight category and gender to demonstrate goodness of fit to the predicted BMI trajectory model. From “Longitudinal Modelling of Body Mass Index from Birth to 14 Years.” By Chivers P, Hands B, Parker H, Beilin L, Kendall G, Bulsara M, 2009, Obesity Facts, 2, p.305. Copyright 2009 S. Karger AG, Basel. Adapted with permission.

### Linear Mixed Modelling Testing of Covariates.

A summary of covariate results are reported separately under individual (Table 68), behavioural (Table 69) and environmental (Table 70) headings below. A comprehensive review of covariate models with summaries is provided in the Technical Report, Part Two.

#### *Individual factors and the BMI linear mixed model.*

Table 68

*Summary Results from Testing of Individual Factors as Covariates in the BMI Linear Mixed Model*

Variable	Covariate only Model		All interactions model		Notable Model			Comments
	AIC	<i>p</i>	AIC	<i>p</i>	AIC	Interaction ( <i>p</i> )	<i>p</i>	
Early Infant Feeding								
Weaning	<b>29659.759</b>	<b>NS</b>	29713.193	NS				
Breastfeeding (4 month cut-off)	<b>29690.838</b>	<b>NS</b>	29713.393	NS	29706.654	*gender (.019) *age (all) (<.001)	NS	
Other Milk (4 month cut-off)	<b>29516.452</b>	<b>NS</b>	29536.679	NS	29531.398	*gender (.011) *age (all) (<.001)	NS	

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction ( <i>p</i> )	<i>p</i>	
	AIC	<i>p</i>	AIC	<i>p</i>				
Breastfeeding and Other Milk (4 month cut-off)	29384.378	NS	29426.275	<.05	<b>29377.240</b>	<b>BF*OM (.011)</b>	<b>&lt;.05</b>	Mixed feeding before four months highest increase in BMI.
Diet								
Fruit	<b>30767.418</b>	<b>NS</b>	30897.400	NS				
Vegetable intake	30762.046	NS	30871.354	.027	<b>30745.871</b>	<b>*BMICat (.006)</b> <b>*gweeks (.028)</b>	<b>.030</b>	
Fat intake at 8 years	<b>23157.746</b>	<b>NS</b>	23228.760	NS				
Fat intake at 14 years	<b>17618.081</b>	<b>NS</b>	17692.676	NS				
Developmental Milestones and Motor Competence								
Parent reported developmental milestones (Overall score).	<b>13360.438</b>	<b>NS</b>	13441.674	NS				
Denver II assessment (years 1-3) <sup>1</sup> .								
Gross Motor (y1)	<b>29097.767</b>	<b>.043</b>	29158.753	NS				Normal gross motor had higher BMI, year one only.



Variable	Covariate only Model		All interactions model		Notable Model			Comments
	AIC	<i>p</i>	AIC	<i>p</i>	AIC	Interaction ( <i>p</i> )	<i>p</i>	
Fine Motor (y1)	<b>29011730</b>	<b>NS</b>	29075.881	NS				Representative of years two and three.
Personal / Social (y1)	28898.056	NS	28952.525	NS	<b>28886.279</b>	<b>*BMICat (.009)</b>	.010	Non-normal development had higher BMI for years one and two.
Language (y1)	29005.828	.043	29069.590	NS	<b>28998.857</b>	<b>*gender (.009)</b>	<b>NS</b>	Non-normal development, obese and female increased BMI in years one and three.
Overall (y3)	<b>20260.410</b>	<b>NS</b>	20349.429	NS				Data only year 3.
Infant Monitoring Questionnaire								
Overall age 2 and 3 years. <sup>1,2</sup>								
Hearing (y2)	<b>25258.458</b>	<b>NS</b>	25278.751	NS				NS for three years.
Talking (y3)	27971.523	NS	28004.896	NS	<b>27967.485</b>	<b>*gender (.019)</b>	<b>NS</b>	Females not similar to peers increased BMI.
Understand speech (y2)	<b>25249.058</b>	<b>NS</b>	25275.873	NS				NS for two years. NS for three years.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction ( <i>p</i> )	<i>p</i>	
	AIC	<i>p</i>	AIC	<i>p</i>	AIC	Interaction ( <i>p</i> )	<i>p</i>	
Walk, run, climb (y2)	25188.912	NS	25194.452	NS	<b>25175.080</b>	<b>*BMICat (.002)</b>	<b>.001</b>	Obese group not like their peers increased BMI. NS for three years.
Worries (y2)	25148.370	NS	25186.053	NS	<b>25143.123</b>	<b>*BMICat (.015)</b>	<b>NS</b>	Obese and no worries increased BMI. NS for three years.
Medical (y2)	25007.665	NS	25037.842	.003	<b>25003.004</b>	<b>*gweeks (*.003)</b>	<b>.004</b>	Gestational age 38 weeks and no problems increased BMI. NS for three years.
Parent perceived child skills at 10 years (eg Run). <sup>3</sup>	<b>29434.900</b>	<b>NS</b>	29532.502	NS				All abilities reported NS covariate and interactions models.
Motor Competence (MAND)								
Total Fine Motor (10y)	26983.601	NS	27066.113	NS				
Total Gross Motor (10y)	<b>26881.924</b>	<b>NS</b>	26956.511	NS	26886.821	<b>*BMICat (.004)</b>	NS	Overweight group only.

Variable	Covariate only		All interactions		Notable Model			
	Model		model		AIC	Interaction ( <i>p</i> )	<i>p</i>	Comments
	AIC	<i>p</i>	AIC	<i>p</i>				
NDI Score (10y)	<b>26832.066</b>	<b>NS</b>	26913.838	NS	26842.847	*BMICat (.040)	NS	Overweight group only.
Total Fine Motor (14y)	<b>30559.227</b>	<b>NS</b>	30635.678	NS	30565.530	*BMICat (.007)	NS	Obese group only.
Total Gross Motor (14y)	<b>30559.656</b>	<b>NS</b>	30639.576	NS	30569.498	*BMICat (.039)	NS	Overweight group only.
NDI Score (14y)	<b>30560.772</b>	<b>NS</b>	30641.989	NS	30571.230	*BMICat (.023)	NS	No subgroups significant.
Physical Activity (14 years)								
Aerobic fitness (PWC 170).	<b>29088.796</b>	<b>&lt;.001</b>	29158.066	NS	29093.113	*BMICat(<.001)	NS	Increase in PWC 170 increases BMI.
<i>Weight adjusted (PWC 170/kg).</i>	<i>28977.651</i>	<i>.003</i>	<i>28939.370</i>	<i>NS</i>	<b>28934.813</b>	<b>*age (.040)</b> <b>*BMICat (NS)</b>	<b>NS</b>	<i>Obese group largest decrease in BMI (p=.022) as fitness increases.</i>
Fitness Tests								
Muscle endurance (curl-ups)	<b>30359.583</b>	<b>NS</b>	30433.652	NS	30370.390	*BMICat (.042)	NS	
Muscle strength(basketball throw)	30536.777	.008	30540.634	NS	<b>30527.312</b>	<b>*BMICat (&lt;.001)</b>	<b>NS</b>	Overall estimates showed for increasing score BMI increased for obese and overweight.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction ( <i>p</i> )	<i>p</i>	
	AIC	<i>p</i>	AIC	<i>p</i>				
Flexibility								
Sit and Reach (left leg)	<b>30509.117</b>	NS	30591.293	NS				
Sit and reach (right leg)	<b>30508.511</b>	NS	30588.870	NS				
Sit and reach (both legs)	<b>30494.348</b>	NS	30575.202	NS				
Shoulder stretch (left)	<b>30534.212</b>	NS	30565.836	NS				
Shoulder stretch (right)	<b>30555.535</b>	NS	30578.538	NS				
Puberty (Pubic Hair)	23948.811	NS	24045.299	NS	<b>23942.456</b>	<b>*BMICat (NS)</b>	<b>NS</b>	Subgroup interaction between group obese and puberty.

*Note.* **Bolded** is best model according to AIC fit. Probability value (*p*) is for the covariate in the model, as defined by variable label in first column. NS=not significant, BMICat=IOTF weight status group categories. Unless specified \*age represents all age transformation interactions with covariate.

<sup>1</sup>Data were collected over the follow-up years, however only the year with the largest sample size is reported usually as representative of other years (refer to comments column specific to variable).

<sup>2</sup> Infant Monitoring Questionnaire results were non-specific across gross and fine motor skills for follow-ups one to three years. Overall results only are presented here.

<sup>3</sup>Parent perception of abilities at follow-up 10 years for skip, throw, catch, kick, strike, ride a bike, balance and coordination reported a non-significant covariate and non-significant interactions.

***Behavioural factors and the BMI model.***

Table 69

*Summary Results from Testing of Behavioural Factors as Covariates in the BMI Linear Mixed Model*

Variable	Covariate only		All interactions		Notable Model			Comments
	Model	p	model	p	AIC	Interaction(p)	p	
<b>Physical Activity Behaviours.</b>								
Frequency of visits to a park (y1)	<b>27006.836</b>	<b>NS</b>	27148.442	NS				Representative of years two and three.
<b>Parent reported activity levels</b>								
6 years organised activity	<b>29075.709</b>	<b>NS</b>	29116.123	NS	29107.063	*log(age)(.043)	NS	
6 years organised sport	<b>29073.524</b>	<b>.009</b>	29117.883	NS				Children not involved had lower BMI.
8 years organised activity	<b>29416.855</b>	<b>NS</b>	29492.653	NS				
8 years organised sport	29259.593	NS	29289.117	NS	<b>29255.266</b>	<b>*gender (.010)</b>	<b>NS</b>	Overall for females BMI decreased if involved.
10 years Total activity	<b>30962.873</b>	<b>NS</b>	31031.901	NS	31005.257	*log(age)(.031)	.038	Non-significant covariate models best fit, although overall increased activity decreased BMI.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction(p)	p	
	AIC	p	AIC	p	AIC			
Physical activity level (Mean daily step count).	13412.776	NS	13573.674	NS	13445.853	*BMICat(NS)	.048	Obese group with low step counts increase BMI.
Physical activity Attitudes and values.								
Encouragement and support.	<b>19533.362</b>	<b>NS</b>	19608.464	NS				
Physical education and exercise.	<b>30687.764</b>	<b>.022</b>	30695.816	NS				As participation score increased, BMI increased.
Effects of physical activity.	<b>30278.367</b>	<b>.034</b>	30360.253	NS	30287.617	*BMICat (.035)	NS	Increase in score resulted in very small increase in BMI.
Importance of physical activity.	<b>29631.513</b>	<b>NS</b>	29713.449	NS				
Excuses for not increasing physical activity in the future.	<b>29933.701</b>	<b>NS</b>	30010.195	NS				
Sedentary Behaviour.								
Television 6 Years	<b>29176.513</b>	<b>NS</b>	29336.511	NS				
Television 8 Years	<b>29439.766</b>	<b>NS</b>	29582.335	NS				
Television 10 Years	<b>29463.027</b>	<b>NS</b>	29628.994	NS				
Television 14 Years	30849.777	NS	30996.701	NS	<b>30849.007</b>	<b>*BMICat (NS)</b>	<b>NS</b>	Higher screen time increases BMI.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction(p)	p	
	AIC	p	AIC	p				
Computer 14 Years	<b>30857.296</b>	<b>NS</b>	31004.643	NS				
Screen Time 14 Years	<b>30834.753</b>	<b>NS</b>	30991.642	NS				
Self Concept (SPPA).								
Athletic competence	<b>30200.253</b>	<b>NS</b>	30248.979	NS				
Global Self Worth	<b>30247.780</b>	<b>NS</b>	30287.923	NS				
Physical appearance	30255.869	.034	30275.957	NS	<b>30249.552</b>	<b>*gender(.002)</b>	<b>NS</b>	As score increases BMI decreases, greatest for females.
Job	<b>30203.930</b>	<b>.030</b>	30250.769	NS				As score increases BMI increases.
Scholastic	<b>30242.001</b>	<b>.013</b>	30293.024	NS				As score increases BMI increases.
Social	<b>30228.282</b>	<b>NS</b>	30273.381	NS				
Romantic	<b>28137.475</b>	<b>NS</b>	28177.397	NS	28167.067	<b>*log(age) .018</b>	<b>NS</b>	
Behaviour	<b>30247.839</b>	<b>NS</b>	30293.510	NS				
Friend	<b>30246.382</b>	<b>NS</b>	30293.398	NS				
Parent reported academic, social and behavioural progress 10y.								
Academic performance	29473.498	NS	29604.437	NS				
Learning skills	29436.917	NS	29554.061	NS	<b>29432.138</b>	<b>*gender (NS)</b>	<b>NS</b>	Significant subgroup <i>dissatisfied</i> .

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction(p)	p	
	AIC	p	AIC	p	AIC			
Social progress	29470.425	NS	29584.652	NS	<b>29462.518</b>	<b>*gender (NS)</b> <b>*BMICat (NS)</b>	<b>NS</b>	Groups obese and overweight <i>neither</i> increase in BMI, with females higher than males.
Behavioural progress	<b>29472.420</b>	<b>NS</b>	29565.752	NS				

*Note.* **Bolded** is best model according to AIC fit. P-value is for the covariate in the model, as defined by variable label in first column. NS=not significant, BMICat=IOTF weight status group categories. Unless specified \*age represents all age transformation interactions with covariate. Where an interaction term is noted, but NS, typically this represented a subgroup interaction only was significant, although overall NS.



***Environmental factors and the BMI model.***

Table 70

*Summary Results from Testing of Environmental Factors as Covariates in the BMI Linear Mixed Model*

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction (p)	p	
	AIC	p	AIC	p	AIC			
<b>Socioeconomic Status</b>								
SEIFA (categorical)	<b>27781.599</b>	<b>NS</b>	277856.721	NS				
Mother's ethnicity	<b>30938.406</b>	<b>.001</b>	31065.057	NS				Non-Caucasian had lower BMI.
Father's ethnicity	30586.288	<.001	30729.332	NS	<b>30565.134</b>	<b>*BMICat (.036)</b>	<b>NS</b>	Generally non-Caucasian had lower BMI.
Family Income (7-category)	<b>28249.604</b>	<b>&lt;.001</b>	Invalid model†					Low income largest decrease in BMI
Mother's education	<b>30958.638</b>	<b>.043</b>	31123.869	NS				Trade and apprentice increased BMI.
Father's education	<b>26753.182</b>	NS	26907.944	NS				
Father's occupation	<b>24835.244</b>	<b>NS</b>	25068.172	NS				
Family employment	<b>26027.042</b>	<b>NS</b>	26116.124	NS	26102.981	*age (<.05) *BMICat (NS)	.026	Subgroup interaction between overweight and neither employed, increasing BMI.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction (p)	p	
	AIC	p	AIC	p				
Minimum family work hours (categorical)	29818.698	NS	29836.171	NS	<b>29815.634</b>	<b>*age (&lt;.05)</b>	<b>.031</b>	BMI decreased with increasing hours.
Mother's work	<b>30351.931</b>	<b>NS</b>	30544.519	NS	30537.015	*age/age^2 (<.01) *BMICat (.021)	.004	Children with non-working mother's increase BMI, pronounced for obese.
Mother's work hours (categorical)	<b>29781.470</b>	<b>NS</b>	29839.047	NS	29820.400	*age(<.05)	.030	
Family Life stress (dichotomous)	25612.415	NS	25650.091	.008	<b>25611.593</b>	<b>*gweeks (.013)</b>	<b>.012</b>	Term babies increased BMI with low stress levels.
Parental Influences								
Maternal smoking during pregnancy	<b>24739.106</b>	<b>NS</b>	24772.576	NS	24763.845	*age (<.05)	.016	Smoking during pregnancy decreased BMI at birth, but higher than non-smoking group thereafter.
Mother's birth weight	<b>23919.167</b>	<b>&lt;.001</b>	24051.880	NS	24002.621	*gender(.036) *age(<.05)	<.001	Increases result in child's BMI increase.
Father's birth weight	<b>17362.859</b>	<b>.017</b>	17496.836	NS				Increases result in child's BMI increase.
Mother's Pre Pregnancy BMI	30928.194	<.001	30992.925	NS				

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction (p)	p	
	AIC	p	AIC	p				
Birth Father's BMI	<b>25910.440</b>	<b>.005</b>	25969.087	NS				Increases result in child's BMI increase.
Y8 Mother's BMI	<b>25969.822</b>	<b>&lt;.001</b>	26036.926	NS	26007.916	*Log(age)(.024)	<.001	Increases result in child's BMI increase.
Y8 Father's BMI	<b>8841.655</b>	<b>NS</b>	8892.534	NS				<i>n</i> =387
Y14 Mother's BMI	<b>23931.802</b>	<b>.001</b>	23985.616	NS				Increases result in child's BMI increase.
Parenting Scale 10y								
Total Score	<b>29472.104</b>	<b>NS</b>	29551.984	NS	29477.233	*BMICat (.005)	.027	Overweight group increased BMI with increase score.
Laxness Factor	<b>29466.057</b>	<b>NS</b>	29507.249	NS				
Over-reactivity Factor	<b>29466.123</b>	<b>NS</b>	29505.700	NS				
Verbosity Factor	<b>29467.674</b>	<b>NS</b>	29507.832	NS				
Child Perception of Parenting 14y								
Smile at me	30240.681	.015	30326.537	NS	<b>30232.640</b>	<b>*BMICat (NS)</b>	<b>.026</b>	Never smiles decreases in BMI, obese and often smiles largest increase in BMI.
Forget rules	<b>30235.986</b>	<b>NS</b>	30352.931	NS				

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction (p)	p	
	AIC	p	AIC	p				
Praise me	30231.418	NS	30344.209	NS	<b>30231.015</b>	<b>*BMICat (NS)</b>	<b>NS</b>	Group obese and sometimes had highest increase in BMI.
Nag me	30183.034	NS	30293.694	NS	<b>30182.681</b>	<b>*BMICat (NS)</b>	<b>NS</b>	Group overweight and sometimes or often small decrease in BMI.
Rules when suit	30150.892	NS	30254.466	NS	<b>30143.747</b>	<b>*gender (.020)</b> <b>*BMICat (.023)</b>	<b>.020</b>	Mixed results with no definitive trend.
Appreciate me	<b>30206.094</b>	<b>NS</b>	30317.240	NS				
Threaten punishment	<b>30229.351</b>	<b>NS</b>	30345.112	.018	30231.570	<b>*gweeks (.017)</b>	<b>.018</b>	
Speak positively	<b>30234.954</b>	<b>NS</b>	30340.644	NS				
Enforce rules inconsistent	<b>30122.704</b>	<b>NS</b>	30226.997	NS				
Threaten or hit	<b>30213.200</b>	<b>NS</b>	30305.990	.011	30213.851	<b>*gweeks (.035)</b>	<b>.035</b>	
Proud of me	<b>30249.805</b>	<b>NS</b>	30353.089	NS				
Built Environment								
Home swimming pool (y1)	<b>29644.912</b>	.011	29684.210	NS				BMI decreased for no pool and representative of years two and three.

Variable	Covariate only		All interactions		Notable Model			Comments
	Model		model		AIC	Interaction (p)	p	
	AIC	p	AIC	p				
Outdoor play space (y1)	29693.064	NS	29714.318	NS	<b>29684.920</b>	<b>*BMICat (.013)</b>	<b>.021</b>	No outdoor play space increases BMI for overweight and obese. Year one only.
Live near a park / playground (y2)	9263.652	.023	9285.976	NS	<b>9257.302</b>	<b>*BMICat (.046)</b>	<b>.001</b>	No park nearly increases BMI for years two and three only.
School Influences (y14)								
Affiliation	<b>21119.192</b>	<b>NS</b>	21189.324	NS				
Coeducation	<b>21116.166</b>	<b>NS</b>	21148.035	NS				
Facilities	<b>21103.059</b>	<b>NS</b>	21164.923	NS				
Representative Sport (categorical)	3073.214	NS	3138.721	NS	3052.438	*gender(NS) *BMICat(NS)	NS	Lowest category and obese interaction (p=.005) with decrease in BMI.

Note. **Bolded** is best model according to AIC fit. SEIFA=Socioeconomic Index for Areas (advantage/disadvantage). Unless specified \*age represents all age transformation interactions with covariate.

† Warning. Iteration was terminated but convergence has not been achieved. The MIXED procedure continues despite this warning. Subsequent results produced are based on the last iteration. Validity of the model fit is uncertain. Predicted estimates were non-sense and many reported ‘.’.

### **Part Three. Structural Equation Modelling (SEM)**

The subset of variables included in this part of the analysis were those with statistically significant results for either weight status group differences or LMM. In addition some prominent obesogenic variables were also included, although the prior analysis showed in this study that they were not statistically significant (e.g. fat intake). All variables were used in the model building mode of structural equation modelling, each with pathways to BMI. Non-significant pathways were then removed followed by single path building using modification indices.

Preliminary interrelationship modelling was investigated at each follow-up. Based on constraints of the data (e.g. sample sizes and variables available), only models for follow-ups age 6 years to 14 years are described in detail.

For each final model the following details are reported:

- A correlation matrix table with means and standard deviations<sup>17</sup>;
- A figure depicting the interrelationships with notated standardised estimates (along path or near arrow head) and squared multiple correlation (top right hand corner of variable box); and
- A table with unstandardised estimates.

Standardised estimates represent changes in standard deviations. That is, as the value of variable X goes up by one standard deviation, the value of variable Y changes by the Z standard deviations. Unstandardised estimates represent changes in the actual values of variables. The figure titles will include model fit information, namely chi-square ( $\chi^2$ ), Root Mean Square Error of Approximation (RMSEA) with upper and lower confidence interval (CI), Comparative Fit Index (CFI) and Tucker Lewis Index (TLI). Their appropriate cut-offs for judging good fit are  $\chi^2 > .05$ , RMSEA  $\leq .05$ , CFI  $\geq .90$  and TLI  $\geq .90$ .

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<sup>17</sup> Note for some variables the mean and standard deviation is based on a categorical scale.

### **Birth to follow-up age 3 years.**

At birth, BMI was, not surprisingly, comprised of height and weight as main predictors. As reported in previous descriptive and modelling results, there were few gender or weight status group differences. However mother's pre-pregnancy BMI was related to birth BMI, and this may be influenced by both the mother's and father's ethnicity.

In the first year of life, SEM demonstrated that the only predictor of BMI was the duration of breastfeeding. Other variables did not play any direct or indirect roles in predicting BMI. Of interest, those children who were breastfed for longer tended to be taken to the park more frequently.

In the second and third years, sample size and number of relevant variables for these follow-ups negated any further investigation. Socioeconomic status and Denver gross motor skills however appeared to be predictive of BMI.

### **Follow-up age 6 years.**

At follow-up age 6 years, variables selected included family income, organised sport (at school or with club), organised activity (music, dancing, kindy gym, other clubs), amount of television watched daily, and child's BMI. Exploratory investigations found all variables important in the prediction of BMI at age 6 years, however only television watching was directly related to BMI. The correlation matrix is reported in Table 71, unstandardised estimates in Table 72, and final model in Figure 15.

Table 71

*Summary of Correlations, Means and Standard Deviations for Variables used in the Final Structural Equation Model at age 6 years*

	1	2	3	4	<i>n</i>	<i>M</i>	<i>SD</i>
1. Family Income	-				1,282	0.74	0.44
2. Organised Sport	.016	-			1,299	0.33	0.47
3. Organised Activity	.093**	.014	-		1,299	0.34	0.47
4. TV	-.053	-.089**	-.111***	-	1,303	2.84	1.0
5. BMI	-.048	.028	-.029	.110***	1,259	15.8	1.8

*Note.* TV = time spent watching television, and BMI = child's BMI.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , two-tailed.

Table 72

*Summary of Unstructured Regression Weights for Variables used in the Final Structural Equation Model at age 6 years*

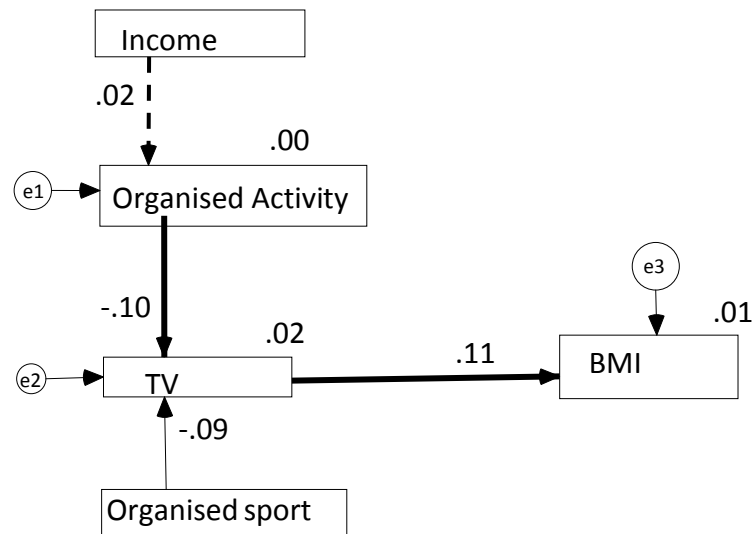
	Constrained equal			Freely estimated					
				Male			Female		
	P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>
Activity <--- Income				.018	.036	.624	.187	.045	<.001
TV <--- Activity	-.232	.058	<.001						
TV <--- Sport	-.187	.059	.001						
BMI <--- TV	.197	.050	<.001						

*Note.* P.E. = parameter estimate, S.E. = standard error, Activity = organised activity, income = family income, TV = amount of television watched daily, Sport = organised sport and BMI = child's BMI.

Gender multi-group analysis found significant differences in the path between income and organised activity, intercept and error variance for organised activity, and mean of organised sport. For males the pathway between income and organised activity was not significant while for females it was significant. All other parameters were not significantly different between males and females. Overall, at age 6 years, the predictors of BMI accounted for one percent of BMI variance for both males and females.



Male



Female

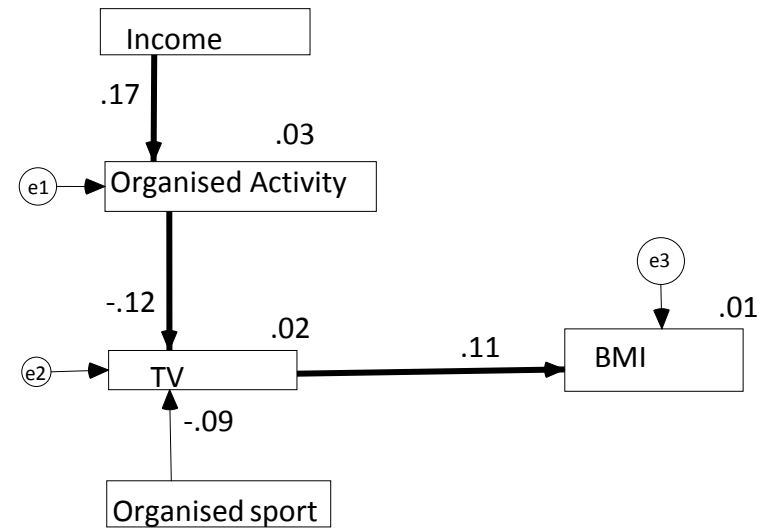


Figure 15. Estimated relations among predictors of BMI at age 6 years for all paths included in the final model for males and females.

Standardised coefficients are reported. Model fit  $\chi^2$  (df=22) = 25.036  $p$ =.295; RMSEA=.010 [CI .010-.025]; CFI=.932; TLI=.907.

TV = time spent watching television, and BMI = child's BMI.

$p > .05$  (---),  $p < .05$  (—),  $p < .001$  (—).

### Follow-up age 8 years.

Exploratory modelling found that self reported fat intake did not have any direct or indirect relationship with BMI and was excluded from further modelling. There was no relationship between family income and mother's BMI at follow-up age 8 years, however mother's BMI was the strongest predictor of child BMI and was related to increased television viewing. Television viewing had no direct effect on BMI, but was indirectly associated with BMI by the reduction in time spent in organised sport or activity. Correlation matrix is reported in Table 73, unstandardised estimates in Table 74, and final model in Figure 16.

Table 73

*Summary of Correlations, Means and Standard Deviations for Variables used in the Final Structural Equation Model at age 8 years*

	1	2	3	4	5	<i>n</i>	<i>M</i>	<i>SD</i>
1. Family Income	-					1,279	.73	.44
2. Mother's BMI	-.032	-				1,154	25.9	5.6
3. Organised Activity	.042	-.053	-			1,308	1.3	0.7
4. Organised sport	.076**	-.034	.347***	-		1,303	.80	.40
5. TV	-.056*	.176***	-.156***	-.072*	-	1,309	2.8	1.0
6. BMI	-.029	.306***	-.120***	.012	.112***	1,271	16.8	2.5

*Note.* TV = time spent watching television, and BMI = child's BMI.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , two-tailed.

Gender invariance testing found no significant differences for any paths, means or error variances. The only significant gender difference was for the intercept organised activity. Overall, at age 8 years, the predictors of BMI accounted for 10% and 11% of BMI variance for males and females respectively.

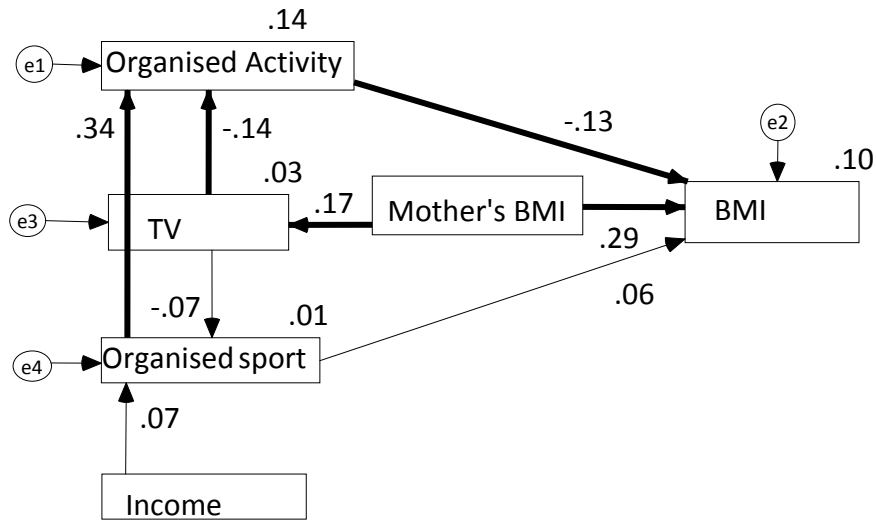
Table 74

*Summary of Unstructured Regression Weights for Variables used in the Final Structural Equation Model at age 8 years*

Regression Path			P.E.	S.E.	<i>p</i>
TV	<---	Mother's BMI	.030	.005	<.001
Organised Sport	<---	Income	.066	.025	.009
Organised Sport	<---	TV	-.028	.011	.015
Organised Activity	<---	Organised Sport	.545	.042	<.001
Organised Activity	<---	TV	-.092	.017	<.001
BMI	<---	Organised Sport	.401	.181	.026
BMI	<---	Organised Activity	-.492	.110	<.001
BMI	<---	Mother's BMI	.133	.012	<.001

*Note.* P.E. = parameter estimate, S.E. = standard error, TV = time spent watching television, and BMI = child's BMI. No gender separated estimates are presented as all pathways were constrained equal due to non-significant gender differences.

Male



Female

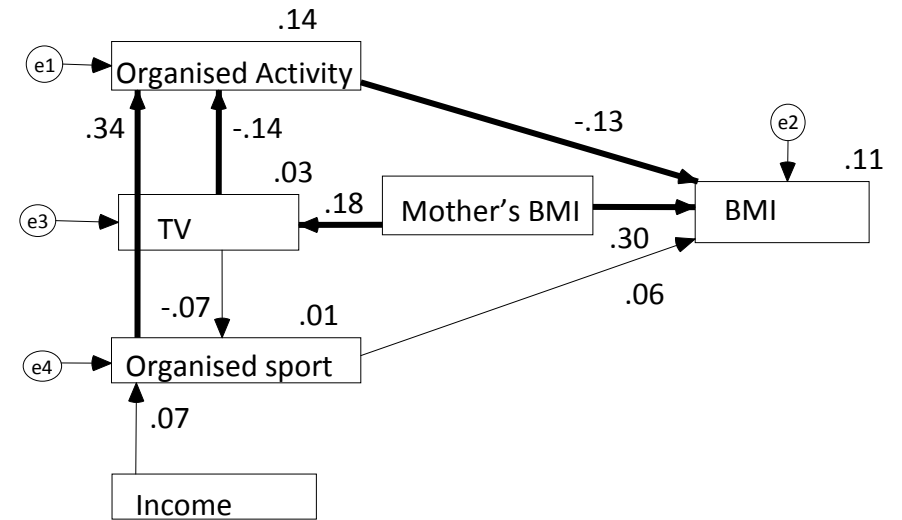


Figure 16. Estimated relations among predictors of BMI at 8 years for all paths included in the final model for males and females.

Standardised coefficients are reported. Model fit  $\chi^2$  (df=32) = 33.326  $p$ =.403; RMSEA=.005 [CI .0 - .021]; CFI=.996; TLI=.995.

TV = time spent watching television, and BMI = child's BMI.

$p > .05$  (---),  $p < .05$  (—),  $p < .001$  (—).

### **Follow-up age 10 years.**

Exploratory modelling found that the parent reported Parent Scale did not have any direct or indirect pathways to BMI and was excluded from further investigation. There was a strong correlation between parent reported movement skill and coordination, and motor competence (MAND NDI). Motor competence (MAND NDI) and coordination were also correlated with family income, a marker of SES. Movement skill was the strongest predictor of BMI followed by time spent watching television. Movement skill also influenced the amount of physical activity, with physical activity having an inverse relationship with BMI. The correlation matrix is reported at Table 75, unstandardised estimates at Table 76, and final model at Figure 17.

Gender multi-group analysis found a significant difference between the path coordination to activity, the mean and covariance of the MAND NDI scores. For males, coordination was more predictive of activity levels. All other parameters were not significantly different between males and females. Overall, at age 10 years, the predictors of BMI accounted for 8% of BMI variance for both males and females.

Table 75

*Summary of Correlations, Means and Standard Deviations for Variables used in the Final Structural Equation Model at 10 years*

	1	2	3	4	5	6	<i>n</i>	<i>M</i>	<i>SD</i>
1. Income	-						1,290	0.74	0.44
2. TV	-.062*	-					1,312	3.0	1.0
3. Activity	.051	-.044	-				1,403	6.4	3.9
4. Movement skill	.025	-.033	.147***	-			1,290	14.0	3.6
5. Coordination	.067*	-.026	.141***	.382***	-		1,312	3.3	0.7
6. Motor competence	.077**	-.093**	.103***	.281***	.177***	-	1,186	94.3	14.4
7. BMI	-.099**	.138***	-.113***	-.233***	-.130***	-.085**	1,246	18.6	3.3

*Note.* TV = time spent watching television, Motor competence = MAND NDI score, Coordination = coordination and developmental progress, and BMI = child's BMI.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , two-tailed.

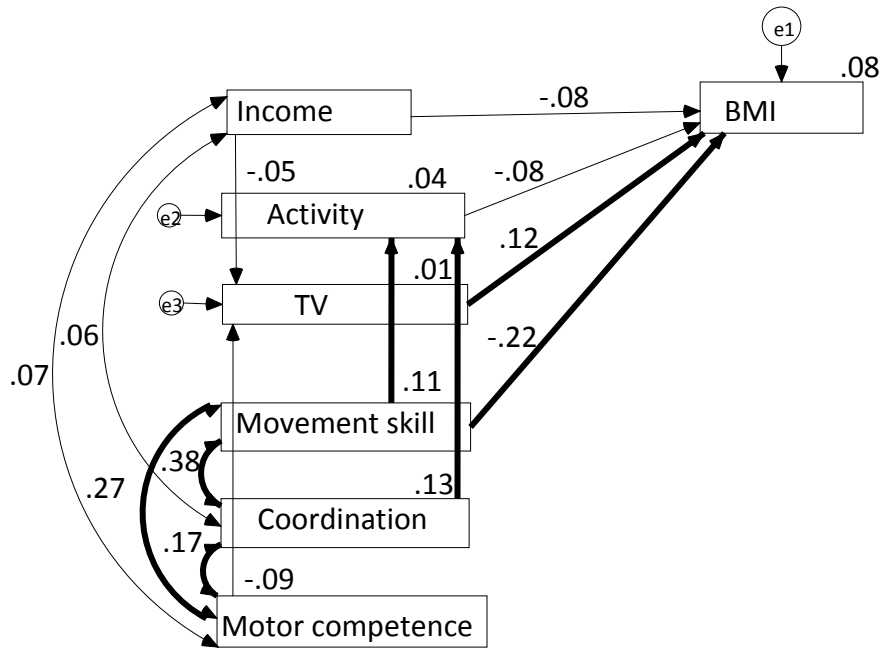
Table 76

*Summary of Unstructured Regression Weights for Variables used in the Final Structural Equation Model at age 10 years*

			Constrained equal			Freely estimated					
						Male			Female		
			P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>
TV	<---	Motor Competence	-.006	.002	.002						
Activity	<---	Movement skill	.121	.032	<.001						
Activity	<---	Coordination				.708	.164	<.001	.458	.164	.005
TV	<---	Income	-.124	.063	.049						
BMI	<---	Activity	-.064	.023	.006						
BMI	<---	TV	.414	.092	<.001						
BMI	<---	Movement skill	-.198	.025	<.001						
BMI	<---	Income	-.623	.209	.003						

*Note.* P.E. = parameter estimate, S.E. = standard error, TV = time spent watching television, Motor competence = MAND NDI score, Coordination = coordination and developmental progress, and BMI = child's BMI.

Male



Female

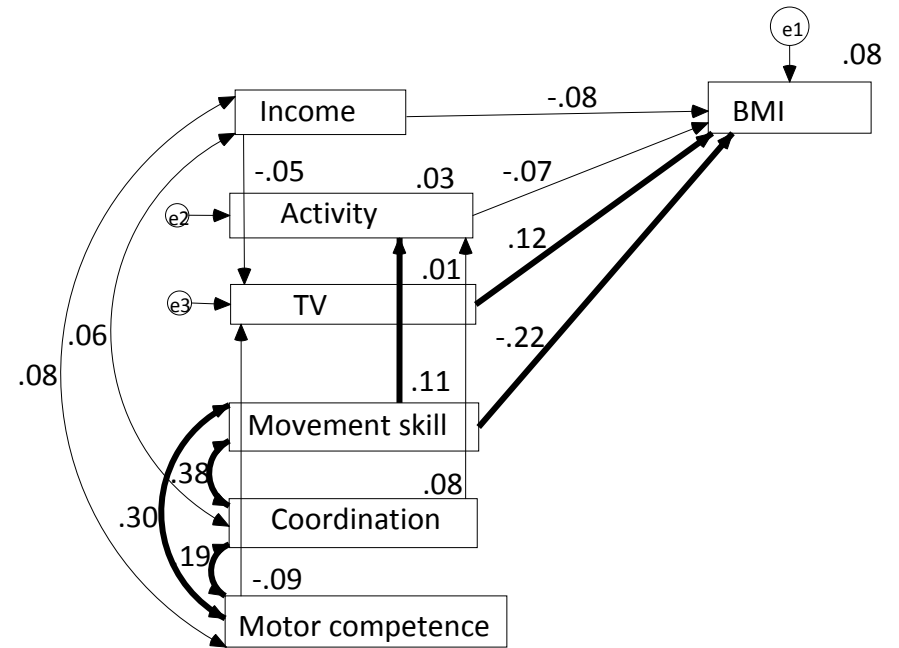


Figure 17. Estimated relations among predictors of BMI at 10 years for all paths included in the final model for males and females.

Standardised coefficients are reported. Model fit  $\chi^2$  (df=40) = 47.820  $p$ =.185; RMSEA=.012 [CI .0 - .023]; CFI=.983; TLI=.976.

TV = time spent watching television and playing computers, Motor competence = MAND NDI score, Coordination = coordination and developmental progress, and BMI = child's BMI.

$p > .05$  (---),  $p < .05$  (—),  $p < .001$  (—).



### **Follow-up age 14 years.**

At age 14 years a much more complex pattern emerged. Attitudes to physical activity (encouragement, effects, and importance of physical activity; physical education) played neither a direct nor indirect role with BMI but seemed to be predicted by self-perceptions, actual motor competence, aerobic fitness, physical activity level, and even the likelihood to make excuses. The school environment also did not play either a direct or indirect role with BMI nor have any relationships with other variables.

Self perceptions of athletic competence, physical appearance and global self worth did have a relationship with BMI however they were the result of other variables. For example, those individuals who were active and had high motor competence scored highly in the athletic domain, or those with high BMI scored low in the appearance domain. Further, poor model fit and errors were reported with these variables present in the model. As a result these domains were removed from the model.

As previously discussed, diet was represented by a derived score for fat intake, fruit and vegetable intake, all self-reported by the child. The derived fat score was not predictive of BMI either directly or indirectly which may relate to the crudeness of this particular measure. Fruit and vegetable intake also did not play a role in predicting BMI. However, the modelling process indicated that other behaviours were predictive of fruit and vegetable intake. Specifically, increased income, motor competence and physical activity all predicted increases in fruit intake. Fruit intake reduced with increased screen time, while vegetable intake reduced with the later stages of pubertal development.

Remaining in the final model were physical activity level, aerobic fitness, motor competence, sedentary activity (screen time), excuses, puberty and mother's BMI in the model. The correlation matrix is reported in Table 77, unstandardised estimates in Table 78, and final model in Figure 18 .

The significant gender differences include: the paths between aerobic fitness and BMI; puberty and aerobic fitness; mother's BMI and BMI, and income and screen time; intercepts for motor competence, physical activity, and screen time; mean for puberty; and error variances for physical activity, aerobic fitness, motor competence and mother's BMI.

Overall mother's BMI and aerobic fitness were the strongest predictors of BMI. Mother's BMI was predictive of the adolescent's motor competence and screen time. Aerobic fitness was predictive of physical activity level and motor competence, and was affected by pubertal development. Screen time had an inverse relationship with BMI and also predicted motor competence. An adolescent's motor competence was inversely related to the number of excuses they made to avoid more physical activity. SES as measured by family income, continued to provide an influence, particularly in respect to mother's BMI, excuses, motor competence and BMI. SES was only related to screen time in males. Overall, these predictors of BMI accounted for 24% and 21% of BMI variance for males and females respectively.

Table 77

*Summary of Correlations, Means and Standard Deviations for Variables used in the Final Structural Equation Model at age 14 years*

	1	2	3	4	5	6	7	8	<i>n</i>	<i>M</i>	<i>SD</i>
1. Income	-								1,338	0.81	0.40
2. Mother's BMI	-.088**	-							1,071	26.4	6.1
3. Screen time	-.049	.067*	-						1,397	3.5	1.2
4. Excuses	-.142***	.062*	.040	-					1,357	12.8	7.0
5. Aerobic fitness	.056*	.016	-.030	-.134***	-				1,322	111.0	29.0
6. Physical Activity	.002	.009	-.064	-.120**	.256***	-			604	10.7	3.9
7. Motor competence	.105***	-.115***	-.087**	-.159***	.181***	.003	-		1,384	97.3	17.3
8. Puberty	-.024	-.007	-.015	-.007	.166***	-.049	-.067*	-	1,082	2.9	0.8
9. BMI	-.108***	.331***	.055*	.156***	.146***	-.097*	-.137***	.121***	1,403	21.4	4.2

*Note.* Aerobic fitness = PWC 170, Physical Activity = mean daily step count, Motor competence = MAND NDI score, Puberty = Tanner stage of pubic hair development, and BMI = child's BMI.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ , two-tailed.

Table 78

*Summary of Unstructured Regression Weights for Variables used in the Final Structural Equation Model at age 14 years*

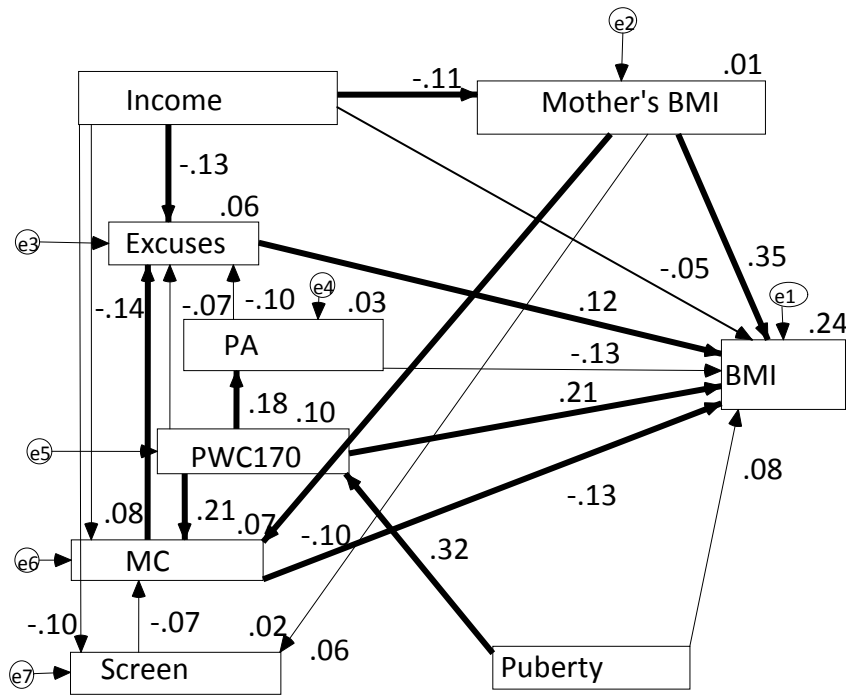
			Constrained equal			Freely estimated					
						Male			Female		
			P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>
M BMI	<---	Income	-1.605	.464	***						
Screen	<---	M BMI	.012	.006	.040						
PWC170	<---	Puberty				11.962	1.028	***	2.250	.954	.018
Screen	<---	Income				-.311	.115	.007	.024	.118	.838
MC	<---	M BMI	-.309	.083	***						
MC	<---	Income	3.593	1.173	.002						
PA	<---	PWC170	.026	.006	***						
MC	<---	PWC170	.127	.018	***						
MC	<---	Screen	-1.046	.380	.006						
Excuses	<---	Income	-2.230	.479	***						
Excuses	<---	PA	-.159	.073	.029						
Excuses	<---	PWC170	-.017	.007	.021						
Excuses	<---	MC	-.054	.011	***						
BMI	<---	Excuses	.072	.015	***						
BMI	<---	Income	-.589	.267	.027						

			Constrained equal			Freely estimated					
						Male			Female		
			P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>	P.E.	S.E.	<i>p</i>
BMI	<---	PWC170				.030	.005	***	.065	.007	***
BMI	<---	PA	-.126	.041	.002						
BMI	<---	M BMI				.261	.023	***	.167	.022	***
BMI	<---	Puberty	.442	.157	.005						
BMI	<---	MC	-.030	.006	***						

*Note.* P.E. = parameter estimate, S.E. = standard error, M BMI = mother's BMI at follow-up 14 years, Screen = total screen time hours/week, PWC 170 = aerobic fitness measure, Puberty = Tanner scale for pubic hair, MC = motor competence measured by MAND NDI score, PA = physical activity measure pedometer mean daily step count (x1000), and BMI = child's BMI.

\*\*\*  $p < .001$ .

Male



Female

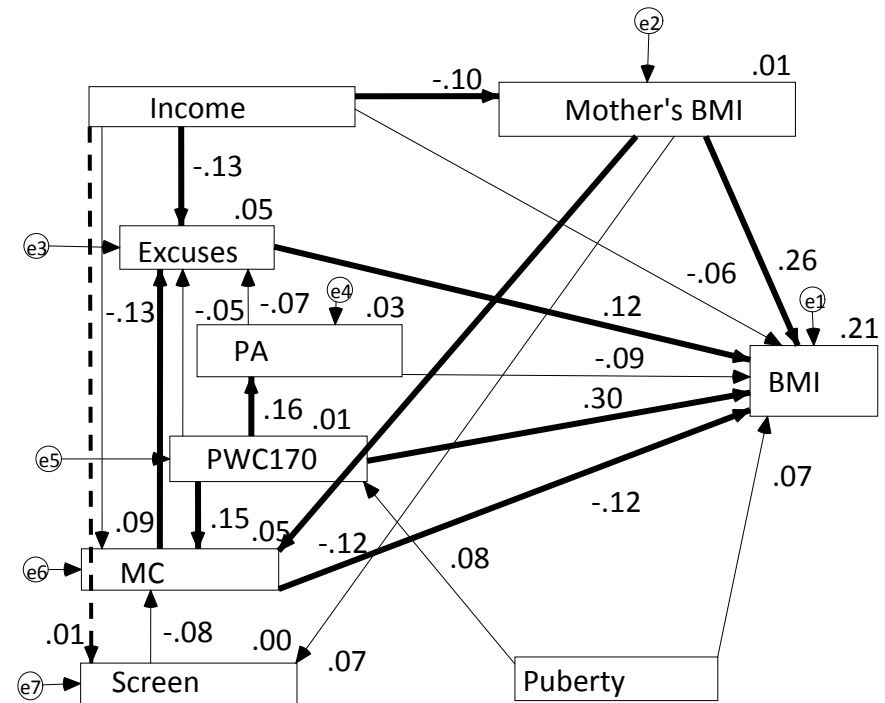


Figure 18. Estimated relations among predictors of BMI at 14 years for all paths included in the final model for males and females.

Standardised coefficients are reported. Model fit  $\chi^2$  (df=57) = 59.487  $p$ =.385; RMSEA=.006 [CI .0 - .018]; CFI=.995; TLI=.992. Screen = total screen time hours/week, PWC 170 = aerobic fitness, Puberty = Tanner scale for pubic hair, MC = Motor competence measure MAND ND score, PA = physical activity pedometer step count (x1000), and BMI = child's BMI.

$p > .05$  (---),  $p < .05$  (—),  $p < .001$  (—).

## **Summary**

In summary, these results provided a comprehensive review of obesogenic variables and their respective roles with adolescent obesity. Descriptive analysis provided evidence for common relationships between gender and among weight status groups. The LMM permitted a more robust assessment of each obesogenic variable and their relative positive or negative contribution to BMI. Finally, SEM explored possible interrelationships between obesogenic variables, and how together they might influence BMI at each follow-up.



### Ten years

"... Early adolescents experience intense and unstable feelings. At least in part because of changes in hormones, they bounce from being silly, happy and exuberant to withdrawn, grouchy and sad. They feel embarrassed very easily and are overly concerned with how they look ..."

By Lynn Blinn Pike



## Chapter Five

### Discussion

This research confirms the multi-faceted nature of obesity, with many complex interactions of family, community, environment, social demographics, genetics, biology, behaviour, diet and psychology, all intricately interwoven in cause, effects and feedback loops. These interrelationships are shown to change over time, differ between boys and girls, and the pathways for normal weight, overweight and obese individuals are distinctly different from birth to adolescence.

Within the rich database of the Western Australian Pregnancy (Raine) Cohort numerous individual, behavioural and environmental variables were investigated with respect to weight status at age 14 years. This investigation involved four main stages: first to validate the adiposity proxy to classify children's weight status at age 14 years, secondly to assess gender and weight status group differences in key variables at each follow-up; thirdly to develop a longitudinal model and assess obesogenic variables and their relationship to weight status at 14 years, and lastly to combine factors into a structural equation model to explore complex interrelationships.

Discussion in this chapter moves away from the simplistic notion of individual causes of obesity, to a multi-faceted perspective with relative contributions. The discussion begins with an overview of adiposity measures, followed by a synopsis of obesogenic variables guided by Bandura's model of individual, behavioural and environmental factors. Lastly, findings are summated according to the longitudinal model of BMI, critical time periods, interrelationship modelling, and gender and weight status group differences.

## Adiposity Measures<sup>18</sup>

The similarities between common adiposity measures of weight, hip girth, waist girth, and the indices WHtR, BMI and WHR, were investigated in a large sample of adolescents using conventional Pearson Product Moment Correlation, the Bland-Altman method (Bland & Altman, 1986) and the Bland regression method (Bland, 2004). In addition, different weight status categorisation methods (Cole et al., 2000; Kahn et al., 2005; McCarthy & Ashwell, 2006; Taylor et al., 2000) were evaluated.

As demonstrated the indices of WHtR and BMI were strongly correlated, with the Bland-Altman method and Bland regression method confirming results. The sub-grouping of the sample by gender and three weight status categories showed that both the BMI (Cole et al., 2000) and WHtR (Kahn et al., 2005) cut-offs provided similar groupings, which were strongly correlated and statistically significant. The BMI cut-offs (Cole et al., 2000) identified slightly fewer individuals as normal weight, more individuals as overweight, and less individuals as obese compared to the WHtR cut-offs (Kahn et al., 2005). Although the two category groupings (McCarthy & Ashwell, 2006) for BMI had a better Kappa value with WHtR, the number of differently categorised individuals was much higher.

Most critically, this analysis examined the similarity and inter-changeability of adiposity measures. Although the Bland-Altman method with z-scores reported strong agreement between some measures, the more appropriate statistical tool, Bland regression method, found large variation in limits that were unacceptable clinically. The adiposity measures examined in this study, although sharing strong relationships, appeared to provide different information. These findings highlight the need to interpret with caution most field-based measures of obesity.

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<sup>18</sup> From "A comparison of field measures of adiposity among Australian adolescents from the Raine Study," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2010, *Malaysian Journal of Sports Science and Recreation*. Copyright 2010 by MJSSR. Adapted and reprinted with permission.

Therefore, based on these findings and the availability of BMI measures, BMI was used as a proxy for adiposity (Chivers et al., in press). Despite some limitations as a measure of fatness (Dehghan et al., 2005; Freedman et al., 2007; Kahn et al., 2005; McCarthy & Ashwell, 2006; Taylor et al., 2002), BMI is still considered a reasonable marker (Bua et al., 2007; Cole et al., 2000; Dehghan et al., 2005; Guo & Chumlea, 1999; Katzmarzyk et al., 2004; Krebs et al., 2003; Taylor et al., 2002; Tremblay et al., 2002; WHO, 2006), and highly reliable (Bua et al., 2007; Guo & Chumlea, 1999; Lindsay et al., 2001).

## Individual Factors

### **Body Mass Index (BMI) - trajectories and adiposity rebound<sup>19</sup>.**

The BMI trajectories followed a distinct pathway from birth to 14 years for individuals within different weight categories. As suggested by others (Rolland-Cachera et al., 2006; Small et al., 2007), the timing of the adiposity rebound can be interpreted as a marker for later obesity. These results show more clearly than those reported by Rolland-Cachera (1984; 2006) the distinct and significantly different pathways followed by the three weight status groups for both the raw means and modelled data (LMM). There were statistically different pathways, in particular between the normal weight group compared to the overweight and obese groups. Major limitations were the absence of data at ages 4 and 5 years, and the smaller sample at age 2 years, which were reflected in the lack of statistical difference between the overweight and obese weight status groups only, although pictorially a difference was shown (Figure 2). However adiposity rebound has been reported in regards to macronutrients with a smaller sized sample ( $n=112$ ) and time points 0.8, 2, 4, 6 and 8 years (Rolland-Cachera et al., 1995). We also found a statistical difference in BMI at nadir, with the normal weight group having a lower BMI at rebound compared to the overweight and obese groups.

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<sup>19</sup> From "Longitudinal modelling of body mass index from birth to 14 years," by P. T. Chivers, B. Hands, H. E. Parker, L.J. Beilin, G.E. Kendall, and M. Bulsara, 2009, *Obesity Facts*, 2, 302-310. Copyright 2009 S. Karger AG, Basel. Adapted and reprinted with permission.

The limitation in the number of data points up to age 6 years and in particular between age 3 and 6 years may play a significant part in the adiposity rebound age and BMI nadirs reported. Although the ages for the obese and normal weight groups appear clear, those for the overweight group are not. The STRIP (Special Turku Coronary Risk Factor Intervention Project for Children) study reported by Lagstrom and colleagues (2008), collected annual measurements until age 13 years, and reported adiposity rebound at 4.3 years for overweight boys and 3.8 years for overweight girls. In reviewing this present study's BMI trajectory plots (Figure 2), it is likely that there may have been a lower BMI point somewhere between assessments at 3 and 6 years of age. Irrespective, it is clear that the overweight adiposity rebound occurs at some point at, or after age 3 years, and before age 6 years in this cohort. This is well before that of the normal group, and those originally reported by Rolland-Cachera and colleagues (1984), yet similar to more recent studies (Lagstrom et al., 2008; Rolland-Cachera et al., 2006).

This identified timing of adiposity rebound supports others who found that the earlier the occurrence of the adiposity rebound the higher the BMI with age (Lagstrom et al., 2008; Rolland-Cachera et al., 1987; Rolland-Cachera et al., 2006). However, unlike Rolland-Cachera and colleagues (1987), children in the present study were pre-classified into weight status at age 14 years and then their respective mean value for adiposity rebound in the earlier years was calculated. Our methodology found that the higher the BMI at year one, the earlier the age of rebound, which may be reflective of children with high centile BMI or upward centile crossing according to BMI growth charts (Cole, 2004). Like others, we believe the occurrence of the earlier adiposity rebound is a marker for early determinants or mechanisms of obesity (Cole, 2004; Dubois & Girard, 2006; Rolland-Cachera et al., 1999; Small et al., 2007).

Following this line of thought, investigation of early feeding patterns was conducted (Chivers et al., 2010). A comparison of each early feeding group trajectory (Figure 3) showed a difference in BMI with those breastfed for more than 4 months having

a lower BMI at 14 years. Although a statistical difference between raw BMI with adiposity rebound measures for age or BMI at nadir were not found, predicted BMI based on the whole sample population accounting for age, gestational age, gender and weight status showed that breastfeeding plays an important role in the timing of the adiposity rebound. Both breastfeeding and the age other milk was introduced were important in the determination of BMI at adiposity rebound. These results support early feeding literature (Oddy et al., 2006b; Singhal et al., 2007) that suggest bottle feeding results in accelerated weight gain in the infant, compared to their breastfed counterparts, as depicted in the adiposity rebound nadir results. Early feeding patterns appear to be one of a number of early determinants or mechanisms for later obesity. These include environmental, behavioural and individual factors (such as diet, physical activity and genetics), that may program later weight status (Hallal et al., 2006; Skinner et al., 2004; Small et al., 2007). To date however the value of identifying the timing of adiposity rebound for use in a clinical setting is yet to be confirmed (Dietz, 2000), although it could be used as a tool to alert health professionals to children at risk.

Although these findings on adiposity rebound are similar to others (Lagstrom et al., 2008; Rolland-Cachera et al., 2006), the meaning of the distinct peaks and troughs in BMI in relation to child behaviour is as yet unclear. Analysis of the height and weight data found that the changes observed in BMI were principally related to changes in weight and not height. This can be accounted for by height and weight growth spurts that occur during childhood (Botton et al., 2008). While height trajectories were very similar for all three weight categories, the overweight and obese groups were gaining weight at a faster rate than their normal weight counterparts. This was most pronounced by 3 years of age, although statistically significant from age one. Adiposity rebound for the obese group occurred at around 2 years, the same as the beginning of the second critical period for adiposity proposed by Botton and colleagues (2008).

The longitudinal model of trajectories of BMI was different between males and females, and different for each weight category (Figure 14). This difference was in

both the rate of change over time (acceleration) and BMI at birth. The interaction effects showed that within the weight categories the pathways for males and females were significantly different, with females slightly leaner than males when young, with a cross over in later childhood / early adolescence. The obese group increased their BMI over time at a faster rate, and this difference began from birth. This analysis, which includes more frequent collection points and with a larger sample, is similar to the results reported by Blair and colleagues (2007) for their New Zealand cohort. These results indicate that the preschool years (before age 6 years) is a critical time period for the development of obesogenic behaviours.

The overlay of mean age-adjusted BMI at each follow-up on the modelled BMI trajectories provided an overview of goodness of fit of our proposed model (Figure 14), with residual diagnostics supporting this (Appendix E). The tight clustering of mean data points in the first few years again highlights the need for greater in-depth analysis of this important period with more frequent data collection points, perhaps at three-monthly intervals.

It is therefore apparent that in the Raine Study cohort, the increased rate in weight gain accounts for later adiposity. The underlying causes of the accelerated weight gain are still unclear, although early feeding appears to be an important factor. An examination of growth patterns may assist in the understanding of the development of obesity (Heude et al., 2006). Parents often observe children undergoing increased hunger and appetite, weight gain, followed by decreased appetite and height growth, or episodes of spurt and lag (Wilson, 1986). One explanation may be that established food behaviours do not follow the pendulum return of decreased appetite during growth for the overweight and obese groups. Physical and social aspects of the home environment may also influence food choices based on types of food available, parent food behaviours and feeding practices (Bryant, Ward, Hales, Vaughn, Tabak, & Stevens, 2008). There may also be an as yet determined developmental switch triggered by the environment, that is present or absent in the overweight and obese (Blair et al., 2007; Eriksson, Forsen, Tuomilehto, Osmond, & Barker, 2001), which may be related to epigenetic

mechanisms (Walley et al., 2006; Waterland et al., 2008). Genetically, a common variant in the FTO (fat mass and obesity associated) gene has been identified that increases the odds of obesity (Frayling et al., 2007). In addition, the level of physical activity and sedentary past-times could be important. Dubois and Girard (2006) concluded that influences of behavioural and social factors were critical to the possible onset of obesity in the early pre-school years. Regardless of cause, rapid weight gain after age one is associated with adverse health effects (Eriksson et al., 2001), and this critical period of development requires intensive investigation in regards to obesity.

### **Infant feeding<sup>20</sup>.**

Early infant feeding investigations using LMM indicated that the age breastfeeding stopped and the age other milk was introduced should be investigated simultaneously. Interestingly, those individuals who were breastfed for more than 4 months, but introduced to other milk at or before 4 months had the highest increase in BMI. Over the time period of interest, those individuals who were breastfed for more than 4 months and started other milk after 4 months did not increase their BMI.

Like Blair and colleagues (2007) for their New Zealand cohort, these data concur that the preschool years are a likely critical time period for the development of obesity (Chivers et al., 2010) and that early feeding plays an important role. These early feeding results extend the findings of Burke and colleagues (2005) on this cohort which showed higher BMI at age 8 years in children breastfed for less than 4 months. Cross sectional analysis of early infant feeding and weight status groups determined at 14 years showed that the proportion of overweight and obese was higher in those who had mixed feeding patterns. A difference was observed in BMI

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<sup>20</sup> From "Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study)," by P. T. Chivers, B. Hands, H. E. Parker, M. Bulsara, L.J. Beilin, G.E. Kendall, and W.H. Oddy, 2010, 34, e1-8, *International Journal of Obesity*. Copyright 2010 Nature Publishing Group. Adapted and reprinted with permission.

peak at age 1 year for the different weight status groups, supporting the theory that early introduction of formula feeding is related to growth acceleration, and overweight or obesity (Oddy et al., 2006b), whereas exclusive breastfeeding meets the individual nutrient and energy requirements of each child (Butte, Lopez-Alarcon, & Garza, 2002). Modelling indicated that breast feeding for longer than 4 months, but introducing other milk before 4 months had the largest increase in BMI over time. This is supportive of the theory that bottle feeding reduces self-regulation as parental behaviour may override satiety (Gillman et al., 2001), whereas breast feeding provides protection against overfeeding (Oddy, Scott, Graham, & Binns, 2006c). Further, the suggestion that breastfeeding develops behavioural mechanisms for food acceptance and control of energy intake (Savage et al., 2007) is supported by the finding that BMI differences between early infant feeding groups remains over time until at least 14 years of age.

Recent reports have thrown into question whether the association between breast feeding and subsequent obesity is causal or in fact uncontrolled bias. This bias has been suggested to be mediated by selection and confounding socioeconomic environmental issues that determine parental family feeding and physical activity (Kramer et al., 2009; Michels et al., 2007). However, we were able to show that breastfeeding remained significantly associated with adolescent BMI when adjusted for maternal education, income and mother's pre-pregnancy BMI (Chivers et al., 2010). Hence, with consideration of some SES confounding, the case is strengthened for the important role early infant feeding has on adolescent obesity.

### **Diet.**

Gender differences in food choices in young adults (aged 17 to 30) (Wardle, Haase, Steptoe, Nillapun, Jonwutiwes, & Bellisle, 2004) were also found in the present study's adolescent group. Males reported higher intake of fats while females reported higher intakes of vegetables. However, both fruit and fat intake were not found to be associated with weight status at 14 years, nor vegetable intake for males. These findings support Hu's (2008) review of evidence which found that no



single dietary factor had a large effect on BMI. In contrast, there were weight status group differences in vegetable intake for females, and longitudinal modelling found that those who consumed fewer vegetables had a larger increase in BMI. This is similar to findings with adult women, where those who increased their intake of fruit and vegetable the most, decreased their risk for obesity (He et al., 2004).

Interrelationship investigations found that although adolescent fruit and vegetable intake was neither directly nor indirectly associated with BMI, there were relationships with other variables. Overall, healthy behaviours (e.g. being physically active) were related to increased fruit intake, while late stages of puberty were related to a decrease in vegetable intake. These relationships are not unusual, as in young adults, particularly women, healthy food choices are thought to be associated with a more positive attitude towards health, with these individuals more likely to put these beliefs into action (Wardle et al., 2004).

Surprisingly, fat intake was consistently unrelated to BMI. This may be due to measures used for fat intake in this study, which were not those typically reported, nor a complete representation of actual dietary fat intake. Dietary pattern analysis is becoming more popular (Ambrosini et al., 2009a; Hu, 2008c), although not available for analysis in the present study, it may be a more appropriate indicator for diet (Ambrosini et al., 2009a). However, the lack of association for fat intake with BMI may be related to specific dietary reporting issues. It is well known that individuals who are overweight or obese tend to under-report true intake because they are being recorded, are inaccurate in detailing items and amounts, and / or reluctant to admit what they are eating (Lissner, Heitmann, & Bengtsson, 2000). Closely associated is the issue of portion size (Rennie et al., 2005; Steenhuis & Vermeer, 2009), information which was not available in the present study, although the use of such data can be limited and restricted by many methodological issues (Hu, 2008c).

### **Developmental milestones and motor competence.**

Early childhood is a particularly important developmental period for autonomous self, social and motor competence (Houck, 1999). Evidence from parent reported developmental milestones from ages 1 to 3 years suggested a weak relationship between the locomotor milestones of stand and totter and weight status.

Interrelationships investigations found that these early milestones were the result of motor competence and did not have any causal pathways to other variables, nor BMI at age 1 year. Similarly, the Denver II Assessment results, over similar follow-up years, did not find any significant weight status differences, although individuals who were overweight or obese at 14 years, were more likely to have been rated *uncooperative* or *suspect* in these early years. These results are similar to Jones and colleagues (2009) who found no relationship between motor development, perceived competence and obesity in 2-6 year olds.

The IMQ results demonstrated some relationships with the overweight and obese individuals who were yet to master some fine motor skills. Interestingly, talking, which could be considered a fine motor skill, was delayed in those individuals who were overweight or obese at 14 years. Toddler speech may reflect a complex cognitive process and has been shown to predict a child's capacity to self-regulate behaviour by school age. Further, developmental competence, as measured by the IMQ, precedes self concept and is enhanced by social competence (e.g. speech) (Houck, 1999).

Findings for locomotor skills suggest a possible link with later obesity. Locomotor skills in individuals overweight or obese at 14 years were more likely to be judged *not like their peers* for the IMQ, and *below average* for parent reported locomotor skills at 10 years. Longitudinal modelling showed a significant negative influence with BMI, with an increased BMI for those with poor locomotor skills. Locomotion, such as walking, is a major developmental milestone occurring during the first two years of life (Malina, 2004). Mastery of locomotor skills may be related to adolescent weight status through future physical activity levels, attitudes and self

concept. Certainly, evidence suggests that intervention at an early age for those with poor motor competence should occur (Houck, 1999), as pre-school children with poor coordination are less physically active (Williams et al., 2008). These early interventions may assist in increasing perceived competence and self concept, and help combat the suggested reciprocal relationship between motor competence and weight status (Okely et al., 2004).

Overall, these earlier motor skill findings were supported by the more accurate (reliable and valid) MAND results at both follow-up ages 10 and 14 years. A clear difference was found, with higher motor competence associated with lower BMI. Importantly, at 14 years, there were significant interrelationships, with low motor competence individuals more likely to make excuses for not increasing physical activity levels and have high screen use, the latter being similar to that found by Graf and colleagues (2004).

From a dynamic systems perspective, these factors probably relate to the specific environmental influences necessary for motor development, namely prior and new movement experiences (Malina, 2004). Those with better skills find it easier, and are more likely to engage in physical activity (Williams et al., 2008). However, although it has been shown that higher physical activity levels provides opportunities to improve motor skills (Reilly et al., 2006; Williams et al., 2008) we found no relationship between physical activity (step count) and motor competence. It is important to note that in contrast to these earlier studies, the physical activity measures differed, and the Raine Study cohort was older. Cohort age and measure of physical activity are important factors to consider when making comparisons (Williams et al., 2008), with differences across age having been shown in this cohort (Hands, Chivers, Parker, Beilin, Kendall, & Larkin, 2010b). Similar to Graf and colleagues (2004) we found a positive relationship between motor competence and aerobic fitness, concurring with a cross-sectional analysis using the Raine Study cohort (Hands et al., 2008). Perhaps the type of measure for physical activity is the key. In respect to the directionality question posed by Graf and colleagues (2004) “do overweight and obesity lead to poor physical performance or

is it the other way around?" (p. 25), the SEM results suggest motor competence predicts BMI. Like others (Williams et al., 2008), we demonstrate that a sedentary lifestyle (as measured by screen time) is interrelated with low motor competence and unhealthy weight.

### **Physical fitness.**

Physical fitness relates to an individual's ability to perform physical activity (Caspersen et al., 1985; Must & Tybor, 2005). The Raine study included measures of absolute aerobic fitness (PWC 170), muscle endurance (curl-ups), upper body strength (basketball throw), and flexibility (sit and reach, shoulder stretch) at 14 years. Gender differences were reported across all variables with males outperforming females in absolute aerobic fitness and endurance, while females had greater flexibility. Except for absolute aerobic fitness, no weight status group differences were found. The longitudinal model showed that the number of curl-ups and basketball throws were related to BMI. Those with higher BMI achieved fewer curl-ups, and this probably relates to actual fat mass restricting movement (Hands et al., 2008). Adolescents with higher BMI scored better in basketball throws and this may be related to the stability provided by body mass to perform this skill (Hands et al., 2008) or more muscle mass.

For both males and females, absolute aerobic fitness was greatest for obese, followed by those overweight, then normal weight. However, there was not a significant difference between the normal weight and overweight males.

Longitudinal modelling replicated this trend with a positive relationship between absolute aerobic fitness and BMI. These results are not surprising and concur with others (Goran et al., 2000; Rowland, 1991). Obese individuals do not necessarily have an impaired cardio-respiratory response to exercise (Goran et al., 2000), but rather that increased fat levels are associated with increased cardiopulmonary exercise capacity (Rowland, 1991), particularly in a weight supported bike ergometer test. However, in non-weight supported activities (e.g. treadmill running) obese individuals require a greater proportion of their aerobic capacity to

participate (Goran et al., 2000), that is, for these tasks their *functional* fitness decreases with excess body fat (Rowland, 1991) and they are likely to find locomotor tasks such as running more difficult.

Pubertal stage has a confounding effect on the measure of aerobic fitness. ANOVA and SEM analyses both demonstrated a strong relationship between stage of pubic hair development and absolute aerobic fitness, with BMI. During puberty, increased body fat (Sloboda et al., 2007) and changes in physical characteristics (e.g. lean body mass, skeletal mass and body fat) occur (Hayward, 2003; McCarthy, Cole, Fry, Jebb, & Prentice, 2006). Regardless, interrelationship investigations at 14 years demonstrated that each of these factors was independently related to BMI both in an indirect and direct way.

### **Puberty.**

Adolescence has been identified as a critical period for the development of obesity (Dietz, 1994), but also coincides with the timing of puberty. With increasing rates of childhood obesity, questions have been raised as to whether increased adiposity is responsible for earlier puberty, although evidence suggests an independent decline is also present (Aksghlaede et al., 2009). However, it is well recognised that puberty is associated with increases in fat mass and muscle mass (Hayward, 2003; McCarthy & Ashwell, 2006; Sloboda et al., 2007).

In this study the overall timing of puberty as measured by first menstruation (menarche) and the Tanner self-ratings for pubic hair and breast development (Tanner, 1962) did not have a direct influence on individual BMI. The cross-sectional lack of association between BMI and age at menarche concur with previous findings in this cohort (Sloboda et al., 2007). However longitudinal modelling found that overweight females with later menarche had lower BMI, which may indicate some confounding with respect to the increase in body fat usually associated with menarche in females (McCarthy et al., 2006; Sloboda et al., 2007). Only female breast development was significantly associated with weight

status, with more obese and overweight individuals being more physically mature at 14 years. Longitudinal modelling of pubic hair development showed that those individuals who were obese and pre-pubertal had lower BMI, similar to the age at menarche finding.

Generally there was some evidence that individuals with higher BMI experienced pubertal changes earlier, which is consistent with previous findings (Styne, 2004). In respect to interrelationships at 14 years, puberty measured by pubic hair development was predictive of aerobic fitness (e.g. PWC 170) and also BMI, and is probably related to changes in physical characteristics (e.g. lean body mass, skeletal mass and body fat) (Hayward, 2003; McCarthy et al., 2006).

## **Behavioural Factors**

### **Physical activity.**

Physical activity is used to describe a body movement or behaviour of varying amounts as described by frequency, intensity, time and type (Caspersen et al., 1985). Physical activity can be measured in a number of ways, for example accelerometers, pedometers, heart rate monitors, direct observation and self report, with each method having its advantages and disadvantages (Molnar & Livingstone, 2000; Must & Tybor, 2005). In addition, attitudes and values to physical activity are seen as modifiable correlates of physical activity, such as encouragement, effects, importance, competence, excuses, intention, enjoyment, and encouragement (Sallis et al., 2000).

Historically, children's development (and activity behaviour) is facilitated by positive early experiences, and can be delayed by negative experiences (Gandour, 1989). In the preschool years, early physical activity behaviours were assessed by how often children were taken to the park or playground (age 1 to 3 years).

Generally males, compared to females, were taken to the park or playground more frequently, corresponding to participation levels in organised sport at age 6 years, but not later. It is well recognised that males tend to be more active than females

from an early age (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006) which may explain why caregivers are more likely to take males to the park to vent some of that excess energy. In the later years (ages 8 and 10 years) males continued to have higher activity levels compared to females. Females though had higher levels of participation in organised activity, but this probably related to the types of activities defined by this survey question, namely music, dancing, kindy gym and other clubs. These gender differences are consistent with other studies (Dollman & Lewis, 2009; Hands et al., 2008; Hands et al., 2004; Martin et al., 2009; Thompson et al., 2009) and are probably also related to attitudes and values to physical activity (Li et al., 2005; Murcia et al., 2007; Sallis, 2000).

Adolescents who were obese at age 14 years were *never* or *seldom* taken to the park or playground as young children. Their parents reported less participation in organised activity at age 6 years, they comprised the highest proportion of inactive and lowest proportion of active at age 8 years, and had the lowest scores for light, moderate and vigorous activity at age 10 years. This appearance of tracking is supported by a concurrent study (Hands et al., 2010b) which depicts tracking of physical activity based on these variables in a longitudinal SEM model. These results support others who have highlighted the importance of early childhood activity participation in the development of physical activity behaviours in later childhood, adolescence and into adulthood (Janz, Dawson, & Mahoney, 2000; Tremblay & Willms, 2003).

In these early years, parents also play a key role in their child's activity levels. The importance of parents as role models has been shown by others (Dollman & Lewis, 2009; O'Dea, 2003a). In the present study, at age 14 years, normal weight children had more active parents who also *helped them to exercise*. Interestingly, LMM found that those individuals who reported higher levels of encouragement from their parents had higher BMI, contrary to the positive role of parent encouragement in overcoming barriers to being physically active (O'Dea, 2003a). These differences may relate to the style of encouragement provided. Evidence in the physical education setting has shown that a style that develops structure,

autonomy support and involvement has positive outcomes, versus a controlled motivation with less autonomy (often used in the teaching setting for those less motivated) (Ntoumanis & Standage, 2009).

Overall pre-school physical activity behaviours did not seem to play an important role when considered with the longitudinal model of BMI, nor did it have interrelationships with other obesogenic variables. However, at age 10 years, a relationship was found between an increasing number of activities and decreasing BMI. Further interrelationship modelling indicated that physical activity in childhood operated indirectly by displacing screen time. Investigations on the displacement hypothesis have been mixed (Hands et al., 2010b; Hohepa et al., 2009; te Velde et al., 2007), however work with this cohort suggests early childhood was an important period for the development and establishment of healthy behaviours.

In this study, physical activity level was measured by pedometer step counts over a minimum 4-day period at age 14 years. Like others (Olds et al., 2004; Steinbeck, 2001), males had higher levels of physical activity (mean daily step count) than females overall, during the week and on weekends. The higher step counts are thought to be generated by being more involved in outdoor play and leisure activity, with outdoor activity associated with higher levels, intensity and frequency of physical activity (Klesges, Eck, Hanson, Haddock, & Klesges, 1990).

An association between physical activity and obesity is not consistently found (Hands et al., 2008; Thompson et al., 2009). In this study, only females showed a significant weight status difference with those who took more mean daily steps having lower BMI. There were no weight status group differences for males, with the overweight group having higher mean daily steps than the normal weight group. Though not significant overall, LMM analysis found subgroup interactions between the obese group and mean daily step count, with the more active obese individuals having a lower BMI. This suggests that physical activity may assist in preventing further weight gain (Fogelholm & Kukkonen-Harjula, 2000), or protect



against accelerated weight gain (Steinbeck, 2001). Interrelationship modelling also demonstrated an inverse relationship between physical activity at ages 6, 8, 10 and 14 years, supporting the inverse relationship between physical activity and BMI reported in young adults (Dragan & Akhtar-Danesh, 2007).

A relationship was found between physical activity and screen time at ages 6 and 8 years, but not at ages 10 and 14 years. In associated investigations with this same cohort sample (Hands et al., 2010b), a longitudinal SEM model demonstrated strong interrelationships between physical activity levels and screen time in the early years, indicating that during the early years screen time may displace physical activity. Evidence of physical activity level tracking from childhood into adolescence in this cohort study, along with other studies (Janz et al., 2000; Kelder, Perry, Klepp, & Lytle, 1994; Telama, 2009), provides additional support that early childhood may be a critical time for the establishment of more physical and less sedentary behaviours (Hands et al., 2010b).

In the present study, the direct influence of physical activity attitudes and values on weight status was examined along with the indirect influence through physical activity behaviour. Significant gender differences were found, consistent with findings for self-perception (Li et al., 2005; Murcia et al., 2007) and physical activity (Sallis et al., 2000). Overall males were more positive towards participation, while girls were more likely to make excuses to avoid participation. Weight status group differences were found, but again, similar to self-perceptions, healthy weight individuals had more positive responses and depicted greater confidence in being physically active. Recently Schmalz (2010) found in adults that perceptions of body weight were predictive of physical activity participation levels. More specifically, weight related stigma had an inverse relationship with perceived physical activity competence, which was also related to BMI. Overall in the present study, longitudinal modelling found either weak sub-group or no relationships with BMI, and similarly in interrelationship modelling. However, those with higher BMI were more likely to make excuses. Here, as the number of excuses increased, so did BMI, which is consistent with findings on perceived barriers and intentions to be active

(Sallis et al., 2000) and weight related stigma (Schmalz, 2010). The low active (step count) and low motor competent (MAND) groups made more excuses for not participating in physical activity, providing further support that positive attitudes and values increase with physical activity participation (Li et al., 2005; Murcia et al., 2007), which in turn is related to healthy body weight.

Parent influences had an association with physical activity attitudes and values, although not always in the direction expected. These inconsistencies were also reported by Sallis and colleagues (2000) in their review of physical activity correlates. The involvement of peers was also significant. Individuals with active friends had lower BMI, supporting Tergerson and King's (2002) study that showed having a friend to exercise with was important. Peer participation was not as important for males as it was for females, who valued the social aspect of physical activity participation. This was particularly true for overweight females aged 8 to 16 years (Zabinski, Saelens, Stein, Hayden-Wade, & Wilfley, 2003). Here, overweight children, particularly females, were more likely to report barriers to physical activity which included negative perceptions about their body; lack of interest, skills, facilities or knowledge; and negative social perspectives (e.g. lack of peers to be active with, teasing, peers not active). Overall, the shown gender differences in social aspects, were consistent with other studies (Hands et al., 2004; Tergerson & King, 2002).

Important differences in physical activity attitudes and behaviours were found between weight status groups. Obese individuals did not value physical activity opportunities, and were more negative about the positive influences of being physically active. The overweight and obese groups were more likely to be taunted by others about their weight or shape, and believed the role of physical activity was principally to control or lose weight. This suggests a different behavioural motivation for physical activity participation. Certainly having fun is more likely to promote a motivation to be physically active (Whitehead, 2008), but having fun may be less likely if an individual is self-conscious, teased, does not have peers to

be active with, lacks resources, or actual participation exhibits negative physical effects (difficult, dislike of how body feels, or uncomfortable) (Zabinski et al., 2003).

The most significant findings were related to perceived barriers or excuses for not increasing physical activity. More obese individuals were more likely to be self-conscious when being active, and therefore developed excuses such as having other interests, not finding physical activity fun, not being good at sport, that people might laugh at them, or that they had poor health. Social aspects and skills were both important for the overweight and obese individuals, with similar barriers reported in a similar aged cohort (Zabinski et al., 2003). Overall the derived scores showed that obese individuals scored less positively across all items. These results provide further evidence that lower self-perceptions are related to lower physical activity levels and unhealthy weight (Li et al., 2005; Murcia et al., 2007).

### **Sedentary behaviours.**

Commonly, screen time, computer time and television watching are used as proxy measures of sedentary behaviour (Jebb & Lambert, 2000; Olds et al., 2004) as these are seen to be the main competitors to time spent participating in physical activity (Olds et al., 2004). Until age 14, gender did not play a role in the amount of time spent in screen based activity. However, at age 14 years more boys reported high levels of screen based activity, compared to girls. This gender difference probably relates to the type of free-time play, with males spending more time playing computer games, while females were more likely to be involved in socialising activities (like chatting on the phone) (Biddle et al., 2004a).

Although cross sectional analysis found more overweight or obese children had higher levels of screen based activities, this was not evident in longitudinal modelling, with all screen based activity models not significant. However there were some specific interactions noted in the estimates which depict specific effects for some subgroups. Consistently, for children classified as obese, those who were involved in less screen based activity (<2 hours per day) had a decrease in BMI

compared to those involved in the highest level of screen based activity (>5hours per day). It would appear that overweight and obese children were more likely to be involved in more screen based activity, a finding reported in a recent study of 2-6 year olds (Jones, Okely, Gregory, & Cliff, 2009). Also, increased screen time may be associated with other negative health behaviours such as increased snacking, with higher snacking shown to occur in unhealthy weight families (Francis et al., 2003).

Interrelationship modelling found that at ages 6, 8, and 10 years higher levels of television watching were associated with increases in BMI. However this relationship was not present at 14 years. At ages 6 and 8 years, physical activity levels appeared to displace the amount of television being watched, with this inverse relationship shown in 2-6 year olds (Jones et al., 2009). It is hypothesised that time spent television watching, especially in the early years, is an indicator of opportunity for physical activity including active play (indoors and outdoors) and active transport (Dehghan et al., 2005), especially during the critical time after school and before dinner (Olds et al., 2004). Certainly in this age group others have shown that increasing time spent in sedentary activity is associated with increased body fat (Blair et al., 2007) and might be related to lowering of resting metabolism (Klesges et al., 1993; Molnar & Livingstone, 2000), and / or snacking behaviours and food choices whilst sedentary (Francis et al., 2003). It may also explain why at 14 years, television watching is no longer important in the overall model, as screen time may now be occurring outside this critical window and later at night. However in this study no information was collected on the time of day for screen time, hence this is speculation only.

There may also be other influences based on the types of programs, advertising, parental supervision and cognitive stimulation (Must & Tybor, 2005). Rennie and colleagues' (2005) review of determinants of obesity related to sedentary behaviours reported links to food advertising of food high in fat and or sugar, lack of advertising for healthy foods (eg. fruit and vegetables), and family lifestyle habits. More poignantly, they suggested without clear secular trends, it is unclear

whether increases in sedentary behaviours per se are obesogenic or it is due to the displacement of active behaviours. Results from this study are suggestive of the latter, particularly in light of evidence that the influence of sedentary behaviour disappears when adjusted for moderate and vigorous physical activity in 12-year-olds (Mitchell et al., 2009).

### **Self concept.**

Self concept is important in understanding individual motivation and behaviour (Hagger et al., 2005; Harter, 1990; Weiss, 1987) with physical activity participation shown to increase when one has positive perceptions of their capacity to contribute (Murcia et al., 2007), and decrease with negative body esteem (Schmalz, 2010). These capacities support the development of perceived competence, which have been described as stemming from early childhood where objective self-awareness and self-conscious evaluations begin (Houck, 1999).

Overall, in this study, males reported higher scores than females across self-perception domains, consistent with a meta-analysis study (Wilgenbusch & Merrell, 1999) and Hagger et al. (2005). Normal weight individuals had higher scores in athletic competence, physical appearance and overall sense of self-worth, than those overweight or obese. Individuals overweight or obese had lower scores in self-worth, consistent with lower self-esteem reported in unhealthy weight children (Waters & Baur, 2003) and lower body esteem in adults (Schmalz, 2010). This construct is regarded as relatively stable and enduring (Hagger et al., 2005; Raudsepp et al., 2004) and is suggestive of an underlying difference in global self-worth for healthy weight versus unhealthy weight. Individuals overweight and obese may be less able to cope (e.g. with family stress), lack motivation (e.g. to be physically active), and may be less happy (e.g. peer related judgements) (Harter, 1990), than their healthy weight counterparts.

Investigation of these domains in the longitudinal model however did not find any significant relationship with BMI. Exploratory SEM also found no direct pathway

between self concept and BMI. However, it was shown that self concept may work in indirect ways through other variables such as attitude to participation and the valuing of physical activity, which impact on participation levels of physical activity (Schmalz, 2010). Certainly these findings support the role of a positive self concept (perceived athletic competence, association with a positive physical appearance, and overall global self-worth) in increasing participation in physical activity (Li et al., 2005; Murcia et al., 2007) which in turn is related to body weight (Dragan & Akhtar-Danesh, 2007). However in this study, without repeated measures of self concept, the direction of the relationship between BMI and self concept could not be discerned.

Related to self concept, parent reported social and behavioural progress at age 10 years found weak associations. Interestingly, more parents of obese girls were dissatisfied with their social progress, supported by a weak but significant interaction in the longitudinal BMI modelling. Overall parents of obese individuals were also dissatisfied with their behaviour progress, although no longitudinal relationship was found. It is unclear what inferences can be made here, particularly considering that these results are based on two survey questions alone. Yet these results may provide further support of the importance of individual motivation and behaviour in building one's self concept for *normal* behavioural and social development (Hagger et al., 2005; Harter, 1990; Weiss, 1987).

## **Environmental Factors**

### **Socioeconomic status (SES).**

Socioeconomic status comprises a number of characteristics that influence behaviour, having both direct and indirect effects on obesity. These include wealth (family income), environment (SEIFA), educational attainment, knowledge (ethnicity, occupation) and life stress (Sanigorski, Bell, Kremer, & Swinburn, 2007). Results from this study support the SES gradients in fatness for Australian children reported in other studies (Dollman et al., 2007; Sanigorski et al., 2007), with those at the lower end more likely to be overweight or obese.

### ***Socioeconomic index for areas (SEIFA).***

Socioeconomic index for areas (Australian Bureau of Statistics, 2007a) was used as an environmental indicator of community SES as it permits the socioeconomic investigation beyond the individual (Bennett, Wolin, & Duncan, 2008). There were no reported gender differences for SEIFA, but significant weight status groups differences, like that reported for the Victorian cohort (Sanigorski et al., 2007) and similar to international trends in neighbourhood socioeconomic position (Bennett et al., 2008). Compared to normal weight individuals, overweight and obese individuals tended to have lower SEIFA scores and therefore lived in communities of more disadvantage. These disadvantaged communities have been shown to have a greater exposure to fast food (advertising and access), are less safe, and their community facilities are often poorly maintained (Dalton, 2007), aspects of the environment considered to not promote healthful behaviours, nor help avoid obesity (Sallis & Glanz, 2006). These findings provide support for the hypothesis that social context may play a more important role in obesity risk than other SES factors (Bleich, Thorpe, Sharif-Harris, Fesahazion, & LaVeist, 2010).

### ***Family Income***

Individuals from families with low income were more likely to be overweight or obese. While not evident in early childhood (birth to age 3 years), this relationship was strong from late childhood (6 to 14 years). Longitudinal modelling however did not find significant influences, although interrelationship modelling suggested family income did play a role with BMI, indirectly through influence on other obesogenic factors.

Exploratory interrelationship models at age 1 year, found high income was associated with increased time spent breast feeding, and may relate to both the opportunity for the mother to stay at home to care for her child and the educative value of breastfeeding one's child for longer (Sloan, Gildea, Stewart, Sneddon, &

Iwaniec, 2007). Another confounder is that low income families tend to be overweight and obese (Australian Bureau of Statistics, 2007c), and mothers who are overweight and obese tend to breastfeed for shorter duration (Burke et al., 2005; Hediger et al., 2001).

At ages 6, 8, 10 and 14 years, income was positively related to activity, and inversely related to the amount of television watched. The number of reported organised activities may relate to the opportunities available due to the costs of activities, cost and availability of transport, availability of the parent to transport the child to the activities (working parents) and also attitudes toward the importance of activity participation (Olds et al., 2004). Similarly, if low income relates to reduced opportunity for participation in organised activities, then television watching may be an easy and affordable alternative.

### ***Parental Employment***

In recent times maternal employment has increased, with resultant changes in parental behaviour, home routines and attitudes (Fertig, Glomm, & Tchernis, 2009). A connection between maternal employment and childhood obesity has been suggested (Fertig et al., 2009; Hawkins, Cole, & Law, 2008). In the present study, children whose mother stayed at home were more likely to be obese, while those whose mothers were working were more likely to be of normal weight, with little difference in the overweight group. These results are generally similar with the Australian Bureau of Statistics (2007b) report which found the proportion of overweight was similar across SES. Interestingly, longitudinal modelling found that a working mother was protective for the obese group only. This suggests that for obese children, there is either a negative influence of the home environment (for non-working mothers), or a positive influence of the care environment (for working mothers), with more maternal work hours shown to be associated with lower BMI if a child spent time in child care (Fertig et al., 2009).



Further, the investigation of hours a mother worked showed very small decreases in child BMI for increasing hours. This concurs with Fertig and colleagues (2009), but is in contrast to Hawkins and colleagues (2008) who reported an increased likelihood of obesity for every 10 hours per week worked. These contrasting findings are probably related to sample size as 18,533 families were recruited for the Millennium Cohort (Hawkins et al., 2008), compared to 1,403 in the current study, and 2,500 children in the study by Fertig and colleagues (2009). It may be that mother's work is a proxy for maternal education and reflective of their related effects (discussed below under Parental education). Such confounding effects, were also identified by Fertig and colleagues (2009) who showed differences between low and high educational attainment mothers, their work hours, and different effects for school and childcare. Overall, maternal employment has a complex relationship with other factors related to obesity.

### ***Parental education and father's occupation.***

Parents with low educational attainment had higher proportions of obese children, concurring with another Australian study (Sanigorski et al., 2007). In the present study, in addition to parental education, father's occupation was also related to weight status, with those involved in sales, plant operations and labouring having a higher proportion of obese children. Trades people had an even distribution of obese, overweight and normal weight for both educational attainment and father's occupation. The mechanisms by which parent education and occupation may operate are unclear. Evidence has shown a positive association between educational level and healthy patterns of behaviour (activity level and cardiovascular fitness) (Cleland et al., 2009; Ferreira, van der Horst, Wendel-Vos, Kremers, van Lenthe, & Brug, 2007) and negative sedentary behaviours (television time) (Hesketh, Crawford, & Salmon, 2006). Paternal occupation mechanisms may be related to family income, role within the family, paternal food preferences (Sanigorski et al., 2007) or occupational culture. Overall though, more in depth considerations of what these operational mechanisms may be need further

investigation, to provide clarity and opportunities for more targeted and appropriate intervention programs.

### ***Ethnicity.***

Often social demographic characteristics such as ethnicity are used to identify disparities in health related outcomes (Bennett et al., 2008), although recent evidence suggests that such disparities are associated with the social context and not ethnicity (Bleich et al., 2010). Although sample size was low and highly skewed, in the present study father's ethnicity was associated with weight status, with proportionally more Caucasians overweight and obese. Longitudinal modelling also found that parental ethnicity was associated with weight status, and Caucasians had a higher BMI. It could be argued that ethnicity may be a proxy for genetic differences (Bennett et al., 2008) or heritable individual genetic-environment susceptibility (Mutch & Clement, 2006; Walley et al., 2006). It might also be a proxy for parent behavioural qualities that may be attributed to ethnic related cultural practices and are therefore directly and indirectly related to patterns of behaviour (Gibson et al., 2007; Olds et al., 2004).

### ***Life Stress.***

The often assumed relationship between stress and obesity is not conclusive. Whether stress or its confounders are responsible is uncertain, with both biological and comfort food mechanisms considered (Bennett et al., 2008). Across the years of this study, money problems was the most associated life stress variable with weight status. Parents who reported money problems had fewer normal weight children. Another study with this cohort found financial stress during pregnancy was associated with a shorter duration of breastfeeding (Li et al., 2008), which itself is a risk factor for future obesity (Chivers et al., 2010). Longitudinal modelling also identified *problems with children* and *other problems* as significantly associated with weight status, both leading to an increased BMI. These two latter stress items may also be indicative of other behavioural mechanisms (such as the ability of the

parent to manage children's behaviour i.e. parenting style), especially since investigation of three or more life stress events did not reveal any relationships, nor any interrelationships using SEM. This finding for overall stress corresponds with the lack of association between negative life events and BMI in another Western Australian cohort of 6-13 year old children (Gibson et al., 2007). Therefore family life stress may not play a significant role in weight status for children. However financial stress, which could occur across SES and be dependent upon level of household debt, may identify a stress burden which has an indirect role with other obesogenic factors related to adolescent obesity such as breastfeeding, financial costs associated with organised sport and quality of diet.

### **Parental influences.**

Parental influences are embedded among many of the variables discussed and work both directly and indirectly on the child's pattern of behaviour (Gibson et al., 2007; Klesges et al., 1990; Olds et al., 2004; Steinbeck, 2001). Probably most critically, parents serve as a role model to their children (Cleland, Venn, Fryer, Dwyer, & Blizzard, 2005; Gibson et al., 2007), particularly in the preschool years (Ventura & Birch, 2008; Wake et al., 2007). Parental exercise habits are positively associated with the 7-15 year old children's activity levels and cardiovascular fitness even after adjustment for age, SES and school type (Cleland et al., 2005). It is proposed that parent influence on exercise may operate at three levels, firstly by modelling behaviour, secondly by providing encouragement and support (transport, equipment, funds), and thirdly through a genetic predisposition to being active (Cleland et al., 2005). Importantly, the role of parents is critical at the earliest stage of child development to support and promote healthy behaviours (Lindsay et al., 2006).

So far parental influence has been shown through early feeding practices, diet choice, participation in organised activity and sport, opportunity for participation (due to financial or time constraints), attitudes and values to active and sedentary

behaviours, encouragement to be active, culture (ethnicity), employment and education. Discussion of other factors now follows.

### ***Maternal smoking during pregnancy.***

Maternal smoking is a modifiable prenatal determinant of child obesity and is thought to reduce fetal growth (Gillman, 2008). The growing evidence that maternal smoking during pregnancy causes later obesity in the children (Gillman, 2008), was also supported by significant cross-sectional weight status group differences, particularly for those overweight. Longitudinal modelling confirmed that smoking during pregnancy reduces fetal growth in this cohort (lower birth weights), with BMI higher after birth. Whether maternal smoking is causative of, or a marker of other SES determinants and behaviours, was not able to be determined in this study.

### ***Parent BMI.***

The association between parent and child BMI is well documented (Bell et al., 2007; Huus et al., 2007) and the results of this study concur with previous research. Significant weight status group differences were found for mother's BMI, with longitudinal modelling showing that as mother's BMI increased so did the child's BMI. A similar result was found for fathers, although the actual increase in BMI was smaller, when compared to mothers. Although there were no weight status group differences for parent birth weight, longitudinal modelling found for both mother's and father's birth weight, there was an incremental increase in BMI. Exploratory SEM at birth suggested that parent birth weight was correlated to child birth weight and gestational age. Both the LMM and SEM results indicate some underlying polygenic factor which suggests heritable individual genetic-environment susceptibility (Mutch & Clement, 2006; Walley et al., 2006).

### ***Parenting styles.***

The role of family characteristics on child obesity has been hypothesised over many decades. There is anecdotal evidence that parenting style, supportiveness and social cohesion may be important in respect to the development of obesity (Gibson et al., 2007; Wake et al., 2007) and that authoritarian parenting may have the highest risk of overweight (Rhee et al., 2006). This study investigated a total score for parenting and individual parenting styles relating to *laxness*, *over-reactivity* and *verbosity*. Overall, no gender or weight status differences in parenting styles were found at age 10 years, although longitudinal modelling indicated that with more negative parenting behaviours there was an increase in BMI for overweight individuals, followed by the obese group.

Adolescents rated their perception of parenting at age 14 years. Compared to males, more females reported positive parenting behaviours. Generally there were weak or no weight status group differences, which corresponds with results for the same parenting scale in 6- to 13-year-old Western Australian children (Gibson et al., 2007). Some negative parenting styles were associated with obesity. Obese males tended to be threatened or hit more often by parents, while obese females found parents were inconsistent in the enforcement of rules, and they felt less appreciated. However longitudinal modelling results suggested that the most adverse parenting behaviours (such as, threatened or hit and threaten punishment) were more likely to result in decreases in BMI, regardless of weight status.

Although these results concur with Gibson and colleagues (2007), a limitation to their study was the absence of child perceived parent support. This factor was evaluated at age 14 years in the present study, but it was not important for weight status. However this study did not differentiate between mother's and father's parenting styles nor investigate parenting and the preschool years, therefore no conclusion can be drawn on how early childhood parenting styles might relate to weight status in adolescence. Wake and colleagues (2007) found father's parenting style was associated with preschool child BMI (Wake et al., 2007) with authoritarian

parenting (high demands for self-control but low levels of sensitivity (Rhee et al., 2006) associated with higher risk of obesity (Rhee et al., 2006; Wake et al., 2007). Perhaps this study's weak findings relate to the low numbers of obese individuals in the cohort, but clearly lend support toward the positive role supportive, consistent and fair parenting has on childhood healthy weight possibly by providing children with positive incentives and guidance to develop effective self-regulation behaviours in respect to diet, exercise and sedentary activity (Rhee et al., 2006)

### **Built environment.**

More recently there has been a growing interest in the built environment as a determinant of obesity in children, especially in respect to its association with diet and physical activity behaviours (Burdette & Whitaker, 2004; Maddison et al., 2009; Maziak, Ward, & Stockton, 2008; Norman, Nutter, Ryan, Sallis, Calfas, & Patrick, 2006; Page et al., 2010). There is evidence to suggest that the more time pre-school children spend outdoors, use local parks and playgrounds, and have access to physical activity equipment, the more active they are (Norman et al., 2006). However research with adolescents reveal that these relationships are complex, and should be considered in context with SES factors (Boone-Heinonen, Evenson, Song, & Gordon-Larsen, 2010). Although built environment variables were not specifically collected in this cohort, and limitations based on Boone-Heinonen and colleagues (2010) are acknowledged, proxies such as early childhood home influences, and school influences were investigated.

### ***Early childhood home influences.***

In this study, having a swimming pool, home garden, or living near a park at ages 1, 2 and 3 years was hypothesised to provide a positive environment for physical activity and healthy weight. Except for living near a park or playground at age 1 year, all home influences had no significant relationship with weight status. These results may relate to the low sample size at follow-up ages 2 and 3 years, as well as the highly skewed data with a *yes* or *no* response. The latter issue was also identified as a limitation by Burdette and Whitaker (2004). The presence of a home

garden does not provide sufficient information on available space for physical activity, nor sufficient evidence for a relationship with yard space, as suggested for investigation by Burdette and Whitaker (2004). This is disappointing considering home yards provide active outdoor free-play opportunities (Veitch et al., 2010). Interestingly, at age 1 year, a higher percentage of healthy weight individuals did not live near the park or playground, which supports other studies that playground proximity is not related to overweight or obese (Burdette & Whitaker, 2004; Potestio et al., 2009; Timperio, Jeffery, Crawford, Roberts, Giles-Corti, & Ball, 2010). In considering these results, it should be noted that there is a poor level of agreement between objective and perceived proximity reports (Lackey & Kaczynski, 2009), and perhaps proximity to active spaces is related more to possible barriers to being healthy and active, with those exhibiting generally healthy behaviours also less likely to find proximity to a park or playground a barrier (Sallis, 2000).

This reasoning may explain why in longitudinal modelling we found an individual's increase in BMI was greater for overweight and obese, compared to normal weight if they did not live near a park. Exploratory SEM at age 1 year also did not find any direct relationships between these early childhood influences, although preliminary investigations found that early walking was related to frequency of park visits, and frequency was associated with proximity. This relationship was hypothesised, but untested, by Burdette and Whitaker (2004). Certainly there is sufficient evidence to question the association and further investigations are warranted.

### ***School influences.***

There is limited literature discussing the impact of the school environment on obesity in childhood and adolescence. In this study, there were no gender differences for co-education, school affiliation, private versus public school, and representative sport opportunities at age 14 years. Weight status group differences were found for each school influence. More obese children attended coeducational schools which probably relates to a greater representation of such schools in the public system, and also overall more children from lower SES backgrounds (O'Dea,

2003b). Independent schools had the lowest overweight and obese individuals compared to Catholic and public schools, and obesity levels in public schools were double those in private schools. Longitudinal modelling did not show any significant trends. Overall these findings probably reflect differences in SES gradients typically seen across private and public school sectors in Australia (O'Dea, 2003b). Based on evidence that compulsory physical activity programs (Cleland et al., 2008b) and school physical activity environments (O'Malley et al., 2009) are not associated with obesity, school influences are likely to be related with SES variables such as family income. Preliminary SEM investigations found that school affiliation (independent, catholic, government) was related with family income, but that school affiliation did not influence BMI.

The sample for school representative sport was small ( $n=142$ ) with a weak weight status group difference found, but no definitive trend. The total school facilities provided an indicator for environmental opportunity for organised sport and activity within the school environment but this was not significant. This result aligns with community environment studies that found little evidence of an association with either BMI (Norman et al., 2006) or physical activity (Maddison et al., 2009) in adolescents. This is contrary to positive community partnership results which demonstrated that community coalitions can increase the likelihood of adolescents being highly active (Leatherdale et al., 2010). In summary, school demographics are most likely the common factor in results found for public versus private schools. Overall, it would seem that the school environment does not directly influence obesity in childhood and adolescence.

### **Synthesis of Overall Findings**

This section collates cross-sectional, longitudinal and interrelationship modelling evidence, with individual, behavioural and environmental perspectives. Research findings are brought together and discussed in respect to the original research questions listed at Chapter One.



## Longitudinal model of BMI.

This study presented a longitudinal model of BMI from birth to adolescents depicting distinct pathways between males and females, as well as between individuals who were normal weight, overweight or obese (Figure 14) (Chivers et al., 2009). Details of this model have been discussed earlier in this chapter, with highlights presented throughout the chapter on the individual influences obesogenic factors had on this trajectory of BMI. Most importantly, this longitudinal model demonstrated that individual, behavioural and environmental factors were cumulative, and had both positive and negative effects on BMI.

These cumulative effects were sometimes specific to gender. Language development (Denver II and IMQ) that was *not normal* in females resulted in increased BMI, possibly reflecting a difficulty in self-regulation behaviour (Houck, 1999), or were more specifically related to the female gender in the development of self-concept (Okely et al., 2004). This was confirmed by an indifferent social progress rating for overweight and obese females at age 10 years, and an inverse relationship between self concept scores (SPPA) and BMI at age 14 years. In addition, increased physical activity levels (e.g. organised sport) had a more pronounced decrease on BMI for females, and may be related to the lower baseline levels of activity in which females are involved, compared to males (Olds et al., 2004; Steinbeck, 2001).

The different cumulative effects were also present for the weight status groups. Individuals who were obese and rated *not like their peers* in their early childhood locomotor skills (IMQ) and had lower activity levels (step count) in adolescence, increased their BMI, distinct from the normal weight and overweight groups. Improved motor competence score (age 10 and 14 years) was also protective, decreasing BMI by 2.2 kg/m<sup>2</sup> and 0.5 kg/m<sup>2</sup> respectively. This suggests a link between motor skill proficiency in mid childhood and later physical activity levels in adolescence, with an association between perceived competence and activity levels previously shown (Barnett, Morgan, van Beurden, & Beard, 2008). Individuals who

were overweight tended to be more affected by low motor competence in late childhood, by having both parents unemployed and by parenting style. More positive parenting scores in particular were most protective for those overweight, with significant decreases in BMI ( $2.2 \text{ kg/m}^2$ ). This association with parental influences suggests that parental role modelling (Gibson et al., 2007), exercise habits (Cleland et al., 2005) and style (Rhee et al., 2006) may be more important for those children who are on the trajectory of overweight.

Overall, a continuum of positive and negative influences on BMI trajectory was shown, and has been discussed in detail previously. These key factors included maternal smoking during pregnancy, parental BMI, early feeding behaviours, aerobic fitness and basketball throw, sedentary behaviour, social progress, and attitudes and values to physical activity. For example, children from normal weight mothers (pre-pregnancy) decreased their BMI by  $0.43 \text{ kg/m}^2$ , and children who were breastfed for less than 4 months increased their BMI by  $1.19 \text{ kg/m}^2$  (Chivers et al., 2010). The diversity in nature of effects in the longitudinal model of BMI confirms the complex and multi-faceted nature of adolescent obesity.

### **Critical time periods.**

The developmental pathway to adolescent obesity was shown to be dynamic with periods of rapid change and periods of relative stability. There is evidence for three main periods to be a critical times for the development of obesity: perinatal, early childhood, and adolescence (Blair et al., 2007; Dietz, 1994; Lawlor & Chaturvedi, 2006). This study provided strong evidence to support a critical developmental period between birth and 8 years with three critical phases: perinatal, from birth to 3 years (early childhood), and 6 to 8 years (early adolescence). Unfortunately, data were only available until age 14 years for this study, and any adolescent and pubertal changes were unable to be examined in detail without another subsequent wave of data.

### ***Perinatal period.***

Several different mechanisms have been suggested to operate in the perinatal period (Dubois & Girard, 2006), with this study confirming the influences of maternal smoking and high birth weight on adolescent obesity. Maternal smoking during pregnancy was associated with lower weight babies, but from birth to adolescence these children had higher BMI, concurring with others (Dubois & Girard, 2006; Gillman, 2008). It is believed that growth restriction due to maternal smoking during pregnancy is then followed by postnatal catch up growth, which continues and is associated with later obesity (Dubois & Girard, 2006).

Higher birth weight was also associated with adolescent obesity. In particular overweight adolescents were more likely to have had higher birth weights compared to normal weight and obese adolescents. Higher birth weights are associated with later obesity (Dietz, 1997), with in utero conditions favouring high weight gain suspected to continue postnatal, suggesting the influence of an environmental factor (Dubois & Girard, 2006).

### ***Early childhood.***

The timing of adiposity rebound is a marker for later obesity (Lawlor & Chaturvedi, 2006; Rolland-Cachera et al., 1984) and in this study began as early as age 3 years, with children on a trajectory of overweight and obese between age 3 and 6 years (Chivers et al., 2009). Rapid growth and weight gain, as depicted in the longitudinal model (Figure 14) have long been suggested to be related to adult obesity (Law, 2001). It has been speculated that adiposity rebound may be an important marker as it is related to the expression of diet and activity behaviours (Dietz, 1997).

Early feeding behaviours, particularly exclusive breast feeding had an important relationship with BMI, which remained until age 14 years (Figure 3). Exclusive breastfeeding influenced the peak BMI at age 1 year, as well as the timing of, and BMI at adiposity rebound, and this influence remained until at least age 14 years (Chivers et al., 2010).

In respect to early motor development behaviours, this study showed that children aged 1 to 3 years were more likely to become overweight or obese if they were not assessed as developing *normally*. By age 10 and 14 years, low motor competence was associated with obesity, suggestive of a possible tracking of motor ability from early childhood. A relationship was also shown between motor ability and physical activity.

Early childhood physical activity behaviours (ages 1, 2, 3, 6 and 8 years), also demonstrated an association with later adolescent obesity. Physical activity behaviours have been shown to track across time, in concurrent work with this cohort (Hands et al., 2010b), as well as in other studies (Janz et al., 2000; Kelder et al., 1994; Telama, 2009). The Hands and colleagues study (2010b) also showed that screen time in early childhood (ages 6 and 8 years) might displace time spent engaged in physical activity. SES was important across years, but individual items for family life stress had particular and consistent effects at age 6 years.

Longitudinal modelling also demonstrated a shift in both males and females BMI trajectories between 6 and 8 years (Chivers et al., 2009). This is considerably earlier than the suggested increased fat deposition and obesity risk for females during adolescence (Dietz, 1994). Together, these results suggest age 6 years might be an important turning point that also happened to coincide with the beginning of full time schooling in the state of Western Australia.

### ***Early adolescence.***

The limit of the data was 14 years for this study, and hence examination of changing influence in early adolescence and beyond was not possible. However, in this period many important changes were occurring. These include physical changes associated with puberty and psychological development of self concept. Both these factors were found to have important co-relationships with other obesogenic factors. Certainly, evidence is suggestive of important mechanisms during this period that are different to earlier periods.

Overall it is clear that major changes in individual, behavioural and environmental factors are occurring in early childhood, and that these complex interactions between biological and behavioural mechanisms (Dietz, 1997) are responsible, at least in part, for adolescent obesity.

### **Interrelationship modelling.**

To further elucidate developmental pathways to obesity this study used exploratory SEM modelling to investigate possible pathways to BMI and interrelationships between factors at follow-up ages 6 to 14 years. SEM is typically used to determine how well a theoretical model is supported by the data (Dragan & Akhtar-Danesh, 2007; Garson, 2007; Schumacker & Lomax, 2004). Since a detailed theoretical model was not hypothesised, the findings from this aspect of the study are speculative at this time. Further investigations with an independent sample would be required to confirm whether these models are supported by other data.

However the exploratory processes provided some valuable insights into possible interrelationships and pathways to BMI. From age 6 years screen time patterns were important in the direct prediction of BMI, but the amount of screen time was influenced by the child's activity levels. By age 8 years, this relationship was reversed, with screen time behaviours now predictive of time spent in organised sport and activity, the latter now influencing BMI. This suggests that screen time patterns are being established by age 6 years, as recently reported for 2- to 6-year-old Australians (Jones et al., 2009). At age 10 years, the importance of motor competence is shown, having an influence on both activity levels, screen time patterns and BMI, findings supported in the present study by cross-sectional and longitudinal modelling results, along with others (Graf et al., 2004). By age 14 years the relationship between motor competence and BMI remained strong, influencing future activity levels and BMI. Overall, motor competence, as determined by early childhood development, may be an integral part of facilitating a healthier lifestyle. Those with better skills are more likely to engage in physical activity (Williams et al.,

2008), less likely to have high screen time patterns (Jones et al., 2009), be more fit (Hands et al., 2008) and hence have healthier diets (Hands et al., 2010a), and a higher self concept (Okely et al., 2004).

Some notable gender differences in pathways were also found. At age 6 years the pathway between activity and income was significant for males but not females. This finding is interesting but might be explained by the nature of the question, with activity representing music, dance, kindy gym and other clubs. These activities may be considered feminine in nature with parents of boys not enrolling their sons in these types of activities. The tendency for boys to participate may be related to parent influenced gender linked stereotypes (Bussey & Bandura, 1992, 1999). In turn, parent influence may be associated with SES factors such as education, occupation and income. At age 10 years, the pathway between motor coordination and activity was more significant for males than females. This suggests that the relationship between better skills and more physical activity (Williams et al., 2008) is stronger for males. At age 14 years, although significant, the influence of fitness on BMI was greater for females than males, whereas mother's BMI was stronger for males than females. Compared to females, the effect of puberty was highly significant for males, and relates to the physical characteristic changes which provide males with increased muscle strength (Hayward, 2003; McCarthy et al., 2006). The pathway between income and screen time was only significant for males, suggestive of SES influences in this behaviour. Specifically this may relate to the availability of screen based games and computers.

This exploratory process also identified other factors not important in strengthening the prediction of BMI in the overall models, this itself being of interest. In the first year of life, breastfeeding was the only strong predictor of BMI. This is not surprising considering the strong link between breastfeeding and obesity in this study (Chivers et al., 2010) and others (Butte et al., 2002; Gillman et al., 2001; Oddy et al., 2006c), which suggests that other factors (e.g. locomotor skills, diet, and physical activity) have a limited impact on BMI in the first year of life. Surprisingly, yet consistent with other results, diet variables at ages 8 and 14 years

did not have any relationships, and probably relate to limitations in the data used, such as lack of information on portion size (Rennie et al., 2005; Steenhuis & Vermeer, 2009) and or more comprehensive dietary pattern information (Ambrosini et al., 2009a; Hu, 2008c). At 14 years, the school environment, self concept and valuing of physical activity, did not have any pathways to BMI, however other factors were seen to influence them. For valuing of physical activity this may reflect a possible lag between thoughts and beliefs and measurable behaviours such as physical activity. As Houck (1999) describes, self concept capacities have already begun to develop in early childhood. For the school environment factors, exploratory results showed that school influence was related to family income, and was not related to BMI, which is similar to findings by others (Cleland, Crawford, Baur, Hume, Timperio, & Salmon, 2008a; O'Malley et al., 2009).

The exploratory processes undertaken for this part of the study provide important information on possible pathways and interrelationships between factors on BMI at several time points in childhood and adolescence. Directionality in pathways provides insights into possible mechanisms of action, and potential opportunities for intervention. SEM results provide evidence-based models available for testing in other datasets, in addition to raising new questions and hypothesis for future research.

### **Gender differences.**

Consistent with previous literature, gender differences were generally common across many variables, and in some obesogenic influences over time (Longitudinal model of BMI). In relation to physical skills, males generally out performed females in performance type tasks such as dodge, object control, bike riding, muscle endurance and strength, while females outperformed males in skipping and fine motor skills. Females were more flexible, and involved in more activities (such as dance, gymnastics), while males played more sport, had higher activity levels at higher intensities and were more aerobically fit.

For self concept domains, females scored higher on behavioural conduct and close friend, while males scored higher in athletic competence, physical appearance, global self worth and romantic appeal. Compared to males, females academic performance, learning skills progress and behavioural progress was more likely to be rated highly by parents or caregivers. Compared to females, males were more positive about physical education classes, the effects of physical activity, and less likely to report excuses for not increasing physical activity in the future.

Until adolescence, screen behaviours were similar between males and females. However, at 14 years more males were now involved in high levels of screen time compared to females. This period was also associated with puberty, with more females in the later stages of pubertal development compared to males.

The longitudinal model of BMI clearly depicted gender as an independent moderator of effects with distinct trajectories and an overlap between ages 8 and 10 years (Chivers et al., 2009). Exploratory SEM models also demonstrated gender differences in pathways to BMI (discussed previously). These included significant differences between males and females in the following paths:

- family income and organised activity, age 6 years;
- motor coordination to activity, age 10 years;
- aerobic fitness to BMI, puberty to aerobic fitness, mother's BMI and BMI, and income and screen time, age 14 years.

Overall, these findings confirm the importance in specifically identifying and acknowledging gender differences in obesity studies. This includes investigations of the possible influence on obesogenic factors and taking into account how these gender influences might change over time. Such basic knowledge is at the core of the development of appropriated and effective public health interventions.



## **Differences between overweight and obese adolescents.**

The weight status group differences identified in this research revealed that overweight and obese adolescents' responses were not always in the direction expected, nor always on a continuum from normal weight to obese. As far as is known, no such evidence has been presented within the literature, and reflects the complex and dynamic nature of the constructs of weight status.

The following findings highlight these non-typical trends:

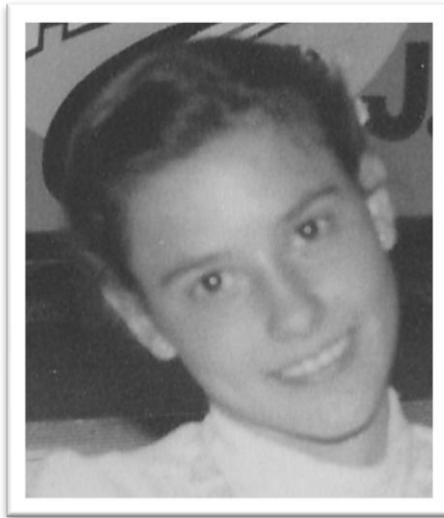
- A high frequency of vegetable intake was greatest for those overweight, and least for those obese, and only for females.
- Obese individuals were more likely to have non-normal developmental milestones, but by late childhood motor abilities across weight status were along the continuum. However, longitudinal modelling found at ages 10 and 14 years low motor competence scores (MAND) resulted in increases in BMI for the overweight group only.
- Overweight and normal weight individuals were similar in their parent reported activity levels at ages 6, 8, 10 and 14 years, with obese individuals less vigorously active, and more inactive.
- Screen time was similar for the overweight and normal weight groups at ages 6, 8, and 10 years, with the obese group watching higher levels of 2 hours or more of television. However at 14 years, the overweight group were now similar to the obese group, watching higher levels of 2 hours or more of television, compared to the normal weight group.
- Stress items revealed that overweight children had a higher frequency of reporting family stress items *pregnancy problems* at follow-up age 6 years, and *residential move* at 8 years than the obese or normal weight adolescents.
- LMM found that where both parents were not employed BMI increased for the overweight group.
- The overweight group had highest proportion of mothers who smoked during pregnancy.

- A higher proportion of overweight adolescents attended Catholic schools, while a higher proportion of obese adolescents attended coeducational government schools.
- LMM at age 10 yrs showed that the overweight group increased BMI with more negative parenting, compared to the obese and normal weight group.

Certainly, findings across individual, behavioural and environmental factors question the practice of combining data for overweight and obese into one group, which may mask true differences. It also highlights that interventions may need to be different for overweight compared to obese individuals and also time-period sensitive.

### **Summary**

Individual, behavioural and environmental factors are all intricately interwoven in the multi-faceted nature of obesity. These interrelationships and obesogenic influences have been shown to change over time, with this data confirming early childhood as a critical period for the development of obesogenic behaviours. Further, pathways differ for males and females, with obesogenic influences different across gender and changing over time. Longitudinal modelling demonstrated clear distinctions in the pathways of normal weight, overweight and obese groups, with obesogenic influences on BMI being both negative and positive. Influences were also shown to be different for individuals who are overweight or obese, and not necessarily on a continuum. Together these results demonstrated complex and dynamic interactions within individuals and among their environmental influences.



Fourteen years

"Adolescence is a new birth,  
for the higher and more completely human traits are now born."

By G. Stanley Hall

## Chapter Six

### Summary and Conclusions

This final chapter revisits the major research questions posed in Chapter One and summarises the key findings. Directions for future research are discussed, along with the practical implications of these results.

The over-arching research question for this study was:

*How do individual, behavioural and environmental factors during childhood contribute to weight status at adolescence?*

This study has shown, within the constraints of available variables that complex interrelationships exist between individual, behavioural and environmental factors. Their relative importance to maintaining healthy weight from birth through to early adolescence varies. Healthy weight maintenance is a complex balancing act maximising positive and minimising negative influences, and an individual's ability (genetic, psychological and behavioural) to be resilient to the impact of negative influences.

Evidence from this study supports the complex, reciprocal interactions occurring over time and among obesogenic factors as per the Bandura (2001) model of Social Cognitive Theory. For example self concept results depict important relationships between motor competence, perceived benefits and barriers to physical activity; and actual physical activity participation. Support is provided for the perspective that human learning occurs through modelling processes and observing others (Lindzey et al., 1978), as shown by the effect of parental and peer influences. Differences in behaviours between individuals, and between weight status groups,

demonstrate the complex interactions of human behaviour involving individual thoughts and actions, beliefs and competencies, in the context of social influences and structures (Davis, 2006). Lastly, the exploratory SEM models concur with Bandura's (2001) overall social cognitive model. BMI was shown to be affected by both direct and indirect effects, among and between factors, which change and evolve over time and with age.

The two main over-arching findings were:

- Individual, behavioural and environmental factors affect adolescent weight status in positive and negative ways. These effects are cumulative over time and vary with gender, age, and weight status.
- Individual, behavioural and environmental factors operate in both direct and indirect ways to influence BMI. The strength of these associations changes over time and varies with age and gender.

Several sub questions were explored during the course of this study and are now discussed, providing further support to these two key findings.

*What is the relative contribution of individual, behavioural and environmental factors to weight status before birth to adolescence?*

Over the life course, the influence of individual, behavioural and environmental factors is ever changing. Prenatally, the mother's eating and smoking behaviour all have a significant impact on the gestational age, weight and health of the child at birth. In the first year after birth, early feeding choices are a significant and dominant predictor of weight and their influence remains until at least 14 years (Chivers et al., 2010). Developmental and coordination progress is critical through early childhood, and contributes to physical activity behaviours and self concepts in later childhood and adolescence. At puberty, the child's body is changing dramatically both physically and emotionally.

Key determinants of adolescent obesity identified in this study were:

- Prenatal environment;
- Early feeding choices;
- Motor development and abilities;
- Physical activity behaviours;
- Screen time viewing patterns;
- Attitudes towards physical activity participation; and
- Mother's BMI.

***What is the relative contribution of individual, behavioural and environmental factors in a longitudinal model of BMI?***

Longitudinal examinations using the LMM demonstrated that individual, behavioural and environmental factors had both positive and negative effects on BMI, sometimes specific to gender and weight status at 14 years (Chivers et al., 2009). This latter finding lends support to the notion of an epigenetic mechanism with obesity (Cami3n et al., 2009). For example adolescents who were classified obese at age 14 years and as a toddler were reported to be *not like their peers* in locomotor skills had higher BMI. For the overweight group, both motor competence and parenting style were more important. Overall, the more negative the influences the higher the BMI, that is, these negative influences were additive. This cumulative effect on BMI change has been suggested by others (Hesketh, Carlin, Wake, & Crawford, 2009).

The key findings were:

- BMI pathways differ statistically between normal weight, overweight and obese adolescents from three years.
- Individual, behavioural and environmental factors affect BMI in a cumulative manner, dependent upon gender, age and weight status.

*Are there any critical phase shifts in the relative contribution of these factors during this age range?*

The early childhood years were shown to be critical periods for the establishment of healthy weight related behaviours. The relationship between maternal smoking and birth weight highlighted the importance of the intrauterine environment to eventual adolescent weight status. In the toddler years the timing of, and BMI at adiposity rebound, early infant feeding patterns, developmental progress, and activity behaviours were associated with later obesity, as well as precursors for later childhood behaviours. Age 6 years in particular was identified as a critical turning point for behavioural patterns, and the point at which more data were available concerning physical activity behaviours. Life stress variables were also important at age 6 years. Also, screen time was critical in the establishment of early physical activity behaviours at age 6 years. This finding provides support to current Australian policy discussions that reducing screen time in young children is a significant health and well being issue (Centre for Community Child Health, 2009). However, even more concerning, was that the obese group was already on a trajectory to unhealthy weight as early as age 3 years, as shown by the BMI LMM and also by adiposity rebound investigations (Chivers et al., 2009).

*Are there any gender differences over time and across factors?*

Not surprisingly, gender differences were shown repeatedly across variables, and were generally consistent with previous literature. Notably, the effect of obesogenic factors worked in different ways for males and females, as well as changed over time. Generally, females were more influenced by social perspectives (e.g. peer support), while males were influenced more by performance (e.g. athletic competence). Screen time patterns were similar only until age 10 years, then at 14 years males spent significantly more time involved in screen activities than females. Physical activity behaviours and levels were different with males being more active, at higher intensities and having more positive attitudes. The changes with puberty

were also notably different, with females being further advanced in pubertal development compared to males at age 14 years.

In respect to obesity, some factors were more important for one gender. For example, the LMM identified interactions with gender for language, organised sport and physical appearance. Their BMI trajectories also differed, with a crossover point between 8 and 10 years, after which females' BMI, on average, was higher than males (Chivers et al., 2009). Exploratory SEM models also demonstrated gender differences in pathways to weight status. For example the pathway from income to screen time was not significant for females but significant for males at 14 years.

Key findings included:

- Scores, ratings or test results were different between males and females, with these differences were usually consistent across weight status groups;
- Females were influenced more by social perspectives of obesogenic factors;
- Males were influenced more by performance perspectives of obesogenic factors;
- The influence of screen time was more important for males at adolescence; and
- The influence of puberty on obesogenic factors began earlier for females.

*How do the interrelationships between individual, behavioural and environmental factors affect BMI?*

Valuable insights were gained from exploratory SEM modelling of the complex interrelationships between individual, behavioural and environmental factors, in the pathways to BMI. In early childhood (age 6 and 8 years), screen time was an important predictor of BMI, but by 10 years the interrelationships were more complex. Motor competency, aerobic fitness and physical activity were highly interrelated and together predictive both directly and indirectly of BMI. Although



in the early years, motor competency was less important in predicting weight status, these early levels were important in the tracking of motor competence at 14 years (Hands et al., 2010b) where they played an important role in the age 14 year model of BMI. Overall, motor competence, as expressed in early childhood development, may be an integral part of facilitating a healthier lifestyle later on. These exploratory processes also identified factors not influential in the overall model. Diet, the school environment, self concept, and valuing of physical activity all had no role in the overall model, although these factors were the result of influence of factors within the model.

The key findings were:

- Interrelationships among individual, behavioural and environmental factors are complex and dynamic.
- Individual, behavioural and environmental factors operate in both direct and indirect ways to influence BMI at each follow-up age.
- Gender differences influence factors and the strength of pathways to BMI.

## **Limitations**

The limitations to this research have been outlined specifically in Chapter One and addressed in the discussion Chapter Five, with a brief summary outlined here. The data for this study was drawn from the Raine Study up to the 14 year collection point and inherits many of the limitations of the data collection process. This cohort may not be truly representative of the metropolitan Western Australian population as it was not drawn randomly. More specific to this research was the restriction of variables to identified obesogenic factors; use of BMI as a proxy measure of adiposity, along with use of IOTF cut-points for weight status; non-standardized observational data; mixture of caregiver and adolescent reporting; changes in how information was collected on variables that tracked over time; limitations in how variable information was originally recorded (e.g. diet); restricted data collection points with sampling frequency low, especially in respect to capturing adiposity rebound; and the exploratory rather than confirmatory

processes used in SEM. Overall however, these limitations are well recognised in studies of obesity and are not considered to adversely affect the generalisability or significance of the results.

## **Strengths**

One of the key strengths of this study was the relatively large sample and longitudinal nature of the research design spanning from birth to 14 years. It provided an opportunity to examine early pathways of weight status, and in particular an examination of the timing of the adiposity rebound in an Australian cohort. The large sample, even with attrition, provided for more accurate distinctions to be made among gender and weight categories (Chivers et al., 2009).

The unique mixed modelling statistics used to model trajectories of BMI accounted for correlated errors normally associated with repeated, continuous and correlated observations. The mixed model permitted the evaluation of age as a covariate, rather than predetermined averaged time points (survey waves), which increased the validity of the model (Chivers et al., 2009).

This modelling approach provided an opportunity to test factors that might drive accelerated and early increases in BMI. Early pathways of weight status were examined, in particular the possible influence of obesogenic factors. Specifically clarifying the influence of early infant feeding on adolescent BMI and its relationship with adiposity rebound demonstrated the value of such statistical testing (Chivers et al., 2010).

The use of exploratory SEM highlighted the importance of early childhood in the development of sedentary and physical activity behaviours (6 years), that track into adolescence, and play a concurrent role with adiposity (Hands et al., 2010b). Model development demonstrated complex interrelationships at ages 6, 8, 10 and 14 years and how these pathways and their influence changed. These models provide

hypothetical models that can now be tested with other data using confirmatory SEM techniques.

## **Future Research**

The ongoing nature of the Raine Study means that currently another wave of data is available (age 18 years). This presents an ongoing opportunity to explore the current study's findings and investigate whether these reported influences continue into early adulthood.

More specifically, based on the findings from this research, as well as identified gaps in knowledge (things that couldn't be tested due to lack of data), three key areas for further research were identified. These include aspects related to the preschool years, parent BMI, and adolescent motivations.

### **Preschool years.**

The preschool years require a more in depth analysis to ascertain key behavioural and environmental influences that may impact on concurrent and future obesogenic behaviours, including protective mechanisms. These may include:

- An investigation into differences in pre-school lifestyle behaviours, particularly for those children overweight and obese in early childhood.
- An investigation of behavioural differences between pre-school children in the home environment with their primary caregiver, in the home environment cared by relatives, or in community based day-care.
- An investigation of differences in parental lifestyle behaviours and their association with early feeding choices and future diet patterns.
- An investigation on how parental behavioural mechanisms impact on their child's physical activity, sedentary activity and lifestyle behaviours in these early years.

### **Parent BMI.**

The behavioural mechanisms in daily living associated with parent BMI, but particularly mother's BMI warrant further investigation.

- An investigation of the differences between behavioural characteristics (physical activity, diet, BMI, attitudes and values) of mothers who smoked during pregnancy, compared to non-smokers, and their relationship to childhood overweight.
- An investigation of how parental behavioural mechanisms impact on their child's physical activity, sedentary activity and lifestyle behaviours?
- An investigation on how parenting styles differ between healthy weight versus unhealthy weight parents?

### **Self concept.**

The development of self concept in the early years, the possible lag effects into adolescence and their relationship with future behaviours (e.g. activity participation, screen time patterns) requires further investigation, particularly differences between healthy weight and unhealthy weight.

- An investigation of external motivators for being active, and their role in obesity.
- An investigation into general motivational differences for exhibiting healthy behaviours (e.g. active, healthy diet), between healthy weight and overweight.
- An investigation into how self concept changes from early childhood into adolescence, with a specific focus on concurrent analysis of activity and sedentary behaviours and possible lag effects.

## **Conclusion**

This study shows, within the constraints of available data and the 14 year time frame of the longitudinal investigation, the complex interrelationships between individual, behavioural and environmental factors, and their relative importance to maintaining healthy weight from birth through to early adolescence. Healthy weight maintenance is a complex balancing act between positive and negative influences, and an individual's ability (genetic, psychological and behavioural) to be resilient to the impact of negative influences. Early childhood is identified as a critical time point for establishing key behaviours that influence weight status in adolescence.

This research demonstrates the complex and dynamic nature of obesity. It highlights that causation is multi-factorial with identifiable patterns for sub groups. As such, prevention and intervention policies and prevention programs need to be multi-faceted, and target gender and weight status groups differently. Clearly, the current concern for obesity rates among Australian children requires a focus on early pre-school behaviours, particularly at home, and in association with maternal behaviours.

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## **Appendix A**

### **Related Publications**

Each paper listed below, is presented in full overleaf.

**Chivers, P. T.**, Hands, B., Parker, H., Beilin, L. J., Kendall, G. E., & Bulsara, M. (2009). Longitudinal modelling of body mass index from birth to 14 years. . *Obesity Facts*, 2, 302-310.

**Chivers, P.**, Hands, B., Parker, H., & Bulsara, M. (2009) The role of physical activity is different for normal weight, overweight and obese 14-year-old adolescents. *Journal of Science and Medicine in Sport*, 12(6),e137-138. Seventh National Physical Activity Conference, Brisbane QLD. October. doi:10.1016/j.jsams.2009.10.286

**Chivers, P. T.**, Hands, B., Parker, H. E., Bulsara, M., Beilin, L. J., Kendall, G. E., & Oddy, W. H. (2010). Body mass index, adiposity rebound and early feeding in a longitudinal cohort (Raine Study). *International Journal of Obesity*, 34, e1-8. doi:10.1038/ijo.2010.61

**Chivers, P. T.**, Hands, B., Parker, H. E., Beilin, L. J., Kendall, G. E., & Bulsara, M. (2010). A comparison of field measures of adiposity among Australian adolescents from the Raine Study. *Malaysian Journal of Sports Science and Recreation*, 6(1), 33-45.

### **Other Publications During Candidature**

- Hands, Beth; **Chivers, Paola**; and Jetson, Tim (2009) 'Book reviews', *Sport, Education and Society*, 14 (1), 141-146.















































































## Appendix B

### Activity Classification Protocol

#### Physical Activity Categories

<b>LIGHT- minimal effort (CODE as 1)</b>	
Bushwalking – leisurely	Sailing, other Boating Activities
Cubs, Scouts etc - team games	Snorkelling
Fishing	Ten pin bowling
Golf	Tree climbing
Kicking a ball (alone)	Trampoline (home)
Playing in a park or playground	Walking/Strolling
Pool/Snooker	Yoga
<b>MODERATE- not exhausting, sweat after 20 minutes (CODE as 2)</b>	
Aerobics (classes or home)	Kayaking
Athletics/Little Athletics	Lifesaving/Little Nippers
Badminton	Rollerblading/Rollerskating/Iceskating
Ballet	Shooting hoops with friends
Baseball	Soccer with friends
Bicycling – Easy	Scooter
Body surfing	Skateboarding
Boogie board riding	Softball
Board surfing	Surfing
Canoeing	Swimming - Recreational
Cricket	Table tennis
Dancing	Teeball training/game
Exercise Bike	Tennis- doubles
Fast walking	Tennis- lessons
Horsriding	Volleyball
<b>VIGOROUS – heart beats rapidly. Light sweat w/i 5 minutes (CODE as 3)</b>	
Basketball training/game/miniball/3 on 3	Orienteering
Bicycling - long distance, racing	Rock climbing
BMX/trail bike	Running
Football training/game	Skipping rope
Gymnastics	Soccer/Rooball training/game
Hiking	Squash training/game
Hockey/Minkey training/game	Swimming training/competition
Jogging	Tennis - singles
Lacrosse/Modcrosse training/game	Touch rugby
Martial arts (Judo, Karate, etc)	Water polo
Netball/Nettaball training/game	

# Appendix C

## Ethics



28 November 2007

Paola Chivers  
 School of Health and Physical Education  
 University of Notre Dame  
 P O Box 1225  
 FREMANTLE WA 6959

Dear Paola

Submission for PhD – Paola Chivers – University of Notre Dame

**PROJECT TITLE: Individual, behavioural and environmental pathways to healthy weight from birth to early adolescence.**

On behalf of the Chief Investigators of the Raine Study we thank you for the opportunity to review your PhD proposal. We are in agreement for you to utilise data previously collected on the Raine Cohort as detailed in your proposal.

Data requests will be made through the Raine Study Co-ordinator and we anticipate that regular contact will be maintained with agreed Raine Investigators.

Yours sincerely

Professor Fiona Stanley AC  
 Director, Teletthon Institute for Child Health Research;  
 Professor, School of Paediatrics and Child Health,  
 The University of Western Australia

Professor Nick de Klerk  
 Head of Division (Biostatistics and Genetic Epidemiology)  
 Teletthon Institute for Child Health Research



14 December 2007

Paola Chivers  
16 Como Court  
Eaton WA 6232

Dear Paola,

I am writing to you in regard to your Application for Ethical Clearance for your proposed research project to be undertaken for the research component of your course at the University of Notre Dame Australia.

The title of this project is: *Individual, behaviour and environmental pathways to healthy weight from birth to adolescence.*

I am pleased to advise that your proposal has been reviewed by the University's Human Research Ethics Committee and has been assessed as having met all expected ethical standards that are relevant to the nature of the intended research and the instrumentation you have chosen to use. Ethical Clearance has been granted for this proposed study.

Should the design of the study, the choice of instrument, or its manner of administration be altered in any significant way as the study progresses, you will be required to provide an update of your clearance application for fresh consideration by the University.

On behalf of the Human Research Ethics Committee, I wish you well with what promises to be a most interesting and valuable study.

Yours sincerely,

**Ms Jaki Creavin**  
Executive Officer,  
Human Research Ethics Committee

cc: Professor Helen Barker, Dean, School of Health Sciences, Fremantle  
Associate Professor Beth Hays, Co-supervisor

**Appendix D**  
**Technical Report CD ROM**

**Appendix E**  
**Linear Mixed Model Development**



Table 1

*Comparison of Higher Order Transformations of Age Models*

Parameter	Model 1	Model 2	Model 3	Model 4	Model 5
	age	age <sup>2</sup>	age <sup>3</sup>	age <sup>4</sup>	age <sup>5</sup>
AIC	32963.958	32355.637	32367.162	32482.669	32609.618
<b>Fixed effects</b>					
Intercept	***	***	***	‘‘	‘‘
Weight Status	*	**	NS	‘‘	‘‘
Gender	***	***	***	‘‘	‘‘
Age	***	***	***	‘‘	‘‘
Age <sup>X</sup>		***	‘‘	‘‘	‘‘
Age*gender	***	NS	*	‘‘	‘‘
Age <sup>X</sup> *gender		**	‘‘	‘‘	‘‘
Age*weight status	***	***	***	***	‘‘
Age <sup>X</sup> *weight status		***	‘‘	‘‘	‘‘
<b>Estimates</b>					
Intercept	15.1(0.04)***	15.5(0.05)***	15.5(.05)***	15.4(.04)***	15.3(0.4)***
Obese	-0.31(0.11)**	0.26(0.11)*	0.10(0.11)NS	0.02(0.11)NS	-0.04(0.11)NS
Overweight	-0.04(0.07)NS	0.25(0.08)**	0.17(0.08)*	0.12(0.08)NS	0.09(0.08)NS
Normal	Ref	Ref	Ref	Ref	

Parameter	Model 1	Model 2	Model 3	Model 4	Model 5
	age	age^2	age^3	age^4	age^5
Gender (F)	-0.31(0.06)***	-0.25(0.06)***	-0.26(0.06)**	-0.27(0.06)***	-0.27(0.06)***
Age	0.02(0.00)***	-0.02(0.00)***	-0.01(0.00)***	-2.1E-3(0.9E-3)*	1.3E-3(8.5E-4)NS
Age^X		2.2E-4(7.8E-6)***	1.0E-7(4.2E-8)*	4.7E-9(1.6E-10)*	2.4E-11(8.6E-13)***
Age*gender	0.01(0.00)***	1.6E-3(1.9E-3)NS	3.2E-3(1.4E-3)*	3.6E-3(1.2E-3)**	3.7E-3(1.1E-3)**
Age^X*gender		2.7E-5(1.0E-5)**	1.0E-7(4.2E-8)*	5.2E-10(2.1E-10)*	2.9E-12(1.1E-12)*
Age*obese	0.07(.00)***	0.03(0.00)***	0.04(0.00)***	0.05(0.00)***	0.05(0.00)***
Age*overweight	0.03(.00)***	0.01(0.00)**	0.02(0.00)***	0.02(0.00)***	0.02(0.00)***
Age*normal	Ref	Ref	Ref	Ref	Ref
Age^X*obese		2.8E-4(1.9E-5)***	1.1E-6(7.8E-8)***	4.9E-9(3.9E-10)***	2.5E-11(2.1E-12)***
Age^X*overweight		1.4E5(1.3E-5)***	5.3E-7(5.4E-8)***	2.4E-9(2.7E-10)***	1.2E-11(1.5E-12)***
Age^X*normal		Ref	Ref	Ref	Ref

*Note.* Standard errors are in parentheses. ^X=the nominated age transformation for that model shown at the top of the column. Variable\*variable indicates an interactions between those variables; F=female; NS=not significant. Ref= the reference group for that variable. '.'=a very small number not reported by output for fixed effects.

\* p<.05, \*\*p<.01, p<.001.

Table 2

*Comparison of Age Transformations Models to Capture the Functional Form*

Parameter	<i>Model 2</i> <i>age<sup>2</sup></i>	Model 6 age <sup>2</sup> and Log(age)	Model 7 age <sup>2</sup> and Sqrt(age)	Model 8 age <sup>2</sup> , log(age) and Sqrt(age) <sup>†</sup>	Model 9 age centred (ageC)and ageC <sup>2</sup> <sup>†</sup>
AIC	32355.637	31003.349	31067.667	30318.473	30457.010
<b>Fixed effects</b>					
Intercept	***	***	***	***	***
Weight Status	**	*	**	NS	***
Gender	***	NS	NS	NS	***
Age	***	***	***	***	***
Age <sup>2</sup>	***	***	***	***	***
log(age)		***		‘‘	
Sqrt(age)			***	‘‘	
Age*gender	NS	***	***	**	NS
Age <sup>2</sup> *gender	**	NS	*	**	*
log(age)*gender		***		‘‘	
Sqrt(age)*gender			***	‘‘	
Age*weight status	***	***	**	***	***
Age <sup>2</sup> *weight status	***	***	***	NS	***

Parameter	Model 2 <i>age</i> <sup>2</sup>	Model 6 age <sup>2</sup> and Log(age)	Model 7 age <sup>2</sup> and Sqrt(age)	Model 8 age <sup>2</sup> , log(age) and Sqrt(age) <sup>†</sup>	Model 9 age centred (ageC)and ageC <sup>2†</sup>
X(age)*weight status		NS		‘‘	
Sqrt(age)*weight status			NS	‘‘	
AR					***
<b>Estimates</b>					
Intercept	15.54(0.05)***	14.12(0.06)***	14.18(0.06)***	13.99(0.06)***	13.37(0.05)***
Obese	0.26(0.11)*	0.30(0.14)*	0.32(0.14)*	0.23(0.14)NS	0.61(0.10)***
Overweight	0.25(0.08)**	0.21(0.09)*	0.22(0.09)*	0.17(0.09)NS	0.34(0.07)***
Normal	<i>Ref</i>	Ref	Ref	Ref	Ref
Gender (F)	-0.25(0.06)***	-0.05(0.07)NS	-0.06(0.07)NS	-0.03(0.07)NS	-0.23(0.05)***
Age	-0.02(0.00)***	-0.12(0.00)***	-0.20(0.00)***	0.48(0.03)***	-0.65(0.02)***
Age <sup>2</sup>	2.2E-4(7.8E-6)***	6.1E-4(1.1E-5)***	7.3E-4(1.4E-5)***	-3.3E-4(4.6E-5)***	0.06(0.00)***
log(age)		1.54(0.03)***		12.69(0.53)***	
sqrt(age)			1.36(0.03)***	-10.11(0.48)***	
Age*gender	1.6E-3(1.9E-3)NS	0.02(0.00)***	0.03(0.01)***	-0.11(0.04)**	0.03(0.02)NS

Parameter	Model 2 <i>age</i> <sup>2</sup>	Model 6 age <sup>2</sup> and Log(age)	Model 7 age <sup>2</sup> and Sqrt(age)	Model 8 age <sup>2</sup> , log(age) and Sqrt(age) <sup>†</sup>	Model 9 age centred (ageC)and ageC <sup>2†</sup>
Age <sup>2</sup> *gender	2.7E-5(1.0E-5)**	-2.7E-5(1.5E-5)NS	-4.2E-5(1.8E-5)*	1.8E-4(6.1E-5)**	3.8E-3(1.5E-3)*
log(age)*gender		-0.22(0.04)***		-2.6(0.70)***	
sqrt(age)*gender			-0.19(0.04)***	2.2(0.63)**	
Age*obese	0.03(0.00)***	0.03(0.01)***	0.03(0.01)**	0.34(0.07)***	0.39(0.04)***
Age*overweight	0.01(0.00)**	5.5E-3(4.5E-3)NS	5.3E-3(7.2E-3)NS	0.17(0.05)***	0.13(0.03)***
Age*normal	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>
Age <sup>2</sup> *obese	2.8E-4(1.9E-5)***	2.7E-4(2.8E-5)***	2.6E-4(3.4E-5)***	-2.3E-4(1.2E-4)*	0.04(0.00)***
Age <sup>2</sup> *overweight	1.4E5(1.3E-5)***	1.4E-4(1.9E-5)***	1.4E-4(2.3E-5)***	-1.2E-4(7.9E-5)NS	0.02(0.00)***
Age <sup>2</sup> *normal	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>	<i>Ref</i>
log(age)*obese		-0.04(0.08)NS		5.89(1.33)***	
log(age)*overweight		0.03(0.06)NS		3.11(0.90)**	
log(age)*normal		<i>Ref</i>		<i>Ref</i>	
sqrt(age)*obese			-0.05(0.07)NS	-5.38(1.21)***	
sqrt(age)*overweight			0.02(0.05)NS	-2.79(0.82)**	

Parameter	Model 2	Model 6	Model 7	Model 8	Model 9
	<i>age</i> <sup>2</sup>	<i>age</i> <sup>2</sup> and Log( <i>age</i> )	<i>age</i> <sup>2</sup> and Sqrt( <i>age</i> )	<i>age</i> <sup>2</sup> , log( <i>age</i> ) and Sqrt( <i>age</i> )†	<i>age</i> centred ( <i>age</i> C)and <i>age</i> C <sup>2</sup> †
Sqrt( <i>age</i> )*normal			Ref	Ref	
AR					3.64(0.05)***

*Note.* Standard errors are in parentheses. <sup>2</sup>=age to the power of two; Log(*age*)=natural log of age; Sqrt(*age*)=square root of age; variable\*variable indicates an interactions between those variables; F=female; NS=not significant; Ref= the reference group for that variable; ‘.’=a very small number not reported by output for fixed effects. AR=adiposity rebound factor, time<1=0, time≥1=0).

\* p<.05, \*\*p<.01, p<.001.

†Although both Model 8 and 9 provided better model fit (AIC), based on null results for fixed effects (Model 8) and the complexity (Model 8 and 9), the decision was made to revert to the simpler model with best model fit.

Table 3

*Results of the Information Criteria Comparison of the Covariance Structure Type for the Final Linear Mixed Model*

Covariance Structure Type	Information Criteria		
	2 Restricted Log Likelihood	Akaike's Information Criterion (AIC)	Schwarz's Bayesian Criterion (BIC)
ARMA (1,1)	32650.874	32656.874	32678.257
Compound Symmetry Correlation Metric	33984.985	33988.985	34003.240
Compound Symmetry	33984.985	33988.985	34003.240
Compound Symmetry Hetrogenous	33064.182	33082.182	33146.331
Diagonal	35215.189	35231.189	35288.210
Factor Analytic Hetrogeneous	31847.131	31879.131	31993.175
First-Order Ante-dependence	31205.815	31235.815	31342.731
First-Order Autoregressive	32667.438	32671.438	32685.694
First-Order Factor Analytic	32592.594	32610.594	32674.744
Hetrogeneous First-Order Autoregressive	31854.966	31872.966	31937.115
Huynh-Feldt†	34384.656	34402.656	34466.806
Scaled Identity	36045.400	36047.400	36054.528
Toeplitz	32631.567	32647.567	32704.589
Heterogeneous Toeplitz	31819.372	31849.372	31956.288
<b>Unstructured correlation metric</b>	<b>30882.625</b>	<b>30954.625</b>	<b>31211.223</b>
<b>Unstructured</b>	<b>30882.625</b>	<b>30954.625</b>	<b>31211.223</b>

Note. Information criteria on model fit where smaller is better form.

† Iteration was terminated but convergence has not been achieved. The MIXED procedure continues despite this warning. Subsequent results produced are based on the last iteration. Validity of the model fit is uncertain.

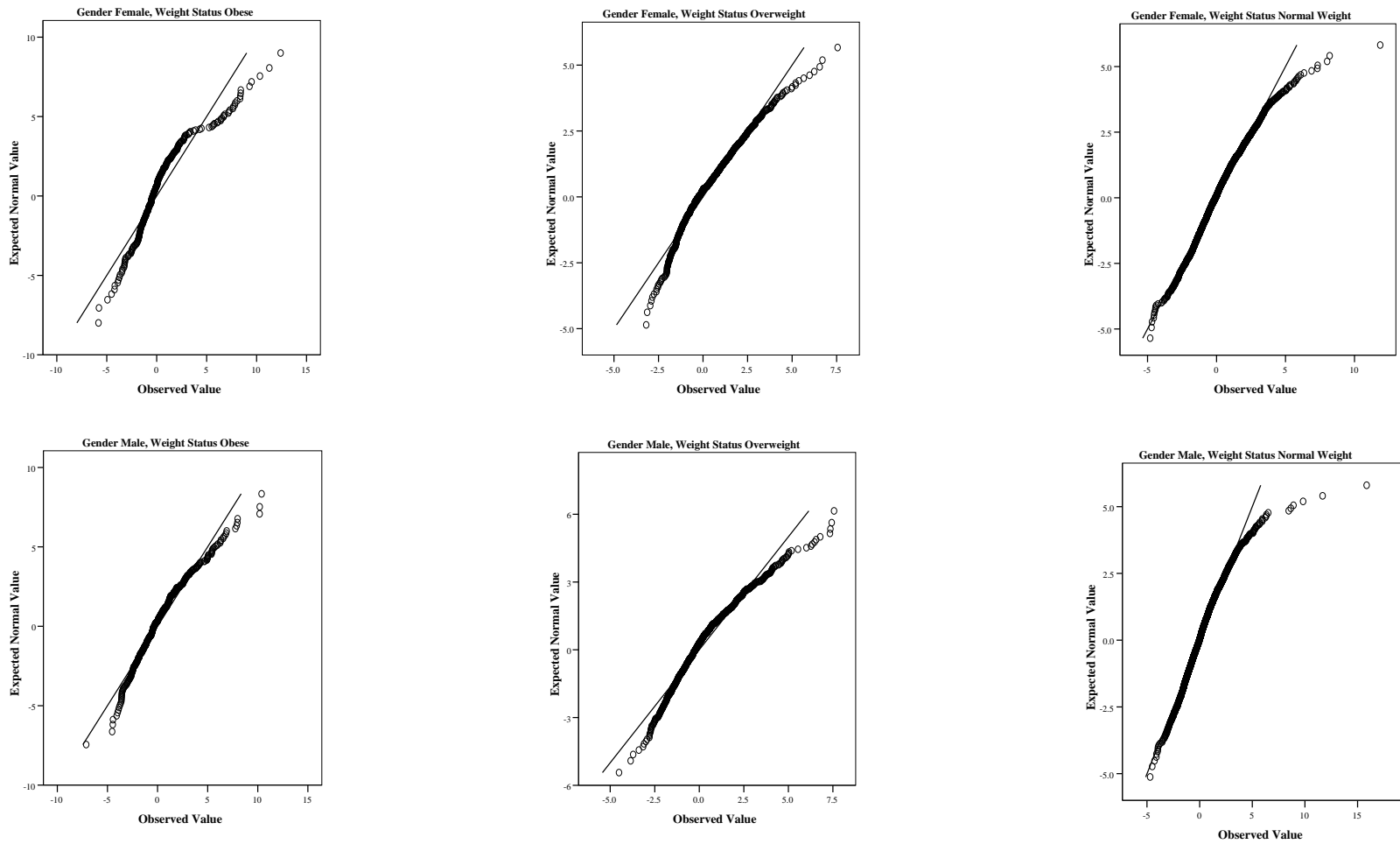


Figure 1. Normal Q-Q plot of residuals for the final BMI linear mixed model, separated by gender and IOTF weight status groups.



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