Type 2 Diabetes Costs in Australia -

the potential impact of changes in diet, physical activity and levels of obesity

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Abbreviations

ABS	Australian Bureau of Statistics
AFNMU	Australian Food and Nutrition Monitoring Unit
AIHW	Australian Institute of Health and Welfare
ANZFA	Australian New Zealand Food Authority
AusDiab	Australian Diabetes, Obesity and Lifestyle Study
BMI	Body mass index
BOD	Burden of disease
CDHAC	Commonwealth Department of Health and Aged Care
CHPE	Centre for Health Program Evaluation
CDHHCS	Commonwealth Department of Health, Housing and Community Services
COI	Cost of illness
DCIS	Disease Costs and Impact Study
DHSF	Department of Health & Family Services
IGT	Impaired glucose tolerance
LCD	Low calorie diet
NHMRC	National Health and Medical Research Council
NHPA	National Health Priority Areas
NHS	National Health Survey, 1995 (Australia)
NNS	National Nutrition Survey, 1995 (Australia)
NZMPH	New Zealand Ministry of Health
OATSIH	Office of Aboriginal & Torres Strait Islander Health
PAF	Population attributable fraction
RR	Relative risk
UQ	University of Queensland
SIGNAL	Strategic Inter-governmental Nutrition Alliance
VLCD	Very low calorie diet
WHO	World Health Organisation

Executive Summary

Close to one million (7.5%) Australian adults have diabetes mellitus and it is estimated that by the year 2010 this will increase to 1.23 million. Diabetes mellitus accounts for 5% of the total burden of disease and is the seventh leading cause of Australian deaths. Type 2 diabetes is the most common form representing 85-90% of those with diabetes mellitus. This report provides estimates for the direct health care costs of Type 2 diabetes attributable to obesity, and the potential impact on costs of diet and physical activity interventions.

There is a growing body of evidence from intervention studies suggesting that lifestyle interventions (diet and/or physical activity) can prevent or delay the onset of Type 2 diabetes in those with impaired glucose tolerance (a strong predictor of Type 2 diabetes). Epidemiological evidence suggests a role of diet in the aetiology of Type 2 diabetes. However, there is not yet consensus regarding the specific dietary factors involved or the magnitude of their impact.

Obesity is an important risk factor for Type 2 diabetes, with increasing BMI associated with greater risk of Type 2 diabetes. The relative risk (RR) of Type 2 diabetes has been estimated to be 1.8 for overweight adults (BMI 25–<30kg/m^2) and 3.2 for obese individuals (BMI 30kg+/m^2). Data from the 1995 National Nutrition Survey showed that approximately 55% of Australian adults were overweight or obese. Based on these figures, it was estimated here that 41% of Type 2 diabetes in Australia is attributable to overweight and obesity (17% for overweight; 24% for obesity).

- The total health care cost of Type 2 diabetes in 1993-94 in Australia was $216.7 million of which $89.1 million (41%) can be attributed to overweight and obesity. The attributed costs are substantially greater if the costs of complications resulting from diabetes are also included (an additional $120.5 million). These will be about 10% greater in terms of current costs.

While the aetiology of obesity is complex and multi-factorial in nature, a review of the literature showed that there is strong evidence that diet and physical activity are important determinants of obesity. The studies considered suggest that a conservative estimate of weight loss in the order of 1 to 5kg can be achieved and maintained for at least one year by lifestyle interventions. Maintenance of long-term weight loss has been achieved by those consuming diets reduced in fat and energy along with a regular physical activity program.

- Interventions that result in a weight loss of 5kg in all Australians who are overweight or obese would reduce the prevalence of overweight and obesity from 55% to ~ 40%. This could reduce the health care costs associated with Type 2 diabetes by $18.6 million per year, or up to $45.1 million per year if the cost of complications is also considered.

- Recent data from the AusDiab Study suggest the prevalence of overweight and obesity is increasing, and consequently that Type 2 diabetes and its associated costs will continue to increase at least in the near future. Therefore, there would be a cost saving for the health care system by simply arresting the increase in prevalence of overweight and obesity.

These estimates do not factor in the cost of implementing the programs to achieve the weight changes, and the cost estimates change according to the assumptions made (eg it is not expected that all cost categories will be equally affected by a reduction in overweight and obesity as assumed in the calculations). However, regardless of the assumptions it was clear that dietary and physical activity habits of the population and the associated obesity contribute substantially to the direct health system costs from Type 2 diabetes. While this does not include indirect costs and intangible costs they do provide a useful indicator of the financial burden attributable to overweight and obesity and the potential impact of lifestyle changes.

1 Introduction

Diabetes mellitus is a chronic disease characterised by insulin insufficiency or resistance to its action, resulting in poor glycaemic control. The recent Australian Diabetes, Obesity and Lifestyle Study (AusDiab) estimated that close to one million (7.5%) Australian adults have diabetes mellitus (Dunstan et al 2001). Type 2 diabetes is the most common form comprising 85 to 90% of those with diabetes mellitus. The most recent cost estimates available suggest approximately $216.7 million in health system costs were attributable to Type 2 diabetes in 1993-94 (Mathers & Penm 1998). In a study of the burden of disease in Australia, diabetes mellitus was estimated to account for 5% of the total burden of disease and was the seventh leading cause of Australian deaths (Mathers, Vos & Stevenson 1999). In addition, complications of diabetes such as heart disease, stroke, blindness, kidney problems, and lower limb amputations, contribute significantly to the overall morbidity and mortality in Australia.

The increasing burden of diabetes in Australia has been recognised by its inclusion as one of the National Health Priority Areas (AIHW & DHFS 1997). In 1999 the Australian Health Ministers' Conference endorsed the National Diabetes Strategy 2000-2004 and identified primary prevention strategies and prevention and reduction of complications as key priority areas.

Researchers have postulated a role for diet in the development of diabetes for some time. A growing body of evidence from intervention studies suggests dietary intervention may prevent or delay the onset of Type 2 diabetes in those with impaired glucose tolerance (IGT), which is a strong predictor of Type 2 diabetes. Further, evidence from observational studies suggests that specific dietary factors may be associated with Type 2 diabetes. However, details of the specific dietary factors and pathophysiological mechanisms by which these affect Type 2 diabetes remain uncertain (see section 1.2). The current scientific consensus suggests that diet may have its greatest effect on Type 2 diabetes via its influence on levels of obesity, in conjunction with the impact of physical activity habits (Report of the NHMRC expert panel on prevention of obesity and overweight, 1997). With the current trends to increasing prevalence and risks associated with overweight and obesity, the indirect effects of diet and physical activity on risk of Type 2 diabetes appear to be substantial.

This report was undertaken as part of a larger study considering the impact of dietary factors on Australia's health problems and health system costs (see Marks et al 2001). Because the effect of diet on Type 2 diabetes is mainly indirect, through its effect on obesity, the report focuses on obesity and interventions to reduce obesity.

Thus the aim of this study was to estimate the direct health care costs of Type 2 diabetes attributable to overweight and obesity and the potential impact on these costs of dietary and physical activity interventions that result in weight loss amongst Australians who are overweight or obese.

1.1 National Food and Nutrition Monitoring and Surveillance Project

This cost of illness report is part of a program of work being funded by the Commonwealth Department of Health and Aged Care that underpins development of a national food and nutrition monitoring system for Australia. This work aims to establish a basis for routinely providing the diet-related evidence needed to guide policy and planning decisions.

Establishment of a food and nutrition monitoring system for Australia is a priority of the Australian *Food and nutrition policy* (CDHHCS 1992). It is also a listed initiative of *Eat Well Australia* and the *National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan* (SIGNAL 2000).

Developmental activity has occurred in two stages. The first stage of development was undertaken by the AIHW in the early 1990's. This resulted in a number of outcomes, including the publication *Plan for a national food and nutrition monitoring system* (Coles-Rutishauser & Lester 1995). This has provided the framework and strategy for subsequent developments. The second stage of development commenced in December 1998 when the Australian Food and Nutrition Monitoring Unit, a consortium led by the University of Queensland, was contracted to undertake an integrated set of projects that implemented key elements of the above *Plan*. This includes the project leading to the current report.

1.2 Diet and Type 2 diabetes

It has long been believed that diet plays a part in the development of Type 2 diabetes. Ecologic and migrant studies suggest a role for environmental factors including diet in the aetiology of Type 2 diabetes (Mann 1980, Feskins 1992). Studies in animal models and humans have demonstrated significant effects on glucose tolerance and insulin sensitivity in response to dietary manipulation of the proportion of fats and carbohydrate consumed (Feskens 1992, Feskens and van Dam 1999).

Relatively few epidemiological studies have investigated the relationship between diet and risk of Type 2 diabetes. A small number of studies have reported increased risk of Type 2 diabetes associated with total and saturated fat intake (Feskens et al 1995, Marshall et al 1994). However, other studies report no significant association with total or saturated fat (Lundgren et al 1989, Feskens & Kromhout et al 1989). Other studies have reported associations between glycaemic index, cereal fibre, vegetable fat, polyunsaturated fat, *trans* fatty acids and vegetables (Colditz et al 1992, Marshall et al 1994, Feskins et al 1995, Salmeron et al 1997, Williams et al 1999, Salmeron et al 2001). Overall, there is an insufficient body of evidence to conclude that specific dietary factors influence the risk of developing Type 2 diabetes, and the physiological mechanisms have yet to be adequately described.

1.2.1 Lifestyle intervention trials

A small number of studies have investigated the effect of lifestyle interventions (diet and /or physical activity) for the prevention of Type 2 diabetes (table 1). These studies have focused on subjects with impaired glucose tolerance (IGT), who are at higher risk of developing Type 2 diabetes. The annual progression rates from IGT to Type 2 diabetes range from 4 to 9% (Edelstein et al 1997). Two studies conducted in Sweden reported reduced incidence of Type 2 diabetes in those treated with diet (Sartor et al 1980) and diet plus exercise (Eriksson and Lindgarde 1991) compared to control groups. The dietary interventions were aimed at reducing intake of dietary fat and simple carbohydrate intakes. In both studies, about 30% of the control group developed diabetes as opposed to just over 10% in the intervention groups.

More recently, larger intervention trials of diet and exercise in people with IGT have been conducted in China, New Zealand and Finland. The Chinese Da Qing study reported a 31% and 42% reduction in risk of developing Type 2 diabetes with diet and diet plus exercise interventions respectively after six years of follow up (Pan et al 1997). In New Zealand, a one year randomised controlled trial of reduced fat ad libitum diet versus usual diet reported a lower conversion from IGT to Type 2 diabetes

(47% vs 67% $p<0.05$). However, after a five year follow-up, no differences were found between the two groups (Swinburn, Metcalf & Ley 2001). The Finnish Diabetes Prevention study reported 23% of the control group progressed to diabetes compared to 11% of those receiving individualised lifestyle counselling (reducing weight, total intake of fat and saturated fat, and increasing fibre and physical activity) (Tuomilehto et al 2001). One large study in the USA investigating the effects of lifestyle intervention (intensive diet and exercise program), metformin and placebo in subjects with IGT is currently underway (The Diabetes Prevention Program Research Group, 2000).

Table 1: Studies of lifestyle interventions (diet with or without physical activity) for the prevention of Type 2 diabetes

Study	Country	Study population	Follow up up	Intervention	Progression to Type 2 diabetes (%)	
					Intervention	Control
Sartor et al 1980	Malmohus, Sweden	267 men with IGT	10 years OR diet only	Diet + placebo	13	29
Eriksson and Lindgarde 1991	Malmo, Sweden	217 men with IGT	6 years	Diet + Exercise	11	29
Pan et al 1997	Da Qing, China	577 men and women with IGT	6 years	Diet	44	68
Swinburn, Metcalf & Ley, 2001	New Zealand	176 men and women	1 year	Reduced Fat ad-libitum Diet	47	67
Tuomilehto et al 2001	Finland	523 overweight men and women with IGT	3.2 years	Diet + Exercise	11	23

These studies provide support for an effect from lifestyle interventions in the prevention or delay of Type 2 diabetes. In addition, modification of diet and physical activity may improve glycemic control in those with established diabetes, thereby reducing the risk of diabetes-related complications (Bourn et al 1994, Pi-Sunyer 1996). Whether lifestyle interventions are effective in delaying or preventing Type 2 diabetes in the long term is unclear until longer term follow up data become available. The National Health Priority Areas report on diabetes mellitus identifies even a short term delay in onset of diabetes as a substantial health gain.

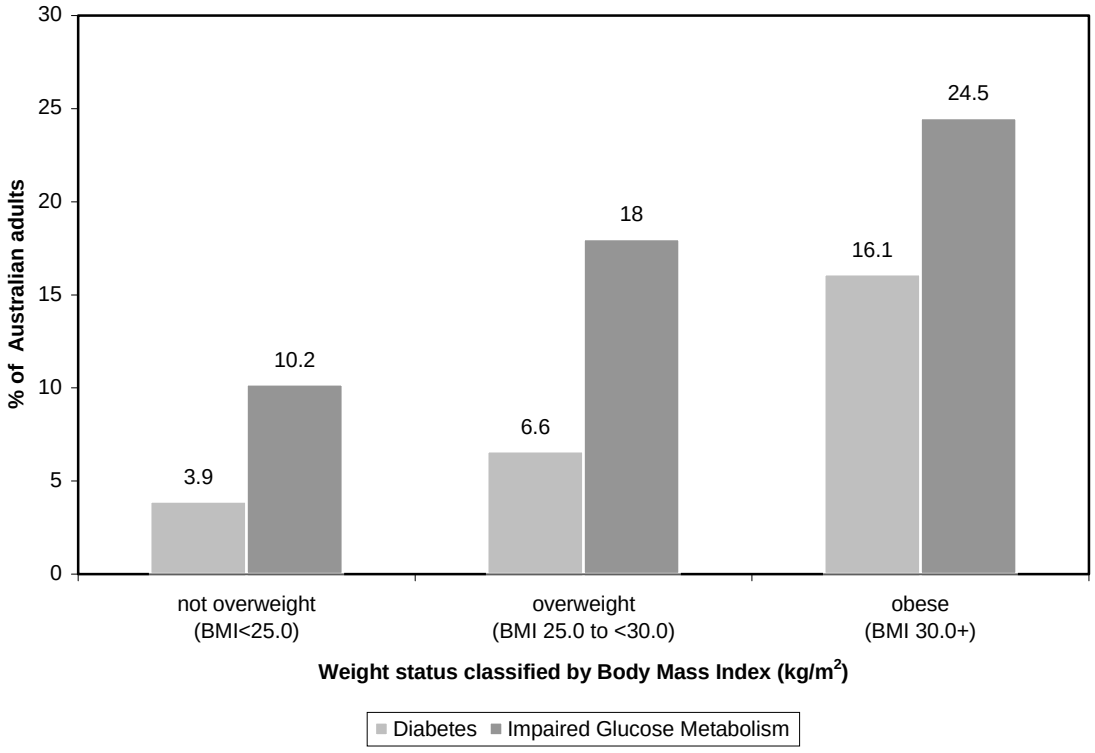
However, it is difficult from these studies to separate the effects of either diet or physical activity, and the direct versus indirect effects through the impact on levels of overweight and obesity. While for many purposes this would not be a limitation, it is necessary for the purposes of this study to be able to clearly specify the type of 'exposure' and size of effect so that the attributable costs can be calculated. For this reason, the next section focuses on the association between obesity and Type 2 diabetes, and the size of the effect of specific dietary and lifestyle interventions on obesity.

1.3 Obesity and Type 2 diabetes

Obesity is a major risk factor for Type 2 diabetes with increasing BMI associated with greater diabetes risk (Colditz et al 1990). This pattern is supported by the results of the AusDiab study which estimates that the prevalence of Type 2 diabetes increases from 3.9% in those not overweight (BMI <25) to 16% among those who are obese (BMI 30+) (Dunstan et al 2001) (figure1). In addition, obesity is an important risk factor for conditions such as hypertension, cardiovascular disease and hyperlipidaemia.

Current strategies and recommendations to reduce levels of overweight and obesity in the population focus on dietary change and physical activity habits (Report of the NHMRC expert panel on prevention of obesity and overweight, 1997).

Figure 1: Proportions of the Australian adult population with impaired glucose metabolism and diabetes mellitus by weight status: 2000 AusDiab report



Source: Dunstan et al 2001

1.3.1 The effect of specific dietary interventions on obesity

There is strong evidence to suggest that diet is an important determinant of obesity and diet plays a major role in the prevention and treatment of overweight and obesity. Dietary fat and total energy intakes have been the main foci of research in this area. Recent interest has surrounded the role of other dietary factors such as fibre, glycaemic index and dietary variety as determinants of overeating, satiety and weight regulation (McCrory et al 2000). However, the relative importance of these factors has yet to be determined.

Dietary studies show marked variations in weight loss ranging from a small weight gain or no change to weight losses of 10kg or more, even in studies with similar interventions. Purnell, Knopp & Brunzell (1999) suggest the variation in weight loss in response to dietary interventions may be explained by the notion of *"dietary responders"*. Genetic factors are suggested to account for 25-40% of the risk of obesity (Report of the NHMRC expert panel on prevention of obesity and overweight, 1997). Dietary factors are only a part of a possible range of environmental influences that affect the genetic expression of obesity. Therefore, it is not unexpected that dietary intervention *"results in a variable amount of weight loss depending on one's genetic background"* (Purnell, Knopp & Brunzell 1999). In addition, small numbers of subjects, lack of a control group, high dropout rates and relative short study duration have limited most dietary intervention studies conducted so far.

Recently, there have been several systematic reviews of dietary fat and obesity interventions. Hill, Melanson &Wyatt (2000) reviewed randomised clinical trials comparing low fat diets fed ad libitum to usual/control diets. The difference in weight between intervention and control groups ranged from no change to -7.5kg. Fat intake was 2-18% less than baseline in the intervention groups. Study duration ranged from three months to two years, with the majority of studies lasting more than one year. In general, greater weight loss was seen in studies of overweight subjects. However, no studies in obese subjects met the inclusion criteria. The greatest effect was seen in a study by Pritchard, Nowson & Wark (1996) of overweight men (mean BMI 29) who reduced their fat intake to ~20%, resulting in a mean weight loss of 6.3kg over 1 year compared to controls who gained 1.2kg.

Bray and Popkin (1998) reviewed 28 studies of low fat diets without energy restriction. A reduction of 10% of energy from fat was associated with a weight loss of 16g/day. However, selected studies ranged from three weeks to two years in duration and some studies did not include a control group.

A recent meta-analysis of low fat ad libitum dietary intervention studies found a reduction in dietary energy from fat was associated with a weight loss of 3.2kg (95%CI 1.9-4.5kg) in the intervention compared to the control group (Astrup et al 2000). Included studies ranged from two to 12 months in duration and the difference in weight loss between intervention and control groups ranged from +0.7 to -11.1kg. In addition, a greater pre-treatment body weight was associated with a greater amount of weight loss. Willet (1998) reviewed studies of low fat ad libitum dietary intervention studies lasting longer than one year. Changes in weight ranged from -0.8kg (National Diet-Heart Study Research Group, 1968) to -2.6kg (Kasim et al 1993) with energy from dietary fat reduced to 30% and 17% respectively (baseline 35% of energy from fat).

Some investigators have questioned the role of dietary fat in weight gain given the limitations of these studies and the increasing prevalence of obesity in the USA and other countries despite declining intakes of dietary fat, (Katan, Grundy & Willett 1997, Willett 1998a&b). Hill, Melanson & Wyatt (2000) have suggested that …*"the greatest potential effect of low fat diets may not be in producing weight loss but in preventing weight gain"*.

However, these studies suggest a conservative estimate of weight loss in the order of 1 to 5kg can be achieved and maintained for at least 1 year by dietary intervention alone and that weight loss is greater in those who are more overweight and obese. A weight loss of 5kg or more is possible in short term studies but weight re-gain is common. Weight losses of 1 to 5kg are used in the present study to estimate the potential impact of interventions on overweight and obesity in the Australian population.

1.3.2 The combined effect of dietary and physical activity changes on overweight/obesity

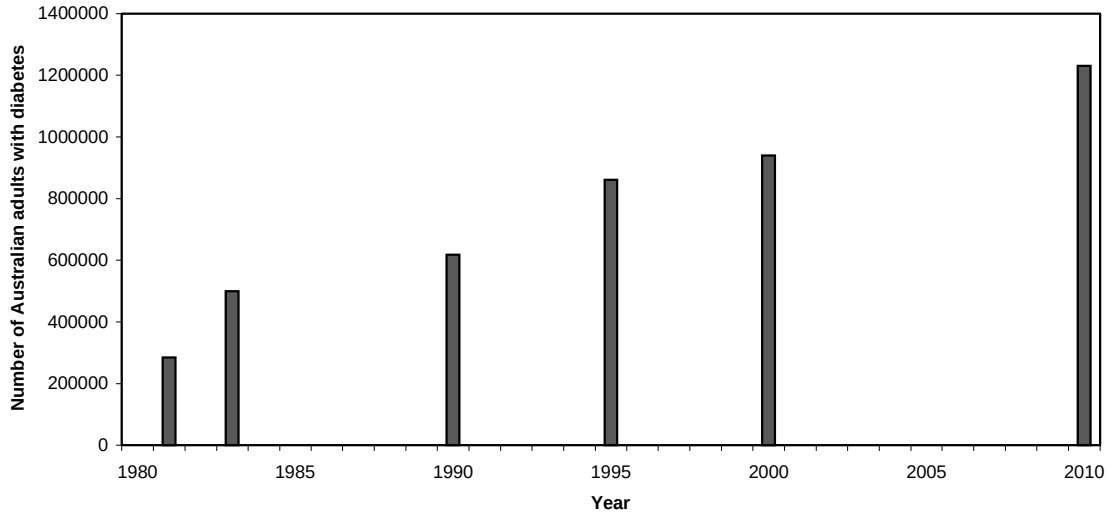
It is important to recognise that the aetiology of obesity is multi-factorial in nature and that diet is only one part of a complex interaction between genetic, behavioural and environmental determinants. As such, it is necessary to address diet as part of overall lifestyle modification. Recent evidence suggests dietary interventions aimed at reducing caloric intake and increasing physical activity in combination result in greater, more prolonged weight loss, than either diet or exercise alone (Glenny et al 1997, Yu-Poth et al 1999). Data from the National Weight Control Registry of persons who have successfully achieved long term loss (13.6+kg for 1+ year) in the USA indicates that of those who have maintained weight loss, 89% consume a diet restricted in fat and energy and participate in regular physical activity compared to 10% by diet alone and 1% by physical activity alone (Klem et al 1997). This is in agreement with current Australian dietary guidelines that state *"maintain a healthy body weight by balancing food intake and regular physical activity"* (NHMRC 1992). Therefore, broad lifestyle modification rather than simply dietary or physical activity changes is considered necessary for the prevention and treatment of overweight and obesity and Type 2 diabetes.

1.4 Prevalence of diabetes

The recent AusDiab report indicates about 940,000 (7.5%) Australian adults (25 years or older) have diabetes mellitus (Dunstan et al 2001). Of these, 85-90% have Type 2 diabetes. In addition, a further 16.3% of Australians have impaired glucose tolerance or impaired fasting glycemia (see appendix: WHO classifications of glucose tolerance status).

Since 1981, the number of Australians with diabetes has increased threefold (figure 2). It is estimated that 1.23 million Australians will have diabetes by the year 2010 (Dunstan et al 2001). This projection was revised from the previous estimate of 950,000 persons, which is close to the current prevalence reported by the AusDiab study.

Figure 2: Estimates and projections of diabetes prevalence in Australia, 1981-2010

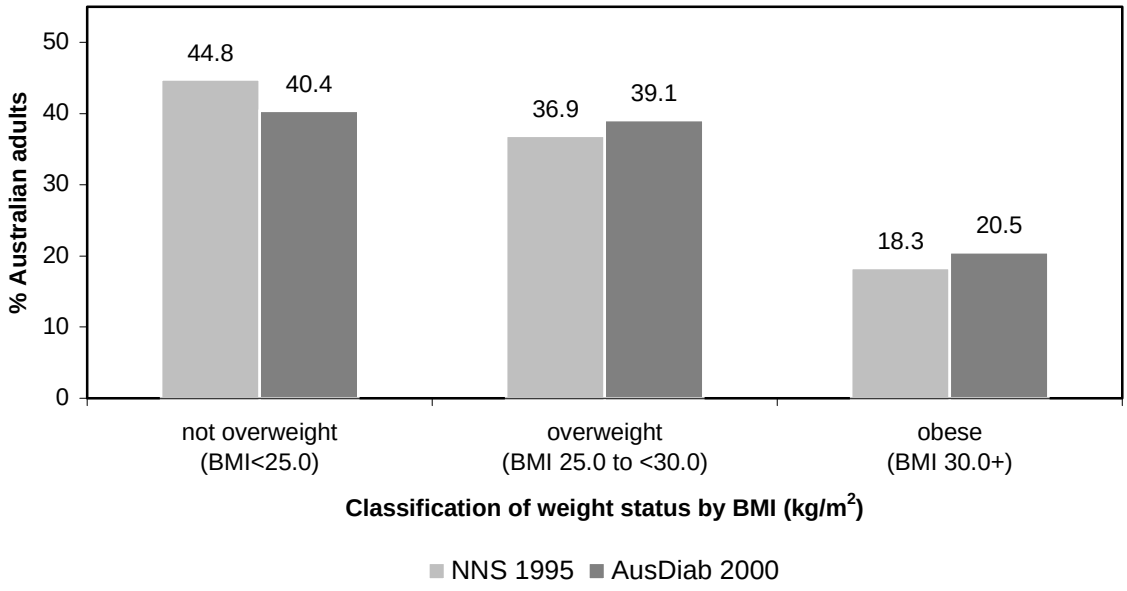


Sources: Busselton Survey 1981 (Glatthaar et al 1985), National Heart Foundation Risk Factor Prevalence Study No 2 - 1983 (NHF 1983), National Health Survey 1989-90 (ABS 1991), National Health Survey 1995 (ABS 1997a), AusDiab Study 2000 (Dunstan et al 2001), projection 2010 (Dunstan et al 2001)

1.5 Prevalence of overweight and obesity

The most recent estimates of the prevalence of overweight and obesity in Australia suggest 59.6% of Australians are overweight or obese (39.1% and 20.5% respectively) (Dunstan et al 2001) (figure 3). These estimates indicate an increase in the prevalence of overweight and obesity since the 1995 NNS, which reported 55.2% of Australians as overweight or obese (figure 3) (ABS 1997). In this study, the prevalence estimates of overweight and obesity from the 1995 NNS were used. These estimates were considered to be more representative of the population and the prevalence of overweight and obesity at the time of the health system cost data reported by the AIHW in 1993-94 (Mathers & Penm 1998). The criteria for defining overweight and obesity used in the 1995 NNS are presented in the appendices (table A2).

Figure 3: Distribution of the Australian adult population by weight status from 1995 National Nutrition Survey and 2000 AusDiab report



Source: 1995 National Nutrition Survey (ABS 1997b), AusDiab Report (Dunstan et al 2001)

1.6 Cost of illness studies

Cost of illness (COI) studies estimate the economic consequences of poor health. They generally distinguish between direct and indirect costs.

> *Economists make a distinction in disease cost studies between the direct costs of providing health care services and the indirect costs, which focus on lost production due to sickness and premature death but can include as well costs impacting outside the health care sector (such as police and courts costs associated with drug abuse, for example). Direct costs thus include all those expenditures on diagnosing, treating and caring for the sick. Indirect costs and intangibles (such as pain and suffering) are not included in the cost estimates … as their meaning is often imprecise and the methodologies for their measurement at the population level are either contentious and/or at an early stage of development.* (Mathers et al 1998)

In the last decade there have been significant developments in methods for cost of illness studies in Australia. The 'first wave' of studies in the early 1990's was associated with the work of the AIHW for the Macro Economic Evaluation Model and included the first work on COI for diet-related disease in Australia by Crowley and coworkers (1992). This work was continued by AIHW as the Disease Costs and Impact Study (DCIS). The DCIS has focused on estimation of direct costs, noting that calculation and inclusion of indirect costs in COI studies remains an area of debate and controversy (Mathers et al 1998). Intangible costs are omitted for similar reasons.

As part of the DCIS, the basic methodology for estimating and attributing direct costs was substantially revised in the period 1994-96. The AIHW has published direct cost estimates based on disaggregation of the national health expenditure for 1993-4 into the major disease categories of the International Classification of Diseases (Version 9). The study was able to allocate 90% of the national health expenditure into these categories[1] (Mathers et al 1998). In 1998 the AIHW began publishing a series of reports describing the health system costs for different disease categories. These are the most recent direct cost estimates available for Australia and the report describing the health system costs of diabetes mellitus in Australia for 1993-94 was used as the source of costs data in the current study (Mathers et al 1998).

The approach used for estimating the costs attributable to particular risk factors typically follows a series of steps that involve estimating the proportion of the disease attributable to the risk factor and then using this to apportion the share of the total costs accordingly (Crowley et al 1992, Stephenson et al 2000). This study firstly estimated the proportion of Type 2 diabetes and health care system costs attributable to overweight and obesity. It then considered the potential impact of dietary interventions to prevent or reduce levels of overweight and obesity in the population and the potential impact of this on health care costs, but not including the costs of implementing these interventions. The methods are considered further in chapter 2.

1.7 Use and interpretation of direct cost of illness information

The AIHW reports describing the health system costs recommend caution in use and interpretation of this information (Mathers et al 1998). They note that:

- existing health expenditure on a disease does not of itself give an indication of the loss of health due to that disease, or the priority for intervention or need for additional health services expenditure;

- care should be taken in interpreting direct costs associated with disease treatment as an estimate of the savings that would result from prevention of the disease. The conversion of the opportunity cost of resources being devoted to disease treatment, or benefits forgone, into expenditure savings involves a number of additional considerations; and

1The total cost of each disease is divided into its major components, namely hospital, medical, pharmaceutical, allied health, research, public health and other. Expenditure not able to be disaggregated included community health services, ambulance services, capital works and public health programs apart from national cancer screening programs (breast and cervix) and lung and skin cancer prevention programs (Mathers et al 1998).

- although the expenditure estimates provide a broad picture of the health system resources usage, they should be interpreted with caution for specific diseases because the methodology is a comprehensive national accounts approach, which, while providing consistency, good coverage and totals that add up to known expenditures, is not as sensitive or accurate for any specific disease as a detailed analysis of actual costs incurred by patients with that disease.

The health system cost estimates do not purport to provide a comprehensive assessment of the impact of disease on the welfare of society. However, they do provide useful indicators of the financial burden associated with individual diseases, and in this case, the costs attributable to obesity in the causation of Type 2 diabetes. This can help to identify and analyse how health resources are allocated amongst different types of costs and services and where potential gains can be made with further investigation using other priority setting and economic evaluation techniques.

2 Overview of methods

The study firstly estimated the proportion of Type 2 diabetes and health care system costs attributable to overweight and obesity. It then considered the potential impact of interventions to prevent or reduce levels of overweight and obesity in the population and the potential reduction in costs associated with this.

2.1 The proportion of Type 2 diabetes and health system costs attributable to overweight and obesity

The approach used for estimating the costs attributable to particular risk factors typically follows the following steps (Crowley et al 1992, Stephenson et al 2000):

1. *identify those disease categories that are related to the risk factor.* In this case the risk factor is overweight and obesity and the disease category is Type 2 diabetes.

2. *quantify the relationship between the risk factor and disease morbidity and mortality using population attributable fractions (PAF).* This is based on estimates of the level of risk associated with overweight and obesity (Relative Risk or RR), and the prevalence of overweight and obesity in the Australian population. The RR estimates used were those presented by Mathers and others (1999). Results from the 1995 National Nutrition Survey (NNS) were used to provide estimates of the prevalence of overweight and obesity (ABS 1997). These are discussed further below.

3. *identify the relevant cost categories so as to estimate the costs of episodes and person years of life lost.* This report used those presented by Mathers and Penm (1998).

4. *quantify the total direct costs associated with the disease categories.* This report used those presented by Mathers and Penm (1998).

5. *use the PAF to apportion that share of these total costs directly attributable to the risk factor.* Presented in chapter 3.

6. *undertake sensitivity analysis of key epidemiological and economic parameters (or assumptions) to provide a range of cost estimates.* Presented in chapter 3.

2.2.1 The population attributable fraction for overweight/obesity and Type 2 diabetes

The population attributable fraction (PAF) can be used to determine the proportion of illness that can be attributed to a risk factor in a population. The PAF is calculated using the risk of exposure (RR) and the prevalence of that risk factor in the population.

The measure is useful for assessing the public health importance of risk factors because it represents the proportion of cases, deaths or other outcomes that can be prevented if the risk factor is eliminated (ie everyone in the population achieves a weight consistent with the 'low risk' category). The PAFs were determined using methods described by English et al (1995) and are presented below.

The aetiological fraction among those exposed (F_r) is:

$$F_r = (RR-1)/RR$$

The aetiological fraction among the total population (F_a) also known as the population attributable fraction (PAF) is:

$$F_a = p_e(RR-1)/p_e(RR-1)+1$$

where p_e is the proportion of the total population exposed to the risk factor.

Aetiological fractions for various levels of weight exposure (overweight, obesity) can be determined from the following equations as described by English et al (1995).

F_{ri} the partial aetiological fraction for i^{th} level of exposure.

$$F_{ri} = (RR_i-1)/RR_i$$

F_{ai} the partial aetiological fraction in the general population.

$$F_{ai} = p_i(RR_i-1)/\sum_{j=0}^{k} p_j(RR_j-1)+1$$

The relative risk estimates of overweight/obesity and Type 2 diabetes used here are those reported in the Australian Burden of Disease and Injury Study (table 2). These estimates were derived by extrapolation from published data of relative risks of Type 2 diabetes associated with overweight/ obesity (Colditz et al 1990, Colditz et al 1995, Carey et al 1997, Njolstad, Arnesen & Lund-Larsen 1998). This was done because the relative risks reported in these studies were based on different BMI cut-offs and so were not directly comparable between studies. In addition, the excess relative risks were discounted by 50% to take into account confounding factors, such as physical activity, which were often not adequately controlled for in these studies.

Table 2: Relative risks of Type 2 diabetes associated with overweight and obesity

Overweight				Obese			
Males		Females		Males		Females	
<65	65+	<65	65+	<65	65+	<65	65+
1.80	1.80	1.80	1.80	3.20	3.20	3.20	3.20

Source: Mathers C, Vos T & Stevenson C 1999, *The Burden of disease and injury in Australia,* Australian Institute of Health and Welfare, AIHW cat no PHE 17, Canberra.

The International Obesity Taskforce (IOTF) is currently undertaking a systematic review of the relationship between overweight/obesity and specific diseases including Type 2 diabetes. This will provide updated and potentially more accurate RR estimates when available.

Data on the prevalence of overweight and obesity in the Australian population used for calculation of the PAF are those from the 1995 NNS (ABS 1997) as presented in section 1.5. The population attributable fractions for Type 2 diabetes associated with overweight and obesity are presented in table 3. The overall PAF indicates that 41% of Type 2 diabetes in Australia can be attributed to overweight and obesity.

Table 3: Population attributable fractions (PAF) for Type 2 diabetes associated with overweight and obesity

Risk factor for Type 2 diabetes	PAF (%)
Overweight	17
Obesity	24
Overall PAF (overweight & obesity)	**41**

2.2 Potential impact of lifestyle interventions to prevent or reduce levels of overweight and obesity

Section 1.3 considered the potential impact of lifestyle interventions on levels of overweight and obesity and concluded that a conservative estimate would be that weight loss in the order of 1 to 5kg could be achieved and maintained for at least 1 year by dietary/physical activity interventions. Data from the 1995 NNS were used to estimate changes in the levels of overweight and obesity that would occur as a result of weight changes of this magnitude. This was done by simply subtracting a specified number of kilograms from the individual body weights recorded for the sample of the NNS and recalculating the prevalences of overweight and obesity. The impact of this weight reduction on the prevalence of overweight and obesity will be similar regardless of whether the weight loss is for the whole population or only for those that are currently overweight or obese.[1]

The potential impact of these weight changes on the extent of Type 2 diabetes in the Australian population was estimated by recalculating the PAFs using the 'new' levels of overweight and obesity. This is discussed further in chapter 4.

[1] Note that this in not a comment on the relative effectiveness of strategies that focus on high risk groups versus whole of population approaches, but rather the effect on levels of overweight and obesity when weight loss in the order of 1 to 5kg is achieved, regardless of the strategy used.

3 Cost of Type 2 diabetes attributable to overweight and obesity

Mathers and Penm (1998) present estimates of the total health system costs associated with Type 2 diabetes in Australia and the costs according to health system sector in 1993-94. They also provide estimates of the additional costs from complications of Type 2 diabetes. The PAF of 41% is used to apportion these costs and estimate those attributable to overweight and obesity. These amounts should be interpreted with care as they assume that all cost categories are equally affected by overweight and obesity. This could be refined in the future by consideration of a disease model that explicitly assesses the impact of overweight and obesity on each stage of the disease process including both its role in prevention and in management of the disease. The implications of this assumption are considered in relevant sections.

3.1 Total health system costs

The total health system costs for Type 2 diabetes alone in Australia, 1993-94 was estimated to be $216.7 million (excluding the costs of complications). Applying the PAF (41%) directly to the total health system costs indicates that overweight and obesity in the population accounted for approximately $89.1 million of the total health system costs for Type 2 diabetes in 1993-94 (table 4).

Table 4: Health system costs for Type 2 diabetes and the proportion attributable to overweight and obesity, Australia, 1993-94

Cost description	Total cost ($m)	Cost ($m) attributable to overweight/obesity
Type 2 diabetes	216.7	89.1

Source: Mathers & Penm 1998

3.2 Health system costs by health sectors

The health system costs by sector for Type 2 diabetes and the cost attributable to overweight and obesity are presented in table 5. Prescription pharmaceuticals alone accounted for $23.8 million of Type 2 diabetes costs attributable to overweight and obesity. Similarly, total hospital costs contributed $22 million of the total costs of Type 2 diabetes attributable to overweight and obesity. The assumption that all cost categories will be equally affected by a reduction in the levels of overweight and obesity is likely to be in error, particularly because weight reduction is known to have an impact on both prevention and management of Type 2 diabetes. It is likely that this analysis underestimates the overall impact of weight reduction.

Table 5: Health system costs by health system sector for Type 2 diabetes and the proportion attributable to overweight and obesity, Australia, 1993-94

Cost description	Total cost of Type 2 diabetes $m	Cost attributable to overweight/obesity $m	Percent of total cost (%)
Hospital inpatients			
Public hospitals	29.9	12.3	14
Private hospitals	7.0	2.9	3
Non-inpatients	16.7	6.9	8
Total hospital	**53.6**	**22.0**	**25**
Medical services			
General practitioners	14.9	6.1	7
Specialists	22.9	9.4	11
Total medical	**37.8**	**15.5**	**17#**
Pharmaceuticals			
Prescription	54.6	22.4	25
Over-the-counter	3.4	1.4	2
Total pharmaceuticals	**58.0**	**23.8**	**27**
Allied health	10.9	4.5	5
Research	11.5	4.7	5
Public health	0	0	0
Other	32.4	13.3	15
Nursing homes	12.3	5.1	6
Total costs	**216.7#**	**89.1#**	**100**

Apparent inconsistency in totals due to rounding

Source: Mathers & Penm 1998

3.3 Costs of complications of Type 2 diabetes

The cost of complications attributable to Type 2 diabetes accounted for a further $293.3 million in health system costs (table 6). These costs were estimated by Mathers and Penm (1998) from attributable fractions derived by Huse et al (1989) for the United states (see appendix table A3). Cerebrovascular disease, ischaemic heart disease, heart failure due to diabetic complications and hypertension accounted for three quarters of these costs.

Applying the PAF of Type 2 diabetes attributable to overweight and obesity (PAF=41%) to the total cost of diabetes-related complications would result in $120.5 million attributable to overweight and obesity. However, as for the costs by health sector, this figure should be interpreted with caution. The assumption that all categories of complications will be equally affected by a reduction in the levels of overweight and obesity is likely to be in error. As noted above, weight reduction is used in the management of Type 2 diabetes and so the impact of overweight and obesity on many of these costs is likely to be underestimated.

Table 6: Health system costs for complications of Type 2 diabetes, Australia, 1993-94

Complication of Type 2 diabetes	Total cost of complications of Type 2 diabetes ($m)
Hypertension	31.9
Ischaemic heart disease	68.2
Cerebrovascular disease	75.1
Heart failure due to diabetic complications	37.1
Atherosclerosis	5.8
Peripheral vascular disease	4.5
Glaucoma	3.8
Cataract	19.9
Blindness	4.2
Nephropathy	15.0
Chronic skin ulcer	25.1
Absence of extremities	2.7
Total	**293.3**

Source: Mathers & Penm 1998

4 Potential impact of lifestyle interventions on health system costs

Section 1.3 concluded that weight loss in the order of 1 to 5kg can be achieved and maintained for at least 1 year by lifestyle interventions and that weight loss is greater in those who are more overweight and obese. The impact of these weight changes on the extent of Type 2 diabetes in the Australian population was assessed by estimating the levels of overweight and obesity that would result from such weight losses in the Australian population and recalculating the PAFs using the 'new' levels of overweight and obesity. The potential impact on costs is calculated as the difference between costs associated with the initial and new PAFs. Tables 7 and 8 present these for Type 2 diabetes and diabetes-associated complications respectively.

As noted by Mathers et al (1998), care should be taken when interpreting direct costs associated with disease treatment as an estimate of the savings that would result from prevention of the disease. A number of assumptions are made in undertaking the analysis (Stephenson et al 2000). These are as follows:

1. the PAF applies equally to all stages of the disease process, so that there is a proportionally equal reduction in all cost categories.

2. there is no lag between the onset of lifestyle change and the reduction in risk.

3. the health costs are immediately converted to savings when people change their lifestyle.

4. there are no additional financial costs associated with the lifestyle change.

5. there are no differences in the demographic, socioeconomic, behavioural or other risk factors between different segments of the population as defined by level of dietary/physical activity exposure.

6. the marginal cost of health care remains static over time.

Assumptions 1, 2 and 3 would be better addressed if the analysis were able to explicitly incorporate consideration of a disease model for Type 2 diabetes and its management and to adopt an incidence (rather than prevalence) approach to costing, which could examine lifetime costs. This is not possible with the current costing and analysis framework. Assumption 4 is probably valid as far as costs to the consumer are concerned (although only a small part of overall costs). With regard to Assumption 5, some lifestyle behaviours have been reported as independent of socioeconomic status, but some would be expected to be associated with other risk factors. Assumption 6 is likely to become progressively invalid as equipment, treatment and other costs change over time.

Considering the assumptions, the results of the analysis need to be considered as gross estimates, and only indicative of the potential impact on costs.

4.1 Reducing current levels of overweight and obesity

Overweight and obese individuals have an increased risk of diabetes, and other chronic diseases such as coronary heart disease, hypertension and some cancers (Report of the NHMRC expert panel on prevention of obesity and overweight, 1997). Interventions aimed at reducing body weight in this high-risk population are appropriate given that even small reductions in weight have been shown to improve risk factors such as hyperlipidaemia, hypertension and impaired glucose tolerance (Bourn et al 1994).

Tables 7 and 8 present the levels of overweight and obesity observed in the Australian population in the 1995 NNS ('nil'), as well as the levels that would be seen with different levels of weight loss. The PAFs associated with these range from 41% for 'nil' to 33% for the scenario where those overweight and obese have a 5kg weight loss. This reduction in body weight of 1-5kg is associated with reductions in health system costs for Type 2 diabetes of between $3.6 and $18.6 million (table 7).

The combined total cost of diabetes and complications in 1993-94 was $510.0 million. Therefore, a reduction in body weight of 1-5kg could potentially reduce health system costs for Type 2 diabetes and complications by between $8.5 and $43.7 million respectively (table 8).

Table 7: Type 2 diabetes attributable to overweight and obesity: total health system costs and cost reductions by reducing the prevalence of overweight and obesity

Weight change	% Australian adults overweight & obese (1995 NNS)			PAF (%)	Type 2 diabetes attributable to overweight and obesity	
	Overweight	Obese	Total		Costs ($m)	Potential cost reductions ($m)
Nil	36.9	18.3	55.2	41	89.1	
1kg weight loss	35.5	16.7	52.2	39	85.4	3.6
2kg weight loss	33.7	15.1	48.8	38	81.4	7.7
3kg weight loss	31.6	13.9	45.5	36	77.8	11.3
4kg weight loss	30.1	12.8	42.9	34	74.4	14.7
5kg weight loss	28.2	11.6	39.8	33	70.5	18.6

Note: The total cost of Type 2 diabetes in 1993-94 was $216.7 million. The cost of Type 2 diabetes attributable to overweight and obesity= total cost of Type 2 diabetes x PAF/100.

Table 8: Type 2 diabetes and complications attributable to overweight and obesity: total health system costs and potential cost reductions by reducing the prevalence of overweight and obesity

Weight change	% Australian adults overweight & obese (1995 NNS)			PAF (%)	Type 2 diabetes attributable to overweight and obesity	
	Overweight	Obese	Total		Costs ($m)	Potential cost reductions ($m)
Nil	36.9	18.3	55.2	41	209.6	
1kg weight loss	35.5	16.7	52.2	39	201.1	8.5
2kg weight loss	33.7	15.1	48.8	38	191.5	18.1
3kg weight loss	31.6	13.9	45.5	36	183.0	26.1
4kg weight loss	30.1	12.8	42.9	34	175.1	34.5
5kg weight loss	28.2	11.6	39.8	33	165.9	43.7

Note: The total cost of Type 2 diabetes and complications in 1993-94 was $510.0 million (ie $216.7 million + $293.3 million). The cost of Type 2 diabetes and complications attributable to overweight and obesity= total cost of Type 2 diabetes and complications x PAF/100.

4.2 Preventing predicted increases in overweight and obesity

The NHMRC expert panel on prevention of overweight and obesity observed …

> *"…while the treatment of people who are currently overweight and obese should continue, we believe that the current trend of an increasing prevalence of overweight and obesity will be reversed only if urgent steps are taken to prevent people from becoming overweight and obese*." (Report of the NHMRC expert panel on prevention of obesity and overweight, 1997)

Studies in children show increasing rates of overweight and obesity (Magarey et al 2001) and recent estimates from the AusDiab study indicate that prevalences of overweight, obesity and diabetes in Australian adults continues to increase (Dunstan et al 2001). The trend and projections for diabetes are shown in Figure 1.

Based on this, it is reasonable to expect that the costs associated with Type 2 diabetes and its complications will grow for at least the near future. Therefore, there would be cost savings for the health care system from simply arresting this increase in rates of overweight and obesity. That is, the 'nil' weight gain described in tables 6 and 7 would reduce expected costs and the potential cost reductions for weight loss is underestimated if judged against a trend to increased levels of overweight and obesity and their associated costs.

4.3 Comparison with other cost of illness studies

The previous study of diet-related cost of disease in Australia by Crowley et al (1992) estimated a direct cost of $83-249 million in 1989-90 for Type 2 diabetes. However, this previous study used sensitivity analysis to provide a range of estimates of PAF. These estimates were based on epidemiological evidence, current practice and expert opinion. The low, middle and high PAF bounds were 25, 50 and 75% for Type 2 diabetes attributable to obesity respectively. Our PAF of 24% for Type 2 diabetes attributable to obesity (PAF=41% for type 2 diabetes attributable to overweight and obesity) is close to the lower bound estimate of Crowley et al (1992).

In 1995, the AIHW and the Centre for Health Program Evaluation (CHPE) estimated the costs of obesity-related diseases in Australia as part of the work associated with the Macro Economic Evaluation Model (section 1.6). This included the above work by Crowley et al (1992) on the cost of diet-related disease. The PAF of Type 2 diabetes attributable to obesity only was estimated at 66%. This differs from the PAFs presented here (17% and 24% for overweight and obesity respectively) as the RR estimates in the CHPE work were derived from the USA economic cost of obesity study (RR= 16.6, Colditz 1992).

Both the PAF estimates by Crowley et al (1992) and the AIHW/CHPE are higher than the 24% of diabetes attributable to obesity (BMI 30.0+) reported here. This probably reflects both an improved knowledge base regarding obesity and Type 2 diabetes relationships and the conservative approach used in the current study, probably leading to an overall underestimate of PAF values and obesity-related costs.

Glossary

Body Mass Index (BMI)

Body mass index (BMI) is the body weight in kilograms divided by the square of height in metres (kg/m^2). The groups used are those recommended by the World Health Organization (1995).

Category	BMI range (kg/m^2)
Underweight or thinness	<18.5
Normal or acceptable weight range (a)	18.5-<20
	20-<25
Overweight	25-<30
Obesity	30+

(a) The normal or acceptable range has been split to enable comparison with NHMRC categories

Cost of illness (COI)

Cost-of-illness refers to the economic consequences of poor health.

Diabetes mellitus

Diabetes mellitus is a chronic disease characterised by hyperglycemia or high levels of glucose, which is caused by deficient insulin production and/or resistance to its action. (The criteria used to classify glucose tolerance status are presented in the appendix.)

Direct costs

Direct costs refer to the health sector costs for prevention, diagnosis and treatment of diseases and may include costs for ambulance, inpatient, nursing home, outpatient, rehabilitation, allied health, research, community health and medical services and consumption of pharmaceuticals.

Impaired glucose tolerance (IGT)

Impaired glucose tolerance is defined by both fasting glucose levels and two hour post oral glucose load results, which fall between normal values and those that define diabetes. (The criteria used to classify glucose tolerance status are presented in the appendix.)

Population attributable fraction (PAF)

Population attributable fraction refers to the proportion of illness in a population that can attributed to a particular risk factor.

Relative risk (RR)

The ratio of risk of disease in the exposed group compared with those not exposed to the risk factor.

Type 2 diabetes

Type 2 diabetes is a type of diabetes mellitus characterised by insulin resistance or impaired insulin production. (The criteria used to classify glucose tolerance status are presented in the appendix.)

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Appendices

Appendix 1: WHO Classifications for glucose tolerance status

The disease costing methodology used to derive cost data for Type 2 diabetes comes from the Disease Costs and Impact Study (DCIS). The costing methodology is described in detail elsewhere (Mathers et al 1998). The WHO International Classification of Disease-Ninth Revision (ICD-9 code 250.0) and International Classification of Primary Health Care (ICPC code T90) were used to identify costs associated with Type 2 diabetes[1].

Table A1: Glucose concentration for diagnosis of diabetes mellitus and other categories of hyperglycaemia

	Glucose concentration, mmol l^{-1} (mg dl^{-1})		
	Whole Blood		Plasma #
	Venus	Capillary	Venus
Diabetes Mellitus			
Fasting	6.1+ (110+)	6.1+ (110+)	7.0+ (126+)
OR			
Two hour post-glucose load	10.0+ (180+)	11.1+ (200+)	11.1+ (200+)
OR BOTH			
Impaired Glucose Tolerance (IGT)			
Fasting concentration (if measured)	<6.1 (<110)	<6.1 (<110)	<7.0 (<126)
AND			
Two hour post-glucose load	6.7+ (120+) and <10.0 (<180)	7.8+ (140+) and <11.1 (<200)	7.8+ (140+) and <11.1 (<200)
Impaired Fasting Glycaemia (IFG)			
Fasting	5.6+ (100+) and <6.1 (<110)	5.6+ (100+) and <6.1 (<110)	6.1+ (110+) and <7.0 (<126)
Two hour (if measured)	<6.7 (<120)	<7.8 (<140)	<7.8 (<140)

Corresponding values for capillary plasma for Diabetes Mellitus: fasting 7.0+ (126+), two hour 12.2+ (220+)

Source: WHO 1999

[1] Classification/cut-offs used define diabetes in ICD 9 have been revised from >7.8mmol l^{-1} to >7.0mmol l^{-1}. The incidence of diabetes will most likely increase using this new classification/cut-off.

Appendix 2: Criteria for defining overweight and obesity

In this report, body mass index (BMI) categories used to define overweight and obesity are based on those recommended by the WHO Expert Committee on Physical Status: the Use and Interpretation of Anthropometry (1995) (table A2). The 1995 NNS and 2001 AusDiab estimates of overweight and obesity were based on these categories.

Table A2: Weight categories as defined by BMI cut-offs

Category	BMI range (kg/m^2)
Not overweight	<25
Overweight	25 to <30.0
Obese	30+

Source: WHO Expert Committee on Physical Status: the Use and Interpretation of Anthropometry 1995

Appendix 3: Complications attributable to Type 2 diabetes

The costs of complications attributable to Type 2 diabetes were derived by applying PAFs determined by Huse et al (1989) for the United States (table A3). Mathers and Penm (1998) note that the reported prevalence of Type 2 diabetes in Australia in 1995 was broadly comparable than that for the US population in 1984-86. At present, the PAF of complications attributable to Type 2 diabetes for the Australian population have not been estimated.

Table A3: Population attributable fractions for complications attributable to Type 2 diabetes by age and sex

Condition	ICD-9 codes	PAF (%)			
		<65 years		65 years and over	
		Males	Females	Males	Females
Circulatory disorders					
Hypertension	401-405	2.0	2.2	4.1	6.4
Ischemic heart disease	410-414	4.8	6.8	8.4	9.8
Cerebrovascular disease	430-438	4.8	5.0	17.0	10.1
Heart failure	428, 429.2-429.3, 429.9	4.8	6.8	8.4	9.8
Atherosclerosis	440	6.1	5.3	11.5	10.0
Peripheral vascular disease	443, 459.8-459.9	6.1	5.3	11.5	10.0
Visual disorders					
Glaucoma	365	7.5	8.4	9.5	9.8
Cataract	366	5.0	5.6	5.7	5.9
Blindness	369	11.6	12.9	48.1	49.1
Other disorders					
Nephropathy	580-586	18.0	18.0	18.7	19.3
Chronic skin ulcer	707	5.0	5.6	26.9	27.6
Absence of extremities	736	3.1	3.5	18.5	19.1

Source: Mathers & Penm 1998, Huse et al 1989