

**TITLE: DETERMINATION OF THE GLYCAEMIC INDEX
OF THREE TYPES OF ALBANY SUPERIOR™ BREAD**

**MARTHA JACOMINA VAN ZYL
B.Sc. Dietetics**

**Mini-dissertation submitted in partial fulfillment of the
requirements for the degree Magister Scientiae in Dietetics**

**in the
Faculty of Health Sciences
Department of Human Nutrition
University of the Free State
Bloemfontein
South Africa
November 2006**

Supervisor: Prof. M. Slabber-Stretch

Co-supervisor: Dr. C.M. Walsh

DECLARATION

I declare that the dissertation hereby submitted by me for the Magister degree at the University of the Free State is my own independent work and has not previously been submitted by me to another university/faculty. I further cede copyright of this research report in favour of the University of the Free State

Martha Jacomina van Zyl

November 2006

**To my beloved husband and
four furry friends**

ACKNOWLEDGEMENTS

This study would not have been possible without the mercy of our Heavenly Father, who gave me the strength, courage and perseverance to complete this study.

My gratitude and sincere thanks are expressed to the following people and organizations. Without their support this project could not have been possible:

- My supervisor Prof. M. Slabber, for her knowledge, advice and assistance as well as excellent guidance
- Dr. C.M. Walsh, for her patience, excellent advice and encouragement in completion of this mini-dissertation
- Dr. J.H. van der Linde and Sister Pearl for their time and professional assistance in the execution of the study
- R. Nel from the Department of Biostatistics at the University of the Free State, for the statistical analysis of the data
- Letsia Kruger, from the Department of Surgery at the University of the Free State
- The subjects who participated in the study
- My parents, family and friends for their encouragement, support and interest. Special thanks to my husband John, without whom I could not have completed this study. Thank you for your understanding and support.
- Tiger Brands for financial support of this research.

ABSTRACT

Introduction

The glycaemic index (GI) concept was introduced as a means of classifying different sources of carbohydrates (CHO) and CHO-rich foods in the diet, according to their effect on postprandial glycaemia since different carbohydrate containing foods have different effects on blood glucose responses. The GI is defined as the incremental area under the blood glucose response curve of a 50 g glycaemic (available) carbohydrate portion of a test food expressed as a percentage of the response to the same amount of glycaemic CHO from a standard food taken by the same subject. Though not the only factor that will determine whether the food should be included in the diet or not, the GI can be used alongside current dietary guidelines like the Food Based Dietary Guidelines and exchange lists to guide consumers in choosing a particular food with a predicted known effect on blood glucose levels and homeostasis.

Variation in the GI values for apparently similar foods may reflect both methodologic factors as well as true differences in the physical and chemical characteristics of the specific food. Differences in GI values of similar foods could also be due to inherent botanical differences from country to country. Two similar foods may also have different ingredients, different processing methods or different degree of gelatinisation resulting in significant variation in the rate of CHO digestion and consequently the GI value. Methodological variables which include food-portion size, the method of blood sampling, sample size and subject characteristics, standard food, available CHO, volume and type of drinks consumed with test meals can markedly affect the interpretation of the glycaemic responses and the GI value obtained.

Tiger Brands commissioned an independent assessment of the GIs of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread carried out under strictly standardised conditions using methods complying with the most recent internationally accepted methodology.

Methods

Twenty healthy, fasting male volunteers, aged 18-27 years, each randomly consumed six different test meals consisting of 50 g available carbohydrates from three different test foods (three types of

Albany Superior breads) and one type of standard food (glucose) (repeated three times in each subject) according to a Latin square design. Finger-prick capillary blood was collected fasting and within 10-15 min after the first bite was taken for every 15 min time interval for the first hour and thereafter for every 30 min time interval for the second hour, using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™). The AUC and GI for the three different breads, were calculated using the mean of the three glucose responses (standard meals) as standard. Statistically significant differences were also determined.

Results

The mean GIs were 78.44, 72.01 and 79.62 for Whole Wheat™, Brown™ and Best of Both™ bread respectively. No statistically significant differences were found between the GIs of the three different Albany Superior™ breads.

Conclusions

From the study it can be concluded that the three different Albany Superior™ breads fell between the intermediate and high categories.

Recommendations

It is recommended that the methodological guidelines determined by the GI Task Force should be followed. It is also important to inform patients and consumers that in using the GI to choose CHO foods it is a fact that physiological responses to a food may vary between individuals and that it is normal for a specific food to have a high GI in some individuals and a medium or even a low GI in others. For labeling purposes it is recommended that the GI is presented as a mean with 95% confidence intervals.

Keywords: glycaemic index, three Albany Superior breads

OPSOMMING

Inleiding

Die glukemiese indeks (GI) -beginsel is in gebruik geneem ten einde verskillende bronne van koolhidrate en koolhidraatryke voedsel te klassifiseer volgens hul effek op post-prandiale bloedglukose aangesien verskillende koolhidraat-bevattende voedsel verskillende effekte het op bloedglukose reaksies. Die GI word gedefinieer as die inkrementele area onder die bloedglukoseresponskurwe vir 'n toetsvoedsel wat 'n 50 g glukemiese (beskikbare) koolhidraatporsie bevat, in verhouding tot (uitgedruk as persentasie) die ooreenstemmende area onder die kurwe nadat dieselfde koolhidraatporsie van 'n standaardvoedsel deur dieselfde persoon ingeneem is. Alhoewel die GI nie die enigste faktor is wat bepaal of 'n voedselsoort in die dieet ingesluit moet word of nie, kan die GI met huidige dieetriglyne bv. die "Food Based Dietary Guidelines" en Ruillyssisteem geïntegreer word om sodoende verbruikers by te staan in hul keuse van voedsel met 'n bekende geskatte effek op bloedglukosevlakke en homeostase.

Variasie in die GI vir skynbaar soortgelyke voedsel kan beide metodologiese faktore asook werklike verskille in die fisiese en chemiese kenmerke van spesifieke voedsel reflekteer. Verskille in GI-waardes van soortgelyke voedsel kan moontlik toegeskryf word aan verskille in botaniese kenmerke eie aan 'n spesifieke land. Twee soortgelyke voedsels kan moontlik ook verskil wat betref bestanddele, prosesseringsmetode en graad van gelatinisasie wat tot variasie in die tempo van CHO-vertering en dus gevolglik die GI-waarde kan lei. Metodologiese veranderlikes wat insluit voedselporsiegrootte, die metode van bloedinsameling, steekproefgrootte en proefpersoonkenmerke, standaardvoedsel, beskikbare CHO, volume en tipe vloeistof wat tydens toetsmaal ingeneem word, kan die interpretasie van glukemiese response en die GI waarde wat verkry word noemenswaardig beïnvloed.

'n Voedselmaatskappy het opdrag gegee dat 'n onafhanklike bepaling van die GIs van drie Albany Superior™ brode naamlik "Best of Both™", "Brown™" en "Whole Wheat™" gedoen word, onder streng gestandaardiseerde toestande deur gebruik van mees onlangse internasionaal aanvaarbare metodologie.

Metodes

'n Groep van 20 gesonde, vastende manlike vrywilligers, 18-27 jaar oud, het elk ewekansig 50 g beskikbare koolhidrate vanaf drie verskillende toetsvoedsels (drie tipes Albany Superior brode) en een tipe standaardvoedsel naamlik glukose wat drie keer herhaal is in elke proefpersoon, in ses verskillende toetsmaaltye ingeneem, volgens 'n Latynse vierkantontwerp. Kapillêre bloed, d.m.v. vingerprik, deur gebruik te maak van One Touch Ultra™ toetsstrokies en One Touch Ultra™ glukosemeters (Lifescan™), is versamel vastend, binne 10-15 min nadat die toetsmaal 'n aanvang geneem het vir elke 15 min tydsinterval van die eerste uur en daarna vir elke 30 min tydsinterval van die daaropvolgende uur. Die area onder die kurwe (AUC) en GI vir die drie verskillende brode, is bereken deur die gemiddeld van die drie glukose response (standaardvoedsel) as standaard te gebruik. Statisties betekenisvolle verskille is ook bepaal.

Resultate

Die gemiddelde GIs was respektiewelik 78.44, 72.01 and 79.62 vir "Whole Wheat™", "Brown™" and "Best of Both™" brood. Geen statisties betekenisvolle verskille is tussen die GIs van die drie verskillende Albany Superior™ brode gevind nie.

Gevolgtrekkings

Die gevolgtrekking kan uit die studie gemaak word dat die GIs van drie verskillende Albany Superior™ brode tussen die intermediêre tot hoë GI kategorieë val.

Aanbevelings

Dit word aanbeveel dat die metodologiese riglyne soos opgestel deur die GI Werkgroep gevolg moet word. In die gebruik van die GI om koolhidraatvoedsel te kies, moet pasiënte en verbruikers bewus gemaak word van die feit dat fisiologiese response tot 'n voedsel tussen individue mag varieer en dat dit normaal is vir 'n spesifieke voedsel om tot 'n hoë GI in sommige individue en tot 'n medium of selfs lae GI in ander, aanleiding te gee. Die aanbeveling word gemaak dat die GI vir etiketteringsdoeleindes, as 'n gemiddeld met 'n 95% vertrouensinterval voorgestel word.

Sleutelwoorde: glukemiese indeks; drie Albany Superior brode

TABLE OF CONTENT

Declaration of independent work	ii
Acknowledgements	iv
Abstract	v
Opsomming	vii
List of tables	xiii
List of figures	xiv
List of appendices	xv
List of abbreviations	xvi
CHAPTER 1: INTRODUCTION	1
1.1 Introduction	1
1.2 Objectives of the study	4
1.3 Structure of the mini-dissertation	4
CHAPTER 2: LITERATURE REVIEW	5
2.1 Introduction	5
2.2 Definition of the glycaemic index (GI) and glycaemic load (GL)	5
2.2.1 Glycaemic index (GI)	5
2.2.2 Glycaemic load (GL)	6
2.3 Criticism and misconceptions of the GI	6
2.4 Methodology used to determine the GI	10
2.4.1 Subjects	10
2.4.1.1 Within- and between-subject variation	11
2.4.1.2 Type of subjects	12
a) Health status	12

b) Age	13
c) Ethnicity	13
d) Gender	13
e) Body mass index	14
2.4.1.3 Number of subjects	14
2.4.2 50 g carbohydrate portion	14
2.4.3 Standard food	15
2.4.4 Pre-test meal	16
2.4.5 Test foods	17
2.4.6 Volume and type of drinks consumed with test meals	17
2.4.7 Blood sampling	18
2.4.8 Calculation of the area under the curve (AUC)	18
2.5 Factors influencing GI determination	19
2.5.1 Non-food factors	20
2.5.2 Food factors	20
2.5.2.1 Carbohydrates	21
a) Nature of the monosaccharide	21
b) Nature of the starch (chemical structure)	22
2.5.2.2 Dietary fibre and resistant starch	23
2.5.2.3 Protein and Fat	24
2.5.2.4 Food processing	25
2.5.2.5 Anti-nutrients	26
2.5.2.6 Organic acids	27
2.5.2.7 Ripening and food storage	27
2.5.2.8 Other factors	27
a) The influence of mixed meals on the GI	27
b) Second meal effect	28
2.6 Health benefits of low-GI diets	29
2.6.1 Diabetes mellitus	29
2.6.2 Coronary heart disease	31
2.6.3 Carbohydrate metabolism	32

2.6.4	Lipid metabolism	33
2.6.5	Obesity	34
2.6.6	Cancer	35
2.6.7	Exercise performance	36
2.6.7.1	Pre-exercise	36
2.6.7.2	During exercise	37
2.6.7.3	Recovery after exercise	37
2.7	Labelling	38
2.8	Summary	40
CHAPTER 3: METHODOLOGY		43
3.1	Introduction	43
3.2	Objectives	43
3.3	Methods	43
3.3.1	Subjects	43
3.3.2	Study design	44
3.3.3	Operational definitions	44
3.3.3.1	Glycaemic Index	44
3.3.3.2	Food-portion size	44
3.3.3.3	Glycaemic carbohydrates	45
3.3.4	General procedures	45
3.3.5	Detail on capillary whole blood sampling	48
3.3.6	Standard food	49
3.3.7	Test food	49
3.3.8	Methodological and measurement errors	50
3.3.8.1	Validity and reliability	51
a)	Validity	51
b)	Reliability	51
3.3.9	Pilot study	52
3.3.10	Statistical analysis	52

3.3.11	Implementation of findings	52
3.3.12	Ethical aspects	53
3.3.13	Limitations of the study	53
CHAPTER 4: RESULTS, DISCUSSION AND RECOMMENDATIONS		55
4.1	Introduction	55
4.2	Subject characteristics	55
4.3	Within-subject and between-subject variation in blood glucose responses to three glucose reference tests	57
4.4	Blood glucose responses of glucose and the three different Albany Superior™ breads	58
4.5	The AUCs for the three different Albany Superior™ breads	59
4.6	The GIs of the three different Albany Superior™ breads	60
4.7	Conclusions	64
4.8	Recommendations	65
BIBLIOGRAPHY		68
APPENDICES		87

LIST OF TABLES

PAGE

• Table 2.1 Factors that influence the glycaemic index	21
• Table 3.1 Latin square design for subjects	44
• Table 3.2 Pre-evening test meal	48
• Table 3.3 Time schedule for blood sampling per test day	49
• Table 3.4 Macronutrient composition of the three different breads	50
• Table 4.1. Subject characteristics	56
• Table 4.2. The AUC for individual subjects for the three glucose reference tests (GRT) using capillary blood	57
• Table 4.3. The mean AUC for the three glucose reference tests using capillary blood	58
• Table 4.4. Blood glucose responses after every time interval for the three Albany Superior™ breads as well as glucose (standard food)	59
• Table 4.5. The mean AUC for the three different Albany Superior™ breads and glucose using capillary blood.	60
• Table 4.6. The GIs for individual subjects for the three different Albany Superior™ breads using capillary blood.	61
• Table 4.7. The mean GI for the three different Albany Superior™ breads using capillary blood	63
• Table 4.8 Suggestions on how the GI may be incorporated into current dietary advice	66
• Table 4.9 Substituting lower-GI foods for high-GI foods	67

LIST OF FIGURES

PAGE

- Figure 2.1 Proposed form of GI labeling 40
- Figure 3.1 The procedure for the study 46

LIST OF APPENDICES

PAGE

- APPENDIX A: Permission letter 87
- APPENDIX B: Recruitment form 91
- APPENDIX C: Informed consent form 92
- APPENDIX D: Glycaemic index testing information form (data form) 98

LIST OF ABBREVIATIONS

%	percentage
&	and
α	alfa
β	beta
<	less than
>	greater than
≤	less and equal than
≥	greater and equal than
=	equal
±	plus minus
°C	degree celcius
ADA	American Dietetic Association
AUC	area under the curve
AUC_{min}	area under the curve (minimum as baseline)
AX	arabinoxylan
BMI	body mass index
CHD	coronary heart disease
CHO	carbohydrate
DoH	Department of Health
<u>et al.</u>	Et alii
ETOVS	Ethics Committee, Faculty of Health Science, UFS
FAO/WHO	Food and Agricultural Organization/World Health Organization
FFA	free fatty acids
Fig.	figure
GI	glycaemic index
GRT	glucose reference test
GL	glycaemic load
g	gram
HbA_{1c}	glycosylated haemoglobin
HDL	high density lipoproteins
HDLC	high density lipoprotein cholesterol

IGFs	insulin-like growth factors
kJ	kilojoules
kg	kilogram
kg/m²	kilogram per square meter
LDL	low density lipoproteins
LDLC	low density lipoprotein cholesterol
ml	millilitre
min	minutes
mmol/L	millimol per litre
NSP	nonstarch polysaccharides
OS	oxidative stress
P	product (testfood)
P1	Whole wheat™ bread
P2	Brown™ bread
P3	Best of Both™ bread
RAG	rapidly available glucose
RS	resistant starch
S	standard food (glucose)
SAG	slowly available glucose
SCFA	short-chain fatty acids
TC	total cholesterol
TG	triacylglycerols
UFS	University of the Free State
UK	United Kingdom
VLDL	very low density lipoproteins

CHAPTER 1: INTRODUCTION

1.1 Introduction

The glycaemic index (GI) concept was introduced in 1981 (Jenkins et al., 1981), as a means of classifying different sources of carbohydrates (CHO) and CHO-rich foods in the diet, according to their effect on postprandial glycaemia since different carbohydrate containing foods have different effects on blood glucose responses (Brouns et al., 2005; Jenkins et al., 1981; Wolever, 1990).

The effect of different carbohydrate foods on the glycaemic response of healthy and diabetic subjects has been studied extensively. In essence, the GI is a ranking of foods which indicates a food's ability to raise blood glucose concentrations, relative to a standard food (glucose or white bread) (Jenkins et al., 1981). According to the GI Task Force (2002) the GI is defined as "the incremental area under the curve for the increase in blood glucose after the ingestion of 50g of glycaemic carbohydrates of a test food (unless the total volume exceeds 300ml when 25g of glycaemic [available] carbohydrate from the test food and reference food will be acceptable) in the 2-hour for healthy and 3-hour for diabetic individuals from the start of the test meal, as compared with ingestion of the same amount of glycaemic (available) carbohydrate from glucose taken with 300 ml of water spread over a 10-15 minute period, tested according to a defined procedure by an accredited laboratory in the same individuals under the same conditions using fasting blood glucose concentrations as a baseline."

According to Brouns et al., (2005) low-GI CHO's are classified as those CHO's that are digested and absorbed slowly and lead to a low glycaemic response, whereas high-GI CHO's are rapidly digested and absorbed and show a high glycaemic response. The rate of glucose entry into blood and the duration of the elevated blood glucose are known to induce many hormonal and metabolic changes that may affect health and disease parameters. In this respect, low-GI foods have often been found to induce benefits on risk factors for certain chronic diseases. Because of these observations it was proposed that GI data for foods could be used to make priorities for food selection within food groups.

Several studies have shown that eating a low GI diet has health benefits. Evidence from prospective studies shows that low GI-diets are associated with reduced risk of diabetes (especially type 2 diabetes) (Hodge *et al.*, 2004; Frost *et al.*, 1998; Salmerón *et al.*, 1997a; Salmerón *et al.*, 1997b), cardiovascular disease (Liu *et al.*, 2000), cancer (Augustin *et al.*, 2001; Augustin *et al.*, 2002; Augustin *et al.*, 2003a; Augustin *et al.*, 2003b; Augustin *et al.*, 2004a; Augustin *et al.*, 2004b; Augustin *et al.*, 2004c), and the metabolic syndrome (McKeown *et al.*, 2004). Low GI foods improve overall blood glucose control in people with type 2 diabetes (Wolever *et al.*, 1992), reduce serum lipids in people with hypertriglyceridaemia (Jenkins *et al.*, 1987) and improve insulin sensitivity (thus reducing insulin demand) (Frost *et al.*, 1998; Riccardi and Rivellese, 2000; Augustin *et al.*, 2002; Slyper, 2004). In addition, the intake of a low GI-diet is associated with higher concentrations of high-density lipoprotein (HDL) cholesterol (Frost *et al.*, 1999) and significant reductions in low-density lipoprotein (LDL) cholesterol as well as total cholesterol (Opperman *et al.*, 2005).

When incorporated into an energy restricted diet, low glycaemic CHO's, compared to higher glycaemic CHO's, leads to a reduction in insulin resistance that cannot be accounted for by weight-loss alone (Slabber *et al.*, 1994). According to Agus *et al.* (2000), low-GI diets also influence body weight and resting energy expenditure independently of energy intake in young moderately overweight subjects. Several studies (Roberts, 2000; Gulliford *et al.*, 1989) have shown that high-glycaemic CHO also leads to hunger and CHO craving. Ludwig (2000, as referred to by Slyper, 2004) who recorded 15 studies in the adult literature, demonstrated increased satiety, delayed return of hunger, and decreased food intake, after ingestion of low-GI compared with high-GI foods. Low-GI diets may thus also play an extensive role in weight loss.

The usefulness of the GI in diet planning has been endorsed by the Joint FAO/WHO expert consultation "Carbohydrates in Human Nutrition" (1998) and Riccardi and Rivellese (2000) due to these beneficial effects to health. However, according to Pi-Sunyer (2002) there are still many questions being asked regarding the validity of the GI for determining what foods are "good" and "bad" for one's health. Much more definitive data from controlled clinical trials are needed before any such dietary recommendations are made as part of standard treatment modalities (Pi-Sunyer, 2002).

Controversial opinions have been made public regarding the relevance of classifying foods according to their glycaemic responses by using the GI (Bessenen, 2001). Part of the controversy is due to methodological variables that can markedly affect the interpretation of glycaemic responses and the

GI values obtained (Wolever, 1990). Methodological variables that affect the GI value include food-portion size, the method of blood sampling, sample size and subject characteristics [within-subject variation, body mass index (BMI)], standard food, available CHO, volume and type of drinks consumed with test meals (Wolever *et al.*, 1991; Venter *et al.*, 2003).

The GI concept was endorsed in the Joint Food and Agriculture Organization/World Health Organization (FAO/WHO) report (1998) that reviewed the available research evidence regarding the importance of CHO in human nutrition. Afterwards other international expert groups including the European Dietetic Association and the Canadian Diabetes Association also endorsed the GI concept in their dietary guidelines. Health professionals in Australia (Foster-Powell and Miller, 1995) have developed official dietary guidelines for healthy consumers as well as a GI trademark certification program for food labelling, in Australia, the most advanced country in terms of knowledge of GI of foods (Venter, Slabber and Vorster, 2003). Locally, the GI concept is acknowledged in the South African Food-Based Dietary Guidelines (Vorster and Nell, 2002) referring to the beneficial effects of low-GI foods in the context of preventing chronic disease. The inclusion of high-GI foods is also highlighted in these guidelines, as the preferred choice in specific circumstances such as restoring glycogen stores after exercise (Vorster and Nell, 2002). A task force was appointed in 2002 by the Directorate of Food Control to standardise the procedure for determining the GI in South Africa, as there is currently no international standard besides the method described by the FAO/WHO (1998).

Requirements for claims regarding the GI value of carbohydrate-rich foods are included in a concept regulation regarding food packaging in South Africa (Amended Foodstuffs, Cosmetics and Disinfectants Act, 54/1972). Although it is still a draft regulation, the GI concept seems to be acceptable and useful according to research in South Africa as well as internationally (Venter, *et al.*, 2003). According to Pieters and Jerling (2005), an expert group was assembled in 2002 by the Department of Health to develop a standardised method for GI determination. This was meant for use in South Africa, paving the way for GI labelling and subsequent consumer education. Such issues include the practice of unstandardised methodology in determining the GI (number of subjects, standard food, white bread or glucose, venous or capillary blood, method of determining glucose, calculation/measurement of glycaemic CHO in foods, etc.) and how to express the GI on the food label (mean, standard deviation, 95% confidence interval, low versus medium versus high and cut-off-points of these categories, etc.), as well as how to handle the often large variations in the GI of a specific food and the day-to-day variations in glycaemic responses of individuals. GI values are generally reproducible from country to country, but in some instances there are variations

due to inherent botanical differences of foods. To date the GI of a large number of South African products, especially certain breads, remains undetermined or not determined by using the prescribed methodology. Therefore, the Department of Human Nutrition, UFS was requested by Tiger Brands to determine the GI values of three Albany Superior™ breads, namely Best of Both™, Brown™ and Whole Wheat™ bread.

1.2 Objectives of this study

The aim of this study was to determine the GI of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread using capillary blood sampling to determine whether there were significant difference between the GIs of the products mentioned.

1.3 Structure of the mini-dissertation

The mini-dissertation is divided into five chapters.

The first chapter summarizes the methodological issues regarding the determination of the GI and the objectives for the study. The structure of the mini-dissertation is then outlined.

The second chapter of the mini-dissertation consists of a review of the relevant literature.

In Chapter 3 the methodology used during the study is presented according to the most recent laboratory guidelines based on the results of international studies and the recommendations of the South African GI Task Force (2002).

In Chapter 4 the results are presented, including the area under the curve (AUC) and the GI of the three different Albany Superior™ breads. Thereafter results of the study are discussed and conclusions are drawn, after which recommendations are made for further research.

Chapter 2: LITERATURE REVIEW

2.1 INTRODUCTION

Until recently CHO have been classified as 'simple' and 'complex' based on their degree of polymerisation, however, their effects on health may be better described on the basis of their physiological effects (e.g. ability to raise blood glucose), which depend both on the type of constituent sugars (e.g. glucose, fructose, galactose) and the physical form of the CHO (e.g. particle size, degree of hydration). This classification is referred to as the glycaemic index (GI). The GI is a quantitative assessment of foods, originally introduced as a means of classifying different sources of CHO and CHO-rich foods in the diet, according to their effect on postprandial glycaemia (Jenkins et al., 1981, 1984; Augustin et al., 2002). The systemic classification of foods according to their glycaemic responses was first undertaken by Otto and Niklas in 1980 (Wolever et al., 1991). One year later, Jenkins and co-workers independently developed the concept known as the GI (Jenkins et al., 1981).

2.2 DEFINITION OF THE GLYCAEMIC INDEX (GI) AND GLYCAEMIC LOAD (GL)

2.2.1 Glycaemic index (GI)

The GI is a classification of the blood glucose-raising potential of CHO foods. It is defined as the *incremental area* under the blood glucose response curve of a *50 g glycaemic (available) carbohydrate portion* of a test food expressed as a percentage of the response to the same amount of glycaemic CHO from a *standard food* taken by the same subject (Pieters and Jerling, 2005; Nell et al., 2003; FAO/WHO Expert Consultation Group, 1998; GI Task Force, 2002). Low-GI CHO are classified as those that are digested and absorbed slowly and lead to a low glycaemic response, whereas high-GI CHO are rapidly digested and absorbed and show a high glycaemic response (Brouns et al., 2005). The italicised items are discussed later in Chapter 2 because different interpretations of these concepts may profoundly affect the GI obtained.

2.2.2 Glycaemic load (GL)

The term glycaemic load (GL) was introduced in 1997 by researchers from Harvard University. It is defined as the GI multiplied by the amount (grams) of available CHO in a specific portion of a CHO-containing food (Salmerón *et al.*, 1997a, b; Wylie-Rosett *et al.*, 2004). According to Shikany *et al.* (2006) GL is a measure that incorporates both the quality and quantity of dietary CHO. Wylie-Rosett *et al.* (2004) stated that GL was developed as a way of comparing the glucose-raising effect of foods with widely differing amounts of CHO's. The higher the GL, the greater the expected elevation in blood glucose and insulinogenic effect of the food. Long term consumption of a diet with a relatively high GL is associated with an increased risk of type 2 diabetes and coronary heart disease (CHD) (Lui *et al.*, 2000). Thus according to Wylie-Rosett *et al.* (2004), the fact that a low-GL diet slows absorption and lessens hyperinsulinaemia, suggests that it would promote appropriate weight loss, improve cardiovascular health, and reduce diabetes.

The following example illustrates the GL concept. An example of a food with a high GI but low GL is pumpkin. The GI of pumpkin is 75. However, a serving size of 80 g is recommended which denotes 4 g available CHO resulting in a GL of only 3. Due to this concept it is thus unnecessary to exclude the above and many other fruits and vegetables with high GI values from the healthy diet (Foster-Powell *et al.*, 2002). Barclay *et al.* (2005) categorised foods with a $GL \leq 10$ as low-GL and ≥ 20 as high GL. Thus, by adding the glycaemic loads of individual foods together, the total glycaemic load of a complete meal or the whole diet can be calculated (Salmeron *et al.*, 1997a). Barclay *et al.* (2005) expressed his concern that the use of GL or glycaemic response in isolation may lead to the habitual consumption of lower-CHO diets.

2.3 CRITICISMS AND MISCONCEPTIONS OF THE GI

Foods with a low GI produce a lower peak in postprandial glucose and a lesser overall blood glucose increase during the first 2 hours after consumption compared with foods with a high GI. The principle is that a slower rate of CHO absorption from low-GI foods results in a lower rise in blood glucose. Controversy about the clinical utility of classifying foods according to their glycaemic responses by using the GI method has however been reported and critics further suggest that the GI concept adds further restriction to the dietary management of diseases (Coulston and Reaven, 1997; Daly *et al.*, 1997). In contradiction to the complaint that a low-GI diet is too complex for clinical

use, several studies involving self-selection of food by patients who found the diets “simple and practical” have been reported (Wylie-Rosett *et al.*, 2004).

Some of the main concerns were that published GI values did not always agree because of different methodologies used to determine the GIs of individual foods (Raben, 2002), and that differences between the GI values of different foods are lost once these foods are consumed in a mixed meal (Coulston *et al.*, 1987). Methodological variables can markedly affect the interpretation of the glycaemic responses and the GI values obtained (Wolever, 1990). According to Wolever *et al.* (1991), variables that affect the GI value include food-portion size, the method of blood sampling and subject characteristics.

Except for the Food and Agriculture Organization (FAO) guidelines (FAO/WHO, 1998) there is currently no internationally approved, detailed and standardised method for determination of the GI. In order to formulate a scientifically sound and standardised method of GI determination, the South African Department of Health convened a working group consisting of scientists and delegates from the industry. The main purpose of this process was to enable comparison of GI between foods and to provide health professionals with a scientifically sound dietary tool (Pieters and Jerling, 2005).

The GI values of many common foods are still unknown and different GI values for similar foods are often reported by different investigators. As is immediately apparent from examinations of GI tables (Foster-Powell *et al.*, 2002), values for the GI of foods can be rather broad. For example the published GI for boiled white rice varied from 45 to 112 (glucose = 100), and bananas ranged from 30 to 70, partially depending on their degree of ripeness. White durum-wheat semolina spaghetti varied from 46 to 65, depending on length of cooking time (Wylie-Rosett *et al.*, 2004). Venter and co-workers (2005) emphasized the fact that differences in GI values of similar foods could be due to inherent botanical differences from country to country, different testing methods, or the effects of random variation. Differences in testing methods include use of different types of blood samples (capillary whole blood or venous plasma) and different portions of foods (50 g of total carbohydrate rather than of glycaemic carbohydrate).

It is acknowledged by experts on the GI that macronutrient recommendations remain the primary concern in diabetes nutrition management (Jenkins, 1984; Perlstein *et al.*, 1997). Opponents to the GI concept admit that the concept of ‘simple’ and ‘complex’ CHO is not scientific and is outdated, but they are still against the general practical implementation of the GI (American Diabetes Association,

2001). The recent South African Food-Based Dietary Guidelines is in agreement with current recommendations for diabetes mellitus that advocates dietary variety and a diet high in CHO (with emphasis on increasing intake of cereals and grains) but low in fat content (Vorster *et al.*, 2001). Slabber (2005) therefore suggests that the GI concept should be used alongside and not in opposition to these guidelines.

Slabber (2005) reported that critics of the clinical utilisation of the GI argue that the use of technical terms will confuse clients' understanding of the GI concept. Pi-Sunyer (2002) questions how customers are to be informed about a food's method of preservation and processing as well as technical terms like retrogradation. Slabber (2005) clearly states that there is no need to use these terms when educating patients. Health professionals who successfully use the GI concept in patient education emphasise the fact that consumers should not be burdened with technical terms and they suggest that technical terms be avoided (Pawlak *et al.*, 2002; Pi-Sunyer, 2002; Katanas, 1999). Many food factors, such as the extent to which a starch is processed and gelatinised by home cooking or commercial preparation, may affect the rate of digestion and thus the GI of the starch. Slabber (2005) asked the question, "Why should we be more technical when advising consumers to ingest certain CHO in preference to others? Despite the fact that preservation methods may influence the omega-3 fatty acid content of fish, consumers are still advised to eat fish at least twice a week in order to increase their omega-3 fatty acid intake."

General agreement has been reached among most nutritionists and dieticians regarding the place of sugar in the diabetic diet. The GI of sucrose is relatively low at 68 ± 5 (mean of 10 studies using glucose as standard) (Forster-Powell *et al.*, 2002). The ingestion of 30 g sucrose per day does not compromise carbohydrate or lipid metabolism and these findings initiated the liberation of sugar intake in diabetic diets over the past decades (Slabber, 2005). The American Diabetes Association (2003) regarded the evidence that sucrose does not increase glycaemia to a greater extent than isocaloric amounts of starch as A-level evidence, but as reported by Slabber (2005), many health professionals still believe that sugar should be avoided in the diabetic diet. Brand-Miller and co-workers (Brand-Miller *et al.*, 1995) state that theoretically the addition of sucrose will lower the overall GI of the diet if it replaces wheat flour or high-GI foods. When the starch in a high-GI breakfast cereal was replaced with sucrose, Brand-Miller and Lobbzoo (1994) demonstrated a decrease in glucose and insulin responses. Thus, according to Slabber (2005) if sugar is used in the diabetic diet within the context of current dietary guidelines, it need not be an issue at all.

It is stated by opponents to the practical utility of the GI concept that differences in GIs between foods are lost once these foods are ingested in a mixed meal and they also argue that mixed meals contain fat that may greatly alter the GI of the meal (Coulston et al., 1987). Added fat had a negligible influence on the predicted glycaemic response in studies in which 8-24 g fat was fed in mixed meals containing 38-104 g carbohydrate. Over time large deviations in the dietary macronutrient profile will occur, but these differences will decrease as time goes by. Changes in the dietary GI are likely to be obscured only in those subjects with substantial differentiations in daily macronutrient intake, and in such individuals any meaningful attempt at dietary modification is also likely to be difficult (Jenkins et al., 2002). According to Willet et al. (2002), by using the GL, the total dietary GI of mixed diets can be calculated as a weighted average of the GI values of the individual foods with the weights corresponding to each food's CHO content. This may be very complex for the consumer, but if higher-GI foods are replaced with lower-GI alternatives in a meal, consumers do not have to be burdened by this technical task.

According to Slabber (2005), the expression of the GI as numerical figures that may adversely affect food choices is one of the major misuses of the GI concept. Clients may for example view all foods with a low GI as suitable and include low-GI foods with a high fat content, such as chocolate, freely. Clients may also avoid foods with high GIs that contain important nutrients and phytochemicals such as potatoes, enriched mealie meal porridge and carrots. Many health professionals unfortunately regard the numerical list of GI values as the primary factor in determining a food's suitability in dietary management (Perlstein et al., 1997). According to Slabber (2005) the actual GI figure or number is not the most important consideration, but rather that clients should be educated that the ranking (i.e. whether the food has a low, moderate or high GI) holds the real key to correctly applying the GI concept in dietary advice. Slabber (2005) also stated that providing clients with lists of numbers for GIs may be confusing and complicate dietary education and therefore a range of low-, medium- and high-GI foods should rather be provided, because it best describes the glycaemic response to foods and should therefore also be used by health professionals in client education.

Earlier on the American Diabetes Association (1998) and some health professionals (Beebe, 1999; Franz, 1999) were concerned about the practical utility of the GI, in particular the fact that a low-GI diet limits food choices and places another burden on individuals with diabetes. However, a recent large long-term prospective study in children with type 1 diabetes showed that those who were given flexible low-GI dietary advice did not lower dietary quality or food choices compared with children who received more traditional measured carbohydrate dietary advice (Gilbertson et al.,

2003). Since 1998 the American Diabetes Association (Sheard *et al.*, 2004) stated, however, that blood glucose level is determined by both the amount (grams) of CHO as well as the type of CHO in a food. Therefore, in order to achieve glycaemic control, recording total grams of CHO, whether by use of exchanges or CHO-counting, remains important, largely due to the fact that the total amount of CHO consumed is a strong predictor of glycaemic response.

Slabber (2005) emphasises that although a wider variety of low-GI products may be needed to implement a low-GI diet and suitable alternatives are not always available, health professionals can utilise the current range of food listed within low-, medium, and high-GI ranges as a valuable tool in client education. It is thus of great importance that the food industry regards the development of lower-GI starch substitutes as a challenge, especially in view of the current draft labelling legislation, which advocates the use of standardised methodology for the determination of the GI in CHO rich foods. According to Slabber (2005), some health professionals consider all low-GI foods as appropriate and all high-GI foods as unsuitable, which may well lead to *ad libitum* use of low-GI foods and exclusion of high-GI, thus limiting food choices and resulting in a deterioration of dietary quality.

Beebe (1999) states that Americans are eating low-fat foods, but in unlimited quantities. They are replacing fat with CHO, but ignoring total energy intake and this practice implies misuse of the GI concept. Because portion sizes remain of utmost importance, Slabber (2005) strongly emphasised that health professionals should strictly avoid suggesting to overweight clients and diabetic patients that low-GI CHO may be eaten in unlimited quantities without overt risk of increasing obesity and/or hyperglycaemia.

It is also premature to recommend the avoidance of high-GI foods to the general population. However, substituting certain CHO with 'better' choices will not discard any current dietary guidelines. It is thus, important for health professionals to emphasise that individuals should not overindulge on low-GI foods, that portion sizes are important, and that the GI of food is not the only factor determining whether the food should be included in the diet or not (Slabber, 2005).

2.4 METHODOLOGY USED TO DETERMINE THE GI

2.4.1 Subjects

2.4.1.1 Within- and between-subject variation

The glycaemic response to a particular food is subject to both within individuals and between individual variation (Pi-Sunyer, 2002; Brand-Miller *et al.*, 2003; Frost and Dornhorst, 2000; Wolever *et al.*, 1985). The variability of the glycaemic response for a given food for any one individual is similar to that seen for the oral glucose tolerance test (Wolever, 1990). Within-subject variation refers to the day to day variation of glycaemic response in the same subject, when consuming standard test meals under standardised conditions (Pieters and Jerling, 2005). Studies have shown that within-subject variation of healthy subjects to glucose varied from 19% (Nell, 2001) to 63% (Aginsky *et al.*, 2000), while a fairly consistent picture of fasting plasma glucose variability of 14-20% in type 2 diabetics was shown to be in agreement with those of similar studies (Venter *et al.*, 2003). According to Wolever *et al.* (1985) the mean within-subject variation of the glycaemic response after consumption of either glucose or white bread is 30% in type 1 diabetics.

Pieters and Jerling (2005) stated that it is of utmost importance that the glucose response of the standard food is measured correctly, since the GI is the individual's glucose response to a test food versus the individual's glucose response to a standard food. For this reason it is essential that there is no change in glucose homeostasis from the time the standard food is consumed until the test food is consumed. Some factors might influence glucose homeostasis such as exercise pattern (Dunstan *et al.*, 2002), weight change (Conceicao de Oliveira *et al.*, 2003; Harder *et al.*, 2004), presence of infection (Peach, 2001; Sougleri *et al.*, 2001), changes in alcohol consumption patterns (Meyer *et al.*, 2003), change in stress levels (Mizock, 1995), seasonal variation in glucose and insulin levels (Mavri *et al.*, 2001), use of certain medications, e.g. corticosteroids (Meticorten), oestrogens (Premarin), diuretics (Dyazide), nicotinic acid, beta-blockers (Inderal or Tenormin) and even aspirin (Pieters and Jerling, 2005). Three measurements of the standard food are thus essential for the accurate calculation of the GI because of the high within-subject variation of the glucose response (Pieters and Jerling, 2005). According to Wolever *et al.* (2002) the average of the three measurements of the standard food has been shown to reduce the variation of the mean GI values.

Differences in the physical and chemical characteristics of specific foods, as well as differences in methodology (e.g. type of blood sample, the experimental time period and the portion of food) may also be shown by variation in individual glucose response, thus, influencing the GI of a given food (Sheard *et al.*, 2004). Similar GI values can be obtained when methodology is standardised

(Wolever et al., 2003), although some foods continue to show wide variation in response secondary to botanical differences (Foster-Powell et al., 2002).

Variation between individual subjects in the glycaemic response to a food and in the GI of the same food also exists. According to Wolever (1990) the variability between individuals is larger than within individual subjects. Venter et al. (2003) stated that some studies confirmed this phenomenon (Wolever et al., 1985; Wolever et al., 1989), while other studies proved the opposite in finding greater within- than between-subject variation in both healthy subjects (Nell, 2001) as well as in type 2 diabetics (Kruger et al., in press). According to Venter et al. (2003) the latter findings have an important practical implication in GI determination for research or labelling purposes, as this proposes that, as long as the group is homogeneous, it would not be necessary to use the same subjects repeatedly and that larger groups of subjects could be used less often. Between-individual variation can be reduced to ~10%, if the glycaemic response is expressed as a percentage of an individual's response to a standardised food (i.e. 50 g white bread or glucose) (Jenkins et al., 1981; Jenkins et al., 1983; Wolever et al., 2003).

2.4.1.2 Type of subjects

Many subject characteristics may affect the glycaemic response to a given food and might contribute to variation including health status, type and treatment of diabetes, body mass index (BMI), age, gender and ethnicity (Jenkins et al., 1984; Venter et al., 2003) and will subsequently be discussed.

a) Health status

According to Pieters and Jerling (2005) consensus has not yet been reached on the issue of whether subjects for GI determination may include both normal and diabetic individuals. Although correlation exist between the GI of normal healthy individuals, type 1 and type 2 diabetics, the absolute values may differ significantly and for that reason the three types of subjects should not be combined into one test group for GI determination. Brouns et al. (2005) stated that routine testing is recommended in healthy human volunteers as variation of the values may differ in various groups, being highest in individuals suffering from type 1 diabetes. Another reason for not combining diabetics and healthy individuals is the fact that the calculation of the area under curve (AUC) for

diabetics is done over 3 hours, while for normal healthy individuals it is done over 2 hours (Venter et al., 2003).

The health status of subjects included should preferably be in agreement with those of the target population, (e.g. type 1 diabetics or athletes) if a specific food formula or feed is developed for a specific target population. Before subjects can be classified as either in good health or diabetic, their individual glucose tolerance should be determined. Healthy subjects should not take any drugs that may affect glucose tolerance. Type 2 diabetics' glycosylated haemoglobin (HbA_{1c}) should be measured and be within the acceptable range of 7-8% to ensure diabetic subjects are well controlled. Serum and urine creatinine should also be within normal ranges to ensure that the subjects have normal renal function. To decrease variability, type 2 diabetics should be treated with diet alone or diet and metformin rather than sulphonylureas (Venter et al., 2003).

b) Age

According to Venter et al. (2003) dietary changes and lower physical activity may affect glucose tolerance with increasing age, but no significant differences in the glycaemic responses between adults and children were found by Wolever and colleagues (1988).

c) Ethnicity

Data is lacking on the effect of ethnicity independent of background diet. Walker and Walker (1984) could not find significant differences in blood glucose response between different race groups. Summerson and co-workers (1992) have, however, shown race-related differences in the control of diabetes in adults. It might therefore, be advisable to use subjects from the same ethnic group only in studies on diabetic subjects (Venter et al., 2003).

d) Gender

Rasmussen and co-workers (1992) failed to show a significant influence of gender on glycaemic responses in middle-aged male and female type 2 diabetics.

e) **Body mass index**

Obese subjects may show altered glucose tolerance due to insulin resistance that is associated with abdominal obesity and the presence of obesity as a variable has not been studied adequately (Castillo *et al.*, 1994). Therefore, according to FAO/WHO (1998) in a non-diabetic study sample subjects in normal BMI range of 18.5-24.9 kg/m² should be included. Approximately 80% of type 2 diabetics have a history of obesity at the time of diagnosis or are currently obese (Marion and Franz, 2000, p.745). When determining the GI a reference BMI range of 20-35 kg/m² will, therefore, be more representative of the general population. To optimise results, the study population should be homogeneous with regard to age, weight, height and BMI (Venter *et al.*, 2003).

2.4.1.3 Number of subjects

Most GI studies have been done with five to ten subjects (Foster-Powell *et al.*, 2002). However, Nell (2001) indicated that if a 10% range for a GI of a food is sought with 80% confidence, between 24 and 90 subjects should be included in a study using venous plasma samples. Brouns *et al.* (2005) advises that the inclusion of ten subjects provides a reasonable degree of power and precision for most purposes of measuring GI, but that the number of subjects can be increased if the aim of the study is to detect small differences in GI or when greater precision is required. The GI Task Force (2002) recommends a minimum of 10-20 subjects to be recruited based on willingness to comply with the protocol, inclusion and exclusion criteria.

2.4.2 50 g carbohydrate portion

Food portion size has a major effect on the GI value because glycaemic responses are related to the CHO load. According to Pieters and Jerling (2005), not all CHO ingested contribute to the blood glucose response. Free sugars and starch are the main contributors to blood glucose while resistant starch (RS) and non-starch polysaccharides move through to the colon where they are either fermented to short-chain fatty acids (SCFAs) (mainly RS and soluble fibre) or excreted (mainly lignin and cellulose). Fructose and galactose are mainly converted to glucose only once they pass through the liver and are not immediately available as glucose after absorption. Thus, galactose and fructose

play a smaller part in the immediate glucose response. Not all ingested CHO should therefore be included in the 50 g portion.

Confusion might also be caused by terms like 'glycaemic' and 'available' CHO which are not synonymous. Available CHO also include resistant starch and soluble fibre, because they are available to the body, although not as glucose, but as SCFAs. Glycaemic CHO include only CHO that provide CHO for metabolism and is a summation of the analytical values of mono-, di-, and oligosaccharides, starch and glycogen but excludes fructo-oligosaccharides and other non-digestible oligosaccharides and resistant starch (FAO/WHO, 1998; Brand-Miller and Gilbertson, 2001). Pieters and Jerling (2005) support the proposal that in the determination of the 50 g portion only glycaemic CHO should be used, since this is the CHO fraction that elicits the blood glucose response. South African Food Composition Tables calculation of the 'CHO by difference' value was not directly measured and should not be used. The Englyst *et al.* (1999) method is an example of an analytical technique that can be used to determine different starch fractions in a product (e.g. free sugar glucose, rapidly available starch, slowly available starch and RS).

2.4.3 Standard food

According to the FAO/WHO (1998), either glucose or white bread can be used as the standard food. The DoH working group decided to use glucose as the standard food for labelling purposes since it was the chosen food used in an international inter-laboratory study where the aim was to evaluate the method recommended by FAO/WHO in order to determine the magnitude and source of variation in the GI values obtained by experienced investigators in different international centres (Pieters and Jerling, 2005).

Brouns *et al.* (2005) recommend that the GI be expressed relative to glucose =100. For practical purposes Brouns and co-workers (2005) feel that it is acceptable to use standard foods other than glucose, such as white bread, during the measurement of GI as long as they have been calibrated against glucose and the condition of preparation of this food is standardised. It is easy to standardise glucose whereas differences in locally produced white breads might add to analytical variation (Pieters and Jerling, 2005). According to Pieters and Jerling (2005) the variation in GI of locally produced white bread (one of the test foods) did not differ from the variation in GI of other

centrally produced test foods (instant mashed potato, white spaghetti and pot barley) and therefore might still be a viable option in the selection of a standard food.

According to Venter *et al.*, (2003) if glucose is used as standard it should be selected from the same batch and purchased in bulk. Fifty grams of glucose powder should be weighed in separate portions and dissolved in 200-250 ml water. Glucose solutions should be served at the same temperature. If white bread is used as standard food, each sample should provide 50 g available glycaemic CHO. All breads used should come from the same batch and supplier to avoid differences in the quality and quantity of CHO load. Because of the influence of the Maillard reaction on the availability of CHO from the bread crusts, all crusts must be removed. White bread ingested on different days as standard food should be frozen and thawed according to methods prescribed for test foods to ensure uniformity. This is because bread is not a consistent food and it may go stale, losing water when standing at usual indoor temperatures.

The mean area under the curve (AUC) of three trials of the standard (reference) food should be used to calculate the GI (FAO/WHO, 1998), because the mean of these three trials is more likely to be representative of a subject's true glycaemic response to the standard food than the result of a single trial (GI Task Force, 2002).

2.4.4 Pre-test meal

On the evening before testing all subjects should consume a standardised pre-test meal no later than 22h00. Proposed standardised methodology contains examples of such meals. The fundamental reason behind this standardised pre-test meal is to prevent constituents of the evening meal, before testing, from affecting the glycaemic response of the test meals (second meal effect) (Pieters and Jerling, 2005). It seems, however, that a standardised pre-evening test meal may not be essential. A recent study by Campbell *et al.* (2003) demonstrated no difference in the mean incremental area under the blood glucose curve of 13 subjects following either a standardised or non-standardised pre-evening test meal. A small amount of doubt does exist since the study needs to be verified and also seems to be somewhat underpowered (Pieters and Jerling, 2005).

2.4.5 Test foods

According to Venter *et al.* (2003), test foods should be given on separate days in random order and should provide 50 g of available glycaemic CHO. Test foods selected from the same batch should be purchased in bulk to ensure uniformity of shelf life and similarity of management during production, maturity and processing procedures. Cooked test foods should be prepared beforehand, frozen in portioned amounts in plastic bags or sealed containers at -18-30°C. Required food should be removed from the freezer on the night before the test session, thawed at room temperature and reheated if necessary in a microwave oven at precise times (Nel, 2001). Individual dry food portions are weighed into precise portions containing 50 g glycaemic CHO each by using a digital scale. Standardised equipment, cooking methods and utensils should be used to prepare cooked food products (Venter *et al.*, 2003).

2.4.6 Volume and type of drinks consumed with test meals

The volume and type of beverage consumed with a test meal may affect the blood glucose response. Young and Wolever (1998) studied the influence of 50, 250, 500, 750 or 1000 ml water or 250 ml coffee or tea and concluded that the volume and type of beverage consumed with a test meal influenced the pattern of blood glucose response but has no effect on the incremental area under the curve (AUC). They, however, suggested that a standardised volume of beverage must be established in order to design a definitive procedure for blood glucose testing.

Brouns and co-workers (2005) recommend supplying a standard amount of 250 ml water to the subject with the test portions and with the white bread portion if it is used as standard reference food. If glucose is the standard reference food, they recommended using a solution of 50 g glucose diluted into 250 ml water. They advised that fluid ingestion should take place within 5-10 min. Solids and semi-solids should be ingested within 10-20 min, depending on the type and taste of the food. The first blood sample should be taken exactly 15 min after the first bite of the food or first sip of the drink.

Venter *et al.* (2003) suggested that an accompaniment could be given with dry test foods, because otherwise they might be unpleasant to consume. However, this accompaniment should be low in energy, very low in CHO and kept the same for different foods compared (Truswell, 1992). Clear

statements should be made regarding the accompaniment used in the experimental protocol, especially for labelling purposes (Venter et al., 2003). The GI Task Force (2002) recommends that the standard reference food be taken with 300 ml of water in a 10-15 minute period.

2.4.7 Blood sampling

According to Pieters and Jerling (2005) consensus has been reached on the use of capillary blood for glucose determinations, provided that the capillary blood sample is obtained in a standardised manner. Venter et al. (2005) found that capillary blood samples had a lower coefficient of variation (CV) than venous samples and were on average higher than in venous plasma.

Brouns et al. (2005) as well as the GI Task Force (2002) recommend the following blood sampling schedule in subjects without diabetes: fasting (0) and at 15, 30, 45, 60, 90 and 120 min after starting to eat the test meal.

2.4.8 Calculation of the area under the curve (AUC)

Several possible ways exist for calculating the area under the blood glucose response curve (FAO/WHO, 1998; Wolever et al., 1991). Four different methods have been documented by different research groups to calculate the area under the curve (AUC), which includes 1) incremental AUC, 2) net incremental AUC, 3) incremental area with the lowest glucose values as baseline (AUC_{min}) and 4) total AUC (Venter et al., 2003).

The *incremental AUC* (IAUC) calculates the area under the curve (AUC) starting from the fasting value as baseline and therefore excluding any part of the curve that drops below the fasting value, and is a measure of the change of blood glucose from the fasting condition (Vorster et al., 1990; Pieters and Jerling, 2005). According to Wolever (2003; 1990), the GI is only based on the incremental area below the curve and above the fasting level and he considers IAUC as the only method to calculate the GI. This method was chosen by the DoH working group as the method of choice because it is used most often internationally and is also recommended by the FAO/WHO Expert Consultation Group on Carbohydrates in Human Nutrition (Pieters and Jerling, 2005).

The *net incremental AUC* was used by several researchers and is a variant of Wolever's method. In this method the area under the fasting blood glucose curve is subtracted from the area above the fasting blood glucose curve. A difference between the incremental and the net incremental areas will only be detected in cases where the postprandial blood glucose concentration drops below the fasting value (Venter et al., 2003).

Total AUC, on the other hand is a measure to calculate the total physiological response to a CHO load, and includes the area under the curve down to a blood glucose of zero and is a measure of the average blood glucose concentration during the period of test starting from the lowest glucose concentration in the response curve (including hypoglycaemic values, lower than the fasting value) (Vorster et al., 1990; Pieters and Jerling, 2005). The method has been criticized as being insensitive for detecting differences between the postprandial glycaemic responses of different meals (Venter et al., 2003).

The main source of error in determining the GI could be the method of calculating the AUC (Venter et al., 2003). According to Jerling et al. (2002) two main streams of approaches currently exist including the Wolever and Potchefstroom approach. In the Potchefstroom approach the incremental area with the lowest glucose value is used as baseline to calculate GIs since hypoglycaemia will not be reflected when the area below fasting level is ignored (Vorster et al., 1990; Venter et al., 2003). Nell (2001) recently found that the AUC_{min} method showed less variation than the incremental AUC method above the fasting level only and suggested that the AUC_{min} method is a more relevant physiological method to use in GI-calculations. According to Wolever et al. (1991) the GI is, however, based on the area under the blood glucose response curve above baseline. The overall equation simplifies to: $Area = (A+B+C+D/2)t + D^2t/2(D+\{E\})$, where A, B, C, D and E represent positive blood glucose increments; t is the time interval between blood samples. The Wolever approach has the longest history and is therefore used more often in scientific literature.

2.5 FACTORS INFLUENCING GI DETERMINATION

The glycaemic and insulin responses to food are influenced by either physiological individual factors or food factors (Vorster et al., 1990; Wolever et al., 1991). These factors influence the rate of absorption or digestion and, in turn, the glycaemic responses. It is thus, for these reasons that the GI range, rather than an absolute value, may be linked to each food as differences of 10 to 15 units

are within the error associated with the measurement of the GI (Wolever, 1991; Perlstein et al., 1997).

2.5.1 Non-food factors

Processes such as chewing and swallowing are included in non-food factors. Chewing leads to the reduction in food particle size which increases absorption rate as well as constituency of food such as bread, while pasta, on the other hand, retains its structure on swallowing which slows the absorption (Jenkins et al., 1988a). Gastric emptying is the major determinant of nutrient delivery to the small intestine and variation in the rate of gastric emptying accounts for 35% of the variance in peak blood concentration after ingestion of 75 g of oral glucose in both healthy as well as type 2 diabetic subjects (Horowitz et al., 1993). When white bread is used as standard food, GIs can range from less than 20% to approximately 120%. Differences in the rate of digestion or absorption of the CHO, as well as the digestive/fermentation fate of CHO in the small and large gut (to glucose vs. short-chain fatty acids) are usually the cause of these large differences in GI (Vorster et al., 2003).

2.5.2 Food factors

Some of the food factors that influence the GI include food form, particle size, cooking, processing and starch structure (Augustin et al., 2002). CHO with different physical forms, chemical structures, particle sizes and fibre content induce distinct plasma and glucose responses (Nell, 2001). Some of the factors that can influence the GI are summarized in table 2.1.

Table 2.1 Factors that influence the glycaemic index (Augustin *et al.*, 2002)

Factors that affect the GI	Factors that decrease the GI	Factors that increase the GI
<ul style="list-style-type: none"> ▪ Nature of starch ▪ Nature of monosaccharide components ▪ Viscous fibre ▪ Cooking/food processing ▪ Particle size ▪ Ripeness and food storage ▪ α-Amylase inhibitors ▪ Nutrient-starch interactions 	<ul style="list-style-type: none"> ↑Amylose/amylopectin Fructose Galactose ↑Guar ↑β-glucan Parboiling Cold extrusion Large particles Unripeness Cooling ↑Lectins ↑Phytates ↑Protein ↑Fat 	<ul style="list-style-type: none"> ↓Amylose/amylopectin Glucose ↓Guar ↓β-glucan Extruding Flaking Popping Grinding (small particles) Ripeness ↓Lectins ↓Phytates ↓Protein ↓Fat

2.5.2.1 Carbohydrates

Historically 'complex' CHO has been thought to be beneficial in slowing the glycaemic response. Various diabetes associations advocated absolute elimination of sucrose as well as limited intake of 'simple' CHO (Wolever and Brand-Miller, 1995). Jenkins *et al.* (1981) proved this concept wrong by finding that sucrose elicits a lower glycaemic response than glucose, whole meal bread, muesli and many other starch-containing foods.

a) Nature of the monosaccharide

Low GI foods are not the same as foods based on high complex CHO and fibre, nor are high GI foods those based on simple sugars. In foods that produce the highest GI the starch is fully gelatinised and can be rapidly digested and absorbed. Sugary foods often cause lower levels of glycaemia per g of CHO than the common starchy staples of western diets, because up to half of the weight of CHO is fructose, a sugar that has little effect on glycaemia and that produces a lower

glycaemic response than sucrose (Brand-Miller and Foster-Powell, 1999; Wolever and Brand-Miller, 1995).

Vorster et al. (1987) proved that moderated amounts of sucrose can be added to low GI foods and can improve the palatability of these low GI foods without detrimental effects on the glycaemic response. The GI of sucrose is relatively low at 68 ± 5 (mean of 10 studies using glucose as standard) (Forster-Powell et al., 2002). The ingestion of 30 g sucrose per day does not compromise CHO or lipid metabolism and these findings initiated the liberation of sugar intake in diabetic diets over the past decades (Slabber, 2005).

Brand-Miller and co-workers (Brand-Miller et al., 1995) state that theoretically the addition of sucrose will lower the overall GI of the diet if it replaces wheat flour or high-GI foods. In practice, Brand-Miller and Lobbezoo (1994) demonstrated a decrease in glucose and insulin responses when the starch in a high-GI breakfast cereal was replaced with sucrose. Thus, according to Slabber (2005), if sugar is used in the diabetic diet within the context of current dietary guidelines, it need not be an issue at all.

Galactose is actively absorbed in the small intestine and is converted into glucose in the liver, while very little appears in the blood after oral or intravenous galactose. The glycaemic response of galactose is, however, much lower in the presence of glucose, as both of these CHO compete for active transport (Wolever and Brand-Miller, 1995).

Another important factor affecting the glycaemic response is that of the different CHO fractions. Englyst et al. (1999) proposed a chemically based classification which divides dietary CHO into sugars, starch fractions and non-starch polysaccharides (NSP) and which groups the latter starch into rapidly available glucose (RAG) and slowly available glucose (SAG). This chemically based classification takes into account the likely site, rate and extent of digestion.

b) Nature of the starch (chemical structure)

The type of starch (amylose content) present in a food influences the glycaemic response. Amylose and amylopectin are both polymers of glucose which occur in linear and branched form respectively. Studies have shown that the open, branched structure of amylopectin starch makes it easier to digest than the (linear) amylose starch (Wolever, 1990). A higher ratio of amylose to amylopectin

produces a slower rate of digestion due to the extensive hydrogen bonding of amylose (Perlstein *et al.*, 1997).

Higher amylose content would lead to induction of a decreased postprandial plasma glucose- and insulin response compared to amylopectin (Behall *et al.*, 1989; Byrnes *et al.*, 1995; Granfeldt *et al.*, 1994). Additionally, a high-amylose diet also resulted in significantly lower fasting triglycerides and cholesterol levels (Behall *et al.*, 1995). According to Granfeldt *et al.* (1994) the proposed mechanism for lowered metabolic responses in the presence of high amylose starch was probably due to a decreased rate of amylolysis. Amylose also has the tendency to recrystallize or to interact with lipids.

2.5.2.2 Dietary fibre and resistant starch

Various epidemiological studies have shown that ingestion of high-fibre foods reduces the risk of type 2 diabetes and CHD, and therefore, it was recommended by various diabetic associations that diets contain fibre-rich food (Wolever, 1990). Complex networks of fibre render the food particle less accessible for absorption. Non-digestible complex CHO are commonly known as dietary fibre, although the correct terminology is NSPs (Englyst *et al.*, 1987). According to ADA (2002), NSP are divided into soluble and insoluble fibre. Various mechanisms are involved in the effect of fibres on glycaemic response that depends on the structure of the food (mostly the integrity of the cell walls in non-fractionated foods) and reduced accessibility of α -amylase to its substrate.

Pulses, which result in the lowest GIs, also have the most resistant cell walls (Jenkins *et al.*, 1980b). β -glucans (especially uncooked), found in oats and barley as well as gums, especially guar gums may exhibit a reduced glycaemic response. In gums the reduced glycaemic response is due to their high viscosity (Guillon and Champ, 2000; Liljeberg *et al.*, 1996b). Liljeberg *et al.* (1996b) suggested that enrichment of cereal products with a genotype containing high β -glucan content would be favourable and acceptable to enhance fibre intake. Léclere *et al.* (1994) found that guar gum reduced the rate of starch degradation by pancreatic amylase and slowed gastric emptying.

Arabinoxylan (AX), of which wheat grain is a rich source, is another major component of dietary fibre. Wheat bran contains 64-69% AX and 15-31% cellulose, whereas NSPs in wheat endosperm are 88% AX. The physiological effect of AX is relatively unknown, but Lu and co-workers (2000)

found that the addition of as little as 6 g AX-rich fibre to bread in a breakfast meal significantly lowered postprandial glucose and insulin responses in healthy subjects. The precise mechanism by which AX-rich fibre flattens the postprandial glucose response is not yet known, but it is thought that because AX is a soluble fibre, it is likely that its effect is exerted similarly to other such fibres. Further research is required to determine whether AX-rich fibre will be of value to people with type 2 diabetes (Lu et al., 2000). Plantago Psyllium mucilage and α -glucosidase inhibitor (acarbose) decreased the GI of bread, the effect being greater with acarbose (Munari et al., 1998).

Insoluble NSP have little effect on gastric emptying and no effect on glucose absorption, therefore high fibre diets are not synonymous with low GI foods (Jenkins et al., 1983). Resistant starch (RS) is defined as "the fraction of starch that passes undigested to the large bowel" (Englyst et al., 1987). RS produced a significant lowering of postprandial plasma glucose (Raben et al., 1994). Venter and co-workers (1990) found that RS in cooled maize porridge resulted in a significantly smaller response in blood glucose compared to that of hot or reheated porridge. Potatoes and other moist-heated starchy foods are incompletely digested when cooled because of retrogradation of the starch (amylose) during cooking. The digestion decreases from 97% to 88% after cooling off these foods (Wolever, 1990; Englyst and Hudson, 2000, p.72). Although further studies are needed to clarify the effect of RS in mixed meals, it is in essence still advisable to recommend that diabetics ingest 25-30 g of diverse types of fibre sources daily as fibre has many other health benefits (Guillon and Champ, 2000).

2.5.2.3 Protein and fat

Fat and protein may modify the glycaemic response to a CHO food by slowing gastric emptying, due to the delay in CHO absorption (Welch et al., 1987; Franz, 1997) and increasing insulin secretion, respectively (Nuttall et al., 1984; Gannon et al., 1988). Fat is also known to reduce jejunal motility and postprandial flow rates in the intestine, and hence decreases the glycaemic response (Perlstein et al., 1997). High fat intakes appear to result in insulin resistance leading to increased glucose levels, while the ingestion of modest amounts of fat has minimal effect (Franz, 1997). According to Perlstein and co-workers (1997) protein may also increase the osmolarity of stomach content, thereby reducing the rate of gastric emptying.

Several factors may account for the influence of dietary fat on glucose and insulin responses, such as differences in gastric emptying, dietary fatty acids which may interact with food digestion by modulating digestive enzyme activities, as well as the type of fat (Wolever, 1990; Armand *et al.*, 1995; Joannic *et al.*, 1997). According to Joannic and co-workers (1997), the degree of saturation of a fat plays a conclusive role in influencing the metabolic response, and the greater the degree of unsaturation (e.g. polyunsaturated and monounsaturated), the more profound the insulin secretion, which is unfavourable. Whether the effect persists when the fat content is lower is debatable.

It has been found that neither fat nor protein in the amounts found in most foods (with the exception of peanuts and most nuts) significantly alter the glycaemic response (Wolever *et al.*, 1994). Protein levels of 30-50 g and fat levels of 50 g per 50 g of available CHO may decrease the GI (Wolever *et al.*, 1994; Nuttall *et al.*, 1984).

2.5.2.4 Food processing

The gross physical form of food, the susceptibility of starch granules to enzymatic hydrolysis and the susceptibility of starch granules to retrogradation after cooking have a major effect on the rate and extent of starch digestion and absorption in the small intestine. The way food is processed and prepared in the factory and/or at home is thus of great importance. Starch contained within discrete structures such as whole grains and seeds, is physically inaccessible to pancreatic amylase. Crushing, chopping and milling all increase the accessibility of the starch, i.e. the rate of digestion is influenced by the final particle size. Physical inaccessibility may cause the rate of starch hydrolysis to be so slow that some starch enters the large intestine or in extreme cases is excreted in the faeces (Englyst and Hudson, 2000, p.71).

Various methods of processing such as milling, dry heating, extrusion, cooking and puffing, flaking and rolling lead to structural changes in the food particle and disrupt the cellular architecture and fibrous structure rendering a product with a higher GI, which results in faster digestion and absorption and higher blood glucose and insulin responses (Brand *et al.*, 1985; Holt and Brand-Miller, 1994; Wolever, 1990). Extrusion, flaking, grinding, canning, storing and cooking of CHO-containing foods can affect the particle size and the integrity of the starch granules (Jenkins *et al.*, 1988a) and plant cell walls (Ellis *et al.*, 1991), making the CHO portion more accessible to digestive

enzymes (Wolever, 1990; Collins *et al.*, 1981). It is thus of utmost importance to maintain botanical structures to induce lowered glycaemic and insulin responses.

It is hypothesised that the high pressure used in the canning process could alter the physical nature of the starch and antinutrient content. Canning can increase the GI of dried beans by 17 units. The physical form of food also appears to influence the GI. Higher proportion of whole intact grain decreases the glycaemic response of food. The disruption to the grain increases the availability for enzymatic digestion and starch gelatinisation and hence elevates the GI (Perlstein *et al.*, 1997).

Cooking facilitates the hydrolysis of starch through gelatinisation and dispersion of the starch granules. Gelatinisation of starch granules is an obvious process whereby enzymes have greater opportunity to degrade the starch, leading to rapid digestion and absorption. Foods eaten raw retain their starch as granules, which show varying degrees of resistance to digestion and raw cereal starch is digested slowly within the small intestine, resulting in a modest glycaemic response. Retrogradation also slows digestion, and retrograded starch (mainly amylose) from processed cereal and potato products has been shown to pass through the small intestine (Englyst and Hudson, 2000, p.71).

Baking flour into shortbread, which involves cooking in the presence of very little water results in limited disruption of the granular structure and provides a product that also digests slowly. Baking flour into bread is, however, a process that requires a long cooking time in the presence of water, which results in extensive gelatinisation of the starch granules, leading to rapidly digestible starch granules as well as a rapidly digestible product (Englyst and Hudson, 2000, p.72).

2.5.2.5 Anti-nutrients

Anti-nutrients are food components which, in large amounts reduce the bioavailability of nutrients in food and which can impair growth in experimental animals. These anti-nutrients are present in many foods, especially legumes. The GI of these foods is closer related to their enzyme inhibitors, lectin, phytate (can inhibit amylase) and polyphenol (tannin) content, which also affect blood glucose response (Wolever, 1990; Thompson, 1988). Acarbose, a stable α -glucosidase inhibitor of bacterial origin delays the digestion and intestinal absorption of sucrose and starches (Munari *et al.*, 1998).

2.5.2.6 Organic acids

Organic acids and salts in question include lactic acid, tartaric acid and sodium propionate as well as sodium acetate and acetic acid (found in vinegar). Sourdough fermentation with the addition of lactic acid (organic acid) and sodium propionate (salt) to bread resulted in a decreased postprandial blood glucose and insulin rise as well as a possible delayed gastric emptying as a result of above organic acid and salt (Liljeberg and Björck, 1996a). The addition of tartaric acid to sorghum porridge decreased the GI by 43% (Mbhenyane, 1997).

According to Brighenti et al. (1995), acetate produced by colonic fermentation could be a mechanism by which fermented dietary fibre decreases postprandial glycaemia. Sodium acetate and acetic acid from vinegar produced the same effect as the above acids and salts, but sodium acetate has more profound effects. Brighenti and co-workers (1995) concluded that the mechanism by which vinegar influences glycaemic response to a mixed meal is related to acidity and not to gastric emptying. Traditional eating habits, such as the use of vinegar for the preservation of vegetables as well as in salad dressings, may decrease glycaemic response of meals consisting of CHO and fat.

2.5.2.7 Ripening and food storage

The composition of food changes as they ripen, and this makes a difference to their GI. The starch content of unripened banana is 37%, which decreases to 3% when ripe. Only 10% of this starch is then digested by the human gastrointestinal tract. These findings and others concerning ripening, food storage and various cultivars, suggest that there are many factors that cannot be controlled (Wolever, 1990).

2.5.2.8 Other factors

a) The influence of mixed meals on the GI

As already discussed, opponents to the practical utility of the GI concept state that differences in GIs between foods are lost once these foods are ingested in a mixed meal and they also argue that mixed meals contain fat that may greatly alter the GI of the meal (Coulston et al., 1987). Added

fat had a negligible influence on the predicted glycaemic response, in studies in which 8-24 g fat was fed in mixed meals containing 38-104 g carbohydrate. Over time large deviations in the dietary macronutrient profile will occur, but these differences will decrease as time goes by. Changes in the dietary GI are likely to be obscured only in those subjects with substantial differentiations in daily macronutrient intake, and in such individuals any meaningful attempt at dietary modification is also likely to be difficult (Jenkins et al., 2002).

Collier et al. (1986) also found that the relative glycaemic effect of mixed meals can be predicted from the GI of their carbohydrate components. Coulston et al. (1984a) stated that the GI concept remains discriminating in the context of a mixed meal in type 2 diabetics, which validates the use of the GI for choosing foods even in mixed meals and that the insulin response does not bring greater discrimination between CHO foods, but remains of interest in physiological studies. According to Willet, Manson and Lui (2002) by using the GL, the total dietary GI of mixed diets can be calculated as a weighted average of the GI values of the individual foods with the weights corresponding to each food's CHO content.

b) Second meal effect

Rapid absorption of CHO results in a large rise in blood glucose and insulin. The large insulin response causes peripheral glucose utilisation to increase to such extent that absorption from the gut cannot keep up so that the blood glucose level undershoots the baseline. This causes a counterregulatory response, with a rise in relative insulin resistance. On the other hand when CHO absorption is slow and prolonged, there is a less rapid rise of blood glucose, a smaller insulin response, and less of a tendency for the blood glucose to undershoot (Wolever, 1990). Thus, low GI foods as part of a meal can improve the CHO tolerance to subsequent meals.

This effect has been shown to occur between breakfast and lunch and between dinner and breakfast. A study by Wolever and colleagues (1988) showed that low GI CHO foods eaten at an evening meal reduced the acute post-prandial blood glucose response to the evening meal and to the subsequent standard breakfast. The blood glucose response to the lunch after a low-GI breakfast was significantly less than that of the lunch after the high-GI breakfast. The effect was mimicked by slowly nibbling the same high-GI breakfast over the entire 4 h period. However, when only one quarter of the high-GI breakfast was taken, the glycaemic response to lunch was markedly

impaired (Wolever, 1990; Franz, 1999). This aspect was also studied by Wolever and Bolognesi (1996b) in which they concluded that lunch time responses were influenced by many factors, such as the nature and composition of the previous meal (second meal effect).

2.6 HEALTH BENEFITS OF LOW-GI DIETS

Several studies have shown that eating a low GI diet has health benefits. Evidence from prospective studies shows that low GI-diets are associated with reduced risk of diabetes (especially type 2 diabetes) (Hodge *et al.*, 2004; Frost *et al.*, 1998; Salmerón *et al.*, 1997a; Salmerón *et al.*, 1997b), cardiovascular disease (Liu *et al.*, 2000), cancer (Augustin *et al.*, 2001; Augustin *et al.*, 2002; Augustin *et al.*, 2003a; Augustin *et al.*, 2003b; Augustin *et al.*, 2004a; Augustin *et al.*, 2004b; Augustin *et al.*, 2004c), and the metabolic syndrome (McKeown *et al.*, 2004).

2.6.1 Diabetes mellitus

The long-term effects of the GI on the development of type 2 diabetes were investigated in the Nurses' Health Study (Salmeron *et al.*, 1997a), the Health Professionals Study (Salmeron *et al.*, 1997b) and the Iowa Women's Health Study (Meyer *et al.*, 2000). Salmeron and colleagues (1997a, b) found a positive association between GI and the development of type 2 diabetes in both women and men after adjustment for age, body mass index (BMI), smoking, physical activity, family history of diabetes, alcohol and cereal fibre intake, as well as total energy intake. Comparing the highest with the lowest GI quintile of the diet, the relative risk (RR) of diabetes in women and men was 1.37 (95% confidence interval (CI):1.09, 1.71, p trend =0.05) and 1.37 (95% (CI):1.02, 1.83, p trend =0.03) respectively. However, no association between GI and the risk of developing diabetes was reported in the Iowa Women's Health Study.

The body responds by secreting insulin after the intake of high-GI foods. Nutrient absorption from the gastrointestinal tract declines within 2-4 hours after a high-GI meal and the high circulating insulin levels result in a reactive hypoglycaemic situation. Constant hypersecretion of insulin after intake of high-GI-meals could lead to pancreatic beta-cell dysfunction, resulting in insulin resistance (Ludwig, 2002; Wolever, 2000). According to Ludwig (2002), various longitudinal studies found that the risk for diabetes was higher among individuals in the highest quintile of GI than in those in the lowest quintile.

A number of long-term implications exist when altering the rate of breakdown and absorption, or GI of dietary CHO (Venter *et al.*, 2003). According to Augustin *et al.* (2002) the link between high-GI and high-GL diets and diabetes may relate to glucose peaks and increased insulin demand. High-GI foods lead to rapid rises in blood glucose and insulin levels. Hyperinsulinaemia, in turn, may down regulate insulin receptors and therefore reduce insulin efficiency, resulting in insulin resistance, which may act in a vicious circle by increasing blood glucose concentrations and insulin secretion. Insulin resistance is a risk factor for type 2 diabetes.

Low-GI diets tend to delay glucose absorption thereby resulting in reduced peak insulin concentrations and overall insulin demand and several studies have found improvements in glycaemic control with low-GI diets (Augustin *et al.*, 2002). In other studies, low-GI diets reduced blood glucose levels and urinary C-peptide output, as a measure of insulin secretion in healthy subjects (Burke *et al.*, 1982; Jenkins *et al.*, 1987b). Low-GI diets also improved glycaemic control in type 1 diabetic patients in the cross-sectional EURODIAB Complication Study, as indicated by reductions in glycosylated proteins (glycosylated haemoglobin (HbA_{1c}) concentrations). Compared with the highest GI quartile (GI 89), HbA_{1c} concentrations in the lowest quartile (GI 75) were 11% lower in patients from southern European centres and 6% in patients from the rest of the European centres (Buyken *et al.*, 2001). The Framingham cohort furthermore showed a strong positive association between prevalence of CHD and increased HbA_{1c} concentrations, suggesting the importance of hyperglycaemia in the development of CHD (Singer *et al.*, 1992).

A number of investigators on meal frequency also supported the health benefits of a low-GI diet as a model for a reduced rate of CHO absorption. In both diabetic and non-diabetic subjects, increasing meal frequency in isocaloric diets has been shown to reduce postprandial glucose rise (Jenkins *et al.*, 1992; Bertelsen *et al.*, 1993; Jones *et al.*, 1993), daily insulin levels (Jenkins *et al.*, 1992; Bertelsen *et al.*, 1993; Jones *et al.*, 1993), and 24 hour urinary C-peptide output (Jenkins *et al.*, 1989, 1992). A review by Opperman and co-worker's (2005) presented convincing evidence to recommend the use of the GI as a scientifically based tool when choosing CHO-containing foods to improve overall metabolic control of diabetes.

According to Augustin *et al.* (2002), two main mechanisms of action could be involved in low-GI diets, namely:

- Free fatty acid (FFA) levels: Rapidly absorbed CHO stimulate a large insulin rise, followed by a rapid blood glucose fall, often below baseline values. This could result in counter-

regulatory response with the release of FFA, creating an insulin resistant environment and reduced glucose tolerance. Ingestion of slow release CHO food (e.g. uncooked cornstarch) at bedtime was shown to produce substantial suppression of nocturnal FFA-levels and postprandial improvements in breakfast glucose levels, possibly due to reduced nocturnal lipolysis. Nocturnal hypoglycaemia type 1 diabetics can be prevented by the ingestion of slow-release CHO, taken in the evening.

- Oxidative stress (OS): OS is defined as a disturbance in the balance between free radical production and antioxidant capacity and it may play a role in the micro- and macro-angiopathic complications of diabetes. A direct link has been found between post-prandial glycaemia and the induction of OS that can be reversed by antioxidants. Possible mechanism of action of low-GI diets include reduction of (a) glucose toxicity (i.e. the effect of high glucose levels in depressing pancreatic function through free radical damage of β cells) and (b) glycosylation of proteins and key enzymes responsible for metabolic processes.

According to Rizkalla, Bellisle and Slama (2002) a 10% fall in the GI of a diet could result in a 30% increase in insulin sensitivity. Increased insulin sensitivity and reduced hepatic gluconeogenesis following low-GI diets, could all contribute to improved glucose control.

2.6.2 Coronary heart disease

Epidemiological evidence suggests that low-GI diets may decrease the risk of CHD independently and as part of a healthy lifestyle (Lui *et al.*, 2000). According to Augustin *et al.* (2002), possible beneficial effects of a low-GI diet in the prevention of CHD may be explained by improvements in blood lipid profiles, insulin levels, thrombotic factors and endothelial function. Low-GI foods are associated with reduced hepatic gluconeogenesis, suppression of FFA release and therefore increases in the high-density lipoprotein cholesterol (HDL) fraction, demonstrating an inverse association between serum HDL and dietary GI (Rizkalla *et al.*, 2002; Wolever, 2000).

Proposed mechanisms for lipid modulation by low-GI foods versus high-GI foods may include lower insulin-stimulated HMG-CoA reductase activity, the rate limiting enzyme in cholesterol synthesis, due to a reduced rate of CHO absorption; impaired bile acid and cholesterol reabsorption from the ileum due to the typically high fibre content of low-GI foods; inhibition of hepatic cholesterol synthesis by the short chain fatty acid (SCFA) propionate, a by-product of colonic fermentation; and reduced

inflammatory response (Augustin et al., 2002). Counterregulatory hormone secretion is triggered by resulting hypoglycaemia within 4-6 hours after ingestion of a high-GI meal. This results among other things in glucagon release which stimulates gluconeogenesis leading to elevated levels of FFA in the circulation. Increased hepatic uptake of FFA results in increased hepatic secretion of very-low-density lipoproteins (VLDL) triglyceride secretion. High levels of VLDL production result in reduced HDLC levels and an increase in the formation of low-density lipoproteins cholesterol (LDLC) fractions (Blaauw, 2003).

Regulating insulin levels in diabetics as well as healthy subjects may be of utmost importance as hyperinsulinaemia has been directly associated with CHD in previously healthy populations. When looking at thrombotic factors some evidence suggests that hyperglycaemia and hyperinsulinaemia may lead to impaired fibrinolysis and thrombosis, thereby increasing the risk for CHD. In relation with endothelial function some evidence exists for a role of hyperglycaemia in endothelial cell dysfunction possibly through increased generation of oxygen free radicals, especially in diabetics (Augustin et al., 2002).

According to Opperman and colleagues (2005), a low HDLC concentration is a strong independent predictor of CHD and has several causes, many which are associated with insulin resistance, elevated triacylglycerols (TGs), overweight and obesity, physical inactivity and type 2 diabetes. Opperman et al (2005) reviewed several studies, of which some showed improvements in HDLC concentrations (Ford and Liu, 2001; Buyken et al., 2001), while others found a significant negative relationship between dietary GI and HDLC concentrations (Frost et al., 1999). One other study showed no association between GI and HDLC concentrations (Van Dam et al., 2000).

2.6.3 Carbohydrate metabolism

Fructosamine is measured as a useful short-term (2-week) index of glycaemic control. Glycosylated albumin is the main constituent of fructosamine and has a half-life of only 12 days. It seems that the longer low-GI diets are followed, the larger the observed decreases in fructosamine concentrations (Opperman et al., 2005). According to Jones et al. (1983), 4-6 weeks are necessary for maximum changes in fructosamine to occur and a more profound decrease was documented in diabetic than healthy subjects.

Glycosylated haemoglobin (HbA_{1c}) is a longer-term marker of CHO metabolism than fructosamine and provides an index of the average blood glucose concentration over a half-life of the haemoglobin molecule (approximately 6 weeks). One may conclude from HbA_{1c} that low-GI diets beneficially influence long-term glycaemic control. Incorporation of more than one type of low-GI food into the diet may be needed to achieve measurable long-term improvements in glycaemic control (Opperman *et al.*, 2005).

A greater incidence of long-term macrovascular complications has been associated with poor glucose control in both type 1 and type 2 diabetic patients (Opperman *et al.*, 2005). How the GI improves markers of CHO metabolism and prevents the onset of type 2 diabetes is not yet clear, but several mechanisms have been proposed. Firstly, high-GI diets have been associated with high postprandial blood glucose concentrations and increased insulin demands (Ludwig, 2002; Willet *et al.*, 2002). Insulin resistance, which reduces insulin sensitivity, is primarily caused by hyperinsulinaemia. Additional habitual consumption of high-GI meals over the long term initiates a cycle of hyperinsulinaemia and insulin resistance leading to a loss of pancreatic β -cell function (Ludwig, 2002) that can result in glucose intolerance and an irreversible state of diabetes (Willet *et al.*, 2002). Hyperglycaemia also has deleterious effects on counterregulatory hormone secretion, as discussed above it increases late postprandial serum FFA concentrations and leads to the occurrence of oxidative stress. On the other hand, low-GI diets tend to delay glucose absorption, therefore resulting in reduced peak insulin concentrations and overall insulin demand (Augustin *et al.*, 2002).

According to a review by Opperman and colleagues (2005), significant improvements in carbohydrate metabolism could be expected in fructosamine of -0.1 mmol/L with a GI reduction of 24 ± 9 GI units, and HbA_{1c} will improve by -0.27% with a reduction of 21 ± 7 GI units.

2.6.4 Lipid metabolism

When looking at lipid metabolism, low-GI diets significantly decrease LDLC concentrations by -0.24 mmol/L with a GI reduction of 21 ± 10 units and total cholesterol (TC) by -0.33 mmol/L with a GI reduction of 20 ± 9 units (Opperman *et al.*, 2005).

In studies reviewed by Opperman *et al.* (2005), low-GI diets showed a statistically significant improvement in TC concentrations, while non-significant improvements were observed in LDLC. No

significant change was found in triacylglycerols and HDLC with low-GI diets, although an inverse relationship was found in epidemiological studies between the GI and HDLC with lower GI diets (Buyken *et al.* 2001; Ford and Lui, 2001; Frost *et al.*, 1999). Low-GI diets have favourable effects on LDLC concentrations of type 2 diabetic subjects (Opperman *et al.*, 2005).

The mechanism by which low-GI diets may reduce TC concentrations remains unclear, but as already discussed above it may include a decrease in insulin stimulated HMG-CoA reductase activity, impaired bile acid and cholesterol reabsorption and/or inhibition of hepatic cholesterol synthesis by SCFAs such as propionate (Opperman *et al.*, 2005).

A possible mechanism by which low-GI diets contributes to lower LDLC concentrations may be that insulin resistance may occur with consumption of a high-GI diet due to the direct effects of hyperglycaemia (Ludwig, 2002). Insulin resistance impairs normal suppression FFA release from adipose tissue in the postprandial state. According to Timar and co-workers (2000), increased FFA released from abdominal adipose tissue delivered to the liver, offers an efficient substrate for enhanced synthesis of TG and VLDL, resulting in elevated cholesterol concentrations as explained previously.

The LDL-receptor activity is reduced with the prevalence of insulin resistance as seen in type 2 diabetics, which results in less LDLC removal from the blood, therefore contributing to higher LDLC concentrations (Garg, 1996). Barakat *et al.* (1996) explain that reduced receptor activity may be attributed to glycosylation of the low-density lipoprotein (LDL) particle in the presence of hyperglycaemia. Glycosylated LDLC cannot bind as efficiently as non-glycosylated LDLC because of impairments in the binding of the LDL particles to LDL receptors and glycosylated LDL particles will therefore remain longer in circulation.

2.6.5 Obesity

Obesity is a risk factor for several chronic diseases including type 2 diabetes, CHD and some types of cancers (Augustin *et al.*, 2002). According to Brand-Miller and colleagues (2002) weight loss can be achieved by any means of energy restriction, but current dietary guidelines have not prevented weight regain or population-level increase in obesity and overweight. Marked increase in postprandial hyperglycaemia and hyperinsulinaemia may lead to the belief that many high-CHO, low-

fat diets may be counterproductive to weight control. Many high-CHO foods common to Western diets produce a high glycaemic response (high-GI foods), promoting postprandial CHO oxidation at the expense of fat oxidation, thus altering fuel separation in a way that may lead to body fat gain. According to Ludwig (2002), hyperinsulinaemia and hypoglycaemia may stimulate the consumption of high-GI foods, which have a low satiety level, and which increase hunger as well as stimulate eating. The resultant cycles of hypoglycaemia and hyperphagia contribute to the development of obesity.

Diets based on low-fat foods that produce a low glycaemic response (low-GI foods) may in contrast enhance weight control by promoting satiety, minimizing postprandial insulin secretion, and by maintaining insulin sensitivity (Brand-Miller *et al.*, 2002). When low glycaemic CHO are incorporated into an energy restricted diet, there is a greater fall in insulin resistance that cannot be accounted for by weight-loss alone (Slabber *et al.*, 1994). According to Agus *et al.* (2000), low- glycaemic index diets also influence body weight and resting energy expenditure independently of energy intake in young moderately overweight subjects. Several studies (Roberts, 2000; Guliford *et al.*, 1989) have shown that high-glycaemic carbohydrate also leads to hunger and carbohydrate craving. Ludwig (2000) has recorded 15 studies in the adult literature demonstrating increased satiety, delayed return of hunger, and decreased food intake after ingestion of low-GI compared with high-GI foods (Slyper, 2004). Low-GI diets may thus also play an extensive role in weight loss.

2.6.6 Cancer

At present the amount of evidence on the relationship between GI and cancer is scarce. Some epidemiological studies found direct associations for colorectal and breast cancer. Several lines of evidence point to a possible role of the GI in the development of cancer (Augustin *et al.*, 2002). McKeown- Eyssen (1994) and Giovannucci (1995) hypothesized that hyperinsulinaemia/insulin resistance may promote colorectal cancer and possibly other types of cancers related to Western lifestyle (Bruning *et al.*, 1992). High intakes of energy and refined CHO, low intake of vegetables, fruit and dietary fibre, lack of physical activity, obesity, diabetes, hyperinsulinaemia and high levels of insulin-like growth factors (IGFs) have been implicated in the aetiology of various types of cancer (Giovannucci, 1995).

The more refined the CHO in the habitual diet, the greater the risk for colorectal cancer (Franceschi *et al.*, 2001). Hyperinsulinaemia/ insulin resistance has been hypothesized to play a role in development of cancer of the breast (Kaaks, 1996). Insulin may act as a mitogen in breast cells through the insulin receptor in a dose-dependent manner (Del Giudice *et al.*, 1998; Bruning *et al.*, 1992). Obesity, particularly central obesity, is one of the major risk factors for insulin resistance and hyperinsulinaemia and is positively associated with breast cancer risk in postmenopausal women (Sellers *et al.*, 1992; Galanis *et al.*, 1998). Possible reasons for association may be related to hormonal factors such as oestrogen synthesis from androstenedione (in adipose tissue), which is increased with greater body fat and the fact that obesity often leads to a state of hyperinsulinaemia with potential consequences on oestrogen and IGFs (Madigan *et al.*, 1998; Newcomb *et al.*, 1995).

2.6.7 Exercise performance

CHO is the main fuel for exercising muscles, therefore the amount, timing and type of carbohydrate (CHO) food ingested is an important part of an athlete's daily dietary intake. The amount and timing of CHO ingestion for increased performance and optimal glycogen storage has been investigated extensively (Jentjens and Jeukendrup, 2003; Jentjens *et al.*, 2001). The GI of CHO foods has been used when selecting foods and CHO-containing fluids to optimise CHO availability during exercise. It has also been suggested that the GI of CHO foods influences CHO availability during exercise and the rate of glycogen synthesis post-exercise (Wright, 2005).

2.6.7.1 Pre-exercise

Although low-GI (Low-GI, <40%) CHO foods are mostly recommended for the pre-exercise meal, ingesting high-GI (High-GI, >70%) CHO foods pre-exercise mostly does not result in hypoglycaemia in healthy individuals during exercise (Wright, 2005). Wright (2005) also suggested that if the total amount of CHO ingested pre-exercise is sufficient (1.1-2 g CHO/kg, 1-2 hours pre-exercise) and if CHO is ingested during exercise, the type of CHO in the pre-exercise meal can be determined according to the individual's preference and previous experience. However, in a small percentage of athletes that are sensitive to CHO ingestion during the hour before exercise the ingestion of a substantial amount of low-GI CHO (>70 g) may prevent rebound hypoglycaemia and

hyperinsulinaemia, including exaggerated CHO oxidation, decreased blood glucose levels, rapid onset of fatigue that is experienced by these athletes at the onset of exercise (Wright, 2005).

2.6.7.2 During exercise

CHO ingestion during prolonged submaximal intensity and intermittent intensity exercise has been associated with increased performance (Wright, 1991; Coggan and Coyle, 1991; Vergauwen *et al.*, 1998). It might also be beneficial for high-intensity exercise (Below *et al.*, 1995). Mechanisms for improved exercise performance as a result of CHO ingestion during exercise of \pm 1 hour are unclear, since only 5-15 g of ingested glucose could have been oxidised at the end of exercise. This suggests that benefits to 'central performance' involving the brain and nervous system may be involved (Carter *et al.*, 2004). High-GI and low-GI CHO foods yield similar results in terms of exercise performance and perceived rate of exertion, but Wright (2005) recommends high-GI and moderate GI CHO foods during exercise.

The argument that the consumption of fructose is often promoted while exercising because of the low insulinaemic response (Samols and Dormandy, 1963) is not well founded since insulin secretion is suppressed during exercise. A low insulinaemic response would, however, increase free fatty acid (FFA) oxidation, thereby sparing CHO oxidation (decreased glycogen depletion) and contributing to prolonged time to exhaustion (Guezennec *et al.*, 1989). Fructose oxidation is slower than that of glucose (High-GI) probably due to a lower rate of absorption and because it first needs to be converted to glucose in the liver before it can be metabolised (Jeukendrup and Jentjens, 2000). Although fructose can be used to increase the palatability of drinks, as well as water and CHO absorption, high concentrations of fructose is not recommended owing to increased risk of gastrointestinal distress (Wright, 2005).

2.6.7.3 Recovery after exercise

The recovery phase post-exercise is characterised by glycogen resynthesis and whole-body protein synthesis (Piehl, 1974). During short-term recovery periods (<8 hours) the type and timing of CHO consumed is more important than during longer recovery periods (>8 hours). The amount of CHO ingested remains equally important in both above mentioned recovery periods (Burke *et al.*, 1998).

Current recommendations is to ingest 1.0-1.85 g/kg/hour immediately post-exercise and at 15-60 minute intervals thereafter, for up to 5 hours post-exercise (Jentjens and Jeukendrup, 2003) during a short-term recovery period. During a longer recovery period total daily CHO intake should be sufficient (7-10 g/kg/day) (Coyle, 1991).

The GI is also thought to influence the rate of glycogen resynthesis post-exercise, thereby potentially enhancing exercise performance. High-GI CHO foods are currently recommended during the recovery period due to their high insulinaemic and glycaemic response (Brand-Miller *et al.*, 1996). Low-GI CHO foods are not recommended during a short recovery period (<6 hours) because of their rate of absorption and indigestible CHO, which seems to be a poor substrate for glycogen synthesis (Wright, 2005).

The effect of the GI on whole-body protein synthesis has not been investigated extensively. According to Wright (2005), combining a high-insulinogenic and high-GI CHO with protein immediately post-exercise is a potent stimulator for protein synthesis, probably due to increased insulin secretion and amino acid availability. A recent study by Suzuki (2003) concluded that high-GI CHO is more effective than low-GI CHO for skeletal muscle formation.

2.7 LABELLING

South Africa is in the process of legislating food labelling for the GI. This paves the way for inclusion of specific health messages regarding the GI on product labels. The main aims of food labelling are to inform consumers of the composition of the food, and to assist in the selection of a healthy diet. It is not only the comparison of two individual products, but rather the use of the label for continuous healthy food choices in order to improve the quality of the diet as a whole. These aims are not always easy to reconcile because the health benefit of different carbohydrate-containing foods cannot readily be communicated simply from a description of their composition (Venter *et al.*, 2003; Pieters and Jerling, 2005).

According to Pieters and Jerling (2005), the role of the GI when making food choices must be well understood by the consumer. When making these choices the GI should definitely not be seen as the only guideline to consider or to contradict or replace the Food Based Dietary Guidelines. The GI only comes into play when choosing carbohydrate-rich foods. Therefore the recommendation by the

DoH working group has been made that only foods containing 40% or more of the total energy value as glycaemic CHO, should be labelled. This working group also stipulated that fat content must not exceed 30% of total energy and the protein content not exceeding 42%. Only a selected group of products therefore qualify for labelling. Misuse of GI labelling will thus be prevented, for instance producers of products with a low CHO, high fat content will not be able to label them 'low-GI' foods.

Together, the GI and information on food composition can be used to guide food choices. Informing consumers of the GI on labels helps them to choose carbohydrate-containing foods based on expected physiological effects (e.g. blood glucose-raising potential) and may eliminate confusion surrounding the effect of different types of carbohydrates without using complex terminology (Venter et al., 2003).

To constitute a proper well-balanced low-GI diet, a wider range of low-GI products will be required. Substitution of one high-GI food with one low-GI food will probably not result in any clinical gains, but if the GI of the whole diet can be decreased then improvement in clinical symptoms can be expected, as had been proven in several studies (Jenkins et al., 1983; Jenkins et al., 1986; Wolever et al., 1987, Cole, 1997; Leeds, 2002; Miller, 1994). This should be kept in mind when discussing the labelling of products for GI. According to Venter et al. (2003) when planning a low-GI diet consumers should be advised that, whether a food should be included in such a diet or not, is not only dependant on the GI of the particular food. Some low-GI foods may not be a good choice because they are high in fat and/ or low in other nutrients and phytochemicals. It is obvious that the GI influences management of diabetes, impaired glucose tolerance as well as dyslipidaemia, in spite of variations in methodology for GI determination and many logical objections to its use in clinical practice (Pieters and Jerling, 2005; Opperman et al., 2005).

According to Pieters and Jerling (2005), to prevent misinterpretation, means and confidence intervals (CIs), the estimated range of values likely to include the means should be viewed together. It is of great importance to remember that the CIs sometimes span across two or even all three of the suggested categories, namely 0-55 = low, 56-69 = intermediate and 70-100 = high. Therefore, two types of products may seem to be in two different categories according to the means but statistically they do not differ when looking at their large CIs, indicating that there is no difference between these two products which makes the application of these GI categories to labelling, more complex. Pieters and Jerling (2005) stated, for labelling purposes a more precise reflection of the GI is to

express the GI values on a continuum and not categorized with fixed cut-off values. Fig.1 is an example of such a continuum.

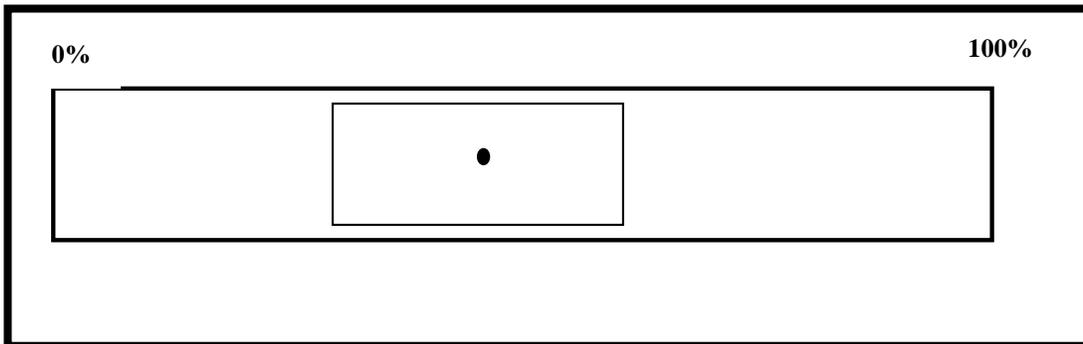


Figure 2.1 Proposed form of GI labelling (Pieters and Jerling, 2005).

The GI is then presented as a mean (dot) with 95% CI (square). Neutral colours should be used when labelling packages. The colour red commonly indicates danger and should be avoided when signifying a high GI, because customers might interpret such products as unhealthy. In marketing of foods this misinterpretation has the potential to be misused. This concurs with the philosophy that there are no good or bad foods, only good or bad diets (Pieters and Jerling, 2005).

Pieters and Jerling (2005) emphasized that by expressing the GI as a continuum, comparison between two products can still be done without classifying them as high or low GI. Products can be evaluated by comparing the position of the GI value on the continuum. Customers can thus make an educated decision about which of the two products has a higher or lower GI.

2.8 SUMMARY

Concerns have been raised about the variation in published GI values for foods that appear to be similar. This variation may reflect both true differences in the physical and chemical characteristics of the food and methodological factors. Differences in testing methods include the use of different types of blood samples (capillary or venous), experimental time periods and food portion sizes (Foster-Powel *et al.*, 2002). Correct application of the GI is dependant on correct GI calculation. The real public health impact in both diabetic and other populations will only be known so long as the GI is calculated in a scientifically expectable way (Pieters and Jerling, 2005).

The glycaemic and insulin responses to food are influenced by either physiological individual factors or food factors (Vorster et al., 1990; Wolever et al., 1991). Factors influence the rate of absorption or digestion and, in turn, the glycaemic responses. Some of the food factors that influence the GI include food form, particle size, cooking, processing and starch structure etc. (Augustin et al., 2002). It is thus, for these reasons that the GI range rather than an absolute value may be expected for each food as differences of 10 to 15 units are within the error associated with the measurement of the GI (Wolever, 1991; Perlstein et al., 1997).

Several studies have shown that eating a low GI diet have health benefits. Evidence from prospective studies shows that low GI-diets are associated with reduced risk of diabetes (especially type 2 diabetes) (Hodge et al., 2004; Frost et al., 1998; Salmerón et al., 1997a; Salmerón et al., 1997b), cardiovascular disease (Liu et al., 2000), cancer (Augustin et al., 2003a; Augustin et al., 2003b; Augustin et al., 2004a; Augustin et al., 2004b; Augustin et al., 2004c; Augustin et al., 2001), and the metabolic syndrome (McKeown et al., 2004). Low GI foods improve overall blood glucose control in people with type 2 diabetes (Wolever et al., 1992), reduce serum lipids in people with hypertriglyceridaemia (Jenkins et al., 1987;) and improves insulin sensitivity (thus reduces insulin demand) (Frost et al., 1998; Riccardi and Rivellese, 2000; Augustin et al., 2002; Slyper, 2004). In addition, the intake of a low GI-diet is associated with higher concentrations of high-density lipoprotein (HDL) cholesterol (Frost et al., 1999) and significant reductions in low-density lipoprotein (LDL) cholesterol as well as total cholesterol (Opperman et al., 2005). Diets based on low-fat foods that produce a low glycaemic response (low-GI foods) may in contrast enhance weight control by promoting satiety, minimizing postprandial insulin secretion, and by maintaining insulin sensitivity (Brand-Miller, 2002). When low glycaemic carbohydrates are incorporated into an energy restricted diet, there is a greater fall in insulin resistance that cannot be accounted for by weight-loss alone (Slabber et al., 1994).

Various hurdles should first be overcome by health professionals and customers, because only then can eating according to the GI, make an impact on the diet and lifestyle of diabetics as well as other clients. Therefore nutritional advice should be adequately conveyed in a basic and understandable way without misuse of the GI concept. It is thus imperative that cultural and ethnic preferences and traditions should be re-examined, and health professionals must therefore move away from a 'good food', 'bad food' approach (Slabber, 2005).

Pieters and Jerling (2005) stated, for labelling purposes a more precise reflection of the GI is to express the GI values on a continuum and not categorized with fixed cut-off values. They also emphasized that by expressing the GI as a continuum, comparison between two products can still be done without classifying them as high or low GI. Products can be evaluated by comparing the position of the GI value on the continuum. Customers can thus make an educated decision about which of the two products has a higher or lower GI.

According to Venter et al. (2003) if a food carries a GI given to it by an accredited laboratory and if that food meets specific nutrition criteria, it is acceptable to label the GI value and give a short explanation near the nutrition information panel of the packaging. This may inform customers on how to construct a balanced diet through use of the GI concept and challenge the food industry to develop low-GI versions (for the benefit of diabetics, persons with cardiovascular disease and metabolic syndrome) and high-GI versions (for the benefit of physically active persons) of a specific product. If the industry complies, consumers and health professionals will be able to make educated choices about the quality of carbohydrates in foods.

Chapter 3: METHODOLOGY

3.1 INTRODUCTION

A well known food company commissioned the Department of Human Nutrition at the Free State University to carry out a scientific measurement of the GI of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread. This study was conducted in agreement with the latest guidelines based on the results of international studies (Wolever *et al.*, 2003) and the recommendations of the South African GI Task Force (2002).

3.2 OBJECTIVES

The aim of this study was to determine the GI of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread using capillary blood sampling and to determine whether there were significant differences between the GIs of the products mentioned.

3.3 METHODS

3.3.1 SUBJECTS

Twenty healthy male volunteers, aged 18-27 years, with a mean body mass index (BMI) of 24.85 ± 2.02 kg/m² were recruited to take part in the study. The GI Task Force (2002) suggests a minimum of 10-20 subjects to be recruited based on willingness to comply with the protocol, inclusion and exclusion criteria. Each subject acts as his own control. The GI Task Force (2002) also stipulates that a homogenous group of subjects should be included. Therefore only male volunteers were included and each subject acted as his own control. Exclusion criteria included: any known disease, abnormal glucose tolerance (i.e. fasting plasma glucose is 7.8 mmol/L or higher or a random plasma glucose concentration of 11.1 mmol/L or higher and/or ≥ 11.1 mmol/L after 2 hours or more on oral glucose tolerance testing (Franz, 2004, p. 799), the use of any medication that may affect blood glucose tolerance and a BMI greater than 28 kg/m².

3.3.2 STUDY DESIGN

A randomized cross-over intervention study was conducted. Volunteers randomly consumed six different test meals consisting of three different test foods (three types of bread) and one type of standard food (glucose) (repeated three times in each subject) according to a Latin square design. Six combination sets were used, four sets repeated three times, while two sets repeated four times (because 20 subjects were used). It must however be noted that glucose was not randomized because every subject received the same standard food. Glucose (standard food) was given in week one, three and five as indicated in table 3.1. Randomisation was done according to Table 3.1 using a Latin square design.

Table 3.1 Latin square design for subjects (as received from Dept. Biostatistics)

STUDENT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
WEEK	1					2					3					4				
Day	1					2					3					4				
1	S1																			
2	P2	P3	P2	P1	P3	P1	P2	P2	P3	P3	P1	P1	P2	P2	P1	P3	P3	P2	P1	P2
3	S2																			
4	P3	P1	P1	P3	P2	P2	P3	P1	P1	P2	P3	P2	P3	P1	P3	P1	P2	P3	P2	P1
5	S3																			
6	P1	P2	P3	P2	P1	P3	P1	P3	P2	P1	P2	P3	P1	P3	P2	P2	P1	P1	P3	P3

P= Product (test food) (three different products = P1, P2, P3)

P1=Whole wheat (A)

P2=Brown (B)

P3=Best of Both (C)

S= Standard food

3.3.3 OPERATIONAL DEFINITIONS

For the purpose of this study the following operational definitions were used:

3.3.3.1 Glycaemic Index: The glycaemic index (GI) is defined as the incremental area under the curve for the increase in blood glucose after the ingestion of 50g of glycaemic (available) carbohydrate of a test food in the 2-hour for healthy individuals post ingestion period as compared with ingestion of the same amount of glycaemic (available) carbohydrate from glucose taken with 300ml of water spread over a 10-15 minute period, tested according to a defined procedure in the same individuals under the same conditions using the fasting blood glucose concentration as a baseline (GI Task Force, 2002).

3.3.3.2 Food-portion size: 50g of glycaemic CHO of a test food (GI Task Force, 2002).

3.3.3.3 Glycaemic carbohydrate is defined as the carbohydrate available for metabolism and the summation of the analytical values of mono-, di- and oligosaccharides, starch and glycogen but excluding fructo-oligosaccharides and other non-digestible oligosaccharides and resistant starch (Brand-Miller and Gilbertson, 2001). Englyst Carbohydrates Research and Services Ltd., Cambridge, United Kingdom used the Englyst method (Englyst *et al.*, 1999) to determine 50g of available glycaemic CHO in the test foods included in the present study. Each meal contained 50 g available glycaemic carbohydrate.

Method of calculation to determine 50 g available glycaemic carbohydrate from the three Albany Superior™ breads as eaten:

Whole Wheat™ bread:

100 g Whole Wheat™ bread = 43.3 g available glycaemic CHO

115.5 g rounded to 116 g Whole Wheat™ bread = 50 g available glycaemic CHO

Brown™ bread:

100 g Brown™ bread = 43.0 g available glycaemic CHO

116.3 g rounded to 116 g Brown™ bread = 50 g available glycaemic CHO

Best of Both™ bread:

100 g Best of Both™ bread = 42.6 g available glycaemic carbohydrates

117.4 g rounded to 117 g Brown™ bread = 50 g available glycaemic CHO

3.3.4 GENERAL PROCEDURES

Figure 3.1 illustrates the procedure of the study.

PROCEDURES OF THE STUDY

Submission for evaluation to the Research Committee of the School for Allied Health Professions

Application for funds

Approval by Ethics Committee, Faculty of Health Science

Recruitment of subjects

Informed consent (Appendix B)

Standardized Pre-evening test meal

Fasting (10-12 hours)

Blood Sampling

Fasting capillary finger prick

Ingestion of test food or standard food (50 g of glucose)

Capillary blood sampling

- Six times on every subject
- Over a study period of six weeks, once a week per subject

15 min after ingestion

30 min after ingestion

45 min after ingestion

60 min after ingestion

90 min after ingestion

120 min after ingestion

Analysis of data

Documentation

Blood Sampling Procedure

Once a week over a six-week period

Figure 3.1 The procedure for the study

The Research Committee of the School for Allied Health Professions evaluated the protocol where after it was approved by the Ethics Committee, Faculty of Health Science, UFS (ETOVS no:195/05). The protocol with the budget was also submitted to the management of the food company that commissioned the research.

Twenty male volunteers were recruited via announcements in UFS male hostels and on bulletin boards. After consent was given by the subjects or their parents a random finger prick blood glucose test was taken by the researcher during recruitment to ensure normal glucose tolerance. The recruitment form (containing the inclusion and exclusion criteria) was completed during recruitment by the researcher (Appendix B). Volunteers who met all inclusion criteria were asked to fill in the necessary informed consent and indemnity forms (Appendix C). All rules, procedures regarding the execution of the study and factors that might influence glucose response were carefully explained. The study took place over a six week period. Every morning, Tuesday to Friday, five subjects (per day) were studied. Capillary blood sampling was performed on a specific day, every week, over a six week study period. Thus, each subject participated in the study once a week, on a specific day for a six week period. Volunteers randomly consumed six different test meals consisting of three different test foods (three types of bread) and one type of standard food (glucose) (repeated three times in each subject) according to a Latin square design.

Subjects did not smoke, use alcohol or exercise 12 hours prior to testing and did not consume anything other than water from 10h00 p.m. the night prior to testing (GI Task Force, 2002). All medication, including complementary or natural medicines and the use of supplements used by subjects before and during the test, remained the same during the test period. Records was kept of all medications (Appendix B) used by all subjects (GI Task Force, 2002). Every pre-test evening the subjects gathered at the seminar room, Department of Surgery to consume a standard pre-evening test meal (containing 60% of the total kJ from CHO; 25% from fat; 15% from protein) as set out in Table 3.2 to optimize CHO metabolic enzyme induction and to standardize potential "second meal" effects (GI Task Force, 2002; Gresse and Voster, 1992). The subjects were instructed to spend the evening relaxing at home and to be in bed by 23h00.

Table 3.2 Pre-evening test meal*

Food	Amount	Carbohydrates (g)	Protein (g)	Fat (g)	Energy (kJ)
Milk, fat free	250mL	12.3	8.5	0.5	372.5
Apple (peeled)	80g	11.8	0.2	0.2	212.8
Bread, white (crusts removed)	6 x 30g	94.3	15.3	3.2	1983.6
Jam	35g	24.5	0.1	0.0	419.3
Cheese, medium fat	60g	1.3	14.9	16.4	855
Margarine, medium fat	10g	0.0	0.0	6.4	240
Total		144.2	39.1	26.7	4083
Total kJ		2422.56	656.8	1014	4083
% of kJ		59%	16%	25%	100%

* Calculated according to the MRC Food Composition Tables (Langenhoven *et al.*, 1991; Jerling *et al.*, 2002).

3.3.5 DETAIL ON CAPILLARY WHOLE BLOOD SAMPLING

For four days per week, five of the twenty subjects per day participated in the study on that specific day and consumed the test food or standard food as well as for blood sampling. The researcher (a trained registered dietician) with the aid of one other trained registered nurse or medical doctor obtained whole capillary blood glucose samples via finger-pricks, using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™) (7 times). This procedure was done in strict compliance with the protocol recommendations of the manufacturer as well as good laboratory practice as described by the GI Task Force (GI Task Force, 2002). All One Touch Ultra™ glucometers (Lifescan™) were calibrated each morning before testing with One Touch Ultra™ glucose control solution (Lifescan™). All subjects' hands were washed with soap and water before testing started. As recommended, no alcohol swabs were used to disinfect the finger. The side of the finger was pricked and by allowing the hand to hang, blood gravitated to the finger. The finger was gently squeezed (without milking the finger) and one large drop of blood was applied to the test strip on the blood glucose sensor electrode (GI Task Force, 2002). The standard and test food was given in the seminar room, Department of Surgery. The glucose values were noted on a data form (Appendix D) by the researcher and one trained registered nurse or medical doctor.

On the test days whole capillary blood glucose samples via finger-pricks were obtained while subjects remained seated, and relaxed (for example reading, conversing with fellow subjects). The following whole capillary blood glucose samples were obtained:

- Fasting blood glucose
- Subjects then randomly consumed a test meal, within 10-15 min, which consisted of either a **standard food** of 50g glucose powder dissolved in 300mL of water or an amount of **test food** containing 50 g of available CHO without crusts with 300 ml water in the seminar room of Department of Surgery.
- A blood sample was taken every 15 min for 1 hour (15 min, 30 min, 45 min, 60 min).
- Thereafter every 30 min for 1 hour (90 min, 120 min). The time schedule for blood sampling per test day (5 students per day) is set out in Table 3.3.

Table 3.3 Time schedule for blood sampling per test day

Finger prick	Fasting blood glucose	15 min after ingestion	30 min after ingestion	45 min after ingestion	60 min after ingestion	90 min after ingestion	120 min after ingestion
<i>Student</i>							
1	8:00	8:15	8:30	8:45	9:00	9:30	10:00
2	8:10	8:25	8:40	8:55	9:10	9:40	10:10
3	8:20	8:35	8:50	9:05	9:20	9:50	10:20
4	8:30	8:45	9:00	9:15	9:30	10:00	10:30
5	8:40	8:55	9:10	9:25	9:40	10:10	10:40
6	8:50	9:05	9:20	9:35	9:50	10:20	10:50

3.3.6 STANDARD FOOD

Standard food refers to 50 g glucose powder dissolved in 300 ml of bottled still water. The standard food was prepared in the seminar room of the Department of Surgery by the researcher and one trained registered nurse.

According to Venter *et al.* (2003), if glucose is used as standard it should be selected from the same batch and purchased in bulk. Glucose solutions should be served at the same temperature. These procedures were followed in the study.

3.3.7 TEST FOODS

The foods tested included three Albany Superior™ breads namely Best of both™, Brown™ and Whole Wheat™ bread. The test foods were prepared in the seminar room of the Department of

Surgery. Each test meal contained 50g available CHO (consumed with 300 ml bottled still water). The nutritional composition of the three different breads is summarized in Table 3.4. However, to determine the amount of glycaemic CHO, samples of the three breads were couriered to Englyst Carbohydrates Research and Services Ltd. in Southampton, UK to be analysed.

Subjects had to consume the test foods or standard food within 10-15 minutes after the fasting blood glucose value was obtained and the timer was started. The timer started with the first bite of the test meal – so the first blood sample was taken after 15 min. Time was kept by the researcher and one trained registered nurse or medical doctor.

According to Venter *et al.* (2003) all breads used should come from the same batch and supplier to avoid differences in the quality and quantity of CHO load. Because of the influence of the Maillard reaction on the availability of CHO from the bread crusts, all crusts were removed. Bread ingested on different days was frozen and thawed according to methods prescribed for test foods to ensure uniformity. This was done because bread is not a consistent food and may go stale, losing water when standing at usual indoor temperatures. This procedure was followed throughout the study.

Table 3.4 Macronutrient composition of the three different breads (Albany bakeries, 2006).

BREAD	BROWN™	WHOLE WHEAT™	BEST OF BOTH™
Slice weight (g)	37.5	40	37.5
Kilojoules (KJ)	368	375	338
Carbohydrates (g)	15.9	16.24	15
Protein (g)	3.12	3.74	2.89
Fats (g)	0.715	0.96	1.09
Fibre (g)	2.37	2.9	2.48

3.3.8 METHODOLOGICAL AND MEASUREMENT ERRORS

Errors that may occur: On the test days subjects were asked whether they smoked, took alcohol or ate after 22:00. If a subject smoked or failed to stay fasting, he was omitted from the study for that particular day, only to be included again on a different day later in the week (this happened to none of the subjects). However, through the course of the study, five subjects wrote exams during the test-day time-interval (8:00-11:00 am) and requested to participate later in the week.

3.3.8.1 Validity and reliability

a) Validity

Validity as a concept of measurement refers to the degree to which a research procedure or tool measures what it is supposed to measure (Koh and Owen, 2000, p.175).

Instrumentation: Instrumentation accuracy is confounded when more than one person is involved in measuring the GI. The accuracy of the instrumentation was controlled by the fact that measurements were taken by the researcher (a trained registered dietician) and one other trained registered nurse or medical doctor. The procedures were standardized in measuring capillary whole blood glucose using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™) during a pilot study.

Interaction of selection bias and experimental treatment (population sample difference): To control the threats of this type of external validity, only healthy, male volunteers between the ages of 18 and 27 years, with a mean body mass index (BMI) of 20-28 kg/m² were recruited to take part in this study. Males from different socio economic classes and degrees were included. According to FAO/WHO (1998) and GI Task Force (2002) within-subject variability will be reduced by three required measurements of the standard food and one required measurement for the test food. This was done in the study. The above mentioned bias was thus be compensated for by using randomization.

b) Reliability

Reliability refers to the degree to which an instrument accurately and consistently measures a given phenomenon from one time to another (Koh and Owen, 2000, p.171). Reliability can be obtained by ensuring that the same persons (registered nurse and the researcher) measures the whole capillary blood glucose concentration using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™) according to the standardised procedures.

3.3.9 PILOT STUDY

A pilot study was performed in five healthy male volunteers who were not included in the study. Procedures were standardized in measuring capillary whole blood glucose using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™).

3.3.10 STATISTICAL ANALYSIS

The statistical analysis was done by the Department of Biostatistics, Faculty of Health Sciences. Results were expressed as incremental areas under the glucose curves, ignoring the area beneath the fasting level (AUC) and the GI. Description statistics namely means and standard deviations for continuous data were calculated per bread. The breads were compared by means of 95% confidence intervals. The mean GI per bread was described by means of 95% confidence interval for the mean. Repeated measures analysis, with covariance structure chosen as compound symmetry was calculated and the F-statistics of the type 3 tests of fixed effects was noted.

3.3.11 IMPLEMENTATION OF FINDINGS

Results of this study supplied scientific GI values for three well known bread types on the market. The GI of a food can be used to guide consumers in choosing a particular food with a predicted known effect on blood glucose levels and homeostasis. According to Pieters and Jerling (2005) it is, however, of utmost importance to inform patients and consumers that in using the GI to choose CHO foods, physiological responses to a food may vary between individuals. Therefore, if food is indicated as having a high GI it does not mean that it will have a high GI in everybody. They also concluded that it is quite normal for a specific food to have a high GI in some individuals and a medium or even a low GI in others.

3.3.12 ETHICAL ASPECTS

The protocol was submitted to the Ethics Committee, Faculty of Health Sciences, UFS for approval. If the subjects met the inclusion criteria during recruitment, the necessary informed consent and indemnity forms were completed by the subjects or their parents (Appendix C). The consent form contained information with respect to the study and the selection criteria. All rules and procedures regarding their trial were carefully explained to minimize factors which may influence glucose responses. If a person's blood glucose tested high (random plasma glucose concentration of 11.1 mmol/L or higher) during the recruitment period, they would have been referred for medical treatment, but this was not necessary for anybody.

Confidentiality was ensured throughout the study. Forms (recruitment and data forms) were only numbered and no names were used on the data forms (Appendix B & D). A letter asking permission to use healthy male student volunteers for participation in this study was sent to the Vice-Dean: Student Affairs and to the Vice-rector: Academic Planning, where after they gave permission for students to partake in the study. Please refer to Appendix A.

3.3.13 LIMITATIONS OF THE STUDY

Acceptability of the meals raised some concerns. Some of the subjects complained about the glucose water (300 ml + 50g glucose) saying it was too sweet and some about the dry (without margarine or any other spread) bread. In some ethnic groups dry fresh bread is acceptable, but in other ethnic groups it is unacceptable. Despite this, all the subjects consumed their glucose water (standard food) and dry bread (3 types of test food) without any problems.

One could have used white bread as a standard food, but due to limited time it was decided on glucose as standard food. According to Pieters and Jerling (2005) it is easy to standardise glucose whereas differences in locally produced white breads might add to analytical variation. These three breads, however, still need to be tested in relation to mixed meals.

Some of the students wrote tests during the time of the study which could have led to some stress. Most of the subjects (17) were students and three were working young men. The study was

conducted between 8:00 and 11:00 in the morning after a 10-12 hour fast. The GI Task Force recommends a 10-12 hour fast before testing. Brouns and colleagues (2005), however, recommend that the test should take place before 10:00 in the morning, after a 10-14 h fast. Brouns et al. (2005) recommend both the measurement of glycaemic responses and insulinaemic responses for completeness if budget limitations do not play a role. However, they concluded that for routine use of the GI method, glucose measurement is enough.

Unfortunately, due to financial constraints, subjects were free-living and not in a metabolic unit, where better controlled conditions would have been easier to enforce. The rules of the study were, however, explained to each subject via informed consent. At the start of each test day subjects were also asked whether they complied with the rules or not, to which they answered affirmatively.

CHAPTER 4: RESULTS, DISCUSSION AND RECOMMENDATIONS

4.1 INTRODUCTION

An independent assessment of the GIs of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread was commissioned by Tiger Brands. The determination of the three GIs was carried out under strictly standardised conditions using methods complying with the most recent internationally accepted methodology. The objectives of this study were to determine the GI of three Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread using capillary blood sampling and to determine whether there were significant differences between the GIs of the products mentioned. The area under the curve (AUC) and GI of Best of Both™, Brown™ and Whole Wheat™ bread were determined using capillary whole blood. The amount of bread needed to supply 50 g glycaemic CHO was calculated based on the analysis of the glycaemic carbohydrate content of the products, as determined by the Englyst Carbohydrate Research and Services Ltd. in the UK.

Different testing methods used in different parts of the world can markedly affect the interpretation of the glycaemic responses and the GI value obtained for foods (Wolever, 1990; Foster-Powell *et al.*, 2002). Methodological variables that affect the GI value include food-portion size, the method of blood sampling, sample size and subject characteristics [within-subject variation, body mass index (BMI)], standard food, available CHO, volume and type of drinks consumed with test meals (Wolever *et al.*, 1991; Venter *et al.*, 2003).

4.2 SUBJECT CHARACTERISTICS

Table 4.1 summarises subject characteristics of the twenty healthy males that were recruited to take part in the study.

Table 4.1. Subject characteristics

Characteristics	Mean	Standard deviation (SD)	Lower Quartile (Q1)	Median	Upper Quartile (Q3)	Min	Max
Age (years)	21	2.14	20	21	22	18	27
BMI (kg/m ²)	24.85	2.02	23.6	24.60	26.80	20.51	27.68
Random glucose (mmol/L)	5.3	0.66	4.80	5.3	5.75	4.6	7.4

BMI =body mass index

Study compliance was good, therefore data of all twenty subjects is presented. Most GI studies have been done with five to ten subjects (Foster-Powell et al., 2002). However, Nell (2001) indicated that if a 10% range for a GI of a food is sought with 80% confidence, between 24 and 90 subjects should be included in a study using venous plasma samples. Brouns et al. (2005) advises that the inclusion of ten subjects provides a reasonable degree of power and precision for most purposes of measuring GI, but that the number of subjects can be increased if the aim of the study is to detect small differences in GI or when greater precision is required. The GI Task Force (2002) recommends a minimum of 10-20 subjects to be recruited based on willingness to comply with the protocol, inclusion and exclusion criteria.

The median age of participants included in this study was 21 years. According to Venter et al. (2003) dietary changes and lower physical activity may affect glucose tolerance with increasing age, but no significant differences in the glycaemic responses between adults and children were found by Wolever and colleagues (1988). Median BMI was 24.60 kg/m², which fell within the WHO's normal range of 18.5 to 24.9 kg/m² (DoH, 2001). According to FAO/WHO (1998) in a non-diabetic study sample subjects in normal BMI range of 18.5-24.9 kg/m² should be included. Median random glucose was 5.3 mmol/L, which is within the normal range (random plasma glucose of < 11.1 mmol/L) as described by Franz (2004, p. 799).

Medications for common colds and flu were used during the study. Subjects were instructed to phone the researcher before using any medication, in order to exclude medications which may have an effect on blood glucose. None of the medication used during the duration of the study had any effect on blood glucose.

4.3 WITHIN-SUBJECT AND BETWEEN-SUBJECT VARIATION IN BLOOD GLUCOSE RESPONSES TO THREE GLUCOSE REFERENCE TESTS

It is a well known fact that glycaemic responses vary substantially within as well as between subjects. As indicated in Table 4.2, subjects 5, 7, 11, 12, 16, and 19 showed the largest variation in AUC between glucose reference test (GRT) 1 and GRT 2, while subjects 4, 6, 7, 9, 10 and 13 showed the largest variation in AUC between GRT2 and GRT 3. The fasting blood glucose was used as baseline to measure the AUCs for three glucose tests for each subject. Although the standard food was consumed under standardised conditions, day to day variation of the glycaemic response to glucose also occurred within the same subject.

Table 4.2. The AUC for individual subjects for the three glucose reference tests (GRT) using capillary blood

Subjects	AUC GRT 1	AUC GRT 2	AUC GRT 3
1	105.09	89.44	59.34
2	113.16	128.46	131.30
3	163.50	132.71	104.85
4	204.06	249.75	178.30
5	328.63	221.17	216.90
6	136.60	161.75	240.36
7	306.75	400.50	509.25
8	121.50	144.75	111.47
9	106.88	97.71	188.08
10	118.61	112.72	203.10
11	205.50	42.75	109.50
12	144.39	229.27	249.50
13	108.75	154.75	71.25
14	133.04	151.09	143.69
15	102.94	153.00	106.04
16	399.00	209.39	217.03
17	78.38	122.33	83.35
18	193.15	273.83	270.50
19	190.35	298.35	234.17
20	169.23	104.42	139.85

GRT = glucose reference test

As indicated in table 4.2, it is clear that the ingestion of oral glucose on three different occasions resulted in large areas under the curve for six subjects, thus indicating that there were large differences within-subjects as well as between-subject. The mean AUC, 95% CI and SD of the three different standard meals, determined by using capillary blood glucose values are listed in Table 4.3.

Table 4.3. The mean AUC for the three glucose reference tests using capillary blood

	AUC GRT 1	AUC GRT 2	AUC GRT 3
Mean	171.47	173.91	178.39
95%CI	[131.89; 211.05]	[134.23; 213.59]	[131.21; 225.57]
SD	84.57	84.79	100.81
Lower Quartile (Q1)	110.96	117.52	107.77
Median	140.49	152.05	160.99
Upper Quartile (Q3)	198.61	225.22	225.60
Minimum	78.38	42.75	59.34
Maximum	399.00	400.50	509.25

SD=standard deviation; CI=confidence interval for the mean; GRT = glucose reference test

The 95% confidence interval (CI) for the median difference between the AUCs for paired data of GRT1 and GRT2 are [-46; 15.66]. The 95% CI for the median differences between the AUCs for paired data of GRT1 and GRT3 are [-77.35; 37.5]. The 95% CI for the median differences between the AUCs for paired data of GRT2 and GRT3 are [-35.43; 33.3]. Thus, the 95% CI for the median differences between the AUCs for paired data show that there is no statistically significant difference between the AUCs of the three different glucose reference tests.

4.4 BLOOD GLUCOSE RESPONSES OF GLUCOSE AND THE THREE DIFFERENT BREADS

The mean capillary blood glucose response (measured at 0, 15, 30, 60, 90, and 120 minutes) for the three different types of Albany Superior™ breads and glucose (standard food) are listed in Table 4.4. The 95% CI for the mean difference per time interval blood glucose responses for paired data show that there is no statistically significant difference between these blood glucose responses of the three different Albany Superior breads.

Table 4.4. Blood glucose responses after every time interval for the three Albany Superior™ breads as well as glucose (standard food)

Time intervals (min)	Whole Wheat™ (n=20)	Brown™ (n=20)	Best of Both™ (n=20)	Glucose (n=60)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
T ₀	4.89 ± 0.39	5.07 ± 0.40	4.87 ± 0.36	5.08 ± 0.51
T ₁₅	5.36 ± 0.77	5.53 ± 0.48	5.30 ± 0.49	6.56 ± 0.93
T ₃₀	6.60 ± 0.77	6.73 ± 1.13	6.69 ± 0.74	8.32 ± 1.27
T ₄₅	7.14 ± 0.99	7.02 ± 1.00	7.11 ± 1.08	8.07 ± 1.61
T ₆₀	6.31 ± 0.87	6.33 ± 0.94	6.38 ± 0.78	7.04 ± 1.55
T ₉₀	5.46 ± 0.72	5.60 ± 0.79	5.57 ± 0.53	5.31 ± 1.18
T ₁₂₀	5.35 ± 0.67	5.55 ± 0.68	5.20 ± 0.58	4.55 ± 0.75

4.5 THE AUCs FOR THE THREE DIFFERENT BREADS

The *incremental AUC* (IAUC) calculates the area under the curve starting from the fasting value as baseline and therefore excluding any part of the curve that drops below the fasting value, and is a measure of the change of blood glucose from the fasting condition (Vorster *et al.*, 1990; Pieters and Jerling, 2005). According to Wolever *et al.* (2003; 1990), the GI is only based on the incremental area below the curve and above the fasting level. This method was chosen by the DoH GI working group as the method of choice because it is used most often internationally and is also recommended by the FAO/WHO Expert Consultation Group on Carbohydrates in Human Nutrition (Pieters and Jerling, 2005). In this study this approach was used to calculate the GI. The mean AUC, 95% CI and SD of the test meals, determined by using capillary blood glucose values are listed in Table 4.5.

Table 4.5. The mean AUC for the three different Albany Superior™ breads and glucose using capillary blood.

	Whole Wheat™	Brown™	Best of Both™	Glucose (total)
Mean	126.76	117.06	128.33	174.59
95%CI	[94.04; 159.48]	[87.77; 146.35]	[104.35; 152.29]	[154.23; 194.95]
SD	69.91	62.57	51.22	78.83
Lower Quartile (Q1)	76.21	71.13	92.06	122.48
Median	112.50	103.00	120.40	140.22
Upper Quartile (Q3)	181.20	172.88	161.01	225.83
Minimum	10.50	18.96	45.38	84.63
Maximum	319.50	219.00	259.50	405.50

SD=standard deviation; CI=confidence interval for the mean

The 95% confidence intervals (CI) for the median difference between the AUCs for paired data of Whole Wheat™ and Brown™ bread are [-36.4; 55.7]. The 95% CI for the median differences between the AUCs for paired data of Whole Wheat™ and Best of Both™ bread are [-65.2; 48.9]. The 95% CI for the median differences between the AUCs for paired data of Brown™ and Best of Both™ bread are [-57.4; 26.3]. Thus, the 95% CI for the median differences between the AUCs for paired data show that there is no statistically significant difference between the AUCs of the three different Albany Superior™ breads. However, a tendency exists for Brown™ bread to have a lower AUC than Best of Both™.

4.6 THE GIs OF THE THREE DIFFERENT ALBANY SUPERIOR™ BREADS

The GIs of the three different Albany Superior™ breads namely Best of Both™, Brown™ and Whole Wheat™ bread, using capillary blood for each subject are listed in Table 4.6. The glycaemic response to the three different Albany Superior™ breads are subject to both within-subject and between-subject variation. Physiologic responses to the three breads varied widely within- as well as between individuals. The variability of the glycaemic response for a given food for any one individual is similar to that seen for the oral glucose tolerance test (Wolever, 1990). In other studies, within-subject variation of healthy subjects to glucose varied from 19% (Nell, 2001) to 63% (Aginsky *et al.*, 2000). Variation between individual subjects in the glycaemic response to a food and in the GI of same food also exists. According to Venter *et al.* (2003) the latter findings have an

important practical use in GI determination for research or labelling purposes, as this proposes that, as long as the group is homogeneous, it would not be necessary to use the same subjects repeatedly and that larger groups of subjects could be used less often. Between-individual variation can be reduced to ~10%, if the glycaemic response is expressed as a percentage of an individual's response to a standardised food (i.e. 50 g white bread or glucose) (Jenkins *et al.*, 1981; Jenkins *et al.*, 1983; Wolever *et al.*, 2003).

Table 4.6. The GIs for individual subjects for the three different Albany Superior™ breads using capillary blood.

Subjects no.	Whole Wheat™	Brown™	Best of Both™
1	12.41	42.54	128.51
2	48.91	15.26	67.38
3	95.37	53.68	33.94
4	48.46	39.00	79.38
5	65.78	67.50	57.23
6	102.33	112.77	56.38
7	78.79	49.94	64.00
8	94.71	28.59	73.86
9	151.27	36.26	96.31
10	72.51	119.64	52.88
11	105.66	131.72	179.87
12	86.29	39.91	81.96
13	94.77	63.18	81.67
14	37.31	69.94	109.50
15	154.16	119.44	113.58
16	21.85	79.60	41.71
17	104.65	200.41	72.08
18	37.27	55.22	67.48
19	23.48	38.60	53.54
20	132.88	77.08	81.11

Controversy about the clinical utility of classifying foods according to their glycaemic responses by using the GI method has been reported (Bessenen, 2001). Variation in the GI values for apparently similar foods may reflect both methodologic factors as well as true differences in the physical and chemical characteristics of the specific food (Foster-Powell *et al.*, 2002). Venter and co-workers

(2005) emphasised that differences in GI values of similar foods could also be due to inherent botanical differences from country to country. Two similar foods may also have different ingredients (type of flour used), may have been processed with a different method (cooking time may vary) or may have differences in moisture content, which can result in differences in the degree of starch gelatinisation, resulting in significant differences in the rate of CHO digestion and consequently the GI value (Foster-Powell et al., 2002).

The mean GIs for the three different Albany Superior™ breads are listed in Table 4.7. The mean GIs using capillary blood samples were 78.44, 72.01 and 79.62 for Whole Wheat™, Brown™ and Best of Both™ bread respectively. No statistically significant differences were found between the GIs of the three different Albany Superior™ breads. With 95% confidence, the conclusion can be made that the true mean GI for Whole wheat bread was between 58.9% and 97.9% (intermediate to high) for Brown bread between 51.1% and 92.9% (low to high) and for Best of Both bread between 63.9% and 95.3% (intermediate to high) measured. The three different Albany Superior™ breads fell between the intermediate and high categories as defined by the draft Regulations Relating to Labelling and Advertising of Foodstuffs in the Foodstuffs, Cosmetics and Disinfectants Act of 1972 (Act no.54 of 1972), which was published for comment on August 8, 2002 (Jerling et al., 2002).

As included in the most recent international tables (using glucose as standard) the mean GI of whole wheat flour bread as determined in thirteen studies is 71 ± 2 (52- 87), 53 ± 3 (48-58) for wheat bread (cracked wheat kernel) as determined by the mean of two studies and 68 ± 1 (67-69) for white fibre enriched bread (white, high-fibre, because Best of Both is a white bread with added wheat fibre) as determined by the mean of two studies (Foster-Powell et al., 2002). As discussed above it is difficult to compare the GI values of similar foods because of the variation in ingredients used and due to inherent botanical differences that vary from country to country as well as the differences in methodological variables that can also affect the GI value (Foster-Powell et al., 2002; Venter et al., 2003, Wolever, 1990; Wolever et al., 1991). As already mentioned in chapter 3, to some ethnic groups dry fresh bread (without margarine or any other spread) is acceptable, but in other ethnic groups it is unacceptable. It should however be noted that usually bread is not consumed alone and that the fat in margarine or butter could modify the glycaemic response (lower the GI) to bread, by slowing gastric emptying.

According to Mbhenyane *et al.* (2001), mealiemeal porridge, with or without sugar, traditional fermented Sorghum porridge (*ting*) and nowadays acid-added porridge (with added vinegar or tartaric acid) are just a few examples of South Africa's indigenous staple foods and dishes. As determined by using venous blood sampling (white bread as standard), the GI of mealiemeal porridge, with or without sugar, traditional fermented Sorghum porridge (*ting*) and acid-added sorghum porridge are 123.3 ± 47.5 or 117.6 ± 51.8 , 113.2 ± 61.3 and 64.3 ± 20.0 respectively. Venter *et al.* (2005) found that capillary blood samples had a lower coefficient of variation (CV) than venous samples and were on average higher than in venous plasma. The mean GIs using capillary blood samples were 78.44, 72.01 and 79.62 for Whole Wheat™, Brown™ and Best of Both™ bread respectively. These three Albany Superior breads are also staple foods to some cultures and have lower GIs when compared to mealiemeal porridge, with or without sugar and traditional fermented Sorghum porridge (*ting*), and higher GIs than acid-added sorghum porridge.

Currently low-GI variations of bread are already available on the market (e.g. Sasco and Albany). The GI and the GL can be lowered by adding protein spreads, low-fat protein meat or cheese to the bread. Another way of lowering GI is by using a spread that contains fat, like peanut butter (which also contains protein). Fat and protein may modify the glycaemic response to a CHO food by slowing gastric emptying, due to the delay in CHO absorption (Welch *et al.*, 1987; Franz, 1997) and increasing insulin secretion, respectively (Nuttall *et al.*, 1984; Gannon *et al.*, 1988). Fat is also known to reduce jejunal motility and postprandial flow rates in the intestine, and hence decreases the glycaemic response (Perlstein *et al.*, 1997). According to Perlstein and co-workers (1997) protein may also increase the osmolarity of stomach content, thereby reducing the rate of gastric emptying.

Table 4.7. The mean GI for the three different Albany Superior™ breads using capillary blood

	Whole Wheat™	Brown™	Best of Both™
Mean	78.44	72.01	79.62
95%CI	[58.90; 97.90]	[51.10; 92.90]	[63.90; 95.30]
SD	41.56	44.73	33.54
Lower Quartile (Q1)	42.88	39.46	56.81
Median	82.54	59.20	72.97
Upper Quartile (Q3)	103.49	96.18	89.13
Minimum	12.41	15.26	33.94
Maximum	154.16	200.40	179.87

SD=standard deviation; CI=confidence interval for the mean GI

The 95% confidence intervals (CI) for the median difference between the GIs for paired data of Whole Wheat™ and Brown™ bread are [-26.1; 34.7]. The 95% CI for the median differences between the GIs for paired data of Whole Wheat™ and Best of Both™ bread are [-30.1; 32.6]. The 95% CI for the median differences between the GIs for paired data of Brown™ and Best of Both™ bread are [-42.0; 10.3]. Thus, the 95% CI for the median difference between the GIs for paired data shows that there is no statistically significant difference between the GIs the three different Albany Superior™ breads. However, a tendency exists for Brown™ bread to have a lower GI than Best of Both™ bread.

Foods that meet specific nutritional criteria and have been tested for their GI by an accredited laboratory may be eligible to label the GI value and give a short explanation near the nutrition information panel. This may inform the customer about the use of the GI concept in a balanced diet and challenge the food industry to reformulate and develop low-GI versions (for the benefit of persons with diabetes, cardiovascular disease and metabolic syndrome) and high-GI versions (for the benefit of physically active persons) of a specific product. If this is done the customers and health professionals will be able to make informed choices about the quality of CHO in foods (Venter *et al.*, 2003).

4.7 CONCLUSIONS

From the results of this study it can be concluded that:

- The mean GIs using capillary blood samples were 78.44, 72.01 and 79.62 for Whole Wheat™, Brown™ and Best of Both™ bread respectively. With 95% confidence, the conclusion can be made that the true mean GI for Whole wheat bread was between 58.9% and 97.9% (intermediate to high) for Brown bread between 51.1% and 92.9% (low to high) and for Best of Both bread between 63.9% and 95.3% (intermediate to high) measured in capillary blood.
- No statistically significant differences were found between the GIs of the three different Albany Superior™ breads.

- No statistically significant differences were found between the three different Albany Superior™ bread's AUCs and GIs using the median differences for paired data. However, a tendency exists for Brown™ bread to have a lower AUC and GI than Best of Both™ bread.
- Physiologic responses to the three breads varied widely between individuals.
- The three different Albany Superior™ breads fell between the intermediate and high categories as defined by the draft Regulations Relating to Labelling and Advertising of Foodstuffs in the Foodstuffs, Cosmetics and Disinfectants Act of 1972 (Act no.54 of 1972), which was published for comment on August 8, 2002.
- The prevalence of diabetes mellitus is still increasing in South Africa. Bread is consumed by many healthy South Africans as well as diabetic customers. Therefore, everybody will be able to make informed choices about the quality of CHO in foods, if the GI of breads that are bought, are known. According to the most recent international tables (Foster-Powell *et al.*, 2002) when looking at lowering the GI of breads, recipes in the industry could be changed by adding resistant starch, seeds (linseeds, sesame seeds etc.), soy and multigrain.

4.8 RECOMMENDATIONS

- It is recommended that subjects stay in a metabolic unit throughout a study of this nature to ensure that controlled conditions are maintained.
- It is recommended that the methodological guidelines as determined by the GI Task Force should be followed in all research investigating GI. In order to minimize variations in determining the GI of foods it is of utmost importance to continue further research in this field.
- As noted by Venter *et al.* (2005) it is important to inform patients and consumers that in using the GI to choose CHO foods it is a fact that physiological responses to a food may vary between individuals. Therefore, if food is indicated as having a high GI it does not mean that it will have a high GI in everybody. It thus is quite normal for a specific food to have a high GI in some individuals and a medium or even a low GI in others.
- The consumer should be educated on the fact that the GI of food is not the only factor that will determine whether the food should be included in the diet or not. It is, therefore, clear that current dietary guidelines need not be abolished when incorporating the GI concept in

practical advice. In table 4.8 suggestions are made by Slabber (2005) to incorporate the GI into current dietary advice.

***Table 4.8 Suggestions on how the GI may be incorporated into current dietary advice
(Slabber, 2005)***

- ☺ Always keep in mind that current dietary guidelines form the basis of any good diet and apply the GI concept alongside these guidelines
- ☺ Emphasise that a healthy diet is a diet high in CHO and low in fat
- ☺ Change the staple food in the diet to a low-GI alternative (cooled reheated mealie meal porridge, wholegrain and seed breads, wholegrain cereals)
- ☺ Include two low-GI foods daily
OR
Include one low-GI food at each meal
OR
Replace 50% of CHO in the diet with low-GI choices
- ☺ Spread CHO throughout the day
- ☺ For in-between snacks substitute high-GI foods with low-GI foods
- ☺ Adhere to the prescribed amount/portions/exchanges of starch or carbohydrate but make better choices (lower GI choices)
- ☺ Distinguish between low-GI CHO and fatty low-GI CHO. Fatty CHO should be avoided or used as 'sometimes' foods
- ☺ No food is good or bad – eating the low-GI way means eating a variety of foods
- ☺ High-GI foods do not have to be avoided completely. Some high-GI foods contain important nutrients like vitamins, minerals and phytochemicals. The golden rule is to combine high-GI foods with low-GI in the same meal
- ☺ Use dry beans, peas and lentils more often; also as thickening agent in soups, stews and curries
- ☺ Use low-oil or oil-free salad dressing containing vinegar to lower the GI of a meal
- ☺ Enjoy foods in moderate amounts. Avoid overindulgence in any food
- ☺ Low-fibre, low-GI choices include pasta, semolina, high-amylose rice like Basmati
- ☺ Sugar may constitute 10 % of daily energy (1.5-2.5 tablespoons/day)
- ☺ Plain low-fat yoghurt or artificially sweetened fat-free yoghurt are good low-GI choices
- ☺ Athletes who are too nervous to eat solid foods as a pre-event meal may take low-GI liquid supplements

- As stated by Slabber (2005) a range of low-, medium- and high-GI foods should rather be provided to customers than lists of numbers for GIs that may be confusing and complicating

dietary education. Suggestions of how higher-GI foods might be replaced with lower-GI alternatives are made in table 4.9.

Table 4.9 Substituting lower-GI foods for high-GI foods (Slabber, 2005)

Higher-GI foods	Lower-GI alternatives
⇒ Bread: whole-wheat, brown or white	⇒ Bread with lots of whole grains, seed loaf and seed buns
⇒ Mealiemeal porridge	⇒ Cooled reheated mealiemeal porridge and/or add dried beans, lentils, and chickpeas to porridge and/or add any vegetables to meal
⇒ Breakfast cereals: Puffed cereals, Weetbix (including sugar free), Shredded wheat	⇒ All Bran, Raisin Bran, High Fibre Bran, Oat Bran, Raw muesli
⇒ Biscuits and crackers: plain biscuits and crackers, rice cakes	⇒ Biscuits (low fat) with oats or oat bran, dried fruit, whole grains
⇒ Rice: Sticky white or brown rice	⇒ Basmati rice, crushed wheat ('stampkoring'), corn, green mealies, sweetcorn, lentils
⇒ Samp	⇒ Add dried beans or lentils
⇒ Potato	⇒ Sweet potato, pasta, baked beans
⇒ Muffins/scones: white or brown	⇒ Muffins or scones (low fat) made with oat bran, oats, fruit, dried fruit
⇒ Fruit: watermelon, tropical fruits (paw-paw, mangos, litchis, melons, bananas)	⇒ Citrus fruits (oranges, lemons, naartjies, grapefruit), apples, pears, apricots, peaches, plums, kiwi, sultanas, cherries, grapes
NOTE: the riper the banana the higher the GI	NOTE: the more tart/sharp/acid the fruit the lower the GI

- Lastly, for labelling purposes as noted by Pieters and Jerling (2005) it is recommended that the GI is presented as a mean (dot) with 95% CI (square). When labelling packaging, neutral colours should be used. The colour red should not be used to signify a high GI, as red commonly indicates, danger, and customers might interpret such products as unhealthy. That is an important misconception, which has the potential to be misused in the marketing of foods. This is in agreement with the philosophy that there are no good or bad foods, only good or bad diets.

BIBLIOGRAPHY

AGINSKY, J. VISSER, M.E. and LEVITT, N.S. 2000. The inter- and intra-individual variation in glycemic response to glucose and white bread in healthy male students. Journal of the European Medical and Dental Association, vol. 5, pp. 53.

AGUS, M.S., SWAIN, J.F., LARSON, C.L., ECKERT, E.A., LUDWIG, D.S. 2000. Dietary composition and physiological adaptations to energy restriction. American Journal of Clinical Nutrition, vol. 71, pp.901-907.

ALBANY BAKERIES. 2006. The importance of bread in the diet- advertisement. South African Journal of Clinical Nutrition, vol. 19, no. 3, p.117.

AMERICAN DIABETES ASSOCIATION. 1998. Position statement: nutrition recommendations and principles to people with diabetes mellitus. Diabetes Care, vol. 21, pp. 32S-35S.

AMERICAN DIABETES ASSOCIATION. 2001. Position statement: nutrition recommendations and principles to people with diabetes mellitus. Diabetes Care, vol. 24, pp. 44S-47S.

AMERICAN DIABETES ASSOCIATION. 2003. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care, vol. 26, suppl.1, pp. S51-S61.

AMERICAN DIETETIC ASSOCIATION (ADA). 2002. Upcoming DRI report: new ways of defining fibre. Journal of the American Dietetic Association, vol.102, no. 4, p. 468.

ARMAND, M., HAMOSH, M. and DiPALMA, J.S. 1995. Dietary fat modulates lipase activity in healthy humans. American Journal of Clinical Nutrition, vol. 62, pp. 74-80.

AUGUSTIN, L.S., DAL MASO, L., LA VECCHIA, C., PARPINAL, M., NEGRI, E., VACCARELLA, S., KENDALL, C.K.W., JENKINS, D.J.A. and FRANCESCHI, S. 2001. Dietary glycemic index and glycemic load in breast cancer risk: a case-control study. Annals of Oncology, vol. 12, pp.1533-1538.

AUGUSTIN, L.S., FRANCESCHI, S., JENKINS, D.J.A., KENDALL, C.W.C. and LA VECCHIA, C. 2002. Glycemic index in chronic disease: a review. European Journal of Clinical Nutrition, vol. 56, pp.1049-1071.

AUGUSTIN, L.S.A., POLESEL, J., BOSETTI, C. and KENDALL, C.W., LA VECCHIA, C., PARPINEL, M., CONTI, E., MONTELLA, M., FRANCESCHI, S., JENKINS, D.J. and DAL MASO, L. 2003a. Dietary glycemic index, glycemic load and ovarian cancer risk: a case-control study in Italy. Annals of Oncology, vol. 14, pp.78-84.

AUGUSTIN, L.S.A., GALLUS, S. BOSETTI, C., LEVI, F., NEGRI, E., FRANCESCHI, S., DAL MASO, L., JENKINS, D.J., KENDALL, C.W. and LA VECCHIA, C. 2003b. Glycemic index and glycemic load in endometrial cancer. International Journal of Cancer, vol.105, pp. 404-407.

AUGUSTIN, L.S.A., GALLUS, S., FRANCESCHI, S., NEGRI, E., JENKINS, D.J., KENDALL, C.W., DAL MASO, L., TALAMINI, R. and LA VECCHIA, C. 2004a. Glycemic index and load and risk of upper aero-digestive tract neoplasms (Italy). Cancer Causes Control, vol. 14, pp. 657-662.

AUGUSTIN, L.S.A., GALLUS, S., NEGRI, E., LA VECCHIA, C. 2004b. Glycemic index and load and risk of gastric cancer. Annals of Oncology, vol. 15, pp. 581-584.

AUGUSTIN, L.S., GALEONE, C., DAL MASO, L., PELUCCHI, C., RAMAZZOTTI, V., JENKINS, D.J., MONTELLA, M., TALAMINI, R., NEGRI, E., FRANCESCHI, S. and LA VECCHIA, C. 2004c. Glycemic index and load and risk of prostate cancer. International Journal of Cancer, vol. 112, pp. 446-450.

BARAKAT, H.A., VADLAMUDI, S., MACLEAN, P., MACDONALD, K. and PORIES, W.J. 1996. Lipoprotein metabolism in non-insulin dependent diabetes mellitus. Journal of Nutrition and Biochemistry, vol. 7, pp. 586-598.

BARCLAY, A.W., BRAND-MILLER, J.C. and WOLEVER, T.M.S. 2005. Glycemic index, glycemic load, and glycemic response are not the same. Diabetes Care, vol. 28, no. 7, p. 1839.

BEEBE, C. 1999. Diets with a low glycemic index: not ready for practice yet! Nutrition Today, vol. 34, pp. 82-86.

BEHALL, K.M., SCHOLFIELD, D.J. and HALLFRISCH, J. 1999. The effect of particle size of whole-grain flour on plasma glucose, insulin, glucagons and thyroid stimulating hormone in humans. American Journal of Clinical Nutrition, vol. 67, suppl., pp. 772S-778S.

BELOW, P., MORA-RODRIGUES, R. and GONZALEZ -ALONSO, J. and COYLE, E.P. 1995. Fluid and carbohydrate ingestion independently improve performance during 1 h of intense cycling. Medicine and Science in Sports and Exercise, vol. 27, pp. 200-210.

BERTELSEN, J. CHRISTIANSEN, C., THOMSEN, C., PAULSEN, P.L., VESTERGAARD, S., STEINOV, A., RASMUSSEN, L.H., RASMUSSEN, O. and HERMANSEN, K. 1993. Effect of meal frequency on blood glucose, insulin, and free fatty acids in NIDDM subjects. Diabetes Care, vol. 16, pp.4-7.

BESSENER, D.H. 2001. The role of carbohydrate in insulin resistance. Journal of Nutrition, vol. 131, suppl., pp. 2782S-2786S.

BLAAU, R. 2003. Health benefits of the glycaemic index. South African Journal of Clinical Nutrition, vol.16, no. 1, pp. 7-8.

BRAND, J., NICHOLSON, P.L., THORBURN, A.W. and TRUSWELL, A.S. 1985. Food processing and the glycaemic index. American Journal of Clinical Nutrition, vol. 42, pp. 192-196.

BRAND-MILLER, J., FORSTER-POWELL, K., COLAGIURI, S. 1996. The GI factor. Sydney: Hodder and Stoughton.

BRAND-MILLER, J. and FOSTER-POWELL, K. 1999. Diets with a low GI: from theory to practice. Nutrition Today, vol. 34, no. 2, pp. 64-71.

BRAND-MILLER, J. and GILBERTSON, H. 2001. Practical aspects of meal planning using the glycaemic index. FAO/Danone Vitapole Workshop. Glycaemic index and health: the quality of the evidence. Brandol, France: Danone Vitapole.

BRAND-MILLER, J.C., HOLT, S.H.A., PAWLAK, D.B. and MCMILLAN, J. 2002. Glycemic index and obesity. American Journal of Clinical Nutrition, vol. 76, suppl., pp. 281S-285S.

BRAND-MILLER, J., LOBBEZOO, I. 1994. Replacing starch with sucrose in a high glycaemic breakfast cereal lowers glycaemic and insulin responses. European Journal of Clinical Nutrition, vol. 48, pp. 749-752.

BRAND-MILLER, J., PANG, E. and BROOMHEAD, L. 1995. The glycaemic index of foods containing sugar: comparison naturally-occurring v. added sugars. British Journal of Nutrition, vol. 73, pp. 613-623.

BRAND-MILLER, J.C., THOMAS, M., SWAN, V., AHMAD, Z.I., PETOCZ, P. and COLAGIURI, S. 2003. Physiological validation of the concept of glycemic load in lean young adults. Journal of Nutrition, vol. 133, pp. 2728-2732.

BRIGHENTI, F. CASTELLANI, G., BENINI, L., LEOPARDI, E., CROVETTI, R. and TESTOLIN, G. 1995. Effect of neutralized and native vinegar on blood glucose and acetate responses to a mixed meal in healthy subjects. European Journal of Clinical Nutrition, vol. 49, pp. 242-247.

BROUNS, F., BJORCK, I., FRAYN, K.N., GIBBS, A.L., LANG, V., SLAMA, G. and WOLEVER, T.M.S. 2005. Glycaemic index methodology, Nutrition Research Reviews, vol. 18, pp. 145-171.

BRUNING, P.F., BONFRER, J.M.G., VAN NOORD, P.A.H., HART, A.A.M., DE JONG-BAKKER, M. and NOOIJEN, W.J. 1992. Insulin resistance and breast-cancer risk. International Journal of Cancer, vol. 52, pp. 511-516.

BURKE, L. GREGORY, R.C. and HARGRAEVES, M. 1998. Glycemic index – a new tool in sports nutrition? International Journal of Sports Nutrition, vol. 8, pp. 401-415.

BURKE, B.J., HARTOG, K.W. and HOOPER, S. 1982. Assessment of the metabolic effects of dietary carbohydrates and fibre by measuring urinary excretion of C-peptide. Human Nutrition and Clinical Nutrition, vol. 36, pp. 373-380.

BUYKEN, A.E., TOELLER, M., HEITKAMP, G., KARAMANOS, B., ROTTIERS, R., MUGGEO, M. and FULLER, J.H. 2001. Glycemic index in the diet of European outpatients with type 1 diabetes: relations to glycosylated hemoglobin and serum lipids. American Journal of Clinical Nutrition, vol. 73, pp. 574-581.

BYRNES, S.E., BRAND-MILLER, J.C. and DENYER, G.S. 1995. Amylopectin starch promotes the development of insulin resistance in rats. Journal of Nutrition, vol. 125, pp. 1430-1437.

CAMPBELL, GLOWCZESKI, T. and WOLEVER, T.M. 2003. Controlling subjects' prior diet and activities does not reduce within-subject variation of postprandial glycemic responses to foods. Nutrition Research, vol. 23, pp. 621-629.

CARTER, J.M., JEUKENDRUP, A.E. and JONES, D.A. 2004. The effect of carbohydrate mouth rinse on 1-h cycle time trail performance. Medicine and Science in Sports and Exercise, vol. 36, pp. 2107-2111.

CASTILLO, M.J., SCHEEN, A.J., JANDRIAN, B. and LEFEBVRE, P.J. 1994. Relationship between metabolic clearance rate of insulin and body mass index in a female population ranging from anorexia nervosa to severe obesity. Obesity Related Metabolic Disorders, vol. 18, pp. 47-53.

COGGAN, A.R. and COYLE, E.F.1991. Carbohydrate ingestion during prolonged exercise: effects on metabolism and performance. Exercise Sports Science Review, vol. 19, pp.1-40.

COLE T.J. 1997. Sampling, study size, and power. In: Design Concepts in Nutritional Epidemiology. Ed. by Margetts, B.M. and Nelson, M. 2nd ed. Oxford: Oxford University Press, pp. 64-86.

COLLIER, G.R., WOLEVER, T.M.S., WONG, G.S. and JOSSE, R.G. 1986. Predictions of glycaemic response to mixed meals in noninsulin-dependent diabetic subjects. American Journal of Clinical Nutrition, vol. 44, pp. 349-352.

COLLINS, P., WILLIAMS, C. and MACDONALD, I. 1981. Effect of cooking on serum glucose and insulin responses to starch. British Medical Journal, vol. 282, pp. 1032-1033.

CONCEICAO DE OLIVEIRA, M., SICHIERI, R. and SANCHEZ MOURA, A. 2003. Weight loss associated with a daily intake of three apples or three pears among overweight women. Nutrition, vol. 19, pp. 253-256.

COULSTON, A.M., HOLLENBECK, C.B. and REAVEN, G.M. 1984a. Utility of studies measuring glucose and insulin responses to various carbohydrate-containing foods. American Journal of Clinical Nutrition, vol. 39, pp.163-165.

COULSTON, A.M., HOLLENBECK, C.B., SWASLOCKI, A.L. and REAVEN, G.M. 1987. Effect of source of dietary carbohydrate on plasma glucose and insulin responses to mixed meals in subjects with NIDDM. Diabetes Care, vol.10, pp. 395-400.

COULSTON, A.M. and REAVEN, G.M. 1997. Much ado about (almost) nothing. Diabetes Care, vol. 20, pp. 241-243.

COYLE, E.F. 1991. Timing and method of increased carbohydrate intake to cope with heavy training, competition and recovery. Journal of Sport Science, vol. 9, (suppl.), pp. 29-52.

DALY, M.E., MILLER, J.C.B. and DENYER, G.S. 1997. Dietary carbohydrates and insulin sensitivity: a review of the evidence and clinical implications. American Journal of Clinical Nutrition, vol. 66, pp. 1072-1085.

DANSTAN, D.W., DALY, R.M., OWEN, N. JOLLEY, D., DE COURTEN, M., SHAW, J. and ZIMMET, P. 2002. High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. Diabetes Care, vol. 25, pp. 1729-1736.

DEL GIUDICE, M.E., FANTUS, I.G., EZZAT, S., MACKEOWN-EYSSEN, G., PAGE, D. and GOODWIN, P.J. 1998. Insulin and related factors in premenopausal breast cancer risk. Breast Cancer Research Treatment, vol. 47, pp. 111-120.

DEPARTMENT OF HEALTH. 2001. SASSO Draft Guidelines for the Prevention and Management of Overweight and Obesity.

ELLIS, P.R., DAWOUD, F.M. and MORRIS, E.R. 1991. Blood glucose, plasma insulin and sensory responses to guar-containing wheat breads: effects of molecular weight and particle size of guar gum. British Journal of Nutrition, vol. 66, pp. 363-379.

ENGLYST, K.N., ENGLYST, H.N. HUDSON, G.J., COLE, T.J. and CUMMINGS, J.H. 1999. Rapidly available glucose in foods: an *in vitro* measurement that reflects the glycaemic response. American Journal of Clinical Nutrition, vol. 69, pp. 448-454.

ENGLYST, H.N. and HUDSON, G.J. 2000. Carbohydrates. In: Human Nutrition Dietetics. Ed. by Garrow, J.S., James, W.P.T. and Ralph, A. 10th Ed. Philadelphia: Churchill Livingstone.

ENGLYST, H.N., KINGMAN, S.M. and CUMMINGS, J.H. 1992. Classification and measurement of nutritionally important starch fractions. European Journal of Clinical Nutrition, vol. 46, suppl. 2, pp. S33-S50.

ENGLYST, H.N., KINGMAN, S.M., HUDSON, G.J. and CUMMINGS, J.H. Measurement of resistant starch *in vitro* and *in vivo*. British Journal of Nutrition, vol. 75, pp. 749-755.

ENGLYST, H.N., TROWELL, H., SOUTHGATE, D.A.T. and CUMMINGS, J.H. 1987. Dietary fibre and resistant starch. American Journal of Clinical Nutrition, vol. 46, pp. 873-874.

FAO/WHO EXPERT CONSULTATION. 1998. Carbohydrates in human nutrition: Report of a Joint FAO/WHO Expert Consultation. Rome, Italy, 14-18 April 1997.

FORD, E.S. and LIU, S. 2001. Glycemic index and serum high-density lipoprotein cholesterol concentration among US adults. Archives of Internal Medicine, vol. 161, pp. 572-576.

FOSTER-POWELL, K., HOLT, S.H.A. and BRAND-MILLER, J.C. 2002. International table of glycaemic index and glycaemic load values. American Journal of Clinical Nutrition, vol. 76, pp. 5-56.

FOSTER-POWELL, K. and MILLER, J.B. 1995. International tables of glycemic index. American Journal of Clinical Nutrition, vol. 62, pp. 87S-93S.

FRANCESCHI, S., DAL MASO, L., AUGUSTIN, L., NEGRI, E., PARPINNEL, M., BOYLE, P., JENKINS, D.J. and LA VECCHIA, C. 2001. Dietary glycemic load and colorectal cancer risk. Annals of Oncology, vol. 12, pp. 173-178.

FRANZ, M.J. 1997. Protein: metabolism and effect on blood glucose levels. Nutrition Update, vol. 23, no. 6, pp. 643-648.

FRANZ, M.J. 1999. In defense of the American Diabetes Association's recommendations on the glycemic index. Nutrition Today, vol. 34, pp. 25-30.

FRANZ, M.S. 2000. Medical nutrition therapy for diabetes mellitus and hypoglycemia of nondiabetic origin. In Krause's Food, Nutrition, & Diet Therapy. Ed. by Mahan, L.K. and Escott-Stump, S. 10th ed. Philadelphia: W.B. Saunders.

FRANZ, M.S. 2004. Medical nutrition therapy for diabetes mellitus and hypoglycemia of nondiabetic origin. In Krause's Food, Nutrition, & Diet Therapy. Ed. by Mahan, L.K. and Escott-Stump, S. 11th ed. Philadelphia: W.B. Saunders.

FROST, G. and DORNHORST, A. 2000. The relevance of glycaemic index to our understanding of dietary carbohydrates. Diabetic Medicine, vol. 17, pp. 336-345.

FROST, G., LEEDS, A.A., DORE, C.J., MADEIROS, S., BRANDING, S. and DORNHORST, A. 1999. glycaemic index as determinant of serum HDL-cholesterol concentration. Lancet, vol. 353, pp. 1029-1030.

FROST, G., LEEDS, A., TREW, G., MARGARA, R. and DORNHORST, A. 1998. Insulin sensitivity in women at risk of coronary heart disease and the effect of a low glycaemic diet. Metabolism, vol. 47, pp. 1245-1251.

GALANIS, D.J., KOLONEL, L.N., LEE, J. and LE MARCHAND, L. 1998. Anthropometric predictors of breast cancer incidence and survival in a multi-ethnic cohort of female residents of Hawaii. United States. Cancer Causes Control, vol. 9, pp. 217-224.

GANNON, M.C., NUTTALL, F.Q., NEIL, B.J. and WESTPHAL, S.A. 1988. The insulin and glucose responses to meals of glucose plus various proteins in type II diabetic subjects. Metabolism, vol. 37, pp. 1081-1088.

GARG, A. 1996. Insulin resistance in the pathogenesis of dyslipidemia. Diabetes Care, vol. 19, pp. 387-389.

GILBERTSON, H.R., THORBURN, A.W., BRAND-MILLER, J.C., CHONDROS, P. and WERTHER, G.A. 2003. Effect of low-glycemic index dietary advice on dietary quality and food choice in children with type 1 diabetes. American Journal of Clinical Nutrition, vol. 77, pp. 83-90.

GIOVANNUCCI, E. 1995. Insulin and colon cancer. Cancer Causes Control, vol. 6, pp. 164-179.

GLYCAEMIC INDEX (GI) TASK FORCE. 2002. Standard operating procedure for the determining of the glycaemic index. (Unpublished report.)

GRANFELDT, Y., LILJEBERG, H., DREWS, A., NEWMAN, R. and BJÖRCK, I. 1994. Glucose and insulin responses to barley products: influence of food structure and amylose-amylopectin ratio. American Journal of Clinical Nutrition, vol. 59, pp. 1075-1082.

GRESSE, A. and VORSTER, H.H. 1992. The glycaemic index and second meal effect of a typical African meal in black non-insulin-dependent diabetic subjects. South African Journal of Food Science and Nutrition, vol. 4, no. 3, pp. 64-69.

GUEZENNEC, C.Y., STABIN, P., DUFOREZ, F., MERINO, D., PERONNET, F. and KOZIET, J. 1989. Oxidation of corn starch, glucose and fructose ingested before exercise. Medicine and Science in Sports and Exercise, vol. 21, no. 1, pp.45-50.

GUILLON, F. and CHAMP, M. 2000. Structural and physical properties of dietary fibres, and consequences of processing on human physiology. Food Research International, vol. 33, pp. 233-245.

GULLIFORD, M.C., BICKNELL, E.J. and SCARPELLO, J.H. 1989. Differential effect of protein and fat ingestion on blood glucose responses to high- and low-glycemic-index carbohydrates in noninsulin diabetic subjects. American Journal of Clinical Nutrition, vol. 50, pp. 773-777.

HARDER, H., DINESEN, B. and ASTRUP, A. 2004. The effect of a rapid weight loss on lipid profile and glycemic control in type 2 diabetic patients. International Journal of Obesity Related Metabolic Disorders, vol. 28, pp. 180-182.

HODGE, A.M., ENGLISH, D.R., O'DEA, K. and GILES, G.G. 2004. Glycemic index and dietary fibre and the risk of type 2 diabetes. Diabetes Care, vol. 27, no.11, pp. 2701-2706.

HOLT, S.H.A. and BRAND-MILLER, J. 1994. Particle size, satiety and the glycaemic response. European Journal of Clinical Nutrition, vol. 48, pp. 496-502.

HOROWITZ, M., EDELBROEK, M.A.L., WISHART, J.M. and STRAATHOF, J.W. 1993. Relationship between oral glucose and gastric emptying in normal healthy subjects. Diabetologia, vol. 36, no. 9, pp. 857-862.

JERLING, J.C., PIETERS, M., LESSING, M.C., NELL, T.A. and VAN HEERDEN, Y. 2002. The determination of the GI of three oats porridges. Potchefstroom. p.12 (Unpublished dissertation).

JENKINS, D.J.A. 1984. Dietary carbohydrates and their glycemic responses. Journal of the American Medical Association, vol. 252, pp. 2829-2831.

JENKINS, D. and JENKINS, A. 1987a. The glycaemic index, fibre, and the dietary treatment of hypertriglyceridemia and diabetes. Journal of the American College of Nutrition, vol. 6, pp. 11-17.

JENKINS, D.J.A., KENDALL, C.W.C., AUGUSTIN, L.S.A., VUKSAN, V. and SMITH, U. 2002. Glycemic index: overview of implications in health and disease. American Journal of Clinical Nutrition, vol. 76, pp. 266S-273S.

JENKINS, D.J.A., OCANA, A., JENKINS, A.L., WOLEVER, T.M.S., VUKSAN, V., KATZMAN, L., HOLLANDS, M., GREENBERG, G., COREY, P., PATTEN, R., WONG, G. and JOSSE, R.G. 1992. Metabolic advantages of spreading the nutrient load: effect of meal frequency in non-insulin-dependent diabetes. American Journal of Clinical Nutrition, vol. 55, pp. 461-467.

JENKINS, D.J.A, WOLEVER, T.M.S., BUCKLEY, G., LAM, K.Y., GIUDICI, S., KALMISKY, J., JENKINS, A.L., PATTEN, R.L., BIRD, J., WONG, G.S. and JOSSE, R.G. 1988a. Low-glycemic index starchy foods in the diabetic diet. American Journal of Clinical Nutrition, vol. 48, pp. 248-254.

JENKINS, D.J.A., WOLEVER, T.M.S., COLLIER, G.R., OCANA, A., RAO, A.V., BUCKLEY, G., LAM, Y., MAYER, A. and THOMPSON, L.U. 1987b. Metabolic effects of a low-glycemic-index diet. American Journal of Clinical Nutrition, vol. 46, pp. 968-975.

JENKINS, D.J., WOLEVER, T.M., JENKINS, A.L., THORNE, M.J., LEE, R., KALMUSKY, J., REICHERT, R. and WONG, G.S. 1983. The glycaemic index of food tested in diabetic patients; a new basis for carbohydrate exchange favouring the use of legumes. Diabetologia, vol. 24, pp. 257-264.

JENKINS, D.J., WOLEVER, T.M., JENKINS, A.L., GIORDANO, C., GIUDICI, S., THOMPSON, L.U., KALMUSKY, J., JOSSE, R.G. and WONG, G.S. 1986. Low glycaemic response to traditionally processed wheat and rye products: bulgur and pumpernickel bread. American Journal of Clinical Nutrition, vol. 43, pp. 516-520.

JENKINS, D.J.A., WOLEVER, T.M.S., RAO, A.V., HEGELE, R.A., MITCHELL, S.J., RANSOM, T.P.P. BOCTOR, D.L., SPADAFORA, P.J., JENKINS, A.L., MEHLING, C., RELLE, L.K., CONNELLY, P.W., STORY, J.A., FURUMOTO, E.J., COERY, P. and WÜRSCH, P. 1993. Effect on blood lipids of very high intakes of fibre in diets low in saturated fat and cholesterol. New England Journal of Medicine, vol. 329, pp. 21-29.

JENKINS, D.J.A., WOLEVER, T.M.S., TAYLOR, R.H., BARKER, H.M. and FIELDEN, H. 1980b. Exceptionally low blood glucose response to dried beans: comparison with other carbohydrate foods. British Medical Journal, vol. 281, pp. 578-580.

JENKINS, D.J.A., WOLEVER, T.M.S., TAYLOR, R.H., BARKER, H., FIELDEN, H., BALDWIN, J.M., BOWLING, A.C., NEWMAN, H.C., JENKINS, A.L. and GOFF, D.V. 1981. Glycaemic index of foods: a physiological basis for carbohydrate exchange. American Journal of Clinical Nutrition, vol. 34, pp. 362-366.

JENKINS, D.J.A., WOLEVER, T.M.S., VUKSAN, V., BRIGHENTI, F., CUNNANE, S.C., RAO, A.V., JENKINS, A.C., BUCKLEY, G., PATTEN, R., SINGER, W., COREY, P. and JOSSE, R.G. 1989. Nibbling versus gorging: metabolic advantages of increased meal frequency. New England Journal of Medicine, vol. 321, pp. 929-934.

JENTJENS, R.L. and JEUKENDRUP, A.E. 2003. Determinants of post-exercise glycogen synthesis during short-term recovery. Sports Medicine, vol. 33, pp. 117-144.

JENTJENS, R.L., VAN LOON, L.J.C., MANN, C.H., WAGENMAKERS, A.J.M. and JEUKENDRUP, A.E. 2001. Addition of protein and amino acids to carbohydrates does not enhance post-exercise muscle glycogen synthesis. Journal of Applied Physiology, vol. 91, pp. 839-846.

JEUKENDRUP, A.E., JENTJENS, R. 2000. Oxidation of carbohydrate feedings during prolonged exercise: current thoughts, guidelines and directions for future research. Sports Medicine, vol. 29, pp. 407-425.

JOANNIC, J., AUBOIRON, S., RAISON, J., BASSDEVANT, A., BORNET, F. and GUY-GRAND, B. 1997. How the degree of unsaturation of dietary fatty acids influence the glucose insulin responses to different carbohydrates in mixed meals. American Journal of Clinical Nutrition, vol. 65, pp. 1427-1433.

JONES, P.J., LEITCH, C.A. and PEDERSON, R.A. 1993. Meal frequency effects of plasma hormone concentrations and cholesterol synthesis in humans. American Journal of Clinical Nutrition, vol. 57, pp. 868-874.

JONES, I.R., OWENS, D.R., WILLIAMS, S., RYDER, R.E.J., BIRTWELL, A.J., JONES, M.K., GICHERNU, K. and HAYES, T.M. 1983. Glycosylated serum albumin: an intermediate index of diabetic control. Diabetes Care, vol. 6, pp. 501-503.

KAAKS, R. 1996. Nutrition, hormones, and breast cancer: is insulin the missing link? Cancer Causes Control, vol. 7, pp. 605-625.

KATANAS, H. 1999. Diets with a low-glycemic index are ready for practice. Nutrition Today, vol. 34, pp. 87-88.

KOH, E.T. and OWEN, W.L. 2000. Measuring Research Variables. In Introduction to Nutrition and Health Research. Ed. by Kluwer Academic Publishers: London.

KRUGER, L., SLABBER, M., JOUBERT, G., et al. (in press). The intra- and inter individual variation of blood glucose response to white bread and glucose as determined in patients with type 2 diabetes mellitus. South African Journal of Clinical Nutrition.

LÉCLERE, C.J., CHAMP, M., BOILLOT, J., GERARD, G., LECANNU, G., MOLIS, C., BORNET, F., KREMPF, M., DELORT-LAVAL, J. and GALMICHE, J.-P. 1994. Role of viscous gums in lowering the glycemic response after a solid meal. American Journal of Clinical Nutrition, vol. 59, pp. 914-921.

LEEDS, A.R. 2002. Glycemic index and heart disease. American Journal of Clinical Nutrition, vol. 76, no.1 , pp. 286S-289S.

LILJEBERG, H.G.M. and BJÖRCK, I. 1996a. Delayed gastric emptying rate as a potential mechanism for lowered glycemia after eating sourdough bread: studies in humans and rats using test products with added organic acids or an organic salt. American Journal of Clinical Nutrition, vol. 64, pp. 886-893.

LILJEBERG, H.G.M., GRANFELDT, Y.E. and BJÖRCK, I.M.E. 1996b. Products based on a high fibre barley genotype, but not on common barley or oats, lower postprandial glucose and insulin responses in healthy humans. Journal of Nutrition, vol. 126, no. 2, pp. 458-466.

- LIU, S., WILLETT, W.C., STAMPFER, M.J., HU, F.B., FRANZ, M., SAMPSON, L., HENNEKENS, C.H. and MANSON, J.E. 2000. A prospective study of dietary glycemic load, carbohydrate intake and risk of coronary heart disease in US women. American Journal of Clinical Nutrition, vol. 71, no. 6, pp. 1455-1461.
- LU, Z.X., WALKER, K.Z., MUIR, J.G., MASCARA, T. and O'DEA, K. 2000. Arabinoxylan fibre, a byproduct of wheat flour processing, reduces the postprandial glucose response in normal glycaemic subjects. American Journal of Clinical Nutrition, vol. 71, pp. 1123-1128.
- LUDWIG, D.S. 2000. Dietary glycaemic index and obesity. Journal of Nutrition, vol. 130, suppl. 2, pp. 280S-283S.
- LUDWIG, D.S. 2002. The glycaemic index. Physiological mechanisms relating to obesity, diabetes and cardiovascular disease. Journal of the American Medical Association, vol. 287, pp. 2414-2423.
- LUDWIG, D.S. 2002. The glycaemic index. Journal of the American Medical Association, vol. 287, pp. 2414-2423.
- MADIGAN, M.P., TROISI, R., POTISCHMAN, N. et al. 1998. Serum hormone levels in relation to reproductive and lifestyle factors in postmenopausal women (United States). Cancer Causes Control, vol. 9, pp. 199-207.
- MAVRI, A., GUZIC-SALOBIR, B., SALOBIR-PAJNIC, B., KEBER, I., STARE, J. and STEGNAR, M. 2001. Seasonal variation of some metabolic and haemostatic risk factors in subjects with and without coronary artery disease. Blood Coagulation & Fibrinolysis, vol. 12, no.5, pp. 359-365.
- MBENYANE, X.G. 1997. The glycaemic index of indigenous South African foods. Ph.D thesis, Potchefstroom: University for Christian Higher Education: pp. 235.
- MCKEOWN-EYSEN, G. 1994. Epidemiology of colorectal cancer revisited: are serum triglycerides and/or plasma glucose associated with risk? Cancer Epidemiology, Biomarkers & Prevention, vol. 3, pp. 687-695.
- MCKEOWN, N.M., MEIGS, J.B., LIU, S., SALTZMAN, E., WILSON, P.W.F. and JACQUES, P.F. 2004. Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring cohort. Diabetes Care, vol. 27, pp. 538-546.

MEYER, K.A., CONIGRAVE, K.M., CHU, N.F., RIFAI, N., SPIEGELMAN, D., STAMPFER, M.J. and RIMM, E.B. 2003. Alcohol consumption patterns and HbA1c, C-peptide and insulin concentrations in men. Journal of American College of Nutrition, vol. 22, pp. 185-194.

MEYER, K.A., KUSHI, L.H., JACOBS, D.R., SLAVIN, J., SELLERS, T.A. and FOLSOM, A.R. 2000. Carbohydrates, dietary fibre, and incidence of type 2 diabetes in older women. American Journal of Clinical Nutrition, vol. 71, pp. 921-930.

MILLER, J.C. 1994. Importance of glycemic index in diabetes. American Journal of Clinical Nutrition, vol. 59, suppl. 3, pp. 747S-752S.

MIZOCK, B.A. 1995. Alterations in carbohydrate metabolism during stress: a review of the literature. American Journal of Medicine, vol. 98, no. 1, pp. 75-84.

MUNARI, F., ALBERTO, C., WILLIAM, B.P., ANDRACA, A., RAUL, C. and MOISES, C. 1998. Lowering glycaemic index of food by acarbose and plantago Psyllium mucilage. Archives of Medical Research, vol. 29, pp. 137-142.

NELL, T.A. 2001. The variation and application of the glycaemic index of foods. Ph.D thesis, Potchefstroom: University for Christian Higher Education: pp. 1-145.

NELL, T., VENTER, C., VORSTER, H., BOTES, I. and STEYN, F. 2003. Intra- and inter-individual variation in glucose response to white bread and oral glucose in healthy women. The South African Journal of Clinical Nutrition, vol. 16, no. 2, pp. 58-64.

NEWCOMB, P.A., KLEIN, R., KLEIN, B.E., HAFFNER, S., MARES-PERLMAN, J., CRUICKSHANKS, K.J. and MARCUS, P.M. 1995. Association of dietary and life-style factors with sex hormones in postmenopausal women. Epidemiology, vol. 6, pp. 318-321.

NUTTALL, F.Q., MOORADIAN, A.D., BILLINGTON, M.C. and KREZOWSKI, P. 1984. Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. Diabetes Care, vol. 7, pp. 465-470.

OPPERMAN, M., VENTER, C.S., OOSTHUIZEN, W. 2005. Some health benefits of low-glycaemic index diets – a systematic review. The South African Journal of Clinical Nutrition, vol. 18, no. 3, pp. 214-221.

PAWLAK, D.B., EBBELING, C.B. and LUDWIG, D.S. 2002. Should obese patients be counseled to follow low-glycaemic index diet? Yes. Obesity Reviews, vol. 3, pp. 235-243.

PEACH, H.G. and BARNETT, N.E. 2001. Helicobacter pylori infection and fasting plasma glucose concentration. Journal of Clinical Pathology, vol. 54, 466-469.

PERLSTEIN, R.W.J., WILLCOX, J., HINES, C. and MILSAVLJEVIC, M. 1997. Dietitians Association of Australia review paper: Glycaemic index in diabetes management. Australian Journal of Nutrition and Dietetics, vol. 54, no. 2, pp. 57-63.

PI-SUNYER, F.X. 2002. Glycemic index and disease. American Journal of Clinical Nutrition, vol. 76, pp. 290S-298S.

PIEHL, K. 1974. Time course for refilling glycogen stores in human muscle fibres following exercise-induced glycogen depletion. Acta Physiologica Scandanavica, vol. 90, pp. 297-302.

PIETERS, M. and JERLING, J.C. 2005. Measuring the glycaemic index – consensus and issues of debate. South African Journal of Clinical Nutrition, vol.18, no.3, pp. 232-236.

PI-SUNYER, F.X. 2002. Glycaemic index and disease. American Journal of Clinical Nutrition, vol. 76 suppl., pp. 290S-298S.

RABEN, A. 2002. Should obese patients be counseled to follow a low-glycaemic index diet? No. Obesity Reviews, vol. 3, pp. 245-266.

RABEN, A., TAGLIABUE, A., CHRISTENSEN, N.J., MADSEN, J., HOLST, J.J. and ASTRIP, A. 1994. Resistant starch: the effect on postprandial glycemia, hormonal response and satiety. American Journal of Clinical Nutrition, vol. 60, pp. 544-551.

RASMUSSEN, O.W., GREGERSEN, S., DORUP, J. and HERMANSEN, K. 1992. Day to day variation of blood glucose and insulin responses in type 2 diabetic subjects after starch-rich meal. Diabetes Care, vol. 15, pp. 522-524.

RICCARDI, G. and RIVELLESE, A.A. 2000. Dietary treatment of the metabolic syndrome – the optimal diet. British Journal of Nutrition, vol. 83, suppl., pp. S143-S155.

RIZKALLA, S.W., BELLISLE, F. and SLAMA, G. 2002. Health benefits of low glycaemic index foods, such as pulses, in diabetic patients and healthy individuals. British Journal of Nutrition, vol. 88, suppl. 3, pp. S255-S262.

ROBERTS, S.2000. High-glycaemic index foods, hunger and obesity: is there a connection? Nutrition Reviews, vol. 58, pp. 163-169.

SALMERON, J., ASCHERIO, A., RIMM, E.B., COLDITZ, G.A., SPEIGELMAN, D., JENKINS, D.J. ST5AMPFER, M.J., WING, A.L. and WILLET, W.C. 1997a. Dietary fibre, glycaemic load, and risk of NIDDM in men. Diabetes Care, vol. 20, no. 4, pp. 545-550.

SALMERON, J., MANSON, J.E., STAMPFER, M.J. COLDITZ, G.A., WING, A.L. and WILLETT, W.C. 1997b. Dietary fibre, glycemic load, and risk of non-insulin dependent diabetes mellitus in women. Journal of the American Medical Association, vol. 277, pp. 472-477.

SALMOLS, E. and DORMANDY, T.L. 1963. Insulin response to fructose and galactose. Lancet, vol. I, pp. 478-479.

SELLERS, T.A., KUSHI, L.H., POTTER, J.D., KAYE, S.A., NELSON, C.L., MCGOVERN, P.G. and FOLSOM, A.R. 1992. Effect of family history, body-fat distribution, and reproductive factors on the risk of postmenopausal breast cancer. New England Journal of Medicine, vol. 326, pp. 1323-1329.

SHEARD, N.F., CLARK, N.G., BRAND-MILLER, J.C., FRANZ, M.J., PI-SUNYER, F.X., MAYER-DAVIS, E., KULKARNI, K. and GEIL, P. 2004. Dietary carbohydrate (amount and type) in the prevention and management of diabetes. Diabetes Care, vol. 27, pp. 2266-2271.

SHIKANY, J.M., THOMAS, S.E., HENSON, C.S., REDDEN, D.T. and HEIMBURGER, D. 2006. Glycemic index and Glycemic load of popular weight-loss diets. Medscape General Medicine, vol. 8, no.1, pp. 22-30.

SINGER, D.E., NATHAN, D.M., ANDERSON, K.M., WILSON, P.W. and EVANS, J.C. 1992. Association of HbA_{1c} with prevalent cardiovascular disease in the original cohort of the Framingham Heart Study. Diabetes, vol. 41, pp. 202-208.

SLABBER, M. 2005. Complexities of consumer understanding of the glycaemic index concept and practical guidelines for incorporation in diets. The South African Journal of Clinical Nutrition, vol. 18, no. 3, pp. 252-257.

SLABBER, M., BARNARD, H.C., KUYL, J.M., DANNHAUSER, A. And SHALL, R. 1994. Effects of a low-insulin-response, energy- restricted diet on weight loss and plasma insulin concentrations in hyperinsulinaemic obese females. American Journal of Clinical Nutrition, vol. 60, pp. 48-53.

SLYPER, A.H. 2004. The Pediatric Obesity Epidemic: Causes and Controversies. The Journal of Clinical Endocrinology & Metabolism, vol. 89, no. 6, pp. 2540-2547.

SOUGLERI, M., LABROPOULOU-KARATZA, C., PARASKEVOPOULOU, P., FRAGOPANAGOU, H. and ALEXANDRIDES, T. 2001. Chronic hepatitis C virus infection without cirrhosis induces insulin resistance in patients alpha-thalassaemia major. European Journal of Gastroenterology & Hepatology, vol. 13, pp. 1195-1199.

SOUTH AFRICA. DEPARTMENT OF HEALTH (DoH). 2002. Government Notice. Regulations relating to labelling and advertising of foodstuffs. (R. 1055). Government Gazette: 23714, 8 August. p. 87.

SUMMERSON, J.S., KONAN, J.C. and DIGNAN, M.B. 1992. Race related differences in metabolic control among adults with diabetes. South African Medical Journal, vol. 85, pp. 953-956.

SUZUKI, M. 2003. Glycemic carbohydrates consumed with amino acids or protein right after exercise enhances muscle formation. Nutrition Reviews, vol. 61, pp. S88-S94.

THOMPSON, L.U. 1988. Anti-nutrients and blood glucose. Food Technology, vol. 42, pp. 123-130.

TIMAR, O., SESTIER, F. and LEVY, E. 2000. Metabolic syndrome X: A review. Canadian Journal of Cardiology, vol. 16, pp. 779- 789.

TRUSWELL, A.S. 1992. Glycaemic index foods. European Journal of Clinical Nutrition, vol. 46, no. 2(suppl.), pp. S91-S101.

VAN DAM, R.M., VISSCHER, A.W.J., FESKENS, E.J.M., VERHOEF, P. and KROMHOUT, D. 2000. dietary glycemic index in relation to metabolic risk factors and incidence of coronary heart disease: the Zutphen Elderly Study. European Journal of Clinical Nutrition, vol. 54, pp. 729-731.

VENTER, C.S. 2005. The glycaemic index – scientific evidence on the practical use. The South African Journal of Clinical Nutrition, vol. 18, no. 3, pp. 211-212.

VENTER, C.S., JERLING, J.C., VAN HEERDEN, Y. and PIETERS, M. 2005. More evidence for capillary sampling in the determination of the glycaemic index. South African Journal of Clinical Nutrition, vol. 18, no. 3, pp. 238-242.

VENTER, C.S., SLABBER, M. And VORSTER, H.H. 2003. Labelling of foods for glycaemic index: Advantages and problems. South African Journal of Clinical Nutrition, vol. 16, pp.118-126.

VENTER, C.S., VORSTER, H.H., VAN ROOYEN, A., KRUGER-LOCKE, M.M. and SILVIS, N. 1990. Comparison of the effects of maize porridge consumed at different temperatures on blood glucose, insulin and acetate levels in healthy volunteers. South African Journal of Food Science and Nutrition, vol. 2, no. 1, pp. 2-5.

VERGAUWEN, L., BROUNS, F. and HESPEL, P. 1998. Carbohydrate supplementation improves stroke performance in tennis. Medicine and Science in Sports and Exercise, vol. 30, pp. 1289-1295.

VORSTER, H.H. 2005. The glycaemic index in practice – consensus statement of a small group of South African dietitians. South African Journal of Clinical Nutrition, vol.18, no. 3, pp. 260-264.

VORSTER, H.H., VAN TONDER, E., KOTZE, P. and WALKER, A.R.P. 1987. Effects of graded sucrose additions on taste preference, acceptability, glycaemic index, and insulin response to butter beans. American Journal of Clinical Nutrition, vol. 45, pp. 575-579.

VORSTER, H.H., VENTER, C.S. and SILVIS, N. 1990. The glycaemic index of foods: a critical evaluation. The South African Journal of Food Science and Nutrition, vol. 2, no.1, pp. 13-17.

VORSTER, H.H., LOVE, P. and BROWNE, C. 2001. Development of food-based dietary guidelines for South Africa – the process. South African Journal of Clinical Nutrition, vol. 14, pp. 3S-6S.

WALKER, A.R.P. and WALKER, B.F. 1984. Glycemic index of South African foods determined in rural blacks- a population at low risk to diabetes. Human Nutrition and Clinical Nutrition, vol. 38C, pp. 215-222.

WELCH, I.M., BRUCE, C., HILL, S.E. and READ, N.W. 1987. Duodenal and ileal lipid suppresses postprandial blood glucose and insulin responses in man: possible implications for the dietary management of diabetes mellitus. Clinical Science (London), vol. 72, pp. 209-216.

WILLET, W., MANSON, J.A. and LIU, S. 2002. Glycemic index, glycemic load and risk of type 2 diabetes. American Journal of Clinical Nutrition, vol. 76, pp. 274S-1280S.

WOLEVER, T.M.S. 1990. The glycaemic index. World Review of Nutrition and Dietetics, vol. 62, pp. 120-185.

WOLEVER, T.M.S. 2000. Dietary carbohydrates and insulin action in humans. British Journal of Nutrition, vol. 83 (suppl 1), pp. S97-S102.

WOLEVER, T.M.S. 2003. Carbohydrate and the regulation of blood glucose and metabolism. Nutrition Reviews, vol. 61, no. 5, pp. S40-S48.

WOLEVER, T.M.S. and BOLOGNESI, C. 1996b. Time of day influences relative glycaemic effect of foods. Nutrition Research, vol. 16, no. 3, pp. 381-384.

WOLEVER, T.M.S. and BRAND-MILLER, J. 1995. Sugars and blood glucose control. American Journal of Clinical Nutrition, vol. 62 (suppl.) pp. 21-227.

WOLEVER, T.M., CSIMA, A., JENKINS, D.J., WONG, G.S. and JOSSE, R.G. 1989. The glycemic index: variation between subjects and predictive differences. Journal of American College of Nutrition, vol. 8, pp. 235-247.

WOLEVER, T.M.S., JENKINS, D.J.A., COLLIER, G.R., LEE, R., WONG, G.S. and JOSSE, R.B. 1988. Metabolic response to test meals containing different carbohydrate foods: Relationship between rate of digestion and plasma insulin response. Nutrition Research, vol. 8, pp. 573-581.

WOLEVER, T.M.S., JENKINS, D.J., JENKINS, A.L. and JOSSE, R.G. 1991. The glycaemic index: methodology and clinical implications. American Journal of Clinical Nutrition, vol. 54, no. 5, pp. 846-854.

WOLEVER, T.M.S., JENKINS, D.J.A., JOSSE, R.B., WONG, G.S. and LEE, R. 1987. The glycaemic index: similarity of values derived in insulin-dependent and non-insulin-dependent diabetic patients. Journal of American College of Nutrition, vol. 6, pp. 295-305.

WOLEVER, T.M.S., JENKINS, D.J., VUKSAN, V., JENKINS, A.L., WONG, G.S. and JOSSE, R.G. 1992. Beneficial effect of low-glycaemic index diet in overweight NIDDM subjects. Diabetes Care, vol. 15, pp. 562-564.

WOLEVER, T.M., NUTTALL, F.Q., LEE, R., WONG, G.S., JOSSE, R.G., CSIMA, A. and JENKINS, D.J. 1985. Prediction of the relative blood glucose response of mixed meals using the white bread glycaemic index. Diabetes Care, vol. 8, pp. 418-428.

WOLEVER, T.M.S., VORSTER, H.H., BJÖRK, I., BRAND-MILLER, J., BRIGHENTI, F., MANN, J.I., RAMDATH, D.D., GRANFELDT, Y., HOLT, S., PERRY, T.L., VENTER, C. and WU, X. (2002) Determination of the glycaemic index of foods: interlaboratory study. European Journal of Clinical Nutrition, vol. 57, pp. 475–482.

WOLEVER, T.M.S., VOSTER, H.H., BJÖRK, I., BRAND-MILLER, J., BRIGHENTI, F., MANN, I.J., RAMDATH, D.D., GRANDFELDT, Y., HOLT, S., PERRY, T.L., VENTER, C. and XIAOMEI, Wu. 2003. Determination of glycaemic index of foods: Interlaboratory study. European Journal of Nutrition, vol. 57, no. 3, pp. 475-482.

WOLEVER, T.M.S., KATZMAN-RELLE, L., JENKINS, A.L., VUKSAN, V., JOSSE, R.G. and JENKINS, D.J.A. 1994. The glycaemic index of 102 complex carbohydrate foods in patients with diabetes. Nutrition Research, vol. 14, pp. 651-669.

WRIGHT, H.H. 2005. The glycaemic index and sports nutrition. South African Journal of Clinical Nutrition, vol. 18, no. 3, pp. 222-228.

WRIGHT, D.A., SHERMAN, W.M. and DERNBACH, A.R. 1991. Carbohydrate feedings before, during or in combination improve cycling endurance performance. Journal of Applied Physiology, vol. 71, pp. 1082-1088.

WYLIE-ROSETT, J., SEGAL-ISAACSON, C.J. and SEGAL-ISAACSON, A. 2004. Carbohydrates and increases in obesity: Does the type of carbohydrate make a difference? Obesity Research, vol. 12 (suppl. Nov), pp. 124S-129S.

YOUNG, K.W.H. and WOLEVER, T.M.S. 1998. Effect of volume and type of beverage with a standard test meal on postprandial blood glucose responses. Nutrition Research, vol. 18 no. 11, pp. 1857-1863.

Appendix A

Department of Human Nutrition (G24)
P.O. Box 339
UFS
Bloemfontein
9300

Dr.

Re: Permission to perform study to obtain master's degree by using campus students as subjects

I, Marinda van Zyl am a master's degree student in dietetics and I am performing this study in order to complete my postgraduate degree. The aim of this study is to determine the glycaemic index (GI) of three Albany Superior™ breads namely Best of Both™, Brown bread™ and Whole Wheat™ bread.

The methodology of the study is basically as follows: Twenty healthy male student volunteers between the ages of 21-27 years will be recruited through advertising the study on bulletin boards and via announcements in hostels. To be included in this study a random finger prick blood glucose test will be done by the researcher (myself) and one registered nurse or doctor. Only subjects with normal random blood glucose test values will be included. Those who are glucose intolerant or classifiable as diabetics will be referred for medical treatment. Each subject will be required to participate once a week over a period of six weeks.

Participation entails taking a standard pre-evening test meal, being fasting from 22h00 and in bed by 23h00. No smoking, alcohol usage or exercise is allowed twelve hours prior to testing. The next morning after the 10-12 hour fast, a blood sample will be obtained from each subject. Thereafter further samples will be taken after subjects randomly consumed a test meal of either 50 g of glucose powder dissolved in 300 ml of water or 50 g available glycaemic carbohydrates from one of the three different types of bread (consumed with 300 ml of bottled still water). Thus, finger-prick capillary blood samples will be taken before (fasting) and every 15 minutes for one hour after the test meal was consumed, , and thereafter every 30 minutes for another hour.

This whole procedure will be repeated on one specific day of the week [Tuesday to Friday], on a weekly basis over a period of 6 weeks. Thus, each subject will participate in the study one day of the week, every week for a period of six weeks. This procedure is free of any medical complications or side effects.

The study is of great importance in that the results of this study will supply scientific GI values for three well known types of bread on the market. I will therefore greatly appreciate it if I could obtain permission to conduct this study.

Yours sincerely

Marinda van Zyl

Student nr: 1997221302

P.O. Box 339
UFS
Bloemfontein
9300

Prof.

Re: Permission to perform study to obtain master's degree by using campus students as subjects

I, Marinda van Zyl am a master's degree student in dietetics and I am performing this study in order to complete my postgraduate degree. The aim of this study is to determine the glycaemic index (GI) of three Albany Superior™ breads namely Best of Both™, Brown bread™ and Whole Wheat™ bread.

The methodology of the study is basically as follows: Twenty healthy male student volunteers between the ages of 21-27 years will be recruited through advertising the study on bulletin boards and via announcements in hostels. To be included in this study a random finger prick blood glucose test will be done by the researcher (myself) and one registered nurse or doctor. Only subjects with normal random blood glucose test values will be included. Those who are glucose intolerant or classifiable as diabetics will be referred for medical treatment. Each subject will be required to participate once a week over a period of six weeks.

Participation entails taking a standard pre-evening test meal, being fasting from 22h00 and in bed by 23h00. No smoking, alcohol usage or exercise is allowed twelve hours prior to testing. The next morning after the 10-12 hour fast, a blood sample will be obtained from each subject. Thereafter further samples will be taken after subjects randomly consumed a test meal of either 50 g of glucose powder dissolved in 300 ml of water or 50 g available glycaemic carbohydrates from one of the three different types of bread (consumed with 300 ml of bottled still water). Thus, finger-prick capillary blood samples will be taken before (fasting) and every 15 minutes for one hour after the test meal was consumed, , and thereafter every 30 minutes for another hour.

This whole procedure will be repeated on one specific day of the week [Tuesday to Friday], on a weekly basis over a period of 6 weeks. Thus, each subject will participate in the study one day of the

week, every week for a period of six weeks. This procedure is free of any medical complications or side effects.

The study is of great importance in that the results of this study will supply scientific GI values for three well known types of bread on the market. I will therefore greatly appreciate your permission to perform above mentioned study. I will therefore greatly appreciate it if I could obtain permission to conduct this study.

Yours sincerely

Marinda van Zyl

Student nr: 1997221302

Appendix B

TITLE:

DETERMINATION OF THE GLYCAEMIC INDEX OF THREE TYPES OF ALBANY SUPERIOR™ BREAD

Recruitment form:

I, the undersigned (full name) _____
give permission to have finger-prick blood glucose testing performed on me.

Ek, die ondergetekende (volle name en van) _____
gee toestemming dat vingerprik bloed glukose toetse op my uitgevoer mag word.

Signature/Handtekening: _____

- 1. Subject no.:.....
- 2. Age:.....years
- 3. Weightkg
- 4. Heightcm
- 5. BMI:.....kg/m²
- 6. Anyknown disease? _____
- 7. Random glucose test value _____ mmol/L
- 8. Healthy? Yes/No

- 9. Medication usage (particularly those affecting glucose tolerance)

- 10. Dietary supplements usage

- 11. INFORMED CONSENT AND INDEMNITY FORM SIGNED (YES (1)/NO (2))

Appendix C

DETERMINATION OF THE GLYCAEMIC INDEX OF THREE TYPES OF ALBANY SUPERIOR™ BREAD

INFORMED CONSENT FORM**SUBJECT NUMBER:** _____

Thank you for sacrificing your time and participating in this study. We want to determine the glycaemic index (GI) of three types of bread. The GI is a measure of a food's power to raise the blood-glucose concentration after a meal. The purpose of the study is to determine scientific GI values for three well known bread types on the South African market and the results of the study may be used for scientific publications. A potential advantage of the study is that the GI of a food can be used to guide consumers in choosing a particular food with a predicted known effect on blood glucose levels and homeostasis. No costs will be payable by the participants in the study. What we need from you is:

- Every pretest evening every participant will gather at the seminar room, Department of Surgery, Faculty of Health Science to consume a standard pre-evening test meal and the participant will be studied after a 10-12 hour fast once a week (five subjects per day, 4 days a week, over a six week period[Tuesday to Friday]).
- All rules and procedures regarding what you're allowed to do and what not, will carefully be explained to minimize factors which may influence glucose responses. No smoking, alcohol usage or exercise is allowed 12 hours prior to testing.
- You will be instructed to spend the evening relaxing and be in bed by 23h00.
- The next morning after the 10-12 hour fast (on the test days), a blood sample will be obtained from each subject and further samples will be taken after you randomly consumed a test meal of either 50g glucose powder dissolved in 300mL of bottled still water or 50 g available glycaemic carbohydrates from one of the three different types of bread (consumed with 300 ml of bottled still water). Finger-prick capillary blood samples will be taken before (fasting) and every 15 minutes for one hour after the test meal was consumed, and thereafter every 30 minutes for another hour.
- The researcher and one registered nurse or medical doctor will measure whole capillary blood glucose, using One Touch Ultra™ test strips and One Touch Ultra™ glucometers (Lifescan™). This procedure will be done in strict compliance with the protocol recommendations of the manufacturer as well as good laboratory practice as described by the GI Task Force (GI Task Force, 2002).

The study is strictly confidential. This whole procedure will be repeated on one specific day of the week [Tuesday to Friday], on a weekly basis over a period of 6 weeks. Thus, each subject will participate in the study one day of the week, every week for a period of six weeks. This procedure is free of any medical complications, risks or side effects. Participation is voluntary and subjects may withdraw from the study at any time. If any further information is needed please call Marinda van Zyl.

Title of project:

DETERMINATION OF THE GLYCAEMIC INDEX OF THREE TYPES OF ALBANY SUPERIOR™ BREAD

I, the undersigned (full names)

have read the above information regarding the project and also heard the oral version thereof and hereby declare that I fully understand it. I was given the opportunity to discuss relevant aspects of the project with the project leader and hereby declare that I am participating in this project on my own free will. I therefore give my permission to be included in this study as subject (participant).

I hereby indemnify the University of the Free State and also any employee or student of the University, against any liability projected upon me during the course of this study. I undertake not to put forward any claim against the University for damage to property or disadvantage to me personally that may develop as a result of this study, whether it may be due to neglect by the University, its employees or students or other subjects.

Signature of study participant (subject)_____

OR

Parents of study participant (subject)_____ **(if younger than 21 years)**

WITNESSES;

1.....2.....

SIGNED AT.....ON.....

Appendix C

**BEPALING VAN DIE GLUKEMIESE INDEKS VAN DRIE
TIPES ALBANY SUPERIOR™ BROOD**

INGELIGTE TOESTEMMINGSVORM PROEFPERSOON NR.:_____

Dankie dat u , u tyd opoffer om deel te neem aan hierdie studie. Ons wil die glukemiese indeks (GI) van drie tipes brood bepaal. Die GI is 'n aanduiding van 'n sekere tipe voedsel se vermoë om die bloedglukose konsentrasie na 'n maaltyd te verhoog. Die doel van die studie is die bepaling van wetenskaplike GI waardes vir drie welbekende broodsoorte op die Suid-Afrikaanse mark. Die resultate van die studie mag moontlik gebruik word in wetenskaplike publikasies. 'n Potensiële voordeel van die studie mag wees dat aangesien die GI van 'n spesifieke voedsel (die drie brode) dus bekend sal wees, dit verbruikers kan help om 'n voedselkeuse te maak ten opsigte van die effek wat die betrokke voedsel op bloedglukose waardes en -homeostase kan hê. Geen kostes sal van proefpersone verhaal word vir deelname aan die studie nie. Ons benodig die volgende van jou:

- Elke deelnemer sal op 'n weeklikse basis die aand voor die toetsing vergader in die seminaarkamer van die Chirurgie Departement, Fakulteit Geneeskunde. 'n Standaard pre-toets maaltyd sal dan ingeneem word en deelnemers sal dan getoets word na 'n 10-12 uur vastende periode (vyf proefpersone per dag, vier dae per week, oor 'n tydperk van ses weke [Dinsdag tot Vrydag]).
- Reëls en regulasies rakende moets en moenies tydens die studies sal breedvoerig verduidelik word om faktore wat glukose response mag beïnvloed tot 'n minimum te beperk. Geen rook, alkohol gebruik of oefening word in die 12 ure voordat glukose toetsing plaasvind, toegelaat nie.
- U sal versoek word om die aand vooraf ontspanne deur te bring en teen 23h00 te gaan slaap.
- Die volgende oggend na die 10-12 uur vastende periode (tydens die toetsdag), sal bloedmonsters vastend geneem word, asook verskeie bloedmonsters nadat 'n toetsmaal van of 50 g glukose poeier opgelos in 300 ml gebottelde minerale water, of 50 g beskikbare glukemiese koolhidrate van een van die drie verskillende soorte brood (saam met 300 ml gebottelde minerale water), ingeneem is. Dit wil sê dat 'n vingerprik kapillêre bloedmonster vastend en daarna elke 15 minute vir die eerste uur, gevolg deur elke 30 minute vir die tweede uur, geneem sal word.
- Die navorser en een geregistreerde staff verpleegster of dokter sal kapillêre bloedglukose (vingerprik glukose) deur middel van One Touch Ultra™ toetsstrokies en One Touch Ultra™ glukometers (Lifescan™) bepaal. Protokol aanbevelings sal streng tydens die prosedure nagevolg word, soos uiteengesit deur die vervaardiger en in ooreenstemming met goeie laboratoruim-praktyk soos beskryf deur die GI-Taakspan (GI Task Force, 2002).

Die studie is streng vertroulik. Hierdie prosedure sal weekliks herhaal word op 'n spesifieke dag van die week [Dinsdae tot Vrydae] vir 'n periode van ses weke. Dieselfde proefpersoon sal dus een keer per week, elke week vir 'n periode van ses weke, aan die studie deelneem. U as proefpersone word aan geen mediese komplikasies, risiko's of nuwe-effekte blootgestel nie. Deelname aan die studie is vrywillig en proefpersone kan op enige tydstip onttrek aan die studie. Indien enige verdere inligting benodig word kan u Marinda van Zyl kontak.

Title of project:

**BEPALING VAN DIE GLUKEMIESE INDEKS VAN DRIE Tipes ALBANY SUPERIOR™
BROOD**

Ek, die ondergetekende (volle name)

het die bogenoemde inligting in verband met die projek aandagtig deurgelees en ook die na mondelinge weergawe geluister en dus verklaar ek dat ek die inligting tenvolle verstaan. Ek is die geleentheid gegun om relevante aspekte in verband met die projek met die studieleier te bespreek en verklaar hiermee dat ek uit my eie vrye wil aan die projek deelneem. Ek gee hiermee my toestemming om as proefpersoon in die studie ingesluit te word.

Ek vrywaar hiermee die Universiteit van die Vrystaat asook enige werknemers en studente van die Universiteit teen enige aanspreeklikheid wat teenoor my, in die loop van die projek mag ontstaan. Ek onderneem verder om geen eise teen die Universiteit in te stel weens skade of persoonlikheidsnadeel wat ek weens die projek mag ly, hetsy dit aan die nalatigheid van die Universiteit, sy werknemers of studente, of ander proefpersone mag ontstaan nie.

Handtekening van proefpersoon _____

Of

Proefpersoon se ouers _____ **(indien jonger as 21 jaarige ouderdom)**

GETUIES;

1.....2.....

ONDERTEKEN TE.....OP.....

Appendix D

TITLE

**DETERMINATION OF THE GLYCAEMIC INDEX OF THREE TYPES OF ALBANY SUPERIOR™
BREAD**

INSTRUCTIONS

FOR OFFICE USE

Mark the appropriate block with an X or write your answer on the space provided

1. Product to be tested _____

1-3

2. Date of test (dd/mm/yy)/...../.....

4-9

3. Subject no. _____

10-11

4. Birth date? (dd/mm/yy)/...../.....

12-17

TEST RESULTS

5. Name of glucose measuring device used for test_____

18-19

6. Calibration strip lot no_____

20-21

7. Name of control solution used_____

22-23

8. Expiry date of control solution_____

24-25

9. Range of control solution_____

26-27

10. Control value 1_____

28-29

11. Control value 2_____

30-31

12. Capillary glucose in mmol/L

Time in minutes	Glucose reading	Unit					
0		mmol/L			.		32-35
15		mmol/L			.		36-39
30		mmol/L			.		40-43
45		mmol/L			.		44-47
60		mmol/L			.		48-51
90		mmol/L			.		52-55
120		mmol/L			.		56-59