

**The glycaemic index of muffins baked with extruded
dried bean flour compared to muffins baked with
whole wheat flour**

**JACQUELINE GOUWS
B.Sc. (Dietetics)**

**Mini-dissertation submitted in partial fulfilment of the
requirements for the degree Magister Scientiae in Dietetics
at the Potchefstroomse Universiteit vir Christelike Hoër
Onderwys**



Supervisor: Prof. C.S. Venter
Co-supervisor: Prof. W. Oosthuizen

2003
Potchefstroom



Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

This dissertation is dedicated to my husband Pierre, my parents, David and Yvonne and my children, Rowan and Myles.

ACKNOWLEDGEMENTS

I would like to thank my Lord Jesus Christ for giving me the privilege, ability and divine help to complete this work. My gratitude and sincere thanks are expressed to the following individuals and organizations:

Prof Christine Venter, who continues to give her support, guidance and encouragement as well as knowledge and wisdom.

Dr Theo Nell for his assistance, his impartation of knowledge and sense of humour throughout the research period.

Prof Welma Oosthuizen for her assistance, knowledge, guidance and support.

Celia Matthews, my research assistant, who always is the same willing, kind and supportive person.

Sister Chrissie Lessing, for collection of blood samples, other assistance, kindness and gentleness.

Prof. Faans Steyn for his assistance in analysing the data statistically and continuous help. All the subjects, without whom I would not have had a research project.

The Dried Bean Producers Organization, especially Ms Engela van Eyssen.

The staff of Ferdinand Postma Library, especially Ms Helah van der Walt for professionalism, perseverance and outstanding support and service.

My husband Pierre, for his support and encouragement, my children Rowan and Myles for being patient and understanding.

My parents, especially for their continuous support, encouragement and love and for standing in for me while I was conducting the research.

My brother, sisters and friends for support in prayer and in so many other ways.

My nephews Juan and Jaco and our secretary Carien a special word of thanks and gratitude.

Prof Lesley Greyvenstein from PU vir CHE, for editing the document.

The views expressed in this dissertation are my own and do not concur with that of the Dried Bean Producers Organization.

ABSTRACT

Introduction: Emphasis on using the glycaemic index (GI) in addition to carbohydrate exchange lists has led to a greater variety of foods from which to choose for the diabetic population. Breakfast is regarded as the most important meal of the day and the glycaemic response to lunch can be improved by decreasing the GI of breakfast. However, most conventional breakfast cereals and bread exhibit a high GI. Dried beans have a low GI and various processes such as cooking and canning increase GI values, but still in the low GI range. In recent years, extrusion cooking has become one of the popular new processes developed by the food industry. Extrusion provides a convenient alternative for the ingestion of dry beans in the diet. Muffins are eaten by many South Africans and may be an ideal alternative for breakfast cereals and bread, especially if the GI of the muffins is low. The aim of this study was to determine the GI of a muffin baked with extruded bean flour and compare it to the GI of a muffin baked with whole wheat flour.

Subjects and methodology: The study cohort consisted of ten healthy males and ten healthy females. Subjects randomly consumed test meals of glucose (the reference), bean muffins and whole wheat muffins on different days. Each test meal provided 50g available carbohydrate as analysed by the Englyst method.

Results: The GI of the muffin baked with extruded bean flour (mean 53.0%, Confidence intervals (CI): 41.7; 64.2) was not significantly different from that of the whole wheat muffin (mean 55.5%, CI: 41.8; 69.2) but still in the low to intermediate GI category.

Conclusion: Extrusion of dried beans results in a fine flour with relatively no intact starch which may explain the very low resistant starch content (1.6/100g) of the muffins. The small particle size of the fine flour could further have contributed to the higher than expected GI of the bean muffin because the size of the particle is inversely related to glycaemic response. Muffins baked with extruded dried bean meal are nevertheless regarded as an excellent choice for breakfast and as part of the prudent diet. Beans have additional health benefits and are included in the South African Food Based Dietary Guidelines.

Keywords: glycaemic index, dried bean muffin, whole wheat muffin, extrusion.

OPSOMMING

Inleiding: Klem op die gebruik van die glukemiese indeks (GI), bykomend tot die koolhidraatruillyste, het gelei tot 'n groter verskeidenheid van voedsels om van te kies vir die diabetiese bevolking. Ontbyt word beskou as die belangrikste maaltyd van die dag en die glukemiese respons tot middagetes kan verbeter word deur die GI van ontbyt te verlaag. Die meeste van die konvensionele ontbygtraankossoorte en brood het egter 'n hoë GI. Droëbone beskik oor 'n lae GI. Die inmaakproses verhoog die GI-waardes maar dit is steeds in die lae GI-grens. In onlangse jare het ekstrusiegaarmaak ontwikkel deur die voedselindustrie een van die gewilde nuwe prosesse geword. Ekstrusie verskaf 'n gerieflike alternatief vir die inname van droëbone in die dieet. Muffins word deur baie Suid-Afrikaners geëet en mag 'n ideale alternatief vir ontbygtrane en brood wees, veral as die GI van muffins laag is. Die doel van die studie was om die GI van 'n muffin gebak met geëkstrueerde droëboonmeel te bepaal en dit te vergelyk met die GI van 'n muffin gebak met volgraanmeel.

Proefpersone en metodologie: Die studiegroep het bestaan uit tien gesonde mans en tien gesonde dames. Proefpersone het beurtelings toetsmaaltye van glukose (standaard), boonmuffins en volgraanmuffins ingeneem op verskillende dae. Elke toetsmaal het 50g beskikbare koolhidrate verskaf, soos met die Englyst-metode geanaliseer.

Resultate: Die GI van die muffin gebak met geëkstrueerde droëboonmeel (gemiddeld 53.0%, Vertrauensinterval, (VI): 41.7; 64.2) was nie betekenisvol verskillend van die volgraanmuffin nie (gemiddeld 55.5%, VI: 41.8; 69.2) maar nog steeds in die lae tot intermediêr kategorie.

Gevolgtrekking: Ekstrusie van droëbone lei tot 'n fyn meel met relatief geen intakte stysel nie, wat die baie lae weerstandbiedende styselinhoud (1.6g/100g) van die muffins mag verklaar. Die klein partikelgrootte van die fyn meel kon verder bygedra het tot die hoër as verwagte GI van die bonemuffin omdat die grootte van die partikel omgekeerd eweredig verwant is aan die glukemiese respons. Muffins gebak met geëkstrueerde droëbonemeel word nogtans beskou as 'n uitstekende keuse vir ontbyt en as deel van die omsigtige dieet. Bone het addisionele gesondheidsvoordele en is ingesluit in die Voedselgebaseerde Dieetriglyne vir Suid-Afrikaners.

Sleutelwoorde: glukemiese indeks, droëbonemuffin, volgraanmuffin, ekstrusie.

TABLE OF CONTENTS

	Page
ABSTRACT	ii
OPSOMMING	iii
LIST OF TABLES	viii
LIST OF FIGURES	ix
LIST OF APPENDICES	x
LIST OF ABBREVIATIONS	xi

CHAPTER 1

INTRODUCTION

1.1	CARBOHYDRATES AND THE GLYCAEMIC INDEX.....	1
1.2	BACKGROUND AND MOTIVATION FOR THIS STUDY.....	2
1.3	OBJECTIVES OF THIS STUDY.....	4
1.4	STRUCTURE OF THE MINI-DISSERTATION.....	4

CHAPTER 2

LITERATURE REVIEW

2.1	INTRODUCTION.....	6
2.2	THE GLYCAEMIC INDEX.....	6
2.2.1	The glycaemic load.....	9
2.3	FACTORS INFLUENCING THE GLYCAEMIC INDEX.....	9
2.3.1	Non-food factors.....	10
2.3.2	Food factors.....	10
2.3.3	Chemical structure of carbohydrates.....	12

2.3.4	Dietary fibre and resistant starch.....	13
2.3.5	Anti-nutrients.....	15
2.3.6	Co-ingestion of macronutrients.....	16
2.3.7	Organic acids.....	17
2.3.8	Other food factors.....	18
2.3.9	Processing.....	19
2.3.10	The influence of a mixed meal on the glycaemic index.....	20
2.4	PHYSIOLOGICAL AND THERAPEUTIC IMPLICATIONS OF THE GLYCAEMIC INDEX.....	21
2.4.1	Second-meal effect.....	21
2.4.2	Blood glucose and insulin resistance.....	23
2.4.3	Coronary heart disease.....	25
2.4.4	Obesity.....	27
2.4.5	Cognitive performance.....	29
2.4.6	Application in sports nutrition.....	30
2.5	CRITICISM REGARDING THE PRACTICAL APPLICATION AND CLINICAL UTILITY OF THE GLYCAEMIC INDEX.....	32
2.6	FUTURE GLYCAEMIC INDEX RESEARCH.....	34
2.7	SUMMARY.....	35
2.8	DRIED BEANS.....	35
2.8.1.	Introduction.....	35
2.8.2.	Composition and nutrient value of dried beans.....	35
2.8.3.	Health benefits of dried beans.....	38
2.8.4.	The glycaemic index of dried beans and the practical incorporation into the diet.....	39

2.9.	THE EXTRUSION PROCESS.....	41
2.9.1	Introduction.....	41
2.9.2	Physicochemical and structural changes as a result of extrusion.....	42
2.9.3.	Advantages of extrusion.....	44
2.9.4.	Future potential.....	45
2.10.	THE GLYCAEMIC INDEX OF WHOLEGRAIN KERNEL PRODUCTS VERSUS THAT OF MILLED FLOUR.....	46

CHAPTER 3

METHODOLOGY

3.1	INTRODUCTION.....	47
3.2	METHODS.....	47
3.2.1	Subjects.....	47
3.2.2	Study design.....	48
3.2.3	Pre-test meals.....	49
3.2.4	Test foods.....	49
3.2.5	Biochemical analyses.....	50
3.2.6	Glycaemic index.....	50
3.2.7	Statistical analyses.....	50
3.2.8	Limitations of the study.....	51
3.2.8.1	Acceptability of meals.....	51
3.2.8.2	Other limitations.....	51
3.2.8.3	Conclusion.....	51

CHAPTER 4

RESULTS

4.1	INTRODUCTION.....	53
4.2	SUBJECT CHARACTERISTICS.....	53
4.3	INTRA AND INTER-INDIVIDUAL VARIATION IN THE GLUCOSE RESPONSES TO TWO GLUCOSE TESTS.....	53

4.4	MEAN GLUCOSE CURVES FOR DIFFERENT TEST FOODS.....	54
4.5	CALCULATION OF GLYCAEMIC INDICES.....	55
4.6	CONCLUSION.....	59
 CHAPTER 5		
DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS		
5.1	INTRODUCTION.....	60
5.2	DISCUSSION.....	60
5.3	CONCLUSION.....	62
5.4	RECOMMENDATIONS.....	62
	REFERENCES.....	65
	APPENDICES.....	78

LIST OF TABLES

Table 2.1	The nutrient composition of dry and soy beans, compared to Dietary Reference Intakes.....	37
Table 3.1	Latin square design for male subjects.....	48
Table 3.2	Latin square design for female subjects.....	49
Table 3.3.	Macronutrient analysis of bean muffin and whole wheat muffin.....	50
Table 4.1	Areas under the curves (AUCs) in mmol/L.min for test foods and reference.....	56
Table 4.2	Glycaemic indices(%) of bean muffins and whole wheat muffins for each subject (n=20).....	58
Table 4.3	Mean glycaemic indices of the test foods.....	59
Table 5.1	The glycaemic load of a bean muffin and whole wheat muffin.....	63

LIST OF FIGURES

Figure 4.1 Differences within and between some subjects in the areas under the glucose curve with the fasting level as baseline in response to two glucose test meals..... 54

Figure 4.2 Mean glucose levels for different test foods (n=20)..... 55

LIST OF APPENDICES

APPENDIX A	Informed consent form.....	78
APPENDIX B	Food Fundi for Windows used to analyse macronutrient content of foods.....	80
APPENDIX C	Recipes for test foods.....	81
APPENDIX D	The Englyst method for analysis of carbohydrate fractions....	84

ABBREVIATIONS

ADA	American Diabetes Association
AUC	incremental area under the glucose curve with the fasting glucose as baseline
ANF	anti-nutritional factors
AX	arabinoxylan
BM	bean muffin
BIRKO	β cell-specific insulin receptor knockout
BMI	body mass index
CHD	coronary heart disease
CI	confidence intervals
CV	coefficient of variation
DRI	Dietary Reference Intakes
et al.	et alii
FAO	Food and Agriculture Organization
FDA	Food and Drug Administration
FFA	free fatty acids
GE	gastric emptying
GI	glycaemic index
GL	glycaemic load
HDL	high-density lipoprotein
HPF	high protein fractions
HSF	high starch fractions
HTC	hard-to-cook
HTST	high-temperature, short time
II	insulinaemic index
kg	kilogram
LDL	low-density lipoprotein
MIN	minute
mmol/L	millimol per litre
MUFA	monounsaturated fatty acids
NIRKO	neural insulin receptor knockout
NPU	net protein utilisation
NSP	non-starch polysaccharides

PDCAAS	Protein Digestibility Corrected Amino Acid Score
PPBG	preprandial blood glucose
PER	protein-efficiency ratio
PUFA	polyunsaturated fatty acids
PAI-1	plasminogen activator inhibitor-1
RAG	rapidly available glucose
SAA	Sulphur containing amino acid
SAG	slowly available glucose
SD	standard deviation
SI	satiety index
TG	triglycerides
TVP	texturised vegetable protein
WWM	whole wheat muffin
WHO	World Health Organization

CHAPTER 1

INTRODUCTION

1.1 CARBOHYDRATES AND THE GLYCAEMIC INDEX

Carbohydrates are a diverse group of substances with varied physiological properties of differing importance to health. The physiological properties include carbohydrates as an energy source, as increasing satiety, in controlling blood glucose and insulin, in protein glycosylation (possibly affecting the process of ageing), as cholesterol lowering, in bile acid dehydroxylation, as a laxative, in fermentation leading to production of short chain fatty acids as well as increasing microbial biomass, controlling of colonic epithelial function and selective stimulation of microbial growth (Cummings *et al.*, 1997).

Based on the different chemical properties, a new classification for carbohydrates was proposed (Cummings *et al.*, 1997). Otto *et al.* (1973 & 1980) (as quoted by Wolever, 1990) first discovered that apart from the above functions and properties, different carbohydrate foods also produce different glycaemic responses, although the macronutrient composition is the same. Many similar studies followed, but a lack of standardization led to incomparable results (Wolever, 1990). Jenkins *et al.* (1981) was the forerunner in proposing the glycaemic index (GI) as a method to assess and classify responses to foods. This GI concept led to many years of research and debate, particularly regarding individual responses, methodology, practical application and clinical benefits (reviewed by Wolever, 1990).

The low GI diet has various physiological and therapeutic implications that are hotly debated. One of these is the second-meal effect, where the subsequent meal results in lower glycaemic responses when the previous meal has been of low GI composition (Jenkins *et al.*, 1982).

A low GI diet has many benefits and it was found that a low GI meal leads to lower blood glucose and insulin responses, resulting in reduced blood glucose profiles (Jenkins *et al.*, 1987) in healthy subjects and in type 2 diabetes mellitus subjects, respectively (Frost *et al.*, 1994; Jenkins, 1988a; Järvi *et al.*, 1999). Furthermore, a low GI diet has been found to decrease total serum cholesterol, low-density lipoprotein (LDL) cholesterol (Jenkins *et al.*, 1987) and triglycerides (TG) (Järvi *et al.*, 1999; Liljeberg & Björck, 2000). In addition, low GI

foods are associated with high high-density lipoprotein (HDL) cholesterol (Frost *et al.*, 1994) and reduced risk for developing diabetes (Salmerón *et al.*, 1997a,b) and cardiovascular disease (Liu *et al.*, 2000). It is also possible that low GI foods lead to a longer period of satiety, which may be beneficial for weight control (Holt *et al.*, 1995; Roberts, 2000). Studies on glycaemic control and cognitive performance have been conducted and results indicate that a low GI diet may play an important role in the above (Benton & Parker, 1998; Benton *et al.*, 2003; Fischer *et al.*, 2001). Finally, the low GI diet may possibly be applicable in sports nutrition (Burke *et al.*, 1998a; Burke *et al.*, 1998b; Burke *et al.*, 1993; Garcin *et al.*, 2001; Gretebeck *et al.*, 2002; Stannard *et al.*, 2000; Kiens *et al.*, 1990; Noakes, 2000; Thomas *et al.*, 1994; Wee *et al.*, 1999).

Criticism regarding the practical application and clinical utility varies, the main one being that of the applicability in mixed meals (American Diabetes Association (ADA), 1994; Coulston *et al.*, 1984b; Pi-Sunyer, 2002). However, support for the use of the GI is also evident (Brand Miller *et al.*, 1997; Giaco *et al.*, 2001; Gilbertson *et al.*, 2001; Vermeulen & Turnbull, 2000; Wolever, 1997). Future aspects of research include that of the relationship of the GI and oxidative stress and long-term studies will focus on the effect the GI has on chronic diseases (Jenkins *et al.*, 2002).

1.2 BACKGROUND AND MOTIVATION FOR THIS STUDY

It has been suggested that breakfast is the most important meal of the day and that ingestion thereof influences tasks requiring aspects of memory, but a lack of breakfast does not affect performance on an intelligence test (Benton & Parker, 1998). Fischer *et al.* (2001) state that macronutrients ingested in the morning improve cognitive performance depending on their glycaemic effect. Benton *et al.* (2003) confirm in their studies that a low GI rather than a high GI breakfast allows better cognitive performance later in the morning. Furthermore, the glycaemic response to lunch can be improved by decreasing the GI of breakfast (Jenkins *et al.*, 1982). The explanation postulated by Wolever *et al.* (1990) is that when carbohydrate is slowly absorbed there is less rapid rise in blood glucose, a smaller insulin response, and less of a tendency for the blood glucose to undershoot. This results in a smaller counter-regulatory response and improved glucose disposal after the next meal. Most breakfast cereals, particularly those intended for children, have high GIs (Foster-Powell *et al.*, 2002).

It is for the above reasons that a low GI alternative is sought. Dried beans, (*Phaseolus Vulgaris*) have a low GI of 28 (Foster-Powell *et al.*, 2002). Various processes such as cooking and canning lead to increased GI values but still denote a low GI (Foster-Powell *et al.*, 2002). Baked beans on toast is a favourite English breakfast dish. However, there are certain limitations in the utilization of dried beans (Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994; Wang & McIntosh, 1996). Adverse storage conditions of beans leads to hardening, which in turn results in longer cooking periods, a decrease in protein digestibility and, thus, available essential amino acids (Reyes-Moreno & Paredes-Lopez, 1993). Beans also contain low levels of sulphur containing amino acids, but can be complemented with other sources of plant or animal protein. An alternative method has been adopted by the World Health Organization (WHO) and US Food and Drug Administration (FDA) for evaluating protein as protein quality has been underestimated in the past (as reviewed by Messina, 1999).

Another factor i.e. heat stable antinutrients also contributes to limited availability of amino acids (Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994; Wang & McIntosh, 1996). Antinutrients, however, do have certain benefits (Messina, 1999; Harland & Morris, 1995) and various methods of processing lead to removal of this factor (Messina, 1999; Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994). From the above review, it is clear that these factors are easily overcome and that the primary constraints are due to lack of variety of dishes (Aguilera *et al.*, 1984; Harper, 1995) and extended cooking times (for those using the raw/unprocessed product). Further reasons are due to mild abdominal discomfort, bloating and increased flatulence, which is as a result of increased production of short-chain fatty acids, but the latter may have independent beneficial metabolic effects to man (Vorster & Venter, 1994).

The process of extrusion cooking, which dates back to the 1800s, is used extensively in the food industry. The extrusion of dried beans, which is a relatively new process, provides an economical and convenient alternative to cooking and canning as well as a variety of options for ingestion of beans. Muffins are popular for breakfast, especially muffins baked with whole wheat flour. The idea arose to utilize the extruded dried bean flour in muffins, which might well be found to be a suitable alternative. Extrusion of dried beans leads to certain desirable changes such as use of fractions thereof as ingredients in the development of snack products (Aguilera *et al.*, 1984). Further advantages include that of decreased cooking time as well as

increased protein and mineral value (Steel *et al.*, 1995; Wang & McIntosh, 1996), positive effects on body weight gain, plasma cholesterol, texture and flavour (Wang & McIntosh, 1996) and, finally, decreased plasminogen activator inhibitor (PAI-1) levels (Oosthuizen *et al.*, 2000). It is possible, however, that certain unfavourable changes in the GI of the dried bean may also occur.

1.3 OBJECTIVES OF THIS STUDY

The objective of this study was to determine whether muffins baked with extruded bean flour would have a lower GI compared to muffins baked with whole wheat flour. Dried beans have very low GIs which is attributed to many factors including their fibre, resistant starch (RS) tannin and phytic acid contents and high ratio amylose to amylopectin starch.

1.4 STRUCTURE OF THE MINI-DISSERTATION

Chapter 2 provides a review on the GI pertaining to methodology and influencing factors (with special emphasis on processing techniques). The glycaemic load (GL) will also be defined. The metabolic and therapeutic effects, as well as the practical application, will be discussed. Finally, criticism regarding the latter as well as newer aspects of GI research will be reviewed.

The nutrient value and health benefits of dried beans will be reviewed, as well as constraints in its utilization. The extrusion process and resulting physical and chemical changes, and the advantages, disadvantages and future of the above process will also be reviewed, including a brief review of the GI of wholegrain kernel products versus that of milled flour products.

Chapter 3 entails the description of the methodology used during the study, subject characteristics, test meals, blood sampling, statistical analysis and study limitations.

Chapter 4 gives the reported results in the form of tables and graphs. These results include brief details on intra and inter-individual variation in blood glucose and oral glucose solution and the GIs of dried bean and whole wheat muffins respectively.

The results are discussed in detail in Chapter 5 and include reviewing the factors influencing the GI of the muffins. Finally the conclusions are drawn and recommendations are made for future research and development.

CHAPTER 2

LITERATURE REVIEW

2.1 INTRODUCTION

This chapter provides a review of the most recent proposed methods to determine the GI, the definition of the glycaemic load (GL), as well as the non-food and food factors and other influencing variables with special emphasis on processing techniques. Physiological and therapeutic implications of the GI and criticism regarding the practical application and clinical utility are included. Newer aspects of GI research are mentioned. Furthermore, the health benefits and GI of dried beans, as well as practical incorporation thereof will be discussed. The extrusion process and resultant changes, advantages and future potential are also reviewed. Finally, the GI of wholegrain kernel products versus that of milled flour is included.

2.1.1 THE GLYCAEMIC INDEX

The term GI was coined by Jenkins *et al.* (1981). Its aim was to recognize carbohydrates not only as portions on diabetic exchange lists, but also according to their individual physiological actions. The first list of GI values for 62 foods was published (Jenkins *et al.*, 1981). The definition of the GI describes this tool as the area under the glucose curve (AUC) after the ingestion of 50g carbohydrates from a particular food, divided by the AUC resulting from the ingestion of 50g carbohydrate from a reference food, multiplied by 100 (Jenkins *et al.*, 1981). The Food and Agriculture Organization (FAO) and WHO (1998) recommends the use of 50g available carbohydrates except when the volume of a low carbohydrate food dictates a smaller load such as a 25g carbohydrate portion. Since glycaemic responses are related to the amount of carbohydrate ingested, use of a smaller portion size will result in a lower GI value (Wolever & Bolognesi, 1996a).

Available or glycaemic carbohydrate is measured as the total carbohydrate minus the dietary fibre (FAO/WHO, 1998). Englyst *et al.* (1999) describe available carbohydrate as the glycaemic fraction, which is absorbed in the small intestine and measured as the sum of

sugar and starch, excluding RS. This classification will be reviewed further in the literature section. Resistant starch fractions 1 and 2 are included in the Association of Official Analytical Chemists (AOAC) method of analysis for measurement of residue of plant materials after solvent extraction, digestion with dilute acid and alkali and correction for minerals (Olson *et al.*, 1987). This is deemed as incorrect in terms of the required definition of available carbohydrate (FAO/WHO, 1998; Englyst *et al.*, 1987). The methodology states that a 50g available carbohydrate portion of the test and standard food be administered to an individual in random order on different days after an overnight fast.

Type 1 diabetes, type 2 diabetes or healthy subjects may be included. Glucose was originally used as the standard food. Due to the osmotic effect, which may lead to delayed gastric emptying, the presence of nausea and perceived stimulation of cortisol secretion (which in turn may increase blood glucose levels, as stated by Thompson *et al.*, 1982), it was suggested that white bread of known composition be utilized (Wolever, 1990). White bread in contrast contains some protein and thus stimulates insulin secretion, despite the lower blood glucose responses. Also, variation in the composition and digestibility characteristics of white bread according to location and time would reduce its usefulness as the reference food (Wolever *et al.*, 2003). Despite this, the variability of the GI values for bread was similar to those for the other foods. Nevertheless, glucose is a more logical and easily standardized reference food for international use. Thus, for international standardization, it is recommended that GI values of foods be expressed relative to glucose (Wolever *et al.*, 2003). Consequently, results can be adjusted depending on the standard used. Multiplication by a conversion factor of 0.7 is used for white bread to compare it to a glucose standard and 1.4 for glucose to compare it to that of white bread (Wolever, 1990).

It is recommended that blood glucose levels are measured via capillary blood sampling as greater within-subject variation of both glycaemic responses and GI values occurs with venous plasma samples (Wolever *et al.*, 2003). Capillary samples are taken at fasting level and then every fifteen to thirty minutes for two hours in non-diabetic subjects and up to three hours in diabetic subjects, respectively. The normal dose of insulin or oral hypoglycaemic agent (if any at all) is taken after the fasting blood sample and 5 - 10 minutes before starting to eat the test meal (Wolever *et al.*, 1991). Glycaemic index values also vary from centre to centre and it is for this reason that attempts have been made to standardize methods (Wolever, 2003). Several methods of calculating the GI exist. The method used most often in

the scientific literature is that of Wolever *et al.* (1991), which states that the incremental area under the blood glucose response curve (IAUC) is that area above the fasting blood glucose. Any area below the fasting blood glucose concentration is ignored (Wolever *et al.*, 1991).

The GI value of a food is determined by repeating the procedure with a number of subjects. The resulting values for each subject are averaged to obtain the GI value for the food (Wolever *et al.*, 1991). The formula stated above is applied to calculate the GI (Jenkins *et al.*, 1981). The GI ranges are then categorized when glucose is used as standard as follows: low GI foods-below 55, intermediate GI foods-between 55 and 70 and high GI foods-more than 70 (Brand-Miller *et al.*, 1996).

In addition to the above, and of particular consequence, is the factor of intra and inter-individual variation, although not researched in this study. Individual variations exist in the glycaemic response to foods due to differences that arise among individuals and within the same individual (Wolever, 1990).

Type 2 diabetic subjects are the least variable followed by normal and then type 1 subjects who are nearly twice as variable as type 2 subjects (Wolever *et al.*, 1985). Botes (2000) concluded from her study that three repeats of the standard food which is preferably white bread should be used to reduce variation.

Nell's (2001) study involving normal, healthy subjects led to the findings that high intra and inter-individual variations for both glucose and white bread occur. Nell (2001) recommended that larger subject groups be used rather than repetitive tests due to week-to-week intra-individual variations not being smaller than the variation between individuals. The symptom/presence of nausea and possible stimulation of cortisol secretion (a stress hormone), as already mentioned, may in turn increase blood glucose levels on different test occasions in the same individual (Nell, 2001; Thompson *et al.*, 1982).

It was concluded from the studies of Wolever *et al.* (2003) that finding ways to reduce within-subject variation in glycaemic responses may be the most effective strategy to improve the precision of measurement of GI values.

Other factors that may affect the glucose response include subject characteristics, for example, age, gender, body mass index (BMI), glucose tolerance status, dose and timing of insulin or oral hypoglycaemic agents, the degree of diabetes control - particularly in type 1 diabetics and the fasting blood glucose value on the day of the test (Wolever *et al.*, 1991).

Rasmussen *et al.* (1992) researched the influence of gender on glucose and insulin responses in patients with type 2 diabetes. Their study did not demonstrate a significant influence of gender on glycaemic and insulinaemic responses in middle-aged subjects with type 2 diabetes matched for age, duration of diabetes (BMI) and metabolic control. Nielsen and Nielsen (1989) found that preprandial blood glucose (PPBG) >13mmol/L were negatively correlated to the net glycaemic responses. Thus, they recommend that participants with PPBG >13mmol/L be excluded from such studies and that the number of participants be increased substantially which concurs with the recommendation of Nell (2001).

2.2.1 The glycaemic load

The term glycaemic load (GL) was introduced in 1997 by researchers from Harvard University and is defined as the product of the amount of available carbohydrate in that serving and the GI of the food (Salmerón *et al.*, 1997a,b). The higher the GL, the greater the expected elevation in blood glucose and in the insulinogenic effect of the food. The 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) (Liu *et al.*, 2000). An example of a food with a high GI but low GL is that of pumpkin. The GI of pumpkin is 75. However, a serving size of 80g is recommended which denotes 4g of available carbohydrate resulting in a GL of only 3. It is thus unnecessary to exclude the above and many other fruit and vegetables due to this concept (Foster-Powell *et al.*, 2002). The GL is categorized as follows: low GL<143, medium GL 143-165 and high GL>165 (Salmerón *et al.*, 1997a,b).

2.3 FACTORS INFLUENCING THE GLYCAEMIC INDEX

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 response. It is for these reasons that a 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 GI (Wolever, 1991).

2.3.1 Non- food factors

Non-food factors include processes such as chewing and swallowing. Chewing leads to the reduction in food particle size which increases absorption rates as well as constituency of food such as bread. Pasta, on the other hand, retains its structure on swallowing which slows the absorption (Jenkins, 1988a).

Apart from the above processes, gastric emptying (GE) is the major determinant of nutrient delivery to the small intestine and variation in the rate of GE accounts for 35% of the variance in peak blood concentration after ingestion of 75g of oral glucose in both healthy and type 2 diabetic subjects (Horowitz *et al.*, 1993).

Of particular interest is the effect that time of the day has on glycaemic response. Wolever and Bolognesi (1996b) questioned this aspect and subsequently studied the comparison between glycaemic responses measured at breakfast and lunch time. The researchers found that breakfast glycaemic responses were less variable. Lunch time responses were influenced by many factors, one of which was the nature and composition of the previous meal (second meal effect). They concluded that these findings indicate that the interpretation of studies would also be affected. Physical activity as well as ingestion of various diets are also contributing factors (Vorster *et al.*, 1990).

2.3.2 Food factors

Historically “complex” carbohydrate has been thought to be beneficial in slowing the glycaemic response. Absolute elimination of sucrose as well as limited intake of “simple” carbohydrates was advocated by various diabetes associations (Wolever & Brand-Miller, 1995).

However, Jenkins *et al.* (1981) showed different effects in their studies. Their studies resulted in the finding that sucrose elicits a lower glycaemic response than glucose, whole meal bread, muesli and many other starchy foods. In contrast fructose produces a lower glycaemic response than sucrose. Heacock *et al.* (2002) observed that 10g of fructose fed 30-60 minutes prior to a meal of instant mashed potatoes lowered the glycaemic responses as compared to either immediate or no fructose treatments. The possible benefit is that this

aspect could result in a practical application as this amount is easily obtainable from fruit. The question is what amount is deemed optimal? Galactose, on the other hand, is actively absorbed in the small intestine and is converted to glucose in the liver. However, very little glucose appears in the blood after oral or intravenous galactose. Glycaemic response of the latter is much lower in the presence of glucose as both galactose and glucose compete for active transport (Wolever & Brand Miller, 1995).

Wolever and Brand Miller (1995) reviewed various studies concerning the addition of sucrose to other foods and the comparison of sucrose and naturally occurring sugars. Evidently the glycaemic response would only increase if the carbohydrate content was not reduced and if it was dependent on the existing GI of the food. In other words, the glycaemic load would be affected. Sugar that occurs naturally in fruit and fruit juices has approximately the same effect as sucrose.

Vorster *et al.* (1987) investigated the effects of the addition of 10%, 20% and 30% sucrose to cooked dried butter beans on taste preference and acceptability in 29 diabetic patients and 11 control subjects. The addition of 10% and 20% of total carbohydrate as sucrose rendered no adverse effects on the GI of the bean dishes. It was, therefore, proven in this study that the addition of moderate amounts of sucrose to a low GI food may improve palatability (since the diabetic patients preferred the beans with added sucrose) without detrimental effects on the glycaemic response. The 30% additions increased the GI from 28.8 to 53.7, which was significant on a 5% level.

Finally, Brand Miller and Lobbezoo (1994) tested the hypothesis that replacing starch with sugar in a processed breakfast cereal that has a high GI could significantly decrease glycaemic and insulin responses. Amounts of 0g, 21g and 43g of sucrose were added, respectively, to puffed rice cereal. The glycaemic and insulin responses to the meal containing 43g of sucrose were significantly lower compared with the non-sweetened cereal, the opposite being proven to what is commonly believed about addition of sugar to the diet. The meal that contained 43g of sugar produced lower glycaemic responses than the 21g sugar meal. The authors concluded that sweetened breakfast cereals may not compromise glycaemic control more than the unsweetened counterpart and that total avoidance of sucrose replaced by high GI foods may lead to higher levels of postprandial glycaemia. Another important factor affecting the glycaemic response is that of the different carbohydrate

fractions. A chemically based classification was proposed by Englyst *et al.* (1999) which divides dietary carbohydrate into sugars, starch fractions and non-starch polysaccharides (NSP) and which groups the latter 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.

2.3.3 Chemical structure of carbohydrates

Differences in starch structure also affect the glycaemic response. Amylopectin and amylose are both polymers of glucose which occur in a branched and linear form respectively. Studies have shown that the open, branched structure of amylopectin starch makes it easier to digest than the (linear) amylose starch (as reviewed by Wolever, 1990).

Legumes contain 30% to 40% amylose and 60% to 70% amylopectin in their starch granules, while most other carbohydrate foods contain 25% to 30% amylose and 70% to 75% amylopectin (Thorne *et al.*, 1983). Therefore the amylose would lead to induction of a decreased postprandial plasma glucose response compared to the amylopectin (Behall *et al.*, 1989; Byrnes, 1995; Granfeldt *et al.*, 1994).

All of the above researchers concluded that the higher amylose content was responsible for the decreased rise in postprandial glucose and insulin response. Additionally, a high-amylose diet also resulted in significantly lowered fasting triglycerides and cholesterol levels (Behall *et al.*, 1995).

The results of Byrnes *et al.* (1995) suggested that amylopectin leads to the development of insulin resistance in rats. Granfeldt *et al.* (1994) proposed that the 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.

2.3.4 Dietary fibre and resistant starch

Numerous epidemiological studies have shown that ingestion of high-fibre foods reduces the risk of type 2 diabetes and CHD and it was, therefore, recommended by various diabetic associations that diets contain fibre-repleted foods (Wolever, 1990). It was hypothesized that low-fibre diets lead to higher glucose levels due to rapid absorption (Jenkins *et al.*, 2000). In contrast, the complex network of fibre renders the food particle less accessible for absorption. Non-digestible complex carbohydrates are commonly known as dietary fibre, although the correct terminology is NSPs (Englyst *et al.*, 1987). Non-starch polysaccharides are divided into soluble and insoluble fibre, although this term does not denote physicochemical characterization (Anon, 2002).

Leguminous seeds are rich sources of dietary fibre (Vorster & Venter, 1994) and the seed fibre guar gum has been shown to reduce urinary glucose loss in diabetics (Jenkins *et al.*, 1980b). Pulses, which exhibit the lowest GIs also have the most resistant cell walls (Jenkins *et al.*, 1980b). Certain factors, other than fibre, which will be discussed further in Section 2.3.5 and 2.3.9, may also contribute to the reduced glycaemic response of legumes (Thorne *et al.*, 1983). Other than the cell wall components, the β -glucans found in oats may also result in a reduced glycaemic response. Due to their high viscosity, gums and mainly guar gums also exhibit this property (Guillon & Champ, 2000). Certain treatments can lead to reduced viscosity, which in turn reduce the benefits as shown by Granfeldt *et al.* (1995). Their studies resulted in the finding that neither incomplete gelatinization in rolled oats nor naturally occurring viscous dietary fibre in oats affected post-prandial glycaemia positively, unless the oats and wheat kernels remained intact (Granfeldt *et al.*, 1995).

When a common barley, oats or barley genotype containing high levels of β -glucan was tested (Liljeberg *et al.*, 1996b) to determine the respective glycaemic responses, it was found that common oats or barley porridges produced similar glycaemic and insulin responses to that of white bread. The high fibre (20g/100g) barley genotype induced significantly lower glycaemic responses. The authors further suggested that enrichment of cereal products with such a genotype with high β -glucan content would be favourable and acceptable to enhance fibre intake.

Van der Sluijs *et al.* (1999) had showed similar but less detrimental effects in their study. Various cooling processes were undertaken on puddings containing high β -glucan content. The β -glucans in the boiled and baked preparations, although still producing beneficial effects on plasma glucagons, are not as effective as soluble β -glucans that are not cooked.

Another major component of dietary fibre is that of arabinoxylan (AX) of which wheat grain is a rich source. The NSPs in wheat bran are \approx 64-69% AX and 15-31% cellulose, whereas NSPs in wheat endosperm are \approx 88% AX. The physiologic effect of AX is unknown, therefore, Lu *et al.* (2000) studied the effect of AX fibre extracted from the by-product of wheat flour processing on glucose and insulin responses in humans. Addition of as little as 6g AX-rich fibre to bread in a breakfast meal significantly lowered postprandial glucose and insulin responses in healthy persons. The mechanisms by which AX-rich fibre flattens the postprandial glucose response are as yet unknown, but because AX is a soluble fibre, it is likely that its effects are exerted similarly to other such fibres. Moreover, the low-GI, AX-rich fibre bread proved to be palatable and acceptable to subjects. The authors concluded that further research is required to determine whether AX-rich fibre is of benefit to people with type 2 diabetes.

Munari *et al.* (1998) investigated the effect of the ingestion of 15g Plantago Psyllium mucilage and an α -glycosidase inhibitor (acarbose) on the GI of white bread. Both P. Psyllium and acarbose led to a decreased GI of bread, the effect being greater with acarbose, which the authors state is dose dependent. They concluded that further studies are required to ascertain whether these substances can be used to impair intestinal absorption of carbohydrates.

The removal of fibre from food and also its physical disruption such as shown in the study by Haber *et al.* (1977), resulted in apple juice being consumed 11 times faster than intact apples and four times faster than apple puree. Similarly, grinding raw legumes and then cooking them would destroy the seed and seed coat, possibly allowing faster and greater swelling of the starch granules (Thorne *et al.*, 1983). Studies on the disruption of fruit fibre in grapes and oranges by Bolton *et al.* (1981) led to the suggestion that glucose and insulin responses to the latter was due not only to the fibre but fructose content as well. Most pectins are of high viscosity and those which have ion exchange capacity also exhibit similar effects (Guillon & Champ, 2000).

The mechanisms involved in the effect of fibres in the glycaemic response are multiple and dependent 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 substrates as a result of increased viscosity of gut contents; modified intestinal motility, as well as slower gastric emptying (which has been suggested as the major factor, although it is still being debated). Léclere *et al.* (1994) tested the role of guar gum in lowering the glycaemic response and found that guar gum reduced the rate of starch degradation by pancreatic amylase and slowed gastric emptying.

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).

The effect of RS, which is defined as “the fraction of starch that passes undigested to the large bowel” (reviewed by Englyst *et al.*, 1987), was investigated to determine its effect on glycaemic response. Venter *et al.* (1990) in their study found that resistant starch in cooled maize porridge resulted in a significantly smaller response in blood glucose than hot or reheated porridge. The GI of the cooled maize porridge was also the lowest of the three meals. The authors recommended that the GI of porridge made with high amylose maize meal be studied.

Raben *et al.* (1994) undertook a similar study whereby 54% RS (in raw potato starch) was compared to pregelatinised starch (0% RS). The results were reproducible as there was a significant lowering of postprandial plasma glucose with ingestion of the 54% RS product. Resistant starch is also formed during the baking process of which 1.7% is found in the crust of bread (Rabe & Sievert, 1992). Conclusive further studies are needed to clarify the effect of RS in a mixed meal. In essence it is still advisable to recommend that the diabetic population ingest 25-30g of diverse types of fibre sources daily as the latter has many other benefits (Guillon & Champ, 2000).

2.3.5 Anti-nutrients

Acarbose is a stable, α -glucosidase inhibitor of bacterial origin which delays the digestion and intestinal absorption of sucrose and starches (Munari *et al.*, 1998). Munari *et al.* (1998, as described in Section 2.3.4) also incorporated acarbose into their study to determine the effect

glycaemic response curves.

However, the amount of fat was not sufficient to reduce the overall glycaemic response. Several factors may account for the influence of dietary fat on glucose and insulin responses, such as differences in GE, where it has been shown that the GE of a protein and

carbohydrate meal is delayed either by adding fat or by infusing lipids into the ileum or duodenum (as reviewed by Wolever, 1990). Dietary fatty acids may also interact with food digestion by modulating digestive enzyme activities. A study by Armand *et al.* (1995) showed that a high-fat diet compared to a low-fat diet had a tendency for higher output of gastric lipase and significantly increased the gastric lipase activity in healthy humans.

Joannic *et al.* (1997) examined the influence of the type of fat on glucose and insulin responses. Two kinds of fat were used, namely monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs), combined with rice or mashed potatoes that had a GI of 47 and 83 respectively. The total fat was 47% of total energy intake. Polyunsaturated fatty acid meals produced significantly lower postprandial glucose and insulin responses compared with the MUFA meals, regardless of the type of carbohydrate ingested. The MUFA meals resulted in similar glucose responses regardless of the GI of the carbohydrate. The degree of unsaturation of a fat plays a conclusive role in influencing the metabolic response and the greater the degree of unsaturation, the more profound the insulin secretion, which, as mentioned previously, is unfavourable. Whether this effect persists when the fat content is lower is debatable (Joannic *et al.*, 1997). The authors concluded that in healthy subjects, the GI of the CHO contained in mixed meals cannot accurately predict the glucose and insulin responses because the degree of unsaturation of dietary fatty acids also influences these metabolic responses.

2.3.7 Organic acids

The effect that the addition of lactic acid or sodium propionate had on bread was of particular interest (Liljeberg & Björck, 1996a). Sourdough fermentation resulted in a flattened postprandial blood glucose and insulin rise. A delayed gastric emptying might also have occurred as a result of the above organic acids and salts (Liljeberg & Björck, 1996a). The researchers suggested that the role of these acids and salts should be explored further in the food industry. Mbhenyane (1997) added tartaric acid to sorghum porridge so as to produce a similar fermented sorghum. This addition was found to decrease the GI by 43% compared to the fermented product.

Sodium acetate and acetic acid from vinegar produce the same effect as the above acids and salts, but sodium acetate has more profound effects (Brighenti *et al.*, 1995). Brighenti *et al.* (1995) concluded that the mechanism by which vinegar influences glycaemic response to a mixed meal is related to acidity but not to gastric emptying.

Östman *et al.* (2001) examined the possible effects of regular milk and lactic acids in fermented milk products on the glycaemic and the insulinaemic responses as well as the acute metabolic effect of fermented milk (yoghurt) and pickled cucumber as supplements to a traditional breakfast based on a high GI bread. Firstly, the lactic acid in the fermented milk products did not lower the GI and insulinaemic indexes (II) and regular milk products led to high IIs despite having low GIs. Secondly, the yogurt and pickled cucumber meal lowered postprandial glycaemia and insulinaemia, whereas the addition of regular milk and fresh cucumber had no favourable effect on the metabolic responses (Östman *et al.*, 2001).

2.3.8 Other food factors

Wolever (1990) states that the composition of fruits changes as ripening occurs and quotes a study by Englyst and Cummings (1986), where it was found that the starch content of unripened banana is 37% but decreases to 3% when ripe. These findings and others concerning ripening, food storage and various cultivars, suggest that there are many factors that cannot be controlled (Wolever, 1990).

Lastly, the volume and type of beverage consumed with a test meal may affect the blood glucose responses. Young and Wolever (1998) embarked upon a study in which 12 normal subjects ate a standard meal with 50, 250, 500, 750 or 1000ml water or 250ml coffee or tea. The findings resulted in the conclusion that the volume and type of beverage consumed with a test meal influence the pattern of blood glucose response but has no effect on the IAUC. The authors suggest that a standardized volume of beverage must be established in order to design a definitive procedure for blood glucose testing.

2.3.9 Processing

Various methods of processing such as milling, extrusion cooking and puffing, flaking and rolling lead to structural changes in the food particle rendering a product with a higher GI (Brand *et al.*, 1985). Gelatinization of starch granules is an obvious process whereby enzymes have greater opportunity to degrade the starch leading to rapid digestion and absorption. Lintas and Cappelloni (1992) found that cooking resulted in 69% to 84% of legume starch becoming RAG.

Collings *et al.* (1981) confirmed that cooked starch produces greater glucose and insulin response in comparison to raw starch. The aim of a similar study undertaken by Ross *et al.* (1987) was to determine the glycaemic and insulin responses of seven processed wheat products. The conclusion was that the degree of starch gelatinization and processing led to differences in glycaemic and insulin responses.

Reproducible results were also shown in a study by Granfeldt *et al.* (2000). The importance of the degree of gelatinization and product thickness in rolled oats and barley on postprandial glycaemic and insulinaemic responses was examined. Conclusively, all thin (0.5mm) flakes elicited high glucose and insulin responses and all varieties of thick oat flakes gave significantly lower metabolic responses, thus implying once again that the degree of gelatinization and product thickness affects the final GI.

The influence of particle size is also of importance and this aspect was investigated by Jenkins and colleagues (1988b). They found that breads containing cracked or whole cereal grains produced lower postprandial glycaemic responses when substituted for milled flour. On a positive note, the wholegrain bread was not less palatable compared to white bread.

Researchers such as Liljeberg *et al.* (1992), Holt and Brand-Miller (1994) and Behall *et al.* (1999) undertook similar studies where the effect of various particle sizes was examined on postprandial glycaemic and insulin responses. Holt and Brand Miller (1994) and Behall *et al.* (1999) studied different grades of whole grain flour and Liljeberg *et al.* (1992) compared coarse bread made from wheat, rye and barley (intact kernels) to that of whole meal barley and white wheat flour respectively. As expected, decreased particle size led to increased glycaemic and insulin responses in all three studies (Behall *et al.*, 1999; Holt & Brand Miller, 1994; Liljeberg *et al.*, 1992).

Mourat *et al.* (1998) (as quoted by Liljeberg *et al.*, 1992) state that once particles have been reduced to less than 2mm, digestible solids empty from the stomach and thus encapsulated starch in legumes is the reason that they exhibit “lente” properties.

A study by Järvi *et al.* (1995) compared not only bread (such as that used in the above studies), but also parboiled rice versus sticky rice and red kidney beans versus the ground product. Similarly, products undergoing various processes resulted in different glycaemic responses. It is clear from the above research that it is imperative to maintain botanical structure to induce lowered glycaemic and insulin responses.

2.3.10 The influence of a mixed meal on the GI

Coulston *et al.* (1984b) reported that glycaemic responses to mixed meals did not differ significantly, rendering the GI of minimal clinical importance. Wolever *et al.* (1985) refuted these conclusions as there were no significant differences in the glycaemic and insulin responses to three of the four mixed meals tested in their study.

Parillo *et al.* (1985) assessed whether foods containing similar amounts of carbohydrate with different glycaemic responses render differing glycaemic responses when consumed in a mixed meal. The test meals of pasta, bread and potatoes were ingested with a standardized meal. The researchers showed to the contrary that the glycaemic response was significantly higher after ingestion of bread than after the spaghetti meal. The glycaemic response to the potato meal was similar to that for bread at 2 hours and intermediate between the two other test meals at 5 hours. Collier *et al.* (1986) found that the relative glycaemic effects of mixed meals can be predicted from the GI of their carbohydrate components.

Coulston *et al.* (1984a) also states that the GI concept neglects the insulin responses and has not been studied in the context of mixed meals. It was for this reason that Bornet *et al.* (1987) were one of the first to study both the GI and II of foods taken alone and in a mixed meal. They concluded with the statement 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 II does not bring greater discrimination between carbohydrate foods, but remains of interest in physiological studies.

Chew *et al.* (1988) reported that there were significant differences in the glycaemic and insulin responses of healthy individuals to different mixed meals of ethnic origins. The glycaemic and insulin indices were highest for the Lebanese meal (unleavened bread, hummus, falafel and tabouleh) and lowest for the Greek meal (lentil stew). The authors caution that these results might not always be reproducible due to varying amounts of fat and protein content, different processes and methods of cooking.

In conclusion, Wolever and Bolognesi (1996c) in a later study showed that both amount and source of carbohydrate determine the glucose and insulin responses of lean, young, non-diabetic subjects after different mixed meals with variable GI and, furthermore, variation in protein and fat intake over the range as tested in this study appeared to have negligible effect on postprandial glucose and insulin.

2.4 PHYSIOLOGICAL AND THERAPEUTIC IMPLICATIONS OF THE GLYCAEMIC INDEX

2.4.1 Second meal effect

The long-term clinical benefit of the GI has been questioned and for the GI to exhibit any clinical utility it was advised that not only the metabolic effect, but also the mechanisms be proven (Wolever, 1990). Subsequently, studies were undertaken to determine the effect of slow absorption of carbohydrate over a longer period of time as the immediate reduction in glycaemic response is understood (Wolever, 1990). This was then termed the second-meal effect (Jenkins *et al.*, 1982). The second-meal effect is based on the hypothesis that rapid absorption of carbohydrate leads to a large rise in blood glucose as well as insulin secretion. The large insulin response causes peripheral glucose utilisation to increase to such an extent that absorption from the gut cannot keep up and that the blood glucose level undershoots the baseline. This, in turn causes a counter-regulatory response with a rise in free fatty acid (FFA) levels and relative insulin resistance. Slow and prolonged carbohydrate absorption, on the other hand, leads to a slower increase in the blood glucose level, a smaller insulin response and less of a tendency for the blood glucose to undershoot. This causes a smaller counter-regulatory response and improved glucose disposal after the next meal (Wolever,

1990). Jenkins and Co-workers (1980a) were of the first researchers to provide evidence for the proposed mechanism of the second-meal effect, where guar gum was used to slow absorption of glucose. Free fatty acids and β -hydroxybutyrate levels were lower four hours after the guar-containing test meal than after the glucose test meal alone.

The second-meal effect was also demonstrated between breakfast and lunch by Jenkins *et al.* (1982) in a subsequent study. Breakfasts consisting of lentils (low GI) or whole meal bread (high GI) were compared to determine their effect on a standard lunch eaten four hours afterwards. The results proved that a low GI breakfast improved the carbohydrate tolerance of the meal which followed as the blood glucose response to the lunch was significantly less than that of the meal following the high GI breakfast (Jenkins *et al.*, 1982). The same effect was created by slowly nibbling the same GI breakfast over the entire 4-hour period.

Liljeberg *et al.* (1999) also examined this concept by testing the effects of the ingestion of indigestible carbohydrate (RS and dietary fibre) content of seven breakfast cereals with known GIs (ranging from 52-99) on the glucose tolerance at a subsequent meal (lunch) in healthy subjects. Two of four low GI test meals improved the glycaemic response as well as the in-between meal fasting states, which may be an important determinant of improvements in four-hour second-meal glucose tolerance. The researchers concluded that slow absorption and digestion of starch from the breakfast meal, but not content of indigestible carbohydrates in the breakfast meal, improved glucose tolerance at lunch and within a single day. Furthermore, they stated that postprandial glycaemia may be an important factor in the cumulative metabolic effect of starchy foods and that foods with similar GIs of between 52 and 64 may differ in their capacity to modify second-meal glucose tolerance. The mechanisms remain to be clarified. A further, similar study by Liljeberg and Björck (2000) led to evidence in support of the influence of a low GI spaghetti meal on a subsequent lunch where improved glucose tolerance was observed.

The effects of low GI carbohydrates consumed the previous night on the glycaemic responses to a standard test meal eaten at breakfast were studied by Wolever *et al.* (1988). The glycaemic responses to breakfast were significantly lower on mornings after the low GI dinners than after high GI dinners. Wolever *et al.* (1988) are of the opinion that breakfast carbohydrate tolerance is improved when low GI foods are eaten the previous evening.

Finally, in a more recent study, Östman *et al.* (2002) evaluated whether a low GI breakfast with lactic acid had an effect on glucose tolerance and insulinaemia at a subsequent high GI lunch meal. Significant decreases of the incremental glycaemic area and of the glucose response at 95 minutes were found after the lunch meal when the barley bread with lactic acid was given as a breakfast. The insulin level after 45 minutes was also significantly lower after the test meal compared to the meal without lactic acid (Östman *et al.*, 2002).

2.4.2 Blood glucose and insulin resistance

The role of carbohydrate and subsequently the GI in insulin resistance have been studied at length. There is sufficient and strong evidence linking the metabolic disorder with disease risk which includes diabetes, hypertension, CHD and obesity (Bessesen, 2001). Insulin action is complex and controversies surrounding carbohydrates and insulin sensitivity still remain. Insulin stimulates the disposal of ingested glucose into skeletal muscle and adipose tissue and decreases the production of glucose by the liver by reducing glycogenolysis and gluconeogenesis. Insulin also suppresses the release of FFA from adipose tissue by suppressing lipolysis (Bessesen, 2001; Frost & Dornhorst, 2000). Recent studies in β -cell specific insulin receptor knockout (BIRKO) mice have shown loss in insulin secretory capacity after removal of insulin receptors and development of a syndrome similar to type 2 diabetes (Bessesen, 2001). Furthermore, neural insulin receptor knockout (NIRKO) mice were likewise to present with obesity, mild insulin resistance and hyperinsulinaemia. The results of the study suggest that pancreas, brain and adipose tissue play an important role as insulin-sensitive organs (Bessesen, 2001).

Several recent studies suggest that diets which have a low GI may improve insulin sensitivity by their ability to reduce adipocyte FFA release (reviewed by Frost & Dornhorst, 2000) and that consuming a low GI diet may be associated with a lower risk for type 2 diabetes (Bessesen, 2001; Salmerón *et al.*, 1997a,b). The metabolic effects of increased circulating FFA are multiple, some of which include adverse lipoprotein and coagulation changes and lipotoxic effects on the β -cell. A relationship between increased adipocyte FFA release and insulin resistance has been shown in subjects with coronary heart disease (CHD) (reviewed by Frost & Dornhorst, 2000).

Bessesen (2001) points out that the amount of carbohydrate and influence on glucose tolerance is also of importance. A prospective study by Swinburn *et al.* (2001) showed that a low-fat (26% of energy) high-carbohydrate (54% of energy) diet was associated with improved glucose tolerance. Bessesen (2001) concludes that fat (in particular saturated fat) appears to promote insulin resistance in animals and that low GI diets or RS may prove to be beneficial at some stage in development of type 2 diabetes. This statement, however, is still controversial.

Jenkins *et al.* (1987) were the first to conduct a study on the metabolic effects of a low GI diet versus a high GI diet. These diets were given in random order to healthy subjects over a two week period. A 37% reduction in mean postprandial glycaemia was observed during the low GI period with a concomitant 47% reduction in insulin secretion as measured by C-peptide excretion. In addition to this, a significant reduction occurred in the glycosylated serum protein (fructosamine) levels, a marker of the average blood glucose level per day, with a low GI diet. A further reduction in fructosamine levels might have taken place had the studies continued for longer than three weeks.

Jenkins *et al.* (1987) also observed reduced urinary creatinine and urea outputs on the low GI diet, as well as blunted postprandial amino acid responses. This may possibly imply that the latter diet may be associated with reduced renal perfusion, which in early renal disease, for example, may be advantageous in maintaining renal function.

A subsequent study by Jenkins *et al.* (1988a) investigated whether a low GI diet utilised in the short term (i.e. two weeks) in type 2 diabetics accrued any benefit. Over the low GI period, significant reductions were observed in fasting blood glucose, glycosylated haemoglobin (HbA1c), serum fructosamine and urinary C-peptide:creatinine ratio. In contrast, no significant changes in the above measurements were found over the high GI period.

Järvi *et al.* (1999) also evaluated the effects of both low and high GI diets on metabolic control in type 2 diabetic patients. Both test diets comprised similar energy, macronutrient and fibre content. During both the low and high GI period the plasma glucose concentration fell significantly (by 14%). The effect observed during the latter period could have been attributable to the current prescribed dietary recommendations of the ADA (1994), which stresses that the total amount of carbohydrates rather than the source of carbohydrate consumed is of priority. Significant changes also occurred in serum fructosamine and insulin

sensitivity. C-peptide levels were significantly higher with the low GI diet, compared with the high GI diet. This study further confirms the results as found by previous researchers. Giaco *et al.* (2000) researched the feasibility of long term treatment with fibre-rich, low GI foods on glycaemic control and incidence of hypoglycaemic events in type 1 adult diabetic patients. The study took place over a period of 24 weeks. Subjects also received acarbose as part of another study. The mean daily blood glucose concentration was significantly reduced and HbA1c levels were lower, but not of statistical significance. In addition to this, the number of hypoglycaemic events were significantly lower compared to that of a low-fibre, high GI diet. Giaco *et al.* (2000) also point out that acarbose did not affect any of these results and that the test diet was practical and reproducible in a normal clinical setting.

A study of similar design was embarked upon by Gilbertson *et al.* (2001). The subjects were type 1 diabetic children and the carbohydrate exchange diets were compared to a low GI diet over twelve months. Those subjects consuming the low GI diet had significantly better HbA1c levels than those following the carbohydrate exchange diet. There were also significantly lower rates of excessive hyperglycaemia and these results were not related to variations in insulin therapy. Hypoglycaemic episodes were also not increased (Gilbertson *et al.*, 2001).

In this review it is important to discuss not only the relationship between the GI and risk of type 2 diabetes but also that of the glycaemic load (GL). The reason is that the GI only represents the quality of carbohydrate and not the quantity. The GL, on the other hand, denotes a combination of both quantity and quality. For this reason Salmerón *et al.* (1997a,b) set out to design and conduct a prospective study over a period of six years to examine the relationship between high GL and low cereal fibre content with risk of type 2 diabetes. The GL is an indicator of a global dietary insulin demand. The results proved that diets with high GL and low cereal fibre content were positively associated with risk of type 2 diabetes, independent of other dietary factors and currently known risk factors (Salmeron *et al.*, 1997a,b).

2.4.3 Coronary heart disease

Coronary heart disease is the most common cause of death in Western society and is increasing. The relationship between CHD and fat intake is well established, but the role of

carbohydrate is less well explained. Concern has arisen that high carbohydrate diets promote hypertriglyceridaemia and lower insulin sensitivity, but this occurs only when high GI foods are ingested (Frost *et al.*, 1999), with the exception of fructose and sucrose in dietary amounts of > 20% energy and > 35% of energy, respectively. This effect is, however, dose dependent (Anderson, 1997). Numerous studies have been undertaken to examine the effect of low GI versus high GI diets on various risk factors known to worsen the incidence of CHD. The risk factors studied were amongst others: total cholesterol, LDL and HDL cholesterol, TG, apoprotein B and A-1 and plasminogen activator inhibitor-1 (PAI-1) (Oosthuizen, 1999).

Jenkins *et al.* (1987) compared the effect of a low versus a high GI diet on serum lipids. Total serum cholesterol levels decreased on the low GI diet by $15\pm 3\%$ ($p < 0.01$). A significant decrease was also seen in serum LDL cholesterol on the high GI diet, but the change was identical to that seen in the low GI diet, which was not significant. No significant changes were seen in HDL cholesterol. Mean changes in serum TG were not significant and had decreased in five volunteers over the low GI period and increased in four subjects over the high GI period.

Frost *et al.* (1994) also studied the effect of a low GI diet on metabolic control in type 2 diabetic patients. Results indicated a significant decrease in total serum cholesterol, from 6.1 mmol/L to 5.4 mmol/L in the low GI group. Järvi *et al.* (1999) conducted a similar study in type 2 diabetic patients and serum cholesterol was reduced in both dietary groups (low GI diet versus that of current recommendations by the ADA, 1994). However, the reduction in subjects on the low GI diet was significantly more pronounced than those on the high GI diet (-5% , $p < 0.01$). Serum TG values were reduced to the same extent on both diets. HDL cholesterol levels were also decreased after both diet periods. LDL cholesterol was reduced by 29% ($p < 0.01$) after the low GI diet and by 22% ($p < 0.01$) after the high GI diet when compared with that on admission. When comparing the two periods, LDL cholesterol was 8% ($p < 0.01$) lower on the low GI diet than on the high GI diet. PAI-1 activity decreased by 58% on the low GI diet but remained unchanged on the high GI diet. Reductions in PAI-1 activity may have been due to the reduced insulin levels rather than to decreased TG levels. ApoA-1 decreased significantly over both periods, but slightly more over the low GI period. Apoprotein B concentration was markedly reduced after both diet periods but significantly more after the low GI period. The fasting values of FFA showed no changes, but significant differences occurred between the dietary periods throughout the day (Järvi *et al.*, 1999).

Frost *et al.* (1999) further examined the role of the GI as a determinant of serum HDL cholesterol concentration. There were no significant negative relations between HDL cholesterol concentration and both total carbohydrate intake and GI and the association was stronger in women than men. Frost *et al.* (1999) state conclusively that these findings were compatible with the hypothesis that a low GI diet increases HDL cholesterol concentration by improving insulin sensitivity. Liljeberg and Björck (2000) in their study on the second-meal effect showed no differences in total serum cholesterol and HDL cholesterol, however, the TG level was reduced in the subsequent meal.

Leeds (2002) reviewed various studies concerning this aspect and asserted that prospective (>4 weeks) as well as long-term trials with clinical endpoints should be undertaken to ascertain the true clinical value of low GI diets in relation to CHD.

The role of GL in CHD had not been researched in humans until Liu *et al.* (2000) embarked upon a ten year follow-up (prospective study) of healthy subjects to investigate the relation of GL with CHD. This was known as the Nurses Health Study in which 75521 female subjects participated. Glycaemic load contribution was mostly mashed or baked potatoes and cold breakfast cereals. Adjustments for other CHD risk factors were made and a high GL was significantly associated with increased risk of CHD and was most evident among women with (BMI) > 23kg/m², whereas little relationship was found among women with BMI < 23kg/m². GI was a stronger predictor of CHD risk than was simple versus complex carbohydrates.

In a review by Jenkins *et al.* (2002) the Zutphen study of 2000 showed no significant association of GI or GL and CHD, although there were discrepancies in the study design.

2.4.4 Obesity

Many diets are available to induce weight loss, but the question is whether they are sustainable and practical in the long term. In spite of high-carbohydrate, low-fat diets being advocated to reduce weight, obesity rates have still increased. Decreasing fat intake leads to a compensatory increase in carbohydrate ingestion, which tends to have high glycaemic and insulin responses (Ludwig, 2000).

High GI foods, as already mentioned, are rapidly absorbed resulting in decreased satiety. Many studies have examined whether low GI foods reduces hunger and/or promotes satiety,

as the latter is an important factor in any energy restricted diet to prolong the time between meals and reduce over-consumption (summarised by Roberts, 2000).

Holt *et al.* (1995) investigated the aspect of a satiety index (SI) of commonly eaten foods and assessed the relationship of satiety and metabolic responses to meals which were equivalent in composition. It appears that foods with a high SI such as legumes, oranges and apples are also low GI foods suggesting that a low GI diet may produce greater satiety and thus control energy intake. It has been hypothesized that consumption of high-GI foods promote a more rapid return of hunger due to induction of high circulating levels of insulin. The latter suppresses fat mobilization from adipose tissue further promoting hunger as implied by the Friedman model of energy regulation (Roberts, 2000).

Roberts (2000) reviewed short term studies which showed that there was no consistent effect of GI on satiety, but energy intake was nevertheless on average 29% less after consumption of low GI ones. Roberts (2000) included his own investigation in overweight adolescent boys in the review, which revealed that energy intake in the five hour period after consumption of two high GI test meals was 53% higher than after consumption of two medium GI meals. Consumption of energy after the high GI meals was also 81% higher than after consumption of low GI meals. Consumption of high GI foods may contribute to maintenance of excess weight in the obese and perhaps also to the etiology of weight gain in susceptible individuals (Roberts, 2000).

Brand-Miller *et al.* (2002) reviewed certain studies relating to the GI and obesity, one of them being that of Slabber *et al.* (1994). The study of Slabber *et al.* (1994) included diets with a prescription of 50% carbohydrate, 20% protein, and 30% fat for obese, hyperinsulinaemic women who were assigned to one of two treatment groups for 12 weeks. One of the groups was instructed to follow a diet which evoked a low insulin response (ID) and the other group followed a balanced low-energy diet (ND). Both diets supplied \approx 4200-5000 kJ. Both diets resulted in significant weight loss but the ID showed greater weight loss than the ND. The fasting insulin concentrations decreased more after ID compared with ND. In conclusion, the authors state that the mechanisms by which the ID causes more loss of weight and lower insulin response are not known and that an II could be of use for dietary planning (Slabber *et al.*, 1994).

Brand-Miller *et al.* (2002) further reviewed that healthy pregnant women also gained significantly more weight by full-term with the high GI diet than those following a low GI diet. Long-term studies (32 weeks) in animals have also been conducted and all results indicated conclusively that total fat mass was significantly higher and lipolytic capacity was reduced in those fed high GI meals compared to a low GI diet (Brand-Miller *et al.*, 2002). Epidemiological studies revealed lower waist-to-hip ratio and waist circumference in nearly 3000 type 1 diabetics consuming a low GI diet, independent of carbohydrate, fat and fibre intake (Brand-Miller *et al.*, 2002).

Agus *et al.* (2000) studied the effects of energy restricted low and high GL diets in an inpatient cross-over study of one week duration. The subjects were overweight to moderately obese young men and had maintained current weight for the previous 6 months. Subjects were observed over two days while following their usual diets, thereafter a high or low GL diet containing 50% of estimated total energy requirements was given. After six days it was found that resting energy expenditure was significantly lower after energy restriction on the high compared to the low GL diet. Negative nitrogen balance occurred on the high GL diet compared to the low GL diet. Agus *et al.* (2000) suggest that low GL diets may play a role in physiological adaptations to energy restriction in the long-term.

2.4.5 Cognitive performance

Not much is understood about how the multitude of postprandial metabolic changes affect simple and complex cognitive functions as well as different mood states. Glucose is the predominant brain fuel and previous studies such as that of Benton and Parker (1998) have demonstrated the positive effect of an increased blood glucose on certain aspects of cognitive functioning. Benton and Parker (1998) indicated that eating breakfast affects tasks that require the retention of new information but did not influence performance in an intelligence test. The above researchers questioned whether an increase in blood glucose associated with breakfast consumption enhances other types of cognitive functioning and whether the nutritional composition of the previous meal influences memory to a greater or lesser extent. In a later study by Fischer *et al.* (2001), the effects of macronutrients on cognitive performance were researched and they found that ingestion of carbohydrates in the morning

resulted in better short-term memory and accuracy of tasks concomitant with low metabolic activation. Protein, on the contrary, led to better attention and efficiency of tasks related to higher metabolic activation. The researchers concluded that good (and stable) cognitive performance was related to a balanced glucose metabolism, suggesting that glycaemic control is imperative and can occur due to ingestion of a low GI diet. This, in turn, suggests a role in cognitive performance.

An assessment of the benefit of a low GI versus a high GI breakfast on cognitive performance within a period of four hours was undertaken by Benton *et al.* (2003). Their findings resulted in a low GI diet rather than a high GI diet improving memory in humans, especially in the late morning (150 and 210 minutes after breakfast). Similarly, rats displayed better learning performance 180 minutes after they were fed with a low rather than a high GI diet. Benton *et al.* (2003) conclude with the statement that although performances appeared to be only remotely related to blood glucose, the data provide evidence that a low GI breakfast allows better cognitive performances later in the morning.

2.4.6 Application in sports nutrition

The aim of preparing body carbohydrate stores, making provision of fuel during prolonged exercise bouts and restoring glycogen stores during recovery after exercise is to maintain carbohydrate availability to the muscle and central nervous system, during prolonged moderate and high-intensity exercise (Burke *et al.*, 1998b).

Burke *et al.* (1998b) suggested that by applying the GI correctly, the carbohydrate availability for exercise could be optimised, especially during prolonged moderate intensity exercise. Prolonged strenuous exercise leads to increases in the oxidation of plasma glucose when muscle glycogen nears depletion. Pre-exercise nutrition should, therefore, optimise muscle and liver glycogen stores in order to provide the energy required during prolonged exercise sessions, without causing any gastrointestinal discomfort (Burke *et al.*, 1998a). Thomas *et al.* (1994) found that pre-exercise low GI diets led to higher glucose and free fatty acid concentrations after 90 minutes of exercise compared to high GI diets. According to Wee *et al.* (1999), a shift in substrate utilization from fat to carbohydrates occurs when a high GI meal is consumed prior to an event.

Burke *et al.* (1998b) states that carbohydrate-rich foods or beverages seem to be the most effective and appropriate source to ingest during prolonged exercise but the effect of the GI of carbohydrates during exercise has not been systematically studied. Burke *et al.* (1998a) proved in a study that when carbohydrate is ingested during exercise, in amounts of 10g / 100ml glucose solution for a total of 24ml / kg body mass (as recommended by sports nutrition guidelines), that the GI of the pre-exercise meal has little effect on metabolism or on subsequent performance during prolonged sessions of endurance exercise. It seems, however, that a high GI meal following exercise increases the rate of muscle glycogen repletion as opposed to a low GI diet (Burke *et al.*, 1993; Kiens *et al.*, 1990). According to Kiens *et al.* (1990), a high carbohydrate diet based on high GI foods produced greater storage of glycogen during 6 hours of recovery than a diet based on low GI carbohydrate-rich foods as the muscle cells are more permeable to glucose after exercise and its sensitivity to insulin is increased. Stannard *et al.* (2000) conducted research on the GI and high-intensity exercise. They tested the effect of the consumption of a high GI, low GI and non-carbohydrate meal 65 minutes before a cycling test to exhaustion. Their study showed that consumption of a high GI meal is not detrimental to exercise at high intensities as no difference was observed in incremental exercise time to fatigue following ingestion of a high GI or low GI food compared to placebo. They speculate that a high GI food induces a higher rate of glycolysis. The low GI food, on the other hand, increases plasma lactate levels even though plasma glucose concentrations were not decreasing, which could be of benefit.

Noakes (2000) asserts that fatigue during prolonged exercise is not only related to carbohydrate metabolism but is governed by other factors. He proposes that performance is regulated by a central (brain) governor so as to prevent bodily damage. This governor regulates the mass of skeletal muscle that may be activated less in hypoglycaemia and more with replete muscle glycogen stores. Burke *et al.* (1998a) conclude from their study that the GI of carbohydrates ingested prior to exercise does not influence exercise performance, provided that carbohydrate is ingested during exercise so that hypoglycaemia is prevented.

Noakes (2000) points out that although carbohydrates with different GIs induce slightly different metabolic responses, there is still no evidence implying that these differences will affect exercise performance except when hypoglycaemia exists.

Further research by Garcin *et al.* (2001) led to the findings that a low GI versus a high GI food ingested three hours prior to exercise showed no influence on perceived exertion during a one-hour exercise bout at high-intensity. Plasma glucose concentration decreased at the middle of exercise with the high GI food but remained above 4.5mmol/L and did not decrease with the low GI food. Therefore, Garcin *et al.* (2001) suggest that ingestion of the latter food may be a good strategy to avoid glycaemic decrease during a one-hour high-intensity exercise.

Gretebeck *et al.* (2002) points out that although the role of the GI is still being debated in sports nutrition, these recommendations have already been incorporated into some sports nutrition guidelines. They add further that these guidelines are difficult to follow as the GI of specific foods used by athletes has not been determined. Therefore, they determined the GIs of some of these foods ingested by athletes and three categories of sports foods were considered, namely, sports drinks, energy bars and meal replacement drinks. Gretebeck *et al.* (2000) state that health professionals can use the current information if they choose to apply the GI in dietary advice. The authors state further that the II of foods needs to be determined as the insulin response has large effects on metabolism.

Conclusively, in all of the above studies it appears that the GI of foods does not exert an effect on exercise performance. However, it is apparent that high GI foods are of benefit in post-exercise so as to replenish glycogen stores (Burke *et al.*, 1993., 2000; Kiens *et al.*, 1990; Noakes, 2000).

2.5 CRITICISM REGARDING THE PRACTICAL APPLICATION AND CLINICAL UTILITY OF THE GLYCAEMIC INDEX

There is much criticism regarding the application and clinical utility of the GI. The ADA (1994) states that choices are limited if foods are to be chosen on a GI basis. Evidence against this statement is mounting as food choices are in fact wider (Wolever, 1997). Wolever (1997) asserts that there is no limitation in using high GI foods as there is an appropriate utility for this category as well. He points out that both low and high GI foods may be used, depending on the situation.

Coulston and Reaven (1997) stated that one of the reasons the GI was not incorporated into the U.S dietary recommendations was the fact that it was too complex for both the professional and the layperson. The relevancy of this statement is questionable as two separate studies on both adult type 1 diabetics (Giacco *et al.*, 2000) and children (Gilbertson *et al.*, 2001), as reviewed in Section 2.4.2, showed a long-term satisfactory compliance in above 70% of cases and favourable response by children, respectively. These findings rule out the assumption that low GI diets might restrict variety and increase fat and sugar intake.

Another study by Vermeulen and Turnbull (2000) was conducted to assess the use of the GI guide as an educational tool when introducing the GI in practice. Both professionals and diabetics (lay population) were involved and the "GI food pyramid" was utilised. The mean change in knowledge in both the professional and lay population after education was highly significant, but the expert population commented that despite the increase of knowledge, they still found it difficult to grasp the full concept and it is consequently deemed important to keep the education as basic as possible, to avoid confusion.

Another reason cited by Coulston, Hollenbeck and Reaven (1984) is that the GI lacks clinical utility and that differences in GIs between foods do not persist when consumed as a mixed meal (Jenkins *et al.*, 2002; Wolever, 1997). They based this statement on their study results. However, Wolever (1997) reanalysed their data and concluded the opposite.

The ADA cited three studies to support their statement that the GI lacks clinical utility: those of Laine *et al.* (1987), Hollenbeck *et al.* (1988) and Nuttall *et al.* (1983). Wolever (1997) argues that the data of the above studies support the GI and stresses that variation in glycaemic responses do exist both intra and inter-individually on a daily basis, as reviewed in Section 2.2.

On the other hand, Brand-Miller *et al.* (1997) points out that beneficial effects of the GI have been reported in 15 studies internationally. She concludes that the type and amount of carbohydrate and the fat content are still of great importance because certain low GI foods contain high amounts of fat. It is also important to bear in mind the non-food and food factors that lead to varying GIs for similar foods. However, she regards the GI as applicable as long as a few simple substitutions and sound education about the GI is made. Health

professionals should strongly consider that patients are entitled to information on all new research findings (Brand-Miller *et al.*, 1997).

Pi-Sunyer (2002) reviewed the relationship of GI and disease. He argues that greater insulin resistance did not develop with a high GI diet as opposed to a low GI diet as found in a study by Kiens and Richter (1996). Pi-Sunyer (2002) asserts further that a study by Thompson *et al.* (1978) showed that a higher carbohydrate diet resulted in greater insulin sensitivity than the low-carbohydrate diet. Furthermore, Pi-Sunyer (2002) points out that to conclude that high GI diets result in diabetes, it must be shown that these diets result in insulin resistance firstly to such an extent that eventually pancreas exhaustion occurs. Salmerón *et al.* (1997a), however, state that hyperinsulinaemia (a manifestation of insulin resistance) is one of the best predictors of type 2 diabetes and that populations at high risk of type 2 diabetes have higher insulin levels. In contrast to the above study, numerous studies regarding high-fat, low carbohydrate (low glycaemic load) diets have shown to increase the risk of diabetes, therefore, the reviewing author remains unconvinced that high carbohydrate diets lead to increased evidence of diabetes.

Pi-Sunyer (2000) also reviewed studies associating CHD with GL and concluded that present data is insufficient to warrant a public health recommendation and that controlled clinical trials are needed to prove causality.

2.6 FUTURE GLYCAEMIC INDEX RESEARCH

Jenkins *et al.* (2002) reviewed the latest research on the GI which studies the relation of insulin resistance and oxidative stress as well as tissue damage and promotion of the inflammatory process which also play a role in CHD. Depression of serum antioxidants, including lycopene and vitamin E occurs with a postprandial rise in glucose. Greater depression of serum antioxidants is related to increased glycaemia levels. Glycaemic control was also improved with vitamin E supplements.

In conclusion, long-term studies are still required to determine the effect of GI on chronic diseases including cancer (Jenkins *et al.*, 2002).

2.7 SUMMARY

The GI is subject to many varying factors making it confusing at times. However, there are many studies supporting its benefits and clinical application. A challenge lies ahead, not only for health professionals, but also for the food industry to educate and develop products to expand the use of the GI. The GI has sparked much debate and research will no doubt continue as the GI concept will most certainly remain as one of the most talked about nutrition revolutions ever.

2.8 DRIED BEANS

2.8.1 Introduction

Dried beans (*Phaseolus Vulgaris*) are extremely nutritious and economical foods. The GI of dried beans is low, but there are many constraints in utilising this product. Processes such as extrusion can overcome these hurdles but result perhaps in a not so desirable GI. In this section, the nutrient value, constraints and end results of extrusion will be reviewed

2.8.2 Composition and nutrient value of dried beans

In Table 2.1 the nutrient composition of dried beans, haricot and soy beans is given and compared to the Dietary Reference Intakes (DRI).

Dried beans are good sources of the macronutrients protein and carbohydrates as well as minerals and vitamins. The dietary fibre, or NSP, is also substantial and both soluble and insoluble components are present. The major storage protein is phaseolin, which contains the essential amino acids lysine, threonine, leucine, isoleucine, phenylalanine, valine and tryptophan (Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994). Although legumes are recognised as being high in protein, the quality of bean protein is often underestimated. This is because the protein-efficiency ratio (PER), which is based on the growth of laboratory animals, was the standard method of evaluating protein quality until recently. Rats have a methionine requirement that is \approx 50% higher than that of humans.

Consequently, because bean proteins are relatively low in sulphur containing amino acids (SAA), the PER of beans is quite low (reviewed by Messina, 1999). However, the WHO and the US FDA have adopted an alternative method for evaluating protein quality called the protein digestibility corrected amino acid score (PDCAAS). This method uses the amino acid score and a correction factor for digestibility to arrive at a value for protein quality (FDA, 1991). Messina (1999) states further that the PDCAASs of most beans are reasonably good, although their overall value is reduced somewhat by their lower digestibility. Ironically, the relatively low SAA of beans may actually provide an advantage in terms of calcium retention. The reported hypercalciuric effect of protein is likely to be at least partially due to the metabolism of SAAs. Thus, bean protein relative to animal and grain proteins may improve calcium retention (Messina, 1999). Nevertheless, the combination of legumes with cereal grains, nuts, seeds or animal protein sources which are rich in methionine and cystine will ensure that the legumes are complemented (Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994). Legumes contain small amounts of a wide variety of potentially harmful substances referred to as anti-nutrients. These are divided into isolectins and enzyme inhibitors as well as tannins, other phenolic compounds, phytic acid, saponins and some vitamin antagonists (Vorster & Venter, 1994). More recent information, however, suggests that the anti-nutrient label may be an oversimplification, especially in the case of oligosaccharides and saponins (Messina, 1999). Trypsin inhibitors from beans can interfere with protein digestion and in some species of animals do cause pancreatic enlargement and enhance chemically induced pancreatic tumors (Messina, 1999). However, boiling dry beans generally inactivates the heat-sensitive factors such as the trypsin and chymotrypsin inhibitors (Messina, 1999; Reyes-Moreno & Paredes-Lopez, 1993). Messina (1999) states that the amount of trypsin inhibitors obtained by eating commonly consumed beans does not exert any adverse effect. Phytates, on the other hand, are thought to contribute to the poor mineral bioavailability of beans. Although this is an important consideration, phytates and the other components mentioned possibly exert health benefits (Messina, 1999). This will be discussed in the following section.

Table 2.1: The nutrient composition of dry and soy beans* compared to Dietary Reference Intakes** Adapted from Vorster & Venter (1994)

Nutrient	100 GRAM COOKED DRY BEANS			DRI**
	HARICOT	KIDNEY	SOY	
Moisture (%)	69.6	70.5	71.0	
Energy (kJ)	396	405	544	
Protein (g)	6.6	7.1	11.0	56
Fat (g)	0.5	0.3	5.7	
Saturated	-	-	0.8	
Monounsaturated	-	-	1.4	
Polyunsaturated	-	-	3.2	
Carbohydrate (g)	16.6	17.1	9.2	130
Dietary fibre (g)	7.4	5.6	1.6	38
NSP# Total (g)	8.3	6.7	-	
Soluble	3.7	3.2	-	
Insoluble	4.6	3.5	-	
Calcium (mg)	65	19	73	1000
Iron (mg)	2.5	1.7	2.7	8
Magnesium (mg)	45	33	-	400
Phosphorus (mg)	120	87	179	700
Potassium (mg)	320	400	540	
Sodium (mg)	15	16	2	
Zinc (mg)	1.0	1.0	1.2	11
Copper (mg)	0.14	0.16	0.74	0.9
Vitamins: Thiamin	0.11	0.14	0.21	1.2
Riboflavin	0.06	0.07	0.09	1.3
Niacin	0.7	0.7	0.6	16
A (ug)	-	-	30	900
E (mg)	-	-	20.41	15
Folate (ug)	-	-	171	400

*South African Food Tables, Gouws & Langenhoven, 1986

NSP: Non-starch polysaccharides*

** DRI: Dietary Reference Intakes for adult men, Institute of Medicine, 2003.

2.8.3 Health benefits of dried beans

Diets that contain mainly legumes may contribute to decreased cholesterol and lipoprotein levels as indicated in controlled studies on humans, rats and rabbits (Vorster & Venter, 1994). This decrease may also be related to the protein fraction and not to a replacement of dietary lipids (Vorster & Venter, 1994). Kritchevsky (1977) suggested one of several mechanisms proposed, such as the ratio of arginine to lysine which may exert an influence on lipid metabolism. Aljawad *et al.* (1991), in contrast, assert that supplementation of casein, soy and whey proteins with sulphur containing amino acids had greater effects on cholesterol variables than supplementation with arginine and lysine. Their findings indicated that HDL cholesterol increased with methionine supplementation and VLDL cholesterol decreased with cystine. Many studies have shown legumes to have hypocholesterolaemic effects and that the soluble fibre fraction may play a major role. Indirect and direct effects may take place and the major proposed mechanism might be that of the absorption and increased excretion of bile acids (Vorster & Venter, 1994). It was found in a study by Oosthuizen *et al.* (2000) that the inclusion of 91.9g of extruded dry beans daily led to significantly decreased PAI-1 levels, but no significant effects on serum lipoproteins and plasma fibrinogen levels and viscosity were found.

The insoluble fibre fraction leads to a laxation effect through increased water binding capacity, stimulation of microbial growth and fibre residue. Soluble components contribute to lowered glycaemic responses. Fibre also displaces energy content of foods and may induce and maintain optimal body weight through a satiating effect, subsequently further reducing energy consumption (Vorster & Venter, 1994).

As mentioned, the anti-nutritive components may play a beneficial role. Trypsin and chymotrypsin inhibitor (Bowman-Birk inhibitor) found in beans, especially soybeans, has been studied as an anti-cancer agent (as reviewed by Messina, 1999). Furthermore, epidemiological studies have shown that populations consuming vegetarian type diets have lower incidence of cancer. The probable mechanism of action is not understood, however, one is that of the complexing of phytates with iron, which may bring about a favourable reduction in the formation of hydroxyl radicals in the colon (Harland & Morris, 1995) and perhaps lower the risk of breast cancer (Messina, 1999). The full impact of phytate on

carbohydrate digestibility and human health remains uncertain and may be interrelated with dietary fibre and not due to phytate *per se* (Harland & Morris, 1995). The oligosaccharides are responsible for gas production which leads to abdominal discomfort. However, the beneficial effects of the former are associated with growth promotion of bifidobacteria, which has been hypothesized to promote the health of the colon, increase longevity and decrease colon cancer risk (Messina, 1999; Vorster & Venter, 1994). Saponins may also exhibit anti-cancer properties. Finally, although saponins were shown to lower cholesterol in some animal species, the hypocholesterolaemic effects in humans are more speculative (Messina, 1999).

2.8.4 The glycaemic index of dried beans and the practical application and incorporation into the diet

Legumes are particularly beneficial to health due to the effect on glycaemic response (as reviewed in Sections 2.3.4 and 2.3.5). The GI of the dried bean (*Phaseolus Vulgaris*) is 28, depending on the process involved (Foster-Powell *et al.*, 2002). Legumes contain large amounts of RS (from 21% to 44%) and 33% to 53% SAG (Lintas & Cappelloni, 1992). The so-called "anti-nutrients" such as enzyme inhibitors, tannins, lectins and phytic acid also affect blood glucose response (Thompson, 1988). The anti-nutrient content of several leguminous and non-leguminous foods was analysed by Thompson (1988) and tested to determine the effect on glycaemic response. The legumes, which contained the highest concentration of phytic acid, lectins and tannins were digested the slowest and produced the lowest blood glucose response.

The mechanisms whereby the above anti-nutrients affect glycaemic response are not fully elucidated (Thompson, 1988; Thorne *et al.*, 1983), but may be partly attributed to phytic acid that binds with protein, which is associated closely with starch; an inhibitory effect on digestive enzymes which are protein in nature; chelation of calcium, which in turn is required for the activity of amylase; direct binding with starch; an effect on starch gelatinization during cooking or processing and lastly, through direct or indirect influence *in vivo* on gastric emptying (Thompson, 1988). Tannins are suggested to be analogous to that of phytic acid and a direct polyphenol starch interaction is suspected. Lectins, on the other hand, may affect the luminal phase of digestion by inhibition of pancreatic and mucosal enzymes. Conclusively, the

reduction in glycaemic responses can be attributed to the soluble whey fraction which is rich in anti-nutrients (Thompson, 1988).

Wolever *et al.* (1987) showed that canning of beans led to an increase in the GI of 17 units. They hypothesized that the high pressure used in the canning process could alter the physical nature of the starch and anti-nutrient content. It was observed that canned beans led to increases in glycaemic responses in some cases, but not in others.

Many agronomic and cultural factors influence the final quality of dried beans, namely, variety, seed source, agronomic conditions, handling and storage of the dry product and processing procedures (Reyes-Moreno & Paredes-Lopez, 1993).

Acceptability characteristics include grain size, shape, colour, appearance, stability under storage conditions, cooking properties, quality of the product obtained and flavour. Adverse storage conditions of beans lead to hardening. This is termed the "hard-to-cook" (HTC) defect (Reyes-Moreno & Paredes-Lopez, 1993). The HTC defect results in longer cooking periods, a significant decrease in the PER, *in vivo* and *in vitro* protein digestibility and availability of essential amino acids, thus rendering a less acceptable product to the consumer (Reyes-Moreno & Paredes-Lopez, 1993). Tannins lead to the depression of food/feed intake, formation of tannin complexes with dietary protein and other food components, inhibition of digestive enzymes, increased excretion of endogenous protein and minerals (Reyes-Moreno & Paredes-Lopez, 1993). Other factors, however, such as heat stable anti-nutrients, specific structure of the protein, interactions with carbohydrate, dietary fibre components and some minerals also contribute to the limited availability of amino acids from legume protein (Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994; Wang & McIntosh, 1996). Various methods of processing, however, lead to the removal of these factors (as reviewed by Messina, 1999; Reyes-Moreno & Paredes-Lopez, 1993; Vorster & Venter, 1994).

Aguilera *et al.*, (1984) also explain that drying the final product through wet processing involves significant energy usage. Waste by-products are produced which contain high amounts of organic matter and yields are reduced by losses in by-product streams.

Another probable concern is the increase in the risk of gallstone disease. Nervi *et al.* (1989) demonstrated that high legume intake had an effect on biliary lipids and cholesterol saturation and may be related to the prevalence of gallstones in the Pima Indians and Chileans. Thijs and Knipschild (1990) conducted a retrospective case control study on lifestyle-risk factors for

gallstone disease. The intake of saponins was evaluated as it was hypothesized that the saponins might affect lipoprotein metabolism. Apart from legume intake, spinach consumption (which contains high amounts of saponins) was also assessed. Findings led to the conclusion that legume and spinach intake was negatively associated with gallstone risk. The authors acknowledge that all foods containing saponins were not included and that components from pods and pulses might have a different effect. Further studies, therefore, need to be designed to clarify this aspect (Thijs & Knipschild, 1990).

2.9 THE EXTRUSION PROCESS

2.9.1 Introduction

Extrusion implies the process of shaping plasticized materials using pressure to force them through dies or holes. Cooking extrusion is a high temperature, short-time (HTST) process for foods, transforming starch and protein ingredients into marketable products while destroying undesirable enzymes, micro-organisms and heat labile substances (Dziezak, 1989; Harper, 1995).

Extrusion dates back to the mid to late 1800s, when processed meats and similar products were produced. Extrusion has several different functions/applications, namely, cooking, forming, conveying and puffing or drying, depending on the extruder design and the process condition. Various products such as pasta, snack-foods, ready-to-eat breakfast cereals, pet foods, fish and animal feed, soup and beverage bases, weaning foods, pre-gelatinised starches, texturised vegetable protein (TVP) and confectionery are manufactured using this process (Dziezak, 1989; Harper, 1995).

Extruders can be categorized into three main types: piston, roller and single or twin screw extruders. Single screw extruders include five types and twin screw extrusions encompass a variety of machines, which will not be reviewed as it is too extensive. However, the single screw extruders process will be highlighted as it was applied to produce the dried bean flour used in this study (Dziezak, 1989; Harper, 1995). Products like pastas as mentioned in the previous paragraph are difficult to mix, resulting in the extruder being equipped with a preconditioner. The latter is a chamber where raw and granular food ingredients are

uniformly moistened and/or heated by contact with water or live steam before entering the extruder. As the material is moved along the screw in extrusion cooking, it is transformed under conditions of heat, pressure and mechanical shear which results in a discharged product with a higher viscosity (Dziezak, 1989; Harper, 1995). Specific product characteristics are produced by controlling various parameters for example screw geometry, length of barrel and die configuration (Dziezak, 1989; Harper, 1995).

Expanded extrudates are cut at the die face and then dried in a hot air oven to 2-12%. Finished products can then be coated with colours, flavours, oil, sugar, vitamins and minerals (Harper, 1995).

2.9.2 Physiocochemical and structural changes as a result of extrusion

Dramatic transformation of protein and carbohydrates takes place during the extrusion process (Aguilera *et al.*, 1984; Guskja & Khan, 1990; Huber, 1991; Lintas *et al.*, 1995; Steel *et al.*, 1995).

Due to the numerous constraints associated with the utilisation of dried beans, Aguilera *et al.* (1984) conducted a study to produce and utilise navy bean fractions as ingredients in the development of a snack product through extrusion cooking. High protein fractions (HPF) containing 51.4% protein and high-starch fractions (HSF) containing about 50% starch were produced resulting in a feasible process. The process of cracking and dehulling the dried beans prior to extrusion cooking also leads to loss of cell wall components, which renders the starch molecules more accessible to digestion by amylase (Aguilera *et al.*, 1984). Steel *et al.* (1995) conducted a study with the objective to use extrusion technology as an alternative way of overcoming undesirable properties of hardened beans such as unacceptable texture. They also tried to improve nutritive value by decreasing cooking time and by preparing mixed flours of beans and rice which could be of higher nutritive value than either bean or rice flour alone.

Extrusion cooking led to the inactivation of the bean lectins and trypsin-chymotrypsin inhibitors (Steel *et al.*, 1995). Digestibility of the extruded bean flour protein was significantly lower than that of the extruded flour blended with rice and dried beans proteins. The PER did not differ among the samples and the net protein utilisation (NPU) did not differ statistically among the bean and the blended bean-rice flours. This was not readily explainable, since

total body weight gain and PER for the blended flours were significantly greater than when the bean flours were the only sources of protein. The comparative data suggest that growth and PER were influenced by dietary factors other than nitrogen retention and utilisation which may be explained by differences in the rice and bean composition and physiological properties (Steel *et al.*, 1995). Extrusion cooking conclusively produced favourable effects on the nutritive value of beans after twelve months of storage. Extruded bean-rice flours exhibited considerably improved protein nutritive value, texture and flavour, including a significantly decreased bitter taste (Steel *et al.*, 1995).

Wang and McIntosh (1996) evaluated the effect of processing methods of boiling and extrusion and the effect of nutritional complementarity of peas (*Pisium sativum*) or chickpeas (*Cicer arietinum*) and wheat on 1) the improvement of the protein quality of these legumes as indicated by the growth of rats and the PER; 2) the capacity to lower plasma cholesterol of rats; and 3) the growth (weight) of organs. Both processes led to denaturation of anti-nutritional factors (ANF) and it was found that extrusion was more effective in reducing trypsin inhibitors than the boiling method. Rats fed extruded legume diets did not show any deleterious effects compared with rats fed boiled legumes since there were no differences in increased body weight gains (growth), plasma cholesterol concentrations (which were lowered) and increased organ weights between these dietary groups. The researchers concluded that the nutritional value of peas and chickpeas can be improved by removal of ANF and by addition of wheat.

As far as carbohydrates are concerned, Bhattacharya and Hanna (as quoted by Huber, 1991) reported that starches with a 50% blend of amylose and amylopectin (such as that of beans) gave the best expansion properties. Amylose will complex with lipids during extrusion cooking and the formation of the latter complexes reduces digestibility and water solubility of cooked starches.

During low-moisture extrusion, the disruption of the starch granule depends on a combination of heat and mechanical shear. Gelatinisation occurs and the crystalline interior of starch granules is exposed through shear induced by the mechanical action of the screw. Some of the large starch molecules are also dextrinised and broken into shorter chains, which exhibit greater solubility in water (Camire *et al.*, 1990; Gujska & Khan, 1990; Harper, 1995).

Extruded product characteristics are largely affected by the extent of starch transformation during extrusion cooking (Harper, 1995).

Lintas and Cappelloni (1992) researched the effect of processing on legume RS and starch digestibility. Extrusion cooking led to a reduction of RS from between 1% to 7%. Lintas *et al.* (1995) in a further study evaluated the effects of extrusion and boiling cooking on dietary fibre fractions. Three varieties of beans and chickpeas were analysed as well as white and mottled beans (*Phaseolus Vulgaris*). During both processes a redistribution from insoluble to a more soluble fibre was observed, more so in extrusion. A partial solubilisation seemed to have resulted with both hemicelluloses and insoluble pectin substances.

Contrary to the above finding, Artz *et al.* (1990, as quoted by Lintas *et al.*, 1995) did not observe any change in soluble fibre content. Lintas *et al.* (1995) found that the insoluble:soluble ratio in raw samples varied from approximately 1:1 in beans to 2:1 in other legumes. The main fibre component was found to be arabinose in beans and lentils. The amount of sugars and uronic acids (pectins) showed some changes after processing. This was difficult to explain by Lintas *et al.* (1995). Huber (1991) observed a 3% increase in soluble dietary fibre in high-fibre formulations. Guillon and Champ (2000) stated that extrusion leads to changes in hydration capacity which results in fibres hydrating more quickly. However, alteration and collapsing of the fibre matrix also takes place which decreases water retention and thus absorption. Porosity and fragility also increases (Guillon & Champ, 2000; Gujska & Khan, 1990). Combination of thermal and mechanical energy can dramatically change the structure of dietary fibre at all structural levels leading possibly to new functional properties (Camire *et al.*, 1990; Guillon & Champ, 2000). Effect on the solubility of fibre depends on the source of fibre and energy input. The soluble material may have high molecular weight and may in certain conditions form a gel (Guillon & Champ, 2000).

2.9.3 Advantages of extrusion

Processing methods such as extrusion may have positive effects on nutrient availability (reviewed by Frølich, 1995). Mineral utilisation may potentially be affected due to the breakdown of phytic acid which acts as a chelator of iron, zinc and calcium amongst other

factors. The other factors are due to the reorganisation of the dietary fibre components. Extrusion-cooking is applicable in development of products where either nutrition is of little concern (such as the production of confectioneries), destruction of ANF and/or toxic factors is necessary (precooked soy flours) and in the preparation of nutritionally enriched or balanced foods (dietetic foods, for example, gluten-free flour) as well as when increased starch digestibility and avoidance of nutrient destruction is paramount (as reviewed by Camire *et al.*, 1990; Harper, 1995).

Finally, Reyes-Moreno and Paredes-Lopez (1993) discussed the usage of extruded bean flour with improved nutritional and technological characteristics in a variety of bean-based products or in a blend with other foods to improve functional properties and nutritional quality. Furthermore, promising prototypes of extrusion-cooked, high-density formed beans that will readily hydrate and soften when reconstituted are being developed.

2.9.4 Future potential

Many operational characteristics of extrusion cooking lead to the manufacture of numerous products (Harper, 1995; Huber, 1991). The characteristics are as follows (Harper, 1995):

- lower costs - less labour and floor space required
- variety of product characteristics - inducing shape, colour, texture
- energy efficiency - less energy required, less waste, greater yields
- waste minimization - few solids are lost, less effluent discharges
- product innovation - enhanced product development
- continuous processing - replaces many batch heating systems
- process control - thermal/mechanical environment can be adjusted to control changes in food ingredients.

Dziezak (1989) reviewed the application of extrudates in a non-traditional manner, functioning as a chemical reactor, sterilizer and bioreactor. Extrusion processing of pet foods and special foods for aquaculture is also expanding (Harper, 1995).

2.10 THE GLYCAEMIC INDEX OF WHOLEGRAIN KERNEL PRODUCTS VERSUS THAT OF MILLED FLOUR PRODUCTS

The GI of bread made from whole meal flour in South Africa is 75, obviously categorizing it as a high GI item (Foster-Powell *et al.*, 2002). As reviewed in Section 2.3.8, products which contain milled flour lead to increased glycaemic and insulin responses (Behall *et al.*, 1999; Brand Miller, 1994; Liljeberg *et al.*, 1992). Although this is the case it is of interest that phytates are present in large amounts in whole wheat flour, but are destroyed during the leavening process by the action of yeast (Cheryan, 1980). Phytates are also heat stable, but once subjected to cooking methods using water, can be leached out of foods (Cheryan, 1980). However, Militzer (1946, as quoted by Thorne *et al.*, 1983) states that the amylase inhibitor in wheat can withstand cooking in bread, therefore, suggesting that the method of baking is of benefit compared to that of the former. This finding is in contrast to that of Cheryan concerning the addition of yeast.

Currently, there is no GI known for whole wheat muffins. A bran muffin with a GI of 60 (Foster-Powell *et al.*, 2002) is probably closest. Therefore, the GI of whole wheat muffins was also determined in this study.

CHAPTER 3

METHODOLOGY

3.1 INTRODUCTION

In this chapter the methods used in food sampling, food preparation, testing, biochemical and statistical analyses are described. In this study the GI of muffins baked with extruded dried bean flour versus that of muffins baked with whole wheat flour was determined. The prerequisite to entry into this study was a normal blood glucose tolerance. Subjects resided in a metabolic ward overnight, where a standardised meal was given. Three test meals were given according to two Latin square designs for ten males and ten females, respectively. Capillary blood samples were taken the following morning. The AUCs were analysed by a computerised programme. Statistical analyses were performed to test for significant differences and correlations. The limitations of the study will also be discussed.

3.2. METHODS

3.2.1 Subjects

Approval for the study was given by the Ethics Committee of the PU for CHE (project number HHK4M5). Twenty healthy students were recruited and subsequently signed a consent form after explanation of the study and objectives (Appendix A). Nineteen subjects were non-smoking and one smoked once per week. A qualified nurse was responsible for recruitment and screening of all subjects. Exclusion criteria were: diabetes mellitus, abnormal glucose tolerance (i.e. if fasting plasma glucose is 7.8 mmol/L or higher or a random plasma glucose concentration is 11.1 mmol/L or higher and/or ≥ 11.1 mmol/L after 2 hours or more on oral glucose tolerance testing (WHO, 1985), pregnancy, renal problems and BMI greater than 25kg/m². Finally, subjects had to be between the ages of 21 and 26 years. The study cohort consisted of ten males and ten females. Each subject acted as his or her own control.

3.2.2 Study design

A randomised intervention study was conducted. Volunteers randomly consumed three different test meals according to a Latin square design: Table 3.1: male subjects and Table 3.2: female subjects. Glucose was used as a standard test food and consumed twice by all subjects. Subjects stayed overnight in the Metabolic Unit of the PU for CHE and received the same standardised meal in the evening (10-12 hours prior to the test) in order to control the effect that the previous meal might have on the glycaemic response (as reviewed in Section 2.4.1). Subjects were requested to remain inactive and to obtain adequate rest prior to the test. Each subject was required to remain in bed the morning of the test. A fasting capillary blood sample was taken via a lancet, ingestion of the test foods took place and then further capillary blood samples were taken at 15, 30, 45, 60, 90 and 120 minutes after ingestion of test foods. Subjects were required to consume the test meals within 10 minutes and with 200ml of room temperature water.

Table 3.1. Latin square design for male subjects

Subject number	11	12	13	14	15	16	17	18	19	20
Day1	G	WM	BM	G	P	G	WM	BM	G	P
2	WM	BM	G	P	G	WM	BM	G	P	G
3	BM	G	P	G	WM	BM	G	P	G	WM
4	G	P	G	WM	BM	G	P	G	WM	BM
5	P	G	WM	BM	G	P	G	WM	BM	G

G=Glucose, BM=Bean muffin, WM=Whole wheat muffin, P=Pasta. The pasta test food indicated here was part of another study.

Table 3.2 Latin square design for female subjects

Subject number	1	2	3	4	5	6	7	8	9	10
Day 1	G	WM	BM	G	BM	G	WM	BM	G	BM
2	WM	BM	G	BM	G	WM	BM	G	BM	G
3	BM	G	BM	G	WM	BM	G	BM	G	WM
4	G	BM	G	WM	BM	G	BM	G	WM	BMG
5	BM	G	WM	BM	G	BM	G	WM	BM	G

G =Glucose, BM=Bean muffin, WM=Whole wheat muffin

3.2.3 Pre-test meals

The standardised meal was calculated according to the guidelines for the prudent diet for both male and female subjects respectively, i.e. 55-60% carbohydrates, 30% or less fat and 10-15% protein to provide approximately one third of the total recommended energy intake. The Food Fundi for Windows (PentaMedical Systems (Pty) Ltd.) (Appendix B) was used to analyse the meal. The crusts of bread were removed in order to diminish the effect of RS on glycaemic response (reviewed in Section 2.3.2).

3.2.4 Test foods

Each test food provided exactly 50g available carbohydrate which was compared to 50g carbohydrate from glucose. The glucose solution was prepared according to the method as shown in Appendix C. The muffins were baked according to the recipes as supplied by the Dried Bean Producers Organisation (Appendix C). Muffins were then frozen and taken out of the freezer the evening prior to the test to thaw. The Englyst method (Englyst *et al.*, 1999) was used to analyse the different carbohydrate fractions (as reviewed in Section 2.3.2) (Appendix D) by Dr. Klaus Englyst from Englyst Carbohydrates Research and Services Ltd, Southampton. The nutritional content of each test food was analysed by Senwesko Feeds (Table 3.3).

Table 3.3 Macronutrient analysis of bean and whole wheat muffin

Test food	Bean muffin	Whole wheat muffin
Serving (g)	105	101
Protein (g)	11.12	7.92
Carbohydrate (g)	43.16	47.02
Fat (g)	10.9	11.55
Ash (g)	3.71	2.14
Fibre (g)	2.70	1.9
Moisture (g)	28.41	29.17

3.2.5 Biochemical analyses

Capillary blood samples were taken and glucose was determined by means of glucose strips (Surestep of Lifescan) and a calibrated glucometer (Surestep of Lifescan, Johnson and Johnson, Sandton, Milpitas CA95035, California, USA). Glucometers were calibrated prior to each test day and were used for the same subject during the entire research period.

3.2.6 Glycaemic index

The areas under the glucose curve using the fasting values as baseline (Wolever *et al.*, 1991) were calculated by the Statistical Consultation Service, PU for CHE, for each test with a computerised programme. The glycaemic indices were then calculated according to the following formula (Jenkins *et al.*, 1981):

$$GI = \left[\frac{\text{incremental area under 2 hours glucose curve for meal}}{\text{incremental area under 2 hours plasma glucose curve for 50g glucose}} \right] \times 100$$

3.2.7 Statistical analyses

All statistics were compiled and analysed by the Statistical Consultation Service at the PU for CHE. The SAS System for Windows Release 8.02 TS level (1999-2001) computer

programme was used to analyse the data. A two-way analysis of variance (ANOVA) with repeated measures over time was used to calculate the differences between time points within treatments for each test meal. The Tukey test was used to establish significant differences between group means.

3.2.8 Limitations of the study

3.2.8.1 Acceptability of meals

Two of the subjects did not like the raisins in the muffins but still consumed the entire meal. Three of the subjects experienced nausea when ingesting the glucose solution but still consumed the solution over the set time period. It was shown by Thompson *et al.* (1982) that the high osmotic activity of glucose solutions affect gastric emptying and, therefore the glycaemic response.

3.2.8.2 Other limitations

Due to the study design and time frame, it was not possible to give glucose three times as a reference food as recommended (Wolever, 1990) in order to reduce intra and inter-individual variation. One subject ingested some medication due to slight illness. Three subjects were accidentally not given 200ml water to consume with their test food (on one occasion). Only once during the study three subjects did not obtain adequate rest the evening prior to the intervention. The subjects who were students were also experiencing stress related to preparation for exams.

3.2.8.3 Conclusion

This study was conducted in accordance with the most recent interlaboratory guidelines based on the results of international studies (Wolever *et al.*, 2003). The analyses of test foods includes that of Englyst *et al.* (1999) which are of importance in GI studies to exclude the RS fraction (AOAC method). This is deemed important as the latter fraction exerts a

possible effect on the GI. The macronutrient analysis is also paramount to determine the effect of the protein and fat content on GIs. It would, however, have been of interest to determine the soluble and insoluble fibre fractions of the dried beans before and after extrusion. Furthermore, the raisins should be excluded from the recipe as they were unevenly distributed. Sensory evaluation was not done. Apart from the limitations of this study, an important one being that of each subject not repeating the reference food three times, the positive aspects were that the glycaemic carbohydrate of the test foods was determined by the Englyst method, as recommended by FAO/WHO (1998), and that all subjects remained in the metabolic ward which led to stricter control. The results will be presented in the next chapter.

CHAPTER 4

RESULTS

4.1 INTRODUCTION

Data is presented of all the subjects as compliance was good. Some of the subjects had high AUCs for oral glucose. Although it is not the aim of this study to describe intra and inter-individual variation in blood glucose responses, it will be shown. In addition to the above, the mean glucose curves for all test meals at set time intervals will be explained together with resultant AUCs and calculated GIs.

4.2 SUBJECT CHARACTERISTICS

The ten male subjects had a mean age of 20.8 years and mean BMI of 24.6kg/m² and all were non-smoking. The ten female subjects had a mean age of 20.5 years and mean BMI of 22kg/m² and all were non-smoking except for one subject whom smoked once per week. The mean age for the total group was 20.6 years and mean BMI was 23.3kg/m².

4.3 INTRA AND INTER-INDIVIDUAL VARIATION IN THE GLUCOSE RESPONSES TO TWO GLUCOSE REFERENCE TESTS

It is well known that glycaemic responses vary substantially within as well as between subjects. Subjects 3, 6, 7, 9, 17 and 19 showed the largest variation in AUC between the two oral glucose reference tests. Their results are depicted graphically in Figure 4.1. The fasting blood glucose concentration was used as baseline to measure the AUCs for two glucose tests for each subject.

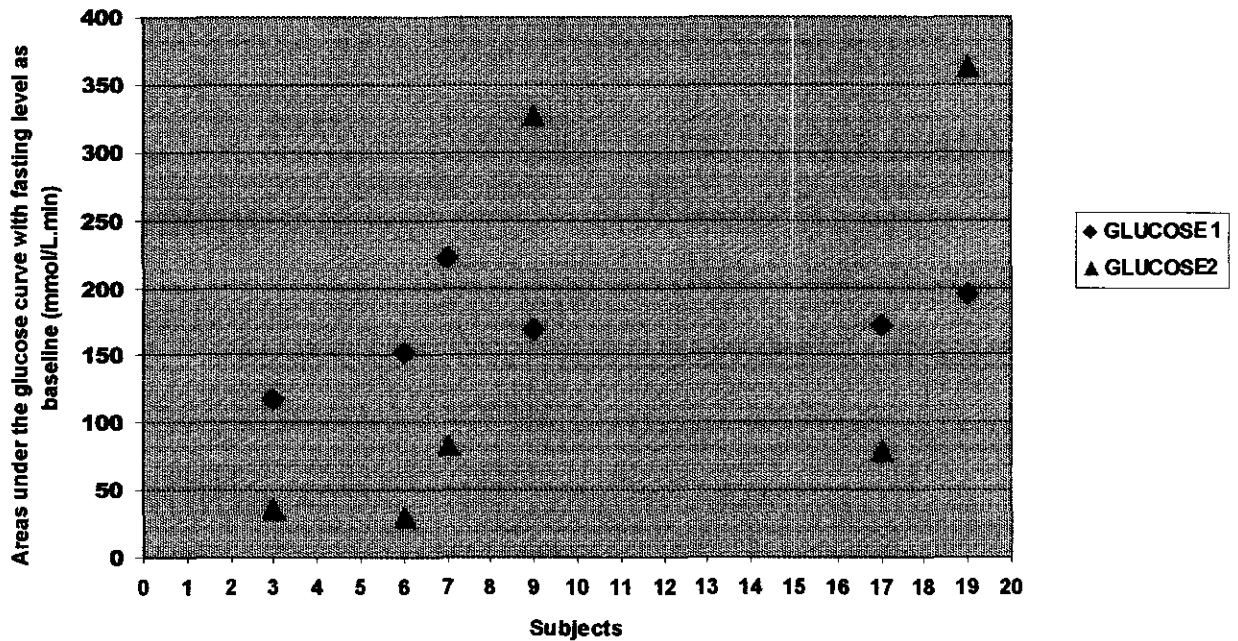


Figure 4.1 Differences within and between some subjects in the areas under the glucose curve with the fasting level as baseline, in response to two glucose reference meals. It is clear from Figure 4.1 that ingestion of oral glucose on different occasions resulted in large areas under the curve for six subjects, indicating that there were large differences intra-individually and inter-individually.

4.4 MEAN GLUCOSE CURVES FOR DIFFERENT TEST FOODS

Figure 4.2 shows that the glucose curve after oral glucose peaked at 30 minutes, whereas the curves for bean and whole wheat muffins peaked at 45 minutes. The curve for the bean muffin (mean of two tests for the female subjects) showed the least fluctuations and glucose the most fluctuations, with a maximum value of 7.9 mmol/L. The value of glucose at 120 minutes was 4.2 mmol/L which was lower than that of baseline. The values for the muffins were slightly higher at 120 minutes indicating a smaller counter-regulatory response. The glucose curve for the muffins seems to be flatter compared to oral glucose, indicating slower glucose absorption.

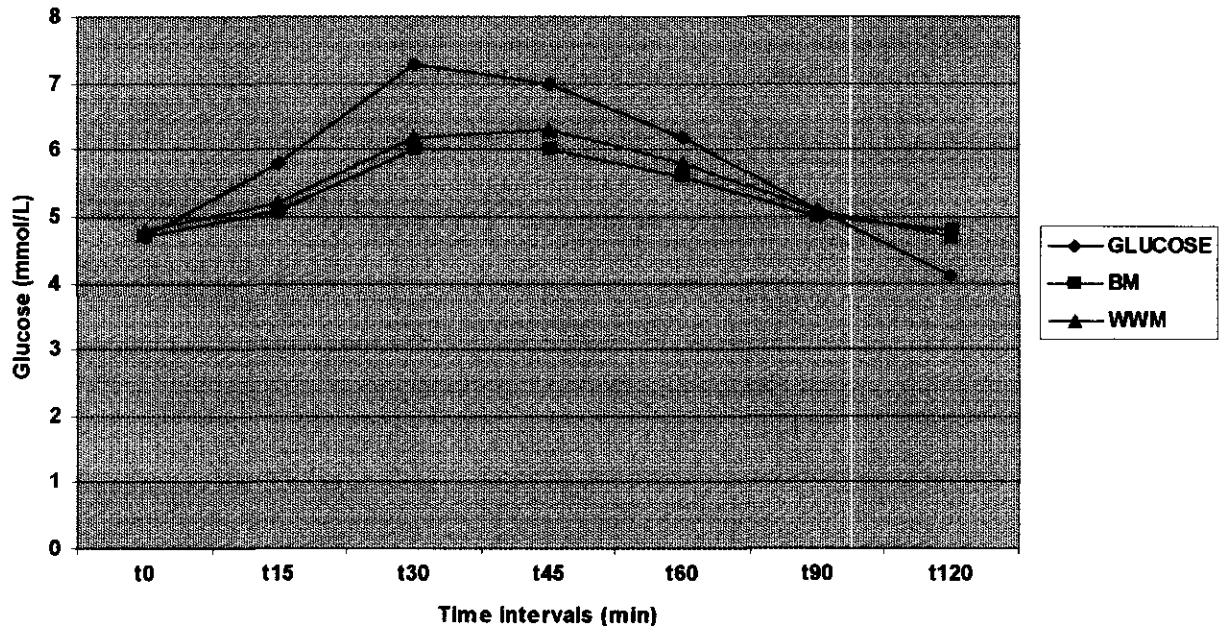


Figure 4.2 Mean glucose levels for different test foods (n=20).

G=glucose, WWM=whole wheat muffin, BM=bean muffin. Glucose concentrations after oral glucose differed significantly from the concentrations after the muffins at each time interval, except at t90 ($p < 0.05$)

4.5 CALCULATION OF GLYCAEMIC INDICES

Table 4.1 displays the individual AUCs for each subject, used to calculate the mean GIs of the bean muffin and whole wheat muffin respectively. The GIs of the products are shown in Table 4.2

The mean AUC for bean muffins was 80.3, for whole wheat muffins 86.4 and for oral glucose 165.6 mmol/L.min (significantly different from the AUC for the muffins, $p < 0.05$). Subjects 5, 7, 11, 14, 17 and 20 showed much higher glycaemic responses than the other subjects for the bean muffin and subjects 7, 11, 15 and 18 showed much higher glycaemic responses for the whole wheat muffin. The mean glycaemic responses for glucose were higher in subjects 4, 9, 16, 18 and 19. The GI values of the male and female subjects did not differ significantly. Therefore, the mean GI for the whole subject group is reported.

Table 4.1 Areas under the curves (AUCs) in mmol/L.min for test foods and reference

Subjects	AUC BM	AUC WWM	AUC GLUCOSE
1	58.4	32.3	65.1
2	55.4	52.1	155.9
3	25.3	19.0	77.12
4	60.8	74.3	233.4
5	121.5	53.0	142.5
6	46.9	75.0	91.3
7	140.0	197.9	152.5
8	84.4	80.3	183.9
9	60.0	54.8	248.3
10	70.9	105.4	130.0
11	104.3	147.0	154.6
12	120	95.3	134.9
13	66.3	67.6	155.3
14	103.5	99.8	181.7
15	46.5	130.5	193.2
16	70.78	38.5	212.3
17	107.3	106.5	125.2
18	86.3	151.2	209.4
19	71.7	65.4	278.7
20	105	81.8	187.2
Mean (SD)	80.3 (29.1)^a	86.4 (43.3)^b	165.6 (54.1)^{a,b}

AUC = area under the curve, BM = bean muffins, WWM = whole wheat muffins, SD = standard deviation. a,b :Means with the same superscript differed significantly ($p < 0.05$).

Table 4.3 displays the mean GIs for both the bean muffin and the whole wheat muffin. The GI can be categorised as follows (using glucose as reference) (Brand Miller *et al.* 1996): below 55% as low GI, between 55 and 70% as intermediate and more than 70% as high GI. The mean GI's for the bean muffin and whole wheat muffin was 53% and 55.5%, respectively.

Both the extruded dry bean and whole wheat muffins fell on the top border of the low GI category. The means, standard deviations (SDs) and 95% confidence intervals (CIs) for the muffins are shown in Table 4.3.

The variability of each subject's glycaemic responses can be expressed as the coefficient of variation (CV) which is the standard deviation expressed as a percentage of the mean (Wolever, 1990). The coefficient of variation (CV) in GIs were 45.5% for the bean muffin and 52.8% for the whole wheat muffin.

Because of this variability it may be more useful to assign a range within which the GI of a food product may vary rather than to assign a single number (a mean) to a product. The 95% CI in Table 4.3 may be used for this range. Looking at the 95% CI in the table, it can be said with 95% confidence that the true mean GI for extruded dry bean muffins lies between 42% and 64% (low to intermediate category) and for whole wheat muffins between 42% and 69% (low to intermediate category).

There were no significant differences ($p > 0.05$) between the GI of the bean muffin and whole wheat muffin but significant differences ($p < 0.05$) between the AUC after glucose and the AUC after the bean muffin as well as that of the whole wheat muffin.

Table 4.2 Glycaemic indices (%) of bean muffins and whole wheat muffins for each subject (n=20)

N	BM	WWM
1	89.6	49.5
2	35.5	33.4
3	32.7	24.6
4	26.1	32.0
5	85.2	37.2
6	51.3	82.1
7	92.0	130.0
8	46.0	43.7
9	24.1	22.0
10	54.5	81.0
11	67.4	95.1
12	89.0	71.0
13	43.0	44.0
14	57.0	55.0
15	24.1	68.0
16	33.4	18.2
17	86.0	85.0
18	41.2	72.2
19	28.7	23.5
20	56.1	43.7
MEAN	53.0	55.5

N=subjects; BM=Bean muffin; WWM=Whole wheat muffin.

Table 4.3 Mean glycaemic indices of the test foods.

Intervention	n	Mean \pm SD	Lower 95% CI	Upper 95% CI
BM	20	53.0 \pm 24.1	41.7	64.2
WWM	20	55.5 \pm 29.3	41.8	69.2

BM=Bean muffin; WWM=Whole wheat muffin; SD=Standard deviation, CI =Confidence interval

4.6 CONCLUSION

As expected, intra and inter-individual variation in the glucose response to the two glucose reference tests as well as the GI of both muffins occurred in some subjects in spite of adhering to protocol. The intake of both muffins resulted in significantly lower glucose response as illustrated by the glucose response curves and AUCs (Fig. 4.2 and Table 4.1 respectively). The mean GIs for the bean and whole wheat muffins were 53% (CI 41.7;64.2) and 55.5% (CI: 41.8;69.2) respectively, showing no significant differences. The RS content of the bean muffin was 1.6g/100g and that of the whole wheat muffin 0.8g/100g.

CHAPTER 5

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 INTRODUCTION

The objective of this study was to determine the GIs of muffins baked with extruded bean flour and whole wheat flour respectively. The main findings of this study were that extruded dry bean muffins and whole wheat muffins had comparable GIs that fell within the low to intermediate categories for GI.

5.2 DISCUSSION

The GI of the bean muffins reported in this study was higher than expected, probably due to the extreme process of extrusion that was applied. Extrusion cooking, firstly, affects particle size (Carnovale & Lintas, 1995), whereby disruption of cell wall components takes place (Guillon & Champ, 2000). This effect renders a higher glycaemic response as seen in this study, consistent with numerous studies by Haber *et al.* (1977) with apples, Järvi *et al.* (1995) with ground red kidney beans, Jenkins *et al.* (1998b) with milled flour breads made from whole grains, Liljeberg *et al.* (1992) with breads baked with whole meal barley flour and white wheat bread, Behall *et al.* (1999) and Holt and Brand Miller (1994) with breads baked with varying grades of wheat which all exhibited higher glycaemic responses due to the reduced particle size.

Secondly, extrusion leads to gelatinisation of the starch (Camire *et al.*, 1990; Carnovale & Lintas, 1995; Harper, 1995), which, in turn, elicits high glycaemic and insulin responses (Collings *et al.*, 1981; Granfeldt *et al.*, 1994; Granfeldt *et al.*, 2000; Ross *et al.*, 1987) being consistent with the results of this study.

Thirdly, the lesser digestibility of legume starch is in the soluble whey and most likely includes anti-nutrients such as phytic acid, tannins and lectins, which are present in the highest concentrations in this fraction (Thompson, 1988). Extrusion cooking has dramatic results on the above factors as the ANF is destroyed and trypsin inhibitors in particular are reduced to

negligible levels (Steel *et al.*, 1995; Wang & McIntosh, 1996). This has been reflected in the results of this study.

Finally, soluble fibre fractions increase by 3% for high fibre foods during extrusion (Huber, 1991), which would possibly increase viscosity and thus lead to a consistent glycaemic response (Léclere *et al.*, 1994; Liljeberg *et al.*, 1996b; Van Der Sluijs *et al.*, 1999). However, this was not evident in these results. It is not known what changes took place as neither soluble nor insoluble fibre fractions were measured, but the RAG fractions which were 26.6/100g and 30.1g/100g in the bean muffin and whole wheat muffin respectively, confirmed the hypothesis of Englyst *et al.* (1999) that this former fraction is an important food determinant of the glycaemic response. It also supports the findings of Lintas and Cappelloni (1992) where the rapidly digestible starch increased up to 89%. The SAG fractions for the bean muffin and the whole wheat muffin in this study were 1.8/100g and 1.5/100g, respectively, which is minimal. The RS fraction was also present in minimal quantities (1.6g/100g for the bean muffin and 0.8g/100g for the whole wheat muffin). Consensus in the international literature has not been reached whether RS should be counted as part of the 50g available carbohydrate portion. Recently, Wolever (2003) recommended that RS should be excluded from the portion size. In practice, however, this is difficult because the analytical method for the determination of RS is not widely available and the RS content of most foods is, therefore, unknown. According to Elmstahl (2002), intact dry beans have a high RS content (11.1% for white beans) and the inclusion thereof, as has been the case in the past, may have a large impact on the GI value, underestimating the GI of dried beans substantially.

It is not clear which factors dominated in having had the most dramatic effect in increasing the glycaemic response. In spite of the altered physicochemical and structural changes brought about by the extrusion process, the GI of the bean muffin (53) is still categorized as a low GI food, whereas the whole wheat muffin (GI of 55) falls into the lower end of the intermediate category.

A puzzling aspect, though, is that one would expect that the whole wheat muffin would have a much higher GI due to the high GI of bread made from whole meal flour (Foster-Powell *et al.*, 2002). Both the bean and whole wheat muffins consisted of exactly the same ingredients, apart from the two different flours. The addition of protein and fat would also not elicit a lower

glycaemic response as the latter ingredients constituted only 11.12g and 10.9g protein as well as 7.92g and 11.55g fat for the bean muffin and whole wheat muffins, respectively. According to the studies of Nuttall *et al.* (1994) and Wolever *et al.*(1994), 30-50g of protein and 22g of fat are the amounts that lead to reduction in glucose response after 50g carbohydrate load. A further explanation could possibly be the fact that whole wheat flour consists of high amounts of phytates (Cheryan,1980), which in turn binds calcium and reduces the activity of amylase (which is a calcium dependent enzyme) (Thompson, 1998). Alternatively, phytates could bind to the starch or protein portion of the starch-protein complex (Thompson,1998). According to Militzer, (as quoted by Thorne *et al.*, 1983), the amylase inhibitor in wheat can withstand cooking in bread. Therefore, possibly suggesting that the method of cooking may affect anti-nutrient activity and thus starch digestibility (Thorne *et al.*, 1983).

Finally, extrusion cooking definitely holds benefits due to the increased PER and thus digestibility of protein and increased mineral absorption. The question arises, however, whether muffins baked from extruded bean flour could hold any benefit when there was no significant difference between that of muffins made from whole wheat flour. Furthermore, are muffins a practical item for breakfast (apart from freezing the product) in terms of preparation possibly being time consuming? This product and possible utilization thereof will be discussed further in the recommendation section.

5.3 CONCLUSION

Although the GI of the bean muffin was higher than expected, it still fell in the low GI category. Considering the health benefits of dried beans (as discussed in Section 2.8.3), these muffins (and other products made from extruded dried beans) can be recommended by dietitians to all their patients/clients to improve their health status.

5.4 RECOMMENDATIONS

5.4.1 Currently, a greater proportion of the diabetic population probably consumes maize at breakfast, which has a high-GI (71-74) (Foster-Powell *et al.*, 2002) except when cooled after cooking (Venter *et al.*, 1990). Extruded bean flour could possibly be utilised as a

cereal (porridge) for both children and adults in the diabetic and non-diabetic population so as to incorporate more variety in the diet.

5.4.2 Extruded bean flour could also be utilised in baked products such as breads, muffins, pizza bases, biscuits and rusks (which are a traditional breakfast item in the Afrikaner diet),

5.4.3 Extruded bean flour may be used for patients with coeliac disease or wheat allergy. The prevalence of coeliac disease among type 1 diabetics in the United Kingdom varies between 1 in 20 and 1 in 50 (Packer *et al.*, 2000). The incidence of these disease relationships in South Africa is, however, not known. It is difficult to achieve the goal of adhering to a low GI, gluten-free diet without compromising glycaemic control as such foods are limited. It is for the above reason that the extruded bean flour can be used as a suitable gluten-free alternative.

5.4.4 Although the possible uses of dried beans as such have not been investigated in this study, pre-cooked beans could be drenched in a mixture of sodium propionate, sucrose and flavourings and then dried to produce a "bean breakfast cereal" for those who prefer a non-cooked cereal.

5.4.5 The recommendation for a serving size and calculated GL for the muffins are summarised in Table 5.1 and are based on the attached recipes (Appendix C).

Table 5.1 The glycaemic load of a bean muffin and a whole wheat muffin

Recommendation	Serving size	Available carbohydrate	GL (per serving)
Bean muffin	60g	28.4g	15
Whole wheat muffin	64g	31.6	18

5.4.6 Finally, it is highly recommended that the food industry formulate extruded bean flour products due to the advantages thereof. Björck *et al.* (2000) recently stated that "to fully exploit the metabolic potential of a low GI, a wider range of low GI foods are necessary." The shortage of low GI alternatives is particularly pronounced among bread and breakfast cereals. The technological means exist to provide such foods and the development of low-GI foods is a challenge for the food industry. Furthermore, the GIs should be tested and it is of great

importance to bear in mind that the final GIs of these products will be dependent on the non-food and food factors, as previously reviewed. If it is found that any of the products elicit an intermediate or high GI, then additions of either sodium propionate, β -glucan or AX can be made to lower the GI. It is also necessary that the sensory acceptability of these products should be determined.

REFERENCES

ADA see American Diabetes Association

AGUILERA, J.M., CRISAFULLI, E.W., LUSAS, M.A., UEBERSAX, M.A. & ZABIK, M.E. 1984. Air classification and extrusion of navy bean fractions. Journal of food science, 49:543-546.

AGUS, M.S.D., SWAIN, J.F., LARSON, C.L., ECKERT, E.A. & LUDWIG, D.S. 2002. Dietary composition and the physiologic adaptations to energy restriction. American journal of clinical nutrition, 71:901-907.

ALJAWAD, N.S., FRYER, E.B. & FRYER, H.C. 1991. Effects of casein, soy and whey protein and amino acid supplementation on cholesterol metabolism in rats. Journal of nutrition and biochemistry, 2:15-24.

AMERICAN DIABETES ASSOCIATION (ADA): 1994. Nutrition recommendations and principles for people with diabetes mellitus (Position Statement). Diabetes care, 17:519-522.

ANDERSON, G.H. 1997. Sugar and health: A review. Nutrition research, 17(9):1485-1498.

ANON. 2002. Upcoming DRI report: New ways of defining fibre. Journal of the American Dietetic Association, 102(4):468.

ARMAND, M., HAMOSH, M. & DiPALMA, J.S. 1995. Dietary fat modulates lipase activity in healthy humans. American journal of clinical nutrition, 62:74-80.

BEHALL, K.M., DANIEL, J.S., YUHANIYAK, I. & CANARY, J. 1989. Diets containing high amylose vs. amylopectin starch: effects on the metabolic variables in human subjects. American journal of clinical nutrition, 49:337-440.

BEHALL, K.M. & HOWE, J.C. 1995. Effects of long-term consumption of amylose vs. amylopectin on metabolic variables in human subjects. American journal of clinical nutrition, 61:334-440.

BEHALL, K.M., SCHOLFIELD, D.J. & HALLFRISCH, J. 1999. The effect of particle size of whole-grain flour on plasma glucose, insulin, glucagon and thyroid stimulating hormone in humans. Journal of the American College of Nutrition, 18(6):591-597.

BENTON, D. & PARKER, P.Y. 1998. Breakfast, blood glucose and cognition. American journal of clinical nutrition, 67(suppl):772S-778S.

BENTON, D., RUFFIN, M., LASSEL, T., NABB, S., MESSAOUDI, M., NINOY, S., DESOR, D. & LANG, V. 2003. The delivery rate of dietary carbohydrates affects cognitive performance in both rats and humans. Psychopharmacology, 166(1):86-91.

BESSESSEN, D.H. 2001. The role of carbohydrates in insulin resistance. Journal of nutrition, 131:2782S-2786S.

- BJORCK, I., LILJEBERG, H. & OSTMAN, E. 2002. Low glycaemic foods. British journal of nutrition, 83: S149-S155.
- BOLTON, R.P., HEATON, K.W. & BURROUGHS, L.F. 1981. The role of dietary fibre in satiety, glucose, and insulin studies with fruit and fruit juice. American journal of clinical nutrition, 34:211-217.
- BORNET, F.R.J., COSTAGLIOLA, D., SALWA, W.R., BLAYO, A., FONTVIEILLE, A., HAARDT, M., LETANOUX, M., TCHOBROUTSKY, G., & SLAMA, G. 1987. Insulinaemic and glycaemic indexes of six starch-rich foods taken alone and in a mixed meal by type 2 diabetics. American journal of clinical nutrition, 45:588-595.
- BOTES, I. 2000. Intra-and inter-individual variations in the glucose response to different standards and test foods. Potchefstroom: PU for CHE. (Mini - dissertation-M.Sc.) 190p
- BRAND, J., NICHOLSON, P.L., THORBURN, A.W. & TRUSWELL, A.S. 1985. Food processing and the glycaemic index. American journal of clinical nutrition, 42:192-196.
- BRAND MILLER, J. & LOBBEZOO, I. 1994. Replacing starch with sucrose in a high glycaemic index breakfast cereal lowers glycaemic and insulin responses. European journal of clinical nutrition, 48:749-752.
- BRAND-MILLER, J., FOSTER-POWELL, K. & COLAGIURI, S. 1996. The G.I. factor. Sydney, Australia: Hodder and Stoughton. 250p.
- BRAND-MILLER, J., COLAGIURI, S. & FOSTER – POWELL, K. 1997. The glycaemic index is easy and works in practice. Diabetes care, 20(10):1628-1629.
- BRAND-MILLER, J.C., HOLT, S.H.A., PAWLAK, D.B. & McMILLAN, J. 2002. Glycaemic index and obesity. American journal of clinical nutrition, 76(suppl):281S-285S.
- BRIGHENTI, F., CASTELLANI, G., BENINI, L., LEOPARDI, E., CROVETTI, R. & 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, 49:242-247.
- BURKE, L.M., COLLIER, G.R. & HARGREAVES, M. 1993. Muscle glycogen storage after prolonged exercise: effect of the glycaemic index of carbohydrate feedings. Journal of applied physiology, 75:1019-1023.
- BURKE, L.M., CLAASEN, A., HAWLEY, J.A. & NOAKES, T.D. 1998a. Carbohydrate intake during cycling minimizes effect of glycaemic index of pre-exercise meal. Journal of applied physiology, 85(6):2220-2226.
- BURKE, L.M., COLLIER, G.R. & HARGREAVES, M. 1998b. Glycaemic index – a new tool in sports nutrition. International journal of sports nutrition, 8:401-415.
- BYRNES, S.E., BRAND MILLER, J.C. & DENYER, G.S. 1995. Amylopectin starch promotes the development of insulin resistance in rats. Journal of nutrition, 125:1430-1437.

- CAMIRE, M.E., CAMIRE, A. & KRUMHAR, K. 1990. Chemical and nutritional changes in foods during extrusion. Critical reviews in food science and nutrition, 29:5-37.
- CARNOVALE, E. & LINTAS, C. 1995. Dietary fibre: effect of processing and nutrient interactions. European journal of clinical nutrition, 49(suppl. 3):S307-S311.
- CHERYAN, M. 1980. Phytic acid interactions in food systems. Critical reviews in food science and nutrition, 13(4):297-335.
- CHEW, I., BRAND, J.C., THORBURN, A.W. & TRUSWELL, A.S. 1988. Application of glycaemic index to mixed meals. American journal of clinical nutrition, 47:53-56.
- COLLINGS, P., WILLIAMS, C. & MACDONALD, I. 1981. Effects of cooking starch on serum glucose and the insulin responses to starch. British medical journal, 282:1032.
- COLLIER, G.R., WOLEVER, T.M.S., WONG, G.S. & JOSSE, R.G. 1986. Prediction of glycaemic response to mixed meals in noninsulin-dependent diabetic subjects. American journal of clinical nutrition, 44:349-352.
- COULSTON, A.M., HOLLENBECK, C.B. & REAVEN, G.M. 1984a. Utility of studies measuring glucose and insulin responses to various carbohydrate-containing foods. American journal of clinical nutrition, 39:163-165.
- COULSTON, A.M., HOLLENBECK, C.B. & LIU, G.C. 1984b. Effects of source of dietary carbohydrate on plasma glucose, insulin and gastric inhibitory polypeptide responses to test meals in subjects with noninsulin-dependent diabetes mellitus. American journal of clinical nutrition, 40:965-970.
- COULSTON, A.M. & REAVEN, G.M. 1997. Much ado about (almost) nothing (Editorial). Diabetes care, 20:241-243.
- CUMMINGS, J.H., ROBERFROID, M.B., ANDERSSON, H., BARTH, C., FERRO-LUZZI, A., GHOOS, Y., GIBNEY, M., HERMONSEN, K., JAMES, W.P.T., KORVER, O., LAIRON, D., PASCAL, G. & VORAGEN, A.G.S. 1997. A new look at dietary carbohydrate: chemistry, physiology and health. European journal of clinical nutrition, 51:417-423.
- DZIEZAK, J.D. 1989. Single and twin screw extruders in food processing. Food technology, 43:163-174.
- ELMSTAHL, H.L. 2002. Resistant starch content in a selection of starchy foods on the Swedish market. European journal of clinical nutrition, 56:500-505.
- ENGLYST, H.N., & CUMMINGS, J.H. 1986. Digestion of the carbohydrates of banana (*Musa paradisiaca sapientum*). American journal of clinical nutrition, 44:42-50.
- ENGLYST, H.N., TROWELL, H., SOUTHGATE, D.A.T. & CUMMINGS, J.H. 1987. Dietary fibre and resistant starch. American journal of clinical nutrition, 46:873-874.

ENGLYST, K.N., ENGLYST, H.N., HUDSON, G.H., COLE, T.J. & CUMMINGS, J.H. 1999. Rapidly available glucose in foods: an in vitro measurement that reflects the glycaemic response. American journal of clinical nutrition, 69:448-454.

FAO/WHO - see Food and Agriculture Organization.

FDA – see Food and Drug Administration.

FISCHER, K., COLOMBANI, P.C., LANGHANS, W. & WENK, C. 2001. Cognitive performance and it's relationship with postprandial metabolic changes after ingestion of different macronutrients in the morning. British journal of clinical nutrition, 85:393-405.

FOOD AND AGRICULTURE ORGANIZATION/WORLD HEALTH ORGANIZATION. 1998. Carbohydrates in human nutrition Report of a Joint FAO/WHO Consultation. FAO Food and Nutrition Paper 66. Rome: FAO.

FOOD AND DRUG ADMINISTRATION, DEPARTMENT OF HEALTH AND HUMAN SERVICES. 1991. Food labeling: general requirements for health claims for food. Federal register, 56:60537-60566.

FOSTER-POWELL, K., HOLT, S.H.A. & BRAND-MILLER, J.C. 2002. International table of glycaemic index and glycaemic load values. American journal of clinical nutrition, 76: 50-56.

FRANZ, M.J. 1997. Protein: metabolism and effect on blood glucose levels. Nutrition update, 23(6):643-648.

FRØLICH, W. 1995. Bioavailability of micronutrients in a fibre-rich diet, especially related to minerals. European journal of clinical nutrition, (Suppl. 3):116-122.

FROST, G. & DORNHORST, A. 2000. The relevance of the glycaemic index to our understanding of dietary carbohydrates. Diabetes medicine, 17:336-345.

FROST, G., WILDING, J. & BEECHAM, J. 1994. Dietary advice based on the glycaemic index improves dietary profile and the metabolic control in type 2 diabetic patients. Diabetes medicine, 11(4):397-401.

FROST, G., LEEDS, A.A., DORE, C.J., MADEIROS, S., BRADING, S. & DORNHORST, A. 1999. Glycaemic index as a determinant of serum HDL-cholesterol concentration. The Lancet, 353:1045-1048.

GARCIN, M., BRÉSILLION, S., PITON, A. & PÉRÈS, G. 2000. Does perceived exertion depend on glycaemic index of foods ingested throughout three hours before a one hour high-intensity exercise? Perceptual and motor skills, 93:599-608.

GIACO, R., PARILLO, M., RIVELLESE, A.A., LASORELLA, G., GIACO, A., D'EPISCOPO, L. & RICCARDI, G. 2000. Long-term dietary treatment with increased amounts of fibre-rich low-

glycaemic index natural foods improves blood glucose control and reduces the number of hypoglycaemic events in type 1 diabetic patients. Diabetes care, 23(10):1461-1471.

GILBERTSON, H.R., EVANS, S., BRAND-MILLER, J.C., THORBURN, A.W. CHONDRA, P. & WERTHER, G.A. 2001. The effect of flexible low glycaemic index dietary advice versus measured carbohydrate exchange diets on glycaemic control in children with type 1 diabetes. Diabetes care, 24(7):1137-1142.

GOUWS, E. & LANGENHOVEN, M. 1986. NRIND Food composition tables. 2nd ed. Parow: South African Medical Research Council.

GRANFELDT, Y., LILJEBERG, H., DREWS, A., NEWMAN, R. & BJÖRCK, I. 1994. Glucose and insulin responses to barley products: influence of food structure and amylose – amylopectin ratio. American journal of clinical nutrition, 59:1075-1082.

GRANFELDT, Y., HAGANDER, B. & BJÖRCK, I. 1995. Metabolic responses to starch in oat and wheat products. On the importance of food structure, incomplete gelatinization or presence of viscous dietary fibre. European journal of clinical nutrition, 49:189-199.

GRANFELDT, Y., ELIASSON, A.C. & BJÖRCK, I. 2000. An examination of the possibility of lowering the glycaemic index of oat and barley flakes by minimal processing. Journal of nutrition, 130:2207-2214.

GRETEBECK, R.J., GRETEBECK, K.M & TITTELBACH, T.J. 2002. Glycaemic index of popular sport drinks and energy foods. Journal of the American Dietetic Association, 102(3):415-417.

GUILLOIN, F. & CHAMP, M. 2000. Structural and physical properties of dietary fibres, and consequences of processing on human physiology. Food research international, 33:233-245.

GUJSKA, E. & KHAN, K. 1990. Effect of temperature on properties of extrudates from high starch fractions of navy, pinto and garbanzo beans. Journal of food science, 55(2):466-469.

HABER, G.B., HEATON, K.W., MURPHY, D. & BURROUGHS, L.F. 1977. Depletion and disruption of dietary fibre: Effects on satiety, plasma glucose and serum insulin. The Lancet, 2(8040):679-682.

HARLAND, B.F. & MORRIS, E.R. 1995. Phytate: a good or bad food component. Nutrition research, 15(5):733-754.

HARPER, J.M. 1995. Extrusion technology: current status and future potential. The South African journal of food science and nutrition, 7(4):135-141.

HEACOCK, P.M., HARTZLER, S.R. & WOLF, B.W. 2002. Fructose refeeding reduces the glycaemic response to a high glycaemic index, starchy food in humans. Journal of nutrition, 132:2601-2604.

HOLLENBECK, C.B., COULSTON, A.M. & REAVEN, G.M. 1988. Comparison of plasma glucose and insulin responses to mixed meals of high, intermediate, and low glycaemic potential. Diabetes care, 11:323-329.

HOLT, S.H.A. & BRAND-MILLER, J. 1994. Particle size, satiety and the glycaemic response. European journal of clinical nutrition, 48:496-502.

HOLT, S.H.A., BRAND-MILLER, J.C., PETOCZ, P. & FARMAKALIDAS, E. 1995. A satiety index of common foods. European journal of clinical nutrition, 66:675-690.

HOROWITZ, M., EDELBROEK, M.A.L., WISHART, J.M. & STRAATHOF, J.W. 1993. Relationship between oral glucose and gastric emptying in normal healthy subjects. Diabetologia, 36(9):857-862.

HUBER, G.R. 1991. Carbohydrates in extrusion processing. Food technology, 45:160-168.
INSTITUTE OF MEDICINE, FOOD AND NUTRITION BOARD. 2003. Dietary Reference Intakes. Washington: National Academy Press.

JÄRVI, A.E., KARLSTROM, B.E., GRANFELDT, Y.E., BJÖRCK, I.M.E. & VESSBY, B.O.H. 1995. The influence of food structure on postprandial metabolism in patients with non-insulin dependent diabetes mellitus. American journal of clinical nutrition, 61:837-842.

JÄRVI, A.E., KARLSTROM, B.E., GRANFELDT, Y.E., BJÖRCK, I.M.E., ASP, N.G.L. & VESSBY, B.O.H. 1999. Improved glycemic control and lipid profile and normalized fibrinolytic activity on a low-glycemic index diet in type 2 diabetic patients. Diabetes care, 22(1):10-18.

JENKINS, D.J.A., WOLEVER, T.M.S., NINEHAM, R., SARSON, D.L., BLOOM, S.R., AHERN, J., ALBERTI, K.G.M.M. & HOCKADAY, T.D.R. 1980a. Improved glucose tolerance four hours after taking guar with glucose. Diabetologia, 19:21-24.

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

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. & GOFF, D.V. 1981. Glycemic index of foods: a physiological basis for carbohydrate exchange. American journal of clinical nutrition, 34:362-366.

JENKINS, D.J.A., WOLEVER, T.M.S., RODNEY, B.M., TAYLOR, R.H., GRIFFITHS, C., KRZEMINSKA, K., LAWRIE, J.A., BENNETT, C.M., GOFF, D.V., SARSON, D.L. & BLOOM, S.R. 1982. Slow release dietary carbohydrate improves second meal tolerance. American journal of clinical nutrition, 35:1339-1346.

JENKINS, D.J.A., WOLEVER, T.M.S., JENKINS, A.L. LEE, R., WONG, G.S. & JOSSE, R.G. 1983. Glycemic response to wheat products: reduced response to pasta but no effect of fibre. Diabetes care, 6:155-159.

JENKINS, D.J.A., WOLEVER, T.M.S., COLLIER, G.R., OCANA, A., VENKETESHWER, A., BUCKLEY, G., LAM, Y., MAYER, A. & THOMPSON, L.U. 1987. Metabolic effects of a low-glycemic index diet. American journal of clinical nutrition, 46:968-975.

JENKINS, D.J.A., WOLEVER, T.M.S., BUCKLEY, G., LAM, K.Y., GIUDICI, S., KALMUSKY, J., JENKINS, A.L., PATTEN, R.L., BIRD, J., WONG, G.S. & JOSSE, R.G. 1988a. Low-glycemic index starchy foods in the diabetic diet. American journal of clinical nutrition, 48:248-254.

JENKINS, D.J.A., WESSON, V., WOLEVER, T.M.S., JENKINS, A.L., KALMUSKY, J., GUIDICI, S., CSIMA, A., JOSSE, R.G. & WONG, G.S. 1988b. Wholemeal versus wholegrain breads: proportion of whole or cracked grain and the glycaemic response. British medical journal, 297:958-960.

JENKINS, D.J.A., AXELSEN, M., KENDALL, C.W., AUGUSTIN, L.S.A., VUKSAN, V. & SMITH, U. 2000. Dietary fibre, lente carbohydrates and the insulin-resistant diseases. British journal of nutrition, 83 (suppl.1): S157-S163.

JENKINS, D.J.A., KENDALL, C.W.C., AUGUSTIN, L.S.A., FRANCESCHI, S., HAMIDI, M., MARCHIE, A., JENKINS, A.L. & AXELSEN, M. 2002. Glycemic index: overview of implications in health and disease. American journal of clinical nutrition, 76(suppl):266-273.

JOANNIC, J., AUBOIRON, S., RAISON, J., BASSDEVANT, A., BORNET, F. & GUY-GRAND, B. 1997. How the degree of unsaturation of dietary fatty acids influences the glucose and insulin responses to different carbohydrates in mixed meals. American journal of clinical nutrition, 65:1427-1433.

KIENS, B., RABEN, A.B. & VALEUR, A.-K. 1990. Benefit of dietary simple carbohydrates on the early post exercise muscle glycogen repletion in male athletes. Medicine and science in sport and exercise, 22(Suppl):588.

KIENS, B. & RICHTER, E.A. 1996. Types of carbohydrate in an ordinary diet affect insulin action and muscle substrates in humans. American journal of clinical nutrition, 63:47-53.

KRITCHEVSKY, D. 1977. Vegetable proteins and atherosclerosis. Journal of American Chemistry Society, 56:135-140.

LAINE, D.C., THOMAS, W., LEVITT, M.D. & BANTLE, J.P. 1987. Comparison of predictive capabilities of diabetic exchange lists and glycemic index of foods. Diabetes care, 10:387-394.

LÉCLERE, C.J., CHAMP, M., BOILLOT, J., GERARD, G., LECANNU, G., MOLIS, C., BORNET, F., KREMPF, M., DELORT-LAVAL, J. & GALMICHE, J.-P. 1994. Role of viscous

gums in lowering the glycemic response after a solid meal. American journal of clinical nutrition, 59:914-921.

LEEDS, A. 2002. Glycemic index and heart disease. American journal of clinical nutrition, 76(suppl):286-289.

LILJEBERG, H., GRANFELDT, Y. & BJÖRCK, I. 1992. Metabolic responses to starch in bread containing intact kernels versus milled flour. European journal of clinical nutrition, 46:561-575.

LILJEBERG, H.G.M. & 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, 64:886-893.

LILJEBERG, H.G.M., GRANFELDT, Y.E. & 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, 126(2):458-466.

LILJEBERG, H.G.M, ÅKERBERG, A.K.E. & BJÖRCK, I.M.E. 1999. Effect of the glycemic index and content of indigestible carbohydrates of cereal-based breakfast meals on glucose tolerance at lunch in healthy subjects. American journal of clinical nutrition, 69:647-655.

LILJEBERG, H. & BJÖRCK, I. 2000. Effects of a low glycaemic index spaghetti meal on glucose tolerance and lipaemia at a subsequent meal in healthy subjects. European journal of clinical nutrition, 54(1):24-28.

LINTAS, C. & CAPPELLONI, M. 1992. Effect of processing on legume resistant starch. European journal of clinical nutrition, 46(suppl. 2):103-104.

LINTAS, C., CAPPELLONI, M., MONTALBANO, S. & GAMBELLI, L. 1995. Dietary fibre in legumes: effect of processing. European journal of clinical nutrition, 49(suppl. 3):298-302.

LIU, S., WILLET, W.C., STAMPFER, M.J., HU, F.B., FRANZ, M., SAMPSON, L., HENNEKENS, C.H. & 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, 71:1455-1461.

LU, Z.X., WALKER, K.Z., MUIR, J.G., MASCARA, T. & O'DEA, K. 2000. Arabinoxylan fibre, a byproduct of wheat flour processing, reduces the postprandial glucose response in normoglycaemic subjects. American journal of clinical nutrition, 71:1123-1128.

LUDWIG, D.S. 2000. Dietary glycemic index and obesity. Journal of nutrition, 130(suppl.):280-283.

MBENYANE, X.G. 1997. The glycaemic index of indigenous South African foods. Potchefstroom: PU for CHE. (Thesis - D.Sc.) 235p.

- MESSINA, M.J. 1999. Legumes and soybeans: overview of their nutritional profiles and health effects. American journal of clinical nutrition, 70(suppl):439S-450S.
- MUNARI, F., ALBERTO, C., WILLIAM, B.P., ANDRACA, A., RAUL, C. & MOISES, C. 1998. Lowering glycaemic index of food by acarbose and plantago psyllium mucilage. Archives of medical research, 29:137-142.
- NELL, T.A. 2001. The variation and application of the glycaemic index of foods. Potchefstroom: PU for CHE. (Thesis - D.Sc.) 145p.
- NERVI, F., COVARRUBIAS, C., BRAVO, P., VELASCO, N., ULLOA, N., CRUZ, F., FAVA, M., SEVERIN, C., DEL POZO, R., ANTEZANA, C., VALDIVIESO, V. & ARTEAGA, A. 1989. Influence of legume intake on biliary lipids and cholesterol saturation in gallstone formation in a highly prevalent area. Gastroenterology, 96:825-830.
- NIELSEN, P.H. & NIELSEN, G.L. 1989. Preprandial blood glucose values: influence on response studies. American journal of clinical nutrition, 49:1243-1246.
- NUTTALL, F.Q., MOORADIAN, A.D., DeMARAIS, R. & PARKER, S. 1983. The glycemic effect of different meals approximately isocaloric and similar in protein, carbohydrate and fat content as calculated using the ADA exchange lists. Diabetes care, 6:432-435.
- NUTTALL, F.Q., MOORADIAN, A.D., BILLINGTON, M.C. & KREZOWSKI, P. 1984. Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. Diabetes care, 7:465-470.
- NOAKES, T.D. 2000. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. Scandinavian journal of medicine and science in sport, 10:123-145.
- OLSON, A., GRAY, G.M. & CHIU, M. 1987. Chemistry and analysis of soluble dietary fibre. Food technology, 41:71-79.
- OOSTHUIZEN, W. 1999. The effect of nutrition on risk factors for coronary heart disease. Potchefstroom: PU for CHE. (thesis-PhD.) 46p.
- OOSTHUIZEN, W., SCHOLTZ, C.C., VORSTER, H.H., JERLING, J.C. & VERMAAK, W.J.H. 2000. Extruded dry beans and serum lipoprotein and plasma haemostatic factors in hyperlipidaemic men. European journal of clinical nutrition, 54:373-379.
- ÖSTMAN, E.M., LILJEBERG ELMSTÅHL, H.G.M. & BJÖRCK, I.M.E. 2001. Inconsistency between glycemic and insulinemic responses to regular and fermented milk. American journal of clinical nutrition, 74:96-100.
- ÖSTMAN, E.M., LILJEBERG, E.H.G. & BJÖRCK, I.M. 2002. Barley bread containing lactic acid improves glucose tolerance at a subsequent meal in healthy men and women. Journal of nutrition, 132(6):1173-1175.

- PACKER, S.C., DORNHORST, A. & FROST, G.S. 2000. The glycemic index of a range of gluten-free foods. Diabetes medicine, 17:657-660.
- PARILLO, M., GIACCO, R., RICCARDI, G., PACIONI, D. & RIVELLESE, A. 1985. Different glycemic responses to pasta, bread, and potatoes in diabetic patients. Diabetes medicine, 2:374-377.
- PI – SUNYER, F.X. 2002. Glycemic index and disease. American journal of clinical nutrition, 76(1):290-298.
- RABE, E. & SIEVERT, D. 1992. Effect of baking, pasta production, and extrusion cooking on formation of resistant starch. European journal of clinical nutrition, 46(suppl.2):105-107.
- RABEN, A., TAGLIABUE, A., CHRISTENSEN, N.J., MADSEN, J., HOLST, J.J. & ASTRUP, A. 1994. Resistant starch: the effect on postprandial glycemia, hormonal response and satiety. American journal of clinical nutrition, 60:544-551.
- RASMUSSEN, O.W., GREGERSEN, S., DØRUP, J. & HERMANSEN, K. 1992. Blood glucose and insulin responses to different meals in non-insulin-dependent diabetic subjects of both sexes. American journal of clinical nutrition, 56:712-715.
- REYES-MORENO, C. & PAREDES-LOPEZ, O. 1993. Hard-to-cook phenomenon in beans - a review. Critical reviews in food science and nutrition, 33(3):227-286.
- ROBERTS, S.B. 2000. High glycemic index foods, hunger, and obesity: is there a connection? Nutrition reviews, 58(6):163-169.
- ROSS, S.W., BRAND, J.C., THORBURN, A.W. & TRUSWELL, A.S. 1987. Glycemic index of processed products. American journal of clinical nutrition, 46:631-635.
- SALMERÓN, J., MANSON, J.E., STAMPFER, M.J., COLDITZ, G.A., WING, A.L. & WILLETT, W.C. 1997a. Dietary fibre, glycemic load and risk of non-insulin-dependent diabetes mellitus in women. Journal of the American Medical Association, 277(6):472-477.
- SALMERÓN, J., ASCHERIO, A., RIMM, E.B., COLDITZ, G.A., SPIEGELMAN, D., JENKINS, D.J., STAMPFER, M.J., WING, A.L. & WILLET, W.C. 1997b. Dietary fibre, glycemic load and risk of non-insulin-dependent diabetes mellitus in men. Diabetes care, 20:545-550.
- SLABBER, M., BARNARD, H.C., KUYL, J.M., DANNHAUSER, A. & SCHALL, R. 1994. Effects of a low-insulin response, energy-restricted diet on weight loss and plasma insulin concentrations in hyperinsulinemic obese females. American journal of clinical nutrition, 60:48-53.
- STANNARD, S.T., THOMPSON, M.W. & BRAND-MILLER, J.C. 2000. The effect of glycaemic index on plasma glucose and lactate levels during incremental exercise. International journal of sport nutrition and exercise metabolism, 10:51-61.

- STEEL, C.J., SGARBIERI, V.C. & JACKIX, M.H. 1995. Use of extrusion cooking to overcome undesirable properties of hard-to-cook dry beans (*Phaseolus vulgaris*L.). Journal of agriculture and food chemistry, 43:2487-2492.
- SWINBURN, B.A., METCALF, P.A. & LEY, S.J. 2001. Long-term (5-year) effects of a reduced fat diet, intervention in individuals with glucose intolerance. Diabetes care, 24(4):619-624, April.
- THIJS, C. & KNIPSCHILD, P. 1996. Legume intake and gallstone risk: results from a case-control study. International journal of epidemiology, 19(30):660-663.
- THOMAS, D.E., BROTHERHOOD, J.R. & BRAND-MILLER, J. 1994. Plasma glucose levels after prolonged strenuous exercise correlate inversely with glycaemic response to food consumed before exercise. International journal of sport nutrition, 4:361-373.
- THOMPSON, L.U. 1988. Anti-nutrients and blood glucose. Food technology, 42:123-130.
- THOMPSON, R., HAYFORD, J. & DARNEY, M. 1978. Glucose and insulin responses to diet: effect of variations in source and amount of carbohydrate. Diabetes, 27:1020-1026.
- THOMPSON, D.G., WINGATE, D.L., THOMAS, M. & HARRISON, D. 1982. Gastric emptying as a determinant of the oral glucose tolerance test. Gastroenterology, 82:51-55.
- THORNE, M.J., THOMPSON, L.U. & JENKINS, D.J.A. 1983. Factors affecting starch digestibility and the glycemic response with special reference to legumes. American journal of clinical nutrition, 38:481-488.
- VAN DER SLUIJS, A.M.C., BEHALL, K.M., DOUGLASS, L., PRATHER, E., SCHOLFIELD, D.J. & HALLFRISCH, J. 1999. Effect of cooking on the beneficial soluble β -glucans in Oatrim. Cereal foods world, 44(4):194-198.
- VENTER, C.S., VORSTER, H.H., VAN ROOYEN, A., KRUGER-LOCKE, M.M. & SILVIS, N. 1990. Comparison of the effects of maize porridge consumed at different temperatures on blood glucose, insulin and acetate levels in healthy volunteers. The South African journal of food science and nutrition, 2(1):2-5.
- VERMEULEN, A. & TURNBULL, W.H. 2000. Feasibility of The GI guide to increase knowledge about the glycaemic index in practice. Journal of human nutrition and dietetics, 13(6):397-405.
- VORSTER, H.H., VAN TONDER, E., KOTZE, P. & 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, 45:575-579.
- VORSTER, H.H. & VENTER, C.S. 1994. Health benefits of dry beans (*Phaseolus vulgaris*): a review. The South African journal of food science and nutrition, 6(2):72-76.

VORSTER, H.H., VENTER, C.S. & SILVIS, N. 1990. The glycaemic index of foods: a critical evaluation. The South African journal of food science and nutrition, 2(1):13-17.

WANG, Y.H.A. & McINTOSH, G.H. 1996. Extrusion and boiling improve rat body weight gain and plasma cholesterol lowering ability of peas and chickpeas. Journal of nutrition, 126:3054-3062.

WEE, S.L., WILLIAMS, C., GRAY, S. & HORABIN, J. 1999. Influence of high and low glycaemic index meals on endurance running capacity. Medicine and science in sports and exercise, 31(3):393-399.

WHO – see World Health Organization.

WOLEVER, T.M.S. 2003. Carbohydrate and the regulation of blood glucose and metabolism. Nutrition reviews, 61:S40-S48.

WOLEVER, T.M.S. & BOLOGNESI, C. 1996a. Source and amount of carbohydrate affect postprandial glucose and insulin in normal subjects. Journal of nutrition, 126:2798-2806.

WOLEVER, T.M.S. & BOLOGNESI, C. 1996b. Time of day influences relative glycaemic effect of foods. Nutrition research, 16(3):381-384.

WOLEVER, T.M.S. & BOLOGNESI, C. 1996c. Prediction of glucose and insulin responses of normal subjects after consuming mixed meals varying in energy, protein, fat, and carbohydrate and glycaemic index. The journal of nutrition, 126:2807-2812.

WOLEVER, T.M.S. & BRAND-MILLER, J. 1995. Sugars and blood glucose control. American journal of clinical nutrition, 62(suppl):21-227.

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

WOLEVER, T.M.S., JENKINS, D.J.A., THOMPSON, L.U., WONG, G.S. & JOSSE, R.G. 1987. Effect of canning on the blood glucose response to beans in patients with type 2 diabetes. Human nutrition: Clinical nutrition, 41C:135-140.

WOLEVER, T.M.S., JENKINS, D.J.A., OCANA, A.M., RAO, V.A. & COLLIER, G. R. 1988. Second-meal effect: lowglycemic index foods eaten at dinner improve subsequent breakfast glycemic response. American journal of clinical nutrition, 48:1041-1047.

WOLEVER, T.M.S., JENKINS, D.J.A., JENKINS, A.L. & JOSSE, R.G. 1991. The glycemic index: methodology and clinical implications. American journal of clinical nutrition, 54:846-854.

WOLEVER, T.M.S., KATZMAN-RELLE, L., JENKINS, A.L., VUKSAN, V., JOSSE, R.G. & JENKINS, D.J.A. 1994. Glycemic index of 102 complex carbohydrate foods in patients with diabetes. Nutrition research, 14:651-669.

WOLEVER, T.M.S., VORSTER, H.H., BJÖRCK, I., BRAND-MILLER, J., BRIGHENTI, F., MANN, J.I., RAMDATH, D.D., GRANFELDT, Y., HOLT, S., PERRY, T.L., VENTER, C.S. & XIAOMEI, W. 2003. Determination of the glycaemic index of foods: interlaboratory study. European journal of clinical nutrition, 57:475-482.

WOLEVER, T.M.S. 1990. The glycemic index. World review of nutrition and dietetics, 62:120-185.

WOLEVER, T.M.S. 1997. The glycemic index: flogging a dead horse? Diabetes care, 20(3):452-455.

WORLD HEALTH ORGANIZATION EXPERT COMMITTEE ON DIABETES MELLITUS: WHO Technical report, Series 727. Geneva, Switzerland: World Health Organization; 1998.

YOUNG, K.W.H. & WOLEVER, T.M.S. 1998. Effect of volume and type of beverage with a standard test meal on postprandial blood glucose responses. Nutrition research, 18(11):1857-1863.

APPENDIX A

Informed consent form

TOESTEMMING: BONEMEEL PROJEK

September 2002

Titel van projek: Die bepaling van die glukemiese indeks van droëbone produkte (muffin, pasta).

Ek, die ondergetekende (volle name)

.....
.....

het die voorafgaande gegewens in verband met die projek gelees en ook die mondelige weergawe daarvan aangehoor en ek verklaar dat ek dit verstaan. Ek was die geleentheid gegun om tersaaklike aspekte van die projek met die projekteier te bespreek en ek verklaar hiermee dat ek vrywillig aan die projek deelneem. Ek gee my toestemming om as proefpersoon in bogenoemde projek op te tree.

Ek vrywaar hiermee die Universiteit asook enige werknemer of student 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
Onderteken

Te.....op.....

GETUIES;

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

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

APPENDIX B

Food fundi for Windows used to analyse macronutrient content of foods

FoodFundi for Windows

Menu 1

Analysis on:- 2002/08/28

Recipe Name : Standard meal Male

Foods	Amount (g)	Energy (kJ)	CHO* (g)	Protein (g)	Fat (g)	Alc (g)
Milk - Low Fat/ Semi Skimmed (2%	25.0	52.0	1.2	0.8	0.5	0.0
Sugar - White Granulated	10.0	169.8	10.0	0.0	0.0	0.0
Tea [Brewed]	180.0	9.0	0.5	0.0	0.0	0.0
Banana [Raw and peeled]	80.0	305.6	16.4	1.0	0.2	0.0
Mustard - Yellow Prepared	5.0	31.0	0.6	0.4	0.4	0.0
Pepper, black	3.0	42.3	1.9	0.3	0.1	0.0
Salt - Table	3.0	0.0	0.0	0.0	0.0	0.0
Egg Chicken [Boiled]	50.0	308.0	0.6	6.3	5.2	0.0
Tomato [Raw]	125.0	113.8	5.0	1.1	0.3	0.0
Margarine - Medium Fat More than	30.0	721.8	0.4	0.1	19.3	0.0
Bread / Rolls - White	180.0	1983.6	94.3	15.3	3.2	0.0
Total		3736.8	130.9	25.4	29.2	0.0
% Energy for meal time			58.5	11.3	29.4	0.0

Foot note:

* Carbohydrate value includes fibre.

FoodFundi for Windows

Menu 1

Analysis on:- 2002/08/28

Recipe Name : Standard meal Female

Foods	Amount (g)	Energy (kJ)	CHO* (g)	Protein (g)	Fat (g)	Alc (g)
Milk - Low Fat/ Semi Skimmed (2%	25.0	52.0	1.2	0.8	0.5	0.0
Sugar - White Granulated	10.0	169.8	10.0	0.0	0.0	0.0
Tea [Brewed]	200.0	10.0	0.6	0.0	0.0	0.0
Banana [Raw and peeled]	80.0	305.6	16.4	1.0	0.2	0.0
Mustard - Yellow Prepared	5.0	31.0	0.6	0.4	0.4	0.0
Pepper - Hot Chili Red [Raw]	2.0	4.8	0.2	0.1	0.0	0.0
Salt - Table	2.0	0.0	0.0	0.0	0.0	0.0
Egg Chicken [Boiled]	50.0	308.0	0.6	6.3	5.2	0.0
Tomato [Raw]	90.0	81.9	3.6	0.8	0.2	0.0
Margarine - Medium Fat More than	20.0	481.2	0.2	0.1	12.9	0.0
Bread / Rolls - White	120.0	1322.4	62.9	10.2	2.2	0.0
Total		2766.7	96.3	19.7	21.5	0.0
% Energy for meal time			58.1	11.9	29.3	0.0

Foot note:

* Carbohydrate value includes fibre.

APPENDIX C

Recipes and portion sizes of all test foods

RECIPES AND PORTION SIZES

GLUCOSE

INGREDIENTS	AMOUNT
Glucose	50g
Water	250ml

METHOD OF PREPARATION

Dissolve glucose in 250ml water the previous night and keep overnight at room temperature.

DRY BEAN FLOUR MUFFINS

INGREDIENTS	AMOUNT
Dry bean flour	200ml
Baking powder	10ml
Salt	2ml
Sugar	100ml
Cake flour	50ml
Pitted raisins	125g
Over-ripe bananas	2
Oil	60ml
Eggs	2
Vanilla flavouring	10ml

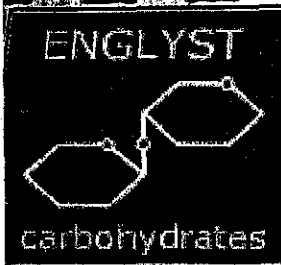
METHOD

1. Preheat oven to 180c.
2. Mix dry bean flour, baking powder and salt together.
3. Add the other dry ingredients and mix well.

4. Mix banana, oil, eggs and vanilla flavouring together.
5. Mix the banana mixture well with the flour mixture.
6. Fill greased muffin tins and bake for 30 minutes

RECIPE FOR WHOLE WHEAT FLOUR MUFFINS

The same recipe as for the dry bean flour muffins. The dry bean flour should be replaced with the same amount of nutty wheat.



Englyst Carbohydrates
 Research and Services Ltd
 2 Venture Road, Chilworth Science Park, Southampton, SO16 7NP, UK
 Tel: 02380 769650
 Fax: 02380 769654
 E-mail: enquiries@englyst.co.uk

Analytical report reference: Potch002

Analytical Report: Free Sugars, RAG, SAG and Starch fractions
 Date: 2-Sep-02
 Prepared by: K.ENGLYST

Request from :
 Potchefstroom (SA) - W. OOSTHUIZEN

Product Name	Carb (g)	Dry Wt (%)	Fru	RAG	SAG	RS(glu)	FSG	RDS(glu)	GG	TS(glu)	TG	SAG
/100g product as analysed												
Bean Pasta		28.4	0.0	19.6	0.5	0.8	0.0	19.6	20.1	20.9	20.9	2.2%
		0.1	0.0	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1	
Brown Muffin		73.2	17.8	28.8	1.8	1.6	17.1	9.5	38.4	14.3	39.0	3.7%
		1.1	2.9	0.2	0.3	0.0	2.6	0.2	0.3	0.2	0.4	
Wheat Muffin		71.9	17.4	30.1	1.5	0.8	17.1	13.0	31.6	15.3	32.4	3.1%
		0.1	0.2	0.1	0.2	0.1	0.1	0.1	0.3	0.3	0.3	

Data are based on triplicate analysis except for Fru and FSG which are duplicates. Analytical standard deviations are provided.

Dry Wt	Dry Weight	FSG	Free Sugar Glucose (including glucose from sucrose)
Fru	Fructose (including fructose from sucrose)	RDS(glu)	Rapidly Digestible Starch (expressed as glucose equivalent)
RAG	Rapidly Available Glucose	GG	Glycemic Glucose (RAG + SAG)
SAG	Slowly Available Glucose	TS(glu)	Total Starch (expressed as glucose equivalent)
RS(glu)	Resistant Starch (expressed as glucose equivalent)	TG	Total Glucose

Reference: Englyst et al (1999), Am J Clin Nutr, 69, 448-454.