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The Pediatric Obesity Epidemic: Causes and Controversies

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Obesity in children and adolescents has reached alarming proportions in the United States. Nutritional surveys do not indicate a significant increase in caloric intake in children and adolescents over the last 3 decades, although caloric intake has increased recently in adolescent females. Dietary fat has also been falling. There is no conclusive evidence linking physical inactivity to the obesity epidemic, and longitudinal studies indicate that physical inactivity may be the result of obesity rather than its cause. Hence, attention should be focused on dietary carbohydrate. Carbohydrate intake has increased as a result of the decrease in dietary fat. Indirect evidence also indicates that the quality of carbohydrate has been changing, so that American children are eating more carbohydrates with a higher glycemic index. It is proposed that high-glycemic-index diets lead to excessive weight gain as a consequence of postprandial hyperinsulinemia. Low-glycemic-index diets lower postprandial insulin levels and insulin resistance. It seems likely that diets restricted in sweetened sodas and noncitrus juices and containing ample whole grains, vegetables, and fruit could have a major impact on the prevalence of pediatric obesity. (*J Clin Endocrinol Metab* 89: 2540–2547, 2004)

OBESITY IN CHILDREN and adolescents has reached alarming proportions in the United States (1). Increasing overweight and obesity has been noted since surveys were begun in 1963, but the rate of increase has been accelerating over the last 2½ decades (1, 2). By 1998, the prevalence of childhood and adolescent overweight, defined as a body mass index (BMI) greater than the 95th percentile for age and sex using current growth curves, had reached 21.5% for African-Americans, 21.8% for Hispanics, and 12.3% for non-Hispanic whites (1). Overweight children are also heavier than they have been in the past (1). The latest published National Health and Nutrition Examination Survey for years 1999–2000 (3) indicates that overweight now affects 15.5% of 12- to 19-yr-olds, 15.3% of 6- to 11-yr-olds, and 10.4% of 2- to 5-yr-olds (Table 1). All racial groups have become heavier, but Mexican-Americans and African-Americans are bearing the brunt of this epidemic. African-American females age 6–19 yr have been particularly affected, with a prevalence of obesity of 26.6% (4).

Caloric intake and the obesity epidemic

It is often assumed that the increase in pediatric obesity has occurred because of an increase in caloric intake. However, the data do not substantiate this. The Bogalusa Heart Study has been following the health and nutrition of children in Bogalusa, Louisiana since 1973. This study noted that total caloric intake for 10-yr-old children remained virtually unchanged from 1973–1988, with a slight but significant decrease evident when energy intake was expressed per kilogram body weight (5). National Health and Nutrition Examination Surveys also show that the mean energy intake for children and adolescents has changed little from the 1970s to 1988–1994 (Table 2) (6). However, the National Health and Nutrition Examination Survey III for the years 1988–1994 indicated for the first time an increase in caloric intake for white and black adolescent females, although not for other age groups (6). The latest National Health and Nutrition Examination Survey for 1999–2000 shows an even greater increase for adolescent females, suggesting that this increase is very real and not just a result of improved data collection methods (7). In line with this, the U.S. Department of Agriculture's National Food Consumption Surveys and Continuing Survey of Food Intake by Individuals also indicates a downward trend in the mean energy intake for the general population from 1965–1991 but an increase in the caloric intake for the years 1994 and 1995 (8).

These data pose two fundamental questions: 1) How was it possible for the prevalence of pediatric obesity to increase for so many years without a concomitant increase in calories, and 2) Why did mean caloric consumption increase for adolescent females in the mid-1990s?

Could decreased physical activity be to blame?

The observation that the weight of the pediatric population has been increasing without an increase in calories has suggested to many that decreased caloric expenditure must be a significant contributor to the current obesity epidemic. However, to date, this hypothesis remains unproven. There is very little longitudinal data on children's activity levels over the last few decades. A dramatic decrease in self-reported activity levels was evident from the National Heart, Lung, and Blood Institute Growth and Health Study as 10-yr-old black and white girls recruited in 1985 matured into adolescence (9). Nevertheless, demonstration of a decrease in activity level in the transition from childhood to adolescence is not necessarily indicative of longitudinal changes at all age groups.

Abbreviation: BMI, Body mass index.

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TABLE 1. Prevalence of overweight among children and adolescents ages 6–19 yr for years 1963–1965 to 1999–2000 (3)

	NHES 2 (1963–1965)	NHES 3 (1966–1970)	NHANES I (1971–1974)	NHANES II (1976–1980)	NHANES III (1988–1994)	NHANES 1999–2000	*P* values for NHANES III *vs.* NHANES 1999–2000
6–23 months[a]							
Total				7.2 (1.0)	8.9 (0.7)	11.6 (1.9)	0.09
Male				8.2 (1.4)	9.9 (0.8)	9.8 (2.2)	0.48
Female				6.1 (1.3)	7.9 (1.0)	14.3 (3.5)	0.04
2–5 yr[a]							
Total			5.0 (0.6)	5.0 (0.6)	7.2 (0.7)	10.4 (1.7)	0.04
Boys			5.0 (0.9)	4.7 (0.6)	6.1 (0.8)	9.9 (2.2)	0.06
Girls			4.9 (0.8)	5.3 (1.0)	8.2 (1.1)	11.0 (2.5)	0.16
6–11 yr[a]							
Total	4.2 (0.4)		4.0 (0.5)	6.5 (0.6)	11.3 (1.0)	15.3 (1.7)	0.02
Boys	4.0 (0.4)		4.3 (0.8)	6.6 (0.8)	11.6 (1.3)	16.0 (2.3)	0.05
Girls	4.5 (0.6)		3.6 (0.6)	6.4 (1.0)	11.0 (1.4)	14.5 (2.5)	0.11
12–19 yr[a]							
Total		4.6 (0.3)	6.1 (0.6)	5.0 (0.5)	10.5 (0.9)	15.5 (1.2)	<0.001
Adolescent boys		4.5 (0.4)	6.1 (0.8)	4.8 (0.5)	11.3 (1.3)	15.5 (1.6)	0.02
Adolescent girls		4.7 (0.3)	6.2 (0.8)	5.3 (0.8)	9.7 (1.1)	15.5 (1.6)	0.002

Values are expressed as percentage (SE). Overweight is defined as at or above the 95th percentile of BMI for age. The BMI-for-age growth charts were developed from five national data sets (NHES2, NHES3, NHANES I, NHANES II, and NHANES III for children <6 yr).

[a] A weight-for-length at the 95th percentile or higher is considered overweight.

[b] A body mass index for age at the 95th percentile or higher is considered overweight.

TABLE 2. Energy intake (kcal) for children and adolescents in the United States (6, 7)

Age (yr)/sex	NHANES I (1971–1974)	NHANES II (1976–1980)	NHANES III (1988–1994)	NHANES 1999–2000
2–5/males	1696 ± 23.4	1565 ± 18.6	1611 ± 20.3	1559 ± 26.3
2–5/females	1445 ± 22.0	1470 ± 17.0	1492 ± 20.3	1393 ± 37.2
6–11/males	2179 ± 29.9	2088 ± 25.6	2156 ± 41.6	2025 ± 97.2
6–11/females	1903 ± 26.1	1833 ± 28.0	1787 ± 25.6	1889 ± 43.7
12–19/males	2836 ± 49.5	2789 ± 47.8	2864 ± 50.7	2686 ± 56.4
12–19/females	1815 ± 33.5	1750 ± 23.9	1975 ± 33.7	2475 ± 45.7

Mean ± SEM. Estimates are based on a single 24-h dietary recall review.

A strong argument against decreased physical activity being a major cause of the current obesity epidemic is that longitudinal studies have been unable to demonstrate that physical inactivity in children predicts excessive weight gain. In fact, the opposite seems to be the case. The National Heart, Lung, and Blood Institute Growth and Health Study found that a higher baseline BMI in black and white females was directly associated with a decline in physical activity scores, particularly for the black females (10). Similarly, a 5-yr longitudinal study of 138 obese 5-yr-old Pima Indian children found that obesity at baseline was associated with decreased participation in sports and increased television viewing, but not with decreased physical activity level (11). Physical activity level was assessed as the ratio of total energy expenditure to basal metabolic rate. At age 10 yr, the associations between body weight, television viewing, and participation in sports were unchanged, but there was a negative association with physical activity level. These results suggested to the investigators that a decrease in physical activity follows rather than precedes the development of obesity.

By contrast, cross-sectional analyses have shown modest to strong associations between obesity and the television viewing of children (12–14), although a minority of studies have been unable to demonstrate this association (15, 16). A relationship between television viewing and obesity has also been evident in longitudinal studies, but there are some exceptions. Hence, a 2-yr longitudinal study of several hundred sixth- and seventh-grade girls found that hours of after-school television watching were not associated with baseline or longitudinal changes in BMI or triceps skinfold thickness (10). At baseline, there was a weak negative association between television watching and physical activity, but not with change in level of physical activity over time. A 1-yr longitudinal study of almost 200 3- to 4-yr-old children also found that television watching was weakly negatively associated with physical activity level but not with body composition (17). On the other hand, a 1-yr longitudinal study of over 10,000 children age 9–14 yr found that watching television and videos and playing computer games during the time of the study did have a small influence on change in BMI in both boys and girls (18). A 3-yr longitudinal study in adolescents found that BMI in the third year was directly related to baseline hours of television viewed, with adolescents who watched more than 2 h of television a day being twice as likely to be overweight than adolescents who watched less than 2 h (19). The Framingham Children's Study followed 106 children from childhood until early adolescence and found watching television and playing video games to be independent predictors of BMI and skinfold thickness, and these were related to the number of hours of television viewed (20). After controlling for physical activity, as assessed by means of an electronic motion sensor, television viewing still remained an independent predictor of changes in skinfold thickness, suggesting that the adverse effect of television viewing on obesity was not solely due to inactivity.

It would be difficult to conclude from the above studies

that decreased physical activity is the prime cause of the current pediatric obesity epidemic, although physical activity undoubtedly has a role in body weight regulation and could be a contributory factor. Nevertheless, it is relevant to ask why sedentary activities such as television viewing and playing video games appear to show a stronger relationship to weight gain in longitudinal studies than self-reported measures of physical activity. Firstly, the validation of self-reported measures of physical activity is open to question (21). Secondly, the relationship between television watching, playing video games, and body weight is undoubtedly a complex one, and these particular sedentary activities only weakly reflect overall level of physical activity (11, 17). It has been suggested that children who watch a lot of television may have different patterns of snacking and food consumption than other children (20). Their food purchases may also be influenced by advertising (12). Children who watch a small amount of television also have parents who are slimmer and have a higher level of education than those who view a lot of television (20).

The role of dietary fat

Excess consumption of dietary fat is a strong contender as an etiological factor for the development of obesity. Indeed, there are strong theoretical reasons why this should be so. The efficiency of nutrient utilization is higher for fat than for carbohydrate or protein, reflecting the low amount of energy required for the synthesis of triglyceride (15). In addition, when energy balance is positive, a proportion of dietary fat is deposited directly into adipose tissue without oxidation (15).

Many pediatric studies have shown a significant association between fat consumption and adiposity (22–26). There is also a wealth of ecological and interventional data linking adult obesity to dietary fat (27). However, the few prospective studies in children and adults that have looked at dietary fat have obtained contradictory results. A 1-yr longitudinal study of over 10,000 children age 9–14 yr was unable to show any relationship between change in BMI and dietary fat (18). A 3-yr longitudinal study in 294 adult men and women found that the percentage of dietary fat and changes in fat intake did predict changes in body weight in men and women (28). A 5-yr prospective study in 12,699 adult Finns, in whom one sixth of the subjects had a dietary assessment at baseline, revealed that daily energy intake significantly predicted weight gain in women, but this was not solely attributable to fat intake (29). In men, weight gain showed no association with either energy intake or macronutrient intake. The Nurses' Health Study followed 121,700 female nurses for 8 yr with a semiquantitative food frequency questionnaire being administered half-way through the study (30). Prior weight change and age explained 8.4% of the variance in subsequent weight change. Addition of specific nutrients to the model increased the percentage of variance that could be explained by only 0.4%. Nevertheless, these authors make the important point that prospective studies examining relationships between weight gain and specific nutrients may be difficult to interpret, because dietary modifications are often made in response to changes in body weight.

The major objection to the notion that dietary fat is responsible for the current pediatric obesity epidemic is the fact that fat consumption in American children has fallen over the last 3 decades (Table 3). In 1977, in an attempt to reduce blood cholesterol levels in this country and thereby reduce the incidence of cardiovascular disease, the U.S. Senate Select Committee on Nutrition and Human Needs issued dietary goals for the United States in which it was recommended that the adult American population reduce cholesterol intake to about 300 mg per day, dietary fat to 30% of calories, and saturated fat to about 10% of total energy intake (31). When these recommendations were issued, dietary fat consumption in adults was approximately 40% of total calories. These recommendations were initially targeted at adults, but by 1991, it was felt appropriate to extend these guidelines to children age 2 yr and older (32). After this and subsequent reports, dietary fat consumption in 10-yr-old American children fell from 38.4–34% of total calories between 1973 and 1993, according to data from the Bogalusa Heart Study (33). The National Health and Nutrition Examination Survey also recorded a decrease in the percentage of fat consumed by children, from between 35.9 and 37% of total calories in 1971–1974 to between 32.7 and 33.8% of total calories in 1988–1994 (Table 3) (34). The 1999–2000 National Health and Nutrition Examination Survey shows a continuing decrease in the percentage of dietary fat consumed by adolescents, and this is now between 32.0 and 32.1% of total calories (Table 3) (7).

Hence, at the very time that the prevalence of pediatric obesity was accelerating, the consumption of dietary fat in the population was decreasing. Dietary fat, when eaten in excess, undoubtedly leads to obesity in predisposed individuals. However, it seems unlikely that increased fat consumption is responsible for the large increase in pediatric obesity.

Focus on dietary carbohydrate

There are at least two reasons why attention should now focus on dietary carbohydrate: 1) The amount of carbohydrate in children's diets has been increasing. This was an

TABLE 3. Trends in age-adjusted mean fat intake as a percentage of energy for children and adolescents in the United States (7, 34)

Age (yr)/sex	NHANES I (1971–1974)	NHANES II (1976–1980)	NHANES III (1988–1994)	NHANES 1999–2000
2–5/males	36.0 ± 0.33	34.9 ± 0.33	32.7 ± 0.31	32.9 ± 0.47
2–5/females	36.5 ± 0.33	35.7 ± 0.26	32.9 ± 0.28	32.8 ± 0.53
6–11/males	36.7 ± 0.21	35.7 ± 0.34	33.8 ± 0.31	33.0 ± 0.54
6–11/females	35.9 ± 0.32	35.8 ± 0.20	33.6 ± 0.35	32.8 ± 0.54
12–19/males	37.0 ± 0.29	36.4 ± 0.28	33.6 ± 0.34	32.0 ± 0.42
12–19/females	36.7 ± 0.27	36.4 ± 0.32	33.8 ± 0.42	32.1 ± 0.61

Mean ± SEM. Estimates are based on a single 24-h dietary recall review.

inevitable consequence of the recommendations to decrease dietary fat; and 2) The type of carbohydrate that children are eating also appears to have changed. Much of the evidence for this is indirect but nevertheless quite suggestive.

In 1992, the Bogalusa Heart Study pointed out that children who occupied the lowest percentiles of fat consumption had increased their intake of simple sugars (35). Twelve years earlier, Gibney (36) had also pointed out, on the basis of cross-sectional data from several European countries, that low-fat diets are invariably associated with an increase in the consumption of simple sugars.

As an example of this phenomenon, it is instructive to examine what happened to milk consumption in the United States between 1965 and 1996. During this time, milk consumption declined by 36% because the saturated fat content of dairy foods rendered milk a suspect food. These lost calories were replaced mainly by soft drinks, soda, and fruit drinks (37, 38).

There is growing concern that this increased consumption of sweetened drinks is having a detrimental effect on children's health. Ludwig *et al.* (39) studied 548 schoolchildren in a 19-month prospective study and reported that baseline consumption of sugar-sweetened drinks was independently associated with an increase in BMI of 0.18 kg/m^2 for each daily serving ($P = 0.02$). Each additional serving over baseline was associated with an increase in BMI of 0.24 kg/m^2 and an increased frequency of obesity ($P = 0.02$ for both variables).

Carbohydrates are usually categorized as either simple sugars or complex carbohydrate. However, in terms of their postprandial metabolic and hormonal responses, many complex carbohydrates do not behave much differently than simple sugars. From a physiological standpoint, therefore, it makes considerable sense to classify carbohydrates in terms of their glucose and insulin responses. This is conveniently expressed in terms of their "glycemic index."

The glycemic index of a carbohydrate-containing food describes the area under the postprandial glucose curve for 50 g of that carbohydrate in relation to 50 g of glucose or white bread and reflects the ease with which that carbohydrate is digested. In general, starches made up of whole grains have low glycemic indices, as do whole beans and most green vegetables. Factors responsible for the glycemic index of a food relate to its method of preparation, the characteristics of the starch and starch granules, cooking, and susceptibility to α-amylase action (40). Pasta, for example, has a low glycemic index relative to bread because of reduced amylolysis (41). Dietary fiber is a moderate predictor of glycemic index, although the relationship is not always a close one because gel-forming fibers decrease postprandial glucose excursions, whereas insoluble fibers such as wheat bran do not (40–42). Among 18 starchy foods, fiber accounted for 40% of the variance in glycemic index (43). For dietary carbohydrate, there is a moderately high correlation between postprandial glucose and insulin responses (44).

An important question is whether differences in glycemic index are expressed when carbohydrates are eaten within a mixed meal. Wolever *et al.* (45) have shown that the quantity of a carbohydrate together with its glycemic index account for approximately 90% of the glucose and insulin responses of mixed meals of varying carbohydrate, protein, and fat content. On the other hand, Hollenbeck and Coulston (46) have pointed out that differences in glycemic index are lost within the context of a mixed meal and ascribe this to the influence of dietary fat and protein. Protein, for example, is a weak insulin secretagogue (47), and the postprandial insulin response of a protein-containing mixed meal reflects a synergism between the carbohydrate and protein content of the food (48). This would lead to an attenuation of the glucose response. Despite the reservations of Hollenbeck and Coulston (46), it is clear from numerous studies that diets composed of low-glycemic carbohydrate have profound effects on intermediary metabolism compared with high-glycemic diets. In diabetics, for example, low-glycemic-index or high-fiber diets consistently lead to lower glucose and insulin profiles compared with isocaloric high-glycemic-index or low-fiber diets (49–52).

There is much indirect evidence that the glycemic index of children's diets has increased in the United States over the last few decades. The frequency of snacking as well as the contribution of snacks to total caloric intake has increased in children of all ages (53, 54). More people are eating fast foods outside the home (55, 56). Bread consumption at breakfast has declined, particularly for whole grain bread, and has been replaced by ready-to-eat cereals, particularly highly refined breakfast cereals (57). Ready-to-eat breakfast cereals are increasingly being eaten at times other than at breakfast (58). Vegetable intake has increased, but primarily from high-glycemic-index white potatoes (37). Soft-drink consumption among adolescent boys tripled between 1977–1978 and 1994, and soft drinks now contribute about 8% of the total energy intake of adolescents (34).

Between 1989 and 1991, the top 10 categories of carbohydrate for 2- to 18-yr-old children were, in descending order of importance, yeast bread (13% of carbohydrates), soft drinks/soda (8.5%), milk (7.9%), ready-to-eat cereal (7.4%), cakes/cookies/quick breads/donuts (7.2%), sugars/syrups/jams (6.0%), fruit drinks (4.3%), pasta (3.9%), white potatoes (3.7%), and orange/grapefruit juice (2.9%) (38). As pointed out by Ludwig (59), other than the milk, pasta, and orange juice, these carbohydrates are high-glycemic carbohydrates and as such are associated with large postprandial excursions in blood glucose and insulin.

Three mechanisms can be suggested to explain the relationship between high-glycemic-index carbohydrate and obesity: 1) high-glycemic carbohydrate leads to postprandial hyperinsulinemia, and this may lead to excessive weight gain; 2) sugar-containing drinks tend not to displace energy from other food sources, leading to an increase in energy intake (60–63); 3) high-glycemic-index foods promote hyperphagia.

Hyperinsulinemia and weight gain

There is evidence, albeit limited, that postprandial hyperinsulinemia may result in excessive weight gain. There are also indications that part of this effect may be due to a change in resting energy expenditure. It is known that a changing hormonal milieu, for example GH replacement in GH-defi-

cient adults, can lead to large changes in resting energy expenditure (65, 66). The notion that the glycemic index of a diet could influence resting energy expenditure through its effect on insulin secretion is quite speculative but nevertheless warrants serious consideration because it could explain how the prevalence of obesity could rise without a concomitant increase in calories.

In young genetically obese animals such as the Zucker rat, weight gain and the subsequent development of insulin resistance is preceded by increased insulin secretion in response to iv glucose and arginine but not fasting hyperinsulinemia (67). Diabetic patients on the sulfonylurea drug glibenclamide, a potent insulin secretagogue, gain more weight than those on insulin, and patients treated with insulin gain more weight that those on the insulin-sensitizing agent metformin (68). In the 6-yr United Kingdom Prospective Diabetes Study Group, weight gain was 6 kg on glibenclamide, 4 kg on insulin, and 1 kg on metformin or diet alone (68). Unwanted weight gain was also an unintended consequence of the intensive insulin therapy branch of the Diabetes Control and Complications Trial, and this occurred despite a decrease in caloric intake from baseline (69). Excessive weight gain was more frequent in those subjects who experienced one or more severe hypoglycemic episodes (69). Carlson and Campbell (70) studied body composition, energy expenditure, glycosuria, and substrate kinetics in six adult patients with insulin-dependent diabetes who had been changed from conventional to intensive insulin therapy. Body weight and fat mass increased on intensive insulin therapy with no change in caloric intake. It was estimated that elimination of glycosuria contributed 70% to the positive energy balance, and a reduction in 24-h energy expenditure contributed the remainder. The latter was the result of a decrease in triglyceride/free fatty acid cycling and nonoxidative glucose and protein metabolism. Lustig *et al.* (71) showed that children with hypothalamic obesity, in whom there is excessive insulin secretion, lose weight when placed on the somatostatin agonist octreotide, with the weight loss correlating with the reduction in insulin levels on an oral glucose tolerance test.

The consequences of postprandial hyperinsulinemia need to be differentiated from those of fasting hyperinsulinemia. Fasting hyperinsulinemia reflects insulin resistance. There is considerable debate in the literature as to whether the insulin-resistant state is weight promoting, or whether insulin resistance is an adaptive mechanism permitting weight maintenance in the presence of hyperinsulinemia (72, 73). This is still an open question, although much of the evidence in adults favors the latter view. One study in Chinese men found that insulin resistance estimated from fasting insulin and glucose values (homeostasis model of assessment) predicted weight gain independently of baseline weight and age (74). However, the Atherosclerosis in Risk in Communities Study, a longitudinal study of over 11,000 45- to 64-yr-old subjects, found that lower fasting insulin was associated with greater weight gain (75). A study in mainly obese but otherwise healthy adult Pima Indians also found that insulin-sensitive subjects gained more weight than those who were more insulin resistant (76). Ferrannini *et al.* (77) found the prevalence of insulin resistance in nondiabetic, normotensive obese adults was relatively low and was exceeded by the prevalence of insulin hypersecretion, as determined from a euglycemic insulin clamp technique. Sigal *et al.* (78) followed weight gain over 16 yr in 107 glucose-tolerant adult offspring of two parents with type 2 diabetes and showed that a high first-phase insulin response to iv glucose was a risk factor for long-term weight gain. Weight gain occurred in particular in insulin-sensitive individuals. Giacco *et al.* (79) also showed that insulin sensitivity is increased in normal-weight healthy men with a strong familial predisposition to obesity.

Studies in children have also produced contradictory results. One longitudinal study in 328 young, moderately obese Pima Indian children found that baseline fasting insulin predicted weight gain and triceps skinfold thickness 9 yr later (80). On the other hand, a 3-yr prospective study in 111 early pubertal children demonstrated that sc fat gain was greater in the more insulin-sensitive children, although they were unable to demonstrate a difference in BMI (81). Observations from the Bogalusa Heart Study indicate that adolescent offspring of parents with coronary artery disease compared with controls have lower fasting insulin levels from childhood until age 20, despite being heavier and having an increased skinfold thickness (82). Only in young adulthood do these offspring manifest fasting hyperinsulinemia in association with their increasing body weight.

The effect of low-glycemic-index and low-carbohydrate diets on hyperinsulinemia, body weight, and resting energy expenditure

Low-glycemic-index diets decrease postprandial hyperinsulinemia and insulin resistance independently of changes in body weight or caloric intake. In 32 patients with advanced coronary disease, 4 wk of a low-glycemic diet reduced the glucose and insulin response to an oral glucose tolerance test compared with a high-glycemic-index diet without any change in body weight (83). Frost *et al.* (84) randomized 28 premenopausal women, some of whom had a parental history of coronary artery disease, to a low- or high-glycemic-index diet, the diets being otherwise identical in their macronutrient content. The low-glycemic-index diet led to a significant increase in insulin sensitivity after 3 wk as demonstrated by a short insulin tolerance test.

Low-glycemic-index diets also influence body weight and resting energy expenditure independently of caloric intake. In a short cross-over study, Agus *et al.* (85) compared a high-glycemic-index energy-restricted diet with an isocaloric low-glycemic-index diet in moderately overweight young men and demonstrated that resting energy expenditure declined by 10.5% on the high-glycemic-index diet compared with 4.6% on the low-glycemic-index diet. In obese hyperinsulinemic women, Slabber *et al.* (86) demonstrated lower fasting insulin levels and greater weight loss after 12 wk of an energy-restricted low-glycemic diet compared with an isocaloric conventionally balanced American diet. In a preliminary report, Bouche *et al.* (87) showed that fat mass and abdominal fat, as demonstrated by dual-energy x-ray absorptiometry, was reduced on 5 wk of a low-glycemic-index diet compared with a high-glycemic-index diet in healthy,

slightly overweight men, there being no difference in energy intake or body weight.

The influence of low-carbohydrate diets on body weight is also of considerable interest in this respect. As for low-glycemic-index diets, low-carbohydrate diets decrease fasting insulin levels (88). It is now well established that weight loss over 6 months is greater with low-carbohydrate diets than with conventional low-fat diets (89–91). A systematic review of over 100 articles relating to low-carbohydrate diets concluded that their success was due primarily to their anorexic effect (92). However, results from three studies raise the possibility that low-carbohydrate diets may also influence energy expenditure. Sondike *et al.* (93) randomly assigned 30 adolescents to a low-carbohydrate diet (<20 g of carbohydrate per day) or low-fat diet (<30% of energy from fat) over a 3-month period and obtained the surprising result that the greater weight loss on the low-carbohydrate diet was associated with a greater consumption of calories ($P = 0.03$). Samaha *et al.* (89) compared a low-carbohydrate diet with a calorie-restricted low-fat diet in severely obese adults and found significantly greater weight loss and improvement in insulin sensitivity on the low-carbohydrate diet, there being no significant difference in caloric intake between the two groups. Essentially similar results were obtained by Brehm *et al.* (91) in a study in obese adult females. Weight loss was greater over 6 months on a very-low-carbohydrate diet compared with a calorie-restricted low-fat diet, and this effect could not be explained by caloric intake, which was similar for the two diets. A number of reasons can be suggested for the improved weight loss while on the low-carbohydrate branch of these studies, including biased reporting of calories while on one of the diets and increased urinary ketone excretion (94). However, a testable hypothesis is that diets that lower blood insulin levels also influence resting energy expenditure.

The influence of high-glycemic carbohydrate on hunger and carbohydrate craving

The National Health and Nutrition Examination Survey III from 1988–1994 reported an increase in caloric intake for female adolescents compared with previous surveys, and the National Health and Nutrition Examination Survey for 1999–2000 indicated a further increase in caloric intake for this group (Table 2) (6, 7). This raises the question as to why this has occurred. It could conceivably be related to cultural influences on food portion sizes and snacking. Although adolescent females decreased the percentage of fat in their diets, their total fat consumption actually increased in tandem with the increase in total calories (Table 3). However, the phenomenon may also have a physiological basis. Excessive consumption of sweetened drinks leads to loss of the ability to precisely regulate caloric intake (60–63). High-glycemic carbohydrate also leads to hunger and carbohydrate craving.

The effect of insulin and glycemic index on hunger and satiety has been the subject of several reviews (47, 95). When insulin is infused into normal subjects, it induces hunger, heightens the palatability of sweet solutions, and increases food intake, even in the absence of hypoglycemia (96). Ludwig (59) has recorded 15 studies in the adult literature demonstrating increased satiety, delayed return of hunger, and decreased food intake after the ingestion of low-glycemic-index compared with high-glycemic-index foods. A recent study in obese adolescents demonstrated a prolongation of satiety after a low-glycemic-index meal compared with a high-glycemic-index meal (97). Ludwig *et al.* (98) demonstrated in obese adolescents that voluntary food intake after breakfast and lunch was higher after high-glycemic-index meals compared with isocaloric medium-glycemic-index meals, whereas medium-glycemic meals resulted in a higher food intake than isocaloric low-glycemic-index meals.

Conclusions

There is no evidence that an increase in caloric or fat intake has been responsible for the current epidemic of pediatric obesity, although an increase in calories may be contributing to the rising prevalence of obesity in adolescent females. The contribution of physical inactivity remains ambiguous. Attention should therefore be focused on dietary carbohydrate. It is proposed that an increase in the amount of carbohydrate in the American. diet, coupled with a change to higher-glycemic-index carbohydrate, may be the most important contributors to our pediatric obesity epidemic. High-glycemic loads lead to postprandial hyperinsulinemia, and this may result in excessive weight gain in susceptible children.

When "Dietary Goals for the United States" (31) was published in 1977, it was reasonable to assume that low-fat diets would reduce the prevalence of obesity at the same time as lowering blood cholesterol levels. This assumption was clearly fallacious. The role of the glycemic index in nutritional management remains controversial, although its role as a research tool is well established. This controversy should not obscure the message that the prevalence of pediatric obesity could be significantly decreased by diets restricted in sweetened sodas and noncitrus juices and containing ample whole grains, vegetables, and fruit (59, 99–103).

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