Effect of high-fat and low-fat diets on voluntary energy intake and substrate oxidation: studies in identical twins consuming diets matched for energy density, fiber, and palatability1-3

Edward Saltzman, Gerard E Dallal, and Susan B Roberts

ABSTRACT There remains controversy over the effects of dietary fat content on voluntary energy intake. Additionally, the question of whether there is a genetic susceptibility to overeating high-fat diets has not been resolved. To address these issues, we designed two diets: a low-fat diet providing 20% of energy as fat and a high-fat diet with 40% of energy as fat. The diets were matched for energy density, fiber, and palatability. In a two-phase, 18-d intervention study, voluntary energy intakes and macronutrient oxidation rates during the fasting and fed states were determined in seven pairs of identical male twins. In contrast with results of previous intervention studies, in which low-fat and high-fat diets were not matched for energy density and other associated variables, we observed no significant difference in voluntary energy intake between the low-fat and high-fat phases, and mean daily intakes were similar (10.3 and 10.7 MJ/d, respectively). Postprandial rates of fat oxidation tended to reflect fat intakes in the two dietary phases, thus helping to explain the lack of a difference in mean energy intakes. There was also a significant twin-pair similarity in differences in energy intakes between dietary phases (P = 0.013). These results suggest that dietary fat content does not have a major influence on voluntary energy intake when dietary variables usually associated with fat are controlled for and that there may be a familial influence on the effects of dietary fat content on energy intake. Am J Clin Nutr 1997;66:1332-9.

KEY WORDS Energy intake, fat, twins, fiber, energy density, macronutrient oxidation, glycogenostatic model of energy regulation

INTRODUCTION

The median body mass index (BMI) of adults in the United States is increasing (1) in tandem with parallel changes around the world (2). There remains controversy, however, concerning the underlying causes of this problematic trend. Of all the dietary variables that have the potential to influence body fatness, dietary fat has received particular attention. To date, however, the role of dietary fat in energy regulation has not been resolved.

Several types of studies suggested a positive effect of dietary fat content on energy intake and body fat mass, thus supporting the suggestion that a high intake of dietary fat promotes weight gain and obesity. In particular, cross-sectional studies linking body mass to dietary fat independently of energy intake (3-5), prospective studies linking dietary fat to future body weight change (6), and intervention studies that examined the effects of dietary fat content on energy intake directly (7-12) all observed positive associations between dietary fat content and energy intake, body fat mass, or both. These observations were consistent with the glycogenostatic model of energy regulation, which has as its central tenet the theory that body glycogen stores are regulated more tightly than are body fat stores (13, 14). The logical extension of this theory (13, 14) is that consumption of high-fat diets encourages hyperphagia because the resultant more rapid depletion of carbohydrate stores triggers the cascade of mechanisms leading to hunger.

However, there are also several lines of evidence against such a role for fat. In particular, although the median BMI and prevalence of overweight have increased in the United States during recent years (15), the reported percentage of energy supplied by fat in the national diet has decreased (16, 17). Additionally, comparisons of dietary fat intake and relative weight in different countries did not yield consistently positive associations and the associations that were positive were relatively weak (18). Further evidence against a role for dietary fat in the causation of obesity was also provided by the finding of no effect of dietary fat content on energy intake in short-term studies that emphasized careful control of psychologic variables and food palatability (19-22) or energy density (23-25). Finally, attempts to provide support for the glycogenostatic model of energy regulation by manipulating body glycogen stores and measuring subsequent energy intake were not successful (26, 27).

One possible explanation for the conflicting evidence on the effects of dietary fat is that dietary factors other than fat may be

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Received January 8, 1996.
Accepted for publication June 30, 1997.

important. Although some studies of dietary fat and voluntary energy intake controlled these variables, this has not universally been the case. High-fat diets usually differ from low-fat diets in a number of respects, although this is not inevitable; in particular, they tend to have a higher energy density and lower fiber content and they may be more palatable. Each of these other dietary factors possibly influences energy intake (28, 29) and so conclusions from previous studies that did not control all these variables may be spurious.

One dietary factor suggested as being particularly important is energy density (30). Three studies of the effects of energy density on energy intake found no effect of dietary fat content on voluntary energy intake when energy density was controlled (23–25). However, in each of these studies, the period of comparison was only 24 h and so uncertainty remains about the long-term effects of dietary fat content on energy intake independent of the effects of fat on dietary energy density.

This study was designed to test the hypothesis that dietary fat does not exert a long-term influence on energy intake when other dietary factors, including energy density, are controlled. The investigation enrolled pairs of monozygotic twins as subjects so that a possible familial influence on the susceptibility to overeating high-fat diets could also be examined.

SUBJECTS AND METHODS

Subjects

Seven pairs of male monozygotic twins aged 20–44 y who were recruited through twin registries participated in the study. Characteristics of the subjects are given in Table 1. All subjects reported having a stable weight (within 3 kg) over the 6 mo preceding admission and were healthy and free of important acute or chronic disease as assessed by physical examinations, routine laboratory tests, and electrocardiography. No subject smoked or took medications suspected of influencing energy regulation. The protocol was approved by the Human Investigations Review Committee of Tufts University and subjects provided written informed consent before participation. The study was conducted in the metabolic research unit (MRU) at the Jean Mayer US Department of Agriculture Human Nutrition Research Center on Aging at Tufts University.

Protocol

Subjects were admitted to the MRU for two 11-d periods separated by ≥ 4 wk. Only data collected during the first 9 d of each phase are reported here because procedures known to influence energy regulation were conducted during the last 2 d of each phase. During each dietary phase, subjects resided at the MRU and were encouraged to maintain their habitual levels of physical activity except for the 24 h before measurements of metabolic rate, when they avoided strenuous activity. The subjects were told that the purpose of the study was to investigate metabolic variables in identical twins and were blinded to manipulation of the study diets.

In a randomized crossover design, twin pairs were provided ad libitum with a low-fat diet during one of the 11-d periods and a high-fat diet during the other 11-d period. Voluntary energy intake was measured as described below. Subjects also underwent tests of energy expenditure, body composition, and metabolic variables during each dietary phase.

Diets

Two menus were developed for this study that differed primarily in the amount of energy supplied by fat and carbohydrate. Each menu consisted of 13 normal food items (Table 2). With the exception of one item (juice), the macronutrient content of each food item closely reflected that of the whole menu. The low-fat menu supplied ~20% of energy as fat and the high-fat menu supplied ~40% of energy as fat; carbohydrate was substituted for fat in the low-fat diet. As in previous dietary fat–density studies (11), different fat contents combined with comparable energy densities were achieved by varying the fat and water contents of the foods. In other respects, the diets were similar. Compositions of the diets were calculated by using standard dietary composition software (FOOD DATABASE version 7A and NUTRIENT DATABASE version 22; Minnesota Nutrition Data System, Nutrition Coordination Center, University of Minnesota, Minneapolis).

To ensure comparable palatability of the two menus, a 2-d pilot study was conducted in which the palatability of each food item was rated by 20 adults (employees of the Human Nutrition Research Center unconnected with this study). On each of the 2 d of the pilot study, palatability of half of the foods was assessed at 1100 after an overnight fast and consumption of the subjects’ usual breakfasts. Assessments were made by using standard 10-cm analogue scales anchored with the descriptions “extremely pleasant” (rating 10.0) and “extremely unpleasant” (rating 0.0). Palatability ratings for the two menus were not significantly different.

Twins were offered but were not required to consume three meals and two snacks per day. They were required to consume only food provided by the MRU and to eat in a communal dining area. They were not allowed to eat next to any other subject participating in the same study. By use of this requirement, we hoped to prevent twins from influencing each other’s dietary choices and energy intakes. As many different menu items as desired could be requested at each eating event, except for juice, of which only ≤ 200 g/d was allowed. Other food items were served in small units of 204–408 kJ each (with equivalent sizes and energy contents for comparable foods in the low-fat and high-fat menus) and multiple units of requested food items were served on a tray at each meal and snack. The number of units given always exceeded anticipated or previous consumption by some units and additional servings could be requested. At the end of each meal or snack, the total number of units of each item consumed was recorded. If a unit was only partly consumed, the remainder was weighed and the weight was recorded.

**TABLE 1**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>26 (20–44)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.7 (64.7–86.2)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179 (170–190)</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23.7 (20.3–29.5)</td>
</tr>
<tr>
<td>Body fat (% of weight)</td>
<td>16.4 (8.2–22.8)</td>
</tr>
<tr>
<td>Maximum oxygen consumption (mL · kg⁻¹ · min⁻¹)</td>
<td>44.9 (34.2–67.5)</td>
</tr>
</tbody>
</table>

*F; range in parentheses.*
TABLE 2
Menu items and composition for the high-fat and low-fat diets

<table>
<thead>
<tr>
<th>Menu items</th>
<th>Low-fat diet</th>
<th>High-fat diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oatmeal with milk (5.12)</td>
<td>Oat-bran cereal with milk (5.28)</td>
<td></td>
</tr>
<tr>
<td>Cold oat cereal with milk (5.98)</td>
<td>Granola with milk (5.98)</td>
<td></td>
</tr>
<tr>
<td>Bagel with spread (5.97)</td>
<td>Bagel with spread (6.10)</td>
<td></td>
</tr>
<tr>
<td>Tuna salad sandwich (6.95)</td>
<td>Chicken salad sandwich (6.99)</td>
<td></td>
</tr>
<tr>
<td>Chicken gumbo (2.31)</td>
<td>Potato and ham stew (2.32)</td>
<td></td>
</tr>
<tr>
<td>Pasta with sauce (2.34)</td>
<td>Chicken stew (2.40)</td>
<td></td>
</tr>
<tr>
<td>Turkey and rice casserole (2.39)</td>
<td>Vegetarian chili (2.44)</td>
<td></td>
</tr>
<tr>
<td>Macaroni salad (6.05)</td>
<td>Macaroni salad (6.09)</td>
<td></td>
</tr>
<tr>
<td>Garlic bread (11.32)</td>
<td>Garlic bread (11.79)</td>
<td></td>
</tr>
<tr>
<td>Cake with strawberries (6.13)</td>
<td>Cake with blueberries (6.45)</td>
<td></td>
</tr>
<tr>
<td>Ice cream and cake (6.60)</td>
<td>Ice cream sundae (6.63)</td>
<td></td>
</tr>
<tr>
<td>Orange and cranberry juices (1.88, 2.42)</td>
<td>Orange and cranberry juices (1.88, 2.42)</td>
<td></td>
</tr>
<tr>
<td>Milk, as a beverage (1.60)</td>
<td>Milk, as a beverage (1.86)</td>
<td></td>
</tr>
</tbody>
</table>

Diet composition

- Fat (% of energy)                 | 20.0 ± 0.2*                         | 40.2 ± 0.3*                  |
- Carbohydrate (% of energy)        | 63.8 ± 2.8                           | 45.6 ± 2.7*                  |
- Protein (% of energy)             | 16.2 ± 2.9                           | 14.1 ± 2.7                   |
- Energy density (kJ/g)             | 5.23 ± 0.79                          | 5.36 ± 0.81                  |
- Fiber (g/MJ)                      | 2.38 ± 0.74                          | 2.30 ± 0.68                  |
- Palatability, pilot study (mm)    | 5.9 ± 0.2                            | 5.9 ± 0.3                    |

* Energy density (kJ/g) in parentheses.
* Excluding juice, which was identical in the two diets.
* SEM; n = 12 foods.
* Significantly different from low-fat diet, P < 0.001.

To monitor changes in subjective palatability of the menus over the course of each dietary phase, subjects were required to taste and rate each food within that study phase on days 1, 5, and 9. Ratings were done just before usual meal times and, as in the pilot study, the palatability of each food was determined by using a standard 10-cm analogue scale.

Substrate oxidation

Substrate oxidation in the fasting and fed states was measured on days 2, 4, and 9 of each dietary phase by using a ventilated-hood indirect calorimeter (Deltatrac TM; Sensor-Medics Corporation, Anaheim, CA) calibrated at regular intervals with mixed test gases of known concentrations. For these measurements, each subject fasted overnight for 10 h, awakened at 0600, voided all urine, and then rested in bed for 30 min. Oxygen uptake and carbon dioxide production at rest were measured under thermoneutral conditions while subjects rested quietly in bed without fidgeting or sleeping.

After this measurement, a liquid test beverage was consumed that contained 2508 kJ and had the same proportions of energy supplied by each macronutrient as the menu for that study phase. Calorimetry was resumed 10 min after the start of the meal and continued for 4 h (with alternating 20-min measurement periods and 10-min breaks) while subjects continued to rest supine. Urine was collected throughout the calorimetry measurement and analyzed for nitrogen (31). Fat, protein, and carbohydrate oxidation was calculated from the data on respiratory gas exchange and urinary nitrogen output by using the coefficients of Livesey and Elia (32).

Maximal aerobic capacity

Maximal aerobic capacity was determined once in each subject 2–3 h after breakfast. Subjects exercised until exhaustion on an electrically braked cycle ergometer at 70 rpm with increasingly high workloads, according to a standard Bruce protocol (33, 34).

Body composition

Hydrostatic weighing was done while subjects were fasting. Measurements were repeated until at least four were within 1% body fat of each other, according to our usual procedure, and fat-free mass was calculated by using the equations of Siri (35).

Statistics

Data are expressed as means ± SEMs unless otherwise specified, with SEMs based on the variability among twin-pair means where specified. Comparisons between dietary phases and the effect of twin-pair membership on differences between phases were made by using repeated-measures analysis of variance (ANOVA) with between-subject factors of study day and dietary phase. Twin-pair membership was treated as a random factor when forming the F ratio test statistics. Because of the complexity of the design, the possibility of an order effect was examined in the table of adjusted means for each twin pair and dietary phase. The association between the phase difference in energy intake and other variables (body fat and phase differences in diet palatability and fat oxidation) was assessed by applying Student’s t test to regression coefficients obtained from each twin pair.

RESULTS

Effects of low-fat and high-fat diets

Energy intakes of subjects in each dietary phase are shown in Figure 1. In the two-way ANOVA, there was no significant
difference in energy intake between phases for either the mean values for 9 d or for the last 4 d (when there appeared to be a slight divergence between phases). Additionally, there was no significant difference between phases in the change in energy intake over time and no significant order effect (ie, which diet was consumed during the first 9-d period and which was consumed during the second 9-d period). Mean intakes were 10.3 ± 0.2 MJ/d during the low-fat phase and 10.7 ± 0.3 MJ/d during the high-fat phase (NS).

Subjects were able to choose which foods they wanted to consume from the menus in each phase. There was a significant linear relation between palatability ratings for each food (average values from study days 1, 5, and 9) and voluntary energy intake of that food, as shown in Figure 2 (Spearman correlation coefficient = 0.72, \( P < 0.01 \)), and variability in the relation increased with palatability rating. The ranges of subjects’ palatability ratings in the low-fat and high-fat phases were comparable. There was also no significant difference in overall palatability ratings between dietary phases. Palatability ratings weighted for energy of each food item were 8.3 ± 0.2 mm for the low-fat menu and 8.6 ± 0.3 mm for the high-fat menu.

Fasting and postprandial rates of fat and carbohydrate oxidation are shown in Figure 3 and Figure 4, expressed as percentages of total energy expenditure during the measurement periods. There were no significant trends in proportions of fat and carbohydrate oxidized in either fasting or fed states over the course of each dietary phase. Additionally, there was no significant difference between dietary phases in fat oxidation in the fasting state. In contrast, fat oxidation during the fed

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**FIGURE 1.** Mean (± SEM) voluntary energy intake over 9 d of consumption of low-fat (●) and high-fat (■) diets.

**FIGURE 2.** Relation between mean palatability rating by analogue scale and mean energy intake per 9-d study phase in 14 subjects consuming low-fat (●) and high-fat (■) menu items.

**FIGURE 3.** Mean (± SEM) fat oxidation during the fasting and fed states on 3 d of 9-d dietary phases in which 14 subjects ate low-fat or high-fat diets. Fasting-state measurements in subjects who consumed low-fat (○) and high-fat (□) diets, respectively; fed-state measurements during low-fat (●) and high-fat (■) diets. Repeated-measures ANOVA found significant effects of diet type on fat oxidation during the fed state (\( P < 0.001 \)) but no significant effects of study day or twin group. None of these variables were significant for fasting measurements.

**FIGURE 4.** Means (± SEM) carbohydrate oxidation during the fasting and fed states on 3 d of 9-d dietary phases in which 14 subjects ate low-fat or high-fat diets. Fasting-state measurements in subjects who consumed low-fat (○) and high-fat (□) diets, respectively; fed-state measurements during low-fat (●) and high-fat (■) diets. Repeated-measures ANOVA found significant effects of diet type on carbohydrate oxidation during the fed state (\( P < 0.004 \)) but no significant effect of study day or twin group. None of these variables were significant for fasting measurements.
state was significantly higher during the high-fat dietary phase than during the low-fat dietary phase ($P = 0.001$); the mean difference in energy expenditure was 13.2% (compared with a 19.9% difference in fat proportions in the two test meals and dietary phases). Similarly, carbohydrate oxidation in the fasting state was not different in the low-fat and high-fat phases but postprandial values were significantly higher in the low-fat phase than in the high-fat phase ($P = 0.004$) and again tended to reflect the proportion of carbohydrate in the test meal and dietary phase.

**Twin-pair effects**

Although there was no significant difference in energy intake between low-fat and high-fat phases, there were significant differences among twin pairs ($P = 0.013$) and individual twin pairs had notably different energy intake responses. As shown in Figure 5, three twin pairs consumed more energy in the high-fat phase than in the low-fat phase, three pairs consumed more in the low-fat phase than in the high-fat phase, and one pair consumed very similar amounts in both phases. It should be noted that this similarity within twin pairs was found only for 9-d mean phase intakes. Day-to-day energy intake patterns were not similar within twin pairs, except for one pair. This suggests that our experimental requirement of separating twins at meals was successful in preventing twin pairs from influencing each other with regard to their daily energy intakes. The removal from the ANOVA of the one twin pair who did have day-to-day similarities in energy intake did not negate the importance of the twin effect on the difference in energy intake between the low-fat and high-fat diets.

To explore possible explanations for the apparent twin-pair influence on differential consumption of low-fat and high-fat diets, we plotted differences in energy intake between phases against body composition (Figure 6), phase differences in palatability (Figure 7), and lipid oxidation in the fed state (Figure 8). Within twin pairs, there was a significant relation between proportion of body fat and difference in energy intake between low-fat and high-fat phases, with fatter twins tending to consume more energy during the low-fat diet than during the high-fat diet ($P = 0.038$). Concerning palatability ratings (which were determined by using a weighting procedure to account for the relative proportion of energy derived from each menu item), there was a tendency for differences in palatability ratings between twin pairs to be reflected in phase differences in energy intake, although the difference was not significant. Finally, there was no twin-pair effect on differences in fat oxidation between phases and no relation between differences within twin pairs in fat oxidation and differences in energy intake.
DISCUSSION

This study addressed two related issues: the effects of dietary fat on voluntary energy intake and familial effects on the propensity to overeat high-fat diets. In contrast with several previous cross-sectional, prospective, and intervention studies (3, 4, 6–11), we found no significant difference in voluntary energy intake between separate 9-d periods during which low-fat and high-fat diets were consumed. Furthermore, although the small mean increment in energy intake during the high-fat diet (4%) could possibly lead to weight gain if it persisted, it was only one-fifth to one-third of mean increments reported in previous intervention studies in which energy density was not controlled (7–11). Instead, the increment was more consistent with values in short-term studies in which energy density was matched between diets with different fat contents (23–25). This consistency between short-term and more long-term studies of the effects of dietary fat content on voluntary energy intake when energy density is controlled is important because it suggests strongly that energy density may be a more important determinant of energy intake than is dietary fat in both day-to-day variability in food intake as well as in long-term control of food intake.

There are important practical issues relating to the finding that consumption of lower-fat diets does not reduce energy intake when separated from the usual coexisting dietary conditions of low energy density, high fiber content, and perhaps low palatability (relative to higher-fat diets). In particular, the previous emphasis on dietary fat as a determinant of hyperphagia has encouraged the view that low-fat commercial products—even extremely palatable low-fat products that do not have low energy density or a high fiber content—help to reduce energy intake. However, if dietary fat by itself is not a major determinant of voluntary energy intake, as our results suggested, commercial products that separate dietary fat from dietary variables usually associated with fat can be expected to be ineffective in preventing hyperphagia.

This suggestion is consistent with the observation that the prevalence of obesity increased in the US population during a time in which commercial products specifically designed to be low in fat but high in palatability became more available (1). On the basis of observations in this study and previous investigations (23–25, 30), it seems appropriate to suggest that high dietary fat per se may not be a primary cause of positive energy balance and obesity but that, instead, dietary fat promotes obesity indirectly through its effects on energy density. More research is needed to determine whether dietary fat really has any effect on voluntary energy intake distinct from other dietary variables usually associated with fat. This is particularly important because the mean increment in energy intake in our study during consumption of the high-fat diet (4%) was not significant although, if this difference were real, it could possibly cause significant weight gain over the long term.

In parallel with the finding of no effect of dietary fat content on voluntary energy intake when energy density and diet palatability were controlled, we found that subjects in this study had postprandial fat and carbohydrate oxidation values that tended to reflect the macronutrient ratios in their diets. Thus, postprandial fat oxidation was higher in the high-fat phase than in the low-fat phase when test meals of equal energy content but different fat contents were consumed. This was observed on all study days on which substrate oxidation was measured (days 2, 5, and 9 of each dietary phase), indicating that the switch between fat and carbohydrate oxidation was rapid. This finding is consistent with those of recent studies that reported a similar observation (36, 37) and with the recognized phenomenon that high concentrations of circulating lipids increase fat oxidation (38).

The other question addressed in this study was whether there is a measurable familial influence on the propensity to overeat high-fat diets, an area that is currently controversial. A recent prospective epidemiologic study of 361 women (39) indicated that not all individuals gain weight when consuming high-fat diets and that those who do so have other overweight individuals in their family, a finding that suggests a familial association. In contrast with this suggestion, other studies found no significant genetic influence on the proportion of dietary energy supplied by fat (40) and inconsistent results were reported in investigations of familial and genetic influences on food preferences (41–43).

In the intervention study reported here, we studied pairs of identical male twins to assess whether there is a familial influence on the extent to which energy intake is affected by fat content of consumed foods. We hypothesized that familial similarities in energy-intake responses to diets with different fat contents would be observed. A striking twin-pair similarity in energy intake response to high-fat compared with low-fat diets was indeed observed, despite comparable levels of physical activity in the two dietary phases. Three of the twin pairs had consistently higher energy intakes in the high-fat phase than in the low-fat phase, three had consistently lower energy intakes in the high-fat phase than in the low-fat phase, and one twin pair had intakes that were very similar in the two phases. This finding is consistent with the suggestion of a familial and probably genetic influence on voluntary energy intake response to diets with different fat contents.
In searching for possible explanations for the twin effect on energy intake responses to high-fat compared with low-fat diets, we considered body composition, twin similarities in perceived diet palatability, and the ability to reach fat oxidation to fat intake during the two diets as possible candidate mechanisms. Contrary to expectations, there was a significant association within twin pairs between body fat and the tendency to overeat low-fat foods relative to high-fat foods. Additionally, even though diet palatability was significantly related to energy intake in the study as a whole, individual and twin-pair differences in palatability ratings between high-fat and low-fat diets were not significantly related to differences in energy intake.

Similarly, we did not observe a significant relation between phase differences in energy intake and fat oxidation. Thus, twin pairs with a tendency to overeat the high-fat diet relative to the low-fat diet did not have the smallest differences in fat oxidation between high-fat and low-fat phases, as would be predicted by the glycogenostatic hypothesis of food intake regulation (because low fat oxidation would be linked to elevated carbohydrate oxidation and depletion of carbohydrate stores) (14). This indirect evidence that overeating high-fat diets relative to low-fat diets is not linked to depressed fat oxidation is consistent with results of studies that found no effect or a negligible effect of experimental alterations in body glycogen stores on subsequent voluntary energy intake (11, 26, 27).

Further studies are needed both to confirm the findings of twin-pair similarities in the extent to which dietary fat content influences energy intake and to address the mechanisms underlying this tendency. Ideally, such studies should enroll twins who are separated from each other completely, an approach not possible in this study.

In conclusion, in this 9-d study that separated dietary fat content from other dietary variables usually linked to fat content, including energy density, palatability, and fiber content, we observed no significant difference in voluntary energy intake between diets with ~20% of energy from fat and those with ~40% of energy from fat. Our results are thus contrary to the widespread belief that fat per se is the dominant factor influencing voluntary energy intake. Instead, they are consistent with previous experimental work that suggested that dietary fat content has no short-term effect on energy intake when energy density is controlled (23–25).

We thank our dedicated volunteers, without whom this study would not have been possible. We are also extremely grateful for the expert technical assistance of the staff of the metabolic research unit and for the assistance of the National Organization of Mothers of Twins, T Bouchard, M Neale, and D Allison in recruiting subjects.

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