

**The effects of glycaemic index of mixed meals on
postprandial appetite sensation, cognitive
function; and metabolic responses during
intermittent exercise**

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for the degree of Doctor of Philosophy in Sport and Health Sciences

September 2013

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.....Mei Yi Wu

ABSTRACT

Glucose is the primary fuel for the brain and also important for exercising muscle. The purpose of the thesis was to investigate the effects of the glycaemic index (GI) of mixed meals on appetite, cognitive performances and metabolic responses during intermittent exercise in recreationally active adults.

Study one investigated whether a low GI (LGI) breakfast (GI = 42.5) could suppress appetite and reduce energy intake (EI) of 12 recreationally active females (28.2 ± 8.0 years) more than a high GI (HGI) breakfast (GI = 73.5). Area under the curve of the appetite score (AS AUC) following LGI breakfast was significantly greater than the HGI trial during the 60-min postprandial (pp) period (2568 ± 1027 vs. 2198 ± 821 mm·min, $p = 0.025$). The HGI breakfast facilitated a stronger appetite suppressing effect up to eight hours post breakfast than the LGI trial (18834 ± 3906 vs. 21278 ± 3610 mm·min, $p = 0.028$). The EI on the LGI trial day was significantly higher than on the pre-trial day ($2,215 \pm 576$ vs. $1,748 \pm 464$ kcal, corrected $p = 0.008$).

Fourteen recreationally active males (34.5 ± 8.9 years) in study two consumed the LGI (GI = 41.3) and HGI (GI = 74.3) breakfasts in the laboratory and then prescribed LGI and HGI meals in the free living environment. In line with study one, the AS AUC was significantly smaller following HGI than LGI breakfast over the 60-min pp period ($2,989 \pm 1,390$ vs. $3,758 \pm 1,290$ mm·min, $p = 0.027$). The HGI meals (GI = 76.9) suppressed appetite more than the LGI meals (GI = 39.6) over 12 hours on the trial day ($35,454 \pm 9,730$ vs. $41,244 \pm 8,829$ mm·min, $p = 0.009$) although energy balance was not different between trials.

Study three investigated whether following a LGI breakfast (GI = 42.2) providing $1 \text{ g CHO kg}^{-1} \text{ BM}$ could result in a better vigilance and attention than a HGI breakfast (GI = 72.4), and reduced lunch EI in 16 recreationally active males (24.4 ± 3.6 years). A significant trial x time effect in the interference time of the Stroop Colour Word Task (SCWT) ($p = 0.039$) showed that the LGI breakfast maintained the attentional performance up to 90-min pp. Both high pre-task glucose concentration ([Glucose]) at 15-min pp and low pre-task [Glucose] at 105-min pp in the HGI trial were associated with unfavourable outcomes in vigilance in the Rapid Information Processing Task (RIPT). The LGI pre-task [Glucose] returning back to fasting level at 60-min pp was associated positively with the response time. The pre-lunch AS was a significant predictor of the lunch EI per fat free mass which explained 21% and 26% of variance in the LGI and HGI trials respectively. No significant difference was found in the *ad libitum* lunch EI between trials.

Sixteen recreationally active males (27.8 ± 7.7 years) in study four consumed a LGI (GI = 42) and a HGI breakfast (GI = 72.8) providing $1.2 \text{ g CHO kg}^{-1} \text{ BM}$ consumed 60 minutes prior to intermittent running on two separate mornings.

Better attentional performance at 150-min pp was found following LGI than HGI breakfast. The significant trial x time interaction in the SCWT ($p = 0.045$) showed the shortest interference time performed after the last exercise session in the LGI trial. The amounts of CHO and fat being oxidized were comparable between trials during three sessions of 16-min intermittent running with an average intensity of 65% $\dot{V}O_2$ max.

In conclusion, the pre-meal appetite sensation is more predictive of the subsequent meal EI than the pre-meal [Glucose]. The meal strategy for weight management in recreationally active adults may focus on greater appetite suppression by selecting HGI foods whilst maintaining healthy eating guidelines. Recreationally active males performing sports requiring high levels of vigilance and selective attention with low physical activity levels can benefit up to 60–90 min pp from the LGI breakfast. Their attentional performance can benefit from the LGI breakfast with moderate to high intermittent intensities in the late exercise period at 150–min pp. Recreationally active adults should consider the timing of meal consumption in relation to performing intermittent exercise, in order to maximize the advantages from the LGI or HGI breakfasts for cognitive performance or appetite suppression. They may be more liberal in pre-exercise food choices if substrate oxidation during intermittent running is only of their concern.

ACKNOWLEDGEMENT

By this opportunity I would like to thank all who led me to complete my Ph.D.. Without their huge supports, the path to the doctorate would have never ended.

First and foremost, I would like to express my gratitude to Professor Craig Williams and Dr. Jo Bowtell. It was a very difficult decision to start a new research project one year after I started the Ph.D. study. I am fortunate to have Craig as my new first supervisor. This thesis would not have been completed without his insightful guidance and support. Although Jo became my second supervisor halfway through my study, her positivity and constructive advice are invaluable in speeding up my progress. I would also like to thank to the staff at the Sport and Health Sciences, particularly David Childs, James Blackwell, Len Maurer and Dr. Sarah Jackman; and Dr. Jonathan Fulford from the University of Exeter Medical School for technical supports. Thanks go to my officemates Andrew Williams, Zoe Saynor and Bert Bond who showed me the British culture; and Sandra Jonasdottir who encouraged me at the end of my Ph.D. journey. I would like to thank all participants in taking part in my studies.

I am deeply indebted to my family. Throughout this long, long journey my brother and sister have shared my family role; my father has endured my temper; and my mother has always been in my heart. Thanks for their patience and understanding. Thanks must go to my friends in Hong Kong and people I met in the church in Exeter for spiritual support.

May God bless us all with good health and wisdom.

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SUMMARY OF PRESENTATIONS

Paper

Wu, M. Y., Bowtell, J. L., & Williams, C. A. (2013). Glycaemic index of meals affects appetite sensation but not energy balance in active males. *Eur J Nutr.* doi: 10.1007/s00394-013-0529-3

Presentations

Wu, D.M.Y., Rowlands, A.V. & Williams C.A. (2011). Effect of low glycaemic index meals on appetite sensation and energy balance of male adult athletes. *Oral presentation at the British Association of Sport and Exercise Sciences (BASES) Student Conference, Chester, U.K.*

Wu, D.M.Y., Rowlands, A.V., Williams, C.A. (2011). Effect of low glycaemic index meals on appetite sensation and energy balance of male adult athletes. *Oral presentation at the European College of Sport Science Congress, Liverpool, U.K.*

Wu, D.M.Y., Bowtell, J.L., Williams, C.A. (2013). Glycaemic index breakfasts affect cognition but not lunch intake in athletic males. *Oral presentation at 18th annual Congress of the European College of Sport Science, Barcelona, Spain.*

Wu, D.M.Y., Bowtell, J.L., Williams, C.A. (2013). Relationships between glycaemic index, cognitive responses and intermittent running performance in adult athletes. *Oral presentation at BASES Conference 2013, Lancashire, U.K..*

DEFINITIONS AND ABBREVIATIONS

For the purposes of this research, the following terms and abbreviations were defined with respect to their usage in this thesis

ANOVA	Analysis of variance, Statistical measure to test for significant differences between means
AS	Appetite score, is a composite score assessed by VAS with five appetite related elements (Chaput, Gilbert, Gregersen, Pedersen, & Sjodin, 2010)
AOAC	Association of Official Analytical Chemists
AUC	Area under the curve
BH	Body stature; expressed in metre
BM	Body mass; expressed in kg
BMI	Body mass index; expressed in $\text{kg}\cdot\text{m}^{-2}$
CHO	Carbohydrate, an organic compound containing carbon, hydrogen and oxygen; a macronutrient which converts into glucose in body
CRT	Choice Reaction Time, a cognitive task to detect selective attentional performance
DEBQ	Dutch Eating Behaviour Questionnaire (Van Strien, Frijters, Bergers, & Defares, 1986)
EAT-26	Eating attitude test (Garner, Olmsted, Bohr, & Garfinkel, 1982)
EI	Energy intake, expressed in MJ or kcal
EE	Energy expenditure, expressed in MJ or kcal
FFA	Free fatty acid
[FFA]	Free fatty acid concentration
FFM	Fat free mass
FFQ	Food frequency questionnaire
g	Gramme

GI	Glycaemic index, a ratio of the incremental area under the curve of postprandial glucose of a test food containing 50 g CHO to that of the reference food containing 50 g CHO (Jenkins et al., 1981)
GL	Glycaemic load, an extension of GI which represents the overall glycaemic effect of the diet; calculated by multiplying the GI of the diet by available CHO in g
[Glucose]	Glucose concentration
Hb	Haemoglobin
Hct	Haematocrit
HGI	High glycaemic index, the overall value of a mixed diet or an individual food or drink is equivalent to 70 or more
hr	hour
HR	Heart rate; expressed in beats per minute
HRmax	Maximal heart rate; expressed in beats per minute
IAUC	Incremental area under the curve
[Insulin]	Insulin concentration
kcal	Kilocalories
kg	Kilogramme
kJ	Kilojoules
km	Kilometres
km·hr⁻¹	Kilometres per hour
[Lactate]	Lactate concentration
LGI	Low glycaemic index, the overall value of a mixed diet or an individual food or drink is equivalent to 55 or below
LT	Lactate Threshold, the transition between moderate and heavy intensity exercise that the oxygen uptake above which blood lactate exceeds the resting concentration during incremental exercise; expressed in mL·kg ⁻¹ ·min ⁻¹
MET	Metabolic equivalent, the amount of oxygen consumed while sitting at rest and equivalent to 3.5 mL·kg ⁻¹ ·min ⁻¹
min	Minute
mmol	Milimole
MJ	Megajoule

mL	Millilitre
mm	Millimetre
ms	Millisecond
n	Statistical measure that refers to the number of participants in a sample
NSP	Non-starch polysaccharide, is a dietary fibre defined by H. N. Englyst, Wiggins, and Cummings (1982)
PAL	Physical activity level
<i>p</i>	Statistical measure that denotes significance
pmol	Picomole
POMS	Profile of mood states (McNair, Lorr, & Droppleman, 1971)
pp	Postprandial
PS	Palatability score, is a composite score assessed by VAS with four palatability related elements (Sørensen, Moller, Flint, Martens, & Raben, 2003)
PV	Plasma volume
R	Reliability coefficients; statistical measure that express correlation between two measures
R²	Statistical measure that explain the percentage of variance of variables.
RIPT	Rapid Information Processing Task, a cognitive task to detect the vigilance performance
RER	Respiratory exchange ratio, the ratio of the rate of carbon dioxide production over the rate of oxygen consumption
RMR	Resting metabolic rate, the minimum energy required to sustain life and perform vital function at rest
RT	Response time; expressed in ms
SCWT	Stroop Colour Word Task, a cognitive task to detect selective and sustained attentional performance
SD	Standard deviation; statistical measure that reflects dispersion around the mean
SEM	Standard error; statistical measure that reflects the standard deviation of a measure divided by the square root of the number of measurements
TFEQ-R21	21-item Three Factor Eating Questionnaire (Cappelleri et al., 2009)

μL	Microlitre
VAS	Visual analogue scale, a 100-mm horizontal line to measure a characteristic or attitude subjectively
$\dot{V}\text{CO}_2$	Volume of carbon dioxide production
vLT	The speed reached LT; expressed in $\text{km}\cdot\text{hr}^{-1}$
$\dot{V}\text{O}_2$	Volume of oxygen consumption
$\dot{V}\text{O}_2\text{max}$	Maximal volume of oxygen output that reached plateau even further increase in the workload, expressed in a relative term as $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$
$\dot{V}\text{O}_2\text{peak}$	Maximal oxygen consumption at exertion, expressed in $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$
$v\dot{V}\text{O}_2\text{max}$	The speed reached $\dot{V}\text{O}_2\text{max}$; expressed in $\text{km}\cdot\text{hr}^{-1}$

CHAPTER 1 INTRODUCTION

Blood glucose is the primary energy fuel for the human body following carbohydrate (CHO) ingestion as a main source. According to the glucostatic theory developed over a half century ago, low blood glucose concentration ([Glucose]) is associated with initiation of food intake (Mayer, 1955). Even a small decline in plasma glucose concentration ([Glucose]) (6–12%) is suggested to predict meal initiation (Stubbs, 1999). There exists a reciprocal relationship between [Glucose] and appetite; and cognition (Fischer, Colombani, & Wenk, 2004; Stubbs, 1999). In addition, consumption of adequate CHO prior to an exercise event facilitates the delay of muscular glycogen depletion and/or the maintenance of blood [Glucose] for both muscle and brain as an energy fuel for cognitive, emotional, motivational and motor skill performance (Welsh, Davis, Burke, & Williams, 2002).

A functional carbohydrate classification system called glycaemic index (GI) has been established for over 30 years which ranks CHO rich foods based on the postprandial bioavailability of glucose (Jenkins et al., 1981). Consumption of low GI (LGI) foods sustains CHO bioavailability and produces more gradual rise in postprandial blood glucose level. Association of LGI diets with health benefits in individuals with chronic conditions have been widely reported due to their potential beneficial effect on regulating body weight (FAO, 1998; WHO/FAO, 2003) via better appetite suppression. However, not all previous studies found the advantage of LGI diets on weight management and appetite suppression (Raben, 2002; Roberts, 2000; USDA Evidence Analysis Library,

2012). Much of the previous research did not control confounding variables such as macronutrient composition, energy density and dietary fibre. In addition, a single drink or food rather than a mixed meal is not ecologically valid on a daily basis in a free living condition (Anderson, Catherine, Woodend, & Wolever, 2002; Stevenson, Williams, McComb, & Oram, 2005). There exists considerable inconsistency in the literature which requires clarification (Esfahani, Wong, Mirrahimi, Villa, & Kendall, 2011; Ford & Frost, 2010; Thomas, Elliott, & Baur, 2007).

Cognitive performance can be affected by psychological and physiological factors (Gilsenan, de Bruin, & Dye, 2009). There are relationships among blood [Glucose], mood and cognition. The specific effect of postprandial glycaemic response from different types of CHO on cognitive performance has not been fully investigated. Increases in circulating blood [Glucose] are reported to improve learning and memory (Donohoe & Benton, 1999; M. A. Smith & Foster, 2008) and cognitive function (Nabb & Benton, 2006), particularly under stress (Fairclough & Houston, 2004). The rapid and drastic decline in blood [Glucose] following high GI (HGI) foods may impair cognitive performance similarly to the hypoglycaemic condition (Fairclough & Houston, 2004). However, research seems to focus more on the associations between glucose profile and cognitive functions in people with impaired glucose tolerance or regulation (Lampert, Dye, Mansfield, & Lawton, 2012; Papanikolaou, Palmer, Binns, Jenkins, & Greenwood, 2006), children (Brindal et al., 2012; Cooper, Bandelow, Nute, Morris, & Nevill, 2012; Ingwersen, Defeyter, Kennedy, Wesnes, & Scholey, 2007; Micha, Rogers, & Nelson, 2010), and middle-aged to older adults (Nilsson, Radeborg, & Bjorck, 2009, 2012; Papanikolaou et al., 2006).

Optimal cognitive function is required for many sports that require the players to combine optimal physical performance and multi-dimensional coordination involving mood, physical movement and cognition for success. Certain skilled sports such as motor racing and rifle shooting require participants to perform fast decision making and optimise sustained and selective attention during an event with low or moderate physical activity. Other skill sports such as ball games like rugby and football; combat sports like taekwondo and boxing require players to keep alert with high levels of concentration, fast reaction and decision making during an event with moderate to high volume of movement. The effect of mixed meal GI on subsequent cognitive performance is therefore of importance, and has not previously been investigated in either recreationally active adults or athletes.

Besides the importance of glucose on appetite and cognition, glucose is also the main energy source to support physical activity. The reliance on CHO oxidation as an energy source increases with increasing exercise intensity (Coyle, 1995). The importance of consuming adequate CHO prior to an endurance exercise event for optimal exercise performance by maximizing the availability of CHO during exercise has been emphasized by sports and health concerned organisations (Rodriguez, DiMarco, & Langley, 2009). The performance advantages of controlling the pre-exercise meal GI have been investigated mostly on endurance exercise (Donaldson, Perry, & Rose, 2010). Although the maintenance of an adequate supply of CHO for muscle and the central nervous system is also important for reducing the risk of injury, optimising exercise and potentially psychological performance during exercise, only limited research investigating the effect of CHO consumption on both

physical and mental performance during intermittent exercise has been conducted (Welsh et al., 2002; Winnick et al., 2005). In addition little research has been conducted on the effects of GI foods taken prior to intermittent exercise (Cocate et al., 2011; Hulton, Gregson, Maclaren, & Doran, 2012; Little et al., 2010).

There are discrepancies in the effects of GI on appetite and cognition among studies which result from the differences in study design and populations. It has been suggested that physical activity, age, gender, body composition, physiological and environmental conditions can affect appetite (Stensel, 2010). It remains unclear whether the previous findings of the effects of GI on appetite and cognitive from sedentary, overweight or obese; young or older populations are applicable to recreationally active adults; and whether any occurrence of transient hypoglycaemia during the early stage of the onset of intermittent exercise affects mood, cognition and perceptual skill performance during exercise. To date the effects of GI at breakfast and at mixed meals on appetite and EI over a single day; cognitive performances and substrate oxidation during intermittent running in recreationally active adults have not been well investigated.

The main aim of the thesis was therefore to investigate the effects of GI on appetite, cognition and exercise performance in recreationally active adults. The purpose of study one was to investigate the effect of GI at breakfast on subsequent appetite sensation and energy homeostasis of recreationally active females. The purpose of study two was to investigate the effects of LGI and

HGI meals consumed over a single day on appetite and energy balance in recreationally active males. The purpose of study three was to investigate the acute effects on glucose response, appetite and subsequent EI; and mood and cognitive performance following LGI and HGI breakfasts in recreationally active males. Finally, the purpose of study four was to investigate the cognitive performances and metabolic responses during intermittent running following LGI and HGI breakfast taken an hour prior to running in recreationally active males.

CHAPTER 2 LITERATURE REVIEW

This chapter includes the background of the key terms and a comprehensive review of 1) the physiological effects of macronutrients and dietary fibre on appetite; 2) confounding variables related to GI values; 3) the effects of GI on various outcome measures, particularly on appetite, EI, cognitive function, and metabolic response and performances during exercise; and 4) measurement issues.

2.1 Appetite

2.1.1 What is Appetite?

Appetite is a driving force for the pursuit, selection and ingestion of food. Appetite can be divided into three components: hunger, satiation and satiety (Mattes, Hollis, Hayes, & Stunkard, 2005; Roberts, 2000). Hunger is considered as a sensation that makes an individual feel deprived of food to a level and motivates the individual to think and desire to obtain and consume foods. Satiation is a process of regulating food intake that develops while a person is ingesting food. Satiation determines meal volume, weight or size. A meal with a high effect of satiation inhibits the person from further food intake and leads to the termination of that meal. Satiety is a process following a meal to suppress the feeling of hunger and the urge to consume more food. Satiety determines the intervals between meals. A meal with high effect of satiety extends the time until the next meal or reduces the amount consumed at the next meal (Blundell, Lawton, Cotton, & Macdiarmid, 1996; Burton-Freeman, 2000). Appetite sensation can be determined subjectively by assessing the

degree of the feelings of hunger, desire to eat, prospective food consumption (PFC) and fullness (Anderson et al., 2002; Blundell et al., 2010; Chaput et al., 2010; Rogers & Blundell, 1979). Desire to eat is an urge to eat. Prospective food consumption indicates the supposed quantity of food to be eaten. Fullness is a sensation of the degree of gastric filling.

2.1.2 Eating behaviour and appetite

Eating patterns involve meal size, meal time, frequency of eating episodes and food selection (King, Lluch, Stubbs, & Blundell, 1997). A conceptual framework named 'Satiety cascade' was originally developed two decades ago to examine the impact of food ingestion on appetite (Blundell, Rogers, & Hill, 1987). The cascade shows that sensory, cognitive, pre-absorptive and post-absorptive factors play roles in satiation and satiety (Figure 2.1). The stomach is able to sense the amount of food being ingested which can lead to the termination of a meal, i.e. satiation (Stubbs, 1999). Sensory factors result from environmental stimuli such as smell, taste, palatability, etc. that drives the psychological state of 'liking' for variety and food choice. Cognitive factors involve the mood states such as at stress or at pleasantness. The energy density of meals plays role in appetite control as a pre-absorptive factor. Furthermore, pre-absorptive and post-absorptive factors physiologically involve the secretion of appetite related hormones, osmotic load, the ingested nutrient composition and the [Glucose]. Those physiological, psychological and environmental factors influence the initiation of food consumption (Blundell et al., 2010). However, there is considerable overlap and interaction among factors and thus it is difficult to categorise the variables into a single model.

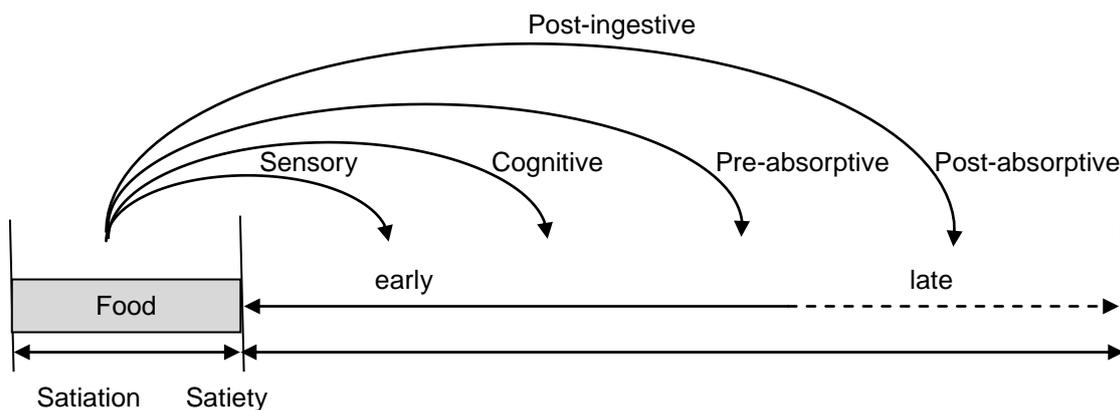


Figure 2.1 The Satiety Cascade (Blundell et al., 1987, pp. 205-219)

Previously de Castro (2004) found that low energy density intake reduced overall intake at any time of a day. In addition, breakfast produced greater levels of satiety and was associated with reduced total intake; whereas any intake in the late evening had less satiating and could lead to greater total intake. The author emphasized that the satiating effect is most effective when the high proportion of CHO was taken in the morning. Hasz and Lampert (2012) quoted that breakfast is defined as the first meal of the day being consumed prior to or at the start of daily activities within two hours of waking. Breakfast eaters can be categorised as having a breakfast providing 10–25% of recommended daily allowance for energy; at least three times of breakfast per week; or at least two major food groups (Murphy, 2007).

The number of food items in a meal was associated with the disruption of habituation that individuals eat more from meal with a variety of foods than from meals with a single food (De Graaf, Blom, Smeets, Stafleu, & Hendriks, 2004). Food variety affects the satiety provided by a meal and this involves a psychological effect from the sensory pleasantness which needs to be

controlled when studying the physiological variables to appetite and food intake. Meals with various food items were associated with higher EI when compared to monotonous meals, without significant differences in pre-meal hunger ratings (Sørensen et al., 2003). The varieties, not more than four items, were found to reduce disruption of habituation (Hetherington, Foster, Newman, Anderson, & Norton, 2006).

2.1.3 Exercise Pattern and appetite

Following exercise can have an influence of eating habits, both food preference and quantity, for energy compensation. An exercise-induced change in the eating pattern can suppress EI (King, Lluch, et al., 1997; King, Tremblay, & Blundell, 1997) or enhance EI (Stubbs, Sepp, Hughes, Johnstone, Horgan, et al., 2002; Stubbs, Sepp, Hughes, Johnstone, King, et al., 2002). There tends to have greater energy compensation over seven days in females (Stubbs, Sepp, Hughes, Johnstone, King, et al., 2002) than males (Stubbs, Sepp, Hughes, Johnstone, Horgan, et al., 2002). In addition, Mettler, Lamprecht-Rusca, Stoffel-Kurt, Wenk, and Colombani (2007); Mettler, Vaucher, Weingartner, Wenk, and Colombani (2008); Mettler, Wenk, and Colombani (2006) found that the postprandial glycaemic responses can be affected by the fitness level of males, but not females.

The above section summarised the elements of appetite sensation, the potential impact of physical activity, physiological and environmental conditions on appetite and these factors could be a confounder to affect appetite measurement (Stensel, 2010).

2.2 Carbohydrate structure and bioavailability

Carbohydrate provides $16 \text{ kJ}\cdot\text{g}^{-1}$ ($3.75 \text{ kcal}\cdot\text{g}^{-1}$) and contributes 40-75% of daily EI of healthy adults (WHO/FAO, 2003). Glucose becomes metabolically available to cells after the ingestion, digestion and then absorption of CHO. There are several classification systems for CHO: available and unavailable CHO; monosaccharide, disaccharide and polysaccharide; simple sugar and complex CHO, etc. (Table 2.1). Of which the Food Agriculture Organization has a comprehensive description of the classification of CHO (FAO, 1998).

Table 2.1 Classification of Carbohydrates

Classifications	Carbohydrates	Definition
Physicochemical (Asp, 1995)	Monosaccharide	<ul style="list-style-type: none"> • most basic units of CHO • cannot be decomposed by hydrolysis
	Disaccharide	<ul style="list-style-type: none"> • two monosaccharides undergo a condensation
	Oligosaccharide	<ul style="list-style-type: none"> • 3–10 monosaccharides bound together by glycosidic bonds • can be hydrolysed to yield basic units of CHO
	Polysaccharide	<ul style="list-style-type: none"> • more than 10 monosaccharides linked by glycosidic bonds • Starch and NSP
Traditional & Structural (FAO, 1998; Jenkins, Jenkins, Wolever, Thompson, & Rao, 1986)	Simple sugar	<ul style="list-style-type: none"> • Purified sucrose as refined sugar or added sugar
	Complex CHO	<ul style="list-style-type: none"> • Starch alone or the combination of all polysaccharides • Starch, dietary fibre and non-digestible oligosaccharides

Table 2.1 (Continued)

Classifications	Carbohydrates	Definition
Degree of polymerization (FAO, 1998)	Sugars	<ul style="list-style-type: none"> • Monosaccharides, disaccharides, polyols
	Oligosaccharides	<ul style="list-style-type: none"> • Malto-oligosaccharides • Other oligosaccharides including fructo-oligosaccharides
	Polysaccharides	<ul style="list-style-type: none"> • Starch and NSP
Physiological (FAO, 1998)	Available	<ul style="list-style-type: none"> • able to be hydrolysed to monosaccharides for metabolism
	Unavailable	<ul style="list-style-type: none"> • not able to be hydrolysed in human body • some may be fermented in the large intestine to varying extents
Nutritional (K. N. Englyst, Liu, & Englyst, 2007)	Available	<ul style="list-style-type: none"> • able to be digested and absorbed in the small intestine
	Resistant	<ul style="list-style-type: none"> • resist digestion • poorly absorbed or metabolized

The CHO in all studies in the current thesis was available CHO determined via the locally developed nutrient dietary analysis software (Microdiet 2.0, Downlee Systems Ltd., High Peak, U.K.) (Food Standards Agency, 2002; McCance, Holland, Widdowson, & Royal Society of Chemistry and Ministry of Agriculture Fisheries and Food, 1991).

2.3 Dietary fibre

There is a strong link between the definition of CHO and dietary fibre. No unique analytical method can be applied to determine the dietary fibre content in foods from the nutritional, chemical and physiological aspects. The dietary

fibre content in foods are commonly defined using the Englyst method (H. N. Englyst et al., 1982) and the enzymic gravimetric method by the American Association of Analytical Chemists (AOAC) (AOAC International, 2000; McCleary et al., 2012). The Englyst method determines non-starch polysaccharide (NSP), with an exclusion of lignin and resistant starch, as dietary fibre. The AOAC method defines dietary fibre as edible parts of plants or analogous CHO resistant to digestion and absorption in the human small intestine with complete or partial fermentation in the large intestine. The dietary fibre by the AOAC method thus includes soluble and insoluble oligosaccharides, NSP, lignin and resistant starch. Generally the AOAC method provides higher fibre content than the Englyst method (Figure 2.2).

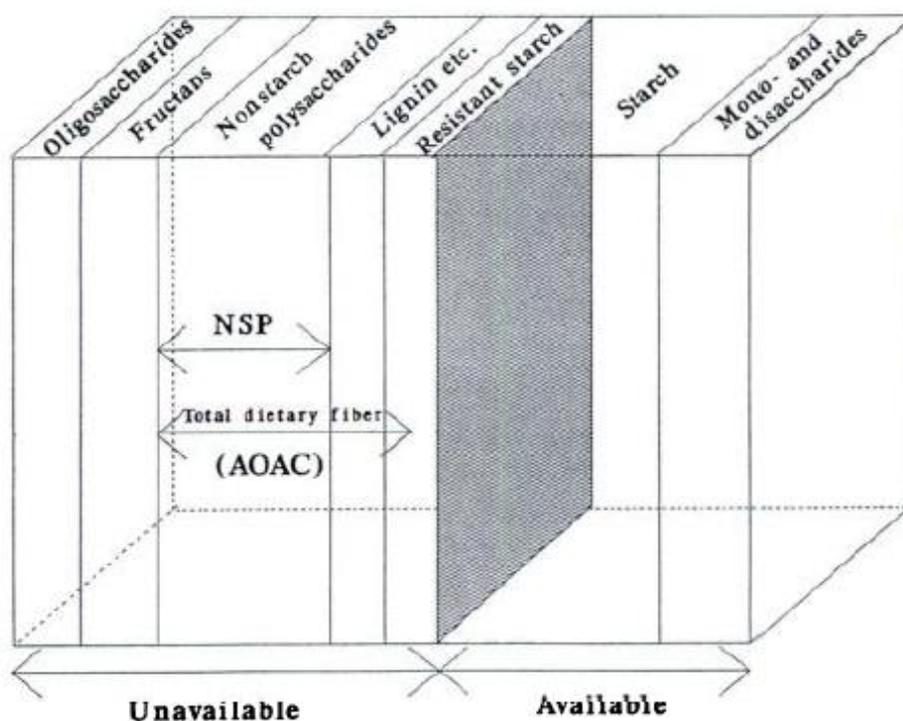


Figure 2.2 Definitions of carbohydrate and dietary fibres according to the methods of Englyst (NSP) and Association of Official Analytical Chemists (AOAC) (Source: Asp (1995, pp. 930S-937S))

The current recommendation for dietary reference intake of dietary fibre in the U.K. is NSP, based on the Englyst method (H. N. Englyst et al., 1982; Food Standards Agency, 2002; McCance et al., 1991). The dietary fibre by the AOAC method is recommended for the purposes of nutrition labelling (Food Standards Agency, 1999). The Food Standards Agency emphasized that the claims for dietary fibre measured by the AOAC method should not be related to the dietary reference value in the U.K. (Food Standards Agency, 1999). The contents of dietary fibre in all studies in the thesis were using the Englyst method (Food Standards Agency, 2002; McCance et al., 1991).

2.4 Glycaemic Index

The GI system is a measure of the postprandial bioavailability of glucose from CHO rich foods, usually contributing > 80% of energy (Jenkins et al., 1981). The ranking system classifies available CHO according to the rate of digestion and absorption in the human body after ingestion. The GI is defined as “the incremental area under the blood glucose response curve (IAUC) expressed as a percentage of the response to the same amount of CHO from a reference food taken by the same subject”. The GI value is a ratio of the two IAUC from 50 g CHO of a test food over 50 g of either white bread or glucose acting as the reference food (Brouns et al., 2005; FAO, 1998; Jenkins et al., 1981). The glucose IAUC includes the area above the fasting level only.

$$\text{GI} = \frac{\text{IAUC for 50 g CHO from a test food}}{\text{IAUC for 50 g CHO from the reference food}} \times 100$$

Previously it was thought that ingestion of simple CHO produced rapid and higher increases in postprandial glucose response than do complex CHO. The previous classification by structure, i.e. simple and complex CHO, to estimate the postprandial glucose profile has been recognised as over simplified. White bread and glucose are the reference foods which are classified as complex CHO and simple sugar by structure; whereas both belong to the HGI foods.

When glucose is used as the reference food, the GI of glucose is assigned a value of 100 arbitrarily. A CHO rich food with the GI value lower than 55, between 55 and 70; and higher than 70 is defined as low, medium and high GI food respectively (The University of Sydney, 2010). Using white bread as the reference food requires multiplication of the GI values obtained when glucose is the reference food, by 1.43, i.e. $GI(\text{bread}) = GI(\text{glucose}) \times 1.43$ (Foster-Powell, Holt, & Brand-Miller, 2002). Foods containing small amount of CHO are not tested for determining the GI values due to the infeasibility of participants to consume the amount providing 50 g of CHO (Schakel, Schauer, Himes, Harnack, & Van Heel, 2008). Nearly 2,000 foods have been assessed based on the GI ranking system in the International Tables (Atkinson, Foster-Powell, & Brand-Miller, 2008; Foster-Powell et al., 2002) and from the official GI website (The University of Sydney, 2010).

It was reported that white bread as a reference food was more preferable than glucose as the rate of gastric emptying of glucose was slowed down due to the osmotic effects (WHO/FAO, 2003). Pure glucose has been now recommended as a better reference food in recent years (Brouns et al., 2005; Louie, Flood,

Turner, Everingham, & Gwynn, 2011). The reference food was switched from white bread to glucose in a GI validation study with 121 single foods during 1995 and 2008 (Bao, Atkinson, Petocz, Willett, & Brand-Miller, 2011). It is therefore reasonable to consider glucose is superior to be a reference food than white bread due to less influence by the extrinsic factors (Louie et al., 2011). The glycaemic response can be affected by several intrinsic and extrinsic factors (Table 2.2).

Table 2.2 Factors influencing glycaemic responses

Factors	Examples	Decrease GI	Increase GI
Nature of the monosaccharide	Glucose, fructose, galactose	Fructose	Glucose
Nature of the starch	Ratio of amylose to amylopectin	Amylose	Amylopectin
	Resistant starch	Resistant starch	
Food processing	Particle size	Large particles	Small particles
	Degree of starch gelatinization	Raw/uncooked	Cooked
	Food form	Immature	Ripen
		Cold	Heated
	Re-crystallization of the starch chains	Toasting, freezing or defrosting	
Others	Other macronutrients	Fat, protein	
	Anti-nutrients	Phytate	
	Acid	Vinegar	

It is important to standardize the methodology for determination of GI in CHO rich foods (Granfeldt, Wu, & Bjorck, 2006). Ideally test foods selected for a

study should be matched for the GI values in the International Tables or the online database geographically and botanically. Those selected foods not found directly in the International Tables or the online database can be matched to available foods with similar characteristics from those sources to estimate the GI values. The current thesis selected the foods with GI values obtained from healthy participants from the International Tables (Atkinson et al., 2008; Foster-Powell et al., 2002) and the official GI website (The University of Sydney, 2010).

2.5 Nutrients and Appetite

2.5.1 Carbohydrate

The glucostatic theory proposes that low blood [Glucose] is associated with a trigger of the initiation of food intake (Mayer, 1955). Thus it is expected a sustained postprandial glucose profile suppresses the feeling of hunger better than a drastic fluctuation of postprandial glucose response. However, Stubbs (1999) reflected that blood glucose level and glycogen stores exhibited a reciprocal relation to appetite and food intake. The review stated that high blood glucose level was associated with reduction of appetite and food intake. The EURODIAB Complications Study including nearly 3000 IDDM female and male adults found that higher CHO intakes were related to a lower BMI, waist-to-hip ratio and waist circumference (Toeller et al., 2001). High CHO intakes with higher postprandial glucose level, compared to low CHO intakes, appeared to result in lower body weight.

Nevertheless, Stubbs, Mazlan, and Whybrow (2001) queried the protective effect of CHO intake against weight gain as different types of CHO have

different influences on EI and sensory sensation. Anderson and Woodend (2003) raised concerns that the prevalence of obesity has been increasing despite the reduction of total EI from decreasing fat intake and increasing CHO consumption. The authors highlighted that quality of CHO intake should be investigated, as peripherally blood glucose might be the major determinant of satiety. High CHO Western diets are generally based on HGI foods such as potato and bread (Brand-Miller, Holt, Pawlak, & McMillan, 2002). Hasz and Lamport (2012) quoted that the intake of high sugar and / or high fat energy dense foods such as sweetened beverages has been increased while the intake of milk and pure fruit juice has gradually been decreased by children over the last decade in the U.S. The GI values of sweetened beverages generally are higher than those of milk and pure fruit juice, except from watermelon and pineapple, which imply that the food intakes have been shifted towards higher GI diets.

2.5.2 Protein

Protein provides $17 \text{ kJ}\cdot\text{g}^{-1}$ ($4 \text{ kcal}\cdot\text{g}^{-1}$) and is a building block in human body, though it does not play an important role for energy. Following protein ingestion there is a synergistic effect with leptin in the hypothalamus and thus protein is suggested as the most satiating macronutrient despite the mechanism being unclear (Stubbs, 1999; Westerterp-Plantenga, Rolland, Wilson, & Westerterp, 1999). Protein is also reported to be insulinotropic with or without changing postprandial glycaemic responses (Flint et al., 2004; Jenkins et al., 1981).

Stubbs (1998) reported that protein induced supercaloric compensation, i.e. under eating, when compared to fat which resulted in subcaloric compensation, i.e. over eating, and exceeded EI, per unit of energy ingested. Weigle et al. (2005) found from 19 overweight sedentary male and female (aged 41 ± 11 years) following high protein (30% energy) low fat (20% energy) fixed-meal diets for two weeks increased feeling of fullness and decreased feeling of hunger during the periods compared to the baseline 15%-protein 35%-fat fixed-meal diets. The authors further found that 30%-protein 20%-fat *ad libitum* diets resulted in sustained lower EI and loss of body weight and body fat; whereas the satiety levels remained during the subsequent 12-week period. The insulinotropic effect induced by high protein diet in that study did not change the postprandial glucose response compared with the baseline diets. Weigle et al. (2005) concluded that a high protein, low fat intake had a greater anorexic effect than a high CHO low fat diet. However, the 30%-protein fixed-meal diet was nearly 60% heavier in food mass than the baseline 15%-protein diet. In addition, the dietary fibre contents between diets were not reported. Thus the maintenance of satiety level might be due to both food volume and energy density differences.

Hochstenbach-Waelen, Veldhorst, Nieuwenhuizen, Westerterp-Plantenga, and Westerterp (2009) found that following a diet providing 35% energy from protein for three days increased EE, protein balance and satiety when compared with a three-day diet having 10% protein for energy contribution on 12 male and 12 female non overweight participants aged 25 ± 7 years. The authors concluded that protein was the most satiating macronutrient, followed by CHO and fat.

Nevertheless it should be noted that high protein diets may be accompanied by reduction of the CHO intake so as to maintain energy balance (Tipton, 2011).

2.5.3 Fat

Fat is the most energy dense macronutrient that provides $37 \text{ kJ}\cdot\text{g}^{-1}$ ($9 \text{ kcal}\cdot\text{g}^{-1}$). Fat enters the small intestine slowly and leads to a decline in the absorption of CHO which lowers the glycaemic response (Jenkins et al., 1981). Cholecystokinin secretion is sensitive to fat in small intestine. It is proposed that cholecystokinin is a potential mediator of fat induced satiety (Burton-Freeman, 2000). Stubbs (1999) stated that the satiety signal from that hormone appeared not to be strong enough to prevent excessive EI; thus considering fat as a potent suppressor of EI was a paradox. High fat diets were reported to be less satiating than isocaloric portions of high-CHO or high-protein diets due to its inhibiting property of physiological satiety signals (Brand-Miller et al., 2002; Lawton, Delargy, Brockman, Smith, & Blundell, 2000). The body poorly auto-regulates fat storage and thus the fat intake did not increase fat oxidation and was more likely to increase body fat mass.

Stubbs et al. (2001) stated that fat precipitated subcaloric compensation (over eating) and promoted excessive EI. Reduction of dietary fat intake for prevention and treatment of obesity has been advocated for three decades (Brand-Miller et al., 2002). Reducing 10% total EI from fat was associated with weight loss (Bray & Popkin, 1998), but weight rebound often occurs (Brand-Miller et al., 2002). There is an argument that the obesity rate increases in the U.S. despite the reduction of fat intake (Willett, 1998; Willett & Leibel, 2002).

However, a previous review with data from 20 countries found a strong and significant positive relationship (adjusted $R^2 = 0.78$, $p < 0.001$) between the percentage of energy contributed from fat and the prevalence of overweight (Bray & Popkin, 1998). Furthermore, different degrees of fatty acid saturation had different effects of satiety. The degree of fatty acid saturation was found to be inversely associated with satiety (Lawton et al., 2000). Toeller et al. (2001) found that higher saturated fat intakes predicted a higher waist-to-hip ratio in IDDM adults.

Individuals were found to increase the intake of foods rich in fat and sugar due to the highly palatability (Bell, Castellanos, Pelkman, Thorwart, & Rolls, 1998; Green, Burley, & Blundell, 1994; Miller, Niederpruem, Wallace, & Lindeman, 1994; Stubbs, Prentice, & James, 1997). Furthermore, it was more likely to have overeating at the subsequent meal following high sugar and high fat intakes (Blundell, Burley, Cotton, & Lawton, 1993; Green et al., 1994). The modern high fat Western diets are deemed to be the main cause of obesity due to high palatability and energy density of high fat foods (Stubbs et al., 2001; van Dam & Seidell, 2007). Excess EI, from any macronutrient, promotes fat storage. Positive energy balance is still the main contributor to obesity.

2.5.4 Dietary fibre

Many countries and health organisations advocate high fibre intake in diets. High fibre diets have been found to slow down stomach emptying and decelerate small intestinal transit time leading to an increase in satiety (Roberfroid, 1993). The delayed rate of stomach emptying was correlated with

the postprandial hunger (Warwick, Hall, Pappas, & Schiffman, 1993). The strength of the evidence of the NSP intake, rather than being mentioned as total dietary fibre intake, has been suggested to be convincing against weight gain and obesity (WHO/FAO, 2003).

An observational study with 23 lean and 23 obese males; 17 lean and 15 obese females showed that the amount of dietary fibre intakes in the obese males (20.9 ± 1.8 g) and females (15.7 ± 1.1 g) were significantly lower than in the lean males (27 ± 1.8 g) and females (22.7 ± 2.1 g) (Miller et al., 1994). After an adjustment of fibre intake as g of fibre per 1,000 kcal, the significant difference remained between the lean (12.8 ± 0.9 g per 1,000 kcal) and the obese (7.9 ± 0.8 g per 1,000 kcal) females although the significant difference diminished in the male group. Pereira and Ludwig (2001) reviewed 17 out of 27 experimental studies between 1984 and 2000 that showed a positive effect of dietary fibre on satiety. Short term appetite control appears to be determined by stomach distension rather than energy density of a meal (De Graaf et al., 2004). Rye bran bread (32.2% fibre) induced feeling of satiety and decreased hunger greater than wheat bread (2.5% fibre) in a within-subject comparison study (Isaksson, Fredriksson, Andersson, Olsson, & Aman, 2009). The authors claimed that diets rich in dietary fibre were associated with a lower risk of gaining weight. Among different types of dietary fibre, viscous fibre was found to induce satiety (Kristensen & Jensen, 2011). Another recent systemic review summarised that more soluble fibres had a greater impact on reducing acute EI than less soluble fibres (59% vs. 25%) (Wanders et al., 2011).

Since macronutrients and dietary fibre have great impact on affect the appetite, it is of importance to minimize these confounders by matching the amounts of macronutrients and dietary fibre in mixed meals

2.6 Benefits of Glycaemic Index

2.6.1 Glycaemic index and weight management

Association of LGI diets with health benefits in individuals with chronic conditions are widely reported. Some literatures have reported the effects of GI on improving glucose control in diabetic subjects, blood lipid profiles in hyperlipidaemic patients and weight control (Jenkins et al., 1981; Kochan et al., 2012; Ludwig, 2000, 2002, 2007; Wolever, Jenkins, Collier, et al., 1988). The World Health Organization (WHO) and Food and Agriculture Organization (FAO) of the United Nations stated that "Thus avoiding obesity and increasing intakes of a wide range of foods rich in non-starch polysaccharide and carbohydrate-containing foods with a low glycemic index offers the best means of reducing the rapidly increasing rates of NIDDM [non-insulin dependent diabetes mellitus] in many countries" (FAO, 1998, p. 19) and "Low-glycaemic foods have been proposed as a potential protective factor against weight gain and there are some early studies that support this hypothesis" (WHO/FAO, 2003, pp. 66-67). The rationale of LGI diets for weight control is due to its potential satiating effect leading to a reduction of subsequent EI. Several studies of the impact of GI on weight loss in overweight and obese adults have been reviewed (Table 2.3).

Slabber, Barnard, Kuyl, Dannhauser, and Schall (1994) recruited 30 obese and sedentary females in a 12-week parallel study involving energy restricted diets

either CHO with low insulin-response as the intervention or the conventional balanced as the control. The authors concluded that the intervention diet significantly reduced body weight to a greater extent than the control diet (-9.3 ± 2.5 vs. -7.4 ± 4.2 kg); however the difference in the weight loss did not reach statistical significance ($p = 0.14$). Several subsequent papers failed to recognise the non-significance and misreported the findings as significant and promoted LGI diets on weight control (Raatz et al., 2005; van Dam & Seidell, 2007).

A cross-over study demonstrated a significantly greater body fat loss following LGI diets than HGI diets (-520 vs. -20 g, $p < 0.05$) in 11 overweight males (86 ± 3 kg) for five weeks with another five weeks of washout (Bouché et al., 2002). No difference was found in the body weight between trials at the end of the period (86.5 ± 2.7 vs. 85.7 ± 2.9 kg). It should be noted that the authors did not report any change in body weight during the five-week washout period. In addition, there existed significant difference in the four-hour post breakfast glucose IAUC at the LGI and HGI baselines (65 ± 23 vs. 152 ± 22 $\text{mmol}\cdot\text{L}^{-1}\cdot\text{hr}^{-1}$, $p < 0.05$) and at week five (109 ± 22 vs. 187 ± 23 $\text{mmol}\cdot\text{L}^{-1}\cdot\text{hr}^{-1}$, $p < 0.05$) whilst no differences of the four-hour glucose IAUC following breakfast between the baseline and at week five for the same diet were found. The four-hour glucose IAUC following LGI breakfast at week five was 67% higher than at the baseline (65 ± 23 vs. 109 ± 22 $\text{mmol}\cdot\text{L}^{-1}\cdot\text{hr}^{-1}$) but no significant difference was reached. The authors did not explain explicitly the location of the significant difference by multiple analysis of variance ($p < 0.001$) between diets at baseline and at week five.

Conversely Wolever and Mehling (2003) provided staple LGI and HGI foods to 24 male and female participants with impaired glucose tolerance in a parallel study for four months. Despite an insignificant difference in EI between diets, greater weight losses were achieved following the HGI than the LGI diets. The authors agreed that the finding did not support the popular notion that reducing LGI diets resulted in weight loss.

In a 10-week study investigating the effect of GI on weight change and appetite with LGI and HGI test foods provided (Sloth et al., 2004), the author concluded that LGI diets were more effective than HGI diets with respect to appetite and body weight regulation and suggested a longer study duration, 6–12 months, for substantiating the findings. There are several pitfalls to draw such a conclusion. Firstly, results showed the existence of under-reporting. Secondly, although participants were requested to complete daily appetite ratings, the results of the appetite rating were not shown. Finally, no difference in the body weight loss was found between trials.

Carels, Darby, Douglass, Cacciapaglia, and Rydin (2005) also did not find a significant difference in body weight loss in 44 obese participants between the GI and the control groups in a parallel study in 20 weeks. Raatz et al. (2005) provided LGI and HGI foods with energy restriction to 19 participants for 12 weeks in a parallel study. However, no significant differences were found in the losses of body weight and fat mass between diets.

Ebbeling et al. (2005) conducted a parallel study for six months in 23 overweight and obese participants to investigate the effect of low glycaemic load (LGL) on weight loss between *ad libitum* LGL and low fat control diets. The author reduced the GL values for the experimental group by reducing the percentages of CHO and increasing the percentage of fat contribution for energy. No significant difference in body weight loss was found between groups. Although the study applied the concept of GL on the diet, de facto it was an investigation of the effect of lower CHO higher fat diet vs. the higher CHO lower fat diet on weight loss. Moreover, the LGL group was reported to have $47 \pm 2\%$ CHO and $33 \pm 1\%$ fat for energy contribution which is less likely to be adequate for recreationally active adults to support exercise training and performance. Despite a significant difference in the mean difference of GI between trials, the difference was only 6.3 units. Previously Holt, Brand, Soveny, and Hansky (1992) had suggested that any research investigating the effects of GI should have the difference of GI of at least 28 units.

Maki, Rains, Kaden, Raneri, and Davidson (2007) recruited 86 overweight and obese participants to investigate the effect of LGL on weight loss between *ad libitum* LGL and low fat control diets with energy restriction for 12 weeks in a parallel study. Participants in the LGL group lost more body weight, fat mass and FFM than the control group. The CHO intake of the LGL group was significantly lower than the control group (108 ± 6 vs. 171 ± 14 g, $p < 0.001$). This parallel study showed a common error of study design, as Ebbeling et al. (2005) manipulated the GL values between trials via advising participants to reduce the CHO intake. Although participants were advised to adopt low fat diets in the control group, no difference was found in the fat intake between

groups. It should also be noted that low CHO diets, i.e. $< 130 \text{ g}\cdot\text{day}^{-1}$, are not recommended in the treatment of overweight or obesity (American Diabetes Association, 2007).

Another similar study with 32 obese participants received LGI and HGI diet advices with energy restriction for eight weeks (Abete, Parra, & Martinez, 2008). The baseline plans provided $1,495 \pm 245$ and $1,568 \pm 225 \text{ kcal}\cdot\text{day}^{-1}$ for the LGI and HGI groups respectively but this did not take account of any significant variation as a baseline. Furthermore, the EI at the end of both trials were not reported. In addition, participants consumed more fibre in the LGI than the HGI groups (24.9 ± 5.1 vs. $18.5 \pm 5.1 \text{ g}$, $p = 0.002$) during the intervention period. However, the authors did not report the baseline advice of fibre intake.

A 12-week randomised crossover study without washout period provided LGI and HGI foods with well-matched energy, macronutrient and fibre intakes to 19 overweight and obese hyperinsulinaemic females (Aston, Stokes, & Jebb, 2008). No differences were found in the satiety and the EI between trials. However, there were significant differences in the fibre content and the percentage of available CHO contribution for energy after adjustment to the final EI. The final intake of CHO in both trials was reduced from the planned 60% to the actual $< 50\%$ of total EI. Despite a mean difference of GI units was adequate between trials as advised by Holt et al. (1992), the mean difference of GI between breakfasts was only 12 units. In addition, the measurement of venous plasma rather than capillary blood [Glucose] is another weakness in the study. The venous blood [Glucose] had a greater within-subject variation of postprandial

plasma glucose responses than the capillary blood [Glucose] (FAO, 1998; Wolever et al., 2003). It is therefore no surprise that there was no significant difference in the two-hour post breakfast AS and glucose responses. Aston et al. (2008) argued that larger GI differences between breakfasts very often would lead to differences in other macronutrient and fibre contents.

Another study investigated the effects of GI of LGI and HGI breakfasts on satiety in 16 females and 5 males who were overweight or obese for 21 days (Pal, Lim, & Egger, 2008). The authors found greater satiety after breakfast and before lunch following the LGI than the HGI breakfasts without the difference in total EI. It should be noted that the dietary fibre content in the LGI breakfast was nearly three folds higher than the HGI breakfast (9.9 vs. 3.2 g).

Livesey (2005) reviewed and suggested that low glycaemic CHO resulted in a lower body weight than the high glycaemic CHO diet in *ad libitum* environment in the term of GL. Lower GL diets were found to be associated with reduction of body weight linked to a greater scope for higher rate of fat oxidation. Despite the previous suggestion of the higher satiating effect of LGI foods, several recent reviews claimed that it lacks clear and insufficient evidence on whether low glycaemic foods as a strategy for managing body weight or satiety due to the presence of potential contribution of certain confounding factors such as fibre and palatability (Esfahani et al., 2011; Ford & Frost, 2010). Although LGI may be more effective in weight reduction in certain populations, there is still considerable inconsistency in the literature which requires clarification (Esfahani et al., 2011; Ford & Frost, 2010; Thomas et al., 2007). It may be helpful to

investigate if the appetite suppressing effect of GI in a shorter term would lead to weight loss without energy restriction (Esfahani et al., 2011).

Table 2.3 Summary of the long term effect of low GI diets on body weight in adults

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Slabber et al. (1994)	I: 15 F; 35.9 ± 5.8 yr; BMI 35 ± 5 Control: 15 F; 34.5 ± 6 yr; BMI 34.6 ± 3.4 (\bar{x} ± SD)	Parallel 12 wk	Diets supplied: ~ 4200–5000 kJ / day; 50% CHO I: CHO with low insulin response, CHO & protein <i>ad libitum</i> in separate meals, no snacks MO: Daily diet records	No difference in BW loss
Bouché et al. (2002)	11 M; 46 yr ± 3 yr BMI 28 ± 1 (\bar{x} ± SEM)	Randomized cross-over 2 x 5 wk with 5 wk washout interval	I: LGI (GI < 45) & HGI (GI > 60) food lists MO: 7-day dietary recall at wk 5	No difference in EI Reported GI: LGI < HGI (41.0 ± 1 vs. 71.3 ± 1.3; $p < 0.001$) 4-hr glucose IAUC after breakfasts at baseline & at week 5: HGI > LGI (152 ± 22 vs. 65 ± 23 mmol·1 ⁻¹ ·4 hr ⁻¹ , $p < 0.05$; 187 ± 23 vs. 109 ± 22 mmol·1 ⁻¹ ·4 hr ⁻¹ , $p < 0.05$) FM loss: LGI > HGI (-0.52 vs. -0.02 kg, $p < 0.05$) No difference in BW
Wolever and Mehling (2003)	All IGT LGI: 3 M & 10 F; 55.2 ± 3 yr; BMI 29.7 ± 1.2 HGI: 2 M & 9F; 58.8 ± 4 yr; BMI 29.3 ± 2.2 (\bar{x} ± SEM)	Randomized parallel 4 mth	I: at least 1 LGI or HGI food per meal at <i>ad libitum</i> basis; key foods provided MO: 3-day diet records at the last week of each mth, mthly fasting blood samples, BW measurement & consultation	No difference in EI Breakfast GI: LGI < HGI (50.7 ± 0.9 vs. 64.8 ± 0.6; $p < 0.05$) BW loss: LGI < HGI (-0.19 ± 0.4 vs. -0.49 ± 0.29 kg, $p < 0.05$)
Sloth et al. (2004)	LGI: 23 F; 28.9 ± 1.3 yr; BMI 27.6 ± 0.3 HGI: 22 F; 30.8 ± 1.3 yr; BMI 27.6 ± 0.3 (\bar{x} ± SEM)	Randomized parallel 10 wk	I: foods provided in 7-day rotation, covered ~ 75% of total CHO intake + <i>ad libitum</i> intake; +CHO food list; LGI (GI = 78.6); HGI (GI = 102.8) MO: 7-day weighed dietary records & 24-hr urine at wk 5 & 10; daily AS rating; bi-wkly BW measurement	> 95% provided food consumed EI decreased with time Under-reporting was found No difference in BW and FM loss

Table 2.3 (Continued)

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Carels et al. (2005)	GI group: 21 F; 43.4 ± 9 yr; BMI 38 ± 13.4 Control: 23 F; 43.5 ± 9.8 yr; BMI 37.2 ± 5.1 ($\bar{x} \pm SD$)	Randomized parallel 20 wk	I: Wkly meeting, GI education on GI group MO: Knowledge assessment test, attendance, diet records, wkly meeting	Better GI knowledge in GI group Diet GI: LGI < HGI (51.5 ± 4.7 vs. 56.5 ± 4.3, $p < 0.05$) EI decreased No difference in BW loss
(Raatz et al. (2005)	LGI: 3 M & 7F; 99.6 ± 5.9 kg; BMI 36.5 ± 1.8 HGI: 2 M & 7 F; 102 ± 4.7kg; BMI 34.6 ± 1.4 ($\bar{x} \pm SEM$)	Randomized parallel 12 wk	I: Provided LGI (GI = 33) high fibre (16.7 g / 4184 kJ) or HGI (GI = 63) lower fibre (9.1 g / 4184 kJ) hypocaloric meals; requested to eat all MO: anthropometric, biochemical measurement at wk 4, 8 & 12; daily dietary questionnaire	No difference in BW & FM loss
Ebbeling et al. (2005)	LGI: 11F; 93.3 ± 5.3 kg; 165.6 ± 2.1 cm Control: 1M 11F; 83.2 ± 3.3 kg; 163.3 ± 2.1 cm ($\bar{x} \pm SEM$)	Randomised parallel 6 mth	I: 45-50% CHO LGL (GL = 54) vs. 55-60% CHO low fat (GL = 78); <i>ad libitum</i> diet advice for 250-500 kcal/day deficit MO: bi-wkly counselling, 7-day food dairy at mth 3 & 6	GL & GI: LGI < control (54.4 ± 2.0 vs. 78.4 ± 1.4 per 1,000 kcal; 46.2 ± 1.6 vs. 52.8 ± 0.9) No difference in BW loss & energy deficit
Maki et al. (2007)	LGL: 14 M & 29 F; 91.2 ± 2 kg; BMI 32.1 ± 0.6 Low fat: 14 M & 29F; 88.7 ± 1.8 kg; BMI 31.6 ± 0.5 ($\bar{x} \pm SEM$)	Randomised parallel 12 wk	I: LGL group: avoid high CHO foods & alcohol x 2 wk, then LGI high fibre foods & moderate alcohol Control group: energy deficit, daily -500-800 kcal MO: anthropometry, 3-day food at wk 0, 2, 6 & 12; FFQ at wk 0 & 12; fasting insulin and glucose at wk -1 & 12	1 withdrew from each group BW, fat mass & FFM loss: LGL > control group (-4.9 ± 0.7 vs. -2.6 ± 0.9 kg, $p = 0.002$), (-1.9 ± 0.3 and -0.9 ± 0.3 kg, $p = 0.016$) & (-2.2 ± 0.2 vs. -1 ± 0.3, $p < 0.001$). No difference in the energy deficit CHO intake: LGL < control group (108 ± 6 vs. 171 ± 14 g, $p < 0.001$)
Abete et al. (2008)	LGI: 10M & 16F; 94.4 ± 13.1 kg; BMI 32.2 ± 4.4 HGI: 8M & 8F; 94.3 ± 16.1 kg; BMI 32.8 ± 4.3 ($\bar{x} \pm SD$)	Randomized parallel 8 wk	I: Hypocaloric diet advice (-30%); LGI (40-45) vs. HGI (60-65) MO: wkly meeting, 3-day dietary record, AS ratings, blood, 12-hr urine & anthropometry at wk 8	BW loss %: LGI > HGI (-7.5 ± 2.9 vs. -5.3 ± 2.6%; $p = 0.03$) Fibre intake: LGI > HGI (24.9 ± 5.1 vs. 18.5 ± 5.1 g, $p = 0.002$)

Table 2.3 (Continued)

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Aston et al. (2008)	19 F; 51.9 ± 1.7 yr; BMI 33.1 ± 4.9 ($\bar{x} \pm SD$)	Randomized cross over No washout period 12 wk x 2 times	I: LGI (GI = 55.5) vs. HGI (GI = 63.9) food provided MO: 4-day dietary record at the last wk	GI difference of 28.5 CHO intake: LGI > HGI (51.4 ± 6 vs. 47.6 ± 6.1%, $p = 0.01$) Fibre intake: LGI > HGI (18.4 ± 5 vs. 15.6 ± 4.5 g, $p = 0.04$) No difference in BW gain, lunch and snack EI; & post breakfast glucose response & AS
Pal et al. (2008)	16 F 5 M: 25-65 yr; 84.1 ± 4.9–85.3 ± 4 kg; BMI 32.8 ± 1–30.5 ± 0.2 ($\bar{x} \pm SEM$)	Randomized crossover 21-day washout ~ 21 hr x 2 times	I: LGI (35) & HGI (79) isocaloric breakfasts; 1081 vs. 1065 kJ; 9.9 vs. 3.2 g fibre MO: Satiety rating after breakfast, before lunch & dinner; plasma [Glucose], serum [Insulin], food record	Satiety change after breakfast and before lunch: LGI > HGI ($p < 0.05$) No difference in total EI, [Insulin] & BW changes Post 3-wk fasting [Glucose]: LGI < HGI (4.9 ± 0.1 vs. 5.1 ± 0.1 mmol·L ⁻¹ , $p < 0.05$)

Abbreviations: AS, appetite score; IAUC incremental area under curve; BW, body weight; BMI, body mass index in kg·m⁻²; CHO, carbohydrate; EI, energy intake; Lunch EI, lunch energy intake; F, female; FFM, fat free mass; FFQ, food frequency questionnaire; FM, fat mass; [Glucose], glucose concentration; HGL, high glycaemic load; HGI, high glycaemic index; I, intervention method; IGT, impaired glucose tolerant; [Insulin], insulin concentration; LGL, low glycaemic load; LGI, low glycaemic index; M, male; min, minute; SEM, standard error of mean; SD, standard deviation; MO, monitoring; mth, month; T2DM, type II diabetes; wk, week; \bar{x} , mean; yr, year.

2.6.2 Glycaemic index and appetite

The relationship between the glycaemic index and appetite was based on the glucostatic theory developed by Mayer (1955). It was suggested that the initiation of eating is controlled by brain, thus the primary fuel for brain plays role in appetite regulation. Previously, Ludwig et al. (1999) found that the ratings of hunger in 12 obese male adolescents aged 15.7 ± 1.4 years were higher following a HGI breakfast compared with a LGI breakfast. Furthermore, the subsequent EI five hours after the HGI breakfast was 81% greater than after the LGI breakfast (5.8 MJ vs. 3.2 MJ; $p < 0.05$). Several studies investigating the short term effect of GI on appetite in adults was summarised in Table 2.4.

Anderson et al. (2002) recruited 14 non obese male adults to investigate the subjective appetite and glucose responses to drinks with different GI. The authors found that 75 g glucose drink suppressed subjective appetite leading to lower subsequent EI and food intake in one hour postprandially than 75 g fructose-glucose (80/20) mixture drink. Glucose and fructose have the GI values of 100 and 20 respectively (Atkinson et al., 2008; Foster-Powell et al., 2002). Anderson et al. (2002) considered the glucose drink and the mixture drink as HGI and LGI CHO respectively in the study. In addition, AS at 60 minutes postprandially was positively correlated with the subsequent EI ($r = 0.45$, $p < 0.01$) and negatively correlated with the 60-min glucose AUC ($r = -0.24$, $p = 0.05$). A negative correlation was also observed between 60-min AUC of glucose and EI ($r = -0.24$, $p < 0.05$).

Arumugam et al. (2008) also found greater pre-lunch AS in 14 overweight females aged 47.7 ± 5.6 years after a bolus feeding of glucose drink (rapid) than small sipping of glucose drinks for eight times (slow) over four hours in the morning. The results also showed that the correlations between AS and the glucose response; and the insulin response were stronger in the rapid than in the slow conditions. Bolus feeding and small regular sipping of glucose simulated the rapid increase and gentle postprandial glucose responses as following HGI and LGI foods, respectively. Moreover the glucose response explained more of the variance than the insulin response. The authors supported the glucostatic theory (Mayer, 1955) that the postprandial glucose were negatively correlated with the feeling of hunger and PFC; and positively correlated with fullness. In addition, there seems to have a second meal effect that the AS patterns three to five hours post lunch were similar to post breakfasts.

A significant improvement in satiety was found in the LGI mixed meals compared to the HGI mixed meals in eight sedentary females (Stevenson, Astbury, Simpson, Taylor, & Macdonald, 2009). Participants consumed the LGI and HGI breakfasts on two different occasions at a basis of $1 \text{ g CHO kg}^{-1} \text{ BM}$ three hours before walking for 60 minutes. The dietary fibre content was higher in the LGI than HGI breakfasts. Although the 3-hour glucose IAUC following the HGI breakfast was significantly higher than following the LGI breakfast (253 ± 30 vs. $156 \pm 22 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{min}$, $p < 0.05$), no difference in the subjective appetite sensations between breakfasts was evident during the period. However a greater fullness rating was found following standard lunch in the LGI than HGI

trials. The authors speculated that the greater LGI post lunch fullness sensation resulted from the residual effect of the high-fibre LGI breakfast.

Kristensen et al. (2010) investigated the effect of fibre content of four isocaloric breakfasts on appetite, glucose responses and subsequent EI three hours postprandially in 16 young males and females with normal BMI ($21.7 \pm 2.2 \text{ kg}\cdot\text{m}^{-2}$). The whole grain bread meal (GI = 105, 11.7g fibre, weighed 181g) was found to have greater AUC for satiety (22%, $p < 0.01$) compared to the refined wheat bread meal (GI = 100, 3.6 g fibre, weighed 170 g); whereas refined wheat pasta meal (GI = 38, 2.2 g fibre, weighed 214 g) and wholemeal pasta meal (GI = 79, 5 g fibre, weighed 227 g) gave scores at level between refined wheat bread meal and whole grain bread meal without significant difference. Although the GI value of refined wheat pasta meal (GI = 38, 2.2 g fibre, weighed 214 g) was low, it did not result in increased satiety compared to the refined wheat bread meal (GI = 100, 3.6 g fibre, weighed 170 g). Increased satiety did not reduce *ad libitum* EI three hours following the wholegrain bread meal compared to the other three test meals. The authors concluded that factors other than GI, such as fibre content and volume of the meal, may be more important to satiety. Besides, the authors explained that the fibre content had no effect on glycaemia when comparing the refined and the whole grain meals. It is too general to correlate the fibre content with the glycaemia. The GI system has already involved the factors affecting the postprandial glucose response (Table 2.2). The GI value of white bread and wholemeal bread are similar (Foster-Powell et al., 2002). Kristensen et al. (2010) focused the study on the effect of dietary fibre; however the type of fibres in the test meals was not differentiated. It is known that viscous fibre slows down the stomach emptying

and decelerates small intestinal transit time leading to an increase in satiety (Kristensen & Jensen, 2011). In an aspect of investigation of the effect of GI, this study appears to have variables including GI value, fibre content, weight or volume to confound the results.

Makris et al. (2011) recruited 12 black, two white and two Hispanic overweight adults aged 37.8 ± 10.5 years to consume breakfast with LGI (GI = 38 and 40) and HGI (GI = 79 and 82); and high (28% for energy) and low (14% for energy) protein in four separate occasions. Focusing on investigating the effects of LGI and HGI low protein breakfasts, no difference was found in the subjective appetite sensation and voluntary lunch intake four hours after breakfasts between trials. It should be aware that the postprandial glucose levels returned to the fasting levels after four hours which seemed to take longer duration than healthy people. Although the inclusion criteria of the recruitment included normal fasting glucose levels, it is still suspected if the postprandial glycaemic responses of overweight participants differed from the individuals with normal BMI. In addition, the voluntary lunch EI (1,250–1,725 kcal) in the study appeared to be much higher than the reference intakes for general population. The food provided at lunch was in a buffet style and thus it was possible that the wide variety of food choices at lunch masked the effects of satiation despite repeated-measure design (Hetherington et al., 2006).

Table 2.4 Summary of the short term effect of low GI foods on appetite and energy intake in adults

References	Participants	Design and Duration*	Intervention and monitoring	Outcomes and comments
Anderson et al. (2002)	14 M: 18-35 yr; BMI 20-25 ($\bar{x} \pm \text{SEM}$)	Each ~ 1 hr	LGI: 75 g fructose-glucose (80/20); HGI: 75g polycose, sucrose, glucose; 200 mL water <i>ad libitum</i> pizza lunch 60 min after tested drinks MO: [Glucose] & appetite rating	60-min glucose AUC: glucose ~ polycose > sucrose > fructose-glucose > sucralose (191 ± 19 vs. 178 ± 19 vs. 132 ± 17 vs. 72 ± 8 vs. 8 ± 2 mmol·L ⁻¹ ·min); significant time x treatment effect ($p < 0.05$) Lunch EI: glucose < sucrose, fructose-glucose & polycose ($p < 0.05$); AS at 60 min correlated with EI ($r = 0.45$, $p < 0.01$); 1-hr glucose AUC correlated with EI ($r = -0.24$, $p < 0.05$)
Arumugam et al. (2008)	14 F: 47.7 ± 5.6 yr; BMI 29.5 ± 2.5 ($\bar{x} \pm \text{SD}$)	Each ~ 9 hr 2-3 wk washout	I: breakfast: 60 g bolus glucose (rapid) vs. 60 g glucose in 8 sips (slow); lunch: 80 g glucose MO: Appetite ratings, blood sample	Post breakfast glucose: rapid > slow (1 hr); slow > rapid (3-4 hr); AS before lunch: rapid > slow; AS 3-5 hr after lunch: rapid > slow [Glucose] & AS correlation: rapid (partial $r^2 = 0.31$, $p = 0.001$); [Insulin] & AS correlation: rapid > slow (partial $r^2 = 0.03$, $p < 0.001$ vs. 0.09, $p < 0.001$)
Stevenson et al. (2009)	8 F: 23.8 ± 7.2 yr; BMI 21.3 ± 1.9 ($\bar{x} \pm \text{SEM}$)	Each ~ 7 hr	I: pre-trial evening meal Breakfast: 1 g CHO kg ⁻¹ BW LGI (GI = 44) & HGI (GI = 78); 3.5 vs. 1.5 g fibre on a 50-kg person; standard lunch MO: regular arterial venous blood sampling, appetite ratings	3-hr glucose IAUC:HGI > LGI (253 ± 30 vs. 156 ± 22 mmol·L ⁻¹ ·min, $p < 0.05$); 3-hr insulin IAUC:HGI > LGI (30,475 ± 4,260 vs. 17,375 ± 3,336 pmol·L ⁻¹ ·min, $p < 0.05$) No difference in AS post breakfast Oxidation during exercise: CHO: LGI < HGI (42.5 ± 4.0 g vs. 51.6 ± 2.7 g, $p < 0.005$); Fat: LGI > HGI (7.4 ± 1.3 vs. 3.7 ± 0.8 g, $p < 0.001$) Lunch: Fullness ratings: LGI > HGI
Kristensen et al. (2010)	6 M 10 F: 24.1 ± 3.8 yr; BMI 21.7 ± 2.2 ($\bar{x} \pm \text{SD}$)	Each 3 hr	I: RWB (GI = 100), WWB (GI = 105), RWP (GI = 38), WWP (GI = 79); contained 50g available CHO MO: Appetite ratings, blood [Glucose]	Fibre intake: RWB (3.6 g), WWB (11.7 g), RWP (2.2 g), WWP (5.0 g) 3-hr glucose AUC: RWB > RWP ($p < 0.01$) & WWB > WWP ($p < 0.01$) Satiety: WWB > RWB ($p < 0.001$) No difference in the subsequent EI

Table 2.4 (Continued)

References	Participants	Design and Duration*	Intervention and monitoring	Outcomes and comments
Makris et al. (2011)	6 M 10 F: 37.8 ± 10.5 yr; BMI 30.9 ± 3.7 (x̄ ± SD)	Each 5 hr	I: high protein LGI, low protein LGI, low protein LGI & low protein HGI (LGI = 38, GL = 17) (HGI = 79, GL = 35) MO: Appetite by VAS, plasma [Glucose], serum [Insulin]	4-hr glucose IAUC: HGI > LGI No difference in hunger and EI ratings No difference in lunch EI

*Randomised crossover. Abbreviations: AS, appetite score; AUC area under curve; BW, body weight; BMI, body mass index in kg·m⁻²; CHO, carbohydrate; EI, energy intake; Lunch EI, lunch energy intake; F, female; FFM, fat free mass; FM, fat mass; [Glucose], glucose concentration; HGL, high glycaemic load; HGI, high glycaemic index; I, intervention method; IGT, impaired glucose tolerant; [Insulin], insulin concentration; LGL, low glycaemic load; LGI, low glycaemic index; M, male; min, minute; RWB, refined wheat bread; RWP, refined wheat pasta; SEM, standard error of mean; SD, standard deviation; MO, monitoring; mth, month; wk, week; WWB, wholegrain bread; WWP, wholegrain pasta; x̄, mean; yr, years.

Besides the above mentioned papers, investigating the short term effect of GI on appetite and / or subsequent EI have been investigated (Anderson et al., 2002; Kristensen et al., 2010; Ludwig et al., 1999; Makris et al., 2011), several studies investigated the effects of GI on glucose tolerance at subsequent meals in Sweden (Liljeberg, Akerberg, & Bjorck, 1999; Liljeberg & Bjorck, 2000; Nilsson, Ostman, Preston, & Bjorck, 2008; Nilsson, Ostman, Granfeldt, & Bjorck, 2008; Rosen, Ostman, & Bjorck, 2011).

Four males and six females aged between 22 and 57 years with normal BMI ($21.6 \pm 1.5 \text{ kg}\cdot\text{m}^{-2}$) consumed eight different breakfasts in separate occasions. A HGI lunch was then consumed four hours post breakfast (Liljeberg et al., 1999). The authors found that the glucose and insulin responses 45 minutes after lunch were lower following the spaghetti breakfast (GI = 52) than the reference wheat bread breakfast (GI = 100). Higher satiety scores were reported 45–95 minutes after lunch following the high-amylose barley bread (HAB) baked for a long time at a low temperature with barley flakes breakfast (GI = 60), 45 minutes after the lunch following the HAB baked for a long time at a low temperature breakfast (GI = 74), and 95 minutes after the lunch following the HAB baked for a long time at a low temperature made with pre-boiled flour breakfast (GI = 85.5) than the reference breakfast. Although the contents of the macronutrient and the dietary fibre of the spaghetti breakfast matched with the reference breakfast, the authors could not explain the lack of improvement of the glucose and insulin tolerance in another LGI breakfast (GI = 64). In addition, the dietary fibre contents of the four HAB breakfasts were much higher than that of the reference wheat bread breakfast (11–25 vs. 2 g) which might be confounding to the satiety score.

Liljeberg and Bjorck (2000) recruited two males and eight females aged between 24 and 51 years with normal BMI ($21.2 \pm 1.4 \text{ kg}\cdot\text{m}^{-2}$) consumed the spaghetti breakfast (GI = 52) and the above reference breakfast (GI = 100) in two separate occasions. The above mentioned HGI lunch was consumed four hours post breakfast. The blood [Glucose] and insulin concentration [Insulin] just before lunch were significantly lower after the spaghetti than the reference breakfasts. Similarly the responses of the glucose 45–95 minutes and insulin 30–45 minutes after lunch were lower following the spaghetti breakfast (GI = 52) than the reference wheat bread breakfast (GI = 100).

Nilsson, Ostman, Granfeldt, et al. (2008) investigated the second meal effect by recruiting seven males and five females aged 28.3 ± 5.1 years with normal BMI ($22.1 \pm 2 \text{ kg}\cdot\text{m}^{-2}$) to consume breakfasts with different dietary fibre contents, weights and GI values at seven separate occasions. The barley and rye kernel breakfasts resulted in lower blood glucose peak increments than that of the reference wheat bread breakfast (1.5 ± 0.2 and 1.6 ± 0.2 vs. $3.2 \pm 0.4 \text{ mmol}\cdot\text{L}^{-1}$, $p < 0.001$). The blood glucose increment 30–60 minutes after the standardised HGI lunch following the barley kernel breakfast was also lower than the reference breakfast ($p < 0.05$). The authors found that a significant but weak positive association between the two-hour blood glucose IAUC after the standardised lunch and that after the test breakfasts ($r = 0.3$, $p < 0.05$). The second series provided the test evening foods followed a standardised HGI breakfast. The glucose increment 45 minutes after the standardised breakfast following the barley kernel evening meal was lower than that of WWB evening meal.

Another similar study with 10 males and 10 females aged between 19 and 30 years with normal BMI ($22.1 \pm 2 \text{ kg}\cdot\text{m}^{-2}$) found that the two-hour glucose IAUC and three-hour glucose total AUC after the standardised HGI breakfast following spaghetti enriched with double amount of barley extracted dietary fibre evening meal (GI = 58) were lower than following the barley porridge evening meal, but not following the reference wheat bread evening meal (GI = 100) (Nilsson, Ostman, Preston, et al., 2008). No difference in the fasting glucose level was found. However, the three-hour post breakfast glucose total AUC below the fasting level following the spaghetti evening meal was significantly larger than following the reference evening meal ($p < 0.05$). No second meal effect from the spaghetti enriched with barley extracted dietary fibre evening meal here appeared to contradict the finding of another study by the same authors which provided barley kernel evening meal (Nilsson, Ostman, Granfeldt, et al., 2008). Nilsson, Ostman, Preston, et al. (2008) suspected that the LGI features of the spaghetti evening meal was not sufficient to elicit a benefit on overnight improvement of glucose tolerance after HGI breakfast.

Five males and five females aged 26 ± 1.1 years with normal BMI ($22.6 \pm 0.4 \text{ kg}\cdot\text{m}^{-2}$) consumed a standardised pasta lunch 270 minutes following 50 g available CHO test breakfasts on seven different occasions (Rosen et al., 2011). All test breakfasts differed in portion, protein and dietary fibre contents. All rye containing breakfasts (GI = 64 to 79) and wholegrain wheat kernels breakfast (GI = 68) induced lower insulinaemic indices than the reference wheat bread breakfast (GI = 100) ($p < 0.05$). The GI values of the breakfast were significantly associated with the early post breakfast (60-min AUC) feeling of fullness ($r = -0.48, p < 0.001$), hunger ($r = 0.44, p < 0.001$) and desire to eat ($r =$

0.39, $p < 0.001$); but not the lunch EI. In addition, the FFA level 180–270 minutes after rye kernel breakfast was lower than that following the reference breakfast. The rye kernel breakfast also induced lower voluntary EI with 16% than the reference breakfast ($p < 0.05$). However, it should be noted that the portion size and the content of indigestible CHO (dietary fibre and resistant starch) in the rye kernel breakfast were nearly double (227 vs. 124 g) and six fold higher (25.2 vs. 4.4 g) than the wheat bread breakfast respectively. The lunch EI was significantly associated with water content ($r = -0.36$, $p = 0.002$), portion size ($r = -0.36$, $p = 0.002$) and indigestible CHO ($r = -0.34$, $p = 0.005$) of the breakfast.

Several reviews have summarised the effects of GI on appetite, EI or weight control in the last few decades (Bornet, Jardy-Gennetier, Jacquet, & Stowell, 2007; Brand-Miller et al., 2002; Ludwig, 2000; Raben, 2002; Roberts, 2000; van Dam & Seidell, 2007). Roberts (2000) reviewed 20 short-term studies published between 1977 and 1999, and found that the LGI foods or drinks promoted post meal satiety in 12 out of 20 studies, of which one study was published in 1977 when the concept of the GI system had not been developed. The author concluded that the effects of LGI and HGI meals on post meal satiety and satiation were equivocal.

Ludwig (2000) reviewed 16 studies regarding glycaemic or insulinaemic response with changes in hunger, satiety and EI published between 1977 and 1999. Some modified dietary factors of the 16 studies included the manipulation of the dietary fibre content. The author concluded that an increase

in satiety and an decrease in hunger or reduction of voluntary EI after LGI foods compared with HGI foods in 16 studies, consistent to the previous review (Roberts, 2000). It should be noted that 11 out of 16 studies reviewed by Ludwig (2000) were adapted from Roberts (2000).

Brand-Miller et al. (2002) reviewed that HGI diets affected appetite and fat storage due to high postprandial insulin responses leading to the shift of fuel partitioning. The authors suggested that LGI might have an influence on shifting the oxidation for energy from CHO towards fat. Raben (2002) systematically reviewed 31 studies shorter than a day regarding the effect of GI on appetite or subsequent EI and summarised that 15 studies supported the consumption of LGI foods for either lessening hunger or decreasing food intake. Despite that, only 7 out of 15 studies achieved significant reductions of *ad libitum* food intake.

In line with the reviews, many of these studies (Table 2.3 and 2.4) did not control confounding variables such as macronutrient composition, energy density and dietary fibre. The inconsistent results between LGI and HGI CHO intakes may have resulted from the differences in the study design such as the post meal time point of the measurements of appetite sensation. Bornet et al. (2007) reviewed and suggested that HGI foods have higher satiating effect within acute postprandial phase where the appetite suppressive effect of LGI foods might last up to six hours. Brindal et al. (2012) criticised the use of the studies providing different macronutrient compositions (Ball et al., 2003; Ludwig et al., 1999) which found reduced feelings of hunger following LGI meals in

overweight children and adolescents compared to HGI meals. Many longer-term studies employed overweight or obese participants that many of whom were glucose intolerant and / or hypercholesterolaemic.

The effects of GI on weight management in the longer term appear to be disappointing. The USDA Evidence Analysis Library (2012) stated there is a strong and consistent evidence that GI and / or GL are not associated with better weight management. The discrepancy may be due to the differences in the study design. Blundell et al. (2010) stated that errors in the data collection in the free-living conditions are high. Longer term investigation of the GI and appetite related studies in the field are prone to bias. Although the tightly controlled laboratory condition can reduce confounding variables to food intake, the author agreed that the laboratory studies cannot replace the field studies and is worthy to have both research approaches.

Appetite sensation, biochemical parameters and EI responses might vary across different sample groups. For instance, physical activity levels (PAL) of participants were not described (Anderson et al., 2002; Ludwig et al., 1999). Postprandial levels of free fatty acid (FFA), glucose and insulin responses differ between the lean and the obese; as well as the lean and the impaired glucose tolerant overweight (Wolever & Mehling, 2003). Differences were also evident in the perceived appetite sensation between obese and non-obese (Cardello, Schutz, Leshner, & Merrill, 2005). Arumugam et al. (2008) also agreed that the postprandial peak [Glucose] of the postmenopausal, overweight and obese women differed from the non-obese men. It has been suggested that physical

activity, and other factors such as age, gender, body composition and environmental conditions effect appetite sensation (Stensel, 2010). The investigation of effects of GI should therefore be restricted to a shorter study period with adequate control of confounding variables. To date, the short term effect of GI on appetite and subsequent EI has not been widely investigated in athletic or recreationally active adults.

2.6.3 Glycaemic index and cognitive performance

There is a strong link between nutrition and cognitive performance. Of which glucose is the primary energy fuel to brain for cognitive activity. Although the mass of a human brain is only around 2% of the total body mass, the energy requirement of the brain can reach 20–30% of the whole body at rest (Benton, Parker, & Donohoe, 1996). However, the storage capacity of glucose in brain is very limited and the brain glucose can be depleted within 10 minutes without supply (Benton & Parker, 1998). Participants having lower blood [Glucose] reported to have greater level of tension during short term task than whom having higher blood [Glucose] (Benton & Owens, 1993). An impairment of the cognitive performance has been demonstrated on healthy participants after insulin induced hypoglycaemia (McCrimmon, Deary, Huntly, MacLeod, & Frier, 1996). In addition, fluctuations of blood glucose level, even within normative range, may also exert influences on cognitive performances (Fairclough & Houston, 2004).

Skipping breakfast has been known to be associated with higher levels of self-reported stress, more depression and anxiety than regular breakfast taking in

childhood or adolescence (Murphy, 2007). Comparatively less number of studies investigated the effect of having breakfast on appetite and cognition in adults (Lluch, Hubert, King, & Blundell, 2000; A. P. Smith, 2003). Having a breakfast eating habit does not imply a healthier eating habit. There are arguments surrounding the consumption of more natural and more highly processed foods at breakfast and the health consequences (Murphy, 2007). The current thesis focused on the impact of CHO intake and cognition. The effect of CHO consumption on cognitive performance in healthy adults is summarised in Table 2.5.

Eighty undergraduate females participated in one of the four breakfast and glucose conditions to investigate the intakes on a trigram test (Benton & Parker, 1998). The authors concluded that a glucose drink could nullify the negative effect of breakfast skipping in the trigram recall test, whereas an additional glucose intake following a breakfast did not have an additional effect. Another study in the same paper recruited 47 male and 137 female young adults for one of the four breakfast and glucose conditions to investigate the intakes on three cognitive tasks. The authors found that participants having breakfast recalled more words and more of the Wechsler story (intelligence test) than the fasting participants. A glucose drink had no additional effect on breakfast eaters on the word recall test and had no effect on fasting participants and breakfast eaters on Wechsler story recall. The authors concluded that mechanisms other than glucose supply affected certain cognitive abilities. One possible reason could be due to the time lag between the breakfast and the glucose drink intakes. Also the protocol of having breakfast intake at participants' home did not control for the nutrient contents. Another study with 17 males and 16 females with a

mean age of 21.3 years found that the [Glucose] was negatively associated with the length of response time (RT) and number of errors in the Spatial memory test which indicated that lower [Glucose] was related to increasing RT and number of error in a memory task (Benton & Parker, 1998).

Table 2.5 The effects of carbohydrate consumption on cognitive performances

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Benton and Parker (1998)	47 M 137 F; 22 yr	Randomised double blind ~ 1.5 hr Test started 20 min pp	1. 50g glucose drink + CHO BF (n = 28); 2. BF + placebo drink (n = 25); 3. 50g glucose drink (n = 12); 4. placebo drink (n = 15) 70-min tests: Word list test, Wechsler story test, abstract reasoning test	Word recall: group 3 > group 4 ($p < 0.01$); group 2 > group 4 ($p < 0.01$); no difference between group 1 & 2 Story recall: breakfast > fasted ($p < 0.02$) No difference in abstract reasoning scores
Benton and Parker (1998)	80 F; 22.6 yr ($\bar{x} \pm SD$)	Randomised double blind 8 trials x 18s Test started 20 min pp	4 trials: BF: 42.6 ± 30.3 g CHO 1. 50g glucose drink + CHO BF (n = 55); 2. BF + placebo drink (n = 51); 3. 50g glucose drink (n = 38); 4. placebo drink (n = 40); Tests: Brown-Peterson task	No difference among trials Improvement in group 3 ($p < 0.001$) Low recall in group 2
Benton and Parker (1998)	17 M 16 F; 21.3 yr	Repeated measures 2 hr x 2	37.7g CHO habitual breakfast vs. fast Tests: Spatial memory test, word list test	Spatial memory test: Negative correlation between [Glucose] & length of time ($r = -0.48$, $p < 0.004$), and error numbers ($r = -0.42$, $p < 0.01$)
Donohoe and Benton (1999)	67 F; 21.8 ± 5.1 yr	Randomised double blind ~ 4 hr x 2 Tests started 20 min pp	Habitual breakfast + 50 glucose solution vs. placebo [Glucose] Tests: Water jars test, embedded figures test, logical reasoning test	No difference in all test performances Water jars test time: Higher [Glucose] group < lower [Glucose] group ($p < 0.02$)
Donohoe and Benton (1999)	69 F; 20.2 ± 2.1 yr	Randomised double blind ~ 4 hr x 2 Tests started 20 min pp; one verbal frequency test started after the drink	Habitual breakfast, + 50 glucose solution vs. placebo [Glucose] Tests: Porteus Maze, block design test, verbal frequency test	Number of verbal frequency: glucose group > placebo ($p < 0.001$) Block design test-difficult design time: [Glucose] drop group < [Glucose] rise drop ($p < 0.02$)

Abbreviations: BMI, body mass index in $\text{kg}\cdot\text{m}^{-2}$; CHO, carbohydrate; F, female; [Glucose], blood glucose concentration; hr, hour; M, males; min, minutes; pp, postprandial; SEM, standard error of mean; SD, standard deviation; \bar{x} , mean; yr, years.

Recently, more research has investigated the effect of CHO consumption on cognitive performance on the young populations at different ranges of age than addressing the effects of GI on cognitive functions (Cooper et al., 2012; Ingwersen et al., 2007; Micha et al., 2010). Some studies have investigated the effects of GI or GL at breakfast on cognitive performance since millennium (Table 2.6).

Ingwersen et al. (2007) found that the LGI breakfast prevented the decline of the attention and memory of 26 boys and 38 girls aged 6.8–11.7 years. It should be noted that the two trials were arranged on two consecutive days without a washout period. The authors did not mention if the breakfasts were provided in a randomised order. Furthermore, the LGI breakfast contained higher protein, fat but lower CHO and energy contents than the HGI breakfast in the study. Protein has been found to increase alertness and lead to better attention and efficiency of cognitive tasks (Fischer, Colombani, Langhans, & Wenk, 2001).

Micha et al. (2010) divided 24 boys and 36 girls with a mean age of 13 ± 0.1 years into four groups (LGI-LGL, LGI-HGL, HGI-LGL and HGI-HGL) to investigate the effects of GI and GL on cognitive performances in an inter-subject comparison study. The children took breakfast at home. The authors found positive associations between the HGI breakfast and the immediate recall test reflecting short term memory ($p = 0.045$); the LGI breakfast and the matrices performance reflecting inductive reasoning ($p = 0.012$); the LGL breakfast and the speed of information processing reflecting vigilance ($p =$

0.031) and sustained attention ($p = 0.001$); and the HGI breakfast and the serial sevens performance reflecting vigilance ($p = 0.009$) and working memory ($p = 0.055$). Serial sevens and speed of information processing were considered to be the most mentally demanding of tasks. The LGI breakfast was associated with improving vigilance tasks but poorer memory in the study. The results appeared not to be consistent with the previous studies that LGI food was associated with better memory performance (Ingwersen et al., 2007; Nilsson et al., 2009; Papanikolaou et al., 2006). The study possessed a number of methodological weaknesses. The start time of the tests differed among children and groups; as it depended on the snack times of children. The authors manipulated the GI and GL values by adjusting the CHO contents. The LGI-HGI breakfast provided the highest amount of CHO content compared to the other three breakfasts and the energy content was not matched. Additionally, the four breakfasts had a trend to differ in protein content ($p = 0.053$) which was the same limitation as Ingwersen et al. (2007).

Cooper et al. (2012) found that the LGI breakfast providing $1.5 \text{ g CHO kg}^{-1} \text{ BM}$ enhanced the response time and the accuracy at the late postprandial period than following the HGI breakfast on a cognitively demanding task in 18 boys and 23 girls with a mean age of 12.8 ± 0.4 years. Although the RT of the Stroop task were quicker on the HGI trial when compared to the LGI trial; the numbers of response accuracy on the HGI trial were lower than on the LGI trial which reflected a speed-accuracy trade off.

Several GI related studies on cognition employed the middle to old age adults. Three breakfasts with different GI and a placebo were provided in separate occasions to 10 males and 10 females with a mean age of 72.3 ± 1.4 years to investigate the effect of GI on cognitive performances in the elderly (Kaplan, Greenwood, Winocur, & Wolever, 2000). The authors commented that LGI foods had a stronger relation baseline performance and memory improvement despite the lack of significant difference in cognitive performance between trials.

There appears to have been less research on adults with glucose intolerance when compared to on the children and adolescents. The cognitive performance following LGI and HGI breakfasts providing 50 g available CHO was assessed in 10 male and 11 female type 2 diabetes aged 65 ± 7.3 years (Papanikolaou et al., 2006). Cognitive performances of various tasks generally were better following the LGI breakfast compared to the HGI breakfast, although no differences were found in sustained attention. Independently to the GI values, glucose AUC was negatively associated to the memory performance. Higher levels of glycosylated haemoglobin were associated with poorer delayed memory recall on paragraph recall test which indicated that poorer glucose regulation was associated with poorer cognitive performances in type 2 diabetic elderly.

Nilsson et al. (2009) investigated the effects of a bolus consumption of 50 g glucose drink and a regular sipping of 8.3 g glucose drink every 30 minutes on the cognitive performance on 20 males and 20 females with normal BMI ($23.8 \pm 2.7 \text{ kg}\cdot\text{m}^{-2}$) and a mean age of 59 years. The bolus drink was considered as

the HGI breakfast whilst the small and regular sip provided more gentle postprandial glucose response simulated the glucose response as following LGI breakfast. The authors further sub-divided the participants into groups with lower and higher glucose tolerance. Working memory test at 90 minutes and selective attention test at 170 minutes post breakfast were performed better in the LGI than the HGI trial. High blood glucose increments at the early postprandial period of the LGI trial were negatively associated with the working memory performances. Furthermore, participants with higher glucose tolerance performed the cognitive tests better than the lower group. The authors concluded that the efficiency of glucose tolerance might influence the cognitive performance in healthy adults.

A recent study compared the effects of bread with low (guar gum enriched white bread) and high (white bread) postprandial glucose responses on cognitive performances on 49–71 year old adults (Nilsson et al., 2012). Similar results were found that the LGI food led to better overall selective attention performance than the HGI food, particularly in the later postprandial period at 120 minutes post breakfast.

Lamport et al. (2012) investigated the effects of GL at breakfast on cognitive performance on four male and six female obese participants with normal glucose tolerance (aged 56.2 ± 2 years); and 12 male and 12 female type 2 diabetic obese participants (aged 61 ± 1.9 years). Eight cognitive tests were measured. Normal glucose tolerant participants generally had better cognitive performance in most of the tasks than the diabetics. Only the time of

psychomotor skill test was found to be shorter in the LGL than the HGL trials. It should be noted that although the energy content and the food weight between breakfasts were matched, the CHO content of the LGL breakfast was only half of that of the HGL glucose drink. In addition, protein and fat were provided only in the LGL breakfast which might also be a pitfall in the study design confounded from the other macronutrients on cognitive performances and efficiency (Fischer et al., 2001).

The improvements observed in one population group might not be applied to another population group. Sensitivity to the benefit of glucose might also differ between genders. It was suggested that memory was improved when a task started 15–20 minutes after ingestion of ~ 50 g glucose where blood [Glucose] peaked at 8–10 mmol·L⁻¹ (Kaplan et al., 2000). It is anticipated there exists an optimum time for performing cognitive tasks to enhance certain cognitive performances following CHO containing breakfast. There occurs a rebound hypoglycaemia 30–60 minutes following CHO ingestion. The decline in glucose level to reach hypoglycaemia was considered to impair cognitive performance (Dye & Blundell, 2002). Jeukendrup and Killer (2010) pointed that the hypoglycaemic symptoms are most likely related to a reduction of the glucose delivery to the brain, which might affect cognitive performance when cognitive task performed during rebound hypoglycaemia. Thus, the time of performing cognitive tasks should be taken in the consideration of the postprandial glucose profile.

A recent review by Gilsenan et al. (2009) critically evaluated eight studies regarding the association between CHO containing meals with different GL, rather than pure glucose drinks, and cognitive performance. Those studies used either a within-subject design for children, elderly and diabetic elderly or a between-subject design for adults. In addition, physical activity level (PAL) and any control of the pre-trial evening meals had not been reported. The authors suggested that consistent methodologies and detailed techniques of food interventions should be employed (Gilsenan et al., 2009).

Table 2.6 The effects of glycaemic index on cognitive performances in different populations

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Kaplan et al. (2000)	Elderly 10 M 10 F; 72.3 ± 1.4 yr; BMI 25.1 ± 0.9 ($\bar{x} \pm SEM$)	Randomised Counter-balanced 105 min x 4 Tests started before breakfast Tests started 15 min pp	4 Trials: 50 g glucose (GI = 142), barley (GI = 36) & potato mash (GI = 118) with 50 g available CHO; & placebo (GI = 0) Capillary [Glucose], [Insulin] Tests at 15, 60 & 120 min pp: Verbal memory tasks: word list recall Visuomotor test, Immediate & delay paragraph recalls	Glucose peaked at 60 min No difference in overall cognitive performance among CHO trials Barley: improvement of delayed recall & total recall at 15 min correlated with glucose IAUC ($p < 0.04$)
Benton, Slater, and Donohoe (2001)	Young adults 150 F; 21.3 yr	Randomised Non crossover 4.5 hr Test started 15-20 min pp	Breakfast trials (n = 25 each): 1. fast; 2. 10 g CHO; 3. 50 g CHO Snacks 1.5 hr pp breakfast: 1. 10 g CHO; 2. 50 g CHO Mood by VAS Capillary [Glucose] Tests: Word recall memory test	Significant snack meal x time interaction on [Glucose]; breakfast x snack interaction on hunger 50 g CHO: poorer mood later Low [Glucose] associated with better mood Snack increased elated feeling in 50 g CHO breakfast ($p < 0.04$) Snack improved immediate memory
Papanikolaou et al. (2006)	Elderly T2DM: 10 M 11 F; 65 ± 7.3 yr; BMI 29.3 ± 5.0 ($\bar{x} \pm SD$)	Randomised Repeated measures ~ 2.5 hr x 3 Tests started 15 min pp	LGI (pasta) & HGI (bread) BF: 50g CHO, water as placebo Finger [Glucose] Tests: Word list, paragraph recall, verbal paired associates, digit span forward, trial-making test, test of everyday attention	Glucose AUC: HGI > LGI ($p = 0.035$) Word list & paragraph recalls: LGI > HGI ($p = 0.002$ & $p = 0.001$) Negative association between low glucose AUC and 30-min delay paragraph memory in both trials ($R^2 = 0.34$, $p = 0.01$); digit span forward: LGI > HGI ($p = 0.045$); trial-making test performance: LGI > HGI ($p = 0.006$)
Ingwersen et al. (2007)	Children: 26 M, BMI 17.1; 38 F, BMI 17.9; 9.3 yr (6.8-11.7 yr) ($\bar{x} \pm SEM$)	Counter-balanced ~ 3 hr x 2 Tests started 30 min before breakfast & then 10 min pp	LGI (all bran) & HGI (coco pops) cereal: 35g + 125 mL milk Tests: 25-min 11 attention & memory tests before breakfast, 10, 70, 130 min pp	Higher CHO in HGI (GI = 77) than LGI (GI = 42) cereals (29.8 vs. 16.1 g CHO) Trial x time interaction of accuracy of attention ($p = 0.026$); decline in performance HGI > LGI at 130 min pp ($p = 0.021$); secondary memory performance: LGI > HGI ($p = 0.02$)

Table 2.6 (Continued)

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Nilsson et al. (2009)	Middle to old-aged adults: 20 M 20 F; 59 yr (49-70 yr); BMI 23.8 ± 2.7 19 completed WM test at 120 min at LGI	Crossover Counter-balanced Repeated measure ~ 3.5 hr x 2 Tests started 35 min pp	LGI breakfast: 8.3 g glucose every 30 min HGI breakfast: 50 g glucose in bolus Standard pre-trial evening meal (bread) Finger [Glucose] Tests: 4 WM test (35, 90, 120, 150 min pp), SA test (170 min pp)	Glucose AUC: HGI > LGI (0-90 min, $p < 0.001$); LGI > HGI (90-180 min, $p < 0.001$) WM test performance: LGI > HGI at 90 min pp ($p < 0.05$); SA test performance: LGI > HGI ($p < 0.05$), second trial > first trial
Micha et al. (2010)	Adolescents: 26 M 36 F; 13 ± 0.1 yr; BMI 20.7 ± 0.6 ($\bar{x} \pm SEM$)	Inter-subject comparison Non crossover	Habitual breakfasts: LGI-HGI (n = 11): GI = 53, GL = 43; HGI-HGL (n = 19): GI = 68, GL = 44; LGI-LGL (n = 19): GI = 58, GL = 31; HGI-LGL (n = 11): GI = 64, GL = 23 Finger [Glucose] 7 cognitive tasks	[Glucose] before the tests: HGL groups > LGL groups ($p = 0.025$) Positive associations with [Glucose]: HGI & immediate recall ($p = 0.045$); HGL & matrices ($p = 0.012$); LGI, HGL & speed of information processing ($p = 0.031$ & $p = 0.001$); LGI, HGL & serial sevens ($p = 0.009$ & $p = 0.055$)
Cooper et al. (2012)	Adolescents: 18 M 23F; 12.8 ± 0.4 yr (12-14 yr); BMI 20.5 ± 3.3 ($\bar{x} \pm SD$)	Randomised Cross-over Order balanced ~ 2.5 hr x 3 Test started 30 & 120 min pp	LGI (GI=48, GL=36) & HGI (GI=72, GL=54) breakfasts: 1.5g CHO / kg BM; placebo Capillary [Glucose], [Insulin] Tests: 15-min Stroop task, Stenberg paradigm & Flanker task at 30 & 120 min pp	[Glucose] over the time & glucose AUC: HGI > LGI ($p < 0.05$) Stroop response time, accuracy, decline in accuracy: HGI < LGI ($p = 0.031$), LGI > HGI ($p = 0.039$), HGI > LGI ($p = 0.033$) Stenberg paradigm complex level response: LGI > HGI ($p = 0.002$) Flanker complex level accuracy: LGI > HGI ($p = 0.014$)

Table 2.6 (Continued)

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Lamport et al. (2012)	Middle to old aged adults versus old aged T2DM: 4 M 6 F NGT; 56.2 ± 2 yr; BMI 31.4 ± 2.6 T2DM: 12 M 12 F; 61 ± 1.9 yr; BMI 34.8 ± 1.3 ($\bar{x} \pm SD$)	Randomised Cross-over ~ 3 hr x 3 Tests started 30 min pp	LGL (GI = 32, GL = 12, 37.3 g CHO) and HGL (GI = 95, GL = 71, 75 g CHO) breakfast & water Standard pre-trial evening meal Finger [Glucose], perceived stress scale Tests: Visual spatial learning test, visual verbal learning test, Corsi block tapping test, Tower of Hanoi, Grooved Peg board, psychomotor test, source monitoring test, paragraph recall	LGL breakfast contained protein and fat. Half CHO content in LGL than HGL breakfasts 2 hr pp glucose level: HGL > LGL in NGT ($p < 0.05$) and in T2DM ($p < 0.05$) Psychomotor skill time: LGL < HGL ($p < 0.05$)
Nilsson et al. (2012)	Old-aged adults 12 M 28 F 62.9 ± 5 yr ($\bar{x} \pm SD$)	Randomised cross-over ~ 3 hr x 3 Tests started 30 min pp	LGI (WWB, GI = 45) & HGI (G-WWB, GI = 100), 50g available starch Standard pre-trial evening meal Finger [Glucose] Tests: ~ 8 min WM test (90, 135, 180, 225 min pp); ~ 10 min SA test (72, 120, 165, 210 min pp)	SA test performance: LGI > HGI at 120 min ($p < 0.01$) Main trial effect of WM test 75-225 min: LGI > HGI ($p < 0.01$)

Abbreviations: AUC, area under the curve; BMI, body mass index in $\text{kg}\cdot\text{m}^{-2}$; CHO, carbohydrate; F, female; [Glucose], blood glucose concentration; G-WWB, guar gum enriched white wheat bread; HGI, high glycaemic index; HGL, high glycaemic load; [Insulin], insulin concentration; LGI, low glycaemic index; LGL, low glycaemic load; hr, hour; M, males; min, minutes; NGT, normal glucose tolerance; pp, postprandial; SA, selective attention; SEM, standard error of mean; SD, standard deviation; T2DM, type 2 diabetes VAS, visual analogue scale;; WM, working memory; WWB, white wheat bread, \bar{x} , mean; yr, years.

Besides cognition, glucose profile has been reported to affect mood state. Benton et al. (2001) suspected that the falling [Glucose] back to baseline was associated with poorer mood state. Dye and Blundell (2002) also stated that CHO ingestion was associated with increasing feeling of fatigue. Pasman, Blokdijk, Bertina, Hopman, and Hendriks (2003) investigated whether lower perception of fatigue, which is one of the mood states; and higher degree of satiety were obtained after the consumption of complex CHO breakfast when compared to simple CHO breakfast in 26 middle-aged males with BMI. Previously it was believed that simple CHO produced greater increases in the postprandial [Glucose] than complex CHO. It has been known that the terms simple sugar and complex CHO do not reflect the bioavailability of CHO after consumption (Jenkins et al., 1981). No difference was found between the complex and simple CHO breakfast on postprandial blood glucose and insulin levels in the study by (Pasman et al., 2003), however the authors still postulated that consumption of complex CHO was favourable to the mood states and satiety.

Athletes were found to have vigour scores higher than and scores for tension, depression, anger, fatigue; and confusion lower than the population norms (Morgan, 1980, 1985). To my knowledge no study has used within-subject design, to investigate the effect of CHO with different glycaemic responses on mood state and cognitive performance in recreationally active males.

2.6.4 Glycaemic index and exercise

Although the maintenance of an adequate supply of CHO for muscle and the central nervous system is important for reducing the risk of injury, optimising exercise and potentially psychological performance during exercise, the difference in bioavailability of CHO following foods with different GI taken prior to an exercise event should be of concern. Hypoglycaemia is known to cause deterioration in cognitive performance in healthy participants (McCrimmon et al., 1996); whilst acute hyperglycaemia was reported to impair cognitive performance in type 2 diabetic adults (Sommerfield, Deary, & Frier, 2004). Moreover, fluctuations of blood glucose level, even within normative range, may also exert influences on cognitive performances (Fairclough & Houston, 2004).

The effects of GI of a pre-exercise meal taken on endurance exercise performance have been investigated since 1990's (Donaldson et al., 2010). Thomas, Brotherhood, and Brand (1991) were the first authors to compare the effects of LGI and HGI CHO on biochemical and physiological responses as well as on endurance exercise performance. The consumption of LGI CHO prior to the endurance exercise was found to increase exercise time and provide higher concentrations of plasma fuels towards the end of exercise. However, some studies did not support the improvement of the endurance exercise performance or the change of substrate metabolism from CHO towards fat by LGI trials (Febbraio & Stewart, 1996; Hulton et al., 2012; Little et al., 2010) A recent review summarised that pre-exercise LGI diets shifted the rates of oxidation from CHO towards fat during subsequent prolonged exercise (Donaldson et al., 2010).

Many athletes such as rugby and soccer players perform intermittent and high intensity exercise modes. Intermittent exercise seems to be performed with more cognitive functions like quick decision making matching with the physical activities like motor skill than endurance exercise. The occurrence of transient hypoglycaemia during the early stage of the onset of exercise potentially affects mood, cognition and perceptual skill performance during exercise. However, limited research investigated the effects of CHO on cognitive and intermittent exercise performances (Welsh et al., 2002; Winnick et al., 2005) (Table 2.7).

Welsh et al. (2002) recruited 5 males and 5 females aged 24.3 ± 3.8 years to investigate the effect of intermittent supply of CHO solution versus placebo on intermittent running and cognitive performance. Despite differences in the levels of glucose, insulin and FFA, no significant difference were found in the Stroop Colour Word Task (SCWT) scores of various components including colour, word, colour-word and interference between trials. Nevertheless, lower (better) motor skill score and faster RT were found at the fourth quarter of exercise following CHO consumption than the placebo. With the CHO supply, participants ran longer time to fatigue and reported less fatigue than the placebo trial.

Another similar study recruited 10 males and 10 females aged 23.9 ± 2 years investigating the effect of CHO consumption prior to and throughout an exercise session on the high-intensity intermittent running and cognitive performances (Winnick et al., 2005). Four speeds on a treadmill ranging between 30% $\dot{V}O_2\text{max}$ and 120% $\dot{V}O_2\text{max}$ as well as vertical jump were designed. Faster

sprint time, higher vertical jump, greater force output and better mood were achieved at the last quarter of the exercise in the CHO than the placebo trials. These studies provided glucose drinks for investigating the impact of CHO on cognitive performance throughout intermittent exercise (Welsh et al., 2002; Winnick et al., 2005). However glucose drinks cannot represent the real life condition. Stevenson, Williams, McComb, et al. (2005) identified that the majority of the previous studies investigating the effect of GI via single foods. Often it is not practical to take single foods alone as main meals on a daily basis in a free living condition.

Less research has evaluated the effect of GI on high-intensity intermittent exercise performance (Cocate et al., 2011; Hulton et al., 2012; Little et al., 2010) (Table 2.7). No significant difference was found in the 90-min sprint performance in 16 males aged 22.8 ± 3.2 years with peak oxygen uptake ($\dot{V}O_{2\text{peak}}$) of $55.4 \pm 4.3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ following LGI and HGI breakfasts providing $1.5 \text{ g CHO kg}^{-1} \text{ BM}$ two hours prior to the intermittent running exercise; whilst both trials improved the sprint performance than in the fast control (Little et al., 2010). Both trials had lower fat oxidation than the control in different times throughout the running. The running protocol was composed of two 45-min sessions separated by a 15-min break; therefore the exercise protocol should be considered as endurance exercise.

Cocate et al. (2011) provided LGI and HGI meals two times per day for four days to 15 athletic males aged 24.4 ± 3.8 years in a crossover study with a washout period of seven days. Ninety minutes after the test breakfasts,

participants completed the cycling race equivalent to 85–95% theoretical maximal heart rate (HR_{max}) for 30 minutes on day one and day five. No significant difference was found in the biochemical variables and the substrates oxidation during exercise between trials. However, the HGI breakfast favoured fat oxidation and while the LGI breakfast led to higher CHO oxidation postprandially prior to the exercise. The findings contrasted to the previous study that HGI foods resulted in higher CHO and lower fat oxidation (Stevenson et al., 2009; Stevenson, Williams, Nute, Humphrey, & Witard, 2008).

The most recent study investigated the effect of LGI and HGI meals providing 2 g CHO kg⁻¹ BM being consumed 3.5 hours prior to a 90-min intermittent running event on exercise performance by eight young male adults (Hulton et al., 2012). The shift of substrates oxidation from CHO towards fat during the intermittent high intensity exercise was not found at the LGI trial compared to the HGI trial. Similar to the previous studies (Cocate et al., 2011; Little et al., 2010), the authors did not find any advantage of pre-exercise LGI meal on the intermittent endurance exercise performance.

Besides the prominent result of the advantage of pre-exercise CHO consumption for improving intermittent exercise performance, the choice of CHO should be considered. It is widely known that different types of CHO lead to different postprandial glycaemic and insulin responses (Jenkins et al., 1981); as well as the shifts of substrate oxidation (Stevenson, Williams, & Nute, 2005; Stevenson et al., 2008). To my knowledge the effects of CHO mixed meals with

different GI on cognitive performance and intermittent exercise performance have not been investigated.

Table 2.7 The effects of carbohydrate consumption or glycaemic index on intermittent exercise and cognitive performances

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Welsh et al. (2002)	5 M 5 F: 24.3 ± 3.8 yr 66.4 ± 8 kg; 171.2 ± 8.6 cm; $\dot{V}O_2\text{max}$ 50.1 ± 3.4 mL·kg ⁻¹ ·min ⁻¹ (\bar{x} ± SEM)	~ 3 hr x 2 Ex: 15 min x 4 HIT quarters + vertical jump + run to fatigue 20 min breakfast after 2 nd quarter	CHO: 6% CHO in 5 mL·kg ⁻¹ BM 1.5 hr before Ex; 6% CHO in 3 mL·kg ⁻¹ BM before the next quarter and run to fatigue; 18% CHO in 5 mL·kg ⁻¹ after 2 nd quarter. 127.5 ± 4.9 g total CHO, water Blood [Glucose], [Insulin], [Lactate], [FFA]; HR Tests: POMS, SCWT, MS-test	Shuttle runt to fatigue time: CHO > placebo (3.58 ± 0.47 vs. 2.61 ± 0.42 min, $p < 0.001$) Fatigue: CHO < placebo [Glucose], [Insulin]: CHO > placebo FFA: CHO < placebo MS-test score & time: CHO < placebo ($p = 0.02$ & $p = 0.0002$); no difference in SCWT components
Winnick et al. (2005)	10 M 10 F: 23.9 ± 2.0 yr; 68 ± 11 kg; 170.9 ± 10.1 cm; $\dot{V}O_2\text{max}$ 51.3 ± 4 mL·kg ⁻¹ ·min ⁻¹ (\bar{x} ± SD)	~ 3 hr x 2 Ex: 15 min x 4 HIT quarters + vertical jump + run to fatigue 20 min breakfast after 2 nd quarter	CHO: 6% CHO in 5 mL·kg ⁻¹ BM before Ex and after the 2 nd quarter; 6% CHO in 3 mL·kg ⁻¹ BM before the next quarter 41 g·hr ⁻¹ total CHO, water Blood [Glucose], [Insulin], [Lactate], [FFA]; HR Test: POMS, SCWT, MS-test, force sensation task	Sprint time at quarter 4: CHO < placebo ($p < 0.05$); MS-test corrected time at quarter 3 & 4: CHO < placebo ($p < 0.01$ & $p < 0.02$); Vertical jump height & forced output at quarter 4: CHO > placebo ($p = 0.02$ & $p < 0.05$); no difference in SCWT scores External POMS: CHO < placebo
Little et al. (2010)	16 M: 22.8 ± 3.2 yr; BMI 21.9 ± 1.4; $\dot{V}O_2\text{max}$ 55.4 ± 4.3 mL·kg ⁻¹ ·min ⁻¹ 13M completed (\bar{x} ± SD)	Randomised counter-balanced ~ 4 hr x 3 Ex: 45 min x 2 + 15 min break in between	Test meals: LGI (26) & HGI (76), 1.5 g CHO kg ⁻¹ BM 2 hr before Ex; & control Blood [Glucose], [Insulin], [Lactate], [FFA], epinephrine, non-epinephrine; HR Sprint test	Sprint distance: LGI ($p = 0.01$) & HGI ($p = 0.04$) > control Fat oxidation: LGI < control at 63-70 min ($p = 0.01$); HGI < control at 33-40 min ($p = 0.005$); trial x time interaction in [Glucose] ($p < 0.001$) before Ex & catecholamine during Ex ($p < 0.05$)
Cocate et al. (2011)	15 M: 24.4 ± 3.8 yr; BMI 21.9 ± 1.4; $\dot{V}O_2\text{max}$ 70 ± 5.3 mL·kg ⁻¹ ·min ⁻¹ (\bar{x} ± SEM)	Crossover 5 days x 2 Ex: 1.5 hr after breakfast; 30 min	LGI (28) & HGI (79) BF & L x 4 days Ex day LGI & HGI breakfasts: 2 g CHO kg ⁻¹ BM 3.5 hr pre-ex 83.5% energy from CHO Ex: 30-min cycloergometric at 85-95% HRmax Blood [Glucose], [Insulin], [Lactate], [FFA]; HR	Post meal: glucose AUC & INS AUC: HGI > LGI; fat oxidation: LGI < HGI; CHO oxidation: HGI < LGI Ex: no difference in glucose AUC

Table 2.7 (Continued)

References	Participants	Design and Duration	Intervention and monitoring	Outcomes and comments
Hulton et al. (2012)	9 M: 21 ± 3 yr; 74.4 ± 4.4 kg; 180 ± 8 cm ($\bar{x} \pm SD$)	Randomised counter-balanced 8.5 hr Ex: 3.5 hr after test meal; 45 min x 2 sessions	Pre-ex LGI (44) & HGI (80) meals: 2 g CHO kg ⁻¹ BM, ~ 62.5% energy from CHO Ex: 45-min high intensity intermittent x 2 Blood [Glucose], [FA], [Insulin], glycerol, [Lactate], b-hydroxybutyrate; HR, RPE	No difference in performance & substrate oxidation Post-Ex b-hydroxybutyrate: LGI > HGI

Abbreviations: AUC, area under the curve; BH, body height; BF, breakfast; BM, body mass; BMI, body mass index in kg·m⁻²; CHO, carbohydrate; Ex, exercise; [Glucose], glucose concentration; [FFA], free fatty acid concentration; HGI, high glycaemic index; HIT, high intensity intermittent; HR, heart rate; HRmax, maximal heart rate; [Insulin], insulin concentration; L, lunch; [Lactate], lactate concentration; LGI, low glycaemic index; MS-test, motor skill test; SCWT, RPE, rate of perceived exertion; Stroop colour word test; POMS, profile of mood states; $\dot{V}O_2$ max, maximal oxygen uptake.

This section summarised the potential benefit of GI on weight management, appetite, cognitive and exercise performances. There are discrepancies in the effects of GI on appetite and cognition among studies which may be due to the wide variation in study design employed and the study population characteristics. To date the effects of GI, based on the difference in the postprandial glucose responses following breakfasts as well as other main meals with LGI and HGI, on appetite and EI over a single day; cognitive performances and substrate oxidation during intermittent running in recreationally active adults have not been well investigated.

2.7 Measurement Issues

2.7.1 Energy intake

Nutrition related studies rely heavily on self-reported dietary intake data. There are several instruments, such as 24-hour dietary recall, food frequency questionnaire (FFQ) and self-reported food record, to assess the energy and nutrient intakes. Both 24-hour dietary recall and dietary record are self-reporting of food intake which is a subjective measure of EI. The description, the pros and cons of these methodologies has been reviewed comprehensively elsewhere (Thompson & Byers, 1994). Briefly speaking, the costing for assessing the FFQ is lower than 24-hour dietary recall and therefore it is popular to be used in epidemiological studies due to its low administrative cost (Neuhouser et al., 2006). The FFQ generally represents the dietary intake over a certain period of time, from over a month to a year. Besides, it was reported that the within individuals coefficient of variation of the differences were much higher for an FFQ (28.5%) than for three-day food records (16.5%) and for a dietary history (18.6%) (Livingstone & Black, 2003).

It is of concern that self-reported food intake is prone to high error levels due to intentional or unintentional misreporting, either under or over, of EI (Livingstone et al., 1990), particularly measuring in free living conditions (Livingstone & Black, 2003). Misreporting of the food intake can cause significant systematic bias (Black, 2000a). Under-reporting is a complex phenomenon. Many factors such as body composition, eating behaviour, body image and social desirability lead to the results of under-reporting (Scagliusi et al., 2009). Lean females appeared to have a better correlation between the reported EI and the total EE; whereas participants who were overweight or having problem of disordered eating were more likely to under-report food intake (Livingstone et al., 1990). Thus the probability of under-reporting appeared to increase with BMI, particularly female (Thompson & Byers, 1994). Eating behaviour questionnaires help to identify individuals who are more likely to under-report their intakes (Karlsson, Persson, Sjostrom, & Sullivan, 2000; Rennie, Siervo, & Jebb, 2006; Stunkard & Messick, 1985; Van Strien et al., 1986). However, Goldberg and Black (1998) pointed out that the problems of under-reporting occur at all levels of EE and in any populations.

Several studies identified the condition of under-reporting of dietary intake. Goldberg et al. (1991) and Goldberg and Black (1998) determined the cut-off limits of under-reporting of EI as less than 110–120% of estimated basal metabolic rate (BMR) for sedentary population or less than 155% for universal population with light activity where BMR is determined from the equations of Schofield (1985):

$$\text{BMR (18–30 years)} = 14.8 \times \text{BM (kg)} + 485$$

$$\text{BMR (30–60 years)} = 8.1 \times \text{BM (kg)} + 842$$

The FAO/WHO used the PAL values for identification of under-reporting (PAL = 1.56, < 1.6 for low activity level, 1.64, 1.6–1.73 for medium activity level, 1.82, > 1.73 for heavy activity level). Black (2000b) and Livingstone et al. (2003) suggested the use of individualised PAL value as the cut-off. The Goldberg cut-off was criticized about its specificity and sensitivity because the PAL factor was relied on the determination of investigators to choose a suitable PAL factor (Black, 2000b). The author analysed the database of 429 subjects from 22 studies of reported EI and doubly labelled water technique determined EE. The ratio of EI and EE less than 0.76, between 0.76–1.24; and over 1.24 were considered to be 'true' under reporters, acceptable reporters and over reporters respectively. The author also pointed out that the cut-off calculated for the mean population should not be used to identify individual under-reporters. Measuring EE to obtain individualized PAL in studies with small sample sizes was therefore recommended (Black, 2000a; Livingstone et al., 2003).

2.7.1.1 Dietary recall

The dietary recall has been considered as one of the optimal methods for collecting the data of food intake in dietary surveillance under a circumstance of being conducted by trained interviewers such as by a research dietician (Subar et al., 2007). The recall usually included free report of dietary intake over the past 24 hours without prompts, followed by prompted report so that the subjects could recall the missing information, if any. The interviewer could also be able

to get the specific portion by prompts after participants finished the recall. Although it was recommended to collect more than one day of recall to assess the usual intake (Guenther, Kott, & Carriquiry, 1997; Tooze et al., 2006), the long interview time and questions might lead to fatigue of participants.

2.7.1.2 Dietary record

Dietary record is a retrospective method to record the food intake, after the consumption. Participants can improve the estimation of food portions by being instructed about the technique for recording food intake (Howat et al., 1994). An appropriate length of the number of food record day can improve the accuracy of self-reported dietary intake. The validity of these methods depends on the accuracy with which participants record their food intakes, assessors enter the food records, as well as the database of food analysis software (Rennie, Coward, & Jebb, 2007).

The dietary records in the thesis were assessed by the same research dietician, to minimize the inter-researcher variation, facilitating the skill of the dietary recall to cross check any missing items (Braakhuis, Meredith, Cox, Hopkins, & Burke, 2003).

2.7.2 Energy expenditures

Total daily EE is comprised of three components: resting metabolic rate (RMR) (60–75%), thermic effect of diet (10%) and physical activity EE (10–30%) (Starling, 2002). Physical activity level is generally characterized by the type, duration, intensity and frequency. Physical activity EE can be measured via

assessing the PAL subjectively and objectively. Energy expenditure is commonly expressed as metabolic equivalents (MET) or in terms of calories burned. More than 30 techniques have been developed for assessing PAL or EE (Table 2.8) (LaPorte, Montoye, & Caspersen, 1985).

Table 2.8 Common measurement methods of energy expenditures

Measurement methods	Examples
Biochemical and physiological	<ul style="list-style-type: none"> • Heart rate monitor • Indirect calorimetry • Doubly-labelled water
Passive motion sensors	<ul style="list-style-type: none"> • Pedometer • Accelerometer • Global positioning system
Direct observation	<ul style="list-style-type: none"> • behavioural observation
Subjective	<ul style="list-style-type: none"> • Recall questionnaire • Physical activity records • Quantitative history • Task-specific diary

2.7.2.1 Accelerometry

Accelerometry is one of the widely accepted techniques for assessing daily EE under free-living conditions due to its light weight, easy, non-invasive, objective, valid and reliable characteristics (Brage et al., 2004; Welk, 2005). The principle of the accelerometry is to detect the movement based on the degree of acceleration. The ActiGraph accelerometer is a small and lightweight (3.8 cm x 3.7 cm x 1.8 cm, 27 g) device to detect motion ranging in magnitudes from 0.05 to 2 g with a band frequency between 0.25 and 2.5 Hz (ActiGraph GT1M

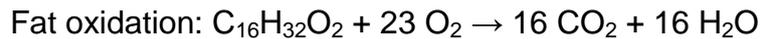
accelerometer, ActiGraph LLC, Pensacola, FL, US). Originally the GT1M was a uniaxial accelerometer which could only detect vertical accelerations along the human body with respect to time ($m \cdot s^{-2}$). The GT1M has now enabled to collect along the X axis as well through a firmware update that this device has been biaxial since October 2008.

The accelerometry data can reflect the frequency, duration and intensity of movement of human subjects (Swartz et al., 2000). The counts are collected at different time sampling intervals (epoch) and then data are transformed into EE with count cut points values corresponding to predetermined MET levels. There are many cut points of the intensity of activity to counts and MET (Jeremey, 2012); as well as the regression models for use with the ActiGraph accelerometer (Crouter, Churilla, & Bassett, 2006; Crouter, Kuffel, Haas, Frongillo, & Bassett, 2010; Freedson, Melanson, & Sirard, 1998). Crouter et al. (2006) compared 14 ActiGraph prediction models used to estimated MET. The results found that energy prediction model of Freedson et al. (1998) provided the closest estimate of both light and moderate physical activity whereas the rest of prediction models underestimated the duration spent in vigorous PA. A recent developed two-segment regression model has been shown to improve accuracy of EE estimates when compared with a simple regression (Crouter et al., 2010).

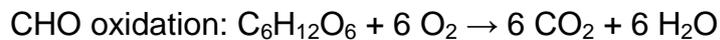
2.7.2.2 Indirect calorimetry

Indirect calorimetry calculates the respiratory exchange ratio (RER) based on the ratio of the volume of oxygen consumption ($\dot{V}O_2$) and carbon dioxide

production ($\dot{V}CO_2$) via the collection of the expired gases through a face mask to estimate EE. The RER usually ranges between 0.7 and 1.0. The RER for fat and CHO are based on the combustions of palmitic acid and glucose, respectively:



$$\text{RER for fat} = 16 CO_2 / 23 O_2 = 0.7$$



$$\text{RER for CHO} = 6 CO_2 / 6 O_2 = 1.0$$

Correspondingly, RER equal to 0.7 and 0.85 represent fat; and 50% and 50% CHO and fat respectively (Starling, 2002). The RER can be below 0.7 or above 1.0 in some conditions such as at starvation or peak exercise respectively. The error is reported to be 2–3% (LaPorte et al., 1985). Participants should perform exercise under a steady state condition so that the $\dot{V}CO_2$ and $\dot{V}O_2$ reflected the metabolic exchange of gases for estimating substrates oxidation during exercise. Due to the small contribution of energy from protein, non-protein stoichiometric equations or figures have been developed to calculate the rates of CHO and fat oxidation (Frayn, 1983).

2.7.2.3 Physical activity log

Physical activity log is a self-report of physical activity (Appendix B). Ainsworth et al. (2011) updated the Compendium of Physical Activities which provides 821

codes covering a wide variety of physical activity to identify the energy cost of physical activity in term of the MET. The EE can then be expressed by multiplying the MET values with BM and the time occupied by that specific physical activity.

2.7.3 Subjective sensation

2.7.3.1 Appetite

Self-report of appetite sensation includes rating attempted to capture specific elements of sensation at a specific time duration (Blundell et al., 2010). The rating scale can measure subjective or perceived appetite sensations such as hunger, desire to eat, PFC, and fullness; as well as the physical comfort of the subjects and the palatability of a meal (Drapeau et al., 2007). A set of three to seven sensational elements has been widely accepted and used in appetite related research (Blundell et al., 2010; Mattes et al., 2005).

The VAS can be used not only for the measurement of human appetite, but also supported for the prediction of subsequent food intake (Cardello et al., 2005). Ratings of hunger and satiety are related to meal frequency and meal interval. Fullness might be synchronized with satiety, whereas the portion being consume, the desire to eat, and appetite might be synchronized with hunger. Previous works investigated the reproducibility and the validity of using VAS questionnaire for assessment of appetite sensation (Flint, Raben, Blundell, & Astrup, 2000; Raben, Tagliabue, & Astrup, 1995; Stubbs et al., 2000). Raben et al. (1995) found that the coefficients of repeatability for subjective appetite ratings in nine participants were large due to day-to-day variation. The

coefficients of repeatability varied in different subjective appetite elements that feelings of fullness and satiety produced lower coefficients of repeatability than hunger and prospective food consumption. Flint et al. (2000) found that the coefficient of variation of the 4.5-hour mean appetite ratings were 9–21% in non-diet participants. The authors suggested that 8–32 participants would be required to detect a 10% difference in fasting, peak and 4.5-hour mean appetite ratings between two foods. Of which, the peak appetite ratings required the greatest number of participants to detect the difference between two foods. The authors explained that meals with the same amount of energy were provided to participants regardless the body size. Thus it was expected that the variation would be reduced if the body size was considered when deciding the meal size.

Stubbs et al. (2000) also reviewed the ability of VAS to predict feeding behaviour, the sensitivity to experimental manipulations, and the reliability, validity and reproducibility. The authors stated a good reproducibility in within-participant designs under laboratory conditions. Subjective feelings of appetite can be assessed reproducibly and predict the food intake when planned appropriately after ~ 80 studies were reviewed (De Graaf et al., 2004). Mattes et al. (2005) illustrated that the pre-lunch appetite ratings could explain 8–12% of the variance in subsequent EI whereas the mean ratings over 4.5 hours post breakfast could explain 23% of variance of subsequent EI.

Blundell et al. (2010) advised that VAS is a valid, sensitive and unbiased measurement tool to translate and interpret the appetite sensation under standardised practices. It is not appropriate to selectively pick a single element

to support the research hypotheses de facto. The author also suggested that measuring the AUC calculated from mean score of appetite elements over the study period is better than mean scores at single time points in terms of repeat-reliability, unless a specific time point is hypothesised to be examined in *priori*.

2.7.3.2 Mood

The mood state can be assessed by the Profile of Mood States (POMS). The original POMS is a 65-item questionnaire with proven validity and reliability to assess human mood among various populations (McNair et al., 1971). A shortened version of POMS consisting of 37-items developed to be easier for administration time management (Shacham, 1983). The shortened versions remained the assessment of six psychological issues of mood state: anger-hostility, confusion-bewilderment, depression-dejection, fatigue-inertia, tension-anxiety and vigour-activity. The POMS was reported to be sensitive to nutritional manipulation (Fischer et al., 2001). Lower blood [Glucose] was shown to negatively alter the mood in terms of increasing fatigue and decreasing vigour (Degoutte et al., 2006).

2.7.4 Eating behaviour

Many diet related questionnaires have been developed to assess eating behaviours (Garner et al., 1982; O'Neil et al., 1979; Stunkard & Messick, 1985; Van Strien et al., 1986). Rennie et al. (2006) suggested that the use of eating behaviour questionnaire is not only to assess the type of eating behaviour but is also able to identify and evaluate the occurrence of under-reporting at an individual level. It might also be suggested as a standard practice to exclude under- or over-reporting participants with diet related questionnaire scores over

set thresholds, unless an eating disorder or restrained eating behaviours are the main aims of the appetite related research (Blundell et al., 2010).

The Eating Attitudes Test–26 (EAT–26) (Garner et al., 1982) is a 26-item self-report questionnaire (Appendix C). The total score over 20 or more is usually considered as a cut-off of having the risk of eating disorder but it is not designed as a diagnostic tool to identify eating disorder.

Three Factor Eating Questionnaire contains 51 questions to identify three different types of eating behaviour: cognitive restraint, disinhibition and emotional eating. The cognitive restraint of eating that is a perception that an individual controls food intake intentionally for body weight management. The disinhibition is a tendency of an individual losing control over eating in response to hunger or external cognitive or emotional stimulus. The emotional eating is a participative response that an individual loses control over eating when facing the negative moods such as feeling lonely or depressed (Stunkard & Messick, 1985). A modified version of TFEQ has been developed with 21 questions which is a shorter version of the 51-item version which was recommended using in non-obese populations (Cappelleri et al., 2009) (Appendix D).

Dutch Eating Behaviour Questionnaire is comprised of 33 questions to identify three types of eating behaviour: restrained eating, i.e. eating less than desired to lose or maintain body weight; emotional eating, i.e. eating in response to

negative emotions; and external eating, i.e. eating in response to the stimulus such as sight and smell of food (Van Strien et al., 1986) (Appendix D).

2.7.5 Anthropometry

Many techniques and equations have been developed for quantifying body composition, such as body fatness, muscle mass and fat free mass, of individuals or population groups in health or sports related studies (Table 2.9). There is no 'gold standard' methodology for body composition assessment (Ackland et al., 2012). Every method has its advantage and limitation. For instance, dual energy X-ray absorptiometry detects negative readings of fat on the torso in elite athletes; magnetic resonance imaging is a costly, sophisticated and time consuming technique; the unnecessary radiation exposure using computerised tomography is of concern. Having considered issues of accuracy, cost, complexity, reliability, precision, repeatability and utility, high correlations and low coefficients of variation allow for good predictions of body composition in the laboratory or in the field areas.

The choice of technique needs to fit the purpose for which data are to be used. The purpose of the assessment of body composition in the thesis was to monitor the homogeneity of the participants being recruited, to use the variables of body composition as covariates as well as to determine the trial regimes. Skinfold thickness is a doubly indirect assessment of body composition in sports. Eston, Rowlands, Charlesworth, Davies, and Hoppitt (2005) found that lower body skinfolds were highly related to the percentage of body fat in fit and healthy young adults. The authors recommended the inclusion of the thigh

skinfold to enhance the estimation of body fatness. The predictions equations of Durnin and Womersley (1974), Jackson and Pollock (1978) and Jackson, Pollock, and Ward (1980) have been widely used in different populations (Peterson, Czerwinski, & Siervogel, 2003). The thigh and the subscapular site are not included in the equations of the Durnin and Womersley (1974); and Jackson and Pollock (1978) and Jackson et al. (1980) respectively. Male and female athletes were found to have highest values of skinfold thickness at the subscapular and at thigh respectively (Ackland et al., 2012). The equation of Peterson et al. (2003) using the four-compartment model including the skinfold at iliac crest, subscapular, triceps and mid-thigh sites had better prediction of the percentage of body fat.

Table 2.9 Common assessment methods of body fatness

Approaches	Methods	Measurement error
Laboratory	Dual energy X-ray absorptiometry	2–3%
	Air-displacement plethysmography	1.8–3.7%
	Hydrodensitometry	2–2.5%
	Ultrasound	3%
Field	Anthropometry	3–3.5%
	Bio-electrical impedance analysis	3.5%

2.7.6 Cognitive Performance

Cognitive performance can examine a number of cognitive functions: perception, memory, attention and arousal, information processing, accuracy, speed of movement and vigilance. The assessments of the performance comprise two main measures: speed and accuracy (Dye, Lluch, & Blundell, 2000). Speed can be measured via the RT whilst accuracy can be measured via the number

of correct and wrong answers (Dye & Blundell, 2002). Reaction time is measured as a response to a variety of visual stimuli. The effect of independent variables for RT appears to be most demonstrable effectively in breakfast studies that the rise in blood glucose was suggested to improve performance of reaction time tasks (Dye & Blundell, 2002). Any factors affecting the substrate provision is therefore expected to affect the cognitive performance (Pollitt & Mathews, 1998). Attention may be affected by macronutrient intake. Of which the oxidation of glucose for energy is the greatest among macronutrients and thus it is expected to be more of importance to be investigated.

In choosing a cognitive task, simpler approach and a purpose of investigating the short-term effect of food are the focus of most research (Dye et al., 2000). Cognitive performance can be assessed verbally, in written form; or in a computerized format by using specific psychological test software (E-prime version 2, Psychology Software Tools, Inc.).

The Stroop colour word task (SCWT) was firstly developed in 1930's to assess selective attention (Stroop, 1935). A modified version of SCWT was developed by Golden (1975) to assess selective attention via the sensitivity to interference and the ability to suppress an automated response. The selective attention refers to the ability to pay attention on completing one task while ignoring other irrelevant factors such as external distracters or stimuli (Evans, Hopkins, Macdonald, & Amiel, 2004; MacLeod, 1991, 1998; MacLeod & MacDonald, 2000; Stroop, 1935; Wenzel & Rubin, 2005).

The highly demanding Rapid information processing task (RIPT) can assess vigilance and sustained attention (Foulds et al., 1996; Owens, Parker, & Benton, 1997). Vigilance and sustained attention are the abilities to maintain alert at high levels for an extended period of time. The RIPT requires high mental effort from participants to detect the target sequence of even or odd digits within a pseudorandom sequence of single digits (Benton & Donohoe, 2011).

The Choice reaction time test (CRT) is commonly used during exercise to evaluate the exercise effect on selective attention (D'Anci K, Vibhakar, Kanter, Mahoney, & Taylor, 2009; McMorris & Hale, 2012) and focused attention (Tomporowski, 2003).

2.7.7 Biomarkers

The glucostatic theory suggested that the appetite could be controlled by the glucose responses (Mayer, 1955). Capillary blood samples were found to have smaller within-subject variation than venous blood samples (Cooper et al., 2012; Granfeldt et al., 2006; Wolever et al., 2003). Besides the postprandial glycaemic response, some intestinal hormone responses may be also related to appetite. The small intestine affects the satiation of a meal and satiety for the subsequent meal by generating signals of hunger and satiety. Satiety related peptides such as glucagon-like peptide-1, ghrelin, and peptide YY are biomarkers of appetite regulation (De Graaf et al., 2004).

Flint et al. (2006) found greater insulin responses related to greater sensation of fullness; whereas no such association were evident between glucose and appetite ratings in 28 healthy male adults. However, glycaemic responses were positively associated with lunch EI, but not the insulinaemic responses in that study. Despite these findings, a review by the same authors suggested that insulin, but not glucose, was associated with the short term regulation of appetite (Flint et al., 2007). Another study found that the postprandial glucose, insulin and glucagon-like peptide-1 responses were lower after 10-week *ad libitum* intake of the LGI diet and the HGI diet; however no difference was found in the leptin, ghrelin and glucagon in 29 healthy but overweight females (Krog-Mikkelsen et al., 2011), In addition, the difference in the glucagon-like peptide -1 responses between LGI and HGI diets did not promote a difference in appetite and EI.

Measurement of appetite related biomarkers provides an objective way of understanding appetite sensation response to a meal. Although these appetite peptides and hormones are related to appetite, there are myriads of such substances in human body and their interactions are poorly understood. Selection of a single measure may be inappropriate. The choice of hormones being investigated needs to fit the purpose for which data are to be used. Insulin sensitivity varies even in healthy adults, resulting in altered postprandial glucose responses modifying the biomarker changes.

There is no 'gold standard' methodology for assessment of appetite, energy intake, expenditures, cognitive performance and metabolic responses. The

choice of measurement tools needs to fit the purpose for which data are to be used. Every method has its advantage and limitation. Having considered issues of accuracy, cost, complexity, reliability, precision, repeatability and utility; the studies in the current thesis employed VAS to assess appetite sensation. The correlation between the subjective appetite and the capillary blood [Glucose] was then determined. The energy intake and expenditure were estimated using a locally developed nutrient analysis and accelerometry assisted with physical activity log, respectively. Vigilance and attention were assessed which are more likely to be ecologically relevant to skill sports, with or without high level of physical activity. Assessment of glycaemic and insulinaemic response to the effects of GI on appetite, cognition and metabolic responses were more financially appropriate other costly appetite related hormones.

2.8 Conclusions

This review has summarised the physiological factors affecting appetite, the impacts of GI on appetite, cognitive and exercise performances as well as the measurement considerations employed to investigate the effects of GI on appetite, cognitive and metabolic responses.

Previous findings about the effects of GI on appetite or weight management in the longer term are equivocal. It has even been suggested that modifying GI does not improve weight management (USDA Evidence Analysis Library, 2012). All these studies recruited overweight and obese participants many of whom were glucose intolerant. The metabolism and eating behaviours might differ

from healthy adults. Inconsistent results of the effects of GI on appetite at the short term in healthy adults have also been reported. Many of those studies did not tightly control confounders such as macronutrient composition, energy density and dietary fibre. The inconsistency may be due to weaknesses in both the study designs and execution. Therefore, the aims of the study one, two and three was to investigate the effects of GI on appetite; as well as energy intake in a shorter experimental duration with improvement and tighter monitoring of the limitations in the previous studies.

Limited research has been conducted into the effects of breakfast GI on cognitive functions. Of those limited research in healthy adults that has been carried out, memory improvement has been found following LGI foods. However, assessment of attentional performance appears to be more ecologically relevant for sporting success, with low or high physical activity. There is a lack of research with consistent methodologies and detailed techniques of food interventions about the effects of mixed breakfast GI on cognitive performances, especially vigilance and attention, in recreationally active males.

Previous research predominantly investigated the effects of pre-exercise meals with different GI on endurance performance. However, the designs of these studies usually determined moderate-high intensity using the percentage of the $\dot{V}O_2\text{max}$ which has high individual variability (Xu & Rhodes, 1999). In addition, many sports encompass physical and cognitive loads simultaneously. The previous investigation of substrate oxidation during intermittent exercise is

comparatively limited; whereas the results might have a great implication in weight management. There has been no study that specifically compares the effects of LGI and HGI at breakfasts taken prior to an intermittent exercise on physiological measures and cognitive function during and after an intermittent running in recreationally active males.

In this thesis the effects of breakfast GI alone or in combination with the regular intake of meals with different GI on:

- appetite and EI over a single day;
- cognitive performance with minimal physical activity and during intermittent running; and
- substrate oxidation during intermittent running

were investigated in recreationally active adults to fill this gap in the world of exercise.

CHAPTER 3 GENERAL METHODS

This chapter describes the main equipment and materials that were used in more than one study. Any methodologies that were used only in a specific study were described within the section of the methodology of that specific chapter.

3.1 Experiments

All the main trials of the studies employed a single-blind, randomized, crossover design with repeated-measure design. All testing took place within the area of the Children's Health and Exercise Research Centre of the Sport and Health Sciences of the University of Exeter. All main trials were performed on the same day of the week for each participant whenever possible (Morris & Payne, 1996) under similar and controlled environmental conditions.

3.2 Participants

All participants were recruited in the Exeter area. The recruitment strategy included distributing recruitment leaflets (Appendix E) at the Streatham and the St. Luke's campuses of University of Exeter, as well as local sports centres. Participants were informed of the testing procedures and the possible risks of the study (Appendix F). They were required to complete a medical and health questionnaire (Appendix G). Physical activity level was assessed by self-report at the health questionnaire. Written informed consent was obtained prior to data collection (Appendix H). The studies were all approved by the Research and Ethic Committee of the Sport and Health Sciences in accordance with the

sixth version of the 2008 Declaration of Helsinki. The general inclusion criteria were:

- Aged 18 to 45 years.
- Recreationally active for at least three occasions of moderate intensity exercise each with 30 minutes per week, ideally in the sports of endurance, aesthetic or with weight categories (Slater, Rice, Mujika, et al., 2005).
- Absence of medication or supplements affecting metabolism, taste, smell or appetite (Ford et al., 2011); or designed for weight management.
- No known metabolic, menstrual or eating disorders.
- Non smoker.
- Stable body weight and that the fluctuation of body weight was less than 10% over the last three months prior to recruitment (Isaksson et al., 2009).
- No known food allergy or aversion to study foods screened by the food acceptability check list (Appendix I).
- Habit of eating breakfast at least three times per week in the last month (Murphy, 2007).

General exclusion criteria were:

- Aged under 18 years or over 45 years at recruitment.
- Sedentary lifestyles.

- Smoker.
- Presence of medication or supplements known to affect metabolism, taste, smell or appetite (Ford et al., 2011); or designed for weight management.
- Being diagnosed with known genetic disorder of metabolism, menstrual or eating disorders.
- Change in body weight more than 10% over the last three months prior to recruitment (Isaksson et al., 2009).
- Known food allergy or aversion, or dislike of more than half of the study foods.
- Non-regular breakfast eater.

3.3 Glycaemic index in meals

The GI values of the LGI and HGI test breakfasts in study one, two, three and four; test lunches at study two and *ad libitum* HGI lunch at study three; test snacks and dinners at study two; and pre-trial LGI dinner at study three were calculated using the mixed-meal method of Wolever and Jenkins (1986). The GI values of the CHO rich food items were selected from the published sources (Atkinson et al., 2008; Foster-Powell et al., 2002; The University of Sydney, 2010) and counted when the items contained more than 5 g CHO per 100 g food weight. When the GI value of a branded item was not available in the published literatures (Atkinson et al., 2008; Foster-Powell et al., 2002; Henry, Lightowler, Strik, Renton, & Hails, 2005), the GI value of the generic item was applied (Olendzki et al., 2006). When the generic item had several GI values, a mean value was calculated (Ramel, Gudmundsdottir, & Thorsdottir, 2012).

The proportion of the CHO in g from a CHO rich food component in a meal was divided by the total amount of CHO in g in that meal and then multiplied by the GI for that food component. Glucose was used as the reference food (GI = 100) (Atkinson et al., 2008; Foster-Powell et al., 2002).

The contents of all macronutrients, energy and dietary fibre were calculated using the locally developed nutrient dietary analysis software. The dietary fibre in the data base of the software using the Englyst method presented as NSP (H. N. Englyst, Quigley, & Hudson, 1995; Food Standards Agency, 2002; McCance et al., 1991).

3.4 Energy intakes

Participants recorded all food and drink intakes upon request. They were asked to describe the intake in detail including meal times, locations, portions and brand names (Rennie et al., 2007). A standardised written instruction and food record sheets were given (Appendix J). A research dietician went through the food records, as well as clarifying any unclear or missing information with participants. Food records were analysed for calculating EI by the nutrient analysis software by the locally developed nutrient dietary analysis software (Microdiet 2.0, Downlee Systems Ltd., High Peak, U.K.).

3.5 Energy expenditure

3.5.1 Accelerometry

The ActiGraph GT1M accelerometer was used in the study one and two. The data were initially collected in 5-second epochs and then converted to 10-second epochs. A valid day of data contained at least 10 valid hours of wear time. Non wear time was defined as a minimal interval of 60 consecutive minutes of inactivity (Crouter et al., 2010). The EE was estimated using the manufacturer's software (V5.6.4 ActiLife, FL, US) with the cutpoints of Troiano et al. (2008) (Table 3.1).

Table 3.1 The relation of intensity of physical activity to counts and metabolic equivalents (MET)

Intensity of activity	Accelerometer Counts	MET values
Sedentary	0–199	< 1.5
Light	200–2,019	1.5–3
Moderate	2,020–5,998	3–5.9
Vigorous	≥ 5,999	≥ 6

3.5.2 Physical activity log

Physical activity log was completed in the study one and two. The energy cost in term of the MET was estimated using the Compendium of Physical Activities (Ainsworth et al., 2011). One MET is equivalent to $3.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and the consumption of one litre of oxygen equates to 20.35 kJ. The EE was then be calculated by multiplying the MET values with BM and the time occupied by that specific physical activity.

3.5.3 Indirect calorimetry

The method of indirect calorimetry was used in study two, three and four. Expired air was collected through a face mask and a sample line connected to a real time gas analyser (Cortex Metalyser 3B, Leipzig, Germany). Following the manufacturer's guidelines, all pressure, gas and volume were calibrated prior to each measurement by ambient air and a standard calibration gas (16% O₂, 4.96% CO₂). Flow calibration was performed by attaching a turbine to a volume cuff as well as a sample line to a 3-litre calibration syringe (Hans Rudolph, U.K.). The rates of oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$); and the RER were calculated synchronously. The rates of CHO and fat oxidation were estimated using the following equations (Frayn, 1983):

$$\text{Rate of CHO oxidation (g}\cdot\text{min}^{-1}) = 4.585 \times \dot{V}CO_2 - 3.226 \times \dot{V}O_2$$

$$\text{Rate of fat oxidation (g}\cdot\text{min}^{-1}) = 1.695 \times \dot{V}O_2 - 1.701 \times \dot{V}CO_2$$

3.6 Subjective sensations

3.6.1 Visual analogue scale

The VAS was used in study one, two and three to assess sensation of appetite, palatability to the meal and physical comfort. The VAS consisted of a 100 mm horizontal line anchored at each end with opposing statements (Appendix K). Questions such as 'How hungry are you?', 'How full are you?' and 'How strong is your desire to you?' were asked. The two ends of the statements were such as 'Not at all', 'Very weak' and 'Completely empty' vs. 'Very full', 'Very strong' and 'Cannot eat another bit', respectively. A vertical slash was placed across the horizontal scale to indicate their feeling to each statement at that point in

time. The VAS rating was calculated by measuring the distance in mm from the beginning of the left side of the line to the vertical slash.

The appetite score (AS) (Chaput et al., 2010) and the palatability score (PS) (Flint et al., 2000) were obtained from the mean score composited from five elements of appetite sensation and four sensational elements related to palatability, respectively:

$$AS = [\text{Desire to eat} + \text{Hunger} + \text{PFC} + (100 - \text{fullness}) + (100 - \text{Satiety})] / 5$$

$$PS = (\text{visual} + \text{smell} + \text{taste} + \text{palatability}) / 4$$

3.6.2 Mood state

Mood states were assessed using the 37-question POMS to assess six mood states: tension, depression, anger, vigour, fatigue and confusion (Shacham, 1983). Each question was based on a 5-point Likert scale consisting of 0 for not at all, 1 for a little, 2 for moderately, 3 for quite a bit, and 4 for extremely.

3.7 Eating behaviour

The 26-item Eating Attitudes Test–26 (EAT–26) (Garner et al., 1982) was used in all studies (Appendix C). Questions were presented in a 6-point Likert scale from 1 (never) to 6 (always). The total score below 20 was the cut-off point as one of the inclusion criteria for the studies.

An integrated eating behaviour questionnaire combined from the 21-item Three Factor Eating Questionnaire (21-TFEQ) (Cappelleri et al., 2009) and the Dutch Eating Behaviour Questionnaire (Van Strien et al., 1986) was used in the study one and two to identify different types of eating behaviour (Appendix D). Questions were presented in a 5-point Likert scale from 'Never' to 'Very often' for the first 33 questions, in a 4-point Likert scale from 'Definitely true' to 'Definitely false' between question 34 and 49; and five miscellaneous questions between question 50 and 54.

3.8 Anthropometry

Body composition was measured in all studies. Body stature, BM, skinfold thickness at four anatomical locations: iliac crest, subscapular, triceps and mid-thigh were measured using standard procedures (Eston & Reilly, 2009). The standing stature was measured by a stadiometer to the nearest 0.1 cm (Novel Products, Rockton, IL). Body mass was measured using a double-beam balance scale (Fairbanks Scales, Kansas City, MO) to the nearest 0.01 kg. No adjustment was made for the weight of clothing when participants wear minimal clothing such as short pants and short-sleeved T-shirts. Body stature and mass were measured in duplicate and the mean values were used.

Each skinfold site was marked with a cross by a water soluble marker and measured at the right side of the body with a skinfold calliper (Holtain Ltd., Crymych, U.K.). The measurements were taken in rotation from one site to the next until the measurements of the four skinfold sites were completed. Three readings were taken at each site to get a median for analysis (Slater, Rice,

Sharpe, et al., 2005). The percentage of body fats were estimated using the following equations (Peterson et al., 2003):

$$\text{Female BF\%} = 22.18945 + (\text{age} \times 0.06368) + (\text{BMI} \times 0.60404) - (\text{BH} \times 0.14520) + (\sum 4 \times 0.30919) - (\sum 4^2 \times 0.00099562)$$

$$\text{Male BF\%} = 20.94878 + (\text{age} \times 0.1166) - (\text{BH} \times 0.11666) + (\sum 4 \times 0.42696) - (\sum 4^2 \times 0.00159)$$

where BF% is percentage of body fat, age is the age in year, BH is the body stature in cm, $\sum 4$ was the sum of iliac crest, subscapular, triceps, mid-thigh

The body mass index (BMI) and the fat free mass (FFM) were calculated. All devices, except the measuring tape, were routinely calibrated for accuracy and precision by in house technicians.

3.9 Cognitive tasks

All cognitive tasks performed in study three and four using a psychological software (E-prime version 2, Psychology Software Tools, Inc., Sharpsburg, PA, U.S.) via a 15-inch computer monitor. The monitor was placed horizontally to the eye levels of participants. Participants provided all responses via a labelled keyboard. Written instructions were shown on the screen before the start of the tasks. Verbal explanation was also given to participants to reassure they fully understood how to perform the tasks prior to the start of the first task.

Participants were told that the performance was based on the response speed and response accuracy.

3.9.1 Stroop colour word task (SCWT)

The SCWT was performed in study three and four. The task consisted of four conditions in two levels. The first level was for practice in four different images, “red,” “green,” “yellow” or “blue”, appearing one at a time in white colour on the black computer screen. Participants were required to respond by “v” at the keyboard for “green”, “b” at the keyboard for “yellow”, “n” at the keyboard for “red” and “m” at the keyboard for “blue”. Labels were stuck to replace the original position of “v”, “b”, “n” and “m” by “g”, “y”, “r” and “b”, respectively. The second level was a colour-interference level which included the conditions of neutral, congruent and incongruent. The neutral, congruent and incongruent conditions consisted of “XXXXX” in the above four mentioned colours, the “red,” “green,” “yellow” or “blue” in their own colours; and the “red,” “green,” “yellow” or “blue” in contrasting colours, respectively. The stimulus duration was 1,500 ms; whilst the stimulus disappeared once the labelled key was pressed for response. Playing back to correct the errors was not allowed. An inter-stimuli interval was set with a white fixation cross in the centre of the screen.

The software recorded the response accuracy and the errors; and the RT for the correct responses and the wrong responses. The response accuracy and the errors were considered as dependent variables. A correct response quicker than 150 ms was excluded and then considered as an error. The interference and facilitation scores were calculated (MacLeod, 1991, 1998):

Interference time = incongruent RT – neutral RT

Facilitation time = neutral RT – congruent RT

Interference score for accuracy = number of correct incongruent response – number of correct neutral response

Facilitation score for accuracy = number of correct congruent response – number of correct neutral response

3.10 Blood biochemistry

Capillary blood glucose and lactate concentrations were measured in study three and four. The blood sampling was collected using a single-use puncture device (Safety-Lancet, Sarstedt Limited, Leicester, U.K.). Blood samples were collected into a 300 µl fluoride containing capillary tube (Microvette® CB 300, Sarstedt Limited, Leicester, U.K.). Three readings were measured from each sample to obtain a median value. Whole blood [Glucose] and [Lactate] were determined by an automated glucose and lactate analyser (YSI 2300 STAT Plus™ Glucose and Lactate Analyser, YSI (UK) Limited, Hampshire, U.K.).

3.11 Statistical Analysis

All statistical analyses were conducted using PASW Statistics 18 (version 18.0, SPSS Inc., Chicago, IL, U.S.). All values were presented as means and standard deviations (SD) unless as standard error of mean (SEM) for graphic presentation. A two-way repeated-measures analysis of variance (ANOVA) was used to assess trial, time and trial by time effects of different variables. Greenhouse-Geisser corrected *p*-values were reported when the

assumption of sphericity was not met. All statistics were performed at the significant level of $\alpha = 0.05$. All statistical analysis has been described separately within each study.

CHAPTER 4 STUDY ONE: EFFECT OF GLYCAEMIC INDEX AT BREAKFASTS ON APPETITE SENSATIONS AND ENERGY HOMEOSTASIS IN RECREATIONALLY ACTIVE FEMALES

4.1 Introduction

Glycaemic index is a ranking system of the postprandial bioavailability of glucose following the intake of CHO rich foods (Jenkins et al., 1981). The rationale of LGI diets for weight control is due to its sustained CHO bioavailability and more gradual rise in postprandial blood glucose levels, based on the glucostatic theory (Mayer, 1955). Association of LGI diets with health benefits in individuals with chronic conditions have been widely reported with its potential beneficial effect on regulating body weight (FAO, 1998; WHO/FAO, 2003). Appetite, including satiation and satiety, may be altered by varying the GI of ingested meals (De Graaf et al., 2004; Fischer et al., 2004; Kissileff, Gruss, Thornton, & Jordan, 1984).

Although following LGI diets are expected to control appetite suppression, there is considerable inconsistency in the literature which requires clarification (Esfahani et al., 2011; Ford & Frost, 2010; Thomas et al., 2007). A major limitation to the interpretation of previous literature is that one or more aspects of the research design lacked control of the confounding variables such as macronutrient composition, energy density and dietary fibre. In addition, a single drink or food rather than a mixed meal is not ecologically valid for the free living condition.

Previous studies have reported an association of consumption of LGI foods on weight management and appetite in overweight or obese adults (FAO, 1998; WHO/FAO, 2003), or appetite and subsequent EI in healthy males (Anderson et al., 2002). To date the effects of GI on appetite and subsequent EI has not been investigated in recreationally active females. The purpose of the study was to investigate the effect of GI breakfast to subsequent appetite sensation and energy homeostasis of recreationally active females. The objectives were to determine and compare the difference between trials in:

- subjective appetite sensation one hour after breakfast and throughout the trial day,
- total voluntary EI; and
- total EE in the trial days.

It was hypothesized that a single LGI breakfast compared to a single HGI breakfast would provide greater appetite suppression; and reduce the total EI while maintaining the EE in a trial day.

4.2 Methodology

4.2.1 Participants

Participants were briefed that the study was to investigate general aspects of meals on appetite sensation; but not the specific purpose of investigating the effects of GI on appetite. They were screened by the 26-item EAT-26 (Garner et al., 1982) (Appendix C) for eligibility. Additional criteria were Caucasian female and EAT-26 score below 20 (Garner et al., 1982).

4.2.2 Study design

The purpose of the study was to examine the effects of a LGI and a HGI breakfasts on the subsequent appetite sensation in recreationally active females over a single day. Participants acted as their own controls and were assigned to receive the two test breakfasts, LGI and HGI, on two separate mornings. Each trial was completed with at least two days in between (Brouns et al., 2005). Both trials were scheduled within the follicular phase of the menstrual cycle, day one to day 14 (Appendix L), to reduce the day-to-day variation in EI and EE (Korth et al., 2007; Morris & Payne, 1996; St-Pierre et al., 2006; Warwick et al., 1993; Webb, 1986).

4.2.3 Preliminary procedures

Participants recorded two days of breakfast intake for estimation of habitual breakfast EI before the first trial (Mettler et al., 2007). The energy content of the test breakfasts was determined from the habitual breakfast EI.

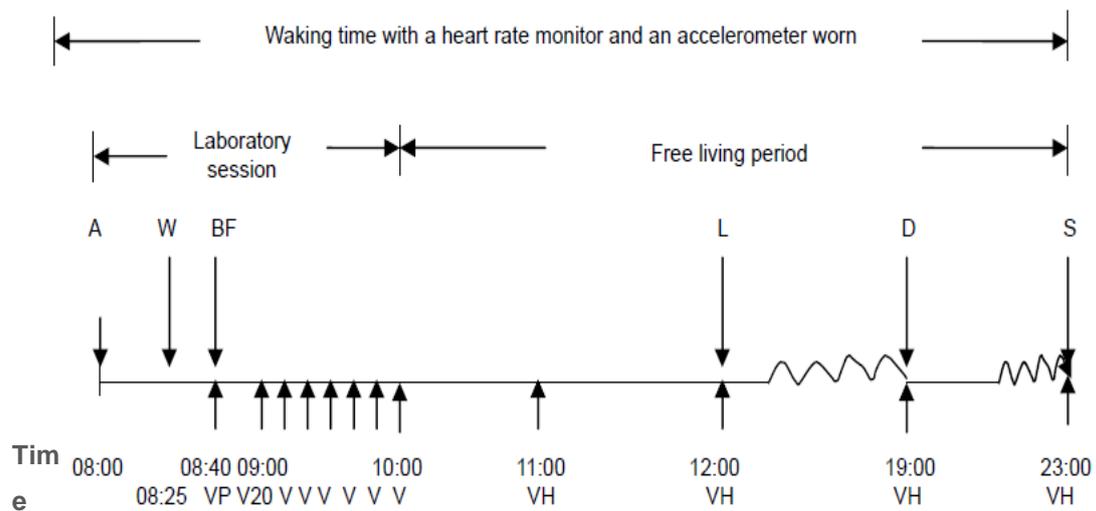
Participants were required to record food and drink intakes, to wear an accelerometer, to maintain their habitual physical activities and similar dietary patterns two days prior to each trial. They were required to keep similar activity patterns two days before every trial and to take a similar meal the evening before every trial so as to minimize the potential evening meal effect on appetite and subsequent glycaemic responses (Brouns et al., 2005; Gilsenan et al., 2009; Lee et al., 2006; Mettler et al., 2007; Wolever, Jenkins, Ocana, Rao, & Collier, 1988). Unusual strenuous exercise and alcohol were avoided for the 24 hours prior to each trial (Brouns et al., 2005; Lee et al., 2006; Mettler et al.,

2007). Participants were advised to fast for 10 hours before arrival at the laboratory for the main trial (Brouns et al., 2005).

4.2.4 Trial procedures

Participants arrived at the laboratory between 0700 and 0900 hours on the trial days with the accelerometer being worn. The anthropometric measurements were taken. The BM measured included the mass of the accelerometer and light clothing. Participants consumed a glass of water (250 mL) to minimize dehydration and difference in the gastric emptying afterwards. They then waited 15 minutes for the test breakfasts in a quiet room (Kissileff et al., 1984).

Participants rated their levels of appetite sensations and physical comfort, and the impression of the breakfast via the VAS questionnaire prior to breakfast consumption. The same VAS questionnaire was rated again 20 minutes from the start of the test breakfast consumption (Brouns et al., 2005). All appetite related ratings were reported every 10 minutes postprandially for an hour at the laboratory (Cardello et al., 2005; Drapeau et al., 2007). Participants were allowed to read, sit and browse over the internet between ratings, with an exception of any activities related to foods and drinks (Cardello et al., 2005). Both trials took place in the same room in order to let participants focus on eating and reduce disruption from habitual satiation processes (Hetherington et al., 2006).



Keys:

- | | | | |
|----|--|-----|----------------|
| A | Arrival and anthropometric measurement | W | Water served |
| BF | Breakfast time | VP | Pre-meal VAS |
| L | Suggested lunch time | V20 | Post-meal VAS |
| D | Suggested dinner time | V | Subsequent VAS |
| S | Sleep time | VH | Hourly VAS |

Figure 4.1 Schematic representation of the study protocol on the trial day

Participants were instructed not to consume any foods or calorie containing drinks for two hours after leaving the laboratory (Mattes et al., 2005). They were advised to take lunch and dinner between 1200 and 1400 hours; and between 1800 and 2000 hours respectively so as to minimize any mealtime effect on rating the appetite sensation. The appetite ratings were reported on hourly basis for the rest of the trial day. Participants were asked to record the food intake and wear the accelerometer until bed time (Figure 4.1). An eating behaviour questionnaire (Appendix D) was completed during the trial period.

4.2.5 Test breakfasts

The majority of the items in the LGI and HGI breakfasts had GI values lower than 55 and over 71, respectively. The calculation of the GI values of the test breakfast was described previously in section 3.3. The overall calculated GI values for the planned LGI and HGI breakfasts were 42.2 ± 0.4 and 73.6 ± 0.2 respectively with a mean difference of 31.4.

The dietary composition of the meals was calculated using the commercial nutrient analysis software. The calculated CHO content was available CHO. The energy contributions from CHO, protein and fat were set as 60%, 15% and 25%, respectively. Plain yoghurt and strawberry yoghurt were alternatives for participants who did not take milk and rice milk in the LGI and HGI breakfasts, respectively. The test breakfasts matched the contents of energy, protein and fats (saturate, monounsaturated and polyunsaturated) by providing non CHO containing foods including macadamia, cheese, cherry tomatoes. The energy density was matched by adding water into liquid items (Table 4.1).

Table 4.1 The low glycaemic index (LGI) and high glycaemic index (HGI) food in the test breakfasts

LGI food items	HGI food items
Semi-skimmed fat milk ¹	Rice milk ⁵
Muesli ² mixed with fructose ³	Corn flakes ⁶ mixed with fruits & fibre ⁷
Dried apricot ⁴	Cherry tomato
Macadamia	Light cheese

¹ Tesco organic 1.7% semi-skimmed pasteurised milk; ² Alpen Swiss muesli; ³ Fruisana fruit sugar; ⁴ Tesco Ready to Eat Dried Apricots; ⁵ Rice Dream Organic Original Rice Milk; ⁶ Kellogg's cornflakes and ⁷ Tesco Fruit & Fibre

Based on the food preference questionnaire, no foods reported as 'No' were provided. The number of the variety of food item was limited to not more than four to reduce the effects of variety which are associated with the disruption of habituation (Hetherington et al., 2006).

4.2.6 Subjective sensations

The pre- and post-meal VAS questionnaires covered the questions for appetite (AS), physical comfort and palatability (PS). The AS AUC during period at the laboratory and throughout the trial days were calculated using the trapezoidal rule (Brouns et al., 2005; Mettler et al., 2007; Mettler et al., 2008). The postprandial AS AUC in mm·min at the laboratory and throughout the trial day were calculated to investigate the satiating effect of the test breakfasts on the subsequent EI (Drapeau et al., 2007).

4.2.7 Energy intake and energy expenditure

The dietary records and the accelerometry data recorded two days prior to each trial were treated as the EI and EE baselines, respectively. The detail of the method of estimation EI and EE were described in the section 3.4 and 3.5.1. Verbal and written instruction of the use of the accelerometer was given (Appendix M). The same serial accelerometer was provided to participants during the trial periods to minimize between-device variations whenever possible. The data were used to estimate the EE described in section 3.5.1. Participants were also required to complete the 24-hour physical activity log sheet (Appendix B) to fill the gaps from the limitations of accelerometry described in section 3.5.2.

4.2.8 Eating behaviour

The self-reported eating behaviour questionnaire (Appendix D) included 54 questions integrated from the DEBQ (Van Strien et al., 1986) and the 21-TFEQ (Cappelleri et al., 2009). The three types of eating behaviour described in section 3.7 were accounted as covariates.

4.2.9 Anthropometry

The anthropometric measurement was completed at arrival. The procedures were described in section 3.8.

4.2.10 Statistical analysis

4.2.10.1 Sample size calculation

The sample size was calculated based on the previous papers (Holt et al., 1992; Ludwig et al., 1999) at an alpha level of 0.05 with a power of 80% using the equation from National Research Council Committee (2003) (Appendix N). The sample sizes calculated to detect significant change of 26 mm in hunger rating (Ludwig et al., 1999) and 34 VAS unit·min in AUC of satiety over 180 minutes (Holt et al., 1992) were 10 and six, respectively. Brouns et al. (2005) recommended that at least ten participants should be involved to obtain sufficient statistical power in any research involving GI.

4.2.10.2 Data analyses

Descriptive statistics were calculated for group characteristics. A two-way ANOVA was used to test AS and EI for trial, time and trial by time effects. The AS AUC was calculated (mm·min) using Graphpad Prism® (GraphPad Prism

version 4.00 for Windows, GraphPad Software, San Diego, CA, U.S.). Differences in the AS AUC at the laboratory and in the free living condition; and the post breakfast PS between trials were assessed using paired sample *t*-tests. Pearson's correlation analyses were applied to examine the associations among AS, breakfast EI, total EI and PS. Multiple linear regression analyses, with adjustment of covariates, PS and type of eating behaviour, were used to predict postprandial AS by breakfast EI with respect to the GI. Any trial order effect, i.e. the first trial versus the second trial, was also examined. A z-score was calculated to substitute the missing VAS data. In total, 28 sets AS data were replaced out of the total 426 sets. All statistics were performed at the significant level of $\alpha = 0.05$.

4.3 Results

4.3.1 Characteristics of the participants

Thirteen participants initially enrolled in the study. One participant withdrew from the study after the first trial due to personal reasons. The data from this participant were excluded from analysis. Twelve recreationally active females completed the study successfully. The demographic characteristics of participants were shown in Table 4.2.

Table 4.2 Anthropometric and physiological characteristics of the participants

Variables	n = 12
Age (year)	28.2 ± 8.0
Body mass (kg)	62.4 ± 11.2
Body mass index (kg·m ²)	22.5 ± 3.2
Body fat percentage (%)	30.8 ± 4.8
Fat free mass (kg)	42.9 ± 6.0

Values are means ± SD; n, number of participants

4.3.2 Glycaemic index of meals

The actual GI values of the test breakfasts were calculated from the actual consumption after adjustment for any leftovers. No significant differences were found in the planned GI value of a test breakfast and the corresponding actual value. The GI values of the planned LGI and the actual LGI at breakfast remained significant lower than the HGI breakfast (Table 4.3).

Table 4.3 The planned and the actual glycaemic index of the test breakfasts

Meal	Planned LGI	Planned HGI	Actual LGI	Actual HGI	Difference in Actual GI
Breakfast	42.2 ± 0.4*	73.6 ± 0.2	42.5 ± 0.7*	73.5 ± 1.1	31 ± 1.5

Data presented as mean ± SD (n = 12). LGI = low glycaemic index; HGI = high glycaemic index; * significantly lower ($p < 0.001$) than the corresponding HGI GI values.

4.3.3 Subjective appetite sensation

No significant difference was found between the LGI and HGI pre-breakfast AS (74.3 ± 12.1 vs. 67.3 ± 16.8, $t(11) = 1.2$, $p = 0.27$). Under the laboratory

condition, the two-way repeated-measure ANOVA showed significant effects of trial ($F(1,11) = 8.0, p = 0.016$) and time ($F(2.2, 23.4) = 48.6, p < 0.001$); however there was no trial \times time effect ($p = 0.4$). Throughout the trial day, significant time effect remained ($F(4.6, 50.6) = 12.9, p < 0.001$) whereas only a trend of trial effect was observed ($F(1,11) = 4.1, p = 0.067$) and no trial \times time effect was found ($p = 0.1$) (Figure 4.2).

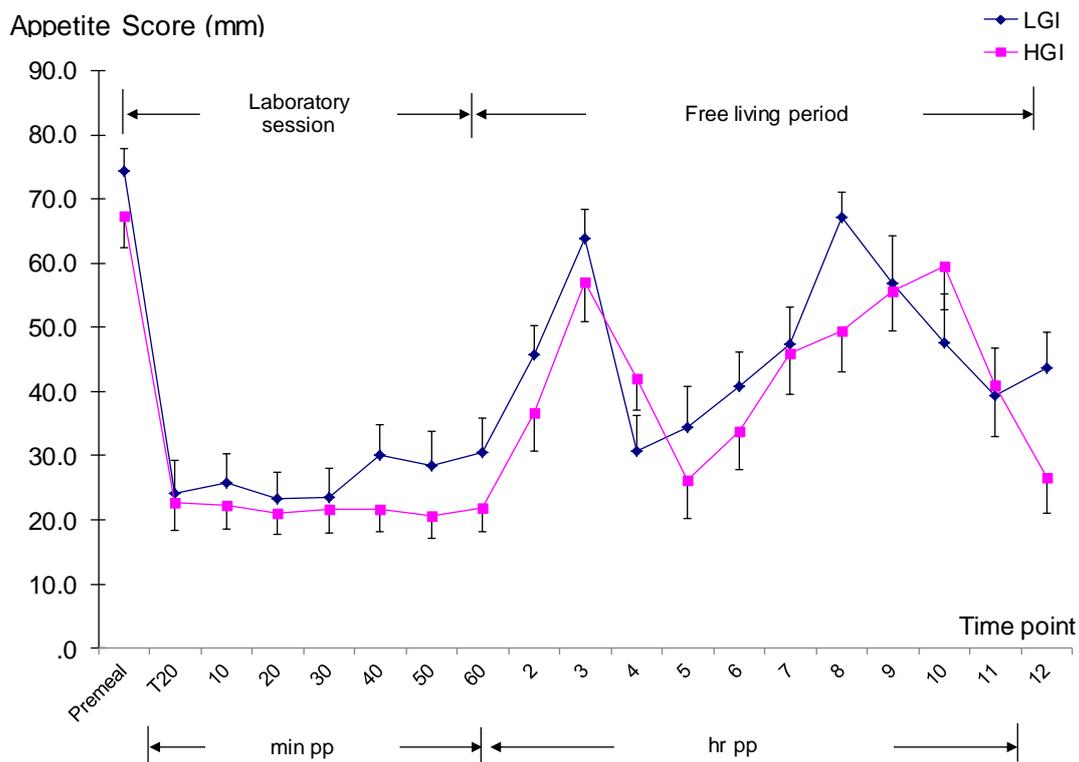


Figure 4.2 Effect of the low glycaemic index (LGI) and high glycaemic index (HGI) breakfasts on subjective appetite sensation

Data presented as mean \pm SD ($n = 12$); T20 = 20 minutes from the start of the breakfast; pp = post breakfast.

The AUC for AS following LGI was significantly greater than the HGI under the laboratory condition over 60 minutes ($2,568 \pm 1,027$ vs. $2,198 \pm 821$ mm·min, $t(11) = 2.6, p = 0.025$) but not throughout the trial day over 12 hours ($33,230 \pm 6,024$ vs. $30,490 \pm 5,465$ mm·min, $t(11) = 1.3, p = 0.2$). The AUC for AS

following LGI remaining significantly greater than the HGI for eight hours was detected ($21,278 \pm 3,610$ vs. $18,834 \pm 3,906$ mm·min, $t(11) = 2.5$, $p = 0.028$).

There was no significant effect of trial order on AS either under the laboratory conditions ($p = 1.0$) or throughout the trial days ($p = 0.4$). The order effect was not analysed for the subsequent variables.

4.3.4 Intake of macronutrients and energy and energy expenditure

Two sets of the LGI pre-trial and trial food records were missed, thus the number of data on the LGI trial was ten. Due to a technical fault with the accelerometer, eight sets of accelerometer reading could not be used. Thus the accelerometry data was not used for estimation of the EE. Alternatively the EE reported were estimated from the physical activity log sheet.

The two-way ANOVA showed that there were significant effects of time ($F(1,9) = 7.1$, $p = 0.026$) and trial \times time ($F(1,9) = 6.8$, $p = 0.028$) in the EI between trials on the pre-trial and trial days. The *post-hoc t* test showed that the LGI trial EI was significantly higher than the LGI pre-trial EI ($2,215 \pm 576$ vs. $1,748 \pm 464$ kcal, $t(9) = 3.8$, corrected $p = 0.008$) (Figure 4.3).

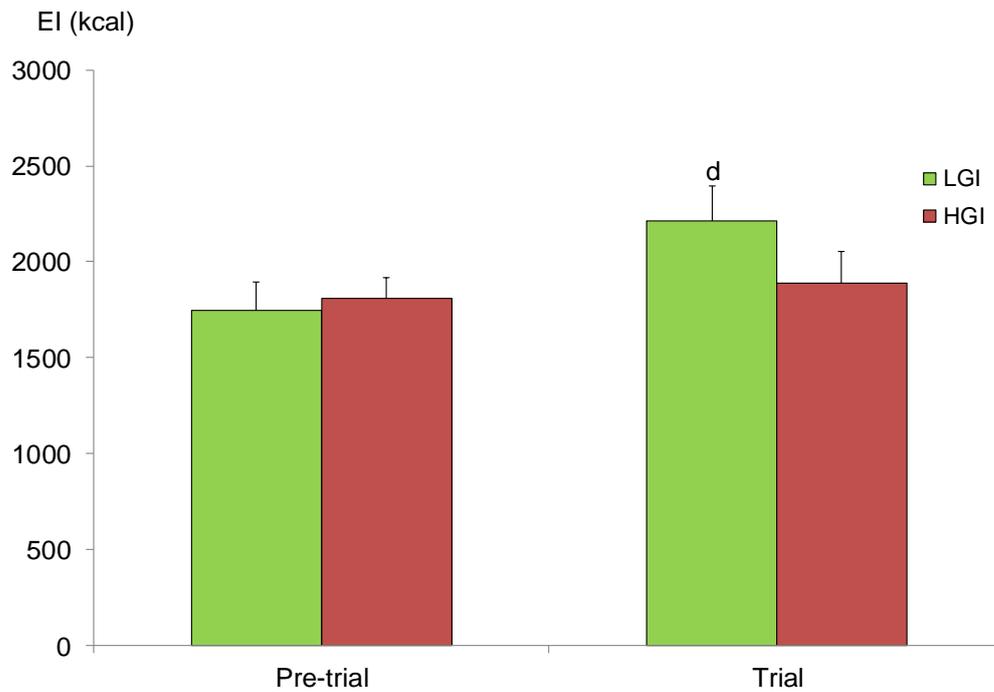


Figure 4.3 Energy intake (EI) between trials on the pre-trial and trial days

Data presented as mean \pm SD (n = 10); ^d significantly different to the corresponding pre-trial EI (corrected $p = 0.008$).

Table 4.4 The intakes of energy (EI) and the macronutrient as well as the energy expenditure (EE) on the pre-trials and trial days

Variables	Pre LGI	LGI trial	Pre HGI	HGI trial
CHO intake (g)*	231 \pm 77 ^b	278 \pm 79 ^{ab}	248 \pm 36	216 \pm 54 ^a
Protein intake (g)*	73 \pm 16	83 \pm 21	67 \pm 12	76 \pm 2.8
Fat intake (g)*	63 \pm 18 ^c	85 \pm 26 ^c	66 \pm 25	79 \pm 33
EI (kcal)*	1,748 \pm 464 ^d	2,215 \pm 576 ^d	1,808 \pm 338	1,886 \pm 531
EI per FFM (kcal·kg ⁻¹)*	42.3 \pm 11.9 ^d	53.9 \pm 15.5 ^d	43.6 \pm 7.9	45.1 \pm 11.3
EE (kcal)	2,192 \pm 597	2,358 \pm 507	2,315 \pm 535	2,358 \pm 488
EE per FFM (kcal·kg ⁻¹)	51 \pm 12.1	55 \pm 9	54 \pm 10.5	55.3 \pm 10.9

Data presented as mean \pm SD (n = 12 except * that n = 10 in the LGI); ^{a,b,c,d} significant differences sharing with the same subscript at the same row (corrected $p < 0.05$). CHO, carbohydrate; EI, energy intake; EE, energy expenditure; FFM, fat free mass; HGI, high glycaemic index; LGI, low glycaemic index.

There were no significant differences in the pre-trial intakes of macronutrients (CHO, protein, fat) between trials. A significant trial \times time interaction effect ($F(1,9) = 75.7, p < 0.001$) was found in the CHO intakes on the pre-trial and trial days (Table 4.4). The *post-hoc t* tests showed that the LGI trial CHO intake was significantly higher than the HGI trial CHO intake ($t(9) = 3.2$, corrected $p = 0.02$); and the LGI pre-trial CHO ($t(9) = 3.3$, corrected $p = 0.016$) (Figure 4.4). The LGI trial fat intake was higher than the LGI pre-trial fat intake ($t(9) = 3.1$, corrected $p = 0.026$). The two-way ANOVA did not find any significant effects of trial, time and trial \times time interaction in the EE (Table 4.4).

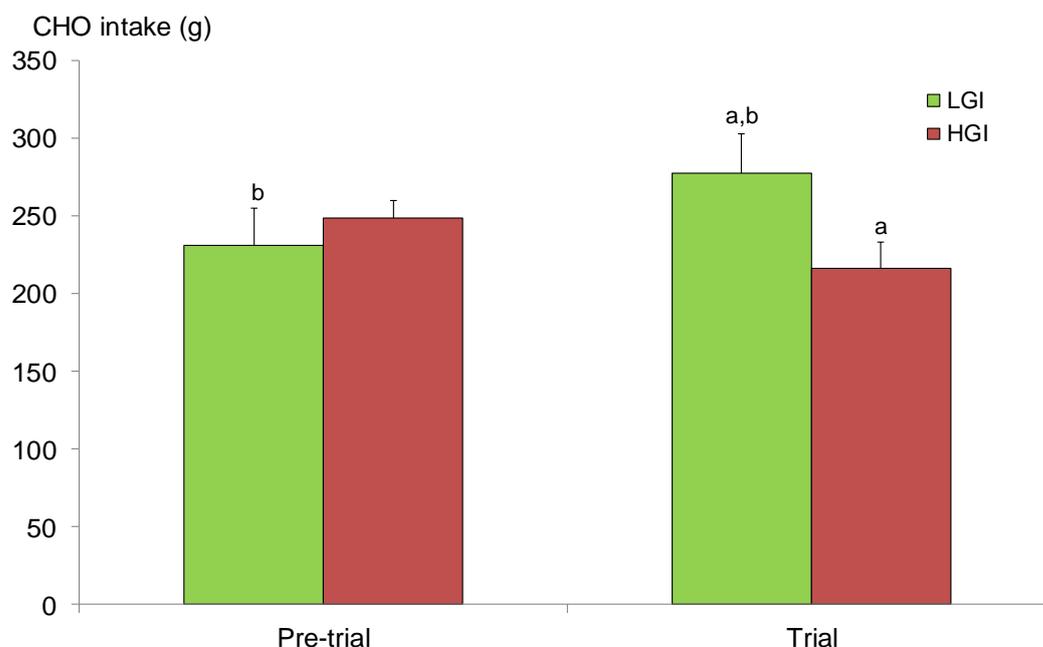


Figure 4.4 Carbohydrate (CHO) intakes between trials

Data presented as mean \pm SD ($n = 10$); ^{a,b} significant differences existed with the same subscript (a: corrected $p = 0.02$, b: corrected $p = 0.016$).

4.3.5 Palatability

No significant differences were found in the post breakfast PS between trials ($t(11) = -0.37, p = 0.7$). Pearson's correlation did not show any correlation

between the post breakfast AS and the post breakfast PS in either the LGI ($p = 0.4$) or the HGI trial ($p = 0.3$).

4.3.6 Relationship between body composition and energy intake

When both trials were included, Pearson's correlation showed a significant negative correlation between the post breakfast AS and the breakfast EI which explained 18.0% of variance ($p = 0.039$). Body mass and FFM had significant positive associations with EI, respectively. No significant correlations were found after the trials were separated.

When both trials were included, the linear regression analysis showed that the post breakfast AS was predicted by the breakfast EI after the adjustment of AS for body composition variables using the following equation: post breakfast AS = $-396 - 0.14.5 \times \text{BM} + 11.9 \times \text{body fat percentage} + 22.9 \times \text{FFM} - 0.06 \times \text{Breakfast EI}$ (SEE = 12.3, $p = 0.002$). The breakfast EI explained 34.8% of variance in post breakfast AS after adjustment of these body composition variables ($p = 0.002$).

4.4 Discussion

The findings did not support the hypothesis that the GI manipulations would give rise to different subjective appetite ratings, with LGI foods expected to exert greater appetite suppressing power than the HGI foods (Arumugam et al., 2008; FAO, 1998; Ludwig et al., 1999; WHO/FAO, 2003). The current study found that the HGI breakfast suppressed appetite in comparison to the LGI

breakfast in recreationally active females. The significantly lower AS AUC and the trial effect of the AS following the HGI breakfast indicated that the appetite satiating effect of the HGI breakfast was sustained for eight hours post breakfast. The significant interaction effect in the EI illustrated a significantly higher EI on the trial day compared to the pre-trial EI in the LGI trial, although the EI on the trial days between trials were comparable.

4.4.1 Appetite and energy intake

The current findings are not the only evidence contradicting the general belief of the higher satiating effects of LGI foods compared to the HGI foods. Previously Anderson et al (2002) demonstrated that 75 g glucose drink suppressed the appetite for one postprandial hour when compared to 75 g fructose-glucose (80/20) mixture drink in non-overweight males. The subsequent EI was positively associated with the 60-min postprandial AS; and was negatively associated with the 60-min glucose AUC. The results suggested that the higher the pre-meal AS, the higher the subsequent EI. Furthermore the authors found a negative association between the postprandial AS at 60 minutes and the 60-min glucose AUC ($r = -0.24$, $p = 0.05$) which implicated that higher glucose levels suppressed the appetite in healthy adults (Anderson et al., 2002).

The intakes of energy and the CHO on the LGI trial day were significantly higher than on the pre-trial day as well as on the HGI trial day in the current study. Most CHO rich foods commonly consumed in western countries, for instance in the U.S. and Canada, are HGI foods in recent decades (Ludwig, 2000, 2002; Ludwig et al., 1999). A crossover study found that female participants had

higher percentage of CHO intake of LGI foods than that of the HGI foods *ad libitum* for 12 weeks despite insignificant difference in EI (Aston et al., 2008). One speculation is that if the habitual choices of participants were HGI foods, LGI test breakfasts provided on the trial day indicated a change from habitual HGI to test LGI. This speculation implicated that a change from a habitual HGI breakfast to a LGI breakfast resulted in an increase in the intakes of energy and CHO on the LGI trial day and the total EI intake was maintained following the HGI breakfast. The speculation remains open until the GI values of pre-trial CHO foods can be examined. Dietary data collection software with GI values has been developed in the U.S. (Schakel et al., 2008); however the data base might not be applicable to the U.K. diets. Currently there is no locally developed dietary analysis software with GI values for examining the GI values of foods taken on the pre-trial and the trial days. Without further empirical evidence, it remains unclear if the lack of significant difference in the EI between trials was due to large standard deviation or this speculation. Nevertheless, the significant interaction effect in the EI that the EI on the trial day was significantly higher than the pre-trial EI in the LGI trial should not be ignored. Not only the intake but also the GI values should have been involved when evaluating associations between diets and body weight and non-communicable diseases outcomes in future (Louie et al., 2011; Olendzki et al., 2006).

Raben (2002) reviewed 15 published studies examining the GI effect on EE, of which nine studies found the increases in the EE following LGI foods. Krog-Mikkelsen et al. (2011) speculated that any GI effect on energy balance, if present, would result from the change in appetite and EI; but not from EE.

Adapting a HGI breakfast suppressed the appetite for a couple of post breakfast hours better than a LGI breakfast, despite a failure to a decrease in total voluntary EI in the current study. The appetite suppression induced by a single HGI meal a day might not therefore be sufficient to change the total EI (Pal et al., 2008). Therefore, further research is necessary to determine whether the suppression of appetite and reduction in EI observed in the HGI trial in the short term in the present study, would be sustained in the longer term.

4.4.2 Impact of body composition on appetite

Anderson et al. (2002) found the short-term satiating effect by HGI CHO in participants with normal BMI whereas the previous studies finding greater appetite suppression lasted for 4–5 hours post LGI breakfast having overweight participants (Arumugam et al., 2008; Ludwig et al., 1999). The discrepancy might be due to the health conditions and body compositions of participants. A recent study showed that FFM, but not fat mass or BMI, was associated with meal size and EI (Blundell et al., 2012b). The authors found that total daily EI was positively correlated with FFM in 41 overweight and obese adults ($R^2 = 0.35$). Blundell et al. (2012a) proposed that FFM was the major determinant, among several body composition related variables, of the EI at a free living environment. The current study also found that FFM was positively correlated with daily EI independently to the GI values. Another recent study found a significant positive correlation between BMI and insulin AUC difference between two meals with different postprandial glucose responses (Ramel et al., 2012). The authors concluded that the greater difference in postprandial insulin response was associated with an increase in BMI. Thus any study related to effect of GI on appetite and total EI should have homogenous group of

participants with similar body composition to minimize any confounding factors from these variables.

4.4.3 Strengths and limitations

The plausibility of the dietary record is always considered as a potential limitation in epidemiological studies (Stubbs et al., 2001). A subsample of 312 women in the Third National Health and Nutrition Examination Survey in the U.S. providing two 24-hour dietary recalls with one month apart during 1988–1994 showed that 58% of women who under-reported on the first occasion also under-reported after one month (Livingstone & Black, 2003). One quarter of the food records in the current study were under-reported using the Goldberg cut-off (data not shown). Nevertheless, participants acted as their own controls in this randomised repeated measured design study which implicated a relative, rather than an absolute, magnitude of the EI estimate. Thus the strength here was any underestimation of daily EI was expected to affect both trials equally and not therefore confound the comparison between trials.

Flint et al. (2004) queried the accuracy of determining the GI values of a mixed meal via calculation. The authors did not find an association between the predicted GI and measured GI. In addition, they claimed that the GI value of mixed meals was more strongly correlated with fat and protein than CHO alone. The authors measured venous glucose levels which is known to be less sensitive to glycaemic responses and have greater within-subject variation than arterial glucose levels (Granfeldt et al., 2006; Wolever et al., 2003). The macronutrient distribution and the energy density of a meal can alter energy

balance (Blundell et al., 2012a). The current study carefully matched energy, CHO, protein, fats and fibre contents; and energy density between breakfasts. The matched test breakfasts minimized any confounding factors on predictability of GI of mixed meals. Moreover, the trial order effect had also been examined and no significant difference in the AS was found between the first and second trials. Any differences in the satiating effect being observed here can be confidently attributed to the GI.

Besides controlling the protein and fat contents to minimize their effects on the postprandial glycaemic and insulinaemic responses, other non-energy containing foods such as vinegar was also minimised in the current study as it is also known to reduce the GI values (Ramel et al., 2012). Schakel et al. (2008) reported that the calculated GI values using the mixed-meal method of Wolever and Jenkins (1986) tended to be lower than values obtained in vivo for some unsweetened breakfast cereals. On the contrary, the calculated GI values were higher than the known literature values in vivo for several types of dairy products such as sweetened yogurt. The deviation between the mean calculated GI and the mean analysed GI in 102 multi-ingredient foods was less than 5% (Schakel et al., 2008). Strawberry yogurt was an alternative to rice milk in the HGI breakfast in the current study; therefore the GI values of the HGI test breakfast might be even higher than the analysed method which implicated that the difference in the GI values between trials may be greater than the current calculated values.

Currently there are not enough published data of GI values for every single food, particularly for a mixed meal. Wolever and Jenkins (1986) developed a calculation method to predict the GI values of mixed meals. Thus it seems not to be feasible to access the GI value of test meals with sufficient sample size prior to the trial if the confounding variables are well controlled. In addition, the daily intra-subject variation in the glycaemic response was reported to be greater than the inter-subject variation in the glycaemic response (Wolever et al., 2003). Although measuring the postprandial glycaemic responses can verify the GI values of the trial meals, the aim of the current study would then have been distracted to become a validation of the GI values of mixed meals against the known literature values. Highly processed grains and cereal products may deviate the actual GI values. The current study selected natural or minimally processed foods to minimize any potential discrepancy and provide more consistent GI values matched to the published databases.

The current study has some other limitations. The sample size was based on a power calculation using the appetite score effect size and might not therefore be sufficient to detect EI effects. In an attempt to control any effect of variation in the pre-trial evening meal on outcomes, participants were requested to replicate the pre-trial evening food choice and portions. However it is not possible to ensure compliance in the free-living setting. Besides the known dietary factors mentioned previously, there existed other factors such as the eating behaviour like the mastication rate, the meal consumption time and the frequency of food consumption that lead to the intra-subject variation (Flint et al., 2004; Wolever, Jenkins, Ocana, et al., 1988). Sleeping quality prior to the trial day also has an influence on the insulin sensitivity which may affect postprandial glycaemic

responses (Wolever, Leung, Vuksan, & Jenkins, 2009). The GI value appears to be affected by insulin sensitivity and the level of physical activity of participants (Brouns et al., 2005; Mettler et al., 2007; Mettler et al., 2008; Mettler et al., 2006). Recreationally active females were recruited for the present study; whether the findings were applicable to male needs further investigation. The current study design should be applied to males and with larger samples so as to further investigate the GI effect on EI.

It has been a decade since Foster-Powell et al. (2002) published the first GI table systemically. A time gap is present here such that the GI values might differ from the published GI values due to the changes in the manufacturing or processing conditions; or the formulas of commercial products over the period while the published GI values have not been updated (Ludwig & Roberts, 2006).

It has been well known that HGI foods result in rapid and higher increases in blood glucose and insulin than do LGI foods (Jenkins et al., 1981). Previously a negative correlation was found between the postprandial insulin AUC response and *ad libitum* food intake in two hours (Holt, Brand Miller, & Petocz, 1996). The authors suggested that high insulin response was associated with less food intake. Flint et al. (2007) suggested that postprandial insulin response may be a better predictor of satiety than postprandial glucose level in a meta-analysis study. Glucose and insulin are closely correlated with each other and thus insulin might also play a role in appetite suppression. However blood glucose, insulin and hormonal response were not analysed to investigate any underlying mechanism of GI on appetite sensation in the current study. This is an

empirical study investigating the GI effect at breakfast on appetite in recreationally active females. Biochemical analysis may provide more information on understanding the influence of GI on appetite and EI which were assessed in Chapter 6.

4.5 Conclusion

The study showed that HGI CHO rich breakfast facilitates a stronger appetite suppressing effect in the postprandial phase in recreationally active females than following LGI CHO rich breakfast. The claim that the LGI foods have specific effects on weight and food intake control is not applicable to recreationally active females. The effect found here should not be generalized without further empirical evidence. The appetite suppressing effect by a single HGI breakfast might not be strong enough to reduce the total EI. Further investigation by providing HGI meals for a whole day is required to examine whether the advantage of HGI foods on appetite suppression persists in the longer term in the free living environment. Examining the relationship among [Glucose], appetite and subsequent intake can also help to understand the GI impact of HGI CHO rich foods on appetite and EI in active individuals.

CHAPTER 5 STUDY TWO: EFFECT OF LOW AND HIGH GLYCAEMIC INDEX MEALS ON APPETITE SENSATION AND ENERGY BALANCE IN RECREATIONALLY ACTIVE MALES

(Published paper: Glycaemic index of meals affects appetite sensation but not energy balance in active males (Appendix A))

5.1 Introduction

Appetite, including satiation and satiety, could potentially be altered by varying the GI values of ingested meals (De Graaf et al., 2004; Fischer et al., 2004; Kissileff et al., 1984). The rationale of the advantage of LGI diets for weight management is due to its potential satiating effects leading to a reduction of subsequent EI. However, Anderson et al. (2002) found that HGI foods suppressed subjective appetite leading to lower subsequent EI than following LGI foods in non-obese healthy males. Study one (Chapter 4) also found that HGI CHO rich breakfast facilitates a stronger appetite suppressing effect in the postprandial phase in recreationally active females than following LGI CHO rich breakfast. The lower subsequent EI in the study of Anderson et al. (2002) was assessed 60 minutes post breakfast which was not ecologically valid in the real practice. To date the effect of GI on appetite and subsequent EI at least three hours post breakfast has not been investigated in recreationally active males.

The purpose of this study therefore was to compare the short term effects of LGI and HGI meals over a day on subsequent subjective appetite sensation, EI, EE, energy balance and RMR in recreationally active males. The objectives were to determine and compare the difference between trials in:

- subjective appetite sensation one hour after breakfast in the laboratory and throughout the rest of the trial day in a free living setting;
- and energy balance in the trial days.

It was hypothesized that HGI meals would suppress appetite and lead to a negative energy balance compared to LGI meals in a trial day.

5.2 Methodology

5.2.1 Participants

Participants were briefed that the study was to investigate general aspects of meals on appetite sensation; but not the specific purpose of investigating the effects of GI on appetite. They were screened by the EAT-26 (Appendix C) for eligibility. Additional inclusion criteria were Caucasian male aged between 18 and 50 years and with EAT-26 score below 20 (Garner et al., 1982).

5.2.2 Study design

The design of the study was to examine the effects of LGI or HGI meals on the subjective appetite sensation and energy balance in recreationally active male adults over a single trial day. Participants ingested either a LGI or HGI breakfast at the laboratory and then left with a take-away bag containing test foods and drinks for lunch, dinner and snacks. They returned to the laboratory the next morning for a post-trial RMR measurement. Participants received the alternative test meals after a wash-out period of at least two days (Brouns et al., 2005; Warwick et al., 1993).

5.2.3 Pre-trial arrangement

Participants recorded two days of food and drink intake prior to the first trial for estimation of the habitual EI. The energy contents of the test meals were based on the habitual EI of each participant. Two days prior to each trial day, they were required to record food intakes and to wear the GT1M accelerometer. Participants were instructed to keep similar physical activities and dietary patterns; and refrain from unusual strenuous exercise for the 24 hours preceding each trial day (Mettler et al., 2007). No alcohol was permitted during this period (Brouns et al., 2005). In order to minimize any evening meal effect on RMR and appetite on the trial days, participants were instructed to consume a similar meal the evening before every trial (Brouns et al., 2005; Mettler et al., 2007). Participants were required not to eat or drink anything for 10 hours before arrival at the laboratory for each trial day (Brouns et al., 2005).

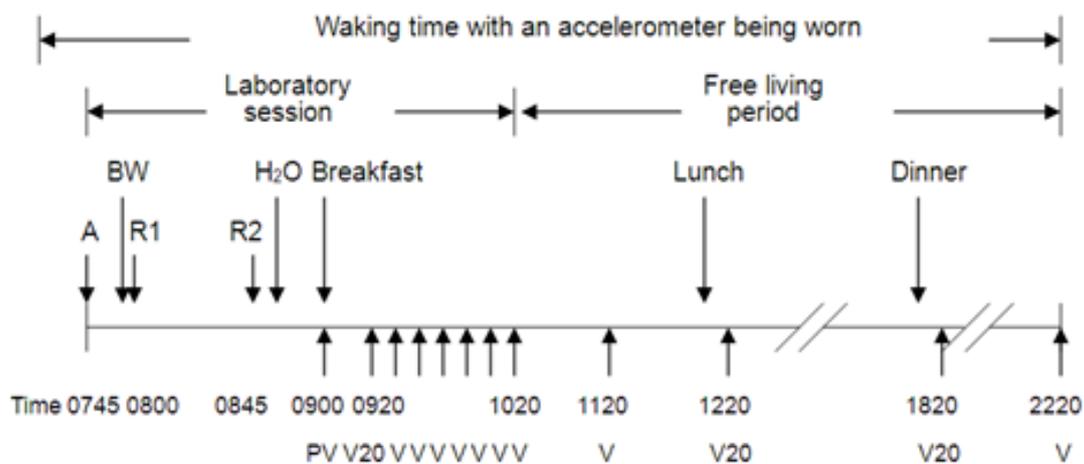
5.2.4 Trial procedures

Participants arrived at the laboratory between 0700 and 0900 hours on the trial days after an overnight fast for 10 hours with the accelerometer being worn. Body mass and body stature were taken wearing light clothing. Participants then lay in a supine position on a tilt table for 45 minutes for the RMR measurement. A glass of water (250 mL) was served to participants afterwards to minimize dehydration and any difference in the gastric emptying before the consumption of the test breakfast. Participants then stayed in a quiet room for 15 minutes for breakfast serving (Kissileff et al., 1984).

Participants rated their levels of appetite, physical comfort, and impression of the breakfast on the VAS questionnaire before consuming the breakfast. Participants were required to consume the breakfast within 20 minutes (Brouns et al., 2005). Water was provided *ad libitum* with the breakfast. The same VAS questionnaire was completed immediately following breakfast, which was 20 minutes from the start of the meal. All VAS for appetite sensations were rated every 10 minutes postprandially for an hour (Cardello et al., 2005; Drapeau et al., 2007). Participants were allowed to read, relax and browse over the internet between ratings, with an exception of any reference to literature related to foods and drinks (Cardello et al., 2005). Participants remained in the same room alone in both trials in order to let them focus on eating, to reduce social influences or any disruption from habitual satiation processes (Hetherington et al., 2006).

Participants were provided with packed test foods and drinks before departure. A free food list was enclosed for participants to top up extra foods when they felt hungry after all foods of each meal provided had been consumed (Pereira, Swain, Goldfine, Rifai, & Ludwig, 2004). Non-calorie containing drinks such as water, tea and coffee were allowed *ad libitum*. On leaving the laboratory, participants were required to rate their appetite on the VAS on an hourly basis for the rest of the trial day until bed time plus the VAS for palatability after lunch and dinner. Participants were instructed to take lunch and dinner between 1200 and 1400 hours; and between 1800 and 2000 hours respectively, to minimize any mealtime effect on appetite sensation rating. Participants were free to consume the provided snacks two hours after each main meal (Figure 5.1).

Participants returned to the laboratory the same time the next day after the overnight fast. A 45-min post-trial RMR was measured. The anthropometric measurements were completed before departure. Participants continued to record the food intake and to wear the accelerometer during the waking hours for the day following each trial.



Keys:

- | | | | |
|------------------|-----------------------|-----|---------------------------|
| A | Arrival | R1 | RER measurement started |
| BW | Body weight | R2 | RER measurement completed |
| H ₂ O | 250 mL water intake | PV | Pre-meal VAS |
| L | Suggested lunch time | V20 | Post-meal VAS |
| D | Suggested dinner time | V | Subsequent VAS |

Figure 5.1 Schematic representation of the study protocol on the main trial day

5.2.5 Anthropometry

The anthropometric measurement was completed at arrival on the post trial day of the first trial. The procedures were described in section 3.8.

5.2.6 Test meals

The acceptability of food was assessed by a food acceptability questionnaire (Appendix I). The energy content of the test meals was estimated from the two-day food records of each participant prior to the first trial using the nutrient analysis software. The two breakfast meals differed in GI values but matched macronutrient contents. The ratio of energy contribution from CHO:protein:fat ratio was 60:15:25. Yoghurt and strawberry yoghurt were alternatives for participants who could not take milk and rice milk respectively. Macadamia, low fat cheese, cherry tomatoes were added to match the fibre, the protein and the fat (saturated, monounsaturated and polyunsaturated) contents between breakfasts. Water was added to match the caloric density of the two test breakfasts (Table 5.1). Both breakfasts were presented with not more than four items with the number of food items matched to reduce the effects of variety which are associated with the disruption of habituation (Hetherington et al., 2006).

The energy content of the food and drink bags following breakfasts for the rest of the trial days was calculated from the difference in the total energy content and the energy contributed from breakfasts. Each food bag packed with lunch, dinner and snack. A cooking instruction sheet for dinner was provided for participants to minimize the cooking effects on GI values. The energy contribution from protein at lunch and dinner remained 15%; and from CHO and fat were 65% and 20%, respectively. The majority CHO rich foods had GI equivalent to 55 or below for the LGI and to 65 or above for the HGI trials. Non CHO containing foods including beef, tuna, vegetables, nuts, cheese, oil or fats were added to standardize the fibre, protein and fat (saturated,

monounsaturated and polyunsaturated fats) contents between the test meals (Table 5.1). Diet drink was provided to match the energy density of meals and snacks between trials. All meals and snacks were freshly prepared and packed in the morning of each main trial day; and kept in the refrigerator until departure. The brands and the nutrition labels of the foods and drinks were either torn away or covered to make participants difficult to estimate their EI. Participants were encouraged to consume all provided foods. Nevertheless they did not have to consume all if they were full. Conversely, after consumption all provided foods at each meal time, participants could add foods selected from a provided list (Appendix O) such as green leafy vegetable and tomatoes.

Table 5.1 An example of the low (LGI) and high (HGI) glycaemic index meal plans for a participant requiring 2,255 kcal

LGI breakfast			HGI breakfast		
Items	GI	Quantity (g)	Items	GI	Quantity (g)
Semi-skimmed milk ¹	32	341	Rice milk ⁵	79	195
Muesli ²	55	60	Corn Flakes ⁶	93	25.2
Dried apricots ⁴	32	26	Fruit 'n Fibre ⁷	67	50
Fructose ³	19	13.4	Light cheese		42.8
Macadamia		6.2	Macadamia		5.1
Water		31.4	Tomatoes		160
	41.9	478		72.6	478

Table 5.1 (Continued)

LGI lunch			HGI lunch		
Items	GI	Quantity (g)	Items	GI	Quantity (g)
White tortilla ⁸	30	146.5	Wholemeal bread ¹⁰	74	137
Apple juice ⁹	44	330	Glucose drink ¹¹	93	380
Dried apricots	32	21	Cucumber		65
Tuna in brine		46.6	Tuna in oil		28.5
Almonds		7.4	Almonds		6.2
Water		72.2	Butter		7
	34.4	624		82.9	624
LGI dinner			HGI dinner		
Items	GI	Quantity (g)	Items	GI	Quantity (g)
Fusilli ¹³	54	70	New potatoes ¹⁵	80	220
Apple juice	44	330	Glucose drink	93	380
Milk Chocolate ¹⁴	40	28.5	Plain Chocolate ¹⁶	49	30
Dried apricots	32	38	Spinach		92
Roasted Beef		50	Roasted Beef		65.6
Almonds		5.5	Almonds		7.3
Diet drink		272			
Olive oil		1.3			
	46.1	795		80.4	795

Table 5.1 (Continued)

LGI snack			HGI snack		
Items	GI	Quantity (g)	Items	GI	Quantity (g)
Canned peaches ¹⁷	40	190	Canned lychees ¹⁹	79	304
Apple juice	44	330	Strawberry yogurt ¹²	85	125
Fructose	19	15.4	Cheese		40.5
Natural yogurt ¹⁸	35	42	water		150
Cheese		42			
	37.4	619		80.5	620

¹ Tesco organic 1.7% semi-skimmed pasteurised milk; ² Alpen Swiss muesli; ³ Fruisana fruit sugar; ⁴ Tesco Ready to Eat Dried Apricots; ⁵ Rice Dream Organic Original Rice Milk; ⁶ Kellogg's cornflakes; ⁷ Tesco Fruit & Fibre; ⁸ Discovery Mexican Plain Flour Tortillas; ⁹ Tesco Apple Juice from Concentrate; ¹⁰ Hovis Medium Sliced Wholemeal Bread; ¹¹ Lucozade energy original; ¹² Tesco Everyday Value Low Fat Strawberry Yogurt; ¹³ Tesco tricolour fusilli; ¹⁴ Cadbury Dairy Milk; ¹⁵ Tesco new potatoes; ¹⁶ Cadbury Bournville Plain Chocolate; ¹⁷ Tesco Peach Slices in Juice; ¹⁸ Tesco Low Fat Natural Yogurt; ¹⁹ Princes Lychees in Syrup

The mean planned GI values of breakfast, lunch and dinner between the LGI and HGI trials were 41.1 ± 1.2 vs. 74.1 ± 1.9 ; 34.3 ± 0.7 vs. 81.2 ± 2.3 and 45.1 ± 1.9 vs. 77.0 ± 2.8 with differences of 33, 47 and 30, respectively. The overall calculated GI values for the LGI and HGI trials were 39.6 ± 0.7 and 76.9 ± 2.8 respectively with a difference in mean of 37.3 ± 2.7 . Based on the food preference questionnaire, no food scored as 'dislike very much' was provided (a 1 on a 5 point Likert Scale).

5.2.7 Subjective appetite sensations

Subjective appetite sensation (AS); physical comfort, as well as, palatability (PS) were assessed using the 100-mm VAS. The intra-variation using the pre-meal

AS at the first and the second visit was calculated as 13.8%. This coefficient of variation was considered as within an acceptable range compared to the coefficient of variation of 9–21% found in a previous study assessing the reproducibility of VAS on appetite sensation (Flint et al., 2000).

5.2.8 Energy intake, energy expenditure and energy balance

Participants were briefed about the format of recording intakes and to provide detailed description of food and drink items being consumed including mealtime, location, portion and brand name (Rennie et al., 2007). A research dietician went through the food records and clarified any unclear or missing information with participants. Food records were analysed using the commercially available nutrient analysis software to estimate EI described in section 3.4.

The expired air was collected from a face mask and sample line connected to the real time gas analyser (Cortex Metalyser 3B, Leipzig, Germany). Participants were instructed to remain awake in a supine position on a tilt table in a quiet and temperature and humidity controlled room for 45 minutes after wearing a gas collection mask (Korth et al., 2007). The calibration procedures and the calculation of CHO and fat oxidation for estimating the RMR were described in section 3.5.3.

Participants were required to wear the accelerometer for estimation of the baseline EE two days prior to each trial day, on the trial as well as post trial days. The same serial accelerometer was provided to participants during the

trial periods to minimize between-device variations wherever possible. The data were used to estimate the EE described in section 3.5.1. The energy balance was calculated from the difference of EI and EE.

5.2.9 Eating behaviour

Participants completed the eating behaviour questionnaire (Appendix D) in order to assess the eating behaviours of participants during the trial period. The detail of the questionnaire was described in section 3.7.

5.2.10 Statistical analysis

5.2.10.1 Sample size calculation

The calculation of the sample size was based upon two experimental papers (Holt et al., 1992; Ludwig et al., 1999) using the equation from National Research Council Committee (2003) (Appendix N) described in section 4.2.10.1. A required sample size of 10 was suggested to achieve a statistical power of 80% at an alpha level of 0.05.

5.2.10.2 Data analyses

Descriptive statistics were calculated for group characteristics. A two-way ANOVA was used to test for trial effect, time effect and trial by time interaction on the mean AS and EI. The AS AUC was examined (mm·min). Pearson's correlation analyses were applied to examine the associations between appetite, breakfast EI, lunch EI, dinner EI and total EI; and palatability. Multiple linear regression analysis was used to determine the predictive value of EI for the post meal AS with an adjustment for PS as a covariate. Differences between trials in

the AS AUC under the laboratory and the free living conditions; and the post meal PS were assessed using paired sample *t*-tests. Any trial order effect was also examined. A z-score was calculated to substitute the missing VAS data. Fourteen sets of AS data were replaced out of total 588 sets. All statistics were performed at a two-tailed level of the significance at the 95% or 0.05.

5.3 Results

5.3.1 Characteristics of the participants

Fifteen participants initially enrolled for the study. One participant withdrew from the study after the first trial due to sickness. The data collected from this participant were excluded from analysis. Another participant who smoked occasionally was still recruited in the study but was reminded not to smoke on the day prior to and each trial day. Fourteen recreationally active males completed the study successfully (Table 5.2).

Table 5.2 Anthropometric and physiological characteristics of the participants

Variables	Participants (n = 14)
Age (year)	34.5 ± 8.9
Body mass (kg)	71.9 ± 10.6
Body mass index (kg·m ⁻²)	22.8 ± 2.1
Body fat percentage (%)	20.7 ± 4.8
Fat free mass (kg)	56.9 ± 7.6

Data presented as means ± SD; n = number of participants.

5.3.2 Glycaemic index of the meals

The actual calculated GI values of each meal were determined after accounting for the final actual food consumption. The GI values of the planned LGI and the actual LGI at each main meal and in overall remained significantly lower than those of the corresponding HGI mealtimes. The actual HGI breakfast GI value was significantly higher than the planned ($t(13) = 2.2, p = 0.05$). No significant differences were found in the GI values between the planned and the actual values at lunch and at dinner in both trials (Table 5.3).

Table 5.3 Planned and actual glycaemic index values of the test meals

Meal	Planned LGI	Planned HGI	Actual LGI	Actual HGI	Difference in actual GI
Breakfast	41.1 ± 1.2*	73.9 ± 1.7**	41.3 ± 1.4*	74.3 ± 1.4	33.1 ± 2.2
Lunch	34.3 ± 0.8*	81.2 ± 2.3	34.1 ± 1.1*	81.4 ± 2.9	47.3 ± 3.1
Dinner	45.1 ± 1.9*	76.9 ± 2.8	44.9 ± 2.1*	75.1 ± 4.6	30.3 ± 3.4
Overall	39.6 ± 0.7*	76.9 ± 2.8	39.6 ± 1.0*	76.2 ± 2.8	36.6 ± 2.9

Data presented as mean ± SD (n = 14). LGI = low glycaemic index; HGI = high glycaemic index; *significantly lower than the corresponding HGI mealtimes ($p < 0.001$); ** significantly lower than the actual HGI GI ($p = 0.05$).

5.3.3 Appetite sensation

There was no significant difference between trials in the AS prior to breakfast respectively (79.3 ± 10.1 vs. $77.6 \pm 14.0, t(13) = 0.41, p = 0.69$). Independently to the GI values, the post breakfast AS decreased significantly compared to the pre-breakfast AS (78.4 ± 12.0 vs. $31.7 \pm 18.0, t(27) = 11.9, p < 0.001$). No trial x time interactions were found under the laboratory condition ($p = 0.2$) and throughout the trial day ($p = 0.3$). There existed significant main trial effects under the laboratory condition ($F(1,13) = 6.0, p = 0.03$) and throughout the trial

day ($F(1,13)= 13.5, p = 0.003$). Significant time effects were also found under the laboratory condition ($F(2,26.5) = 54.5, p < 0.001$) and throughout the trial day ($F(4.5, 57.9) = 10.2, p < 0.001$). The average AS immediately after the three HGI main meals (breakfast, lunch and dinner) was significantly lower than that in the LGI trial (27.2 ± 12.5 vs. $34.0 \pm 15.6, t(13) = -2.5, p = 0.024$) (Figure 5.2).

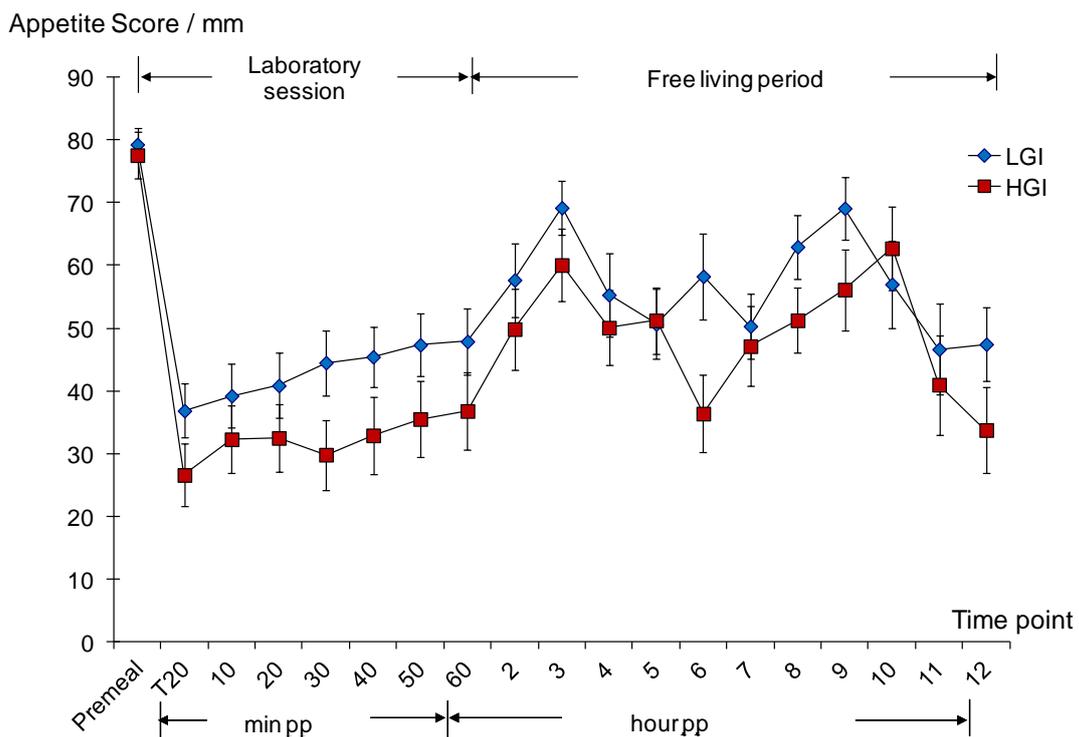


Figure 5.2 The effect of GI values on subjective appetite sensation

Data presented as mean \pm SD ($n = 14$). LGI = low glycaemic index; HGI = high glycaemic index; T20 = 20 minutes following the breakfast; pp = postprandial.

The HGI AS AUC was significantly smaller than the LGI AS AUC under the laboratory condition over 60 minutes ($2,989 \pm 1,390$ vs. $3,758 \pm 1,290$ mm·min, $t(13) = -2.5, p = 0.027$) and throughout the trial day over 12 hours ($35,454 \pm 9,730$ vs. $41,244 \pm 8,829$ mm·min, $t(13) = -3.1, p = 0.009$) (Figure 5.3).

No trial order x time interactions were found under the laboratory condition ($p = 0.4$) and throughout the trial day ($p = 0.7$).

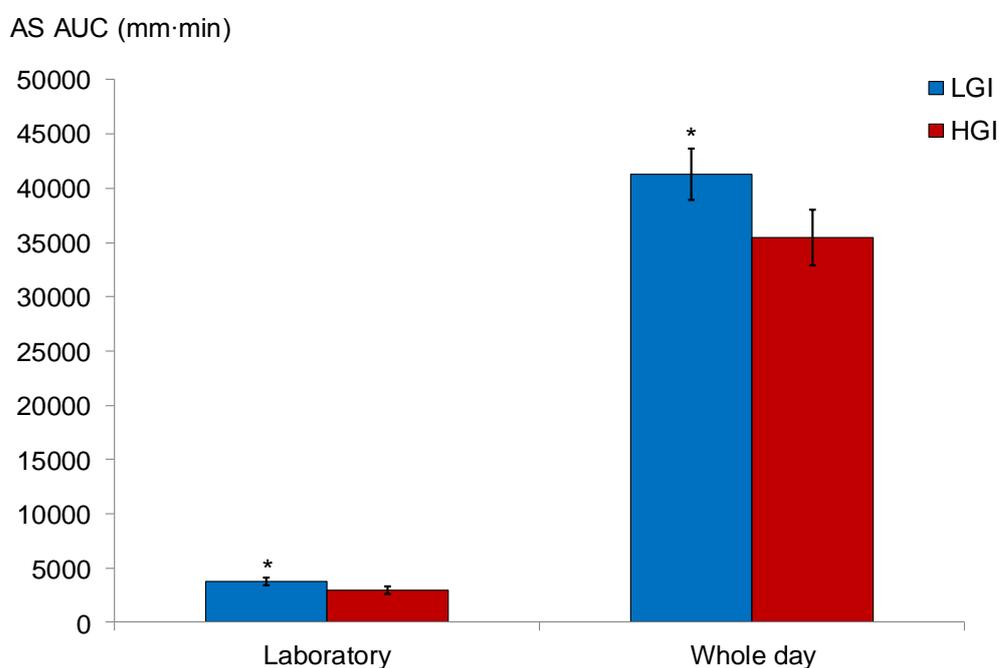


Figure 5.3 The areas under the curve of the appetite score between the low glycaemic index (LGI) and high glycaemic index (HGI) trials

Data presented as mean \pm SD ($n = 14$); AS AUC = area under the curve of the appetite score; * significantly higher to the corresponding HGI AS AUC ($p < 0.05$).

5.3.4 Energy intake, energy expenditure and energy balance

There was no significant difference between the LGI and HGI breakfast EI (572 ± 236 vs. 587 ± 282 kcal, $t(13) = -0.66$, $p = 0.5$). The LGI lunch EI was found to be significantly lower than the HGI lunch EI (751 ± 168 vs. 834 ± 217 kcal, $t(13) = -2.4$, $p < 0.033$) whereas the LGI snack EI was significantly higher than the HGI snack EI (476 ± 368 vs. 404 ± 322 kcal, $t(13) = 2.4$, $p = 0.035$) leading to no significant difference in the total EI ($2,652 \pm 513$ vs. $2,645 \pm 600$ kcal, $t(13) = 0.1$, $p = 0.9$) (Figure 5.4). The macronutrient and the dietary fibre intakes between trials were comparable (Table 5.4). Due to technical faults with the

accelerometers on the pre-trial, and the trial as well as the post-trial days only $n = 13$ and 12 respectively were available. There were no effects of trial, time or trial x time interaction for EI, EE, and energy balance (Table 5.5).

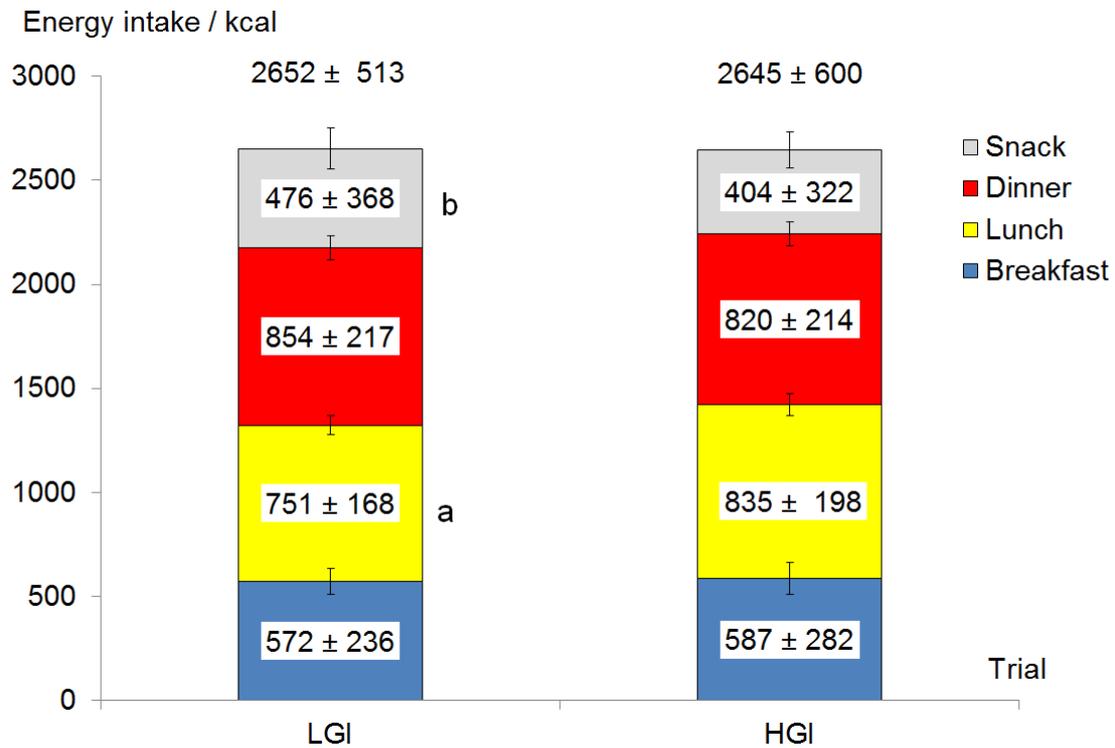


Figure 5.4 Energy intakes at different meals over the trial days

Data presented as mean \pm SD ($n = 14$). LGI = low glycaemic index; HGI = high glycaemic index; ^{a,b} significantly lower ($p = 0.033$) and higher ($p = 0.035$) to the corresponding HGI lunch and dinner respectively.

The rate of fat oxidation in the HGI post-trial morning tended to be higher than that in the HGI trial morning (0.103 ± 0.03 vs. 0.089 ± 0.03 $\text{g}\cdot\text{min}^{-1}$, $t(13) = 1.98$, $p = 0.069$).

Table 5.4 Comparison of the energy and the macronutrient intakes between trials on the trial days

Intakes	Trial	
	LGI	HGI
Carbohydrate (g)	444.7 ± 87.8	445.9 ± 102.7
Protein (g)	102.5 ± 21.7	101.6 ± 21.3
Fat (g)	63.9 ± 13.6	63 ± 16.6
Saturated	26.5 ± 5.6	26.1 ± 8.1
Mono-unsaturated	25.3 ± 6.3	23.6 ± 6.1
Poly-unsaturated	8.3 ± 1.7	9.1 ± 2.2
Fibre (g)	25.4 ± 3.8	24.3 ± 4.4
Energy intake (kcal)	2,652 ± 513	2,645 ± 600

Values are mean ± SD (n = 14). LGI = low glycaemic index; HGI = high glycaemic index.

Table 5.5 Comparison of the energy intakes, energy expenditure and energy balance in kcal between trials

Day	Energy profile	Trial	
		LGI	HGI
Pre-trial	Intake	2,681 ± 659	2,719 ± 699
	Expenditure*	2,644 ± 564	2,516 ± 577
	Balance*	-51 ± 840	205 ± 820
Trial	Intake	2,652 ± 513	2,645 ± 600
	Expenditure**	2,519 ± 554	2,631 ± 799
	Balance**	23 ± 593	-104 ± 717
Post trial	Intake	2,417 ± 487	2,527 ± 486
	Expenditure**	2,594 ± 493	2,512 ± 639
	Balance**	-174 ± 619	-39 ± 520

Values are mean ± SD (n = 14, except *n = 13 and **n = 12); LGI = low glycaemic index; HGI = high glycaemic index; EE = energy expenditure; EI = energy intake.

5.3.5 Palatability

No significant differences were found between trials in the PS following breakfast ($p = 0.78$) or lunch ($p = 0.68$). However, participants reported that the HGI dinner was significantly more tasty (74.7 ± 17.0 vs. 58.6 ± 23.5 , $t(13) = 3.7$, $p = 0.003$) and more palatable (72.0 ± 18.6 vs. 56.4 ± 22.9 , $t(13) = 3.1$, $p = 0.009$) leading to greater HGI dinner PS (69.0 ± 20.7 vs. 56.1 ± 23.1 , $t(13) = 2.6$, $p = 0.024$) than the LGI dinner PS (Appendix P). Significant negative correlations were found between the post dinner PS and AS at both LGI and HGI dinners which explained 56% ($p = 0.002$) and 48% of the variance ($p = 0.007$) respectively.

5.3.6 Relationships amongst appetite sensation and energy intake

In order to remove any confounding effect due to the PS difference, the correlations between AS and the EI at each meal were assessed after an adjustment for PS. When both trials were included, significant negative correlations were found between breakfast EI ($p = 0.002$), lunch EI ($p = 0.008$) and their corresponding postprandial AS; and between the total EI without snack EI ($p = 0.025$) and the overall postprandial AS which explained 32.5%, 24.6% and 13.4% of variances respectively. Only the LGI breakfast EI was found to be negatively correlated to the post breakfast AS which explained 40.9% of variance. The breakfast EI ($p = 0.035$) and the total EI without snack EI ($p = 0.046$) at the HGI trial were negatively correlated to the corresponding post meal AS which explained 34.2% and 23.8% of variance respectively (Table 5.6). The negative correlations between EI and AS meant the higher the EI at one meal, the lower the rating of appetite sensation following that meal.

Table 5.6 Correlations between energy intakes of breakfast, lunch and dinner; and total energy intake without snack and the corresponding postprandial appetite scores

Energy intake	Grouped	LGI	HGI
Breakfast	$r = -0.57$ $p = 0.002$	$r = -0.64$ $p = 0.01$	$r = -0.58$ $p = 0.035$
Lunch	$r = -0.46$ $p = 0.011$	$r = -0.44$ $p = \text{n.s.}$	$r = -0.59$ $p = 0.012$
Dinner	$r = -0.13$ $p = \text{n.s.}$	$r = -0.29$ $p = \text{n.s.}$	$r = -0.06$ $p = \text{n.s.}$
Total without snack	$r = -0.39$ $p = 0.025$	$r = -0.3$ $p = \text{n.s.}$	$r = -0.49$ $p = 0.046$

LGI = low glycaemic index; HGI = high glycaemic index; n.s. = non significance; n = 14, except *n = 28.

5.4 Discussion

The results supported the hypothesis that HGI meals suppressed appetite to a greater extent than LGI meals in recreationally active males, both the period in the laboratory and throughout the trial days. However, no significant difference was found in the EI between trial days.

5.4.1 Appetite and energy intake

The current findings were in line with study one and Anderson et al. (2002) that recruited males with normal BMI. The authors found that HGI CHO resulted in greater subjective appetite suppression than LGI CHO and the effect could last for an hour. Thus, the discrepancy of the current findings to the previous preferences on LGI foods on appetite suppression might result from the differences in body composition. In addition, the current findings showed that

when consumed frequently the appetite suppressing effects of HGI foods appeared to last for a whole day, longer than in study one and in Anderson et al. (2002).

Due to the lower AS AUC in the HGI trial, it was surprising that the LGI lunch EI was significantly lower than the HGI lunch EI without any significant difference in the post lunch AS between trials. The current study provided energy fixed meals conditions, rather than an *ad libitum* environment, although participants were asked to return any leftover foods if they felt full. Participants scored the appetite ratings after consuming those test foods. In accordance with the time sequence the EI should be considered as a determinant of the post meal AS at each meal time. A negative correlation was evident between the HGI post lunch AS and the HGI lunch EI ($p = 0.012$) whilst the LGI post lunch AS was not significantly correlated to the LGI lunch EI ($p = 0.13$). Although the reason for the absence of such an association after the LGI lunch remained unclear, the increased LGI snack EI appeared to compensate for the reduced LGI lunch EI leading to lack of difference in the total EI between trials.

In their review, McKiernan, Houchins, and Mattes (2008) reported that only one third of the papers identified showed an association between appetite related questions and food intake. The authors suggested that hunger was only a weak predictor of EI ($r = 0.3$) and the association between hunger and EI was even weaker under free-living conditions. There is evidently a complex relationship between appetite sensations and dietary intake. In the current study using the AS composited from five appetite related elements, the postprandial AS AUC

was consistently lower following the HGI foods than the LGI foods in both laboratory and free-living conditions. Since a counterbalanced design was employed, these significant differences between trials could not be attributed to any effect of trial order.

5.4.2 Subsequent energy intake

Stevenson, Williams, Nute, Swaile, and Tsui (2005) found that a LGI evening meal elicited a higher gut fullness score and lower rating of hunger in the following morning than following an HGI evening meal. Consumption of LGI foods has been proposed to promote weight loss due to elevation of the metabolic rate, as well as, the fat oxidation when compared to HGI foods (McMillan-Price & Brand-Miller, 2006). Raben (2002) reviewed the GI effect on EE. Energy expenditure was increased in nine and seven out of the 15 studies had reduction of *ad libitum* food intake after the consumption of LGI foods. Krog-Mikkelsen et al. (2011) speculated that any GI effect on energy balance, if it existed, would result from the change in appetite and EI; but not from EE.

Previous studies investigated the effect of evening meals with different GI values on substrate oxidation and glucose tolerance in the morning after the consumption of the standardized HGI meals (Stevenson et al., 2008; Stevenson, Williams, Nute, et al., 2005; Wolever, Jenkins, Ocana, et al., 1988). The insignificant difference in the RER in the current study was consistent with the baseline fasting results of these previous studies. Lower concentration of free fatty acid [FFA] was reported following HGI diets throughout the day compared to LGI diets (Brand-Miller et al., 2002). Nilsson, Ostman, Granfeldt, et al. (2008)

found that the fasting glucose level following a LGI evening meal was lower than that following a HGI evening meal. Low glucose level was found to be negatively associated with [FFA] (Liljeberg & Bjorck, 2000; Nilsson, Ostman, Preston, et al., 2008). The [FFA] was suggested to induce peripheral and hepatic insulin resistance. The [FFA] was not measured here but there was a trend for elevation of the fat oxidation in the post-trial morning after the consumption of HGI foods on the trial day ($p = 0.069$). Elevation of fat oxidation appears to be desirable for promoting weight loss. Nevertheless, fat oxidation can be elevated by low levels of circulating glucose. Thus, the higher rate of fat oxidation following the HGI evening meals in longer term (> 10 hours) may be due to a greater drop in postprandial glucose, even within normal range in the current study. If such speculation was correct, it might suggest that HGI meals would lead to higher hunger rating in the subsequent morning despite greater suppression of appetite sensation by the HGI foods on the trial day, which perhaps warrants further investigation.

5.4.3 Strengths and limitations

Only one participant's reported food intake was below the cut-off point in the current study. Nevertheless, this participant returned some leftovers. de Castro (2006) suggested that self-report dietary record is a reliable and valid method as all analyses were performed within subjects. The energy content of test meals were based on the individuals' reported food intakes prior to the first trial. Participants were not required to record food intake on the trial day unless extra foods were taken. This study design therefore could minimise under reporting of food intake. Furthermore, participants acted as their own controls in this crossover repeated-measure design study. Therefore, any underestimation of

the EI and food provided would affect both trials equally and not confound the comparison between trials. Blundell et al. (2010) has suggested that it is essential to advance laboratory and field research together when measuring food intake so as to reduce the inherent limitations in the two approaches and help bridges the gap between them.

Another strength of this study was that other than the manipulated characteristic of GI, the three main test meals as well as the snack were matched for energy, CHO, protein, fats including saturated, monounsaturated and polyunsaturated fats; fibre content and energy density between trials. It should be noted that the actual GI values may be different from the published values if the manufacturing processing conditions or formulas of commercial products were changed over time while the published values have not been kept up-to-date (Ludwig & Roberts, 2006). It was sought to minimize any potential discrepancy by determining GI values in a more consistent way via the selection of natural or minimally processed foods.

Besides the current study has the common limitations as mentioned in study one, unexpectedly the HGI dinner was reported to be more palatable than the LGI dinner which led the palatability to become a confounding variable in the current study. Breakfast, lunch and snack in both trials were ready to be consumed; whereas participants were required to cook the dinner. Although written cooking instruction was given, it was still difficult to control the palatability under home cooking conditions. Despite higher palatability related scores following the HGI dinner, participants did not significantly increase their

EI. Accordingly, the post dinner PS of both trials was negatively correlated with the corresponding post dinner AS which implies that the more palatable foods suppressed the appetite to a greater extent in this fixed meal condition. Warwick et al. (1993) previously stated that highly palatable meals rich in CHO might facilitate the subsequent control of EI. Holt, Miller, Petocz, and Farmakalidis (1995) found that palatability ratings were negatively correlated with a satiety index which explained 41% of variance implicating the highly palatable foods unfavoured to induce satiety. However, De Graaf, De Jong, and Lambers (1999) suggested that the effect of the pleasantness of the foods on hunger rating and food intake lasted for two hours postprandially. Potato was reported to have the highest satiety score among 38 common foods including some LGI high dietary fibre foods (Holt et al., 1995). In addition, its satiating effect lasted at least to two hours. No evening snack after dinner was provided in the current study and thus any effect of the palatability on subsequent EI is not known.

There are different interpretations of the term palatability and to date there is no clear and consistent definition. The influence of palatability on appetite sensation needs more clarification although it would be difficult to achieve in practice (Blundell et al., 2010). Moreover, the mean difference in the GI values between the LGI and HGI dinners was smallest among the main meals. Further investigation is needed to establish whether the lack of a significant correlation between the dinner EI and the postprandial AS is due to the confounding effect from PS or due to insufficient difference in the GI values between trials for this meal.

5.5 Conclusion

In summary, the consumption of the HGI CHO rich foods facilitates stronger appetite suppression in the early postprandial phase and throughout a day compared to the LGI CHO rich foods. These apparent satiating effects of HGI foods are applicable to recreationally active males when HGI foods are consumed frequently over one day. Nevertheless, the findings should not be generalized without further empirical evidence. The lack of difference in total EI between trials despite the lower appetite scores in the HGI trial is most likely due to the non *ad libitum* experimental setting. Further investigation of the relationships between GI and EI and balance by examining the physiological aspects of GI on substrate oxidation; and whether the advantage of HGI foods on appetite suppression persists in an *ad libitum* environment is necessary to understand the impact of HGI CHO rich foods on appetite and weight management in active individuals in the longer term.

CHAPTER 6 STUDY THREE: ACUTE EFFECT OF LOW AND HIGH GLYCAEMIC INDEX BREAKFASTS ON APPETITE, MOOD AND COGNITIVE PERFORMANCE OF RECREATIONALLY ACTIVE MALES

6.1 Introduction

Glucose is the main energy source for the brain with positive associations amongst blood [Glucose] and feelings of hunger, as well as mood and cognition. An impairment of cognitive performance has been demonstrated in healthy participants during hypoglycaemia (McCrimmon et al., 1996). Participants having lower blood [Glucose] were reported to have a greater level of tension during short term tasks compared to those having higher blood [Glucose] (Benton & Owens, 1993). Lower blood [Glucose] negatively altered the mood in terms of increasing fatigue and decreasing vigour (Degoutte et al., 2006); and feelings of hunger (Mayer, 1955), whilst negative mood increased feelings of hunger and the urge to eat (Hepworth, Mogg, Brignell, & Bradley, 2010). Elevated blood [Glucose] within the normal healthy ranges therefore appeared to be beneficial for cognitive function, particularly during stressful conditions (Fairclough & Houston, 2004; M. A. Smith & Foster, 2008).

Positive effects of the LGI CHO intake on postprandial cognitive functioning has been demonstrated in people with diabetes (Papanikolaou et al., 2006), children (Ingwersen et al., 2007; Micha et al., 2010) and middle-aged adults (Nilsson et al., 2009) when compared to HGI CHO foods. A consensus of the published literature appears to support LGI CHO food as being more beneficial to reduce the decline in performance on cognition such as attention and memory, whilst

hyperglycaemia following HGI food was associated with a negative impact on cognitive function. The rapid and drastic decline in blood [Glucose] following high GI (HGI) foods may impair cognitive performance similarly to the hypoglycaemic condition (Fairclough & Houston, 2004). However, no within-subject study has investigated the effect of CHO with different glycaemic responses on cognitive performance in recreationally active adults.

Wolever, Jenkins, Ocana, et al. (1988) found that a LGI dinner lowered the postprandial glycaemic response following a breakfast the next day than after a HGI dinner. The effect of a second meal on the postprandial glycaemic response was not investigated in the previous studies. The second meal, if presented, might confound the cognitive performances and affect the subsequent lunch intake following LGI and LGI breakfasts.

Certain sports such as car racing and rifle shooting require the players to perform fast decision making, maintain sustained attention and optimal selective attentional performance under demanding tasks, whilst the physical motion is not high during these sports. The purpose of this study was therefore to compare the acute effects of consuming either a LGI or a HGI breakfast on mood and cognitive performance; and subsequent EI in recreationally active males. In an extension of the findings in study one and study two, the subsequent EI following LGI and HGI breakfasts were examined under free living conditions. The objectives were to determine and compare the difference between trials in:

- the post breakfast appetite sensation and glucose responses,
- the cognitive performances and mood states; and
- the voluntary lunch EI three hours following test breakfasts.

It was hypothesized that a LGI breakfast would induce a lower postprandial glucose response, stabilise mood, improve cognitive performance, and reduce subsequent EI three hours post breakfast in recreationally active males compared to a HGI breakfast. It was also hypothesized that the HGI pre-lunch [Glucose] would be lower than the LGI trial. The critical postprandial period most beneficial to perform cognitive tasks after breakfast was also examined.

6.2 Methodology

6.2.1 Participants

Participants were briefed that the study was to investigate general aspects of meals on appetite sensation and cognitive performances; but not the specific purpose of investigating the effects of GI on appetite and cognition. They were screened by the 26-item EAT-26 (Garner et al., 1982) (Appendix C) for eligibility. Additional inclusion and exclusion criteria were males and colour blind, respectively. In order to encourage participants to perform the tasks, participants were informed that they would receive incentives based on the number of correct responses of the cognitive tasks at the end of the second trials. The incentive was at the rate of 2 p per correct response (\approx £10); however participants did not know the total number of the correct response and the incentive rate.

6.2.2 Study design

This study examined the effects of LGI and HGI breakfasts on post breakfast glucose responses, subjective appetite sensation, cognitive performances and lunch intake. Participants received either a LGI or HGI breakfast, and then the alternative breakfast after a washout period of not less than two days (Brouns et al., 2005; Warwick et al., 1993). The capillary [Glucose] and cognitive performances were assessed at specific time points. A standardised HGI lunch was provided three hours following breakfast.

6.2.3 Pre-trial arrangement

Participants completed a food acceptability questionnaire (Appendix I) before the familiarization trial to ensure all foods being provided were acceptable. Participants performed the computerized cognitive tasks at the familiarisation visit, arranged within a week prior to the first main trial, until they felt confident on performing the tasks to negate any potential learning effects.

Participants recorded two-day pre-trial food intake. They were required to keep similar food choices, portions and meal times and physical activity patterns two days before every trial (Brouns et al., 2005). Alcohol and unusual strenuous exercise were not allowed 24 hours prior to each trial day in order to control baseline conditions (Brouns et al., 2005). A standardised LGI pre-trial evening meal was provided in order to minimize the evening meal effect on appetite and the subsequent glycaemic responses (Brouns et al., 2005; Gilsenan et al., 2009; Lee et al., 2006; Mettler et al., 2007; Wolever, Jenkins, Ocana, et al., 1988). No other foods were allowed after the evening meal. No water was allowed 10

hours before the arrival of the laboratory. Participants were also asked to sleep for at least eight hours if possible (Fischer et al., 2001).

6.2.4 Trial procedures

Participants arrived at the laboratory between 0700 and 0900 hours after an overnight fasting for 10 hours (Brouns et al., 2005). The anthropometric measurement was taken by the standard procedures described in section 3.8. A glass of water (250 mL) was then served. While waiting 15 minutes for the breakfast (Kissileff et al., 1984), participants reported current mood state and the subjective appetite sensation using the Profile of Mood States (POMS) (Shacham, 1983) (Appendix Q) and VAS questionnaires respectively.

Fasting capillary blood samples were taken and analysed for [Glucose] and [Lactate]. Participants then consumed breakfast within 20 minutes (Brouns et al., 2005). A glass of water (250 mL) was consumed *ad libitum*. Participants consumed the breakfasts at the same place so that they could focus on the eating episodes and reduce the disruption from habitual satiation processes (Hetherington et al., 2006). Participants were requested to finish the breakfast unless they felt too full. Any leftover was weighed and recorded. The energy density and the macronutrient contents of the second test breakfast were then adjusted to match the first trial.

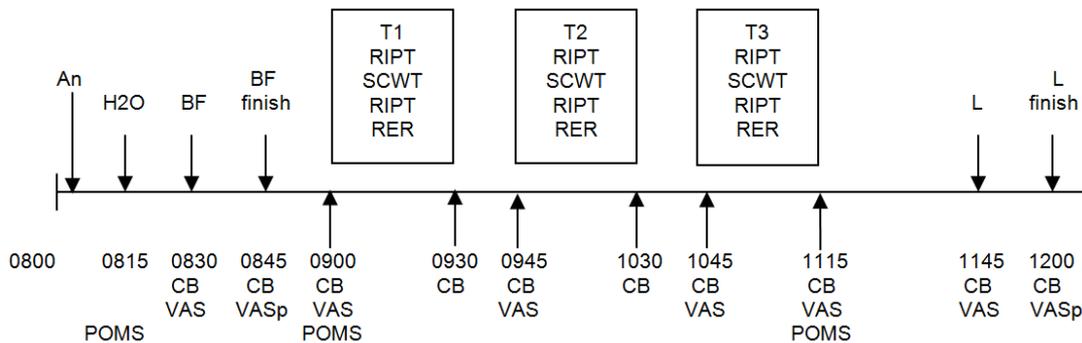
Capillary blood samples were taken and the VAS for appetite, physical comfort and palatability were completed immediately after breakfast and 15 minutes

postprandially (15-min pp). Participants started the first battery of the cognitive tasks after the assessment of the mood states at 15-min pp. A HR monitor and a gas collection mask were worn to assess the stress level and the rates of substrate oxidation of participants during the cognitive tasks, respectively. Capillary blood was collected for [Glucose] and the VAS was rated immediately after tasks. Participants completed three sessions of cognitive tasks with a 15-min break in between. Capillary blood was collected again prior to the next cognitive task. Mood states were assessed after the last task.

Participants then remained seated for 30 minutes. Capillary blood sample was collected and the appetite VAS was completed prior to the HGI lunch. They were asked to eat until they felt comfortably full within 15–20 minutes. They were free to ask for topping up after the initial portion, mentioned later, was all consumed. Capillary blood samples and the post meal VAS questionnaire were collected immediately after lunch.

Water was available at a volume of $2 \text{ mL} \cdot \text{kg}^{-1} \text{ BM}$ during the break. Participants were allowed to consume water *ad libitum* throughout the first trial. Same volume of water was provided for the second trial. Participants were encouraged to consume all water provided in the second trial to standardize the stomach occupancy. The procedures of the two trials were identical, except no skinfold measurement was taken at the second trial. In lieu, participants sat for 15 minutes at the second trial before the fasting capillary blood was taken (Figure 6.1). At the end of the second trial, participants were given the result of

the number of correct responses for all cognitive tasks so as to receive the incentives.



Keys:

An	Anthropometric measurement	VASp	Post meal visual analogue scales
BF	Breakfast	POMS	Profile of mood states
CB	Capillary blood collection	T	Cognitive task
H ₂ O	Water	RER	Expired gas collection
L	Lunch	RIPT	Rapid information process task
VAS	Visual analogue scales	SCWT	Stroop colour word task

Figure 6.1 Schema of the trial procedure

6.2.5 Test meals

Only the food and drinks accepted by participants were provided in the study according to the food acceptability questionnaire.

Evening meal. The pre-trial evening meal provided 1 g CHO kg⁻¹ BM. The energy contributions from CHO, protein and fat were around 55.5%, 14.5% and 30% respectively (Table 6.1). The GI value of the meal was around 51 (Henry

et al., 2005). The protein and fat contents met the average EI of the U.K. National Household Food Survey (Henderson, Gregor, Irving, & Swan, 2003).

Table 6.1 An example of the energy and nutrient compositions of the standardized low glycaemic index evening meal for a 75 kg participant

Evening meal	GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Tea finger ¹	40	10	7	0.8	1.4	44	0.3	3.7
Spaghetti bolognese ²	52	500	68	22	17.5	535	8.5	47.1
		510	75	23	19	579	9.1	51

CHO, carbohydrate; GI, glycaemic index; NSP, non-starch polysaccharide. ¹ Tesco Rich Tea Finger Biscuits; and ² Tesco Everyday Value Spaghetti Bolognese

Table 6.2 An example of the energy and nutrient contents of the low glycaemic index breakfast for a 75 kg participant

Food Name	GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Semi-skimmed milk ¹	32	346.3	16.3	12.1	5.9	158.8	0	6.9
Muesli ²	55	51.3	34.4	5.4	3.0	186.3	3.6	25.2
Apricots, dried ³	32	18.8	8.0	0.9	0.1	35.0	1.4	3.4
Fructose ⁴	19	11.3	10.8	0	0	38.8	0	2.7
Raisin ⁵	54	7.5	5.5	0.1	0	21.3	0.1	3.9
Macadamia	0	3.8	0.1	0.3	2.9	27.5	0.3	0
		438	75	19	13	468	5.4	42

CHO, carbohydrate; GI, glycaemic index; NSP, non-starch polysaccharide. ¹ Tesco organic 1.7% semi-skimmed pasteurised milk; ² Alpen Swiss muesli; ³ Tesco Ready to Eat Dried Apricots; ⁴ Fruisana fruit sugar; and ⁵ Sun-Maid Natural California Raisins

Test breakfasts. Both breakfasts consisted of 1 g CHO kg⁻¹ BM. The energy contribution from CHO, protein and fat was 60%, 15% and 25%, respectively. The calculated GI values of the LGI and HGI breakfasts were around 42 and 72.5 respectively with a difference of 30.5. The protein, fat and fibre contents and the energy density were matched between trials (Table 6.2 and 6.3).

Table 6.3 An example of the energy and nutrient contents of the high glycaemic index breakfast for a 75 kg participant

Food Name	GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Rice milk ¹	79	145	13.8	0.1	1.5	68.8	0.1	14.4
Corn Flakes ²	93	17.5	15.9	1.4	0.3	67.5	0.1	19.7
Fruit & Fibre ³	67	58.8	42.5	5.3	2.8	207.5	4	38.0
Light cheese	0	43.8	0.1	11.1	5.1	92.5	0	0
Cherry tomato	0	86.3	2.6	0.6	0.4	16.3	0.9	0
Macadamia	0	2.5	0.1	0.3	2	18.8	0.1	0
Water		85	0	0	0	0	0	0
		438	75	19	13	469	5.3	72

CHO, carbohydrate; GI, glycaemic index; NSP, non-starch polysaccharide. ¹ Rice Dream Organic Original Rice Milk; ² Kellogg's cornflakes; and ³ Tesco Fruit & Fibre

Ad libitum lunch. The subsequent lunch had a GI value of 90. The energy contributions from CHO, protein and fat were 60%, 15% and 25% respectively. The initial portion was set as 3 g CHO kg⁻¹ BM (Table 6.4). Any leftover was measured. The lunch EI was calculated from the difference in weight between prior to and after consumption based on the assumption of a homogenous distribution of energy and macronutrients.

Table 6.4 An example of the energy and nutrient contents of the high glycaemic index lunch for a 75 kg participant

Lunch	GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Potato mash ¹	95	240	211.3	25.9	2.9	972.5	16.8	89.2
Butter	0	13.8	0.1	0.1	10.5	95	0	0
Cheese	0	65	0.1	16.6	22.8	271.3	0	0
Gravy	0	22.5	13.6	0.5	3.6	90	0.3	0
Water	0	960	0	0	0	0	0	0
		1,301	225	43	40	1,429	17.1	89

GI, glycaemic index; CHO, carbohydrate; NSP, non-starch polysaccharide. ¹ Sainsbury's Basics Instant Mashed Potato

6.2.6 Cognitive tasks

Three batteries of computerised cognitive function tasks were administered using the E-prime psychological software. Each round of the tasks lasted for 25 minutes approximately included the RIPT (Owens et al., 1997) alternated with the SCWT (MacLeod, 1991, 1998; MacLeod & MacDonald, 2000). No feedback or results were provided to participants until the end of the second trial.

6.2.6.1 Rapid information processing task (RIPT)

Nine hundred digits, from one to eight, each at the rate of 100 digits per minute was popped out in a pseudo-random order on the monitor. Participants were required to press the spare bar as quickly and accurate as possible when a sequence of three consecutive odd or even digits as the required response was detected. A gap from 5 to 30 digits was separated between each required response. The number and the percentage of the correct and the wrong responses; and the correct RT were recorded and then analysed. An adjusted

number of the correct response was calculated by the difference between the number of correct response and the number of wrong response. Following the onset of the third digit of a target sequence 1,500 ms was allowed as a buffer time for participants to make a response. Another batch of the RIP task was presented following the SCWT that the sequence differed to the first batch.

6.2.6.2 Stroop colour word task (SCWT)

The first level was for practice which had 72 stimuli and the second level included the conditions of neutral, congruent and incongruent each contained 24 stimuli. An inter-stimuli interval of 1000, 2,000 or 3,000 ms was set randomly with a white fixation cross in the centre of the screen in order to avoid settling habituation (Coco, Di Corrado, Calogero, Perciavalle, & Maci, 2009). The difference in RT on incongruent and congruent trials provides an interference score, related in part to the inhibition of an automatic tendency to respond the word that a delayed response or an increased RT reflected inhibition. The design and the calculation of the SCWT interference and facilitation scores were described in section 2.7.6 and 3.9.1.

6.2.7 Blood biochemistry

Participants were required to put their hands in a warm water bath to improve the blood circulation prior to blood sampling. Capillary blood samples were collected before and immediately after breakfasts (0-min pp), prior to (15-, 60- and 105-min pp) and after each cognitive session (45-, 90- and 135-min pp); before (165-min pp) and immediately after lunch (180-min pp). Whole blood [Glucose] and [Lactate] were determined by the automated glucose and lactate

analyser. The procedures of the capillary blood sampling were described in section 3.10.

6.2.8 Subjective sensations

The POMS was completed at fasting, 15- and 135-min pp. The design of the POMS was described in section 3.6.2.

The subjective appetite sensation (AS), physical comfort and palatability (PS) to the meals were assessed using the VAS questionnaire. The appetite and the physical comfort were assessed at fasting; 0-, 15-, 45-, 90-, 135-, 165- and 180-min pp. The sensations of the palatability were rated immediately after breakfast and lunch. The AS and the PS were calculated using the methods explained in the section 3.6.1. No significant difference was found in the PS between the two breakfasts in the previous studies.

6.2.9 Substrate oxidation

Expired gas was collected whilst participants were performing the cognitive tasks. The substrate oxidation was estimated by the indirect calorimetry. The calibration of the gas analyser and the measurement of the substrate oxidation from the expired gas were described in the section 3.5.3.

6.2.10 Statistical analysis

6.2.10.1 Sample size calculation

Sample size was calculated at an alpha level of 0.05 with a power of 80% using the equation from National Research Council Committee (2003) (Appendix N) using the paper by Micha et al. (2010). The sample size required was 16 to detect a significant shorter completion time between the HGL-LGI and HGL-HGI trials.

6.2.10.2 Data analyses

Descriptive statistics were calculated for group characteristics. The AS AUC was examined, expressed in mm·min. The glucose IAUC ignoring the negative values were calculated, expressed in mmol·L⁻¹·min. The blood [Glucose], the cognitive outcomes and the AS were analysed via two way ANOVA to detect the effects of trial, time and trial by time interactions on the glucose, lactate and substrate oxidation; and the cognitive outcomes. If any time x trial interaction was found, Tukey's *post-hoc* test was computed to locate the significant difference with the Bonferroni correction. Pearson's correlation analyses were applied to examine the associations among AS, breakfast EI, the capillary blood outcomes, the cognitive outcomes and palatability. The PS was inputted as a covariate to the post breakfast AS. Non-parametric Friedman ANOVA, Spearman's correlation and Wilcoxon signed-rank test were used when the parameters were not normally distributed.

The coefficient of variation of the reliability of the VAS was assessed using the PS obtained in the first and second trials. The intra-variation of VAS to detect the subjective sensation was calculated using the post lunch PS at the first and second visits. The coefficient of variation of the VAS was 16.8%. The effect of

trial order on AS, mood states, cognitive outcomes and lunch EI was also examined. Criterion for statistical significance was $p < 0.05$ at the two-tailed level.

6.3 Results

6.3.1 Characteristics of the participants

Sixteen male participants completed the trials successfully. Six participants were Caucasian and ten were Chinese. No significant differences were found in the anthropometric and physiological characteristics between the two ethnic groups (Table 6.5). All data were grouped for subsequent analyses.

Table 6.5 Anthropometric and physiological characteristics of the participants

Characteristics	Total (n = 16)	Caucasian (n = 6)	Chinese (n = 10)
Age (year)	24.4 ± 3.6	26.3 ± 3.6	23.2 ± 3.2
Body mass (kg)	73.4 ± 11.2	76.7 ± 9.3	71.4 ± 12.3
Body mass index (kg·m ⁻²)	22.9 ± 3.3	23.9 ± 2.7	22.2 ± 3.6
Body fat percentage (%)	23.2 ± 4.5	24.5 ± 5.6	22.4 ± 3.7
Fat free mass (kg)	56.0 ± 6.4	57.5 ± 3.4	55.1 ± 7.7

Values are means ± SD; n = number of participants.

6.3.2 Glycaemic index of meals

The GI value of the HGI breakfast was significantly higher than that of the LGI breakfast (72.4 ± 0.6 vs. 42.2 ± 0 ; $t(15) = 201$, $p < 0.001$). The difference of GI values between the HGI and LGI breakfast was 30.2 ± 0.6 . All test breakfasts were completely consumed.

6.3.3 Subjective sensations

There were no significant differences between the LGI and HGI pre-meal AS (71.9 ± 13.8 vs. 70.2 ± 16.7 , $t(15) = 0.63$, $p = 0.5$). Neither effects of trial ($F(1,15) = 4.3$, $p = 0.6$) nor trial x time interaction ($F(4.5, 67.9) = 1.3$, $p = 0.3$) in the AS were found during the whole trial period whilst a significant time effect existed ($F(3.3, 49.7) = 38.8$, $p < 0.001$) (Figure 6.2). The post breakfast AS decreased significantly compared to the pre-meal AS (31.1 ± 13.7 vs. 71 ± 15.1 , $t(31) = -13.8$, $p < 0.001$). No significant difference was found between the LGI and HGI AS AUC over 180 minutes before lunch ($9,121 \pm 2,297$ vs. $8,565 \pm 2,279$ mm·min, $t(15) = 1.4$, $p = 0.18$).

There were no significant differences between the LGI and HGI trials in the breakfast PS (61.4 ± 18.1 vs. 65.2 ± 21 , $p = 0.5$) and the lunch PS (49 ± 18.4 vs. 46.7 ± 16.2 , $p = 0.4$).

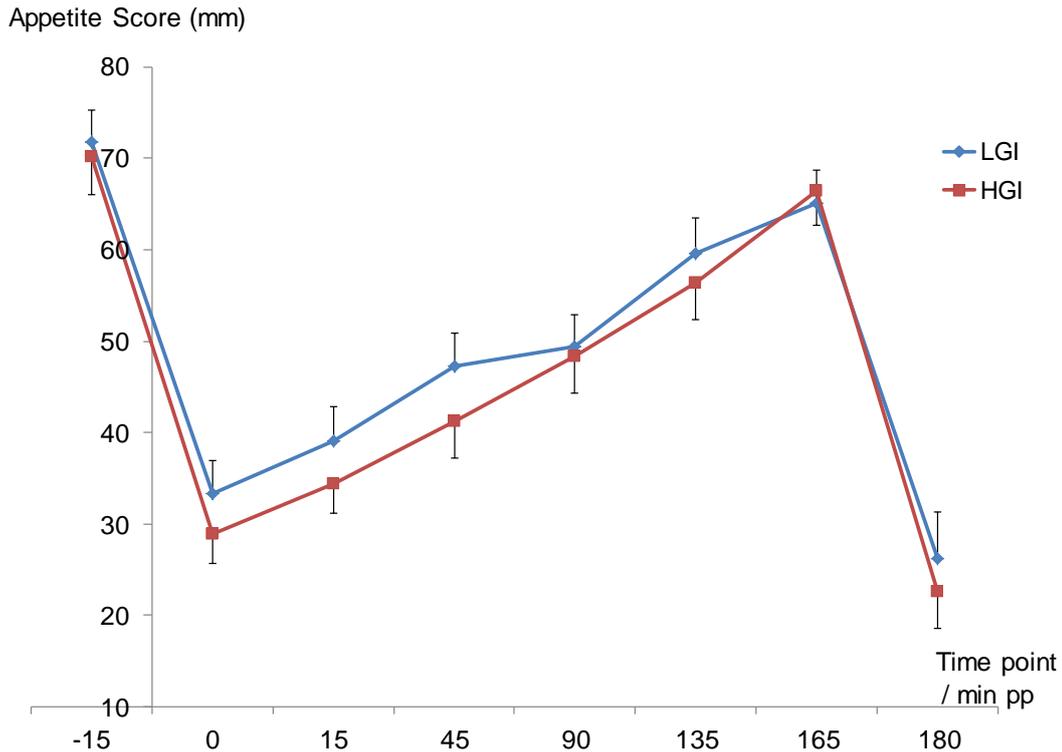


Figure 6.2 Effect of the low glycaemic index (LGI) and high glycaemic index (HGI) breakfasts on appetite score

Data presented as mean \pm SD (n = 16).

6.3.4 Glucose responses

All [Glucose] were normally distributed except at the LGI 60-min pp and at the HGI 45-min pp. There were significant effects of trial x time interaction ($F(3, 44.5) = 8.4, p < 0.001$) and time ($F(2.9, 42.8) = 68.3, p < 0.001$) during the whole trial period (Figure 6.3). The LGI and HGI fasting [Glucose] were comparable (4.4 ± 0.2 vs. 4.3 ± 0.1 mmol·L⁻¹, $t(15) = 1.2, p = 0.24$). The peak [Glucose] reached at 15-min pp was significantly higher in the HGI than the LGI trial (8.1 ± 1.3 vs. 6.9 ± 1.1 mmol·L⁻¹, $t(15) = -3.5$, corrected $p = 0.03$). No significant differences were found during 45- and 105-min pp between trials. The HGI [Glucose] after the last task at 135-min pp was significantly lower than in the LGI trial (4.0 ± 0.5 vs. 4.3 ± 0.3 mmol·L⁻¹, $t(15) = -3.4$, corrected $p = 0.04$).

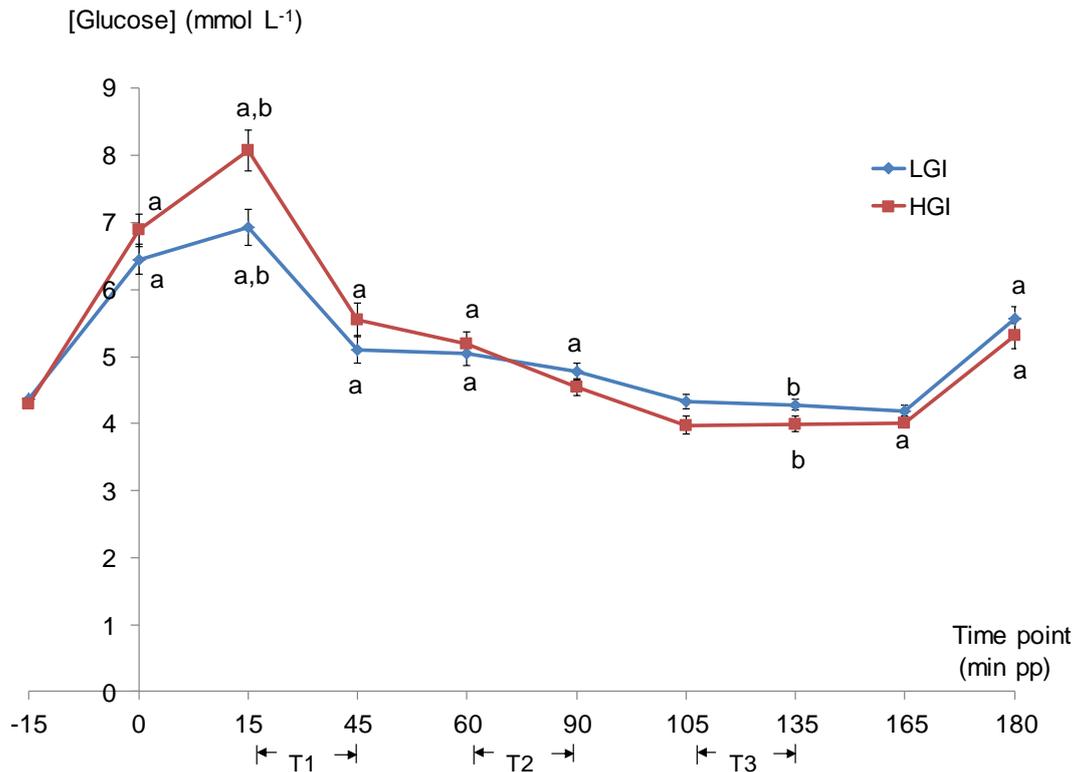


Figure 6.3 Effects of the low glycaemic index (LGI) and high glycaemic index (HGI) breakfasts on the glucose responses

Data presented as mean \pm SD ($n = 16$). [Glucose], glucose concentration; min pp, minutes postprandially; ^a significant different to the corresponding fasting level (corrected $p \leq 0.03$); ^b significant difference when sharing the same letter on the same time point (corrected $p \leq 0.04$).

The LGI and HGI [Glucose] returned to the corresponding fasting levels at 105-min pp (4.3 ± 0.4 vs. 4.4 ± 0.2 mmol·L⁻¹, $t(15) = -0.2$, $p = 0.8$) and at 90-min pp (4.5 ± 0.5 vs. 4.3 ± 0.1 mmol·L⁻¹, $t(15) = 1.9$, $p = 0.08$), respectively. The HGI [Glucose] at 165-min pp dropped significantly below the fasting level (4.0 ± 0.2 vs. 4.3 ± 0.1 mmol·L⁻¹, $t(15) = -5.7$, corrected $p < 0.01$). No difference was found in between the LGI [Glucose] at 165-min pp and the fasting level. The HGI [Glucose] at 165-min pp became comparable again with the LGI (4.0 ± 0.2 vs. 4.2 ± 0.3 mmol·L⁻¹, $t(15) = -2.1$, $p = 0.051$) (Appendix R).

There was a significant time effect ($F(1,15) = 63.2, p < 0.001$) such that the post lunch [Glucose] was significantly higher than the pre-lunch [Glucose] (5.4 ± 0.7 vs. $4.1 \pm 0.3 \text{ mmol}\cdot\text{L}^{-1}$, $t(31) = 9.9$, corrected $p < 0.01$). However, no difference was found between the LGI and HGI post lunch [Glucose] (5.6 ± 0.7 vs. $5.3 \pm 0.8 \text{ mmol}\cdot\text{L}^{-1}$, $t(15) = 1.3, p = 0.2$).

The LGI total glucose AUC during task 1, over 30 minutes, was significantly lower than in the HGI (181 ± 26 vs. $204 \pm 31 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{min}$, $t(15) = -2.8, p = 0.013$) while the LGI total glucose AUC during task 3 was significantly higher than in the HGI (129 ± 10 vs. $120 \pm 14 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{min}$, $t(15) = 3.3, p = 0.005$). The HGI glucose IAUC was significantly greater than in the LGI trial between the period from fasting to 180-min pp (189 ± 65 vs. $147 \pm 56 \text{ mmol}\cdot\text{L}^{-1}\cdot\text{min}$, $t(15) = 2.8, p = 0.012$).

6.3.5 Lactate responses

There were significant effects of trial ($F(1,15) = 9.8, p = 0.007$), time ($F(2.9, 43.6) = 98.6, p < 0.001$) and trial x time interaction ($F(2.7, 40.4) = 9.5, p < 0.001$) during the whole trial period (Figure 6.4).

The LGI and HGI fasting [Lactate] were comparable (0.59 ± 0.23 vs. $0.58 \pm 0.22 \text{ mmol}\cdot\text{L}^{-1}$, $t(15) = 0.2, p = 0.85$). The LGI and HGI [Lactate] reached peaks at 15-min pp and 45-min pp, respectively. The post-hoc paired t -test showed that the LGI 15-min pp [Lactate] was significant higher than the HGI trial (1.1 ± 0.27 vs. $0.8 \pm 0.22 \text{ mmol}\cdot\text{L}^{-1}$, $t(15) = 4.1$, corrected $p = 0.009$). The LGI and HGI

[Lactate] returned to the corresponding fasting levels from 105-min pp. The HGI post lunch [Lactate] was significantly below the pre-lunch level (0.43 ± 0.11 vs. 0.57 ± 0.19 mmol·L⁻¹, $t(15) = -4.9$, corrected $p < 0.01$) but the significance was not found in the LGI.

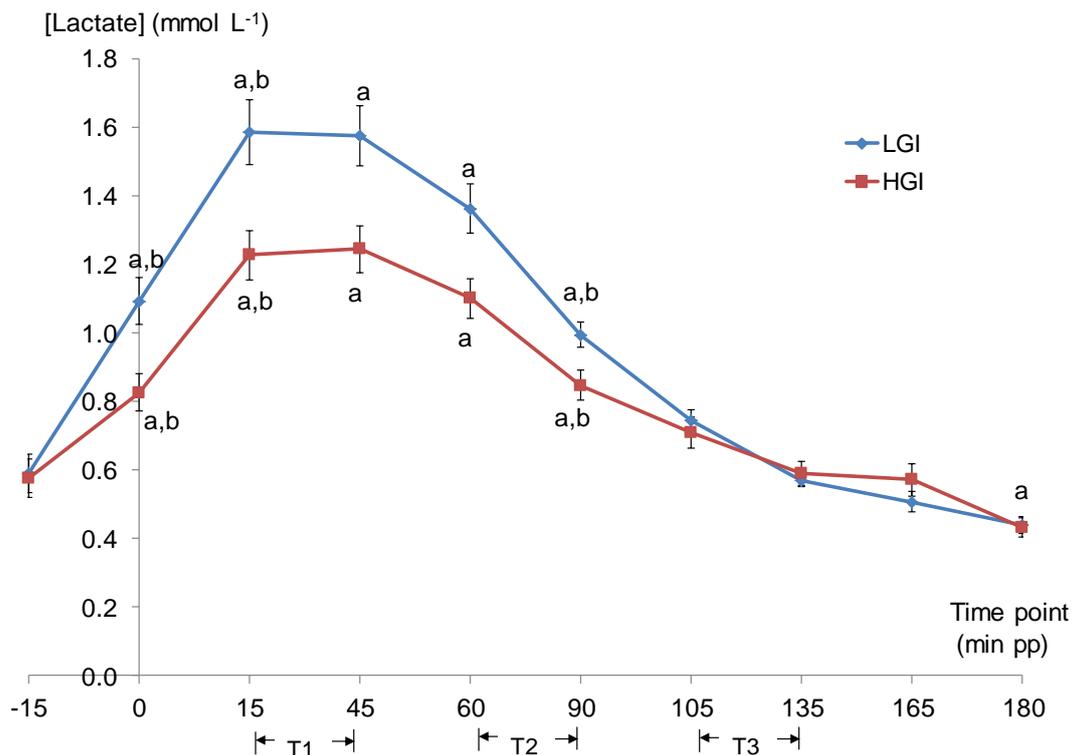


Figure 6.4 Effects of low glycaemic index (LGI) and the high glycaemic index (HGI) breakfasts on lactate responses

Data presented as mean \pm SD (n = 16). [Lactate], lactate concentration; min pp, minutes postprandially; ^a significant different to the corresponding fasting level (corrected $p < 0.05$); ^b significant difference when sharing the same letter on the same time point (corrected $p < 0.05$).

6.3.6 Energy intake and expenditure

No significant difference was found in the pre-trial evening energy and macronutrient intakes between trials. Significant effects of trial x time interaction ($F(1.5, 21.9) = 5.4$, $p = 0.019$) and time ($F(1.7, 25.3) = 22.8$, $p <$

0.001) were found in the CHO oxidation. No trial effect was found ($F(1, 15) = 4.1, p = 0.06$). The *post hoc t*-test showed that the CHO oxidation significantly reduced gradually in the LGI trial, but not in the HGI trial. The LGI task 1 CHO oxidation was significantly higher than the HGI (7.6 ± 1.7 vs. 6.2 ± 1.9 g, $t(15) = 3.1$, corrected $p = 0.003$) (Figure 6.5) (Appendix S).

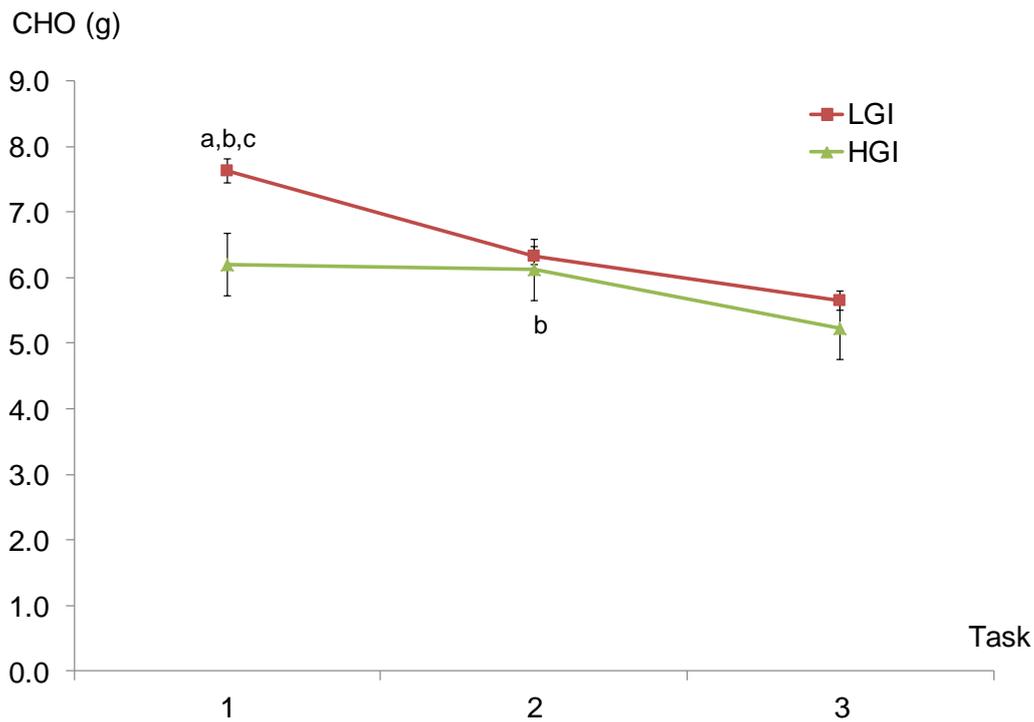


Figure 6.5 Carbohydrate (CHO) oxidation in the low glycaemic index (LGI) and high glycaemic index (HGI) trials

Data presented as mean \pm SD ($n = 16$). ^a significantly higher than the corresponding Task 2 (corrected $p < 0.001$); ^b significantly higher than the corresponding Task 3 (corrected $p < 0.004$); ^c significantly higher than the HGI (corrected $p = 0.003$).

No trial \times time interaction was found in the fat oxidation ($F(1.8, 26.5) = 1.4, p = 0.25$); whilst significant effects of trial ($F(1, 15) = 4.9, p = 0.04$) and time ($F(1.9, 29.2) = 12.5, p < 0.001$) were found (Figure 6.6). The amount of the fat oxidation at the LGI task 1 (0.6 ± 0.7 g) was significantly lower than that at the

LGI task 3 (1.2 ± 0.6 g, $t(15) = -4.8$, corrected $p < 0.01$); and was lower than the HGI task 1 (1.3 ± 0.7 g, $t(15)$ corrected $p = 0.05$). The HGI task 2 fat oxidation was lower than the corresponding task 3 (1.14 ± 0.76 vs. 1.34 ± 0.73 g, $t(15) = -2.8$, corrected $p = 0.04$) (Appendix S).

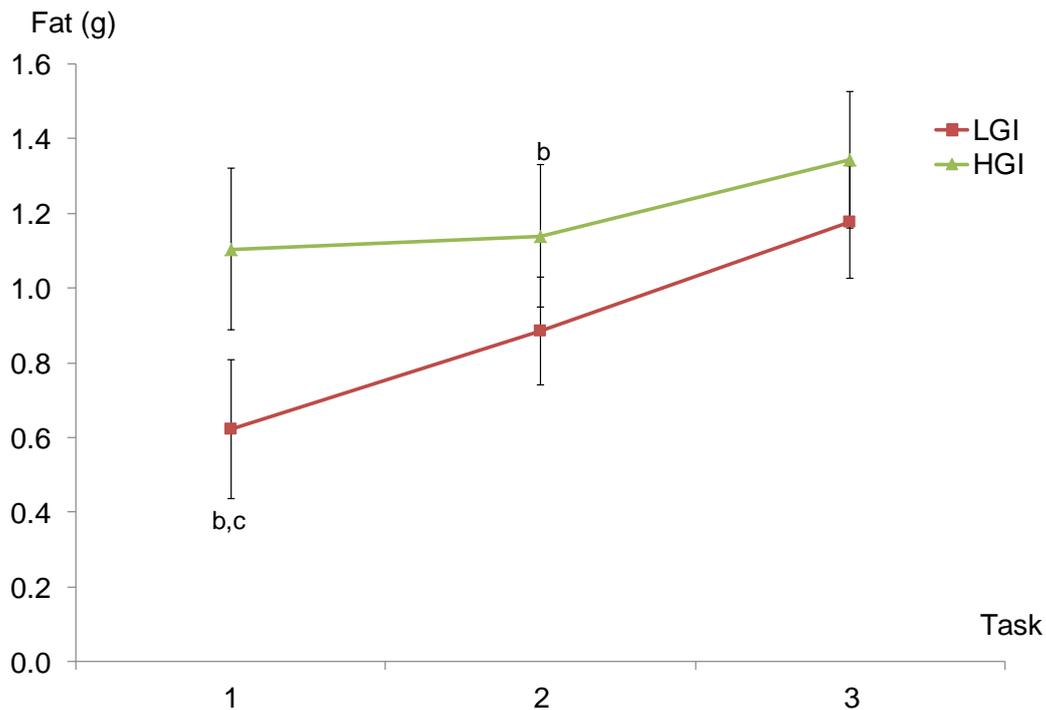


Figure 6.6 Fat oxidation in the low glycaemic index (LGI) and high glycaemic index (HGI) trials

Data presented as mean \pm SD ($n = 16$). ^b significantly lower than the corresponding Task 3 (corrected $p < 0.05$); ^c significantly lower than the HGI (corrected $p = 0.05$).

Significant effects of trial \times time interaction ($F(1.8, 26.7) = 4$, $p = 0.034$) and time ($F(1.6, 23.7) = 23.2$, $p < 0.001$) were found in the RER, but no trial effect was found ($F(1, 15) = 3.3$, $p = 0.09$) (Figure 6.7) (Appendix S).

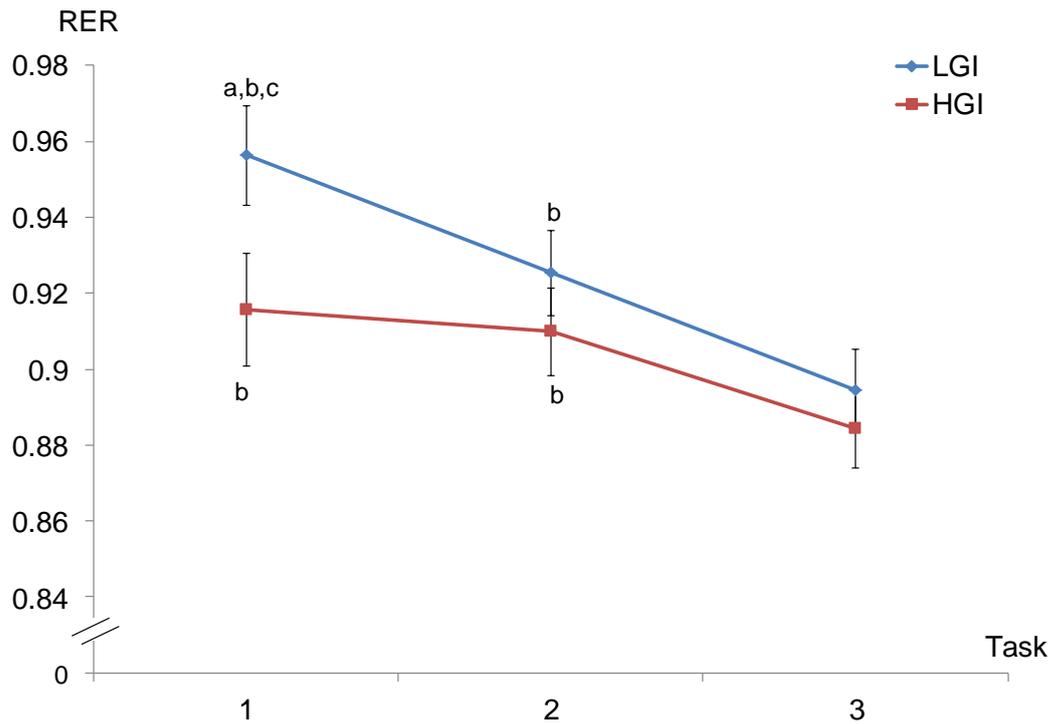


Figure 6.7 Respiratory exchange ratio (RER) in the low glycaemic index (LGI) and high glycaemic index (HGI) trials

Data presented as mean \pm SD (n = 16). ^a significantly higher than the corresponding Task 2 (corrected $p \leq 0.014$); ^b significantly higher than the corresponding Task 3 (corrected $p \leq 0.017$); ^c significantly higher than the HGI (corrected $p = 0.047$).

Table 6.6 The pre-trial intakes of the energy and the macronutrients between trials

Intakes	LGI	HGI
Dinner energy (kcal)	592 \pm 131	549 \pm 81
Carbohydrate (g)	90.9 \pm 24.7	83.5 \pm 17.6
Protein (g)	19.3 \pm 3.6	17.7 \pm 1.7
Fat (g)	18.9 \pm 3.6	17.8 \pm 3.1
Total energy (kcal)	2,278 \pm 534	2,141 \pm 437
Dinner energy (%)	26.3 \pm 8.2	26.7 \pm 7.8

Data presented mean \pm SD (n = 16). HGI, high glycaemic index; LGI, low glycaemic index.

No significant differences were found in the energy and macronutrients intakes at the pre-trial evening meal (Table 6.6). There were no significant differences between the LGI and HGI breakfast EI (462 ± 67 vs. 463 ± 67 kcal, $t(15) = -0.9$, $p = 0.4$) and the *ad libitum* lunch EI (710 ± 374 vs. 679 ± 337 kcal, $t(15) = 0.6$, $p = 0.5$).

6.3.7 Cognitive and psychological performances

6.3.7.1 Rapid information processing task

The two-way repeated-measure ANOVA did not find effects of trial, time and trial x time interaction in the number of correct responses. Pearson's correlation found that the HGI task 1 adjusted response accuracy was negatively associated with the pre-task [Glucose] ($r = -0.68$, $p = 0.003$); and with the total glucose AUC during task 1 ($r = -0.64$, $p = 0.008$) (Table 6.7). Significant negative correlations were found between the total glucose AUC during the three tasks and the total number of the adjusted correct response in the HGI trial ($r = -0.61$, $p = 0.012$). The paired *t*-test found a trend that the total number of the adjusted correct response in the LGI was higher than the HGI trials (237 ± 99 vs. 225 ± 96 , $t(15) = 1.9$, $p = 0.08$).

All RT were not normally distributed. Non-parametric tests were performed for analysing the RT. The Friedman's ANOVA did not find a significant difference in RT ($\chi^2(5, n = 16) = 3.8$, $p = 0.59$). Spearman's correlation found that the HGI task 1 RT was positively correlated with the corresponding high pre-task [Glucose] ($r = 0.58$, $p = 0.019$). Conversely the HGI task 3 RT was negatively correlated with the corresponding low pre-task 3 [Glucose] ($r = -0.61$, $p = 0.01$);

and with the total glucose AUC during task 3 ($r = -0.52, p = 0.04$). The LGI task 2 RT was positively correlated with its pre-task 2 [Glucose] ($r = 0.58, p = 0.019$); and with the total glucose AUC during task 2 ($r = 0.52, p = 0.04$) (Table 6.7).

Table 6.7 Correlations between the glucose concentration and the Rapid Information Processing Task performances

Task		Number of adjusted correct response		Reaction time (ms)	
		LGI	HGI	LGI*	HGI*
Pre-task glucose concentration (mmol·L ⁻¹)	1	$r = -0.04$	$r = -0.68^a$	$r = 0.2$	$r = 0.58^a$
	2	$r = -0.41^*$	$r = -0.45$	$r = 0.58^a$	$r = 0.33$
	3	$r = -0.39$	$r = 0.25$	$r = 0.23$	$r = -0.61^a$

$n = 16$. HGI, high glycaemic index; LGI, low glycaemic index; * Spearman's correlation; ^a significant correlation with the pre-task corresponding glucose concentration ($p < 0.05$)

6.3.7.2 Stroop colour word task

The repeated-measure ANOVA found a significant trial x time effect in the Stroop interference time ($F(1.6, 24.6) = 4.0, p = 0.039$). No effects of trial ($F(1, 15) = 0.1, p = 0.74$) and time ($F(1.8, 27.4) = 2, p = 0.16$) were found. The LGI task 3 interference time (110 ± 60 ms) was longer than that at the task 1 (66 ± 64 ms, $t(15) = 2.4$, corrected $p = 0.086$); as well as at the task 2 (63 ± 67 ms, $t(15) = 2.4$, corrected $p = 0.12$), but the differences did not reach significance after the Bonferroni correction (Figure 6.8). Pearson's correlation found that the LGI task 3 interference time had significant positive correlations with the pre-

task [Glucose] ($r = 0.59, p = 0.015$); and with the total glucose AUC during task 3 ($r = 0.51, p = 0.046$). After an adjustment of the [Lactate] as a covariate, the association with the pre-task [Glucose] ($r = 0.51, p = 0.09$) and with the total glucose AUC ($r = 0.31, p = 0.2$) weakened. A significant positive correlation was found between the LGI task 3 interference time and its CHO oxidation ($r = 0.54, p = 0.032$). The significant correlation remained after the adjustment of the pre-task [Lactate] ($r = 0.57, p = 0.01$). No significant correlations between the glucose response and the SCWT interference time in all HGI tasks were found.

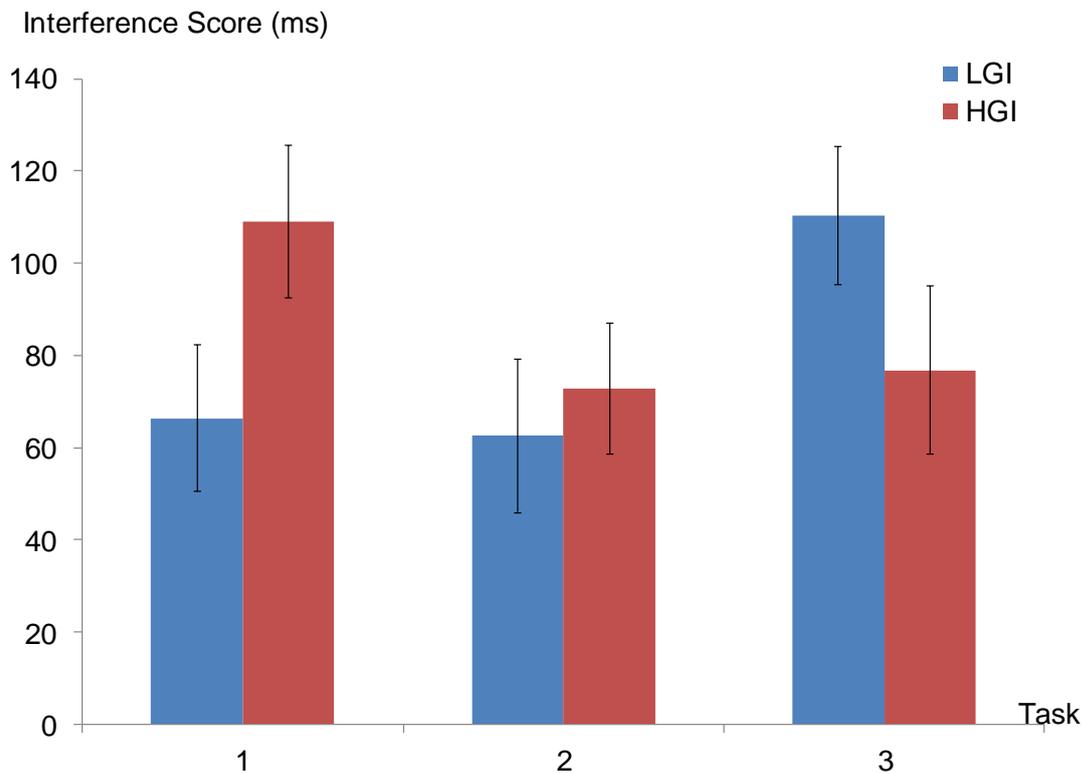


Figure 6.8 Stroop Colour Word Task Interference Score between trials. Data presented as mean \pm SD (n = 16)

LGI, low glycaemic index; HGI, high glycaemic index.

All interference scores for accuracy were normally distributed except at the LGI task 2 and task 3; and at the HGI task 3. No trial x time effect was found in the

Stroop interference for accuracy ($F(1.5, 22.7) = 0.66, p = 0.49$); however, there existed a significant trial effect ($F(1, 15) = 8.2, p = 0.012$).

6.3.7.3 Mood state

Eleven out of 36 mood data were normally distributed; the Friedman's ANOVA was used to detect the differences. The Friedman's ANOVA did not find any significant differences in all mood elements. Spearman's correlation found a significant positive correlation between the HGI 15-min pp [Glucose] and the corresponding depression score ($r = 0.72, p = 0.002$).

There was a significant trial order x time interaction in the confusion score ($F(1.6, 23.6) = 3.9, p = 0.044$). The *post hoc t*-test found that participants felt confused the most, prior to the breakfast in the first visit (2.7 ± 1.8) when compared to that in the second visit ($0.9 \pm 0.9, t(15) = 3.6, p = 0.003$), and prior to the start of the first cognitive tasks in the first visit at 15-min pp ($1.6 \pm 1.9, t(15) = 4.1, p = 0.001$).

6.3.8 Relationships among appetite, energy intake, glucose and lactate concentrations

When the data between fasting and the 165-min pp were included, significant negative correlations were found between the AS and the [Glucose] in the LGI ($r = -0.46, p < 0.001$) and HGI ($r = -0.51, p < 0.001$) trials which explained 20.7% and 26.4% of variance, respectively (Figure 6.9). The variances weakened when the post lunch data were included in the LGI ($r^2 = 0.15, n = 128, p < 0.001$) and HGI ($r^2 = 0.19, n = 128, p < 0.001$) trials. No significant

correlations were found between the AS AUC and the corresponding glucose IAUC in the LGI and HGI trials.

Pearson's correlation did not find significant correlations between post breakfast AS and the corresponding breakfast EI in the LGI and HGI trials. The LGI pre-lunch [Glucose] was negatively associated with the pre-lunch AS ($r = -0.51$, $p = 0.045$), but no significant correlation was found in the HGI trial. The pre-lunch AS were significant predictors of the lunch EI kg^{-1} FFM in the LGI ($r = 0.51$, $p = 0.05$) and HGI ($r = 0.66$, $p = 0.005$) trials.

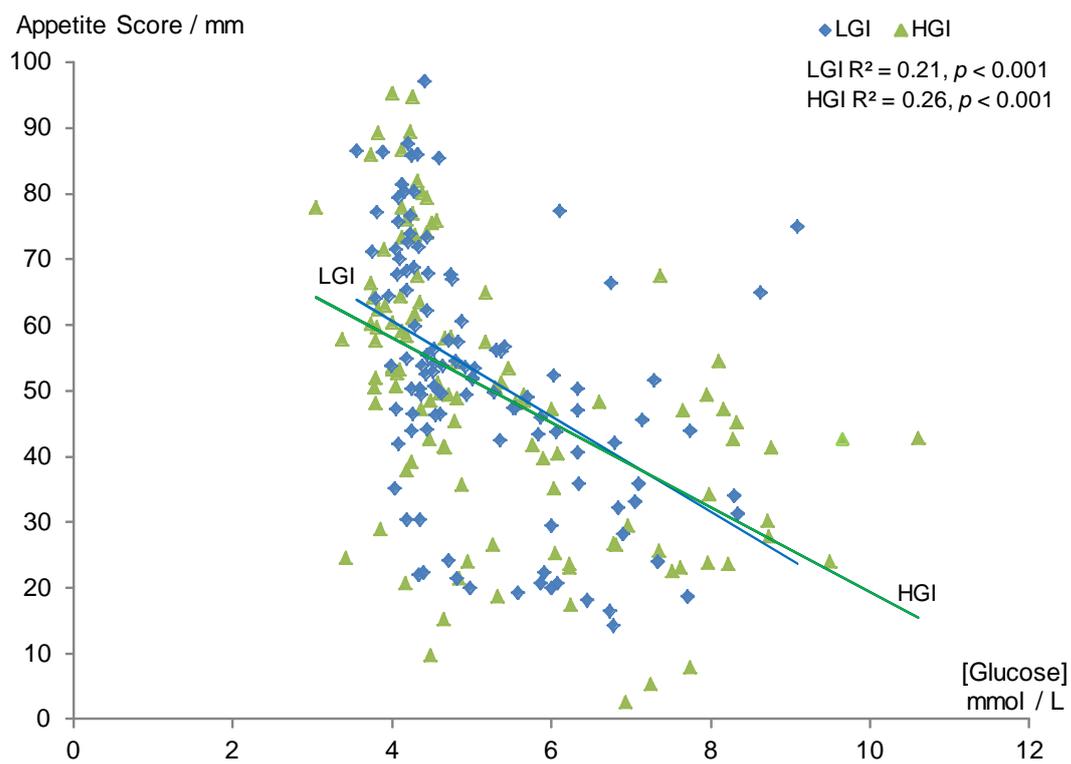


Figure 6.9 Correlation between the glucose concentration and the appetite scores in the low glycaemic index (◆ LGI) (n = 112) and in high glycaemic index trials (▲ HGI) (n = 112)

The LGI post lunch AS was positively associated with its post lunch [Glucose] ($r = 0.51$, $p = 0.04$); whereas the correlation became insignificant after the adjustment of the post lunch PS ($p = 0.06$). A significant negative correlation was found between the HGI post lunch AS and the lunch EI ($r = -0.55$, $p = 0.001$), but no significant correlation was found in the LGI trial ($p = 0.97$). Both lunch EI did not find significant correlations with the [Glucose] before lunch; and after lunch.

6.3.9 Heart rate

Due to the technical fault of the HR transmission, the HR was collected successfully from 12 participants only. No effects of trial ($F(1, 10) = 1.2$, $p = 0.29$) and trial x time interaction ($F(1.4, 14.3) = 0.56$, $p = 0.53$) were found between trials. A significant time effect ($F(1.8, 18.1) = 25.2$, $p < 0.001$) that the HR at task 1 and task 2 were significantly higher than those at the corresponding task 3.

6.4 Discussion

The current study found that the LGI breakfast had cognitive advantages on vigilance over the HGI breakfast, but not on selective attention at late post breakfast period at 105–135 min pp. Cognitive performance was found to be varied with the time interval after food ingestion. However, the second hypothesis of the reduction of lunch EI three hours following LGI breakfast was not supported.

6.4.1 Cognitive performances

In line with previous studies in other age populations (Benton et al., 2003; Cooper et al., 2012; Micha et al., 2010; Nilsson et al., 2009), the vigilance and selective attention were better following LGI than HGI breakfast in recreationally active males in the current study. The main findings were that the HGI postprandial [Glucose] was associated with vigilance impairment at the early and late postprandial periods while the LGI breakfast maintained the sustained and selective performances up to 60–90 minutes post breakfast in RIPT. The advantage of the LGI breakfast on RIPT performance up to 90 minutes postprandially was illustrated from the positive correlations between the task 2 RT and the pre-task 2 [Glucose] equivalent to fasting level in the LGI trial; the positive correlations between the task 1 RT and the high pre-task [Glucose]; and the high total glucose AUC during task 1; as well as from the negative correlations between the number of the adjusted response accuracy and those glucose related variables in the HGI trial. Furthermore, the HGI task 3 RT was negatively correlated with the low pre-task [Glucose]. These correlations implicated that the relatively low pre-task [Glucose] at task 2 in the LGI trial favoured the RIPT RT; whilst either high pre-task [Glucose] at task 1 or low pre-task [Glucose] at task 3 in the HGI trial did not favour the RIPT performances.

A decrement in the SCWT selective attentional performance reflected from the increased interference time at the late postprandial period following the LGI breakfast here was different to the maintenance of the Stroop accuracy at 170-min pp following a simulated LGI trial in older adults (Nilsson et al., 2009). The authors simulated the LGI CHO by providing sips of 8.3 g glucose drink for six times throughout the trial period. The last sip was arranged at 150 minutes

postprandially which was 20 minutes prior to the selective attention test. The simulated LGI [Glucose] remained higher than the fasting level after the completion of the selective attention test whilst the current LGI pre-task 3 [Glucose] was comparable to the fasting level. It might be argued that the HGI task 3 SCWT interference time was maintained despite the comparable pre-task [Glucose] to the fasting level. However, the [Lactate] appeared to have an effect of co-linearity with glucose on the SCWT performances at the LGI task 3. The correlations between the LGI task 3 SCWT interference time and the pre-task [Glucose] ($r = 0.51$, $p = 0.09$); and the total task 3 glucose AUC ($r = 0.24$, $p = 0.3$) were weakened after the adjustment of the pre-task 3 [Lactate]. A previous study found that blood [Lactate] was negatively correlated with performances in the attentional tasks after lactate was infused (Coco et al., 2009). The authors found that the cognitive performance was comparable between the exercise induced hyperlactataemia with the level of 10.1 ± 2.6 mmol·L⁻¹ and the infusion condition with the level of 4.5 ± 0.4 mmol·L⁻¹. Although the current LGI pre-task 3 [Lactate] was not as high as Coco et al. (2009), the additive effect of the [Lactate] on the LGI task 3 interference score for RT should not be ruled out. More research might help to investigate any additive effect of lactate via the metabolism of fructose containing LGI breakfast on the cognitive performances.

The reduced LGI task 3 CHO oxidation was correlated moderately with the increased SCWT interference time. Fischer et al. (2001) investigated the effect of ingestion of different macronutrients on cognitive performances on 15 male aged 26.5 ± 3.3 y with BMI of 21.9 ± 1.7 kg·m⁻². Fat consumption resulted in a highest score in central short-term memory accuracy than following pure CHO

and pure protein consumption; whilst following CHO ingestion had better short-term memory but poorer attention as well as efficiency than following protein. The rates of substrates oxidation differed following different macronutrient consumptions. The findings might implicate that the differences in substrates oxidation had effects on cognitive performances in different areas, such as memory and attention. The authors concluded that optimal cognitive performances were related to stable glucose metabolism and a constant ratio of glucagon to insulin. The current study did not measure the insulin or glucagon concentrations. The mechanism of the association between CHO oxidation and cognitive performances therefore remained unclear.

6.4.2 Appetite and energy intake

Brindal et al. (2012) also did not find a significant difference in the *ad libitum* lunch intake three hours following the breakfasts with different GI and GL. The lack of difference in the lunch intake between trials might be due to the lack of difference in the pre-lunch AS. Although the glucostatic theory suggested low blood [Glucose] initiates food consumption (Mayer, 1955), no significant correlation was found between the lunch EI and the 165-min pp [Glucose]; and the glucose IAUC before lunch. A review suggested that a meal initiation was prompted by transient declines in blood [Glucose] (Campfield & Smith, 2003). The current study fixed the start time of the lunch. The significant positive association between the pre-lunch AS and the lunch EI kg^{-1} FFM in both trials here was in line with Aston et al. (2008). The current subjective appetite sensation was a more sensitive predictor of subsequent EI than the pre-meal [Glucose].

6.4.3 Mood state

It was reported that blood [Glucose] altered the mood state (Benton & Owens, 1993; Degoutte et al., 2006). Sommerfield et al. (2004) suggested that mood was positively affected by high blood [Glucose] including the decrease in the feelings of happiness. The authors found that acute hyperglycaemia deteriorated mood states in type 2 diabetic adults. The current study also found a significant correlation of the depression score with the high HGI 15-min pp [Glucose]. Thus the acute hyperglycaemia induced following a HGI breakfast can also deteriorate the mood state.

6.4.4 Strengths and limitations

A breakfast providing 25% of the daily energy requirement was reported to improve cognitive performance related to creativity compared to the provision of less than 10% in children (Murphy, 2007). The author suggested that energy availability, rather than glucose, influenced the cognitive activities. The test breakfast EI was equivalent to 22% of EI on the pre-trial days which ruled out the provision of low energy test breakfasts here.

Protein is known to be insulinotropic and the postprandial glycaemic response can be affected potentially (Flint et al., 2004; Jenkins et al., 1981), as well as to worsen the RT for the first hour of the cognitive tasks (Fischer et al., 2001). In line with the previous two studies, all meals provided in the current study matched the contents of macronutrient and dietary fibre, as well as energy density between trials. Moreover, a standard pre-trial evening meal was given

in the current study to minimise any meal effects on the trial days (Lamport et al., 2012).

Blundell et al. (2010) advised avoiding a buffet style food provision in an appetite related research, unless a specific hypothesis relating to food choice is to be investigated as the buffet style serving foods with a variety of physical state, energy density, macronutrient composition and sensory characteristics might confound the effect of the pre-load. Conversely, serving a single course would strengthen the focus on the assessment of the intakes of food and energy rather than nutrients which facilitated the investigation of the regulation of EI in short term based. In line with Blundell et al. (2010), lunch was served as a single course in the current study. Furthermore, the significant trial x time interaction in the postprandial glucose profiles, the peak [Glucose] and the glucose IAUC also implied a success in using the calculated method of Wolever and Jenkins (1986) to set the test breakfasts to have significant difference in postprandial glucose response in study one, two and three.

There existed some limitations in the current study design that should also be taken into account. The measurement of the capillary blood [Glucose] is a measure of the peripheral [Glucose] rather than the brain [Glucose]. In addition, the glucose responses during the tasks were measured discretely. Dickinson, Colagiuri, Faramus, Petocz, and Brand-Miller (2002) found that the [Glucose] reached peak at 30–45 minutes in young lean adults following 75 g glucose consumption. The current postprandial glucose profiles might not be closely reflected enough and correlated with the cognitive performances. A continuous

monitoring of [Glucose] might help to better investigate the correlation between [Glucose] and cognitive performances.

The current study did not assess the cognitive performance at fasting conditions as the advantage of breakfast consumption on cognition has already been thoroughly investigated. Nevertheless, testing the cognitive performances at fasting as baselines might help to observe the magnitude of the Stroop interference time in the LGI and HGI trials. In addition, the metabolic activity in prefrontal areas of brain was reported to be correlated with the SCWT performances (Volkow et al., 2009). Assessment of the brain activity or brain energy metabolism might help to further investigate the association between the GI and the cognitive performances.

6.5 Conclusion

The results showed the potential impairment in the sustained attention and vigilance performance evaluated by the RIPT was found to be associated with the HGI breakfast at the early post breakfast period in both response time and accuracy; and at the late post breakfast period in the response time. The selective attention evaluated by the SCWT interference time was not maintained following the LGI breakfast at the late postprandial period. Recreationally active males participating in a sport with low physical activity level may benefit from the LGI breakfast if vigilance and selective attention need to be maintained within 1.5 hours post breakfast. Furthermore, pre-lunch appetite sensation was a more sensitive predictor of the lunch intake. Suppressing the pre-meal appetite would be a more efficient strategy to control

the upcoming meal intake than monitoring the pre-meal glucose level. It is necessary to further investigate any dose-response relationship between CHO dose at breakfasts and cognitive performances; and the lunch intake; the timing for task performance and the GI effect at breakfasts on brain utilization of glucose during demanding cognitive tasks so as to optimise the cognitive performance for recreationally active males.

CHAPTER 7 STUDY FOUR: COGNITIVE AND METABOLIC RESPONSES DURING INTERMITTENT EXERCISE AFTER CONSUMPTION OF A LOW AND A HIGH GLYCAEMIC INDEX BREAKFASTS IN RECREATIONALLY ACTIVE MALES

7.1 Introduction

The reliance on CHO oxidation as an energy source increases with increasing exercise intensity (Coyle, 1995). A depletion of glycogen during intense exercise due to higher metabolic need over the provision of energy can lead to fatigue (Rodriguez, Di Marco, & Langley, 2009). The benefits from pre-exercise CHO intakes include the delay of muscular glycogen depletion and the maintenance of blood [Glucose] for both muscle and brain as an energy fuel for cognitive, emotional, motivational and motor skill performance (Welsh et al., 2002). Although the maintenance of an adequate supply of CHO for muscle and the central nervous system is important for reducing the risk of injury, optimising exercise and potentially psychological performance during exercise, the difference in bioavailability of CHO following foods with different GI taken prior to an exercise event should be of concern. Hypoglycaemia is known to cause deterioration in cognitive performance in healthy participants (McCrimmon et al., 1996); whilst acute hyperglycaemia was reported to impair cognitive performance in type 2 diabetic adults (Sommerfield et al., 2004). Moreover, fluctuations of blood glucose level, even within normative range, may also exert influences on cognitive performances (Fairclough & Houston, 2004).

The effects of the pre-exercise consumption of CHO with different GI have been investigated mainly on endurance exercise (Donaldson et al., 2010; O'Reilly, Wong, & Chen, 2010). However, the majority of research has focused on the investigation of the benefits of GI manipulation on endurance exercise performance. Limited research has investigated the effect of the CHO consumption on both physical and mental performance during intermittent exercises in recreationally active adults (Welsh et al., 2002; Winnick et al., 2005). Not until recently has the acute effect of a pre-exercise meal with different GI values on intermittent exercise been investigated (Cocate et al., 2011; Hulton et al., 2012; Little et al., 2010). However there existed discrepancy in the substrates oxidation and exercise performances in those previous literatures. The discrepancy may be related to the time that exercise started following a meal and is worthy to be further investigated.

An appropriate strategy to consume CHO is important for physically active populations to reduce performance impairment resulting from poor mood state, fatigue or stress. No CHO intake is generally required during exercise less than 60 minutes (Burke & Deakin, 2010). Thus the pre-exercise CHO intake plays an important role in substrate oxidation during exercise less than 60 minutes. Although ecologically pre-exercise CHO intake was recommended to be consumed 3-4 hours prior to an event for athletes (Hulton, Edwards, Gregson, Maclaren, & Doran, 2013; Hulton et al., 2012), it might not be appropriate for recreationally active males. A meal taken one hour prior to exercise results in increased secretion of insulin (Brun, Dumortier, Fedou, & Mercier, 2001). The occurrence of hypoglycaemia during the early exercise period, probably resulted from the reduced production of glucose in liver, as well as during the

later exercise period might affect the overall exercise performance. It remains unknown if a LGI breakfast is advantageous for cognitive function during exercise compared to a HGI breakfast for recreationally active males. Many sports such as team sports, contact sports and rackets sports encompass physical and cognitive loads simultaneously. There has been no study that specifically compares the effects of LGI and HGI breakfasts on physiological measures and cognitive function during and after an intermittent running in recreationally active males.

The aim of this study was therefore to compare the effects of a LGI or a HGI breakfast on metabolic responses and cognitive performances in recreationally active males during intermittent running. The objectives were to determine and compare the difference between trials in:

- substrate oxidation over time during exercise; and
- selective attention and vigilance during and after exercise.

It was hypothesized that the metabolic responses and mental function during high-intensity intermittent exercise would be significantly enhanced following a LGI breakfast, compared to a HGI breakfast in recreationally active males.

7.2 Methodology

7.2.1 Participants

Participants were informed that the purpose of the study was to investigate the difference in the exercise and cognitive performance between two different breakfasts, without debriefing the main principles of the investigation of the effects of GI on cognitive performance and metabolic response. Additional inclusion and exclusion criteria were Caucasian males and colour blindness, respectively. All participants were healthy and suitable to participate in the lactate threshold (LT) and $\dot{V}O_2$ max tests as assessed by the physical activity readiness questionnaire (Appendix T).

7.2.2 Study design

The study design was to examine the effects of a LGI and a HGI breakfasts on cognitive performances and substrates oxidation during intermittent running. Participants visited the laboratory on two occasions for the trials with a washout period of not less than two days (Brouns et al., 2005; Warwick et al., 1993). Participants were pseudo-randomly assigned in a counter-balanced order to receive a LGI or a HGI breakfast for the first trial with the remaining test breakfast consumed on the second trial. At each trial, participants completed three sessions of intermittent running and cognitive tests one hour onwards after breakfast. Capillary and venous blood samples were taken and mood states were assessed at assigned time slots. Whenever possible, both trial days were on the same day of the week to help control for day-to-day variation in EI and EE (Warwick et al., 1993).

7.2.3 Preliminary arrangements

Participants visited the laboratory at least a week before the start of the main trials to perform a LT test and a $\dot{V}O_2$ max test, as well as to be familiar with the computerised cognitive tasks to eliminate potential learning effects during the experimental sessions (Welsh et al., 2002).

7.2.3.1 Lactate threshold

The LT test was performed on a programmable motorised treadmill (Woodway PPS 55 Sport slat-belt treadmill, Woodway GmbH, Weil am Rhein, Germany) to determine the speed at LT (vLT). Capillary blood [Lactate] was determined using a portable lactate analyser (Lactate Pro, Arkray, Japan) prior to the start of the test. The analyser was calibrated by a calibration strip according to the manufacturer's guidelines before use. Participants then warmed up involving stretching and walking on the motorised treadmill at their self-selected walking speeds. Participants wore a respiratory mask connected to the automated gas analyser. Expired air collected over the last 15 seconds of each stage was used for analysis of the oxygen consumption and RER. Participants started at their own fast walking speed on a treadmill with a constant inclination of 1% (Jones & Doust, 1996). The speed increased $1 \text{ km}\cdot\text{hr}^{-1}$ every three minutes. A one-min break was provided between speed increments for capillary blood lactate measurement (Bentley, Newell, & Bishop, 2007; Billat, 1996). The LT test was completed when the blood [Lactate] increased at least $1 \text{ mmol}\cdot\text{L}^{-1}$ after one speed level and was above $4 \text{ mmol}\cdot\text{L}^{-1}$; and the HR of participants reached 85-90% of their age-predicted HRmax ($\text{HRmax} = 220 - \text{age}$) (Nicholas, Williams, Lakomy, Phillips, & Nowitz, 1995).

7.2.3.2 Maximal oxygen uptake test

The $\dot{V}O_{2\max}$ test started after a break of 10–15 minutes from the LT test to establish the relationship between $\dot{V}O_2$, expressed relative to BM ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and running speed so as to determine the $\dot{V}O_{2\max}$ and the speed at the $\dot{V}O_{2\max}$ ($v\dot{V}O_{2\max}$). The running speed was the last speed at the LT test minus $2\text{ km}\cdot\text{hr}^{-1}$ and kept constant during the test. The slope started from 1% and then increased 1% every minute. Borg's 6–20 rating of perceived exertion (RPE) was reported at the last 15 seconds of each grade (Borg, 1970). The test was completed when participants expressed that they could not continue on for the next grade and were volitionally exhausted. The RPE was reported again at exhaustion. A capillary blood sample was collected and analysed for [Lactate] at exhaustion.

Regression equation via extrapolation of the data provided the $\dot{V}O_{2\max}$ estimate. The $\dot{V}O_{2\max}$ was determined when the two of the following criteria were met (British Association of Sports Sciences, 1988):

- a plateau $\dot{V}O_2$, an increase in $\dot{V}O_2$ less than 5% or $2\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ was noted when increasing exercise intensity,
- the HR was within $10\text{ beats}\cdot\text{min}^{-1}$ of the age predicted HRmax ($220-\text{age}$),
- the RER was above 1.15, and
- the post-exercise blood [Lactate] was above $8\text{ mmol}\cdot\text{L}^{-1}$ at exhaustion.

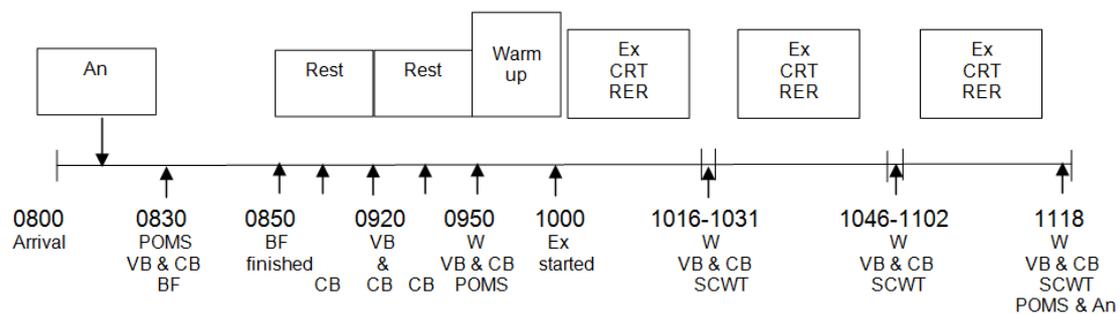
The $\dot{V}O_2$ value at maximal work was referred to as $\dot{V}O_{2peak}$, when the above criteria were not met. The vLT and the $v\dot{V}O_{2max}$ were determined which were later employed in the main trials.

Participants were instructed to consume no alcohol and participate in no vigorous exercise 24 hours prior to the trial visit. They were required to record and keep similar food intake prior to every trial (Brouns et al., 2005; Little et al., 2010). Food records were analysed by the same research dietician using the locally developed nutrient analysis software. Ten hours prior to the arrival of the laboratory and in order to minimize dehydration before each main trial, participants were reminded to drink around 500 mL of water.

7.2.4 Trial procedures

Participants reported to the laboratory between 0700 and 0900 hours after an overnight fast for 10 hours (Brouns et al., 2005). The anthropometric measurement was taken by the standard procedures described in section 3.8. A glass of water (250 mL) was served afterwards. A capillary blood and a venous blood sample were then taken. Afterwards participants completed the POMS questionnaire (Shacham, 1983). In order to standardize the meal finishing time, participants were instructed to consume the meal over a period of 20 minutes once the breakfast was served (Brouns et al., 2005). A glass of water (250 mL) was served aside to be consumed *ad libitum*. Any leftovers were recorded. The alternative breakfast with the same energy content and density; and dietary fibre was provided at the second trial.

Participants remained seated in the laboratory for an hour after breakfast. They were asked to keep their physical activity minimum. Capillary blood samples were taken every 15 minutes for an hour after breakfast. Venous blood samples were collected at 30- and 60-min pp. Participants were encouraged to drink water during the rest time at the first trial. The same volume of water was provided at the second time.



Keys:

An	Anthropometric measurement	POMS	Profile of mood state assessment
BF	Breakfast	RER	Expired gas collection
CB	Capillary blood collection	VB	Venous blood collection
CRT	Choice reaction time task	SCWT	Stroop colour word task
Ex	Exercise session	W	Water

Figure 7.1 Schema of the trial procedure

Participants completed the POMS questionnaire again before the start of the first exercise session. A 10-min warm up consisted of light stretching and motion on the treadmill at the participants' self-selected speeds. Participants then performed a 16-min intermittent exercise on a treadmill with a HR transmitter and a gas collection mask being worn. Expired gas was collected via the gas collection mask with a sampling line connected to the gas analyser. The HR was recorded synchronously by the gas analyser. Participants

performed the CRT task during walking motion. The RPE was rated immediately after each exercise session. A 15-min break was given. During the break, capillary and venous blood samples were taken. Water was provided at a volume of 3 mL·kg⁻¹ BM after each exercise session. Participants consumed water on an *ad libitum* basis throughout the first trial. The volume of water consumed during the first trial was recorded. The same volume of water was provided at the second trial. Participants were encouraged to consume all water provided at the second trial to standardize the hydration status. Participants then performed the SCWT after blood collection. After the last SCWT, participants were asked to complete the last POMS. Body mass was measured again before departing the laboratory (Figure 7.1).

7.2.5 Exercise protocol

The exercise period included three sessions being performed on the programmable motorised treadmill. Each exercise session comprised eight 2-min bouts of intermittent running, consisting of sprinting, interspersed with less intense periods of running and walking (Hulton et al., 2012; Nicholas, Nuttall, & Williams, 2000). Each bout included four different speeds at even time length in a sequence: walking, jogging, sprinting and then running (Table 7.1). A constant incline of 1% was used to reflect and mimic the energy cost of outdoor running (Jones & Doust, 1996). Exercise intensity was expressed as a percentage of $\dot{V}O_2\text{max}$.

Table 7.1 An example of the sequence of one exercise cycle

Mode	Speed equivalent	Intensity	Duration (s)
Walk	20% vLT	Low	30
Jog	80% vLT	Moderate	30
Sprint	120% $\dot{V}O_2$ max	High	30
Run	50% ($\dot{V}O_2$ max-vLT) + vLT	Moderate-high	30
Total			120

7.2.6 Cognitive tasks

Three batteries of computerised cognitive function tasks were administered using the E-prime psychological software during the exercise period. The setting for performing the computerised cognitive tasks was described in section 3.9.

7.2.6.1 Choice reaction time

The CRT was performed during each walking motion. Four different images, red circle, blue circle, red square and blue square, appeared one at a time randomly on the white background computer screen. The right responses were the red circle or the blue square. Participants were required to press the labelled key designated for red circle or blue square as quickly as possible and to ignore the red square and the blue circle when they appeared. Each 2-min bout had 16 stimuli. The stimulus duration was 1,500 ms; with the stimulus disappearing once participants pressed the labelled key for a response. A fixed inter-stimuli interval of 100 ms was set. The software recorded the response accuracy and the errors; and the RT for the correct responses and the wrong responses. The response accuracy and the errors were considered as dependent variables. An error was defined as incorrect responses to a non-

desired response and a failure to respond to a requested response. A correct response quicker than 150 ms was also considered as an error (Davranche & Audiffren, 2004; Luciano et al., 2004). The calculation of the number of adjusted correct responses was described as follows:

Number of adjusted correct response = number of correct response + number of null response to the non-requested response – number of missing response – number of wrong response

7.2.6.2 Stroop colour word task

The SCWT was performed within five minutes after each exercise session. The first level, for practice, had 36 stimuli in four different images in white. The second level was a colour-interference level which included the conditions of neutral, congruent and incongruent each contained 12 stimuli. The stimulus duration was 1,500 ms; whilst the stimulus disappeared once participants pressed the labelled key for response. The design and the calculation of the SCWT scores were explained in section 3.9.1.

7.2.7 Test breakfasts

The test breakfasts provided 1.2 g CHO kg⁻¹ BM. The energy contributions from CHO, protein and fat were 60%, 15% and 25% respectively. The calculated GI values of the LGI and HGI breakfasts were 42 and 72.5 respectively with a difference of 30.5. The energy density and fibre contents were matched between breakfasts (Table 7.2 and 7.3).

Table 7.2 An example of the energy content and nutrient profile of a LGI breakfast (for a 75 kg participant)

Food Name	Quantity GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Semi-skimmed milk ¹	32	415	19.5	14.5	7.1	191	0.0	6.9
Muesli ²	55	62	41.3	6.5	3.6	223	4.3	25.2
Apricots, dried ³	32	22	9.5	1.1	0.2	41	1.7	3.4
Fructose ⁴	19	13	12.9	0.0	0.0	47	0.0	2.7
Raisin ⁵	54	10	6.6	0.2	0.0	26	0.2	3.9
Macadamia	0	5	0.2	0.4	3.5	34	0.2	0.0
		525	90	22.6	14.4	561	6.4	42.2

CHO, carbohydrate; GI, glycaemic index; NSP, non-starch polysaccharide. ¹ Tesco organic 1.7% semi-skimmed pasteurised milk; ² Alpen Swiss muesli; ³ Tesco Ready to Eat Dried Apricots; ⁴ Fruisana fruit sugar and ⁵ Sun-Maid Natural California Raisins

Table 7.3 An example of the energy content and nutrient profile of a HGI breakfast (for a 75 kg participant)

Food Name	Quantity GI	Quantity (g)	CHO (g)	Protein (g)	Fat (g)	Energy (kcal)	NSP (g)	Item GI
Rice milk ¹	79	174	16.5	0.2	1.8	82	0.2	14.4
Corn Flakes ²	93	21	19.1	1.7	0.3	80	0.2	19.7
Fruit & Fibre ³	67	70	51.1	6.3	3.4	249	4.8	38.0
Light cheese	0	52	0.2	13.3	6.2	111	0.0	0.0
Cherry tomato	0	103	3.1	0.8	0.4	19	1.0	0.0
Macadamia	0	3	0.1	0.2	2.3	22	0.2	0
Water	0	102	0.0	0.0	0.0	0	0.0	0.0
		525	90	22.5	14.4	563	6.4	72.2

CHO, carbohydrate; GI, glycaemic index; NSP, non-starch polysaccharide. ¹ Rice Dream Organic Original Rice Milk; ² Kellogg's cornflakes and ³ Tesco Fruit & Fibre

7.2.8 Substrate oxidation

Expired gas was collected whilst participants were performing intermittent exercise. The calibration of the gas analyser and the measurement of the substrate oxidation from the expired gas were described in the section 3.5.3.

7.2.9 Biochemical analyses

7.2.9.1 Capillary blood sampling

Capillary blood samples were taken before breakfast, 15-, 30-, 45- and 60-min pp; and after each exercise session (90-, 120- and 150-min pp). Samples were analysed for the whole blood [Glucose] and [Lactate] using the automated glucose and lactate analyser. The procedures of the capillary blood sampling were described in section 3.10.

7.2.9.2 Venous blood sampling

All venous blood samples were taken by trained and experienced staff. An indwelling catheter (18 G x 32 mm BD Venflon™ Pro Peripheral IV Catheter with Injection port, Becton Dickinson Ltd., U.K.) was inserted into the antecubital vein of the forearm attached to a 10 cm extension tubing to allow for repeated blood sampling. Venous blood samples were collected before breakfast, 30- and 60-min pp; and after each exercise session. The cannula was kept patent by flushing with 2.5 mL isotonic saline (PosiFlush™ Syringe 0.9% sodium chloride, Beckton Dickinson, Oxford, U.K.) after each blood sample collection. Prior to the subsequent blood sampling, the first 2.5 mL of blood was taken by an empty syringe and then discarded. A venous blood sample was collected by a 6 mL green top lithium heparin vacutainer (Beckton Dickinson, Oxford, U.K.) at each time point. The vacutainers were shaken following the manufacturer's

instruction and then immediately placed in ice before centrifuging at 4000 r.p.m. for 10 minutes at 4°C to separate plasma (Stevenson, Williams, & Nute, 2005; Stevenson et al., 2008). The plasma was then stored at -80°C in multiple aliquots for later analysis for electrolytes (Roche 9180 electrolyte analyser, Roche Diagnostics GmbH, Mannheim, Germany) and insulin using the commercially available enzyme-linked immunoassay (ELISA) kits (Insulin ELISA (RE53171), IBL International GmbH, Germany), respectively.

Whole blood haemoglobin (Hb) was determined by dipping a small amount of venous blood sample on a microcuvette (HemoCue Hb 201 Microcuvette, HemoCue Ltd., Derbyshire, U.K.) and then immediately analysed (HemoCue Hb 201⁺ Analyser, HemoCue Ltd., Derbyshire, U.K.). Heparinised capillary tube was used to collect ~ 100 µl blood for measuring haematocrit (Hct) by microcentrifugation (Haematospin 1300, Hawksley Ltd., Sussex, U.K.). The Hct was then determined using an Hct reader (Haematocrit tube reader, Hawksley Ltd., Sussex, U.K.). The change in the plasma volume (Δ PV) was calculated using the equation of Costill, Branam, Eddy, and Fink (1974):

$$\Delta PV = \left[\left(\frac{Hb_1}{Hb_2} \right) \times \left(\frac{100 - Hct_2}{100 - Hct_1} \right) \right] - 1 \times 100\%$$

where Δ PV = change in plasma volume; ₁ = baseline and ₂ = end values

Venous blood parameters were collected in duplicate and a mean reading was taken for analysis if possible. The plasma insulin were analysed by the same kit, if possible, to eliminate the inter-assay variability (Ford et al., 2011). The

manufacturer of the insulin ELISA kit reported the intra- and the inter-assay coefficient of variation to be 1.8–2.6% and 2.9–6.0%, respectively. The coefficient of variations for glucose, lactate, Hb and Hct were 4.4%, 7.9%, 1.6% and 3.7%, respectively (Appendix U).

7.2.10 Subjective sensations

The POMS was completed at fasting, before the start of exercise at 60-min pp and at the end of the last SCWT at around 155-min pp. The design of the POMS was described in section 3.6.2. The RPE was rated at the end of each exercise session.

7.2.11 Heart rate

Heart rate was monitored continuously at a one-second interval default by the gas analyser using the Polar telemetry. Mean HR was calculated for each 16-min exercise session.

7.2.12 Statistical analysis

7.2.12.1 Sample size calculation

Sample size was calculated at an alpha level of 0.05 with a power of 80% using the equation from National Research Council Committee (2003) (Appendix N) from two previous papers (Little et al., 2010; Stannard, Constantini, & Miller, 2000). Fourteen participants were required to show a significant difference in the muscle glycogen concentration of $92.2 \text{ mmol}\cdot\text{kg}^{-1}\cdot\text{dw}^{-1}$ after 75 min of intermittent running between the LGI and HGI trials (Little et al., 2010) and a power number of 11 was calculated to have a significant difference between a

pasta and glucose meals in the RER of 0.08 (Stannard et al., 2000). Thus the mean sample size required was 13 in order to achieve a statistical power of 80% at statistically significance at two-tailed level at 95% or 0.05.

7.2.12.2 Data analyses

Descriptive statistics were calculated for group characteristics. Data were analysed in two time periods: the pre-exercise period (fasting to 60-min pp) and the exercise period (60- to 150-min pp).

The glucose IAUC ignoring the negative values was calculated (Wolever, 2004; Wolever & Jenkins, 1986). The [Glucose], [Lactate], [Insulin] and [FFA]; the cognitive outcomes; and substrates oxidation were analysed via two-way (trial x time) repeated-measure ANOVA to detect the effects of trial, time and trial x time interaction. If any interaction effect was found, Tukey's *post-hoc t*-test was computed to locate the significant difference with the Bonferroni correction. Pearson's correlation analyses were applied to examine the associations among the [Glucose], [Lactate], [Insulin] and [FFA]; the cognitive outcomes; and the substrates oxidation. Non-parametric Friedman ANOVA, Spearman's correlation and Wilcoxon signed-rank test were used when the parameters were not normally distributed.

A z-score was calculated for substituting any missing data when appropriate. One set of SCWT data were replaced by the z-score due to a fire alarm within

the building during a trial. Criterion for statistical significance was $p < 0.05$ in two-tailed level.

7.3 Results

7.3.1 Characteristics of the participants

Originally 18 recreationally active males were recruited in the study. Two participants withdrew because of illness after the completion of the pre-trial $\dot{V}O_2$ max running test. Those data were not used for analysis.

Table 7.4 Anthropometric and physiological characteristics of the participants

Variables	n = 16
Age (year)	27.8 ± 7.7
Body mass (kg)	76.7 ± 10.3
BMI (kg·m ⁻²)	23.6 ± 3
Body fat percentage (%)	22.1 ± 6.3
Fat free mass (kg)	59.3 ± 4.9
$\dot{V}O_2$ at LT (mL·kg ⁻¹ ·min ⁻¹)	34.1 ± 6.3
vLT (km·hr ⁻¹)	9.6 ± 1.5
HR at $\dot{V}O_2$ max (beats·min ⁻¹)*	192 ± 8
$\dot{V}O_2$ max (mL·kg ⁻¹ ·min ⁻¹)	50.5 ± 8.3
v $\dot{V}O_2$ max (km·hr ⁻¹)	13.8 ± 2.6

Values are means ± SD; n, number of participants, * n = 15; BMI, body mass index; HR, heart rate; LT, lactate threshold; vLT, speed at lactate threshold; $\dot{V}O_2$ max, maximal oxygen uptake; v $\dot{V}O_2$ max, speed at maximal oxygen uptake

Sixteen participants aged 27.8 ± 7.7 years completed the study successfully.

The anthropometric and physiological characteristics of participants were shown

in Table 7.4. Body mass changes during exercise were comparable between the LGI and HGI trials (0.05 ± 0.5 vs. 0.06 ± 0.6 kg, $t(12) = -0.04$, $p = 1.0$). Due to the technical fault of the HR transmission, one HR reaching $\dot{V}O_{2\max}$ at the $\dot{V}O_{2\max}$ test was not recorded.

7.3.2 Glycaemic index of meals

The GI value of the LGI breakfast was significantly lower than the HGI breakfast (42.0 ± 0.7 vs. 72.8 ± 1.1 , $t(15) = -72.7$, $p < 0.001$). All test breakfasts were completely consumed.

7.3.3 Biochemical analyses

7.3.3.1 Glucose

There was no significant difference in the fasting [Glucose] between the LGI and HGI trials (4.3 ± 0.34 vs. 4.2 ± 0.36 mmol·L⁻¹, $t(15) = 0.56$, $p = 0.59$). Significant effects of trial ($F(1,15) = 12.7$, $p = 0.003$), time ($F(2.4, 35.2) = 57.6$, $p < 0.001$) and trial x time interaction ($F(2.8, 41.8) = 9.6$, $p < 0.001$) were found during the pre-exercise period. The blood [Glucose] reached peak at 15-min pp at both trials. The *post hoc t*-test showed that HGI peak [Glucose] was significantly higher than the LGI peak [Glucose] (7.8 ± 0.69 vs. 6.7 ± 0.99 mmol·L⁻¹, $t(15) = 5.6$, corrected $p < 0.005$). The LGI [Glucose] at 45-min pp (4.9 ± 0.92 mmol·L⁻¹; $t(15) = 2.5$, corrected $p = 0.13$); and at 60-min pp (4.7 ± 0.68 mmol·L⁻¹; $t(15) = 1.9$, $p = 0.073$) were comparable to the fasting level (4.3 ± 0.34 mmol·L⁻¹); whilst the HGI [Glucose] at 45-min pp (5.4 ± 0.99 mmol·L⁻¹, $t(15) = 3.6$, corrected $p = 0.007$) and at 60-min pp (5.0 ± 0.75 mmol·L⁻¹, $t(15) = 0.72$, corrected $p = 0.013$) remained significantly higher than the fasting level

($4.2 \pm 0.36 \text{ mmol}\cdot\text{L}^{-1}$) (Figure 7.2). No significant difference was found between the LGI and HGI 60-min pp [Glucose] (4.7 ± 0.68 vs. $5.0 \pm 0.75 \text{ mmol}\cdot\text{L}^{-1}$, $t(15) = -1.19$, $p = 0.25$) (Appendix V).

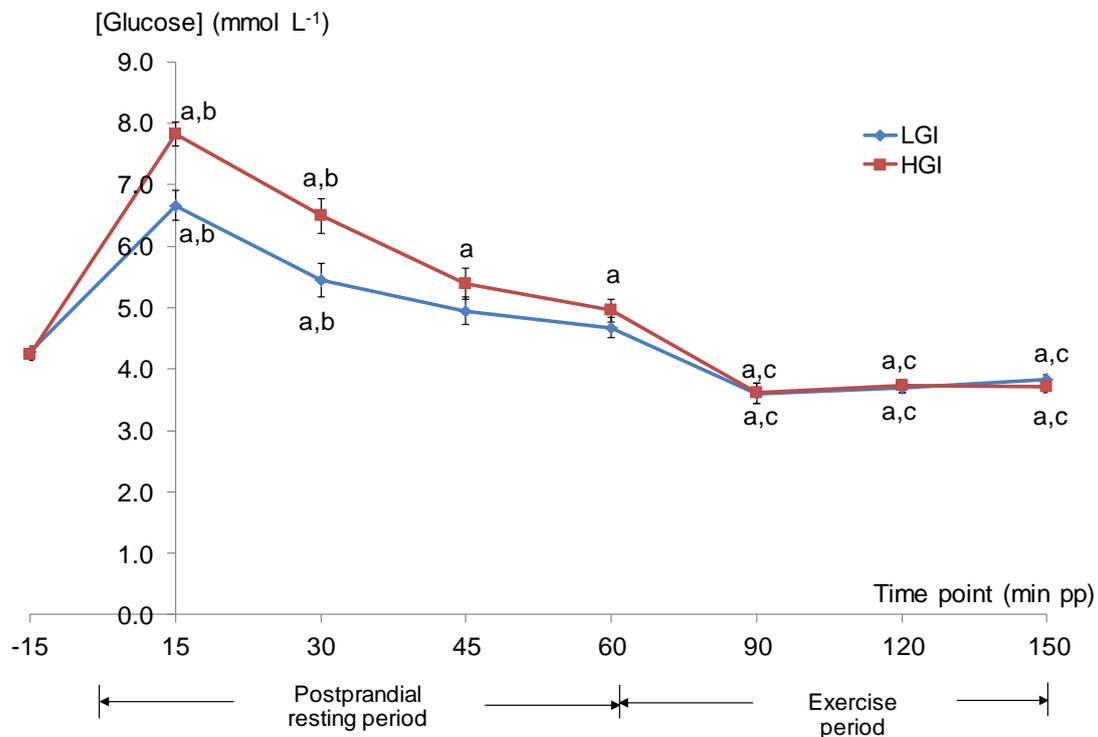


Figure 7.2 Effect of the low glycaemic index (LGI) and the high glycaemic index (HGI) breakfasts on the glucose responses

Data presented as mean \pm SD ($n = 16$). min pp, minutes postprandially; ^a significantly different to the corresponding fasting level (corrected $p \leq 0.013$); ^b significant difference when sharing the same superscript at the same time point (corrected $p \leq 0.01$); and ^c significantly different to corresponding pre-exercise levels (corrected $p \leq 0.003$).

No time \times trial effect existed in the period between 60-min pp and the exercise session. Only a time effect was found during this period ($F(2.1, 31.4) = 25.2$, $p < 0.001$). The post exercise [Glucose] at each session were significantly lower than the corresponding pre-exercise [Glucose] in both trials and remained at the hypoglycaemic levels (Figure 7.2). The LGI glucose IAUC to 60-min pp was

significantly lower than the HGI (86 ± 50 vs. 137 ± 49 mmol·L⁻¹·min, $t(15) = -4.2$, $p = 0.001$).

7.3.3.2 Lactate

The LGI fasting lactate level did not differ to that of the HGI trial (0.62 ± 0.05 vs. 0.56 ± 0.05 mmol·L⁻¹, $t(15) = 1$, $p = 0.33$). Significant main effects of trial ($F(1,15) = 32.4$, $p < 0.001$), time ($F(2.6, 38.3) = 77.9$, $p < 0.001$) and trial x time interaction ($F(2.7, 40.4) = 6.2$, $p = 0.002$) were found in the pre-exercise period (Figure 7.3). The [Lactate] increased significantly following test breakfasts in both trials and maintained at peak levels since 15-min pp before the onset of exercise after the Bonferroni correction. The LGI peak [Lactate] remained significantly higher than the corresponding HGI peak [Lactate]; of which the LGI [Lactate] was significantly higher when compared to the HGI [Lactate] at 60-min pp (1.38 ± 0.07 vs. 1.12 ± 0.05 mmol·L⁻¹, $t(15) = 3.6$, corrected $p = 0.013$) (Appendix V).

There were effects of time ($F(1.6, 24.3) = 28.7$, $p < 0.001$) in the exercise period (Figure 7.3). Blood [Lactate] reached the second peak after the first exercise session when compared to the pre-exercise levels in both trials. The LGI post exercise [Lactate] at each session did not differ to the corresponding HGI levels.

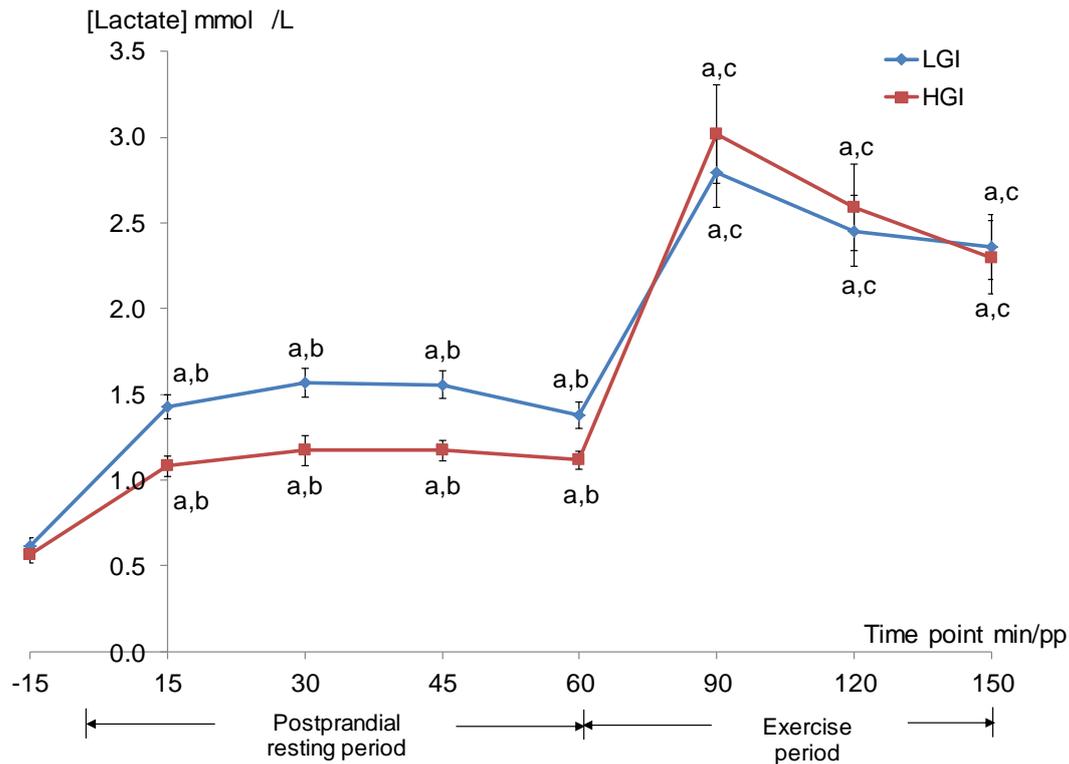


Figure 7.3 Effect of the low glycaemic index (LGI) and the high glycaemic index (HGI) breakfasts on the lactate concentration ([Lactate])

Data presented as mean \pm SD ($n = 16$). min pp, minutes postprandially; ^a significantly different to the corresponding fasting level (corrected $p < 0.001$); ^b significant difference when sharing the same superscript at the same time point (corrected $p \leq 0.013$); and ^c significantly different to corresponding levels pre-exercise level (corrected $p < 0.002$).

Since the LGI [Lactate] at the 60 min-pp was significantly higher than the HGI, changes in the post exercise session [Lactate] (Δ [Lactate]) from the 60-min pp were analysed. Only the LGI post third session Δ [Lactate] was normally distributed. The Friedman ANOVA found a significant effect on the Δ [Lactate] during exercise period ($\chi^2(5, n = 16) = 33.8, p < 0.001$). However, the Wilcoxon signed-rank test did not detect significant differences in the Δ [Lactate] at each exercise session between trials after the Bonferroni correction (corrected $p > 0.05$).

7.3.3.3 Plasma insulin

No significant differences were found between the LGI and HGI fasting plasma [Insulin] (101 ± 62 vs. 85 ± 41 pmol·L⁻¹, $t(15) = 1$, $p = 0.3$). No significant trial x time interaction were found during the pre-exercise ($F(2,30) = 2.6$, $p = 0.88$) and exercise ($F(3,45) = 0.5$, $p = 0.67$) periods. Significant time effects were found that the [Insulin] reached a peak at 30 minutes post breakfast during the pre-exercise ($F(1.6, 23.9) = 41.5$, $p < 0.001$) and the exercise period ($F(1.5, 22.7) = 37.6$, $p < 0.001$) (Figure 7.4). The LGI insulin IAUC to 60-min pp did not differ to the HGI ($12,900 \pm 10,555$ vs. $16,305 \pm 6,792$ pmol·L⁻¹·min, $t(15) = -1.8$, $p = 0.09$).

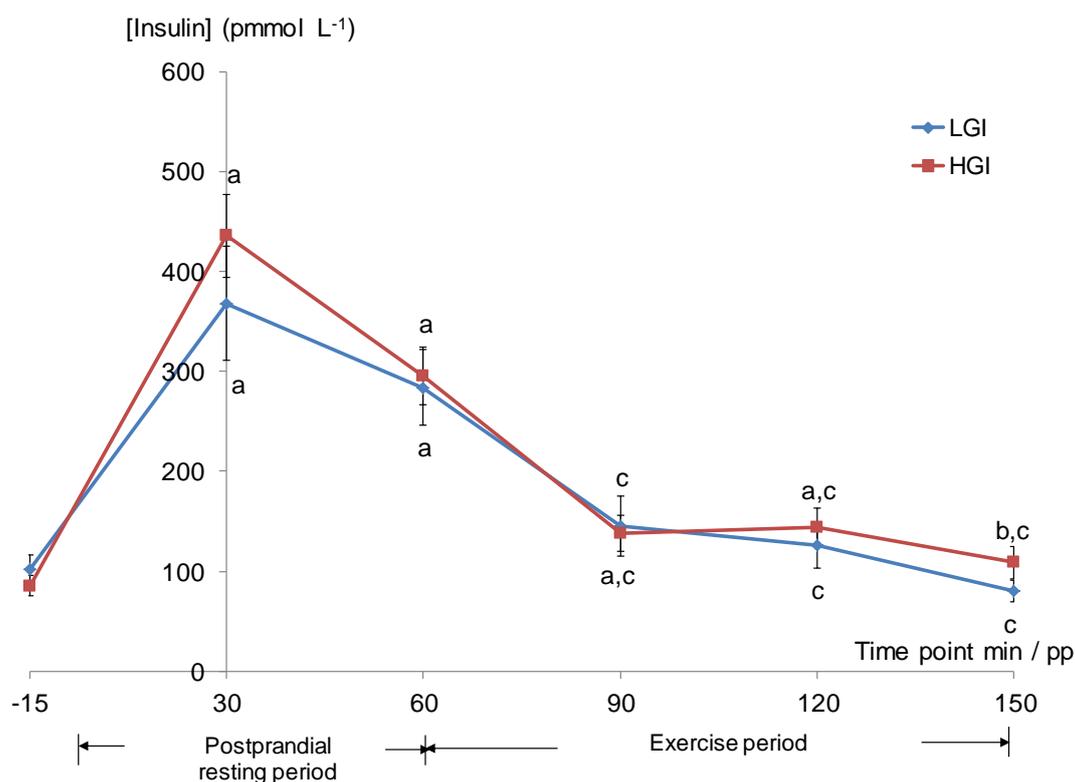


Figure 7.4 Effect of the low glycaemic index (LGI) and the high glycaemic index (HGI) breakfasts on the insulin concentration ([Insulin])

Data presented as mean \pm SD ($n = 16$). min pp, minutes postprandially; ^a significant difference to the corresponding fasting levels (corrected $p \leq 0.002$); ^b significantly higher than the LGI level at the same time point (corrected $p = 0.009$); and ^c significantly different to the corresponding level at 60-min pp (corrected $p \leq 0.001$).

7.3.3.4 Plasma volume and electrolyte

The two-way ANOVA did not find any significant effects of trial x time interaction ($F(2.0, 29.8) = 2.3, p = 0.1$), trial ($F(1, 15) = 0.2, p = 0.67$) and time ($F(1.6, 24.5) = 1.2, p = 0.3$) in the ΔPV between trials during exercise. No significant trial x time interactions were found in the plasma [Na] ($F(2.5, 37.7) = 1.1, p = 0.37$) as well as [K] ($F(2.1, 31.6) = 0.27, p = 0.78$) between trials in the exercise period.

7.3.4 Cognitive performances

7.3.4.1 Choice reaction time

Only the LGI Ex 1 RT was normally distributed. The Friedman ANOVA (two trials x three exercise sessions) found a significant difference in the RT ($\chi^2(5, n = 16) = 21.8, p = 0.001$). The Wilcoxon signed-rank test found the longest RT at the first exercise session when compared to at the second and third sessions in both trials (Figure 7.5). Spearman's correlation only found a significant negative association between the number of the correct response and the RT during the LGI session 2 ($r = -0.53, p = 0.04$). No significant difference was found in the number of correct response ($\chi^2(5, n = 16) = 2.1, p = 0.83$).

There was a significant difference in the RT ($\chi^2(5, n = 16) = 30.1, p < 0.001$) between the first and second order trials. The Wilcoxon signed-rank test found that the RT was significantly longer than at the second visit (537 ± 85 vs. 492 ± 73 ms, $Z = -3.3$, corrected $p = 0.003$). The Friedman ANOVA did not detect a significant difference in the number of correct responses between the first and second order trials.

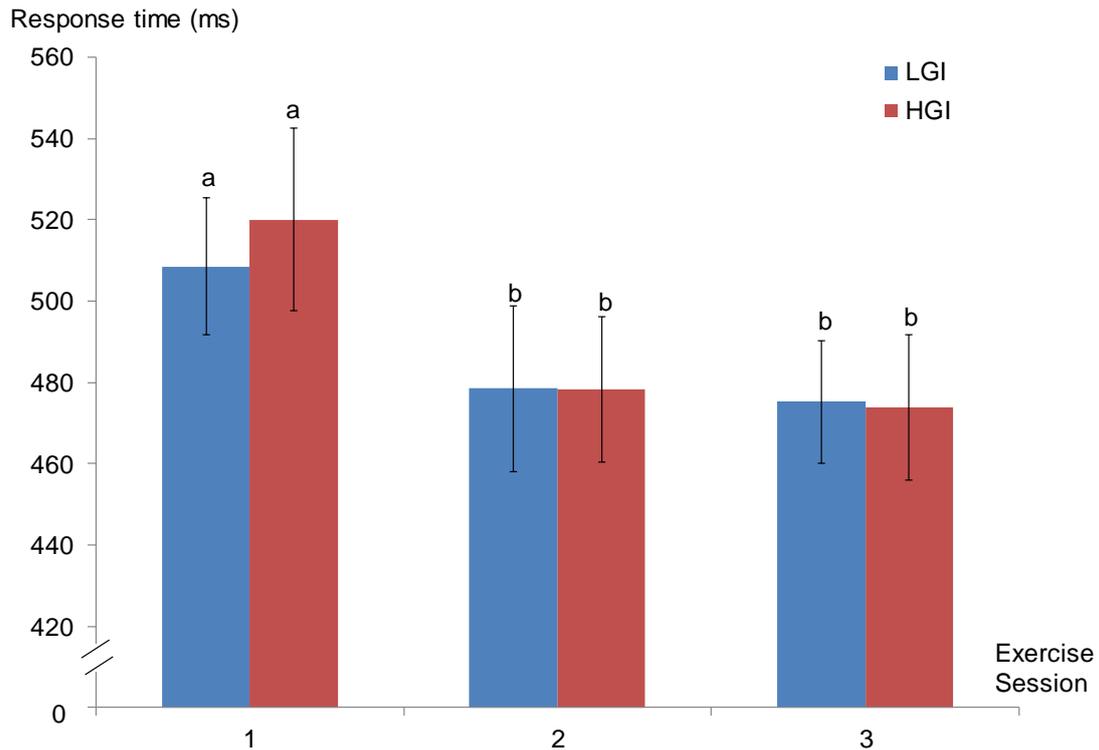


Figure 7.5 Response time in the Choice Reaction Time between trials

Data presented as mean \pm SD ($n = 16$). HGI, high glycaemic index; LGI, low glycaemic index; ^{a,b} significantly different when sharing different superscripts within trial (corrected $p < 0.035$).

7.3.4.2 Stroop colour word task

A significant trial \times time interaction was found in the SCWT interference time ($F(1.8, 27.4) = 3.6, p = 0.045$) (Figure 7.6). The post hoc t -test showed that LGI interference time (59 ± 73 ms) after the last exercise session was lower than that after the corresponding first exercise session (145 ± 130 ms, $t(15) = -2.5$, corrected $p = 0.078$); and that in the HGI trial (113 ± 77 ms, $t(15) = -2.3$, corrected $p = 0.11$) without reaching significance after the Bonferroni correction. No significant trial \times time effects were found in the interference and facilitation scores for response accuracy.

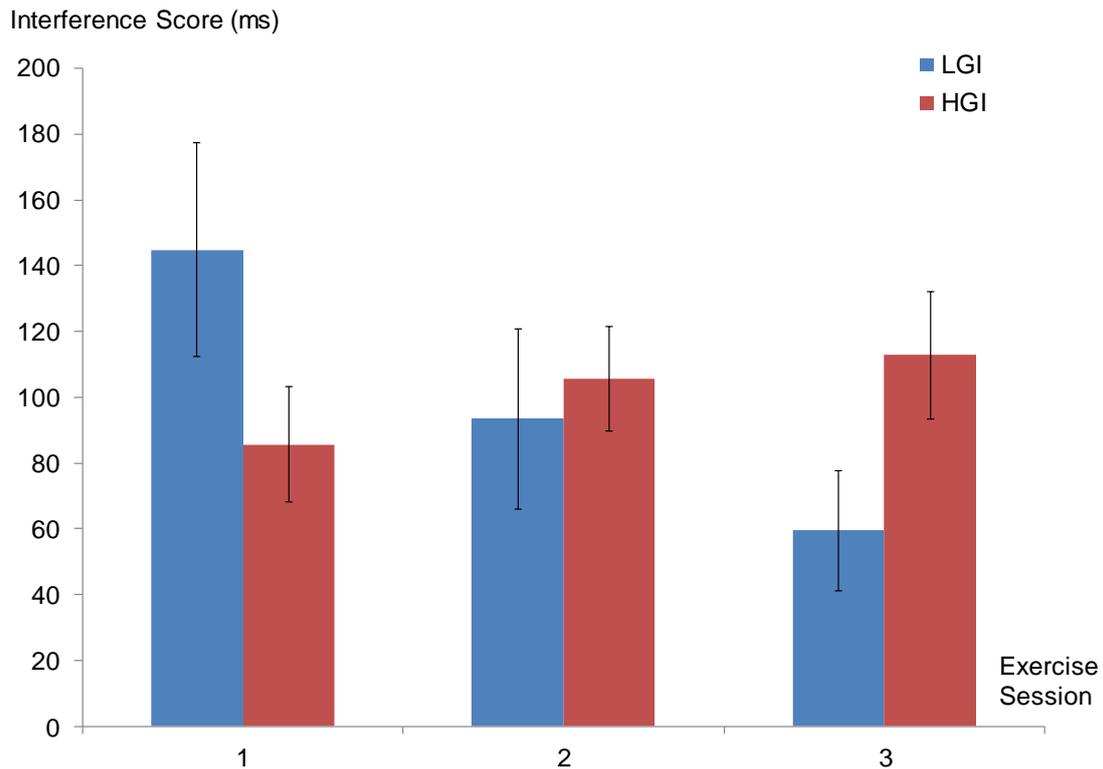


Figure 7.6 Interference Score in the Stroop Colour Word Task between trials

Data presented as mean \pm SD (n = 16). HGI, high glycaemic index; LGI, low glycaemic index.

The ANOVA did not detect any effects of the trial order x time interaction, order and time in the SCWT facilitation speed, the interference and the facilitation scores for accuracy between the first and second visits.

7.3.4.3 Profile of Mood States

A significant trial x time effect was found in the tension score at the pre-exercise period ($F(1, 15) = 4.6, p = 0.05$); however the *post-hoc t*-test did not detect the significant differences between and within trials after the Bonferroni correction.

Significant time effects were found in the scores of confusion ($F(1.5, 20.5) = 5.2, p = 0.023$) and fatigue ($F(1, 1.5) = 5.7, p = 0.03$) during the pre-exercise period;

and depression ($F(1, 15) = 7.1, p = 0.018$), fatigue ($F(1, 15) = 10.1, p = 0.006$) and tension ($F(1, 15) = 9.4, p = 0.008$) during the period between 60-min pp and exercise. The LGI fatigue (4.6 ± 3.7) and the tension (1.3 ± 1.5) scores after the last exercise session were significantly different to the corresponding scores at 60-min pp (fatigue: $1.7 \pm 1.9, t(15) = 4.2$, corrected $p = 0.002$; tension: $3.1 \pm 2.6, t(15) = -3.8$, corrected $p = 0.004$). No such differences were found in the fatigue ($p = 0.07$) and the tension ($p = 0.4$) scores in the HGI trial. The HGI fasting tension score was found to be significantly higher than that at 60-min pp (2.9 ± 2.4 vs. $1.7 \pm 2.0, t(31) = 3.4$, corrected $p = 0.008$) (Table 7.5).

Table 7.5 The Profile of the Mood States between trials

	LGI			HGI		
	Fasting	Before exercise	After exercise	Fasting	Before exercise	After exercise
Confusion	1.5 ± 1.5	1.0 ± 1.6	1.1 ± 1.4	1.8 ± 2.1	1.0 ± 1.5	1.4 ± 1.8
Depression	0.3 ± 0.6	0.3 ± 0.6	0	1.3 ± 2.8	0.8 ± 1.6	0.1 ± 0.3
Fatigue	2.4 ± 2.3	$1.7 \pm 1.9^*$	$4.6 \pm 3.7^*$	3.5 ± 3.8	2.2 ± 1.8	4.0 ± 4.1
Tension	3 ± 2.2	$3.1 \pm 2.6^*$	$1.3 \pm 1.5^*$	$2.9 \pm 2.4^*$	$1.7 \pm 2^*$	1.3 ± 1.5

Data presented as mean \pm SD ($n = 16$). HGI, high glycaemic index; LGI, low glycaemic index; * significant difference when sharing the same superscript at the same type of score within trial (corrected $p < 0.008$).

No significant trial order \times time effects were found during the pre-exercise period; and the exercise period in all POMS variables between the first and second visits.

7.3.5 Relationships between biochemical variables and cognitive performances

Glucose

When all exercise sessions were included, Pearson's correlation found a significant negative correlation was found in the CRT in the HGI trial between the post exercise session [Glucose] and the number of the correct response ($r = -0.36$, $p = 0.012$) (Figure 7.7a); and the number of the CRT adjusted correct response ($r = -0.43$, $p = 0.002$) (Figure 7.7b).

Significant associations were found between the post exercise session [Glucose] and the number of correct responses ($r = -0.64$, $p = 0.008$); and the number of the adjusted correct responses ($r = -0.59$, $p = 0.016$) at the first exercise session of the HGI trial. The LGI trial did not find any associations between the CRT performances and the post exercise [Glucose].

No significant correlations were found in both trials between the post exercise [Glucose] and the SCWT interference scores.

Lactate

When all exercise sessions were included in each trial, the post exercise [Lactate] were not normally distributed in both trials. Spearman's correlation found a significant negative association between the LGI post exercise [Lactate] and the CRT RT ($r = -0.31$, $p = 0.034$). Nevertheless, no significant correlations were found when the exercise sessions were separated in the LGI trial.

Spearman's correlation found significant negative correlations between the HGI post exercise [Lactate] and the number of the correct responses ($r = -0.33$, $p = 0.022$); and the adjusted correct responses ($r = -0.37$, $p = 0.01$) in the CRT (Table 7.6). Of which, the significance was found at the HGI first session with the number of correct response ($r = -0.56$, $p = 0.02$).

Table 7.6 Correlations between post exercise biochemical variables and Choice Reaction Time performances

	Trial	Response time (ms)	Number of the accuracy	Number of the adjusted accuracy
[Glucose] (mmol·L ⁻¹)	LGI	$r = -0.06$ $p = \text{n.s.}$	$r = 0.13$ $p = \text{n.s.}$	$r = -0.03$ $p = \text{n.s.}$
	HGI	$r = -0.005$ $p = \text{n.s.}$	$r = -0.36$ $p = 0.012$	$r = -0.43$ $p = 0.002$
[Lactate]* (mmol·L ⁻¹)	LGI	$r = -0.31$ $p = 0.034$	$r = 0.15$ $p = \text{n.s.}$	$r = -0.14$ $p = \text{n.s.}$
	HGI	$r = -0.09$ $p = \text{n.s.}$	$r = -0.33$ $p = 0.02$	$r = -0.37$ $p = 0.01$

n = 48. HGI, high glycaemic index; LGI, low glycaemic index; * Spearman's correlation

The post exercise [Lactate] did not predict the SCWT interference score in response time and accuracy in both trials.

7.3.6 Substrates oxidation and intakes

No significant differences were found between the LGI and HGI trials in the percentage of the $\dot{V}O_2$ max (65.3 ± 6.6 vs. $64.5 \pm 6.1\%$, $t(15) = 0.89$, $p = 0.39$) and the $\dot{V}O_2$ (2.48 ± 0.32 vs. 2.45 ± 0.33 L·min⁻¹, $t(15) = 1.2$, $p = 0.25$). No significant effects of trial x time interaction and trial were found in the RER, the oxidations of CHO and fat. Significant time effects were found in the oxidations of CHO ($F(1.6, 23.6) = 42.3$, $p < 0.001$) and fat ($F(1.7, 26) = 52$, $p < 0.001$); and in RER ($F(1.6, 24.1) = 88.9$, $p < 0.001$) across exercise sessions (Figure 7.8).

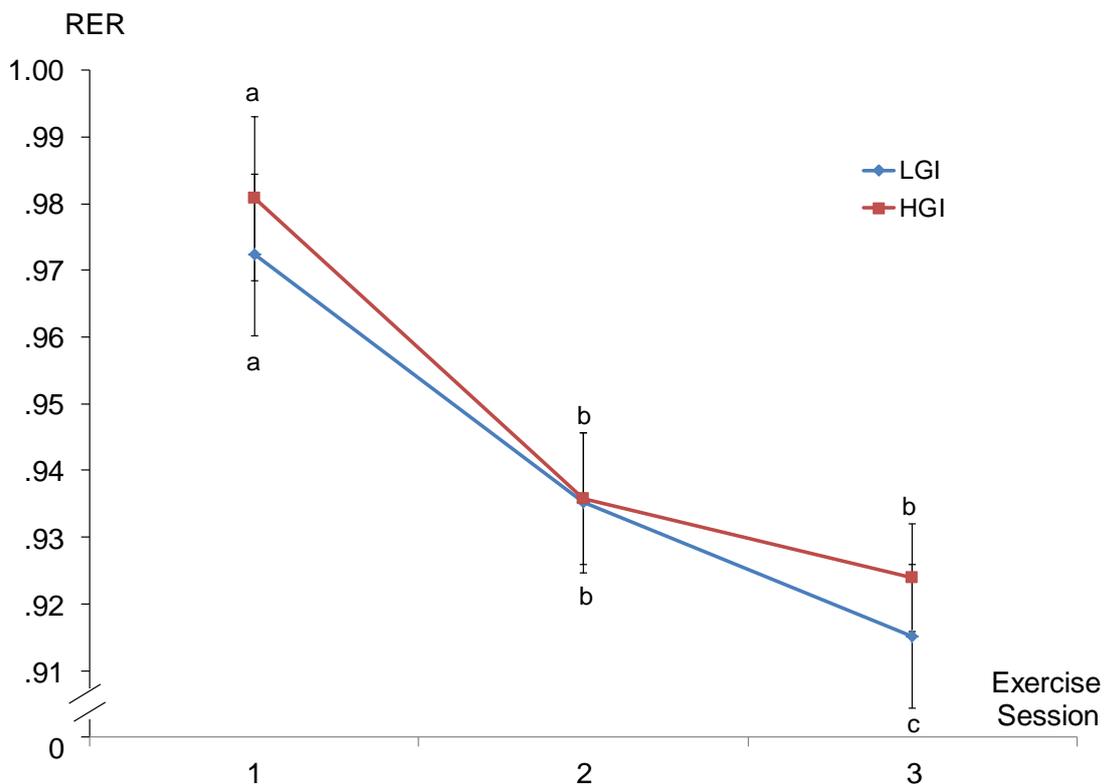


Figure 7.8 Respiratory exchange ratio (RER) during exercises

Data presented as mean \pm SD ($n = 16$). HGI, high glycaemic index; LGI, low glycaemic index; ^{a,b} significant difference when sharing the different superscripts within trial (corrected $p < 0.003$).

The amount of CHO oxidized was reduced as exercise sessions progressed (corrected $p < 0.003$), i.e. the amount during the subsequent exercise session was lower than those during the preceding rounds (Figure 7.9). The amount of CHO oxidation was significantly higher than the CHO intake in the LGI (108 ± 18 vs. 92 ± 12 g, $t(15) = 3.0$, corrected $p = 0.016$) and HGI trials (110 ± 20 vs. 91 ± 13 g, $t(15) = 3.1$, corrected $p = 0.016$); whereas no significant difference between trials was found (Appendix W).

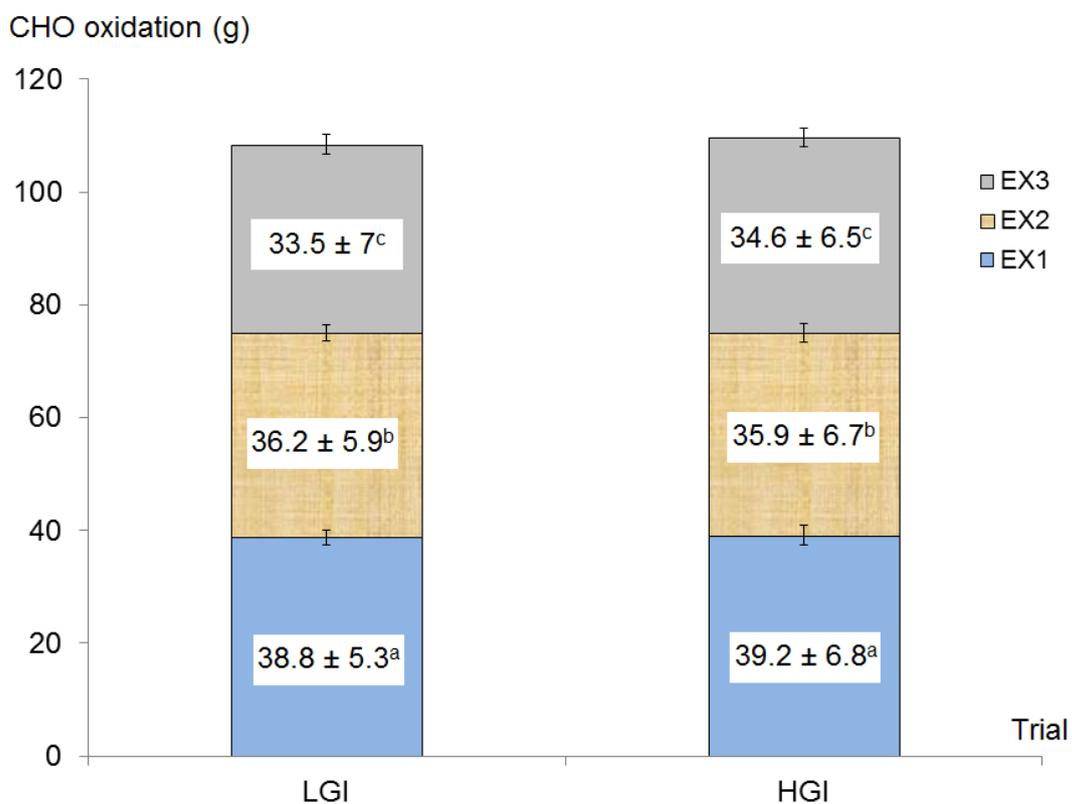


Figure 7.9 Oxidation of carbohydrate (CHO) during exercises between trials

Data presented as mean \pm SD ($n = 16$). Ex, exercise session; HGI, high glycaemic index; LGI, low glycaemic index; ^{a,b,c} significantly different when sharing the different superscripts within trial (corrected $p < 0.02$).

The amount of fat being oxidized was increased as exercise sessions progressed between the first and second sessions in both trials (corrected $p <$

0.003). The LGI oxidation of fat remained significantly higher during the third than second sessions (corrected $p < 0.003$) but the difference did not reach a significant level in the HGI after the Bonferroni correction (corrected $p = 0.13$) (Figure 7.10). No significant difference was found between the amount of fat being consumed at breakfast and being oxidized during exercise in both trials (Appendix W).

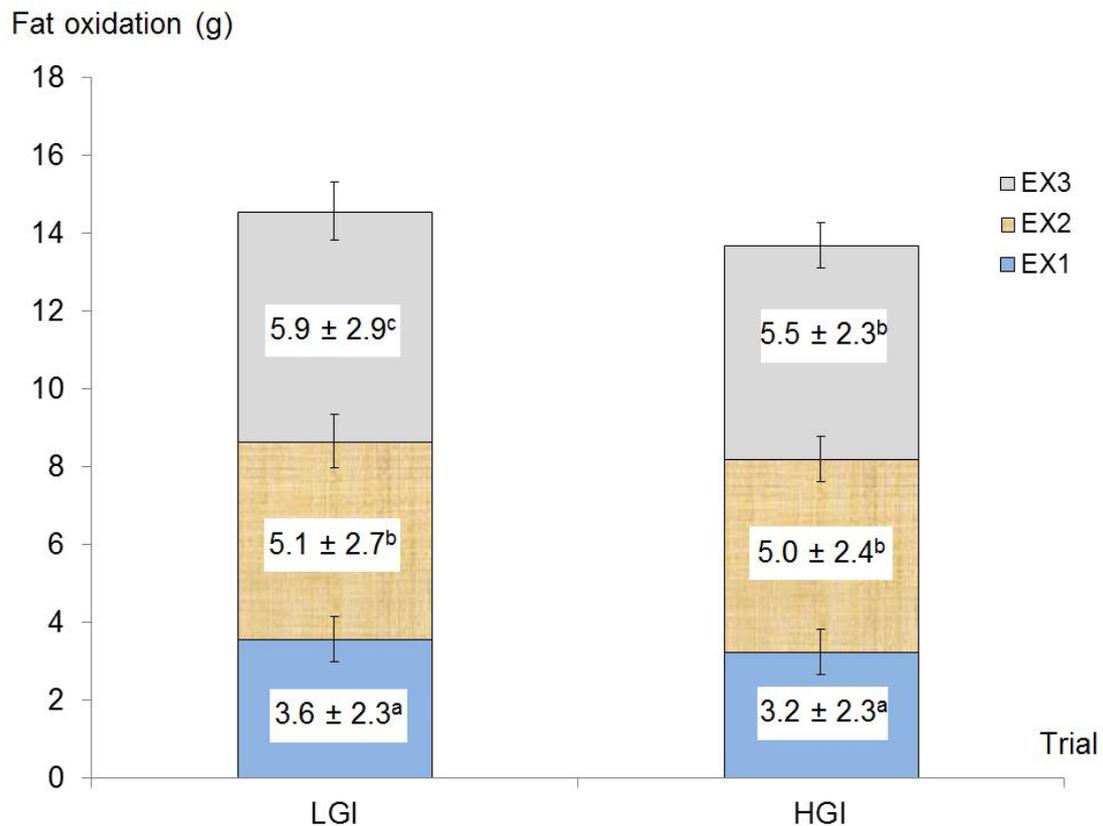


Figure 7.10 Oxidation of fat during exercises between trials

Data presented as mean \pm SD ($n = 16$). Ex, exercise session; HGI, high glycaemic index; LGI, low glycaemic index; ^{a,b,c} significantly different when sharing the different superscripts within trial (corrected $p < 0.003$).

There was no significant difference between the LGI and HGI trials in the breakfast EI (574 ± 77 vs. 572 ± 81 kcal, $t(15) = 0.5$, $p = 0.6$) and the EE during exercise (582 ± 72 vs. 579 ± 79 kcal, $t(15) = 0.5$, $p = 0.6$). No significant differences were found between the breakfast EI and the EE during exercise in

the LGI (574 ± 77 vs. 582 ± 72 kcal, $t(15) = -0.3$, $p = 0.8$) and HGI (572 ± 81 vs. 579 ± 79 kcal, $t(15) = -0.2$, $p = 0.8$) trials (Appendix W).

One participant did not return the pre-trial food record; thus the number of completed food records was 15. There was no difference between the LGI and HGI pre-trial EI ($2,674 \pm 486$ vs. $2,631 \pm 626$ kcal, $t(14) = 0.36$, $p = 0.72$), intakes of CHO (342 ± 71 vs. 344 ± 86 g, $t(14) = -0.12$, $p = 0.91$), protein (113 ± 32 vs. 112 ± 44 g, $t(14) = 0.19$, $p = 0.85$) and fat (100 ± 34 vs. 98 ± 37 g, $t(14) = 0.26$, $p = 0.8$).

7.3.7 Physical responses

No effects of trial and trial x time interaction in the RPE and in the HR were found between trials. A significant time effect was found in the RPE ($F(1.8, 27.5) = 36.7$, $p < 0.001$), the HR ($F(1.4, 15) = 59.6$, $p < 0.001$), and the HR relative to the HR at the $\dot{V}O_{2\max}$ ($F(1.4, 13.6) = 48.6$, $p < 0.001$) that those variables at the subsequent sessions were higher than the preceding sessions (corrected $p < 0.05$) (Table 7.7).

Table 7.7 The physical responses during exercise between trials

	LGI			HGI		
	Ex 1	Ex 2	Ex 3	Ex 1	Ex 2	Ex 3
RPE	13 ± 2 ^a	14 ± 2 ^b	15 ± 2 ^c	13 ± 2 ^a	14 ± 2 ^b	15 ± 2 ^c
HR*	144 ± 14 ^a	151 ± 12 ^b	152 ± 12 ^c	142 ± 12 ^a	149 ± 11 ^b	152 ± 11 ^c
HR % to HR at VO ₂ max (%)**	75.6 ± 4.9 ^a	78.9 ± 4.1 ^b	79.9 ± 4 ^c	74.2 ± 4.8 ^a	77.9 ± 4.3 ^b	80 ± 3.8 ^c

Data presented mean ± SD (N = 16, except *N = 12, **N = 11). Ex, exercise session; HGI, high glycaemic index; HR, heart rate, LGI, low glycaemic index; RPE, rate of perceived exertion; ^{a,b,c} significant difference when sharing the different superscripts in the same row within the trial (corrected $p < 0.05$)

7.4 Discussion

The current study found no significant differences in the substrates oxidation during intermittent running between the LGI and HGI trials providing 1.2 g CHO kg⁻¹ BM 60 minutes taken prior to the exercise. Nevertheless, the study found that the selective and sustained attentional performances were improved when performing after moderate intensity intermittent running following the LGI breakfast.

7.4.1 Substrates oxidation

A rapid and higher increase in postprandial [Glucose] following HGI CHO was expected to lead to higher rates of CHO oxidation during exercise. Several previous studies found significant differences in substrate oxidation during exercise between LGI and HGI pre-exercise meals (Moore, Midgley, Thurlow, Thomas, & Mc Naughton, 2010; Stevenson, Williams, Mash, Phillips, & Nute, 2006; Wee, Williams, Tsintzas, & Boobis, 2005; Wong et al., 2008; Wu, Nicholas, Williams, Took, & Hardy, 2003), but some did not (Febbraio & Stewart,

1996; Hulton et al., 2012; Little et al., 2010). The discrepancy may be due to the differences in study designs such as the mode of exercise like intermittent exercise (Hulton et al., 2012; Little et al., 2010) and endurance exercise (Moore et al., 2010; Stevenson et al., 2006; Wee et al., 2005; Wong et al., 2008; Wu et al., 2003); and the CHO dose that ranged as low as 1.0 g CHO kg⁻¹ BM (Febbraio & Stewart, 1996; Fielding et al., 1987) to as high as 2.5 g CHO kg⁻¹ BM (Wee et al., 2005). No differences in substrates oxidation were found with the dose of 1.5 g CHO kg⁻¹ BM or below (Febbraio & Stewart, 1996; Fielding et al., 1987; Little et al., 2010) when compared to the dose above 1.5 g CHO kg⁻¹ BM (Stevenson et al., 2006; Wong et al., 2008; Wu et al., 2003). Wee et al. (2005) speculated that low CHO dose might not be sufficient to elicit the shift in substrate oxidation (Febbraio & Stewart, 1996; Fielding et al., 1987). However, a recent study found a significant difference in the substrate oxidation between the pre-exercise LGI and HGI trials in which 1 g CHO kg⁻¹ BM was provided (Moore et al., 2010) with higher CHO oxidation after a pre-exercise LGI compared to the HGI meal during a 40-km time trial cycling which was contradictory to the previous significant findings (Stevenson et al., 2006; Thomas et al., 1991; Wong et al., 2008; Wu et al., 2003). A recent review also suggested a trend of an increase in CHO oxidation during exercise following HGI CHO intake whereas no conclusion can be drawn for any GI effect on performance (Jeukendrup & Killer, 2010).

In the study of DeMarco, Sucher, Cisar, and Butterfield (1999), the HGI, LGI and the placebo RER ranged between 0.92 and 0.94; 0.83 and 0.87; and 0.83 and 0.88 at the first 60-min cycling at 70% $\dot{V}O_2$ max, respectively. The LGI and the placebo RER illustrated the shifts of substrate oxidation from CHO towards

fat (DeMarco et al., 1999). The higher the RER indicated the higher the rate of CHO oxidation; whilst lower RER might be resulted from the pre-exercise LGI CHO intake or during the fasting status. The current LGI and the HGI RER ranged between 0.92 and 0.98; and between 0.92 and 0.97, respectively, without reaching an RER as low as at the LGI and placebo trials in the previous study (DeMarco et al., 1999). The current findings indicated that the exercise sessions relied mainly on CHO oxidation without significant trial difference. Another possible reason for the similar substrate oxidation rates between trials was the higher fructose content in the LGI breakfast. Sun, Wong, Huang, Chen, and Tsang (2011) found higher fat and lower CHO oxidation in the LGI trial, whereas both the LGI with fructose and the HGI providing 25% energy resulted in comparable substrate oxidation during brisk walking at 50% $\dot{V}O_{2max}$. Thus the current comparable substrate oxidation might be due to the presence of fructose in the LGI breakfast. The maximal rate of fat oxidation was reported to be at around 63% $\dot{V}O_{2max}$ corresponding to 73% HRmax (Achten, Gleeson, & Jeukendrup, 2002; Achten & Jeukendrup, 2004). The average percentage of $\dot{V}O_{2max}$ and the HRmax were 65% and 75.6–79.9% in the LGI and 74.2–80% in the HGI trials in the current study. However the highest running speed in the current protocol was equivalent to 120% $\dot{V}O_{2max}$. Here had another speculation that the exercise intensity of the current regimen might be too high to elicit significant difference in substrates oxidation.

Kelman, Maughan, and Williams (1975) found that the [Lactate] during short bouts of exercise at 65–70% $\dot{V}O_{2max}$ reached 1.5–2.5, 2.5–3.0 and 3.5–4.0 mmol·L⁻¹ following low, moderate and high CHO diets, respectively. Maughan et al. (1997) considered CHO contributing 65-75% of energy in a diet as a high

CHO diet. It is now known that judging as percentage of energy is poorly correlated to the actual amount of CHO consumed and the energy needed to support exercise events (Burke, Kiens, & Ivy, 2004). Consumption of 75 g CHO an hour prior to submaximal exercise at 62–72% $\dot{V}O_2$ max was considered to be a moderate amount (Jeukendrup & Killer, 2010). The muscle glycogen synthetic rate was found to be similar between an intake of 1.2 g CHO kg⁻¹ BM with protein and 1.5 g CHO kg⁻¹ BM (Burke, Hawley, Wong, & Jeukendrup, 2011). The current breakfasts containing 1.2 g CHO kg⁻¹ BM (92 ± 12 g, 63% energy) and 13% protein for energy was expected to enhance the glycogen storage as efficient as following the consumption of 1.5 g CHO kg⁻¹ BM (Burke et al., 2011). However, the glucose might be sequestered primarily in the liver after an overnight fast as the current amount of the CHO oxidized during exercise was found to be significantly greater than the CHO intake. Nevertheless, the RER maintained high in the last exercise session (> 0.9) which ruled out the body used up all exogenous CHO and running under the condition similar as at a fasting condition at the last exercise session.

7.4.2 Cognitive performances

A bout of submaximal exercise has been demonstrated to improve cognitive performance. Very often cognitive performance has been investigated during or immediately after an exercise protocol including aerobic and anaerobic exercise lasting less than 20 minutes (Tomprowski, 2003). Exercise induced arousal improved the cognitive efficiency in term of the speed of decision-making and mood but not response accuracy (Davranche & Audiffren, 2004; Tomprowski, 2003). The LGI and HGI breakfasts appeared to influence the performances in the CRT in different aspects. When all exercise sessions were included in each

trial, both [Glucose] and [Lactate] were negatively associated with the response accuracy in the HGI trial whilst the [Lactate] had a negative association with the response time in the LGI trial. Lactate might be a substitute fuel for brain during hypoglycaemic condition (Evans et al., 2004). Postprandial blood [Lactate] was significantly higher after consuming the current fructose containing the LGI than HGI breakfasts. Ingested fructose is rapidly phosphorylated in the liver by fructokinase to fructose-1-phosphate which then stimulate the activation of pyruvate kinase leading to the stimulation of the production of lactate, as well as pyruvate (Jentjens, Moseley, Waring, Harding, & Jeukendrup, 2004).

The SCWT interference effect reduced gradually in the LGI trial as exercise sessions progressed; whereas a reverse effect was found in the HGI trial that the interference time tended to increase progressively (Figure 7.6). It might therefore be speculated that the LGI breakfast had an advantage on the selective attentional performance when compared to the HGI breakfast. Both exercise and cognitive activities reduced the cerebral ratio of the oxygen / glucose uptake by brain activation (Dalsgaard, 2006). The increase in the cerebral lactate uptake was parallel with the arterial [Lactate]. When the blood [Lactate] increased with exercise, the cerebral uptake of lactate from blood increased even if the elevation in the arterial [Lactate] was as small as 2.5–3.5 mmol·L⁻¹. The amount of the cerebral lactate uptake could be similar to that of glucose at maximal exercise as the increased uptake of lactate was not accumulated within the brain. Those uptakes were therefore metabolised as an energy substrate for the brain (Dalsgaard, 2006; Dalsgaard & Secher, 2007).

The current average $\dot{V}O_2$ per exercise session was close to the $\dot{V}O_2$ at LT (32.8 ± 6.5 vs. $34.1 \pm 6.3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Moderate exercise or exercise closed to the LT was believed to optimise the cognitive functions (Chmura, Nazar, & Kaciuba-Uscilko, 1994). A U shape effect suggested that the shortest RT was achieved at moderate levels of arousal and the RT was longer at rest or during too tense activities. Kashihara and Nakahara (2005) found an improvement of the RT for the first eight minutes of a 20-min CRT task after a 10-min exercise intensity at LT without the difference in the response accuracy when compared to the control condition. Lambourne and Tomporowski (2010) also suggested that cognitive performance was impaired in the early stage of exercise; whereas the RT, the memory storage and retrieval were improved following exercise. Nevertheless, the authors also mentioned that a treadmill run impaired cognitive performance during the run but achieved a small improvement following exercise. The current CRT performed during the run found neither an improvement nor a decrement in performances whereas the SCWT performed five minutes after each running session showed a significant trial x time effect in the interference score in RT.

The study aimed to investigate the GI effect across times on the exercise and the cognitive performances. The current [Glucose] following all exercise sessions in both trials reached below $3.9 \text{ mmol}\cdot\text{L}^{-1}$ ($3.6\text{-}3.8 \text{ mmol}\cdot\text{L}^{-1}$) which is a general cut-off value of hypoglycaemia (American Diabetes Association, 2007). The plasma [Glucose] of one participant in the current study reached $2.9 \text{ mmol}\cdot\text{L}^{-1}$ immediately after the first session of exercise but did not report any discomforts. The occurrence and magnitude of rebound hypoglycaemia during exercise is individualised and unrelated to the amount of CHO taken

(Jeukendrup & Killer, 2010). The authors explained that it remained unclear the factors contributing to the susceptibility to the development of hypoglycaemia in individuals (Jeukendrup & Killer, 2010). The authors also highlighted that some participants had very low plasma [Glucose] but did not report any hypoglycaemic symptoms. It was therefore speculated that any hypoglycaemia effect, if present in the current study, did not contribute a detrimental effect on running and cognition as the post exercise [Glucose] were comparable between trials.

7.4.3 Physical response

The current [Lactate] remained significantly higher than in the LGI before the onset of the exercise. The [Lactate] reached the second peak after the first exercise session in both trials. The Δ [Lactate] tended to be greater in the HGI than the LGI trial during the first (1.9 ± 1.15 vs. 1.4 ± 0.94 mmol·L⁻¹, $Z = -2.3$, corrected $p = 0.06$) and the second exercise sessions (1.5 ± 0.97 vs. 1.1 ± 0.98 mmol·L⁻¹, $t(15) = 2.5$, corrected $p = 0.069$). The [Lactate] then declined slightly as exercise trials progressed but maintained the decline within 1.0 mmol·L⁻¹ in both trials. The current exercise protocol (3 x 16 minutes) was structured with four different speeds which did not allow participants to slow down unless upon request due to volitional exhaustion. Significant but small progressive increases in the HR and the RPE indicated that the level of fatigue physiologically as well as psychologically gradually increased. Previously high [Lactate] was often considered as an indicator of fatigue. However, Maughan et al. (1997) suggested that the accumulated H⁺ ions might be more likely to induce fatigue than the [Lactate]. The current gradual reduction of the [Lactate] appeared to be incoherent with the gradual increase in the RPE and the HR as the exercise

sessions progressed leading to the speculation of the use of lactate as a substrate for energy.

7.4.4 Ecological validity

Studies have commonly employed an exercise intensity equivalent to 70–75% $\dot{V}O_2$ max for investigating the GI effect on endurance exercise performance (Donaldson et al., 2010; O'Reilly et al., 2010) as exercise performance at these intensities are closely related to the pre-exercise muscle glycogen (Bergstrom, Hermansen, Hultman, & Saltin, 1967; Maughan et al., 1997). Nicholas et al. (2000) designed a controlled field test, the LIST, simulating the activity patterns of a 90-min soccer game which was physiologically and metabolically (RER: 0.92 ± 0.01 ; $\dot{V}O_2$: 2.86 ± 0.16 L min^{-1} ; CHO: 215 ± 13 g; fat: 49 ± 11 g) similar to those reported during soccer matches. McMorris, Sproule, Turner, and Hale (2011) advised that exercising at a level $> 80\%$ $\dot{V}O_2$ max increase the response time and error. It was therefore speculated that the results of the response time and accuracy would not be confounded by the current exercise mode. The RER (0.92–0.98) and CHO oxidation (108–110g) in the current study was comparable to the LIST protocol (Nicholas et al., 2000). Although the current HR, the $\dot{V}O_2$; the [Glucose] and the fat oxidation appeared to be lower than in the LIST (142–159 vs. 166–172 beats min^{-1} ; 2.46 vs. 2.86 L min^{-1} ; 3.6–3.8 vs. 6.3 $\text{mmol}\cdot\text{L}^{-1}$; and 49 g in 90 minutes vs. 14–15 g in 48 minutes), participants in the LIST study had BM loss nearly to 3% which could lead to HR acceleration (Sawka et al., 2007). Submaximal aerobic exercise longer than 60 minutes is associated with increasing psychological stress and compromised cognitive performance especially under circumstances of dehydration and glycogen depletion (Tomprowski, 2003). No changes in the BM and the plasma volume

in both trials in the current study ruled out the potential influence of dehydration on cognitive performances. Nicholas et al. (2000) also reported low [Glucose] of $3.8 \text{ mmol}\cdot\text{L}^{-1}$ at the end of a national level soccer match elsewhere and the $\dot{V}\text{O}_2$ of $2.5\text{--}3.5 \text{ L}\cdot\text{min}^{-1}$ could be reached during matches. The exercise protocol in the current study tried to follow the LIST. However, the treadmill employed here restricted the quickest speed change programmed as 30 seconds which was a limitation of setting the protocol same as the LIST. The current physiological and metabolic outcomes appeared to be comparable to those in the LIST. The protocol was therefore successful in mimicking metabolic and physiological responses to intermittent moderate-to-high intensity exercise like the LIST.

7.4.5 Strengths and limitations

It has been known from the $\dot{V}\text{O}_2$ kinetics that it is not reliable to determine moderate-high intensity using the percentage of the $\dot{V}\text{O}_2\text{max}$ (Xu & Rhodes, 1999). The current study applied the running velocity at delta 50% but the concept of delta 50% was not applied in previous literatures.

In line with the previous studies, all meals provided in the current study matched the contents of macronutrient and dietary fibre, as well as energy density between trials. Furthermore, the significant trial x time interaction in the postprandial glucose profiles, the peak [Glucose] and the glucose IAUC also implied a success in using the calculated method of Wolever and Jenkins (1986) to set the test breakfasts to have significant difference in postprandial glucose response. As mentioned previously, participants maintained at the status of

euhydration reflected from the maintenance of BM and the plasma volume at the end of the exercise period; as well as the osmolality maintained at the normal range which minimized the confounding effect of dehydration on cognitive performances.

Many sports such as rugby and soccer require the players to perform fast decision making, maintain sustained attention and optimal selective attentional performance whilst with motions with various intensities during games. Although memory performances such as verbal learning, immediate and delayed recall were suggested to be influenced more likely by postprandial glucose response than non-mnemonic tasks (Messier, 2004), memory performance was a less detrimental element in success in skill sports. The current thesis assessed attention and vigilance which are more relevant to the skill sports in practice.

There are some limitations in the current study. There was a trial order effect irrespective of condition on CRT response time during the first exercise session; and between the first and second sessions in the first trial. Although participants were asked to practise until they felt confident in performing the cognitive tasks at the familiarisation trial, the CRT was performed at rest during familiarisation. The running protocol was explained verbally again with graphic presentation given prior to the treadmill run. Additional tasks performed prior to breakfast might provide a last opportunity for participants to practise. Moreover, the results at the fasting condition could be the baseline for investigating the changing direction of the cognitive performance influenced by the GI breakfasts.

Evans et al. (2004) suggested that different regions of the brain utilize different fuel sources for energy to support the cognitive functions differentially, particularly at the times of low glucose supply. The current findings on the cognitive performance cannot be simply explained by the peripheral plasma [Glucose]. Other substrates, besides glucose and lactate, or hormones influencing the brain substrate uptake might affect the cognitive performances centrally..

7.5 Conclusion

The current study suggests that a LGI breakfast providing 1.2 g CHO kg⁻¹ BM taken one hour prior to three 16-min intermittent running sessions reduces the SCWT interference time at the last exercise session which indicates an improvement in the selective attention. The negative correlations between the post exercise [Glucose] and the CRT performances require further investigation. Recreationally active males can benefit from a LGI breakfast during the latter periods of moderate-to high intermittent intensity exercise on selective attention compared to a HGI breakfast. The current CHO dose might not reveal or trigger differential shifts in substrate oxidation between trials during subsequent exercise. Therefore, the findings should not be generalized without further empirical evidence as differences in the measurement time, the type of cognitive task selected, and the type, the intensity and the duration of exercise performed might result in differences in cognitive and exercise outcomes.

CHAPTER 8 GENERAL DISCUSSION

This chapter commences with a brief overview of the main findings and their significance within the studies. Methodological considerations and limitations with respect to the recruitment of participants and the study designs are also discussed. Finally, potential applications and implications for practice; and recommendations for further investigation are suggested.

8.1 Summary of main findings

The first three repeated-measure counter-balanced studies investigated the effects of GI, LGI vs. HGI, on appetite sensation. In the first study, 12 recreationally active female participants (28.2 ± 8.0 years) consumed the test breakfasts, (LGI = 42.5; HGI = 73.5), in the laboratory and then left the laboratory one hour post breakfast with their own choice of meals afterwards. The study found a greater appetite suppression following the HGI breakfast lasting for eight postprandial hours. No significant difference was found in the total EI on the trial day between trials (Table 8.1).

Manipulation of the GI of one meal in a day in study one showed significant appetite suppression, although the total EI following LGI and HGI breakfast was comparable. The appetite suppression induced by a single HGI meal a day might not therefore be sufficient to change the total EI (Pal et al., 2008). Therefore study two extended the investigation of the postprandial appetite sensation and the energy balance by providing additional meals at free living settings for the trial day. Fourteen recreationally active male participants (34.5

± 8.9 years) consumed the test breakfast at the laboratory and then consumed the test lunch, dinner and snack in a free living environment for the rest of the trial day. In line with the findings of study one, HGI meals (GI = 76.2) provided greater appetite suppressive effects over the trial day when compared to the LGI meals (GI = 39.6). Although the LGI lunch EI was significantly lower than the HGI lunch EI, the LGI and HGI total EI were still comparable due to the energy compensation from a higher LGI snack EI (Table 8.1).

The measurement of capillary blood [Glucose] in study three verified the significant difference in the GI values between the LGI (GI = 42.2) and the HGI (GI = 72.4) breakfasts resulting from a significant higher peak [Glucose] and greater glucose IAUC following the HGI than LGI breakfasts. The study did not find significant differences either in appetite sensation or in the *ad libitum* lunch intake between trials in 16 recreationally active males (24.4 ± 3.6 years). Significant positive correlations were observed between the lunch EI kg^{-1} FFM and the pre-lunch appetite score in both trials, but not with the pre-lunch [Glucose]. Additionally, study three investigated the effects of GI on the vigilance, selective and sustained attention by RIPT and SCWT in a sitting position. The significant trial x time effect in the SCWT interference time showed that the LGI breakfast maintained the attentional performance up to 90 minutes post breakfast; whilst the HGI breakfast had no effect on the interference time. Despite the lack of significant trial x time effects in the RT and the response accuracy in the RIPT, the high pre-task 1 [Glucose] as well as the low pre-task 3 [Glucose] following HGI breakfast did not favour the RT and the response accuracy at 15 minutes post breakfast; and the response accuracy at 105 minutes post breakfast. A favourable performance in RT

following LGI breakfast was implicated from the positive correlation with the LGI pre-task 2 [Glucose] dropping to the fasting level at 60 minutes post breakfast (Table 8.1).

Study four extended to investigate the effects of GI on cognitive performances during exercise and post exercise. Sixteen recreationally active males (27.8 ± 7.7 years) started the first session of a 16-min intermittent running 60 minutes post breakfast. The LGI breakfast (GI = 42) resulted in a reduction in the SCWT interference time across the exercise sessions compared to the HGI breakfast (GI = 72.8). No significant differences were found in the CRT performances between trials. The test breakfasts providing $1.2 \text{ g CHO kg}^{-1} \text{ BM}$ could not maintain the post-exercise [Glucose] above the fasting levels. The LGI breakfast resulted in comparable rates of CHO and fat oxidation during three intermittent running sessions with an average intensity of 65% $\dot{V}O_{2\text{max}}$ to the HGI trial (Table 8.1).

Table 8.1 Summary of the main findings of the effects of GI on postprandial appetite, cognitive performances and metabolic responses

Study	Participants	Design*	Measures	Main outcomes
One	n = 12 F 28.2 ± 8.0 yr, 62.4 ± 11.2 kg, 22.5 ± 3.2 kg·m ⁻²	LGI (42.5) & HGI (73.5) breakfast: as habitual EI	AS VAS: Pre- & post-breakfast; 10-min pp x 60 min; then hourly; post breakfast PS VAS EI: Food record	1-hr AUC: LGI > HGI (2,568 ± 1,027 vs. 2,198 ± 821 mm·min, <i>p</i> = 0.025). n.s. in total EI & EE; EI: LGI trial day > LGI pre-trial day (2,215 ± 576 vs. 1,748 ± 464 kcal, <i>p</i> = 0.004, <i>n</i> = 10)
Two	n = 14 M 34.5 ± 8.9 yr, 71.9 ± 10.6 kg, 22.8 ± 2.1 kg·m ⁻²	LGI (39.6) & HGI (76.2) meals: breakfast as habitual EI; free-living lunch & dinner: 30% of total EI; snack EI: total EI – main meal EI	AS VAS: Pre- & post-breakfast; 10-min pp x 60 min; then hourly; post meal PS VAS EB: Food record & accelerometry	1-hr & 12-hr AS AUC: LGI > HGI (3,758 ± 1,290 vs. 2,989 ± 1,390 mm·min, <i>p</i> = 0.027) & (41,244 ± 8,829 vs. 35,454 ± 9,730 mm·min, <i>p</i> = 0.009); n.s. in total EI & EE; Lunch EI: LGI < HGI (751 ± 168 vs. 835 ± 198 kcal, <i>p</i> < 0.033); snack EI : LGI > HGI (476 ± 368 vs. 404 ± 322 kcal, <i>p</i> = 0.035) Correlation: main meal EI & post meal AS (<i>r</i> = -0.49, <i>p</i> = 0.046)
Three	n = 16 M 24.4 ± 3.6 yr, 73.4 ± 11.2 kg, 22.9 ± 3.3 kg·m ⁻²	LGI (42.2) & HGI (72.4) breakfast: 1 g CHO kg ⁻¹ BM: <i>ad libitum</i> lunch at 165-min pp 25-min RIPT & SCWT started at 15-, 60- & 105-min pp	Capillary [Glucose] & [Lactate]: pre-breakfast, 0-, 15-, 45-, 60-, 90-, 105-, 135-, 165-min pp; & post lunch AS VAS: Pre- & post-breakfast & lunch; & post task ; post meal PS VAS POMS: pre-breakfast, 15- & 135-min pp	n.s. in AS AUC [Glucose]: trial x time effect (<i>p</i> < 0.001); 3-hr glucose IAUC: LGI < HGI (147 ± 56 vs. 189 ± 65 mmol·L ⁻¹ ·min, <i>p</i> = 0.012) RIPT: pre-task [Glucose] correlation: HGI T1 with adjusted response accuracy (<i>r</i> = -0.68, <i>p</i> = 0.003); & with RT (<i>r</i> = 0.58, <i>p</i> = 0.019); LGI task 2 with RT (<i>r</i> = 0.58, <i>p</i> = 0.019); HGI T3 with RT (<i>r</i> = -0.61, <i>p</i> = 0.01) SCWT interference time: trial x time effect (<i>p</i> = 0.039); LGI CHO oxidation was correlated with T3 (<i>r</i> = 0.57, <i>p</i> = 0.01) Correlation: pre-lunch AS and lunch EI kg ⁻¹ FFM (LGI & HGI, <i>p</i> < 0.05); n.s. in lunch EI
Four	n = 16 M 27.8 ± 7.7 yr, 76.7 ± 10.3 kg, 23.6 ± 3 kg·m ⁻² , 50.5 ± 8.3 mL·kg ⁻¹ ·min ⁻¹ $\dot{V}O_2$ max	LGI (42.0 ± 0.7) & HGI (72.8 ± 1.1) breakfast 1.2 g CHO kg ⁻¹ BM Ex start 60-min pp 8 x 2-min x 3-running sessions, CRT during ex & SCWT 5 min post ex	Capillary [Glucose] & [Lactate]; pre-breakfast, 15-, 30-, 45-, 60-, 90-, 120- & 150-min pp Venous [Insulin]: fasting, 30-, 60-, 90-, 120- & 150-min pp Post ex RPE, HR during ex: fasting, 60- & 150-min pp POMS; substrates oxidation	[Glucose]: trial x time effect at pre-ex period (<i>p</i> < 0.001); 1-hr glucose IAUC: LGI < HGI (86 ± 50 vs. 137 ± 49 mmol·L ⁻¹ ·min, <i>p</i> = 0.001); [Lactate]: trial x time effects at pre-ex (<i>p</i> = 0.002) & ex periods (<i>p</i> = 0.02) A difference in CRT RT (<i>p</i> = 0.001). HGI post ex 1 [Glucose] was correlated with the CRT response accuracy (<i>r</i> = -0.64, <i>p</i> = 0.008) A trial x time effect SCWT interference time (<i>p</i> = 0.045); LGI T1 > LGI T3 (145 ± 130 vs. 59 ± 73 ms, corrected <i>p</i> = 0.078) Gradual declines in RER & CHO oxidation; and increases in fat oxidation, RPE & HR (corrected <i>p</i> > 0.05) LGI & HGI CHO intake < usage (corrected <i>p</i> = 0.016); n.s. in EI & EE

Table 8.1 (Continued)

All were randomised, repeated-measure counter-balanced designs. Breakfast CHO:protein:fat = 60:15:25. Meals were matched with caloric density, macronutrient & fibre contents between trials. Mean \pm SD. AS, appetite score; IAUC, incremental area under the curve; BM, body mass; CHO, carbohydrate; CRT, choice reaction time; EB, energy balance; EE, energy expenditure; EI, energy intake; ex, exercise; FFM, fat free mass; [Glucose], glucose concentration; HGI, high glycaemic index; hr, hour; HR, heart rate; LGI, low glycaemic index; IAUC, incremental area under the curve; [Insulin], plasma insulin concentration; [Lactate], lactate concentration; min pp, minutes postprandially; n.s., non significance; PS, palatability score; POMS, profile of mood state; RER, respiratory exchange ratio; RIPT, rapid information processing task; RPE, rate of perceived exertion; RT, response time; SCWT, Stroop colour word task; T, task; VAS, visual analogue scale; $\dot{V}O_2$ max, maximal oxygen uptake; yr, year.

8.2 Significance of study findings

There are to date no published papers specifically addressing the effects of GI on appetite; and cognitive performance in a condition with low physical activity and under an intermittent running in recreationally active adults. The findings in the thesis are of value in providing practical suggestions for recreationally active adults to facilitate appetite control, cognitive and exercise optimum (Figure 8.1).

8.2.1 GI and appetite

High GI foods had greater appetite suppressing effects than LGI foods in recreationally active adults. The studies supported the glucostatic theory by Mayer (1955) that high blood postprandial [Glucose] was correlated with appetite suppression. The current findings were contradictory to the previous literatures with overweight and / or obese participants (Arumugam et al., 2008; Stevenson et al., 2009). In line with the study by Anderson et al. (2002), the rising [Glucose] in the early postprandial phase was accompanied by greater satiety. Furthermore, the studies in the thesis had a longer experimental duration. Even a single meal, a HGI breakfast, can suppress the appetite for a couple of hours postprandially. It might be easier to manipulate the first meal of the day to meet the busy schedules of recreationally active adults (Pal et al., 2008). In addition, the studies provided mixed meals which are more ecologically relevant to reflect the practical situation than the study by Anderson et al. (2002) which compared the effects of GI using fructose-glucose and glucose drink to represent LGI and HGI trials.

The frequency of eating during a day has been found to be increased in the last few decades (Popkin & Duffey, 2010). The mean time interval between eating occasions has been shortened from 4.5 to 3.5 hours between 1977 and 2006 in the U.S.. Frequent and regular consumption of HGI CHO found here suppressed appetite greater than the LGI CHO throughout a day. Although the HGI food consumption failed to reduce the EI or achieve negative energy balance over a single trial day, recreationally active adults might still benefit from distributing the HGI CHO intake over a day to achieve better appetite control.

8.2.2 GI and cognition

The SCWT interference time increased across the morning in study three; whilst the interference time reduced across the exercise sessions in study four in the LGI trials. Since the CHO dose and the physiological conditions between studies were different, it was not valid to compare those findings. Consistently the LGI SCWT interference time were not correlated with pre-task [Glucose] in both studies. The mechanisms of the effect of LGI breakfast on the Stroop performance remained unclear due to the potential effect of co-linearity by lactate. Lactate might be a substitute fuel for brain (Evans et al., 2004). How the effect of lactate on attentional performances needs further investigation. Recreationally active males participating in a sport with low physical activity level should therefore be aware that the selective attention cannot be maintained in the late post breakfast period following a LGI breakfast with fructose. Contrarily, recreationally active males can benefit on better selective attention from a LGI breakfast in the late intermittent exercise period when compared to a HGI breakfast.

Currently, it is too early to conclude that there are no cognitive effects of GI on the RIPT performance due to the lack of significant trial x time effects between the LGI and HGI trials. The significant unfavourable cognitive performances correlated with the pre-task [Glucose] in the RIPT; and the post exercise [Glucose] in the CRT in the HGI trials could still partially explain the variances. Recreationally active males should be aware of the potential effects of GI on vigilance and attention. Additionally, recreationally active males participating in a sport with low physical activity level may benefit overall from the LGI breakfast, if vigilance and selective attention needed to be maintained within 1.5 hours post breakfast.

Previous studies of the cognitive effects of glucose have mainly focused on memory improvement (Foster, Lidder, & Sunram, 1998; Greenwood, Kaplan, Hebblethwaite, & Jenkins, 2003; Meikle, Riby, & Stollery, 2004; Riby et al., 2006; M. A. Smith & Foster, 2008); whereas the SCWT, the RIPT and the CRT employed in the thesis are cognitive tasks which reflect vigilance, sustained and selective attention. Although a review suggested that memory performance was more likely to be influenced by glucose than non-mnemonic tasks (Messier, 2004), memory tasks such as verbal learning, immediate and delayed recall are less likely to be ecologically relevant to the skill sports for sports than attention and vigilance in practice.

McMorris and Hale (2012) suggested that either the RT or the response accuracy should be considered separately as a dependent variable due to the difference in the effect sizes between the RT and the response accuracy. The

SCWT interference scores were associated with the test breakfasts in terms of RT but not response accuracy which is in line with the previous literature that any changes in the cognitive efficiency are more likely to be observed in the RT rather than the response accuracy (Davranche & Audiffren, 2004; Tomporowski, 2003).

Recreationally active males participating in low or moderate-to-high physical activity sports need to possess good perceptual skills for feedback and adjust their responses continuously to achieve their goals. This thesis has provided some practical implications to sport players to ultimate the cognitive performances and metabolic responses via appropriate strategies on selecting an appropriate type of breakfast and the right timing of breakfast consumption prior to an exercise event with different physical activity levels.

Recreationally active adults looking for better appetite control can distribute the intake of HGI CHO over a day. Pre-meal appetite sensation is a more sensitive predictor of the intake than pre-meal [Glucose]. Recreationally active males performing sports with low physical requirements can benefit overall from the LGI breakfast when vigilance and selective attention needed to be maintained up to 90 minutes post breakfast. Recreationally active males performing sports requiring higher selective attention with moderate to high intermittent intensities can benefit from a LGI breakfast, particularly in the late exercise period. A LGI breakfast providing. A breakfast providing $1.2 \text{ g CHO kg}^{-1} \text{ BM}$ consumed an hour prior to intermittent running with an average intensity of $65\% \dot{V}O_2\text{max}$ could not maintain the post-exercise [Glucose] above the fasting levels,

regardless of GI values of the breakfast. Comparable rates of CHO and fat oxidation during three intermittent running sessions between following LGI and HGI breakfast implicated that recreationally active males may be more liberal in pre-exercise food choices if cognition is not of concern during exercise.

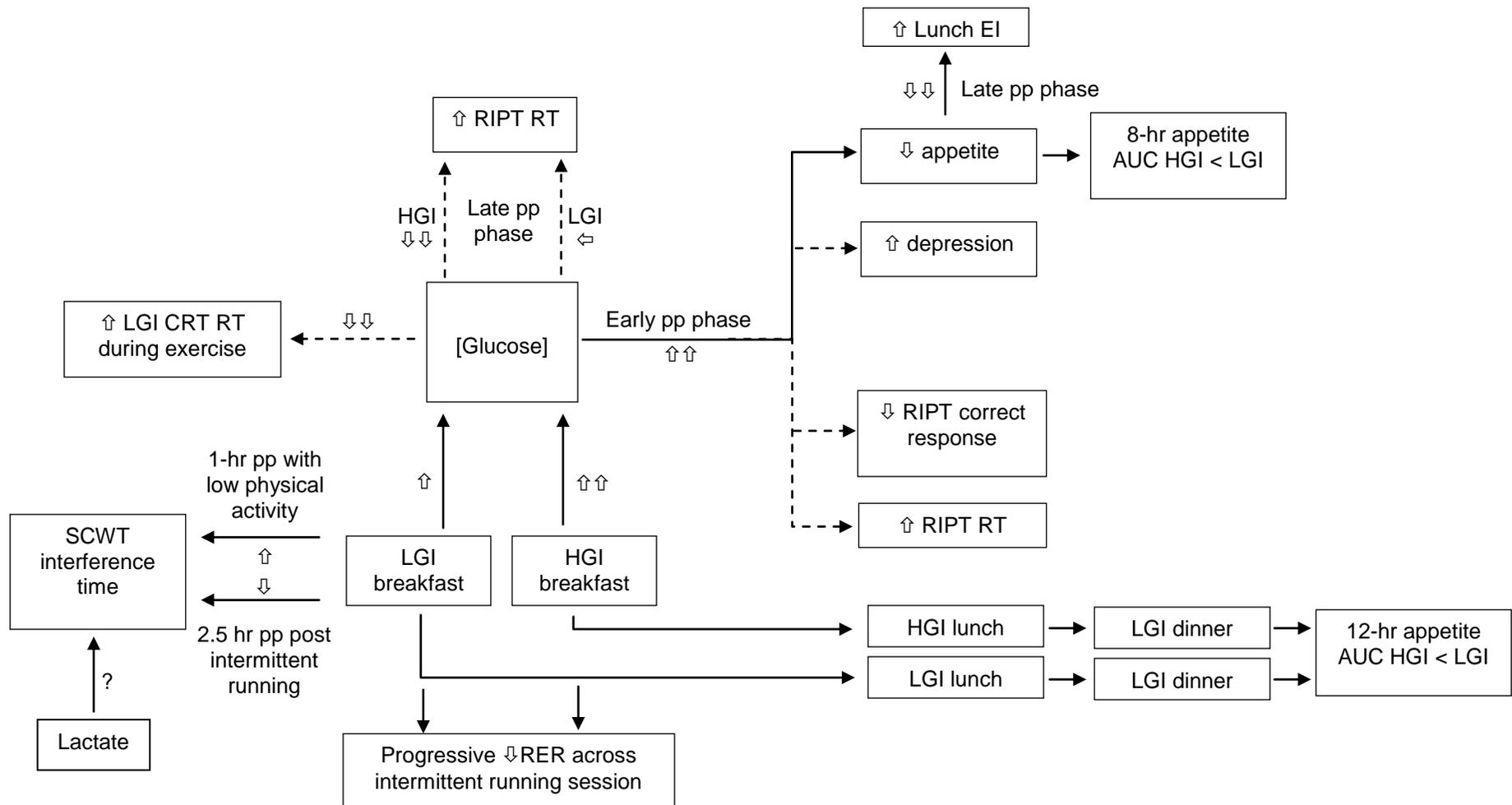


Figure 8.1 Summary of the overall findings

AUC, area under the curve; CRT, choice reaction time task; [Glucose], glucose concentration; HGI, high glycaemic index; hr, hour; LGI, low glycaemic index; pp, postprandial; RIPT, rapid information processing task; RER, respiratory exchange ratio; RT, response time; SCWT, Stroop colour word task; ↑, increase; ↓, decrease; ⇐, reaching fasting level; --->, correlation only without significant trial difference.

8.3 Methodological considerations and limitations

8.3.1 Strengths

8.3.1.1 Appetite sensation

The thesis adhered to research good practice guidelines and recommended statistical standards as suggested by a recent review for appetite assessment (Blundell et al., 2010). The AS in the thesis was composited from the five appetite related elements, desire to eat, hunger, satiety, PFC and fullness, via the VAS questionnaire (Chaput et al., 2010). The AS AUC calculated in the current thesis followed the suggestion not to calculate the IAUC or net AUC (Blundell et al., 2010).

Holt et al. (1995) found that boiled potato, a HGI food, has the highest satiety index among 38 common foods including some LGI high dietary fibre foods such as brown pasta. Furthermore, the satiety effect of potato was reported to last at least for two hours postprandially. Neither is it yet possible to conclude that low GI diets are beneficial for weight loss (Pi-Sunyer, 2002), nor to label HGI diets as bad if the fat and dietary fibre contents meet the healthy guidelines. Previous inconsistent results may result from poor control of variables of other food components which have been known to affect the postprandial glycaemic responses and satiety. Other factors such as inadequate sample number to reach statistical power, dietary confounders such as food size, caloric density, fibre and other macronutrient contents; and insufficient difference in GI between trials have resulted in the inconsistencies (Ludwig, 2007). A methodological strength in this thesis is that the contents of macronutrient, dietary fibre and energy density were controlled (Wolever et al., 2009).

8.3.1.2 Energy intake

It is not uncommon to have intentional or unintentional self-reporting bias in EI. An earlier study observed the reporting bias in self-reported habitual EI and subsequently validated by the doubly labelled water technique (Livingstone et al., 1990). The doubly labelled water technique is considered to be a “gold standard” to assess the energy requirement (Goldberg & Black, 1998), to detect the reporting bias of EI (Trabulsi & Schoeller, 2001); as well as to validate other measurement techniques of EE (Starling, 2002). However, the technique is highly expensive (Achten & Jeukendrup, 2003; Dale, Welk, & Matthew, 2002). The technique can only obtain the average EE but cannot identify the activity pattern such as frequency, intensity and duration. In addition, the return of the urine $^2\text{H}_2$ and ^{18}O levels back to the baseline takes around 7 to 14 days (Starling, 2002). Conversely, de Castro (2006) supported self-report dietary record as a reliable and valid method when all analyses were performed within subjects. Blundell et al. (2010) also recommended that studies investigating the regulation of food intake in short term are best conducted using a within subject repeated measure design. Any underestimation of daily EI in the current thesis affected both trials equally and did not confound the comparison between trials within participants. In addition, energy and CHO intakes were the least variable items in a study analysing 1456 food records of 52 athletes from a variety of sports (Braakhuis et al., 2003).

8.3.1.3 Estimation of GI

The GI values of all meals here were calculated using the method of Wolever and Jenkins (1986). It is important that the difference of the GI values of the mixed meals between the LGI and HGI trials was large enough as a buffer, just

in case the GI values of the single food components varied to narrow the difference of the GI values in the mixed diets between trials, than the actual GI values in the LGI and the HGI mixed diets.

Though all studies in the thesis were well designed, based on previous literatures; there are always further questions raised which need to be answered or explored. All the current studies were short-term and relatively small-scale and investigated healthy and recreationally active adults whilst GI was only one of the factors correlated with appetite. That is the decision of recreationally active adults or coaches to select the diets according to whether the RT or the response accuracy is more of a priority. Below are some suggested areas for further work and new research.

8.3.2 Limitations

8.3.2.1 Study populations

Previous advocates of the beneficial effect of LGI diets have targeted populations groups such as those with obesity or metabolic syndromes (WHO/FAO, 2003). All studies in the present thesis employed a cross-over randomised design. All participants were recruited in the Exeter area. It might be expected that a selection bias exists that only healthy and weight conscious participants were recruited (Rothman, Greenland, & Lash, 2008). Some participants were students in the Sport and Health Sciences, affiliated with sport clubs whilst some others enjoyed exercise more than sport. During briefing the study protocols regarding to the meal intake and appetite sensation, a few participants mentioned about GI on appetite. Nevertheless, all oral and written

messages delivered to participants did not disclose any wordings of glycaemic index. It was attempted to classify the eating behaviours of participants in the study one and two but the sample sizes were still too small for analysing the type of eating behaviour as covariates.

The original aim of the thesis was to investigate the effects of GI in recreationally active Caucasians. Due to the poor response rate of participation in study three, six Caucasian and 10 Chinese recreationally active males were recruited which was the only study with mixed ethnicities. Dickinson et al. (2002) found that the 120-min pp glucose responses remained higher in the Chinese (three males and seven females) than the Caucasian (seven males and 13 females) lean young adults after consumption of 75 g glucose. The south-east Asian (five males and five females) were found to have slowest glucose clearance rate; therefore the south-east Asian was not recruited here. The HGI 45- and LGI 60-min pp [Glucose] were not normally distributed in the current study. No significant differences between the Caucasian and the Chinese in the [Glucose] at the HGI 45-min pp (4.8 ± 0.4 vs. 5.2 ± 0.8 mmol·L⁻¹, $t(14) = -1.1$, $p = 0.2$); and at the LGI 60-min pp (5.3 ± 0.5 vs. 5.7 ± 1.1 mmol·L⁻¹, $t(14) = -0.9$, $p = 0.3$) were found by the independent *t*-test. As the study had a repeated-measure crossover design, participants acted as their own control for comparison. The difference in the postprandial glucose responses between ethnicities were expected to be offset.

Participants were reported as recreationally active at recruitment. The fitness level was not verified except for the $\dot{V}O_2$ max and LT of participants measured in

study four. The findings in study three and four might be of particular interest for recreationally active males in sports with fast decision making, vigilance, selective and sustained attention. The studies did not limit the inclusion criteria to participants accustomed at maintaining high levels of concentration as it might result in another issue of selection bias (Rothman et al., 2008).

8.3.2.2 Study designs

Test meals

The first two studies provided test breakfasts based on the reported habitual breakfast EI of participants and differed from study three in which test breakfasts were provided at a fixed dose of 1 g CHO kg⁻¹ BM. There was a tendency for the HGI AS across the trial morning to be lower than the LGI in study three, but a statistical significant difference was not reached. The discrepancy aroused a concern that a fixed dose provided at breakfast, if below the habitual breakfast EI, might fail to detect the effects of GI on appetite.

All food and drink items provided in the thesis were reported as acceptable by participants via the questionnaire. However, the foods did not match the expectation of several participants when presented. A few participants commented on the potato mash provided at lunch in study three. The feedbacks included the mash was not made by fresh potatoes; the initial portion looked too big which upset the appearance; the taste was too bland and black pepper was requested for flavour enhancing.

Individual preferences on food, food size and taste can affect the appetite and subsequent intake (Popkin & Duffey, 2010). The volume of foods could be a predictor of satiation and satiety regardless of energy density when a constant weight of food was consumed (van Dam & Seidell, 2007). However, it might be no longer to present foods as real meals after foods were blended for meal size and volume control.

The dietary fibre contents were determined via the information provided from the dietary analysis software and food manufacturers. The Englyst defined dietary fibre excludes the content of resistant starch. Wholemeal bread and tortilla were reported to provide 1.2–1.5% resistant starch in some European countries (Liljeberg Elmstahl, 2002; Van Der Kaaij et al., 2009) and 2.1–3.8% resistant starch in a Mexican study (Sayago-Ayerdi, Tovar, Osorio-Diaz, Paredes-Lopez, & Bello-Perez, 2005), respectively. Wholemeal bread and tortilla were provided at lunch in study two. The extent that resistant starch affects the correlation between appetite and GI is unclear here. In addition, some non-CHO components are considered as fibre by AOAC analytical methods for dietary fibre (AOAC International, 2000; McCleary et al., 2012). For instance, lignin is neither a CHO substance nor a viscous fibre but has been defined as a dietary fibre in Europe (EFSA Panel on Dietetic Products Nutrition and Allergies, 2010). The food label of the food items imported from the other European countries did not indicate the definition of the dietary fibre. Besides the issue of the dietary fibre, Livesey (2005) raised the concern of using available CHO in the assessment of the glycaemic response. The classification of available CHO varies amongst authorities which might confound the determination of the GI at mixed meals.

Burke and Deakin (2010) suggested a daily intake of 3–5 and 5–7 g CHO kg⁻¹ BM for skill-based activities and ~ 60-min moderate exercise, respectively. The average pre-trial CHO intake were ~ 386 and ~ 348 g CHO, equivalent to 5.4 and 4.5 g CHO kg⁻¹ BM, in study two and four, respectively. The CHO doses seemed to be lower than the energy requirement for running in study four. The CHO dose-response effect was not tested and it therefore remained unclear whether the current CHO doses were inadequate to elicit the differences between the LGI and HGI trials in substrates oxidation shift or maintain [Glucose] above fasting levels in the late postprandial phase (Wee et al., 2005).

Measurement of appetite

Participants rated the appetite related scores the VAS, during the free living setting which inevitably created an opportunity to examine at the preceding ratings prior to the next rating, amend or rate the scores retrospectively. The accuracy of the ratings from participants who exhibited less compliance could be challenged. A female participant withdraw from study one after the first trial due to the cumbersome completion of the VAS over the trial day.

Biochemical measurements

The methodologies of measuring and interpreting blood glucose responses is important to meet the purpose of the studies (Wolever, 2004). The pre-task [Glucose] were more likely to find significant correlations with the cognitive performances than the post-task [Glucose] in study three; whereas, the pre-exercise [Glucose] was not measured in study four. Blood [Glucose] during the second postprandial hour were more variable within-subjects from day-to-day

than during the first postprandial hour (Wolever, 2004). The intra-individual variability of the [Glucose] therefore was expected to increase across the trial morning. Although the break between exercise sessions in study four was 15 minutes only, it was still not appropriate to apply the post exercise [Glucose] from the previous exercise session as the pre-exercise [Glucose] of the subsequent exercise session for investigating the correlations between pre-exercise session [Glucose] and cognitive performances.

Besides blood [Glucose] as a satiety indicator, insulin was found to have effects on satiety (Anderson & Woodend, 2003). The authors found that high insulin AUC was associated with lower subsequent food intakes in two hours postprandially. Nevertheless, the correlation between AS and [Insulin] was reported not to be as strong as between AS and [Glucose] (Arumugam et al., 2008). The postprandial glucose response explained more of the variance than the insulin response. The current thesis focused on investigating the correlation between appetite and [Glucose], but not [Insulin].

Measurement of energy balance

Although all studies were not conducted in double-blind conditions, the test meals were served by a research dietician, who compiled them according to standard practices and research ethics. The food records were analysed by the same researcher which minimized the inter-researcher variability of dietary assessment. Nevertheless, errors could still not be ruled out through mis-interpretation of the food record and mis-selecting of the alternative food items from the dietary software (Braakhuis et al., 2003).

The lack of significant differences in the energy balance in study one and two may be as a result of the variability of EI and EE. Braakhuis et al. (2003) found the EI was one of the least variable items from 52 athletes that the coefficient of variation within-participants for a 3-day food record was 18%. Finally, the use of accelerometry was limited in its ability to detect certain physical activities e.g., cycling, rowing, upper body weight lifting due to little involvement of hip motion. In addition, all water sports or activities could not be measured due to the non-water proof design.

Exercise

The exercise protocol in study four aimed to mimic the running mode of football that involved low, moderate and high intensities; and short bouts of supramaximal run (Nicholas et al., 2000). Due to the restraint of the programmed treadmill, the shortest duration of each speed could only be set as 30 seconds. Further testing is required to investigate the ecological validation of the protocol of exercise mode in the thesis in the field setting.

8.4 Recommendations for further research

Physiological, psychological and environmental factors can play roles in influencing appetite sensation and cognitive performances (Blundell et al., 2010). The methodology for assessing the sensory initiated appetite is still not well developed. Both the central nervous and the peripheral systems are responsible for responding to the physiological and psychological requirements of metabolic fuels (Stubbs, 1999). It has to be accepted that there is a difficulty in separating the physiological and psychological components on eating

behaviour (Blundell et al., 2010) as well as cognitive performance. The current studies have only investigated the GI effect on the appetite control and cognitive performances based on the peripheral [Glucose] profile following test meals. Below some suggestions are outlined which may help for further exploring the effects of GI and understanding the GI applications:

1. The CHO dose response relationship for appetite, cognitive responses and substrate oxidation during intermittent exercise in field based settings.
2. Continuous monitoring of postprandial [Glucose] and brain activity using electro-encephalograms and functional magnetic resonance imaging whilst performing cognitive tasks.
3. Recruitment of recreationally active females to verify the applicability of the current findings shown in the male participants.
4. A coding system for participants and researcher for reducing the variability of assessment of energy and macronutrient intakes (Braakhuis et al., 2003).
5. A combined use of HR and accelerometry for improving the accuracy of EE estimation (Bassett, 2000; Brage et al., 2004; Strath et al., 2000); and for filling the gap between the restraint of the accelerometry on certain physical activities as well as, better accuracy of estimating the physical activity EE by HR on an individual level rather than on a group level (Achten & Jeukendrup, 2003).
6. An introduction of the electronic VAS for appetite, mood, cognitive and sensory stimuli (Jamison et al., 2002; Whybrow, Stephen, & Stubbs, 2006): the electronic VAS with its reminding function facilitates participants to rate

on time; and prevents from retrospective rating or corrections. The controversial findings of the interchangeability of the data between the electronic and the paper VAS should be tested (Jamison et al., 2002; Whybrow et al., 2006).

7. Performing additional cognitive tasks at the fasting phase to differentiate the direction of the changes in cognitive performances across time.
8. Applications of other cognitive tasks with similar cognitive characteristics such as vigilance and selective attention, to verify whether the differences in cognitive performances between trials were task specific.

CHAPTER 9 CONCLUSIONS

Many sports require the players to optimally perform physically fit, with multi-dimensional coordination involving mood, physical movement and cognition for success. These results provide further evidence to support the glucostatic theory links between postprandial changes in blood [Glucose] and appetite sensation. High blood [Glucose] was correlated with appetite suppression. Frequent consumption of HGI CHO suppressed appetite more than the LGI CHO. Recreationally active adults looking for better appetite control can distribute the intake of HGI CHO over a day. The findings here suggest that the claim of the benefit of LGI foods on weight management and appetite control is not applicable to the recreationally active adults. Pre-meal appetite sensation is a more sensitive predictor of the intake than pre-meal [Glucose]. In future, dietary strategies for weight management should focus more on greater postprandial appetite suppression whilst following healthy eating guidelines, than monitoring the pre-meal glucose level.

The LGI and HGI breakfasts had different effects on the RIPT, CRT and the SCWT performances. The potential compromise in the sustained attention and vigilance performance evaluated by the RIPT was found to be associated with the HGI breakfast at the early post postprandial period in both response time and accuracy and at the late postprandial period in response time. The selective attention evaluated by the SCWT interference time was not maintained following the LGI breakfast at the late postprandial period under a condition of low physical activity. Cognitive performance during and post

intermittent exercise can also be affected by a pre-exercise meal. A LGI breakfast providing 1.2 g CHO kg⁻¹ BM taken one hour prior to three 16-min intermittent running sessions reduced the SCWT interference time at the last exercise session which indicated an improvement in the selective attention. The current CHO doses lead to comparable substrate oxidation during intermittent running between trials. Recreationally active males performing sports with low physical requirements can benefit overall from the LGI breakfast when vigilance and selective attention needed to be maintained up to 90 minutes post breakfast. Recreationally active males performing sports requiring higher selective attention with moderate to high intermittent intensities can benefit from a LGI breakfast, particularly in the late exercise period.

In summary, both LGI and HGI breakfasts had advantages on appetite and cognition at different aspects. The current findings provided practical implications applicable to sports persons. Recreationally active adults should consider the timings of the meal consumption and sport types to maximize the advantages from the LGI or the HGI breakfasts. It remains open to further investigate any dose-response relationship, the changes in blood [Glucose], the timing for task performance, the brain utilization of glucose during demanding cognitive tasks, as well as, the combined effects of macronutrients between the LGI and HGI fixed meals.

CHAPTER 10 PRACTICAL APPLICATIONS OF LOW AND HIGH GLYCAEMIC INDEX MEALS ON APPETITE, COGNITIVE PERFORMANCES FUNCTION AND METABOLIC RESPONSES DURING INTERMITTENT EXERCISE IN RECREATIONALLY ACTIVE ADULTS

“Good food choices will not make a mediocre athlete into a champion, but poor food choices may prevent the potential champion from realising his/her potential” (Maughan & Shirreffs, 2012). Many nutrition and sports associations such as the Medical Commission of the International Olympic Committee, the English Institute of Sport and the Professionals in Nutrition for Exercise and Sport, etc. have provided position papers and statements on sports nutrition to emphasize the importance of diet on athletic performance. The current thesis aimed to investigate the effects of GI on appetite, cognitive performances and substrates oxidation during intermittent exercise so as to provide some practical use of GI to recreationally active adults, coaches, sports and nutrition organisations.

1. Recreationally active adults aiming to have better appetite control can distribute HGI CHO foods throughout a day.
2. A single HGI breakfast can induce greater appetite suppression until mid-afternoon.
3. The appetite suppression does not guarantee a reduction in EI or to achieve negative energy balance.

4. Recreationally active males performing sports requiring high levels of vigilance and selective attention with low physical activity levels can benefit from a LGI breakfast up to 90 minutes post breakfast.
5. Recreationally active males performing sports requiring high level of selective attention with moderate to high intermittent intensities can benefit from a LGI breakfast, particularly in the late exercise period.
6. Following LGI and HGI breakfasts providing 1.2 g CHO kg⁻¹ BM resulted in comparable substrates oxidation in intermittent running performance of less than 50 minutes. Recreationally active males may be more liberal in pre-exercise food choices if substrate oxidation during intermittent running is their only concern.

All findings in the thesis were short-term and laboratory based investigations in healthy and recreationally active adults. Whether athletes competing at the elite level or recreationally active adults enjoying exercise are suggested to consult qualified professionals in sports and nutrition appropriately for more sport specific nutritional strategies of GI on reaching optimum body composition, mental, cognitive and physical performances; and better health.

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APPENDICES

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Appendix A: Published paper

Eur J Nutr
DOI 10.1007/s00394-013-0529-3

ORIGINAL CONTRIBUTION

Glycaemic index of meals affects appetite sensation but not energy balance in active males

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Received: 21 January 2013 / Accepted: 19 April 2013
© Springer-Verlag Berlin Heidelberg 2013

Abstract

Background Foods with low glycaemic index (LGI) are reported to suppress appetite mainly in overweight population but have not been investigated in athletic adults.

Objective The aim of this study was to compare the short-term effects of LGI and high GI (HGI) meals over a day on subsequent subjective appetite sensation, energy intake, energy expenditure, energy balance and resting metabolic rate in physically active males.

Methods This cross-sectional randomized crossover study included 14 active males (mean ± SD; age 34.5 ± 8.9 years, body mass index $22.8 \pm 2.1 \text{ kg m}^{-2}$) to consume LGI and HGI meals on two separate days. On each trial day, participants consumed a breakfast in the laboratory and then left with a packed lunch, dinner and snacks. Appetite scores, energy intake and expenditure were assessed.

Results The area under the curve for appetite scores of the HGI trial was significantly smaller than that of the LGI trial during the laboratory period ($p = 0.027$) and throughout the day ($p = 0.009$). No significant differences in energy intake, energy expenditure, energy balance and resting metabolic rate were found between groups, between the trial days and between the corresponding post-trial days.

Conclusions These results show that frequent ingestion of the HGI meals, contrary to the previous reports, suppresses appetite more than that of LGI meals, but did not affect energy balance in physically active normal-weight males.

Keywords Energy intake · Energy expenditure · Hunger · Satiety · Carbohydrate · Body weight

Abbreviations

AS	Appetite score
AUC	Area under the curve
BMI	Body mass index
CHO	Carbohydrate
EB	Energy balance
EE	Energy expenditure
EI	Energy intake
FFM	Fat-free mass
GI	Glycaemic index
HGI	High glycaemic index
LGI	Low glycaemic index
PS	Palatability score
RMR	Resting metabolic rate
VAS	Visual analogue scale

Introduction

Weight management strategies are undertaken by overweight and obese individuals, but they are also performed by athletes [1]. The regulation of energy intake (EI) is multi-factorial, including environmental, biological and individual influences, as well as a strong genetic component. Therefore, a precise understanding of the relationship between food intake, appetite and weight management is important for athletes wishing to develop appropriate dietary regimens to control body weight while optimizing exercise performance.

Appetite is a driving force for the pursuit, selection and ingestion of food. Appetite suppression, as a weight

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management strategy, might help to control EI without the feeling of being deprived of food intake. Appetite, including satiation and satiety, could potentially be altered by varying the glycaemic index (GI) of ingested meals [2–4]. The GI system was established in the early 1980s as a measure of the bioavailability of carbohydrate (CHO)-rich foods [5]. Previous studies have reported an association of low GI (LGI) food consumption with health benefits in individuals with chronic conditions such as hypercholesterolaemia, diabetes or obesity [5, 6] and have been recommended for weight management and better glycaemic control in the last two decades [7, 8]. To date, nearly 2,000 foods have been assessed based on the GI ranking system [9].

The rationale of the advantage of LGI diets for weight management is due to its potential satiating effects leading to a reduction in subsequent EI. Some studies have suggested that LGI foods have higher satiating effects compared to the high GI (HGI) foods [10, 11]. Ludwig et al. [10] found that the ratings of hunger in 12 obese male adolescents were higher following a HGI breakfast than following a LGI breakfast. Furthermore, the subsequent EI 5 h after the HGI breakfast was 81 % greater than after the LGI breakfast. On the contrary, another study found that HGI foods suppressed subjective appetite leading to lower subsequent EI than following LGI foods in non-obese male adults [12]. These conflicting findings may relate to the different sample population since there appear to be different in the perceived appetite sensation between obese and non-obese adults and between physically active and sedentary populations [13, 14]. To date, the effect of GI on appetite and subsequent EI has not been investigated in male athletes. Therefore, the purpose of this study was to compare the short-term effects of LGI and HGI meals over a day on subsequent subjective appetite sensation, EI, energy expenditure (EE), energy balance (EB) and resting metabolic rate (RMR) in physically active males. It was hypothesized that LGI meals would suppress appetite and lead to a negative EB compared to HGI meals in a trial day.

Methods

Design

A randomized, crossover, within-subject design was utilized to investigate the effects of GI on the subjective appetite sensation and EB of physically active male adults over a single trial day. Participants ingested either a LGI or HGI breakfast at the laboratory and then left with a take-away bag containing test food and drinks for lunch, dinner and snacks. Participants returned to the laboratory the next morning for a follow-up assessment of RMR. Participants

received the alternative test meals after a washout period of at least 2 days [15, 16].

Participants

Volunteers completed an eating attitude test (EAT-26) [17], a health questionnaire and a food preference questionnaire as part of the eligibility screening. Eligibility was based on the following criteria: male, Caucasian, 18–50 years of age, less than 10 % fluctuation of body weight in the last 3 months, to exercise at moderate intensity for more than 30 min at least three times a week, non-regular smoker, no known metabolic or eating disorder, no food allergy or aversion to the test meals, and to consume breakfast at least three times per week in the last month. Participants were informed of the testing procedures and the possible risks of the study and were briefed that the aim of the study was to investigate general aspects of meals on appetite sensation, but not the specific purpose of investigating the GI effects on appetite. Written informed consent was obtained prior to the first trial. A power calculation based on previous papers [10, 18] indicated that 10 participants would be needed in order to detect a significant change of 26 mm in hunger rating [10] and of 34 VAS units of 180 min in area under the curve of satiety [18] at an alpha level of 0.05 with a power of 80 %. The study was approved by the Ethics Committee of the institution in accordance with the Declaration of Helsinki.

Preliminary procedures

Participants recorded 2 days of food and drink intake prior to the first trial for determination of energy content of the test meals. Two days prior to each trial day, they were required to record food intakes and to wear an accelerometer (ActiGraph GT1M accelerometer, ActiGraph LLC, Pensacola, FL, US). Participants were instructed to maintain their habitual physical activities and dietary patterns and refrain from unusual strenuous exercise for the 24 h preceding each trial day [19]. No alcohol was permitted during this period [16]. In order to minimize any evening meal effect on RMR and appetite, participants were instructed to replicate the pre-trial evening meals of the first trial prior to the second trial [16, 19]. Participants were not allowed to eat or drink anything for 10 h before arrival at the laboratory for each trial day [16].

Trial procedures

Participants arrived at the laboratory between 0700 and 0900 hours on the trial days after an overnight fast for 10 h with the accelerometer being worn. Body mass and body stature were taken wearing light clothing. Participants then

rested supine for 45 min to collect gas for the RMR measurement. A glass of water (250 mL) was served to participants afterwards to minimize dehydration and difference in the gastric emptying before the consumption of the test breakfast. Participants then stayed in a quiet room for 15 min before the test breakfast was served [2].

Participants rated their levels of appetite, physical comfort and impression of the breakfast on a visual analogue scale (VAS) questionnaire before consuming the breakfast. Participants were required to consume the breakfast within 20 min [16]. Water was provided ad libitum with the breakfast. Then the same VAS questionnaire was completed immediately following the test breakfast, which was 20 min from the start of the meal. All VAS questionnaires for appetite sensations were rated every 10 min postprandially for an hour [13, 20]. Participants were allowed to read, relax and browse the internet between ratings, with an exception of any reference to literature related to foods and drinks [13]. Participants stayed in the same room alone in both trials in order to let them focus on eating and to reduce social influences or any disruption from habitual satiation processes [21].

Participants were provided with packed test foods and drinks before departure. A free food list was enclosed for participants to top up extra foods when they felt hungry after all foods of each meal provided had been consumed [22]. Non-calorie-containing drinks such as water, tea and coffee were allowed ad libitum. On leaving the laboratory, participants were required to rate their appetite on the VAS hourly during the waking hours for the remainder of the trial day plus the VAS for palatability after lunch and dinner. Participants were instructed to take lunch and dinner between 1200 and 1400 hours and between 1800 and 2000 hours, respectively, to minimize any mealtime effect on appetite sensation rating. Participants were free to consume the provided snacks 2 h after each main meal (Fig. 1).

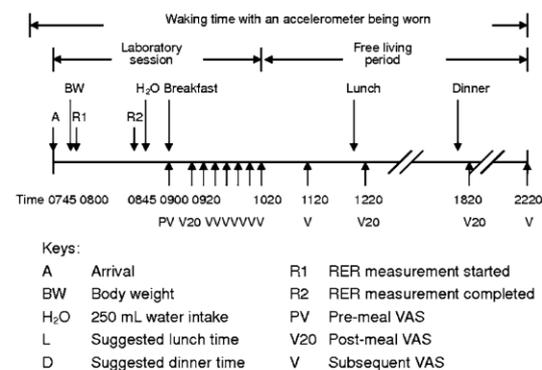


Fig. 1 Schematic representation of the study protocol on the main trial day

Participants returned to the laboratory the same time the next day after the overnight fast. A 45-min post-trial RMR was measured. The anthropometric measurements were completed before departure. Participants continued to record the food intake and to wear the accelerometer during the waking hours for the day following each trial.

Anthropometry

Stature, body mass and four sites of skinfold thickness (iliac crest, subscapular, triceps and medial calf) were measured by using standard procedures [23]. The sum of the skinfold thickness was used to estimate the percentage of body fat [24] and fat-free mass (FFM).

Test meals

The GI values of the meals were calculated from the weighted means of the GI values of individual CHO-containing foods from the published sources [9] using the mixed-meal method [25]. The overall GI values for the planned LGI and HGI trial were 39.6 ± 0.7 and 76.9 ± 2.8 , respectively, with a difference of 37.3 ± 2.7 between trials. The majority of LGI and HGI CHO-rich foods and drinks had a GI lower than 55 and over 71, respectively (Table 1). Based on the food preference questionnaire, no food scored as ‘dislike very much’ was provided (1 on a 5-point Likert Scale).

The total energy content of the test meals was determined from the 2-day pre-trial food record. The energy content of the test breakfast ($E_{\text{breakfast}}$) was determined from the reported breakfast intake. The energy contributions of breakfasts from CHO, protein and fat were set as 60, 15 and 25 %, respectively. Both LGI and HGI breakfasts matched with contents of saturated, monounsaturated and polyunsaturated fats; fibre; and caloric density and presented the same number of food items. The variety was limited to not more than four items per meal to reduce the effects of variety associated with the disruption of habituation [21].

The energy content in the packed foods was set from the difference in the total energy content and the energy contributed from the test breakfast. The packed lunch and dinner each contributed 30 % of the total energy content. The energy ratio from CHO/protein/fat for lunch, for dinner and for the trial day was set as 65:15:20. A cooking instruction sheet for dinner was provided for participants to minimize any cooking effects on GI values. Non-CHO-containing foods were added to match the fibre, protein and fat contents similar between the test meals. A low-calorie drink was provided to match the caloric density between the test meals at each main mealtime (Table 1). The brands and the nutrition labels of the foods and drinks were either

Table 1 Breakdown of the low glycaemic index (LGI) meal and the high glycaemic index (HGI) meal plans for a participant requiring 9.4 MJ (2,255 kcal)

LGI (GI = 40)			HGI (GI = 79.5)		
Breakfast	GI	Quantity/g	Breakfast	GI	Quantity/g
Low fat milk	32	341	Rice milk	79	195
Muesli	55	60	Corn flakes	93	25.2
Dried apricots	32	26	Fruit'n fibre	67	50
Fructose	19	13.4	Light cheese	0	42.8
Macadamia	0	6.2	Macadamia	0	5.1
Water	0	31.4	Tomatoes	0	160
Breakfast GI	41.9	478		72.6	478
Lunch	GI	Quantity/g	Lunch	GI	Quantity/g
White tortilla	30	146.5	Wholemeal bread	74	137
Apple juice	44	330	Glucose drink	93	380
Dried apricots	32	21	Cucumber	0	65
Tuna in brine	0	46.6	Tuna in oil	0	28.5
Almonds	0	7.4	Almonds	0	6.2
Water	0	72.2	Butter	0	7
Lunch GI	34.4	624		82.9	624
Dinner	GI	Quantity/g	Dinner	GI	Quantity/g
Fusilli	54	70	New potatoes	80	220
Apple juice	44	330	Glucose drink	93	380
Milk Chocolate	40	28.5	Plain chocolate	49	30
Dried apricots	32	38	Spinach	0	92
Roasted beef	0	50	Roasted beef	0	65.6
Almonds	0	5.5	Almonds	0	7.3
Diet drink	0	272			
Olive oil	0	1.3			
Dinner GI	46.1	795		80.4	795
Snack	GI	Quantity/g	Snack	GI	Quantity/g
Canned peaches	40	190	Canned lychees	79	304
Apple juice	44	330	Strawberry yogurt	85	125
Fructose	19	15.4	Cheese	0	40.5
Natural yogurt	35	42	Water	0	150
Cheese	0	42			
Snack GI	37.4	619		80.5	620

torn away or covered to minimize participants accessing their EI. All meals and snacks were freshly prepared and packed in the morning of each main trial day and kept in the refrigerator until departure. Participants were encouraged to consume all provided foods. Nevertheless, they did not have to consume all if they were full. Conversely, after consumption of all provided foods at each mealtime, participants could add foods selected from a provided list such as green leafy vegetables and tomatoes if they still felt hungry.

Appetite and palatability

Subjective appetite sensation with the elements of hunger, desire to eat, satiety, prospective food consumption and fullness [26], physical comfort, as well as palatability with the sensation variables of visual, smell, taste and palatability of the test meals were assessed using the 100-mm VAS [20, 27]. The use of VAS for assessing subjective appetite sensation has been widely used and accepted [28]. An appetite score (AS) [26] was developed from the VAS.

Lower composite AS is associated with greater suppression of appetite. Palatability also includes several sensational elements such as visual, smell and taste [27]. A palatability score was calculated from an average of these elements. Higher composite PS represents the meal is more palatable. The equations of calculating the AS [26] and the PS were as follows:

$$AS = [\text{Desire to eat} + \text{hunger} + \text{prospective food consumption} + (100 - \text{fullness}) + (100 - \text{satiety})] / 5 [26]$$

$$PS = (\text{Visual} + \text{smell} + \text{taste} + \text{palatability}) / 4.$$

Energy intake, energy expenditure and energy balance

The 2-day dietary records prior to each trial day were treated as a baseline. The dietary records on the trial day as well as on the post-trial day were treated as intervention data. Participants were briefed about the format of recording intakes and to provide detailed description of food and drink items being consumed including mealtime, location, portion and brand name [29]. Written instruction and food record sheets were given. A registered dietitian went through the food records and clarified any unclear or missing information with participants. Food records were analysed using a commercially available nutrient analysis software (Microdiet 2.0, Downlee Systems Ltd., High Peak, UK).

Participants were required to wear the accelerometer for estimation of the baseline EE 2 days prior to each trial day. They were required to continue to wear the accelerometer on the trial day as well as on the post-trial day so as to collect data as intervention. Verbal and written instruction of the use of the accelerometer was given. The same serial accelerometer was provided to participants during the trial periods to minimize between-device variations wherever possible. The data were initially collected in 5-s epochs and then converted to 1-min epochs for estimating the EE by the software (V5.6.4 ActiLife, FL, US) using the cutpoints of Troiano et al. [30]. The EB was calculated from the difference in EI and EE.

Resting metabolic rate was measured by indirect calorimetry (Cortex Metalyser 3B, Leipzig, Germany). The gas analyser was calibrated prior to each measurement by ambient air and a standard gas (16 % O₂, 4.96 % CO₂). Flow calibration was performed using a 3-L syringe (Hans Rudolph, UK). Participants were instructed to remain awake in a supine position on a bench in a quiet and temperature- and humidity-controlled room for 45 min after wearing a gas collection mask [31]. Readings were taken continuously, and the readings of the first 10 min were not used for analysis. The volumes of oxygen consumption and carbon dioxide production were recorded. The rates of CHO and fat oxidation were then calculated using non-protein stoichiometric equation [32, 33].

Data analyses

Descriptive statistics were calculated for group characteristics. A two-way repeated-measures analysis of variance was used to test for trial effect, time effect and trial-by-time interaction on the mean AS and EI. Greenhouse–Geisser corrected *p* values were reported when the assumption of sphericity was not met. The total area under the curve of the AS (AS_{AUC}) was examined (mm min) using Sigma-Plot® 10.0 (version 10.0, Systat Software Inc, UK). Pearson's correlation analyses were applied to examine the associations between appetite, EI_{breakfast}, EI at lunch (EI_{lunch}), EI at dinner (EI_{dinner}) and total EI, and palatability. Multiple linear regression analysis was used to determine the predictive value of EI for the post-meal AS in the different trials. Differences between trials in the pre-breakfast AS, the AS_{AUC} at the laboratory and at the free-living condition, and the post-meal AS and the post-meal PS at each mealtime were assessed using paired sample *t* tests. Any trial order effect, that is, the first trial versus the second trial, was also examined. All values were presented as means and standard deviations (SD) using PASW Statistics 18 (version 18.0, SPSS Inc., Chicago, IL, US) unless specified as standard error of mean (SEM). All statistics were performed at the significance level of $\alpha = 0.05$.

Results

Descriptive characteristics

Fifteen participants initially enrolled for the study. One participant withdrew from the study after the first trial due to sickness. The data collected from this participant were excluded from analysis. Fourteen physically active males completed the study successfully (Table 2).

Glycaemic index of the meals

The actual calculated GI values of each meal were determined after accounting for the final actual food consumption. The GI

Table 2 Anthropometric and physiological characteristics of the participants

Variables	Participants (<i>n</i> = 14)
Age (years)	34.5 ± 8.9
Body mass (kg)	71.9 ± 10.6
Body mass index (kg m ⁻²)	22.8 ± 2.1
Body fat percentage (%)	20.7 ± 4.8
Fat-free mass (kg)	56.9 ± 7.6

Values presented as mean ± SD

n Number of participants

Table 3 Planned and actual glycaemic index values of the test meals

Meal	Planned LGI	Planned HGI	Actual LGI	Actual HGI	Difference in actual GI
Breakfast	41.1 ± 1.2*	73.9 ± 1.7**	41.3 ± 1.4*	74.3 ± 1.4	33.1 ± 2.2
Lunch	34.3 ± 0.8*	81.2 ± 2.3	34.1 ± 1.1*	81.4 ± 2.9	47.3 ± 3.1
Dinner	45.1 ± 1.9*	76.9 ± 2.8	44.9 ± 2.1*	75.1 ± 4.6	30.3 ± 3.4
Overall	39.6 ± 0.7*	76.9 ± 2.8	39.6 ± 1.0*	76.2 ± 2.8	36.6 ± 2.9

Values presented as mean ± SD ($n = 14$)

LGI low glycaemic index, HGI high glycaemic index

* Significantly lower than the corresponding HGI mealtimes ($p < 0.001$)

** Significantly lower than the corresponding actual GI ($p = 0.05$)

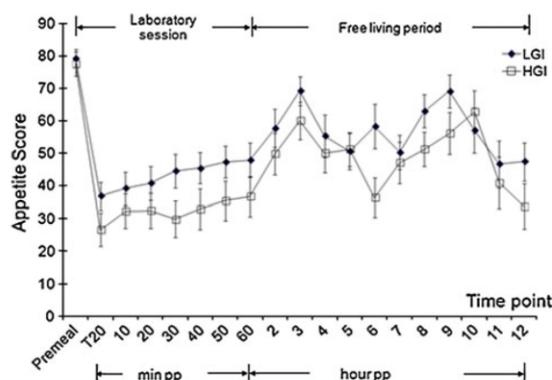


Fig. 2 The effect of GI values on subjective appetite sensation. Values presented as mean ± SEM ($n = 14$). LGI low glycaemic index, HGI high glycaemic index, T20 20 min following the breakfast, pp postprandial

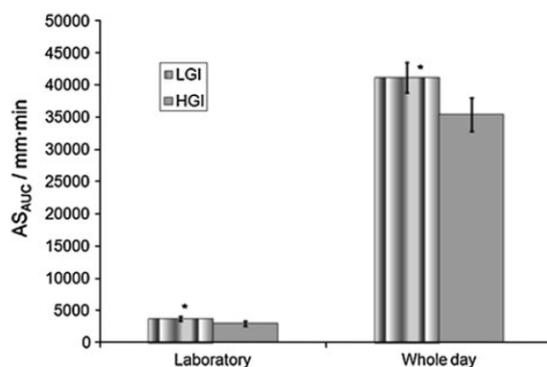


Fig. 3 The area under the curve of the appetite score at the laboratory and throughout the trial days between trials. Values presented as mean ± SEM ($n = 14$). LGI low glycaemic index, HGI high glycaemic index, AS_{AUC} area under the curve of the appetite score. * Significantly higher than the corresponding HGI AS_{AUC} ($p < 0.05$)

values of the planned LGI and the actual LGI at each main meal and in overall remained significantly lower than those of the corresponding HGI mealtimes. The actual HGI breakfast

GI value was significantly higher than that of the planned GI value ($t(13) = 2.2$, $p = 0.05$), whereas no significant differences were found in the calculated GI values between the planned meals and the actual consumption of lunch and dinner at both trials (Table 3).

Appetite sensation

There was no significant difference between trials in the AS prior to breakfast respectively (79.3 ± 10.1 vs. 77.6 ± 14.0 , $t(13) = 0.41$, $p = 0.69$). Independently of the GI values, the AS immediately after the breakfasts decreased significantly compared to the pre-meal AS (78.4 ± 12.0 vs. 31.7 ± 18.0 , $t(27) = 11.9$, $p < 0.001$). There were no trial-by-time interactions during the laboratory session ($p = 0.2$). The averaged AS immediately after the three HGI main meals (breakfast, lunch and dinner) was significantly lower than that in the LGI trial (27.2 ± 12.5 vs. 34.0 ± 15.6 , $t(13) = -2.5$, $p = 0.024$). There existed main trial effects that the HGI AS was significantly lower than the LGI AS in the laboratory session ($F(1,13) = 6.0$, $p = 0.03$) and throughout the trial day ($F(1,13) = 13.5$, $p = 0.003$). Significant time effects during the period in the laboratory ($F(2,26.5) = 54.5$, $p < 0.001$) and throughout the trial day ($F(4.5, 57.9) = 10.2$, $p < 0.001$) were also found. No trial-by-time interactions throughout the trial day were observed ($p = 0.3$) (Fig. 2).

The HGI AS_{AUC} was significantly smaller than the LGI AS_{AUC} during the laboratory session ($2,989 \pm 1,390$ mm min vs. $3,758 \pm 1,290$ mm min, $t(13) = -2.5$, $p = 0.027$) and throughout the trial day ($35,454 \pm 9,730$ mm min vs. $41,244 \pm 8,829$ mm min, $t(13) = -3.1$, $p = 0.009$) (Fig. 3). There was no trial order effect for the AS either during the laboratory session ($p = 0.4$) or throughout the day ($p = 0.7$).

Energy intake, energy expenditure and energy balance

There was no significant difference in the $EI_{\text{breakfast}}$ between trials ($t(13) = -0.66$, $p = 0.5$) (Table 4). In the

Table 4 Comparison of the energy and the macronutrient intakes between trials on the trial days

Intakes	Trial	
	LGI	HGI
Breakfast (MJ)	2.4 ± 1.0	2.5 ± 1.2
Lunch (MJ)	3.2 ± 0.7 ^a	3.5 ± 0.8
Dinner (MJ)	3.6 ± 0.9	3.5 ± 0.9
Snack (MJ)	2.0 ± 1.6 ^b	1.7 ± 1.4
Carbohydrate (g)	444.7 ± 87.8	445.9 ± 102.7
Protein (g)	102.5 ± 21.7	101.6 ± 21.3
Fat (g)	63.9 ± 13.6	63 ± 16.6
Saturated (g)	26.5 ± 5.6	26.1 ± 8.1
Monounsaturated (g)	25.3 ± 6.3	23.6 ± 6.1
Polyunsaturated (g)	8.3 ± 1.7	9.1 ± 2.2
Fibre (g)	25.4 ± 3.8	24.3 ± 4.4
Energy intake (MJ)	11.2 ± 2.2	11.1 ± 2.5

Values are mean ± SD (*n* = 14)

LGI low glycaemic index, HGI high glycaemic index

^{a,b} Significantly lower and higher than the corresponding HGI variables (*p* < 0.05)

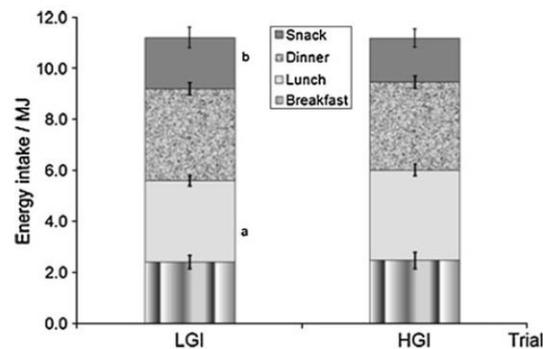


Fig. 4 Energy intake at different meals over the trial days. Values presented as mean ± SEM (*n* = 14). LGI low glycaemic index, HGI high glycaemic index. ^{a,b} Significantly lower (*p* = 0.033) and higher (*p* = 0.035) than the corresponding HGI meals, respectively

free-living setting, the LGI EI_{lunch} was found to be significantly lower than the HGI EI_{lunch} (*t*(13) = -2.4, *p* < 0.033), whereas the LGI snack EI (EI_{snack}) was significantly higher than the HGI EI_{snack} (*t*(13) = 2.4, *p* = 0.035), leading to no significant difference in the total EI (*t*(13) = 0.1, *p* = 0.9) (Fig. 4).

Due to technical faults with the accelerometers, pre-trial and the intervention data were only available for *n* = 13 and 12, respectively. There were no trial, time and trial-by-time interaction effects for EI, EE and EB among the baseline, the trial day and the post-trial day between trials (Table 5).

Table 5 Comparison of the energy intakes, energy expenditure and energy balance between trials

	Trial	
	LGI	HGI
<i>Pre-trial</i>		
EI (MJ)	11.3 ± 2.8	11.5 ± 2.9
EE (MJ)*	11 ± 2.4	10.5 ± 2.4
EB per FFM (kJ kg ⁻¹)*	-1.0 ± 64.3	-19.7 ± 63.9
<i>Trial</i>		
EI (MJ)	11.2 ± 2.2	11.1 ± 2.5
EE (MJ)**	10.5 ± 2.3	11.0 ± 3.3
EB per FFM (kJ kg ⁻¹)**	-6.5 ± 45.3	-2.9 ± 53.6
<i>Post-trial</i>		
EI (MJ)	10.2 ± 2.0	10.6 ± 2.0
EE (MJ)**	10.8 ± 2.1	10.5 ± 2.7
EB per FFM (kJ kg ⁻¹)**	-11.3 ± 45.3	0.3 ± 38.6

Values are mean ± SD (*n* = 14, except * *n* = 13 and ** *n* = 12)

LGI low glycaemic index, HGI high glycaemic index, EI energy intake, EE energy expenditure, EB energy balance, FFM fat-free mass

There was no significant trial effect on RMR between trials (data not shown). The rate of fat oxidation in the HGI post-trial morning tended to be higher than that in the HGI trial morning (0.103 ± 0.03 vs. 0.089 ± 0.03 g min⁻¹, *t*(13) = 1.98, *p* = 0.069).

Palatability

No significant differences were found between trials in the PS following breakfast (*p* = 0.78) or lunch (*p* = 0.68). However, participants reported that the HGI dinner was significantly more tasty (74.7 ± 17.0 vs. 58.6 ± 23.5, *t*(13) = 3.7, *p* = 0.003) and more palatable (72.0 ± 18.6 vs. 56.4 ± 22.9, *t*(13) = 3.1, *p* = 0.009), leading to greater HGI dinner PS (69.0 ± 20.7 vs. 56.1 ± 23.1, *t*(13) = 2.6, *p* = 0.024) than the LGI dinner PS. Significant negative correlations were found between the post-dinner PS and AS at both LGI and HGI dinners, which explained 56 % (*p* = 0.002) and 48 % of the variance (*p* = 0.007), respectively.

Relationships between appetite sensation and energy intake

In order to remove any confounding effect from the PS, the correlations between AS and the EI at each meal were assessed after an adjustment of the PS. Independently of the GI values, significant negative correlations were found between EI_{breakfast} (*p* = 0.002), EI_{lunch} (*p* = 0.008) and their corresponding postprandial AS, and between the total EI without EI_{snack} (*p* = 0.025) and the overall postprandial

Table 6 Correlations between energy intakes of breakfast, lunch and dinner; and total energy intake without snack and the corresponding postprandial appetite scores

Energy intake	Independent of GI*	LGI	HGI
Breakfast	$r = -0.57$ $p = 0.002$	$r = -0.64$ $p = 0.01$	$r = -0.58$ $p = 0.035$
Lunch	$r = -0.46$ $p = 0.011$	$r = -0.44$ $p = n.s.$	$r = -0.59$ $p = 0.012$
Dinner	$r = -0.13$ $p = n.s.$	$r = -0.29$ $p = n.s.$	$r = -0.06$ $p = n.s.$
Total without snack	$r = -0.39$ $p = 0.015$	$r = -0.3$ $p = n.s.$	$r = -0.49$ $p = 0.046$

LGI low glycaemic index, HGI high glycaemic index, n.s. non-significance, $n = 14$, except * $n = 28$

AS, which explained 32.5, 24.6 and 13.4 % of variances, respectively. In an aspect of the GI effect, only the LGI $EI_{\text{breakfast}}$ was found to be negatively correlated to its post-breakfast AS, which explained 40.9 % of variance, whereas $EI_{\text{breakfast}}$ ($p = 0.035$) and the total EI without EI_{snack} ($p = 0.046$) at the HGI trial were negatively correlated to the corresponding post-meal AS, which explained 34.2 and 23.8 % of variance, respectively (Table 6). The negative correlations between EI and AS meant the higher the EI at one meal, the lower the rating of appetite sensation following that meal.

Discussion

This is the first study to investigate the regulation of short-term appetite and energy balance following consumption of LGI or HGI foods by physically active male adults. We hypothesized that LGI foods could suppress appetite to a greater extent than HGI foods; however, the current findings did not support our hypothesis. The results illustrated that HGI meals suppressed appetite to a greater extent than LGI meals in physically active males. Previous studies had demonstrated the advantage of LGI foods in overweight participants on appetite suppression [10, 11], whereas physically active males were recruited in our study. Our findings matched with the finding of another study that involved men with normal BMI [12]. The authors found that HGI CHO resulted in greater subjective appetite suppression than LGI CHO and the effect could last for an hour. Thus, the discrepancy in the current findings to the previous preferences on LGI foods on appetite suppression might result from the differences in body composition. In addition, the current findings showed that when consumed frequently, the appetite-suppressing effects of HGI foods appeared to last for a whole day, longer than observed previously [12].

In the light of the lower AS_{AUC} in the HGI trial, it was surprising that the LGI EI_{lunch} was significantly lower than the HGI EI_{lunch} without any significant difference in the post-lunch AS between trials. The current study provided energy-fixed meal conditions, rather than an ad libitum environment, although participants were asked to return any leftover foods if they felt full. Participants scored the appetite ratings after consuming those test foods. In accordance with the time sequence, the EI should be considered as a determinant of the post-meal AS at each meal time. A negative correlation was evident between the HGI post-lunch AS and the HGI EI_{lunch} ($p = 0.012$), while the LGI post-lunch AS was independent of the LGI EI_{lunch} ($p = 0.13$). Although the absence of such an association after the LGI lunch remained unclear, the increased LGI EI_{snack} appeared to compensate for the reduced LGI EI_{lunch} leading to lack of difference in the total EI between trials.

In their review, McKiernan et al. [34] reported that only one-third of the papers identified showed an association between appetite-related questions and food intake. The authors suggested that hunger was only a weak predictor of EI ($r = 0.3$) and the association between hunger and EI was even weaker under free-living conditions. There is evidently a complex relationship between appetite sensations and dietary intake. In the current study using the AS composited from five appetite-related elements, the post-prandial AS_{AUC} was consistently lower following the HGI foods than the LGI foods in both settings of the laboratory and the free living. Since a counterbalanced design was employed, these significant differences between trials could not be attributed to any effect of trial order.

Unexpectedly, the HGI dinner was reported to be more palatable than the LGI dinner which led the palatability to become a confounding variable in the current study. Breakfast, lunch and snacks in both trials were ready to be consumed; however, participants were required to cook the dinner. Although written cooking instruction was given, it was still difficult to control the palatability under home cooking conditions. Despite higher palatability-related scores following the HGI dinner, participants did not significantly increase their EI. Accordingly, the post-dinner PS of both trials was negatively correlated with the corresponding post-dinner AS, which implies that the more palatable foods suppressed the appetite greater at the fixed meal condition. Warwick et al. [15] previously stated that highly palatable meals rich in CHO might facilitate the subsequent control of EI. However, De Graaf et al. [35] suggested that the effect of the pleasantness of the foods on hunger rating and food intake lasted for 2 h postprandially. No evening snack after dinner was provided in the current study, and thus, any effect of the palatability on subsequent EI is not known. There are different interpretations of the term palatability, and to date, there is no clear and

consistent definition. The influence of palatability on appetite sensation needs clarification. Moreover, the mean difference in the GI values between the LGI and HGI dinners was smallest among the main meals after recalculation from the actual consumption of dinner. Further investigation is needed to establish whether the lack of correlation between the EI_{dinner} and the postprandial AS is due to this confounding effect or insufficient difference in the GI values between trials for this meal.

Stevenson et al. [36] found that an LGI evening meal elicited a higher gut fullness score and lower rating of hunger in the following morning than following an HGI evening meal. Consumption of LGI foods has been proposed to promote weight loss due to elevation of the metabolic rate, as well as the fat oxidation when compared to HGI foods [37]. Raben [38] reviewed the GI effect on EE. In nine out of 15 studies, EE was increased after the consumption of LGI foods. Krog-Mikkelsen et al. [39] speculated that any GI effect on energy balance, if it existed, would result from the change in appetite and EI, but not from EE. However, there was no effect of GI on RMR in the post-trial mornings in the present study. Previous studies investigated the effect of evening meals with different GI values on substrate oxidation and glucose tolerance in the morning after the consumption of the standardized HGI meals [33, 36, 40]. The insignificant difference in the respiratory exchange ratio (RER) in the current study was consistent with the baseline fasting results of these previous studies with male and female participants. No significant difference was found in the RER, fat and CHO oxidation in the fasted state in recreationally active males in another study, whereas the free fatty acid (FFA) concentration was higher in the HGI than in the LGI trial [36]. The FFA concentration was not measured in this study, but there was a trend for elevation of the fat oxidation in the post-trial morning after the consumption of HGI foods on the trial day ($p = 0.069$). Elevation of fat oxidation appears to be desirable for promoting weight loss. Fat oxidation can be elevated from low levels of circulating glucose. Thus, the higher rate of fat oxidation following the HGI evening meals in the longer term (>10 h) may be due to a greater drop in postprandial glucose, even within normal range in the current study. If such speculation was correct, it might suggest that HGI meals would lead to a higher hunger rating in the subsequent morning despite greater suppression of appetite sensation by the HGI foods on the trial day, which perhaps warrants further investigation. Larger sample sizes in the current study as well as in the previous study [36] might help to detect the difference.

Self-reporting of food intake is a subjective measure of EI, and reporting bias does occur as shown when comparing habitual EI measured by doubly labelled water and

self-reported [41]. Goldberg et al. [42] developed a cut-off point for distinguishing underreporting of EI as less than 110 % of estimated basal metabolic rate. Only one participant's reported food intake was below the cut-off point in the current study. Nevertheless, this participant returned some leftovers. de Castro [43, 44] suggested that self-report dietary record is a reliable and valid method as all analyses were performed within subjects. The energy content of test meals was based on the individuals' reported food intakes prior to the first trial. Participants were not required to record food intake on the trial day unless extra foods were taken. This study design therefore could minimize under-reporting of food intake. Furthermore, participants acted as their own controls in this crossover repeated-measure design study. Therefore, any underestimation of daily EI and food provided would affect both trials equally and not confound the comparison between trials.

Another strength of this study was that other than the manipulated characteristic, GI, the test meals were matched for energy; CHO; protein; fats including saturated, monounsaturated and polyunsaturated fats; fibre content and energy density between trials. It should be noted that the actual GI values may be different from the published values if the manufacturing processing conditions or formulas of commercial products were changed over time while the published values have not been kept up to date [45]. We sought to minimize any potential discrepancy by determining GI values in a more consistent way via the selection of natural or minimally processed foods.

In summary, the consumption of the HGI CHO-rich foods facilitates stronger appetite suppression in the early postprandial phase and throughout a day compared to the LGI CHO-rich foods. These apparent satiating effects of HGI foods are applicable to physically active males when HGI foods are consumed frequently over 1 day. Nevertheless, the findings should not be generalized without further empirical evidence. The lack of difference in total EI between trials despite the lower appetite scores in the HGI trial is most likely due to the non-ad libitum experimental setting. Further investigation into the relationships between GI and energy intake and balance by examining the physiological aspects of GI on substrate oxidation, and whether the advantage of HGI foods on appetite suppression persists in an ad libitum environment is necessary to understand the impact of HGI CHO-rich foods on appetite and weight management in active individuals in the longer term.

Acknowledgments We thank the volunteers for participation. All authors have read and approved the final manuscript.

Conflict of interest The authors declare that they have no conflict of interest.

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Appendix B: Physical activity log sheet for study one and two

Physical Activity Log Sheet

Participant ID: _____ (internal use)

Date: _____

Time & Quarter	First	Second	Third	Forth
0:00am				
1:00am				
2:00am				
3:00am				
4:00am				
5:00am				
6:00am				
7:00am				
8:00am				
9:00am				
10:00am				
11:00am				
12:00noon				
1:00pm				
2:00pm				
3:00pm				
4:00pm				
5:00pm				
6:00pm				
7:00pm				
8:00pm				
9:00pm				
10:00pm				
11:00pm				

Example:

Time & Quarter	First	Second	Third	Forth
7:00am	←	Sleeping,	monitors off	→
8:00am	Wake up, monitor on	on and breakfast	Taking bus	→
9:00am	←			
10:00am		Office work		
11:00am	←			→

Appendix C: Eating attitudes test questionnaire

Eating Attitudes Test

Date of Birth (dd/mm/yy): Participant ID:(for internal use)

Please tick once from each question which best represents your views over the last **twelve** months. The questions are designed solely to determine whether you are suitable to participate in the study. Your answers will be treated as strictly confidential.

Questions	Always	Usually	Often	Sometimes	Rarely	Never
1. Enjoy trying new rich foods.	<input type="radio"/>					
2. Take longer than others to eat my meals.	<input type="radio"/>					
3. Display self-control around food.	<input type="radio"/>					
4. Feel uncomfortable after eating sweets.	<input type="radio"/>					
5. Cut my food into small pieces.	<input type="radio"/>					
6. Avoid foods with sugar in them.	<input type="radio"/>					
7. Eat diet foods.	<input type="radio"/>					
8. Particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.).	<input type="radio"/>					
9. Feel that food controls my life.	<input type="radio"/>					
10. Feel that others pressure me to eat.	<input type="radio"/>					
11. Am terrified about being overweight.	<input type="radio"/>					
12. Avoid eating when I am hungry.	<input type="radio"/>					
13. Find myself preoccupied with food.	<input type="radio"/>					
14. Have gone on eating binges where I feel that I may not be able to stop.	<input type="radio"/>					
15. Aware of the calorie content of foods that I eat.	<input type="radio"/>					
16. Feel that others would prefer if I ate more.	<input type="radio"/>					
17. Like my stomach to be empty.	<input type="radio"/>					
18. Feel extremely guilty after eating.	<input type="radio"/>					
19. Am preoccupied with a desire to be thinner.	<input type="radio"/>					
20. Think about burning up calories when I exercise.	<input type="radio"/>					
21. Other people think that I am too thin.	<input type="radio"/>					
22. Am preoccupied with the thought of having fat on my body.	<input type="radio"/>					
23. Give too much time and thought to food.	<input type="radio"/>					
24. Engage in dieting behaviour.	<input type="radio"/>					
25. Have the impulse to vomit after meals.	<input type="radio"/>					
26. Vomit after I have eaten.	<input type="radio"/>					

Appendix D: Integrated Eating Behaviour Questionnaire

Integrated Eating Behaviour Questionnaire

Participant ID: Date of Birth (dd/mm/yy):

Please tick one from each statement that best applies to you in the last **twelve** months.

Question	never	rarely	sometimes	often	very often
1. If you have put on weight, do you eat less than you usually do?	<input type="radio"/>				
2. Do you have the desire to eat when you are irritated?	<input type="radio"/>				
3. Do you try to eat less at mealtimes than you would like to eat?	<input type="radio"/>				
4. Do you have a desire to eat when you have nothing to do?	<input type="radio"/>				
5. How often do you refuse food or drink offered because you are concerned about your weight?	<input type="radio"/>				
6. Do you have a desire to eat when you are depressed or discouraged?	<input type="radio"/>				
7. Do you watch exactly what you eat?	<input type="radio"/>				
8. If food tastes good to you. Do you eat more than usual?	<input type="radio"/>				
9. Do you have a desire to eat when you are feeling lonely?	<input type="radio"/>				
10. Do you deliberately eat foods that are slimming?	<input type="radio"/>				
11. Do you have a desire to eat when somebody lets you down?	<input type="radio"/>				
12. If food smells and looks good, do you eat more than usual?	<input type="radio"/>				
13. When you have eaten too much, do you eat less than usual the following days?	<input type="radio"/>				
14. Do you have a desire to eat when you are cross?	<input type="radio"/>				
15. If you see or smell something delicious, do you have a desire to eat it?	<input type="radio"/>				
16. Do you deliberately eat less in order not to become heavier?	<input type="radio"/>				
17. Do you have a desire to eat when you are approaching something unpleasant to happen?	<input type="radio"/>				
18. If you have something delicious to eat, do you eat it straight away?	<input type="radio"/>				
19. How often do you try not to eat between meals because you are watching your weight?	<input type="radio"/>				
20. Do you get the desire to eat when you are anxious, worried or tense?	<input type="radio"/>				
21. If you walk past the baker to you have the desire to buy something delicious?	<input type="radio"/>				
22. How often in the evening do you try not to eat because you are watching your weight?	<input type="radio"/>				
23. Do you have a desire to eat when things	<input type="radio"/>				

are going against you or when things have gone wrong?					
24. If you see others eating do you also have the desire to eat?	<input type="radio"/>				
25. Do you take into account your weight with what you eat?	<input type="radio"/>				
26. Do you have a desire to eat when you are frightened?	<input type="radio"/>				
27. If you walk past a snack bar or café, do you have the desire to buy something delicious?	<input type="radio"/>				
28. Do you have a desire to eat when you are disappointed?	<input type="radio"/>				
29. Can you resist eating delicious foods?	<input type="radio"/>				
30. Do you have the desire to eat when you are emotionally upset?	<input type="radio"/>				
31. Do you eat more than usual when you see others eating?	<input type="radio"/>				
32. Do you have a desire to eat when you are bored or restless?	<input type="radio"/>				
33. When you are preparing a meal are you inclined to eat something?	<input type="radio"/>				

Question	Definitely true	Mostly true	Mostly false	Definitely false
34. I deliberately choose small helpings to control my weight.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
35. I start to eat when I feel anxious.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
36. Sometimes when I start eating, I just can't seem to stop.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
37. When I feel sad, I often eat too much.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
38. I do not eat some foods because they make me fat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
39. Being with someone who is eating often makes me want to also eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
40. When I feel tense or "wound up", I often feel I need to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
41. I often get so hungry that my stomach feels like a bottomless pit.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
42. I am always so hungry so it is hard for me to stop eating before I finish the food on my plate.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
43. When I feel lonely, I console myself by eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
44. I consciously restrict how much I eat during meals to avoid gaining weight.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
45. When I smell appetizing food or see a delicious dish, I find it very difficult not to eat- even if I have just finished a meal.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
46. I am always hungry enough to eat at any time.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
47. When I feel nervous, I try to calm down by eating.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
48. When I see something that looks very delicious, I often get so hungry that I have to eat right away.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
49. When I feel depressed, I want to eat.	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>

50. How often do you avoid "stocking up" on tempting foods?

Almost never <input type="radio"/>	Seldom <input type="radio"/>	Usually <input type="radio"/>	Almost always <input type="radio"/>
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51. How likely are you to make an effort to eat less than you want?

Unlikely <input type="radio"/>	A little likely <input type="radio"/>	Somewhat likely <input type="radio"/>	Very likely <input type="radio"/>
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52. Do you go on eating binges even though you are not hungry?

Never <input type="radio"/>	Rarely <input type="radio"/>	Sometimes <input type="radio"/>	At least once a week <input type="radio"/>
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53. How often do you feel hungry?

Only at meal times <input type="radio"/>	Sometimes between meals <input type="radio"/>	Often between meals <input type="radio"/>	Almost always <input type="radio"/>
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54. On a scale of 1 to 8, where 1 means no restraint in eating and 8 means total restraint, what number would you give yourself?

Circle the number that best applies to you

1	2	3	4	5	6	7	8
I eat whatever and whenever I want it							I am constantly limiting food intake and never "giving in"

The end of the questionnaire.

Appendix E: Leaflets and flyers for participant recruitment for study one to four



Volunteers Required



Hey gals! We are looking for you!

- Are you a Caucasian and 18 to 45 years old?
- Do you participate in any sports actively and regularly?
- Would you like to know how food intake interacts with your appetite and weight management?
- Would you be willing to spend around two hours in a morning, once a week for two weeks to help us with our research at the St Luke's Campus, Heavitree Road?

If all answers are yes, come and join us!

- ✓ You will be served a breakfast meal at the campus.
- ✓ You will be asked to complete a questionnaire on your appetite sensation during the test day.
- ✓ You will be asked to complete a health questionnaire, a 3-day food record and to wear a heart rate monitor and an accelerometer to measure your activity level.

Contact us if you are interested in joining us or getting more information:

Daphne Wu e-mail: mw328@exeter.ac.uk or Tel: 07551934027

Please note that you may not be suitable if you:

- are pregnant;
- are under 18 years old or over 45 years old of age;
- are a smoker;
- have been diagnosed with any metabolic, menstrual or eating disorder;
- have known of any food allergy or intolerance;
- have taken breakfast less than five times a week in the last month.

Daphne mw328@ex.ac.uk Appetite Study				
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Contact us if you are interested in joining the study or getting more information:

Daphne Wu

PhD Researcher
School of Sport and Health Sciences,
University of Exeter
State Registered Dietitian

E-mail: mw328@exeter.ac.uk

or

Tel: 07551 934027

This project has been reviewed and approved by the Ethics Committee of School of Sport and Health Sciences, University of Exeter.

Flyer V2 DW 060610



Female Volunteers Required

Are you a Caucasian and 18 -45 years old?

Are you physically active at exercise or sports?

Would you like free breakfasts?

Would you like to know how food intake interacts with your appetite and weight management?

What is the study about?

This is an important study for physically active females which aims to investigate whether different breakfast meals lead to differences in appetite sensation and energy metabolism.

Taking part in this study will help you make an informed decision on how food consumption can lessen hunger and promote satiety.



Who is taking part?

We are recruiting female Caucasian volunteers aged 18 to 45 years who participate regularly in sports.

How will taking part in the study benefit me?

- ✓ You will have an opportunity to meet a dietitian graduated from the International Olympic Committee (IOC) Sports Nutrition to find out how nutrition impacts on weight strategies and exercise performance.
- ✓ You will be given the results of your body composition, energy intake and expenditure which you may find useful for setting goals of weight management.

What does the test involve?

- The study involves two 2-hour trials at the St Luke's Campus, University of Exeter, Heavitree Road.
- You will be asked to complete questionnaires on health and eating behaviour.
- You will be served a breakfast after assessment of your body composition.
- You will be asked to record food intakes, to give ratings on your appetite sensation, and to wear activity monitors during the testing period.

Male Volunteers Required

MALE Athletes! We are looking for you!

- Are you a Caucasian and 18 to 45 years old?
- Do you participate in any sports actively and regularly?
- Would you like to get free meals?
- Would you like to know how food consumption interacts with your appetite and weight management?

If all answers are yes, come and join us!

- ✓ You will be served a breakfast meal at the campus and a take-away food box at the trial day.
- ✓ Your body composition will be measured.
- ✓ You will be asked to rate your appetite sensation, to complete food records, questionnaires on health and eating behaviour; to wear a heart rate monitor and an activity monitor (accelerometer).
- ✓ You will be given the results of your body composition, energy intake and expenditure.

The study involves two 2-hour trial visits and two 1-hour post-trial visits at the St. Luke's campus, Heavitree. Contact us if you are interested in joining the study or getting more information:

Daphne Wu e-mail: mw328@exeter.ac.uk or Tel: 07551 934 027



Please note that you may not be suitable if you:

- are under 18 years old or over 45 years old of age;
- are a smoker;
- have been diagnosed with any metabolic or eating disorder;
- have any food allergy or aversion;
- have taken breakfast less than three times a week in the last month.

Daphne mw328@ex.ac.uk Appetite Study				
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Contact us if you are interested in joining the study or getting more information:

Daphne Wu

PhD Researcher
School of Sport and Health Sciences
University of Exeter
State Registered Dietitian

E-mail: mw328@exeter.ac.uk

or

Tel: 07551 934027

This project has been reviewed and approved by the Ethics Committee of School of Sport and Health Sciences, University of Exeter.

Flyer V2 DW 050610



**Male
Volunteers
Required**

Are you a Caucasian and 18 to 45 years old?

Are you physically active at exercise or sports?

Would you like to get free meals?

Would you like to know how food intake interacts with your appetite and weight management?

What is the study about?

This is an important study which aims to investigate whether different meals lead to differences in appetite sensation and energy metabolism.

The study will take place at the St Luke's campus, University of Exeter, Heavitree Road.

Taking part in this study will help you make an informed decision on how food consumption can lessen hunger and promote satiety.

Who is taking part?

We are recruiting male Caucasian volunteers aged 18 to 45 years who participate regularly and actively in sports.

What does the test involve?

- The study involves two 2-hour trial visits and two 1-hour post-test visits.
- You will be asked to complete questionnaires on health and eating behaviour.
- Your body composition will be measured.
- You will be served a breakfast at the campus and then given a take-away food box on the trial day.
- You will be asked to record food intakes, to give ratings on your appetite sensation, and to wear activity monitors.
- You will visit the laboratory the next day after the trial day for a post-test assessment.

How will taking part in the study benefit me?

- ✓ You will be served free meals for two whole days.
- ✓ You will have an opportunity to meet a dietitian graduated from the International Olympics Committee (IOC) Sports Nutrition to find out how nutrition impacts on weight strategies and exercise performance.
- ✓ You will be given the results of your body composition, energy intake and expenditure which you may find useful for setting goals of weight management.

Breakfast and Mental Performance Study **Male Volunteers Required**

- Are you a physically active Caucasian male aged between 18 and 45 years?
- Would you like to get free meals?
- Would you like to know how food affects your mood state and mental performance?

If all answers are yes, come and join us!

- ✓ Two trial breakfasts will be served at campus and two take-away pre-trial evening meals will be provided.
- ✓ You will be asked to complete several computerized psychological tests and to wear a heart rate monitor during the trial after breakfast.
- ✓ Several venous blood samples will be taken.
- ✓ You will be given £20 supermarket coupon (limited availability), the results of your body composition and energy intake after completion of the whole study.

The research involves two trials of around 4 hours each at the St. Luke's campus, Heavitree.

Contact us if you are interested in joining the study or getting more information.

Please note that you may not be suitable if you:

- are under 18 years old or over 45 years old of age;
- are not physically active;
- are a smoker;
- have been diagnosed with any metabolic or eating disorder;
- have any food allergy or aversion;
- are colour blind;
- have taken breakfast less than three times a week in the last month.

Daphne mw328@ex.ac.uk Food & Psychological Study

Contact us if you are interested in joining the project or getting more information:

Daphne Wu

Ph.D. Researcher
State Registered Dietitian
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or

Tel: 07551 934027

This project has been reviewed and approved by the SHS Ethics Committee of The University of Exeter.

Flyer S3V1.4 07062012



Male Volunteers required for Breakfast and Mental Performance Project

Are you a male aged between 18 and 45 years?

Do you exercise regularly?

Would you like to get free meals and supermarket gift card?



What is the project about?

The project involves food consumption, computerized assessment of mental performance; and blood collection for measuring appetite levels.

The aim of the project is to assess how different breakfasts affect appetite, mood state and psychological function.

The project involves two 4-hour trials taking place at the St Luke's campus, University of Exeter, Heavitree Road.

This project is being undertaken as part of the requirements of a Ph.D. in Sport and Health Sciences.

What does the trial involve?

- A visit for familiarization of the computerized psychological tests will be required.
- A take-way evening meal prior to the trial will be provided.
- Body composition will be measured.
- A breakfast will be served at the campus.
- A heart rate monitor and a gas collection mask will be worn during the trial during the psychological tasks and the mood assessment.
- Small samples of blood will be taken.
- A subsequent meal will served after the psychological tasks.

Who is taking part?

We are recruiting male volunteers aged 18 to 45 years who participate regularly and actively in sports.

How will taking part in the project benefit me?

- ✓ You will be served free meals.
- ✓ You will be given the results of your body composition and nutrient intake which you may find useful for setting goals of your own nutrition.
- ✓ You will get £10 supermarket gift card by attending the project plus another up to £10 based on the scores of the psychological tasks.

Breakfast and Exercise Performance Study Male Volunteers Required

- Are you a physically active Caucasian male aged between 18 and 45 years?
- Would you like to get **breakfasts**, results of your body composition, **maximal oxygen uptake (VO₂max)** and **dietary analysis** for **free**?
- Would you like to know how food consumption affects your mood state and running performance?

If all answers are yes, come and join us!

- ✓ You will be asked to complete a running test to exhaustion before the main trial.
- ✓ Two trial breakfasts will be provided at the St. Luke's campus.
- ✓ Several computerised psychological tests and sprint running tests on a treadmill will be required after breakfast with small samples of blood be taken.

Two main trials each of around 3.5 hours and one 1-hour pretrial visit for VO₂max will be taken place at the St. Luke's campus, Heavitree.

Contact us if you are interested in joining the study or getting more information.

Please note that you may not be suitable if you:

- are under 18 years old or over 45 years old of age;
- are a non-Caucasian;
- are a smoker;
- have been diagnosed with any metabolic or eating disorder;
- have any food allergy or aversion;
- are colour blind;
- have taken breakfast less than three times a week in the last month.

Daphne mw328@ex.ac.uk Breakfast, mood & exercise				
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Contact us if you are interested in joining the study or getting more information:

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This project has been reviewed and approved by the SHS Ethics Committee of The University of Exeter.

Flyer S4V2a 12082011



**Male Volunteers
required for
Breakfast, Mood and
Exercise Performance
Study**

Are you a Caucasian male aged between 18 and 45 years?

Do you exercise regularly?

Would you like to get free breakfasts?

Would you like to know how breakfast affects your mood state, cognitive function and exercise performance?

What is the study about?

The study involves food consumption, assessment of psychological performance, intermittent exercise test; and blood collection.

The aim of this study is to assess how differences in breakfast composition affect mood state, psychological function and exercise performance.

The study involves two 3.5-hour trial visits and one 1-hr preliminary test taking place at the St Luke's campus, University of Exeter, Heavitree Road.

This study is being undertaken as part of the requirements of a Ph.D. in Sport and Health Sciences.

What does the test involve?

- The preliminary visit involves a familiarisation of the computerised psychological tasks and a running test to measure the maximal oxygen uptake.
- Body composition will be measured.
- A breakfast will be served.
- An intermittent running test alternating with psychological assessment will be performed; with a heart rate monitor being worn during the trial.
- Small samples of blood will be taken.
- Food record, questionnaires on health and eating attitude will be completed.

Who is taking part?

We are recruiting male Caucasian volunteers aged 18 to 45 years who participate regularly and actively in sports.

How will taking part in the study benefit me?

- ✓ You will be served two free breakfasts.
- ✓ You will be given the results of your body composition, nutrient intake and maximal oxygen uptake.
- ✓ You will meet a dietitian graduated from the International Olympic Committee (IOC) Sports Nutrition to know how nutrition impacts on mood state, psychological and running performance.

Appendix F: Information sheets for study one to four



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Effect of breakfast on appetite sensation and subsequent food intake on female athletes INFORMATION SHEET FOR PARTICIPANT

Caucasian females aged between 18-45 years with regular physical training interested in knowing more about weight management are invited to take part in a study about the relationship between food consumption, appetite and metabolism.

Thank you for showing an interest in participating in the study. Please read this information sheet carefully before deciding whether to participate. If you decide to volunteer we thank you for your participation. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the aim of the project?

The purpose of the study is to investigate whether different breakfast meals lead to different appetite sensation and energy metabolism. We understand that eating is not just for nutrients and energy, it also links to personal emotions, comfort and exercise performance. Taking part in this study will help you make informed decision on how food consumption can lessen hunger and promote satiety. This study is being undertaken as part of the requirements of a Ph.D. degree in School of Sport and Health Sciences, University of Exeter.

What type of participants are needed?

We are seeking for female Caucasian (white) volunteers aged 18 to 45 years participating regularly in sports at least 30 minutes and three days per week.

What will participants be asked to do?

If you agree to participate in this project, you will be asked to attend two morning sessions at St. Luke's Campus, each will last not more than two hours. The trial days follow into follicular phase of menstrual cycle (i.e. <10 days of end of menstruation). Thus the first trial will be arranged on around 4-6th day of your menses.

Protocol

You will be required to record three breakfasts consecutively with food photos being taken a month prior to the first trial so that we can prepare the appropriate portion and choices of breakfast. You will complete an eating behaviour questionnaire within one week after the cessation of the menses.

You will be required to record the food intake with food photo taking, carry a heart rate monitor and an accelerometer two days before the trial visit.

On arrival of the trial visit between 7am and 9am after an overnight fasting, you will be briefed the testing procedure. Following this, your body height, weight and body composition will be measured. You will be then served a glass of water and will have a rest of 15 minutes to get ready for the meal. You will then be required to finish the meal within 20 minutes. You will be asked to rate your appetite and physical comfort on a questionnaire before and after breakfast for one hour.

You are free to take your lunch and dinner between 12 noon and 2 p.m.; and 6 p.m. and 8 p.m. respectively. You will record all food intakes with food photo. You will also rate your appetite and physical comfort hourly during the waking hour of the trial day after your leave. You will wear the heart rate monitor and the accelerometer during the waking hours of the trial day.

You will come back to the laboratory and repeat the trial again in one week.

Summary of testing procedures

Pre-trial Preparation - One week after the cessation of the menses (first menses)

Consent form

Three breakfasts with food photo

Eating behaviour questionnaire

Two days before your first trial visit

Two-day food diary

Heart rate monitor and accelerometer, physical activity log

First Trial Visit – Forth to sixth day of the next menses (second menses)

Height, weight and body composition measurement

Test meal

Rating appetite sensation and physical comfort

Food diary with the food photo, exclusive of breakfast

Heart rate monitor and accelerometer, physical activity log

Two days before your second trial visit

Two-day food diary

Heart rate monitor and accelerometer, physical activity log

Second Trial Visit – One week after the first trial

Weight and body composition measurement

Test meal

Rating appetite sensation and physical comfort

Food diary with the food photo, exclusive of breakfast

Heart rate monitor and accelerometer, physical activity log

What are the possible risks of taking part in the study?

There are no risks additional to those usually associated with food consumption. There may be naturally some discomfort such as hunger and bloating in some individuals. Food will be prepared by a registered dietitian who has been trained in food hygiene.

As we are only looking to recruit participants who are free from food allergy, all risks and safety points associated with food consumption have been covered.

What if you decide you want to withdraw from the project?

You may decide not to take part in the project at any time you wish, then you can. It is entirely up to you and there will be no disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not used either now or in the future. Please be aware that any data having been published at the time you requested cannot be withdrawn.

What will happen to the data and information collected?

All information collected and results will be held securely at the University of Exeter and will only be accessible to relevant University staff. The University will securely hold the data recorded for a period of five years. Following this it will be destroyed. Results of this project may be published, but any data included will in no way be linked to any specific participant. Your anonymity will be preserved. You are most welcome to request a copy of the results of the project/copy of any transcripts etc. should you wish.

What if I have any questions, worries or complaints?

If you have any questions about this project, please feel free to contact me by email or phone. If you feel your treatment either prior to, during or after the study is of your concern in any way, please contact my supervisors Associate Professor Craig Williams on 01392 264890 or Senior Lecturer Dr. Ann Rowlands on 01392 722878 during office hours.

Should you like to participate in this project, please complete the consent form prior to the beginning of the study.



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This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences

Effect of Meals on appetite sensation and metabolism on male athletes

INFORMATION SHEET FOR PARTICIPANT

Male Caucasian adults with regular physical activities interesting in knowing more about weight management are invited to take part in a study about the relationship between food consumption, appetite and metabolism.

Thank you for showing an interest in participating in the study. Please read this information sheet carefully before deciding whether to participate. If you decide to volunteer we thank you for your participation. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the aim of the project?

The purpose of the study is to investigate whether different meals lead to different appetite sensation and energy metabolism. We understand that eating is not just for nutrients and energy, it also links to personal emotions, comfort and exercise performance. Taking part in this study will help you make informed decision on how food consumption can lessen hunger and promote satiety. This study is being undertaken as part of the requirements of a Ph.D. in Sport and Health Sciences.

What type of participants are needed?

We are seeking for male Caucasian (white) volunteers aged 18 to 45 years participating regularly in sports at least 30 minutes and three days per week.

What will participants be asked to do?

The tests involve food consumption. If you agree to participate in this project, you will be asked to attend four morning sessions for two trials at St. Luke's Campus of University of Exeter at Heavitree. Each main trial lasts around two hours following a one-hour post-trial session.

Protocol

You will be required to record the food intake with food photo taking, wear a heart rate monitor and an accelerometer (physical activity monitor) two days before the trial visit.

On arrival of the trial visit between 7am and 9am after an overnight fasting, you will be briefed the testing procedure. Following this, your resting energy expenditures, body height and weight; and body composition (body fat and muscle mass) will be measured. A test meal will then be provided. You will be required to finish the meal within 20 minutes. You will be asked to rate your appetite and physical comfort on a questionnaire before and after breakfast for one hour.

You will provide a food bag when you leave the laboratory. You will be required to consume only the food provided and to take your lunch and dinner between 12 noon and 2 p.m.; and 6 p.m. and 8 p.m. respectively. You are free to eat the snacks provided anytime. You will rate your appetite and physical comfort hourly during the waking hour of the rest of the trial day. You will continue to wear the heart rate monitor and the accelerometer during the trial day.

You will visit the laboratory the next day morning to assess your resting energy expenditures and body composition. You will continue to record your food intake; and wear the heart rate monitor and the accelerometer this day, i.e. the day after the trial day.

You will be asked to come back to the laboratory and repeat the whole trial in one week apart.

Summary of testing procedures of a trial

Two days before the trial visit

- Food diary with food photos
- Heart rate monitor and accelerometer

The trial visit

- Anthropometric and resting energy expenditure measurement
- Test meal
- Questionnaire of appetite sensation, palatability and physical comfort
- Heart rate monitor and accelerometer
- Food box provided for the whole day

The day after the trial visit

- Anthropometric and Resting energy expenditure measurement
 - Food diary with food photos
 - Heart rate monitor and accelerometer
-

What are the possible risks of taking part in the study?

There are no risks additional to those usually associated with food consumption. There may be naturally some discomfort such as hunger and bloating in some individuals. Food will be prepared by a registered dietitian who has been trained in food hygiene.

As we are only looking to recruit participants who are free from food allergy and aversion, all risks and safety points associated with food consumption have been covered.

What if you decide you want to withdraw from the project?

You may decide not to take part in the project at any time you wish, then you can. It is entirely up to you and there will be no disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not used either now or in the future. Please be aware that any data having been published at the time you requested cannot be withdrawn.

What will happen to the data and information collected?

All information collected and results will be held securely at the University of Exeter and will only be accessible to relevant University staff. The University will securely hold the data recorded for a period of five years. Following this it will be destroyed. Results of this project may be published, but any data included will in no way be linked to any specific participant. Your anonymity will be preserved. You are most welcome to request a copy of the results of the project/copy of any transcripts etc. should you wish.

What if I have any questions, worries or complaints?

If you have any questions about this project, please feel free to contact me either by email or by phone. If you feel your treatment either prior to, during or after the study is of your concern in any way, please contact my supervisors Associate Professor Craig Williams on 01392 264890 or Senior Lecturer Dr. Ann Rowland on 01392 722878 during office hours.

Should you like to participate in this project, please complete the attached consent form prior to the beginning of the study.



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This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences

Acute effect of breakfasts on appetite, mood and mental performance to adult athletes

INFORMATION SHEET FOR PARTICIPANT

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the aim of the project?

The purpose of the project is to investigate whether different meals lead to different levels of appetite, mood state and mental performance. Taking part in this study will help you make informed decision on how food consumption can affect psychological test performance and mood state. This project is being undertaken as part of the requirements of a Ph.D. in Sport and Health Sciences.

What types of participants are needed?

We are seeking for male volunteers aged 18 to 45 years who participate exercise with moderate to high intensity at least 30 minutes and three days per week. You may not be suitable to participate in the study if you:

- are under 18 years old or over 45 years old of age;
- are a smoker;
- do not have regular exercise;
- have been diagnosed with any metabolic or eating disorder;
- have any food allergy or aversion;
- are colour blind; or
- have taken breakfast less than three times a week in the last month.

What will participants be asked to do?

The project involves food consumption, computerized psychological tests and blood collection. If you agree to participate in this project, you will be asked to attend two morning sessions for two trials at the St. Luke's Campus of the University of Exeter at Heavitree. Each main trial lasts around four hours. You will be asked to attend a familiarization trial for the computerized psychological tests and to record the food intake prior to each trial. An evening meal will be provided prior to each trial day.

Summary of testing procedures of a trial

Prior to the trial visit

- Food diary
- Familiarization to the psychological tests
- Standardized evening meal

The trial visit

- Anthropometric and mood state measurement
 - Test meal
 - Psychological tests with monitoring of heart rate and breathing
 - Capillary blood sampling
 - Second test meal
-

Protocol

After overnight fasting and on arrival at the laboratory between 7am and 9am, body height and weight and body composition (body fat and muscle mass) will be measured. Mood state will then be assessed by a questionnaire. A capillary blood will be taken from your fingertip. A test breakfast will then be provided. You will be required to complete three sessions of psychological tests (30 minutes) with a heart rate monitor and a gas collection mask being worn. Capillary blood samples will be taken before and after each session. Mood state will be assessed before the start and after the last set of psychological test.

After the last psychological test, you will be seated for 30 minutes until the second meal was served. You will be required to consume as much as you can until you feel very full.

What are the possible risks of taking part in the study?

Taking blood from the finger tip might lead to small area of bruising. There are no risks additional to those usually associated with food consumption. There may be some discomfort such as hunger and bloating in some individuals. Food will be prepared by a registered dietitian who has been trained in food hygiene. As we are only looking to recruit participants who are free from food allergy and aversion, all risks and safety points associated with food consumption will be covered.

What if you decide you want to withdraw from the project?

You may decide not to take part in the project at any time. It is entirely up to you and there will be no disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not used either now or in the future. Please be aware that any data having been analyzed or published at the time you requested cannot be withdrawn.

What will happen to the data and information collected?

All information collected and results will be held securely at the University of Exeter and will only be accessible to relevant University staff. The University will securely hold the data recorded for a period of five years. Following this it will be destroyed. Results of this project may be published, but any data included will in no way be linked to any specific participant. Your anonymity will be preserved. You are most welcome to request a copy of the results of the project/copy of any transcripts etc. should you wish.

What if I have any questions, worries or complaints?

If you have any questions about this project, please feel free to contact me either by email or by phone. If you feel your treatment either prior to, during or after the study is of your concern in any way, please contact my supervisor Associate Professor Craig Williams on 01392 724890 or Associate Professor Joanna Bowtell on 01392 722869 during office hours.

Should you like to participate in this project, please complete the consent form prior to the beginning of the project.



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This project has been reviewed and approved by the SHS Ethics Committee.

Physiological and cognitive performances of adult athletes after consumption of a breakfast

INFORMATION SHEET FOR PARTICIPANTS

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether to participate. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the aim of the project?

The purpose of the study is to investigate whether different breakfasts lead to different levels of mood states, and cognitive and physiological performances during an intermittent exercise event. Taking part in this study will help you make informed decisions on how food consumption can affect exercise performance. This study is being undertaken as part of the requirements of a PhD in Sport and Health Sciences.

What types of participants are needed?

We are seeking male Caucasian (white) volunteers aged 18 to 45 years who participate regularly in decision making sports (e.g. team sports, combat sports and racket sports) for at least 30 minutes on three days per week. You may not be suitable to participate in the study if you:

- are under 18 years old or over 45 years old of age;
- are a non-Caucasian;
- are a smoker;
- have been diagnosed with any metabolic or eating disorder;
- have any food allergy or aversion;
- are colour blind; or
- have taken breakfast less than three times a week in the last month.

What will participants be asked to do?

The trials involve food consumption, psychological tests, an intermittent running test and blood collection. If you agree to participate in this project, you will be asked to attend two morning sessions for two trials at St. Luke's Campus, University of Exeter. Each trial will last approximately 3.5 hours. You will be asked to report your food intake for one day prior to each trial. You will visit the laboratory, prior to the first trial, to get familiar with the computerized psychological tasks and to complete a maximal oxygen uptake ($\dot{V}O_2\text{max}$) test. The $\dot{V}O_2\text{max}$ test will require you to run at different levels of intensity until feeling exhausted which will last for around 30 minutes.

Testing procedures of the main trial

Prior to the trial visit

Familiarization test for the cognitive tasks

Running to exhaustion

Food diary

The trial visit

Anthropometric and mood state measurement

Test breakfast

Protocol

After an overnight fast on arrival to the laboratory between 7am and 9am, body height and weight; and body composition (body fat and muscle mass) will be measured. Mood state will then be assessed by a questionnaire. A small venous blood sample of 10 millilitres (this is about 2 teaspoons) from a vein just below the elbow and a capillary blood sample from the fingertip will be taken before the breakfast. Afterwards, you will ingest the breakfast over a period of 20 minutes. Venous blood and capillary blood will be taken again 30 and 60 minutes following the breakfast. You will be required to walk leisurely on a treadmill or sit for rest during this period. Following a warm up, you will then take three 30-minute sessions of intermittent exercise test on a treadmill. Venous and capillary blood sample will be taken after every session. Mood state will be assessed again before and after the run. The procedure will then be repeated one week later.

What are the possible risks of taking part in the study?

Taking blood from a vein can be uncomfortable and typically a sharp scratch is felt as the needle is inserted into the arm. Blood will be taken by trained staff, all risks and safety points associated with blood collection will have been covered.

There are no risks additional to those usually associated with food consumption. There may be naturally some discomfort such as hunger and bloating in some individuals. Food will be prepared by a registered dietitian who has been trained in food hygiene.

As we are only looking to recruit participants who are free from food allergy and aversion, all risks and safety points associated with food consumption have been covered.

What if you decide you want to withdraw from the project?

You may decide not to take part in the project at any time you wish. It is entirely up to you and there will be no disadvantage to yourself of any kind. You may also request that any information collected from you to date be destroyed or deleted and not used either now or in the future. Please be aware that any data having been analyzed or published at the time you requested cannot be withdrawn.

What will happen to the data and information collected?

All information collected and results will be held securely at the University of Exeter and will only be accessible to relevant University staff. The University will securely hold the data recorded for a period of five years. Following this it will be destroyed. Results of this project may be published, but any data included will in no way be linked to any specific participant. Your anonymity will be preserved. You are most welcome to request a copy of the results of the project/copy of any transcripts etc. should you wish.

What if I have any questions, worries or complaints?

If you have any questions about this project, please feel free to contact me either by email or by phone. If you feel your treatment either prior to, during or after the study is of your concern in any way, please contact my supervisor Associate Professor Craig Williams on 01392 724890 during office hours.

Should you like to participate in this project, please complete the attached consent form prior to the beginning of the study.



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Appendix G: General health questionnaires



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SSHS General Health Questionnaire (Female)

Name: Date of Birth (dd/mm/yy):

Body Height:(in cm)Current Weight: (kg / lb / stone)

Address:
.....

Phone No.:Email:

Types of Sport, duration and frequency:

Please read the following carefully and answer as accurately as possible. The questions are designed solely to determine whether you are suitable to participate in the study. Your answers will be treated as strictly confidential. If you have any doubts or difficulties with any of the questions please contact the person responsible for the study.

	Medical History:	YES	NO
1.	Has a doctor ever said you have diabetes or metabolic disorders? If yes, please specify.....		
2.	Are you currently taking any prescribed medications? If yes, please specify.....		
3.	Do you have been told of any other diseases which last more than 6 months? If yes, please specify.....		
4.	Has a doctor ever said you have food allergy? If yes, please specify.....		
5.	Do you ever feel faint or have spells of dizziness?		
6.	Do you have any physical disability?		
7.	Are you pregnant?		

	Lifestyles:	YES	NO
8.	Do you have taken breakfast five times or more a week in the last month?		
9.	Do you get more than 30 minutes of physical activity on at least 3 days per week in the last three months?		
10.	Were your body weight stable (within 10% change) in the last 12 months? If no, the highest wasand the lowest was(kg / lb / stone)		
11.	Do you smoke, or have you quit smoking in the last six months?		
12.	Are you vegetarian? If yes, the type of vegetarian isand foryears		
13.	Has there been any time when your periods have stopped for a time of more than six months? If yes, please state the duration.....		

I have completed the questionnaire to the best of my knowledge and any questions that I have raised have been answered to my full satisfaction.

Signed: Date:



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General Health Questionnaire (Male)

Name: Date of Birth (dd/mm/yy):
 Body Height:(in cm)Current Weight: (kg / lb / stone)
 Address:
 Phone No.: Email:

Types of Sport, duration and frequency:

Please read the following carefully and answer as accurately as possible. The questions are designed solely to determine whether you are suitable to participate in the study. Your answers will be treated as strictly confidential. If you have any doubts or difficulties with any of the questions please contact the person responsible for the study.

Medical History:		YES	NO
1.	Have you seen your doctor in the last 6 months? If yes, please specify.....		
2.	Has a doctor ever said you have diabetes or metabolic disorders? If yes, please specify.....		
3.	Are you currently taking any prescribed medications? If yes, please specify.....		
4.	Do you have been told of any other diseases which last more than 6 months? If yes, please specify.....		
5.	Has a doctor ever said you have food allergy or do you have any food aversion? If yes, please specify.....		
6.	Do you ever feel faint or have spells of dizziness?		
7.	Do you have any physical disability?		
8.	Lifestyles:	YES	NO
9.	Do you have taken breakfast at least three times a week in the last month?		
10.	Do you get more than 30 minutes of physical activity on at least 3 days per week in the last three months?		
11.	Were your body weight stable (within 10% change) in the last 12 months? If no, the highest wasand the lowest was(kg / lb / stone)		
12.	Do you smoke, or have you quitted smoking in the last six months?		
13.	Are you vegetarian? If yes, the type of vegetarian isand foryears		

I have completed the questionnaire to the best of my knowledge and any questions that I have raised have been answered to my full satisfaction.

Signed: Date:

Appendix H: Consent forms for study one to four



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EFFECT OF BREAKFAST ON APPETITE SENSATION AND METABOLISM ON FEMALE ATHLETES CONSENT FORM FOR PARTICIPANT

NAME:.....(Participant)

I have read the Information Sheet concerning this project and understand what it is about. All my further questions have been answered to my satisfaction. I understand that I am free to request further information at any stage. I know that:

1. my participation in the project is entirely voluntary;
2. I am free to withdraw from the project at any time without having to give out a reason, without disadvantage or prejudice.
3. the data will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for a period of five years from collection;
4. I will be required to attend two sessions to complete the project. As part of the study I will have to :
 - consume two breakfast meals at two occasions;
 - provide food records with food photos; and
 - wear a heart rate monitor and an accelerometer for three consecutive days – the two days prior to the trial day and the trial day;
5. I am aware of any risks that may be involved with the project; and
6. the results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

..... (Signature of participant and Date)
..... (Signature of Witness and Date)

This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences



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EFFECT OF MEALS ON APPETITE SENSATION AND METABOLISM ON MALE ATHLETES

CONSENT FORM FOR PARTICIPANT

NAME:.....(Participant)

I have read the Information Sheet concerning this project and understand what it is about. All my further questions have been answered to my satisfaction. I understand that I am free to request further information at any stage. I know that:

1. my participation in the project is entirely voluntary;
2. I am free to withdraw from the project at any time without having to give out a reason, without disadvantage or prejudice;
3. the data will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for a period of five years from collection;
4. I will be required to attend four sessions to complete the project. As part of the study I will have to:
 - consume two breakfast meals with a week apart;
 - consume the food at the food box provided for the rest of the trial day;
 - provide food records with the food photo; and
 - wear a heart rate monitor for four consecutive days – two days before and one day after the trial day; the trial day;
 - visit the laboratory the next day of the trial day;
5. I am aware of any risks that may be involved with the project; and
6. the results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

..... (Signature of participant and Date)

..... (Signature of witness and Date)

This project has been reviewed and approved by the Ethics Committee of the School of Sport and Health Sciences



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**ACUTE EFFECT OF BREAKFASTS ON APPETITE, MOOD AND
MENTAL PERFORMANCE TO ADULT ATHLETES
CONSENT FORM FOR PARTICIPANTS**

NAME:.....(Participant)

I have read the Information Sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage. I know that:

1. my participation in the project is entirely voluntary;
2. I am free to withdraw from the project at any time without having to give a reason and without disadvantage or prejudice;
3. the data will be destroyed at the conclusion of the project, but any raw data on which the results of the project depend will be retained in secure storage for a period of five years from collection;
4. I will be required to attend three sessions to complete the project. As part of the study:
 - I will visit the laboratory once for familiarisation of the computerised psychological tests;
 - I will record food intakes prior to each main trial;
 - I will visit the laboratory twice for the main trial, a week apart;
 - I will consume an evening meals prior to, a test breakfast and a test lunch at each trial;
 - I will complete a battery of psychological tests at assigned time slots, wear a heart rate monitor and a gas collection mask during the trial; and;
 - ten capillary blood samplings will be taken.
5. I am aware of any risks that may be involved with the project;
6. the results of the project may be published but my anonymity will be preserved; and
7. the amount of honorary incentive will be based on my performance in the project (up to £20).

I agree to take part in this project.

..... (Signature of participant and Date)

This project has been reviewed and approved by the SHS Ethics Committee.



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**ACUTE EFFECT OF BREAKFASTS ON APPETITE, MOOD AND
MENTAL PERFORMANCE TO ADULT ATHLETES**

RECEIPT

NAME:.....(Participant)

I hereby confirm that I received _____ cash / supermarket gift
card after the completion of the above mentioned project.

..... (Signature of participant and Date)

This project has been reviewed and approved by the SHS Ethics Committee.



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PHYSIOLOGICAL AND EXERCISE PERFORMANCES OF ADULT ATHLETES AFTER CONSUMPTION OF BREAKFAST

CONSENT FORM FOR PARTICIPANTS

NAME:.....(Participant)

I have read the Information Sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage. I know that:

1. my participation in the project is entirely voluntary;
2. I am free to withdraw from the project at any time without having to give a reason and without disadvantage or prejudice;
3. the data will be destroyed at the conclusion of the project, but any raw data on which the results of the project depend will be retained in secure storage for a period of five years from collection;
4. I will be required to attend three sessions to complete the project. As part of the study:
 - I will provide food records prior to each trial;
 - I will visit the laboratory to take a psychological task for familiarisation and a running testing to exhaustion for the measurement of maximal oxygen uptake, prior to the first trial;
 - I will consume two test breakfasts, a week apart;
 - I will complete a running test alternating with a battery of psychological tasks; and wear a heart rate monitor during each trial; and;
 - approximately 12 teaspoons (2 teaspoons x 6 times) of venous and capillary blood will be taken on each trial day.
5. I am aware of any risks that may be involved with the project; and
6. the results of the project may be published but my anonymity will be preserved.

I agree to take part in this project.

..... (Signature of participant and Date)

This project has been reviewed and approved by the SHS Ethics Committee.

Appendix I: Food acceptability check lists for study one to four

Food Acceptability Check List (Study one)

Date of Birth (dd/mm/yy):

Date of completion:.....

Please tick one from each question of your acceptability of food. You are free to tick same item both cold and hot if you wish.

Cold Food Item	Yes	No	Cold Food Item	Yes	No	Hot Food Item	Yes	No
Cereals and grains			Milk and other dairy products			Cereals and grains		
• plain oat porridge	<input type="radio"/>	<input type="radio"/>	• milk – regular full fat	<input type="radio"/>	<input type="radio"/>	• plain oat porridge	<input type="radio"/>	<input type="radio"/>
• sweet oat porridge	<input type="radio"/>	<input type="radio"/>	• milk – skimmed (0%)	<input type="radio"/>	<input type="radio"/>	• sweet oat porridge	<input type="radio"/>	<input type="radio"/>
• oat porridge with milk	<input type="radio"/>	<input type="radio"/>	• chocolate milk	<input type="radio"/>	<input type="radio"/>	• oat porridge with milk	<input type="radio"/>	<input type="radio"/>
• sweet oat porridge with milk	<input type="radio"/>	<input type="radio"/>	• Yogurt – regular	<input type="radio"/>	<input type="radio"/>	• sweet oat porridge with milk	<input type="radio"/>	<input type="radio"/>
• weetabix	<input type="radio"/>	<input type="radio"/>	• Yogurt – non fat/skimmed	<input type="radio"/>	<input type="radio"/>	• muesli	<input type="radio"/>	<input type="radio"/>
• cheerios	<input type="radio"/>	<input type="radio"/>	• Cheese	<input type="radio"/>	<input type="radio"/>	• muesli with milk	<input type="radio"/>	<input type="radio"/>
• corn flake	<input type="radio"/>	<input type="radio"/>	• Rice milk	<input type="radio"/>	<input type="radio"/>	• toasted white bread	<input type="radio"/>	<input type="radio"/>
• bran flake	<input type="radio"/>	<input type="radio"/>	Others			• toasted wholegrain bread	<input type="radio"/>	<input type="radio"/>
• muesli	<input type="radio"/>	<input type="radio"/>	• baked bean	<input type="radio"/>	<input type="radio"/>	Milk and other dairy products		
• muesli with milk	<input type="radio"/>	<input type="radio"/>	• marmalade	<input type="radio"/>	<input type="radio"/>	• milk – regular full fat	<input type="radio"/>	<input type="radio"/>
• white bread	<input type="radio"/>	<input type="radio"/>	• jam	<input type="radio"/>	<input type="radio"/>	• milk – skimmed (0%)	<input type="radio"/>	<input type="radio"/>
• wholegrain bread	<input type="radio"/>	<input type="radio"/>	• butter	<input type="radio"/>	<input type="radio"/>	• chocolate milk	<input type="radio"/>	<input type="radio"/>
Fruits and juices			• margarine	<input type="radio"/>	<input type="radio"/>	• Rice milk	<input type="radio"/>	<input type="radio"/>
• apple	<input type="radio"/>	<input type="radio"/>	• ketchup	<input type="radio"/>	<input type="radio"/>	Others		
• pear	<input type="radio"/>	<input type="radio"/>	• sugar	<input type="radio"/>	<input type="radio"/>	• baked bean	<input type="radio"/>	<input type="radio"/>
• banana	<input type="radio"/>	<input type="radio"/>	• honey	<input type="radio"/>	<input type="radio"/>			
• grapefruit	<input type="radio"/>	<input type="radio"/>	• egg	<input type="radio"/>	<input type="radio"/>			
• tomato	<input type="radio"/>	<input type="radio"/>	• nuts	<input type="radio"/>	<input type="radio"/>			
• fruit juice / drink	<input type="radio"/>	<input type="radio"/>						
• dried apricot	<input type="radio"/>	<input type="radio"/>						

Food Acceptability Check List (Study two)

Date of Birth (dd/mm/yy):

Date of completion:.....

Please complete the following food preference questionnaire.

Food Item	Dislike Very Much	Dislike a bit	Neither Like nor Dislike	Like	Like Very Much	Remarks
Cereals and grains						
• Biscuits/ cookies (digestives, tea biscuit, etc.)	o	o	o	o	o	
• Bran flakes	o	o	o	o	o	
• Cereal bar	o	o	o	o	o	
• Cheerios	o	o	o	o	o	
• Corn flakes	o	o	o	o	o	
• Crackers (cream cracker, rice cake, rice cracker, etc.)	o	o	o	o	o	
• Muesli	o	o	o	o	o	
• Pasta (wholegrain/ white)	o	o	o	o	o	
• Porridge	o	o	o	o	o	
• Rice (white / brown)	o	o	o	o	o	
• Tortillas	o	o	o	o	o	
• Weetabix	o	o	o	o	o	
• White bread (sliced / pita)	o	o	o	o	o	
• Wholegrain bread (sliced / pita)	o	o	o	o	o	
Fruits and fruit juices						
• Apple	o	o	o	o	o	
• Banana	o	o	o	o	o	
• Canned fruits (pineapple, lychee, peach, etc.)	o	o	o	o	o	
• Citrus (grapefruit / orange.)	o	o	o	o	o	
• Dried fruits (apricot, raisin, dates, etc.)	o	o	o	o	o	
• Fruit juice/ drinks	o	o	o	o	o	
• Pear	o	o	o	o	o	
Milk and Diaries						
• Cheeses (soft / hard)	o	o	o	o	o	
• Milk (skimmed / semi-skimmed)	o	o	o	o	o	
• Rice milk	o	o	o	o	o	
• Yoghurt (plain / flavoured)	o	o	o	o	o	

Food Item	Dislike Very Much	Dislike a bit	Neither Like nor Dislike	Like	Like Very Much	Remarks
Vegetables						
• Corn (canned)	0	0	0	0	0	
• Green (spinach, lettuce, etc.)	0	0	0	0	0	
• Mushrooms	0	0	0	0	0	
• Onions	0	0	0	0	0	
• Potatoes (canned / mashed)	0	0	0	0	0	
• Sweet peppers	0	0	0	0	0	
• Tomatoes (canned / fresh)	0	0	0	0	0	
• Vegetable juices	0	0	0	0	0	
Meat, poultry and alternatives						
• Baked beans	0	0	0	0	0	
• Boiled Eggs	0	0	0	0	0	
• Canned tuna (in oil / brine)	0	0	0	0	0	
• Beef (roast/ corned)	0	0	0	0	0	
• Other canned beans (red kidney, chick peas, etc.)	0	0	0	0	0	
• Nuts and nut butter (hazelnut, peanuts, almonds, cashews, etc.)	0	0	0	0	0	
• Pork (ham/ bacon)	0	0	0	0	0	
• Poultry (chicken/ turkey)	0	0	0	0	0	
Others						
• Butter / margarine	0	0	0	0	0	
• Chocolate	0	0	0	0	0	
• Crisps (potato / tortilla)	0	0	0	0	0	
• Dressings / condiments (mayonnaise, vinegar, ketchup, etc.)	0	0	0	0	0	
• Diet drinks	0	0	0	0	0	
• Jam / marmalade	0	0	0	0	0	
• Sugar / honey	0	0	0	0	0	
• Sugary / sports drinks	0	0	0	0	0	
• Olive oil	0	0	0	0	0	

The list may not fully recover your food preferences and dislikes. Please specify any foods not shown on the list that you definitely do not wish to be served.

Food Acceptability Check List (Study three and four)

Date of Birth (dd/mm/yy):

Date of completion:.....

Please tick as many as you can accept the foods listed below (Do not leave blank).

Cereals and grains	Yes	No
• cheerios	0	0
• corn flake	0	0
• bran flake	0	0
• muesli	0	0
• muesli with milk	0	0
• wholegrain bread	0	0
Fruits and juices		
• apple	0	0
• banana	0	0
• tomato	0	0
• fruit juice / drink	0	0
• dried apricot	0	0
Milk and other dairy products		
• milk – low fat	0	0
• Yogurt – low fat plain	0	0
• Yogurt – low fat flavoured	0	0
• Cheese	0	0
• Rice milk	0	0
Others		
• ready meal	0	0
• mashed potato	0	0
• pasta	0	0
• rice	0	0
• sugar	0	0
• honey	0	0
• nuts	0	0
• curry chicken	0	0
• gravy / sauces	0	0

Appendix J: Food Record sheets

INSTRUCTIONS FOR COMPLETING THE FOOD RECORD

Thank you very much for providing us your food records. Please read the followings which help you to complete the food record before turning the page.

- Record everything you ate and drank, not served, as detail as you can.
- Do remember to include any snacks and drinks, when being taken e.g. tap water, milk, cheese stick, soup....
- Write down the amount and brand name (except the fresh produce) that you might find the information from the packing.
- Estimate the portion size if you are not sure - a guess is better than leaving a blank!
- Record right after food and drink was taken rather than wait until the end of day that you might miss some items.
- When you finished the record, please check if you have completed the followings:
 - ✓ the date you recorded the intake, the day as well as if it was a school or working day;
 - ✓ the meal time, e.g. 7am, 10:30am, 12:45pm ...;
 - ✓ where you took the food or drinks, e.g. home, school, office, other place...;
 - ✓ the brand name of the foods/drinks, the cooking method if possible, e.g. if you ate an egg, did you write down it was scrambled, fried or boiled...? If you drank a glass of milk, did you write down it was full fat, skimmed or semi-skimmed....?
 - ✓ the portion with *number for quantity* and *unit for size* such as 100 gram / 12-ounce / 2 slices / 1/2 tablespoon / 1 plate / 2 pieces / 4 sets ... for solid food; and 100 mL / 1 pint / 3/4 cup for liquid respectively;
- Take photos of the foods and drink before consumption if possible.
- Specify the type and the duration of any planned exercise if you have done on the record day.
- Feel free to attach a recipe or food label to improve the accuracy.

Food Record

Meal	Time	Place	Food and Drink	Brand Name	Portion
<i>Was it a breakfast, lunch, dinner or snack?</i>		<i>Where did you eat? Home (H), school (S), or other place (O)</i>	<i>Describe the cooking method, e.g. frozen, fresh, boiled, fried.... List all ingredients in dishes, soups, sandwiches, etc. whenever possible.</i>		<i>Quantity and size being eaten, not served</i>

Ruler (each cell in 1 cm) that might facilitate you to measure the size of foods.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
---	---	---	---	---	---	---	---	---	----	----	----	----	----	----	----	----	----	----	----

Have you done any planned exercise today? No / Yes (please circle) I did (type of exercise) for (time in minutes).

Thank you for completing the records! Please take a moment to check that you have completed it as detail as you can.

Appendix K: Visual Analogue Scales

Pre-meal Visual Analogue Scales:

Participant ID: _____

Date: _____

Time: _____

Please put a vertical slash (/) across the line of each question most represents you right now.

	How strong is your desire to eat?	
Very weak	_____	Very strong
	How hungry do you feel?	
Not hungry at all	_____	Very hungry
	How thirsty are you?	
Not thirsty at all	_____	Very thirsty
	How full are you?	
Not full at all	_____	Very full
	How satiated are you?	
Completely empty	_____	Cannot eat another bite
	How much do you think you can eat?	
Not at all	_____	A large amount
	How comfortable do you feel physically?	
Not comfortable at all	_____	Very comfortable
	How do you feel about the visual appeal of the meal?	
Very bad	_____	Very good
	How do you feel about the smell of the meal?	
Very bad	_____	Very good
	How do you think about the taste of the meal?	
Very bad	_____	Very good
	How palatable do you think about the meal in overall?	
Very bad	_____	Very good

Time 20 Visual Analogue Scales:

Participant ID: _____

Date: _____

Time: _____

Please put a vertical slash (/) across the line of each question most represents you right now.

Very weak	How strong is your desire to eat?	Very strong
Not hungry at all	How hungry do you feel?	Very hungry
Not thirsty at all	How thirsty are you?	Very thirsty
Not full at all	How full are you?	Very full
Completely empty	How satiated are you?	Cannot eat another bite
Not at all	How much do you think you can eat?	A large amount
Not comfortable at all	How comfortable do you feel physically?	Very comfortable
Very bad	How do you feel about the visual appeal of the meal?	Very good
Very bad	How do you feel about the smell of the meal?	Very good
Very bad	How do you feel about the taste of the meal?	Very good
Very bad	How palatable do you feel about the meal in overall?	Very good

Post-meal Visual Analogue Scales:

Participant ID: _____

Date: _____

Time: _____

Please put a vertical slash (/) across the line of each question most represents you right now.

Very weak	How strong is your desire to eat?	Very strong
Not hungry at all	How hungry do you feel?	Very hungry
Not full at all	How full are you?	Very full
Completely empty	How satiated are you?	Cannot eat another bite
Not at all	How much do you think you can eat?	A large amount
Not at all comfortable	How comfortable do you feel physically?	Very comfortable

Appendix L: Guideline for Breakfast Record and Laboratory Visits for study one

Guideline for Breakfast Record and Laboratory Visits

Breakfast Record Day

As you are required to record three breakfasts consecutively before your laboratory visit, the following guidelines will help you to find the appropriate breakfast record day.

The first day of breakfast record starts between the fourth day of your menses and one week after the cessation of the menses. In the example below, if your menses starts on Monday June 30th, you could record your breakfast intake any three consecutive days in the grey zone, i.e. between July 3rd and July 10th. When you complete the food record with photo taking of your breakfast, please return the materials within one week.

June 2010						
Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
30	31	1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	1	2	3

Laboratory Visit

Please contact us for making an appointment of the trial. If your next menses starts on Tuesday March 30th, your first trial visit will be arranged between the fourth and eighth day from the start of the menses, July 3rd and 7th (the Italic underline days) Your second visit will be arranged approximately one week after your first trial visit.

July 2010						
Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
27	28	29	30	1	2	<u>3</u>
<u>4</u>	<u>5</u>	<u>6</u>	<u>7</u>	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

Calendar for food record and laboratory visit

- Circle the first date of menses.
- **Underline** the three day of breakfast records.

July 2010

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
27	28	29	30	1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

August 2010

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31	1	2	3	4

September 2010

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
29	30	31	1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30	1	2

Appendix M: Reminder for Laboratory Visits at CHERC for study one and two



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Reminder for Laboratory Visits at CHERC

Trial Day: _____

Participant ID: _____ (internal use)

Prior to the Trial

- Record food intake (meals, drinks, snacks) for 2 days prior to the trial day with photos taking.
- Wear the heart rate monitor and the accelerometer from the morning 2 days prior to the trial day.
- Maintain habitual food intake for two days before the trial day.
- No alcohol 24 hours prior to the trial day.
- No unusual rigorous exercise on the day prior to the trial day.
- Consume the same evening meal before each trial day.
- No food and fluids including water 10 hours before the trial, i.e. _____
- Use minimal physical activity to travel to the CHERC.
- Bring along short pants and vest.

The use of heart rate monitor

- Attach the Polar Coded Transmitter to the elastic strap.
- Adjust the strap length to fit snugly and comfortably.
- Wet the grooved electrode areas on the backside of the Polar Coded Transmitter.
- The key to flawless operation is to wet the grooved electrode areas carefully.
- Secure the strap around your chest, below the breasts. Lock the buckle.
- Check that the grooved and wet electrode areas are against your bare skin.
- Do not press the receiver buttons under water despite the property of water resistance.
- Do not stretch or bend the Transmitter. This may damage the electrodes.
- Wear the monitor all day (from getting up until bedtime).
- START
 - ❖ In the *Time of Day* mode, press SELECT.
 - ❖ The search for transmission code starts.
 - ❖ The heart rate reading with a heart symbol appears on the bottom of the display in a few seconds.
 - ❖ START the stopwatch by pressing SET/START/STOP.
 - ❖ Stopwatch starts running.
 - ❖ Automatic recording of heart rate starts.
- STOP
 - ❖ STOP the stopwatch by pressing SET/START/STOP and then SELECT three times to go back to the *Time of Day* mode, at the bed time or when the monitor cannot be worn.
- When you take off the monitor to sleep, record the times in your activity log.

The use of the accelerometer

- Snap the belt around your waist.
- Place the monitor over your RIGHT HIP.
- Make sure IT IS THE RIGHT WAY UP (Note – you should be able to read the “Actigraph” label on the device).
- DO NOT GET THE MONITOR WET (Sweat is okay).
- Wear the monitor all day (from getting up until bedtime).
- When you take off the monitor to shower, bathe, or swim, record the times in your activity log.

Appendix N: Sample size calculation

Sample size calculation

Power calculation for repeated-measured study from National Research Council Committee (2003):

$$n = 2 + C (s/d)^2$$

where n was the sample size; C was the constant dependent on the value of significance level (α) and power ($1-\beta$); s was the standard deviation mean; d was the mean difference

The constant C obtained from the value of α and $1-\beta$. Assuming the significance level (α) and statistical power ($1-\beta$) is 0.05 and 80% respectively, C equal to 7.85 was used for calculating the sample size.

		α	
		0.05	0.01
$1-\beta$	0.8	7.85	11.68
	0.9	10.51	14.88

Appendix O: Free food list for study two

Free food list

If you still felt hungry after consuming all the provided foods of each meal, you could choose from the following list:

Fruits

Cherries
Plums
Grapefruit
Peaches
Apples
Pears
Dried apricots
Dried apple

Vegetables

Eggplant/Aubergine
Broccoli
Cauliflower
Cabbage
Mushrooms
Tomatoes
Lettuce
Green Beans
Sweet pepper
Onions
Spinach
Cucumbers

Snacks

Chocolate
Potato crisps
Nuts and Raisins

Milks & alternative

Whole milk
Semi skimmed / skimmed milk
Plain yoghurt
Artificially Sweetened Yoghurt
Cheese
Cottage cheese
Unsweetened Soy Milk

Beans & Nuts

Baked Beans
Soybean in can
Hummus
Peanuts
Walnuts
Macadamia
Almonds

*Others (any times)

Olive oil
Butter
Margarine
Balsamic vinegar
Fresh lemon juice
Fresh lime juice
Mustards
Pepper
Garlic
Chicken broth
Diet drinks

*Water, coffee and tea can be consumed at any times.

Free food list

If you still felt hungry after consuming all the provided foods of each meal, you could choose from the following list:

Cereals & grains

Cornflakes
Sultana Bran
Branflakes
Coco Pops
Cheerios
Rice Krispies
Weetabix
Fresh Mashed Potatoes
French Fries
Bagel
French Baguette
Wholemeal wheat bread
Instant White Rice
Pretzels
Soda crackers
Rice crackers

*Others (any times)

Olive oil
Butter
Margarine
Mustards
Pepper
Garlics
Chicken broth

Fruits

Watermelon
Canned lychee

Vegetables

Mashed potato
Broccoli
Cauliflower
Cabbage
Mushrooms
Tomatoes
Lettuce
Sweet pepper
Onions
Spinach
Cucumbers
Baked potatoes

Milks & alternatives

Cheese
Rice milk

Drinks

Sports / Isotonic drinks
Lucozade

* Water, tea and coffee could be consumed at any times.

Appendix P: Palatability Score between trials in study two

Post meal Palatability

Elements	Breakfast		Lunch		Dinner	
	LGI	HGI	LGI	HGI	LGI	HGI
Visual	75.2	69.1	54.1	41.6	55.3	68.1
	± 12.6	± 19.2	± 25.1	± 24.8	± 26.2	± 27.1
Smell	67.2	66.5	54.5	47.1	54.0	61.1
	± 15.3	± 18.8	± 21.4	± 23.3	± 24.3	± 25.0
Taste	67.9	76.1	58.6	61.9	58.6	74.7
	± 20.2	± 18.1	± 20.0	± 21.8	± 23.5 ^a	± 17.0
Palatability	71.9	73.4	58.1	55.1	56.4	72.0
	± 19.3	± 20.1	± 14.7	± 25.8	± 22.9 ^b	± 18.6
Overall palatability	70.5	71.3	56.3	51.4	56.1	69.0
	± 13.2	± 16.7	± 16.4	± 20.8	± 23.1 ^c	± 20.7

Data presented as mean ± SD (n = 14); ^a significantly different ($t(13) = -3.7$, $p = 0.003$) in the taste to the HGI dinner; ^b significantly different ($t(13) = -3.1$, $p = 0.009$) in the palatability to the HGI dinner; and ^c significantly different ($t(13) = -2.6$, $p = 0.024$) in the overall palatability to the HGI dinner.

Appendix Q: Profile of Mood States for study three and four

PROFILE OF MOOD STATES

Participant ID: _____

Date of Birth (dd/mm/yy): _____

Directions: Below is a list of words that describe feelings people have. For each word, choose the answer that best describes how much you have experienced that feeling *right now*.

Feeling	Not at all	A little	Moderately	Quite a bit	Extremely	Feeling	Not at all	A little	Moderately	Quite a bit	Extremely
Tense	o	o	o	o	o	Annoyed	o	o	o	o	o
Angry	o	o	o	o	o	Discouraged	o	o	o	o	o
Worn out	o	o	o	o	o	Resentful	o	o	o	o	o
Unhappy	o	o	o	o	o	Nervous	o	o	o	o	o
Lively	o	o	o	o	o	Miserable	o	o	o	o	o
Confused	o	o	o	o	o	Cheerful	o	o	o	o	o
Peeved	o	o	o	o	o	Bitter	o	o	o	o	o
Sad	o	o	o	o	o	Exhausted	o	o	o	o	o
Active	o	o	o	o	o	Anxious	o	o	o	o	o
On Edge	o	o	o	o	o	Weary	o	o	o	o	o
Grouchy	o	o	o	o	o	Bewildered	o	o	o	o	o
Blue	o	o	o	o	o	Furious	o	o	o	o	o
Energetic	o	o	o	o	o	Full of pep	o	o	o	o	o
Hopeless	o	o	o	o	o	Worthless	o	o	o	o	o
Uneasy	o	o	o	o	o	Forgetful	o	o	o	o	o
Restless	o	o	o	o	o	Vigorous	o	o	o	o	o
Unable to concentrate	o	o	o	o	o	Uncertain about things	o	o	o	o	o
Fatigued	o	o	o	o	o	Bushed	o	o	o	o	o
Helpless	o	o	o	o	o	<i>The end of the questionnaire.</i>					

Appendix R: Glycaemic and lactate responses between trials in study three

Glycaemic and lactate responses between trials

Time point	[Glucose] (mmol·L ⁻¹)		[Lactate] (mmol·L ⁻¹)	
	LGI	HGI	LGI	HGI
Fasting	4.4 ± 0.23	4.3 ± 0.13	0.59 ± 0.23	0.58 ± 0.22
Post breakfast (0-min pp)	6.4 ± 0.89 ^{a,d}	6.9 ± 1.0 ^{a,d}	1.09 ± 0.27 ^{a,b}	0.83 ± 0.22 ^{a,b}
Before 1 st task (15-min pp)	6.9 ± 1.06 ^{a,b}	8.1 ± 1.25 ^{a,b}	1.59 ± 0.38 ^{a,b}	1.23 ± 0.29 ^{a,b}
After 1 st task (45-min pp)	5.1 ± 0.82 ^a	5.5 ± 1.0 ^a	1.58 ± 0.35 ^{a,d}	1.25 ± 0.27 ^{a,d}
Before 2 nd task (60-min pp)	5.0 ± 0.7 ^a	5.2 ± 0.7 ^a	1.36 ± 0.29 ^{a,d}	1.10 ± 0.23 ^{a,d}
After 2 nd task (90-min pp)	4.8 ± 0.48 ^a	4.5 ± 0.53	1.00 ± 0.14 ^{a,b}	0.85 ± 0.18 ^{a,b}
Before 3 rd task (105-min pp)	4.3 ± 0.4 ^d	4.0 ± 0.53 ^{c,d}	0.74 ± 0.13 ^c	0.71 ± 0.18
After 3 rd task (135-min pp)	4.3 ± 0.35 ^b	4.0 ± 0.48 ^{b,c}	0.57 ± 0.08	0.59 ± 0.14
Before lunch (165-min pp)	4.2 ± 0.32	4.0 ± 0.17 ^a	0.51 ± 0.12	0.57 ± 0.19
Post lunch (180-min pp)	5.6 ± 0.73 ^a	5.3 ± 0.76 ^a	0.44 ± 0.09 ^a	0.43 ± 0.11 ^c

Data presented as mean ± SD (n = 16). [Glucose], glucose concentration; [Lactate], lactate concentration; HGI, high glycaemic index; LGI, low glycaemic index; min pp, minutes postprandially. ^a Significant different to the corresponding fasting level (corrected $p < 0.05$); ^b significant difference when sharing the same letter on the same time point (corrected $p < 0.05$); ^c different to the corresponding fasting level, but not reach significance after the Bonferroni correction (corrected $p > 0.08$); and ^d different when sharing the same letter on the same time point, but not reach significance after the Bonferroni correction (corrected $p > 0.07$).

Appendix S: Substrates oxidation and respiratory exchange ratio during cognitive tasks in study three

Substrates oxidation and respiratory exchange ratio during cognitive tasks

	LGI			HGI		
	Task 1	Task 2	Task 3	Task 1	Task 2	Task 3
CHO (g)	7.6 ± 1.7 ^{a,b,c}	6.3 ± 1.9 ^b	5.6 ± 1.7	6.2 ± 1.9	6.1 ± 1.9	5.2 ± 1.9
Fat (g)	0.62 ± 0.75 ^{b,c}	0.88 ± 0.58	1.18 ± 0.61	1.1 ± 0.86	1.14 ± 0.76 ^b	1.34 ± 0.73
RER	0.96 ± 0.05 ^{a,b,c}	0.93 ± 0.04 ^b	0.89 ± 0.04	0.92 ± 0.06 ^b	0.91 ± 0.05 ^b	0.88 ± 0.04

Data presented as mean ± SD (n = 16). CHO, carbohydrate; LGI, low glycaemic index; HGI, high glycaemic index; RER, respiratory exchange ratio. ^a significantly different to the corresponding Task 2 (corrected $p \leq 0.0014$); ^b significantly different to the corresponding Task 3 (corrected $p \leq 0.04$); ^c significant different to the HGI (corrected $p < 0.05$).

Appendix T: Physical activity readiness questionnaire (PAR-Q)

Physical Activity Readiness
Questionnaire - PAR-Q
(revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of <u>any other reason</u> why you should not do physical activity?

If
you
answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____

DATE _____

SIGNATURE OF PARENT _____
or GUARDIAN (for participants under the age of majority)

WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

Appendix U: Coefficient of variation of biochemical analysis in study four

Coefficient of variation of biochemical analysis

Variables	LGI	N	HGI	n	Group	n
[Glucose](%)	3.5	254	5	254	4.4	508
[Lactate] (%)	7.9	254	7.9	254	7.9	508
Haemoglobin (%)	1.8	188	1.4	184	1.6	372
Haematocrit (%)	2.8	166	4.4	146	3.7	312

HGI, high glycaemic index; LGI, low glycaemic index; n = number of samples

Appendix V: Glycaemic and lactate concentration between trials in study four

Glycaemic and lactate concentration between trials

Time point	[Glucose] (mmol·L ⁻¹)		[Lactate] (mmol·L ⁻¹)	
	LGI	HGI	LGI	HGI
Fasting	4.3 ± 0.34	4.2 ± 0.36	0.62 ± 0.05	0.56 ± 0.05
15-min pp	6.7 ± 0.99 ^{ae}	7.8 ± 0.77 ^{ae}	1.43 ± 0.07 ^{be}	1.08 ± 0.06 ^{be}
30-min pp	5.4 ± 1.08 ^{ae}	6.5 ± 1.13 ^{ae}	1.57 ± 0.08 ^{be}	1.17 ± 0.09 ^{be}
45-min pp	4.9 ± 0.92	5.4 ± 0.99 ^e	1.56 ± 0.08 ^{be}	1.17 ± 0.06 ^{be}
60-min pp	4.7 ± 0.68	5.0 ± 0.75 ^e	1.38 ± 0.07 ^{be}	1.12 ± 0.05 ^{be}
90-min pp	3.6 ± 0.66 ^{ce}	3.6 ± 0.66 ^{ce}	2.79 ± 0.2 ^{ce}	3.02 ± 0.29 ^{ce}
120-min pp	3.7 ± 0.37 ^{ce}	3.7 ± 0.42 ^{ce}	2.45 ± 0.21 ^{cde}	2.59 ± 2.25 ^{cde}
150-min pp	3.8 ± 0.34 ^{ce}	3.7 ± 0.38 ^{ce}	2.36 ± 0.19 ^{ce}	2.3 ± 0.21 ^{cde}

Data presented as mean ± SD (n = 16). [Glucose], glucose concentration; HGI, high glycaemic index; [Lactate], lactate concentration; LGI, low glycaemic index; min pp, minutes postprandially. ^a Significant difference in the [Glucose] when sharing the same superscript on the same row ($p \leq 0.002$); ^b significant difference in the [Lactate] when sharing the same superscript on the same row ($p \leq 0.003$); ^c significant different to corresponding levels at 60-min pp ($p \leq 0.003$); ^d significantly different to corresponding levels at 90-min pp ($p \leq 0.003$); and ^e significantly different to the corresponding fasting level ($p \leq 0.003$) after the Bonferroni correction.

Appendix W Intake and macronutrient oxidation rates and energy expenditure for LGI and HGI trials in study four

Intake and macronutrient oxidation rates and energy expenditure for LGI and HGI trials

	LGI		HGI	
	Intake	Usage	Intake	Usage
Carbohydrate (g)	91.9 ± 12.2	108.4 ± 17.6*	91.2 ± 13.2	109.6 ± 19.7*
Fat (g)	14.9 ± 2.1	14.6 ± 7.7	14.9 ± 2.1	13.7 ± 6.8
Energy (kcal)	574 ± 77	582 ± 72	572 ± 81	579 ± 79

Data presented mean ± SD (n = 16). HGI, high glycaemic index; LGI, low glycaemic index; * significantly different to the corresponding intake ($p < 0.05$).