Is dietary fat a major determinant of body fat?[1,2]

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ABSTRACT The percentage of energy from dietary fat is widely believed to be an important determinant of body fat, and several mechanisms have been proposed to account for such a relation. Comparisons of both diets and the prevalence of obesity between affluent and poor countries have been used to support a causal association, but these contrasts are seriously confounded by differences in physical activity and food availability. Within areas of similar economic development, regional intake of fat and prevalence of obesity have not been positively correlated. Randomized trials are the preferable method to evaluate the effect of dietary fat on adiposity, and are feasible because the number of subjects needed is not large. In short-term trials, a modest reduction in body weight is typically seen in individuals randomly assigned to diets with a lower percentage of energy from fat. However, compensatory mechanisms appear to operate because in trials lasting ≥1 y, fat consumption within the range of 18–40% of energy appears to have little if any effect on body fatness. Moreover, within the United States, a substantial decline in the percentage of energy from fat consumed during the past two decades has corresponded with a massive increase in obesity. 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 fat will not be a solution. *Am J Clin Nutr* 1998;67(suppl):556S–62S.

KEY WORDS Dietary fat, obesity, adiposity, overweight, ecologic studies, randomized trials

INTRODUCTION

Excess body fat is the largest nutrition-related problem in the United States as well as many other affluent countries. Excess adiposity can account for ≈30–40% of heart disease (1, 2), many cancers of several types (3), most cases of adult-onset diabetes (4), and a substantial proportion of disabling osteoarthritis (5). Whereas genetic factors influence which individuals within a population will develop adiposity, diet and lifestyle factors are clearly the primary reason for the high rates of excessive body fat in our population. Evidence for this is provided by the dramatic changes in prevalence of overweight in migrants from countries with minimal adiposity who come to the United States; for example, the prevalence of obesity was threefold higher in Japanese men living in San Francisco than in those living in Japan (6). Also, the major increases in adiposity within many populations over time, including the general US population (7), cannot be explained by genetic factors. Dietary fat is among the aspects of lifestyle often said to be responsible for these high rates of adiposity. Indeed, reduction in obesity has often been a primary justification for recommendations to reduce dietary fat.

Several mechanisms have been proposed to explain why high fat intake should lead to greater body fat (8, 9). Dietary fat is the most energy-dense macronutrient, providing ≈38 kJ/g as opposed to 17 kJ/g for carbohydrate or protein; this could lead to overconsumption of energy if food weight or volume is regulated. Also, fats are often said to lend greater flavor and palatability to foods, which could lead to greater consumption of them. In addition, when studied under careful metabolic conditions, carbohydrates produce a greater thermogenic effect than fat, suggesting that dietary fat may be utilized more efficiently and accumulate as body fat more readily (10, 11). Finally, Flatt (12) suggested that carbohydrate intake is regulated, but fat intake is not; thus, individuals eating a high-fat diet will tend to consume more total energy to gain the same amount of carbohydrate as someone eating a low-fat diet. For all these reasons, dietary fat composition must be considered as a possible important determinant of body fat. If a reduction in the percentage of energy from fat in the diet in fact has a substantial effect on body fatness, this would be a major reason to advocate low-fat diets.

EMPIRICAL EVIDENCE

Between-population (ecologic) studies

The observation that the prevalence of overweight tends to be higher in affluent countries with high fat intakes than in poorer regions of the world with low fat intakes has frequently been mentioned to support a relation between dietary fat and body fat. Because such observations are exceedingly confounded by the availability of food and the level of physical activity in the subjects, such comparisons can be completely misleading. More relevant are comparisons within regions of the world with similar degrees of economic development. Among European countries, no association was observed between the national percentage of energy from fat and median body mass index in men, even though fat intake varied from ≈25% to 47% of energy (14). However, a clear inverse relation was observed in women (**Figure 1**) (14). In a study of 65 counties in China, no correlation was found between dietary fat intakes, which ranged from ≈8% to 25% of energy, and body weight (15).

Ecologic studies such as these have many limitations, including variable quality of dietary data and confounding by unmeasured variables such as activity levels, smoking, and cultural atti-

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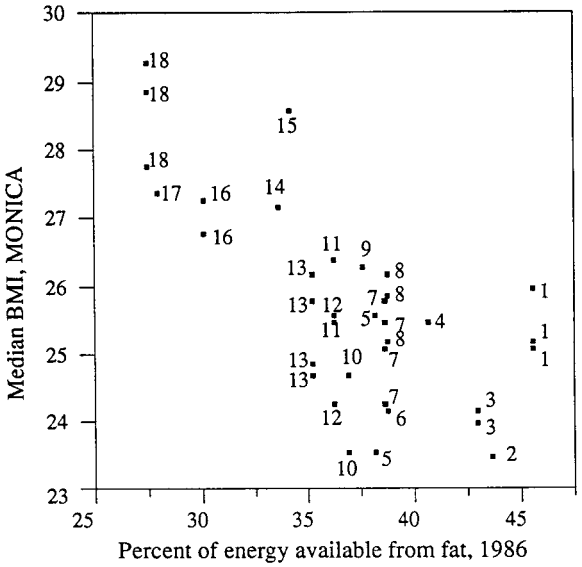

FIGURE 1. Median BMI of women taken from surveys in Europe from 1982 to 1986 versus percentage of energy available from fat.MONICA, World Health Organization MONICA Project [monitoring trends and determinants in cardiovascular disease (13)]; 1, Belgium; 2, Denmark; 3, Switzerland; 4, United Kingdom; 5, France; 6, Iceland; 7, West Germany; 8, Finland; 9, Spain; 10, Sweden; 11, Hungary; 12, Italy; 13, East Germany; 14, Czechoslovakia; 15, Malta; 16, Poland; 17, Yugoslavia; 18, USSR. Reproduced with permission (14).

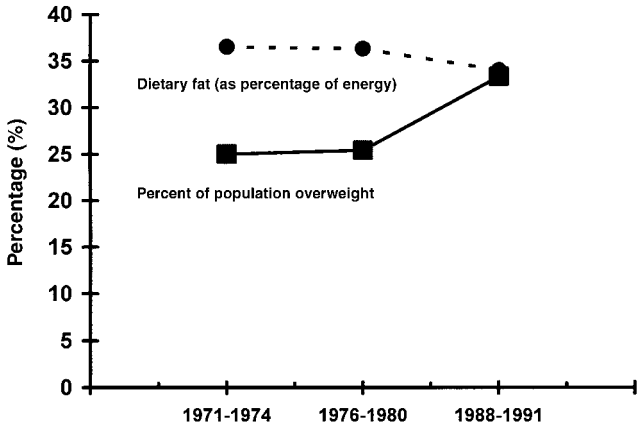

FIGURE 2. Changes in dietary fat (as percentage of energy) and the percentage of population that is overweight (3, 7, 16).

tudes toward body fat. Nevertheless, the failure to observe any clear, positive association between dietary fat intake and obesity among areas with roughly similar degrees of affluence weighs against finding an important causal relation. Because higher fat intake typically accompanies newly gained affluence, time trends in dietary and body fat within countries in transition from poverty or agrarian lifestyles to greater affluence are likely to be confounded by changes in food availability and level of physical activity. Thus, such correlations in time are generally not informative regarding the causal role of dietary fat and obesity. However, in the United States and several other northern European countries, fat intake and affluence have been disassociated by conscious efforts to reduce fat intake. Thus, it is notable that as fat intake has declined as a percentage of energy over the past 25 y the prevalence of obesity has dramatically increased in the United States (**Figure 2**) (3, 7, 16).

Within-population correlations

Numerous cross-sectional studies, reviewed by Lissner and Heitmann (14), were conducted to examine the correlation between dietary fat and body fatness within populations. Results are inconsistent; positive associations were seen in some studies (17–27), but not in others (20, 21, 25, 27, 28).

Unfortunately, most cross-sectional studies within populations are also prone to confounding that is almost uniquely intractable. In most populations studied, both dietary fat and the desirability of avoiding overweight are strongly linked with general health consciousness in recent years. The confounding is particularly problematic because health-conscious persons do have awareness and influence over the primary determinants of body weight, specifically the amount they eat and their level of physical activity, which are both measured imperfectly in free-living populations and thus difficult to control for statistically. Therefore, it should not be surprising that the fat composition of the diet is positively associated with body fat in many studies. Unlike many other health-related indexes, body fatness is readily apparent to the individual, who may alter dietary intake because of weight or weight gain.

Prospective studies are generally considered a substantially stronger epidemiologic design than cross-sectional studies. However, they are similarly susceptible to severe confounding when individuals are aware of the dependent variable (weight status) and also have conscious control over its primary determinants (physical activity, total energy intake, and the percentage of energy from fat). Relatively few prospective studies of dietary fat and weight change have been published (23, 26, 27, 29, 30), and the findings are inconsistent.

The study of Colditz et al (23) provided indirect support for confounding by health consciousness and an intent to lose weight. In this large prospective cohort, weight gain was weakly positively associated with animal fat (generally viewed as unhealthy) but weakly negatively associated with vegetable fat (generally viewed as healthier) even though there is little reason to believe these two fats would be metabolically distinct in relation to weight gain. Because of the serious potential for confounding that is extremely difficult to control, both cross-sectional and prospective studies are likely to be particularly unhelpful in determining the causal relation between the fat composition of diets and body fat.

RANDOMIZED TRIALS

Because of the potential for confounding in studies both among populations and among individuals within populations, randomized trials are the most desirable way to determine the effects of dietary fat on body fatness. Although this relation has great potential importance, surprisingly few studies have been designed to address this issue (8).

In short-term, randomized trials (those lasting from a few weeks up to 6 mo), modest weight losses (< 1–4 kg) are typically seen when 10–15% of energy from fat is replaced by carbohydrate (17, 31–35). In a recent 6-mo trial in which 45 full-fat foods were replaced with low-fat versions, fat intake was reduced by 7% of energy and weight was reduced by 0.6 kg (36). Although the effects

of fat reduction on body weight in short-term studies were modest, these could potentially be important if they were cumulative over periods of years. Thus, long-term studies are critical.

Long-term, randomized trials studying fat reduction and body weight are few, and most data are secondary observations from studies in which body weight was not the primary outcome. Most were pilot studies of fat reduction for the prevention of cancer or cardiovascular disease (**Table 1**) (30, 37–41).

The only double-blind, long-term study of fat reduction appears to be the National Diet-Heart Study (37) conducted in 900 individuals, in which foods with variable fat content were provided to participants. The difference in fat intake between groups was 30% compared with 35% of energy and after ≈1 y the difference in weight was only 0.8 kg (**Figure 3**) (37). Although the difference in fat intake was not large, this is similar to the difference between current US diets and US dietary goals.

Two long-term trials of fat reduction were pilot studies for interventions targeting breast cancer. In the Women's Health Trial (30), fat intake was to be reduced from ≈38% to 20% of energy. Reported compliance was nearly perfect by 6 mo and decreased only modestly by 24 mo. Women in the low-fat group lost 3.2 kg of body weight by 6 mo; however some of this was regained so that by 24 mo they had lost only 1.9 kg, and the difference between the intervention and control group was 1.8 kg. This study strongly suggests that weight losses with low-fat diets are not cumulative over time and indeed seem to be transient, at least in part. In a trial of fat reduction in the prevention of skin cancer (40), the reduction in body fat with a low-fat diet relative to the control diet was only ≈1 kg.

The study of Kasim et al (39) included only overweight women, and thus, presumably those who would be susceptible to higher-fat diets. Fat intake was reduced to 17.6% of energy at 1 y, therefore providing a major contrast in diets. This study included more detailed data on body composition than most other investigations (**Table 2**) (39). The difference in weight change between intervention and control groups was somewhat greater than in the other studies, being 2.6 kg. However, lean mass as well as fat mass was lost so that the difference in change between treatment groups in percentage of body fat at 1 y was only 0.7%, and no effect was observed on the waist-to-hip circumference ratio. Thus, despite a large contrast in diet, the change in adiposity was minimal. Importantly, the maximum difference in weight was seen at 3 mo, with no subsequent divergence between groups.

A major limitation of most long-term studies of fat reduction is that the control groups did not receive dietary instruction and motivation comparable to the intervention groups. In these studies the intervention subjects were generally given state of the art individual and group instruction and support to increase consciousness about dietary fat, sometimes including the provision of scales to weigh food and control portion sizes. Thus, changes in body weight, to the extent that they occurred, could have been the result of greater attention to intake of total energy rather than just attention to fat. To determine whether dietary fat reduction reduces body fatness, an appropriate control group would receive dietary instruction and counseling of similar intensity but directed at the reduction of carbohydrate or total energy. Such a study was recently reported by Jeffery et al (41). Participating women, who were initially moderately obese, received counseling either to reduce fat to 20 g/d or to reduce overall energy intake to 5000 kJ/d. Both groups lost weight initially but after 6 mo they also gained weight in parallel (**Figure 4**) (41). By 18 mo

TABLE 1
Long-term trials studying fat reduction and body weight

Study	Duration	Fat in diet	Greatest weight loss	Change in weight at end trial	Comments
	mo	*%*	*kg*		
National Diet-Heart Study Research Group (37)					
Intervention (n = 450)	12–20	30	−2.8	−2.3	The only double-blind study.
Control (n = 450)	12–20	35	−2.3	−1.5	
Sheppard et al (30)					
Intervention (n = 171)	24	20	−3.2	−1.9	A somewhat large difference at 6 mo became smaller with time.
Control (n = 105)	24	38	−0.4	−0.1	
Boyd et al (38)					
Intervention (n = 100)	12	21	−2.0	−1.0	—
Control (n = 106)	12	37	0	0	
Kasim et al (39)					
Intervention (n = 34)	12	17	—	−3.4	Weight differences reflect changes in lean as well as fat mass as there were no differences between groups in changes in percentage body fat, BMI, or waist-to-hip ratio.
Control (n = 38)	12	36	—	−0.8	
Black et al (40)					
Intervention (n = 38)	24	21	−3.0	−2.0	No significant difference in weight at anytime during the 24 mo.
Control (n = 38)	24	40	−1.0	−1.0	
Jeffrey et al (41)					
Intervention (n = 39)	18	26	−4.6	+0.4	Fat counseling for < 20 g/d, energy reduction to 1000–1200 kcal/d (4184–5021 kJ/d). Thirty three percent of original participants failed to finish trial. Adherence to both fat and energy reduction regimen was poor after 6 mo.
Control (n = 35)	18	33	−3.7	+1.8	

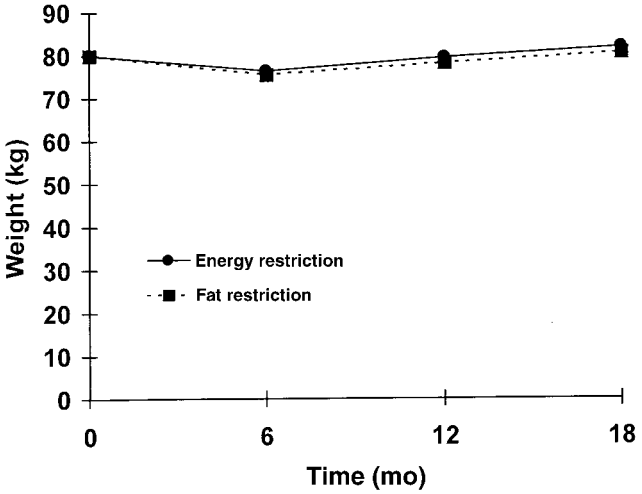

FIGURE 3. Changes in fat and energy intake and body weight over 1 y (37).

TABLE 2
Randomized trial of a low-fat diet in 72 women[1]

	Baseline	12 mo	Difference
Dietary fat (% of energy)			
Low-fat diet	36.3	17.6	—
Control	35.6	33.8	—
Weight (kg)			
Low-fat diet	66.8	63.4	−3.4
Control	72.7	71.9	−0.8
Percentage of body weight (%)			
Low-fat diet	31.8	30.3	−1.5
Control	35.1	34.3	−0.8
Waist-to-hip ratio			
Low-fat diet	0.74	0.73	−0.01
Control	0.77	0.76	−0.01
HDL cholesterol (mmol/L)			
Low-fat diet	1.56	1.44	−0.12
Control	1.47	1.56	+0.09

[1] From reference 39.

there was no significant or material difference between groups. It is notable in this trial that women eating the low-fat diet indicated greater palatability of the low-fat food, which could presumably enhance compliance. Nevertheless, this was not reflected in long-term lower weight, and might have contributed to overeating of low-fat foods.

An important concern in any long-term study of dietary change is that compliance may deteriorate with time so that the lack of a substantial effect on body weight could simply be the result of a lack of difference in diets. In general, studies of fat reduction have been hindered by a lack of a well-documented measure of compliance. However, reduced concentrations of HDL cholesterol occur consistently with low-fat diets (42). Although depression of HDL cholesterol is not specific to fat reduction on a group basis, it does appear to be reasonably sensitive to changes in dietary fat (depressed HDL cholesterol can also result from reduced physical activity, cigarette smoking, abstinence from alcohol, and weight gain). Thus, it is notable that the studies by Kasim et al (39) and Lee-Han et al (43) both showed reductions in HDL concentrations that were maintained over time. Using the data on changes in HDL concentrations at 12 mo in the Kasim study (Table 2) and the Mensink and Katan meta-analysis of metabolic studies on dietary fat and blood lipids (42), it can be back-calculated that the difference in fat intake at 12 mo was ≈20% of energy, which is similar to the reported difference (**Appendix A**). Thus, these studies do not support the notion that failure to observe a substantial weight loss with long-term, low-fat diets is simply the result of noncompliance.

SHORT-TERM WEIGHT-LOSS TRIALS

One hypothesized reason to expect lower body fat with a diet with a lower percentage of energy from fat is the difference in metabolic efficiency of processing fat compared with carbohydrate or protein. Three studies of weight loss are germane to this proposed mechanism (44–46). In the larger of these studies, Powell et al (44) provided 5000-kJ diets to women randomly assigned to consume 10%, 20%, 30%, or 40% of energy from fat. No significant differences were seen in weight change, although the magnitude of reduction was actually somewhat less with the lowest fat intake (**Table 3**). The studies by Alford et al (45) and Lehmann et al (46) also found no effect of the fat composition of the diet on weight loss. These data indicate that under realistic circumstances the theoretical differences in metabolic efficiency associated with different levels of fat intake do not account for important differences in rate of weight loss.

DISCUSSION OF INTERVENTION TRIALS

The long-term randomized trials of fat reduction published thus far provide strong evidence that the effect of dietary fat over a range of 18–40% of energy on body fatness is, at most, small. The findings are particularly inconsistent with the hypothesis that body fat is proportional to dietary fat (47, 48). The lack of major effects is particularly notable because, with the exception of the National Diet-Heart study (37) and the study by Jeffery et al (41), the designs of the other trials were seriously biased in favor of finding an effect of fat reduction.

Other issues in many studies, in addition to the lack of an appropriate control group, complicate what would seem to be a simple hypothesis to test. One of these issues is that fat reduction may be confounded by differences in the fiber content or the energy density of the diet. For example, in most of the fat reduction trials, subjects were generally counseled to consume high amounts of fruit and vegetables as well as whole grains and legumes. In one trial of fat reduction in elderly individuals (35), subjects continuing a baseline high-fat diet (35% of energy from fat) were compared with subjects consuming an isoenergetic low-fat diet. No weight loss was observed over a 12-wk period, again arguing against any important difference in efficiency of energy utilization. However, when subjects were allowed to eat the low-fat diet ad libitum for another 12 wk, a modest weight reduction was observed. This diet was exceptionally high in fiber and had a low energy density, and subjects complained of fullness and abdominal discomfort with the isoenergetic low-fat diet. Thus, in this study, and potentially in other studies, low-fat diets might be confounded by high fiber or low energy density.

It might be argued that this is simply a desirable and inevitable consequence of a low-fat diet and thus should not be

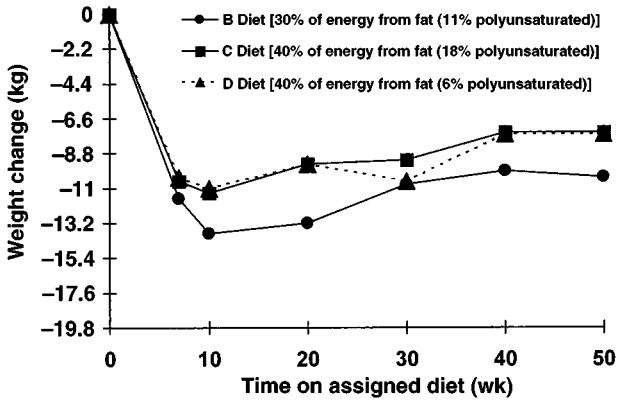

FIGURE 4. Fat and energy restriction and weight change over 18 mo (41).

controlled for in the study design. However, low energy density is not an inevitable characteristic of low-fat diets; as many of the low-fat foods presently being promoted in our commercial food supply are based on sugar or highly refined carbohydrates and often have energy values similar to those of their high-fat counterparts. On the other hand, in the Mediterranean tradition, abundant amounts of vegetables are consumed along with whole grains and olive oil, thus providing a high-fat, high-fiber, and high-volume diet. Because there is not an inevitable relation between the percentage of fat and the energy density of diets, it is important to distinguish between these effects in the design of studies to assess the effect of the percentage of energy from fat on body weight. Whether the energy density of the diet has an important effect on long-term body fat is, of course, an important question in itself, and short-term studies suggest that this may be the case (17, 20, 35, 49–51). However, the contrasts between short- and long-term studies of dietary fat indicate the need for long-term studies of energy density and body weight before any conclusions can be drawn. Also, it is possible that in highly sedentary populations with low energy demands, the energy density may need to be extremely low to have any important effect.

Subtle aspects of the diets being compared could potentially confound randomized trials of dietary fat composition and body weight. Studies in animals fed cafeteria-style diets suggest that palatability, flavor, and texture may influence over- or undereating (52). More subtle aspects of palatability may be difficult to control in any particular circumstance, but at least an attempt to do so should be made so as not to artificially favor one group or another. The relative palatability of the diets may be specific to the study population.

Finally, the possibility exists that individuals vary in their genetic susceptibility so that some will gain weight with high-fat diets and others will not. A recent study suggests that a family history of obesity might be an indicator of susceptibility to weight gain with high-fat diets (53). Although it is possible that susceptible individuals exist, the lack of any substantial overall effect of fat reduction on body weight in the long-term randomized trials suggests that the susceptible subgroup is not large or that other persons are susceptible to weight gain with high-carbohydrate diets.

The fact that studies of dietary fat fail to explain the prevalence and increase in excess body fat in our population indicates the need to consider alternative causes. Abundant evidence

TABLE 3
Twelve-week weight change in 35 women consuming 5000 kJ diets[1]

Percentage of energy from fat	Baseline weight	Change in weight
	kg	
10%	95.0	−4.5
20%	88.2	−6.8
30%	101.4	−6.9
40%	85.0	−6.8

[1] From reference 44.

clearly supports a central role of physical activity in the regulation of body fat. Numerous cross-sectional studies indicate an equilibrium between physical activity and body fatness, and intervention studies show that increased physical activity at least stabilizes body weight and may lead to modest reductions (54). It has been noted that the magnitude of physical activity in these intervention studies was small in relation to the levels of activity typical of nonindustrialized countries, so the full potential effect of higher activity levels has not been adequately addressed in such investigations (54).

The characteristics and availability of food and the social context of food consumption are also likely to have important effects on body fatness, but these are difficult to study in free-living populations. The food industry has invested greatly in research on texture, color, sweetness, saltiness, and flavor of food, as well as on its packaging and promotion, all of which has been designed to enhance consumption. It seems highly unlikely that these efforts have not contributed to overweight, although these contributions are difficult to quantify. A national character that values quantity and abundance over quality and presentation may further aggravate the situation. Finally, the almost ubiquitous presence of food in our life and the convenience with which it is eaten is likely to contribute further to overconsumption, but again quantification of such subjective factors is probably impossible.

CONCLUSIONS

In short-term studies, a modest reduction in body weight is typically seen in individuals assigned to diets with a lower percentage of energy from fat. However, compensatory mechanisms appear to operate such that in the longer term, fat consumption within the range of 18–40% of energy appears to have little if any effect on body fatness. The nature of these compensatory mechanisms is presently unknown. The possibility that individuals vary in their susceptibility to high-fat or high-carbohydrate diets deserves further examination. Nevertheless, diets high in fat are not the primary cause of the high prevalence of excess body fat in our society, nor are reductions in dietary fat a solution. Other means will be needed to reduce substantially the prevalence of obesity; enhancement of physical activity appears to be the most effective physiologic alternative.

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APPENDIX A

In the meta-analysis by Mensink and Katan (42), the change in serum HDL (mg/dL) = 0.47 (carbohydrate $\rightarrow$ saturated fat) + 0.34 (carbohydrate $\rightarrow$ monounsaturated fat) + 0.28 (carbohydrate $\rightarrow$ polyunsaturated fat), where substitutions of fat for carbohydrate are in percentage of energy. A value of 0.4 was used as an approximate weighted average representing a typical mix of dietary fats and assuming the proportions were not substantially changed. If a more substantial weight loss occurs, a correction could be made for this, ideally by using the data on changes in weight and HDL concentrations internal to the study.