Diet composition, energy intake, and nutritional status in relation to obesity in men and women

WAYNE C. MILLER

Department of Kinesiology,
Indiana University,
Bloomington, IN 47405

ABSTRACT

MILLER, W. C. Diet Composition, energy intake, and nutritional status in relation to obesity in men and women. Med. Sci. Sports Exerc., Vol. 23, No. 3, pp. 280–284, 1991. There are dietary factors besides the total energy value of food that can affect adiposity by disrupting the balance between energy intake and expenditure. The purpose of this paper was to examine how perturbation of these dietary factors that control energy balance affects adiposity. There is a substantial amount of evidence suggesting that obesity is not associated with overeating, but with a high dietary fat-to-carbohydrate intake ratio. Physiological adaptations to energy-reduced dieting facilitate both weight regain and make it more difficult to lose weight during subsequent dieting attempts. Since obesity may be better characterized by diet composition than by energy intake, successful weight-loss programs should include diet compositional changes in their regimes.

WEIGHT LOSS, DIETARY FAT, DIETARY CARBOHYDRATE, BODY FATNESS, ADIPOSITY

Heredity, diet, and energy expenditure are the three factors usually accepted as controlling fat deposition in the development of obesity. Although diet and energy expenditure are considered direct inducers of obesity, heredity is indirect, regulating an individual’s response to diet and/or activity. Regardless of the obesity-promoting factor, the underlying assumption in fat deposition is the principle of energy balance: if an individual consumes more energy than he or she expends, weight gain follows, and, if an individual expends more energy than he or she consumes, the result is weight loss. Thus, it is commonly believed that adipose tissue accumulation is simply due to a disequilibrium in energy balance, resulting from energy intake exceeding energy expenditure.

However, there are other dietary factors besides the total energy value of food that can disrupt this balance between energy intake and expenditure. For example, studies have shown that dietary fat and dietary sugar can promote obesity without excessive energy intake (10,11,13,18,19). Other studies have demonstrated a negative relationship between carbohydrate consumption and adiposity (6,15). In addition, consuming 5–24 g·d⁻¹ of indigestible fiber has been shown to assist in weight loss (14,24). These research findings imply that diet composition may be as important as diet energy content in promoting obesity. The purpose of this paper was to examine the relationships among diet composition, energy intake, and body fatness and how perturbation of these dietary factors that control energy balance affects adiposity.

ENERGY INTAKE AND BODY FATNESS

It is generally assumed that the primary cause of obesity is overeating. However, the literature suggests that obese individuals do not consume more joules than their lean counterparts (3,6,12,15,21,23). Research from our laboratory supports the notion that excessive energy intake, in and of itself, is not the principal cause of obesity (15,18,19). In a recent study (15), the relationships among diet composition, energy intake, and body fatness were examined in males (N = 107) and females (N = 109) ranging in age from 18 to 71 yr. All subjects were hydrostastically weighed for determination of body fat content (5.7–49.0%) and had their diets evaluated by use of a 3-d dietary recall and a food frequency questionnaire. Correlational analysis revealed that there was no significant relationship between daily energy intake, expressed as kJ·kg⁻¹ body weight, and adiposity for either sex. When energy intake was expressed as kJ·kg⁻¹ lean body weight, increased adiposity was negatively related to energy intake for both males (r = −0.42, P < 0.001) and females (r = −0.37, P < 0.001).

Additional research was performed where adult men and women were divided into lean and obese subgroups (15). For the males, lean was defined as ≤15% body fat and obese was defined as ≥25% body fat. For the
females, the values were \( \leq 20\% \) and \( \geq 35\% \) body fat, respectively. Group analysis showed that daily energy intakes of lean and obese adults were identical when expressed as \( \text{kJ}\cdot\text{kg}^{-1}\) lean body weight (Fig. 1). Lean individuals actually consumed more energy than the obese when daily intake was expressed as \( \text{kJ}\cdot\text{kg}^{-1}\) total body weight (Fig. 1). It is interesting to note that there were no significant differences in energy intake between genders. In other words, relative energy intake of obese males was the same as that of obese females, and relative intakes of lean males and lean females were identical (Fig. 1).

Other investigators have produced similar results. Dreon et al. (6) studied dietary intake and body composition of 155 middle-aged men and found no association between energy intake and body weight, body fat, or lean body mass. Romieu et al. (23) also found no relationship between energy intake and obesity in 141 middle-aged females when age and exercise were eliminated as cofactors. Large data-based studies from the National Health and Nutrition Examination Survey (3), the German Democratic Republic (21), and the Netherlands (12) have all failed to demonstrate that obesity is related to overeating. This research suggests that differences in energy intake are not the major cause of the variations in adiposity in adults and that it is important to consider other factors that confound the relationship between energy intake and body fatness as promoters of obesity.

Even if the cause of increased adiposity is not overeating, treatment for the overweight individual usually involves a reduction in daily energy intake. However, energy-reduced dieting may not be the best weight-loss therapy because of the many problems associated with reduced energy consumption. Severe restriction in energy intake reduces metabolic rate, which may shift the energy balance back in the direction of energy storage, counteracting any attempts to lose weight by “cutting calories” (4). In addition, significant amounts of fat-free body weight can be lost along with body fat when reduced energy consumption is imposed (17,25). Both of these problems may be attenuated if exercise is incorporated into the treatment program (16,25).

The energy conservation process resulting from severe dieting can persist after the dieting period, which causes body weight to be regained at a rapid rate until it reaches the pre-dieting level (1,11,22). This can lead to repeated dieting or a cycle of rapid weight loss followed by rapid weight gain. The effect of weight cycling has been studied in both animals and humans. In an animal study (5), laboratory rats were fed a high-fat diet to promote the development of obesity. They were then food-restricted and refed for two cycles. This weight cycling affected the rates of weight loss and weight regain in these animals. It took only 21 d for the rats to lose 130 g of body weight on their first dieting attempt but 46 d to lose 130 g on the second attempt. Following the first diet, the rats regained their lost body weight within 46 d, compared with only 14 d after the second diet.

Similar findings have been demonstrated in humans (2). A multidisciplinary weight-loss program that included a very-low-calorie diet (\(< 3.35 \text{MJ} \cdot \text{d}^{-1}\) was used on both an outpatient and inpatient basis. A retrospective review was conducted with the patients in both groups who had participated in the same weight-loss program at least twice (both times as either an outpatient or an inpatient). In each case, the second dieting period was characterized by a lower rate of weight loss than the first dieting period. For the inpatients, weight loss was at a rate of \(0.47 \pm 0.09 \text{kg} \cdot \text{d}^{-1}\) during the first diet and \(0.37 \pm 0.03 \text{kg} \cdot \text{d}^{-1}\) during the second diet (\(P < 0.05\)). The outpatients lost weight at a rate of \(0.19 \pm 0.03 \text{kg} \cdot \text{d}^{-1}\) on the first diet compared with \(0.15 \pm 0.003 \text{kg} \cdot \text{d}^{-1}\) on the second diet (\(P < 0.004\)). These data suggest that weight cycling through energy-restrictive dieting is characterized by an increased difficulty to lose weight and a facilitated ability to regain body weight.

All of these problems associated with very restricted dieting lead one to question the effectiveness of food intake restriction as a mode of treatment for obesity. Our first insights into alternative methods for obesity

![Figure 1](https://example.com/figure1.png)

**Figure 1**—Daily energy intake of lean and obese men and women. Values are means \(\pm\) SEM. *Significant difference between lean and obese (\(P < 0.05\)). Data are from Miller et al. (15).
treatment emerged as we summarized several of our animal studies dealing with diet composition and the development of obesity (Table 1). Note that the animals on the fat-rich/sugar-rich, fat-rich, and sugar-rich diets consumed the same or fewer joules than those animals on the control diet but were severely obese. Also note that, when the total energy intake of each group was divided by the grams of body fat deposited, the control animals could consume twice as many joules per gram of body fat as the fat-rich and/or sugar-rich groups. These data imply that decreasing energy intake by at least 50% may be necessary just to maintain a reduced body fat content if the diet is high in fat and/or sugar. This inference is supported by research demonstrating that obesity persists after a period of energy-reduced dieting when the composition of the diet remains high in fat (11,22).

**DIETARY FAT AND BODY FATNESS**

The data presented in Table 1 demonstrate that animals on a fat-rich and/or sugar-rich diet can become severely obese without overeating. Similar findings have been reported by other investigators (10,11,13). This implies that diet composition may be just as important as energy content in the promotion of obesity.

We have previously seen how the literature reports that normal-weight and overweight persons ingest about the same number of joules. If this is the case, then there must be some other factor(s) in the diet that disrupt the balance between energy intake and expenditure in order to favor body fat deposition. Dietary fat may be one such factor. We previously discussed a study where the relationships among diet composition, energy intake, and body fatness were examined in 216 adult men and women (15). Correlational analysis revealed that there was no relationship between daily energy intake and adiposity, but there was a significant correlation between dietary fat content and body fat content for both the males (r = 0.38, P < 0.001) and the females (r = 0.37, P < 0.001). In a second experiment where males and females were classified into subgroups of lean and obese, the obese derived a greater portion of their daily energy intake from fat when compared with the lean (15). Lean males and females consumed 28.7 ± 1.0% and 28.6 ± 1.5% of their joules, respectively, in the form of fat, compared with 33.1 ± 1.0% and 36.3 ± 1.5%, respectively, for the obese (Fig. 2). The relative energy contents of the diets were similar among all groups.

Dreon et al. (6) have found a significant correlation (r = 0.21, P < 0.01) between dietary fat content and percent body fat in middle-aged men. Analyzing further, they found that adiposity was significantly (P < 0.05) related to saturated fatty acid content of the diet (r = 0.20) and monosaturated fatty acid content (r = 0.19). There was no relationship between polyunsaturated fatty acid consumption and body fatness. Romieu et al. (23) reported similar results for middle-aged women. The Quetelet index (weight/height²) was significantly related to total fat intake (r = 0.20, P < 0.02) and saturated fatty acid intake (r = 0.16, P < 0.05) but not to polyunsaturated fatty acid intake.

The next logical step in our search for reducing dietary fat-induced obesity is to determine whether reducing the dietary fat intake of the obese will actually

![Figure 2](image)

**Figure 2**—Dietary fat and carbohydrate intake of lean and obese men and women. Values are means ± SEM. *Significant difference between lean and obese (P < 0.05). Data are from Miller et al. (15).

<table>
<thead>
<tr>
<th>Diet</th>
<th>Energy Intake (kJ·wk⁻¹)</th>
<th>Carcass Weight (g)</th>
<th>Body Weight (g)</th>
<th>% Body Fat</th>
<th>Total Intake (kJ·g⁻¹ body fat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2,512 ± 41</td>
<td>665 ± 20</td>
<td>216 ± 18</td>
<td>31 ± 2</td>
<td>670</td>
</tr>
<tr>
<td>Fat-rich/sugar-rich</td>
<td>2,520 ± 29</td>
<td>880 ± 16</td>
<td>452 ± 17</td>
<td>51 ± 1</td>
<td>335</td>
</tr>
<tr>
<td>Fat-rich</td>
<td>2,198 ± 71</td>
<td>806 ± 28</td>
<td>374 ± 28</td>
<td>46 ± 2</td>
<td>322</td>
</tr>
<tr>
<td>Sugar-rich</td>
<td>2,156 ± 59</td>
<td>780 ± 24</td>
<td>357 ± 21</td>
<td>45 ± 1</td>
<td>331</td>
</tr>
</tbody>
</table>

Values are means ± SEM. Control diet = 11% kJ from fat, 40% kJ from sugar. Fat-rich/sugar-rich diet = 35-45% kJ from fat, 35% kJ from fat, 55% kJ from sugar. Data are from Oscai et al. (18,19).
DIETARY CARBOHYDRATES AND BODY FATNESS

Our analysis of 216 men and women ranging in body fat content from 5.7 to 49.0% revealed that dietary carbohydrate intake was inversely related to body fatness (15). This relationship was significant \( (P < 0.001) \) for both males \( (r = -0.30) \) and females \( (r = -0.39) \). Group analyses among lean and obese subjects of each gender produced similar results (15). Lean men and women derived 52.6 \( \pm 1.3\% \) and 52.9 \( \pm 1.5\% \), respectively, of their energy from carbohydrates, as compared with 48.0 \( \pm 1.1\% \) and 44.3 \( \pm 1.7\% \) for obese men and women, respectively (Fig. 2). Obesity in middle-aged men was also characterized by a reduced carbohydrate intake (5); however, no correlation between carbohydrate consumption and adiposity was found in middle-aged women (23).

Carbohydrate intake, primarily in the form of refined sugar, has been implicated as a fat-producing agent. It is generally believed that, since refined sugar is virtually void of vitamins and minerals, any energy derived from refined sugar would be excess energy, contributing to body fat storage. Since the majority of evidence indicates either an inverse relationship or no relationship between body fatness and total carbohydrate intake, it seems unlikely that refined sugar would contribute significantly to increased body fat storage, especially if overweight individuals did not overeat, as the literature shows. Thus, the only way refined sugar could contribute substantially to the development of obesity would be either in the absence of hyperphagia or if hyperphagia were specific to refined sugar intake only. In other words, do obese people consume less total carbohydrates but more refined carbohydrates than lean people?

Data in Table 1 demonstrate that rats can become severely obese in the absence of hyperphagia by consuming a sugar-rich diet. Research on spontaneous food intake in humans, on the other hand, is more difficult to interpret than the animal research. One study found a significant negative relationship between sucrose intake and body weight (20), while another found no relationship between sucrose intake and percent body fat or body mass index (6). A third study reported an inverse relationship between sucrose consumption and body mass index (7). The results from the animal study cited previously (19) are easy to interpret and difficult to refute because all of the refined sugar in the diet was sucrose, which was accurately measured and controlled. In contrast, sucrose is only one of various refined sugars found in the American diet. In fact, more and more of the sugar added to our diet is refined fructose and corn syrup rather than sucrose. Thus, in order to determine the contribution of refined sugar to the development of obesity, we must examine the contribution of each individual sugar to body fat deposition. In addition, we will need to distinguish between sugars consumed in their natural form (e.g., corn syrup from sweet corn) and sugars consumed in their refined form (e.g., corn syrup added to soft drinks). It may be that obesity is characterized by excess consumption of “added” sugars, whereas leanness is characterized by high consumption of “natural” sugars. (Work in this area is in progress in our laboratory.)

An experiment more specific to natural carbohydrate consumption and adiposity was recently undertaken (8). Overweight women who were at least 20% overweight were placed on either an expanded-whole-wheat protein diet (4,186 \( \text{kJ} \cdot \text{d}^{-1} \)) or a traditional low-calorie diet (4,186 \( \text{kJ} \cdot \text{d}^{-1} \)) for 12 wk. The group of women consuming the whole-wheat product lost an average of 4.0 kg during the first 6 wk of the diet, compared with 2.9 kg lost on the traditional diet. During the second 6 wk, women on the whole-wheat product lost an additional 1.5 kg, whereas the traditional dieters lost only 0.4 kg. These data suggest that consumption of natural carbohydrate products, such as whole grains, assists in weight loss.

Dietary fiber consists of indigestible carbohydrates usually found in grain products, vegetables, and fruits. An experiment that shows how dietary fiber can assist in weight loss was reported from Denmark (24). In the first part of the experiment, overweight women were placed on either a 5,860 \( \text{kJ} \cdot \text{d}^{-1} \) diet or a 5,860 \( \text{kJ} \cdot \text{d}^{-1} \) diet with a fiber supplement of 5 g·d⁻¹. The group not receiving the fiber supplement was given a placebo. Both groups lost a significant amount of weight during the 8-wk diet \( (P < 0.01) \). However, the group receiving the fiber lost significantly \( (P < 0.05) \) more weight (7.0 kg) than the placebo group (6.0 kg). The investigation was repeated in the second part of the experiment,
except that the daily energy intake during a 12-wk period was increased to 6,598 kJ·d⁻¹ and the fiber supplement was increased to 7 g·d⁻¹. Once again, both groups lost a significant amount of weight (P < 0.001). The average weight loss in the fiber group was 6.2 kg, compared with 4.1 kg in the placebo group (P < 0.05).

In an earlier study (14), a high-fiber/low-calorie bread was used to assist college-aged males in losing weight. Sixteen overweight males were randomly assigned to either an enriched-white bread or a reduced-calorie bread group, unaware of their actual assignment. All subjects were instructed to reduce their total energy intake in order to lose weight. Following 8 wk of their respective diets, the reduced-calorie bread group lost 8.77 ± 0.88 kg, compared with 6.26 ± 0.44 kg for the enriched-white bread group (P < 0.05). These studies suggest that a high fiber intake, possibly through increased consumption of "natural" carbohydrates, assists in weight loss.

SUMMARY

It is generally accepted that adipose tissue accumulation is simply due to a disequilibrium in energy balance, resulting from energy intake exceeding energy expenditure. Treatment of obesity usually focuses upon reducing the total number of joules consumed in order to create a negative energy balance. However, there are other factors that shift the balance between energy intake and expenditure, favoring either body fat deposition or metabolism. The purpose of this paper was to examine how perturbation of some of these dietary factors that influence energy balance affects body fatness. The research presented supports the following conclusions:

1) obesity is not necessarily caused by overeating;
2) diet composition may be just as important as diet energy content in the promotion or reduction of obesity;
3) weight cycling through energy-restrictive dieting may lead to an increased difficulty to lose weight and a facilitated ability to regain body weight; and
4) optimal weight loss may be achieved through a combination of reducing dietary fat intake and increasing complex carbohydrate and fiber consumption, with minimal restrictions in total energy intake.

Address for correspondence: Dr. Wayne C. Miller, Department of Kinesiology, UPASS 295-H, Indiana University, Bloomington, IN 47405.

REFERENCES
