The cost of diet-related disease in Australia

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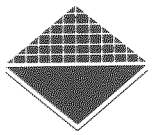

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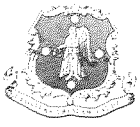

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FOREWORD

This paper has been prepared on a collaborative basis by the Australian Institute of Health and Welfare and the National Centre for Health Program Evaluation. While cognisant of the scope and limitations of the information presented, the authors believe it will help to stimulate discussion and provide a useful contribution to the work being undertaken on the development of a National Food and Nutrition Policy. Comment on the paper, its assumptions and methodologies is most welcome.

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SUMMARY

- The focus of this report is the provision of estimates of the cost to the health delivery system of diet-related disease in the year 1989-90.

- The major causes of death, illness and disability in Australia thought to have a nutrition component in their etiology and for which some form of prevention is likely to be applicable are: coronary heart disease, stroke, hypertension, atherosclerosis, some forms of cancer (stomach, colon, rectal, breast and endometrial), diabetes (non-insulin dependent), osteoporosis, dental caries, gallbladder disease, and non-cancer disorders of the large bowel (diverticular disease, constipation and hemorrhoids).

- With the exception of alcohol-related disease and cancer, there is limited information in the literature on the population attributable fractions necessary to ascertain what proportion of these diseases is directly due to diet. This study has utilised three values - high and low estimates of the population attributable fractions to indicate upper and lower bounds, with the mid value representing the most likely relationship.

- Using the mid value of the population attributable fractions, premature deaths in 1989-90 due to poor diet contributed 36,604 potential years of life lost (PYLL) to age 65 and 100,055 to age 75.

- PYLL due to poor diet (excluding alcohol) is 70 per cent as large as PYLL due to smoking. If alcohol-related disease is included in the diet category, PYLL is 180 per cent as large as PYLL due to smoking.

- Using the mid value population attributable fractions, the direct cost (ie. the cost of health care services - hospital, medical, pharmaceutical, allied professional and nursing home) of diet-related disease is $1,520 million in 1989-90. The indirect cost (ie. earnings foregone through illness and premature death) is $746 million in 1989-90, giving total costs of $2,267 million.

- If the total cost of diseases due to alcohol is added, the total estimate in 1989-90 rises to $3,620 million.

- This cost-of-illness study is useful for a number of purposes, including use as an indicator of public health significance of diet-related disease. It should not be used to justify health promotion activity without regard to allocational efficiency, which requires a realistic consideration of both costs and outcomes of individual projects.

- The direct cost of diet-related disease ($1,520 million in 1989-90) represents the maximum possible annual "savings" that health promotion programs focusing on food and nutrition policy could hope to achieve. It should not be interpreted as an estimate of financial cost savings realisable by government in the short term, but rather as an approximate estimate of the "opportunity cost" of resources devoted to the treatment of preventable disease, that could be available for the treatment of non preventable disease. Conversion of "opportunity costs" (ie benefits foregone) into expenditure savings involves a number of difficult and complex considerations beyond the scope of this paper.

- The scope and limitations of this study are specified in the text and should be carefully noted before utilising the cost-of-illness estimates.

1. INTRODUCTION

The health status of a population, which can be expressed in various ways, is usually not described in terms of health, but rather in terms of the absence of health - mortality and morbidity. Numerous indicators are available to give some impression of mortality and morbidity, but none can assess all of their dimensions, particularly the cost in terms of human suffering.

This is an important issue for economic analysis, which is not primarily concerned with dollars, but rather with total community welfare. The main economic burden of diet-related disease falls on individuals and their families through premature death and loss in quality of life. Any reduction in health status due to diet-related disease will also result in a number of undesirable second order effects. These will include an altered pattern of resource allocation within the health care delivery system, as well as wide ranging effects on consumption and production of private and public goods and services. Costs imposed on the health system represent only one part of the impact of a change in health status. While the impact of disease on quality of life is difficult to measure and value, it is important that this central component of the economic burden not be forgotten.

The purpose of this discussion paper is to provide soundly based estimates of the cost of diet-related disease on the health care delivery system. Preliminary estimates are also provided on the impact of diet-related disease on the broader economy in terms of sick leave and foregone earnings due to premature death, and of the health status impact using the indicator "potential years of life lost".

"Disease costing" or "cost-of-illness" is a technique available to estimate the cost impact of disease on a community. Economists make a distinction in this work between the "direct" costs of providing health care services (both to patients and their families and to service providers) and the "indirect" costs, which include lost production due to absenteeism and premature death. Disease costings are generally not able, however, to provide a comprehensive assessment of the impact of disease on the total welfare of society. That would need to incorporate the effects on quality of life or human suffering, for which satisfactory measures are still being developed. By including information in this paper on potential years of life lost due to premature death, aspects of this issue are addressed, but not the quality of these years, particularly the diminished quality of life due to disease morbidity.

Disease costing estimates can nonetheless be useful to health planners for a variety of purposes. In the case of a specific disease or diagnostic group, they may wish to know the relationship between its incidence or prevalence and the consequential utilisation of health services and costs, in order to identify where potential improvements in health status and/or where "cost savings" might be achieved by means of prevention activities. Disease costing may also be used by planners for comparisons between the relative burden of different diseases or diagnostic groups, and these comparisons may assist in setting priorities in prevention.

Predictions of financial savings in health expenditure from prevention should be interpreted carefully. While individual prevention activities may save money, a longer or better life does not necessarily imply that fewer resources are spent on health care when expenditure over a whole lifetime is taken into account. There are important choices about where to spend available resources and the priority attached to citizens being able to lead healthy and long lives. The extent of preventable illness and premature mortality suggests that considerable potential still exists to improve health outcomes for the community. This does not imply, however, that all health promotion programs are necessarily value for money, or that they are necessarily cheaper than cure. The decision to carry out a prevention program (or any health program) should ideally be based on a careful consideration of all the costs, all the possible risks, and all the possible benefits of the program.

Cost-of-illness studies are particularly useful for identifying and analysing how resources are currently allocated between different types of costs, between different types of services and between different diseases. However, they do not provide sufficient information on their own to permit a decision on whether the resource allocation is efficient. This can only be done using evaluation techniques that combine both costs and outcomes. There is little point pursuing a case for further expenditure on prevention without taking the benefits and costs of prevention into account. These benefits include both the improvements in mortality, morbidity and quality of life, as well as the extent and timing of the cost-of-illness explored in this paper. Cost-of-illness studies should not be used to justify further expenditures without regard to allocational efficiency.

It is generally recognised that diet, smoking and alcohol consumption are three major life style factors in the etiology of many chronic diseases. Despite the mounting evidence linking diet and disease, there have been only a few attempts to quantify the economic burden of diet-related morbidity and mortality. This is in marked contrast to the effects of smoking and alcohol consumption, where a number of studies have been undertaken, both in Australia and overseas, identifying the magnitude of the economic burden they impose on the community.

The only estimate previously available for Australia is that cited in the report of the Nutrition Taskforce of the Better Health Committee[1]. The Taskforce estimated that in 1984 diet-related chronic diseases accounted for $5 billion in health care costs. This figure was extrapolated from a 1977 United States Senate Report, "Dietary Goals for the United States" and was later updated in the "Health For All Australians" report[2] to $6 billion for 1988. The US Senate report, a comprehensive attempt at quantifying the health related costs of poor diet, maintained that improved nutrition might reduce that nation's health bill by one third.

In addition to the lack of cost-of-illness studies, there is also a dearth of information on the economic evaluation of nutrition intervention programs. One notable exception is a study by Hall et. al.[3] which looked at the cost-effectiveness analysis of alternative strategies for the prevention of coronary

heart disease. Whilst the alternate strategies analysed in that study targeted multiple risk factors, modification of diet was an important component. The study by Hall et. al. is discussed in Section 5. Obtaining evidence regarding the cost-effectiveness of nutrition intervention programs in reducing diet-related morbidity and mortality is important for analysing the feasibility of the nutrition goals and targets established in the "Health For All Australians" report[2] (refer Appendix A).

There are a number of basic steps to be followed for a cost-of-illness study on diet-related disease. These are to:

(i) identify those diseases that are diet-related;

(ii) quantify the relationship between dietary risk factors and disease mortality and morbidity (i.e. the population attributable fractions);

(iii) identify the relevant economic cost categories to be estimated;

(iv) quantify the total costs associated with diet-related disease;

(v) use the population attributable fractions to apportion that share of these total costs which is due to diet; and

(vi) undertake sensitivity analysis of key epidemiological and economic parameters (or assumptions) to provide a range of cost estimates.

The remainder of the paper is in four sections, and generally follows these steps. Section 2 identifies diet-related diseases, summarises the evidence identifying the etiological relationship between diet and disease, and quantifies the impact of diet-related diseases in terms of preventable years of life lost.

Section 3 identifies the relevant economic costs, briefly describes the methodology used to estimate the costs-of-illness and overviews the results. This section includes information on the costs of health care that can be reasonably attributed to diet. This estimate then becomes the maximum potential "savings" in any one year that prevention programs can seek to realise. It is important to realise that this "cost savings" estimate is based on the economic notion of "opportunity cost". It is probably best conceived of as the cost of resources devoted to the treatment of preventable diseases that could be available for the treatment of non preventable disease. The notion of "opportunity cost" is essentially one of benefits foregone, and conversion into actual dollar cost savings introduces a number of additional and quite complex considerations.

Section 4 compares the results obtained with previous cost-of-illness studies undertaken in Australia and overseas, and Section 5 reviews evaluation studies of specific nutrition intervention programs and outlines a macro economic evaluation approach that the AIHW is developing.

2. THE RELATIONSHIP BETWEEN DIET AND DISEASE

Identification of Diet Related Disease

Over recent years a number of reports in Australia and other countries have reviewed the relationship between diet and disease [1,4,5,6,7,8].

The major causes of death, illness and disability in Australia thought to have a nutrition component in their etiology and for which some form of prevention is likely to be applicable are: coronary heart disease; stroke; hypertension; atherosclerosis; some forms of cancer (stomach, colon, rectal, breast and endometrial); diabetes (non-insulin dependent); osteoporosis; dental caries; gallbladder disease; non-cancer disorders of the large bowel; (such as diverticular disease and constipation) and iron deficiency anemia.

It is beyond the scope of this paper to discuss in any detail the evidence for and against the relationship between nutrition and disease. Judgements must be made on the basis of evidence from many sources, including clinical observation, animal experiments, epidemiological studies and a limited number of experimental studies on humans. The most compelling evidence would, in theory, be supplied by controlled life-time feed experiments on humans, and is obviously unobtainable. Much of the strong evidence of the relationship between diet and disease comes from epidemiological studies. Well designed and conducted intervention studies can provide direct evidence on which to judge whether diet causes (or prevents) a disease.

It is difficult to prove that the links between dietary components and disease are causal, or to assess the exact proportion of disease incidence attributable to diet alone. The evidence to date more appropriately defines a strong association between diet and many of the chronic diseases of modern lifestyle. These diseases are caused by a combination and interaction of multiple environmental, behavioural, biological, social and genetic factors. The exact proportion that can be attributed directly to diet is uncertain.

The multifactorial etiology of coronary heart disease (CHD) illustrates this. The following are established or suggested risk factors for this disease:

- raised blood lipids (cholesterol and triglycerides)
- hypertension
- cigarette smoking
- overweight/obesity
- diabetes mellitus
- physical inactivity
- psychological stress
- genetic predisposition

Diet can be linked to each of these risk factors with the probable exception of cigarette smoking, genetic predisposition and stress.

If just one of these diet-related risk factors, say hypertension, is considered, it is not known with certainty what the precise role of diet is in its development. Excess dietary sodium, obesity and alcohol are the best known of the dietary factors implicated in the development of hypertension. The overall contribution of sodium to the etiology of hypertension is still unclear. There have been well designed randomised clinical trials undertaken in mildly hypertensive patients which have shown that sodium restriction can improve this condition. Correlation studies also show that countries with high salt intakes have a higher prevalence of hypertensive people. But it cannot be said with certainty that high sodium intake causes hypertension. It is extremely difficult to disentangle the individual effects of the risk factors for the development of coronary heart disease.

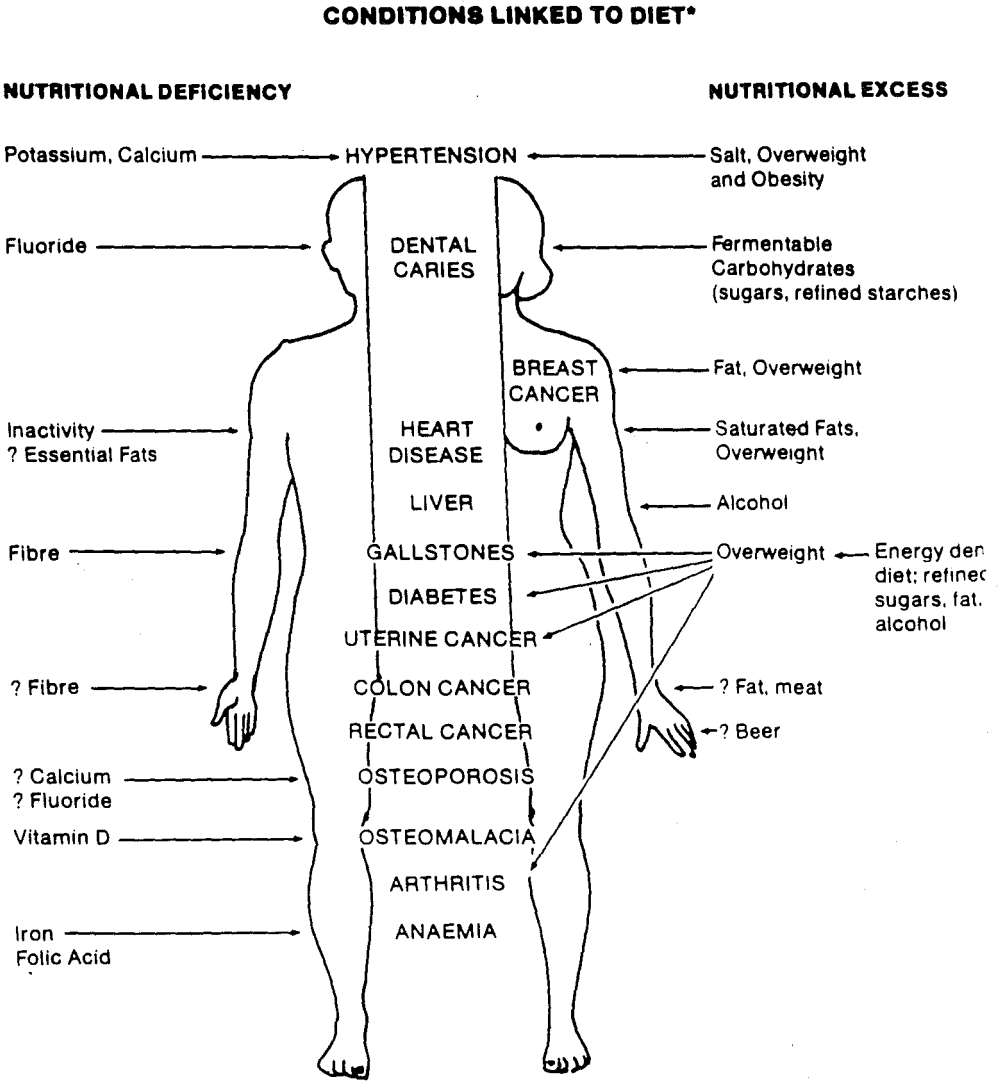
Figure 1 summarises the conditions linked to nutritional deficiency or excess nutrition. The prevailing evidence suggests that in the developed world the diseases of concern are due to an over-consumption of total energy and the macronutrients of fats and refined carbohydrates, as well as excessive alcohol and low fibre intakes. In Australia, for example, there is less evidence of frank nutritional deficiencies except in population sub-groups known to be at extra risk, such as vegetarians, and pregnant and lactating women. The latter two groups have higher iron requirements and if their diet is marginal in this nutrient, they may be at higher risk of developing iron-deficient anemia. In addition, studies have shown that mild under-nutrition occurs in Aboriginal children [9,10].

The present study reports on the economic costs of the following diseases:

- Coronary heart disease
- Hypertension
- Atherosclerosis
- Stroke
- Diabetes mellitus, non-insulin dependent
- Certain cancers
  - stomach
  - colon
  - rectal
  - breast
  - endometrium
- Osteoporosis
- Non-cancer disorders of the large bowel (diverticular disease, hemorrhoids, constipation)
- Dental caries
- Gallbladder disease
- Iron deficiency anemia
- Alcohol related diseases

Deaths related to hypertension have been variously classified over recent years. They have either been considered as a separate entity or combined with such classes of atherosclerotic cardiovascular diseases as CHD and stroke.

FIGURE 1



*Adapted from: James. W.P.T. 1985. *Guidelines for health nutrition and for the prevention of diseases of major public health importance in Europe*. 4th draft WHO. Copennagen.

Using mortality as a vital statistic alone in discussing the epidemiology of hypertension has its limitations. Many researchers treat hypertension as primarily a risk factor for CHD and stroke, rather than as a disease entity in itself, and often do not consider its associated morbidity or economic burden.

Hypertension has a separate disease classification in the ICD9-CM and is the most common cardiovascular condition. The preliminary analysis of the 1989 National Heart Foundation Risk Factor Prevalence Study[11], for example, found that one in six men, and one in eight women were hypertensive. Despite its low unit health care costs, the high prevalence of hypertension means that its aggregate costs are considerable. For this reason, estimates have been made of the costs to the health sector and the Australian economy attributable to the current management of hypertensive disease.

Quantifying the Relationship Between Diet and Disease

Whilst the reports cited above comprehensively address the evidence regarding the dietary risk factors for chronic disease and review their prevalence and incidence, they give little attention to reviewing the estimates of the proportion of disease directly attributable to diet. The statistic most commonly cited in Australia (often inappropriately) to describe the relationship between diet and disease is that 56.6% of all deaths in Australia in 1983 (based on ABS Deaths data) are diet-related. This in no way should be interpreted as meaning that 56.6% of all deaths are attributable to diet. The most that can be said is that diet is indicated as a risk factor for this proportion of all deaths.

For cost-of-illness studies the fundamental epidemiological statistic that is necessary to quantify the direct relationship between a risk factor of interest and disease (and thus quantify its associated economic costs) is the population attributable fraction (PAF). It has been defined as the proportion of total events (e.g. deaths or morbidity) in a population that could be prevented if a particular risk factor could be eliminated.

The PAF reflects the overall impact of morbidity and mortality from a risk factor in the specified population. Thus it can be interpreted from an etiological viewpoint (causal outcomes attributed to a particular risk factor) or from a prevention viewpoint (the maximum number of events that could be prevented). Many epidemiologists use the concept of "preventable proportion" as a useful generalisation of the concept of "population attributable fraction".

For the situation where only one category of exposure is present the formula for the PAF is:

$$PAF = [p\,(RR- 1)]\,/[p\,(RR-1) + 1]$$

where p = prevalence of exposure in age group

RR = relative risk = $(I_e)/(I_o)$

I_e = the incidence of the condition among those exposed to the risk factor

I_o = the incidence among those not exposed

The formula can be extended to deal with multiple-category exposure, such as different relative risks associated with different categories of cholesterol or of obesity[12].

$$PAF = 1 - (1 / [\sum_{i=0}^{k} P(RR_i)])$$

where p = the proportion of the population in the ith risk-factor category

RR_i = the relative risk for the disease in the ith risk-factor category compared with that of persons who do not have the risk factor.

The PAF can be presented as a fraction or as a percentage. Thus a PAF of 0.73 means that 73 per cent of the incidence of the disease could be eliminated by removal of the risk factor (or conversely, that the risk factor contributes to 73 per cent of the incidence of the disease).

PAF's can be calculated provided the prevalence of the risk factor in the community is known, as well as its relative risk for the outcome of interest. A number of studies have estimated PAF's for tobacco and alcohol-related behaviour. Such calculations are more straightforward as dose-response relationships in terms of health impacts have been demonstrated. With diet-related behaviour, however, calculation of PAF's is more complex. Certain food components have been found to have harmful effects, while others have shown protective effects. Exposure to the risk factors is not simply dichotomous - absent or present - but is present on an ordinal scale[13].

Armstrong and Holman[14] have calculated PAF's, which they have termed etiological fractions, on a detailed age-sex breakdown for both alcohol and tobacco. Their PAF's for alcohol are set out in Appendix B. Other studies give an overall attributable fraction for a risk factor and the development of disease, depending on the availability of relative risk and prevalence data for various age and sex cohorts.

Apart from alcohol-related disease, and certain cancers, there is scarce information in the literature applying PAF's to diet-related disease. Given the lack of hard quantifiable evidence, the present study relies on a sensitivity analysis to provide a range of estimates based on differing assumptions regarding the values that the population attributable fractions are likely to take. Rather than giving a single value for that part of each disease that is attributable to diet, three estimates are given. The high and low estimates indicate the likely upper and lower bounds with the mid value representing the most likely value. The setting of these upper and lower bounds was based on a number of criteria including consideration of empirical evidence from epidemiological studies, consideration of current practice in the literature and judgements from experts in the field.

The evidence on PAF's for particular diseases is set out in Appendix C, together with an outline of the rationale for selecting the range of estimates of the proportion of disease attributable to diet. Table 1 summarises the estimates that have been used.

TABLE 1: PROPORTION OF DISEASE ONSET ATTRIBUTABLE TO DIET

DISEASE	RANGE OF ESTIMATES		
	HIGH %	MIDDLE %	LOW %
Coronary heart disease	60	40	20
Hypertension	75	50	25
Atherosclerosis	75	50	25
Stroke	60	40	20
Diabetes mellitus (non- insulin dependent)	75	50	25
Cancers - overall	35		
- stomach		50	15
- colon		35	15
- rectum		35	15
- breast		30	10
- endometrium		25	10
Osteoporosis	30	20	10
Diverticular disease	75	50	25
Hemorrhoids	75	50	25
Dental caries	75	50	25
Gallbladder disease	75	50	25
Constipation	75	50	25
Iron deficiency anemia	75	50	25
Alcohol-related disease	Refer Appendix A		

While causality has not necessarily been proven in the work on diet-related disease it is considered that this approach provides a useful input for policy development and analysis. If research input to the planning process was to wait until fully acceptable and precise estimates of PAF's were available, there could be an indefinite delay because of the difficulties in proving causality. Policy development and analysis needs to be based upon the best information available, providing that this is not used out of context, and that the limitations are borne firmly in mind. The approach outlined in this paper enables a start to be made.

Mortality and morbidity attributable to diet-related diseases

Numerous health indicators that give some impression of the nature and degree of ill health in the community have been developed. The number of deaths and the mortality rate per 100,000 persons are common mortality indicators. The disadvantage of these indicators is that they do not give an adequate reflection of the importance of premature death. The concept of "potential years of life lost" (PYLL) is important, because it puts more emphasis on premature death than death at old age. Using PYLL to age 65 and age 75 are both useful because they involve different levels of morbidity for the extra years of life lived.

The person years of life lost due to diet-related deaths have been estimated using the same methodology as that of Holman et. al.[14]. This methodology uses a life table approach to estimate the extra years of life which would otherwise have been lived by each person who died of a diet-related disease, taking into account the risk of death at each subsequent age from all other diseases. An adjustment for death from other causes is necessary, if the mortality benefit from individual health promotion activities is not to be grossly over-stated.

Table 2 provides mortality information for diet-related diseases prior to application of the PAF's, while Table 3 provides information on that part of the potential years of life lost (PYLL) due to these diseases that can be attributed to nutrition.

If the diseases are ranked according to PYLL attributable to diet using the 'medium' or 'low' PAF's, then the order is coronary heart disease followed by neoplasms, stroke, diabetes and hypertension. If the high PAF's are used, however, neoplasms moves ahead of coronary heart disease.

Table 4 provides a further comparison of PYLL to age 65 for diet-related diseases as a group compared to cancers, injury, cardiovascular disease and smoking related disease. PYLL due to poor diet is 70 per cent as large as PYLL due to smoking, and indicates that poor diet is an important source of premature mortality. If alcohol is included as part of diet, then their combined PYLL is 180 per cent of PYLL due to smoking. It is arguable that poor diet as a risk factor, has been under-rated relative to the attention given smoking.

One of the difficulties with mortality indicators is that they fail to take into account the degree to which chronic, non-fatal diseases and handicaps affect a person's sense of well-being.

Indicators of morbidity in a population can be grouped together broadly as:

- incidence and prevalence for certain diseases;
- prevalence of impairments, disabilities and handicaps;
- subjective well-being and other answers to health questionnaires (quality-of-life etc.);

- utilisation of health services ;
- sickness, absenteeism and work disability; and
- prevalence of known determinants of disease.

While most of these indicators differ in nature, they do overlap. Incidence and prevalence rates and answers to health questionnaires give an impression of the presence of morbidity itself, whereas the use of health care facilities and absenteeism can be considered the consequences of morbidity. Table 2 includes morbidity information for the diet-related diseases as reflected by public hospital bed days, and the number of hospital separations.

TABLE 2: MORBIDITY AND MORTALITY OF DIET-RELATED DISEASE IN AUSTRALIA (PRIOR TO USING POPULATION ATTRIBUTABLE FRACTIONS)

	Deaths (1)	PYLL to 65 (2)	PYLL to 75 (2)	Hospital bed days (3)	No. hospital separations (3)
Coronary heart disease	32,639	43,289	127,156	732,290	99,025
Stroke	12,579	12,347	32,360	752,786	38,997
Hypertension	1,150	1,139	3,159	75,934	8,925
Diabetes mellitus (non-insulin dependent)	2,079(4)	3,165	8,682	154,553	13,496
Cancers					
- stomach	1,300	3,265	7,640	43,614	3,586
- colon	3,100	8,299	20,158	111,186	8,319
- rectum	1,001	2,618	6,694	85,834	6,049
- breast	2,448	14,217	27,092	127,402	14,252
- endometrium	235	474	1,301	19,120	2,021
Dental caries	-	-	-	15,834	13,431
Gallbladder disease	109	667	1,754	238,804	29,137
Non cancer disorders of the large bowel					
- Constipation	7	0	2	25,165	6,289
- Diverticular disease	246	162	557	65,918	11,033
- Hemorrhoids	4	2	17	74,944	18,328
Osteoporosis	867(5)	338	1,124	57,708(6)	3,032(6)
Iron deficiency anemia	31	3	27	25,988	4,929
TOTAL	**57,795**	**89,985**	**237,723**	**2,607,080**	**280,849**

Source: AIHW

Notes:

(1) No. of deaths for males and females, 1989, taken from the ABS mortality data.

(2) Potential years of life lost (undiscounted) due to deaths in 1989 from diseases for which diet is a risk factor (before applying population attributable fractions).

(3) Estimated from hospital morbidity data held by the AIHW. For public hospitals includes NSW 88/89, VIC 88/89, SA 88/89, NT 87/88, ACT 87/88. For private hospitals includes NSW 88/89, and SA 88/89. Data for other States/Territories estimated by pro-ration.

(4) Estimated by excluding deaths due to diabetes mellitus at ages prior to 40 years(to separate non-insulin dependent from insulin dependent).

(5) Estimated assuming 51%, 71% and 91% of deaths due to accidental falls as a consequence of osteoporosis for age groups 45-59, 60-74, 75+ respectively (refer Appendix C).

(6) These estimates are for osteoporosis only, excluding fractures.

TABLE 3: POTENTIAL YEARS OF LIFE LOST (PYLL) DUE TO PREMATURE DEATH IN AUSTRALIA IN 1989 ATTRIBUTED TO DIET USING HIGH, MEDIUM AND LOW ESTIMATES OF POPULATION ATTRIBUTABLE FRACTIONS(1)

	POTENTIAL YEARS OF LIFE LOST (PYLL)		
DIET RELATED DISEASES	HIGH	MIDDLE	LOW
Coronary Heart Disease			
to age 65	25,847	17,190	8,575
to age 75	75,237	49,814	24,738
Stroke			
to age 65	9,256	6,168	3,083
to age 75	24,229	16,126	8,050
Hypertension deaths			
to age 65	865	569	285
to age 75	2,369	1,579	789
Diabetes (non-insulin dependent)(3)			
to age 65	5,217	3,476	1,737
to age 75	17,061	11,358	5,671
Neoplasms			
to age 65	39,187	8,433	2,787
to age 75	83,760	18,064	5,984
Total PYLL(2)			
to age 65	79,808	36,604	16,961
to age 75	205,358	100,055	46,716

Source: C Mathers, unpublished

Notes:

(1) For population attributable fractions refer Table 1.

(2) PYLL due to a number of diseases analysed as a group are always greater than the sum of the individual PYLL calculated for each condition separately[14].

(3) Whittall et. al.[15] estimated that one in three deaths due to diabetes is coded with diabetes as the cause of death in Australia. Deaths due to diabetes in persons aged 40 years or more have been multiplied by three.

TABLE 4: COMPARISON OF PYLL TO AGE 65 FROM CANCERS, INJURY, CARDIOVASCULAR DISEASE, DIET-RELATED DISEASE AND SMOKING-RELATED DISEASE, FOR AUSTRALIA, 1989

	Non-discounted			Discounted(1)		
Disease category(2)	Male	Female	Total	Male	Female	Total
Injuries	145,882	46,282	192,164	68,931	21,234	90,165
Cancers	59,251	53,818	113,069	39,487	34,996	74,483
Cardiovascular	54,133	20,561	74,694	38,373	14,241	52,614
Smoking related	37,720	14,219	51,939	25,916	8.860	34,776
Diet related(3)	22,774	13,830	36,604	16,818	9,946	26,763
Alcohol related	40,394	16,257	56,651	20,023	8,532	28,555

Source: C Mathers, unpublished

Notes:

(1) Discount rate of 5% was used
(2) These disease categories not mutually exclusive
(3) Medium estimates of population attributable fractions utilised.

3 ECONOMIC COSTS OF DISEASE

Identification of the economic costs of disease

Disease costing is the estimation of the costs-of-illness, direct and indirect, per diagnostic group. The direct costs-of-illness are the costs of health care services for diagnosing and treating illness as well as those for rehabilitation, research, training and capital investment in medical facilities. Direct costs impact on both patients and their families, as well as on service providers. For the health services sector, direct costs are the costs of foregone alternatives: if there were less illness then a proportion of the resources spent on diagnosing, treating and caring for the sick could be put to other uses. For the patient and family, these costs are the time and expenditure consumed by activities associated with ill health, that could have been devoted to other pursuits.

Indirect costs are usually defined as the value of the output that is lost because people are too ill to work or have died prematurely. Under this definition they represent losses to the total output of an economy and are often measured using the human capital approach . There are important issues about the validity of such measures, including whether total production for an economy is reduced by absenteeism or death, whether losses in the form of household services should be included, and whether earnings are an adequate measure of value.

The advantages and disadvantages of this approach and an alternative "willingness-to-pay" approach, are further explored in Appendices D and E. It is important to note (for those interested in financial "savings") that net present value estimates of foregone production do not estimate the resources that would become available to the community for expenditure on other health programs. Only direct costs should be used in that financial context.

There is also an important third category of costs, often called "intangibles" (e.g. pain, bereavement, suffering, anxiety) but more recently explored in the quality of life literature. While difficult to measure and express in money terms, this category is relevant when discussing the impact of illness or death.

A summary of the relevant costs included in cost-of-illness studies is set out in Table 5. Those for which estimates have been made in this study are marked with an asterisk. The estimates in this paper under "Direct Costs" cover 82 per cent of total recurrent expenditure on health services as reported in the AIHW Health Expenditure Bulletin No.7[16]. The areas of recurrent health expenditure that make up the 18 per cent not yet included are research (1.4 per cent), administration (3.5 per cent), health promotion and illness prevention (1.2 per cent), aids and appliances (2.0 per cent), community health services (2.9 per cent), ambulance services (1.5 per cent), public psychiatric institutions (3.6 per cent) and other (2.4 per cent).

TABLE 5: COST CATEGORIES INCLUDED IN DISEASE COSTING[1]

TYPE OF COST	SOURCE
(A) DIRECT COSTS	**Health Services** - hospital inpatient* and outpatient costs * - pharmaceutical costs * - medical services (general practitioners and specialists) * - ambulance costs - nursing home costs * - allied professional services * **Patient costs** - out-of-pocket medical, pharmaceutical and hospital costs * - direct travel costs **Carer costs**
(B) INDIRECT COSTS	**Morbidity costs** - include costs to industry (worker absenteeism *[2], staff turnover, reduced worker productivity) **Mortality costs** - foregone earnings due to premature death * **Other indirect costs** - costs of road traffic accidents due to alcohol - legal and police costs due to alcohol
(C) INTANGIBLE COSTS	**Pain, suffering, anxiety and reductions in quality of life**

Notes:

(1) Costs marked with an asterisk (*) have been included in the current study.
(2) Includes only those absenteeism costs associated with a visit to a medical practitioner or a stay in hospital.

Summary of disease costs attributable to diet

A summary of the direct costs of health care attributable to diet in 1989-90 is presented in Table 6. Estimates are presented for each of the high, medium and low population attributable fractions. Table 7 provides a summary of the total costs measured in this study (refer Table 5) attributable to diet in 1989-90, including both the direct costs of health care, the net present value of foregone earnings (due to premature mortality) and the cost of sick leave. Appendix G provides details on the various components of the disease cost estimates prior to application of the population attributable fractions, while Appendices H, I, and J provide the same detail on the costs attributable to diet.

For those who are interested in what part of current Australian annual expenditure on the care of patients with diet-related diseases is potentially "savable" through health promotion activities centred on nutrition and food policy, Table 6 is the relevant table. Table 6 summarises the direct costs of hospitals, medical expenses, allied professional services, pharmaceutical expenses and nursing homes attributable to diet for each disease.

Caution should, of course, be used in interpreting these estimates. They represent the maximum possible "savings" that health promotion programs could hope to realise. Apart from issues related to the cost and efficacy/effectiveness of individual interventions, there are also various policy and practical limitations on what percentage of this potential could be realised, and how quickly such savings could be achieved through time. These should not be interpreted as estimates of financial cost savings realisable by government in the short term, but rather as approximate estimates based on the economic notion of "opportunity cost". They are probably best conceived of as the cost of resources devoted to the treatment of preventable disease, that could be available for the treatment of non preventable disease.

The notion of "opportunity costs" is essentially one of benefits foregone, and conversion into dollar cost estimates would necessarily introduce a number of difficult and quite complex considerations (economic, financial, political, and practical) beyond the scope of this paper.

TABLE 6: SUMMARY OF DIRECT HEALTH CARE COSTS ATTRIBUTABLE TO DIET IN AUSTRALIA, 1989-90

	High Estimate ($M)	Middle Estimate ($M)	Low Estimate ($M)
Coronary heart disease	291	194	97
Hypertension	414	276	138
Atherosclerosis	16	11	5
Stroke	267	178	89
Diabetes mellitus (non-insulin dependent)	249	166	83
Cancers			
- Stomach		12	4
- Colon	191	16	7
- Rectum	(All)	12	5
- Breast		17	6
- Endometrium		3	1
Osteoporosis	74	50	25
Diverticular disease	34	23	11
Hemorrhoids	41	27	14
Dental Caries	712	475	255
Gallbladder disease	71	47	24
Constipation	10	6	3
Iron deficiency anemia	11	7	4
SUB TOTAL	2,381	1,520	771
Alcohol-related diseases	470	470	470
TOTAL	**2,851**	**1,990**	**1,241**

Note: Figures rounded to the nearest $ million.

TABLE 7: SUMMARY OF TOTAL COSTS ATTRIBUTABLE TO DIET IN AUSTRALIA, 1989-90

	High Estimate ($M)	Middle Estimate ($M)	Low Estimate ($M)
Coronary heart disease	710	474	237
Hypertension	546	364	182
Atherosclerosis	20	14	7
Stroke	404	270	135
Diabetes mellitus (non-insulin dependent)	372	248	124
Cancers			
- Stomach		35	11
- Colon	735	62	26
- Rectum	(All)	24	10
- Breast		72	24
- Endometrium		5	2
Osteoporosis	86	57	29
Diverticular disease	46	30	15
Hemorrhoids	59	39	20
Dental caries	717	478	257
Gallbladder disease	118	79	39
Constipation	11	8	4
Iron deficiency anemia	13	9	4
SUB TOTAL	3,837	2,267	1,126
Alcohol-related diseases	1,353	1,353	1,353
TOTAL	**5,190**	**3,620**	**2,479**

Notes: Figures rounded to the nearest $ million.

For those who are interested in ranking diet-related diseases in order of importance, careful thought should be given to the basis of the ranking. Table 8 illustrates how the rankings change according to the ranking criterion chosen. The "big three" (CHD, neoplasms, stroke) dominate mortality related indicators but not the diet-related health care cost indicator. Dental caries and hypertension (high medical and pharmaceutical costs) dominate the health care costs ranking.

Disease costing estimates do not provide sufficient information on their own to permit a decision on whether resource allocation is efficient. This can only be done using evaluation techniques that combine both costs and outcomes. The AIHW is currently developing a macro economic evaluation approach that incorporates both disease costing and impact on life expectancy. Illustrative results from this macro economic appraisal work for diet-related interventions are included in Tables 9 and 10.

TABLE 8: TOP SIX DIET-RELATED DISEASES IN AUSTRALIA, 1989-90

RANKED BY PYLL TO AGE 65		RANKED BY TOTAL COSTS		RANKED BY HEALTH CARE COSTS	
	Years		$M		$M
CHD	17,190	Dental caries	478	Dental caries	475
Certain neoplasms	8,433	CHD	474	Hypertension	276
Stroke	6,168	Hypertension	364	CHD	194
Diabetes (NIDDM)	3,476	Stroke	270	Stroke	178
Gallbladder disease	667	Diabetes (NIDDM)	248	Diabetes (NIDDM)	166
Hypertension	569	Certain neoplasms	198	Certain neoplasms	60

Methodology for quantifying the costs

The methodology utilised for quantifying the costs associated with diet-related diseases is explained in detail in Appendix D. The basic approach for direct costs has been to take known aggregate expenditures and apportion those on a disease specific basis. Indirect costs due to premature death have been estimated using the human capital approach. The advantages and disadvantages of this approach are explained in Appendices D and E. The costs due to sick leave associated with disease morbidity are estimated from doctor visits, hospital beddays and recuperation time. Absenteeism due to sick leave not involving a medical practitioner has not been included.

For non-insulin dependent diabetes (NIDDM), Australian and overseas studies have indicated that the cost of complications are significant. To estimate the population attributable fractions for hospital costs associated with chronic complications the United States estimates by Huse et. al.[17] have been used. These are outlined in Appendix C. As an example, for males less than 65 years of age, two per cent of hospital related admissions for hypertension are directly related to NIDDM. These attributable fractions are also assumed to be applicable to medical costs and nursing home costs.

An average cost per hospital separation has been used to calculate the high estimate of diet-related cancer rather than the weighted DRG approach utilised for all other diet-related disease costs (refer Appendix D). This was necessary because the high PAF estimate was expressed as a percentage of all cancers, rather than in relation to specific cancers. Thus to obtain the high estimate of cancer related hospital inpatient costs, the total number of cancer separations is multiplied by the average cost per separation and population attributable fraction of 0.35.

For dental caries, the methodology detailed in Appendix C covers only medical and hospital costs, and not the costs of dentists per se. Therefore for non hospital/medical dental costs, the information published in the AIHW Australian Health Expenditure Information Bulletin[16] on total dental services was used. To apportion that amount of total dental services expenditure that relates to dental caries, a value of 75% of total expenditure is used (Spencer, personal communication).

An estimate of the portion of total dental services expenditure that relates to dental caries has been derived from the 1988 Longitudinal Study of Labourforce Participation and Productivity of Dentists in Australia[18]. A weighted random sample of dentists in general private practice across Australia yielded information from 1133 dentists on the services they provide on a typical day.

The mix of services was dominated by Restorative (31 per cent), Diagnostic (29 per cent) and Preventive (19 per cent) services. Those services directly identified as related to the treatment of sequelae of dental caries amounted to 32 per cent, while other specifically identified areas amounted to approximately three per cent. On the basis that 70 per cent of non-specific services would be related to the diagnosis, prevention or treatment of dental caries, a further 46 per cent is added, thus resulting in a total share of services related to dental caries of 78 per cent. This share of dental services may be a little higher than the corresponding share of dental expenditure. An estimate of 75 per cent has been used in calculating the dental services expenditure related to dental caries.

Scope and limitations

This section summarises some selected issues of scope and interpretation for cost-of-illness studies. It is not possible to provide comprehensive advice on this issue in the current paper. Several other reports do provide such information[19,20,21,22,23]. A central issue to be aware of in any cost-of-illness study

is what costs are included or excluded. Table 5 identified the range of costs included in this paper (refer page 16).

Scope of the costing

One important economic cost not included in our current estimates, is the cost which families and friends incur as a result of undertaking the caregiving function for chronically ill and handicapped people. These costs are "incremental costs" because they are in addition to those which would have been incurred in the absence of the condition. Costs can be direct expenditures (money outlays for home costs on recurring items, travel costs related to patient condition, costs for durable equipment and home renovation) or indirect, including costs resulting from lost opportunity for the caregivers.

The elderly have a very heavy reliance on family and community care services. A study which followed-up 125 patients over the age of 75 years in a Sydney hospital showed that at three months after discharge from hospital only 34 per cent of men and 17 per cent of women were fully independent. Eighty eight per cent of patients were in daily contact with family carers who were providing for many of their elderly relatives' needs[24].

These costs are important and need to be considered in a comprehensive economic study. If families cannot afford to provide home care for relatives, increased institutionalisation may result with consequent increase in the direct costs to government. Conversely, government cut-backs for institutionalised care will result in cost shifting back to family carers or private institutions.

A further category of cost often not included in cost-of-illness studies is the personal costs incurred by the patient and family in being diagnosed and treated for disease. Such costs may include the Medicare gap, travel costs, costs associated with taking unpaid leave from work and the opportunity costs of the time spent travelling to and from the place of consultation. The current study includes the personal financial cost associated with hospitals, medical services and pharmaceuticals, but does not include the opportunity cost of travelling and waiting time, or travel expenses. Such costs are not trivial. For example, a 1988 study estimated personal costs to women attending a mammographic screening program in Australia to be in the order of $20 per attendance[25].

The important cost category of "intangible costs" (pain, suffering, anxiety) or quality of life impacts, has not been costed in this study. This is an important issue for economic analysis, which is not primarily concerned with dollars, but rather with total community welfare. Measuring and valuing the quality of life impact of disease is as an important area for research in cost-of-illness studies, as it is for economic evaluation. The use of "willingness-to-pay" approaches, which have a strong grounding in welfare economics, have particular attraction in this regard (refer Appendix E).

The indirect costs so far measured for both diet and alcohol-related diseases include foregone earnings due to premature death and sick leave involving a

visit to a medical practitioner or hospital. The costs of absenteeism not involving a medical practitioner, of diminished productivity and other non-health sector costs (e.g. from road accidents) are not included. For alcohol-related diseases these as yet uncosted elements are likely to be quite substantial. These cost elements are also very difficult to measure with any certainty, and estimates have yet to be completed.

Incidence-based versus prevalence-based costings

In the now extensive literature on cost-of-illness methodology, two general approaches can be identified: prevalence based approaches and incidence based approaches. The choice between the two is a major issue of study design. The current study is based on a prevalence cost approach.

Prevalence based costs provide an estimate of the direct and indirect economic burden incurred in a period of time (the 'base period') as a result of the prevalence of disease during this same base period, usually a year. Included are the costs incurred in the base year of manifestations or sequelae of disease that may have had its onset in the base year or at any time prior to the base year. Prevalence based costing involves:

- the measurement of all health care costs (such as medical diagnosis and treatment) and productivity losses (such as absenteeism) due to sickness that accrued during a given year to persons suffering from that condition; and

- estimates of the net present value of lost expected future earnings of persons who died of the condition in that year.

Major overseas studies of disease costs using the prevalence approach have been undertaken by Cooper and Rice[26], and Hodgson and Kopstein[27]. In Australia, studies have been undertaken on coronary heart disease by Gross[28] and on drug abuse by Collins and Lapsley[29].

The incidence approach also has health care cost and lost earnings components, but requires that these costs be measured from the time of onset of the disease until cure/death, which for many diseases is a number of years later. Only cases which commence in the base year are included, and the costs incurred in subsequent years are discounted so they may be added to the costs in the given year.

Incidence costs are difficult to estimate because they require knowledge of the likely course of a disease and its duration, including survival rates since onset; medical care that will be used and its cost during the duration of the disease; and the impact of the disease on employment, housekeeping and earnings. These factors often vary greatly even within a specific disease category such as cancer, and will depend on organ site, type of cellular change, and stage of disease development when treatment commences.

Incidence cost studies often suffer from limitations of data and knowledge, but have been enhanced by the DRG developments for the hospital cost component of direct costs. Incidence studies require agreement about alternative clinical event profiles and the associated resource utilisation profiles.

Despite its greater difficulty, the incidence approach is more appropriate for analyses that seek to measure the savings or benefits of preventing a new case of disease. The incidence-based information is also necessary to determine the reduction in health costs which would result from incremental changes in conditions that lower the incidence or ameliorate the severity of disease.

Estimates based on the prevalence of disease can inflate costs because they include the continuing costs of treatment for persons with established disease who are unlikely to benefit from a primary or secondary prevention program. On the other hand, prevalence estimates may understate potential savings to the extent that new cases prevented involve long episodes of care that extend beyond one year.

The estimation of cost based on incidence rather than prevalence establishes a more appropriate ceiling against which health initiatives to prevent disease can be assessed. In practice, however, the extra information required by the incidence approach limits its utility. Sensible interpretation of prevalence studies, often combined with some simplifying assumptions, can also increase their usefulness for a broader range of purposes.

In summary, both the prevalence and incidence approaches have advantages and disadvantages. The prevalence approach is easier to implement because it largely relies on existing data bases, but there are greater limitations on inferences that can be drawn from it for health prevention and policy purposes. Both approaches can sensibly be used to estimate the financial burden-of-illness, rank alternative interventions, or estimate potential cost savings, providing the assumptions on which they are based are kept firmly in mind, and the results interpreted accordingly.

The Problem of Double Counting

Taxes and transfer payments, such as welfare and unemployment benefits are not included in costs-of-illness studies. They do not represent costs to the community as a whole since they do not involve resource usage[30]. They merely represent transfers of income from one section of the community (the taxpayers) to another (e.g. the unemployed or disabled). That is, the net cost to society in terms of resources used (and thus unavailable for other alternatives) is zero.

A second example of double counting to be avoided relates to disease overlapping. For example, in costing non insulin dependent diabetes and its sequelae, the cost of coronary heart disease that results as a direct long term complication of diabetes will be included. If estimates are made of the total cost to the economy of both diabetes and coronary heart disease the cost of diabetic associated coronary heart disease must be subtracted from the final total costs of

the two diseases combined. This paper overcomes this particular problem by apportioning the extent of total coronary heart disease that is due to diet-related behaviour and also apportioning that extent of coronary heart disease that is attributable to the longer term complications of non-insulin dependent diabetes. Thus it is possible to add the two associated costs.

Eliminating a Cause of Death

Calculation of the gains in life expectancy due to the "elimination" of a cause of death or risk factor is a theoretical exercise, because the main causes of death in the developed world cannot be eliminated. Cardiovascular disease and cancer are more or less normal causes of death that can at the most only be partially eliminated. The purpose of the PYLL calculations is to visualise the potential impact of prevention programs, thereby incorporating an approximate estimate of the replacement of one cause of death by another.

It makes an important difference in such calculations whether the probabilities of dying of certain diseases are "independent" or not (ie whether the mortality rates of one disease are unaffected by changes in the mortality rates of other diseases). Although it is quite possible that individuals who run a high risk of developing cancer, also run an increased risk of developing cardiovascular disease, calculation of the increase in PYLL due to eliminating a cause of death is usually based on an assumption of independent risks. That assumption has been used in this study.

If the probability of dying of one disease is positively associated with the probability of dying of another, however, (ie competing death risks) then the elimination of the first disease will increase the mortality (and morbidity) rate from the other. On the other hand, different assumptions about the dependency between causes of death (through common risk factors or otherwise) may lead to better results from the elimination of one cause of death than in the case of independent causes. The literature is not clear on the notions of "substituting" and "competing" causes of death, and adequate models to predict their effects are still being developed.

It also makes a difference whether the gain in life expectancy is calculated for the total population or only that proportion of the population that is "saved" by eliminating that cause of death, ie those who will not die of that specific cause. Since our mortality pattern is dominated by typical old-age causes, such as cardiovascular disease and cancer, the elimination of one cause will quickly lead to an exponential increase in the risks for other causes, and the gain in life expectancy for the total population may be quite limited. This does not apply for the saved population, however, especially when a less common cause of death or a cause that frequently occurs at a younger age is eliminated. The PYLL calculations in this report are for the "saved" population.

4 COMPARISONS WITH PREVIOUS STUDIES

Previous Estimates of the Cost of Diet-Related Disease

Whilst there have been numerous studies, both overseas and in Australia, estimating the costs of many of the chronic diseases, little work has been done on assessing the overall cost of diet-related disease. The most comprehensive attempt at quantifying the health related costs of poor diet is that quoted in the 1977 United States Senate Report, "Dietary Goals of the United States"[31], which estimated that improved nutrition might reduce that nation's health bill by one third. This estimate was based upon the assumption that improved diet would result in a reduction of death and morbidity from chronic diseases of the following magnitudes:

Disease	Per cent reduction
Cardiovascular disease	25
Non-insulin dependent diabetes	50
Obesity	80
Alcoholism	33
Osteoporosis	75
Cancer	20
Renal disease	20

These figures were based upon the best estimates then available of the degree to which diet directly causes disease.

The "Health For All Australians"[3] report updated the Nutrition Taskforce of the Better Health Committee's[1] estimate of $5 million in 1984 to $6 billion in 1988. These estimates were extrapolated from United States data. The results of the present study for the diet-related costs of health care are approximately half those of this early estimate. The current estimates are based on Australian primary data bases (refer Appendix D) and current epidemiological knowledge, and are the most rigorous currently available for this country.

It is also possible to account for some of the difference between the previous Australian estimates and those given in this paper. Firstly, in comparing costs between countries the different per unit health care costs must be taken into account, as well as the differing estimates for incidence and prevalence of the diseases under study. The USA undoubtedly experiences higher per unit health costs than Australia. This may account for some of the difference.

Secondly, our estimate of the health care costs of diet-related diseases should be considered conservative for a number of reasons. Not all diseases that have a possible nutrition element in their etiology have been costed. Obesity, for example, is associated with a number of conditions that have not been costed in this study. These include increased post anesthetic complications, compromised pulmonary function, complications during pregnancy, a range of bone and joint

disorders exacerbated by increased weight, and a number of cancers (prostate, endometrial, cervical, ovarian, and gallbladder). In addition, the two major eating disorders, anorexia nervosa and bulimia, whilst increasingly recognised as serious psychiatric conditions with physiological consequences, could be considered under a diet-related cost-of-illness study.

Further, the number of hospital cases costed for those diseases included in this study are based on primary diagnoses only. This would under-estimate diseases that present as significant secondary diagnoses. Sometimes these secondary effects can be quite subtle. Patients with non-insulin dependent diabetes undergoing any surgery, for example, would have their blood glucose levels monitored, a cost which is attributable to their diabetes rather than to the nominated surgery.

Finally, while all major expenditure categories have been costed (hospitals, medical services, nursing homes, pharmaceuticals and allied professionals), a number of minor categories have not. These comprise research, health promotion, aids and appliances, community health services, psychiatric institutions and ambulance services, which together constituted 18% of total recurrent health expenditure in 1989-90.

Alcohol Related Disease

Two recent Australian studies have been undertaken on the economic costs of alcohol-related disease. The first, by Crowley and Richardson[32], extrapolated from overseas studies to the Australian context. Their estimates were in the range of $6.7 billion to $17.9 billion per year depending on the assumptions made and the country of study. This range of results illustrates the dangers inherent in simple extrapolation of overseas studies to Australia or not carefully considering underlying assumptions.

The second study, by Collins and Lapsley[29], concluded that alcohol abuse cost the Australian economy approximately $6 billion annually. This study included a broader range of direct and indirect costs than those considered in this present report, such as the police and legal costs of accidents, and "expenditure on alcohol by abusers net of taxes and subsidies". A comparison of common cost components, between this current study and that of Collins and Lapsley, yields comparable estimates where methodologies are similar - $470 million versus $581 million, respectively, for health care costs; and $169 million versus $229 million, respectively for sick leave involving a medical attendance/hospital stay. The methodologies for costing foregone earnings are not sufficiently similar to be able to compare results.

Neither the study by Collins and Lapsley nor the work reported here included the costs to industry through absenteeism not involving a medical practitioner, or reduced productivity, which have been estimated in overseas studies to contribute to between 50 and 80 per cent of all alcohol-related costs. Our current estimate of $1.4 billion for alcohol-related disease would yield estimates of approximately $2.8 to $7.0 billion if prorated up for those as yet uncosted industry effects.

Cost Studies of Individual Diseases

Coronary Heart Disease

A few cost studies of individual disease conditions have been undertaken in Australia. For example, it has been estimated that the cost to Australia of cardiovascular disease was of the order of $1,500-$2,000 million in 1984[28]. A breakdown of these costs included hospital, medical and pharmaceutical treatment costs ($600 million) and costs associated with loss of economic output attributable to illness and premature death ($900-$1,400 million).

These figures compare with the estimate for 1989-90 in the present report of $449 million for the direct costs of coronary heart disease (the major component of cardiovascular disease) and $677 million for the indirect costs. The estimates presented in this current paper are clearly more conservative, but of a similar magnitude.

A further study relating to the costs of cardiovascular disease was undertaken by Goldstein et. al.[33]. This study costed acute myocardial infarcts (AMI) in New South Wales based upon incidence rather than prevalence, the usual costing method used in Australia. This study found that the cost for AMI in 1979 for NSW was $301.0 million, made up of $32.3 million as direct costs and $268.7 million as indirect costs.

Osteoporosis

In 1989, Leeder and Salkeld[34] estimated the direct costs of fracture of the femoral neck, wrist fractures and those associated with vertebral collapse. Estimates were derived from New South Wales and Victorian hospital statistics of the incidence of fracture related to osteoporosis, stratified by age and sex. These figures were extrapolated to the national population using demographic statistics.

The total direct health care costs were estimated to be $172 million for 1988. Included in the direct cost measurement were acute public hospital, and public and private nursing home costs. For hospital stay, an average cost per bed day approach was used (based on the estimate by Mathers and Harvey of $282 in 1988) and this was adjusted for outpatient costs. For public and private nursing home costs a similar approach was used (based on the Mathers and Harvey 1988 cost of $65 per bed day).

The estimate in the present report of $248 million for national health care costs in 1989-90 for osteoporosis and related fractures is of a similar magnitude. This estimate includes $46 million for medical and allied professional services that were not included in the Leeder/Salkeld study.

Lord and Sinnett[35] estimated the extent of inpatient femoral neck fractures in New South Wales for 1981. Although the study did not explicitly look at the associated costs, it provided important data that are necessary to undertake a comprehensive cost-of-illness study. For example, it highlighted that length of stay and bed use increased significantly with age, as did the proportion of patients with femoral neck fractures who were transferred to other hospitals or institutions.

5 ECONOMIC EVALUATION OF NUTRITION INTERVENTION PROGRAMS

Cost-of-illness studies provide an estimate of the magnitude of the cost burden of disease to the Australian population. Of particular relevance to a National Food and Nutrition Policy is the extent to which nutrition intervention programs can reduce preventable morbidity and premature mortality and thus lead to a reduction in these costs.

This section of the paper reviews evidence regarding the potential reductions in diet-related chronic diseases that could be reasonably expected if the Australian population changed its food habits in line with the Australian Dietary Guidelines and Nutrition Goals and Targets.

Achievement of nutrition goals and targets

As mentioned earlier (refer page 7), the maximum possible potential reductions in disease that could occur in a population is that estimated by the population attributable fraction. The population attributable fraction, for a particular risk factor, is the proportion of disease that could be eliminated if the total risk factor was eliminated in a population.

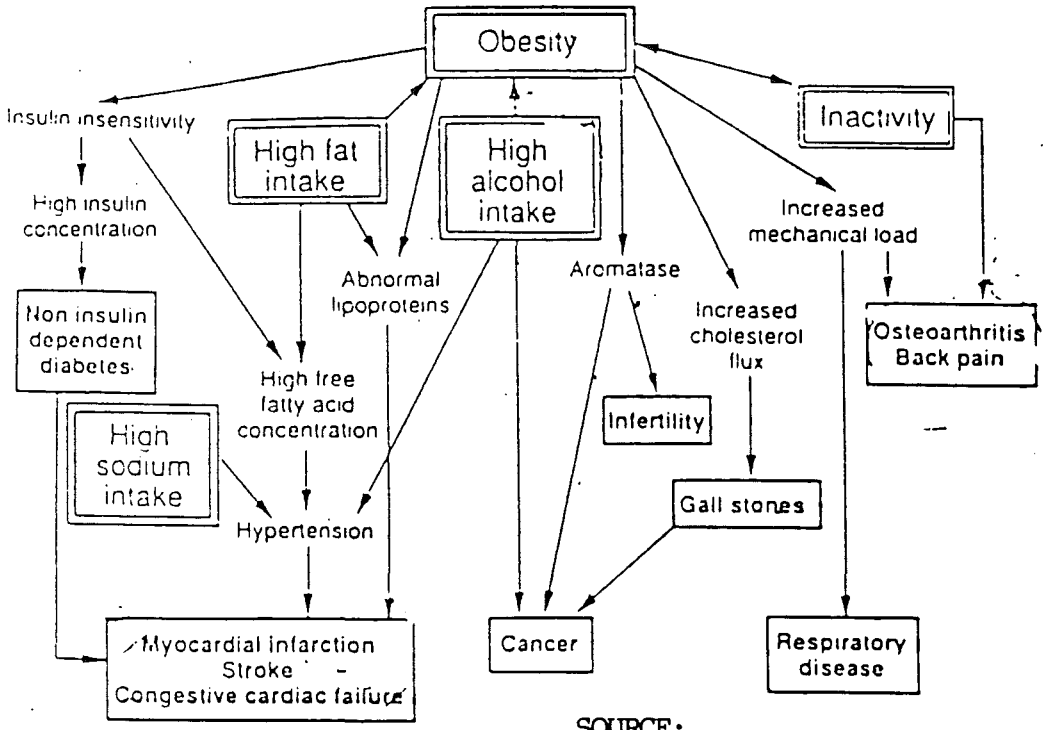
Total elimination of major risk factors is unlikely to occur with diet-related disease, particularly in the short and medium term. Estimation of what might be achieved in reality requires:

- a knowledge of the causal factors and the extent to which some increment in exposure causes an increase in the rate of disease, i.e. including figures for relative risk that are precise and generalisable to the whole Australian population and its age structure;
- a knowledge of the extent of present exposure of the population to the relevant risk factor;
- the extent to which the present exposure might be plausibly reduced and is capable of reversing the effects of previous exposure as well as the length of time necessary for this to occur[14]; and
- the effect of the reduction in disease incidence on health care utilisation and practice patterns, and subsequent health care costs.

Estimation of the reduction in exposure to risk factors that might be achieved in reality has been termed "actual" preventability or the "impact fraction". It is the proportion by which the incidence of the disease might be reduced by a credible change in known risk factors.

It is difficult to predict precisely the effects of the Australian population achieving the nutrition targets by the year 2000. For example, obesity is associated with the development of many chronic diseases, but the exact proportion of these diseases attributable to diet is uncertain. Figure 2 depicts the influence of obesity on chronic diseases.

FIGURE 2: INFLUENCE OF OBESITY ON CHRONIC DISEASE



Effect of obesity and activities leading to obesity on important causes of death and illness

SOURCE:

Garrow, J. Importance of obesity

BMJ 1991:303:704-6

Some very crude estimates for the effect of obesity on non-insulin dependent diabetes can be made. For example, assume the target for obesity (body mass index (BMI) > 30) is to reduce its prevalence by approximately 20 per cent over the next ten years. Colditz[37] has estimated that the relative risk of diabetes for obese women aged 30 to 64 years is approximately 17 times greater than that for women of acceptable weight or lower (BMI <= 25). The current prevalence of obesity in Australia for women aged 30 to 64 years is approximately 12 per cent. Reducing the prevalence to 10 per cent would reduce the PAF% from 66 per cent to 60 per cent (refer page 7). Thus, reducing obesity in women by 20 per cent could achieve an 6 per cent reduction in the incidence of non-insulin dependent diabetes in Australia.

This does not mean that the net economic benefit to the community is 6 per cent of the current direct costs of non-insulin dependent diabetes. The costs of achieving such benefits (e.g. costs of screening, education campaigns) over time must be netted out and there are various economic, political and practical realities that would affect how potential savings could be realised.

Another goal of the National Better Health Program is the reduction in the contribution of fat to dietary energy intake from 38 per cent to 33 per cent or less by the year 2000[2]. Again, it is difficult to predict the extent of the impact of such a reduction on chronic disease. The relationship between diet, serum cholesterol and later development of coronary heart disease is not fully understood. There is conflicting evidence about the extent to which lowering fat in the diet will reduce serum cholesterol levels, despite the fact that the general consensus from primary intervention trials is that for every 1 per cent reduction in cholesterol levels there is a reduction in the risk of coronary heart disease of 1 to 2 per cent.

Most large primary prevention trials utilise multiple risk factor interventions, and only one study, the Los Angeles Veterans Administration Domiciliary Study, fully evaluated the effect of a change in diet alone. This study showed a 14 per cent reduction in cholesterol in the intervention group versus the control group. In addition, the intervention group's diet was more restrictive than that set out in the Nutrition Goals and Targets. The control group ate a typical North American diet (e.g. 40% energy as fat). The intervention group diet contained reduced fat to less than 20% of energy, half the dietary cholesterol intake level of the controls (i.e. <200mg/day) and a high polyunsaturated/saturated fat ratio of 1.5. Extrapolating from this study to the context in Australia is thus difficult.

Controlled intervention trials which advocated diets similar to that of the nutrition goals and targets have been shown to be less successful in reducing cholesterol levels. For example, the North Karelia trial and the Stanford Five City trials, both mass education approaches, showed more modest reductions in serum cholesterol levels - with 2 per cent reduction in 10 years and 0.6 per cent over 5 years respectively. These results have led some researchers to believe that practical population dietary fat modifications in line with the Nutrition Goals and Targets may only produce a 1 to 2 per cent reduction in cholesterol levels, leading to a 2 to 3 per cent reduction in coronary heart disease events. To produce much larger reductions would require more restrictive changes and diets which

have not been tested in long-term controlled trials in the general population. Further, even if such changes in diet were achievable, they may not lead to the results expected because of the "competition in causes of death" explained earlier (refer page 25) that may influence the impact of prevention programs.

The evidence of reduced incidence of chronic disease with salt reduction is more promising. The Australian NHMRC Dietary Salt Study[38] showed that dietary modification to reduce sodium intake does lower systolic and diastolic blood pressure, and that these effects are specifically attributable to the reduction in salt intake. The low sodium group (80 mmol sodium/day) had mean systolic and diastolic blood pressure falls of 6.1 and 3.7 mm Hg respectively over the eight week intervention phase. The normal sodium group had falls of 0.6 and 0.9 for systolic and diastolic blood pressure respectively. Multivariate analysis taking into account the effects of pre-diet blood pressure, weight and age, reduced the effect from 5.5 to 4.8 for systolic blood pressure, but the effect on diastolic blood pressure was not changed significantly. A change in systolic blood pressure of 1.5 mm Hg was found for every kg lost.

Grobbee and Hofman[39] reviewed 13 sodium studies and also came to the conclusion that dietary sodium restriction is associated with a small hypotensive effect. This effect is greater for systolic blood pressure and is directly related to age and initial blood pressure.

Law et. al.[40] analysed data taken from published reports of blood pressure and sodium intake for 24 different communities throughout the world. They measured the difference in blood pressure due to a 100 mmol/day reduction in sodium intake and reported similar conclusions. They point out that the association of blood pressure with sodium intake is substantially larger than is generally appreciated and increases with age and initial blood pressure. The following shows the mean differences in systolic and diastolic blood pressure for a reduction in 100 mmol sodium intake per day according to age:

Age	Systolic	Diastolic
15 - 19	5.0	1.8
20 - 29	4.9	2.6
30 - 39	5.5	3.0
40 - 49	6.6	3.6
50 - 59	9.2	4.7
60 - 69	10.3	4.3

The same authors determined the impact of reducing dietary salt on mortality from stroke and ischemic heart disease. Simple dietary manipulation, that is avoiding salty foods and not adding salt in cooking or at the table, reduces sodium intake by approximately 50 mmol (3 g) per day. In people aged 50 - 59 this would, after a few weeks, lower systolic blood pressure by an average 5 mm Hg, and 7 mm Hg in those with high blood pressure (170 mm Hg). Diastolic blood pressure would be lowered by half as much.

This reduction in blood pressureis estimated to result in a reduction in the incidence of stroke by 26 % and of ischemic heart disease by 15 % if such a reduction of salt could be achieved by a whole Western population. Law et. al. concluded that the reductions in mortality would be larger than those achieved by drugs. For example, lowering sodium intake by 100 mmol/day (avoiding many processed foods and manufacturers not adding salt to their food product) would achieve twice the blood pressure fall and would prevent some 70,000 deaths a year in Britain. If these studies are generalisable to the Australian population the reduction in the economic costs of hypertension and stroke brought about by primary intervention programs may be quite significant.

The current average sodium intake of the Australian population is not known with certainty. The Better Health Committee[2] cites a value of 165 mmol per day as the latest data available and have set a target of 100 mmol per day by the turn of the century. If these reductions are achieved by the general population only time will tell whether the reductions in coronary heart disease and stroke suggested by Law et. al. are possible. Even if these reductions do occur it will be difficult to ascertain whether they are a direct result of salt restriction or due to other factors.

Cost-effectiveness studies of diet-related interventions

An Australian study by Hall et. al.[3] used the results of a number of international intervention trials[41] aimed at reducing ischemic heart disease, to calculate the cost-effectiveness of a series of alternative preventive strategies. This study has been utilised by the AIHW as part of its Macro Economic Evaluation Project.

The AIHW analysis examines the cost-effectiveness of four of the five interventions that had relevance to diet. All interventions were designed to prevent coronary heart disease (CHD) by modifying the risk factors known to be associated with this condition. The risk factors are obesity, smoking, lack of exercise, high blood pressure and high blood cholesterol levels. The AIHW cost estimates for the interventions and the effectiveness rates are based on those used by Hall et. al. The costs of the interventions for the initial year only have been inflated from 1986 prices to 1991 prices and expanded to a national basis for the target group of males aged 40-59 years. Costs used are those for the medium range for the media campaign and Medicare Benefit Schedule fees for screening and medical counselling interventions.

The estimate for reductions in cost-of-illness (which in this study is limited to savings in direct health care costs) has been extracted from the data being generated for cost-of-illness by the AIHW Macro Economic Evaluation Project. The reductions in cost-of-illness attributable to the interventions differ from the study by Hall et. al.[3] in that they are estimated on a national basis and include hospital, medical, pharmaceutical and nursing home costs for CHD (Hall et. al. estimated savings in health care costs for hospitals only).

Whilst the interventions examined address multiple risk factor modification, dietary modification is a major contributor to reducing the risk factors of high

blood pressure, elevated blood cholesterol and obesity. Although the effectiveness rates of the interventions could not be attributed wholly to dietary modification, the results are nonetheless indicative of the upper range of effectiveness of preventive interventions addressing CHD which have an important dietary component. The AIHW study maintains the simplifying assumption by Hall et. al. that the number of myocardial infarctions prevented would be distributed evenly over the five year period; (and therefore applies the same effectiveness ratio as the Hall et. al. study for the one year 1990-91).

The four interventions which are evaluated target whole populations, high risk individuals and high risk groups. They attempt to reduce either the average level of risk of the general population, or to identify by population screening individuals and groups at high risk and to reduce their level of risk. The four interventions (as per the Hall et. al. study) are:

Strategy 1 - Whole population approach

Media campaigns are used to encourage the target audience to make behavioural changes which will modify risk factors. Examples of such campaigns in Australia are the North Coast Healthy Life-style Programme[42] and the "Quit for Life" campaign[43].

Strategy 2 - Identification of High Risk Individuals

The population is screened for risk factors relevant to CHD, such as high blood cholesterol levels, in order to identify the individuals who are at high risk. The target population consists of those who are in the top 15 per cent of the distribution for obesity, smoking, lack of exercise, high blood pressure and high cholesterol. Fifteen per cent of individuals in the population would receive long term counselling, consisting of 2.5 long consultations with a general practitioner in the first year and one standard length consultation in each subsequent year. Advice would be able to be tailored individually.

Strategy 3 - Combined Approach

Advice on risk factor modification is offered to the total population. In addition, screening for risk factors identifies a high risk group requiring individual, long term counselling as in Strategy 2. It is assumed that this requires an initial examination and counselling in a long consultation with a general practitioner. Follow-up is as for Strategy 2.

Strategy 4 - Identification of High Risk Groups

Groups at high risk may be identified according to what is known of the epidemiology of CHD rather than by screening of individuals. Advancing age, low socio-economic status and a family history of CHD are known to be associated with an increased level of risk. Identification of persons belonging to high risk groups may be established at routine consultations with their general practitioners. Persons in this group (epidemiologically high risk) may then be offered individual counselling.

Results

Table 9 summarises the costs and effectiveness rates for each intervention. The media campaign is the least expensive, followed by the identification of high risk groups by general practitioners, then screening for high risk individuals and lastly the combined approach.

Table 10 shows the cost estimates for the interventions when applied nationally, the potential reduction in direct health sector costs attributable to the intervention, the number of cases prevented by the intervention and the macro model cost-effectiveness indices (in this instance using cases prevented rather than life years saved as the outcome measure).

In terms of the number of cases of myocardial infarction prevented, the most effective strategy is the combined approach, which is predicted to prevent 10,945 cases on a national basis. The media campaign is the next most effective strategy, predicted to prevent 8303 cases, followed by high risk individuals (5315 cases) and finally high risk groups (4246 cases).

In terms of costs and cost-effectiveness measures; the media campaign is the only strategy which is estimated to produce a net saving in health care costs of $480 per case prevented; other strategies are estimated to result in a cost per case prevented ranging from $11,790 to $20,500. Strategy 3, the combined approach, is estimated to be the next most cost-effective with a cost per case prevented of $11,790 and the highest number of cases prevented (10,945). Strategy 2 (screening high risk individuals) is estimated to be the least cost-effective at $20,500 per case saved but is estimated to save more cases (5315) than Strategy 4 (screening high risk groups).

Strategy 1, the media campaign,is estimated to produce net health care savings and the second highest number of predicted cases prevented (8303) and on this basis must be considered the preferable strategy on the basis of economic analysis.

There remains, however, the question of whether whole population strategies are sufficient, given that the number of cases prevented remains a small proportion of the total number of myocardial infarctions occurring. The combined approach is the most cost-effective of the screening strategies and also the most effective in terms of the number of cases prevented.

Table 11 summarises the results of the Hall et. al. study, price adjusted and applied to a national target population. The cost-effectiveness ranking by strategy is the same in both studies, with the media campaign showing net savings in both. The "cost savings" estimates in the macro model results differ from comparable Hall et. al. study estimates, but not substantially. The difference might well be due to the limited time scale of our analysis (1 year versus the Hall et. al. 5 years) and the impact of discounting.

TABLE 9: POTENTIAL COST AND EFFECTIVENESS RATES FOR DIET RELATED INTERVENTIONS

STRATEGY	COST(1) ($'000)	EFFECTIVENESS RATE (i.e. cases prevented)(2)
1. Whole Population (Media Campaign)	7703	10.0%
2. High Risk Individuals Identified	116,428	6.4%
3. Combined Approach of 1. and 2.	144,468	13.2%
4. High Risk Groups Identified	75,095	5.1%

Notes:

(1) Costs in $1990-91, on a national basis. Costs of interventions and effectiveness rates adapted from Hall, J. et. al.[3]

(2) Effectiveness rates for these interventions have been modelled on the basis of a population of 500,000 people, with a target group of all males aged 40-59 years (n=60,000). Estimates of benefits of risk factor reduction are based on the findings of the large European Multifactoral Prevention Trial. Effectiveness rates measure the proportion of cases prevented by the intervention, of the number of cases of myocardial infarction which would have otherwise occurred.

TABLE 10: THE MACRO COST-EFFECTIVENESS INDICES (Indicative only)
CORONARY HEART DISEASE HEALTH PROMOTION STRATEGIES

Components of index	INTERVENTIONS			
	Strategy 1 Whole Population	Strategy 2 High Risk Individuals	Strategy 3 Combined Approach of 1 and 2	Strategy 4 High Risk Groups Identified
A. Cost of intervention	7,703	116,428	144,468	75,095
B. Reductions in the cost-of-illness attributable to the intervention	11,669	7,468	15,403	5,951
C. Number of cases prevented by the intervention	8,303	5,315	10,945	4,246
D. Macro cost-effectiveness index	Net saving of 3,966			
$\frac{[A - B]}{[C]}$	Saving per case prevented = 0.48	Cost per case prevented = 20.50	Cost per case prevented = 11.79	Cost per case prevented = 16.28

Note: Costs in $'000 (1990-91). Costs and effectiveness rates adapted from Hall J et. al.[3] (refer Table 9).

Strategy 1 is estimated to result in a net saving and effectiveness is measured as "savings" per case prevented. Other strategies result in a "cost per case prevented" measure.

TABLE 11: COST-EFFECTIVENESS INDICES OF STRATEGIES FOR THE PREVENTION OF CORONARY HEART DISEASE

	STRATEGY			
	1	2	3	4
A. Cost of intervention	7703	116,428	144,468	75,095
B. Savings	11,004	7,042	14,480	5,633
C. Number of cases prevented	8,303	5,315	10,945	4,246
D. Cost per prevented case $\frac{[A - B]}{[C]}$		20.58	11.88	16.35
Net saving	3,301			
Savings per prevented case	0.40			

Source: Adapted from Hall, J. et.al. 1988.

Notes: Costs are expressed in $'000 (1990-91)

Strategy 1 is estimated to result in a net saving, and cost-effectiveness is measured as a "saving per prevented case".

Original costs in $1986 have been price adjusted to $1991 using the total health expenditure deflator and multiplied by a factor of 31.45 to reach a national target group of males 40-59 years [1,887,000]. Hall's original target group of males aged 40-59 years was 60,000 in a population of 500,000.

6.3 Brief overview of AIHW Macro Economic Evaluation Approach

The purpose of this section is to provide a brief outline of the Macro Economic Evaluation Approach being developed by the AIHW.

A prime function of the approach is to assist in prioritising a wide range of interventions. The approach does this by applying the basic principles of economic evaluation to a macro setting. The two basic principles are:

- that both costs and outcomes are identified, measured and valued; and
- that a comparison between alternatives is made.

The principles are embodied in the basic equation (i) that is used to generate an economic index to rank the various projects being considered. In its simplest form the equation is

$$(i) \qquad E_1 = \frac{[\text{cost of project}] - [\text{reductions in cost-of-illness attributable to project}]}{[\text{improvement in life expectancy attributable to project}]}$$

This equation includes both costs and outcomes, and a base case/project case comparison (by measuring the change in life expectancy and the change in costs of health care brought about by a reduction in disease incidence attributable to the project).

In this basic form, the equation yields an index based on cost- effectiveness evaluation methodology, which can be interpreted in the normal manner; i.e. a project with a lower cost per life year is ranked higher than a project with a higher cost per life year. It can also be written as a cost-utility or cost-benefit index:

$$(ii) \qquad E_2 = \frac{[\text{cost of project}] - [\Delta \text{ cost-of-illness}]}{[\Delta \text{ quality adjusted life years saved}]} \qquad \text{(cost/utility)}$$

$$(iii) \qquad E_3 = \frac{[\text{cost of intervention}]}{[\Delta \text{ cost-of-illness}] + [\Delta \text{ life expectancy measured as } \Delta \text{ foregone earnings} + \Delta \text{ productivity}]} \qquad \text{(cost/benefit)}$$

It is important to note that the mortality, morbidity, cost, and economic burden data subsumed in the index, could also be used as information sets in their own right. The advantage of the macro index, is simply that it offers a concise and consistent approach based on economic principles that is reproducible through time and across interventions. It provides a way of combining disparate pieces of information, and making explicit the weightings by which the various inputs are put together. Figure 3 illustrates the components of the model in schematic form.

Two basic tasks are involved in utilising the equations set out above. These are:

- working out the cost and efficacy/effectiveness of individual interventions; and

- working out the costs-of-illness (i.e. the disease costing task) and life expectancy impact of the various diseases.

The steps in applying the formulae for nutrition are:

1. to identify those diseases, in ICD9-CM format, that are nutrition related;

2. to work out the total costs-of-illness and life years lost for those diseases;

3. to work out that part of the incidence of the diseases which is attributable to diet (the population attributable fractions);

4. to work out the cost of the interventions being considered on the assumption of national application; and

5. to work out the impact of the interventions on the prevalence of the diet-related risk factors and then disease incidence, and from this calculate

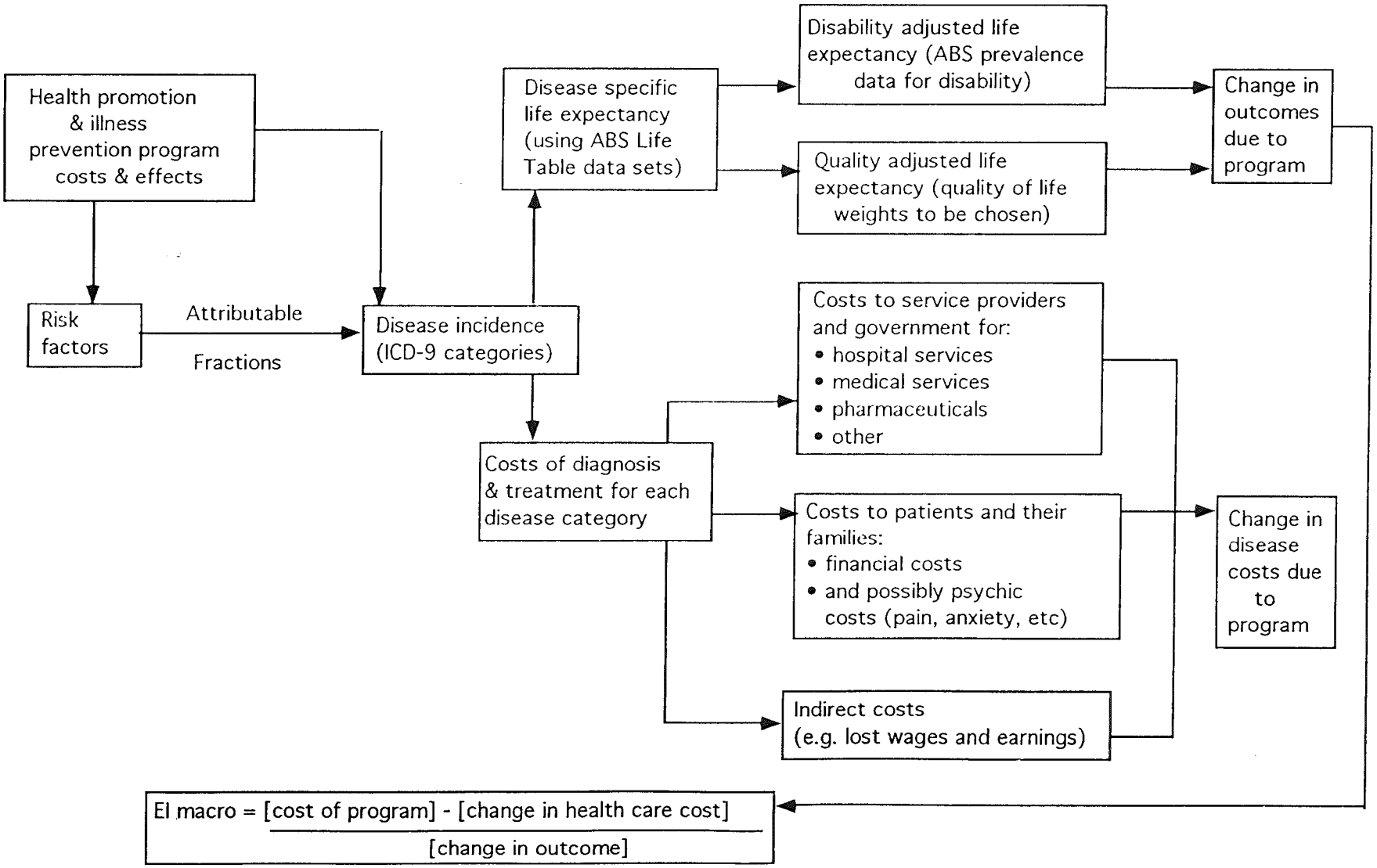
    a. expected increase in life years lived; and

    b. reductions in cost-of-illness achievable by the intervention.

Preliminary results of applying this approach were provided in Table 10. A pilot study involving smoking related interventions is also available.

FIGURE 3

CONCEPTUAL OVERVIEW OF MACRO ECONOMIC EVALUATION



Appendixes

APPENDIX A

NUTRITION GOALS AND TARGETS

NUTRITION TARGETS FOR IMPROVING THE HEALTH OF AUSTRALIANS

	0BJECT1VE	LATEST DATA AVAILABLE	1995	2000
1.	Reduce the prevalence of overweight and obesity in 25-64 years age group	38%	30%	25%
2.	Reduce the contribution of total fat to the total energy of the Australian diet	38% contribution	35% contribution	33% contribution
3.	Reduce the contribution of refined sugars to the total energy of the Australian diet	14% contribution	12% contribution	12% contribution
4.	Increase the dietary fibre content of the Australian diet	17% day content	25g day content	30g day content
5.	Reduce the contribution of alcoholic beverages to the total energy of the Australian diet	6% contribution	5% contribution	5% contribution
6.	Reduce the intake of dietary sodium	165 mmol day intake	130 mmol day intake	100 mmol day intake
7.	Increase prevalence rates for breast-feeding:			
	. at discharge from hospital	88%	95%	95%
	. at 3 months	55%	75%	80%
8.	Increase the percentage of the Australian population using fluoridated water supplies	66%	85%	90%

Source: Health For All Australians Report[2]

APPENDIX B

ETIOLOGICAL FRACTIONS FOR ALCOHOL

Taken from:

The quantification of drug caused mortality in Australia 1989

Prepared by the
Department of Community Services and Health

as an initiative of the
National Campaign Against Drug Abuse

Australian Government Publishing Service
Canberra

3. THE NEW METHODOLOGY

3.1 Alcohol and tobacco

The aetiological fractions developed by Holman and Armstrong (1988) have been used by the Commonwealth Department of Community Services and Health, with some minor modifications as discussed in the previous chapter, on the basis that they represent the latest research findings linking alcohol and tobacco with various morbid conditions.

These replace, to a great extent, the fractions described by L.R.H. Drew in *Technical Information Bulletin no. 69* (1982).

In their report, Holman and Armstrong calculated age-specific aetiological fractions for conditions they identified as being caused or prevented by alcohol and tobacco for each five-year age group from 0–4 to 80–84 and 85+ years. For most conditions, aetiological fractions were derived by the indirect method (Holman & Armstrong 1988, p.18) by which pooled estimates of relative risk were combined with measures of the prevalence of alcohol and tobacco use. For some conditions, lack of published analytical studies necessitated the use of aetiological fractions reported from clinical or other case studies. For conditions identified as having alcohol or tobacco use as a necessary cause (e.g. alcoholic gastritis), a fraction of 1.00 was attributed.

The indirect method

Relative risk (RR) is an epidemiological measure which indicates the strength of the effect of exposure to some external effect (e.g. alcohol or tobacco use) on contracting a particular condition.

$$RR = (IR_e)/(IR_o)$$

where IR_e is the incidence rate of the condition among those exposed to the external effect and IR_o is the incidence rate among those not exposed.

Holman and Armstrong calculated pooled estimates of RR from studies of the relationship between alcohol or tobacco and a particular condition using precision based weighting (Kleinbaum et al., 1982; Rothman 1986). This method assigns greater weight to studies which make more precise estimates of RR, the pooled estimate of RR being a weighted average of the estimates from a pool of studies.

Except for a few conditions, there were insufficient studies which provided data or results within consistently grouped age strata to enable the calculation of pooled estimates of age-specific RRs. However, data on age-specific prevalence of alcohol use and tobacco use were available from the National Heart Foundation *Risk factor prevalence study no. 2, 1983* for alcohol and from Hill D.J. (1988) *Australian patterns of tobacco smoking in 1986* for tobacco, and were combined with the overall pooled estimates of RRs to obtain age-specific aetiological fractions. This method assumes that while exposure prevalence varies with age, the RR for a given level of exposure is not modified by age.

Given a pooled RR for a condition, the age-specific aetiological fraction is computed by:

$$F = [p(RR-1)] / [p(RR-1)+1] \quad (I)$$

where p is prevalence of exposure in age group.

The aetiological fractions used by the NDAIC within these categories are at 3.1.1 to 3.1.4.

3.1.1 Age-specific aetiological fractions, males, alcohol

Cause of death	*ICD-9*	*0–14*	*15–34*	*35–64*	*65+*
Lip cancer	140	–	0.18	0.17	0.17
Oral cancer	141,143–145	–	0.56	0.55	0.54
Pharyngeal cancer	146–149	–	0.66	0.65	0.63
Oesophageal cancer	150	–	0.51	0.49	0.48
Colon cancer	153	–	0.15	0.15	0.14
Rectal cancer	154	–	0.05	0.05	0.05
Hepatic cancer	155	–	0.36	0.35	0.34
Pancreatic cancer	157	–	0.17	0.17	0.16
Laryngeal cancer	161	–	0.57	0.55	0.54
Breast cancer	174,175	–	0.22	0.21	0.20
Pellagra	265.2	–	0.83	0.83	0.83
Alcoholic psychosis	291	1.00	1.00	1.00	1.00
Alcohol dependence	303	1.00	1.00	1.00	1.00
Alcohol abuse	305.0	1.00	1.00	1.00	1.00
Alcohol polyneuropathy	357.5	1.00	1.00	1.00	1.00
Hypertension	401–405	–	0.22	0.21	0.20
Ischaemic heart disease	410–414	–	–0.28	–0.26(a)	–
Alcoholic cardiomyopathy	425.5	1.00	1.00	1.00	1.00
Cardiac dysrhythmias	426–427	–	–0.28	–0.26(a)	–
Heart failure	428–429	–	–0.08	–0.22(a)	–
Stroke	430–438	–	0.15	0.15	0.14
Oesophageal varices	456.0–456	–	0.51	0.51	0.51
Gastro-oesophageal haem.	530.7	–	0.47	0.47	0.47
Alcoholic gastritis	535.3	1.00	1.00	1.00	1.00
Alcoholic liver cirrhosis	571.0–571.3	1.00	1.00	1.00	1.00
Unspecified liver cirrhosis	571.5–571.9	–	(b)	(b)	(b)
Cholelithiasis	574	–	–0.34	–0.32	–0.30
Acute pancreatitis	577.0	–	0.36	0.36	0.36
Chronic pancreatitis	577.1	–	0.70	0.69	0.68
Low birthweight	764,765	0.03	0.03	0.03	0.03
Ethanol toxicity	980.0	–	1.00	1.00	1.00
Methanol toxicity	980.1	–	1.00	1.00	1.00
Road injuries	E810–E819	0.30	0.34	0.25	0.16
Alcoholic beverage poisoning	E860.0	1.00	1.00	1.00	1.00
Other eth/meth poisoning	E860.1,E860.2	–	1.00	1.00	1.00
Fall injuries	E880–E888	–	0.35	0.35	0.35
Fire injuries	E890–E899	–	0.40	0.40	0.40
Drowning	E910	–	0.26	0.26	0.26
Aspiration	E911	–	1.00	1.00	1.00
Machine injuries	E919,E920	–	0.13	0.13	0.13
Suicide	E950–E959	–	0.23	0.23	0.23
Assault	E960,E965,E966,E968,E969	0.51	0.51	0.51	0.51
Child abuse	E967	0.15	0.15	0.15	0.15

(a) For ischaemic heart disease, cardiac dysrhythmias and heart failure the fraction appearing in the 35–64 column should only be applied to 35–60 year olds.

(b) Fractions for this condition are calculated as follows:

Let A = total male deaths in 571.0–571.3
Let B = total male deaths in 571.5–571.9
If A/(A+B) > 0.51 then insert 0.00 as the fraction where (b) appears
If A/(A+B) < 0.51 then insert [0.51(A+B)–A]/B as the fraction where (b) appears

3.1.2 Age-specific aetiological fractions, females, alcohol

Cause of death	*ICD-9*	*0–14*	*15–34*	*35–64*	*65+*
Lip cancer	140	–	0.19	0.18	0.16
Oral cancer	141,143–145	–	0.19	0.17	0.15
Pharyngeal cancer	146–149	–	0.64	0.61	0.58
Oesophageal cancer	150	–	0.48	0.45	0.42
Colon cancer	153	–	0.14	0.13	0.11
Rectal cancer	154	–	0.05	0.04	0.04
Hepatic cancer	155	–	0.34	0.31	0.29
Pancreatic cancer	157	–	0.16	0.14	0.13
Laryngeal cancer	161	–	0.54	0.51	0.48
Breast cancer	174,175	–	0.20	0.18	0.16
Pellagra	265.2	–	0.83	0.83	0.83
Alcoholic psychosis	291	1.00	1.00	1.00	1.00
Alcohol dependence	303	1.00	1.00	1.00	1.00
Alcohol abuse	305.0	1.00	1.00	1.00	1.00
Alcohol polyneuropathy	357.5	1.00	1.00	1.00	1.00
Hypertension	401–405	–	–	–	–
Ischaemic heart disease	410–414	–	–0.25	–0.21(a)	–
Alcoholic cardiomyopathy	425.5	1.00	1.00	1.00	1.00
Cardiac dysrhythmias	426–427	–	–0.25	–0.21(a)	–
Heart failure	428–429	–	–0.08	–0.16(a)	–
Stroke	430–438	–	0.14	0.13	0.11
Oesophageal varices	456.0–456.2	–	0.47	0.47	0.47
Gastro-oesophageal haem.	530.7	–	0.47	0.47	0.47
Alcoholic gastritis	535.3	1.00	1.00	1.00	1.00
Alcoholic liver cirrhosis	571.0–571.3	1.00	1.00	1.00	1.00
Unspecified liver cirrhosis	571.5–571.9	–	(b)	(b)	(b)
Cholelithiasis	574	–	–0.30	–0.26	–0.22
Acute pancreatitis	577.0	–	0.36	0.36	0.36
Chronic pancreatitis	577.1	–	0.68	0.65	0.62
Low birthweight	764,765	0.03	0.03	0.03	0.03
Ethanol toxicity	980.0	–	1.00	1.00	1.00
Methanol toxicity	980.1	–	1.00	1.00	1.00
Road injuries	E810–E819	0.30	0.34	0.25	0.16
Alcoholic beverage poisoning	E860.0	1.00	1.00	1.00	1.00
Other eth/meth poisoning	E860.1,E860.2	–	1.00	1.00	1.00
Fall injuries	E880–E888	–	0.16	0.16	0.16
Fire injuries	E890–E899	–	0.40	0.40	0.40
Drowning	E910	–	0.04	0.04	0.04
Aspiration	E911	–	1.00	1.00	1.00
Machine injuries	E919,E920	–	0.13	0.13	0.13
Suicide	E950–E959	–	0.23	0.23	0.23
Assault	E960,E965,E966,E968,E969	0.51	0.51	0.51	0.51
Child abuse	E967	0.15	0.15	0.15	0.15

(a) For ischaemic heart disease, cardiac dysrhythmias and heart failure the fraction appearing in the 35–64 column should only be applied to 35–60 year olds.

(b) Fractions for this condition are calculated as follows:

Let A = total female deaths in 571.0–571.3
Let B = total female deaths in 571.5–571.9
If A/(A+B) > 0.47 then insert 0.00 as the fraction where (b) appears
If A/(A+B) < 0.47 then insert [0.47(A+B)–A]/B as the fraction where (b) appears

APPENDIX C

EVIDENCE ON POPULATION ATTRIBUTABLE FRACTIONS FOR DIET-RELATED DISEASE

Cancer:

Doll and Peto[44] provide the most frequently quoted estimate of the proportion of cancer directly related to diet. They give 35 per cent as the best estimate of the proportion of cancer deaths in the United States which could be attributed to diet, with acceptable estimates ranging from 10 to 70 per cent. Doll himself in a recent overview of the epidemiological evidence linking diet and cancer admits that this value is indeed a guess[45]. His estimate is based partly on the knowledge that the diet of experimental animals has a major influence on the incidence of cancer and partly on the simplistic belief that what you put into your mouth and pass into or through the digestive tract is likely to play a large part in the production of cancer in the corresponding organs.

A more recent comprehensive review attempting to quantify the relationship between diet and various forms of cancer was undertaken by the WHO International Agency For Research On Cancer (IARC) in Lyon[46]. This publication estimated the theoretical preventability and estimated reduction in risk for cancers at selected sites. A summary of their results is at Table 12.

The IARC concluded that the increase in risk associated with specific food items and nutrients was likely to be low. This does not deny the possibility that dietary habits as a whole could be responsible for substantial differences in cancer risk, but it further emphasises the need for, and the difficulty of, evaluating the whole diet, taking into account energy providing nutrients (fat, carbohydrates, protein and alcohol) as well as other components (e.g. vitamins, mineral salts, chemical contaminants).

Doll and Peto's estimate was based on cancer interventions that address the totality of diet-related risk factors. This may remove a larger proportion of excess risk, although cancer etiology is a complex process by which independent risk factors do not contribute independently. The IARC and other preventable proportion estimates cited below refer only to a single, more well defined risk factor.

Wahrendorf[13] estimated the proportion of colo-rectal and stomach cancers that might be prevented by certain changes in dietary habits. He based his estimates on a selected number of case control studies of different designs and conducted in different cultural settings. He concluded that changes in population dietary practices may lead to elimination of about 15 to 20 per cent of the excess incidence. For breast cancer a study among Dutch Caucasian women by Van't Veer et. al.[47] estimated that there was a 30 per cent increased risk per 10 per cent of energy derived from fat. These results suggest that in a country with a high fat

TABLE 12 : SUMMARY OF STUDIES ESTIMATING ATTRIBUTABLE FRACTIONS FOR DIET RELATED CANCERS

Disease	Author	Diet Component	Attributable Fraction	Comments
Cancer overall	Doll and Peto[44]	0verall population	10-90% with best estimate of 35%	
Breast	WHO (IARC)[46]	Low fat and animal consumption	Uncertain	
	WHO (IARC)[46]	Weight reduction for the obese	10%	
	Van't Veer et. al.[47]	% Fat/energy	30% increased	Concluded that a country reducing overall fat/energy ratio from 40 to 30% may lower breast cancer by 10 to 30%
	Miller (1985)	Reductions in fat and elimination of obesity	26% incidence reduction over next 60-70 years	
Endometrium	WH0 (IARC)[46]	Weight reduction for the obese	25%	
Stomach	Wahrendorf[13]	Various dietary components	15-20%	
	WH0 (IARC)[46]	High consumption of fruit and vegetables	Up to 50%	
Colon/Rectum	WH0 (IARC)[46]	Low fat and animal protein consumption, high vegetable consumption	Up to 35%	
	Wahrendorf[13]	Various dietary components	15 - 20%	

consumption, a reduction of dietary fat intake from 40 to 30 per cent of energy intake may lower breast cancer incidence by 10 to 30 per cent.

The current study has used Doll and Peto's best estimate of 35 per cent of all cancer deaths due to diet as the "high" estimate. The "middle" and "low" estimates are based on the range of values for specific cancer sites linked to diet as outlined in Table 12.

Cardiovascular disease (especially coronary heart disease, atherosclerosis and stroke):

For cardiovascular diseases a number of studies have estimated population attributable fractions for diet related risk factors (high blood cholesterol and hypertension) but not directly for individual dietary components. A study by Plant et. al.[12] calculated the population attributable fraction for cholesterol levels using relative risk estimates from the United States' Multiple Risk Factor Intervention Trial and blood cholesterol prevalence levels from the 1983 National Heart Foundation Risk Factor Prevalence Study Trial. The population attributable fraction expressed as a percentage was found to be 54 per cent. The authors used sensitivity analysis to show that even if the relative risk was over estimated by 10 per cent the proportion of people who suffer a cardiovascular event attributable to high blood cholesterol levels was in the order of 48 per cent.

Al-Room et. al.[48] estimated the magnitude of the effect of hypertension as a risk factor for acute myocardial infarct (AMI). In the Hunter Region of NSW they studied 250 patients aged between 35 and 64 who presented with a first AMI. The cases were matched by age, sex, and residential area, and control subjects were obtained from a random population sample from the same region. They estimated that 24 per cent of the first AMI's were attributable to hypertension (after adjusting for smoking).

Estimates for the USA of the percentage of coronary heart disease attributable to elevated serum cholesterol and hypertension are similar to the Australian estimates above. Various estimates for cholesterol fall in the 30 to 40 per cent range with blood pressure accounting for between 20 to 25 per cent of all coronary heart disease[49].

The current study provides three estimates for the relationship between diet and coronary heart disease - 60%, 40% and 20%. These figures were based on the assumption that PAFs for raised serum cholesterol and hypertension are additive (54% + 24% $\doteqdot$ 80%) and that diet either contributes 75%, 50% and 25% to the development of these two risk factors (i.e. 0.75 x 80% equals PAF of approximately 60%).

Stroke

Approximately 80%-90% of strokes have been attributed to hypertension[50]. Although the precise relationship between diet and hypertension has not been established, obesity and high sodium intake are the two dietary factors most

commonly implicated in the development of the disease. Based on the literature and consultation the current study provides cost estimates based on the assumption that 75%, 50% and 25% of hypertension is caused by dietary factors, and thus PAFs of 60%, 40% and 20% were used (0.75 x 80% to 90% equals PAF of approximately 60%).

Diabetes (non-insulin dependent):

Obesity (and adiposity) has been described as the major risk factor for non-insulin dependent diabetes. Prevalence estimates are available in Australia but little work has been undertaken in determining the relative risk and population attributable fractions for obesity and diabetes.

Some studies in Pacific Islanders have attempted to quantify the relationship between obesity and diet. The impact of obesity in these studies showed that obesity as a risk factor appears to vary among different populations[51]. In one study the population attributable fractions (potential decrease in disease frequency that might follow from changes in risk factors) for NIDDM in women were 37 per cent for a reduction in BMI from the upper to midtertile, and 52 per cent for such a reduction in the waist-hip ratio. For men the corresponding figures were 42% and 19%[52]. It is not appropriate to use these figures in the Australian context due to the possibility that the Pacific Islanders have a higher genetic pre-disposition for diabetes.

Colditz et. al.[53] examined the relationship between body mass index (weight/height^2) with the risk of non-insulin dependent diabetes in women in the United States. The authors analysed data from a cohort of 114,000 US women aged 30-55 years who were free of diagnosed diabetes. The cohort was followed up over an 8 year period from 1976-1984. Among women of average body mass index (23 - 23.9), the relative risk was 3-6 times that of women with a BMI less than 22. The level of risk continued to increase above this level of BMI. For example, a BMI of 29.0 to 30.9 showed a relative risk of 20.0. Within the total cohort, 90.4% of diagnoses of diabetes were attributable to a body mass index greater than 22. For women with a BMI of 33 or more, 98% of diabetes diagnoses were found to be attributable to obesity.

If it is assumed that the same relative risk for obesity holds true for Australian women and given the fact that 12 per cent of women aged 30 to 64 years are obese[11] the corresponding population attributable fraction percent is 66 per cent (assuming a relative risk of 17). This figure could be higher if females of average body mass index (i.e. those above 22) are included, as was the case in the Colditz study[37].

Studies estimating corresponding PAF for men could not be found in the literature. Thus for the current study it was assumed that obesity has the same effect for males as it did for females in the Colditz study. It has been assumed that obesity directly causes 75%, 50% and 25% of the prevalence of non-insulin dependent diabetes.

The cost of long term complications for non-insulin dependent diabetes has been estimated in the current study. To estimate the attributable fractions associated with chronic complications this paper uses United States estimates by Huse et. al[17]. These are outlined in Table 13. For example, in males less than 65 years of age, 2 per cent of hospital related admissions for hypertension are directly related to NIDDM. It has been assumed that these attributable fractions are also applicable to medical, pharmaceutical, allied professional, nursing home, and indirect costs in the current study.

Whittall et. al.[15] estimated that one in three deaths caused by diabetes in Australia were coded with diabetes as the cause of death in the ABS mortality data. For the purpose of calculating PYLL due to diet-related diabetes, the current study has multiplied the number of deaths with cause of death given as diabetes by three before applying the above PAFs.

TABLE 13 : COMPLICATIONS ATTRIBUTABLE TO NON INSULIN DEPENDENT DIABETES (NIDDM) BY AGE AND SEX

	% of Prevalence attributable to NIDDM			
	< 65 year Old		≥ 65 year Old	
Condition	Male	Female	Male	Female
Circulatory disorders				
Hypertension	2.0	2.2	4.1	6.4
Cardiovascular disease	4.8	6.8	8.4	9.8
Cerebrovascular disease	4.8	5.0	17.0	10.1
Peripheral vascular disease	6.1	5.3	11.5	10.0
Visual disorders				
Glaucoma	7.5	8.4	9.5	9.8
Cataract	5.0	5.6	5.7	5.9
Blindness	11.6	12.9	48.1	49.1
Other disorders				
Nephropathy	3.2	3.6	18.7	19.3
Chronic skin ulcer	5.0	5.6	26.9	27.6
Absence of extremities	3.1	3.5	18.5	19.1

Source: Huse et. al.[17]

Gallbladder disease

Obesity is the dietary factor most commonly associated with the development of gallbladder disease. Berstein et. al.[54] found that development of gallbladder disease was significantly positively associated with body weight, age and the number of children in women. These three risk factors account for up to 88 per cent of the variation in the frequency of occurrence of the disease. Obesity was found to be the dominant factor, with obese women (20-30 years of age) having six times the risk of developing gallbladder disease compared to normal weight women. If this relative risk is assumed to be the same in Australia, the population attributable fraction per cent for women in this age group is 25%, given that the prevalence of obesity in just over 6% in this age group[11]. Since the incidence of gallstones, and the prevalence of obesity increases with age, the PAF for older women due to obesity is likely to be higher than 25%.

Additional quantitative evidence in relation to obesity and gallstone disease has come from early analysis of the Framingham study. People who were 20 per cent above median weight for height had almost twice the relative risk of developing gallbladder disease than those who were less than 90 per cent of the median weight for height.

Given the reported strong relationship between obesity and development of gallstone disease, the 75%, 50%, 25% PAF assumptions have been used in this current study.

Dental caries

Like most other diet-related diseases, the development of dental caries is multifactorial in nature. Even though the etiological role of sugar may have been weakened with the introduction of fluoridated water supplies, sugar intake still poses as a risk factor for approximal caries in children[55]. Fluoride has been shown to be the most effective agent for caries prevention but the incidence of dental caries is still high.

Dietary sugar (fermentable carbohydrates, especially sucrose) along with oral microflora are the two factors most implicated in carie development[56]. Other potential factors are the stimulation of salivary flow, saliva composition, chemical structure of the enamel and the degree of water fluoridation[5]. In terms of carbohydrates, as well as total quantities consumed, the frequency of ingestion (particularly between meals) and the relative stickiness are important factors.

Quantifiable conclusions on the specific relationship between the role of sugar and development of caries is not available in the literature. One recognised difficulty in estimating the relationship is the concept referred to as "background noise". This refers to the difficulty in assessing the impact of one particular food against a background of generally high sugar intakes[56]. Control groups in studies often have only marginally different total sugar intakes compared to the intervention groups. Despite these difficulties some authors talk of a sucrose threshold of about 15kg per year or 40 grams per day as a safe limit.

Given the lack of quantifiable evidence on the relationship between diet and dental caries, PAF of 75%, 50% and 25% have been used in this current paper to determine the diet related costs of dental caries.

Osteoporosis:

Osteoporosis is a condition in which progressive decline of bone mineral results in brittle bones which are more susceptible to fracture than healthy bones. The clinical presentation of osteoporosis is usually from the symptoms caused by the resultant fractures, although there is a separate ICD9-CM code for osteoporosis. The most common fractures which can result from osteoporosis are: proximal fractures of the neck of femur (hip fracture), vertebral fractures and Colles fractures (wrist). Bone mineral loss occurs throughout the skeleton, however, and osteoporosis can also contribute to a lesser extent to fractures in other sites. Table 14 summarises the disease groups included in the economic costing of osteoporosis.

Women are more susceptible to developing osteoporosis for several reasons:

- they have smaller bones and therefore less bone mineral to lose than men;
- they attain peak bone mineral content (BMC) earlier; and
- they suffer an accelerated rate of loss immediately after the menopause associated with declining endogenous oestrogen.

Most deaths associated with osteoporosis result from hip fracture and its aftermath. Various overseas surveys report that between 12 and 40 per cent of all patients with hip fractures die within six months. The large range highlights the difficulty in establishing the cause of death in a group who are often frail, and hence have a higher risk of death from all causes.

Factors associated with a higher risk of post-menopausal osteoporosis have been summarised by Larkins (1990)[57]:

Established

- thin body habitus
- premature menopause
- physical inactivity
- familial factors
- caucasian (as opposed to Negroid or Asian) race

Possible

- low calcium intake
- tobacco use
- alcohol use

There is much controversy on the role of sub-optimum calcium intake and the development of osteoporosis. Available data suggest that a low calcium intake in early life may limit the peak bone mass reached in adult life and therefore be a significant determinant of the prevalence of post-menopausal osteoporosis in the community. However, once peak bone mass is reached, the evidence suggests that although calcium supplements given to menopausal women may have a slight effect in slowing the rate of cortical bone loss, there is little or no reduction of the rate of trabecular bone loss.

TABLE 14: DISEASE GROUPS AND ATTRIBUTION FRACTIONS USED IN COSTING OF OSTEOPOROSIS

ICD-9-CM CODE	DESCRIPTION	OSTEOPOROSIS ATTRIBUTION RATE BY AGE		
		45-59	60-74	75+
733	Osteoporosis	0.82	0.89	0.90
733.1	Pathological fracture of the vertebrae	0.82	0.88	0.89
820	Fracture* of neck of femur	0.51	0.71	0.91
813.4	Fracture* of distal forearm	0.70	0.78	0.84
805	Vertebral fractures*(1)	0.51	0.71	0.84

Source: Phillips et. al.[58]

Notes:

* The following ICD-9-CM E codes were used to select those fractures most likely to be related to osteoporosis:
- accidental falls E880-E888
- struck against or by an object or person E917
- over-exertion and strenuous movements E927
- unspecified accident E928.9
- late effect of fall E929.3

(1) Attribution rates estimated in the current study to be lower rate of fracture of neck of femur or distal forearm fracture.

In conclusion, although it is important for women to maintain an adequate calcium intake throughout life, which may in turn affect peak bone mass and reduce cortical bone loss post-menopausally, the effect of calcium

supplementation for menopausal women in preventing osteoporosis would be slight. Due to the uncertain nature of the role of calcium in the development of osteoporosis, PAFs of 30%, 20% and 10% have been assumed.

Alcohol-related diseases:

For alcohol-related mortality and morbidity, etiological fractions estimated by Armstrong and Holman[14] have been used. These provide detailed 5 year age and sex etiological fractions for some 43 causes of death and morbidity (Refer Appendix B). Given the general acceptance of these estimates by the scientific community the current paper does not use sensitivity analysis to vary these estimates. Rather the one set of estimates of the cost of alcohol are given using the Armstrong and Holman fractions.

Other diet-related diseases

For diverticular disease, constipation, development of hemorrhoids and iron deficiency anemia, PAF's of 75%, 50% and 25% are used as there is little evidence in the literature quantifying their relationship to diet. Whilst some may view 75% as unrealistically high, discussion with experts would suggest that values of 50% and 25% appear to be conservative estimates.

APPENDIX D

METHODOLOGY FOR COST ESTIMATES

Direct Costs

Hospital costs - government and private expenditure

The hospital estimates were based on unit costs being allocated to the number of cases of each disease as indicated in the hospital morbidity data collections held by the AIHW. The hospital morbidity data collections classify disease according to the International Classification of Diseases (ICD-9 CM). The principal diagnosis for cases was used in this study. The methodology applied population attributable fractions, diagnostic related group (DRG) cost weights, average cost per separation, number of DRG separations and adjustments for length of stay differences between the DRGs and principal diagnosis. The DRG cost weights are based on US weights, adjusted for differences in average length of stay variations between the US and NSW DRG data.

Adjustments for costing DRG length of stay outliers were undertaken using criteria for outliers applied by the Department of Victoria, the Yale refinement project, and the Australian DRG refinement project at the University of NSW.

The average cost per separation for public hospitals include non-salary recurrent expenditure, salaries, wages and related payments, and the medical costs for treating private patients in public hospitals. The DRG costs have been adjusted to include outpatient services. Capital has not been included. DRG methodology is under active consideration in Australia and it is likely that the DRG weights will be consistently upgraded for some time to come. Source of funds for the public hospitals include public sector outlays by the Commonwealth and State Governments, health insurance funds, workers compensation, and motor vehicle third party insurance.

Public hospital morbidity data were available for ACT, NT, NSW, VIC and SA. DRG costing was undertaken for each of these States. Such expenditure for each of the remaining States (WA and QLD) was estimated from the State with similar hospital servicing per capita (separations per 1000 population) by diet-related ICD code.

NSW and SA were used to estimate public hospital costs for the other States. Per capita case-mix adjusted hospital expenditure for diet-related diseases was calculated by ICD-9- CM 3 digit code, age, sex for NSW and SA. This expenditure in the relevant State was multiplied by the population in each age/sex group in the `estimated' State. The costs were adjusted for the interstate difference in the public hospital operating costs per 1000 population.

Private hospital sources of funds covered those outlined above for public hospitals, except State government public sector outlays. Private hospital

morbidity data were available for NSW and SA. DRG costing was undertaken for these States. Expenditure in the remaining States was estimated using similar methodology to that for the public hospitals.

States with similar per capita servicing were determined by comparisons of occupied bed days per 1000 population and average length of stay. Per capita case mix adjusted expenditure by ICD-9-CM 3 digit code, age and sex were applied to the population structure of the `estimated' State. Adjustments were made for interstate differences in the cost structure through applying a ratio of the two states' cost per occupied bed day for total non-capital costs.

Medical costs

Government costs

The medical costs (1989-90) were attributed to disease categories based on data from the Australian Morbidity and Treatment Survey (AMTS) carried out by the Division of Family Medicine in the University of Sydney. This involved a national survey of 100,000 patient encounters with GPs over a year. 526 GPs were recruited into the survey from a stratified random sample of 2,100 practitioners who claimed at least 1,500 general practice items of service during 1989. The survey collects data on GPs as well as referrals to specialists, drugs prescribed and investigations for pathology and x-rays.

The diet-related ICD-9-CM codes were converted to the comparable International Classification of Primary Care (ICPC) codes used in the Sydney University survey. The total number of diagnoses/problems managed for these codes was obtained from data provided by the University. Analyses of total medical services utilisation derived in the GP survey indicated that there are approximately 1.5 diagnoses/problems managed in each encounter/visit. The number of diagnoses/problems managed for the ICPC codes was adjusted accordingly to calculate the approximate number of visits that can be attributed solely to each code. This process avoided overestimating the costs associated with the diet related diagnoses, since diagnoses/problems other than those relating to diet may be managed in each visit.

The number of visits estimated for each diet-related disease was divided by the total number of visits in the sample to determine the proportion of total visits relating to the disease. This proportion was applied to out-of-hospital medical utilisation data for specialists and GPs which were analysed separately for the Department of Veterans' Affairs and the Department of Health, Housing and Community Services. The utilisation data derived were adjusted by multiplying by the population attributable fractions to determine the proportion of the utilisation that is attributable to diet. The utilisation data were multiplied by the average benefit to determine the cost estimates.

The medical services costed included GP and specialist attendances, obstetric and gynaecology, anaesthesia, pathology, diagnostic imaging, operations and others.

Services provided for dental and optometry were excluded since they were analysed under allied professional services.

Private costs

Private expenditure on medical services was derived from total private expenditure estimates on medical services compiled in AIHW Bulletins of National Health Expenditure, which use a National Accounting framework. This includes expenditure by health insurance funds, individuals, workers compensation and motor vehicle third party insurance funds.

The proportion of total government medical services expenditure for diet related diseases was used to distribute total private medical expenditure by disease.

Pharmaceuticals

Government and Private costs

The estimation of pharmaceutical costs for diet-related disease also utilised the AMTS data. The following methodology was applied:

(i) For each disease, the number of scripts from the AMTS data set ($NSBW_d$) was identified based on the ICPC codes used for the medical services analysis.

(ii) The proportion of total scripts attributable to each diagnosis ($Prop_d$) was calculated by dividing NSBWd by the total number of scripts in the AMTS data base (98,562 scripts).

$Prop_d = NSBW_d/98,562$

(iii) $Prop_d$ was applied to aggregated pharmaceutical expenditure figures obtained from AIHW health expenditure bulletins (TEPH) to calculate the pharmaceutical expenditure attributable to each disease ($PEXP_d$). The latest year data available was adjusted with a deflator to calculate total pharmaceutical costs for 1989-90. This was undertaken before applying the proportions.

$PEXP_d = Prop_d$ *TEPH

Government and Private pharmaceutical expenditure for 1988-89 was $2,170 million. Applying the deflator:

1989-90 expenditure = 2170*1.3708/1.3160 = $2,260.36 million = TEPH

(iv) The figures derived in (iii) were applied to the before etiological fraction estimate.

(v) The low, middle and high etiological fractions were applied to $PEXP_d$

Expenditure after etiological fraction(s) = $PEXP_d * ET_d$

Nursing homes - government and private expenditure

Estimates of nursing home costs were based on the diagnosis, age, sex and utilisation (bedday) patterns of patients who transfer from hospitals to nursing homes. The analysis assumes that the bedday utilisation patterns of these transferring patients is the same as the whole group of nursing home patients. Previous analyses undertaken by the AIHW indicate that 63% of patients that apply to go to nursing homes are in hospital.

Total beddays for patients transferring from public and private hospitals in each State were compiled by age, sex and diet-related diagnosis at either the three or four digit level. A percentage distribution of these data was calculated to show, in each diagnostic, age and sex cell, the proportion of total beddays for all ICD-9 CM codes in the State that transferred into nursing homes.

In States where there are no private hospital data, the percentage distribution was based on the public hospital transfers only. For States where there is no public hospital data, the percentage distribution was based on the public hospital bedday distribution of the State that had the most similar servicing per capita (separations per 1000 population) for the diet-related diseases.

Total nursing home beddays for 1989-90 was obtained for each State. The utilisation percentage distributions discussed above were applied to the total bedday figures by State. This calculated the total nursing home beddays by State, diet-related diagnostic group, age and sex.

The average bedday cost for nursing homes was multiplied by the number of beddays in each "cell" for each State. The average bedday cost was derived from total costs for nursing homes divided by the total number of beddays. The total costs for 1988-89 nursing homes was extracted from health expenditure compiled by the AIHW, which uses a national accounting framework. It includes private (payments by individuals, workers compensation, and motor vehicle third party insurance funds) and public components of expenditure (Commonwealth, State and Local government). The cost figure was inflated to expenditure in 1989-90 using the Hospital and Clinical deflator compiled by the Australian Bureau of Statistics.

Data for each State was aggregated to determine a national estimate of nursing home costs.

Allied professional services

The "Allied Professional Services" expenditure figures were derived from Health Expenditure Information Bulletin No.7, Australian Health Expenditure to 1990-91 by the AIHW. The expenditure includes paramedical services funded by the Commonwealth Department of Health, Housing and Community Services and

also the Commonwealth Department of Veterans' Affairs. Private sector expenditure covers fund benefit payments by the Health Insurance Funds, payments by individuals, Workers Compensation and motor vehicle third party.

The Health Insurance Funds cover payments for optical, orthoptics, physiotherapy, occupational therapy, speech therapy, psych/group therapy, hypnotherapy, natural therapy, other therapies, dietetics, podiatry, chiropractic and acupuncture. The Department of Veterans' Affairs covers domiciliary nursing, physiotherapy, podiatry, occupational therapy, optical etc. Most of the expenditure by the Commonwealth Department of Health, Housing and Community Services is for optometry.

During 1988-89, $852 million was spent on "Allied Professional Services". This cost excludes those associated with dental services. Of this total, $760 million was spent in the private sector, with $93 million expended in the public area. The total cost ($852 million) was inflated to expenditure in 1989-90, using the Private Final Consumption Expenditure Medical Services - Other Medical deflator compiled by the Australian Bureau of Statistics.

For 1989-90, the estimate for total expenditure on "Allied Professional Services" was $898.18m. The proportion of medical expenditure attributable to diet-related diseases is 12.50% (corresponds to $112.27 million). **This excludes the amount attributable to dental services.** This proportion was used to estimate the costs of diet-related diseases attributable to the "Allied Professional Services" sector.

The proportion of total medical expenditure attributable to alcohol related diseases is 12.28% (before application of population attributable fractions) and 2.2% (after application of population attributable fractions). These proportions were used to estimate the costs ($110.30 million before and $19.83 million after fraction application) of "Allied Professional Services" that were alcohol-related.

Indirect Costs

Morbidity

The methodology outlined below has been based on that by Collins and Lapsley[29]. The authors identified three types of absences from work:

(i) associated with hospital episodes
(ii) associated with receipt of medical services
(iii) not associated with any health care services

The value of production loss resulting from morbidity (types (i) and (ii)) can be calculated by estimating the number of work days lost as a result of each hospital bed-day and medical services visits of people in the workforce[29].

Collins and Lapsley estimate that each hospital bed-day used by a member of the workforce involves on average a further absence of two days work and that each medical service supplied to a member of the workforce involves on average a

loss of half a day's work. These authors were unable to locate any information about absenteeism unassociated with the delivery of health care services.

Outlined below is the methodology developed for this current study.

(i) Absenteeism costs attributable to hospitalisation

The total number of beddays, by age and sex, associated with the disease of interest were determined from hospital morbidity data. Added to this figure was an additional two days for each bedday. Work force participation rates and average daily wage rates were then applied to give total costs for morbidity associated with hospitalisation.

(ii) Absenteeism costs attributable to medical services

An average loss of 0.37 of a day's work was calculated from an analysis of the ABS National Health Survey 1989-90. This proportion was used instead of the estimate of 0.5 of a day determined by Collins and Lapsley.

AMTS data was used to determine the number of medical services, by age and sex, for each disease of interest. The fraction of 0.37 of a day's work was then applied to the number of medical services together with work force participation rates and average daily wage rates to give total costs for morbidity associated with a medical service.

The total costs calculated for hospitalisation and medical services were summed to derive total costs attributable to absenteeism (lost productivity from morbidity) for the disease of interest.

Indirect costs due to morbidity were calculated for housekeepers assuming a "housekeeper" work force participation rate of 32 per cent. This was based on an earlier study by Richardson[32].

Mortality

The indirect costs of mortality relates to the cost of productive capacity lost when people die prior to reaching the normal end of their productive life.

The criterion or meaning of value when calculating the "value" of lost productivity is the loss of output times the amount people would be prepared to pay for that output (i.e. the price). With loss of life there are two approaches that may be adopted. The first (Human Capital) values life as the value of foregone lost production. The second, which is arguably more consistent with the usual meaning of value noted above, is the "Willingness-To-Pay" to preserve life. The two approaches may produce quite different results, with the Willingness-To-Pay approach often producing much larger estimates (see Steadman and Bryon, 1988). However, for practical reasons this study has used the Human Capital approach. Estimates could, therefore, be considered conservative. The methods available to calculate indirect costs are considered more fully in Appendix E.

The estimated cost due to premature mortality is also different from other estimates for another reason. While other costs are incurred in the current year, the mortality costs here are estimates for the total loss of production for all subsequent years lost by the individual. They are calculated by multiplying the potential years of life lost between the ages of 15 and 65 attributable to diet by the workforce participation rate and by the average weekly earnings. A discount rate of 5% is used to calculate the net present value of this foregone stream of future earnings. The data used in the calculations are set out in a table in Appendix F.

The Costs of Housekeeping

The Australian National Accounts in measuring economic production includes the production of all goods and services which legally enter the market. In addition it includes certain economic activities which do not enter the market but for which it is possible to impute values using closely related or analogous market transactions, such as imputed rent of owner-occupied houses. Production within the household sector has been excluded from estimates of GDP.

In measuring unpaid household work it has been traditionally assumed that housekeepers are always females. There are no accurate statistics on the number of housekeepers in Australia. For the purposes of this paper it is assumed that 32% of the PYLL lost by females in each female cohort between the ages of 15 and 65 due to diet-related disease is lost by housekeepers. The average wage rate assigned to housekeepers is assumed to be the wage rate for a paid housekeeper, currently $11 per hour ($440 for 40 hour week). There is little doubt that on using the above method the role of housekeeping is under-valued.

The Australian Bureau of Statistics in 1990 published an information paper titled "Measuring Unpaid Household Work: Issues and Experimental Estimates". The paper was concerned with the measurement of unpaid household work such as cleaning, cooking, child care and gardening which takes place within the household sector. It presents results from the 1987 ABS "Time Use Pilot Survey" which conducted a two week survey covering 1000 private dwellings in the Sydney Statistical Division.

Two methods for valuing time were used, the replacement cost and opportunity cost approaches. The replacement cost approach values time spent on household production according to the cost of hiring a housekeeper to undertake those activities. The opportunity cost method values household work in terms of the earnings foregone by devoting time to unpaid production rather than to paid employment, with foregone market wage being the appropriate valuation for time spent on household activities.

The study found that unpaid household production involved a daily average of 5 hours for all females and two and a half hours for all males. Employed females spent a daily average of 4 hours on unpaid household activity compared with 6 hours by females who were not employed. For employed and unemployed males the corresponding estimates were 2 and 3 hours respectively.

Using these estimates would substantially increase the overall costs of mortality using the human capital approach. More work is needed to clarify the extent of unpaid productive output of households.

APPENDIX E

METHOD USED TO CALCULATE INDIRECT COSTS

There are two principal methodologies for estimating the indirect cost of illness: the human capital method and the willingness-to-pay method. The former method, used in most of the studies undertaken, is called the human capital or output accounting approach because an employed person is seen as producing a stream of output over the years that is valued at the individual's earnings. The main criticism of this methodology is that it excludes intangibles, only counts earnings, and undervalues some groups relative to others because earnings may not accurately reflect one's ability to produce. Thus males are more highly valued than females, white persons more than black persons, and middle-aged people more than the young and elderly, with part of the difference a result of racial and sexual discrimination.

The willingness-to-pay method values human life according to the amount people are willing to spend to obtain reductions in the probability of death[59,60]. This method could be helpful in indicating how individuals value health and life, in deriving social preferences regarding public policy, and in assessing the burden of pain and suffering, which have an intangible quality that is less amenable to evaluation in terms of the monetary value of resources used or foregone[21]. Objections to this method are that the value of individual lives depends on the income distribution, with the rich able to pay more than the poor, and that it is exceedingly difficult for persons to place a value on small reductions in the probability of death. The strengths and weaknesses of these two methodologies are discussed fully in articles on the state of the art of cost-of-illness estimation by Hodgson and Meiners[20] and by Hodgson[19].

The human capital and willingness-to-pay methods are not simply alternatives. Together or separately, each can contribute to a greater understanding of the burden of disease and other hazards. Unfortunately, the precise nature of the relationship between values calculated by the human capital approach and those implied by the willingness-to-pay method has not been determined. Although it is not known to what extent the two values would differ if willingness-to-pay for small reductions in mortality risk could be calculated, lifetime earnings as estimated by the human capital method may at lest be a lower bound to a person's willingness to pay for a decreased risk of death[61,62,63]. Most cost-of-illness studies use the human capital approach as it provides valuable information based on reliable statistics, so long as its limitations[64] are realised. Most micro economic evaluations in the health area, for example, prefer not to cost the value of life, but rather to put direct costs over `life years gained' or 'QALYS' as the decision index. This goes to the heart of the differences between cost/benefit, cost/effectiveness, and cost/utility analysis.

Cost-of-illness studies, nonetheless, are usually answering a different research question to micro economic evaluations, and including indirect costs is more

widely accepted in the disease costing context. Standards have been set for the methodology of cost-of-illness studies by a US Task Force on Disease Costing, in order to make studies in different centres more comparable and permit evaluation of cost estimates presented in publications[20]. Despite this, there are a number of methodological issues about which researchers continue to debate, particularly in the indirect costs category, and a recent Australian study[29] makes interesting reading in this regard.

APPENDIX F

SUPPORTING TABLES

TABLE 15: ASSUMPTIONS USED IN LOSS OF PRODUCTION COST ESTIMATES DUE TO PREMATURE MORTALITY IN 1989 BY AGE COHORT (MALES) FROM DIET-RELATED DISEASES

	AGE COHORT						
	15-19	20-24	25-34	35-44	45-54	55-59	60-64
1. Average years to Retirement	48	43	35	25	15	8	3
2. Workforce Participation Rates	0.53	0.82	0.90	0.91	0.87	0.71	0.41
3. Average Weekly Earnings ($) (Nov 1989)	547	547	547	547	547	547	547
4. PYLL 15-65							
Non-discounted	47,662 (High)		22,098 (Medium)		10,670 (low)		
Discounted 5%	34,110 (High)		16,179 (Medium)		7,815 (low)		

TABLE 16: ASSUMPTIONS USED IN LOSS OF PRODUCTION COST ESTIMATES DUE TO PREMATURE MORTALITY BY AGE IN 1989 COHORT (FEMALES) FROM DIET-RELATED DISEASES

	AGE COHORT					
	15-19	20-24	25-34	35-44	45-54	55-59
1. Average years to Retirement	43	38	30	20	10	3
2. Workforce Participation Rates	0.51	0.71	0.62	0.68	0.58	0.31
3. Average Weekly Earnings ($) (Nov 1989)	454.50	454.50	454.50	454.50	454.50	454.50
4. PYLL 15-65						
Non-discounted	27,895 (High)		13,730 (Medium)		5,930 (low)	
Discounted 5%	19,461 (High)		9,777 (Medium)		4,246 (low)	

TABLE 17: ASSUMPTIONS USED IN LOSS OF PRODUCTION COST ESTIMATES DUE TO PREMATURE MORTALITY IN 1989 BY AGE COHORT (HOUSEKEEPING) FROM DIET-RELATED DISEASES

	AGE COHORT					
	15-19	20-24	25-34	35-44	45-54	55-59
1. Average years to Retirement	43	38	30	20	10	3
2. Workforce Participation Rates	0.32	0.32	0.32	0.32	0.32	0.32
3. Average Weekly Earnings ($) (Nov 1989)	440	440	440	440	440	440
4. PYLL 15-65 Non-discounted Discounted 5%	[see text in Appendix D for assumptions used]					

HEALTH CARE AND OTHER COSTS ASSOCIATED WITH DIET RELATED DISEASES ($M) 1989/90 (BEFORE POPULATION ATTRIBUTABLE FRACTIONS APPLIED)

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
Coronary heart disease (1)	262.57	37.35	299.92	57.92	48.38	30.25	12.26	**448.73**	117.62	559.80	**677.42**	**1,126.15**
Hypertension (1)	14.44	4.63	19.07	262.40	183.55	10.92	55.46	**531.40**	155.94	14.16	**170.10**	**701.50**
Atherosclerosis (1)	7.59	1.50	9.09	1.18	0.40	8.63	0.20	**19.50**	3.29	1.79	**5.08**	**24.58**
Stroke (1)	100.61	21.65	122.26	7.76	2.08	253.83	1.68	**387.61**	73.00	144.15	**217.15**	**604.76**
Diabetes	31.03	3.59	34.62	55.54	23.70	27.34	11.79	**152.99**	56.45	42.82	**99.27**	**252.26**
Diabetes complications												
Hypertension	0.59	0.24	0.83	10.03	7.01	0.72	2.13	**20.72**	5.08	0.24	**5.32**	**26.04**
Cardiovascular disease	26.87	3.74	30.61	7.33	6.31	11.38	1.57	**57.20**	12.12	24.84	**36.96**	**94.16**
Cerebrovascular disease	12.49	2.95	15.44	2.06	0.54	38.25	0.45	**56.74**	6.62	5.71	**12.33**	**69.07**
Peripheral vascular disease	3.19	0.86	4.05	1.79	0.24	3.57	0.34	**9.99**	1.86	0.21	**2.07**	**12.06**
Glaucoma	0.24	0.31	0.55	0.40	0.29	0.12	0.11	**1.47**	0.34	0.00	**0.34**	**1.81**
Cataract	1.14	2.31	3.45	0.25	0.01	0.22	0.04	**3.97**	0.78	0.00	**0.78**	**4.75**
Blindness	0.76	0.05	0.81	0.22	0.00	0.08	0.04	**1.15**	0.17	0.00	**0.17**	**1.32**
Nephropathy	3.16	0.36	3.52	1.07	0.26	1.79	0.22	**6.86**	1.26	0.39	**1.65**	**8.51**
Chronic skin ulcer	5.63	1.78	7.41	3.75	1.41	7.00	0.79	**20.36**	4.06	0.00	**4.06**	**24.42**
Absence of extremities	0.27	0.13	0.40	0.33	0.00	0.16	0.11	**1.00**	0.26	0.00	**0.26**	**1.26**
Total Diabetes	85.37	16.32	101.69	82.77	39.77	90.63	17.59	**332.45**	89.00	74.21	**163.21**	**495.66**
Neoplasms												
Breast	27.70	10.95	38.65	4.49	1.60	10.10	0.97	**55.81**	25.38	160.25	**185.63**	**241.44**
Stomach	15.85	3.78	19.63	0.86	0.48	2.99	0.22	**24.18**	4.98	41.56	**46.54**	**70.72**
Colon	24.99	10.30	35.29	2.59	0.72	7.34	0.56	**46.50**	25.15	104.31	**129.46**	**175.96**
Rectum	19.59	8.51	28.10	(2)	(2)	6.08	(2)	**34.18**	(2)	33.79	**33.79**	**67.97**
Endometrium	5.10	1.14	6.24	0.73	0.27	4.38	0.11	**11.73**	2.99	5.24	**8.23**	**19.96**
Sub-total slected neoplasms	93.23	34.68	127.91	8.67	3.07	30.89	1.86	**172.40**	58.50	345.15	**403.65**	**576.05**
Total all neoplasms	298.32	115.13	413.45	NYA	15.42	116.89	NYA	**545.76**	232.77(3)	1,320.90	**1,553.67**	**2,099.43**

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
Osteoporosis	7.61	3.64	11.25	35.22	23.65	9.74	9.09	**88.95**	18.02		**18.02**	**106.97**
Distal forearm fracture	0.84	0.00	0.84	NYA	NYA	2.48	NYA	**3.32**	1.38(3)		**1.38**	**4.70**
Hip Fracture	43.94	4.36	48.30	0.94	0.43	102.30	0.34	**152.31**	17.89		**17.89**	**170.20**
Vertebral fracture	0.04	0.00	0.04	NYA	NYA	3.71	NYA	**3.75**	1.73(3)		**1.73**	**5.48**
Sub-total osteoporosis	52.43	8.00	60.43	36.16	24.08	118.23	9.43	**248.33**	39.02	NYA	**39.02**	**287.35**
Diverticular disease	18.96	7.77	26.73	7.18	4.57	5.67	1.57	**45.72**	13.05	1.89	**14.94**	**60.66**
Hemorrhoids	14.61	19.31	33.92	11.45	5.50	1.39	2.47	**54.73**	23.29	0.00	**23.29**	**78.02**
Dental caries	6.49	3.89	10.38	7.50	5.33	0.12	925.74	**949.07**	7.37	0.00	**7.37**	**956.44**
Gallstones	53.91	25.60	79.51	6.69	1.76	5.38	1.42	**94.76**	52.28	10.58	**62.86**	**157.62**
Constipation	7.57	2.63	10.20	NYA	NYA	2.63	NYA	**12.83**	2.45(3)	0.00	**2.45**	**15.28**
Iron deficiency anemia	9.06	1.64	10.70	NYA	NYA	3.89	NYA	**14.59**	2.63(3)	0.00	**2.63**	**17.22**
Total diet(5)	726.84	184.97	911.81	489.68	318.49	562.46	1,029.68	**3,312.12**	637.44	1,151.73	**1,789.17**	**5,101.29(6)**
Alcohol related disease	1,122.36	215.18	1,337.54	521.74	348.20	750.05	110.30	**3,067.83**	828.94(7)	2,537.84	**3,366.78**	**6,434.61(6)**

NOTES:

(1) Costs related to complications of diabetes for coronary heart disease, hypertension atherosclerosis and stroke have been netted out.

(2) Colon and rectum are both included in this estimate.

(3) Includes hospital component of absenteeism only.

(4) Preliminary estimates only.

(5) Includes only selected neoplasms (breast, stomach, colon, rectum, endometrium). If all neoplasms were included the total would have been $6,624.67 million.

(6) Totals for diet related diseases and alcohol related diseases in this "prior to attribution" table should not be added together or double counting will occur.

(7) Does not include medical component of absenteeism for alcohol related injuries, suicide, assault or child abuse.

KEY: NYA - Not Yet Available

APPENDIX H

HEALTH CARE AND OTHER COSTS ASSOCIATED WITH DIET RELATED DISEASES ($M) 1989/90 (HIGH ESTIMATE OF POPULATION ATTRIBUTABLE FRACTIONS APPLIED)

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
Coronary heart disease	169.62	24.06	193.68	37.55	31.36	19.99	7.95	**290.53**	74.97	344.82	**419.79**	**710.32**
Hypertension	11.27	3.65	14.92	204.33	142.92	8.73	43.19	**414.09**	120.76	10.80	**131.56**	**545.65**
Atherosclerosis	6.26	1.25	7.51	0.97	0.33	7.28	0.17	**16.26**	2.68	1.37	**4.05**	**20.31**
Stroke	67.86	14.76	82.62	5.89	1.57	175.25	1.28	**266.61**	47.78	89.92	**137.70**	**404.31**
Diabetes	23.27	2.69	25.96	41.66	17.78	20.50	8.84	**114.74**	42.34	32.12	**74.46**	**189.20**
Diabetes complications												
-Hypertension	0.45	0.18	0.63	7.52	5.26	0.54	1.60	**15.55**	3.81	0.18	**3.99**	**19.54**
-Cardiovascular disease	20.16	2.81	22.97	5.50	4.73	8.53	1.18	**42.91**	9.09	18.63	**27.72**	**70.63**
-Cerebrovascular disease	9.37	2.21	11.58	1.54	0.40	28.69	0.34	**42.55**	4.97	4.28	**9.25**	**51.80**
-Peripheral vascular disease	2.39	0.64	3.03	1.34	0.18	2.68	0.26	**7.49**	1.40	0.16	**1.56**	**9.05**
-Glaucoma	0.18	0.24	0.42	0.30	0.22	0.09	0.08	**1.11**	0.26	0.00	**0.26**	**1.37**
-Cataract	0.86	1.73	2.59	0.19	0.01	0.16	0.03	**2.98**	0.59	0.00	**0.59**	**3.57**
-Blindness	0.57	0.04	0.61	0.16	0.00	0.06	0.03	**0.86**	0.14	0.00	**0.14**	**1.00**
-Nephropathy	2.37	0.27	2.64	0.80	0.20	1.34	0.16	**5.14**	0.94	0.29	**1.23**	**6.37**
-Chronic skin ulcer	4.22	1.33	5.55	2.81	1.06	5.25	0.59	**15.26**	3.04	0.00	**3.04**	**18.30**
-Absence of extremities	0.20	0.10	0.30	0.25	NYA	0.12	0.08	**0.75**	0.20	0.00	**0.20**	**0.95**
Total Diabetes	64.04	12.24	76.28	62.07	29.84	67.96	13.19	**249.34**	66.78	55.66	**122.44**	**371.78**
Neoplasms (35% of all neoplasms)	104.41	40.30	144.71	NYA	5.40	40.91	NYA	**191.02**	81.47(1)	462.31	**543.78**	**734.80**

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
OSTEOPOROSIS:												
-Osteoporosis	2.29	1.09	3.38	10.57	7.10	2.92	2.73	**26.70**	5.41		**5.41**	**32.11**
-Distal forearm fracture	0.25	0.00	0.25	NYA	NYA	0.74	NYA	**0.99**	0.41(1)		**0.41**	**1.40**
-Hip Fracture	13.18	1.31	14.49	0.28	0.13	30.69	0.05	**45.64**	5.37		**5.37**	**51.01**
-Vertebral fracture	0.01	0.00	0.01	NYA	NYA	1.11	NYA	**1.12**	0.52(1)		**0.52**	**1.64**
Sub-total osteoporosis	15.73	2.40	18.13	10.85	7.23	35.46	2.78	**74.45**	11.71	NYA	**11.71**	**86.16**
Diverticular disease	14.22	5.83	20.05	5.39	3.43	4.26	1.18	**34.31**	9.79	1.42	**11.21**	**45.52**
Hemorrhoids	10.95	14.48	25.43	8.59	4.13	1.04	1.85	**41.04**	17.46	0.00	**17.46**	**58.50**
Dental caries	4.87	2.92	7.79	5.63	4.00	0.09	694.30(2)	**711.81**	5.54	0.00	**5.54**	**717.35**
Gallstones	40.43	19.20	59.63	5.02	1.32	4.04	1.06	**71.07**	39.21	7.93	**47.14**	**118.21**
Constipation	5.68	1.97	7.65	NYA	NYA	1.97	NYA	**9.62**	1.84(1)	0.00	**1.84**	**11.46**
Iron deficiency anemia	6.79	1.23	8.02	NYA	NYA	2.92	NYA	**10.94**	1.97(1)	0.0	**1.97**	**12.91**
Total diet	522.13	144.29	666.42	346.29	231.53	369.90	766.95	**2,381(3)**	481.96	974.23	**1,456.19**	**3,837.28**
Alcohol related diseases	165.27	33.64	198.91	93.77	49.50	108.00	19.83	**470.01(4)**	169.22(5)	713.68	**822.90(6)**	**1,352.91**
Total												**5,190.19**

NOTES:

(1) Includes hospital component of absenteeism only.

(2) Preliminary estimate only.

(3) Does not yet include medical or allied health professional costs for neoplasms.

(4) For alcohol related diseases a number of studies have shown that direct health care costs are only a minor part (10-30%) of total economic costs.

(5) Does not include medical component of absenteeism for alcohol related injuries, suicide, assault or child abuse.

(6) Other indirect costs (absenteeism, lost productivity, road accidents) are likely to add substantially to this estimate.

KEY: NYA - Not Yet Available

APPENDIX I

HEALTH CARE AND OTHER COSTS ASSOCIATED WITH DIET RELATED DISEASES ($M) 1989/90 (MID ESTIMATE OF POPULATION ATTRIBUTABLE FRACTIONS APPLIED)

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
Coronary heart disease	113.08	16.04	129.12	25.03	20.91	13.32	5.30	193.68	49.98	229.88	279.86	473.54
Hypertension	7.52	2.43	9.95	136.22	95.28	5.82	28.80	276.07	80.51	7.20	87.71	363.78
Atherosclerosis	4.17	0.83	5.00	0.65	0.22	4.85	0.11	10.83	1.78	0.91	2.69	13.52
Stroke	45.24	9.84	55.08	3.93	1.05	116.83	0.85	177.74	31.85	59.95	91.80	269.54
Diabetes	15.51	1.79	17.30	27.77	11.85	13.67	5.90	76.49	28.23	21.41	49.64	126.13
Diabetes complications												
-Hypertension	0.30	0.12	0.42	5.01	3.51	0.36	1.07	10.37	2.55	0.12	2.67	13.04
-Cardiovascular disease	13.44	1.87	15.31	3.66	3.16	5.69	0.79	28.61	6.05	12.42	18.47	47.08
-Cerebrovascular disease	6.25	1.48	7.73	1.03	0.27	19.12	0.23	28.38	3.31	2.85	6.16	35.54
-Peripheral vascular disease	1.60	0.43	2.03	0.89	0.12	1.78	0.17	4.99	0.93	0.10	1.03	6.02
-Glaucoma	0.12	0.16	0.28	0.20	0.15	0.06	0.06	0.75	0.17	0.00	0.17	0.92
-Cataract	0.57	1.15	1.72	0.13	0.00	0.11	0.02	1 98	0.40	0.00	0.40	2.38
-Blindness	0.38	0.02	0.40	0.11	0.00	0.04	0.02	0.57	0.09	0.00	0.09	0.66
-Nephropathy	1.58	0.18	1.76	0.53	0.13	0.90	0.11	3.43	0.63	0.20	0.83	4.26
-Chronic skin ulcer	2.81	0.89	3.70	1.87	0.71	3.50	0.40	10.18	2.03	0.00	2.03	12.21
-Absence of extremities	0.13	0.07	0.20	0.17	NYA	0.08	0.06	0.51	0.13	0.00	0.13	0.64
Total Diabetes	42.69	8.16	50.85	41.37	19.90	45.31	8.83	166.26	44.52	37.10	81.62	247.88
Neoplasms												
-Breast	8.31	3.29	11.60	1.35	0.49	3.03	0.29	16.76	7.61	48.07	55.68	72.44
-Stomach	7.92	1.89	9.81	0.43	0.24	1.50	0.11	12.09	2.49	20.78	23.27	35.36
-Colon	8.75	3.60	12.35	0.91	0.25	2.57	0.20	16.28	8.80	36.51	45.31	61.59
-Rectum	6.86	2.98	9.84	(1)	(1)	2.13	(1)	11.97	(1)	11.82	11.82	23.79
-Endometrium	1.27	0.29	1.56	0.18	0.07	1.10	0.11	3.02	0.75	1.31	2.06	5.08
Sub-total selected neoplasms	33.11	12.05	45.16	2.87	1.05	10.33	0.71	60.12	19.65	118.49	138.14	198.26

DIET RELATED	PUBLIC HOSPITAL $M	PRIVATE HOSPITAL $M	TOTAL HOSPITAL $M	MEDICAL $M	PHARM-ACEUT-ICAL $M	NURS-ING HOME $M	ALLIED PROFES-SIONAL $M	TOTAL DIRECT $M	SICK LEAVE $M	FORE-GONE EARNINGS $M	TOTAL INDIRECT $M	TOTAL $M
OSTEOPOROSIS												
-Osteoporosis	1.52	0.72	2.24	7.04	4.73	1.95	1.82	17.78	3.61		3.61	21.39
-Distal forearm fracture	0.17	0.00	0.17	NYA	NYA	0.50	NYA	0.67	0.28(2)		0.28	0.95
-Hip Fracture	8.79	0.87	9.66	0.19	0.09	20.46	0.03	30.43	3.58		3.58	34.01
-Vertebral fracture	0.01	0.00	0.01	NYA	NYA	0.74	NYA	0.75	0.35(2)		0.35	1.10
Sub-total osteoporosis	10.49	1.59	12.08	7.23	4.82	23.65	1.85	49.63	7.82	NYA	7.82	57.45
Diverticular disease	9.48	3.89	13.37	3.59	2.29	2.84	0.79	22.88	6.53	0.94	7.47	30.35
Hemorrhoids	7.30	9.65	16.95	5.73	2.75	0.69	1.24	27.36	11.65	0.00	11.65	39.01
Dental caries	3.25	1.95	5.20	3.75	2.67	0.06	462.87(3)	474.55	3.69	0.00	3.69	478.24
Gallstones	26.96	12.80	39.76	3.34	0.88	2.69	0.71	47.38	26.14	5.29	31.43	78.81
Constipation	3.79	1.32	5.11	NYA	NYA	1.32	NYA	6.43	1.23(2)	0.00	1.23	7.66
Iron deficiency anemia	4.53	0.82	5.35	NYA	NYA	1.95	NYA	7.30	1.32(2)	0.00	1.32	8.62
Total diet	311.61	81.37	392.98	233.71	151.82	229.66	512.06	1,520.23	286.67	459.76	746.43	2,266.66
Alcohol related disease	165.27	33.64	198.91	93.77	49.50	108.00	19.83	470.01(4)	169.22(5)	713.68	882.90(6)	1,352.91
Total												3,619.57

NOTES:

(1) Colon and rectum are both included in this estimate.

(2) Includes hospital component of absenteeism only.

(3) Preliminary estimates only.

(4) For alcohol related diseases a number of studies have shown that direct health care costs are only a minor part (10-30%) of total economic costs.

(5) Does not include medical component of absenteeism for alcohol related injuries, suicide, assault or child abuse.

(6) Other indirect costs (lost productivity, road accidents) are likely to add substantially to this estimate.

KEY: NYA - Not Yet Available

HEALTH CARE AND OTHER COSTS ASSOCIATED WITH DIET RELATED DISEASES ($M) 1989/90 (LOW ESTIMATE OF POPULATION ATTRIBUTABLE FRACTION)

DIET RELATED	PUBLIC HOSPITAL	PRIVATE HOSPITAL	TOTAL HOSPITAL	MEDICAL	PHARM-ACEUT-ICAL	NURS-ING HOME	ALLIED PROFES-SIONAL	**TOTAL DIRECT**	SICK LEAVE	FORE-GONE EARNINGS	**TOTAL INDIRECT**	**TOTAL**
	$M	$M	$M	$M	$M	$M	$M	**$M**	$M	$M	**$M**	**$M**
Coronary heart disease	56.54	8.02	64.56	12.52	10.45	6.66	2.65	**96.84**	24.99	114.94	**139.93**	**236.77**
Hypertension	3.76	1.22	4.98	68.11	47.64	2.91	14.40	**138.04**	40.25	3.60	**43.85**	**181.89**
Atherosclerosis	2.09	0.42	2.51	0.33	0.11	2.43	0.06	**5.44**	0.90	0.46	**1.36**	**6.80**
Stroke	22.62	4.92	27.54	1.97	0.52	58.42	0.43	**88.88**	15.93	29.97	**45.90**	**134.78**
Diabetes	7.76	0.90	8.66	13.89	5.93	6.83	2.95	**38.26**	14.12	10.71	**24.83**	**63.09**
Diabetes complications												
Hypertension	0.15	0.06	0.21	2.51	1.75	0.18	0.53	**5.18**	1.27	0.06	**1.33**	**6.51**
Cardiovascular disease	6.72	0.94	7.66	1.83	1.58	2.84	0.39	**14.30**	3.03	6.21	**9.24**	**23.54**
Cerebrovascular disease	3.12	0.74	3.86	0.52	0.14	9.56	0.11	**14.19**	1.65	1.43	**3.08**	**17.27**
Peripheral vascular disease	0.80	0.21	1.01	0.45	0.06	0.89	0.09	**2.50**	0.46	0.05	**0.51**	**3.01**
Glaucoma	0.06	0.08	0.14	0.10	0.07	0.03	0.03	**0.37**	0.08	0.00	**0.08**	**0.45**
Cataract	0.29	0.58	0.87	0.06	0.00	0.05	0.01	**0.99**	0.20	0.00	**0.20**	**1.19**
Blindness	0.19	0.01	0.20	0.06	0.00	0.02	0.01	**0.29**	0.05	0.00	**0.05**	**0.34**
Nephropathy	0.79	0.09	0.88	0.27	0.07	0.45	0.06	**1.73**	0.31	0.10	**0.41**	**2.14**
Chronic skin ulcer	1.41	0.44	1.85	0.94	0.35	1.75	0.20	**5.09**	1.01	0.00	**1.01**	**6.10**
Absence of extremities	0.07	0.03	0.10	0.08	NYA	0.04	0.03	**0.25**	0.07	0.00	**0.07**	**0.32**
Total Diabetes	21.36	4.08	25.44	20.71	9.95	22.64	4.41	**83.15**	22.25	18.56	**40.81**	**123.96**
Neoplasms												
Breast	2.77	1.10	3.87	0.45	0.16	1.01	0.10	**5.59**	2.53	16.02	**18.55**	**24.14**
Stomach	2.38	0.57	2.95	0.13	0.07	0.45	0.03	**3.63**	0.75	6.23	**6.98**	**10.61**
Colon	3.75	1.54	5.29	0.39	0.11	1.10	0.08	**6.97**	3.78	15.64	**19.42**	**26.39**
Rectum	2.94	1.28	4.22	(1)	(1)	0.91	(1)	**5.13**	(1)	5.07	**5.07**	**10.20**
Endometrium	0.51	0.11	0.62	0.07	0.03	0.44	0.01	**1.17**	0.32	0.52	**0.84**	**2.01**
Sub-total selected neoplasms	12.35	4.60	16.95	1.04	0.37	3.91	0.22	**22.49**	7.38	43.48	**50.86**	**73.35**

DIET RELATED	PUBLIC HOSPITAL	PRIVATE HOSPITAL	TOTAL HOSPITAL	MEDICAL	PHARM-ACEUT-ICAL	NURS-ING HOME	ALLIED PROFES-SIONAL	TOTAL DIRECT	SICK LEAVE	FORE-GONE EARNINGS	TOTAL INDIRECT	TOTAL
	$M	$M	$M	$M	$M	$M	$M	$M	$M	$M	$M	$M
Osteoporosis	0.76	0.37	1.13	3.52	2.37	0.97	0.91	**8.90**	1.80		**1.80**	**10.70**
Distal forearm fracture	0.08	0.00	0.08	NYA	NYA	0.25	NYA	**0.33**	0.14(2)		**0.14**	**0.47**
Hip fracture	4.39	0.44	4.83	0.09	0.04	10.23	0.02	**15.21**	1.79		**1.79**	**17.00**
Vertebral fracture	0.00	0.00	0.00	NYA	NYA	0.37	NYA	**0.37**	0.17(2)		**0.17**	**0.54**
Sub-total osteoporosis	5.23	0.81	6.04	3.61	2.41	11.82	0.93	**24.81**	3.90	NYA	**3.90**	**28.71**
Diverticular disease	4.74	1.94	6.68	1.80	1.14	1.42	0.39	**11.43**	3.27	0.47	**3.74**	**15.17**
Hemorrhoids	3.65	4.83	8.48	2.86	1.38	0.35	0.62	**13.69**	5.82	0.00	**5.82**	**19.51**
Dental caries	1.62	0.97	2.59	1.88	1.33	0.03	249.42(3)	**255.25**	1.85	0.00	**1.85**	**257.10**
Gallstones	13.48	6.40	19.88	1.67	0.44	1.35	0.36	**23.70**	13.07	2.64	**15.71**	**39.41**
Constipation	1.89	0.66	2.55	NYA	NYA	0.66	NYA	**3.21**	0.61(2)	NYA	**0.61**	**3.82**
Iron deficiency anemia	2.26	0.41	2.67	NYA	NYA	0.97	NYA	**3.64**	0.66(2)	NYA	**0.66**	**4.30**
Total diet(5)	151.59	39.28	190.87	116.50	75.74	113.57	273.89	**770.57**	140.88	214.12	**355.00**	**1,125.57**
Alcohol related disease	165.27	33.64	198.91	93.77	49.50	108.00	19.83	**470.01(4)**	169.22(5)	713.68	**882.90(6)**	**1,352.91**
Total												**2,478.48**

Notes:

(1) Colon and Rectum are included in this estimate.

KEY: NYA - Not Yet Available

(2) Includes hospital component of absenteeism only.

(3) Preliminary estimate only.

(4) For alcohol related diseases a number of studies have shown that direct health care costs are only a minor part (10-30%) of total economic costs.

(5) Does not include medical component of absenteeism for alcohol related injuries, suicide, assault or child abuse.

(6) Other indirect costs (absenteeism, lost productivity, road accidents) are likely to add substantially to this estimate.

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