



**Heart Foundation**

**A review of the relationship between  
dietary fat and overweight/obesity**

**Nutrition and Metabolism Advisory Committee**

**National Heart Foundation of Australia**

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This paper was prepared by the National Heart Foundation of Australia's Nutrition and Metabolism Advisory Committee (NMAC) with the assistance of Veronique Droulez who was contracted for this process.

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This evidence-based paper was developed through an extensive review and consultation process. A Working Group consisting of Dr Manny Noakes, A/Prof David Crawford, Veronique Droulez, Susan Anderson and Barbara Eden guided the development of this review paper. The NMAC considered several drafts of the review paper and the paper was subsequently revised based on the comments from committee members.

A position statement on Dietary fat and overweight/obesity was developed based on this evidence-based paper. Dr Manny Noakes, Chair of the NMAC and Barbara Eden, Executive Officer of the National Nutrition Program, with Veronique Droulez, developed the position statement. The position statement was reviewed and modified by the working group and then circulated to National Nutrition Staff followed by the NMAC for comment. Comments were incorporated and the statement was reviewed again by the Working Group.

The NMAC approved both the review paper and position statement in July 2002. The review paper and position statement were presented to the National Heart Foundation of Australia Cardiovascular Health Advisory Committee (CVHAC) and after minor amendments, the CVHAC members approved both documents in November 2002. The medical members of the National Board also provided comment and the National Board approved the Heart Foundation's review paper and position statement in February 2003.

The Heart Foundation's Position Statement on 'Dietary fat and overweight/obesity', based on this evidence-based review paper is published in *Nutrition & Dietetics* 2003;3:174-6.

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## 1. Background

The National Heart Foundation of Australia's review of the relationship between dietary fat and cardiovascular disease (CVD) found good evidence of an association between an increased intake of saturated fat and increased risk of coronary heart disease (CHD) and that replacing the saturated fat with polyunsaturated, and to a lesser extent monounsaturated, type fats improves CVH risk factors (National Heart Foundation of Australia 1999). This review also indicated little evidence linking coronary events or death to the amount of total fat in the diet. Consequently, the National Heart Foundation of Australia (NHFA) policy on dietary fat does not include a recommendation for intakes of total dietary fat for the prevention of CVD. However, it has been suggested that dietary fat intake could increase the risk of CVD indirectly by increasing the risk of overweight and obesity.

Obesity is an independent risk factor for morbidity and mortality related to coronary heart disease (CHD) (World Health Organization (WHO) 1998; Krauss et al. 1998; Willett 1995; Rimm et al. 1995). It is associated with risk factors of CHD such as hypertension, non insulin dependent diabetes mellitus, alterations in homeostatic variables, hypertriglyceridaemia and reduced high density lipoprotein cholesterol (HDL) levels (Hubert 1983; Krauss et al. 1998). Weight gain increases the risk of heart disease and mortality in both moderate and severe overweight (Calle et al. 1999; WHO, 1998). Short term studies suggest weight loss (of up to 10%) improves several health biomarkers, including blood pressure and serum lipids (WHO, 1998). However, the benefits of long term weight loss in terms of morbidity and premature mortality is yet to be demonstrated in well-designed studies (Institute of Medicine 1995; WHO 1998).

Body Mass Index (BMI) is recommended for assessing overweight and obesity and monitoring changes in body weight (National Institute of Health (NIH) 1998). In adults, a BMI greater than or equal to 25 is classified as overweight and a BMI greater than or equal to 30, obesity (WHO 1998; NIH 1998). In 1995, 63.7% of adult Australian men and 47.0% of women were overweight or obese (Australian Bureau of Statistics (ABS) 1997). In children aged seven to 15 years, 15.0% of boys and 15.8% of girls were overweight and a further 4.5% of boys and 5.3% of girls were obese (Magarey et al. 2001). In both adults and children, the prevalence of overweight and obesity has increased substantially since the 1980s (Eckersley 2001; Magarey et al. 2001).

There are many factors implicated in weight gain, including genetic, metabolic, psychosocial and environmental influences (NIH 1998). It has been argued that the dramatic increase in the prevalence of obesity around the world is primarily due to environmental factors, primarily sedentary lifestyles and consumption of high-fat energy-dense diets (WHO 1998; Walker et al. 1999b).

Dietary fat reduction has been recommended as a useful weight management strategy (Bray & Popkin 1998; WHO 1998). More recently, the association between dietary fat and obesity has been questioned (Shah & Garg 1996b; Willett 1998). According to Willett (1998), the increasing prevalence of obesity in the US since

1976, despite an apparent decline in percent energy from dietary fat, suggests that there is no association between dietary fat intake and overweight. However, Bray & Popkin (1999) argue that the decline in fat intake might not be sufficient to influence the incidence of obesity. Furthermore, underreporting of higher fat foods may underestimate percent energy from fat.

To determine whether a recommendation for dietary fat intake was required in terms of CVD risk reduction, a rigorous review of the scientific literature was conducted to investigate the relationship between dietary fat and overweight/obesity. Interactions between dietary fat and other dietary factors as well as physical activity levels, body weight status, gender and age were considered.

## 2. Methodology

The specific research questions of this review were:

- To determine whether dietary fat\*, independent of energy intake, is a risk factor for the development and progression of overweight and obesity
- To assess the effectiveness of fat reduction strategies relative to other dietary strategies for achieving weight loss in overweight and obese individuals and weight maintenance in normal weight, overweight and obese individuals.

\*Dietary fat intake measured as either % energy from fat (% E fat) or/and absolute fat intake (grams), adjusted for energy intake, were examined.

Articles were identified by searching Medline and the Cochrane Library up until July 2001. Keywords included “dietary fat”, “dietary protein”, “dietary carbohydrate”, “dietary fibre”, “glycaemic index”, “body weight”, “BMI”, “overweight”, “satiety” and “energy density” as well as key authors. Citations were identified from key reviews on the topic, clinical guidelines for the management of overweight and obesity and through consultation with colleagues working in the area.

Abstracts were assessed to determine whether the study was designed specifically to investigate the effect of diet composition on body weight. Animal studies were excluded. Studies were included if they contained a measure of body weight at baseline and post intervention (i.e. BMI, absolute weight loss or percentage of body weight loss). The effect of weight loss on CVD risk factors was not examined.

Long-term follow up of a representative cohort is considered the ideal study design for answering questions related to aetiology and risk factors (National Health and Medical Research Council (NHMRC) 2000). To determine whether the association between dietary fat intake and the development of overweight and obesity is causal, it is recommended that issues such as biological plausibility and the existence of a dose-response relationship be considered. Randomised controlled trials are recommended for determining the effects of an intervention. Controlled trials (ad libitum and energy controlled), of at least 6 weeks in duration, were reviewed to assess the effectiveness of fat reduction compared to other dietary strategies on weight reduction or weight maintenance.

The following criteria were considered in appraising the evidence (Sempos et al. 1999; Seidell 1998; NHMRC 2000):

- Consistency in the evidence across a range of study designs.
- The quality of the study, particularly relating to measurement bias.
- The size of the effect.
- Demonstration of a biologically plausible mechanism.
- Relevance of the evidence to the Australian population.

The following framework was used to assess the evidence:

	<b>'Good Evidence'</b>	<b>'Moderate Evidence'</b>	<b>'Little Evidence'</b>
<b>Level of evidence</b>	Consistency across several study designs, including long term intervention studies	Inconsistency across study designs; Use of surrogate measures; Limited number and type of studies	Inconsistency across study designs; Limited number and type of studies
<b>Quality of the evidence</b>	Measurement bias adequately minimised	Limited in quality	Limited in quality
<b>Size of the effect</b>	Statistically significant.	Effect possibly due to measurement bias	Effect possibly due to measurement bias
<b>Mechanism</b>	Metabolic studies in humans	Metabolic studies in humans	Lack of metabolic studies in humans

### 3. Evidence from epidemiological studies

#### 3.1 Ecological studies

Several ecological or between-population studies have used aggregate level population data (usually national food balance data) to correlate dietary fat intakes with prevalence of overweight and obesity (Bray & Popkin 1998; Lissner & Heitmann 1995). A positive association between consumption of dietary fat (% E fat) and BMI have been reported in some studies, but not in others (Bray and Popkin, 1998; Lissner & Heitmann 1995). It is generally acknowledged that evidence from ecological studies is limited due to differences in physical activity levels, smoking, availability of food and affluence, as well as the variable quality of the dietary intake data (Willett 1999a; Bray & Popkin 1998; Lissner & Heitmann 1995).

### **3.2 Cross-sectional studies**

Many cross-sectional studies have been conducted in individuals within the same population to examine the correlation between intake of dietary fat (mainly % E fat) and body fatness (mainly BMI). Most cross-sectional studies provide statistical evidence of an association (correlation coefficients ranging from 0.17 to 0.38) between % E fat and body fat in free-living populations (Lissner & Heitmann 1995). However, a number of studies have reported no association (Lissner & Heitmann 1995). Similarly, results from cross-sectional studies in children are inconsistent (Davies 1997; Gazzaniga & Burns 1993; Guillaume et al. 1998; Lissner & Heitmann 1995). Inconsistency in study findings may be explained by differences in diet methodology and in the measurement of confounders, such as energy intake, physical activity, age and cigarette smoking (Shah & Garg 1996b). Shah and Garg (1996b) identified four cross-sectional studies which controlled for all potential confounders. Two studies reported an inverse and no relationship between obesity and energy or fat intake, whereas the other two found obese individuals consumed more energy and more dietary fat (independent of energy intake) than lean individuals. However, % E fat explained only 1.6% of the total variation in body fat.

Since indices of obesity and dietary fat intake are measured simultaneously, cross-sectional studies are not considered sufficient to establish causation (Sempos et al. 1999).

### **3.3 Cohort studies**

In adults, weight change is a better indicator of changes in energy balance and body fat than a single measure of body weight (Seidell 1998). Consequently, the evidence from cohort studies, where dietary fat intake has been measured at baseline in a group of individuals and changes in body weight monitored over time, has been examined.

#### **3.3.1 Adults**

Relevant cohort studies identified as part of the literature search are described in Table 1. Participants were selected from health surveys (Rissanen et al. 1991; Paeratakul et al. 1998; Klesges et al. 1992; Jorgensen et al. 1995), on-going cohorts (Colditz et al. 1990; Lissner et al. 1997; Heitmann et al. 1995; Jorgensen et al. 1995) or recruited via advertisements (Kant et al. 1995). Follow-up varied from around 70 to 90%. Baseline dietary fat intake was measured using a range of methodologies and averaged approximately 37% energy from fat (% E fat) in most studies, except for one study conducted in China (about 20% E fat) (Paeratakul et al. 1998). Only one study measured dietary fat intake more than once (Klesges et al. 1992). Most studies analysed the association between weight gain and dietary fat as continuous variables. Lissner et al. (1997) compared weight change in high fat consumers (>38.5% E fat) with low fat consumers (<38.5% E fat). Only one study stratified dietary intake according to quartiles of % E fat but found that there were no clear trends between quartiles of % E fat and weight change in men or women (Kant et al. 1995). Although all studies controlled for total energy intake, not all possible confounding factors were controlled making it difficult to compare study results.

One study reported no association between dietary fat (% E fat) and weight change (Jorgensen et al. 1995). Another study reported a positive association between dietary fat (% E fat) and weight change (Klesges et al. 1992). The positive association between dietary fat and weight change reported in other studies was dependent on age (Kant et al. 1995), gender (Rissanen et al. 1991; Paeratakul et al. 1998; Kant et al. 1995), health status (Kant et al. 1995), activity levels (Lissner et al. 1997), and genetic predisposition (Heitmann et al. 1995).

The association between % E fat and weight change in both men and women appears to be stronger in younger people. Kant et al. (1995) found an inverse association between % E fat and weight change in women aged 50 years and over ( $P=0.55$ ), which became significant in women less than 50 years of age ( $P=0.04$ ).

Several studies reported differences in the association between % E fat and weight change according to gender. In Finland, neither the intake of energy nor that of any of the macronutrients predicted weight gain in men. In Finnish women, the risk of gaining five or more kilograms in 5.7 years in the highest quintile of intake of energy (RR=2.0 (95% CI,1.2-3.3), fat (RR=1.7 (95% CI,1.1-2.7), carbohydrate (RR=1.7 (95% CI,1.0-2.6), and protein (RR=2.0 (95% CI,1.2-3.3), was almost twice that of the lowest quintile, after adjusting for potential confounders (Rissanen et al. 1991). In contrast, studies in China and the US suggest that the association is positive in men but not in women (Kant et al. 1995; Paeratakul et al. 1998).

The Nurses' Health Study suggests dietary intake changes as a result of weight gain (Colditz et al. 1990). Prior to weight gain, there was a positive relationship between weight gain and intakes (grams per day) of total and type of dietary fat as well as sucrose. After subsequent weight gain, an inverse relationship was found between weight gain and total fat, vegetable fat, trans fatty acid, oleic acid, linoleic acid and sucrose. Similarly, the inverse relationship between intakes (grams per day) of protein and dietary fibre and prior weight gain was reversed to a positive relationship with subsequent weight gain.

Physical activity and genetic predisposition was shown to modify the effect of dietary fat on weight gain in women (Lissner et al. 1997; Heitmann et al. 1995). Dietary fat intake was associated with weight gain in sedentary women but not in active women (Lissner et al. 1997). Sedentary women consuming a high fat diet (42.3% E fat and 100.2 g fat/day) gained 2.6 kg over 6 years whereas those on a low fat diet (34.1% E fat and 73.2 g fat/day) lost 0.6 kg (see Table 1).

Genetically predisposed women, with one or more obese parents, were more susceptible to weight gain when exposed to high dietary fat intakes (Heitmann et al. 1995). Women predisposed to obesity who consumed a diet consisting of 40-45% E fat gained 3.5-5.2 kg over 6 years.

The effect of diet on weight gain is small when compared to the effect other non dietary factors have on weight gain. Diet was less predictive of body fat or weight than non dietary factors, particularly physical activity, age and prior weight gain (Rissanen et al. 1991; Colditz et al. 1990; Jorgensen et al. 1995). In men, an increase in fat intake of 100 Kcal was associated with an increase in BMI of 0.036 kg/m<sup>2</sup>. In comparison, an increase in physical activity by one level (physical activity

levels were divided into sedentary, moderate and strenuous according to self reported physical activity level at occupation) was associated with a decrease in BMI of 0.12 kg/m<sup>2</sup> in women (Paeratakul et al. 1998). For both genders, an average increase in age of about two years was associated with an increase in BMI of about 0.11 kg/m<sup>2</sup> (Paeratakul et al. 1998). Colditz et al. (1990) found that age and prior weight gain were much stronger predictors of weight change than dietary intake.

### **3.3.2 Children**

Several cohort studies have been conducted in children. Measurement of obesity in children is problematic due to differences in growth patterns. A simple measure of body fat, such as BMI, is not ideal for assessing obesity in children because it covaries with height (Bellizzi and Dietz, 1999). Until recently, there has not been an internationally acceptable index to assess childhood obesity nor an established cut-off to define overweight in children (Bellizzi and Dietz, 1999). Consequently, obesity has been assessed and defined differently in studies, making it difficult to compare results.

A 3-year study in 146 American preschool children aged 4 years found baseline levels of % E fat were positively associated with changes in obesity status based on 1987 Department of Health and Human Services Norms ( $P=0.05$ ), after adjusting for known risk factors. BMI increased by 0.168 kg/m<sup>2</sup> as % E fat increased by 5% over the 3 yrs (Klesges et al. 1995). However, in a cohort of 112 French children, protein intake (% E protein) at 2 yrs of age, but not dietary fat intake, was positively correlated with BMI and subscapular skinfold at 8 years after adjustment for energy intake and parental BMI (Rolland-Cachera et al. 1995).

A study in 4 year old children, selected according to their familial risk of obesity, reported significantly higher dietary fat intakes (34.4% E fat;  $P=0.0004$ ) in the high risk group (defined as having one to two overweight parents) compared to the low risk group (32.1% E fat) (Eck et al. 1992). After 1 year, the high risk group gained marginally more weight than the low risk group ( $P=0.05$ ). However, the difference in weight gain was small (2.5 vs 2.2 kg) and may have been affected by physical activity, which was marginally lower in the high risk group (Eck et al. 1992).

A 4-year study in 112 children aged 8.6 years found that parents' obesity was the main risk factor for obesity and that dietary intake did not significantly affect risk of obesity (defined as relative BMI >120%) (Maffeis et al. 1998).

A recent analysis of data from an Australian longitudinal study of children (2 to 15 years) over a 13 year-period reported no significant associations between BMI (converted to standard deviation scores) and intake of any macronutrient estimated from three-day and four-day weighed food records (Magarey et al. 2000). However, there was a positive association between dietary fat intake (g/day) and tricep ( $P<0.05$ ) as well as subscapular ( $P<0.01$ ) skinfolds (converted to standard deviation scores). Dietary fat intake (g/day) at 6 years was a significant predictor of BMI (SD score) at 8 years ( $P<0.01$ ) and dietary fat intake (g/day) at 2 years was a significant predictor of subscapular skinfolds (SD score) at 15 yrs ( $P<0.01$ ). Previous body fatness had the greatest effect on subsequent body fatness (Magarey et al. 2000).

The risk of a high fat mass, (defined as percentage of body fat >20% for males and >30% for females and BMI > 25kg/m<sup>2</sup>) estimated from the sum of four skinfolds and body mass, was measured in a Dutch cohort of 13 year-old children who were followed over a period of 20 years until the age of 32 years (Kemper et al. 1999). Lifestyle factors discriminating high- from low-risk participants included physical activity [OR=0.81 (0.69 to 0.96)] and % E protein [OR=1.5 (1.2 to 1.8)] but not % E fat.

### **3.4 Measurement bias in epidemiological studies**

The variables involved in studying the association between dietary fat and obesity are measured by proxy and hence vulnerable to measurement bias (Seidell, 1998). Differences in the methods used to measure obesity and especially dietary fat intake may explain some of the inconsistency across studies.

#### **3.4.1 Under-reporting**

Accurate collection of dietary intake is particularly problematic in nutritional epidemiology. A comparison of total energy intake (indicated by food records) with total energy expenditure (doubly labelled water technique) suggests total energy intake is under-reported by up to 20% in both men and women (Martin et al. 1996). Underreporting may be more prevalent in overweight and obese subjects (Ballard-Barbash et al. 1996) and in women (Macdiarmid et al. 1998). It has been suggested that obese subjects underreport dietary fat intake (measured by a diet history) more than total energy intake (Heitmann & Lissner 1995). Selective underreporting of dietary fat intake (measured by food records) has been reported in obese men (Goris et al. 2000). Failure to report between-meal snack foods suggests that under-reporting may differ according to the type of food (Krebs-Smith et al. 2000; Poppitt et al. 1998). It is unclear whether underreporting differs according to the diet methodology used.

Cross-sectional studies suggest under-reporting can change the relationship between dietary fat and obesity (Gibson, 1996; Macdiarmid et al. 1998; Samaras et al. 1998). Excluding underreporters weakened the association between dietary fat intake and BMI in men. In women, the relationship between absolute dietary fat intake and BMI became significant and the negative relationship (P=0.02) between consumption of high fat sweet products and BMI was reversed to a positive association (P=0.04) when underreporters were excluded (Macdiarmid et al. 1998). A study in 436 middle-aged female twins reported no relationship between intake of total or type of dietary fat and BMI, total or central adiposity after excluding underreporters and controlling for environmental and genetic factors (Samaras et al. 1998).

Dietary methods, such as 24 hour recall, may not capture usual dietary intakes of overweight people who are more likely to be on an energy restricted diet (Ballard-Barbash et al. 1996). Few studies monitored secular changes in dietary intake and physical activity.

### **3.4.2 Absolute dietary fat intake vs percent energy from fat**

The way in which dietary fat intake was measured may also explain differences in the reported association between dietary fat and overweight and obesity. Macdiarmid et al. showed that absolute dietary fat intake was significantly related to BMI in both men ( $P=0.010$ ) and women ( $P=0.003$ ). However, % E fat was significantly related to BMI in men ( $P=0.004$ ), but not in women. Similarly, the inverse relationship between BMI and % E fat, carbohydrate or sucrose was reversed to a positive association when dietary fat was expressed in grams/day (Macdiarmid et al. 1996; Drewnowski et al. 1997).

### **3.4.3 Adjusting for energy intake**

Since dietary fat and energy intake are highly correlated, it is necessary to adjust for total energy intake in order to determine whether the relationship between dietary fat intake and obesity is independent of the effect of energy intake (Beaton et al. 1997). Methods used for adjusting energy intake include the standard multivariate model, the multivariate nutrient density model, the nutrient residual model and the energy-partition model (Willett et al. 1997). Although most cohort studies and recent cross-sectional studies have controlled for total energy intake, different methods for adjusting total energy intake have been used which will influence study results (Beaton et al. 1997). Even so, because dietary fat is so highly correlated with total energy intake, controlled trials are required to distinguish the effects of dietary fat independent of total energy intake (Sempos et al. 1999; Beaton et al. 1997).

### **3.5 Summary of evidence from epidemiological studies**

The association between dietary fat and obesity reported in cohort studies, which were conducted in different population groups, was inconsistent. Underreporting and the limitations of diet methods can partly explain the inconsistency in these findings. The majority of cohort studies measured dietary fat intake only once and none excluded underreporters. Most studies have measured dietary fat intake in terms of dietary fat concentration (% E fat). It is unclear whether the relationship between body weight and dietary fat differs when expressed as energy-controlled absolute dietary fat intake (grams/day).

Few studies reported an association between total energy intake and weight gain, possibly due to the confounding effect of physical activity (Shah & Garg 1996b). Physical activity has generally been inadequately assessed, if at all, relying mainly on self-reported activity levels. Although most studies controlled for energy intake, few controlled adequately for physical activity levels, making it difficult to separate the effect of dietary fat from that of physical activity (Seidell, 1998). In children, physical activity was shown to be an important determinant of overweight and obesity.

The lack of variation in dietary fat intake in study populations and the large within-person variation in dietary intake make it difficult to find an association between dietary fat intake and weight gain in cohort studies (Sempos et al. 1999). Furthermore, the relationship between dietary fat and weight gain may differ according to level of physical activity, age, gender and genetic background. However, compared to the effect of non dietary factors, such as physical activity, on weight change, the effect of dietary fat is small and may not make a significant impact on body weight over the long term (Shah & Garg 1996b; Seidell, 1998). No study reported a dose response relationship between dietary fat intake and weight gain.

Due to these methodological limitations, an evaluation of the relationship between dietary fat intake and overweight and obesity requires consideration of information from observational epidemiology as well as experimental and clinical intervention studies (Sempos et al. 1999).

## 4. Mechanisms

To confirm whether dietary fat plays a role in the aetiology of overweight and obesity suggested in some cohort studies, biologically plausible mechanisms must be identified which explain how dietary fat might increase the risk of weight gain.

It is well established that body weight remains stable as long as energy intake matches energy expenditure (WHO, 1998). Energy intake in excess of energy expenditure results in energy storage and weight gain. Hence dietary fat could affect energy balance by influencing energy storage, energy expenditure or energy intake.

### 4.1 The effect of dietary fat on energy storage

Several experimental studies have shown that protein and carbohydrate promote their own oxidation whereas fat influences its own oxidation only weakly or not at all (Schutz, 1995; Tremblay et al. 1989). An increase in dietary fat intake produces a positive fat balance, whereas an increase in carbohydrate intake results in a negative fat balance (Proserpi et al. 1997; Schutz, 1995). It has therefore been suggested that dietary fat is more efficiently stored than carbohydrate (Proserpi et al. 1997; Schutz, 1995).

The effect of dietary fat on risk of overweight may vary according to the type of fatty acid. Since the type of dietary fatty acid consumed, influences membrane lipid composition, it may also affect metabolic activity (Pan et al. 1994). Some research suggests that saturated fats are predominantly stored whereas n-3 polyunsaturated fats are preferentially oxidized (Storlien et al. 1998). A study in 7 lean and 8 obese subjects over 14 days investigated the effect of meals differing in the ratio of saturated to polyunsaturated fatty acids on components of the thermic effect of food. Overall, total dietary fat, irrespective of type of fat, and carbohydrate oxidation did not differ significantly across groups (Jones et al. 1992).

The effect of macronutrient composition on fat storage may only be evident when excess energy is available for storage in the adipose tissue. Under conditions of energy balance, the body seems capable of adapting to variations in fat and carbohydrate intake without displacing macronutrient stores (Stubbs et al. 1993). Normal weight subjects can adjust fat oxidation in response to increased fat intake within 7 days, depending on physical activity levels (Schrauwen et al. 1997). A study which provided 7 pairs of normal weight identical twins with either a low fat (20% E fat) or high fat (40% E fat) diet over 18 days reported no differences in fat oxidation rates between the two diets. Instead, post-prandial fat and carbohydrate oxidation rates tended to reflect the diet's macronutrient ratios (Salzman et al. 1997).

In conditions of overfeeding, short term studies (conducted over 24 hours or less) suggest that fat overfeeding results in fat storage, whereas carbohydrate overfeeding results in increased carbohydrate oxidation and limited de novo lipogenesis (Shah & Garg 1996b). However, longer term studies (>24 hours) indicate that habitual overfeeding leads to a gain in fat stores, irrespective of the fat and carbohydrate content of the diet (Shah & Garg 1996b). Horton et al. (1995) calculated energy storage in an overfeeding study lasting 14 days. Although carbohydrate overfeeding resulted in 75% to 85% of excess energy being stored compared to 90 to 95% with

fat overfeeding, there was no significant difference between diets in body weight or body composition. By the end of the 14 days there was no difference in the proportion of energy stored as body fat. More recently, a 96-hour continuous whole-body calorimetry study provided 50% excess energy as either fat or different sources of carbohydrate and reported no differences in macronutrient oxidation or fat balance in lean and obese women (McDevitt et al. 2000). The authors concluded that the effect of diet type on fat balance is not mediated through differences in their metabolic actions with respect to disposal or total energy expenditure.

Flatt (1995) proposed that energy regulation is dependent on carbohydrate balance due to its limited storage capacity in the body. It is unlikely that food intake is regulated to maintain carbohydrate balance as proposed by Flatt (Proserpi et al. 1997; Stubbs et al. 1993).

#### **4.2 The effect of dietary fat on energy expenditure**

The components of energy expenditure consist of diet-induced thermogenesis, the basal metabolic rate (BMR) and physical activity. The contribution of diet-induced thermogenesis remains constant at about 10% whereas the contribution of BMR varies from 40 to 60%, depending on the level of physical activity (WHO, 1998).

A study comparing energy balance of 6 normal weight men in a calorimeter with that of free-living conditions found subjects on the medium fat diet (40% E fat) in the sedentary condition were in positive energy balance whereas in the free-living condition, they were in negative energy balance (Stubbs et al. 1995). This study demonstrates the critical role physical activity can play in preventing positive energy balance.

In conditions of energy balance, no differences in energy expenditure have been reported over a range of diets containing from 10-80% E fat (Prentice, 1998). However, a recent study in 12 mildly obese men over four days found that substitution of carbohydrate with 17-18% E as either pork meat or soy protein produced a 3% higher twenty-four hour energy expenditure (Mikkelsen et al. 2000).

#### **4.3 The effect of dietary fat on energy intake**

Since dietary fat may be highly correlated with energy density, it has been proposed that together with its palatability, dietary fat creates a "fat-related hyperphagia", overriding normal signals of satiety and leading to passive overconsumption (Blundell et al. 1999). Several studies suggest that dietary fat exerts relatively weak effects on satiation and satiety compared to carbohydrate and protein (Blundell et al. 1993). Satiety refers to the effects of a specific food or meal on subsequent food intake after eating has ended. Satiation refers to the processes involved in the termination of a meal.

Studies conducted over 2 to 11 weeks in normal weight women and men reported significantly higher energy density and energy intake on high fat diets (40-60% E fat) than on low fat diets (20-25% E fat) resulting in positive energy balance and weight gain (Stubbs et al. 1995; Lissner et al. 1987; Kendall et al. 1991). However, in these

studies, both macronutrient composition and energy density varied, making it difficult to separate the effect of dietary fat from energy density.

Studies in which energy density and palatability were kept constant found no effect of dietary fat content on voluntary energy intake in normal weight women and men (van Stratum et al. 1978; Stubbs et al. 1996; Salzman et al. 1997). A 2-week study provided 22 normal weight women with liquid formula diets and snacks varying in carbohydrate and fat content, but similar in energy density, appearance and palatability (van Stratum et al. 1978). There was no significant difference in energy intake on the high fat (47% E fat) compared to the low fat (24% E fat) diets and body weight was maintained on both diets. A study in which 6 normal weight men had ad libitum access to a low-fat (20% E fat), medium fat (40% E fat) and high fat (60% E fat) diets over 14 days found that the increase in energy intake reported on high fat, high energy dense diets in previous studies no longer existed when energy density was controlled (Stubbs et al. 1996). Similarly, dietary fat intake (20% E vs 40% E) had no significant effect on voluntary energy intake when energy density (5 kJ/g), fibre and palatability were controlled in seven normal weight identical male twins over 9 days (Salzman et al. 1997). These studies suggest that high fat diets do not result in excess energy intake when confounding dietary factors, such as energy density and palatability, are held constant.

#### **4.4 The effect of energy density on energy intake**

Studies where energy density was manipulated independently of fat content confirm that energy density affects energy intake directly. Stubbs et al. (1998) used maltodextrin to manipulate the energy density of high carbohydrate diets (66% E carbohydrate) over 14 days in 6 normal weight men. Voluntary energy intake was higher on the high (6.3 kJ/g) compared to the low (3.6 kJ/g) energy dense diet despite similar proportions of dietary fat in both diets (Stubbs et al. 1998). A study in 18 normal weight women manipulated energy density by changing the proportion of low-fibre vegetables and pasta, hence the water content of foods, whilst keeping palatability and macronutrient composition constant. The amount of food consumed remained constant, so that more energy was consumed when high energy dense foods (4.8 to 5.9 kJ/g) were consumed compared to low energy dense foods (3.1 to 4.0 kJ/g) (Bell et al. 1998).

The independent effect of energy density and dietary fat was determined in a study of 17 lean and 17 obese females over 4 days. Energy density was manipulated by varying the water and fibre content of foods. Both lean and obese women reduced energy intake by 16% in the low (4.4 kJ/g) compared to the high (6.7 kJ/g) energy density condition. In contrast, there was no difference in energy intake when the fat content of foods was manipulated from 16% to 36% E fat (Rolls et al. 1999a). A recent study in lean and obese women examined the effect of energy density on energy intake using meals representative of the typical American diet. There were no differences between lean and obese women. Both consumed 20% less energy on the low (5.23 kJ/g) than on the high (7.32 kJ/g) energy density condition, irrespective of the fat content of the meals (Bell & Rolls 2001). These studies suggest that when palatability and energy density are controlled, fat and carbohydrates have similar effects on energy intake. Hence, energy density, rather than dietary fat per se, is a major determinant of energy intake regardless of macronutrient content (Rolls 2000).

Since high energy dense foods tend to be smaller in amount (by weight or volume) than low energy dense foods, the volume or weight of food may regulate food intake (Rolls, 2000; Drewnowski, 1998). Several studies have shown that people eat a constant weight of food which implies that energy density is a critical determinant of energy intake (Rolls, 2000; Kendall et al. 1991; Stubbs et al. 1996; Stubbs et al. 1998a). However, differences in the weight of food consumed have been reported, suggesting that other factors may affect energy intake. A recent study in 6 normal weight men compared the effect on food intake over 14 days of mixed diets differing in energy density but not in macronutrient composition (39% E fat; 48% E carbohydrate; 13% E protein). Solid food intake decreased as energy density increased where subjects consumed 12% and 19% less solid food on the medium energy dense diet (5.5 kJ/g) and high energy dense diet (7.4kJ/g) than on the low energy dense diet (3.7 kJ/g). Nevertheless, this degree of caloric compensation was insufficient to prevent an increase in energy intake with increasing energy density and weight gain of 0.95 kg on the high energy dense diet (Stubbs et al. 1998a). Subjects lost 1.2 kg on the low energy dense diet and maintained weight on the medium energy dense diet. The authors noted that subjects on the high energy dense diet consumed more fluids and hence failure to differentiate between fluid and food intake may explain differences in study results.

Energy intake has been shown to differ according to the volume of food consumed. The effect of food volume on energy intake may differ from the effect of food weight on energy intake (Bell & Rolls 2001). A study in 20 normal weight men measured energy intake at lunch after preloads varying in volume but similar in palatability, energy and fat content. Energy intake was less after the 600ml preload than after the 300ml preload suggesting that the volume of foods can affect satiety independent of its energy or macronutrient content (Rolls et al. 1998). Another study showed how the water content of the food (eg. casserole vs soup), but not drinking water with a meal, affects energy intake (Rolls et al. 1999). According to Rolls (2000), the water content of foods is a critical determinant of energy density, having a larger effect than fat or fibre.

Changing the energy density of only one or a few foods at a single meal is unlikely to affect energy intake to the same extent as when the energy density of a significant portion of the diet is decreased (Rolls et al. 1999a). A study compared the effect on energy intake and energy density of reducing fat in a single meal compared to all meals consumed over 12 days by 6 normal weight men (Poppitt, 1998). Reducing the fat content of only a portion of the diet was insufficient to reduce overall daily energy intake.

#### **4.5 The effect of palatability on energy intake**

Energy dense foods are generally palatable but not satiating making it difficult to separate these factors from energy density in real life (Drewnowski, 1998). A study in 11 normal weight and 9 post obese subjects over 14 days compared the impact of a high sucrose (29% E fat; 59% E carbohydrate, 23% E sucrose) to a high starch (28% E fat; 59% E carbohydrate; 2% E sucrose) and high fat diet (46% E fat; 41% E carbohydrate; 2% E sucrose) on ad libitum energy intake and body weight (Raben et al. 1997). The energy density of the high sucrose diet (6.9 kJ/g) was comparable to

the high starch diet (6.4 kJ/g) and significantly lower than the high fat diet (8.2 kJ/g). However, average energy intake was significantly higher on the high sucrose (10.3 MJ/d) than on the high starch diets (9.1 MJ/d) and comparable to the high fat diet (10.2 MJ/d). Consequently, there was a significant decrease in body weight (0.7 kg,  $P < 0.05$ ) on the high starch diet compared to the high sucrose and high fat diets. Although the high sugar and high fat diets differed significantly in terms of energy density, they had similar impacts on energy intake. This study therefore suggests that factors affecting satiety such as the palatability of the diet and the form of food consumed may also affect energy intake. The high sucrose diet was the most preferred by subjects and contained large amounts of sucrose-containing drinks which may be less efficient at increasing satiety and suppressing food intake compared with solid foods.

Energy density, palatability and energy intake are highly interrelated. Path analysis showed that the influence of energy density on energy intake was in part direct, and in part indirect and mediated by palatability (McCrorry et al. 2000).

#### **4.6 The effect of other factors on energy intake**

It is necessary to consider the effect of non-dietary factors on energy intake, such as visual, cognitive and physiological cues. For instance, knowledge of eating an amount of food that constitutes a culturally acceptable meal or knowledge of portion size appropriate for the satisfaction of hunger can both affect food intake (Rolls & Bell 1999b). Studies have shown that food labels can influence food intake, particularly in restrained eaters (Miller et al. 1998; Rolls et al. 1992).

The association between the variety of foods consumed and body fatness was analysed using 6-month dietary intake reports from a food frequency questionnaire completed by men and women and accurate measurements of body fatness by underwater weighing. Dietary variety was positively associated with energy intake where individuals who consumed a high variety of sweets, snacks, condiments, entrees and carbohydrates and a low variety of vegetables were more likely to have high levels of body fat (McCrorry et al. 1999). In another study, a positive association between frequency of consuming restaurant foods and body fatness was reported, possibly due to the high energy density, large portion size, palatability and variety of foods available in the restaurant setting (McCrorry et al. 2000). Large portion sizes have been shown to increase energy intake (McCrorry et al. 2000).

Energy intake in response to dietary fat manipulations may be influenced by genetic factors. Familial differences in response to dietary fat manipulations have been reported (Salzman et al. 1997). Within twin pairs, there was a significant relation between proportion of body fat and difference in energy intake between low fat and high fat diets of the same energy density. In this study, three twin pairs consumed more energy in the high-fat phase than in the low-fat phase, three pairs consumed more in the low-fat phase than in the high-fat phase and one pair consumed similar amounts in both phases. Within twin pairs, there was a significant relation between proportion of body fat and difference in energy intake between low-fat and high-fat phases, with fatter twins tending to consume more energy during the low fat diet than during the high fat diet ( $P = 0.038$ ).

#### **4.7 Summary of mechanisms explaining the relationship between dietary fat and obesity**

Experimental studies suggest that energy density, rather than dietary fat per se, is an important determinant of energy intake. The effect of diet on fat balance is not mediated through differences in metabolic action with respect to energy storage and total energy expenditure. High fat diets do not result in excess energy intake when energy density and palatability are held constant (Stubbs et al. 1996; Salzman et al. 1997; van Stratum, 1978). Together, the evidence suggests that factors promoting excess energy intake, rather than the differential metabolic effect of macronutrients on energy expenditure and energy storage, are more likely to increase the risk of weight gain.

Energy density appears to be a major determinant of energy intake. Energy intake is reduced when the energy density of the diet is low ( $\leq 4\text{kJ/g}$ ) and increased when energy density is high ( $\geq 6\text{kJ/g}$ ) (Rolls et al. 1999; Bell & Rolls 2001; Bell et al. 1998; Stubbs et al. 1998a). The effect of energy density on energy intake appears to be similar in both obese and lean individuals.

Energy density is mainly determined by the water, fat and fibre content of foods. More evidence is required to determine the relative effect of the total water, fat and fibre contents of foods on the energy density of the diet.

Other factors such as palatability, the physical form of food and the amount of food (portion size) consumed as well as behavioural and genetic factors may also influence energy intake. Eating patterns, in terms of food variety and frequency of restaurant meals may also affect energy intake.

## **5. Intervention studies**

### **5.1 Energy-controlled dietary intervention studies**

Several studies have investigated the effect on weight loss of varying the fat and carbohydrate content of isocaloric energy restricted diets (Table 2). The studies were mainly conducted in obese women. Treatment allocation was randomized in some studies (Powell et al. 1994; Golay et al. 1996a; Lean et al. 1997) but not in others (Alford et al. 1990; Golay et al. 1996b). One study was conducted in a controlled environment and included exercise and a behavioural program (Golay et al. 1996a). Dietary compliance, based on actual intakes, was measured in free-living subjects in some studies (Powell et al. 1994; Golay et al. 1996b). Alford et al. (1990) monitored dietary compliance without reporting actual dietary intakes. Lean et al. (1997) did not measure dietary compliance and analysis was based on treatment allocation. Only one study controlled for potential confounders, such as physical activity, baseline scores, caloric intake and deficit in caloric intake between baseline and intervention diets (Powell et al. 1994).

In these studies, short-term weight loss in overweight and obese individuals was related to total energy intake. Diet composition, in particular the amount of fat and carbohydrate in energy restricted diets, did not influence short term weight loss. A recent systematic review by Summerbell et al. (1998), compared low-energy, low fat diets (less than or equal to 30% E fat) with low energy diets which were not low in dietary fat (Baron et al. 1986; Lean et al. 1997; Pascale et al. 1995; Racette et al. 1995). Summerbell et al. (1998) concluded that a low energy, low fat diet was as effective as a low energy diet, which is not low in fat, in achieving weight loss in overweight or obese subjects. Intervention studies therefore confirm that energy intake, rather than macronutrient composition, is the major determinant of weight loss in overweight and obese individuals.

Studies suggest that factors other than diet composition may be more important for weight loss. A 3-month randomized controlled trial in 135 overweight subjects compared dietary advice differing in fat, carbohydrate and fibre, but not energy intake (Baron et al. 1986). Club membership was a better predictor of weight loss than diet allocation (Baron et al. 1986). A 6-week study compared the effect of adding aerobic exercise to hypocaloric diets, differing in fat and carbohydrate content, on body composition and weight loss in 23 obese women. There was no significant difference in the effect of diet composition on body composition, whereas aerobic exercise (45 minute sessions three times/week) had a significant effect on fat loss (Racette et al. 1995).

### **5.2 Weight maintenance dietary interventions**

The effect of dietary fat on body weight may differ according to conditions of energy balance. Prewitt et al. (1991) investigated the effect of diet composition on weight maintenance in 18 premenopausal women ranging in BMI from 18 to 44. Women were placed on a high fat diet (37% E fat; 44% E carbohydrate) for 4 weeks followed by 20 weeks on a low fat diet (21% E fat; 59% E carbohydrate). Body weight after 4 weeks on the high fat diet was compared to the last 4 weeks on the low fat diet.

Obese women (BMI>30) lost 2.1 kg on the low fat diet and those with BMI<30 lost 2.0 kg. However, it is difficult to draw conclusions from this study since the analyses did not control for differences in dietary compliance and physical activity levels and the order in which the two diets were fed was not randomized and differed in duration (Prewitt et al. 1991).

Schaefer et al. placed 27 free-living men and women on a low fat, weight maintenance diet (15% E fat; 68% E carbohydrate) for 5 to 6 weeks followed by a low fat ad libitum diet for 10-12 weeks. No weight change occurred on the weight maintenance diet compared to the baseline diet when energy intake was controlled. However, when subjects were allowed to adjust the amount of food consumed, they ate less and lost 3.6 kg (Schaefer et al. 1995) suggesting that fat reduction strategies may facilitate reduced energy intake.

### **5.3 Low fat ad libitum intervention studies**

Several ad libitum dietary intervention studies have investigated the effect of changing dietary fat intake without restricting energy intake, on total energy intake and subsequent body weight in free-living individuals. Studies examined were those specifically designed to investigate the effect of low fat or reduced fat dietary advice on ad libitum energy intake and body weight compared to usual dietary intake in normal weight and overweight individuals (Table 3).

Interventions included those conducted in a worksite environment (Siggaard et al. 1996; Swinburn et al. 1999); a study of diet-related lifestyle changes to reduce breast cancer in at risk-women; and instructions to purchase and consume reduced fat food products (Westerterp et al. 1996; Gatenby et al. 1995). Swinburn et al. (1999) also provided instructions on behavioural skills such as goal setting and self-monitoring. Allocation was either self-selected (Siggaard et al. 1996), according to sex, body weight and household (Gatenby et al. 1995) or randomized (Swinburn et al. 1999; Sheppard et al. 1991; Westerterp et al. 1996). The control group was generally given no special dietary instructions and followed their habitual diet (Gatenby et al. 1995; Siggaard et al. 1996). Some provided the control group with general nutrition advice on healthy eating at the start of the trial (Swinburn, et al. 1999; Sheppard et al. 1991). In one study, the control group was instructed to consume full fat food products and a minimum of 37 g of fat/day (Westerterp et al. 1996).

Dietary intake was measured using 4-day or 3-day diet records with one study also using a food frequency questionnaire at baseline (Sheppard et al. 1991). Some studies measured dietary intake before and after the intervention (Siggaard et al. 1996; Swinburn et al. 1999), whilst others included more than 4 dietary measures during the intervention (Gatenby et al. 1995; Westerterp et al. 1996). Dietary compliance was assessed by grocery receipts (Gatenby et al. 1995), average attendance rate at monthly meetings and percent completion of shorthand food diaries (Swinburn et al. 1999) but it was not measured in the other studies. Under-reporters were not identified in any study and all participants were included in the analysis.

Studies conducted in normal weight individuals suggest that reducing dietary fat intake to less than 30% E fat results in reduced energy intake and subsequent weight

loss of 1 to 2.5 kg (Gatenby et al. 1995; Siggaard et al. 1996; Sheppard et al. 1991). Reducing dietary fat intake from 35% to 33% E (decrease of 6 g fat/day) resulted in weight maintenance (Westerterp et al. 1996). Increasing % E fat from 35% to 40% E fat (increase of 30 g fat/day) in normal weight subjects resulted in increased energy intake and weight gain of 0.9 kg in women and 1.2 kg in men (Westerterp et al. 1996). Dietary fat reduction achieved greater weight loss (3 to 5 kg) in overweight individuals (Siggaard et al. 1996; Swinburn et al. 1999).

Meta-analyses of short-term low fat ad libitum intervention studies including both normal weight and overweight individuals have been conducted. A meta-analysis of 37 low fat dietary intervention studies in free-living subjects lasting more than 3 weeks reported a 0.28 kg decrease in body weight for every 1% decrease in energy as total fat (Yu-Poth et al. 1999). A review of 28 short term trials of the effect of dietary fat reduction on weight loss in obese individuals showed that a reduction of 10% E fat was associated with a reduction in weight of 16 g/day (Bray and Popkin 1998). A more restrictive meta-analysis of weight loss in 16 ad libitum low fat intervention trials lasting more than 2 months showed that for every 1% reduction in dietary fat, a weight loss of 0.37 kg (95% CI, 0.15 to – 0.60 kg/%) was achieved (Astrup et al. 2000). Astrup et al. (2000) reported a weighted difference in weight loss between intervention and control groups of 2.55 kg (95%CI, 1.5 to 3.5; P<0.001). Similarly, Yu-Poth et al. (1999) found that weight loss in the intervention groups was 2.79 kg larger than in the control group.

According to Astrup et al. (2000), the effect of ad libitum dietary fat reduction on weight loss is dose-dependent. However, Knopp et al. (1997) compared diets providing 28%, 26%, 22% and 18% E fat and found statistically significant mean reductions in body weight of 2 to 3 kg in each group after 12 months. Energy intake decreased statistically in all diets suggesting that the effect of dietary fat restriction on energy intake was not dose-dependent. Reducing dietary fat to 23% E fat did not achieve greater weight loss in long term (Sheppard et al. 1991). It is possible that the effect of ad libitum dietary fat reduction on energy intake and hence weight loss has a threshold effect with little further gain achieved below 27% E fat (Hill et al. 2000).

Since most of these studies were short term studies (<6 months) it is difficult to predict the long term effect of ad libitum dietary fat reduction on body weight (Willett, 1999a). Intervention studies assessing the long term effect of dietary fat reduction on risk of breast cancer in women suggest weight loss occurs in the first six months, with no further weight loss (Lee-Han et al. 1988; Boyd et al. 1990; Kasim et al. 1993). Overall, women following the low fat diet, consumed about 1050 kJ (250 kcal) less than the control group and lost about 1 to 2 kg over 12 months compared to the control group. Women who reduced dietary fat from 36.3% E to 17.6% E lost 3.4kg in 3 months, with no further weight gain reported after 1 year (Kazim et al. 1993). Although women lost more weight on the low fat diet, they regained most of the weight after 12 months, despite no significant dietary changes occurring over the 12 months of the intervention (Boyd et al. 1990). At the end of 2 years, the difference in weight loss between the intervention and control groups was only 1.8 kg compared to 2.8 kg after 6 months (Sheppard et al. 1991).

It is unclear whether absolute dietary fat intake (grams) or % E fat, is more predictive of weight change. Sheppard et al. (1991) reported that changes in % E fat were more

predictive of weight change than changes in total energy intake. Fat reduction of 1% E fat in the intervention group resulted in weight loss of 0.1 to 0.25 kg. However, Westerterp et al. (1996) reported that change in absolute fat intake explained 70% of the variance in fat mass compared to the fat to carbohydrate ratio which explained only 15%.

Results suggest there is a wide variation in the effect of dietary fat restriction on weight loss ranging from 11.4 to -0.77 kg (Astrup et al. 2000). The effect of dietary fat reduction on weight loss explained only 57% of the total variance (Yu-Poth et al. 1999).

Several studies confirm the significant positive correlation ( $r=0.52$ ;  $p<0.05$ ) between baseline BMI and weight loss reported by Astrup et al. (2000) (Siggard 1996; Bray and Popkin 1998). The separate effect of ad libitum fat reduction on body weight in normal weight and overweight individuals was not reported in the meta-analyses.

Factors other than dietary fat intake explain some of the weight change. Westerterp et al. (1996) compared the separate effect of dietary fat reduction and found that men were more sensitive to changes in dietary fat intake than women. Eating behaviour has been shown to influence the effect of fat reduction on weight change. Restrained eaters were less likely to gain weight on a high fat diet (40% energy from fat) than unrestrained eaters. A reduced fat diet (33% energy from fat) resulted in weight loss in restrained eaters, whereas body weight was maintained in unrestrained eaters (Westerterp-Plantenga et al. 1998). Other factors, such as physical activity and smoking cessation, may also have influenced weight change. Few studies measured and controlled for differences in physical activity, dietary compliance and smoking behaviour. Yu-Poth et al. (1999) reported significantly greater weight loss in diet intervention with exercise compared to those without exercise. Body weight decreased by 5.66kg in intervention groups with exercise and by 2.79kg in intervention groups without exercise (Yu-Poth et al. 1999). Few studies controlled adequately for these potential confounding effects.

In many studies, the control group did not receive dietary instruction of the same intensity as the intervention group. Only one study treated intervention and control groups equally (Westerterp et al. 1996). Furthermore, many of these studies included other behavioural and lifestyle changes, making it difficult to determine the independent effect of fat reduction on body weight. The Hawthorne effect describes the modest weight loss achieved during interventions, regardless of the dietary manipulation, as a result of the attention, monitoring and recording provided to the treatment group. In order to determine the independent effect of dietary fat manipulations on weight change, it is preferable to provide the control group with a similar level treatment intensity as the treatment group (Willett, 1999b).

These ad libitum studies suggest that fat restriction may offer a simple behavioural strategy for achieving energy restriction. It is therefore possible that carbohydrate restriction may equally restrict energy intake.

#### 5.4 Energy restriction compared to low fat ad libitum diets

Table 4 describes randomized controlled studies in which the effect of a low fat, ad libitum diet on weight loss is compared to energy restriction in obese subjects. An exercise and behavioural program was included in some studies (Harvey-Berino, 1998; Schlundt et al. 1993; Hammer et al. 1989). One study compared the effect of low fat ad libitum versus energy restriction on weight maintenance after an initial weight loss (Toubro and Astrup, 1997). Overall, the low fat ad libitum intervention group was instructed to reduce dietary fat intake to around 20 to 25 g fat/day. The energy restricted intervention group was required to reduce caloric intake to between 3360 kJ and 7800 kJ.

Dietary intake was measured using food records (Hammer et al. 1989; Toubro and Astrup, 1997; Harvey-Berino 1998; Schlundt et al. 1993), 24 hour recall (Jeffery 1995) and a food frequency questionnaire (Jeffery, 1995). Dietary intake was measured at baseline and post-intervention in some studies (Schlundt et al. 1993; Harvey-Berino, 1998), whilst others measured dietary intake more than 4 times during the intervention (Hammer et al. 1989; Jeffery 1995). Jeffery (1995) showed that dietary intakes differed depending on the dietary measurement tool used.

Dietary compliance was measured in some studies using a dietary adherence questionnaire (Harvey-Berino, 1998), completion of food diaries (Schlundt et al. 1993; Jeffery 1995; Hammer et al. 1989), personal interviews, attendance at treatment visits (Hammer et al. 1989; Jeffery 1995), and percent of days on which participants reported achieving their dietary goals (Jeffery 1995). Toubro and Astrup, (1997) did not report dietary intake.

Subjects in the low fat ad libitum intervention group generally did not achieve their dietary fat targets, consuming between 30 to 40 g fat/day (20-27% E fat), whereas subjects in the energy restriction groups generally achieved energy targets. Overall, there were small differences in dietary intake between fat and energy restricted groups. Energy restricted groups tended to reduce total energy intake by restricting both dietary fat and dietary carbohydrate. In fat restricted groups, dietary fat reduction accounted for most of the decrease in energy intake (Shah et al. 1994). Harvey-Berino, (1998) reported that carbohydrate intake accounted for 73% of the difference in energy reduction between the two groups.

Short term studies (16 to 20 weeks) reported significantly greater weight loss in energy restricted than in low fat interventions (Hammer et al. 1989; Schlundt et al. 1993; Harvey-Berino 1998). Men lost more weight than women and women seemed to lose more weight on the energy restricted diet than the low fat ad libitum diet (Schlundt et al. 1993). Energy restriction combined with an exercise program resulted in the most weight loss indicating the critical role of physical activity (Hammer et al. 1989).

Long term studies (>6months) show no significant differences in weight loss between energy and fat restriction since weight lost in the short term is regained (Jeffery 1995; Shah et al. 1994; Shah, 1996a). Changes in dietary fat intake over time was significantly correlated with weight change and predicted weight change better than energy restriction, even though fat restriction did not achieve weight loss in the long term (Jeffery, 1995; Shah, 1996a). Toubro and Astrup (1997) reported that weight

loss was less likely to be maintained on an energy restriction regime compared to a low fat ad libitum regime (RR=1.7 (95%CI, 0.8 to 3.7).

Results showed variation in weight loss despite similar compliance suggesting other factors influence weight loss (Hammer et al. 1989). Energy deficits could only explain 31% of the variability in weight loss. Inconsistency in results may be explained by the lack of control of confounders including physical activity and baseline body weight. Some studies reported no differences in physical activity between intervention groups without providing specific details (Schlundt et al. 1993; Harvey-Berino, 1998; Jeffery et al. 1995). Others did not discuss differences in physical activity between intervention groups (Toubro & Astrup, 1997). In some studies, there were significant differences in baseline body weight between intervention groups (Schlundt et al. 1993; Hammer et al. 1989). Preference for a dietary intervention may impact on weight loss. Shah et al. (1994) found that some subjects reported better quality of life measures and palatability on the low fat regime (Shah et al. 1994), whilst others preferred the energy restricted regime (Harvey-Berino, 1998). However, dietary compliance was generally poor in both types of dietary interventions, especially in the long term. As a result, there were small differences in dietary intake between intervention and control diets making it difficult to detect a significant difference in weight change between energy and fat restriction.

Considering these methodological limitations, the evidence suggests reducing dietary fat intake to around 25% E fat or 30 to 35 g fat/d is as effective as restricting energy intake to around 5040 kJ/day for achieving weight loss in obese women (Jeffery 1995). A (preliminary) systematic review found that low fat diets were as efficacious as other types of low-energy diets in promoting weight loss in overweight and obese individuals (Summerbell et al. 1998).

## **5.5 Low fat ad libitum compared to high monounsaturated fat ad libitum diets**

Several isoenergetic studies comparing the effect of a low fat, high carbohydrate diet with a high monounsaturated fat (MUFA) diet in people with diabetes suggest low fat diets are less advantageous than higher fat diets in terms of glycaemic control and lipid metabolism (Golay et al. 1996a; Noakes & Clifton 2000). However, studies in free living subjects suggest that reducing dietary fat intake from 41% to 31% E and increasing carbohydrate intake from 38% to 46% E over 6 months results in significant weight loss (2.7 kg) in obese insulin dependent women, but not in those who were not obese (de Bont et al. 1981). Several studies have therefore investigated the optimal diet for managing both body weight and glycaemic and lipid control in people with diabetes.

Randomized studies conducted in free-living overweight women with type 2 diabetes suggests no significant difference in weight loss between low fat, high carbohydrate (22% E fat; 52% E carbohydrate) and high MUFA (varying from 33% to 36% E fat; 40% E carbohydrate) diets over 3 months (Walker et al. 1995; Walker et al. 1999a). Weight loss was small (less than 1.5 kg) and not statistically different between groups (Walker et al. 1995; Walker et al. 1999a). However, there were significant differences in regional fat loss. Whilst loss of upper body fat was similar on both

diets, women on the high carbohydrate diet lost significantly more lower body fat ( $p=0.04$ ) (Walker et al. 1999a).

There were no significant differences in total energy intake between the high MUFA and low fat groups (Walker et al. 1999a). Although usual physical activity patterns were maintained, differences in physical activity levels were not considered in the analyses (Walker et al. 1999a). The diets differed significantly in the proportion of protein, fat and carbohydrate. The low fat diet was significantly higher in % E protein and % E carbohydrate whereas the high MUFA diet was significantly higher in % E from MUFA and PUFA. Dietary fibre intake on the high MUFA (25 g/day) was similar to that on the low fat diet (29 g/day) (Walker et al. 1999a). The MUFA diet was based on a Mediterranean eating style which included plenty of vegetables and legumes. Consequently, the energy density of the MUFA diet was comparable to that of the low fat ad libitum diet (Walker et al. 2000). This study suggests that dietary fat intakes of up to 36% E fat can be consumed as long as the diet is low in energy density.

Research is required to further investigate the effect of dietary composition on body fat distribution. Research is also required to determine whether similar effects are achieved using polyunsaturated fats and in people without diabetes.

## **5.6 Low fat, high carbohydrate ad libitum compared to low fat, high protein ad libitum diets**

Few studies have investigated the optimal relative proportions of dietary carbohydrate and protein in a low fat (30% E fat) ad libitum diet. Short term studies suggest that protein exerts a more powerful effect on satiety than carbohydrate (Blundell et al. 1993). Increasing the protein content of a hypocaloric diet may reduce loss of lean body mass (Durrant et al. 1980). A randomized trial was therefore conducted to determine whether increasing the protein content of a low fat diet would improve weight loss in overweight individuals.

The study was conducted in 65 overweight and obese men and women to investigate the effect on weight loss of replacing dietary carbohydrate with protein in a low fat (29% E fat) ad libitum diet using a validated laboratory shop system to supply diets. After six months, average weight loss was 5.0 kg (3.6 to 6.4 kg) with the low fat, high carbohydrate diet (30% E fat; 12% E protein; 59% E carbohydrate) and 8.7 kg (7.3 to 11.3 kg) with the low fat, high protein diet (30% E fat; 25% E protein; 45% E carbohydrate). Baseline weights were similar in all groups and no significant weight change occurred in the control group. There was a significant difference of 3.7 kg (1.3 to 6.2 kg;  $P=0.0002$ ) in weight loss between intervention groups, mainly due to body fat loss. After 6 months, more subjects had lost greater than 10 kg body weight in the high protein group (35%) than in the high carbohydrate group (9%). (Skov et al. 1999).

The total energy intake of those on the low fat, high protein diet (9.3 MJ/d) was significantly less than those on the low fat, high carbohydrate diet (11.2 MJ/d), despite the latter providing significantly more dietary fibre (22.8 g vs 18.6 g on average) (Skov et al. 1999). There were no significant differences in palatability or energy density with both diets providing 5.0 kJ/g. Dietary compliance was assessed using 24 hour urinary nitrogen excretion. The authors attributed the greater weight

loss in subjects on the high protein diet compared to the high carbohydrate diet, to a greater reduction in energy intake, possibly due to the effect of protein on satiety and partly due to the slightly higher thermogenic effect of protein (Skov et al. 1999).

This study suggests that dietary factors other than energy density and dietary fat affect energy intake. In general, protein intake in the intervention studies conducted to date provided around 17% energy from protein, showing little variation between studies and suggesting that the protein intake is generally constant. Due to concerns in relation to potential adverse effects of high protein intake, further research is recommended before these results can be extrapolated to public health recommendations. However, this study does highlight the complexity of factors which regulate energy intake.

### **5.7 Low fat, complex carbohydrate compared to low fat, simple carbohydrate diets**

Whilst current weight management guidelines emphasize replacing dietary fat with carbohydrate, there has been little research conducted to investigate the effect of reducing carbohydrate on body weight. Preliminary research suggests that there may be differences in the effect of type of carbohydrate on satiety (Holt and Brand-Miller, 1995) and energy intake (Raben et al. 1997). However, few studies have investigated the effect of different types of carbohydrate on body weight. Since many low fat products may include high levels of sugar, the effect of sucrose on body weight is of interest.

The effect of type of carbohydrate in a low fat ad libitum diet (<30% E fat) on body weight regulation was investigated in a European multi-centre trial (the Carbohydrate Ratio Management in European National diets or CARMEN trial) (Saris et al. 2000). Moderately obese adults (BMI between 26 and 35) were randomised to either a seasonal control group (no intervention to control for seasonal variation in body weight), or to one of three experimental diet groups: a control diet (typical of the average national intake, 37-38% E fat), low fat, high simple carbohydrate diet or a low fat, high complex carbohydrate diet. Food was supplied by a validated laboratory shop system. Subjects following the low fat, complex carbohydrate diet (n=83) lost 1.8 kg (P<0.001), 2.6 kg more than the control diet (n=77) and 1.9 kg more than the seasonal control group (n=80). Those in the low fat, simple carbohydrate diet (n=76) lost 0.9 kg (P<0.05), 1.7 kg more than the control group. Differences in weight loss between the low fat, simple carbohydrate and low fat, complex carbohydrate diets were not statistically significant. There were no significant differences in total, low density lipoprotein cholesterol (LDL) or HDL-cholesterol, glucose, insulin or leptin levels among the groups. Energy density on the low fat, simple carbohydrate diet (3.68 kJ/g) was significantly higher (P<0.05) than on the low fat, complex carbohydrate diet (3.36 kJ/g), but it was significantly (P<0.05) lower than on the control diet (3.83 kJ/g).

In a similar study, in which subjects from the CARMEN trial were included, one quarter of daily fat intake was replaced with complex or simple carbohydrate. Thirty-nine overweight individuals (22% drop-out rate) with metabolic syndrome were randomly assigned to one of three ad libitum diets for six months: control diet; low fat, high complex carbohydrate diet (LFCC), and a low fat high simple carbohydrate diet (LFSC) (Poppitt et al. 2002). A grocery store system supplied about 60% of predicted

energy requirements during the intervention. Dietary intake was estimated from 7-day and 3-day weighed-food records collected on 5 occasions during the study. Baseline dietary fat intake varied from 32 % E in the control to 29.8% E in the LFCC and 35.8% E in the LFSC. Mean dietary fat intake during the intervention was significantly lower in both LFCC (24.1% E) and LFSC (21.1% E) than the control (31% E) ( $P < 0.0001$ ). Complex carbohydrate was significantly higher in the LFCC (35.5% E) than in either the control (28.6% E) and LFSC (28.5% E) ( $P < 0.0001$ ). Simple carbohydrate intake was higher in the LFSC (28.9% E) than in the control (20.6% E) and LFCC (17.6% E). Although mean energy intake during the intervention was higher in the LFSC (9578 kJ/d) than the control (8022 kJ/d) and LFCC (8108 kJ/d), there was no significant difference in energy intake between groups. It is interesting to note that the LFSC group made the greatest reduction in dietary fat intake yet had the highest intake of energy. Physical activity levels (reported energy intake/predicted BMR) were slightly higher, but not significantly different in the LFSC than in the LFCC and control. There was evidence of underreporting of energy intake in all groups. Weight loss was greatest with the LFCC diet. Body weight loss from baseline was -4.25 kg ( $P < 0.01$ ) in the LFCC group, -0.28 kg in LFSC group and 1.03 kg (NS) in the control group. Weight loss was not accompanied by significant changes in abdominal obesity. Differences in weight loss between the three diets in this study cannot be explained by any differential in reported energy intake. There were no significant changes in LDL-cholesterol, whereas HDL cholesterol decreased over time in all three groups ( $P < 0.0001$ ). Triglyceride concentrations were higher in the LFSC than in the other two groups ( $P < 0.05$ ).

## 5.8 Summary of evidence from intervention studies

Evidence from intervention studies suggests that energy balance is the most important determinant of weight loss in overweight and obese individuals.

Randomized controlled trials in obese individuals reported no significant differences in short-term weight loss between energy-restricted diets (1000-1200 kcal) varying in fat content from 15% to 75% E fat (Powell et al. 1994; Lean et al. 1997; Golay et al. 1996a). An interim systematic review concluded that low-energy, low-fat diets are not more efficacious than low-energy diets which are not low in fat, in terms of weight loss in overweight or obese individuals (Summerbell et al. 1998).

Controlled trials suggest that low-fat ad libitum dietary interventions may reduce energy intake leading to short-term weight loss in overweight individuals (Swinburn et al. 1999; Siggaard et al. 1996). A meta-analysis of low-fat ad libitum dietary intervention studies, in which weight loss was not the primary aim of the majority of these studies, reported a weighted difference in weight loss between intervention and control groups of 2.55 kg (95% CI: 1.5-3.5;  $P < 0.0001$ ) (Astrup et al. 2000). In many studies, it was not possible to separate the effect of dietary fat from the behavioural aspects of the treatment diet. In particular, ad libitum approaches are also testing the use of fat restriction as a simple behavioural strategy for achieving energy restriction, the success of which is dependent on the eating patterns of the targeted population. Few studies separated the effect of dietary fat on weight loss from other confounding factors such as physical activity, other dietary factors and behavioural influences.

Randomized controlled trials suggest that there is a wide variation in short-term weight loss achieved in obese individuals on low-fat (30% E fat) ad libitum diets

(Skov et al. 1999; Saris et al. 2000). Differences in the protein content, and to a lesser extent, the type of carbohydrate in the low-fat ad libitum diet resulted in significant (3.7 kg 95% CI: 1.3-6.2 kg;  $P=0.0002$ ) and non-significant differences in weight loss, respectively. A randomised cross-over study found no significant difference in short-term weight loss in overweight women on a low-fat diet (22% E fat) compared to those on a high monounsaturated fat diet (36% E fat) (Walker et al. 1999a). It is possible that dietary fat intakes of up to 35% E fat can be consumed as long as the overall diet is low in energy density.

Reducing dietary fat intake to 25% E (30-35 g/day) appears to be as effective as restricting energy intake to 5040 kJ/day for achieving short term (6 months) weight loss in obese women (Summerbell et al. 1998). A randomized controlled trial suggests that short-term weight loss achieved with both low fat ad libitum dietary interventions (dietary fat reduced to 25% E fat or 30-35 g fat/day) and energy restricted diets (around 5040 kJ/day) in free-living obese individuals is not maintained in the long-term, after controlling for confounding factors (Jeffery et al. 1995). Other factors, such as physical activity, social support and preference for the dietary regime have been shown to also influence weight loss.

Few controlled trials have measured the independent effect of dietary fat reduction on weight maintenance in normal, overweight and obese individuals. A randomized controlled trial suggests energy intake is significantly decreased in normal weight men ( $P<0.01$ ) and women ( $P<0.0001$ ) when dietary fat intake is reduced from 35% E fat to 33% E fat, whereas energy intake is significantly increased in women only ( $P<0.01$ ) when dietary fat intake is increased from 35% E fat to 41.2% E fat (Westerterp et al. 1996). Body weight was significantly increased on the high fat diet (40% E or 122 g/day) in both men ( $P<0.001$ ) and women ( $P<0.01$ ) but remained unchanged on the low fat diet (33% E fat or 89 g fat/day). Eating behaviour may explain differences in the effect of dietary fat reduction on energy intake and consequent weight change in normal weight individuals (Westerterp-Plantenga et al. 1998).

The long-term effect of dietary fat reduction strategies on weight maintenance in normal weight and overweight individuals has not been demonstrated. It is therefore unclear whether dietary fat reduction prevents excess energy intake and hence helps to prevent the development or progression of overweight and obesity.

Some studies suggest that energy intake is reduced when the overall energy density of the diet is reduced to less than 5 kJ/g. Low fat ad libitum diets may help to reduce the energy density of the diet and in this way reduce energy intake. However, a low energy dense diet can be achieved with dietary fat intakes of up to 36% E. The relative effectiveness of dietary fat reduction compared to increasing the water and/or fibre content of foods on lowering the energy density of the diet is unclear. Furthermore, the long-term effectiveness of a low energy dense diet on weight loss or maintenance has not been demonstrated.

Physical activity is clearly critical in weight loss and weight maintenance. More weight loss is achieved when energy intake is reduced along with increased physical activity (Yu-Poth et al. 1999). Further research is required to determine the optimal combination of increased energy expenditure and reduced energy intake required for achieving weight loss and weight maintenance in normal weight, overweight and obese individuals.

## 6. Conclusions

The association between dietary fat and overweight and obesity, reported in cohort studies conducted in different population groups, was inconsistent. The findings of cohort studies were limited due to the quality of the studies, particularly with respect to the measurement of dietary fat intake. Few studies excluded under-reporters and dietary intake was generally measured only at baseline. Since dietary fat is highly correlated with energy, it is difficult to separate the effect of dietary fat from that of energy. Furthermore, few studies controlled for the confounding effect of physical activity. Overall, the effect of dietary fat intake on body weight was small and may be explained by measurement bias.

Evidence from experimental studies suggests that factors promoting excess energy intake, rather than the differential effect of macronutrients on energy expenditure and energy storage, are more likely to increase the risk of weight gain. Energy density appears to be a major determinant of energy intake. The energy density of the diet is mainly determined by the dietary fat, fibre and water contents of foods. It is possible that dietary fat may increase the energy density of the diet, facilitating excess energy intake and hence indirectly increase the risk of overweight and obesity. However, other factors may also influence energy intake including the palatability and the physical form of food, the amount (portion size) and volume of food consumed as well as behavioural and genetic factors.

There is therefore moderate evidence suggesting that dietary fat is not an independent risk factor for the development and progression of overweight and obesity.

Short-term (up to 6 months) studies indicate that energy balance is the most important determinant of weight loss in overweight and obese individuals. The macronutrient composition of low-energy diets does not influence short-term weight loss in overweight and obese individuals (Powell et al. 1994). Other reviews have also concluded that energy balance is the major determinant of weight loss (Freedman et al. 2001).

Low fat ad libitum studies suggest that reducing dietary fat may facilitate reduced energy intake resulting in modest short term weight loss (Astrup et al. 2000). However, measurement bias in many low fat ad libitum dietary intervention studies make it difficult to examine the independent effect of dietary fat reduction on weight loss. Studies comparing energy restricted diets (around 5040kJ/day) with low fat ad libitum diets (25% E or 30 - 35 g/day) suggest that they are equally effective in reducing energy intake and achieving short term (6 months) weight loss in overweight and obese individuals. However, weight loss is not maintained in the long term due to poor dietary compliance with both diets (Jeffery et al. 1995).

Intervention studies suggest that reducing the overall energy density of the diet to around 5kJ/g may facilitate reduced energy intake and hence weight loss (Skov et al. 1999; Saris et al. 2000; Walker et al. 1999a). A low energy dense diet may be achieved on a moderate dietary fat intake (around 35% E fat) by including plenty of vegetables (Walker et al. 1999a). Further research is required to determine the relative effectiveness of manipulating the dietary fat, fibre and water content of foods on reducing the energy density of the overall diet. Long-term studies are also

required to determine whether strategies for reducing the energy density of the diet are useful for achieving weight loss in overweight and obese individuals.

There was a wide variation in the amount of short-term weight loss achieved with ad libitum low fat diets suggesting that other factors also influence weight loss. Dietary factors such as the protein content of the diet have been shown to increase short-term weight loss in obese individuals. Non dietary factors which influence hunger, appetite and subsequent food intake include neurochemical, genetic, environmental and emotional factors and the hedonistic qualities of food (Freedman et al. 2001). Psychological issues are probably implicated in dietary compliance (Freedman et al. 2001). Social support has been shown to be an important determinant of weight loss and maintenance (Jeffery et al. 1984). Physical activity is also a very important determinant of weight loss. Combining reduced energy intake with increased physical activity had the greatest impact on weight loss in overweight and obese individuals (Hammer et al. 1989). Few studies controlled adequately for physical activity. Further research is required to determine effective strategies for combining dietary and physical activity interventions in weight management.

There is therefore moderate evidence suggesting dietary fat reduction alone, without energy restriction, will not achieve weight loss in overweight and obese individuals. According to the NIH (1998), reducing dietary fat alone, without reducing energy intake, is not sufficient for weight loss in overweight and obese individuals. In particular, replacing dietary fat with carbohydrate will not produce the energy deficit required to achieve weight loss. Several authors have recommended that low fat foods must be low in energy density to contribute to reduced energy intake (Salzman et al. 1997; Bray and Popkin, 1998; Levine, 2001; Roberts & Heyman, 2000).

Few controlled trials have measured the independent effect of dietary fat reduction on short-term weight maintenance in normal, overweight and obese individuals. The long-term effect of dietary fat reduction strategies on weight maintenance in normal weight and overweight individuals has not been demonstrated. High dietary fat intakes (around 40% E fat) may facilitate excess energy intake and hence weight gain. Reducing dietary fat intake to 33% E fat may prevent excess energy intake and assist in achieving weight maintenance (Westerterp et al. 1996). The effectiveness of dietary fat reduction strategies on weight maintenance has been shown to differ according to eating behaviour (Westerterp-Plantenga et al. 1998).

There is therefore little evidence to indicate whether dietary fat reduction alone will prevent weight gain in normal weight, overweight and obese individuals. Although dietary fat may increase the energy density of the diet, facilitating excess energy intake and hence weight gain, other dietary and non-dietary factors need to be considered. Further research is required to determine the long-term effectiveness of manipulating the energy density of the diet on weight maintenance. Research is also required to determine the relative effectiveness of reducing the energy density of the diet by manipulating the dietary fat, fibre and water content of foods. The usefulness of other dietary strategies for reducing total energy intake, such as increasing the protein content of the diet, reducing the intake of sugar-containing beverages and reducing portion sizes, requires further investigation. Eating patterns, in particular food variety and frequency of consumption of restaurant meals may also need to be considered. Physical activity is clearly critical for weight maintenance. In children,

physical activity was shown to be an important determinant of overweight and obesity. It is recommended weight management include both strategies to control energy intake and increase physical activity (NIH, 1998; WHO, 1998; NHMRC, 1997). Further research is required to determine the most effective public health strategies for achieving energy balance by combining both dietary and physical activity strategies.

Public health strategies for the prevention of overweight and obesity have encouraged strategies for reducing dietary fat intake (NHMRC, 1997). According to the 1995 National Nutrition Survey, Australians are consuming, on average, 32% E fat, 12.7% E SAFA and 4.9% PUFA (ABS 1997). The evidence reviewed suggests that dietary fat intake of 30 to 35% E do not seem to be associated with excess energy intake. Since dietary fat restriction may help to facilitate energy restriction, public health recommendations should ensure that dietary fat intake remain at less than 35% energy from fat.

Intake of total dietary fat and polyunsaturated fatty acids in the Australian diet has decreased since 1983, whilst intake of total energy and saturated fatty acids has increased (Cook et al. 2001). Behavioural research suggests that low fat dietary messages have been successfully communicated. Consumers associated dietary fat with overweight and generally believed that fat restriction was of benefit to health (Shanahan et al. 2000). They were aware of low fat dietary strategies, including restricting dietary fat intake by using less fat and oil in cooking, trimming meat fat and chicken skin, grilling instead of frying, using low fat food alternatives and avoiding intake of fried foods, takeaways, full cream dairy foods, cakes, biscuits and salad dressing. In particular, low fat dietary messages have had an impact on use of added fats and oils. A comparison of nutrition surveys in 1983 and 1995 suggest that consumption of fats and oils has decreased (Cook et al. 2001). Anecdotal evidence from the food industry is consistent with a decreasing use of added fats, particularly margarine (Bill Shrapnel, consultant to Unilever Australia, personal communication 2001).

Dietary modelling, based on 24-hour recalls of adults in the 1995 National Nutrition Survey, indicated that lowering intake of added fats and oils to less than 10g/day made it difficult to achieve the NHFA's policy on dietary fat with respect to the ALA and PUFA recommendations (Cobiac et al. 2000; Droulez et al. in review (a)). Public health messages which do not consider type of fat, risk further reducing intake of PUFA without affecting intake of SAFA in the Australian diet. Dietary modelling indicates that it is possible to include 25g/day of added unsaturated fats and oils in an energy restricted (6000 kJ) diet.

The emphasis of food-based recommendations for the prevention of heart disease is on reducing intake of foods high in saturated fatty acids, particularly full fat dairy products, takeaway meals, pastries, snacks and cakes. Dietary modelling indicates that reducing intake of saturated fatty acids tends to also reduce total dietary fat intake to less than 35% E (Droulez et al. in review (b)). Furthermore, since many foods high in saturated fatty acids are also high in energy density, restricting their intake also facilitates a reduction in energy intake. The NHFA's food-based recommendations are based on an eating pattern which includes a higher proportion of vegetables, fruit and cereals and regular intake of fish, legumes, nuts/seeds,

unsaturated fats and oils, low fat dairy products and lean meat. This eating pattern, which has been shown to be protective against heart disease, encourages intake of low energy dense foods such as fish, legumes, vegetables and grain-based foods and limits intake of energy dense foods such as takeaway meals, snacks, cakes and biscuits (Hu et al. 1999). Food-based recommendations developed for the prevention of CHD therefore may also provide a useful guide to lowering the energy density of the diet and maintaining the total dietary fat intake of the Australian diet to less than 35% E fat.

Since a general low fat dietary message may prevent achievement of the NHFA policy on dietary fat for the prevention of heart disease, it is recommended that the emphasis of public health nutrition strategies be placed on reducing intake of SAFA.

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**Table 1: Cohort Studies investigating the relationship between dietary fat intake and weight change**

	Sample size (% follow-up)	Age	Baseline weight (BMI)	Follow-up	Dietary Intake (Diet method)	Weight change	Confounders considered	Association between % E fat and obesity
Rissanen et al. 1991	Finnish 6165 F 6504 M (NA)	25-64 yrs	Females: 25.9±4.4 Males: 25.5±3.3	5.7 yrs	NA (Diet history in 1289 F and 1382 M at baseline)	Females: 0.06 ±4.95kg;  Males: 0.6±4.73kg	education, energy intake, smoking, alcohol, coffee consumption, leisure physical activity, health status, parity, gender	Positive association in Females only RR=1.7 (1.1-2.7)
Colditz et al. 1990	American 31940 F (82%)	30-55 yrs	81.5% with BMI<24.9	8 yrs	NA (FFQ)	1.9±4.6kg in first 4 yrs  1.6±4.7kg in next 4 yrs	Age, total calorie intake and other nutrients, baseline BMI but not physical activity	Positive association between fat (g/day) and prior weight change but not subsequent weight change
Klesges et al. 1992	American 152 F 142 M (70.5%)	33.07 ± 4.15 years in F 34.78 ± 4.73 years in M	Females: 24.83 ± 4.96 Males: 27.8 ± 4.32)	3 yrs	<b>% E fat:</b> M:35.97±5.4  F: 36.80±6.12 (FFQ annually)	Females: 1.37±5.89 kg  Males: 0.265 ±4.546 kg	Age, sports activity, work activity, leisure activity, energy intake, smoking, alcohol use, smoking, familial risk, pregnancy status, gender	Positive association in men and women (P=0.0010)
Paeratakul et al. 1998	Chinese 3484 M and F (74.5%)	20-45 yrs	mean = 21.5kg/m <sup>2</sup>	2 yrs	<b>% E fat:</b> M: 19.8(0.25) F: 19.9 (0.24)  (24-hr recall over 3 consecutive days)	Change in BMI: M: +0.2 (1.6) F: +0.2 (1.8)	Reported physical activity level at occupation, smoking, socio- economic, age, energy intake, gender	Positive association in men (P=0.0001) but not in women
Kant et al. 1995	American 4 567 F 2 580 M (68.5%)	25 – 74 yrs	Males: 25.9±0.1 Females: 25.2±0.1	10.6 yrs	<b>%E fat:</b> F: 25.6 – 47.2 M: 26.2 – 47.9	Males: 2.11kg ±0.2  Females: 2.49±0.2	Baseline energy intake, physical activity level, baseline BMI, race, education, age, smoking status, morbidity, alcohol intake, and special diet status, gender	No association in men and women Positive association in men without any morbidity at baseline (P=0.05) Inverse association in women aged <50 yrs (P=0.04)
Jorgensen et al. 1995	Danish 1008 F 1001M (87%)	30 – 60 yrs	Ht and Wt measured  Baseline data NA	5 yrs	NA (7 day food records)	39.8% increased BMI	Age, baseline BMI, gender, familial predisposition of obesity, education level, social status, baseline BMI, total energy intake, alcohol and smoking habits	No association with %E fat

**Table 1 continued**

	Sample size	Age	Baseline weight	Follow-up	Dietary Intake	Weight change	Confounders considered	Association between fat and obesity
Heitmann et al. 1995	Swedish obese women with obese parents (n=56) and lean parents (n=53) Lean women with obese parents (n=87) and lean parents (112) (89%)	38-60 yrs	29.6±4.0 28.0 ±2.7 22.4 ±1.7 21.9± 1.9	6 yrs	% E fat: 38± 5% 38± 5% 38± 6% 39± 5%  (Diet history at baseline)	Δ BMI: 0.5 ± 2.5 0.3 ± 2.2 0.9 ± 1.4 0.8 ± 1.4	Energy intake, smoking, physical activity, menopausal status	Positive association in genetically predisposed women (P=0.002)
Lissner et al. 1997	Swedish 361 F	38-60 yrs	<b>Low fat consumers</b> (<38.5% E fat) BMI=24.6(±4.1)  <b>High fat consumers</b> (>38.5% E fat) BMI=24.1(±4.1)	6 yrs	NA (Diet history)	<b>Low fat, sedentary :</b> -0.59kg <b>High fat, sedentary:</b> consumers: +2.24kg (P=0.06)	Baseline BMI, smoking, age and total energy	Positive association in sedentary women only

**Table 2: The effect on weight loss of hypocaloric diets (1000 – 1200 kcal) varying in diet composition but not energy content**

<b>Study</b>	<b>Sample</b>	<b>Time</b>	<b>% E Fat</b>	<b>% E CHO</b>	<b>Weight loss</b>
Powell et al. 1994	35 free-living obese women	12 wk	15% 20% 28% 34%	65% 58% 50% 43%	4.8kg 7.3kg 6.0kg 6.5kg (NS)
Golay et al. 1996b	68 obese outpatients	12 wk	26% (34g) 45% (57g)	45% (132g) 25% (70g)	8.6kg 10.2kg (NS)
Golay et al. 1996a	43 obese men and women in-patients	6 wk	26% (30g) 53% (60g)	45% (115g) 15% (37g)	7kg 8.3kg (NS)
Alford et al. 1990	35 free-living sedentary over-weight women	10 wk	75% 45% 25%	NA	4.8kg 5.6kg 6.4kg (NS)
Lean et al. 1997	110 obese women	6 mnths	58% 35%	20% 35%	5.6kg 6.8kg (NS)

**Table 3: Low or reduced fat ad libitum intervention diets compared to usual diets in free-living normal weight and overweight subjects**

Study	Sample	Time	% E Fat	Energy intake	Weight change
<b>Gatenby et al. 1995</b>	Men and women aged 40 years (average) BMI: <b>I = 24.1</b> (0.9) (n=15) <b>C = 26</b> (1.2) (n=14)	6 wks	<b>Baseline:</b> 38% (90g)  <b>I: 30.4%</b> (63g)  <b>C: 39.4%</b> (81g)	2156kcal  1939kcal  1887kcal	-1.1kg  +0.3kg (P<0.001)
<b>Siggaard et al. 1996</b>	Mainly men aged 32 to 47 yrs BMI: <b>I = 28.4 ±0.7</b> (n=50) <b>C = 27 ±1.0</b> (n=16)	12 wks  F/U: 24 wks 52 wks	<b>Baseline:</b> 39% (84g) <b>I: BMI&gt;25:</b> 28% (54g) <b>BMI&lt;25:</b> 28% (60g) <b>C: 37%</b> (81g)	8.2MJ  <b>BMI&gt;25:</b> 7.5MJ  <b>BMI&lt;25:</b> 8.0MJ  <b>Control:</b> 8.4MJ	<b>BMI&gt;25:</b> -5.2kg  <b>BMI&lt;25:</b> -2.5kg  <b>Control:</b> -0.8kg (P<0.001)
<b>Westerterp et al. 1996</b>	Men and women aged 19 to 55 yrs  <b>BMI:</b> <b>Reduced Fat:</b> 24.8 ±2.1 (n=116) <b>Full Fat:</b> 25.0 ± 2.1 (n=101)	6 mths	<b>Baseline:</b> 35% (95g)  <b>RF:</b> 33.1% (89g)  <b>FF:</b> 41.2% (122g)	10.1  10.1MJ  11.1MJ	<b>RF:</b> F: +0.6kg M:+0.1kg  <b>FF:</b> F: +0.9kg P<0.01 M:+1.2kg P<0.001
<b>Swinburn et al. 1999</b>	men and 30 women aged 45-60 yrs  BMI: <b>I = 29.7±5.2</b> (n=49) <b>C = 27.8±4.3</b> (n = 61)	1 yr	<b>Baseline:</b> 35% (86g)  <b>I: 25.9%</b> (52g)  <b>C: 33.8%</b> (90g)	2195kcal  1832kcal  2307kcal	-3.1kg  +0.4kg (P<0.001)
<b>Sheppard et al. 1991</b>	women aged 45 – 69 yrs  BMI: <b>I = 26 ± 0.4</b> (n=171) <b>C = 25 ± 0.4</b> (n=105)	2 yrs	<b>Baseline:</b> 39% (77g)  <b>I: 22.8%</b> (34g)  <b>C: 36.5%</b> (66g)	7293kcal  5640kJ  6748kJ	-1.9kg  -0.1kg

**Table 4: Randomized controlled trials in free-living subjects comparing the effect of low fat ad libitum interventions to energy restriction on weight loss**

Study	Sample	Time	% E Fat	Energy intake	Weight loss
<b>Hammer et al. 1989</b>	27 obese women aged 25 to 42 yrs	16 wks	<b>LF:</b> 23% (37g)  <b>ER:</b> 24% (22g)	6090kJ  3258kJ	<b>LF:</b> 5.9kg [6.72kg (+ Ex)]  <b>ER:</b> 9.4kg [13.1kg (+ Ex) (P<0.05)]
<b>Schlundt et al. 1993</b>	49 obese men and women aged 27 to 56 yrs	16 to 20 wks  Follow up 9 to 12 mnths	<b>LF:</b> 19% (30g)  <b>ER:</b> 20% (28g)	5969kJ  5292kJ	<b>LF:</b> 8kg (M) 3.9kg (F)  <b>ER:</b> 11.8kg (M) 8.2kg (F) (p<0.001) <b>Follow-up (mean):</b> LF:2.6kg ER:5.5kg
<b>Harvey-Berino, 1998</b>	15 obese men and 65 women aged 25 to 45 yrs	6 mnths	<b>LF:</b> 21% (39g)  <b>ER:</b> 27% (45g)	6902kJ  6180kJ	<b>LF:</b> 5.2kg  <b>ER:</b> 11.8kg (p<0.0001)
<b>Jeffery et al. 1995</b>	74 obese women aged 25 to 45 yrs	6 mnths  12 mnths  18 mnths	<b>LF</b> 24% (30g) <b>ER</b> 32% (37g) <b>LF:</b> 27% (33g) <b>ER:</b> 34% (41g) <b>LF:</b> 26% (34g) <b>ER:</b> 33% (43g)	4725kJ  4364kJ  4670kJ  4633kJ  5036kJ  4960kJ	4.6kg  3.7kg  2.1kg  0.7kg  +0.4kg  +1.8kg (NS)
<b>Toubro and Astrup, 1997</b>	2 obese men and 35 women aged 39 to 45 yrs	6 mnths  12 mnths  24 mnths	<b>LF:</b> 20 to 25%  <b>ER:</b> not reported	<b>LF:</b> not reported  <b>ER:</b> <b>7.8MJ</b>	2.3kg +0.5kg  +0.3kg +4.1kg  +5.4kg +11.3kg (P=0.08)

ER= energy restriction  
LF = low fat ad libitum  
Ex = exercise