CHAPTER 1: INTRODUCTION

1.1 Background

According to the Australasian Society for the Study of Obesity (1995), some 40 per cent of the Australian population are either overweight or obese. The medical literature has clearly demonstrated that obesity is an independent risk factor for a number a serious medical conditions including type 2 diabetes mellitus, hypertension, coronary heart disease (CHD), elevated cholesterol levels, depression, and musculoskeletal pain (Bray, 1992; Colditz, 1992; Kopelman, 2000; Pi-Sunyer, 1996; VanItallie, 1985). Since these conditions can be costly to treat, obesity could clearly have substantial economic impacts. Epidemiological estimates of the aggregate economic costs associated with specific obesity-related diseases for Australia have estimated the economic cost to society as $395 million per annum (Segal et al. 1994).

There are no published Australian studies that have used a microeconomic approach to estimating the overall economic impact of obesity on the cost of medical care utilisation (MCU). This study is based upon the method developed in an earlier study of the costs of obesity in the United States (Kortt, 1997). The techniques developed in that study are refined, expanded, adapted, and applied to an Australian setting and, in particular, to a different dataset. This project attempts to contribute to the literature by addressing several questions related to the overall impact of obesity on health care costs through an analysis of individual-level data on MCU and risk factors, rather than summing up population or aggregate estimates for specific (obesity-related) diseases. These questions explore the complex set of relationships between obesity, obesity-related risk

factors, different types of medical care utilisation (MCU), and the costs associated with MCU. An important feature of this project is to identify preventable diseases associated with obesity and estimate the cost of related MCU to assist in health policy decisions. This is the first time such a study has been undertaken to explore these relationships in Australia.

1.2 Statement of Problem

Previous economic studies of obesity have generally focused on estimating the economic burden of this disease, usually within a prevalence-based cost-of-illness framework (Colditz, 1992; Lévy et al. 1993; Seidell & Deerenberg, 1994; Wolf & Colditz, 1994, 1996). These cost-of-illness studies are based on the underlying assumption that obesity is a causative factor in a number of serious medical conditions including type 2 diabetes mellitus, coronary heart disease (CHD), elevated cholesterol levels, hypertension, depression, and musculoskeletal pain (Bray, 1992; Colditz, 1992; Kopelman, 2000; Pi-Sunyer, 1996; VanItallie, 1985). The objective of these cost studies was to estimate the cost attributable to obesity for these specific diseases. In essence, these studies estimate the costs attributable to obesity by using the population attributable risk (PAR). As the PAR is used in epidemiological studies it would be useful to derive and provide a numerical example of the population attributable risk.

According to Hennekens and Buring (1987), the population attributable risk is of interest because it estimates the "excess rate of disease in the total study population of exposed and non-exposed individuals that is attributable to the exposure" (Hennekens & Buring, 1987, p. 90). The population attributable risk (PAR) is calculated as the rate of disease in the population (I_T) minus the rate in the unexposed group (I_0):

$$PAR = I_T - I_0$$

The concept of the population attributable risk is illustrated in Table 1.1. Data are from a cohort study of oral contraceptive use (OC) in relation to the development of bacteriuria among women aged 16 to 49 years (Hennekens & Buring, 1987).

Table 1.1: Oral contraceptive (OC) use and bacterium among women

	Bacteriuria		
	Yes	No	Total
OC use			
Yes	27	455	482
No	77	1831	1908
Total	104	2286	2390

Source: Hennekens and Buring (1987).

The results from this study showed that of the 2390 women aged between 16 to 49 years who were free of bacteriuria, 482 were OC users in the original survey, while 1908 were not. A second survey indicated that 27 of the OC users have developed bacteriuria, as had 77 non-OC users (Hennekens & Buring, 1987). The number of women in this study who did not develop bacteriuria among OC users and non-OC users can be obtained by subtraction. The data in Table 1.1 allow, the population attributable risk of bacteriuria associated with OC use to be calculated as follows:

$$\begin{aligned} PAR &= I_T - I_0 \\ &= 104/2390 - 77/1908 \\ &= 316/10^5/\text{year} \end{aligned}$$

Thus, if oral contraceptive use were stopped, the excess annual incidence of bacteriuria that could be removed among women aged 16 to 49 is 316 per 100,000 (Hennekens & Buring, 1987). Mathematically, the PAR can also be expressed as the product of the relative risk (RR) and the prevalence of the risk factor (P):

$$PAR = P(RR - 1) / [P(RR - 1) + 1]$$

The relative risk (RR) estimates the strength of an association between exposure and disease and indicates the likelihood of developing the disease in the exposed group compared to those who are not exposed. The prevalence (P), which is a measure of disease frequency, is used to estimate the proportion of individuals in a population who have a disease at given point in time (Hennekens & Buring, 1987). Thus, the prevalence of a disease is calculated by dividing the number of existing cases of a disease by the population (at a given point in time).

Using obesity as a risk factor, PARs were first estimated by Segal et al. (1994). Using information on the relative risk of a disease (such as type 2 diabetes mellitus) in obese individuals, the PAR can then be calculated to estimate the direct cost of treating obesity (Hughes & McGuire, 1997). The cost estimate is based on the excess population in the specific disease areas (such as type 2 diabetes mellitus) who "have the condition as a result of obesity and then the proportion of costs of treating these co-morbidities [such as type 2 diabetes mellitus] that ought to be attributed to these conditions" (Huges & McGuire, 1997, p. 259).

This approach has generally been used to estimate the aggregate cost attributable to obesity. However, there have been no economic studies using Australian survey (or individual-level) data that have attempted to examine and quantify the association between medical care utilisation and the level of obesity (as measured by the body mass index). Although obesity cost-of-illness evaluations indicate that the costs attributable to obesity are substantial, there has been little applied economic research into the relationships between the cost of resource utilisation, the level of obesity, and the diseases associated with being obese. Given the dearth of studies in this area, it is desirable to examine these relationships and to estimate the potential cost savings associated with weight reduction.

A useful starting point will be to examine the association between medical care utilisation (MCU) and the level of obesity using individual-level data. As indicated above, obesity is associated with a number of serious medical conditions. Furthermore, within the medical literature, it has been noted that these risk factors are at least partly caused by obesity (Sjöström, 1993). Given this association, it is important to take these risk factors into consideration when modelling the association between MCU and the level of obesity. Furthermore, there is evidence to suggest that: (i) obesity leads to risk factors such as type 2 diabetes mellitus, hypertension, and high cholesterol levels; (ii) these risk factors affect MCU (and, hence costs); and (iii) obesity has little direct (or independent) effect on MCU. The primary benefit in examining and quantifying these relationships will be in estimating the potential savings that could be realised from weight loss and maintenance strategies.

1.3 Significance of the Problem

Data from the Australasian Society for the Study of Obesity (1995) has revealed that a significant proportion (40 per cent) of the Australian population is either overweight or obese. Estimates from the Australian Bureau of Statistics (ABS) indicate that among adults 18 years and over the rate of overweight and obesity has increased from 38 per cent in 1989-1990 to 42 per cent in 1994-1995 (Australian Institute of Health and Welfare, 1995).

These statistics clearly indicate that a substantial proportion of the Australian population is overweight and is therefore at an increased risk of developing a number of serious medical conditions including type 2 diabetes mellitus, coronary heart disease (CHD), and hypertension. In turn, these diseases will lead to an increase in medical care utilisation (MCU) and hence costs. If over 40 per cent of the Australian population are at an increased risk of developing any one of these medical conditions, then the costs associated with increased MCU could be substantial.

Although obesity can lead to costly treatment for disease, there is also a social stigma associated with being obese, especially for women (Enzi, 1994; Friedman, 2000). Enzi (1994) indicates that obese individuals are adversely affected in their marital, educational, and economic endeavours and may be perceived as being "dirty, stupid, lazy, ugly, cheats, and liars" (Enzi, 1994, p. 54).

With increasing pressures on the national health care budget, it can be argued that there is a need to examine the economic (and health-related) benefits associated with a reduction in obesity among the Australian population. The evidence indicates that a

reduction in excess body fat will assist in the prevention of obesity-related risk factors (such as type 2 diabetes mellitus and hypertension) and hence reduce health care costs.

1.4 Purpose of Study

The purpose of this study is to examine and quantify the association between MCU and the level of obesity (as measured by the body mass index) for a sample of the Australian population. In the analysis, the main components measured as MCU are: (i) the number of doctor visits in 1995, (ii) the number of other health care professional visits in 1995 and (iii) the number of hospital visits in 1995. To evaluate these relationships, a recursive statistical model was specified and logistic and Poisson regression analysis was used to examine the relationships between MCU and the level of obesity. Following this analysis, the Poisson regression coefficients were combined with unit cost data to assess the association between *the cost* of MCU and the level of obesity. This approach was undertaken to provide estimates associated with the potential annual cost savings related to a reduction in body size (i.e., the level of obesity). Data drawn from the 1995 National Health Survey from the Australian Bureau of Statistics (ABS) were used to model these associations.

1.5 Research Questions

Within the context of this research project the following research questions were addressed:

(1) Is there evidence of a causal relationship between obesity (as defined by the body mass index) and the following risk factors: type 2 diabetes mellitus, hypertension,

coronary heart disease (CHD), elevated cholesterol levels, depression, and musculoskeletal pain?

(2) Do these obesity-related risk factors lead to increased medical care utilisation (MCU)?

(3) Does obesity have an independent effect on medical care utilisation? and

(4) To what degree might weight loss reduce the cost of medical care utilisation?

1.6 The Scope of this Study within the Health Economics Literature

This research project can be broadly classified as an exercise in both applied economics and econometrics. Ultimately, the objective is to examine and quantify the relationship between medical care utilisation and the level of obesity (BMI) for a sample of the Australian population. Policy analysis was conducted to assess the potential cost saving associated with a reduction in body size. In terms of the health economics literature, this study fits into the *cost of disease* genre. The cost of disease literature is primarily concerned with the following question: what is the aggregate economic cost of a particular disease or medical condition? (Bulter & Doessel, 1981).

In an early study, Fein (1958) examined the aggregate economic costs associated with mental disorders. Fein (1958) distinguished between direct costs (such as government expenditure on mental illness) and indirect costs (measured as foregone earnings). Weisbrod (1961), undertook a similar approach to evaluate the economic costs associated with cancer, tuberculosis, and poliomyelitis. In a follow-up study, Weisbrod (1971) used this earlier data (1961) to assess the costs and benefits associated with research and treatment expenditure " . . . which led to the development of vaccines (Salk and Sabin) against poliomyelitis" (Weisbrod, 1971, p. 527). Reynolds (1958) used a

similar approach to that of Fein (1958) to examine the economic burden associated with road accidents. Around this time, Muskin and Collings (1959) also outlined the necessary steps involved in undertaking this type of economic evaluation. In each of the above studies, the estimation of total costs did not include intangible costs (most notably the costs associated with pain and suffering). Reynolds (1958) states that ". . . it is beyond the competence of the economist to assign objective values to the losses suffered [due to pain from road accidents]" (Reynolds, 1958, p. 393).[1] Interestingly, this literature also formed part of the background to the argument that *health* should be viewed and treated as an investment (Mushkin, 1962).

It is also worth noting that, at the time, it seemed clear that the economic *benefits* could be measured by the *averted costs* associated with the reduction or elimination of a particular disease or medical condition. This proposition, however, was not without its critics, especially in relation to the *valuation of life* by using *averted* lost earnings (e.g., see Linnerooth, 1979; Mishan, 1971). In an interesting paper, Bulter and Doessel (1981) attempted to sort out, theoretically, the necessary conditions for this proposition to hold.

This project fits into the cost of disease *genre* though it only focuses on one dimension of cost. Its purpose is to examine the potential direct cost savings (or benefit) associated with a reduction in body size. In other words, if obesity could be reduced, what is the magnitude of the potential cost savings?

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[1] Today, standard textbooks on health economic evaluations (e.g., see Drummond et al. 1987) classify costs into three components: direct, indirect, and intangible.

1.7 Thesis Outline

This chapter has presented an overview of the problem and statement of the research questions. Chapter 2 provides an overview of the obesity literature covering issues ranging from the definition of obesity to a review of obesity cost-of-illness studies. Chapter 3 examines obesity issues in Australia and provides an overview of the 1995 National Health Survey, which is the preferred dataset for this project. This is followed by chapter 4, which presents a discussion of the methods used in this research, including the use of individual-level (or micro) data, the specification of the econometrics models, the selection of the variables, and a discussion of the actual data used. Chapter 5 presents the results from the econometric models. Chapter 6 presents the results from the policy scenarios to provide an indication of the magnitude of the potential cost savings associated with a reduction in body size. Finally, chapter 7 provides an overview of the main findings, addressed study limitations, and identifies future economic research areas.

CHAPTER 2: THE OBESITY LITERATURE

2.1 Introduction

This chapter the obesity literature is reviewed and the following areas are discussed: (i) the definitions of overweight and obesity, (ii) worldwide epidemiology, (iii) aetiology, (iv) the relationship between obesity and both mortality and morbidity, and (v) the economic costs associated with obesity.

2.2 Definition of Obesity

The word 'obesity' is derived from the Latin 'ob', meaning over, and 'edere', meaning to eat (Pemberton, 1984, p. 71). However, in a medical context, obesity is defined as an excess amount of body fat or adipose tissue, while overweight is defined as an increased body weight in relation to height, often expressed as the body mass index (Lissner, 1994; Sjöström, 1993). Accurate estimates of adiposity usually require sophisticated techniques that are often expensive and need specialised equipment (Bray, 1992; Lissner, 1994). Consequently, weight-for-height indices are the most commonly used indicators of overweight and obesity in clinically based research (Lissner, 1994).

The body mass index (BMI) is defined as the weight in kilograms divided by height in meters squared:

$$\text{BMI} = \text{weight (kg)} / \text{height (m}^2\text{)}$$

This relationship was originally defined by the Belgium astronomer Quetelet over 100 years ago (Garrow, 1988). According to Quetelet, in 'normal' adults the ratio of weight (W) to height squared (H^2) is roughly constant. Therefore, a person with a relatively high W/H^2 ratio is "over-weight-for-height" (Garrow, 1988, p. 2). The index was named the body mass index in a paper by Keys et al. (1972).

A variety of BMI categories have been selected as operational definitions of overweight and obesity. BMI categories for overweight and obesity have been proposed by the World Health Organisation (WHO) Expert Committee for the classification of overweight (Seidell & Flegal, 1997), the National Center for Health Statistics in the United States (VanItallie, 1994), and Stunkard (1992). Of these three, both the Australian Bureau of Statistics (ABS) and the Australian Institute of Health and Welfare (AIHW) use the WHO-endorsed international classification for obesity (and overweight). This classification system is presented in Table 2.1.

Table 2.1: WHO classification of obesity/overweight

Body mass index	*WHO classification*	*Popular description*
< 18.5	Underweight	Thin
18.5 to 24.9	—	'Healthy', 'normal', 'acceptable', or 'desirable' weight
25 to 29.9	Grade 1 obesity	Overweight
30 to 39.9	Grade 2 obesity	Obese
≥ 40	Grade 3 obesity	Morbidly obese

Source: Adapted from Seidell & Flegal (1997).

The BMI of an individual is used to classify whether or not they are overweight or obese. This classification system covers four different categories ranging from underweight to grade 3 (or morbid) obesity. In this classification system being

overweight is defined as a BMI between 25 and 30, and obesity is defined as BMI greater than 30 (Table 2.1).

Although the WHO classification system is widely used, it should also be noted that the National Center for Health Statistics in the United States defines overweight as a BMI greater than or equal to the 85th percentile of men and non-pregnant women between the ages of 20 to 29 years. Severe overweight is defined as a BMI greater than or equal to the 95th percentile (Kuczmarski, 1992; Lissner, 1994; Wolf & Colditz, 1994). As shown in Table 2.2, men are classified as overweight when their BMI is greater than or equal to 27.8, and they are judged to be severely overweight (obese) when their BMI is greater than or equal to 31.1. For women, the corresponding cut-off points for overweight and severely overweight (obese) are BMI scores of 27.3 and 32.3, respectively. This classification system is similar to the WHO-endorsed classification system of obesity is presented in Table 2.2.

Table 2.2: National Center for Health Statistics definition of overweight

	Overweight (85th percentile)	*Severe overweight (95th percentile)*
BMI for men	27.8	31.1
BMI for females	27.3	32.3

Source: Kuczmarski (1992).

Stunkard (1992) has developed another obesity classification system defined by the degree of severity. This classification system is presented in Table 2.3. Stunkard (1992) proposes that obesity (as measured by BMI) should be classified into three distinct categories ranging from 'mild' to 'severe' obesity.

Table 2.3: Classification of obesity by degree of severity

Body Mass Index	*Class of Obesity*	*Per cent Overweight*
27 to 30	Mild	20-40%
31 to 35	Moderate	41-100%
> 35	Severe	>100%

Source: Martin et al. (1995) adapted from Stunkard (1992).

Although BMI is generally used as a proxy for the degree of obesity in epidemiological studies, "it has been proposed that the weight-for-height function best corresponding to adiposity may not in fact be BMI, but weight to some other power of height" (Lissner, 1994, p. 9). Regardless of the power function used however, any BMI criterion selected for an operational definition of obesity is to a large extent arbitrary, "because body fatness is naturally distributed in a continuum" (Lissner, 1994, p. 9).

In defining overweight and obesity it is also important to consider the concept of regional adiposity. There is evidence to suggest that an individual with a relatively high BMI may not be at excess health risk if the distribution of body fat exhibits a gynoid (or pear-shaped) pattern (Lissner, 1994). A gynoid pattern is characterised by body fat distribution in the hip and femoral regions. Women tend to have a greater amount of gluteal fat and consequently have relatively larger hip circumferences, giving them the gynoid fat distribution pattern. On the other hand, an android (or apple-shaped) pattern may be associated with an increased health risk. An android pattern is characterised by body fat distribution in the torso and abdomen. Men generally tend to have a greater amount of abdominal fat, giving them the android fat distribution pattern (Bray, 1992). In other words, it is thought that people's shape (i.e., their body fat distribution) as well as their weight is an important factor with respect to possible health risks. On the basis

of these different distributions, a measure of central body fat distribution can be obtained by calculating the waist-to-hip ratio (WHR) by dividing the waist measurement by the hip measurement (Bray, 1992; Lissner, 1994). It has also been proposed that a waist-to-hip ratio of above 1 in men and 0.85 in women is a meaningful cut-off point associated with increased health risks (Lissner, 1994). West (1994) indicates that it is relatively easier for android shaped people to alter their WHRs because abdominal body fat is more easily broken down than subcutaneous fat. In addition, gynoid shaped people may encounter other health problems such as varicose veins.

In summary, there are a number of criteria for defining overweight and obesity. The most commonly used definition of obesity in epidemiological and research papers is the WHO classification based on the body mass index. Nevertheless some studies use different BMI values to define whether individuals are overweight or obese, and this complicates the comparisons of data and results.

2.3 Worldwide Epidemiology

According to VanItallie (1994), average body mass index (BMI) values and prevalence data for obese populations display complex patterns that vary with age, sex, race, geography, and socioeconomic status. International comparisons of the prevalence of obesity are difficult to make because obesity has been defined differently across different studies (VanItallie, 1994). Nonetheless, "a number of global trends are evident when the available data, however imperfect, are compared" (VanItallie, 1994, p. 1).

Based on the WHO classification system for obesity, the Australasian Society for the Study of Obesity (1995) has reported that some 40 per cent of the Australian population is either overweight or obese. Estimates from the Australian Bureau of Statistics (ABS) indicate that among Australian adults 18 years and over the rate of overweight and obesity has increased from 38 per cent in 1989-1990 to 42 per cent in 1994-1995 (Australian Institute of Health & Welfare, 1995).

International comparisons of the prevalence of obesity have been compared for eight developed countries and six developing Latin American countries (Epstein & Higgins, 1992; VanItallie, 1994). The prevalence of obesity, defined as a BMI value greater than or equal to 30, for selected countries is presented in Table 2.4.

Table 2.4: Prevalence of obesity (BMI ≥ 30): A selective international comparison

Country	*Age in Years*	*Prevalence (%)*	
		Male	*Female*
Developing Countries of Latin America			
Costa Rica	40-45	5.7	14.4
El Salvador	40-45	0	1.5
Guatemala	40-45	0	5.6
Honduras	40-45	2.8	6.0
Nicaragua	40-45	3.1	16.4
Panama	40-45	2.3	1.7
Developed/Industrialised Countries			
Australia	25-64	7	7
Canada	25-64	9	8
Finland	20+	10	18
Italy	15-44	4.8	3.9
	45-64	9.9	11.1
The Netherlands	20-34	1.9	2.0
	35-49	4.2	5.0
	50-64	5.4	10.3
South Africa	15-64	14.7	18.0
United Kingdom	20-64	8	9
United States	20-64	12	15

Source: Adapted from VanItallie (1994) after Epstein & Higgins (1992)

Although only broad international comparisons can be made, it appears that obesity is most prevalent in North Americans and white South Africans (VanItallie, 1994). For other industrialised countries such as Australia, the United Kingdom (UK), and Canada, the prevalence of obesity for both men and women are comparable. On the other hand, the prevalence of obesity in Latin American countries varies considerably.

It also should be noted that the prevalence of obesity in Asian countries is relatively lower than in industrialised Western countries. For example, the prevalence of obesity in Japan is among the lowest in the world, with an average BMI for both men and women less than 25 (Stamler, 1993).

Data have been collected by Epstein and Higgins (1992) from the Intersalt and WHO MONICA Project (*Moni*toring trends and determinants in *car*diovascular disease) to provide a guide to the relationship between average BMI and obesity (defined as a BMI value $\geq$ 90th percentile) throughout the world (VanItallie, 1994). These data are presented in Table 2.5.

These data indicate that there is limited variation between average BMI and obesity throughout Europe, while both North American and European values tend to be relatively high. Conversely, the mean BMI and obesity values for Asia are relatively low. Furthermore, females throughout Europe, North America, and Asia have relatively higher BMI values – at the 90th percentile point – than their male counterparts.

According to VanItallie (1994) race also affects the prevalence of overweight and obesity. For instance, the percentages of white, black, and Mexican-American US

adults aged between 20 and 74 years who were classified as being overweight were 24.4 per cent, 26.3 per cent, and 31.2 per cent, respectively (VanItallie, 1994). In addition, Martin et al. (1995) has reported that nearly 50 per cent of black women in the United States were overweight. The 1994 Aboriginal and Torres Strait Islander survey indicated that 60 per cent of men and 58 per cent women were either overweight or obese (National Health and Medical Research Council, 1997).

Table 2.5: Mean BMI and obesity (BMI ≥ 90th percentile), based on the Intersalt and WHO MONICA studies

Geographic Area	*Mean BMI*[a]	*BMI at 90th percentile point*[b]	
		Males	*Females*
Northern Europe	24.5-25.3	29.1-31.7	29.6-33.0
Western and Central Europe	24.4-25.9	29.8-32.5	30.8-33.6
Southern Europe	25.4-28.0	30.3-32.7	30.8-33.2
Eastern Europe	24.9-26.2	30.4-32.4	32.0-36.2
North America	25.1-30.1	29.9	31.8
South and Central America	21.2-28.2		
Asia	20.1-23.8	27.6	29.5
Africa[c]	20.8-26.1		

a Age-adjusted value of men and women aged between 30 and 59 years.
b Men and women aged 35 and 64 years.
c Kenya and Zimbabwe only.

Source: VanItallie (1994) after Epstein & Higgins (1992)

It is postulated that modernisation is a contributing factor to the increased prevalence of obesity in population groups such as female Samoans, young Saudi Arabian women, and Australian Aborigines (VanItallie, 1994).

Based on the available data, it appears that obesity is becoming more prevalent worldwide, not only in industrialised countries but also in the populations of developing

countries (VanItallie, 1994). These data also indicate that the prevalence of obesity is more pronounced in female than male populations.

Causes of Trends in Obesity

A number of reasons have been put forward to explain the worldwide increase in the prevalence of obesity. Reduced physical activity, diets high in fat and "inadequate adjustments of energy intakes to diminished energy requirements are likely to be major determinants of the observed changes" (Seidell and Flegal, 1997, p. 249). In a study conducted in the United Kingdom, Prentice and Jebb (1995) suggest that, on a population level, limited physical activity may be a more important factor, than fat consumption, in explaining the increasing trend of obesity.

Changes in smoking behaviour may also contribute to overall changes in body weight on a population level (Seidell and Flegal, 1997). For example, data from the US indicate that, smoking cessation can explain some of the increase in the prevalence of overweight, but this alone cannot explain the major proportion of the increase (Flegal et al. 1995). Other studies have also shown that the increase in the prevalence of obesity may, in fact, be independent of an individual's smoking status (Boyle et al. 1994; Wolk et al. 1995).

Seidell and Flegal (1997) also indicate that dramatic increases in the prevalence of obesity in countries such as The Netherlands could be attributable to relatively minor increases in mean (average) body weight. For example, if height had remained constant, a mean weight increase of "only slightly less than 1 kg over 10 years could account for the increased prevalence of obesity observed" (Seidell and Flegal, 1997, p.

249). This gradual increase in body weight could reflect a relatively small change in energy balance on a daily basis (Seidell and Flegal, 1997).

2.4 Aetiology of Obesity

Obesity can be viewed as a energy imbalance disorder, "which develops as a consequence of excess energy input with respect to output" (Lissner, 1994, p. 9). According to West (1994), in an average sedentary individual, approximately three-quarters of total energy expenditure is accounted for by the body's basic activities which include breathing and the maintenance of body temperature. About one-tenth of energy is expended digesting food, while the remaining energy is used for all other activities such as exercise (West, 1994).

It has been suggested that there are two different forms of obesity (Bierman, 1979). The first form is characterised as a life-long condition commencing during early childhood (with weight gains occurring during puberty), and for women, during pregnancy (with weight gains made over the long-term). The second form of obesity, occurs in middle-age adults who gain weight because of a more sedentary lifestyle. It should be noted, however, that these propositions have not been supported by the scientific literature (West, 1994).

It has been postulated that a variety of factors may be associated with obesity including: (i) genetic predisposition, (ii) low socioeconomic status, and (iii) psychological factors (West, 1994). These factors are discussed below.

2.4.1 Genetic Factors

There is a growing body of evidence indicating that an individual's body weight has a substantial genetic component (Barsh et al. 2000; Brownell & Wadden, 1992; Hawks & Richins, 1994). Floch and McClearn (1980) examined the question of whether genetic factors were a cause of obesity. They reviewed several studies and noted that two out of three obese individuals had at least one parent who was also obese, indicating a genetic predisposition. West (1994) indicates that these findings could be explained by environmental factors such as family upbringing and socioeconomic status. However, other studies support the conclusions of Floch and McClearn (1980). In 1964, Withers identified a correlation between the weights of adopted children and their biological parents rather than their adoptive parents, giving support to the genetic predisposition premise (Withers, 1964). More recent studies involving adoptive children (Stunkard et al. 1986) and twins reared separately (Stunkard et al. 1990) provide further evidence that obesity is related to genetic susceptibility, as opposed to environmental factors such as family upbringing.

The current dogma is that genetic predisposition is insufficient alone to cause obesity (West, 1994), although genetically inclined individuals who are exposed to adverse environmental factors have a greater chance of becoming obese (Lissner, 1994; Meyer & Stunkard, 1993). Although some individuals may be genetically predisposed to obesity, their problems may be overcome (or at least controlled) by a program consisting of dietary modification and regular exercise.

2.4.2 Socioeconomic Factors

Socioeconomic status (SES) is conceptualised as a combination of financial, educational, and occupational influences (Winkleby et al. 1992). Socioeconomic factors such as low education level, high-school alcohol consumption, and loss of employment appear to be correlated with obesity (Garrow, 1992). Sobal and Stunkard (1989) reviewed 144 published studies that examined the relationship between the SES and the level of obesity. They found a strong inverse relationship between obesity and SES among women in industrialised societies such as the United States and Europe. However, among men and children the relationship was less consistent (Sobal & Stunkard, 1989). On the other hand, in developing countries, a strong direct relationship between SES and obesity has been identified (Sobal and Stunkard, 1989; West, 1994), "probably reflecting the availability of food as a major determinant of nutritional status including the degree of obesity" (Sorensen, 1995, p. S6).

Based on these studies, the observed association between relatively low SES and the level of obesity may be explained by three different types of causal relationships. Sorensen (1995) suggests that: (i) low SES may promote or encourage the development of obesity, (ii) SES may be reduced by obesity, or (iii) obesity and low SES "may share some causes that both promote the development of obesity and tend to reduce the SES" (Sorensen, 1995, p. S6). Enzi (1994), who concludes that obese individuals (especially women) are adversely affected in their marital, educational, and economic prospects, also supports the relationship between SES and obesity.

2.4.3 Psychological Factors

Psychological problems probably results from obesity. A number of studies have concluded that obese individuals may experience social discrimination and psychological distress as a result of their weight (Enzi, 1994; Perri et al. 1992; Wadden & Stunkard, 1985). In fact, society's emphasis (or obsession) with being 'thin' and its prejudice towards obese individuals have probably contributed to the "disdain that overweight people have for their own physical appearance" (Perri et al. 1992, p. 13).

It is also argued that psychological factors or emotional disturbances are a causative component in the development of obesity (Jebb 1997; West, 1994). For instance, reactive obesity is "said to exist when over-eating takes place as a reaction to a distressing situation or event" (West, 1994, p. 17). It was also believed that obese individuals may over-eat to compensate for feelings such as inferiority, insecurity, and even sexual inadequacy (Perri et al. 1992). Since the 1980s, however, this view has changed substantially. Clinically-based studies comparing obese patients to nonobese controls have failed to demonstrate a clinically significant difference in general psychological disturbance (Perri et al. 1992; Wadden & Stunkard, 1985).

However, while obese subjects may not differ from non-obese subjects in terms of general psychological disturbances, it has been reported that numerous obese individuals suffer from a range of emotional difficulties (Perri et al. 1992). Obese individuals usually suffer from psychological disturbances that are related to being obese. Negative emotional feelings in obese individuals can range from frustration due to failed attempts at weight loss to "severe body-image disparagement" (Perri et al.

1992, p. 14). So the current wisdom is that psychological factors may play a small role in causing obesity, but obesity certainly causes psychological problems.

2.5 The Relationship between Obesity and Mortality

The role of obesity as a risk factor of premature mortality has been discussed for over 30 years (Sjöström, 1993). The relationship between obesity and decreased life expectancy has been demonstrated in both insurance studies and population-based studies. Increased risk of premature mortality has not only been documented in the severely obese but also for individuals who experience significant weight change over time. Moreover, obesity is also associated with increased prevalence of cardiovascular risk factors such as elevated cholesterol levels, hypertension, and type 2 diabetes mellitus (VanItallie, 1985).

2.5.1 Insurance Studies

United States insurance statistics, over the 20th century have "indicated that obesity was associated with increased mortality" (Sjöström, 1993, p. 14). The first studies to report an association between body weight and mortality were published in the early 1950s (Dublin, 1953; Williamson & Pamuk, 1993). These results were similar to the findings of a prospective study conducted in 1950 by the Metropolitan Life Insurance Company, with the results that the insurance company charged overweight individuals extra life insurance premiums because "they were too fat to be taken at the standard rates" (Dublin, 1953, p. 971). These original findings were also supported by the 1959 Build and Blood Pressure Study and the 1979 Build Study (Sjöström, 1993; Williamson & Pamuk, 1993).

2.5.2 Population-Based Studies

Despite the association indicated by insurance statistics and the subsequent insurance-based studies (Williamson & Pamuk, 1993), approximately 40 per cent of all random population-based studies performed during the 1960s and 1970s, failed to show a relationship between body weight (i.e., obesity) and mortality (Sjöström, 1993). In spite of this, Sjöström (1993) emphasised than many of these papers had a number of limitations including: (i) relatively small sample sizes, (ii) a short-term focus, (iii) failure to control for cigarette smoking, and (iv) inappropriate control of intermediate risk factors (Sjöström, 1993).

Sjöström (1993) points out "that all studies with more than 20,000 participants and 20 of 21 cohorts larger than 7,000 show a positive relationship between overweight and mortality" (Sjöström, 1993, p. 22). Furthermore, in relatively larger size studies (with a follow-up period of about 5 years) the association between body-weight and mortality is more evident (Sjöström, 1993). In other words, smaller sample size studies with a short follow-up period are less likely to demonstrate a positive relationship between body weight and mortality.

Another study limitation is that negative population studies did not control for cigarette smoking (Sjöström, 1993). Epidemiological studies indicate that cigarette smoking not only increases the risk of mortality, but is also more common among non-obese individuals. Therefore, failure to control for cigarette smoking will produce a higher mortality rate in non-obese subjects. Consequently, when non-obese and obese mortality rates are compared "obesity-related mortality will thus be underestimated"

(Sjöström, 1993, p. 24). For example, in a study by Manson et al. (1990) controlling for cigarette smoking led to a more evident association between body weight and mortality.

A further limitation identified is that negative population studies failed to adequately control for intermediate risk factors. Sjöström (1993) cites some early studies (Chapman & Massey, 1964; Rosenman, 1975) that found a crude relationship between body weight and mortality, but concluded that body weight had no impact on mortality rates because the association was insignificant when hyperlipidemia, hypertension, and type 2 diabetes mellitus were controlled. However, the crucial point stressed by Sjöström (1993) is that "these conditions are not confounders, but intermediate risk factors that are – at least partly – caused by obesity and through which obesity exerts its damaging effects" (Sjöström, 1993, p. 24).

Although about 40 per cent of population-based studies indicate a negative association between body-weight and mortality, it is interesting to note that over the past 10 to 15 years, approximately 60 per cent of all "employee, community, and random population studies have found a relationship between overweight and mortality" (Sjöström, 1993, p. 25). According to Sjöström (1993), the most important studies, during this time, were long-term in design and had relatively large sample sizes (Hubert et al. 1983; Lew & Garfinkel, 1979; Waaler, 1984).

Lew and Garfinkel (1979) reported on obesity in the American Cancer Society Study, which followed a total of 750,000 people over the period 1959 to 1972. This total population consisted of 340,000 men and 420,000 women between the ages of 38 and 89. The key finding in the American Cancer Society Study was that the positive

association between body weight and mortality was similar to the relationships observed in 1959 Build and Blood Pressure Study and the 1979 Build Study (Williamson & Pamuk, 1993). Another significant finding observed in this study was the 25-fold increase in the mortality of obese young women with type 2 diabetes mellitus (Sjöström, 1993).

Hubert et al. (1983) analysed the Framingham Heart Study population, and discovered that obesity was related to both mortality and morbidity even after controlling for other medical conditions such as hypertension, elevated cholesterol levels, and type 2 diabetes mellitus. Within this analytical framework, obesity was considered to be an independent risk factor. However, as Sjöström (1993) has argued, these conditions should not be thought of as confounders, but more appropriately as intermediate risk factors.

In another study, Waaler (1984) reported on 'The Norwegian Experience' in which all Norwegian citizens over the age of 15 years were required to have an x-ray examination. These x-ray examinations were performed between 1967 and 1976. Originally, the objective of this study was to screen for tuberculosis but all subjects also had their weight and height recorded. Approximately 816,000 and 902,000 men and women were followed, on average, for 10 years. During this time, approximately 177,000 deaths were recorded. A U-shaped relationship between BMI and mortality was observed for both men and women. For instance, in men aged between 40 and 50 years, the risk of mortality associated with a BMI value of between 23 and 25 was doubled for men with a BMI value of 34 (Waaler, 1984). Furthermore, the mortality

rates for the Norwegian diabetic mellitus population were similar to the findings reported in the American Cancer Society Study (Waaler, 1984).

Although the study by Waaler (1984) indicated a U-shaped relationship between BMI and mortality, other studies report diverse findings (Manson et al. 1995). For example, Manson and colleagues (1995) examined the association between BMI and mortality in a cohort of 115,195 United States women between the ages of 30 and 55 enrolled in the prospective Nurses' Health Study. A BMI value of less than 19 was used as the reference category (i.e., relative risk = 1.0) to examine the association between increased BMI values and mortality. The authors concluded that body weight and mortality were directly related among middle-age women, while lean women (i.e., BMI < 22) did not have excess mortality rates. Similar findings were supported by Willett et al. (1995).

An overview of these studies shows that a variety of relationships between BMI and mortality have been documented including no association (Vandenbroucke et al. 1984), a J-shaped relationship (Folsom et al. 1993), a direct relationship (Lindsted et al. 1991), and even an inverse association (Wilcosky, 1990).

Although there is still debate as to the true nature of the relationship between body weight and mortality, there is strong evidence to suggest that obesity affects the mortality rate of the general population (Sjöström, 1993). The most compelling evidence comes from long-term and large sample size population studies (Hubert et al. 1983; Lew & Garfinkel, 1979; Waaler, 1984).

2.5.3 The Relationship between Severe Obesity and Mortality

As obesity is a general term, it is important to define and distinguish between severe obesity and other forms of milder obesity. Various terms have been used to describe the uppermost level of obesity including very obese, super obese, gross obesity, extreme obesity, and morbid obesity (Kuczmarski, 1992). This degree of obesity may carry its own consequences (Kral, 1985; Bray, 1992). Severe obesity is a term used to "denote an absolute (45.4 kg) or relative (100 per cent) excess weight above a 'desirable' weight that is statistically associated with maximum life expectancy" (Kral, 1985, p. 1043). This degree of obesity is usually associated with a BMI value of between 40 and 50 (Kuczmarski, 1992).

Severe obesity is also associated with an increased risk of sudden death (Bray, 1992), an observation initially made by Hippocrates some 2,000 years ago (Bray, 1990). Subsequently, this association has been supported by findings in epidemiological studies including the Framingham and Wadsworth Veterans Administration Study (Bray, 1992). Kral (1985) reports that sudden death is at least 13 times more prevalent in severely obese women than in normal women. Severe obesity is also associated with a number of medical conditions including cardiomyopathy, Pickwickian/sleep apnea syndrome, pituitary/gonadal dysfunction, acanthosis nigricans, and osteoarthritis (Bray, 1992). If severe obesity remains untreated, the individual may become physically disabled (Martin et al. 1995).

2.5.4 The Relationship between Weight Change and Mortality

According to Sjöström (1993) weight gains should be associated with an increased risk of mortality "since the obese state is necessarily preceded by weight increase"

(Sjöström, 1993, p. 28). For instance, in a prospective study, Mason et al. (1990) examined the relationship between obesity and the risk of coronary heart disease in 115,886 US female nurses. Nurses whose weight had changed less than ± 3 kilograms (kg) since the age of 18 were used as a reference category in the analysis, with a relative risk of 1.0 for myocardial infarction plus fatal coronary heart disease combined. The authors reported that substantial weight gains in the range of 10 to 19.9 kg and 20 to 34.9 kg were associated with relative risks of 1.7 and 2.5, respectively (Mason et al. 1990).

Although the association between weight gain and mortality has been reported, a number of studies have observed a relationship between weight loss or weight variation and increased mortality (Hamm et al. 1989; Lee & Paffenbarger, 1992; Lissner et al. 1991). However, according to Iribarren et al. (1995) "this is not a universal finding" (Iribarren et al. 1995, p. 686). In fact, a review of the literature by Goldstein (1992) indicated that weight loss has been associated with reductions in both mortality and obesity-related medical conditions.

Iribarren et al. (1995) examined the long-term relationship between weight loss, weight variation, and mortality over a 6-year period in 6,537 Japanese American men enrolled in the Honolulu Heart Program. The authors reported that weight loss and weight fluctuations were not significantly related to mortality in healthy men who had never smoked. On the other hand, weight loss and weight variation was associated with mortality in men with pre-existing health problems and current cigarette smokers. Therefore, concerns about weight loss and weight variation may not be applicable for healthy men.

However, the major limitation with studies examining the association between weight loss and mortality has been their failure to distinguish between intentional and unintentional weight loss (Iribarren et al. 1995). This limitation has been cited as the key reason why intentional weight loss has been observed in clinical trials and not in the majority of epidemiological studies (Williamson et al. 1995). Failure to account for intentional and unintentional weight loss "may bias the estimated effect of weight loss toward an adverse association because unintentional weight loss is often caused by illness" (Williamson et al. 1995, p. 1128).

The study by Williamson et al. (1995) was the first study to report on the association between mortality and intentional weight loss. The authors analysed prospective data from 43,457 overweight (never smoking) US white women aged between 40 and 64 years. These women completed questionnaires on weight, weight change, and intention to lose weight between 1959 and 1960. The statistical analysis employed Cox proportional hazards modelling[2] to estimate the mortality ratios for women who intentionally lost weight compared with women who had no weight change. In addition, the analysis was stratified according to pre-existing illness adjusted for age, initial BMI, alcohol consumption, educational level, physical activity, and health conditions (Williamson et al. 1995).

[2] The Cox proportional hazards model (named after D.R. Cox who developed the approach) allows for multivariable comparisons of hazards functions between two groups. The Cox proportional hazards model makes it possible to evaluate the impact of each independent variable on differences between two hazard functions (Selvin, 1995).

Williamson et al. (1995) reported that in women with obesity-related health conditions such as type 2 diabetes mellitus, cancer, and cardiovascular disease who intentionally lost between 0.5 and 9 kg had a 20 per cent decrease in all-cause mortality (as did women who lost in excess of 9 kg). These reductions in overall mortality were due to decreased cancer mortality among women who intentionally lost weight. Women who lost between 0.5 and 9 kg had a 37 per cent reduction in cancer mortality, while women who lost in excess of 9 kg had a 29 per cent reduction in mortality. Although, the mortality rate from cardiovascular disease was reduced by only 9 per cent among women who intentionally lost weight, the diabetes-related mortality rate was reduced by 44 per cent among women who lost between 0.5 and 9 kg and by 31 per cent in women who lost in excess of 9 kg (Williamson et al. 1995). On the other hand, in women with no pre-existing illnesses, the authors report "little difference in the rates for all-cause, cardiovascular, and diabetes associated mortality between those with no weight change and those with intentional weight loss" (Williamson et al. 1995, p. 1128).

The Williamson et al. (1995) study indicates the importance of distinguishing between intentional and unintentional weight loss. Based on the findings in the Williamson et al. (1995) study, intentional weight loss in women with obesity-related conditions is generally associated with a reduction in the mortality rate.

2.5.5 The Relationship between Obesity and Morbidity

The medical literature has clearly demonstrated that obesity is an independent risk factor for a number of serious medical conditions including type 2 diabetes mellitus, hypertension, coronary heart disease, elevated cholesterol levels, depression,

musculoskeletal pain, and several cancers (Bray, 1992; Colditz, 1992; Kopelman, 2000; Pi-Sunyer, 1993, 1996; VanItallie, 1985). The conclusions from a number of different studies indicate that there is an association between weight reduction and decreased mortality, and according to Sjöström (1993) has concluded that "it is well known that obesity-related risk factors are reduced by weight reduction" (Sjöström, 1993, p. 28). Medical conditions associated with obesity are discussed in detail below.

2.5.5.1 Diabetes Mellitus

The causative factor in the association between diabetes mellitus and obesity appears to be obesity "which brings about abnormal glucose disposal and impaired effectiveness of insulin" (Drenick, 1980, p. 391). Therefore, promoting weight loss results in an improvement of glucose tolerance and a reduction of insulin resistance, "suggesting that obesity either causes or enhances obesity-diabetes" (Drenick, 1980, p. 391).

A strong association between type 2 diabetes and obesity has been observed in a number of studies (Colditz et al. 1995; Goldstein, 1992; Lean et al. 1990; Rimm et al. 1995), and it has been postulated that obesity is the strongest modifiable risk factor for individuals with type 2 diabetes mellitus (Rimm et al. 1995). It has also been documented that that diabetic-obese patients who lose about 10 per cent of their body-weight show reductions in hyperinsulinemia "and improve [their] responsiveness to glucose challenge" (Goldstein, 1992, p. 398).

Lean et al. (1990) who examined the association between obesity and weight loss for individuals diagnosed with type 2 diabetes mellitus found similar results. Medical records from the Aberdeen diabetic clinic for 263 subjects with type 2 diabetes mellitus,

who died in 1985 or 1986, were used in the analysis. For the 233 who lived more than one year (189 were classified as being overweight) a survival analysis, using stepwise multiple regression techniques, was performed The analysis showed that for the average patient a 1 kg weight loss was associated with a prolonged survival of between three to four months. A 10 kg weight loss "would predict the restoration of about 35 per cent in life expectancy" (Lean et al. 1990, p. 232).

More recently, Colditz et al. (1995) examined the association between weight change and the risk of type 2 diabetes mellitus among US middle-age women based on a prospective cohort study from 1976 to 1990. Using BMI as an indicator of obesity the results indicate that BMI was the dominant predictor of risk for type 2 diabetes mellitus. The relative risk for type 2 diabetes mellitus for women who gained between 5.0 and 7.0 kg was 1.9. On the other hand, women who lost in excess of 5 kg reduced their risk of type 2 diabetes mellitus by 50 per cent or more (Colditz et al. 1995).

2.5.5.2 Hypertension

Hypertension, or high blood pressure, is a common problem in modern society that may result in a number of medical conditions including blurred vision and dizziness (Wiklund, 1996). High blood pressure has also been established as a strong risk factor for heart disease in both men and women (Wiklund, 1996). A number of studies such as the Swedish Trial in Old Patients with Hypertension (STOP - Hypertension) (Johannesson et al. 1993), the Systolic Hypertension in the Elderly Program (SHEP), and the European Working Party on High Blood Pressure in the Elderly (EWPHE) (Wiklund, 1996) have shown that lowering elevated blood pressure reduces the risk of stroke and myocardial infarction (Wiklund, 1996).

Since excess body weight is associated with increased blood pressure, weight loss can reduce elevated blood pressure in hypertensive individuals who are more than 10 per cent over ideal body-weight (Langford et al. 1991). It has been shown that a reduction in blood pressure can be achieved with a weight loss of about 4.5 kg (Schotte & Stunkard, 1990) and in his review Goldstein (1992) has concluded that "most studies assessing the effect of weight reduction on hypertension have supported the contention that substantial (> 10 per cent) weight reduction lowers blood pressure" (Goldstein, 1992, p. 411).

2.5.5.3 Coronary Heart Disease

There is evidence to suggest that obesity contributes to coronary heart disease (CHD). In the United States, cross-sectional studies such as the second National Health and Nutritional Examination Survey (NHANES-II) has clearly demonstrated an association between the level of obesity and CHD (Perri et al. 1992; Pi-Sunyer, 1993). Several other large-scale prospective studies have also examined the association between the level of obesity and CHD (Perri et al. 1992). For example, in the Nurses' Health Study, Manson et al. (1990) examined the incidence of CHD in a prospective cohort over 8 years. The sample consisted of 115,886 nurses who were 30 to 55 years of age in 1976 and who were free of CHD, cancer, and stroke. Results from this study indicate that women in the heaviest weight category (i.e., BMI > 29), were three times more likely to develop CHD than those in the lightest group (i.e., BMI < 21).

2.5.5.4 Elevated Cholesterol Levels

Cholesterol is a fat-like substance (lipid) that is present in cell membranes (Working Group Report, 1991). Cholesterol travels through the blood, containing both lipids and proteins, commonly referred to as lipoproteins. There are three major forms of lipoproteins in the blood of an individual: (i) low density lipoproteins (LDL), (ii) high-density lipoproteins (HDL), and (iii) very low-density lipoproteins (VLDL). The LDL consists of between 60 to 70 per cent of the total serum cholesterol, the HDL consists of between 20 to 30 per cent of total cholesterol while the VLDL consists of between 10 to 15 per cent of the total serum cholesterol (Working Group Report, 1991). Since LDL cholesterol is the major atherogenic lipoprotein, it is usually the primary target of many cholesterol-lowering strategies. An individual's cholesterol level is determined partly by genetic history and their health status and social factors such as diet, calorie intake, and physical activity.

Obese individuals tend to have higher concentrations of total cholesterol in the form of LDL cholesterol and lower concentrations of HDL cholesterol (Goldstein, 1992). Elevated cholesterol levels have been associated with an increased risk of cardiovascular diseases (Oster & Epstein, 1986), and specifically low HDL cholesterol levels are also associated with cardiovascular morbidity (Goldstein, 1992). Although there has been considerable controversy regarding the effect of cholesterol-lowering on the incidence of cardiovascular disease (Oster & Epstein, 1986), the findings from the Lipid Research Clinics Coronary Primary Prevention Trial in 1984 provided strong scientific evidence of an association between elevated cholesterol levels and cardiovascular disease (Amarasingham, 1991; Kortt & Armstrong, 1998; Stason, 1990).

Therefore, reducing elevated LDL cholesterol levels and/or increasing low levels of HDL cholesterol may reduce the risk of cardiovascular disease (Goldstein, 1992) and a number of clinical studies have demonstrated that moderate weight loss is associated with an increase in the HDL cholesterol levels (MacMahon et al. 1985; Sopko et al. 1985). Barakat et al. (1990) observed that severely obese subjects referred for gastric by-pass surgery demonstrated that substantial reductions in weight improved cardiovascular risk by decreasing elevated LDL cholesterol levels and increasing HDL cholesterol levels. Similar results (i.e., relative levels of LDL and HDL cholesterol) were achieved in a study examining the effects of modest weight loss in subjects with type 2 diabetes mellitus (Goldstein, 1992; Wing et al. 1987).

2.5.5.5 Depression

Although clinical studies have indicated that obese individuals are no more psychologically disturbed that non-obese individuals (Foreyt & Goodrick, 1994), many obese individuals suffer from both binge eating and depression, which may be a result of unrealistic attempts to lose weight. It is these individuals who are more likely to have significant personality disturbance (Foreyt & Goodrick, 1994). Furthermore, there is evidence to suggest that heavier individuals are more likely to gain weight if depressed (Stunkard et al. 1991). The Swedish Obesity Study (SOS) has also indicated that obese adults report a worse mental health state (Sjöström, 1992). Causality appears to be reversible in this case.

2.5.5.6 Musculoskeletal Pain

A number of studies have reported that there is evidence linking obesity to a variety of musculoskeletal disorders ranging from osteoarthritis (in both the knee and hip) to joint pain (Bray, 1985; Colditz, 1992; Jung, 1997; Pi-Sunyer, 1993). For example, the prevalence of osteoarthritis with increasing body weight has been reported in several cross-sectional studies (Goldin et al. 1976; Leach et al. 1973). Furthermore, in morbidly obese subjects, McGoey et al. (1990) have reported that a 6-10 kg weight loss is associated with a relief from pain in the lower back, ankles, and feet.

2.5.5.7 Obesity and Other Risk Factors

There is also evidence to suggest that obesity is associated with several other medical conditions including gall bladder disease, respiratory disease, and some forms of cancer (Pi-Sunyer, 1993).

Pi-Sunyer (1993) indicates that increasing body weight is associated with a greater prevalence of gall bladder disease (Stampfer et al. 1992; Khare et al. 1995). This association has been documented in both longitudinal (Friedman et al. 1966) and cross-sectional (Burnett, 1971) studies. Furthermore, there is evidence to suggest that gall stones occur three to four times more often in obese individuals compared to their non-obese counterparts (Bray, 1985; Burnett, 1971, Friedman et al. 1966). Maclure et al. (1989) point out that the results from the Nurses' Health Study indicate that middle-age women are particularly at risk of developing gall bladder disease and Stampfer et al. (1992) report that obese women (i.e., a BMI > 30) had a yearly symptomatic gallstone incidence rate of greater than 1 per cent.

According to Pi-Sunyer (1993) obesity is also associated with respiratory disease. Increased adiposity in the chest and abdomen reduces lung volume, alters the respiratory system, and causes a decreased compliance of the respiratory system (Jung, 1997; Kopelman, 2000; Pi-Sunyer, 1993). Furthermore, as the level of obesity increases, sleep apnea occurs with greater frequency (Davies & Stradling, 1990; Pi-Sunyer, 1993; Shepherd, 1992).

There is also evidence to suggest that obesity is associated with certain types of cancer including endometrial, breast, prostate, and colon (Chu et al. 1991; Giovannucci et al. 1996; Helmrich et al. 1983; Hunter et al. 1993; Huang et al. 1997; Pi-Sunyer, 1993; Willett et al. 1985). For instance, in a prospective study, the American Cancer Society followed 750,000 men and women for 12 years and reported that the mortality ratio for men who were 40 per cent (or more) overweight was 1.33. For women, the corresponding mortality ratio was 1.55 (Garfinkel, 1985). Overweight men in general showed higher mortality ratios for colorectal and prostate cancer, and overweight women had higher rates for gall bladder, ovarian, cervical, and breast cancer (Garfinkel, 1985; Pi-Sunyer, 1993).

2.6 Costs of Obesity

The costs associated with obesity can be classified into three groups: (1) direct, (2) indirect, and (3) intangible.

The direct costs associated with obesity fall into two distinct categories (Drummond et al. 1987), namely:

• Organising and operating costs borne directly by the health care system (such as health care resources consumed in the diagnosis and treatment of obesity and obesity-related medical conditions); and

• Costs borne by the patient and their families. Examples of these costs include out-of-pocket medical expenses, pharmaceutical and hospital costs, and direct travel costs.

Obesity also results in indirect costs to society such as production losses. Losses in productivity due to obesity-related illness, disability, and sudden death (Gorstein & Grosse, 1994) result in lost income by the patient and their family (Langley, 1993). Therefore, production losses such as lost work time or time spent seeking treatment are referred to as indirect costs (Drummond et al. 1987).

The intangible costs associated with obesity include the psychological element of treating obesity and range from pain and discomfort resulting from surgical intervention to potential adverse drug reactions from pharmacotherapy.

To date, 13 published studies have estimated the costs associated with obesity within a cost-illness-framework (Allison et al. 1999; Birmingham et al. 1999; Colditz, 1992; Colditz, 1999; Gorsky et al. 1996; Lévy et al. 1995; Segal et al, 1994; Seidell, 1995; Swinburn et al. 1997; Wolf & Colditz, 1994, 1996; 1998; West, 1994). Table 2.6 presents the salient features of these studies. The categories in Table 2.6 include: authour/year/country, (ii) BMI criteria used to define obesity, and (iii) the principal findings

Cost-of-illness studies are designed to quantify the cost to society of a given medical condition during a given time period (Wolf, 1998). This type of analysis can either be prevalence-or incidence-based (Hughes & McGuire, 1997; Drummond, 1992).

Table 2.6: Obesity cost-of-illness studies

Author(s), Year, Country	BMI value used to define obesity	Principal Findings
Colditz, (1992) US	≥ 28.7 (men); ≥ 28.3 (women)	Direct costs attributable to obesity totalled $39.9 billion in 1986 or 5.5% of US health-care expenditure.
Wolf and Colditz, (1994), US	≥ 28.7 (men); ≥ 28.3 (women)	Direct costs attributable to obesity totalled $45.8 billion in 1990 or approximately 6% of US health-care expenditure.
Wolf and Colditz, (1996), US	≥ 28.7 (men); ≥ 28.3 (women)	Increased costs associated with type 2 diabetes and coronary heart disease were attributable to increases in BMI.
Wolf and Colditz, (1998), US	≥ 29	Direct costs attributable to obesity totalled $51.46 billion in 1995, representing 5.7% of US health care expenditure.
Colditz, (1999), US	≥ 30	Direct costs attributable to obesity totalled $70 billion in 1995, representing 7% of total health-care expenditure.
Allison et al, (1999), US	≥ 29	Percentage of lifetime cost attributable to obesity could vary from 0.89% to 4.32%.
Gorsky et al, (1996), US	≥ 29	$16.1 billion would be spent in the US over the next 25 years for the treatment of obesity-related medical conditions for overweight women in the 40-44 year age group.
Birmingham et al, (1999), Canada	≥ 27	Direct costs attributable to obesity totalled $1.8 billion in 1997, representing 2.4% of total health-care expenditure in Canada.
West, (1994), UK	≥ 30	Total cost of obesity was estimated to be in excess of £165.25 million
Seidell[17], (1995), The Netherlands	> 30	Costs of obesity were estimated at 1 billion Dutch guilders, representing about 4% of health-care costs.
Lévy et al, (1995), France	≥ 27	Direct costs attributable to obesity were estimated at 11.89 in 1992 French francs, representing 2% of health-care costs.
Segal et al, (1994), Australia	>30	Costs of obesity were estimated at $A395 million in 1989, accounting for 2% in health-care expenditure.
Swinburn et al, (1997), New Zealand	> 30	Costs attributable to obesity totalled $NZ135 million or 2.5% of total health-care costs.

Source: Adapted from Kortt et al. (1998) and Kortt (2000).

All the studies presented in Table 2.6 estimate the costs attributable to obesity using a prevalence-based cost-of-illness framework, expect for the study by Gorsky et al., (1996) which used an incidence-based approach.

Colditz (1992) has argued that a prevalence-based approach is well suited to estimating the economic burden of an acute medical condition on an annual basis. This approach, however, does not quantify the long-term consequences of diseases such as obesity. Thus, an incidence-based cost-of-illness evaluation would be more appropriate. This particular approach estimates the lifetime costs of cases diagnosed in a given year (Colditz, 1992; Drummond 1992). Drummond indicates that this approach is more "demanding in terms of data, since it requires estimates to be made of disease progression" (Drummond, 1992, p. 1). However, incidence-based cost-illness estimates also provide "a baseline against which new therapy interventions can be assessed"(Drummond, 1992, p. 1).

2.6.1 Cost of Obesity in North America

This problem has been examined extensively. Colditz (1992) conducted the first study on the economic costs associated with obesity in the US. In this study, the costs of the following obesity-related medical conditions including type 2 diabetes mellitus, gall bladder disease, CHD, hypertension, and several cancers. The cost of treating each medical condition attributable to obesity was estimated by assuming that a certain proportion of each disease occurred in obese individuals and, more importantly, that a certain proportion of those cases could be attributable to obesity. For example, Colditz (1992) assumed that 27 per cent of CHD were diagnosed in obese individuals, and that

70 per cent of these cases were attributable to obesity. Thus, 19 per cent (i.e., 0.27 x 70 per cent) of the aggregate cost of treating CHD could be attributable to obesity. The estimated costs attributable to obesity in 1986 were $22.2 billion for CHD, $2.4 billion for gall bladder disease, $1.9 billion for cancer, $1.5 billion for hypertension, and $11.3 billion for type 2 diabetes. Overall the total direct cost attributable to obesity was $39.9 billion, which "represents 5.5% of the total cost of illness in 1986" (Colditz, 1992, p. 503S).

In a follow-up study, Wolf and Colditz (1994) reported that the direct costs associated with treating obesity in 1990 were $45.8 billion. The authors also estimated that the indirect costs associated with lost productivity and mortality at $23 billion in 1990. Revising these earlier estimates, Wolf and Colditz (1998) updated and extended their analysis to estimate that the direct medical costs attributable to obesity amounted to approximately $51.64 billion in 1995, which represents 5.7 per cent of total health-care expenditure in the US. The indirect costs associated with lost productivity were estimated to be $47.56 billion. Together, the direct and indirect costs attributable to obesity were estimated at $99.2 billion in 1995.

The study by Wolf and Colditz (1998) was reanalysed by Allison and colleagues (1999). The method employed by the authors differed from Wolf and Colditz (1998) in that it examined whether the direct health-care costs associated with obesity were, in fact, offset by the increased mortality rate among obese persons (defined as a BMI $\geq$ 29). Accounting for the differing mortality rates in obese and nonobese individuals, the authors estimated the percentage of lifetime cost (from age 20-85) attributable to obesity to be 4.32 per cent. Sensitivity analyses were also conducted, indicating the direct costs

attributable to obesity could vary from 0.89 per cent to 4.23 per cent. Allison et al (1999) indicated that mortality rates among obese individuals should be taken into account so as not to overestimate the direct health-care costs attributable to obesity.

Gorsky et al (1996) used an incidence-based approach to estimate the costs associated with overweight (BMI 25-28.9) and obesity (BMI ≥ 29) among a hypothetical cohort of 10,000 women age 40-65 years. Compared to the healthy weight group (defined as a BMI range 20-24.9), the excess health-care costs associated with the overweight group were estimated at $22 million, while the obese group incurred an excess cost of $53 million. These findings were then extrapolated to the US population. Gorsky et al (1996) reported that $16.1 billion would be spent in the US over the next 25 years for the treatment of obesity-related medical conditions for overweight women in the 40-44 year age group. Moreover, each woman who maintains a BMI ≥ 25 between the age 40-65 was expected to amass $4,132 in excess health-care costs.

The cost of obesity in Canada has also been estimated by Birmingham et al (1999). To estimate the costs attributable to obesity, the authors used population attributable fractions (PAFs). The PAF for each obesity-related medical condition (such as type 2 diabetes) is the proportion of the condition that is attributable to obesity. The PAF is calculated using the formula PAF = P(rr-1)/[P(rr-1) + 1]. The PAF is the product of the prevalence of obesity (P) and the relative risk (rr) of the corresponding obesity-related medical condition (which is type 2 diabetes in this example) [Birmingham et al. 1999; Huges & McGuire, 1997; Segal et al. 1994; Swinburn et al. 1997]. In this study, obesity was defined as a BMI ≥ 27. The following obesity-related medical conditions included: coronary artery disease, hyperlipidemia, hypertension, pulmonary embolism, stroke,

type 2 diabetes, and several cancers. The authors estimated that the direct costs attributable to obesity in 1997 was in excess of $1.8 billion, representing 2.4 per cent of Canada's total health-care expenditure for all diseases.

2.6.2 Impact of Weight Gain on the Cost of Obesity in the US

The social and economic effects of weight gain in American adults aged 18 and over has been examined by Wolf and Colditz (1996). In their analysis of earlier work (Colditz, 1992; Wolf & Colditz, 1994) they addressed the following questions: (i) at what body weight do we implement preventative weight gain strategies?, and (ii) what are the direct costs associated with weight gain? The authors also examined the indirect costs associated with obesity: the impact of obesity on restricted-activity, bed days, and work loss days was analysed. These questions were addressed using a prevalence-based cost-of-illness framework.

The population attributable risk per cent was used to estimate the proportion of obesity-related diseases within the following BMI categories: (i) 23-24.9, (ii) 25-29, and (iii) greater than or equal to 30. Within this framework, the direct and indirect costs associated with type 2 diabetes, coronary heart disease (CHD), hypertension, and gall stones at three different levels of BMI were estimated. It is interesting that the estimated direct costs associated with type 2 diabetes and CHD are directly attributable to increases in weight gains. For weight gains of 5-10 kg, 11-19.9 kg, or greater than or equal to 20 kg the estimated costs of NIDDM increased by $1.56, $4.61, and $6.88 billion, respectively. The estimated costs of CHD of $2.99 and $4.76 billion for the two lower weight gain categories were as expected but the relatively lower cost of $4.2 billion for the highest gain may reflect a higher death rate (Wolf & Colditz, 1996).

The results for this study are consistent with the conclusion that direct costs rise with increases in BMI. A similar relationship was also observed for indirect cost estimates, although these estimates may be confounded by smoking status and pre-existing illness. This study suggests that a healthy body weight appears to be a BMI less than 25, and that weight gain should be kept to less than 5 kg throughout a lifetime (Wolf & Colditz, 1996).

2.6.3 Cost of Inactivity and Obesity in the US

Colditz (1999) has also examined the economic costs of inactivity and obesity. A range of obesity-related diseases were examined and PAFs were used to calculate the proportion of disease that could be averted by eliminating inactivity or obesity. Inactivity was defined as the median prevalence (28.8 per cent) for adults who reported no leisure time devoted to physical activity in 1995. The prevalence of inactivity and the relative risk associated with a number of obesity-related diseases (such as type 2 diabetes, hypertension, and CHD) were used to calculate the PAFs. For example, it was estimated that 22 per cent of CHD was attributable to inactivity, representing a direct cost of $8.9 billion. Overall, Colditz (1999) estimated that the direct costs associated with inactivity total $24.3 billion per annum, representing 2.4 per cent of total health-care expenditure in the US. Moreover, Colditz (1999) also updated an earlier estimate of the costs attributable to obesity (Wolf & Colditz, 1998). The revised estimate indicates that the direct costs attributable to obesity totalled $70 billion, representing 7 per cent of total health-care costs in the US (Colditz, 1999).

2.6.4 Cost of Obesity in United Kingdom and Europe

The cost of obesity has also been estimated for the United Kingdom. West (1994), using a similar approach to that of Colditz (1992) and Wolf and Colditz (1994), estimated the total cost of obesity to be in excess of £165.25 million. McIntyre (1998) has reported that the estimated cost of obesity in the UK represents between 1 per cent and 5 per cent of total health care expenditure.

The costs attributable to obesity have also been estimated for several European countries. Seidell (1995) used a prevalence-based approach to report on the direct cost of treating obesity in The Netherlands. It was estimated that the cost of treating obesity amounted to about 1 billion Dutch guilders, approximately 4 per cent of the total Dutch health care cost.

Lévy and associates (1995) estimated the economic cost of obesity for France using a prevalence-based cost-of-illness model. In this study, obesity was defined as a BMI greater than or equal to 27. A prevalence-based cost-of-illness model was used and the direct costs and indirect costs were estimated in this analysis. Direct costs included personal health care, hospital care, physician services, and drugs, and the indirect costs were measured as lost output due to a reduction of productivity caused by morbidity and mortality (Lévy et al. 1995). The method used by Lévy et al (1995) was similar to that of Wolf and Colditz (1994, 1996) and population attributable risk percentages were used to estimate the costs of obesity-related diseases in 1992 French Francs. The direct cost of obesity for France in 1992 was estimated at 11.89 billion Francs while the indirect cost estimate was 0.6 billion Francs. These obesity-related costs represent approximately 2 per cent of the total health care costs in France.

2.6.5 Cost of Obesity in Australasia

Segal et al. (1994) have reviewed the Australian direct health care costs associated with obesity. The Australian Institute of Health and Welfare estimated that, in 1989, obesity and obesity-related diseases were responsible for 50,931 hospital admissions, 433,165 hospital bed-days, 3.1 medical consultations, 6.7 millions pharmaceutical prescriptions, and 164,903 referrals to health practitioners. Again, population attributable risk percentages were used to estimate the cost of diseases such as NIDDM, gall stones, CHD, hypertension, breast cancer, and colon cancer that are directly attributable to obesity. The cost attributable to obesity in 1989 was estimated to be $A395 million. However, Segal et al. (1994) indicate that this figure is an underestimate "as about 15% of total health expenditure is not captured in the categories costed by the model" (Segal et al. 1994, p. 48).

In 1997, Swinburn et al. (1997) estimated the costs attributable to obesity in New Zealand. As with the previous studies, this New Zealand study was designed to estimate the costs of obesity-related diseases such as type 2 diabetes mellitus, coronary heart disease, hypertension, post-menopausal breast cancer, and colon cancer. The costs associated with these medical conditions were then "multiplied by the population attributable risk factor for obesity for each condition" (Swinburn et al. 1997, p. 891) using the method described by Segal et al. (1994). The results suggest that the costs attributable to obesity totalled $NZ135 million, representing approximately 2.5 per cent of total health care expenditure.

2.7 Discussion of Cost Studies

The majority of studies reviewed have estimated the costs attributable to obesity within a prevalence-based cost-of-illness framework. While it is difficult to make comparisons across the different studies, it is clear that the costs associated with obesity are substantial. Estimates from a number of countries indicate that obesity represents between 2 and 7 per cent of total health-care expenditure in industrialised countries.

However, while these studies provide an estimate of the economic burden associated with obesity they have limitations. To begin with, a number of studies used different BMI criteria to defined obesity, which clearly influence the estimates. Different definitions of obesity also make it difficult to make comparisons across the different studies.

A similar point can also be made for the selection of the obesity-related medical conditions. Estimates of the costs attributable to obesity are influenced by the selection of obesity-associated diseases. For example, exclusion of obesity-related risk factors would tend to underestimate the economic burden associated with obesity. However, as Lévy et al (1995) indicate, a lack of suitable data will also result in an underestimate. Another point raised by Allison et al (1999) is that the mortality rates among obese and nonobese individuals should be taken into account so as not to inflate the estimated economic burden of obesity.

There is a dearth of studies that attempt to explicitly model the association between the cost of medical care utilisation and the level of obesity. In fact, only two studies have used individual-level data to examine the relationship between health-care expenditure

and the level of BMI for a sample of the US population (Heithoff et al. 1997; Quesenberry et al. 1998). The study by Heithoff et al (1997) reported a positive relationship between health-care expenditures and body mass using data from the 1987 US Nation Medical Expenditure Survey. Quesenberry et al (1998) also reported, in study among members of a health maintenance organisation (HMO), a positive statistical association between health-care costs and the level of BMI. For example, compared to individuals in a healthy BMI range (20-24.9), the average annual total costs were 25 per cent higher for those individuals with a BMI range of 30 to 34.9, and 44 per cent greater for those individuals with a BMI in excess of 35. The individual-level data that were used in these studies are particularly useful as it can provide a range of information on personal characteristics and resource utilisation to aid economic analysis.

Although numerous studies have indicated that the costs associated with obesity are substantial, further research is needed. Specifically, there is a need to examine, in greater detail, the relationship between the cost of medical care utilisation and the level of BMI. Considering the availability of individual-level data (especially in the US), regression techniques can be used not only to examine the relationships between BMI and obesity-related medical conditions but also the potential cost savings associated with a reduction in body size. Public health authorities and providers of health-care services would be particularly interested to know the potential cost savings associated with a reduction in weight.

As outlined in chapter 1, the purpose of this study is to examine the relationships between MCU, obesity, and obesity-related medical conditions. This is the first

economic study in Australia that explicitly models the association between MCU and obesity using micro-level household survey data and it examines a number of issues. The first issue to be examined is whether there is a positive relationship between MCU and obesity to establish that MCU and hence costs increase with obesity. The second issue is to establish if the relationship operates partly or totally through obesity-related risk factors. A third issue is to examine the cost implications of weight reduction in obese individuals.

CHAPTER 3: OBESITY ISSUES IN AUSTRALIA

3.1 Introduction

This chapter addresses the issues of obesity in Australia and is divided into two major sections. First, the empirical literature on obesity in Australia is reviewed and issues ranging from current trends in obesity to the socioeconomic characteristics of obesity are addressed. Second, a detailed description (and discussion) of the Australian Bureau of Statistics (ABS) 1995 National Health Survey is presented.

3.2 Obesity Trends in Australia

Are Australian's becoming more obese? According to data collected from the National Heart Foundation of Australia (NHFA) Risk Factor Prevalence Studies, since the early 1980s there has been an increase in the proportion of the Australian population that is classified as being either overweight ($25 < BMI \leq 30$) or obese ($BMI > 30$).

In a recent study, Bennett and Mangus (1994) examined the impact of hypertension, blood lipids, height and weight, smoking status, alcohol consumption, dietary behaviour, and exercise on the cardiovascular risk factor profile of Australian adults aged 25 to 64. The data for this analysis were drawn from the Risk Factor Prevalence Study conducted by the National Heart Foundation of Australia (National Heart Foundation of Australia & Australian Institute of Health, 1991; Risk Factor Prevalence Study Management Committee, 1990) and the authors identify the "changes in levels of risk factors which might be associated with the continuing fall in cardiovascular mortality rate" (Bennett & Mangus, 1994, p. 520).

This study is relevant to the present work as Bennett and Mangus (1994) reported on the changes in both height and weight (BMI) for men and women during the 1980s. The data for weight (in kilograms) and BMI (i.e., weight for height) for men and women is presented in Tables 3.1 and 3.2, respectively.

Table 3.1: Weight and BMI for men (age 25-64)

	1980	*1983*	*1989*	*1980-1989*
Age in Years		*Weight (kg) Mean*		*Difference*
25-34	75.6	75.1	77.5	+1.7*
35-44	78.8	77.2	79.0	-0.1
45-54	77.4	78.8	80.4	+2.9*
55-64	76.4	76.9	78.6	+2.2*
25-64	77	77.0	78.8	+1.7*
BMI Categories		*BMI Crude (%)*		*Odds Ratio*
BMI < 20	4.8	4.7	3.1	0.72*
BMI 20-25	45.4	46.2	41.3	0.86**
25< BMI ≤ 30	40.6	40.0	44.1	1.13*
BMI > 30	9.3	9.1	11.5	1.24*
Overweight/Obese BMI > 25	49.8	49.1	55.6	1.23**

Notes:
1. *$p < .05$. **$p < .01$.
2. Differences and odds ratios are adjusted for survey design factors.
Source: Adapted from Bennett and Mangus (1994)

The data presented in Table 3.1 indicate that Australian men in 1989 were over 1.7 kg heavier, on average, compared with their 1980 counterparts (Bennett & Mangus, 1994). Men in the age group 45 to 54 experienced the largest weight increase – 2.9 kg – during the 1980s. For Australian men, the odds of being overweight (25 < BMI ≤ 30) or obese (BMI >30) increased by 13 per cent and 24 per cent, respectively.

The data presented in Table 3.2 indicate that Australian women were also heavier – over 3 kg on average – compared with women of the same age in 1980. Women in the age group 45 to 54 experienced the largest weight increase – 4 kg – during the 1980s. For

women, the odds of being overweight or obese increased by 32 per cent and 70 per cent, respectively.

In general, although women on average experienced greater weight gains during the 1980s the prevalence of overweight and obesity in adult men was considerably greater than when compared with women. According to Bennett and Mangus (1994), 55.6 per cent of Australian men in 1989 aged 25 to 64 were classified as being either overweight or obese. By comparison, 38.3 per cent of non-pregnant Australian women in 1989 were classified as being either overweight or obese.

Table 3.2: Weight and BMI for non-pregnant women (age 25-64)

	1980	*1983*	*1989*	*1980-1989*
Age in Years		*Weight (kg) Mean*		*Difference*
25-34	58.7	60.4	61.8	+2.8*
35-44	61.4	62.5	63.9	+2.3*
45-54	63.2	64.8	67.3	+4.0*
55-64	63.7	64.6	67.0	+3.2*
25-64	61.7	63.0	64.8	3.1*
BMI Categories		*BMI Crude (%)*		*Odds Ratio*
BMI < 20	16.7	14.1	11.8	0.69**
BMI 20-25	55.0	53.4	49.8	0.81**
25< BMI ≤ 30	20.2	22.0	25.1	1.32**
BMI > 30	8.0	10.5	13.2	1.70**
Overweight/Obese BMI >25	28.3	32.5	38.3	1.58**

Notes:
1. $*p < .05$. $**p < .01$.
2. Differences and odds ratios are adjusted for survey design factors.
Source: Adapted from Bennett and Mangus (1994)

There is evidence to suggest that the trend towards weight gain has continued into the 1990s. More recently, an examination of self-reported survey data from the Australian Bureau of Statistics (ABS) indicates that prevalence of being overweight or obese has increased substantially over five years (Abraham et al. 1995; Australian Institute of

Health and Welfare, 1995). Tables 3.3 and 3.4 provide relatively recent estimates of the percentage of Australian adults who are overweight or obese.

Table 3.3: Percentage of adults who are overweight or obese

		1989-90	*1994-95*	*Change over 5 years (%)*
Sex:	Men	44.4	49.7	11.9*
	Women	30.9	33.5	8.5
Age:	18-34	27.1	32.1	18.4*
	35-54	43.9	46.9	6.8
	55+	45.0	49.2	9.4

Notes:
1. * indicates that the difference between the 1989 and 1994 percentage is statistically significant at the 95 per cent level.
2. All proportions have been age-adjusted using the total 1991 Australian population.
3. The 1989-90 estimates are based on the ABS National Health Survey that was conducted between October 1989 and September 1990.
4. The 1994-95 estimates are based on the ABS 4. Population Survey Monitor that was conducted in May, August, and November of 1994.
Source: Adapted from the Australian Institute of Health and Welfare (1995).

The data presented in Table 3.3 indicate that the rate of overweight and obesity among adult men aged 18 to 34 has increased substantially from 27.1 per cent in 1989-90 to 32.1 per cent in 1994-95. Over a five-year period this represents a significant increase (18.4 per cent). According to these data, Australian men, on average, have experienced a greater increase than women in the rate of overweight and obesity. Similar results, are observed for the age group 25 to 64 presented in Table 3.4 (Abraham et al. 1995)

Table 3.4: Percentage of adults (aged 25-64) classified and overweight and obese

		1989-90	*1994-95*	*Change over 5 years (%)*
Sex:	Men	48.6	54.5	12.1
	Women	32.9	34.9	6.1

Notes:
1. The 1989-90 estimates are based on the ABS National Health Survey that was conducted between October 1989 and September 1990.
2. The 1994-95 estimates are based on the ABS Population Survey Monitor that was conducted in May, August, and November of 1994.
3. The proportions are age-adjusted using the total Australian population as at June 30, 1991.
Source: Adapted from Abraham et al. (1995).

The data in Table 3.4 clearly demonstrates that the proportion of overweight and obese Australian men and women has increased. For men, the rate of overweight and obesity, increased from 48.6 per cent in 1989-90 to 54.5 per cent in 1994-95, representing a 12.1 per cent increase over a five year period. On the other hand, women, over the same five-year period, experienced a 6.1 per cent increase.

3.3 Overweight and Obesity in Australian Adults

This statistical evidence clearly shows that Australians are becoming heavier and that a substantial proportion of Australians, especially men, are either overweight or obese. Estimates of the prevalence of overweight and obesity "based on measured height and weight come from the National Heart Foundation's risk factor survey in 1989" (National Health and Medical Research Council, 1997, p. 73) and the principal results from this survey are present in Table 3.5.

Table 3.5: Overweight and obesity based on measured height and weight from men and non-pregnant women (aged 20-69), 1989

	Age										
BMI (%)	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	20-69
Men											
Overweight	19	30.3	36.3	42.9	42.9	46.2	44.6	44.6	49.0	49.5	38.6
Obese	6.4	5.9	5.8	7.9	9.3	12.1	15.4	16.0	10.9	11.5	9.3
Overweight or Obese	25.4	36.2	42.1	50.8	52.2	58.3	60.0	60.6	59.9	61.0	47.9
Women											
Overweight	13.1	13.3	14.5	20.6	23.6	29.0	30.0	28.8	37.2	36.3	22.4
Obese	4.4	7.8	7.7	8.0	6.8	14.9	19.3	23.0	15.2	20.6	11.1
Overweight or Obese	17.5	21.1	22.2	28.6	30.4	43.9	49.3	51.8	52.4	56.9	33.5

Source: Adapted from the Risk Factor Prevalence Study Management Committee (1990).

These results indicate that 48 per cent of men and 34 per cent of non-pregnant women were either overweight or obese in 1989. The prevalence of overweight and obesity was lowest among adults aged 20 to 24 and increased with age. Moreover, a greater proportion of men when compared with women were either overweight or obese.

These findings are supported by the more recent estimates provided by the Population Survey Monitor conducted by the Australian Bureau of Statistics in May, August and November 1994, and February 1995 (Australian Institute of Health & Welfare, 1995). The estimates derived from this survey are based on self-reported height and weight and indicate that 50 per cent of men and 34 per cent of women (aged 18 and over) were classified as being either overweight or obese (Australian Institute of Health & Welfare, 1995).

Recent estimates on the prevalence of overweight and obesity are also available from the 1995 National Health Survey which have been published by the Australian Bureau of Statistics (ABS, 1997a). This survey also used self-reported height and weight to estimate the prevalence of overweight and obesity for the Australian population and found that 41 per cent of men and 30 per cent of women aged 25 to 64 were either overweight or obese during the survey period (ABS, 1997a), a result consistent with other recent surveys.

3.4 Overweight and Obesity in Australian Sub-Populations

The majority of obesity studies in Australia have focused upon the general Australian population. However, a limited number of studies that have examined both the prevalence of overweight and obesity for various Australian sub-populations. Several

studies, in particular, have examined the prevalence of obesity in both Australian Aboriginals and Torres Strait Islanders, and Australian immigrants.

3.4.1 Overweight and Obesity in Aboriginal and Torres Strait Islander People

Several New South Wales Better Health Program projects have set out to measure the prevalence of overweight and obesity among a sample of Aboriginal people. The results from these finding, summarised by Boyle and Dobson (1992), are presented in Table 3.6. Again it should be noted that as with previous studies on this subject the BMI cut off point used to define overweight and obesity differed in each study, making it difficult for comparisons to be made.

The Cardiovascular Disease Risk Factor Screening undertaken in 1989 indicated that 64 per cent of Aboriginal women were either overweight or obese as shown in Table 3.6. It was estimated that 46 per cent of Aboriginal men were either overweight or obese (Boyle & Dobson, 1992). However, the other studies indicate that the proportion of Aboriginal men who are either overweight or obese is substantially lower. These results should be treated with caution as several factors such as: (i) the regions studied, and (ii) the relatively small sample size may actually under-estimate the proportion of Aboriginal adults who are either overweight or obese.

More recent estimates derived from the National Aboriginal and Torres Strait Islander Survey (National Health and Medical Research Council, 1997) indicate that a higher proportion of Aboriginal and Torres Strait Islanders are either overweight or obese. In 1994, the ABS conducted a National Aboriginal and Torres Strait Islander Survey (National Health and Medical Research Council, 1997), in which height and weight

were measured. The results of this survey indicate that for adults aged 18 years and older, 60 per cent of men and 58 per cent of women were classified as either being overweight or obese (i.e., a BMI > 25) [National Health and Medical Research Council, 1997].

Overall, the studies undertaken to date indicate that a substantial proportion of Australian Aboriginals and Torres Strait Islanders are above what is considered to be an 'acceptable' body weight (i.e., a BMI > 25) and that the prevalence of obesity and overweight is greater than for the population as a whole. A consequence of this is that the cost of treating obesity-related risks factors in this sub-population are likely to be substantial and higher than for the rest of the Australian population.

Table 3.6: Prevalence of overweight and obesity among Aboriginal people in the New South Wales Better Health Program projects

				Percentage overweight or obese	
Region	*Project*	*Age*	*Sample*	*Men*	*Women*
Orana/Far West	Cardiovascular Disease Risk Factor Screening, 1989	20-70	209	46 (BMI > 26)	64 (BMI > 25)
New England	Purfleet, Forster Aboriginal Diabetes Study, 1985	18+	118	27 (BMI > 27)	53 (BMI > 25)
Orana/Far West	Bourke Enngonia -- Aboriginal Diabetes Study, 1982-83	20+	294	30 (BMI > 27.5)	42 (BMI > 27)

Source: Boyle & Dobson (1992) after the National Health and Medical Research Council (1997)

3.4.2 Overweight and Obesity in Australian Immigrants

NHMRC surveys also indicate that there is a prevalence of overweight and obesity "among immigrants to Australia from Southern Europe" (National Health and Medical Research Council, 1997, p. 77). Results from the National Heart Foundation's risk

factor survey in 1989 point out that among 20 to 69 years old the odds (or likelihood) of being overweight or obese was almost two times greater (i.e., odds ratio = 1.96) for men born in Southern Europe compared with native-born Australian men. Moreover, the odds of being overweight or obese was three times greater (i.e., OR = 3) for women born in Southern Europe compared to women born in Australia. In contrast, the odds of being overweight or obese were lower for both Asian born men (OR = 0.52) and women (OR = 0.84) when compared to native-born Australian men and women, respectively. Details of these survey results are presented in Table 3.7.

Table 3.7: Region of birth differentials in the prevalence of overweight and obesity based on measured height and weight for men and women (aged 20-69) in 1989

Region of Birth	*Sample Size*	*Crude % of overweight and obese*	*Odds ratio (OR)*
Men			
Australia	3265	49	1
Untied Kingdom	490	44.7	0.7
Northern Europe	214	67.4	1.52
Southern Europe	249	68.5	1.96
Asia	202	34.5	0.52
Other	77	40.8	0.84
Women			
Australia	3452	31.4	1
Untied Kingdom	426	34.3	1.02
Northern Europe	192	42.2	1.14
Southern Europe	246	56.4	3
Asia	189	30.4	0.84
Other	69	36.6	1.63

Source: National Health and Medical Research Council (1997).

Mathers (1994a; 1994b; 1996) has reported similar results based on self-reported height and weight from the National Health Interview Survey that was conducted by the ABS from October 1989 to September 1990 (National Health and Medical Research Council, 1997). The three reports by Mathers were designed to document the health differentials in Australia for each of the following immigrant groups: (i) young adults aged 15 to 24, (ii) adults aged 25 to 64, and (iii) older adults aged 65 years and over (Mathers, 1994a, 1994b, 1996).

Several points are worth noting about the results included in the three reports. The first is that many of the health indicator rates, such as the prevalence of overweight and obesity, rise with age (Mathers, 1994b). Therefore, the age distribution of population sub-groups can greatly influence the crude rates. To avoid this bias all health indicators were age standardised to the total mid-year Australian population in 1988, enabling Mathers (1994b) to conclude that this "produces an estimate of the rate that would have prevailed in the standard population if it had experienced the age-specific rates for the given indicator, thus eliminating from the differentials reported here the effects of the age distributions of the population subgroups" (Mathers, 1994b, p. 4).

A second point worth noting is that the differentials between populations are expressed in terms of rate ratios. For instance, country of birth differentials are given in terms of the foreign-born rate to the Australian-born rate. In other words, the Australian-born category is defined as the reference group. Therefore, a ratio of 1 indicates that there is no difference between the proportion of overweight and obese people between the two groups. Table 3.8 presents the region of birth differentials in overweight and obesity derived from the 1989 National Health Interview Survey.

The results in Table 3.8 indicate that men and women in the age group 25 to 64 who were born in "Other Europe" were respectively 1.15 and 1.35 times more likely to be either overweight or obese compared with their Australian counterparts (Mathers, 1994b). It is also noteworthy that for men and women in the age group 65 years and older who were born in "Other Europe" were respectively 1.35 and 1.40 times more likely to be either overweight or obese compared with their Australian counterparts.

Table 3.8: Region of birth differentials in the prevalence of overweight and obesity based on self-reported height and weight for men and women, 1989

Age Group / Region of Birth	*Men*		*Women*	
	Prevalence rate (%)	*Rate ratio*	*Prevalence rate (%)*	*Rate ratio*
18-24				
Australia	26.33	1.00	15.42	1.00
UK, Ireland	31.64	1.20	14.40	0.93
Other Europe	27.67	1.05	12.25	0.79
Asia	24.45	0.93	11.73	0.76
Other	16.27	0.62	10.39	0.67
25-64				
Australia	49.31	1.00	32.39	1.00
UK, Ireland	43.47	0.88**	28.64	0.88*
Other Europe	56.47	1.15**	43.77	1.35***
Asia	36.78	0.75***	28.73	0.89
Other	45.20	0.92	28.00	0.86
65+				
Australia	42.50	1.00	36.46	1.00
UK, Ireland	39.22	0.92	33.61	0.92
Other Europe	57.58	1.35**	50.89	1.40**
Asia	33.29	0.78	40.70	1.12
Other	46.68	1.10	31.59	0.87

Notes:
1. $*p < .05$. $**p < .01$. $***p < .0001$
2. All estimates are age-standardised by the mid-1988 Australian population.
3. The reference group is Australia.
4. Regions are defined as: (i) UK, Ireland: United Kingdom and Ireland; (ii) Other Europe: includes Eastern Europe, USSR, and Baltic States; (iii) Asia: Middle East, South East Asia, Southern Asia; (iv) Other: North East Asia (including China, Hong Kong, Japan, and Korea), Northern and Southern Africa, the Americans, New Zealand, and the Pacific region.
Source: Mathers (1994a, 1994b, 1996).

Bennett (1993) has examined the inequalities in risk factors and cardiovascular mortality among Australian immigrants. A sample consisting of 6,116 immigrants to Australia and 14,941 native-born Australians was drawn from the 1980, 1983, and 1989 National Heart Foundation's Risk Factor Prevalence surveys. Bennett systematically compared "the levels of biomedical and behavioural risk factors among a range of immigrant groups in Australia, using native-born Australians as a reference group" (Bennett, 1993, p. 251). After adjusting for both age and survey design, Bennett (1993) reported statistically significant differences between immigrants to Australian and

native-born Australians. In particular, differences were noted for systolic blood pressure, behavioural risk factors and, more importantly, obesity.

This study is of interest because like Mathers' (1994a, 1994b, 1996) work, the results for obesity (BMI) "suggest marked differences in overall obesity among immigrant groups" (Bennett, 1993, p. 253). For example, compared with native-born Australians, men from the Asian regions and the UK had statistically significantly lower average BMI levels (Bennett, 1993). For women, the results were similar but less pronounced. Moreover, men and especially women from Southern European regions and the Middle East had, on average, higher BMI levels than native-born Australians.

3.5 State and Regional Characteristics of Overweight and Obesity

Data drawn from the National Heart Foundation's risk factor survey indicates that the prevalence of overweight and obesity varies between major cities in Australia. For example, men and women residing in Adelaide had the greatest prevalence of overweight and obesity in 1989, with 55 per cent of men and 38 per cent of women classified as being either overweight or obese. In contrast, the prevalence of overweight and obesity was lowest for men residing in Sydney and women residing in Darwin. Data comparing the proportion of overweight and obese individuals by cities are presented in Table 3.9.

Mathers (1994a, 1994b) has also examined the differentials in the prevalence of overweight and obesity by State and Territory, which are summarised in Table 3.10. Although no statistically significant differentials were found, those individuals who were more likely to be either overweight or obese, compared to the national average,

were older males (aged 65+) residing in the Australian Capital Territory (ACT), and South Australian (SA) and older females (65+) residing in the Northern Territory (Mathers, 1994a; Mathers, 1994b).

Table 3.9: Overweight and obesity based on measured height and weight from men and non-pregnant women (aged 20-69) in Australian capital cities, 1989

	Sydney	*Melbourne*	*Brisbane*	*Adelaide*	*Perth*	*Hobart*	*Darwin*	*Canberra*
Men				%				
Overweight	37.1	37.5	40.1	42.9	40.3	40.4	37.0	40.0
Obese	7.5	10.7	9.2	12.0	9.4	13.0	9.5	9.8
Overweight or Obese	44.6	48.2	49.3	54.9	49.7	53.4	46.5	49.8
Women								
Overweight	20.2	22.8	24.1	24.2	23.3	24.8	20.4	24.5
Obese	10.4	11.8	10.2	14.1	10.7	12.8	8.4	12.2
Overweight or Obese	30.6	34.6	34.3	38.3	34.0	37.6	28.8	36.7

Source: Adapted from the Risk Factor Prevalence Study Management Committee: Cities Analysis (1991).

Thompson et al. (1995) examined the regional differences in obesity (as measured by BMI) in Tasmania. The authors used data drawn from the Hobart component of the National Heart Foundation's risk factor survey conducted in 1989 and used these data as an indicator of the risk factor profile of the southern region of Tasmania. These data were then compared with data collected in the north and north-west regions of Tasmania. Results indicate that for both men and women, the mean BMI was greatest in the north-west region of Tasmania (a BMI of 26.5 for men and a BMI 25.8 for women, respectively) and lowest in the southern region (as a BMI of 25.7 for men and a BMI of 24.8 for women, respectively).

Table 3.10: State and Territory differentials in the prevalence of overweight and obesity based on self-reported height and weight for men and women, 1989

State / Territory	*Men*		*Women*	
Age	*Prevalence rate (%)*	*Rate ratio*	*Prevalence rate (%)*	*Rate ratio*
25-64				
Australia	48.64	1.00	32.80	1.00
NSW	47.49	0.98	32.01	0.98
Victoria	49.85	1.02	34.42	1.05
Queensland	50.20	1.03	32.52	0.99
WA	46.45	0.95	32.01	0.98
SA	50.43	1.04	33.84	1.03
Tasmania	49.03	1.01	32.79	1.00
ACT	44.67	0.92	27.33	0.83
NT	50.41	1.04	27.14	0.83
65+				
Australia	43.58	1.00	37.43	1.00
NSW	40.52	0.93	36.09	0.96
Victoria	45.54	1.04	36.87	0.99
Queensland	41.25	0.95	39.65	1.06
WA	46.47	1.07	39.92	1.07
SA	50.76	1.16	39.35	1.05
Tasmania	41.24	0.95	35.53	0.95
ACT	52.78	1.21	31.31	0.84
NT	25.61	0.59	48.37	1.29

Notes:
1. All estimates are age-standardised by the mid-1988 Australian population.
2. The reference group is Australia.
3. Where: (i) NSW: New South Wales; (ii) WA: Western Australia; (iii) SA: South Australia; (iv) ACT: Australian Capital Territory; and (v) NT: Northern Territory.

Source: Mathers (1994a) and Mathers (1994b).

3.6 Socioeconomic Characteristics of the Overweight and Obese

Waters and Bennett (1995) report on the socioeconomic differentials related to the prevalence of overweight and obesity for Australian men and women aged 20 to 69 based on data were drawn from the 1989 Risk Factor Prevalence Survey. Broadly speaking, the prevalence of overweight and obesity in adult men and women is associated with the level of education, martial status, and occupation. The socioeconomic differentials in the prevalence of overweight and obesity for men (aged 20 to 69) is presented in Table 3.11.

Table 3.11: Socioeconomic differentials in the prevalence of overweight and obesity based on measured height and weight for men, 1989

Socioeconomic variable	*Sample Size*	*Crude (%) overweight or obese*	*Adjusted rate ratio for overweight or obese*	*Crude (%) obese*	*Adjusted rate ratio for obese*
Level of education					
Tertiary	1380	46.4	1.0	8.2	1.0
Completed high school	1314	51.1	1.3	9.9	1.2
Some high school	1435	56.9	1.4	12.4	1.5
Never attended school or attended primary school only	368	66.6	1.5	19.8	2.2
Marital Status					
Never married	917	35.0	1.0	7.9	1.0
Now married	3215	57.6	1.9	11.7	1.2
Separated	108	53.7	1.6	7.4	0.7
Divorced	205	54.6	1.6	14.6	1.5
Widowed	51	60.8	1.6	13.7	1.1
Occupation					
Professionals	767	43.6	1.0	7.2	1.0
Managers or Administrators	700	60.0	1.8	12.1	1.7
Para-professionals	350	46.9	1.2	8.6	1.2
Tradepersons	660	50.8	1.3	10.5	1.5
Clerks	347	47.0	1.2	9.5	1.3
Salespersons	304	50.3	1.3	9.2	1.3
Plant, machine operators, & drivers	278	60.8	1.9	15.8	2.4
Labourers	307	56.0	1.6	12.7	1.8
Not employed full or part -time.	755	59.1	1.1	14.0	1.5

Notes:
1. Adjusted for age and city.
2. Reference group for variables includes those individuals who are classified as being: (i) tertiary educated, (ii) never married, and (iii) professionals.
Source: Waters and Bennett (1995).

It is noteworthy that a number of socioeconomic factors were associated with increased odds of being obese (i.e., a BMI > 30). For instance, for adult men the odds of being obese was 2.2 times greater for those men who never attended school or attended primary school compared to tertiary educated men. Furthermore, the odds of being obese were 1.5 times greater for men who attended some high school compared to tertiary educated men (Waters and Bennett, 1995). In addition, it is interesting to note,

that the odds of being obese were 1.5 times greater for men who were divorced compared to men who never married.

In occupational terms for adult men, the odds of being obese were 2.4 times greater in plant/machinery operator/driver occupations compared with those men in professional occupations (Waters and Bennett, 1995). Also, the odds of being obese was 1.7 times greater for men in management and administration compared to those men in professional occupations.

For women, the socioeconomic differentials in the prevalence of overweight and obesity are presented in Table 3.12. This table is also categorised by socioeconomic variables. With reference to the level of education, the odds of being obese were 2.2 times greater for women who never attended school or only attended primary school compared to those women who were tertiary educated (Waters and Bennett, 1995). Furthermore, the odds of being obese was 1.7 times greater for women who attended some high school compared to those women who were tertiary educated.

In terms of the type of occupation, the odds of being obese were 2.9 times greater for women in plant/machinery, operator/driver occupations compared to those women in professional occupations (Waters and Bennett, 1995). Moreover, the odds of being obese was 2.3 times greater for women labourers compared to those women in professional occupations.

Table 3.12: Socioeconomic differentials in the prevalence of overweight and obesity based on measured height and weight for women, 1989

Socioeconomic variable	*Sample Size*	*Crude (%) overweight or obese*	*Adjusted rate ratio for overweight or obese*	*Crude (%) obese*	*Adjusted rate ratio for obese*
Level of education					
Tertiary	1135	26.8	1.0	7.9	1.0
Completed high school	1268	32.2	1.2	9.8	1.2
Some high school	1737	40.8	1.6	14.8	1.7
Never attended school or attended primary school only	434	62.0	2.6	22.4	2.2
Marital Status					
Never married	808	26.6	1.0	7.8	1.0
Now married	3062	39.3	1.0	13.3	1.2
Separated	125	28.8	0.7	10.4	1.0
Divorced	336	30.7	0.7	11.3	1.0
Widowed	241	54.4	1.0	19.1	1.2
Occupation					
Professionals	562	26.5	1.0	6.6	1.0
Managers or Administrators	189	36.5	1.5	10.6	1.6
Para-professionals	228	26.8	1.1	9.2	1.5
Tradepersons	76	39.5	1.7	10.5	1.6
Clerks	973	30.2	1.2	9.2	1.5
Salespersons	430	35.0	1.6	10.5	1.7
Plant, machine operators, & drivers	54	35.2	1.3	18.5	2.9
Labourers	238	41.2	1.7	15.1	2.3
Home Duties	1416	44.2	1.5	16.7	1.7
Not employed full or part -time.	397	48.8	1.4	16.4	2.1

Notes:
1. Adjusted for age and city.
2. Reference group for variables includes those individuals who are classified as being: (i) tertiary educated, (ii) never married, and (iii) professionals.
Source: Waters and Bennett (1995).

3.7 Summary of Evidence on Obesity in Australia

By way of summary, a number of conclusions can be made. First, the Australian population, over time, is becoming heavier. There is sufficient evidence to suggest that, on average, Australian men (compared to women) are becoming more obese. There is also a growing body of empirical literature indicating that the rate of being overweight

or obese among the Australian population is influenced by a variety of factors ranging from racial background to socioeconomic status. Most of the empirical data on obesity are primarily have been drawn from two major data sources: (i) the National Heart Foundation of Australia Risk Factor Prevalence Surveys, and (ii) the 1989 Australian Bureau of Statistics National Health Survey. A major difference in these surveys is that those conducted by the National Heart Foundation calculated their BMI values based on measured height and weight while the BMI values calculated from the 1989 National Health Survey were based on self-reported height and weight. Finally, these data were primarily collected during the 1980s. Recently, the Australian Bureau of Statistics (ABS) published the results from the 1995 National Health Survey, which contains the most up to date data on the health of the Australian population. In addition, this survey also contains recent estimates of the rate of overweight and obesity. As this National Health Survey will be used to examine the relationship between medical care utilisation (MCU) and level of obesity (for a sample of the Australian population) it is important to describe and discuss the salient features of this survey.

3.8 The 1995 National Health Survey: An Overview

The Australian Bureau of Statistics (ABS) conducted the 1995 National Health Survey (NHS) from January 1995 to January 1996. This is the second in a series of five-yearly surveys designed to collect a range of health-related information based on a sample of the Australian population (Australian Bureau of Statistics, 1996). The major benefit of these surveys is that health trends in Australia can be monitored over time. The principal aims of the 1995 NHS were to collect data on the following: (i) the health status of the Australian population, (ii) the use of health care services, and (iii) health-related aspects of lifestyle.

Taking into account sample loss, approximately 23,800 households, which represent about 1 in 310 of the non-institutionalised population in Australia, were selected in the 1995 National Health Survey. This sample design ensured that in each State or Territory each individual "had a known . . . [and] equal chance of selection" (Australian Bureau of Statistics, 1996, p. 1). Trained ABS interviewers personally interviewed each member of the selected households aged 18 years and over. While the survey was conducted over a 12 month period, selected households were interviewed only once. In addition, medical records were not required and no tests were administered to respondents.

Information collected during the interview process included details of both recent and long-term medical conditions experienced by respondents, actions individuals had taken in response to these medical conditions (such as consulting a physician), and aspects of their lifestyle that could affect or contribute to their current health status such as cigarette smoking, alcohol consumption, and exercise. Approximately half of the respondents were invited to complete the Medical Outcomes Study Short Form health status questionnaire (referred to as the SF-36). This questionnaire has been designed to assess the overall health status and well-being of an individual. In addition, approximately half of adult female respondents were invited to complete an additional survey relating to specific aspects and issues pertinent to women's health.

3.8.1 Survey Sample Design and Selection

For the 1995 National Health Survey, a base sample size "approximately one-third of 1 per cent of the population was *initially* chosen" [emphasis added] (Australian Bureau of Statistics, 1996, p. 6). This sample size would approximately be equal to 16,400 private and non-private dwellings units distributed across the States and Territories in Australia. This sample size was selected to provide (i) detailed information on each State, Territory, and Australia, (ii) relatively detailed information for capital cities, (iii) broad level estimates for regions within the more populous States (such as Victoria and New South Wales), and (iv) estimates for those characteristics which are relatively common and sub-populations which are relatively large and spread fairly evenly over a geographic area (Australian Bureau of Statistics, 1996).

To improve the statistical reliability of regional estimates, and "the disaggregation of data which could be provided at the regional level", (Australian Bureau of Statistics, 1996, p. 6) the original base sample was increased in Victoria, South Australia, the Northern Territory and the Australian Capital Territory (ATC). Specifically, in Victoria the base sample was doubled in Melbourne and increased by 50 per cent in the rest of the State. In South Australia, the base sample was doubled for the entire State. For the Northern Territory, the base sample was increased six-fold in Darwin and Alice Springs and for the ACT the base sample was increased by 75 per cent. Details of the sample selected for the 1995 National Health Survey are presented in Table 3.13.

Table 3.13: Sample selected for the 1995 NHS

State / Territory	*Private and Special Dwelling Units*	*Sampling Fraction Metropolitan*	*Sampling Fraction Ex-metropolitan*
New South Wales	4476	1/500	1/550
Victoria	7061	1/240	1/320
Queensland	3697	1/390	1/390
South Australia	4871	1/140	1/140
Western Australia	2805	1/290	1/290
Tasmania	1589	1/150	1/150
Northern Territory	1981	1/30	1/150
Australian Capital Territory	2156	1/90	n.a.
AUSTRALIA	28636	1/240	1/290

Source: Australian Bureau of Statistics (1996).

Households were chosen at random using a multi-stage area sample "which ensured that persons within each State and Territory had a known and, in the main, an equal chance of selection in the survey" (Australian Bureau of Statistics, 1997b, p. 1). Based on a multi-stage approach to sampling, each State and Territory in Australia was divided into a number of 'strata' consisting of a Local Government Area (LGA) or group of LGAs. In turn, each stratum contained a number of Census Collector's Districts (CDs) determined by the 1991 Population Census. Each CD consisted of about 250 dwellings. Consequently, by employing this sampling technique, the ABS ensured that "each dwelling within the same stratum had the same probability of selection" (Australian Bureau of Statistics, 1996, p. 7).

For each dwelling selected, information was obtained by trained ABS interviewers who interviewed each adult member in the household. The following four questionnaires were developed and used in the 1995 NHS: (i) the household form, (ii) the personal interview questionnaire, (iii) the general health and well-being form, and (iv) the women's health supplementary form (Australian Bureau of Statistics, 1996).

The 'household form' was used in the 1995 NHS sample to collect basic demographic data (such as age, sex, and birthplace) and the details of the relationship between the individuals in each household (Australian Bureau of Statistics, 1996). This information was obtained from any adult that was present in the household at the time of the survey.

The 'personal interview questionnaire' was developed and administered in the 1995 NHS to collect information from individuals on (i) health-related actions they had taken (such as the number doctor visits and hospital visits), (ii) recent and long-term illness or medical conditions, and (iii) selected lifestyle behaviours (Australian Bureau of Statistics, 1996).

Furthermore, the 'general health and well-being form' was also given to adults (aged 18 years and over) in selected households for completion prior to the administration of the main questionnaire. This form is more commonly referred to as the SF-36 (or short-form with 36 items). The SF-36 is a general survey designed to measure the health status of a population. The SF-36 scales yield a profile of eight attributes, including physical functioning, role-functioning, bodily pain, general health, vitality, social functioning, role-emotional, and mental health (Ware, 1996). The SF-36 was included in the 1995 NHS to analyse the health status of the Australian population.

Finally, the 'women's health supplementary form' was given to female respondents aged 18 years and over who were not selected in the SF-36 sample (Australian Bureau of Statistics, 1996). This questionnaire contained thirty questions that were specifically related to women's health issues.

3.8.2 Survey Response

Although 28,636 dwellings were selected this sample size was reduced to an "active sample" of 23,817 households after "sample loss in the field stage" (Australian Bureau of Statistics, 1996, p. 14). As presented in Table 3.14, 21,787 of these were either fully or partly responding, resulting in a response rate for households of 91.5 per cent.

Table 3.14: Response rate for the 1995 NHS

	Private Dwelling Households	*Special Dwelling Households*	*Metropolitan*	*Ex-metropolitan*	*Total*	
	No.	No.	No.	No.	No.	%
Active Sample	23313	504	16396	7421	23817	100
Responses						
Fully refusal	737	9	523	223	746	3.1
Fully non-contact	738	120	563	295	858	3.6
Fully non-response for other reasons	420	6	322	104	426	1.8
Fully or partly responding	21418	369	14988	6799	21787	91.5

Source: Australian Bureau of Statistics (1996)

From the fully or partly responding households in the sample, there were 57,633 individuals in the coverage of this survey. However, of this number, a further 2,356 records were deleted from the sample during the processing because "they did not form part of complete households" (Australian Bureau of Statistics, 1996, p. 15). The number of individual records for each State and Territory are presented in Table 3.15.

Table 3.15: Number of records for each State and Territory

State / Territory	*Number of Records*
New South Wales	8,268
Victoria	13,964
Queensland	6,671
South Australia	8,906
Western Australia	5,203
Tasmania	2,658
Northern Territory	3,457
Australian Capital Territory	4,624
AUSTRALIA	53,751

Source: Australian Bureau of Statistics (1996)

However, it should be noted that the figures presented in Table 3.15 were presented in an ABS report issued on December 19, 1995. At the time the report was issue the ABS indicated that at ". . . the time of writing, the possibility of re-instating some records from incomplete households was under investigation" (Australian Bureau of Statistics, 1996, p. 15).

In a subsequent ABS publication released on December 23, 1997, an additional 77 individual records were re-instated (Australian Bureau of Statistics, 1997b). The revised number of records for each State and Territory are presented in Table 3.16. In comparing Tables 3.15 and 3.16 it can be seen that (i) only one record was removed from the Tasmanian sample, (ii) 71 records were re-instated in the Western Australian sample, and (iii) 7 records were re-instated in the Northern Territory sample. In sum, a total of 77 records were re-instated, increasing the total number of individual records to 53,828.

Table 3.16: Number of records for each State and Territory (revised)

State / Territory	*Number of Records*
New South Wales	8,268
Victoria	13,964
Queensland	6,671
South Australia	8,906
Western Australia	5,274
Tasmania	2,657
Northern Territory	3,464
Australian Capital Territory	4,624
AUSTRALIA	53,828

Source: Australian Bureau of Statistics (1997b)

In 1997, the revised individual records were released by the Australian Bureau of Statistics as a "Confidentialised Unit Record File" (Australian Bureau of Statistics, 1997b, p. 1) and these individual-level (or micro) data are now available (Australian Bureau of Statistics, 1997c).

3.8.3 Survey Contents

The 1995 NHS contains data on the following topics: (i) health status; (ii) health-related actions; (iii) health risk factors; (iv) women's health supplementary items; and (v) sample characteristics.

Health Status

The World Health Organisation (WHO) defines health as "a state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity" (WHO, 1958, p. 459). Surveys such as the 1995 NHS attempt to collect data on the health status of the Australian population. In attempting to describe the health status of the Australian population, the 1995 NHS focused on the following measures of ill-health: (i) the number and type of medical conditions (such as illness or injury) recently

experienced by respondents; and (ii) the number and type of long-term medical conditions (such as asthma or type 2 diabetes mellitus) experienced by respondents (Australian Bureau of Statistics, 1996).

Recent and long-term medical conditions were also reported in the 1995 NHS and are presented as a single code. The classification system adopted in the 1995 NHS is based on the International Classification of Diseases, 9th Revision (ICD9). However, many of the ICD9 codes were "collapsed into broader groupings of conditions which reflect the type and level of information provided in a household survey situation and which were expected to yield sufficient observations in the survey to provide useable estimates" (Australian Bureau of Statistics, 1996, p. 117).

The 1995 NHS also collected information on self-assessed health status, and included the SF-36 form which, using scores derived from 36 survey items, provides indicators for eight attributes of health and well-being (Australian Bureau of Statistics, 1996). With respect to self-assessed health status, respondents were asked directly to rate their general health from excellent to poor (Australian Bureau of Statistics, 1996). The purpose of administering the SF-36 form was to collect data that could be used as (i) a means of monitoring the health of the general population, (ii) a method of estimating the burden of different health outcomes, (iii) a tool to assist in evaluating the effectiveness of alternative treatment options, and (iv) a tool for monitoring outcomes in a clinical environment (Australian Bureau of Statistics, 1996). This is the first time that the ABS has administered the SF-36 in association with the National Health Survey.

Health-Related Actions

The 1995 NHS also contains information on the health-related actions taken by individuals during the survey period. In particular, a broad range of health-related actions were cover in this survey including: (i) hospital episodes, (ii) doctor consultations, (iii) dental consultations, (iv) consultations with other health professionals, (v) consultations with other persons and/or organisations, (vi) days away from school or work, (vii) other days or restricted activity, and (viii) the use of medications (including vitamins and natural preparations) [Australian Bureau of Statistics, 1996]. Therefore, the 1995 NHS contains a wealth of information on health-related actions. More specifically, these health-related action data can be used to examine the association between medical care utilisation (MCU) and level of obesity. In fact, it is probably reasonable to argue that 'hospital episodes' and 'consultations with doctors' are the driving factors behind medical care utilisation. As these health-related actions are of central importance they will be discussed in greater detail.

In the 1995 NHS, a hospital episode is defined as either (i) admissions to a hospital as an in-patient, and (ii) the use of the emergency and outpatient services at a hospital (Australian Bureau of Statistics, 1996). The second part of this definition excludes admissions and consultations at dental hospitals (which may be attached to a hospital as part of their outpatient setting) (Australian Bureau of Statistics, 1996). Furthermore, for the purpose of this survey, a hospital is defined "as an institution which offers residential health care, other than a nursing or convalescent home" (Australian Bureau of Statistics, 1996, p. 38).

According to the ABS, hospital episode data collected by the 1995 NHS can assist in the analysis of usage patterns (i) across different groups within the community, and (ii) of emergency, outpatient, and day clinic services relative to other health-related actions that individuals may undertake in response to injury or illness (Australian Bureau of Statistics, 1996).

In the 1995 NHS, 'doctor consultations' refer to any occasions in which the survey respondent discussed his/her health with, and/or received treatment from, a doctor (Australian Bureau of Statistics, 1996). Included in this definition are face-to-face consultations, telephone consultations, and another individual consulting on behalf of the survey respondent. The term 'doctor' refers not only to general practitioners but specialists such as surgeons and psychiatrists (Australian Bureau of Statistics, 1996).

According to the ABS, doctor consultations collected in the 1995 NHS can assist in (i) detailing the level (or number) of doctor consultations relative to other health-related actions undertaken by the individual, (ii) identifying medical conditions for doctor consultations, (iii) outlining the patterns of doctor consultations for specific groups in the community, and (iv) indicating the association between doctor consultations lifestyle factors such as smoking and exercise (Australian Bureau of Statistics, 1996).

Health Risk Factors

The 1995 NHS also collected information in relation to health risk factors. This is because a number of factors are associated with an increased risk of ill-health or injury (Australian Bureau of Statistics, 1996). In particular, the 1995 NHS focused on the following areas presented in Table 3.17 below.

Table 3.17: Health risk factor: Area of focus

Area of Focus	*Associated Health Risk Factor*
Cardiovascular	Smoking, physical activity, diet/nutrition, high blood cholesterol, overweight/obesity, high blood pressure
Cancer	Sun protection, smoking, diet/nutrition, alcohol, obesity
Injury	Alcohol misuse, quality of medicines, mental health, safety behaviours, physical activity
Mental Health	Diet/nutrition, quality of medicines, alcohol misuse, illicit drug use

Source: Australian Bureau of Statistics (1997b)

As shown in Table 3.17, the four areas of focus include (i) cardiovascular disease, (ii) cancer, (iii) injury, and (iv) mental health. Each of these areas is also associated with a variety of health risk factors ranging from smoking to overweight/obesity. According to the ABS, the decision to focus on these health risk factors was a result of consultation with health professionals, administrators, and policy makers. Based on these consultations it was these health risk factors that were considered to be "major issues of concern" (Australian Bureau of Statistics, 1996, p. 59).

The principal advantage of the 1995 NHS is that these health risk factor data were collected "side by side with indicators of health status and the usage of health services and other health related actions" (Australian Bureau of Statistics, 1996, p. 60). More importantly, this means that data on health risk factors can be considered (i) independently, (ii) in relation to other health risk factors, (iii) in relation to medical conditions, and (iv) in relation to health-associated actions (Australian Bureau of Statistics, 1996). However, it is important to note that while data from the 1995 NHS may indicate a statistical association between, for example, a particular risk factor and a

certain medical condition, this statistical association should not be interpreted as a causal relationship (Australian Bureau of Statistics, 1996).

Of these health risk factors, the measurement of height, weight, and the classification of body mass index (BMI) is of particular interest. The 1995 NHS calculated the BMI based on self-reported height and weight. According to the ABS, the analysis of body mass can assist in (i) identifying the prevalence of being either overweight or obese, (ii) enable BMI to be analysed in association with health status indicators, health service utilisation, and health risk factors (Australian Bureau of Statistics, 1996). Furthermore, the BMI classification system used in the 1995 NHS is consistent with the World Health Organisation (WHO) endorsed classification system for overweight/obesity.

Women's Health Supplementary Items

The 1995 NHS also contained a supplementary women's health form. As indicated previously, this questionnaire consisted of 30 questions specifically related to women's health issues. Information was collected on the following topics: (i) screening for breast and cervical cancer, (ii) breastfeeding of infants, (iii) use of hormone replacement therapy, and (iv) hysterectomy (ABS, 1996). The collection of this type of information can be used to assist in the monitoring of women's health and formulation of health policy directed at women.

Survey Characteristics

The 1995 NHS also contains a range of information pertaining to both the demographic and socioeconomic characteristics of the survey sample. These characteristics can be linked with other health-related data collected in the survey. The characteristics

recorded in the 1995 NHS include: (i) demographics (such as age and marital status); (ii) education; (iii) employment; (iv) income; (v) health insurance cover; and (vi) housing (Australian Bureau of Statistics, 1996).

Assessment of the 1995 National Health Survey

As previously indicated the 1995 NHS is a personal interview household survey that provides a wealth of health-related information for a sample of the Australian population. Therefore, it would be useful to review the advantages and limitations of using this survey.

To begin with, the NHS uses a complex multistage design, with over-sampling for some sub-populations, especially minority populations. Consequently, sampling weights have been developed to account for survey design and to provide an adjustment for survey non-response. In other words, the NHS, is essentially using a cluster sampling framework to collect data. There are two important reasons why cluster sampling is widely used in practice, especially in surveys that cover a large geographical area: feasibility and cost (Fowler, 1993; Lohr, 1999).

Cluster sampling may be the only feasible method of sampling because the only sampling frames readily available for the target population are a list of clusters. This is true in surveys where the 'households' serve as the listing unit. Moreover, cluster sampling may cost less than other survey designs. For example, if a cluster is a geographic unit such as a census tract, then once households are selected within the tract, the cost of travelling (with the tract) to each household is relatively low. This

usually why cluster sampling is less expensive than a random sample survey design (Fowler, 1993).

The advantage, from a research perspective, is that interviewer-household surveys, such as the NHS, in general collect a wide range of self-reported data that are available on CD. In addition, these data can be extracted and analysed using a number of statistical packages. However, there is one primary disadvantage of cluster sampling – high standard errors. For instance, households on the same block will often be similar in terms of race, income, and a range of other characteristics. Therefore, because of this homogeneity among listing units, high standard errors may result. To account for sampling design, the NHS contains 'weights' that take "account of the person's probability of selection in the sample from their region, with adjustment to account for undernumeration at the age, sex and metropolitan rest of State level" (Australian Bureau of Statistics, 1997c, p. 6).

These 'weights' can be used in regression analysis; however, there is considerable debate as to the appropriate use of 'weights' regression analysis (see Korn & Graubard, 1991; Korn & Graubard, 1991; Lohr, 1999). Furthermore, the use of 'weights' in the field of econometrics is not standard practice and specialised statistical programs are required to estimated regressions using 'weights' (Lohr, 1999). As a result, a decision was made not to use 'weights' in the regression analysis. However, for the subsequent policy analysis 'weights' were used to generate cost estimates for segments of the Australian *population*.

3.9 Summary

This chapter provided an overview of the empirical literature on overweight/obesity in Australia. Two key points are worth noting (i) the Australian population, on average, is becoming more obese; and (ii) the rate of being overweight or obese among the Australian population is influenced by a variety of factors ranging from racial background to socioeconomic status.

Furthermore, the 1995 National Health Survey was also addressed in this chapter. This survey, which was conducted by the Australian Bureau of Statistics, contains the most recent data on the health of the Australian population. Furthermore, contained within this survey are the most recent estimates of the rate of overweight/obesity. This dataset will be used to examine the relationship between medical care utilisation and the level of BMI for a sample of the Australian population. The next chapter provides a detailed discussion of the methods used to examine and estimate these relationships.