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Higher breakfast glycaemic load is associated with increased metabolic syndrome risk, including lower HDL-cholesterol concentrations and increased TAG concentrations, in adolescent girls

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ABSTRACT

Almost all previous studies examining associations between glycemic load (GL) and metabolic risk have used a daily GL value. The daily value does not distinguish between peaks of GL intake over the day, which may be more closely associated with metabolic risk. We aimed to investigate cross-sectional associations between daily and mealtime measures of GL and metabolic syndrome risk, including metabolic syndrome components, in adolescents. Three-day food records and metabolic assessments were completed by adolescents participating in the 14-year follow-up of the Western Australian Pregnancy Cohort (Raine) Study. Breakfast GL, lunch GL, dinner GL and a score representing meal GL peaks over the day were determined in 516 adolescents. Logistic regression models investigated whether GL variables were independent predictors of metabolic syndrome in this population based cohort (3.5% prevalence of metabolic syndrome). Breakfast GL was predictive of metabolic syndrome in girls (OR = 1.15; 95% CI = 1.04,1.27; $P<0.01$) but not in boys. Other meal GL values and daily GL were not significant predictors of metabolic syndrome. When breakfast GL was examined in relation to each of the metabolic syndrome components in girls, it was negatively associated with fasting HDL cholesterol ($P=0.037$; $\beta=-0.004$; 95% CI= -0.008, -0.002) and positively associated with fasting triglycerides ($P=0.008$; $\exp(\beta)=1.002$; 95% CI=1.001, 1.004). Our results suggest that there may be a link between breakfast composition and metabolic syndrome components in adolescent girls. These findings support further investigation into including lower GL foods as part of a healthy breakfast in adolescence, particularly for girls.

INTRODUCTION

The metabolic syndrome is a cluster of metabolic disturbances that increases the risk of developing type 2 diabetes and cardiovascular disease [1; 2]. In Australia, prevalence of metabolic syndrome in adolescents has been previously reported at 3.6% using International Diabetes Federation (IDF) paediatric diagnostic criteria [3], increasing to 22.1% in adulthood (adult IDF criteria) [4]. Diet is one of the factors that may have the ability to influence this progression from adolescence to adulthood.

The glycemic index (GI) was developed 30 years ago with the aim of improving postprandial glycemia in the diabetic population [5]. The GI ranks foods or beverages on their ability to raise blood glucose levels compared to ingestion of the same quantity of carbohydrate, expressed as a percentage. A high GI food consumed in a small amount can have a minimal impact on blood glucose concentrations, and conversely a low GI food consumed in a large amount can have a major impact on blood glucose concentrations. The glycemic load (GL) is a product of the quantity of carbohydrate present in food and the GI; by taking the carbohydrate into consideration, it represents the total impact of the food on blood glucose concentrations [6]. Hence, the GL is better able to distinguish impact on postprandial glycemia compared with the GI.

Habitual dietary intake of a diet with high postprandial glycemia may lead to hyperinsulinemia and disturbed lipid metabolism [7], with increased risk of developing metabolic syndrome [8; 9]. Diets lower in GI/GL have been associated with improved health outcomes for various metabolic risk factors and chronic diseases in studies and meta-analyses [10; 11; 12]. Other studies have not found significant associations between low GI/GL diets and reduced risk of diabetes [13; 14], perhaps in part because the use of daily values has some limitations in representing metabolic processes resulting from habitual dietary carbohydrate intake over the course of the day [13]. Studies investigating associations with dietary GI and GL often use food frequency questionnaires, which can estimate daily GI/GL but not individual meal values. We identified two studies which were able to assess meal values using either a food record (Hong Kong children aged 6-7 years [15]) or diet history (older Australian women [16]). The latter considered a new measure of high glycemic carbohydrate impact, the GL peak score, based on the summation of individual mealtime GLs that scored a peak above the daily GL mean [16]. To date, no published adolescent studies appear to have

examined mealtime patterns of glycemic impact, including investigation of periods when GL intake may peak substantially.

Determining patterns of carbohydrate intake may provide insight into potential glycemic impacts for adolescents, who are also undergoing the stresses of growth, and metabolic and hormonal changes. In this explorative study, we aimed to investigate mealtime measures of GL intake in relation to metabolic syndrome risk, as well as components of the metabolic syndrome, in the 14-year follow-up of the Western Australian Pregnancy Cohort (Raine) Study in Perth, Western Australia. We hypothesised that individual meal GL values and a score representing peaks in meal GL would be better predictors of metabolic syndrome risk than a daily GL value.

RESEARCH DESIGN AND METHODS

Study population

This study is a cross-sectional analysis of adolescents who participated in the 14-year follow-up of the Raine Study. As previously described [17], 2900 pregnant women were enrolled in a controlled trial from public and private antenatal clinics at or near King Edward Memorial Hospital in Perth, Western Australia between May 1989 and November 1991. The resulting 2868 children were recruited for cohort follow-up. The 14-year follow-up (mean age 14.0 ± 0.2 years, age range 13.0–15.0 years) occurred from 2003 to 2005, and was the first to collect comprehensive dietary data allowing nutrient analysis of individual meals in habitual diet. Adolescents with type 1 or type 2 diabetes mellitus or implausible energy intakes (< 3000 or > 20 000 kJ/ day, as previously used in studies of adolescents [18; 19]) were excluded from the study. Informed written consent for the 14-year follow-up procedures was provided by study participants and a parent/guardian, and approval was obtained from the ethics committees of King Edward Memorial Hospital and Princess Margaret Hospital for Children.

Dietary glycemic intake assessment

Three-day food records were completed by the adolescents, with parental support if requested. Intakes were recorded in household measures. Subjects were provided with written and verbal instructions, as well as metric measuring cups and spoons. Consumption away from home was recorded in relation to serve size (for example, two slices of a large pizza or one Whopper hamburger) or estimated in household measures. A checklist ascertained whether each of the three days recorded was typical of the subject's usual intake, and only those records completed and classified as representative were used. A dietitian checked each food diary as it was returned and sought clarification via follow-up telephone calls [20]. Food record data were entered into FoodWorks dietary analysis software (Professional Version 4.00, Xyris Software, Brisbane, Queensland, Australia). Food composition data that were not available through FoodWorks were obtained from a Australian nutrition website with a customized GI database [21]. Where GI values for a specific product were not available, the GI value was imputed from a product or subgroup of products that was assessed by the researchers to be sufficiently similar in terms of type of starch, molecular monosaccharide components, ingredients, including amounts of protein and fat, amount of dietary fibre present, and degree of cooking or processing. If a product was too specialised to be a good

match, (for example, a specific type of body building powder) no GI value was given. GI values for mixed foods and recipes were estimated from component foods, for example, the GI for trifle was based on a weighted GI calculation of the carbohydrate containing ingredients (sponge, jelly and custard). The formula used to calculate the composite GI of meals based on relative weighting of carbohydrate content does not take into account the effect of the whole dish, and there is likely to be a variable loss of discrimination of individual GI values in composite foods.

To ensure that food records were representative, 80% or more of the total daily dietary carbohydrate required an assigned GI value for the record to be included. GL values for individual meals comprised the sum of GL values for all foods and beverages in that meal. Meal GL values were obtained by averaging the values for each particular meal over the three days recorded, to produce daily breakfast, morning tea, lunch, afternoon tea, dinner and supper GL values for each subject. Limited availability of GI values may affect the results of studies examining associations between GI/ GL and chronic disease, particularly when local/ traditional foods are involved. In our cohort, GI values were able to be assigned to 92% of all carbohydrate foods and beverages [(3)]. This meant that for some subjects, carbohydrate foods or beverages in a meal were not able to be allocated a GI value. Non-allocation of a GI meant the contribution of these foods or beverages to the GL for the meal was unable to be calculated (despite having a likely effect on blood glucose levels). To ensure that the GL values we were using were as representative as possible of the food being consumed, we decided that 80% or more of the dietary carbohydrate per meal should be assigned a GI value in order for the meal GL to be used in the study. This was based on methods used in previous research and professional opinion of clinical relevance, whereby a value of lower than 80% was thought to potentially compromise the validity of the data [(3)]. Subjects were excluded if this meant that two or more meals of the same type (eg breakfast) out of the three-day record period did not have usable GL values.

Mean breakfast GL, morning tea GL, lunch GL, afternoon tea GL, dinner GL and supper GL values were calculated for each subject where possible. Together with the mean meal GL (the mean of the above six meal GLs), these were used to produce the peak score GL. Meal peak GL values were calculated for each subject by subtracting the mean meal GL from each meal GL value, and are represented graphically as a set of positive and negative peaks with the

mean set to zero. Peak score GL was calculated by adding all the positive meal peak values [16] (see **Figure 1**). For the purposes of this study, we investigated five GL variables: 1) breakfast GL, 2) lunch GL, 3) dinner GL, 4) peak score GL, 5) daily GL.

Metabolic syndrome definition

Prevalence of metabolic syndrome in this adolescent cohort at the 14-year follow-up has previously been reported as 3.6% or 4.0% [3], using age-specific adolescent definitions from the IDF and the National Cholesterol Education Program Adult Treatment Panel III respectively [22]. While no consistent adolescent definition for the metabolic syndrome exists, the American Heart Association recommends using the IDF paediatric definition for adolescents [23], and this has been used in the current study. The IDF metabolic syndrome definition requires the presence of a high waist circumference in addition to two or more of the following: high systolic or diastolic blood pressure; high fasting serum triglycerides; low fasting serum high-density lipoprotein (HDL) cholesterol; or high fasting plasma glucose concentrations. Cut points for categorization of these high and low subgroups vary by gender and age, as published previously [22]. A research nurse took at waist measurements at the level of the umbilicus from adolescents standing in the anatomical position, to the nearest 0.1 cm until two readings were within a centimetre of each other. Phlebotomists visited adolescents at their homes to obtain fasting blood samples. Serum glucose was measured using an automated Technicon Axon Analyzer (Bayer Diagnostics, Sydney, NSW, Australia), triglycerides were measured using the Cobas MIRA analyser (Roche Diagnostics, Basel, Switzerland), and HDL-C was determined on a heparin–manganese supernatant. PathWest Laboratories at Royal Perth Hospital conducted the biochemistry assays. Six measurements seated blood pressure readings were taken at rest over a 10-minute period using a Dinamap ProCare 100 automatic oscillometric recorder (GE Healthcare Technologies, Rydalmere, NSW, Australia). The first measurement was disregarded, and the mean of the next five measurements was calculated to give diastolic and systolic blood pressure values.

Potential confounding variables

Information regarding potential confounding variables was collected from adolescents themselves and their parents/guardians [3]. Information on physical and sedentary activity was assessed by time spent outside school hours participating in physical activity that caused

breathlessness or sweating (categorized as less than once a week = low exercise, once to three times a week = moderate exercise, or four times or more per week = high exercise), and time spent watching television/videos and using computers for school, work and recreation (categorized as less than two hours per day = low screen use, two to four hours per day = moderate screen use, or over four hours per day = high screen use). These variables were combined into a five category summary variable, which ranged from low screen use with high exercise to high screen use with low exercise. Family characteristics including family structure, family income, maternal age, maternal education and family history of diabetes and cardiovascular disease were supplied by parental report. The Tanner stages of pubic hair development was used to assess puberty status in the cohort [24; 25]. Adolescents were asked to select their corresponding developmental stage from a set of standard drawings depicting Tanner stages two (sparse) to five (adult), in a privately completed questionnaire. Stage one was omitted as an option as this corresponds to a pre-pubescent period (<10 years of age). Dietary variables considered as potential confounding factors in the models included average daily intakes of total energy, total fat, saturated fat, and protein. Body mass index (BMI), calculated as weight in kilograms divided by height in meters squared, was also considered. Trained researchers measured weight to the nearest 100 g using a Wedderburn Digital Chair Scale, and height to the nearest 0.1 cm with a Holtain Stadiometer. Due to the narrow age range in the 14-year follow-up, age was not considered as a confounding factor.

Statistical analysis

Nutrient intakes, including GL measures, were adjusted for total energy using the residuals method to control for confounding and reduce extraneous variation [26]. Continuous measures were expressed as mean ± standard deviation. Student's independent sample t-tests, Mann-Whitney U-tests and Chi-square tests were used to compare subject characteristics between included and excluded adolescent populations. Logistic regression models were used to analyse the relationship between mealtime GL measures and metabolic syndrome, adjusted for potential confounding variables and split by gender (due to significant interaction effects between sex and GL measures). Potential confounding variables were tested in the models. Nagelkerke R^2 values were compared between models, with increasing values indicating better fit [27]. Variables were retained as confounders in the model if they were significant or improved the fit of the model. Models were fitted with and without BMI to allow

comparisons, because BMI is associated with the metabolic syndrome - the definition of metabolic syndrome includes waist circumference. Odds ratios (ORs) and 95% confidence intervals (CIs) were obtained for all variables. Where GL measures were found to be significant predictors of metabolic syndrome, regression models were used to examine associations with continuous measures of metabolic syndrome components (waist circumference, blood pressure, fasting serum triglycerides, fasting HDL-cholesterol and fasting plasma glucose). Components were logged as required to normalise data. BMI was included in each of these analyses, with the exception of waist circumference. No mathematical correction was made for multiple comparisons. Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS Statistics for Windows, version 19.0, IBM corp, New York, USA) and tests used a significance level of 0.05.

RESULTS

Study population

From the original cohort of 2868 at birth, 1286 adolescents in the 14-year follow-up agreed to complete the 3-day food record. Adolescents who completed the 3-day food record were more likely to have older mothers, a higher family income and a lower BMI compared with other adolescents in the follow-up who did not complete a food record [28]. Completed records were returned by 962 subjects [3]. Of these, 822 were considered complete and representative of usual diet. Five subjects were excluded as they had diagnosed diabetes, no subjects were excluded for implausible energy intakes. A total of 516 non-diabetic adolescents provided records where all six meals had at least two GL values to average, and this "two-meal valid" group was used in the statistical models. **Table 1** shows a comparison of subject characteristics for the adolescents between the included (n=516) and excluded (n=306) groups, from the total of 822 adolescents with food dairies that were considered complete and representative of usual diet. Daily dietary carbohydrate intake was found to be significantly higher in the excluded subject group (P=0.028).

Mealtime glycemic carbohydrate intake

Meal GL values are described in **Table 2**. Dinner was the meal with the highest GL value (mean ± SD, 44.9± 20.1), followed by lunch (31.6± 16.5), breakfast (30.9± 14.9), afternoon tea (23.9± 18.6), morning tea (15.5± 13.2) and supper (10.7± 11.5). **Table 2** also provides a breakdown of dietary intake and metabolic syndrome by mean meal GL tertile for boys and girls. Boys and girls with higher mean meal GL values were more likely to have higher energy adjusted carbohydrate intakes and lower protein and fat intakes when compared with boys and girls with lower mean meal GL values ($P<0.05$). From the group of 516 adolescents, 480 had data available to assess metabolic syndrome, which was identified in 17 subjects out of 480 (3.5%). Increasing risk of metabolic syndrome with increasing mean meal GL tertiles was observed in boys but not girls (**Table 2**).

Associations with metabolic syndrome

Final logistic regression models included BMI, single parent family, physical activity and daily protein intake as confounding variables. The other factors investigated did not contribute significantly to the fit of the models, so were not included as confounders. Results of the logistic regression analyses are shown in **Table 3**; there was little difference in odds ratios and significance when BMI was included or excluded as a confounder in these models. Daily GL was not a significant predictor of metabolic syndrome. Breakfast GL was associated with increased risk of metabolic syndrome (OR=1.15; 95% CI=1.04-1.27; P<0.01) in girls. That is, for each unit increase in breakfast GL, the odds of metabolic syndrome increased by a factor of 1.15 (or equivalently, by 15%). With BMI removed from the model, breakfast GL was still a significant predictor (OR=1.06; 95% CI=1.00-1.12; P=0.04). Breakfast GL was not a significant predictor of metabolic syndrome in boys (P=0.15). No other GL values were significant predictors of metabolic syndrome. When breakfast GL was examined in relation to each of the components of the metabolic syndrome in girls, it was negatively associated with fasting HDL cholesterol (P=0.037; β=-0.004; 95% CI= -0.008, -0.002) and positively associated with fasting triglycerides (P=0.008; β=0.002 for logged triglyceride values; exp(β)=1.002; 95% CI=1.001-1.004). That is, for each unit increase in breakfast GL there was a mean decrease in HDL cholesterol of 0.004 mmol/L and a 0.2% increase in the geometric mean fasting triglyceride level.

DISCUSSION

In this study we aimed to explore mealtime measures of GL intake in relation to metabolic syndrome risk as well as components of the metabolic syndrome, in 14-year old adolescents. We hypothesised that meal based GL values would be better predictors of metabolic syndrome risk than a daily GL value. In our group of 516 adolescents, no significant association was found with daily GL values and metabolic syndrome. However, breakfast GL was a significant independent predictor of metabolic syndrome in the same group. As we were comparing GL values on a meal basis, we excluded adolescents where it was not possible to accurately and consistently allocate meal GL values. In a previously published study of the larger Raine Study cohort, a significant association was found with daily GL and metabolic syndrome [3]. It is likely that a reduced sample size meant we were no longer able to detect a significant association with daily GL. We would expect a low prevalence from a paediatric population cohort study rather than a clinical group, and caution must be taken when interpreting the results due to low statistical power to find associations with dietary components [29]. However, our current findings suggest that breakfast GL may be a more sensitive predictor than daily GL in our adolescent group.

Breakfast GL was found to be significantly associated with odds of metabolic syndrome in girls, but not in boys. This association was seen independently and dependently of BMI, so BMI does not appear to mediate the observed association. To put these associations into perspective, our results suggest that if an additional slice of white bread (GL = 12) were added on top of the girls' existing breakfast, the theoretical associated odds of metabolic syndrome would be 5.35 times greater, with an associated 95% CI of 1.60-17.6 times. It must be noted that the confidence interval here is large, due in part to the low prevalence of metabolic syndrome in the study group (n = 17 adolescents; n = 9 girls). Breakfast GL was also found to be significantly associated with two components of the metabolic syndrome, decreased fasting HDL cholesterol and increased fasting triglycerides.

Almost all previous studies using daily GI/GL values have not been able to distinguish between different mealtime effects on glucose and insulin responses, and this may have contributed to conflicting results on whether dietary glycemic carbohydrate intake is a useful

predictor of chronic disease risk [9; 11; 12; 13; 14; 30; 31; 32; 33; 34]. Our findings suggest that breakfast GL may be particularly important. Blood glucose and insulin responses have been shown to be proportional to breakfast GL in clinical trials [31; 32]. Bao et al. [31] suggest that breakfast metabolic responses may not necessarily reflect responses to other meals. In adolescents, clinical trials have shown the benefits of consumption of low-GI carbohydrate at breakfast [35], with increased satiety and reduced consumption at an *ad libitum* lunch, while breakfasts with sufficiently low-GI, multi-grain cereals may produce second meal effects that can last through to lunch or beyond [36]. It is possible that a low-GL breakfast may have the benefit of decreasing the amount eaten at lunch (and potentially the lunch GL), thus reducing the metabolic risk associated with both meals. Effects may differ by age - in older women, O'Sullivan et al. [16] showed that increasing lunch GL was significantly associated with increased risk of insulin resistance, along with peak score GL.

We found that two components of the metabolic syndrome, decreased fasting HDL cholesterol and increased fasting triglycerides, were significantly associated with increasing breakfast GL. Other studies in both youth and adults have also found similar associations with GL. In a randomised controlled trial involving 32 healthy 11 to 25 year olds, higher GL diets were associated with lower HDL cholesterol [37]. In adults, a systematic review and meta-analysis [10] concluded that reduced fasting plasma triglycerides were associated with lower GL diets in adults. In an adult male population, fasting triglycerides were found to increase with increasing dietary GI but not GL, while HDL cholesterol decreased with increasing GL [38]. Risk of developing metabolic syndrome was related to daily GI and GL in Korean women (but not men), with high triglyceride and low HDL cholesterol the components that were associated with high intakes. Although more research is needed to expand on our findings, there are potential mechanisms to explain our results. Habitual intake of high meal GLs can result in hyperglycemia, hyperinsulinemia and disturbed lipid metabolism [7], which have been linked to the development of chronic diseases such as metabolic syndrome and consequent type 2 diabetes and heart disease [31; 39; 40; 41]. Following a high peak in glucose and subsequently insulin, post-prandial hypoglycaemia is common four to six hours after a high GL meal. This can stimulate counter-regulatory hormone secretions that raise glucose and free fatty acids levels [7]. This is linked to increased levels of inflammatory mediators and triglycerides, and decreased HDL cholesterol [42].

In our study we found significant associations in girls, but not in boys. Higher GL diets have been previously associated with a greater risk of the metabolic syndrome in women, but not men [43]. Females may be more innately insulin-resistant than males due to specific sex-linked gene expression, leading to changes in receptor and signalling pathways [44]. In puberty, there is a natural tendency for girls to have more fat gain relative to boys [44; 45]. Hormones in girls such as oestradiol favour fat deposition while those of boys favour muscle tissue accumulation [45]. Increased oestradiol is associated with an increased subcutaneous fat deposition and insulin response, and decreases fatty acid oxidation [46]. Higher fat stores and insulin levels in turn increase secretion of leptin; increased leptin leads to increased oestradiol and subsequent IGF-1 (insulin-like growth factor 1), further increasing insulin secretion and fat storage [45]. Although highly speculative, the effect of hormonal surges at a key stage in puberty is a possible reason for an increased sensitivity to GL in relation to metabolic risk at this time.

Daily dietary protein intake was noted as an important confounding factor in the association of breakfast GL with metabolic syndrome in girls. The adolescents in our study were observed to consume breakfasts with a relatively high GL but low protein content when compared to lunch and dinner. Increasing protein consumption at meals lowers the glycemic response by delaying gastric emptying [47]. A high-protein, low-GI diet produced a combined beneficial effect attributed to reduced insulin response, increased satiety and decreased energy intake in children (5 to 18 years) in the DiOGenes dietary study [48], while higher versus lower protein intake was associated with lower waist circumference and lower LDL cholesterol levels in another paediatric subset of this study [49].Higher protein breakfasts may have the ability to attenuate high-GL responses sufficiently to reduce metabolic syndrome risk. Quality protein for breakfast may lower the meal GL by promoting satiety and by displacing carbohydrate. Further research is required to test this concept

Strengths and limitations

Strengths of this study include the use of three-day food records, which enabled investigation of GL at a mealtime level. Our study also allowed for gender-specific analysis of the group. Limitations of this study include the inability to generalise to other Western adolescent

populations, with the adolescents completing food records in our study more likely to have lower BMIs and older mothers, and come from households with a higher annual income. In reducing the sample size to 480 adolescents to ensure accurate and consistent GL meal data across the three-day record, 17 remained with diagnosed metabolic syndrome, of which nine were female. Subjects excluded had significantly higher intakes of carbohydrates (**Table 1**), and this may have meant that some associations with higher intakes went undetected. The bulk of published GI values come from Australia and the USA [(50)], and despite the high representation of Australian foods, there is a need for a larger GI database of carbohydrate foods commonly consumed by younger populations, such as fast foods and snack bars. Consumption of foods that did not have GI values often occurred at the same mealtime on two consecutive days, which effectively removed a subject from the study each time (via the previously-determined exclusion criterion requiring at least two GL values to average for any one meal). Although many adolescents were removed due to our strict criteria, this method helped to maintain accuracy of the data by ensuring the meal GL values represented a true reflection of the foods reported. Although we attempted to minimise under- and over-reporting through the use of cut-offs previously used in adolescent studies, this method is imprecise and it is possible that we included adolescents in our study who were misreporting their intake. It has been suggested that adolescents with higher BMIs (and therefore at higher risk of metabolic syndrome) are more likely to misreport dietary intake [(18)] and this could affect the associations observed. In addition, this study is a cross-sectional snapshot of the prospective cohort, and as such causality cannot be established.

Implications

In this study we hypothesised that meal based GL values would be better predictors of metabolic syndrome risk than a daily GL value; breakfast GL did appear to have a more sensitive association. Adolescence is an important time for establishing dietary patterns into adulthood, and insight into their impact on disease processes may provide meaningful data to formulate dietary advice. Although we cannot determine causality from our study, it is possible that the addition of low GL foods to breakfast may be beneficial for girls. Our findings support previous recommendations made in this regard around consumption of a low GL breakfast [(51)] [(52)].

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WO, LB and TM were involved in data collection, TO'S conceptualised and supervised the research, AN, MD, AB and TO'S were involved in data analysis and drafting the manuscript, all authors were involved in review of the manuscript.

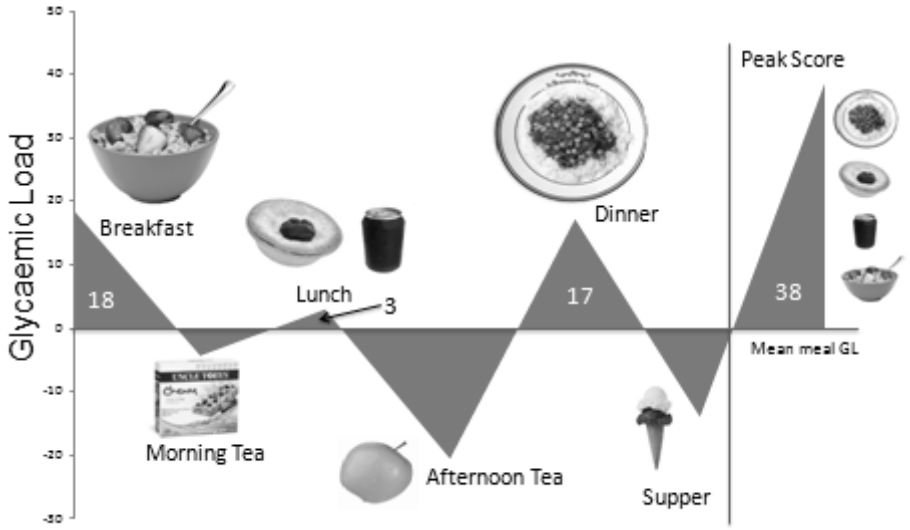

Figure 1. *Glycemic load (GL) variables and food intake for a sample subject in the Raine study (chosen for illustrative purposes only). The mean meal GL was set to zero, producing both positive and negative peaks. For this subject, positive peaks are seen at breakfast (18), lunch (3) and dinner (17). These are summed to create the peak GL score, which is 38 (sum of positive peaks).*

Table 1. *A comparison of adolescent subject characteristics between the populations included (minimum of two-meals with valid GL) and excluded (due to >20% dietary carbohydrate not assigned a GI, insufficient valid meal GL values, or diabetes) from the study, out of the group that returned complete and representative food diaries (n = 822)*

Subject characteristics	**Two-meal valid population** n = 516	**Excluded population** n = 306	**P value** [a]
Characteristics			
Gender (female - n; %)	252; 48.8 %	149; 48.7 %	0.968
Weight categories [b] (n; %)			
Underweight	30; 5.8%	25; 8.2%	
Normal weight	352; 68.5%	225; 73.8%	0.053
Overweight	102; 19.9%	45; 14.7%	
Obese	30; 5.8%	10; 3.3%	
Physical activity participation (n; %)			
4+ times/week	179; 34.8 %	108; 35.4 %	0.980
1-3 times/week	288; 56.0 %	170; 55.7 %	
≤ 1 time/month	47; 9.1 %	27; 8.8 %	
Screen time – computers, TV, video (n; %)			
4+ hours/day	159; 31.2 %	94; 31.1 %	0.766
2-4 hours/day	201; 39.5 %	126; 41.7 %	
< 2 hours/day	149; 29.3 %	82; 27.2 %	
Single parent family (n; %)	97; 19.0 %	47; 15.5 %	0.210
Annual family income (pa, $AUD) (n; %)			
< $35 000	106; 20.9 %	63; 20.9 %	0.956
$35 001 - $70 000	180; 35.6%	110; 36.5 %	
> $70 001	220; 43.5 %	128; 42.5 %	
Maternal education (n; %)			
< Year 12	240; 46.6 %	146; 47.7 %	0.758
≥ Year 12	275; 53.4 %	160; 52.3 %	
Dietary variables			
Energy (kcal/d)	2225 ± 579	2303 ± 584	0.067
Carbohydrate (g/day)	277 ± 79	291 ± 87	0.028
Protein (g/day)	88.4 ± 26.1	89.6 ± 26.5	0.529
Total fat (g/day)	80.7 ± 24.7	82.7 ±23.8	0.249
Saturated fat (g/day)	34.3 ± 12.3	35.4 ± 11.6	0.231

a: All comparison of means for normally-distributed scale variables used Student's t-test for independent samples; Mann-Whitney U tests were used where scale variables were not normally distributed. The Chi-square test of contingencies was used to compare categorical variables between the two populations. P (2-tailed) <0.05 in all cases. b: Standard adolescent criteria were used to classify participants into BMI categories of underweight, normal weight, overweight, and obese [(53; 54)]

Table 2. *GL variables and prevalence of the metabolic syndrome in Raine Study adolescents arranged according to tertiles of mean meal GL*

Variable	Total group n = 516	BOYS Low meal GL[b] n = 88	BOYS Medium meal GL[b] n = 82	BOYS High meal GL[b,c] n = 94	GIRLS Low meal GL[b] n = 95	GIRLS Medium meal GL[b] n = 94	GIRLS High meal GL[b,c] n = 78
Daily nutrient intakes [a]							
Energy (kcal)	2225±579	2533±609	2430±637	2497±541	1892±378	1910±435	2057±398*
Carbohydrate (g)	277±79	249±28	277±20	304±22 *	253±20	278±17	303±25 *
Protein (g)	88.4±26.1	97.8±17.5	91.0±13.9	84.2±14.7 *	93.9±11.2	85.4±11.1	78.4±12.7 *
Total fat (g)	80.7±24.7	89.3±11.2	79.7±9.0	71.2±9.4 *	88.8±8.7	81.9±8.4	73.9±9.8 *
Saturated fat (g)	34.3±12.3	38.7±7.7	34.5±5.8	29.6±6.0 *	36.8±5.4	35.1±5.7	31.2±5.8 *
Daily GI (%)	54.6±4.9	51.3±4.1	55.0±3.5	57.5±4.2 *	51.6±4.9	54.3±3.8	58.1±4.5 *
Daily GL	152±45	126±16	152±10	175±15 *	131±13	150±9	175±16 *
GL variables [a]							
Breakfast GL	30.9±14.9	26.5±11.8	34.6±14.8	35.2±16.5 *	25.9±9.0	30.1±10.5	32.6±12.6 *
Morning Tea GL	15.5±13.2	10.7±11.4	13.5±11.1	17.6±13.7 *	13.5±9.4	15.8±10.6	21.9±15.2 *
Lunch GL	31.6±16.5	29.8±16.8	27.5±14.7	38.9±15.6 *	26.7±11.8	32.2±12.1	33.6±14.4 *
Afternoon tea GL	23.9±18.6	17.2±15.1	25.3±15.9	26.6±20.8 *	20.0±10.6	22.6±11.9	32.1±19.4 *
Dinner GL	44.9±20.1	37.3±14.0	42.6±16.2	53.9±24.0 *	39.1±13.2	44.1±13.9	51.8±18.8 *
Supper GL	10.7±11.5	7.7±8.9	11.8±12.8	11.8±14.7 *	9.0±6.1	11.4±8.4	12.4±9.2 *
Peak score GL	42.3±15.6	38.5±14.5	41.7±15.6	50.7±19.5 *	36.4±10.2	40.3±1.6	46.2±16.0 *
Metabolic Syndrome [d] (n; %)							
Yes	17; 3.5 %	2; 2.4 %	0; 0.0 %	6; 6.8 % *	2; 2.6 %	5; 5.7 %	2; 2.8 %
No	463; 96.5%	81; 97.6%	73; 100 %	82; 93.2 %	74; 97.4 %	83; 94.3 %	70; 97.2 %

Abbreviations:- GI: glycemic index; GL: glycemic load

a: Daily intakes adjusted for energy

b: Arranged into tertiles of mean meal GL, where mean meal GL = Σ(Breakfast GL. + Morning tea GL+ Lunch GL + Afternoon tea GL + Dinner GL + Supper GL)/6

c: Comparison between highest and lowest tertiles; all comparison of means for normally-distributed scale variables used Student's t-test for independent samples; Mann-Whitney U tests were used where scale variables were not normally distributed. The Chi-square test of contingencies was used to compare categorical variables between the two populations. P (2-tailed) <0.05 in all cases, with significance indicated by an asterisk (*)

d: International Diabetes Foundation definition of metabolic syndrome i.e. high waist circumference and any 2 or more of the following: high systolic or diastolic blood pressure; high fasting serum triglycerides; low serum high-density lipoprotein cholesterol, or high plasma glucose concentrations; cut points for categorization of these high and low subgroups vary by gender and age, as published previously [(22)]

Table 3. *Meal, peak score and daily GL[a] variables and risk of metabolic syndrome[b] in Raine Study adolescents (n = 516) in unadjusted and adjusted logistic regression models (with and without BMI)[c]*

Meal GL Variable (BMI excluded/included) [c]	**GIRLS (n=252)**		**BOYS (n=264)**	
	OR (95% CI)	**P**	**OR (95% CI)**	**P**
Breakfast GL				
Unadjusted	1.05 (0.99 – 1.11)	0.07	1.01 (0.97 – 1.06)	0.51
Adjusted, BMI excluded	1.06 (1.00 – 1.12)	0.04	1.04 (0.98 – 1.09)	0.18
Adjusted, BMI included	1.15 (1.04 – 1.27)	< 0.01	0.83 (0.64 – 1.07)	0.15
Lunch GL				
Unadjusted	1.04 (0.99 – 1.09)	0.06	1.03 (0.99 – 1.07)	0.09
Adjusted, BMI excluded	1.04 (0.99 – 1.08)	0.15	1.04 (1.00 – 1.09)	0.06
Adjusted, BMI included	1.04 (0.99 – 1.10)	0.14	1.05 (0.97 – 1.15)	0.24
Dinner GL				
Unadjusted	1.00 (0.95 – 1.04)	0.84	0.99 (0.95 – 1.03)	0.56
Adjusted, BMI excluded	0.98 (0.94 – 1.03)	0.44	0.97 (0.93 – 1.01)	0.14
Adjusted, BMI included	0.97 (0.91 – 1.04)	0.43	0.96 (0.89 – 1.02)	0.19
Peak Score GL				
Unadjusted	1.01 (0.97 – 1.07)	0.58	1.01 (0.97 – 1.05)	0.70
Adjusted, BMI excluded	1.00 (0.94 – 1.05)	0.94	0.99 (0.95 – 1.04)	0.78
Adjusted, BMI included	1.01 (0.95 – 1.08)	0.71	0.95 (0.86 – 1.04)	0.24
Daily GL				
Unadjusted	1.00 (0.98 – 1.02)	0.77	1.01 (0.99 – 1.02)	0.48
Adjusted, BMI excluded	1.00 (0.98 – 1.02)	0.90	1.00 (0.99 – 1.02)	0.64
Adjusted, BMI included	1.01 (0.99 – 1.04)	0.44	1.03 (0.99 – 1.06)	0.19

Abbreviations:- GL: glycemic load; OR: odds ratio; 95% CI: 95% confidence interval

a: All GL variables were adjusted for energy

b: Using the age-specific International Diabetes Foundation definition of metabolic syndrome [(22)]

c: Logistic regression models were adjusted for single parent family, physical activity and energy-adjusted daily protein intake, with BMI excluded or included as an additional confounder

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