Individual, behavioural and environmental pathways to adolescent obesity

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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, energy balance = energy intake – 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 Organisation (2006) report that in 2005, globally, there were about 1.6 billion overweight adults (based on BMI ≥25), with at least 400 million adults considered obese (BMI ≥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 (10th 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).\(^1\)

\(^1\) 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>
<thead>
<tr>
<th>Factor</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>Krebs, et al., 2003</td>
</tr>
<tr>
<td>Bullying and social exclusion</td>
<td>Centre for Community Child Health, 2007; Krebs, et al., 2003; Gillman, Rifas-Shiman, Camargo Jr, &amp; Berkey, 2001</td>
</tr>
<tr>
<td>Factor</td>
<td>Source</td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Hepatic disorders</td>
<td>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</td>
</tr>
<tr>
<td>High blood pressure / hypertension</td>
<td>Dietz, 1998; Guo &amp; Chumlea, 1999; Access Economics, 2006; Dehghan, Akhtar-Danesh, &amp; Merchant, 2005; Krebs, et al., 2003; Baur, 2002; Olds et al., 2004</td>
</tr>
<tr>
<td>Hyperinsulinemia</td>
<td>Dietz, 1998; Olds et al., 2004</td>
</tr>
<tr>
<td>Infertility</td>
<td>Dehghan, Akhtar-Danesh, &amp; Merchant, 2005</td>
</tr>
<tr>
<td>Lower growth hormone secretion</td>
<td>Dollman, J., Ridley, K., Magarey, A. and Hemphill, E., 2007; Olds et al., 2004</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>(Ford, Kohl, Mokdad, &amp; Ajani, 2005; Huang et al., 2009; Vanhala, Vanhala, Kumpusalo, Halonen, &amp; Takala, 1998)</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>Access Economics, 2006</td>
</tr>
<tr>
<td>Polycystic Ovary Disease</td>
<td>Dietz, 1998</td>
</tr>
<tr>
<td>Sleep disorders</td>
<td>Dietz, 1998; Centre for Community Child Health, 2007; Krebs, et al., 2003; Baur, 2002</td>
</tr>
</tbody>
</table>
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\(^2\), 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

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

<table>
<thead>
<tr>
<th>Factors reported in the aetiology of obesity</th>
</tr>
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<tbody>
<tr>
<td>Adiposity rebound³ (timing and BMI) (4)</td>
</tr>
<tr>
<td>Birth weight (4,16)</td>
</tr>
<tr>
<td>Breastfeeding duration / formula feeding (5,11,16)</td>
</tr>
<tr>
<td>Diet / nutrition (4,5,6,10,11)</td>
</tr>
<tr>
<td>Early maturation (5,7)</td>
</tr>
<tr>
<td>Ethnicity (4)</td>
</tr>
<tr>
<td>Genetics (4,5,13,14)</td>
</tr>
<tr>
<td>Income (9)</td>
</tr>
<tr>
<td>Maternal age (1)</td>
</tr>
<tr>
<td>Parent’s education (9)</td>
</tr>
<tr>
<td>Parental feeding style (4)</td>
</tr>
<tr>
<td>Parental weight status (1,2,3,4,5,6)</td>
</tr>
<tr>
<td>Physical activity (4,10,11,12)</td>
</tr>
<tr>
<td>Prenatal environment (1,15)</td>
</tr>
<tr>
<td>Rapid weight gain (1)</td>
</tr>
<tr>
<td>Sedentary activity (1,3,4,5,9,10,11,12,17)</td>
</tr>
<tr>
<td>SES (4,5,8)</td>
</tr>
<tr>
<td>Sleep duration (4)</td>
</tr>
<tr>
<td>Smoking during pregnancy (11)</td>
</tr>
</tbody>
</table>


³ 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.

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.
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, Guilloud-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 diary, 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” (Stoddan 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 KTK⁴). 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, 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’.

⁴
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, Sothern, 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 10th, but by 1994 it is ranked second, retaining this rank in 2000. For boys, TV was ranked 2nd in 1994, but was 4th 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 (≥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
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

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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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**

<table>
<thead>
<tr>
<th>Description</th>
<th>Advantages</th>
<th>Disadvantages</th>
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<tbody>
<tr>
<td>BMI</td>
<td>Ratio of height over weight.</td>
<td></td>
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<tr>
<td>Skinfold</td>
<td>Indirect measure of subcutaneous adipose tissue, usually over a number of body sites.</td>
<td></td>
</tr>
<tr>
<td>Waist Girth</td>
<td>Measurement of waist circumference.</td>
<td></td>
</tr>
<tr>
<td>Hip Girth</td>
<td>Measurement of hip circumference.</td>
<td></td>
</tr>
<tr>
<td>WHTR</td>
<td>Ratio of waist girth over height.</td>
<td></td>
</tr>
<tr>
<td>WHR</td>
<td>Ratio of waist girth over hip girth.</td>
<td></td>
</tr>
</tbody>
</table>

#### Advantages
- Most common indicator for adiposity.
- Reliable
- Easy to measure.
- Cheap to measure.
- International recognised cut-points (Cole et al., 2000).
- BMI-for-age z-score option.
- High correlation with fat mass.
- High correlation with DXA.
- High correlation with skinfold measures.
- Similar to waist girth.

#### Disadvantages
- 12 site measurement provides similar results to magnetic resonance imaging.
- Good predictor of percentage fat.
- Triceps, subscapular, check and abdominal are best predictors.
- Sum of skinfolds correlated with BMI.
- Simple and better estimation of central fatness than BMI.
- Correlates with DXA fat distribution.
- Associated with metabolic risk.
- Measures central subcutaneous and visceral fat.
- Associated with a number of adverse health outcomes.
- Better predictor of cardiovascular risk compared to BMI.
- Less gender specific.
- Measures overall fat distribution.
- Associated with a number of adverse health outcomes.
- Similar predictive ability to BMI.
- High correlation with BMI.
- Equivalent cut-point to BMI cut-points.
- Simpler and better associations with metabolic risk.
- Does not require age and gender centiles.
- A better measure of weight status than BMI.
- Cut-points exist.
<table>
<thead>
<tr>
<th>BMI</th>
<th>Skinfold</th>
<th>Waist Girth</th>
<th>Hip Girth</th>
<th>WHtR</th>
<th>WHR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disadvantages</td>
<td>• Limitations in children (growth spurts, gender, maturity).</td>
<td>• Measurements are highly investigator dependent.</td>
<td>• Does not have accepted cut-points.</td>
<td>• Reproducibility good but lower than BMI.</td>
<td>• Similar limitations to BMI.</td>
</tr>
<tr>
<td></td>
<td>• Underestimates obesity prevalence.</td>
<td>• Not usually recommended for obese.</td>
<td>• Different measurement protocols used internationally.</td>
<td></td>
<td>• Prognostic value in children low.</td>
</tr>
<tr>
<td></td>
<td>• Less accurate in normal weight.</td>
<td>• Skinfold equations tend to underestimate body fat.</td>
<td></td>
<td></td>
<td>• Does not appear to accurately reflect intra-abdominal fat mass.</td>
</tr>
<tr>
<td></td>
<td>• No age and gender cut-points before age two years.</td>
<td>• Prediction equations not suitable in children.</td>
<td></td>
<td></td>
<td>• Value as health risk predictor unclear.</td>
</tr>
<tr>
<td></td>
<td>• Problems with self report measures, typically underestimated.</td>
<td>• Misclassification problematic.</td>
<td></td>
<td></td>
<td>• Does not have accepted cut-points.</td>
</tr>
</tbody>
</table>

(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, Knower, & 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, Raatz, Moran, Hong, & Sinaiko, 2005; Taylor, Jones, Williams, & Goulding, 2002; te Velde et al., 2007; Tremblay, Kastzmarzy, & Willms, 2002; Yarnell, Patterson, Thomas, & Sweetnam, 2001)
Population studies commonly use body mass index (BMI), a weight for height ratio \( \text{wt/ht}^2 \) 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 kg/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\(^{th}\) percentile, healthy weight 5\(^{th}\) – 85\(^{th}\) percentile, overweight 85\(^{th}\) – 95\(^{th}\) percentile, and obese ≥95\(^{th}\) 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 Resarch 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.
"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