Dietary fat intake does affect obesity![1–3]

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See corresponding editorial on page 1149.

ABSTRACT There is a difference of opinion about whether the percentage of dietary fat plays an important role in the rising prevalence of obesity. Evidence from epidemiologic studies, from controlled trials, and from epidemiologic and ecologic analyses provides strong evidence that dietary fat plays a role in the development and treatment of obesity. A reduction in fat intake reduces the gap between total energy intake and total energy expenditure and thus is an effective strategy for reducing the present epidemic of obesity worldwide. A review of the results from 28 clinical trials that studied the effects of a reduction in the amount of energy from fat in the diet showed that a reduction of 10% in the proportion of energy from fat was associated with a reduction in weight of 16 g/d. We thus conclude that dietary fat plays a role in the development of obesity. To reduce the prevalence of obesity, there must be an increase in energy expenditure, a reduction in total energy intake, or both. This goal can be facilitated by reducing the amount of fat in the diet. *Am J Clin Nutr* 1998;68:1157–73.

KEY WORDS Dietary fat, energy intake, obesity, humans, low-fat diets, low-energy diets, review

INTRODUCTION

Two related questions about the role of dietary fat intake and the development of obesity are being asked by the health community: *1*) Can we prevent obesity or slow the rapid increase in its prevalence by preventing the progression toward higher-fat diets in low-income countries? *2*) Can a reduction in energy density in the diet through a decrease in total fat intake contribute to the reduction in energy intake and the very high prevalence of obesity in countries with high intakes of dietary fat?

A recent review argued provocatively that "Diets high in fat do not appear to be the primary cause of the high prevalence of excess body fat in our society, and reductions in [dietary] fat will not be a solution" (1). Willett's argument is built around both epidemiologic evidence that shows a poor association between total fat intake and obesity, and an assessment of published clinical trials that focused on changes in the fat composition of the diet.

A major argument for the hypothesis that there is no relation between dietary fat and the development of obesity is that the epidemic of obesity is occurring in affluent countries at a time when intakes of dietary fat are decreasing. This reasoning is open to the equally plausible counterarguments that if dietary fat intakes were increasing in these countries, the prevalence of obesity might increase more rapidly, and that fat intakes may have increased in those segments of society in whom the prevalence focus on dietary fat intake may have been overemphasized at the expense of total energy intake. This is a critical point when it comes to placing the role of dietary fat intake into its proper context. Total energy balance is what matters most and the focus on dietary fat consumption must be seen through its effects on total energy intake. We are convinced from our review of the literature that if people eat more fat they consume more energy. This is the result of both passive overconsumption and the low thermic effect of fat. Diets with a low energy density are thus associated with greater satiety. In addition, we believe that if people eat less fat they will on average consume less energy.

We present some important biological reasons for why fat must be considered a crucial part of the energy balance equation. Without an understanding of the crucial role that fat intake plays in the etiology of obesity as well as in the design and implementation of its treatment, the likelihood of preventing a further worsening of the obesity epidemic will be hindered. In effect, we might lose an important weapon in what is now a limited arsenal of tools for the prevention and treatment of obesity. At the same time it is important to recognize that decreasing physical activity at work and leisure also plays an important role in the genesis of obesity. The first section of this paper summarizes the major argument against the primary role of dietary fat in the development of obesity (1) and is followed by a discussion of the role of low-fat diets in preventing obesity and the role of dietary fat intake in reversing obesity.

THE ARGUMENT THAT DIETARY FAT IS NOT IMPORTANT IN THE DEVELOPMENT OF OBESITY

There are 3 parts to the argument that dietary fat is not important in the development of obesity: *1*) the prevalence of obesity

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has increased in the United States despite reductions in dietary fat, *2*) other ecologic studies have found no relations between dietary fat intake and body fatness, and *3*) a reduction in dietary fat has little effect on the reduction in body weight.

Fat intake in the United States has begun to decline; however, rates of obesity have continued to increase. Thus, one can question the role of dietary fat intake in the epidemic of obesity. This decline in the proportion of energy from fat is small, only 3–5%, and would not necessarily be associated with a decline in body weight based on the clinical evidence reviewed later. We are apprehensive about drawing any conclusions with respect to these national trends because obesity is caused by many underlying biological factors. In addition, there is the potential that the emphasis on reductions in dietary fat has led consumers to underreport consumption of higher-fat foods and fat added during food preparation. Similar arguments were made related to the role of the decline in breast-feeding at a time when infant mortality was declining in low-income countries. The argument was that if breast-feeding affected infant mortality, then infant mortality would not have declined. In reality, infant mortality results from many underlying biological factors, as does obesity. If breast-feeding behavior had stayed steady or increased, infant mortality rates would have declined more. Clearly the same argument can be made for the role of dietary fat in the epidemic of obesity.

Other ecologic and population data can also be interpreted to show that dietary fat does not play a primary role in the epidemic of obesity. Studies in Europe that used the median body mass index (BMI; in kg/m^2) as a measure of obesity and national food disappearance data found no association between BMI and dietary fat as a proportion of energy (1). However, we view the use of median BMI values as an inadequate way to test the hypothesis that dietary fat is related to obesity. In addition, it is useful to look at a larger set of countries with nationally representative data.

The Chinese data collected in 1983 are a poor example of correlational relations between dietary fat and body weight because the prevalence of obesity was limited at the time and fat intakes then were lower than they are currently (2, 3). This study consisted of dietary data that reflected total household intakes for clusters of 30 rural families in each of the 65 rural Chinese counties selected for an ecologic analysis of cancer. Correlations between the proportion of fat in the diet and mean body weight ($r = 0.10$, $P > 0.05$) and between total fat intake and mean body weight ($r = 0.21$, $P > 0.05$) were low (3). In this review, we provide recent nationwide, longitudinal data from China that relate individual dietary changes over time to obesity to show that increased dietary fat intake is an important cause of variance in the development of obesity in Chinese adolescents and adults.

A review of several randomized clinical trials that addressed the role of reductions in dietary fat in lowering body weight suggests that the associated weight loss is modest. Long-term trials are few and most did not focus on obesity but rather on cardiovascular disease or cancer. In these long-term trials, it is often impossible to separate the effect of fat reduction from that of a reduction in total energy intake. We argue that there is little potential for weight change from dietary interventions that focus on reductions in dietary fat but no reductions in energy intake. Changes in fat intake are an important pathway to changes in total energy intake. Fat reduction must be accompanied by an increase in fiber and carbohydrates to be effective. To argue otherwise would be contrary to current knowledge. There is also little evidence that long-term dietary changes can be maintained because there have been few studies of the relation between maintenance of weight loss and behavioral changes.

We argue that there is evidence for an important role for dietary fat in preventing the rising prevalence of obesity and in treating currently existing obesity. We feel that changes in fat intake are an important means of reducing energy density and increasing the intake of other important foods and food constituents. To prevent or reduce obesity one must focus on both sides of the energy balance equation and consider total energy intake and its food components along with physical activity. For this review, articles were chosen based on an exhaustive search of the literature focusing on dietary interventions that manipulated fat intake. That there are no clear-cut, long-term, large-scale successful interventions to reduce obesity does not mean that dietary fat intake does not play a role in the development and reduction of obesity. We do not accept the conclusion that consumption of 18% of energy from fat is no different from the consumption of 40% of energy from fat in terms of the effect on body fatness, as implied elsewhere (1).

INCREASING DIETARY FAT ACCELERATES THE DEVELOPMENT OF OBESITY

Obesity is characterized by the accumulation of excess body fat. The body can adjust the mix of metabolic fuels it oxidizes so that alcohol, carbohydrate, and protein intakes are tightly regulated. In effect, the body can achieve carbohydrate and protein balances quickly. However, the body has a poor autoregulatory system for fat and an almost unlimited ability to store fat. Although positive energy balance results in obesity, fat intake is an important contributor to energy balance. There are several important ways in which fat intake and the consumption of high-fat diets play a significant role in the development of obesity. We begin with the presentation of experimental animal and clinical human research and then proceed to discuss controlled trials and epidemiologic issues.

Experimental data

Obesity in animals eating low-fat diets

As a rule, experimental animals eating low-fat diets do not become obese. The major exceptions are animals with genetic forms of obesity, animals with neuroendocrine disorders, and animals treated with drugs or peptides. These exceptions underline the fact that the development of experimental obesity while consuming a low-fat diet is the exception (4).

Animals eating high-fat diets

Development of obesity in animals eating high-fat diets is the expected outcome (4, 5). Whether animals become obese or are resistant to obesity when eating high-fat diets has strong genetic components. From an epidemiologic perspective, a high-fat diet can be viewed as the agent that acts on the susceptible host animal to produce the noninfectious disease obesity. Some strains of mice and rats are exquisitely susceptible to developing obesity when eating high-fat diets or high-fat, high-carbohydrate diets. Other strains of mice (SWR) and rats (S5B/Pl) are resistant to developing obesity when eating either high- or low-fat diets. A similar, differential responsiveness in humans is noted below.

Clinical data

It is the slow but continual overconsumption of dietary energy relative to daily needs that leads to obesity. An increase in dietary fat increases this tendency to overconsume. When dietary fat intakes increase, the body reacts in 1 of 2 ways to maintain energy balance. First, the extra fat in the diet can be oxidized by the body for its energy needs. Alternatively, the increased dietary fat can be sensed by the body in such a way that subsequent intake of high-fat foods is reduced; thus, energy balance is maintained. The evidence reviewed below argues that periodic exposure to a high-fat meal, particularly when hunger is high, may be sufficient to lead to overconsumption of energy from fat that is not compensated for by reduced energy intake (6).

Addition of fat to a meal might increase fat oxidation and thus metabolize the extra fat. This possibility has been tested in several ways. The conclusion from these experiments is that when fat is added to a meal, there is no increase in fat oxidation (7, 8). In contrast with the data on fat, there is a substantial body of data showing that addition of carbohydrate to the diet will increase carbohydrate oxidation.

Because fat intake does not stimulate fat oxidation, maintenance of energy balance during consumption of a high-fat diet requires a reduction in fat intake. Several approaches have been used to examine the effect of fat on satiety and on subsequent food intake. One question requiring an answer is whether fat and carbohydrate have different satiety values. That is, does the intake of a similar amount of energy from fat or as carbohydrate reduce appetite and food intake to a similar degree? In trials with pure nutrients, fat and carbohydrate in equienergetic amounts produced similar suppressions of food intake 15 min later, but fat was more effective 3.5 h later (9). When fat is part of the food in the diet, suppression of future food intake depends on the composition of the food, postingestional signals, and the cognitive response to these foods. Another question is whether the degree of reduction in food intake after ingestion of meals high in fat is similar to that after ingestion of meals high in carbohydrate. Preloads high in carbohydrate reduce food intake proportionally 30 min after the meal, but the degree of compensatory reduction declines as the interval between the preload and the test meal lengthens (10). In laboratory settings, healthy men compensate at subsequent meals for reductions in energy in experimentally manipulated meals. However, there was no compensation for variations in macronutrients (11, 12). Consumption of lunches providing low or high energy intakes, in which each energy combination contained sucrose or sucrose and fat, showed that individuals eating the high-sucrose, high-fat combination, particularly the lunch providing the high energy intake, were most likely to overconsume at the subsequent meal (13, 14).

The effectiveness of compensating for ingested energy by reducing the subsequent intake of energy in diets high in fat or carbohydrate varies between men and women and between individuals who consciously restrain food intake. In healthy, young men, the compensation for preloads is accurate (15). In women and men who are restrained eaters as well as in overweight men and women, the compensation for preloads is less adequate than in unrestrained eaters, which may lead to passive overconsumption of energy (15–18). There is limited understanding of the mechanisms that link energy density with total energy intake. The ability to reduce food intake appropriately to compensate for food eaten earlier is impaired when the food choices are high in fat (19, 20) and when they are high in both sugar and fat (14, 16). These findings point to one reason why sweet, high-fat foods are problematic for obesity. People tend to overconsume them rather than compensate for them.

The adaptation to a high-fat or low-fat, isoenergetic diet has been studied in several experiments by measuring changes in body composition or changes in metabolism in a respiratory chamber. The effect of a transition from a low-fat to a high-fat diet or vice versa in individuals living in a respiratory chamber was studied by Schrauwen et al (21) and by Hill et al (22). Schrauwen et al (21) reported that it takes between 4 and 9 d for the transition to asymptote at the new respiratory quotient, which is used as an index of fat intake. Careful overfeeding and underfeeding to achieve a new steady state has shown a shift in energy expenditure. In a study by Leibel et al (23), when the weight of obese volunteers plateaued at 10% above their baseline weight after they overate, energy expenditure increased; when their weight decreased to 10% or 20% below their baseline weight, energy expenditure decreased.

Most of the metabolic studies of overfeeding have used mixed diets, but a few compared high- and low-fat diets. Because carbohydrate stores are limited, overfeeding results first in glycogen stores being filled and next, to the extent that there is excess dietary protein, the protein stores. Beyond this point, overfeeding results in metabolism of the available carbohydrate with any excess being converted to fat. In most metabolic studies, subjects were in positive energy balance because respiratory chambers restrict energy expenditure, and thus energy expenditure has been difficult to match accurately to intake. Some overfeeding studies that compared high-fat and high-carbohydrate diets are summarized in **Table 1**. These data argue that metabolic adaptations to changes in fat in the diet are slow. If there is a genetic predisposition to fat storage, a high-fat diet may enhance the likelihood of obesity.

Postobese individuals usually regain weight and thus they are good candidates for studying what forces drive weight upward because obesity may modify the metabolic response to fat. Metabolic expenditure, as evaluated by a meta-analysis of published studies, decreased slightly after subjects had stabilized at a lower body weight (27). A similar reduction in total daily energy expenditure was observed in nonhuman primates that had stable weights for 10 y (28). More important for the present review is the inability of postobese patients to metabolize fat. In subjects that consumed meals containing 20%, 30%, and 50% of energy from fat, Astrup et al (26) found that postobese subjects had a smaller rise in energy expenditure with the 50%-fat meal than never-obese control subjects who were matched by sex, age, height, body weight, and body composition. A defect in the ability to oxidize fat by formerly obese individuals and by obese individuals overfed fat while in a metabolic chamber (29) suggests that consistent consumption of a diet higher in fat than in carbohydrate, which occurs when anything other than low-fat meals are eaten, results in the gradual accumulation of fat until fat stores have expanded sufficiently to bring fat and carbohydrate intakes back into balance.

Ecologic analysis

Ecologic studies use large populations and statistical models to examine various relations. This most basic type of epidemiologic analysis is useful for raising hypotheses but has many weaknesses, including failure to control for various confounders and an inability to truly relate individual levels to the population

TABLE 1
Studies on nutrient balance in nonobese, obese, and postobese subjects

Reference and subjects	Design	Comments
Abbott et al (24)		
Nonobese (n = 27M, 27F)	Metabolic chamber for 24 h	Energy balance related to fat balance; carbohydrate and protein oxidized
Flatt et al (7)		
Nonobese (n = 7M)	Meals with and without 50 g margarine; fat oxidation measured over 9 h with a ventilated-hood system	Additional fat intake did not increase fat oxidation
Thomas et al (25)		
Nonobese (n = 6M, 5F) and obese (n = 5M, 5F)	Low-fat, high-carbohydrate diet High-fat, low-carbohydrate diet ad libitum for 1 wk	Energy intake higher with the high-fat than with the high-carbohydrate diet; oxidation = carbohydrate intake; nonobese subjects showed relation between high fat intake and oxidation, but obese subjects did not
Astrup et al (26)		
Postobese (n = 9F) and never obese (n = 9F)	Varied fat and carbohydrate; 20%, 30%, or 50% of energy from fat	Postobese subjects had a smaller rise in fat oxidation
Hill et al (22)		
Nonobese (n = 3M, 5F)	Randomized crossover; 60% of energy as carbohydrate or fat; calorimetry on days 3 and 7	Isoenergetic substitution did not change energy expenditure but rapidly changed substrate oxidation

level. To date, the ecologic research that relates dietary fat to obesity has been based on the use of median weight or BMI (30, 31). We used a large set of nationally representative surveys that provided adult weight and height data. In addition to surveys conducted in several countries (Russia, Kyrgyzstan, and China) by one of us, we used data from published surveys conducted in all regions of the world. We selected all countries for which large, nationally representative samples of adults were available (30, 32) and also included data representative of a region for 2 African (South Africa and the Congo) and 2 Asian (India and Thailand) countries to allow more countries to be represented but with minimal data points. For adults, the cutoff we used to delineate overweight was a BMI $\geq$25.0 (33). The proportion of energy from fat comes from the Food and Agriculture Organization of the United Nations/Food and Agriculture Statistics database. These statistics are from national food balance data and are derived from data for the year preceding the date the obesity data were collected.

Data documenting the prevalence of BMI $\geq$25 were available for 20 countries (**Figure 1)**. An ordinary least-squares regression that weighted each country with its population was used to relate the BMI to the proportion of dietary energy from fat. The regression coefficient of 2.53 ($P < 0.001$, adjusted $R^2 = 0.78$) indicated a relation between the increase in the proportion of dietary energy from fat and the prevalence of overweight in a country in the sample available to us. There was a large significant, positive association between dietary fat consumption and the proportion of the population who was overweight. Note the rapid shift from the countries with low overweight and low dietary fat consumption such as India, China, and the Philippines, to the countries with moderate overweight and moderate dietary fat consumption, such as Brazil, Cuba, Saudia Arabia, and Tunisia. Clearly, some countries such as Russia and Saudia Arabia were outliers because they had much higher levels of overweight than expected on the basis of the percentage of energy from fat consumed by the population. Other countries, such as Malaysia and the Congo, had much lower levels of overweight than expected on the basis of the percentage of energy from fat consumed by the population. Nevertheless, except for the Congo, there was a marked increase in overweight in countries with higher fat intakes. These results support the potential role of reducing dietary fat intake as a means of preventing increases in the level of obesity in low- and middle-income countries.

Consistent with the results in Figure 1 are results from research on the nutrition transition in China, Brazil, South Africa, and other countries (2, 34, 35). In each case, at a time when fat intake was low (eg, China's food supply did not provide it), the level of obesity was very low. Increases in the proportion of energy from fat consumed have been consistent with increases in obesity in each country. Migration studies have a special place in epidemiology and have been most important in implicating several lifestyle components in the etiology of various cancers and cardiovascular disease. A recent reanalysis of the famous Ni-Hon-San migration study of Japan examined the role of diet in the etiology of obesity (36); 8006 Japanese men living in Honolulu were compared with 2183 men living in Hiroshima and Nagasaki. Mean BMI and subscapular skinfold thickness were both greater in the men living in Honolulu. In addition, more of the men living in Honolulu had a BMI >27.8 [a measure of obesity based on the National Center for Health Statistics 85th percentile in 20–29-y olds from the 1976–1980 survey (37)]. The mean energy intake in men aged 45–64 y was only slightly greater in Honolulu; however, the percentage of energy from fat was 2 times greater in Honolulu than in Japan. Although this study did not examine relations at the individual level, ignoring cause and effect, and did not control for crucial confounders such as physical activity, the results indicated the potential role that dietary fat plays in the development of obesity.

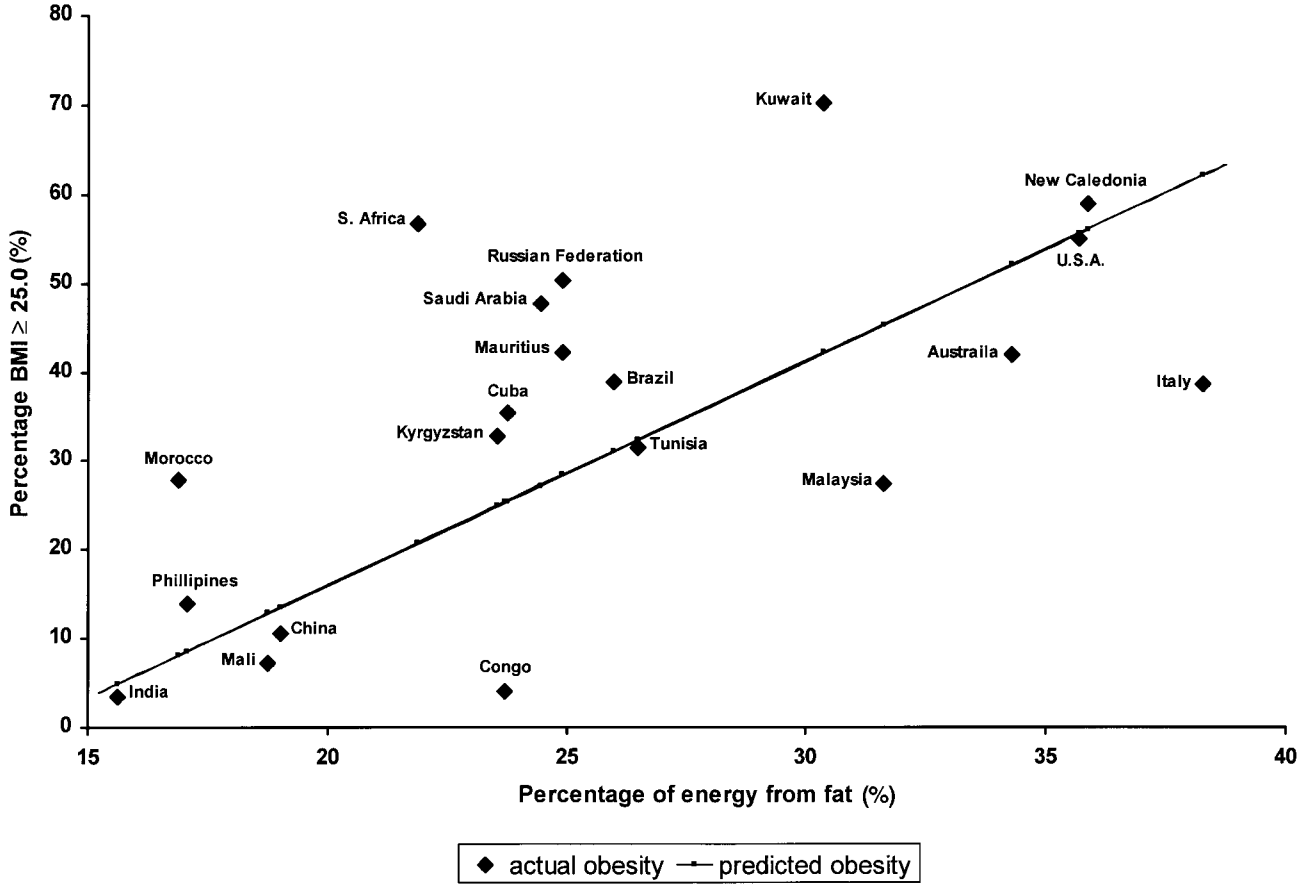

FIGURE 1. Relation between the percentage of the population that is obese and the proportion of energy intake from fat.

Collectively, fat and carbohydrate make up 85% of the diet. An increase in the percentage of one nutrient will decrease the percentage of the other as long as protein remains constant. When both nutrients were examined together over the past 25 y, an increasing intake of both fat and carbohydrate sources was shown, which correlated with the increased level of obesity.

The Leeds Fat Study provides additional data (16). When the frequency distribution of BMI in individuals who consumed a high-fat diet (>45% of energy) was plotted, the tail was skewed to the right compared with those who ate a low-fat diet (<35% of energy). With use of a BMI cutoff of 30, there were 19 times as many consumers of a high-fat diet than of a low-fat diet above this cutoff. However, note that there were large numbers of individuals eating the high-fat diet whose BMI was entirely normal, suggesting that there were important underlying physiologic factors, probably with a genetic basis, that were influencing whether there was an increase in BMI in the consumers of a high-fat diet.

Another ecologic approach is to undertake time-trend analysis of the relations between the proportion who are obese and the preceding diet. A study of 377 200 Danish military recruits from the years 1943–1974 followed the shift in obesity (defined as a BMI >31) and related it to the proportion of energy from fat (31, 38). There was a marked parallel between the increase in the percentage of energy from fat and the subsequent increase in obesity (BMI >31).

A similar set of studies was conducted among the Pima Indians in the United States (39). Parallel changes in the proportion of obesity and the proportion of energy from fat were found. Of course, many other factors changed along with diet, eg, physical activity may have declined.

As discussed in the second section of this review, the Chinese data used by Willett (1) to support the hypothesis that fat is not important are inappropriate for this purpose. Shifts in dietary fat intake in China led to increased body fatness, as shown by data from the China Health and Nutrition Survey, an ongoing, longitudinal survey of 8 provinces in China. A multistage, random, cluster-sampling procedure was used to draw the sample from each province. Additional details on the research design of this survey are presented elsewhere (35). This study collected detailed dietary data by using weighing and measuring methods for each individual over a 3-d period and also measured weight, height, and physical activity. We present data for adults collected between 1989 and 1993 and for adolescents aged 10–18 y collected between 1991 and 1993 (**Table 2**). Note that by examining changes in BMI and changes in other confounders (eg, activity and smoking in adults), we controlled for many unmeasurable factors that do not vary over time.

To test the hypothesis that energy from fat has an independent effect on body fatness, we controlled for total energy intake in one set of regressions and then examined the effect of the proportion of energy from fat. In a separate regression, we examined the effects of changes in energy from fat while controlling for the effect of changes in energy from all nonfat sources. In both cases, we found a significant independent effect of energy from

TABLE 2
Analysis of change in diet as a predictor of change in BMI with use of data from the China Health and Nutrition Survey[1]

Predictors	Adolescents, 10–18 y old[2] (n = 742)	Adults, 20–45 y old[3] (n = 6667)
Model A		
Change in energy from fat	0.0005 ± 0.0003[4]	0.0001 ± 0.00005[4]
Change in energy from nonfat	0.0001 ± 0.0001	0.000007 ± 0.00003
Model B		
Change in percentage of energy from fat	0.01 ± 0.008[5]	0.003 ± 0.002[5]
Change in total energy	0.0002 ± 0.0001[5]	0.00002 ± 0.00002

[1] Coefficient ± SE. Data from 1991 and 1993 survey used for adolescents and from 1989, 1991, and 1993 used for adults.
[2] Adjusted for age (in y), sex, and residence.
[3] Adjusted for age (in y), sex, residence, smoking, and physical activity.
[4] $P < 0.05$.
[5] $P < 0.10$.

fat on BMI. This was consistent with other studies in this same population (35). These results also showed that there was a considerably larger effect of energy from fat than of energy from nonfat sources on changes in BMI. These data are entirely consistent with the hypothesis that increasing amounts of dietary fat put a significant fraction of the population, who have a genetic susceptibility, at risk of obesity (4, 5, 31, 38, 40).

CAN A REDUCTION IN DIETARY FAT PREVENT OR REVERSE OBESITY?

Experimental data

High-fat diets can increase fat cell numbers, which limits weight loss

Lemonnier (41) was the first to show that feeding mice a high-fat diet will increase the number of adipocytes, with the intraabdominal perirenal depot showing the greatest response. Subsequently, Faust et al (42) and others (43) showed this as well. When they switched mice from a high-fat to a low-fat diet, the number of adipocytes increased. Thus, when the fat cells returned to normal size, the animals were still obese (44). Data from a study by Hill et al (44), who compared weight gain between rats who consumed a high–saturated fat diet with those who consumed a high–unsaturated fat diet, confirmed that 60% of rats in both groups became fat and both groups had an increased number of fat cells, which was particularly evident in the animals eating the high–saturated fat diet.

Switching from a high-fat to a low-fat diet may reverse obesity, but not always

Switching from a high-fat diet to a low-fat diet might be expected to reverse fat-induced obesity, unless the number of adipocytes increases. In this case, weight reduction may be incomplete. Rolls et al (45) were the first to show that rats fed a high-fat diet did not return to their baseline weight when switched to a lower-fat diet. Hill et al (44) expanded on this finding by showing that animals switched to a low-fat diet after having been fed a high-fat diet for a short time (4 mo) reduced their weight to levels the same as that of controls not eating the high-fat diet. However, animals fed the high-fat diet for 7 mo failed to reduce their weight to control values. Others also reported that body weight did not return to the level of control animals maintained on a low-fat diet throughout the study (46, 47). The extent to which genetic factors are involved in the enlargement of fat cells and in this reversal has not yet been determined, but they are likely to play an important role. These data suggest that dietary fat may be particularly important in inducing obesity, whereas a reduction in dietary fat has less of an effect on weight loss.

Clinical data

Energy density

Fat substitutes (sucrose polyester, eg, olestra) have been used to facilitate research on the degree to which covert changes in energy density alter total energy intake and macronutrient selection. Short-term studies of the substitution of indigestible fat substitutes for fat in the diet showed 2 patterns of adaptation (**Table 3**). When olestra was substituted for fat in a single breakfast meal, there was energy compensation over the next 24–36 h in healthy, young men (48, 49). When fat intake was lowered from 40% to 30% of energy by substituting fat for olestra in the noon or evening meal, there was no energy or nutrient compensation over the next 24 h (50). However, when substitution with olestra lowered the fat intake from 30% to nearly 20% of energy over 3 meals, healthy subjects felt less satisfied at the end of the substitution and compensated for nearly 75% of the energy deficit over the next day (51).

In longer-term experiments, lasting 2 wk or 3 mo, the substitution of olestra for ≈33% of the fat in a diet containing 40% of energy from fat reduced energy consumption by ≈15% [8.4 MJ (2000 kcal) reduced to 3.2 MJ (1750 kcal)] for the same weight of food. In each experiment there was only partial compensation for energy, suggesting that when the energy density of the diet did not change, the subjects continued to eat the same mass of food, even though it provided less metabolizable energy. Weight loss in the 2-wk experiment was 1.5 kg and in the 3-mo experiment was nearly 5 kg, which was significantly greater than the amount of weight lost in the control group (52, 53).

Effect of diet composition on weight loss in nonobese subjects

Decreasing total fat in the diet without intentionally decreasing total energy in many cases results in a lower total energy intake in both obese and nonobese individuals (**Table 4**). Two short-term, randomized trials found moderate weight loss with a low-fat diet containing 22–29 g fat/d (54, 55). Subjects consumed 1 of 3 diets with different fat contents (15–20%, 30–35%, or 45–50% of energy from fat) for 2 wk each. Those who consumed the lowest-fat diet lost weight and those who consumed the highest-fat diet gained weight (54). The subjects who consumed the lowest-fat diet (15–20% of energy from fat) did not compensate for the energy lost as fat. A longer-term study was conducted to explore

TABLE 3
Effect of replacing dietary fat with olestra on subsequent food intake

Reference and subjects	Design	Comment
Rolls et al (48)		
Nonobese (n = 24M)	Replacement of fat with 20 or 30 g olestra at breakfast; crossover, self-selected diet	Energy compensation occurred over the next 3 meals; no macronutrient adjustment
Burley and Blundell (49)		
Nonobese (n = 24M)	Replacement of fat with 20 or 30 g olestra at breakfast	Energy compensation occurred over the next 24 h
Cotton et al (50)		
Nonobese (n = 16M)	Replacement of fat with 55 g olestra at lunch or dinner in 2 × 2 design	No nutrient or energy compensation over 24–36 h
Cotton et al (51)		
Nonobese (n = 16M)	50 g olestra and 50 g triacylglycerol over 3 meals on day 1; no olestra on day 2; 2 × 2 design	Subjects hungrier on day 2; 74% energy compensation by end of day 2
Bray et al (52)		
Nonobese (n = 10M)	Replacement of 33% of fat with olestra in a diet containing 40% of energy from fat for 2 wk	No change in fat oxidation; significant weight loss of 1.5 kg
Roy et al (53)		
Nonobese (n = 15F)	Replacement of 33% of fat with olestra in a diet containing 40% of energy from fat for 3 mo	Olestra group lost significantly more weight than the full-fat group

further the ability to compensate for this lost energy. Thirteen women consumed both a low-fat (20–25% of energy from fat) and a high-fat (35–40% of energy from fat) diet for 11 wk each; energy intake was not controlled for (55). The low-fat group consumed significantly less energy and lost significantly more weight than the high-fat group. In a 6-wk study of Finnish men and women randomly assigned to either a low-fat diet or their usual (control) diet, energy intake was supposed to have been kept constant; however, the intervention group spontaneously decreased their energy intake and lost 0.7 kg (67).

In studies whose primary purpose was not to reduce body fat but to decrease cancer and cardiovascular disease risk, subjects eating a low-fat diet nonetheless lost modest amounts of weight. However, weight loss with the low-fat diet in these studies was confounded by several factors that usually accompany preventive treatment of these diseases, such as exercise and smoking cessation. Three long-term, randomized trials in women at risk of breast cancer found that those receiving advice to eat a low-fat diet (15% of energy from fat) lost more weight than did the control group (57, 58, 64). Twelve months after consuming the low-fat diet, however, several of the women had regained most or all of the weight they had lost (57, 58). In the Women's Health Trial Feasibility Study, neither weight reduction nor exercise was emphasized, but a low-fat, ad libitum diet as a preventive measure for breast cancer was (61). One hundred seventy-one women were randomly assigned to either a low-fat diet (20% of energy from fat) or a usual diet. All the weight lost in this study was lost during the first 6 mo. After 2 y, ≈41% of the weight lost had been regained by the low-fat group; compliance also declined slightly over the 2 y. In a study of 28 breast cancer patients randomly assigned to either a low-fat diet (15% of energy from fat) or a control diet for 3 mo, the mean weight loss of the low-fat group was twice that of the control group (59). These studies were confounded by the fact that the control group did not receive the same counseling. Healthy behaviors associated with the dietary intervention could have accounted for the weight lost.

Adherence to low-fat, ad libitum diets is also a function of the frequency of dietary counseling. Twenty breast cancer patients were instructed to consume a low-fat, high-fiber diet ad libitum. The diet was reinforced by a dietitian through monthly group sessions. Even though total energy intake increased, after decreasing initially at the start of the diet, fat intake was maintained and the patients lost weight steadily over the 6-mo study period, with a final weight loss of 5.1 kg (56).

Several intervention trials aimed at improving lipid profiles and cardiovascular disease risk in hypercholesterolemic patients found that weight loss was a secondary benefit of a low-fat, low-cholesterol diet. Women consuming the National Cholesterol Education Program Step II diet (15% of energy from fat) for 9 wk weighed 1.4 kg less than when they were consuming a high-fat diet (63). Even though total energy was meant to remain constant between diets, these patients consumed 2887 kJ (690 kcal) less with the low-fat diet than with the high-fat diet. After consuming a low-fat diet (15% of energy from fat) for 10–12 wk ad libitum, moderately hypercholesterolemic men and women lost 3.6 kg (65). After consuming a low-fat diet (≈30% of energy from fat) for 5 wk, 39 hypercholesterolemic patients lost significantly more weight than 41 hypercholesterolemic patients consuming a control (usual) diet; this loss was maintained at 26 wk (60). In another study, the reduced-fat group lost 1.3 kg, even though their energy intake increased (68). In patients who had experienced myocardial infarctions, a control (usual diet) and an

TABLE 4
Clinical trials of the relation between dietary fat intake and weight change in nonoverweight subjects

Reference and subjects	Percentage of energy from fat[1]	Energy content[1]	Duration of intervention	Mean change in weight	Weight change	Comment
	%	*MJ (kcal)*		*kg*	*g/d*	
Lissner et al (54)						
Healthy, nonsmoking women (age: 22–41 y; *n* = 24)						Maintenance of habitual exercise requested
Low-fat diet	15–20	8.7 (2087)	2 wk	−0.4	−29	
Medium-fat diet	30–35	9.8 (2352)	2 wk	−0.03	−2	
High-fat diet	45–50	11.4 (2717)	2 wk	+0.32	+23	
Kendall et al (55)						
Women (age: 25–46 y; *n* = 13)						Maintenance of habitual exercise requested; activity not different between groups
Low-fat diet	20–25	7.6 (1816)	11 wk	−2.54	−33	
High-fat diet	35–40	8.6 (2055)	11 wk	−1.26	−16	
Boyar et al (56)						
Women with breast cancer (mean age: 58 y; *n* = 20)						No mention of physical activity
	34→21	6.3→5.1 (1504→1230)	1–2 mo	−1.9		
	→21	→5.6 (→1347)	2–4 mo	−4.5		
	→21	→5.5 (→1324)	4–6 mo	−5.1		
Lee-Han et al (57)						
Women with breast dysplasia (mean age: 41.9 y)						Change in activity not controlled for
Low-fat diet (*n* = 29)	36.42→23.49	7.8→6.9 (1872→1654.2)	6 mo	−1.16	−6	
	→25.83	→6.9 (→1655.2)	12 mo	−0.93	−3	
Control diet (*n* = 28)	35.73→34.35	7.6→7.8 (1828→1867)	6 mo	+0.07	0	
	→35.88	→7.9 (→1881)	12 mo	+0.62	+2	
Boyd et al (58)						
Women with breast dysplasia (mean age: 44 y)						Physical activity not controlled for
Low-fat diet (*n* = 101)	37→21	7.3→6.4 (1753→1529)	4 mo	−1.0	−8	
	→21	→6.4 (→1528)	8 mo	−2.0	−8	
	→21	→6.5 (→1543)	12 mo	−1.0	−3	
Control diet (*n* = 106)	37→36	7.3→7.2 (1742→1717)	4 mo	0	0	
	→36	→7.2 (→1713)	8 mo	0	0	
	→35	→7.3 (→1742)	12 mo	0	0	
Buzzard et al (59)						
Women with breast cancer (age: 50–75 y)						No mention of physical activity
Low-fat diet (*n* = 17)	38.4→22.8	7.7→5.7 (1840→1365)	3 mo	−2.8	−31	
Control diet (*n* = 11)	39.4→35.4	ND[2]	ND	−1.3	−14	
Bloemberg et al (60)						
Men at risk for cardiovascular disease (age: 20–60 y)						No mention of physical activity
Low cholesterol (*n* = 39)	38.5→30.8	ND	5 wk	−1.05	−30	
	→33.5		26 wk	−0.95	−27	
Control diet (*n* = 41)	38.3→37.1		5 wk	−0.18	−1	
	→36.8		26 wk	−0.06	+0.3	
Sheppard et al (61)						
Women at risk for breast cancer (age: 45–69 y)						No emphasis on weight loss or exercise; baseline physical activity levels controlled for
Low-fat diet (*n* = 171)	39.2→20.9	7.3→5.5 (1743→1323)	6 mo	−3.2	−18	
	→21.6	→5.4 (→1302)	12 mo	−3.0	−8	
	→22.8	→5.6 (→1348)	24 mo	−1.9	−3	
Control diet (*n* = 105)	38.9→37.9	7.2→6.5 (1720→1565)	6 mo	−0.4	2.2	
	→37.2	→6.6 (→1578)	12 mo	−0.4	1.1	
	→36.5	→6.7 (→1613)	24 mo	−0.1	0	

(Continued)

TABLE 4
(Continued)

Reference and subjects	Percentage of energy from fat[1]	Energy content[1]	Duration of intervention	Mean change in weight	Weight change	Comment
	%	*MJ (kcal)*		*kg*	*g/d*	
Singh et al (62)						
Men and women with myocardial infarction						Both diets fat-modified; both groups received general advice on the importance of regular physical activity
Diet A (mean age: 50.5 y; *n* = 204)	26→24	8.8→7.8 (2110→1812)	12 mo	−6.3	−17	Advice about physical activity reinforced regularly
Diet B (mean age: 50.2 y; *n* = 202)	24→28	9.0→8.1 (2153→1940)		−2.4	−7	Advice about physical activity not reinforced
Hunninghake et al (63)						
Men (mean age: 65 y; *n* = 58) and women (mean age: 40 y; *n* = 39) with moderate hypercholesterolemia	41.4→25.8	9.5→6.6 (2270→1580)	9 wk	−1.4	−22	No washout period after lovostatin; 16 obese subjects had a weight loss of 1.8 kg; no mention of physical activity
Kasim et al (64)						
Women at risk for breast cancer (mean age: 46 y)						No mention of physical activity
Low-fat diet (*n* = 34)	36.3→17.6	8.0→6.6 (1927→1572)	12 mo	−3.4	−9	
Control diet (*n* = 38)	35.6→33.8	7.1→6.3 (1697→1499)	12 mo	−0.8	−2	
Schaefer et al (65)						
Moderately hypercholesterolemic men and women (age: 52–79 y; *n* = 14F, 13M)	35→15	ND	10–12 wk	−3.6	ND	No mention of physical activity
Westerterp et al (66)						
Nonobese men and women (age: 19–55 y)						
Lower-fat diet (*n* = 57F, 59M)	35→33[3]	10.1→10.1 (2414→2414)	6 mo	1.4	+2	Most of the energy increase explained by increases in fat intake
Full-fat diet (*n* = 51F, 50M)	36→41	10.3→11.1 (2462→2653)	6 mo		+6	

[1] Arrows indicate change from one level of energy intake to another.
[2] Not determined.
[3] Converted energy from fat (g/d) to percentage of energy from fat.

intervention (low-fat diet) group received initial counseling to consume a low-fat, high-complex-carbohydrate diet; to exercise regularly; and to stop smoking. The low-fat group received regular reinforcement of this advice but the control group did not (61). After 1 y, the low-fat group had lost 6.3 kg and the control group had lost 2.4 kg. One of the best-known studies of the effects of dietary fat on body weight is the study by Ornish et al (69). In this study, fat intake was markedly low and subjects were encouraged to make many changes to their lifestyles, particularly to increase their physical activity. The intervention group in this study lost 10.1 kg.

Although there is a mixed picture of successes and failures in the above-mentioned studies, there is no evidence that those individuals who consumed low-fat diets gained weight. As Astrup et al (26) noted in a review on this topic, there is substantial evidence that the fat and carbohydrate contents of the diet will affect body fat and, moreover, that a reduction of even a few kilograms in body weight will cause a significant decrease in the prevalence of obesity and its associated risks. Despite the variations in dietary fat intake in these studies, we conclude that dietary fat does matter; however, note that these studies did not focus on long-term weight maintenance and most did not focus on obesity. It is also important to note that weight loss rarely exceeded 100 g/d and typically ranged from 20 to 30 g/d. Expectations for greater weight loss should not be promoted; even Ornish et al's highly successful intervention was associated with a daily weight loss of only 28 g/d.

Effect of diet composition on weight loss in overweight subjects

Several trials have examined the effects of a low-fat diet with and without energy restriction on overweight subjects. The rate of weight loss was generally greater when low-fat dieting was coupled with a reduction in energy intake. Convincing evidence that variations in dietary fat intake can affect body weight is provided in **Table 5**. Because fat is an energy-dense nutrient, one way to decrease total energy in the diet is to reduce the fat content. Although the rate of weight loss with an ad libitum, low-fat, high-carbohydrate diet may not be as rapid as weight loss with diets low in total energy, ad libitum, low-fat, high-carbohydrate diets have been found to provide greater satiety; subsequently,

TABLE 5
Clinical trials of the relation between dietary fat intake and weight change in overweight subjects

Reference and subjects	Percentage of energy from fat[1]	Energy content[1]	Duration of intervention	Mean change in weight	Weight change	Comments
	%	*MJ (kcal)*		*kg*	*g/d*	
Puska et al (67)						
Men and women (age: 30–50 y)						Subjects requested to not change exercise or smoking habits
Low-fat diet (*n* = 35)	38.6→23.3	10.42→8.37 (2490→2001)	6 wk	−0.7	−17	
Control diet (*n* = 38)	37.2→38.0	10.96→10.87 (2609→2598)	6 wk	+0.2	+5	
Hammer et al (70)						
Obese, premenopausal women (mean age: 39 y)						
Low-fat, ad libitum diet (*n* = 4)	36→23	8.1→6.1 (1934→1450)	4 wk	−2.7	−96	Supervised exercise
			8 wk	−4.3	−77	
			12 wk	−4.8	−57	
			16 wk	−5.8	−52	
Low-fat, energy-restricted diet (*n* = 8)	36→24	8.1→3.5 (1934→840)	4 wk	−5.1	−182	Refrained from physical activity
			6 wk	−7.1	−169	
			12 wk	−8.4	−100	
			16 wk	−9.5	−85	
Low-fat, ad libitum diet (*n* = 8)	36→23	8.1→6.1 (1934→1450)	4 wk	−3.5	−125	Supervised exercise
			6 wk	−4.6	−110	
			12 wk	−6.0	−71	
			16 wk	−6.7	−60	
Low-energy, energy-restricted diet (*n* = 6)	36→24	8.1→3.5 (1934→840)	4 wk	−5.7	−204	Refrained from physical activity
			6 wk	−8.5	−202	
			12 wk	−10.7	−127	
			16 wk	−12.9	−115	
Alford et al (71)						
Overweight women						Subjects sedentary and agreed to remain so during the study
Moderate-carbohydrate diet (*n* = 11)[2]	35	5.0 (1200)	10 wk	−5.6	−80	
Low-carbohydrate diet (*n* = 12)[3]	45	5.0 (1200)	10 wk	−6.4	−91	
High-carbohydrate diet (*n* = 12)[4]	10	5.0 (1200)	10 wk	−4.8	−69	
Prewitt et al (72)						
Women (age: 20–48 y; *n* = 18)						Exit and entry levels of physical activity did not increase; subjects rated their daily activity levels
High-fat diet	37	7.6 (1820)	1–4 wk	+0.7	ND[5]	
Low-fat diet	21.4	8.2 (1953)	5–8 wk	0	ND	
		8.0 (1921)	9–12 wk	−0.4	ND	
		8.2 (1961)	13–16 wk	−1.3	ND	
		8.6 (2055)	17–20 wk	−1.2	ND	
		9.1 (2171)	21–24 wk	−1.3	ND	
Rumpler et al (73)						
Overweight men (mean age: 35 y; *n* = 8)						
Low-fat diet (*n* = 4)	20	12.9→6.4 (3095→1542)	28 d	−5.0	−178	
High-fat diet (*n* = 4)	40	13.3→6.6 (3182→1590)		−5.2	−185	
Shintani et al (74)	32→7	10.9→6.6 (2594→1569)	3 wk	−7.8	−371	
Obese Native Hawaiian men and women (age: 25–64 y; *n* = 19)						No mention of physical activity
Schlundt et al (75)						
Overweight men and women (age: 30–37 y)						Moderate exercise; both groups reported the same mean number (5.2) of weekly exercise sessions
Low-fat diet (*n* = 21F, 4M)	38→19[6]	9.2→6.0 (2200→1426)	16–20 wk	−4.6	ND	
Low-energy diet (*n* = 20F, 4M)	39→20	8.4→2.6 (1999→1265)	16–20 wk	−8.3	ND	

(Continued)

TABLE 5
(*Continued*)

Reference and subjects	Percentage of energy from fat[1]	Energy content[1]	Duration of intervention	Mean change in weight	Weight change	Comments
	%	*MJ (kcal)*		*kg*	*g/d*	
Powell et al (76)[7]						
Obese women (age: 25–45 y)						
10% of energy from fat (n = 8)	35.0→15.7	8.1→4.7 (1942→1116)	6 wk	−3.6	−86	
20% of energy from fat (n = 8)	39.6→20.8	6.9→4.9 (1642→1162)	6 wk	−5.0	−119	
30% of energy from fat (n = 9)	36.6→27.9	8.7→5.0 (2081→1188)	6 wk	−4.6	−110	
40% of energy from fat (n = 10)	37.2→33.5	7.8→4.9 (1865→1178)	6 wk	−4.5	−107	
Harris et al (77)						
Overweight men and women (age: 25–45 y; n = 157)						Exercise assessed and controlled for
Low-fat diet	36→31[6]	8.1→5.7 (1945→1356)	6 mo	−2.9[8]	ND	
	→32	→5.8 (→1382)	12 mo	−2.2	ND	
	→32	→5.9 (→1417)	18 mo	−1.7	ND	
Shah et al (78)						
Obese women (age: 25–45 y)						Physical activity not significantly different between groups; adjustment for physical activity did not change results
Low-fat diet (n = 47)	33.8→21.0	7.9→6.6 (1893→1580)	6 mo	−4.4	−24	
Low-energy diet (n = 42)	34.4→30.4	8.9→2.4 (2119→1550)		−3.8	−21	
Jeffery et al (79)						
Obese women (age: 25–45 y)						Duration of intervention diminished from weekly to monthly after 6.5 mo; subjects kept exercise diaries for 7 d; physical activity given modest emphasis; energy expenditure assessed with a physical activity questionnaire
Low-fat diet (n = 39)	35→22	8.2→6.7 (1965→1602)	6 mo	−4.6	−25	
	→25	→6.6 (→1580)	12 mo	−2.1	−6	
	ND	ND	18 mo	+0.4	+1	
Low-energy diet (n = 35)	35→30	9.3→6.3 (2224→1514)	6 mo	−3.7	−20	
	→31	→7.2 (→1726)	12 mo	−0.5	−1	
	ND	ND	18 mo	+1.8	+3	
Pascale et al (80)						
Obese women with type 2 diabetes (mean age: 56.5 y)						Physical activity encouraged 2 Subjects resumed oral medications
Low-fat, low-energy diet (n = 15)	32.2→22.4	6.9→5.0 (1658→1201)	16 wk	−7.7	−69	
Low-energy diet (n = 16)	35.5→30.1	7.7→5.8 (1837→1392)	16 wk	−4.6	−41	7 Subjects resumed oral medications
Obese women with risk of type 2 diabetes (mean age: 42.7 y)						
Low-fat, low-energy diet (n = 16)	39.2→22.2	8.5→5.2 (2024→1246)	16 wk	−7.5	−67	
Low-energy diet (n = 13)	36.5→30.8	9.3→5.0 (2220→1190)	16 wk	−6.9	−61	
Raben et al (68)						
Men and women (mean age: 23.9 y)						All subjects instructed to maintain physical activity levels
Low-fat diet (n = 6F, 18M)	37.4→25.6	12.8→13.4 (3059→3203)	11 wk	−1.3	−17	
Control diet (n = 8F, 16M)	→35.4	→11.5 (→2749)	11 wk	0	0	Maintained habitual diet
Golay et al (81)						
Obese men and women						Subjects hospitalized to ensure compliance; in addition to low energy, diet program included 1 h of aerobic exercise/d and 1 h of underwater physical activity/d
45% of energy as carbohydrate (mean age: 45 y; n = 15F, 6M)	26	4.3 (1027)	6 wk	−7	−167	
15% of energy as carbohydrate (mean age: 41 y; n = 19F, 3M)	53	4.2 (1007)	6 wk	−8	−190	

(Continued)

TABLE 5
(Continued)

Reference and subjects	Percentage of energy from fat[1]	Energy content[1]	Duration of intervention	Mean change in weight	Weight change	Comments
	%	*MJ (kcal)*		*kg*	*g/d*	
Siggaard et al (82)						
Overweight men and women						No significant changes in physical activity or smoking habits
Low-fat diet (mean age: 41.5 y; *n* = 44)	39→28	8.2→77 (1960→1840)	12 wk	−4.2	−50	
Control diet (mean age: 36.3 y; *n* = 15)	38.3→36.7	8.3→8.4 (1984→2008)	12 wk	−0.8	−9	
Harvey-Berino (83)						
Overweight men and women (age: 25–45 y)						All subjects received behavior -modification therapy and
Restricted-fat diet (*n* = 28)	33.3→27.2	9.1→6.2 (2171→1477)	24 wk	−11.5	−68	
Low-fat diet (*n* = 29)	30.1→20.9	8.1→6.9 (1929→1650)	24 wk	−5.2	−31exercised	

[1] Arrows indicate change from one level of energy intake to another.
[2] The diet contained 45% of energy from carbohydrate and 20% from protein.
[3] The diet contained 25% of energy from carbohydrate and 30% from protein.
[4] The diet contained 75% of energy from carbohydrate and 15% from protein.
[5] Not determined.
[6] Converted energy from fat (MJ/d) to percentage of energy from fat.
[7] Diets were intended to provide 10%, 20%, 30%, or 40% of energy from fat, but actual values are given in next column.
[8] Change in BMI (in kg/m^2).

compensation for the decrease in energy content is not complete (54, 78, 79, 84). Even in studies in which the goal is to maintain a constant energy intake, energy intake often is reduced unintentionally when a low-fat diet is consumed (67). Despite the modest weight loss and poor dietary compliance associated with long-term trials of low-fat diets, these diets may still be important for weight maintenance (66, 84). Toubro and Astrup (84) randomly assigned patients who had lost weight by means other than a low-fat diet into a group that received a low-fat diet ad libitum or a fixed-energy (weight-maintenance) diet. After 1 y, the low-fat group was 3.5 kg lighter than the fixed-energy group. At the 1-y follow-up, the fixed-energy group had regained 11.3 kg, whereas the low-fat group had regained only 5.4 kg. These data, coupled with the findings of DeGraaf et al (85) that long-term consumption of reduced-fat products leads to lower energy and fat consumption than does the consumption of full-fat products, suggest that low fat intakes may make it easier to maintain a low-energy diet and thus slow down the rate of weight gain or weight regain.

As we noted above, small reductions in weight have been shown in subjects consuming a low-fat diet when energy intake is held constant. Because of the lower thermic effect of fat and the higher energy cost of converting carbohydrate to fatty acids, fat is more readily stored in the adipose tissue than is carbohydrate. A major reason why some low-fat isoenergetic diets were not shown to cause weight loss was because the subjects were in negative energy balance (76, 81). In a study of isoenergetic diets with different proportions of carbohydrate, fat, and protein, overweight women were assigned to one of three 5.0-MJ (1200-kcal) diets: 25%, 45%, or 75% of energy from carbohydrate (71). Over the 10-wk study period, the group consuming the 25%-carbohydrate diet lost the most weight (6.4 kg), followed by group consuming the 45%-carbohydrate diet (5.6 kg). There were no significant differences among the 3 groups. It is unfortunate that in this study the individual effects of the macronutrients on weight loss could not be distinguished from each other because their proportions in each diet differed.

Powell et al (76) randomly assigned 35 obese women to 1 of 4 diets that had different fat contents (10%, 20%, 30%, or 40% of energy) but about the same amount of energy (5.0 MJ). All participants performed the same supervised workout regimen, with additional exercise restricted. All subjects lost weight and there was no significant difference in the amount of weight or body fat lost among the groups at 6 and 12 wk. In a similar study by Golay et al (81), obese men and women were randomly assigned to a 4.2-MJ (1000-kcal) diet containing either 45% or 15% of energy from carbohydrate. All participants were hospitalized for 6 wk to ensure dietary compliance and participated in 2 h of controlled physical activity daily. Both groups lost a considerable amount of weight; however, the amount of weight lost was not significantly different between the 2 groups. In both of these studies, total energy deficit was the primary factor responsible for weight loss, which was independent of the percentage of fat consumed (76, 81). However, the authors noted that it was impossible to test whether there was a differential effect between fat and carbohydrate on weight loss because no excess energy was available for storage in the adipose tissue.

Several intervention studies of overweight women who were randomly assigned to consume either a low-fat, high-carbohydrate diet ad libitum or a low-energy diet found that the reduction in dietary fat alone had a smaller effect than did the reduction in energy; subjects consuming the low-energy diet experienced more weight loss. In 2 such studies, lasting 16 wk to 6 mo, both the low-energy and the low-fat, high-carbohydrate diets produced substantial weight loss; however, in 2 studies lasting 16–20 wk, both diets resulted in weight loss, but the low-energy diet resulted in a more significant amount of weight loss than did the low-fat, high-carbohydrate diet (70, 75). Changes in physical activity pat-

terns may have been responsible for the weight loss associated with the diets but not the differential weight loss between the diets. Hammer et al (70) accounted for physical activity levels by studying groups with and without supervised exercise. The low-fat, energy-restricted group who did not exercise lost considerably more weight than the low-fat group who performed supervised exercise. Schlundt et al (75) found that the physical activity level of their subjects did not vary by diet (low-energy diet compared with low-fat diet) on the basis of daily records. Subjects consuming either diet lost a notable amount of weight, ranging from 4.6 to 9.5 kg; however, those consuming the low-energy diet lost significantly more weight. Most of the weight lost was fat. Fat-free mass remained the same in both groups in Hammer et al's (70) study, whereas fat-free mass decreased in both groups in the study by Schlundt et al (75). In both studies, even though carbohydrate was consumed ad libitum by those in the low-fat group, compensation for energy lost from the reduction in dietary fat was incomplete. The energy-restricted groups were also hungrier. The most recent comparison of the effects of a low-fat diet with those of an energy-restricted diet found that subjects in the energy-restricted group reduced their weight by 11.5 kg compared with 5.2 kg in the low-fat group after 24 wk (83). In this study, both groups received behavior-modification therapy and exercised. Thus, the consumption of low-fat diets for 6 mo has been shown to produce significant weight loss; however, this weight loss may not be as great as would be possible with energy-restricted diets. However, the long-term effects of consuming low-fat diets may be more long lasting.

In a 16-wk randomized trial, different results were found between obese women with type 2 diabetes or a family history of type 2 diabetes who were counseled to consume either a low-energy or a low-energy, low-fat diet. Both groups were encouraged to increase their physical activity and received behavior-modification therapy. Women with type 2 diabetes lost more weight with the low-fat, low-energy diet (7.7 kg) than with the low-energy diet (4.7 kg) (80). However, there was no significant difference in weight loss between the 2 diet groups in women with a family history of type 2 diabetes; the reasons were unclear.

In studies lasting >6 mo, low-fat diets resulted in greater weight loss than low-energy diets, even with diminishing compliance, and were associated with a slower rate of weight regain and a better acceptance rating by the dieters. In 2 long-term studies, Shah et al (78) and Jeffery et al (79), using the same population of obese women, found that the low-fat group had lost slightly more weight than the low-energy group after 6 mo. At this time, the energy content of the 2 diets was not significantly different but the fat and carbohydrate contents were. By 18 mo, more weight than had been lost had been regained; however, the low-fat group did not gain as much weight as the low-energy group. This difference in weight regain was attributed to a decrease in dietary compliance, more so in the low-energy than in the low-fat group, and to the diminished frequency of the intervention from weekly to monthly. In addition, the low-fat group had higher satiety scores and significantly higher palatability and quality-of-life scores.

It has generally been found that a reduction in dietary fat concomitant with ad libitum carbohydrate consumption results in weight loss. A remarkable weight loss of 7.8 kg was observed in 19 obese native Hawaiian men and women who were fed a traditional low-fat Hawaiian diet ad libitum for 3 wk (74). The percentage of fat in their diet was reduced from 32% to 7% of energy. A continued decline in body weight and percentage of body fat over 20 wk was reported in 18 women who consumed a diet containing 21.4% of energy from fat, whereas their total energy intake increased steadily to 119% of their original intake. In a study of men and women at a Danish work site, a 12-wk low-fat diet resulted in a 4.2-kg loss as opposed to a 0.8-kg weight loss in the control (usual diet) group (82). Entry and exit activity reports did not show an increase in activity levels. Harris et al (77) examined the effects of activity and diet on weight gain in overweight men and women. Using multivariate models, they found that a positive change in BMI was significantly associated with a positive change in fat when exercise was controlled for.

In the studies listed in Tables 4 and 5, we examined the energy content of the diets studied relative to the energy content of the subjects' usual diets. We found a consistent drop in energy intake of 11–31% in all studies in which energy intake was not restricted. We summarized the effects of reductions in dietary fat on weight loss in **Figure 2**. In this figure, we used as our outcome the reduction in weight loss per day for the entire set of studies that focused on reductions in dietary fat in obese individuals. Our key explanatory variable was the change in the proportion of energy from fat. We did not include isoenergetic studies. We accounted for the differential size of each clinical trial and included the proportion of men to women and the mean initial weight of the subjects as covariates in the regression. For several studies that did not provide initial fat intakes, we assumed the proportion of energy from fat to be 36%. The data depict the relation between the percentage reductions in the proportion of energy from fat in the diet and the resultant weight change. The regression did not include the study by Shintani et al (74) because it was based on a unique sample of grossly overweight Hawaiians and acted as an outlier that shifted the results inordinately. The final regression indicated that men and those with a higher initial weight lost more weight, as was expected. The most important result is shown by the slope of the regression line, which indicated that each additional percentage point reduction in dietary fat produced a daily weight loss of 1.6 g ($t = 2.02$, $P = 0.05$). The total R^2 value for the equation was 0.45; thus, the overall fit was good. Because all the studies shown were short term, the effects of long-term interventions were not addressed. Nevertheless, the results indicate a biologically important and significant relation between the percentage reduction in dietary fat and weight loss.

To check the plausibility of the relation shown in Figure 2, we calculated the quantity of fat needed from fat stores to make up for the reduction in fat in the diet. We divided the assumed intake of 8.4 MJ/d (2000 kcal/d) by 20% (protein), 40% (carbohydrate), and 40% (fat); therefore, the amount of fat ingested was ≈90 g, which provided an energy intake of 3.35 MJ/d (800 kcal/d). A reduction in the fat intake to 30% of energy without a change in the amount of the other nutrients would reduce the energy intake to 7.2 MJ/d (1715 kcal/d). Thus, 1.1 MJ (285 kcal) would be needed from other nutrient stores, mainly fat. Assuming that only fat stores would be used to compensate for the energy reduction, then 31.6 g fat would be needed, which was one-half the estimated daily loss. If carbohydrate stores or protein stores contributed to the mobilized energy reserves, then weight loss would be somewhat faster. A reduction in fat from 30% to 20% of energy would reduce energy expenditure to 7.3 MJ/d (1750 kcal/d) and would produce a fat loss of 27.8 g/d. A reduction in fat from 20% to 10% of energy would reduce energy

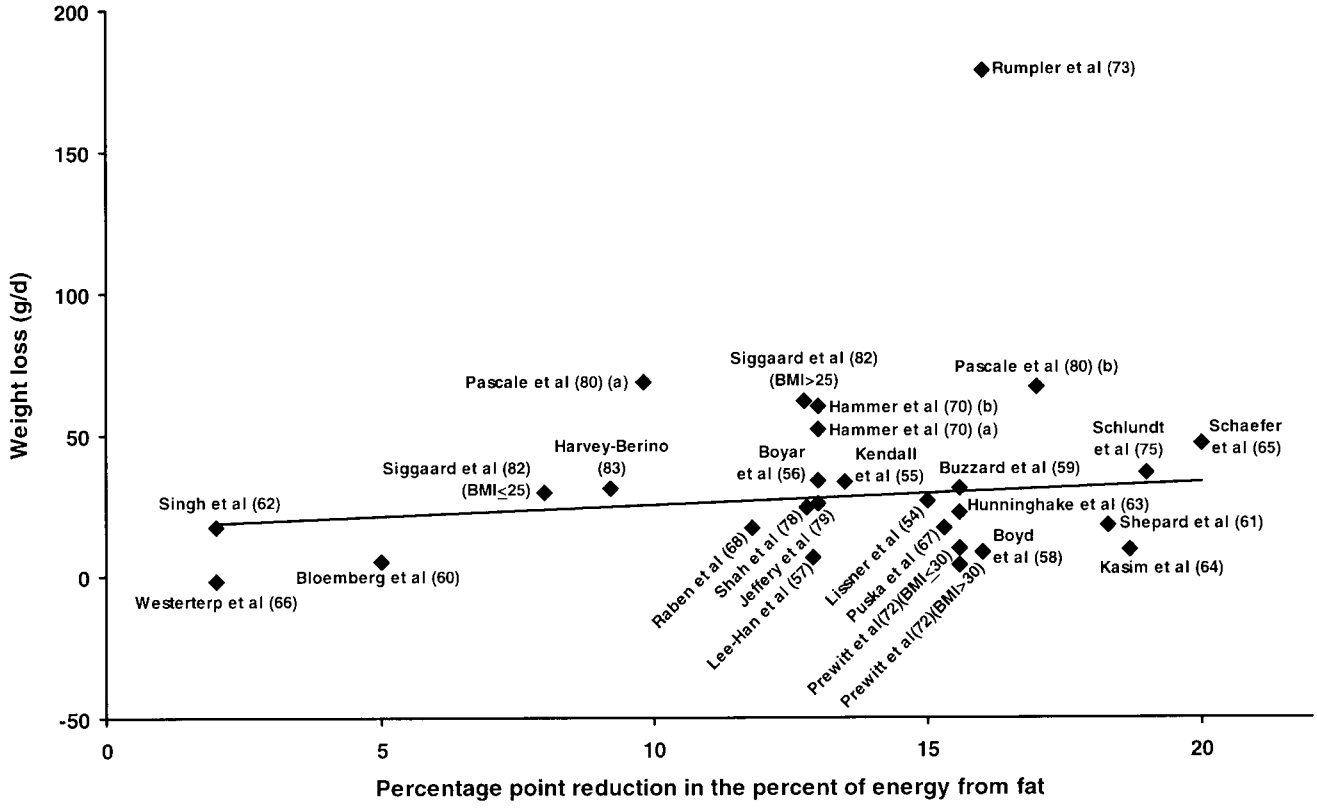

FIGURE 2. The effect of a reduction in the percentage of energy from fat on grams of weight loss per day.

expenditure to 0.9 MJ/d (222 kcal/d) and thus 24.7 g/d would be needed from reserves. It is clear that the rate of weight loss in the studies reviewed was about twice that depicted by the regression line in Figure 2, suggesting that nearly 50% of the energy reduction was compensated for. In other words, the dietary interventions were ineffective. With a marked reduction in dietary fat, there may be significant changes in fluid stores in muscle and liver that would be expected to maximize the rate of weight loss in the first few weeks.

Two points of caution are needed to interpret this analysis. The degree to which we can reduce the proportion of energy from fat by >5% or 10% is unclear. Moreover, it is possible that weight status before the dietary intervention affected the extent of weight loss. We could not explore the latter point with this small data set.

CONCLUSIONS

This review focused on the role of dietary fat in the development and treatment of obesity. It is difficult for animals and humans to become obese if they consume low-fat diets, and countries where the population consumes low-fat diets do not experience rapid increases in the level of obesity. We followed adolescents and adults in China and military recruits in Denmark during a time of increasing fat intake and found that the increase in dietary fat intake was significantly related to an increase in BMI.

High-fat diets produce obesity by enhancing passive overconsumption of energy and increasing the energy density of the diet. Reduction of dietary fat is one of the most practical ways to reduce energy density and is the approach nutritionists and dietitians frequently use to promote a reduction in total energy intake. Results from studies of fat substitutes provide one of the more rigorous data sets from which to make this assertion. Our review of the data from studies using low-fat, non-energy-restricted diets showed a reduction in energy intake from the usual diet of ≈11–31%. Moreover, the regression in Figure 2 shows a biologically plausible weight loss of 1.6 g/d per each percentage point reduction in energy from fat. This review shows that there is inadequate support for the assertion that increased fat intakes do not lead to increased energy intakes (1).

Although a reduction in dietary fat should not be expected to entirely reverse the development of obesity, a reduction in dietary fat plays an important role once obesity has developed. Although low-fat, isoenergetic diets may be helpful in reducing adiposity, we did not find the evidence for such to be convincing. Rather, the evidence indicates that dietary fat reduction should be seen as a means to reduce total energy intake and to reduce the energy density of the diet. Low-fat diets are more satiating because they usually contain high amounts of complex carbohydrate. It is clear that the increased satiety and improved palatability of low-fat diets tend to improve compliance. The experimental data in humans suggest that passive overconsumption occurs more readily when sweet, high-fat foods rather than low-fat foods are consumed. Thus, satiety may occur more consistently and sooner with low-fat, nonsweet foods.

We subscribe to the 1996 Surgeon General's report on physical activity that documents convincingly the important role that a sedentary lifestyle plays in the development of obesity and the importance of regular physical activity in the treatment of obesity (86). We concur with the sentiment that recommendations to increase the consumption of energy-dense, reduced-fat foods

will not reduce total energy intake and might, in some circumstances, actually mislead the public into consuming more energy than they would have otherwise.

In summary, data from animal and clinical research, several controlled trials, and ecologic and epidemiologic studies were reviewed and discussed to support our assertion that dietary fat intake does affect obesity. The data show that a reduction in fat intake as a means to reduce total energy intakes can result in a weight loss of 20–100 g/wk—a weight loss that is clinically significant when it exceeds 5% of body weight.

Energy balance is maintained when energy intake equals energy expenditure. There is no doubt that obesity results from an imbalance between energy intake and energy expenditure. Both the costs of decreasing energy expenditure and the benefits of increasing energy expenditure have been shown in many studies. Energy intake, particularly the nutrient-dense portion of the diet provided by fat, is equally important. Overall, this review had 3 main theses: *1*) obesity rarely occurs when very-low-fat diets are consumed; *2*) when fat intakes increase, the likelihood of obesity also increases; and *3*) the effects of fat intake on weight gain are not equal to the effects of fat reduction on weight loss.

We have argued that the increasing prevalence of obesity in developing countries can be explained in part by increases in the availability of high-fat products and by increases in the amount of ingested fat over the past 2 decades. It also appears that the risk of becoming obese in countries with high dietary fat intakes is a function of the amount of fat eaten and the genetic makeup of the individual. For some people, consumption of a high-fat diet does not put them at risk of obesity. For many others, particularly those with little restraint, consumption of a high-fat diet and a genetic predisposition to obesity increases the risk of obesity. Losing weight by lowering dietary fat intakes has a modest but predictable effect.

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