Resting metabolic rate in obese and nonobese Chinese Singaporean boys aged 13–15 y[1–3]

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ABSTRACT

Background: Previous studies investigating the hypothesis that a low resting metabolic rate (RMR) is a cause of obesity yielded discrepant findings. Two explanations for these findings are the use of imprecise methods to determine obesity and a failure to control for differences in fat mass (FM) and fat-free mass (FFM) when comparing RMR values.

Objective: This study tested the hypothesis that RMR is lower in obese than in nonobese boys (with the use of precise methods to quantify body fatness and with adjustment for differences in both FM and FFM).

Design: Forty Chinese Singaporean boys aged 12.8–15.1 y were recruited. Boys were classified as obese (n = 20) or nonobese (n = 20) on the basis of their adiposity index (ratio of FM to FFM: >0.60 = obese, <0.40 = nonobese) determined by dual-energy X-ray absorptiometry. RMR was determined by using indirect calorimetry. RMR values were compared by using both linear (analysis of covariance) and log-linear (analysis of covariance with log-transformed data) regression to control for differences in FM and FFM.

Results: Age, height, and FFM did not differ significantly between groups. Body mass was 13 kg greater and FM was 16 kg greater in the obese boys than in the nonobese boys ($P < 0.001$). After control for FFM and FM, RMR did not differ significantly between the groups.

Conclusion: When body composition is appropriately controlled for, RMR does not differ significantly between obese and nonobese boys. *Am J Clin Nutr* 2001;74:369–73.

KEY WORDS Resting metabolic rate, obesity, Chinese Singaporean boys, fat mass, fat-free mass, analysis of covariance, log-linear regression, dual-energy X-ray absorptiometry, indirect calorimetry

INTRODUCTION

Cross-sectional studies examining resting metabolic rate (RMR) in obese and nonobese children have yielded discrepant findings. Usually, RMR is found to be higher in obese than in nonobese children when absolute values (kJ/d) are compared (1–7), but there are exceptions (8, 9). After control for fat-free mass (FFM), most studies reported that RMR did not differ significantly between obese and nonobese children (3, 4, 6, 7, 9) although some studies still report higher RMR values in the obese children (1, 5).

Explanations for the disparate findings presented above include *1*) the methods used to measure body composition, *2*) the criteria used to determine obesity, and *3*) the means of controlling for differences in body size or composition when RMR values are compared. Most studies measured skinfold thickness to assess body composition (3–6, 9), but weight and height (2), total body water (1), and dual-energy X-ray absorptiometry (DXA) (7, 8) have also been used. Criteria used to classify children as obese included weight-for-height (2, 3, 5, 7, 9), body mass index (4, 6), skinfold thickness (8), and percentage of ideal body weight determined from measurements of total body water (1). The predominant method used to adjust for differences in body size or composition in comparisons of RMR values was analysis of covariance with FFM as the covariate (1, 3, 4, 6, 7, 9). This practice was promoted by Poehlman and Toth (10) in a paper published in this journal. It is now clear, however, that fat mass (FM) also contributes significantly to RMR (11, 12) and that differences in FM should be controlled for in comparisons of RMR between groups. Only one of the studies cited above (5) controlled for both FM and FFM when comparing RMR in obese and nonobese children. This study found no difference in RMR between groups, but the imprecise method used to determine body composition (skinfold thickness) and the criterion for obesity (weight-for-height) mean that the findings require verification.

Therefore, we decided to reexplore the relation between RMR and obesity in 40 Chinese Singaporean boys by using DXA to determine body composition and both linear and log-linear regression to control for FM and FFM. We hypothesized that with accurate determination of body composition and appropriate

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Received June 22, 2000.

Accepted for publication January 3, 2001.

TABLE 1
Age, height, weight, and adiposity of obese and nonobese boys[1]

	Obese (n = 20)	Nonobese (n = 20)
Age (y)	13.6 ± 0.5	13.7 ± 0.6
Height (m)	1.64 ± 0.09	1.64 ± 0.06
Weight (kg)	74.8 ± 12.3	61.8 ± 10.9[2]
Adiposity index[3]	0.75 ± 0.12	0.32 ± 0.07[2]

[1] $\bar{x}$ ± SD.
[2] Significantly different from obese, $P < 0.001$.
[3] Ratio of fat mass to fat-free mass.

adjustment for both FM and FFM, RMR would be significantly lower in obese than in nonobese Chinese Singaporean boys.

SUBJECTS AND METHODS

Subjects

The subjects in this study were 40 Chinese Singaporean boys aged 12.8–15.1 y (**Table 1**). The boys were year 1 and year 2 students at Catholic High School, Singapore. Written informed consent was obtained from the boys and from their parents before the start of the study. The Ethical Advisory Committee of the School of Physical Education (Nanyang Technological University) also gave its permission for the study. Boys were classified as obese (n = 20) or nonobese (n = 20) on the basis of their adiposity index (ratio of FM to FFM). Boys with an adiposity index >0.60 were classified as obese whereas those with an adiposity index <0.40 were classified as nonobese. These values were chosen arbitrarily to create 2 distinctly different groups with respect to body fatness. Boys with an adiposity index of 0.40–0.60 were excluded from the study. The pubertal status of the boys was not determined, but the groups did not differ significantly with respect to age, height, or FFM.

Body composition

Height was measured to the nearest 0.1 cm by using a wall-mounted stadiometer (Holtain, Dyfed, United Kingdom). The mass of the subjects, who were wearing socks and shorts, was measured to the nearest 0.01 kg with an electronic weighing scale (IDL Plus; Mettler Toledo, Giessen, Germany). Body mass index was calculated as weight (kg)/height^2 (m). The waist-to-hip ratio was determined from the waist (minimal circumference of the abdomen) and hip (maximal circumference over the greater trochanters) circumferences measured to the nearest 1.0 cm by using a plastic measuring tape. A DXA instrument (LUNAR DPX-L model 1.31; Lunar Corp, Madison, WI) was used to measure each subject's FM, FFM, and percentage body fat. The DXA scans were performed in the Orthopaedic Diagnostic Centre at the National University Hospital, Singapore.

Resting metabolic rate

RMR was determined by using indirect calorimetry. The boys were asked to avoid strenuous exercise for 1 d before the test and to fast for 12 h the night before the test. On the morning of the test, the boys were collected from school at 0700 and transported by car to the laboratory of the School of Physical Education. On arrival at the laboratory, the boys sat quietly for 30 min before RMR was measured by using a metabolic cart (model 2900Z; SensorMedics, Yorba Linda, CA). This metabolic cart was calibrated each morning before the tests began. A half-face mask with a 2-way breathing valve (model 2700; Hans Rudolph Inc, Kansas City, MO) was fitted to the subjects' mouths and connected by respiratory tubes to the metabolic cart. Testing was conducted with the boys in a seated position. Expired air was collected for ≥30 min. RMR (kJ/d) was calculated from oxygen consumption and carbon dioxide production (13) without measurement of protein oxidation. The values used to calculate RMR included initial steady state values and all values after this. Steady state was defined as ventilation oxygen consumption and the respiratory exchange ratio each varying by <3% during 2 consecutive minutes. Steady state was reached generally between 5 and 15 min of the collection period. If a steady state was attained after 15 min, the duration of the collection period was extended. If a steady state was not reached at any point during the collection period, then the first 5 min of data were discarded; RMR was calculated from the remaining data. The mean duration of data collection used to calculate RMR for both groups was 20 min.

Statistical analysis

SPSS (version 9.0; SPSS, Inc, Chicago) was used to analyze the data. RMR values were divided by body mass and FFM so that values could be expressed as $kJ \cdot kg\ bodywt^{-1} \cdot d^{-1}$ and $kJ \cdot kg\ FFM^{-1} \cdot d^{-1}$, respectively. These values were then compared with the use of two-tailed t tests for independent samples. In addition, analysis of covariance was used to compare RMR in the obese and nonobese boys with the use of only FFM as a covariate and then with both FFM and FM as covariates. Finally, log-linear regression was used to compare the RMR values of the groups. For this comparison, analysis of covariance was again used to control for FFM alone and for FFM in combination with FM but using log-transformed data (eg, ln RMR, ln FFM, and ln FM). This approach was based on the assumption that RMR was likely to be proportional to the subject's body size (similar to the relation between peak oxygen consumption and body mass), and hence, best expressed by the allometric model $Y = am^b$, described previously (14, 15). Pearson product-moment correlation coefficients were used to examine relations between variables. Significance was set at $P < 0.05$. Data are given as means ± SDs.

RESULTS

Body-composition variables for the obese and nonobese boys are shown in **Table 2**. All variables were significantly different between groups except for FFM. The minimum and maximum values for each group for each body-composition variable shown in Table 2 indicated that there was some overlap between groups for each variable with the exception of percentage body fat, for which the maximum value in the nonobese group (28.3%) was 11% less than the minimum value in the obese group (39.0%). Conversely, minimum and maximum values for FFM indicated considerable overlap between groups.

The relations between RMR and body mass, RMR and FFM, and RMR and FM are shown in **Figures 1–3**. All relations were significant. The highest correlations were between RMR and body mass. Correlations between RMR and FFM were only slightly higher than those between RMR and FM in each group.

RMRs are expressed in 7 different ways in **Table 3**. Absolute values did not differ significantly between groups. When absolute

TABLE 2
Body-composition variables for obese and nonobese boys[1]

	Obese (n = 20)	Nonobese (n = 20)
Percentage body fat (%)	42.5 ± 3.5 (39.0–51.3)	24.1 ± 4.3 (12.4–28.3)[2]
BMI (kg/m²)	27.7 ± 2.9 (23.1–35.4)	22.8 ± 3.0 (19.3–30.6)[2]
Waist circumference (cm)	90.8 ± 8.1 (76.2–110.2)	76.1 ± 6.0 (66.7–89.8)[2]
Waist-to-hip ratio	0.90 ± 0.04 (0.80–0.96)	0.84 ± 0.04 (0.77–0.91)[2]
Fat mass (kg)	30.1 ± 6.2 (19.4–41.7)	14.1 ± 4.1 (7.5–23.0)[2]
Fat-free mass (kg)	40.5 ± 6.9 (26.7–56.0)	44.1 ± 7.6 (31.7–58.4)

[1] $\bar{x}$ ± SD; range in parentheses.
[2] Significantly different from obese, $P < 0.001$.

values were divided by body mass (kJ·kg bodywt^{-1}·d^{-1}), they were significantly lower in the obese than in the nonobese boys; significantly higher values were obtained in the obese boys when absolute values were divided by FFM (kJ·kg FFM^{-1}·d^{-1}). The use of linear or log-linear regression yielded significantly higher RMR values in the obese boys when FFM was controlled for. The fitted exponent for FFM based on the log-linear model was $\beta = 0.50$ (95% CI: 0.34, 0.67). Conversely, when both FFM and FM were controlled for by using either linear or log-linear regression, RMR values did not differ significantly between groups. The fitted exponents for FFM and FM based on the log-linear model were $\beta = 0.39$ (95% CI: 0.20, 0.56) and $\beta = 0.15$ (95% CI: 0.03, 0.28), respectively. Interestingly, the covariates of FFM and FM contributed significantly to the prediction of RMR in the above regression analyses.

DISCUSSION

The main finding of this study was that after FM and FFM were controlled for, RMR did not differ significantly between the obese and nonobese boys. This is consistent with previous results of cross-sectional studies that did not show a lower RMR in obese than in nonobese children (1–9). It is also consistent with the finding that infants (16) and children (17–21) who are genetically predisposed to obesity do not have lower RMR values than infants and children who are not predisposed to obesity.

Although the results of the present study are consistent with data from previous cross-sectional studies in showing that obese children do not have lower RMRs, the findings of the present study differ in several ways. Most previous comparisons observed that absolute RMR values were higher in obese than in nonobese children (1–7). In the present study, absolute RMR values did not differ significantly between groups. A likely explanation for this is that FFM did not differ significantly between obese and nonobese boys. In fact, the mean FFM value of the obese group was slightly lower than that of the nonobese group (40.5 compared with 44.1 kg; NS). All previous studies found that FFM was higher in obese than in nonobese children. In some studies these differences were significant (1, 4–7) and in others they were not (3, 8, 9).

It is possible that the lower FFM values in the obese boys were a peculiarity of the selection criteria that were used (ie, adiposity index >0.60). This may have encouraged recruitment of boys with a particularly low FFM to the obese group. Another possibility is that DXA did not accurately measure FFM differences between the obese and nonobese boys. There is evidence that tissue thickness affects the accuracy of DXA measurements (22); therefore, it is possible that FFM was underestimated in the obese boys or overestimated in the nonobese boys. However, Svendsen et al (23) found excellent agreement between DXA (Lunar DPX-L, version 1.31) measures of FFM and values determined by chemical analysis in 7 pigs weighing 35–95 kg and ranging in body fat from 10% to 50%. A further possibility is that the hydration status of the obese and nonobese boys differed before DXA analysis. Hydration influences DXA measurements of FFM because DXA

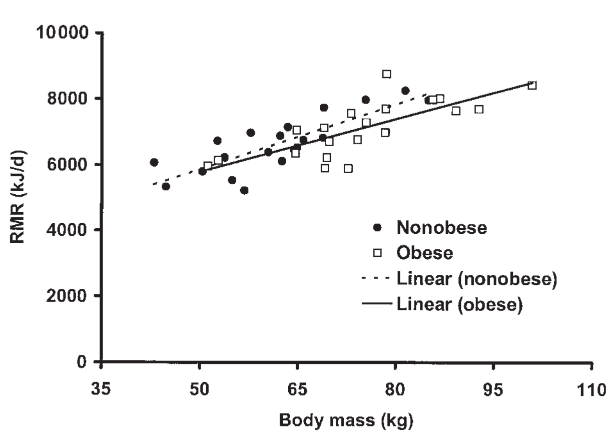

FIGURE 1. Relation between body mass and resting metabolic rate (RMR) in 20 obese boys ($r = 0.76$, $P < 0.0001$) and 20 nonobese boys ($r = 0.83$, $P < 0.0001$).

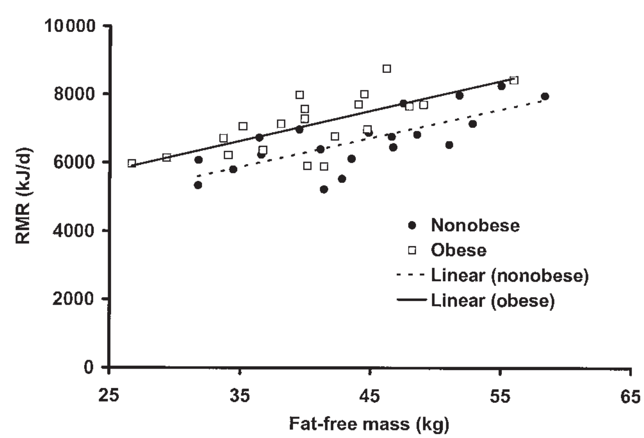

FIGURE 2. Relation between fat-free mass and resting metabolic rate (RMR) in 20 obese boys ($r = 0.71$, $P < 0.001$) and 20 nonobese boys ($r = 0.74$, $P < 0.0001$).

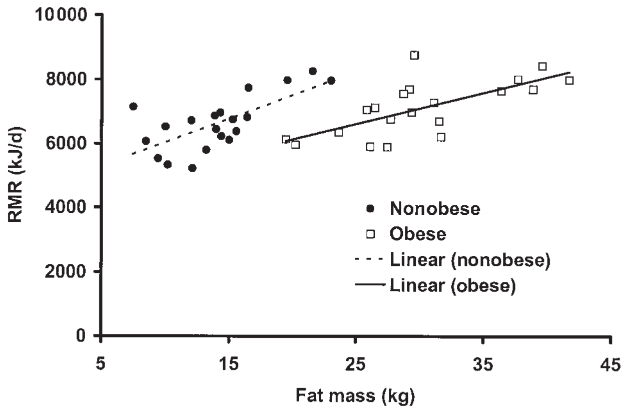

FIGURE 3. Relation between fat mass and resting metabolic rate (RMR) in 20 obese boys (r = 0.69, P < 0.001) and 20 nonobese boys (r = 0.70, P < 0.001).

assumes that water is a constant fraction of bone-free lean tissue mass (0.73 mL/g). Hence, dehydration results in a reduction in FFM measured by DXA (24). However, we have no reason to suspect that the obese boys were dehydrated at the time of measurement. Furthermore, although the water content of lean tissue changes during childhood, any difference between groups in the present study should have had a small effect on the assessment of FFM by DXA (22).

When analysis of covariance was used to control for FFM, some studies reported that RMR values were still higher in obese children (1, 5) whereas others did not (3, 4, 6, 7, 9). In the present study, RMR values were higher in the obese boys when we controlled for FFM using either linear or log-linear regression. This is consistent with the results of the study of Bandini et al (1) and that of Molñar and Schutz (5) and is most likely due to the difference in FM between the obese and nonobese groups. This difference was 16 kg on average in the present study. In the study of Bandini et al, FM values were not reported but were based on body mass and percentage body fat data; the difference between the obese and nonobese groups was 29 kg. In the study of Molnar and Schutz (5), the difference was 18 kg. Although FM makes only a small contribution to RMR, it is an important contribution (12). Thus, a large FM would be expected to elevate RMR. This is supported by the fact that both FFM and FM predicted RMR when analysis of covariance was performed in the present study. In studies that did not show elevated RMR values in obese children after controlling for FFM (3, 4, 6, 7, 9), differences in FM were much smaller—ranging from 6.0 to 10.4 kg and, hence, would have had a smaller effect on RMR.

TABLE 3
Resting metabolic rates (RMR) in obese and nonobese boys[1]

RMR	Obese (n = 20)	Nonobese (n = 20)
(kJ/d)	7117 ± 863	6648 ± 870
(kJ · kg body wt · d^{-1})	96.3 ± 10.9	108.8 ± 12.2[2]
(kJ · kg FFM^{-1} · d^{-1})	178.5 ± 22.5	152.7 ± 19.2[3]
Adjusted for		
FFM by ANCOVA (kJ/d)	7276 ± 615	6489 ± 615[3]
FFM and FM by ANCOVA (kJ/d)	6723 ± 1031	7042 ± 1031
FFM by log-linear ANCOVA (kJ/d)[4]	7222 ± 664	6451 ± 600[3]
FFM and FM by log-linear ANCOVA (kJ/d)[4]	6707 ± 644	6946 ± 649

[1] $\bar{x}$ ± SD; FM, fat mass; FFM, fat-free mass; ANCOVA, analysis of covariance.
[2,3] Significantly different from obese: [2] P < 0.001, [3] P < 0.0001.
[4] Antilogs.

The preceding discussion highlights the importance of 2 factors when conducting studies of this nature: *1*), the criteria used to determine obesity, and *2*) more important, the method of expressing RMR. To our knowledge, only one previous study controlled for both FM and FFM when comparing the RMR of obese and nonobese boys (5). Our findings confirm that with control for FM and FFM there is no difference in RMR between groups. Thus, obese Chinese Singaporean boys who did not differ in age or height from their nonobese counterparts but carried 16 kg of excess fat appeared to have normal RMRs. This finding is perhaps not surprising in light of obesity-prevalence data. The prevalence of obesity (defined by weight-for-height with regard to age and sex) in Singaporean schoolchildren increased from 5.4% in 1980 to 15.1% in 1991 (25). It is unlikely that such a sharp increase in a short amount of time could be explained by a sudden alteration in RMR.

It is possible that RMR was low in the obese boys in the present study before they became obese. Such a hypothesis is supported by the results of Griffiths and Payne (26). Moreover, the results of a study by Roberts et al (27) suggest that low total energy expenditure in infants is predictive of future weight gain. However, more recent cross-sectional (16–21) and prospective (28, 29) studies do not support the hypothesis that a low RMR is predictive of obesity, and there are limitations to the earlier work. Griffiths and Payne's (26) study of 2 groups of children matched for body size found lower RMRs per kilogram body mass in the group of 3–5-y-old children of obese parents than in the group of children of nonobese parents. However, a follow-up study conducted 12 y later showed no difference in percentage body fat between subgroups of the original children (30). The study of Roberts et al (27) examined infants predisposed to obesity by virtue of having an overweight mother. It was observed that infants who became overweight by 1 y of age had a low total energy expenditure (measured by using doubly labeled water) at 3 mo of age. However, RMR was not measured in this study and the postprandial metabolic rate did not differ significantly between groups. Thus, the evidence that a low RMR in infancy or early childhood is a cause of obesity is limited.

In conclusion, the present study addressed the limitations of previous cross-sectional studies examining RMR in obese and nonobese children by using more appropriate methods and criteria to determine body composition and obesity and by controlling for FM as well as for FFM when comparing RMR between groups. Our findings indicate that RMR is neither lower nor higher in obese boys than in nonobese boys and therefore suggest that the RMR of obese children is not abnormal.

We thank the students from Catholic High School who participated in the study; the staff at Catholic High School, particularly Parameswari Thambusamy and Benjamin Kwok for allowing us into their school and assisting in the administration of this study; and Joyce Tan Bee Lian and Gillian Ng Bee Kit for their help with the RMR measurements.

REFERENCES

1. Bandini LG, Schoeller DA, Dietz WH. Energy expenditure in obese and non-obese adolescents. Pediatr Res 1990;27:198–203.
2. Epstein LH, Wing RR, Cluss P, et al. Resting metabolic rate in lean and obese children: relationship to child and parent weight and percent overweight change. Am J Clin Nutr 1989;49:331–6.
3. Maffeis C, Schutz Y, Zoccante L, Micciolo R, Pinelli L. Meal-induced thermogenesis in lean and obese prepubertal children. Am J Clin Nutr 1993;57:481–5.
4. Maffeis C, Pinelli L, Schutz Y. Increased fat oxidation in prepubertal obese children: a metabolic defence against further weight gain? J Pediatr 1995;126:15–20.
5. Molnár D, Schutz Y. The effect of obesity, age, puberty and gender on resting metabolic rate in children and adolescents. Eur J Pediatr 1997;156:376–81.
6. Schutz Y, Rueda-Maza CM, Zaffanello M, Maffeis C. Whole-body protein turnover and resting energy expenditure in obese, prepubertal children. Am J Clin Nutr 1999;69:857–62.
7. Treuth MS, Figueroa-Colon R, Hunter GR, Weinsier RL, Butte NF, Goran MI. Energy expenditure and physical fitness in overweight vs non-overweight prepubertal girls. Int J Obes Relat Metab Disord 1998;22:440–7.
8. DeLany JP, Harsha DW, Kime JC, Kumler J, Melancon L, Bray GA. Energy expenditure in lean and obese prepubertal children. Obes Res 1995;3(suppl):67–72.
9. Maffeis C, Micciolo R, Zoccante L, Zaffanello M, Pinelli L. Basal energy expenditure in obese and normal weight schoolchildren. Acta Paediatr Scand 1991;80:1145–9.
10. Poehlman ET, Toth MJ. Mathematical ratios lead to spurious conclusions regarding age- and sex-related differences in resting metabolic rate. Am J Clin Nutr 1995;61:482–5.
11. Goran MI, Kaskoun M, Johnson R. Determinants of resting energy expenditure in young children. J Pediatr 1994;125:362–7.
12. Tataranni PA, Ravussin E. Variability in metabolic rate: biological sites of regulation. Int J Obes Relat Metab Disord 1995;19(suppl): S102–6.
13. Consolazio CF, Johnson RE, Pecora LJ. Physiological measurements of metabolic functions in man. New York: McGraw Hill, 1963.
14. Nevill AM. The appropriate use of scaling techniques in exercise physiology. Pediatr Exerc Sci 1997;9:295–8.
15. Welsman JR, Armstrong N, Nevill AM, Winter EM, Kirby BJ. Scaling peak VO_2 for differences in body size. Med Sci Sports Exerc 1996;28:259–65.
16. Davies PS, Wells JC, Fieldhouse CA, Day JM, Lucas A. Parental body composition and infant energy expenditure. Am J Clin Nutr 1995;61:1026–9.
17. Fontvieille AM, Dwyer J, Ravussin E. Resting metabolic rate and body composition of Pima Indian and Caucasian children. Int J Obes Relat Metab Disord 1992;16:535–42.
18. Goran MI, Carpenter WH, McGloin A, Johnson R, Hardin JM, Weinsier RL. Energy expenditure in children of lean and obese parents. Am J Physiol 1995;268:E917–24.
19. Goran MI, Kaskoun M, Johnson R, Martinez C, Kelly B, Hood V. Energy expenditure and body fat distribution in Mohawk children. Pediatrics 1995;95:89–95.
20. Treuth MS, Butte NF, Wong WW. Effects of familial predisposition to obesity on energy expenditure in multiethnic prepubertal girls. Am J Clin Nutr 2000;71:893–900.
21. Wurmser H, Laessle R, Jacob K, et al. Resting metabolic rate in preadolescent girls at high risk of obesity. Int J Obes Relat Metab Disord 1998;22:793–9.
22. Kohrt WM. Body composition by DXA: tried and true? Med Sci Sports Exerc 1995;27:1349–53.
23. Svendsen OL, Haarbo J, Hassager C, Christiansen C. Accuracy of measurements of body composition by dual-energy X-ray absorptiometry in vivo. Am J Clin Nutr 1993;57:605–8.
24. Horber FF, Thomi F, Casez JP, Fonteille J, Jaeger P. Impact of hydration status on body composition as measured by dual energy X-ray absorptiometry in normal volunteers and patients on haemodialysis. Br J Radiol 1992;65:895–900.
25. Ministry of Health. The Health of Singaporeans. Singapore: Research and Evaluation Department, Ministry of Health, 1993.
26. Griffiths M, Payne PR. Energy expenditure in small children of obese and non-obese parents. Nature 1976;260:698–700.
27. Roberts SB, Savage J, Coward WA, Chew B, Lucas A. Energy expenditure and intake in infants born to lean and overweight mothers. N Engl J Med 1988;318:461–6.
28. Goran MI, Shewchuk R, Gower BA, Nagy TR, Carpenter WH, Johnson RK. Longitudinal changes in fatness in white children: no effect of childhood energy expenditure. Am J Clin Nutr 1998;67:309–16.
29. Stunkard AJ, Berkowitz RI, Stallings VA, Schoeller DA. Energy intake, not energy output, is a determinant of body size in infants. Am J Clin Nutr 1999;69:524–30.
30. Griffiths M, Payne PR, Stunkard AJ, Rivers JPW, Cox M. Metabolic rate and physical development in children at risk of obesity. Lancet 1990;336:76–8.