

Nutrient balance and body composition

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Summary — The prevalence of obesity in industrialized countries is increasing in spite of decreased energy and fat intakes. This trend might be mainly a consequence of a decline in energy expenditure. It is suggested here that it might also be accounted for by the increasing proportion of protein in the diet, affecting the hormonal status. The nutrient imbalance is particularly apparent in early childhood, when a low fat and high protein diet is not justified because of high energy needs for growth and because it is the period of high rate of myelinization of the nervous system. At later ages, the proportion of fat exceeds the recommended level, and the protein intake remains high. A diet containing less animal and more vegetable products would reduce both protein and saturated fat excesses and could help decrease metabolic risk factors.

body composition / nutrition / macronutrients / obesity / humans

Résumé — **Équilibre alimentaire et composition corporelle.** La prévalence de l'obésité ne cesse d'augmenter dans les pays industrialisés, bien que depuis une vingtaine d'années les apports en énergie et en lipides diminuent régulièrement. L'augmentation de l'obésité est sans doute principalement la conséquence de la baisse des dépenses énergétiques, mais elle peut également s'expliquer par une modification de la répartition des nutriments dans la ration. Les taux élevés de protéines relevés dans les enquêtes récentes pourraient influencer le statut hormonal. Les apports faibles en graisse et élevés en protéines relevés chez les jeunes enfants ne sont pas adaptés à leurs besoins. Une alimentation riche en graisse, comme le lait maternel, est mieux adaptée aux besoins élevés en énergie au début de la vie et pendant la période de myélinisation rapide du système nerveux. Paradoxalement, le taux de lipides augmente ensuite avec l'âge et dépasse les recommandations à partir de l'âge de 4 ans. Les apports protéiques restent élevés à tout âge, et les taux de glucides sont insuffisants à partir de 4 ans. Une alimentation contenant moins d'aliments d'origine animale et plus de produits végétaux, permettrait de réduire, chez l'adulte, les taux élevés de protéines et de graisse, en particulier de graisses saturées, et permettrait sans doute de réduire les risques de maladies métaboliques.

composition corporelle / nutrition / macronutriments / obésité / humains

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INTRODUCTION

Relationships between adiposity and food intake have been widely investigated. It is often suggested that high energy, fat or carbohydrate intakes could increase the risk of obesity. However, no clear epidemiological evidence emerges from the literature to account for these suggestions (Stefanick et al, 1959; Keen et al, 1979; Kromhout et al, 1983; Rolland-Cachera et al, 1990). Moreover, an unexpectedly large number of studies report a positive association between the proportion of proteins in the diet and adiposity (Frank et al, 1978; Fehily et al, 1984; Rolland-Cachera and Bellisle, 1986; Valoski and Epstein, 1990; Slattery et al, 1992; Spyckerelle et al, 1992). As most studies are cross-sectional, these results are often interpreted as a consequence of food restriction in the obese. Indeed, such studies can hardly explain the development of obesity. In order to elucidate the relationships between nutritional intake and fatness development, longitudinal studies conducted during growth are more appropriate. In the present paper, we analysed the pattern of growth in children who became normal or overweight adults as well as the nutritional factors that could account for abnormal growth processes in the obese.

Development of growth parameters

Mean changes

Body weight increases with age, mainly as a consequence of increased stature. It does not reflect fatness variations. The weight/height² body mass index (BMI) development is very different from weight development. As with skinfold thickness, a measure of subcutaneous adiposity, the BMI increases during the first year of life, decreases between 1 and 6 years of age and subsequently increases till the end of

growth (Sempé et al, 1979; Rolland-Cachera et al, 1982). Before the age of 6 years, adiposity variations correspond to adipocyte size variations (an increase during the first year followed by a subsequent decrease), while after the age of 6 years, increased adipose tissue corresponds to increased adipocyte number (Knittle et al, 1979) (fig 1).

Changes in growth pattern recorded in the adipose tissue also occur in various other tissues. For example, the period of low increase in adipocyte number corresponds to the period of steep decrease in height velocity, while a high increase in adipocyte number corresponds to the period of increased height velocity. These changes correspond to changes in hormonal status (Rolland-Cachera, 1995). During the first years of life, when the adipocyte number is stable and height velocity decreases, the insulin-like growth factor 1 (IGF1) level is low (Rosenfeld et al, 1986), while subsequently, when both adipocyte number and height velocity increase, IGF1 increases, suggesting a relationship between these developments.

Individual patterns

Individual development in adiposity is characterized by a low tracking from early childhood to adulthood. In particular, most fat infants do not remain fat during growth (Poskitt and Cole, 1977). These changes of weight status can be clearly understood by examining BMI patterns. An increase in BMI (the 'adiposity rebound') occurs by the age of 6 years on average, but can take place earlier or later (Rolland-Cachera et al, 1984). A fat child at the age of 1 year can join the average BMI after a late adiposity rebound, while a thin child can become overweight at adolescence after an early adiposity rebound (fig 2). An early adiposity rebound reflects advanced maturation (Rolland-Cachera et al, 1984).

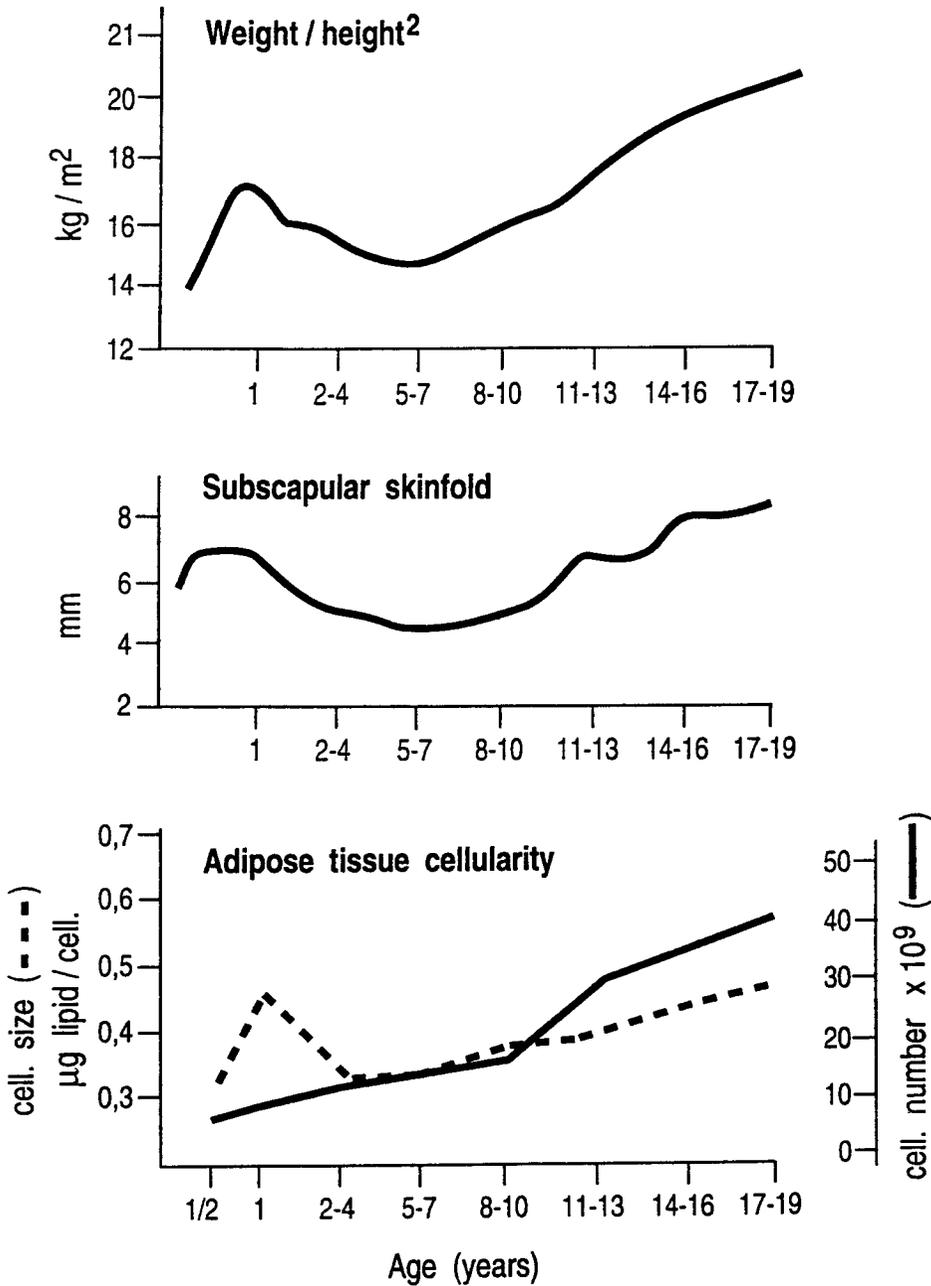


Fig 1. Development of fatness as assessed by BMI and skinfolds and changes of adipose tissue cellularity (from Rolland-Cachera et al, 1982; Sempé et al, 1979; Knittle et al, 1979).

Advanced maturation (Garn and Clark, 1975) and an early adiposity rebound are strong characteristics of childhood obesity.

Factors influencing adiposity

Chronic protein-energy deficit affects growth by slowing down height velocity,

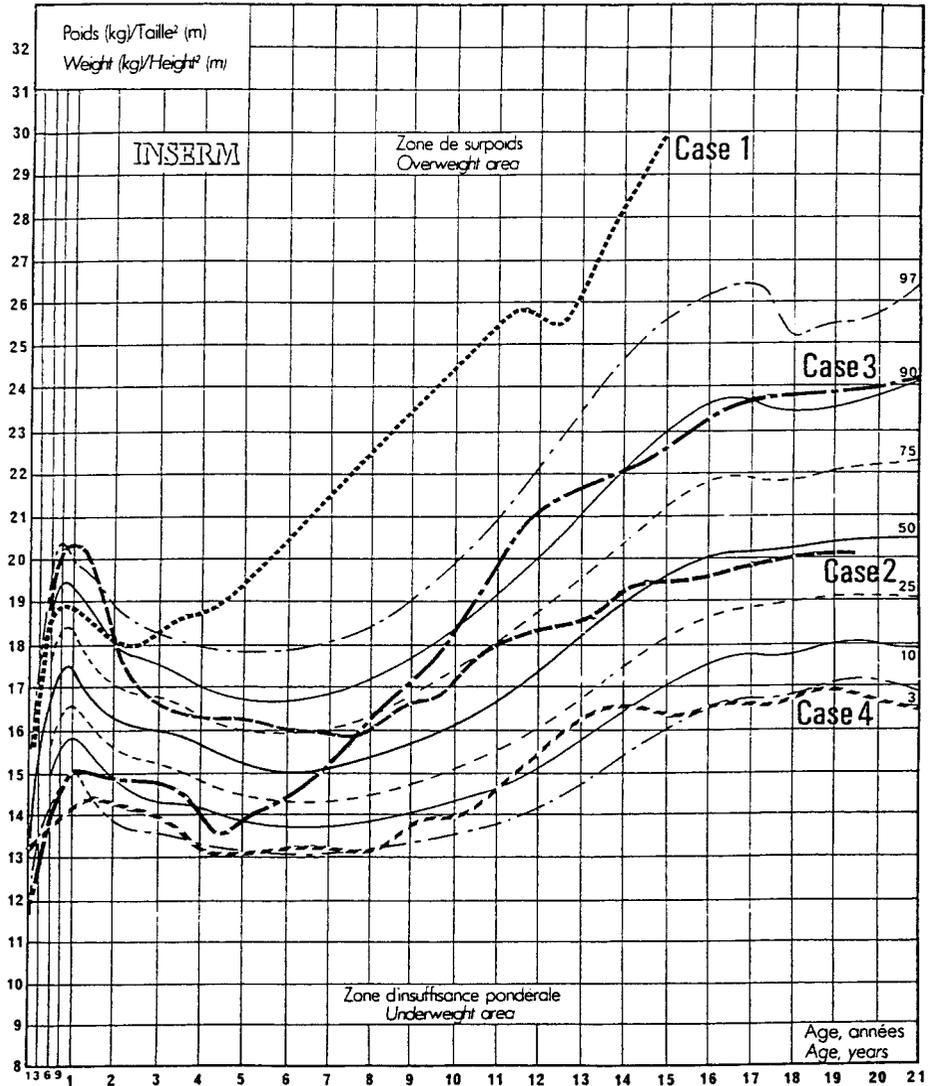


Fig 2. Four examples of BMI development: case No 1, fat child at 1 year, remained fat after an early adiposity rebound (2 years); case No 2, fat child at 1 year, did not stay fat after a late adiposity rebound (8 years); case No 3, lean child at 1 year, did not stay lean after an early adiposity rebound (4.5 years); case No 4, lean child at 1 year, remained lean after a late adiposity rebound (8 years) (after Rolland-Cachera et al, 1987).

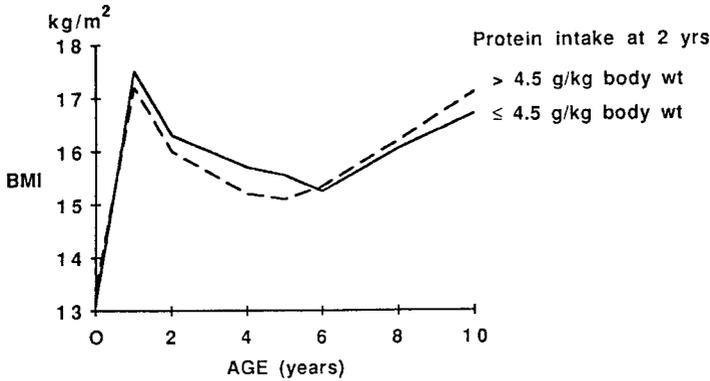


Fig 3. BMI development in two groups constructed on the basis of protein intake (g/kg body weight) at the age of 2 years. This figure illustrates the association between protein intake at the age of 2 years and age at adiposity rebound: the higher the protein intake at the age of 2 years, the earlier the age at adiposity rebound (Rolland-Cachera et al, 1995).

but at the same time, weight-for-height can be normal. In the case of severe energy deficit, weight-for-height will also be affected. By contrast, in industrialized countries, where food is easily available, children grow taller and heavier from generation to generation (Eveleth and Tanner, 1990). In a longitudinal study of nutrition and growth, we examined the influence of early nutrition on growth processes. As expected, height velocity was associated with high protein intake early in life (Rolland-Cachera et al, 1991) but, unexpectedly, an early adiposity rebound, as well as a subsequent high body fatness, were also associated with high protein intake in early life (Rolland-Cachera et al, 1995). These associations are illustrated in figure 3 presenting the BMI development in two groups of children constructed on the basis of protein intake at the age of 2 years. At first, fatness decreased more rapidly in the high protein intake group, but subsequently exceeded that of the other group after an early adiposity rebound.

Given that protein intake promotes IGF1 (Clemmons and Underwood, 1981) and that

IGF1 promotes adipocyte multiplication (Ailhaud et al, 1992), our results suggest that a high protein intake in early life might promote a precocious multiplication of adipocytes, responsible for the characteristic early adiposity rebound in the obese. Indeed, IGF1 levels are high in obese children (Roskamp et al, 1987). In addition, we have suggested (Rolland-Cachera, 1995) that in contrast with the situation of protein-energy undernutrition, which promotes an increase in the lipolytic growth hormone (GH) level, protein excess could promote a decrease in GH level, contributing to the development of obesity. Other factors affect the hormonal status. Exercise decreases IGF1 (Smith et al, 1987) and increases GH levels (Jungmann et al, 1991). Consequently, a sedentary lifestyle may have a similar influence to that of protein excess on the hormonal status, promoting accelerated growth, decreased lipolysis and the development of obesity.

A high protein intake is often reported in obese children (Frank et al, 1978; Valoski and Epstein, 1990; Rolland-Cachera and Bellisle, 1986), but this excess is not

usually thought to be a causal factor of obesity development. Nevertheless, this excess may well account for the accelerated growth usually reported in obese children (Garn and Clark, 1975; Forbes, 1977). In addition, over the last decade or two, the prevalence of obesity has increased steeply in industrialized countries, in spite of no reported increase in energy intake, and a general decrease in fat intake (Nicklas et al, 1993; Prentice and Jebb, 1995; Rolland-Cachera et al, 1996b; Heini and Weinsier, 1997). This trend is the probable consequence of decreasing energy expenditure, but might also be due to a high proportion of protein in the diet, which can compromise the benefits obtained from fat restriction. In studies where adiposity is assessed in different sites, protein excess is associated with fat located at the abdominal level, rather than with fat located in other sites (Rolland-Cachera et al, 1996a), suggesting an association between protein excess and metabolic complications of obesity such as insulin resistance and cardiovascular diseases (Vague, 1956; Björntorp, 1996).

A high proportion of protein in the diet can be the consequence of a high consumption of animal products or else a restriction of fat intake. Fat restriction is not justified during early life, when energy needs for growth are very high (Holliday, 1978), and because it is the period of high rate of myelination of the nervous system (Agostoni et al, 1992). Human milk contains a moderate amount of proteins (7%) and a high proportion of fat (50%). This composition may account for the beneficial effects of breast feeding on growth processes (Rolland-Cachera, 1995). By the age of 5–12 months, children consume more than 15% of energy from proteins and less than 30% of energy from fat (Deheeger et al, 1996; Räsänen et al, 1985). In this age range, it is recommended that proteins represent 5–6% of the total energy intake (FAO, WHO, UNU, 1985). In the French study (Deheeger et al, 1994), 10-month-old children consumed

27.6% of their energy from fat. The percentage of fat was lower than 30% in 72% of infants, and was lower than 25% in a quarter of the population. The proportion of fat in the diet of infants seemed low. Moreover, the ratio of polyunsaturated/saturated fat was also low. Paradoxically, the proportion of fat in the diet is low in early life and increases with age, whereas it should be high in early life and then should gradually decrease. In later childhood, and in adulthood, the proportions of protein and fat in the diet are usually high. Energy provided by proteins is close to 17% (Hercberg et al, 1991) instead of the recommended 12%, and energy provided by fat is usually greater than 35%. An excess of animal products in the diet may account for this nutrient imbalance. Less animal and more vegetable products would decrease both protein and saturated fat and favor carbohydrates in the diet. Such a nutrient balance is a characteristic of the Mediterranean diet, whose protective effects on various pathologies has been widely demonstrated (Keys, 1995; Willett et al, 1995).

CONCLUSION

An increased prevalence of obesity has been observed in most industrialized countries. Over the last decade or two, however, there has been no increase in energy or fat intake to account for this trend. The decline in energy expenditure is very probably the major factor accounting for this apparent paradox. However, other factors may play a role. The increased proportion of proteins in the diet may affect hormonal status. The possibility of a relationship between excess protein intake and obesity is consistent with the observation that obese children tend to be taller and have a larger muscle mass. A reduced protein and high fat diet (such as human milk) is probably more adapted for infants whose energy needs are very high and because fat is necessary during the phase

of high rate of myelinization of the nervous system. Fat intake restrictions are not justified during the first years of life and excessive protein intake should be avoided at all ages. A diet containing less animal and more vegetable products would decrease both protein and saturated fat intakes. Improved nutrient balance, taking into account the variations in specific needs at different ages, and more exercise should be the main strategies to decrease the prevalence of obesity in industrialized countries.

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