Factors associated with weight gain and the development of obesity

An overview of existing scientific literature

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Executive summary

The prevalence of obesity continues to rise within Australia and it remains a serious public health problem. Obesity results from a prolonged period of energy imbalance where the energy intake from food and drink exceeds energy expenditure for metabolic processes and physical activity. Excess energy is stored as fat within the body and is associated with a wide range of health, social and psychological problems.

Energy balance within the body is usually well regulated by a range of physiological responses that monitor food intake, metabolism and storage and send signals to influence appetite and, to a lesser extent, energy expenditure. The body attempts to protect existing weight status by making small adjustments but once this capacity is overwhelmed by persisting energy excess, weight gain will begin and usually continue until a new weight results in increased energy expenditure (metabolism) and the re-establishment of the energy balance. This new weight will then be defended.

The number and extent of the signals which encourage increased food intake greatly outweigh those that promote satiety. Thus energy balance will be more difficult to achieve in individuals who have smaller energy requirements and those with a smaller food intake, as they are more reliant on the weaker physiological mechanisms that influence satiety.

Although there has been substantial effort invested in identifying the determinants of obesity, we still have an incomplete understanding of all the drivers of obesity. It is clear that diet and physical activity are the central elements of the energy balance equation. However, in contrast to popular belief, obesity is not solely caused by a lack of cognitive control over personal dietary and physical activity behaviours. It is now understood that physiology and genetics have a critical role in driving or attenuating these behaviours and the physical, social and political environment in which we live greater influences our ability to maintain behaviours appropriate to weight control.

A number of analyses have attempted to define the key determinants of obesity and there remains a degree of controversy over which factors have made the greatest contribution to the recent rise in the rates of obesity in Australia today. What is accepted is that there is
no simple single cause of obesity and that a complex array of interacting factors is driving changes in weight status at a societal level. The complexity and inter-relationship between the diverse drivers of obesity is best represented by the ‘obesity systems map’ which was produced by the Foresight Programme of the UK Government Office for Science. At the core (or engine) of the map is energy balance surrounded by variables that directly or indirectly influence this key process.

Food intake and physical activity behaviours are the two key factors that have potential to directly influence energy balance and weight status and much discussion has revolved around the relative influence of specific behaviours. Key dietary behaviour linked to creating energy excesses include: a high intake and increasing portion size of energy-dense foods, especially high fat foods, snack foods and food eaten away from home; a low intake of high fibre, low energy-dense foods, especially vegetables and fruit; and a high consumption of sugar-sweetened beverages.

Physical activity changes over time are hard to document but it appears that leisure time physical activity has not declined substantially over the last three decades, while there have been substantial reductions in occupational and incidental physical activity. At the same time, the amount of time spent sitting or being sedentary has increased dramatically.

The changing social, political and physical environment in which we live tends to encourage obesity-promoting behaviours and discourage appropriate eating and physical activity behaviours. Urban design and the built environment discourage physical activity and active travel and influence the ease of access to appropriate food. Changes to the food supply have led to the wide availability of cheap, high kilojoule processed foods that are aggressively marketed. The portion size of snack foods, sweetened drinks and takeaway foods has increased and their relative cost has decreased, while the relative cost of fresh produce has increased. Changes to occupational structures and work environments have led to the replacement of physically active workplaces with desk-bound and sedentary occupations. In addition, longer working hours leave less time for food preparation and family recreation and physical activity. Disrupted sleep patterns exacerbate the lack of time for planned activity and food preparation.
Further, powerful genetic and physiological processes which are geared to the accretion of energy for survival undermine attempts to limit energy intake and increase energy expenditure. In particular, our genes and nutrition exposures in utero and early in life have profound effects on our regulation of energy balance and how and where we store fat generated by excess kilojoules.

There are certain times in a person’s life when they are more prone to weight gain and thus require special focus in addressing the problem. Critical life stages for weight gain include: prenatal; time of adiposity rebound (5–7 years); adolescence; early adulthood; pregnancy; and menopause.

In addition, there are some specific groups identified at higher risk of weight gain. These include: those with a family history of weight problems; the socio-economically disadvantaged; Indigenous Australians and certain minority ethnic groups; recent successful weight reducers; and recent past smokers.
1. The project brief

The Boden Institute within the University of Sydney was contracted by RaggAhmed to produce an outline of the key factors associated with weight gain and development of obesity. This was part of a larger project being undertaken by RaggAhmed to assist the Department of Health and Ageing with the development of a healthy weight guide.

The objective of this brief report was not to produce a comprehensive analysis of a plethora of factors implicated in the aetiology of obesity but rather to focus on a brief description of the key drivers of obesity. In addition, the report addresses the physiology of weight gain and the critical life stages and high risk groups in which weight gain is more common.

1.1. The process in generating this report

This report is not intended to a comprehensive or systematic review of the literature but rather a summary of the current state of knowledge. No systematic search strategy was employed and instead, information was drawn from a range of existing reports and comprehensive reviews of the literature on this issue, including work from:

- World Health Organization
- World Cancer Research Fund
- US Institute of Medicine
- The UK Foresight Project
- The International Obesity Task Force
- National Preventative Health Taskforce (Australia)

In addition, significant reviews and individual papers have been sourced.
2. The problem of obesity in Australia

During the last 100 years there have been enormous advances in the health status of most Australians. Better nutrition, improved living conditions and advances in therapeutic options have led to gains in infant, child and adult health. Today most Australians can expect to live an average of 80 years, most of them relatively healthy. As the impact of infectious diseases and under-nutrition has diminished in Australia, these conditions are being replaced by chronic diseases of over-nutrition as the greatest cause of morbidity and premature mortality. Although there has been improvement in the management of these chronic diseases, their impact on the health of Australians remains significant and is likely to remain so for some time.

It is clear that the lifestyles of Australians today are closely associated with the rise in chronic disease, and the increasing levels of overweight and obesity in Australia have been implicated as a major contributor. A large increase in obesity rates among Australians has the potential to erode many of the recent health gains, as excessive fatness has been associated with a wide range of chronic and debilitating illnesses such as diabetes, heart disease, some cancers, sleep apnoea, and osteoarthritis. It is now clear that there are numerous health benefits to be gained by individuals and the community as a whole by maintaining a healthy weight throughout life and preventing excessive weight gain.

Urgent action is required to help address the problem of increasing levels of overweight and obesity but this action needs to be well planned, targeted and based on a good understanding of the problem in the community. It is also important that interventions avoid contributing to any existing problems of under-nutrition and disturbed eating in the community.

2.1. Definition of obesity

Measuring weight may be relatively simple but defining when it has become a health risk is more difficult. Obesity can be defined as a condition of excess body fat where the fat has accumulated to an extent that it is likely to be detrimental to health (WHO 2000). However, obese individuals are not all the same; they vary considerably in the degree of
excess body fat, the distribution of the fat within the body, and the health risks associated with the excess fat.

The two most useful measures for characterising excessive fat are body mass index (BMI) and waist circumference. BMI is calculated from a person’s weight and height and gives a reasonable estimate of total adiposity. A BMI between 18.5 and 25 kg/m$^2$ is considered acceptable, while a BMI equal or greater than 30 is usually considered to indicate obesity. A waist circumference measurement gives an indication of the amount of fat stored within the abdomen, and this distribution of fat has been associated with worse health outcomes. In males, a waist circumference equal or greater than 102 cm is usually considered very high risk, while in females the corresponding value is 88 cm.

Defining weight status in children is more difficult, as children’s height and weight and the relationship between them varies as they grow and develop. Therefore, BMI for age percentiles based on standard growth curves are used in most countries to define weight status, with the 85th percentile equivalent to overweight and the 95th and above percentiles equivalent to obesity. Recently an international reference population has been developed and cut-off points have been devised based on percentiles equivalent to a BMI of 25 and 20 in adults. This is now widely used to define the prevalence of overweight and obesity in children at a population level (Lahti-Koski and Gill 2004).

2.2. Obesity and weight gain as a health problem

There is a very strong association between increasing BMI and premature death and the risk of developing a number of chronic non-communicable diseases such as type 2 diabetes, cardiovascular disease, gallbladder disease, and certain types of cancer. Obesity is also associated with a range of debilitating conditions, which can drastically reduce quality of life. These include arthritis, back pain, respiratory difficulties, skin problems and sleep apnoea. Excessive body weight is also frequently associated with psychosocial problems, many of which appear to result from the negative cultural bias and prejudice directed against obese people in Australia (Catford and Caterson 2003).
In 2005, overweight and obese Australian adults cost the Australian economy $21 billion in direct healthcare and direct non-healthcare costs, plus an additional $35.6 billion in government subsidies (Colagiuri et al. 2010).

2.3. The current problem of obesity

Results from 2007–08 National Health Survey (NHS) revealed that in 2007–08, 61.4% of the Australian population were either overweight or obese, with 42.1% of adult males and 30.9% of adult females classified as overweight and 25.6% of males and 24% of females classified as obese (ABS 2008). Aboriginal and Torres Strait Islander Australians are 1.9 times as likely as non-Indigenous Australians to be obese. The same survey also found that 24.9% of children aged 5–17 years (25.8% of boys and 24.0% of girls) were either overweight or obese (ABS 2008).

2.4. Current and projected weight gain in Australians

Data from the cohort follow-up within the AusDiab study indicated that the average weight gain of Australian adults aged 25 years and older was 1.4 kg over the five-year period (AusDiab 2006). However, this ranged from a mean weight gain of 3.5 kg (or 0.7 kg per year) in those aged 25–34 and 2.5 kg (0.5 kg per year) in those aged 35–44 to a loss of 2.2 kg (0.44 kg per year) in those aged over 75 years.

The prevalence of obesity in Australia has more than doubled in the past 20 years, being 2.5 times higher now than in 1980. For males, the prevalence of overweight and obesity increased from 39% in 1980 to 67% in 2008, and for females, the prevalence of overweight and obesity increased from 28% to 64% in the same time period (ABS 2008). This is supported by self-reported data from the National Health Surveys, in which data has been collected more regularly. Figure 2.1 shows a consistent increase in levels of overweight and obesity from 1980 through to 2008.
Self-reported height and weight, 18 years plus, various NHS surveys

Figure 2.1 Trends in BMI in Australia Self-reported data 1980–2008

The projected progression of the prevalence of obesity in Australia will mean that if the current rates of weight gain continue, then 42.4% of adults will be overweight and 33.9% will be obese by 2025, and only 28.1% will be an acceptable weight (Walls et al. 2010).
3. Energy balance and the regulation of weight

Energy imbalance is the root cause of changes in weight status, but significant weight gain and obesity occur only when energy intake from food has exceeded energy expenditure from physical activity and metabolic processes over a considerable period of time. A complex and diverse range of factors can give rise to a positive energy balance but it is the interaction between a number of these influences rather than any single factor acting alone that is thought to be responsible. Discussions on the genesis of obesity often focus on a lack of cognitive control over personal dietary and physical activity behaviours and ignore the critical role played by physiological processes in driving or attenuating these behaviours.

3.1. Energy balance

Despite its critical function in weight status, the mechanisms which influence the achievement of energy balance are not completely understood. Energy status in humans is often represented as a simple equation governed by the first law of thermodynamics (that energy can neither be created nor destroyed).

\[ \text{Energy intake} = \text{energy expenditure} + \text{energy stores} \]

Energy status is considered in ‘balance’ when energy intake (EI) = energy expenditure (EE), in deficit when EI < EE and in surplus when EI > EE. Energy balance results in stable body energy stores (and thus body fat and weight) while an energy deficit will create a weight loss and an energy surplus will lead to weight gain.
Figure 3.1 Common representation of energy balance

In most people, however, body weight remains much more stable over time than would be predicted from this simple model of energy balance, as individuals have considerable variations in energy intake and expenditure on a daily basis. It is now clear that there are a range of metabolic processes that act to try and keep energy status in balance by producing compensatory changes in energy intake or expenditure. When energy intake decreases, changes occur which create small decreases in energy expenditure; when energy intake increases, there are small increases in energy expenditure. There is also a wide array of biological systems which attempt to regulate energy balance by creating signals to encourage changes in energy intake as energy expenditure increases or decreases (Hill 1998). However, these regulatory processes are capable of only small adaptations. They evolved to deal with a different food and physical activity environment and work best in a situation of high energy expenditure and restricted opportunity for energy intake.
3.2. Physiological regulation of weight

The maintenance of energy balance involves various processes including satiety responses, appetite control systems, and other homeostatic mechanisms. Energy balance and ultimately, body fat, is regulated by a negative feedback system in which the body responds to changes in energy stores (i.e., body fat) by changes in appetite, energy intake and energy expenditure. These physiological mechanisms are incompletely understood but it is known that signalling mechanisms within the intestine, adipose tissue, brain and other tissues sense food intake, metabolism and storage. Key to the regulation of these systems are hormones with energy-regulating roles (e.g. ghrelin, leptin, insulin) that sense changes in energy intake and stores and convey information to central processing centres in the brain. This results in increased or reduced secretion of neurotransmitters (e.g. neuropeptide Y, agouti-related protein) within these sections of the brain which stimulate or suppress energy intake and energy expenditure.

3.3. Asymmetry in appetite control

Physical inactivity influences weight gain through a variety of processes but its strongest impact is on the body’s ability to more effectively regulate appetite and energy intake at higher energy expenditure levels. Prentice and Jebb (2004) suggested that physically active people have higher energy needs and thus above-average food energy intake, allowing hunger signals (which are strong) to drive food intake up to the levels needed. Inactive people, however, tend to have energy needs below the norm for food consumption, so their energy homeostasis relies on physiologically inefficient satiety signals to try and limit their energy intake. These weaker satiety signals are further confounded by diets that are very energy-dense, leading to more kilojoules being consumed before signals to cease eating become effective.

3.4. The process of weight gain

Unfortunately, this physiological regulation is insufficient to maintain energy balance, and weight gain occurs in the face of an energy surplus that is sufficiently large and sustained for long periods. It is not well defined how much of an energy surplus is required to initiate
weight gain but it is known that the relationship between excess energy and the amount of body energy stored in fat is not linear or direct. When energy intake increases above expenditure, weight gain occurs but does not continue indefinitely. This is partly because as new adipose tissue is created, the energy cost of maintaining that new tissue also increases, thus decreasing the overall energy surplus. Over a period of time, the increases in weight will lead to a situation where the increased energy expenditure has totally eroded the energy surplus and a new equilibrium is created (see Figure 3.2).

Figure 3.2 The dynamic equilibrium of weight gain

This new energy balance at a higher body weight will be defended by the same physiological mechanisms that sought to maintain energy balance at the lower body weight, and these will defend against weight loss by creating adaptations to an energy deficit. Further increases in energy intake or decreases in energy expenditure will lead to another round of weight gain which will continue until a new energy balance is reached at an even higher body weight. Thus, slow but regular weight gain occurs in cycles which are hard to reverse without considerable intervention.
3.5. Patterns of weight gain

Weight gain and obesity may develop after a prolonged period of a small energy imbalance but it is also possible that it may be a result of periodic bouts of large, positive energy balance, achieved by temporary increases in intake or decreases in physical activity. There are few longitudinal studies that have assessed weight status regularly enough to define the true pattern of weight gain and this is likely to vary from individual to individual; however, small studies have shown that a large proportion of weight gain can occur in a short period of the year, with more weight gain occurring over celebratory periods such as Christmas (Schoeller, 2009).
4. The drivers of weight gain

Physiological energy regulation mechanisms operate within each person to keep weight and body fat stores stable in the long term. However, powerful societal and environmental forces influence energy intake and expenditure through effects on dietary and physical activity patterns, and may overwhelm the physiological control of body weight. The susceptibility of individuals to these forces is influenced by genetic and other biological factors such as gender, age and hormonal activities, over which they have little or no control.

The breadth of these ‘drivers of weight gain’ is addressed in this section but discussed in more detail in later sections.

4.1. Influences on energy balance and weight gain

A number of analyses have attempted to define the key determinants of obesity and there remains a degree of controversy over which factors have made the greatest contribution to the recent rise in the rates of obesity in Australia. Comprehensive assessment of the situation has been undertaken by the World Health Organization in the *Expert Report on Diet, Nutrition and the Prevention of Chronic Disease* (WHO 2003) and the World Cancer Research Fund report *Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective* (WCRF 2007).

These reports examined the current literature and identified a range of key factors which either increase or decrease the risk of weight gain and the development of obesity. The results of both assessments are summarised in Table 4.1.
Table 4.1 Summary of the strengths of evidence on behaviours that might promote or protect against weight gain and obesity – agreement from WHO 2003 and WCRF 2007 reports

<table>
<thead>
<tr>
<th>Evidence</th>
<th>Decreases risk</th>
<th>Increases risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rated convincing or likely in both reports</td>
<td>Regular physical activity</td>
<td>Sedentary lifestyles</td>
</tr>
<tr>
<td></td>
<td>High intake of low energy-dense foods*</td>
<td>* High intake of energy-dense foods</td>
</tr>
<tr>
<td>Rated probable or possible in both reports</td>
<td>High dietary fibre intake</td>
<td>Sugar-sweetened soft drinks and juices</td>
</tr>
<tr>
<td></td>
<td>Promoting linear growth</td>
<td>High proportion of food prepared outside of homes</td>
</tr>
<tr>
<td></td>
<td>Breastfeeding</td>
<td>High exposure to television (marketing)</td>
</tr>
<tr>
<td>Rated possible in one report only</td>
<td>Low glycaemic index foods</td>
<td>Adverse social and economic conditions in developed countries (especially for women)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Large portion sizes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rigid restraint/periodic disinhibition eating patterns</td>
</tr>
<tr>
<td>Rated insufficient</td>
<td>Increased eating frequency</td>
<td>Alcohol</td>
</tr>
</tbody>
</table>

* Energy-dense foods are high in fat/sugar, and energy-dilute foods are high in fibre and water such as vegetables, fruits, legumes and whole grain cereals. Source: Developed from WHO 2003 and WCRF 2007.

4.2. The complex web

Although the WHO and WCRF reviews touched upon the array of behavioural and environmental influences in the aetiology of obesity at an individual and population level, they did not capture the interplay of these factors and how they operate at a societal level. One of the first attempts to represent the nature of the prevailing, multi-layered environmental factors that influence energy balance in the modern world was the International Obesity Task Force ‘causal web’ (Kumanyika 2001). The causal web illustrated that although food intake and energy expenditure ultimately influence energy balance, there are an array of forces operating at different layers of society that impact directly or indirectly upon these behaviours (Figure 4.1). The implications of this representation are apparent. Addressing obesity prevention will require action at many levels and must include a focus on many of the distal factors that influence our food and activity environment.
Societal policies and processes influencing the population prevalence of obesity

**Figure 4.1 The IOTF Causal web**
The Foresight obesity system map

Although the causal web suggests that the genesis of and thus, the solution to obesity is complicated, its linear format does not clearly illustrate the complexity of the interactions between the various layers. The Foresight Programme of the UK Government Office for Science expanded on the causal web approach by using a systems approach to produce a complex conceptual model with 108 variables known as the ‘obesity systems map’ (Vandenbroek et al. 2007). The relationships between the variables are illustrated with more than 300 solid or dashed lines to indicate positive and negative influences. All the variables are interconnected and these connections give rise to feedback loops. At the core (or engine) of the map is energy balance, surrounded by variables that directly or indirectly influence this key process.

The Foresight map approach to defining the broad-ranging drivers of obesity and the inter-relationships between these factors has not met with universal approval. It has been criticised for being overly complex to the extent that it creates a sense of confusion and despair, when clarity is what is needed to effectively address this problem. Others have also questioned how comprehensive such a map could be, in that it implies that all potential drivers of obesity are captured within the map. It is probably true that very few people completely understand all the intricacies of the system it describes and that the map is not truly complete, as it reflects only the perceptions of the stakeholders engaged in its development. However, it has served some useful objectives in improving our perception of the nature of the obesity problem and the approach that will be required to successfully address it. Some of the principles that the Foresight map and process have reinforced are:

- the wide range of political, social, environmental, behavioural and physiological factors that influence individuals’ and society’s capacity to achieve energy balance, and the complex, multifactorial nature of the systems that give rise to obesity
- the breadth of action that will be required to restore energy balance
• the futility of attempting to address obesity by focusing attention solely on individual behaviour change or within one domain of action

• the need to consider the interaction between factors that either enable or amplify, or conversely, inhibit the behaviour change process required to achieve energy balance

• the interaction between factors within the system, which is currently driving energy accretion and disrupting individual efforts to achieve energy balance.

Figure 4.2 Domains of action within the Foresight obesity system map
5. **Behavioural factors**

Food intake and physical activity behaviours are the two key factors that have potential to directly influence energy balance and weight status. Historically they have been considered a product of free will under the direct cognitive control of the individual. However, as previous sections have indicated, there are a range of biological as well as social and environmental forces that constrain these behaviours in individuals. However, an appreciation of the dietary and activity behaviours that have been linked to weight gain and the development of obesity is important if we are to usefully define these problems and decide how best to address them.

Both energy expenditure and energy intake contribute to weight gain and the development of obesity, and it is not possible to clearly apportion the contribution that each makes to the problem. There has been a lot of unnecessary debate over which factor is more important in the genesis of obesity. Attempts to selectively promote one factor over the other as the major cause are counterproductive, as both will need to be addressed in tackling the problem.

5.1. **The formation of habits**

When a behaviour relating to food intake or activity is repeated often for a long period of time, it becomes a habit, meaning that it becomes almost an automatic response to certain cues or situations. Habits often remain well after the original reason for adoption of the behaviour has passed, making them difficult to change. Often, people passively adopt or continue a behaviour rather than making an active decision to do so. Once habits are formed, individuals show little inclination to change them. In addition, attitudes and intentions have less of an impact when a habit has been established, making changes to inappropriate food and activity behaviours less likely even when the need for such a change is accepted (Vandenbroeck 2007).

Food and activity habits are often associated with an increased energy intake, and as environments become more ‘obesogenic’ (obesity-promoting), the behaviours that lead to obesity are increasingly the default or automatic ones.
5.2. Dietary behaviours

A number of dietary factors have been identified as potential contributors to weight gain and obesity by undermining the innate regulatory control of body weight. There are multiple mechanisms by which this can occur, including satiety, palatability, food availability or low energy needs as a consequence of physical inactivity. Despite great interest in this area, evidence linking specific dietary behaviours to weight gain is limited and is largely restricted to observational and intervention studies.

5.2.1 Energy density

Although surplus energy intake is an obvious cause of weight gain, there is little evidence to link high kilojoule intake directly to obesity. This may be the result of large individual variability in energy requirements, with higher requirements often being associated with higher levels of activity. In addition, total kilojoule intake is not reported accurately or consistently in most dietary surveys. This has led to a focus on the role of the energy density (i.e., the energy or kilojoules per 100 g) of food on weight gain. The theory is that energy-dense foods undermine appetite and food intake regulatory systems, leading to the ‘passive overconsumption’ of kilojoules as individuals do not adjust the volume of food in response to its higher kilojoule content. There is strong experimental and observation evidence to support this hypothesis (WCRF 2007, WHO Euro 2007).

5.2.2 Macronutrient composition of the diet

Fat

Dietary fat is readily stored as body fat, with minimal energy costs of conversion relative to protein or carbohydrate. Fat is less satiating than iso-energetic quantities of other nutrients, and habitual consumption of a high fat diet may down-regulate some elements of the appetite control system. Early studies suggested that those who consumed a high fat diet were more at risk of weight gain but the data from prospective studies is inconsistent. Intervention studies show modest but significant spontaneous weight loss in people who reduce the fat content of their diet. The specific influence of fat on weight gain
is believed to be largely mediated through the high energy density of fat and fat-containing foods (WHO 2000, Swinburn et al. 2004).

It has been suggested that different types of fat have different effects on the risk of weight gain. Animal studies suggest that saturated fatty acids may be preferentially stored, while more unsaturated fats are more likely to be oxidised. However, there is insufficient research on this issue in humans for any firm conclusions (WHO Euro 2007).

**Carbohydrates and glycaemic index**

The proportion of carbohydrate in the diet tends to vary reciprocally with fat and it is difficult to segregate the impact of the total amount of carbohydrate in the diet from total fat. While some studies show a protective effect of eating a diet containing a high proportion of carbohydrate, many others, particularly studies of children, show no such association. The evidence relating the intake of sugar, per se, to weight change is inconsistent. In part, this may be due to the various sources of sugar in the diet, including fruit and milk as well as ‘added’ sucrose (Jebb 2007, WCRF 2007).

Currently, there is particular interest in the glycaemic index of the diet. A recent systematic review found a positive association between a high glycaemic index and weight gain, and experimental studies show that food with a low glycaemic index is associated with increased satiety and short-term reductions in energy intake. However, results from longer-term intervention studies have been inconsistent (WHO Euro 2007, Jebb 2007).

**Fibre**

A number of reviews have shown an inverse association between fibre intake and weight gain (although their definitions of dietary fibre are somewhat variable). Intervention studies also show that a high intake of dietary fibre may assist in weight loss. There are many possible mechanisms for this effect, with the influences of fibre on nutrient absorption and appetite regulation processes already established. Intake of wholegrain foods has also been negatively associated with BMI and weight gain, although the size of this effect is usually attenuated after adjustment for the fibre content of the diet (WHO 2003, Swinburn et al. 2004).
Protein

Interest in the effect of protein on weight gain has arisen out of intervention studies that have shown improved weight loss maintenance with high protein diets. There are also animal studies which suggest a specific appetite for protein with low protein diets, leading to increased energy intake so as to achieve the animals’ usual protein requirement. However, data in humans on protein and weight gain remains too limited to enable any firm conclusions (Jebb 2007, WHO Euro 2007).

Alcohol

The exact relationship between alcohol and weight gain remains unclear. In observational studies, high alcohol intake is not associated with increased body weight, although experimental studies suggest that energy from alcohol supplements rather than substitutes for food energy. However, there are many confounding socio-economic and demographic factors that influence the consumption of alcohol (Swinburn et al. 2005, WHO Euro 2007).

5.2.3 Fruit and vegetable consumption

Given the emphasis placed on fruit and vegetables in dietary recommendations, surprisingly few studies have reported on the relationship between fruit and vegetable consumption and weight change. Results from prospective studies have been conflicting but the largest and more recent studies suggest that the consumption of fruit and vegetables may have a modest protective effect against weight gain (Jebb 2007).

5.2.4 Consumption of sugar-sweetened beverages

The association between consumption of sugar-sweetened beverages and weight gain has been the focus of a number of systematic reviews. The findings remain controversial but generally support a firm link between increased consumption of sugar-sweetened drinks and increased risk of weight gain and obesity. In short-term intervention studies, energy consumed in liquid form appears to supplement habitual food intake, leading to increases in body weight (WHO 2003, Jebb 2007).
5.2.5 Portion size

Habituation to large portion sizes is likely to be a risk factor, although this has been poorly studied in large cohorts, which frequently rely on food frequency questionnaires to collect dietary data. However, experimental studies suggest that large portions tend to increase energy intake at a meal, with no increase in satiety and little compensation at subsequent eating episodes (Swinburn et al. 2004).

5.2.6 Increased consumption of takeaway foods and foods prepared away from home

There is now reasonable evidence linking the consumption of fast food to weight gain and obesity, and a number of studies have consistently documented that fast food consumption is associated with an increase in total energy intake (Rosenheck 2008). Higher energy density and portion size in takeaway foods has been proposed as the mechanism for this relationship, however the associations have been found with the frequency of eating in fast food venues rather than with the foods chosen.

5.2.7 Eating frequency

A number of early consumption studies suggested an inverse relationship between the number of daily meals and obesity, but more recent studies have shown greater variability in this association, and it is not supported by studies using the doubly labelled water technique to gauge total energy intake. Experimental studies using iso-energetic diets have shown that eating frequency does not affect energy expenditure. The effect on body weight probably depends on the nature of the food consumed rather than the number of eating occasions per se.

5.3. Physical activity behaviours

Although it may appear obvious that inactive people will gain more weight over time than active people, the true impact of reduced activity on weight gain is not easy to clarify for a number of reasons (WHO Euro 2007, Wareham 2007, Fox and Hilsdon 2007).
Unfortunately, we do not have high quality data on the amount of physical activity that Australians have undertaken over time and most of the data we do have is self-reported, with little objective validation. In addition, the relationship between physical inactivity/sedentary behaviour and obesity is complex and is subject to a wide array of confounding factors which are difficult to exclude.

Although data is now available from large prospective studies, it is difficult to exclude the possible influence of reverse causality; that is, a high BMI at the start of the study may be a cause of decreasing physical activity and may also be independently associated with an increased risk of weight gain.

Most studies have examined only leisure time physical activity and have ignored the impact of occupational and incidental activity.

Despite these limitations, most reviews of research consistently show the expected inverse relationship between leisure time physical activity and obesity in all but the youngest children.

There remains some controversy around the state of participation rates in leisure time physical activity among Australians, with some studies suggesting a decline in recent years while others indicate stable levels of activity. There appears to be more consistency in studies of children that have indicated steady or improved rates of participation in organised physical activity programs. However, it is likely that among adults, the shift in occupational structure and workplace functioning have contributed to a significant decline in physical activity and an increase in sedentary behaviours. This is likely to be compounded by a drop in incidental physical activity as a consequence of reduced use of active transport modes and an increase in the mechanisation of usual tasks such as stair walking, although these contribute only a modest amount to total energy expenditure. In children, a reduction in free play and its replacement by sedentary pursuits is likely to be the major contributor to changes in physical activity in recent years (NPHT 2008).
5.4. Sedentary behaviours

Sedentary behaviours are not merely the inverse of being physically active. They are different but interlinked behaviours. For example, television viewing has been shown to have an association with obesity in childhood and this may be a consequence of inactivity, but it may also be a result of a decrease in metabolic rate or increased food intake which has been shown to accompany TV viewing. Other key sedentary behaviours to consider include: schoolwork at home, video and computer games, reading, sedentary hobbies and chores and sitting in powered transport. Unlike physical activity, the direct relationship between sedentary behaviour and the development of obesity is seen consistently only before adolescence (Fox and Hilsdon, 2007).

The most commonly utilised indicator of sedentary behaviour is television viewing time and many (but not all) studies have found a positive association between hours of TV watched and increased body weight. Each additional hour of TV watched by an Australian child at age six was associated with a 40 per cent increase in the odds of being overweight at age eight. Associations remained significant after adjusting for birth weight, maternal BMI, maternal smoking, and children’s physical activity level. Another Australian study by Hardy et al. (2009) indicated that more than two hours a day of screen time was associated with reduced fitness, increased weight and a greater risk of ill health in schoolchildren. Similar studies are now being undertaken in adults involved in sedentary occupations. Australian studies have identified the high level of sitting time among Australian workers and have found that sitting for more than six hours a day doubles a person’s risk of being overweight compared to those who spend less than one hour a day sitting (Brown et al. 2003, Mummery et al. 2005).
6. **Environmental and structural factors**

The external social, political and economic environment in which people live has a profound effect on the way people live and behave. Each day, people interact with a wide range of services, systems and pressures in settings such as schools, the workplace, home, restaurants and takeaway food outlets. In addition, these settings are influenced by laws, policies, economic imperatives and attitudes of governments, industry and society as a whole. Each of the features of this complex system, which shapes the environment we live in, has the capacity to inhibit or encourage appropriate dietary and physical activity patterns. This has led some researchers to term the environment in Australia today as ‘obesogenic’ or ‘toxic’ because it inhibits appropriate dietary and physical activity patterns and encourages energy imbalance (Egger and Swinburn 1997).

6.1. **Changing physical activity environment**

The availability of open space and access to public transport, the design of suburbs, access to buildings, perceived levels of safety, provision of lighting and many other factors influence our capacity and desire to be more physically active in our daily lives. In recent years, changes in the occupational structure, urban and building design and technology have contributed to a situation where opportunities to be more physically active are now more limited.

The built environment encompasses a range of physical and social elements that make up the structure of a community and may influence obesity-related behaviours. Several characteristics of the urban form (natural and built environment) tend to be associated with physical activity, and possibly with nutrition behaviours. These include:

- mixed land use and density
- footpaths, cycleways and other facilities for physical activity
- street connectivity and design
- transport infrastructure and systems linking residential, commercial and business areas.
A number of Australian and international studies have found that access to proximate and large public open space with attractive attributes such as trees, water features and bird life is associated with higher levels of walking (Gebel et al. 2005). The characteristics of the neighbourhood also have an impact on physical activity. For instance, street layout and the level of connectivity of streets (suburban or more traditional layout) determine route choices and distances, thereby making walking or cycling more or less practicable. Urban design features such as the location of residential, business and commercial areas, in combination with transport services, influence people’s access to food.

‘Active travel or transport’ refers to physical activity undertaken as a means of transport. This can include walking and cycling. It also refers to the use of public transport, as most public transport trips require a walk or cycle trip at either end. Recent research in New South Wales found that people who drove to work were 13% more likely to be overweight or obese than those who walked, cycled or used public transport, regardless of their income level. Additionally, the further people had to drive each day, the greater their weight increase, as shown in Figure 6.1 (Wen et al. 2006). International comparisons of active transport and obesity rates yield similar findings (Basset et al. 2008).
6.2. Changing food environment

Access to appropriate food outlets, advertising pressures, school food policies, nutrition information and labelling all potentially influence food selection. In past years, the usual environment throughout much of Australia allowed only a limited food choice, while today, we have access to a wide variety of cheap, high fat/energy-dense foods that are aggressively marketed.

There is widespread acceptance that the current food supply is not conducive to the maintenance of energy balance. The widely available processed foods have levels of trans fats, saturated fats, salt and sugar well above those recommended for good health and weight control, and provide excess kilojoules. Short-term experimental studies have shown
that larger portions of energy-dense food are associated with increased energy intake (Rolls et al. 2007). Specifically, they have shown that both children and adults consume more at a single eating episode when offered larger portions of energy-dense food. Studies from Denmark (Matthiessen 2003) and the USA (Neilsen and Popkin 2003) indicate that:

- the portion sizes of many (but not all) commercial energy-dense foods and beverages and fast food meals seem to have increased over time, particularly in the last 10 years
- the number of super-sized food items available in grocery stores and supermarkets seems to have increased substantially
- conventional and fast food restaurants serve larger so-called value meals and offer all-you-can-eat buffets in the competition for customers.

Australians now spend around one third of their household food expenditure on food consumed away from the home and, in 2009, 3.7 billion meals were served by commercial food service outlets (BIS Shrapnel 2009). Many people, even nutrition professionals, underestimate the number of kilojoules in fast food meals and when people eat out, they tend to consume more kilojoules and fat, and fewer vegetables, fruits and fibre (Rudd Center 2008). Economic theory suggests that food prices affect food intake. Therefore, manipulating the price of food is likely to have both short- and long-term consequences for body weight (Goldman et al. 2009).

Some researchers suggest that the relative cheapness of high energy-dense foods is a major driver of their consumption by less affluent consumers (Drewnowski 2004). Conversely, the high relative cost of low energy-dense foods, such as fruit and vegetables, is seen as a barrier to their increased consumption.

The majority of studies that have examined the relationship between store access and dietary intake find that better access to a supermarket or large grocery store is associated with healthier food intakes (Larson et al. 2009). Aboriginal people and Torres Strait Islanders face difficulties in accessing an adequate and healthy diet, particularly in remote areas of Australia. Only a very limited variety of food is offered in remote community
stores, relative to what’s available in larger rural towns and urban centres. Perishable items such as dairy foods, fruit and vegetables are frequently in short supply.

Numerous systematic reviews have concluded that the marketing of unhealthy (or energy-dense, nutrient-poor) foods and beverages to children negatively influences children’s eating behaviour, dietary intake and beliefs, and purchase requests to their parents (Cairns et al. 2009, IOM 2006). Much of this literature has focused on the effects of marketing energy-dense, nutrient-poor (EDNP) foods on television, given that this is the main media outlet to which children are exposed. However, marketing has now spread to multiple media platforms including print, the Internet, sports (via sponsorship) and outdoors. (IOM 2006).

Australian studies consistently show that most food advertising is for EDNP foods. The advertising of EDNP foods typically includes persuasive and appealing features, including cartoon and celebrity characters, jingles and themes of fun and family, thus ‘normalising’ the consumption of these foods.

Furthermore, the EDNP foods and beverages advertised to children are not consistent with national dietary guidelines and food selection guides for children. This contributes to a consumer information environment that has conflicting and confusing nutrition information, and compromises the integrity and potential efficacy of any healthy eating social marketing campaigns and consumer education messages.

6.3. Changed living and working environment

Some of the most profound changes to our way of life in recent decades have occurred around the living and working environment. Not only have occupational structures changed but so have working hours and the gender balance within the workforce. Longer working hours, living further from work and the involvement of both partners or parents in the workforce have led to a situation where individuals and families have less time to devote to planned nutrition and physical activity behaviours. As a consequence, certain changes have occurred, which research has identified as having had a negative impact on weight control, and which may have contributed to the rise in obesity (Vandenbroeck 2007, NPHT 2008, Keith et al. 2006). These changes include:
• reduced time for personal physical activity and unavailability to supervise the play and active pursuits of children

• a greater reliance on sedentary leisure time pursuits

• the loss of regular meal patterns and family meals, which has been shown to result in greater snacking and a higher kilojoule intake

• disrupted sleep patterns (short sleep duration has been shown to create metabolic disturbance that interferes with the body’s systems for appetite control and has been associated with weight gain, especially in children).
7. Biological factors

Most of the existing scientific information suggests that the increasing rate of obesity within the Australian population is a result of changes in our food intake and physical activity behaviours driven by social, political and economic environments which promote overconsumption of food and limit opportunities for physical activity. However, it is also clear that heritable biological factors explain why some individuals or groups are more at risk of developing obesity compared to others within the community. In particular, our genes and the nutrition and physical activity environment we are exposed to in utero and early in life have profound effects on our regulation of energy balance and how and where we store fat generated by excess kilojoules.

7.1 Genetic influences

One of the strongest predictors of a child’s weight status is the weight status of their parents. Overweight parents are more likely to have overweight children. This could be a result of the shared family environment, but the fact that adopted children have a weight status closer to their biological rather than adoptive parents suggests a strong role of genetics in weight status. This relationship has been confirmed by examining the closeness in body weight of monozygotic (identical) twins, who have exactly the same genes and very similar weight status, versus dizygotic (non-identical) twins, who share only 50% of their genes on average and have much greater variability in weight status (Bouchard 2007). This does not imply that a certain genetic makeup inevitably leads to obesity but rather, that the propensity to gain weight and become obese is increased. Estimates of how much of the difference in weight status between individuals can be explained by their genes vary depending on what group is being studied and what aspect of weight is being measured – it can be as low as 5% and as high as 70%.

Unlike other inherited conditions, it is extremely rare for a single gene difference to result in obesity. Rather, the inheritance of obesity is thought to be the result of a wide number of genetic variations which result in a series of small but important disruptions to our energy balance regulatory systems (Farooqi and O’Rahilly 2007). So far, around 30,000 genes have been identified in the human genome, and a large number of these have the
potential to influence factors associated with energy balance regulation. However, research does not support the commonly held view that genetics endows some people with slow metabolisms, meaning that they can eat hardly anything without gaining weight, and delivers others with a super metabolism, allowing them to expend significantly more energy than others just by existing. Most of the common gene defects that have thus far been associated with obesity influence appetite and satiety regulation or the way the body handles food and stores fat. Some of these genes may have been associated with positive outcomes in past times when food was scarce and work was hard but now, with our changed environment, they contribute to a negative outcome of weight gain.

7.2. Epigenetic influences on weight gain

It is now understood that inheriting a particular gene does not mean that it will always result in the same outcome in different individuals. Part of the reason for this is that it is possible to alter the way the gene is expressed, or translated into action within the body, by subtle changes to that gene. This process is called epigenetics and is a result of small chemical modifications to the DNA material which can result from a range of factors, such as poor nutrition, a lack of physical activity, smoking, exposure to certain toxins, ageing etcetera. It is thought that these epigenetic changes can predispose individuals to obesity by influencing the way food is handled or energy balance is regulated (Campion et al. 2009). It is unclear whether these epigenetic modifications can be reversed but they are clearly heritable and are passed on to the next generation, leading to poor health outcomes. Epigenetic changes in response to poor nutrition and physical activity environments may explain why rates of obesity have increased so rapidly in recent generations, who have inherited physiological traits that exacerbate weight gain as a result of their own exposure to the obesogenic environment.

7.3. In utero and early life influences

It has long been known that good nutrition during pregnancy is important for the growth and development of the foetus, and the health and wellbeing of the mother as well as the ease of delivery. However, recent evidence has revealed that in utero exposures, together with early life experiences, have profound effects on the weight status and health of the
infant that last well into adulthood. Some of the key perinatal nutrition factors that influence later development of obesity include the following.

**Low birth weight and smoking during pregnancy**

Low birth weight infants experience a range of health problems usually associated with under-nutrition and failure to thrive, but it is also clear now that low birth weight infants are at higher risk of a range of chronic conditions later in life (Barker 2004). Such children are more prone to develop abdominal obesity and early metabolic disease, especially if exposed to over-nutrition in childhood. Low birth weight is common among babies born to women who smoke during pregnancy, but smoking is also associated with a 50% increase in the risk of childhood obesity (Oken et al. 2008).

**Excessive weight gain during pregnancy**

Women who gain more than the recommended levels of weight during pregnancy are at greater risk of gestational diabetes and having a high birth weight child. Such children have a greater rate of obesity later in childhood (Gillman et al. 2005). Women who enter pregnancy with an existing weight problem and have excessive weight gain during pregnancy further increase their risk of gestational diabetes and the obstetric complications associated with high birth weight children (Whittaker 2004).

Accelerated weight gain during the first weeks or months of life is associated with higher BMI or obesity later in life. A systematic review by Baird et al. (2005) found that infants with more rapid early growth had a higher risk of later obesity than infants with normal growth.

**Breastfeeding**

A number of reviews have concluded that exclusive breastfeeding for a period of at least six months is associated with a reduced level of obesity later in childhood. A meta-analysis of 17 studies of breastfeeding duration found that each additional month infants were breastfed was associated with a 4% lower risk of later-life obesity (Harder et al. 2005). However, there remains some controversy around the role of breastfeeding as results of
studies have been inconsistent and it is hard to separate associated factors socio-economic status and cultural factors in any analysis.

**Short sleep duration in infancy**

There is some indication from a small number of studies of an association between short sleep duration in infants and higher levels of overweight in later childhood (Taveras et al. 2008). However, separating the effect of sleep from the wide range of potential confounders in this relationship is difficult.

Other research shows that short sleep times appear to have no relationship with obesity in teenagers aged 10–16 years, and it is unclear whether longer sleep times are beneficial for obese teenagers (Sung et al. 2011).
8. Population groups and life stages most susceptible to weight gain

Weight gain generally occurs slowly over a person’s lifetime. However, there is evidence to show that some life stages and some life events are likely to increase the risk for weight gain. In addition, certain groups within the community appear to be at higher risk for weight gain and obesity as a result of strong social or biological forces which tend to interfere with effective energy balance regulation.

8.1. Critical life stages

There are certain times in a person’s life when they are more prone to weight gain and thus require special focus in addressing the problem. The WHO report *Obesity: Preventing and Managing the Global Epidemic* (WHO 2000) identified a range of critical life stages for weight gain (see Table 7.1). These included:

- prenatal
- adiposity rebound (5–7 years)
- adolescence
- early adulthood
- pregnancy
- menopause.
### Table 8.1 Identifying at-risk groups for obesity in terms of life stages

<table>
<thead>
<tr>
<th>Critical ages/life stages</th>
<th>Reason for increased risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prenatal</td>
<td>There is evidence to suggest that in utero development has permanent effects on later growth and energy regulation.</td>
</tr>
<tr>
<td>Adiposity rebound (5-7 years)</td>
<td>Body mass index (BMI) begins to increase rapidly after a period of reduced adiposity during pre-school years. Food and activity patterns change as a result of exposure to other children and school. Early and rapid weight rebound often precedes the development of obesity.</td>
</tr>
<tr>
<td>Adolescence</td>
<td>Period of increased autonomy which is often associated with irregular meals, changed food habits and periods of inactivity during leisure, combined with physiological changes that promote increased fat deposition, particularly in females.</td>
</tr>
<tr>
<td>Early adulthood</td>
<td>Early adulthood usually correlates to a period of marked reduction in physical activity and significant changes in diet and alcohol consumption. In women, this usually occurs between the ages of 15 and 19 years but in men, it may be as late as the early 30s.</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Excessive weight gain during pregnancy often results in retention of weight after delivery, particularly with early cessation of breastfeeding. This pattern is often repeated after each pregnancy.</td>
</tr>
<tr>
<td>Menopause</td>
<td>In Western societies, weight generally increases with age, but it is not certain why menopausal women are particularly prone to rapid weight gain. The loss of the menstrual cycle does affect food intake and reduces the metabolic rate slightly.</td>
</tr>
</tbody>
</table>

Source: adapted from Gill 1997

### 8.2. High risk groups

The risk of weight gain is not equal across all sections of the population, and some specific groups have been identified as being at higher risk of weight gain. These include the following.

**Individuals with a family history of weight problems**

As previous sections have shown, there is clear evidence that some individuals are more prone to depositing fat than others, even when these individuals live in the same
environment. Having one or more parent with a weight problem is a firm indication of possible genetic predisposition to obesity or a family environment conducive to weight gain. The basis of these differences in individual susceptibility to obesity is yet to be fully elucidated but is believed to involve a number of physiological processes associated with fat deposition and oxidation and involuntary energy expenditure.

**Certain ethnic groups**

The National Preventative Health Task Force (2008) identified a range of ethnic groups within Australia with disproportionately high rates of overweight and obesity. These include Aboriginal and Torres Strait Islander people; people of different cultural backgrounds, particularly from Asia (India and China), Pacific Islands and the Middle East; and recent migrants from Africa and Asia, who are at higher risk of rapid weight gain.

**Socially or economically disadvantaged individuals**

In Australia, there is an inverse association between income and education levels and obesity that is most pronounced among women and children. It is argued that cheaper foodstuffs are usually high in fat and energy-dense and that those with more limited financial resources spend more time engaged in sedentary activities such as watching TV.

**Recent successful weight reducers**

Successful weight loss is usually followed by the regain of one third to one half of the weight loss over the following year. It is believed that biological and behavioural processes act to drive body weight back to baseline levels.

**Recent past smokers**

Smokers are usually thinner than non-smokers because smoking tends to depress appetite, increase the basal metabolic rate and, after each cigarette, induce a surge in heart and metabolic rates. The effect on metabolism of smoking 24 cigarettes per day has been estimated at around 200 kcal per day. Therefore the cessation of smoking is often accompanied by a return in appetite and a reduction in energy expenditure leading to increased risk of weight gain.
9. Conclusion

Although substantial effort has been invested in identifying the determinants of obesity, we still have an incomplete understanding of all the drivers of obesity. It is clear that diet and physical activity behaviours are the central elements of the energy balance equation. However, in contrast to popular belief, obesity is not caused solely by a lack of cognitive control over personal dietary and physical activity behaviours. It is now understood that physiology and genetics play critical roles in driving or attenuating these behaviours and that the physical, social and political environment in which we live greatly influences our ability to maintain behaviours appropriate to weight control.

Improving weight control will require attention by individuals to key dietary and physical activity behaviours. However, it is important to understand that some individuals will be physiologically less able to make such changes, and that changes to the prevailing physical, social and economic environment will be necessary to encourage and support such change and the maintenance of new, healthier habits.
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