Fat intake and adiposity in children of lean and obese parents

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ABSTRACT  We examined the relations between obesity in parents and fat intake in their children, and the effect of fat intake on fat mass in these children. Our heterogeneous sample (± SD: 20.2 ± 3.4 kg; 5.2 ± 1.3 kg fat mass) consisted of 56 white and 15 Mohawk children 4–7 y of age (35 girls and 36 boys). Dietary intake was assessed with the Willett food-frequency questionnaire revised for children. Body composition was measured by bioelectrical resistance and subscapular and triceps skinfold thicknesses. Physical-activity energy expenditure was estimated by the difference between total energy expenditure (measured over 14 d by the doubly labeled water method) and postprandial resting energy expenditure (measured by indirect calorimetry). Before statistical analysis, fat mass was adjusted for fat-free mass, and fat intake was adjusted for nonfat intake. There was no effect of sex or ethnicity on fat intake, and no effect of ethnicity on the relation between fat intake and fat mass. Adjusted mean (± SE) fat intakes for the groups of children, based on parental obesity status, were as follows: 2.65 ± 0.09 MJ/d (nonobese mother and father), 2.85 ± 0.12 MJ/d (obese mother, nonobese father), 2.58 ± 0.10 MJ/d (obese father, nonobese mother), and 2.79 ± 0.10 MJ/d (obese mother and father). We found an influence of maternal obesity on dietary fat intake in children (P = 0.052) and a significant correlation between fat mass and fat intake in boys (r = 0.48, P < 0.01) but not in girls after adjustment for physical-activity energy expenditure. Our data suggest that 1) mothers may contribute to the development of obesity in children by influencing their dietary fat intake, and 2) dietary fat intake contributes to obesity in boys, independent of physical-activity energy expenditure. 


KEY WORDS  Obesity, children, parental influence, dietary fat, body composition

INTRODUCTION

Data from the National Health Examination Surveys from 1963 to 1965 compared with data from the National Health and Nutrition Examination Surveys from 1976 to 1980 indicate that pediatric obesity was considerably higher in the period from 1976 to 1980, with a 54% increase in children 6–11 y of age (1). This increase may be due, in large part, to environmental factors (1). Recent evidence from a study in 3416 schoolchildren in Birmingham, AL, indicates that the prevalence of pediatric obesity remains elevated. Thirteen percent of white girls were obese by age 5 y and 29% were obese by age 10 y; 10% of white boys were obese at age 5 y and 22% were obese by age 10 y (2). Pediatric obesity is a critical health concern because obesity in childhood increases the risk for adult obesity and numerous associated diseases (3). Approximately 26–41% of obese preschool children are obese as adults (4). Obesity status in the biological parents is a good predictor of the development of obesity in children (5, 6). The probability of a child becoming obese has been estimated to be 7% if neither parent is obese, 40% if one parent is obese, and 80% if both parents are obese (5).

The diet is considered to be an important variable in controlling weight status, but findings with respect to dietary intake are inconclusive. Because studies in adults (7–9) and children (10–14) have failed to find a significant relation between total energy intake and adiposity, focus has turned to diet composition. Flatt (15) proposes that fat oxidation occurs as a function of the difference between total energy expenditure and the oxidation of protein and carbohydrate. Studies in humans have confirmed that oxidation of protein and carbohydrate is similar to their intake, whereas fat oxidation does not parallel fat intake (16, 17). Dietary fat intake is therefore hypothesized to play a key role in the long-term regulation of energy balance and the development of obesity (15).

Because an effect of dietary fat intake on body fat has been shown in many studies of adults (18), the high prevalence of obesity in America can be explained in part by a high consumption of dietary fat. However, we identified only a few studies examining the influence of dietary fat intake on body composition in children (19–21). More importantly, dietary intake patterns within the family may contribute to the development of obesity in families with children who have obese parents. Eck et al (22) reported that children of obese parents consumed a higher percentage of energy from fat (34.4% compared with 32.1%) and less from carbohydrate (51.7%)


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compared with 55.0%) than did children with no obese parent. Because children of obese parents are at higher risk of developing obesity than are children of nonobese parents, and because dietary fat intake significantly contributes to body fat in adults, it is important to understand the role of diet composition in the pathogenesis of obesity. Thus, the objectives of this study were twofold: 1) to investigate the association between the obesity status of parents and dietary fat intake in their offspring, and 2) to determine the relation between dietary fat intake and body fat in children, taking into the account the potential independent effects of physical-activity energy expenditure (AEE).

SUBJECTS AND METHODS

Subjects

The subjects in this study were 65 white and 15 Mohawk children (4–7 y of age) from 52 white and 13 Mohawk families. Families were recruited by newspaper advertisement and word of mouth and resided in Burlington, VT, or Akwesasne, NY. Children were recruited and divided into four groups based on their parents’ obesity status: 1) nonobese mother and father (n = 22); 2) obese mother, nonobese father (n = 13); 3) nonobese mother, obese father (n = 20); and 4) obese mother and father (n = 16). Parents were defined as being obese if their body mass index (BMI) was above the 85th percentile for age and sex, and nonobese if their BMI was below the 85th percentile for age and sex (23). We previously reported data on energy expenditure for these children (24, 25). The study was approved by the Committee on Human Research for the Medical Sciences at the University of Vermont.

Macronutrient intake

Macronutrient intake was measured by administration of the Willett 111-item food-frequency questionnaire (FFQ) for children. This questionnaire is similar to the FFQ for adults, except that it does not contain questions about alcohol intake (26). Because use of this questionnaire in children over a 1-y period produces results comparable with those of an FFQ administered multiple times throughout the year (r = 0.70, P < 0.001), it was used as a substitute in measuring intake over this extended time (27). The dietary intake of children, based on parental response to the FFQ, has been found to be reasonably reliable (r = 0.67) for all nutrients (28). Foods mentioned in the FFQ are divided into eight groups: dairy, fruits, vegetables, meats, sweets/baked goods, bread/cereals/starches, carbonated beverages, and miscellaneous. Nine frequency choices for consumption ranging from never or less than once per month up to six times or more per day are listed with spaces available for foods consumed but not included on the questionnaire, i.e., uncommon foods or ethnic foods. Responses to the FFQ were analyzed by the Channing Laboratory at the Harvard School of Public Health, Boston.

Parents were instructed on how to complete the questionnaire and informed that intake was to represent the child’s eating habits over a 1-y period. The questionnaire was returned the following day or 2 wk later when the subjects reported back to the laboratory. Responses to the questionnaire were completed primarily by the child’s mother, with occasional input from the child’s father and/or the child. Missing responses were obtained either by phone or by direct conversation with the mother.

Body composition and anthropometry

Body composition was measured in the children by using bioelectrical resistance and skinfold-thickness measurements. Whole-body resistance was measured in duplicate 14 d apart with an RIL bioelectric analyzer (Mi Clemens, MI) with electrodes placed at sites recommended by the manufacturer. The system was calibrated before each test. Skinfold thickness was measured in the children at eight sites (axilla, chest, abdomen, triceps, calf, subscapula, suprailiac, and thigh). All skinfold-thickness measurements were taken in triplicate by the same research assistant and with the same calipers (Lange, Cambridge, MD) according to the procedures of Lohman et al (29). Height was measured to the nearest 0.5 cm with a wall-mounted ruler and weight to the nearest 0.1 kg on an electronic scale before bioelectrical resistance measurements. Body fat was estimated from subscapular and triceps skinfold thicknesses, weight, height²/resistance, and sex by using an equation developed for children, and using dual-energy X-ray absorptiometry (DXA) as a criterion method (R² = 0.91) (30).

Energy expenditure

Total energy expenditure was measured over 14 d by using the doubly labeled water method as described previously (31). A baseline urine sample was taken before the oral dose of 0.15 g H₂¹⁸O and 0.12 g ²H₂O/kg body mass was administered. Two urine samples were collected the morning after dosing and two samples in the morning 14 d later. Samples were analyzed in triplicate for H₂¹⁸O and ²H₂O by isotope-ratio mass spectrometry at the Biomedical Mass Spectrometry Facility, Clinical Research Center, University of Vermont. Carbon dioxide production was derived from the equation of Speakman et al (32) and converted to total energy expenditure by using the equation of de Weir (33).

Resting energy expenditure was measured postprandially in duplicate 14 d apart by indirect calorimetry with a Deltatrac metabolic monitor (Sensormedics Corp, Yorba Linda, CA) as described previously (31). Postprandial resting energy expenditure rather than the typical 12-h postabsorptive energy expenditure was measured because, from our experience, young children are more relaxed in this environment than they are after an overnight fast; therefore, an accurate measurement is more likely. We have shown that repeat measurement of resting energy expenditure in the postprandial state is reliable (intraclass correlation coefficient = 0.91) for measurements 14 d apart (31), and 11% higher than when measured under 12-h postabsorptive conditions (34). Thus, the additional cost of meal-induced thermogenesis was included in our measurements of resting energy expenditure by design. AEE (MJ/d) was estimated as the difference between total energy expenditure and postprandial resting energy expenditure.

Statistical analyses

Data are presented as means ± SDs or as means ± SEs. In the preliminary analysis, fat intake was adjusted for nonfat intake (i.e., protein plus carbohydrate) and fat mass was adjusted for fat-free mass by using linear-regression models. Data were screened and values removed if they were considered to be
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RESULTS

Data from nine subjects were considered outliers, on the basis of the criterion previously stated, and thus were excluded from all analyses. There were complete data for 71 children and their subject characteristics are shown in Table 1. All subject characteristics were similar by sex and ethnic groups, except fat mass, which was greater in females than in males after adjustment for fat-free mass ($P < 0.01$). There was no effect of sex or ethnic group on dietary fat intake adjusted for nonfat intake; therefore, the data from both sex and ethnic groups were pooled for the first objective and separated by sex for the second objective.

### Objective 1

Results of the two-way ANCOVA assessing the effect of parental obesity on adjusted fat intake in the four groups of children were as follows ($\bar{x} \pm SE$): 2.65 ± 0.09 MJ/d (nonobese mother and father), 2.85 ± 0.12 MJ/d (obese mother, nonobese father), 2.58 ± 0.10 MJ/d (obese father, nonobese mother), and 2.79 ± 0.10 MJ/d (obese mother and father). A main effect for maternal obesity was nearly significant ($P = 0.052$), but no significant group effect was found in post hoc analysis. There was a trend toward significance between the group with an obese mother and nonobese father and the group with an obese father and nonobese mother ($P = 0.078$).

### Objective 2

The results of the multiple-regression model assessing the association between fat mass and fat intake in all children and by sex are shown in Table 2. After adjustment for variability due to fat-free mass, fat intake, nonfat intake, and AEE, a significant relation remained between fat mass and fat intake in boys ($r = 0.48$, $P < 0.01$) but not in girls. These relations are shown separately for girls and boys in Figure 1. There was no difference between white and Mohawk children for the association between fat mass and fat intake.

### DISCUSSION

Our aim was to examine the relation between obesity in parents and dietary fat intake in their children, and the effect of dietary fat intake on fat mass in children. We observed the following major findings: 1) obesity in mothers tends to positively influence fat intake in their children ($P = 0.052$), and 2) fat intake was positively and significantly correlated with body

### TABLE 2

Parameter estimates for the multiple-regression model assessing the association between fat mass (dependent variable) and indicated independent variables.

<table>
<thead>
<tr>
<th>All children ($R^2 = 0.47$)</th>
<th>Girls ($R^2 = 0.43$)</th>
<th>Boys ($R^2 = 0.69$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept (kg)</td>
<td>-3.0</td>
<td>-1.70</td>
</tr>
<tr>
<td>Fat-free mass (kg)</td>
<td>0.35</td>
<td>0.31</td>
</tr>
<tr>
<td>Dietary fat intake (MJ)</td>
<td>0.0027</td>
<td>0.0002</td>
</tr>
<tr>
<td>Nonfat intake (MJ)</td>
<td>-0.0009</td>
<td>0.00003</td>
</tr>
<tr>
<td>AEE (MJ)</td>
<td>-0.0005</td>
<td>0.00008</td>
</tr>
<tr>
<td>Parameter estimates</td>
<td>SE</td>
<td>$P$</td>
</tr>
<tr>
<td>0.87</td>
<td>&lt;0.0001</td>
<td>1.16</td>
</tr>
<tr>
<td>0.05</td>
<td>&lt;0.0001</td>
<td>0.07</td>
</tr>
<tr>
<td>0.0011</td>
<td>&lt;0.05</td>
<td>0.91</td>
</tr>
<tr>
<td>0.0005</td>
<td>0.53</td>
<td>0.0008</td>
</tr>
</tbody>
</table>

1 Fat-free mass and fat mass were calculated from skinfold-thickness measurements and bioelectrical resistance. TEE, total energy expenditure measured over 14 d by the doubly labeled water method; REE, postprandial resting energy expenditure; AEE, physical-activity energy expenditure calculated from the difference between TEE and REE.

2 Significantly different from boys, $P < 0.01$. AEE, physical-activity energy expenditure.
fat in boys \( (r = 0.48, P < 0.01) \) but not in girls, after adjustment for AEE.

**Objective 1**

The first observation that obesity in mothers influences fat intake in their children \( (P = 0.052) \) supports previous findings. Eck et al (22) also found that children with obese mothers and normal-weight fathers consumed a higher percentage of energy in the diet from fat than did children with no overweight parent \( (P < 0.05) \). Although we found a main effect of paternal influence on fat intake in children, we were unable to detect a significant difference between groups by maternal and paternal obesity. The difference between fat intake in the group with an obese mother (nonobese father) and the group with an obese father (nonobese mother) was nearly significant \( (P = 0.078) \). The absence of a group effect may be due to the small sample size in the groups with obese mothers or to the marginally significant main effect of maternal influence on fat intake in children.

On the basis of two-way analysis of variance, our data suggest that the parental influence of obesity on dietary fat intake in children is likely to be associated with maternal rather than paternal factors. The lack of effect of the father’s obesity on his children’s fat intake is consistent with the findings of Oliveria et al (35), who determined using 3-d food records that the diets of mothers and their children were more similar than were the diets of fathers and their children. Furthermore, a positive correlation with fat intake was noted in mother-child relations \( (r = 0.46, P < 0.001) \) but not in father-child relations. Other researchers (14) found, by direct observation, that mothers serve larger portions of food to their obese children than to their nonobese children, suggesting that the mothers’ behavior may aid in perpetuating weight gain in their children.

One limitation of studies examining children’s intake is the possibility of recall bias. Because mothers usually report their children’s intake, the mothers’ description of their children’s intake may be influenced by their own dietary habits and biases. However, it has been reported that mothers are somewhat more accurate in reporting their children’s fat intake than are fathers (36). In addition, mother-child interactions have been found to be related to the child’s weight (37), and mothers’ (not fathers’) child-feeding practices are highly correlated with their child’s ability to regulate intake (38). The recent findings of Fisher and Birch (20) support our hypotheses that parents influence their children’s fat intake, and dietary fat intake contributes to obesity. They found that children with a greater preference for dietary fat consumed more fat in the diet \( (r = 0.54, P < 0.05) \), had higher skinfold-thickness measurements \( (r = 0.61, P < 0.01) \), and had parents with higher BMIs \( (r = 0.67, P < 0.01) \) than did children with less of a preference for fat.

We were surprised to find that the interaction of obesity in both parents did not affect dietary fat intake in their children because children with two obese parents are at greater risk of becoming obese than are children with one or no obese parent (5). A possible explanation for the lack of interaction effect may be that whereas the fat content of the diet promotes the expression of obesity, dietary fat may have a greater effect in children with a genetic predisposition to obesity, i.e., children who have at least one obese parent. Specifically, children with two obese parents may consume an amount of fat similar to that consumed by children with one or no obese parent, but the fat content may have a more potent effect on their body composition. This hypothesis is supported by cross-sectional studies with previously obese women, who were found to have a reduced ability to increase fat oxidation in response to high fat intakes compared with nonobese control subjects (39, 40). Although these studies have limitations in their application to children, they indicate that there are individuals who may be more sensitive than others to the effects of a high-fat diet. In addition, recently published results from a 12-y prospective study of 361 women revealed that a higher dietary fat intake was associated with a greater BMI \( (P = 0.003) \) only in women who were predisposed to obesity (defined as those with a family history of obesity and currently obese)(41).

Evidence from an animal study by Lim et al (42) also indicates that a high dietary fat intake may contribute to obesity within families. Offspring of rats fed a high-fat (40%) diet accumulated significantly more body fat than did offspring of rats fed a low-fat (5%) diet \( (P < 0.001) \) even when the offspring were fed an isonenergetic medium-fat diet (22.5% of energy as fat, 52.5% of energy as carbohydrate, and 25% of energy as protein). This effect was seen in offspring beginning with the third generation, and the outcome was affected by neither the mother’s milk nor diet during the pregnancy period. The results of this study suggest that dietary fat intake over generations influences energy metabolism and the subsequent increase in adipose tissue deposition that occurs in response to a high-fat diet. Moreover, these data support the hypothesis that dietary fat contributes to familial aggregation of obesity. Further longitudinal studies in children are needed to examine individual susceptibility to dietary fat content.

**Objective 2**

Our second major finding was the significant positive correlation between fat mass and fat intake in boys but not in girls. When we accounted for AEE, the correlation between fat
intake and fat mass remained significant in boys ($r = 0.48$, $P < 0.01$). Although AEE did not significantly affect body fat in the children in our study, others have shown the opposite to be true (43, 44). It is recognized that children are not as physically active as they were in previous decades and, at the same time, are more obese (1).

The association between fat mass and fat intake in our study corresponds with earlier studies in adults (7–9, 18) and children (19, 20), though most have shown a relation in both sexes. Consistent with previous studies (19, 44), fat mass in girls was higher than in boys in our study. Yet this greater fat mass in girls could not be attributed to a higher fat intake. The absence of a relation between fat mass and dietary fat intake in girls in our study was most likely complicated by the presence of maternal bias when mothers report their daughter’s intake. Although studies show that obese individuals underreport intake compared with nonobese individuals (45), and women underreport compared with men (46), little is known about parents’ under- or overreporting of their children’s intake. We showed previously that there is no relation between mothers’ or children’s body composition and the estimation of intake by the FFQ (47). Klesges et al (48) found that neither paternal nor maternal weight status affected the accuracy with which parents recalled their children’s intake. Unfortunately, the question remains as to whether sex-specific differences exist when parents report their children’s intake and, more specifically, if the mother’s or daughter’s weight status alters this estimation. We speculate that differences may exist. Mothers with obese daughters may be underreporting their daughter’s fat consumption because weight appears to be a greater concern for women than for men (49). In the aforementioned study by Oliveria et al (35), mother-child correlations for nutrient intake were higher than father-child correlations, whereas most mother-daughter correlations were even stronger than mother-son correlations. The difference in the association between the diets of boys and their mothers and girls and their mothers suggests that girls may be more influenced by their mother’s dietary patterns and behavior than are boys. In fact, the high prevalence of dietary behavior in women compared with men may be due, to some extent, to learned behavior in childhood (49). Maloney et al (50) found that in elementary school-aged children, 41% of girls reported that they had attempted to lose weight, whereas 31% of boys reported weight-loss attempts. Moreover, it has been noted that mothers who are more restrictive of their own intake may have daughters who are less able to self-regulate intake ($r = -0.37$, $P < 0.08$) (38).

A 3-y longitudinal study in adults by Klesges et al (51) examining the influences of fat intake and physical activity on weight status also revealed a difference between sexes. The consistent cross-sectional predictor of men’s increase in body mass was fat intake, whereas in women it was fat intake and familial obesity. The authors recognized the possibility that the variations between sexes may be attributed to differences in reporting between females and males, or to inherent sex differences. Additional studies are needed to evaluate the possible differences that exist between sexes, particularly in children, and their influence on body composition. Further studies examining the influence of parental bias on reports of their children’s intake are also warranted because the parents’ report of their children’s diet may not be a completely accurate representation of their child’s intake. Future studies examining the effect of parental influence on their children’s diets and its relation to adiposity should also consider parental socioeconomic status. Although we are unaware of any studies that suggest that parental socioeconomic status may influence macronutrient intake in their offspring, socioeconomic status is a potentially confounding factor that deserves exploration.

Thus far, most studies examining the association between fat mass and dietary fat intake rely on standard dietary intake techniques. As discussed, discrepancies may occur in these studies because measurements of intake are dependent on subject recall and report. With this limitation in mind, we attempted to control for inaccuracies inherent in the FFQ technique used in our study. The FFQ has been shown to overestimate energy intake in children (47), but this overestimation is likely due to the use of adult (47) and fixed (52) portion sizes. The sensitivity of the technique increases when “energy-adjusted” variables are used, as described by Willett and Stampfer (52). Our approach in this study was therefore to examine reported fat intake relative to reported nonfat intake.

The magnitude of the effect of dietary fat intake on fat mass measured by the FFQ in our study was higher than that found in some studies in adults ($r = 0.20–0.38$) (7–9), but similar to that found by Gazzaniga and Burns (19). Smaller cross-sectional studies and larger collections of epidemiologic data indicate that dietary fat promotes obesity (18). Although dietary fat appears to explain only a small portion of the variance in fat mass, results from most studies invariably agree with experimental data, which provide the most convincing argument that fat intake has an effect on body fat; ie, rats become obese when fed isoenergetic diets with different fat contents (53). On the other hand, some strains of mice become obese when fed a high-fat diet whereas others are resistant (54), suggesting that genetic mechanisms are involved. The congruency of all studies investigating the association between fat mass and dietary fat intake suggests that 1) dietary fat is only one contributor to the development of obesity and/or 2) methodologic limitations of dietary intake measurement techniques may explain the consistent, but weak, effect of dietary fat intake on the variability in fat mass.

Metabolic factors other than diet composition are likely to be involved in the development of obesity. Studies have investigated differences in metabolic rates in children at risk for obesity (24, 55, 56), but the data have been inconsistent. According to our previous finding, there is currently no evidence from cross-sectional analyses to indicate that obesity in parents is related to energy expenditure components in children (24). Results of longitudinal studies should provide insight into changes that may occur in energy expenditure as children develop. Although not examined in our study, a high respiratory quotient (RQ), independent of decreased energy expenditure, has been found to be a predictor of weight gain in a group of adult Pima Indians (57). Moreover, the high RQ was a familial trait, so it is not unreasonable to speculate that children who are at risk for developing obesity may have a high RQ. Finally, some studies suggest that specific genetic factors are linked to human obesity (58, 59). Thus, it appears that although the etiology of obesity is complex, it may be modifiable by dietary factors. Our results indicate that obesity in children may not be due entirely to the fat content of the diet. Taken together with the studies discussed, it appears that the development of
obesity may result from an interaction of environmental and genetic factors.

In summary, our results suggest that obesity in mothers tends to influence dietary fat intake in their children, and that fat intake is significantly related to fat mass in boys. These findings have significant implications when educating parents and children about the importance of obesity prevention. Our data agree with the data of many studies in adults and the limited data in children. The sparsity of these types of studies in children indicates a need for further research in this area.

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