Growth-Healthy status and active food model in pediatrics

International Journal of Obesity (2005) 29, S14–S18
 0307-0565/05 $30.00
www.nature.com/ijo

PAPER

Growth: healthy status and active food model in pediatrics

NC Battistini[1]*, M Malavolti[1], M Poli[1] and A Pietrobelli[2]

[1]*Human Nutrition Chair, Modena and Reggio Emilia University, Italy; and* [2]*Pediatric Unit, Verona University Medical School, Verona, Italy*

OBJECTIVE: The brain integrates peripheral signals of nutrition in order to maintain a stable body weight. Nutritional status defined as the results of introduction, absorption, and utilization of the nutrients could be interpreted with the base of the relationship between nutritional status and healthy status. In this view, energy balance, body function, and body composition are three entities correlated to each other to the healthy status.
AIM: To discuss the nutritional status in relation with healthy status, and its relationship with growth and nutrients.
METHODS: A review of the available literature on food patterns and active food model was carried out.
RESULTS: In the reviewed studies, strategies that could offer promising results to prevent overweight and obesity were discussed, in particular in the light of functional foods that effect energy metabolism and fat partitioning.
CONCLUSION: At this moment it is necessary to proactively discuss and promote healthy eating behaviors among children at an early age and empower parents to promote children's ability to self-regulate energy intake while providing appropriate structure and boundaries around eating.
International Journal of Obesity (2005) **29**, S14–S18. doi:10.1038/sj.ijo.0803077

Keywords: pediatric; healthy food; growth; active food model

Introduction

The brain integrates peripheral signals of nutrition in order to maintain a stable body weight.[1] However, this physiological system is not simple and easy to describe. Brain centers including the hypothalamus, brainstem, and reward centers using neuropeptides are able to regulate energy homeostasis. Peripheral signals of adiposity such as leptin, insulin, and adiponectin 'represent' the nutritional status of the body and could influence these circuits. Peripheral signals from the gastrointestinal tract (ie, ghrelin, pancreatic-polypeptide, proglucagon products, cholecystokinin) modulate these pathway, and the final product is either appetite stimulation or satiety effects.[1]

The definition of nutritional status that is defined as the results of introduction, absorption, and utilization of the nutrients could be interpreted with the base of the relationship between nutritional status and the healthy status. In this view, energy balance, body function, and body composition are three entities correlated to each other (Figure 1).[2]

*Correspondence: Professor NC Battistini, Applied Dietetic Technical Science Chair, Modena and Reggio Emilia University, Via Campi 287, Modena 41100, Italy.
E-mail: battisti@unimore.it

Nutrients are usually divided into macro- and micronutrients in relation to their presence in the normal body. Macronutrients (ie, protein, lipids, and carbohydrates) have energetic and structural functions, while micronutrients (ie, vitamins and minerals) do not have energetic functions; they could however take part in structural functions but they have in particular homeostatic functions.[2] Also, water is another fundamental element of the human body, with no energetic function, but it is by far the most abundant of the constituents of the body. The percentage of body weight as water varies from 70 to 75% at birth to less than 40% in obese adults.[3]

The aim of this paper is to discuss the nutritional status in relation to the healthy status, and its relationship with growth and nutrients.

Growth

Significant changes, both external and internal, occur during the years spanning infancy through young adulthood.[4] External changes (ie, body proportion, height, weight, and pubertal status) are measured appropriately using physical examination and anthropometric measurements, while internal changes (ie, body composition and hormonal status) require specific and specialized tests. Compared with

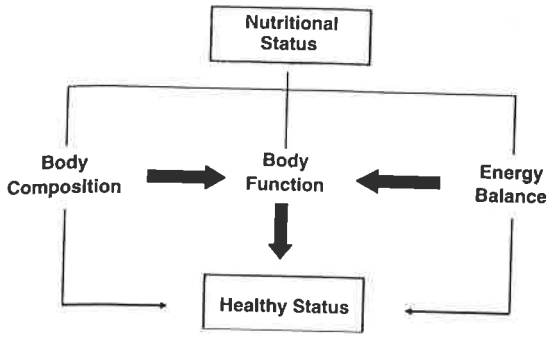

Figure 1 Functional definition of active healthy status in relation with body function, energy balance, and body composition. Adapted from Bedogni *et al.*[2]

adults, children have much greater variation in body composition attributable to growth and development from infancy to adolescents. We need to remember that our population is not 'mature' and growth is the first factor that influences the pediatric body composition measurements. Boys and girls grow differently and it is fundamental to assess this natural aspect when we measure young patients. In fact a certain amount of fat is fundamental in girls to reach or develop puberty.[5] During growth fat-free mass hydration changes from birth (80.6%) to adult age (73%), and it is used an indicator of growth and cellular development.

Early identification of adolescents at risk for obesity and its related metabolic complications requires reliable, simple, and specific measures of excess body fat for this age group.[6] Waist circumference seems to be the best simple anthropometric predictor for the screening of the metabolic syndrome in children and adolescents.[6,7] Recently Cole *et al*[8] compared the performance of different measures of body mass index (BMI) measured longitudinally in a group of kindergarten children over a 9-month period. This issue has implications for short-term studies targeting the prevention of weight gain in children at risk for obesity or the treatment of children that are already obese.[8] They concluded that it is fundamental to look for an appropriate method to assess adiposity change when following children at risk of obesity.[8]

It is important during growth to assess the effect of energy expenditure, including resting metabolic rate, total energy expenditure, and activity energy expenditure, as well as substrate oxidation (respiratory quotient). Energy intake and energy expenditure are difficult to assess, and many factors have influenced the measurements *per se*. However, it is to measure the fat intake that it is critical in regulation of energy intake and body weight.[1,3,4]

The fundamental issue is also to know body composition changes during pregnancy in order to determine the safety and development of the fetus and the newborn. Data on fat and water gain during pregnancy would be a beneficial adjunct to regular prenatal care. Since high gestational fat gain could contribute to obesity development and consequently is not particularly beneficial to fetal growth,

Table 1 Changes in body composition during pregnancy and recommended weight gain based on pre-pregnancy weight

Prepregnancy weight	*Underweight (BMI < 19.8)*	*Normal weight (BMI 19.8–26)*	*Over weight (BMI > 26–29)*	*Obese (BMI > 29)*
Recommended weight gain (kg)	12.5–18	11.5–16	7–11.5	7–9.2
Weight gain weeks 14–37 (kg)	12.6	12.2	11.0	8.7
Fat gain weeks 14–37 (kg)	4.8	3.9	2.8	0,2

BMI = body mass index (kg/m^2). Based on Lederman *et al*[9], and IOM Institute of Medicine[10].

reducing maternal fat gain is highly important.[9] Table 1 reported the recommended weight gain during pregnancy based on prepregnancy weights.[9,10]

Kramer and colleagues showed for the fist time the association between breastfeeding and protection against overweight.[11] Recent meta-analyses[12,13] showed that breast-feeding has a protective effect against childhood obesity due to several potential mechanisms. First of all, breast-fed infants control the amount of milk they consume and it implies that infants could learn earlier the capacity to respond to satiety rather than infants who are bottle fed that are encouraged by the parents to finish their bottle. Also, high protein intake of formula feed could influence higher insulin secretion.[14] Finally, leptin levels, the hormone that controls body fatness, were lowest in subjects who received breast milk.[15] We may conclude that breastfeeding could be promoted as one strategy among others to prevent overweight in children.

On the other hand, it is important to also consider that maternal control during feeding may promote childhood obesity or disordered eating patterns.[16] It is well known that restricting children's access to preferred food increases the probability that children will eat those foods when given free access.[17] Faith *et al*[18] using the National Longitudinal Survey of Youth in a cohort of 3–6-y-old children showed that mother allotted child food choice and child eating compliance were associated with child BMI percentile. This finding is fundamental in order to underline that maternal feeding practices are associated with variations in child weight status, and it is important when we treat pediatric overweight subjects.[18]

Finally, it is fundamental to underline the potential merits of studying related individuals (ie, twins and siblings) when conducting pediatric treatment/prevention studies on obesity.[19] In fact, these studies have the potential to inform us about between-child variations in treatment response, providing unique information that cannot always be gleaned by studying unrelated children.[20]

Food patterns

Definition of food/eating patterns is related to patterns of meals, frequency of meal consumption, snaking habits,

beverage consumption, portion size, and daily dietary quality. It is well known that individuals who consumed the highest variety of food from all food groups had the most adequate nutrient intake.[21] In a healthy diet, carbohydrates should constitute 55–65% of daily caloric intake, protein should range from 8% on 6–10 y of age to 13–15% on 13–14 y period, and total lipid should not exceed 30% of daily intake.[22,23] It has been observed that dietary variety obtained from fruit and vegetable consumption was inversely associated with body fatness, while dietary variety obtained by sweets, snacks, and carbohydrates was directly associated with body fatness.[24] It was recently shown that trends in childhood nutrition over the past 20–30 y, such as changes in fast food and soft drink consumption, could explain the increasing prevalence of overweight in children and could critically address the issues contributing to these changes in nutrient intake, knowing the link between childhood obesity and metabolic abnormalities.[25]

Popkin and Gordon-Larsen[26] described the 'nutrition transition' closely related to the demographic and epidemiological transition. The diets of the developing world are shifting rapidly, particularly with respect to fat, caloric sweeteners, and animal source foods.[27] Also, in developed countries daily energy intake appears to be increasing in particular from energy-dense, nutrient-poor foods and an increase in snacks.[28] More meals are being consumed away from home and portion size offered in restaurants has increased significantly.[29] On the basis of several studies, increasing fruit and vegetable intake, reducing sweet drink consumption, and decreasing portion sizes are strategies that could offer promising results to prevent overweight and obesity.[30–32]

Active food model

There is evidence that children's diet is still too high in fat and too low in complex carbohydrates, and it is difficult to reduce the intake of energy-dense food, especially dietary fat intake.[16] Recently, functional foods that effect energy metabolism and fat partitioning were suggested as adjuncts to a dietary approach to body weight control.[33] Among functional foods we briefly reviewed beverages (ie, water, tea, and milk) that could attempt to prevent weight gain and/or promote weight loss.

Water

Water is essential for life, serving as a solvent for biochemical reaction and as a transport media. It is often neglected because the volume of total body water is well regulated in health. A 15% decrease in body water due to dehydration is life threatening,[34] and it is fundamental to keep in mind this information in pediatrics because even a small change in total body water can produce a measurable change in body weight.

Table 2 Distribution of water in the reference young male

Model	*Compartment*	*kg*	*%Total body water*
Molecular	total body water	40	100
Cellular	Intracellular	23	57
	Extracellular	17	43
Tissue	Intracellular	23	57
	Plasma	2.8	7
	Interstitial	8.0	20
	Bone	2.8	7
	Dense connective tissue	2.8	7
	Trans-cellular	1.6	4

Adapted from Snyder *et al.*[36]

Water is an important constituent at the molecular, cellular, and tissue levels according to the classical models describing body composition.[35] In fact, at the molecular level, water constitutes a well-defined component, hydrogen oxide. At the cellular level, water is found in two compartments: the body cell mass and the extracellular fluid compartment. At the tissue level, water is present in five compartments: intracellular water, plasma water, interstitial water, dense connective tissue water, and *trans*-cellular water. Table 2 summarizes the distribution of water and each of the levels in a 'young reference' male.[36]

Water is the food component that has high capacity to reduce energy density as it increases weight without adding energy.[37]

Tea

It is well known that tea polyphenols are antioxidants that could reduce LDL-cholesterol oxidation.[38] Several studies analyzed the relationship between tea consumption and energy expenditure and found that tea could positively influence energy expenditure, both 24 h and fasting, and fat oxidation in normal-weight adults.[39,40] More studies are needed to confirm the role of tea in weight maintenance, but there are promising results from the published studies.[39,40] Two important factors, however, must be taken into account, the caffeine content in the tea and the addition of sugar. Both factors could reduce the potential of tea as a functional food for weight maintenance.

Milk

It has been hypothesized that high Ca^{2+} diets protect against fat gain by creating a balance of lipolysis over lipogenesis in adipocytes.[41] An increase in dietary calcium could reduce 1,25-dihydroxyvitamin D concentrations, resulting in the downregulation of calcium transfer into adipose and pancreatic cells. It is important to underline that in the adipocytes, a reduction in intracellular calcium decreases fatty acid synthetase transcription that results in

lipogenesis decrease and lipolysis increase.[42] In pancreatic cells, a reduction of intracellular calcium concentration decreases insulin output and the result is a lipogenesis reduction and lipolysis improvement in adipocytes.[42] The final outcome is that high intakes of calcium are associated with lower levels of fat mass.[43] It is also important to note that the beneficial role of dietary Ca^{2+} in weight management is markedly greater from dairy *vs* nondairy sources of Ca^{2+}.[41] Several studies confirmed the inverse relationship between calcium intake and body weight in children [44] that supports the role of dietary calcium and dairy foods in controlling excess adiposity.[45] Recently, Zemel *et al*[46] showed in young adults that three six-ounce servings of yogurt in the daily diet markedly augments fat loss secondary to modest energy restriction and results in a selective increase in the loss of central adiposity. On the other hand, Lappe *et al*[47] found that calcium-rich diets do not cause excessive weight gain in pubertal girls, but do contribute positively to overall nutrition.

Discussion

The fundamental importance of changing the nature of the environment toward greater inducement of obesity has been reported in the WHO Technical Report[48] as follows: 'Changing in the world food economy has contributed to shifting dietary patterns, for example, increased consumption of energy-dense diets high in fat, and low in unrefined carbohydrates. These patterns are combined with a decline in energy expenditure that is associated with a sedentary lifestyle'.[48]

Food insecurity may contribute to the inverse relation of obesity prevalence with socioeconomic status,[49] as well as lack of consistent access to healthy food choices, particularly fruit and vegetables. Absence of family meals is associated with lower fruit and vegetables consumption as well as consumption of more fried food and carbonated beverages.[49]

At this moment it is necessary to proactively discuss and promote healthy eating behaviors among children at an early age and empower parents to promote children's ability to self-regulate energy intake while providing appropriate structure and boundaries around eating.

Conclusion

It is important for the clinician to know nutritional factors, energy intake, and composition of the diet, nutrition and hormonal status, food preference, and behavior, and the influence of non-nutritional factors.[50] When these are all taken together with an accurate and precise body composition assessment, it may be possible to control growth process and to predict adult status in order to reduce the risk factors of various diseases.[51] Lissau *et al*[52] mentioned that there are essentially six relevant levels, which could be involved in the prevention of child and adolescent obesity: family (child, parents, siblings, etc), schools, health professionals, government, industry and media. Therefore, it is incumbent on the pediatric community to take the leadership role in prevention and early recognition of pediatric obesity.

References

1 Wynne K, Stanley S, McGowan B, Bloom S. Appetite control. *J Endocrinol* 2005; **184**: 291–318.
2 Bedogni G, Borghi A, Battistini NC (eds). *Principi di valutazione dello stato nutrizionale*. EDRA Medical Publishing & New Media: Milan, Italy; 1999.
3 Forbes GB. Methods for determining composition of the human body. With a note on the effect of diet on body composition. *Pediatrics* 1963; **29**: 477–494.
4 Sopher A, Shen W, Pietrobelli A. Pediatric body composition. In: Heymsfield SB, Lohman TG, Wang ZM, Going SB (eds). *Human body composition*. Human Kinetics: Champaign, IL; 2005. pp 129–140.
5 Fomon SJ, Haschke F, Ziegler EE, Nelson SE. Body composition of reference children from birth to age 10 years. *Am J Clin Nutr* 1982; **35**: 1169–1175.
6 Rodriguez G, Moreno LA, Blay MG, Blay VA, Garagorri JM, Sarria A, Bueno M. Body composition in adolescents: measurements and metabolic aspects. *Int J Obes Relat Metab Disord* 2004; **28**: S54–S58.
7 Moreno LA, Pineda I, Rodriguez G, Fleta J, Sarria A, Bueno M. Waist circumference for the screening of the metabolic syndrome in children. *Acta Paediatr* 2002; **91**: 1307–1312.
8 Cole TJ, Faith MS, Pietrobelli A, Heo M. What is the best measure of adiposity change in growing children: BMI, BMI%, BMI z-score or BMI centile? *Eur J Clin Nutr* 2005; **59**: 419–425.
9 Lederman SA, Paxton A, Heymsfield SB, Wang J, Thornton J, Pierson Jr RN. Body fat and water changes during pregnancy in women with different body weight and weight gain. *Obstet Gynecol* 1997; **90**: 483–488.
10 IOM (Institute of Medicine), National Academy of Sciences. *Nutrition during pregnancy*. National Academy Press: Washington, DC; 1990.
11 Kramer MS. Do breast-feeding and delayed introduction of solid foods protect against subsequent obesity? *J Pediatr* 1981; **98**: 883–887.
12 Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics* 2005; **115**: 1367–1377.
13 Arenz S, Ruckerl R, Koletzko B, von Kries R. Breast-feeding and childhood obesity — a systematic review. *Int J Obes* 2004; **28**: 1247–1256.
14 Scaglioni S, Agostoni C, Notaris RD, Radaelli G, Radice N, Valenti M, Giovannini M, Riva E. Early macronutrient intake and overweight at five years of age. *Int J Obes Relat Metab Disord* 2000; **24**: 777–781.
15 Singal A, Farooqi IS, O'Rahilly S, Cole TJ, Fewtrell M, Lucas A. Early nutrition and leptin concentrations in later life. *Am J Clin Nutr* 2002; **75**: 993–999.
16 Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. *Pediatrics* 1998; **101**: 539–549.
17 Fisher JO, Birch LL. Restricting access to foods and children's eating. *Appetite* 1999; **32**: 405–419.
18 Faith MS, Heshka S, Keller KK, Sherry B, Matz PE, Pietrobelli A, Allison DB. Maternal-child feeding patterns and child body weight. *Arch Pediatr Adolesc Med* 2003; **157**: 926–932.
19 Faith MS. Development and modification of child food preferences and eating patterns: behavior genetics strategies. *Int J Obes Relat Metab Disord* 2005; **29**: 549–556.

20 Pietrobelli A. Putting related individual studies into childhood obesity intervention trials. *Int J Obes Relat Metab Disord* 2005; **29**: 547–548.
21 Kant A, Schatzkin A, Block G, Ziegler R, Nestle M. Food group intake patterns and associated nutrient profiles of the US population. *J Am Diet Assoc* 1991; **91**: 1532–1537.
22 *National research council. Recommended dietary allowances* 10th Edn. National Academy: Washington, DC; 1989.
23 Società Italiana di Nutrizione Umana (SINU). Livelli di assunzione raccomandati di Energia e Nutrienti per la Popolazione Italiana (LARN), 1996.
24 McCrory MA, Fuss PJ, McCallum JE, Yao M, Vinken AG, Hays N, Roberst SB. Dietary variety within food groups: association wit energy intake and body fatness in men and women. *Am J Clin Nutr* 1999; **69**: 440–447.
25 St-Onge MP, Keller KK, Heymsfield SB. Changes in childhood food consumption pattern: a cause for concern in light of increasing body weights. *Am J Clin Nutr* 2003; **78**: 1068–1073.
26 Popkin BM, Gordon-Larsen P. The nutricion transitino: worldwide obesity dynamics and their determinants. *Int J Obes Relat Metab Disord* 2004; **28**: S2–S9.
27 Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences!. *Public Health Nutr* 2002; **5**: 205–214.
28 Nielsen S, Siega-Riz A, Popkin BM. Trends in energy intake in the US between 1977 and 1996: similar shifts seen across age groups. *Obes Res* 2002; **10**: 370–378.
29 French S, Story M, Neumark-Sztainer D, Fulkerson JA, Hannan P. Fast food restaurant use among adolescents: associations with nutrient intake, food choice, and behavioural and psychosocial variables. *Int J Obes Relat Metab Disord* 2001; **25**: 1823–1833.
30 Nestle M. The ironic politics of obesity. *Science* 2003; **299**: 781.
31 Nestle M. Increasing portion sizes in American diets: more calories, more obesity. *J Am Diet Assoc* 2003; **103**: 39–40.
32 Flodmark CE, Lissau I, Pietrobelli A. Child and adolescent obesity: why we need to fight!. *Acta Paediatrica* 2005; **94**: 4–7.
33 St-Onge MP. Dietary fats, teas, dairy and nuts: potential functional foods for weight control? *Am J Clin Nutr* 2005; **81**: 7–15.
34 Shoeller DA. Hydrometry In: Roche AF, Heymsfield SB, Lohman TG (eds). *Human body composition*. Human Kinetics: Champagne, IL; 1996. pp 25–43.
35 Wang ZM, Pierson Jr RN, Heymsfield SB. The five level model: a new approach to organizing body-composition research. *Am J Clin Nutr* 1992; **56**: 19–28.
36 Snyder WS, Cook MJ, Nasset ES, Karhausen LR, Howells GP, Tipton IH. *Report of the task group on reference man*. Pergamon Press: Oxford, UK; 1975.
37 Grunwald GK, Seagle HM, Peters JC, Hill JO. Quantifying and separating the effects of macronutrient composition and non-macronutrients on energy density. *Br J Nutr* 2001; **86**: 265–276.
38 Weisburger JH. Tea and health: the underlying mechanism. *Proc Soc Exp Biol Med* 1999; **220**: 271–275.
39 Rumpler W, Seale J, Clevidence B, Judd J, Wiley E, Yamamoto S, Komatsu T, Sawaki T, Ishikura Y, Hosoda K. Oolong tea increases metabolic rate and fat oxidation in men. *J Nutr* 2001; **131**: 2848–2852.
40 Komatsu T, Nakamori M, Komatsu K, Hosoda K, Okamura M, Toyama K, Ishikura Y, Sakai T, Kunii D, Yamamoto S. Oolong tea increases energy metabolism in Japanese females. *J Med Invest* 2003; **50**: 170–175.
41 Zemel MB, Shi H, Greer B, Dirienzo D, Zemel PC. Regulation of adiposity by dietary calcium. *FASEB J* 2000; **14**: 1132–1138.
42 Zemel MB. Mechanism of dairy modulation of adiposity. *J Nutr* 2003; **133**: 252:2–252:6.
43 Melanson EL, Sharp TA, Schneider J, Donahoo WT, Grunwald GK, Hill JO. Relation between calcium intake and fat oxidation in adults humans. *Int J Obes Relat Metab Disord* 2003; **27**: 196–203.
44 Skinner JD, Bounds W, Carruth BR, Ziegler P. Longitudinal calcium intake is negatively related to children's body fat indexes. *J Am Diet Assoc* 2003; **103**: 1626–1631.
45 Carroth BR, Skinner JD. The role of dietary calcium and other nutrients in moderating body fat in pre-school children. *Int J Obes Relat Metab Disord* 2001; **25**: 559–566.
46 Zemel MB, Richards J, Mathis S, Milstead A, Gebhardt L, Silva E. Dairy augmentation of total and central fat lkoss in obese subjects. *Int J Obes Relat Metab Disord* 2005; **29**: 391–397.
47 Lappe JM, Rafferty KA, Davies KM, Lypaczewski G. Girls on a high-calcium diet gain weight at the same rate as girls on a normal diet: a pilot study. *J Am Diet Assoc* 2004; **104**: 1361–1367.
48 World Health Organization. *Report of a Joint WHO/FAO Expert Consultation. Diet nutrition and the prevention of chronic diseases* WHO Technical Report Series no. 916 WHO: Geneva; 2002.
49 American Academy of Pediatrics. Policy statement. prevention of pediatric overweight and obesity. *Pediatrics* 2003; **112**: 424–430.
50 Flodmark CE, Lissau I, Moreno LA, Pietrobelli A, Widhalm K. New insight into the field of children and adolescents' obesity: the European perspective. *Int J Obes Relat Metab Disord* 2004; **28**: 1189–1196.
51 Forbes GB. Body composition in infancy, childhood and adolescence. In: Forbes GB (ed). *Human body composition: growth, aging and activity*. Springer Verlag: New York, NY USA; 1987. pp 125–196.
52 Lissau I, Burniat W, Poskitt E, Cole TJ. Prevention. In: Burniat W, Cole TJ, Lissau I, Poskitt E (eds). *Child and adolescent obesity*. Cambridge University Press: Cambridge; 2002. pp 243–269.