The Adiposity Rebound: Its Contribution to Obesity in Children and Adults

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The increasing prevalence of childhood obesity is a major problem affecting both industrialized and developing countries (1). Given the poor prognosis of treatment in adults, prevention or early intervention are desirable. Most obese children, however, will not remain fat as adults, and certain children who are not overweight will develop obesity later (2,3). It is therefore important to be able to identify those overweight children who will remain fat and those nonobese children who may become fat later. Indicators predicting adult adiposity are of great potential value. They are useful for pediatricians as guides for intervention and are also useful for researchers. Studying these indicators should allow progress in understanding the origins of obesity.

Body measurements at different ages and their temporal variations are used to predict adult fatness. These include weight, skinfold thickness, and weight and length gain (2,4,5). The weight/height², Quetelet or body mass index (BMI) is now widely used to assess nutritional status in children.

Changes in the BMI pattern have been proposed as an indicator predicting adult adiposity (6,7). Dietz suggests that childhood overweight and obesity develop during critical periods (8)—the prenatal period, during the adiposity rebound, and in adolescence. Available information related to the adiposity rebound and its value in predicting adult obesity will be considered in this chapter.

ADIPOSITY DEVELOPMENT

During childhood, body composition changes substantially. The percentage of body fat is about 12% at birth, 22% in 9-month-old infants, and 16% at the age of 5 years. This percentage rises again up to the age of 10 years. It then decreases at adolescence in boys and continues to increase in girls (9).

Physical development in childhood is generally followed on the basis of weight and height measurements. Weight-for-age charts are used to assess nutritional status (10). As weight is strongly associated with height, weight measurements should be
related to height. Weight-for-height charts, however, do not take age into account. Adjustment of weight for both height and age can be achieved using power indices of the form weight/height$^2$. The selection of indices was based on low correlations with height, and high correlations with weight and body fat. As a rule, weight/height$^2$ shows the lowest correlation with height, except at adolescence in boys, where weight/height$^2$ shows lower correlation with height (6). BMI charts have been constructed for all ages throughout childhood (6). As opposed to other measures based on weight and height (weight for age or weight for height), BMI/age changes reflect real changes in the child’s body shape during growth. By the age of 1 year, a child whose BMI is in the mean range looks chubby. Over the next few years, the child will slim down and look thin by the age of 6 years, but will later put on fat again. The similarity between the development of the BMI and more direct measures of fatness such as skinfold thickness (11) (Fig. 1) is a major argument supporting BMI over other indices.

When examining individual BMI patterns, it was noted that the nadir of the BMI curves, which on average takes place by the age of 6 years, could occur earlier or later. The point of minimum BMI value was named the “adiposity rebound” (7). The age at adiposity rebound is associated with BMI development. As a rule, the earlier the adiposity rebound, the greater the degree of adult adiposity (3,4,7,12–17).

Changes in weight status can be clearly understood by examining individual BMI patterns (Fig. 2). For example, a fat 1-year-old child may become a normal-weight adult after a late adiposity rebound, whereas a lean 1-year-old child may become a

![Body Mass Index and Subscapular skinfold](image)

**FIG. 1.** Development of fatness as assessed by body mass index and subscapular skinfold and changes of adipose tissue cellularity. (Data from Rolland-Cachera et al., 1982 [6]; Sempé et al., 1979 [11]; and Knittle et al., 1979 [19].)
fat adult after an early adiposity rebound. These individual BMI patterns explain why, before the adiposity rebound, the child’s BMI predicts adult fatness only poorly (2–4). Case 3 in Fig. 2 (a thin child at 4 years becoming overweight after an early adiposity rebound) shows that overweight diagnosed at adolescence actually can have its origin much earlier in life. The onset of obesity can be defined as the time when the BMI overlaps the cutoff centile defining obesity. In those children who are not fat from early childhood, the age of adiposity rebound is generally before that at which obesity is established. It then constitutes useful information for the pediatrician and the researcher.

**METHODOLOGY**

The age at adiposity rebound corresponds to the point of minimum BMI value, the nadir of the curve preceding the steep increase in the curve. Different methods are used to assess age at adiposity rebound. In some studies (3,7,13,16), BMI curves were drawn for each subject, and the timing of the adiposity rebound was assessed by visual inspection. This involves identifying an upward trend in the BMI after the nadir. In some cases, the descending phase of BMI is followed by a plateau (e.g., case 4 in Fig. 2). In this case, 8 years was considered to be the age of adiposity rebound, because at this age the steep increase in BMI begins. To identify the upward trend in
BMI, Dorosty et al. (16) specified that all consecutive measurements of the BMI after the nadir should show an increase and that any increase in the BMI after the nadir had to equal or exceed 0.1 kg/m². Gasser et al. (4) determined the adiposity rebound from the velocity curve of the BMI. Age at adiposity rebound corresponds to the point at which the velocity curve becomes positive after the loss of BMI in infancy and early childhood.

Siervoel et al. (12) chose a polynomial model to describe the pattern of change in BMI. Age at minimum BMI was derived from this model. Other investigators have used the same approach (14,15,17).

ADIPOSITY REBOUND INVESTIGATIONS

An international growth study was initiated in 1953 (18). In the early 1980s, on the basis of the French sample, Rolland-Cachera et al. described the BMI pattern, emphasizing the ascending and descending phases at different stages of growth (6). They then established that an early adiposity rebound was associated with a high BMI at the age of 16 years. This association was observed wherever the BMI at age 1 year (7). In this study the association between age at adiposity rebound and bone age was also investigated. The changes in the pattern of BMI and adipose tissue cellularity (19) during growth were discussed. The increase in BMI during the first year of life followed by a decrease corresponds to variations in adipocyte size, whereas after the age of 6 years the rise in BMI corresponds to the increase in adipocyte number (Fig. 1). It was suggested that the age at adiposity rebound may reflect the time at which adipocytes start to increase in number (7,20).

Subsequently, we analyzed the association between adiposity rebound and fatness development up to the age of 21 years (3) (Fig. 3). Adiposity rebound was significantly associated with both BMI and subscapular skinfold at age 21. We also investigated the association between age at adiposity rebound and previous measurements.

![Image](image_url)

**FIG. 3.** Mean body mass index curves for three groups of girls classified as early (——), average (——), or late (—–) adiposity rebound. NS: $p > 0.05$; *$p < 0.05$; **$p < 0.01$. (From Rolland-Cachera et al., 1984, 1987 [3,7].)
FIG. 4. Distribution of the age at adiposity rebound in the French reference population (N, 227; mean age at adiposity rebound, 6.2 ± 1.3 years) and in a sample of children treated for obesity in a department of pediatric endocrinology (N, 62; mean age at adiposity rebound, 3.2 ± 1.2 years). In the reference population, age at adiposity rebound was ≤ 6 years in 51% of children; in obese children age at adiposity rebound was ≤ 3 years in 55% of children (3).

An opposite association was found between age at adiposity rebound and BMI at the age of 1 year: The earlier the rebound, the lower the BMI at 1 year. This trend was recorded in both boys and girls, but it reached significance only in girls.

The association between age at adiposity rebound and fatness at adult age emerged even in a sample including mostly normal-weight children. In this sample, mean age at adiposity rebound was 6.2 ± 1.3 years, varying from 3 to 9.5 years (Fig. 4) (3). The discussion of this paper reported unpublished data collected in a department of pediatric endocrinology where children were treated for obesity. Mean age at adiposity rebound was 3.2 ± 1.2 years. Of the 62 children, none had an adiposity rebound later than the age of 6 years. Two children had their adiposity rebound when they were 6 (i.e., average age at adiposity rebound) but more than half of them (55%) had their rebound at or before the age of 3 (Fig. 4). A similar mean age at adiposity rebound recorded in obese children (2.9 years) has been reported elsewhere (21).

On the basis of data recorded in the Fels longitudinal study in children between the ages of 2 and 18 years, Siervogel et al. (12) investigated individual changes in body fatness during childhood and adolescence. They reported that both the age when children reach their BMI nadir (the age at adiposity rebound) and BMI level at adiposity rebound were associated with the BMI value at 18 years. Correlations between age at adiposity rebound and adult BMI are negative, as a younger age at adiposity rebound is associated with a greater adult BMI.

In a longitudinal study conducted in Czech children from 1 month to 18 years of age, Prokopec and Bellisle (13) reported similar association between early adiposity rebound and later increased fatness. In that study, most overweight adults (15 of 18 with a BMI of more than 25 kg/m² at 18 years of age) had an early adiposity rebound
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(before the age of 6 years), and all lean adults had late adiposity rebound (after 6 years).

On the basis of the Zurich sample from the international growth study initiated in 1954 (18), Gasser et al. (4) examined the prediction of adult measurements from measurements recorded in infancy and through adolescence. They found that the age at adiposity rebound correlated significantly with adult BMI.

Whitaker et al. conducted a retrospective study in adults (14). BMI curves were fitted for each subject between 1.5 and 16 years. Age and BMI level at adiposity rebound were recorded from these curves. The BMI values of parents were also available. Adult obesity was associated with early adiposity rebound, high BMI at adiposity rebound, heavy mothers, and heavy fathers. After adjusting for parent BMI and BMI at adiposity rebound, the odds ratio for adult obesity associated with early versus late adiposity rebound was 6.0 (95% confidence interval, 1.3 to 26.6). Both parent BMI and BMI z score at adiposity rebound were associated with a younger age at rebound. This study showed that an early adiposity rebound was associated with an increased risk of adult obesity independent of parental obesity and the BMI at adiposity rebound.

In a follow-up study conducted in children between the ages of 3 and 18 years, Williams et al. (15) reported that BMI in early adulthood was associated with both age at adiposity rebound and BMI at that age. Skeletal maturity was also assessed in that study.

On the basis of a study conducted in the United Kingdom, Dorosty et al. (16) investigated the nutritional determinants of early adiposity rebound in children followed from birth to 61 months. Children with very early, early, and later adiposity rebound were compared. There was no evidence of a difference in absolute BMI between the three groups before the adiposity rebound, except in girls with a very early rebound. After the rebound had occurred, BMI was significantly higher in those who had rebounded very early. The association between parental BMI and adiposity rebound was also examined.

On the basis of the Fels longitudinal study, Guo et al. (17) examined BMI patterns from early childhood to 35 to 45 years. They showed that changes in childhood BMI were related to adult overweight and adiposity, more so in women than in men. The odds ratios for age at adiposity rebound in relation to adult BMI overweight status (BMI ≥ 25 versus BMI < 25) were 0.88 in male subjects and 0.44 in female subjects, showing that an early adiposity rebound was associated with an increased risk of adult obesity in women but not in men.

All the studies described here reported a highly significant association between an early adiposity rebound and the risk of developing obesity at a later age. However, the magnitude of the association varied between studies. In the Fels longitudinal study (12), the correlation between age at adiposity rebound and BMI at 18 years was −0.50. In the study conducted in New Zealand (15), correlations with measurements at 21 years were −0.56 for boys and −0.43 for girls. In the Zurich study (4), correlations between age at adiposity rebound and BMI at adult age were −0.31 for boys and −0.30 for girls.
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Some investigators (4,15) noticed that the correlation between the BMI level at the age of 6 or 7 years (mean age at adiposity rebound) and BMI at adult age was similar or higher than the correlation between age at adiposity rebound and BMI at adult age. Williams et al. (15) concluded that BMI level recorded by the age at adiposity rebound was a more practical way of predicting BMI in adulthood than assessing age at adiposity rebound. In fact, different aspects must be considered. BMI level by the age at adiposity rebound and the age at adiposity rebound are independent predictors of adult adiposity (12,14). They may give complementary information on the factors initiating the development of fatness. A high BMI at 6 years and an early adiposity rebound correspond to different BMI patterns. BMI at the age of 6 years is significantly associated with later BMI, but also with previous BMI levels. In the Zurich study (4), the correlation between BMI at 6 years and the BMI at adult age was 0.60. It was of the same magnitude ($r = 0.66$) between the BMI at 6 years and the BMI at 1 year. In the French sample of the international growth study, an early adiposity rebound was significantly associated with higher BMI at the age of 21 years, but no such a trend—or even an opposite trend—was observed for the correlations between age at adiposity rebound and measurements at the age of 1 year (Fig. 3). Similarly, Dorosty et al. (16) did not find a clear trend of high fatness before an early adiposity rebound. On the basis of the data from a longitudinal study of nutrition and growth (20), we found that both an early adiposity rebound and a high BMI at 6 years predicted a high subsequent BMI level. The association with previous measurements, however, was different for the two indicators. BMI at 6 years was positively and significantly correlated with birth weight ($r = 0.23; p = 0.003$), whereas age at adiposity rebound was not correlated with birth weight ($r = -0.06; p = 0.53$) (Rolland-Cachera MF et al., unpublished data). Consequently, as a rule a fat child at age 6 years is likely always to be fat, whereas a child with an early adiposity rebound develops fatness only from the time of the rebound and is likely to have been thin before rebound (Fig. 3).

An inverse association between BMI level before and after adiposity rebound has been reported in various circumstances (22). For example, comparing different countries, the mean BMI level showed an opposite rank order before and after adiposity rebound. The differences in BMI patterns (permanently high BMI, or low BMI followed by high BMI after adiposity rebound) are probably associated with different obesity-promoting factors. The use of both anthropometric indicators (BMI at 6 years and age at adiposity rebound) may improve the identification of the various determinants of obesity.

FACTORS ASSOCIATED WITH ADIPOSITY REBOUND

Differences Between Studies

Mean age at adiposity rebound varies between studies from 5 to 7 years. As age at adiposity rebound varies according to the method used (15), it is difficult to make valid comparisons between studies. Three studies have used the same method (visual inspection). Two were conducted in France, and one in the Czech Republic. Mean age
at adiposity rebound was 6.2 years in children born in 1955 (6) and 5.6 years in those born in 1985 (20). In Czech children, mean age at adiposity rebound was 7 years (13). Three studies used a polynomial model to assess age at adiposity rebound. In American children, mean age at rebound was 5.2 years (12) or 5.5 years (14). In New Zealand it was 6.2 years (15). In a study conducted in China it was 5 years (23). In this last study measurements were recorded until the age of 7 years only. As adiposity rebound may occur up to the age of 10 years, this mean value may be an underestimate.

In addition to differences attributable to the methods used, factors such as the date at measurements, the prevalence of obesity in each sample, and country-specific environmental conditions may affect mean age at adiposity rebound.

**Gender**

No consistent gender differences emerge from the available data regarding either mean age at adiposity rebound or the association between age at rebound and adult fatness. In the French sample of the international growth study (6,18), age at adiposity rebound was earlier in girls than in boys (6.05 versus 6.26 years). In a study started 30 years later (20), adiposity rebound occurred later in girls than in boys (5.9 versus 5.4 years). In the Fels longitudinal study (12), the rebound took place later in girls (5.34 versus 5.13 years), and in the New Zealand study (15), it was earlier in girls (6.1 versus 6.3 years). In the retrospective cohort conducted in the United States (14), girls tended to reach adiposity rebound earlier than boys (5.4 versus 5.8 years). In the Czech study, no difference was recorded between the sexes (7 years in both boys and girls). The sex differences observed in all these studies are probably not significantly different. A sex difference was observed in the predictive value of the adiposity rebound in the Fels longitudinal study (17). An early adiposity rebound was associated with a greater risk of adult obesity in female subjects but not in male subjects. In other studies no such sex differences appeared (4,7,12–16).

**Parental BMI**

Two studies have examined the association between parental BMI and age at adiposity rebound. The retrospective study of Whitaker et al. (14) showed that parent BMI was associated with a younger age at adiposity rebound. In the study conducted by Dorosty et al. (16), mean BMI of parents was significantly higher in children with a very early adiposity rebound. Having at least one obese parent was also associated with both very early and early adiposity rebound.

**Skeletal Maturity**

Two studies have analyzed the association between age at adiposity rebound and skeletal maturity. An early adiposity rebound was associated with advanced skeletal maturity. In the French longitudinal study (7), a trend for older bone age to be associated with an early adiposity rebound has appeared from the age of 2 years, though this is not yet significant. The trend is clearer between 8 and 13 years of age in girls
and between 11 and 15 years in boys. The greatest differences are observed at age 12 years in girls ($p < 0.001$) and at age 15 years in boys ($p < 0.005$). Williams et al. (15) also found that an earlier adiposity rebound was associated with more advanced skeletal maturity, but the association was significant for boys only. These observations are consistent with the accelerated growth observed in childhood obesity.

**Socioeconomic Status and Parental Education**

De Spiegelaere et al. (24) found no association between socioeconomic status and adiposity rebound. Dorosty et al. (16) reported that there was no evidence of any association between parental education or socioeconomic status and the timing of the adiposity rebound. This is consistent with previous analyses showing that, compared with adult obesity, which is related to lower socioeconomic status, in children such an association is less clear (25).

**Physical Activity**

In a longitudinal study of nutrition and growth, Deheeger et al. (26) investigated the association between physical activity and child development. Physical activity was assessed at the age of 10 years. In spite of a higher energy intake, active children had a lower percentage of fat body mass than nonactive children. A retrospective analysis showed that adiposity rebound occurred later in active children. While physical activity was assessed after the age of adiposity rebound (at 10 years), the higher energy intake and greater lean body mass recorded at all ages in active children, and the results of previous studies showing good tracking of physical activity, suggested that active children at age 10 years were also active at earlier ages. These observations are speculative, but they suggest a role of physical activity on the timing of the adiposity rebound.

**Secular Trends**

Secular trends have appeared for various measures of growth (27). Children are getting larger, becoming taller (attributable mainly to increased leg length), and reaching maturity more rapidly. A longitudinal growth study was initiated in the early 1950s (6,18). Another longitudinal study, following similar protocol (children recruited in health centers in the Paris area) started 30 years later. Mean age at adiposity rebound was $6.2 \pm 1.3$ years in children born in 1955 (6) and $5.6 \pm 1.9$ years in children born in 1985 (20). The trend in age at adiposity rebound is consistent with the secular trend of earlier maturation.

**Nutrition**

*Energy and Nutrient Intakes*

In a longitudinal study of nutrition and growth we investigated the nutritional factors associated with age at adiposity rebound (20). Nutritional intakes were assessed at the
age of 2 years. Correlations were computed between intakes at 2 years of age at adiposity rebound. No association was found between energy intake, percentage of energy from fat or carbohydrate, and age at adiposity rebound. A negative and significant association was found between the percentage of protein intake and adiposity rebound—that is, the higher the percentage of protein consumed at the age of 2 years, the earlier the adiposity rebound.

More recently, Dorosty et al. (16) conducted a similar analysis in a prospective cohort of children followed from birth to 5 years in the United Kingdom. They compared very early (before the age of 3.5 years), early (from 4 to 5 years), and later rebound (after 5 years). They found no evidence of associations between dietary protein or any other dietary variable recorded at the age of 18 months and the timing of the adiposity rebound. They did not confirm the association between protein intake and age at adiposity rebound. They also failed to show any association with either energy or fat intake. A study conducted in Italian children has shown an association between early high protein intake and increased BMI at the time of adiposity rebound (28).

The unexpected results of the French study regarding the association between protein intake and age at adiposity rebound (20) have drawn attention to the inadequate nutrient balance of the infant diet in industrialized countries (29) (Table 1). By the age of 1 year, the infant diet is characterized by high intakes of protein (approximately 4 g/kg body weight or 16% of total energy) and low intakes of fat of about 28%). Protein intake represents about three to four times the protein needs (30). The nutrient imbalance is remarkable because the diet during the first months of life, when human milk is the only food, provides 7% of energy as protein and 54% as fat (9). Paradoxically, the proportion of fat in the infant diet is low at a period of high energy needs. Figure 5 shows the actual nutrient changes from early childhood (20) to adult life (31). The percentage of energy provided by fat increases with age, whereas it should be high in early life and gradually decrease with age.

How would an increased protein intake lead to an early adiposity rebound? Childhood obesity is characterized by accelerated growth and increased stature and lean body mass (19). An early high protein intake may account for these characteristics. These changes in body composition could have occurred through changes in hormonal status (29,32). High plasma, insulin-like growth factor 1 (IGF-1) concentrations and reduced growth hormone secretion (spontaneous or in response to a wide

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From Rolland-Cachera et al., 1999 (29).
FIG. 5. Actual nutrient changes from early childhood (21) to adulthood (31). The percentage of energy provided by fat increases from infancy to adult age, whereas it should be high in early life and decrease gradually with age.

variety of stimuli) are characteristic features of children with simple obesity (33). Nutritional intake affects hormonal status. Fasting decreases serum IGF-1 concentrations and restoration of IGF-1 is directly associated with the energy and protein content of the diet (34). In protein–energy malnutrition, growth hormone levels are raised and the high growth hormone values are not affected by increasing the energy intake. They fall only when protein is added to the diet (35). These results suggest that protein deprivation acts as a stimulus to growth hormone secretion.

We have previously proposed (32) that the altered hormonal status of obese children (high IGF-1 and reduced growth hormone) could be the mirror image of protein deprivation and thus a consequence of an excess protein intake. Indeed, a high protein intake is often recorded in obese children (36–38). It may increase IGF-1 levels, stimulating protein synthesis and cell proliferation in all body tissues. This could account for the characteristic accelerated growth and increased muscle mass of obese children. As IGF-1 promotes the differentiation of preadipocytes into adipocytes (39), a high protein intake may induce hyperplasia in adipose tissue. The early increase in adipocyte number reported in obese children (19) may be responsible for the early adiposity rebound recorded in the obese. In addition, a high protein intake may, at any age, decrease growth hormone levels, thus decreasing lipolysis and promoting the development and maintenance of high fat stores. A high protein intake may also promote an android type of body fat distribution (40) and hence the development of metabolic complications of obesity such as insulin resistance or cardiovascular disease (41).

*Metabolic Adaptation to a Relative Energy Deficit in Infancy*

Over the previous few decades, energy and fat intakes have decreased at a population level (42) in children (43), and even in young children (44,45), while the percentage of energy from protein has increased (44).
Energy intake in 1.5- to 2.5-year-old English children declined from 1,264 to 1,045 kcal/day between 1967 and 1993 (44). At the same time, the percentage of energy obtained from protein increased (43,44). The trend to decreased energy intake is the probable consequence of decreasing energy expenditure in older children but is less likely in young children, in whom a reduced energy intake may be related to the composition of the diet. A low fat intake reduces the energy density of the diet and may reduce the total energy intake (46). In addition, a low-fat/high-protein diet can reduce energy intake, as young children prefer flavors associated with high dietary fat (47) and because of the satiating effect of protein (48). Mean energy intakes in infants are generally below estimated requirements (49).

A relative energy deficit could be generated by both a low fat intake and by the increased energy expenditure required for the high protein-associated accelerated growth. The BMI pattern associated with an early adiposity rebound (low fatness in infancy, followed by an early adiposity rebound and subsequent increased fatness) could be explained by the dietary changes with age (Fig. 5); that is, a low-fat/high-protein intake during the first years of life, followed by high-fat intake in later childhood.

The low energy density of the infant's diet could create a relative energy deficit and induce metabolic adaptations (thrifty metabolism). These adaptations may have adverse effects when, at later ages, children eat an adequate or more abundant high-fat diet. Similar patterns of consuming low-fat weaning foods, followed by a more adequate diet, may also occur in developing countries.

The fatness pattern of low BMI followed by increased BMI after the adiposity rebound is consistent with other observations. Low weight for length in early life has been found in men and women with syndrome X (50), and it has been suggested that changes from poor to abundant nutrition could increase the risk of developing diabetes and ischemic heart disease (51). The hypothesis of an association between risk factors and accelerated growth promoted by an excessive early protein intake is also consistent with the association between increased stature, particularly leg length (52), early maturation (2), and health risks. Overweight at adolescence, whatever the weight status by adulthood, is associated with increased risk factors for later diseases (53), showing the importance of the period of growth. Accelerated growth may play a major role in the risks associated with obesity.

CONCLUSIONS

The increasing prevalence of obesity has stimulated interest in the identification of early markers for this condition. Age at adiposity rebound, recorded on individual BMI curves, has been found to be associated with adult weight status. Numerous studies have reported that an early adiposity rebound is associated with an increased risk of obesity.

Some studies have investigated the factors associated with age at adiposity rebound. An early adiposity rebound is associated with advanced skeletal maturity and parental obesity, and there is evidence to suggest an association with a sedentary lifestyle. No association was recorded with socioeconomic status. Studies on nutritional intakes have failed to show any association between energy or fat intake in
early childhood and age at adiposity rebound. One study reported an association with a high protein intake at the age of 2 years.

The early adiposity rebound recorded in obese individuals suggests that the determinants act in early life. The results of nutritional studies suggest that infant diets in many industrialized countries are not adapted to the specific needs of children during the different phases of growth.

More research on the factors associated with age at adiposity rebound may help to identify factors promoting overweight and may be useful in preventing the development of obesity at an early stage of life.

SUGGESTIONS FOR FUTURE RESEARCH ACTIVITIES IN THE FIELD

Associations between adiposity rebound and environmental and genetic factors should be investigated. Previous investigations were conducted in populations with homogeneous weight status, feeding practices, and physical activity. The association between adiposity rebound and environmental factors should be conducted in samples that include a broader range of BMI and more varied behaviors.

Mean BMI pattern reflects changes in fatness, but changes in body composition at the time of adiposity rebound may vary according to various factors (e.g., sex, nutrition, genetics, physical activity). Studies on changes in body composition in relation to age at adiposity rebound could be of interest.

An early adiposity rebound and a high BMI by the time of the adiposity rebound relate to different BMI patterns—these two indicators are independent predictors of adult fatness. Comparisons of the association of these indicators with biological markers and long-term health risks should be investigated.

KEY POLICY IMPLICATIONS

Research on adiposity rebound has stressed the role of early determinants of adult obesity and inadequate nutritional intakes in early life.

The combination of decreasing energy intake and increasing prevalence of obesity suggests that rather than energy restriction, improvements in nutrient balance—taking into account the specific needs at the different periods of growth—and promotion of an active lifestyle should be encouraged.

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DISCUSSION

*Dr. Dallou*: This is a fascinating phenomenon. In relation to protein intake, a correlation coefficient of 0.2—that is, an $r^2$ value of 0.04—means that protein explains less than 5% of the total variation. Even if that's significant, it's a very weak determinant. What was the correlation coefficient in the other study in which there was also a positive relation, the one by Scaglioni (1)? Was it stronger?

*Dr. Rolland-Cachera*: Nutrition is just one of many aspects of lifestyle and it is obviously difficult to find associations between nutritional intake and obesity. We have summarized 176 correlations from 34 studies. The main result was that there was no association between nutritional intake and adiposity. I agree that the correlation in your study is very weak; nevertheless, it is still there, and that isn't the case with the other nutrients. It is true that the correlation
is too weak to allow us to say that protein increases the risk of obesity, but it does open a window onto a research area.

The study by Scaglioni was not linked to adiposity rebound but to BMI level. The association was weak also.

Dr. Singhal: What is the genetic contribution to the adiposity rebound? Have there been any studies in twins to indicate whether the phenomenon is related to nutrition or to genetics?

Dr. Rolland-Cacher: I don’t know of any twin studies, but an association between adiposity rebound and parental BMI suggests that there might be a genetic component, as does the difference between countries.

Dr. Freedman: You showed a slide in which there were eight curves representing different countries. A low BMI at age 2 was associated with an earlier adiposity rebound and then a high BMI at age 18. Would you find the same thing if you were to study individuals? Surely there would not be an inverse association between BMI at age 2 and BMI at age 18, wouldn’t there?

Dr. Rolland-Cacher: Yes, children with an early adiposity rebound tend to have a lower BMI before the rebound. To illustrate that, I described the case of a very thin child with an early adiposity rebound who later became fat. What I really wanted to stress was that there may not be only one process of obesity. We should look more carefully at these individual BMI patterns. They might be linked with different determinants—genetic, nutritional, and so on. The U-shaped birth weight curve in relation with risk factors has been mentioned previously. Adiposity rebound may be related to this phenomenon. We should really be looking at the patterns of development that are associated with different risk factors.

Dr. Bellizzi: In your correlations with nutrients, have you ever looked at carbohydrates, and in particular complex carbohydrates?

Dr. Rolland-Cacher: There was no association at all. The association with fat was also nonsignificant, but the trend was in favor of an inverse trend between fat intake in infancy and adiposity at older ages. This is consistent with a beneficial effect of human milk. It is important that advice to decrease fat intake in the population as a whole exclude infants.

Dr. Dulloo: From your graph of adiposity rebound in different countries it seemed that the phenomenon occurs in Senegal and Burundi but not in India. What is your explanation for that?

Dr. Rolland-Cacher: I think the explanation probably lies in the lifelong low intakes of food in the Indian population.

Dr. Cole: I would like to comment on the interpretation of the adiposity rebound, putting it in a different context. I asked myself the question, “Why should an early age at adiposity rebound predict later obesity?” The answer is because at about this age, the median value of the BMI goes down and then goes up again. For this reason, what is in fact an upward centile crossing has the appearance of an early adiposity rebound, and if you are crossing centiles upward, this will predict later obesity. I can illustrate this with two examples [Figs. 6 and 7].

The first example, from Dr. Rolland-Cacher’s French study, is that of a child reported to have a late adiposity rebound. BMI centiles are crossed upward until about age 1 year, and then they fall. It’s hard to say exactly when the adiposity rebound is but it’s certainly late. If you plot that same chart using the BMI z score (right), each centile curve becomes a horizontal straight line, as I explained in an earlier discussion, so it’s easier to see what is going on. You can now see that there is upward crossing of the centiles until about 1 year, and then a more or less linear fall until the age of around 10 or 12, so what looks like a late adiposity rebound is simply the downward centile crossing of the BMI.

The second example is that of a child with an extremely early adiposity rebound, at about age 2. If you look on the right hand side of the graph, you can see that the early adiposity rebound is again just simply upward centile crossing. So the child is crossing centiles more or
FIG. 6. Late adiposity rebound and centile crossing downward.
FIG. 7. Early adiposity rebound and centile crossing upward.
less linearly from the age of 1 year to the age of 6 years, after the same centile is maintained.

If you understand that “adiposity rebound” is simply a statement about centile crossing, then three things follow. First, an earlier adiposity rebound will be associated with a low BMI, because you are starting at a low centile and crossing upward. Second, it will be correlated with a high BMI later. Third, whether your BMI is high or low early will correlate with whether your BMI is high or low later.

Dr. Rolland-Cachera: I agree that we should compare data in different ways to gain a better understanding of the phenomenon. Thank you for this additional information.

REFERENCES