II REVIEW OF LITERATURE

The review of literature pertaining to the study entitled, “Determination of Glycemic Index of Selected Foods and Formulation of Low Glycemic Index Food Products”, is discussed under the following headings:

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2.1. THE GLYCEMIC INDEX

2.1.1. Meaning of Glycemic Index

The concept of glycemic index (GI) was first proposed by Jenkins and colleagues in 1981 to study the impact of the rate of carbohydrate absorption on blood glucose level after a meal (Jenkins et al., 1981). It was initially proposed as a method of ranking carbohydrate-containing foods based on the extent blood glucose level is raised, as learning the glycemic effects of specific food products may allow an in-depth understanding of physiological effects of whole diets (Wolever, Jenkins, Jenkins and Josse, 1991).

The GI of food products is divided into three classifications: high (>70), medium (55-70), and low (<55) (Kalergis, Grandpre and Andersons, 2005). Digestion and absorption of low GI foods occurs slowly, whereas for high GI foods, digestion and absorption occurs rapidly, resulting in varied glycemic responses. For this reason, low GI foods were proposed to induce many health benefits (Brouns et al., 2005; Kalergis et al., 2005; Granfeldt, Xu and Bjorck, 2006; Wolever et al., 2008).

2.1.2. Glycemic Index Determination – Internationally Accredited Protocol

GI is defined as the incremental area under the blood glucose response curve (AUC) of a 50g carbohydrate portion of a test food ingested expressed as a percentage of the response to the same amount of carbohydrate from a standard food consumed by the same subject (FAO/WHO, 1998). The higher the AUC produced from a particular food, the faster the rise in blood glucose level and larger insulin secretion, therefore, the higher the GI of the food. This result in a lower blood glucose concentration over the late (2 – 3h) postprandial period compared to the mechanism of a low GI food (Opperman, Venter, Oosthuzien, Thompson and Vorster, 2004).

GI is the AUC measured by the ingestion of 50g of available carbohydrate of a test food after an overnight fast, followed by the ingestion of a similar quantity of carbohydrate of a reference food after an overnight fast on an alternate day.

Variations in GI values have been observed in studies, which led to the questioning on the methodology used for measurement of GI. Within-subject
variability has also sparked a concern for researchers as this could eventually lead to a misclassification of GI values. Thus, an expert panel, Food and Agriculture Organization (1998), have discussed and designed a reference methodology for GI testing. According to FAO, the test should be carried out in six or more subjects to obtain the average GI value. In addition, to minimize within-subject variability, the reference food will be tested two to three times. This was further concluded by Brouns et al. (2005) that using ten subjects presents a reasonable measure of power and precision, with at least two blood sample values obtained for each timing.

Healthy subjects will have their fasting blood sample drawn, followed by consumption of the test meal within 10 minutes, and lastly, have their blood samples drawn within two hours at 15, 30, 45, 60, 90 and 120 min after starting to eat. Diabetic subjects, on the other hand, will have their blood samples drawn every half hourly for three hours (Wolever et al., 1991). Each test meal will be served with a standard beverage, water, coffee or tea with milk if desired but no sugar (Wolever et al., 2003). Subjects are given a choice of beverage to be served; however, the beverage chosen will be the same for all tests. It was reported that while caffeine is able to decrease insulin sensitivity in an acute manner (Graham et al., 2001), coffee and tea being the choice of beverage will not significantly affect the incremental area under the glucose response curve (Young and Wolever, 1998; Brouns et al., 2005; Wolever et al., 2008).

It was recommended that capillary blood be obtained for GI measurement rather than venous blood. This is for several reasons, for instance, finger prick capillary blood was easier to obtain (FAO, 1998), and the results were more consistent (Venn and Green, 2007). The use of capillary blood is further recommended by Brouns et al. (2005) as finger prick capillary blood provides the greatest sensitivity which minimizes disturbances such as fluctuations due to ambient temperature.

There have been studies investigating the glycemic responses and GI of foods with the use of glucose meters (Batra, Sharma and Seth, 1994; Anderson, Catherine, Woodend and Wolever, 2002; Heilbronn, Noakes and Clifton, 2002), reason being glucose meters are cheap, portable, produces rapid results, and requires little training for usage (Velangi, Fernandes and Wolever, 2005). Consequently, performance and reliability of glucose meters have been evaluated.
Blood samples measured using the glucose meter was found to be associated with a lower intra-laboratory SD value of GI. This suggested that the glucose meter was less precise; resulting in a reduced accuracy of results as compared to blood samples analyzed using the reference method, Yellow Springs Instruments Glucose Analyzer (YSI) (Velangi, Fernandes and Wolever, 2005; Wolever et al., 2008).

To date, there were up to ten different reference foods used for the measurement of GI (Brouns et al., 2005). Nevertheless, majority of the studies used glucose or white bread as the reference food. It has been commonly argued that bread be the choice of reference food because it allows GI measurement to be done in a more physiological manner. However, the nutritional composition of a white bread used in a particular study may differ from the other, hence, resulting in varied results which thus affect the GI value. Even so, this can be overcome by calibrating the white bread against glucose with standardized procedures such as the mode of preparations.

According to the FAO protocol, the amount of test food should contain 50g of available carbohydrate. In usual practice, available carbohydrate is obtained by subtracting dietary fiber from the total carbohydrate (FAO, 1998; Wolever et al., 2003; Brouns et al., 2005). Aston, Gambell, Lee, Bryant and Jebb (2008) conducted a study to determine the GI value of several carbohydrate-containing foods in the UK, and the portion size served was determined according to the manufacturer’s nutrition information. To minimize discrepancies, Brouns et al. (2005) has recommended the following steps for determination of available carbohydrate as: 1) Identifying carbohydrate-containing ingredients from food packaging or manufacturer’s information, 2) determination of contents (i.e. total starch, resistant starch, total sugars, sugar alcohol and detailed sugars) by analytical methods, 3) calculation of available carbohydrate with reference to contents, 4) calculation of test load for 50g available carbohydrate.

2.1.3. Factors affecting the Glycemic Index of Foods

Any process that disrupts the physical or botanical structure of food ingredients will affect digestion and absorption, leading to an increase in plasma glucose and ultimately, insulin responses (Bjorck, Grandfeldt, Liljeberg, Tovar and Asp, 1994). The main food factors involved in lowering postprandial glycemia is
through the rate of gastric emptying and/or rate of CHO digestion and absorption (Ek, Brand-Miller and Copeland, 2012).

Factors that can influence the GI of CHO-containing food products include its processing such as rolling, grinding or pressing that disrupts the outer germ layer and granules in grains leading to an increase in GI (Pi-Sunyer, 2002), preparation and cooking methods, physical form such as whole fruits vs pureed or juice, type of sugars and starch present, macronutrients such as fat and protein, anti-nutrients, resistant starch and fibre, as well as the ripeness of food (Aston, Bryant, Gambell, Jebb and Lee, 2008; Venn and Green, 2007). Theoretically, starch which has a generally higher GI is converted to sugar as fruits ripens, hence, more readily absorbed resulting in a lower GI decreases (Pi-Sunyer, 2002). However, this contradicts with what is commonly known that GI increases with the maturity of fruits (Kalergis et al., 2005; Schakel et al., 2008).

The size of particles, also contributes to the variations in GI. For example, the mashing of a 1-inch cube potato results in an increment of GI by 25% (Wolever et al., 2001, as cited in Pi-Sunyer 2002). Also, the thickness, amylose:amylopectin ratio, as well as types/shapes of pasta have different GI values. It has been explained by Pi-Sunyer (2002) that any disruption to starch that exists in large granules will allow the amylose or amylopectin molecules to become available for hydrolysis. The extent of cooking whereby the greater amount heat, water and duration of cooking the starch granules are exposed to, more will become available for hydrolysis and digestion, therefore, affecting its GI.

In grains, the extent of milling increases rate of starch digestion by increasing access of enzymes and water for starch, increasing rate of gelatinization (Ek, Brand-Miller and Copeland, 2012). For example, rice with a higher amylose-amylopectin ratio will produce a lowered metabolic response by inducing a slower digestion rate, producing a lowered glycemic and insulin response, hence, playing an important role in low GI diet (Brand-Miller, Pang and Bramall, 1992).
2.2. GLYCEMIC INDEX IN CHRONIC DISEASE

2.2.1 Glycemic Index and Diabetes Mellitus

Adverse metabolic effects of high GI foods may be exacerbated in sedentary, overweight or genetically susceptible people (Willett, Manson and Liu, 2002).

It has been postulated that the long-term intake of high carbohydrate (CHO)-containing foods with high GI that are rapidly absorbed as glucose in the body increases the risk of type II diabetes (Hodge, English, O'Dea and Giles, 2004; Ludwig, 2002; Willett, Manson and Liu, 2002). This effect can be explained by two main mechanisms: 1) increased insulin resistance or 2) exhaustion of the pancreatic β-cell by increasing insulin demand leading to the overstimulation of insulin secretion. Ludwig (2002) has explained that insulin resistance results in compensatory hyperinsulinemia following the habitual consumption of high GI foods that eventually results in β-cell failure and hence, type II diabetes.

Most of the international diabetes organizations promotes the use of GI in preventing and managing diabetes, however, not by the American Diabetes Association due to the lack of evidence to support the relationship between GI and development of diabetes (Sahyoun et al., 2008).

Willett, Manson and Liu has reported that large prospective epidemiologic studies such as Nurses' Health Study (Salmeron et al., 1997) and the Health Professional’s Follow-up Study (Salmeron, Ascherio and Rimm 1997) have found that the GI of overall diet plays a positive role in the development of diabetes (Brand-Miller, 2004; Hodge et al., 2004; Willett, Manson and Liu, 2002). In these two studies, it was found that white bread, potatoes and soda beverages led to a significant higher risk of diabetes, whilst cold breakfast cereal was associated with a reduced risk. Following the control of BMI and associated risk factors with diabetes in the Nurses' Health Study, women in the top quintile of whole grain consumption had a 27% lowered risk as compared to the lowest quintile. However, this association was not observed in the Iowa Women’s Health Study (Meyer et al., 2000), Atherosclerosis Risk in Communities Study (Stevens et al., 2002, as cited in Brand-Miller, 2004), as well as the Health, Aging
and Body Composition Study in older adults (Sahyoun et al., 2008). The findings were due to self-reporting of diabetes in the Iowa Women's Health Study, and also, extrapolation may play a significant role in the source of error in a GI database. In addition, Sahyoun et al. (2008) reported that GI was negatively associated with saturated- and total fat, as well as alcohol, which influences glucose metabolism. Higher dietary GI was also thought to be compatible with the recommended dietary guideline on limiting saturated fat intake (Sahyoun et al., 2008).

It was also reported that the replacement of high GI carbohydrates with a low GI version resulted in an improved glycemic control and the reduction of hypoglycemic episodes in patients on insulin (Willett, Manson and Liu, 2002). Hodge et al. (2004) has suggested the substitution of white bread with low GI bread as a diet high in CHO and lower GI may reduce the risk of diabetes. Brand-Miller, Hayne, Petocz and Colagiuri (2003) conducted a meta-analysis which found that a low GI diet is capable of reducing insulin secretion in type II diabetics, and in type I diabetes, it may enable the reduction in requirement of daily insulin. In addition, a diet low in GI reduced the level of glycated proteins by 7.4% as compared to a high GI diet (Brand-Miller et al., 2003).

### 2.2.2. Glycemic Index and Cardiovascular Disease

A high GI diet increases the risk for cardiovascular diseases characterized by raised postprandial blood glucose and insulin levels, contributing to oxidative stress (Ludwig, 2002; Mirrahimi et al., 2014), and lipid metabolic disorders in overweight and the obese (Dong, Zhang, Wang and Qin, 2012). It has been observed in various studies that there is a significant association between the consumption of high GI meals and increased CVD risk, especially with increasing adiposity and possibly, diabetics, however, noted only in women (Dong et al., 2012; Mirrahimi et al., 2014). In a study conducted by Hosseininpour-Niazi et al. (2013) on the association between GI and CVD risk factors, it was found that there was a positive relationship between GI, high serum triglycerides and a low HDL cholesterol level amongst participants who were obese. Also, in the non-obese participants, high GI was related to an enlarged waist circumference. A meta-analysis conducted by Mirrahimi et al. (2014) has found
that high GI diet increased the risk for coronary heart disease and stroke, with a higher risk observed in those with higher BMI, in both men and women (Fan et al., 2012, as cited in Mirrahimi et al., 2014).

On the other hand, Mirrahimi et al. (2014) has found that a diet low in GI may reduce the risk by a reduction in total and LDL cholesterol level, raising the concentration of HDL cholesterol at the same time. The level of high-sensitivity C-reactive protein (Wolever et al., 2008) and plasminogen activator inhibitor-1 (Jarvi et al., 1999) was also reduced following the consumption of a low GI diet. Ludwig (2002) also observed that triglyceride and LDL cholesterol levels were lowered, accompanied by a lower ratio of total to HDL cholesterol concentration.

### 2.2.3. Glycemic Index and Cancer Risk

GI is associated with several types of cancer, which includes breast, ovarian, endometrial, pancreatic and colorectal cancer (Biddinger and Ludwig, 2005). It is suggested that a diet high in GI increases the risk of cancer by the modulation of insulin-like growth factor (IGF) axis (Brand-Miller, Liu, Petocz and Baxter, 2005; George et al., 2009; Gnagnarella, Gandini, La Vecchia and Maisonneuve, 2008).

In the study by Brand-Miller et al. (2005) on ten lean young adults who consumed 50g of CHO portion of two foods (low GI pearled barley vs high GI potato) with similar macronutrient composition but differing GI, it was observed that a low GI meal resulted in a greater decrease in insulin-like growth factor binding protein (IGFBP)-1 as compared to the high GI meal, whereas an increase in IGFBP-3 following the consumption of a low GI meal. It was then suggested that high GI CHO-containing foods may contribute to a metabolic environment that promotes the growth of tumour, and on the contrary, IGFBP-3 acts as an anti-proliferative and pro-apoptotic factor on human cancer cells (Biddinger and Ludwig, 2005; Brand-Miller et al., 2005).

Hu et al. (2012) found that GI was positively associated with an increased risk of prostate cancer, explaining that a high GI diet causes a higher level of glucose and insulin, promoting glucose intolerance, insulin resistance and hyperinsulinemia. The relationship between insulin and cancer development is via
the exertion of mitogenic effects, both directly and indirectly, modifying the levels of IGFBPs, increasing the bioactivity of IGF-1, stimulating cell proliferation and differentiation (Kaaks and Lukanova 2001, as cited in Gnagnarella et al., 2008) inhibiting apoptosis (Pollak, 2008; Suh and Kim, 2011, as cited in Hu et al., 2012).

In a study conducted by George et al. (2009), findings suggested that GI was not strongly associated with cancer incidence. For total cancer risk, it was found that GI was positively associated with total cancer among both women and men with a high BMI. Augustin et al. (2001) found that high GI food products increased the risk for breast cancer, however, the association was not observed between medium-low-GI foods. In the Netherlands Cohort Study, Weijenberg et al. (2008), on the other hand, found no clear associations with sub-sites of colorectal cancer, hence, did not support the hypothesis that a high GI diet increases the risk of colorectal cancer. Folsom, Demissie and Harnack (2009) has found no association between GI and occurrence of endometrial cancer in women. Presently, findings on the association between GI and cancer risk are still scant and under further investigations, however, suggesting the importance of insulin in carcinogenesis (Augustin et al., 2002).

A Legume Inflammation Feeding Experiment, conducted by Hartman et al. (2010), testing the effects of a legume-enriched, low GI diet in men at risk for colorectal adenomas and insulin resistance, found relationship between the diet and improvement of fasting biomarkers of insulin sensitivity and inflammation after four weeks. However, a reduction in rate of absorption of CHOs, lowering postprandial glycemic and insulinemic responses, reducing C-peptide level (Hartman et al., 2010).

2.3. GLYCEMIC INDEX AND HEALTH

2.3.1 Glycemic Index and Athletic Performance

The concept of GI was cited as a potential concept in improving exercise performance and enhancement of exercise capacity (O’Reilly, Wong and Chen, 2010). Large glycemic and insulinemic responses following the ingestion of high GI foods promotes muscle glycogen re-synthesis, which is recommended during the recovery period. Conversely, low GI foods has been shown to produce lower
glycemic and insulinenic responses during rest in the postprandial period (Brown et al., 2013; Stevenson et al., 2005).

There have been several studies on the beneficial effects of pre-exercise ingestion of low GI CHO food products that results in a decrease in postprandial hyperglycemic and hyperinsulinemia, increasing oxidation of free fatty acids and possibly a better maintenance of plasma glucose concentrations, sustaining CHO availability during exercise (O’Reilly, Wong and Chen, 2010).

In a study conducted on eight endurance-trained male runners who consumed an isocaloric meal containing either low GI or high GI CHO foods two hours before running and after an overnight fast, Wong et al. (2008) observed that there is an improved performance time following the ingestion of a low GI meal, with blood glucose and serum free fatty acids concentration higher throughout the performance run. A lowered CHO and fat oxidation was observed as well. Consumption of the high GI pre-exercise meal resulted in a greater rate of muscle glycogen degradation and higher serum insulin concentrations during the performance run (Wong et al., 2008).

In a similar study, Moore et al. (2010) tested the effects of consumption of low GI and high GI foods 45 minutes prior to a 40 km time trial on ten male cyclists to determine the effects on metabolism and subsequent endurance performance. A greater CHO oxidation was observed after the consumption of low GI food, coupled with increased CHO availability throughout the exercise period, sustaining energy production, improving exercise performance.

On recovery period, Stevenson et al. (2005) found that the ingestion of a low GI CHO recovery diet consumed within the 24 h period following prolonged heavy exercise results in a greater endurance capacity during steady state exercise in the post-absorptive state the following day, as compared to a high CHO diet. This is possibly explained by a greater lipid oxidation rate.

2.3.2. Glycemic Index and Cognitive Function

The use of low GI meals has been encouraged for cognitive function, such as memory and attention due to better control of blood glucose concentration (Cooper, Bandelow, Nute, Morris and Nevill., 2012). Moderate increase in
circulating glucose enhanced learning and memory (Korol and Gold, 1998, as cited in Mahoney et al., 2005) and it was suggested that a high fiber, low GI and more slowly digested meal will maintain a more sustained release of glucose into the blood stream and brain.

The effects of a low and high GI breakfast was tested on 52 adolescents by the admission of three cognitive function tests, and it was found that there was greater improvement in response times and accuracy on the three tests was better maintained on the low GI breakfast. A lower glycemic response was observed too, however, there were no difference in insulinaemic response (Cooper et al., 2012).

Papanikolaou et al. (2006) studied the relationship between postprandial increase in blood glucose following the consumption of low (pasta) and high GI (bread) meals on postprandial cognitive performance, on subjects with type II diabetes. It was found that high postprandial blood glucose was associated with poorer verbal memory which was seen in subjects who consumed bread. In particular, the subjects with the highest increase in postprandial blood glucose level presented the poorest memory performance.

These findings were in line with Micha, Rogers and Nelson (2011, 2010) who found that a low GI, high GL breakfast was associated with better performance on a speed of information processing in school children, whereas high GI breakfast was associated with response times, in terms of better performance on immediate word recall task (Cooper et al., 2012; Micha, Rogers and Nelson, 2011, Micha, Rogers and Nelson, 2010).

Additionally, Ingwersen, et al. (2007) investigated the effect of low and high GI of breakfast cereal differentially affects children’s attention and memory and findings suggested that GI plays a role on children’s performance on accuracy of attention and on secondary memory.

2.3.3. Glycemic Index and Weight Management

The primary focus in dietary prevention and weight management of overweight and obesity has been on the reduction of fat intake, because high dietary fat intakes is associated with increased obesity rates. However, reducing
fat intake while increase the intake of CHO leads to an increase in glycemic effect of the diet (Brand-Miller, Holt, Pawlak and McMillan, 2002). This is explained by a typical Western diet that is high in GI, CHO food products like potatoes and bread which are rapidly digested and absorbed, increasing glycemic load and therefore, stimulating insulin secretion (Brand-Miller et al., 2002).

The benefit of a low GI diet for weight management works by reducing body mass, fat mass, BMI, total and LDL cholesterol (Thomas, Elliott and Baur 2007, as cited in Zerlin and Li, 2008; Kong et al., 2011). It was also reported that low GI food products also increased satiety, delaying the return of hunger, reducing *ad libitum* food intake (Ludwig 2000; Zerlin and Li, 2008), as well as promoting oxidation of fat at the expense of CHO oxidation (Brand-Miller et al., 2002). A low GI diet contributes to weight loss by influencing appetite, metabolism and levels of insulin (Ludwig, 2002), thus playing an important role in weight and obesity management (Henry, Simonite and Warren, 2003). On the other hand, high GI food products as a result of refining of wheat has been found to decrease postprandial satiety (Holt and Miller, 1994, as cited in Brand-Miller et al., 2002), increasing hunger and subsequent food intake (Ludwig, 2002).

In the adult DANES cohort study conducted in both men and women, it was found that GI was positively associated with body weight, percentage body fat and waist circumference in sedentary women, however, not observed in men (Hare-Bruun, Flint and Heitmann, 2006). Also, Ma et al. (2005) also found that body mass index (BMI) is positively associated with GI, reinforcing the importance of GI and weight management. Turner-McGrievy et al. (2011) examined the effect of GI on weight loss and glycated haemoglobin (HbA1c) levels in type II diabetics by designing a low-fat, low-GI vegan diet or diets based on the 2003 American Diabetes Association dietary recommendations. Findings of this dietary intervention study showed the association between reduction in GI and weight loss, implying that overweight type II diabetics may benefit from a low GI diet which is predictive of weight loss, which in turn is predictive of the reduction in HbA1c levels. Additionally, weight gain was positively associated with high intake of refined grains, who are commonly high in GI, however, negatively associated with high intake of fiber and whole-grain products that are commonly low in GI (Liu, Willett and Mason, 2003, as cited in Marsh and Brand-Miller, 2008).
On the contrary, Sloth et al. (2004) investigated the long-term effects of a low and high GI low-fat, high-carbohydrate diet in overweight women, and found that there was a lack of difference between groups in energy intake, body weight and fat mass changes, however, achieving a 10% decrease in LDL cholesterol and a larger decrease in total cholesterol with low GI diet, supporting the concept of a low GI diet being beneficial for ischemic heart disease.

On weight loss maintenance, five ad libitum diets were investigated: 1) low protein-low GI, 2) low protein-high GI, 3) high protein-low GI, 4) high protein-high GI, and 5) control diet based on dietary recommendations of the country. Overweight participants who administered the high protein, low GI diet had a higher rate of weight loss maintenance, therefore, could be employed in preventing regaining of weight in weight management programs (Larsen et al., 2010).

2.3.4. Glycemic Index and Pregnancy

It is recognized that maternal weight and weight gain during pregnancy influences pregnancy outcome, such as fetal growth rate and infant birth weight (McGowan and McAuliffe, 2010; Moses et al., 2006; Scholl, Chen, Khoo and Lenders, 2004). High circulating maternal blood glucose level meant an increased glucose transfer to the fetus, and therefore, initiating an increased compensatory fetal insulin secretion (McGowan and McAuliffe, 2010). There is evidence to show a direct relationship between maternal blood glucose level during pregnancy and foetal growth and size at birth (McGowan and McAuliffe, 2010). It was found that high GI diet resulted in a progressive increase in postprandial glucose response, whereas it remained the same following a low GI diet (Clapp, 1998, as cited in McGowan and McAuliffe, 2010). Furthermore, women consuming a high GI diet gave birth to a large-for-gestational-age (LGA) infant, as compared to women on low GI diet, with a difference mean weight of 1000g more. This is in line with the findings of Moses et al. (2006), furthermore, women who consumed the low GI diet did not gave birth to infants who were SGA or LGA, however, LGA infants were more likely to be those on high GI diet. Thus, it was concluded that the consumption of a diet high in CHO, constituting >50% of one’s daily energy intake with low GI varieties, can help to maintain blood glucose concentrations within the
normal range, lower postprandial glycemia, reducing the incidence of fetal macrosomia (Louie, Markovic, Ross, Foote and Brand-Miller 2013; McGowan and McAuliffe, 2010), also, reducing excessive maternal weight gain (Louie, Brand-Miller, Markovic, Ross and Moses, 2010). Low GI diet works by flattening the progressive increase in insulin resistance and postprandial glucose levels as commonly seen in late pregnancy.

In an intervention study by McGowan, Walsh, Byrne, Curran and McAuliffe (2013) conducted on eight hundred women randomized to receive low GI and healthy eating dietary advice or to receive standard maternity care, it was found that a low GI dietary intervention during pregnancy led to a significant reduction in maternal dietary GI and gained significantly less weight that the control group.

The above findings were, however, not in line with a study by Louie et al. (2011), whereby the effect of a low GI diet or a high-fiber moderate-GI diet was tested on 99 women diagnosed with GDM at 20-32 weeks’ gestation. The study yielded similar result between the low GI and high fiber diet, suggesting that earlier intervention may be beneficial, supporting the concept of a low GI diet to be a safer alternative for women with GDM. Louie et al. (2013), on the contrary, found that low GI breakfast produced a lower postprandial glycemia amongst women with diet-controlled GDM in 30-32 weeks of gestation, potentially reducing the excessive transfer of maternal-fetal blood glucose.

2.4. GLYCEMIC INDEX: APPLICATION TO MIXED MEALS

2.4.1 Effect of individual macronutrients on glycemic responses

The addition of fat and protein to CHO reduces glycemic responses by delaying gastric emptying, mediated by gut hormones such as gastric inhibitory polypeptide and glucagon-like peptide-1 (GLP-1), despite a small glucose effect, and stimulating insulin secretion by the direct effects of fatty and amino acids (Moghaddam, Vogt and Wolever, 2006; Pi-Sunyer 2002; Wolever et al., 1991). It was also stated that 30g of protein and 50g pf fat per 50g of available CHO may decrease the GI (Wolever et al., 1994, as cited in Augustin, Franceschi, Jenkins, Kendall and Vecchia, 2002).
It has been shown that the higher amount of CHO in a food as opposed to protein and fat, the GI will be higher, and a mixed meal will have different glycemic response due to different proportion of the macronutrients (Pi-Sunyer, 2002). Fat also contributes to delayed gastric emptying by promoting the secretion of incretins, thus its ability to reduce glycemic impact by reducing postprandial blood glucose (Owen and Wolever, 2003). Increased amount of protein taken together with CHO will result in an increased insulin response but postprandial glucose will not change much (Pi-Sunyer 2002), as observed when 25g of protein was administered together with a mashed potato or spaghetti meal which resulted in a slight reduction in glycemic response, although increasing insulin responses in noninsulin-dependent diabetics (Gulliford, Bicknell and Scarpello, 1989; Nuttall, Mooradian, Gannon, Billington and Krezowski, 1984). Correspondingly, adding fat to a CHO meal stimulates insulin secretion even though plasma glucose response decreases (Pi-Sunyer, 2002). It was also stated that the relationship between protein and fat with reduced glycemic responses is dependent on the individual's fasting plasma insulin, waist circumference, insulin sensitivity, dietary fiber and fat intake (Wolever et al., 2008). In addition, the source of fat and protein influences postprandial glucose responses differently (Lan-Pidhainy and Wolever, 2010).

In a study conducted by Moghaddam, Vogt and Wolever (2006) in normal and hyperinsulinemic subjects, given 0-30g of corn oil and soy protein concentrate, in 16 different test meals consisting of 50g of glucose with varying ranges of fat and protein in a randomized block design. It was reported that studies may have resulted in a reduction of glycemic response non-linearly, however, there was no evidence for this similar response. Protein and fat had an independent effect of each other, with protein exerting a larger effect by two to three times greater as compared to fat. It was also observed that the effect of fat on glycemic responses was larger in subjects with a lower fasting plasma insulin, while protein exerted a greater effect in subjects with a higher waist circumference and greater intake of fiber. This finding may help to explain that fiber and waist circumference may be associated with glycemic responses, however, other factors such as protein digestibility, specific amino acids components in stimulating insulin should be taken into consideration as well. Furthermore, effect of fat on glycemic response was not different between the control and subjects with
hyperinsulinemia, however, noting a significant correlation between fat and fasting plasma insulin in individual subjects, in line with studies showing that fat played no role in glycemic responses in diabetic subjects. This suggests that insulin resistance was the possible cause for the absence of fat effect (Moghaddam, Vogt and Wolever, 2006).

A similar study was also conducted on health non-diabetic patients given 50g of oral glucose with 0-30g doses of canola oil and whey protein over a period of 11 separate mornings, it was found that glucose level was significantly reduced by the intake of protein, increasing insulin and GLP-1, without any significant effect on C-peptide or rate of insulin secretion. Protein intake occurs with a significant progressive reduction in GI. Conversely, the addition of fat to CHO did not lower glucose response, and this may be due to the difference between solid and liquid meals or the increment in insulin following fat ingestion may be too small to decrease glucose level, or also, the role of fat and release of cholecystokinin and peptide YY. However, findings were in line with Moghaddam, Vogt and Wolever (2006) that protein exerted twice the effect of fat, also, the hypoglycemic effect of fat and protein was found not to be influenced by the degree of insulin sensitivity (Lan-Pidhainy and Wolever, 2010).

MacIntosh, Holt and Brand-Miller (2003) investigated the effect of fat saturation on ten healthy men given 50g of CHO portion of mashed potato with isoenergetic amounts of butter, Sunola oil (mono-unsaturated fatty acid), or sunflower oil (poly-unsaturated fatty acid). Fat was shown to increase insulinemia, however, it was found that the degree of fat saturation had no significant effect on acute glycemia, insulinemic and satiety responses, suggesting that substitution with healthier fat sources played no role in improving acute postprandial hyperglycemia and insulinemia (MacIntosh, Holt and Brand-Miller, 2003).

Additionally, Fernandez-Raudales, Diaz-Rios, Lotton, Chapman-Novakofski and de Mejia (2012) investigated the effect of protein profile on glycemic response of low glycinin soymilk and conventional soymilk, both being low GI, in overweight and obese men. High, regular and non-β-conglycinin beverages produced no differences in postprandial glucose response, hence, concluding that beverages with different protein profiles with similar GI values had no differences in glycemic
responses, and also, suggesting that the high content of β-conglycinin did not produce any effect in postprandial blood glucose response in overweight men.

2.4.2. Effect of combination of fat and protein on glycemic responses

In the study by Wolever, Yang, Zeng, Atkinson and Brand-Miller (2006), it was found that varying the amount of protein and fat (0-18g) had a negligible effect on mean glycemic response in mixed meals, also in line with Wolever and Bolognesi (1996), implying that CHO content and GI of the different test meals should be varied over a reasonably large range and that the variability of fat and protein content should be similar to that of CHO in order to examine the effect of fat and protein on glycemic response.

Co-ingestion of mashed potato and/or spaghetti with 25g of fat (Flora margarine) and 25g of protein (tuna fish), were administered to six non-insulin-dependent diabetic subjects, compared to protein ingestion with a CHO meal alone. Results showed that blood glucose response was reduced, suggesting that gastrointestinal motility is a limiting factor, however, this effect was noted only in the mashed potato meal and not the spaghetti meal. This finding suggests that the physical structure of pasta is associated with reduced rates of starch hydrolysis, lowering the blood glucose responses, and that the effect of fat was not limited by rate gastrointestinal transit alone. Also, findings also show that varying meal composition will vary the glycemic response as well. Hence, blood glucose responses to CHO-rich foods are not equally modified by the co-ingestion of fat and protein (Gulliford, Bicknell and Scarpello, 1989).

The glycaemic and insulinaemic responses to a mashed potato meal with the addition of rapeseed oil, chicken breast, and/or salad was examined in 11 healthy subjects. GI of pure mashed potato was 108, however, when combined with the other side dishes, GI is reduced to 54. It was found that chicken breast increased insulinaemic response, however, the addition of rapeseed oil led to a decrease instead. Findings also showed that adding fat and/or protein to a CHO dish will result in a lower glycaemic responses. Hence, estimation of GI of a mixed meal was considered to be imprecise due to the presence of fat and protein (Hatonen et al., 2011).
Nevertheless, Ning, Brown, Venn, Williams and Green (2010) found that variations in the fat and CHO contents on evening meal preceding GI testing held the next morning played no significant role in affecting glycemic response or GI. In addition, when both fat and protein are administered together, Moghaddam, Vogt and Wolever (2006) were unable to rule out the existence of a fat and protein interaction as it was insignificant, and recommended a larger sample size in the future for investigation.

2.4.3. Calculation of mixed meal or diet GI

Since the development of GI, there were controversies pertaining to its application in mixed meals. Many studies on low GI meals that have been shown to reduce hunger and increase satiety are confounded by many barriers such as macronutrients content, and this is because people usually consume complex mixed meal rather than single CHO food item. Hence, a formula is derived to calculate the GI of a mixed meal, for example, containing three CHO foods. This can be calculated using the sum of GI of the individual food that is multiplied by the proportion of available CHO, divided by the total available CHO in the entire meal, to give the GI of whole meals (Wolever and Jenkins, 1986).

Debates have been sparked as some researchers have found that the GI of certain foods may be able to predict the glycemic and insulin response in mixed meals, whereas some have found otherwise (Kalergis et al., 2005). However, this can be due to disparities between the methodologies used. GI of individual foods is a significant determinant of meal GI (Wolever et al., 2006), which is virtually proportional to the mean glycemic response, despite the hypoglycemic effect of fat and protein presented (Collier et al., 1986). This is demonstrated also in a study by Chew, Brand, Thorburn and Truswell (1988) on the application of GI in mixed meals by using the glycemic and insulin response of healthy individuals to six different mixed meals and compared the results with published GI indices. Results from the study were in line with several studies (Wolever et al., 1985; Collier, Wolever, Wong and Josse, 1986; Wolever, Yang, Zeng, Atkinson and Brand-Miller, 2006) which shows that the method evaluated is successful in predicting the glycemic responses in both healthy and diabetic individuals to mixed meals.
Flint et al. (2004) conducted a similar study to investigate the applicability of GI in the context of mixed meals and have deduced that there was no correlation. However, this finding could be due to methodology differences as it was identified that an array of GI values for foods could account for misclassification errors as the wrong value was chosen for the specific food item (Brouns et al., 2005; Wolever et al., 2006). It was recommended by Brouns et al. (2005) that the GI of individual foods should be measured through analytical procedures to achieve the most precise GI of mixed meals.

To ascertain whether GI and CHO content of individual foods played a role in the glucose and insulin responses elicited by realistic mixed meals, 14 test meals with varied energy, protein, fat, available CHO and GI were tested. Wolever et al. (2006) have found that for mixed meals containing 0-18g of fat, 0-18g of protein, ≈ 220-450 kcal and 16-79g available CHO, the CHO content and meal GI explains ≈90% of the variation in mean glycemic response, with negligible effects of protein and fat. Therefore, GI is a significant determinant of glycemic effect of mixed meals conducted in normal subjects.

The effects of mixed meals with different GI and CHO content on postprandial glycemic and insulinemic response was investigated in overweight or obese adults. Four isocaloric meals were served throughout a day: 1) high GI-high CHO, 2) high GI-low CHO, 3) low GI-high CHO, and 4) low GI-low CHO. It was found that all except the high GI-high CHO diet resulted in a significant reduction of glucose and insulin area under the curve, however, no significant differences observed in terms of ratings of hunger, fullness or satiety. It was also observed that glycemic and insulinemic responses were the highest after breakfast with the high GI-high CHO breakfast meal ranked the highest, also, suggesting that lowering GL of mixed meals is effective in controlling blood glucose and insulin levels throughout the day, making it a good predictor for postprandial responses overweight and obese participants (Liu et al., 2012).
2.5. GLYCEMIC LOAD

2.5.1 Definition of Glycemic Load

The glycemic load (GL) is defined as the product of GI and total available CHO content in a given amount of food, (available CHO content food item) X [(given amount of food) X (GI)].

GI compares equal quantities of CHO and provides a measure of the quality of CHO, however, not quantity. Therefore, researchers at Harvard University introduced the concept of GL in order for the quantification of overall glycemic effect of a portion of food consumed, allowing the comparison between realistic portion sizes of food (Foster-Powell, Holt and Brand-Miller, 2002; Venn and Green, 2007). The higher the GL, the greater the elevation in blood glucose level and insulinemic response is expected to be. However, blood glucose incremental area under curve (AUC) does not increase directly in proportion to the amount eaten (Venn and Green, 2007), as explained by Brand-Miller et al. (2003) and Venn and Green (2002), increasing the amount of food consumed decreases the rate of increase in AUC at high doses, for example, four and six slices of bread consumed has been shown to result in flattening of blood glucose response.

For the consumption of individual CHO-containing food products, GL will be more relevant as compared to GI. This is because, taking carrot for example, it has a high GI but low GL as it contains only a small amount of CHO which has a minimal effect on blood glucose or insulin level. This is also in the case for watermelon. Hence, suggesting the significance and applicability of GI when one’s diet is high in CHO (Willett, Manson and Liu, 2002). It has also been recommended that researchers and health professionals calculate their own GL data using the appropriate serving sizes and CHO-composition data because portion sizes between different people and countries may vary (Foster-Powell, Holt and Brand-Miller, 2002).
2.6. DEVELOPMENT OF LOW GLYCEMIC INDEX FOOD PRODUCTS

2.6.1 Selection of Low Glycemic Index Ingredients

Research studies have yielded evidence that establishes a low GI diet being associated with many beneficial health outcomes with the increase in awareness of the GI concept; this in turn led to the development of foods with a low GI (Brouns et al., 2005; Thondre and Henry, 2009). Low GI food products include legumes, pasta, rice, sourdough bread and burger-type products (Kumar and Prabhasankar, 2014).

Therefore, low GI ingredients play an important role in the promotion of low GI diet. Modification of starches reduces GI by changing its structure via physical, enzymatic, and/or chemical means. Therefore, changing its properties as such that they are able to withstand extreme heat, acidity, shear, time, temperature, increasing visco-stability, or to lengthen or shorten gelatinization time (Kumar and Prabhasankar, 2014).

Saczzina, Siebenhendl-Ehn and Pellegrini (2013) has reviewed possible strategies to reduce the GI of bread: 1) high beta-glucan genotype flour of barley or oat, 2) addition of soluble fibres such as guar gum, arabinoxylans, high molecular weight beta-glucans, psyllium or resistant maltodextrin, 3) addition of whole or cracked kernels, 4) high resistant starch and amylose content, 4) addition of fractions enriched in fibre by water extraction, and 5) sourdough fermentation.

The use of intact cereal kernels and kernel-based breads are characterized as low GI, and this is due to obstructed amylolysis, a result of botanical encapsulation of starch or limited starch swelling (Bjorck and Elmstahl, 2003). A low-temperature long-duration baking promotes the growth of crystalline amylose, also known as annealing (Eerlingen et al., 1993, as cited in Bjorck and Elmstahl, 2003), with the use high-amylose barley flour led to an increase in resistant starch content and reduction in GI. In the same article, it was also noted that sourdough fermentation or addition of organic acids (e.g. acetic acid) can be utilized to lower the GI of wholegrain bread products (Bjorck and Elmstahl, 2003), affecting glucose response by partly slowing gastric emptying (Pi-Sunyer, 2002).
Soluble viscous fibers have been known to increase the amount of unavailable carbohydrate, reducing the GI of diet by flattening postprandial glycemia via the reduction in rate of absorption. The viscosity of fiber is directly associated with the degree it flattens postprandial glycemia (Jenkins et al., 1978, as cited in Jenkins et al., 2002). This includes beta-glucan, which is found in several cereal products which comprises of oats, barley, rye and wheat (Casiraghi et al., 2006; Jenkins et al., 2010).

A prototype beta-glucan-enriched breakfast cereal and bar, sweetened with fructose to ensure palatability was developed and tested on type II diabetes, and it was found that both prototypes resulted in a lowered postprandial glycemia and a significantly lower glycemic response as compared to commercial oat bran breakfast cereal that is naturally high in beta-glucan. The reduction in GI of food was four units per gram of beta-glucan, which was similar to oat bran that has undergone minimal processing (Jenkins et al., 2002).

Another study involving the use of barley beta-glucan was the development of chapatis, a form of unleavened Indian flatbread. Thondre and Henry (2009) used high molecular weight barley beta-glucan in this study and it was shown that with four and eight grams of dosage per serving, GI of chapatis were reduced significantly. Also, as compared to what is shown in literature, four grams of beta-glucan is sufficed to bring about a significant reduction in GI.

Furthermore, Jenkins, Kacinik, Lyon and Wolever (2010) investigated the use of a novel viscous polysaccharide fiber, PolyGlycopleX (PGX) and its effect on GI in commonly eaten foods. However, with increasing amount of fiber, palatability of a food product decreases as the viscosity is higher. With the development of PGX, a commercial product which is able to delay the onset of viscosity, palatability of fiber increases when it is incorporated in food. It was shown in the study that with the addition of PGX, many of the test foods could be reclassified with the new GI value achieved. Furthermore, GI of yogurt was reduced moderately with the incorporation of PGX despite it being low GI originally.

The use of unripe banana flour represents a source of indigestible CHOs, i.e. resistant starch, and also, antioxidant properties. In a study using unripe
banana flour in the development of cookies, it was found that the amount of rapidly
digested starch decreases whereas slowly digestible starch was increased with an
increased amount of unripe banana flour, resulting in lowered hydrolysis percentage
and predicted GI (Agama-Acevedo et al., 2012).

2.6.2. Processing and other factors affecting GI of food products

Food processing methods are associated with loss of functional properties of
food ingredients, which in turn influence the GI of carbohydrate-containing food
products. For example, the physical state of starch will have the greatest impact on
GI rating of foods, The GI of foods varies with degree of starch gelatinization, nature
of starch, amylose to amylopectin ratio, particle size, pH, fiber, fat and protein
content, ripeness, storage, cooking method and time (Kalergis et al., 2005; Schakel
et al., 2008). Bjorck et al. (1994) has explained that gelatinization of starch increases
its availability to amylases (Holm, Lundquist and Bjorck, 1991, as cited in Bjorck et
al., 1994), and on the other hand, low processing temperature and moisture content,
or the enclosure of food ingredients in its intact botanical tissue will obstruct the
hydration of starch in product, both factor which plays a role in glucose and insulin
responses.

Aman, Rimsten and Andersson (2004) developed pasta and pancakes with
the incorporation of oats, and have found that effects of food processing on beta-
glucan degradation were more pronounced after undergoing heat treatment.

Increasing the proportion of resistant starch is effective in lowering the GI of
foods. The effects of cooking methods and storage conditions has been explored
by Yadav (2011) on tubers and bread. As compared to shallow frying, deep frying
resulted in a more pronounced effect in reducing the resistant starch content of
potatoes and sweet potatoes. This was explained by the marked reduction in
resistant starch content in deep frying because more fat becomes available for the
lipid-amylose complex formation, hence, absence of water inhibited the process
amylose chain crystallization, reducing the amount of resistant starch. On the other
hand, long-time low-temperature baking of bread increased resistant starch content
as endogenous enzymes remained active for an extended period, resulting in
possible de-branching of amylopectin producing short chains. Increasing storage
time of bread also allowed retrograded amylose to contribute to the formation and therefore, increasing the amount of resistant starch (Yadav, 2011).

Effects of popping on sorghum starch digestibility and predicted GI has also been studied by Saravanabavan, Shivanna and Bhattacharya (2013). Unprocessed sorghum is high in its resistant starch content and it was found that popping of sorghum resulted in increased total starch content and a decrease in amylose and resistant starch content, hence, increasing starch digestibility, yielding a high GI due to a higher soluble amylose content.

In spite of the deleterious effects of food processing, Casiraghi et al. (2006) investigated the glycemic and insulin responses of crackers and cookies made from barley flour enriched with beta-glucan. It was illustrated in the experimental study that although simple sugars in the cookie dough may lower water activity, resulting in a lower degree of starch gelatinization which impacts on the GI of cookies, the functionality of beta-glucan being able to entrap available water could be a possible explanation for the significant reduction in GI.

In addition, the effects of processing on GI was tested on oats and barley, whereby they were processed under conditions simulating commercial production. Oat grains were steamed and roasted, and barley grains were steamed, before rolling to thin flakes. These heat treatment resulted in a low degree of gelatinization, however, resulting in a high postprandial glucose and insulin responses, therefore, causing a high GI. Thin oat flakes with a low degree of gelatinization was found to be insufficient to lower the glycemic and insulinenic responses, however, thick oat flakes with a similar degree of gelatinization as the thin version produced a significantly lower GI. This finding was thought to be a reduced access to amylase as the outer layer of endosperm or cell walls are less disrupted. Still, this effect was not observed in thick barley flakes which yielded a high GI (Granfeldt, Eliasson and Bjorck, 2000).