Original Article

# Validation of prediction equations for estimating resting energy expenditure in obese Chinese children

Dorothy FY Chan FHKAM[1], Albert M Li FHKAM[1], Michael HM Chan FRCPA[2], Hung Kwan So PhD[1], Iris HS Chan BSc[2], Jane AT Yin Bsc[1], Christopher WK Lam PhD[2], Tai Fai Fok FRCPCH[1], Edmund AS Nelson FRCPCH[1]

[1]*Department of Paediatrics, The Chinese University of Hong Kong, Shatin, Hong Kong SAR*
[2]*Department of Chemical Pathology, The Chinese University of Hong Kong, Shatin, Hong Kong SAR*

Objectives: (1) To examine the validity of existing prediction equations (PREE) for estimating resting energy expenditure (REE) in obese Chinese children, (2) to correlate the measured REE (MREE) with anthropometric and biochemical parameters and (3) to derive a new PREE for local use. Design: Cross-sectional study. Subjects: 100 obese children (71 boys) were studied. Measurements: All subjects underwent physical examination and anthropometric measurement. Upper and central body fat distribution was signified by centrality and conicity index respectively, and REE was measured by indirect calorimetry. Fat free mass (FFM) were measured by DEXA scan. Thirteen existing prediction equations for estimating REE were compared with MREE among these obese children. Fasting blood for glucose, lipid profile and insulin were obtained. Results: The overall, male and female median MREEs were 7.1 mJ/d (IR 6.2-8.4), 7.3 mJ/d (IR 6.3-9.7) and 6.9 mJ/d (IR 5.6-8.1) respectively. No sex difference was noted in MREE ($p=0.203$). Most of the equations except Schofield equation underestimated REE of our children. By multiple linear regression, MREE was positively correlated with FFM ($p<0.0001$), conicity index ($p<0.001$) and centrality index ($p=0.001$). A new equation for estimating REE for local use was derived as: REE = (17.4*logFFM) + (11.4*conicity index) – (2.4*centrality index) – 31.3. The mean difference of new PREE-MREE was -0.011 mJ/d (SD 1.51) with an interclass correlation coefficient of 0.91. Conclusion: None of the existing prediction equations were accurate in their estimation of REE, when applied to obese Chinese children. A new prediction equation has been derived for local use.

**Key Words: obese, children, body fat distribution, resting energy expenditure, insulin resistance**

## INTRODUCTION

The problem of obesity, and its related complications, has become a major health concern globally.{WHO, 2000 4 /id} In Hong Kong, it was estimated that the prevalence of obesity for children aged 11 years rose from 21% and 10% in 1993 to 34% and 13% in 1998[2] (defined as >120% median weight–for–height using local reference ranges)[3] for boys and girls, respectively.

Prevention and treatment of this global problem is proving to be a challenge. Monitoring total energy expenditure (TEE) is important in understanding the potential for the occurrence of under- or over-nutrition in populations. Resting energy expenditure contributes 60-70% of TEE,[4] and this is often used to estimate the TEE. The gold standard for measuring resting energy expenditure (REE) is indirect calorimetry which is not widely available as it is a time consuming and labor intensive procedure. Consequently, several prediction equations using simple anthropometric parameters have been derived to estimate REE.[5-10]

Most existing equations were derived from Caucasian population and appear to overestimate REE in Asians.[11,12] As stated in Table 1, most of these prediction equations are contributed positively by body weight and body height and negatively by age. The taller statue of Caucasian population may contribute to the overestimation. Leung and colleagues derived a new validated prediction equation for Hong Kong Chinese adults.[13] However, equations derived from adult populations are not applicable for children[7,8] and furthermore specific REE equations for children do not allow cross-racial comparisons.[7,9,14,15] REE has been shown to be significantly less in blacks than in Whites in the United States.[16] REE has also been shown to be higher in obese than non-obese Caucasian children.[14] At the time of manuscript compilation, there is a lack of race and age specific equation for local use.

REE was reported to be associated with metabolic risk factors including high blood pressure[17] and insulin resistance[18,19] Moreover, adults with metabolic syndrome had 10% higher of REE than normal healthy adults.[20] Conflicting negative correlation of REE and insulin resistance

**Corresponding Author:** Dr Dorothy FY Chan, Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong SAR.
Tel: (852) 26322859; Fax: (852) 2636 0020
Email: dorothychan@cuhk.edu.hk
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was demonstrated in obese children and young adults by Ten et al.[21] The association was unclear in the Chinese population.

This study aimed to (1) validate 13 existing prediction equations for estimating REE of local obese Chinese children, (2) correlate measured REE with anthropometric and biochemical parameters including insulin resistance, and (3) derive a new prediction equation for local obese children.

## MATERIALS AND METHODS

Children with primary obesity, aged 7 to 18 years, were recruited consecutively during their first visit at a specialist for medical assessment between 1 January 2000 and 31 December 2004. Referrals were from primary health care physicians and children with secondary cause of obesity were excluded. All recruited children had a body mass index (BMI) above 95$^{th}$ percentile according to local sex- and age-specific reference ranges.[22] The study was approved by the Clinical Research Ethics Committee of the Chinese University of Hong Kong and informed consents were obtained from the subjects and their parents prior to assessment.

Each child underwent a complete physical examination including anthropometric measures. Weight and standing height were measured with a calibrated weighing scale and stadiometer respectively using standard methods.[23] The percentage of body fat was determined by the multiple skinfold technique performed by a single observer (DC) using a Holtain Skinfold Caliper (Holtain Ltd, Crosswell, United Kingdom).[24] Skinfold thickness was measured at the biceps, triceps, subscapular and suprailiac regions. Each skinfold was measured three times and reported as the average of the three measurements. Waist and hip girth measurements were obtained using an inelastic cloth measuring tape around the area of greatest girth of the abdomen and hip.[25] Assessment of obesity included the four skinfold measurements and BMI (kg/m$^2$). Measurements of body central fat distribution included the waist to hip ratio (WHR) and conicity index (ConI) which is a function of waist circumference, weight, and height.[26] The formular for ConI is waist circumference (m)/ 0.109 sq root [weight (kg)/height (m)]. The upper body fat distribution was demonstrated by centrality index (CenI) which is calculated from the ratio of the subscapular to triceps skinfolds. The 50$^{th}$ centile of the BMI curve according to age and sex reference stated by Cole was defined as ideal BMI.[27] The degree of obesity was presented by percentage above the ideal BMI. Overweight and obesity were defined as age and sex specific BMI corresponding to cuff off point of 25 kg/m$^2$ and 30 kg/m$^2$ at the age of 18.

Subjects were kept fasting for at least 4 hours before measurement of REE were conducted. Measured REE (MREE) was assessed by using open-circuit indirect calorimetry performed with the use of a respiratory metabolic cart (Deltatrac II MBM-200; Instrumentarium Corp, Helsinki, Finland). Before each test, the calorimeter was calibrated with the use of a reference gas mixture provided by the manufacturer. Measurements for MREE were performed during sleep in a quiet and thermo-regulated room. During measurement, the subject was covered with an airtight transparent plastic canopy. The metabolic monitor generated a constant airflow of 40 l/min through the canopy. All exhaled gas was collected into this constant flow. VO2 and VCO2 were calculated every minute from the difference in the oxygen and carbon dioxide concentration of inhaled and exhaled gas respectively, after adjustment to standard temperature (0°C) and pressure (760 mmHg or 101.3 kPa). Each measurement was continued

**Table 1.** Thirteen existing prediction equations (MJ/d) and difference with REE

| Source | Age group | Equation | Difference | ICC: Alpha |
|---|---|---|---|---|
| Harris-Benedict | Adults | | -0.82(1.79) | 0.7827 |
| Boys | | 4.18*[(66.437+(13.752*wt)+(5.003*ht)-(6.755*age)]/1000 | | |
| Girls | | 4.18*[(655.096+(9.563*wt)+(1.85*ht)-(4.676*age))/1000 | | |
| Jia | Adults | (2912+56.9*wt-25.1*age)/1000 | -1.63(1.96) | 0.7200 |
| Liu | 20 to 78 years | (58.074*wt +17.405*ht -14.35*age+277.36)/1000 | -1.08(1.76) | 0.7966 |
| Maffesis | 6-10 years old | | -1.70(2.14) | |
| Boys | | ((28.6*wt)+(23.6*ht)-(69.1*age)+1287)/1000 | | |
| Girls | | ((35.8*wt)+(15.6*ht)-(36.3*age)+1552)/1000 | | |
| Monlar 1 | 10-16 years old | ((50.2*wt)+(29.6*ht)-(144.5*age)-(550*sex)+594.3)/1000 | -1.06(1.88) | 0.7425 |
| Monlar 2 | | | | |
| Boys | | ((50.9*wt)+(25.3*ht)-(50.3*age)+26.9)/1000 | -1.06(1.84) | 0.7614 |
| Girls | | ((51.2*wt)+(24.5*ht)-(207.5*age)+1629.8)/1000 | | |
| Schofield | Newborns to adults | | 0.33(1.61) | 0.8531 |
| Boys | | (0.082*wt)+(0.545*(ht/100))+1.736 | | |
| Girls | | (0.071*wt)+(0.677*(ht/100))+1.553 | | |
| WHO | Adults | | -0.31(1.87) | 0.7648 |
| Boys | | ((16.6*wt)+(77*(ht/100)+572))*4.18/1000 | | |
| Girls | | ((7.4*wt)+(482*(ht/100))+217)*4.18/1000 | | |
| Tverskaya | 3-18 years old | (775+(28.4*FFM)-(37*age)+(3.3*FM)+(82*sex))*4.18/1000 | -0.79(1.91) | 0.7831 |
| Leung 1 | 16-88 years old | (72.345*FFM-20.423*age+3261.8)/1000 | -1.68(2.06) | 0.6654 |
| Leung 2 | | (57.562*FFM-26.795*age+3340.2)/1000 | -0.70(1.92) | 0.7233 |
| DB 1 | 3-18 years old | 0.1096*FFM+2.8862 | -0.24(1.88) | 0.7896 |
| DB 2 | | 0.1371*FFM-0.1644*age+3.3647 | -0.60(1.93) | 0.7922 |

Wt, weight (kg); ht, height (cm); FM, fat-free mass. Age is given in years. For sex, male=0 and female=1. FM and FFM are given in grams. Difference given in mean (SD). ICC, Interclass correlation coefficient.

for at least 15 minutes, and the mean value of each variable obtained.

Fat free mass were measured by a total body scanner (QDR4500A. Hologic, Waltham, MA, USA) using array mode. This equipment used a switched pulse stable dual-energy-X-ray operating at 100 and 140 kV. An automatic internal reference system with a calibration wheel achieved the fat free mass calculation.

Thirteen existing prediction equations (Table 1) derived from different populations were used for comparing with the measured REE of our subjects.

All subjects had blood samples taken in the morning, after an overnight fast, for the estimation of plasma glucose (GLU), serum insulin (INS) concentrations and plasma lipid profiles [total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL) and low-density lipoprotein cholesterol (LDL) concentrations]. Lipid dysfunction was defined as either TC ≥ 5.2 mmol/L, LDL ≥ 3.4 mmol/L, HDL ≤ 1.2 mmol/L or TG ≥ 2.0 mmol/L. Insulin resistance was assessed by homeostasis model assessment (HOMA) based on fasting serum insulin and plasma glucose concentrations. Homeostasis model assessment was calculated as INS in mIU/l x GLU in mmol/l divided by 22.5. In the absence of sex and age adjusted reference for our population we used HOMA > 4 to classify insulin resistance as this level has been shown to provide a reliable cut-off between healthy children and those at risk of developing type 2 diabetes.[28]

### *Statistics*

SPSS for Windows (14, SPSS, Inc., Chicago, IL) was used in the analysis, and the level of significance was set at 5% for all comparisons.

The demographic data and laboratory results were expressed as median with inter-quartile ranges (IR). Because fat free mass was not normally distributed, logarithmic transformation was applied. Mann-Whitney U test was used to explore the relationship of the factors with gender. The factors associated with resting energy expenditure with a *p*-value less than 0.25 were then analyzed by multiple linear regression analysis, using a forward stepwise selection strategy. When two or more potential factors were highly correlated, the factor that was clinically important was selected for entry. Interclass correlation coefficient was used to evaluate the reliability with the available equations.

## RESULTS

A total of 100 children, 29 girls and 71 boys were recruited. All subjects had no evidence of underlying disease that could have caused secondary obesity on review of the history and clinical examination. Their median age was 12.1 years (IR 9.9 – 14.2) and the median BMI was 29.1 kg/m$^2$ (IR 26.0 – 32.5). The median of percentage above ideal BMI was 163.8% (IR 151.1 – 175.8). There were no sex differences in most of the anthropometric parameters except WHR and conicity index which may not have any clinical significance as the median values were the same in both genders. There was no gender difference in fat free mass and all of the biochemical parameters. The characteristics of the subjects are shown in Table 2.

Biochemical parameters were available in 76 subjects. Half of the missing data were due to refusal for taking

**Table 2.** Descriptive data by gender

| | Male (n=71) | Female (n=29) | *p*-value | Overall |
|---|---|---|---|---|
| Age (year) | 11.7 (9.6-14.0) | 13.4 (11.0-15.0) | 0.120 | 12.1 (9.9-14.2) |
| Weight (kg) | 64.5 (55.0-82.0) | 73.2 (57.4-84.4) | 0.382 | 67.4 (55.8-82.5) |
| Height (cm) | 150.0 (143.0-163.0) | 153.0 (146.5-161.0) | 0.548 | 150.5 (144.0-161.0) |
| Body mass index | 28.8 (25.8-32.5) | 30.0 (27.0-33.8) | 0.272 | 29.1 (26.0-32.5) |
| BMI z-Score | 2.9 (2.7-3.4) | 3.0 (2.6-3.2) | 0.668 | 2.9 (2.6-3.4) |
| % ideal BMI | 163.5 (151.0-176.0) | 164.0 (151.7-175.8) | 0.906 | 163.8 (151.1-175.8) |
| Waist circumference (cm) | 86.5 (81.5-96.0) | 89.3 (78.6-93.8) | 0.880 | 87.5 (80.0-94.5) |
| Hip circumference (cm) | 97.0 (88.5-107.0) | 104.9 (89.8-112.6) | 0.133 | 99.0 (89.5-109.0) |
| Waist-hip ratio | 0.9 (0.9-0.9) | 0.9 (0.8-0.9) | 0.003 | 0.9 (0.8-0.9) |
| Biceps skinfold thickness (mm) | 19.0 (14.5-25.0) | 20.2 (17.0-24.9) | 0.744 | 19.8 (15.5-25.0) |
| Triceps skinfold thickness (mm) | 27.0 (22.0-33.3) | 29.4 (26.1-33.0) | 0.337 | 28.0 (22.5-33.0) |
| Subscapular skinfold thickness (mm) | 33.0 (28.5-36.5) | 34.5 (30.3-40.0) | 0.086 | 34.0 (28.5-38.0) |
| Suprailiac skinfold thickness (mm) | 34.5 (30.0-38.0) | 33.8 (30.5-37.4) | 0.712 | 34.0 (30.0-37.5) |
| Centrality index | 1.1 (1.0-1.3) | 1.2 (1.0-1.3) | 0.624 | 1.1 (1.0-1.3) |
| Conicity index | 1.2 (1.2-1.3) | 1.2 (1.1-1.2) | 0.044 | 1.2 (1.2-1.2) |
| Total % of Fat | 35.9 (31.2-39.8) | 39.0 (36.6-41.3) | 0.010 | 37.3 (33.7-41.1) |
| Fat free mass (kg) | 40.7 (34.9-52.4) | 42.5 (34.9-48.6) | 0.997 | 41.0 (34.9-49.4) |
| Fat mass (kg) | 23.9 (18.1-30.5) | 30.3 (21.0-33.7) | 0.057 | 25.3 (19.5-32.7) |
| Resting Energy Expenditure (MREE) MJ/d | 7.3 (6.3-9.7) | 6.9 (5.6-8.1) | 0.203 | 7.1 (6.2-8.4) |
| Insulin | 21.0 (14.4-37.2) | 26.7 (15.8-36.3) | 0.206 | 22.2 (14.6-37.2) |
| Insulin pmol/L | 150.7 (103.5-267.1) | 191.6 (113.4-260.1) | 0.206 | 158.9 (104.8-266.9) |
| Glucose | 5.0 (4.9-5.3) | 5.2 (4.8-5.4) | 0.727 | 5.1 (4.9-5.3) |
| Glu (mg/dL) | 90.5 (88.7-95.9) | 93.2 (86.9-97.7) | 0.727 | 91.4 (88.7-95.9) |
| FGIR | 4.3 (2.5-6.8) | 3.6 (2.8-5.3) | 0.250 | 4.1 (2.5-6.4) |
| QUICKI | 0.3 (0.3-0.3) | 0.3 (0.3-0.3) | 0.309 | 0.3 (0.3-0.3) |
| HOMA | 4.8 (3.2-8.3) | 5.8 (3.4-8.2) | 0.255 | 4.9 (3.3-8.3) |
| Cholesterol mmol/L | 4.6 (4.2-5.3) | 4.6 (4.1-5.0) | 0.572 | 4.6 (4.2-5.2) |
| TAG mmol/L | 1.3 (1.0-1.6) | 1.4 (1.3-1.6 | 0.332 | 1.4 (1.1-1.6) |
| HDL mmol/L | 1.2 (1.1-1.3) | 1.2 (1.1-1.4) | 0.522 | 1.2 (1.1-1.4) |
| LDL mmol/L | 2.8 (2.4-3.3) | 2.7 (2.0-3.2) | 0.474 | 2.8 (2.4-3.2) |

**Table3.** Multivariate linear regression: R2 = 0.7

| | B | SE | *p*-value |
|---|---|---|---|
| Log(Fat free mass) | 17.37 | 1.29 | <0.0001 |
| Conicity index | 11.36 | 2.55 | <0.0001 |
| Centrality index | -2.38 | 0.67 | 0.001 |

Dependent variable: Resting energy expenditure MJ/d

blood and the remaining did not fast prior to taking blood. Some abnormalities of the serum lipid profile were found in 69% of the subjects namely low HDL (50%), high LDL (15%) and high TAG (10%). The medians of TC, TAG, HDL and LDL were 4.6 mmol/L (IR 4.2 – 5.2), 1.4 mmol/L (IR 1.1 – 1.6), 1.2 mmol/L (IR 1.1 – 1.4) and 2.8 mmol/L (IR 2.4 – 3.2), respectively. The medians of fasting glucose, insulin and HOMA were 5.1 mmol/L (IR 4.9-5.3), 158.9 pmol/L (IR 104.8 – 266.9) and 4.9 (IR 3.3 – 8.3), respectively. A third of subjects (n=33) had insulin resistance defined as a HOMA value greater than 4. There were no correlations between MREE and insulin resistance or serum lipid abnormalities.

The median MREE for all study participants was 7.1 mJ/d (IR 6.2 – 8.4) and that for boys and girls were 7.3 mJ/d (IR 6.3 – 9.7) and 6.9 mJ/d (IR 5.6 – 8.1), respectively. There was no sex difference in MREE. The medians of the predicted REE calculated from the 13 existing equations are listed in Table 1. The Derumeaux-Burel (DB) 1 equation was shown to have the lowest MREE-PREE difference of -0.24mJ/d with a interclass correlation coefficient of 0.79. Apart from the Schofield equation, all the remaining equations underestimated REE in our obese Chinese children subjects. Except for the Leung 1 equation, the interclass correlation coefficients for all the remaining equations were above 0.72 showing the reliability of the equations was high. (Table 1)

In multiple regression analysis, fat free mass, conicity and centrality indexes were independent predictors of MREE. ($R^2 = 0.7$). (Table 3)

A new equation for local obese children was derived as: REE = (17.4*logFFM) + (11.4*conicity index)$^a$ – (2.4*centrality index)$^b$ – 31.3. {$^a$Conicity index = waist circumference (meters)/ 0.109 sq root [weight (Kg)/height (Meters)]; $^b$Centrality index = subscapular/triceps skinfold}. The mean difference of new PREE-MREE was -0.01 mJ/d (SD 1.51) with an inter-class correlation of 0.91.

## DISCUSSION

There is currently no reliable and validated equation for estimating REE in obese Chinese children. In this study, we have derived a new equation using free fat mass (FFM), conicity index and centrality index as predictors for use in our locality.

Double labelled water is the only method that directly measures energy expenditure but it is both labour and time consuming thus this method is limited to laboratory-based research. Indirect calorimetry remains the only simple, accurate and clinically feasible method for measuring energy expenditure. The procedure involves sophisticated equipment and experienced personnel are required for its proper operation. Prediction equations have since been derived in serving the purpose to simplify REE estimation without the need to rely on machinery.

The Derumeaux-Burel (DB) equations were derived from 752 obese Caucasian subjects aged 3-18 years. That study was broken into 2 phases, the first involved 471 subjects. The equation derived from the first phase was validated in the second phase that included 211 subjects. Fat free mass (FFM) was the single largest contributing factor in equation 1 (REE = 0.1096FFM + 2.8862) while the more minor factor of age was considered in equation 2 (REE = 0.1371FFM – 0.1644age + 3.3647).[29] In our cohort, FFM was shown to be an independent predictor of REE instead of age, and this may explain why equation 1 was superior in assessing REE in our cohort. Resting energy expenditure (REE) is well known to relate to age and pubertal onset. The age range of our cohort was narrow and may contribute to the contradictory result when compared to previous published studies. The Harris-Benedict (HB) as well as the Food and Agriculture/World Health Organization/United Nation University (FAO/WHO/UNU) equations were mainly derived from a large adult population, when up to 7000 adults were recruited.[5,6] The lack of children's data in forming these two equations could explain their poor performance in our cohort, both equations under-estimated REE in our children. The Jia, Liu and Leung equations derived from Asian and Chinese adult populations also underestimated REE in our obese Chinese children.[11-13] This is consistent with other studies that show that adults have significantly lower REE than children. The Maffeis, Monlar and Tverskaya equations were derived from obese Caucasian children with FFM measured either by skinfold or impedance. They also underestimated REE in our study.[7-9] Ethnic differences and more accurate measurement of FFM by DEXA in our study may explain this discrepancy. The Schofield equation which was the only equation that overestimated REE in our children, was derived from a mix of both obese and non-obese Caucasian children.[10] The difference in methodology may explain this discrepancy, as not all the subjects in the Schofield study were pre-prandial prior to the measurement of REE. In contrast to other studies, we detected no sex difference in REE. Our cohort was male predominant (71%), and there may have been insufficient power to detect a difference.

Apart from the degree of adiposity, the way body fat is distributed is also important in estimating REE. Both conicity and centrality indexes were significant predictors for REE in our study. These associations had not been reported previously. In the calculation of REE, body fat distribution seems to play an extra role in children with higher degree of adiposity.

There was no association between insulin resistance and REE. Insulin resistance can be genetically determined or as a consequence to obesity. For the latter, it may lead to a higher REE by increasing protein turnover and gluconeogenesis and hence, serves as defence mechanism for further weight gain.[21] We did not demonstrate any association between REE and insulin resistance in our population. The HOMA cut-off was arbitrarily set at 4 to classify insulin resistance[28] and the median HOMA values were high for both males (4.8) and females (5.8) in our subjects. The narrow range of insulin resistance in our

population may not demonstrate any significant trend of association.

The newly developed REE prediction equation can only be applied to individuals who share similar characteristics as our study subjects. Further validation studies involving healthy Chinese children are needed.

**AUTHOR DISCLOSURES**

All authors declare that they have no conflict of interest in connection with this paper.

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Original Article

# Validation of prediction equations for estimating resting energy expenditure in obese Chinese children

Dorothy FY Chan FHKAM[1], Albert M Li FHKAM[1], Michael HM Chan FRCPA[2], Hung Kwan So PhD[1], Iris HS Chan BSc[2], Jane AT Yin Bsc[1], Christopher WK Lam PhD[2], Tai Fai Fok FRCPCH[1], Edmund AS Nelson FRCPCH[1]

[1]*Department of Paediatrics, The Chinese University of Hong Kong, Shatin, Hong Kong SAR*
[2]*Department of Chemical Pathology, The Chinese University of Hong Kong, Shatin, Hong Kong SAR*

# 評定靜息能量消耗預測方程在肥胖華裔兒童的效度

目的：(1) 評定現存的靜息能量消耗預測方程(PREE)在肥胖華裔兒童的效度，(2) 探討測量的靜息能量消耗(MREE)與體位測量和生化指標的關聯，(3) 建立本地的靜息能量消耗預測方程。設計：橫斷面研究。受試者：100 名肥胖兒童，當中 71 位是男孩。測量：所有受試者均接受身體檢查和體位測量。上身和中央體脂分佈，分別用向心性指標(centrality index)和圓錐度指標(conicity index)表示。靜息能量消耗(REE)是以間接熱量計量度。應用雙能 X 線吸收測量法(DEXA)量度非脂肪組織(FFM)。以測量肥胖兒童得到的靜息能量消耗和 13 項現存的靜息能量消耗預測方程進行比較，並記錄空腹血糖、血脂及胰島素的結果。結果：整體、男孩和女孩的 MREE 中位數分別是 7.1 mJ/d (IR 6.2-8.4)、7.3 mJ/d (IR 6.3-9.7)及 6.9 mJ/d (IR 5.6-8.1)，MREE 未見性別差異($p$=0.203)。除了 Schofield 的方程外，大部份的預測方程都低估了我們兒童的 REE。應用多元線性回歸發現 MREE 與 FFM($p$<0.0001)，圓錐度指標($p$<0.001)及向心性指標($p$=0.001)呈正相關。為本地兒童制定的新方程：REE = (17.4*logFFM) + (11.4*圓錐度指標) – (2.4*向心性指標) – 31.3。新方程的 PREE-MREE 的平均差值是-0.011 mJ/d (SD 1.51)，其組間相關係數為 0.91。結論：所有現存的靜息能量消耗預測方程應用在華裔肥胖兒童時皆不能做出準確預測，因此我們為本地兒童制定新的方程。

**關鍵字：肥胖、兒童、體脂分佈、靜息能量消耗、胰島素阻抗**