**Faster eating rates are associated with higher energy intakes during an *Ad libitum* meal, higher BMI and greater adiposity among 4.5 year old children – Results from the GUSTO cohort.**

Anna Fogel1, Ai Ting Goh1, Lisa R. Fries2, Suresh Anand Sadananthan3,S.Sendhil Velan3,4, Navin Michael3, Mya Thway Tint5, Marielle Valerie Fortier6, Mei Jun Chan3, Jia Ying Toh3, Yap-Seng Chong3,5, Kok Hian Tan7, Fabian Yap7, Lynette P. Shek3,8, Michael J. Meaney1,9, Birit F.P. Broekman 3,10, Yung Seng Lee3, 8, Keith M. Godfrey11, Mary Foong Fong Chong1,12 & Ciarán Gerard Forde1,13\*

1 Clinical Nutrition Research Centre, Singapore Institute for Clinical Sciences, Agency for Science, Technology and Research (A\*STAR), National University Health System, Singapore.

2 Nestle Research Center, Lausanne, Switzerland.

3Singapore Institute for Clinical Sciences, A\*STAR, Singapore.

4Singapore Bio-Imaging Consortium, A\*STAR, Singapore.

5Department of Obstetrics and Gynaecology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore.

6Department of Diagnostic and Interventional Imaging, KK Women’s and Children’s Hospital, Singapore.

7KK Women’s and Children’s Hospital, Singapore

8Department of Paediatrics, Yong Loo Lin School of Medicine, National University of Singapore, Singapore,

9Douglas Mental Health University Institute, McGill University, Montréal, Canada.

10Department of Psychological Medicine, Yong Loo Lin School of Medicine, National University of Singapore, National University Health System, Singapore.

11Medical Research Council Lifecourse Epidemiology Unit and National Institute for Health Research Southampton Biomedical Research Centre, University of Southampton and University Hospital Southampton NHS Foundation Trust, Southampton, United Kingdom

12 Saw Swee Hock School of Public Health, National University of Singapore, Singapore

13Department of Physiology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore.

**Authors' last names:** Fogel Goh Fries Sadananthan VelanMichaelTint Fortier ChanTohSengHianYapShekMeaney Broekman Lee Godfrey Chong Forde

\***Author to whom correspondence should be addressed**: **Ciaran Gerard Forde**; Centre for Translational Medicine, 14 Medical Drive #07-02, MD 6 Building, Yong Loo Lin School of Medicine, Singapore 117599;Tel: +65 64070104; ciaran\_forde@sics.a-star.edu.sg

**Sources of Support:** This work is supported by the Translational Clinical Research (TCR) Flagship Program on Developmental Pathways to Metabolic Disease funded by the National Research Foundation (NRF) and administered by the National Medical Research Council (NMRC), Singapore-NMRC/TCR/004-NUS/2008. Additional funding is provided by the Singapore Institute for Clinical Sciences, A\*STAR and Nestec SA. KMG is supported by the National Institute for Health Research through the NIHR Southampton Biomedical Research Centre and by the European Union's Seventh Framework Programme (FP7/2007-2013), project Early Nutrition under grant agreement n°289346.

**Abbreviations: BMIz- Body Mass Index z-score; BAi- Body Adiposity Index; SAT- Subcutaneous Adipose Tissue; VA- Visceral Adiposity**

**Clinical Trial Registry Number:** NCT01174875; https://clinicaltrials.gov/

**Abstract**

Faster eating rates are associated with increased energy intake, but less is known about the relationship between children’s eating rate, food intake and adiposity. We examined whether children who eat faster consume more energy and whether this is associated with higher weight status and adiposity. We hypothesized that eating rate mediates the relationship between child weight and *ad libitum* energy intake. Children (N=386) from the Growing Up in Singapore towards Healthy Outcomes (GUSTO) cohort participated in a video-recorded *ad libitum* lunch at 4.5 years to measure acute energy intake. Videos were coded for three eating-behaviours (bites, chews and swallows) to derive a measure of eating rate (g/min). Body mass index (BMI) and anthropometric indices of adiposity were measured. A subset of children underwent MRI scanning (n=153) to measure abdominal subcutaneous and visceral adiposity. Children above/below the median eating rate were categorised as slower and faster eaters, and compared across body composition measures. There was a strong positive relationship between eating rate and energy intake (r=0.61, p<0.001) and a positive linear relationship between eating rate and children’s BMI status. Faster eaters consumed 75% more calories than slower eating children (Δ131 kcal, 95%CI [107.6, 154.4], p<0.001), and had higher whole-body (p<0.05) and subcutaneous abdominal adiposity (Δ118.3 cc; 95%CI [24.0, 212.7], p=0.014). Mediation analysis showed that eating rate mediates the link between child weight and energy intake during a meal (b=13.59, 95% CI [7.48, 21.83]). Children who ate faster had higher energy intake, and this was associated with increased BMIz and adiposity.

**Key words:** Eating rate; Energy intake, Adiposity; Childhood obesity, Mastication; Children

**Introduction**

A key recommendation from the 2016 Ending Childhood Obesity (ECHO) committee report (1) is to investigate the behavioural responses of children to the modern obesogenic food environment as a critical element to tackle childhood obesity. The report recognised that eating behaviours emerge and stabilise early in life, and are linked with higher energy intakes and rapid weight gain among children under 5 years of age (2). While a number of genetic, epigenetic and environmental risk factors have been identified in childhood obesity, these often manifest in habitual eating behaviours that support sustained positive energy balance and weight gain (3, 4.). Eating behaviours have been shown to be highly heritable and linked with common obesity related gene variants such as FTO (5-9).

One of the eating behaviours previously studied in the context of energy intake and obesity risk is rate of eating. Research on adults has shown that people who eat faster tend to consume more energy during a meal (10), and longitudinal studies have shown an increased risk of weight gain independently of other lifestyle factors (11), of becoming overweight or obese (12-15) and of a range of metabolic diseases (16-18). Behavioural Susceptibility Theory suggests there is a link between genetic factors, appetitive traits and adiposity, and it has been proposed that faster eating rates are a behavioural marker of appetitive traits that predispose children to higher energy intakes and increased risk of weight gain (19, 20). Obese children tend to eat more rapidly than non-obese children (21) and show less variation in their eating patterns (22), highlighting stable behavioural eating patterns by pre-school age. Using data from the Twins Early Development Study, Llewellyn and colleagues (23) demonstrated a heritable component to eating rate and a positive association with BMI status among school-age children. Comparison of microstructural patterns of eating within a meal has shown that obese children have a faster eating rate compared to healthy weight children, achieved by taking larger bites (24) and fewer chews per bite (22, 25, 26). However, some studies have failed to show a link between eating rate and weight status (27, 28).

Eating rate has also been identified as a behavioural marker of prospective weight and fat mass gain in longitudinal studies of child growth and development. Variations in eating speed can already be observed at 2-4 weeks postpartum, and vigorous suckling, akin to faster eating, has been linked with higher energy intakes and prospective weight gain to age 3 years (29), and was predictive of weight gain at 12 and 24 months (30). In a large population-based study faster feeding at ~3 months of age predicted subsequent weight at 9 months more strongly than weight at 9 months predicted subsequent eating speed (at 15 months), supporting the idea that differences in early feeding speed are likely to influence early weight gain (31). In pre-school children, rate of eating at age 4 years was predictive of prospective weight gain, whole-body adiposity and abdominal adiposity at age 6 years independently of maternal weight status (32), supporting a link between rapid eating and weight gain. Whole-body and abdominal adiposity are important risk factors for type 2 diabetes (33) and metabolic syndrome (34), and are particularly problematic in South Asian populations, who show increased levels of adiposity within the healthy-range of BMI, and onset of metabolic diseases at lower BMI(35). Previous research has highlighted the need for further studies linking children’s eating behaviours, energy intake and body composition measures across different ethnic groups (32). It has been previously demonstrated that self-reported eating rate shows stronger associations with prevalence of overweight in younger compared to older Asian children (36). No study to date has investigated variations in Asian children’s observed eating rates and related this to their energy intake and body composition. Since BMI is a poor summary measure of adiposity among Asian children (35), comprehensive assessments were taken to estimate total adiposity using anthropometry and abdominal adiposity by MRI. The present study investigated the relationship between eating rate and *ad libitum* energy intake during a meal among 4.5 year old Singaporean children. Secondly, we explored whether eating rate was related to children’s BMI z-score (BMIz) and adiposity. We predicted that (i) children who eat at a faster rate would consume more energy during an *ad libitum* meal and that (ii) faster eating would be associated with higher body weight and adiposity. To examine whether faster eating rate is associated with energy intake during a meal independently from energy requirements, we tested a model in which (iii) the association between body weight and energy intake during the meal is mediated by children’s eating rate.

**Subjects**

The 483 mother-child dyads studied were a subset of the larger *Growing Up In Singapore Towards Healthy Outcomes* cohort (GUSTO; N=1247(37)). Participants took part in a video-recorded *ad libitum* buffet lunch task at age 4.5 years (54±2 months). After removal of videos due to non-compliance with the test protocol (e.g. child leaving the room while eating or children sharing their food with a parent; n=97), the final sample consisted of 386 parent-child dyads from three ethnicities: Chinese (n= 210), Indian (n= 68) and Malay (n= 108), and balanced child sex (n=202 boys and n=184 girls). Children whose data were not included in the analyses did not differ from the analysed sample in energy consumed, frequency of foods chosen, gender, ethnicity, BMI or any other anthropometric measures (p>0.05). The study was approved by the Institutional Review Boards of the hospitals involved (clinical trials registry: NCT01174875) and all participants provided informed consent to participate in the meal. A detailed summary of the participant selection and a number of participants considered in various analyses is summarised in the flowchart (Appendix A).

**Methods**

*Ad libitum meal*

Foods served during the meal were provided *ad libitum* in a buffet and comprised 9 commercially available foods and 3 drinks, selected as familiar and accepted products for this age group based on food frequency questionnaire data from the same cohort. The foods and drinks served were: white bread (Gardenia; 2.63 kcal/g; 6 slices), Honey Stars cereal (Nestle; 3.8 kcal/g; 80g), pancakes (Aunty Jemima; 3 kcal/g; 70g), chocolate cake (Sara Lee; 4.3 kcal/g; 80g), cheese (Cowhead; 2.95 kcal/g; 66g), chicken cocktail sausage (Fairprice; 2.95 kcal/g; 192g), chicken nuggets (CP; 2.29 kcal/g; 216g), apple slices (0.44 kcal/g; 204g), canned corn (Hosen; 0.81 kcal/g; 160g), apple juice (Marigold; 0.5 kcal/ml; 6 boxes), full cream milk (Marigold; 0.65 kcal/ml; 6 boxes) and water. Additional portions of each item were also available should any single item have been fully consumed during the meal. Energy content of food items was derived from the Health Promotion Board of Singapore food composition tables (38).

The test room was equipped with video cameras positioned in three corners of the test room. Cameras enabled high resolution video capture of all aspects of the food choice and consumption, and it was possible to zoom in from different angles up to 400% without the loss of resolution, for the behavioural coding of specific eating behaviours. Children were instructed to consume their normal breakfast at home and abstain from consuming foods for the minimum of 3 hours before the meal. Prior to the meal, mothers were requested not to interfere with children’s food choices or portion size selection, but otherwise were free to interact with their child in the usual way. Participants were told that they could eat as much or as little as they wished during the meal, and that they would be given 20 minutes to eat. Extensions of 10 minutes were granted to finish the meal if they run out of time. All products served were weighed before and after the meal, and intake of each food was recorded (g) and later converted to energy consumed using each food’s energy density (kcal).

*Behavioural Coding Analysis*

Previous research has highlighted the validity of behavioural coding analysis of video recordings for use in quantifying eating behaviours (39). Video recordings of food intake were coded for oral processing behaviours using specialized behavioural annotation software (ELAN 4.9.1, Max Planck Institute for Psycholinguistics, The Language Archive, Nijmegen, The Netherlands; (40)). A coding scheme was developed to record the frequencies of three ‘point’ events (frequency counts of bites, chews and swallows), and duration of a single ‘continuous’ event (total oral exposure time in minutes), based on a previous approach (41-43). Using this approach we collected objective oral exposure measures for each food and derived measures to compare eating rates across participants. The time food was in mouth was collected per bite, and cumulated across all bites to produce total oral exposure time (minutes), later used to derive mean eating rate by dividing the grams consumed by the total oral exposure time recorded (g/min). Behavioural video coding was completed by a single trained video-coder and later blind-validated by the second trained video-coder through standard reliability measures to achieve an acceptable level of agreement (≥80%) in line with previously published recommendations (44).

*Anthropometric Measures*

Participants were invited to attend a separate test session for anthropometric measurement, which took place either the next day or within a month from the *ad libitum* buffet task at the same age. These included height, weight, waist and mid-arm circumferences, and triceps, biceps, suprailiac and subscapular skinfold thickness. All measures were taken following standard guidelines and using the recommended anatomical landmarks for children in this age group (45, 46). All measurements were taken twice, and a third measurement was taken if the first two measurements differed by more than 0.5cm. Some children did not assent to some of the measurements and sample size for all of these measurements is outlined in participant flowchart (Appendix A). Height and weight were transformed to BMI z-scores (BMIz) corrected for age and sex according to WHO child growth standards(47) and later used to classify children as healthy weight (BMIz ≤1.96; n=347) or overweight/obese (BMIz > 1.96; n=31). For simplicity overweight/obese children are referred to as “overweight” throughout. Children within the healthy weight group were additionally subdivided into two separate sub-categories to reflect the large variation in their body composition. Children whose BMIz was lower or equal to 0 were classified as lower-healthy weight (n=194) and children whose BMIz was above 0 but lower than the cut-off for overweight were classified as upper-healthy weight range (n=153). Skinfold thickness measures were used to calculate a body adiposity index (BAi) based on the sum of the suprailiac, subscapular, biceps and triceps skinfold thickness (32, 48). Waist and mid-arm circumference were used in the analyses as non-normalised continuous variables.

*MRI Scan for Abdominal Adiposity*

Magnetic resonance (MR) images of the abdomen were obtained for a subset of 158 participants from a 3T MR scanner (Siemens Skyra, VE11A). Sixty axial slices with 5 mm slice thickness and in-plane resolution of 0.94 × 0.94 mm were acquired using a water suppressed HASTE sequence (TR=1000 ms, TE=95 ms) and body matrix coil. A fully automated graph theoretic segmentation algorithm was used to segment and quantify the subcutaneous (SAT) and visceral adipose tissue (VA) depots between the top of liver and top of sacrum(49). The first step of the algorithm employs intensity thresholding to remove non-fat tissues from the fat tissues and create the fat mask. The mask is then classified into SAT and VA compartments using graph cuts. The resultant output images are then edited manually to remove bowel contents, stomach, spleen, liver, sternum, vertebral body, gall bladder and pelvic bones that get misclassified as fat due to their bright appearance in the image. The number of voxels in SAT and VA were summed and multiplied by the resolution to get the fat volumes.

*Statistical analysis*

The cumulative frequency of food choice from the *ad libitum* buffet was compared across the group and showed that all foods were chosen with similar frequency, with the exception of apples and corn, which were chosen with lower frequency. The frequency of foods chosen was compared within the cohort across healthy and overweight children, and faster and slower eaters (chi-square analysis). A correlation analysis (Pearson’s r) was conducted to test the first prediction that children who eat faster consume more calories during the meal. This same analysis was then repeated in a two-step regression adjusting for the control variables, as initial descriptive statistics revealed some variations in eating rate by sex and ethnicity. Sex and ethnicity were then controlled in all subsequent analyses. Some children ate for longer than others during the lunchtime meal, and it was necessary to test that the relationship between eating rate and energy intake was independent from duration of eating. Children were subdivided into four eating time quartiles based on active mealtime duration. Moderated regression with simple slopes analysis was conducted to confirm that eating rate was positively linked with energy intake independently of eating duration. Eating rate values were centred for the purpose of simple slopes analysis.

Further analyses compared group differences between the faster and slower eaters. A median split was used to group children into slower (n=192) and faster eating groups (n=194), based on their eating rates. Comparison of the food choices among slower and faster eaters showed no significant trends in the frequency with which individual food items or drinks were chosen (p>0.2). There were no significant differences in energy intakes from liquids between slower and faster eaters (p=0.44), so comparison of energy intakes were based on intake of solid foods only. ANOVA was then used to analyse adjusted group differences between the slower and faster eaters in energy intake, BMIz, anthropometric measures and abdominal adiposity measured using MRI scanning, while controlling for gender and ethnicity.

A mediation model was used to test the third prediction, that the association between children’s BMIz and energy intake during a meal is partially explained by children’s eating rate. In the mediation analysis direct links between BMIz and energy intake and indirect associations of BMIz and energy intake within the meal through eating rate were compared. The size of the indirect effect and 95% bootstrapped CIs were used as an index of successful mediation. This was additionally confirmed using the Sobel test to determine the significance (using alpha level of p<0.05) and magnitude of the mediation effect (50). Two-tailed tests were used in all analyses and p <0.05 were considered statistically significant. All statistical analyses were performed in SPSS version 23.0 (IBM).

**Results**

*Food Choice from the Ad libitum Buffet*

Children were free to choose from any of the 9 food items served and at a group level chose all foods with similar frequency, with the exception of apples and corn which were chosen less frequently. Overweight and healthy weight children did not differ in the frequency of the foods they chose (χ2≤1.5, p>0.10) or in the variety (number) of foods consumed (t=0.49, p=0.62). Children who ate faster consumed a slightly larger variety of foods (Δ0.5 food item; t= 3.55, p<0.001) and chose most foods more frequently, and some significantly more frequently (corn, nuggets, apple and cheese) though these differences in choice were not biased towards foods with higher energy density (χ2≥4.2, p<0.001). Children who ate at a slower rate selected Honey Stars more frequently (χ2=36.7, p<0.001). Differences in energy intake were therefore not driven by selection frequency of individual food items. Comparison of the eating rates of the different foods demonstrated that seven of the nine foods in the sample were consumed at a similar rate (Range: 9.7-11.6 g/min), while Honey Stars were consumed at a slightly slower rate (5.8 g/min) and cheese at a slightly faster rate (15.08 g/min). Foods consumed slightly slower and slightly faster were of similar energy density (3.8 vs. 2.95 respectively).

*Eating rate and Ad libitum energy intake*

Our first aim was to examine if children who eat at a faster rate consume more energy during an *ad libitum* meal. The mean (± SD) eating rate in the sample was 6.9 (3.2) g/min and mean energy consumed during the meal was 241.4 (131.9) kcal. Children who ate faster consumed more energy, with a strong linear association between eating rate and energy consumed (r=0.61, p<0.001; **Figure 1**). Post-hoc power analysis (G\*power 3.1) using exact data distribution in a bivariate normal model with assumed alpha error probability of 0.05 revealed that, the achieved power to detect this effect was 1.0. R2 obtained in this analysis lied outside the lower and upper critical r levels (-0.09, 0.09), hence the null hypothesis that there is no relationship between children's eating rate and energy intake during the meal could be rejected. This relationship remained strong after controlling for the possible effects of sex (β=-0.11, p=0.033), ethnicity (β=-0.05, p>0.05) and BMIz (β=0.21, p<0.001) in a regression model (β=0.60, p<0.001; R2=0.39; F(4,373)=59.91, p<0.001), highlighting the consistency of this association. These results remained consistent when using alternative measures of eating rate such as kcal/min or bites/ min. This demonstrates that regardless of gender, ethnicity and BMIz, children who ate at a faster rate consumed more energy.

Children were free to eat for as long as they wanted within the allotted time and as some children ate for longer than others it was necessary to test that the relationship between eating rate and energy intake was independent of eating duration. The mean time spent eating was 15.1 (5.1) minutes, but this varied considerably across the sample, ranging from 2.0-35.1 minutes. The length of time spent eating was divided into four eating time quartiles from shorter to longer time, and children’s eating rate was compared within each quartile. In a moderated regression analysis, for all children eating rate was positively linked to energy consumed independently of meal duration (R2= 0.56; F(7,378)=70.93, p<0.001). **Figure 2** shows the positive relationship between eating rate and energy consumed among children in each of the four eating time quartiles. The simple slopes analysis confirmed that children who ate faster consumed more energy, within each of the active eating time quartiles.

*Differences in Energy Intake Based on Faster and Slower Eating Rates*

A median split was used to group children into slower (n=192) and faster eaters (n=194) to quantify specific group differences in the eating behaviours measured. Direct comparison of mean eating rates revealed that faster eaters were eating approximately twice as fast (9.33±2.44 g/min) as the slower eaters (4.43±1.43 g/min). The faster eating group (306.76±9.9 kcal) consumed on average 131.45 kilocalories more than the slower eating group (175.31±6.09 kcal), which equates to 75% more energy consumed during the meal (F(1,381)=121.12, p<0.001, 95%CI [107.6, 154.4]; **Figure 3**). Effect size calculation of differences in energy intake among slower and faster eaters with alpha error probability of 0.05 revealed large effect size of d=1.15 (51). Faster eaters consumed more of all served foods (t≥1.8, p<0.006), except for the Honey Stars which showed higher intakes among the slower eaters (t=4.5, p<0.001).

*Comparison of Eating Rates Across Healthy Weight and Overweight Children*

Our second aim was to examine the association between eating rate and body composition. Child BMIz scores increased with children’s eating rate (r=0.20, p<0.001). Specific analysis of group differences between healthy weight and overweight children (by BMIz) showed that overweight children ate significantly faster than did the healthy weight children (F(1,371)=4.31, p=0.039). When children were further divided into lower-healthy and upper-healthy range of BMIz, there was again a linear increase in eating rate with increase in weight status (F(2,373)=5.33, p=0.005), with children in the lower-healthy range of BMIz eating significantly slower than children in the upper healthy (p=0.046) or overweight groups (p=0.008). Group differences are shown in Figures 4(a) and 4(b).

*Relations between Eating Rates and Anthropometric Indices of Adiposity*

There was a positive relationship between eating rate and all anthropometric adiposity indices (**Table 1**). Examining differences between slower and faster eaters, faster eaters had significantly higher adiposity across all anthropometric measures (Table 1). Adiposity in biceps and suprailiac region and the overall body adiposity index showed similar but non-significant trends. These results highlight that those children who ate at faster rates had higher anthropometric indices of adiposity.

*Relations between Eating Rate and MRI measures of Abdominal Adiposity*

MRI scanning for abdominal adiposity was performed on 158 children. The data revealed five outliers with unusually high subcutaneous and/or visceral adiposity (z>4.0); these children were removed and analyses completed on a reduced sample (n=153). Comparing faster (n=88) and slower eaters (n=65), faster eaters had 26% higher subcutaneous adipose tissue volume than did slower eaters (means 571.0 ± 30.9 vs 452.7± 35.8 cc, F(1,148)=6.14, p=0.014), and a non-significant trend to have higher visceral adiposity (185.8 ± 5.6 vs 172.3 ± 6.5 cc, F(1,148)=2.44, p=0.12; 95%CI [24.0, 212.7; **Figure 5**). Effect size calculation of differences in subcutaneous adiposity between slower and faster eaters with alpha error probability of 0.05 revealed small-to-medium effect size d=0.42 (51).

*Mediating effects of eating rate on the relationship between BMIz and energy intake*

A mediation model was tested which proposes that greater energy intakes occur in the presence of faster eating rates rather than solely as a result of greater BMIz. Mediation analysis showed that there was a weak, non-significant relationship between child BMIz and energy consumed during the meal (direct effect; p=0.072). However, there was an indirect relationship between BMIz and energy intake when children’s eating rate was included in the mediation, indicating a mediating effect of eating rate on the relationship between BMIz and intake. This was, supported by 95% CI [7.48, 21.83], with a medium effect size (ĸ2=0.14, 95% BCa CI [0.08, 0.21](52). The Sobel test confirmed the statistical significance of this mediation (b= 13.58, SE= 3.34, z= 4.06, p<0.001), indicating a significant improvement in the association, and that heavier children tended to eat faster, and faster eating rates were positively linked to increased energy intake. Importantly, the link between BMIz and energy intake was best explained when child eating rate was included as a mediating factor (**Figure 6**). This highlights that children with higher BMIz scores did not necessarily always consume more energy, but they did so when eating at a faster rate. In this regard, greater energy intake occurs in the presence of faster eating rates, rather than strictly as a result of higher body weight and energy needs.

**Discussion**

Children who eat faster had increased *ad libitum* energy intake, and this was associated with increased BMIz and increased levels of whole body and abdominal adiposity. Mediation modelling showed that explaining energy consumed during the meal through children’s eating rates led to a stronger association with energy intake, than the direct link between BMI and energy intake in isolation.

These results confirm that faster eating rates were associated with increased energy intake within a meal and demonstrate an association with higher BMIz scores and adiposity. We found a two-fold difference in calories consumed between the slower and the faster eating children, with faster eaters consuming on average 130 kcal more than the slower eaters. Previous research has demonstrated that slower eating rates occur through smaller bite sizes(53) and longer chewing times(26, 54) and have been suggested to enhance the satiating capacity of energy consumed through longer oro-sensory exposure time(55). Analyses of the microstructural patterns of eating of children from the current study revealed that children who ate faster did so by taking larger bites that were chewed less and had shorter overall oro-sensory exposure time(26). Longer oro-sensory exposure has a dual protective effects against overconsumption by promoting satiation through the earlier termination of eating(56) and by increasing inter-meal satiety(57). The children in our study were free to consume food for as long as they liked within the allotted time, and we compared eating rates among children who varied in their eating duration. Irrespective of eating duration, children who were eating at a faster rate, consumed significantly more energy and those who ate for the longest duration and at the fastest rate, consumed the most energy. It may be that the children who eat at a faster rate have higher motivation to eat, are more food responsive and may be less sensitive to fullness feelings(58). The underlying appetitive traits and motivations that underpin longer and faster eating rates remain unclear, but will be central to a better understanding of the mechanisms behind the obesogenic eating style observed in the current study.

Our study highlights a strong linear relationship between children’s eating rates, energy intake and body composition across a wide range of measurements. This suggests that the higher intake observed among faster eating children may be indicative of stable and habitual faster eating rates, which support higher energy intake and positive energy balance. At age 4.5 years, children within the upper range of healthy BMIz exhibited eating rates similar to those of overweight children, putting them at risk for future weight gain and indicating a possible role of faster eating rates in the transition from normal weight to overweight and obesity. Among South Asian populations, measures of BMIz should be considered alongside more detailed adiposity measures, as obesity related metabolic disorders often present at lower BMIz compared to non-Asian populations (59, 60). This is particularly the case for risk of type 2 diabetes, where previous research has highlighted greater insulin insensitivity and higher circulating levels of blood glucose among Asians compared to Caucasians(61), and onset of type 2 diabetes occurs at lower ranges of BMI among Asian populations compared to Caucasians(62). The largest relative differences between the slower and the faster eaters were noted in abdominal subcutaneous adipose tissue with a mean difference of 121 cc. Abdominal adiposity is a risk factor for metabolic syndrome, type 2 diabetes and other cardiovascular health problems (60, 63, 64) and as adipocytes quantity stabilises early in childhood and translates into metabolic risk during adulthood, preventing early childhood weight gain and adipogenesis is key to decreasing future metabolic risk(65). The relationship between eating rate and fat mass gain is not well understood, although results from animal studies offer potential clues on the mechanisms linking faster eating rates to fat accumulation. Rodent studies on eating rate suggest that faster eating rate may increase glucose response and over time induce problems with glucose metabolism and fat accumulation(66). A faster eating rate and shorter oro-sensory exposure time also decrease post-prandial thermogenesis and promote white adipose tissue accumulation(67). Recent human studies provide further support for metabolic effects of eating rate, with increased post-prandial thermogenesis among slower eaters(68).

When describing the relationship between eating rate, energy intake and body composition it is necessary to consider the directionality of the findings. The current data reflect eating rate measured on a single occasion, therefore it is not possible to confirm whether faster eating rates are causing weight gain, or whether higher weight and adiposity levels encourage faster eating rates to increase energy intake and sustain higher energy needs. Longitudinal research with measures of eating rate, energy intake and body composition over time are needed to further explore the likely direction of the relationship between rate of eating, energy intakes and later risk of excess weight gain and adiposity. Previous research has described faster eating as a heritable trait(23) that can be identified in the first months of life(6, 29). In line with Behavioural Susceptibility Theory, both genetic and environmental factors shape early eating behaviours and appetitive traits, and may emerge across a range of different food related behaviours such as selecting larger portions, plate cleaning behaviours and faster eating rates(69). There is also a strong learned component to the development of stable eating behaviours and appetitive traits and children’s trajectory of learning to eat is influenced by their early experiences with food and the family feeding environment. Feeding practices such as breastfeeding(70) and the timing of complementary food introduction(71) may interact with genetic risk to influence food texture acceptance, orofacial muscle development and oral stamina(72). Parental feeding practices such as verbal or physical prompting to eat during a meal have also been linked to promoting faster eating rates and higher energy intakes and may influence overweight and healthy weight children differently(73). In addition, early life experiences with food textures and feeding practices may influence food preferences and late introduction of food textures may lead to a later aversion to harder foods(74), and through this may encourage selection of softer foods that can be consumed at a faster rate.

Eating rate has been identified as a modifiable risk factor for childhood obesity and a potential target for behavioural intervention (75). Children who eat quickly may naturally have a higher rate of eating, but can also avoid selecting foods that require extensive mastication and can be consumed at a higher rate. Children’s eating rate can be reduced through the use of external timing prompts, and eating rate monitors (76, 77), which have been shown to successfully reduce eating rate and support successful weight loss in adults and children (75-78). Rather than relying on external cues to prompt eating rate changes, a more natural strategy for long-term sustainable reductions in eating rate would be to promote smaller bites and longer chewing cycles through modified food textures or serving utensils. Previous research with adults has demonstrated food influences on eating rate (41, 43, 79, 80) and the impact of substituting equally liked food textures within a meal to promote changes in eating rate and reduce overall energy intake (42, 81). To date no study has demonstrated the efficacy of a food texture intervention to slower eating rates and reducing energy intake and body weight in children.

The current study surveyed a large representative sample of children, with extensive body composition measures and objective measures of eating behaviours conducted in controlled laboratory setting. However a limitation of the study was the measurement of eating rate on one meal occasion in a laboratory setting and the modest sample of overweight children by BMIz score classification, although this is consistent with national childhood overweight rates for Singapore (60). To determine whether faster eating rates are playing a causal role in weight gain and adiposity, future studies will need to explore the stability of eating behaviours longitudinally among the same children, and the impact of faster eating on energy intake, body weight and adiposity over time. Moreover, a better understanding of the causes and eating patterns that reinforce faster eating rates will help guide the development of effective strategies to combat the early emergence of this ‘obesogenic’ eating behaviour(26).

**Conclusions**

Children who ate at a faster rate had increased *ad libitum* energy intake, and this was associated with increased BMIz and increased levels of whole body and abdominal adiposity. Future research is needed to confirm the stability of these behaviours and their impact on body composition overtime, and a better understanding of the appetitive traits and eating patterns that promote faster eating rates is necessary for the development of approaches to slower down and reduce energy intake.

**Acknowledgments**

We thank the contributions of study participants, GUSTO study group and all clinical and home-visit staff involved. The GUSTO study group includes Pratibha Agarwal, Arijit Biswas, Choon Looi Bong, Birit FP Broekman, Shirong Cai, Jerry Kok Yen Chan, Yiong Huak Chan, Cornelia Yin Ing Chee, Helen Y. H Chen, Yin Bun Cheung, Audrey Chia, Amutha Chinnadurai, Chai Kiat Chng, Shang Chee Chong, Mei Chien Chua, Chun Ming Ding, Eric Andrew Finkelstein, Doris Fok, Marielle Fortier, Anne Eng Neo Goh, Yam Thiam Daniel Goh, Joshua J. Gooley, Wee Meng Han, Mark Hanson, Christiani Jeyakumar Henry, Joanna D. Holbrook, Chin-Ying Hsu, Hazel Inskip, Jeevesh Kapur, Ivy Yee-Man Lau, Bee Wah Lee, Yung Seng Lee, Ngee Lek, Sok Bee Lim, Yen-Ling Low, Iliana Magiati, Lourdes Mary Daniel, Michael Meaney, Cheryl Ngo, Krishnamoorthy Naiduvaje, Wei Wei Pang, Anqi Qiu, Boon Long Quah, Victor Samuel Rajadurai, Mary Rauff, Salome A. Rebello, Jenny L. Richmond, Anne Rifkin-Graboi, Lynette Pei-Chi Shek, Allan Sheppard, Borys Shuter, Leher Singh, Shu-E Soh, Walter Stunkel, Lin Lin Su, Kok Hian Tan, Oon Hoe Teoh, Mya Thway Tint, Hugo P S van Bever, Rob M. van Dam, Inez Bik Yun Wong, P. C. Wong, Fabian Yap, George Seow Heong Yeo.

**Authors’ Contributions:** This study was conceived and designed by CGF, AF, MFFC and LRF. Clinical analyses were performed by SS, SV, AF, ATG, and CGF and data analysis and interpretation were carried out by AF and CGF. AF and CGF prepared the draft manuscript and all authors reviewed and approved the final draft. This study was given ethical approval by ethical review boards of the KK Women’s and Children’s Hospital and National University Hospital in Singapore.

**Author disclosures:** Keith Godfrey, Lee Yung-Seng and Yap Seng Chong have received reimbursement for speaking at conferences sponsored by companies selling nutritional products. They are part of an academic consortium that has received research funding from Abbott Nutrition, Nestec and Danone. Lisa Fries is an employee of Nestec SA, working at the Nestlé Research Center. The other authors have no financial or personal conflict of interests.

**References**

1. World Health Organization. Report of the commission on ending childhood obesity. 2016.

2. Nishtar S, Gluckman P, Armstrong T. Ending childhood obesity: a time for action. The Lancet.387(10021):825-7.

3. Silventoinen K, Rokholm B, Kaprio J, Sorensen TIA. The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. International Journal of Obesity. 2009;34(1):29-40.

4. Hebebrand J, Hinney A. Environmental and Genetic Risk Factors in Obesity. Child and Adolescent Psychiatric Clinics of North America. 2009;18(1):83-94.

5. Carnell S, Haworth CM, Plomin R, Wardle J. Genetic influence on appetite in children. International Journal of Obesity. 2008;32(10):1468-73.

6. Llewellyn CH, van Jaarsveld CH, Johnson L, Carnell S, Wardle J. Nature and nurture in infant appetite: analysis of the Gemini twin birth cohort. The American journal of clinical nutrition. 2010;91(5):1172-9.

7. Llewellyn CH, van Jaarsveld CH, Plomin R, Fisher A, Wardle J. Inherited behavioral susceptibility to adiposity in infancy: a multivariate genetic analysis of appetite and weight in the Gemini birth cohort. The American journal of clinical nutrition. 2012;95(3):633-9.

8. Llewellyn CH, Trzaskowski M, van Jaarsveld CH, Plomin R, Wardle J. Satiety mechanisms in genetic risk of obesity. JAMA pediatrics. 2014;168(4):338-44.

9. Wardle J, Llewellyn C, Sanderson S, Plomin R. The FTO gene and measured food intake in children. International journal of obesity (2005). 2009;33(1):42-5.

10. Robinson E, Almiron-Roig E, Rutters F, de Graaf C, Forde CG, Tudur Smith C, et al. A systematic review and meta-analysis examining the effect of eating rate on energy intake and hunger. Am J Clin Nutr. 2014;100(1):123-51.

11. Tanihara S, Imatoh T, Miyazaki M, Babazono A, Momose Y, Baba M, et al. Retrospective longitudinal study on the relationship between 8-year weight change and current eating speed. Appetite. 2011;57(1):179-83.

12. Sasaki S, Katagiri A, Tsuji T, Shimoda T, Amano K. Self-reported rate of eating correlates with body mass index in 18-y-old Japanese women. International Journal of Obesity. 2003;27(11):1405-10.

13. Otsuka R, Tamakoshi K, Yatsuya H, Murata C, Sekiya A, Wada K, et al. Eating fast leads to obesity: Findings based on self-administered questionnaires among middle-aged Japanese men and women. Journal of Epidemiology. 2006;16(3):117-24.

14. Maruyama K, Sato S, Ohira T, Maeda K, Noda H, Kubota Y, et al. The joint impact on being overweight of self reported behaviours of eating quickly and eating until full: Cross sectional survey. BMJ. 2008;337(7678):1091-3.

15. Ohkuma T, Hirakawa Y, Nakamura U, Kiyohara Y, Kitazono T, Ninomiya T. Association between eating rate and obesity: a systematic review and meta-analysis. International Journal of Obesity. 2015;39:1589-96.

16. Sakurai M, Nakamura K, Miura K, Takamura T, Yoshita K, Nagasawa SY, et al. Self-reported speed of eating and 7-year risk of type 2 diabetes mellitus in middle-aged Japanese men. Metabolism: clinical and experimental. 2012;61(11):1566-71.

17. Zhu B, Haruyama Y, Muto T, Yamazaki T. Association between eating speed and metabolic syndrome in a three-year population-based cohort study. Journal of Epidemiology. 2015;25(4):332-6.

18. Lee S, Ko B-J, Gong Y, Han K, Lee A, Han B-D, et al. Self-reported eating speed in relation to non-alcoholic fatty liver disease in adults. European Journal of Nutrition. 2015;55(1):327-33.

19. Llewellyn C, Wardle J. Behavioral susceptibility to obesity: Gene-environment interplay in the development of weight. Physiol Behav. 2015;152(Pt B):494-501.

20. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. Am J Clin Nutr. 2008;88(1):22-9.

21. Drabman RS, Hammer D, Jarvie GJ. Eating styles of obese and nonobese black and white children in a naturalistic setting. Addictive Behaviors. 1977;2(2–3):83-6.

22. Drabman RS, Cordua GD, Hammer D, Jarvie GJ, Horton W. Developmental trends in eating rates of normal and overweight preschool children. Child development. 1979;50(1):211-6.

23. Llewellyn CH, van Jaarsveld CH, Boniface D, Carnell S, Wardle J. Eating rate is a heritable phenotype related to weight in children. The American Journal of Clinical Nutrition. 2008;88(6):1560-6.

24. Laessle RG, Uhl H, Lindel B, Muller A. Parental influences on laboratory eating behavior in obese and non-obese children. International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity. 2001;25 Suppl 1:S60-2.

25. Chei C, Toyokawa S, Kano K. Relationship between eating habits and obesity among preschool children in Ibaraki Prefecture, Japan. Japanese Journal of Health and Human Ecology. 2005;71(2):73-82.

26. Fogel A, Goh AT, Fries LR, Sadananthan SA, Sendhil Velan S, Michael N, et al. A description of an ‘obesogenic’ eating style that promotes higher energy intake and is associated with greater adiposity in 4.5 year-old children: Results from the GUSTO cohort. Physiology & Behavior. 2017.

27. Spiegel T. Rate of intake, bites, and chews—the interpretation of lean–obese differences. Neuroscience & Biobehavioral Reviews. 2000;24(2):229-37.

28. Park S, Shin WS. Differences in eating behaviors and masticatory performances by gender and obesity status. Physiol Behav. 2015;138:69-74.

29. Agras WS, Kraemer HC, Berkowitz RI, Hammer LD. Influence of early feeding style on adiposity at 6 years of age. The Journal of Pediatrics. 1990;116(5):805-9.

30. Stunkard AJ, Berkowitz RI, Schoeller D, Maislin G, Stallings VA. Predictors of body size in the first 2 y of life: a high-risk study of human obesity. International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity. 2004;28(4):503-13.

31. van Jaarsveld CH, Llewellyn CH, Johnson L, Wardle J. Prospective associations between appetitive traits and weight gain in infancy. The American journal of clinical nutrition. 2011;94(6):1562-7.

32. Berkowitz RI, Moore RH, Faith MS, Stallings VA, Kral TV, Stunkard AJ. Identification of an obese eating style in 4‐year‐old children born at high and low risk for obesity. Obesity. 2010;18(3):505-12.

33. Freemantle N, Holmes J, Hockey A, Kumar S. How strong is the association between abdominal obesity and the incidence of type 2 diabetes? International Journal of Clinical Practice. 2008;62(9):1391-6.

34. Shah RV, Murthy VL, Abbasi SA, Blankstein R, Kwong RY, Goldfine AB, et al. Visceral Adiposity and the Risk of Metabolic Syndrome Across Body Mass Index: The MESA Study. JACC Cardiovascular imaging. 2014;7(12):1221-35.

35. Ramachandran A, Wan Ma RC, Snehalatha C. Diabetes in Asia. The Lancet. 2010;375(9712):408-18.

36. Murakami K, Miyake Y, Sasaki S, Tanaka K, Arakawa M. Self-reported rate of eating and risk of overweight in Japanese children: Ryukyus Child Health Study. Journal of nutritional science and vitaminology. 2012;58(4):247-52.

37. Soh SE, Tint MT, Gluckman PD, Godfrey KM, Rifkin-Graboi A, Chan YH, et al. Cohort profile: Growing Up in Singapore Towards healthy Outcomes (GUSTO) birth cohort study. International journal of epidemiology. 2014;43(5):1401-9.

38. Singapore HPB. Food and Nutrient Composition Database. Retrieved 2016, from Health Promotion Board. HPB (2016) 2016;<http://focos.hpb.gov.sg/eservices/ENCF/>.

39. Hennequin M, Allison P, Veyrune J, Faye M, Peyron M. Clinical evaluation of mastication: validation of video versus electromyography. Clinical Nutrition. 2005;24(2):314-20.

40. Lausberg H, Sloetjes H. Coding gestural behavior with the NEUROGES-ELAN system. Behav Res Methods Instrum Comput. 2009;41(3):841-9.

41. Forde CG, van Kuijk N, Thaler T, de Graaf C, Martin N. Oral processing characteristics of solid savoury meal components, and relationship with food composition, sensory attributes and expected satiation. Appetite. 2013;60(0):208-19.

42. Bolhuis DP, Forde CG, Cheng Y, Xu H, Martin N, de Graaf C. Slow food: Sustained impact of harder foods on the reduction in energy intake over the course of the day. PLoS ONE. 2014;9(4):e93370.

43. Ferriday D, Bosworth ML, Godinot N, Martin N, Forde CG, Van Den Heuvel E, et al. Variation in the Oral Processing of Everyday Meals Is Associated with Fullness and Meal Size; A Potential Nudge to Reduce Energy Intake? Nutrients. 2016;8(5):315.

44. Haidet KK, Tate J, Divirgilio-Thomas D, Kolanowski A, Happ MB. Methods to Improve Reliability of Video Recorded Behavioral Data. Research in nursing & health. 2009;32(4):465-74.

45. de Onis M, Onyango AW, Van den Broeck J, Chumlea CW, Martorell R. Measurement and standardization protocols for anthropometry used in the construction of a new international growth reference. Food and nutrition bulletin. 2004;25(1\_suppl1):S27-S36.

46. Phenxtoolkit.

47. WHO. Child Growth Standards 2003 [Available from: <http://www.who.int/childgrowth/standards/Technical_report.pdf>.

48. Nightingale CM, Rudnicka AR, Owen CG, Cook DG, Whincup PH. Patterns of body size and adiposity among UK children of South Asian, black African-Caribbean and white European origin: Child Heart And health Study in England (CHASE Study). International journal of epidemiology. 2011;40(1):33-44.

49. Sadananthan SA, Prakash B, Leow MKS, Khoo CM, Chou H, Venkataraman K, et al. Automated segmentation of visceral and subcutaneous (deep and superficial) adipose tissues in normal and overweight men. Journal of Magnetic Resonance Imaging. 2015;41(4):924-34.

50. Hayes AF. Beyond Baron and Kenny: Statistical Mediation Analysis in the New Millennium. Communication Monographs. 2009;76(4):408-20.

51. Cohen J. A power primer. Psychological bulletin. 1992;112(1):155.

52. Preacher KJ, Kelley K. Effect size measures for mediation models: quantitative strategies for communicating indirect effects. Psychological methods. 2011;16(2):93-115.

53. Zijlstra N, de Wijk R, Mars M, Stafleu A, de Graaf C. Effect of bite size and oral processing time of a semisolid food on satiation. The American journal of clinical nutrition. 2009;90(2):269-75.

54. Zhu Y, Hollis JH. Increasing the number of chews before swallowing reduces meal size in normal-weight, overweight, and obese adults. J Acad Nutr Diet. 2014;114(6):926-31.

55. de Graaf C. Texture and satiation: The role of oro-sensory exposure time. Physiology & Behavior. 2012;107(4):496-501.

56. de Graaf C. Why liquid energy results in overconsumption. The Proceedings of the Nutrition Society. 2011;70(2):162-70.

57. Cecil JE, Francis J, Read NW. Relative Contributions of Intestinal, Gastric, Oro-sensory Influences and Information to Changes in Appetite Induced by the Same Liquid Meal. Appetite. 1998;31(3):377-90.

58. Smith CF, Geiselman PJ, Williamson DA, Champagne CM, Bray GA, Ryan DH. Association of dietary restraint and disinhibition with eating behavior, body mass, and hunger. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity. 1998;3(1):7-15.

59. Meininger JC, Brosnan CA, Eissa MA, Nguyen TQ, Reyes LR, Upchurch SL, et al. Overweight and Central Adiposity in School-Age Children and Links With Hypertension. Journal of Pediatric Nursing. 2010;25(2):119-25.

60. Wulan SN, Westerterp KR, Plasqui G. Ethnic differences in body composition and the associated metabolic profile: A comparative study between Asians and Caucasians. Maturitas. 2010;65(4):315-9.

61. Dickinson S, Colagiuri S, Faramus E, Petocz P, Brand-Miller J. Postprandial hyperglycemia and insulin sensitivity differ among lean young adults of different ethnicities. The Journal of nutrition. 2002;132(9):2574-9.

62. Chiu M, Austin PC, Manuel DG, Shah BR, Tu JV. Deriving ethnic-specific BMI cutoff points for assessing diabetes risk. Diabetes Care. 2011;34(8):1741-8.

63. Gishti O, Gaillard R, Durmus B, Abrahamse M, van der Beek EM, Hofman A, et al. BMI, total and abdominal fat distribution, and cardiovascular risk factors in school-age children. Pediatr Res. 2015;77(5):710-8.

64. Sniderman AD, Bhopal R, Prabhakaran D, Sarrafzadegan N, Tchernof A. Why might South Asians be so susceptible to central obesity and its atherogenic consequences? The adipose tissue overflow hypothesis. International journal of epidemiology. 2007;36(1):220-5.

65. Spalding KL, Arner E, Westermark PO, Bernard S, Buchholz BA, Bergmann O, et al. Dynamics of fat cell turnover in humans. Nature. 2008;453(7196):783-7.

66. Bae C-R, Hasegawa K, Akieda-Asai S, Kawasaki Y, Cha Y-S, Date Y. The Short-Term Effects of Soft Pellets on Lipogenesis and Insulin Sensitivity in Rats. Preventive Nutrition and Food Science. 2014;19(3):164-9.

67. Oka K, Sakuarae A, Fujise T, Yoshimatsu H, Sakata T, Nakata M. Food texture differences affect energy metabolism in rats. Journal of Dental Research. 2003;82(6):491-4.

68. Hamada Y, Kashima H, Hayashi N. The number of chews and meal duration affect diet‐induced thermogenesis and splanchnic circulation. Obesity. 2014;22(5):E62-E9.

69. Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. Pediatrics. 1998;101(Supplement 2):539-49.

70. Viggiano D, Fasano D, Monaco G, Strohmenger L. Breast feeding, bottle feeding, and non-nutritive sucking; effects on occlusion in deciduous dentition. Archives of Disease in Childhood. 2004;89(12):1121-3.

71. Coulthard H, Harris G, Emmett P. Delayed introduction of lumpy foods to children during the complementary feeding period affects child's food acceptance and feeding at 7 years of age. Matern Child Nutr. 2009;5(1):75-85.

72. Wang XT, Ge LH. [Influence of feeding patterns on the development of teeth, dentition and jaw in children]. Beijing da xue xue bao Yi xue ban = Journal of Peking University Health sciences. 2015;47(1):191-5.

73. Drucker RR, Hammer LD, Agras WS, Bryson S. Can mothers influence their child's eating behavior? Developmental and Behavioral Pediatrics. 1999;20(2):88-92.

74. Coulthard H, Harris G, Emmett P. Delayed introduction of lumpy foods to children during the complementary feeding period affects child's food acceptance and feeding at 7 years of age. Maternal & child nutrition. 2009;5(1):75-85.

75. Ford AL, Bergh C, Södersten P, Sabin MA, Hollinghurst S, Hunt LP, et al. Treatment of childhood obesity by retraining eating behaviour: Randomised controlled trial. BMJ (Online). 2010;340(7740):250.

76. Salazar Vázquez B, Vázquez S, López Gutiérrez G, Acosta Rosales K, Cabrales P, Vadillo‐Ortega F, et al. Control of overweight and obesity in childhood through education in meal time habits. The ‘good manners for a healthy future’programme. Pediatric obesity. 2015;6:484-90.

77. Hamilton-Shield J, Goodred J, Powell L, Thorn J, Banks J, Hollinghurst S, et al. Changing eating behaviours to treat childhood obesity in the community using Mandolean: the Community Mandolean randomised controlled trial (ComMando)--a pilot study. Health technology assessment (Winchester, England). 2014;18(47):i.

78. Ferster CB, Nurnberger JI, Levitt EB. The control of eating. 1962. Obesity research. 1996;4(4):401-10.

79. Forde C, Leong C, Chia E, McCrickerd K. Fast or Slow-Foods? Describing Natural Variations in Oral Processing Characteristics across a Wide Range of Asian Foods. Food & Function. 2016.

80. Viskaal-van Dongen M, Kok FJ, de Graaf C. Eating rate of commonly consumed foods promotes food and energy intake. Appetite. 2011;56(1):25-31.

81. Forde CG, van Kuijk N, Thaler T, de Graaf C, Martin N. Texture and savoury taste influences on food intake in a realistic hot lunch time meal. Appetite. 2013;60:180-6.

Figure 1. Relationship between eating rate and energy consumed during lunch (Pearson’s r; p<0.001; N=386).

Figure 2. Simple slopes analysis representing the moderating effects of time spent eating on the relationship between eating rate (z-scores) and energy consumed during lunch (N=386). The four groups represent active mealtime quartiles from 1 (shortest time spent eating) to 4 (longest time spent eating). The following cut-offs were used: 1 (<11.6 minutes), 2 (11.6<15.01 minutes), 3 (15.01<18.8 minutes), 4 (≥18.8 minutes). Interaction 1 (β= 18.26, p<0.001, 95%CI [11.11, 25.42]). Interaction 2 (β=31.99, p<0.001, 95%CI [26.98, 37.00]). Interaction 3 (β= 28.81, p<0.001 95%CI [21.75, 35.87]). Interaction 4 (β=40.18, p<0.001, 95%CI [26.78, 53.58]).

Figure 3. Energy consumed during lunch by children in the slower and faster eating group (adjusted for sex and ethnicity, mean ± SEM; p<0.001; N=386).

Figures 4 (a, b) Group differences in eating rate between children classified as healthy weight (n= 347) and overweight (n= 31) by BMI status (a) and three groups of children classified as lower (n=194) and upper range (n=153) of healthy weight and overweight (n= 31), by BMI status (Adjusted Mean ± SEM). *\*p<0.05;\*\*p<0.01 ns*= non-significant.

Figure 5. Differences between slower (n= 88) and faster eaters (n= 65) in subcutaneous (SAT) and visceral adiposity (VA) in the abdominal area (Means ± SEM). \*p<0.05

Figure 6. Model of children’s BMI as a predictor of energy consumed mediated by eating rate (n=378).

**Table 1**. Relations between eating rate and adiposity indices (Pearson’s r), group differences between slower and faster eaters (mean ± SEM) and group-level summary of the body composition measures (mean ± SD), controlled for sex and ethnicity.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Measure | Group mean  | r | Slower eaters | Faster eaters | F | sig. |
| BAi (mm) (n=360) | 31.23±11.67 | 0.12\* | 30.0±0.9 | 32.4±0.8 | 3.80 | 0.052 |
| Biceps (n=368) | 6.66 ± 2.44 | 0.04 | 6.54±1.81 | 6.80±0.18 | 1.10 | 0.30 |
| Triceps (n=366) | 10.18± 3.26 | 0.14\*\* | 9.78±0.24 | 10.60±0.24 | 5.97 | 0.015 |
| Suprailiac (n=363) | 6.91±3.68 | 0.10 | 6.59±0.27 | 7.24±0.27 | 2.91 | 0.089 |
| Subscapular (n=368) | 7.52±3.50 | 0.11\* | 7.12±2.55 | 7.92±0.25 | 4.85 | 0.028 |
| Waist (cm) (n=377) | 51.64±5.21 | 0.17\*\*\* | 50.7±0.4 | 52.0±0.4 | 11.45 | 0.001 |
| Mid-arm (cm) (n=377) | 16.89±2.64 | 0.18\*\*\* | 16.4±0.2 | 17.4±0.2 | 11.52 | 0.001 |

\**p*<0.05, \*\**p*<0.01, \*\*\**p*<0.001; BAi= Body adiposity index