The relationship between overweight, obesity and cardiovascular disease

A literature review prepared for the National Heart Foundation of Australia

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Preface

The National Heart Foundation of Australia commissioned the Australian Institute of Health and Welfare to undertake the literature review contained in this publication to bring together the diverse material dealing with the impact of excess weight on cardiovascular disease. A range of policy responses to the growing prevalence of excess weight in Australia are now being developed and it is therefore important that the evidence for the relationship between excess weight and cardiovascular disease is well understood.

The literature review covers a large amount of peer-reviewed research, and stands as a tribute to the expertise and commitment of the researchers involved.

While the review was in progress, there was substantial national publicity and debate in the print and electonic media over the health impacts of excess weight. In that context, this thorough review will assist and even stimulate further informed debate.

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This report was prepared by Joanne Davies (Project Co-coordinator), Bonnie Field and Toni Hunt. It was originally prepared in August 2003 for the National Heart Foundation of Australia, and has been revised for publication in 2004.

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1 Summary

This report presents a review of the literature evaluating the association between overweight and obesity and cardiovascular disease (CVD), and whether the relationship is direct or indirect. In many instances the report examines the relationship between CVD or its metabolic risk factors and 'excess body weight', meaning all degrees of overweight taken as one group. This is because the literature does not always focus on traditional categories such as 'overweight' and 'obesity'.

The review draws on major Australian and international studies and data to examine the relationship between excess body weight and CVD, and its major biomedical risk factors. The main findings in this report, based on the literature reviewed in these studies, are summarised below.

1.1 Evidence relating to major biomedical risk factors

The literature reviewed in this study suggests that:

There is **good evidence** of an association between:

- excess body weight and:
  - atherosclerosis
  - high blood pressure, including in children and adolescents
  - high total blood cholesterol, high LDL cholesterol and low HDL cholesterol
  - Type 2 diabetes, particularly central adiposity
- overweight and obesity and:
  - Type 2 diabetes among Aboriginal and Torres Strait Islander peoples.

There is **moderate evidence** of an association between:

- excess body weight and:
  - atherosclerosis in men compared with pre-menopausal women
  - high blood pressure in women compared with men, and younger men compared with older men
  - high total blood cholesterol (and high LDL cholesterol) in younger adults versus older adults
  - high total blood cholesterol (and high LDL cholesterol and low HDL cholesterol) in children and adolescents
  - Type 2 diabetes in children and adolescents
- duration of excess body weight and:
  - Type 2 diabetes in men.

There is **little or weak evidence** of an association between:

- central adiposity and:
  - high blood pressure.

1.2 Evidence relating to cardiovascular disease

The literature reviewed also suggests that:

There is **good evidence** of an association between:

- overweight and obesity and:
  - CVD incidence in young to middle-aged adults
  - coronary heart disease (CHD) incidence in adults.

There is **moderate evidence** of an association between:

- overweight and obesity and:
  - heart failure incidence in adults
  - ischaemic stroke incidence in adults
- obesity (including duration of obesity) and:
  - CVD mortality
- abdominal obesity and:
  - CVD incidence in older men
  - CHD incidence in older men and younger women
  - stroke incidence.

There is **little or weak evidence** of an association between:

- overweight and obesity and:
  - CVD incidence among Aboriginal and Torres Strait Islander peoples
- obesity and:
  - CHD incidence among children and adolescents (particularly among girls) unless the children become obese adults.

The literature reviewed in this study points to a relationship between excess body weight, in particular obesity, and CVD. The relationship links excess body weight with CVD both indirectly as an independent risk factor for the established biomedical risk factors (atherosclerosis, high blood pressure, high blood cholesterol and Type 2 diabetes) and directly as an independent risk factor for CVD, in particular CHD.

The major gaps and deficiencies in the literature relate to 'at risk' populations such as people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people and migrant groups.

Other issues that may require further examination are the effect of the duration of being overweight or obese, location of body fat (such as central adiposity) and extent of excess body weight. It should be noted that most studies in this review did not consider the role of

physical activity and physical fitness as mediating factors in the development of CVD or its risk factors.

2 Introduction

The World Health Organization (WHO) has called the worldwide rise in obesity a global epidemic (WHO 2000) and Australia has not escaped the trend. Rates in Australia have risen dramatically over the past few decades. Around one in five Australian adults was estimated to be obese in 1999–2000, and around two in five overweight but not obese (AIHW 2004a).

The high prevalence of excess weight is not restricted to adults. Levels of child and adolescent overweight and obesity are also on the rise. In Australia, it is estimated that around 5% of children and adolescents are obese (body mass index (BMI) ≥ 30) and 20% are overweight (BMI ≥ 25) (AIHW 2004b).

With excess body weight comes the risk of long-term health consequences. Overweight and obesity is associated with the morbidity and mortality of many health conditions, such as CHD, Type 2 diabetes, gall bladder disease, ischaemic stroke, osteoporosis, sleep apnoea and some types of cancers (WHO 2000). Although the focus of this review is on the relationship between overweight and obesity and CVD, this list of conditions highlights the impact excess body weight has on overall morbidity and mortality; overweight (BMI ≥ 25) is estimated to account for over 4% of the total burden of disease in Australia in 1996 (AIHW: Mathers et al. 1999).

Obesity is associated with a substantial reduction in life expectancy. Recent data from the United States suggest that a severe level of obesity (BMI > 45) during early adulthood (aged 20–30 years) may reduce a man's life expectancy by up to 13 years and a woman's by up to 8 years (Fontaine et al. 2003).

Obesity in childhood and adolescence is a major concern, not only because of the health and social problems in the short term but also because of the increased risk of it continuing into adulthood with associated long-term health effects.

While many factors influence excess body weight, at a basic level overweight and obesity results from a sustained energy imbalance. This occurs when energy intake from the diet exceeds energy expenditure from physical activity and metabolic processes over a considerable period (WHO 2000). Even a slight imbalance over the long term can result in increased weight. The result of excess energy intake relative to energy expenditure is the storage of unused energy as body fat.

2.1 Prevalence of overweight and obesity in Australia

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab) estimated that over 2.6 million (21% of the population aged 25 years and over) were obese (BMI ≥ 30)—this is more than double the rate observed in 1980. When looking at the broader group who are overweight (BMI ≥ 25, which includes the obese group), the same survey estimates that 7.5 million Australians aged 25 years and over (or 60%) are affected.

Men (67%) were more likely to be overweight or obese than women (52%). The proportion of overweight or obese people increased across age groups and peaked at 55–74 years for men (74%) and 65–74 years for women (71%) (AIHW 2002).

Some subgroups in Australia are more at risk of being overweight or obese. Overweight or obesity is more common among women in lower socioeconomic groups and women living in remote areas. Aboriginal and Torres Strait Islander peoples are more likely to be obese than other Australians (AIHW 2001).

Between 1985 and 1997, the prevalence of overweight and obesity doubled among young Australians. Further, obesity alone trebled over the same period among young people (Booth et al. 2003).

2.2 The problem of cardiovascular disease

CVD is Australia's biggest health problem. Based on self-reports from the National Health Survey, an estimated 3.7 million Australians or 19.4% of the population had a current cardiovascular condition. Cardiovascular disease kills more people than any other disease (accounting for 39% of all deaths in 2000) and it accounts for the largest share of health expenditure (AIHW 2004a). Much of the death, disability and illness caused by CVD is preventable.

Many Australians remain at higher risk of CVD through tobacco smoking, being physically inactive, eating a diet high in saturated fats and low in unsaturated fats (Cobiac et al. 2000), being overweight, or having high blood cholesterol or high blood pressure (BP).

A report by Bonow and colleagues (2002) highlighted three factors that could serve to accelerate the rate of CVD and its complications. First is the ageing of the population. Second is the dramatic increase in the rate of obesity and Type 2 diabetes and their related complications of hypertension, hyperlipidaemia and atherosclerotic vascular disease. Finally, there is evidence emerging that risk factors are clustering in young people.

2.3 Aim of this review

Growing awareness of the public health significance of the obesity epidemic has evoked strong interest within and outside the health sector. The recent interest has included the establishment of a National Obesity Taskforce, the running of state-organised obesity forums, and regular newspaper and journal articles on the epidemic (Swinburn 2003). It is timely, then, that the relationship between overweight and obesity and CVD is examined.

The aim of the review is to assess how overweight and obesity relates to CVD, either directly or indirectly. The review draws on major Australian and international studies and data to examine the relationship between overweight and obesity and:

- CVD incidence and mortality
- the major biomedical CVD risk factors.

Particular emphasis is placed on:

- the strength of the association of overweight and obesity with CVD, i.e. its strength as a risk factor
- identifying elements of the association between excess body weight and CVD, such as abdominal obesity and duration of overweight and obesity
- population subgroups at most risk.

The report highlights gaps and deficiencies within the identified literature to assist in determining potential future research avenues.

2.4 Structure of the report

The report is divided into six sections. Sections 1 and 2 are the summary and introduction, respectively. Section 3 describes the methodology used in this review. Section 4 reviews evidence of the association between overweight and obesity and established biomedical risk factors for CVD. Section 5 summarises the current literature examining the association between overweight and obesity and CVD. CVD is discussed in terms of its major components: CHD, stroke and heart failure. Section 6 concludes with recommendations and priority research areas based on gaps and deficiencies in the literature identified by the reviewers. References (Appendix A) and supplementary tables listing included studies (Appendix B) are included at the back of the report.

3 Methods

3.1 Measures of body weight

This section describes the major measures of overweight and obesity used in this report.

Total adiposity

Overall or total adiposity is most commonly measured using the BMI. This is an index of a person's weight relative to their height, and is considered to be a reasonable reflection of overall body fat for most people. BMI is not gender-specific. It is calculated as a person's weight in kilograms divided by the square of their height in metres. BMI does not directly measure percentage of body fat, but it provides a more accurate measure of overweight and obesity than relying on weight alone (NIH 1998).

The WHO has defined categories of BMI based on evidence of increased risk of chronic disease and mortality (Table 1) (WHO 2000) and these categories have recently been adopted as the national standard in Australia (NHDC 2003). This classification is used for people aged 18 years and over, and is not suitable for children and adolescents. In addition, the specific cut-offs shown in Table 1 may not be suitable for all ethnic groups, who may have equivalent levels of risk at a lower (e.g. Asians) or higher (e.g. Pacific Islanders or Polynesians) BMI (Craig et al. 2001; Deurenberg et al. 1998; WHO 2000).

The classification is not suitable for children and adolescents due to age-related growth patterns. Consequently, the International Obesity Task Force has developed internationally accepted cut-off points for BMI which are age-and sex-specific for children aged 2 to 17 years for use in population surveys (Cole et al. 2000; NHDC 2003).

Other measures of excess body weight include absolute body fat, relative body fat (i.e. percentage of body fat) and ratio of actual weight to desirable weight (or the Metropolitan Relative Weight, MRW).

Table 1: Classification of BMI for people aged 18 and over

BMI (kg/m^2)	Classification
< 18.5	Underweight
18.5–24.9	Healthy weight range
≥ 25	Overweight
25.0–29.9	Preobese
≥ 30	Obese
30.0–34.9	Class I obesity
35.0–39.9	Class II obesity
≥ 40	Class III obesity

Sources: WHO 2000; NHDC 2003.

Central adiposity

It has been suggested that central adiposity—where fatness is heavily concentrated around the waistline—is a better measure of disease risk than overall or total adiposity. However, it is difficult to assess which is the better measure of disease risk when total adiposity and central adiposity are so highly correlated (Kuller 1999; Obarzanek 1999).

Certain abdominal skinfold measures, waist-to-hip ratio (WHR) and waist circumference are considered to be markers of central adiposity or intra-abdominal fat. More recently, newer techniques such as scans of visceral fat tissue have been shown to be good measures of central adiposity as well. These newer techniques, although providing more precise measures of body fat, are costly and impractical for use in many research studies or population surveys (WHO 2000; Kuller 1999).

Similar to BMI, health risks increase as waist circumference increases. BMI is used more frequently, particularly in self-report surveys, since people are much more likely to be able to report or estimate their height and weight rather than their waist circumference. Current evidence suggests that, for Caucasians, waist circumferences greater than 94 cm in men and greater than 80 cm in women indicate increased risk (WHO 2000). Waist circumferences greater than 102 cm (men) and 88 cm (women) indicate substantially increased risk. As with BMI, this classification is not suitable for use in people aged less than 18 years and, again, the cut-off points were developed for Caucasians and may not be suitable for all ethnic groups.

3.2 Scope

The association with CVD is explored in terms of overall associations with CVD incidence and mortality, and then in terms of the major components of CVD including CHD, stroke and heart failure. The association with the major biomedical CVD risk factors is also examined.

Where possible, the following population subgroups are examined: male and female, age groups (children and adolescents, early adulthood, middle age, older age), socioeconomic status, Aboriginal and Torres Strait Islander peoples, and selected migrant groups (Pacific Islander peoples, South-East Asians, Mediterraneans).

3.3 Literature identification

Articles included in this review were identified by Medline and ProQuest searches covering the period 1 January 1990 to 30 June 2003. Keywords used were 'BMI', 'overweight', 'obesity', 'cardiovascular disease' and 'heart disease'. Articles were also identified from key reviews on the topic.

Relevant studies were evaluated according to whether the study met the following inclusion criteria:

- literature inclusion criteria:
  - published studies and reviews
  - measured anthropometric data only
  - sufficiently large sample sizes (≥ 1,000 individuals). Smaller studies were included if particularly relevant, such as for population subgroups.
- literature exclusion criteria:
  - how a person became overweight or obese, i.e. nutrition or physical activity levels
  - mechanisms underlying overweight and obesity
  - weight loss and weight cycling (losing and gaining weight repeatedly).

Results from some Australian studies which may not fit these criteria exactly have been included as a means of representing local findings.

3.4 Appraising and summarising the strength of evidence

Evaluation of the evidence centred on several criteria (adapted from NHFA 2003):

- consistency of evidence over a range of study designs
- the quality of the evidence, relating to minimising measurement bias
- the size of the effect
- relevance of the evidence to the Australian population.

Table 2: Appraisal criteria

	Good evidence	Moderate evidence	Little or weak evidence
Level of evidence	Consistency across a range of study designs and measures of overweight and obesity	Inconsistency across a range of study designs; use of surrogate measures; limited number and type of studies	Inconsistency across a range of study designs and measures of overweight and obesity; small number and type of studies
Quality of the evidence	Measurement bias minimised	Limited in quality	Limited in quality
Magnitude of association	Statistically significant	Effect possibly due to measurement bias	Effect possibly due to measurement bias

Source: Adapted from NHFA 2003.

Further criteria that are useful in appraising causality include the presence of a dose–response relationship, the correct time sequence between exposure and outcome, independence and specificity of the effect, and the effect of introducing or removing exposure to the factor. With the exception of the latter criteria each of these are used throughout the review: the dose–response relationship via analysis of the extent of excess body weight and the health outcome; the time between exposure and outcome via a focus on prospective studies conducted over many years; and excess body weight as an independent risk factor for CVD via the extent to which confounding variables are considered in each study. The effect of introducing or removing the exposure (i.e. weight loss or gain) was considered more problematic to include in the review because of additional confounding factors such as baseline BMI and the extent, rate and duration of weight loss, gain or maintenance. The authors acknowledge, however, that this is an important criterion and, if considered, would add to the evidence for the association.

In line with Table 2, at the end of each section this review reports the strength of evidence between overweight and obesity and the CVD-related factor of interest as either good, moderate or little/weak.

4 The association between excess body weight and other major established biomedical CVD risk factors

This section examines whether there is an association between excess body weight (or body fatness) and the development of CVD through its underlying pathology. Links with 'overweight and obesity' are made if there is sufficient evidence from the studies examined that a level of excess body weight equivalent to the definitions of overweight and obesity (described in Section 3.1) have been assessed; if not, the term 'excess body weight' is used.

First, the association between excess body weight and atherosclerosis, the underlying mechanism for CVD, is examined. Second, its association with the traditional biomedical CVD risk factors is examined. Lists of studies used in this review are presented in Appendix B.

4.1 Atherosclerosis

This section examines the relationship between excess body weight and the development of atherosclerosis. Atherosclerosis is the process that gradually clogs arteries, through fatty and fibre-like deposits that build up on the inner walls of the arteries. It is the main underlying condition for CVD. Typically studies which assess atherosclerosis are small. They use CT scans of coronary arteries or investigate coronary arteries or tissues in autopsy examination. In the latter, assessment of coronary risk factors is done retrospectively. Studies identified in this review are presented in Appendix B Table A1.

A cross-sectional study by Takami et al. (2001) of 849 Japanese men aged 20–78 years investigated the relationship between body fatness (particularly abdominal fat) and carotid atherosclerosis. They found that general adiposity (as measured by BMI), waist circumference, WHR, abdominal subcutaneous fat and intra-abdominal fat were all correlated with carotid intimal–medial thickness (IMT) after adjustment for age and smoking habit. Adjustment for general adiposity eliminated all other associations except WHR with IMT, indicating that abdominal fat is not as strongly associated with atherosclerosis as is general body fatness.

The Progetto ATENA study is a large (over 5,000 participants) ongoing investigation of the causes of CVD and cancer in Italian females aged 30 to 69 years. Rubba et al. (2001) examined a subsample of 310 females from within this study and showed that higher BMI (29 versus 27) was associated with common carotid artery plaques, after adjusting for age. De Michele et al. (2002) also reported on a subsample of 310 women from the study and concluded that BMI and WHR were significant predictors of carotid wall thickness independent of other cardiovascular risk factors (age, BP, lipid abnormalities, and fasting insulin). The investigators used ultrasound examination of carotid IMT as a marker of early atherosclerosis. As BMI increased, IMT increased along with other coronary risk factors

(systolic blood pressure (SBP), diastolic blood pressure (DBP), triglycerides, fasting glucose, insulin and lower high-density lipoprotein (HDL) concentrations).

Berenson et al. (1992) showed that atherosclerosis of the aorta and coronary arteries were related to obesity in youth. Autopsies were conducted on 150 children and young adults aged 6 to 30 years in the Bogalusa Heart Study. The study revealed that obesity was associated with the early development of atherosclerotic lesions as evidenced by fatty streaks and/or fibrous plaque lesions, particularly among young men.

Mahoney and colleagues (1996) examined coronary artery calcification in young adults in the Muscatine Study. The longitudinal study measured coronary risk factors in 384 children (mean age 15 years) and twice during adult life (mean ages 27 and 33 years). They used electron beam computed tomography to measure coronary artery calcification as a marker of the atherosclerotic process. They found that increased BMI and triceps skinfold thickness during childhood was more strongly associated with the presence of coronary artery calcification in young adult men than women.

A study by McGill and others (2002) examined early stages of atherosclerosis in the arteries and tissues from around 3,000 people aged 15–34 years who had died from injuries or poisoning. They found that obesity (BMI ≥ 30) and thick panniculus adiposus (central pattern of obesity) were related to accelerated coronary atherosclerosis in adolescent and young men, but not women. When adjustment was made for standard CVD risk factors (cholesterol, smoking and high BP) the size of the effect was reduced by around 15%.

A commentary by Grundy (2002) concluded that the effects of other risk factors, although underestimated in McGill's study, did not detract from the strong and independent association of obesity as a risk factor for coronary atherosclerosis, at least for men. He suggested two reasons why the effects of obesity on atherosclerosis were not observed in women. First, premenopausal women generally have a delay in the progression of atherosclerosis. Second, men generally have a higher accumulation of abdominal adipose tissue that may predispose to higher atherosclerotic risk.

4.1.1 Strength of evidence

The literature reviewed in this study suggests that:

- There is good evidence of an association between excess body weight and atherosclerosis.
- There is moderate evidence that an association between excess body weight and atherosclerosis may be stronger in men than in premenopausal women.

4.2 High blood pressure

This section examines the association between excess body weight and high BP. Although obesity and high BP are both disorders/conditions in their own right, studies have shown that there is a strong and close relationship between the two (Frohlich 1991). BP increases as BMI increases and people who are obese have been found to have a much higher prevalence of high BP (Doll et al. 2002; WHO 2000; Kemper et al. 1999; McCarron & Reusser 1996; Stamler 1991; Garn et al. 1988). The studies included in this review are listed in Appendix B Table A2.

4.2.1 Excess body weight and high BP

In the three US National Health and Nutrition Examination Surveys (NHANES), high BP (defined as 140/90 mmHg or greater or reported use of antihypertensive medication) was independently associated with higher BMI. In all three surveys, higher BMI was responsible for around 30% of the prevalence of high BP (Hajjar & Kotchen 2003). After adjustments were made for age, sodium and potassium excretion, alcohol intake and smoking status, the NHANES II study of American adults found that high BP among those with excess body weight was three times as common as among those with normal weight (van Itallie 1985).

The latest NHANES phase (Hajjar & Kotchen 2003), conducted in 1999–2000, observed an increase in participants with high BP when compared with the previous two phases of the NHANES III survey conducted between 1988 and 1991, and 1991 and 1994. Within this same period, Flegal et al. (2002) also found an approximate increase of 8% in the prevalence of obesity (BMI ≥ 30). After adjustments were made for age, sex and race/ethnicity, BMI was independently and positively associated with high BP prevalence and was found to contribute to more than half the increase (Hajjar & Kotchen 2003).

The INTERSALT Study (Stamler 1991; Dyer & Elliot 1989) examined the relationship between BMI and BP among more than 10,000 people in 52 centres and 32 countries around the world. They found a clearly established, significant and independent relationship between high BP and increased BMI. BMI was positively associated with SBP in 51 of the 52 centres studied among men, and in 47 of the 52 centres among women. This association was significant in 24 and 27 centres for men and women, respectively. BMI was positively associated with DBP in 51 centres for men and 49 centres for women and was significant in 33 and 31 centres, respectively. The INTERSALT study found that every BMI unit increase was associated with an SBP increase of 0.91 mmHg for men and a 0.72 mmHg increase for women (Stamler 1991; Dyer & Elliot 1989). For DBP, this increase was 0.75 mmHg for men and 0.5 mmHg for women per BMI unit. This significantly positive association between BMI and BP was similar between men and women and was significant across age groups as well (Dyer & Elliot 1989). Overall, a ten kilogram difference in body weight was associated with a 3.0 mmHg difference in SBP and a 2.2 mmHg difference in DBP (Dyer & Elliot 1989).

The WHO MONICA study found that although there were ethnic differences in the association between obesity and high BP, in general the risk of an obese person becoming hypertensive is similar worldwide (relative risk (RR) approximately 2–3) when compared with a lean person (WHO 2000).

Sex

Some studies have found the positive association between body weight and BP to be similar among men and women (Folsom et al. 1991; Dyer & Elliot 1989). However, Doll et al. (2002)

established a substantially greater association between overweight and obesity and BP levels in females. Among men, they found a 1 mmHg increase in SBP for every 1.7 kg/m^2 increase in BMI, 4.5 cm increase in waist circumference or 3.4% increase in WHR. Among women, this 1 mmHg SBP increase occurred for every 1.25 kg/m^2 increase in BMI, 2.5 cm increase in waist circumference, or 1.8% increase in WHR—substantially smaller increments than in men, indicating a stronger relationship between BP and measures of overweight and obesity in women.

A similar association was also found in the Tecumseh Community Health Survey where excess body fatness was a stronger predictor for high BP in females than males (Garn et al. 1988). Further, higher rates of high BP and lower rates of BP control were found among women in the 1999–2000 NHANES (Hajjar & Kotchen 2003).

Age

The prevalence of high BP and overweight and/or obesity increases progressively with age (Filipovsky et al. 1993). However, the NHANES II study established that the RR of high BP among overweight people aged 20–44 years was 5.6 times greater than their non-overweight counterparts. Among people aged 45–74, the RR was double that of lean persons of the same age (van Itallie 1985).

Similarly, the INTERSALT study found that the positive association between BMI and BP was significant across age groups; however, there was a stronger association for those aged 20–39 compared with those aged 40–59 (Dyer & Elliot 1989). Therefore, it follows that increased body weight during early adult life may be more hazardous than during middle age in relation to BP.

Garn et al. (1988) in their study of 5,507 people aged 15–75 years, however, found the effect of total adiposity levels on BP was as marked in older adults as in younger adults.

4.2.2 Duration of excess body weight

The People's Gas Company Study (Chicago) followed 746 men over a period of 20 years and found that the relative weight (RW) of young adult men, measured as the ratio of actual weight to desirable weight for height x 100, was related to risk of high BP later in life. Young men with a RW < 105 had lower rates of high BP over the next 20 years compared with young men with a RW = 105–114 (Stamler 1991).

4.2.3 Location of body fat

Studies have reported a link between central adiposity and increased risk of developing high BP (Selby et al. 1989; Blair et al. 1984; Despres et al. 1990). In some studies, central skinfold thickness, WHR and waist circumference have all been revealed to be strong predictors of high BP compared with BMI (Doll et al. 2002; Kemper et al. 1999; Folsom et al. 1991; Blair et al. 1984). However, in other studies, BMI was more strongly associated with BP than central skinfold tests were (Filipovsky et al. 1993; Stamler 1991) or there was no difference across the measures of fat patterning (Garn et al. 1988).

4.2.4 Excess body weight in childhood and adolescence

The Bogalusa Heart study (Freedman et al. 1999) found that overweight children were 2.4 times more likely to have high DBP and 4.5 times more likely to have high SBP, when compared with non-overweight children. Overweight was classified as a BMI above the smoothed 95th percentile of combined data from the Health Examination Survey II and III, and NHANES I, II and III.

Table 3: Estimated RR for overweight children (BMI > 95th percentile) aged 5–17 years and high BP

	White boys	White girls	Black boys	Black girls	Overall
High levels of SBP	7.1	4.5	3.0	3.0	4.5
High levels of DBP	3.1	3.4	1.1	1.6	2.4

Source: Freedman et al. 1999.

Similarly, a study by Rosner et al. (2000) examined eight US epidemiological studies to assess differences in BP between black and white children. They documented consistent associations between body size and high BP among all children, regardless of race, age and gender. Children in the upper decile of BMI had a 2.5–3.7 odds ratio (OR) when compared with children in the lower decile.

A study of 2,460 children aged 12–16 years (Sorof et al. 2002) that examined the association between all risk factors and high BP among children found BMI to be most dominant. Obese children were three times more at risk of high BP than non-obese children were. The gradient of risk increased with increment of BMI. Further, this study found a positive association between high SBP (referred to as 'isolated' high BP) and obesity (Sorof et al. 2002). Thirty-three per cent of obese children had high BP compared with 11% of non-obese children. Of those who were hypertensive, more than 88% had isolated SBP (Sorof et al. 2002).

4.2.5 Strength of evidence

The literature reviewed in this study suggests that:

- There is good evidence of an association between excess body weight and high BP among people of all ages, including children, with those classed as obese at greatest risk.
- There is moderate evidence that the association between excess body weight and high BP is less strong with increasing age and less strong for men compared with women.
- There is little evidence of an association between central adiposity and high BP.

4.3 High blood cholesterol

This section examines the association between excess body weight and high blood cholesterol, including its components. The examination is based on a range of prospective and cross-sectional studies, and various populations. The studies included in this review are listed in Appendix B Table A3.

4.3.1 Total cholesterol (TC) levels

Several studies have shown consistent positive independent associations between excess body weight and TC levels (Owen et al. 2003; WHO 2000; Ferrara et al. 1997; Stamler et al. 1997; Ernst & Obarzanek 1994; Denke et al. 1993; Dattilo & Kris-Etherton 1992; Garn et al. 1988).

The Tecumseh Community Health Survey (Garn et al. 1988) examined 5,507 white people aged 15–75 to study the association of skinfold levels and lipids and BP. They discovered that increased body weight, measured using the sum of four skinfolds (triceps, subscapular, iliac and abdominal), was associated with an increase in lipid levels across all age groups. This study found an average 13 mg/dL increase (around 0.34 mmol/L) in TC from the lowest to the highest level of body fatness. This increase was found to be slightly more marked in males.

Significant correlations between body weight and TC levels were also observed in other studies reviewed by Denke et al. (1993): the Brooks Air Force Base Study and the Chicago Heart Association Detection Project in Industry.

Age

Denke et al. (1993) reported on NHANES II data and assessed the association between BMI and TC levels in white men. They found that among young men (20–44 years), differences in TC levels were most marked between the highest (BMI ≥ 30) and lowest (BMI < 21) BMI groups. Similar results occurred in older men (45–59 and 60–74 years), but to a lesser degree. The results were adjusted for dietary intakes and smoking.

The Chicago Heart Association Detection Project in Industry (reviewed by Denke et al. 1993) found a stronger correlation between body weight and TC levels in those aged less than 35 years.

4.3.2 Low-density lipoprotein (LDL) cholesterol

Significant correlations between body weight and LDL cholesterol levels were found in the Framingham Offspring Study (reviewed by Denke et al. 1993), the Cardiac Study (Denke et al. 1993) and the Rancho Bernardo Study (Ferrara et al. 1997). People with excess body weight are more likely to have increased levels of LDL cholesterol (Ferrara et al. 1997; Stamler et al. 1997; Kuller et al. 1995; Ernst & Obarzanek 1994; Denke et al. 1993; Dattilo & Kris-Etherton 1992).

Age

The NHANES II (Denke et al. 1993) study found that LDL cholesterol levels were not as readily influenced by body weight as was TC. The association between LDL cholesterol levels and BMI was even less significant in the older age groups. Young men (aged 20–44)

with BMI ≥ 30 showed higher LDL levels (3.78 mmol/L) compared with young men in the leanest category (BMI ≤ 21) (2.87 mmol/L). In the same BMI categories in older age groups, there was very little difference in LDL levels.

The Chicago Heart Association Detection Project in Industry (reviewed by Denke et al. 1993) also found stronger correlations between body weight and LDL cholesterol levels among younger people (aged < 35 years). The Framingham Offspring Study (reviewed by Denke et al. 1993) also noted a stronger correlation between LDL cholesterol and BMI for young men aged 20–29 years compared with older men aged 40–49.

4.3.3 High-density lipoprotein (HDL) cholesterol

Excess body weight is associated with lower levels of HDL cholesterol (WHO 2000; Ferrara et al. 1997; Ernst & Obarzanek 1994; Denke et al. 1993; Dattilo & Kris-Etherton 1992).

The Lipid Research Clinics Program Prevalence Study also showed a significant negative correlation between HDL cholesterol and body weight in those aged 12–79 years (reviewed by Denke et al. 1993). The NHANES II study found that HDL cholesterol levels were negatively correlated with BMI and this was significant across age groups (Denke et al. 1993).

4.3.4 Location of body fat

There is some evidence that central adiposity is related to the risk of high blood cholesterol independent of total adiposity (WHO 2000; Despres 1994). Kuller (1999) found that central adiposity compared with total adiposity was more positively associated with increased triglyceride level, lower HDL cholesterol and less positively associated with LDL cholesterol. Total adiposity was more strongly associated with LDL cholesterol.

4.3.5 Excess body weight in childhood and adolescence

Childhood obesity is a major health problem as it commonly leads to obesity in adulthood and is associated with several risk factors for CVD, including high blood cholesterol. Further, high cholesterol levels in childhood often lead to high levels in adulthood (Ernst & Obarzanek 1994).

Data examining the association between lipid levels and body weight in children are rare. However, Ernst & Obarzanek (1994) examined two child and adolescent studies, the Bogalusa Heart Study and the Muscatine study, and found that both demonstrated a positive correlation between BMI or skinfolds and TC and LDL cholesterol levels, and a negative correlation for HDL cholesterol. These studies established that the greater the BMI and skinfold thickness the greater the level of blood cholesterol and other lipids. Further analysis of the Bogalusa Heart Study by Freedman et al. (1999) established that overweight (defined as BMI at levels ≥ 85th percentile) children aged 5–17 years were 2.4 times as likely to have high TC levels as non-overweight school children. Similar ORs were also seen for high LDL cholesterol (3.0) and low HDL cholesterol (3.4).

The Ten Towns Heart Health Study (Owen et al. 2003), involving a cross-sectional analysis of adolescents aged 13–16 years, found that TC levels increased as BMI increased. Social class and ethnic group were not significantly related to TC levels.

4.3.6 Strength of evidence

The literature reviewed in this study suggests that:

- There is good evidence of an association between excess body weight and higher TC and LDL cholesterol levels and lower HDL cholesterol levels.
- There is moderate evidence that the association between excess body weight and higher TC and LDL cholesterol levels is stronger in younger adults compared with older adults.
- There is moderate evidence of an association between excess body weight and higher TC and LDL cholesterol levels and lower HDL cholesterol levels among children and adolescents.

4.4 Type 2 diabetes

This section examines the relationship between excess body weight and the development of Type 2 diabetes, a well-established biomedical risk factor for CVD (Barrett-Connor & Pyorala 2001).

Large prospective and cross-sectional studies using measured overweight and obesity and measured glucose or insulin levels are rare. For this reason, this section draws on studies which may not meet this description. Studies included in this review are presented in Appendix B Table A4.

Assessment of the relationship of obesity and Type 2 diabetes identified certain elements which strengthen the risk of developing Type 2 diabetes amongst the obese, after controlling for age, smoking and family history of Type 2 diabetes (WHO 2000). These characteristics include central adiposity, duration of obesity, and obesity during childhood and adolescence.

4.4.1 Location of body fat

Excess body weight, especially when located in the abdominal region, has a strong association with blood glucose levels, insulin resistance and the development of diabetes. This has been a consistent finding across a range of prospective studies (Despres et al. 2001; Boyko et al. 2000; Njolstad et al. 1998; Chan et al. 1994; Haffner et al. 1991; Charles et al. 1991; Colditz et al. 1990), cross-sectional studies (Janssen et al. 2002; Schmidt et al. 1992; Dowse et al. 1991; Skarfors et al. 1991) and recent reviews (WHO 2003; Hodge et al. 1996; WHO 2000; Kuller 1999; Despres et al. 1990).

Most studies support that central adiposity is the dominant risk factor for the development of Type 2 diabetes, although there are some exceptions. Perry et al. (1995) and Skarfors et al. (1991) found BMI was the dominant risk factor over other measures of central adiposity for risk of developing Type 2 diabetes.

A review by Hodge et al. (2001) concluded that both overall adiposity and central fat distribution were important independent risk factors for Type 2 diabetes.

4.4.2 Duration of excess body weight

Wannamethee and Shaper (1999), in the British Regional Heart Study, showed that for men aged 40–59 years the duration of being overweight and obese (BMI ≥ 25) was a strong and continuous predictor of Type 2 diabetes. Men who had been overweight (defined as

BMI = 25.0–27.9) or obese (defined as BMI ≥ 28) for 5 years or more consistently had a greater risk of diabetes than men who had been in a comparable BMI category for less than 5 years. The RR compared with normal-weight men was threefold for overweight men who had been so for less than 5 years, compared with a RR being fourfold in men who had been obese for 5 years or more. Men who were markedly obese (BMI > 30) for 5 years or more had an eightfold risk of developing Type 2 diabetes compared with men who were not overweight (BMI < 25).

4.4.3 Childhood and adolescence

There is evidence that cases of Type 2 diabetes are emerging in the child and adolescent population. Type 2 diabetes is typically characterised by its onset during middle age; however, small clinical studies are revealing cases of Type 2 diabetes in obese children.

A review of these small clinical studies by Goran and colleagues (2003) discussed the emergence of Type 2 diabetes and CVD in children. They proposed several risk factors for the development of Type 2 diabetes and CVD in youth: increased body fat and abdominal fat, insulin resistance, ethnicity and onset of puberty. Furthermore, they suggested that these factors work in an additive fashion.

In their review, the authors cite evidence from the Bogalusa Heart study, where there was a weak but significant positive association between children with central body fat and fasting insulin. The study also found that there was increased insulin resistance in African-Americans compared with Caucasian children, based on measures of fasting insulin. Their review cites various cross-sectional studies that show that pubertal development was associated with an approximate 25–30% reduction in insulin sensitivity. The authors conclude that further research is required to ascertain the mechanisms for the progression of Type 2 diabetes in both adults and children.

4.4.4 Australian studies

The AusDiab study conducted during 1999–2000 found that obesity (BMI ≥ 30) levels among adults (25 years and over) were more than double the rate observed in 1980 in the National Heart Foundation of Australia's Risk Factor Prevalence Survey (AIHW 2002). Further, obesity was strongly linked to impaired glucose tolerance (31.5% were obese) and diabetes (44.4% were obese) (Dunstan et al. 2001).

The investigators concluded that the increased prevalence of obesity in Australia has been a significant contributing factor in the increasing prevalence of diabetes, specifically Type 2 diabetes.

Aboriginal and Torres Strait Islander peoples

Several small (less than 1,000 participants) regional Australian studies have examined the association of excess body weight and the prevalence of Type 2 diabetes in Aboriginal and Torres Strait Islander peoples.

Daniel et al. (1999) concluded after an 8-year follow-up study that BMI-specific diabetes incidence rates in Australian Aboriginal people were among the highest in the world. In two Central Australian Aboriginal communities, after adjusting for age, sex and community, the population diabetes incidence rate was 20.3 cases/1,000 person-years. Across the BMI

categories (BMI: < 25; 25–28.9; 29–32.9 and ≥ 33), rates ranged from 10.7 to 47.2 cases/1,000 person-years.

Sequential cross-sectional surveys (1987, 1991 and 1995) were conducted in a Central Australian Aboriginal community. McDermott and colleagues (2000) reported that among younger women (15–24 years) in this community there was a trebling in obesity prevalence (BMI ≥ 30) over the period and a four- to fivefold increase in diabetes prevalence.

A cross-sectional study by Guest et al. (1993) compared insulin and obesity levels of 300 southeastern Australian Aboriginals and 553 other Australians from rural and urban Australia. They found that mean insulin levels were much higher in Australian Aboriginals (15.5 mU/L) than other Australians (9.5 mU/L). Obesity (BMI > 30) was more prevalent in Australian Aboriginals—38% of Australian Aboriginal females (25–64 years) were obese compared with 18% of other Australian females. Abdominal obesity (WHR > 0.9 in men and > 0.8 in women) was higher among Australian Aboriginal females (mean 0.87 in persons aged 25–64 years) than other Australian females (mean 0.81, $p < 0.001$). There was a strong and positive association between insulin levels and BMI. The association between WHR and insulin levels was not significant.

Leonard and others (2002) investigated obesity, diabetes and associated CVD risk factors among Torres Strait Islander people. They found a high prevalence of overweight (BMI ≥ 25 and < 30) (81%), obesity (BMI ≥ 30) (51%), abdominal obesity (WHR > 0.9 for men and > 0.8 for women) (70%), diabetes (26%), high blood cholesterol (33%), albuminuria (28%), high BP (32%) and tobacco smoking (45%). In fact, only 8.5% of men and 6.5% of women were considered free of risk factors for heart disease.

O'Dea and others (1990) found a high frequency of diabetes in a small Aboriginal community in Northern Australia despite the community being relatively lean. BMI increased with age for both men and women, but only 25% of the sample was overweight or obese. Type 2 diabetes increased with age—Type 2 diabetes was eight times as common in those 35 years and older as in those aged 15–34 years.

4.4.5 Strength of evidence

The literature reviewed in this study suggests that:

- There is good evidence of an association between excess body weight, particularly in the abdominal region, and Type 2 diabetes.
- There is moderate evidence of an association between the duration of excess body weight and Type 2 diabetes in men.
- There is moderate evidence of an association between excess body weight in childhood and adolescence and Type 2 diabetes.
- There is good evidence of an association between overweight and obesity among Aboriginal and Torres Strait Islander people and Type 2 diabetes.

5 The association between overweight and obesity and CVD

This section examines the association of overweight and obesity and CVD. It discusses the association with overall CVD risk and then examines major CVD components including CHD, stroke and heart failure. The metabolic effects arising from obesity, which may explain its contribution to cardiovascular risk, are outside the scope of this discussion.

The discussion is based on international prospective analyses involving large cohorts, measured criteria of overweight and obesity, and documented outcomes and results assessing the independent and combined contribution of overweight and obesity and CVD risk. Results from Australian studies may not fit these criteria but have been included to represent local findings. Case control studies have also been included where available.

Studies included in this review are presented in Appendix B Table A5.

5.1 Overweight and obesity and risk of CVD

The signs alluding to an association between overweight and obesity and CVD risk have largely originated from analyses of the Framingham Heart Study. One of the earliest of these was by Hubert et al. (1983). After 26 years of follow-up they concluded that obesity, measured as the ratio of actual weight to desirable weight (MRW), was a significant independent predictor of CVD, including CHD, coronary death and congestive heart failure in both men and women; and stroke in women after adjustment for risk factors.

After 44 years of follow-up of the Framingham Heart Study, Wilson et al. (2002) showed that CVD risk (including angina, myocardial infarction, CHD or stroke) was higher among overweight men (RR 1.24; 95% CI: 1.07–1.44), and obese men (RR 1.38; 95% CI: 1.12–1.69) and obese women (RR 1.38; 95% CI: 1.14–1.68) after adjustment for age, smoking, high blood pressure, high cholesterol and diabetes. The association was not significant among overweight women. In this case, overweight was defined as BMI ≥ 25 but < 30, and obesity as BMI ≥ 30.

After 16 years of follow-up, analysis of prospective data from the Nurses' Health Study (Manson et al. 1995) indicated that the risk of death from CVD was significantly greater among women with a BMI ≥ 27 compared with the risk among women with a BMI < 19. Among women with a BMI ≥ 32 the RR of death from CVD was 4.1 (95% CI: 2.1–7.1) after accounting for cigarette smoking and disease-related weight loss. Deaths due to CHD were even more strongly associated with BMI among women in the study than deaths due to CVD, as the death rate began to increase at a BMI above or equal to 22 (RR 1.4); among those with a BMI ≥ 29, the RR of death was 4.6. (Although weight was self-reported in this study, it was highly correlated with measured weight in a small subsample of the cohort.)

5.1.1 Age

Hubert et al. (1983) concluded in their analysis of the Framingham Heart Study that an increase in MRW after the age of 25 years independently predicted the risk of CVD, particularly in men. Kannel, D'Agostino & Cobb (1996) refined this risk based on their

summary of the effect of weight on CVD using the Framingham Heart Study and other studies, by concluding that the amount of weight gained after the age of 25 (or on completion of musculoskeletal growth) carries a proportionately increased risk of CVD.

However, this linear association may not continue into old age. Analysis of results from the American Cancer Society's Cancer Prevention Study I (Stevens et al. 1998b) involving 324,135 participants showed that excess body weight increased the risk of death from CVD in healthy white adults aged 30–74 years followed up over a period of 12 years. Above 74 years, however, the RR of death was not significant for either men or women. Hubert et al. (1983) noted in their analysis of the Framingham Heart Study that whilst an increase in MRW after the age of 25 years increased the risk of CVD, the association was stronger among men and women aged less than 50 years.

Similar results were obtained by Baik et al. (2000) in the Health Professionals Follow-up Study. CVD mortality among obese (BMI ≥ 30) men aged less than 65 years was significantly greater (RR 3.92) after accounting for other risk factors including smoking compared with those with a BMI < 23 (RR ≤ 1). Among men 65 years or older, there was no significant relationship between BMI and risk of CVD mortality.

Similar results have been observed in women. In the Adventist Mortality Study, mortality due to CVD was strongly dependent on age among obese (defined in the study as BMI > 27.4) women. Higher BMI at a younger age (30–54 years at baseline) was associated with an increased risk of CVD mortality after 15–26 years of follow-up, whereas this did not apply in older ages (55–74 years at baseline) (Singh & Linstead 1998).

5.1.2 Duration of excess body weight

The reduced risk at older ages may, however, be mediated by the duration of excess body weight. In 1998, Harris et al. analysed data on non-smoking older persons in the Framingham Heart Study and concluded that risk of death was twofold among those with a BMI at or above the 70th percentile at both 55 and 65 years of age. Those who were heavy only at age 65 years had only a small increase in risk and those who were thin over time had better survival overall. CVD mortality (including deaths due to angina, CHD, stroke and congestive heart failure) showed a similar pattern to all-cause mortality.

Analysis of longitudinal data involving men aged 40–55 years at baseline by Spataro et al. (1996) found that there was no discernible association between adiposity and CHD mortality for the first 14 years of follow-up but after 15–22 years of follow-up adiposity (measured as per cent body fat derived from skinfolds, BMI, subscapular skinfold, and their sum) was significantly related to CHD mortality. Similarly, Hubert et al. (1983), in their analysis of the association between obesity (measured using the MRW) and CVD using data from the Framingham Heart Study, found that the risk was greatest among those who remained the heaviest over the 26 years of follow-up.

5.1.3 Protective effects of 'healthy weight'

From analysis of data from the Nurses' Health Study and the Health Professionals Follow-Up Study, Field et al. (2001) concluded that adults should try to maintain a BMI of between 18.5 and 21.9 to minimise their risk of disease.

Kannel, D'Agostino and Cobb (1996), in their comprehensive analysis of the literature, concluded that the optimal weight for avoidance of CVD and prolonging life corresponds to

a BMI of 22.6 for men and 21.1 for women or a subscapular skinfold thickness < 12 mm in men and < 15 mm in women.

5.1.4 Overweight or obese

In a British study involving 7,735 men aged 40–59 years (Shaper et al. 1997) there was a significant increase in the risk of CVD mortality among men with a BMI of 28.0–29.9 (RR 1.57; 95% CI: 1.09–2.25) and men with a BMI ≥ 30 (RR 2.09; 95% CI: 1.45–3.03) compared with those with a BMI of 20.0–21.9. There were no significant increases in risk among those with a BMI of 22.0–28.0. The analysis adjusted for age, smoking, social class, alcohol consumption and physical activity.

In the Health Professionals Follow-up Study (Baik et al. 2000) CVD mortality among obese men aged less than 65 years was significantly greater (RR 3.92; 95% CI: 1.64–9.36) than among those with a BMI < 23 after accounting for smoking status, family history of myocardial infarction, profession, marital status, alcohol intake and dietary components. Those with a BMI = 23.0–29.9 did not differ significantly from those with a BMI < 23.

5.1.5 Location of body fat

In 1991, Kannel et al. investigated regional obesity compared with total adiposity as a risk of CVD using the Framingham Study. They found that the risk of CVD incidence and mortality increased with the degree of regional, central or abdominal obesity. These were measured as waist-to-height ratio, subscapular skinfold alone, its ratio with triceps skinfold and its ratio with BMI. Abdominal obesity was an independent risk factor for stroke, heart failure and CVD mortality in men. In women, the subscapular-to-triceps skinfold ratio independently contributed to the risk of CHD and CVD mortality.

Location of adiposity may be mediated by age as a risk factor for CVD. Among men aged 65 and over, waist circumference, but not BMI, strongly predicted risk of death from CVD in the Health Professionals Follow-up Study (Baik et al. 2000).

Abdominal adiposity may be a better predictor of stroke risk in men and women than BMI (see Section 5.2.3; Suk et al. 2003; Shinton et al. 1995; Walker et al. 1996) and CHD risk in older men (see Section 5.2.1; Rimm at al. 1995) and younger women (aged less than 65) (see Section 5.2.1; Rexrode et al. 1998).

5.1.6 Other 'non-white' population subgroups

The discussion to date has focused on 'white' populations, where the greater the adiposity the greater the risk of CVD. Among 'non-white' populations the association may not be as simple. Adams-Campbell et al. (1995) noted a greater risk of coronary artery disease among moderately overweight African-Americans (with an approximate BMI of between 27 and 32) but found that there was no apparent increase in the degree of coronary artery disease in the more severe overweight range.

In a large population-based study in China, the results were similar to those in white populations (Zhou et al. 2002). The study involved approximately 100,000 people aged 35–59 years followed up for an average of nine years. The authors concluded that BMI was positively and independently associated with an increased risk in the incidence of both stroke and CHD in the population studied. After adjusting for other CVD risk factors, each

two-unit increase in BMI was associated with a 23% increase in the RR for CHD (95% CI: 1.08–1.41), a 9% increase in the RR for total stroke (95% CI: 1.04–1.13) and a 13% increase in the RR for ischaemic stroke (95% CI: 1.08–1.17).

5.2 Overweight and obesity and risk of specific CVDs

5.2.1 Coronary heart disease

Much of the literature linking overweight and obesity to specific CVDs focuses on CHD.

In 1998 the American Heart Association added obesity to its list of major risk factors for CHD (Eckel & Krauss 1998). Rao et al. (2001) examined the evidence supporting this addition. They concluded that the available evidence indicates that both a high BMI (defined in one study as a BMI > 29 among women) and a high WHR are independent risk factors for CHD and mortality irrespective of the presence of other coronary risk factors. There are numerous studies supporting this conclusion (Harris et al. 1993; Manson et al. 1995; Rimm et al. 1995; Willett et al. 1995; Jousilahti et al. 1996; Rexrode et al. 1998; Field et al. 2001; Rashid et al. 2003).

Age

Unlike the mediating effect of older age on obesity and overall CVD risk, this may not apply to CHD risk and may be inter-related with change in weight throughout adult life, which is currently outside the scope of this review.

Analysis of 'white' people in the Epidemiologic Follow Up Study of the NHANES I survey by Harris et al. (1997) showed that BMI ≥ 27 in late middle age (which may reflect the maximum lifetime weight for most people) was associated with a 70% increased risk of CHD in old age (aged 70–86) among both men and women (95% CI: 1.3–2.1) after adjusting for SBP, total cholesterol at baseline and self-reported diabetes, while BMI ≥ 27 in old age was not (RR 1.1; 95% CI: 0.8–1.5). Heavier weight in old age was, however, associated with an increased risk of CHD once those with substantial weight loss between middle and old age were excluded from the analysis.

Among women in the Nurses' Health Study, Manson et al. (1990) found that the current level of overweight or obesity (BMI ≥ 25) among those aged 30 to 55 was a more important correlate of present risk of CHD (RR 2.0; 95% CI: 1.4–2.8 adjusted for age and smoking) than overweight or obesity (BMI ≥ 25) at 18 years of age (RR 1.5; 95% CI: 1.0–2.3). Significant weight gain during adulthood (of 20–34.9 kg), however, approximately doubled the coronary risk after controlling for the initial relative weight level at age 18 (RR 2.5; 95% CI: 1.7–3.7).

In contrast to weight gain throughout life, 'morbid obesity' (or Class III obesity defined as BMI ≥ 40 based on the WHO classification, see Table 1) early in adult life is emerging as a significant risk factor for CHD mortality. In a recent review of data from the Framingham Heart Study, the Nurses' Health Study, the Buffalo Health Study and the Cancer Prevention Study II involving a total of nearly 400,000 people, Rashid et al. (2003) concluded that 'morbid obesity' significantly increased risk of death from CHD, especially in young men.

Age and sex

In a 15-year follow-up study involving 16,113 men and women aged 30–59 years, obesity was found to be an independent risk factor for CHD mortality among men, after adjusting for age, smoking, serum cholesterol and SBP, and a contributing factor (as BMI was found to interact with SBP) to the risk of CHD death among women (Jousilahti et al. 1996). Starting at a BMI of approximately 22, a unit increase in body weight was related to a 4–5% increase in CHD mortality. Among young men (aged 30–49 years) the association between BMI and CHD mortality was weaker than among older men (aged 50–59 years) although the reverse was true in women.

Age and location of body fat

A large study by Rimm at al. (1995) involving 29,122 men aged 40–75 years found that BMI and WHR were associated with an increased risk of CHD. Among men younger than 65, after adjusting for other coronary risk factors, the RR was 1.7 for men with BMI = 25.0–28.9, 2.6 for BMI = 29.0–32.9 and 3.4 where BMI ≥ 33 compared with a lean BMI < 23. Among men 65 years or above the association between BMI and risk of CHD was much weaker, although WHR was a stronger predictor of risk (RR 2.8 between extreme quintiles). The authors concluded that among younger men, obesity is a strong risk factor for CHD, independent of fat distribution, whereas for older men, measures of fat distribution may be a better predictor of coronary disease.

Extent of adiposity

Analysis of data from 115,886 women in the Nurses' Health Study by Manson et al. (1990) showed that even mild-to-moderate overweight (BMI = 25.0–28.9) increased the risk of non-fatal CHD in middle-aged women after adjustment for age and smoking (RR 1.8; 95% CI: 1.2–2.5). Among those with a BMI ≥ 29 the risk more than trebled (RR 3.3; 95% CI: 2.3–4.5). The effect was substantially reduced after adjusting for other CVD risk factors but remained significant among those with a BMI ≥ 29 (RR 1.9; 95% CI: 1.3–2.6). Further analysis by Manson et al. (1995) showed that CHD mortality increased among women in the Nurses' Health Study with a BMI ≥ 22. Above a BMI = 29 the RR was 4.6.

Analysis of the Nurses' Health Study by Willett et al. (1995) showed similar results. They concluded that higher levels of body weight within the 'normal' range, as well as modest weight gains (more than 5 kg) after 18 years of age, appear to increase risks of CHD in middle-aged women. After controlling for age, smoking, menopausal status, postmenopausal hormone use and parental history of CHD, significant increases in risk were observed among those with a BMI ≥ 23 compared with those with a BMI < 21. The RRs for CHD were 1.5 (95% CI: 1.2–1.8) for a BMI = 23.0–24.9, 2.1 (95% CI: 1.7–2.5) for a BMI = 25.0–28.9 and 3.6 (95% CI: 3.0–4.3) for a BMI ≥ 29.

Harris et al. (1993) assessed the risk of CHD incidence among overweight white women aged 65–74 years in the Epidemiologic Follow Up Study of the NHANES I survey. They found that after an average of 14 years of follow-up women with a BMI ≥ 29 showed an increased risk of CHD after adjusting for other CHD risk factors.

In a 10 year follow-up study of 77,690 middle-aged women in the Nurses Health Study and 46,060 men in the Health Professionals Follow-Up Study, Field et al. (2001) found that the incidence of heart disease increased with the severity of overweight. Compared with men and women with a BMI < 25, men with a BMI ≥ 30 were twice as likely to develop CHD (95% CI: 1.7–2.3) and women 1.5 times as likely (95% CI: 1.3–1.7). However, if the reference BMI is

lowered to between 18.5 and 21.9 the risk among obese men and women increases slightly and is apparent even in the weight range BMI = 22.0–24.9, where the RR of heart disease is 1.2 for women (95% CI: 1.1–1.4) and 1.1 for men (95% CI: 1.0–1.4). The data were adjusted for age, smoking status and race.

In a 15-year follow-up study involving 16,113 men and women aged 30–59 years, Jousilahti and others (1996) found that for each unit increase in BMI above approximately 22 there was an increased risk of CHD mortality of 4–5%.

Location of body fat

In an assessment of abdominal adiposity on CHD risk among women aged 40–65 years in the Nurses' Health Study, Rexrode et al. (1998) concluded that WHR and waist circumference are independently associated with CHD risk after controlling for BMI and other cardiac risk factors. Women in the highest quintile of WHR or waist circumference were about 2.5 times more likely to develop CHD than women in the lowest quintile and the association was more pronounced in women younger than 60.

Other measures of adiposity, apart from BMI and waist circumference, have also been shown to be equally predictive of CHD risk. In a case control study involving middle-aged adults, Kahn et al. (1996) found that the ratio of waist girth to thigh girth and abdominal diameter measured from front to back were more strongly related to CHD than was total adiposity after controlling for smoking, history of high blood pressure, high cholesterol and diabetes.

Childhood and adolescence

The contribution of childhood obesity to CHD risk was examined by Freedman et al. (2001). They found that although participants who were overweight early in life were more obese as adults, the age of onset of obesity did not show consistent (or statistically significant) associations with adult risk factor levels. In their review of the literature they noted the conflicting results of other studies. In one study, obese adolescent boys (but not girls) were at increased risk of CHD mortality, and that this association was independent of adult BMI, whereas other studies have found that the effects of childhood obesity were mediated by adult weight status and some findings suggest that the risk may be highest among normal-weight children who become obese adults. The authors concluded that additional data are needed to assess the independent relationship of childhood weight status and CHD morbidity.

5.2.2 Heart failure

Kenchaiah et al. (2002) examined obesity as a risk factor for heart failure. They concluded that for each unit increment of BMI there was an increased risk of heart failure of 5% for men and 7% for women after controlling for several risk factors including age, smoking status and previous heart attack. Obese women were 2.1 times (95% CI: 1.51–2.97) as likely to develop heart failure compared with those of normal BMI; and obese men 1.9 times (95% CI: 1.30– 2.79) as likely. There was a stepwise increase in risk across increasing categories of BMI with obesity defined as BMI ≥ 30.

He et al. (2001), in their assessment of the NHANES 1 Epidemiologic Follow-Up Study, found that after an average of 19 years of follow-up, the incidence of congestive heart failure was positively and significantly associated with overweight (BMI ≥ 27.8 for men and

BMI ≥ 27.3 for women) (RR 1.30; 95% CI: 1.12–1.52) after adjusting for several risk factors including current cigarette smoking, hypertension, diabetes and low physical activity.

5.2.3 Stroke

Although obesity is an established risk factor for CHD, and has more recently been identified to increase the risk of heart failure, its role as a risk factor for stroke remains controversial, based on differences between sexes, measures of adiposity used and type of stroke.

Men

Analysis of 21,414 US male physicians participating in the Physicians' Health Study (Kurth et al. 2002) found that, compared with participants with BMI < 23, those with BMI ≥ 30 had a RR of 2.0 (95% CI: 1.5–2.7) for ischaemic stroke, and 2.3 (95% CI: 1.0–5.0) for haemorrhagic stroke. The association was mediated by high BP, diabetes and high cholesterol for ischaemic stroke but not for haemorrhagic stroke. They estimated that for each unit increase in BMI there was a significant 6% increase in the RR of both ischaemic and haemorrhagic stroke. Field et al. (2001) also found that obese (BMI ≥ 30) men were twice as likely to suffer a stroke compared with men with a BMI < 25.

By contrast BMI was not a risk factor for stroke in a Swedish study involving a cohort of 7,495 men aged 47–55 years who were followed up for an average of 12 years (Harmsen et al. 1990), nor among 28,643 American male health professionals aged 40–75 followed prospectively for five years (Walker et al. 1996). However, there was a significant age-adjusted risk between extreme quintiles of the WHR (< 0.89 compared with ≥ 0.98) and stroke (RR 2.3; 95% CI: 1.25–4.37) (Walker et al. 1996). These findings are based on self-reported measurements.

Women

Field et al. (2001), in their analysis of a large cohort of women, did not observe an increased risk of stroke with increasing BMI after adjusting for age, smoking, oral contraceptive use, menopausal status, hormone replacement therapy and time period. Overweight and obesity may, however, increase the risk for ischaemic, but not haemorrhagic, stroke in women. During 16 years of follow-up in the Nurses' Health Study involving women aged 30–55, ischaemic stroke was 75% higher in women with a BMI > 27 and 137% higher in those with a BMI > 32, compared with women who had a BMI < 21 (Rexrode et al. 1997).

Location of body fat

Results from two case control studies showed that abdominal fat was associated with an increased risk of stroke. In the Northern Manhattan Stroke Study, involving 576 stroke cases, those with a WHR greater than or equal to the median had an OR of 3.0 (95% CI: 2.1–4.2) for ischaemic stroke (Suk et al. 2003). The association occurred in men and women and in all race-ethnic groups; it was a stronger predictor of stroke than BMI, and had a greater effect among younger persons. In a British case control study involving men and women aged 35 to 74 years, Shinton, Sagar and Beevers (1995) found that excess body fat, in particular abdominal fat, increased the risk of stroke, although cigarette smoking presented a greater risk than abdominal fat alone.

5.3 Australian studies

5.3.1 Women

Large Australian studies investigating the association between overweight and obesity and CVD are rare. Brown et al. (1998) analysed data on adjusted self-reported height and weight from 13,431 women aged 45–49 years in the Australian Longitudinal Study on Women's Health (now known as the Women's Health Australia project). They found that the risk of heart disease (self-reported) doubled among women with a BMI > 40 (OR 2.0; adjusted for confounding variables) compared with a BMI ≤ 30.

5.3.2 Aboriginal and Torres Strait Islander peoples

Wang & Hoy (2002) assessed the association between BMI and risk of disease-specific mortalities over a period of eight years in 744 Indigenous Australians aged 20–77 years in a remote community. Contrary to results on 'white' populations included in this review, they found no association between BMI and death from CVD (including heart failure, heart attack, cardiac arrest and stroke coded as either a primary or secondary cause). In fact, those in the highest BMI quartile (median 30.1) were the least likely to die from CVD (rate ratio of 0.41, adjusted for age, sex, smoking and drinking status) although this result was not significant. The authors suggested that this result may be due to the association of higher BMIs with relative socioeconomic advantage. The findings might also be explained by higher birth weights. Pre-existing disease in those in the lowest BMI quartile (median 18.1) was also not accounted for. They concluded 'that the ability to gain and maintain a moderate to mildly excessive amount of weight indicates a survival advantage for adults in this society'.

5.4 Relative risks

Table 4 summarises the studies reviewed that provide relative risk data on the relationship between overweight, obesity and CVD.

Table 4: Summary of the relative risks for CVD

Study	Population	Outcome	Relative risk	CI	
Baik et al. 2000	Men < 65 yrs (BMI ≥ 30)	CVD death	3.9	1.6	9.4
Brown et al. 1998	Women aged 45–49 (BMI > 40)	Heart disease incidence	Odds ratio 2.0	0.9	4.5
Field et al. 2001	Men (BMI ≥ 30) (RR = 1; BMI < 25)	CHD incidence	2.0	1.7	2.3
	Women (BMI ≥ 30) (RR = 1; BMI < 25)	CHD incidence	1.5	1.3	1.7
	Men (BMI = 22.0–24.9) (RR = 1; BMI = 18.5–21.9)	CHD incidence	1.1	1.0	1.4
	Women (BMI = 22–24.9) (RR = 1; BMI = 18.5–21.9)	CHD incidence	1.2	1.1	1.4
Harris et al. 1997	BMI ≥ 27 in late middle age	CHD incidence	70% increase in old age	1.3	2.1
Kurth et al. 2002	Men (BMI ≥ 30) (RR = 1; BMI < 23)	Ischaemic stroke incidence	2.0	1.5	2.7
	Men (BMI ≥ 30) (RR = 1; BM < 23)	Haemorrhagic stroke	2.3	1.0	5.0
Manson et al. 1990	Women at 30–55 years (BMI ≥ 25)	CHD incidence	2.0	1.4	2.8
	Women at 18 years (BMI ≥ 25)	CHD incidence in middle age	1.5	1.0	2.3
	Women middle aged (BMI ≥ 29)	CHD incidence	1.9	1.3	2.6
Manson et al. 1995	Obese women (BMI ≥ 32)	CVD death	4.1	2.1	7.1
	Women (BMI ≥ 29)		4.6	P < 0.001	
Rimm et al. 1995	Men < 65 yrs (BMI = 25.0–28.9) (RR = 1; BMI < 23)	CHD incidence	1.7	1.1	2.7
	Men < 65 yrs (BMI = 29.0–32.9) (RR = 1; BMI < 23)	CHD incidence	2.6	1.5	4.4
	Men < 65 yrs (BMI ≥ 33) (RR = 1; BMI < 23)	CHD incidence	3.4	1. 7	7.1
	Men aged ≥ 65 yrs (WHR > 0.98) (RR = 1; WHR = 0.70–0.89)	CHD incidence	2.8	1.2	6.2
Shaper et al. 1997	Men (BMI = 28.0–29.9)	CVD death	1.6	1.1	2.2
	Men (BMI ≥ 30)	CVD death	2.1	1.4	3.0
Suk et al. 2003	WHR ≥ median	Ischaemic stroke incidence	Odds ratio 3.0	2.1	4.2
Walker et al. 1996	Men (WHR ≥ 0.98) (RR =1; WHR < 0.89)	Stroke incidence	2.4	1.2	4.6
Willett et al. 1995	Women (BMI = 23.0–24.9)	CHD incidence	1.5	1.2	1.8
	Women (BMI = 25.0–28.9)	CHD incidence	2.1	1.7	2.5
	Women (BMI ≥ 29)	CHD incidence	3.6	3.0	4.3
Wilson et al. 2002	Overweight men (BMI = 25.0–29.9)	CVD incidence	1.2	1.1	1.4
	Obese men (BMI ≥ 30)		1.4	1.1	1.7
	Obese women (BMI ≥ 30)		1.4	1.1	1.7
Zhou et al. 2002	Every 2 unit increase in BMI	CHD incidence	23% increase in RR	1.1	1.4
	Every 2 unit increase in BMI	Total stroke incidence	9% increase in RR	1.0	1.1
	Every 2 unit increase in BMI	Ischaemic stroke	13% increase in RR	1.1	1.2

5.5 Strength of evidence

The literature reviewed in this study suggests that for:

Cardiovascular disease

- There is good evidence of an association between overweight and obesity and CVD incidence among young to middle-aged men and women but not among older people.
- There is moderate evidence of an association between the duration of obesity and CVD mortality among adults, which may mediate the protective effect of overweight in older age.
- There is moderate evidence of an association between obesity and CVD mortality among adults.
- There is moderate evidence of an association between abdominal obesity and risk of CVD, particularly among older men.
- Overweight and obesity as a risk factor for CVD among Aboriginal and Torres Strait Islander peoples remains uncertain.

Coronary heart disease

- There is good evidence of an association between overweight and obesity and increased risk of CHD in adults.
- There is moderate evidence of an association between abdominal obesity and risk of CHD in older men and younger women.
- There is little evidence of an association between childhood and adolescent obesity and risk of CHD (particularly among girls), unless the children become obese adults.

Heart failure

- There is moderate evidence of an association between overweight and obesity and risk of heart failure.

Stroke

- There is moderate evidence of an association between overweight and obesity and increased risk of ischaemic stroke among adults.
- There is moderate evidence of an association between abdominal adiposity and risk of stroke in men and women.

6 Conclusions

6.1 Summary of evidence

Tables 5 and 6 below summarise the extent of evidence of an association between excess body weight and CVD, as suggested in the literature reviewed.

Table 5: A summary of the evidence between excess body weight and major established biomedical risk factors for CVD

The association between excess body weight and…	Level of evidence
Atherosclerosis	Good
• is stronger in men compared with pre-menopausal women	Moderate
High blood pressure	Good
• is stronger in women compared with men	Moderate
• is stronger in younger men compared with older men	Moderate
• in children and adolescents	Good
• in adults with central adiposity	Weak
Blood cholesterol	
High total cholesterol	Good
• is stronger in younger adults compared with older adults	Moderate
• in children and adolescents	Moderate
High LDL cholesterol	Good
• is stronger in younger adults compared with older adults	Moderate
• in children and adolescents	Moderate
Low HDL cholesterol	Good
• in children and adolescents	Moderate
Type 2 diabetes	
• in adults with central adiposity	Good
• in children and adolescents	Moderate
• in men with long duration of excess body weight	Moderate
The association between overweight and obesity and…	
Type 2 diabetes	
• in Aboriginal and Torres Strait Islander peoples	Good

Table 6: A summary of the evidence between overweight and obesity and risk of CVD

The association between overweight and obesity and…	Level of evidence
Cardiovascular disease	
• incidence in young to middle-aged adults	Good
• incidence in Aboriginal and Torres Strait Islander peoples	Weak
Coronary heart disease	
• incidence in adults	Good
Heart failure	
• incidence in adults	Moderate
Stroke	
• ischaemic stroke incidence	Moderate
The association between obesity and…	
Cardiovascular disease	
• mortality in adults (including duration of obesity)	Moderate
• incidence in older men with abdominal obesity	Moderate
Coronary heart disease	
• incidence in older men and younger women with abdominal obesity	Moderate
• incidence in children and adolescents (particularly among girls)	Weak (unless the children become obese adults)
Stroke	
• incidence in adults with abdominal obesity	Moderate

6.2 Further research

The reviewers found limited or no studies on overweight and obesity and:

Atherosclerosis

- between the sexes
- across age groups (including children and adolescents)
- location of body fat: central versus total adiposity
- among the diverse populations, people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people, or migrant groups

High blood pressure

- between the sexes
- across age groups (including children and adolescents)
- among the diverse populations, people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people, or migrant groups

Blood cholesterol

- duration of overweight and obesity
- among the diverse populations, people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people, or migrant groups

Type 2 diabetes

- across age groups (including children and adolescents)
- duration of overweight and obesity
- among the diverse populations, people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people, or migrant groups
- extent of obesity

Cardiovascular disease

- among the diverse populations, people from low socioeconomic backgrounds, Aboriginal and Torres Strait Islander people, or migrant groups
- increased levels of physical activity and its effect on reducing the negative health consequences of overweight.
- among children and adolescents, including low birthweight.

Appendix A: References

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Appendix B: Lists of included studies

Table A1: Studies included for atherosclerosis

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight / obesity	Outcome	Confounders considered	Relationship
Berenson et al.	1992	Bogalusa Heart Study	150	M F	6–30	Cross-sectional	Ponderal index (weight/height^3)	Fatty streaks, fibrous plaque lesions	No	Ponderal index associated with fatty streaks in the aorta ($r = 0.38$; $p < 0.05$) for white males; fatty streaks in the coronary arteries ($r = 0.37$; $p < 0.05$) for white males. Too few observations to present female correlations.
De Michele et al.	2002	Progetto ATENA study	5,062	F	30–69	Cross-sectional	BMI, WHR	Carotid intimal-medial thickness, intimal-medial areas	Age, BP, lipids, fasting insulin. Less than 5% had used hormone replacement therapy	BMI and WHR were significant predictors of carotid wall thickness independently of other cardiovascular risk factors.
Mahoney et al.	1996	Muscatine Study	384	M F	Mean = 15	15	BMI, triceps skinfold thickness	Coronary artery calcification	Age, sex	Obesity was strongly associated with coronary artery calcification.
McGill et al.	2002	Pathobiological Determinants of Atherosclerosis in Youth (PDAY) study	3,000	M F	15–34		BMI, thick panniculus adiposus	Atherosclerotic lesions in right coronary artery (RCA), left anterior descending coronary artery (LAD)	Age, race	Obesity is related to accelerated coronary atherosclerosis in adolescent and young men. Increased BMI in adolescent and young men was associated with fatty streaks in the RCA and stenosis in LAD. RCA lesions were greater in young men with thick panniculus adiposus. BMI was not associated with coronary atherosclerosis in young women although there was a trend among those with a thick panniculus adiposus.

(continued)

Table A1 (continued): Studies included for atherosclerosis

Authors	Year	Study	N	Sex	Age	Follow -up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Rubba et al.	2001	Progetto ATENA study	310	F	30–69	Cross-section-al	BMI	Carotid intimal-medial thickness	Age	Higher BMI associated with common carotid plaques.
Takami et al.	2001	Japanese	849	M	20–78	Cross-section-al	BMI, waist circumference, WHR, abdominal sub-cutaneous fat, intra-abdominal fat	Carotid intimal-medial thickness (IMT)	Age, smoking	All overweight measures correlated with IMT. When adjusted for BMI, only WHR remained correlated.

Table A2: Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Blair et al.	1984	First National Health and Nutrition Examination Survey (NHANES)	5,506	M F	30–59	Cross-sectional	Tricep & subscapular skinfold thickness	HBP	Age	Link between central adiposity WHR & WC and HBP
Despres et al.	1990	Review of numerous studies								Abdominal obesity related to HBP
Doll et al.	2002	MONICA/Swiss data – Vaud-Fribourg – Ticino & – Seychelles Heart Study	3,116 (tot.) 1,065 1,361 690	M F	35–64	Cross-sectional	BMI, WC, WHR, HBP ≥ 140/90 mmHg	Relationship between adiposity and BP	Age	Linear WC & WHR stronger predictors than BMI
Dyer & Elliot	1989	INTERSALT	10,079	M F	20–59	Cross-sectional	HBP ≥ 140/90 mm/Hg	HBP	Age, sex, sodium & potassium excretion, alcohol intake, smoking status	For men, SBP increase of 0.91 mmHg and DBP increase of 0.75 mmHg per BMI unit. For women, this increase was 0.72 and 0.5 for SBP and DBP.

(continued)

Table A2 (continued): Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Filipovsky et al.	1993	Paris Prospective Study	7,312	M	42–53	15–20	BMI & ITCI (iliac-to-thigh circumference index)	Mortality from CVD & cancer	Age, smoking, serum cholesterol	Linear BMI was more strongly related to BP than ITCI
Freedman et al.	1999	Bogalusa Heart Study	9,167	M F	5–17	Cross-sectional	BMI > 95th P, Rohrer index, triceps & subscapular skinfold thickness.	HBP	Age, race, sex, year of study, insulin & cholesterol levels	Positive association
Frohlich	1991	Review of many studies								Association
Garn et al.	1988	Tecumseh (MI) Community Health Survey	5,507	M F	15–75	Cross-sectional	Skinfolds: triceps, sub-scapular, iliac site, abdominal & sum, HBP = DBP ≥ 90	HBP	None	Positive association
Hajjar & Kotchen	2003	NHANES 1999–2000	4,115	M F	≥ 18	Cross-sectional	BMI, HBP ≥ 140/90 mmHg	Hypertension	Age, sex race/ethnicity	Per 1 unit increase in BMI—RR increase of 0.31

(continued)

Table A2 (continued): Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Ireland & Giles	1996	Melbourne Collaborative Cohort Study	> 1,000	M F	40–69	20	BMI	SBP & DBP	Diet	Changes in average BP over time, unlikely to be related to weight gain
Kemper et al.	1999	Amsterdam Growth and Health Longitudinal Study	500	M F	13	20	BMI, sum of four skinfold thickness		Diet, physical activity	Association between skinfolds and HBP
Kuller et al.	1995	The Healthy Women Study	541	F	42–50	Cross-sectional	BMI	SBP & DBP	Pre-menopause	
Martikainen & Marmot	1999	Whitehall II Study	7,973	M F	35–55	6–8	BMI, WHR	HBP	Age, BMI at 25 years	Independent association
McCarron & Reusser	1996	Trials of Hypertension Prevention Phase I (TOPH I)	2,000 +	M F		Cross-sectional	BMI	Weight loss causes reductions in BP	Age	Yes. BMI accounts for 25–30% variation in BP in populations
Rissel & Russell	1993		389	M F		Cross-sectional				
Rosner et al.	2000	Eight large US epidemiological studies	47,196	M F	5–17	Cross-sectional	BMI obesity ≥ 95th P, HBP ≥ 95th P		Height, weight, study	Strong association. RR = 2.5–3.7

(continued)

Table A2 (continued): Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Selby et al.	1989	Kaiser Permanente Medical Care Program	1,031				BMI, subscapular & triceps skinfold		Age, sex, race,	Strong association between centrally deposited body fat, BMI and HBP
Smith et al.	1992a	Kimberley Region Study Mar 88–Sept 89	490	M F	≥ 15	Cross-sectional	HBP ≥ 160/95 mm/Hg, BMI & skinfolds	HBP		Strong association between overweight and HBP. Not so strong for obesity except among middle-age-women.
Smith et al.	1992a	Kimberley Region Study Mar 88–Sept 89	490	M F	≥ 15	Cross-sectional	HBP ≥ 160/95 mm/Hg, BMI & skinfolds	HBP, ECG abnormalities	Age, sex	HBP 2–3 times higher in Indigenous Australians.
Sorof et al.	2002	8 Houston urban public high schools	2,460	M F	12–16	Cross-sectional	BMI obesity ≥ 95th P, HBP ≥ 95th P	HBP		Strong association RR = 3
Stamler	1991	People's Gas Company Medical Department in Chicago	746	M	Young adults	20	Relative weight, HBP = DBP ≥ 95 mmHg	HBP	None	Yes

(continued)

Table A2 (continued): Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Stamler	1991	CARDIA		M F	15–30	Cross-sectional	BMI	HBP	Smoking, alcohol intake, potassium and calcium intake, demographic and psychosocial traits	Linear
Stamler	1991	Chicago Heart Association Detection Project	21,024	M F	25–44 45–64	Cross-sectional	SBP ≥ 160 mmHg; DBP ≥ 95 mmHg	HBP	Age, heart rate, serum cholesterol level, 1-hour postload plasma glucose level, serum uric acid level, smoking	Independent
Stamler	1991	INTERSALT	10,079	M F	20–59	Cross-sectional	HBP ≥ 140/90 mmHg	HBP	Age, sex sodium & potassium excretion, alcohol intake, smoking status	Significantly independent association

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Table A2 (continued): Studies included for high blood pressure

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Stamler	1991	Western Electric Study	1,860	M		8		HBP		Significantly independent association
Trials of Hypertension Research Group	1997	Trials of Hypertension Prevention Phase II (TOPH II)	2,382	M F	30–54	3–4	HBP ≥ 140/90 mmHg Overweight = BMI 110% to 165% of their desirable body weight	HBP	Current hypertension, CVD, NIDDM, renal insufficiency or other serious illness, current/planned pregnancy	Weight loss causes a reduction in BP.
van Itallie	1985	NHANES II		M&F	20–75	Cross-sectional	BMI, triceps & subscapular skinfold thickness, HBP ≥ 160/95	HBP	Age, sodium & potassium excretion, alcohol intake, smoking status	Strong association
WHO	2000	WHO MONICA Study								Yes. RR = 2–3

Table A3: Studies included for high blood cholesterol

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Dattilo & Kris-Etherton	1992	Meta-analysis of 70 studies				Cross-sectional				Weight loss had positive association on TC, triglycerides and LDL cholesterol and an inverse association with HDL cholesterol.
Denke et al.	1993	NHANES II & review of other studies	4,834	M	20–74	Cross-sectional	BMI	High cholesterol levels	Smoking, intake of saturated fats (% of energy), daily dietary cholesterol intake	Linear trend Linear regression
Denke et al.	1993	Brooks Air Force Study	1,264	M	17–64		% of body fat and BMI	High cholesterol levels		BMI positively associated with TC and triglycerides
Denke et al.	1993	Chicago Heart Association Detection Project in Industry	33,648	M F	20–55		Relative weight	High cholesterol levels		Relative weight positively associated with TC
Denke et al.	1993	CARDIA Study	4,955	M F	18–30		BMI	High cholesterol levels		BMI significantly associated with LDL cholesterol
Denke et al.	1993	Lipid Research Clinics Program Prevalence Study	6,865	M F	4–79		BMI	High cholesterol levels		BMI positively associated with triglycerides Ages 12–79 BMI inversely related to HDL cholesterol

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Table A3 (continued): Studies included for high blood cholesterol

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Denke et al.	1993	Baltimore Longitudinal Study of Aging								Changes in BMI causes changes in TC
Denke et al.	1993	Western Electric Study								Changes in BMI causes changes in TC
Denke et al.	1993	Framingham Offspring Study	4,304	M F	16–49		BMI	High cholesterol levels		Changes in BMI causes changes in TC. BMI positively associated with LDL cholesterol esp. aged 20–29
Despres et al.		Review of numerous studies								Abdominal obesity related to cholesterol levels
Ernst & Obarzanek	1994	Bogalusa Heart Study		M F	5–14	11	Ponderal index, BMI, skinfold thickness	High cholesterol levels		Yes
Ernst & Obarzanek	1994	Muscatine Study	2,367	M F	8–18	12	Ponderal index, BMI, skinfold thickness	High cholesterol levels		Yes

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Table A3 (continued): Studies included for high blood cholesterol

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Ferrara et al.	1997	Rancho Bernardo Study	2,344	M F	50–93	8 917 people	BMI	High cholesterol levels	Age	Changes in BMI caused positive association with changes in LDL cholesterol and TC; inversely with changes in HDL cholesterol.
Freedm-an et al.	1999	Bogalusa Heart Study	9,167	M F	5–17	Cross-sectional	BMI and Rohrer Index Overweight = BMI≥27 High lipids: TC > 200 mg/dL LDL > 130mg/dL TG ≥ 130 mg/dL HDL < 35 mg/dL= low	High cholesterol levels	Race, sex, age	Weakly related to TC and LDL cholesterol and strongly related to HDL cholesterol and TG
Garn et al.	1988	Tecumseh (MI) Community Health Survey	5,507	M F	15–75	Cross-sectional	Skinfolds: tricep, subscapular, iliac & abdominal & sum	High blood cholesterol	None	Associated

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Table A3 (continued): Studies included for high blood cholesterol

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Owen et al.	2003	Ten Towns Heart Health Study	1,532	M F	13–16		Birth weight, length of gestation, crown–heel length, head circumference	Total blood cholesterol, BMI at adolescence	Age, sex, town and ethnicity	No association between birth weight and cholesterol levels at adolescence
Stamler et al.	1997	Multiple risk factor intervention trial (MRFIT)	6,428 at baseline	M		1–6	BMI	≥ 10% reduction in serum total cholesterol	Smoking, DBP, baseline lipids, antihypertensives.	Weight loss associated with lowering of cholesterol
WHO	2000	WHO MONICA Study								Yes. RR = 2–3

Table A4: Studies included for Type 2 diabetes

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Brancati et al.	1999	John Hopkins Precursors Study (measured at baseline, self-report at follow-up)	916	M	20–29	27	BMI	NIDDM	Age, physical activity, maternal history of diabetes, smoking	BMI at 25, 35 and 45 years of age all strongly predict diabetes risk (RR for overweight BMI ≥ 25 vs non-overweight were greater than 3.0).
Cassano et al.	1992	Normative Ageing Study cohort	1,972	M	22-80	24	BMI, Waist-to-hip ratio	NIDDM	Age, cigarette smoking, overall adiposity	BMI independently related to the risk of impaired glucose tolerance and risk of diabetes. Men had 1.3-fold greater risk of NIDDM in highest BMI group than lowest BMI group Rate of NIDDM increased by tertile of WHR (across all levels of BMI) At end examination, 22% had impaired glucose tolerance, 11.5% were NIDDM and 66.5% normal.
Chan et al.	1994	Health Professionals' follow-up Study Self report	51,529	M	40–75	5	BMI, WHR, absolute fat mass, % body fat	NIDDM	Age, family history, smoking	Risk of diabetes increased continuously with levels of BMI
Cho et al.	2002	Nurses' Health Study Self-report	5,897	F	40–74 years	20	BMI	NIDDM	Age, smoking and other coronary risk factors	Obesity and weight gain were associated with future risk of CHD among women with NIDDM

(continued)

Table A4 (continued): Studies included for Type 2 diabetes

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Colditz et al.	1990	Nurses' Health Study Self-report	113,861	F	30–55	8	BMI	NIDDM	Age, family history	Increasing risk of NIDDM with increase in BMI.
Daniel et al.	1999	Aboriginal and Torres Strait Islander people	882	M F	15–77	8	BMI	NIDDM	Age, sex, community	BMI-specific rates: 10.7 to 47.2 cases/1,000 person-years. RR for BMI categories: 3.3 (1.5-7.0) for < 25kgm^2 , 2.7 (1.1–6.8) 4.4 (1.7–11.6)
Dunstan et al.	2000	AusDiab	11,247	M F	25+	Cross-sectional	BMI	Glucose tolerance, NIDDM	Age, sex	Diabetes was associated with hypertension (69.3%), obesity (44.4%), LDL (45.9%), HDL (23.1%), triglycerides (42.9%).
Folsom et al.	1991	Coronary Artery Risk Development in Young Adults (CARDIA), Atherosclerosis Risk in Communities (ARIC) studies	CARDIA = 5,115 ARIC = 15,803	M F	18–30 45–64	2 2	BMI, WHR, skinfolds	Glucose, NIDDM	Age, cigarette smoking	Obesity defined by skinfold thickness was positively associated with cholesterol, BP, serum glucose and insulin and the prevalence of Type 2 diabetes. The strength of the association was similar across African-Americans and Caucasians. For middle-aged African-Americans, the risk increased.

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Table A4 (continued): Studies included for Type 2 diabetes

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Ford et al.	1997	First National Health and Nutrition Examination Epidemiological Follow-up Study (NHEFS) (Measured at baseline, self-report at follow-up)	8,545	M F	18+	21	BMI	NIDDM	Age, sex, race, education, smoking and other risk factors	Weight gain over a 10-year period was strongly associated with increased risk for diabetes. Every kg increase, the risk for diabetes increases 4.5%. No differences across age, sex or race.
Guest et al.	1993	Aboriginal and Torres Strait Islander people	306 Aborigines 553 other Australians	M F	13+	Cross-sectional	BMI, WHR			Abdominal obesity is high in urbanised Aborigines. BMI is more associated with glucose intolerance than WHR.
Haffner et al.	1991	San Antonio Heart Study	2,117	M F	25–64	3	BMI, skinfolds	NIDDM	BMI	Central adiposity was more strongly associated with diabetes incidence in women than men, when adjusted for BMI

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Table A4 (continued): Studies included for Type 2 diabetes

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Janssen et al.	2002	NHANES III	14,924	M F	17+	Cross-sectional	BMI, WC	Blood pressure, IRS (insulin resistance syndrome), blood cholesterol	Age, race, alcohol intake, smoking, physical activity, poverty-to-income ratio	Include both measures BMI and WC in studies. High WC (indicative of high abdominal (visceral) fat) is associated with hypertension, Type 2 diabetes, dyslipidemia, and metabolic syndrome. Increases with age. Women higher than men
Leonard et al.	2002	TSI	592	M F	15+	Cross-sectional	BMI	NIDDM		High prevalence of overweight (30%), obesity (51%), abdominal obesity (70%), diabetes (26%), hypercholesterolaemia (33%), albuminuria (28%), hypertension (32%) and tobacco smoking (45%).
Lundgren et al.	1989	Gothenburg	1,462	F	38, 46, 50, 54, 60	12	BMI, skinfolds, WHR	NIDDM	Age, smoking, SBP, intake of antihypertensive drugs, serum cholesterol, triglyceride, and glucose concentrations	BMI, skinfolds, WHR were significantly associated with the incidence of NIDDM.
McDermott et al.	2000	A&TSI	335	M F	15+	8 x cross-sectional	BMI, WHR	NIDDM		In younger women (15–24 years) a trebling in obesity, and four- to five-fold increase in diabetes prevalence.

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Table A4 (continued): Studies included for Type 2 diabetes

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Njolstad et al.	1998	Finnmark Study	11,654	M F	35–52	12	BMI	NIDDM	Age, ethnicity, smoking, BP, physical activity, cholesterol, height	BMI RR = 2.22 (1.98–2.50) for men, RR = 1.72 (1.57–1.88) for women. Strong dose–response relation between diabetes and BMI for both sexes
O'Dea et al.	1990	A&TSI	122	M F	17+	Cross-sectional	BMI, WHR	Glucose tolerance, NIDDM		High levels of diabetes despite relative leanness of sample.
Perry et al.	1995	British regional heart study (Measured at baseline, self-report at follow-up)	7,735	M	40–59	12.8 years	BMI	NIDDM	Age, BMI, risk factors	Risk of NIDDM increased with increasing BMI. RR = 11.6 (5.4, 16.8)
Skarfors et al.	1991	Uppsala, Sweden	2,322	M	47–53	7–14	BMI	NIDDM		BMI independent predictor of diabetes in middle-aged men
Wanna-methee & Shaper	1999	British regional heart study (Measured at baseline, self-report at follow-up)	6,916	M	40–59	12	BMI	NIDDM	Age, initial BMI and other risk factors.	BMI is strong and continuous predictor of NIDDM. Substantial weight gain (> 10%) was associated with significant increase in risk of NIDDM (RR = 1.65).

Table A5: Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Adams-Campbell et al.	1995	African-Americans undergoing coronary angiography	866	M F		Retrosp-ective review of 7 years of data	BMI	Coronary artery disease	Age, smoking, diabetes, lipid levels	Inverted U-shaped relationship between BMI and coronary artery disease
Baik et al.	2000	Health Professionals Follow-Up Study	39,756	M	40–75	10 years	BMI, WC	CVD mortality	Age, smoking status and quantity, family history of myocardial infarction before age 60 years, profession, marital status, height, alcohol intake and dietary components	Among men aged < 65 years, CVD risk increased linearly with greater BMI. Among men aged 65 years or more, there were no significant relations between BMI and CVD risk; however, waist circumference strongly predicted death from CVD among older men.
Brown et al.	1998	Women's Health Australia project	13,431	F	45–49		BMI	CVD	Area of residence, education, smoking, exercise and menopausal status	Healthy weight range for middle-aged women to reduce the risk of CVD is about a BMI of 19 to 24.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Field et al.	2001	Nurse Health Study (women) Health Professionals Follow-Up study (men)	46,060 77,690	M F		10 years	BMI	CHD incidence, stroke	Age, smoking status and race	Risk of incidence increased with severity of overweight
Freedman et al.	2001	Bogalusa Heart Study	2,617	M F	2–17	Mean of 17 years	BMI	CHD	Age, sex, race, examination year and adult BMI	Childhood overweight was related to adverse risk factor levels for CHD but associations were weak and were attributable to the strong persistence of weight status between childhood and adulthood.
Harmsen et al.	1990	Swedish study	7,495	M	47–55	12 years	BMI	Stroke	Stroke risk factors	BMI was not a risk factor for stroke.
Harris et al.	1993	Epidemiologic Follow- Up Study of NHANES I	1,259	F	65–74	14 years	BMI	CHD incidence	Age, smoking	Overweight (BMI > = 29) is an independent risk factor for CHD in older women, finding strengthened after previous weight loss is accounted for.
Harris et al.	1997	Epidemiologic Follow- Up Study of NHANES I	621 960	M F	Mean age 77	Up to 13 years	BMI	CHD incidence	Age, smoking status	Heavier weight in late middle age was a risk factor for CHD in late life. Heavier weight in old age was a risk factor after excluding those with substantial weight loss.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Harris et al.	1988	Framingham Heart Study	597 1,126	M F	55–65	Up to 23 years	BMI	Mortality	SBP,blood glucose, serum cholesterol, smoking, prior CVD	U-shaped (but thinness and mortality dependent on illness at baseline)
He et al.	2001	NHANES I Epidemiologic Follow-Up Study	5,545 8,098	M F	25–74	Average of 19 years	Overweight defined as BMI of 27.8 or greater	Incidence of congestive heart failure	Sex, education, physical inactivity, smoking, diabetes, high blood pressure, valvular heart disease and CHD	Overweight is an independent risk factor for congestive heart failure.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Hubert et al.	1983	Framingham Heart Study	2,252 2,818	M F	28-62	26 years	MRW (ratio of actual weight to desirable weight as %)	CHD Stroke Congestive heart failure	Age Systolic BP Serum cholesterol Cigarettes per day Glucose intolerance Left ventricular hypertrophy	MRW was a risk factor for CHD independent of age, cholesterol, systolic blood pressure, smoking, left ventricular hypertrophy and glucose intolerance.
Jousilahti et al.	1996	Follow-up of middle-aged man and women in Eastern Finland	16,113	M F	30–59	15 years	BMI	CHD mortality	Age, smoking, serum cholesterol, systolic BP	Obesity was an independent risk factor for CHD mortality among men and contributed to the risk of CHD death in women
Kahn et al.	1996	Case-control	217 cases 261 controls	M F	< 70		Ratio of waist to thigh girth, abdominal diameter	CHD	Smoking, history of high BP, high cholesterol or diabetes	Increased mid-thigh girth and subcutaneous fat mass (sum of 3 skinfolds) were associated with a protective effect against CHD.
Kannel, D'Agostino & Cobb	1996	Framingham Heart Study		M F	35–70	26 years	BMI	CHD, stroke		Each SD in relative weight gain confers 15% and 22% increases in men and women, respectively. The optimal weight for avoidance of CVD corresponds to a BMI of 22.6 for men and 21.1 for women.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Kannel et al.	1991	Framingham Heart Study	2,039 2,671	M F	35–70	24 years	Regional, central or abdominal obesity	CHD, stroke, PVD chronic heart failure	Serum cholesterol, SBP, blood glucose, uric acid	Linear trend. CVD is closely linked to abdominal and general adiposity.
Kenchaiah et al.	2002	Framingham Heart Study	2,704 3,177	M F		Mean 14 years	BMI	Incidence of heart failure	Age, smoking status, alcohol consumption, serum cholesterol, valve disease, high BP, diabetes, ECG LVH, MI	Stepwise increase in risk of heart failure across increasing categories of BMI
Kurth et al.	2002	Physicians' Health Study	21,414	M		12.5 years	BMI	Stroke (ischaemic and haemorrhagic)	BP, diabetes, cholesterol	Significant increase in the RR of stroke of total stroke and its two major subtypes with each unit increase of BMI independent of high BP, diabetes and cholesterol.
Manson et al.	1990	Nurses' Health Study	115,886	F	30–55	8 years	BMI	CHD	Age, smoking status and quantity	Strong positive association between obesity and risk of CHD in women.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Manson et al.	1995	Nurses' Health Study	115,195	F	30–55	16 years	BMI	CHD mortality	Age, smoking status and quantity, menopausal status, oral contraceptive and postmenopausal hormone use, parental history of myocardial infarction before age 60 years	The lowest mortality was observed among women who weighed 15% less than the US average for women of similar age and among those whose weight had been stable since early adulthood.
Rao et al.	2001	Review of several studies					BMI, WHR	CHD		Both a high BMI and a high WHR are independent risk factors for CHD and mortality.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Rexrode et al.	1997	Nurses' Health Study	116,759	F	30–55	16 years	BMI	Stroke	Age, smoking status and quantity, oral contraceptive use, menopausal status, hormone replacement therapy and time period	Both obesity and weight gain are important risk factors for ischaemic and total stroke but not for haemorrhagic stroke.
Rexrode et al.	1998	Nurses' Health Study	44,702	F	40–65	12 years	BMI, WHR, WC	CHD	Age, smoking, parental history of heart attack, alcohol intake, physical activity, menopausal status, hormone replacement therapy, oral contraceptive use, aspirin intake, saturated fat intake and antioxidant score	The WHR and WC are independently associated with CHD risk in women.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Rimm et al.	1995	Health Professionals Follow-Up Study	29,122	M	40–75	3 years	BMI, WHR	Incidence of CHD	Baseline BMI, vitamin E use, age, smoking status, alcohol use, total calories, profession and family history of CHD	Among men less than 65, BMI was better than WHR at predicting CHD risk. For older men (≥ 65 years), measures of fat distribution may be better than BMI at predicting CHD risk.
Shaper et al.	1997	Prospective study of a male cohort	7,735	M	40–59	Mean 14.8 years	BMI	CVD mortality	Age, smoking, social class, alcohol intake and physical activity	A healthy BMI among this cohort of men appeared to be about 22.
Shinton, Sagar & Beevers	1995	British case control study	125 cases 198 controls	M F	35–74		Subscapular skinfold thickness	Stroke	Age, smoking, race, saturated fat intake, exercise for those aged 15 to 25	Those in the thinnest and fattest quartiles of subscapular skinfold thickness were at increased risk of stroke compared with those in the middle quartiles.
Singh & Linstead	1998	Adventist Mortality Study	12,576	F	30–74	26 years	BMI (based on self-reported height and weight)	CHD, hypertensive disease, stroke	Age, smoking	U-shaped, particularly among women in the fifth to seventh decade of life.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Spataro et al.	1996	Western Electric Study	1,707	M	40–55	22 years	BMI, % body fat (1), % body fat (2) triceps, subscapular, sum of skinfolds, body weight	CHD mortality	Age, SBP and DBP, serum cholesterol, education, minor and major ECG abnormalities, smoking, alcohol, heart rate	After 15 years follow-up, all adiposity measures, except triceps skinfold, were significantly related to CHD mortality.
Stevens et al.	1998	American Cancer Society's Cancer Prevention Study I	324,125	M F	30–74	12 years	BMI	CVD mortality	Age, education, physical activity and alcohol consumption	Increased BMI increases the risk of death from CVD in adults between 30–74 years of age with RR being greater among younger adults.
Suk et al.	2003	Northern Manhattan Stroke Study		M F			BMI, WHR	Stroke	Stroke risk factors	Abdominal obesity is an independent risk factor for stroke. It is stronger than BMI and has a greater effect among younger persons.
Walker et al.	1996	Health professionals	28,643	M	40–75	5 years	BMI, WHR	Stroke	Age, physical activity, vitamin E, vitamin C and carotene	Abdominal obesity but not increased BMI, predicts risk of stroke in men.

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Table A5 (continued): Studies included for CVD

Authors	Year	Study	N	Sex	Age	Follow-up	Index of overweight/ obesity	Outcome	Confounders considered	Relationship
Wang & Hoy	2002	Australian Aboriginals	744	M F	20–77	5,041 person-years	BMI	CVD	Age, sex, smoking, drinking status	Individuals with a relatively lower BMI have a lower risk of death.
Willett et al.	1995	Nurses' Health Study	115,818	F	30–55	14 years	BMI	CHD	Age, smoking status, menopausal status, current and past use of postmenopausal hormones, parental history of myocardial infarction before 60 years of age	Higher levels of body weight within the 'normal' modest weight gains after 18 years of age appear to increase risks of CHD in middle-aged women.
Wilson et al.	2002	Framingham Heart Study		M F	35–75	Up to 44 years	BMI	CVD incidence	Age, smoking, high blood pressure, high cholesterol and diabetes	Overweight and obesity are associated with increased CVD incidence.
Zhou et al.	2002	14 target populations among the main regions of China	19,741	M F	35–59	~9 years	BMI, WC, hip circumference	Stroke CHD Total death		Overweight was an independent risk factor for stroke and CHD.

Abbreviations

AIHW	Australian Institute of Health and Welfare
AusDiab	Australian Diabetes, Obesity and Lifestyle Study
BMI	Body mass index
BP	Blood pressure
CARDIA	Coronary Artery Risk Development in Young Adults
ARIC	Atherosclerosis Risk in Communities
CI	Confidence interval
CHD	Coronary heart disease
CT	Computerised tomography
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
HBP	High blood pressure
HDL	High-density lipoprotein cholesterol
IMT	Intimal–medial thickness
ITCI	Iliac-to-thigh circumference index
LAD	Left anterior descending coronary artery
LDL	Low-density lipoprotein cholesterol
MONICA	MONItoring trends and determinants of CArdiovascular disease
MRW	Metropolitan relative weight
NHANES	National Health and Nutrition Examination Survey (US)
NHDC	National Health Data Committee
NHFA	National Heart Foundation of Australia
NIDDM	Non-insulin dependent diabetes mellitus
NIH	National Institutes of Health
OR	Odds ratio
RCA	Right coronary artery
RR	Relative risk
RW	Relative weight
SBP	Systolic blood pressure
SD	Standard deviation
TC	Total cholesterol
TG	Triglycerides
WC	Waist circumference
WHO	World Health Organization
WHR	Waist-to-hip ratio

Glossary

Body mass index	A person's weight in kilograms divided by the square of their height in metres.
Metropolitan relative weight	Ratio of actual weight to desirable weight
Relative weight	Ratio of observed weight to desirable weight for height x 100.