

**THE GLYCEMIC INDEX OF SOME TRADITIONAL ETHIOPIAN
FOODS IN MICE**



**BY
MEAZA ASSEFA**

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By

Meaza Assefa

Msc Biochemistry

ID Number: GSR/ 3360/ 04

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Under the supervision of:

DR. Melaku Umeta, PhD (Department of Biochemistry, School of Medicine, Addis Ababa University).

DR. N.Gnanasekran, PhD (Department of Biochemistry, School of Medicine, Addis Ababa University).

University of Addis Ababa

ADDIS ABABA UNIVERSITY

School of Graduate studies

This is to certify that the thesis prepared by **Meaza Assefa Zegeye** entitled: “**The glycemic index of some traditional Ethiopian foods in mice**” and submitted in partial fulfillment of the requirements for the Degree of Master of Science (Medical Biochemistry) complies with regulations of the University and meets the accepted standards with respect to originality and quality.

Signed by the Examining Committee:

Examiner: _____ Signature: _____ Date: _____

Advisor: _____ Signature: _____ Date: _____

Advisor: _____ Signature: _____ Date: _____

Chair of Department or Graduate Program Coordinator.

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LIST OF ACRONYMS

| | |
|-------|--|
| AAU | Addis Ababa University |
| AOAC | Association of Official Analytical Chemists |
| AUC | Area Under the concentration over-time Curve |
| CHO | Carbohydrate |
| CHD | Coronary heart disease |
| CRP | C- reactive protein |
| CVD | Cardiovascular disease |
| DM | Diabetes Mellitus |
| EFSA | European Food Safety Authority |
| EHNRI | Ethiopian Health and Nutrition Research Institute |
| FAO | Food and Agricultural Organization of the United Nations |
| GI | Glycemic Index |
| GIT | Gastrointestinal tract |
| GL | Glycemic Load |
| HCl | Hydrochloric acid |
| HDL | High density lipoprotein |
| Hr | Hours |
| IAUC | Incremental Area Under the Curve |
| IGF-1 | Insulin-like growth factor 1 |
| LDL | Low density lipoprotein |
| MI | Myocardial infarction |
| NIDDM | Non Insulin Dependent Diabetic Mellitus |
| SEM | Standard error of mean |
| TG | Triglycerides |
| UK | United Kingdom |
| WHO | World Health Organization |

ABSTRACT

BACKGROUND : Glycemic index (GI) describes the blood glucose response after consumption of a carbohydrate containing test food relative to a reference food, typically glucose or white bread. Glycemic index was originally designed for people with diabetes as a guide to food selection, advice being given to select foods with a low GI. In Ethiopia information with regard to the glycemic index of commonly consumed traditional foods are not known. Therefore, the current study aims at assessing the glycemic index of some common traditional Ethiopian foods. Thus generating information for the dietary management of diabetes mellitus.

MATERIALS & METHODS: Twelve different traditional Ethiopian foods were randomly selected from the local market and prepared these foods at home following traditional methods. The foods were dried with sun light and oven (<85°C) and then the dried foods were manually grinded and powdered. The powders were kept in clean glasses at room temperature until used for the experiment. Twelve healthy mice (six for control and six for the test group) for each tested foods were used for the study. The mice were divided in to two groups, group 1 is standard (each mouse administered 0.25g of glucose) and group 2 is test (each mouse administered 0.25g of test food). The test food and standard glucose were administered after overnight fasting and the blood glucose were measured at 30 minute intervals for the next two hours (0, 30, 60, 90 and 120minutes). The blood glucose response curve was used to calculate the incremental area under the curve (IAUC) of each food and glucose. Glycemic index of food was calculated as a percentage of incremental area under the curve (IAUC) of each food from standard glucose and expressed as Mean \pm SE of each food.

RESULTS: The result indicated that among the twelve traditional Ethiopian foods eight were found to have low glycemic index ($GI \leq 55$); these include: 1. White teff *enjera*, $GI=35$, 2. Red teff *enjera*, $GI=39$, 3. Maize *enjera*, $GI= 43$, 4. Barley bread, $GI=25$, 5. Qocho bread, $GI=38$, 6. pea sauce, $GI=41$, 7. Chickpeas sauce, $GI=27$, 8. Lentil sauce, $GI=17$, three foods had a moderate glycemic index ($GI=56-69$): 1. Maize bread, $GI= 56$, 2. wheat bread, $GI= 57$, 3. Bulla *genfo*, $GI=60$) and one had a high glycemic index ($GI \geq 70$): 1. White bread, $GI= 73$.

CONCLUSIONS: The examined traditional Ethiopian foods provided important information for the public to guide food choice and could be useful for the prevention of diabetes mellitus.

KEY WORDS: Glycemic index, carbohydrate, blood glucose response, glucose and test food

1. INTRODUCTION

1.1. Carbohydrates

Carbohydrates, often referred to as glycans, play an important role in many biological and biochemical processes, ranging from protein folding to a variety of recognition events, many of which are of immunological importance (Varki *et al.*, 2002; Helenius & Aebi, 2001; Ohtsubo & Marth, 2006). Also carbohydrates (sugars) are organic compounds containing carbon, hydrogen and oxygen with the general formula $C_nH_{2n}O_n$, where, hydrogen and oxygen are present in 1:2 ratio like that in water, i.e. they may be regarded as hydrates of carbons with one molecule of water for each carbon atom, i.e. $C_n(H_2O)_n$. By definition, carbohydrates are aldehyde or ketone (i.e. they have a large number of hydroxyl groups with an aldehyde or a ketone as the main functional group), derivatives of polyhydric alcohols or compounds which yield these derivatives on hydrolysis (Bohne *et al.*, 2001).

Some of the substances are exceptions to the chemical formula but classified as carbohydrates and may not have the above formula, e.g. rhamnose, amino sugars and deoxy sugars, whereas there are some non-carbohydrate compounds which have the same formula as defined for carbohydrates, i.e. $C_nH_{2n}O_n$. The examples include acetic acid (CH_3COOH , $C_2H_4O_2$) and lactic acid ($C_3H_6O_3$) that are not carbohydrates. Carbohydrates constitute the most important domains in protein- and lipid-carbohydrate complexes (glycoprotein's and glycolipids) and participate in the cell-cell interaction and recognition (Helenius & Aebi, 2001).

1.1.1. Classification of carbohydrates

Structurally, "simple" carbohydrates are classified with reference to either the aldotriose glyceraldehyde or the ketotriose dihydroxyacetone and this group containing mostly mono- or oligosaccharides and "complex" carbohydrates when containing mainly polysaccharides or starches. According to the number of sugar units and the size of the molecule, they could be grouped into three types (McWilliams, 2009):

Monosaccharides (simple sugars): They are simplest form of sugars, which cannot be further hydrolyzed. They represent the building units and hydrolytic end products of the more complex carbohydrates.

Oligosaccharides: These are the conjugates of carbohydrates where 2-10 monosaccharide units are linked to each other.

Polysaccharides: These are higher polymers of carbohydrates and contain more than 10 monosaccharide units per molecule.

1.1.2. Importance of carbohydrates

Carbohydrate in the form of glucose is the only sugar used by the body to provide energy for its tissues. Therefore, all digestible polysaccharides, disaccharides, and monosaccharides must eventually be converted into glucose or a metabolite of glucose by various liver enzymes. Because of its significant importance to proper cellular function, blood glucose levels must be kept relatively constant.

Flavor and Sweeteners

A less important function of carbohydrates is to provide sweetness to foods. Receptors located at the tip of the tongue bind to tiny bits of carbohydrates and send what humans perceive as a "sweet" signal to the brain. However, different sugars vary in sweetness. For example, fructose is almost twice as sweet as sucrose and sucrose is approximately 30% sweeter than glucose (Shah & Garg, 2004).

Dietary Fiber

Dietary fibers such as cellulose, hemicellulose, pectin, gum and mucilage are important carbohydrates for several reasons. Soluble dietary fibers like pectin, gum and mucilage pass undigested through the small intestine and are degraded into fatty acids and gases by the large intestine. The fatty acids produced in this way can either be used as a fuel for the large intestine or be absorbed into the bloodstream. Therefore, dietary fiber is essential for proper intestinal health. In general, the consumption of soluble and insoluble fiber makes the elimination of waste much easier. Since dietary fiber is both indigestible and an attractant of water, stools become large and soft. As a result, feces can be expelled with less pressure so fiber helps to prevent constipation (Brisson & Carver, 2001).

Biological Recognition Processes

Carbohydrates not only serve nutritional functions, but are also thought to play important roles in cellular recognition processes. For example, many immunoglobulins (antibodies) and peptide hormones contain glycosyl sequences and are composed of amino acids linked to carbohydrates. During the course of many hours or days, the carbohydrate polymer linked to the rest of the protein may be cleaved by circulating enzymes or be degraded spontaneously. The

liver can recognize differences in glycosyl length, composition and may internalize the protein in order to begin its own degradation. In this way, carbohydrates may mark the passage of time for proteins (Chatwell *et al.*, 2008).

1.1.3. Digestion of carbohydrates

The major energy carrying components in human diet are starches, sugars, fats and proteins, often referred to as macronutrients. These components need to be hydrolyzed in to smaller molecules in the human gastrointestinal tract (GIT) before they can be absorbed and further metabolized in the rest of human body (Bender, 1997). Most of the carbohydrates in the food stuffs are complexes with proteins, lipids or even nucleic acids to form the cellular matrix of the ingested food. According to digestion of foods dietary carbohydrate can be divided in to three:

Ready-to-absorb carbohydrates: The carbohydrate molecules, which do not require digestion and are absorbed as such, e.g. monosaccharide's: glucose, mannose, galactose, fructose and pentose's.

Digestible carbohydrates: These include starch, glycogen, maltose, sucrose, and lactose (oligosaccharides and polysaccharides). They are completely digested into their respective monosaccharide's.

Non-digestible carbohydrates: There are still other carbohydrate molecules which cannot be digested in human gastrointestinal tract. The indigestibility of these dietary fibers is primarily due to the absence of specific digestive enzymes. Most of these indigestible carbohydrates are plant polysaccharides like cellulose, pentose's, hemicelluloses, lignin, gums and pectin's. However, they do absorb water, form a gel and increase the bulk of the stool, which gives a laxative effect.

Carbohydrate digestion:

The digestion of carbohydrates begins in the buccal cavity and continues through the stomach and small intestine (Linda, 2012).

1. Carbohydrate digestion in the mouth: The main function of mouth, in terms of digestion and absorption of carbohydrates; are chewing to increase the surface area of the food particles and initiation of starch hydrolysis catalyzed by amylase enzyme in saliva, and

conversion of carbohydrate in to dextrin and maltose. Food particles are swallowed after chewing and propelled to the stomach using the oesophagus.

2. Carbohydrate digestion in the stomach: The acidic condition denatures the amylase enzyme which was introduced in the mouth, rendering it inactive (Bender, 1997). Some carbohydrates are released during this phase as proteins are denatured and uncoiled, which disrupt various bonds. There is no carbohydrate splitting enzyme in the stomach but hydrochloric acid (HCl) hydrolyses the disaccharides, particularly sucrose into glucose and fructose.
3. Carbohydrate digestion in the small intestine: Once food exits the stomach , it enters in to the small intestine which is responsible for most of the starch hydrolysis by amylase enzyme secreted by pancreas and also carbohydrate in the small intestine digested by hydrolases secreted by intestinal mucosal cells. Undigested food particles continue to the large intestine where water is absorbed and bacteria metabolize some undigested carbohydrates. The undigested contents are stored in the rectum prior to evacuation as feces (Bender, 1997).

1.1.4. Absorption of carbohydrates

The carbohydrate digestion is completed in the small intestine and by the time food bolus reaches the end of small intestine, all the complex carbohydrates have been converted into simpler monosaccharide's. These monosaccharide's are almost completely absorbed from the small intestine (Farrell *et al.*, 2010).

Site of absorption: Mainly upper part of small intestine, i.e. jejunum is involved in the absorption of carbohydrates. The rate of absorption decreases down the intestine i.e. proximal jejunum absorbs much more effectively than the distal portion. A very small amount is absorbed in the stomach or large intestine.

Route of absorption:

The monosaccharide's are absorbed through the mucosal cells into the blood stream and are delivered to the liver by the portal vein chiefly in the form of hexoses (glucose, fructose, mannose and galactose) and as pentose sugars (ribose). Monosaccharide's are not absorbed at the

same rate. However, monosaccharide's are rarely found in normal diets, rather they are derived by enzymatic digestion of more complex carbohydrates within the digestive tube.

Table 1: The rate of absorption of different monosaccharide's are given below (Cumming & Stephan, 2007).

| Monosaccharide's | Absorption rate |
|------------------|-----------------|
| Galactose | 110 |
| Glucose | 100 |
| Fructose | 43 |
| Mannose | 19 |
| Xylose | 15 |
| Arabinose | 9 |

1.1.5. Carbohydrates providing energy and regulating blood glucose

Glucose metabolism is critical to normal physiological functioning. Glucose acts both as a source of energy and as a source of starting material for nearly all types of biosynthetic reactions. The diagram shows the major players in the regulation and utilization of plasma glucose.

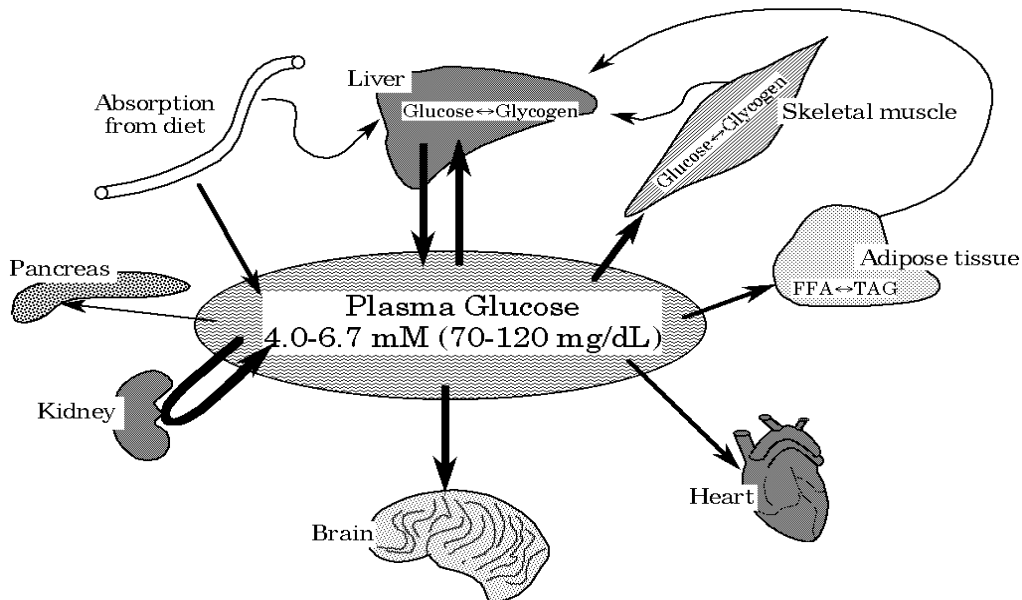


Figure 1: The organs that control blood glucose levels (Stryer, 2003).

The **brain** uses about 60-70% of the total body glucose metabolism. The brain has little stored glucose, and no other energy stores. The brain cannot use fatty acids for energy (fatty acids do not cross the blood brain barrier in significant amount rapidly enough to be used as a fuel for neurons), LCFAs can easily penetrate the blood brain barrier and astrocytes metabolize LCFAs by beta-oxidation. Therefore, fatty acids can gain access to neurons; but due to the low level of enzyme in the neurons for beta-oxidation of fatty acids and anti-oxidant enzyme in the brain, fatty acids take long time for the production of ATP than glucose and ketone bodies, due to these reason ketone bodies can enter the brain and can be used for energy in emergencies. The brain can only use glucose or under conditions of starvation, ketone bodies (acetoacetate and hydroxybutyrate) for energy (Pocai *et al*, 2005).

The **liver** is the major metabolic regulatory organ. About 90% of all circulating glucose not derived directly from the diet comes from the liver. The liver contains significant amounts of stored glycogen available for rapid release into circulation, and is capable of synthesizing large quantities of glucose from substrates such as lactate, amino acids, and glycerol released by other tissues. In addition to controlling plasma glucose, the liver is responsible for synthesis and release of the lipoproteins that adipose and other tissues use as the source of cholesterol and free fatty acids (Stryer, 2003). During prolonged starvation, the liver is the source of both glucose and the ketone bodies required by the brain to replace glucose. The liver uses glycolysis primarily as a source of biosynthetic intermediates, with amino acid and fatty acid breakdown providing the majority of its fuel.

Like the liver, the **kidney** has the ability to release glucose into the blood. Kidney function is critical for glucose homeostasis for another reason; plasma glucose continuously passes through the kidney and must be efficiently reabsorbed to prevent losses.

The **muscle** cannot release glucose into circulation; however, its ability to rapidly increase its glucose uptake is critical for dealing with sudden increases in plasma glucose. Skeletal muscle has an additional role in maintaining plasma glucose levels: it releases free amino acids into circulation to serve as substrates for liver gluconeogenesis. The muscle can use glucose, fatty acids, and ketone bodies for energy (Berteau & Stenutz, 2004).

The **adipose tissue** is the major site of fatty acid storage. Fatty acids are stored in the form of triacylglycerol, which is synthesized in the adipose tissue from glycerolphosphate and free fatty acids. The glycerol-phosphate used must be derived from glycolysis in the adipose tissue. In

conditions when liver gluconeogenesis is necessary the adipose tissue supplies free fatty acid and glycerol to the circulation to be taken up by the liver as substrate (Weickert & Pfeiffer, 2006).

Finally, the **pancreas** is the source of insulin and glucagon, two of the most important metabolic regulatory hormones. During periods of food consumption, pancreatic beta cells sense the rise in blood glucose and begin to secrete the hormone insulin. Insulin binds to many cells in the body having appropriate receptors for the peptide hormone and causes a general uptake in cellular glucose. In the liver, insulin causes the uptake of glucose as well as the synthesis of glycogen (Cheatham & Kahn, 2003).

In contrast, the hormone glucagon is secreted into the bloodstream by pancreatic alpha cells upon sensing falling levels of blood glucose. This hormone inhibits the uptake of glucose by muscle and other cells, promotes the breakdown of glycogen in the liver and promotes gluconeogenesis, a process involving the synthesis of glucose from amino acid precursors (Phillipe, 2001). Through the effects of both glucagon and insulin, blood glucose can usually be regulated in concentrations between 70 and 125mg/100 ml of blood.

1.2. Glycemic index, Insulin index and Glycemic load

Glycemic index (GI); the index measures how quickly and how much glucose levels rise in the blood. In fact, the word "glycemic" means glucose in the blood. GI is an empirical system for classifying carbohydrate-based foods, founded on the degree of glucose release into the blood stream once ingested (Ludwig, 2007). Foods with a high GI break down, are absorbed and cause a rise the blood glucose quickly in the body. The low GI foods, have slower break down, absorption and cause a slower rise the blood glucose. The concept of GI is based on both chemical composition and physiological properties of carbohydrates containing foods (Englyst, Lui & Englyst, 2007).

Glycemic index ranks foods on a scale from 0 to 100. All carbohydrate-containing foods are categorized under high, moderate, and low depending on the numerical value derived from foods. Individual foods with low glycemic index ($GI \leq 55$) release glucose more slowly over several hours. Moderate index foods are between 56 and 69. Foods with high glycemic index ($GI \geq 70$). The GI concept has been developed to obtain a numerical physiological classification of carbohydrate containing foods and meals based on the rate of carbohydrate absorption into the blood, to improve nutritional advice (Laville, 2004; Jenkins *et al.*, 1981).

Insulin Index

Insulin index, which is a measure of the insulin response to foods. This index is used particularly for low-carbohydrate foods with high protein or fat content. The insulin index was calculated from the ratio of the IAUC of the insulin response curve of test food and the same amount of reference food (mean IAUC of two reference glucose or white bread) expressed as a percentage. Food insulin index, measures postprandial increase in insulin secretion of a whole food, and dependent on carbohydrate, quantity and quality of protein and fat and their interactions.

Lean meat, for example, has a low glycemic index because it contains very little carbohydrate, but the amino acids from the meat's protein digestion stimulate insulin secretion by the pancreas and this could contribute to possible harmful effects of meat on insulin metabolism. However, carbohydrates are not the only stimulus for insulin secretion. Especially in combination with carbohydrates, protein and fat act synergistically to enhance insulin secretion and reduce blood glucose concentrations (Ngo *et al.*, 2004).

Glycemic load

The glycemic load (GL) of food is a number that estimates how much the food will rise a blood glucose level after eating it. Glycemic load accounts for how much total carbohydrate is in the ingested food and how much each gram of carbohydrate in the food rises blood glucose levels, so that GL indicates both quality and quantity of carbohydrate consumed in terms of glycemic response. But GI compares equal quantities of foods and provides a measure of carbohydrate quality in terms of glycemic response but not quantity (Das & Gilhooly *et al.*, 2007).

In 1997 the concept of GL was introduced by researchers at Harvard University to quantify the overall glycemic effect of a portion of food. Thus, the GL of a typical serving of food is the product of the amount of available carbohydrate in that serving and the GI of the food. The higher the GL, the greater the expected elevation in blood glucose and in the insulinogenic effect of the food. The long-term consumption of a diet with a relatively high GL (adjusted for total energy) is associated with an increased risk of type 2 diabetes and coronary heart disease. Glycemic load gives a relative indication of how much that serving of food is likely to increase your blood-sugar levels.

1.3. Glycemic index of foods

The following table provides the glycemic index (GI) values of selected foods. Revised International Table of Glycemic Index (GI) value by David Mendosa (2008). The reference food for this table is glucose.

Table 2: The mean glycemic index (GI) of common foods derived from multiple studies by different laboratories (David, 2008).

| SOME COMMON CARBOHYDRATE FOODS | PERCENTAGE (%) OF GLYCEMIC INDEX (GI) |
|---------------------------------------|--|
| Breads & Grains | |
| White wheat bread | 75±2 |
| Whole wheat/whole meal bread | 74±2 |
| Speciality grain bread | 53±2 |
| Unleavened wheat bread | 70±5 |
| Wheat roti | 62±3 |
| Chapatti | 52±4 |
| Corn tortilla | 46±4 |
| White rice, boiled | 73±4 |
| Brown rice, boiled | 68±4 |
| Breakfast Cereals | |
| Cornflakes | 81±6 |
| Wheat flake biscuits | 69±2 |
| Porridge, rolled oats | 55±2 |
| Instant oat porridge | 79±3 |
| Rice porridge/congee | 78±9 |
| Millet porridge | 67±5 |
| Muesli | 57±2 |
| Fruit and fruit products | |
| Apple, raw | 36±2 |

| | |
|--|------|
| Orange, raw | 43±3 |
| Banana, raw | 51±3 |
| Pineapple, raw | 59±8 |
| Mango, raw | 51±5 |
| Watermelon, raw | 76±4 |
| Peaches, canned | 43±5 |
| Strawberry jam/jelly | 49±3 |
| Apple juice | 41±2 |
| Orange juice | 50±2 |
| Vegetables | |
| Potato, boiled | 78±4 |
| Potato, instant mashed | 87±3 |
| Potato, french fries | 63±5 |
| Carrots, boiled | 39±4 |
| Sweet potato, boiled | 63±6 |
| Pumpkin, boiled | 64±7 |
| Plantain/green banana | 55±6 |
| Vegetable soup | 48±5 |
| Dairy products and alternatives | |
| Milk, full fat | 39±3 |
| Milk, skim | 37±4 |
| Ice cream | 51±3 |
| Yogurt, fruit | 41±2 |
| Soy milk | 34±4 |
| Rice milk | 86±7 |
| Legumes | |
| Chickpeas | 28±9 |
| Kidney beans | 24±4 |
| Lentils | 32±5 |
| Soya beans | 16±1 |

| | |
|-----------------------|-------|
| Snack products | |
| Chocolate | 40±3 |
| Popcorn | 65±5 |
| Potato chips | 56±3 |
| Soft drink/soda | 59±3 |
| Rice crackers/crisps | 87±2 |
| Sugars | |
| Fructose | 15±4 |
| Sucrose | 65±4 |
| Glucose | 103±3 |
| Honey | 61±3 |

1.4. Significance of glycemic index

Carbohydrate foods are important for providing the steady fuel supply. Monitoring energy and blood glucose levels is especially important for athletes and people with health implications, such as diabetes mellitus. Due to the importance of balancing blood glucose levels in diabetics, low to moderate glycemic foods are important to control blood glucose in these individuals (Ruth & Diane, 2003).

For athletes CHO-loading regimens prior to endurance exercise, total carbohydrate is a much more important consideration than the GI of the foods/diets chosen. For the food prior to endurance exercise, there appears to be competitive advantage to selecting low-to-moderate GI foods, because low GI foods caused higher fat oxidation rates (Stevenson *et al.*, 2006). Increasing fat oxidation spares CHO thus making the fuel supply last longer. In addition, ingestion of low to moderate GI foods increased the availability of nonessential fatty acids during exercise and decreased the reliance on intramuscular lipid during moderate intensity exercise (Trenell *et al.*, 2008). There was also a slight but limited advantage of a higher GI diet or food for replenishing glucose by restore muscle glycogen after exercise (Dickinson *et al.*, 2008 and Chen *et al.*, 2008).

1.5. Low glycemic index (GI) and health benefits

In 1998 the United Nations FAO/WHO Expert Consultation Group on carbohydrates in human nutrition recommended the use of the GI to classify CHO as low, moderate or high GI and in making food choices. Low GI diet most of which should come from foods that are rich in non-starch polysaccharides (NSP). This is due to the fact that diets that are rich in non starch polysaccharides as components of slowly digested CHO assist in disease prevention, especially the prevention of diabetes mellitus, and other chronic diseases. However, foods should not only be chosen on account of their GI, as some foods might have a low GI, but a high fat content. Therefore, the total amount of CHO, amount & type of fat, fiber, micronutrient of foods should also be considered in deciding whether a food is a healthy choice or not (Pawlak *et al.*, 2004).

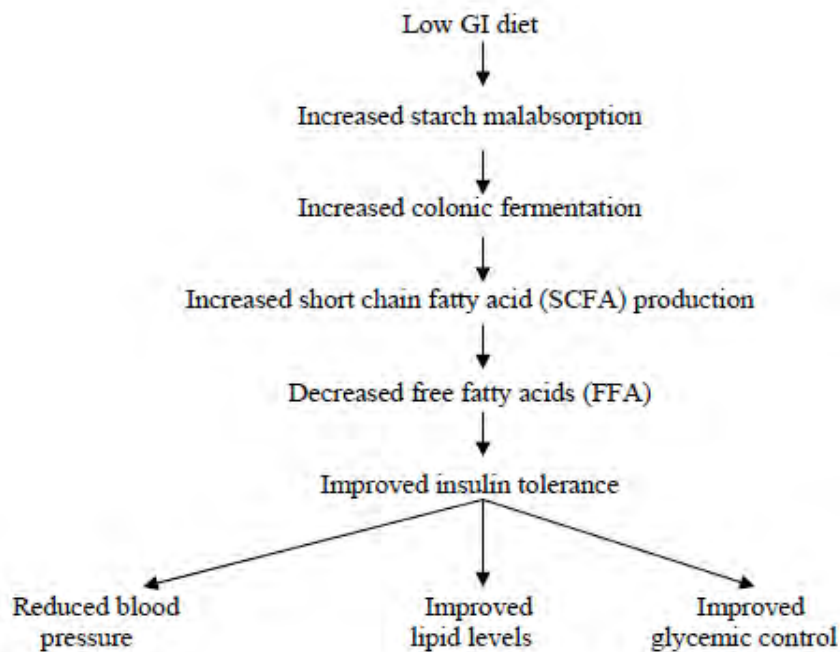


Figure 2: Advantages of a low GI Diet (Frost *et al.*, 1994).

1.5.1. Low glycemic index and diabetes

Diabetes mellitus is a chronic polygenic syndrome with impaired carbohydrate metabolism. The carbohydrate metabolism is impaired due to deficiency or ineffectiveness of insulin (peripheral insulin resistance) or decreased insulin/anti-insulin ratio leading to chronic hyperglycemia and glycosuria. The high blood glucose levels observed in diabetes can be due to higher rates of glucose appearance in the blood stream. The GI of the diet was positively associated with and the best predictor of the risk of developing type 2 diabetes, after adjustment

for factors such as age, BMI, physical activity, family history, smoking, alcohol consumption and total energy intake. It also showed that insulin resistance increased the risk of type 2 diabetes (Sievenpiper *et al.*, 2002).

However, since foods naturally low in GI are often high in fiber and other nutrients are important to avoid the risk factors for developing diabetes such as insulin resistance and obesity by enhancing insulin sensitivity and controlling body weight by decreasing fat storage, so the GI of the food or diet may simply flag a dietary choice that helps to reduce risk of diabetes (T2DM). This concluded that due to the importance of balancing blood glucose levels in diabetes, low to moderate glycemic foods are important to moderate blood glucose in these individuals (Sartorelli & Cardoso, 2006).

1.5.2. Low glycemic index and obesity

The prevention of weight gain in adult life suggested that consumption of diets with low in fat and high in a protein and complex CHOs helped prevent weight gain. Obesity and insulin resistance are risk factors for diabetes and CVD. Obesity and sedentary behavior are also associated strongly with increase insulin requirement, as inactivity causes insulin insensitivity in muscle tissue, so obesity associated with hyperinsulinemia, insulin resistance and type 2 diabetes (Brand-Miller *et al.*, 2002; Lineback, 2005). Low GI foods may benefit weight control in the following ways:

1. **By promoting fat oxidation at the expense of CHO oxidation:** High GI foods yielded lower rates of fat oxidation than low GI foods of similar composition and some evidence supports the hypothesis that the chronic hyperglycemia and hyperinsulinemia induced by high GI diets, can reduce the body's ability to oxidize fat, leading eventually to a significant increase in body fat storage. But low GI foods to induce the fat oxidation as well as lower rates of hepatic lipogenesis and lower liver and muscle glycogen stores (Liu *et al.*, 2000).
2. **By enhancing satiety:** Low GI foods are also generally more satiating than high GI foods and Holt and Brand-Miller (1994) have shown that CHO foods generally have a greater satiety value than fat. According to Lavin *et al.*, (1996), the higher satiety induced by the consumption of low GI foods, can be attributed to the fact that nutrient receptors in the small intestine of the gastrointestinal tract are stimulated for a longer period of time, as these foods are digested and absorbed at a slower rate, which leads to prolonged

feedback to the satiety center in the brain, through signals such as cholecystokinin and Glucagon-like Peptide-1.

1.5.3. Low glycemic index and hypoglycemia

Hypoglycemia is an abnormally low concentration of glucose in the blood. This occurs when a person has eaten too little food, or exercised without food or if they have diabetes and have injected too much insulin. Taking a small amount of a sugar or food with sugar usually helps the person feel better within 10-15 minutes. To prevent hypoglycemia the low glycemic diet is important because it allows slow digestion and absorption of glucose in to the blood stream, thus making the glucose supply last longer. It is important not to eat high glycemic foods as they can trigger an over production of insulin (Jenkins *et al.*, 1981). High insulin levels usually cause a drop in blood glucose and can lead to a hypoglycemic episode. Regular exercise is crucial to help maintain healthy insulin response and healthy blood glucose levels. Kaufman (1999) showed that episodes of nocturnal hypoglycemia were reduced when lower GI foods were incorporated into the diet.

1.5.4. Low glycemic index and blood lipids, markers of coronary risk factors

Dickinson and Brand-Miller (2005) posit that the average dietary GI predict cardiovascular disease risk factors, including HDL cholesterol, TGs and markers of inflammation, therefore they are related to coronary heart disease. Several mechanisms have been suggested as ways that low GI diets can decrease coronary risk factors. First, they can decrease numbers of atherogenic small, dense LDL particles and help to maintain high levels of HDL, primarily by mechanisms that involve decreasing plasma TG concentrations (Siri & Krauss, 2005). Second, they also decrease chylomicron production (Lairon, 2008). Third, by decreasing markers of inflammation such as C-reactive protein.

In one study the data showed that the Canadian Trial of Carbohydrates in Diabetes, a low GI diet did not improve blood glucose, but it did lower the level of C-reactive protein (CRP) (Wolever *et al.*, 2008). Generally, markers of inflammation are strongly correlated with coronary disease, then dietary patterns (low GI) that reduce CRP and other pro-inflammatory substances may be an important aspect of prevention. Low GI concept show that diets moderate in fat and low in saturated fat and CHOs with low GI are important for controlling complications of coronary heart disease and other cardiovascular disease.

1.6. High glycemic index and health hazards

This high glycemic index foods, which means they are rapidly digested and quickly enter to the blood stream as a surge of blood glucose. Surges of blood glucose result in corresponding surges of blood insulin that promote fat storage, increase metabolic syndrome risks, promote the development of type 2 diabetes and promote a certain cancer (Ludwig, 2002). People who eat a lot of high glycemic index foods tend to have greater levels of body fat, as measured by the body mass index (BMI). High BMIs are linked to obesity, heart disease, and diabetes.

1.6.1. High glycemic index and diabetes, insulin resistance

Diabetes mellitus is a chronic polygenic syndrome with impaired carbohydrate metabolism. Diabetes mellitus is characterized by the lack, or relative lack of insulin. This has the effect of increasing blood glucose levels as uptake of glucose by cells is inhibited. The insulin resistance syndrome, or metabolic syndrome (MetS) is a cluster of metabolic abnormalities characterized by high levels of circulating insulin, high blood glucose, high blood pressure, central (visceral) obesity, low HDL cholesterol, high triglycerides (TG), high small, dense LDL particles, elevated markers of inflammation and abnormal fibrinolysis (Feldeisen & Tucker, 2007).

Foods with a high glycemic index break down and send glucose quickly in to the blood. The higher the index, the faster glucose is released. Initially insulin levels are very high, but peripheral insulin resistance and increased hepatic gluconeogenesis make insulin levels inadequate to normalize plasma glucose levels. Insulin production then falls, further exacerbating hyperglycemia. Hyperglycemia itself may impair insulin secretion, because high glucose levels desensitize β -cells, cause β -cell dysfunction (glucose toxicity), or both. These changes typically take years to develop in the presence of insulin resistance (Kim *et al.*, 2008). These leads to NIDDM, so early epidemiological studies associated high GI diets with the development of type 2 diabetes mellitus (T2DM) due to insulin resistance (metabolic syndrome).

1.6.2. High glycemic index and coronary heart disease

Coronary Heart Disease (CHD) ranks number one as the cause of death in the world wide. Therefore, dietary strategies to prevent and manage this disease are of great interest. Dietary components including the amount and quality of the carbohydrate (CHO) and fat are the major factors for the development of CHD. GI has effects on both postprandial hyperglycemia and postprandial insulinemia, both of which are independent risk factors for CVD. High GI foods are

positively associated with serum triglycerides (TG) and LDL cholesterol and also inversely associated with HDL cholesterol. Particularly these high plasma LDL cholesterol and low HDL levels cause atherosclerosis (Ludwig, 2002). Atherosclerosis affects most or all arteries, but some are more medically important: Coronary arteries (arteries feeding the heart tissue): myocardial ischemia and myocardial infarction, these leads to coronary heart disease.

If the quality of CHO would impact CHD risk if it was substituted for a known coronary risk factor, saturated fats (SF). These indicates that when CHOs with high-GI values were substituted for SF, the risk of myocardial infarctions (MI) increased. If CHOs with low-GI values were substituted, there was a non-significant inverse association between GI and risk of MI, and if CHOs with medium-GI values were substituted, there was no measurable effect (Jakobsen *et al.*, 2010).

1.6.3. High glycemic index and cancer

Since 2000, there have been a number of studies looking at the relationship between CHO intake, GI and cancer risk. It has been postulated that because insulin affects levels of insulin-like growth factor (IGF), and IGF promotes growth in normal and malignant tissues, that GI might be associated with cancers. This review will look at the relationship between various forms of cancer and GI. For example, pancreatic, prostate, and endometrial cancers.

Pancreatic Cancer

Epidemiological evidence strongly suggests that glucose intolerance and T2DM are risk factors for pancreatic cancer, thus some predicted that GI or GL would be a risk factor for pancreatic cancer. So high GI diet are positively associated with the development of T2DM, due to these consumption of high GI diets has been associated with Hyperinsulinemia (Holt *et al.*, 1997). Insulin is associated with the activation of mitogenic signals that stimulate pancreatic cell proliferation (Gupta, *et al.*, 2002).

High glycemic index diets are associated with increased insulin secretion, which has been shown to promote pancreatic cancer cell growth in vitro (Byrnes *et al.*, 1995) and to affect pancreatic cancer risk by several mechanisms, including alteration of cell cycle kinetics (insulin facilitates the transit of cells through the G1 phase of the cell cycle) (Gross *et al.*, 1984), inhibition of apoptosis, and down-regulation of insulin-like growth factor binding protein 1 (IGFBP-1) (Musey *et al.*, 1993). Therefore, high GI diets are positively associated with pancreatic cancer.

Prostate Cancer

Insulin may play a role in prostate cancer tumorigenesis. Postprandial blood glucose and insulin responses of foods depend importantly on the carbohydrate quality and quantity. There is good evidence that obesity and perhaps, a high fat intake, due to its strong correlation with obesity, increase the risk of high-grade or aggressive prostate cancer and prostate cancer mortality. High glycemic index diets are associated with increased insulin secretion, which has been shown to promote prostate cancer (Ramon *et al.*, 2000). This high circulating insulin has been shown to play a role in prostate cancer tumorigenesis by inhibiting apoptosis and stimulating cell proliferation. In addition to this insulin may alter tumor development through alterations in sex-hormone metabolism and by influencing the insulin-like growth factor (IGF) axis, i.e. increasing bioactivity of IGF-1, partly by reducing IGF binding protein levels. High circulating IGF-1 was consistently associated with moderately increased risk of prostate cancer (Key *et al.*, 1997).

The effect of carbohydrates on blood glucose and insulin levels depends on the carbohydrate quality, which can be characterized by the diet's glycemic index (GI). However, carbohydrates are not the only stimulus for insulin secretion. Especially in combination with carbohydrates, protein and fat act synergistically to enhance insulin secretion and reduce blood glucose concentrations (Ngo *et al.*, 2004). Generally that a diet characterized by high intake of rapidly absorbable carbohydrates represented by high GI/GL, is associated with greater risk of prostate cancer.

Endometrial cancer

Endometrial cancer is the 5th most common cancer among women worldwide. Insulin resistance may play a role in the etiology of endometrial cancer based on the observation that several risk factors for endometrial cancer including adiposity, low physical activity, elevated levels of blood glucose, and diabetes are also linked to insulin resistance. Carbohydrates are the main dietary component affecting an individual's insulin secretion, therefore glycemic index is also relevant one of the risk factor for the development of endometrial cancer (Ferlay *et al.*, 2010). Diets with high glycemic index (GI) are characterized by fast absorption of their carbohydrate component, with consequent rises in glucose and insulin levels. Although estrogens are the main endometrial cancer risk factors, insulin may disrupt sex hormone balance.

Hyperinsulinemia has been shown to increase ovarian steroid production, to stimulate conversion of testosterone into estradiol (Poretsky, 2003 and Garzo, 1998) and to suppress sex hormone binding globulin (SHBG), thereby increasing estradiol bioavailability (Nestler *et al.*, 2001). Insulin has been shown to act as a cancer promoting agent and it has also affinity for the insulin-like growth factor (IGF) receptor, thereby increasing levels of IGFs (Klotz *et al.*, 2000). Estrogens may also stimulate IGF-1 synthesis. IGF-1 has been shown to stimulate mitogenesis in endometrial cancer cell lines and may therefore, have a role in endometrial carcinogenesis (Ayabe *et al.*, 1997). Therefore, there are direct association between dietary GI and endometrial cancer risk.

Generally, the basic concept is that a higher rate of carbohydrate absorption leads to higher blood glucose and insulin rise, and hence a higher GI index. Foods with higher glycemic indexes are a risk factors for diabetes, obesity, cardiovascular disease, and cancer.

1.7. Factors influencing the glycemic response to foods and the variability of the glycemic index of a foods

The same amount of different CHO does not necessarily cause the same glycemic response. The difference in the GI values of various foods may be explained by several factors. It would appear that the rate of absorption as well as digestibility plays a major role in determining the GI of a particular food.

Table 3: Factors that affect glycemic responses and GI of foods [adapted from FAO/WHO 1998].

| Factors | Specific food component | Effect on GI | Researchers |
|----------------------|--------------------------------|--|---------------------|
| Amount of CHO | Not applicable (N/A) | Postprandial blood glucose and insulin levels ↑ with an ↑ in the amount of glycemic CHO. | Lineback, 2005 |
| Nature of the Starch | Amylose and amylopectin | The linear nature of amylose starch causes it to create compact bundles that exclude water, making it resistant to enzymatic attack. High amylose starches also retrograde more readily on processing, making the starch molecule even more resistant to hydration and enzymatic attack. The branched nature of amylopectin starch causes it to be more open, to hydrate | Jenkins et al, 1995 |

| | | | |
|-----------------------------------|-------------------------|---|--|
| | | easily and to be more susceptible to enzymatic attack. | |
| Cooking/foodprocessing | Not applicable (N/A) | Main effect on starches is disruption of the cellular architecture and fibrous structure of starch granules, causing the starch to gelatinize more easily, but some starch granules disrupt more easily than others. Hydration during cooking also plays a major role in ↑starch digestibility. | Frost et al, 1994; Holt et al, 1994); Jenkins et al, 1995 |
| | Particle size | Intact whole grains usually cause ↓ glycemic responses. When the cereal grains involves milling or grinding, the rate of digestion and absorption is ↑ due to ↓ particle size and thus the glycemic and insulinemic responses will also be ↑ and satiety ↓. In many plant foods (e.g. minimally processed legumes and cereal grains) the encapsulation of starch and sugars within cell walls slows down the digestion and absorption of the starch/sugars therein. | Englyst et al, 2003 ; Holt et al, 1994 |
| | Cellular structure | The cell walls of tropical fruits are softer, more easily digested, release sugars more quickly and have higher GI values than temperate climate fruits, which have tougher cell walls and lower GI values. | Brand Miller et al, 2002 |
| Acidity or organic acids or salts | | High acidity slows down the rate of gastric emptying and therefore ↓ the glycemic response to such a food or meal, as well as its GI. Bread products that contain sodium propionate (e.g. sourdough bread) ↓ blood glucose and insulin responses in healthy individuals, probably due to ↓ gastric emptying rate. | Foster-Powell et al, 2002; Liljeberg et al, 1998 |

1.8. Statement of the problem

The foods with low glycemic index may be beneficial in the management of diabetes mellitus and other chronic disease such as obesity, cardiovascular disease, coronary heart disease and cancer. Physician and health professionals should be equipped with proper knowledge about the glycemic index of foods in order to give proper advice to their respective patients. However, in Ethiopia the glycemic index of traditional prepared foods are not known. Due to this physician and health professionals simply advise the patient without having such information. Therefore, the current study was undertaken to fill these information gap.

1.9. Significance of the study

The glycemic index (GI), first proposed in 1981, is a system of classifying food items by glycemic response. Over the past 35 years, low-GI diets have been associated with decreased risk of cardiovascular disease, type 2 diabetes, metabolic syndrome, stroke, depression, chronic kidney disease, formation of gall stones, neural tube defects, formation of uterine fibroids, and cancers of the breast, colon, prostate, and pancreas. Taking advantage of these potential health benefits can be as simple as sticking with whole, natural foods that are either low or very low in their GI value. The study is important for diabetes, metabolic syndrome (Insulin resistance), athlete and weight management.

To bring literature surveys showing that there is little or no knowledge about the glycemic index of traditional Ethiopia foods. Therefore, the aim of this study was to explore the pattern of glycemic index of commonly consumed traditional Ethiopian foods, so that it may generate some useful information for health professionals during the management of certain disease such as diabetes mellitus.

2. OBJECTIVES

2.1. General objective

- To investigate the glycemic index values of selected traditional Ethiopian foods in mice.

2.2. Specific objectives

- To select some commonly consumed traditional Ethiopian foods.
- To set the proximate nutritional composition of test foods.
- To measure blood glucose concentrations of mice with 30 minutes interval for 2 hrs.
- To determine the glycemic index (%) of selected foods.
- To classify the twelve traditional Ethiopian foods as low, moderate and high glycemic index based on their glycemic response.

3. MATERIALS AND METHODS

3.1. Study design

This study the laboratory experiment involving quantitative and qualitative (descriptive) analysis of data.

3.2. Study area

The study was carried out in Addis Ababa University, College of Health Sciences, and Biochemistry, EPHI and Animal laboratories at Black Lion Specialized Hospital.

3.3. Test foods

Twelve different traditional Ethiopian foods were randomly selected from the local market and the traditional way of preparing these foods were done at home. These includes;

1. Cereal based foods:

Enjera-;

Teff , *Eragrostis tef* (Zucc.) Trott., white enjera & red/brown enjera, and Maize (Corn), *Zea mays L.*, enjera.

Breads-;

Barley, *Hordeum vulgare L.*: bread,

Wheat, *Triticum vulgare Vill.*, bread (traditional *diffo dabo*),

Maize (Corn), *Zea mays L.*, bread (traditional *diffo dabo*), and

White bread (bread from bakery).

2. Root and root based products:

False banana, *Ensete ventricosum.*, Bulla genfo and Qoch'ō bread.

3. Legume based food products:

Pea, *Pisum sativum L.*, Sauce (*Ater Shiro wet*),

Chickpea, *Cicer arietinum L.*, Sauce (*Shimbra Shiro wet*), and

Lentil (split), *Lens culinaris Med.*, Sauce (*Missir wet*).

3.4. Processing of raw materials

3.4.1. Preparation of cereals:-All cereals collected from local market were cleaned and milled in local mills. But white breads were collected from the local bakery.

3.4.1.1. Preparation of enjera [Teff (white and red/brown enjera), and Maize (Corn) enjera]

Measurement and ingredients-; Teff flour, warm water, Maize flour, large bowl and pan.

Preparation method; The teff or maize flour and water mixed with starter culture (a batter from a previous fermentation (Ersho). The mixture was kneaded for 5-10 minutes. The dough was left to ferment for 2 days at room temperature. The yellowish liquid at the top of the dough was discarded after 2 days fermentation. After the liquid is discarded some amount of the dough was boiled (locally known as „absit“). After the absit was cooled it was mixed with the rest of the dough. Then it was left to ferment once again and it took at least 1hr to rise, at room temperature until foam and bubbles were formed. Pour about 500ml of the batter in a circular manner on 50cm diameter hot clay griddle “*mitad*” and bake covered for about 2minutes. Removed the enjera by lifting it off the hot griddle and sliding over a straw mat.

3.4.1.2. Preparation of breads:- [Wheat, Barley, and Maize (Corn) breads]

Measurement and ingredients-; cups of wheat, barley/ maize flour, sugar, commercial yeast, salt, bowl, oil, water and pan,

Preparation method; Combined cups of flour, salt, yeast, and oil in a large mixing bowl. Stir to blend. In a saucepan, heat the sugar and water until very warm. Pour into dry ingredients and mixed for 2 minutes. Stir in enough of the remaining flour to made a soft dough. Transfer dough to a lightly floured surface and kneaded until smooth and elastic. Shape into a ball and placed in a greased bowl. Covered loosely with a towel and let rise until double. Then the dough was kneaded to get rid of the big bubbles and made it uniform. It was placed in loaf pan and allowed to rise again for another hour until it fills the loaf pan. It was baked in an oven preheated to 150-175 °C for about 45minutes until golden brown. Removed from pans and finally to get bread.

3.4.2. Preparation of legumes-: All legumes collected from the local market were cleaned and milled in local mills.

3.4.2.1. Preparation of pea and chickpea sauce

Measurement and ingredients -; pea/ chickpeas powder, onion, cup of vegetable oil, garlic, *berbere*, salt and water.

Preparation method; Placed the onion and garlic in a food processor or in medium pot. Added a little water and heat the oil, saucepan over medium flame. Then *berbere* and simmer added for about few minutes at low heat and put in a dash of water to avoid sticking, until the excess moisture evaporates and the onion loses its raw aroma, about 5-10 minutes. Add the water and mixed the *shiro* by adding a small portion of *shiro* flour at a time and continuously stirring.

Enough amount of salt added and let it cooked until it became thick, but runny for about 30-40 minutes at low heat. Finally to get pea/ chickpea sauce.

3.4.2.2. Preparation of lentil sauce (*Missir wet*)

Measurement and ingredients -; Split red lentil, onions, vegetable oil, garlic, *berbere*, salt and water.

Preparation method; Placed the onion and garlic in a food processor or in medium pot. Added a little water and heat the oil, saucepan over medium flame. Then *berbere* and simmer added for about few minutes at low heat and adding a dash of water to avoid sticking, until the excess moisture evaporates and the onion loses its raw aroma, about 5-10 minutes. Add the water and mixed lentils to the saucepan. Enough amount of salt added and let it cooked and simmer until lentils are cooked through and fall apart, about 40 to 55 minutes at low heat. Added the water to keep the lentils from drying out. Finally to get lentil sauce.

3.4.3. Preparation of root and root product (qocho bread and bulla *genfo*)

All root and root based products collected from local market were manually scraping and squeezing.

Preparation method of qocho bread-; Qocho powder was mixed with water. The mixture was kneaded for 5-10 minutes. This dough was formed into flatbreads by wrapping it in a thin layer of ensete leaves and these dough was baked on a griddles (*mitad*).

Preparation method of bulla *genfo*-; To prepare bulla *genfo* added enough amount of bulla flour to cup of boiling water in a medium pan. Stir it all up and when it was smooth, added a little salt and let it cooked, about 15 to 25 minutes at medium heat. Removed from the pan, finally to get bulla *genfo*.

3.5. Inclusion criteria

3.5.1. Gender: Male and female mice.

3.5.2. Part of the study: healthy mice [Fasting blood glucose value (at time 0) between 70-126mg/dl].

3.5.3. Mice that were used for GI tests should be studied in the morning at breakfast time, after an overnight fast of 10–14hrs on separate days.

3.5.4. When to calculating IAUC, if blood glucose value fell below the baseline, only the area above the fasting level was included.

3.6. Exclusion criteria

3.6.1. Diagnosis of diabetes mellitus (DM).

3.6.2. Use of medications or nutritional supplements known to affect glucose metabolism.

3.6.3. For the purpose of statistical evaluation all tests that were not complete and all tests where the first (i.e. fasting) blood-glucose concentration was 126mg/dl or higher were excluded.

3.7. Experimental design

3.7.1. Experimental animals

Adult male Swiss albino mice aged between 12 and 14 weeks were used for the study. Mice were purchased from Ethiopian Health and Nutrition Research Institute (EHNRI). All the mice were acclimatized to the laboratory condition for one week before commencing the experiments and fed with pellet and free access of water. The animals were housed in 12 hours light and dark cycle at room temperature. The experiment was performed in the laboratory of Pharmacology Department, School of Medicine, Addis Ababa University after ethical approval obtained from Ethical Committee of the Department of Biochemistry, School of Medicine, Addis Ababa University (protocol number 0022/2013). All animal handling and care was done as per the guidelines set by the national academies press, Washington, D.C., USA.

3.7.2. Experimental protocol

Swiss albino mice were divided in to two groups of six animals each.

Group1. This group was kept as control and animals administered with standard glucose (0.25g).

Group 2. This group was a test and administered with test foods (0.25g).

3.8. Proximate composition analysis

The moisture, total nitrogen, protein, fat, carbohydrate, crude fiber, ash, and mineral contents of test foods were analyzed according to AOAC methods. All samples were collected, prepared using traditional methods and stored in -20°C until analysis. Before analysis, samples were equilibrated to room temperature. Care has been taken for components in the food which can easily undergo chemical changes by exposure to air and light.

Moisture:

Moisture content included free water and volatile substances. Dry matter in the food sample was determined by oven drying method at 105°C for about 12hr, removed and then cooled in a dessicator to a constant weight. The difference in weight was moisture content of the sample

(AOAC, 2005). Foods with high water content were first dried at low temperature (65°C) and further drying was carried out. The moisture content of the sample was calculated using the following equation:

$$\%W = \frac{W1 - W2}{W1} \times 100$$

Where:

%W = Percentage of moisture in the sample

W1 = Weight of original sample (grams)

W2 = Weight of dry sample (grams)

The result expressed as the percentage per gram sample.

Total nitrogen:

Total nitrogen was determined using Kjeldahl procedure (AOAC, 2005). The method consists of three steps: 1. Digestion of the sample in sulphuric acid with a catalyst (0.5g Se and 100g K₂SO₄). The nitrogen contained in the sample is converted to ammonia; ammonium sulphate being formed. 2. Distillation of ammonia released from ammonium sulphate by addition of an excess of sodium hydroxide; ammonia being trapped in a trapping solution (sulphuric acid) and 3. Titrated the contents with sodium hydroxide solution to the endpoint. The result should be expressed in g of nitrogen per 100 g of sample.

Total protein:

In all cases, protein values have been calculated from the nitrogen content as determined by the Kjeldahl procedure multiplied by the factor 6.25. i.e, N × 6.25. This might over estimate the values compared to the use of individual factors.

Fat:

Fat refers to the „Ether extractable“ material. Oven dried sample was extracted in soxhlet apparatus using an hydrous diethyl ether as described in the Official Methods of Analysis, Association of Official Analytical Chemists and as described in the Tecator Manual (AOAC, 2005).

$$\text{Crude fat content} = \frac{W3 - W2}{W1} \times 100$$

Where:

W1 = Weight of original sample (grams).

W2 = Weight of the flask before addition of the sample (grams)

W3 = Weight of the flask after concentration of the sample (grams)

Carbohydrate:

The values given for carbohydrate in this table refers to the” total carbohydrate by difference”. i.e. the difference between 100 percent and the sum of the percentage of moisture, protein, fat and ash. The carbohydrate portion that is supposed to be utilized can be obtained by subtracting the crude fiber from the total carbohydrate.

Crude fiber:

The crude fibre was estimated as the portion of the carbohydrate that resist digestion when boiled in dilute sulfuric acid followed by subsequent boiling with dilute alkali and followed by subsequent washing, drying, weighing and igniting as described in Official Methods of Analysis, AOAC (AOAC, 2005). It is largely cellulose, hemicelluloses and lignin.

$$\text{Crude fiber content} = \frac{W_2 - W_3}{W_1} \times 100$$

Where:

W1= Weight of original sample (grams)

W2= Weight of dry sample (grams)

W3= weight of cooled sample after ignited (grams)

Ash:

Refers to the total inorganic material residue after igniting the sample at 550°C to burn off all the organic matter .

$$\text{Ash content} = \frac{W_3 - W_1}{W_2 - W_1} \times 100$$

Where:

W1 = Weight of empty crucible (gram)

W2 = Weight of crucible and sample (gram)

W3= Weight after ashing (gram)

Minerals:**Calcium (Ca) and Iron (Fe):**

A similar procedure was followed for the determination of Ca and Fe. After removing the organic matter by dry ashing, the residue was dissolved in dilute hydrochloric acid followed by an appropriate dilution and the determination of the element were carried by Atomic Absorption Spectrophotometers at a specific wavelength in a Varian Model 10/20 Spectra A. (AOAC, 2005). For Ca determination, 5ml of Lanthanium Chloride solution per 100ml of the solution was added.

Phosphorous:

An aliquot of the digested ash was used for the estimation of phosphorous by the standard ammonium molybdate calorimetric procedure.

3.9. Making tested foods in to Powder

After preparing the tested food with different methods, and then the foods were dried by sun light and oven (<85°C). The dried foods were manually grinded and the powders were kept in clean glasses at room temperature until used for the experiment/test.

3.10. Preparation of food powder solution for administration

The powder food (0.25g) was weight dissolved in 1ml of water administered in to each mouse (six). The same way the standard (glucose) 0.25g was weight dissolved in 1ml of water to administered each mouse (six).



Figure 3: Administration of test food in to mouse.

3.11. Blood glucose measurement- The blood samples were measured using GlucoSure® Plus Blood Glucometer to determine blood glucose levels of foods.

Glucometer

Glucometer used for this study was GlucoSure® Plus Blood Glucose Meter (APEX BIOTECHNOLOGY CORP., Hsinchu, Taiwan, R.O.C.).

Principle-; The GlucoSure® Plus Blood Glucose Meter is designed specifically to detect glucose in whole blood with the Touch-In® Plus Blood Glucose Test Strip. The Touch-In® Plus Blood Glucose Test Strips come in a moisture-proof and light-protected bottle. It is important that the bottle is kept well sealed at all times and the cap is replaced immediately after a strip is removed.

3.12. Estimation of blood glucose concentrations using glucometer

Fasting blood glucose was measured with GlucoSure® Plus glucometer after getting the blood sample from the tail vein of the overnight (12-14 hr) fasted mice and measured blood glucose after administered foods and standard glucose with 30 minute intervals for 2hrs (0, 30, 60, 90, 120min).

3.13. Determination of glycemic index

3.13.1. Blood glucose response graphs

The blood glucose response curve vs. time was obtained by plotted a graph thus: x-axis, time interval and y-axis, blood glucose concentration. The averages of the respective blood glucose response before and after administering the food were used to draw a blood glucose response curve for the two-hour period.

3.13.2. Calculations of glycemic index .

The incremental area under the curve (IAUC) was calculated for each food in every mouse separately (as the sum of the surface of triangles and trapezoids between the blood glucose curve and horizontal baseline going parallel to x-axis from the beginning of blood glucose curve at time 0 to the point at time 120 min) to reflect the total rise in blood glucose response after administering the tested foods. The IAUCg for the standard glucose was obtained similar to the mean from the first two independent IAUCg1 and IAUCg2, in the mice. When a blood glucose value falls below the baseline, only the area above the fasting level is included. In each mouse,

the GI (%) was calculated by dividing the IAUC for the tested food by the IAUCg for the standard glucose and multiplying by 100. The following formula was used (Brouns *et al.*, 2005):
The Glycemic Index (GI) of a food was:

$$\frac{\text{IAUCf (above fasting baseline)}}{\text{Mean IAUCg (above fasting baseline)}} \times \frac{100}{1}$$

where:

IAUCf (above fasting baseline)= Incremental area under the blood glucose response curve above fasting baseline of a food.

Mean IAUCg (above fasting baseline) = Mean incremental area under the curve above fasting baseline of two determinations of the standard glucose (Wolever *et al.*, 1991).

The GI value of a food was the mean of six mice of the percentage expression in each mouse.

3.14. Data management

Everyday, before each experiment the glucosure® plus blood glucose meter was calibrated using the codes by the manufacturer. But the code card found in the test strip package was for use with that particular package only.

3.15. Statistical analysis

All the values of blood glucose and glycemic index were expressed as mean ± standard error (Mean± SE) and were performed using SPSS software package Version 21.0. The values were analyzed by one-way analysis of variance (ANOVA). A value of P < 0.05 was considered to be evidence for statistically significant.

3.16. Ethical consideration

The research proposal was approved and ethically cleared by the Ethical Review Committee of the Departments of Biochemistry, School of Medicine, Addis Ababa University. Supportive letter should be written to the Ethiopian health and nutrition research institute (EHNRI) from the department of Biochemistry, Faculty of Medicine, College of health sciences, Addis Ababa University. Confidentiality of response was maintained throughout the study.

4. RESULTS

The proximate composition of twelve traditional Ethiopian foods are given in Table 4. The carbohydrate of these tested foods ranges from 10.4- 57.3 g/100g. From this range white bread contained high carbohydrate content (grater than 50g/100g) and chickpea and pea sauce were low carbohydrate foods (less than 15g/100g). White and red teff enjera exhibited the highest fiber content (greater than 1.5g/100g) while bulla *genfo* was the lowest fiber (less than1g/100g).

Table 4. Proximate nutritional composition of twelve traditional Ethiopian foods considered in the study (Expressed as gram per 100grams on fresh weight basis).

| Compn Test F | Moisture (%) | Protein gram | Fat gram | CHO gram | Fiber gram | Ash gram | Nit(N) gram | Cal(Ca) (mg) | Phosp(P) (mg) | Iron(Fe) (mg) |
|--------------------|-----------------|-----------------|-------------|-------------|---------------|-------------|----------------|-----------------|------------------|------------------|
| White teff enjera | 56.3 | 4.9 | 1.0 | 36.3 | 2.2 | 1.3 | 0.78 | 73.0 | 164.1 | 56.0 |
| Red teff enjera | 60.2 | 3.4 | 0.7 | 34.0 | 1.8 | 1.7 | 0.58 | 50.0 | 115.0 | 14.7 |
| Maize enjera | 60.2 | 4.4 | 1.3 | 33.5 | 1.2 | 0.6 | 0.71 | 2.4 | 111.1 | 9.8 |
| Wheat bread | 44.8 | 6.6 | 0.7 | 45.6 | 1.7 | 2.3 | 1.06 | 38.4 | 147.7 | 3.4 |
| Maize bread | 52.2 | 4.5 | 1.9 | 40.6 | 1.3 | 0.8 | 0.72 | 10.5 | 126.9 | 22.8 |
| Barley bread | 49.5 | 4.4 | 0.4 | 45.3 | 1.1 | 1.4 | 0.71 | 16.0 | 160.0 | 3.5 |
| Pea sauce | 75.3 | 6.6 | 2.9 | 11.8 | 1.0 | 3.4 | 1.06 | 43.6 | 115.7 | 16.8 |
| Chickpea sauce | 80.9 | 3.4 | 2.0 | 10.4 | 1.4 | 3.3 | 0.55 | 97.6 | 76.9 | 11.7 |
| Lentil sauce | 73.4 | 5.6 | 2.6 | 14.9 | 1.0 | 3.5 | 0.89 | 25.7 | 76.6 | 8.8 |
| Qocho bread | 52.9 | 1.6 | 0.1 | 44.1 | 1.4 | 1.3 | 0.25 | 71.3 | 98.8 | 2.8 |
| Bulla <i>genfo</i> | 77.3 | 0.1 | 3.8 | 17.9 | 0.2 | 0.9 | 0.01 | 18.3 | 1.9 | 0.6 |
| White bread | 31.4 | 6.8 | 4.3 | 57.3 | 0.3 | 0.2 | 1.08 | 3.4 | 45.5 | 0.5 |

The glycemic index of twelve traditional Ethiopian foods were investigated in mice. The results of the mean glycemic index value of different traditional Ethiopian foods are given below.

4.1. The glycemic index of white teff enjera

Table 5 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of white teff enjera was found to be 35, which classifies this food as a low GI food ($GI \leq 55$).

Table 5 : Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of White teff enjera.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic Index (GI) |
|----------|---|---------|---------------------|
| | White teff enjera | Glucose | |
| GI mean | - | - | 35 |
| SE | - | - | 1.2 |
| Mouse 1. | 116.85 | 368 | 32 |
| Mouse 2. | 203.85 | 534 | 38 |
| Mouse 3. | 227.55 | 576.5 | 39 |
| Mouse 4. | 89.5 | 254.35 | 35 |
| Mouse 5. | 109.75 | 301 | 36 |
| Mouse 6. | 112.2 | 354 | 32 |

- ✚ IAUC determined by geometric calculation by applying the trapezoid rule
- ✚ GI calculated as IAUC of enjera expressed as a percentage of IAUC of glucose

Figure 4 graphically presents the blood glucose responses which the white teff enjera and standard glucose samples had on the twelve healthy mice (six for enjera and six for glucose) who administered them. The blood glucose level after administered white teff enjera starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing white teff enjera vs. glucose ($P=0.001$).

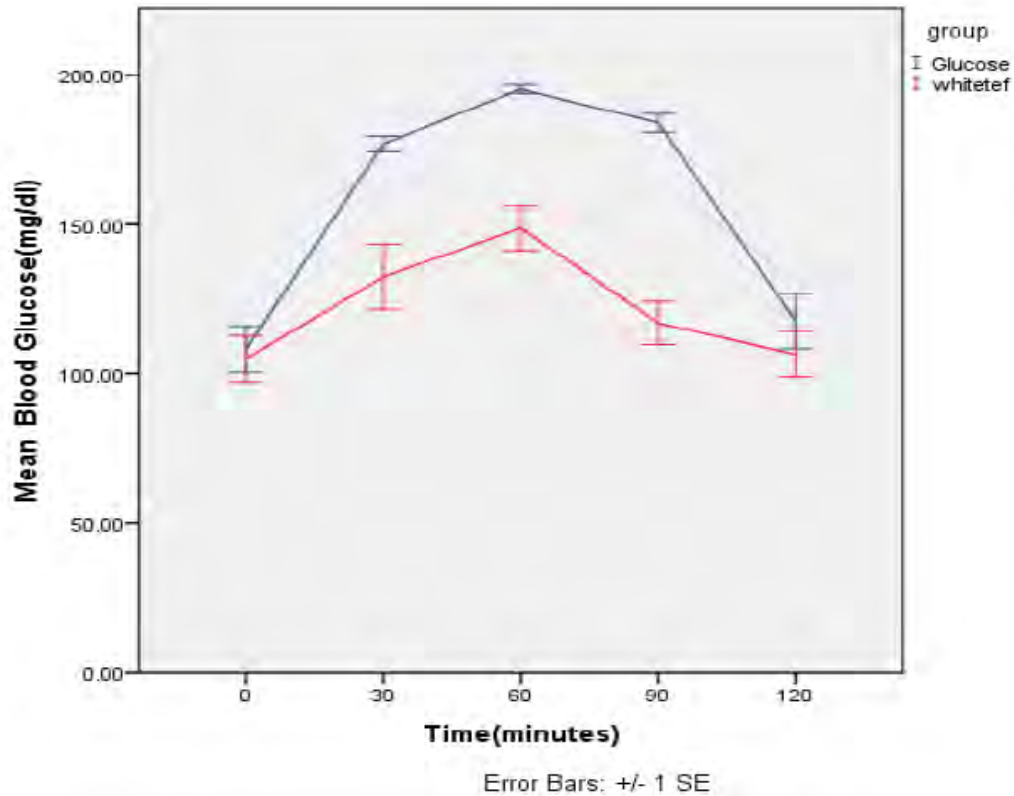


Figure 4: Blood glucose response at different time interval for white teff enjera with standard glucose.

4.2. The glycemic index of red teff enjera

Table 6 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of red teff enjera was 39, which classifies this food as a low GI food ($GI \leq 55$).

Table 6: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Red teff enjera.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | Red tef enjera | Glucose | |
| Mean GI | - | - | 39 |
| SE | - | - | 0.7 |
| Mouse 1. | 143.25 | 365.3 | 39 |
| Mouse 2. | 201.5 | 531.1 | 38 |
| Mouse 3. | 237.67 | 579 | 41 |
| Mouse 4. | 103.02 | 253.3 | 41 |
| Mouse 5. | 120.47 | 304.3 | 40 |
| Mouse 6. | 131.7 | 356.4 | 37 |

Figure 5 indicates that the blood glucose responses after administered red teff enjera starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing red tef enjera vs. glucose (P=0.002).

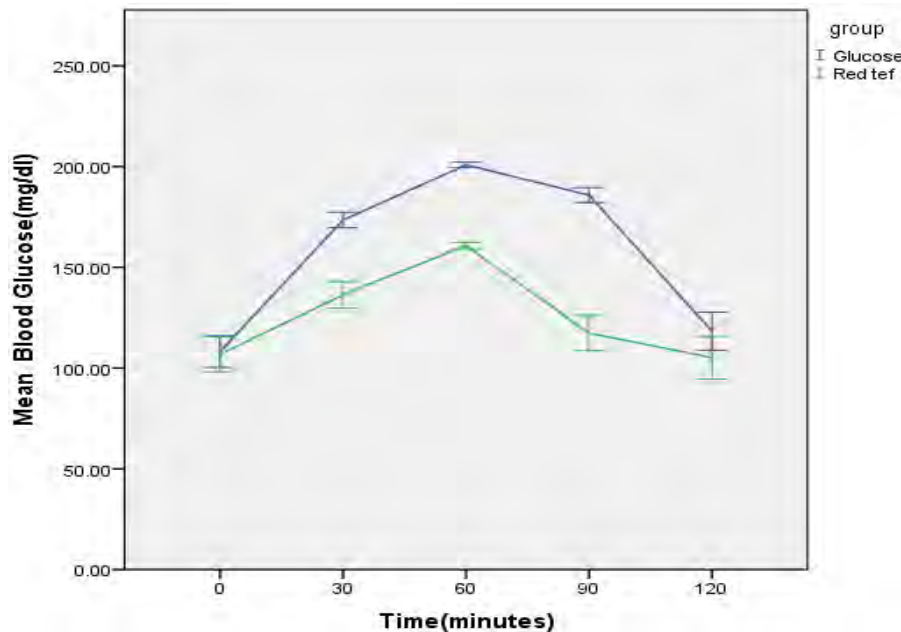


Figure 5: Blood glucose response at different time interval for red teff enjera with standard glucose.

4.3. The glycemic index of maize (corn) enjera

Table 7 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of maize enjera was found to be 43, which classifies this food as a low GI food ($GI \leq 55$).

Table 7: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Maize enjera.

| Mice | Incremental Area Under the Curve (IAUC) | | Glyemic index (GI) |
|----------|---|---------|--------------------|
| | Maize enjera | Glucose | |
| Mean GI | - | - | 43 |
| SE | - | - | 1.3 |
| Mouse 1. | 168.9 | 366.2 | 46 |
| Mouse 2. | 202.17 | 531 | 38 |
| Mouse 3. | 229.4 | 576.5 | 40 |
| Mouse 4. | 112.35 | 257.3 | 44 |
| Mouse 5. | 138.6 | 303.5 | 46 |
| Mouse 6. | 156.64 | 357.3 | 44 |

Figure 6 indicates that the blood glucose level after administered maize enjera starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing maize enjera vs. glucose (0.002).

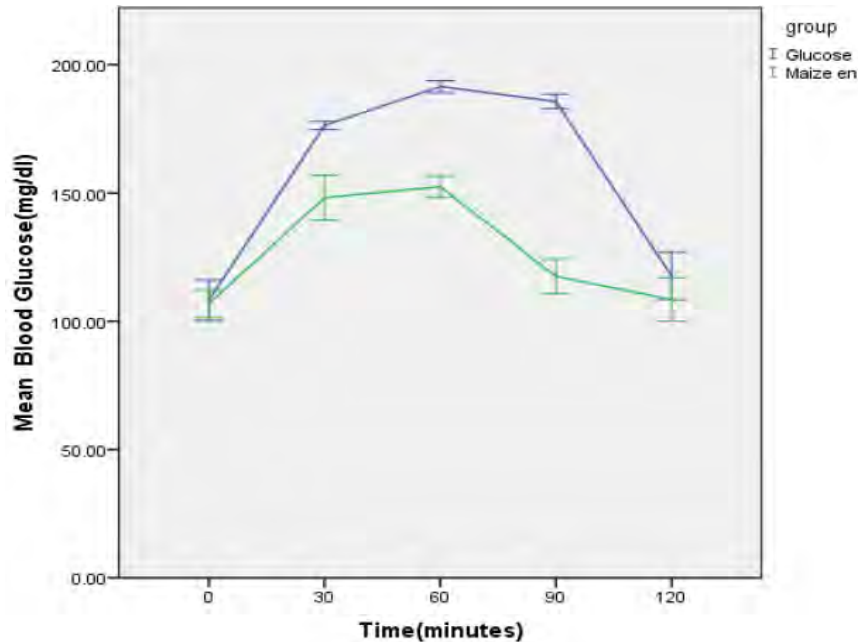


Figure 6: Blood glucose response at different time interval for maize enjera with standard glucose.

4.4. The glycemic index of barley bread

Table 8 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of barley bread was 25, which classifies this food as a low GI food ($GI \leq 55$).

Table 8: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Barley bread.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | Barley bread | Glucose | |
| Mean GI | - | - | 25 |
| SE | - | - | 1.4 |
| Mouse 1. | 78.13 | 364.5 | 21 |
| Mouse 2. | 110.7 | 539 | 21 |
| Mouse 3. | 146.3 | 577.3 | 25 |
| Mouse 4. | 71.22 | 254.35 | 28 |
| Mouse 5. | 88.02 | 305 | 29 |
| Mouse 6. | 84.3 | 349 | 24 |

Figure 7 graphically presents the blood glucose responses which the barley bread and standard glucose samples had on the twelve healthy mice (six for barley bread and six for glucose) who administered them. The blood glucose level after administered barley bread starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing barley bread vs. glucose (0.0004).

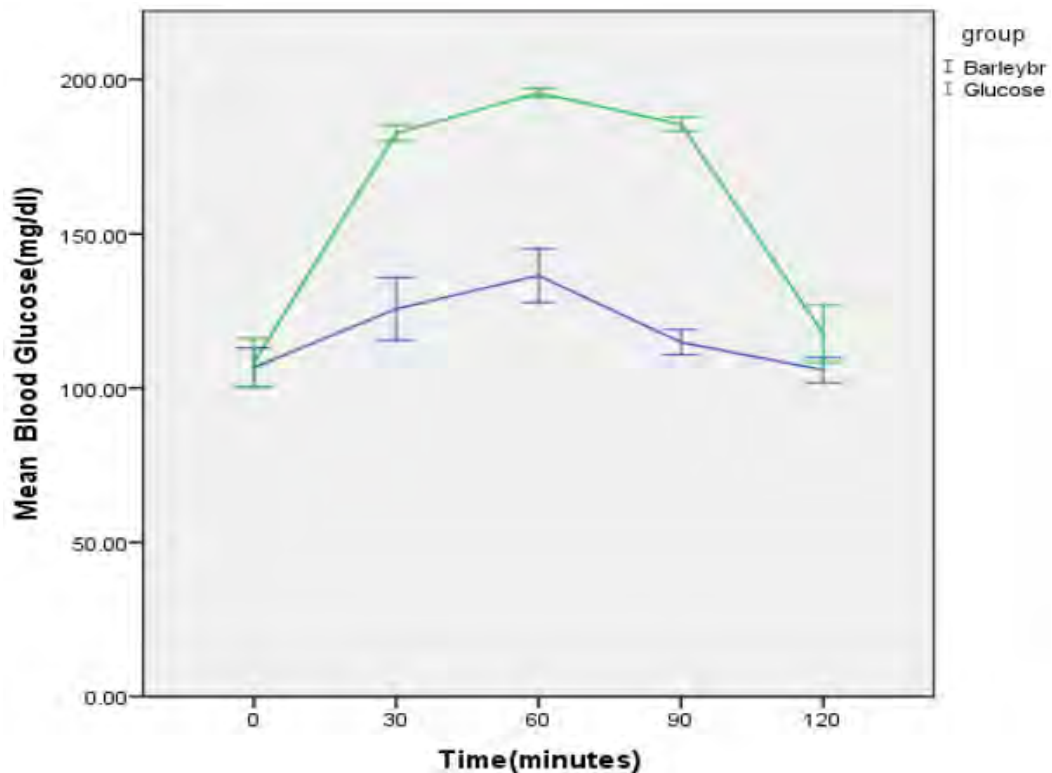


Figure 7: Blood glucose response at different time interval for barley bread with standard glucose.

4.5. The glycemic index of maize bread (Traditional *diffo dabo*)

Table 9 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of maize bread was found to be 56, which classifies this food as a moderate GI food (GI =56-69).

Table 9: Incremental area under the curve (IAUC) & calculated glyceimic index (GI) values of Maize bread.

| Mice | Incremental Area Under the Curve (IAUC) | | Glyceimic index (GI) |
|----------|---|---------|------------------------|
| | Maize bread | Glucose | |
| Mean GI | - | - | 56 |
| SE | - | - | 0.8 |
| Mouse 1. | 204.3 | 364.6 | 56 |
| Mouse 2. | 287.7 | 531.6 | 54 |
| Mouse 3. | 336.3 | 574.9 | 58 |
| Mouse 4. | 147.5 | 253.2 | 58 |
| Mouse 5. | 161.06 | 299 | 54 |
| Mouse 6. | 199.1 | 356.6 | 56 |

Figure 8 indicates that the blood glucose level after administered maize bread starts to rise up at time 0 and reached a glyceimic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing maize bread vs. glucose (0.016).

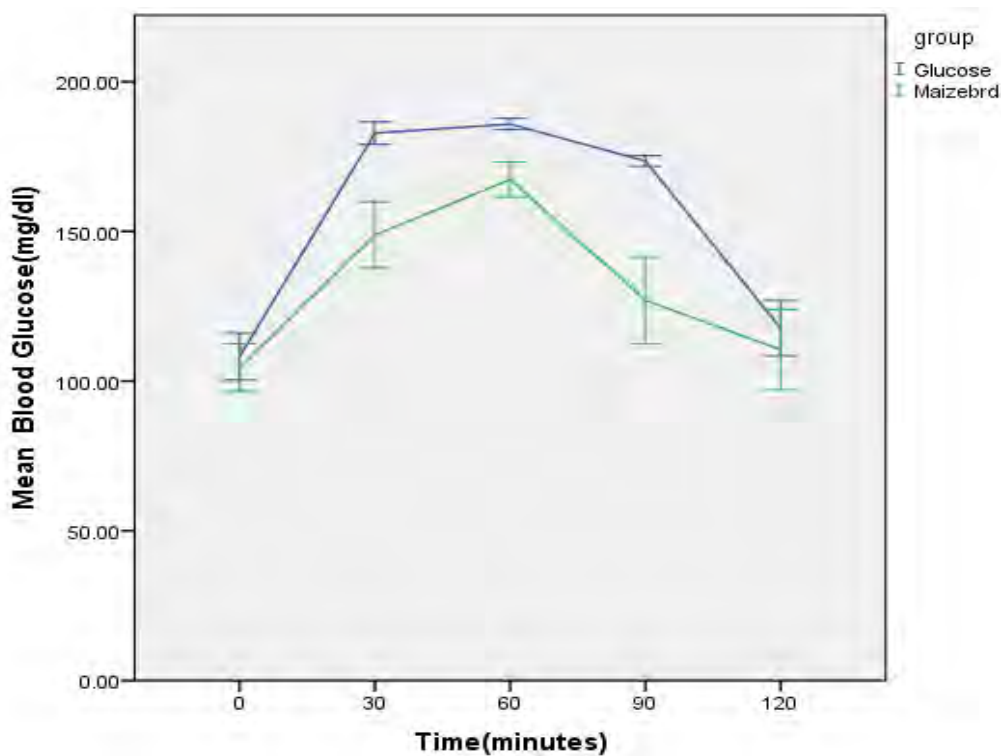


Figure 8: Blood glucose response at different time interval for maize bread with standard glucose.

4.6. The glycemic index of wheat bread (Traditional *diffo dabo*)

Table 10 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of wheat bread was 57, which classifies this food as a moderate GI food (GI =56-69).

Table 10: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Wheat bread.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | Wheat bread | Glucose | |
| Mean GI | - | - | 57 |
| SE | - | - | 1.2 |
| Mouse 1. | 203 | 365 | 55 |
| Mouse 2. | 289.1 | 534.8 | 54 |
| Mouse 3. | 333 | 579.5 | 58 |
| Mouse 4. | 154.4 | 251.6 | 61 |
| Mouse 5. | 163.2 | 304.63 | 54 |
| Mouse 6. | 206.27 | 358.1 | 58 |

Figure 9 indicates that the blood glucose level after administered wheat bread starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120mins. There was a significant difference in the blood glucose increment at different time interval when comparing wheat bread vs. glucose (0.016).

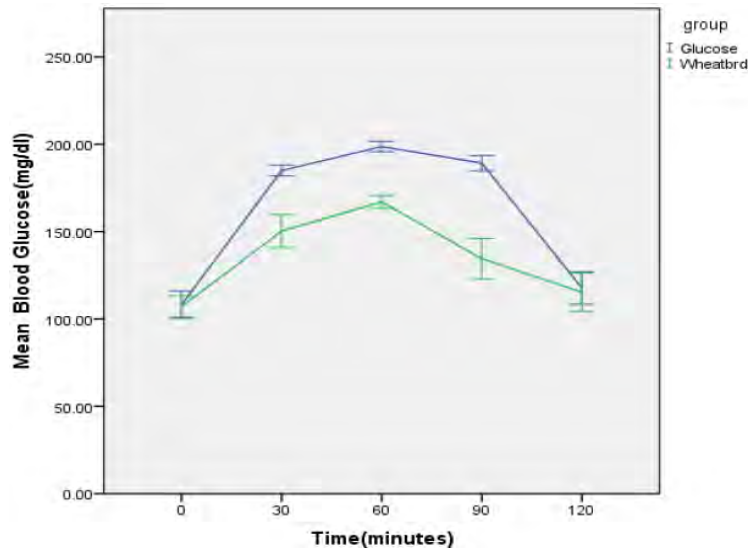


Figure 9: Blood glucose response at different time interval for wheat bread with standard glucose.

4.7. The glycemic index of white bread (bread from bakery)

Table 11 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of white bread was found to be 73, which classifies this food as a high GI food ($GI \geq 70$).

Table 11: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of White bread.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|-----------------------|
| | White bread | Glucose | |
| Mean GI | - | - | 73 |
| SE | - | - | 0.7 |
| Mouse 1. | 263.7 | 368 | 72 |
| Mouse 2. | 372.3 | 534 | 70 |
| Mouse 3. | 413.85 | 575.2 | 72 |
| Mouse 4. | 185.75 | 254.35 | 73 |
| Mouse 5. | 226.3 | 303.2 | 75 |
| Mouse 6. | 258 | 352.2 | 73 |

Figure 10 graphically presents the blood glucose responses which the white bread and standard glucose samples had on the twelve healthy mice (six for bread and six for glucose) who administered them. The blood glucose level after administered white bread starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing white bread vs. glucose (0.039).

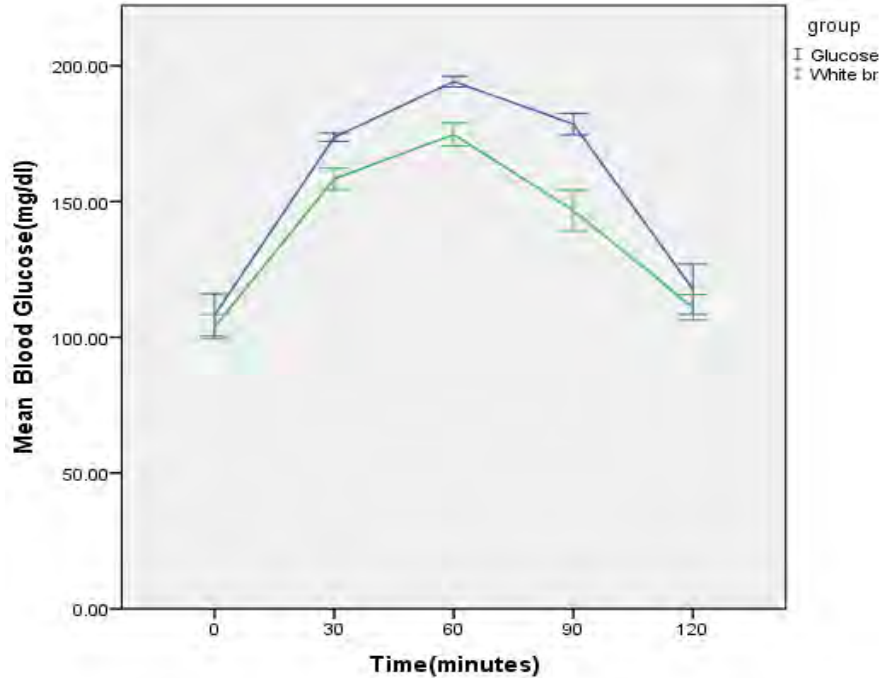


Figure 10: Blood glucose response at different time interval for white bread with standard glucose.

4.8. The glycemic index of qocho bread

Table 12 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of qocho bread was found to be 38, which classifies this food as a low GI food ($GI \leq 55$).

Table 12: Incremental Area under the curve (IAUC) and calculated glycemic index (GI) values of Qoch'o bread.

| Mice | Incremental Area under the curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | Qocho bread | Glucose | |
| Mean GI | - | - | 38 |
| SE | - | - | 1.2 |
| Mouse 1. | 154.15 | 369 | 42 |
| Mouse 2. | 201.7 | 536.3 | 38 |
| Mouse 3. | 227.55 | 578 | 39 |
| Mouse 4. | 87.7 | 254.5 | 34 |
| Mouse 5. | 101.6 | 299.6 | 34 |
| Mouse 6. | 138 | 356.6 | 39 |

Figure 11 indicate that the blood glucose level after administered qocho bread starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing qocho bread vs. glucose (0.002).

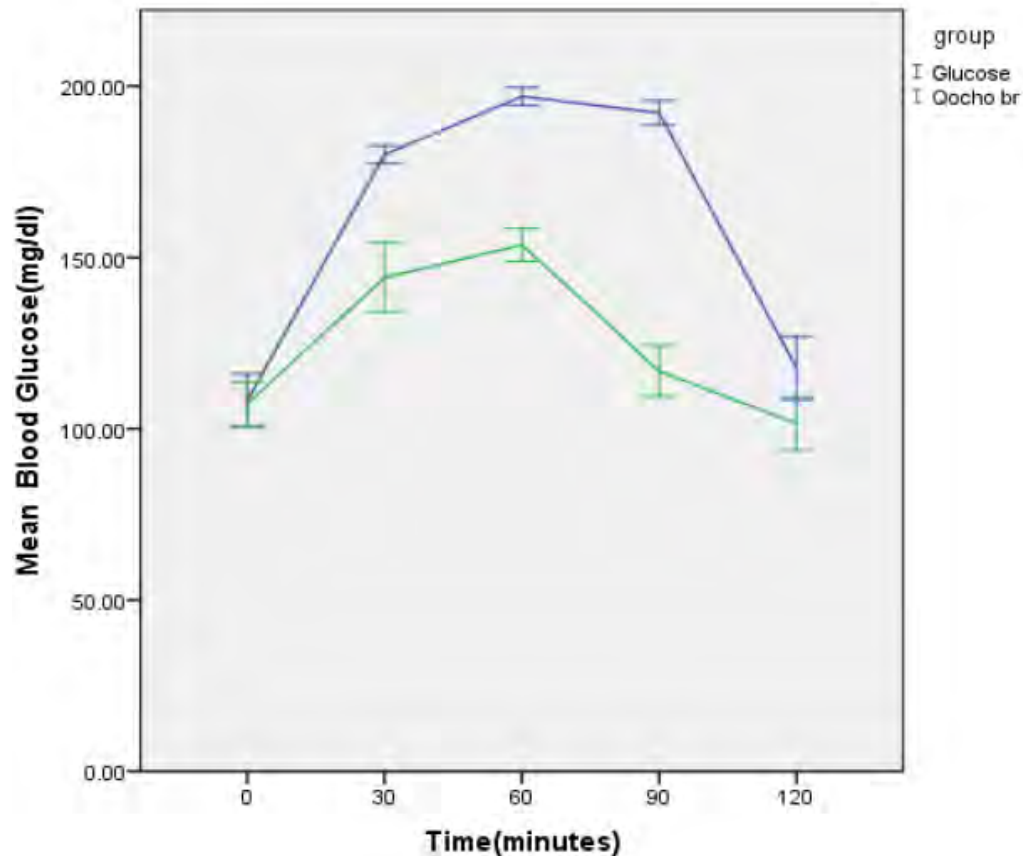


Figure 11: Blood glucose response at different time interval for qoch'o bread with standard glucose.

4.9. The glycemic index of *Bulla genfo*

Table 13 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of *bullu genfo* was found to be 60, which classifies this food as a moderate GI food (GI=56-69).

Table 13: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of *Bulla genfo*.

| Mice | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | <i>Bulla genfo</i> | Glucose | |
| Mean GI | - | - | 60 |
| SE | - | - | 1.3 |
| Mouse 1. | 223.8 | 369 | 61 |
| Mouse 2. | 344.7 | 532.7 | 65 |
| Mouse 3 | 335.8 | 574.3 | 58 |
| Mouse 4. | 154 | 251.9 | 61 |
| Mouse 5. | 169.27 | 303.5 | 56 |
| Mouse 6. | 205.95 | 354 | 58 |

Figure 12 indicates that the blood glucose level after administered *bulla genfo* starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing *bulla genfo* vs. glucose (0.021).

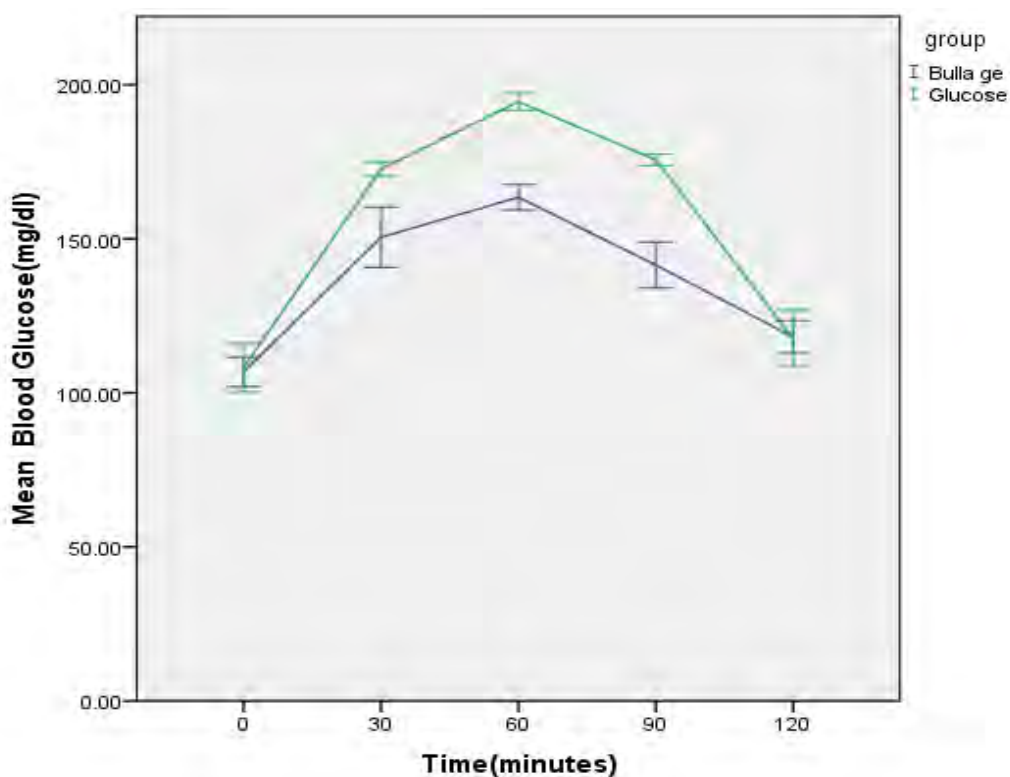


Figure 12: Blood glucose response at different time interval for *bulla genfo* with standard glucose.

4.10. The glycemic index of pea sauce (*Ater shiro wet*)

Table 14 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of pea sauce was found to be 41, which classifies this food as a low GI food ($GI \leq 55$).

Table 14: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Pea sauce.

| Mice | Incremental Area Under the Curve (I AUC) | | Glycemic index (GI) |
|----------|--|---------|---------------------|
| | Pea sauce | Glucose | |
| Mean GI | - | - | 41 |
| SE | - | - | 1.7 |
| Mouse 1. | 143.25 | 369.2 | 39 |
| Mouse 2. | 185.25 | 535.2 | 35 |
| Mouse 3. | 229.35 | 579.2 | 40 |
| Mouse 4. | 114.45 | 251.6 | 45 |
| Mouse 5. | 139.2 | 301 | 46 |
| Mouse 6. | 152.1 | 351.7 | 43 |

Figure 13 graphically presents the blood glucose responses which the pea sauce and standard glucose samples had on the twelve healthy mice (six for sauce and six for glucose) who administered them. The blood glucose level after administered pea sauce starts to rise up at time 0 and reached a glycemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing pea sauce vs. glucose (0.002).

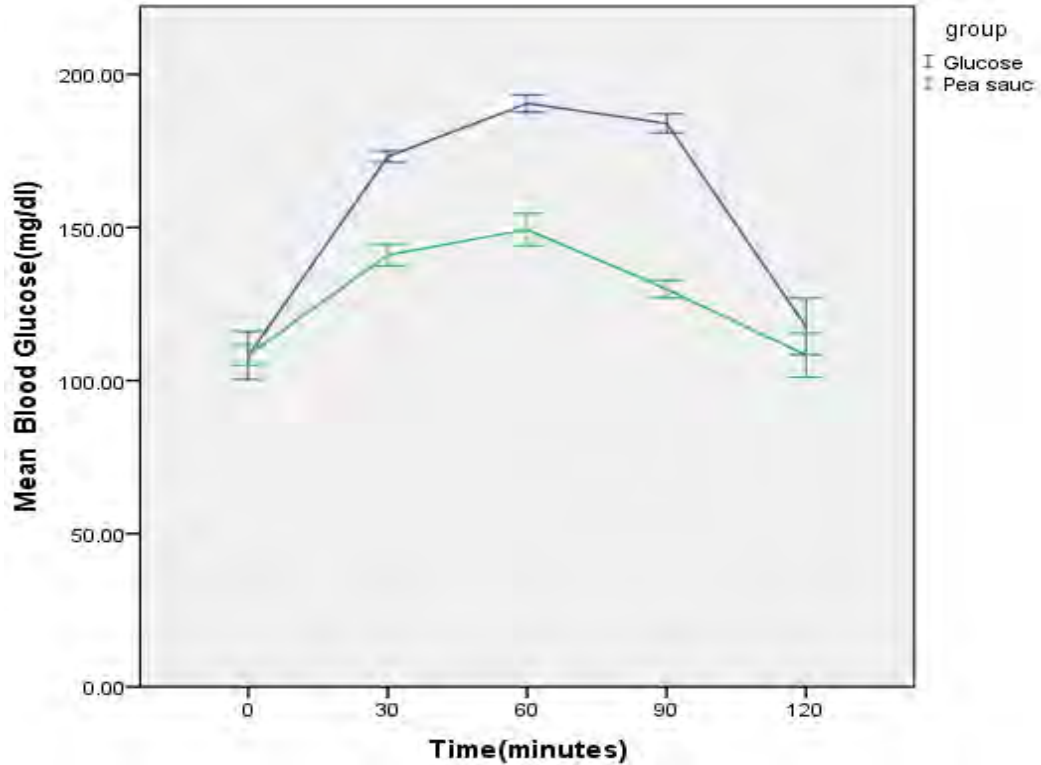


Figure 13: Blood glucose response at different time interval for pea sauce with standard glucose.

4.11. The glycemic index of Chickpeas sauce (*Shimbre shiro wet*)

Table 15 presents the incremental area under the curve (IAUC) and the mean glycemic index (GI) values of each mouse together with SE. The mean glycemic index value of chickpeas sauce was 27, which classifies this food as a low GI food ($GI \leq 55$).

Table 15: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Chickpeas sauce.

| Mean | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|--------------------------|
| | Chickpea sauce | Glucose | |
| Mean GI | - | - | 27 |
| SE | - | - | 1.6 |
| Mouse 1. | 99.5 | 364 | 27 |
| Mouse 2. | 110.7 | 531 | 21 |
| Mouse 3. | 146.26 | 573.8 | 25 |
| Mouse 4. | 71.22 | 254.35 | 28 |
| Mouse 5. | 88.13 | 304.4 | 29 |
| Mouse 6. | 118.65 | 351.3 | 34 |

Figure 14 indicates that the blood glucose level after administered chickpea sauce starts to rise up at time 0 and reached a glycaemic peak value at 60min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing chickpea sauce vs. glucose (0.0005).

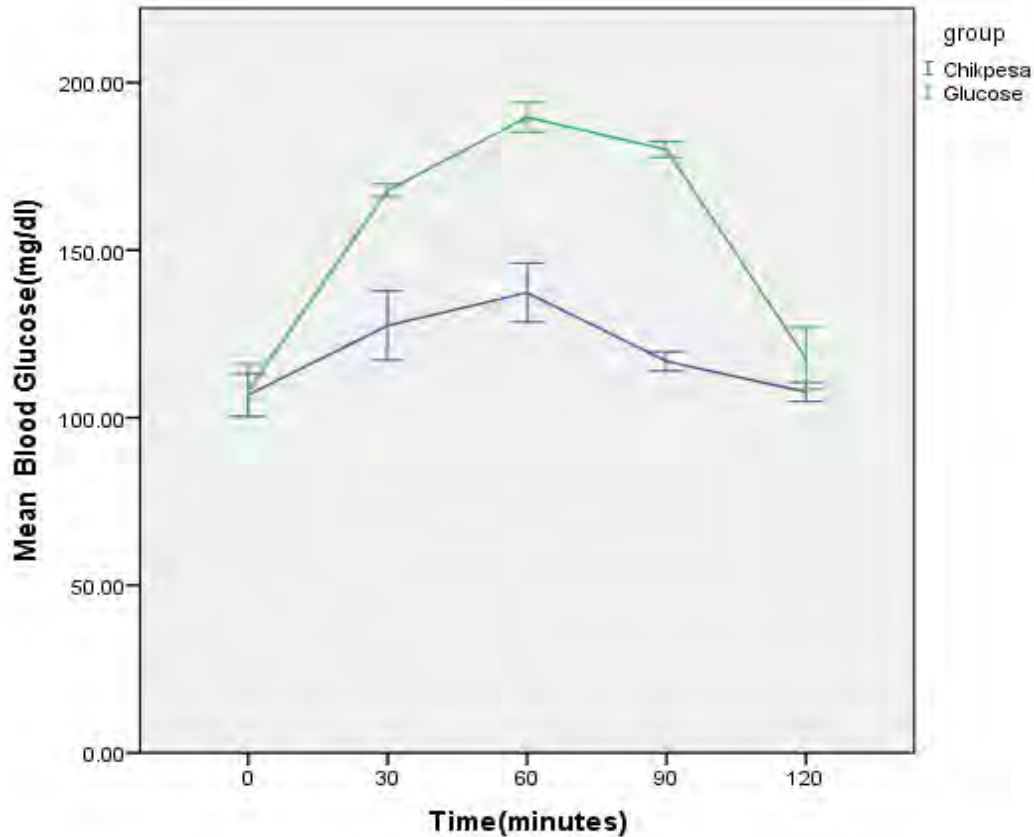


Figure 14: Blood glucose response at different time interval for chickpea sauce with standard glucose.

4.12. The glycaemic index of Lentil sauce (*Missir wet*)

Table 16 presents the incremental area under the curve (IAUC) and the mean glycaemic index (GI) values of each mouse together with SE. The mean glycaemic index value of lentil sauce was found to be 17, which classifies this food as a low GI food ($GI \leq 55$).

Table 16: Incremental area under the curve (IAUC) and calculated glycemic index (GI) values of Lentil sauce.

| Mean | Incremental Area Under the Curve (IAUC) | | Glycemic index (GI) |
|----------|---|---------|---------------------|
| | Lentil sauce | Glucose | |
| Mean GI | - | - | 17 |
| SE | - | - | 1.0 |
| Mouse 1. | 67.4 | 365.3 | 18 |
| Mouse 2. | 84.2 | 532.4 | 16 |
| Mouse 3. | 88 | 573.8 | 15 |
| Mouse 4. | 35.98 | 251.3 | 14 |
| Mouse 5. | 59.7 | 303.6 | 20 |
| Mouse 6. | 71.4 | 357 | 20 |

Figure 15 indicates that the blood glucose level after administered lentil sauce starts to rise up at time 0 and reached a glycemic peak value at 30min and starts slowly dropping down and reached to minimum value at 120min. There was a significant difference in the blood glucose increment at different time interval when comparing lentil sauce vs. glucose (0.00).

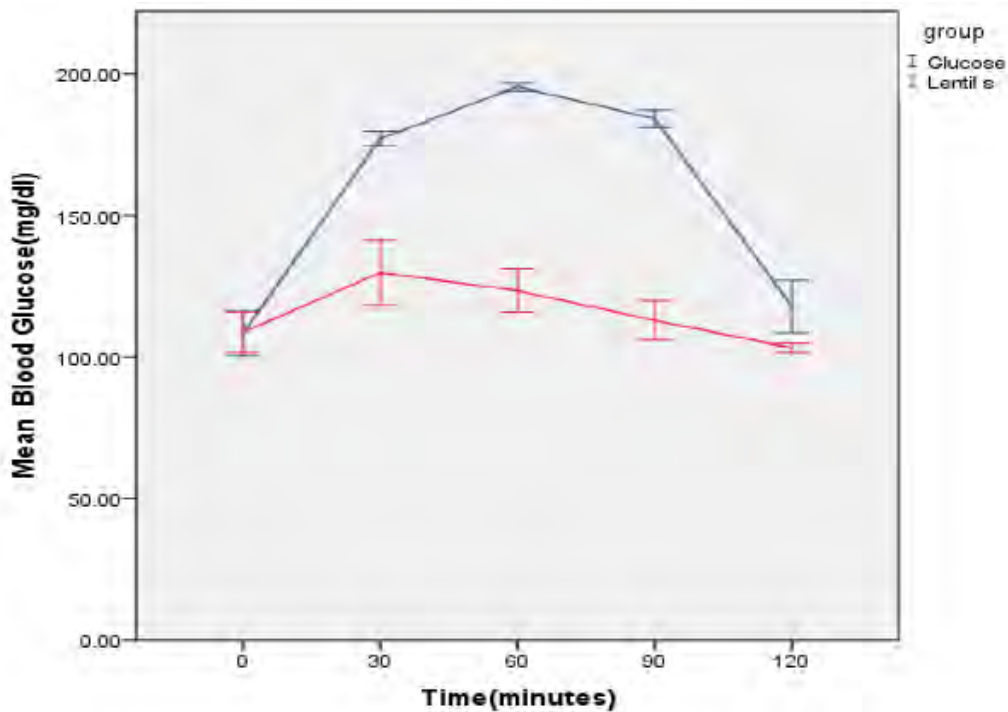


Figure 15: Blood glucose response at different time interval for lentil sauce with standard glucose.

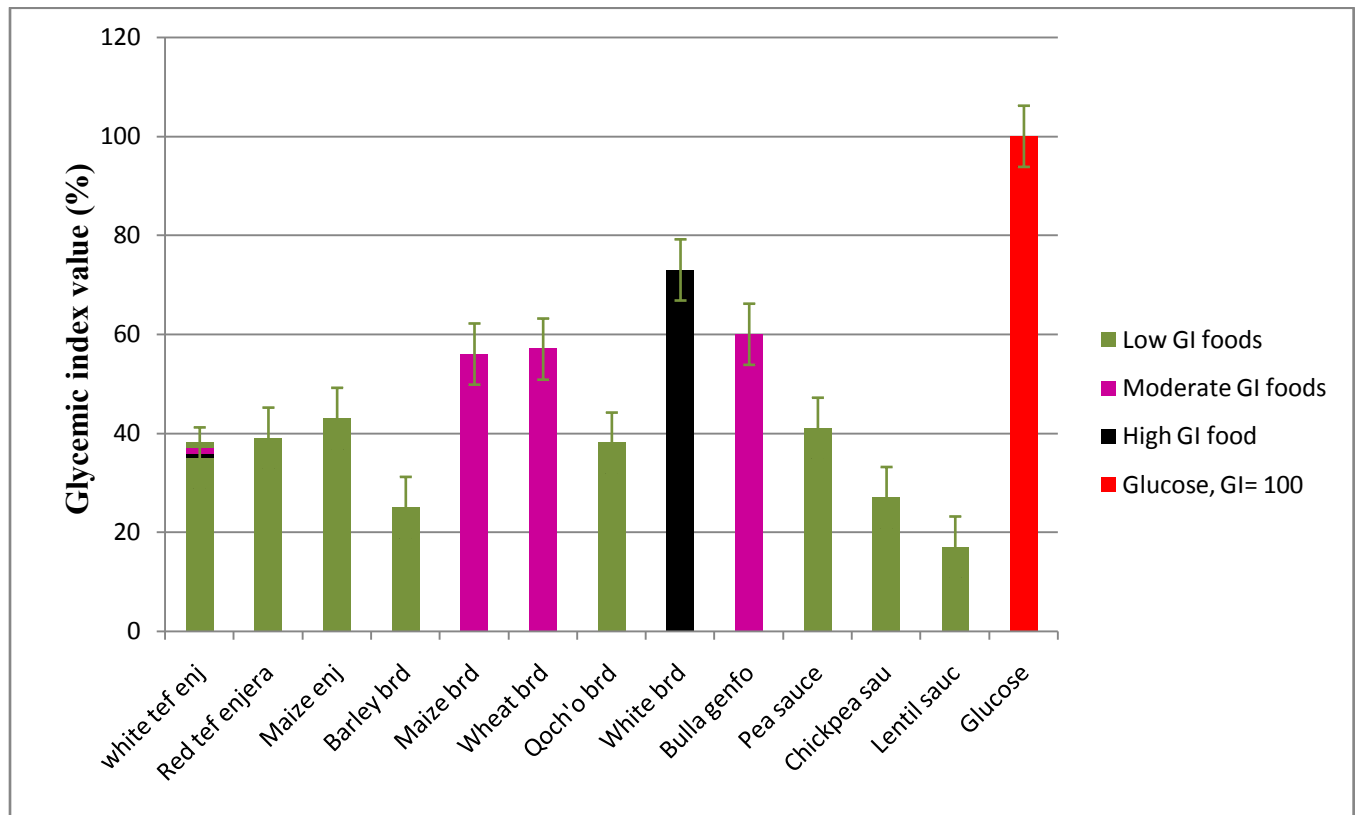


Figure 16: The mean glycemic index (GI) with standard error (SE) values for the standard food (glucose) and the twelve traditional Ethiopian foods.

5. DISSCUSSION

The glycemic index (GI) of foods have been reported to be a marker of some beneficial effects (Pawlak & Kushner *et al.*, 2004). Low glycemic index diets are important in the management of hyperglycemia and hyperinsulinemia. They are more effective per unit of energy, than most other foods in inducing satiety, these foods have a potential role in dietary strategies to avoid and treat diabetes and obesity (Obloh *et al.*, 2010; Anderson *et al.*, 1991). Foods have been reported to produce different glycemic values depending on their chemical structure, particle size, amount and type of dietary fiber, fat, protein, anti-nutrient and food processing which may explain the variation among the carbohydrates of the food (Jenkin *et al.*, 2000).

Processing of food have been reported to have effect on glycemic index (Omoriegie & Osagie 2008). Boiling, cooking, and heating result in alteration of the physical property of carbohydrates through gelatinization and retrogradation, thereby increase the starch availability to amylase (Jimoh *et al.*, 2008). The heat utilized, the amount of water, the time of cooking, all have a significant effect on the glycemic index (Pi-Sunyer, 2002), this could also be attributed to the observed variation in the GI of the test foods. Chewing has been shown to reduce the particle size of foods and facilitates mixture with salivary amylase, thereby reducing digestion time of carbohydrate (Omoriegia and Osogie, 2008). This is in conformity with reports that different foods with similar quality and type of carbohydrate form show different glycemic response (Thorsdottir *et al.*, 2005).

Determination of glycemic index of traditional Ethiopian foods are important for monitoring or balancing the blood glucose levels. The present study was designed to determine the glycemic index of twelve traditional Ethiopian foods. Among the twelve traditional Ethiopian foods, there were eight foods reported as low GI these are-; 1. white tef enjera, GI=35, 2. red tef enjera, GI=39, 3. maize enjera, GI=43, 4. barley bread, GI=25, 5. qocho bread, GI=38, 6. pea sauce, GI=41, 7. chickpea sauce, GI=27 and 8. lentil sauce, GI=17. From these eight low GI foods, maize enjera reported as highest glycemic response, GI=43, and lentil sauce was reported as the lowest glycemic response, GI=17. These low glycemic index (GI) foods have a number of positive health impacts; these include: (1) improved glycemic control in diabetic subjects, which is associated with reduced risk of diabetes, (2) more favorable lipid profiles, which are associated with lower risk of cardiovascular disease, and (3) reduced markers of inflammation, which are associated with lower risk of metabolic syndrome, overweight and other chronic diseases. For

this reason low glycemic index foods are important to assist in disease prevention, especially the prevention of diabetes, cardiovascular disease, obesity, cancer and other chronic diseases of lifestyle (Pawlak & Kushner *et al.*, 2004). From the twelve traditional Ethiopian foods, there were three foods reported as having moderate GI: these are-; maize bread, GI= 56, wheat bread, GI= 57 and bulla *genfo*, GI=60. From the three foods, bulla *genfo* had the highest glycemic response (GI=60) and maize bread had the lowest response (GI=56). These low to moderate GI traditional Ethiopian foods are important for balancing (monitoring) blood glucose levels for diabetes mellitus.

Of the twelve traditional Ethiopian foods only one food was reported as having a high GI; this food was white bread (bread from bakery), GI =73. This high glycemic index food is quickly digested and enters the blood stream, quickly increasing the blood glucose rapidly. Increasing of blood glucose results in corresponding increase of blood insulin, promoting fat storage, increase cardiovascular risks, promote the development of type 2 diabetes and promote certain cancers (Ludwig, 2002). So high glycemic index foods are positively associated with diabetes mellitus, and other chronic disease such as cardiovascular disease, obesity and cancers. Therefore, white bread is not beneficial to control blood glucose for diabetic patients.

Pi-Sunyer, 2002 and David M, 2008 reported wheat bread and maize bread to produce moderate glycemic response. In the present study also categorized wheat and maize bread as moderate GI foods, although higher carbohydrate content was observed.

Oboh *et al.*, 2010 reported the legumes to produce relatively high glycemic response in healthy individuals in Nigeria. The lower GI value of legumes (pea, chickpea and lentil sauce) in this study may be explained by their components particularly the soluble diet fiber and the nature of starch. Jimoh *et al.*, 2008 reported the cereals relatively produced high glycemic index (Kenya). In the present study cereal produced low GI may be explained by high fiber content.

After preparing the tested food with different traditional methods, and then the foods were dried by sun light and oven (<85°C). The dried foods were manually grinded and the powders were kept in clean glasses at room temperature. The powder food (0.25g) was weight dissolved in 1ml of water administered in to each mouse (six). These procedures may affect the value of glycemic index of foods, because making tested foods in to powder causes the decrease in food particle size, leads to increase the rate of digestion and absorption of foods and produced high glycemic index.

According to the Table 4 (proximate nutritional composition) there is no correlation between glycemic index and protein and fat, for example, white bread shows the highest protein and fat contents and categorized under high glycemic index, but qocho bread contains lowest protein and fat and classified as the lowest glycemic index food, although numerous studies have shown that GI is lower with increased fat and protein content of many foods. Therefore, in this study there is no relationship between glycemic index and protein and fat contents of foods.

The Mediterranean Diet (or Med Diet) reflects a way of eating that is traditional in the countries that surround the Mediterranean. Key elements of those choices include: Healthy oils, such as olive oil or canola oil, vegetables and whole grains, fish or seafood twice a week, very little red meat or eggs, e.t.c. There are many reasons to follow the Mediterranean Diet! Scientific evidence shows that it can help you: Achieve weight loss and weight management goals, lower risk of heart disease and high blood pressure, fight certain cancers and chronic diseases, reduce asthma, avoid diabetes, resist depression, nurture healthier babies, and ward off Parkinson's disease.

Ethiopian traditional foods are also important for the prevention of different chronic disease and decrease the risk of development of disease including CVD, CHD and cancers. These foods contain legumes, cereals, especially enjera and root products.

Importance of teff *enjera*:

- High Nutritional Value: Teff is high in protein with a great combination of eight essential amino acids needed for the body's growth and repair. It has high amounts of calcium, manganese, phosphorous, iron, copper, aluminum, barium, thiamin, and vitamin C. The iron from tef is easily absorbed and is also recommended for people with low blood iron levels. Naturally, this grain is very low in saturated fat.
- Gluten-Free: Teff is a gluten-free grain so it can be a great alternative for those living with celiac disease, having gluten intolerance or choosing a gluten-free lifestyle.
- Better Manage Blood Sugars: Teff contains approximately 20 to 40 percent resistant starches and has a relatively low glycemic index (GI) that can help diabetics better to regulate their blood glucose levels.

Teff and celiac disease: This grain has very high calcium content, and contains high levels of phosphorus, iron, copper, aluminum, barium, and thiamin. A big advantage, according to soil and crop, is the fact that the iron from teff is easily absorbed by the body. Teff is high in

carbohydrates and fiber. It contains no gluten, so it is appropriate for those with gluten intolerance or Celiac disease.

A test developed by the Leiden University Medical Center (LUMC) has shown that Teff is completely gluten-free, meaning it can probably be accommodated in the diet of patients suffering from celiac disease. Celiac disease is caused by aberrant T-cell responses to wheat gluten and the gluten-like proteins in barley and rye. The only cure for the disease is a lifelong gluten-free diet. A cereal lacking T-cell-stimulatory peptides would thus be of great value to patients with celiac disease. It is also an alternative grain for people allergic to the gluten in wheat.

According to the proximate nutritional composition (Table 4) white teff enjera shows high iron content than red teff enjera because the iron content of the soil and its possible effect on the iron contents of the grain have been conducted, it may be possible that the high iron content of the soil contributes to the iron content of the grain. Therefore, the red color of red enjera is not due to iron but due to other plant pigments.

When to compare *Mediterranean Diet* and *Ethiopian foods*: both contain cereals and legumes foods with high fiber, high protein and low saturated fats, these are important for healthy food choice, for the prevention of many disease. But in Ethiopia, red meat is a bigger part of the Ethiopian diet compared with the Mediterranean diet, this food is not good choice because contains high amount of saturated fat, leads to increase the risk of cardiovascular disease by increasing cholesterol, TAG, and also increase fat storage, cause obesity and weight gain. These are risk factors for the development of chronic disease such as diabetes mellitus. Therefore, foods with high amount of red meat are not important for the prevention of disease in Ethiopia.

Generally using the glycemic index concept and determined the glycemic index value of Ethiopian traditional foods are important for diet selections, and that GI should be controlled for both treatment and prevention of chronic diseases (diabetes mellitus).

6. CONCLUSIONS

The present study indicates that the scientific basis of the glycemic response of twelve traditional Ethiopian foods. The GI value for twelve tested foods were determined. Results from this study indicates eight foods were low, three foods were moderate and one was high glycemic index value of foods commonly consumed in Ethiopia.

Among the twelve traditional Ethiopian foods eleven foods (Eight were low and three were moderate) had a low to moderate glycemic index; these foods are important for people both with and without diabetes. These include reduction in the risk of developing type 2 diabetes and improvements in metabolic factors associated with long-term complications of type 1 and type 2 diabetes such as reduction of postprandial glycemia and insulinemia, improved glycemic control, improved lipid profile, and reduced risk factors of CVD.

From these traditional Ethiopian foods only one food had a high glycemic index (white bread), and this will likely increase the risk of development of many chronic diseases, especially diabetes mellitus, cardiovascular disease, obesity and cancer.

7. LIMITATIONS OF THE STUDY

- ✚ There is no standardized food processing and preparation methods, so simply traditional way of processing and preparation methods was done at one home, these may not be representative.
- ✚ The study was limited to few traditional Ethiopian foods due to the financial limitation.

8. RECOMMENDATIONS

- ✓ Using glycemic index of foods can help diabetic patients predict their daily diets to control blood glucose levels.
- ✓ In the future study it's recommended that the study should be done in diabetic patient (human model) to explore the beneficial effect of these tested foods.
- ✓ Food processing (preparation and cooking) methods may alter the glycemic index value of foods. Therefore, further study may be recommended to standardize the food processing methods to get the exact glycemic index value of these tested foods.
- ✓ Further study needs to focus measuring glucose concentration for each food could help to explain whether readily absorbable carbohydrate is related to the glucose response and glycemic index.

9. REFERENCES

- Akerberg A, Liljeberg H & Björck I. 1998. An in vitro method based on chewing, to predict resistant starch content in foods allows parallel determination of potentially available starch and dietary fiber. *J Nutr*, 128, 651 – 660.
- Allison DB, Paultre F, Goran MI, Poehlman ET & Heymsfield SB. 1995. Statistical consideration regarding the use of ratios to adjust data. *Int J Obes Relat Metab Disord*, 19, 644 – 652.
- Allen ND. 1997. A new look at dietary carbohydrate: Chemistry, Physiology and Health. *Eur. J Chem. Nutr*, 57, 1 – 7.
- Anderson JW, JA Zeigler, DA Deakins, TL Floure, DW, Dillon, CL Wood & PR Oeltgen. 1991. Metabolic effect of high carbohydrate, high fiber diets for insulin dependant diabetic individual. *Am J Clin Nutr*, 54, 936-943.
- AOAC. 2005. Official Methods of Analysis. Association of Official Analytical Chemists, Washington, D.C.
- AOAC. 2000. Official methods of analysis. 17th ed. Washington, DC: Association of Official Analytical Chemists.
- Asp NG. 1996. Dietary carbohydrates: classification by chemistry and physiology. *Food Chem*, 57, 9 -14.
- Augustin LS, Dal Maso L, La Vecchia C, Parpinel M, Negri E & Vaccarella S. 2001. Dietary glycemic index and glycemic load and bread cancer risk: as case-control study. *Ann of Onc*, 12, 1533–1538.
- Augustin LS, Gallus S, Bosetti C, Levi F, Negri E, Franceschi S, Dak Maso L, Jenkins DJ, Kendal CW & La Vecchia C. 2003. Glycemic index and glycemic load in endometrial cancer. *Int J Can*, 105, 404–407.
- Ayabe T, Tsutsumi O, Sakai H, Yoshikawa H, Yano T, Kurimoto F, Taketani Y. 1997. Increased circulating levels of insulin-like growth factor-I and decreased circulating levels of insulin-like growth factor binding protein-1 in postmenopausal women with endometrial cancer. *Endocr J*, 44, 419 –424.
- Baxter AJ, Coyne T, McClintock C. 2006. Dietary patterns and metabolic syndrome--a review of epidemiologic evidence. *Asia Pac J Clin Nutr*, 15, 134-142.
- Bender DA, 1997. Introduction to nutrition and metabolism. Taylor & Francis Ltd. London.
- Berger M. 1996. Review: the bridge science and patient care in diabetes. *Diab*, 39, 749-757.

- Berteau O. & Stenutz R. 2004. Carbohydrate and energy. *Carbohydr. Res.* 339, 929–936.
- Beulens JW, de Bruijne LM, Stolk RP, Peeters PH, Bots ML, Grobbee DE, van der Schouw YT. 2007. High dietary glyceemic load and glyceemic index increase risk of cardiovascular disease among middle-aged women: a population-based follow-up study. *J Am Coll Cardiol*, 50, 14-21.
- Bohne-Lang A, Lang E, Forster T & von der Lieth CW. 2001. Carbohydrate and glycoprotein three dimensional structures. *Carbohydr Res*, 336, 1– 11.
- Bornet FRJ, Costagliola D, Rizkalla SW, Blayo A, Fontvieille AM, Haardt MJ, Letanoux M, Tchobroutsky G, Slama G. 1987. Insulinemic and glyceemic indexes of six starch- rich foods taken alone and in a mixed meal by type-2 diabetics. *Am J Clin Nutr*, 45, 588–595.
- Brand-Miller JC, Colagiuri S. 1994. The carnivore connection: dietary carbohydrate in the evolution of NIDDM. *Diabet*, 57, 1280–1286.
- Brand-Miller JC, Holt SH, Pawlak DB, McMillan J. 2002. Glyceemic index and obesity. *Am J Clin Nutr*, 76, 2815–2855.
- Brisson J. R. & Carver J. P. 2001. Carbohydrate and protein. *Biochemistry*, 22, 3671–3680.
- Brouns F, Bjorck I, Frayn KN, Gibbs AL, Lang V, Slama G, *et al.* 2005. Glyceemic index methodology. *Nutr Res Rev*, 18, 145–171.
- Byrnes SE, Miller JCB, Denyer GS. 1995. Amylopectin starch promotes the development of insulin resistance in rats. *J Nutr*, 125, 1430–1437.
- Calle-Pascual AL, Gomez V, Leon F. 1988. Foods with a low glyceemic index do not improve glyceemic control of both type 1 and type 2 diabetic patients after 1 month of therapy. *Diabetes Metab*, 14, 629-633.
- Chatwell L, Holla A, Kaufer BB & Skerra A. 2008. Glyceemic effects of carbohydrates. *Mol. Immunol*, 45, 1981–1994.
- Cheatham & Kahn. 2003. “Insulin action and the insulin signaling network.” *Endocr. Rev*, 16, 117-142.
- Chen YJ, Wong SH, Wong CK, Lam CW, Huang YJ, Siu PM. 2008. The effect of a pre exercise carbohydrate meal on immune responses to an endurance performance run. *Br J Nutr*, 9, 1-9.
- Chiasson JL, Josse RG, Gomis R. 2002. Acarbose for prevention of type 2 diabetes mellitus:

- the STOP-NIDDM randomised trial. *Lancet*, 359, 2072-2076.
- Cumming JH & Stephan AM. 2007. Carbohydrate terminology and classification. *Eur J Clin Nutr*, 61(S1), S5-S18.
- David Mandosa. 2008. Revised International Table of Glycemic Index (GI) and Glycemic Load (GL) Values. *Diabet Care*.
- Das SK, Gilhooly CH, Golden JK, Pittas AG, Fuss PJ, Cheatham RA, Tyler S, Tsay M, McCrory MA, Lichtenstein AH, Dallal GE, Dutta C, Bhapkar MV, Delany JP, Saltzman E, Roberts SB. 2007. Long-term effects of 2 energy-restricted diets differing in glycemic load on dietary adherence, body composition, and metabolism in CALERIE: a 1-y randomized controlled trial. *Am J Clin Nutr*, 85, 1023-1030.
- DeRougmont A, Normand S, Nazare JA, Skilton MR, Sothier M, Vinoy S, Laville M. 2007. Beneficial effects of a 5-week low-glycaemic index regimen on weight control and cardiovascular risk factors in overweight non-diabetic subjects. *Br J Nutr*, 1-11.
- Dickinson S, Hancock DP, Petocz P, Ceriello A, Brand-Miller J. 2008. High-glycemic index carbohydrate increases nuclear factor-kappaB activation in mononuclear cells of young, lean healthy subjects. *Am J Clin Nutr*, 87, 1188-1193.
- Dickinson S, Brand-Miller J. 2005. Glycemic index, postprandial glycemia and cardiovascular disease. *Curr Opin Lipidol*, 16, 69-75.
- Englyst KN, Luis & Englyst HN. 2007. Nutritional characterization and measurement of dietary CHO. *Eur J Clin Nutr*, 61(1), 519-539.
- Englyst E, Englyst H, Hudson GJ, Cole TJ & Cummings J. 1999. Rapidly available glucose in foods: an in vitro measurement that reflects the glycemic response. *Am J Clin Nutr*, 69, 448-454.
- Englyst H, Kingman S & Cummings J. 1992. Classification and measurement of nutritionally important starch fractions. *Eur J Clin Nutr*, 46, S33- S50.
- Englyst HN, Veenstra J & Hudson GJ. 1996. Measurement of rapidly available glucose (RAG) in plant foods: a potential *in vitro* predictor of the glycemic response. *Brit J Nutr*, 75, 327-337.
- Englyst K, Vinoy S, Englyst H & Lang V. 2003. Glycaemic index of cereal products explained by their content of rapidly and slowly available glucose. *Brit J Nutr*, 89, 329-340.
- Edcoms. 2007. Review and Analysis of current literature on consumer understanding of

- nutrition and health claims Made on food. COI for FSA.
- Ferlay J, Shin HR, Bray F, *et al.*, 2010. Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. *Int J Cancer*, 127, 2893-2917.
- Farrell JJ, Feldman M, Friedman LS, Brandt LJ (editor). 2010. Digestion and absorption of nutrients and vitamins. Sleisenger & Fordtran's Gastrointestinal and Liver Disease. 9thed. Philadelphia, Pa: Saunders Elsevier: chap 100.
- Feldeisen SE, Tucker KL. 2007. Nutritional strategies in the prevention and treatment of metabolic syndrome. *Appl Physiol Nutr Metab*, 32, 46-60.
- Fernandez G, Velangi A & Wolever TMS. 2005. Glycemic index of potatoes commonly consumed in North America. *J Am Dietet Associ*, 105, 557-562.
- Fiske CH & Subbarow Y. 1925. The calorimetric determination of phosphorous. *J Biol Chem*, 66, 375-400.
- Fontvieille AM, Rizkalla SW, Penformis A. 1992. The use of low glycemic index foods improves metabolic control of diabetic patients over five weeks. *Diabet Med*, 9, 440-460.
- Food and Agriculture Organization/World Health Organization. 1998. Carbohydrates in Human Nutrition. Report of a Joint FAO/WHO Expert Consultation. Rome. *Food and Nutr Paper*, 66, 1-140.
- Foster-Powell K, Brand-Miller J. 1995. International tables of glycemic index. *Am J Clin Nutr*, 62, 871S-893S.
- Foster-Powell K, Holt SH, Brand-Miller JC. 2002. International table of glycaemic index and glycaemic load values. *Am J Clin Nutr*, 76, 5-56.
- Franz MJ, Horton ES, Bantle JP, Beebe CA, Brunzell JD, Coulston AM. 1994. Nutrition principles for the management of diabetes and related complications. *Diabet Care*, 17(5), 490-518.
- Frost G, Wolever TMS, Leeds AR. 1994. Review: The Glycemic Index. Is it time to take a new look? *Dietary Fibre Bibl and Rev*, 67-71.
- Frost G, Domhurst A. 2000. The relevance of the glycaemic index to our understanding of dietary carbohydrate. *Diabet Med*, 17, 336-345.
- Gapsur SM, Gann PH, Lowe W. 2000. Abnormal glucose metabolism and pancreatic cancer mortality. *J Am Med Assoc*, 283, 2552-2558.
- Garzo VG, Dorrington JH. 1998. Aromatase activity in human granulosa cells during follicular

- development and the modulation by folliclestimulating hormone and insulin. *Am J Obstet Gynecol*, 148, 657–662.
- Gavlack, R. E. D. A. Horneck & R. O. Miller. 2005. Soil and Water Reference Methods for the Western Region, 3rd Edition. *West Regi Ext Pub*, WREP-125, Oregon State University.
- Gilbertson HR, Brand-Miller JC, Thorburn AW, Evans S, Chondros P & Werther GA. 2001. The effect of flexible low glycemic index dietary advice versus measured carbohydrate exchange diets on glycemic control in children with type 1 diabetes. *Diabetes Care*, 24, 1137–1143.
- Goni, Garcia-Alonso A & Saura-Calixto F. 1997. A starch hydrolysis procedure to estimate glycaemic index. *Nutr Research*, 17, 427-437.
- Gross G, Boldt DH, Osborne CK. 1984. Perturbation by insulin of human breast cancer cell kinetics. *Cancer Res*, 44, 3570–3575.
- Gupta K, Krishnaswamy G, Karnad A, Peiris A. 2002. Insulin: a novel factor in carcinogenesis. *Am J Med Sci*, 232, 140–145.
- Griffith JA, Ma Y, Chasan-Taber L, Olendzki BC, Chiriboga DE, Stanek EJ 3rd, Merriam PA, Ockene IS. 2008. Association between dietary glycemic index, glycemic load, and high-sensitivity C-reactive protein. *Nutrition*, 24, 401-406.
- Helenius A & Aebi M. 2001. Analysis and validation of carbohydrate. *Science*, 291, 2364- 2369.
- Holt SHA & Brand-Miller J. 1994. Particle size, satiety and the glycemic response. *Eur J Clin Nutr*, 48, 496–502.
- Holt SH, Miller JC, Petocz P. 1997. An insulin index of foods: the insulin demand generated by 1000-kJ portions of common foods. *Am J Clin Nutr*, 66, 1264–1276.
- Irwin T. 2001. A new glycaemic index labeling program for Australia. *Nutrition Builet in*, 26, 317-318.
- Jakobsen MU, Dethlefsen C, Joensen AM, Stegger J, Tjønneland A, Schmidt EB, Overvad K. 2010. Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *Am J Clin Nutr*, 91, 1764-1768.
- Jenkins DJA, Wolever TMS, Taylor RH, Barker H, Fielden H, Baldwin JM. 1981. Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr*, 34, 362–366.

- Jenkins DJA, Wolever TMS, Jenkins AL, Thorne MJ, Lee R, Kalmusky J, *et al.* 1983. The glycemic index of foods tested in diabetic patients; a new basis for carbohydrate exchange favouring the use of legumes. *Diab*, 24, 257–264.
- Jenkins JA, Wolever TMS, Kalmusky J, Giudici S, Giordano C, Wong GS, *et al.* 1985. Low glycemic index carbohydrate foods in the management of hyperlipidemia. *Am J Clin Nutr*, 42, 604–617.
- Jenkins DJA, Wolever TMS, Jenkins AL, Giordano C, Giudici S, Thompson LU, *et al.* 1986. Low glycemic response to traditionally processed wheat and rye products: bulgur and pumpernickel bread. *Am J Clin Nutr*, 43, 516–520.
- Jenkins DJA, Mayer A, Jenkins AL, Wolever TMS, Collier GR, Wesson V, *et al.* 1987. Simple and Complex carbohydrates; lack of glycemic difference between glucose and glucose polymers. *J Clin Nutr Gastroenterol*, 2, 113–116.
- Jenkins DJA, Josse RG, Jenkins AL, Wolever TMS, Vuksan V. 1995. Implications of altering the rate of carbohydrate absorption from the gastrointestinal tract. *Clin Invest Med*, 18, 296–302.
- Jenkins DJ, CW Kendall & M Axelsen. 2000. Viscous and non viscous fibers, non absorbable and low glycemic index carbohydrates, blood lipids and coronary heart disease. *Curr Opin. Lipidol*, 11, 49-56.
- Jenkins AL, Jenkins DJ, Zdravkovitz U, Wursch P, Vuksan V. 2002. Depression of glycemic index by high levels of beta-glucan fiber in two functional foods tested in type 2 diabetes. *Eur J Clin Nutr*, 56, 622–628.
- Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL, Axelsen M. 2002. Glycemic index: overview of implications in health and disease. *Am J Clin Nutr*, 76, 2665–2673.
- Jenkins DJ, Kendall CW, Augustin LS, Vuksan V. 2002. High complex carbohydrate or lente carbohydrate foods? *Am J Med* 113, *Suppl 98*, 30S–37S.
- Jimoh AK, OS Adedliron, SA Adebisi, SA Biliaminu & AB Okesina. 2008. Effect of food processing on glycemic response to white yam meals. *Diabetol Croat*, 37, 67-72.
- Kaaks R, Lukanova A, Kurzer MS. 2002. Obesity, endogenous hormones, and endometrial cancer risk: a synthetic review. *Cancer Epidemiol Biomarkers Prev*, 11(12), 1531–1543.
- Kaufman FR, Epport K, Engilman R, Halvorson M. 1999. Neurocognitive functioning in children

- diagnosed with diabetes before age 10 years. *J Diabet's Complications*, 13, 31–38.
- Key TJ, Silcocks PB, Davey GK. 1997. A case-control study of diet and prostate cancer. *Br J Cancer*, 76, 678-687.
- Kim WY, Kim JE, Choi YJ, Huh KB. 2008. Nutritional risk and metabolic syndrome in Korean type 2 diabetes mellitus. *Asia Pac J Clin Nutr*, 17 Suppl 1, 47-51.
- Klotz DM, Hewitt SC, Korach KS, Diaugustine RP. 2000. Activation of a uterine insulin-like growth factor I signaling pathway by clinical and environmental estrogens: requirement of estrogen receptor-alpha. *Endocr*, 141, 3430 –3439.
- Lairon D. 2008. Macronutrient intake and modulation on chylomicron production and clearance. *Atheroscler Suppl*, 5, 347-349.
- Laville. 2004. Could GI be the basis of simple nutritional recommendation? Invited comment. *Br J Nutr*, 91, 803-804.
- Lavin JH, Wittert G, Sun WM, Horowitz M, Morley JE, Read NW. 1996. Appetite regulation by carbohydrate: role of blood glucose and gastrointestinal hormones. *Am J Physiol*, 271(2 Pt 1), E209-E214.
- Levin RI. 1981. *Statistics for Management*. 2nd ed. Englewood Cliffs: Prentice-Hall.
- Liljeberg H, Bjorck I. 1998. Delayed gastric emptying rate may explain improved glycaemia in healthy subjects to a starchy meal with added vinegar. *Eur J Clin Nutr*, 52, 368-371.
- Liljeberg HGM, Bjorck IME. 1996. Delayed gastric emptying rate as a potential mechanism for lowered glycemia after eating sourdough bread: studies in humans and rats using test products with added organic acids or an organici salt. *Am J Clin Nutr*, 64, 886-893.
- Linda J. Vorvick. 2012. MD, Medical Director and Director of Didactic Curriculum, MEDEX Northwest Division of Physician Assistant Studies, Department of Family Medicine, UW Medicine, School of Medicine, University of Washington. *Am J Clin Nutr*, 12,102-111.
- Lineback DR. 2005. Role of diet in blood glucose response and related health outcomers: summary of a meeting. *Nutr Rev*, 63(4), 126–131.
- Liu S, Willet W, Stampfer M. 2000. A prospective study of dietary glycaemic load, carbohydrate intake and risks of coronary heart disease in US women. *Am J Clin Nutr*,71, 1455-1461.
- Ludwig DS, Majzoub JA,Al-zahrani A,Dallal GE,Blancol & Roberts SB. 1999. High glycaemic index foods, Over eating and obesity. *Pediatrics*, 103(3), 26.

- Ludwig DS. 2000. Dietary glycaemic index and obesity. *J Nutr*, 130, 280-283.
- Ludwig DS. 2002. The glycemic index: physiological mechanisms relating to obesity, diabetes and cardiovascular disease. *JAMA*, 287, 2414-2423.
- Ludwig DS. 2007. Clinical Update; the low glycemic index diet. Comment. *The Lancet*, 369, 890-892.
- MCWilliams M. 2009. Foods; Experimental Perspectives. 6th Edition. Prentice Hall Publishers. London.
- Mercado-Asis, LB, Siguan-Crisaldo A, Flavier AA. 2005. Single blinded randomized clinical trial on comparison of glycemic control and episodes of hypoglycemia in type 2 diabetes mellitus patients on conventional diet versus automatic snacking. *Santo Tomas J of Medic*, 52, 140-146.
- Meyer LH. 1960. Food Chemistry. Reinhold Publishing Corporation, New York, 8.
- Mitchell HL. 2008. The glycaemic index concept in action. *Am J Clin Nutr*, 87, 244S-246S.
- Musey VC, Goldstein S, Farmer PK, Moore PB, Phillips LS. 1993. Differential regulation of IGF-1 and IGF-binding protein-1 by dietary composition in humans. *Am J Med Sci*, 305, 131–138.
- Nestler JE, Powers LP, Matt DW, Steingold KA, Plymate SR, Rittmaster RS, Clore JN, Blackard WG. 2001. A direct effect of hyperinsulinemia on serum sex hormone- binding globulin levels in obese women with the polycystic ovary syndrome. *J Clin Endocr Metab*, 72, 83–89.
- Ngo TH, Barnard RJ, Anton T, *et al.* 2004. Effect of isocaloric low-fat diet on prostate cancer xenograft progression to androgen independence. *Cancer Res*, 64,1252 – 1254.
- Nuttall FQ, Mooradian AD, Gannon MC, Billington C, Krezowski P. 1984. Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. *Diabetes Car*, 7, 465-470.
- Oboh H, A Osagie & A Omotosho. 2010. Glycemic response of some boiled legumes commonly eaten in Nigeria. *Diabet Croat*, 39, 710-716.
- Official Methods of Analysis. Vol 2, 15th edn. 1990. Association of Official Analytical Chemists, Food Composition; Additives; Natural Contaminants. Arlington, Virginia, 783-784.
- Ohtsubo, K. & Marth, J. D. 2006. Carbohydrate structures. *Cell*, 126, 855–867.
- Omorieg ES & AU Osagie. 2008. Glycemic indices and glycemic load of some Nigerian foods.

- Pak J Nutr*, 7, 710-716.
- Osborne DR and Voogt V. 1978. *The Analysis of Nutrients in Foods*. Academic Press Inc. London, 107-232.
- Pawlak DB, Kushner JA, Ludwig DS. 2004. Effects of dietary glycaemic index on adiposity, glucose homeostasis and plasma lipids in animals. *Lancet*, 364, 778–785.
- Phillipe. 2001. “Structure and pancreatic expression of the insulin and glucagon genes.” *Endocr. Rev*, 12, 252-271.
- Pi-Sunyer FX. 2002. Glycemic index and disease. *Am J Clin Nutr*, 76(Suppl), 290S-298S.
- Pocai A, Obici S, Schwartz GJ, Rossetti L. 2005. A brain-liver circuit regulates glucose homeostasis. *Cell Metab*, 1, 53–61.
- Poretsky L, Kalin MF. 2003. The gonadotropic function of insulin. *Endocr Rev*, 8, 132– 141.
- Ramachandran A, Shobhana R, Snehalatha C, Christina A, Murugesan N, Vijay V, Anil K. 2007. Increasing expenditure on health care incurred by diabetic subjects in a developing country – Study from India. *Diabetes Care*, 30, 252- 256.
- Ramon JM, Bou R, Romea S. 2000. Dietary fat intake and prostate cancer risk: a case-control study in Spain. *Cancer Causes Control*, 11, 679 – 685.
- Riccardi G, Rivellese AA, Giacco R. 2008. Role of glycemic index and glycemic load in the healthy state, in prediabetes, and in diabetes. *Am J Clin Nutr*, 87, 269S-274S.
- Ruth L & Diane N. 2003. Prepared by Karin Westberg. Using the glycemic index to compare carbohydrates. www.diabetesdigest.com/dd_nutrition2.htm, [www.diabetesnet.com/food/diabetes diet/ glycemic index.php](http://www.diabetesnet.com/food/diabetes_diet/glycemic_index.php).
- Sartorelli DS, Cardoso MA. 2006. Association between dietary carbohydrates and type 2 diabetes mellitus: epidemiological evidence. *Arq Bras Endocr Metabol*, 50, 415-426.
- Shah M & Garg A. 2004. High fat and high carbohydrate diets and energy balance. *Diabetes Care*, 19, 1142–1152.
- Sievenpiper JL, Jenkins AL, Whitham DL, Vuksan V. 2002. Insulin resistance: concepts, controversies, and the role of nutrition. *Can J Diet Pract Res*, 63, 20-32.
- Siri PW, & Krauss RM. 2005. Influence of dietary carbohydrate and fat on LDL and HDL particle distributions. *Curr Atheroscler Rep*, 7, 455-459.
- Steemburgo T, Dall’Alba V, Gross JL, Azevedo MJ. 2007. Dietary factors and metabolic syndrome. *Arq Bras Endocr Metabol*, 51, 1425-1433.

- Stevenson EJ, Williams C, Mash LE, Phillips B, Nute ML. 2006. Influence of high- carbohydrate mixed meals with different glycemic indexes on substrate utilization during subsequent exercise in women. *Am J Clin Nutr*, 84, 354-360.
- Stryer. 2003. "Chapter 30: Integration of Metabolism." *Biochemistry*, 4th ed. W.H. Freeman & Company, New York.
- Tecator Manual. 1979. Tecator Digestion, Distillation and Titration System. Determination of Kjeldahl Nitrogen by using Kjeltex SystemII. Tecator AB, Hoganas, Sweden.
- Tecator Manual. 1979. Fat extraction using Soxhlet procedure.
- Thorsdottir I, I Bjorck, B Brigisdottir, L Steingrimsdottir & A Flint. 2005. Glycemic index; From research to nutritional recommendation. Ekpresson and Kopiceter, *Denmark*, pp, 1- 84.
- Trenell MI, Stevenson E, Stockmann K, Brand-Miller J. 2008. Effect of high and low glycemic index recovery diets on intramuscular lipid oxidation during aerobic exercise. *Br J Nutr*, 99, 326-328.
- Trout DL, KM Behall & O Osilesi. 1993. Prediction of glycemic index for starchy food. *Am J Clin Nutr*, 58, 873-878.
- Varki A, Cummings R, Esko J, Freeze H, Hart G & Marth J. 2002. Editors. Essentials of Glycobiology. New York: Cold Spring Harbor Laboratory Press.
- Venter CS, Slabber M, Vorster HH. 2003. Labelling foods for glycemic index: Advantages and problems. *S Afr J Clin Nutr*, 16(4),118–126.
- Vonk RJ, Hagedoorn RE, de Graaff R, Elzinga H, Tabak S, Yang YX, Stellaard F. 2000. Digestion of so-called resistant starch sources in the human small intestine. *Am J Clin Nutr*, 72, 432-438.
- Weickert MO, Pfeiffer AF. 2006. Signalling mechanisms linking hepatic glucose and lipid metabolism. *Diabetologia*,49, 1732–1741.
- William B. & Jensen. 2007. The Origin of the Soxhlet Extractor. *J Chem Edu*, Vol, 84(12), 1913-1914.
- Wolever TMS, Jenkins DJA, Jenkins AL, Josse RG. 1991. The Glycaemic index: methodology and clinical implications. *Am J Clin Nutr*, 54(5), 846–854.
- Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG, Leiter LA, Maheux P, Rabasa-Lhoret R, Rodger NW, Ryan EA. 2008. The Canadian Trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in

type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein.
Am J Clin Nutr, 87, 114-125.