# THE EFFECT OF THE GLYCAEMIC INDEX OF A PRE-EXERCISE MEAL ON THE GLYCAEMIC AND INSULIN RESPONSES DURING ACUTE EXERCISE

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*Aan my ouers, Dr. Koos en Hendrien Pieters,*
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# ABSTRACT

Introduction: The objectives of this study were to examine the role of the glycaemic index of a pre-exercise meal, ingested 1 hour before an acute exercise test (15 –20 min), on the metabolic profile of elite athletes compared to control subjects in order to advise sportsmen on the ideal carbohydrate composition of the pre-exercise meal.

Subjects and methods: Fifteen elite long distance male athletes (mean age: 22 yr.) and 11 control subjects (mean age: 21 yr.) participated. Each subject underwent three interventions. 1) Intervention I (baseline): Fasting subjects exercised until exhaustion. 2) Intervention II: Each subject consumed a high glycaemic index meal consisting of hot maize porridge and 10 g glucose one hour prior to exercise. 3) Intervention III: Each subject consumed a low glycaemic index meal consisting of cold samp and 10 g sucrose one hour prior to the exercise session. Interventions II and III were done in random order, with both meals providing 50 g carbohydrate. The exercise session was an acute graded exercise test (15 – 20 min) until exhaustion on a Monark cycle ergometer. Exercise started at 70 rpm against a 50 watt resistance which was increased with 50 watt every four minutes until exhaustion was reached. Exhaustion was determined by using the maximal age adapted heart rate (220 – age) or until the subjects reported exhaustion according to a 10-point Borg scale. Blood samples were taken during all interventions at the following time intervals: fasting, prior to exercise, at exhaustion, and 30 min after rest. Parameters measured included serum glucose, insulin, triglycerides, total protein, albumin and haematocrit (to correct for plasma volume changes that might occur during exercise). A DAX profile was also included.

Results: Results showed the following: a) The athletes were on average significantly more insulin sensitive than the control subjects and they also had significantly lower fasting serum glucose and insulin values than the controls (effects of long-term exercise).

b) Glucose values at the end of the exercise session were significantly higher when the subjects exercised in a fasted state compared to when a pre-exercise meal was ingested.
c) The low glycaemic meal resulted in a better/more stable blood glucose and insulin control than the high glycaemic meal.

Conclusion: It is concluded that for acute bouts of exercise a low glycaemic index pre-exercise meal might be a better option than the high glycaemic meal as far as blood glucose and insulin values are concerned. Partaking in this type of exercise in the fasted state, resulted in higher serum glucose values at the end of exercise, but a sharp decline during the 30 min after exercise (hypoglycaemia did, however, not occur).

**Key words:** exercise, performance, exhaustion, glucose, insulin, glycaemic index, carbohydrate oxidation, pre-exercise nutrition, muscle glycogen

# OPSOMMING


**Die effek van die glukemiese indeks van 'n vooroefeningmaaltyd op die glukemiese en insulienrespons gedurende akute oefening.**

Inleiding: Die doel van hierdie studie was om die rol van die glukemiese indeks van 'n vooroefeningmaaltyd wat 1 uur voor 'n akute oefensessie (15 – 20 min) ingeneem is, op die metaboliese profiel van elite atlete met dié van kontrole proefpersone te vergelyk, ten einde sportmanne omtrent die ideale koolhidraatsamestelling van 'n vooroefeningmaaltyd te adviseer.

Proefpersone en metodes: Vyftien elite manlike langafstandatlete (gemiddelde ouderdom: 22 jr) en 11 kontrole proefpersone (gemiddelde ouderdom: 21 jr) het deelgeneem. Elke proefpersoon het drie intervensies ondergaan: 1) Intervensie I (basislyn): Vastende proefpersone het tot uitputting geoefen. 2) Intervensie II: Elke proefpersoon het 'n hoë glukemiese indeks maaltyd, bestaande uit warm mieliepap en 10 g glukose, 1 uur voor die aanvang van die oefensessie ingeneem. 3) Intervensie III: Elke proefpersoon het 'n lae glukemiese indeks maaltyd, bestaande uit koue stampmielies en 10 g sukrose, 1 uur voor die aanvang van die oefensessie ingeneem. Intervensie 2 en 3 is in ewekansige volgorde gedoen en beide maaltye het 50 g koolhidrate verskaf. Die oefensessie het bestaan uit akute, gegradeerde oefening (15 – 20 min) op 'n Monark fietsergometer, totdat uitputting bereik is. Die oefening het 'n aanvang geneem by 70 opm teen 'n weerstand van 50 watt, wat elke 4 min met nog 50 watt verhoog is, totdat uitputting bereik is. Uitputting is bepaal deur gebruik te maak van die maksimale ouderdom-aangepaste harttempo (220 – ouderdom) of wanneer die proefpersone uitputting volgens 'n 10-punt Borg-skaal gerapporteer het. Bloedmonsters is tydens alle intervensies op die volgende tye geneem: vastend, voor oefening, met uitputting en na 30 min rus. Parameters wat gemeet is, sluit in serumglukose, insulien, trigliseriede, totale

proteïne, albumien en hematokrit (om te korrigeer vir plasmavolume verskuiwings wat as gevolg van oefening mag ontstaan). 'n DAX-profiel is ook geneem.

Resultate: Die resultate het die volgende getoon: a) Die atlete was gemiddeld betekenisvol meer insuliensensitief as die kontroles en het ook betekenisvol laer vastende serumglukose- en insulienwaardes as die kontroles gehad (gevolge van langtermyn oefening). b) Glukosewaardes teen die einde van oefeninng was betekenisvol hoër wanneer die proefpersone vastend geoefen het, as wanneer 'n vooroefeningmaaltyd ingeneem is. c) Die lae glukemiese indeks maaltyd het beter/meer stabiele bloedglukose- en insulienbeheer as die hoë glukemiese indeks maaltyd tot gevolg gehad.

Samevatting: Samevattend kan gesê word dat vir 'n akute oefensessie 'n lae glukemiese indeks vooroefeningmaaltyd 'n beter opsie mag wees as 'n hoë glukemiese indeks maaltyd, vir sover dit bloedglukose- en insulienwaardes betref. Deelname aan hierdie tipe oefening in 'n vastende toestand, het hoër serumglukosewaardes aan die einde van oefening getoon, maar 'n skerp daling gedurende die 30 min rusperiode tot gevolg gehad (hipoglukemie het egter nie voorgekom nie).

**Sleutelwoorde:** oefening, prestasie, uitputting, glukose, insulien, glukemiese indeks, koolhidraatoksidasie, vooroefeningvoeding, spierglikogeen

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| | |
|---|---|
| A | athletes |
| ADA | American Diabetes Association |
| Alb | albumin |
| ANOVA | analysis of variance |
| ATP | adenosine triphosphate |
| BMI | body mass index |
| C | controls |
| °C | Degrees Celsius |
| Ca-ATPase | calcium adenosine triphosphatase |
| Cat. No. | catalogue number |
| CHO | carbohydrate |
| Chol | cholesterol |
| CV | coefficient of variation |
| CVD | cardiovascular disease |
| DAX | discreet analyser |
| DBP | diastolic blood pressure |
| DM | diabetes mellitus |
| EDTA | ethylene diamine-tetra-acetic acid |
| ELISA | enzyme linked immunosorbent assay |
| Exh HR | exhaustion heart rate |
| FAO | Food and Agriculture Organisation of the United Nations |
| FFA | free fatty acids |
| Fig. | figure |
| G | gram |
| g/dl | gram per decilitre |
| GI | gastrointestinal |

| | |
|---|---|
| g/l | gram per litre |
| GI | glycaemic index |
| Glc | glucose |
| GLM | general linear model procedure |
| GR | glycaemic response |
| Hct | haematocrit |
| HDL-C | high density lipoprotein cholesterol |
| HGI | high glycaemic index |
| HPLC | high pressure liquid chromatograph |
| IDDM | insulin dependant diabetes mellitus |
| IU/l | international units per litre |
| J | Joule |
| kJ | kilojoule |
| LDL | low density lipoprotein |
| LDL-C | low density lipoprotein cholesterol |
| LGI | low glycaemic index |
| LPL | lipoprotein lipase |
| mg/l | milligram per litre |
| min | minute |
| ml | millilitre |
| mm | millimetre |
| mmol/l | millimol per litre |
| MUFA | monounsaturated fatty acids |
| ND | not determined |
| NIDDM | non-insulin dependant diabetes mellitus |
| NSP | non-starch polysaccharides |
| PAI-1 | plasminogen activator inhibitor 1 |
| pmol/l | picomol per litre |
| PUFA | polyunsaturated fatty acids |

| | |
|---|---|
| PWC | physical work capacity |
| RBC | red blood cell |
| RER | respiratory exchange ratio |
| Res HR | resting heart rate |
| rpm | revolutions per minute |
| RS | resistant starch |
| S | serum |
| SBP | systolic blood pressure |
| SD | standard deviation |
| SFA | saturated fatty acids |
| SPSS | Statistical Package for Social Sciences |
| T | time |
| t | teaspoon |
| TAS | total antioxidant status |
| % TE | percentage of total energy |
| TG | triglycerides |
| TPA | tissue plasminogen activator |
| T prot | total protein |
| ug/l | microgram per litre |
| µmol/l | micromol per litre |
| USDA | United States Department of Agriculture |
| µU/ml | micro units per millilitre |
| UV | ultra violet |
| VLDL | very low density lipoprotein |
| $VO_2$ max | maximal oxygen uptake |
| *vs* | versus |
| WHO | World Health Organisation |

# CHAPTER 1 INTRODUCTION

## 1.1 BACKGROUND TO THE PROBLEM AND MOTIVATION

Murray & Lopez (1996) stated that cardiovascular disease (CVD) was the main cause of mortality in 1990 world-wide and that it will still be one of the main causes of death in developed and developing countries by the year 2020. This high incidence, besides personal loss, has a major impact on health services and the economy. In South Africa alone, the cost of treating CVD disorders was already between 4 – 5 billion rand in 1991. This excluded rehabilitation and follow-up visits (Pestana *et al.*, 1996). Many studies have been done to determine possible risk factors for CVD in an attempt to decrease the high incidence world wide. These risk factors include hypertension, smoking, dyslipidaemia, obesity, a high fat diet, abnormalities in the haemostatic profile, insulin resistance and lack of exercise (European Atherosclerosis Society, 1993).

The beneficial effect of exercise on other risk factors for CVD such as obesity, hypertension, insulin resistance and dyslipidaemia has been reviewed extensively by Eriksson *et al.* (1997). Exercise can also protect against CVD through its effects on haemostasis (Connely *et al.*, 1992). From the available literature it is difficult to gather which haemostatic effects of exercise are caused by increased energy expenditure (usually coupled with increased energy intake), which are caused by improved energy balance, and which by exercise and physical fitness *per se*. Studies with different intensity and duration of exercise and uncertainties in defining optimal exercise further complicate comparison and interpretation of results (Suzuki *et al.*, 1992).

Nevertheless, there is agreement that fibrinolytic capacity is increased by exercise (Boman *et al.*, 1994), especially because of its effects on plasminogen activator inhibitor 1 (PAI-1) and tissue plasminogen activator (tPA). Effects on plasma fibrinogen are less

clear but Ernst *et al.* (1985) have found that long-term physical activity decreased plasma fibrinogen levels. The effect of long-term exercise on the haemostatic profile has been investigated substantially in the literature (Ernst *et al.*, 1985), but the effect of short-term exercise has not been investigated to the same extent.

Acute and long-term exercise may have separate effects on haemostasis. Acute bouts of dynamic exercise increase tPA activity and decrease PAI-1 activity, but values return to pre-exercise values after 24 hours (Rankinen *et al.*, 1995). Wang *et al.* (1995) showed that acute bouts of strenuous exercise transiently increase platelet adhesiveness and aggregability, but that long-term endurance training suppressed it. There is no information whether this acute effect may be influenced by diet. For optimal dietary recommendations to sportsmen and women, more information on the combined effect of exercise and diet on haemostasis is urgently needed.

The ingestion of a high carbohydrate (CHO), low fat diet together with appropriate amounts of physical activity to maintain or improve weight status is advised by the United States Department of Agriculture (1995). This forms part of the prudent guidelines for the prevention of chronic diseases of lifestyle such as CVD. These are also the guidelines prescribed for optimal nutrition of athletes (Williams, 1993). Because there is no information available on the influences of the glycaemic index (GI) of a meal (different types of CHO) on haemostatic balance, the effects of two types of CHO (a low glycaemic index (LGI) and high glycaemic index (HGI)) were investigated in this study.

Intake of LGI foods may lead to lower but sustained post-prandial blood glucose levels and increased insulin sensitivity (Båvenholm *et al.*, 1995). It will also ensure that fermentable CHO will reach the colon with a resultant increase in short-chain fatty acid production, increased post-prandial blood acetate and decreased free fatty acid (FFA) levels (Cummings & Macfarlane, 1991). All these factors might lead to a decrease in the activation of the clotting system and an increased fibrinolytic potential during exercise (Båvenholm *et al.*, 1995). Juhan-Vague *et al.* (1993) and Vague *et al.* (1986) have shown

that increased concentration and/or activity of PAI-1 (the main cause of impaired fibrinolytic capacity) is possibly caused by increased insulin secretion or insulin resistance. According to Båvenholm *et al.* (1995) insulin and insulin propeptides might be involved in the regulation of plasma fibrinogen concentration, factor VII level and plasma PAI-1 activity.

Haskell (1985) has argued that relatively little is known about the stimuli needed to produce beneficial changes in the overall health status of the individual. Such stimuli may vary between the transient, short-term effects of a single bout of exercise and the long-term effects of exercise training. Moreover, the effects of exercise intensity, frequency and duration remain inconclusive. To increase understanding of the effects of long-term exercise on serum markers for CVD (lipids, glucose and insulin), it is important to study the transient, short-term effects of a single bout of exercise on these parameters. While the effects of exercise training on CHO and lipid metabolism have been documented and reviewed previously (Goldberg & Elliott, 1987; McArdle *et al.*, 1996), the short-term transient effects of exercise on serum glucose, insulin and lipids have not received similar attention (Pronk, 1993).

There is also still controversy regarding the effect of the GI of a pre-exercise meal on substrate utilisation during exercise and peak sporting performance. As will be discussed in Chapter 2, many studies found beneficial results with the ingestion of a LGI compared to a HGI meal (De Marco *et al.*, 1999), whereas other researchers found no difference (Sparks *et al.*, 1998). Many researchers found the ingestion of a HGI pre-exercise meal detrimental to the exercising athlete (Alberici *et al.*, 1993), while others found either a positive, or no effect at all (summarised by Thomas *et al.*, 1991).

In a study undertaken by postgraduate students in the Department of Nutrition of the PU for CHE, the effects of two types of pre-exercise meals (LGI and HGI) and acute exercise on the haemostatic variables, fibrin network structure and markers of CHO and lipid metabolism (as these form part of the risk for CVD) of elite male athletes and controls

were investigated. Two other students report the haemostatic variables and fibrin network structure. The effect of a LGI and HGI meal on the lipid metabolism before an acute exercise test will form part of the dissertation of another student. In this study the effect of the GI of a pre-exercise meal and acute exercise on glucose and insulin responses of elite male athletes and controls will be discussed. Knowledge of these responses is important because of the important role insulin plays in the regulation of many of the hemostatic variables discussed earlier. The adjustment of the GI of the pre-exercise meal might lead to better glucose and insulin control during exercise, which in turn might improve the hemostatic profile.

## 1.2 OBJECTIVES OF THIS STUDY

The main aim of this study was to determine the effect of the glycaemic index of a pre-exercise meal ingested 1 hour before acute exercise on the serum glucose and insulin responses of elite male athletes (that is athletes who have excelled during national competitions) and controls. The subjects also exercised without a pre-exercise meal to determine whether, according to their glucose and insulin responses, it is best to undertake acute exercise (15 – 20 min) with or without a pre-exercise meal. A group of controls consisting of non-athletes was included in the study to distinguish between the effects of long-term and acute exercise.

## 1.3 DELIMITATIONS OF THE STUDY

Due to the large scope of the study and financial constraints, not all the relevant variables for each separate part of the study could be determined. Therefore, if each of the sub-studies were done alone, a more detailed analysis of more variables could have been undertaken. However, only the most important variables were chosen and determined. Consequently, variables such as lactate and FFA levels could not be determined (discussed in Section 5.3). Respiratory exchange ratio (RER) and maximal oxygen uptake $VO_2$ max were also not determined.

Because the ingestion of a pre-exercise meal (especially CHO's) is known to influence performance and endurance, these factors are also reported on, but it should be noted that these were not the main aims of the study.

## 1.4 ORGANISATION OF THIS DISSERTATION

Following this introductory chapter, a review of the literature is presented in Chapter 2 which provides background information on the GI, including the development of the concept, factors influencing the GI of food, the applicability of the GI and especially the role of the GI in exercise.

Chapter 3 describes all the methods used in the collection of data. Details about the recruitment, exclusion criteria and characteristics of the subjects are given. The study design, organisational procedures, anthropometric measurements and clinical examinations are also discussed. Methods used for blood sampling, preparation of serum and plasma and analyses of all samples are also described. The preparation of the standardisation meal the evening before the experiment as well as meals used during each intervention and the composition thereof are also discussed and explained in this chapter. Problems encountered in the execution of the study will be identified and discussed.

In Chapter 4 all results pertaining to this study are presented either in graphs or tables. These results include mean haematocrit, baseline characteristics of the subjects, hemostatic variables, anti-oxidant status, exercise-associated variables as well as the mean serum glucose and insulin values of the athletes and the controls. The values of individual subjects will not be given but only the mean values and standard deviations for the athletes and controls will be reported and discussed. The individual values were, however, taken into account to determine the presence of outliers that could possibly have influenced the mean. No such outliers were however found in the results.

In Chapter 5 these results will be discussed, compared to those results reported in the available literature and possible interpretations of and explanations for the results investigated.

In Chapter 6 a conclusion is reached as to whether a pre-exercise meal should be given for exercise sessions of 10 - 20 min and if so, what type of CHO (HGI or LGI) it should contain. This conclusion will be reached from an extensive study of available literature and the results found in this study. Recommendations for further studies will also be made.

# CHAPTER 2 LITERATURE SURVEY

## 2.1 INTRODUCTION

The term glycaemic index was first documented by Jenkins *et al.* (1981) in an attempt to explain the considerable variation in blood glucose and insulin responses to equal amounts of CHO from different food sources. The GI concept and its development will be discussed in Section 2.2. The GI is influenced by many interacting factors that can be divided into two groups: physiological (or "human") factors and physical ("food") factors (Vorster *et al.*, 1990). The physiological factors will be discussed in Section 2.3. The food factors (Section 2.4) can influence the GI by influencing the digestion of starch and the absorption of glucose. By slowing digestion and absorption, these factors can produce what is called **lente carbohydrate** (Jenkins & Jenkins, 1995) which is a potential useful therapeutic tool. These therapeutic effects, which include nutrition for sportsmen and prevention/control of diabetes mellitus (DM) and CVD, are discussed in Sections 2.5 and 2.6.

## 2.2 THE DEVELOPMENT OF THE GLYCAEMIC INDEX CONCEPT

Historically carbohydrates have been thought of as being digested in the small bowel and the products (sugars) absorbed, oxidised in muscle and other tissue and excreted as water and carbon dioxide. Fibre was thought to pass through the gut and be excreted in the faeces. From the mid 1970's, however, several research groups realised that the same amount of CHO's, from different sources, produced widely different glucose responses after ingestion (Truswell, 1992). This could not be explained by the initial classification of simple CHO's (sugars) and complex CHO's (starch) (Jenkins *et al.*, 1987; Wolever & Brand Miller, 1995). This classification also led to the exclusion of sugar in the diet of patients with DM with the aim to control their blood glucose concentrations (Wolever & Brand Miller, 1995). Today it is known that natural sugars (including sucrose) cause approximately the same and even

lower blood glucose responses as refined starches such as bread and cooked rice and potato (Brand Miller *et al.*, 1995; Wolever & Brand Miller, 1995).

A new classification for CHO's was needed and Otto and colleagues (1973, as quoted by Wolever, 1990) were the first group to classify CHO's according to their glycaemic responses (GR's). Several studies that showed large differences between the GR's of different subjects (depending on their glucose tolerance status) to the same test food (Jenkins *et al.*, 1994; Wolever, 1992) then followed. In answer to this problem, Jenkins *et al.* (1981) developed the idea of the GI. The GI standardises GR to a standard food (white bread or glucose) to correct for between-subject variation (Jenkins *et al.*, 1994; Wolever, 1992) and can therefore be defined (Jenkins *et al.*, 1994; Truswell, 1992; Wolever, 1990) as:

$$\frac{\text{Incremental area under blood glucose response curve to a 50 g glycaemic CHO portion of food}}{\text{Corresponding area after a 50 g glycaemic CHO portion of standard food (white bread/glucose)}} \times 100$$

## 2.3 PHYSIOLOGICAL (HUMAN) FACTORS THAT AFFECT THE GLYCAEMIC INDEX

These factors are responsible for variations in GI, obtained with a specific food in different individuals or in the same individual over a period of time. Studies have shown considerable variability of GI values between different individuals for the same test meal. For example, for lentils the values ranged from 23 to 70 (Wolever, 1990). Because of variability such as this, it has been suggested that the consideration of average GI values is not valid because they conceal large differences between individual subjects (Hollenbeck *et al.*, 1986). This argument, however, fails to take into account the fact that there are two types of individual variation: **between- and within-individual**, that can cause the differences in GI values (McDonald *et al.*, 1965; Wolever, 1990). When subjects have tested foods only once, it is not possible to determine whether the variation is due to true, consistent differences between subjects

(between-individual variation) or to variation from day-to-day within the same subject (within-individual variation).

### 2.3.1 Within-individual variation

When the same subject tests the same food on several occasions, the variability of that subject's GR can be expressed as the coefficient of variation (CV). This is the standard deviation (SD) expressed as a percent of the mean. The variability of diabetic subjects is different from that of normals as can be seen in Table 2.1.

Non-insulin dependent diabetes mellitus (NIDDM) subjects are the least variable, followed by normal, and then insulin dependent diabetes mellitus (IDDM) subjects, who are nearly twice as variable as NIDDM subjects.

Table 2.1 The average coefficient of variation (CV) values of within-individual variation following several repeats of ingestion of 50 g CHO from white bread (adapted from Wolever, 1990).

| Type of subject | Number of subjects | Repeats | Average CV of within-individual variation in GR |
|---|---|---|---|
| Normal | 22 | 8 | 22 % |
| NIDDM + insulin | 12 | 11 | 15 % |
| NIDDM | 10 | 8 | 15 % |
| IDDM | 14 | 9 | 29 % |

CV - coefficient of variation NIDDM - non-insulin dependent diabetes mellitus

GR - glycaemic response IDDM - insulin dependent diabetes mellitus

### 2.3.2 Between-individual variation

The variability of GR between subjects who are reasonably homogeneous is larger than the variability within the individuals (Wolever, 1990). The between-individual CV was 26 % for 11 normal subjects, 34 % for 10 NIDDM subjects on diet or tablets, 23% for 12 NIDDM subjects on insulin, and 34 % for 14 IDDM subjects (Wolever *et al.*, 1985).

It has been suggested that many factors affect the GR to the same food: variations in the background diet (Vorster *et al.*, 1990), the presence of diabetes, type, treatment, weight, age, sex and race (summarised by Wolever, 1990). Many of these have not yet been studied adequately. The effect of diabetes is clear in that diabetic subjects may have GR areas 5 – 10 times that of normal subjects. The effect of the type of diabetes is, however, not yet clear (Wolever, 1990).

The implication of all this, is that it is not reliable to compare the absolute GR of different foods tested in different groups of subjects since the subjects may have different GR to the same food. To solve this problem, the subjects' responses were indexed to a standard (glucose/white bread) and thus the concept of the GI was introduced.

## 2.4 PHYSICAL (FOOD) FACTORS THAT AFFECT THE GLYCAEMIC INDEX

These are the factors which explain the different GI values of different foods. They are related to the physical characteristics as well as the chemical composition of the food and influence starch digestion and glucose absorption (Cherbut, 1995; Jenkins *et al.*, 1995; Wolever *et al.*, 1988; Wolever & Bolognesi, 1996b).

### 2.4.1 Particle size

Scientists seemed to have reached consensus that the particle size of food does in fact influence the GI of food (Frost *et al.*, 1994; Holt & Brand Miller, 1994; Jenkins *et al.*, 1988; Thorne *et al.*, 1983), the reason being that food processing which disrupts the cellular architecture and fibrous structures of food results in faster digestion and absorption, thereby increasing blood glucose and insulin responses (Holt & Brand Miller, 1994).

An illustration of the effect of food processing is the significantly lower GR to wholegrain products than to wholemeal products (Jenkins *et al.*, 1988). Wholegrain

refers to foods containing the intact grain, whereas wholemeal refers to foods that contain appropriate proportions of all the milled constituents (bran, endosperm, germ) of the grain. Confusing these two terms can lead to the incorrect meal being given to the patient, which in turn may lead to the loss of blood glucose control in diabetic patients (Jenkins *et al.*, 1988).

### 2.4.2 Cooking method

Modern methods of food processing can lead to either an increase or a decrease in the GI of food. Greater rises in blood glucose and insulin responses have been reported after consumption of cooked as opposed to raw starch (Thorne *et al.*, 1983; Wolever, 1990). Cooking disrupts the starch granules causing starch gelatinisation, which increases starch susceptibility to enzymatic digestion (Frost *et al.*, 1994; Wolever, 1990). On the other hand, processes such as parboiling of wheat and rice can lead to decreased blood glucose responses (Frost *et al.*, 1994; Wolever, 1990).

### 2.4.3 Non-glycaemic carbohydrates

Not all CHO's are digested in the small intestine. There are two major sources of CHO's that reach the colon, namely non-starch polysaccharides (NSP) and 5 – 20 % of starch in foods (this amount can increase depending on the NSP content of the food) (Frost *et al.*, 1994). According to the Joint FAO/WHO Expert Consultation Group (1998) the term "non-glycaemic carbohydrates" describes CHO's that are not digested and absorbed in the small intestine but rather reaches the large bowel where it is fermented. Non-glycaemic carbohydrates produce fermentation products such as short-chain fatty acids (propionate, butyrate and acetate) instead of CHO (glucose) for metabolism (Cummings & Macfarlane, 1991; Frost *et al.*, 1994; Jenkins *et al.*, 1995). CHO's that are digested and absorbed in the small intestine, however, is termed glycaemic CHO and this provides CHO for metabolism (Joint FAO/WHO Expert Consultation Group, 1998).

For many years CHO's that were not digested in the small intestine were called dietary fibre (Joint FAO/WHO Expert Consultation Group, 1998). However, it has been

suggested that the term "dietary fibre" be phased out gradually because it is not clear which components of CHO's should be included in this term (Cummings *et al.*, 1997; Joint FAO/WHO Expert Consultation Group, 1998). It has therefore been suggested that the components of non-glycaemic carbohydrates should separately be referred to as soluble and insoluble non-starch polysaccharides (which is currently thought to be the principle part of dietary fibre), non-digestible oligosaccharides and resistant starch (Joint FAO/WHO Expert Consultation Group, 1998).

Taking this discrepancy into account, it is easy to understand that no simple correlation between the fibre content of food and its GI has been found (Jenkins *et al.*, 1981; Truswell, 1992). There seems to be consensus that soluble NSP reduces blood glucose and insulin responses but that insoluble NSP does not have that same effect (Cherbut, 1995; Frost *et al.*, 1994; Jenkins & Jenkins, 1995; Jenkins *et al.*, 1995; Truswell, 1992). The proposed mechanism for the effectiveness of soluble NSP is that it reduces the rate of gastric emptying as well as the small intestinal transit rate (Cherbut, 1995; Wolever, 1990). It impairs glucose absorption by reducing the diffusion rate of glucose, from the centre of the lumen to the intestinal mucosa (Cherbut, 1995).

### 2.4.4 Second-meal effect

The glycaemic response of a standard second meal can be influenced by the GI of the first meal. Wolever (1990) explains that rapidly absorbed CHO results in large blood glucose and insulin responses. This large insulin response causes increased peripheral glucose uptake. Glucose levels may then undershoot fasting levels despite glucose absorption from the gut. This may result in a counterregulatory response with a rise in free fatty acids and relative insulin resistance. When CHO's are absorbed slowly, the blood glucose and insulin responses are less rapid and the tendency for blood glucose to undershoot decreases. This also results in a smaller counterregulatory response and improved glucose disposal after the next meal (Frost *et al.*, 1994; Gresse & Vorster, 1992; Wolever, 1990).

### 2.4.5 Nibbling versus gorging

Fabry and Tepperman (1970) noted more than a quarter of a century ago that individuals who ate more meals a day, therefore prolonging absorption time, had reduced rates of CVD, DM and obesity (Jenkins & Jenkins, 1995). Today it is known that reducing the size and increasing the frequency of meals result in lower mean blood glucose and insulin levels over the day in patients with NIDDM. In the longer term in healthy persons, total and low density lipoprotein cholesterol (LDL-C) levels, fasting apolipoprotein B and serum uric acid levels (as risk factors for coronary heart disease) are reduced (Jenkins & Jenkins, 1995; Jenkins *et al.*, 1995). Nibbling instead of gorging is thus one factor that contributes to the slowing down of small intestinal absorption.

### 2.4.6 Nature of starch

There are only two polysaccharides that are hydrolysed in the human small intestine, namely amylose and amylopectin (Frost *et al.*, 1994; Wolever, 1990). Amylose, the smaller polysaccharide, has a regular helical structure with extensive hydrogen bonding. Amylopectin is a larger polysaccharide with irregular branches and few hydrogen bonds (Frost *et al.*, 1994). This structure of amylopectin allows it to be more readily gelatinised and hydrolised by amylase (Wolever, 1990). This partly explains the lower glucose and insulin responses of high amylose rice (Heijnen *et al.*, 1995; Larsen *et al.*, 1995) as well as for legumes, whose starch granules contain 30 – 40 % amylose compared to other starchy foods with 25 –30 % amylose content (Frost *et al.*, 1994; Wolever, 1990). Another explanation for the lower glucose and insulin responses of high amylose products may be that retrogradation (particularly of the amylose component) can lead to the formation of resistant starch (Heijnen *et al.*, 1995). Resistant starch (RS) is defined as starch and starch degradation products not absorbed in the small intestine of healthy humans. The main forms of RS are: physically enclosed starch, within intact cell structures ($RS_1$), some raw starch granules ($RS_2$), and retrograded amylose ($RS_3$) (Joint FAO/WHO Expert Consultation Group, 1998).

### 2.4.7 Anti-nutrients

Non-nutritive components of food can also alter the rate of digestion and the physiological response to food (Jenkins *et al.*, 1995). The best known examples are phytates, lectins and saponins which decrease digestion of starches by inhibiting amylase-starch reactions (Jenkins *et al.*, 1995; Thorne *et al.*, 1983; Wolever, 1990). Another group of anti-nutrients, enzyme inhibitors, also decreases starch digestibility by inhibiting the activity of enzymes such as sucrase, amylase (Thorne *et al.*, 1983; Wolever, 1990) and α-glucosidase (Jenkins & Jenkins, 1995).

### 2.4.8 Non-carbohydrate components – mixed meals

Most researchers seem to have reached consensus that the GI can be applied to mixed meals by calculating the weighted GI values of the individual foods (Joint FAO/WHO Expert Consultation Group, 1998). The accuracy of this calculation depends on the accuracy of the GI values ascribed to the individual foods (Joint FAO/WHO Expert Consultation Group, 1998; Le Floch *et al.*, 1991; Wolever *et al.*, 1990).

However, there are researchers who believe that the GI can not be applied to mixed meals (Coulston *et al.*, 1987; Hollenbeck & Coulston, 1991). Wolever (1997), however, argues that the conclusions drawn by these researchers were wrong and that reanalysis of the same data delivered results that strongly support the use of the GI in mixed meals.

The reason why many researchers feel that the GI is not applicable to mixed meals is because of the effects of protein and fat on the GR. In healthy and NIDDM subjects, protein (and to a lesser extent fat) increases insulin secretion with a resultant reduced GR (Wolever, 1990; Wolever *et al.*, 1990). Fat, on the other hand, decreases the GR by delaying gastric emptying (Wolever, 1990; Wolever *et al.*, 1990). In IDDM patients the inclusion of protein and fat in the test meal resulted in higher GR's through a mechanism that is not yet known (Wolever, 1990; Wolever *et al.*, 1990). Another question is whether different types of protein and fat exert different physiological

responses. Current evidence suggests that different types of protein and fat do not in fact have a major influence on the GI (Wolever *et al.*, 1990; Wolever *et al.*, 1991).

Wolever and Bolognesi (1996a) put the whole issue of protein and fat into perspective when they gave eight non-diabetic subjects five different mixed meals containing variable energy, fat, protein, carbohydrate and GI. Their conclusion was that when protein and fat are ingested in amounts consistent with the recommended allowances for a prudent diet (protein 15 – 20 % and fat 25 – 30 % of total energy), their effect is negligible. Only when large amounts are consumed (protein 33 – 50 % and fat 53 – 69 % of total energy) as had been done in several studies (Le Floch *et al.*, 1991; Westphal *et al.*, 1990) does their effect become significant (Wolever *et al.*, 1991; Wolever & Bolognesi, 1996a).

Although discrepancies still occur, there is strong enough evidence for the use of GI in mixed meals. Further research is, however, advocated.

## 2.5 APPLICABILITY OF THE GLYCAEMIC INDEX

Because the GI ranks food according to its effect on blood glucose, it may be a useful tool in the treatment of diseases that are sensitive to blood glucose values like diabetes (Brand Miller, 1994; Vessby, 1994), post-gastric surgery patients with hypoglycaemia and CHO induced hyperlipidemia (Jenkins *et al.*, 1981). The GI may also play a role in the treatment of cardiovascular disease (CVD) (Frost *et al.*, 1994). The association between raised serum cholesterol and cardiovascular disease is well known (Lipid Research Clinics Program, 1984). It has also been confirmed that raised serum triglyceride levels may be a CVD risk factor (Frost *et al.*, 1994; Hokanson & Austin, 1996; Wood, 1996). Because insulin may stimulate the hepatic production of very low density lipoprotein (VLDL) and while evidence exists that insulin resistance may play a role in the development of CVD (Frost *et al.*, 1994), it seems logical that LGI diets (which reduce insulin secretion) may be of use in the treatment of hyperlipidemia (Jenkins *et al.*, 1995; Wolever, 1997). The ingestion of LGI meals resulted in

reductions in LDL-C, serum triglycerides and apolipoprotein B without changes in high density lipoprotein cholesterol (HDL-C) (Frost *et al.*, 1994; Jenkins *et al.*, 1995). Patients susceptible to hypertriglyceridaemia should however avoid high doses of fructose (a LGI food) because of its potential hypertriglyceridaemic effect (Uusitupa, 1994). It has also been suggested that the formation of short-chain fatty acids in the colon after ingestion of LGI food may further inhibit cholesterol synthesis in the liver (due to the presence of propionate) (Jenkins *et al.*, 1995).

There is currently controversy regarding the clinical utility of classifying foods according to their GR by using the GI. Part of this controversy is due to methodological variables that can markedly affect the GI values obtained.

Some of the different methodological procedures that need to be sorted out are the:

- method of calculation of area under the blood glucose curve;
- method of blood sampling;
- length of time of studies;
- use of different standards (glucose/white bread);
- number of standard tests; and
- subject characteristics (Gannon & Nuttall, 1987; Wolever *et al.*, 1991).

Another factor contributing to the controversy is the position of the American Diabetes Association (ADA) (1994) on the GI. They feel that priority should be given to the amount, rather than the source of CHO and that applying the GI would limit the food choices for patients with DM (ADA, 1994). Some researchers differ from the ADA in this respect (Wolever & Bolognesi, 1996c; Wolever *et al.*, 1995).

It is important to remember that the GI was meant to supplement the information in food tables, and not replace it. The GI is a valid and potentially useful concept, but it is also very complex and therefore when applying the GI in clinical practice it should be done with caution (Wolever & Bolognesi, 1996c). The GI is not the only, nor the

most important criterion by which to judge food. Some LGI foods should be used in moderation because of a high fat content (chocolate and peanuts) and some HGI foods may be good choices because they have a low energy but a high nutrient content (carrots). In other situations HGI foods may be appropriate because they are low in fat and convenient to use (breakfast cereals and bread).

There are therefore still a number of unanswered questions and unresolved problems that concern the general use of the GI as well as the fact that not everybody is familiar with the GI and how to use it. Consensus should thus be reached to enable scientists to give reliable and useful information to the general public who is currently still confused about the GI and its clinical applicability. Such a study is currently underway (Wolever, 1999).

## 2.6 THE GLYCAEMIC INDEX AND EXERCISE

The rate at which glucose enters the bloodstream affects the insulin response to that glucose providing food source. This ultimately affects the fuels available to the exercising muscle (as will be discussed in detail in this section). Because the GI ranks food on the basis of its GR it can therefore influence substrate utilisation during exercise and ultimately during peak sporting performance. A second mechanism through which the GI can influence sporting performance is its possible affect on carbo-loading and glycogen repletion after exercise. Both these effects of the GI will now be discussed.

### 2.6.1 Substrate utilisation during exercise

Before discussing the influence of the GI on substrate utilisation, an overview of substrate utilisation during exercise in fasted subjects will be given as background information before the effect of the GI is investigated.

There are basically three metabolic systems providing energy to exercising muscle (Guyton, 1991). These systems are summarised in Table 2.2. The phosphagen system

will not be discussed in this dissertation because it is not influenced by the diet and the focus is on substrate utilisation and how diet modification and exercise can influence it.

Table 2.2 The muscle metabolic systems in exercise (Guyton, 1991).

| SYSTEM | ENERGY SOURCES | TIME OF MAXIMAL MUSCLE POWER |
|---|---|---|
| Phosphagen system | Phosphacreatine and ATP* | 8 – 10 seconds |
| Glycogen-lactic acid | Anaerobic glycolysis | 1.3 – 1.6 minutes |
| Aerobic system | Glucose, fatty acids and amino acids | Unlimited time (as long as nutrients last) |

* ATP = adenosine triphosphate

The two major substrate sources of energy are CHO and fat because bodily protein does not contribute significantly to energy production (Coyle, 1995). Of all the CHO and fat sources in the body, there are four that contribute largely to the energy needed by exercising muscles: **muscle glycogen, blood glucose, plasma fatty acids and intramuscular triglyceride** (Coyle, 1995).

Apart from the diet, exercise intensity, fitness and the duration of endurance exercise can also influence the contribution of these four sources of energy during exercise (Coyle, 1995).

**Intensity**

Almost all of the energy for exercise at the low intensity of 25 % $VO_2$ max, like walking, is derived from plasma fatty acids, with an additional small contribution from blood glucose (in the fasted state). As exercise intensity increases to 65 % $VO_2$ max (like moderate running) plasma fatty acid turnover does not increase and the additional energy is obtained by utilisation of muscle glycogen, blood glucose and intramuscular triglyceride. At even higher intensity exercise (85 % $VO_2$ max) fat cannot be oxidised at high enough rates to provide most of the energy, therefore CHO oxidation provides more than two thirds of the energy needed (Coyle, 1995; Craig, 1993). Figure 2.1

shows the contribution of the four energy sources after 30 minutes of exercise at different intensities in fasted endurance-trained subjects.

Of the two main fuels, CHO and fat, fat has several characteristics that would make it the substrate of choice. Fat contains more than twice the energy per unit weight than CHO and is not hydrated when stored in the body (Saltin & Åstrand, 1993). However, as has been indicated during exercise, CHO is the main source of energy, as exercise intensity increases. It is still unclear how the use of lipids as an abundant energy source for the muscle during exercise is limited (Coyle, 1995; Saltin & Åstrand, 1993) because the supply of FFA far exceeds what is taken up by the muscle. The mechanism that explains the limited rate of fat oxidation during exercise is still unknown (Coyle, 1995; Saltin & Åstrand, 1993).

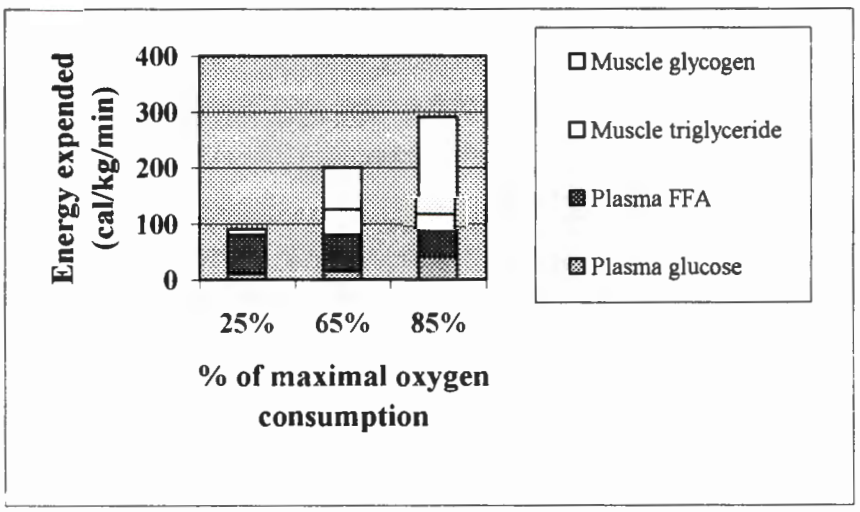



Figure 2.1 Contribution of the four major substrates to energy expenditure after 30 min of exercise at 25 %, 65 % and 85 % of $VO_2$ max respectively (Coyle, 1995).

FFA - free fatty acids 1 cal = 4.186 Joule

**Fitness level**

As stated earlier, apart from the intensity of exercise, the level of fitness also influences substrate utilisation (Figure 2.2). The major difference is that endurance-trained subjects increase their oxidation of intramuscular triglyceride and therefore spare

muscle glycogen (Coyle, 1995). The sparing of glycogen delays the onset of fatigue because fatigue is associated with the depletion of intramuscular glycogen (Sparks *et al.*, 1998). Even though at a given percentage of $VO_2$ max, normally active people oxidise less fat and more muscle glycogen compared to endurance-trained subjects, the pattern of substrate use during prolonged exercise of various intensities (as shown in Figure 2.1) would be similar for the two groups (Coyle, 1995).

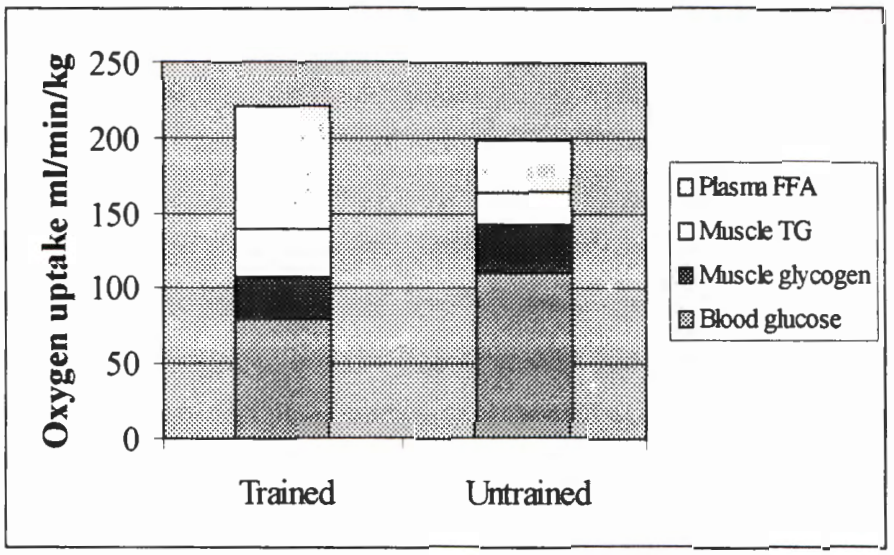



Figure 2.2 Contribution of the four substrates to energy metabolism during exercise when the limb is trained or untrained (Saltin & Åstrand, 1993).

FFA – free fatty acids TG - triglycerides

Saltin and Åstrand (1993) speculated that the following adaptations might occur in trained skeletal muscle, and that this might explain the shift in favour of fat oxidation:

- an increase in the concentration of enzymes for the citric acid cycle, fatty acid oxidation and the electron-transport system;

- an elevation of carnitine and carnitine transferase as transporters for fatty acids within the muscle fibre;

- an increase in fatty acid transporters through the sarcolemma; and

- proliferation in muscle capillarisation, with both a greater number of capillaries per muscle fibre as well as a decrease in the area supplied by a single capillary.

Saltin and Åstrand (1993) discuss these factors in detail. Figure 2.2 shows the contribution of the various substrates for energy in endurance-trained and untrained individuals.

**Exercise duration**

Another factor that can influence the contribution of the four energy sources, is the duration of endurance exercise (Coyle, 1995). Figure 2.3 displays the substrate shifts during prolonged exercise at 65 – 75 % $VO_2$ max in endurance trained subjects after they have fasted overnight.

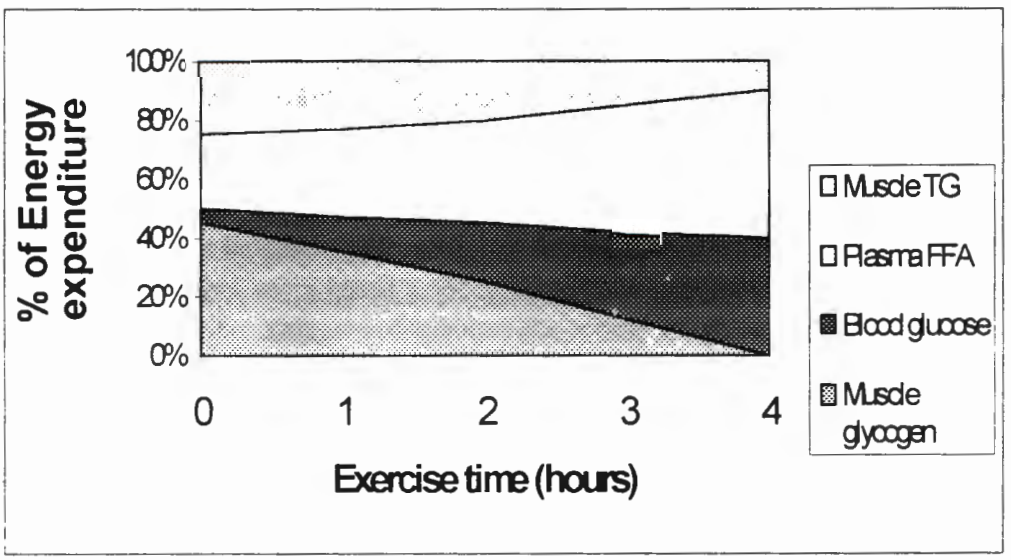



Figure 2.3 Percentage of energy derived from the four major substrates during prolonged exercise at 65 – 75 % of $VO_2$ max (Coyle, 1995).

TG – triglycerides FFA – free fatty acids

About 50 % of the energy for exercise at 70 % $VO_2$ max is derived from fat, with equal contributions from plasma fatty acids and intramuscular triglycerides at the beginning of exercise. There is a small increase in plasma fatty acid contribution over time. CHO provides the remaining 50 % of the energy. During the early phase of

exercise, the majority of CHO energy is derived from muscle glycogen. As the exercise progresses, muscle glycogen is reduced and contributes less to the CHO requirements of exercise and there is increased reliance on blood glucose (Coyle, 1995).

The aim of CHO supplementation prior to and during endurance exercise is to optimise the supply of muscle glycogen and blood glucose late in exercise (Coyle, 1995; Craig, 1993; Guezennec, 1995; Short *et al.*, 1997). Pre-exercise CHO meals have three basic effects during exercise:

- it promotes additional muscle glycogen synthesis when stores are not already super-compensated;

- it replenishes liver glycogen and stores glucose in the body (intestine and glucose space) for oxidation during exercise; and

- it causes increases in CHO oxidation during exercise and decreases in fat oxidation (Burke *et al.*, 1998; Coyle, 1995; Sparks *et al.*, 1998).

The first two responses are beneficial but the resultant increased CHO and decreased fat oxidations are the subject of much controversy amongst researchers. This increased CHO oxidation could be advantageous late in exercise because it provides an energy source to the exercising muscle. It could, however, also be disadvantageous because this increased CHO oxidation could lead to increased glycogen depletion, which will enhance the onset of fatigue (Burke *et al.*, 1998; Coyle, 1995; Thomas *et al.*, 1991). It is, however, not yet clear whether this increased CHO oxidation is due to increased glycogen utilisation or to increased blood glucose uptake by the muscle tissue (Coyle, 1995).

There is currently no consensus as to what the effect of pre-exercise CHO ingestion on exercise performance is. Some studies found it to be beneficial (Kirwan *et al.*, 1998;

Sparks *et al.*, 1998; Thomas *et al.*, 1991); others claimed it to be detrimental (Foster *et al.*, 1979; Kirwan *et al.*, 1998; Short *et al.*, 1997); while others found no effect (Alberici *et al.*, 1993; Febbraio & Stewart, 1996).

Factors that further complicate this issue are:

- the manipulation of the time of HGI food consumption prior to exercise (this may prevent hypoglycaemia at the onset of exercise as will be discussed later) (Guezennec, 1995; Horowitz & Coyle, 1993);
- the type and amount of CHO (Horowitz & Coyle, 1993);
- the consumption of "real foods" *versus* nutrients like sucrose (Horowitz & Coyle, 1993; Sparks *et al.*, 1998);
- the level of training and intensity of exercise (Alberici *et al.*, 1993); and
- the complex effect of insulin on substrate utilisation (as indicated in Table 2.3 compiled from * Jialal *et al.*, 1985, ° Meyer, 1988 and • McCarty, 1994).

Table 2.3 Functions of insulin

| TISSUE | FUNCTIONS |
|---|---|
| **a) Liver** | |
| Not catabolic | ↓ glycogenolysis °<br>↓ gluconeogenesis °<br>↓ ketogenesis ° |
| Anabolic | ↑ glycogen synthesis °<br>↑ fatty acid synthesis °<br>↑ LDL receptor activity ° |
| **b) Muscle** | |
| Not catabolic | ↓ protein catabolism °<br>↓ amino acid output ° |
| Anabolic | ↑ amino acid uptake °<br>↑ protein synthesis °<br>↑ glycogen synthesis °<br>↑ synthesis of Ca-ATPase • |

| TISSUE | FUNCTIONS (CONTINUED) |
|---|---|
| **c) Fat**<br>Not catabolic<br>Anabolic | ↓ lipolysis °<br>↑ glycerol synthesis °<br>↑ fatty acid synthesis °<br>↑ LPL activity °<br>↑ LDL receptor activity °<br>↑ free fatty acid uptake of fat tissue ° |
| **d) General**<br>Anabolic | ↑ glucose transport and metabolism *<br>↑ ion flux (especially potassium) *<br>↑ DNA synthesis *<br>↑ RNA synthesis *<br>↑ cell growth and differentiation * |

↑ - increase ↓ - decrease

LDL – low density lipoprotein LPL – lipoprotein lipase

Ca-ATPase – calcium adenosine triphosphatase

*Acknowledging the GI of the pre-exercise meal may play a role in clearing up some of these issues.*

HGI pre-exercise meals

Intake of HGI food 60 – 30 minutes prior to exercise can lead to hyperinsulinemia at the onset of exercise (Short *et al.*, 1997). This increases glucose uptake by the contracting muscles at a time when liver glucose output may be reduced, causing an imbalance and hypoglycaemia (Coyle, 1995). According to Burke *et al.* (1998) these perturbations in glucose and insulin levels are transient and can be overridden by the metabolic responses to exercise. There may, however, be a small percentage of athletes that are sensitive to exaggerated GR's and they should manipulate their timing as well as the type of CHO intake prior to exercise (preferably LGI).

The resultant hyperinsulinaemia after ingestion of a HGI meal may also reduce lipolysis and FFA availability and thus increase CHO oxidation and glycogen utilisation (Alberici *et al.*, 1993; Sparks *et al.*, 1998). This could lead to a reduced time to exhaustion (Alberici *et al.*, 1993), but several studies have shown no decrease and in

fact, longer times to exhaustion (summarised by Thomas *et al.*, 1991). It is clear therefore, that the use of HGI meals prior to exercise is still controversial.

LGI pre-exercise meals

It is very important not to confuse the terms LGI and complex CHO. Many studies found no benefits with ingestion of complex CHO like potatoes (which is of course a HGI food) and therefore disregarded the concept that the decreased insulin response of complex CHO could lead to increased endurance (Cole, 1995).

Because of the longer absorption time of LGI food, the glycaemic and insulin responses are more moderate than with HGI food. Because of this, it is postulated that LGI food will provide a sustained source of glucose to the blood without the associated insulin surge (Burke *et al*, 1998; Sparks *et al.*, 1998; Thomas *et al.*, 1994). This may lead to higher blood glucose levels at the end of exercise as well as an increased rate of FFA oxidation (insulin has a strong inhibitory effect on FFA mobilisation) (Burke *et al.*, 1998; Kirwan *et al.*, 1998; Saltin & Åstrand, 1993). It may therefore be argued that, due to the higher glucose concentrations and increased lipid oxidation, the ingestion of LGI foods may have a glycogen sparing effect, which in turn may delay the onset of exhaustion (Kirwan *et al.*, 1998).

The hypothesis that pre-exercise ingestion of LGI food would be advantageous during exercise does, however, need further examination because although several studies have shown higher concentrations of fuels in the blood towards the end of exercise (Febbraio & Stewart, 1996; Sparks *et al.*, 1998; Thomas *et al.*, 1994), many showed no increase in endurance time, when compared to HGI foods (Febbraio & Stewart, 1996; Sparks *et al.*, 1998; Thomas *et al.*, 1991; Thomas *et al.*, 1994).

CHO intake during exercise

CHO is generally ingested in a liquid form during endurance exercise in order to simultaneously supply glucose and ensure adequate fluid replacement (Guezennec, 1995; Thomas *et al.*, 1994). The goal for optimal CHO feeding during prolonged

exercise is obtained by ingestion of 40 – 75 g of glucose diluted in 400 – 750 ml of water per hour of exercise (Guezennec, 1995). This optimal feeding is, however, rarely obtained during competitive events.

It is well established that CHO supplementation during exercise can delay the onset of fatigue and therefore increase endurance time (summarised by Sparks *et al.*, 1998). Unlike the intake of CHO 60 – 30 minutes prior to exercise, CHO ingestion during exercise is not affected by the rebound of blood glucose caused by possible hyperinsulinaemia (Walton & Rhodes, 1997). Catecholamines that are released at the onset of exercise may suppress insulin release because of its antagonistic effects.

Because CHO ingested during exercise supplies glucose, it helps to maintain high blood glucose levels and allows CHO oxidation to be maintained at the end of exercise (Burke *et al.*, 1998; Walton & Rhodes, 1997). It appears that exogenous CHO, with the exception of fructose, is oxidised in small amounts during the first hour of exercise (< 20 g) and then reaches a peak rate of oxidation of ± 1 g/min during subsequent exercise (Guezennec, 1995; Hawley *et al.*, 1992). This may lead to sparing of muscle glycogen (Thomas *et al.*, 1994).

Generally, studies examining the effect of CHO intake during exercise have used only HGI sources (Walton & Rhodes, 1997). No studies could be found that compared the intake of HGI foods with LGI foods. Therefore there is no scientific data available to support the theoretical hypothesis. The current hypothesis is that the more rapid digestion of HGI foods will enable blood glucose levels to be elevated during exercise. A LGI source may not increase and maintain blood glucose levels during exercise when they are required. Furthermore, the slow digestion of LGI sources may cause gastrointestinal discomfort (Burke *et al.*, 1998; Walton & Rhodes, 1997). It seems as if the greatest disadvantage of LGI food is its effect on gastric comfort, rather than its GI.

### 2.6.2 The importance of optimal glycogen stores

Because fatigue during prolonged exercise is associated with muscle glycogen depletion, maximal glycogen concentrations are essential for optimal endurance performance (Burke, 1996; Parkin *et al.*, 1997). This includes maximising glycogen stores before the onset of exercise (**carbo-loading**) as well as **glycogen repletion** after exercise, especially with repeated bouts of exercise on the same day or on consecutive days. This will ensure optimal glycogen stores before the start of the next exercise session.

Carbo-loading

A few days before a prolonged and competitive event, athletes should regulate their diets and training in an attempt to maximise (super-compensate or load) muscle glycogen stores. The most practical method of carbo-loading involves altering training and diet for seven days (Coyle, 1995). On days 7, 6, 5 and 4 before competition athletes should train only moderately hard (1 – 2 hours/day) and consume a moderately low CHO diet (± 350 g/day). This will make the muscle CHO deprived and ready to super-compensate. During the third day prior to competition, training should be tapered (30 – 60 min/day of low to moderate intensity) and a high CHO diet should be consumed (± 500 – 600 g/day; >8 g/kg/d) (summarised by Coyle, 1995). Such a regimen will increase muscle glycogen stores 20 – 40 % above normal. This new glycogen loading regimen is as effective as the "classic" regimen which included heavy exercise on days 7, 6, 5 and 4 prior to competition while consuming diets high in protein and fat and very low in CHO (Hamilton *et al.*, 1985). What makes the new regimen more practical is that it does not cause the side effects that were found with the classic regime: abnormal heartbeat, swollen painful muscles because of the accompanying water retention and weight gain (Hamilton *et al.*, 1985). The type of CHO to consume during carbo-loading should be consistent with the CHO indicated in the prudent guidelines and should therefore contain both LGI and HGI foods (Mahan & Escott-Stump, 1996).

Glycogen repletion

**Timing:**

During the first 2 hours after exercise, the rate of muscle glycogen resynthesis is 7 – 8 mmol/kg/h, which is somewhat faster than the normal rate of 6 – 7 mmol/kg/h (Ivy *et al.*, 1988). It therefore seems logical to consume CHO within the first two hours after exercise. Ivy *et al.* (1988) found that muscle glycogen storage 4 hours post-exercise was in fact greater following the immediate ingestion of CHO compared with ingestion after a 2-hour delay. Parkin *et al.* (1997), however, found that delaying the meal by 2 hours had no effect on muscle glycogen resynthesis at 8 and 24 hours post-exercise, providing that sufficient CHO was ingested during the recovery period.

**Type of CHO:**

Results of several studies indicate that a HGI diet, compared to a LGI diet, increases the rate of muscle glycogen repletion after glycogen depleting exercise (Burke *et al.*, 1993; Walton & Rhodes, 1997). The increased muscle glycogen levels seen after a HGI meal may be due to the high blood glucose and insulin levels the meal engenders. Glycogen synthetase is activated by insulin and elevated levels of insulin may increase transport of glucose into cells for storage (Walton & Rhodes, 1997). According to Burke *et al.* (1993), delayed digestion and absorption as well as production of short chain fatty acids in the colon following malabsorption of LGI food may also explain the difference in glycogen resynthesis between LGI and HGI food.

To test the hypothesis that delayed digestion and absorption of LGI foods explain the lower glycogen storage during post-exercise recovery, Burke *et al.* (1996) studied the effect of frequency of CHO feedings on muscle glycogen storage. Feeding HGI foods in a series of small snacks mimics the pattern of delayed digestion and absorption expected with LGI foods while removing extraneous variables such as the nutrient composition of the two meals (Burke *et al.*, 1996). The recovery diets provided 10 g CHO/kg body weight over 24 hours (the amount of CHO considered to be at or above the threshold for glycogen storage) and were composed of HGI foods. In the gorging trial, four meals containing equal amounts of CHO were fed at 0, 4, 8 and 20 hours of

recovery respectively. During the nibbling trial, each meal was divided into snacks of equal CHO content and fed at hourly intervals with a 9-hour break for sleeping (0 - 11, 20 – 23 hours).

No significant difference in muscle glycogen storage over 24 hours was found but the nibbling diet resulted in significantly reduced glucose, insulin and triglyceride concentrations compared to the gorging trial. The results of this study agree with those of Costill *et al.* (1981), who reported no difference in 24-hour muscle glycogen storage when 525 g CHO was consumed by glycogen-depleted runners in two meals or seven meals. The amount of CHO provided to the muscle during recovery appears to be the most critical dietary factor in optimal glycogen synthesis (Burke *et al.*, 1996; Costill *et al.*, 1981). Apparently, when the total CHO intake is adequate, manipulations of glucose and insulin levels within physiological ranges might not be critical in long-term or daily glycogen storage (Burke *et al.*, 1998).

**Co-ingestion of fat and protein**

The effects of protein and fat on insulin secretion have already been discussed (Section 2.3.8). Zawadzki *et al.* (1992) suggested that post-exercise muscle glycogen storage could be enhanced with a CHO-protein supplement as a result of the interaction of CHO and protein on insulin secretion. This is, however, unlikely, as has been explained above. Burke *et al.* (1995) found that the addition of moderate amounts of fat and protein to a recovery diet does not alter glycogen storage over 24 hours, provided CHO intake is adequate.

In conclusion, although the role of the GI in exercise is still unclear there are definite indications that it might be useful and important in the diet of endurance trained athletes. Further research is needed to clear up some of the still controversial issues.

# CHAPTER 3 METHODS

## 3.1 INTRODUCTION

This study was a multidisciplinary investigation into the effects of intense or acute exercise on blood volume shifts (measured as changes in haematocrit), haemostasis (fibrinogen, fibrin monomers, d-dimer, factor $VII_C$ and fibrin networks), serum lipids (triglycerides) and glucose homeostasis (serum glucose and insulin) during fasting conditions and when a high or low glycaemic meal was eaten before exercise commenced. A group of biokineticists, physiologists and sports nutritionists participated as investigators, and three Masters and two Honours dissertations are being written on the results. The author of the present study was responsible for the preparation of all meals consumed during the study, as well as the determination of the glycaemic index of both the high and low glycaemic index meals. The author also participated in all the determinations except those done at the Department of Chemical Pathology of the University of Pretoria (DAX profiles).

To evaluate the study as a whole, the methods of all measurements are given in this chapter (Tables 3.2 – 3.5). Measurements done on haemostasis will, however, not be reported on in this study. In Chapter 4 the results will be reported, with special emphasis on the serum glucose and insulin responses to the high and low glycaemic index pre-exercise meals during acute exercise. In Chapter 5 these results will be discussed, compared to available literature and possible interpretations of and explanations for the results will be investigated.

## 3.2 SUBJECTS

### 3.2.1 Recruitment of volunteers for the study

Healthy male students, athletes and non-athlete controls, were recruited with the help of the Department of Biokinetics at the PU for CHE. Athletes who participated in moderate-to-vigorous physical activity for more than 30 minutes per session for at least four to seven days per week for the previous three months and male students who had not participated in any regular physical activity for at least three months, were recruited. After a motivational explanation of the objectives and methods of the study, the students volunteered to take part and signed a written informed consent form (Addendum A). The study was done under the ethical approval no. HHK4M5-95 given by the Ethics Committee of the PU for CHE.

### 3.2.2 Exclusion criteria

The exclusion criteria for the study were:

(i) any chronic disease that could influence haemostasis;

(ii) smoking;

(iii) use of alcoholic beverages;

(iv) use of any dietary supplements; and

(v) use of any medication that could influence haemostasis.

Therefore, most life-style related factors known to influence haemostasis, lipoprotein metabolism or glucose and insulin levels were controlled in an attempt to measure the independent effect of physical activity.

### 3.2.3 Characteristics of the subjects

Fifteen athletes, aged between 18 and 26 years and 14 non-athletes (controls), aged between 19 and 25 years, volunteered to participate. Baseline measurements were taken in May 1998, while the interventions were done during September 1998. Two athletes

(no. 5 & 6) and three controls (no. 18, 22 & 30) were unable to participate during the September experiments and their data were excluded. Two new replacement athletes (no. 27 & 31) were recruited and their baseline values were determined during September followed by the intervention period. Complete data are therefore available for 15 athletes and 11 controls. The athletes were randomly given numbers 1 to 15; numbers 27 and 31 were later used for the two replacements in the athlete group. The control group received numbers 16 to 30 at random (excluding number 27). The mean baseline characteristics of the two groups are shown in Table 4.2.

## 3.3 STUDY DESIGN

In this case-control intervention trial baseline measurements during which subjects exercised under fasting conditions, were done over a two-week period, while the same measurements during dietary intervention periods where the subjects took either a low or high glycaemic index meal before exercise, were taken over a three-week period.

The study can be divided into 11 time points where a specific time represents the time when blood was taken from the subject. The baseline (Intervention I) (Time 1 – 3) was done within 14 days during May (winter). The subjects exercised until exhaustion. Intervention II (Time 4 - 7) and intervention III (Time 8 - 11) were done according to a random design over a period of 15 days during September (spring). These consisted of a high glycaemic index- (intervention II) and a low glycaemic index- (intervention III) pre-exercise meal after which subjects exercised again until exhaustion.

The study design and division of the different time points when blood samples were taken during the three interventions are shown in Figure 3.1.

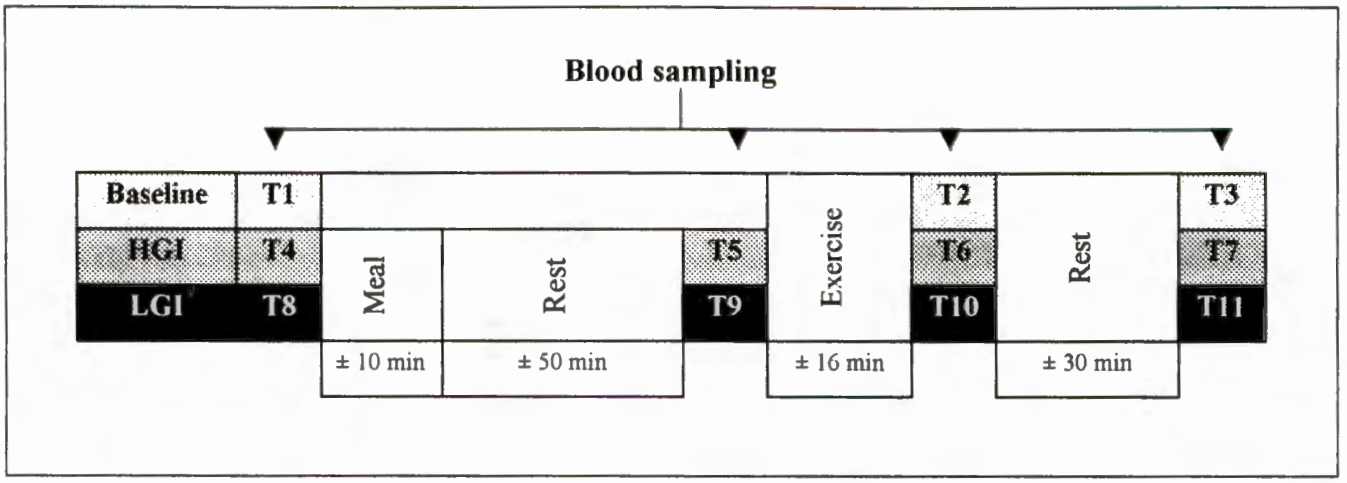



Figure 3.1. The study design.

## 3.4 ORGANISATIONAL PROCEDURES

The subjects arrived in numerical order from 6:00 in the morning at the Biokinetics Institute of the PU for CHE. The study started with only one subject every half-hour to ensure successive evaluation. It took ~2 hours to evaluate each subject. To prevent blood samples from being taken after 10:00 in the morning (Szymanski *et al.*, 1994), only four to five subjects could be tested daily. The subjects had to be fasted since 22:00 the previous evening.

## 3.5 ANTHROPOMETRIC MEASUREMENTS

Anthropometric measurements were taken by post-graduate Biokinetic students. The information was coded on an anthropometric data sheet (Addendum B). Body weight (kg) was determined to the nearest 0.5 kg on a *Seca*® beam balance. Subjects were barefoot and wore only light indoor/athletic clothing. Height in centimetre was measured to the nearest 0.5 cm using a *Seca*® stadiometer. Triceps, subscapular, suprailiac, abdominal, thigh and calf skinfolds were taken in triplicate with a *Slimguide*® skinfold

caliper. These skinfolds were used to calculate fat percentage, using the following formula (McArdle *et al.*, 1996):

Fat % = (Σ6 skinfolds) x 0.1051 + 2.585

## 3.6 CLINICAL EXAMINATIONS

A qualified nursing sister (Mrs. C. Lessing) assisted during the whole of the study. The oral temperature and blood pressure of the subjects were taken while lying on a hospital bed. Blood pressures were taken with a sphygmomanometer (Tycos®, USA) and the first and fifth Korotkoff sounds were recorded as systolic and diastolic pressures, respectively.

## 3.7 BLOOD SAMPLING AND PREPARATION OF SERUM AND PLASMA SAMPLES

Blood samples were drawn by the nursing sister from the *vena cephalica,* using a sterile butterfly 21G-infusion set (Johnson & Johnson, 19 mm) and syringes.

Serum

- For the preparation of serum, 20 ml of blood was allowed to clot in glass tubes (Vacutainer ® SST®), centrifuged (Universal 16R™, HETTICH) at 3 000 rpm for 15 min at 4 °C. For each subject four aliquots were frozen at -84 °C in a Nuaire™ biofreezer.

- For the determination of glucose 5 ml of blood was transferred to a sodium fluoride potassium oxalate tube and centrifuged at 2 700 rpm for 10 min. The serum was aliquoted into Eppendorff tubes and stored at -84 °C.

Plasma

- Citrated blood samples (0.5 ml of 1 mmol/L citrate, pH 4.5 - 4.8 plus 9.5 ml venous blood) were centrifuged in two plastic (medispo) tubes at 2 700 rpm for 10 minutes at 4 °C. The citrated plasma of each subject was divided into seven aliquots and stored at -84 °C.

- Five ml blood was transferred to EDTA glass tubes. Haematocrit and haemoglobin levels were determined on the EDTA blood. Blood cells and EDTA plasma were separated by centrifugation (2700 rpm for 10 min at 4 °C). The aliquoted plasma was stored at -84 °C and the cells at -20 °C.

## 3.8 DIETARY INTERVENTIONS

### 3.8.1 Baseline

No dietary intervention was done on the morning of the baseline test. The subjects followed their normal diet and reported fasting on the morning of testing. Fasting had to start at 22:00 the previous evening. Only water was allowed after that time.

### 3.8.2 Pre-evening meal

The pre-evening meal was only given when dietary intervention took place and not when the baseline testing was done. This meal consisted of a balanced but low-fibre meal given the night before testing, no later than 20:00. A low-fibre meal was given to prevent any possible interference with the glucose response (Wolever, 1990) to the pre-exercise meal on the following day ("second meal effect").

During the baseline testing the 24-hour recall method (see Addendum C) was used to determine the daily energy intake and dietary energy distribution. From this an average energy intake and energy distribution (percentage fat, protein and carbohydrate) of the

diet were determined. This average energy intake was divided by three to estimate the energy content of one meal, using three meals per day as a norm.

Average daily energy (kJ) intake : 12366

Estimated energy (kJ) per meal : 12366 ÷ 3 = 4122 kJ

The energy distribution of the pre-evening meal was a combination of the subjects' existing dietary pattern (average distribution according to the 24-hour-recall) and the distribution recommended for a prudent diet (Ralph, 1993). The energy content and distribution of the pre-evening meal are given in Table 3.1.

Table 3.1 The composition and energy distribution of the evening meal.*

| Amount | Foods | Carbohydrate (g) | Protein (g) | Fat (g) | Energy (kJ) |
|---|---|---|---|---|---|
| 250 ml | Milk, fat free | 12.3 | 8.5 | 0.5 | 373 |
| 80 g | Apple (peeled) | 10.3 | 0.2 | 0.2 | 186 |
| 60 g | Cheese | 1.3 | 14.9 | 16.4 | 899 |
| 30 g (6t) | Sugar/jam | 30 | - | - | 510 |
| 20 g (4t) | Margarine (Canola) | - | - | 16.3 | 619 |
| 5 x 30 g | White bread | 74 | 12.8 | 2.7 | 1578 |
| **TOTAL** | | 127.9 | 36.4 | 36.1 | 4165 |
| kJ | | 2174 | 619 | 1372 | 4165 |
| % of kJ | | 52 | 15 | 33 | 100 |

* Calculated according to the MRC Food Composition Tables (Langenhoven *et al.*, 1991).

3.8.3 High glycaemic index pre-exercise meal

A high glycaemic index meal was given on the morning of testing. It contained porridge prepared with 50 g of raw maize meal and served with 10 g glucose. The recipe is given in Figure 3.2.

| FOOD INGREDIENTS | | | CHO CONTENT | |
|---|---|---|---|---|
| 50 | g | refined maize meal | 40 g | CHO * |
| 5 | ml | salt | | |
| 285 | ml | water | | |
| 10 | g | glucose | 10 g | CHO |
| | | | 50 g | CHO |

METHODS

- Microwave water for two minutes.
- Add maize meal and salt while stirring.
- Microwave porridge for two minutes. Stir well.
- Microwave for another one minute. Stir well.
- Add glucose and serve immediately.

Figure 3.2. Recipe for soft maize porridge.

*(Englyst & Pieters, 1998)

The reasons for the composition of the meal were the following (Joint FAO/WHO Expert Consultation Group, 1997):

- Hot porridge was given because cooled cooked starch contains larger amounts of resistant starch, which will lower the glycaemic index.
- Soft porridge was given because the starch is gelatinised maximally in soft porridge.

- Refined maize meal was chosen because the small particle size favours digestion.
- Glucose was chosen because its glycaemic index (100) is higher than that of sucrose (65) (Brand Miller *et al.*, 1996).
- The glycaemic index of this meal was 64, obtained in two subjects in a pilot study, done by the author, using glucose as a standard.

The CHO content of these meals was not calculated from the Food Composition tables, but it was determined by the researcher self in collaboration with Klaus Englyst, using the method based on that of Englyst *et al.* (1992), which uses a colorimetric end-point. The method used in this study was made more reproducible by the introduction of a high-pressure liquid chromatographic (HPLC) end-point, which allows the use of an internal standard and has the potential advantage of measuring sugars other than glucose (Englyst, 1998).

3.8.4 Low glycaemic index pre-exercise meal

A low glycaemic meal was given on the morning of testing. It contained 50 g of glycaemic CHO. The meal consisted of cooled cooked samp and 10 g sugar. 46.6 g raw samp was used to prepare the meal. The recipe is given in Figure 3.3.

The reasons for the composition of the meal were as follows (Joint FAO/WHO Expert Consultation Group, 1997):

- Cooled samp was given, because cooled cooked starch contains high amounts of resistant starch that lowers the glycaemic response.
- Samp was given because the bigger particle size slows digestion. This will also lead to a lower glycaemic response.
- Sucrose was given because it has a glycaemic index of about half that of glucose. 10 g of sucrose contains ± 5 g of glucose and 5 g of fructose. Fructose has very little effect on the glycaemic response. That leaves only the 5 g of glucose to cause a glycaemic response.

- The glycaemic index of this meal was 40, obtained in a pilot study on the same subjects as mentioned in 3.8.3

| FOOD INGREDIENTS | | | CHO CONTENT | |
|---|---|---|---|---|
| 46.6 | g | raw samp | 40 g | CHO * |
| 5 | ml | salt | | |
| 300 | ml | water | | |
| 10 | g | sugar | 10 g | CHO |
| | | | 50 g | CHO |

METHOD

- Soak samp overnight in 300 ml boiled water.
- Cook samp for 2 ¾ hours on low to medium heat. Stir occasionally.
- Add salt when samp is almost cooked.
- Serve with 10 g sugar.

Figure 3.3 Recipe for samp.

* (Englyst & Pieters, 1998)

## 3.9 EXERCISE INTERVENTION

The subjects exercised to exhaustion. Resting heart rate and blood pressure were taken before exercise. The subjects were asked to do an acute exercise test at maximal effort on a Monark ergometer. At the start of the exercise the subjects cycled at 70 rpm against a resistance of 50 watt (De Scalzi *et al.*, 1987). The resistance was raised with 50 watt every four minutes after blood pressure and heart rate had been taken and after the subjects had reported their levels of exhaustion according the Borg scale (Noble *et al.*, 1983). This increase in resistance was kept up until the maximal age-adapted heart rate

(220 – age) had been reached, or until the subject requested to stop due to exhaustion. This data was used to calculate the physical work capacity (PWC 190) at a heart rate of 190 beats/minute.

## 3.10 ANALYTICAL METHODS

The analytical methods are described in Tables 3.2 to Table 3.5. Table 3.2 gives the non-biochemical methods, Table 3.3 the blood analysis, Table 3.4 the biochemical methods for analyses of serum and Table 3.5 the biochemical methods for analysing of plasma.

## 3.11 STATISTICAL ANALYSES

The Statistical Package for Social Sciences (SPSS) programme was used for the statistical analyses. Paired t-tests were used to obtain significance of differences within groups and analyses of variance (ANOVA) with Tukey's tests (GLM procedure) for significant differences between groups. Results are expressed as means and standard deviations. Spearman correlations were used to determine if glucose and insulin correlated with any of the other variables with and without adjusting for BMI.

## Table 3.2 Non-biochemical methods

| Variables | Normal range | Methods & reference | Place | Apparatus |
|---|---|---|---|---|
| **Dietary intake** | Prudent dietary guidelines (USDA, 1985) | 24 hour recall (Steyn *et al.*, 1994). | Department of Biokinetics, PU for CHE, Potchefstroom. | Dietary intake questionnaire (24 hour-recall) (Steyn *et al.*, 1994). |
| **Dietary intake analysis** | | Food Finder®. | Computer Laboratory. Department Nutrition and Family Ecology, PU for CHE, Potchefstroom. | Food Finder® (Grant *et al.*, 1992). |
| **Glycaemic Index** | Standard = glucose = 100 | GI = Incremental area under the glucose curve for 50 g carbohydrate from test meal; divided by the incremental area under the glucose curve for 50 g carbohydrate from glucose; multiplied by 100 (Jenkins *et al.*, 1981). | Department Nutrition and Family Ecology, PU for CHE, Potchefstroom. | Glucometer®, model 5529. Ames Division (USA). |
| **Fat percentage formula** | 3 - 15 % (McArdle *et al.*, 1996) | ($\sum$6 skinfolds) 0.1051 + 2.585<br>The 6 skinfolds include the<br>• Triceps<br>• Subscapular<br>• Suprailiac<br>• Abdominal<br>• Thigh<br>• Medial calf<br>(Heyward & Stolarczyk, 1996) | Department of Biokinetics, PU for CHE, Potchefstroom. | Slimguide® calliper. |
| **Statistics** | | SPSS – Statistical program<br>ANOVA (paired t-tests); Tukey's test (general linear model procedure).<br>Descriptive statistics; Spearman correlations (non-parametric). | Department Nutrition and Family Ecology. PU for CHE, Potchefstroom. | SPSS programme. |

## Table 3.3 Blood analysis

| Variables | Normal range | Methods & reference | Place | Apparatus |
|---|---|---|---|---|
| **Haematocrit** | 39 - 49 % | Capillary tube (Marienfeld, Germany) and haematocrit centrifuge (Hettich Zentrifugen Haematocrit 24, D-78532, Tutlingen). | Research Laboratory, Department Nutrition and Family Ecology, PU for CHE, Potchefstroom. | Capillary Tube (Marienfeld, Germany) Hettich Zentrifugen Haematocrit 24. |

## Table 3.4 Biochemical methods (Serum)

| Variables | Normal range | Methods & reference | Place | Apparatus |
|---|---|---|---|---|
| **DAX-Profile** | | DAX Profile (discrete analyser). | Institute of Pathology, University of Pretoria. | LX 20 from Beckman® |
| Sodium | 137 - 144 mmol/L | | | |
| Potassium | 3.6 - 4.7 mmol/L | | | |
| Chloride | 98 - 108 mmol/L | | | |
| Total carbon dioxide | 23 - 29 mmol/L | | | |
| Anion gap | 7 - 14 mmol/L | | | |
| Urea | 3.1 - 7.8 mmol/L | | | |
| Creatinine | 81 - 114 mmol/L | | | |
| Uric acid | 0.31 - 0.47 mmol/L | | | |
| Total calcium | 2.2 - 2.55 mmol/L | | | |
| Correlated calcium | 2.2 - 2.55 mmol/L | | | |
| Magnesium | 0.7 - 0.95 mmol/L | | | |
| Phosphate | 0.87 - 1.45 mmol/L | | | |
| Total protein | 66 - 79 g/L | | | |
| Albumin | 39 - 50 g/L | | | |
| Globulin | 18 - 36 g/L | | | |
| Total bilirubin | 4 - 30 umol/L . | | | |

| | | | | |
|---|---|---|---|---|
| Unconjugated bilirubin<br>Conjugated bilirubin<br>Alanine phosphatase<br>Gamma gluthamyl transferase<br>Alanine transferase<br>Aspartate transferase<br>Lactate dehydrogenase<br>Cholesterol-total<br>LDL-Cholesterol<br>HDL-Cholesterol<br>Triglyceride<br>Glucose<br>Osmolarity | 2 - 14 umol/L<br>0 - 8 umol/L<br>38 - 102 IU/L<br>8 - 32 IU/L<br><br>6 - 32 IU/L<br>9 - 34 IU/L<br>90 - 180 IU/L<br><br>3.0 - 5.2 mmol/L<br>2.0 - 3.4 mmol/L<br>0.9 - 1.6 mmol/L<br>0.8 - 1.5 mmol/L<br>3.9 - 5.8 mmol/L<br>275 - 295 mmol/L | | | |
| **Albumin** | 34 - 50 g/L | Boehringer Mannheim;<br>Serum albumin binds quantitatively to the indicator 5,5-dibromo- ø – cresolsulphenophthalein (bromocresol purple, BCP).<br>Cat. No. 1489143<br><br>*Control Precipath U*<br>*Cat. No. 171760*<br>*(Coefficient of variance = 3.23 %)*<br>*Control Precinorm U*<br>*Cat. No. 171735*<br>*(Coefficient of variance = 3.01 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |
| **Total Protein** | 55 - 83 g/L | Boehringer Mannheim;<br>Cat. No. 1553836<br><br>*Control Precipath U*<br>*Cat. No. 171760* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |

| | | | | |
|---|---|---|---|---|
| | | *(Coefficient of variance = 3.1 %)*<br><br>*Control Precinorm U*<br>*Cat. No. 171735*<br>*(Coefficient of variance = 2.49 %)* | | |
| **Triglycerides** | ≤ 2.3 mmol/L | Boehringer Mannheim Enzymatic hydrolysis of triglycerides with subsequent determination of liberated glycerol by colorimetry.<br>Cat. No. 1488872<br><br>*Control Precipath U*<br>*Cat. No. 171760*<br>*(Coefficient of variance = 3.16 %)*<br><br>*Control Precinorm U*<br>*Cat. No. 171735*<br>*(Coefficient of variance = 1.9 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |
| **Total antioxidant status** | 1.3 - 1.77 mmol/L | Boehringer Mannheim Bromocresol Purple Method (BPC).<br>Cat. No. NX 2332<br><br>*Control serum*<br>*Cat. No. NX2331*<br>*(Coefficient of variance = 1.79 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |
| **C-Reactive protein** | < 6 mg/L | The sample is reacted with specific antiserum to form a precipitate which is | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |

| | | | | |
|---|---|---|---|---|
| | | measured turbidimetrically at 340 nm. Boehringer Mannheim. Bromocresol Purple Method (BPC).<br>*Cat. No. CP788*<br>*Control serum*<br>*(Coefficient of variance = 0.87 %)* | | |
| Insulin | 72 - 179 pmol/L (10-25 uU/mL) | Radioimmunoassay for the quantitative determination of insulin concentration in serum. Analytical technique in which radiolabeled insulin competes with unlabeled insulin for binding sites on anti-insulin immobilised to the inside wall of the tube.<br>Cat. No. IC13021<br><br>*Control serum*<br>*(Coefficient of variance = 25.85 %)* | Department of Physiology. PU for CHE, Potchefstroom. | Packard Cobra Auto Gamma Counter. |
| Glucose | 4.2 – 6.4 mmol/L | Glucose was determined after enzymatic oxidation in the presence of glucose oxidase. Boehringer Mannheim. Bromocresol Purple Method (BPC).<br>Cat. No. GL 2623<br><br>*Control Precipath U*<br>*(Coefficient of variance = 2.81 %)*<br><br>*Control Precinorm U*<br>*(Coefficient of variance = 2.75 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein | Operator's Manual; Kinetic reader; model EL 312 U; Biotec Instruments. |

Table 3.5 Biochemical methods (Plasma)

| Variables | Normal range | Methods & reference | Place | Apparatus |
|---|---|---|---|---|
| D-dimer | 4 - 78 ug/L | ELISA method enzygnost® D-dimer micro wavelength 492 nm Behring. Cat. No. OQBC11<br><br>*(Coefficient of variance = 3.57 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Spectrophotometer UV - 1201 Shimadzu (wavelentgh 405 nm). |
| Fibrin monomers | < 3.4 – 14.5 mg/L | Berichrom®FM Kinetic method (enzyme reaction). Soluble fibrin in the sample stimulates activation of plasminogen to plasmin by tissue plasminogen activator (tPA). The resulting plasmin is measured photometrically via its reaction with a chromogenic substrate. Cat. No. OWXZ11<br><br>*(Coefficient of variance = 5.7 %)* | Fibrinogen Unit, Technicon Free State, Bloemfontein. | Spectrophotometer UV - 1201 Shimadzu (wavelentgh 405 nm). |
| Factor VII | 50 - 150 % | Plasma FVIIc was determined with a one-stage clotting assay, calcium thromboplastin extracted from rabbit brain (PT-Fib kit, Instrumentation Laboratories {IL}, Milan, Italy. Cat. No. 84682-10) | Research Laboratory, Department of Nutrition and Family Ecology, PU for CHE, Potchefstroom. | ACL 200 Automated Coagulation Laboratory. |

| | | | | |
|---|---|---|---|---|
| | | Factor VII deficient plasma (IL, Milan, Italy. Cat. No. 84662-50)<br><br>*(Coefficient of variance = 4.12 %)* | | |
| Fibrinogen | 200 - 400 mg/dL | Plasma fibrinogen was measured with the Clauss method, and reagents from Instrumentation Laboratories (IL) (Milan, Italy).<br><br>Fibrinogen standards and controls were purchased from IL and from the National Institute for Biological Standards and Control (code 89/644, NIBSC, Hertfordshire, UK), Cat. No. 1-084691-10 *(Coefficient of variance = 12.8 %)* | Research Laboratory Department of Nutrition and Family Ecology, PU for CHE, Potchefstroom. | ACL 200 Automated Coagulation Laboratory. |

## 3.12 LIMITATIONS OF THE STUDY

The control group had a significantly higher mean body mass index (BMI) than the athletes. As discussed in Section 4.2.2 this might influence several factors such as the lipid profile and fasting glucose values. But it might also indicate the effects of long-term exercise. Possible effects of long-term exercise were also considered in this study when a control group of unfit subjects were included to compare with the responses seen in the athletes.

The amount of CHO given in the pre-exercise meal in this study was only 50 g. This amount is significantly less than the CHO content of the same type of studies cited in the literature. Most of the other researchers gave a CHO portion of 1 g/kg body weight (Goodpaster *et al.*, 1996; Thomas *et al.*, 1994), or even 2 g/kg body weight (Wee *et al.*, 1999). The reason why only 50 g CHO portion was given in this study was because the glycaemic responses of food are related to their CHO load (Wolever *et al.*, 1991). The dose response for bread and glucose appears to be nearly linear up to 50 g available CHO, but the dose response flattens between 50 and 100 g. GI studies are therefore based on 50 g available CHO portions.

Brand Miller *et al.* (1996) classified the GI's of food into three groups namely i) LGI = < 55; ii) intermediate GI = 55 to 70 and iii) HGI > 70 (using glucose as the standard food). The GI of the HGI pre-exercise meal of this study was only 64. It is therefore actually an intermediate GI food. Because the GI of the LGI meal was 40, which is 24 units lower than the 64 of the HGI meal, it was felt that this difference of 24 was large enough to cause a significant difference in the glycaemic and insulin responses of the two meals. The meals were therefore classified as LGI and HGI because of the large difference between the GI's of the two meals. The two meals both consisted out of maize products (porridge and samp). The reason why maize products were chosen is that these are indigenous to South Africa and are the staple foods in many of the provinces including the North West.

Because both the athletes and controls exercised until exhaustion, and the athletes were significantly more fit than the controls, the two groups of subjects did not perform the same amount of work before exhaustion was reached. This difference in workload might influence some of the results, especially the exercise associated variables. Therefore, instead of letting both groups cycle until exhaustion, they could have performed the same amount of work (even though this would mean that the athletes would then probably not have reached exhaustion).

# CHAPTER 4 RESULTS

## 4.1 INTRODUCTION

To evaluate the acute effects of exercise, glucose and insulin responses measured before and after exercise (and the resting period) are compared in both groups (athletes and controls) especially during intervention I (baseline). To evaluate the effect of the glycaemic index of a pre-exercise meal, glucose and insulin responses measured at the same time intervals during interventions I (baseline), II (HGI pre-exercise meal) and III (LGI pre-exercise meal) were compared. Comparison of glucose and insulin response changes during all three interventions between the control and athlete groups, could give an indication of the long-term effects of exercise. Paired t-tests were used to obtain significance of differences within groups and analyses of variance with Tukey's tests (GLM procedure) for significant differences between groups. Results were corrected for changes in plasma volume that occurred during the exercise test (discussed in Section 4.2.1). Both the original and the corrected glucose and insulin values will be given in Tables 4.6 and 4.7, but only the corrected values (Figures 4.1 – 4.22) will be illustrated and discussed.

Subject characteristics are discussed in Section 4.2.2, all variables associated with the exercise intervention are given in Section 4.2.3, and the serum glucose and insulin values of the subjects are reported in Section 4.2.4. In Section 4.2.5 correlations between glucose and insulin and other variables are given. All the above values have been corrected for changes in plasma volume changes. However, in Section 4.2.6, correlations for glucose and insulin and other variables are given after the values have also been adjusted for BMI (Section 4.2.2 explains the effect of the BMI on certain variables).

## 4.2 RESULTS

### 4.2.1 Plasma volume changes

When the levels of serum glucose and insulin are measured over time, a change in blood volume as a direct result of a change in plasma volume may result in an artificial inverse change in the level of the measured parameters. Adjusting for changes in plasma volume will therefore allow a more accurate measurement of the true impact of the manipulated variable of interest (Alberici *et al.*, 1993; Pronk, 1993). Not all researchers, however, correct glucose values for changes in plasma volume. Brouns *et al.* (1989) feel that it is the absolute level of glucose that determines the metabolic effects. For this reason both the original and corrected serum glucose and insulin values are given in Tables 4.6 and 4.7, respectively.

Exercise in the heat may result in substantial losses of total body water, mainly resulting from sweat loss. However, sweat rates have been shown to increase linearly with exercise in any environment, not only heat (Pronk, 1993). Senay and Pivarnik (1985) have shown that during maximal exercise the loss of plasma volume may approach as much as 20 %. In this study the athletes showed only a 5 % loss of plasma volume during the baseline testing after ± 20 min of exercise to exhaustion. The testing was done early in the morning at cool temperatures, which may explain the smaller loss of plasma volume. In this study, changes in haematocrit concentrations were used as an indicator of plasma volume changes. Table 4.1 shows the mean haematocrit values of the athletes and controls at all time intervals (1 – 11) to demonstrate changes in plasma volumes that occurred during this study.

Post-exercise haematocrit values differed significantly ($p \leq 0.05$) from fasted values for every intervention (baseline, HGI and LGI). Therefore corrections were made for changes in plasma volume by using the formula suggested by Rankinen *et al.* (1995):

$$\frac{\text{Hct(1)}}{\text{Hct(2)}} \times \text{Variable(2)}$$

(1) → Fasted value (times 1, 4 and 8)

(2) → All other times subjected to plasma volume changes (times 2, 3, 5, 6, 7, 9, 10 and 11).

Table 4.1 Mean haematocrit values (%) of athletes and controls at all the time intervals (1 – 11).

| | | Time interventions | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | | a | ab≠ | b• | cd | c▪ | cd■ | d+ | e≡ | f♣ | efg# | g° |
| | Mean | 44.13 | 46.33 | 43.4 | 43.2 | 42.27 | 44.33 | 42.13 | 42.43 | 42.3 | 43.97 | 41.9 |
| | SD | 3.58 | 4.48 | 3.96 | 2.93 | 3.58 | 3.04 | 3.23 | 3.8 | 3.66 | 4.08 | 4.17 |
| Controls | | h | hi≠ | i• | j | k▪ | jkl■ | l+ | m≡ | n♣ | mno# | o° |
| | Mean | 46.36 | 50.64 | 46.91 | 45.18 | 45.09 | 47.45 | 44.91 | 45.36 | 45.32 | 47.45 | 45.55 |
| | SD | 4.52 | 4.27 | 4.59 | 3.03 | 2.51 | 3.27 | 2.7 | 2.72 | 2.19 | 3.45 | 2.54 |

abcdefghijklmno means with the same letter differ significantly within the same group

≠•▪■+≡♣#° means with the same symbol differ significantly between the groups ($p \leq 0.05$)

SD - standard deviation

The results of times 5 and 9 (although prior to exercise) were also corrected because of a significant difference in haematocrit values for athletes between times 4 and 5 ($p = 0.014$). The reason for this significant difference is, however, not clear.

Furthermore, the athletes had significant lower haematocrit values ($p \leq 0.05$) than the controls for all time intervals except times 1 and 4. This may be explained by the phenomenon of **sports anaemia** (Mahan & Escott-Stump, 1996). Heavy training can cause a transient sports anaemia, which is characterised by a significant decrease in red blood cell (RBC), haemoglobin concentration and packed cell volume. However,

the RBC morphology remains normal and performance does not appear to deteriorate. Possible causes include a haemodilution effect of expanded blood volume and an increased rate of RBC destruction owing to intravascular haemolysis (Mahan & Escott-Stump, 1996).

### 4.2.2 Subject characteristics

The baseline characteristics of all the subjects are given in Table 4.2, their DAX-profile in Table 4.3 and the mean macronutrient content of their background diet in Table 4.4.

Table 4.2 Mean baseline characteristics of the subjects.

| Variable | Athletes | | Controls | |
|---|---|---|---|---|
| | Mean | SD | Mean | SD |
| Weight (kg) | 69.47 ≠ | 8.68 | 87.73 ≠ | 16.42 |
| Height (cm) | 178.53 | 7.67 | 180.27 | 7.13 |
| BMI (kg/m$^2$) | 21.67 • | 1.84 | 27.00 • | 6.08 |
| Age (years) | 22.13 | 2.70 | 21.27 | 1.90 |
| Fat % | 7.45 * | 1.52 | 17.34 * | 9.40 |
| Rest HR (beats/min) | 66.53 o | 12.93 | 82.86 o | 13.16 |
| SBP (mmHg) | 122 | 12 | 128 | 12 |
| DBP (mmHg) | 73 | 9 | 79 | 8 |
| Fibrin monomers (mg/l) | 15.51 | 7.04 | 13.25 | 4.97 |
| D-dimer (µg/l) | 10.49 | 2.26 | 9.36 | 1.6 |
| Factor VII (%) | 70.53 + | 29.88 | 43.09 + | 26.63 |
| Fibrinogen (g/l) | 3.16 | 1.07 | 2.56 | 0.8 |
| C-reactive protein (mg/l) | 3.12 | 1.18 | 2.99 | 0.51 |
| TAS (mmol/l) | 1.24 | 0.07 | 1.24 | 0.08 |

≠ • * o+ means with the same symbol differ significantly between athletes and controls

SD – standard deviation

BMI - body mass index

Rest HR - resting heart rate

DBP - diastolic blood pressure

SBP - systolic blood pressure

TAS – total antioxidant status

The athletes and controls were comparable regarding mean age and height ($p > 0.05$). However, as expected, the athletes had a significant lower body mass (69.47 ± 8.68 kg *vs* 87.73 ± 16.42 kg) ($p = 0.005$), body mass index (BMI) (21.67 ± 1.84 kg/m$^2$ *vs* 27 ± 6.08 kg/m$^2$) ($p = 0.005$) and fat percentage (7.45 ± 1.52 % *vs* 17.34 ± 9.4 %) ($p = 0.006$) than the controls.

The higher BMI of the control group may be seen as a weak link in the study design. The resting metabolic profile of the subjects reflected the beneficial effect of exercise in that the athletes had significantly lower TC (3.7 ± 0.48 mmol/l *vs* 4.81 ± 1.11 mmol/l) ($p = 0.08$), LDL-C (2.09 ± 0.6 mmol/l *vs* 3.23 ± 1.06 mmol/l) ($p = 0.06$), TG (0.76 ± 0.33 mmol/l *vs* 1.17 ± 0.49 mmol/l) ($p = 0.028$) and glucose (4.67 ± 0.36 mmol/l *vs* 5.05 ± 0.28 mmol/l) ($p = 0.007$) levels and significantly higher HDL-C (1.27 ± 0.25 mmol/l *vs* 1.07 ± 0.19 mmol/l) ($p = 0.028$) levels than the controls (Table 4.3). It could be argued that if differences in response to acute exercise between the two groups are examined, it would not be clear whether these differences are because of long-term exercise (fitness) in the athletes or because of the BMI and metabolic profile differences.

This could have been circumvented by choosing a control group with matched BMI. But on the other hand, it could also be argued that the lower BMI, fat percentage and better metabolic profile of the athletes actually reflect the fact that they exercise and that this is unique to fit athletes. If a group with the same BMI would have been chosen it is doubtful whether they could have reached the same fat percentage and still be in good health.

All data used in this dissertation were therefore corrected for changes in plasma volume changes but not for BMI. In Section 4.2.6, however, correlations between glucose and insulin and other variables are given after adjustment for BMI. These values are only given to be compared with the correlations given in Section 4.2.5, to show the effect that adjustment for BMI would have on the serum glucose and insulin responses.

It is impossible to correct for all possible factors that could influence the data, as this might change the values in such a way that they do not reflect the measured values any more. It is the absolute level of these measured values that determined the metabolic effects that were observed (Brouns *et al.*, 1989).

It is clear from the table that the mean resting heart rate of the athletes was significantly lower than that of the controls, which will be discussed in Section 4.2.3.

The only significant difference between haemostatic variables of the athletes and controls was observed in the baseline factor VII values. The athletes had significantly higher plasma factor VII levels (70.53 %) than the controls (43.09 %). This finding is contradictory to what was expected. According to Marckmann *et al.* (1998) plasma factor VII levels are positively associated with body weight and BMI (both the body weight and BMI of the athletes were significantly lower than those of the controls). A high fat diet is also associated with increased fasting factor VII levels (Marckmann *et al.*, 1998). The percentage of total energy contributed by total fat in the background diet of the athletes was 30.9 % compared to the 36.2 % of the controls as indicated in Table 4.4.

All the haemostatic variables of both the athletes and controls fell within or vary close to their normal ranges (Table 3.4 and 3.5).

Table 4.3 DAX profile of subjects at baseline.

| Variable | Normal range | Athletes | | Controls | |
|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD |
| S-sodium (mmol/l) | 137 – 144 | 141.97 | 1.31 | 141.9 | 0.88 |
| S-potassium (mmol/l) | 3.6 – 4.7 | 3.97 | 0.21 | 4.04 | 0.25 |
| S-chloride (mmol/l) | 98 – 108 | 104.48 | 1.92 | 105.07 | 1.42 |
| S-total carbon dioxide (mmol/l) | 23 – 29 | 23.66 | 1.83 | 23.0 | 1.96 |
| S-anion gap (mmol/l) | 7 – 14 | 13.94 | 1.23 | 13.91 | 1.76 |
| S-urea (mmol/l) | 3.1 – 7.8 | 6.79 * | 1.21 | 5.16 * | 1.06 |
| S-creatinine (mmol/l) | 81 – 114 | 114.99 | 12.78 | 112.45 | 9.44 |
| S-uric acid (mmol/l) | 0.31 – 0.47 | 0.38 | 0.09 | 0.35 | 0.11 |
| S-total calcium (mmol/l) | 2.2 – 2.55 | 2.32 | 0.06 | 2.37 | 0.10 |

| Variable | Normal range | Athletes | | Controls | |
|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD |
| S-calcium corrected (mmol/l) | 2.2 – 2.55 | 2.28 | 0.04 | 2.29 | 0.08 |
| S-magnesium (mmol/l) | 0.7 – 0.95 | 0.91 | 0.06 | 0.87 | 0.06 |
| S-phosphate (mmol/l) | 0.87 – 1.45 | 1.32 | 0.19 | 1.19 | 0.15 |
| S-total protein (g/l) | 66 – 79 | 69.52 | 2.46 | 71.71 | 5.03 |
| S-albumin (g/l) | 39 – 50 | 47.38 | 2.15 | 48.68 | 2.42 |
| S-globulin (g/l) | 18 – 36 | 22.13 | 2.04 | 23.04 | 3.36 |
| S-total bilirubin (umol/l) | 4 – 30 | 9.76 | 3.02 | 11.49 | 8.78 |
| S-bilirubin unconjugated (umol/l) | 2 – 14 | 6.23 | 2.12 | 7.85 | 6.79 |
| S-bilirubin conjugated (umol/l) | 0 – 8 | 3.53 | 0.98 | 3.64 | 2.04 |
| S-alkaline phosphatase (ALP) (IU/l) | 38 – 102 | 72.82 | 26.99 | 84.4 | 26.29 |
| S-gammaglutamyl transferase (GGT) (IU/l) | 8 – 32 | 17.35 | 4.22 | 22.89 | 9.61 |
| S-alanine transaminase (ALT) (IU/l) | 6 – 32 | 19.4 | 13.44 | 25.85 | 13.5 |
| S-aspartate transaminase(AST) (IU/l) | 9– 34 | 33.16 | 32.37 | 26.5 | 9.03 |
| S-lactate dehydrogenase (LD) (IU/l) | 90 – 180 | 156.73 | 44.24 | 147.03 | 31.7 |
| S-cholesterol (mmol/l) | 3 – 5.2 | 3.7 • | 0.48 | 4.81 • | 1.11 |
| S-low density lipoprotein cholesterol (LDL-C) (mmol/l) | 2 – 3.4 | 2.09 • | 0.6 | 3.23 • | 1.06 |
| S-high density lipoprotein cholesterol (HDL-C) (mmol/l) | 0.9 – 1.6 | 1.27 ° | 0.25 | 1.07 ° | 0.19 |
| S-triglyceride (mmol/l) | 0.8 – 1.5 | 0.76 + | 0.33 | 1.17 + | 0.49 |
| S-glucose (mmol/l) | 3.9 – 5.8 | 4.67 ⊗ | 0.36 | 5.05 ⊗ | 0.28 |
| S-osmolarity (mmol/l) | 275 - 295 | 284.4 | 2.53 | 283.09 | 1.58 |

≠ • * ○ + ⊗ means with the same symbol differ significantly between athletes and controls

SD - standard deviation S - serum

The metabolic variables (shown in Table 4.3) of both the athletes and controls fell within the normal ranges, reflecting good health. It is therefore accepted that, for this study, the control group was an appropriate and valid control.

The mean macronutrient content of the background diet of the subjects is shown in Table 4.4

Table 4.4 Mean macronutrient content of the background diet of the subjects.

| | | Kilojoules (kJ) | Total protein (g) | Total fat (g) | SFA (g) | MUFA (g) | PUFA (g) | Chol (mg) | Total CHO (g) | Dietary fibre (g) | Added sugar (g) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Athletes | Mean | 14479 | 112.5 | 124.4 | 44.6 | 42 | 23.8 | 427 | 479 | 30.6 | 161.5 |
| | % TE | | 13.2% | 30.9% | 11.1% | 10.4% | 5.9% | 124 mg/ 4200kJ | 56.2% | | 19% |
| Controls | Mean | 9929 | 86.9 | 99.9 | 37.6 | 35.5 | 17.7 | 330.6 | 281.3 | 18.4 | 98.8 |
| | % TE | | 14.9% | 36.2% | 13.6% | 12.9% | 6.4% | 140 mg/ 4200kJ | 49.2% | | 16.9% |
| Recommendations (USDA, 1995) | | | 10% - 15% TE | 15% – 30% TE | 0% - 10% TE | | 3% - 7% TE | 0 – 300 mg | 55%-75% TE | 27g – 40g | 0% - 10% TE |

SFA - saturated fatty acids MUFA - monounsaturated fatty acids

PUFA - polyunsaturated fatty acids Chol - cholesterol

Total CHO - total carbohydrates % TE - percentage of total energy intakes

Highlighted areas indicate values that are *above* the recommended range. Highlighted areas indicate values that are *under* the recommended range for a prudent diet.

The athletes consumed approximately 5 000 kJ more energy than the controls did to meet their higher daily energy expenditure. The energy distribution of the athletes and controls differed in that the controls consumed a larger percentage of their total energy from protein and fat than the athletes did, while the athletes consumed larger amounts of CHO. The controls also consumed a diet that contained a higher cholesterol content when expressed per 4 200 kJ intake than the athletes did. Together with the larger percentage of CHO, the athletes also consumed larger amounts of dietary fibre and added sugar (sucrose) than the controls did.

4.2.3 Exercise intervention

All variables that are associated with the acute exercise test that took place during the three interventions are given in Table 4.5. Differences between the athletes and controls as well as differences in each group between interventions will be discussed briefly.

**Between groups (athletes *vs* controls)**

The mean resting heart rates of the athletes (63.87; 68.8; 69.87 beats/min) were lower than the mean heart rates of the controls (83.09; 83.64; 76.36 beats/min) for all three interventions and were significantly lower during baseline and the HGI intervention ($p \leq 0.05$). It is known that regular physical activity (long-term exercise) leads to a lower heart rate at any given submaximal intensity (American College of Sports Medicine, 1995). It is therefore clear that the above results support the literature. There were no significant changes in the exhaustion heart rate of the athletes or controls during any of the three interventions.

Physical work capacity (PWC) was measured to determine the level of fitness of the subjects (American College of Sports Medicine, 1995). It is an indication of the work capacity at maximal heart rate, per kilogram body weight. The athletes had a significantly higher mean PWC than the controls for all three interventions (3.27; 2.90; 3.04 watt/kg *vs* 2.04; 1.93; 2.07 watt/kg). The athletes could also cycle against a significantly higher resistance than the controls during all three interventions (240; 210; 223.33 watt *vs* 186.36; 170.45; 177.27 watt). These higher mean PWC and resistance (watt) values are probably the result of long-term exercise that caused a higher level of fitness and performance in the athlete group.

Time to exhaustion was significantly longer for athletes (19.73 ± 2.81 min) at baseline than for controls (14.45 ± 1.97 min) after an acute exercise session. This could be ascribed to the difference in the level of fitness between the athletes and controls. There was no significant difference in time to exhaustion between athletes and controls when a pre-exercise meal (HGI or LGI) was given (14.47; 15.20 min *vs* 13.09; 14.55 min).

Table 4.5 Exercise-associated variables of athletes and controls.

| Variables | | Athletes | | Controls | |
|---|---|---|---|---|---|
| | | Mean | SD | Mean | SD |
| Baseline | Rest HR (beats/min) | 63.87 •[a] | 10.97 | 83.09 • | 14.58 |
| | Exh HR (beats/min) | 197.93 [bc] | 2.66 | 198.82 [i] | 1.94 |
| | Resistance (watt) | 240.0 ≠[d] | 32.46 | 186.36 ≠ | 23.36 |
| | PWC (watt/kg) | 3.27 +[e] | 0.52 | 2.04 + | 0.44 |
| | Exercise time (min) | 19.73 ⊕[fg] | 2.81 | 14.45 ⊕ | 1.97 |
| HGI Intervention | Rest HR (beats/min) | 68.80 • | 10.36 | 83.64 •[h] | 11.55 |
| | Exh HR (beats/min) | 189.47 [b] | 7.42 | 190.91 [i] | 6.47 |
| | Resistance (watt) | 210 ⊕[d] | 45.12 | 170.45 ⊕ | 24.54 |
| | PWC (watt/kg) | 2.90 ≠[e] | 0.47 | 1.93 ≠ | 0.33 |
| | Exercise time (min) | 14.47 [f] | 2.90 | 13.09 [j] | 1.87 |
| LGI Intervention | Rest HR (beats/min) | 69.87 [a] | 9.87 | 76.36 [h] | 13.17 |
| | Exh HR (beats/min) | 189.60 [c] | 12.79 | 186.18 [i] | 5.90 |
| | Resistance (watt) | 223.33 ≠ | 45.77 | 177.27 ≠ | 23.6 |
| | PWC (watt/kg) | 3.04 • | 0.40 | 2.07 • | 0.45 |
| | Exercise time (min) | 15.20 [g] | 2.70 | 14.55 [j] | 2.02 |

•≠+⊕•⊕≠•  means with the same symbol differ significantly between groups

abcdefghij  means with the same letter differ significantly within groups, between interventions

SD - standard deviation

HGI - high glycaemic index

LGI - low glycaemic index

Rest HR - resting heart rate

Exh HR - exhaustion heart rate

PWC - physical work capacity

**Athletes (between interventions)**

The exhaustion heart rate, resistance, PWC and exercise time of the athletes, when exercising fasted was significantly longer than when a HGI pre-exercise meal was ingested. The fasting exhaustion heart rate and exercise time were also significantly

higher than the exhaustion heart rate and exercise time when a LGI pre-exercise meal was ingested. The higher exhaustion heart rate during baseline could possibly be ascribed to the fact that the athletes cycled longer and against a higher resistance than during any of the other interventions. When the HGI and LGI interventions are compared, higher values for resistance, PWC and exercise time were reported with the LGI pre-exercise meal, the difference was, however, not significant.

**Controls (between interventions)**

The same trend was seen with the controls as was found with the athletes. Resistance, PWC and exercise time values were the highest when subjects exercised fasted; second highest when a LGI pre-exercise meal was ingested, while the ingestion of a HGI pre-exercise meal resulted in the lowest values. Exhaustion heart rate was, however, higher when a HGI pre-exercise meal was ingested than when a LGI pre-exercise meal was ingested.

While both groups' exercise variables followed the same trend, more statistically significant differences were observed in variables in the athletes than in the controls.

### 4.2.4 Serum glucose and insulin values of subjects

The mean serum glucose and insulin values of the athletes and controls for all time intervals are shown in Tables 4.6 and 4.7 respectively. The CV for the method of determination of glucose was 2.78 % and for insulin it was 25 %. The different values of each individual subject will not be given and only the mean values and standard deviations will be reported and discussed. The individual values were, however, taken into account to determine the presence of outliers that could possibly have influenced the mean. In both these tables the values before correction for plasma volume changes (original) as well as after correction for the volume changes (corrected) will be given. The reason for this is to show the effect the changes in plasma volume had on the concentrations of the serum glucose and insulin. Significant differences ($p \leq 0.05$) in serum glucose and insulin values over the 11 time intervals will be indicated for the athletes and controls respectively, by using colour co-ordinated alphabetical letters. Significant differences ($p \leq 0.05$) between the

serum glucose and insulin values of the athletes and controls for a specific time interval will also be indicated by using different symbols in black.

In Figures 4.1 – 4.6 the corrected serum glucose and insulin values for each intervention are shown. Thus in each graph the fasted (T1/4/8), postprandial (intervention II and III) (T5/9), post-exercise (T2/6/10) and 30 min rest (T3/7/11) values are shown to present an overall picture of the changes in serum glucose and insulin values during a specific intervention.

In Figures 4.7 – 4.22 the interventions will be divided into the following components: *fasted – post prandial*; *fasted – post-exercise;* and *post-exercise – 30 min rest* value. The significant changes in serum glucose and insulin values of the athletes and controls in each component will also be indicated. Differences between the different time intervals (for the athletes and controls respectively) will be indicated with colour co-ordinated alphabetical letters (**athletes – blue** and **controls – red**) and differences between the athletes and controls for a specific time interval will be indicated with black symbols (see footnote to Figures 4.1 and 4.2 as illustration). The same letters that were used in the tables will be used in the graphs. In these figures, only the corrected values will be given and discussed because they represent a more accurate measurement of the serum glucose and insulin values (as has been discussed in Section 4.2.1).

Significant changes in serum glucose and insulin values during the different interventions will be discussed only with Figures 4.7 – 4.22 to prevent duplication of information.

The absolute glucose or insulin value at a specific time for the athletes could not be compared with the absolute value of the controls, because the fasted value (for each intervention) of the athletes and controls differed. There are two possible ways to deal with this problem:

1. to adjust the values so that the athletes and controls start each intervention from the same fasted value; or

2. to compare the difference (Δ) between the values of two time intervals of the **athletes** with the difference (Δ) between the values of the same two time intervals of the **controls**.

For this study the second (2) option was chosen. The reason for this is that the specific fasted values measured for the athletes and controls, could be a possible effect (result) of long-term exercise (fitness) and could therefore not be adjusted.

Next to each graph that will be discussed (Figures 4.7 – 4.22) it will be indicated if the difference (Δ) between the values of the two time intervals of the athletes differs significantly ($p \leq 0.05$) from the difference (Δ) between the values of the two time intervals from the controls, given in each graph.

An example of how this will be indicated with the graphs is as follows:

$\Delta - \Delta = \mathbf{p \leq 0.05}$ to indicate the difference is significant; and

$\Delta - \Delta = \mathbf{p > 0.05}$ to indicate the difference is not significant.

Table 4.6 Mean serum glucose (mmol/l) values of athletes and controls before (original) and after (corrected) correction for plasma volume shifts.

| | | | Time interventions | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Original | | $a^{\neq}$ | a | $a^{\bullet}$ | $^{\bullet}$ | | | | | | | |
| | | Mean | 4.66 | 5.87 | 4.37 | 4.67 | 4.93 | 4.64 | 4.59 | 4.47 | 4.15 | 4.65 | 4.63 |
| | | SD | 0.37 | 0.83 | 0.55 | 0.24 | 0.77 | 0.90 | 0.57 | 0.40 | 1.30 | 0.76 | 0.36 |
| | Corrected | | $a^{o}$ | ab | b | $^{+}$ | | | | | | | |
| | | Mean | 4.66 | 5.59 | 4.15 | 4.67 | 5.06 | 4.50 | 4.71 | 4.47 | 4.16 | 4.48 | 4.70 |
| | | SD | 0.37 | 0.73 | 0.65 | 0.24 | 0.86 | 0.78 | 0.58 | 0.40 | 1.32 | 0.69 | 0.35 |
| Controls | Original | Mean | $bd^{\neq}$ | bc | $c^{\bullet}$ | $e^{\bullet}$ | | e | | d | | | |
| | | | 5.14 | 5.66 | 5.12 | 4.99 | 5.63 | 4.41 | 4.71 | 4.52 | 4.63 | 4.69 | 4.45 |
| | | SD | 0.27 | 0.74 | 0.55 | 0.35 | 1.45 | 0.70 | 0.74 | 0.35 | 0.97 | 0.82 | 0.44 |
| | Corrected | Mean | $c^{o}$ | | | $de^{+}$ | f | ef | | cd | | | |
| | | | 5.14 | 5.16 | 4.59 | 4.99 | 5.65 | 4.20 | 4.75 | 4.52 | 4.66 | 4.49 | 4.53 |
| | | SD | 0.27 | 0.48 | 1.65 | 0.35 | 1.48 | 0.67 | 0.86 | 0.35 | 1.12 | 0.84 | 0.54 |

abcdeabcdef means with the same letter differ significantly within groups, between interventions

$\neq\bullet*o+$ means with the same symbol differ significantly between groups

SD - standard deviation T - time

Table 4.7 Mean serum insulin (pmol/l) values of athletes and controls before (original) and after (corrected) correction for plasma volume shifts.

| | | | Time interventions | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Original | Mean | ac | ab≠ | b• | cdefg | f | d | e | ghj• | jo | hi | i |
| | | | 144.12 | 168.21 | 125.40 | 181.81 | 300.22 | 233.93 | 228.68 | 141.38 | 206.63 | 193.81 | 142.30 |
| | | SD | 39.84 | 47.45 | 26.36 | 38.10 | 96.04 | 80.68 | 77.20 | 33.81 | 52.35 | 71.75 | 43.27 |
| | Corrected | Mean | ac | ab+ | b⊗ | cdefϕ | egh | g | fh | dij≡ | ik | jl | kl |
| | | | 144.12 | 160.24 | 128.39 | 181.81 | 308.93 | 226.81 | 234.66 | 141.38 | 207.57 | 186.73 | 144.28 |
| | | SD | 39.84 | 44.50 | 31.78 | 38.10 | 103.35 | 73.62 | 78.59 | 33.81 | 53.52 | 67.08 | 44.03 |
| Controls | Original | Mean | k | k≠ | • | lm | l | | | mn• | no | n | n |
| | | | 167.55 | 248.56 | 213.65 | 216.87 | 431.67 | 259.13 | 255.10 | 170.01 | 290.44 | 247.09 | 156.22 |
| | | SD | 70.03 | 67.70 | 104.72 | 63.13 | 248.69 | 100.39 | 103.78 | 35.73 | 128.59 | 62.92 | 34.21 |
| | Corrected | Mean | m | + | ⊗ | mnoϕ | op | | p | nqr≡ | q | r | qr |
| | | | 167.55 | 225.93 | 211.28 | 236.12 | 433.04 | 247.89 | 257.60 | 170.01 | 292.92 | 236.12 | 155.91 |
| | | SD | 70.03 | 54.24 | 106.44 | 68.71 | 248.84 | 97.49 | 108.77 | 35.73 | 135.06 | 60.10 | 35.99 |

abcdefghijklmnabcdefghijklmnopqr means with the same letter differ significantly within groups between interventions

≠•*o+⊗ϕ≡ means with the same symbol differ significantly between groups

SD - standard deviation T - time

The corrected mean serum glucose and insulin values of athletes and controls during intervention I, II and III are illustrated in Figures 4.1 to 4.22 respectively.

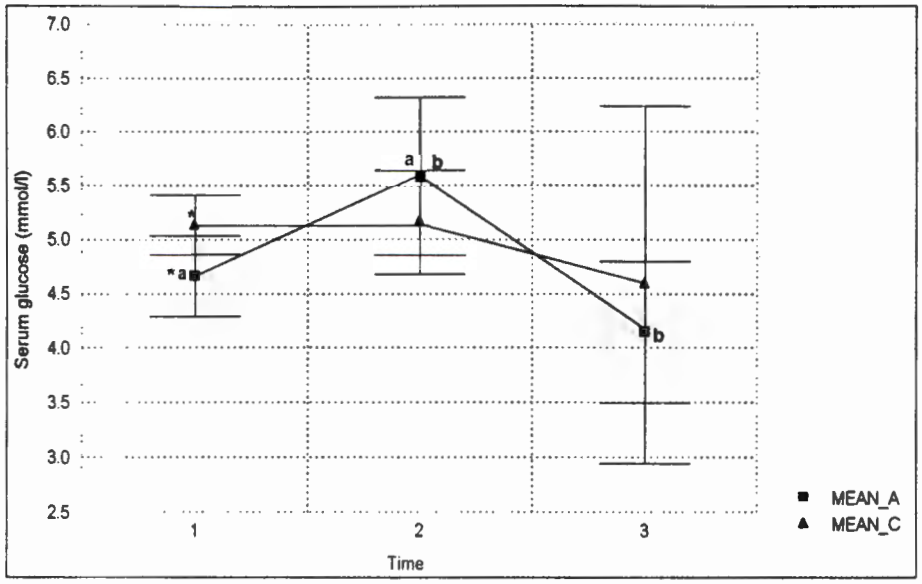



Figure 4.1 Baseline. Serum glucose values of athletes (A) and controls (C).

**ab** means with the same letter differ significantly within groups

* means with the same symbol differ significantly between groups

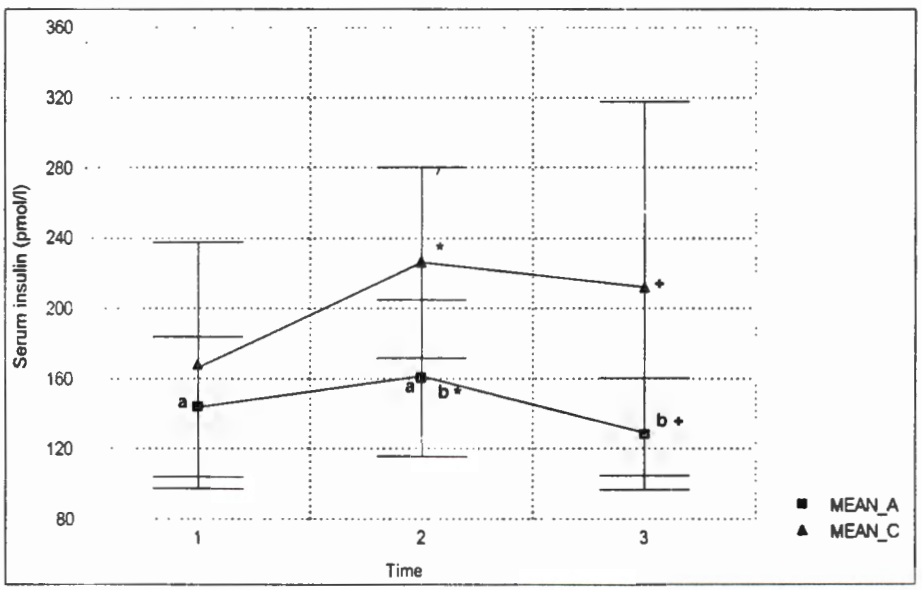



Figure 4.2 Baseline. Serum insulin values of athletes (A) and controls (C).

**ab** means with the same letter differ significantly within groups

*+ means with the same symbol differ significantly between groups

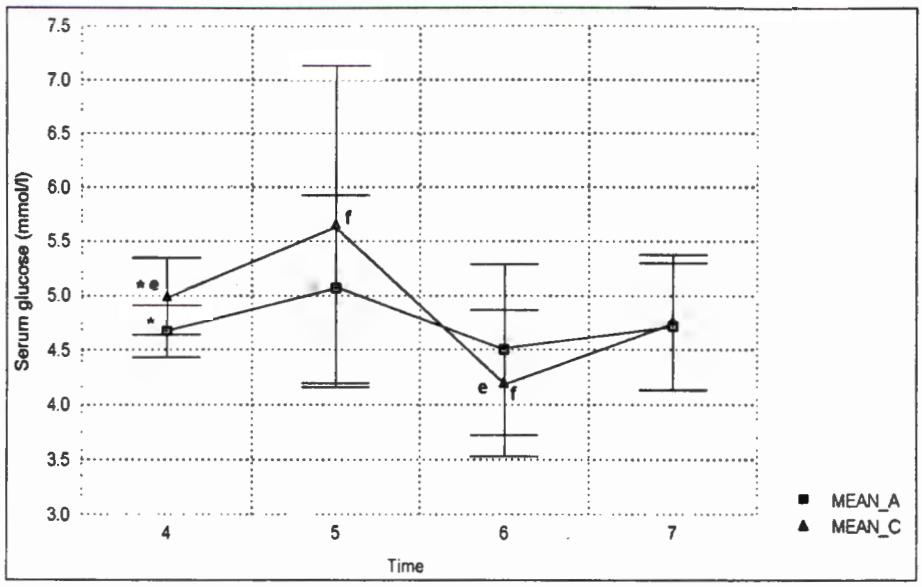



Figure 4.3 HGI intervention. Serum glucose values of athletes (A) and controls (C). (HGI – high glycaemic index pre-exercise meal).

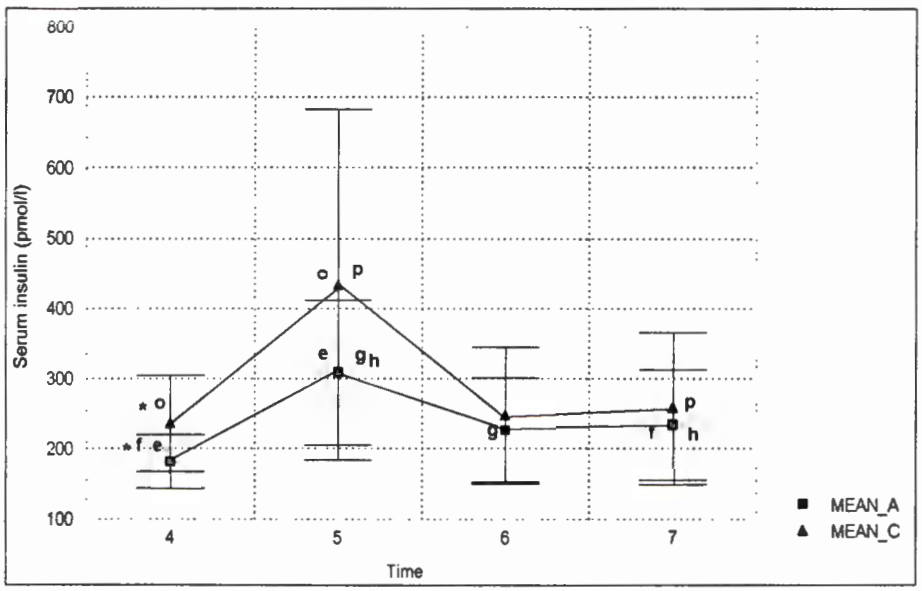



Figure 4.4 HGI intervention. Serum insulin values of athletes (A) and controls (C).

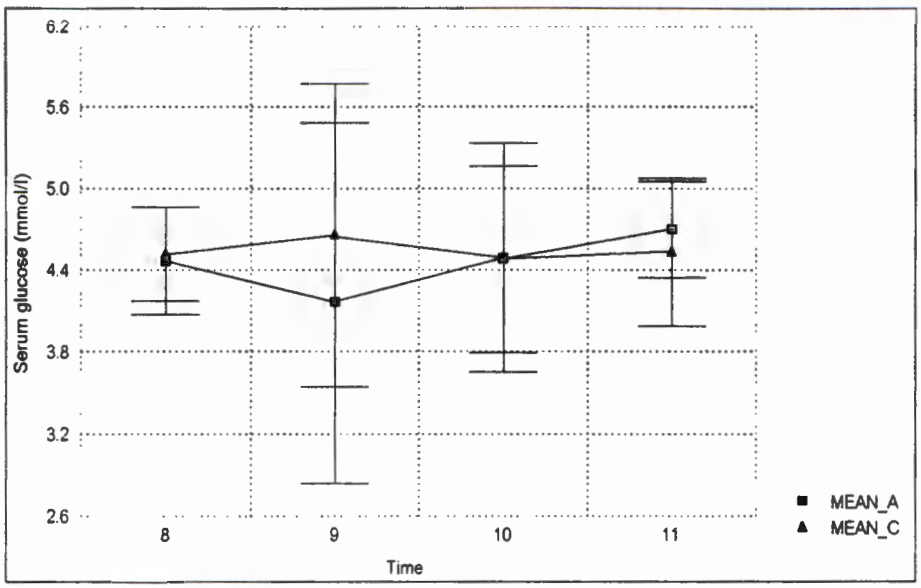



Figure 4.5 LGI intervention. Serum glucose values of athletes (A) and controls (C). (LGI – low glycaemic index pre-exercise meal).

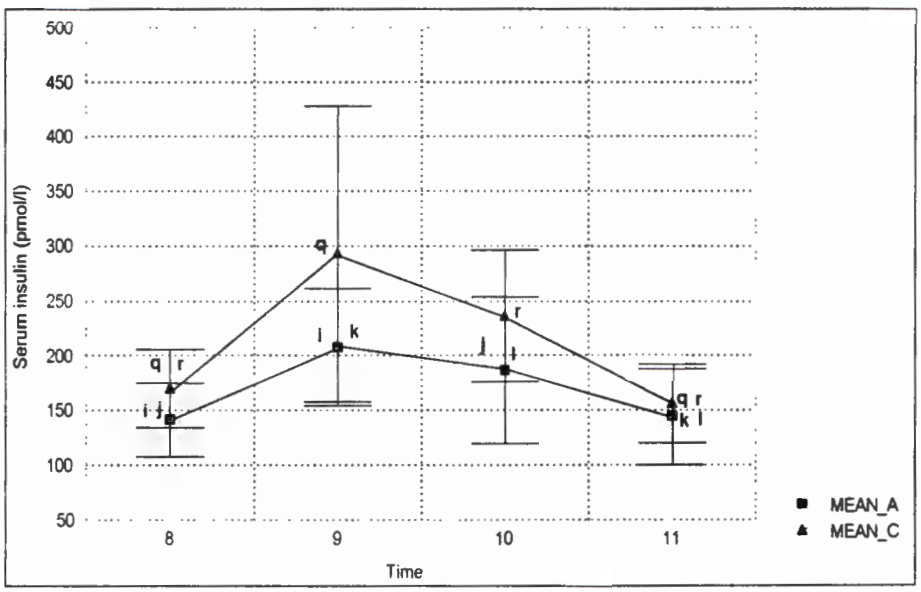
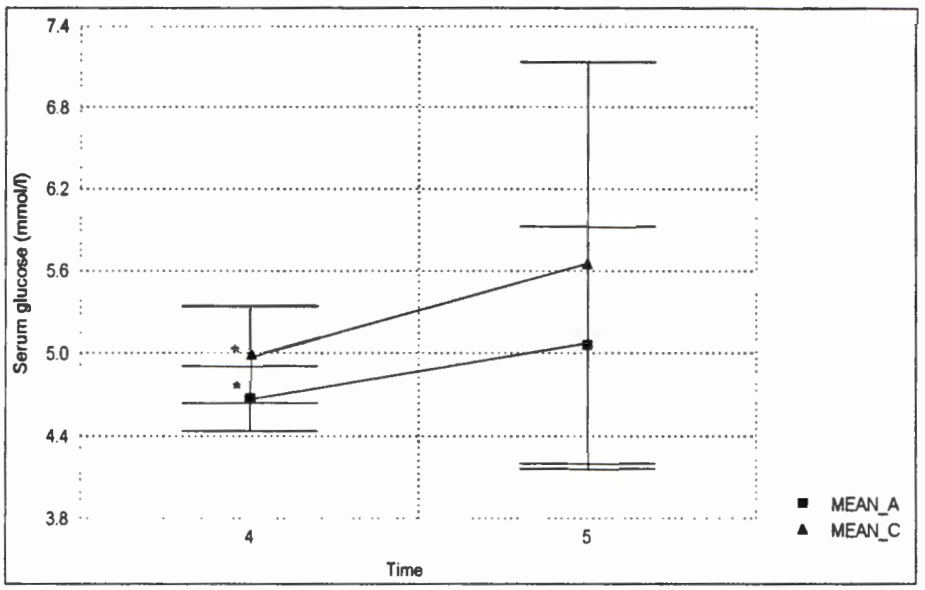


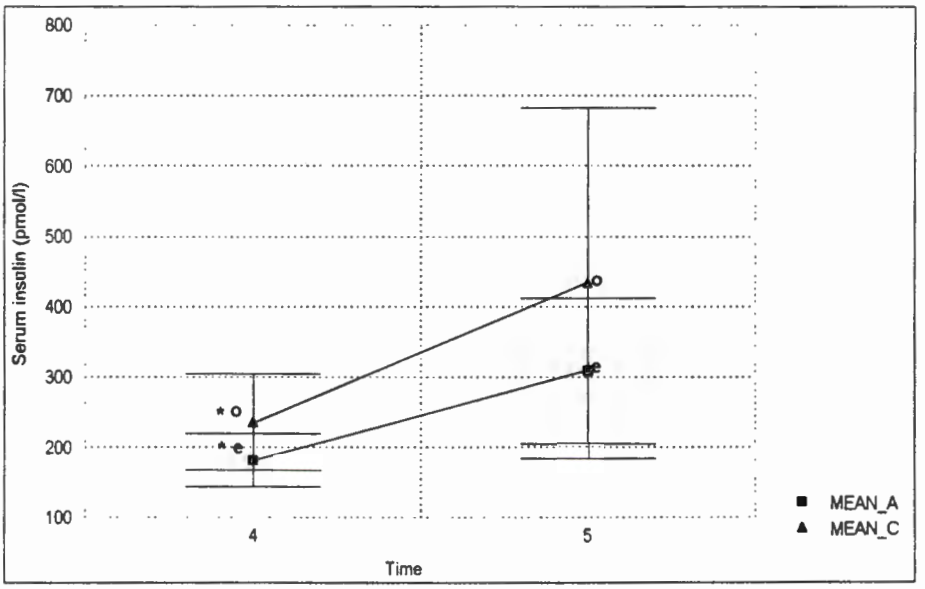

Figure 4.6 LGI intervention. Serum insulin values of athletes (A) and controls (C).

In the next section (Figures 4.7 – 4.22) the changes in serum glucose and insulin over a single time interval (the separate components of each intervention) will first be illustrated as a graph and then be discussed, to simplify interpretation. The two interventions with pre-exercise meals will be presented first.




$\Delta - \Delta = p > 0.05$

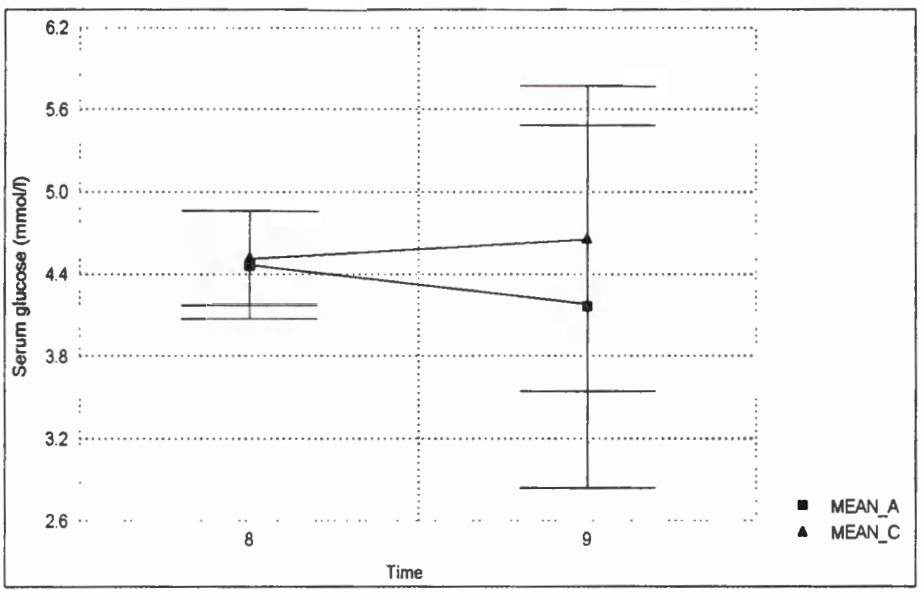
Figure 4.7 HGI intervention. Fasted and 50 min post-prandial serum glucose values of athletes (A) and controls (C)




$\Delta - \Delta = p > 0.05$

Figure 4.8 HGI intervention. Fasted and 50 min post-prandial serum insulin values of athletes (A) and controls (C).

The glucose values of both the athletes and controls increased after the consumption of the HGI meal but not significantly so (Fig. 4.7). The increase in serum glucose of the athletes did not differ significantly from the increase of the controls ($\Delta - \Delta = p > 0.05$). Therefore it does not seem as if long-term exercise (fitness) influences the glucose response after the ingestion of a HGI meal. The same can be said for the insulin responses (Fig. 4.8). The controls had significantly higher fasting glucose ($p = 0.02$) and insulin ($p = 0.032$) values than the athletes did.




$\Delta - \Delta = p > 0.05$

Figure 4.9 LGI intervention. Fasted and 50 min post-prandial serum glucose values of athletes (A) and controls (C).

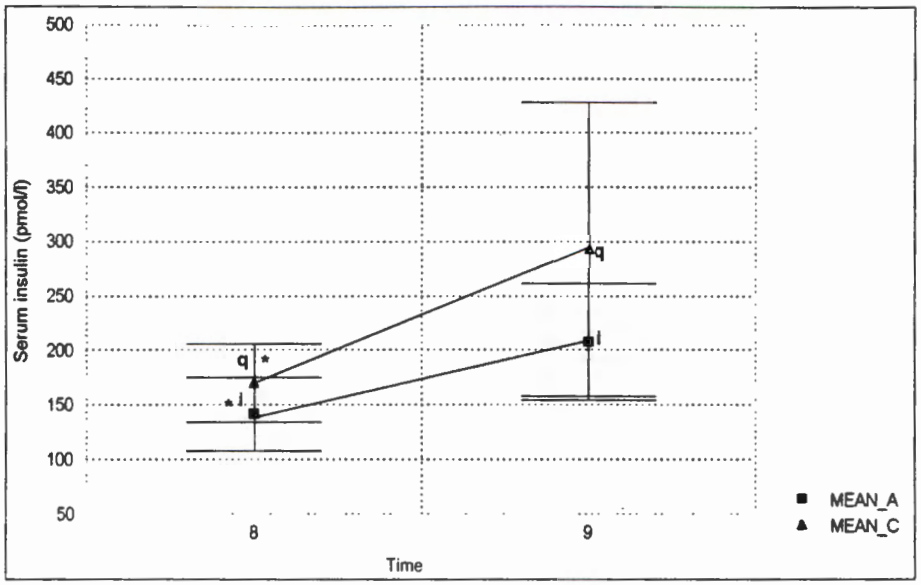



Figure 4.10 LGI intervention. Fasted and 50 min post-prandial serum insulin values of athletes (A) and controls (C).

Neither the post-prandial serum glucose values of the athletes nor the post-prandial serum glucose values of the controls differed significantly from the fasting values, although a slight increase was seen with the glucose values of the controls and a slight decrease in the glucose values the athletes (Fig. 4.9). Once again the glucose response of the athletes and controls after the ingestion of a LGI meal did not differ significantly ($\Delta - \Delta = p > 0.05$). Therefore it does not seem as if long-term exercise (fitness) influenced this glucose response either.

Insulin values were higher at T9 (50 min post-prandial) than the fasting value for both the athletes and controls but not significantly so (Fig. 4.10). The increase in serum insulin values from T8 to T9 also did not differ between the athletes and controls (this correlates with what was seen in the glucose curves).

The controls had a significantly higher ($p = 0.05$) fasting serum insulin value than the athletes did.

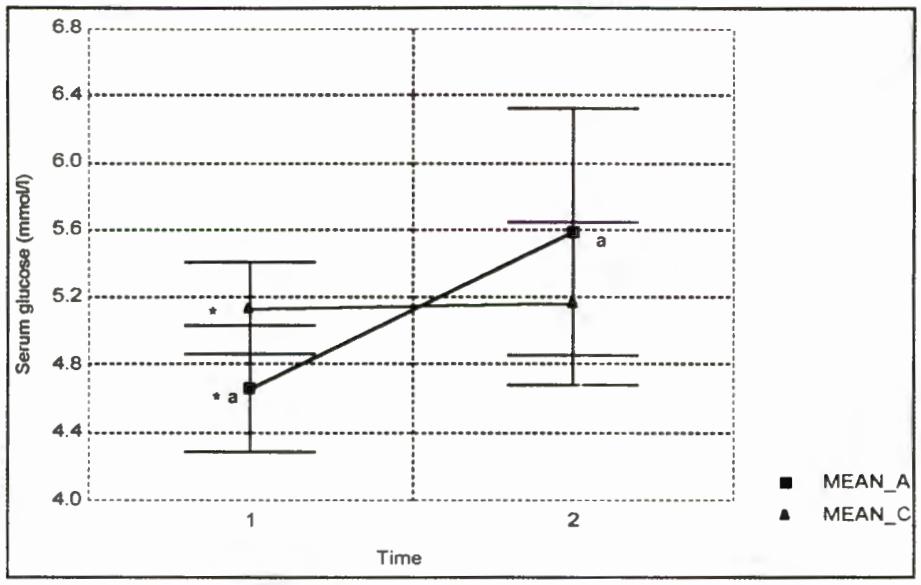



Figure 4.11 Baseline. Fasted and post-exercise serum glucose values of athletes (A) and controls (C).

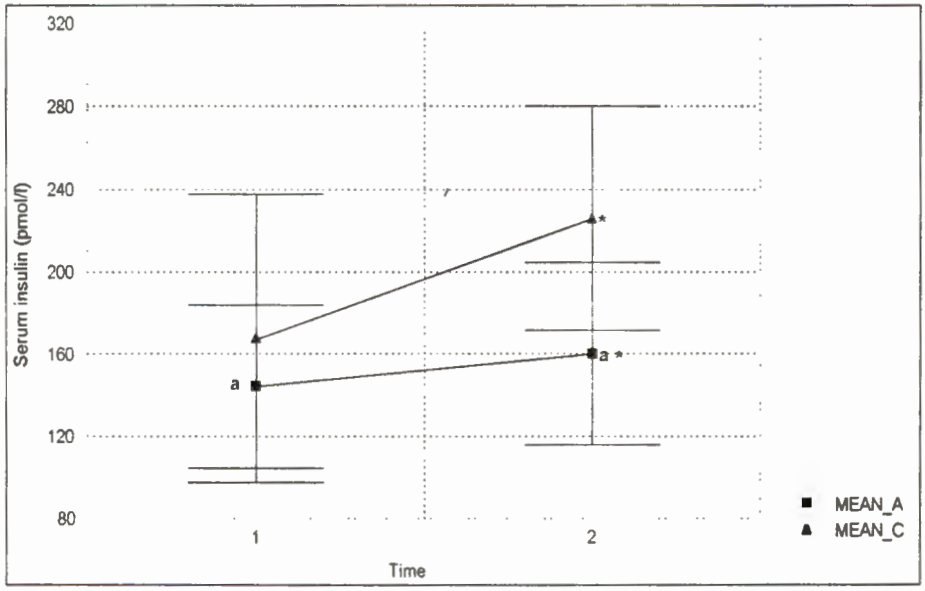


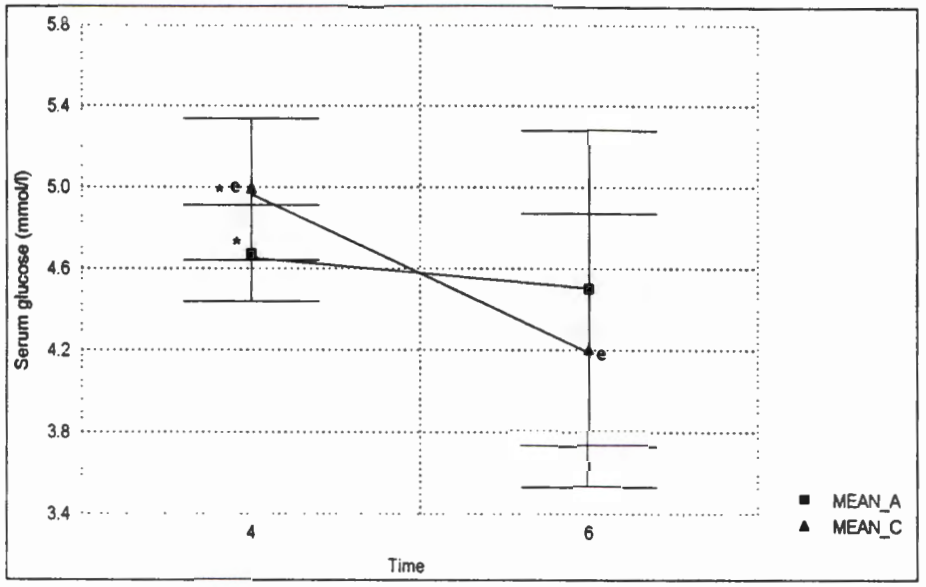

Figure 4.12 Baseline. Fasted and post-exercise serum insulin values of athletes (A) and controls (C).

The post-exercise glucose value of the athletes was significantly higher than the fasted value ($p = 0.00$). This observation suggests that acute exercise has a significant effect on the serum glucose of athletes. The post-exercise value of the controls did not differ significantly ($p > 0.05$) from the fasted value and could therefore be interpreted that acute exercise did not have a significant effect on the serum glucose of non-athletes (Fig. 4.11).

The change in serum glucose of the athletes between T1 and T2 differed significantly ($\Delta - \Delta = p = 0.00$) from the change in the serum glucose values of the controls from T1 to T2. This suggests that long-term exercise (fitness) might influence the effect of acute exercise on serum glucose.

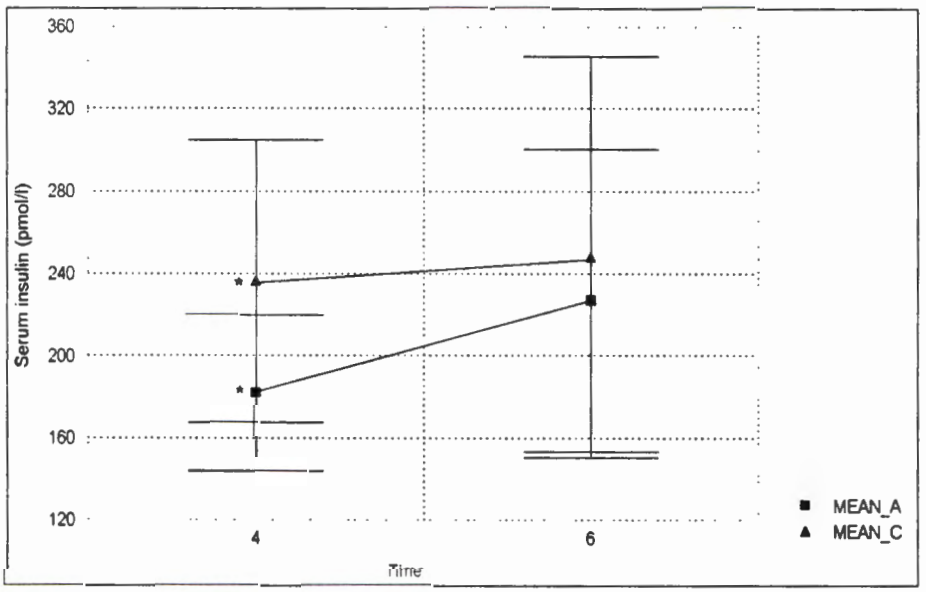
The controls had significantly higher fasting serum glucose values than the athletes ($p = 0.001$).

Acute exercise significantly increased serum insulin values of the athletes ($p = 0.018$) but not that of the controls (Fig. 4.12). The increase in the serum insulin values from T1 to T2 did not differ significantly between the athletes and controls ($\Delta - \Delta = p > 0.05$). Therefore, although long-term exercise (fitness) might influence glucose responses after acute exercise, it does not seem to have an effect on the insulin responses following acute exercise.




$\Delta - \Delta = p = 0.049$

Figure 4.13 HGI intervention. Fasted and post-exercise serum glucose values of athletes (A) and controls (C).




$\Delta - \Delta = p > 0.05$

Figure 4.14 HGI intervention. Fasted and post-exercise serum insulin values of athletes (A) and controls (C).

The mean serum glucose values of the athletes after the ingestion of a HGI pre-exercise meal and acute exercise (the post-exercise value) was lower than the fasting value but not significantly so. The mean post-exercise value of the controls was significantly lower ($p = 0.005$) than the fasting value (Fig. 4.13).

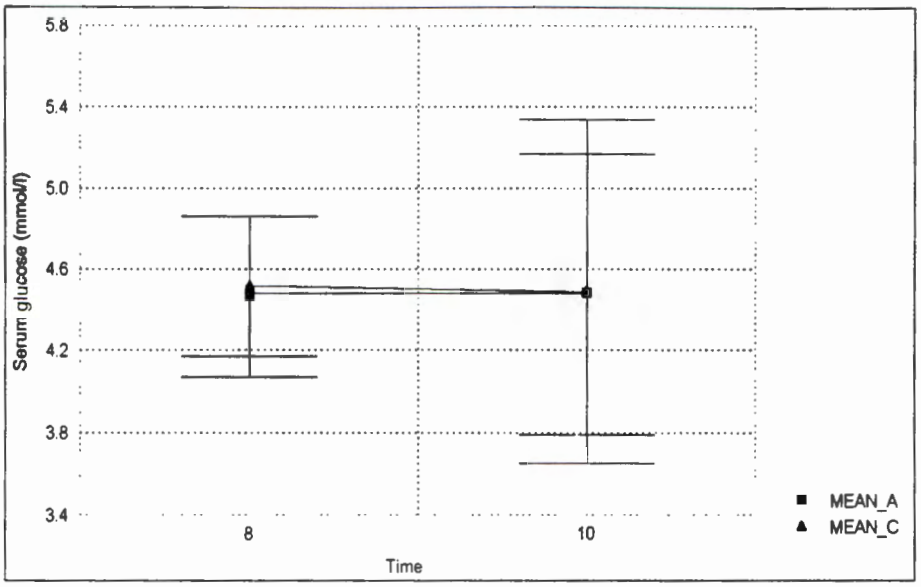
While the post-exercise glucose values were higher than the fasting values at the baseline intervention (Fig. 4.11), the post-exercise glucose values were lower than the fasting values with the ingestion of a HGI pre-exercise meal (Fig. 4.13). This was found in both the athletes and the controls.

The difference between the changes in serum glucose of the athletes and controls between T4 and T6 was significant ($\Delta - \Delta = p = 0.049$). It appears therefore as though long-term exercise might influence the glucose response after acute exercise that was preceded by a HGI meal.

The fasting serum glucose value of the controls was significantly higher than the serum glucose value of the athletes (also indicated in Fig. 4.7).

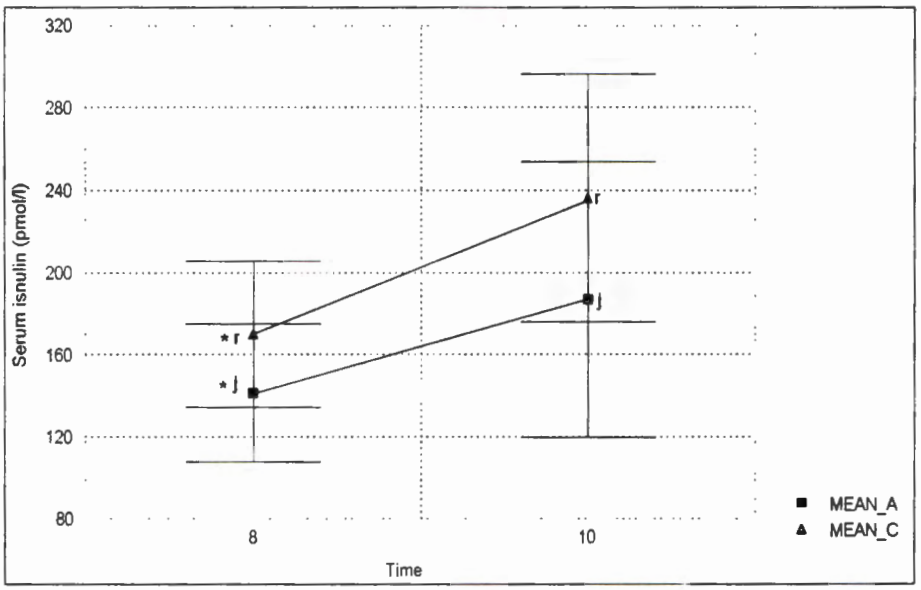
In both the athletes and controls the post-exercise insulin values were higher than the fasting value but not significantly so (Fig. 4.14). It must, however, be kept in mind that the serum insulin level at the onset of the exercise was not the fasted value but a significantly higher post-prandial value (as indicated in Fig. 4.4 and 4.8), therefore there was actually a drop in insulin levels during the exercise period.

The changes in serum insulin from T4 to T6 did not differ significantly between the athletes and controls ($\Delta - \Delta = p > 0.05$). The controls had significantly higher fasting serum insulin levels than the athletes did ($p = 0.032$).




$\Delta - \Delta = p > 0.05$

Figure 4.15 LGI intervention. Fasted and post-exercise serum glucose values of athletes (A) and controls (C).




$\Delta - \Delta = p > 0.05$

Figure 4.16 LGI intervention. Fasted and post-exercise serum insulin values of athletes (A) and controls (C).

No significant changes were seen between the post-exercise and fasted serum glucose values of either the athletes or the controls (Fig. 4.15). The post-exercise glucose value of the athletes was only slightly higher than the fasted value and the serum

glucose value of the controls was slightly lower than their fasted value. This differs from the responses found during the baseline intervention when the subjects exercised fasted (Fig. 4.11). It appears as though the ingestion of a LGI pre-exercise meal had a stabilising effect on the serum glucose responses of both the athletes and controls.

The post-exercise serum insulin values of both the athletes and controls were significantly higher than their fasted values ($p = 0.002$, $p = 0.001$, respectively) (Fig. 4.16). Again, keep in mind that the insulin levels prior to exercise were not the fasted levels but higher values due to the ingestion of a LGI pre-exercise meal (the post-prandial value). For both the athletes and controls the post-prandial values were significantly higher ($p = 0.00$, $p = 0.009$, respectively) than the fasted value (Fig. 4.6 and 4.10). Therefore insulin levels dropped during exercise in both the athletes and controls. The controls had significantly higher fasted serum insulin levels than the athletes ($p = 0.05$).

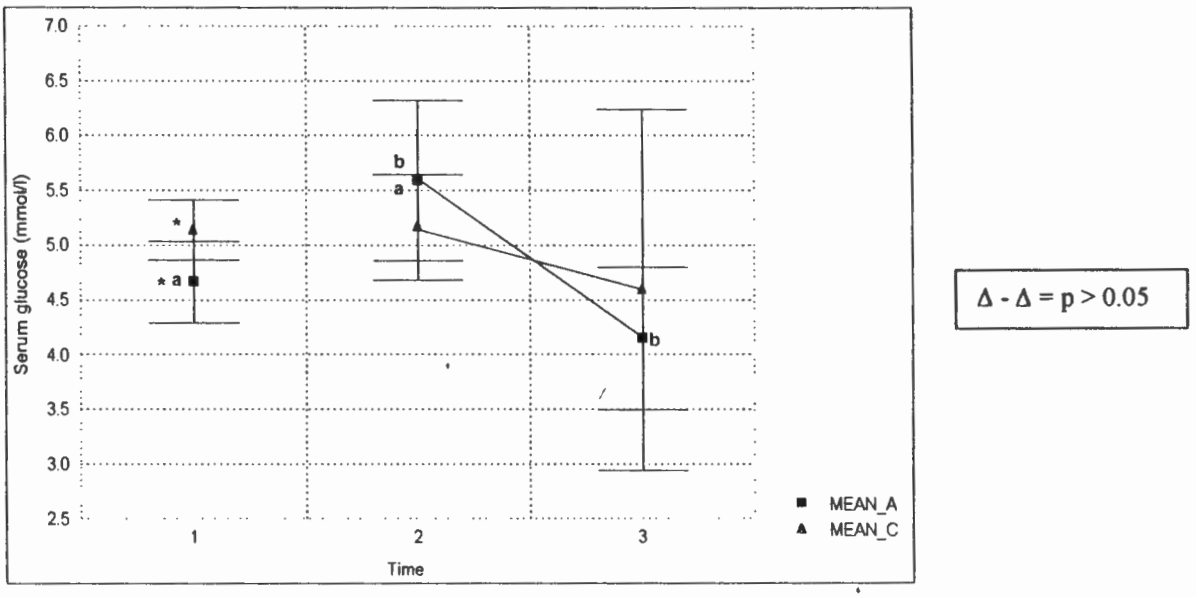



Figure 4.17 Baseline. Post-exercise and 30 min rest serum glucose values of athletes (A) and controls (C). Fasted glucose values are shown on the left.

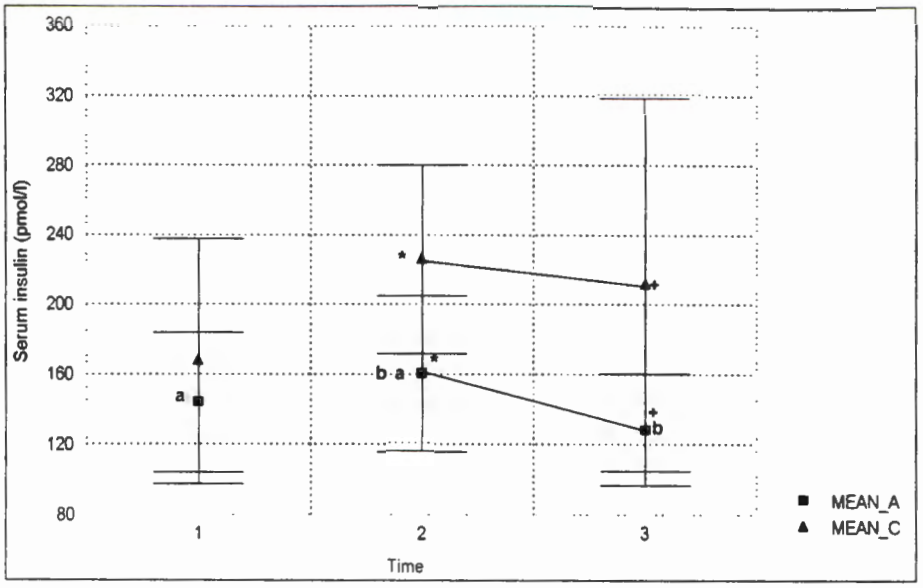



Figure 4.18 Baseline. Post-exercise and 30 min rest serum insulin values of athletes (A) and controls (C). Fasted insulin values are shown on the left.

The serum glucose value of the athletes after 30 min of rest was significantly lower than the serum glucose value just after exercise ($p = 0.00$ Fig. 4.17). The 30 min rest glucose value of the controls also tended to be lower than the post-exercise value but not significantly so. Neither the athletes' nor the controls' serum glucose values after 30 min of rest, differed significantly from the fasted value. The decrease in glucose values during the 30 min rest period did not differ significantly between the athletes and controls ($\Delta - \Delta = p > 0.05$). Therefore long-term exercise did not seem to influence the recovery of serum glucose values during the 30 min rest period after acute exercise.

As was seen with the serum glucose of the athletes, the serum insulin value after 30 min of rest was also significantly lower ($p = 0.005$) than the value just after exercise. The value also decreased in the control group but was not significantly lower (Fig. 4.18).

For both the athletes and controls the insulin levels recovered to a value that did not differ significantly from their respective fasted values.

The decrease in serum insulin values during the 30 min rest period did not differ significantly between the athletes and controls ($\Delta - \Delta = p > 0.05$). Therefore long-term exercise did not seem to influence the recovery of serum insulin values during the 30 min rest period after acute exercise.

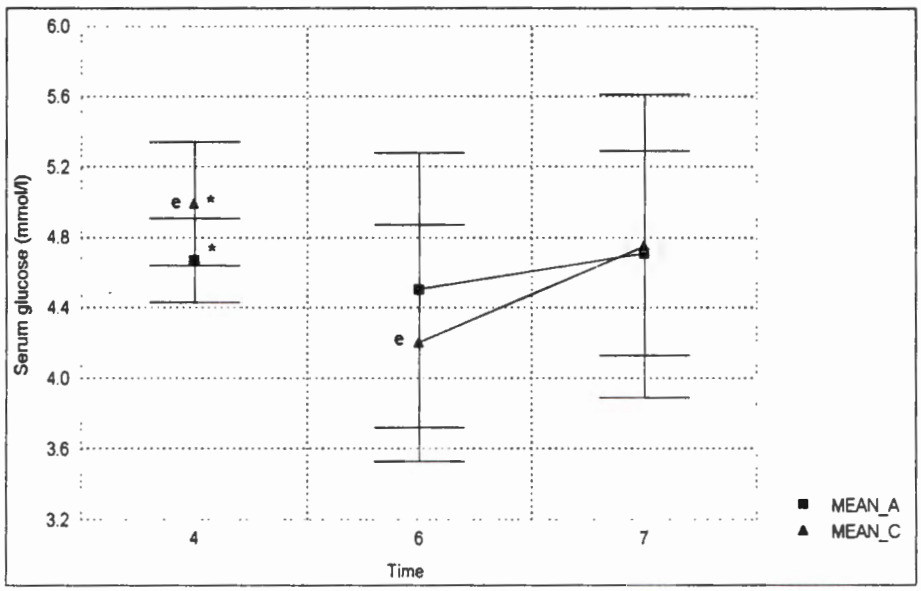



Figure 4.19 HGI intervention. Post-exercise and 30 min rest serum glucose values of athletes (A) and controls (C). Fasted glucose values are shown on the left.

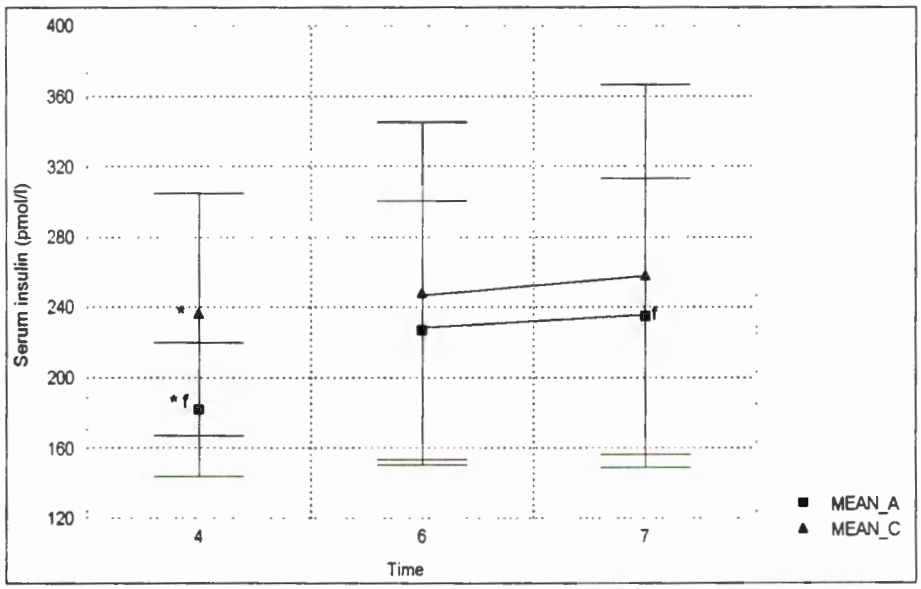
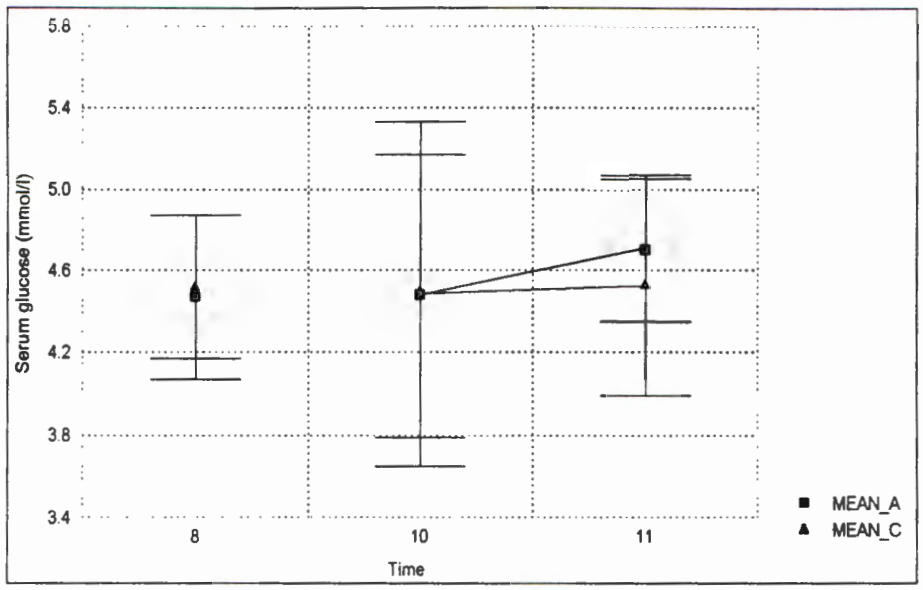



Figure 4.20 HGI intervention. Post-exercise and 30 min rest serum insulin values of athletes (A) and controls (C). Fasted insulin values are shown on the left.

Both the serum glucose values of the athletes and controls increased after 30 min of rest when compared to the serum glucose value just after exercise, but not significantly so (Fig. 4.19). This increase did not differ significantly between the athletes and controls either ($\Delta - \Delta = p > 0.05$). Long-term exercise therefore does not seem to influence the ability of serum glucose to recover after an acute exercise test. The values after 30 min of rest did not differ significantly for either the controls or the athletes from the fasted values.

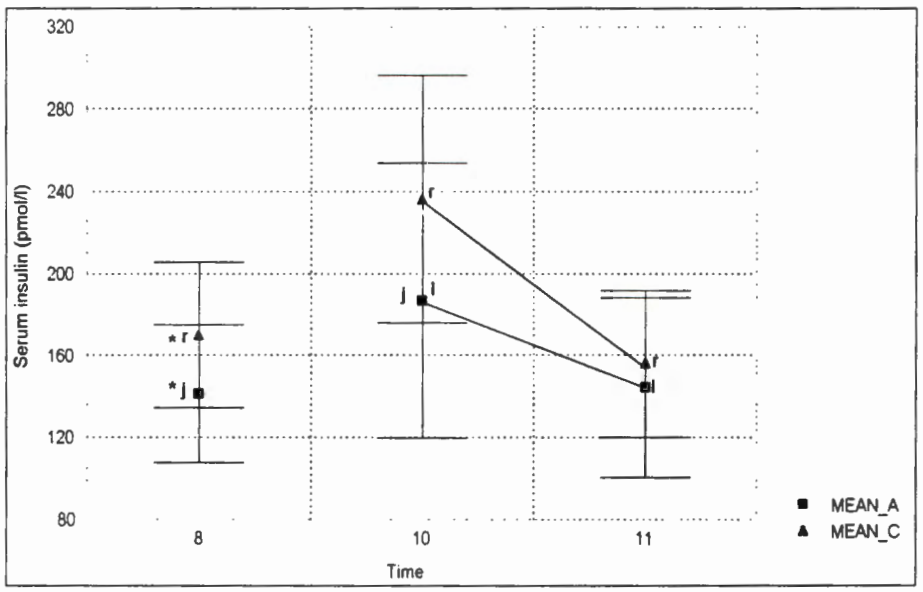
It is important to note that the post-exercise values for both the athletes and controls were higher than the fasted values when the subjects exercised fasted (Fig. 4.11) but were lower than the fasted value when a HGI pre-exercise meal was given (Fig. 4.13). In both cases, however, the serum glucose returned to a value not significantly different from the fasted value, no matter if the post-exercise value was higher or lower. This was the case for both the athletes and controls.

Neither the serum insulin values of the athletes or the controls after 30 min of rest, differed significantly from the value just after exercise (Fig. 4.20). The same trend was seen as with the glucose responses when subjects exercised fasted when compared to exercise after the ingestion of a HGI meal. While the insulin levels decreased during rest after exercising fasted (Fig. 4.18), the insulin levels increased for both the athletes and controls (to a value that did not differ significantly from the fasted values) during the 30 min rest period when they ingested a HGI meal prior to exercise.




$\Delta - \Delta = p > 0.05$

Figure 4.21 LGI intervention. Post-exercise and 30 min rest serum glucose values of athletes (A) and controls (C). Fasted glucose values are shown on the left.




$\Delta - \Delta = p = 0.033$

Figure 4.22 LGI intervention. Post-exercise and 30 min rest serum insulin values for athletes (A) and controls (C). Fasted insulin values are shown on the left.

The serum glucose values after 30 min of rest did not differ significantly from the glucose values just after exercise nor did they differ significantly from the fasted values before the LGI meal was consumed (Fig. 4.21). This was the case for both the athletes and controls. Once again it seems as though a LGI pre-exercise meal had a stabilising effect on all the glucose responses in this intervention: post-prandial (T9), post-exercise (T10) and after 30 min of rest (T11).

The serum insulin values of both the athletes and controls after 30 min of rest, were significantly lower than their respective post-exercise values ($p = 0.001$, $p = 0.00$, respectively) (Fig. 4.22). The serum insulin levels of the athletes after 30 min of rest, recovered (declined) to a value that did not differ significantly from the fasted value, but the 30 min rest insulin value of the controls was significantly lower than their fasted value ($p = 0.014$).

The decline in insulin levels during the 30 min rest period differed significantly between the athletes and controls ($\Delta - \Delta = p = 0.033$). It seems as though long-term exercise (fitness) might influence insulin recovery after an acute exercise test when a LGI meal was ingested prior to exercise.

#### 4.2.4.1 Summary of changes in serum glucose and insulin during all interventions.

The next section summarises the glucose and insulin values of the atheletes and controls for the three interventions. Fig. 4.23 shows the glucose values of the athletes for all three interventions, and Fig. 4.24 shows the glucose values of the controls for all three interventions. Figures 4.25 and 4.26 show the insulin values of the athletes and controls over the three interventions respectively. In this way the glucose and insulin responses of the different interventions can be compared with each other (for the athletes and controls separately). It will also show how the athletes' and controls' glucose and insulin control mechanisms responded to acute exercise without a meal, acute exercise with a HGI pre-exercise meal, and acute exercise with a LGI pre-exercise meal.

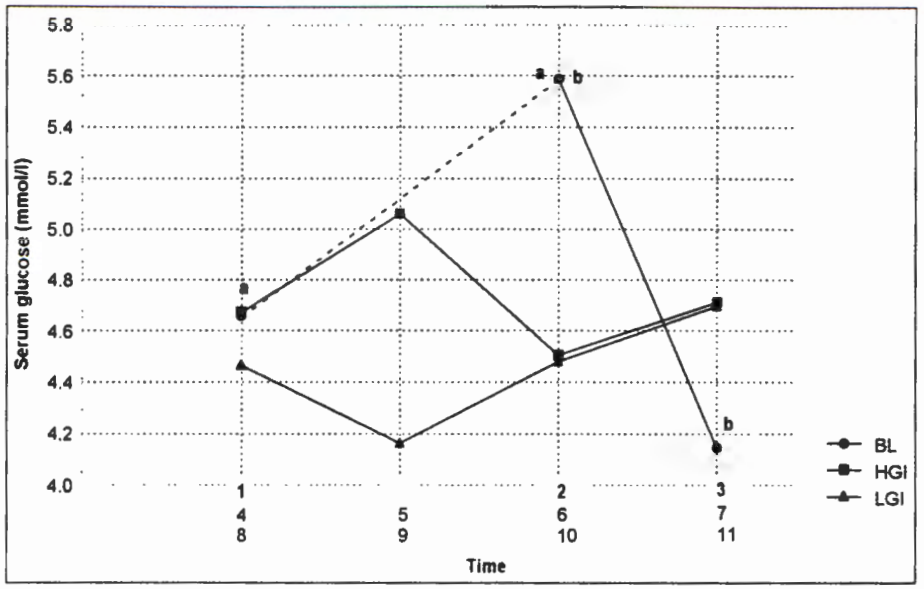



Figure 4.23 Serum glucose responses of the athletes for all three interventions.

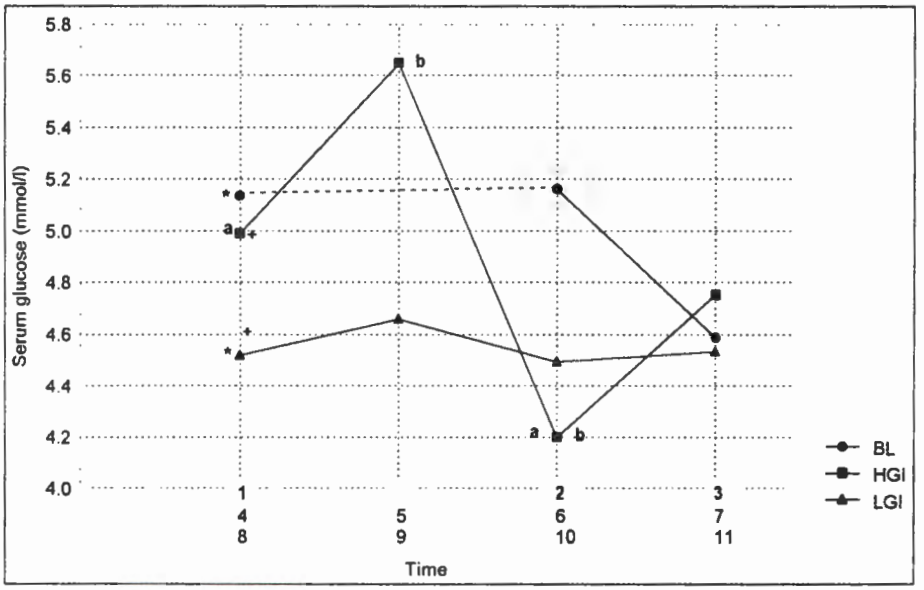



Figure 4.24 Serum glucose responses of the controls for all three interventions.

The serum glucose values of the athletes at baseline (exercise without pre-exercise meal) showed significant changes over the intervention, while no significant changes were seen in serum glucose values with the ingestion of either a HGI or LGI pre-exercise meal (Fig. 4.23).

The serum glucose values of the controls on the other hand, showed the largest fluctuations when a HGI pre-exercise meal was ingested. No significant changes occurred during the baseline or LGI intervention (Fig. 4.24).

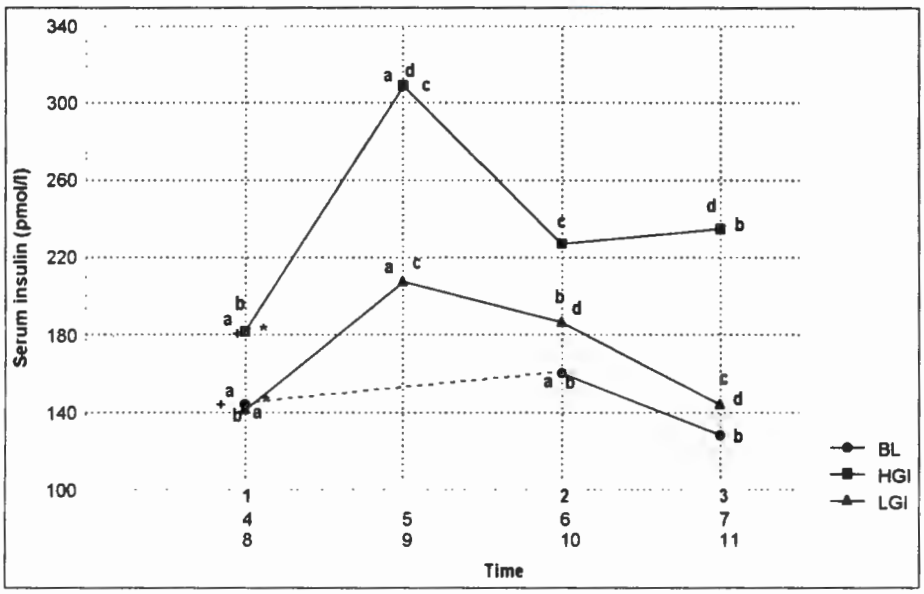



Figure 4.25 Serum insulin responses of the athletes for all three interventions.

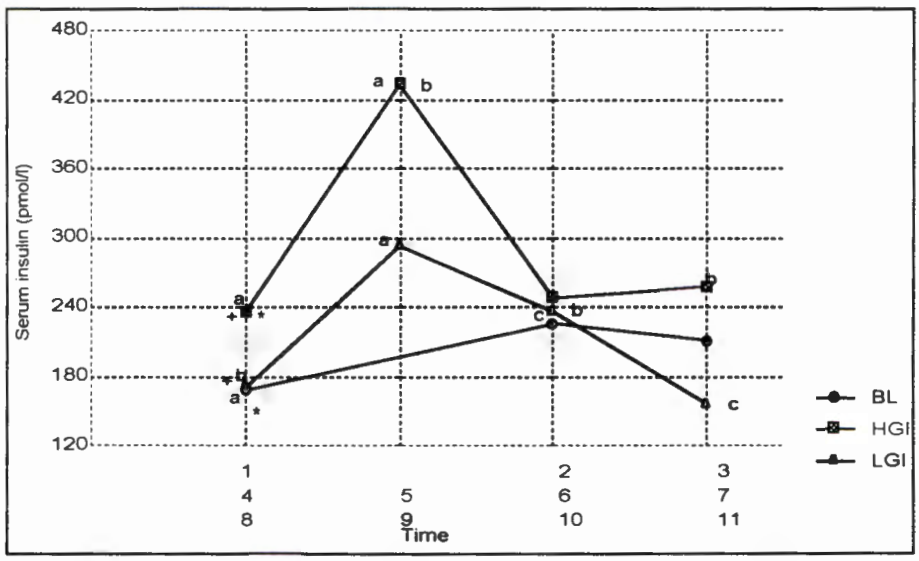



Figure 4.26 Serum insulin responses of the controls for all three interventions.

Significant changes occurred during all three interventions in the serum insulin values of the athletes. Note that the HGI intervention resulted in higher insulin values when compared to the LGI or baseline interventions (Fig. 4.25).

The serum insulin values of the controls changed significantly during the HGI and LGI interventions but not during the baseline intervention. The HGI intervention also resulted in higher insulin values, for the controls, when compared to the LGI and baseline interventions (Fig. 4.26).

### 4.2.5 Correlations between glucose and insulin and other variables

Spearman correlations between glucose and insulin and other variables were done to determine whether glucose and insulin are influenced by other variables and, if so, to what extent. In Table 4.8 and 4.9 the correlation between glucose and insulin respectively and other variables is given.

Table 4.8 Correlations between glucose (mmol/l) and other variables.

| | | Time interventions | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.62* | - | - | - | - | - | - | - | - |
| | Alb | - | - | 0.56* | - | - | - | - | - | - | - | - |
| | Insulin | 0.61* | - | 0.7** | - | - | - | 0.53* | - | 0.58* | 0.6* | - |
| | TG | - | - | 0.66** | - | - | - | - | 0.53* | - | 0.66* | - |
| Controls | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.82** | - | - | - | - | - | - | - | - |
| | Alb | - | - | 0.83** | - | - | - | - | - | - | - | - |
| | Insulin | - | - | 0.84** | - | 0.8** | - | - | - | 0.85** | 0.6* | 0.61* |
| | TG | 0.77** | - | - | 0.8** | - | - | - | - | - | - | 0.64* |

* - significant ($p \leq 0.05$) ** - significant ($p \leq 0.02$) T - time ND - not determined

- no significance T prot - total protein TG - Triglycerides Alb - albumin

Glucose values showed the strongest correlations with insulin values (r = 0.53 to 0.85). There were also correlations with triglyceride values for some time intervals. At T3 (baseline – post exercise) interval both the athletes' and controls' glucose values correlated with total protein and albumin levels, but not at any other time.

Table 4.9 Correlations between insulin (pmol/l) and other variables.

| | | Time interventions | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | - | - | - | - | - | - | - | - | - |
| | Alb | - | - | - | - | - | - | - | - | - | - | - |
| | Glc | 0.61* | - | 0.7** | - | - | - | 0.53* | - | 0.58* | 0.6* | - |
| | TG | - | - | - | - | - | - | - | 0.6* | - | 0.72** | 0.85** |
| Controls | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.77* | - | - | - | - | 0.61* | - | - | 0.62* |
| | Alb | - | - | 0.81** | - | - | - | - | - | - | - | - |
| | Glc | - | - | 0.84** | - | 0.8** | - | - | - | 0.85** | 0.6* | 0.61* |
| | TG | - | - | 0.59* | - | - | - | - | - | - | 0.64* | - |

* - significant ($p \leq 0.05$)

** - significant ($p \leq 0.02$)

- no significance

Glc - glucose

T - time

T prot - total protein

Alb - albumin

ND - not determined

TG - Triglycerides

Strong correlations were found between the insulin and glucose values of both the athletes and controls as mentioned previously. Insulin and triglyceride values also correlated during the LGI interventions in the athletes (r = 0.6 to 0.85) but there were no other correlations between insulin and the other variables in the athlete's group. The control group on the other hand showed stronger correlations between the insulin

values and total protein (r = 0.61 to 0.77) than between insulin and triglyceride values (R = 0.59 to 0.64).

Both the insulin and glucose values showed more correlations with the other variables during the LGI intervention and baseline than during the HGI intervention. In fact, the HGI intervention showed very few correlations when compared to the other two interventions, which may be ascribed to the large fluctuations in glucose and insulin values during the HGI intervention.

### 4.2.6 Correlations between glucose and insulin and other variables after adjustment for body mass index

Because of the significant difference in BMI between athletes and controls and the possibility that the higher BMI of the controls might have influenced biochemical variables, Spearman correlation coefficients were also determined after adjustment for BMI. Table 4.10 gives the correlations between glucose and the same variables as in Table 4.8, but after adjustment for BMI. Table 4.11 represents the correlations between insulin and the same variables as in Table 4.9, also after adjustment for BMI.

When adjusted for BMI, the correlations were slightly stronger for both glucose and insulin. The athletes' glucose and insulin values followed the same trend as when values were not adjusted for BMI: more correlations during baseline and the LGI intervention and less during the HGI intervention. The situation changed slightly for the controls' glucose and insulin values. They still showed many correlations during the baseline intervention, but their insulin values during the HGI intervention correlated more often with the other variables, than did their insulin values during the LGI intervention.

Table 4.10 Correlations between glucose (mmol/l) and other variables, after adjustment for BMI.

| | | Time interventions | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.6** | - | - | - | - | - | - | - | - |
| | Alb | - | - | 0.55* | - | - | - | - | - | - | - | - |
| | Insulin | 0.67** | - | 0.69** | - | 0.6** | - | - | - | 0.66** | 0.74** | - |
| | TG | 0.62** | 0.53** | - | - | - | - | - | 0.7** | - | 0.69** | - |
| Controls | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.94** | - | - | - | - | - | - | - | - |
| | Alb | - | - | 0.95** | - | - | - | - | - | - | - | - |
| | Insulin | - | - | 0.75** | - | 0.86** | - | - | - | 0.74** | - | 0.71** |
| | TG | 0.72** | 0.62* | 0.74** | - | - | - | - | - | - | - | - |

* - significant ($p \leq 0.05$)

** - significant ($p \leq 0.02$)

- no significance

T – time

T prot - total protein

Alb - albumin

ND - not determined

TG - Triglycerides

Table 4.11 Correlations between insulin (pmol/l) and other variables after adjustment for BMI.

| | | Time interventions | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Baseline | | | High glycaemic | | | | Low glycaemic | | | |
| | | T1 Fasted | T2 After acute exercise | T3 30 min rest | T4 Fasted | T5 50 min after meal | T6 After acute exercise | T7 30 min rest | T8 Fasted | T9 50 min after meal | T10 After acute exercise | T11 30 min rest |
| Athletes | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | 0.54* | - | - | - | - | - | - | - | - |
| | Alb | - | - | - | - | - | - | - | - | - | - | - |
| | Glc | 0.67** | - | 0.69** | - | 0.6** | - | - | - | 0.66** | 0.74** | - |
| | TG | 0.67** | 0.58* | - | - | - | - | - | 0.62** | - | 0.82** | 0.68** |
| Controls | Weight | - | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND |
| | T prot | - | - | - | - | - | 0.7** | - | - | - | - | - |
| | Alb | - | - | - | - | - | 0.71** | - | - | - | - | - |
| | Glc | - | - | 0.75** | - | 0.86** | - | - | - | 0.74** | - | 0.71** |
| | TG | - | - | 0.8** | - | 0.67** | - | - | - | - | - | - |

* - significant ($p \leq 0.05$)

** - significant ($p \leq 0.02$)

- no significance

Glc - glucose

T - time

T prot - total protein

Alb - albumin

ND - not determined

TG - Triglycerides

# CHAPTER 5 DISCUSSION

## 5.1 INTRODUCTION

In this chapter, the results given in Chapter 4 will be discussed. The results related to the exercise interventions (heart rate, resistance, PWC and exercise time) will be discussed in Section 5.2, the glucose and insulin responses will be discussed in Section 5.3 and the correlations with other variables in Section 5.4.

Carbohydrate, protein and fat metabolism are controlled and integrated by two functional states or periods: the **post-absorptive state** and the **absorptive state** (Vander *et al.*, 1990). The post-absorptive state is the period during which the gastrointestinal tract is empty of nutrients and energy must be supplied by the body's own stores. The absorptive state is the period during which ingested nutrients are entering the blood from the gastrointestinal tract. During the absorptive period, some of the ingested nutrients supply the energy needs of the body and the remainder is added to the body's energy stores, to be called upon during the next post-absorptive period.

Since the subjects exercised in the post-absorptive state during the baseline intervention (fasted) and in the absorptive state during the HGI and LGI interventions (pre-exercise meal) the factors affecting the serum glucose and insulin values differed as illustrated in Figures 5.1 and 5.2. Since insulin is the most abundant hormone and its effects so important and widespread, the term post-absorptive state could also be replaced with "results of decreased insulin" and the term absorptive state with "actions of insulin" (Vander *et al.*, 1990).

There are still so many confounding variables affecting the results that the reasons for the glucose and insulin responses can only be speculated upon.

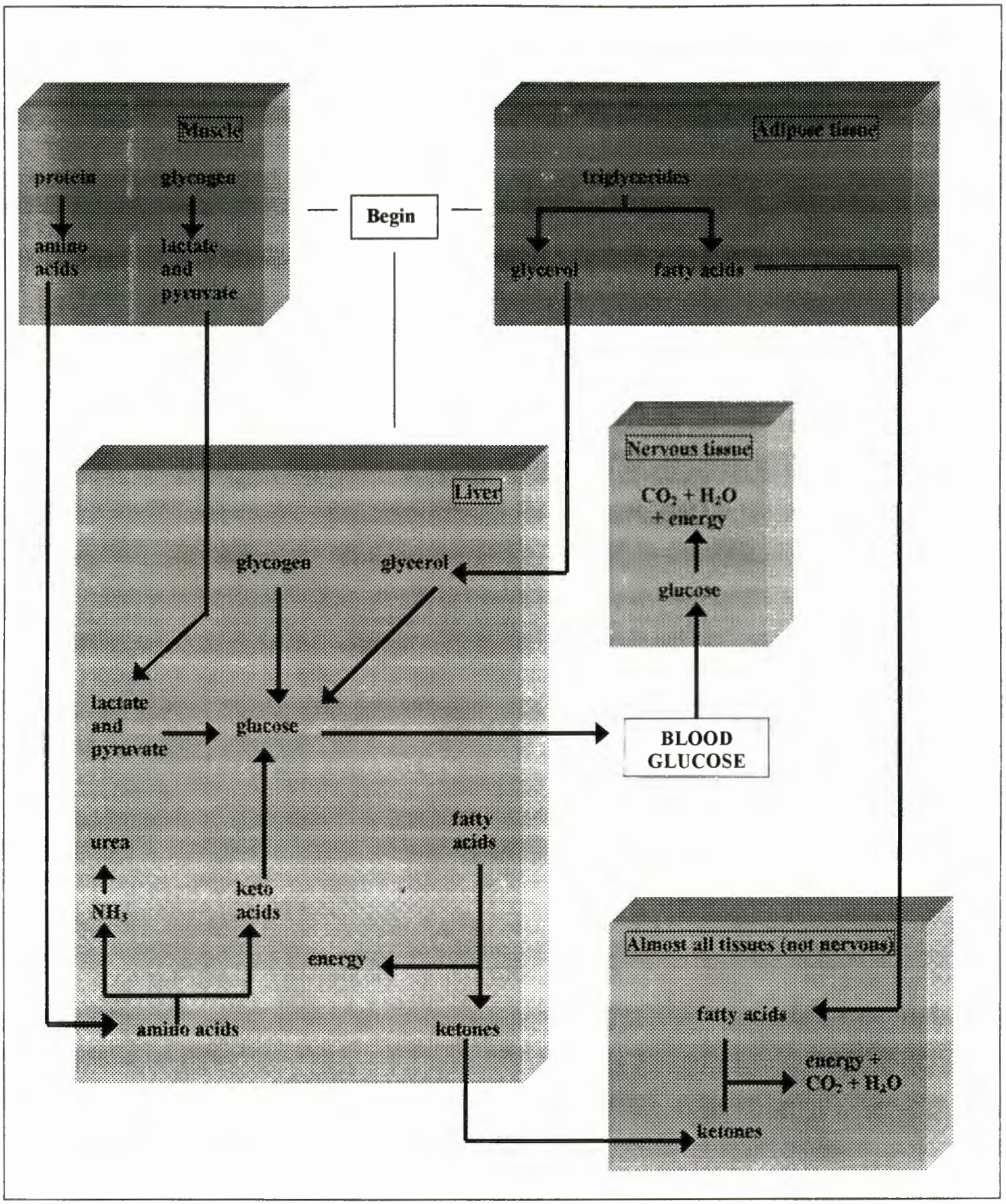



Fig. 5.1 Post-absorptive state (Vander *et al.*, 1990).

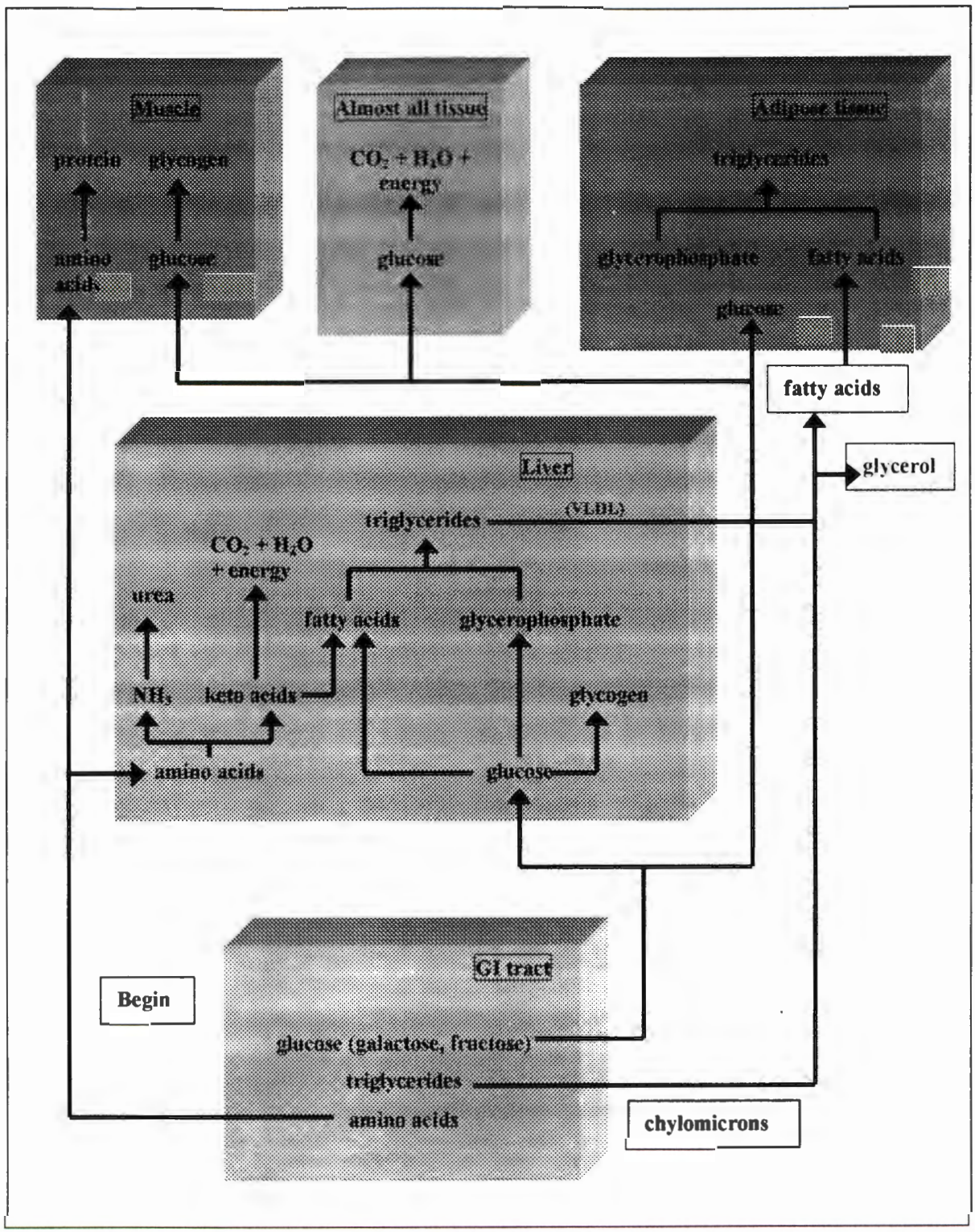



Fig. 5.2 Absorptive state (Vander *et al.*, 1990).

## 5.2 EXERCISE-ASSOCIATED VARIABLES

When discussing the variables associated with exercise and comparing these to comparable values in available literature (studies on GI and exercise), it should be taken into account that the time of exercise in this study was much shorter than the other studies cited in the literature (Alberici *et al.*, 1993; De Marco *et al.*, 1999; Febbraio & Stewart, 1996; Kirwan *et al.*, 1998; Sparks *et al.*, 1998; Thomas *et al.*, 1991; Wee *et al.*, 1999). The exercise time in this study was 15 – 20 min while the shortest time found in other studies was 1 hour. No studies could be found that examined the effect of the GI of a pre-exercise meal on exercise of this short (15 – 20 min) duration. This is important because of the significant effect this difference in exercise time might have on the glycogen stores of the body. The reason for the shorter endurance time is because of the increasing resistance the subjects had to cycle against.

Because of the short exercise time in this study, it could be argued that, although the subjects reported exhaustion, the muscle and liver glycogen of the subjects were most likely not depleted, while in exercise sessions of 1 hour or more, as in other studies glycogen stores were probably depleted.

According to McArdle *et al.* (1996) the average amount of total glycogen in the body of a well-nourished individual is 375 – 475 g. This presents 6 375 – 8 075 kJ (1 g CHO = 17 kJ). Table 5.1 shows the different fractions of CHO in the body.

If the energy expenditure of this study is compared to the amount of energy usually available from glycogen stores in the body, it could theoretically be calculated if the subjects depleted their glycogen stores or not.

Table 5.1 CHO fractions in the well-nourished individual (adapted from McArdle *et al.*, 1996).

| Fraction | Amount of CHO (g) | Energy content (kJ) |
|---|---|---|
| Total | 375 - 475 | 6 375 – 8 075 |
| Muscle | 325 | 5 525 |
| Liver | 90 - 110 | 1 530 – 1 870 |
| Blood | 15 - 20 | 255 - 340 |

CHO – carbohydrate kJ – kilojoule

The energy expenditure was calculated to be approximately 42 kJ/min (for very heavy exercise) according to a scale that classifies exercise according to intensity (McArdle *et al.*, 1996). If this factor of 42 is multiplied by the minutes the subjects exercised, the approximate total amount of energy expended during each intervention can be calculated. These values are given in Table 5.2.

Table 5.2 Total amount of energy expended during the exercise sessions.

| | Intervention | Time to exhaustion (min) | Energy expended (kJ) (exercise time x 42 kJ) |
|---|---|---|---|
| Athletes | Baseline | 19.73 | 828 |
| | HGI | 14.47 | 608 |
| | LGI | 15.2 | 638 |
| Controls | Baseline | 14.45 | 607 |
| | HGI | 13.09 | 550 |
| | LGI | 14.55 | 611 |

HGI – high glycaemic index LGI – low glycaemic index

min – minute kJ – kilojoule

According to the values in the table, the highest amount of energy expended during the whole study was 828 kJ by the athletes during the baseline intervention, which is not

nearly the value of glycogen content in the body. It is also important to remember that the glycogen contents given in Table 5.1 are for "normal" well-nourished individuals and not for endurance trained athletes. The glycogen content of these athletes would be even more, because of their known ability to increase their glycogen stores (McArdle *et al.*, 1996).

It must also be taken into consideration that during interventions II and III a pre-exercise meal was given that would have provided a further source of energy (HGI: 960 kJ and LGI: 980 kJ), therefore even less of the muscle glycogen would have been needed for the exercise.

When the results of this study are compared to those reported in the literature, the following should be noted:

- For both athletes and controls, a higher resistance, PWC and longer exercise times were found when they exercised fasted compared to when a pre-exercise meal was ingested. The difference between the baseline values for these variables and the HGI intervention values was significant.

- For both athletes and controls, a higher resistance, PWC and longer exercise times were found when a LGI pre-exercise meal was ingested compared to when a HGI meal was ingested. These differences were, however, not significant.

- Alberici *et al.* (1993), Febbraio and Stewart (1996), Sparks *et al.* (1998) and Wee *et al.* (1999) found no significant difference in performance of athletes when LGI and HGI pre-exercise meals were compared.

- De Marco *et al.* (1999), Kirwan *et al.* (1998) and Thomas *et al.* (1991) on the other hand found that the ingestion of a LGI compared to a HGI pre-exercise meal resulted

in significantly better performance, increased exercise capacity and longer endurance times.

As has been discussed, the aim of CHO supplementation is to provide an additional source of CHO in the form of blood glucose late in exercise when liver and muscle glycogen stores are low. This supplementation is therefore extremely important in exercise where glycogen stores are depleted. A central question that must be answered, is: How does this affect exercise when glycogen stores are not depleted, as was probably the case in this study?

Figures 4.23 and 4.24 represent the serum glucose values of the athletes and controls respectively. Note that the serum glucose values of both the athletes and controls at the end of exercise (T2, 6 and 10) were significantly higher during the baseline intervention (fasted state) than when a pre-exercise meal was given (the possible reason for this is discussed in the next section). If the exercise variables, such as PWC, resistance and time of exercise of the three interventions are compared (Table 4.4), then all of the above mentioned variables had the highest values when the subjects exercised fasted (Baseline).

It therefore seems as if the subjects' time to exhaustion were the longest when they had the highest serum glucose level at the end of the exercise. This is therefore consistent with the literature that states that high levels of serum glucose late in exercise may delay exhaustion. This might suggest that the serum glucose level at the end of exercise *per se* is the important factor and not the source of this glucose. If the subjects in this study had depleted their glycogen stores, however, the picture would probably have looked completely different. When glycogen stores are depleted, subjects have to rely on the extra energy from the pre-exercise meal to continue their exercise session. Therefore, a possible reason why subjects could exercise longer with the ingestion of a LGI pre-exercise meal compared to fasting (as was seen in the above-mentioned literature), is because the meal provided extra energy to be used after depletion of glycogen stores. The possible reason why subjects could exercise longer after the ingestion of a LGI

compared to a HGI pre-exercise meal (which also provides extra energy), is that the LGI meal caused increased levels of serum glucose late in exercise which was not found with the ingestion of a HGI pre-exercise meal.

## 5.3 GLUCOSE AND INSULIN VALUES

The objective of this study was to determine the effect of a pre-exercise meal with either a LGI or HGI on the glucose and insulin responses during acute exercise and after 30 min of rest in elite male athletes and controls.

**Fasting**

The controls had significantly higher fasting glucose and insulin values than the athletes for almost all the interventions. This might be explained by the fact that endurance exercise increases insulin sensitivity (Donahue *et al.*, 1988; Vander *et al.*, 1990). This might be due to several factors, among them increased numbers of insulin receptors, a change in body composition with a reduction of adipose tissue and an increased muscular mass, which would enhance whole-body glucose disposal as well as changes in blood flow and muscle glycogen metabolism (Tegelman *et al.*, 1996). Thus while the athletes are more insulin sensitive, the controls (with a mean BMI of $27 \pm 6.08$) were more insulin resistant. The relationship between insulin resistance and overweight is well known (Eriksson *et al.*, 1997). According to Slabber *et al.* (1994), weight loss, especially after following a low insulin response, low energy diet, decreases insulin levels in subjects. Therefore, relative insulin resistance might explain the higher fasting glucose values that were found in the controls together with the higher fasting insulin values.

While discussing insulin sensitivity, the background diet of the subjects (Table 4.4) must also be considered. The athletes consumed a larger percentage of their total energy from CHO than the controls did (56.2 % compared to 48.2 %), and apart from this they also consumed 12.2 g more dietary fibre per day than the controls did. It is known that increased amounts of CHO, especially LGI CHO that contains substantial amounts of

dietary fibre, may increase insulin sensitivity while preventing unwanted increases in fasting TG concentrations and lowering of HDL cholesterol (Kiens & Richter, 1996; Rodgers & Vranic, 1998; Smith, 1994). In healthy humans there is, however, evidence that sucrose might have a deleterious effect on insulin sensitivity (Reiser *et al.*, 1979) and the athletes consumed more sucrose than the controls did (19 % of TE compared to 16.9 %). This deleterious effect of insulin was, however, found with a CHO intake of 43 – 44 % of TE whilst the CHO intake of the subjects in this study was substantially higher than 44 % (56.2 % for the athletes and 48.2 % for the controls respectively). Therefore the higher insulin sensitivity seen in the athletes might also be a result of their (healthier) background diet.

Apart from the known relationship between endurance exercise and increased insulin sensitivity, researchers have also found that a single bout of acute exercise enhances insulin sensitivity. This increased sensitivity after a single bout of acute exercise is, however, transient (Burstein *et al.*, 1990; Perseghin *et al.*, 1996).

**After a pre-exercise meal**

The glucose values of the athletes and controls 50 min postprandially, during the HGI intervention, were higher than the fasting value. This was to be expected because the meal provided exogenous CHO and because of its HGI most of the glucose would have been absorbed in the blood stream 50 min after the meal had been ingested. The subjects were therefore in the absorptive state (Fig. 5.2).

This increase in serum glucose explains the significantly increased insulin values for both the athletes and controls that were seen 50 min postprandially.

A possible limitation of this study is that the serum glucose was not measured every 15 min after the ingestion of the meals. It could therefore not be determined if the glucose responses differed significantly for each group between the two meals (LGI and HGI). The first values measured were 50 min after the ingestion of the meals. For both the HGI

and LGI meals the 50 min postprandial serum glucose values of the controls did not differ significantly from the fasting values, while the insulin values for both meals were significantly higher that the fasting value. There was therefore no difference observed 50 min postprandially in the glucose response of the controls to the two meals.

The serum glucose values of the athletes 50 min postprandially did not differ significantly from the fasting value for either the HGI or LGI interventions. In the LGI intervention the serum glucose was, however, **lower** (though not significantly) than the fasting value. One or both of the following reasons can possibly explain this:

- After the LGI meal, not as much CHO has been digested and absorbed as in the HGI meal, as a result of the difference in GI of the two respective meals. According to Thorne *et al.* (1983) it takes more than 3 hours for the complete digestion and absorption of LGI CHO.

- The unexpected decrease may also be attributed to a pronounced insulin sensitivity of endurance trained athletes (Donahue *et al.*, 1988).

**During exercise**

The aim of CHO supplementation prior to endurance exercise is to optimise the supply of muscle glycogen and blood glucose late in exercise and to prevent exhaustion. Some researchers have found, however, that the ingestion of a HGI pre-exercise meal may lead to hyperinsulinaemia and hypoglycaemia in the first 10 – 30 min of exercise, increased muscle glycogen utilisation, decreased lipolysis and FFA availability and reduced endurance performance (Costill *et al.*, 1977; Foster *et al.*, 1979; Short *et al.*, 1997).

No hypoglycaemia (glucose < 3.9 mmol/l, Mahan & Escott-Stump, 1996) was found in either the athletes or the controls, at any of the three interventions after the acute exercise session (exercise time: 15 – 20 min) in this study. Neither did the subjects report any of the symptoms of hypoglycaemia such as light-headedness, weakness, lack of co-

ordination or nausea (Dunford & Saunders, 1993) at any stage during the three interventions. These results agree with results found by Alberici *et al.* (1993) and Horowitz and Coyle (1993). The exercise sessions in both these studies were, however, longer than in the present study. In the Alberici *et al.* (1993) study the subjects exercised for 90 min and in the Horowitz and Coyle (1993) study, the subjects exercised for 60 min.

It should, however, be noted that although hypoglycaemia did not develop, much larger fluctuations in glucose values were found in the control group during the HGI intervention, when compared to the fluctuations in the LGI intervention (Fig. 4.24).

During the baseline intervention when the athletes exercised fasted, the glucose levels increased significantly during the exercise session (Fig. 4.23). This significant increase during exercise was not seen in the control group whose glucose values showed hardly any change (Fig. 4.24). These results agree with some studies found in the literature. Febbraio and Stewart (1996), Kirwan *et al.* (1998) and Sparks *et al.* (1998) also found that when endurance trained athletes exercised fasted, their serum glucose values increased during the first 15 min of exercise. Alberici *et al.* (1993), Goodpaster *et al.* (1996), Horowitz and Coyle (1993) and Short *et al.* (1997), however, found that when endurance trained athletes exercised fasted, their serum glucose values decreased during the first 15 min of exercise, while De Marco *et al.* (1999) and Thomas *et al.* (1991) reported no changes in serum glucose values.

A possible explanation for this increase in serum glucose during the exercise session when the subjects exercised fasted, is that because there was no exogenous CHO from a pre-exercise meal, the athletes had to maintain glucose levels by breaking down liver glycogen. Thus increased liver glycogenolysis during the exercise session could be the cause of the increased serum glucose values. The subjects exercised in the post-absorptive state (Fig. 5.2).

The reason why the serum glucose values of the athletes but not that of the controls, increased during the exercise session, could possibly be due to the fact that endurance trained athletes have larger glycogen stores than untrained individuals and their enzyme and hormone activity are more conditioned because of their training (McArdle *et al.*, 1996; Williams, 1993). Therefore the athletes had enough liver glycogen that could be broken down more easily to increase the serum glucose values, while the controls had only enough liver glycogen to maintain glucose values during exercise and their bodies were probably not as conditioned as the athletes' to provide glucose fast enough.

The serum insulin levels of both the athletes and controls increased during the exercise session when the subjects exercised fasted. The increase in the serum insulin levels of the athletes was significant (Figures 4.25 and 4.26). Because of the increase seen in the glucose values during exercise, insulin secretion was probably also increased to stabilise the serum glucose levels. During exercise sympathoadrenal stimulation occurs and catecholamine levels increase substantially (a "stress response") (Ranallo & Rhodes (1998). Catecholamines are insulin antagonists and therefore actually decrease insulin levels in the blood (this decrease was in fact seen when a pre-exercise meal was given, as will be discussed in the next few paragraphs). The increase seen in the serum insulin levels of the athletes in the fasted state, might be because of the fact that in highly trained individuals the plasma catecholamine levels decrease markedly (Ranallo & Rhodes, 1998). A study done by Winder *et al.* (1979) also demonstrated that in the trained state, the serum insulin level rises during exercise. This is likely to exacerbate the blunted sympathoadrenal response to exercise in the trained state. The higher plasma insulin level in trained individuals increases the utilisation of glucose from liver glycogen. This allows more substrate to be made available to the working muscle, which in turn could increase endurance times (Ranallo & Rhodes, 1998). This does, however, not explain the increase seen in the serum insulin levels of the controls during exercise in the fasted state.

When a pre-exercise meal was ingested (LGI or HGI) the serum glucose values decreased during exercise (Figures 4.23 and 4.24), except in the athletes who had ingested a LGI

pre-exercise meal, but their pre-exercise serum glucose was lower than the fasting value and possibly because of this low glucose level at the onset of exercise, liver glycogen was also broken down to provide serum glucose to the exercising muscle.

This decrease in serum glucose values after the ingestion of a pre-exercise meal agrees with what is reported in the literature (Alberici *et al.*, 1993; De Marco *et al.*, 1999; Goodpaster *et al.*, 1996; Horowitz & Coyle, 1993; Kirwan *et al.*, 1998; Short *et al.*, 1997; Sparks *et al.*, 1998; Thomas *et al.*, 1991; Thomas *et al.*, 1994; Wee *et al.*, 1999). A possible explanation for this is that the pre-exercise meal provides exogenous CHO. This lead to increased serum glucose values at the onset of exercise and because the serum glucose levels were already increased and there was exogenous CHO available to maintain serum glucose levels, the liver did not need to produce extra glucose from glycogenolysis. This increased serum glucose was then available to the exercising muscles that caused the serum glucose value to decrease as it is used up by the exercising muscle. In this case, the subjects exercised in the absorptive state (Fig. 5.1).

The insulin values of both the athletes and controls decreased during exercise when a pre-exercise meal was ingested (Figures 4.25 and 4.26). The decrease was only significant for the athletes when they ingested a HGI pre-exercise meal. These results are consistent with the literature that states that insulin levels decrease during the first 30 to 40 min of exercise (Ranallo & Rhodes, 1998).

It is important to remember that insulin is not required for glucose uptake in the muscle cell during exercise (Brouns *et al.*, 1989). Activity of an insulin-like factor in the muscle cell membrane that has the same effect as insulin, is increased during exercise. This facilitates glucose uptake during exercise. However, it is known that insulin in the presence of muscle contractions has an additive effect on glucose uptake (Brouns *et al.*, 1989).

**Post-exercise**

During the 30 min rest period after the exercise session both the athletes' and controls' serum glucose values during baseline intervention (fasted state) decreased. The decrease observed in the athletes' serum glucose values was significant ($p < 0.05$).

This decrease could possibly be explained by the fact that glycogen stores would have been decreased during exercise to supply substrate to the exercising muscle and therefore needed to be replenished after exercise. No exogenous CHO was available for substrate utilisation. Therefore the muscles had to utilise endogenous CHO (glycogen stores). Serum glucose is taken up from the blood into the muscle to act as substrate in the glycogenesis process (Vander *et al.*, 1990). Glucose is also needed for many other body functions such normal brain function, and this further decreases the serum glucose level. In the fasted state, serum insulin levels in both the athletes and controls decreased during the 30 min rest period after the exercise session (Figures 4.25 and 4.26). The decrease in the serum insulin levels of the athletes was significant. The insulin values probably decreased because of the decrease seen in the glucose values during the resting period. The stimulus for insulin secretion (increased glucose values) was lowered.

The serum glucose levels of both the athletes and controls increased during the 30 min rest period when a pre-exercise meal (HGI and LGI) was given. None of these increases were, however, significant. This increase could possibly be because of further digestion and absorption of the pre-exercise meals. According to Englyst *et al.* (1992) CHO that is rapidly digestible such as HGI food, needs ± 20 min to be digested, while slowly digestible starch like LGI food needs ± 120 min to be digested. Therefore most of the HGI meal should have been digested at the onset of exercise while less than half of the LGI meal would probably have been digested.

Increased activity of the sympathetic nervous system during exercise which causes splanchnic vasoconstriction probably further reduced the rate of absorption of monosaccharides from the LGI meal (Wee *et al.*, 1999). At rest digestion returns to

normal and the rest of the meal can then be digested and absorbed, which explains the increase in serum glucose values during rest. This effect (according to the above-mentioned literature) should be larger for the LGI than the HGI meal, because most of the HGI meal should have been digested before the onset of exercise. None the less, the serum glucose values during rest, in both the athletes and controls after the ingestion of a HGI pre-exercise meal did also increase.

The serum insulin levels of both the athletes and controls increased slightly (but not significantly) during the 30 min rest period when a HGI pre-exercise meal was given. This was probably due to the fact that the serum glucose values increased during the rest period and thus stimulated insulin secretion. Another factor could also be that because of the termination of the exercise session the secretion of the "stress hormones" (insulin antagonists) decreased.

The serum insulin levels of the athletes and controls during the 30 min rest period decreased significantly when a LGI pre-exercise meal was ingested. This was unexpected, as digestion was probably still taking place in the post-exercise period (as has been explained in the discussion of glucose values in the 30 min resting period). It was therefore expected that insulin should increase, because of the increase in glucose (as seen with the ingestion of the HGI meal).

A possible explanation might be the production of short-chain fatty acids from the digestion of LGI food. Fibre and resistant starch are present in most LGI food. These components are not digested and absorbed in the small intestine, but pass into the colon where they are fermented to short-chain fatty acids (see Section 2.4.3). From a survey of the literature (Venter, 1989) it appears as if one of these short-chain fatty acids, propionate, has a significant effect on hepatic CHO metabolism. It stimulates glycogenolysis and glucose utilisation without increasing serum insulin levels. Because of the composition of the LGI meal, short-chain fatty acids would probably have been

formed and the presence of propionate in the liver might therefore explain the lower insulin values observed.

**Athletes *vs* controls**

When the glucose responses for all three interventions are compared between the athletes and the controls (Figures 4.23 and 4.24) it can be seen that the controls showed overall larger fluctuations than the athletes did. This is possibly due to the fact that endurance training "conditions" enzyme and hormone activity (McArdle *et al.*, 1996).

Generally, the magnitude of hormonal response to a standard exercise load declines with endurance training. Target tissue sensitivity and/or responsiveness to a given quantity of hormone increases (McArdle *et al.*, 1996). Training can also increase the number of mitochondria and levels of enzymes involved in aerobic synthesis of ATP (Mahan & Escott-Stump, 1996). These metabolic adaptations enhance the ability of muscle to oxidise all fuels, but especially fat (Mahan & Escott-Stump, 1996).

Another limitation of the study is that lactate levels were not measured because of financial constraints. Lactate production is an indication of anaerobic glycolysis (Sparks *et al.*, 1998). Therefore the level of glycogen use and depletion could not be determined in this study and, consequently the effect of different GI's on muscle glycogen depletion in this study can only be speculated on. Furthermore, FFA levels were also not determined, again because of financial constraints. Measurement of FFA would also have allowed a more detailed understanding of what substrates (muscle glycogen, muscle triglyceride, serum glucose and serum FFA) were used during exercise and to what extent (as has been discussed in Section 2.6).

## 5.4 CORRELATIONS

The correlations between glucose and insulin and other variables were given in Table 4.8 and 4.9 respectively. For all the time intervals and in both groups (athletes and controls)

the strongest correlations were found between glucose, insulin and triglycerides. This was to be expected if one takes into account that insulin secretion is directly controlled by the level of glucose in the blood that passes through the pancreas (McArdle *et al.*, 1996). Furthermore, when considering the effect of insulin on lipid metabolism such as increased fatty acid synthesis, LDL receptor activity, and lipoprotein lipase (LPL) activity as well as decreased lipolysis (as indicated in Table 2.3), the correlation with triglycerides was to be expected.

When comparing the three interventions, the most correlations between glucose, insulin and triglycerides were found for the baseline intervention and when a LGI pre-exercise meal was given. Very few correlations were found between glucose and insulin and triglycerides when a HGI pre-exercise meal was given. It appears as though the HGI meal influences the normal correlations seen in the fasted state (as discussed above). The fact that strong correlations were also found with the ingestion of a LGI meal, could indicate that the lower blood glucose response after the ingestion of a LGI meal caused a relatively stable insulin response during acute exercise (of the same magnitude as in the fasted state). When a HGI pre-exercise meal was ingested, however, the sharp increase in blood glucose caused an over stimulus of insulin, more than was actually needed to regulate the serum glucose levels. Therefore fewer correlations were found with the ingestion of a HGI pre-exercise meal. It is this increased insulin response that could theoretically cause hyperinsulinaemia with resultant hypoglycaemia in the first 10 – 30 min of exercise after the ingestion of a HGI pre-exercise meal (as has been discussed in Section 2.6).

# CHAPTER 6 CONCLUSIONS AND RECOMMENDATIONS

## 6.1 INTRODUCTION

The aim of this study was to determine the effect of the GI of a pre-exercise meal on the serum glucose and insulin responses of elite male athletes and controls during an acute exercise session of 15 – 20 min. The study was divided into three interventions:

1. Intervention I – Baseline: subjects exercised fasted.
2. Intervention II – HGI intervention: subjects exercised after ingestion of a HGI pre-exercise meal.
3. Intervention III – LGI intervention: subjects exercised after ingestion of a LGI pre-exercise meal.

The glycaemic index itself is a relative new concept (Jenkins *et al.*, 1981) and role of the GI in sports and exercise has therefore not been studied extensively. This study differs from other studies cited in the literature in (a) that a control group was included; and (b) that the exercise time of this study was significantly shorter compared to previous studies (Alberici *et al.*, 1993; Brouns *et al.*, 1989; Burke *et al.*, 1993; Burke *et al.*, 1995; Burke *et al.*, 1996; De Marco *et al.*, 1999; Febbraio & Stewart, 1996; Kirwan *et al.*, 1998; Parkin *et al.*, 1997; Short *et al.*, 1997; Sparks *et al.*, 1998; Thomas *et al.*, 1991; Wee *et al.*, 1999; Weydahl *et al.*, 1995; Zawadzki *et al.*, 1992;).

Although the shorter exercise time and inclusion of a control group makes this study unique, the selection of the control group could probably have been done more cautiously. As has been discussed in Section 4.2.2 the control group had a significantly (statistically as well as clinically) higher BMI than the athletes.

In Section 6.2 conclusions will be made from the results of this study and in Section 6.3 recommendations as to whether a pre-exercise meal should be given for an exercise session of 15 – 20 min or not and if so, what type of CHO (LGI of HGI) it should consist of. Section 6.4 will provide recommendations for further study.

## 6.2 CONCLUSIONS

Figures 4.23 to 4.26 summarise the serum glucose and insulin responses for all three interventions. The serum glucose and insulin values of the athletes basically followed the same trend as the values of the controls. The important differences are the following:

- Larger fluctuations were seen in both the glucose and insulin values of the controls compared to the values of the athletes.

- The athletes had significantly lower fasting glucose and insulin levels than the controls.

- The ingestion of a HGI pre-exercise meal caused larger fluctuations in the glucose and insulin values of both the athletes and controls than did the LGI pre-exercise meal.

- Exercising in the fasted state for 15 – 20 min resulted in significantly higher serum glucose values at the end of exercise when compared to the ingestion of a pre-exercise meal.

## 6.3 RECOMMENDATIONS

Recommendations based on serum glucose and insulin values that could possibly be made from the results of this study are given in Table 6.1.

Table 6.1 Recommendations according to results of this study.

| Intervention | Exercise | Recovery |
|---|---|---|
| Fasting | ✓ | ✗ |
| HGI pre-exercise meal | ✗ | ✓ |
| LGI pre-exercise meal | ✓ | ✓ |

Exercising in the fasted state resulted in the highest glucose values at the end of exercise as well as the longest time to exhaustion and the highest PWC. During the 30 min recovery period, however, there was a sharp decrease in serum glucose values. If this drop continues, it might cause hypoglycaemia, which could be detrimental to the athletes (serum glucose values later than 30 min of rest were not determined in this study).

Exercising after the ingestion of a HGI pre-exercise meal resulted in large fluctuations in the serum glucose values of both the athletes and controls. Although hypoglycaemia did not occur in this study, the large fluctuations are a cause for concern. During the 30 min rest period, serum glucose values, however, increased again to the fasting level.

Exercising after the ingestion of a LGI pre-exercise meal resulted in stable serum glucose values both during exercise and during the 30 min resting period. Although time to exhaustion, PWC and resistance were lowered compared to when exercising in the fasted state, this was not significantly so. Therefore, it does not appear as though ingesting a LGI pre-exercise meal before an exercise session of 30 min, inhibits performance significantly. A LGI pre-exercise meal, by maintaining serum glucose concentrations, may help to conserve hepatic glycogen stores.

## 6.4 RECOMMENDATIONS FOR FURTHER STUDY

Further studies are needed to clarify the position of the importance of the GI of the pre-exercise meal.

The inclusion of a control group in this type of study enables researchers at the same time to also determine the effect of long-term endurance training. Therefore the inclusion of a control group has its merits. Care should, however, be taken that the BMI and workload of both groups are compatible. Subjects should therefore not exercise until exhaustion but until the same workload has been reached.

Glycogen use/depletion should be determined by measuring for example lactate levels as well as RER. Measuring glycogen use will enable the researcher to determine which CHO sources (endogenous and exogenous) provided glucose to the blood and how these sources of blood glucose are influenced by exercise in the absorptive or post-absorptive state.

The $VO_2$ max was not stable during the exercise session, because it was a graded exercise test. $VO_2$.max should, however, be determined because substrate utilisation during exercise is not just influenced by the pre-exercise meal but also by the intensity and duration of the exercise as well as the fitness level of the subjects (Section 2.6). Controlling these factors would make a more accurate discussion of substrate utilisation during exercise possible.

The importance of this study lies in the observation that even for a short acute exercise session (15 – 20 min), the ingestion of a LGI pre-exercise meal results in the most optimal serum glucose and insulin levels in athletes as well as non-athletes. Because of the larger fluctuations caused by the ingestion of a HGI pre-exercise meal, foods with a HGI are therefore not recommended for short bouts of exercise. Manufacturers of supplements for athletes should also take note of these findings.

# CONGRESS PRESENTATIONS

The following presentations, based on this dissertation, have been delivered:

Pieters, M., Venter, C.S., Badenhorst, C.J., Snyman C. & Moss, H. The effect of the glycaemic index of a pre-exercise meal on the glycaemic and insulin responses during acute exercise. 8th South African Sports Medicine Association biennial conference. Sports medicine in Africa: Into the next millennium. 6 – 8 September 1999, Midrand.

Pieters, M., Venter, C.S., Badenhorst, C.J., Snyman C. & Moss, H. The effect of the glycaemic index of a pre-exercise meal on the glycaemic and insulin responses during acute exercise. Wageningen Symposium, 20 – 21 August 1999, Pretoria.

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# ADDENDUM A

Informed consent form

## INFORMED CONSENT

The school of Nutrition, Family Ecology and Physiology

1. **Project title**

   Characteristics of the fibrin network structure of sedentary and active males

   **Project leaders**

   Sr. MC Lessing, registered nursing sister

   Prof. HH. Vorster: HOD Research

   Prof. WJH Vermaak: HOD Chemical Pathology

   Prof. CS Venter: HOD Nutrition

2. **The procedures that would be followed**

   The subjects will have to fast for 10h to 12 h. Fasting venous blood samples for the baseline values will be drawn at three different periods. These periods will be before, during maximal activity and 30 minutes after activity was stopped. These procedures would be repeated with the consumption of a high glyceamic index meal before the maximal activity. The test will be repeated with the intervention of a low glyceamic index meal. During the pre-exercise meal intervention testing, four blood samples will be drawn. With these blood samples a total DAX profile will be done together with the determination of fibrinogen, Factor VII, insulin, and fibrin network structure characteristics. Mass, length, percentage body fat, blood pressure and heart rate will be measured.

3. **Inconvenience and danger that is involved in the project**

   Blood has to be drawn as described in paragraph 2. All the necessary precautions will be taken to ensure that the process is performed with the least amount of inconvenience.

3. **Pre-cautions taken to protect the subjects**

   A qualified en experienced nurse will draw the samples according to a set protocol to prevent the contamination of any infectious diseases.

To the undersigned of the informed, consent:

1. You are invited to participate in the research project as described in paragraph 2 in the previous section. It is important that you will read and understand the following principles of the research project that is applicable to all participants.

1.1 Participation in the project is completely voluntarily

1.2 It is possible that you will not gain any personal advantage from the project, although the knowledge that is gained from the project may be to the advantage of other people.

1.3 You are free to with draw from the project without any reason. You are kindly requested not to do so without giving it good thought, as the statistical validity of the project will be influenced.

1.4 A summary of the type of project, risk factors and possible inconvenience that may occur together with the advantages after given in part 1

1.5 You are welcome to ask any questions at any stage concerning the project and the procedures to the project leaders. The project will also be fully discussed with you.

**CONSENT**

I, the undersigned_____have read the preceding information regarding the project as described in part 1 paragraph 2 together with a oral description and declare that I understand it. I was given the opportunity to discuss in short the aspects of the project with the leader. I here with declare that I voluntarily participate in the research project.

_____Signed at_____

(Signature of volunteer) on _____

_____Signed at_____

(Signature of project leader) on _____

_____Signed at_____

(Signature of witness) on _____

# ADDENDUM B

Anthropometric data sheet

INSTITUUT VIR BIOKINETIKA

Menslike Bewegingskunde
PU vir CHO


INSTITUTE FOR BIOKINETICS

Human Movement Science
PU for CHE

SIVIELE PERSONE

PERSOONLIKE INLIGTING/PERSONAL INFORMATION

DATUM VAN EVALUASIE/DATE OF EVALUATION : ___/___/19___

VOORLETTERS EN VAN/INITIALS AND SURNAME : _____

_____

POSADRES/POSTAL ADRESS : _____

_____

_____

_____

WOONADRES/HOME ADRESS : _____

_____

_____

_____

TEL NR WERK/WORK : _____

HUIS/HOME : _____

GENEESHEER/DOCTOR : _____

OUDERDOM/AGE : _____ GESLAG/SEX : _____

BEROEP/OCCUPATION : _____

HUWELIKSTATUS/MARITAL SATUS : _____

REDE VIR BESOEK/REASON FOR VISIT : _____

_____

_____

# INGELIGTE TOESTEMMING

EK, DIE ONDERGETEKENDE _____

VERKLAAR HIERBY DAT EK DIE GEDETAILLEERDE INLIGTING DEURGELEES HET EN DIT TEN VOLLE VERSTAAN. EK HET DIE GELEENTHEID GEHAD OM RELEVANTE VRAE M.B.T. DIE EVALUASIE TE VRA EN NEEM DIE EVALUASIE OP EIE RISIKO.

_____ _____

SIGNED / HANDTEKENING DATE / DATUM

# INFORMED CONSENT

I, THE UNDERSIGNED _____

READ THE DETAILED INFORMATION ABOUT THE EVALUATION AND UNDERSTAND IT FULLY. I HAD THE OPPORTUNITY TO DISCUSS ALL RELEVANT MATTERS CONCERNING THE TEST WITH THE BIOKINETICIAN I PARTICIPATE IN THE EVALUATION AT MY OWN RISK.

1.1 Kan jy sonder om pyn in die bors te kry of baie kortasem te word 2-3 km stap?
Are you capable of walking 2-3 kilos without experiencing chest pains and/or becoming short of breath ?
JA/YES NEE/NO ONSEKER/UNCERTAIN

1.2 Indien NEE, wat is die probleem ? /
If your answer is NO, state problem

---

1.3 Het u geneesheer al ooit gesê dat u 'n hart - probleem het ? / Has your doctor indicated that you have a heart ailment ?
JA/YES ☐ NEE/NO ☐

1.4 Indien JA, wat is die probleem ? /
If so, state problem

---

1.5 Het u geneesheer al ooit gesê dat u bloeddruk te hoog is ? / Do you suffer from high blood pressure ?
JA/YES ☐ NEE/NO ☐

1.6 Neem u medisyne op gereelde basis ? /
Are you on regular medication ?
JA/YES ☐ NEE/NO ☐

1.7 Indien JA, watter soort en vir wat ? /
If so, what type of medicine and for what reason is it being used ?

---

1.8 Het u enige las van pyn laag in die rug ? /
Are you troubled by pain in the lower region of the back ?
JA/YES ☐ NEE/NO ☐

1.9 Indien JA, spesifiseer asb / If so please spesify

---

1.10 Het u las van enige breuke ? /
Are you troubled by any kind of hernia ?
JA/YES ☐ NEE/NO ☐

1.11 Indien JA, spesifiseer asb / If so please spesify

---

1.12 Het u enige van die volgende siektetoestande op die oomblik ? / Do you presently suffer from any of the following ailments ?

| | JA/YES | NEE/NO |
|---|---|---|
| a) Oefeningsgeïnduseerde asma / Exercise induced asthma | ☐ | ☐ |
| b) Gereelde hoofpyn of megraine aanvalle / Regular headaches or migraine attacks | ☐ | ☐ |
| c) Gereelde moegheid / Regular tiredness | ☐ | ☐ |
| d) Depressie / Depression | ☐ | ☐ |
| e) Nierprobleme / Kidney problems | ☐ | ☐ |
| f) Krampe in bene / Cramps in the legs | ☐ | ☐ |
| g) Diabetes mellitus / Diabetes mellitus | ☐ | ☐ |
| h) Verkoue, griep of bronchitus / Flu or bronchitus | ☐ | ☐ |
| i) Het u enige gewrigs- of skeletprobleme soos artritis wat deur oefening vererger kan word ? / Do you have any joint or skeletal problems, eg. arthritis, which are aggrovated by exercise ? | ☐ | ☐ |
| j) Epilepsie / Epilepsy | ☐ | ☐ |
| k) Spatare in bene / Varicose veins in legs | ☐ | ☐ |
| l) Enige longsiektes / Any pulmonary disease | ☐ | ☐ |

Indien JA, spesifiseer / If so, specify

---

| | | |
|---|---|---|
| m) Lighoofdigheid of floutes / Dizziness or fainting | ☐ | ☐ |

JA/ NEE/
YES NO

n) Is enige probleme met bloedsirkulasie al ooit by u geneesheer gediagnoseer ? / Has your doctor ever diagnosed a blood circulatory disorder ? ☐ ☐

1.13 SPANNING / TENSION

Dui asseblief aan watter een van die volgende die beste op u van toepassing is / Please mark the one condition most applicable to you

| | |
|---|---|
| Nooit ooit gespanne nie<br>Never ever tense | |
| Selde gespanne of angstig<br>Seldom tense | |
| Soms gespanne, tydsbewus en gejaagd<br>From time to time tense | |
| Dikwels<br>Frequently tense and/or anxious | |
| Gewoonlik gejaagd/dikwels kwaad<br>Usually tense and/or anxious | |
| Uitermate gejaagd/dikwels kwaad/ baie gedruk/neem kalmeermiddels<br>Exceedingly tense and anxious | |

1.14 Het u geneesheer enige ander mediese probleme gediagnoseer of enige probleem wat u belangrik ag waarvan ons moet kennis neem ? / Has your doctor diagnosed any other medical problem or ailment which you seem necessary to our notice ?

JA/YES ☐ NEE/NO ☐

1.15 Indien JA ,wat is die probleem ? / If so, specify

_____

1.16 ROOKGEWOONTES / SMOKING HABITS

Is u 'n roker ? / Are you a smoker ?

JA/YES ☐ NEE/NO ☐

Indien wel, hoeveel sigarette per dag ? / Is, how many cigarettes per day ? ☐

Indien nie, was u voorheen 'n roker en hoe lank gelede het u opgehou rook ? / If not, did you ever smoke and when did you stop ?

_____

1.17 Neem u deel aan sport ? / Do you participate in any kind of sport ?

JA/YES ☐ NEE/NO ☐

Indien JA, spesifiseer / If so, specify

_____

1.18 Ly u of enige van u bloedverwante aan die volgende. Dui asseblief aan waar van toepassing ? Do you or any of your relatives suffer from any of the following. Please indicate where applicable

| | Vader Father | Moeder Mother | Suster Sister | Broer Brother |
|---|---|---|---|---|
| Verhoogde cholesterol/ Excessive cholesterol | | | | |
| Verhoogde trigliseriede / Increase triglicerides | | | | |
| Koronere hartsiekte onder 60 / Coronary heart disease under 60 | | | | |
| Suikersiekte / Diabetes mellitus | | | | |
| Hoe bloeddruk / Hypertension | | | | |
| Oorgewig / Overweight | | | | |
| Beroerte / Stroke | | | | |

## ERGOLOGIESE EVALUERING

* LEDEMAAT : _____

* TIPE BESERING : _____

_____

_____

* GESKIEDENIS : _____

_____

_____

_____

_____

_____

_____

_____

_____

* ERGOLOGIES : _____

_____

_____

_____

_____

_____

_____

_____

_____

VOORLETTERS EN VAN : _____

_____

DATUM : ___/___/19___ OUDERDOM : _____ JAAR

GESLAG : _____

LENGTE : _____ CM MASSA : _____ KG

| VELVOUMETINGS | | | | |
|---|---|---|---|---|
| TRISEPS | | | | |
| SUBSKAPULA | | | | |
| SUPRA-ILIACA | | | | |
| PARA-UMBILIKUS | | | | |
| BOBEEN | | | | |
| MEDIALE KUIT | | | | |

VET % = _____ %

ILM = _____ KG

(VET % = __)

OPSITTE : _____ SOEPELHEID : _____ CM

HT PERSENTASIE VAN MAKSIMUM

40% = _____ 70% = _____

50% = _____ 80% = _____

60% = _____ 90% = _____

$FWV_{170}$/FOX 5 MIN / /

| STRESVLAK | WEERSTAND | HT | SBD | DBD | BORGSKAAL |
|---|---|---|---|---|---|
| RUSTEND | | | | | |
| 1. | | | | | |
| 2. | | | | | |
| 3. | | | | | |
| 4. | | | | | |
| 1 MIN HERSTEL | | | | | |
| 3 MIN HERSTEL | | | | | |

# ADDENDUM C

Dietary intake questionnaire

(24-hour recall)

## DIETARY INTAKE QUESTIONNAIRE (24HR-RECALL)

Name of child: _____

Date of birth: Day _____ Month _____ Year _____

Day of the week interviewed:

MON ☐1 TUES ☐2 WED ☐3 THUR ☐4 FRI ☐5 SAT ☐6 SUN ☐7

Was yesterday typical/routine for the child? Yes ☐1 No ☐2

If not, why? _____

What kind of margarine does the child usually eat:

- on bread: B-6502 ☐ HM-6508 ☐ MED-6560 ☐ PM-6521 ☐ Ghee-6554 ☐

-in cooking: B-6502 ☐ HM-6508 ☐ MED-6560 ☐ PM-6521 ☐ WF-6545 ☐

Ghee-6554 ☐

What kind of bread does the child usually eat?

White ☐4001 Brown ☐4002 Whole wheat ☐4003

What kind of milk does the child usually drink?

SM-0007 ☐ WM-0006 ☐ BL-0068 ☐ Breast-0029 ☐

COND-WM-0002 ☐ COND-SM-0032 ☐ Goat-0026 ☐ 2%-0069 ☐ Formula: _____

Other (specify) _____

Did this child eat at a feeding scheme or créche yesterday? Yes ☐1 No ☐2

(If yes, fill in page 12)

Name of Interviewer: _____

ow I want you to tell me everything that this child ate and drank yesterday. Lets start with when the child woke up. id he/she have anything to eat or drink? Proceed through the day following the child'sactivities. When you have ished, summarise it for the caretaker. Any forgotten items can then be added.

Instructions: Enter each item eaten in grams under the correct interval of the day eaten. Make sure that the code is entered. Items not on the questionnaire should be looked up in the FOOD COMPOSITION TABLES. Specify fully when new items are entered and look up the code later. Recipes should be added on page 13.

This questionnaire was updated from one developed by N P Steyn (Steyn NP, Dietary intake and nutritional status of 11-year-old children in the Western Cape. Stellenbosch University: Ph D thesis; 1988).

---

**ABBREVIATIONS**

Measures
1t = 1 rounded teaspoon
1T = 1 rounded tablespoon (15ml)
1LS = 1 rounded servingspoon (30ml)
c = measuring cup (250ml)
s/s = small size
m/s medium
l/s = large
E = enriched
P = plain

Milk:
SM = skim milk
WM = whole milk
BL = blend
CON = condensed

Bread:
Wh = white
Br = brown
Ww = wholewheat

Meat:
F = with fat
FT = fat trimmed

Oil/Fat
B = butter
HM = hard margarine
Med = medium fat/light
PM =- polyunsaturated
VO = vegetable oil
WF = white fat

BR = Breakfast (Up to 09h00)
IS = In-between snack
L = Lunch (midday (12h00-14h00)
D = Dinner (evening) ( 17h00 - 19h00)
AD = After dinner
Comm = commercial
Home = homemade
Pot = potato
Cab = cabbage
Carr = Carrot
Fill = filling
Usually = At least 4x/week

| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|
| TEA & COFFEE | Tea: 9514; Rooibos 9560; Coffee 9513 | | Teacup = 180ml; Mug = 250ml | | | | | | |
| | + Sugar | 9012 | 1 t = 6g | | | | | | |
| | + Condensed Milk: SM -0032; WM-0002 | | 1t = 10g | | | | | | |
| | + Non-diary Creamer | 0039 | 1t = 3g | | | | | | |
| | + Milk Blend | 0068 | 20ml - tea in cup | | | | | | |
| | + SM-0007; WM-0006; 2%-0069; Breast-0029; Goat-0026 | | 35ml - tea in mug | | | | | | |
| | + Formula Milk (Specify): | | 40ml - coffee in cup | | | | | | |
| | | | 75ml - coffee in mug | | | | | | |
| MILK & MILK DRINKS | ~~Buttermilk~~ | ~~0001~~ | s/s = 175ml | | | | | | |
| | Maas/Amazi/Sourmilk | 0085 | L/s = 500ml<br>½c = 125g | | | | | | |
| | Custard: SM-0005; WM-0004 | | | | | | | | |
| | Milk: SM-0007; WM-0006; BI-0068; 2%-0069; Breast-0029; Goat-0026; FORMULA (Specify) : _____<br>No. of scoops/bottle used _____ | | To drink ½c = 125ml<br>Baby bottle = 250ml | | | | | | |
| | + Sugar | 9012 | 1t = 6g | | | | | | |
| | + Ice Cream-6548; Sorbet-6516 | | 1 Scoop = 40g | | | | | | |
| | + Sustagen-9722; Complan-9725 | | 2 scoops = 25g; 1T = 15g | | | | | | |
| | + Milo/Cocoa/Horlicks/Ovaltine | 0024 | 1t = 5g | | | | | | |
| | Yoghurt: Plain SM-0022; WM-0045 | | s/s = 175ml<br>Yogisip = 350ml<br>½c = 125g | | | | | | |
| | Flav-0044; Fruit-0020 | | | | | | | | |
| COLD DRINKS/JUICE | ~~Apple Juice - No Sugar~~ | 7080 | Liquifruit s/s = 250ml<br>L/s = 500ml<br>Ceres s/s = 200ml<br>Cartons/bottles<br>s/s = 350ml<br>L/s = 500ml | | | | | | |
| | Apricot: + Sugar-7008; No Sugar-7040 | | | | | | | | |
| | Mango-7162; Granadilla-7159; Grape-7169 | | | | | | | | |
| | Orange: +Sugar-7033; No sugar-7113 | | | | | | | | |
| | Guava: +Sugar-7024; No Sugar-7103 | | | | | | | | |
| | Peach-7117; Pear-7120; Naartjie-7161 | | | | | | | | |
| | Cold drinks: Squash-9002; Magou-9562; Carbonated-9001 | | s/s bottle = 300ml<br>L/s bottle = 500ml<br>s/s can = 340ml | | | | | | |
| | Low-Cal-9013; Syrup (undiluted)-0534 | | | | | | | | |
| BREAKFAST CEREALS | ~~Mabella: Soft or stiff~~ | ~~4034~~ | ~~½c = 125ml~~ | | | | | | |
| | M/Meal: Soft: Plain-4254; Enrich-4330 | | 1c soft = 250g | | | | | | |
| | Stiff: Plain-4255; Enrich-4331 | | 1c stiff = 250g | | | | | | |
| | Crumbly: Plain4256; Enrich-4332 | | 1c crumbly = 140g | | | | | | |
| | Oats-4032; Tastee Wheat-4033; Sour Porridge | | ½c = 125g | | | | | | |
| | Corn Flakes-4036; Sugar Frosted-4218 | | 1c = 40g | | | | | | |
| | Honey Crunch and Muesli | 4122 | ½c = 65g | | | | | | |
| | Pronutro: Great Start-4316; High Energy-4038; Wholewheat-4314 | | ½c = 50g | | | | | | |
| | Puffed Wheat-4149; Sweetened-4221 (Honey Smacks) | | ½c = 12g | | | | | | |
| | Raisin Bran-4217; Fruit Loops-4303 | | Raisin Bran ½c = 45g<br>Fruit Loops ½c = 18g | | | | | | |
| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| BREAKFAST CEREALS | Special K-4146; All Bran-4035 | | ½c = 25g | | | | | | |
| | Rice Crispies-4046; Cocopops-4216 | | ½c = 20g | | | | | | |
| | Weetbix | 4037 | 1 = 25g | | | | | | |
| | + Fat: B-6502; HM-6508; PM-6521; WF-6545; Med-6560 | | | | | | | | |
| | Milk: SM-0007; WM-0006; BI-0068; 2%-0069; Goat-0026<br>Breast-0029; Formula - Specify: | | with instant = 125ml<br>with porridge = 60ml<br>with pronutro = 180ml | | | | | | |
| | + Sugar | 9012 | 1t = 6g | | | | | | |
| BREAD & ROLLS | Bread: Comm & Home:<br>~~Wh-4001; Br-4002~~<br>Ww-4003 | | Wh + Br 10mm = 30g<br>Ww 10mm = 35<br>Wh + Br 20mm = 60g<br>Ww = 20mm = 70g | | | | | | |
| | Cream Crackers-4022; Provita-4027 | | 1 = 7g | | | | | | |
| | Maize Meal Bread | 4083 | m/s = 30g; L/s = 50g | | | | | | |
| | Rolls: Wh-4001; Br-4002; Ww-4003<br>Roti: VO-4199; HM-4198 | | Wh round (10cm) = 30g<br>Wh long (16cm) = 40g<br>s/s = 50g (Roti) | | | | | | |
| | Rusks: Comm Plain | 4206 | 1 = 15g | | | | | | |
| | Comm Buttermilk: Wh-4160; Ww-4161 | | Wh = 35g; Ww = 30g | | | | | | |
| | Home Buttermilk: Wh-4006; Ww-4049 | | Wh = 30g; Ww = 30g | | | | | | |
| | Scones: (Wh) SM-4269; WM-4029 | | 6cm diam = 35g<br>8cm diam = 60g | | | | | | |
| | (Ww) SM-4270; WM-4142 | | | | | | | | |
| | Vetkoek: Wh-4057; Ww-4148 | | 8cm diam = 60g | | | | | | |
| SPREADS ON BREAD | ~~Beef Fat-6519; Mutton Fat-6522; Butter-6502; Ghee-6554; Lard-6520; WF-6545~~ | | Thin: ~~5~~ Med: ~~10~~ Thick: ~~15~~ | | | | | | |
| | Fishpaste-2567 Liver Spread-1517 | | 5 7 10 | | | | | | |
| | Jam-9008; Honey-9007; Syrup-9011 | | 10 20 35 | | | | | | |
| | Marg: H-6508; Med-6560; PM-6521 | | 5 7 10 | | | | | | |
| | Marmite-9502; Meat Spread-9501 | | 2 4 7 | | | | | | |
| | Peanut Butter-6509; Sandwich Spread-6551 | | 5 10 20 | | | | | | |
| EGGS | ~~Eggs: Boiled~~ | ~~1001~~ | ~~1 egg = 50g~~ | | | | | | |
| | Curried | 1037 | 1 egg + sauce (IT) = 75g | | | | | | |
| | Fried: B-1002; HM-1012; PM-1013 | | 1 egg = 52g | | | | | | |
| | VO-1003; Bacon Fat-1004 | | | | | | | | |
| | Scrambled/Omelette: SM + B-1021; SM + HM-1022<br>SM+PM-1023; SM+VO-1024; WM+B-1009<br>WM+HM-1025; WM+PM-1026; WM+VO-1008 | | IT = 35g; 1LS = 80g<br>½c = 115g (± 2 eggs)<br>Omelette = 60g egg (med)<br>120g (L/S) | | | | | | |

| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|
| CH EE SE | Cheddar-0010; Sweetmilk/0011 | | grated: med = 10g<br>Thick = 15g<br>1 cheezi = 20g; cubes = 30g<br>1 slice = 8g | | | | | | |
| | Cheese Spread | 0018 | med = 12g; thick = 25g | | | | | | |
| | Cottage Cheese; Creamed | 0047 | thin = 10g med = 20 | | | | | | |
| | Fat Free-0017; Low Fat-0048<br>(smooth) (Chunky) | | med = 20g; thick = 30g | | | | | | |
| | Macaroni Cheese: SM-4176; WM-4120 | | 1T = 45g; 1LS = 90g;<br>½c = 115g | | | | | | |
| | Pizza (Cheese + Tomato) | 4193 | s/s = 90g; L/s = 340g | | | | | | |
| | Savoury Tart: + Asparagus-4210; + Vienna-4153 | | wedge small = 65g<br>med = 75g<br>large = 110g | | | | | | |
| | + Tuna-4209 | | | | | | | | |
| ME AT | Bacon: Fried: Lean-1510 F-1501 | | 1 rasher = 10g | | | | | | |
| | Beef: Corned/Silverside/Cold cuts:F-1519;<br>Lean-1558; Curry Beef-Combine codes | | 138 x 85 x 3 = 20g<br>½c = 100g | | | | | | |
| | Fillet: F-1528; FT-1524 | | 100 x 70 x 10 = 90g | | | | | | |
| | Mince: Pan Fried F-1505; Lean-1556; Curry-1631 | | T = 40; LS = 85g<br>½c = 100g | | | | | | |
| | - Savoury (Tomato + Onion) | 1585 | | | | | | | |
| | - Cottage Pie: WM + HM | 1623 | | | | | | | |
| | Roast: F-1539; FT-1555 | | 120 x 60 x 5 = 35g<br>120 x 60 x 10 = 70g | | | | | | |
| | Rump: Fried: F-1503; FT-1554 | | S/S 130 x 70 x 15 = 125g<br>LS 165 x 70 x 30 = 270g | | | | | | |
| | Sirlion/T-Bone: Grilled: F-1541; FT-1502 | | | | | | | | |
| | Stew: Cabbage + Onion + Potatoes | 1619 | 1 LS = 105g; ½c = 125g | | | | | | |
| | : Pot + Carrots + Peas + Onions | 1504 | | | | | | | |
| | Biltong: Beef-1506; Game-1507 | | Grated 1LS = 10g<br>Beefeater = 18g<br>Sliced 1LS = 35g | | | | | | |
| | Bobotie: Lean, SM, VO-1628; F, WM, VO-1584 | | 1LS = 85g; ½c = 115g | | | | | | |
| | Chicken: Boiled + Skin-1621; No Skin-1560; Curry-Combine | | breast + skin = 125g<br>thigh = 80g<br>drumstick = 42g<br>foot = 30g<br>wing = 30g<br>pie (comm) = 150g home = 90g<br>liver = 30g; stomach = 20g | | | | | | |
| | Feet-1609; Giblets-1610; Heads-1611 | | | | | | | | |
| | Pie (Comm) | 1549 | | | | | | | |
| | Roast + Skin-1520; No Skin-1545 | | | | | | | | |
| | Stew: Carrot, Peas, Pot-1618<br>Cabbage + Potato-1619<br>Tomato + Onion - 1583 | | 1LS = 90g; ½c = 125g | | | | | | |
| | Batter Dipped-Fried eg. Kentucky | 1634 | 1Ls = 105g; ½c = 125g | | | | | | |
| | Cornish Pie: (Comm) | 1548 | med = 150g | | | | | | |
| | Frankfurter | 1532 | 155 x 20 = 45g<br>168 x 21 = 60g | | | | | | |

| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|
| | Goat meat: Stewed (plain)-1677<br>(+ Veg)-1678 | | 120 x 60 x 5 = 35g<br>120 x 60 x 10 = 70g | | | | | | |
| | Ham-1564; Ham & Tongue loaf-1600 | | med slice = 25g | | | | | | |
| ME AT | Heart: Beef-1565; Sheep-1566<br>Kidney: Beef-1518; Sheep-1551<br>Lung: Beef-1635 | | Sheep heart = 60g<br>Sheep kidney = 30g<br>Beef kidney = 85g | | | | | | |
| | Lasagne: SM-4318; WM-4061 | | T = 40g; LS = 75g; ½c = 120g | | | | | | |
| | Liver: Fried : Beef-1515; Sheep-1550 | | Sheep = 55g<br>Chicken = 30g<br>Beef = 80g | | | | | | |
| | Cooked: Chicken-1567; Patty-1568 (fried) | | | | | | | | |
| | Meat Ball: F + Egg-1562; F-No Egg-1563; Lean-1653 | | 50mm = 60; 75mm = 120g | | | | | | |
| | Meat Loaf: F-1655; Lean-1615 | | 80 x 85 x 15mm slice = 80g | | | | | | |
| | Meat Patty: (Hamburger) | 1581 | s/s = 50g; m/s = 100g | | | | | | |
| | Mutton: Chop (grilled) F-1522; FT-1529 | | Loin chop = 60g<br>Rib chop = 40g | | | | | | |
| | Roast: F-1542; FT-1570 | | s/s slice = 30g<br>Med = 70g | | | | | | |
| | Stew: Plain-1571; Irish-1511 (Carrots + Pot)<br>Curry-1660; Greenbean-1661 | | LS = 105g; ½c = 125g | | | | | | |
| | Offal: Cooked-Tripe; Tongue, Harslag, Pens, Vetderm; (Specify): | 1616 | 1 LS = 105g; ½c = 125g | | | | | | |
| | Oxtail: Stewed | 1573 | | | | | | | |
| | Polony | 1514 | Slice 5mm thick = 8g<br>Comm slice = 16g | | | | | | |
| | Pork: Chop (Grilled) F-1525; FT-1574 | | Chop: 115 x 80 x 20 = 100g<br>Schnitzel: 115 x 80 x 20 =110g<br>Roast: 110 x 65 x 5 = 30g<br>1LS = 105g; ½c = 125g<br>3 Ribs = 130g | | | | | | |
| | Crumbed-1602; Spareribs-1625 | | | | | | | | |
| | Roast: F-1553; FT-1575 | | | | | | | | |
| | Salami and Russians | 1543 | Slice 5mm thick = 12g<br>1 Russian = 50g | | | | | | |
| | Samoosa: with Veg-4272; Meat-4196 | | s/s = 42g | | | | | | |
| | Sausage: Beef: Dry-1544; Fried-1526 (Boerewors) | | Thin x 200mm = 45g<br>Thick x 165mm = 90g | | | | | | |
| | Pork: Fried/Grilled | 1527 | Med = 55g | | | | | | |
| | Roll/Meat Pie (Comm) | 1534 | 25mm Pie = 120g<br>Roll x 135mm = 165g | | | | | | |
| | Spaghetti Bolognaise: Lean-4236; F-4060 | | T=40g; LS = 75g; ½c = 100g | | | | | | |
| | Steak & Kidney: Pie-1552; Stew-1576 | | Comm pie = 120g (30mm)<br>1LS = 100g; ½c = 135g | | | | | | |
| | Tongue: Ox-1530; Sheep:1577 | | Slice 75 x 45 x 10 = 40g | | | | | | |
| | Toppers/Imana: Cooked | 3527 | LS = 85g; ½c = 120g | | | | | | |
| | Veal: Cutlet (Fried): Plain-1671; Crumbed-1580 | | 1 Chop = 90g | | | | | | |
| | Vienna Sausage/Canned Sausage | 1531 | 100mm = 30g; 150mm = 40g | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|
| | Worms/Insects: Mopani-1676; Specify: _____ | | | | | | | | |
| | Wild Birds, Animals; Specify: _____ | | | | | | | | |
| | Other: | | | | | | | | |

| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|
| FISH | Bokkems | 2551 | 1 s/s = 25g (120mm)<br>~~L/s = 40g (135mm)~~ | | | | | | |
| | Fatty Fish: Kipper; Galjoen; Snoek; Shad | | Small 50 x 55 x 30 = 60g<br>Med 100 x 55 x 30 = 120g<br>Stew 1LS = 95; ½c = 140g | | | | | | |
| | : Fried (VO)-2535; Batter-2548; Grill-2533;<br>: Smoked-2570<br>: Salted-2551; Steam-2559;<br>: Stew-2527 (Tomato and Onion) | | | | | | | | |
| | Fish Cakes: (Fried): Home-2552; Comm-2531 | | 65 x 15mm = 50g | | | | | | |
| | Fish Fingers: (Fried) | 2532 | 85mm = 35g | | | | | | |
| | Haddock: Smoked (Boiled) | 2511 | 70 x 70 x 15 = 65g | | | | | | |
| | Pilchards: Tomato Sauce-2557; Brine-2503 | 2503 | 1 = 75g | | | | | | |
| | Sardines: + Sauce-2539; + Oil-2560 | | s/s = 7g; L/s = 25g | | | | | | |
| | Smoorsnoek: | 2525 | 1L/s = 55g; ½c = 80g | | | | | | |
| | Sole: Fried-2542; Grilled-2524 | | Baby sole: 180mm = 70g | | | | | | |
| | Tuna: Oil Pack-2547; Water-2501 | | ¼c = 50g | | | | | | |
| | White Fish: Hake, Haddock, Kingklip; Cod | | s/s piece 50 x 55 x 30 = 60g<br>Med 100 x 55 x 30 = 120g<br>Stew 1LS = 95g; ½c = 140g | | | | | | |
| | : Stew-2527 (Tom + On); Baked-2545 | | | | | | | | |
| | : Grilled-2530; Batter-2523; Fried-2509 | | | | | | | | |
| | Other: eg Fresh Water Fish; Specify:_____ | | | | | | | | |

| | | | T | LS | ½c | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| STARCH | Maize pap: Stiff (E)-4331; Crumbly (E)-4332<br>~~Stiff (P)-4255; Crumbly (P)-4256~~ | | ~~Stiff~~ | ~~75~~ | ~~120~~ | ~~125~~ | | | | |
| | Mabella/Sorghum: | 4315 | Crum | 30 | 75 | 70 | | | | |
| | Maize Rice: | 4043 | | 25 | 45 | 65 | | | | |
| | Samp: (Cooked) | 4043 | | 55 | 125 | 125 | | | | |
| | Rice: Wh-4040; Br-4134 | | | 25 | 60 | 65 | | | | |
| | Spaghetti/Macaroni: (Cooked) | 4062 | | 35 | 70 | 90 | | | | |
| | Spaghetti + Tomato Sauce | 4058 | | 45 | 80 | 125 | | | | |
| | Stamped Wheat/Wheat Rice | 4042 | | 30 | 80 | 80 | | | | |
| | + Fat: B-6502; HM 6508; PM-6521; WF-6545; VO-6510; Med-6560 | | | | | | | | | |
| | Ghee-6554 | | | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| LEGUMES AND SOUP | **Baked Beans** | 3504 | 50 | 105 | 135 | | | | | | | | |
| | **Beans:** (Cooked) Haricot-3515; Sugar-3542; Kidney-3513 | | 50 | 85 | 135 | | | | | | | | |
| | **Breyani:** Rice + Lentils + Ghee-3524; +VO-3523 | | 40 | 80 | 85 | | | | | | | | |
| | **Lentils:** Cooked/curried-3509 | | | | | | | | | | | | |
| | **Samp and Beans** (1:1)-4257 | | | | | | | | | | | | |
| | **Soup:** Comm-3054; | | | | 125 | | | | | | | | |
| | **FOOD ITEMS** | **CODE** | **QUANTITY (g/ml)** | | | **BR** | **IS** | **L** | **IS** | **D** | **AD** | | |
| | Split Pea-3045; Lentil-3041; Beef + Veg-3047; Bean-3033 | | T 35 | LS 80 | ½c 130 | | | | | | | | |
| | **"Sousboontjies"** | 3502 | 40 | 105 | 135 | | | | | | | | |
| | **Stew:** Bean + Potato + Onion | 3508 | 60 | 120 | 125 | | | | | | | | |

| | | Boiled | Fat Added (or Fried) | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | NF | B | HM | PM | VO | | T | LS | ½c | | | | | |
| COOKED VEGETABLES | **Gr Beans** | 8002 | 8080 | 8098 | 8099 | 8100 | | 25 | 60 | 80 | | | | | |
| | **Gr Bean Curry** | 8101 | | | | | 8101 | 40 | 75 | 120 | | | | | |
| | **Gr Bean + Potato + Onion** | | 8003 | 8102 | 8103 | 8104 | | | | | | | | | |
| | **Beetroot + Sugar** | 8005 | | | | | 8005 | 40 | 70 | 80 | | | | | |
| | **- No Sugar** | 8004 | | | | | 8004 | | | | | | | | |
| | **Brinjal** | 8006 | 8109 | 8110 | 8111 | 8112 | | 1 slice = 20g (70mm) + batter = 30g | | | | | | | |
| | **- Fried + Egg** | | | | | | 8113 | | | | | | | | |
| | **- + Tomato + Onion** | | 8105 | 8106 | 8107 | 8108 | | 50 | 100 | 130 | | | | | |
| | **Brocolli** | 8007 | 8114 | 8115 | 8116 | | | 25 | 60 | 75 | | | | | |
| | **Brussels Sprouts** | 8009 | 8117 | 8118 | 8119 | | | 50 | | | | | | | |
| | **Cabbage** | 8066 | 8012 | 8120 | 8121 | 8122 | | 30 | 55 | 80 | | | | | |
| | **Cab + Pot + Onion** | | 8014 | 8123 | 8124 | 8125 | | 35 | 75 | 80 | | | | | |
| | **Carrots** | 8067 | 8074 | 8126 | 8127 | 8082 | | 20 | 50 | 80 | | | | | |
| | **Car + Pot + Onion** | | 8073 | 8132 | 8133 | 8134 | | 35 | 70 | 105 | | | | | |
| | **Carrot + Sugar** | 8067 | 8019 | 8129 | 8130 | 8131 | | 25 | 50 | 85 | | | | | |
| | **Cauliflower** | 8203 | 8021 | 8135 | 8136 | 8137 | | 40 | 65 | 80 | | | | | |
| | **Caul + Cheese** | 8022 | | | | | 8022 | 43 | 70 | 90 | | | | | |
| | **Marog/Imifino*** | 8302 | | | | | 8302 | 40 | 105 | 90 | | | | | |
| | **Mealies** (corn) | 8033 | | | | | 8033 | 30 | 60 | 95 | | | | | |
| | **"Sweetcorn"** | 8034 | | | | | 8034 | 55 | 125 | 135 | | | | | |
| | **Mix Veg** (Tin/Froz) | 8035 | 8144 | 8145 | 8146 | 8147 | | 35 | 75 | 75 | | | | | |
| | **Mushroom** (Sliced) | 8037 | 8148 | 8149 | 8150 | 8151 | | 30 | 65 | 80 | | | | | |

| | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Onions (Sliced) | 8083 | 8153 | 8154 | 8155 | 8038 | | 50 | | | | | | | | | |
| Onion + Batter | | | | | | 8156 | Rings: Med = 40g | | | | | | | | | |
| Peas | 8026 | 8075 | 8166 | 8167 | 8168 | | 30 | 65 | 85 | | | | | | | |
| Peas + Sugar | 8027 | 8032 | 8169 | 8170 | 8171 | | 30 | 65 | 85 | | | | | | | |
| Potato: + Skin | 8046 | | | | | 8046 | s/s = 60; m/s = 90g | | | | | | | | | |
| : Baked + Skin | 8044 | | | | | 8044 | s/s = 60g; m/s = 90g | | | | | | | | | |
| *if indigenous specify local name _____ | | | | | | | | | | | | | | | | |
| FOOD ITEMS | | | | | | CODE | QUANTITY (g/ml) | | | BR | IS | L | IS | D | AD | |
| | NF | B | HM | PM | VO | | T | LS | ½C | | | | | | | |
| : Chips | | | | | 8048 | 8048 | ½c = 50g; med = 80g | | | | | | | | | |
| : Peeled | 8045 | 8076 | 8177 | 8178 | 8179 | | s/s = 60g; m/s = 90g; (90 x 60 x 40) | | | | | | | | | |
| : Sauté | | 8180 | 8181 | 8182 | 8183 | | 3 | 50 | 90 | | | | | | | |
| COOKED VEGETABLES | | | | | | | | | | | | | | | | |
| Potato Cake | | | | | 8234 | 8234 | 1 med = 40g (75 x 30) | | | | | | | | | |
| Pot Mash (SM) | | | 8185 | 8186 | | | 50 | 115 | 125 | | | | | | | |
| Pot Mash (WM) | | 8047 | 8187 | 8188 | | | | | | | | | | | | |
| Pot (Roast): Beef fat-8189; Chicken-8242; Lamb-8043; Pork-8275 | | | | | | | 1 med = 70g | | | | | | | | | |
| Pumpkin & Butternut | 8069 | 8050 | 8202 | 8203 | 8081 | | 45 | 85 | 105 | | | | | | | |
| Pump + Sugar | 8036 | 8051 | 8204 | 8205 | 8206 | | | | | | | | | | | |
| Pump Fritter | | | | | 8094 | 8094 | 75 x 50 x 9 = 25g | | | | | | | | | |
| Spinach | 8071 | 8055 | 8209 | 8210 | 8211 | | 40 | 105 | 90 | | | | | | | |
| Spin + Pot + Onion | | 8056 | 8212 | 8213 | 8096 | | 50 | 105 | 110 | | | | | | | |
| Squash -Gem/Marrow | 8070 | 8052 | 8193 | 8194 | 8195 | | ½ gem = 45g 1LS marrow = 85g | | | | | | | | | |
| Squash + Sugar | 8063 | 8053 | 8196 | 8197 | 8198 | | | | | | | | | | | |
| Sw Potato | 8214 | | | | | 8214 | 50 | 110 | 145 | | | | | | | |
| Sw Pot + Sugar | 8243 | 8072 | 8058 | 8215 | 8215 | | | | | | | | | | | |
| Tomato + Onion | 8244 | | | | | 8244 | 35 | 75 | 140 | | | | | | | |
| Tom + Onion +Sugar | 8221 | | | | | 8221 | | | | | | | | | | |
| Tomato | | 8218 | 8219 | 8220 | 8077 | | 1 Slice 5mm = 15g (thin); Med = 25g | | | | | | | | | |
| Turnips | 8222 | | | | 8222 | | 25 | 45 | 90 | | | | | | | |
| Other | | | | | | | | | | | | | | | | |
| SALADS | | | | | | | | | | | | | | | | |
| SALADS Asparagus-8001; Avocado-7132 | | | | | | | ¼ avo (80 x 50mm) = 40g med asparagus = 15g | | | | | | | | | |
| Beetroot (Grated) + Sugar | | | | | | 8005 | 1T = 25g; LS = 65g | | | | | | | | | |
| Carrot: Raw-8015; + Sugar-8028 (Grated) | | | | | | | 1T = 25g; | | | | | | | | | |
| + Pine + Orange-8016; + Apple + Orange-8017 | | | | | | | 1T = 35g; 1LS = 60g | | | | | | | | | |
| Coleslaw + Mayonnaise | | | | | | 8011 | T = 20g; LS = 40g; ½c = 50g | | | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|
| Cucumber Raw/Pickled | 8025 | med slice = 10g; thick = 15g | | | | | | | |
| Lettuce | 8031 | 1 med leaf = 30g | | | | | | | |
| Mixed (Tom + Cucum + Lett) - No Dressing | 8240 | 1T = 40g; 1LS = 85g | | | | | | | |
| Mixed Green - No Dressing | 8246 | | | | | | | | |
| Potato Salad + Mayonnaise (Comm) | 8247 | T = 45g; 1LS = 105g; ½c = 120g | | | | | | | |
| Tomato (Raw) | 8059 | med = 120g; slice = 15g | | | | | | | |
| Other | | | | | | | | | |

| | FOOD ITEMS | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD |
|---|---|---|---|---|---|---|---|---|---|
| DRESSINGS | French Dressing | 6512 | 1t = 5g; 1T = 15g | | | | | | |
| | Mayonnaise: Home-6535; Comm-6513 | | 1t = 10g 1T = 40g | | | | | | |
| | Mayonnaise: Low Fat | 6514 | | | | | | | |

| | FRUIT | Canned + Sugar | Raw | Dry | Stewed | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| FRUIT | Apple | 7073 | 7001 | 7074 | 7077 | | 1T = 60g; ½c = 120g; 1 med = 150g (52 x 66) | | | | | | |
| | Apricot | 7004 | 7003 | 7005 | 7006 | | 1 med = 35g | | | | | | |
| | Banana | | 7009 | | | 7009 | 1 med = 75g | | | | | | |
| | Dates | | 7012 | | | 7012 | 1 med = 10g | | | | | | |
| | Figs | | 7013 | 7027 | | | 1 med = 40g (45 x 44) 1 dry = 20g | | | | | | |
| | Fruit Salad | 7051 | 7079 | 7066 | 7062 | | ½c = 110g (med) | | | | | | |
| | Granadilla | | 7014 | | | 7014 | 1 med = 22g | | | | | | |
| | Grape Fruit | 7017 | 7016 | | | | ½ med = 125g | | | | | | |
| | Grapes | | 7020 | | | 7020 | med bunch = 230g; ½c =90g | | | | | | |
| | Guava | 7023 | 7021 | | | | med (6cm) = 95g | | | | | | |
| | Litchi | | 7107 | | | 7107 | med (3cm) = 8g | | | | | | |
| | Mango | | 7026 | | | 7026 | 135mm = 350g | | | | | | |
| | Naartjie | 7110 | 7028 | | | | med = (5cm) = 75g | | | | | | |
| | Orange | | 7031 | | | | med (7cm) = 180g | | | | | | |
| | Pawpaw | 7114 | 7034 | | | | wedge 165 x 26 x 27 = 90g | | | | | | |
| | Peach | 7038 | 7036 | 7039 | 7040 | | 1 med = 150g (60 x 65) | | | | | | |
| | Pear | 7054 | 7053 | 7056 | 7057 | | 1 med (80 x 65mm) = 165g | | | | | | |
| | Pineapple | 7123 | 7052 | | | | 1 slice (85 x 10mm) = 40g | | | | | | |
| | Plum | | 7041 | | | 7041 | 1 med = 50g (45 x 40) | | | | | | |
| | Prunes | 7154 | | 7069 | 7035 | | 1T = 50g; ½c = 110g; 1 = 12g | | | | | | |
| | Raisins | | 7022 | | | | handfull = 27g | | | | | | |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Strawberries | 7129 | 7044 | | | | 1 med = 12g; ½c = 80g | | | | | | |
| | Sweetmelon | | 7046 | | | | 1 wedge (145 x 31 x 20mm) = 60g; ¼ = 110g | | | | | | |
| | Watermelon | | 7047 | | | 7014 | Slice (330 x 70mm) = 220g | | | | | | |
| | Wild Fruit, Berries: Specify: _____ | | | | | | | | | | | | |

| | PUDDINGS | SM | WM | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PUDDINGS | Apple + Batter | 4178 | 4154 | | med serving = 70g | | | | | | | |
| | Apple Crumble | | 4165 | | med serving = 70g | | | | | | | |
| | Baked Pudd + Syrup | 4181 | 4131 | | med serving = 30g<br>30 x 65 x 65 = 50g | | | | | | | |
| | - No Syrup | 4180 | 4013 | | | | | | | | | |
| | FOOD ITEMS | | | CODE | QUANTITY (g/ml) | BR | IS | L | IS | D | AD | |
| | | SM | WM | | | | | | | | | |
| PUDDINGS | Blancmange | 4090 | 4089 | | LS = 75; ½c = 95g | | | | | | | |
| | Egg Type eg. Bread, Sago | 4179 | 4063 | | 1T = 50g; ½c = 140g;<br>LS = 100g | | | | | | | |
| | Ice Cream | | | 6507 | Scoop = 40g; 1LS = 65g;<br>½c = 75g | | | | | | | |
| | Instant Pudding | 4133 | 4066 | | T = 45g; LS = 95g; ½c = 145g | | | | | | | |
| | Jelly | | | 9004 | 1T = 35g; 1LS = 75g;<br>½c = 110g | | | | | | | |
| | Jelly + Fruit | | | 9033 | 1T = 40g; 1LS = 90g;<br>½c = 125g | | | | | | | |
| | Jelly Whip | 0037 | 0038 | | 1T = 55g; LS = 95g;<br>½c = 120g | | | | | | | |
| | Pancake/Crumpets | 4177 | 4030 | | 1 crumpet = 25g<br>pancake = 70g | | | | | | | |
| | Soft Serve | | | 6547 | plain = 135g; + flake = 155g | | | | | | | |
| | Sorbet | | | 6516 | 1LS = 65g; ½c = 75g | | | | | | | |
| | Trifle-4130; Vermicelli Pudding-4233 | | | | ½c = 130g (med) | | | | | | | |

| | SAUCES | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| SAUCES | ~~Cream: Plant-6517; Canned-6524~~ | | | | | | | | | |
| | - Light-6504; Heavy-6503 | | | 1T = 30g | | | | | | |
| | Chocolate Sauce | | 3016 | T = 15g | | | | | | |
| | Custard: SM-0005; WM-0004 | | | T = 13g; LS = 40g | | | | | | |
| | Sugar | | 9012 | 1t = 6g | | | | | | |
| | Other | | | | | | | | | |

| | ~~Banana Loaf: WM + HM-4164; SM + PM-4214~~ | | ~~slice = 45g; 90 x 80 x 10mm~~ | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| CAKE | Cake -Carrot | 4244 | 80 x 40 x 40 = 50g | | | | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | - Plain: SM + B-4009; HM-4095; PM-4096 | | single slice = 50g (75 x 75 x 20mm) Double slice = 100g (plain) Icing = 10g per slice | | | | | | |
| | WM + B-4009; HM-4097; PM-4101 | | | | | | | | |
| | Cake Icing: HM-9042; PM-9043 | | | | | | | | |
| | - Chocolate (No Icing) WM-4099; SM-4170 | | | | | | | | |
| | - Fruit: Comm-4102; Home-4305 | | Home: 70 x 85 x 15mm = 70g Comm: 90 x 70 x 15mm = 35g | | | | | | |
| | - Sponge (Plain) | 4011 | 100 x 50 x 50 = 40g | | | | | | |
| | - Swiss Roll | 4103 | slice = 60g; 15cm thick | | | | | | |
| | Cheese Cake: Baked-4108; Unbaked-4109 | | slice 95 x 50 x 30mm = 70g | | | | | | |
| | **FOOD ITEMS** | **CODE** | **QUANTITY (g/ml)** | **BR** | **IS** | **L** | **IS** | **D** | **AD** |
| CO OKI :S | **COOKIES** Comm + fill-4008, Plain-4007 | | plain = 10g + fill = 15g | | | | | | |
| | - Home: Plain HM-4025; PM-4172 | | plain = 15g + fill = 20g hertzog = 50g; cupcake = 35g shortbread = 12g | | | | | | |
| | Jam-4110; Oats-4065; Shortbread-4111 | | | | | | | | |
| | Custard Slice | 4169 | 110 x 45 x 35mm = 250g | | | | | | |
| | Date Loaf; HM-4054; PM-4171 | | slice 90 x 75 x 10mm = 40g | | | | | | |
| | Doughnuts: Jam-4301; Plain-4024 | | med round = 45g med long = 90g | | | | | | |
| | Eclairs + Cream + Chocolate | 4070 | 1 = 120g (160mm) | | | | | | |
| | Gingerbread: HM-4047; PM-4215 | | 90 x 75 x 15 = 70g | | | | | | |
| | Koeksister | 4023 | 100 x 35 = 60g | | | | | | |
| | Raisin Bread | 4005 | slice 85 x 100 x 10mm = 30g | | | | | | |
| TA RT S | **TARTS** Apple: HM-4016, PM-4192 | | 50 x 50 x 50mm = 70g (med) | | | | | | |
| | Coconut | 4020 | Wedge 50 x 100 x 30mm= 55g | | | | | | |
| | Condensed: HM-4109; PM-4317 | | 95 x 70 x 30mm = 90g | | | | | | |
| | Fridge (Fruit): HM-4246; PM-4312 | | | | | | | | |
| | Lemon Meringue: HM-4018; PM-4184 | | 100 x 70 x 35mm = 75g | | | | | | |
| | Milk (Short) WM + HM-4202; SM + PM-4189 | | 120 x 70 x25mm = 75g | | | | | | |
| | Milk (Flaky) WM + B-4321; WM + HM-4021 | | | | | | | | |
| | Savoury: Aspar-4210; Tuna-4209; Vienna-4153 | | 120 x 50 x 25 = 75g | | | | | | |
| | Tipsy: HM-4147; Jam-4017 | | 87 x 70 x 50mm = 90g | | | | | | |
| SW EE TS | **SWEETS** Bubble/Chewing gum | 9019 | See manual | | | | | | |
| | Chocolates: Assorted | 9017 | | | | | | | |
| | Coated Bars eg. Tex, Lunch, Chomp | 9024 | | | | | | | |
| | Milk eg. Smarties, Flake, Aero | 9010 | | | | | | | |

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|---|---|---|---|---|---|---|---|---|---|---|
| | Nuts/Raisins | 9020 | | | | | | | | |
| | Plain | 9030 | | | | | | | | |
| | Dry Fruit Sweets | 9021 | | | | | | | | |
| | Fruit Gums | 9027 | | | | | | | | |
| | Hard/Jelly Sweets eg. Sugus, Jelly Tots, Fruit drops | 9009 | | | | | | | | |
| | Ice Lollies | 9002 | | | | | | | | |
| | Marshmallows | 9028 | | | | | | | | |
| | Meringues | 9035 | | | | | | | | |
| | Peanuts-6007; Peanut Brittle-9029 | | | | | | | | | |
| | Peppermints | 9031 | | | | | | | | |
| | **FOOD ITEMS** | **CODE** | **QUANTITY (g/ml)** | **BR** | **IS** | **L** | **IS** | **D** | **AD** |
| SWEETS | Popcorn: Plain-4163; Sugar Coated-4201 | | SEE MANUAL | | | | | | |
| | Potato Crisps eg. Simba, O'Gradys | 4275 | | | | | | | |
| | Snacks - Fritos, Niknaks | 4067 | | | | | | | |
| | Soft Sweets - Fudge, Toffees, Caramel | 9014 | | | | | | | |
| OTHER | OTHER | | | | | | | | |
| | ~~Cheese Sauce: WM + HM-3012; SM + PM-3015~~ | | ~~LS = 65g; 1T = 25g~~ | | | | | | |
| | Curry Sauce | 3017 | 1T = 25g | | | | | | |
| | Chutney-3057; Atjar-3004 | 3057 | 1T = 14g; 1T = 60g | | | | | | |
| | Gravy: Comm-3006; Meat-3009; NF-3008 | | 1T = 15g; LS = 35g | | | | | | |
| | Mustard | 9509 | 1t = 6g | | | | | | |
| | Tomato Sauce (Comm) | 3027 | 1t = 6g; 1T = 25g | | | | | | |
| | White Sauce: WH + HM-3030; SM + PM = 3029 | | | | | | | | |
| INFANT FOODS | **Baby Cereals** (dry):<br>Nestum 1-0501; 2-0503<br>Purity: Mixed-0511; Wholewheat-0530; Rice-0531<br>Cerelac-0505; Nestum Rice & Maize-0504<br>Junior-0502 | | 1t = 2g<br>1T = 8g<br>½c = 20g | | | | | | |
| | Milk: SM-0007; WM-0006; BI-0068; 2%-0069; Breast-0029;<br>Goat-0026; FORMULA (Specify) : _____<br>No. of scoops/bottle used _____ | | To drink ½c = 125ml<br>Baby bottle = 250ml | | | | | | |
| | + Sugar | 9012 | 1t = 6g | | | | | | |
| | First Food Fruit 0521; First Food Veg-0520 | | Jar = 80g; 1t = 11g | | | | | | |
| | Fruit Juice (Strained)-0529; Fruit Juice-0535 | | ½c = 125ml | | | | | | |
| | Infant Dinners (Dry): Beef + Veg=0510; Chicken + Veg-0509<br>Guava + Custard-0506; Mix Veg-0508;<br>Orange + Banana-0507 | | 1t = 5g<br>1T = 15g<br>½c = 47g | | | | | | |
| | Junior Food (Jar): Veg + Meat-0517; Mix Veg-0518;<br>Pasta + Beef-0519<br>Junior Fruit (Jar): Fruit-0532; Guava-0524<br>Junior Pudding: Fruit+Yog-0527; Vanilla Cust-0528 | | Jar = 200g<br>1t = 11g<br>½c = 125g | | | | | | |

OTHER: