Available online at www.sciencedirect.com

SciVerse ScienceDirect

Journal of Sport and Health Science 1 (2012) 149–159

www.jshs.org.cn

Review

Breakfast, glycaemic index and health in young people

Keith Tolfrey [a,*], Julia K. Zakrzewski [b]

[a] School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough LE11 3TU, UK
[b] Department for Health, University of Bath, Bath BA2 7AY, UK

Received 16 June 2012; revised 20 August 2012; accepted 31 August 2012

Abstract

The often cited phrase "breakfast is the most important meal of the day" may have been largely anecdotal and lacking empirical evidence originally, particularly where children and adolescents (young people) are concerned. However, there is now a large body of evidence demonstrating that regular breakfast consumption is associated with a variety of nutritional and lifestyle-related health outcomes in large diverse samples of young people, which may prevent weight gain, nutrient deficiency and reduce risk factors for chronic disease. This evidence has been reviewed previously, but the link between breakfast composition and health has received less attention. There is emerging evidence in young people that suggests certain breakfasts are particularly beneficial for health, with much of this evidence focusing on ready-to-eat cereals and breakfast glycaemic index (GI). Substituting a high GI (HGI) breakfast for a low GI (LGI) breakfast may be particularly beneficial for overweight young people through increased glycaemic control and satiety. Thus, the purpose of this paper is to extend previous reviews on breakfast consumption and health to provide a greater understanding of the role of breakfast composition, particularly breakfast GI. Unlike the evidence on breakfast consumption, which has often been based on large cross-sectional studies, the evidence on breakfast GI is based primarily on controlled experimental studies, often with relatively small samples. At times, it was necessary to refer to the adult-based literature in this review to support findings from young people or to highlight areas that are particularly lacking in empirical evidence in this population. Since breakfast consumption has declined in young people and also decreases from childhood to adolescence, strategies to promote regular consumption of a healthy breakfast in young people are warranted. Future research in young people should place greater emphasis on breakfast composition, consider the mechanisms controlling relationships between breakfast consumption and health, and investigate the benefits of habitual consumption of LGI compared with HGI breakfasts.

Keywords: Composition; Glucose; Glycaemic index; Insulin; Obese; Overweight

1. Introduction

A definition of breakfast for research has been proposed as "the first meal of the day, eaten before or at the start of daily activities, within 2 h of waking, typically no later than 10:00 in the morning, and of an energy level between 20% and 35% of total daily energy needs".[1] In the past several years, research has shown that regular breakfast consumption has important implications for improving health,[2,3] as well as improving cognitive performance and reducing mental distress[4] in young people. Despite these reported advantages, 10%–35% of young people in many westernised countries regularly skip breakfast;[2,5–10] these numbers are higher in girls compared with boys and increase from childhood to adolescence.[11] It is important to note that this broad range in the numbers of breakfast skippers reported may be attributed to several factors, particularly between-study differences in the method

* Corresponding author.
E-mail address: k.tolfrey@lboro.ac.uk (K. Tolfrey)
Peer review under responsibility of Shanghai University of Sport

2095-2546/$ - see front matter
http://dx.doi.org/10.1016/j.jshs.2012.09.001

of assessment and definition of breakfast consumption.[3,7,8] Although researchers have typically defined breakfast as anything that the participant considers to be "breakfast" using questionnaires,[7–9,11] more specific definitions that have been proposed[1] may help to provide some consistency between studies in the future. Furthermore, there are currently no national recommendations for the frequency of breakfast consumption, with studies defining "habitual" breakfast eaters as those, for example, who consume breakfast on a certain number of days across the week,[11] school days only[9] or on the dietary survey day only.[8]

From a health perspective, breakfast consumption may favourably affect nutrition, body composition, and chronic disease risk markers; all of which have considerable relevance given public health concerns of obesity and associated cardiometabolic disorders in young people, including insulin resistance, dyslipidaemia, and hypertension.[12,13] This is particularly concerning for the younger generation since the transition from adolescence to adulthood appears to be a high-risk period for weight gain[14] and the temporal reduction in insulin sensitivity during the pubertal period.[15,16] It is also clear that childhood obesity tracks into adulthood[14] and can have adverse consequences on mortality and morbidity in later life.[17] Since childhood and adolescence have been identified as critical periods for the establishment of lifestyle behaviors, and it is widely recognised that obesity prevention provides a more effective and realistic solution than treatment, attention should be directed towards young people.[18]

The observed relationships between breakfast consumption and health may not be due to consumption *per se*, but rather the composition or "quality" of breakfast.[19] Ready-to-eat-breakfast-cereals (RTEBC) are often consumed for breakfast in westernised countries[20–22] and studies have distinguished between RTEBC and non-RTEBC consumers.[19,23,24] There has also been considerable interest in the health benefits of whole-grain, fibre-rich, low-energy-dense breakfasts that contain low glycaemic index (LGI) carbohydrates (CHO).[25,26] This complements the large body of research advocating the health benefits of LGI diets in adults[27] and, to a lesser extent, in young people.[28,29]

Previous reviews have comprehensively examined the health benefits of regular breakfast consumption and advocated the consumption of high-fibre breakfasts containing nutrient-rich whole grains in young people, but have only touched upon the effect of breakfast composition and paid little attention to breakfast glycaemic index (GI).[2,3] Thus, the purpose of this review is not to provide an exhaustive overview of the literature on breakfast consumption, but rather to focus on breakfast composition, particularly GI and metabolism. Following an overview of the epidemiological evidence on breakfast consumption, obesity, and health, the concept of glycaemic index is described and experimental evidence examining the effect of breakfast GI on health markers, metabolism, and satiety is reviewed. For clarity, breakfast skipping is defined as a habitual choice in free-living conditions (e.g., a child who chooses, for whatever reason, not to eat breakfast), whereas breakfast omission refers to an experimental manipulation that a study participant has consented to comply with when asked (e.g., an adolescent who normally eats breakfast, but has completed some measurements after an overnight fast as part of an experimental study).

2. Breakfast consumption, weight status, and disease risk markers

Various inter-related factors have contributed to the large multi-national increase in numbers of overweight and obese young people.[30] An imbalance between energy intake and expenditure is, however, often posited as the root of the problem. Breakfast consumption and composition represent modifiable factors that are both directly and indirectly related to the balance between energy intake and expenditure. A large body of cross-sectional evidence has shown consistently an inverse association between breakfast consumption and measures of obesity (most often body mass index (BMI)) in large diverse samples of young people and with the adjustment of potential confounding factors.[3,31,32] Moreover, prospective studies indicate that habitual breakfast consumption is predictive of lower BMI.[11,31] In a longitudinal study with 2216 adolescents and a 5-year follow-up, Timlin et al.[11] reported a dose–response inverse relationship between breakfast frequency and weight gain. Subsequently, a recent systematic review of 16 studies concluded that breakfast consumption was associated with reduced overweight and obesity risk in young people,[3] although it should be noted that these relationships have not always been observed; for example, a reduction in BMI over time was associated with breakfast consumption in non-overweight, but breakfast skipping in overweight adolescents.[33] Further longitudinal research with a 20-year follow-up indicated that skipping breakfast over prolonged periods of time led to more pronounced changes in weight gain and disease risk; participants who skipped breakfast in both childhood and adulthood had a higher BMI, waist circumference, HOMA-IR score, fasting insulin concentration and total and LDL-cholesterol concentration than those who consumed breakfast at both time points.[34] Breakfast consumption has also been associated with lower plasma total cholesterol concentration in young people,[35] but more research is required on relationships between breakfast consumption and cardiometabolic health markers. Nutrition, meal patterns, physical activity (PA), and other lifestyle factors are likely to contribute to the lower BMI and disease risk markers in breakfast consumers. However, it is important to highlight that breakfast consumption may simply be a marker for a healthy lifestyle in general; research that can infer causality between breakfast consumption and health-related variables would be required to refute such claims.

2.1. Nutrition

Since common breakfast foods come from the core food groups (breads and cereals, dairy products, and fruit), breakfast is typically a nutritious meal, low in fat and high in CHO.[36] Indeed, the nutritional benefits of regular breakfast consumption are well established in young people[2] and are

particularly relevant for this population since inadequate nutrient intake during childhood may be associated with adverse health outcomes during childhood, adolescence, and adulthood.[37] Breakfast consumption has been associated with favourable diet quality and nutritional status, reflected by higher micronutrient intakes and a greater likelihood of meeting recommended intakes for vitamins and minerals, including vitamins A and C, riboflavin, calcium, zinc, and iron.[6,7,38] The higher milk and calcium intake in breakfast consumers[31,32] is critical for young people since bone calcium accretion is highest during adolescence.[39] Importantly, young people who skip breakfast do not seem to make up the nutrient deficits through other meals consumed during the day.[6,38] Breakfast consumption is also associated with higher daily total energy, CHO, protein and dietary fibre intake, and lower total and saturated fat intake,[6,11,31,32] whilst the impact of breakfast consumption on sugar intake is unclear.[7,38] Findings that breakfast consumers have lower BMIs and higher energy intakes are somewhat contradictory, but suggest meal patterns and PA may be more important in explaining associations between breakfast consumption and BMI. Importantly, experimental data are emerging in adults, which reported no difference in daily energy intake when adults were asked to consume breakfast for one week and omit breakfast another week.[40] Interestingly, the effect of breakfast varied according to sex and morning eating habits; in the men, daily energy intake was higher in habitual breakfast consumers during the breakfast condition. In the women, however, habitual breakfast consumers ate more and later in the day under the breakfast omission condition.

Breakfasts containing cereal may be particularly beneficial for overall nutrient intake; RTEBC is typically low in fat, a good source of complex carbohydrates, fortified with vitamins and minerals and provides dietary fibre.[41] Nutritional benefits of regular RTEBC consumption are similar to those of breakfast consumption and include higher micronutrient, fibre, CHO, protein and reduced-fat and cholesterol intake,[20–24] as well as improved biochemical indices of nutritional status, i.e., serum vitamin and mineral concentrations.[42]

2.2. Meal patterns

Increased daily energy intake is unlikely to explain the higher BMI associated with breakfast skipping.[7,38,43] It is more likely that skipping breakfast leads to greater high-fat snacking[35,38] and energy intake later in the day to compensate for the energy deficit at breakfast, which predisposes obesity.[43,44] Indeed, consuming more energy earlier compared with later in the day may assist in weight loss in adults.[45] There is evidence that overweight and obese young people skip breakfast more frequently, consume a lower proportion of energy at breakfast, and consume a higher proportion of energy during dinner.[46,47] As sedentary behavior is more prevalent later in the day,[48] it is likely that the energy consumed in the late afternoon or evening will be stored as glycogen and fat, rather than metabolised through PA. Breakfast consumption may be a simple, yet effective, strategy to reduce energy intakes later in the day when young people may be less likely to expend the energy consumed. Conversely, the increased eating frequency associated with breakfast composition results in a more even distribution of energy intake throughout the day.[43] This may increase dietary induced thermogenesis and energy expenditure[48] and, consequently, contribute to healthy weight status. Indeed, meal frequency is inversely associated with obesity in young people.[49–51]

2.3. PA and healthy lifestyle behaviors

Observations that regular breakfast consumers may have higher daily energy intakes than breakfast skippers[7,38,43] suggest that these young people maintain a lower BMI by expending more energy. There is direct evidence that young people who consume breakfast habitually have higher PA and cardiorespiratory fitness levels.[9,52] Cardiorespiratory fitness is protective against chronic disease risk markers in young people[53,54] and morbidity and all-cause mortality in adults.[55] These findings are particularly relevant for girls since PA levels decline during adolescence[56] and are lower compared with boys.[57] Conversely, experimental data in adults have shown no difference in daily activity and energy expenditure when participants consumed or omitted breakfast for one week,[40] suggesting that breakfast consumption may not lead to increase in PA in this population. However, further experimental studies are required to clarify this finding. Although similar data in young people would be valuable, ethical restrictions may present challenges when asking children to omit breakfast over a period of time.

Other health-compromising behaviors associated with breakfast skipping in adolescents include tobacco, alcohol and substance use, although it should be highlighted that it is not possible to infer causality between these relationships.[11,58] It seems plausible that adolescents who readily adopt a variety of unhealthy lifestyle choices also skip breakfast. Those who consume healthier breakfasts containing LGI CHO and whole grain may also be more physically active and generally adopt healthy lifestyle behaviors. However, research investigating relationships between breakfast composition and PA does not appear to be available in young people.

2.4. Breakfast composition

Breakfast composition or “quality” is an important factor mediating the relationship between breakfast consumption and health. Regular RTEBC consumption has been associated with lower BMI and obesity risk in young people.[20] In a study comparing consumption of RTEBC and other breakfasts (foods or beverages other than RTEBC as the first meal) with breakfast skipping, adolescent RTEBC consumers had the lowest prevalence of obesity.[19] Frequent RTEBC consumption may also be protective against risk factors for chronic disease in young people, including reduced blood glucose[52] and low-density lipoprotein cholesterol[23,24] concentrations.

Since "RTEBC consumers" are "breakfast consumers", it is possible that just eating breakfast (but not necessarily RTEBC) may partly explain the reported health benefits of RTEBC consumption.[20] However, differences between RTEBC and other breakfast consumers imply the beneficial effect of breakfast consumption is enhanced with the inclusion of RTEBC. The nutrient fortification and low fat content of cereals may explain relationships between RTEBC consumption and nutrient intake. Compared with other breakfasts, RTEBC consumption is associated with greater nutritional benefits in young people, including higher intakes of total CHO, dietary fibre and several micronutrients and lower total fat and cholesterol intakes.[19,32] Lower fat intakes are associated with lower BMI in young people[47] and may prevent weight gain in adults.[59] Increased dairy calcium consumption that often accompanies RTEBC is also related to lower BMI in children[60] and interventions in adults have shown that increased calcium consumption may accelerate weight loss.[61]

In more recent years, it has been suggested that the association between RTEBC consumption and health may be attributed to the consumption of whole-grain and not refined-grain cereals, particularly regarding diabetes.[25,26] In young people, plasma total cholesterol was lower in those habitually consuming RTEBC with fibre compared with traditional breakfast, crisps ("chips") or sweets, other RTEBC and mixed breakfasts.[35] Indeed, the nutritional content of RTEBC varies considerably and there are concerns that the majority of RTEBC marketed to children fail to meet national nutrition standards. These cereals are typically denser in energy, sugar and sodium, but sparser in fibre and protein compared with cereals that are not marketed specifically for children.[62] Conversely, it is possible that the health benefits of RTEBC offset potential increases in added sugars and, in practice, the convenience and cost of RTEBC as a breakfast food may facilitate the promotion of breakfast consumption.[63] Breakfasts containing LGI rather than high glycaemic index (HGI) CHO typically have a lower energy density and contain higher amounts of dietary fibre.[64,65] However, evidence on the nutrient intakes of young people regularly consuming LGI compared with HGI breakfasts does not appear to be available. The consumption of RTEBC containing LGI CHO may provide an optimal balance of ensuring that breakfasts are nutritious, healthy and convenient for the consumer.

3. GI

Much of the research on the health benefits of breakfasts containing LGI CHO comes from experimental work investigating the acute effect of manipulations in GI on metabolism. The following section reviews this evidence, following an introduction on GI.

The concept of GI was introduced as a method of classifying different CHO-rich foods according to their effect on postprandial glycaemia. It is defined as the incremental area under the 2-h blood glucose curve following ingestion of 50 g available CHO as a percentage of the corresponding area following an equivalent amount of CHO from a standard reference product (glucose or white bread).[66] Typical blood glucose and plasma insulin responses to HGI and LGI breakfasts are displayed graphically in Fig. 1. Values for GI range from 1 to 100 and CHOs can be classified as high (≥70), moderate (56−69) or low (≤55). Foods classified as HGI include refined-grain products, white bread and potato, whereas LGI foods include whole-grain products, legumes and fruits. Numerous published tables now contain GI values for a variety of foods, including the international tables of glycaemic index.[67] As the extent of postprandial glycaemia depends on both the GI and the amount of CHO consumed, the glycaemic load (GL) was later proposed to provide an indication of the total glycaemic effect of the diet and is calculated as the product of the GI and total dietary CHO divided by 100.[68] Critically, the consumption of mixed meals composed of commonly consumed foods more closely reflects "real world" situations than assessing single CHO-containing foods. The GI of mixed meals can be predicted from the GI values of the component CHO foods. The weighted mean of the individual GI values is based on the percentage of the total meal CHO provided by each food and the predicted response is strongly correlated with the actual glucose response.[69,70]

Various food factors influence the GI of CHO-containing foods, which are affected by the method of preparation, processing, variety, origin, maturation, and degree of ripeness.[71,72]

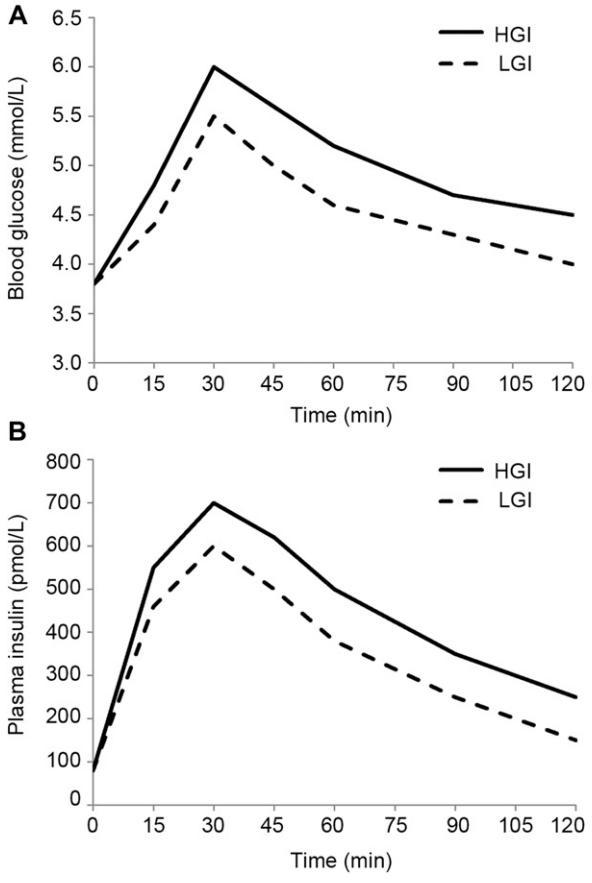

Fig. 1. Typical glycaemic and insulinaemic responses to a high glycaemic index (HGI) and a low glycaemic index (LGI) breakfast.

The term "available carbohydrate" represents parts of the CHO that can be digested and absorbed, excluding dietary fibre. The type of monosaccharide affects the GI, with fructose having a relatively low GI compared with glucose, although it should be noted that diets high in fructose have been implicated in insulin resistance.[64,71] The ratio of amylose/amylopectin in starch is another important factor; the branched amylopectin is more rapidly digested than the unbranched amylose and results in a higher GI.[73] The macronutrient content of foods also affects the GI, with protein and fat reducing the glycaemic response.[74–76] Although soluble fibre may lower the GI,[66,77] controversy generally surrounds the effect of dietary fibre on GI.[72] Glycaemic and insulinaemic responses to consumed CHO are generally well related.[70] However, in certain foods, although the GI can predict the glucose response to a meal it does not necessarily predict the insulin response.[72] The unexpectedly high insulinaemic index of milk[78] may be important when considering postprandial metabolism following breakfasts typically containing milk.

4. Dietary GI and health

The rate of glucose entry into the bloodstream and duration of elevated blood glucose concentration induce hormonal and metabolic changes that may affect health; mounting evidence suggests that the postprandial state contributes to the development of chronic disease.[79] A review in adults concluded that there is now a large body of evidence providing robust support for LGI diets in the prevention of obesity, diabetes and cardiovascular disease.[27] Although, relative to adults, a small number of interventions have assessed the impact of dietary GI on health markers in young people. The available evidence indicates reduced GI diets have implications for lowering BMI, metabolic syndrome and cardiovascular risk factors, hyperglycaemia, fasted glucose and insulin and hunger.[28,29,80] Moreover, a reduced-GL diet may be more effective at improving BMI and insulin sensitivity compared with a reduced-fat diet.[81] However, it should be noted that some have reported dietary GI may not influence health markers in children.[82] Encouragingly, health benefits of reducing dietary GI may be achieved by targeting just the breakfast meal in adults,[83] although these effects have yet to be investigated in young people. Potential health-enhancing effects of reduced GI diets in young people are, therefore, encouraging, but require greater research attention. Evidence on the effect of manipulating habitual GI at the breakfast meal only (rather than the diet as a whole) in young people would be valuable; cross-sectional and prospective associations between breakfast GI, BMI, and health markers should be explored.

5. Acute effect of breakfast GI on metabolism and satiety

Plausible mechanisms explaining relationships between dietary GI and health may arise from the contrasting acute metabolic responses to HGI and LGI foods. Indeed, much of the support for the promotion of LGI breakfasts comes from experimental studies investigating the acute effect of HGI and LGI breakfasts on metabolism and satiety. In young people, the effect of manipulations in breakfast GI on glucose, insulin, satiety, and fat oxidation have been investigated; the following section reviews this evidence and draws on the more extensive adult literature where appropriate.

5.1. Glucose and insulin

Numerous studies in adults have shown that LGI compared with HGI mixed-breakfast meals reduce postprandial glycaemia and insulinaemia[27,84,85] and studies in young people have provided similar findings.[86,87] In adults, LGI CHO consumption may also attenuate glycaemic and insulinaemic responses to standard subsequent meals due to the "second meal effect",[88] although similar evidence in young people appears to be unavailable. Reduced blood glucose decreases the quantity of insulin required to clear glucose from the blood, which may up-regulate insulin receptors on cells and increase insulin sensitivity.[89]

Recent findings in young people indicate that the higher glycaemic response to HGI compared with LGI breakfast consumption was exaggerated in overweight compared with non-overweight girls.[65] This was mainly due to the delayed decline in blood glucose following the postprandial peak and may indicate delayed blood glucose uptake up to 60 min following HGI breakfast consumption in overweight girls, possibly reflecting a reduced ability to cope with the metabolic demands of this breakfast. In support, higher and more sustained postprandial glucose responses have been reported in obese compared with non-obese young people,[90] but these studies did not investigate the potential interaction with the GI of the consumed CHO. 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 overweight girls may have contributed to this exaggerated glycaemic response.

5.2. Satiety

It is not surprising that that breakfast consumption compared with omission reduces feelings of hunger in young people,[91] but there is evidence that LGI breakfasts have additional satiating properties that may reduce subsequent food intake. It is this finding that has prompted much of the interest surrounding GI and body weight regulation and, importantly, there is evidence to support these claims in young people. In a well-controlled study, Warren et al.[92] reported lower lunch time energy intake and hunger ratings after LGI and LGI with added sugar breakfasts compared with HGI and habitual breakfasts (which were also HGI) in girls and boys.

During a 10-week intervention, Henry et al.[93] found a tendency towards reduced energy intake during a lunch time *ad libitum* buffet following LGI compared with HGI breakfast consumption in preadolescent children, although the mean difference was low (75 kJ, 18 kcal) and mainly confined to boys. In addition, data from 3-day food diaries showed a tendency towards a reduced energy intake during the LGI

compared with the HGI study period. However, glucose and insulin responses to the breakfasts were not determined in these studies, thus it is not possible to confirm whether the breakfasts differing in GI induced the expected metabolic responses.[92,93] Nevertheless, studies that have determined postprandial glucose and insulin concentrations support these findings and suggest a dose response; voluntary energy intake and hunger ratings were greatest after an HGI, followed by a moderate GI (MGI) and lowest after an LGI breakfast in obese adolescent boys.[87] However, although the HGI and MGI breakfasts were matched for key variables, the LGI breakfast contained less CHO, more protein and more fat than the HGI breakfast, possibly confounding the GI comparison.[87] In contrast, similar energy intake and hunger ratings were reported when comparing an LGI meal replacement, LGI whole-food meal and HGI meal replacement in overweight adolescents.[86] However, time to request additional food was prolonged following the LGI breakfast,[86] indicating that overweight and obese adolescents are satisfied for a longer time period after LGI compared with HGI breakfast consumption.[86,87]

In younger children aged 4−6 years, a high glycaemic load (HGL) compared with low glycaemic load (LGL) breakfast resulted in higher hunger levels before lunch, but this did not translate into differences in the amount of food and energy consumed during an *ad libitum* lunch.[94] However, between-participant variation may have confounded the between-trial comparisons; since the children were allowed to consume as they desired, those in the LGL group consumed lower amounts of total CHO, energy and dietary fibre and higher amounts of protein and fat at breakfast compared with the HGL group. Thus, the study design did not permit the examination of the independent effect of GL; decreased hunger prior to lunch in the LGL group may be ascribed to the higher protein and fat content of the LGL breakfast. An interesting observation of this study was that the children consumed more energy at breakfast when the HGL test breakfast was served, despite the similar hunger ratings before the two breakfast meals. This may be attributed to the greater energy density of the HGL compared with LGL breakfast, since no difference was found in the amount of food consumed at breakfast between the LGL and HGL intervention groups. Therefore, LGI breakfast consumption may not only reduce food intake later in the day, but also reduce energy intake at the breakfast meal. Further work in this area would be valuable and is required to support these findings.

It should be noted that some studies have shown no effect of breakfast GI on satiety in young people. Following the consumption of HGI and LGI breakfasts matched for energy and macronutrient content, no difference in perceived hunger was reported in overweight and non-overweight girls,[65] although it is possible the 2-h postprandial period was too short for differences to emerge.[86] In another study, refined and whole-grain breakfasts had a similar effect on satiation 2 h after consumption, with breakfast omission resulting in substantially higher hunger and tiredness levels.[91] However, a standard amount of each breakfast was given to all children, independent of individual factors such as size, weight or usual breakfast habits. Furthermore, although breakfast meals were matched for energy content, the children were not instructed to consume all of the breakfast provided and only four of the 28 participants consumed the entire breakfast for both trials. The authors did not compare energy intake statistically between breakfast conditions, but noted that 11 children consumed the entire refined breakfast and left some whole-grain breakfast. Therefore, it appears that the children were equally as hungry after both breakfasts despite many of them consuming less energy during the whole-grain condition, again highlighting that this type of breakfast may be particularly satiating. A prospective study of free-living children reported a higher daily energy intake in children in the lower tertile of breakfast GI who consumed a second early snack within 3 h of breakfast consumption, whereas breakfast GI did not affect subsequent daytime energy intake in children consuming their next meal during the late postprandial phase (>3−4 h).[95] This suggests that, when a morning snack is consumed within 3 h of breakfast consumption, HGI breakfasts may actually be more satiating. However, glucose and insulin responses to breakfast were not measured in this study.[94] Nevertheless, studies in adults also suggest that HGI foods may suppress short term voluntary energy intake more effectively than LGI foods.[96−98]

The lower energy intake and prolonged satiety following LGI breakfast consumption suggest that these breakfasts could have direct implications for weight management and may partly explain reported relationships between dietary GI and obesity.[28,99] Indeed, there is evidence that these acute LGI breakfast effects may translate into longer term reductions in hunger; self-reported hunger was reduced after a 6-week LGL diet (based on the replacement of at least 50% of the high GI foods with LGI foods) in prepubertal children.[80] In turn, reduced BMI may contribute to other health benefits associated with LGI diets, including increased insulin sensitivity and reduced cardiovascular risk factors. The similar palatability between whole and refined[91] and between HGI and LGI breakfasts in young people is encouraging.[65]

Differences in glycaemia might underpin the relationship between GI and satiety, as the lower glucose concentration following an LGI compared with HGI breakfast explained much of the lower voluntary food intake later in the day in obese adolescent boys.[87] The opposing effects of an HGI meal in the early and late postprandial phase can potentially be ascribed to a satiating effect of blood glucose spikes in the early postprandial phase,[97] which ceases once glycaemia drops to concentrations below baseline in the later postprandial phase.[100] Indeed, the rapid absorption of glucose following HGI breakfast consumption stimulates insulin release, which promotes glucose uptake by the liver, skeletal muscle, and adipose tissue, while suppressing both lipolysis in adipocytes and the release of glucose from the liver into the circulation. Subsequently, blood glucose concentration decreases rapidly. The decreased circulating concentrations of metabolic fuels following HGI breakfast consumption would be expected to result in increased hunger and food intake as the body attempts to restore energy homeostasis. In contrast, the attenuated

glucose response following LGI breakfast consumption stimulates more subtle hormonal responses and the prolonged and continued absorption of nutrients means that the fasted state is reached much later. The hunger response is, thus, prolonged following LGI breakfast consumption, promoting longer term satiety.[87,101]

5.3. Substrate metabolism during rest and exercise

Low rates of fat oxidation may be involved in the aetiology of obesity and accumulation of lipid within skeletal muscle can lead to abnormalities in insulin signalling and contribute to insulin resistance.[102,103] Maximising fat oxidation may, therefore, have important health implications for obesity and Type 2 diabetes. It is well established that fat oxidation is maximised in the fasted state, increasing in direct proportion to the duration of fasting[104] and being suppressed by CHO consumption.[105,106] In young people, exogenous CHO utilisation lowers the contribution of fat oxidation to energy expenditure during exercise.[107–110] The mechanisms responsible for the reduction in fat oxidation following CHO consumption relate to the rise in insulin that inhibits lipolysis and free fatty acid (FFA) availability[106] and the increase in blood glucose uptake and, therefore, CHO oxidation, which inhibits the rate of FFA entry into the mitochondria.[111,112] Although it is clear that exercise in the fasted state is preferential for augmenting fat oxidation, this may not be practical for young people and, as discussed throughout this review, regular breakfast consumption should be advocated for health.[1,11,23]

In adults, increased fat oxidation during the immediate postprandial rest period has been reported following an LGI compared with HGI breakfast.[85] However, the majority of studies have not supported this finding.[84,113,114] It was suggested that the lower CHO load in the Stevenson et al.'s[85] study compared with other studies reporting no effect of breakfast GI may have underpinned reported differences in resting fat oxidation. When individuals consumed an HGI or LGI breakfast and lunch, higher resting fat oxidation was reported following the LGI meals after lunch only.[115] However, the consumption of an HGI compared with LGI evening meal did not influence fat oxidation following a standard HGI breakfast the next morning in men[116] or women.[117] Studies examining the more prolonged effect of GI on substrate oxidation have reported no difference in resting fat oxidation over 10 h when obese women consumed an HGI or LGI breakfast and lunch.[113] Furthermore, consuming two HGI compared with LGI meals for 5 consecutive days actually resulted in higher fat oxidation in trained men.[118] In line with this finding, resting fat oxidation was higher after high glucose (HGI) compared with high fructose (LGI) meals in obese adults, despite greater glucose and insulin responses to the high glucose meal,[119] suggesting fat oxidation may depend on the type of LGI CHO consumed.

Unlike resting fat oxidation, the majority of studies support the finding that LGI compared with HGI breakfast consumption results in higher fat oxidation during exercise performed 45 min to 3 h after breakfast.[84,85,114,120] These observations have typically been accompanied by higher plasma FFA and glycerol concentrations following LGI breakfasts.[84,120–122] However, some have reported no effect of breakfast GI on exercise fat oxidation[123,124] and a recent study even reported higher fat oxidation when an HGI breakfast was consumed 45 min before a cycling time trial.[125] The relationship between GI and fat oxidation is further complicated by findings that breakfast GI does not affect fat oxidation during exercise when comparing an MGI and HGI breakfast[126] and no difference in fat oxidation was reported when exercise was preceded by two LGI or HGI meals rather than breakfast alone.[115] Furthermore, exercise fat oxidation was not affected when an LGI or HGI meal was provided the evening before[116,117]; this suggests that the "second meal effect" does not apply to fat oxidation.

In the only study, we are aware of, to investigate GI and substrate oxidation in young people, Zakrzewski et al.[65] examined the effect of HGI and LGI mixed-breakfast meals on fat oxidation in overweight and non-overweight girls. They focused on the 2-h postprandial rest period and a subsequent 30-min walk at 50% VO_{2peak}. Although breakfast GI did not affect postprandial fat oxidation during rest or exercise in either group of girls, it is noteworthy that LGI breakfast consumption resulted in 12% higher exercise fat oxidation (adjusted for fat free mass (FFM)) in both groups, a finding that may have meaningful health-related implications if experienced regularly over an extended period.[102] The similar insulin response between HGI and LGI reported in this study may have underpinned the similarity in fat oxidation.[106] Furthermore, fructose has a lower GI than glucose, but results in higher blood lactate concentrations.[127] It is possible that higher lactate concentrations compromised fat oxidation following the LGI breakfast through direct inhibition of adipose tissue FFA release.[128] Indeed, resting fat oxidation was lower after high fructose compared with high glucose meals in obese adults, despite lower glucose and insulin responses to the high fructose meal.[119] It is also possible that the 1.5 g CHO/kg body mass breakfast, 2-h postprandial period, and 30-min exercise duration at 50% VO_{2peak} was a sub-optimal combination to induce differences in fat oxidation between HGI and LGI. However, higher exercise fat oxidation following LGI breakfasts has been reported 45 min to 3 h[85,120] following breakfasts containing 1–2.5 g CHO/kg body mass during exercise lasting 60 or 30 min at 50%–71% VO_{2peak} in adults.[85,114] It is, therefore, difficult to ascertain which factors contribute specifically to the higher fat oxidation following LGI breakfasts in some adult studies. Furthermore, differences in fat metabolism between adolescents and adults[129] may have resulted in discrepancies between this study and some of the adult literature. Consequently, these results require confirmation with larger independent samples of young people.

It has been suggested that the reduced-fat oxidation following HGI breakfasts is largely due to the higher insulin response, which increases muscle glycogen stores and utilisation, resulting in higher CHO and lower fat oxidation.[114] Indeed, Wee et al.[114] reported increased muscle glycogen

concentration 3 h following an HGI breakfast, with no change following the LGI breakfast, and greater muscle glycogen utilisation during subsequent exercise in the HGI trial. Increased muscle glycogen utilisation following HGI breakfast consumption was reported previously,[130] but not consistently.[124] Contrasting findings may have been due to major differences in study design and, in particular, differences in the timing of the muscle biopsy, which was obtained 30 min[114] or 2 h[124] after exercise. Differences in *FAT/CD36* gene expression following HGI and LGI CHO consumption may be another underlying mechanism controlling differences in fat oxidation. In men, *FAT/CD36* mRNA and protein levels were down-regulated 3 h after the consumption of an HGI post-exercise meal, but were unchanged when an isoenergetic LGI meal with similar macronutrient content was consumed.[131] Conversely, muscle glucose transporter type 4 (*GLUT-4*) expression was reduced similarly following both meals, suggesting that this is not implicated in the relationship between GI and substrate oxidation. The effect of GI on *FAT/CD36* expression may also be mediated through differences in the insulin response to meals differing in GI.[132,133]

6. Summary and recommendations for future research

Regular breakfast consumption is associated with a variety of nutritional and lifestyle-related health outcomes in large diverse samples of young people, which may prevent weight gain, nutrient deficiency, and the development of chronic disease risk factors. Health benefits of breakfast consumption may be enhanced with the inclusion of RTEBC, particularly those containing LGI carbohydrates. Substituting an HGI breakfast for an LGI breakfast may be particularly beneficial for overweight young people through increased glycaemic control, fat oxidation and satiety. Overall, the potential benefits of LGI breakfasts seem to indicate that this could represent a positive factor supplementary to regular breakfast consumption. Breakfast consumption and composition, therefore, represent an important area of research that may have broad public health applications in obesity prevention and health. However, it is noteworthy that breakfast comprises just one component of a healthy lifestyle and those involved in breakfast promotion should highlight this to the target audience.

Research on breakfast consumption and health has typically taken the form of cross-sectional and descriptive prospective studies; controlled, systematic experimental studies are required to infer causality and the mechanisms controlling these relationships require further investigation. However, randomised controlled trials involving the intentional manipulation of breakfast omission over a period of time may be challenging for ethical reasons. Conversely, evidence surrounding breakfast GI and health is most often based on experimental research. There is a notable gap in the literature that has recognised the integrative effect of regular breakfast consumption and breakfast GI. Thus, large observational studies and interventions differentiating between HGI and LGI breakfasts are required and could provide valuable data required to strengthen health claims of LGI breakfast consumption.

References

1. Timlin MT, Pereira MA. Breakfast frequency and quality in the etiology of adult obesity and chronic diseases. *Nutr Rev* 2007;**65**(6 Pt 1):268−81.
2. Rampersaud GC, Pereira MA, Girard BL, Adams J, Metzl JD. Breakfast habits, nutritional status, body weight, and academic performance in children and adolescents. *J Am Diet Assoc* 2005;**105**:743−60.
3. Szajewska H, Ruszczyńskia M. Systematic review demonstrating that breakfast consumption influences body weight outcomes in children and adolescents in Europe. *Crit Rev Food Sci Nutr* 2010;**50**:113−9.
4. Lien L. Is breakfast consumption related to mental distress and academic performance in adolescents? *Public Health Nutr* 2007;**10**:422−8.
5. Graham MV, Uphold CR. Health perceptions and behaviors of school age boys and girls. *J Commun Health Nurs* 1992;**9**:77−8.
6. Nicklas TA, Bao W, Webber LS, Berenson GS. Breakfast consumption affects adequacy of total daily intake in children. *J Am Diet Assoc* 1993;**93**:886−91.
7. Nicklas TA, Reger C, Myers L, O'Neil C. Breakfast consumption with and without vitamin-mineral supplement use favorably impacts daily nutrient intake of ninth-grade students. *J Adolescent Health* 2000;**27**:314−21.
8. Sampson AE, Dixit S, Meyers AF, Houser RJ. The nutritional impact of breakfast consumption on the diets of inner-city African-American elementary school children. *J Natl Med Assoc* 1995;**87**:195−202.
9. Sandercock GR, Voss C, Dye L. Associations between habitual school-day breakfast consumption, body mass index, physical activity and cardiorespiratory fitness in English schoolchildren. *Eur J Clin Nutr* 2010;**64**:1086−92.
10. Siega-Riz AM, Popkin BM, Carson T. Trends in breakfast consumption for children in the United States from 1965−1991. *Am J Clin Nutr* 1998;**67**:748−56.
11. Timlin MT, Pereira MA, Story M, Neumark-Sztainer D. Breakfast eating and weight change in a 5-year prospective analysis of adolescents: project EAT (Eating Among Teens). *Pediatrics* 2008;**121**:e638−45.
12. Burke V. Obesity in childhood and cardiovascular risk. *Clin Exp Pharmacol Physiol* 2006;**33**:831−7.
13. Chiarelli F, Marcovecchio ML. Insulin resistance and obesity in childhood. *Eur J Endocrinol* 2008;**159**:67−74.
14. Gordon-Larsen P, Adair LS, Nelson MC, Popkin BM. Five-year obesity incidence in the transition period between adolescence and adulthood: the National Longitudinal Study of Adolescent Health. *Am J Clin Nutr* 2004;**80**:569−75.
15. Goran MI, Gower BA. Longitudinal study on pubertal insulin resistance. *Diabetes* 2001;**50**:2444−50.
16. Moran A, Jacobs DR, Steinberger J, Steffen LM, Pankow JS, Hong CP, et al. Changes in insulin resistance and cardiovascular risk during adolescence: establishment of differential risk in males and females. *Circulation* 2008;**117**:2361−8.
17. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes (London)* 2011;**35**:891−8.
18. Frieden TRY, Dietz W, Collins J. Reducing childhood obesity through policy change: acting now to prevent obesity. *Health Affairs (Millwood)* 2010;**29**:357−63.
19. Deshmukh-Taskar PR, Nicklas TA, O'Neil CE, Keast DR, Radcliffe JD, Cho S. The relationship of breakfast skipping and type of breakfast consumption with nutrient intake and weight status in children and adolescents: the National Health and Nutrition Examination Survey 1999−2006. *J Am Diet Assoc* 2010;**110**:869−78.
20. Albertson AM, Anderson GH, Crockett SJ, Goebel MT. Ready-to-eat cereal consumption: its relationship with BMI and nutrient intake of children aged 4 to 12 years. *J Am Diet Assoc* 2003;**103**:1613−9.

21. Ruxton CH, O'Sullivan KR, Kirk TR, Belton NR, Holmes MA. The contribution of breakfast to the diets of a sample of 136 primary-schoolchildren in Edinburgh. *Brit J Nutr* 1996;**75**:419−31.
22. Song WO, Chun OK, Kerver J, Cho S, Chung CE, Chung SJ. Ready-to-eat breakfast cereal consumption enhances milk and calcium intake in the US population. *J Am Diet Assoc* 2006;**106**:1783−9.
23. Albertson AM, Affenito SG, Bauserman R, Holschuh NM, Eldridge AL, Barton BA. The relationship of ready-to-eat cereal consumption to nutrient intake, blood lipids, and body mass index of children as they age through adolescence. *J Am Diet Assoc* 2009;**109**:1557−65.
24. Gibson S. Micronutrient intakes, micronutrient status and lipid profiles among young people consuming different amounts of breakfast cereals: further analysis of data from the National Diet and Nutrition Survey of Young People aged 4 to 18 years. *Public Health Nutr* 2003;**6**:815−20.
25. Kochar J, Djoussé L, Gaziano JM. Breakfast cereals and risk of type 2 diabetes in the physicians' health study I. *Obesity* 2007;**15**:3039−44.
26. Kosti RI, Panagiotakos DB, Zampelas A. Ready-to-eat cereals and the burden of obesity in the context of their nutritional contribution: are all ready-to-eat cereals equally healthy? A systematic review. *Nutr Res Rev* 2010;**23**:314−22.
27. Brand-Miller J, McMillan-Price J, Steinbeck K, Caterson I. Dietary glycemic index: health implications. *J Am Coll Nutr* 2009;**28**:446−9.
28. Rovner AJ, Nansel TR, Gellar L. The effect of a low-glycemic diet *vs*. a standard diet on blood glucose levels and macronutrient intake in children with type 1 diabetes. *J Am Diet Assoc* 2009;**109**:303−7.
29. Spieth LE, Harnish JD, Lenders CM, Raezer LB, Pereira MA, Hangen SJ, et al. A low-glycemic index diet in the treatment of pediatric obesity. *Arch Pediat Adol Med* 2000;**154**:947−51.
30. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *Lancet* 2002;**360**:473−82.
31. Affenito SG, Thompson D, Barton BA, Franko DL, Daniels SR, Obarzanek E, et al. Breakfast consumption in black and white adolescent girls correlates positively with calcium and fiber intake and negatively with body mass index. *J Am Diet Assoc* 2005;**105**:938−45.
32. Barton BA, Eldridge AL, Thompson D, Affenito SG, Striegel-Moore RH, Franko DL, et al. The relationship of breakfast and cereal consumption to nutrient intake and body mass index: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Am Diet Assoc* 2005;**105**:1383−9.
33. Berkey CS, Rockett HR, Gillman MW, Field AE, Colditz GA. Longitudinal study of skipping breakfast and weight change in adolescents. *Int J Obes Relat Metab Disord* 2003;**27**:1258−66.
34. Smith KJ, Gall SL, McNaughton SA, Blizzard L, Dwyer T, Venn AJ. Skipping breakfast: longitudinal associations with cardiometabolic risk factors in the Childhood Determinants of Adult Health Study. *Am J Clin Nutr* 2010;**92**:1316−25.
35. Resnicow K. The relationship between breakfast habits and plasma cholesterol levels in schoolchildren. *J School Health* 1991;**61**:81−5.
36. Williams P. Breakfast and the diets of Australian adults: an analysis of data from the 1995 national nutrition survey. *Int J Food Sci Nutr* 2005;**56**:65−79.
37. Viteri FE, González H. Adverse outcomes of poor micronutrient status in childhood and adolescence. *Nutr Rev* 2002;**60**:77−83.
38. Sjöberg A, Hallberg L, Höglund D, Hulthén L. Meal pattern, food choice, nutrient intake and lifestyle factors in The Göteborg Adolescence Study. *Eur J Clin Nutr* 2003;**57**:1569−78.
39. Weaver CM. The growing years and prevention of osteoporosis in later life. *Proc Nutr Soc* 2000;**59**:303−6.
40. Halsey LG, Huber JW, Low T, Ibeawuchi C, Woodruff P, Reeves S. Does consuming breakfast influence activity levels? An experiment into the effect of breakfast consumption on eating habits and energy expenditure. *Public Health Nutr* 2012;**15**:238−45.
41. Cotton PA, Subar AF, Friday JE, Cook A. Dietary sources of nutrients among US adults, 1994 to 1996. *J Am Diet Assoc* 2004;**104**:921−30.
42. Preziosi P, Galan P, Deheeger M, Yacoub N, Drewnowski A, Hercberg S. Breakfast type, daily nutrient intakes and vitamin and mineral status of French children, adolescents, and adults. *J Am Coll Nutr* 1999;**18**:171−8.
43. Dubois L, Girard M, Potvin Kent M, Farmer A, Tatone-Tokuda F. Breakfast skipping is associated with differences in meal patterns, macronutrient intakes and overweight among pre-school children. *Public Health Nutr* 2009;**12**:19−28.
44. Schlundt DG, Hill JO, Sbrocco T, Pope-Cordle J, Sharp T. The role of breakfast in the treatment of obesity: a randomized clinical trial. *Am J Clin Nutr* 1992;**55**:645−51.
45. Keim NL, Van Loan MD, Horn WF, Barbieri TF, Mayclin PL. Weight loss is greater with consumption of large morning meals and fat-free mass is preserved with large evening meals in women on a controlled weight reduction regimen. *J Nutr* 1997;**127**:75−82.
46. Ortega RM, Requejo AM, López-Sobaler AM, Quintas ME, Andrés P, Redondo MR, et al. Difference in the breakfast habits of overweight/obese and normal weight school children. *Int J Vitam Nutr Res* 1998;**68**:125−32.
47. Maffeis C, Provera S, Filippi L, Sidoti G, Schena S, Pinelli L, et al. Distribution of food intake as a risk factor for childhood obesity. *Int J Obes Relat Metab Disord* 2000;**24**:75−80.
48. Biddle SJ, Gorely T, Marshall SJ, Murdey I, Cameron N. Physical activity and sedentary behaviours in youth: issues and controversies. *J Roy Soc Health* 2004;**124**:29−33.
49. Drummond S, Crombie N, Kirk T. A critique of the effects of snacking on body weight status. *Eur J Clin Nutr* 1996;**50**:779−83.
50. Mota J, Fidalgo F, Silva R, Ribeiro JC, Santos R, Carvalho J, et al. Relationships between physical activity, obesity and meal frequency in adolescents. *Ann Hum Biol* 2008;**35**:1−10.
51. Toschke AM, Thorsteinsdottir KH, Kries RV. Meal frequency, breakfast consumption and childhood obesity. *Int J Pediatr Obes* 2009;**4**:242−8.
52. Kafatos A, Linardakis M, Bertsias G, Mammas I, Fletcher R, Bervanaki F. Consumption of ready-to-eat cereals in relation to health and diet indicators among school adolescents in Crete, Greece. *Ann Nutr Metab* 2005;**49**:165−72.
53. Allen DB, Nemeth BA, Clark RR, Peterson SE, Eickhoff J, Carrel AL. Fitness is a stronger predictor of fasting insulin levels than fatness in overweight male middle-school children. *J Pediat* 2007;**150**:383−7.
54. Jiménez-Pavón D, Castillo MJ, Moreno LA, Kafatos A, Manios Y, Kondaki K, et al. Fitness and fatness are independently associated with markers of insulin resistance in European adolescents; The HELENA Study. *Int J Pediatr Obes* 2011;**6**:253−60.
55. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease risk factors: a systematic review. *Obes Rev* 2010;**11**:202−21.
56. Armstrong N, Welsman JR. The physical activity patterns of European youth with reference to methods of assessment. *Sports Med* 2006;**36**:1067−86.
57. Riddoch CJ, Mattocks C, Deere K, Saunders J, Kirkby J, Tilling K, et al. Objective measurement of levels and patterns of physical activity. *Arch Dis Child* 2007;**92**:963−9.
58. Keski-Rahkonen A, Kaprio J, Rissanen A, Virkkunen M, Rose RJ. Breakfast skipping and health-compromising behaviors in adolescents and adults. *Eur J Clin Nutr* 2003;**57**:842−53.
59. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* 2000;**24**:1545−52.
60. Skinner JD, Bounds W, Carruth BR, Ziegler P. Longitudinal calcium intake is negatively related to children's body fat indexes. *J Am Diet Assoc* 2003;**103**:1626−31.
61. Zemel MB, Thompson W, Milstead A, Morris K, Campbell P. Calcium and dairy acceleration of weight and fat loss during energy restriction in obese adults. *Obes Res* 2004;**12**:582−90.
62. Schwartz MB, Vartanian LR, Wharton CM, Brownell KD. Examining the nutritional quality of breakfast cereals marketed to children. *J Am Diet Assoc* 2008;**108**:702−5.
63. Nicklas TA, McQuarrie A, Fastnaught C, O'Neil CE. Efficiency of breakfast consumption patterns of ninth graders: nutrient-to-cost comparisons. *J Am Diet Assoc* 2002;**102**:226−33.
64. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values. *Am J Clin Nutr* 2002;**76**:5−56.
65. Zakrzewski JK, Stevenson EJ, Tolfrey K. Effect of breakfast glycemic index on metabolic responses during rest and exercise in overweight and non-overweight adolescent girls. *Eur J Clin Nutr* 2012;**66**:436−42.

66. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, et al. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* 1981;**34**:362−6.
67. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values. *Diabetes Care* 2008;**31**:2281−3.
68. Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, et al. Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* 1997;**20**:545−50.
69. Wolever TM, Jenkins DJ. The use of the glycemic index in predicting the blood glucose response to mixed meals. *Am J Clin Nutr* 1986;**43**:167−72.
70. Wolever TM, Yang M, Zeng XY, Atkinson F, Brand-Miller JC. Food glycemic index, as given in glycemic index tables, is a significant determinant of glycemic responses elicited by composite breakfast meals. *Am J Clin Nutr* 2006;**83**:1306−12.
71. Englyst KN, Vinoy S, Englyst HN, Lang V. Glycaemic index of cereal products explained by their content of rapidly and slowly available glucose. *Br J Nutr* 2003;**89**:329−40.
72. Pi-Sunyer FX. Glycemic index and disease. *Am J Clin Nutr* 2002;**76**:290−8.
73. Granfeldt Y, Drews A, Björck I. Arepas made from high amylose corn flour produce favorably low glucose and insulin responses in healthy humans. *J Nutr* 1995;**125**:459−65.
74. Ercan N, Gannon MC, Nuttall FQ. Effect of added fat on the plasma glucose and insulin response to ingested potato given in various combinations as two meals in normal individuals. *Diabetes Care* 1994;**17**:1453−9.
75. Nuttall FQ, Mooradian AD, Gannon MC, Billington C, Krezowski P. Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. *Diabetes Care* 1984;**7**:465−70.
76. Nuttall FQ, Gannon MC, Wald JL, Ahmed M. Plasma glucose and insulin profiles in normal subjects ingesting diets of varying carbohydrate, fat, and protein content. *J Am Coll Nutr* 1985;**4**:437−50.
77. Nuttall FQ. Dietary fiber in the management of diabetes. *Diabetes* 1993;**42**:503−8.
78. Ostman EM, Liljeberg Elmståhl HG, Björck IM. Inconsistency between glycemic and insulinemic responses to regular and fermented milk products. *Am J Clin Nutr* 2001;**74**:96−100.
79. Heine RJ, Balkau B, Ceriello A, Del Prato S, Horton ES, Taskinen MR. What does postprandial hyperglycaemia mean? *Diabetic Med* 2004;**21**:208−13.
80. Fajcsak Z, Gabor A, Kovacs V, Martos E. The effects of 6-week low glycemic load diet based on low glycemic index foods in overweight/obese children—pilot study. *J Am Coll Nutr* 2008;**27**:12−21.
81. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediat Adol Med* 2003;**57**:773−9.
82. Cheng G, Karaolis-Danckert N, Libuda L, Bolzenius K, Remer T, Buyken AE. Relation of dietary glycemic index, glycemic load, and fiber and whole-grain intakes during puberty to the concurrent development of percent body fat and body mass index. *Am J Epidemiol* 2009;**169**:667−77.
83. Pal S, Lim S, Egger G. The effect of a low glycaemic index breakfast on blood glucose, insulin, lipid profiles, blood pressure, body weight, body composition and satiety in obese and overweight individuals: a pilot study. *J Am Coll Nutr* 2008;**27**:387−93.
84. Stevenson EJ, Williams C, Mash LE, Phillips B, Nute ML. Influence of high-carbohydrate mixed meals with different glycemic indexes on substrate utilization during subsequent exercise in women. *Am J Clin Nutr* 2006;**84**:354−60.
85. Stevenson EJ, Astbury NM, Simpson EJ, Taylor MA, Macdonald IA. Fat oxidation during exercise and satiety during recovery are increased following a low-glycemic index breakfast in sedentary women. *J Nutr* 2009;**139**:890−7.
86. Ball SD, Keller KR, Moyer-Mileur LJ, Ding YW, Donaldson D, Jackson WD. Prolongation of satiety after low versus moderately high glycemic index meals in obese adolescents. *Pediatrics* 2003;**111**:488−94.
87. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. *Pediatrics* 1999;**103**(3):E26.
88. Liljeberg H, Björck I. Effects of a low-glycaemic index spaghetti meal on glucose tolerance and lipaemia at a subsequent meal in healthy subjects. *Eur J Clin Nutr* 2000;**54**:24−8.
89. Song YJ, Sawamura M, Ikeda K, Igawa S, Yamori Y. Soluble dietary fibre improves insulin sensitivity by increasing muscle GLUT-4 content in stroke-prone spontaneously hypertensive rats. *Clin Exp Pharmacol Physiol* 2000;**27**:41−5.
90. Sinha R, Fisch G, Teague B, Tamborlane WV, Banyas B, Allen K, et al. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *New Engl J Med* 2002;**346**:802−10.
91. Pereira MA, Erickson E, McKee P, Schrankler K, Raatz SK, Lytle LA, et al. Breakfast frequency and quality may affect glycemia and appetite in adults and children. *J Nutr* 2011;**141**:163−8.
92. Warren JM, Henry CJ, Simonite V. Low glycemic index breakfasts and reduced food intake in preadolescent children. *Pediatrics* 2003;**112**(5):e414.
93. Henry CJ, Lightowler HJ, Strik CM. Effects of long-term intervention with low- and high-glycaemic-index breakfasts on food intake in children aged 8−11 years. *Br J Nutr* 2007;**98**:636−40.
94. LaCombe A, Ganji V. Influence of two breakfast meals differing in glycemic load on satiety, hunger, and energy intake in preschool children. *Nutr J* 2010;**9**:53.
95. Buyken AE, Trauner K, Günther AL, Kroke A, Remer T. Breakfast glycemic index affects subsequent daily energy intake in free-living healthy children. *Am J Clin Nutr* 2007;**86**:980−7.
96. Anderson GH, Catherine NL, Woodend DM, Wolever TM. Inverse association between the effect of carbohydrates on blood glucose and subsequent short-term food intake in young men. *Am J Clin Nutr* 2002;**76**:1023−30.
97. Anderson GH, Woodend D. Effect of glycemic carbohydrates on short term satiety and food intake. *Nutr Rev* 2003;**61**(5 Pt 2):S17−26.
98. Holt SH, Brand Miller JC, Petocz P. Interrelationships among postprandial satiety, glucose and insulin responses and changes in subsequent food intake. *Eur J Clin Nutr* 1996;**50**:788−97.
99. Du H, van der ADL, van Bakel MM, Slimani N, Forouhi NG, Wareham NJ, et al. Dietary glycaemic index, glycaemic load and subsequent changes of weight and waist circumference in European men and women. *Int J Obesity (London)* 2009;**33**:1280−8.
100. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA* 2002;**287**:2414−23.
101. Brand-Miller JC, Holt SH, Pawlak DB, McMillan J. Glycemic index and obesity. *Am J Clin Nutr* 2002;**76**:281−5.
102. Holloway GP, Bonen A, Spriet LL. Regulation of skeletal muscle mitochondrial fatty acid metabolism in lean and obese individuals. *Am J Clin Nutr* 2009;**89**:455−62.
103. Rogge MM. The role of impaired mitochondrial lipid oxidation in obesity. *Biol Res Nurs* 2009;**10**:356−73.
104. Montain SJ, Hopper MK, Coggan AR, Coyle EF. Exercise metabolism at different time intervals after a meal. *J Appl Physiol* 1991;**70**:882−8.
105. Achten J, Jeukendrup AE. The effect of pre-exercise carbohydrate feedings on the intensity that elicits maximal fat oxidation. *J Sports Sci* 2003;**21**:1017−24.
106. Horowitz JF, Mora-Rodriguez R, Byerley LO, Coyle EF. Lipolytic suppression following carbohydrate ingestion limits fat oxidation during exercise. *Am J Physiol* 1997;**273**(4 Pt 1):E768−75.
107. Riddell MC, Bar-Or O, Schwarcz HP, Heigenhauser GJ. Substrate utilization in boys during exercise with [13C]-glucose ingestion. *Eur J Appl Physiol* 2000;**83**:441−8.
108. Timmons BW, Bar-Or O, Riddell MC. Oxidation rate of exogenous carbohydrate during exercise is higher in boys than in men. *J Appl Physiol* 2003;**94**:278−84.
109. Timmons BW, Bar-Or O, Riddell MC. Influence of age and pubertal status on substrate utilization during exercise with and without carbohydrate intake in healthy boys. *Appl Physiol Nutr Metab* 2007;**32**:416−25.

110. Timmons BW, Bar-Or O, Riddell MC. Energy substrate utilization during prolonged exercise with and without carbohydrate intake in preadolescent and adolescent girls. *J Appl Physiol* 2007;**103**:995−1000.
111. Coyle EF, Jeukendrup AE, Wagenmakers AJ, Saris WH. Fatty acid oxidation is directly regulated by carbohydrate metabolism during exercise. *Am J Physiol* 1997;**273**(2 Pt 1):E268−75.
112. Sidossis LS, Stuart CA, Shulman GI, Lopaschuk GD, Wolfe RR. Glucose plus insulin regulate fat oxidation by controlling the rate of fatty acid entry into the mitochondria. *J Clin Inves* 1996;**98**:2244−50.
113. Díaz EO, Galgani JE, Aguirre CA, Atwater IJ, Burrows R. Effect of glycemic index on whole-body substrate oxidation in obese women. *Int J Obes* 2005;**29**:108−14.
114. Wee SL, Williams C, Tsintzas K, Boobis L. Ingestion of a high-glycemic index meal increases muscle glycogen storage at rest but augments its utilization during subsequent exercise. *J Appl Physiol* 2005;**99**:707−14.
115. Stevenson E, Williams C, Nute M. The influence of the glycaemic index of breakfast and lunch on substrate utilisation during the postprandial periods and subsequent exercise. *Br J Nutr* 2005;**93**:885−93.
116. Stevenson E, Williams C, Nute M, Swaile P, Tsui M. The effect of the glycemic index of an evening meal on the metabolic responses to a standard high glycemic index breakfast and subsequent exercise in men. *Int J Sport Butr Exer Metab* 2005;**15**:308−22.
117. Stevenson E, Williams C, Nute M, Humphrey L, Witard O. Influence of the glycaemic index of an evening meal on substrate oxidation following breakfast and during exercise the next day in healthy women. *Eur J Clin Nutr* 2008;**62**:608−16.
118. Cocate PG, Pereira LG, Marins JC, Cecon PR, Bressan J, Alfenas RC. Metabolic responses to high glycemic index and low glycemic index meals: a controlled crossover clinical trial. *Nutr J* 2011;**10**:1.
119. Tittelbach TJ, Mattes RD, Gretebeck RJ. Post-exercise substrate utilization after a high glucose *vs*. high fructose meal during negative energy balance in the obese. *Obes Res* 2000;**8**:496−505.
120. Sparks MJ, Selig SS, Febbraio MA. Pre-exercise carbohydrate ingestion: effect of the glycemic index on endurance exercise performance. *Med Sci Sports Exerc* 1998;**30**:844−9.
121. Wee SL, Williams C, Gray S, Horabin J. Influence of high and low glycemic index meals on endurance running capacity. *Med Sci Sports Exerc* 1999;**31**:393−9.
122. Wu CL, Williams C. A low glycemic index meal before exercise improves endurance running capacity in men. *Int J Sport Nutr Exerc Metab* 2006;**16**:510−27.
123. Bennard P, Doucet E. Acute effects of exercise timing and breakfast meal glycemic index on exercise-induced fat oxidation. *Appl Physiol Nutr Metab* 2006;**31**:502−11.
124. Febbraio MA, Stewart KL. CHO feeding before prolonged exercise: effect of glycemic index on muscle glycogenolysis and exercise performance. *J Appl Physiol* 1996;**81**:1115−20.
125. Moore LJ, Midgley AW, Thurlow S, Thomas G, McNaughton LR. Effect of the glycaemic index of a pre-exercise meal on metabolism and cycling time trial performance. *J Sci Med Sport* 2010;**13**:182−8.
126. Backhouse SH, Williams C, Stevenson E, Nute M. Effects of the glycemic index of breakfast on metabolic responses to brisk walking in females. *Eur J Clin Nutr* 2007;**1**:590−6.
127. Moore MC, Cherrington AD, Mann SL, Davis SN. Acute fructose administration decreases the glycemic response to an oral glucose tolerance test in normal adults. *J Clin Endocrinol Metab* 2000;**85**:4515−9.
128. Boyd III AE, Giamber SR, Mager M, Lebovitz HE. Lactate inhibition of lipolysis in exercising man. *Metabolism* 1974;**23**:531−42.
129. Riddell MC, Jamnik VK, Iscoe KE, Timmons BW, Gledhill N. Fat oxidation rate and the exercise intensity that elicits maximal fat oxidation decreases with pubertal status in young male subjects. *J Appl Physiol* 2008;**105**:742−8.
130. Febbraio MA, Keenan J, Angus DJ, Campbell SE, Garnham AP. Pre-exercise carbohydrate ingestion, glucose kinetics, and muscle glycogen use: effect of the glycemic index. *J Appl Physiol* 2000;**89**:1845−51.
131. Cheng IS, Liao SF, Liu KL, Liu HY, Wu CL, Huang CY, et al. Effect of dietary glycemic index on substrate transporter gene expression in human skeletal muscle after exercise. *Eur J Clin Nutr* 2009;**63**:1404−10.
132. Luiken JJ, Arumugam Y, Bell RC, Calles-Escandon J, Tandon NN, Glatz JF, et al. Changes in fatty acid transport and transporters are related to the severity of insulin deficiency. *Am J Physiol Endocrinol Metab* 2002;**283**:E612−21.
133. Smith AC, Mullen KL, Junkin KA, Nickerson J, Chabowski A, Bonen A, et al. Metformin and exercise reduce muscle FAT/CD36 and lipid accumulation and blunt the progression of high-fat diet-induced hyperglycemia. *Am J Physiol Endocrinol Metab* 2007;**293**:E172−81.