

University of the Incarnate Word

The Athenaeum

Theses & Dissertations

5-2017

Effect of Postprandial Walking on the Glycemic Effect of a Meal: Type I Diabetes

Samantha L. Hinojosa

University of the Incarnate Word, samanthaloren16@gmail.com

Follow this and additional works at: https://athenaeum.uiw.edu/uiw_etds



Part of the [Endocrinology, Diabetes, and Metabolism Commons](#), and the [Human and Clinical Nutrition Commons](#)

Recommended Citation

Hinojosa, Samantha L., "Effect of Postprandial Walking on the Glycemic Effect of a Meal: Type I Diabetes" (2017). *Theses & Dissertations*. 4.

https://athenaeum.uiw.edu/uiw_etds/4

This Thesis is brought to you for free and open access by The Athenaeum. It has been accepted for inclusion in Theses & Dissertations by an authorized administrator of The Athenaeum. For more information, please contact athenaeum@uiwtx.edu.

EFFECT OF POSTPRANDIAL WALKING ON THE GLYCEMIC EFFECT OF A MEAL:
TYPE 1 DIABETES

by

SAMANTHA L HINOJOSA, BS, MS

A THESIS

Presented to the Faculty of the University of the Incarnate Word
in partial fulfillment of the requirements for
the degree of

MASTER OF SCIENCE

UNIVERSITY OF THE INCARNATE WORD

May 2017

Copyright by
Samantha L Hinojosa
2016

ACKNOWLEDGEMENTS

This thesis would not have been successful without the help of the Nutrition and Nursing faculty, Athletics Department, and the School of Pharmacy at the University of the Incarnate Word. I would like to thank the primary investigator and my thesis advisor, Dr. Cynthia Heiss, for all the guidance and understanding through this long process. You have taught me so much during our time working together and have given me such a great opportunity; I am grateful to have you as a professor, advisor, and friend. I would like to thank the remainder of the thesis committee, Dr. Joseph Bonilla and Dr. Maureen Rauschhuber, for being there to review the proposal and defense of the thesis. I would like to thank Dr. Beth Senne-Duff and Dr. Neeta Singh for their additional support of the thesis. I would like to thank Nancy Nguyen for participating in our study as a registered nurse. I would like to thank the undergraduate student assistants, Megan Baker and Aaron Luevano, for helping with the preparation and organization of data collection, and researching articles. I would also like to thank Steve Rodriguez for his IT skills that helped format the final thesis.

I would like to dedicate this work to all the individuals in the world living with type 1 diabetes; I have experienced what you all have experienced and because of that I want to dedicate my career to working hard to make diabetes management as easy as it possibly can. We can and will beat diabetes and live long, healthy lives. I would also like to dedicate this work to my mother, Wenoa Genice. Thank you for being the overprotective and strict parent that this type 1 diabetic needed you to be; without you, I wouldn't be in good health and still pursuing my passion to help other individuals with diabetes. Thank you for also being my personal cheerleader; supporting me through the change of my career path and continuing to be proud of

me. I owe so much of my educational success to you and there are not enough words that can express my gratitude for that. You're a great parent and this book is proof of it. I love you bunches mom.

Samantha L Hinojosa, BS, MS

EFFECT OF POSTPRANDIAL WALKING ON THE GLYCEMIC EFFECT OF A MEAL: TYPE 1 DIABETES

Samantha L Hinojosa, BS, MS

University of the Incarnate Word, 2017

This study was conducted to determine if 15 minutes of postprandial light walking has an effect on the glycemic response to a Boost[®] beverage in individuals with type 1 diabetes. Seven participants, 22.3 ± 4.3 yrs, with type 1 diabetes completed the two days of data collection. On day 1, participants measured baseline fasting blood glucose (BG) with a glucometer, consumed a Boost[®] beverage, and sat quietly, repeating BG measurements 15, 30, 60, 90, and 120 minute. On day 2, participants repeated the protocol, but walked 15 minutes at a light pace (50-60% max HR) immediately after beverage consumption. The difference between peak and baseline BG was significantly lower on the walking day compared to the sedentary day (6.4 ± 1.2 vs 4.0 ± 2.4 , respectively, $P < .05$) as was the iAUC (468.6 ± 94.5 mmol/L/120min vs 241.1 ± 155.8 mmol/L/120min, $P < .05$). In conclusion, light walking for 15 minutes postprandially can blunt the spike in BG and overall glycemic response to a breakfast beverage in young adults with type 1 diabetes.

TABLE OF CONTENTS

| | |
|--|------|
| LIST OF TABLES | viii |
| LIST OF FIGURES | ix |
| INTRODUCTION | 1 |
| Statement of the Problem..... | 1 |
| Purpose of the Study | 3 |
| Research Questions..... | 3 |
| Hypotheses | 4 |
| Significance of the Study | 4 |
| LITERATURE REVIEW | 5 |
| Type 1 Diabetes: An Overview..... | 5 |
| Management of Type 1 Diabetes | 6 |
| Diabetes Complications | 8 |
| Glycemic Index and Response..... | 11 |
| Physical Activity and Glycemic Control | 13 |
| Factors Influencing Physical Activity in Individuals with Diabetes | 19 |
| Glycemic Control and The Development of Diabetes' Complications | 20 |
| The Expenses of Type 1 Diabetes..... | 24 |
| Summary | 26 |
| RESEARCH METHODOLOGY | 27 |
| Participants..... | 27 |

Table of Contents—Continued

RESEARCH METHODOLOGY—continued

| | |
|---|----|
| Blood Glucose Measurements | 28 |
| Data Collection | 28 |
| RESULTS | 30 |
| Participants Characteristics | 30 |
| Blood Glucose Indicators With and Without Walking | 30 |
| Tables and Figures | 31 |
| DISCUSSION AND CONCLUSION | 37 |
| REFERENCES | 42 |
| APPENDICES | 48 |

LIST OF TABLES

| | |
|---|----|
| 1. Participant Characteristics..... | 31 |
| 2. Blood Glucose Indicators of Sedentary and Walking Days | 32 |

LIST OF FIGURES

| | |
|--|----|
| 1. Difference Between Baseline and Peak Blood Glucose (mmol/L) for Sedentary and Walking Days..... | 32 |
| 2. iAUC Sedentary VS Walking For Each Participant..... | 33 |
| 3. Comparison of Peak-Baseline BG on Sedentary and Walking Days..... | 34 |
| 4. Comparison of iAUC on Sedentary and Walking Days..... | 34 |
| 5. Glucose Curves for Each Participant..... | 35 |

Introduction

Statement of the Problem

Type 1 diabetes manifests when there is no longer a production of insulin, which is a hormone secreted by the beta cells of the pancreas that normally acts to lower blood glucose (BG) levels, resulting in a hyperglycemic state. Another type of diabetes, type 2, occurs mainly in overweight and obese individuals; in this case, insulin is still produced by the pancreas, but is ineffective in maintaining normal BG. Only about 5-10% of the diabetic population are diagnosed with type 1 diabetes and live with it for majority of their lifetime, with some patients developing it at a very young age.¹ In 2012, 29.1 million Americans had diabetes, with 1.25 million of those with type 1 diabetes.² Forty thousand people are diagnosed with type 1 diabetes every year and 5 million people are expected to develop type 1 diabetes by 2050, in the United States alone.³

Type 1 diabetes is the more severe form of diabetes because the complete lack of insulin production by the pancreas can result in very high BG levels; no cure is available at this time. However, BG levels can be managed with insulin injections and a controlled carbohydrate intake. BG levels can become uncontrolled if insulin dosing and carbohydrate intake are not coordinated with each other, resulting in hypoglycemia or hyperglycemia.¹ Currently, less than one-third of individuals with type 1 diabetes are considered to be in good glycemic control.³ Hyperglycemia, if not treated promptly or left untreated for a long period of time, can result in complications including retinopathy, nephropathy, neuropathy, and cardiovascular disease (CVD), among others.¹

Physical activity after a meal may be a tool in type 1 diabetes management to decrease postprandial BG and avoid hyperglycemia. Because exercising muscle does not require insulin

for glucose uptake, postprandial physical activity may blunt the spike in BG and overall glycemic response to carbohydrate, potentially resulting in better BG control over time, with the possibility of lessening the risk for the long term complications of diabetes and decreasing insulin dosages needed.⁴ Previous research has shown that 30 minutes of light walking after consumption of a candy bar blunted the glycemic response to a candy bar in healthy participants.⁵ Other studies with healthy participants and those at high risk for type 2 diabetes have shown similar effects of physical activity on the glycemic response to food.⁵⁻¹⁰

Few studies have been conducted examining the effect of postprandial physical activity on the glycemic response in individuals with type 1 diabetes. Some of the studies were conducted between 1982 and 1994, before the insulin pump and intensive insulin therapy were commonly used. Intensive insulin therapy involves administering a bolus of fast acting insulin at each meal, in accordance with the carbohydrate content of the meal. This results in more precise glycemic control and greater meal flexibility than previous insulin regimens that involved 2-3 insulin injections a day with a mixture of insulin types that peaked at specific times when meal with controlled carbohydrate content had to be consumed. Studies have found a beneficial effect of postprandial physical activity on glycemic control was found in participants with type 1 diabetes using varying insulin, meal, and physical activity regimens.¹¹⁻¹³ In both of the 1982 studies, participants were hospitalized and administered their usual insulin type and dosage, either intravenously or by artificial pancreas for 48 hours before the study, then commenced activity for 45 minutes on an exercise bike for 30 minutes after the beginning of breakfast.^{11,13} In 1994, Rasmussen and coworkers found favorable effects of 30 minutes of cycling that commenced 15 minutes after a meal was consumed in participants with provided insulin via an artificial pancreas for 2 hours before the meal followed by continuous insulin infusion for the

remainder of data collection.¹² Years later, in 2002, the researchers found a similar beneficial effect of postprandial physical activity in patients using intensive insulin therapy; these participants were hospitalized for the study, but given long acting insulin the night before data collection, then given a bolus of regular insulin 30 minutes before meal consumption, which is not typical current protocol for intensive insulin therapy. In addition, the participants waited 30 minutes after meal consumption to walk for 30 minutes.¹⁴

To date, no studies have been conducted to determine the effect of postprandial physical activity on the glycemic effect of a meal in free-living participants using intensive insulin therapy or the pump following their usual insulin routine. In addition, a shorter bout of physical activity may be more realistic to implement in daily life. Even though postprandial physical activity has been found to be beneficial, there is also a potential risk of hypoglycemia or hyperglycemia in individuals with type 1 diabetes when participating in physical activity.¹⁵⁻¹⁷ Thus, reducing the physical activity duration to 15 minutes would likely reduce the risk of hypoglycemia and be more realistic or convenient as part of the treatment regimen.

Purpose of Study

The purpose of this study is to determine if a 15 minute interval of light physical activity, consisting of walking immediately after consumption, would attenuate the glycemic response to a breakfast beverage in free-living individuals with type 1 diabetes following their usual insulin protocol.

Research Questions

1. Will 15 minutes of light physical activity immediately after consuming a breakfast beverage result in a lower spike in BG compared to being sedentary after consumption in individuals with type 1 diabetes?

2. Will 15 minutes of light physical activity immediately after consuming a breakfast beverage result in a smaller glycemic response, as determined by the incremental glucose area under the curve (iAUC), than being sedentary after consuming the beverage?

Hypotheses

1. Fifteen minutes of light physical activity immediately after consuming a breakfast beverage will result in a lower spike in BG compared to being sedentary in individuals with type 1 diabetes.
2. Fifteen minutes of light physical activity immediately after consuming a breakfast beverage will result in a smaller glycemic response indicated by the incremental glucose area under the curve (iAUC), compared to being sedentary after consumption.

Significance of Study

This study will shed light on the potential benefit of timing of physical activity to enhance glycemic control in individuals with type 1 diabetes. Specifically, this study will examine the effect of postprandial physical activity on the glycemic response to a breakfast beverage as BG increases most dramatically after carbohydrate consumption. Better glycemic control will lead to better management of diabetes and potentially better overall health by possibility of decreasing the risk for secondary complications forming. Achieving better glycemic control with light postprandial walking would also lead to a decrease in the amount of insulin needed to achieve glycemic control and therefore lower the cost of supplies and medications. This study may promote further research to be conducted on this topic with participants with type 1 diabetes to confirm the duration and types of activity that have the most beneficial effects while minimizing hypoglycemia or hyperglycemia. In the end, postprandial physical activity could be included a part of routine diabetes management.

Literature Review

Type 1 Diabetes: An Overview

Type 1 diabetes is the most severe form of diabetes and represents 5-10% of all diabetes diagnoses, occurring when there is no longer a production of the hormone insulin due to the destruction of the beta cells of the Islets of Langerhans in the pancreas. This can be caused by autoimmune destruction of beta islet cells or can be idiopathic, where the cause is unknown. A contributing factor can be genetic predisposition from a family history of type 1 diabetes along with a trigger by an environmental exposure. Insulin is required for the uptake of glucose by the GLUT 4 transporter in muscle and adipose cells. Insulin's normal function is to facilitate glucose entry into cells where it is involved in metabolic pathways, such as glycolysis, and ultimately provides substrate for the production of ATP via aerobic metabolism. Insulin also lowers BG by stimulating the storage of glucose as glycogen in the liver and muscle, and inhibiting glycogen breakdown (glycogenolysis). In addition to glucose metabolism, insulin influences fat metabolism by promoting fat storage and inhibiting fat breakdown, and promotes amino acid entry into cells, enhancing muscle protein synthesis. Thus, insulin, which promotes glycogen formation and fat and protein synthesis is considered an anabolic hormone. When insulin is absent, as in type 1 diabetes, glucose levels in the blood rise because glucose does not enter muscle and adipose cells. In addition, anabolic processes are not inhibited, resulting in a catabolic state where the release of glucose from the liver glycogen stores, and gluconeogenesis from the conversion of some amino acids from catabolized protein and the glycerol portion of catabolized triglycerides further contribute to hyperglycemia.¹ GLUT 4 transporters, however, are not dependent on insulin for the uptake of glucose while skeletal muscle is contracting during physical activity.⁴

The symptoms that present with type 1 diabetes, in addition to hyperglycemia, are polyuria, polydipsia, polyphagia, dehydration, electrolyte disturbance, fatigue, blurry vision, slow healing process, weight loss, and sometimes ketoacidosis.¹ Diabetic ketoacidosis (DKA) is a life-threatening condition that presents with symptoms of dyspnea, abdominal pain, and vomiting.¹⁸ The lab values that determine a type 1 diabetes diagnosis include a fasting plasma glucose of 126 mg/dL on two separate occasions, a casual, non-fasting BG of 200 mg/dL with symptoms present, or a glycated hemoglobin (HbA1c) value 6.5% or greater. A simple oral glucose tolerance test (OGTT) can also be used to diagnose type 1 diabetes. The procedure includes an 8 hour fast previous to the ingestion of a glucose solution that is 1.75 g of glucose per kg of ideal body weight, with the maximum dosage of 75 g. The patient will then have their BG tested two hours after ingestion. There is another version of the OGTT that requires a 12 hour fasting period prior to the test and more frequent BG readings taken: fasting then at 30-60 minute intervals for two to five hours. Diabetes is diagnosed in both tests when the BG reading is greater than 200 mg/dL 2 hours after solution ingestion.¹

Management of Type 1 Diabetes

There is currently no cure for type 1 diabetes, but this condition can be managed by controlling BG levels using an insulin regimen, diet, and physical activity under the guidance of an endocrinologist and a certified diabetes educator (CDE), along with other medical professionals. Medical nutrition therapy goals, established by the American Diabetes Association, include maintaining certain lab values, such as BG levels within a normal range established for individuals with diabetes, a normal blood lipid profile, and normal blood pressure level.¹⁹ Achievement of these goals will help prevent or delay the development of the long-term complications of diabetes. A CDE would teach individuals with diabetes how to achieve these

goals with insulin and a diet that meets the individual's nutritional needs, while keeping food items that they enjoy. The goals to indicate glycemic control include:

- HbA1c less than 7% and less than 7.5% in children and adolescents;
- pre-meal BG of 80-130 mg/d;
- peak postprandial BG less than 180 mg/d;
- LDL cholesterol less than 100 mg/dL;
- HDL greater than 40 mg/dL and 50 mg/dL in men and women respectively;
- triglycerides less than 150 mg/dL;
- blood pressure less than 130 over 90 mmHg^{1,20-21}

Insulin dosages and type of insulin medications used are dependent on the individual's lifestyle with factors such as meal times and daily activity level taken into consideration. There are short acting, rapid acting, intermediate acting, long acting, and mixtures of insulin that can be used in insulin regimens. The type and the number of insulin medications used also depends on if the patient is using two to three insulin injections a day coordinated with meal times, which is the conventional regimen, or using the intensive regimen, including multiple daily injections or an insulin pump. With multiple daily injections, individuals with diabetes would use a long acting once a day and rapid acting insulin at meals and for BG corrections. If the individual were using an insulin pump, then rapid acting insulin would be administered in small doses, automatically, throughout the day to mimic the baseline output of insulin that a healthy pancreas would produce, and additional doses, manually, at meals and for corrections of high BG levels. For an individual with type 1 diabetes, the required insulin dosage is 0.5 to 1 unit/kg of body weight, with 50% of the total daily insulin dose contributing to the basal insulin.¹

In terms of diet, individuals with type 1 diabetes are mainly advised to be conscious of their carbohydrate consumption and to do so by using one of two methods. One method is the carbohydrate counting method where each carbohydrate “exchange” is a type and amount of food that contains 15 grams (g) of carbohydrates. Individuals using the carbohydrate counting method know how many units of insulin are needed for a certain amount of carbohydrates (their insulin to carbohydrate ratio). A more extensive exchange method can be used to control calorie, protein, carbohydrate, and fat intake. The individuals using this method would be prescribed a diet with a defined number of exchanges from the starch, fruit, vegetable, milk, meat, and fat exchange groups at each meal or snack. Then a bolus of insulin would be injected in an amount appropriate for the carbohydrate load consumed at each meal or snack, according to their insulin to carbohydrate ratio. If the individual is on a conventional regimen, 2-3 injections of mixtures of insulin types that peak at different times of the day are administered. They are instructed on the number of carbohydrate exchanges they are allowed at each meal and snack, and aware of the times they must consume carbohydrate; carbohydrate consumption is timed to coordinate with insulin peaks and, therefore, timing is important.¹ Individuals with type 1 diabetes also need to be aware of their fat consumption because of the complications, such as CVD and stroke, that are affected by fat intake. They would be advised to consume more omega-3 fatty acids and limit the consumption of saturated fats, trans fats, and cholesterol to decrease inflammation and their risk of secondary complications.^{22,23}

Diabetes Complications

If glycemic control is poor, complications can develop. Hypoglycemia can occur if too much insulin administered or if an inadequate amount of carbohydrates are consumed for the insulin to carbohydrate ratio given. Hypoglycemia can also occur during or after physical activity

because exercising muscle can take up BG without insulin, which means that individuals with type 1 diabetes will need to monitor their BG often to ensure that glycemic control is maintained. Hypoglycemia can be treated with the consumption of a 15 g carbohydrate glucose tablet or snack. If the BG becomes so low that the individual passes out, then an emergency glucagon shot should be administered by someone. Hyperglycemia is a hallmark of type 1 diabetes diagnosis, but it can also develop if insulin dosages are not adequate to cover a carbohydrate load, leading inadequate BG uptake by cells and catabolism of muscle and fat. Diabetic Ketoacidosis (DKA) can develop from hyperglycemia when there is inadequate glucose in the cell to facilitate normal carbohydrate and fat metabolism, resulting in energy production that mimics starvation. In this case, fatty acids are broken down into two carbon molecules called Acetyl CoA, which are then converted to ketones rather than entering the Krebs cycle for aerobic metabolism. Ketones are acidic and, with prolonged hyperglycemia, the concentration of ketones becomes so high that DKA occurs and the body goes into a toxic, acidic state. Muscle protein and fat breakdown will also occur because the anabolic effect of insulin on fat and muscle protein synthesis is lost, and the body goes into a catabolic state. When muscle protein is catabolized, some of the amino acids can and glycerol from catabolized fat can be converted to glucose as described earlier, further elevating BG levels. When high BG levels are extreme, frequent, and not properly corrected in individuals with type 1 diabetes, the risk of complications increase significantly.¹

Long term complications of type 1 diabetes include microvascular and macrovascular complications. Microvascular disease complications occur more frequently in individuals with type 1 diabetes than macrovascular complications, which include coronary heart disease, peripheral vascular disease, cerebrovascular disease, dyslipidemia, and hypertension. However, individuals with type 1 diabetes are still at risk for developing macrovascular diseases.¹

Microvascular diseases include nephropathy, retinopathy, and neuropathy. These conditions develop from experiencing hyperglycemia for a long period of time; excess glucose in the small blood vessels in the kidney, eye, and nerves will attach to and impair proteins in those areas. When proteins in artery walls, like collagen, are damaged by excess glucose, plaques will build up in small and large blood vessels, decreasing blood flow to the tissues they supply, resulting in tissue damage. In cells that do not require insulin for glucose uptake, the levels of glucose inside the cells becomes too high, and glucose enters the cell following the concentration gradient, which also damages cells and impairs their function. The early evidence of nephropathy is microalbuminuria, which is considered to be a value greater than 30 mg/dL; a slow progression into end stage renal disease usually ensues. Retinopathy occurs when there is a loss of retinal vasculature and ischemia with increased occurrence of hemorrhages, leading to increased ocular pressure and glaucoma formation. The lens of the eye will swell from hyperglycemia and cause a temporary blurred vision. Neuropathy occurs in 60-70% of individuals with diabetes and is characterized by peripheral nerve damage to feet and hands as well as autonomic nerve damage resulting in impairment in the impulses to the heart, and can cause impotence and gastroparesis.¹ Gastroparesis is characterized by delayed gastric emptying caused by the damage of the vagus nerve, which normally allows for the proper movement of food through the digestive tract. This condition will make managing BG levels more difficult since food is not able to pass through the digestive tract normally, altering absorption rate from typical rates. When the food finally enters the small intestine, then the BG will elevate, but the timing of the BG response to carbohydrate will be altered.²⁴ Gangrene can also develop from neuropathy when there is a lack of nerve sensation to the feet resulting in an injury that goes untreated. The poor circulation due to macrovascular complications, including peripheral

vascular disease, limits the delivery of substances needed for proper wound healing. The combination of a wound, poor circulation, and hyperglycemia lead to infection, which sometimes leads to gangrene and the need for amputation.¹

Some of the other complications of diabetes include cognitive impairment and macrovascular complications such as atherosclerosis, which increases the risk for heart attack, stroke, and peripheral vascular disease. Hyperglycemia can also increase the formation of advanced glycation end products (AGEs) in the body, which are produced by the post-translational modification of proteins, lipids, or DNA.^{25,26} AGEs will normally form at a slow, constant rate, from early embryonic development throughout adulthood, accumulating over time. However, AGE formation is accelerated in diabetes due to constant elevated BG levels.^{27,28} AGEs result in the crosslinking of collagen and stiffening of connective tissue, including tissue in the arteries. This will cause irreversible damage to tissues and therefore activate macrophages, monocytes, cardiac fibroblasts, and vascular smooth muscle cells. Not only will hyperglycemia affect the development of atherosclerosis, but it will also increase the risk of the production of reactive oxygen species (ROS) that will then promote the oxidation of DNA and membrane lipid peroxidation.²⁵ Therefore, elevated BG levels stimulate inflammation by promoting the production of free radicals and other pro-inflammatory chemicals. Chronic inflammation is related to the progression of CVD, cancer, and Alzheimer's disease.^{5,29,30} Peppia et al, suggest that AGEs are important pathogenic mediators of almost all of the complications that can form secondary to diabetes, on both macro- and micro- levels.²⁷

Glycemic Index and Response

The glycemic response to food consumption refers to the extent and duration of the increase in BG after eating. The glycemic index (GI) is a dietary tool used to measure the effect a

food has on an individual's BG concentration in comparison to a standard food, such as white bread. The GI is represented as number that reflects the area under the glucose curve (AUC) after consumption of a 50 g load of carbohydrate. Foods that are high in refined and simple carbohydrates that tend to have a high GI, are absorbed quickly and result in BG spikes of greater magnitude than carbohydrate containing foods that have fiber. The processes that influence the changes in BG concentration include the speed of carbohydrate hydrolysis and absorption in the small intestine, insulin secretion, and the tissue uptake of glucose in response to insulin.³¹ It is important to note that the GI of a food is based on a portion that would provide 50 g of carbohydrate, some foods that have a high GI do not affect the BG to the magnitude expected from the GI because a 50 g portion is not typically consumed. For example, carrots have a high GI, but a typical portion provides approximately 5 g of carbohydrate. So some prefer to use the "glycemic load" (GL), which is calculated using the GI of the food multiplied by the amount of carbohydrate per serving. So although the GI of carrots is high, the GL is much lower. In any event, the GI is not normally used in type 1 diabetes management, but it is used to explain the glycemic response to certain food types. In one study of participants with type 1 diabetes who participated in physical activity, the use of the GI was effective towards the prevention of hypoglycemia. When a high GI meal was consumed after 45 minutes of physical activity at 70% VO_2 max in participants with type 1 diabetes, the glycemic response to the meal was similar to the response in the participants that consumed a low GI meal.³² The GI can potentially have beneficial effects in other aspects of diabetes in addition to BG response. Pereira et al, examined the effect of a low GI diet on satiety, insulin resistance, serum triglycerides, C-reactive protein, and blood pressure in comparison to a low fat diet. They found that a low GI diet increased

satiety, and decreased insulin resistance, serum triglycerides, C-reactive protein, and blood pressure.³³

However, foods are not always consumed in isolation and the glycemic response will be reflective of the combination of foods consumed. For example, in one study, three different diets with different combinations of high or low GI foods and varying carbohydrate and calorie contents were consumed by non-diabetic obese and overweight participants. Results indicated that the combination of low GI and low calorie foods with a moderate amount of carbohydrate would be more effective at decreasing body weight and controlling BG levels than consuming a diet with high GI foods.³⁴

Physical Activity and Glycemic Control

As mentioned previously, working, or exercising muscle, does not require insulin for glucose entry; therefore, physical activity influences BG levels, with the magnitude of effect depending on the duration and the intensity of the physical activity. Glucose enters muscle cells by facilitated diffusion via the GLUT4 transport proteins that reside inside muscle cells; glucose can also enter adipose cells through the GLUT4 transporter. GLUT4 receptors cannot facilitate glucose uptake unless they translocate to the surface of the cell. Insulin is the signal for this translocation, but muscle contractions can facilitate this translocation independent of insulin. Once inside the cell, glucose is then converted into energy, via glycolysis, Krebs's Cycle, and the electron transport chain, to help the body maintain energy levels during physical activity. Insulin plus muscle contraction has a synergistic effect that promotes glucose uptake by muscle cells and thus may be particularly effective in mitigating BG spike after carbohydrate consumption in individuals with type 1 diabetes who are using intensive insulin therapy, which includes administering rapid acting insulin with meals. There is also an increased expression of GLUT 4

receptors on the cell surface of the skeletal muscle with physical activity, which improves insulin action, glucose uptake, and glycogen storage in both healthy individuals and individuals with type 1 diabetes. Another reason for increased BG uptake during physical activity is the fact that blood flow is increased during physical activity; BG uptake is directly correlated to blood flow. During physical activity, the blood flow can be increased up to 20 fold compared to at rest. When physical activity is prolonged, the liver is depleted of glycogen and cannot compensate for the glucose being taken in by the skeletal muscle. Therefore, hypoglycemia can occur, especially in individuals with type 1 diabetes. This can be prevented if an individual consumes some form of carbohydrate before the physical activity to provide the arterial blood with glucose so that homeostasis can occur.^{4,35} Exercise physiology is slightly different in individuals with type 1 diabetes than healthy individuals. Because the pancreas does not respond to BG fluctuations, imbalances can occur with the additional hormonal responses involved with physical activity and BG control, such as glucagon, catecholamines, and glucocorticoids. As a result, hypo- or hyperglycemia can occur in individuals with type 1 diabetes in response to physical activity. With moderate aerobic physical activity, the pancreas does not respond to a decrease in BG by limiting insulin secretion, which, in the case of type 1 diabetes, the insulin in the system is from an injection. This can lead to a decrease in BG. With intense anaerobic physical activity, the pancreas of the individual with type 1 diabetes cannot alter insulin secretion to counteract the hormones that increase BG during physical activity, and hyperglycemia could result.³⁶ Hyperglycemia caused by physical activity may be due to low insulin concentrations and the body releasing counter-regulatory hormones including glucagon and epinephrine which stimulate glycogenolysis glycogen in response to increased energy needs; regulating insulin levels during physical activity with a continuous blood glucose monitoring device could prevent this.¹²

As mentioned in the section on the overview of type 1 diabetes, some level of physical activity is a part of the management plan to achieve glycemic control. The American Diabetes Association has included in the 2015 Standards of Medical Care in Diabetes that the time that individuals with diabetes spend sedentary should be broken up by some form of physical activity in order to promote BG control; they suggested less than every 90 minutes.²¹ To support that, in a study with individuals with type 2 diabetes, researchers found that breaking up prolonged sedentary activity with 5 minute, light intensity intervals of standing or walking, for every 30 minutes of sitting, decreased the postprandial BG levels in postmenopausal women.⁶ The National Institutes of Health (NIH) wrote an article on recommendations of exercise as part of the diabetes management plan. Thirty to sixty minutes of moderate to vigorous physical activity most days of the week is recommended; however, it is important for individuals with type 1 diabetes to avoid vigorous physical activity when ketones are present in the blood or urine since hyperglycemia could result.³⁷ The Joslin Diabetes Center advises individuals with diabetes to avoid exercising when BG levels are above 250 mg/dL and ketones are present. If there are no ketones present, the individual with a BG of 300 mg/dL or higher can participate in physical activity if their BG decreases within 5 to 10 minutes. If there is no decrease, they should refrain from exercising.³⁸

The effect of postprandial physical activity on the glycemic response to food has been investigated in healthy individuals mostly, and may potentially help with glycemic control in individuals with type 1 diabetes. In one study, the effect of postprandial walking at a light pace for 30 minutes, compared to being sedentary, on the glycemic response to a Milky Way[®] candy bar in healthy individuals was determined. The investigators found that postprandial walking reduced the BG spike at 30 and 60 minutes after consumption of a high sugar candy bar.⁵ There

was a different approach to intervention of physical activity in another study with elderly participants at risk for glucose intolerance. In this study, there were two types of interventions: three 15-minute intervals of postprandial physical activity bouts vs 45 minutes of sustained walking. The study revealed that the 15-minute interval sessions of walking had a greater effect on lowering BG concentration and, therefore, was the better method of physical activity for glycemic control.⁷ An extensive review was also conducted to describe evidence on strategies to minimize the glycemic effect of food consumption in obese adults at risk or have diabetes. The authors concluded that postprandial activity could be a solution of mitigating the BG spike rather than consuming a low glycemic diet, which was found to be undesirable, unrealistic, and difficult. If postprandial physical activity does prove to be effective at blunting the BG spike postprandially, it could be a low cost and convenient method to prevent the damaging consequences of elevated BG.⁸ Other research has shown that cycling (30-minute 70% maximum heart rate) blunts the BG spike after consumption of corn flakes (1 g of carbohydrate per kg body weight). The physical activity lowered the BG levels with a similar to the effect of hypoglycemic drugs.⁹ Another study examined the effect of postprandial physical activity on BG in women over 50 years old. The participants were sedentary, walked 15 minutes, or 40 minutes after consuming the same meal described in the previous study. The results again showed that postprandial walking after a high carbohydrate meal can decrease the BG spike and the incremental area under the glucose curve (iAUC).¹⁰ The appropriate time allowance of physical activity and intensity still needs to be determined, but from the studies published to date, it seems that between 15-30 minutes of physical activity has the most beneficial effect on glycemic control compared to a longer interval of physical activity.

When comparing physical activity's effect on BG concentration in individuals with type 1 diabetes to healthy individuals, there are several factors to consider. Individuals with type 1 diabetes will not have the insulin production that healthy individuals do after food consumption, and therefore certain metabolic processes will be influenced by the presence or lack of insulin. However, as long as individuals with type 1 diabetes follow their insulin regimen and monitor BG levels often, the glucose concentrations should be near normal. The earliest studies found examined the effect of postprandial physical activity on BG response in participants with type 1 diabetes were published in 1982, 1994, and 2002, before intensive insulin therapy became common. In 1982, Nelson et al examined the BG response to 45 minutes of moderate postprandial physical activity (55% VO_2 max), on a bicycle ergometer, in participants with and without type 1 diabetes. The participants with diabetes used an artificial pancreas during the study, which involves increases or decreases of insulin infusion based on BG level, and consumed a breakfast with an estimated carbohydrate total of 48.4 g. The physical activity commenced 30 minutes after breakfast. In both participants with and without type 1 diabetes, the physical activity resulted in BG levels returning to the fasting level at a rapid rate. The physical activity resulted in a decrease in the amount of insulin needed for glycemic control compared to the amount infused without exercising after eating, providing evidence that postprandial physical activity may indeed reduce insulin dosages needed for glycemic control.¹¹ Another study published in 1982 examined the effect of postprandial physical activity on the glycemic response to breakfast and lunch. The participants were sedentary one day after meal consumption and, another day, performed 45 minutes of moderate physical activity 30 minutes after breakfast on another day. Insulin infusion was used the night before the study days so that fasting BG were similar on both days, then participants administered their usual insulin regimens. Breakfast and

lunch were provided on both days, containing 52.5 and 53.6 g of carbohydrates, respectively. They found an improved glycemic control in response to both breakfast and lunch meals with postprandial physical activity.¹³ In 1994, Rasmussen and coworkers examined the effect of 30 minutes of cycling at 65% VO_2 max commencing 15 minutes after a breakfast composed of 50 g of carbohydrate from white bread. An artificial pancreas was used to achieve normal BG on the test days, then insulin was continuously infused for the remainder of the test periods. The results showed that although the peak BG were similar on both days, the glycemic response (AUC) was decreased by one third.¹² Years later, in 2002, the researchers found a similar beneficial effect of postprandial physical activity in patients using intensive insulin therapy, but these patients were hospitalized for the study, given long acting insulin the night before data collection, then given a bolus of regular insulin 30 minutes before meal consumption, which is not typical current protocol for intensive insulin therapy. In addition, the participants waited 30 minutes after meal consumption to walk for 30 minutes.¹⁴

Other, more recent studies have also found a beneficial effect of postprandial physical activity on glycemic control in those with type 1 diabetes. Manohar et al, compared the glycemic response to a meal with and without 30 minutes of postprandial physical activity in both healthy participants and participants with type 1 diabetes. The results indicated that walking was beneficial for the postprandial blood glycemic control in both those with and without diabetes, but the attenuation of BG with postprandial physical activity was much greater in individuals with diabetes. In this study, the participants with type 1 diabetes were using an artificial pancreas.³⁹ In another study, the association of physical activity (not specifically postprandial) and glycemic control were examined in participants were individuals with type 1 diabetes and their non-diabetic siblings between the ages of 8 and 16. Results indicated that moderate to

vigorous physical activity can result in better glycemic control for individuals with diabetes.¹⁵ To support that conclusion, the study by Campbell, et al. found men with type 1 diabetes who participated in physical activity 45 minutes after consuming a high GI meal, on the day of data collection, experienced a response to postprandial BG concentrations similar to the participants that consumed a low GI diet, meaning that vigorous physical activity can promote better glycemic control.³²

Factors Influencing Physical Activity in Individuals with Diabetes

Health care professionals advise individuals with type 1 diabetes that regular physical activity should be a part of their lifestyle; however, not every patient follows that recommendation. Research has examined the lifestyle habits, including physical activity, of children, adolescents, and young adults with type 1 diabetes. A survey was conducted to gather information on lifestyle habits, such as media consumption and physical activity, and their association with HbA1c. The average weekly time spent exercising among the participants was 5.1 ± 4.5 hours. The results showed that there was not a significant effect of physical activity level on HbA1c, with the HbA1c ranging from 8.6-8.9%. However, the amount of time spent on daily media, how long they had diabetes, and socioeconomic status were related to poorer glycemic control. Even though the data did not find a direct relationship between physical activity and glycemic control, indirectly, media consumption likely resulted in a more sedentary lifestyle, which did affect HbA1c and, therefore, glycemic control.⁴⁰ Level of motivation for physical activity can influence the amount of physical activity accomplished. One research group believes that the reason for the lack of physical activity in patients with diabetes, both type 1 and 2, is the absence of initiative or intention to work out as explained by the social cognitive theory of planned behavior. The social cognitive theory of planned behavior contends that social

influence and its emphasis on external and internal social reinforcement has an affect on behavior outcomes.⁴¹ Other researchers contend fear of hypo- or hyperglycemia occurring during and after physical activity is another reason deterring individuals with type 1 diabetes from participating in physical activity.^{12,15-17} Many factors influence the BG response to physical activity including environment, stress and nutritional status, regimen changes, and physiology. The researchers proposed that an “exercise calculator” device should be developed that would calculate the insulin correction needed for physical activity, either an increase or decrease in insulin, that would prevent prolonged hypoglycemia or hyperglycemia.¹⁶ However, monitoring BG frequently before, during, and after physical activity will have similar effects on avoiding hypo- or hyper-glycemia. BG levels also depend on the when physical activity is performed in relation to the meal consumption. A review article stated that pre-meal physical activity can cause the BG levels to increase immediately following a bout of physical activity and that there was only a slight risk of hypoglycemia, which depended on the level of physical activity in individuals with type 2 diabetes, metabolic syndrome, type 1 diabetes, and insulin resistance. Physical activity performed 30 to 45 minutes postprandial decreased BG in individuals with type 2 diabetes, metabolic syndrome, type 1 diabetes, and healthy individuals. High intensity physical activity at this time did not result in the BG increasing as it did with pre-meal physical activity. However, in the late postprandial period, there was a elevation in BG, but in healthy individuals.⁴²

Glycemic Control and the Development of Diabetes’ Complications

Better glycemic control can be accomplished through proper diet, insulin regimen, and physical activity; with better glycemic control, there can be a decreased risk of developing diabetic complications. As mentioned in an earlier section on diabetes complications, neuropathy

is one of the microvascular complications that can occur with uncontrolled diabetes. The level of physical activity can influence the course of diabetic neuropathy in individuals with type 1 diabetes. In one study, the development of small and large fiber neuropathy in individuals with type 1 diabetes was evaluated to determine if elevated HbA1c and AGEs were contributing factors. The participants were followed for 27 years and the nerve function was evaluated by nerve conduction studies at years 8, 17, and 27. The majority of the participants (81%) were diagnosed with small nerve neuropathy with the remaining of the participants developing large nerve neuropathy. It was found that small nerve fibers might be more sensitive to changes in BG levels; thus, glycemic control is important in order to prevent small nerve damage.⁴³

Two of the other microvascular diabetes complications, retinopathy and nephropathy, were also examined. A study focused on the relationship between retinopathy and nephropathy development in those with type 1 diabetes for six and a half years. The conclusion was that the incidence of the progression of retinopathy was more rapid in participants that had nephropathy compared to participants that did not have nephropathy and vice versa. Therefore, the presence of the microvascular complications increased the risk of each other.⁴⁴ Waden et al, examined the effect of physical activity on the development of nephropathy. A self-evaluation questionnaire was given to determine the level of physical activity for each participant. Even though there was no association found between the progression of renal status and physical activity, the authors concluded that physical activity intensity might have an effect on the initiation and progression of nephropathy.⁴⁵ With persistent hyperglycemia, HbA1c levels will be higher more AGE's will form, contributing to more proteins in the small blood vessels and nerves becoming glycosylated. Postprandial physical activity may help reduce the chances of protein dysfunction, and therefore

decrease the risk of diabetic microvascular complications and other adverse effects such as amputation, blindness, and or kidney failure.^{18,24}

CVD risk is increased in individuals with type 1 diabetes due to several factors, including other complications that come with type 1 diabetes. Hyperglycemia can promote oxidative stress in the body by causing inflammation and therefore promote atherosclerosis formation.^{24,46}

Constant states of hyperglycemia can also cause the arteriole walls to become brittle due to glycosylation or calcification of the arteries. Neuropathy is another factor that increases the risk of CVD in individuals with type 1 diabetes. The impaired function of the nerves can impair the myocardial function and restrict the blood flow to the heart, promoting CVD. Individuals who have type 1 diabetes and follow an unhealthy diet will also have an increased risk for CVD.

When individuals with type 1 diabetes do not inject adequate insulin, BG levels will be elevated and additional insulin will be needed to correct the BG and reach homeostasis. If the carbohydrate intake is over the threshold for energy production, then the remaining glucose will be converted into fat and stored. If this becomes a habitual diet, the increased fat storage will result in an increase in LDL levels, and an increased risk of hypertension, atherosclerosis and CVD, and stroke.²⁴ Continued excessive fat storage will then contribute to weight gain, leading to a body mass index (BMI) of overweight or obese status, which increases the risk of CVD.⁴⁷

The inflammation caused by hyperglycemia can also contribute to the development of CVD. In a population based cohort study with individuals with type 1 diabetes, Grauslund et al examined the relationship between glycemic control and the presence of dyslipidemia and renal dysfunction, as well as all cause and CVD mortality, and ischemic heart disease. The participants had lived with type 1 diabetes for at least 20 years and were followed for a 13-year time period, ample time for complications and or mortality to occur. The study found that, with long-term

glycemic and metabolic control, the individuals with type 1 diabetes had less macrovascular damage. Therefore, glycemic control, dyslipidemia, and renal dysfunction were all related to the all cause mortality outcome and ischemic heart disease development in the study participants.⁴⁸ Some individuals with type 1 diabetes and atherosclerosis need coronary bypass surgery. Therefore, postoperative issues need to be considered as complications from type 1 diabetes. In a study done in Sweden, the researchers observed type 1 diabetic patients' HbA1c levels and the mortality or adverse cardiovascular effects present. The researchers determined that if the bypass patients had poor glycemic control before surgery, then they had a higher risk of mortality and adverse cardiovascular outcomes.⁴⁹ Therefore, not only would postprandial physical activity have a beneficial effect on glycemic control, may reduce postoperative complications and mortality risk.

Obesity does not only occur in type 2 diabetes; the epidemiological shifts in the population and increased calorie intake in individuals have contributed to an increase in the percentage of individuals with type 1 diabetes who are obese; the prevalence of obesity has increased from 1% to 31%, in individuals with type 1 diabetes, as reported in the 1993 Diabetes Control and Complications Trial (DCCT).^{50,51} Clinical trials have provided evidence that intensive insulin therapy can result in excessive weight gain in a subset of patients with type 1 diabetes. Obesity in individuals with type 1 diabetes is directly related to increased risk for CVD by increasing CVD risk factors such as increased visceral adiposity, high blood pressure, increased levels of LDL lipoproteins, and insulin resistance. Insulin resistance is also known to increase the risk in coronary heart disease in individuals with type 1 diabetes as well.⁵⁰ To further support this point, Brazeau et al, indicated in their literature review that individuals with type 1 diabetes are known to decrease their physical activity because of the risk of

hypoglycemia. However, without physical activity present in the lifestyle, individuals with type 1 diabetes can be at risk for weight gain, which can then lead to obesity. This study found that increased physical activity in type 1 diabetics resulted in a decrease in BMI and body fat percentage, although that there was no change in the HbA1c and insulin dosages.⁵²

A stroke, another complication of type 1 diabetes, occurs when there is a blood vessel blockage in the brain, cutting off blood flow, and thus oxygen and nutrient delivery to the brain tissue, which results in tissue death. The blockage occurs when a blood clot gets stuck in an artery narrowed by atherosclerosis. Individuals with type 1 diabetes are at risk for experiencing a stroke at some point in their lives because of the macrovascular damage that is caused by high glucose concentrations, elevated fat and cholesterol intake, and weight status. AGEs can contribute to the potential increased severity of a stroke associated with diabetes by contributing to the neurotoxicity.⁵³ In one study, other diabetes complications, retinopathy and neuropathy, were examined in relation to stroke incidence. The researchers did find a positive correlation between the presence retinopathy and neuropathy, independently, and the risk of stroke occurring in individuals with type 1 diabetes.^{24,54}

Expenses of Type 1 Diabetes

Besides preventing the development of the complications of type 1 diabetes, glycemic control promoted by postprandial physical activity could have a significant effect on the cost of diabetes management. Diabetes comes with many expenses including supplies for insulin injections and BG monitoring, multiple physicians appointments for management and check-ups on complications, and additional medication to manage and treat the complications of diabetes. A 2010 study on the cost of managing type 1 diabetes using a national dataset the estimated cost per year was \$10.6 billion, during the years 1999 and 2005, and \$422.9 billion for a lifetime,

which included both self-payments and third party insurance payments. The expenses included hospital inpatient stays, ambulatory visits, emergency room visits, prescription drugs, medical supplies, home health providers, and vision and dental screening and treatments. These estimates represent the costs of both type 1 and type 2 diabetes combined. The researchers calculated that the overall direct medical cost per capita was \$9,868 per patient year, with \$6,288 attributed to type 1 diabetes.⁵⁵ The Juvenile Diabetes Research Foundation (JDRF) recently reported that annual healthcare costs associated with type 1 diabetes in the United States is estimated to be \$14 billion.³ Between December 2008 and December 2010, researchers reported the cost of medication for diabetes medication and management, medical tests, and consultations with physicians and dietitians for individuals with type 1 diabetes in Brazil. The expenditure on outpatient treatment alone was \$1,216.33 per patient per year, with the average annual cost being \$3,867,915.66, which represents about 92% of the overall direct medical cost. The outpatient treatments included insulin, oral drugs, supplies to monitor BG, insulin pump and supplies, and medical procedures, including hemodialysis. Insulin was estimated to be \$348.78, oral drugs \$27.60, supplies for BG monitoring \$696.78, insulin pumps and supplies \$72.53, and medical procedures \$75.64, all representing per capita per year. The United States is known to have extremely high medical costs compared to some other countries, and therefore the costs would reflect a different amount than Brazil, and taking the inflation over the years into consideration.⁵⁶ According to Accu-Chek[®], the average cost of an insulin pump is \$6,500 in the United States, with an additional average expense of \$1,500 per year for the pump supplies.⁵⁷ However, the cost of type 1 diabetes can be much greater depending on the amount and type of supplies, number of visits to physicians and other health professionals, and the amount that medical insurance covers.

Summary

From this literature review on type 1 diabetes, glycemic control, and physical activity, there appears to be evidence that postprandial physical activity may blunt the glycemic response to a meal containing carbohydrates in individuals with type 1 diabetes. This study can add to the evidence by determining if a short bout, 15 minutes, of low intensity physical activity after consumption of a carbohydrate-containing breakfast beverage specifically can attenuate the spike in BG and the overall glycemic response specifically with participants with type 1 diabetes in a free-living situation. To date, several similar studies have been conducted that show promising effects of postprandial physical activity on the glycemic effect of a carbohydrate load, but no controlled crossover designs have been published with individuals with type 1 diabetes as the participants free-living on intensive insulin therapy regimens with a short bout of light activity. Previous studies involving participants with type 1 diabetes have also used small cohorts of participants in their data collection. A review article listed studies on the effect of the timing of physical activity and glycemic response, and most studies used a cohort of less than 10 participants.⁴² If more studies, such as this one, indicate a beneficial effect of postprandial physical activity, a routine could be used to improve glycemic control, prevent or delay the progression to diabetic complications. With decreased medical costs, including supplies, insulin dosages, and medical visits, not only will money be saved, but quality of life may be improved as well. In summary, the research reviewed for this study suggests that postprandial physical activity may be one method for attenuating the BG increase resulting from carbohydrate intake and therefore, may indirectly reduce the dosage of insulin needed to compensate the carbohydrate intake, prevent or delay complications, and reduce costs related to treatment.

Research Methodology

Participants

Institutional Review Board approval was obtained from University of the Incarnate Word prior to the commencement of this study. Volunteers were recruited from former participants of type 1 diabetes summer camp in San Antonio, as well as the snowball effect. Participants included 7 healthy adult individuals with type 1 diabetes, ages 18 to 30 years old who volunteered to participate. To be included in the study, participants had to indicate on a screening questionnaire that they: were a non-pregnant healthy adult with type 1 diabetes, over the age of 18, weighing at least 110 lb, no allergies or intolerances to soy or milk, willing to consume a breakfast consisting 8 oz container of Boost® (a nutritional beverage, which provides 240 Calories and a 41 g load of carbohydrate, which is mainly composed of corn syrup and sugar, knew how much insulin to inject for 41 g of carbohydrate), did not have any condition that would render them unable to walk at a light pace for 15 minutes, willing to fast for at least 8 hours, but no longer than 12 hours, prior to data collection; and had documentation of an HbA1c reading of <9.5% within the previous month. Participants meeting the inclusion criteria were contacted with instructions, including that they were to refrain from physical activity the 24 hours prior to data collection, and to contact the investigators if their morning BG was not between 70 – 200 mg/dl the day of data collection. If their BG was not in that range, they rescheduled for a different day, or corrected with fast acting insulin if high, or a small glass of juice or a glucose tablet if low. After the correction, they had to wait at least 1 hour for data collection to minimize the impact of the insulin or carbohydrate correction on data collection. Two participants had to perform insulin corrections for one of the days, and one needed to

perform a carbohydrate correction. All participants signed an informed consent form prior to data collection.

Blood Glucose Measurements

Glucometers were calibrated with a test solution for mg/dL prior to data collection. All participants knew how to determine BG using the glucometer (FreeStyle Lite; Abbott Diabetes Care, Inc, Alameda, California) used in this study. The measurement included wiping a finger with an alcohol pad and then allowing the finger to dry. A lance was then used to prick the finger for a small drop of blood. A glucose test strip was placed at the finger where the small drop of blood appeared and then placed in the glucometer for determination of BG level. The finger was then wiped again with the alcohol pad. Glucometers were numbered, and the participants used the same glucometer for each day of data collection.

Data Collection

Data were collected on three Saturdays; participants were required to report for data collection on two of the days. On the first day, participants reported to a designated room on the University of the Incarnate Word campus after an overnight fast and after determining that their morning BG was in the required range. They filled out a form detailing demographic and anthropometric information including gender, age, most recent HbA1c value, and number of years since diagnosis of diabetes. Weight was measured using a beam balance scale, and height was determined using a stadiometer. BMI was calculated as weight in kilograms divided by height in squared meters. The participants measured their BG levels using the glucometer twice. If the 2 measures were not within 10 mg/dL they measured a third time. The 2 values within 10 mg/dL were averaged for the baseline glucose value. Each participant then injected the appropriate amount of insulin, via insulin pump or a syringe; the amount of insulin administered

was based on their carbohydrate to insulin ratio needed for the 41 g Boost beverage and their fasting BG levels. Immediately after insulin was self-administered, participants drank a chilled Boost® beverage within 10 minutes. The participants then sat quietly and repeated BG measurements at 15, 30, 60, 90, and 120 minutes after beverage consumption, and recorded their values on a data sheet. The investigators collected the data sheets at the end of the first day of data collection. On the second day, the participants followed the same protocol as the first day: measured baseline BG, self-administered insulin for 41 g of carbohydrate, and drank a chilled Boost® beverage within 10 minutes. However, the second day included the walking intervention. The participants engaged in 15 minutes of walking at a light pace (50-60% max HR) immediately after consuming the Boost® beverage on the treadmills in the Wellness Center at the University of the Incarnate Word. The max HR range for each participant had been calculated for them and their HR was self-monitored using a Polar® heart rate monitor. After 15 minutes of walking, the participants measured their BG readings at 15, 30, 60, 90, and 120 minutes, as they did the first day. After all the data was collected, the data sheets were collected from the participants by the investigators.

Glucose incremental area under the curve (iAUC) was determined for the walking and sedentary days for each participant. The difference from peak to baseline BG was determined. Descriptive statistics were performed on the demographic, anthropometric, and BG measurements, including iAUC. Paired t-test was used to determine differences in the magnitude of BG spike (peak – baseline), iAUC and baseline BG for the walking and sedentary days. SPSS was used for data analysis (IBM SPSS Statistics 22, Armork, NY), and significance was set at $P < .05$.

Results

Participant Characteristics

Seven participants completed the study; 4 males and 3 females, age 22.3 ± 4.3 years old, with type 1 diabetes diagnosed at age 12.7 ± 3.5 years old. Three were Hispanic and 4 were non-Hispanic Caucasian. BMI ranged from 21.5 to 31.4, with 3 in the healthy range of 18.0-24.9, and 4 were above the healthy range. The HbA1c was 7.4 ± 0.5 %, with a range of 6.6 - 8.1 indicating reasonably good control, although only two were in the target range of <7.0 . See Table 1 for a summary of participant characteristics.

Blood Glucose Indicators With and Without Walking

BG data points were all converted from mg/dL to mmol/L to interpret the results. The mean fasting BG for both days were the same with statistical significance ($P=.012$), but each individual fast BG for each day were different for each participant, but there was no significance in peak BG between the days ($P=.105$). However, the magnitude of the BG spike after consumption of the breakfast beverage (peak BG – baseline BG) was significantly lower on the day the participants walked at a light pace immediately after consumption vs the sedentary day (6.4 ± 1.2 vs 14.4 ± 3.4 , respectively, $P=.045$). The glucose iAUC was lower on the walking day compared to the sedentary day as well (241.1 ± 155.8 vs 486.6 ± 94.5 , respectively, $P=.016$). The trends found with the BG spike and iAUC were found in all participants but one. Thus, the intervention with light physical activity did attenuate the spike in BG and decreased the iAUC, after consumption of a breakfast beverage. The average decrease in the BG spike was 11.8% and the average decrease in iAUC was 47.2%. The average percent decrease in the difference between peak and baseline from day 1 VS day 2 was 36.2%. The spike in BG and iAUC were lower in each participant on the walking day, indicating favorable effect of postprandial walking.

See Table 2 for a comparison of BG indicators between the sedentary and walking days, and a comparison of the spike in BG between the days for each participant is displayed in Figure 1.

Figure 2 shows the iAUC curves for each participant for the sedentary and walking days. For most participants, BG peaked at the 30 minute time point on the walking day and 60 minute on the sedentary day. Participant 3's BG dropped to 5.1 mmol/L at the 90 minute time point, so she consumed a dextrose tablet and her 120 minute BG value was omitted. Otherwise, the 15 minutes of walking did not result in hypoglycemia for any of the participants. Figures 3 and 4 show comparisons of the average Peak-Baseline BG and iAUC, respectively, on sedentary and walking days. Figure 5 shows the BG response in each participant, with each BG reading represented, on both sedentary and walking days.

Table 1. Participant Characteristics

| Variable | Mean \pm SD (<i>N</i> = 7) | Range |
|-----------------------------------|----------------------------------|---------------|
| Age (yrs) | 22.3 \pm 4.3 | 18.0 – 30.0 |
| Height (cm) | 168.0 \pm 7.3 | 160.0 – 180.3 |
| Weight (kg) | 71.9 \pm 10.0 | 55.5 – 85.6 |
| BMI ¹ | 25.4 \pm 3.5 | 21.6 – 31.4 |
| HbA1c (%) (mmol/mol) ² | 7.4 \pm 0.5 | 6.6 – 8.1 |
| Years with Type 1 Diabetes | 12.7 \pm 3.5 | 7.0 – 17.0 |

¹ BMI = Body Mass Index; weight in kilograms divided by height in meters squared

² HbA1c = glycated hemoglobin

Table 2. Blood Glucose Indicators of Sedentary and Walking Days

| Variable | Day1 ¹ Mean \pm SD (N = 7) | Day 2 ¹ Mean \pm SD (N = 7) | P |
|---------------------------------------|---|--|------|
| Baseline BG (mmol/L) ² | 8.0 \pm 2.9 | 8.0 \pm 2.5 | .012 |
| Peak BG (mmol/L) | 14.4 \pm 3.4 | 12.2 \pm 1.9 | .105 |
| Peak - Baseline ³ | 6.4 \pm 1.2 | 4.0 \pm 2.4 | .045 |
| iAUC (mmol/L/120 min) ⁴ | 468.6 \pm 94.5 | 241.1 \pm 155.8 | .016 |

¹Day 1 was the sedentary day; Day 2 participants walked after consuming the beverage.

²BG = Blood Glucose

³The difference between Peak BG and Baseline BG

⁴iAUC = Incremental Area Under the Glucose Curve

Figure 1. Difference between baseline and peak blood glucose (mmol/L) for sedentary and walking days

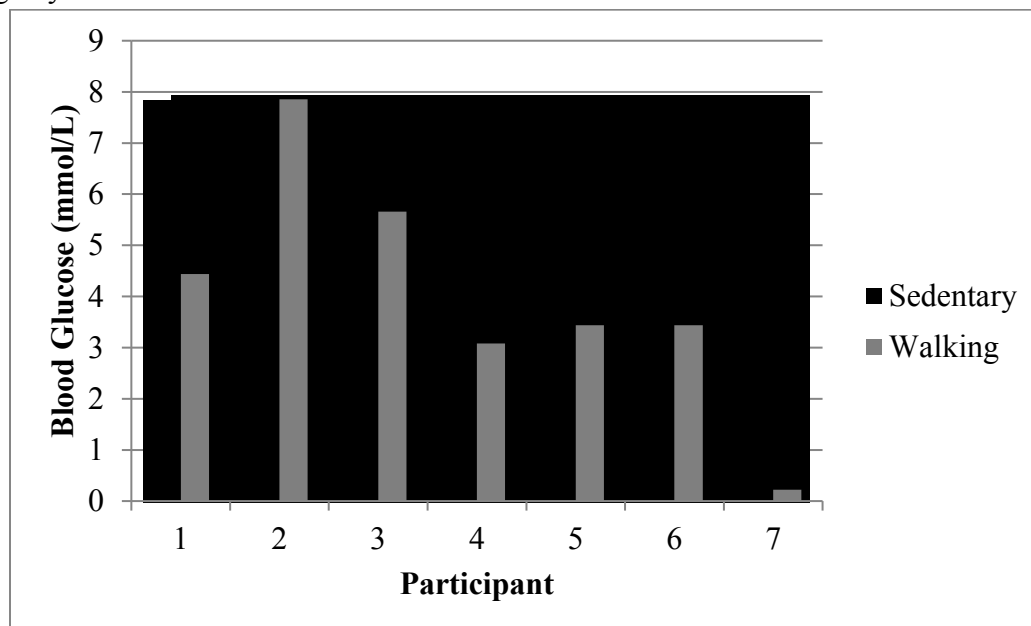


Figure 2. iAUC (mmol/L/120min) Sedentary VS Walking For Each Participant

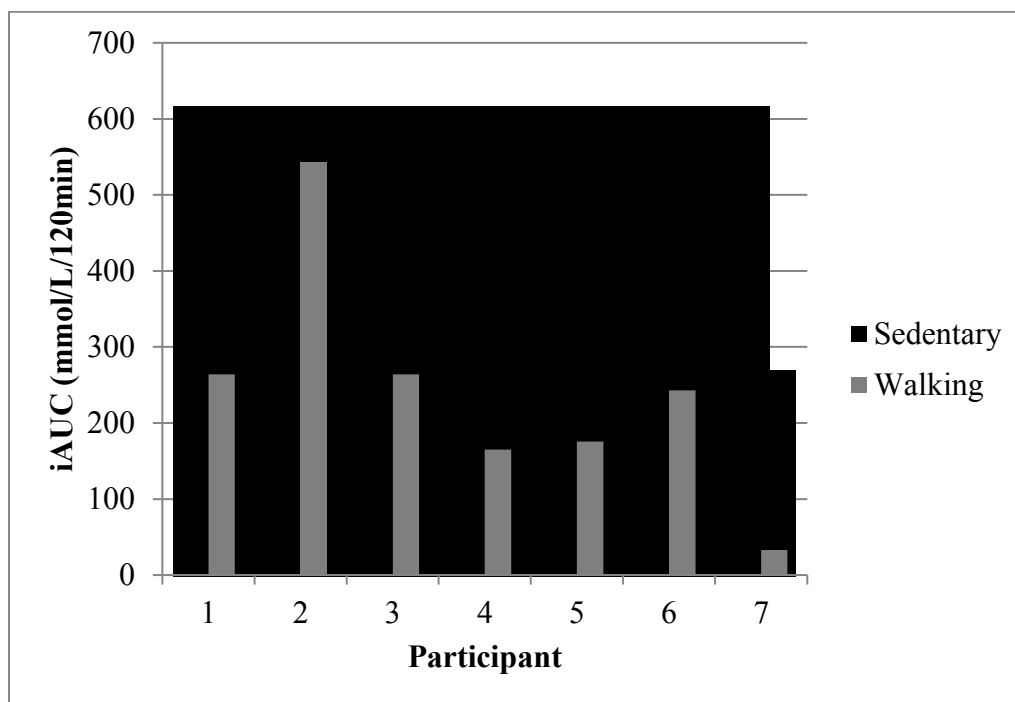


Figure 3. Comparison of Peak-Baseline BG on Sedentary and Walking Days

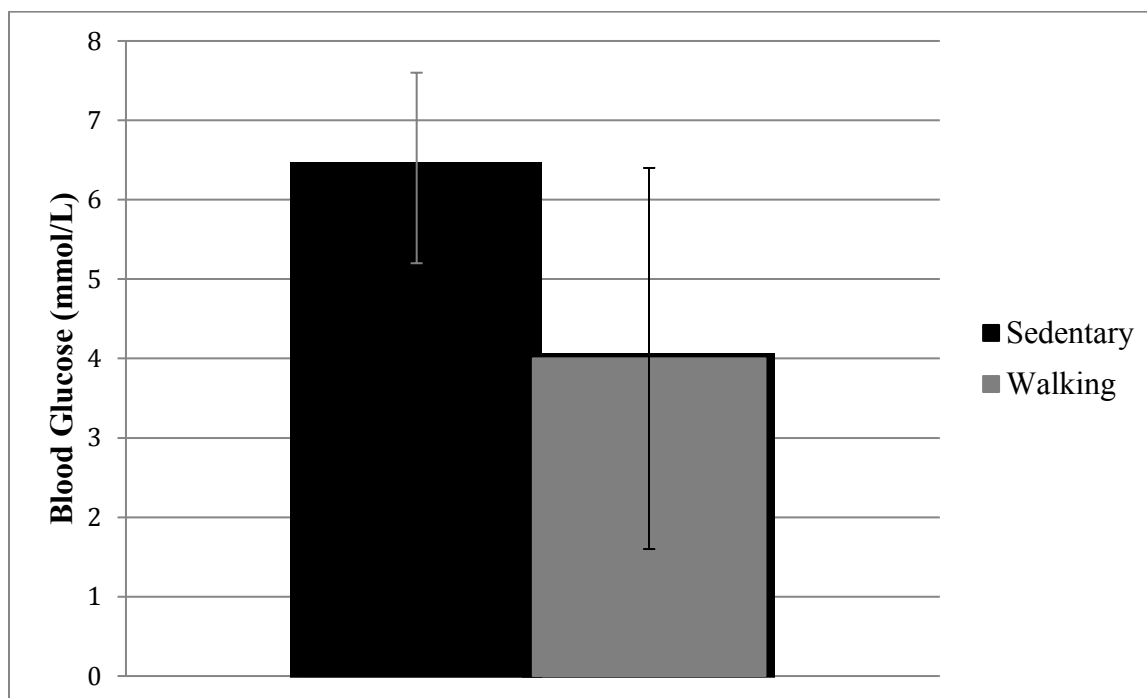


Figure 4. Comparison of iAUC on Sedentary and Walking Days

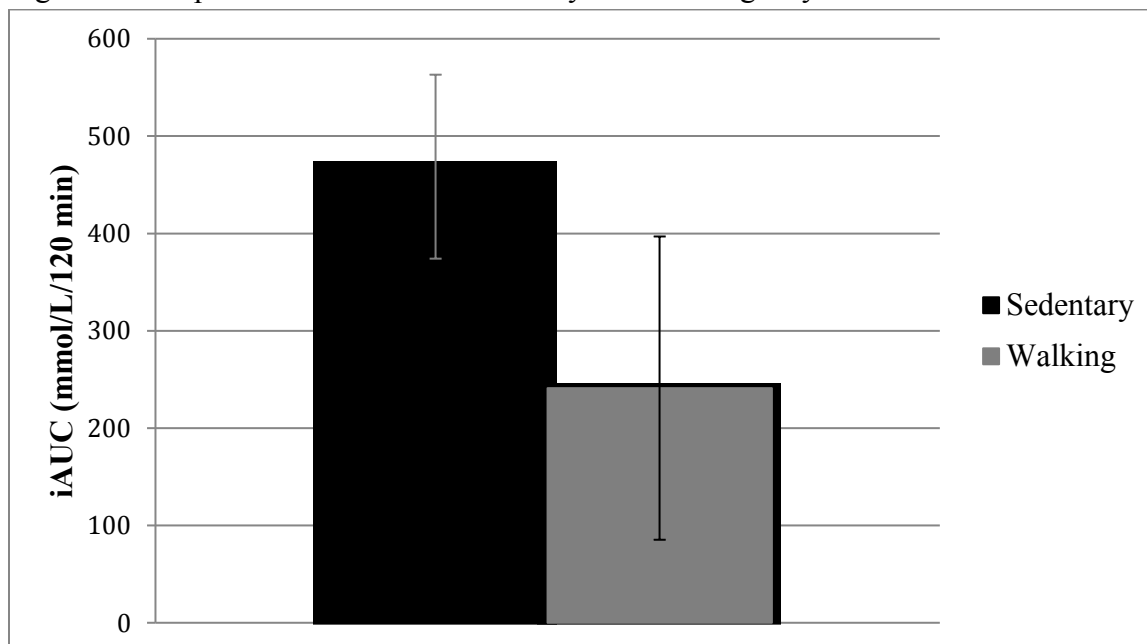
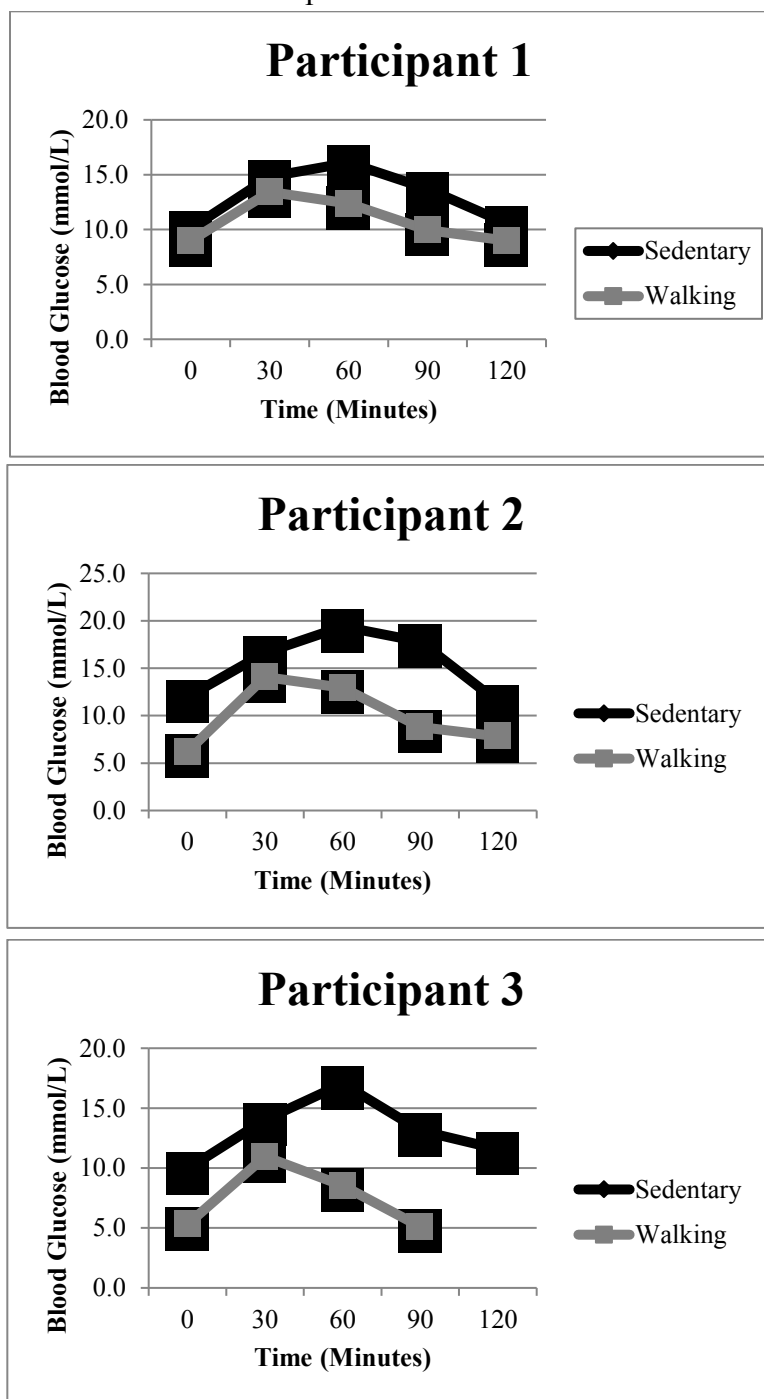
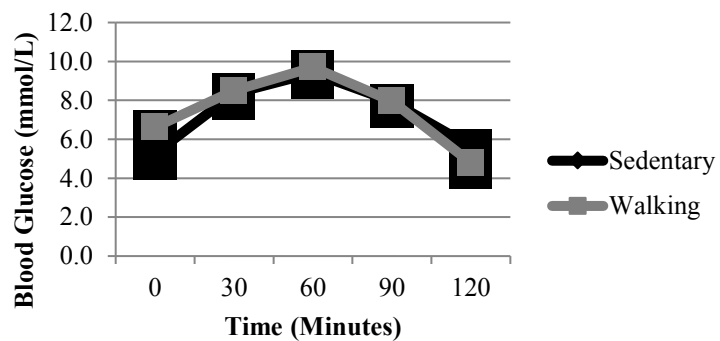


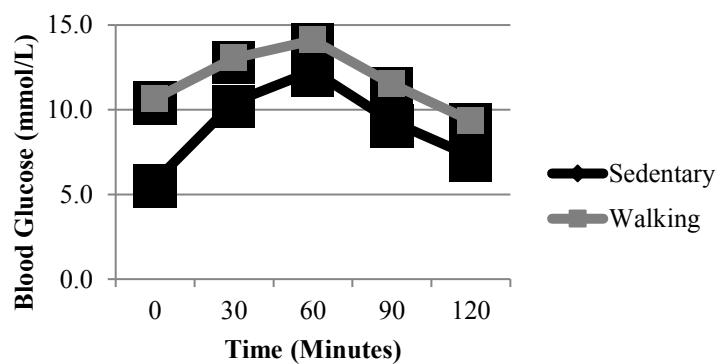
Figure 5. Glucose Curves for Each Participant



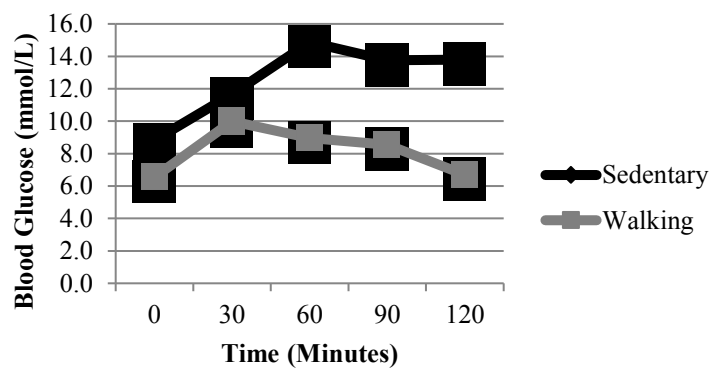
Participant 4

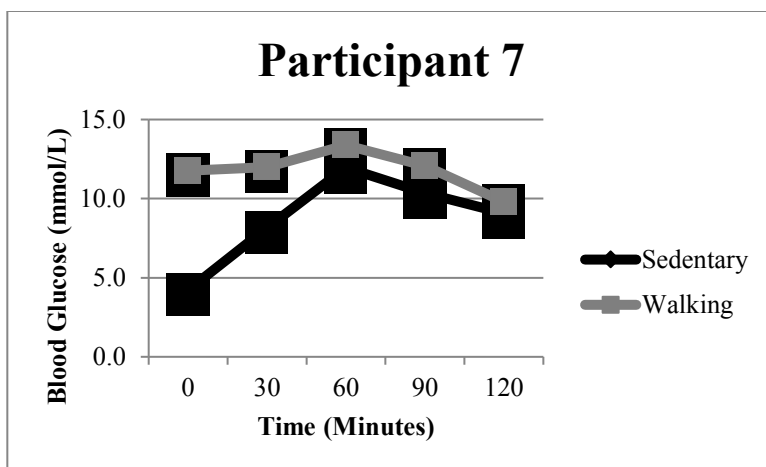


Participant 5



Participant 6





Discussion

The results of the present study confirm the beneficial effect of postprandial walking, at a light pace, on the glycemic response to a meal. In contrast to other studies, the participants in the present study followed their usual insulin regimen, administered a bolus of insulin appropriate for the 41 g carbohydrate load provided by the Boost[®] beverage, and the physical activity was shorter in duration, commencing immediately after meal consumption.^{11-15,32,39} Other studies did control for fasting BG levels by the use of an artificial pancreas or insulin infusions the night before data collection and the present study did not.^{11-14,39} Although the fasting BG levels differed in some of the participants on the two data collection days, all but one of the seven participants had a lower spike in BG (peak - baseline BG) and lower iAUC on the postprandial walking day compared to the sedentary day. The results showed that the spike (peak - baseline) in BG was diminished by an average of 36.2% with the postprandial physical activity intervention. The iAUC was also decreased by an average of 47.2% with postprandial walking. One participant had a 90 minute BG in the 80 mg/dL range, so a dextrose tablet was taken to prevent hypoglycemia, and the 120 minute BG was not used in the data analysis. A decrease in the spike in BG and glycemic response from a carbohydrate intake would be beneficial for individuals with type 1 diabetes because it could possibly decrease the risk for long term complications and improved health status.

The shorter duration of postprandial physical activity in the present study, 15 min of walking immediately after meal consumption at a light pace, may be a more realistic approach for compliance than the studies that used longer durations and intensities of physical activity or a wait period after the meal before commencing with physical activity.^{11-14,32,39} For example, on a

lunch hour, 15 minutes of walking is realistic, whereas 30-45 minutes of physical activity would not be feasible in a lunch hour.

One concern related to physical activity in individuals with type 1 diabetes is hypoglycemia, which is why light walking was chosen as the postprandial physical activity.^{31,34,35} Since exercising muscle and insulin have a synergistic effect on facilitating BG uptake by muscle cells, those administering insulin with meals and performing physical activity right after food consumption could be at increased risk for hypoglycemia. With cautious BG monitoring or the use of a continuous glucose monitor and being mindful of the time of day and food consumption, individuals with type 1 diabetes can successfully partake in physical activity, avoiding hypo- or hyperglycemia.^{16,42} In addition to glycemic control, another benefit of physical activity overall, in individuals with type 1 diabetes, is to potentially decrease the risk of secondary complications, such as CVD, stroke, obesity, micro-, and macrovascular disease by means of the glycemic control.²⁴

In interpreting the results of the present study, some limitations must be considered. The study included a low participant number, although there was enough power to achieve statistical significance. In addition, fasting BG was variable among many participants on the two testing days. Morning insulin injection or a small carbohydrate dose was needed to correct BG levels to meet the range for participation by a few participants, who were required to wait an hour after correction before data collection commencement to minimize confounding effects. The age group of participants, all young adults, ages 18 to 30 years old, limits the generalizability of the results, as older or younger individuals may not respond the same. Another limitation of the study would be the method used to measure BG values, which was via glucometers, which are not as reliable and valid as venous blood samples. The study design component of having the

participants monitor and record their blood glucose readings, and monitor their heart rates is another limitation that brings potential error in the data because the data readings could be easily changed to favor the hypothesis. The participants also determined the amount of insulin themselves and self-administered the insulin without recording the amount of insulin injected for the 41 g of carbohydrates.

Conclusion

Prevention of BG spikes is important for individuals with type 1 diabetes, not only to help with achieving glycemic control, but to also decrease the amount of insulin needed to achieve control and to prevent secondary complications from forming that could potentially impair quality of life, such as CVD, stroke, and microvascular disease. The results of the present study show that a short bout (15 minutes) of light postprandial physical activity, consisting of walking, can blunt the BG spike postprandial to a Boost® beverage in participants with type 1 diabetes on intensive insulin therapy regimens. With mounting evidence, health professionals may consider recommending postprandial physical activity to their patients to optimize glycemic control, with the caution that BG must be closely monitored. A continuous BG monitor would be ideal to monitor BG when exercising. Although there appears to be a beneficial effect of postprandial physical activity on glycemic response to a meal, the long-term effect of regular postprandial activity on glycemic control and development of complications is unknown. Future studies need to examine the effect of light postprandial activity for some or all meals, following participants over a period of time to determine if HbA1c levels are influenced.

References

1. Mahan LK, Escott-Stump S, Raymond JL. *Krause's Food & the Nutrition Care Process*. 13th Edition. St. Louis, MO: Elsevier. 2012; p. 54, 197, 203, 676-709, 743, 758, 769, 885.

2. American Diabetes Association. Statistics About Diabetes. Diabetes.org, 2016. Accessed from: <http://www.diabetes.org/diabetes-basics/statistics/>. Accessed July 2016.

3. Juvenile Diabetes Research Foundation. Type 1 Diabetes Facts. Jdrf.org, 2016. Accessed from: <http://www.jdrf.org/about/fact-sheets/type-1-diabetes-facts/>. Accessed July 2016.

4. McArdle, WD, Katch FI, and Katch VL. *Exercise Physiology; Energy, Nutrition and Human Performance*. 5th ed. Philadelphia, PA, Lippincott, Williams, and Wilkins. 2001; p. 428.

5. Heiss CJ, Tollefson M. Postprandial light exercise attenuates the glycemic effect of a candy bar. *Top Clin Nutr*, 2014;29(2):132-138.

6. Henson J, Davies MJ, Bodicoat DH, Edwardson CL, Gill JMR, Stensel DJ, et al. Breaking up prolonged sitting with standing or walking attenuates the postprandial metabolic response in post menopausal women: A randomized acute study. *Diabetes Care*, 2016;39(1),130-138.

7. DiPietro L, Gribok A, Stevens MS, Hamm LF, Rumpler W. Three 15-min bouts of moderate postmeal walking significantly improves 24-h glycemic control in older people at risk for impaired glucose tolerance. *Diabetes Care*, 2013;36(10):3262-3268.

8. Heiss CJ, Goldberg LR. Post-meal exercise my attenuate the glycemic response to a carbohydrate load: important implications for adults who are obese, with pre-diabetes or diabetes, and/or at-risk for dementia. *OROJ*, 2015;2:81-88.

9. Høstmark, AT, Ekeland, G.S, Beckstrøm, AC, Meen, HD. Postprandial light physical activity blunts the blood glucose increase. *Prev Med*. 2006;42(5): 369–371.

10. Nygaard, H, Tomten, S, Hostmark, A. Slow post meal walking reduces postprandial glycemia in middle-aged women. *Appl Physiol Nutr Metab*. 2009;34(6);1087-1092.

11. Nelson JD, Poussier P, Marliss EB, Albisser AM, Zinman B. Metabolic response of normal man and insulin infused diabetics to postprandial exercise. *Am J Physiol*, 1982;242(5):E309-E316.
12. Rasmussen OW, Lauszus FF, Hermansen K. Effects of postprandial exercise on glycemic response in IDDM subjects. *Diabetes Care*, 1994;17(10):1203-1205.
13. Caron D, Poussier P, Marliss B, Zinman B. The effect of postprandial exercise on meal-related glucose intolerance in insulin-dependent diabetic individuals. *Diabetes Care*, 1982; 5(4): 364-369.
14. Yamanouchi K, Abe R, Takeda A, Yoshihito A, Shichiri M, Sato Y. The effect of walking before and after breakfast on blood glucose levels in patients with type 1 diabetes treated with intensive insulin therapy. *Diabetes Res Clin Pract*, 2002: 11-18.
15. Cuenca-García M, Jago R, Shield JPH, Burren CP. How does physical activity and fitness influence glycemic control in young people with type 1 diabetes? *Diabetic Med*, 2012;29(10):e369-e376.
16. Colberg SR, Laan R, Dassau E, Kerr D. Physical activity and type 1 diabetes: time for a rewire? *J Diabetes Sci Technol*, 2015;9(3):609-618.
17. Chimen M, Kennedy A, Nirantharakumar K, Pang TT, Andrews R, Narendran P. What are the health benefits of physical activity in type 1 diabetes mellitus? A literature review. *Diabetologia*, 2012; 55: 542-551.
18. De Vries L, Oren L, Lazar L, Lebenthal Y, Shalitin S, Phillip M. Factors associated with diabetic ketoacidosis at onset of type 1 diabetes in children and adolescents. *Diabet Med*, 2013;30(11):1360-1366.
19. American Diabetes Association. Checking your blood glucose. Diabetes.org, 2015. Accessed from: <http://www.diabetes.org/living-with-diabetes/treatment-and-care/blood-glucose-control/checking-your-blood-glucose.html>[SH1] . Accessed November 2015.
20. American Diabetes Association. Postprandial blood glucose. *Diabetes Care*, 2001;24(4): 775-778.

21. American Diabetes Association. Standards of medical care in diabetes-2015. *Diabetes Care*. 2015;38(1);S33-S40.
22. Giugliano D, Ceriello A, Esposito K: The effects of diet on inflammation: emphasis on the metabolic syndrome. *J Am Coll Cardiol*. 2006;48(4):677-85.
23. Seaman DR. The diet-induced proinflammatory state: a cause of chronic pain and other degenerative diseases? *J Manipulative Physiol Ther*. 2002;25(3):168-79.
24. American Diabetes Association. Complications. 2015; <http://www.diabetes.org/living-with-diabetes/complications/>. Accessed April 4, 2015.
25. Simm A, Wagner J, Gursinsky T, Nass N, Friedrich I, Schinzel R, Czeslik E, Silber RE, Scheubel RJ. Advanced glycation endproducts: a biomarker for age as an outcome predictor after cardiac surgery? *Exp Gerontol*. 2007;42(7):668-75.
26. Goldin A, Beckman JA, Schmidt AM, Creager MA. Advanced glycation end products: sparking the development of diabetic vascular injury. *Circulation*. 2006;114(6):597–605.
27. Peppia, M, Uribarri, J, Vlassara, H. Glucose, advanced glycation end products, and diabetes complication: What is new and what works. *Clin Diabetes*. 2004;21(4):186-187.
28. Pickup, JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. *Diabetes Care*, 2004;27(3):813-823.
29. Esposito K, Nappo F, Marfella R, et al. Inflammatory cytokine concentrations are acutely increased by hyperglycemia in humans: role of oxidative stress. *Circulation*. 2002;106(16):2067-72.
30. Rakoff-Nahoum, S. Why cancer and inflammation? *Yale J Biol Med*. 2006;79:123-130.
31. Glade MJ, Smith K. A glance at...glycemic index. *J. Nutr*, 2015;31(3):539-541.

32. Campbell MD, Gonzalez JT, Rumbold PLS, Walker M, Shaw JA, Stevenson EJ, et al. Comparison of appetite responses to high- and low-glycemic index postexercise meals under matched insulinemia and fiber in type 1 diabetes. *Am J Clin Nutr*, 2015;101(3):478-486.

33. Pereira MA, Swain J, Goldfine AB, Rifai N, Ludwig DS. Effects of a low-glycemic load diet on resting energy expenditure and heart disease risk factors during weight loss. *JAMA* 2004; 292(20): 2482-90.

34. Juanola-Falgarona M, Salas-Salvadó J, Ibarrola-Jurado N, Rabassa-Soler A, Díaz-López A, Guasch-Ferré M, et al. Effect of the glycemic index of the diet on weight loss, modulation of satiety, inflammation, and other metabolic risk factors: a randomized control trial. *Am J Clin Nutr*, 2014;100(1):27-35.

35. Richter EA, Hargreaves M. Exercise, GLUT4, and muscle glucose uptake. *Physiological Reviews*. 2013; 93(3), 993-1017.

36. Riddell M, Perkins BA. Exercise and glucose metabolism in persons with diabetes mellitus: perspectives on the role of continuous glucose monitoring. *J Diabetes Sci Technol*, 2009; 3(4): 914-923.

37. What I need to know about Physical Activity and Diabetes. 2014. Available at: <http://www.niddk.nih.gov/health-information/health-topics/diabetes/physical-activity-diabetes/pages/physical-activity-diabetes.aspx>. Accessed May 2, 2016.

38. Joslin Diabetes Center. Why do blood glucose levels sometimes go up after physical activity? Joslin.org, 2016. Accessed from: http://www.joslin.org/info/why_do_blood_glucose_levels_sometimes_go_up_after_physical_activity.html. Accessed July 2016.

39. Manohar C, Levine JA, Nandy DK, Saad A, Man CD, McCrady-Spitzer SK, et al. The effect of walking on postprandial glycemic excursion in patients with type 1 diabetes and healthy people. *Diabetes Care*, 2012;35(12):2493-2499.

40. Galler A, Lindau M, Ernert A, Thalemann R, Raile K. Associations between media consumption habits, physical activity, socioeconomic status, and glycemic control in children, adolescents, and young adults with type 1 diabetes. *Diabetes Care*, 2011;34:2356-2359.

41. Plotnikoff RC, Lippke S, Courneya K, Birkett N, Sigal R. Physical activity and diabetes: an application of the theory of planned behavior to explain physical activity for type 1 and type 2 diabetes in an adult population sample. *Psychol Health*, 2010;25(1):7-23.
42. Chacko E. Exercising tactically for taming postmeal glucose surge. *Scientifica*, 2015;2016:1-10.
43. Sveen KA, Karime B, Jørum E, Mellgren SI, Fagerland MW, Monnier VM, et al. Small- and large-fiber neuropathy after 40 years of type 1 diabetes. *Diabetes Care*, 2013;36(11):3712-3717.
44. Kramer CK, Retnakaran R. Concordance of retinopathy and nephropathy over time in type 1 diabetes: an analysis of data from the diabetes control and complications trial. *Diabetic Med*, 2013;30(11):1333-13341.
45. Wadèn J, Tikkanen HK, Forsblom C, Harjutsalo V, Thorn LM, Saraheimo M, et al. Leisure-time physical activity and development and progression of diabetic nephropathy in type 1 diabetes: The FinnDiane Study. *Diabetologica*, 2015;58(5):929-936.
46. Lowe GD: Circulating inflammatory markers and risks of cardiovascular and non-cardiovascular disease. *J Thromb Haemost*. 2005;3(8):1618-27.
47. Schnell O, Cappuccio F, Genovese S, Standl E, Valensi P, Ceriello A. Type 1 diabetes and cardiovascular disease. *Cardiovasc Diabetol*, 2013;12:156-166.
48. Grauslund J, Jørgensen TMM, Nybo M, Green A, Rasmussen LM, Sjølie AK. Risk factors for mortality and ischemic heart disease in patients with long-term type 1 diabetes. *J Diabet Complications*, 2010;24:223-228.
49. Nystöm T, Holzmann MJ, Eliasson B, Kuhl J, Sartipy U. Glycemic control in type 1 diabetes and long-term risk of cardiovascular events or death after coronary artery bypass grafting. *J Am Coll Cardiol*, 2015; 66:535-543.
50. Ferranti SD, de Boer IH, Fonseca V, Fox CS, Golden SH, Lavie CJ, et al. Type 1 diabetes mellitus and cardiovascular disease a scientific statement from the American heart association and American diabetes association. AHA/ADA Scientific Statement, *Circulation*, 2014; 130: 1110-1130.

51. American Diabetes Association. History of Diabetes. Diabetes.org, 2014.
<http://www.diabetes.org/research-and-practice/student-resources/history-of-diabetes.html?referrer=https://www.google.com/>. Accessed July 2016.

52. Brazeau AS, Leroux C, Mircescu H, Rabasa-Lhoret R. Physical activity level and body composition among adults with type 1 diabetes. *Diabetic Med*, 2012;e402-e408.

53. Zimmerman GA, Meistrell M 3rd, Bloom O, Cockcroft KM, Bianchi M, Risucci D, et al. Neurotoxicity of advanced glycation endproducts during focal stroke and neuroprotective effects of aminoguanidine. *Proc Natl Acad Sci USA*. 1995;25;92(9):3744-8.

54. Hagg S, Thorn LM, Putaala J, Liebkind R, Harjutsalo V, Forsblom CM, et al. Incidence of stroke according to presence of diabetic nephropathy and severe diabetic retinopathy in patients with type 1 diabetes. *Diabetes Care*, 2013;36: 4140-4146.

55. Tao B, Pietropaolo M, Atkinson M, Schatz D, Taylor D. Estimating the cost of type 1 diabetes in the U.S.: a propensity score matching method. *PLos ONE*, 2010;5:1-11.

56. Cobas RA, Ferraz MB, Matheus ASM, Tannus LRM, Negrato CA, Araujo LA, et al. The cost of type 1 diabetes: a nationwide multicenter study in Brazil. *WHO*, 2013;91(6):434-440.

57. Accu-Chek. Healthcare Professionals: Frequently Asked Questions. Accessed from: <https://www.accu-chekinsulinpumps.com/ipus/hcp/faqs.html>. Accessed November 2015.

58. Mayo Clinic. Coronary Bypass Surgery. Mayo.org, 2016. Accessed from: <http://www.mayoclinic.org/tests-procedures/coronary-bypass-surgery/home/ovc-20166007>. Accessed July 2016.

59. Huntington's Outreach Project For Education, At Stanford. Inflammation: Glucocorticoids. Web.stanford.edu, 2010. Accessed from: http://web.stanford.edu/group/hopes/cgi-bin/hopes_test/glucocorticoids/#what-are-glucocorticoids. Accessed July 2016.

60. Running For Fitness. VO2 Max. Runningforfitness.org, 2016. Accessed from: <http://www.runningforfitness.org/faq/vo2-max>. Accessed July 2016.

Appendices

Appendix A

GLOSSARY OF ABBREVIATIONS, TERMS, AND VARIABLES

AGEs: Advance glycation end products: the result of the post-translational modification of proteins, lipids, or DNA, which results in the crosslinking of collagen and stiffening of connective tissue, including the tissue found in the arteries, attracting macrophages, monocytes, cardiac fibroblasts, and vascular smooth muscle cells.¹⁰

Atherosclerosis: a disease in which plaque builds up in the arteries caused by atherogenesis that is a chronic and local inflammatory response to lipid profiles.¹

AUC: Area under the glucose curve

Beta cell autoimmune: antibodies have been made against the host's own beta cells found in the pancreas and the body's immune system attacks itself.¹

BG: Blood Glucose

BMI: Body Mass Index; measurement used to determine weight status and is calculated as weight in kilograms divided by height in squared meters.¹

Carbohydrate Exchange: a group of foods that are divided into carbohydrates, which include starches, fruits, milk, sweets, desserts, and other carbohydrates, including non-starchy vegetables, meat and meat substitutes, fats, and free foods.¹

Catecholamines: organic molecule released by the adrenal medulla that include epinephrine and norepinephrine that can act as a hormone or neurotransmitter.¹

CDE: Certified Diabetes Educator

Conventional Regimen: involves 2-3 insulin injections a day with a mixture of insulin types that peaked at specific times

when meal with controlled carbohydrate content had to be consumed.¹

Coronary Bypass: is a procedure that diverts the flow of blood around a section of a blocked artery in your heart to restore regular blood flow.⁵⁸

C Reactive Protein: a positive acute phase protein that is increased during inflammation and contributes to an inflammatory response.¹

CVD: Cardiovascular Disease: a group of interrelated diseases including coronary heart disease, atherosclerosis, hypertension, ischemic heart disease, peripheral vascular disease, and heart failure.¹

DCCT: Diabetes Control and Complications Trial: one of the classic clinical trials that has linked glycemic control and the development of complications in individuals with type 1 and type 2 diabetes, with also signifying the importance to nutrition therapy to achieve glycemic control. 1400 participants were administered insulin either as an intensive regimen, with multiple injections or the use of an insulin pump, or a conventional regimen of one or two injections per day. The results were that if the conventional regimen participants could reach the same glycemic control as the intensive, then the risk of complications could be reduced by 50-75%.¹

DKA: Diabetic Ketoacidosis: a complication from diabetes, characterized by the disruption of carbohydrate, protein, and fat metabolism, in which produces ketones in response to glucose not being

available for metabolism, that can be potentially life threatening, but reversible.¹

Dyspnea: shortness of breath.¹

GI: Glycemic Index: a measurement of the relative area under the postprandial glucose curve of 50 g of carbohydrates, from one food item, compared to 50 g of a standard food of measurement, such as white bread.¹

GL: Glycemic Load; GI multiplied by the amount of carbohydrate per serving that measures the impact of a certain amount of food on glycemic response.¹⁸

Glucagon: a hormone that is counter-reactive to insulin and restores blood glucose to normal levels by promoting glycogenolysis and gluconeogenesis.¹

Glucocorticoids: are steroid hormones (cortisol) with anti-inflammatory properties produced by the human body.⁵⁹

Glucometer: calibrated, hand held device that is used to self monitor blood glucose.¹

GLUT 4: glucose transporter that is insulin dependent found in the adipose and muscle cells.¹

Grams (g): measurement of mass

HbA1c: Glycated Hemoglobin: a test value that reflects long-term glucose concentrations by showing the amount of proteins that are exposed to glucose and the weighted average of plasma glucose concentration over several weeks time period.¹

Hypoglycemia: a state when the blood glucose concentration falls below the normal range.¹

Hyperglycemia: a state when the blood glucose concentration elevates above the normal range.¹

Hypertension: persistently high arterial blood pressure based on the force exerted

per unit area of the artery wall. Diagnosed as a systolic blood pressure of 120 mmHg or higher and a diastolic of 80 mmHg or higher.¹

iGAUC: Incremental glucose area under the curve

Insulin Bolus: administration of insulin used in response to a meal intake and or to correct for hyperglycemia.¹

Insulin: a hormone that is produced by the β cells, which are in the pancreas, in response to food ingested to maintain blood glucose homeostasis.¹

Insulin Pump: an option of insulin therapy used in individuals with type 1 diabetes that provides a basal of rapid acting or short acting insulin continuously and a bolus for when carbohydrates are consumed.¹

Intensive Regimen: involves administering a bolus of fast acting insulin, at each meal, in accordance with the carbohydrate content of the meal.¹

Ischemic Heart Disease: occurs when blood flow is restricted or reduced to the heart due to narrowing of arteries.¹

JDRF: Juvenile Diabetes Research Foundation

Ketones: produced from acetyl CoA when there is inadequate glucose in the cell to facilitate carbohydrate metabolism and energy production, to be used by the brain, heart, and muscle.¹

LDL: Low Density Lipoprotein: a small cell that is made up of a lipoprotein outside that surrounds a large amount of cholesterol inside, which is also labeled as the “bad” cholesterol.¹

Long acting insulin: a type of insulin used in a conventional regimen, with an onset of action being 2-4 hours, with no peak action

period, and a usual effective duration being 18-24 hrs depending on the mixture (Lantus or Levemir); BG should be monitored at 10-12 hrs.¹

Microalbuminuria: low, but abnormal albumin levels present in the urine (30-299 mg/24 hours) that is considered the earliest sign of clinical evidence for nephropathy.¹

Nephropathy: a microvascular disease that involves damage to the small blood vessels in the kidney allowing proteins to enter the urine and present with microalbuminuria caused by excess BG.¹

Neuropathy: a microvascular disease that involves peripheral nerve damage to feet and hands as well as autonomic nerve damage caused by excess BG in the small blood vessels.¹

OGTT: Oral Glucose Tolerance Test: A procedure used to diagnose diabetes that requires the patient to fast prior to testing and then ingesting a glucose solution at testing, followed by a blood glucose reading hours later. The protocol varies based on which version is used.¹

Oxidative Stress: stress in the body caused by an increase in oxidant generation, a decrease in antioxidant production, or a failure to repair oxidative damage.¹

Rapid acting insulin: a type of insulin used in both intensive and conventional regimens

that has an onset of action of less than 15 min, peak action at around 1-2 hrs, and an effective duration of 3-4 hrs; BG should be monitored at 2 hrs.¹

Retinopathy: a microvascular disease that involves the loss of retinal vasculature and ischemia with increased occurrence of hemorrhages, leading to increased ocular pressure and glaucoma formation, caused by excess BG in the small blood vessels.¹

ROS: Reactive Oxygen Species: free radicals, reactive anions containing oxygen atoms, or molecules containing oxygen atoms that can either produce free radicals or are chemically activated by them, which all cause cell damage as a result of oxidative stress.¹

Type 1 Diabetes: a disease characterized by high blood glucose concentrations in the body resulting from pancreatic β -cell destruction by an autoimmune response or idiopathic.¹

Type 2 Diabetes: a disease characterized by gradual elevation of blood glucose concentrations in the body resulting from genetic disposition and environmental factors such as family history, obesity, race, ethnicity, and physical inactivity.¹

VO₂ Max: the measure of the maximum volume of oxygen that an individual can use during exercise.⁶⁰

Appendix B

INFORMED CONSENT

Code_____

Effect of post-meal exercise on the glycemic effect of a meal: Type 1 diabetes **Consent to Participate in a Research Study** University of the Incarnate Word

You are being asked to participate in a research study conducted by Cindy Heiss, PhD, RD, LD. The purpose of this study is to determine if a 15 min bout of light exercise (walking) will minimize the glycemic response to a breakfast in people with type 1 diabetes. In other words, we want to know if the spike in your blood sugar can be minimized by walking after consuming carbohydrates. If this is the case, people with type 1 diabetes may be able to lower their insulin dosages, saving money and possibly reducing side effects, after consuming meals.

If you agree to take part in this study, you will participate in the following procedures:

You will report to room 318, Bonilla Science Hall at the University of the Incarnate Word on two different days at the usual time you consume breakfast for data collection having fasted overnight for at least 8 hours but no more than 12 hours. You will also not have exercised the day before each data collection day. Before coming to campus for data collection, you will need to have tested your blood glucose at home to determine if it is between 70 and 200mg/dl. If it is not in that range, you will contact the investigators by email (heiss@uiwtx.edu) or text/phone (805-766-9055) to reschedule.

1. On day 1 of data collection, you will complete a questionnaire to describe some demographic information such as your gender, age, and ethnicity, as well as how long you have had diabetes. In addition, your height and weight will be measured, and your body composition (percent body fat) will be determined using a bioelectrical impedance analyzer. This measurement involves having 2 electrodes placed on your hand and foot, followed by a non-detectable electrical current that will pass through your body. You will feel nothing, and the measurement takes only a few minutes. You will be given a data collection sheet with a code. You will need to record this code in your cell phone or on a card to keep in your wallet so we can give you the correct data collection form on the second day of data collection. You will measure your blood glucose using a glucometer twice. If the two measures are not within 10mg/dl, you will measure a 3rd time. You will record all measurements on a form provided to you. After obtaining your fasting blood glucose, you will give yourself your appropriate amount of insulin for a 41gm carbohydrate load, then consume 1 container of Boost supplemental beverage (which contains 240 calories and 41 gm carbohydrate. Then you will sit quietly and measure your blood glucose at 15, 30, 60, 90 and 120 minutes after you finished breakfast. You will then give your data collection sheet to the investigator.
2. On day 2 of data collection, you will again measure your fasting blood glucose as described earlier, give yourself your insulin dose, then eat the same beverage as before. Immediately after consumption, you will walk for 15 minutes at a light pace. You will wear a wrist band heart monitor and increase your pace until you achieve the heart rate

University of the Incarnate Word
 IRB Approved
 Application No. 16-01-002

provided to you by the investigators. You will maintain this heart rate until the 15 minutes is up. Then you will sit quietly for the remainder of the data collection. You will measure your blood glucose at 15, 30, 60, 90 and 120 minutes after you finished breakfast. You will then give your data collection sheet to the investigator. You may stay for a 180 minute reading if you wish to ensure your blood glucose is not too low, although this is not likely to happen with 15 minutes of light walking.

Note:

You will be instructed on how to measure blood glucose using the glucometer provided in this study. The measurement includes wiping a finger with an alcohol pad, then allowing the finger to dry. A special lance is then used to prick the finger for a small drop of blood. There will be a "pin-prick" sensation that may result in some slight discomfort. A glucose test strip is placed at the finger where the small drop of blood appears, then placed in the glucometer for determination of blood glucose level. The finger is then wiped again with the alcohol pad. Lancets, glucose test strips, and alcohol pads will be disposed of in a sharps container. Trained technicians and a registered nurse will be available to help with the blood glucose measurement if needed.

Each of the two days of participation will require approximately three hours, for a total time commitment of 6-7 hours for the overall study.

Before you can participate in this study, you will need to complete a screening questionnaire to determine if you are eligible to participate. To be eligible to participate in the study, you:

1. Must have type 1 diabetes and use an insulin pump or multiple daily injections (those who manage their diabetes with 2-3 daily insulin injections do not qualify).
2. Must be a non-pregnant healthy adult over the age of 18, weighing at least 110lb
3. Must have no allergies or intolerances to soy or milk
4. Must be willing to consume a breakfast consisting 1 container of Boost supplemental beverage, which provides 240 Calories and a 41 gram load of carbohydrate
5. Must know how much insulin to inject for this amount (41 grams) of carbohydrate
6. You cannot not have any condition that would render you unable to walk at a light pace for 15 minutes
7. You must be willing to fast for at least 8 hours, but no longer than 12 hours, prior to data collection
8. You will need to have an A1c reading of 7.5-9.5%. You will need to provide documentation of an A1c level of 7.5-9.5% measured in the past month. If you do not have that documentation, you will contact investigator Heiss (contact information below) to make an appointment to have your A1c measured. You will go to the UIW campus, room 302 for an appointment, and investigator Heiss will measure your A1c level, for which a drop of blood is needed. A special lance is then used to prick the finger for a small drop of blood. There will be a "pin-prick" sensation that may result in some slight discomfort. A kit is then used to determine A1c level.
9. You will agree to not exercise the day before each of the 2 data collection days.

As compensation for participating in this study, you will receive \$25 after completion of the first day, and another \$75 after completion of the second day.

University of the Incarnate Word
IRB Approved
Application No. 16-01-02

We will input the data from your questionnaires into a spreadsheet for analysis. Your name will not be included in the data spreadsheet, only your study "code number." All data will be confidential, and no copies will be maintained by the study investigator.

Your identity will be protected and any publication that follows this study will only display data of groups, not of individuals.

Participation is voluntary and you have the right to refuse participation without penalty of any kind. You have the right to stop participating at any time, without penalty of any kind. You have the right, at the end of the study, to be informed of the findings of this study.

If you have questions at any time, before, during, or after the study, please feel free to ask. If you have additional questions later or you wish to report a problem that may be related to this study, contact:

Cindy Heiss, PhD, RD, LD
210-829-3908
heiss@uiwtx.edu

To contact the University of the Incarnate Word committee that reviews and approves research with human subjects, the Institutional Review Board (IRB), and ask any questions about your rights as a research participant, call: UIW IRB, Office of Research Development (210) 805-3036.

If you completely understand the expectations and rights of participants in this study, all of your questions have been answered to your satisfaction, and you are willing to participate in this study please sign and date this consent form in the space provided. To sign this consent form, you must be 18-years-old or older by today's date.

Participant Signature

Date Signed

Witness Signature

Date Signed

Principle Investigator Signature

Date Signed

University of the Incarnate Word
IRB Approved
Application No. 16-01-002

Appendix C QUESTIONNAIRE

Screening Questionnaire Effect of post-meal exercise on the glycemic effect of a meal: Type 1 diabetes

Please answer the following questions by circling or writing your response.

1. Do you have type 1 diabetes? Yes No
2. Are you currently on an insulin pump? Yes No
If yes, what brand of pump? _____
If no, do you manage your blood glucose with multiple daily injections? Yes No
- (If you are on a set insulin injection regimen of 2-3 injections per day, then you do not qualify for this study, and do not have to fill out the remainder of the questionnaire.)
3. Do you know how much fast acting insulin you need for a 41 gram carbohydrate load? Yes No
4. Are you over the age of 18? Yes No
5. Are you pregnant or breastfeeding? Yes No
6. Do you weigh at least 110 pounds? Yes No
7. Are you in general good health? Yes No
8. Are you willing and able to walk 15 minutes? Yes No
9. Do you know your last A1c reading? Yes No
If yes, what was your last A1c reading? _____ Date measured _____
10. Do you have any allergies or intolerances that would make you unable to consume a Boost supplement beverage (Boost contains milk and soy ingredients. For a full list of ingredients, contact the researcher, Samantha Hinojosa by email at shinojo@student.uiwtx.edu or text/phone at (210) 260-5342 if you have any concerns)?
Yes No
11. Are you willing to consume a Boost supplement beverage on two mornings? Yes No
12. Do you know how much insulin you need to take for a 41 gram carbohydrate load? Yes No
13. Are you willing to give yourself a dosage of insulin to cover 41 grams of carbohydrate prior to consuming the Boost beverage? Yes No
14. Are you willing to fast for at least 8, but no longer than 12, hours prior to data collection, which will occur on 2 separate days?

Yes No

15. Are you willing to refrain from exercise the day before each day of data collection?

Yes No

16. Are you willing to refrain from strenuous exercise the day before data collection on both days?

Yes No

If you would like to participate in the study, please contact Samantha Hinojosa at slhinojo@student.uiwtx.edu so that your screening form can be collected (you can email the completed form to her if you wish, or ask to be provided with a self-addressed, stamped envelope to mail the screening questionnaire). If you meet the screening criteria from this questionnaire, we will arrange to measure your A1c if you can't provide documentation of an A1c between 7.5 and 9.5 in the past month as the final screening step if necessary. You will need to sign the Informed Consent Form prior to participation. If you are eligible for the study, you will be contacted with further information to set up data collection.

Your Name _____

Your Contact Information (indicate which is your preferred method for being contacted):

Address:

Phone:

Email

Appendix D

| |
|------|
| Code |
|------|

DATA SHEET

Effect of post-meal exercise on the glycemic effect of a meal: Type 1 diabetes

1. What is your gender? (circle one) male female
2. What is your age in years? _____
3. How many years have you had type 1 diabetes? _____
4. Which one or more of the following would you say is your race? Select all that apply.
____ White
____ Black
____ Asian
____ American Indian or Alaskan Native
____ Native Hawaiian or other Pacific Islander
____ Choose not to answer
5. Are you Hispanic or Latino? Yes No Choose not to answer

Glucometer Number _____

Anthropometric and Blood Glucose Data

Height _____ Weight _____

Day 1: If first two measurements are within 10 units, you don't have to do a third...

Fasting Blood Glucose: 1 _____ 2 _____ 3 _____

15 min blood glucose: 1 _____ 2 _____ 3 _____

30 min blood glucose: 1 _____ 2 _____ 3 _____

60 min blood glucose: 1 _____ 2 _____ 3 _____

90 min blood glucose: 1 _____ 2 _____ 3 _____

120 min blood glucose: 1 _____ 2 _____ 3 _____

Day 2:

Fasting Blood Glucose: 1 _____ 2 _____ 3 _____

15 min blood glucose: 1 _____ 2 _____ 3 _____

30 min blood glucose: 1 _____ 2 _____ 3 _____

60 min blood glucose: 1 _____ 2 _____ 3 _____

90 min blood glucose: 1 _____ 2 _____ 3 _____

120 min blood glucose: 1 _____ 2 _____ 3 _____