5.4 The role of diet in diet-related disease

Health associations of excess energy intake

Obesity

The NHMRC defines obesity as a BMI greater than 30 kg / m^2 (see Box 5.2). Genetic and environmental factors are likely to be involved in the aetiology of obesity,[147] which has been described as 'a heterogeneous disorder with multiple aetiologies, and hence, multiple risk factors'.[148]

Obesity is the result of energy intake being greater than energy expenditure for a sustained period, often years. Energy expenditure is determined by the sum of the resting metabolic rate and level of physical exercise. Loss of excess weight is possible if the level of physical activity is increased and food intake is decreased, although the difficulties for many obese people in changing their behaviours are well recognised.[149] There is growing evidence that total dietary fat levels are positively associated with an increased prevalence of obesity.[87] Dietary fat has only a weak effect on satiety, and the result may be to contribute to passive over-consumption of fat.[147] Factors such as the flavour and texture of food may need to be considered in managing obesity; food variety has been found to be positively associated with a lower waist-to-hip ratio in a survey of Melbourne women of Chinese origin.[150]

Pi-Sunyer[151] has reviewed the health implications of obesity, concluding that the health risks of obesity increase with its severity, and that it is a major health risk factor in mortality, particularly cardiovascular disease mortality but also mortality due to NIDDM, digestive disorders and stroke in men, and cancer in women. Not all the studies reviewed found a relationship between obesity and mortality, although shorter follow-up periods in these studies may partly account for the differences.[151] Lee at al. found a positive association between BMI and all-cause mortality in a 27-year follow-up of middle-aged men from the Harvard Alumni Health Study; the relative risk between the heaviest and lightest quintiles was 1.57 in those who had never smoked and a J-shaped relationship in current smokers, of whom those for which $23.5 < BMI \leq 24.5$ had the least risk.[152] Obesity can potentiate other cardiovascular risk factors, such as hypertension, hyperlipidaemias, low HDL cholesterol, and impaired glucose tolerance with hyperinsulinaemia, and it may increase the risk of cardiovascular disease morbidity independently.[131,153,154]

The relationship between obesity and hypertension is well documented but the pathophysiology is not well defined. Studies such as the Framingham Study have shown that obese individuals have a significantly greater risk of developing high blood pressure than lean individuals. Data from the INTERSALT Study showed correlations in men and women aged 20–59 years between BMI and both systolic and diastolic blood pressure, and a similar correlation between weight and blood pressure if there was direct adjustment for height.[155] A positive association has been found between blood pressure and BMI in people aged 60–87 years.[156]

Obesity is also associated with a greater risk of gall bladder disease, arthritis, gout, pulmonary dysfunction, and some forms of cancer. For example, the American Cancer Study found that overweight men had significantly higher mortality ratios for colorectal and prostate cancers and overweight women had significantly higher rates of endometrial, gall bladder, cervical, ovarian and breast cancers. Not all studies, however, have shown positive associations.[151]

Obesity is the dietary factor most commonly associated with gallstones. Maclure et al.[157] found a strong positive association between obesity and symptomatic gallstones in middle-aged women. Hayes et al. suggest that excess energy intake may lead to expression of a genetic predisposition to gallstone formation in susceptible individuals.[158] Obesity may also be associated with elevated blood lipids.

There are several techniques for a precise determination of the amount of body fat, but they require the use of sophisticated equipment and are normally confined to research use. Anthropometric measures are used for clinical or surveillance purposes as indicators of adiposity. The most common measure of overall obesity is BMI.

Body fat distribution

Body fat distribution has emerged as an important predictor of obesity-related morbidity and mortality. Bjorntorp et al. [159–161] concluded that cardiovascular disease, stroke, premature death and NIDDM are closely associated with abdominal obesity, and that this association appears to be independent of overall obesity. In addition, increased abdominal obesity has been linked to some carcinomas found only in women, increased plasma cholesterol, increased blood pressure, increased triglycerides, insulin insensitivity, increased fibrinogen levels, and low sex-hormone-binding globulin.[162,163] Abdominal fat is the type of fat most common in men when they become obese. Gluteal-femoral obesity, which is more common in women, has been found to be associated only with varicose veins and joint problems. The common measure used to estimate body fat distribution is the waist-to-hip ratio (WHR), which is calculated from waist circumference measured in the horizontal plane, at the end of normal expiration and around the narrowest point between the ribs and hips when viewed from the front. Hip circumference was measured at the point of maximum extension of the buttocks, viewed from the side and measured in the horizontal plane.

Because it is a measure of abdominal obesity, WHR correlates better with cardiovascular disease risk.[164,165] Neither BMI nor WHR, however, are considered to be sensitive indicators of total or abdominal fat mass, and comparisons are made more difficult by differences in application from study to study.[166–169] Several other indexes of abdominal obesity have been advocated but there is no consensus.

Rodin et al. reported a positive correlation between WHR and variability in an individual's weight.[170] Masuda et al. reported an association between WHR and abdomen-to-hip circumference with cardiovascular disease risk factors (systolic blood pressure and serum lipids).[171]

Weight loss and weight cycling

In a review of six epidemiological studies, Williamson and Pamuk concluded that a relationship between mortality and weight reduction by overweight persons was equivocal; the studies lacked power primarily because of the difficulties in conducting randomised, controlled studies and a failure to distinguish between voluntary and involuntary weight loss.[172] A review by Andres et al. concluded from a review of 13 studies in which participants were weighed at 'two separate times during adult life' that those who gain some weight during adulthood tended to survive longer than those who maintain or lose weight; long-term weight loss ‘was generally associated with high mortality rate’.[173]

Data on the health effects of weight cycling—repeated weight gains and losses—are inconclusive,[174] but energy metabolism appears to be affected and this may result in faster regaining of weight.[175]

In the Western Electric Study men who experienced large gains and losses in body weight during young adulthood had an increased risk of coronary death, although the subsample was small (n=98).[176] Weight cycling was associated with higher all-cause mortality in the Baltimore Longitudinal Study,[177] the Framingham Study[178] and the Gothenburg Study.[179]

In a prospective study of body weight change in a cohort of 11 703 men from the Harvard Alumni Health Study (mean age 57.6 years in 1977), Lee and Paffenberger found that, compared with those whose weight was stable, weight fluctuation was associated with a higher 12-year mortality from all causes and coronary heart disease but not from cancer.[180] Those in the highest quintile of BMI were at higher risk than those in the lowest quintile, but adding BMI as a variable in the analysis did not alter the association between weight change and mortality; the authors noted that there was no correlation between BMI and weight change.[180] Weight cycling was associated with higher all-causes and cardiovascular mortality risk in some men participating in the Multiple Risk Factor Intervention Trial; the risk from weight cycling was greater in leaner men.[181] The association between weight change and higher all-causes and coronary heart disease (but not cancer) mortality was consistent with some data from the Framingham population,[178] although Higgins et al. found that weight loss was also associated with improvements in blood pressure and reduced serum cholesterol levels.[182] Some studies on smaller samples did not find an association.[177,183] Ernsberger and Koletsky note that in animal models weight cycling is associated with adverse cardiovascular and metabolic changes.[184]

Garrow agrees that there is good evidence that weight cycling is associated with an increased risk of heart disease and premature death. Nevertheless, he warns, ‘This observation … has been used as an argument that it would be better not to try to lose weight in the first place. However, there is no evidence that the association between weight variation and heart disease is causal, nor that weight regain is inevitable, whereas there is good evidence that obesity is a serious health risk.’[185]

Physical activity

The NHMRC statement on the role of exercise in nutrition and health noted that inactivity was a health and nutrition risk and that exercise had a beneficial effect.[91] In 1992 the American Heart Association, in a position statement on the known benefits and recommendations for physical activity programs for all Americans, took the same view.[186] There is no evidence to suggest that physical inactivity causes obesity, but the increased mechanical load associated with obesity contributes to reduced exercise tolerance.[148,187]

Exercise can help control blood lipid abnormalities, diabetes and obesity. Bauman and Owen[188] reported significant independent associations between levels of exercise participation and HDL cholesterol level, the HDL-to-total-cholesterol ratio, and serum triglyceride levels for both men and women. Cox et al. found that a concurrent exercise regime did not offset the decrease in HDL cholesterol level brought about by a reduced alcohol intake in 75 sedentary men.[189] They also observed an association between physical activity and systolic blood pressure in older men.

Exercise is also related to increased mineral content of bones, and a decline in physical activity may increase the rate of osteoporosis.[91,188]

Exercise levels in the community

The following are the most recent sources of data on participation in leisure-time exercise by Australians:

- the National Heart Foundation Risk Factor Prevalence Study;[27,190,191]
- the National Health Survey, 1989–90;[192]
- the Pilot Survey of the Fitness of Australians, 1990–91;[193]
- six national surveys of physical activity undertaken by the Department of the Arts, Sport, the Environment, Tourism and Territories between 1984 and 1987;[194]
- the 1985 Australian Health and Fitness Survey.[24]

A significant proportion of the Australian adult population can be classified as sedentary or undertaking no exercise for sport, recreation or fitness. The 1989 Risk Factor Prevalence Survey found that 36 per cent of men and women did not exercise during leisure time in the previous two weeks.[27] The corresponding figure for the National Health Survey[192] was 36 per cent of adults and for the Pilot Survey of Fitness[193] 20 per cent. Bauman et al.[195] found a statistically significant decline between 1984 and 1987 in the number of people reported to be totally sedentary; inactivity was more common in women and older people and was associated with lower education and income status. The National Health Survey and the Pilot Survey of Fitness confirmed the association with age, sex and educational status.[192,193] The National Health Survey also found that overweight and obese individuals were less likely to exercise than people with a BMI within the normal range. Smokers were less likely to exercise than ex-smokers and people who had never smoked. Males were more likely to undertake vigorous or moderate exercise; females were more likely to walk for exercise. The older age groups of both sexes were more likely to use walking as their form of exercise. People who walked for exercise tended to exercise more often and for longer periods than those who engaged in vigorous or moderate exercise.

Alcohol intake

Heavy drinking

Heavy alcohol consumption is a risk factor for coronary heart disease. An association has been reported in men who were alcoholics or problem drinkers,[196–198] and Hanna et al. found that the rate of early cardiovascular disease mortality among women who are heavy drinkers was similar to that of men who are heavy drinkers.[198] An increased risk of sudden cardiac death for heavy drinkers has also been reported,[199–201] although the association was not significant in one of the three studies.[199] Heavy drinkers have an increased risk of stroke, and particularly sub-arachnoid haemorrhage. Comparisons between studies are rendered difficult by the use of different definitions of ‘heavy drinking’, but generally the relative risk of stroke for the heaviest drinkers compared with non-drinkers ranged from 0.5 to 4.2, depending on the type of stroke examined.[201–206]

Alcoholic beverage consumption has also been linked to many forms of cancer including cancer of the breast,[207–210] lip, larynx, oro-pharynx, oesophagus, stomach, colon, rectum, liver, gall bladder and pancreas.[211,212] Rectal cancer risk has been linked to the consumption of beer.[87]

Heavy, long-term alcohol consumption causes cirrhosis of the liver, portal hypertension, and oesophageal varices and their complications (such as anaemia due to chronic bleeding from varices). Batey et al.[213] compared the risk of liver cirrhosis from alcohol consumption in 43 men with an earlier study of women. It was shown that the risk of cirrhosis was apparent for men and women at intakes in excess of 40 g ethanol per day, but that women were more susceptible: the odds for women of developing cirrhosis at intakes of 41–80 g ethanol per day (odds ratio = 57) greatly exceeded the odds for men at intakes in excess of 80 g ethanol per day (odds ratio = 22).

Heavy, long-term consumption can also cause alcohol-related brain damage (Wernicke's encephalopathy and Korsakoff's psychosis), polyneuropathy, cardiomyopathy, gastritis and acute poisoning.[81,87,214] Alcohol consumption in pregnancy can lead to foetal alcohol syndrome.[81,87,214] There may be an association with osteoporosis because bone density is less and rate of bone loss greater in those who drink large amounts of alcoholic beverage.[87] Other morbid conditions associated with alcohol abuse include malnutrition, injury and accidental death and, since alcohol is a drug, its use may also lead to dependence, emotional disturbances or suicide.[81,211]

Alcohol is also associated with other health risk factors such as hypertension, obesity and hyperlipidaemia. Moderate and heavy drinkers have higher blood pressure compared with non-drinkers;[196,201,215,216] blood pressure fell with cessation of alcohol intake.[8] Dyer et al. reported a weak but positive association between alcohol intake and systolic and diastolic blood pressure in young adults from the CARDIA Study,[217] and a causal relationship between regular alcohol consumption and high blood pressure has been established in a number of cross-sectional studies and randomised trials reviewed by Beilin and Puddey.[218]

Light-to-moderate drinking

Many epidemiological studies have shown light-to-moderate alcohol consumption to have a protective effect against coronary heart disease,[196,202,219–227] although other studies—for example the Oxford Vegetarian Study—found no difference in cardiovascular disease risk factors between moderate drinkers, heavy drinkers, lifelong teetotallers and ex-drinkers.[228] Alcohol consumption at low levels has been shown to raise HDL cholesterol levels in American adults.[229] There is concern that the U-shaped relationship between coronary heart disease and alcohol may divert or dilute action to reduce the social and health problems of alcohol abuse.[230,231] Recently Gaziano et al. conducted a case-control study that demonstrated a decreased risk of myocardial infarction mediated by increases in the HDL cholesterol subfractions HDL_2 and HDL_3, noting, however, that 'further research should be directed to finding safer methods of raising the level of this lipid'.[232]

Cullen et al. have undertaken a number of analyses of data from the Busselton population studies. Mortality from all causes between 1966 and 1979 was higher for non-drinkers than for drinkers, both men and women,[233] and 10 years later there was again a significant inverse association between alcohol consumption and 23-year mortality data.[234] Relative risks for all-cause mortality in moderate drinkers compared with non-drinkers were 0.71 for men and 0.85 for women; corresponding relative risks for cardiovascular disease mortality (and coronary heart disease mortality) were 0.69 (and 0.69) for men and 0.59 (and 0.35) for women.[234]

Levels of alcohol intakes in the population are examined in Chapter 4.

Dietary sodium and hypertension

Three studies by Law et al. investigated the relationship between sodium intake and hypertension based on data from a large number of studies undertaken throughout the world. It was found that blood pressure varied according to sodium intake and that a reduction in dietary salt intake lowered blood pressure to an extent that increased with age and with initial blood pressure.[235,236] Age and initial blood pressure were directly related to the degree of change in blood pressure for a given difference in sodium intake. Within-population studies failed to demonstrate an association between blood pressure and sodium intake, but when allowance was made for a systematic underestimation bias the within-population studies collectively showed a highly significant association between blood pressure and sodium intake.[237]

Law et al. concluded that avoidance of salty foods and not adding salt during cooking or at the table could reduce sodium intake by about 50 mmol per 24 hours (about 3 g of sodium chloride) and that after a few weeks such a reduction would be expected to lower systolic blood pressure by 5–7 mmHg in people aged 50 to 59 years, depending on their initial blood pressure.[236] A universal reduction in sodium intake of 50 mmol per 24 hours would reduce the incidence of stroke by one-fifth and that of ischaemic heart disease by one-sixth and would be a more effective strategy than targeting only those in the population with high blood pressure. Law et al. also suggested that a reduction in the amount of salt added to processed food by manufacturers and the stating of salt content on food labels could reduce dietary sodium intake by a further 50 mmol per 24 hours, with further public health benefits,[236] and lead to a greater reduction in the incidence of stroke and ischaemic heart disease.

Although the work of Law et al. provides support for a causal relationship between sodium intake and blood pressure, the issue remains controversial.

Apart from the effect on blood pressure, a high level of consumption of salt and salty foods is positively associated with stomach cancer.[137]

Dietary fat and fatty acids

Diets high in saturated fatty acids raise serum cholesterol levels, although not all saturated fatty acids are equally likely to cause hypercholesterolaemia. Diets high in stearic acid (C18:0) and short-chain saturated fatty acids (C<12) do not raise serum cholesterol; lauric acid (C12:0) and myristic acid (C14:0) are thought to be the major cholesterol-raising fatty acids.[238] Most opinion is that *trans*-fatty acids (resulting from processing polyunsaturates) tend to raise blood cholesterol levels.[82,83,239] Willett et al. calculated intakes of *trans*-fatty acids in the diets of 85 095 respondents in the Nurses' Health Study and found a relative risk for coronary heart disease of 1.50 between the highest and lowest quintiles; in 69 000 women who reported a stable margarine consumption over 10 years the relative risk was 1.67.[240] A cross-sectional study of middle-aged and elderly men indicated an independent association between *trans*-fatty acid intake and total serum cholesterol and LDL cholesterol; there was an inverse association with HDL cholesterol.[241] It should be noted that margarines manufactured in Australia have a lower level of *trans*-isomers than American margarines.[82]

Of two recent studies that included tests on palmitic acid (C16:0), one study suggested that palmitic acid may be neutral in relation to serum cholesterol levels[242] and the other showed an increase in HDL cholesterol compared with habitual fat intake.[243] Mono-unsaturated fatty acids (chiefly oleic acid, C18:1n-9) and n-6 polyunsaturated acids (chiefly linoleic acid, C18:2n-6) have a cholesterol-lowering effect.[82,83118,239] HDL cholesterol levels are lowered by linoleic acid but not by oleic acid.[238,239] Total fat intake may promote the development of hypertension,[87] but marine n-3 (ω-3) fatty acids may have a blood-pressure-lowering effect.[82,83] There is also an association between total fat intake and some forms of cancer.[137]

Gallstone disease risk differs with sex; polyunsaturated fatty acid intake may be positively associated with risk in men and negatively associated with risk in women.[158]

Dietary fibre

Diets high in fibre have been shown to lower systolic and diastolic blood pressure in hypertensive individuals.[105] Epidemiological evidence suggests that dietary fibre, and in particular soluble fibres (gums and pectin) found primarily in oats, legumes and fruit, have potential cholesterol-lowering properties, whereas insoluble fibres such as cellulose, hemicellulose, and lignin from grains and some vegetables generally do not.[244] Diets high in complex carbohydrates and water-soluble fibre may reduce blood cholesterol levels by 20–32 per cent and fasting triglycerides by 10–20 per cent.[105] Such intakes also appear to be protective against diverticular disease and constipation.[244]

Vegetable and cereal fibre intakes appear to have a protective effect against colorectal cancer.[137] A change to a vegetarian diet reduced systolic blood pressure in normotensive men,[245] but a low-fat, high-fibre diet in hypertensive men reduced blood cholesterol levels and not blood pressure.[246]

Dietary fibre intake may also be protective against gallstone formation, but the nature of the relationship between dietary fibre, cholesterol metabolism and gallstones is not well understood.[157]

Other dietary factors

Potassium intake may be inversely related to hypertension, suggesting a possible protective effect.[83] Witteman et al. have reported independent and significant inverse associations of both dietary calcium and magnesium with hypertension but no independent inverse association between potassium and hypertension.[154]

Wahlqvist et al.[239] draw attention to possible effects on serum lipids of other macronutrients—protein and carbohydrates—as well as the non-nutrient components of foods (see Section 4.6). Antioxidants, especially vitamins C and E, and carotenoids may also provide a beneficial effect by protecting LDL cholesterol against oxidation.[238,239]

5.5 Morbidity and mortality associated with diet-related disease

Morbidity

The 1989 National Heart Foundation Risk Factor Prevalence Survey and previous surveys in 1980 and 1983 are major recent sources of national anthropometric and physiological data, although the survey populations were limited to the capital cities. In 1989 height, weight, waist and hip circumference, diastolic and systolic blood pressure, and plasma triglycerides, total cholesterol and HDL cholesterol measurements were taken for all participants.[27]

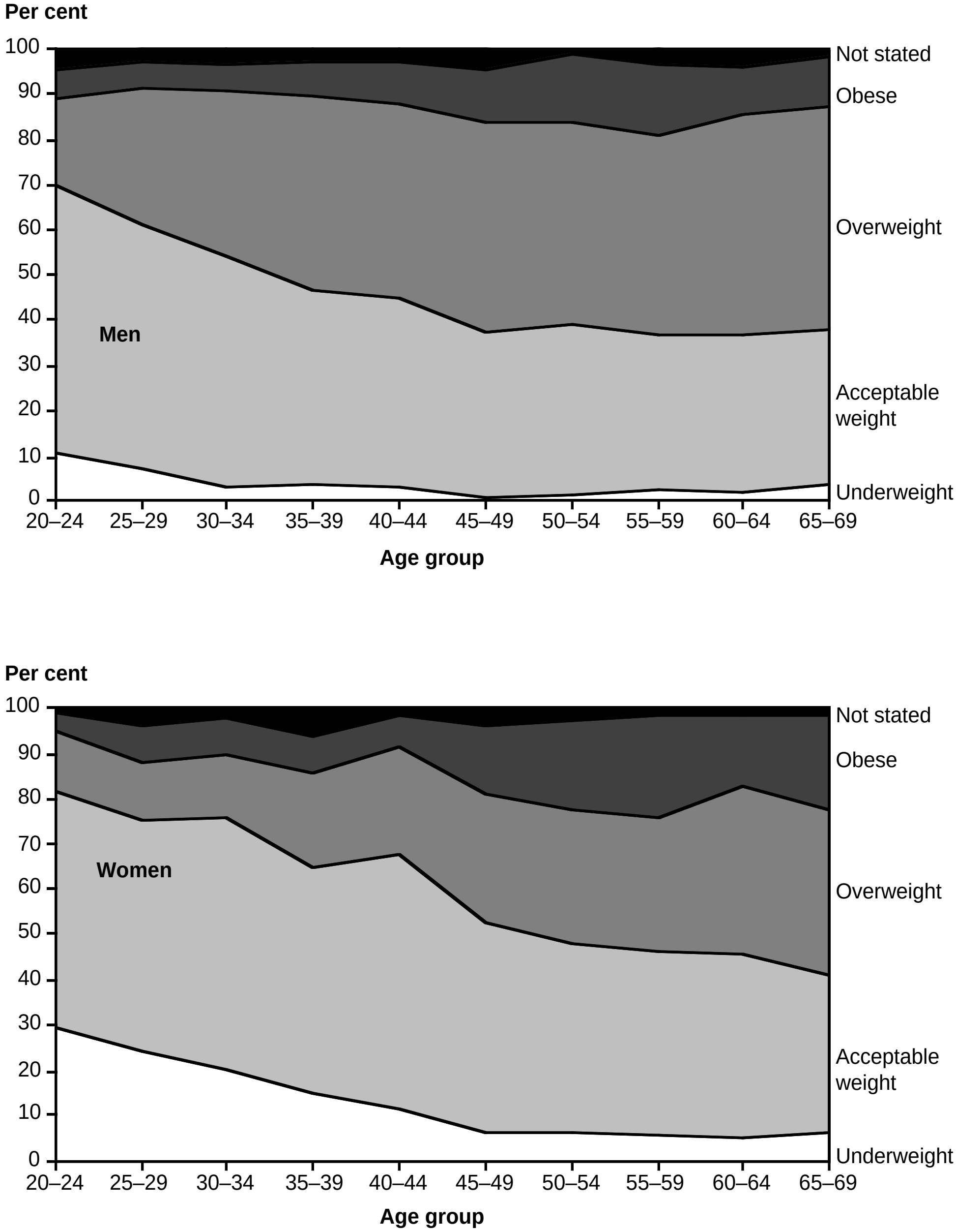
The prevalence of obesity generally increased with age (see Figure 5.3). Overall obesity was more prevalent among women than men.[27] Trends analysis data, corrected to allow direct comparison between the 1983 and 1989 surveys, show an increase in the prevalence of obesity (BMI >30 kg/m^2) of 15 per cent for women and slightly more for men to 11.1 per cent for women and 9.3 per cent for men; the prevalence of overweight (25< BMI <30 kg/m^2) increased by only 5–7 per cent to 22.4 per cent for women and 36.8 per cent for men.

Boyle et al. calculated waist-to-hip ratios for the 1989 survey, using waist and hip circumference measured over light street clothing and standardised sites for minimum waist and maximum hip circumferences.[164] Over 40 per cent of men surveyed in 1989 had a WHR of 0.90 or greater, while 5 per cent had a WHR of 1.00 or more. Almost 25 per cent of women had a WHR of 0.80 or more. These figures indicate that excessive WHR is more prevalent among urban Australians than is excessive BMI; that is, abdominal obesity is more prevalent.[164]

An estimated 17 100 people aged 25–69 years had a first heart attack during 1991; an additional 5400 had a second or subsequent heart attack. Of the 500 who had heart attacks in 1991, 76 per cent (17 000) were males.[247]

In the 1989 Risk Factor Prevalence Survey, those with hypertension, defined as having a diastolic blood pressure of 95 mmHg or more, and those who stated that they were taking tablets for blood pressure constituted 17 per cent of men; 13 per cent of women were found to be hypertensive (see Table 5.5). If the definition of hypertension was expanded to include those whose systolic blood pressure was 160 mmHg or more, 18 per cent of men and 14 per cent of women were hypertensive. The prevalence of hypertension increased steadily with age in both sexes.

An estimated 37 000 Australians per year have a stroke, with about half of these occurring in those aged more than 75 years.[247] The Perth Community Stroke Study found age-adjusted rates of 132 per 100 000 for men and 77 per 100 000 for women.[248]



Source: Risk Factor Prevalence Survey Management Committee[27]

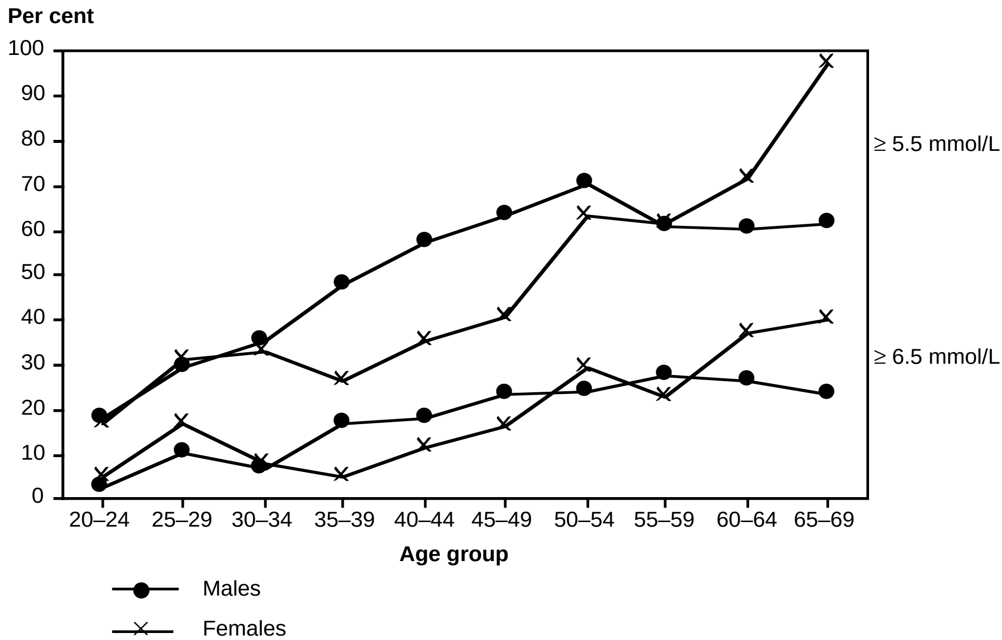
Figure 5.3: ***Body mass index, by age group and sex, 1989***

Hypercholesterolaemia, defined as having a total plasma cholesterol level of 6.5 mmol/L or more, was found in 16 per cent of men and 14 per cent of women; 47 per cent of men and 39 per cent of women had plasma cholesterol levels of 5.5 mmol/L or more. The prevalence of raised plasma cholesterol levels increased overall with age in both men and women (see Figure 5.4).

Diet-related cancers

In Australia, cancer incidence is 1.3 times higher for men than for women. The risk of cancer generally increases with age. Excluding non-melanocytic skin cancer, the most common types of cancers for men are lung, prostate, colon, bladder, rectal and stomach cancers and melanoma. For women the most common types of cancers are breast, colon, lung and rectal cancers, melanoma, and cancers of the cervix and uterus.

Between 1982 and 1985 in Australia the number of cancers in men increased from 24 637 to 28 243 (3.5 per cent) and the number of cancers in women rose from 21 071 to 24 431 (3.8 per cent). Small increases were observed in age-standardised incidence; the average annual change was 1.4 per cent for men and 1.7 per cent for women. The age-standardised incidence for men at 1985 was 305 per 100 000; for women it was 233 per 100 000.[249] Set against this general increase, diet-related cancers have changed in an inconsistent manner. Table 5.6 shows increases of 2.1 per cent for breast cancer and 5.8 per cent for male colon cancer and decreases in stomach cancer for both men and women.



Source: Risk Factor Prevalence Survey Management Committee 1990[27]

Figure 5.4: ***Raised plasma cholesterol, by age group and sex, 1989***

Table 5.5: ***Incidence of selected diet-related cancers, number, age-standardised rate[(a)] and annual rate of change,[(b)] by sex, Australia, 1985***

ICD-9 classification		Males			Females		
		Number	ASR	Annual change (%)	Number	ASR	Annual change (%)
151	Stomach	1220	13.1	–0.3	645	5.1	–3.6
152	Small intestine	50	0.5	–4.8	55	0.5	+10.6
153	Colon	2694	28.9	+5.8	2725	23.6	+2.4
154	Rectum, anus	1542	16.7	+0.6	1191	10.7	+1.0
156	Gall bladder	177	1.9	–1.7	250	2.1	+1.0
174	Female breast	–	–	–	5837	59.2	+2.1
179, 182	Uterus	–	–	–	1019	10.0	+10.7

– Not applicable

(a) Age-standardised to the World Standard Population

(b) Average annual rate of change in age-standardised incidence rate, 1982–85

Source: Australian Institute of Health and Welfare and the Australian Association of Cancer Registries[249]

Breast cancer has been shown to be more common among women of higher socioeconomic status but the reverse is true for its mortality. Urban areas have shown higher incidence rates for breast cancer than rural areas. Colon cancer has been shown to have an association with higher socio-economic status, whereas stomach cancer has been shown to have an inverse relationship. Cancers of the gall bladder and small intestine showed little urban–rural variation in an examination of Victorian cancer registry data.[137]

Cancer incidence varies between those born overseas and those born in Australia. Those born in the British Isles, Germany and Greece have a higher incidence of cancer of the stomach than the Australian-born. Breast cancer incidence is relatively high in Australian-born women (53 per 100 000 in New South Wales) but lower than in women born in the British Isles. Colon cancers for both men and women exhibit higher incidence among those born in the British Isles, Germany and Northern Europe. Cancer of the rectum, on the other hand, has shown high rates in men born in New Zealand and low rates in Italian and Southern European women.[249]

Hospital morbidity data

Each State collects information about hospitalisation: the process of admission and separation from hospital, length of stay and reason for admission. This statistical collection is an important data source for information on diet-related diseases, although it does have limitations that affect its usefulness as an indicator of morbidity. These include aspects of differential access to hospitals, provision of level and type of services, medical practice, and repeated admissions. There have been encouraging developments towards the standardisation of core data items and most States provide data to the Australian Institute of Health and Welfare for inclusion in national data bases. Recent data for New South Wales have been used in this analysis to provide an indication of the impact of diet-related diseases on the hospital system.

Coronary heart disease represents the main diet-related cause for hospitalisation (see Table 5.6). Cardiovascular disease was the major disease group, accounting for 41.2 per cent of hospital separations for diet-related diseases in 1991–92 (see Table 5.6 and Figure 5.5). Hospital separations related to digestive tract conditions—diverticular disease, constipation, haemorrhoids, non-alcohol-related liver/biliary disease and malabsorption—accounted for 33.3 per cent of separations. Diet-related cancers accounted for 7.8 per cent.

Age-standardised hospital separation rates for diet-related diseases were greater for males than females and increased between 1988–89 and 1991–92, the increase being greater for females (12.2 per cent) than for males (7.1 per cent) (see Table 5.8). Compared with men, women are more likely to be treated in hospital for cancers, digestive tract conditions, eating disorders and skeletal diseases. Men are more likely to be treated for heart disease and alcohol-related diseases.

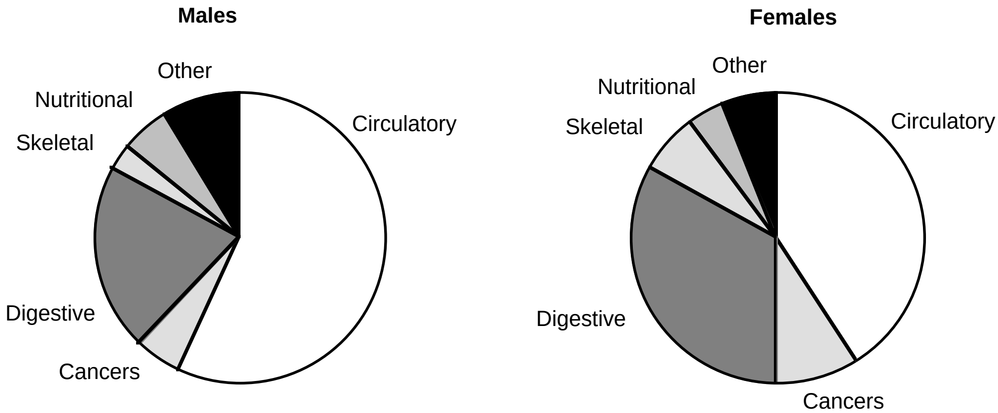
The hospital morbidity data base can be used to monitor trends in the use of hospital services for diet-related diseases and provides useful estimates to supplement morbidity data from regular health surveys and specific disease registers.

Table 5.6: ***Diet-related diseases, separations from New South Wales hospitals, 1991–92***

ICD-9 classification		Number	Per cent
410–414	Ischaemic heart disease	43 900	27.2
574–575	Gall bladder disorders	17 601	10.9
430–438	Cerebrovascular disease	15 313	9.5
428–429	Cardiovascular disease (excl. CHD)	12 596	7.8
558	Non-infective gastroenteritis and colitis	12 013	7.4
455	Haemorrhoids	9 320	5.8
562.1	Diverticular disease	7 805	4.8
820–821	Fracture of neck of femur	6 666	4.1
174–175	Cancer of the breast	4 363	2.7
783	Symptoms concerning nutrition, metabolism & development	3 802	2.4
521.0	Dental caries	3 759	2.3
401–405	Hypertensive disease	3 408	2.1
153	Cancer of the colon	3 330	2.1
250	Diabetes mellitus (NIDDM)	3 054	1.9
564.0,7	Constipation	2 507	1.6
443, 459	Peripheral vascular disease (excl. athero)	2 337	1.4
154	Cancer of the rectum	2 093	1.3
291	Alcoholic psychoses	1 274	0.8
151	Cancer of the stomach	1 137	0.7
733.1	Vertebrae collapse	957	0.6
440	Atherosclerosis	773	0.5
182	Cancer of the endometrium	614	0.4
003–005, 009	Intestinal infectious diseases	521	0.3
733.0	Osteoporosis	484	0.3
303	Alcohol dependence syndrome	435	0.3
156	Cancer of the gall bladder	300	0.2
278.0	Obesity	285	0.2
260–269	Nutritional deficiencies	169	0.1
281	Other deficiency anaemias	154	0.1
152	Cancer of the small intestine	100	0.1
988	Toxic effect of noxious substances eaten as food	94	0.1
366.41	Diabetic cataract	88	0.1
270.1, 271.3	Other nutritional disorders	72	–
280	Iron deficiency anaemias	4	–
	All diet-related diseases	**161 329**	**100.0**
	All other diseases	**1 068 893**	
	All diseases	**1 230 221**	

Note: Numbers may not add exactly due to rounding

Source: Australian Institute of Health and Welfare (unpublished data)



Source: Australian Institute of Health and Welfare (unpublished data)

Figure 5.5: ***Distribution of hospital separations for diet-related diseases, New South Wales, by sex, 1991–92***

Table 5.7: ***Diet-related diseases, age-standardised separation rates for New South Wales hospitals,[(a)] all ages, 1988–89 and 1991–92***

	Rate per 100 000 population	
Type of disease	**Males**	**Females**
1988–89		
Circulatory disease	540.7	322.6
Cancers	49.2	82.3
Skeletal diseases	37.1	55.2
Digestive diseases	156.0	227.4
Nutritional diseases	56.9	52.9
Other diet-related diseases	82.0	40.9
All diet-related diseases	**882.4**	**782.3**
1991–92		
Circulatory disease	608.2	358.7
Cancers	51.5	86.2
Skeletal diseases	36.0	57.1
Digestive diseases	156.0	311.8
Nutritional diseases	51.3	39.6
Other diet-related diseases	82.0	58.9
All diet-related diseases	**1033.0**	**910.8**

(a) Age-standardised to 1988–89 New South Wales population
Source: Australian Institute of Health and Welfare (unpublished data)

Mortality

The relative impact of coronary heart disease and stroke differs between men and women, breast cancer is a significant cause of death among women, and death from alcohol-related diseases is more prominent among men. Men and women are at equal risk of dying from cancer of the colon and diabetes mellitus (see Table 5.8).

The likelihood of death from diet-related diseases increases with age, with the partial exception of acute intestinal infections (see Table 5.9).

Recent national trends

Australia's death registration system (see Box 5.4) can be used to monitor mortality associated with diet-related disease. The Australian Institute of Health and Welfare has recently implemented a comprehensive mortality surveillance system, which examines the most recent Australian mortality data for evidence of trends and unexpected observations, using a relatively fine classification of diseases that includes the major diet-related diseases. Mortality rates from most cardiovascular diseases have fallen in recent years (see Table 5.8), as have rates for several diet-related cancers. Breast cancer mortality has shown a small but persistent increase among women. Death rates from cirrhosis of the liver have fallen for men and women. Diabetes mellitus mortality rates have remained stationary.

Table 5.8: ***Mortality from selected diet-related diseases, number and annual rate of change,***(a) ***by sex, Australia, 1991***

ICD-9 classification		Number			Annual change (%)	
		Males	Females	Persons	Males	Females
Cardiovascular diseases						
410–414	Ischaemic heart disease	16 753	13 570	30 323	–3.5*	–2.4*
430–438	Cerebrovascular disease	4829	7054	11 883	–4.7*	–4.7*
401–405	Hypertensive disease	408	621	1029	–5.5*	–4.7*
440	Atherosclerosis	312	564	876	–10.8*	–9.5*
443	Other peripheral vascular disease	243	246	489	1.2*	2.2*
Cancers						
153	Cancer of colon	1583	1519	3102	0.2	–1.4*
174	Cancer of female breast	na	2513	2513	na	0.5*
151	Cancer of stomach	782	496	1278	–3.8*	–3.5*
154	Cancer of rectum and anus	654	473	1127	–1.1*	–2.3*
156	Cancer of gall bladder	139	222	361	–1.9*	–0.9
182	Cancer of body of uterus	na	220	220	na	–1.4
152	Cancer of small intestine	40	31	71	1.3	1.0
Alcohol-related diseases						
571	Cirrhosis of liver	798	296	1094	–2.8*	–3.1*
303	Alcohol dependence syndrome	147	38	185	1.0	0.1
291	Alcoholic psychoses	33	5	38	1.7	0.3
Diabetes mellitus						
250	Diabetes mellitus	1113	1175	2288	0.7	–0.6

* Statistically significant ($p<0.01$)

(a) Average annual rate of change in age-standardised mortality rate, 1980–91

Source: Australian Bureau of Statistics; Australian Institute of Health and Welfare (unpublished data)

Table 5.9: ***Number of deaths in Australia due to intestinal infectious disease (ICD-9:001–009), by age group, 1979–1990***

Age	1979	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990
<1	18	16	16	14	8	9	8	3	3	2	2	2
1–4	12	5	8	8	4	2	5	3	3	6	3	1
5–14	1	1	4	1	–	–	–	–	2	1	–	–
15–54	6	4	1	6	5	1	3	3	4	6	5	4
55–74	16	25	13	10	7	11	12	13	20	13	14	10
75+	35	27	42	30	30	18	26	25	20	25	18	29
Total	**88**	**78**	**84**	**69**	**54**	**41**	**54**	**47**	**52**	**53**	**42**	**46**

– Zero

Source: Bennett et al.[83]

The surveillance system covers males and females separately and provides age-specific estimates of trends for all age groups. The system could usefully be integrated into a national food and nutrition monitoring and surveillance system.

Box 5.4 : Registration of deaths in Australia

Registration of deaths in Australia is the responsibility of the State Registrars of Birth, Deaths and Marriages. Information on the cause of death is supplied by the medical practitioner certifying the death or by a coroner. Other information about the deceased person is supplied by a relative or other personal acquaintance or by an official of the institution in which the death occurred. The information is provided by the Registrars to the Australian Bureau of Statistics for compilation into aggregate statistics.

Registration of death is a legal requirement and is virtually complete. With the exception of deaths of foreign diplomatic personnel, all deaths occurring within Australia come within the scope of the collection. Published mortality statistics generally relate to the year of registration of the death rather than the year of occurrence—usually 5–6 per cent of deaths are not registered until the following year or later.

Nairn et al. provide a detailed description of the data collection process in their study of the quality of the cause of death data in young Australian men.[250]*The Australian Bureau of Statistics employs a variety of control measures to ensure that the statistics are as reliable as possible, among them seeking further information where necessary to enable accurate classification of the underlying cause of death, check-coding of the cause of death, detailed computer editing of the data, and checks on statistical output at the individual record and aggregate levels. Nevertheless, the data are not error free (the Bureau detailed the potential sources of error in a 1989 review of the causes of death collection.* [251]

More recently attention has been given to the accuracy of death certificates for specific causes such as diabetes,[252] *testis cancer*[253]*and AIDS.*[254]*Maclaine et al. compared clinical diagnoses with autopsy findings in the Australian Capital Territory and found a discrepancy rate of 23 per cent.*[256] *Appreciable discrepancies were also found in an audit of a metropolitan hospital.*[256]

Despite these limitations, the National Death Register is potentially a powerful tool for clarifying the links between mortality and dietary intakes or nutritional status.

5.6 Quantifying the relationship between diet and disease

Measuring attribution

The fundamental epidemiological statistic that is necessary to quantify the direct relationship between a risk factor of interest and disease is the 'population attributable fraction', which has been defined as the proportion of total illness or ill-health events in a population that could be prevented if a particular risk factor were eliminated.

The population attributable fraction reflects the overall impact of a risk factor on morbidity and mortality in a specified population. It can thus be interpreted from an aetiological 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'. Risk attributable to diet in the Melbourne Colorectal Cancer Study was calculated to be 46 per cent where there were at least five dietary risk factors; the risk attributable to beer consumption for rectal cancer was 11 per cent in women and 31 per cent in men.[257] Holman et al. have calculated population attributable fractions, which they have termed 'aetiological fractions', on a detailed age–sex breakdown for alcohol; these can be found in Crowley et al.[258] as well as in the original publication.[211]

A recent study jointly undertaken by the Australian Institute of Health and Welfare and the National Centre for Health Program Evaluation derived population attributable fractions for the diet-related diseases.[258] The study relied on a sensitivity analysis to provide a range of estimates based on differing assumptions about the values that the population attributable fractions are likely to take. Rather than a single value being given for that part of each disease attributable to diet, three estimates were given. The high and low estimates indicate the probable upper and lower bounds; the mid-value represents the most probable value. The setting of these upper and lower bounds was based on a number of criteria, including empirical evidence from epidemiological studies, current practice in the literature, and judgements from experts in the field. As Table 5.10 shows, for most conditions population attributable fraction ranges are broad.

The cost to the community of diet-related disease

Crowley et al. estimated the economic cost of diet-related coronary heart disease, hypertension, atherosclerosis, stroke, non-insulin-dependent diabetes mellitus, some cancers (stomach, colon, rectal, breast and endometrial), osteoporosis, diverticular disease, haemorrhoids, constipation, dental caries, gall bladder disease, and iron deficiency anaemia.[258] The estimates include both direct health care costs attributable to diet in 1989–90 (costs of hospitals, medical expenses, allied health professional services, pharmaceutical expenses and nursing homes) and indirect costs (costs due to sick leave and the net present value of forgone earnings due to premature death). Nevertheless, these costs do not take into account all diet-related diseases, nor can they account, for example, for effects such as exacerbation of musculoskeletal or respiratory dysfunction due to obesity. Nor were all cost categories estimated. Two categories not included were the costs accrued to individuals in time spent in diagnosis or treatment and the costs incurred by family and friends in visiting or caring for the ill or disabled.

Estimates of the direct health care costs attributable to diet in 1989–90 were calculated using the high, middle and low population attributable fractions (see Table 5.10). Similarly, total costs—that is, direct and indirect costs—of diet-related disease in 1989–90 are provided (see Table 5.11). Using the mid-value population attributable fractions, the direct cost of diet-related disease is estimated to be $1432 million in 1989–90. The indirect cost is $605 million in 1989–90, giving total costs of $2037 million.

Table 5.10: ***Proportion of disease onset attributable to diet (per cent)***

	Range of estimates		
Type of disease	**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			
Stomach	–	50	15
Colon	–	35	15
Rectum	–	35	15
Breast	–	30	10
Endometrium	–	25	10
All cancers	35	–	–
Osteoporosis	30	20	10
Diverticular disease	75	50	25
Haemorrhoids	75	50	25
Dental caries	75	50	25
Gall bladder disease	75	50	25
Constipation	75	50	25
Iron deficiency anaemia	75	50	25

– Not applicable

Source: Crowley et al.[258]

Alcohol-related disease

The total cost of alcohol-related disease was revised in 1993, to give a 1989–90 estimate of $1243 million,[259] giving a total cost for diet- and alcohol-related disease based on the mid-value population attributable fraction of $3280 million.

These costs are likely to underestimate the true cost of alcohol-related morbidity and death. Collins and Lapsley estimated the cost at $6000 million in 1989–90;[260] Crowley and Richardson had a higher estimate of $6700 million to $17 400 million in 1989–90.[262] The major differences between estimates depend on what may be included in indirect costs to industry, estimated in several overseas studies to be 50–80 per cent of total costs. These costs derive from absenteeism, staff turnover and reduced productivity, and indirectly from unemployment.

Obesity

The Australian Institute of Health and Welfare extended the earlier analysis of costs by Crowley et al.[258] to include an estimate of the costs of obesity. This was based on United States studies of the cost of obesity-related disease. Conditions included were non-insulin-dependent diabetes mellitus, cardiovascular disease, hypertension, gall bladder disease, and colorectal and breast cancers, and the cost was estimated by Colditz at 5.5 per cent of the cost of illness in the United States in 1986.[261]

Table 5.11: ***Direct and indirect health care costs attributable to selected diet-related diseases, middle estimate, 1989–90 ($ million)***

	Cost		
Type of disease	**Direct[(a)]**	**Indirect[(b)]**	**Total**
Coronary heart disease	167	268	435
Hypertension	211	44	255
Atherosclerosis	12	2	14
Stroke	163	69	232
Diabetes mellitus (non-insulin dependent)[(c)]	139	53	192
Cancers			
Stomach	12	24	36
Colon	16	42	58
Rectum	13	15	28
Breast	16	49	65
Endometrium	3	2	5
All cancers	61	132	193
Osteoporosis and fractures	37	1	38
Diverticular disease	23	4	27
Haemorrhoids	25	10	35
Dental caries	518	1	519
Gall stones	50	17	67
Constipation	15	2	17
Iron deficiency anaemia	12	2	14
Alcohol-related disease	430	813	1243
Total	**1862**	**1419**	**3280**

Note: Figures may not add exactly due to rounding

na Not available

(a) Costs include public and private hospital, medical, pharmaceutical, nursing home and allied professional

(b) Costs include sick leave and forgone earnings

(c) Includes diabetes complications

Source: Australian Institute of Health and Welfare; National Centre for Health Program Evaluation (unpublished data)

The Australian estimate of the direct costs of obesity was derived by combining Colditz's relative risk estimates with Australian prevalence data. The health care costs of treating obesity were also estimated, and the total cost in 1988–89 was estimated at $672 million, with obesity-related coronary heart disease and hypertension accounting for 62 per cent of that amount. The cost of treatment of obesity within the health care system was $393 million.[259]

The obesity estimate did not include the costs incurred through use of commercial weight-loss organisations or of special foods or other products used to reduce weight. The Consumer Advocacy and Financial Counselling Association of Victoria estimates that approximately 300 000 people purchase commercial weight-control programs each year, at a cost of $500 million.[263] The additional cost of anorectic agents, most of which do not fall within the Pharmaceutical Benefits Scheme, could be up to $18 million per year. This estimate is based on an estimated 410 000 prescriptions filled for such agents in 1989–90 and an average cost of $45 per prescription.[264] A further cost to be considered is through the fitness industry; the attributable fraction of this cost due to obesity will be offset by savings through improved health, but the overall amount is unknown. These costs of obesity fall directly on individuals in the community; few other morbid conditions incur such a high personal cost.

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