Predicting the Glycaemic Index of Mixed Meals

Hayley Ellen Dodd

A thesis submitted in partial fulfilment for the degree of

Master of Science

at the University of Otago, Dunedin, New Zealand

January 2011
Abstract

The Glycaemic Index (GI) provides a measure of the rise in blood glucose following consumption of a test food relative to a reference food. Various associations have been found between certain dietary GIs and a number of diseases. GI has also been used to assist people with diabetes to choose foods that help to stabilise their blood glucose levels. The overall GI of a whole meal or diet has been estimated using a summation model of the individual components. The GI of each component is weighted according to its available carbohydrate contribution.

\[
\text{Meal GI} = \sum \frac{\text{GI}_n \times \text{AvailCHO}_n (g)}{\text{AvailCHO}_{\text{Meal}} (g)}
\]

The validity of this model has not been thoroughly tested. Two simple meals of bread and beans have been used to test the model, with one study finding good agreement, while the other found little agreement between predicted and observed GI. Therefore the aim of this study was to robustly assess how well the summation model predicted the GI of typical mixed meals. A secondary aim was to compare the GI of three meals containing meat, vegetables and sauce that differed in their major carbohydrate source.

Thirty healthy participants aged between 21-49 years old were recruited from the public via posters and e-mails throughout the University of Otago. Fifteen people from each sex including ten from three age brackets (18-30yr, 30-40yr, 40-50yr) were recruited. Four reference glucose beverages (two 50g and two 25g), seven test foods (potato, rice, pasta, kumara, peas, carrots, sauce) and three meals containing potato, rice or pasta plus the other vegetables, sauce and 50g pan-fried chicken were tested by all participants. Nutrient analysis to determine carbohydrate content of the seven test foods was performed. Capillary blood glucose was measured before eating and over two hours post-prandially. Incremental areas under the blood glucose curve were calculated, and a mean GI was obtained for all foods and meals. Meal GI was predicted by inserting the observed GI for each food into the summation model and this was compared to the observed GI for each meal.

Mean (95% CI) GI values for the foods were: potato 72 (62, 85), rice 48 (41, 62), spaghetti 56 (48, 66), kumara 84 (72, 98), peas 29 (25, 34), carrots 31 (27, 36), and sauce 35 (30, 41). Observed and predicted mean (95% CI) GI values for the potato, pasta and rice based meals were: 53 (46, 62) cf 63 (56, 70), 38 (33, 45) cf 51 (45, 56) and 38 (33, 44) cf 55 (49, 61).
respectively. The predicted meal GIs were greater than the observed meal GIs in all three cases (p<0.001). The summation model overestimated GI by 19-45% when applied to mixed meals.

The present study provides reliable information regarding the ability of the summation model to predict a composite GI. Using measured and published GI values for foods resulted in significant and variable overestimation of measured meal GIs. Researchers using this model to predict meal or dietary GI should be aware of the limitations associated with the model.
Preface

The research study undertaken in this thesis was conceived by the candidate’s supervisor, Dr Bernard Venn, who was also responsible for applying for funding, sample size determination, study design and research protocols. Dr Rachel Brown was involved in study design and protocols. Associate Professor Sheila Williams was involved in sample size determination and the statistical analysis of the results. This included the mixed model analysis to calculate GI of foods and meals and predict the GI of the mixed meals using values obtained in this study. Gribbles Veterinary Pathology, Mosgiel, and AsureQuality, Auckland, were responsible for nutrient analysis of the test foods.

In 2009, funding was obtained from the University of Otago Performance-Based Research Fund (PBRF) fund to carry out a study investigating whether the existing summation model could predict the GI of meals.

The candidate was responsible for the following under supervision:

- Completing and submitting the ethics application to the University of Otago Human Ethics Committee.
- Study design and protocols
- Preparing foods and conducting initial tests to determine carbohydrate content.
- Preparing foods and organising nutrient analysis with external laboratories.
- Testing glycaemic responses to pan-fried chicken to ensure the chicken would not elicit a glycaemic response.
- Choosing the test foods and designing the meals.
- Calculating the amount of food required to obtain the correct amount of available carbohydrate in the test foods or meals (either 25g or 50g available carbohydrate).
- Pre-testing the foods and meals to make sure they were palatable and could be eaten within the 10-12 minute time frame. A brief questionnaire on food acceptability was designed by the candidate and given to 5 volunteers who pre-tested several foods and meals. Comments were taken into account in final meal design.
- Recruiting all participants, with correct ages and sex accounted for.
- Employing and training all GI laboratory staff and cooking assistants.
- Scheduling all work rosters.
- Budgeting of clinic supplies, food and reimbursement costs.
• Ordering laboratory supplies and food.
• Setting-up and co-ordinating clinics.
• Preparing and cooking all test foods and meals, in accordance with food safety guidelines. Food preparation was also completed with the help of a research assistant when needed.
• Taking anthropometric measurements of all participants.
• Arranging reimbursement for all participants.
• Data collection and entry.
• Meeting with the statistician and completing part of the statistical analysis.
• Writing this thesis.

The research was presented as an oral presentation at the 2010 New Zealand Nutrition Society Conference in Wellington, New Zealand before the submission of this thesis. This research has also been submitted as a paper to the American Journal of Clinical Nutrition. The manuscript can be viewed in Appendix 10.
Acknowledgements

I would like to acknowledge the following people, whose support and generosity enabled me to complete this thesis:

To my participants, thank you. I am so grateful for your kindness and willingness to get up early and eat whatever we placed in front of you! This research would not have been possible without you.

Thanks to Danielle, Rose, Jessie, Kerryn, Kavitha, Asher and Pauline; my super-star finger pricking team for working so hard and making those early mornings more bearable. Thanks also to Danielle and Michelle for donning the apron and joining me in the kitchen at outrageous hours of the morning.

My sincere gratitude goes to Bernard; you have been an amazing supervisor. I couldn’t have asked for more from you and I truly admire your enthusiasm and keenness for knowledge and science. Thank you for your continued support, patience and guidance through this project.

To Rachel, thank you for always being there when I needed that extra bit of input and advice, especially towards the end. I am truly grateful for your help.

To Sheila, I sincerely appreciate your readiness to help and fresh ideas you have brought to this project.

To Kieran, Ivy and Marlenne, thank you for helping me with carbohydrate testing and putting up with my frustrations and mess.

To the postgraduate students and staff of the Human Nutrition department, for always being so positive, baking the most delicious treats and for helping to make this experience enjoyable.

Thank you to all of my friends, especially Michelle, Camilla and Shonelle, for the countless cups of tea, squares of chocolate, cookies and dream sharing. Thank you for getting me through those tough times. You are amazing monkeys.

My family, thank you so much for your continued support and belief in me through all my studies. And special thanks to my most wonderful companion, Warren, for just being awesome.
# Table of Contents

Abstract ......................................................................................................................................... ii

Preface ........................................................................................................................................... iv

Acknowledgements ....................................................................................................................... vi

Table of Contents ........................................................................................................................... vii

List of Tables and Figures ............................................................................................................. ix

Abbreviations ................................................................................................................................. x

1 Introduction .................................................................................................................................. 1

2 Literature Review ....................................................................................................................... 3
   2.1 Introduction ............................................................................................................................ 3
   2.2 The Glycaemic Index ............................................................................................................. 4
       2.2.1 Reliability of Glycaemic Index Values .......................................................................... 8
       2.2.2 Dietary Glycaemic Index ............................................................................................. 10
   2.3 Application of the Glycaemic Index to Mixed Meals .......................................................... 14
       2.3.1 Predicting Meal Glycaemic Index ............................................................................... 14
       2.3.2 Effect of Glycaemic Index of Major Carbohydrate Source on Glycaemic Response ...... 19
   2.4 The Glycaemic Index In Human Health and Disease ............................................................ 23
       2.4.1 Intervention Studies Involving the Glycaemic Index ................................................. 23
       2.4.2 The Use of Glycaemic Index in Cohort Studies ......................................................... 25
   2.5 Summary ............................................................................................................................... 27

3 Methods ...................................................................................................................................... 28
   3.1 Ethics ..................................................................................................................................... 28
   3.2 Participants ............................................................................................................................ 28
       3.2.1 Sample Size Calculation ............................................................................................. 28
       3.2.2 Recruitment ................................................................................................................... 28
   3.3 Nutrient Analysis ................................................................................................................... 29
       3.3.1 Pre-testing of Chicken ............................................................................................... 30
   3.4 Study Protocol ....................................................................................................................... 31
       3.4.1 Blood Glucose Concentration ..................................................................................... 31
       3.4.2 Anthropometric Measurements ................................................................................. 32
List of Tables and Figures

Table 2.1 Factors affecting glycaemic response ................................................................. 10
Table 2.2 Calculating meal GI ............................................................................................. 12
Table 2.3 Predicted and observed mean±SD GI for meals containing bread and beans .......... 15
Table 2.4 Predicted and observed mean GI for six ethnic mixed meals ................................. 16
Table 2.5 Predicted and observed mean GI values for composite breakfast meals ................. 17
Table 2.6 Studies testing predictability and rank of glycaemic response ............................. 19
Table 2.7 Intervention studies on GI and markers for diabetes ............................................ 23
Table 2.8 Overview of recently published cohort studies and risk of T2DM ......................... 25
Table 3.1 Nutrient content of the foods from laboratory analysis, company data (chicken only) and
reference values .................................................................................................................. 29
Table 3.2 Amount of food (g) required to obtain 25g or 50g available carbohydrate per test food or
meal ...................................................................................................................................... 30
Table 3.3 Total energy (kJ) and nutrient content of potato, rice and pasta meals .................. 30
Table 4.1 Mean (SD) values for characteristics of study sample (n=30) ............................... 36
Table 4.2 Observed IAUC and GI values (presented as geometric mean (95% CI) for all items and
published mean GI (range) values for single foods ............................................................ 37
Table 4.3 Mean (95% CI) observed and predicted meal GI .................................................. 38
Table 4.4 Predicted meal GIs from published values (Atkinson et al., 2008) ......................... 38
Table 4.5 Predicted mean (95% CI) meal GIs using main carbohydrate source plus kumara or
vegetables only ....................................................................................................................... 39

Figure 2.1 Blood glucose increments over time ................................................................... 5
Figure 2.2 Blood glucose responses of hypothetical high and low GI foods .......................... 7
Figure 4.1 Mean changes in plasma glucose for potato, rice and pasta meals ...................... 37
Figure 4.2 Variation in IAUC values for reference foods ..................................................... 39
## Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ApoA</td>
<td>Apolipoprotein A</td>
</tr>
<tr>
<td>ApoB</td>
<td>Apolipoprotein B</td>
</tr>
<tr>
<td>AOAC</td>
<td>Association of Official Analytical Chemists</td>
</tr>
<tr>
<td>AUC</td>
<td>Area Under the Curve</td>
</tr>
<tr>
<td>AvailCHO</td>
<td>Available Carbohydrate</td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>CHO</td>
<td>Carbohydrate</td>
</tr>
<tr>
<td>CV</td>
<td>Coefficient of variation</td>
</tr>
<tr>
<td>DDR</td>
<td>Day Diet Record</td>
</tr>
<tr>
<td>FAO/WHO</td>
<td>Food and Agriculture Organisation/World Health Organisation</td>
</tr>
<tr>
<td>FFQ</td>
<td>Food Frequency Questionnaire</td>
</tr>
<tr>
<td>GI</td>
<td>Glycaemic Index</td>
</tr>
<tr>
<td>GIP</td>
<td>Gastric Inhibitory Peptide</td>
</tr>
<tr>
<td>GL</td>
<td>Glycaemic Load</td>
</tr>
<tr>
<td>HbA₁c</td>
<td>Glycated Haemoglobin</td>
</tr>
<tr>
<td>HDL</td>
<td>High Density Lipoprotein Cholesterol</td>
</tr>
<tr>
<td>HGI</td>
<td>High Glycaemic Index Diet</td>
</tr>
<tr>
<td>IAUC</td>
<td>Incremental Area Under the Curve</td>
</tr>
<tr>
<td>LDL</td>
<td>Low Density Lipoprotein Cholesterol</td>
</tr>
<tr>
<td>LGI</td>
<td>Low Glycaemic Index Diet</td>
</tr>
<tr>
<td>MUFA</td>
<td>Monounsaturated Fatty Acids</td>
</tr>
<tr>
<td>PPG</td>
<td>Postprandial Glycaemia</td>
</tr>
<tr>
<td>TAG</td>
<td>Triacylglycerides</td>
</tr>
<tr>
<td>T1DM</td>
<td>Type 1 Diabetes Mellitus</td>
</tr>
<tr>
<td>T2DM</td>
<td>Type 2 Diabetes Mellitus</td>
</tr>
</tbody>
</table>
1 Introduction

The glycaemic index (GI) has been widely researched over the past 40 years. A high dietary GI has been associated with increased risk of diseases such as type II diabetes, obesity, cardiovascular disease, the metabolic syndrome and various forms of cancer (Augustin et al., 2003; 2004a; 2004b; Cho et al., 2003; Folsom et al., 2003; Grau et al., 2010; Higginbotham et al., 2004a; Jonas et al., 2003; Levitan et al., 2010; Meyer et al., 2000; Salmeron et al., 1997a; 1997b; Schulze et al., 2004; Silvera et al., 2005a; 2005b). Although there is a considerable body of literature suggesting benefit from a low dietary GI, there are also a number of studies in which GI had no discernable effect on disease risk (Higginbotham et al., 2004b; Hodge et al., 2004; Johnson et al., 2005; Mosdol et al., 2007; Sahyoun et al., 2008; Similä et al., 2010; Stevens et al., 2002). A low GI diet has shown to be beneficial in reducing disease markers and symptoms in those with certain diseases such as type II diabetes and cardiovascular disease (De Natale et al., 2009; Jarvi et al., 1999; Jenkins et al., 2008; Jimenez-Cruz et al., 2003; Rizkalla et al., 2004; Wolever et al., 2008b). However, the literature in relation to disease risk and GI is inconsistent and cannot be overlooked, especially when several public health agencies including the World Health Organisation (WHO) recommend GI to guide food choices and help prevent certain diseases (FAO/WHO, 1998).

Although GI is tested with individual foods, this data has been used to obtain the GI of a whole diet in which each food’s GI is weighted according to its carbohydrate contribution (Salmeron et al., 1997a; 1997b). This dietary GI has been used in studies to link GI with certain diseases. However, the validity of this current method has not been tested thoroughly. The model has been tested twice using simple meals of bread and beans. One study found good agreement between predicted and observed GI, while the other found little agreement (Jenkins et al., 1984b; Wolever et al., 1985). Two other studies used published GI values to predict meal GI, and again one found a good correlation while the other found a very poor correlation between predicted and observed GI (Chew et al., 1988; Flint et al., 2004). Several factors may limit the application of the summation model including the presence of other nutrients (namely fat and protein), processing (cooking, milling) and food variety, ripeness and origin. In addition the use of published nutrient profiles of the test foods, small sample sizes and variable methodologies limit the current literature on this topic.
Although the summation model has been extensively used to predict the GI of meals and diets, at the present time, no study has robustly tested the model. It can therefore not be determined if the formula is suitable to predict a composite GI. The primary aim of this study is to test how well the GI of cooked meals can be predicted by summing the weighted GI of individual foods contributing to the meals. It has also been suggested that the main carbohydrate source can predict meal GI and that a combination of foods and nutrients will slow the rate of absorption of carbohydrate. Therefore, a secondary aim is to compare the GI of three meals containing meat, vegetables and sauce but differing in their major carbohydrate source - a low, medium and high GI food.
2 Literature Review

2.1 Introduction

Differences in the glycaemic response to various carbohydrate containing foods was first investigated by Otto et al. (1973; 1980). These discoveries led to the development of the glycaemic index; a concept that ranks carbohydrate-containing foods based on their effect on blood glucose levels (Jenkins et al., 1981). GI is usually tested with individual foods but as very few foods are consumed solely as a meal it is important for GI to apply to mixed meals also. A summation model has been developed to predict the GI of meals. This model weights each food’s GI according to it available carbohydrate contribution. The summation model has also been used in observational research to predict total dietary GIs that have subsequently been linked with certain diseases (Salmeron et al., 1997a). However, research focusing exclusively on the summation model and its capacity to predict the GI of mixed meals is limited and inconsistent. Thus, further investigation into this matter is warranted.

The following literature review aims to:

1. Provide a brief overview of GI;
2. Describe the reliability and variability of GI;
3. Discuss the application of the GI and glycaemic response to mixed meals;
4. Discuss the relationship between GI and human health and disease.

Relevant literature published in English between 1980-2010 and using human participants was sought via the databases PubMed, Medline via Ovid and Scopus using key MeSH of ‘glycemic index’ or ‘glycaemic index’ combined with the following words: dietary intake, factors affecting, methodology, determining dietary, type 2 diabetes, obesity, cancer, randomized controlled trial, diet, mixed meals, glycemic response, sample-size, intra-personal variation, inter-personal variation, reliability. Additional literature was found through reference lists of published journal articles. The Australian Standard referred to throughout this thesis is the Australian Standard on glycemic index of foods, published in 2007 by Standards Australia. It will be referred to as ‘Australian Standard (2007)’. 
2.2 The Glycaemic Index

Definition
The GI is defined as the “incremental area under the blood glucose response curve of a portion of carbohydrate from a test food expressed as a percent of the response to the same amount of carbohydrate from a reference food taken by the same subject” (FAO/WHO, 1998; Jenkins et al., 1981; Truswell, 1992). The carbohydrate referred to is carbohydrate that contributes to postprandial glycaemia or ‘available’ carbohydrate and excludes dietary fibre.

Glycaemic Index Protocol
GI values have been obtained using non-diabetic, type I and type II diabetic participants; the concept appears to rank foods similarly in these three groups (Crapo et al., 1981; Wolever et al., 1987). A brief overview of the protocol to measure GI is as follows (Australian Standard, 2007; Brouns et al., 2005; Wolever et al., 1991; FAO/WHO, 1998):

1. Two baseline blood samples are taken before the food is ingested.
2. A food portion containing up to 50g available carbohydrate is given to the participant after an overnight (10hr) fast.
3. Blood samples are taken to test blood glucose levels over a 2-hr period for non-diabetic participants. Samples are collected at 15, 30, 45, 60, 90, 120 minutes after the test food is consumed.
4. Capillary or venous blood is used to determine blood glucose concentration, however capillary blood is preferred because it has been found to be less variable (FAO/WHO, 1998; Vrolix & Mensink, 2010; Wolever et al., 2003).

Calculating the Glycaemic Index
The incremental area under the blood glucose response curve (IAUC) is the sum of the areas between blood collection time-points calculated using the trapezoidal rule. Areas below baseline are ignored. The equation is as follows (Wolever & Jenkins, 1986):

\[
\text{Area} = \left( A + B + C + \frac{D}{2} \right) t + \frac{(D + E) T}{2} + \frac{E^2 T}{2(E + F)}
\]

Where A-E represent positive blood glucose increment values from baselines, F equals the first negative increment value, t equals the 15-minute time interval between blood samples and T is the 30-minute time interval (Figure 2.1) (Source: Wolever & Jenkins, 1986). The blood
glucose concentration often falls below fasting levels, which results in a negative blood glucose increment value. In this case the area is calculated as the portion of the triangle \(0.5 \times (T \times (E + F))\) which is above zero.

![Figure 2.1 Blood glucose increments over time.](image)

To calculate the GI of a food the following equation is used (Jenkins et al., 1988):

\[
GI = \frac{\text{IAUC of test food}}{\text{Mean IAUC of reference food}} \times 100
\]

Results for the GI of foods are commonly expressed as mean±SEM. The food may be classified into one of three categories depending on its mean GI value. Foods are classified as low GI if the GI value is less than 55; moderate GI if between 55-69; and high GI if greater than 70 (Brand-Miller et al., 2003). The categories are arbitrary and do not necessarily relate to healthy food choices. For example, foods with a high sugar, high fat content such as ice cream, have a low GI, whereas many fruits have a medium GI (Atkinson et al., 2008).

**Available Carbohydrate Portion**

Not all carbohydrate is available for digestion (i.e. broken down into monosaccharides and absorbed), so only the available, or glycaemic portion of carbohydrate is measured and used (FAO/WHO, 1998). Note that fructose is the exception as it is relatively non-glycaemic (Englyst et al., 2003; Henry et al., 1991; Wolever & Brand-Miller, 1995). Available carbohydrate can be defined as ‘total carbohydrate less dietary fibre’ (Brouns et al., 2005) and determined ‘by difference’ (FAO/WHO, 1998). This method involves measuring the food components of water, fat, protein, ash and total dietary fibre, summing the values and subtracting the sum from 100; the remainder is then taken as the available carbohydrate.
content of the food (AOAC, 2005). The method has inaccuracies because each separate test for the components is associated with error and the errors accumulate in the summation. Additionally, the leftover fraction may also contain a small amount of non-available carbohydrate from the assay (Brouns et al., 2005; FAO/WHO, 1998). An alternative method is to measure the carbohydrate amounts of sugars and starch directly, although this also involves a number of separate determinations and the summation of monosaccharide fractions. Nevertheless, it is considered to be better than the ‘by difference’ method (Brouns et al., 2005).

When testing the GI of foods, a 50g portion size is most commonly used; this is the portion size on which GI is based upon (Brouns et al., 2005; Jenkins et al., 1981; Wolever et al., 1991). A dose-response effect is seen between increasing amount of carbohydrate and IAUC, but this relationship is curve-linear and plateaus after 50g carbohydrate (Brouns et al., 2005; Venn et al., 2006). Occasionally smaller portions of 25g available carbohydrate are used (Jenkins et al., 1981). This amount is particularly useful when the required portion size of the food, for example non-starchy vegetables, have a small carbohydrate content and a portion containing 50g available carbohydrate would be too large to consume in one sitting.

Reference Food

White bread, glucose beverages and white rice have been used as reference foods, and therefore assigned a GI value of 100 (Sugiyama et al., 2003; Wolever et al., 1985). In theory, any carbohydrate-rich food could be used, but in practice the choice is limited to facilitate comparability among studies. These reference foods do not have the same glycaemic responses as each other even though they are assigned the same GI value. The calculated GI of test foods are therefore relative to the reference food in which they were compared with, and this must be taken into account when comparing the GI of foods.

There have been concerns regarding the digestion of glucose beverages as they have an osmotic effect, which may lead to more rapid gastric emptying (Truswell, 1992; Wolever, 1990; Wolever et al., 1991). In addition, they contain only carbohydrate, whereas other reference foods contain protein and fat, both of which affect glycaemic response and gastric emptying (Wolever, 1990; Wolever et al., 1991). Often, the choice of reference food is determined by the usual practice or preference of individual GI testing laboratories.

The use of white bread as a reference food compared with a glucose beverage has been investigated (Wolever et al., 1985; 1991). Healthy participants (n=23) consumed 21 different
foods, with each food being tested between 4-23 times. The glycaemic response was 38% higher with glucose, hence GI values obtained when using white bread as the standard are deemed to be 1.38 times that of GI values from glucose (Wolever et al., 1991). The FAO/WHO report (FAO/WHO, 1998) states that white bread is an appropriate reference food, and the International tables of glycemic index and glycemic load values (Atkinson et al., 2008) present values for foods obtained from both white bread and glucose. Conversely, it has been shown that bread may not be a suitable reference food as it is not a consistent food and loses water at an indoor temperature affecting the weight and carbohydrate portion (Pi-Sunyer 2002; Truswell, 1992). Differing bread volumes with the same macronutrient composition can also alter glycaemic response and GI (Burton & Lightowler 2006); hence the GI value of white bread relative to glucose is not constant (Bornet et al., 1987; Pi-Sunyer 2002; Wolever et al., 1991).

**Measuring the Blood Glucose Response**

The glycaemic index is based on the glycaemic response to food being a function of the rates of digestion and blood clearance of the carbohydrate in a food. The most commonly accepted and known concept of a high GI food is one that has a large proportion of carbohydrate that is readily available and easily digested. The blood glucose concentration will peak soon after the consumption of high GI foods (Figure 2.2) (Source: Wolever, 1990). This response will decrease quickly, often dipping below the baseline blood glucose concentration. Likewise, if the food has a small proportion of easily digested carbohydrate, the glucose response will be delayed, and return to baseline over a longer period of time (Jenkins et al., 1980b; Venn & Green, 2007; Wolever et al., 1991). Low GI foods rarely fall below baseline glucose concentration.

![Figure 2.2](image)

**Figure 2.2** Blood glucose responses of hypothetical high and low GI foods.
The glycaemic response to the food should be recorded in the two hours following food consumption for normal participants, and in the following three hours for diabetic participants (Wolever et al., 1991). Measurements can be taken until blood glucose levels are back to baseline (Wolever et al., 1991) but it has been suggested that this would lead to greater within-subject variation (Wolever et al., 2003). Testing blood glucose concentrations for longer, for example up to 4 hours, would result in little difference in the IAUC between high and low GI foods, hence the significance of the earlier rise in glucose concentrations would be lost (Pi-Sunyer 2002).

### 2.2.1 Reliability of Glycaemic Index Values

**Sample Size**

Earlier studies examining the glycaemic potential of carbohydrate containing foods used sample sizes that were adequate to determine differences attributable to the sample examined. For example, glycaemic responses of diabetic and healthy participants differ greatly, and hence a small sample size (n~6) was suitable to note postprandial glycaemic differences between these groups (Coulston et el 1984; Jenkins et al., 1981; 1984; Wolever et al., 1985; 1989). With a growing interest to determine GI differences between foods or diets rather than people, sample size numbers have not increased suitably. The 1998 FAO/WHO Consultation on Carbohydrates states that 6 or more people are required to determine the GI of a food (FAO/WHO, 1998) with no explanation given as to why this number is appropriate. However, a recent overview (Brouns et al., 2005) concluded a sample of 10 people would provide a “reasonable degree of power and precision for most purposes”, but if more precise GI results are required a larger sample (20-30) should be tested. In line with this statement, the Australian Standard (2007) requires a minimum of 10 healthy subjects to be tested.

There is potential to misclassify foods when the measure of variance for individual foods overlaps (Brouns et al., 2005). In addition, an inherent property of GI is that the variance increases with the mean, and showing differences in GI between foods is dependent on sample size therefore (Venn & Green, 2007; Wolever et al., 2003). An inter-laboratory study demonstrated the margin of error as well as the difference in GI that can be detected (80% power, p<0.05) decreased with increasing number of subjects over a range of GI values (Wolever et al., 2003). Thus, if GI is tested in small samples, the results will be imprecise and the GI may be inaccurate. This may lead to misclassification, particularly for medium to high
GI foods. This effect was apparent in the same inter-laboratory study in which different laboratories classified the same rice as being low, medium or high GI (Wolever et al., 2003).

Issues arising from poor reliability of GI can be improved by increasing the sample size (Williams et al., 2008). In order to achieve better precision and to gain more confidence in the classification of a food, more than 10 participants are required. More participants may become practically and financially prohibitive, but as a guide it has been suggested that 25 participants should provide reasonable estimates (Venn & Green, 2007).

Repeated Testing of the Reference Food
The Australian Standard (2007) as well as the FAO/WHO (1998) specifies that the reference food should be tested three times in all participants. Repeated testing improves the reliability of results (Brouns et al., 2005; Williams et al., 2008), however it also incurs extra cost. The greatest decrease in variability is obtained when the reference food is repeated once; lesser gains are achieved with additional replicates (Venn et al., 2006; Hättonen et al 2006; Williams et al., 2008; Wolever et al., 2003). Due to the cost of multiple replicates and the observation that the greatest gain in precision occurs with just one replicate, it is suggested in a document on GI methodology that the reference food should be tested at least twice in each participant (Brouns et al., 2005).

Intra- and Inter-personal Variation
A substantial amount of the variability surrounding GI is associated with intra-individual variation (Brouns et al., 2005; Vega-Lopez et al., 2007; Vrolix & Mensink, 2009; Williams et al., 2008; Wolever et al., 1989; 2003; 2008a). Large confidence intervals and poor reliability of GI tests could be due almost entirely to variation within a person (Williams et al., 2008). This indicates that GI is more suitably applied to groups of people rather than individuals (Williams et al., 2008). As each person acts as his or her own control, intra-individual variation should be a null issue, however greater intra-individual variation is consistently seen compared to inter-individual variation (Brouns et al., 2005; Vega-Lopez et al., 2007; Vrolix & Mensink, 2009; Williams et al., 2008; Wolever et al., 2003). It is difficult to reduce intra-individual variation to a great degree, even when using standardised testing protocols.

Conclusion
Increasing the sample size, number of replicates of the food and minimising intra-individual variation will provide greater precision and confidence in GI results. These are issues that should be addressed if GI is to be applicable to the public as a tool to encourage healthy
**Table 2.1 Factors affecting glycaemic response.**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Effect on Glycaemic Response</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nature of carbohydrate</strong></td>
<td></td>
</tr>
<tr>
<td>Available sugars</td>
<td>Glucose is more glycaemic than sucrose$l&gt;$lactose$l&gt;$fructose. Fructose exhibits a lower glucose response compared to glucose and has a low GI (20).</td>
</tr>
</tbody>
</table>
| Starch structure            | Starch crystalline structure can affect the rate of hydrolysis. The higher the amylopectin:amylose ratio, the faster the digestions as digestive enzymes more easily access the branched structure of amylopectin. Starch structure can be altered by:  
  • Gelatinisation- disrupts the cell structure making it more available to digestive enzymes.  
  • Retrogradation- digestive enzymes may not be able to act effectively on recrystallised starch  
  • Degree of ripening- higher glycaemic response and GI with riper fruits and vegetables. |
| Amount                      | The larger the total amount of carbohydrate the longer digestion will take (associated with glycaemic load).                                                                                                                                 |
| **Presence of other nutrients** |                                                                                                                                                                                                                           |
| Fibre                       | Total fibre and GI do not correlate, as different types of fibre have different effects, for example viscous gel-forming fibres lower the glycaemic response. Fibre is not digested in the small intestine, and does not contribute to immediate glucose supply. |
| Fat                         | The inclusion of fat in meals lowers the glycaemic response and GI by delaying gastric emptying and/or reduces starch gelatinisation. Some have noted that the amount required to make a significant difference to the glycaemic response is larger than what would be usually consumed (e.g. >40g/50g CHO portion). |
| Protein                     | Protein may lower the glycaemic response and GI by increasing insulin secretion.                                                                                                                                              |
| Anti-nutrients (e.g. tannins) | Decreases the rate of starch digestion.                                                                                                                                                                                    |
| **Processing**              |                                                                                                                                                                                                                             |
| Milling and grinding        | Starch particles within an intact grain are less accessible to digesting enzymes. Starch from milled particles that have had their cell walls removed are more easily accessible and therefore digested more quickly. |
| Cooking                     | Increases the digestibility of the starch and therefore GI of some foods.                                                                                                                                                   |
| Fermentation                | Organic acids formed during fermentation of some bread delays gastric emptying.                                                                                                                                              |
eating. Thus findings will be more useful when ranking and classifying foods based on GI for groups of people.

### 2.2.2 Dietary Glycaemic Index

#### Effect of Dietary Factors on the Glycaemic Index

Several dietary factors that affect the glycaemic response and therefore the GI of foods include the nature of the carbohydrate, the consumption of carbohydrate with other nutrients, and processing of the carbohydrate (Arvidsson-Lenner et al., 2004; Bjorck et al., 1994; FAO/WHO, 1998; Gulliford et al., 1989; Jenkins et al., 1981; 1988; Liljeberg et al., 1995; Normand et al., 2001; Owen & Wolever 2003; Pi-Sunyer 2002; Sugiyama et al., 2003; Truswell, 1992; van Loon 2000; Wolever 1991). It is difficult to control for all of these factors when testing GI. A summary of several dietary factors and their effect on the glycaemic response is presented in Table 2.1.

The addition of fat and protein to carbohydrate containing foods flattens the glycaemic response and lowers overall GI (Brand et al., 1985; Collier & O’Dea 1983; Flint et al., 2004; Henry et al., 2006; Jenkins et al., 1981; Nuttall et al., 1984; Ross et al., 1987; Sugiyama et al., 2003; Trout et al., 1993). However, it has been suggested that the amount of fat and protein required to have a substantial effect on the glycaemic response may be greater than that consumed in a normal dietary pattern (Brand et al., 1985; Collier et al., 1984; Collier & O’Dea 1983; Gannon et al., 1993a; 1993b; Goddard et al., 1984; Jenkins et al., 1984a; Ross et al., 1987; Truswell, 1992; Wolever, 1990; Wolever et al., 1990; 1991). A large amount of fat, for example 40-50g fat to every 50g carbohydrate has been shown to significantly reduce IAUC and blood glucose peak rise (Gannon et al., 1993a; 1993b; Owen & Wolever 2003; Wolever et al., 1994). Likewise, differences in glycaemic responses are not seen until about 25g of protein per 50g CHO is added (Wolever, 1990). The smaller addition of fat and protein is likely to be the reason why no difference in postprandial glycaemia has also been documented (Jenkins et al., 1983; Miller et al., 2003; Wolever & Bolognesi, 1996). However, significant inverse correlations between protein (5-28g) and fat content (3-42g) and glycaemic response were found when thirteen breakfast meals were tested, with $r^2$ values of 0.65 and 0.66 respectively ($p<0.001$) (Flint et al., 2004). This indicates relatively small amounts of protein and fat may influence post-prandial glycaemia. In addition, the effect of fat and carbohydrate contents of the evening meal before GI testing has been shown to have no influence on GI (Ning et al., 2010).
The proposed mechanisms by which these nutrients affect blood glucose concentration are:

- Protein produces greater gastric inhibitory peptide (GIP) and insulin responses resulting in a lower postprandial glucose peak and a reduced glycaemic response of high GI foods (Bornet et al., 1987; Gannon et al., 1988; Gulliford et al., 1989; Simpson et al., 1985).
- Fat delays gastric emptying, thereby slowing digestion and absorption of glucose (Gulliford et al., 1989; Henry et al., 2006; Normand et al., 2001). It may also affect the interaction among plasma glucose, insulin and GIP (Gannon et al., 1993a; 1993b; Owen & Wolever 2003; Simpson et al., 1985).

Calculating the Glycaemic Index of Mixed Meals
Although GI is tested with individual foods, a summation model has been used to predict a composite GI in which each food’s GI is weighted according to its available carbohydrate contribution (Salmeron et al., 1997a). As very few meals consist solely of a single food, it is important for the GI to apply well to mixed meals. Meals often contain several carbohydrate containing foods and each of these is taken into account proportionally in the model in order to determine total meal GI. The GI of each individual food should be determined when predicting total meal GI (FAO/WHO, 1998), but in some circumstances published GI values have been used (Chew et al., 1998; Flint et al., 2004).

The GI of a meal is expressed as “the weighted mean of the GI values of each of the component foods, with the weighting based on the proportion of the total meal carbohydrate provided by each food” (FAO/WHO, 1998; Wolever et al., 1985; 1991). The following equation was therefore designed to estimate composite GI (Salmeron et al., 1997a):

$$\text{Meal GI} = \frac{\sum_n (\text{GI}_n \times \text{AvailCHO}_n \text{ (g)})}{\text{AvailCHO_{Meal} (g)}}$$

Where ‘n’ represents the foods in the meal and AvailCHO is the available carbohydrate content in the food or meal. An example of the model is presented in Table 2.2.

The validity of this model has not been robustly tested and issues arising from the use of this model are covered more thoroughly in Section 2.3.
Table 2.2 Calculating meal GI.

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>GI of food</th>
<th>Available food CHO (g)</th>
<th>Available meal CHO (g)</th>
<th>Weighted meal GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food 1</td>
<td>80</td>
<td>× 25</td>
<td>/</td>
<td>50</td>
</tr>
<tr>
<td>Food 2</td>
<td>60</td>
<td>× 15</td>
<td>/</td>
<td>50</td>
</tr>
<tr>
<td>Food 3</td>
<td>20</td>
<td>× 10</td>
<td>/</td>
<td>50</td>
</tr>
<tr>
<td>Total available meal CHO (g)</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Total meal GI** 62

CHO = carbohydrate

Ascertainment of Dietary Glycaemic Index

There is considerable interest in the relationship between dietary GI and risk of chronic diseases such as diabetes, cancer and heart disease (Augustin et al., 2003; 2004a; 2004b; Barclay et al., 2008a; Cho et al., 2003; Folsom et al., 2003; Grau et al., 2010; Higginbotham et al., 2004a; 2004b; Jonas et al., 2003; Larsen et al., 2010; Levitan et al., 2010; Mann, 2007; Meyer et al., 2000; Salmeron et al, 1997a; 1997b; Schulze et al., 2004; Silvera et al., 2005a; 2005b; Stevens et al., 2002). In order to link GI with disease, dietary GI is assessed most commonly with food frequency questionnaires (FFQs) by which published GI values of foods are assigned. The food GIs are inserted into the summation model, thereby estimating total dietary GI (Augustin et al., 2003; 2004a; 2004b; Flood et al., 2006; Grau et al., 2010; Schulz et al., 2005; van Bakel et al., 2009). A similar method is used when there are multiple food items per FFQ line. A GI value is assigned to each food in the line and an estimated GI of the line is obtained from the weighted average GI values (Schulz et al., 2005). The weighted GIs were approximated from the prevalence of estimated population consumption of these foods. In addition, some authors have intentionally not included important carbohydrate sources such as some vegetables into overall dietary GI analysis (Grau et al., 2010). This approach would tend to alter the dietary GI estimation and obscure the subsequent results linking GI with disease. Another issue is that food composition data, which is used when testing GI or to obtain a dietary nutrient profile, can be inaccurate as databases do not include a full range of foods and brands, types or varieties may differ depending on country, region or season (Athar et al., 2006; FSA 2002). The accuracy of dietary assessment of GI is reduced when using approximate methods such as those mentioned.
Several other factors reduce the accuracy of dietary GI assessment. The original purpose of most FFQs that have been used in epidemiological research was not to assess dietary GI. Currently, there are a few FFQs that have been adapted from existing FFQs and validated against diet records to specifically assess dietary GI (Barclay et al., 2008b; Barrett & Gibson, 2010). These validation studies were conducted in samples similar to, or were sub-samples, of the population that were evaluated in the subsequent epidemiological studies. It has been suggested, “correlation values of about 0.5 for most nutrients is good evidence that the FFQ has the ability to rank individuals according the nutrient intake” (Brunner et al., 2001).

Dietary GI estimated from a 145-item FFQ correlated moderately well to that from four-day diet records in one study ($r^2 = 0.53$) (Barclay et al., 2008b). In another study, average GI obtained from a 297-item FFQ related reasonably well to that from four one-week diet records ($r^2 = 0.637$) (Barrett & Gibson, 2010). These study demonstrate that in their samples the FFQ correlated well the diet records. This does not mean that the FFQ is suitable to assess dietary GI, as it has not yet been determined if the assessment of a composite GI is valid. Another issue is that not all foods can be included in a FFQ, and the contribution of excluded foods to the total dietary GI is voided. A FFQ has been designed to include foods that contribute most to dietary GI (95% of carbohydrate intake in study location) (van Bakel et al., 2009). Although some progress has been made in designing a more robust approach to the dietary assessment of GI, the FFQs are still a major limitation of studies that have aimed to estimate dietary GI.

The GI values used in dietary assessment are often taken from published values, which have been compiled over time and are from different laboratories that can have rather different testing protocols for measuring GI. Often the GI values are derived from foods of different origin, brand or type, and are tested in small samples that differ from the study population in which dietary GI is being assessed. Many of the published GI values are obtained in small samples ($n=6-10$) and the large variation related to this further decreases the reliability of dietary assessment. There is a lack of standardised GI values that are applicable and relevant for study populations. The difficulty in obtaining an accurate and comparable dietary GI is partly due to the fact there are numerous items for the same food presented and there is large variation in GI values for the same food, thus it is the researchers choice as to which items from the tables are suitable. In a study examining insulin resistance and atherosclerosis, a review of eligible studies that tested the GI of foods was conducted and the mean GI from all eligible studies was assigned to the food (Schulz et al., 2005). For example bread, which is
considered a universal food, has almost 200 items, many of which are experimental products and designed to be ‘low-GI’ (Atkinson et al., 2008). The GI values of boiled white rice, another commonly consumed and standard food, range from 43-89. Published GI values are often based on small samples meaning they are imprecise, they may be inaccurate and predicted dietary GI can be viewed as indicative of a true dietary GI value (Williams et al., 2008)

More recently, dietary GI data have been added to databases and a dietary GI obtained using more robust approaches of dietary assessment such as diet records and careful assignment of GI values to foods (Aston et al., 2010; Flood et al., 2006; Martin et al., 2008; Neuhouser et al., 2006; Olendzki et al., 2006; Schakel et al., 2008; Similä et al., 2009). For example, GI values that the investigators have measured themselves have the highest level of confidence, while foods with no published GI value have the least level of confidence (Aston et al., 2010; Similä et al., 2009). The Diogenes Project assessed the diet of study participants using diet records and assigned measured GI values they had obtained themselves where possible (Aston et al., 2010). Likewise, an intervention study that assessed dietary GI and weight loss outcomes actually measured the GI of the some of the ‘key’ foods used in the intervention (Venn et al. 2010). Although not all foods were measured, this approach combined with the compilation of more reliable GI databases may help in providing a more accurate dietary GI.

2.3 Application of the Glycaemic Index to Mixed Meals

The summation model was originally designed to predict a composite meal GI and it has also been used to rank glycaemic responses to mixed meals based on their major carbohydrate source. This model has not been thoroughly tested and there are inconsistencies within the literature. For the GI to have clinical utility, the concept should remain robust in the testing of mixed meals containing carbohydrate, fat and protein.

2.3.1 Predicting Meal Glycaemic Index

Summation Model Based on Measured Response of Components and Meals

Two studies to date have tested the individual GI of the meal components as well as the GI of the meal (Jenkins et al., 1984b; Wolever et al., 1985). In both studies, the GI of beans alone and in a meal with bread was tested, with the beans and bread each contributing half of the available carbohydrate to the meal which contained a total of 50g starch carbohydrate.
In six type I diabetics, the observed GI (mean±SEM) of a meal containing bread and white pea beans was 77±7 which differed to the predicted meal GI of 92 (Table 2.3) (Jenkins et al., 1984b). Similarly, in seven type II diabetics, an observed meal GI of 60±8 differed to the predicted GI of 70 (Jenkins et al., 1984b). In another study a comparable meal of navy beans and bread was well predicted from the observed individual GIs. In six healthy participants, there was only one GI unit difference between predicted and observed meal GI (78 cf 77±11 respectively).

**Table 2.3 Predicted and observed mean±SD GI for meals containing bread and beans.**

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Food</th>
<th>Predicted GI</th>
<th>Observed GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jenkins et al., 1984b</td>
<td>6 T1DM</td>
<td>Beans 84±10 Bread 100</td>
<td>92</td>
<td>77±7</td>
</tr>
<tr>
<td></td>
<td>7 T2DM</td>
<td>Beans 41±5 Bread 100</td>
<td>70</td>
<td>60±8</td>
</tr>
<tr>
<td>Wolever et al., 1985</td>
<td>6 healthy</td>
<td>Beans 56±16 Bread 100</td>
<td>78</td>
<td>77±11</td>
</tr>
</tbody>
</table>

T1DM= type I diabetes mellitus; T2DM= type II diabetes mellitus

The discrepancies of the results between these two studies could be explained by the different participants (i.e. diabetic vs healthy) tested, however as each participant acts as their own control with GI testing, no differences in GI should be seen. It has also been shown the GI values obtained in people with and without type II diabetes correspond well (Crapo et al., 1981; Mehalski 1997; Wolever et al., 1987). Potentially as a result of the three different sample types that were tested, the GI of the beans was 84, 41 and 56. The effect of fat and protein on the glycaemic response does not offer an explanation for the different results. This is because the GI of the bread and beans were tested individually and thus the effect of these nutrients on GI is accounted for. The more likely reason for such a range in results is the small sample sizes used which inherently introduces variability.

There are several of limitations to these findings:

- Small samples were used (Table 2.3), reducing the power of the study and introducing variability as explained in Section 2.2.2.
- The tested meal contained the reference food. White bread was used as the reference food and as part of the meal, so was assigned a value of 100 in the prediction of meal GI. By assigning white bread a value of 100 within the meal, the variability of the GI for the bread is not accounted for, decreasing the variability of the meal GI. When predicting meal GI, larger standard deviations and 95% CI are produced if the GI of one meal
component is set at 100 compared to if measured GI values are used (BJ Venn, unpublished data).

- The carbohydrate contents of the foods were not tested. This is an important step in determining the GI of meals, as food composition can differ substantially depending on where and when the nutrient analysis was completed.

**Summation Model Based on Published Values for Foods and Measured Meal GI**

Correlation coefficients or ‘r values’ are often used as a measure of association between two variables, and in the cases below this is between predicted and measured GI. It is important for the correlation line to intersect the origin, or ‘zero’, for a meaningful association to be present (i.e. x-axis (number) correlates linearly with the y-axis (number)).

A significant correlation was observed between predicted GI using published values (Jenkins et al., 1981) and observed GI of six ethnic meals when tested in eight healthy participants ($r^2=0.88; p<0.01$) (Chew et al., 1988). The meals consisted of a main carbohydrate source (e.g. rice, bread, spaghetti) mixed with other foods such as vegetables, meat and pulses to obtain similar amounts of carbohydrate, protein and fat in all meals. Although an overall significant association was reported, the line did not intersect the origin and several predicted and observed GI results varied substantially (Table 2.4). Although the observed and predicted GI of four out of six meals was within 10 GI units, the other meals differed considerably. One example is a simple meal of bread and chickpeas. The mean predicted meal GI was 69 whereas the observed mean±SEM meal GI was 86±12. This range of values essentially classifies this meal as either moderate GI (predicted value), or high GI (observed value), which holds true for most of the meals as the summation model generally underestimated observed outcome.

*Table 2.4 Predicted and observed mean GI for six ethnic mixed meals.*

<table>
<thead>
<tr>
<th>Main sources of carbohydrate</th>
<th>Predicted GI</th>
<th>Observed GI (mean±SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bread, lentils</td>
<td>38</td>
<td>40±5</td>
</tr>
<tr>
<td>Spaghetti</td>
<td>40</td>
<td>52±9</td>
</tr>
<tr>
<td>Rice, lentils</td>
<td>60</td>
<td>60±10</td>
</tr>
<tr>
<td>Rice</td>
<td>65</td>
<td>73±17</td>
</tr>
<tr>
<td>Potato</td>
<td>69</td>
<td>66±12</td>
</tr>
<tr>
<td>Bread, chickpeas</td>
<td>69</td>
<td>86±12</td>
</tr>
</tbody>
</table>
Using published GI values obtained from Foster-Powell and Brand-Miller (1995) no association between predicted GI and observed GI in a range of commonly consumed European breakfast meals was found ($r^2=0.002; p=0.88$) (Flint et al., 2004). This is illustrated by an example of a meal containing oats, sugar and milk where the predicted GI was 74, but a mean GI of 57 was observed. Overall, a much larger range in measured GI values was seen (26-116) compared to the range for predicted meal GI (55-100) (Table 2.5). This study had several strengths. A pool of 28 participants was used, and due to the randomised, crossover design of this study, each meal was tested in 18-21 people. This number is two to three times larger than any previous study conducted on this topic. In addition, data were transformed, which is recommended when data is not normally distributed (Williams et al., 2008).

**Table 2.5 Predicted and observed mean GI values for composite breakfast meals.**

<table>
<thead>
<tr>
<th>Meal</th>
<th>Predicted GI</th>
<th>Observed GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finnish bread, butter, cheese</td>
<td>91</td>
<td>26</td>
</tr>
<tr>
<td>German bread, butter, cheese</td>
<td>91</td>
<td>27</td>
</tr>
<tr>
<td>Reference bread, butter cheese</td>
<td>99</td>
<td>30</td>
</tr>
<tr>
<td>Italian biscuits, coffee, milk</td>
<td>74</td>
<td>45</td>
</tr>
<tr>
<td>Reference bread, butter</td>
<td>100</td>
<td>49</td>
</tr>
<tr>
<td>All-bran plus, milk</td>
<td>55</td>
<td>51</td>
</tr>
<tr>
<td>Reference bread, butter, jam</td>
<td>96</td>
<td>56</td>
</tr>
<tr>
<td>Rolled oats, sugar, milk</td>
<td>74</td>
<td>57</td>
</tr>
<tr>
<td>Frosties, milk</td>
<td>77</td>
<td>63</td>
</tr>
<tr>
<td>All-bran, milk</td>
<td>86</td>
<td>65</td>
</tr>
<tr>
<td>French bread, butter, jam</td>
<td>94</td>
<td>71</td>
</tr>
<tr>
<td>Cornflakes, milk</td>
<td>97</td>
<td>81</td>
</tr>
<tr>
<td>Porridge, apple sauce, water</td>
<td>76</td>
<td>116</td>
</tr>
</tbody>
</table>

Energy (kJ), protein (g) and fat (g) were inversely related to meal GI ($r^2=0.93$, $r^2=0.65$, $r^2=0.88$, respectively; $p<0.001$) and carbohydrate content (%energy) was positively associated with meal GI ($r^2=0.80$; $p<0.001$). This effect on GI was present even with smaller additions of fat and protein to the meals and the authors attributed the lack of association between predicted and observed meal GIs to the metabolic effect of fat and protein in the meal. These nutrients reduce the glycaemic response to meals by increasing insulin production and glucose
disposal, thereby lowering postprandial blood glucose concentration. These mechanisms result in a lower blood glucose response curve and GI (refer Section 2.2).

There are many limitations associated with using published values to predict a composite GI. The disparity in the results of these two studies may be partially explained by the choice of published GI values. In addition, the study by Chew et al. (1988) was conducted at a time when GI was a relatively new concept and limited published values were available. This resulted in the use of values that would be considered less suitable by more recent standards. For example, there would have been a more limited choice of foods and a more restricted choice of country of test, which is probably why some of the published GI values were from foods tested in Canada. In comparison to this, the more recent study by Flint et al. (2004) used values that were more representative of the test foods such as Finnish and German breads and Italian biscuits. Moreover Flint et al. (2004) disclosed the item number/s of the foods used in their study, which is a practice that is not commonly implemented.

Another limitation of these studies is the available carbohydrate was estimated from published nutrient data. Chew et al., (1988) tested the GI of their foods in Australia, but the nutritional information was obtained from English food composition tables (Paul & Southgate, 1978). This method is likely to have introduced error into the calculation of the 50g available carbohydrate portion. Flint et al. (2004) also used published nutritional information but this data was sourced from Danish food composition tables (Møller & Saxholt 1996) or the suppliers, which is more suitable as the foods were of Scandinavian and European origin. When relying on published values for the carbohydrate content of the foods, one cannot be certain the data is representative of the test food and whether the correct amount of food will be tested in order to provide the required available carbohydrate portion. Hence, there may be uncertainty as to whether the true glycaemic response for that food has been observed.

Conclusion
The capacity of the summation model to predict the GI of meals is limited. The findings regarding this topic thus far are inconsistent, with some avidly supporting the use of the summation model while others do not. The model does not account for the effects of fat and protein on glycaemic response and a different GI is often observed to that predicted. Methodological issues such as small samples (n~6) and not testing the GI or available carbohydrate content of the foods limit the current findings.
### Table 2.6 Studies testing predictability and rank of glycaemic response.

<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bornet et al., 1987</td>
<td>3 T2DM</td>
<td>GI ranking remains, in foods alone and in meals.</td>
</tr>
<tr>
<td>Collier et al., 1986</td>
<td>6 T2DM</td>
<td>Glycaemic responses to mixed meals were predicted from the GI of the individual meal components ($r^2=0.9875$, $p&lt;0.01$).</td>
</tr>
<tr>
<td>Coulston et al., 1980</td>
<td>22 Healthy</td>
<td>No significant differences in glycaemic response to two meals containing different carbohydrate sources.</td>
</tr>
<tr>
<td>Coulston et al., 1984</td>
<td>8 T2DM</td>
<td>Differences in GI between foods are lost when applied to a mixed meal.</td>
</tr>
<tr>
<td>Coulston et al., 1987</td>
<td>6 Healthy</td>
<td>The glycaemic response to mixed meals was not predicted by varying the carbohydrate source and published GI values.</td>
</tr>
<tr>
<td>Henry et al., 2006</td>
<td>10 Healthy</td>
<td>Lower GI with addition of toppings to potato, pasta and bread.</td>
</tr>
<tr>
<td>Hollenbeck et al., 1988</td>
<td>9 T2DM</td>
<td>Plasma glucose concentrations were almost identical after the ingestion of meals differing in their GI.</td>
</tr>
<tr>
<td>Parillo et al., 1985</td>
<td>7 T2DM</td>
<td>AUC of spaghetti meal significantly different to both potato and bread ($p&lt;0.05$).</td>
</tr>
<tr>
<td>Wolever &amp; Bolognesi, 1996</td>
<td>8 Healthy</td>
<td>Predicted meal GI did not correctly rank glycaemic response. Amount and source of CHO positively correlated with glycaemic response ($r^2=0.929$, $p=0.022$).</td>
</tr>
<tr>
<td>Wolever &amp; Jenkins, 1986</td>
<td>8 T2DM</td>
<td>GI applies well to mixed meals ($r^2=0.987$, $p&lt;0.02$).</td>
</tr>
<tr>
<td>Wolever et al., 1990</td>
<td>3 T1DM</td>
<td>Overall mean±SD glycaemic responses to mixed meals were significantly different from each other ($p&lt;0.01$). Proportion of correct ranking of meal GI on glycaemic response was 71%.</td>
</tr>
<tr>
<td>Wolever et al., 2006</td>
<td>26 Healthy</td>
<td>Carbohydrate content ($p=0.002$) and GI ($p=0.022$) were related to the glycaemic responses.</td>
</tr>
</tbody>
</table>

AUC = area under the blood glucose response curve; T1DM = type I diabetes mellitus; T2DM = type II diabetes mellitus.
2.3.2 Effect of Glycaemic Index of Major Carbohydrate Source on Glycaemic Response

Low glycaemic foods are often recommended to diabetics to maintain normoglycaemia and help with glycaemic control. As very few foods are eaten alone, it is important the glycaemic responses to carbohydrate containing foods are predictable when consumed with other foods. This is particularly important if a purpose of the GI is to facilitate food choice. The GI and glycaemic response to carbohydrate foods can generally be ranked; for example, potato usually elicits a greater response than rice or pasta. This ranking often remains when consumed with other foods, but occasionally is altered or there are no differences between foods. This is likely due to the effect on the glycaemic response of the addition of nutrients such as fat and protein. An overview of studies focussing on predicting glycaemic response and ranking of GI or glycaemic responses of meals is presented in Table 2.6.

Predicting Glycaemic Responses to Mixed Meals

Early work in this area produced some conflicting results. When predicting the glycaemic response to mixed meals published GI values from earlier work were often used instead of testing the GI of the individual foods (Jenkins et al., 1983). Another issue is that total rather than incremental area under the glucose curve was used in some early studies. Total area under the curve is “considered to be a less sensitive measure of differences in post-prandial blood glucose responses to different meals” (Wolever, 1990). The use of this method may be the reason why no difference in mean glycaemic response was seen when healthy participants consumed either a low GI meal containing corn and rice, or a high GI meal containing potato and gelatine (Coulston et al., 1980).

Similarly, in type II diabetics there was no difference between the GI of potato, rice, spaghetti or lentils when consumed alone or in a meal containing similar amounts of fat, protein and energy (Coulston et al., 1984). When this same data was reanalysed using IAUC, the GI values of the meals were significantly correlated with the IAUC ($r^2=0.987$, $p<0.02$) (Wolever & Jenkins, 1986). The authors conclude the GI concept applies well to mixed meals containing fat and protein and the addition of these nutrients to a meal does not obscure the glycaemic effect of carbohydrate foods in the meal. This conclusion is contrary to many other findings (Bornet et al., 1987; Flint et al., 2004; Gulliford et al., 1989; Henry et al., 2006; Normand et al., 2001, Simpson et al., 1985).

Following on from their previous work, Coulston and colleagues tested three carbohydrate rich meals with varying glycaemic potential in healthy and diabetic participants (Coulston et
al., 1987). These meals were designed to be either high GI with carbohydrate contributions from white rice, banana and carrots, medium GI with spaghetti, orange and canned beetroot, and low GI with lentils, cooked apple. Predicted meal GI based on published values differed substantially (high=71, medium=48, low=34), however no significantly different glycaemic responses were seen between meals when tested directly. This was also found in the assessment of daylong plasma glucose concentrations. Type II diabetic participants consumed either a low, moderate or high GI breakfast, lunch and dinner on three separate days, with no significant differences in plasma glucose after the meals or daylong levels (Hollenbeck et al., 1988). Furthermore, the responses to breakfast and dinner were “essentially identical” regardless of the GI of the meal. In contrast to these findings, an Italian study on seven type II diabetics resulted in glucose responses to a potato meal and a bread meal that were significantly higher than a spaghetti meal (Parillo et al., 1985). All three meals contained the same ingredients with the exception of the main carbohydrate source. This may explain the differences in results in this study compared to the work by Coulston et al. (1980; 1984; 1987) who had a range of carbohydrate foods in their meals. That approach is thought to introduce more varied effects on the glycaemic response, as a lesser number of variables such as foods and nutrients are controlled for.

The amount of carbohydrate as well as the source predicted glycaemic responses of five meals that varied in their energy, protein and fat content (Wolever & Bolognesi, 1996). Predicted glycaemic response correlated well with the measured glycaemic response ($r^2=0.929$ $p=0.022$) however the predicted meal GI did not correctly rank the glycaemic responses of the meals. The glycaemic responses to two of the meals, based on an omelette and barley, were significantly lower than those consisting of spaghetti and oatmeal and these four meals were significantly lower than cornflakes. This rank differed to the predicted rank for lowest to highest GI and glycaemic response, which was barley, spaghetti, omelette, oatmeal and cornflakes. The omelette resulted in the lowest response although it was predicted to give a relatively large response. This outcome is probably because of the omission of eggs from the model as eggs contain very little carbohydrate. The other carbohydrate containing foods in the meal, such as bread, therefore determined the predicted meal GI; thus highlighting a limitation of the model.

It has been suggested that the proportional difference between predicted and measured glycaemic responses of meals differing in GI is unpredictable (Gulliford et al, 1989; Henry et al., 2006). This was demonstrated in a study whereby the glycaemic responses to potato, toast
and pasta consumed alone or with different toppings were assessed in 10 healthy participants (Henry et al., 2006). The addition of toppings resulted in a lowering effect on the GI of the main carbohydrate source, which is likely to be due to the fat and protein addition from the toppings. For example, the observed mean±SEM GI for potato alone was high at 93±8, but when consumed with 120g of cheddar cheese the GI was low with a value of 39±5. The proportional differences between the meals varied, with the glycaemic response to potato and toast consumed alone or with the cheddar cheese topping differing by 58% and 43% respectively. In addition, the glycaemic response to potato and pasta alone compared to when consumed with 120g of tuna differed by 18% and 54% respectively. It is established that the addition of fat and protein to a carbohydrate source alters the glycaemic response (refer to Section 2.2), but it appears that the relative difference is variable.

Predictability of the GI of meals was assessed from a pool of data on glycaemic responses to six composite breakfast meals with varying carbohydrate contents in sixteen participants in Australia, and eight meals in ten participants in Canada (Wolever et al., 2006). Meal carbohydrate content and predicted meal GI was related to glycaemic responses ($r^2$=0.568, p=0.0018 and $r^2$=0.367, p=0.022 respectively). Contrary to previous findings, fat and protein were not related to glycaemic response ($r^2$=0.068, p=0.37 and $r^2$=0.199, p=0.11 respectively). The authors conclude “when properly applied in realistic settings, the GI is a significant determinant of the glycaemic effect of mixed meals in normal subjects”. However, some of the results do not support this conclusion. Two meals that had similar carbohydrate contents, one consisting of a bagel with cream cheese and orange juice, and the other rye bread, margarine, cereal, milk, sugar and orange juice had very similar glucose responses measured as AUC (148 ± 14 and 143 ± 13 mmol/Lmin$^{-1}$ respectively), but the predicted GIs using published values differed (67 and 51 respectively).

**Ranking Glycaemic Index and Response**

When main carbohydrate sources such as potato, bread, pasta and rice are consumed alone a ‘rank’ of the glycaemic responses to these foods can be produced and is usually maintained. For example mashed potato repeatedly produces higher responses than spaghetti pasta. The rank of foods has also shown to be maintained when consumed as part of a mixed meal. Six type II diabetic participants tested six meals that differed only in their carbohydrate source (potato, white bread, rice, spaghetti, barley, lentils) (Collier et al, 1986). The mean glycaemic responses to the meals could be correctly ranked from the GI of the individual meal components ($r^2$=0.9875, p<0.01). In contrast to these findings the ranking of GI was not
strictly maintained when six foods were tested alone and in a mixed meal in three type II diabetic participants (Bornet et al., 1987). The glycaemic response to the foods when consumed alone could be ranked as white bread, instant potato flakes, spaghetti, white rice, lentils, beans, whereas when combined in a meal with butter and cheese the rice was ranked higher than the spaghetti. A consistent lowering effect on glycaemic response to the mixed meals was seen compared to the single food meal. Overall differences between the foods alone and within the mixed meal were not statistically significant, however the reliability of these results are questionable due to the high variability introduced by the small sample size.

Work by Wolever’s group has shown that under test conditions the GI ranking of carbohydrate foods is preserved within a meal setting (Wolever & Jenkins, 1986; Wolever et al., 1990). Twelve type II diabetic participants consumed three mixed meals with a fixed 50g carbohydrate from bread, rice or spaghetti (Wolever et al., 1990). The mean glycaemic response areas could be ranked bread>rice>spaghetti from the predicted GI of the meals and the mean glycaemic responses were significantly different from each other when presented as IAUC (p<0.01) or GI (p<0.01). These findings differ to that of a previous study by Wolever and Bolognesi (1996) whereby predicted meal GI did not correctly rank the glycaemic responses of five meals, highlighting the inconsistent findings within this body of literature.

Conclusion
Despite 20 years of research in this area, there is no consensus on whether the GI differences among foods are preserved when combined into a meal or if the rank of the GIs for foods is maintained in a meal. It is important that GI studies are performed in the context of mixed meals, as very few foods are eaten alone. In addition, if the lowering effect of fat and protein is more substantial for high GI foods, food choices may be broadened to include these foods with the condition they are consumed within a mixed meal.
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Participants n</th>
<th>Characteristics</th>
<th>Intervention duration</th>
<th>Methods</th>
<th>GI reduction (units)</th>
<th>Outcomes reported</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alfenas and Mattes (2005)</td>
<td>Parallel</td>
<td>39</td>
<td>Healthy</td>
<td>8 days</td>
<td>Only key high or low foods were consumed in laboratory.</td>
<td>Low 30-50; High 73-169</td>
<td>No change in any outcomes (plasma glucose or insulin responses).</td>
</tr>
<tr>
<td>De Natale et al. (2009)</td>
<td>Crossover</td>
<td>18</td>
<td>T2DM</td>
<td>4 weeks</td>
<td>High CHO/LGI diet or low CHO high MUFA diet.</td>
<td>27</td>
<td>Decrease in PPG, insulin responses, and glycaemic variability in high CHO/LGI GROUP.</td>
</tr>
<tr>
<td>Gutschall et al. (2008)</td>
<td>Pre-post</td>
<td>109</td>
<td>T2DM</td>
<td>9 weeks</td>
<td>Group based behavioural intervention with an immediate or delayed start.</td>
<td>-2.1 immediate group; +1.7 delayed group</td>
<td>Improvements to diet, anthropometric and biochemical were seen in immediate group (p&lt;0.05)</td>
</tr>
<tr>
<td>Jarvi et al. (1999)</td>
<td>Crossover</td>
<td>20</td>
<td>T2DM</td>
<td>24 days</td>
<td>A controlled HGI or LGI diet.</td>
<td>25.9</td>
<td>Fasting plasma glucose decreased from baseline for both diets; HbA1c decreased after LGI diet; insulin sensitivity increased after both diets.</td>
</tr>
<tr>
<td>Jenkins et al. (2008)</td>
<td>Parallel</td>
<td>210</td>
<td>T2DM</td>
<td>6 months</td>
<td>High cereal fibre or LGI dietary advice.</td>
<td>13.9</td>
<td>HbA1c decreased -0.50% and HDL increased 0.7mg/dl in LGI group.</td>
</tr>
<tr>
<td>Jimenez-Cruz et al. (2003)</td>
<td>Crossover</td>
<td>14</td>
<td>T2DM</td>
<td>6 weeks</td>
<td>HGI or LGI diet.</td>
<td>12</td>
<td>HbA1c improved 0.4% on LGI diet; no change on HGI diet.</td>
</tr>
<tr>
<td>Riskalla et al. (2004)</td>
<td>Crossover</td>
<td>12</td>
<td>Men with T2DM</td>
<td>4 weeks</td>
<td>Either a HGI or LGI diet.</td>
<td>32.3</td>
<td>Improved IAUC, postprandial plasma glucose, insulin profiles, fasting plasma glucose and HbA1c after the LGI diet compared to HGI diet.</td>
</tr>
<tr>
<td>Shikany et al. (2006)</td>
<td>Crossover</td>
<td>24</td>
<td>Overweight men</td>
<td>4 weeks</td>
<td>HGI/GL or LGI/GL diet.</td>
<td>25.5</td>
<td>No significant differences in glucose metabolism, inflammatory or coagulation markers.</td>
</tr>
<tr>
<td>Solomon et al. (2009)</td>
<td>Parallel</td>
<td>32</td>
<td>Obese</td>
<td>7 days</td>
<td>Controlled HGI or LGI diet combined with aerobic exercise</td>
<td>39.8</td>
<td>Fasting glucose, insulin, TAG, cholesterol decreased in both groups.</td>
</tr>
<tr>
<td>Wolever et al. (2008)</td>
<td>Parallel</td>
<td>156</td>
<td>T2DM</td>
<td>1 year</td>
<td>High CHO/HGI, high CHO/LGI or low CHO/ high MUFA diet.</td>
<td>7</td>
<td>No change in HbA1c.</td>
</tr>
</tbody>
</table>

CHO=carbohydrate; HGI=high GI; LGI=low GI; PPG=post-prandial glycaemia; TAG=triacylglycerides; T1DM=type I diabetes mellitus; T2DM=type II diabetes mellitus
2.4 The Glycaemic Index In Human Health and Disease

2.4.1 Intervention Studies Involving the Glycaemic Index

One of the major criticisms of the GI concept is that it lacks clinical application in those with chronic diseases such as diabetes and cardiovascular disease (Alfenas & Mattes 2005; Coulston et al., 1984a; 1987, Coulston & Reaven 1997; Franz et al., 2002; Laine et al., 1987; Shikany et al., 2009). In contrast to this view, significant improvements in disease markers have been seen in those following lower GI diets in intervention studies (De Natale et al., 2009; Jarvi et al., 1999; Jenkins et al., 2008; Larsen et al., 2010; Rizkalla et al., 2004; Wolever et al., 2008b) and many reviews are also in favour of a low GI diet, (Jenkins et al., 2002; Esfahani et al., 2009; Mann, 2004; 2006; 2007; Riccardi et al., 2008; Sheard et al., 2004; Thomas & Elliot 2009).

As with all studies concerning dietary GI, the major flaw in intervention studies is the form of dietary assessment to estimate dietary GI. Various methods for dietary GI determination have been used and there is no established protocol making comparisons among studies difficult (Esfahani et al., 2009). Many intervention studies assess the diet at intervals via diet records, and overall dietary GI obtained using published values. While researchers strive to maintain the same proportions of macronutrients in the study diets, it is not always achieved. Although some of the differences in nutrient composition of the diets are controlled for, the varying amounts of the nutrients may be partly responsible for the affect on the measured outcomes. For example, low GI diets are commonly associated with an increase in the consumption of high fibre foods and decrease in sugar intake. It may be the effect of changes such as these, combined with a shift towards a healthier diet that often accompanies a low GI diet, which affects the study outcomes.

A major practical issue is the small difference in dietary GI units (<10 units) between the high and low GI diets that is commonly seen, as a major change in dietary GI is difficult to achieve. Perhaps unexpectedly, this can still lead to significant differences in disease biomarkers between high and low GI groups (Gutschall et al., 2009; Jimenez-Cruz et al., 2003; Larsen et al., 2010; Miller & Gutschall 2009; Wolever et al., 2008b). Further variation in the literature is introduced through small samples (6-30) and differing forms of study design. While many studies are conducted in free-living population, some are conducted in the laboratory, in hospitals and some treatment diets are combined with exercise. It is difficult to review and interpret this body of literature when such inconsistencies and variations are present. The
differences in methodology and findings for the key intervention studies are presented in Table 2.7.

**Diabetes**

Intervention studies often focus on the effect of the intervention on disease markers, and in diabetes research the most commonly assessed marker for related complications is HbA1C. Significant improvements in HbA1C were seen in several short-term studies with relatively large sample sizes and substantial changes in dietary GI (Alfenas & Mattes 2005; De Natale et al., 2009; Jarvi et al., 1999; Jimenez-Cruz et al., 2003; Rizkalla et al., 2004; Shikany et al., 2009). The dietary GI in these studies was substantially altered, which is likely due to the short-term duration and the ability of participants to tolerate large changes to their diet for a short period of time. For example, in several trials with an intervention period of 4 weeks the difference in dietary GI between the high and low GI study groups ranged from 26-32 GI units.

These findings contrast to a recent long-term study, in which a large change in dietary GI was more difficult to obtain (Wolever et al., 2008b). ‘Key foods’ for a low or high GI diet were given to participants with a seven dietary GI unit difference seen between the two diet groups over a one-year intervention period. No significant effect on HbA1C levels was found (Wolever et al., 2008b). However a long-term trial over six months achieved a dietary GI change of 14 units through dietary advice and low or high GI food checklists. This change in dietary GI resulted in a significant -0.3% difference in HbA1C levels in the low compared to high GI diet group (Jenkins et al., 2008). The findings from this study show that improvements in disease markers can be gained from dietary GI changes.

**Cardiovascular Disease**

A meta-analysis on the effects of low GI compared to high GI diets on markers for lipid metabolism was conducted with various outcomes for different markers (Opperman et al., 2004). Criteria for selection were adapted from the ‘Effective Practice and Organisation of Care Cochrane Group’ and also specified that feeding periods were longer than fourteen days, food intake had to have been controlled and described, the GI of the diet was indicated, and subject population was homogenous. Changes in the GI of study diets ranged from 5-35 GI units. An average difference of 22 dietary GI units resulted in a significant mean change in total cholesterol concentration of -0.33mmol/L. No significant change in mean HDL cholesterol (-0.03mmol/L) or triacylglyceride concentrations (0.03mmol/L) was found.
<table>
<thead>
<tr>
<th>Study</th>
<th>Number of cases/total cohort</th>
<th>Length of follow-up (year)</th>
<th>GI and disease risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nurses Health Study (1997)</strong></td>
<td>915/65173</td>
<td>6</td>
<td>1.37 (1.09, 1.71)</td>
</tr>
<tr>
<td><strong>Health Professionals Study (1997)</strong></td>
<td>523/42759</td>
<td>6</td>
<td>1.37 (1.02, 1.83)</td>
</tr>
<tr>
<td><strong>Iowa Women Study (2000)</strong></td>
<td>1141/35998</td>
<td>6</td>
<td>0.89 (0.72, 1.10)</td>
</tr>
<tr>
<td><strong>ARIC Study (2002)</strong></td>
<td>1447/12251</td>
<td>9</td>
<td>1.00 (0.99, 1.02)</td>
</tr>
<tr>
<td><strong>Nurses Health Study II (2004)</strong></td>
<td>741/91249</td>
<td>8</td>
<td>1.46 (1.12, 1.92)</td>
</tr>
<tr>
<td><strong>Melbourne Collaborative Cohort Study (2004)</strong></td>
<td>365/31641</td>
<td>4</td>
<td>1.23 (0.98-1.54) (OR/10GI units)</td>
</tr>
<tr>
<td><strong>Whitehall II study (2007)</strong></td>
<td>329/7321</td>
<td>13</td>
<td>0.94 (0.72, 1.22) (HR)</td>
</tr>
<tr>
<td><strong>Health, Aging and Body Composition Study (2008)</strong></td>
<td>99/1898</td>
<td>4</td>
<td>1.0 (0.5, 2.0)</td>
</tr>
<tr>
<td><strong>ATBC Study (2010)</strong></td>
<td>1098/25,943</td>
<td>12</td>
<td>0.87 (0.71, 1.07)</td>
</tr>
</tbody>
</table>

HR = Hazard Ratio; OR = Odds Ratio

* Fully adjusted model
+ The RR is the risk between the lowest \( \text{RR}=1.00 \) and highest quintile of intake for GI
However, in seven out of ten studies an improvement in LDL cholesterol concentrations was observed (-0.15 mmol/L) in those following a low GI diet. Although this finding was not statistically significant, mean LDL cholesterol concentrations decreased to a greater extent in those who had heart disease than those who were healthy.

2.4.2 The Use of Glycaemic Index in Cohort Studies

The interest in the relationship between GI and disease has risen in recent years, which has led to a large body of literature with inconsistent findings (Barclay et al., 2008a; Feskens & Du, 2006; Mann, 2007). A summary of recent prospective studies focussing on dietary GI and the development of type II diabetes is presented in Table 2.8.

The Health Professionals and Nurses Health studies are similar in methodology and design (Salmeron et al., 1997; 1997b). These two large cohorts were each followed up over 6 years, and dietary GI was assessed by FFQs. In both groups, the highest quintile of GI intake was associated with a 37% increase in risk of developing type II diabetes. Similarly, a more recent study of the Nurses Health cohort found a positive correlation between GI and the risk of type II diabetes and reported a higher risk for disease (RR=1.46) (Schulze et al., 2004). The estimated GI level could be classified as high for almost all quintiles of intake, ranging from 65-79 (Health Professionals), 64-77 (Nurses Health) and 71-82 (Nurses Health II). These values are relatively high for an average dietary GI, and although no specified are therefore likely to be based on GI values obtained from a white bread reference.

In direct contrast to the above findings, the Iowa Women Study found a high dietary GI was associated with a significantly decreased risk of type II diabetes (Meyer et al. 2000). A larger range of median GI for each quintile was observed compared to other cohort studies, with a median GI of 53 for quintile 1, ranging to 89 for quintile 5. The ARIC Study (Stevens et al., 2002) examined both female and male participants, with no significant association seen between dietary GI and risk of type II diabetes. Likewise, data from the 12-year follow-up of the ATBC study found no significant association between GI and type II diabetes in men (Similä et al., 2010). Several other studies have also found no association between dietary GI and type II diabetes (Hodge et al., 2004; Mosdol et al., 2007; Sahyoun et al., 2008). A recent meta-analysis compared all relevant literature on GI and risk of type II diabetes, with an overall RR of 1.40 (95% CI 1.23, 1.59) (Barclay et al., 2008a). These contrasting results may be due to the various methods used to predict the GI of diets.
Most studies assessing dietary GI have a major shortcoming in that FFQs, which were not designed to measure the GI of the diet, were used to assess dietary data (issues with this form of assessment were described in Section 2.2). The FFQs are usually validated for energy, total carbohydrate or fibre intake, which is justified by “GI is a characteristic of the carbohydrate in different foods” (Barclay et al., 2008a). A correlation coefficient of 0.45 for a weighed food record and FFQ was obtained in the ARIC study, which is not considered as evidence of high enough quality to suggest the FFQ is able to rank individuals based on nutrient intake (Brunner et al., 2001).

There is currently no standardised method for authors to use when choosing published GI values in order to predict a dietary GI (Aston et al., 2010). The approach to assign GI values to foods consumed by the study populations is somewhat subjective and extremely variable. The Insulin Resistance Atherosclerosis Study (IRAS) along with many others used a FFQ to estimate dietary GI. However, this study assigned a GI value of 100 to sources that contained little carbohydrate such as meat, oils, fish and some vegetables (Schulz et al., 2005). An explanation for this approach is given as “[this method] will not greatly affect the estimated daily GI due to small amounts of carbohydrates consumed with these foods, but allows for the fact that whatever carbohydrate to be found in meats or fish is glycogen” (Schulz et al., 2005). A fair point, but the effects of the fat and protein content of these foods on glycaemia or the fact that vegetables will contribute other carbohydrate forms is not taken into account. The GI of mixed dishes was estimated from the main carbohydrate source, again not considering the nutritional contribution from other foods in the meal, or the effect of the consumption of a whole meal on post-prandial glycaemia. An example is that sushi was assigned a GI of plain rice, and spaghetti dishes were assigned a value of plain spaghetti. Furthermore, “the infrequent consumption of other mixed dishes in our study population was assumed to be of minor importance for the estimation of dietary GI” (Schulz et al., 2005), which is surprising considering the large body of evidence that shows the decrease in glycaemic response when mixed meals are consumed compared to the main carbohydrate source alone (Bornet et al., 1987; Flint et al., 2004; Gulliford et al., 1989; Henry et al., 2006; Normand et al., 2001; Sugiyama et al., 2003; Owen & Wolever 2003). With common methodological flaws such as those mentioned, as well as the controvertible use of the summation model to predict dietary GI, the associations between GI and disease may be less clear than previously thought.

Another major weakness in this area of research is that most of the studies that showed a positive association with GI and type II diabetes originated from one research group.
These studies all used very similar methodology and FFQs. For the relationship between GI and disease to be convincing one would expect similar results published from other groups of researchers, preferably from other countries. Accordingly, a WHO report states the strength of evidence that a low-GI diet is associated with diseases such as obesity and diabetes was classed as “possible” (FAO/WHO, 2002).

2.5 Summary

There are gaps in the current literature on GI- particularly where the summation model and predictability of the GI in mixed meals is concerned. Many of the downfalls of the GI concept with regard to mixed meals have arisen from small sample sizes, limited use of standardised GI testing protocols, large intra-individual variation and not testing the carbohydrate content or GIs of the individual foods. The summation model has been utilised by numerous studies to link GI with disease, but only two early studies have specifically tested the model, albeit somewhat inadequately and with varying results (Jenkins et al., 1984b; Wolever et al., 1985). Furthermore, if the GI is to have clinical utility, composite GIs obtained from published and measured values should be similar when inserted into the model. Robust testing of the model is essential if it is to be used in the clinical setting to guide food choice, in dietary GI prediction in epidemiological research and to help improve the methods of dietary GI assessment.
3 Methods

3.1 Ethics

The University of Otago Human Ethics Committee approved this study and granted permission to recruit participants from the general public. Relevant participant information outlining study requirements and risks was included in the application to the committee (Appendix 1-4). Potential participants were required to read the information sheet and were screened for eligibility using the participant questionnaire. Any concerns or questions were dealt with via e-mail, phone or on the morning of their tests. Informed written consent was obtained from the participants.

3.2 Participants

3.2.1 Sample Size Calculation

The variability of GI testing is known from previous work in the Department of Human Nutrition, University of Otago (Williams et al., 2008). Data from 30 people would have 80% power to detect a difference of 10 GI units using the 5% level of significance. The study was underpowered to detect a smaller difference but a difference of less than 10 GI units is of limited clinical significance. The sample size was also large enough to predict the GI of a meal from the GI of its components.

3.2.2 Recruitment

Participants were recruited from the public via flyers and e-mails to University of Otago staff and post-graduate students. Participants were required to meet the following criteria: male or female aged between 18-50 years, free from chronic disease, not taking any medications or nutritional supplements known to affect glucose metabolism, do not suffer from food allergies and women who were not pregnant.
Table 3.1 Nutrient content of the foods from laboratory analysis, company data (chicken only) and reference values.

<table>
<thead>
<tr>
<th>Food</th>
<th>Moisture %</th>
<th>Ash %</th>
<th>Protein %</th>
<th>Fat %</th>
<th>CHO %</th>
<th>Fibre (crude) %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Analysis</td>
<td>Reference Values</td>
<td>Analysis</td>
<td>Reference Values*</td>
<td>Analysis</td>
<td>Reference Values</td>
</tr>
<tr>
<td>Potato</td>
<td>82.2</td>
<td>77-78.8</td>
<td>0.9</td>
<td>-</td>
<td>1.5</td>
<td>1.6-2.1</td>
</tr>
<tr>
<td>Rice</td>
<td>56.9</td>
<td>76</td>
<td>0.1</td>
<td>-</td>
<td>3.1</td>
<td>3.1</td>
</tr>
<tr>
<td>Pasta</td>
<td>65.9</td>
<td>63</td>
<td>0.3</td>
<td>-</td>
<td>5.5</td>
<td>5.2</td>
</tr>
<tr>
<td>Kumara</td>
<td>73.7</td>
<td>78</td>
<td>0.5</td>
<td>-</td>
<td>1.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Peas</td>
<td>77.7</td>
<td>81</td>
<td>0.4</td>
<td>-</td>
<td>5.8</td>
<td>5.4</td>
</tr>
<tr>
<td>Carrots</td>
<td>91.2</td>
<td>90-91.1</td>
<td>0.3</td>
<td>-</td>
<td>0.7</td>
<td>0.6-0.9</td>
</tr>
<tr>
<td>Sauce</td>
<td>81.7</td>
<td>85</td>
<td>2.1</td>
<td>-</td>
<td>2</td>
<td>1.4</td>
</tr>
<tr>
<td>Chicken</td>
<td>-</td>
<td>62</td>
<td>-</td>
<td>-</td>
<td>19.8</td>
<td>31.2</td>
</tr>
</tbody>
</table>

* No reference values for ash content are available.
+ Reference value refers to total fibre.
3.3 Nutrient Analysis

Cooked foods (except chicken) were sent to an external laboratory (Rice: AsureQuality, Auckland, NZ; Potato, Pasta, Kumara, Peas, Carrots, Sauce: Gribbles Veterinary Pathology, Mosgiel, NZ) for basic nutritional analysis. The following methods were used to determine nutrient content:

- Moisture content of the food was determined by AOAC method 950.46 (AOAC, 2005).
- AOAC method 942.05 (modified to 4 hour ash time) (AOAC, 2005) was used to determine the ash content of the foods.
- The AOAC method 981.10 (AOAC, 2005) was used for protein concentration determination. This method, the Kjeldahl Method, determines the amount of nitrogen present in the food and multiplies this by 6.5 to obtain percent protein.
- Total fat was determined as described in Pearson's Chemical Analysis of Foods (Egan, Kirk & Sawyer 1987).
- Crude fibre was determined as described in Foss Analytical Application Note 380 (2005).
- The carbohydrate content was determined via the by difference method (AOAC, 2005). This method is briefly described in Section 2.2.

The approximate reference values were taken from the NZ Food Composition Tables (Athar et al., 2006). Results for both the present analysis and reference values are shown in Table 3.1. The nutrient content of the chicken breast (Rangitikei corn fed, free range, boneless chicken breast skin off, Tegal Foods Ltd, Auckland, NZ) was obtained directly from the company (Tegal Foods Ltd). These values are also shown in Table 3.1.

The amount of food required to obtain the correct available carbohydrate content was determined by the following equation:

\[
\text{Amount of food} = \frac{100}{x} \times y
\]

Where \(x\) is the amount of available carbohydrate in grams per 100g of the food and \(y\) is the amount of available carbohydrate that food is required to provide. For example, to determine the amount of pasta required to obtain 50g available carbohydrate, given that pasta contains 27.1g available carbohydrate per 100g (Table 3.1): \(100/27.1 \times 50 = 184.5\)g pasta. The amount of each food that was tested is shown in Table 3.2.
Total energy content (kJ) and nutrient contents in grams of protein, fat, carbohydrate and fibre for the meals are shown in Table 3.3. Total energy content was estimated using the energy conversion factors described in the Concise NZ Food Composition Tables (Athar et al., 2006). The energy conversion factor used for fat was 37.7kJ/g, protein 16.7kJ/g, carbohydrate 16.7kJ/g and fibre 8kJ/g.

*Table 3.2 Amount of food (g) required to obtain 25g or 50g available carbohydrate per test food or meal.*

<table>
<thead>
<tr>
<th>Food</th>
<th>25g Available CHO</th>
<th>50g Available CHO</th>
<th>Meal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amount (g) of food required</td>
<td>Amount (g) of food required</td>
<td></td>
</tr>
<tr>
<td>Potato</td>
<td>388g</td>
<td>25</td>
<td>194g</td>
</tr>
<tr>
<td>Rice</td>
<td>130g</td>
<td>25</td>
<td>65g</td>
</tr>
<tr>
<td>Pasta</td>
<td>184.5g</td>
<td>25</td>
<td>92g</td>
</tr>
<tr>
<td>Kumara</td>
<td>213g</td>
<td>10</td>
<td>43g</td>
</tr>
<tr>
<td>Peas</td>
<td>194g</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Carrots</td>
<td>417g</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Sauce</td>
<td>287g</td>
<td>-</td>
<td>8</td>
</tr>
<tr>
<td>Chicken</td>
<td>-</td>
<td>-</td>
<td>0</td>
</tr>
</tbody>
</table>

*Table 3.3 Total energy (kJ) and nutrient content of potato, rice and pasta meals.*

<table>
<thead>
<tr>
<th>Meal</th>
<th>Energy (kJ)</th>
<th>Protein (%)</th>
<th>Fat (%)</th>
<th>Carbohydrate (%)</th>
<th>Fibre (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>2229.8</td>
<td>17.4 (17.4)</td>
<td>28.4 (28.5)</td>
<td>50.1 (50.2)</td>
<td>3.9 (3.9)</td>
</tr>
<tr>
<td>Rice</td>
<td>2069.5</td>
<td>16.5 (17.3)</td>
<td>24.6 (25.9)</td>
<td>50.1 (52.7)</td>
<td>3.8 (4)</td>
</tr>
<tr>
<td>Pasta</td>
<td>2135.0</td>
<td>19.6 (19.9)</td>
<td>25.1 (25.5)</td>
<td>50.0 (50.9)</td>
<td>3.5 (3.6)</td>
</tr>
</tbody>
</table>

### 3.3.1 Pre-testing of Chicken

The chicken breast, prepared in the same method as described below (Section 3.4.3), was given to three participants to assess what affect, if any, it had on postprandial glycaemia. Due to meat containing no carbohydrate, it was proposed there would be little or no effect on blood glucose levels (Athar et al., 2006). An outline of the methods and results for this test is
presented in Appendix 5. In summary, there was no apparent glycaemic response to the chicken; therefore it was not included in the summation model equation.

3.4 Study Protocol

This study was performed in general accordance with the Australian Standard (2007), with the exception of testing the reference food twice instead of three times.

Participants attended early morning appointments on non-consecutive weekdays at the University of Otago, Human Nutrition Department GI Testing Facilities. To ensure blood glucose concentrations and patterns remained as similar as possible over the study duration, participants were regularly reminded of their responsibility to arrive at the clinic in a fasting state (≥10 hours), to have not drunk alcohol or exercised the night before tests, and to drive to the GI clinic if possible. If this was not feasible, and participants had biked or walked to the clinic, they were required to sit for approximately 20 minutes on arrival due to changes in blood glucose concentration induced by the exercise. This rest period let blood glucose levels stabilise.

Many measures were taken to preserve procedural consistency:

- All tests were performed over a 12-week period,
- The research assistants were trained by the candidate, and obtained all blood samples,
- The candidate and two assistants cooked all test foods and meals,
- All foods were prepared to a standardised recipe,
- The candidate took all anthropometric measurements in duplicate,
- All foods were bought from the same shop or business, and when appropriate at the same time to ensure the same batches were being used and to limit seasonal variation.

3.4.1 Blood Glucose Concentration

On each clinic day, participants reported at their scheduled time. Wheat sacks were used to heat fingertips. Fingers were sanitised using 70% isopropyl alcohol swabs (Axis® Alcohol Swab, Tollot Pty Ltd; Australia) and wiped afterwards with a non-woven swab (Multisorb®, BSN medical Ltd; China). Disposable 1.8mm lancets (Unistik® 3 Normal, Owen Mumford Ltd; England) were used to prick fingers by pressing firmly on the side of the fingertip. The finger was gently massaged to form a droplet of blood and the first drop of blood was wiped
off using a non-woven swab to remove broken cells. An additional drop of blood was massaged out and the tip of a microcuvette (HemoCue®; Sweden) placed onto the blood drop. Blood was drawn up the microcuvette via capillary action. The flat sides of the microcuvette were wiped to ensure that there was no excess blood that might interfere with the blood glucose reading. The microcuvette was immediately placed into the HemoCue® Glucose 201+ Analyser (Aktiebolaget Leo, Helsingborg, Sweden) to determine the participants blood glucose concentration in mmol/L. Each morning before and after testing the machine was calibrated using a solid control that was provided by the manufacturer.

Two fasting capillary finger-prick samples were taken 5 minutes apart via the finger prick method described above. If these two baseline blood glucose concentration readings were ±0.5mmol/L different, the participant rested a further 5 minutes and another glucose reading obtained. The average of two readings was used as the baseline value. The last baseline reading was used as time ‘zero’, which is also when the participant received their food. After fasting glucose concentrations were measured, the participants consumed a test food, a meal or a glucose beverage within 12 minutes. Blood glucose concentration was measured over a 2-hour period at 15, 30, 45, 60, 90 and 120 minutes from time zero.

3.4.2 Anthropometric Measurements
Trained personnel measured height and weight using standardised procedures (Gibson, 2005) after one of the 25g glucose beverages were consumed. Participants were asked to remove shoes and socks, and wear no excess clothing for both measurements. Height was measured using a freestanding stadiometer (Wedderburn, Dunedin) and weight was measured using electronic scales (Seca alpha, Model 770; Germany).

3.4.3 Preparation and Administration of Reference and Test Foods
Foods were not administered in a random order, as any advantage of randomisation of foods in GI studies is not clear (Brouns et al., 2005). In addition, the logistics of preparing up to 13 different foods or meals for 15 people in one morning was not feasible.

All meals were prepared in the metabolic kitchen of the Human Nutrition department, University of Otago. Water (200ml) was served alongside the food with no extra water allowed during the 120-minute test period. All foods, except the chicken, were prepared the morning of testing. The cooked weight of the food was used to ensure the correct amount of carbohydrate was present. Amounts of food required for each test or meal can be seen in Table 3.2. A brief description of preparation procedures for the foods follows:
**Reference Glucose Beverages:**

300ml bottles containing 50g dissolved glucose (CarboTest, Lomb Scientific Pty Ltd; Australia) were given to participants on 2 out of 14 mornings.

Half of the 300ml, 50g glucose beverage was measured and topped up to 300ml with water. This was used as the 25g reference glucose test and also given to participants on 2 out of 14 mornings.

**Potato:**
Dried potato mash (Potato Mash Homestyle, Continental; Uniliever Australasia) was mixed with a small amount of butter (Pams; New Zealand) and water just off the boil. For every 50 g dried potato, 5g of butter and 250ml water were used.

**Rice:**
A white rice type, Doongara (SunRice™ CleverRice®, Rice Growers Co-op., Australia) was cooked with water in a 1:3 ratio in an automatic rice cooker (Tefal Automatic Rice Cooker). The rice cooker was automatic and turned off once the rice was cooked.

**Pasta:**
Dried spaghetti pasta (Budget, Safeway Traders Ltd; New Zealand) was added to boiling water and boiled for 15 minutes. For every 100g dried pasta 1L boiling water was used.

**Kumara:**
The kumara (red, *Ipomoea batatas*; New Zealand grown) was peeled, rinsed under cold water and chopped into 1cm cubes. Sufficient water to cover the kumara whilst cooking was put onto boil. The kumara was cooked for 7 minutes then drained.

**Peas:**
One portion of frozen peas (plain, Talleys Group Ltd, New Zealand) was added to 250ml boiling water and cooked for 10 minutes in a vegetable steamer (Tefal Automatic Rice Cooker).

**Carrots:**
One portion of diced frozen carrots (Talleys Group Ltd, New Zealand) was added to 400ml boiling water and cooked for 15 minutes in a vegetable steamer (Tefal Automatic Rice Cooker).
*Sauce:*
The sauce (Tomato with Extra Cheese Pasta Bake, Dolmio®, Mars Food; Australia) was heated on the stovetop in a pot for 10 minutes. To increase the palatability of the sauce, 100ml boiling water was added to each individual portion to make a soup-like consistency. For the meal the sauce was served without added water.

*Chicken:*
Raw boneless and skinless chicken breast (Rangitikei corn fed, free range, boneless chicken breast skin off, Tegal Foods Ltd, Auckland, NZ) was chopped into 5 cm long, 1 cm wide strips and stir fried in canola oil. For every 100g of chicken breast 5g of oil was used. The cooked chicken was divided into 50g portions then blast frozen for use at a later date. On the morning of consumption one portion of chicken was reheated in a microwave for 1 minute.

*Potato Meal:*
The potato, kumara, peas, carrots, sauce and chicken were prepared using the methods described above in order to make a simple meal. Cooking times were arranged so that individual foods were ready at similar times and minimal cooling occurred.

*Rice Meal:*
Doongara rice was used in place of the potato in the meal described above.

*Pasta Meal:*
Spaghetti pasta was used in place of the potato in the meal described above.

### 3.5 Selection of Foods from the International Tables of GI

Several approaches were used to determine predicted meal GI based on values from the International tables of glycemic index and glycemic load values (Atkinson et al., 2008). The highest, lowest and average values as well as values that suited a type fit were selected and inserted into the summation model.

For the ‘type fit’ foods, a selection hierarchy was used:

1. The description and preparation method matched the description and preparation of foods in the present study.
2. The food was from and/or tested in New Zealand or Australia.
3. Description or preparation for most or all items was lacking therefore the average was selected.

4. The closest description of a food was used if the food did not have a specific published value.

Selected items and the breakdown of each model are presented in Appendix 6.

3.6 Statistical Analysis

All data was entered into the statistical programme Microsoft Excel (Microsoft® Excel ®. Version 12.0. Microsoft Corporation 2007) to determine IAUC for each food or meal. IAUC was calculated using the trapezoidal rule and ignoring area beneath the baseline concentration. A paired-sample t-test was performed on characteristics (weight, height, BMI, fasting glucose) to test for differences between age groups and gender.

The glycaemic responses to all test foods and meals were not normally distributed. Due to the skewed distribution and the innate property of GI where variance increases with the mean, the IAUC data were log-transformed, analysed for GI and back-transformed. GIs for test foods and meals are presented as geometric mean with 95% CI. With the assistance of the statistician (Sheila Williams) a mixed model analysis using the log-transformed IAUC was performed to analyse differences between GIs of observed and predicted meals.

Two methods were used to estimate meal GI, with no differences between the two approaches:

1. The GI values for meal components were inserted into a model weighted for the amount of CHO the food contributed to the meal and divided by the amount of CHO in the meal (Salmeron et al., 1997a; 11997b; Wolever et al., 1985)

2. The GI values for meal components were inserted into a constituent model weighted for the amount of CHO the food contributed to the meal (e.g. 0.5GIpotato x 0.2GIkumara x 0.8GI peas… etc).

Statistical analyses were performed in Microsoft Excel (Microsoft® Excel ®. Version 12.0. Microsoft Corporation 2007) and STATA statistical programme (StataCorp. Stata Statistical Analysis Software Release 9.2. Stata Corporation 2007). Statistical significance was set at p<0.05.
4 Results

4.1 Participant Characteristics

Thirty healthy participants aged between 21-49 participated in this study. Participant characteristics are shown in Table 4.1. Data for individual participant characteristics are presented in Appendix 7. All participants were of New Zealand European ethnicity. Each age group consisted of five male and five female participants. According to BMI, all groups except 30-40 year old males were in the healthy weight range. Mean overall fasting glucose was 4.92mmol/L.

Table 4.1 Mean (SD) values for characteristics of study sample (n=30).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>18-30yr</th>
<th>30-40yr</th>
<th>5</th>
<th>40-50yr</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average age (yr)</td>
<td>23.8 (1.8)</td>
<td>33.8 (1.7)</td>
<td>43.8 (1.7)</td>
<td>33.8 (8.69)</td>
<td></td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>72.9 (11.6)</td>
<td>81.9 (10.4)</td>
<td>70.2 (12.6)</td>
<td>75.0 (15.1)</td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.76 (0.1)</td>
<td>1.70 (0.1)</td>
<td>1.71 (0.1)</td>
<td>1.72 (0.1)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.6 (2.3)</td>
<td>28.0 (2.3)</td>
<td>24.1 (3.3)</td>
<td>25.2 (4.3)</td>
<td></td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>4.90 (0.1)</td>
<td>4.85 (0.1)</td>
<td>5.00 (0.1)</td>
<td>4.92 (0.2)</td>
<td></td>
</tr>
</tbody>
</table>

5.1 IAUC and GI of All Foods and Meals

Mean IAUC and log-transformed GIs are presented in Table 4.2, and changes in blood glucose concentration over time in response to the three meals are presented in Figure 4.1. For a single food item, kumara and peas had the highest and lowest average IAUC and GI respectively. Kumara also had the largest 95% CI and range. When examining the major carbohydrate source only, the GI of potato was significantly different to both rice and pasta (p<0.001), however the difference in GI units between rice and pasta was not significantly different (p=0.08). Individual GI values for each participant and IAUC graphs for each food are presented in Appendix 8 and 9. A comparison of values obtained in our study to those
from the international tables of GI is also presented in Table 4.2. All observed GI values or 95% CI for foods, except for the peas and sauce, were within range of the corresponding published value.

*Table 4.2* Observed IAUC and GI values (presented as geometric mean (95% CI) for all items and published mean GI (range) values for single foods.

<table>
<thead>
<tr>
<th>Food Item</th>
<th>IAUC</th>
<th>GI</th>
<th>Published Mean GI Value (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>148.7 (127.7, 173.1)</td>
<td>72 (62, 85)</td>
<td>87 (79-97)</td>
</tr>
<tr>
<td>Rice</td>
<td>99.0 (84.8, 115.5)</td>
<td>48 (41, 62)</td>
<td>54 (48-64)</td>
</tr>
<tr>
<td>Pasta</td>
<td>115.1 (97.0, 136.6)</td>
<td>56 (48, 66)</td>
<td>41 (41-58)</td>
</tr>
<tr>
<td>Kumara</td>
<td>172.5 (143.3, 207.6)</td>
<td>84 (72, 98)</td>
<td>70 (44-94)</td>
</tr>
<tr>
<td>Peas</td>
<td>37.9 (30.6, 46.8)</td>
<td>29 (25, 34)</td>
<td>52.5 (51-54)</td>
</tr>
<tr>
<td>Carrots</td>
<td>40.3 (32.1, 50.6)</td>
<td>31 (27, 36)</td>
<td>39 (33-49)</td>
</tr>
<tr>
<td>Sauce</td>
<td>45.5 (36.3, 56.9)</td>
<td>35 (30, 41)</td>
<td>52 (-)</td>
</tr>
<tr>
<td>Potato Meal</td>
<td>109.3 (90.7, 131.8)</td>
<td>53 (46, 62)</td>
<td>-</td>
</tr>
<tr>
<td>Rice Meal</td>
<td>78.6 (65.5, 94.4)</td>
<td>38 (33, 45)</td>
<td>-</td>
</tr>
<tr>
<td>Pasta Meal</td>
<td>78.0 (65.1, 93.4)</td>
<td>38 (33, 44)</td>
<td>-</td>
</tr>
</tbody>
</table>

![Figure 4.1](image) Mean changes in plasma glucose for potato, rice and pasta meals.
5.2 Application to the Summation Model

Using values obtained in our study the predicted meal GI was significantly higher than the observed meal GI in all three instances (p<0.001). Observed meal GI was overestimated by 10-17 GI units or by 19-45% (Table 4.3). As shown in Table 4.4, the resulting values for predicted meal GI using published values differed greatly, with overlaps between different GI classification groups depending on the chosen item/s. The selected items used to estimate the different meal GIs are presented in Appendix 6. It appears that the lowest values selected from the International Tables of GI and GL values best matched those measured in our study, but even the lowest values still overestimated observed GI. All but one of the predicted GIs using published values were outside of the 95% CI of the corresponding observed value (Table 4.3, Table 4.4).

Table 4.3 Mean (95% CI) observed and predicted meal GI.

<table>
<thead>
<tr>
<th></th>
<th>Observed</th>
<th>Predicted</th>
<th>P value</th>
<th>Unit Difference (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato Meal</td>
<td>53 (46, 62)</td>
<td>63 (56, 70)</td>
<td>&lt;0.001</td>
<td>10 (19%)</td>
</tr>
<tr>
<td>Rice Meal</td>
<td>38 (33, 45)</td>
<td>51 (45, 56)</td>
<td>&lt;0.001</td>
<td>13 (34%)</td>
</tr>
<tr>
<td>Pasta Meal</td>
<td>38 (33, 44)</td>
<td>55 (49, 61)</td>
<td>&lt;0.001</td>
<td>17 (45%)</td>
</tr>
</tbody>
</table>

Table 4.4 Predicted meal GIs from published values (Atkinson et al., 2008).

<table>
<thead>
<tr>
<th>Meal</th>
<th>Highest values</th>
<th>Lowest values</th>
<th>Average values</th>
<th>Best type fit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato Meal</td>
<td>79</td>
<td>63</td>
<td>72</td>
<td>73</td>
</tr>
<tr>
<td>Rice Meal</td>
<td>63</td>
<td>47</td>
<td>56</td>
<td>57</td>
</tr>
<tr>
<td>Pasta Meal</td>
<td>60</td>
<td>44</td>
<td>53</td>
<td>51</td>
</tr>
</tbody>
</table>

The predicted meal GIs when not using all carbohydrate containing foods, for example only potato and kumara, were all higher than predicted meal GIs using all carbohydrate foods (Table 4.5). When using only the main carbohydrate source plus kumara the observed meals GIs were overestimated by 24-29 GI units or by 47-76%. When using all sources of
Figure 4.2 Variation in IAUC values for reference foods.
carbohydrate except for the sauce, the overestimation was ranged from 17-23 GI units or 32-60%.

Table 4.5 Predicted mean (95% CI) meal GIs using main carbohydrate source plus kumara or vegetables only.

<table>
<thead>
<tr>
<th></th>
<th>Plus kumara</th>
<th></th>
<th>Plus vegetables (no sauce)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Potato</td>
<td>Rice</td>
<td>Pasta</td>
<td>Potato</td>
</tr>
<tr>
<td></td>
<td>78</td>
<td>62</td>
<td>67</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>(72, 83)</td>
<td>(55, 69)</td>
<td>(61, 73)</td>
<td>(65, 75)</td>
</tr>
</tbody>
</table>

It was determined how often the model correctly predicted measured meal GI according to GI classification (low, moderate, high) using values obtained in our study. The model ‘worked’ 38 times out of 90 or 42.2% of the time using this approach.

5.3 Variability

The reference foods (50g and 25g glucose beverages) elicited variation in IAUC (Figure 4.2), with overlaps in average IAUC values for 25g and 50g glucose beverages in several participants. The mean intra-personal coefficients of variation (CV) for the 25g and 50g reference tests were 23.0% and 19.4% respectively.
6 Discussion

This study is the first to robustly test if the summation model can predict the GI of meals, as it was designed to do. The GIs of three meals differing in their major carbohydrate source were predicted using the model and compared to the observed GI for each meal. There are several novel aspects of this research, specifically the testing of carbohydrate content and GI of the individual foods in a relatively large group of people. The GI of the main carbohydrate source was lowered when combined in a mixed meal containing protein and fat. The individual food GIs were inserted into the summation model, which subsequently overestimated directly measured meal GIs by 19-45%. Inserting a range of published values into the model generally resulted in a greater overestimation than this. The findings question the clinical utility of using published values to help determine food choices and also in predicting dietary GI in epidemiological studies. The overestimation was not consistent, and even with reliable knowledge of the GI of individual foods and the composition of the meal, it was still difficult to estimate a composite GI.

5.1 The Summation Model

In the present study, the observed meal GIs were significantly lower than the predicted meal GIs when inserting both measured and published values for individual foods into the summation model. The effect of protein and fat contributed from the chicken is likely to have been the main factor that caused lower than predicted meal GIs. The individual testing of each food accounted for the effects of all other fat and protein sources on GI. The chicken contributed approximately 56% of total protein and 39% of total fat content to the meals. The effects of these nutrients on the glycaemic response are well documented. A dose-response effect for the addition of fat to the meal has been previously shown (Owen & Wolever 2003) and a significant inverse relationship between protein, fat and GI exists even with small amounts of these nutrients in the meal (5-28g and 3-42g per 50g carbohydrate portion respectively) (Flint et al., 2004). This may apply to the effect on glycaemic response in the present study as the protein and fat content lie within this range. The summation model only accounts for carbohydrate containing foods, and the results from our study suggest this is the main limitation of the model. The GI is a measure of the glycaemic effect of foods, and it is this measure that is of interest in research and clinical use. Although GI is not measured in
foods that contain little or no carbohydrate, for the model to predict a composite GI more accurately, these other foods should be accounted for. If the glycaemic effect and therefore GI of the carbohydrates is used in a clinical setting and to promote low GI diets as a factor to reduce disease risk, then the effects that other nutrients have on the glycaemic potential of carbohydrate foods should be taken into account.

In addition, when lower GI foods are consumed with a high GI food, the meal GI is lowered appreciably. In line with Wolever’s work (Wolever & Jenkins, 1986; Wolever et al., 2006), the major carbohydrate source in the present study partly accounted for the response to the meal containing the same carbohydrate food. For example, when consumed alone the potato had the highest GI compared to rice and pasta, and when consumed as a meal this rank remained among the meals. The GI for pasta consumed alone was higher than the GI of rice consumed alone (56 cf 48). However when these foods were consumed within meals there was no difference in the GIs of the two meals (GI=38). Hence the rank order of pasta>rice was not maintained when these foods were part of a meal. In another study the rank order of rice and spaghetti pasta was altered in the same manner when butter and cheese were added to the carbohydrate food (Bornet et al., 1987).

The proportional contribution of available carbohydrate is an important factor to consider when predicting meal glycaemic responses. If the main carbohydrate source is contributing most of the meal carbohydrate then the meal glycaemic response is likely to be similar to that of the main carbohydrate. A significant correlation between predicted and observed GI was seen with six meals that contained 81-86% carbohydrate from the main carbohydrate source (Chew et al., 1988). When the source of carbohydrates in the meal is more evenly spread among the contributing foods, the glycaemic response of the meal will be more attributable to the combination and amounts of foods present. These previously tested meals differ to the ones in the present study in that 50% of total meal carbohydrate came from the main carbohydrate source. Given the variation seen in our results, it does not appear that the main carbohydrate source in a typical mixed meal can predict the glycaemic response or GI of the meal.

The FAO/WHO Consultation on Carbohydrates document (FAO/WHO, 1998) states that to obtain an accurate estimate of meal GI all carbohydrate sources should be accounted for. However, it has been suggested that only the main carbohydrate sources need to be used to predict a composite GI (Wolever 2002). In a study looking at dietary GI and risk of heart
disease, only staple carbohydrate foods were included in the dietary GI estimation, meaning that some vegetables were not (Grau et al., 2010). An inverse association between dietary GI and CVD morbidity and mortality in men was observed (Grau et al., 2010). In comparison to this, the Diogenes project included all foods that contributed >0.1g carbohydrate to the diet in the dietary GI prediction (Aston et al., 2010). In that study a significant improvement in weight loss with a low GI/high protein diet was observed with a small change in dietary GI (-4 GI units) (Larsen et al., 2010). Although the formula overestimated meal GI in the present study, this effect would have been even greater had we not accounted for all foods containing carbohydrate. The overestimation of predicted GI using the main carbohydrate source plus kumara ranged from 24-29 GI units (47-76%) (Table 4.5). When omitting the sauce but including the vegetables in the equation the overestimation was marginally less, ranging from 17-23 units (32-60%). Therefore when predicting a composite GI, all foods that contribute to the available carbohydrate in the meal or diet should be included.

5.1.1 Using Observed GI Values of Foods

The findings in the present study compare well to the findings from one previous study by Jenkins et al., (1984), but contrast to the findings by Wolever et al., (1985). As described in Section 2.3.1, these two previous studies were very similar in design, with virtually the same meals tested and same sample sizes used. There are several issues with these studies, with one being that very simple meals were tested. The meals contained only bread and beans and no non-carbohydrate sources or other vegetables. The predicted and observed meal GIs for the participants who had type I diabetes were 92 cf 77 (Jenkins et al., 1984b). Differences in predicted and observed GIs were also seen in participants who had type II diabetes 70 cf 60 (Jenkins et al., 1984b). In normal participants predicted and observed GI correlated well with GIs of 78 cf 77 respectively (Wolever et al. 1985). Wolever et al., (1985) criticised the former study in that the participants who had type I diabetes had a higher than usual fasting glucose on the test day for beans alone and this caused the differences in meal GI between the three study samples. However, very small samples were used in both of these studies (n= 6 or 7), which is more likely to account for the conflicting findings of these two studies. Our study mitigates the issues from these studies by testing a more typically consumed meal containing a carbohydrate source, vegetables, meat and a sauce and by using a large sample size (n=30). However, the results from Wolever et al., (1985) have been used in GI guidelines that state the GI of meals can be predicted by the GI of its components (FAO/WHO, 1998), and has also been used in other research areas, namely in the prediction of dietary GI (Salmeron et al.,
The present study helps to provide a more robust answer as to the extent that meal GI can be predicted from the GI of the individual components of the meal.

Using the measured values for foods obtained in our study, the summation model always overestimated the meal GI, however the amount of overestimation varied by 19-45% among the meals. This variable effect has been previously found when the addition of tuna to baked potato or pasta reduced the glycaemic response by 18% and 54% respectively (Henry et al., 2006). Contrary to these findings tuna added to a potato and spaghetti meal resulted in a significantly decreased glycaemic response to the potato meal only (Gulliford et al., 1989). When margarine was added a further lowering effect on the glycaemic response occurred for the potato meal but there was no change in blood glucose response to the spaghetti meal. This variable and unpredictable result may affect the classification of quintiles of dietary GI by misclassifying participants - the consequences of this have great importance to epidemiology studies, and are explained below in the section on ‘Human Health and Disease’.

5.1.2 Using Published GI Values of Foods

The GI has been tested on thousands of foods worldwide, and most have been incorporated into one document, with the most recent being the “International tables of glycemic index and glycemic load values: 2008” (Atkinson et al., 2008). Previous researchers have inserted published values into the summation model to predict meal GI and glycaemic response with variable results (Chew et al., 1988; Coulston et al., 1987; Flint et al., 2004). Coulston et al. (1987) designed three meals that differed not only in their main carbohydrate source but also in the other carbohydrate foods in the meals. The predicted meal GIs were high (71), medium (48) and low (32). However, no differences in post-prandial glycaemia were seen (Coulston et al., 1987). Likewise, the predicted GIs for thirteen breakfast meals did not correlate with the respective observed meal GIs (Flint et al., 2004). The overestimation in the present study differs to one other study where predicted meal GIs were underestimated by the model, yet were significantly correlated to observed meal GIs (Chew et al., 1988). This may be due to the variation introduced from the small sample size in that study (n=8), or because the main carbohydrate source/s contributed the majority of total carbohydrate content (~80%). The use of published values, as opposed to directly testing the GI of individual foods, introduces a further error into GI estimation.

In the present study, published values (Atkinson et al., 2008) were examined for the lowest, highest, average and best type fit GI values for the foods tested. Depending on the approach
used, the predicted meal GIs differed substantially. This introduces discrepancies among studies, as it is the investigators choice as to which values are used and often this information is not disclosed in published research (Aston et al., 2010). Depending on which set of published GI values was chosen, the range of predicted meal GIs from published values were outside the confidence interval of the tested observed meal GIs (Table 4.3, Table 4.4). This disparity between observed and predicted GI using published values highlights a major limitation in the use of the summation model to predict composite GIs.

Although the GI classification system should be considered arbitrary, the use of published GI values classed the meals as either moderate/high GI for the potato meal, or low/moderate GI for the rice and pasta meals depending on the values used. For example, the range of possible GIs for our meals was 63-79, 47-63 and 44-60 for the potato, rice and pasta meals respectively, resulting in a range of 16 units for all three meals. This illustrates the extraordinary uncertainty that is introduced by predicting a composite GI from published values. The potato meal could be classed as low, medium or high GI (53 cf 63 cf 73) depending on whether the meal GI was directly measured, calculated from the individually tested food GIs or calculated using published values. An assessment of the potato meal GI based on published values (73) would completely misclassify the GI of this meal. This has major consequences for those who use published values to design low GI meals and researchers who use published values to classify people based on their dietary GI. If potato provided the majority of carbohydrate in the diet for a participant in an epidemiological study, there is the possibility that the dietary GI will be overestimated substantially. This also applies for rice and pasta, but potentially to a lesser extent.

The use of published values to predict a composite GI has several very important consequences, explained below.

**Human Health and Disease**

Published GI values are inserted into the summation model for use in cohort and intervention studies. There is considerable heterogeneity within the current literature with positive, negative and no associations between GI and disease or disease markers found, and the use of published GI values and the summation model may partially account for this. For meals in the present study, the predicted GIs overestimated observed GI in an inconsistent manner, an effect that has been documented previously (Gulliford et al., 1989; Henry et al., 2006). If the overestimation was consistent, then the associations between dietary GI and disease would
possibly be weakened. However, as the overestimation was not consistent, the extent of
misclassification is unknown and associations are likely to be considerably weaker. For
example, the dietary GI of a person in quintile 5 of dietary GI intake could be overestimated
and should actually be classed in quintile 2. The disparity in findings attempting to link GI
with chronic disease may be partially explained by our results.

The summation model was tested as robustly as possible in the present study and it was found
that the model was unable to predict the GI of typical mixed meals. As this model is the basis
for predicting a dietary GI, it can be assumed that the model is not suitable to predict the GI
of diets. This conclusion takes into account that many more errors, including the use of
published GI values, are also introduced into dietary GI estimations. Furthermore, the
overestimation using the most suitable published values in the present study was 13 GI units
for the pasta meal, 19 for the rice meal and 20 for the potato meal. A difference of more than
10 GI units has been shown to result in changes to disease markers (Jenkins et al., 2008;
Jimenez-Cruz et al., 2003). With the variation in predicted GIs one could not be certain that
the selected GI values were true representations of the dietary GI. Hence, it becomes more
difficult to ascertain whether it was the effect of dietary GI on the outcomes of interest. Other
issues such as the choice of dietary assessment method and a lack of standardized GI values
for foods limit the ability to accurately assess dietary GI. Thus, it is possible that the
association between disease or outcome of interest and dietary GI is less reliable than initially
believed. There is a need for better dietary assessment protocol, more reliable published
values, universal criteria for selection of GI values and an improved model to predict dietary
GI.

Clinical Utility
The published GI values, which are readily available to the public and health practitioners,
are often used in the clinical setting to calculate a low GI diet for patients. Although over-
estimation is better than under-estimation for clinical use, the published values should be
useful and accurate if people are to make decisions about foods and meals they are to
consume. The overestimation shown in our study is a substantial finding considering the
observed meals resulted in a lower than predicted GI. It is assumed that this is because of the
effects of other food components such as fat and protein. This may mean that the GI of the
main carbohydrate source is of less importance than current guidelines suggest. In order to
predict the glycaemic potential of carbohydrates and the effect on health outcomes, a more
inclusive model is essential. A perfect model is unlikely due to the inconsistent nature of GI
testing, the variable nature of foods and intra-individual variation, however an improved model to predict a composite GI is plausible.

Potatoes are commonly avoided by those following a low GI diet as it has previously been shown that hot potatoes have a large amount of easily digestible carbohydrate and subsequently a high GI (Atkinson et al., 2008, Buyken & Kroke 2005; Garcia-Alonso & Goni 2000; Tahvonen et al., 2006). However, the present research demonstrates that it may be suitable to be combined in reasonable amounts into a meal. Potato had a higher glycaemic response measured as IAUC and GI than the rice or pasta, both as a single food and in a mixed meal. Even though the higher response was significant, it was relatively small, and it cannot be said with any certainty what the clinical relevance of this larger response may be. As a single food the IAUC of potato, rice and pasta were 148.7, 99.0, 115.1 mmol/Lmin⁻¹, and as a meal 109.3, 78.6 and 78.0 mmol/Lmin⁻¹ respectively. An appreciable amount of mashed potato (194g) was consumed as part of a meal in the present study and a substantial lowering effect on the GI of the potato resulted. All three meals could be considered ‘low GI’ with values of 53, 38 and 38 for the three meals, which may be helpful in a clinical setting where low GI meals are advised. While it is not being suggested that all potato meals will be low GI, recommendations that advise avoiding the consumption of potatoes may be adjusted. This adjustment could be that a reasonable amount of potato eaten within a typical mixed meal is suitable for those following a low GI diet.

Another aspect of the use of published values for the GI of mixed meals is the testing of mixed meals only. A variety of meals and convenience foods have been tested for GI, primarily for commercial purposes. A section on mixed meals is presented in the International tables of glycemic index and glycemic load (Atkinson et al., 2008). For comparison to the meals tested in the present study, those containing a form of meat are mostly low GI, with a few medium GI. For example, the GI of a sweet and sour chicken dish served with noodles (item # 1257) is 41, which is similar to our pasta meal GI of 38. Comparable dishes and GI values also exist for the rice and potato meal GIs. Thus it may be expected that the form of a meal as well as its nutritional composition are factors determining meal GI, contrary to several findings whereby the major carbohydrate source was the main determinant of meal GI (Wolever & Jenkins, 1986; Wolever et al., 1990). This may also help to inform those who commonly consume specifically developed low GI products.
5.2 Variation

Coefficient of Variation for Repeated Tests

GI testing is associated with large inter- and intra-personal variation but repeated measures can decrease the variability (Williams et al., 2008). The CVs obtained in the current study from the repeated 25g and 50g reference tests were 23.0% and 19.4% respectively. This variation is similar to or smaller than previously published CVs for similar study samples. Moderate intra-personal variation was seen in a sample from a recent inter-laboratory study where CVs ranged from approximately 12-33% (Wolever et al., 2008a). A conclusion from Wolever et al. (2008a) is that intra-individual variation, which is the largest contributor to overall variation in GI testing, should be less than 30%. The CVs in the present study were considerably lower than this indicating a reliable sample and standardised testing conditions.

Confidence Intervals

The confidence intervals obtained in the present study exhibit what has previously been found with GI testing; that variance increases with the mean. This is demonstrated by two of the test foods, peas and kumara, which had the lowest and highest GI values and accordingly the 95% CI for peas and kumara are the smallest and largest, respectively (Table 4.6). This effect of increasing variance with the mean was also seen for almost all the other foods, with the exception of rice and pasta. Overall, the 95% confidence intervals obtained in this study are relatively small (6 and 8 for the meals; 4-13 for the foods) when compared to other work in the literature (approximately 20) (Chew et al., 1988; Jenkins et al., 1984b; Wolever et al., 1985). A large 95% CI means a wide range of possible GI values exist for those foods. A simple meal of bread and beans tested in 6 healthy people resulted in a mean GI of 77 with a 95% confidence interval of 55-99 (Wolever et al., 1985), meaning the meal could have been low GI or could have had essentially the same effect as the reference food (GI=100). The small 95% CI values in the present study are attributable to the low variability of our sample and the larger sample. The low overall variability in our large study sample adds confidence to the study findings in that the results are relatively precise.
5.3 Methodology

There are several unique aspects of the methodology employed in the present study, which have allowed us to expand on the current literature investigating the use of the summation model. It also adds new knowledge into the assessment of a composite GI and will provide insight into the use of the summation model when used to predict dietary GI.

The present study was conducted according to standard GI testing protocol as described by the joint FAO/WHO report (1998) and the Australian Standard (2007).

Recruitment, Sample Size and Characteristics

The major advantage of the methodology used in the current study is the large subject numbers that tested each food. Thirty participants were recruited from the public, and this sample size meets the power calculation and aims. The number of participants recruited was greater than other studies examining GI and mixed meals (Chew et al., 1988; Flint et al., 2006; Jenkins et al., 1986; Wooley et al., 1985). Ten or more participants have been recommended when testing GI (Australian Standard, 2007; Brouns et al., 2005; Truswell, 1992), but for greater precision 25 participants should be used (Venn & Green, 2007; Williams et al., 2008). In the International Table of GI and GL many foods were tested once in less than ten participants and values presented in the ‘reliable’ list are mostly derived from eight or more subjects although some values were obtained in 6-7 subjects (Atkinson et al., 2008).

It was intended that we recruit fifteen males and females as well as ten people per age group (18-30yr, 30-40yr, 40-50yr). We recruited those aged up to 50 years to assist the generalisability of the results of the present study. As the study was powered for 30 people to show a difference of 10 GI units among foods and meals, it cannot be determined if there are between sex or among age group differences. It has been suggested that there are differences in glycaemia between males and females, however this has not been fully substantiated. Accordingly we used even numbers of each sex as recommended in the Australian Standard (2007).

Nutrient Analysis

Using recommended methods, external laboratories performed the nutritional analysis of the macronutrients present in all test foods. This has not been done before in previous studies that have investigated the summation model. To obtain the nutrient content of foods and meals,
published nutrient values for the foods are typically used because they are inexpensive and the
time to analyse the nutrient content of foods can be prohibitive (Chew et al., 1988; Flint et al.,
2004; Jenkins et al., 1984b; Wolever et al., 1985). The major issue with using published
nutrient values is that nutrient contents, in particular the carbohydrate content, can vary
substantially depending on factors such as food variety, ripeness, seasonal variation, processing
and cooking method. In addition, nutrient content for the same food has been shown to differ
when sourced and/or tested in different countries, even when the same variety or type of food
is being tested (Athar et al., 2006; Atkinson et al., 2008; FSA 2002). Chew et al. (1988)
performed their study on Australian foods, but obtained some of the GI values from Canadian
data and the carbohydrate content values from published values of foods sourced and tested in
England. If this approach or similar is taken, the food nutrient content and GI may vary
substantially to the true values, thereby introducing further error into the GI test. In general,
published nutrient values contain limited information and therefore values are only
approximations. As GI testing is based on a portion of food designed to deliver 50g of
available carbohydrate, one cannot be certain that this is achieved when using published
nutrient values of the test foods.

GI Testing

Reference tests were completed two times in each individual, which is acceptable if tested in a
large sample (Williams et al., 2008). Additionally the greatest decrease in variability of the
reference food is between the first and second test with lesser gains seen with each additional
test (Venn et al., 2006; Williams et al., 2008).

The meals tested were typical of meals that would be eaten in many Westernised countries.
These generally consist of a main carbohydrate source, non-starchy vegetables, meat, and are
often combined with sauces/gravies. The meals differed to those that have previously been
used to test the summation model, which have consisted of bread and beans, a range of typical
breakfast foods, and ethnic dishes (Chew et al., 1988; Flint et al., 2004; Jenkins et al., 1984b;
Wolever et al., 1985). The choice of the main carbohydrate source in the present study was
also typical of foods consumed worldwide as grains and root crops are the first and third most
consumed sources of carbohydrate respectively (sugar-cane is the second most consumed
Foods

Chicken was used as part of the meal to obtain a typical Western meal, which was useful in order to test the summation model when applied to a usual meal. Little of the surrounding literature on this topic has used meat before and therefore our findings are difficult to compare to others. Three meals differing in their main carbohydrate and meat source were tested in 8 healthy volunteers by Chew et al. (1988), however the predicted meal GI generally underestimated observed meal GI in this case. For example, the GI of spaghetti bolognaise was predicted to be 40, whereas a GI of 52 was observed. The main carbohydrate portion of this meal (pasta) contributed 81% of total meal carbohydrate, which is an unrealistic portion of carbohydrate content in most meals. It also means the observed GI was similar to that of pasta alone. The amount of meat in the meal, the GI of the main and other contributing carbohydrate sources, or the disparities between predicted and observed GI for each individual meal are not discussed in the paper. As the main carbohydrate source contributed the majority of meal carbohydrate one would expect the GI of the meals to be similar to that of the main carbohydrate, with negligible effects of the other foods. There is potential for the meat to have had an effect, but the results differ to ours in that observed meal GI was actually underestimated by the model.

The meals with meat that were tested by Chew et al. (1988) had carbohydrate contributions from the main source ranging between 81-86%. In addition, the meals contained similar protein amounts but approximately 10g less fat per meal when compared to the meals in the present study. It is assumed that the decrease in glycaemic response of the meals seen in the present study is likely to be due to the effects of the protein and fat in the chicken, as all other sources of fat and protein were accounted for by testing the foods individually. Nonetheless, if the summation model is to be applicable to those who need to make food choices based on GI, then the inclusion of meat should be a factor that is predictable and accounted for. To accurately determine the effect of meat on meal GI, two meals, one with meat and one without meat that were identical in all other aspects would need to be tested.

The values obtained for the GI of foods in our study are, on the whole, comparable to those previously published (Atkinson et al., 2008). It is however, uncertain why the GI of peas (29) is much lower in our study compared to the published values (51-54). Differing varieties, ripeness, seasonal effects, cooking method and times are probable reasons for the dissimilar GI values of peas. The likely reason for the sauce having a different GI to the soup used from the tables (item # 1555) is that they were not totally comparable items. This was considered the
closest food product to sauce and the predicted meal GIs using published values were not likely to have been altered by this approach due to the sauce contributing only 16% of the available carbohydrate to the meal. This further highlights the difficulties when trying to substitute the most suitable GI value when the actual food item is not available in tables. Using a sample of 30 is likely to determine more reliable GI values than the published values in which small samples were used.

**Non-randomisation**

The order in which participants consumed the reference tests, test foods and meals was not randomised to each participant. This was largely due to the logistical difficulties if one had to prepare different foods for different people each morning. It is believed that the potential for bias to be introduced by the manual allocation of the reference tests, foods and meals would be minimal (Brouns et al., 2005). No purposeful pattern of food allocation was intended when administering the test foods, with the foremost reason being ease of preparation and availability of staff. Apart from logistics, it was thought better to prepare foods and meals in batches to produce a standardised product and reduce day-to-day variability.
7 Conclusions, Recommendations and Directions for Future Research

This study assessed if the summation model could predict the GI of three meals differing in the main carbohydrate source. The GI of the foods and meals were tested in the same group of people, the carbohydrate contents of the foods were tested and a relatively large sample was used. Using this approach, directly measured meal GIs were overestimated when inserting individual food GIs into the summation model. The overestimation was generally greater when inserting published values into the model.

In the literature, variable methods have been used and inconsistent results found in determining the glycaemic responses and GI of mixed meals. The present study provides reliable information regarding the ability of the summation model to predict a composite GI. This has important research-related and clinical implications.

• There is a high probability that dietary GIs predicted by inserting published values into the summation model may not be representative of the true dietary GI. Misclassification between quintiles of dietary GI intake has probably occurred, which weakens the current literature linking GI with disease. Future researchers involved in investigations in this area should be aware of limitations associated with the model.

• Those following a low GI diet may be overestimating the GI or misclassifying some meals and a wider food choice may be possible.
7.1 Directions for Future Research

Areas for future research based on the findings from the present study would include:

1. Testing the capacity of the summation model to predict a composite GI when the amounts of foods in the meal are varied, as food portion was kept constant in the present study. Also varying the amounts of carbohydrate, fat and protein would be of interest.

2. Testing a range of meals, identical in all aspects except for one version with meat and one without meat. This would assist in determining the effect that meat has on meal glycaemic response and GI of typical meals.

3. Testing the amount of overestimation in a larger range of mixed meals. This could determine whether there is any correlation between the overestimation and the main carbohydrate source.

4. Designing an equation that incorporates dietary effects, such as those introduced by fat and protein, on the glycaemic response and GI. Other factors that influence GI such as seasonal variation of foods could also be accounted for.
8 References


Barrett JS & Gibson PR. Development and validation of a comprehensive semi-quantitative food frequency questionnaire that includes FODMAP intake and glycaemic index. J Am Diet Assoc, 2010; 110;1469-1476.


Coulston AM, Hollenbeck CB, Swislocki ALM, Reaven GM. Effect of source of dietary carbohydrate on plasma glucose and insulin responses to mixed meals in subjects with NIDDM. Diabetes Care, 1987; 10:395-400.


Foss Analytical AB ©. Foss Analytical Application Note AN380, Revision 2.3, 2005. (Supplied by Gribbles Veterinary Pathology, Mosgiel, New Zealand)


Martin CL, Murphy SP, Au DLM. Compiling glycemic index and glycemic load values for addition to a food composition database. J Food Compos Anal, 2008; 21:469-73.


Mosdol A, Witte DR, Frost G, Marmot MG, Brunner EJ. Dietary glycemic index and glycemic load are associated with high-density-lipoprotein cholesterol at baseline but not with increased risk of diabetes in the Whitehall II study. Am J Clin Nutr, 2007; 86:988-94.


Thomas D, Elliot EJ. Low glycaemic index, or low glycaemic load, diets for diabetes mellitus. Conchrane Database Syst Rev, 2009; CD006296.


Wolever TM. Low carbohydrate does not mean low glycaemic index!. Br J Nutr, 2002; 88:21112.


Wolever TMS, Yang M, Zeng XY, Atkinson F, Brand-Miller JC. Food glycemic index, as given in glycemic index tables, is a significant determinant of glycemic responses elicited by composite breakfast meals. Am J Clin Nutr, 2006; 83:1306-12.
9 Appendices

1. Ethics Application
2. Participant Information Sheet
3. Consent Form
4. Participant Questionnaire
5. Testing the glycaemic response to Chicken
6. Predicted Meal GIs Using Values from the International Tables of GI and GL Values
7. Individual Characteristics of Study Sample
8. Individual GI Values for All Foods and Meals for Each Participant
9. Figures of IAUC of Foods and Meals
10. Manuscript: Calculating meal glycemic index (GI) using measured and published food values compared with directly measured meal GI.
Appendix 1

Ethics Application
APPLICATION TO THE UNIVERSITY OF OTAGO HUMAN ETHICS COMMITTEE FOR ETHICAL APPROVAL OF A RESEARCH OR TEACHING PROPOSAL INVOLVING HUMAN PARTICIPANTS

1. University of Otago staff member responsible for project:

   (surname)  (first name)  (title)

   Venn    Bernard    Dr

2. Department:  Human Nutrition

3. Contact details of staff member responsible:

   ph. 479 5068

   email: bernard.venn@otago.ac.nz

4. Title of project:   Predicting the Glycaemic Index of meals

5. Brief description in lay terms of the purpose of the project:

   The Glycaemic Index (GI) provides a measure of a person’s rise in blood glucose following consumption of a test food relative to a reference food. Data from some observational studies suggest that consuming high GI food is a risk factor for obesity and type II diabetes. Although GI is tested with individual foods, this data has been used to obtain the GI of a
whole diet in which each food’s GI is weighted according to its carbohydrate contribution. The validity of this current model has not been robustly tested. Therefore the objective of this study is to assess how well the GI of meals can be predicted by a summation model and to compare directly the GI of three meals, which will contain similar amounts of fat and protein, but differ in their major carbohydrate source.

6. **Indicate type of project and names of other investigators and students:**

   **Staff Research**
   Department of Human Nutrition
   
   Dr Rachel Brown, Lecturer
   
   Department of Preventive and Social Medicine, Dunedin School of Medicine
   
   Assoc. Prof. Sheila Williams, Research Associate Professor
   
   **Student Research**
   
   This will be Hayley Dodd’s Masters project in 2010.

7. **Is this a repeated class teaching activity?**

   No

8. **Intended start date of project:**

   February 2010

   **Projected end date of project:**
   
   May 2010

9. **Funding of project.**

   University of Otago, Human Nutrition Department PRBF Grant

10. **Aim and description of project:**

    The primary aim of this study is to test how well the GIs of cooked meals can be predicted by summing the individual GI of foods contributing to the meals. A secondary aim is to compare the GI of two meals containing meat, vegetables and gravy but differing in their major
carbohydrate source— one a low to medium GI food (eg. rice) and the other a high GI food (eg. potato). Previous studies have found that the GI of a meal is largely determined by its primary carbohydrate source. This is yet to be robustly tested under controlled laboratory conditions. In addition, it has been suggested that the combination of foods, including fat and protein, will slow the rate of absorption of carbohydrate.

The GI of individual foods will be tested and a total meal GI predicted based on the mathematical model. The foods will then be combined into meals and the GI of two meals will be directly obtained from blood glucose levels from participants taken over a period of two hours. The results from both methods will be compared and tested statistically as to whether the summation of individual GIs is a valid approach. The mixed meal approach will test the slowing effect of other nutrients, namely fat and protein, on glucose absorption. It is hypothesized that the expected slowing effect will be proportionally greater in the high GI meal such that the GI of the two meals would tend to converge.

The results will have important practical significance. If the GI summation model works well, this study will support the practice of its use in epidemiological studies. If not, this would lead to further research to improve dietary GI assessment. For the meal comparison, if the glycaemic response to a mixed meal is largely independent of the GI of the major source of carbohydrate, this would indicate that people following a low GI diet may be able to broaden their range of foods.

For GI determinations, capillary blood is collected by finger pricking using a sterilised disposable lancet. During each test, a series of eight blood samples are collected over a period of two hours following the consumption of the foods or meals. To determine the GI of the foods and two meals, the participants must attend the clinic on 14 occasions. The test days are non-consecutive. The Department of Human Nutrition will use trained personnel to do the finger pricking.

11. Researcher or instructor experience and qualifications in this research area:

The method for conducting GI testing is well established at the University of Otago. The University has an accredited GI testing laboratory. Dr. Bernard Venn and Dr Rachel Brown are experienced in conducting research trials involving human participants. GI testing will be carried out according to our standard procedure in the Department of Human Nutrition Undergraduate Laboratories.
12. Participants

12(a) Population from which participants are drawn:

Participants will be members of the public voluntarily recruited through advertisement.

12(b) Specify inclusion and exclusion criteria:

Inclusion: Men and women in the age range of 18 - 40 years inclusive (n = 30).

Exclusions: People diagnosed with chronic disease including diabetes mellitus, cardiovascular disease, cancer, and diseases of the digestive system; who are taking any medications that affect glucose tolerance; that suffer from food allergies; and women who are pregnant.

12(c) Number of participants:

The clinical utility of dietary GI has been used to calculate the necessary sample size. In population studies, the range of dietary GI is up to 15 GI units. Data from 30 people would have 80% power to detect a difference of 10 GI units using the 5% level of significance. It would be underpowered to detect a smaller difference, but a difference of less than 10 GI units is of limited clinical significance. The study will also be large enough to predict the GI of a meal from the GI of its components.

12(d) Age range of participants:

18 - 50 years.

12(e) Method of recruitment:

Recruitment will be by advertisement in local newspapers and flyers posted around the University of Otago.

12(f) Please specify any payment or reward to be offered:

Participants will be reimbursed for their time at a rate of $35 per test equivalent to $490 for a complete set of 14 tests. Those who do not complete all tests will be paid pro-rata.
13. **Methods and Procedures:**

When volunteers first make contact in response to the advertisement an information sheet and participant questionnaire will be sent out (documents attached). The participants will return the completed questionnaire and if interested and eligible, will be booked in for their 14 appointments. At the first appointment, research staff will be available to answer questions regarding the study. If respondents are willing to continue, a consent form (attached) will be given to them. Participants will have their height and weight measured in a screened-off area to ensure the participants privacy.

Participants will attend the glycaemic index facility after an overnight fast of at least 10 hours on 14 occasions. On the evenings preceding each of these test days, participants will be advised not to exercise and to ensure that their evening meal contains a carbohydrate-rich food. On each of the test days, two finger-prick blood samples will be taken five minutes apart as a baseline blood glucose concentration. This method of collecting blood for analysis causes minimal discomfort to the participant. Human Nutrition Department personnel who are experienced in this method of blood sampling will perform the finger pricking. Blood glucose concentrations will be determined from a drop of blood using a Hemocue Glucose 201 Analyzer. Following this, a reference or test food will be consumed over a fifteen minute period and a series of six more finger-pricks will be undertaken at 15, 30, 45, 60, 90 and 120 min. In the event of an abnormal result, a repeat fingerprick may be required. Adhesive plasters will be provided to hold in place a cotton wool swab covering the small incision. The total volume of blood extracted from the finger-pricks will be less than one millilitre. Participants will be asked to remain seated for the duration of the tests. At the end of two hours the participants will be offered a light breakfast before leaving.

The Ngāi Tahu Research Consultation Committee has suggested that the researchers consider the Otago District Health Board’s Tikaka Best Practice document with regard to participant engagement. We have a copy of this document. The procedures involved with GI testing are relatively non-invasive and it is not anticipated that
culturally sensitive issues will arise. However, Māori volunteers will be asked if they would like their blood samples disposed of using standard methods or with a karakia (prayer). These options are included in the participant questionnaire.

14. **Compliance with The Privacy Act 1993 and the Health Information Privacy Code 1994** imposes strict requirements concerning the collection, use and disclosure of personal information. These questions allow the Committee to assess compliance.

14(a) **Are you collecting personal information directly from the individual concerned?**

We will be collecting contact details comprising name, mailing address, email and telephone numbers. Basic demographic and anthropometric data will be collected to enable us to describe the population groups. This will involve collecting data on age, smoking habits and gender and measuring height and weight. We will include the census question on ethnicity, a recommendation of the Ngāi Tahu Research Consultation Committee. We will confirm that the participants have not been diagnosed with diabetes mellitus, cardiovascular disease, cancer, and diseases of the digestive system. We will also confirm participants have met inclusion criteria.

14(b) **If you are collecting personal information directly from the individual concerned, specify the steps taken to make participants aware of the following points:**

- **the fact that you are collecting the information:**

  Participants will receive the information sheet and questionnaires (both are attached). Research staff will be available to answer any questions.

- **the purpose for which you are collecting the information and the uses you propose to make of it:**

  Participants will receive the information sheet and will be asked to confirm that they understand what is required of them. Research staff will be available to answer questions. All data and information will be kept in a locked room, with access limited to the researchers.
• who will receive the information:

No information containing a person’s identity will be distributed. Anonymous group demographics and statistical results may be published and/or used in future studies.

• the consequences, if any, of not supplying the information:

If the participant chooses not to supply any information, it may exclude them from the study.

• the individual's rights of access to and correction of personal information:

The participant will have rights to access the personal information they have provided may also correct or change this information. They will be advised they can request a copy of the results of the project if they wish.

14(c) If you are not making participants aware of any of the points in (b), please explain why:

N/A

14(d) Does the research or teaching project involve any form of deception?

No.

14(e) Please outline your storage and security procedures to guard against unauthorised access, use or disclosure and how long you propose to keep personal information:

The information will remain confidential to the study investigators. Paper copies will be kept in a lockable office and electronic data stored on departmental computers. The results of this study may be published but no individual’s identity will be revealed.

At the end of the project any personal information will be destroyed immediately except that, as required by the University's research policy, any
raw data on which the results of the project depend will be retained in secure storage for five years, after which it will be destroyed.

14(f) Please explain how you will ensure that the personal information you collect is accurate, up to date, complete, relevant and not misleading:
Participants will fill out their own personal details onto a participant questionnaire (attached). Height and weight will be measured by research staff, recorded and checked in the presence of the participant. The blood samples will be collected directly from the participants.

14(g) Who do you propose will have access to personal information, under what conditions, and subject to what safeguards against unauthorised disclosure?
Only study personnel directly involved in the testing will have access to personal information. The paper versions will be kept in a filing cabinet in a secure office. Electronic versions will be maintained on staff computers. The statistician will be given anonymous data.

14(h) Do you intend to publish any personal information and in what form do you intend to do this?
A person’s identity will remain anonymous in any form of published data. Demographic and anthropometric data will be presented only as group means.

14(i) Do you propose to collect information on ethnicity?
Ethnicity data will be collected. The information will not be used to draw comparisons between Māori and other ethnic groups; it is being collected only to characterize the ethnic composition of the groups.

15. Potential problems:
There will be minimal discomfort to participants from the fingerprick blood glucose test. The Department of Human Nutrition staff involved will be available throughout the test should any problems arise.

16. **Informed consent**

Please refer to consent form (attached).

17. **Fast-Track procedure**  Do you request fast-track consideration?  **No**

18. **Other committees**

   **N/A**

19. **Applicant's Signature:** .................................................................

    **Date:** ........................................

20. **Departmental approval:**  *I have read this application and believe it to be scientifically and ethically sound. I approve the research design. The Research proposed in this application is compatible with the University of Otago policies and I give my consent for the application to be forwarded to the University of Otago Human Ethics Committee with my recommendation that it be approved.*

    **Signature of *Head of Department:** .................................................................

    **Date:** ........................................
Appendix 2

Participant Information Sheet
Glycaemic Index Study- Predicting the GI of meals

INFORMATION SHEET

Please read this information sheet carefully before deciding whether or not to participate. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for considering our request.

What is the Aim of the Project?
The aim of the project is to test how well the glycaemic index (GI) of meals can be predicted by the GI of individual foods. We also aim to compare the GI when different carbohydrate sources (such as potatoes or rice) are mixed in a meal.

Project Design and Methods
The project requires attending the Department of Human Nutrition on 14 occasions. During the first visit you will be provided with information about the study. If you agree to participate and sign a consent form, we will collect some personal information from you comprising demographics, height and weight. Following this, the first GI test will be conducted. GI testing is conducted in the morning with a start time of between 7-8 am. You will be required to fast, ie: to have no food or drinks except water after 10 pm on the night before the test. We would prefer that you did not walk to the University. If you do walk or cycle we would like you to arrive 20 minutes early so that your heart rate and blood glucose have a chance to settle down before you start the test. On arrival and five minutes after, a finger-prick blood sample will be taken in the fasting state. You will then be given a glucose drink or a small meal to eat. After this, additional finger-prick blood samples will be taken at 15, 30, 45, 60, 90, and 120 min. In the event of an abnormal result, a repeat finger-prick may be required. The total volume of blood collected will amount to less than half a teaspoon. During this two hours we would like
you to remain seated in the room with the exception of toilet visits if necessary. You are free to read or talk and there will be some magazines available. At the end of two hours there will be a light breakfast available for you to eat on the premises or to take away.

Can Participants Change their Mind and Withdraw from the Project?
You may decide not to participate or withdraw from participation in the project without any disadvantage to yourself of any kind.

What Data or Information will be Collected and What Use will be Made of it?
We will collect data on your age, ethnicity, smoking habits and gender and we will be measuring your height and weight. We will also get you to confirm that you have not been diagnosed with diabetes mellitus, cardiovascular disease, cancer, diseases of the digestive system, you are not pregnant and you do not suffer from food allergies. We will collect data on medication and supplements that you are taking. The purpose of collecting this information is to describe the overall characteristics of the study population. From your blood samples we will be testing glucose concentration. The information will remain confidential to the study investigators. Paper copies will be kept in a lockable office and electronic data stored on departmental computers. The results of this study may be published but no individual’s identity will be revealed. At the end of the project any personal information will be destroyed immediately except that, as required by the University's research policy, any raw data on which the results of the project depend will be retained in secure storage for five years, after which it will be destroyed. If you choose not to supply information this may exclude you from taking part in the study. You have rights of access to the personal information that you have given to us and you may correct or change this information.

Reimbursement
There will be reimbursement for your time at a rate of $35 per test or $490 per complete set of tests. Reimbursement will be paid at the end of the study.

If you have questions about this project, either now or in the future, please contact:

Dr. Bernard Venn      Tel: 479-5068      Email: bernard.venn@otago.ac.nz

This project has been reviewed & approved by the University of Otago Human Ethics Committee.
Appendix 3

Consent Form
Glycaemic index study- Predicting the GI of meals

CONSENT FORM

I have read the Information Sheet and understand the procedures. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

I consent to:

• Attending the glycaemic index facility on 14 days following an overnight fast
• Consuming a test food, meal, or beverage on 14 occasions
• Providing eight blood samples obtained by finger pricking over two hours on each glycaemic index test day.

I know that:

• The data may be published but my name will not be disclosed
• My participation is voluntary
• I am free to withdraw from the project at any time without any disadvantage
• I will be reimbursed at the end of the study

I agree to take part in this project. Date ........................

Name ................................. Signature .................................

This project has been reviewed & approved by the University of Otago Human Ethics Committee.
Appendix 4

Participant Questionnaire
PARTICIPANT QUESTIONNAIRE

Name:

Are you male or female?

Postal address:

Email address: (if applicable)

Telephone numbers: (Work/Home/Mobile)

Date of birth:

Are you a non-smoker, past smoker, current cigarette smoker, cigar smoker or pipe smoker?

Frequency of smoking (if applicable)
Have you been diagnosed with diabetes mellitus, heart disease, stroke, cardiovascular disease, cancer, diseases of the digestive system?

Please list current medicines, dose and frequency:

Please list current supplements, brand and frequency:

Are you pregnant?

Please list any food allergies:

Please indicate to which ethnic group you belong:

New Zealand European, Māori, Samoan, Cook Island Maori, Tongan, Niuean, Chinese, Indian

Other. Please state:

Please circle whether you would like your blood samples to be disposed of using:
 a) standard methods
 b) with a karakia (prayer)
Appendix 5

Testing the Glycaemic Response to Chicken
Testing the Glycaemic Response to Chicken

It is established that meats such as chicken contain no carbohydrate, and are therefore presumed to have little effect on glycaemic responses when consumed alone (Athar et al., 2006). When combined in a meal with carbohydrate containing foods, fat and protein has been shown to have an effect on the glycaemic response. We tested the glycaemic response to chicken consumed alone to be certain its effect on the glycaemic response was only present when consumed in addition to carbohydrate containing foods.

Three participants were asked to attend the GI testing facilities for one early morning session and to have fasted the previous 10 hours. Chicken breast was cooked by shallow frying in 5g oil/50g chicken. Two fasting capillary blood glucose samples were obtained from the participants. The chicken pieces were then consumed hot over 10 minutes. Blood glucose concentration was measured every 15 minutes over the following 60 minutes.

No change in blood glucose levels, other than what would be expected for normal variation around baseline blood glucose values, was seen over the 60 minutes following consumption of the chicken.

* Table 8.1 Baseline glucose concentrations and glycaemic responses over 60 minutes.

<table>
<thead>
<tr>
<th>Participant</th>
<th>Time</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>60</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>5.3</td>
<td>5.6</td>
<td>5.1</td>
<td>5.1</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>4.9</td>
<td>4.6</td>
<td>4.8</td>
<td>4.6</td>
<td>4.8</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>5.3</td>
<td>5</td>
<td>5.7</td>
<td>5.6</td>
<td>5</td>
</tr>
</tbody>
</table>

*Figure 8.1* Glycaemic responses of three healthy individuals to chicken.
Appendix 6

Predicted Meal GIs Using Published Values from the International Tables of GI and GL: Different Approaches Presented
Table 8.2 Meal GI for potato and components using lowest published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>25</td>
<td>79</td>
<td>25 x 79/50</td>
<td>39.5</td>
<td>1660</td>
<td>12</td>
<td>65.3-92.7</td>
<td>Instant mashed potato, Idahoan Foods, USA</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>44</td>
<td>10 x 44/50</td>
<td>8.80</td>
<td>1684</td>
<td>7</td>
<td>-</td>
<td>Sweet potato, boiled, Australia</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>33</td>
<td>3 x 33/50</td>
<td>1.98</td>
<td>1623</td>
<td>8</td>
<td>23.2-42.8</td>
<td>Carrots, peeled, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>38.2-62.8</td>
<td>Pea, frozen, boiled, Canada</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>63</td>
<td>Medium GI meal</td>
<td></td>
</tr>
</tbody>
</table>

Table 8.3 Meal GI for potato and components using highest published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>25</td>
<td>97</td>
<td>25 x 97/50</td>
<td>48.5</td>
<td>1665</td>
<td>10</td>
<td>85.2-108.8</td>
<td>Instant mashed potato, Idahoan Foods, USA</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>77</td>
<td>10 x 77/50</td>
<td>15.4</td>
<td>1687</td>
<td>9</td>
<td>53.5-100.5</td>
<td>Sweet potato, kumara, New Zealand</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Carrots, peeled, diced, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>54</td>
<td>4 x 54/50</td>
<td>4.32</td>
<td>1612</td>
<td>12-15</td>
<td>26.6-81.4</td>
<td>Pea, green, (Pisum sativum), India</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>79</td>
<td>High GI meal</td>
<td></td>
</tr>
</tbody>
</table>

Table 8.4 Meal GI for potato and components using averages from published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>25</td>
<td>87</td>
<td>25 x 87/50</td>
<td>43.5</td>
<td>1660-1665</td>
<td>6-47</td>
<td>81.1-92.9</td>
<td>Only instant mash with NO added fat.</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>70</td>
<td>10 x 70/50</td>
<td>14.0</td>
<td>1684-1692</td>
<td>7-10</td>
<td>58.2-81.8</td>
<td>Includes all types of kumara and forms of cooking, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>39</td>
<td>3 x 39/50</td>
<td>2.34</td>
<td>1621-1624</td>
<td>7-8</td>
<td>31.2-46.8</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>52.5</td>
<td>4 x 52.5/50</td>
<td>4.20</td>
<td>1611, 1612</td>
<td>6-15</td>
<td>-</td>
<td>Both entries for ‘green pea’.</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>72</td>
<td>High GI meal</td>
<td></td>
</tr>
</tbody>
</table>
### Table 8.5 Meal GI for potato and components using published values with best type fit

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potato</td>
<td>25</td>
<td>86</td>
<td>25 x 86/50</td>
<td>43</td>
<td>1660-1665</td>
<td>6-47</td>
<td>81.1-92.9</td>
<td>Instant mashed potato (Edgell’s Potato Whip), Australia</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>75</td>
<td>10 x 75/50</td>
<td>15</td>
<td>1692</td>
<td>9</td>
<td>65.2-84.8</td>
<td>Sweet potato, purple skin, white flesh, peeled, cut into piece, boiled for 8 min, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Carrots, peeled, diced, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>39.2-62.8</td>
<td>Pea, frozen, boiled, Australia</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>73</strong></td>
<td></td>
<td></td>
<td></td>
<td>High GI meal</td>
</tr>
</tbody>
</table>

### Table 8.6 Meal GI for rice and components using lowest published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>25</td>
<td>48</td>
<td>25 x 48/50</td>
<td>24</td>
<td>552</td>
<td>9</td>
<td>40.2-55.8</td>
<td>Doongara rice, cooked in a rice cooker (2007), Australia</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>44</td>
<td>10 x 44/50</td>
<td>8.8</td>
<td>1684</td>
<td>7</td>
<td>-</td>
<td>Sweet potato, boiled, Australia</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>33</td>
<td>3 x 33/50</td>
<td>1.98</td>
<td>1623</td>
<td>8</td>
<td>23.2-42.8</td>
<td>Carrots, peeled, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>39.2-62.8</td>
<td>Pea, frozen, boiled, Canada</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>47</strong></td>
<td></td>
<td></td>
<td></td>
<td>Low GI meal</td>
</tr>
</tbody>
</table>

### Table 8.7 Meal GI for rice and components using highest published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>25</td>
<td>64</td>
<td>25 x 64/50</td>
<td>32</td>
<td>556</td>
<td>8</td>
<td>43.4-81.6</td>
<td>Doongara, white (1992), Australia</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>77</td>
<td>10 x 77/50</td>
<td>15.4</td>
<td>1687</td>
<td>9</td>
<td>53.5-100.5</td>
<td>Sweet potato, kumara, New Zealand</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Carrots, peeled, diced, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>54</td>
<td>4 x 54/50</td>
<td>4.32</td>
<td>1612</td>
<td>12-15</td>
<td>26.6-81.4</td>
<td>Pea, green, (Pisium sativum), India</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>63</strong></td>
<td></td>
<td></td>
<td></td>
<td>Medium GI meal</td>
</tr>
</tbody>
</table>
### Table 8.8 Meal GI for rice and components using averages from published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>Gl of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>25</td>
<td>54</td>
<td>25 x 54/50</td>
<td>27</td>
<td>552-556</td>
<td>8-10</td>
<td>48.1-59.9</td>
<td>Doongara, white (SunRice CleverRice™ brand, Rice Growers Co-op., Australia)</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>70</td>
<td>10 x 70/50</td>
<td>14</td>
<td>1684-1692</td>
<td>7-10</td>
<td>58.2-81.8</td>
<td>Includes all types of kumara and forms of cooking, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>39</td>
<td>3 x 39/50</td>
<td>2.34</td>
<td>1621-1624</td>
<td>7-8</td>
<td>31.2-46.8</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>52.5</td>
<td>4 x 52.5/50</td>
<td>4.2</td>
<td>1611, 1612</td>
<td>6-15</td>
<td>-</td>
<td>Both entries for ‘green pea’.</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>56</strong></td>
<td></td>
<td></td>
<td></td>
<td>Medium GI meal</td>
</tr>
</tbody>
</table>

### Table 8.9 Meal GI for rice and components using Published values with best type fit

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>Gl of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>25</td>
<td>54</td>
<td>25 x 54/50</td>
<td>27</td>
<td>552-556</td>
<td>8-10</td>
<td>48.1-59.9</td>
<td>Doongara, white (SunRice CleverRice™ brand, Rice Growers Co-op., Australia)</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>75</td>
<td>10 x 75/50</td>
<td>15</td>
<td>1692</td>
<td>9</td>
<td>65.2-84.8</td>
<td>Includes all types of kumara and forms of cooking, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>39.2-62.8</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>57</strong></td>
<td></td>
<td></td>
<td></td>
<td>Medium GI meal</td>
</tr>
</tbody>
</table>

### Table 8.10 Meal GI for pasta and components using lowest published values

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>Gl of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasta</td>
<td>25</td>
<td>41</td>
<td>25 x 41/50</td>
<td>20.5</td>
<td>1368</td>
<td>10</td>
<td>-</td>
<td>100% durum semolina spaghetti, boiled 15 min, Canada</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>44</td>
<td>10 x 44/50</td>
<td>8.8</td>
<td>1684</td>
<td>7</td>
<td>-</td>
<td>Sweet potato, boiled, Australia</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>33</td>
<td>3 x 33/50</td>
<td>1.98</td>
<td>1623</td>
<td>8</td>
<td>23.2-42.8</td>
<td>Carrots, peeled, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>38.2-62.8</td>
<td>Pea, frozen, boiled, Canada</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>44</strong></td>
<td></td>
<td></td>
<td></td>
<td>Low GI meal</td>
</tr>
</tbody>
</table>
### Table 8.1
**Meal GI for pasta and components using highest published values**

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasta</td>
<td>25</td>
<td>58</td>
<td>25 x 58/50</td>
<td>29</td>
<td>1364</td>
<td>8</td>
<td>40.4-75.6</td>
<td>White, durum wheat, boiled 10 min in salted water, Italy</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>77</td>
<td>10 x 77/50</td>
<td>15.4</td>
<td>1687</td>
<td>9</td>
<td>53.5-100.5</td>
<td>Sweet potato, kumara, New Zealand</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Carrots, peeled, diced, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>54</td>
<td>4 x 54/50</td>
<td>4.32</td>
<td>1612</td>
<td>12-15</td>
<td>26.6-881.4</td>
<td>Pea, green, (<em>Pisium sativum</em>), India</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td>60</td>
<td></td>
<td></td>
<td></td>
<td>Medium GI meal</td>
</tr>
</tbody>
</table>

### Table 8.12
**Meal GI for pasta and components using averages from published values**

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasta</td>
<td>25</td>
<td>49</td>
<td>25 x 49/50</td>
<td>24.5</td>
<td>1363-1377</td>
<td>6-47</td>
<td>-</td>
<td>Spaghetti, white or type NS, boiled in salted or unsalted water.</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>70</td>
<td>10 x 70/50</td>
<td>14</td>
<td>1684-1692</td>
<td>7-10</td>
<td>58.2-81.8</td>
<td>Includes all types of kumara and forms of cooking, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>39</td>
<td>3 x 39/50</td>
<td>2.34</td>
<td>1621-1624</td>
<td>7-8</td>
<td>31.2-46.8</td>
<td>Includes cooked and raw values, various methods of cooking.</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>52.5</td>
<td>4 x 52.5/50</td>
<td>4.2</td>
<td>1611,1612</td>
<td>6-15</td>
<td>-</td>
<td>Both entries for ‘green pea’.</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>53</strong></td>
<td></td>
<td></td>
<td></td>
<td>Low GI meal</td>
</tr>
</tbody>
</table>

### Table 8.13
**Meal GI for pasta and components using published values with best type fit**

<table>
<thead>
<tr>
<th>Meal Component</th>
<th>Avail. CHO (g)</th>
<th>GI of food</th>
<th>Equation</th>
<th>Weighted meal GI</th>
<th>Item</th>
<th>N</th>
<th>95% CI</th>
<th>Description/Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pasta</td>
<td>25</td>
<td>41</td>
<td>25 x 41/50</td>
<td>28.5</td>
<td>1368</td>
<td>10</td>
<td>21.4-60.6</td>
<td>100% durum semolina spaghetti, boiled 15 min, Canada</td>
</tr>
<tr>
<td>Kumara</td>
<td>10</td>
<td>75</td>
<td>10 x 75/50</td>
<td>15</td>
<td>1692</td>
<td>9</td>
<td>65.2-84.8</td>
<td>Sweet potato, purple skin, white flesh, peeled, cut into piece, boiled for 8 min, Australia.</td>
</tr>
<tr>
<td>Carrots</td>
<td>3</td>
<td>49</td>
<td>3 x 49/50</td>
<td>2.94</td>
<td>1624</td>
<td>7</td>
<td>45.1-52.9</td>
<td>Carrots, peeled, diced, boiled, Australia</td>
</tr>
<tr>
<td>Peas</td>
<td>4</td>
<td>51</td>
<td>4 x 51/50</td>
<td>4.08</td>
<td>1611</td>
<td>6</td>
<td>39.2-62.8</td>
<td>Pea, frozen, boiled, Canada</td>
</tr>
<tr>
<td>Sauce</td>
<td>8</td>
<td>52</td>
<td>8 x 52/50</td>
<td>8.32</td>
<td>1555</td>
<td>10</td>
<td>44.1-59.8</td>
<td>Tomato soup, condensed, prepared with water, USA</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>50</strong></td>
<td>-</td>
<td>-</td>
<td><strong>51</strong></td>
<td></td>
<td></td>
<td></td>
<td>Low GI meal</td>
</tr>
</tbody>
</table>
Appendix 7

Individual Characteristics of Study Sample
### Table 8.14 Individual participant characteristics

<table>
<thead>
<tr>
<th>Participant #</th>
<th>Sex</th>
<th>Age</th>
<th>Height</th>
<th>Weight</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>21</td>
<td>1.58</td>
<td>57.8</td>
<td>23.2</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>21</td>
<td>1.81</td>
<td>92.9</td>
<td>28.4</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>22</td>
<td>1.87</td>
<td>80.3</td>
<td>23.0</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>22</td>
<td>1.88</td>
<td>82.9</td>
<td>23.5</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>24</td>
<td>1.89</td>
<td>75.2</td>
<td>21.1</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>24</td>
<td>1.64</td>
<td>62.1</td>
<td>23.1</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>24</td>
<td>1.8</td>
<td>72.8</td>
<td>22.5</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>24</td>
<td>1.82</td>
<td>80.5</td>
<td>24.3</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>28</td>
<td>1.59</td>
<td>66</td>
<td>26.1</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>28</td>
<td>1.68</td>
<td>58.5</td>
<td>20.7</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>31</td>
<td>1.77</td>
<td>75.8</td>
<td>24.2</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>31</td>
<td>1.78</td>
<td>102.9</td>
<td>32.5</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>32</td>
<td>1.76</td>
<td>70.5</td>
<td>22.8</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>32</td>
<td>1.59</td>
<td>83.3</td>
<td>32.9</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>32</td>
<td>1.83</td>
<td>108.9</td>
<td>32.5</td>
</tr>
<tr>
<td>16</td>
<td>F</td>
<td>34</td>
<td>1.6</td>
<td>52.5</td>
<td>20.5</td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>36</td>
<td>1.69</td>
<td>81.3</td>
<td>28.5</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>36</td>
<td>1.76</td>
<td>115.6</td>
<td>37.3</td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>37</td>
<td>1.65</td>
<td>60.7</td>
<td>22.3</td>
</tr>
<tr>
<td>20</td>
<td>F</td>
<td>37</td>
<td>1.6</td>
<td>67</td>
<td>26.2</td>
</tr>
<tr>
<td>21</td>
<td>F</td>
<td>41</td>
<td>1.67</td>
<td>70</td>
<td>25.1</td>
</tr>
<tr>
<td>22</td>
<td>F</td>
<td>41</td>
<td>1.605</td>
<td>77.5</td>
<td>30.1</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>41</td>
<td>1.785</td>
<td>64.1</td>
<td>20.1</td>
</tr>
<tr>
<td>24</td>
<td>F</td>
<td>42</td>
<td>1.68</td>
<td>56.6</td>
<td>20.1</td>
</tr>
<tr>
<td>25</td>
<td>F</td>
<td>43</td>
<td>1.6</td>
<td>66.4</td>
<td>25.9</td>
</tr>
<tr>
<td>26</td>
<td>M</td>
<td>43</td>
<td>1.77</td>
<td>78.5</td>
<td>25.1</td>
</tr>
<tr>
<td>27</td>
<td>M</td>
<td>44</td>
<td>1.785</td>
<td>71.6</td>
<td>22.5</td>
</tr>
<tr>
<td>28</td>
<td>F</td>
<td>47</td>
<td>1.68</td>
<td>62.7</td>
<td>22.2</td>
</tr>
<tr>
<td>29</td>
<td>M</td>
<td>47</td>
<td>1.82</td>
<td>84.6</td>
<td>25.5</td>
</tr>
<tr>
<td>30</td>
<td>M</td>
<td>49</td>
<td>1.69</td>
<td>69.9</td>
<td>24.5</td>
</tr>
</tbody>
</table>
Appendix 8

Individual GI Values for All Foods and Meals for Each Participant
Table 8.15 Individual GI results for all test foods and meals.

<table>
<thead>
<tr>
<th>Participant #</th>
<th>Potato</th>
<th>Rice</th>
<th>Pasta</th>
<th>Kumara</th>
<th>Peas</th>
<th>Carrots</th>
<th>Sauce</th>
<th>Potato Meal</th>
<th>Rice Meal</th>
<th>Pasta Meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>67</td>
<td>78</td>
<td>47</td>
<td>61</td>
<td>13</td>
<td>59</td>
<td>35</td>
<td>69</td>
<td>21</td>
<td>31</td>
</tr>
<tr>
<td>2</td>
<td>49</td>
<td>11</td>
<td>43</td>
<td>43</td>
<td>48</td>
<td>48</td>
<td>30</td>
<td>24</td>
<td>19</td>
<td>27</td>
</tr>
<tr>
<td>3</td>
<td>48</td>
<td>36</td>
<td>53</td>
<td>57</td>
<td>38</td>
<td>34</td>
<td>19</td>
<td>40</td>
<td>46</td>
<td>25</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>61</td>
<td>33</td>
<td>127</td>
<td>23</td>
<td>22</td>
<td>45</td>
<td>92</td>
<td>60</td>
<td>74</td>
</tr>
<tr>
<td>5</td>
<td>106</td>
<td>70</td>
<td>69</td>
<td>57</td>
<td>62</td>
<td>45</td>
<td>55</td>
<td>34</td>
<td>39</td>
<td>30</td>
</tr>
<tr>
<td>6</td>
<td>71</td>
<td>73</td>
<td>92</td>
<td>112</td>
<td>23</td>
<td>19</td>
<td>25</td>
<td>61</td>
<td>52</td>
<td>46</td>
</tr>
<tr>
<td>7</td>
<td>81</td>
<td>30</td>
<td>56</td>
<td>74</td>
<td>44</td>
<td>9</td>
<td>32</td>
<td>88</td>
<td>30</td>
<td>47</td>
</tr>
<tr>
<td>8</td>
<td>51</td>
<td>49</td>
<td>44</td>
<td>57</td>
<td>27</td>
<td>16</td>
<td>54</td>
<td>55</td>
<td>38</td>
<td>41</td>
</tr>
<tr>
<td>9</td>
<td>88</td>
<td>40</td>
<td>53</td>
<td>64</td>
<td>29</td>
<td>19</td>
<td>17</td>
<td>76</td>
<td>23</td>
<td>51</td>
</tr>
<tr>
<td>10</td>
<td>99</td>
<td>54</td>
<td>91</td>
<td>129</td>
<td>30</td>
<td>52</td>
<td>32</td>
<td>37</td>
<td>75</td>
<td>22</td>
</tr>
<tr>
<td>11</td>
<td>65</td>
<td>96</td>
<td>82</td>
<td>108</td>
<td>35</td>
<td>15</td>
<td>47</td>
<td>28</td>
<td>56</td>
<td>52</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>50</td>
<td>44</td>
<td>51</td>
<td>8</td>
<td>25</td>
<td>22</td>
<td>69</td>
<td>45</td>
<td>40</td>
</tr>
<tr>
<td>13</td>
<td>57</td>
<td>85</td>
<td>45</td>
<td>85</td>
<td>29</td>
<td>59</td>
<td>36</td>
<td>37</td>
<td>47</td>
<td>30</td>
</tr>
<tr>
<td>14</td>
<td>81</td>
<td>50</td>
<td>72</td>
<td>90</td>
<td>37</td>
<td>70</td>
<td>26</td>
<td>41</td>
<td>40</td>
<td>29</td>
</tr>
<tr>
<td>15</td>
<td>72</td>
<td>25</td>
<td>50</td>
<td>66</td>
<td>30</td>
<td>29</td>
<td>62</td>
<td>31</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>16</td>
<td>70</td>
<td>29</td>
<td>64</td>
<td>94</td>
<td>49</td>
<td>20</td>
<td>50</td>
<td>48</td>
<td>42</td>
<td>37</td>
</tr>
<tr>
<td>17</td>
<td>95</td>
<td>63</td>
<td>60</td>
<td>69</td>
<td>36</td>
<td>70</td>
<td>75</td>
<td>99</td>
<td>54</td>
<td>51</td>
</tr>
<tr>
<td>18</td>
<td>62</td>
<td>39</td>
<td>54</td>
<td>156</td>
<td>9</td>
<td>29</td>
<td>64</td>
<td>28</td>
<td>39</td>
<td>38</td>
</tr>
<tr>
<td>19</td>
<td>99</td>
<td>72</td>
<td>79</td>
<td>93</td>
<td>50</td>
<td>46</td>
<td>43</td>
<td>88</td>
<td>34</td>
<td>28</td>
</tr>
<tr>
<td>20</td>
<td>90</td>
<td>85</td>
<td>76</td>
<td>102</td>
<td>10</td>
<td>20</td>
<td>21</td>
<td>50</td>
<td>20</td>
<td>34</td>
</tr>
<tr>
<td>21</td>
<td>114</td>
<td>92</td>
<td>68</td>
<td>133</td>
<td>38</td>
<td>20</td>
<td>19</td>
<td>43</td>
<td>54</td>
<td>41</td>
</tr>
<tr>
<td>22</td>
<td>75</td>
<td>50</td>
<td>73</td>
<td>82</td>
<td>15</td>
<td>6</td>
<td>18</td>
<td>63</td>
<td>37</td>
<td>35</td>
</tr>
<tr>
<td>23</td>
<td>59</td>
<td>23</td>
<td>25</td>
<td>112</td>
<td>39</td>
<td>76</td>
<td>74</td>
<td>49</td>
<td>48</td>
<td>60</td>
</tr>
<tr>
<td>24</td>
<td>66</td>
<td>33</td>
<td>55</td>
<td>70</td>
<td>34</td>
<td>28</td>
<td>23</td>
<td>50</td>
<td>46</td>
<td>25</td>
</tr>
<tr>
<td>25</td>
<td>81</td>
<td>47</td>
<td>67</td>
<td>50</td>
<td>25</td>
<td>24</td>
<td>32</td>
<td>76</td>
<td>40</td>
<td>55</td>
</tr>
<tr>
<td>26</td>
<td>57</td>
<td>47</td>
<td>34</td>
<td>96</td>
<td>33</td>
<td>75</td>
<td>40</td>
<td>41</td>
<td>25</td>
<td>26</td>
</tr>
<tr>
<td>27</td>
<td>89</td>
<td>40</td>
<td>60</td>
<td>103</td>
<td>62</td>
<td>50</td>
<td>51</td>
<td>70</td>
<td>22</td>
<td>35</td>
</tr>
<tr>
<td>28</td>
<td>95</td>
<td>38</td>
<td>57</td>
<td>75</td>
<td>47</td>
<td>20</td>
<td>48</td>
<td>102</td>
<td>89</td>
<td>52</td>
</tr>
<tr>
<td>29</td>
<td>45</td>
<td>50</td>
<td>76</td>
<td>118</td>
<td>29</td>
<td>32</td>
<td>28</td>
<td>52</td>
<td>38</td>
<td>49</td>
</tr>
<tr>
<td>30</td>
<td>69</td>
<td>48</td>
<td>26</td>
<td>80</td>
<td>13</td>
<td>46</td>
<td>14</td>
<td>58</td>
<td>24</td>
<td>42</td>
</tr>
</tbody>
</table>
Appendix 9

Figures of IAUC of Individual Foods
Figure 8.2 Changes in blood glucose to consumption of main CHO sources.

Figure 8.3 Changes in blood glucose to other foods in the meal.
Manuscript: Calculating meal glycemic index (GI) using measured and published food values compared with directly measured meal GI.