HAL
archives-ouvertes.fr

Effect of breakfast glycemic index on metabolic responses during rest and exercise in overweight and non-overweight adolescent girls

Keith Tolfrey, Julia Kirstey Zakrzewski, Emma Stevenson

▶ To cite this version:

Keith Tolfrey, Julia Kirstey Zakrzewski, Emma Stevenson. Effect of breakfast glycemic index on metabolic responses during rest and exercise in overweight and non-overweight adolescent girls. European Journal of Clinical Nutrition, Nature Publishing Group, 2011, 10.1038/ejcn.2011.175 . hal-00685383

HAL Id: hal-00685383
https://hal.archives-ouvertes.fr/hal-00685383

Submitted on 5 Apr 2012

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Effect of breakfast glycemic index on metabolic responses during rest and exercise in overweight and non-overweight adolescent girls

Julia K Zakrzewski (MSc)[1], Emma J Stevenson (PhD)[2] and Keith Tolfrey (PhD)[1]

[1]School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, LE11 3TU, UK

[2]School of Life Sciences, Northumbria University, Newcastle Upon Tyne, NE1 8ST

Corresponding author:
Dr. Keith Tolfrey
School of Sport, Exercise and Health Sciences
Loughborough University
Loughborough
LE11 3TU
UK
k.tolfrey@lboro.ac.uk
Phone +44(0)1509 226355
Fax +44(0)1509 226301

Source of support: The study was funded by Tanita Grant In Aid.

Running title: Effect of glycemic index on metabolism in girls

Abstract

Background/Objectives: The metabolic responses to mixed breakfast meals with different glycemic indexes (GI) and their effects on substrate metabolism during exercise in adolescent girls have not been examined. The interaction with weight status also warrants investigation. The present study investigated the effect of mixed breakfast meals containing high GI (HGI) or low GI (LGI) carbohydrates on metabolic responses and fat oxidation during rest and exercise in overweight (OW) and non-overweight (NO) adolescent girls.

Subjects/Methods: Eight OW and 12 NO adolescent girls consumed an isoenergetic HGI (GI=73) or LGI (GI=44) breakfast 120 min before completing a 30-min treadmill walk at 50% $\dot{V}O_{2\,\text{peak}}$.

Results: Peak blood glucose concentration was higher for HGI compared with LGI in OW (P=0.023), but not NO (P>0.05) girls. Blood glucose total area under the curve (TAUC) was 13% higher in HGI compared with LGI in OW (P=0.006), but only 4% higher in NO (P=0.072) girls. Plasma insulin data were $\log_e$ transformed (lninsulin). Plasma lninsulin concentrations were not different between HGI and LGI (P>0.05). Peak plasma lninsulin concentration (P=0.016) and TAUC (P=0.001) were greater in OW than NO girls. Fat oxidation during postprandial rest and exercise was not different between breakfasts (P>0.05).

Conclusions: The elevated glycemic response in HGI compared with LGI was more pronounced in OW girls, suggesting a reduced ability to cope with the metabolic demands of the HGI, but not LGI, breakfast. Manipulation of breakfast GI did not alter fat oxidation during rest or subsequent moderate intensity exercise in OW and NO adolescent girls.

Keywords: glycemic index, overweight, adolescents, females, metabolism, exercise

46 **Introduction**

47 Several lines of evidence have demonstrated benefits associated with regular breakfast
48 consumption in children and adolescents, relating to academic performance (Lien, 2007),
49 nutrition (Barton *et al*., 2005; Song *et al*., 2006), cardiorespiratory fitness and obesity
50 (Sandercock *et al*., 2010). However, the relationship between breakfast and health benefits
51 may not be due to consumption *per se*, but rather breakfast composition (Cho *et al*., 2003).
52 There are concerns that ready-to-eat cereals commonly eaten by children and adolescents
53 (Song *et al*., 2006) fail to meet national nutrition recommendations (Schwartz *et al*., 2008). In
54 contrast, there has been considerable interest in potential health benefits of breakfasts
55 containing low glycemic index (LGI) carbohydrates (Ludwig *et al*., 1999; Willet *et al*., 2002).

56

57 Manipulation of the GI of a mixed breakfast meal affects postprandial glycemic and
58 insulinemic responses (Ludwig *et al*., 1999; Stevenson *et al*., 2009). Evidence that breakfasts
59 rich in LGI carbohydrates promote satiety in obese adolescents (Ball *et al*., 2003; Ludwig *et*
60 *al*., 1999) suggest that LGI breakfast consumption could have direct implications for pediatric
61 weight management. In adults, the reduced glucose and insulin response to a LGI compared
62 with HGI breakfast has also been shown to result in increased fat oxidation during rest
63 (Stevenson *et al*., 2009) and subsequent exercise (Stevenson *et al*., 2006; 2009; Wee *et al*.,
64 2005). However, breakfast GI does not affect fat oxidation during rest or exercise when
65 comparing a moderate GI and HGI breakfast (Backhouse *et al*., 2007) or when exercise is
66 preceded by two LGI meals rather than breakfast alone (Stevenson *et al*., 2005). A recent
67 study even reported higher fat oxidation during a cycling time trial following a HGI compared
68 with LGI breakfast (Moore *et al*., 2010). Therefore, the influence of carbohydrate GI on
69 postprandial fat oxidation remains unclear.

70

71 Reductions in fat oxidation (Zunquin *et al*., 2009), glucose tolerance (Sinha *et al*., 2002), and
72 insulin sensitivity (Weiss *et al*., 2004) have been shown in overweight and obese young
73 people. Substituting a HGI breakfast for a LGI breakfast may, therefore, be particularly
74 beneficial for overweight individuals through increased glycemic control (Willet *et al*., 2002),
75 fat oxidation (Stevenson *et al*., 2009) and satiety (Ludwig *et al*., 1999). However, the majority
76 of studies investigating the impact of GI on fat oxidation have included endurance trained or
77 recreationally active adults as participants (Stevenson *et al*., 2006; Wu *et al*., 2003) and we
78 are unaware of similar studies including overweight individuals or young people, despite well
79 recognised differences in metabolism between adolescents and adults (Riddell *et al*., 2008).
80 Therefore, we examined the effect of mixed breakfast meals providing HGI or LGI
81 carbohydrates on metabolic responses and substrate utilization during rest and exercise in
82 overweight and non-overweight adolescent girls.
83

Subjects and Methods

Subjects

After gaining approval from the University Ethical Advisory sub-Committee, eight overweight (OW) and twelve non-overweight (NO) girls aged 11-13 y participated in the study. Overweight status was defined using age and sex specific body mass index (BMI) reference points (Cole *et al*., 2000). Written informed consent was obtained from the primary carer and the subjects provided their "willingness to participate". Subjects were screened using a health history questionnaire. Exclusion criteria included: known congenital heart disease, musculoskeletal problems, uncontrolled exercise-induced asthma, diabetes and epilepsy. Stature was measured to the nearest 0.01 m using a stadiometer (Holtain, Holtain Limited, Dyfed, UK) and body mass (BM) to the nearest 0.1 kg using a beam balance scale (Seca Model 888, Hamburg, Germany). Skinfold thickness was measured over the triceps, subscapular and medial calf sites to the nearest 0.2 mm (Harpenden, Baty International, England). Each site was measured three times and median values were used to estimate percentage body fat (Slaughter *et al*., 1988); fat free mass (FFM) was estimated subsequently. Waist circumference was measured midway between the 10th rib and the iliac crest (McCarthy *et al*., 2005) using a Gulick tape measure (Creative Health Products, Plymouth, MI). With the assistance of a primary home-based carer, the girls provided a self-assessment of their physical maturation using pubic hair (Tanner, 1962).

Preliminary measurements

Subjects completed two preliminary tests on a treadmill (RunRace, TechnoGym, Gambettola, Italy) to determine 1) the relation between running speed and oxygen uptake ($\dot{V}O_2$) and 2) peak oxygen uptake ($\dot{V}O_{2\,\mathrm{peak}}$). The speed eliciting 50% of each subject's $\dot{V}O_{2\,\mathrm{peak}}$ was determined subsequently.

109

110 **Test breakfasts**

111 Subjects were provided with a breakfast consisting of either HGI or LGI foods and containing
112 1.5 g CHO·kg BM^{-1} (Table 1). The breakfasts were matched for energy, macronutrients and
113 fluid, but the LGI breakfast contained more fibre. The GI values for individual foods were
114 taken from the International Table of GI and Glycemic Load Values (Atkinson *et al*., 2008)
115 and breakfast GI was calculated from the weighted means of the GI values for the component
116 foods (Wolever and Jenkins, 1986). The calculated GI for the breakfasts were 73 (HGI) and
117 44 (LGI).

118

119 **Experimental protocol**

120 Subjects completed two experimental trials (HGI and LGI) in a counter-balanced order. Trials
121 were conducted a maximum 48 h apart for the girls who had irregular menstruation to
122 minimise the potential influence of menstrual cycle phase on within-subject comparisons
123 (Oosthuyse and Bosch, 2010). Due to the sporadic nature of the menstrual cycle in young
124 adolescent girls, other studies have not accounted for menstrual cycle phase (Timmons *et al*.,
125 2007). The girls consumed the same diet and minimised physical activity in the 24 h prior to
126 experimental trials.

127

128 Subjects reported to the laboratory at 08:00 following a 12 h fast. Following fasted measures,
129 the girls consumed a HGI or LGI breakfast (Table 1) within 15 min. Blood, expired air
130 samples and subjective ratings of hunger were collected at regular intervals during the 120
131 min postprandial period. Subsequently, the girls completed a 30 min treadmill walk at 50%
132 $\dot{V}O_{2\,\mathrm{peak}}$. Water was available ad libitum throughout the first trial and the girls drank the same
133 volume during the second trial.

134

135 **Blood sampling and analysis**

136 Capillary blood samples were obtained from a pre-warmed hand by finger prick (Unistik 2,
137 Owen Mumford, UK) into collection tubes (Microvette CB300 EDTA, Sarstedt Ltd, UK).
138 Capillary rather than venous blood sampling is preferred for reliable GI testing (Wolever *et*
139 *al*., 2003). Duplicate 25 µl aliquots of whole blood were deproteinised in 250 µl of ice cooled
140 perchloric acid (PCA; 2.5%), centrifuged for 4 min at 2415 x g and stored at -20°C for blood
141 glucose analysis. The remaining whole blood was centrifuged for 4 min at 2415 x g. Plasma
142 was extracted and stored at -20°C for insulin analysis.

143

144 Blood glucose concentration was determined spectrophotometrically using the glucose
145 oxidase method (GOD-PAP, Randox, Ireland). Plasma insulin was measured using an
146 enzyme-linked immunosorbent assay (ELISA, Mercodia. Sweden). Total 2-h area under the
147 curve (TAUC) for blood glucose and plasma insulin was calculated using the trapezium rule
148 (Wolever and Jenkins, 1986). Homeostasis model assessment-insulin resistance (HOMA-IR)
149 was calculated (Mathews *et al*., 1985). The intra-assay coefficient of variation was 2.4% for
150 blood glucose and 6.3% for plasma insulin.

151

152 **Expired air and indirect calorimetry**

153 Expired air was sampled continuously during exercise tests and for 10 min periods for resting
154 measurements. Breath-by-breath data were displayed online using a portable metabolic cart
155 (K4 b^2, Cosmed, Rome, Italy) and interpolated into 1 sec intervals for subsequent analyses.
156 Calibration procedures were carried out prior to each experimental test, as described
157 previously (Zakrzewski and Tolfrey, 2011). Fat oxidation rates were calculated using
158 stoichiometric equations, with the assumption that the urinary nitrogen excretion rate was

159 negligible and a physiological stready-state had been attained (Frayn, 1983). The total area
160 under the fat oxidation versus time curve (TAUC) for the 120 min rest period was calculated
161 using the trapezium rule and included in subsequent analyses.

162

163 **Perceived hunger**

164 Perceptions of hunger, satisfaction, fullness, prospective food consumption and breakfast
165 palatability were assessed using 100 mm visual analogue scales.

166

167 **Statistical analyses**

168 Statistical analyses were completed using SPSS (v16 SPSS Inc, Chicago, IL, USA). The
169 insulin data were transformed using a natural logarithm (lninsulin) to normalise them and
170 homogenize the variances between the groups. Breakfast by time (2 x 7) repeated measures
171 ANOVA were used to examine differences between HGI and LGI over time for glucose and
172 lninsulin; these were conducted separately for OW and NO girls. Breakfast by group (2 x 2)
173 mixed measures ANOVA with breakfast as the repeated factor were used to compare the two
174 groups directly for glucose and lninsulin TAUC. For resting and exercise fat oxidation,
175 breakfast by group (2 x 2) mixed measures ANCOVA with estimated FFM as the covariate
176 were used. Homogeneity of regression slopes was confirmed prior to each ANCOVA. Paired
177 sample t-tests with Bonferroni correction were used to compare glucose and lninsulin
178 concentrations at different time points and to follow-up significant two-way interactions.
179 Values are expressed as mean(SD), unless stated otherwise, and effect sizes (ES) were
180 calculated. Statistical significance was accepted at $P \leq 0.05$.

181

182 **Results**

183 **Subject characteristics**

184 Complete data for 8 OW and 12 NO girls were available for analyses. Body fat, BMI, body
185 mass, fat free mass, waist circumference and hip circumference were higher in OW compared
186 with NO girls (P≤0.05), whereas $\dot{V}O_{2\,peak}$ (mL·kg^{-1}·min^{-1}) was higher in NO girls (P≤0.0005)
187 (Table 2).

188

189 **Blood glucose concentration**

190 Following breakfast, blood glucose concentrations increased and peaked at a median
191 (interquartile range) time of 30(0) min for all trials, except in the OW HGI trial where it
192 peaked at 45 min in 4 girls (median 37.5(15) min) (Figure 1). Breakfast by time interactions
193 were found for OW (P≤0.001) and NO (P=0.001); concentrations were higher in HGI
194 compared with LGI at 45 (P=0.004) and 60 (P≤0.001) min in OW girls and at 90 (P=0.006)
195 and 120 (P=0.001) min in NO girls.

196

197 There were no differences in fasting or postprandial glucose between OW and NO girls at any
198 time points (P>0.05). However, breakfast by group interactions for peak blood glucose
199 (P=0.053, ES: 0.44) and TAUC (P=0.026, ES: 0.50) were found. Peak blood glucose was
200 higher for HGI compared with LGI in OW (6.1 vs. 5.5 mmol·L^{-1}; P=0.023, ES: 0.74), but
201 similar between breakfasts in NO (5.8 vs. 5.9 mmol·L^{-1}; P=0.741). There were no between
202 group differences in peak blood glucose after the HGI (P=0.404) or LGI (P=0.122) breakfasts.
203 Blood glucose TAUC was 13% higher in HGI compared with LGI in OW (P=0.006, ES: 0.82)
204 but only 4% higher in NO (P=0.072, ES: 0.51). Moreover, HGI TAUC was 9% higher in OW
205 compared with NO (P=0.070, ES: 0.41), but LGI TAUC was similar between the groups

206 (P=0.831, ES: 0.05). Similarly, the pattern of blood glucose over time differed between the
207 OW and NO girls for HGI (P=0.047, ES: 0.24), but not LGI (P=0.119).
208

209 **Plasma insulin concentration**

210 Following breakfast, plasma lninsulin concentration increased and peaked at a median
211 (interquartile range) time of 30(15) and 30(0) min for HGI and LGI in NO and 30(15) min in
212 the OW LGI trial, but 45(15) min in the OW HGI trial (Figure 2). Breakfast by time
213 interactions were found for OW (P=0.012) and NO (P≤0.005); however, pairwise
214 comparisons only revealed a single significant difference in NO girls at 120 min (P=0.001).
215 Furthermore, neither the main effect for breakfast nor the breakfast by group interaction for
216 lninsulin TAUC were significant (P>0.05).
217

218 Although a strong statistical trend in fasting lninsulin between the OW and NO girls was
219 found (P=0.054, ES: 0.45), pairwise analyses were not significant (P>0.025). Peak lninsulin
220 (P=0.016) and TAUC (P=0.001) were higher in OW than NO. Whilst Bonferroni follow-up
221 indicated that peak lninsulin was only significantly different following the HGI breakfast
222 (P≤0.025), it was clear that both breakfasts led to significant differences in TAUC (P≤0.025)
223 between OW and NO. HOMA-IR was higher in OW compared with NO (3.2 vs. 2.9,
224 P=0.054).
225

226 **Fat oxidation**

227 The resting and exercise fat oxidation results by group and breakfast are in Table 3. During
228 both postprandial rest and subsequent exercise, absolute and ANCOVA FFM adjusted fat
229 oxidation were not different between HGI and LGI breakfast conditions in either group of
230 girls (P>0.05).

During the postprandial rest period, absolute fat oxidation was higher in the OW compared with NO girls in HGI (P=0.004, ES: 0.61) and LGI (P=0.005, ES: 0.60). However, once between group differences in FFM were accounted for, resting fat oxidation was similar in the two groups of girls (P>0.05). During subsequent exercise, absolute and ANCOVA FFM adjusted total fat oxidation were not different when comparing the OW and NO girls for the HGI and LGI conditions (P>0.05).

Hunger

Perceptions of hunger, satisfaction, fullness, prospective food consumption and breakfast palatability were similar between trials (P>0.05).

Discussion

The main finding of the present study was that the higher glycemic response in HGI compared with LGI was more pronounced in OW than NO girls, possibly reflecting a reduced ability to cope with the metabolic demands of a HGI breakfast in OW girls. Breakfast GI did not affect fat oxidation during the 120 min postprandial rest period or subsequent moderate intensity exercise in OW and NO adolescent girls.

A novel finding was that the higher glycemic response in HGI compared with LGI was exaggerated in the OW girls, mainly due to the delayed decline in blood glucose following the postprandial peak. This may indicate a delayed blood glucose uptake up to 60 min following HGI breakfast consumption in OW girls. Previous work has reported higher glycemic responses to HGI compared with LGI breakfasts in obese adolescents, but these studies did not include non-overweight participants for direct comparison (Ball *et al*., 2003; Ludwig *et al*., 1999). Furthermore, higher and more sustained postprandial glucose responses have been reported in obese compared with non-obese children (Sinha *et al*., 2002). However, we were unable to locate another study that has investigated whether these differences between OW and NO young people are dependent on the GI of the consumed carbohydrate. Perälä *et al*. (2011) recently reported that the higher glycemic response to a HGI compared with LGI meal was similar in OW and NO 62 to 72 year olds. However, the meals only contained 50 g CHO and were not scaled to body mass. It is, therefore, difficult to compare our findings directly with those of Perälä *et al*. (2011) due to differences in study design and participants.

It is possible that the combination of readily absorbed glucose from the HGI (but not LGI) breakfast and higher insulin resistance (HOMA-IR) in the OW girls contributed to the larger glycemic response in the OW HGI trial. Furthermore, plasma insulin peaked earlier and

268 returned towards baseline values for LGI, but remained elevated for HGI in the OW girls. The
269 earlier peak in plasma insulin may have contributed to the more rapid decline in blood glucose
270 in LGI, as suggested previously in adults (Schenk *et al*., 2003).
271
272 Collectively, these findings indicate that LGI breakfasts may be beneficial for blood glucose
273 control in OW girls. Furthermore, the elevated glycemic response in the OW HGI trial may
274 also increase voluntary food intake later in the day (Ludwig *et al*., 1999). Encouragingly,
275 adults with higher postprandial glycemic responses have a greater postprandial reduction
276 when changing from HGI to LGI foods (Høstmark, 2007) and lowering breakfast GI for 21
277 days reduced fasting glucose and satiety in obese adults (Pal *et al*., 2008). Moreover, the
278 similar palatability between breakfasts in our study indicates LGI breakfast promotion for
279 OW girls may be feasible. As the higher fibre content in the LGI breakfast may have
280 contributed to the lower glycemic response to this breakfast (Pi-Sunyer, 2002), it may be
281 more appropriate to recommend LGI high-fibre breakfasts (rather than LGI breakfasts) for
282 OW girls. This is feasible since LGI foods typically contain more fibre than HGI foods.
283 Nevertheless, confirmation of these results in larger groups of young people, including boys,
284 is required.
285
286 Breakfast GI did not affect postprandial fat oxidation during rest or exercise in either group of
287 girls. However, it is noteworthy that LGI resulted in 12% higher exercise fat oxidation
288 (ANCOVA adjusted for FFM) in both groups on average, a finding that may have meaningful
289 health-related implications since higher rates of fat oxidation may ameliorate the development
290 of obesity and type 2 diabetes (Holloway *et al*., 2009). During exercise, studies in adults have
291 reported higher fat oxidation following LGI breakfasts (Stevenson *et al*., 2009; Wee *et al*.,
292 2005), no effect of GI (Backhouse *et al*., 2007; Stevenson *et al*., 2005) or even higher fat

293 oxidation following a HGI breakfast (Moore *et al*., 2010). During rest, most have reported no
294 effect of breakfast GI on fat oxidation (Díaz *et al*., 2005; Stevenson *et al*., 2005; Wee *et al*.,
295 2005), although higher fat oxidation following LGI breakfasts has been shown (Stevenson *et*
296 *al*., 2009). Discrepancies between studies may be due to differences in breakfast size or
297 composition, exercise mode, intensity and duration, postprandial time period and subject
298 characteristics. However, higher exercise fat oxidation following LGI breakfasts has been
299 reported 45 min to 3 h (Sparks *et al*., 1998; Stevenson *et al*., 2009) following breakfasts
300 containing 1 to 2.5 g CHO·kg BM^{-1} during exercise lasting 60 or 30 min at 50-71% $\dot{V}O_{2\,\mathrm{peak}}$
301 (Stevenson *et al*., 2009; Wee *et al*., 2005). It is, therefore, difficult to ascertain which factors
302 contribute specifically to the higher fat oxidation following LGI breakfasts in some adult
303 studies. It is possible that the 1.5 g CHO·kg BM^{-1} breakfast, 120 min postprandial period and
304 30 min exercise duration at 50% $\dot{V}O_{2\,\mathrm{peak}}$ used in the present study was a sub-optimal
305 combination to induce differences in fat oxidation between HGI and LGI. Furthermore,
306 differences in fat metabolism between adolescents and adults (Riddell *et al*., 2008) may have
307 resulted in discrepancies between our study and some of the adult literature. Therefore, further
308 examination of the relationship between breakfast GI and fat oxidation in young people is
309 warranted.
310
311 The similar insulin response between HGI and LGI in our study may have underpinned the
312 similarity in fat oxidation (Horowtiz *et al*., 1997). Furthermore, fructose has a lower GI than
313 glucose, but results in higher blood lactate concentrations (Moore *et al*., 2000). It is possible
314 that higher lactate concentrations compromised fat oxidation following the LGI breakfast
315 through direct inhibition of adipose tissue free fatty acid release (Boyd *et al*., 1974). Indeed,
316 resting fat oxidation was lower after high fructose compared with high glucose meals in obese
317 adults, despite lower glycemic and insulinemic responses to the high fructose meal (Tittelbach

318 *et al*., 2000). Although we did not measure blood lactate, higher postprandial lactate
319 concentrations have been reported following LGI compared with HGI breakfasts (Stevenson
320 *et al*., 2006). In addition, blood lactate can affect the validity of indirect calorimetry for fat
321 oxidation estimations (Rowlands, 2005). However, it is unlikely that this was a factor in the
322 present study since the girls exercised at a moderate intensity (50% $\dot{V}O_{2\,\mathrm{peak}}$) and additional
323 steps were taken to increase the validity of indirect calorimetry (e.g. removing individual $\dot{V}O_2$
324 and $\dot{V}CO_2$ values ≥ 3 SD's from the mean and verifying a steady state in $\dot{V}O_2$ and $\dot{V}CO_2$).
325
326 Although we found no difference in perceptions of hunger between breakfasts, the 120 min
327 postprandial period may have been too short for differences to emerge (Anderson and
328 Woodend, 2003; Stevenson *et al*., 2009). Furthermore, prolongation of satiety based on time
329 to request food was found >3 h following LGI compared with HGI foods in obese
330 adolescents, despite no difference using hunger scales (Ball *et al*., 2003).
331
332 In summary, the higher glycemic response in HGI compared with LGI was more pronounced
333 in OW girls, suggesting a reduced ability to cope with the metabolic demands of a HGI
334 breakfast in this population. This provides further evidence for potential health benefits of
335 LGI foods and suggests LGI breakfast promotion for OW girls is warranted. Breakfast GI did
336 not affect fat oxidation during rest or exercise in OW and NO adolescent girls, although
337 further examination of this relationship young people is required.
338

339 **Acknowledgments**

340 We thank Tanita Grant In Aid for funding the research, all of the participants and their parents
341 for their commitment to our study, and Professor Clyde Williams for his advice. We also
342 thank Mr Graham Bett, Mr Ian Smith, and Mrs Sophie Diaper from Woodbrook Vale High
343 School, Loughborough for their support with the study.

344

345 **Conflict of interest**

346 The authors declare no conflict of interest.

347

348 **References**

349 1. Anderson GH, Woodend D (2003). Effect of glycemic carbohydrates on short term satiety
350 and food intake. *Nutr Rev* **61**, S17–S26.
351 2. Atkinson FS, Foster-Powell K, Brand-Miller JC (2008). International tables of glycemic
352 index and glycemic load values: 2008. *Diabetes Care* **31**, 2281-2283.
353 3. Backhouse SH, Williams C, Stevenson E, Nute M (2007). Effects of the glycemic index
354 of breakfast on metabolic responses to brisk walking in females. *Eur J Clin Nutr* **61**, 590-
355 596.
356 4. Ball SD, Keller KR, Moyer-Mileur LJ, Ding YW, Donaldson D, Jackson WD (2003).
357 Prolongation of satiety after low versus moderately high glycemic index meals in obese
358 adolescents. *Pediatrics* **111**, 488-494.
359 5. Barton BA, Eldridge AL, Thompson D, Affenito SG, Striegel-Moore RH, Franko DL *et*
360 *al*. (2005). The relationship of breakfast and cereal consumption to nutrient intake and
361 body mass index: the National Heart, Lung, and Blood Institute Growth and Health Study.
362 *J Am Diet Assoc* **105**, 1383-1389.
363 6. Boyd AE, Giamber SR, Mager M, Lebovitz HE (1974). Lactate inhibition of lipolysis in
364 exercising man. *Metabolism* **23**, 531-542.
365 7. Cho S, Dietrich M, Brown CJ, Clark CA, Block G (2003). The effect of breakfast type on
366 total daily energy intake and body mass index: results from the Third National Health and
367 Nutrition Examination Survey (NHANES III). *J Am Coll Nutr* **22**, 296-302.
368 8. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH (2000). Establishing a standard definition for
369 child overweight and obesity worldwide: international survey. *BMJ* **320**, 1240-1243.
370 9. Díaz EO, Galgani JE, Aguirre CA, Atwater IJ, Burrows R (2005). Effect of glycemic
371 index on whole-body substrate oxidation in obese women. *Int J Obes (Lond)* **29**, 108-114.

372 10. Frayn KN (1983). Calculation of substrate oxidation rates in vivo from gaseous exchange.
373 *J Appl Physiol* 55, 628-634.
374 11. Holloway GP, Bonen A, Spriet LL (2009). Regulation of skeletal muscle mitochondrial
375 fatty acid metabolism in lean and obese individuals. *Am J Clin Nutr* **89**, S455-S462.
376 12. Horowitz JF, Mora-Rodriguez R, Byerley LO, Coyle EF (1997). Lipolytic suppression
377 following carbohydrate ingestion limits fat oxidation during exercise. *Am J Physiol* **273**,
378 E768-E775.
379 13. Høstmark AT (2007). Variations in the glycemic response to carbohydrates: do high
380 responders have a special benefit of using low glycemic foods? *Open Nutr J* **1**, 1-4.
381 14. Lien L (2007). Is breakfast consumption related to mental distress and academic
382 performance in adolescents? *Public Health Nutr* **10**, 422-428.
383 15. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB (1999). High
384 glycemic index foods, overeating, and obesity. *Pediatrics* **103**, E26.
385 16. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC (1985).
386 Homeostasis model assessment: insulin resistance and beta-cell function from fasting
387 plasma glucose and insulin concentrations in man. *Diabetologia* **28**, 412-419.
388 17. McCarthy HD, Jarrett KV, Emmett PM, Rogers I (2005). Trends in waist circumferences
389 in young British children: a comparative study. *Int J Obes (Lond)* **29**, 157-162.
390 18. Moore MC, Cherrington AD, Mann SL, Davis SN (2000). Acute fructose administration
391 decreases the glycemic response to an oral glucose tolerance test in normal adults. *J Clin*
392 *Endocrinol Metab* **85**, 4515–4519.
393 19. Moore LJ, Midgley AW, Thurlow S, Thomas G, Mc Naughton LR (2010). Effect of the
394 glycaemic index of a pre-exercise meal on metabolism and cycling time trial performance.
395 *J Sci Med Sport* **13**, 182-188.

396 20. Oosthuyse T, Bosch AN (2010). The effect of the menstrual cycle on exercise
397 metabolism: implications for exercise performance in eumenorrhoeic women. *Sports Med*
398 **40**, 207-227.
399 21. Pal S, Lim S, Egger G (2008). The effect of a low glycaemic index breakfast on blood
400 glucose, insulin, lipid profiles, blood pressure, body weight, body composition and satiety
401 in obese and overweight individuals: a pilot study. *J Am Coll Nutr* **27**, 387-393.
402 22. Perälä MM, Hätönen KA, Virtamo J, Eriksson JG, Sinkko HK, Sundvall J *et al*. (2011).
403 Impact of overweight and glucose tolerance on postprandial responses to high- and low-
404 glycaemic index meals. *Br J Nutr* **25**, 1-8.
405 23. Pi-Sunyer FX (2002). Glycemic index and disease. *Am J Clin Nutr* **76**, S290-S298.
406 24. Riddell MC, Jamnik VK, Iscoe KE, Timmons BW, Gledhill N (2008). Fat oxidation rate
407 and the exercise intensity that elicits maximal fat oxidation decreases with pubertal status
408 in young male subjects. *J Appl Physiol* **105**, 742-748.
409 25. Rowlands DS (2005). Model for the behaviour of compartmental CO_2 stores during
410 incremental exercise. *Eur J Appl Physiol* **93**, 555-568.
411 26. Sandercock GR, Voss C, Dye L (2010). Associations between habitual school-day
412 breakfast consumption, body mass index, physical activity and cardiorespiratory fitness in
413 English schoolchildren. *Eur J Clin Nut*r **64**, 1086-1092.
414 27. Schwartz MB, Vartanian LR, Wharton CM, Brownell KD (2008). Examining the
415 nutritional quality of breakfast cereals marketed to children. *J Am Diet Assoc* **108**, 702-
416 705.
417 28. Schenk S, Davidson CJ, Zderic TW, Byerley LO, Coyle EF (2003). Different glycemic
418 indexes of breakfast cereals are not due to glucose entry into blood but to glucose removal
419 by tissue. *Am J Clin Nutr* **78**, 742-748.

420 29. Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K *et al*. (2002).
421 Prevalence of impaired glucose tolerance among children and adolescents with marked
422 obesity. *N Engl J Med* **346**, 802–810.
423 30. Slaughter MH, Lohman TG, Boileau RA, Horswill CA, Stillman RJ, Van Loan MD *et al*.
424 (1988). Skinfold equations for estimation of body fatness in children and youth. *Hum Biol*
425 **60**, 709-723.
426 31. Song WO, Chun OK, Kerver J, Cho S, Chung CE, Chung SJ (2006). Ready-to-eat
427 breakfast cereal consumption enhances milk and calcium intake in the US population. *J*
428 *Am Diet Assoc* **106**, 1783-1789.
429 32. Sparks MJ, Selig SS, Febbraio MA (1998). Pre-exercise carbohydrate ingestion: effect of
430 the glycemic index on endurance exercise performance. *Med Sci Sports Exerc* **30**, 844-
431 849.
432 33. Stevenson EJ, Williams C, Nute ML (2005). The influence of the glycaemic index of
433 breakfast and lunch on substrate utilisation during the postprandial periods and subsequent
434 exercise. *Br J Nutr* **93**, 885-893.
435 34. Stevenson EJ, Williams C, Mash LE, Phillips B, Nute ML (2006). Influence of high-
436 carbohydrate mixed meals with different glycemic indexes on substrate utilization during
437 subsequent exercise in women. *Am J Clin Nutr* **84**, 354-360.
438 35. Stevenson EJ, Astbury NM, Simpson EJ, Taylor MA Macdonald IA (2009). Fat oxidation
439 during exercise and satiety during recovery are increased following a low-glycemic index
440 breakfast in sedentary women. *J Nutr* **139**, 890-897.
441 36. Tanner JM (1962). Growth at adolescents. Oxford, UK: Blackwell Scientific.
442 37. Timmons BW, Bar-Or O, Riddell MC (2007). Energy substrate utilization during
443 prolonged exercise with and without carbohydrate intake in preadolescent and adolescent
444 girls. *J Appl Physiol* **103**, 995-1000.

445 38. Tittelbach TJ, Mattes RD, Gretebeck RJ (2000). Post-exercise substrate utilization after a
446 high glucose vs. high fructose meal during negative energy balance in the obese. *Obes Res*
447 **8**, 496-505.
448 39. Wee SL, Williams C, Tsintzas K, Boobis L (2005). Ingestion of a high-glycemic index
449 meal increases muscle glycogen storage at rest but augments its utilization during
450 subsequent exercise. *J Appl Physiol* **99**, 707-714.
451 40. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW *et al*. (2004).
452 Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* **350**,
453 2362-2374.
454 41. Willett W, Manson J, Liu S (2002). Glycemic index, glycemic load, and risk of type 2
455 diabetes. *Am J Clin Nutr* **76**, S274-S280.
456 42. Wolever TM, Jenkins DJ (1986). The use of the glycemic index in predicting the blood
457 glucose response to mixed meals. *Am J Clin Nutr* **43**, 167-172.
458 43. Wolever TM, Vorster HH, Björck I, Brand-Miller J, Brighenti F, Mann JI *et al*. (2003).
459 Determination of the glycaemic index of foods: interlaboratory study. *Eur J Clin Nutr* **57**,
460 475-482.
461 44. Wu CL, Nicholas C, Williams C, Took A, Hardy L (2003). The influence of high-
462 carbohydrate meals with different glycaemic indices on substrate utilisation during
463 subsequent exercise. *Br J Nutr* **90**, 1049-1056.
464 45. Zakrzewski J, Tolfrey K (2011). Exercise protocols to estimate Fatmax and maximal fat
465 oxidation in children. *Ped Ex Sci* **23**, 122-135.
466 46. Zunquin G, Theunynck D, Sesboüé B, Arhan P, Bouglé D (2009). Comparison of fat
467 oxidation during exercise in lean and obese pubertal boys: clinical implications. *Br J*
468 *Sports Med* **43**, 869-870.
469

470 **Figure legends**

471 **Figure 1** Blood glucose concentration response to the HGI and LGI breakfasts for the
472 overweight (OW; figure 1a) and non-overweight (NO; figure 1b) girls.
473 Breakfast was consumed between 0 and 15 min. Breakfast x time interactions
474 were significant for OW ($P \leq 0.001$) and NO ($P=0.001$) girls.
475 [a] significant difference between HGI and LGI (Bonferroni correction;
476 significance was $P \leq 0.025$).

477 **Figure 2** Plasma insulin concentration response to the HGI and LGI breakfasts for the
478 overweight (OW) and non-overweight (NO) girls. Breakfast was consumed
479 between 0 and 15 min. Breakfast x time interactions were significant for OW
480 ($P=0.012$) and NO ($P \leq 0.005$) girls.
481 [a] significant difference between HGI and LGI in NO girls (Bonferroni
482 correction; significance was $P \leq 0.025$).

483

484

485 **Table 1** Composition of test breakfasts for a 45 kg girl

Breakfasts	Description	Macronutrient content
HGI	45 g Cornflakes[a] + 135 g skimmed milk, 44 g white bread, 4 g jam, 5 g margarine[a],124 g water (total weight = 356 g)	1498 kJ energy, 68 g CHO, 4.2 g fat, 11.6 g protein, 2.7 g fibre GI = 73[b]
LGI	41 g muesli[a] + 90 g skimmed milk, 107 g apple, 169 g apple juice, 87 g yoghurt (total weight = 493 g[c])	1498 kJ energy, 67 g CHO, 4.3 g fat, 11.7 g protein, 5.3 g fibre GI = 44[b]

486 CHO - carbohydrate
487 [a] Cornflakes, Kellogg's; Flora original margarine spread, Unilever; Alpen no added sugar,
488 Weetabix Ltd.
489 [b] Calculated according to Wolever and Jenkins (1986) with GI values taken from Atkinson et
490 al. (2008).
491 [c] Total weight of LGI breakfast higher than HGI breakfast ($P \leq 0.005$).

492

493 **Table 2** Subject characteristics

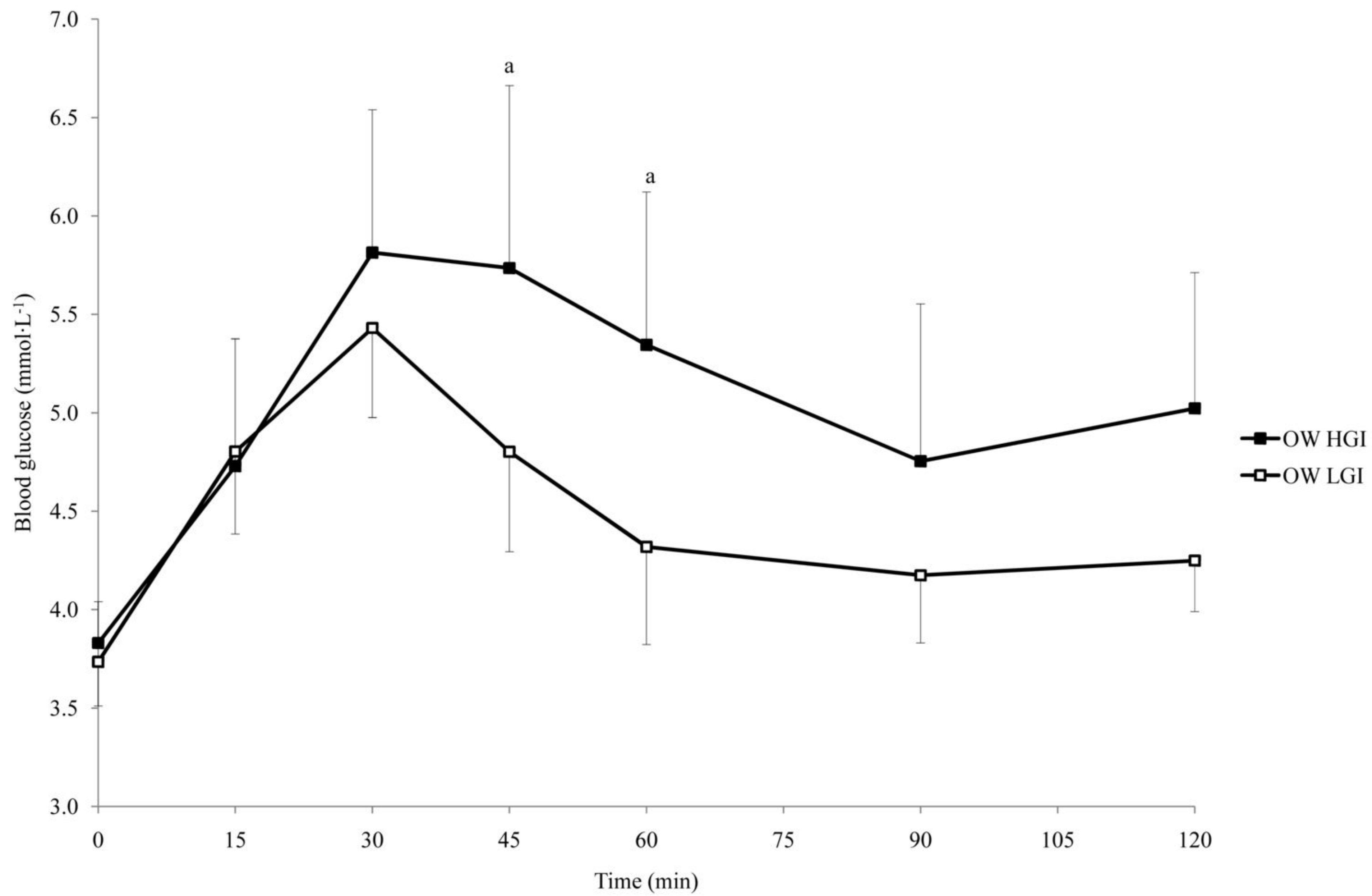
	OW n=8	NO n=12
Age (y)	12.6(0.5)	13.1(0.4)
Body mass (kg) [a]	70.9(19.4)	45.5(8.4)
Stature (m)	1.61(0.08)	1.56(0.09)
Body mass index ($kg·m^{-2}$) [a]	27.0(5.8)	18.5(2.0)
Body fat (%) [a]	35.7(6.0)	19.2(3.9)
Fat free mass (kg) [a]	45(9)	37(6)
Waist circumference (cm) [a]	84.6(13.5)	63.0(4.7)
Hip circumference (cm) [a]	99.5(12.3)	82.1(8.2)
Tanner Stage (pubic hair) [b]	3(1)	3(1)
Treadmill $\dot{V}O_{2\,peak}$ ($mL·kg^{-1}·min^{-1}$) [a]	32(7)	45(6)

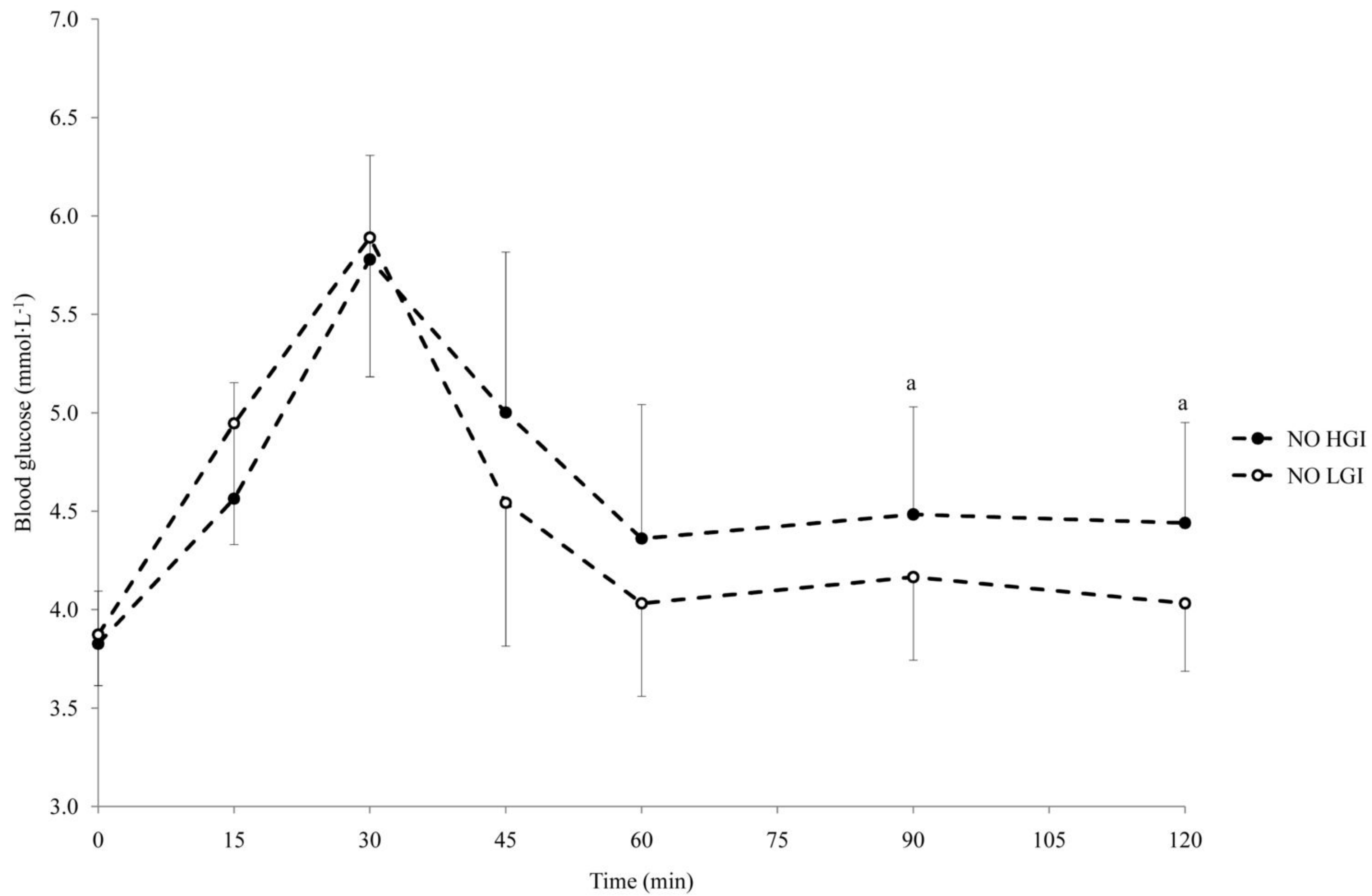
494 Tanner stage is an estimation of secondary sexual characteristics (Tanner, 1962).
495 $\dot{V}O_{2\,peak}$ – peak oxygen uptake.
496 [a] significantly different between OW and NO.
497 [b] median (interquartile range).
498
499

Table 3 Resting and exercise fat oxidation (area under curve over time): comparisons between breakfasts and groups

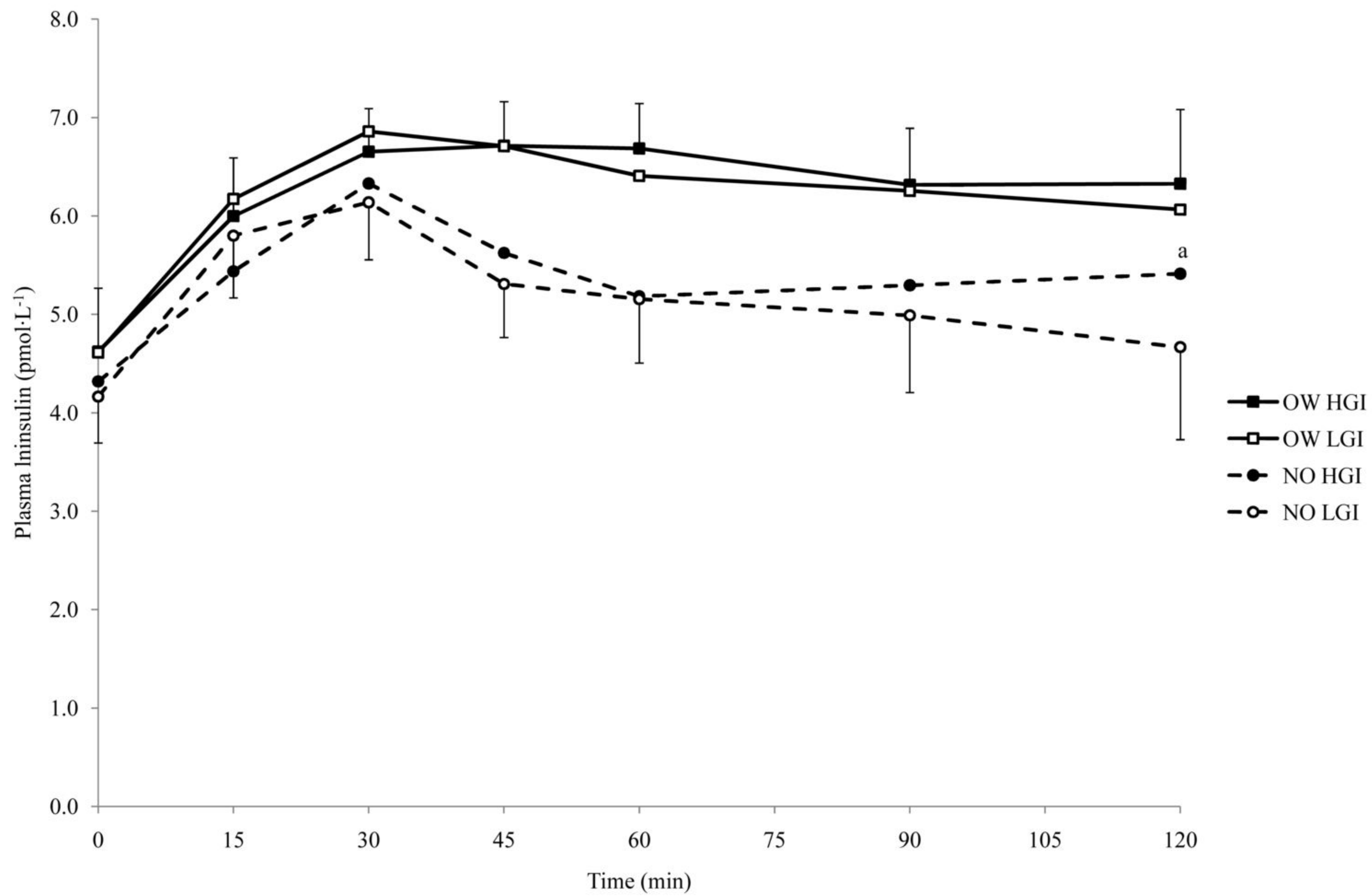
Group	Breakfast	Rest ($g \cdot 120\ min^{-1}$)		Exercise ($g \cdot 30\ min^{-1}$)	
		Absolute	FFM[1]	Absolute	FFM[1]
OW	HGI	0.25(0.10)[a]	0.20(0.05)	5.05(1.53)	4.49(1.71)
	LGI	0.26(0.13)[b]	0.18(0.07)	6.03(3.49)	5.08(2.38)
NO	HGI	0.15(0.04)	0.16(0.09)	4.76(1.83)	5.14(1.66)
	LGI	0.13(0.05)	0.14(0.07)	5.23(1.63)	5.86(2.31)

All values are mean(SD)
OW – overweight girls; NO – non-overweight girls; HGI – high glycemic index; LGI – low glycemic index
[1]ANCOVA adjusted values with FFM (fat free mass) as the covariate
[a]Significantly higher in OW compared with NO girls after consuming the HGI breakfast (P=0.004)
[b]Significantly higher in OW compared with NO girls after consuming the LGI breakfast (P=0.005)


Blood glucose (mmol·L⁻¹)
7.0
6.5
6.0
5.5
5.0
4.5
4.0
3.5
3.0
a
a
OW HGI
OW LGI
0
15
30
45
60
75
90
105
120
Time (min)



7.0
6.5
6.0
5.5
5.0
4.5
4.0
3.5
3.0
Blood glucose (mmol·L-1)
0
15
30
45
60
75
90
105
120
Time (min)
a
a
NO HGI
NO LGI



Plasma Ininsulin (pmol·L-1)
8.0
7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
0
15
30
45
60
75
90
105
120
Time (min)
a
OW HGI
OW LGI
NO HGI
NO LGI