Abstract

CORBITT, ALEXIS DANIELLE. Characterization of the Glycemic Index of Raw and Thermally Processed Sweet Potatoes (*Ipomea batatas* L.) (Under the direction of Dr. Jonathan C. Allen.)

Diabetes mellitus is one of the common and serious chronic diseases across the globe. There are 20.8 million children and adults in the United States, or 7% of the population, who have diabetes. While an estimated 14.6 million have been diagnosed, 6.2 million people are unaware that they have the disease.

Type-2 diabetes, non insulin-dependent diabetes mellitus (NIDDM), formerly adult-onset diabetes, is a disease that occurs when the body produces enough insulin but cannot use it effectively. This form of diabetes has primarily been observed to develop in adults over the age of 40, but is gradually becoming a threat across all age groups. About 90% to 95% of people with diabetes have type 2; of which 80 percent are overweight. Diabetes is a life-long disease, but it can be self-managed if people take responsibility for their daily care. Through diet management, exercise, self-testing of blood glucose and in many cases oral medication and/ or insulin, people with diabetes can assist their bodies to keep blood glucose near normal levels.

The objective of our study was to investigate the short term glycemic effect of Beauregard sweet potato *(Ipomea batatas L.*) in normal subjects through measuring the glucose response of sweet potato with and without skin, and of skin alone, in comparison with the more commonly consumed white potato. The study investigated the decrease in glycemic response of white potato vs. white potato plus Caiapo, an antidiabetic acidic glycoprotein component in a Japanese sweet potato cultivar. The first study focused on C-peptide and insulin analysis via Enzyme-Linked ImmunoSorbent Assay (ELISA) (ALPCO Diagnostics) from subjects at 0, 60, and 120 minutes after consumption of potato samples. Glucose levels were measured at interval times of 0, 30, 60, 90, 120 minutes post prandially with glucometers (Therasense). These data

illustrated that differences in insulin, and C-peptide response due to sweet potato and white potato were constant over time resulting in graph parabola trends similar to glucose response. The comparison of C-peptide, serum insulin, and glucose response showed that the mechanism for the hypoglycemic effect in sweet potato or Caiapo is not due to improved insulin production, response to blood glucose, or increased insulin uptake by target cells. The study suggested that the blood glucose lowering effects occur in the small intestine (duodenum) by retarding absorption of glucose to the bloodstream.

The subsequent study investigated the thermo-stability, through cooking, of the low glycemic index effect previously determined in Beauregard sweet potato by measuring the glycemic index after baking at 190 °C for 1 hour, steaming at 100 °C for 45 minutes, dehydration at 40 °C overnight, and microwaving for 5 minutes. Participants consumed 25 g of available carbohydrate, determined through AOAC proximate analysis of lipid, ash, moisture, and total dietary fiber levels in each preparation method. Comparing the heat treatments (steaming, baking, dehydrating, and microwaving) of Beauregard sweet potato skin and flesh illustrated the extent to which cooking affects the glycemic index. Results showed that steamed skin, baked skin, and dehydrated flesh do not have statistically different glycemic indices ($p > 0.05$) from those of raw sweet potatoes ($n=12$). Dehydrated sweet potato flesh, along with the aforementioned cooked sweet potatoes, retained the low glycemic index of raw sweet potatoes. The glycemic index of steamed flesh elevated to that of a medium glycemic index food. Baked and microwaved flesh also elicited blood glucose responses equivalent to that of medium glycemic index foods. This information will lead to further understanding of preferred foods for diabetics to assist in blood glucose management.

Understanding the glucose response and glycemic index elicited by NC sweet potatoes will improve recommendations for their health promoting properties, such as reducing the risk of diabetes or insulin resistance.

CHARACTERIZATION of the GLYCEMIC INDEX of RAW AND THERMALLY PROCESSED SWEET POTATOES (*Ipomea batatas* L.)

By

ALEXIS DANIELLE CORBITT

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APPROVED BY:

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Dr. Leon C. Boyd Dr. Van Den Truong

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Dr. Jonathan C. Allen
Chairman of Advisory Committee

Biography

Alexis Danielle Corbitt was born on May 30, 1984 in St. Croix, United States Virgin Islands. She is the only daughter of Archie (Guy) Corbitt Jr. and Pauline B. Corbitt. After finishing elementary school at Pearl B. Larsen, she completed junior high school at Elena Christian, and secondary school at the St. Croix Educational Complex. She received the early admission scholarship to the University of the Virgin Islands where she began her Bachelor of Science studies in Biology. During this time, she received the Park Scholarship to attend North Carolina State University to complete her studies. She joined Alpha Kappa Alpha Sorority, Incorporated, Peer Mentor Program, Undergraduate Student National Dental Association (USNDA), the Wolfpack Club, and Black Student's Board during undergraduate years at NC State. She served as an ambassador for the College of Agriculture and Life Sciences for two years and vice president of USNDA. Research under the direction of Dr. Jonathan C. Allen during the summer of 2005 exposed her to studies in food science and nutrition. She began graduate coursework during her senior year to expedite the completion of graduate school. Alexis completed her B.S. in Biological Sciences at North Carolina State University, Raleigh, NC in 2006. After graduation, she continued research with Dr. Allen as a graduate research assistant. Joining organizations like the Food Science Club and Minorities in Agricultural, Natural Resources, and Related Sciences (MANNRS) broadened her knowledge of the field and made her continued studies enjoyable. She was awarded her Master of Science degree in Nutrition in August 2007. Alexis was accepted to the University of North Carolina at Chapel Hill's dental school and plans to improve dental health care in underserved North Carolina.

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Chapter I

Literature Review

Introduction

Consumption of low glycemic index foods is suggested to be effective for the prevention and control of diabetes (Little, 2003). The Glycemic Index (GI) expresses the rise in blood glucose elicited by a carbohydrate food as a percentage of the rise that would occur if an equal amount of carbohydrate from white bread or glucose was consumed (Brand-Miller et al., 2003). Foods with low glycemic indices provide a slow release of glucose into the blood, thus preventing a rapid increase in glucose levels which should be avoided by diabetic patients. Previous studies indicate a range of glycemic indices for sweet potatoes across various cultivars to be 48 to 78 (Foster- Powell et al, 2002).

Several reviews have been published discussing the health benefits from consuming low glycemic index (GI) foods for diabetics (American Diabetes Association, 2006). Low GI foods are able to release glucose in such a manner that prevents hyperglycemia for diabetics. Consumption of foods with these properties have been shown to reduce post prandial hyperglycemia (Brand-Miller et al., 2003).

This review will discuss carbohydrate metabolism in diabetic patients and the possible pharmacological effects from the glycemic index of sweet potatoes vs current diabetic therapy and complications. Research is needed to differentiate the short and long term effects from low glycemic index (GI) diets for possible nutraceutical effects and bioactive components (Jenkins et al., 2002). It is also not known whether the low GI measured in some sweet potato cultivars is maintained in cooked products.

Carbohydrate Metabolism

Carbohydrates are the macronutrients that are the primary source of energy for human metabolism. In general, carbohydrate intake should be 50%-60% of total calories (5-13 g/kg)

(Ash, 2005). Glucose, known as “blood sugar” and “dextrose” on food labels can be used by any cell and is absorbed in the small intestine. Dietary sources of carbohydrate are fruits and vegetables, corn syrup, lactose, sucrose, fiber and starches. Starch is the polysaccharide form of glucose and energy storing component for plants. Spare roots, such as sweet potatoes, are vegetables which are very rich in starch. Starches have different thickening and gel-forming characteristics based on number of glucose molecules and degree of “branching”. Pancreatic amylase digests the polymer into dextrins and maltodextrins. Brush border hydrolases of the small intestinal lumen further digest these disaccharides into glucose.

Polysaccharides and disaccharides must be digested into monosaccharides of glucose prior to absorption. The key enzymes in these processes are the brush border hydrolases, which include maltase, lactase and sucrase. Dietary lactose and sucrose are digested by their respective brush border enzymes. Starch, as discussed previously, is first digested to maltose by amylase in pancreatic secretions and, in some part, by salivary amylase. Dietary lactose, sucrose, and maltose derived from digestion of starch, diffuse into the small intestinal lumen and come in contact with the surface of absorptive epithelial cells covering the villi where they engage with brush border hydrolases. Maltase cleaves maltose into two molecules of glucose, lactase cleaves lactose into a glucose and a galactose and sucrase cleaves sucrose into a glucose and a fructose. At this point, the monosaccharides of glucose can be absorbed into the bloodstream. The glucose transporter (GLUT-1) mediates transport of glucose down its concentration gradient; thus, acting as it's a facilitative transporter.

Glucose is absorbed by muscle and adipose cells via GLUT 4 transporters. Transporter activity is contingent upon the binding of insulin to cell surface insulin receptors. Non diabetic individuals are able to bind insulin that is secreted by groups of cells within the

pancreas called islet of Langerhan cells. Insulin is released in the presence of glucose in the bloodstream. Once released, insulin binds to specific insulin receptors, such as those on muscles, and activates the GLUT 4 transporters by increasing the quantity present on the cell surface for glucose absorption.

Diabetic Carbohydrate Metabolism

Both insulin resistance and impaired insulin secretion are required for diabetes mellitus to become manifest. Insulin is produced by the beta cells in the islets of Langerhans in the pancreas. When glucose enters the blood, the pancreas should automatically produce the appropriate amount of insulin to stimulate the absorption of glucose into cells. As long as the pancreatic beta cells can compensate for the degree of insulin resistance, glucose tolerance remains normal (Groop, 1999).

Type I Diabetes Mellitus (IDDM)

People with type 1 diabetes mellitus (previously known as insulin-dependent or childhood-onset; IDDM) produce very little or no insulin from the ß cells of the pancreas. The cells of patients with type 2 diabetes are resistant to the insulin the pancreas secretes. The disease causes hyperglycemia (high blood glucose levels). Certain complications include retinopathy, nephropathy, and neuropathy. Some people with diabetic retinopathy have blood vessels that may swell and leak fluid. In others, abnormal new blood vessels grow on the surface of the retina. Diabetes is the leading cause of blindness among American adults (National Eye Institute, 2006). Diabetes complicated by diabetic neuropathy is a risk factor initiating chronic foot ulceration, which may lead to amputation (Young et al., 1994).

Without daily administration of insulin, Type 1 diabetes is rapidly fatal. Symptoms include excessive excretion of urine (polyuria), thirst (polydipsia), constant hunger, weight

loss, vision changes and fatigue. These symptoms may occur suddenly. IDDM results when the immune system attacks and destroys the insulin-producing ß cells of the pancreas. Cells in the body are unable to take up glucose from the blood for use as an energy source. The results of this attack are a pancreas that produces little to no insulin and an inability to regulate the level of sugar in the blood (Scriver et al., 1995). It is hypothesized that environmental factors trigger the immune system to destroy the insulin-producing cells.

Type II Diabetes Mellitus (NIDDM)

Type 2 diabetes (formerly called non-insulin-dependent or adult-onset) results from the body's ineffective use of insulin in skeletal muscle, adipose tissue, and the liver (Groop, 1999). Type 2 diabetes accounts for 90% of people with diabetes around the world. About 80% of type 2 diabetics are overweight, largely the result of excess caloric intake and physical inactivity. Symptoms may be similar to those of type 1 diabetes, but are often less marked. Clustering of type 2 diabetes into certain ethnic populations and families points to a strong genetic background for the disease. However, environmental factors such as obesity and a sedentary lifestyle are usually required to unmask the genes (Groop, 1999).

The pathogenesis of type 2 diabetes involves insulin resistance, increased hepatic glucose output, and impaired insulin secretion (Ludvik et al., 2003). The disease may be diagnosed several years after onset, once insulin resistance has already become manifest. Until recently, this type of diabetes was seen only in adults but it is now also occurring in obese children. The majority of patients with type 2 diabetes are obese, which aggravates insulin resistance (Campbell & Carlson 1993; Bogardus et al., 1985). Several who are not obese by standard weight criterion may have a higher percentage of adipose tissue concentrated in the abdominal region (Kissebeh et al., 1982).

Impaired insulin-stimulated glucose metabolism (especially non-oxidative) in skeletal muscle represents a key feature of type 2 diabetes and is observed in the pre-diabetic state. It is not clear whether this is an inherited defect of the muscle or develops secondary to abdominal obesity. The latter hypothesis of abdominal obesity and low metabolic rate seem to precede the development of insulin resistance in offspring of type 2 diabetic patients (Groop, 1999). Early in the disease, insulin resistance is present and patients are hyperinsulinaemic, but not hyperglycemic. Over time, compensatory mechanisms break down and patients progress to type 2 diabetes (Groop, 1999). Insulin resistance in diabetic patients leads to impairment of both the suppression of glucose production and stimulation of glucose uptake. Hepatic glucose production remains high despite increasing concentrations of insulin.

Patients with type 1 diabetes require exogenous insulin to live; people with type 2 diabetes can be treated with oral medications, subcutaneous insulin, and/ or controlled diets, but may also require blood pressure control. Diabetes prevention interventions include: Screening for retinopathy (which causes blindness); blood lipid control (to regulate cholesterol levels); nephropathy (diabetes-related kidney disease), and neuropathy (peripheral nerve complications) (Young et al., 1994).

Global Diabetes Impact

Diabetes mellitus is one of the more common and serious chronic diseases across the world. "The World Health Organization (WHO) estimates that more than 180 million people worldwide have diabetes. This number is likely to more than double by 2030. WHO projects the diabetes death rates to increase by more than 50% in the next 10 years. Most notably,

diabetes deaths are projected to increase by over 80% in upper-middle income countries between 2006 and 2015" (WHO, 2006).

There are 20.8 million children and adults in the United States (7% of the population) who have diabetes. While an estimated 14.6 million have been diagnosed, unfortunately, 6.2 million people (nearly one-third) are unaware they have the disease (American Diabetes Association – ADA, 2007). Its prevalence is increasing throughout America and developed countries. Of people under the age of 20, 0.22% were diagnosed with diabetes mellitus, 9.6% are diagnosed in the "over 20" age group, and 20.9% are diagnosed who are over 60 years of age. Of men and women, 10.5% and 8.8% respectively, are diagnosed with diabetes, making it a very serious concern.

Clinically-based reports and regional studies suggest that type 2 diabetes, although still rare, is being diagnosed more frequently in children and adolescents, particularly in American Indians, African Americans, and Hispanic/Latino Americans (American Diabetes Association, 2007). The increased incidence of diabetes may be directly linked to the changed in dietary consumption patterns. The insurgency of the fast food industry over recent times may produce epidemic increases of obesity (Bahado-Singh, 2006).

Diabetes Medications

Diet and exercise are the foundation for management of hyperglycemica in type II diabetes mellitus. Patients who do not achieve adequate glycemic control through these methods are candidates for pharmacological agents to help manage glucose levels. There are several medications for the control of diabetes. Combination treatments with oral pills or strict subcutaneous insulin are today's currently accepted methods for the management of diabetes. Insulin is the more common form of diabetes control used by most patients. Oral

or intravenous methods of improving insulin levels in the body allow the proper amount of glucose into the body's cells and control of blood glucose levels. Pharmacological treatment options include orally administering sulfonylurea, a biguanide (metformin), α-glucosidase inhibitor (acarbose), or a thiazolidinedione (troglitazone). Even though pharmacological therapy is practiced, attention to diet and activity must be observed (Prisant, 2004).

Since the 1950's, sulfonylureas have been used to stimulate the beta cells of the pancreas to release more insulin. Earlier agents, such as chlonpropamide, caused hypoglycemic with prolonged treatment. Glipizide (brand names Glucotrol and Glucotrol XL), glyburide Micronase, Glynase, Diabeta, and glimepiride (Amaryl) are the current second generation sufonylureas that have the same glucose reducing mechanism of action. They are traditionally the first line of therapy for diabetics. According to Lebovitz (1995), they may also increase insulin receptor sensitivity.

Metformin (brand name Glucophage) is a biguanide that may also be opted as a first-line pharmacological treatment for patients with NIDDM. Biguanides lower blood glucose levels primarily by reducing the hepatic glucose production. Metformin also helps to lower blood glucose levels by making muscle tissue more sensitive to insulin for improved glucose intake at the cellular level. It may be used in monotheraphy or in conjunction with insulin treatments. Gastrointestinal side effects that include nausea, diarrhea, and abdominal discomfort are seen in 30% of patients (Vijan et al., 1997).

Acarbose (brand name Precose) and meglitol (Glyset) are alpha-glucosidase inhibitors. These drugs slow the digestion of dietary carbohydrates in the small intestine. Delay of glucose absorption into the blood stream and a decrease in postprandial blood glucose are observed through their mode of action. Prescription details state they should be

taken with the first bite of a meal. Reported gastrointestinal side effects include pain, flatulence, and diarrhea (Vijan et al., 1997).

Rosiglitazone (Avandia), troglitazone (Rezulin), and pioglitazone (ACTOS) form a group of drugs called thiazolidinediones. These drugs help improve insulin sensitivity in muscle and adipose tissue and inhibit hepatic glucose production. Caution should be taken when using a combination of troglitazones and insulin as they can produce hypoglycemia. Rosiglitazone may also increase risk of cardiovascular disease and myocardial infaction (Nissan & Wolski, 2007).

Pramlintide (brand name Symlin) is a recently FDA approved synthetic form of the hormone amylin, which is produced along with insulin by the beta cells in the pancreas. Amylin, insulin, and glucagon work in an interrelated fashion to maintain normal blood glucose levels (ADA, 2007).

When combination therapy fails to achieve patients' glycemic targets, treatment may be changed to strict daily use of subcutaneous insulin. Optimal modern therapy of type 2 diabetes uses a "treat-to-target" approach of moving patients along a sequence of therapies to achieve better diabetes control (Blonde et al., 2002). Therapy may be intensified as needed with twice daily mixed insulin, three times daily insulin therapy, or multiple daily injections. New methods of reducing the amount of insulin required recommends mixing long-acting or short acting insulin with dosages of sugar in the form of juices, sodas, etc., to prevent hypoglycemia and hyperinsulinemia (Shohet, 1995). This methodology aims to reverse the negative effects already caused by diabetes. According to Shohet, when a combination of insulin-plus-sugar is prescribed in precise amounts and adjusted daily based on both blood

and urine tests for sugar, not only is hypoglycemia prevented, but also the need for continued insulin can be steadily and predictably reversed (Shohet, 1995).

Predictors of the Disease

BMI

Body mass index (BMI) is the strongest risk factor for the development of NIDDM (Rios, 1998). Both obesity and NIDDM are major causes of morbidity and mortality from atherogenic macrovascular disease. Accepted etiological factors include genetic, autoimmunity, viral infections, and obesity, the latter of which clearly contributes to insulin resistance (Walter et al., 1991). The risk of developing NIDDM is more closely associated with the regional distribution of body adipose tissue. Visceral obesity is correlated to a higher degree of risk than peripheral obesity due to metabolic and circulatory changes. Visceral body fat accumulations generate intra-abdominal, high hip-waist ratios associated with high risk of cardiovascular disease (Chan et al., 1994). The change in population of lipoproteins in the blood and alterations in the levels of oxidative stress lead to the development of insulin resistance, cardiovascular and macrovascular risk (Rios, 1998). Increased flow of free fatty acids to the liver lead to the development of insulin resistance and increased lipoprotein synthesis, resulting in glucose intolerance, hypertension, and hyperlipidaemia (Bonora, 1997). It is hypothesized that increased adipocytokine production (TNF-alpha or leptin) might have unfavorable circulatory effects of further contribution to insulin resistance and hypertension (Rios, 1998). Most important, there is evidence that weight loss, even by only ten pounds, may reverse or prevent virtually all circulatory and metabolic instability caused by obesity-insulin resistance syndrome (Albie et al., 1995).

Family

It is now well accepted that the lifetime risk of any offspring developing diabetes is 40% if one parent has diabetes, with risk being slightly higher if the mother has the condition (Groop, 1999). Moreover, it is not clear what is inherited. Current hypotheses take account of the predisposition to obesity, insulin resistance, or a link between the two, and a predisposition to impaired Beta-cell function.

Groop (1999) suggests that the thrifty gene, hypothesized from the increased type 2 diabetes prevalence on the island of Nauru, allows for storage of energy in abdominal fat. The evolutionary advantage of maximizing survival from a non constant food supply can bring about the pathogenesis of insulin resistance in type 2 diabetes. Conversely, in an affluent society, this gene should not be required but would be deleterious and associated with impaired health.

Research by Morris et al. (1989) found a relationship between obesity, heredity, and risk of diabetes. The occurrence of diabetes increased consistently with increasing reports of either obesity or family history by a study with 32,662 white women in the United States and Canada who were members of the TOPS (Take Off Pounds Sensibly) Club, Inc. Heredity and obesity appeared to have independent risks for diabetes. Women with a family history of diabetes and overweight reported overall odds ratio of 22.8 when compared to women with no family history and only 10% about ideal weight.

Diabetes Health Complications

Complications of persistent hyperglycemia due to diabetes mellitus have been shown to lead to vascular complications involving large and small blood vessels, such as arteriosclerosis, glomerulosclerosis, and retinopathy (Covington, 2001). Diabetic

neuropathy, characterized by pain and parasthesias, is among the most frequent complications of longstanding, poorly controlled diabetes and is often associated with a reduction in physical activity with sleep disturbances (Berman et al., 1999; Hamza et al., 2000).

Nephropathy

Optimization of glycemic control acts as primary prevention of diabetic nephropathy (Klein et al., 1995). Renal disease in patients with type 2 diabetes mellitus is becoming increasingly recognized. Renal disease was previously more closely associated with type 1 diabetes, but evidence has since revealed similar risk in both groups of patients (Ritz & Orth, 1999). Cross-sectional and longitudinal studies have identified a number of factors that may increase the progression of renal failure in type 2 diabetics (Kirchner, 2000). These factors take account of elevated blood pressure, poor glycemic control, smoking, and black or Native American ethnicity. Less defined risk factors are male gender, high intake of dietary protein, and hyperlipidemia. Hyperlipidemia is a risk factor for arthrosclerosis as well. The recent U.K. Prospective Diabetes Study found that moderately lowering blood pressure led to major reductions in the risk of renal and cardiovascular events. Research noted that blood pressure control was more beneficial than tight glucose control in preventing adverse secondary outcomes (Ritz & Orth, 1999).

Patients with microalbuminuria (urinary albumin excretion rate of 20 to 200 mg per 24 hours) are at high risk for renal complications. Proteinuria has also been identified to be an early sign of diabetic nephropathy (Vijan et al., 1997). Furthermore, the high rate of micro-and macrovascular problems that occur in type 2 diabetics cause incidences such as foot problems, impotence, diarrhea, constipation, coronary heart disease, and cerebrovascular

disease. The creation of vascular access is recommended to patients who show indications of failing kidneys and general circulation.

Retinopathy

Controlled trials demonstrated that improving glycemic control can reduce the incidence of diabetic retinopathy (Vijan et al, 197). Improvement of glycemic control also slows the advancement of established retinopathy in diabetic patients. Progression of the "diabetic eye disease" to proliferative retinopathy and macular edema frequently leads to severe visual loss (Vijan et al., 1997). Randomized controlled trials have shown that laser therapy of proliferative retinopathy significantly reduces the incidence of visual loss in patients with diabetes (Vijan et al., 1997).

Neuropathy

Fifteen percent of patients with type II diabetes mellitus for more than 15 years develop peripheral nephropathy (Vijan et al., 1997). Diabetes patients are also at risk for diabetic foot ulcers. A diabetic foot ulcer is defined as any interruption of the integrity of the skin that extends through the entire dermis. The pathology of neuropathy is the slowing of nerve conduction velocity. Nerve fiber regeneration via nerve growth factors slows the progression of neuropathy and aid in the prevention of diabetic foot ulcers (Sima, 1988; Greene, 1996). Drugs capable of nerve regeneration are not yet available for use in the United States.

Minerals

The clinical manifestation of the diabetes mellitus as a heterogeneous disease includes hyperglycemia, glycosuria, altered protein fat, and carbohydrate metabolism, as well as chronic complications resulting from macro- and microvascular pathology. Alterations in

the metabolism of several trace elements; copper, zinc, manganese, and the macro-element magnesium, have been associated with impaired insulin release, insulin resistance, and glucose intolerance (Walter et al., 1991). It had been suggested that complications are more pronounced when metabolic control is compromised. The stepwise regression analysis of the study performed by Walter et al revealed age, obesity, and duration of the disease as non-significant predictors of mineral status in diabetics.

Overall, diabetic subjects had higher plasma copper concentrations compared to control participants. Copper levels were higher in diabetic women. Type 1 diabetic subjects had lower concentrations of copper in muscle biopsies, suggesting copper depletion in the muscle. Furthermore, plasma zinc concentrations and the zinc-copper ratio were lower in diabetic subjects. Although a 24-hour recall of food intake did not reveal differences in mineral intake between diabetic and non-diabetic subjects, the difference in zinc levels could be due to the nearly six-fold increase in urinary zinc excretion in diabetic subjects. This finding could be disconcerting for diabetics because thymulin, a biologically active zinc-dependent thymic hormone involved in the maturation and differentiation of the thymus-derived T-lymphocyte line, is reduced in diabetes (Mocchegiani et al., 1989).

Obese diabetic subjects had lower plasma magnesium than normal-weight diabetic subjects (Walter et al., 1991). A positive correlation was found between duration of diabetes and plasma magnesium levels. Hyperzincuria and hypermagesuria were evident in diabetic compared with control subjects (Walter et al., 1991).

Magnesium

Diabetic magnesium deficiency has a negative effect of the post-receptor signaling of insulin, which can further compromise glucose metabolism (Lopez-Ridaura, Ruy et al.,

2004). Magnesium supplementation has a beneficial effect on insulin action. Magnesium intake is positively associated with intakes of fiber and inversely associated with intakes of fat and processed meat (de Valk, 1996; Paolisso, 1992). Consistent with previous data, Fung et al (2003) found an inverse association between magnesium intake and fasting insulin levels. The inverse association between magnesium intake and risk of type 2 diabetes is strong among men. Intracellular magnesium is a critical cofactor for several enzymes in carbohydrate metabolism (Paolisso, 1990). Those involved in tyrosine-kinase have been shown to affect the post receptor pathway of insulin in muscle cells (Paolisso, 1990). Altered mineral status of diabetics may be due in part to nephropathy.

Iron

Hepatic iron overload, steatosis (cellular lipid retention), and increased serum ferritin levels suggests a role of insulin resistance in iron metabolism (Gostout et al., 2000). Type II diabetes is one of the metabolic conditions with the higher rate of insulin resistance and is frequently associated with increased serum ferritin levels. There exists a relationship between glucose metabolism, fatty liver, serum ferritin, and hepatic iron activity in diabetic patients (Wilson et al., 2003). The manifestation of insulin resistance favors the accumulation of free fatty acid in the liver and increases the risk of steatosis. Iron reduction therapy in hereditary hemochromatosis and transfusional iron overload is associated with improved glucose tolerance and reduced incidence of secondary diabetes. Trials of iron reduction therapy in diabetes mellitus, although limited and inconclusive, have shown clinical improvement in some patients (Wilson et al., 2003).

Since excess glucose is present in the blood during diabetes, glucose reacts with hemoglobin to form glycosylated hemoglobin (Al Yassin & Ibrahi, 1981; Sheela & Augusti,

1992). Venkateswaran and Pari (2002) demonstrated that decreasing the levels of glucose can reduce the level of glycosylated haemoglobin in diabetic rats.

Non Pharmacological Diabetes Mellitus Treatments

Ginseng Species

Medical plants play an important role in the management of diabetes especially in developing countries where resources are scarce (Bnouham et al., 2006). Ginseng root, often described as "king herb", is a traditional Oriental medicinal plants used in many countries. It is now cultivated in China, Korea, Japan, Russia, and in the United States and Canada (Lee, 1992; Xie et al., 2005). The active component of the herb is attributed to ginesnosides, a family of steroids named steroidal saponins (Attele et al., 1999; Huang, 1999). Ginseng is noted for elevating mood, improving psycho-physiological performance and physical activity, and reducing body weight (Sotaniemi, 1995). Clinical evidence suggests the blood glucose lowering effects can be credited to panaxans I, J, K, and L of ginsenoside Rb-2 for type 1 diabetic patients (Konno, 1984; Konno et al., 1985; Yokozawa et al., 1985).

According to Vusjan et al. (2001) American ginseng, when consumed 40 minutes before consuming a test meal, significantly lowers postprandial blood glucose levels in non-diabetic and type 2 diabetic patients. Conversely, reduced postprandial glucose levels were not observed when ginseng was consumed with added with meals. Proposed mechanisms are that ginseng eaten alone retards the digestion of food, thus reducing the rate of carbohydrate absorption into portal hepatic circulation (Yuan, 1998; Suzuki, 1991), or affects nitric oxide (NO) mediated glucose transport (Ohnishi et al., 1996; Hasegawa et al., 1994; Gillis, 1997; Roy et al., 1998), or alters secretion of NO-mediated insulin (Kimura et al., 1981).

Considering the risk of hormone like or hormone- inducing effects, some authors recommend limiting medicinal treatment to three month (Schulz et al., 1998).

***Momordica charantia* (Bitter Melon)**

Referred to as a vegetable and a fruit, *Momodrica charantia* is widely cultivated across Asia, Africa, and South America (Dey et al., 2002). Common names include breadfruit, balsam pear, karela, and bitter melon. Of its confirmed anti-diabetic components, alcohol extracted charantin is comprised of mixed steroids and was found to be more potent than the oral hypoglycemic pharmacological drug tolbutamide in an animal study (Sakar, 1996). There exists an insulin-like polypeptide, polypeptide P, with a similar structure to bovine insulin which decreased blood sugar levels when injected into type 1 diabetic patients (Baldwa, 1977). Welihinda et al. (1986) showed that 57 g of the juice improved glucose tolerance in 73 percent of type 2 diabetic patients.

Trigonelle foenum graecum (Fenugreek)

Particularly utilized in India, the defatted portion of *Trigonella foenum graecum* seed has been used as a remedy for diabetes (Miller, 1998). Alkaloid trigonelline, nicotinic acid, and coumarin are contained in this fraction. Consuming 1.5- 2.0 g daily of the defatted seed was shown to reduce fasting and postprandial blood glucose levels, glucagon, somatostatin, insulin, total cholesterol, and triglycerides, and increased HDL-cholesterol levels in diabetic dogs (Ribes et al., 1984). The fact that seeds are comprised of 50 percent fiber alludes to the possibility of glycemic influences related to total dietary fiber.

Coccinia indica

Administering of *Coccinia indica* leaf extract to normal and diabetic animals demonstrated significant hypoglycemic and antihperglycemic outcomes and reversed

biochemical difficulties (Hossain, 1992). Significant reduction in blood glucose, glycosylated haemoglobin and an increase in total haemoglobin and plama insulin were reported after oral doses of 200 mg/ kg of the ethanol extract was given to diabetic animals (Venkateswaran & Pari, 2002). Experimental diabetes studies marked altered enzymes for glucose and fatty acid metabolism in diabetics. Plausible mechanisms to *Coccinia indica* hypoglycemic effects are potentiating secretion of insulin from the β-cells or release from bound form which was illustrated by increased insulin levels in diabetic rats (Venkateswran & Pari, 2002).

Chromium

Chromium is an essential micronutrient for humans (Dey et al., 2002). Epidemiological studies identify chromium as a significant determinant of insulin sensitivity, as it is a cofactor in insulin-regulating activities via facilitating insulin binding and subsequent uptake of glucose into the cell (Offenbacher et al., 1980). Supplementation of chromium had demonstrated fasting glucose lowering effects, decreasing total cholesterol and triglycerides, while increasing HDL cholesterol in normal, elderly, and type 2 diabetic subjects (Mooradian et al., 1994). Lee and Reasner (1994) reported a decrease in triglycerides, but no statistical difference between control and chromium-treated subjects with respect to measured parameters of glucose control when given 200 mcg/ day of chromium picolinate for six months. Although there is no recommended daily allowance for chromium, over 200 µg/day appears necessary for optimal blood sugar regulation (Dey et al., 2002). The Adequate Intake (AI) set by the DRI committee of Institute of Medicine is 20- 35 µg/day (Food & Nutrition Board and Institute of Medicine, 2000).

Glycemic Index Therapy for Diabetes Mellitus

Whereas pharmacological therapies are effective for diabetes control, which function by reducing blood glucose levels along with optimal diet, the GI of foods has been associated with prevention and control of diabetes mellitus (Brand-Miller et al., 2003). GI is a ranking of carbohydrate containing foods according to their immediate effects on blood sugar levels (Williams, 2004). High glycemic indices are characterized by the greatest elevation in blood sugar when a food is eaten. Pure glucose is used as the standard to which other foods are compared and is given the glycemic index of 100. The glycemic loads of foods, meals, and diets are calculated by multiplying the glycemic index of the constituent foods by the amounts of carbohydrate in each food and then totaling the values for all foods over a meal or a day. Although all dietary carbohydrates provide the same amount of energy, they are not all handled with equal efficiency by the body (Zakir, 2005). The American Diabetes Association says that the use of glycemic index and glycemic load may provide a modest additional benefit over that observed when total carbohydrate is considered alone. "Conventionally, management of diabetes mellitus was to reduce carbohydrate consumption to avoid increased blood sugar levels. Further studies emphasize that carbohydrates with low glycemic indices may be helpful in controlling blood glucose" (Little, 2002). The ADA acknowledges that use of low-GI food may reduce postprandial hyperglycemia, but asserts that there is not sufficient evidence of long term benefit to recommend their use as a primary strategy (ADA, 2002). According to Little et al., "the incremental benefit is clinically significant and similar to that offered by newer pharmacological agents."

Studies with large numbers of diabetics have indicated that those who maintain their blood sugar under tight control best avoid complication from the disease (Gilbertson et al.

2001). Difficulty managing the disease may be associated with the consumption of carbohydrates with high glycemic indices. Research into GI has clearly proven that equal amounts of carbohydrate do not elicit similar glycemic responses. The classification of simple and complex carbohydrates has been dispelled for the more appropriate high and low glycemic index foods. Starch digestion is the rate-limiting step for the determination of the glycemic index of most foods (Brand et al., 1985). Fried starches are exceptions to this rule because fat decreases stomach emptying. High total dietary fiber in starches can also alter the absorption of glucose thus skewing glycemic index predictions based solely on carbohydrate content (Brand et al., 1985; Bahado-Singh, 2006).

Factors Affecting GI

The blood glucose absorbed from any given food is affected by physiological and nutritional factors, which include the digestibility of the starch, interactions of starch with proteins, amounts and kinds of fat, sugar and fiber in the presence of constituents, and the level and type of food processing (Bahado-Singh, 2006; Whitney et al., 2002; Englyst et al., 1987). It was also observed that changes in the physiological state of the food, from green to ripe, increases its GI (Bahado-Singh, 2006). Several other factors are the source and class of carbohydrate, resistant starches, amylose and amylopectin levels, fiber content, and cooking.

Preprandial Blood Glucose

Normal fasting glucose is 4.44 to 5.55 mmol/L, equal to 90 mg/dL to 95mg/dL. Fasting glucose levels from 110 to 125 mg/dL identifies patients with impaired fasting glucose (Prisant, 2004). Studies reveal an inverse relationship between preprandial glucose levels and glycemic response. Wolever et al. (1985) attributed this association as the result of increased spilling of glucose into the urine due to a saturation of blood glucose and

decreased renal capacity for reabsorption. A reduced retention of glucose due to the renal threshold being exceeded causes a decrease in observed glycemic response with increasing fasting glucose levels of diabetic patients. However, this low glycemic response does not negate the fact that glucose levels are at unhealthy concentrations in the blood.

Class of Carbohydrate

The classes of carbohydrates are the monosaccharides, such as glucose, and disaccharides, such as fructose. Free sugars are readily available and are the components that form disaccharides and longer polymers of sugars. These different classes of carbohydrates are metabolized differently (Asp,1995) though digested and absorbed in the duodenum (Vosloo, 2005; Cummings & Englyst ,1995). Fructose, a disaccharide, has a lower blood glucose response (GI=23) (Brand-Miller et al., 1996) than glucose, a monosaccharide, (GI=100) (Brand-Miller et al., 1996). Sucrose, a disaccharide, has a GI of 69 (Brand-Miller et al., 1996). The GI of raw and cooked tubers is significantly affected by the levels of these mono- and disaccharides.

Resistant Starches

Starchy foods generally yield lower glycemic values (Truswell, 1992). Starch hydrolysis varies from quite rapid to very slow. It is becoming appreciated that small intestinal starch digestion may be so retarded that starch can escape into the large bowel. These starches pass through the small intestine without being digested (Cumming & Englyst, 1995). This fraction is termed *resistant starch*. Factors under current investigation look at physical and chemical attributes of either the starch or the whole food (Annison & Topping, 1994). This is a new direction to research on complex carbohydrates.

The main theory behind resistant starch formation is cooling. During cooking, gelatinization occurs with heat and excess water. Disruption of the crystalline structure takes place at temperature of 60°- 70° (Colonna et al., 1985). Above 90 °C, fragments of amylopectin and amylose are suspended in water due to the significant loss of granular structure (Jing-ming et al., 1990). Granules high in amylose swell more slowly than those rich in amylopectin due to amylopectin increased branching.

Cooked food is almost always stored for variable lengths of time under moderate or low temperatures before consumption. Amylose and amylopectin molecules can associate to form a gel (Miles et al., 1985). The exact nature of the gel depends on amylose:amylopectin ratio, amount of water, and time and temperature of storage (Annison & Topping, 1994). The gel network is created by glucan chains that retrograde (recrystallise) in a helical structure. The helical structures further associate forming an extended network of polysaccharides detected by X-ray diffraction displaying a ß-type pattern (Colonna et al., 1992). It may take hours or days to form this gel. Retrogradation may also be increased by replicated heated and cooling (Sievert et al., 1991). Annison and Topping (1994) found that ß-type pattern polysaccharides are composed of almost pure linear ß-glucan chains of degree of polymerization.

The change in structure of starches during heating and cooling has a significant influence on starch digestibility in the gastrointestinal tract. Some evidence from intubated humans indicates that free glucose may pass to the colon from traces found in the terminal ileum (Stephen et al., 1983). This too is termed resistant starch since it escapes digestion. Englyst et al (1992) classified resistant starches according to the cause which allows them to pass to the large bowel: chemically resistant starch (i.e. enzyme resistant starch) and

physiologically resistant starch (i.e. starch that passes undegraded through the small intestine and into the large bowel). Raw starches are highly resistant to enzymatic hydrolysis compared with gelatinized starches (Annison & Topping, 1994).

Physical Barriers

Starch granule size can affect digestion by amylase as a result of surface area. Smaller particles are digested more rapidly than larger ones. Cell wall components and other plant material surrounding starch granules can inhibit enzyme access to the granule. Processing by heating can even encapsulate the starch granules. Proteases will have to digest the protein before starch digestion can occur. Tovar et al. (1990) revealed that amylolysis was enhanced by wet homogenization and pepsin treatment indicating that disruption of cell walls is a prerequisite for efficient digestion. Some processing conditions can cause long ß-glucan chains to form inclusion complexes with fatty acids (Holm et al., 1983). These complexes can affect the GI of starches.

Starch Structure

Higher levels of amylose in starch granules do not appear to affect digestibility. Fujita et al (1989) found that the starch digestibility from some plant species is much less than of others. Rice and tapioca starches (Cone & Wolters, 1990) are digested particularly well in vitro, but have low levels of amylose. Potato starch granules are predominately ß-type crystalline structure are less susceptible to ß-amylase hydrolysis. The available carbohydrate portion of foods should not include resistant starches, but in practice this can be difficult because resistant starches are difficult to measure (Foster- Powell et al., 2002).

Brand-Miller et al. (1996) has shown that the ratio of amylose to amylopectin molecules in food has an effect on the glycemic response. They are semicrystline aggregates

called starch granules. Their size, shape, and structure vary among botanical sources. Amylose has a straight chain of glucose units linked together by alpha-1,4-glycosidic bonds which are densely arranged, while amylopectin is much larger and contains branched chains of glucose units linked together by alpha-1,6-glycosidic bonds. Research has shown that the more open structure and lower density of amylopectin allows for quicker digestion (Brand-Miller et al., 1996).

Implications of Resistant Starches

Incomplete digestion of carbohydrates in the small intestine can result in symptoms similar to those of lactose intolerance; diarrhea, abdominal pain, and cramps. An accumulation of sugars in the large bowel beyond fermentation capabilities of the microflora lead to osmotic diarrhea (Holtug et al., 1992). Reduced absorption of carbohydrates in the small intestinal tract can pose health problems. Foods high in resistant starches limit the amount of glucose absorbed in the small intestine, thus lowering the glycemic index in theory, but data are contradictory. Brand- Miller et al (1992) carried out studies on white and brown rice from a cultivar (*Doongara*) which showed a lower glycemic index than comparable products from other cultivars. Truswell (1992) listed factors that could contribute to these inconsistencies, including quantity of food that was fed and day-to-day variations within the same individual.

Processing

Canning of starchy foods, such as potatoes (Soh & Brand-Miller, 1999), may cause retrogradation due to cooling. Competition for moisture can also lead to resistant starches. High dietary fiber levels retain moisture when cooking thereby limiting water for gelatinization (McWilliam, 2001). Sucrose also retains water during cooking and reduces the

amount of gelatinization that can occur. Resistant starches are more prevalent when crystalline granules are not gelatinized.

Fiber Type & Content

Dietary fiber is derived from various tissues of fruits, vegetables, cereals, and legumes. General conclusions about the physiochemical characteristics are not easily derived. Research done by Nishimune et al. (1991) and Trout et al. (1993) showed that an increase in total dietary fiber can retard the glycemic response. Studies by Cummings (1995) suggests that consumption of dietary fiber, particularly viscous fiber, alters the digestion and absorption of carbohydrates whereas lipids delaying gastric emptying to the small intestine by and/or interaction with digestive enzymes. There exists debate on the effect of blood glucose lowering between soluble fiber and uronic acids in insoluble fiber. Nishimune et al. (1991) further explained the effect of total dietary fiber on polysaccharide absorption through five mechanisms. Fiber delays the digestion of starch in the stomach; secondly, fiber will delay the transition time of stomach contents to the duodenum; thirdly, fiber will delay the diffusion of different saccharides in the duodenum, and fourthly, fiber will delay the hydrolysis of polysaccharaides in the upper parts of the duodenum. Finally, fiber will lower the rate of absorption of monosaccharides through the microvilli of the epithelial cells in the jejunum and upper ileum (Nisimune et al., 1991). It is explained that dietary fiber, with increasing concentration, acts as a competitor of sugar uptake through a membrane because both sugars released by cooking and fiber compete for the same binding site. This effect illustrates how total dietary fiber in foods lowers the post-prandial blood glucose response.

Cooking

The starch in raw food is stored in hard, compact granules which is difficult to digest (Brand- Miller et al., 1996). The cooking process causes intermolecular bonds of starch molecules in the presence of water and temperature and allowing the hydrogen bonding sites (the hydroxyl hydrogen and oxygen) to engage more water releasing individual molecules (Vosloo, 2005). Gelatinization occurs when starch molecules enter the aqueous solution followed by total disruption of the granules in a sequential process (Bennion & Scheudle, 2000). Starch is gelatinized at 60-90 °C region and becomes susceptible to hydrolysis by α and ß amylase. Incomplete cooking processes would result in starch being resistant to digestion (Asp & Bjork, 1992) leading to slow digestion and lower glucose response.

Sweet Potato

Sweet potatoes (*Ipomoea batatas* L.) are perennial dicots in the morning glory family (Convolvulaceae) which are cropped as annuals. Sweet potatoes should not be confused with yams which are monocots in the family Dioscoreaceae. Yams are grown as a staple in many tropical countries, but are seldom grown in the continental United States. Sweet potatoes probably originated in Central or South America but are now grown in many tropical, subtropical, and temperate regions. Sweet potato plants produce primary fibrous roots, pencil roots and storage roots. Storage roots are the only part eaten in the United States, but in parts of Asia the leaves are cooked like spinach and eaten as a green vegetable. Storage roots are attached to the stem by a stalk of thinner root which is usually initiated at the stem node just below the soil line. Skin color of storage roots ranges from white to brown to red-orange. Flesh color of storage roots can be red-orange, orange, yellow, purple, or white. The flesh can be either “moist” or “mealy” after cooking. In the southern states, commercial

types are soft-fleshed, developing a moist, sugary consistency during cooking as starches are converted into maltose and dextrins (Census of Agriculture, 1992). The *Beauregard* cultivar has the following advantages: pox resistance; high quality; attractive appearance; reduced need for N application, and is harvest ready in 90 rather than 120 day (Peet, 2001).

The sweet potato (*Ipomoea batatas* L.), often called a yam in the United States, is a crop plant whose large, starchy, sweet tasting tuberous roots are has a low glycemic index of 48 ± 6 according to Foster-Powell et al. (2002). It is the 7th most important food crop worldwide (Chalfant et al., 1990). There is a high yield (kg/ha/day), nutritional value (e.g. vitamins, nutriceuticals, glycemic index, and dietary fiber), and resistance to production stress (high temperature, water deficit, and insect and disease pressure). However the consumption per capita has declined progressively over the past 42 years (Kays, 2004).

Sweet Potato for Glycemic Control

Foods of the same botanical classification can elicit very different glycemic responses. For example, sweet potatoes and potatoes are both tuberous, but potato has a high GI value (<70) (Soh & Brand-Miller, 1999), while sweet potatoes have a low GI value (<55) (Brand-Miller & Foster-Powell, 2000; Vosloo, 2005). Furthermore, the crystalline structure of a given starch affects its digestion rate, ultimately influencing its GI value. Cumming & Englyst (1995) explained crystallinity as the difference in size and diffraction pattern (A, B, and C) differs with starch type. These authors suggested that starch primarily consisting of Type B (banana, potato, and other tubers) and C (legumes) X-ray diffraction patterns, will be more resistant to the action of alpha-amylase, thus retarding starch digestion (Vosloo, 2005). This would be illustrated in flattened blood glucose response curves.

Although generally considered sweet by definition, there is a large range of perceived sweetness, depending on sugar components and starch conversion during cooking of sweet potatoes (Takahata et al., 1992). Collins (1985) estimated available sugar values between 30-35%. Raw sweet potato tissue has sucrose, glucose, and fructose. The principal change in sugar composition with cooking is the production of maltose from starch. Much of the starch is converted into dextrins and maltose by alpha amylase and beta amylase; however the degree of starch conversion can differ across cultivars (Walter et al., 1975).

Caiapo

In addition to drugs, which act on insulin secretion, hepatic glucose output and intestinal absorption, new compounds that improve insulin sensitivity are currently under investigation or already in clinical use (Ludvik et al., 2003). Ludvik et al. (2002) previously reported (2002) the beneficial effects of Caiapo, an extract of the white-skinned sweet potato (*Ipomoea batatas* L.), on fasting plasma glucose, as well as on total and low-density lipoprotein (LDL) cholesterol in type 2 diabetic patients. This sweet potato has been cultivated in the Kagawa Prefecture in Japan and the extract of the skin of the root is used for the treatment of type 2 diabetes in Japan. The isolated antidiabetic component of Caiapo is an acidic glycoprotein that is similar to the proteins found in Beauregard sweet potatoes (*Ipomea batatas* L.) (Ludvik et al., 2002). Following treatment with high dose Caiapo, 4 g/d, insulin sensitivity significantly ameliorated when assessed with oral glucose tolerance test and frequently sampled intravenous glucose tolerance test ($P > 0.05$). This pilot study demonstrated that the ingestion of high dose Caiapo reduced fasting blood glucose levels and LDL cholesterol in male Caucasian type 2 diabetic patients previously treated by diet alone. They reported no significant changes in any parameters related to insulin dynamics; c-

peptide, distribution, and clearance. Caiapo consistently improved metabolic control of type 2 diabetes patients by decreasing insulin resistance without affecting body weight. In general, improved glycemic control can be attributed to a number of different mechanisms, including reduced intestinal glucose absorption, increased insulin secretion, and improved insulin sensitivity. Their studies argue against the delay in absorption because reduced glucose levels were not visible in the first phase of the experiment but only in the second later tests. Furthermore, their results exclude the stimulatory effects of insulin due to consistent levels of secretion parameters in the blood throughout the test. The discussion surrounding the effects of Caiapo indicates improved effects on insulin sensitivity.

Effects of Processing on Sweet Potatoes

Glycemic Index (GI)

Soh and Brand-Miller (1999) concluded no significant difference in GI of potatoes between boiling, oven-baking, microwaving, or mashing. Conversely, Lunetta et al. (1995) found that baked potatoes produced a significantly lower incremental glycemic response compared with boiled potatoes. Wolever et al. (1994) also found no differences in baking, boiling, and canned potatoes, but found that mashing significantly increased the glycemic response (by 15-20%). The variability noted in potatoes, although of a different species than sweet potatoes, leads to questions surrounding the effect of cooking methods on glycemic index with sweet potatoes.

According to Bahado-Singh et al. (2006) baked sweet potato elicited a high GI of 94 when studied with 14 West Indian carbohydrate rich foods. This study found boiled sweet potatoes to have a low GI of 46 (SE 5). Likewise, the roasted tubers were all high (82 for sweet yam). Blood glucose response curves for a low, intermediate, and high GI foods were

similar to the relationship shown in the response curves of boiled, roasted, and fried sweet potato. These results suggest the different processing method used (boiling, roasting, baking, frying) may influence the GI of sweet potatoes.

Studies investigated by Brand et al. (1985) revealed that extrusion cooking, explosion puffing, and instantization appear to make the starch of rice, potato, and corn products more readily digested. Methods that are known increase the digestibility are hydration of granules (gelatinization) and disruption of organized granule structure (Booher et al., 1951). These conditions increase the availability of starch to amylase digestion and are more likely to occur in factory processing cooking conditions of these starches when higher temperatures and pressures are utilized. The more processed a food is, the higher the glycemic response it will produce (Thorne et al., 1983). Conventionally cooked starches may be recommended for diabetics to achieve glycemic indices lower than those processed in factories.

Starch

The boiling processing uses wet heat that causes leaching of glucose monomers from amylase-amylopectin degredation. Starch content of sweet potatoes is approximately 63% of available carbohydrate (USDA, 2006). The loss of the readily digestible sugars due to leaching had no direct implication on the amount of carbohydrate used to calculate the GI of boiled or steamed foods (Bahado-Singh, 2006). Using wet heat to cook a potato has been shown to contain greater amounts of resistant starches. Under baking, the foods are processed using dry heat, causing loss of water and concentrating of free sugars. The degradation of starches further increases the sugar content increasing the glycemic index to high values.

Cooking methods further affect conversion of starch to digestible form. Researchers have shown that cutting sweet potatoes into strips and cooking rapidly retain significant amounts of starch, whereas the cooking of whole roots allows more complete conversion of starch into sugars and dextrins (Collins & Walter, 1985). Conversely it has been shown that sugar concentrations are similar in roots cooked in microwaved and convection ovens (Picha, 1985).

The recrystallization of dispersed starches is formed as a result of intermolecular hydrogen bonds resulting in stronger hydrogen bonds (Englyst, 1982). The cook-cool-re-warming of the potatoes affects about 7% of the starch and allows it to escape digestion in the small intestine compared to about 3% in freshly cooked potato (Englyst & Cummings, 1987). Earlier studies on potatoes in vitro showed cooling, freezing, or drying produces partially resistant starches to α-amylase as well. Digestibility of starch made resistant to α-amylase by cooling has been shown to improve on reheating. The increased resistance to amylase on cooling appeared to relate to changes in crystalline structure of starch rather than overall physical form (Englyst & Cummings, 1987). The resistant starches do significantly affect the glycemic index.

Maturity

The maturity of a tuber affects digestion of starches. As potatoes mature, the quantity of amylose increases but the difference is small and not likely to affect the glycemic response. (Soh & Brand-Miller, 1999) On the other hand, the degree of amylopectin branching increases with maturity. Amylopectin has an irregular branching structure and it is more readily gelatinized than the linear amylose molecule leading to higher glycemic responses. However, higher levels of amylose (less branching) do not affect digestibility.

Total Dietary Fiber

GI was negatively correlated with total dietary fiber (TDF) and with certain fiber components, i.e. soluble and insoluble fiber (Trout et al, 1993). The addition of fiber, especially viscous soluble fiber, to a glucose solution or to a semisolid food, reduces the glycemic response of normal or diabetic subjects (Jenkins et al, 2002). Fiber can stabilize the gastric chyme and prevent separation of the solid from the liquid phase. Fiber has little, if any, direct acute effect on the secretory function of the exocrine pancreas suggesting that the primary effect of fiber on carbohydrate digestion is exerted in the intestinal lumen.

Insoluble dietary fiber is not soluble in water and is relatively unchanged as it passes through the digestive tract. The decrease in total dietary fiber is primarily due to the reduction in insoluble dietary fiber. However, the effects of concentrating the food due to water loss accounts for the higher percentage of total dietary fiber. This higher percentage of TDF impedes the digestion of sugars and their absorption.

Gelatinization

The softening and sweetening of sweet potato during the cooking process is brought about by cell separation (Binner et al., 2000). Sweet potatoes are unique in containing a heat-activated α-amylase that breaks down starch under suitable conditions of gelatinization. The mechanism to which amylose leaches out of the sweet potato is through micropores formed in the starch granules during the expansion phase of gelatinization. The low-molecular weight dextrins and malto-dextrins from amylose and amylopectin breakdown are able to escape the cell wall, resulting in the sweet and non-mealy texture which is desired (Freeman et al., 1992). Modified cell walls that are amenable to dextrin release were observed by cooking at 100 °C than at 70 °C, suggesting that sweet potato gelatinization occurs near the

upper spectrum of the gelatinization region. After cooking, spongy-like structures are observed in sweet potato parenchyma cells due to gelatinization (Valetudie et al., 1995).

The temperature at which gelatinization occurs is influenced by the sugars found in sweet potatoes. Sucrose is the main component of the soluble sugars, while glucose and fructose are minor components (Taira & Yaui, 1987; Truong et al., 1986). It is well accepted that sugar shifts the gelatinization to higher temperatures. Research by Nishmuni et al. (1991) observed gelatinization occurring at increased temperatures with increasing sucrose concentrations while testing fiber content in potatoes.

A starch with high sucrose levels is more likely to reduce the length of cooking time within its gelatinization temperature range. This will promote more incomplete breakdown of the starch resulting in a lower glycemic value. On the other hand, it will delay retrogradation, thereby delaying the formation of resistant starch, and increasing available sugars for digestion and increasing the glucose response. There are varying outcomes that depend on the length of cooking, temperature, and carbohydrate composition.

Protein

Reports on the amino acid content of "Jewel" sweet potato grown in Clayton, North Carolina by Purcell and Walter (1982) suggest that their protein has a high chemical score. The total protein content on a dry matter basis is about 7.52% raw and 5.55 % baked. Lysine is the primary amino acid studied in sweet potatoes. Other amino acids such as tyrosine, leucine, isoleucine, and phenylalanine are slightly lower in cooked vs. raw sweet potatoes. Destruction of lysine was considered to be the major change of amino acids caused by processing of sweet potatoes (Purcell & Walter, 1982). Purcell and Walter's results indicate <26% less of this essential amino acid in canned and flaked puree of sweet potatoes. They

hypothesized that proteins leached into the canning syrup resulting in lower nitrogen value. Further analysis was recommended to determine if resulting protein levels after cooking is suitable for supplement recommendation.

Beta-Carotene

Beta–carotene is a naturally occurring pigment in sweet potatoes providing the yellow-orange color. It is the major precursor of vitamin A (Francis, 1969; Simpson 1980; Klaui & Bauernfeind 1982). Beta-carotene is subject to degradative changes during food processing and cooking due to oxidation (Gregory, 1985; Simpson, 1985). The conversion of natural trans-ß-carotene to cis-ß-carotene lowers the pro-vitamin A activity (Sweeney & March, 1971). Presumably tissue morphological changes of gelatinization allow penetration of organic solvents and release of carotenes (Chandler & Schwartz, 1988).

Summary

It is through our understanding of the health benefits of low glycemic index foods as treatment for diabetic patients and improved glucose control that our study chose to investigate the glycemic index found in Beauregard sweet potatoes. More comprehensive data are needed to quantify the true GI of North Carolina Beauregard sweet potatoes with the intention of making recommendations for their health improvement qualities. Furthermore, our study considered the fact that sweet potatoes are commonly consumed after cooking in the United States and therefore examined the changes processing induces on the glycemic index of sweet potatoes. The objectives of the research described in the following chapters are to add to the literature on these points.

Literature Cited

Al Yassin D, Ibrahim K (1981): A minor haemoglobin fraction and the level of fasting blood glucose. J Fac Med University Baghdad 23: 373-380.

Albie J, Konnarides CH, Pi-Sunyer X. (1995) Weight control. Meabolic and cardiovascular effects. Diabetes Review. 3:335-44.

American Diabetes Association: (2002) Evidence based nutrition principles and recommendation for the treatment and prevention of diabetes and related complications. Diabetes Care. 24:202-212.

American Diabetes Association (2006) Nutrition Recommendations and Interventions for Diabetes – 2006. Diabetes Care. 29(9)2140-2157.

American Diabetes Association (2007) All About Diabetes. < http://www.diabetes.org/about-diabetes.jsp> accessed June 5, 2007.

Annison G, Topping DL. 1994. Nutritional role of resistant starch: chemical structure vs. physiological function. Ann Rev Nutr. 14:297-320.

Ash, SL. Human Nutrition: An Inquiry Guided Approach. 8th edition. 2005; 87

Asp N-GL (1995) Classification and methodology of food carbohydrates as related to nutritional effects. Am J Clin Nutri 61(suppl) 930S-937S.

Attele AS, Wu JA, Yuan CS. (1999) Ginseng pharmacology: multiple constituents and multiple action. Biochem Pharmacol 58:1685-193.

Bahado-Singh (2006) Food processing methods influence the glycaemic indices of some commonly eaten West Indian carbohydrate-rich foods. Br J Nutr. 9(3) 476-81.

Baldwa VS, Bhandari CM, Pangaria A, Goyal RK, (1977) Clinical trial in patients with diabetes mellitus of an insulin-like compound obtained from plant sources. Upsala J Med Sci. 82:39-41.

Bennion M, Scheule B (2000) Introductory foods. 11th Edition. New York. Prentice-Hall

Berman BM, Kaczmarczk J, Swyers JP: (1999) Complementary and alternative medicine herbal therapies for diabetes. J Assoc Acad Minor Phys 10:10-14.

Binner, S; Jardine, WG; Renard, CMCG; Jarvis, MC (2000) Cell wall modifications during cooking of potatoes and sweet potatoes. J Sci Food Agric. 80:216-218.

Blonde L, Ginsberg BH, Horn S. (2002) Frequency of Blood glucose monitoring in relation to glycemic control in patients with type II diabetes. Diabetes Care. 25(1)245.

Bnouham M, Ziyyat A, Mekhfi H, Tahri A, Legssyer A (2006) Medicinal plants with potential antidiabetic activity- A revew of ten years of herbal medicine research (1990-2000) Internaional J Diabetes Metabol 14:1-25.

Bogardus C, Lillioja S, Mott DM, Hollenbeck C, Reaven G (1985) Relationship between degree of obesity and in vivo action in man Amer J Physiol, Endocrinol Metabol 248(3): 286-E291.

Bonadonna RC, Bonora E. (1997) Glucose and free fatty acid metabolism in human obesity. Diabetes Review. 5:21-51.

Brand JC, Nicholson, PL, Thornburn AW, Truswell ASl. (1985) Food processing and the glycemic index. Amer J Clin Nutr. 42: 1192-1196.

Brand-Miller J, Pang E, Bramall L. (1992) Rice: a high or low glycemic index food? Proc Nutr So. Austr 17:226.

Brand-Miller J, Foster-Powell K, Cola-Giuri S. (1996) The GI factor: The glycemic index solutions. Tydelmere. Hodder and Stoughton:34

Brand-Miller J, Foster-Powell K (2000) The glucose revolution. GI plus. Sydney. Hodder Headline: 58

Brand-Miller J, Petocz, P, Hayne S, Colaguri S (2003) Low-glycemic index diets in the management of diabetes. Diabetes Care. 26(8): 2261-2267.

Chalfant R, Jansson R, Seal D, Schalk J (1990) Ecology and Management of Sweet Potato Insects. Annual review of entomology 35: 1:157.

Campbell PJ, Carlson MG (1993) Impact of obesity on insulin action in NIDDM. Diabetes. 42(3)405-410.

Chan JM, Rim EB, Colditz GA et al. (1994) Obesity, fat distribution and weight gain as risk factors for clinical diabetes in men. Diabetes Care. 17:961-9.

Chang MC, Morris WC (1990) Effect of heat treatments on chemical analysis of dietary fiber. J Food Sci. 55(6)1647-1675.

Chandler LA, Schwartz SJ (1988) Isomerization and Losses of *trans-B-Carotene* in Sweet Potatoes as Affected by Processing Treatments. J Agric Food Chem 36:129-133.

Collins WW, Walter Jr WM (1985) Fresh roots for human consumption. Sweet potato products: a natural resource for the tropics. Bouwkamp JC ed. Florida CRC Press 205-218.

Colonna P, Leloup V, Buleon A. (1992) Limiting factors of starch hydrolysis. Eur J Clin Nutr. 46(suppl.2): S17-S32.

Cone JW, Wolters MGE. (1990) Some properties and degradability of isolated starch granules. Starch/Starke 42:298-31.

Covington MB. (2001) Traditional Chinese medicine in the treatment of diabetes. Diabetes Spectrum 12(3) 155-159.

Cummings JH, Englyst HN (1995) Gastrointestinal effects of food carbohydrate. Amer J Clin Nutr 61(4) (suppl): 938S- 945S.

De Valk HW (1996) Magnesium in diabetes mellitus. Neth J Med 54: 139-146.

Dey L, Attele AS, Yuan S. (2002) Alternative therapies for Type II Diabetes. Alternative Medicine Review 7(1)45-58.

Englyst HN, Wiggins HS, Cummings JH (1982) Determination of the non-starch polysaccharides in plant foods by gas-liquid chromatography of constituent sugars as alditol acetates. Analyst. 107:307-318.

Englyst HN, Cummings JH (1987) Digestion of polysaccharides of potato in the small intestine of man. Am J Clin Nutr 45:423-431.

Englyst HN, Kingman SM, Cummings JH. (1992) Classification and measurement of nutritionally important starch fractions. Eur J Clin Nutr. 46(Suppl2):S33-S50.

Foster-Powell K, Holt SHA, Brand-Miller JC (2002) International table of glycemic index and glycemic load values: Am J Clin Nutr. 76:5-56.

Francis FJ (1969) Pigment content and color in fruits and vegetables. Food Technol 23:32.

Freeman M, Jarvis MC, Duncan HJ (1992) The textural analysis of cooked potato. 3 Simple methods to determining texture. Potato Res. 35:103-109.

Fujita S, Glover DV, Okuno D, Fuwa H. (1989) In vitro and in vivo digestion of high amylose type starch granules. Starch/Starke 41:221-24.

Fung TT, Manson JF, Solomon CG, Liu S, Willett WC, Hu FB (2003) The association between magnesium intake and fasting insulin concentration in healthy middle-aged women. J Am Coll Nutr. 22(6) 533- 38.

Gilbertson HR, Brand-Miller JC, Thornburn 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 II diabetes. Diabetes Care. 24: 1137-1143.

Gillis DN. (1997) Panax ginseng pharmacology: a nitric oxide link? Biochem Pharmacology; 54:1-8.

Gostout CJ, Lindor KD, Sorbi D (2000) Insulin resistance influences iron metabolism and hepatic steatosis in type II diabetes. Gastroenterology 118;5:986-988.

Greene D, Arezzo J, Brown M, and the Zenarestat Study Group. Dose-related effects of the aldose reductase inhibitor Zenarestat on nerve sorbitol levels, nerve conduction velocity and nerve fiber density in human diabetic neuropathy. Diabetes. 1996;45:A9. Abstract.

Gregory GF III. (1985) "Chemical Changes of Vitamins during Food Processing". In Chemical Changes in Food Processing. Richardson T, Finley JW, Eds AVI. Westport CT, 1985 p 393.

Groop L.C. (1999) Insulin resistance: the fundamental trigger of type 2 diabetes. Diabetes, Obesity Metab. 1:S1-S7.

Hamza MA, White PF, Criag WF, Ghonome EA, Ahmed HE, Proctor TJ, Noe CE, Vakharia AS, Gajraj N (2000) Percutaneous electrical nerve stimulation: a novel analgesic therapy for diabetic neuropathic pain. Diabetes Care 23:365-370.

Hasegawa H, Matsumiya S, Murakami C, Kurokawa T, Kasai R, Ishibashi S, Yamasaki K. (1994) Interaction of ginseng extract, ginseng separated fractions, and some tritepenois saponins with glucose transporters in sheep erythrocytes. Planta Med 60(2)153-157.

Holm J, Bjorck I, Ostrowska S, Eliasson AC, Asp NG, et al. (1983). Digestibility of amylose lipid complexes in vitro and in vivo. Starch/Starke 35:294-97.

Holtug K, Clausen MR, Hove J, Christiansen J, Mortensen PB. (1992). The colon in carbohydrate malabsorption: short-chain fatty acids, pH, and osmotic diarrhea. Scan J Gastroenterol. 27:545-52.

Hossain MZ, Shibib BA, Rahman R (1992): Hypoglycemic effects of *Coccinia indica* : Inhibition of key gluconeogenic enzyme, glucose-6-phosphate. India J Exper Biol 30:418-420.

Huang KC. (1999) The Pharmacology of Chinese Herbs. Boca Raton, FL; CRC Press;.

Jing-ming L, Sen-lin Z. (1990). Scanning electron microscopy study on gelatinization of starch granules in excess water. Starch/Starke 42:96-98.

Jenkins D, Cyril JA, Kendall WC, Augustin LSA, (2002) Glycemic index: overview of implications in health and disease. Am J Clin Nutr, 76(1), S266-S273.

Kays SJ (2004) Sweet Potato Production Worldwide: Assessment, Trends, and the Future. I International Symposium on Root and Tuber Crops: Food Down Under. (abstract).

Kimura M, Waki I, Chujo T, Kikuchi T, Hiyama C, Yamazaki K, Tanaka O (1981) Effects of hypoglycemic components in ginseng radix on blood insulin level in alloxan diabetic mice and on insulin release from perfused rat pancreas. J Pharmacobiodyn;4:410-417.

Kirchner Jeffrey T. (2000) Nephropathy in patients with type II diabetes mellitus. American Family Physician. http://findarticles.com/p/articles/mi_m3225/is_5_61/ai_61432769/pg_1>

Kissebah, AH, Vydelingum, N, and Murray, R (1982) Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metabol. 54:254-260.

Klaui M, Bauernfeind JC (1981) Carotenoids as Food Colors. In Carotenoids as Colorants and Vitamin A Precursors. Baurnfein JC, Ed; Academic. New York p 50.

Klein R, Klein BED, Moss SE. (1996) Relation of glycemic control to diabetic microvascular complication in diabetes mellitus. Amer Intern Med.124 (1 pt 2) 90-6.

Konno C, Sugiyama K, Kano M (1984) Isolation and hypoglycaemic activity of panaxans A,B,C,D,E, glycans of Panax ginseng roots. Plata Med 50:434-436.

Konno C, Murakami M, Oshima Y, Hikino H. (1985) Isolation and hypoglycemic activity of panaxans W,R,S,T, and U, glycans of Panax ginseng roots. J Ethnopharmacol 14:69-74.

Lebovitz HE. Sulfonylurea drugs. In: Lebovitz He, ed. (1995) Therapy for Diabetes and Related Disorders. 2nd ed. Alexandria, Va: American Diabetes Association;116-23.

Lee, FC. Facts About Ginseng: (1992) The Elixir of Life. Hollyn International Corporation, Elizabeth, NJ,.

Lee NA, Reasner CA. (1994) Beneficial effect of chromium supplementation on serum triglycerides levels in NIDDM. Diabetes Care 17:1449-1452.

Little, D' (2003) Non-pharmacological management of diabetes: the role of diet and exercise. Geriatrics and Aging. 6;1:27-29.

Lopez-Ridaura R, Willett WC, Rimm EB, Liu S. (2004) Magnesium intake and risk of type II diabetes in men and women. Diabetes Care 27(1:134-140.

Ludvik, Bernhard, Mahdjoobian Katja, Waldhaeusi Werner, Hofer Astrid, Prager Rudolf, Kautzky-Willer Alexandra,Pacini Giovanni (2002) The effect of *Impomea batatas* (Caiapo) on Glucose Metabolism and Serum Cholesterol in patients with type II diabetes. Diabetes Care. 25(1):239-240.

Ludvik B, Mahdjoobian K, Waldhaeusi W, Hofer A, Prager R, Kautzky-Willer A, Pacini G (2003) Mode of Action of *Impomea Batatas* (Caiapo) in type II diabetic patients. Metabolism. 52;7:875-880.

Lunetta
McWilliams M. (2001) Foods. Experimental perspectives. 4th Edition. Upper Saddle River, New Jersey. Prentice-Hall.

Miles MJ, Morris VJ, Orford PD, Ring SG (1985). The roles of amylose and amylopectin in the gelation and retrogradation of starch. Carbohydr Res. 135:271-81.

Miller LG. (1998) Herbal medications, nutraceuticals, and diabetes. In; Miller LG, Murray WJ, eds. Herbal Medicinals, A Clinician's Guide. Binghamton, NY: Pharmaceutical Products Press, Imprint of the Haworth Press, Inc. 155-133.

Mocchegiani E, Boemi M, Fumelli P, Fabris N (1989) Zinc-dependent low thymic hormone level in type I diabetes. Diabetes. 38:932-37.

Mooradian AD, Failla M, Hoogwerf B (1994) Selected vitamins and minerals in diabetes. Diabetes Care; 17:464-479.

Morris R, Rimm D, Hartz A, Kalkhoff R, Rimm A (1989) Obesity and Heredity in the etiology of non-insulin-dependent diabetes mellitus. Am J Epidemiol 130: 112-121.

National Eye Institute, 2006 Diabetic Retinopathy. US National Institute of Health

Nissan SE, Kwolski (2007) Effect of Nosigliazone on risk of myocardial infarction and death from cardiovascular causes. New Engl J Med 356:2457- 2471.

Nishimune T, Yakushiji T, Sumimoto T, Taguchi S, Konishi Y, Nakahara S, Ichikawa T, Kunita N (1991) Glycemic response and fiber content of some foods. Am J Clin Nutr 54:414-9.

Offenbacher EG, Pi-Sunyer FX. (1980) Beneficial effect of chromium-rich yeast on glucose tolerance and blood lipids in elderly subjects. Diabetes; 29:919-925.

Ohnishi Y, Takagi S, Miura T, Usami M, Kako M, Ishihara E, Yano H, Tanigawa K, Seino Y. (1996) Effect of ginseng radix on GLUT 2 protein content in mouse liver in normal and epinephrine-induced hyperglycemic mic. Biol Pharm Bull19:1238-1240.

Paolisso G, Scheen A, D'Onofrio F, Lefebvre P (1990) Magnesium and glucose homeostasis. Diabetolgia 33:511-514

Paolisso G, Sgamato S, Gambardella A, Pizza G, Tesauro P, Varricchio M, D'Onofrio F (1992) Daily magnesium supplements improve glucose handling in elderly subjects. Am J Clin Nutr 55:1161-1167.

Peet M. (2001) Sweet Potato. Sustainable Practices for Vegetable Production in the South < http://www.ncsu.edu/sustainable/profiles/c18swpot.html>

Picha DH (1985) HPLC Determinationof sugars in raw and baked sweet potatoes. J Food Sci. 50: 1189-1190, 1210.

Prisant LM (2004) Preventing type II Diabetes Mellitus. J Clin Pharmacol 44: 406-413.

Purcell AE, Walter WM (1982) Stability of Amino Acids during Cooking and Processing of Sweet Potatoes. J Agric Food Chem 30: 443-444.

Ribes G., Sauvaire Y, Baccou JC, Valette G, Chenon D, Trimble ER Loubatieres MM (1984) Effects of fenugreek seeds on endocrine pancreatic secretion in dogs. Ann Nutr Metab 28:37-43.

Rios MS (1998) Relationship between obesity and increased risk of major complication in non-insulin-dependent diabetes mellitus. Euro J Clin Invest 28 (Suppl. 2): 14-18.

Ritz E, Orth Sr. (1999) Nephropathy in patients with type II diabetes mellitus. N Engl J Med. 341:1127-33.

Roy D, Perreault M, Marette A. (1998) Insulin stimulation of glucose uptake in skeletal muscle and adipose tissue *in vivo* in NO dependent. Amer J Phsiol.274: E692-E69

Sakar S, Pranava M, Marita R. (1996) Demonstration of the hypoglycemic action of *Momordica charantia* in a validated animal model of diabetes. Pharmacol Res 33:1-4.

Schulz V, Hansel R, Tyler VE. (1998) Rational phytotherapy. In: Agents that increase resistance to disease. New York, NY: Springer-Verlag 269-272.

Scriver CR, Kaufman S, Eisensmith RC, Woo SLC (1995) The hyperphenylalaninemias. In: Scriver CR, Beaudet AL, Sly WS, Valle D (eds) The metabolic and molecular basis of inherited diseases, 7th edn, vol 1. McGraw-Hill, New York, pp 1015–1075.

Sheela GC, Augusti KT (1992): Antidiabetic effects of S-allyl cysteine sulphoxide isolated from garlic *Allium sativum* Linn. India J Experim Biol 30: 523-526.

Sievert D, Czuchajowaska A, Pomeranz Y. (1991) Enzyme resistant starch. III. X-ray diffraction of autoclaved amylomaize. Cereal Chem. 68:86-91.

Sima AA, Bril V, Nathaniel V, McEwen TA, Brown MB, Lattimer SA, Greene DA (1988) Regeneration and repair of myelinated fibers in sural nerve biopsies from patients with diabetic neuropathy treated with an ARI. N Engl J Med. 319:548-55.

Simpson KL, Chichester CO (1980) Metabolism and Nutritional Significance of Carotenoids. Ann Rev Nutr. 1:351.

Simpson KL (1985) Chemical changes in natural food pigments. *Chemical Changes in food during processing;* Richardon T, Finley JW. AVI Westport CT. 411

Soh N. & Brand-Miller J. (1999). The glycaemic index of potatoes: The effect of variety, cooking method and maturity. European J Clin Nutr*, 53*, 249-254.

Shohet IH (1995) Method of Controlling Diabetes Mellitus. United States Patent. #5,420,108.

Sotaniemi EA, Haapakoski E, Tautio A. (1995) Ginseng therapy in non-insulin-dependent diabetic patients. Diabetes Care 18:1373-1375.

Stephen AM, Haddad AC, Philips SF. (1983) Passage of carbohydrate into the colon. Direct measurement in humans. Gastroenterology 85:589-95.

Suzuki Y, Ito Y, Donno C, Furuya T. (1991) Effects of tissue cultured of ginseng on gastric secretion and pepsin activity. Yakugaku Zasshi; 111:770-774 (translated from Japanese).

Sweeney SJ, Marsh AC (1971) Effect of Processing on Provitamin A in Vegetables. J Am. Diet Assoc. 59:238.

Taira H, Yaui T. (1987) Evaluation of proximate comonents in new conversion products and raw materials. Annual Report of Biomass Conversion Program; Agriculture, Forestry, and Fisheries Research Council Secretaria: Tokyo 36-37 (translated from Japanese).

Takahata, Y; Noda T, Nagata T, (1992) Varietal diversity of free sugar composition in storage root of sweet potato . Jpn. J. Breed. 42:515-521

Tovar J, Bjorck IM, Asp N-G. (1990) Analytical and nutritional implications of limited enzymic availability of starch in cooked red kidney beans. J Agric Food Chem. 38:488-93.

Truong, V D; Biermann, Christpopher J, Marlett, Judith (1986) Simple sugars, Oligosaccharides, and starch concentrations in raw and cooked sweet potato. J Agric Food Chem. 34:421-425.

Truout Daid L, Behall Kay M, Osilesi Odutola (1993) Prediction of glycemic index for starchy foods. Am J Clin Nutr. 58:873-8.

Truswell AS. (1992) Glycemic index of foods. Eur. J. Clin. Nutr. 46(Suppl.2):S91-S101.
USDA National Nutrient Database for Standard Reference, Release 19 (2006) website

U.S. Dept. of Commerce, Bureau of the Census. 1992 Census of Agriculture. Table 29. Vegetables, Sweet corn and melons harvested for sale: 396-421

Valetudie, Jean-Claude; Guadeloupe La; Colonna, Paul; Bouchet, Brigitte; Gallant, Daniel J (1995) Gelatinization of Sweet potato, Tania, and Yam Tuber Starches. Starch. 47;8:298-306.

Vankateswaran S, Pari L (2002) Effect of *coccinia indica* on Blood Glucose, Insulin and Key Hepatic Enzymes in Experimental Diabetes. Pharamaceutical Biology. 40;3:165-70.

Vijan Sandeep MD, Steven Deryth L MD, Herman William H MD MPH, Funnel Martha M MS RN, Standiford MD (1997) Screening, Prevention, Counceling, and Treatment for the Complication of Type II Diabetes Mellitus. Journal General Internal Medicine. 12:567-579.

Vosloo Charlyn M (2005) Some factors affecting the digestion of glycaemic carbohydrates and the blood glucose response. Journal of Family Ecology and Consumer Sciences 33: 1-9.

Vuksan V, Sievenpiper JL, Xu Z, Wong EYY, Jenkins AL, Beljan-Zdravkovic U, Leiter LA, Josse RG, Stavro MP (2001) Konjac-Mannan and American ginseng: Emerging alternative therapies for type 2 diabetes mellitus. J Am Coll Nutr;20(5 Suppl):370S– 380S; discussion 381S–383S.

Walter RM, Uriu-Hare JY, Olin KL, Oster MH, Anawalt BD, Critchfirled JW, Keen CL (1991) Copper, Zinc, Maganese, and Magnesium Status and Complication of Diabetes Mellitus. Diabetes Care. 14(11)1050-1056.

Walter WM, Purcll AE, Nelson AM (1975) Effects of amylolytic enzymes on “moistness” and carbohydrate changes of baked sweet potato cultivars. J Food Sci 40: 793-796.

Welihinda J, Karunanayake EH, Sheriff MH, Jayasinghe KS. (1986) Effect of *Momordica charantia* on the glucose tolerance in maturity onset diabetes. Journal of Ethnopharmacology;17:277-282.

Wheatley AO, Asemota HN, Morrison EY (2002) Glycemic Index: not all carbohydrates are created equal. West Indian Medical Journal. 51 (suppl) 60.

Williams Pauline, Stubbs Wendy (2004) The glycemic index. Extension Utah State University website

Wilson JG, Lindquist JH, Grambow SC, Crook ED, Maher JF(2003) Potential Role of Increased Iron Stores in Diabetes. American Journal of the Medical Sciences. 325(6):332-339

Wolever TMS, Nuttal FQ, Lee R, et al. (1985) Prediction of the relative blood glucose response of mixed meals using the white bread glycemic index. Diabetes Care;8:418-28.

Wolever TMS, Katzman-Relle L, Jenkins AL, Vuksan V, Josee RG and Jankins DJA (1994): Glycaemic index of 102 complex carbohydrate foods in patients with diabetes. Nutr Res. 14: 651-669.

Xie Jing-Tian, Mehendale Sangeeta, Yuan Chun-Su (2005) Ginseng and Diabetes. Amer J Chinese Med 33;3:397-404.

Yokozawa T, Kobayashi T, Oura H, Kawashima Y. (1985) Studies on the mechanism of the hypoglycemic activity of ginsenoside- Rb2 in streptozoticin – diabetic rats. Chemical Pharmacy Bulletin33:869-872.

Young MJ, Breedy JL, Veves A, Boulton AJ (1994) The prediction of diabetic neuropatic foot ulceration using vibration perception thresholds. A prospective study. Diabetic Care 17:557-560.

Yuan CS, Wu JA, Lowell T, Gu M (1998) Cut and brain effects of American ginseng root on brainstem neuronal activities in rat. Amer J Chinese Med 26:47-55.

Zakir S, Sarwar M, Allen JC, Butt MS, Allen H. (2005) Effect of sweet potato on insulin efficiency of normal and diabetic subjects in Pakistan. Eur J Sci Res 10(1):87-97.

Chapter 2

Sweet Potato and Caiapo in Relation to Post-prandial Glucose Level

Abstract

Long term studies have found that consumption of a high glycemic load diet is a risk factor for type 2 diabetes mellitus. Low glycemic index foods maintain steady blood glucose levels and aid in the control of diabetes. The glycemic index of white potato is 85 (high). The glycemic index for sweet potatoes cultivars is approximately 48 ± 6. An active ingredient in the skin of a Japanese cultivar lowers blood glucose levels in diabetic subjects. This antidiabetic component, Caiapo, has been reported by Ludvik et al. in 2003 to increase insulin sensitivity, thereby increasing cellular glucose uptake.

This study aims to investigate the blood glucose lowering capabilities of North Carolina sweet potatoes. Our research objective was to quantify the GI produced from the Beauregard cultivar. Twelve volunteers consumed white potato, white potato with Caiapo, whole Beauregard sweet potato, Beauregard sweet potato skin, and Beauregard sweet potato flesh while monitoring blood glucose levels in thirty minute increments for two hours with glucometers. C-peptide and insulin levels were measured to assess the response to a rise in absorbed glucose. All samples were analyzed in duplicate.

Comparison between the levels of glucose, C-peptide, and insulin release all illustrated peaks at sixty minutes or earlier and values returning to fasting levels after two hours. The difference between these parameters due to potato sample was constant over time indicating equal trends regardless of potato or inclusion of Caiapo. Lower levels of serum insulin and C- peptide were observed when lower concentrations of glucose were in the blood, suggesting that sweet potato or Caiapo did not directly affect insulin release. The GI for raw Beauregard sweet potatoes is low, especially when consumed with the peel or

eaten alone. The addition of Caiapo to white potato lowered the average GI although the difference was not statistically significant.

Introduction

Attention to the quantity and source of dietary carbohydrate recommended for diabetic patients has been the cornerstone for therapy since the disease was first described. Low calorie and low carbohydrate meals were first recommended, followed by high carbohydrate meals intended to reduce total saturated fat levels for improved glucose tolerance (Brunzell et al., 1971; Brunzell et al., 1974). These recommendations were later amended to take into account the type of carbohydrate; simple verses complex. Simple carbohydrates are defined as either monosaccharides or relatively low-molecular-weight polymers of the monosaccharides, and are also referred to as sugars (Crapo, 1985). Complex carbohydrates are polysaccharides comprised of a large number of monosaccharide molecules.

The assumption was that complex carbohydrates were digested and absorbed more slowly than simple sugars, resulting in a flattened glucose response curve. The fallacy was revealed when researchers discovered that blood glucose and insulin responses varied greatly, independent of diet classification as simple or complex carbohydrate (Schauberger et al., 1977; Jenkins et al., 1980; Jenkins et al., 1981; Jenkins et al., 1983).

Jenkins and others (1981) proposed the glycemic index as a measurement of carbohydrate quality that considers the physiological response to food. Initial studies found that baked potato elicited a blood glucose response not significantly different from that of an equivalent amount of glucose (Crapo et al., 1977). Today, white bread or glucose is used as the standard against which other foods are compared for glycemic index calculations. Glycemic index foods are separated into low (<55), medium (56 to 69), and high (>70) categories (Canadian Diabetes Association, 2007).

Alternative methods for diabetes mellitus management using diet are becoming increasingly important and are gaining popularity. The glycemic index has become a prescribed method to help maintain low blood glucose levels for diabetic patients. However, the American Diabetes Association (ADA) (2002) asserts that there is not sufficient evidence of long-term benefit to recommend their use as a primary strategy. Conversely, the standard retrospective meta-analysis by Brand-Miller et al. (2003) found that there is evidence that low-GI diets improve glycemic control over and above that obtained by conventional or high-GI diets with incremental benefit that are clinically significant and similar to that offered by newer pharmacological agents.

Evidence suggests that sweet potatoes (*Ipomea batatas* L.) may be useful in controlling blood glucose of persons with diabetes (Ludvik, 2004; Zakir, 2005). Chemical analysis conducted by Zakir (2005) indicated that Beauregard (US) contains similar acidic glycoproteins to those found in the dietary supplement Caiapo that is marketed for anti-diabetic properties. These findings represent an opportunity for North Carolina sweet potato growers to market Beauregard sweet potatoes for potential anti-diabetic qualities as well.

Further understanding of the post-prandial glycemic response from Beauregard sweet potatoes is needed to determine the blood sugar lowering mechanisms of the inherent glycoprotein. Zakir (2005) studied the blood glucose lowering effects found in a Pakistan and US sweet potato cultivars in Pakistan. The focus of this study was to observe the glycemic response elicited by Beauregard sweet potato (skin, flesh, and whole) including the effect when Caiapo is added in its powdered form.

Materials & Methods

Reagents

Glucose oxidase/ peroxidase and o-dianisidine reagents were provided in the Glucose (GO) assay kit by SIGMA- Aldrich (GAGO-20) (St. Louis, MO). C-peptide (10-11136-01) and insulin (10-1113-01) ELISA assay kits were manufactured by Mercodia (Sweden, ALPCO Diagnostics, Windham, NH).

Instrumentation

A Cuisinart ® MP-14-N (East Windsor, NJ) 14-cup food processor was used to blend samples. Blood glucose response levels were obtained using Therasense Freestyle Glucometers (Alameda, CA). Self retracting safety lancets (Arta Plast AB, Fisher HealthCare, Houston, TX) used to obtain blood samples were purchased from Fisher. Insulin and c-peptide levels were analyzed on microwell plates and read on a Thermo Electron Corporation Multiskan MCC microplate reader (Shanghai, China).

Participants

Eligibility to participate in this study required willingness to adhere to the research protocol and absence of any chronic diseases. Participants were recruited by direct requests and emails to the North Carolina State University (NCSU) Food Science Department. Volunteers were all considered normal for research purposes. The study began on June 29, 2005 and ended on August 10, 2005 when 10 healthy volunteers completed the study. A consent form approved by the NCSU Institutional Review Board was signed by each volunteer. Financial compensation was provided for the time subjects participated in the study. A questionnaire covering age, gender, ethnicity/ culture, medical history, and

medications was used to screen potential volunteers. Body Mass Index (BMI) kg/m^2 was calculated from height and weight based on volunteer recall.

Proximate analysis of Beauregard (US) sweet potato and white potato were completed to determine the amount that would contain 50 grams of available carbohydrate from the skin, flesh, and or whole grated potato (Table 2.1).

Beauregard sweet potato roots grown in Clinton, North Carolina were, washed, skinned, and prepared by grating using a Cuisinart ® food processor (East Windsor, NJ). All 200-g portions of sweet potato (skin, flesh, or whole) were weighed, placed in Ziplock bags, and stored in food grade freezers for a maximum of one week until needed for participants. The treatments consumed for this study were T1= Glucose drink, T2= whole sweet potato, T3= sweet potato flesh, T4= sweet potato skin, T5= white potato, T6= white potato plus Caiapo. All sweet potato samples were consumed raw, unexposed to any cooking heat. White potato samples were microwaved in the food grade lab for 3 minutes at 750 watts to reduce potential solanine toxicity. Frozen samples were thawed each morning for 1.5 hours at room temperature (22 ^{o}C) before consumption. Approximately 4 grams of Caiapo was added to white potato and mixed prior to eating. Each treatment was consumed on two different days by each subject to determine glycemic index.

Volunteers were seated at desks and asked to record the last meal consumed prior to the required eight hour fasting and arriving at the test site. Fisher® HealthCare Brand (Arta Plast AB, Fisher HealthCare, Houston, TX) self retracting safety lancets were provided to prick the tips of their fingers for fasting glucose level determination by Therasense® Freestyle glucometers (Alameda, CA). Participants were then asked to consume 50-g of

Fisherbrand Sun-Dex Glucose Tolerance Test Beverages (Houston, TX) to obtain the standard against which sweet potato samples were measured. Blood glucose levels were subsequently measured at times 30, 60, 90, and 120 minutes after they finished eating the sample. An additional 400 µL of blood were collected in BD Microtainer serum separator tubes, centrifuged ten minutes, and frozen for further analysis of c-peptide and insulin concentrations. Participants consumed one treatment (T2 to T6) on weekday mornings. They sampled each treatment twice over the course of a month and a half.

The data on glucose levels were entered into Microsoft Excel and to graph glucose response curves. The positive area under the curve AUC between 0 – 120 minutes were measured for all treatments and the average area for treatments T2 through T6 was calculated as a percentage of the area under the curve (AUC) for the glucose drink treatment in order to determine the glycemic index as outlined by Jenkins et al. (1981). This seemingly older method was used for accuracy in lieu of previous inaccurate calculations. The graphed glucose responses were printed for each participant, cut, and weighed to represent the area under the curves. The glycemic index was calculated as a fraction of the weight measured from the treatments to the glucose standard. This methodology simulated the measurement by Jenkins et al. (1981) which calls for the percentage of each sample's area to that of the glucose drink for determination of its glycemic index number.

Serum insulin and C-peptide concentrations from times 0, 60, and 120 minutes were measured from thawed blood samples using the Mercodia Insulin ELISA 10-1113-01 (Sweden) assay and Mercodia C-peptide ELISA (10-11136-01) kits by Mercodia (Sweden, ALPCO Diagnostics, Windham, NH).

Glucose and Insulin levels were read at 450 nm. Serum glucose concentrations were also measured in these samples using a glucose assay kit (GAGO-20, Sigma, St. Louis, MO). Graphs were made from values of insulin and C-peptide to check for trends, relationships, and interactions. The glucose oxidase assay was used to verify blood glucose concentrations displayed from the Theresense glucometers to ensure accuracy of their readings.

Statistical Methods

ANOVA tables from GLM means provided p values considering treatments and subjects as main variables. Duncan's Multiple Range procedure calculated the mean glycemic index for each treatment and indicated statistical similarities among them ($P< 0.05$). The means slopes of trend lines from 0 – 60 minutes and 60 – 120 minutes for insulin and C-peptide concentrations were graphed and analyzed. The slopes from the two time intervals were also analyzed. Duncan's procedure was used any treatments with significantly different insulin or C-peptide release or absorption.

The Type III Sum of Squares *P* values were used to assess the probability of obtaining results similar to those in our study. Type II Sum of Squares corrects for as many factors in the model and where there are unequal numbers of observations in each group.

Results and Discussion

Anthropometric Measurements

The mean age for participants was 29.7 ± 9.69 with a range of 21 to 53 years. The mean weight of volunteers was 179.5 ± 33.49 lb (81.4 ± 15.2 kg). The height average of the ten participants was 5'8" ± 3.30" (1.71± .08M). The average BMI was 27.04 ± 4.74. This

average BMI is considered overweight by the U.S. Dietary Guidelines (2005). There were 4 males and 6 females, all of whom were non-smokers.

Proximate Analysis

Sweet potatoes were analyzed for macro nutrients and compounds in order to calculate 50 grams carbohydrate by difference as outlined in Brand-Miller et al (1992). GI testing requires that portions of both the reference foods and the test foods contain the same amount of available carbohydrate, typically 25 or 50 g (Foster– Powell et al., 2002). There are higher percentages of fiber and ash in the skin of Beauregard sweet potatoes as shown in Table 2.1. There is more flesh than skin per tuber of sweet potato. Crude protein levels and the percentage of fat are constant in both whole and skin portions of sweet potato. The consistency could indicate that the majority of protein in sweet potatoes is located in the skin. The Caiapo protein, which was studied by Ludvik (2003, 2004) for antidiabetic properties, was extracted from the skin of the Japanese white skinned sweet potato.

Table 2.1 Proximate Analysis of Beauregard, White Star, and White Potatoes

Sweet Potato (Cultivars)	Crude Protein	Fat %	Fiber %	Ash %	NFE %	Dry Matter %	BCA Protein %
Beauregard Whole	2.57	1.26	2.33	3.71	90.13	17.57	
Beauregard Skin	2.41	1.44	9.2	7.36	79.59	18.61	1.8
White Potato Whole	2.31	0.39	1.23	0.67	95.4	17.86	

Descriptive Analysis of Glucose Responses

Table 2.1 shows the calculated GI's for the five samples studied and glucose standard. The GI of raw white potato was reduced from 52 to 30 when 4 g of Caiapo was added.

A blood glucose lowering effect was observed at two hours after consuming each sample by the decreased blood glucose levels in all treatments. Each treatment reduced glucose response values returning to levels comparable to those of fasting without rising to levels similar to the glucose standard. It seems that the substances that lower the blood glucose are in the sweet potato skin.

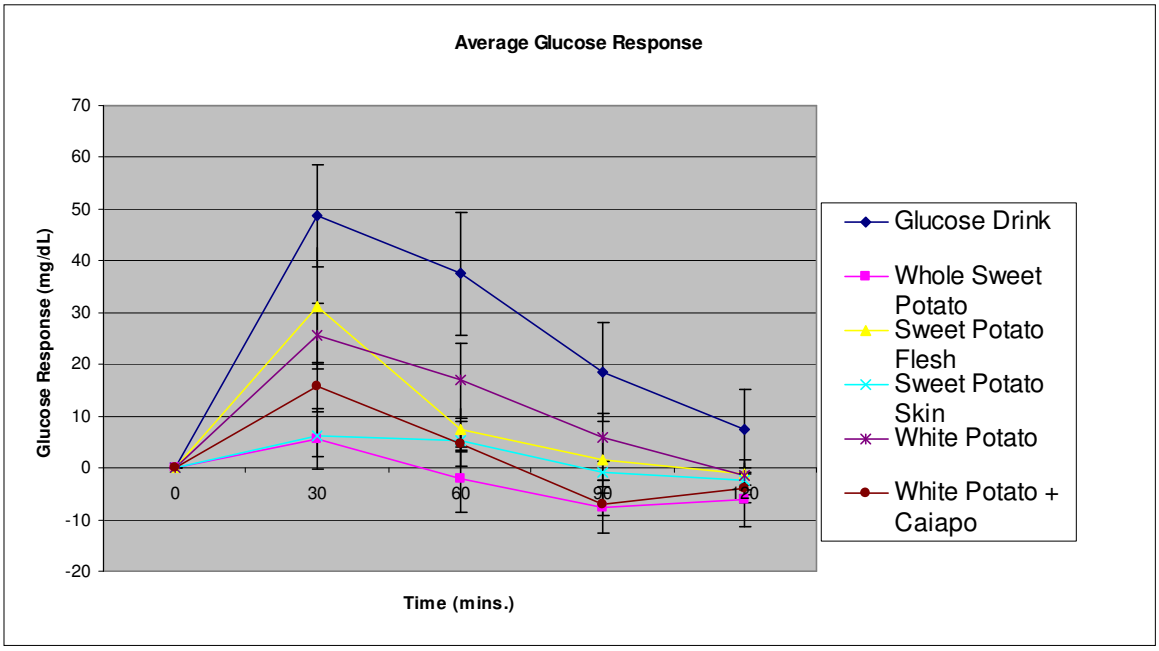

Figure 2.1 Average glucose response curve relative to fasting blood glucose concentrations for each subject.

Glycemic Indices

Treatments with glycemic indices of zero were not excluded from the data because previous data from Zakir (2005) revealed the possibility of obtaining a decrease in blood

glucose levels after sweet potato consumption, especially in skin fractions. That occurrence would calculate a GI of zero which was still important to calculating the mean glycemic response across subjects. Figure 2.1 shows the mean glycemic indices for the five treatments studied which were compared to the glucose standard. The GI of raw white potato was reduced from 52 to 30 when 4g of Caiapo was added. The glycemic indices for all samples are low when compared to the GI standard according to GI standards. Type III Sum of Squares indicated differences among subjects (P= 0.004; α = 0.05), but no significant difference among treatments (P= 0.06).

Table 2.2 Glycemic Index of the Treatment Studied.

Sample	GI	SEM
Glucose	100	
Whole Sweet Potato	39 ^{A,B}	15.36
Sweet Potato Flesh	49 ^{A}	12.11
Sweet Potato Skin	26 ^{A,B}	6.70
Whole White Potato	52 ^{A}	13.71
White Potato + Caiapo	30 ^{A,B}	8.09

Values with the same superscript letter are not significantly different ($P > 0.05$)

Descriptive Analysis of Insulin Response

Figure 2.2 shows the mean insulin response to sweet potato and white potato samples. Insulin response to the sweet potato and white potato samples were all 9.32 ± 1.36 mU/L less than the glucose standard. The slopes of insulin response levels for the first and second

hours were analyzed using Duncan's statistical grouping of means to determine differences in the rate of insulin release in the first hour and rate of insulin uptake in the second hour.

There were no significant differences in insulin concentration means by treatment over a two-hour period (P= 0.319; α = 0.05). The lack of significant difference between samples indicates that Caiapo did not significantly increase pancreatic insulin release in the first hour. The results from white potato + Caiapo contradict with conclusion of Ludvik et al. (2002, 2003) for increased insulin efficiency. Whole sweet potato had the fastest rate of concentration increase among samples (0.081 mU/L/min), but not significantly higher than white potato (0.079 mU/L/min). White potato + Caiapo had an insulin release rate of 0.073 mU/L/min, sweet potato skin rose at 0.034 mU/L/min, and sweet potato flesh increased at 0.027mU/L/min. The slope of glucose treatment (T1) was significantly greater than the other treatments showing that the greater rise in blood glucose caused greater rise in insulin.

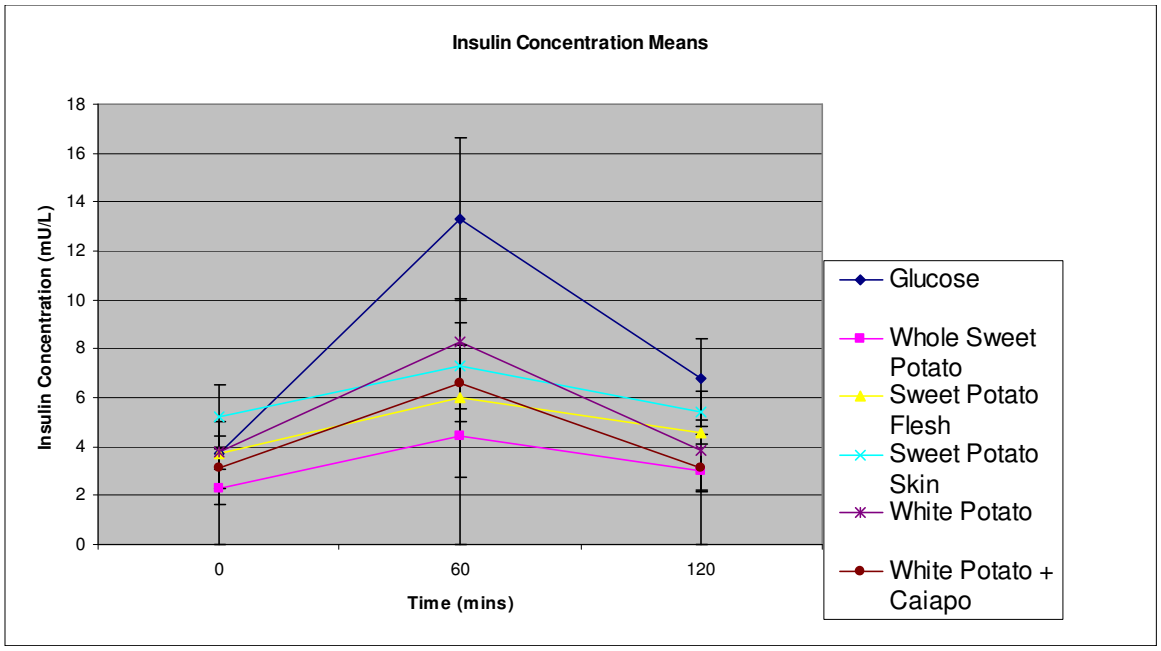

Figure 2.2 Mean Serum Insulin Levels from Subjects Consuming 50 carbohydrate from Glucose; Beauregard Sweet Potato and White Potato (n=10)

Participants consuming white potato with 4 g of Caiapo had a mean fasting insulin level of 3.14 ± 2.49 mU/L. Their mean post-prandial insulin level rose to 6.57 ± 4.68 mU/L at 60 minutes and decreased to 3.14 ± 2.87 mU/L at 120 minutes. A difference of 3.43 mU/L was observed between fasting and 60 minutes. Participants consuming white potato alone had mean fasting insulin levels at 3.79 ± 3.66 mU/L which rose to 8.29 ± 5.22 mU/L after 60 minutes and declined to 3.84 ± 3.02 mU/L after 120 minutes. A difference of 4.5 mU/L was observed between fasting and 60 minutes.

The Type III Sum of Squares analysis on the rate of insulin concentration increase, represented by slopes indicated insulin uptake pointed to no significant differences among subjects or treatment; P= 0.0530, P=0.2562 respectively (α= 0.05). Duncan's grouping of

slopes clustered all slope means under one category (A). Whole sweet potato flesh increased at the fastest rate of 0.0812 mU/L/min, whole white potato increased at 0.0789 mU/L/min.

The Type III Sum of Squares analysis of decreasing insulin concentrations indicated no significant differences in subjects or treatments: P= 0.3915, P= 0.3299 respectively (α= 0.05). Duncan's grouping of means categorized all means in one cluster (A). White potato decreased fastest (-0.081 mU/L/ min) and not statistically different from white potato + Caiapo (-0.068 mU/L/ min).

Sweet potato skin displayed a more plateau-like insulin response curve in Figure 2.2 indicating minimal insulin release in response to very low glucose increment. Research investigating the mechanisms causing this low insulin response despite the high starch content in sweet potatoes can further explain these phenomenon. Fiber content, starch granule structure, or bioactive components may play a role in the low glucose and insulin response of these sweet potatoes. These components can hinder enzymatic digestion of the starch creating lower glycemic indices.

Descriptive Analysis of C-Peptide Response

Figure 2.3 displays the mean C-peptide response of the samples (T1-T6). C-peptides are the protein fraction of pro-insulin that is not metabolized immediately after release, because it does not react with specific receptors.. Quantifying C-peptide levels post prandially specifies how much insulin was released from the pancreas. The trend lines in Figure 2.3 demonstrate that all raw Beauregard sweet potato samples elicited lower C-peptide response levels than white potato and white potato plus Caiapo. The mean C-peptide increase among sweet potato samples was 181 ± 43.95 pmol/L. Conversely, white potato samples showed a mean increase of 457 pmol/L from fasting to 60 minutes, while white

potato plus Caiapo samples had a mean increase of 419 pmol/L during the fasting to 60 minute time interval. These differences show the lack of a short term increase from adding Caiapo.

Analysis of the 60 to 120 minute time interval reveal that white potato plus Caiapo samples had a greater decrease in c-peptide levels (466 pmol/L) than white potato alone (344 pmol/L). There was greater variation in the raw Beauregard sweet potato samples in the last hour of c-peptide metabolism. Whole sweet potato samples had a mean c-peptide reduction of 234 ± 1.08 pmol/L, mean c-peptide levels in sweet potato flesh increased 6 ± 1.77 pmol/L, and levels decreased by 64 ± 23.4 pmol/L in sweet potato skin samples.

The GLM statistical analysis comparing c-peptide concentration increases indicated differences among subjects and treatments (P= 0.0002, P= 0.0142 respectively, α= 0.05). White potato + Caiapo (7.422 pmol/L/min) had the fastest increase in c-peptide concentrations. Duncan's Multiple Range test groups classified white potato (6.084 pmol/L/min), and sweet potato flesh (5.553 pmol/L/min) as not being statistically different. White potato and sweet potato flesh were not statistically different from sweet potato skin (2.659 pmol/L/min) and whole sweet potato (2.636 pmol/L/min).

There were no statistical differences in the rate of c-peptide concentration decrease by treatment (P= 0.0870), but there were significant differences between subjects (P= 0.0133). Concentration decreases in sweet potato skin (-2.078 pmol/L/min), whole sweet potato (-.2.398 pmol/L/min), white potato (-4.427 pmol/L/min), sweet potato flesh (-4.618 pmol/L/min), and white potato + Caiapo (-6.412 pmol/L/min) were all statistically similar.

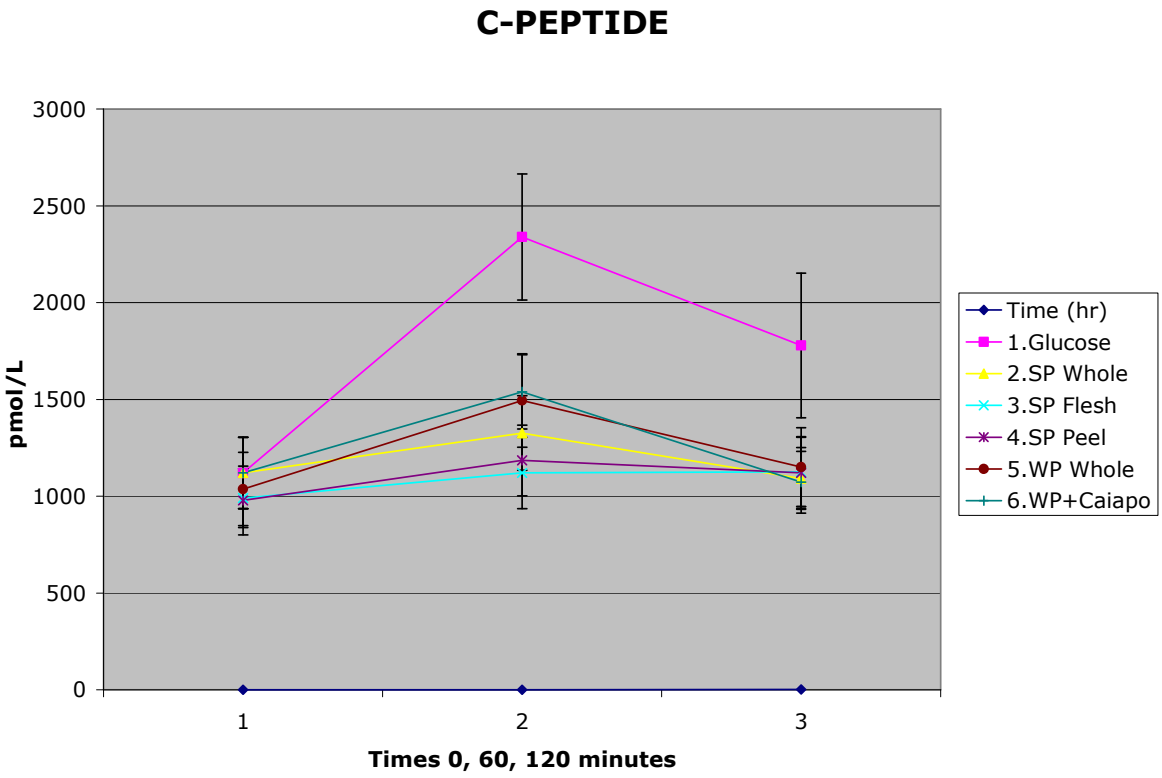

Figure 2.3 Mean Serum C-Peptide Levels from Subjects Consuming 50 g carbohydrate from Glucose; Beauregard Sweet Potato and White Potato (n=10)

Glucose Analytical Methods

Serum glucose was measured by the glucose oxidase/ glucoperoxidase reaction on the 0, 60, and 120 minute samples to verify the glucometer readings. Glucose concentrations for the enzyme coupled assay and those displayed on the glucometers were similar across glucometers (p= 0.1673) using paired t test comparison. The mean value observed from glucometers was 97.15 mg/dL and mean glucose response level from glucose oxidase reactions is 94.28 mg/dL. The standard error of their difference was 2.88 mg/dL (n=180). The slight difference between the assays could have been due to glucose metabolism by blood cells before the samples could be cooled. Lack of significant differences between

glucometers and glucose oxidase reactions points to the accuracy of the Therasense glucometers for purposes of our glycemic index calculations.

Investigation of the rate to which insulin and C-peptide concentrations increase and decrease reveals that all sweet potato and white potato roots studied elicit statistically similar results. An increase in glucose uptake would have been illustrated in a graph by significantly lower insulin values in white potato + Caiapo when compared to insulin levels elicited from white potato alone at 120 minutes. Since trend lines rose and declined at statistically similar rates, Caiapo cannot be attributed to any change in insulin uptake in a short term, one hour interval.

The GI of these samples reveals the extent to which these samples actually would maintain low blood glucose levels. The statistical differences in subjects may be explained by variations in daily carbohydrate intake, time to finish eating samples, or degree to which samples were thawed. Literature on the GI of sweet potatoes cultivars gives a range from 48 to 78 across cultivars (Foster-Powell et al, 2002). The Beauregard sweet potato studied is in the lower range of GI's observed across cultivars.

Summary

The results from this study provide values for the glycemic index from NC Beauregard sweet potatoes with respect to skin, flesh, and whole. Further data on insulin and c-peptide responses suggests that glucose concentration is closely associated with insulin release. Similarly, there appeared to be lack of a significant effect of Caiapo in changing insulin and c-peptide release rates in a two hour interval post-prandially. This suggests that the short term effect of Caiapo treatment on the glucose response curve in this study was not

due to altered insulin metabolism. Knowledge that raw Beauregard sweet potatoes show a low glycemic index is beneficial for diabetic patients who eat them. It is noteworthy that there were no significant differences between raw sweet potatoes and raw white potatoes. Investigation of the effect cooking my change glycemic index values and create statistical differences can add more significance to the recommendation for sweet potatoes.

Most individuals consume sweet potatoes after some type of cooking process. Further research is needed to understand how cooking affects the glycemic index of sweet potatoes. Understanding how much the glycemic index increases will improve recommendations to sweet potato consumption, especially for diabetic patients. Should glycemic indices remain low (<55), cooked sweet potatoes may aid in controlling blood glucose levels.

Literature Cited

AOAC, (1990) Official Methods of Analysis. The Association of Official Analytical Chemists. 15th Edition, Arlington, USA.

Brand Miller J, Pang E, Bramall L. (1992) Rice: a high or low glycemic index food? Proc. Nutr Soc Austr 17:226.

Brand-Miller, Jennie; Petocz, Peter; Hayne, Susan; Colaguri, Stephen (2003) Low-glycemic index diets in the management of diabetes. Diabetes Care. 26;8:2261-2267.

Brunzell JD, Lerner RL, Hazard WR. (1971) Improved glucose tolerance with high carbohydrate feeding in mild diabetes. New Engl J Med 284:521

Crapo PA, Reaven G, Olefsky JM (1977) Postprandial glucose and insulin responses to different complex carbohydrates. Diabetes 26:1178-83.

Crapo PA., (1985) Simple Versus Complex Carbohydrate use in the diabetic diet. Ann Rev Nutr 5:95-114.

Cryer PE (1997) Hypoglycemia: Pathophysiology, Diagnosis and Treatment. Oxford University Press: New York.

Food & Nutrition Board and Institute of Medicine (2000) Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. The National Academies Press.

Foster-Powell K, Holt SHA, Brand-Miller JC (2002) International table of glycemic index and glycemic load values Am J Clin Nutr. 76:5-56.

Jenkins DJA, Wolever TMS, Taylor RH, et al. (1981) Glycemic index of foods: a physiological basis for carbohydrate in non-insulin-dependent diabetic patients. Am J Clin Nutr; 34:362-6

Jenkins DJA, Wolever TMS, Taylor RH. (1980) Rate of digestion of foods and postprandial glycaemia in normal and diabetic subjects. Br Med J 281:14-17.

Jenkins DJA, Wolever TMS, Jenkins AL (1983) The glycaemic index of foods tested in diabetic patients: A new basis for carbohydrate exchange favoring the use legumes. Diabetolgia; 24:257-64.

Ludvik B, Mahdjoobian K, Waldhaeusi W, Hofer A Prager R, Kautzky-Willer A ,Pacini G (2002) The effect of *Ipomea batatas* (Caiapo) on glucose metabolism and serum cholesterol in patients with type II diabetes. Diabetes Care. 25;1:239-240.

Ludvik B, Mahdjoobian K, Waldhaeusi W, Hofer A, Prager R, Kautzky-Willer A, Pacini G (2003) Mode of Action of *Ipomea Batatas* (Caiapo) in type II diabetic patients. Metabolism. 52;7:875-880.

Ludvik B, Neuffer B, Pacini G. (2004) Efficacy of *Ipomoea batatas* (Caiapo) on diabetes control in type 2 diabetes subjects treated with diet. Diabetes Care. 27(2)436-40.

Nielsen H, Nielsen Gunnar L (1989) Preprandial blood glucose values: influence on glycemic response studies. Am J Clin Nutr; 49: 1243-6.

US Dietary Guidelines (2005)

http://www.health.gov/dietaryguidelines/dga2005/document/html/chapter3.htm . accessed June 20, 2007.

WHO, (1999) Diabetes. World Health Organization Fact Sheet No. 312. (http:www.who.int/medicacentre/Factsheets/FS312leu? Accessed 06/19/2007.

Zakir S, Sarwar M, Allen JC, Butt MS, Allen H. (2005) Effect of sweet potato on insulin efficiency of normal and diabetic subjects in Pakistan. Eur J Sci Res 10(1):87-97.

Table 2.3 Descriptive Statistics of Blood Glucose (mg/dL), Insulin (IU/L), and C-peptide pmol/L levels with Various Treatments of Normal Subjects.

Parameters	**Glucose (Control)** (T1)	**Whole Sweet Potato** (T2)	**Sweet Potato Flesh** (T3)	**Sweet Potato Skin** (T4)	**White Potato** (T5)	**White Potato + Caiapo** (T6)
Blood Glucose Fasting						
Mean	96.30	99.40	93.60	88.78	89.50	93.39
SD	10.47	22.56	13.57	12.24	18.17	12.16
30 Minutes						
Mean	144.90	105.00	124.80	96.11	113.06	107.78
SD	31.51	8.58	36.56	8.44	24.59	16.90
One Hour						
Mean	133.80	97.15	100.90	94.17	108.22	98.28
SD	39.31	13.44	16.77	11.57	23.84	14.94
90 Minutes						
Mean	114.80	91.85	95.30	87.83	96.33	86.44
SD	29.10	10.03	14.10	10.38	11.48	10.01
Two Hours						
Mean	103.80	93.35	92.50	86.61	89.56	88.78
SD	24.65	10.42	13.39	9.29	17.28	13.40
Insulin Fasting						
Mean	3.74	2.27	3.73	5.24	3.79	3.14
SD	2.10	1.89	2.77	3.92	3.66	2.49
One Hour						
Mean	13.29	4.45	6.01	7.31	8.29	6.57
SD	9.93	5.08	3.55	5.29	5.22	4.68
Two Hour						
Mean	6.77	2.98	4.58	5.40	3.84	3.14
SD	4.97	2.47	3.50	2.67	3.02	2.87
C-Peptide Fasting						
Mean	1120	1120	990	978	1037	1120
SD	184	184	152	179	190	184
One Hour						
Mean	2339	1327	1120	1184	1494	1539
SD	326	192	184	182	242	210
Two Hour						
Mean	1779	1092	1126	1120	1151	1073
SD	375	159	181	184	203	202

Appendix Table 2.1 GI Means for Subjects by Treatment Analysis of the Trial Means

The GLM Procedure

Duncan's Multiple Range Test for (2005)

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	36
Error Mean Square	0.053451

Number of Means	2	3	4	5
Critical Range	.2097	.2204	.2275	.2325

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt
A	0.5202	10	wp
A			
A	0.4887	10	spf
A			
B A	0.3915	10	spw
B A			
B A	0.3014	10	wpc
B			
B	0.2561	10	sps

Appendix Table 2.2 Insulin Slopes for the First Hour

The GLM Procedure

Dependent Variable: time

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	12	0.10527658	0.00877305	1.92	0.0747
Error	29	0.13268874	0.00457547		
Corrected Total	41	0.23796531			

R-Square	Coeff Var	Root MSE	time Mean
0.442403	118.3320	0.067642	0.057163

Source	DF	Type I SS	Mean Square	F Value	Pr > F
person	8	0.07951147	0.00993893	2.17	0.0605
trt2	4	0.02576511	0.00644128	1.41	0.2562

Source	DF	Type III SS	Mean Square	F Value	Pr > F
person	8	0.08221254	0.01027657	2.25	0.0530
trt2	4	0.02576511	0.00644128	1.41	0.2562

Appendix Table 2.3 Insulin Slopes for the First Hour

The GLM Procedure

Duncan's Multiple Range Test for time

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	29
Error Mean Square	0.004575
Harmonic Mean of Cell Sizes	8.181818

NOTE: Cell sizes are not equal.

Number of Means	2	3	4	5
Critical Range	.06840	.07187	.07413	.07573

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt2
A	0.08108	6	wsp
A			
A	0.07896	9	wp
A			
A	0.07296	9	wpc
A			
A	0.03376	9	sws
A			
A	0.02702	9	spf

Appendix Table 2.4 Insulin Slopes for the Second Hour

The GLM Procedure

Dependent Variable: time

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	12	0.09375465	0.00781289	1.04	0.4406
Error	30	0.22542299	0.00751410		
Corrected Total	42	0.31917764			

R-Square	Coeff Var	Root MSE	time Mean
0.293738	-184.9012	0.086684	-0.046881

Source	DF	Type I SS	Mean Square	F Value	Pr > F
person	8	0.05902040	0.00737755	0.98	0.4691
trt2	4	0.03473424	0.00868356	1.16	0.3499

Source	DF	Type III SS	Mean Square	F Value	Pr > F
person	8	0.06603646	0.00825456	1.10	0.3915
trt2	4	0.03473424	0.00868356	1.16	0.3499

Appendix Table 2.5 Insulin Slopes for the Second Hour

The GLM Procedure

Duncan's Multiple Range Test for time

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	30
Error Mean Square	0.007514
Harmonic Mean of Cell Sizes	8.571429

NOTE: Cell sizes are not equal.

Number of Means	2	3	4	5
Critical Range	.08552	.08987	.09269	.09470

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt2
A	-0.00955	8	spf
A			
A	-0.03322	9	sws
A			
A	-0.03792	8	wsp
A			
A	-0.06793	9	wpc
A			
A	-0.08065	9	wp

Appendix Table 2.6 C-Peptide Slopes for the First Hour

The GLM Procedure

Dependent Variable: Time Time

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	13	774.107279	59.546714	4.69	0.0001
Error	36	456.970039	12.693612		
Corrected Total	49	1231.077318			

R-Square	Coeff Var	Root MSE	Time Mean
0.628805	73.14654	3.562810	4.870783

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Person	9	590.8322224	65.6480247	5.17	0.0002
trt2	4	183.2750566	45.8187642	3.61	0.0142

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Person	9	590.8322224	65.6480247	5.17	0.0002
trt2	4	183.2750566	45.8187642	3.61	0.0142

Appendix Table 2.7 C-Peptide Slopes for the First Hour

The GLM Procedure

Duncan's Multiple Range Test for Time

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	36
Error Mean Square	12.69361

Number of Means	2	3	4	5
Critical Range	3.231	3.397	3.505	3.583

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt2
A	7.422	10	wpc
A			
B A	6.084	10	wp
B A			
B A	5.553	10	spf
B			
B	2.659	10	sws
B			
B	2.636	10	wsp

Appendix Table 2.8 C-Peptide Slopes for the Second Hour

The GLM Procedure

Dependent Variable: Time Time

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	13	486.818037	37.447541	2.62	0.0112
Error	36	513.925564	14.275710		
Corrected Total	49	1000.743601			

R-Square	Coeff Var	Root MSE	Time Mean
0.486456	-94.77533	3.778321	-3.986608

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Person	9	360.4234474	40.0470497	2.81	0.0133
trt2	4	126.3945896	31.5986474	2.21	0.0870

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Person	9	360.4234474	40.0470497	2.81	0.0133
trt2	4	126.3945896	31.5986474	2.21	0.0870

Appendix Table 2.9 C-Peptide Slopes for the Second Hour

The GLM Procedure

Duncan's Multiple Range Test for Time

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	36
Error Mean Square	14.27571

Number of Means	2	3	4	5
Critical Range	3.427	3.603	3.717	3.800

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt2
A	-2.078	10	sws
A			
A	-2.398	10	wsp
A			
B A	-4.427	10	wp
B A			
B A	-4.618	10	spf
B			
B	-6.412	10	wpc

Chapter 3

Effect of Cooking Methods

On Glycemic Response of Sweet Potato

Abstract

Previously, glycemic index of raw Beauregard sweet potatoes was found to be 39 ± 15.36 (whole), 48 ± 12.11 (flesh), 25 ± 6.70 (skin). An understanding of the effect cooking has on the amount of available glucose will aid in the recommendation for including sweet potatoes as a regular component in American diets. Processing of starch breaks down amylopectin and amylose starch granules bringing about maltose and dextrins that are more readily digested by pancreatic amylase. This phenomenon, theoretically, should increase the glycemic index (GI) of a sweet potato.

Twelve volunteers consumed 25 g of available carbohydrate from Beauregard sweet potato skin and flesh separately that were subjected to conventional cooking methods; baking 190.5 °C for 1 hour, microwaving for five minutes in a 1000 watt microwave, dehydrating at 60 °C for 16 hours, and steaming at 100 °C for 45 minutes. Available carbohydrate was determined by difference from proximate analysis of lipid, total dietary fiber, moisture, and ash. Fasted participants measured blood glucose levels at 0, 30, 60, 90, and 120 minutes after consuming 25 g of carbohydrate from test foods or glucose. Calculations of the elicited glycemic indices from these methods revealed that the GI of steamed flesh was 63 ± 8.4, a medium glycemic index food. Blood glucose responses to baked and microwaved flesh also indicated medium glycemic index foods, with GI of 64 ± 10.11 and 66 ± 13.29, respectively. However, dehydrated sweet potato flesh had a low glycemic index (40 ± 8.24) similar to that of raw sweet potato flesh (28 ± 7.26). Steamed skin, baked skin, and dehydrated flesh did not have a statistically different glycemic index ($p > 0.05$) from that of raw sweet potatoes. This study suggests that North Carolina Beauregard sweet potato skin and dehydrated sweet

potato flesh may be consumed as low and medium GI foods even when cooked, which may prove beneficial for diabetic or insulin resistant consumers.

Introduction

Our previous study on whole raw sweet potatoes found a low GI of 39 ± 15.36. We do not know the components associated with the low GI in raw sweet potatoes. Proteins, fiber, or starch are components that could be attributed to the low glycemic index of sweet potatoes. However, sweet potatoes are rarely consumed raw in developed and developing countries due to the starchy flavor. In developing countries, the consumption of sweet potato appears to be inversely proportional to income level, and they are typically viewed as a food to be consumed only for survival (Truong, 1986). Although generally considered sweet by definition, there is potentially a large range in perceived sweetness amongst cultivars, depending on sugar components and starch conversion at cooking (Takahata et al., 1992).

Theoretically, an increase in simple sugars should cause higher glycemic indices due to improved sugar availability. Other compounds, such as fiber, can confound this effect by maintaining low GI values. This study aims to investigate the change in glycemic index after cooking sweet potatoes under conventional domestic methods.

Sweet potatoes are more commonly consumed cooked using various methods. Baked sweet potatoes are popular in the Americas. The North Carolina Sweet Potato Commission recommends baking at a temperature of 350 °F (190 °C) for forty minutes. Sweet potatoes are also boiled and steamed in many Asian countries. Dehydrated sweet potatoes are consumed as chips for snacks. This objective of this study was to determine the effect of cooking by different methods on the GI of sweet potato on the GI of sweet potato skin and flesh.

Materials & Methods

Reagents

Hydrocholoric acid (HCl) was purchased from Fisher Scientific (Fair Lawn, NJ) to digest dehydrated samples. Sodium sulfate (anhydrous) was used to dry lipid samples dissolved in n-hexane, also purchased from Fisher Scientific. Heat stable α- amylase, protease, and amyloglucosidase were provided in a Megazyme total dietary fiber assay procedure kit AOAC 991.43 (Ireland, American Association of Cereal Chemists, St. Paul, MN). MES/TRIS buffer (2N-morpholino ethanesulfonic acid and tris(hydroxymethyl) aminomethane) (0.05 M) at pH 8.2 adjusted with 6N HCL was prepared using reagents from Sigma- Aldrich (St. Louis, MO). Celite, acid-washed, pre-washed from World Minerals was also used in total dietary fiber assay. Ethanol (95%), 78% ethanol (AAPER Alcohol, Shelbyville, Kentucky), and acetone used for washing of total dietary fiber samples were reagent grade (Fisher Scientific, St. Louis, MO).

Instrumentation

Sweet potato roots were skinned using a household potato peeler. Conventional ovens were used in for baking. Temperature was checked by an oven thermometer. Sweet potato samples were dehydrated in a Precision Scientific economy oven (Chicago, IL) that operated by mechanical convection. A 10 cup household steamer was used to steam sweet potato slices. Sweet potato samples were microwaved at full power (750 watt) in General Electric 'Hotpoint' ® (Fairfield, CT) in food grade areas of the lab.

A Cuisinart ® 14-cup food processor blended samples after cooking for freeze drying. Cooked sweet potato samples were prepared for proximate analysis using a vacuum bottle type 4.5 Liter benchtop freeze dryer (Labconco) (Kansas City, Missouri) to ensure complete

dehydration of pureed sweet potato samples. Coffee grinders were used to create a sweet potato powder suitable for analysis of macronutrients.

Digestion conditions were simulated in hot water baths made by Precision Scientific. A rotary evaporator was utilized to remove n-hexane solvent from solution in lipid extraction procedures. Buffer pH was adjusted using 6N HCL and 6N NaOH. Fritted Pyrex 50 mL ASTM 40-60 C crucibles by Corning filtered samples were used in total dietary fiber separation extraction methods. Small vacuum pumps were used for suction of crucibles. Samples were ashed at 525 ^{o}C in a muffle furnace by Barnstead/ Thermolyne and cooled in desicators. Therasense ® Freestyle glucometers were used to monitor changes in blood glucose levels of volunteers.

Cooking Methods

Sweet potatoes were grown at the NCSU Research Farm in Clinton, North Carolina and held in a temperature controlled cooler (55 °C) in the North Carolina State University Horticulture Department after harvest. Samples were retrieved from storage coolers weekly, washed and allowed to dry at room temperature (22 °C) for 10 minutes. Inedible portions were removed and not used in cooking. Samples were then skinned for flesh and skin fractions to be separated and weighed to 25 g available carbohydrate quantities determined from proximate analysis. The conventional cooking methods investigated in this study were baking, dehydrating, steaming, and microwaving. These cooking methods were intended to be representative of common sweet potato preparations.

Raw sweet potato samples were sliced and baked to 325 °F (190 °C) for one hour in sealed aluminum foil packets. Peeled raw sweet potatoes were thinly sliced using a Cuisinart ® food processor with a 4 mm blade. The slices were then placed on baking sheets in a

dehydrator at 60 °C for 16 hours. Other slices were placed in a home steamer for 45 minutes. Sweet potatoes were sliced and placed in a domestic microwave for 5 minutes.

Research has shown that if sweet potato roots are cut into strips and cooked rapidly, significant amounts of starch remain, whereas the cooking of whole roots allows a more complete conversion of starch into dextrins and sugars (Collins & Walter, 1985). Since processing of sweet potato into pieces before cooking is standard method used in both household and industrial sweet potato preparation (Lewthwaite et al., 1997), it was used in this study.

After cooking, samples were placed in a refrigerator (13 °C) for a maximum of a week until needed then reheated for one minute in a microwave for volunteer consumption. The shelf life was one week and thereafter any un-used samples were discarded.

Proximate Analysis

Our study began with quantifying carbohydrate content in raw, baked, steamed, and dehydrated sweet potatoes. Analysis was conducted after cooking to observe the possible change in macro nutrient levels through leaching or other manner. The proximate compositions of total dietary fiber, ash, lipid, and moisture contents for the different cooking methods of sweet potatoes were determined using a standard Association of Official Analytical Chemists (AOAC, 1995) method and available carbohydrate content was calculated by difference (Brand- Miller et al., 1992; Ramdath et al., 2004).

Total Dietary Fiber

The AOAC 991.43 Megazyme kit (Ireland, American Association of Cereal Chemists, St. Paul, MN) methods required samples to be cooked at 100 °C with heat stable α–amylase to give gelatinization, hydrolysis and depolymerization of starch. Incubation at 60 °C with protease followed to solubilize and depolymerise proteins and amyloglucosidase to hydrolyze starch fragments to glucose. Treatment with ethanol precipitated the soluble fiber and removed depolymerized protein and glucose from starch. The residue was then filtered and washed with 78% ethanol, 98% ethanol, and acetone. One duplicate was analyzed for protein and another for ash. Total dietary fiber was then calculated by difference from filtered and dried residues.

Lipid

Acid hydrolysis was used to release bound lipids, polar and non- polar, by dissociating lipid-starch and lipid-protein intermolecular forces. Samples were hydrolyzed with hydrochloric acid under heat. Extraction of lipids using hexane retained lipid in the organic solvent. The organic layer was filtered through sodium sulfate and evaporated at 40 °C under reduced pressure using a rotary evaporator. The resulting weight was subtracted revealing the amount of lipid present.

Moisture

Sweet potatoes were first cooked and then placed in a moisture determination dehydrator. The sample was automatically weighed, dehydrated to a constant weight, and the percentage of moisture was then calculated and displayed.

Protein

The NC State University soil science department determined dry matter nitrogen levels as a part of the analysis using a C-H-N 2400 CO2 Elemental Analyzer (Perkin Elmer, Norwalk, CT). The amount of protein was determined from N content x 6.25 (16% N in sweet potato protein).

Ash

Ash was measured in the total dietary fiber procedure by placing digested starch residue in a muffle furnace for five hours at 525^{o}C (Megazyme; Ireland, American Association of Cereal Chemists, St. Paul, MN). The high constant temperature destroyed all compounds other than minerals found in the sweet potato.

Human Subject Panel

The experimental procedure was approved by the NCSU Institutional Review Board. Once 25 grams of carbohydrate was determined by proximate analysis of other macronutrients and components, 12 volunteers were recruited to participate in the feeding trial. All volunteers were healthy participants, free of chronic carbohydrate metabolism disease, who consented to the approved protocol of the research. The study began on February 19th, 2007 and ended on April 20, 2007. Participants were financially compensated for each completed day of the panel. Questionnaires were provided to the volunteers for age, gender, medical history, and normal daily carbohydrate consumption information. Anthropometric data measured were for weight and height for subjects at the commencement of the study. BMI was calculated from the measured data.

Samples were warmed for one minute in a microwave oven on the day they were needed for each volunteer as done in research by Singh et al. (2006). Three hundred μL of blood was collected on the first day of the study to be analyzed for fasting insulin levels to remove participants with hyperinsulemia. Volunteers began between 7 a.m. and 9 a.m. after fasting for at least 7 hours. A fasting blood glucose level was taken using Therasense ® Freestyle glucometers and followed by participants consuming their samples in 15 to 20 minutes. Research by Wallace et al. (2006) found no difference between venous and capillary blood samples using continuous glucose monitoring systems for determining the blood glucose response to food. The time when participants completed each sample was recorded and blood glucose levels were taken in thirty minute increments thereafter, stopping at two hours.

Glycemic Index Analysis

Each subject consumed each food sample on two separate days for improved statistical accuracy. The incremental areas under the curve, excluding the area beneath the fasting level, were calculated by weighing geometrically (Wolever et al., 1991). Glucose responses were graphed for each individual using Microsoft Excel and printed on Staples 8.5" x 11" acid-free paper. The area under the curve, not considering less than zero, was cut and weighed on Denver Instrument and Sartorius Analytical balances. The GI was calculated by expressing the glucose response area for the test foods as a percentage of the mean response area of the reference food (glucose drink) taken by the same subjects (Wolever et al., 1991, 1994).

Statistical Analysis

A t-test (LSD) using SAS 9.1 compared glycemic index means for each treatment to calculate statistical differences (Zakir, 2005). A program considering the interaction of treatment (baking, steaming, and raw) and part (skin vs. flesh) was used to analyze their effect on the glycemic index.

Results

Anthropometric and demographic description of subjects

Participants completed questionnaires regarding age, date of birth, weight, height, carbohydrate metabolism deficiencies, smoking habits, carbohydrate source, physical activity, and medical history. Twelve volunteers began and ended the study; seven female and five male. Volunteers were not screened for high or low carbohydrate intake or source. All were non-diabetic and considered normal for the purposes of the study. The average age was 32 ± 12. Ages ranged from twenty-two to sixty-three. Body mass index (BMI) was calculated from the weights and heights. The mean BMI among participants was 24.63 ± 3.62, which is considered a healthy BMI (US Dietary Guidelines, 2005). The average weight among volunteers was 161.14 ± 26.87 lb (73.1 ± 12.2 kg) and height was 5'7" ± 3.74" (1.701 ± .095 M). No participants reported that they currently smoked.

Proximate Analysis of Sweet Potatoes

Variability was noted in proximate analysis results. Calculations were on dry matter basis due to dehydration requirements for assay procedures. Some crucible weights with Celite™ and dried residue were lower than the weights of crucibles and Celite™ alone. These discrepancies caused negative or inflated values when calculating ash, protein, total

dietary fiber, and lipid resulting in certain values that could not be used. The positive residue values were used to replace those that were negative within the same treatment in order to maintain accurate values of lipid, protein, and ash. Table 3.1 displays proximate analysis values for each cooking method.

Research by Lanza et al. (1987) found that sweet potatoes have an average dietary fiber content of 2.4 g/ 100g fresh weight using the neutral detergent fiber plus water soluble fraction and Southgate procedure for extracting the fiber. The value was average from compiled literature sources. The data presented in Table 3.1 shows values at dry weights that are slightly higher in fiber content than the average sweet potato in the study by Lanza (1987).

Table 3.1 Dry matter percentage of sweet potato components for proximate analysis.

	% Ash[1]	%TDF[1]	% Fat[1]	% Protein[1]	% Dry Matter[2]	25 g Carbohydrate[3]
Raw Skin	6.29	23.62	1.16	9.5	16.8	264.610 g
Raw Flesh	2.16	27.79	0.27	6.4	20.1	189.155 g
Steamed Skin	3.69	49.78	0.23	7.8	13.3	445.299 g
Steamed Flesh		16.64	0.89	7.9	21.8	147.831 g
Baked Skin		43.95	1.22	7.9	33.8	146.441 g
Baked Flesh		26.09	0.41	7.3	12.8	261.178 g
Dehydrated Skin	3.47	43.82	1.95	6.8	75.3	63.612 g
Dehydrated Flesh		13.06	2.00	7.4	59.7	52.724 g

1= Dry Matter Basis, 2= As Served Basis, 3= Quantity of sweet potato containing 25 g of carbohydrate

Glucose Response

Blood samples were drawn and analyzed thirty minutes increments after participants completed consuming samples. Figure 3.1 shows the mean glucose response levels and standard deviations. Dehydrated skin was not consumed by all subjects due to severe gastrointestinal difficulty by three volunteers. Symptoms included cramping, nausea, vomiting, and constipation. These symptoms were reported to only last for a maximum of six hours. An analysis of interaction between cooking method (steamed, baked, raw) and part (skin vs. flesh) using a comparison of means revealed that GI values for part were dependent on the method of cooking and vise versa (P= 0.001). Furthermore, there were significant differences in glycemic indices based on cooking method (P= 0.001) and part (P=0.001).

Table 3.2 shows the calculated glycemic index for each sample. Glucose was given the value of 100. The Type III Sum of Squares analysis of subjects indicated no differences in glycemic indices calculated from subjects for each treatment (P= 0.573). Significant differences were observed by treatment (P= 0.0001). A t-test on least squares (LSD) of GI means determined that some samples produced values significantly different from others. Microwave flesh produced a medium glycemic index of 66 followed by baked flesh (64), and steamed flesh (63). These values were statistically similar to one another. Dehydrated flesh had a glycemic index of 40, which was grouped with baked skin (32), raw flesh (28), and steamed skin (25). The final group consisted of baked (32), steamed (25), and raw skin (19) and raw flesh (28).

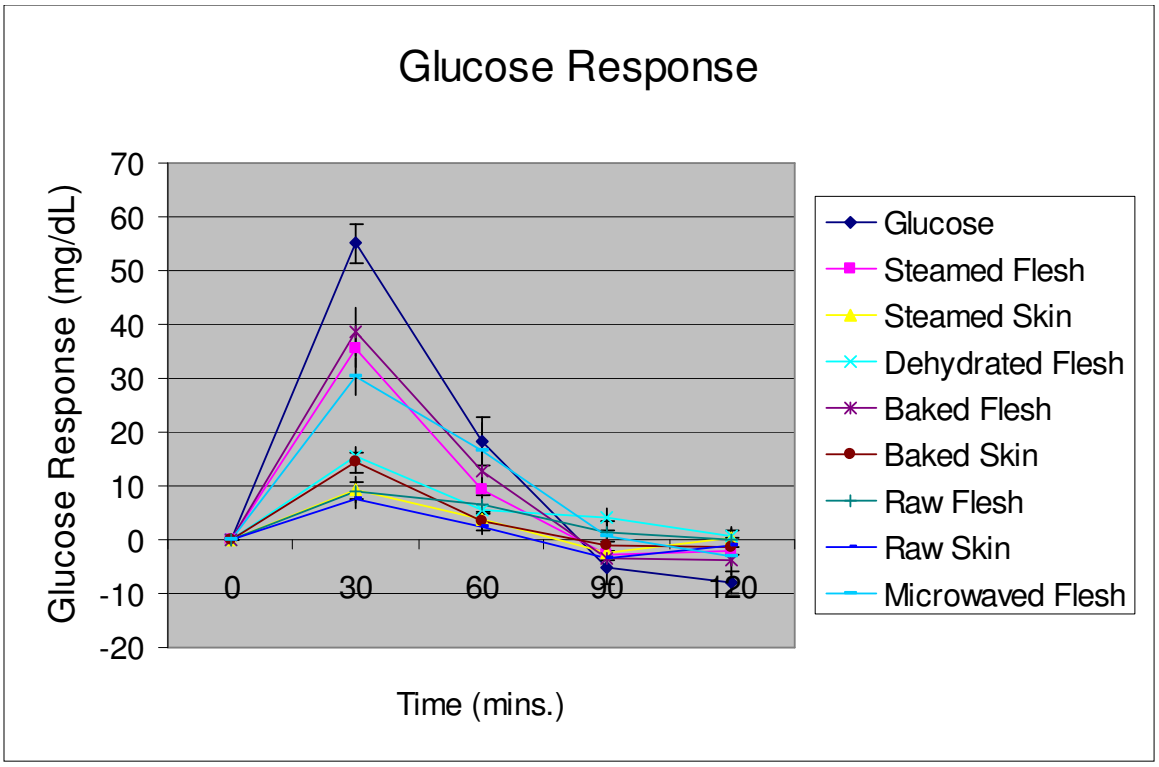

Figure 3.1 Mean Glucose Response from Participants consuming cooked Beauregard Sweet Potato Samples

Table 3.2 Calculated Glycemic Indices of Cooked Beauregard Sweet Potato

Sample	GI	SEM
Steamed Flesh	63 [A]	8.4
Steamed Skin	25 [B,C]	6.51
Dehydrated Flesh	40 [B]	8.24
Baked Flesh	64 [A]	10.11
Baked Skin	32 [B,C]	5.95
Raw Flesh	28 [B,C]	7.26
Raw Skin	19 [C]	6.29
Microwaved Flesh	66 [A]	13.29

Discussion

Lipid and protein levels remained fairly constant regardless of cooking methods. Total dietary fiber values were more variable across sample preparation. Total dietary fiber was higher in the skin of cooked samples; however raw sweet potato skin had levels similar to that of raw flesh. The amount of fiber content quantified by Bahado- Singh (2006) as 3 to 14 grams (SE 0.02) per 100 gram sample of sweet potato fresh weight. The amount of total dietary fiber is important because of its influence on glucose absorption, post-prandial glucose levels, and GI.

High levels of total dietary fiber can cause low GI levels, as observed in the study. Leaching of sugars can occur during heat processing concentrating the components the fiber components of the skin (Reddy & Sistrunk, 1980). The amount of lipid found in sweet

potatoes has been determined to be 0.30 grams per 100 gram raw sample (SE 0.02) (Bahado-Singh, 2006), a value specific for Beauregard sweet potatoes. The proximate analysis was comparable to Bahado-Singh's data, where higher concentrations of lipid compounds were extracted in the sweet potato skin.

Chang and Morris (1990) found no observed statistical differences in protein content between samples or processing treatments of autoclaving and microwaving. Similarly, this study's proximate analysis revealed protein concentrations that remained relatively constant (range= 7.406% to 9.531%). Bahado- Singh (2006) determined that protein levels in sweet potatoes was 2.15 g per 100-g dry matter (SE 0.05), calculated by multiplying nitrogen content by 6.25.

Glycemic indices of zero were not omitted from the study nor were values greater than 100; negative glycemic response was given a GI of zero. Individuals who had glycemic responses that created GI values equal to zero indicate that breakdown of sweet potato samples could have taken longer than two hours to elicit a glucose response. GI values greater than 100 can mean that the rate at which an individual absorbed glucose from the sweet potato was faster than their response to the glucose drink standard.

The GI values for raw sweet potato skin and flesh are different from the first study, in which the glycemic index for raw sweet potato flesh was calculated at 49 ± 12.11 and skin at 26 ± 6.70. This second study produced values of 28 ± 7.26 and 19 ± 6.29 respectively. The variability in glycemic index could be the result of storage and preparation method differences between the two studies. Samples were frozen and allowed to thaw or be slightly warmed in microwaves prior to eating in the first study. Minimal cooking could have taken

place, or freezing may have disrupted the cell structure. The protocol in this study required that raw samples be kept in refrigerators and let chill and re-warm without being subjected to any heat.

A t-test comparing glycemic index means from raw sweet potato skin showed statistical differences between this and first study from 2005 in reference to the glycemic index of skin. Conversely, sweet potato flesh glycemic indices were not statistically different. The variability shown in sweet potato skin glycemic indices may be due to the microwaving done to thaw frozen samples in the first study. The temperature in the microwave can evenly warm the external areas of the sweet potato in one minute.

The glycemic index calculated by Zakir (2005) for dehydrated Beauregard sweet potato was 30, which is not too different from the value determined in this study (40). Both values are low according to U.S. Dietary Guidelines (2005). The reproducible low values are beneficial for sweet potato consumers. The low GI will allow for a slow rate of glucose absorption, thus maintaining low blood glucose levels. Zakir's research considered the presence of α amylase inhibitor protein in sweet potato skin that could cause low glycemic index values. The quantity of total dietary fiber may also have an effect on the glycemic index produced from Beauregard sweet potatoes.

Summary & Suggestions for Future Work

Beauregard sweet potato samples produced glycemic indices that remained low to medium despite cooking. This may prove beneficial for diabetic patients who consume sweet potatoes. Knowledge on the glycemic index can help diabetic patients predict their

daily diets to control blood glucose levels. The total dietary fiber content of sweet potatoes is also enough to affect the glycemic index elicited by these roots.

Further research quantifying the amount of maltodextrins produced from starch after various cooking procedures using HPLC can give insight to starch breakdown by cooking. The concentrations of the resulting sugars can help explain the readily absorbed available carbohydrate and how the related to the glucose response and glycemic index.

Literature Cited

AOAC, (1990) Official Methods of Analysis. The Association of Official Analytical Chemists. 15th Edition, Arlington, USA.

Bahado-Singh (2006) Food processing methods influence the glycaemic indices of some commonly eaten West Indian carbohydrate-rich foods. Brit J of Nutrition. 9: 476-81.

Brand-Miller J, Pang E, Bramall L. (1992) Rice: a high or low glycemic index food? Proc. Nutr Soc Austr 17:226.

Chang MC, Morris WC (1990) Effect of heat treatments on chemical analysis of dietary fiber. J Food Science. 55:1647-1675.

Collins WW, Walter Jr WM (1985) Fresh roots for human consumption. Sweet potato products: a natural resource for the tropics. Bouwkamp JC ed. Florida CRC Press; 205-218.

Kohyama K, Nishinari K (1991) Effect of soluble sugars on gelatinization and retrogradation of sweet potato starch. J A Food Chem. 39:1406-1410.

Reddy N, Sistrunk W (1980) Effect of Cultivar, Size, Storage, and cooking method on carbohydrates and some nutrients of sweet potatoes Journal of Food Science 45; 3: 682-684.

Lanza E, Jones D, Block G, Kessler L (1987) Dietary fiber intake in the US population. The American Journal of Clinical Nutrition. 46;5:790-797.

Lewthwaite SL, Sutton KH, Triggs CM (1997) Free sugar composition of sweet potato cultivars after storage. New Zealand J of Crop Hori Sci 25:33-41.

Picha, DH. (1986) Sugar content of baked sweet potatoes from different cultivars and length of stage. J Food Sci. 51:845-845,948.

Ramdath DD, Isaacs RLC, Teelucksing S & Wolever TMS (2004) Glycaemic index of selected staples commonly eaten in the Caribbean and the effects of boiling v. crushing. Br J Nutr 91: 971-977.

Takahata Y; Noda T, Nagata T, (1992) Varietal diversity of free sugar composition in storage root of sweet potato. Jpn J Breed 42:515-521

Thorne MJ, Thompson LU, Jenkins DJA. (1983) Factors affecting starch digestibility and the glycemic response with special references to legumes. Am J Clinical Nutrition.; 38:481-8.

Truong, VD (1986) Processing of Sweet Potato for food and industrial uses in sweet potato Research State-of-the-Art; Philippine Council for Agriculture and Resources Research: Los Banos, Philippines.

Wallace AJ, Willis JA, Monro JA, Frampton CM, Hedderley DI, Scott RS (2006) No difference between venous and capillary blood sampling and the minimed continuous glucose monitoring system for determining the blood glucose response to food. Nutrition Research. 26:403-408.

Wolever TMS, Jenkins DJA, Jenkins AL, Josse RG. (1991) The glycemic index: methodology and clinical implications. Am J Clin Nutr 54:846-54.

Wolever TMS, Katzman-Relle L, Jenkins AL, Vuksan V, Josee RG and Jankins DJA (1994): Glycaemic index of 102 complex carbohydrate foods in patients with diabetes. Nutr Res 14: 651-669.

Zakir S, Sarwar M, Allen JC, Butt MS, Allen H. (2005) Effect of sweet potato on insulin efficiency of normal and diabetic subjects in Pakistan. Eur J Sci Res 10(1):87-97.

Appendix Table 3.1 Mean Glucose Responses for Volunteers in Trials 1 from cooked Beauregard sweet potatoes.

Trial I

Parameters	Glucose Control	Steamed Flesh	Steamed Skin	Dehydrated Flesh	Baked Flesh	Baked Skin	Raw Flesh	Raw Skin	Microwaved Flesh
Blood Glucose Fasting									
Mean	92.75	90.67	92.67	91.83	95.75	91.58	91.42	94.25	94.42
SD	10.75	8.82	8.52	9.13	8.85	10.36	8.49	5.46	6.86
30 Minutes									
Mean	146.17	126.42	104.42	107.33	126.00	109.92	101.92	101.83	119.25
SD	22.16	22.98	16.10	12.12	26.07	17.20	11.29	8.95	21.66
One Hour									
Mean	108.67	98.75	97.92	99.08	106.42	103.67	96.75	94.50	107.17
SD	19.08	20.09	8.46	8.86	22.90	21.51	7.93	8.72	21.30
90 Minutes									
Mean	86.67	87.00	89.42	96.67	90.00	90.58	93.92	88.83	96.00
SD	4.94	9.03	10.22	10.25	13.19	15.83	7.77	5.11	6.22
Two Hours									
Mean	85.67	90.08	92.58	95.08	90.58	88.58	91.67	93.83	92.58
SD	5.84	8.47	10.68	12.49	8.67	10.00	7.35	10.11	7.15

Appendix Table 3.2 Mean Glucose Responses for Volunteers in Trials 2 from cooked Beauregard sweet potatoes.

Trial II

Parameters	Glucose Control	Steamed Flesh	Steamed Skin	Dehydrated Flesh	Baked Flesh	Baked Skin	Raw Flesh	Raw Skin	Microwaved Flesh
Blood Glucose Fasting									
Mean	88.83	91.25	91.92	92.33	92.58	92.42	91.92	91.83	96.75
SD	9.94	10.05	11.18	6.93	8.73	11.33	6.95	7.32	4.99
30 Minutes									
Mean	145.58	126.83	98.67	108.00	128.08	110.83	102.83	100.08	118.08
SD	22.61	18.88	14.34	9.50	22.77	25.38	10.64	9.97	19.31
One Hour									
Mean	109.33	101.67	94.25	95.83	102.17	93.25	99.25	96.67	111.67
SD	22.24	25.00	8.69	9.64	16.39	6.78	10.84	9.16	33.94
90 Minutes									
Mean	84.83	89.17	90.08	96.00	92.75	90.33	95.92	88.17	97.33
SD	10.40	11.41	5.37	8.87	9.31	7.08	10.45	12.33	18.08
Two Hours									
Mean	80.08	87.75	92.92	90.75	90.58	92.75	91.50	91.75	93.42
SD	8.26	9.53	11.66	6.82	9.42	10.30	8.02	4.94	9.88

Appendix Table 3.3 2007 Glycemic Indices by Part

The GLM Procedure

Dependent Variable: GIMean

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	18	3.47871936	0.19326219	5.19	<.0001
Error	77	2.86783070	0.03724455		
Corrected Total	95	6.34655006			

R-Square	Coeff Var	Root MSE	GIMean Mean
0.548128	45.99107	0.192988	0.419622

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Subject	11	0.35653060	0.03241187	0.87	0.5724
trt	4	1.60880114	0.40220028	10.80	<.0001
Part	1	0.75703420	0.75703420	20.33	<.0001
trt*Part	2	0.75635343	0.37817671	10.15	0.0001

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Subject	11	0.35653060	0.03241187	0.87	0.5724
trt	4	1.25772981	0.31443245	8.44	<.0001
Part	1	0.75703420	0.75703420	20.33	<.0001
trt*Part	2	0.75635343	0.37817671	10.15	0.0001

Appendix Table 3.4 Subject and treatment statistical differences

The GLM Procedure

Dependent Variable: GIMean

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	18	3.47871936	0.19326219	5.19	<.0001
Error	77	2.86783070	0.03724455		
Corrected Total	95	6.34655006			

R-Square	Coeff Var	Root MSE	GIMean Mean
0.548128	45.99107	0.192988	0.419622

Source	DF	Type I SS	Mean Square	F Value	Pr > F
Subject	11	0.35653060	0.03241187	0.87	0.5724
trt2	7	3.12218876	0.44602697	11.98	<.0001

Source	DF	Type III SS	Mean Square	F Value	Pr > F
Subject	11	0.35653060	0.03241187	0.87	0.5724
trt2	7	3.12218876	0.44602697	11.98	<.0001

Appendix Table 3.5 Duncan's grouping of Glycemic Index means

The GLM Procedure

Duncan's Multiple Range Test for GIMean

NOTE: This test controls the Type I comparisonwise error rate, not the experimentwise error rate.

Alpha	0.05
Error Degrees of Freedom	77
Error Mean Square	0.037245

Number of Means	2	3	4	5	6	7	8
Critical Range	.1569	.1651	.1705	.1745	.1775	.1800	.1820

Means with the same letter are not significantly different.

Duncan Grouping	Mean	N	trt2
A	0.65766	12	Microwa_Flesh
A			
A	0.64073	12	Baked_Flesh
A			
A	0.62558	12	Steamed_Flesh
B	0.39855	12	Dehydra_Flesh
B			
C B	0.31585	12	Baked_Skin
C B			
C B	0.27513	12	Raw_Flesh
C B			
C B	0.25179	12	Steamed_Skin
C			
C	0.19169	12	Raw_Skin