Diet composition quality of high, moderate, and low carbohydrate diets among subjects with Type 1 Diabetes

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Thesis

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Contents

Abstract

In type 1 daibetes there is the popular belief that a low-carbohydrate diet can lead to better glycemic outcomes. There is little research that captures glycemic results and looks at diet quality. This thesis aims to investigate if the low-carbohydrate diet does improve time in range and overall mean glucose for those with type 1 diabetes. It also aims to examine the diet quality of those who were following a low-carbohydrate/high fat diet. The T1DEXI data was examined using SPSS 29 and looked at means, 95% confidence intervals, and standard deviations. The key variables examined were time in range, mean percent glucose, and Healthy Eating Index (HEI) scores. We found that the group with the low-carbohydrate/high fat diet had the best glycemic results with more time in range and a lower mean glucose. We also found that the HEI scores throughout the low-carbohydrate, moderate-carbohydrate, and high-carbohydrate diets were within 1-2 points, showing little difference. Due to the similarities between the HEI scores and the improved glycemic outcomes we also examined insulin per kilogram and found that the amount of insulin was the same across the three diet groups despite the difference in carbohydrate intake. This led us to the conclusion that insulin/kg had played the largest role in glycemic outcomes. More randomized controlled studies looking at the impact of the low-carbohydrate diet's impact on type one diabetics needs to take place before certain conclusions are drawn.

Chapter 1: Specific Aims

Type 1 diabetes (T1D) is an autoimmune disease characterized by the loss of beta cells in the pancreas resulting in the loss of near complete or total loss of insulin secretion.[1] Currently, the medical nutrition therapy (MNT) recommendations for those diagnosed with TID includes practicing carbohydrate counting, eating an overall balanced diet, and monitoring blood glucose frequently.[2] People with TID use exogenous insulin in personalized carbohydrate-to-insulin ratios to manage blood glucose concentrations post-prandially.[3] Due to the impact of dietary carbohydrates on blood glucose and hemoglobin A1C (HbA1c) there has been an increased interest in consuming low-carbohydrate diets. Even with the increase in popularity, there has been very little data to support the use of a low-carbohydrate diet to improve glycemic control for those with TID.

Most of the low-carbohydrate diet research studies conducted have reported results among those who have been diagnosed with type two diabetes, or they focus on children with TID. There are a few studies reporting the impact of a low-carbohydrate diet on glycemic control among adults with TID. In addition, the few existing published studies do not use consistent definitions of a low-carbohydrate diet, and include diets with wide ranges of carbohydrate intake. In a case report assessing the efficacy and safety of long-term use of a ketogenic diet in an individual person with TID they define the ketogenic diet as less than 50 grams of carbohydrates a day.[4] In a real-world observational study looking at 42 adults with T1D they defined a ketogenic diet as one with less than 50 grams of carbohydrates per day.[5] In a recent systematic review of varying carbohydrate diets among those with TID, they defined three different levels of carbohydrate intake: very low carbohydrate diets, low carbohydrate diets, and

moderate carbohydrate diets. They considered a very low-carbohydrate diet to be 50 grams of carbohydrates or less per day, or less than 10% of their total daily calories. They defined the low-carbohydrate diet as 130 grams of carbohydrates or less a day, or under 26% of their total caloric intake. For moderate carbohydrate intake, they defined it as 130 to 230 grams of carbohydrates per day, or 26% to 45% of their total daily calories.[6] The majority of these studies include small sample sizes, unclear results, or have additional limitations, making interpretation of the evidence for low-carbohydrate diets in TID challenging. Data remains very limited regarding the use of a low-carbohydrate diet for those with TID.

Because of this gap in the literature, the goal of this project is to provide additional evidence on how low-carbohydrate and moderate-carbohydrate diets impact overall nutrient intake and glycemic control among people with diabetes. We proposed assessing how varying carbohydrate intakes affect overall diet quality. We further proposed investigating whether a moderate carbohydrate diet improves blood glucose control compared with a low carbohydrate diet among adults with TID.

This was a secondary analysis of the T1Dexi data. T1Dexi was a free-living observational study of people with TID randomized to various types of exercise: high intensity interval training, aerobic or resistance exercise. Glycemia was measured by continuous glucose monitoring (CGM). Dietary intake was measured by the remote food photography method using the T1Dexi app. Insulin dose was measured from insulin pump data or from Clipsulin captured insulin injections of those who manage their diabetes with multiple daily injections (MDI).

The specific aims of this project were:

Aim 1: Assess dietary quality among high, moderate, or low carbohydrate intakes using HEI scores.

Hypothesis: The normal and moderate will be at or above the average HEI score of 58, but the low carbohydrate diet will be below the average.

Aim 2: Compare total time in range (TIR) and mean glucose, between the high, moderate, and low carbohydrate intakes.

Hypothesis: Low and moderate carbohydrate intakes will have a greater TIR and lower mean glucose compared to high carbohydrate diets.

Identifying the impact of low-carbohydrate diets on both glycemia control and any associated nutrient insufficiencies that increase risk for chronic diseases will allow us to inform guidelines and recommendations for those with TID. This will allow clinical practices to inform patients on the answer of low-carbohydrate diets and the impact that they have on hemoglobin A1C.

Chapter 2: Background and Review of the Literature

Type 1 diabetes (T1D) is an autoimmune disease that can be characterized by the loss of pancreatic secretion of insulin, and when untreated, leads to hyperglycemia and death.[1] This is a chronic condition, and insulin replacement is required to maintain blood sugar levels. Because little to no insulin is being produced, glucose uptake into insulin-sensitive tissues to use for energy or stored for later use is substantially impaired.[7] The most common forms of diabetes management include exogenous insulin delivery, both basal insulin and bolus dosing for meals, with carbohydrate counting.[3]

Dietary intake, including carbohydrate counting, is an incredibly important factor in the management of T1D.[2] Consistent and well-balanced meals can make it easier to maintain euglycemia.[2] The insulin-to-carbohydrate ratio indicates how many grams of carbohydrates one unit of rapid-acting insulin can cover.[3] An insulin-to-carbohydrate ratio could be 1:10, meaning that 1 unit of insulin will cover 10 grams of carbohydrates. This ratio affects the insulin bolus taken.[8] Overall, more time in range reduces the complications that follow severe highs or lows of blood sugar levels. The goal TIR recommendation from the American Diabetes Association (ADA) is to have at least 70% of readings within the target range of around 70 to 180 mg/dL.[9]

Because of the large impact dietary carbohydrate intake plays into the management of T1D, there has been interest in alternative diets that could increase time in range. One of the diets that has gained interest is the low-carbohydrate diet, or the ketogenic diet. This diet is known not only for being low in carbohydrates, but also high in fat. While this diet has gained popularity in the diabetes community, few studies have reported glycemic outcomes among people with T1D. Current studies mostly focus on low-carbohydrate diets and do not assess moderate and high (regular) carbohydrate diets and their impacts. Much of the current research focuses on people with type two diabetes (T2D) and adolescents with T1D. The goal of this project is to evaluate how low, moderate, and high (regular) carbohydrate diets impact glycemia among people with T1D. We assessed diets with varying carbohydrate content, overall diet quality and glycemic outcomes.

2.1 Insulin and Glucose Metabolism

Insulin deficiency results in decreased glucose uptake into tissues and increased hepatic glucose output resulting in profound and prolonged hyperglycemia.[10] The liver works in

maintaining blood glucose levels by using glycogenolysis and gluconeogenesis.[11] The lack of insulin in T1D there are no signals that inhibit either process leading to elevated blood sugar levels. In muscle and heart tissues, insulin furthers glucose uptake through the translocation of GLUT4.[12] Insulin joins to its receptor on the cell exterior leading to the movement of GLUT4-containing vesicles to the plasma membrane.[12] GLUT4 allows glucose to enter the cells to either be used for energy or stored as glycogen. Adipose tissue requires insulin to stimulate GLUT4 to the cell membrane to facilitate glucose uptake as well.[12] Glucose can be converted to glycerol, which combines with fatty acids to form triglycerides. Insulin inhibits hepatic glucose production through deregulating gluconeogenesis and glycogenolysis, and by upregulating glycogen synthesis.[12]

2.2 Pathophysiology of T1D

T1D is the result of an autoimmune response that destroys the pancreatic β-cell and various genetic factors play a role in the risk of developing T1D.[13] T1D can develop at any age but tends to happen around a couple of peak ages: four to seven years, ten to fourteen years, and early adulthood.[13] The autoimmune response is activated by the β-cells which in turn interact with the T-cells that seek out and destroy the beta cells that secrete insulin.[13,14] The loss of pancreatic b-cells causes reliance on exogenous insulin for a person with T1D.[13,14]

T1D cause both acute and chronic complications if unmanaged. In the absence of adequate intracellular glucose, hepatocytes use fatty acids for energy and produce ketones that are released into systemic circulation. Extreme elevations of blood ketone concentrations can cause a life-threatening condition called diabetic ketoacidosis or DKA.[13] DKA is characterized by high blood acidity, dehydration, and electrolyte imbalances. Treatment of acute DKA may

require hospitalization, electrolyte and fluid resuscitation and insulin therapy.[11] Additionally, poor chronic management of T1D can lead to extended periods of hyperglycemia, and/or risk of insulin associated hypoglycemia.[11] Chronic hyperglycemia among people with T1D can damage blood vessels and nerves leading to diabetic retinopathy and neuropathy and cardiovascular disease.[11] People with T1D are also at risk for developing metabolic syndrome due to the complications created by their insulin deficiency.[11] Preventing these long-term complications and promoting normal glycemia is achieved with blood glucose monitoring and insulin therapy.[3]

2.3 T1D Management

The current management for TID includes monitoring blood glucose levels and providing exogenous insulin.[3] Blood glucose levels are monitored either by finger prick testing or with CGM. Finger-prick testing is a traditional method that uses a lancet to prick the fingertip to attain a drop of blood.[3] The blood is then deposited on a test strip, which is inserted into a glucometer. This method accurately measures blood glucose concentrations at the time of testing. CGMs offer continuous monitoring of blood glucose levels, typically measured every 5 minutes, providing insight into daily glucose fluctuations.[3] CGMs use a small sensor inserted under the skin that measures glucose levels in interstitial fluid and reports this data to a receiver. This allows CGMs to provide alerts for high and low interstitial glucose levels, detailed evaluation of time glucose is within the recommended range (70-180mg/dl), time above and time below the recommended range, and time above and time below the range over a 24-hour period.[3] The granular detail provided by CGM improves a patient's ability to adjust and manage insulin delivery and maintain better euglycemia.[15]

Insulin replacement is required for people with T1D and can be delivered via multiple daily injections (MDI) or insulin pumps. MDI involves injecting insulin multiple times throughout the day using a syringe or insulin pen. MDI typically uses a combination of long-acting insulin (basal) administered once or twice per day and rapid-acting insulin (bolus) administered with meals. MDI was the historical standard of insulin delivery for many years and remains the chosen delivery method for many patients with T1D.[3] However, insulin pumps have become the predominant method of insulin delivery among patients with T1D in recent years.[3,15] Insulin pumps are tiny, computerized devices that continuously deliver insulin throughout the day. Insulin pumps deliver a small, continuous basal dose of insulin via a catheter placed under the skin. Bolus doses can be programmed by the patient and administered at mealtimes or to counteract high blood sugar levels. This ability to couple insulin delivery with real-time glucose monitoring by CGM has improved overall glycemic control for patients with T1DM.[15] Despite these technological advancements, patients with T1D continue to struggle with glycemic control and with balancing insulin delivery with dietary intake, particularly carbohydrate intake.[16]

With effective management of T1D, a person can avoid extreme causes of hyperglycemia and hypoglycemia while maintaining optimal HbA1c levels. HbA1c, or glycated hemoglobin, is a key marker of lasting blood glucose control, reflecting average blood glucose levels over the past 2 to 3 months.[1,3] While insulin therapy is effective at lowering HbA1c, it can also increase the risk of hypoglycemia. Hypoglycemia arises when blood glucose levels fall below 70 mg/dL. In more severe cases, it can even drop down below 54 mg/dL, classified as severe hypoglycemia.[1,3] Frequent or severe hypoglycemia can have a great impact on the quality of life of people with

T1D. This is due to the not only to the danger that hypoglycemia can poise but also the mental burden of worrying about hypoglycemia.

2.4 Types of Carbohydrates:

Different types of dietary carbohydrates have differential impacts on post-prandial glycemia. For example, carbohydrates differ in their effects on post-prandial blood glucose concentration, with simple sugars and rapidly digestible starches producing the greatest glycemic rise, while dietary fiber has marginal impact. Sugars, including monosaccharides and disaccharides, are composed of one or two sugar molecules, are rapidly digested and contributed heavily to post-prandial glycemic responses.[2] Following ingestion of monosaccharides or disaccharides, in post-prandial glycemia, there would be a rapid spike leading to the need for exogenous insulin. Foods high in sugar tend to be high-glycemic index (GI) foods. They cause rapid glycemic elevations and significant glucose excursions.[17] Starches are composed of longer chains of sugar molecules and are digested more slowly. This leads to a slower, more sustained increase in glycemia. Most foods high in starches are going to have a lower GI impact, leading to more stable glucose excursions. Fiber is resistant to digestion and absorption, and thereby helps manage postprandial glycemia by regulating carbohydrate digestion and absorption. The two types of fiber are soluble and insoluble. Soluble fiber will slow the digestion and absorption of carbohydrates, which in turn leads to gradual rises in post-prandial glycemia. It can help flatten the post-prandial curve, reducing glucose excursions.[2] Insoluble fiber slows digestion by reducing glycemic spikes and glucose excursions.[2]

Protein also plays a significant role in glycemia levels. Protein has a moderate impact on post-prandial glycemic levels. It does not cause rapid spikes, but it can contribute to a gradual

increase. Dietary fats slow the digestion and absorption of carbohydrates, leading to a steadier rise in glycemic levels.[2] Given the impact that protein and fat have on glycemia, combining all macronutrients can help moderate post-prandial glucose excursions.[2]

2.5 Carb-To-Insulin Ratio

Current T1D management uses an individualized carb-to-insulin ratio (CIR) to determine the bolus insulin dose for specific meals. A person's CIR indicates one unit of insulin for a specific number of grams of carbohydrates. A patient would estimate the grams of carbohydrate in a meal, divided by their CIR and administer that number of units of insulin via an insulin pump or an injection. CIR is prescribed by the medical provider for that patient and is based on insulin sensitivity, activity level, and overall health.[18] Because CIR allows variable insulin dosing based on the size and carb content of the meal, this improves post-prandial glycemic control.[3,18] People with T1D often use tools such as food labels and mobile apps to estimate the carbohydrate content of their meals. Healthcare professionals such as dietitians often provide education on carbohydrate counting and how to improve estimations. Effectively mastering carbohydrate counting and using a CIR can allow for reduced health risks for those with T1D.[19]

2.6 Introduction to Low-Carbohydrate Diet

Because carbohydrate intake has a large impact on post-prandial glycemia, modifying the amount of carbohydrates consumed has been investigated as an alternative dietary approach for people with T1D. This includes consuming either a low-carbohydrate or moderate-carbohydrate diet. Altering the carbohydrate intake as such has an inverse relationship to fat intake causing high fat consumption. Specific restriction of an entire class of foods, such as carbohydrates, increases the chance for a poor diet quality that lacks essential micronutrients.

Because the impact of low-carbohydrate diets among people with T1D is still gathering evidence of the short-term and long-term impacts, the goal of this project was to assess how varying carbohydrate diets impact the overall diet quality and post-prandial glycemia among people with T1D. We examined three levels of fat intake: high, moderate, and low. For the remainder of this background, a high carbohydrate diet will be defined as 45 to 65% of daily caloric intake from carbohydrate; a low carbohydrate diet will be defined as less than 10% of total daily calories from carbohydrate. Current research comparing low and high-carbohydrate diets is reviewed below.

2.7 Low-Carbohydrate Diet Current Research

Several studies have investigated the impact of low-carbohydrate diets on clinical outcomes among participants with T1D. A cross-sectional study of 285 participants examined the relationships among carbohydrate intake, glycemic control, and cardiovascular health in adults with T1D, with participants scored by macronutrient intake. A score of 0-10 was assigned to each macronutrient based on intake. The particpants in the lower carbohydrate diet were consuming around 50% of their intake from fat and about 30% from carbohydrates. Participants who consumed a lower carbohydrate diet, defined as <30% of total daily energy, had better glycemic control, and a higher odds of not experiencing level 3 hypoglycemia, and higher odds of achieving recommended HbA1c targets without adverse hypoglycemic events. They also reported no adverse effects on participants' cardiovascular health, but noted that further studies are needed to assess long-term effects.[40] A mixed methods study evaluated the effect of the LCD on glycemic control and quality of life before and after a 12-week LCD (<100 g/d) and about 55% of intake from fat. Twenty-two participants completed the study, and an LCD

improved HbA1c but did not impact life quality. Additionally, the LCD induced weight loss and decreased daily bolus insulin needs without adverse events.[20]

A randomized crossover study that had 15 participants with T1D compared the effects of three different diets: high protein/ low carbohydrate (20% of daily calories from carbohydrates, 40% of daily calories from fat), Mediterranean/ low GI (40% of daily calories from carbohydrates, 35% of daily calories from fat), and a reference diet (50% of daily calories from carbohydrates, 30% of daily calories from fat). Each arm lasted 3 weeks, with a 7-day washout period between dietary interventions, totaling 9 weeks for each participant. They found that the LCD performed better than the reference diet in regard to time spent in range; 73.3% of participants spent time with their glucose levels within 70-140.[21] A 12 week non-randomized single-arm design trial compared the effects of a professional supporteded LCD (25-75 g/day of digestible carbohydrates and about 60% of intake from fats) with diets higher in carbohydrates on clinical markers including HbA1c, glycemic variability, frequency of hypoglycemia, total daily insulin, and quality of life. The participants included 20 adults and found that HbA1c levels reduced by 0.6 + 0.7% from post-control to post-intervention, and total daily insulin dosing was reduced by 16 + 11 U/day.[22] While these four studies showed improved glycemia, they also were limited by the short duration of the studies, and further research is needed to verify that low-carbohydrate diets could improve glycemic control.

Many of the studies about the low-carbohydrate diets had very small sample sizes with the potential for bias. A retrospective, real-life study collected data from 33 participants with T1D who switched to a eucaloric very low-carb diet (EVLCD) from their usual diet for a period of 12 months. The EVLCD diet is defined as a diet with a daily caloric intake equal to total daily

energy expenditure (TDEE) and a carbohydrate content of <50 g/day with 70% of daily intake coming from fat. This study found a mean reduction of HbA1c from 8.34 ± 1.73% to 6.8 + 0.78%.[23]

Several reviews and meta-analyses have evaluated evidence for low carbohydrate diets among T1D. These articles call out the lack of randomized controlled trials comparing LCD with high carbohydrate diets among people with T1D but claim that LCD's could have beneficial effects when practiced in a short-term setting.[24,25] Other authors suggest a LCD diet may be helpful for some but not all people with T1D but they would need to be carefully selected and monitored when using this diet as a management tool. Many reviews call for further research, stating the current research is inconclusive.[26] Specifically, the need for high-quality randomized controlled trials is needed to make a conclusion about the efficacy of LCD for T1D management.

Potential harms of an LCD must also be considered, and several publications suggest a low-carbohydrate (ketogenic) diet might increase the risk of developing an eating disorder.[27] These articles bring forth risks that have not yet been evaluated from a psychological standpoint. Other potential harms include restriction of micronutrients typically consumed with carbohydrates that might increase the risk of nutrient deficiencies and the potential to compromise a person with T1D's cardiac health due to the high fat intake.[28]

A handful of studies were conducted among pediatric patients instead of adults with T1D. Some studies also provided valuable contextual information, including parents' experiences with using a low-carbohydrate diet as a management strategy for their child with T1D. Parents reported preferring LCD for their child, feeling less worried and sleeping better,

decreasing their overall anxiety surrounding their child. Authors of this study push for more education surrounding this topic to be available to families who have a child with T1D and for more education surrounding T1D in general.[29] In contrast, a single case report found that the use of an LCD for a child with T1D may have a negative impact on growth and cardiovascular health. The case in the report followed a LCD diet that consisted of a mean of 1342 kcal/d, 23% carbohydrates, 23% protein, and 54% fat for two years. The participant experienced growth retardation and developed dyslipidemia, which increases the risk of cardiovascular disease.[30]

Another review mentioned how even if a child has epilepsy and T1D, there is not enough literature to fully back a low-carbohydrate diet as completely safe and recommends treating each patient individually.[31] One study found that children following the LCD had slightly better glycemic control compared to the control group on a normal carbohydrate diet, with improved time in range.[32] These improvements were modest at best and the overall outcomes regarding their HbA1c were neutral. While this one study offered a slightly positive impact, there were several studies that highlighted the challenges of implementing an LCD with children. Another study emphasized the need for clinical protocols to ensure the medical safety of children following a LCD.[33]

Few studies have explored the practicality and impact of LCD in children and adolescents with T1D. One study that tried to implement an LCD was met with almost no compliance with following an LCD.[34] These studies also pointed out the potential risks associated with LCD, such as developing disordered eating, nutrient deficiencies, and negative long-term impacts on cardiovascular health. While a few of these studies did list some benefits, more time in range

and better glycemic control, they also were much more upfront the potential harm this diet could do to do children with TID.

A meta-analysis and systematic review of the safety of a carbohydrate-restricted diet summarized several trials. The review included 9 randomized studies and 143 participants, aged 15.5 to 47.2. The definition of LCD differed, with one being 60-80 g/d, another 25% carbohydrates, and the others being <100 g/d. This review found that in the 9 studies that they looked at, carbohydrate restriction did increase time in range and found that low-carbohydrate diets were associated with weight loss and reduced insulin use. However, even with the results, the review warned that they may be skewed by the limitations of the randomized controlled trials included.[35]

One study looked at the impact of low-carbohydrate diets and their impact on micronutrient intake among adolescents with T1D. They aimed to provide 50-80 g/day of carbohydrates for each patient and about 55% of daily intake coming from fat. This study found that while a low-carbohydrate diet did lower the amount of processed food intake and lower BMI, they also found that there was a decrease in iron, calcium, vitamin B1, and folate intake with the low-carbohydrate intervention. They concluded that if a person with T1D were to follow a low-carbohydrate diet, they would need frequent monitoring and guidance by medical professionals.[36]

One of the key limitations of this literature is the inconsistent definition of an LCD. Some studies used less than fifty grams of carbohydrates a day, while others included up to one hundred and thirty grams of carbohydrates a day.[23,37] Most of these studies also failed to

examine any micronutrient intakes and overall diet adequacy. It does seem there is potential for better glycemic control and fewer insulin boluses when following a low-carbohydrate diet.[21,23,25,35,38-41] There are also the potential risks, including the increased chance of developing an eating disorder, potential exacerbation of cardiovascular disease, growth inhibition in children, and the potential for the diet itself to be overall inadequate.[23,24,28,35-37,40,42-44]

2.8 Moderate Carbohydrate Diet Current Research

While the moderate carbohydrate diet would be less restrictive than the low carbohydrate diet it may offer some of the same benefits. In a randomized, crossover study, 50 participants with T1D followed two diets, moderate and traditional carbohydrate diets, for 4 weeks each, separated by a 4-week washout period. The moderate carbohydrate diet was defined as having around 30% of total daily caloric intake from carbohydrates, while the traditional diet was defined as having 40% of total daily caloric intake from carbohydrates. The results showed an overall lack of adherence to the diets, resulting in only a 30-gram difference in carbohydrate intake between the diets. A difference in mean glucose of 0.6 mmol/L favored the moderate carbohydrate diet.[45] Because of the lack of research surrounding a more moderate carbohydrate diet, it is possible that results would look somewhat like the benefits that were being seen with the low-carbohydrate diet. The diet is less restrictive, potentially reducing risk for disordered eating, nutrient deficiencies, or cardiovascular disease. This gap is where this project will help provide some perspective on a more moderate approach to restricting carbohydrates for people with TID.

2.9 High Carbohydrate Diet Current Research

While there were almost no studies on a moderate-carbohydrate diet, a few compare an LCD to a high- or regular-carbohydrate diet. A 12-week randomized study compared the effects of an LCD and a high carbohydrate diet on glycemic control and cardiovascular risks. The LCD was defined as limiting daily carbohydrate intake to under 100 g/d while the high carbohydrate diet was defined as over 250 g/d. There was a total of 14 participants, and they found that the time spent in the range did not differ between the LCD and the HCD groups.[41] A randomized 2-period crossover design study had a 12-week intervention where they assigned 10 different participants either an LCD (<100 g/d) or a high-carbohydrate diet (>250 g/d). They compared the effects of each diet on the participants' lipidome. They found that 11 lipid species changed between the two diets, and that all 11 were elevated during the LCD. In the LCD, there was an increase in sphingomyelins and phosphatidylcholines, which the authors imply are thought to reduce dyslipidemia.[46] With both of these studies looking at a low-carbohydrate diet having additional benefits for people with T1D, there needs to be more research done to determine which carbohydrate-modified diet would be the most effective, realistic, and beneficial out of the three.

The studies highlighted above suggest potential benefit for a low carbohydrate diet for people with T1D, but many gaps in the literature remain. With the proposed benefits that are mentioned in some of the studies on the low-carbohydrate diet's impact on people with TID, it would be worth it to complete more research looking at restricted-carbohydrate diets. It would also be worth making sure that each diet's micronutrient content and fiber are within the National Institute of Medicine's Acceptable Macronutrient Distribution Range (AMDR) and meet

the recommendations for micronutrient intake. Filling these gaps could help bring awareness to the gaps that are currently being overlooked when looking at low carbohydrate diets for people with TID. This would allow researchers to determine whether the diets should be recommended, not only based on glycemic control and blood work, but also on their composition and whether they are nutritionally insufficient.

Chapter 3: Methods

We performed a secondary analysis of a publicly available data set, the Type 1 Diabetes and Exercise Initiative (T1DEXI). This study included adult participants with T1D. Participants used the T1DEXI smartphone application that was cloud-connected to enter health information about sleep, physical activities, and report food intake. Participants used a CGM, and interstitial glucose levels were measured and collated via a cloud-based data collection system. Participants also had an activity watch to monitor physical activity and heart rate monitoring, a Verily watch. Data from the watch was also aligned via a cloud-based data collection system. The activity entries included time of day, duration, activity type, intensity rating, whether the activity was competitive, and the timing since the last meal. The food intake data included categorical estimates of carbohydrates, fats, and protein, as well as meal size, and required participants to capture a photograph of the food consumed. A trained human rater used a computer-assisted method to identify foods and amounts in images. Participants used either their personal Dexcom C6 CGM or a blinded Dexcom C6 CGM, along with a Verily Study Watch, to continuously monitor heart rate. One of three exercises (aerobic, resistance, and interval) was assigned to participants and was completed following video instruction for at least 6

sessions over the 4-week observation. During exercise, participants wore a Polar H10 chest strap heart rate monitor. The inclusion criteria included: 18 to 70 years of age, use of MDI or an insulin pump, and a diagnosis of T1D of at least 2 years.

3.1 Primary Study

In the T1-Dexi, participants learned how to use the app to record their exercise and, before and after meals, their food intake using photography. They were instructed to rank their meals as low, typical, or high in protein; low, typical, or high in fat; and small, medium, or large in meal size. They also provided an estimation of carbohydrates in grams for each meal. Tidepool/Medtronic was used to collect meal insulin bolus data if the participant used an insulin pump. Clipsulin, an insulin dose recorder, or written records were used by an MDI user. The Ingestive Behavior Laboratory at the Pennington Biomedical Research Center analyzed 2,731 photos of meals that were submitted using the Remote Food Photography Method© (RFPM©).

3.2 CGM

Participants in this study used their personal CGMs (50% Dexcom; 10% Medtronic, 2% Abbott) and those who were not CGM users received a blinded CGM (38% Dexcom G4 with 505 or G5). CGM captured interstitial glucose concentration every 5 minutes. Results were captured via a cloud-based system and aligned in a central data center. We will include days for which at least 18 hours of CGM data are available; days with >6 hours of missing glucose data will be excluded. Time in range will be calculated as the number of glucose readings between 70-140 mg/dL total number of readings. Mean glucose will be the average of all CGM readings for the 24-hour period.

3.3 Food Photography

Participants used their smartphones to capture images of their meals and snacks before and after consuming food. Participants were given a reference card before data collection as a measurement reference point, instructing participants to capture their images at an arm's distance away and at a 45-degree angle. For each meal, the two pictures were collected and sent to the data collection server for assessment. The photos were analyzed by a trained human rater using proprietary software that matched pictures to images of food. The rater reviewed the images, identify the foods, and estimate the amount consumed using the photo database. The food type and amounts were analyzed using the nutrient source in the Food and Nutrient Database for Dietary Studies.[47] Daily records that included 2 or less recorded meals/photos, were excluded as underreported in the main study.

3.4 Statistical Methods

Statistical software, SPSS 29, was used for analyses. Key variables are summarized using mean, standard deviation, and 95% confidence intervals. The primary dependent variables include percent glucose time in range and mean glucose levels. The population was divided into groups based on fat intake levels.

Dietary records are often inaccurate. We used the Goldberg cutoff method to test for underreporting and exclude inaccurate data. We estimated basal metabolic rate for each participant using the Schofield equation. We then calculated the reported kcal intake/ estimated BMR. Exclusions were any day with a reported kcal intake/basal metabolic rate (BMR) ratio less than 1.2 and greater than 2.4.[42] Participants with a kcal intake/BMR between 1.2 and 2.4 and 18 hours or more of CGM data are included. The final dataset includes time and range and mean of

all data glucose points. Potential confounding variables include physical activity, baseline HbA1c, use of other medications, insulin delivery method, and meal timing.

We focused on fat intake due to the popularity of the ketogenic diet and the correlation between high fat intake and low carbohydrate intake. The total population was divided into low, moderate and normal fat intake. SPSS was used to divide the total population into three tertiles based on their fat intake. Tertile 1 was the highest fat intake with a maximum of 69.26% and a minimum of 46.46% of total fat intake. Tertile 3 was the lowest fat intake with a maximum of 40.42% and a minimum of 23.27% of total intake. Moderate carbohydrate intake is defined as any carbohydrate intake that falls between the two groups. A one-way ANOVA was used to compare the diet quality (HEI score), TIR and mean blood glucose between these 3 groups.

Descriptive Statistics were used to summarize the glycemic control metrics for each carbohydrate intake group. We compared our primary outcome variables: time in range, and average glucose by tertiles using a one-way ANOVA test with a post-hoc. Box plots were created to visualize the differences between tertiles for each outcome. We conducted another analysis to look at the 13 individual HEI factors. All 13 factors were averaged such as the participants data and graphed on a radar plot to see the key differences in scores

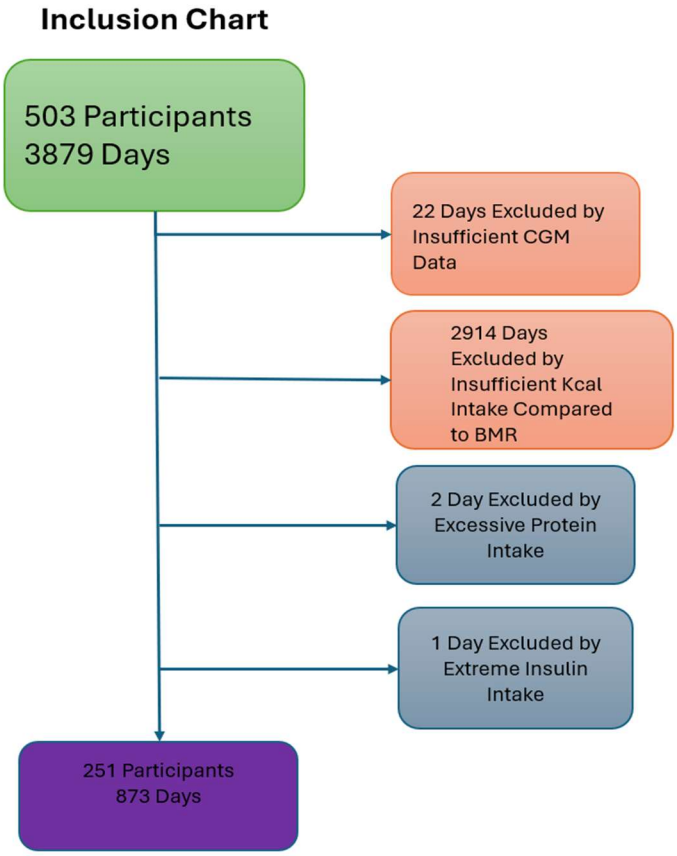
Chapter 4: Results

4.1 Inclusion Criteria

The complete T1Dexi data had 503 participants with a total of 3879 days of dietary intake data. Exclusion criteria includes insuffiecient caloric intake per the Goldberg cutoff for BMR, insuffiecient CGM data, excessive protein intake, and extreme insulin intake. Dietary

intake of total energy <1.3 estimated BMR was excluded as underreported leading to exclusion of 2914 days of dietary data. Days with <18 hours of CGM data were excluded due to excessive missing glycemic data leading to removal of a total of 25 days of data leaving the final dataset with 251 participants and 873 days of data (Figure 1). Individual participants had variable numbers of data in the whole dataset and all days of data for an individual participant were averaged to create the participant mean values (Table 1).

Table 1: Inclusion Chart



4.2 Comparison of Demographics in Each Tertile

The participant means were divided into tertiles based on the percentage of energy from fat consumed (Table 2).

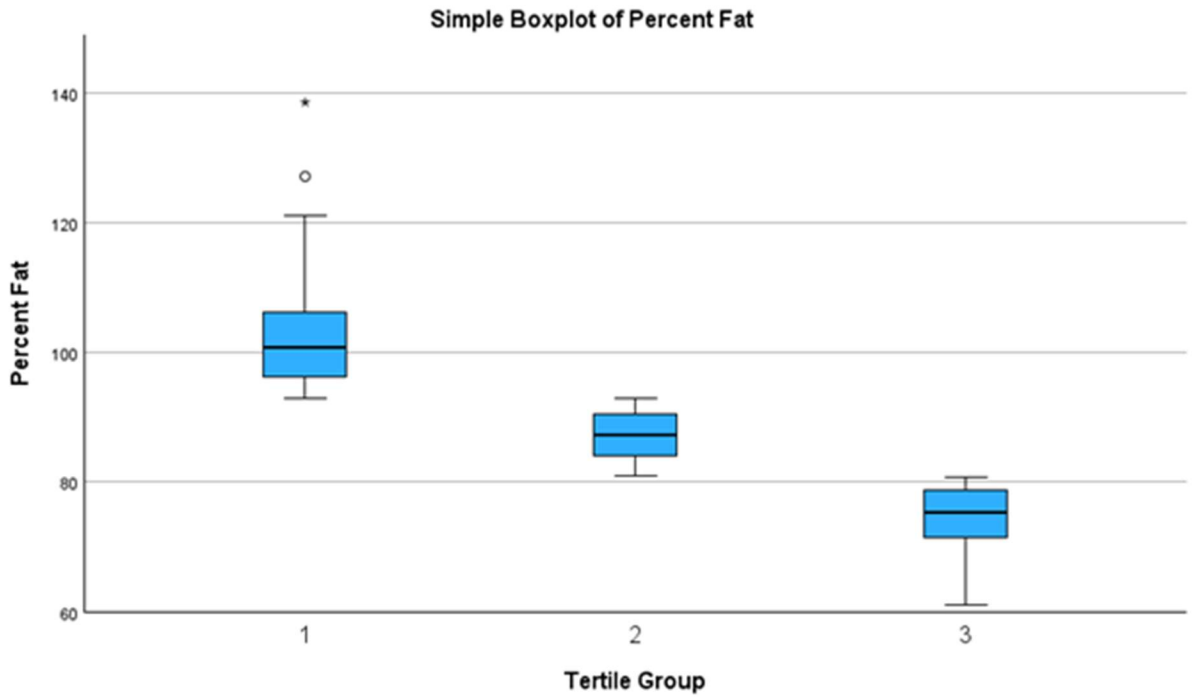
Table 2: Demographics

Demographics

Variable	Tertile 1	Tertile 2	Tertile 3	Total
HEIGHT (inches)				
Mean	66.33	66.50	66.40	66.41
SD	3.334	3.233	3.560	3.375
Min	58	58	59	58
Max	76	75	75	76
WEIGHT (kg)				
Mean	72.40	71.86	70.72	71.66
SD	13.21	11.75	11.78	12.24
Min	46.82	50.91	51.65	46.82
Max	114.55	104.09	104.55	114.55
BMI (kg/m^2)				
Mean	25.22	25.13	24.76	25.04
SD	3.96	3.28	3.36	3.54
Min	18.28	19.98	19.78	18.28
Max	35.58	34.35	38.35	38.35
SEX				
Female n (%)	63 (75.9%)	65 (77.4%)	59 (71.1%)	187 (74.8%)
Male n (%)	20 (24.1%)	19 (22.6%)	24 (28.9%)	63 (25.2%)
EXERCISE TYPE				
Aerobic n (%)	28 (33.7%)	28 (33.3%)	33 (39.8%)	89 (35.6%)
Interval n (%)	24 (28.9%)	30 (35.7%)	24 (28.9%)	78 (31.2%)
Resistance n (%)	31 (37.3%)	26 (31.0%)	26 (31.3%)	83 (33.2%)
AGE AT ENROLLMENT (years)				
Mean	40.20	37.79	35.28	37.76
SD	13.46	14.12	14.40	14.09
Min	20	19	19	19
Max	67	69	69	69
DIABETES DURATION (years)				
Mean	20.37	19.81	17.02	19.07
SD	11.97	13.18	9.27	11.64
Min	1	1	0.5	0.5
Max	56	66	44	66

Tertile 1 is the highest percentage of fat consumed while tertile 3 is the lowest amount of fat percentage consumed (Figure 1).

Figure 1: Percent Fat From Total Caloric Intake



Tertile 1 includes 83 participants with 63 being female and 20 being male. Tertile 2 includes 84 participants with 65 being female and 19 male. Tertile 3 includes 83 participants with 59 being female and 24 male. Demographic characteristics were similar between tertiles.

Table 3: Macronutrient Percent by Tertile

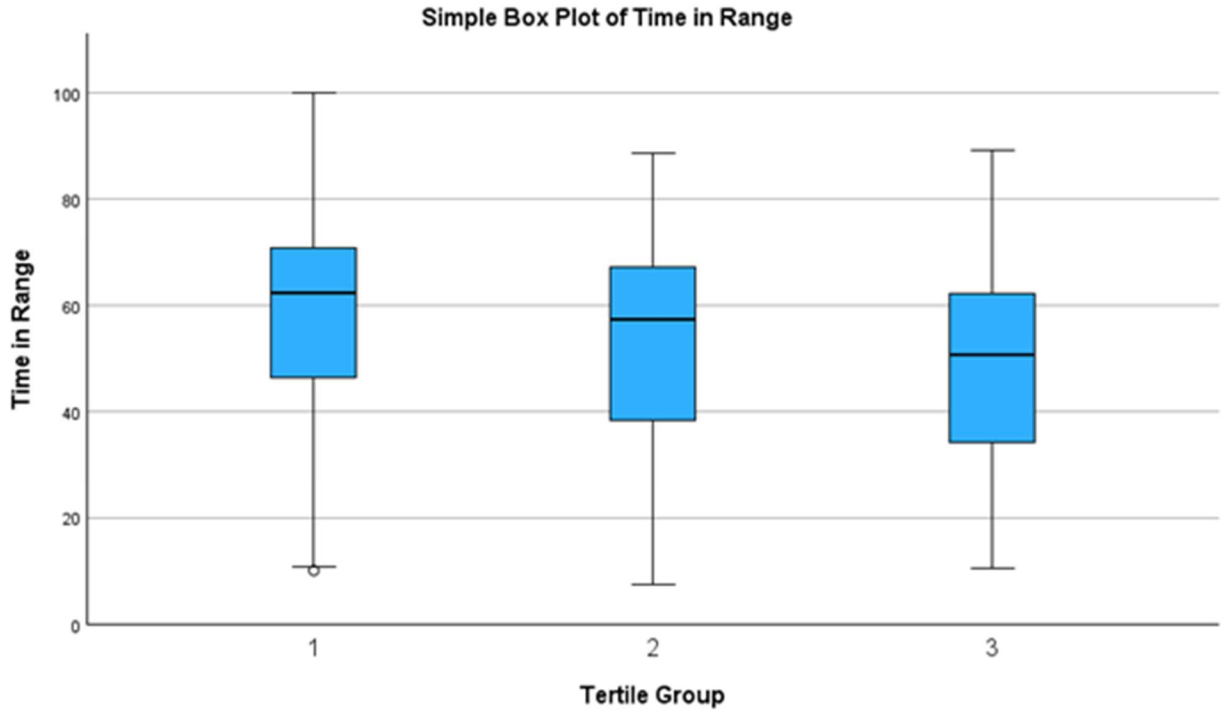
Macro Mean	Tertile 1	Tertile 2	Tertile 3	Total
Protein	17.49	16.64	16.14	16.75
CHO	31.99	40.67	47.07	39.91
Fat	51.35	43.51	36.81	43.89

4.3 Glycemic Outcomes

There is a pattern of decreasing time in range across the tertiles. Tertile 1 has a mean time in range of 59.19 while Tertile 3 has a mean of 49.23. The standard deviations are similar

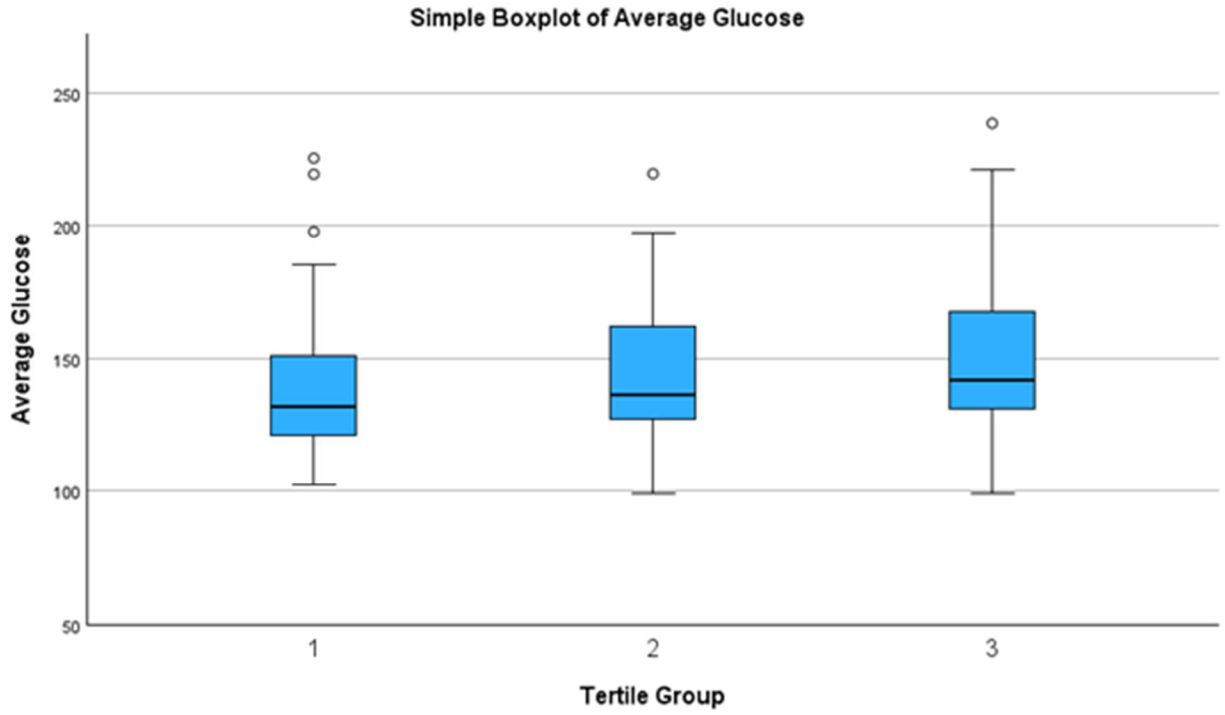
throughout the tertiles staying around 18-20. Total time in range was significantly different between Tertile 1 and 3 with Tertile 1 having more time in range (p-value of 0.003; figure 2).

Figure 2: Time in Range by Tertile



Mean Average Glucose (mg/dL) increases across the tertiles, which is opposite to the Time In Range results. Tertile 1 has a mean average glucose of 138.61 while tertile 3 has a mean of 147.68. There are higher medians and upper quartiles in Tertile 3 which suggests that participants in tertile 3 had higher average glucose (Figure 3).

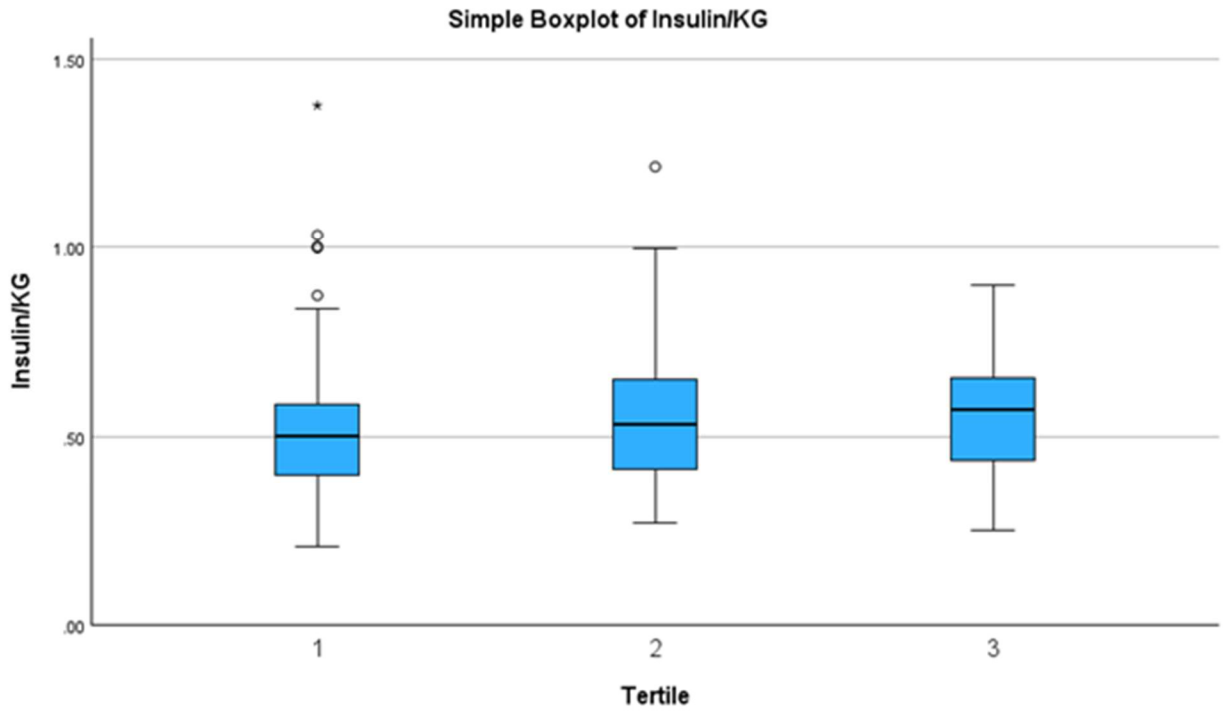
Figure 3: Average Glucose by Tertile



Participants who consumed a higher percentage of energy from carbohydrates in tertile 3 had less time in range and a higher mean average glucose.

An essential component of managing glycemia for patients with T1D is the carb to insulin ration or the insulin on board. We examined insulin per kilogram of body weight per day as a measure of insulin on board. Insulin per Kilogram showed no differences across the three tertiles (Figure 4, p=0.458).

Figure 4: Insulin Over Kilogram by Tertile



Even with the reduction in carbohydrate intake and higher fat intake, the overall amount of insulin administered was the same. For participants in Tertile 1 with the lower carbohydrate and high fat intake, a similar amount of insulin was administered as Tertile 3 with the higher carbohydrate and low fat intake, suggesting a higher carbohydrate-to-insulin ratio.

4.5 Mixed-Effects Model

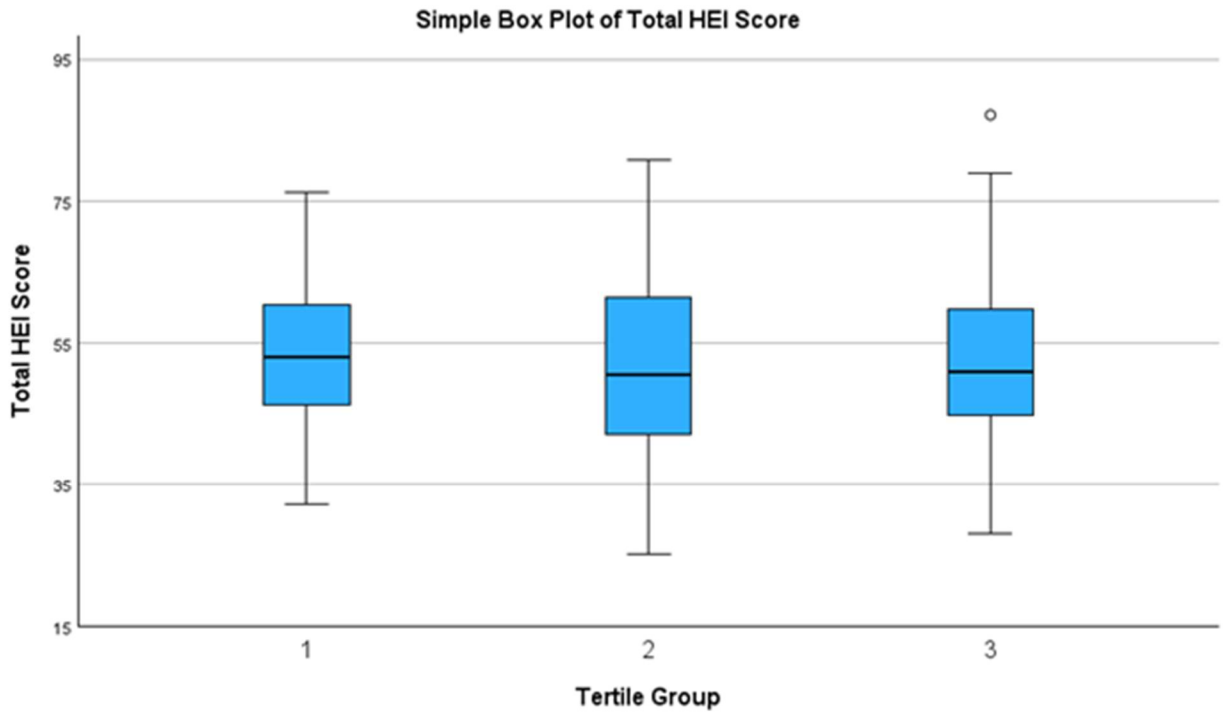
A mixed-effects model was also conducted as a secondary statistical approach that includes each individual day for each participant and not a participant's averaged total. Because there are multiple days per participant, the observations are not independent. The fixed effects in the final model include percent calories from fat, insulin/kg, BMI, HEI score, and the length of time since a T1DM diagnosis. Random effects are participant and the dependent variables

include time in range, and average glucose. The results from this test did not provide any additional explanations compared to our participant mean analysis above.

4.6 HEI Scores

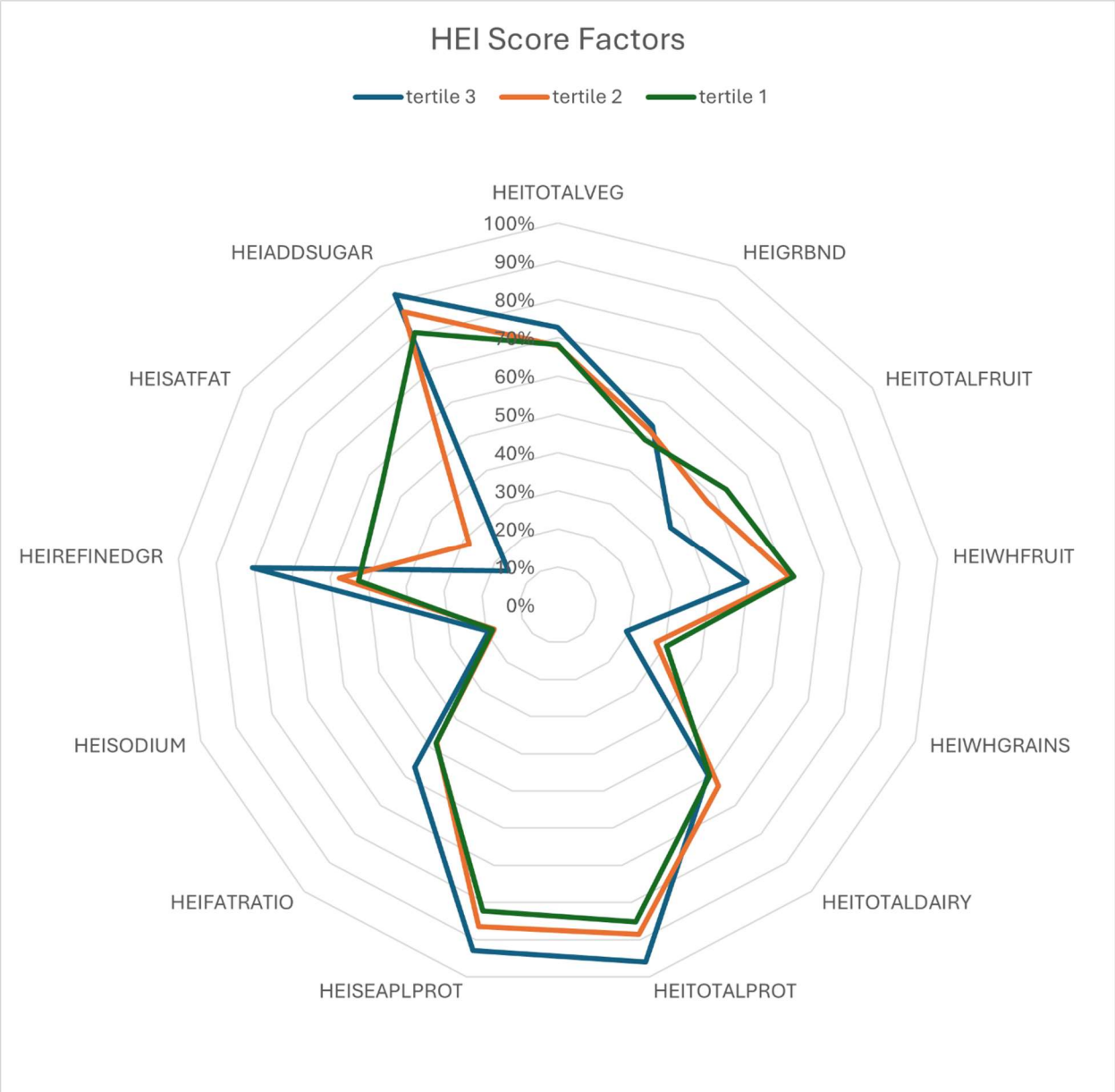
Despite differences in percent of energy from fat, HEI Scores were relatively similar across the tertiles with only a difference of 1 to 2 points. The standard deviations did have a moderate spread ranging from 9.8 ± 12.5 points (Figure 5).

Figure 5: Total HEI Score by Tertile



There are not many notable differences between the tertiles in the radar plot other than a slightly higher saturated fat intake with Tertile 1 and Tertile 3 having a higher intake of refined grains. Despite differences in macronutrient content, HEI scores were similar across tertiles.Between all three tertiles there was less than 1 point of difference (Figure 5), with the mean for tertile 1 being 53.25, tertile two being X, and tertile 3 being 52.39

Figure 6: HEI Score Factors by Tertile



Chapter 5: Discussion

The overall goal of this project is to examine whether dietary fat intake in persons with type 1 diabetes is associated with glycemic outcomes. The glycemic outcomes are described as time in range, and mean glucose. We also looked at the average HEI score for each tertile

showing us the overall diet quality. The final dataset was broken down into 3 tertiles based on the percentage of total energy from fat. In essence, there was a low fat-intake group, a moderate fat-intake group, and a high-fat intake group. Tertile 1 had the highest fat intake at a mean of 51.35% while Tertile 3 had the lowest fat intake at a mean of 36.81%. None of the tertiles align with current dietary recommendations which suggest a range of 20 ± 35% of daily caloric intake come from fat sources. Analysis was conducted for both participants mean values and mixed-effects models using individual day data. The results showed that Tertile 1, with the highest fat intake, had better overall glycemic outcomes with almost the same HEI score as Tertile 3, the low-fat group. These findings opposed what we were expecting to see in the results by showing that all three tertiles had a similar HEI score that was close to the average score. The findings did confirm what we expected to see regarding the better glycemic outcomes for the LCD/high fat diet.

5.1 Insulin Intake Per Kilogram

As shown above the insulin on board was the same for Tertile 1 and Tertile 3. Even with the lower carbohydrate and higher fat intake Tertile 1, participants in this group were administering the same amount of insulin per kilogram. This leads to more questions such as, if Tertile 3 had adminstered more insulin to account for their higher carbohydrate intake would the glycemic outcomes have been more aligned with Tertile 1? The only significant factor in the mixed-effects model was insulin on board. In this small data set, insulin administration is driving factor associated with glycemic outcomes.

5.2 Interpretations of the Findings

The tertile differences in time in range and mean glucose could reflect behavioral or physiological patterns but the small effect sizes suggest that the differences are not large. Before completing the results we believed that even if we saw better glycemic outcomes for Tertile 1 they would be minor and would result in a more poor HEI score. The results showed a different outcome of improved glycemic outcomes with an HEI score that was the same as Tertile 3. All three tertiles scored amount the same, around the average for Americans: 58.[48]

It was assumed that the HEI score would be poor for Tertile 1 due to the macronutrient composition. When eating high fat that allows little room for whole grains, whole fruits, and other complex carbohydrates that add to the overall HEI score. It also typically leads to higher consumption saturated fat and trans fat. Tertile 3 would be assumed to have a higher HEI score due to having more carbohydrates being consumed allowing for more whole fruit and complex carbohydrate consumption. Due to Tertile 1 consuming around 51% of their daily caloric intake from fat and Tertile 3 consuming around 36% of their daily intake from fat, it was surprising that the HEI scores could be so similar across tertiles (Figure 5). These assumptions ended up being wrong with both of these groups with all three tertiles scoring similarly throughout the HEI score factors. Tertile 1 did have a higher saturated fat intake, but Tertile 3 had a higher refined carbohydrate intake which seemed to offset the difference between the two tertiles.

There was also the expectation that the moderate intake group would have higher time in range which ended up being the group with the highest fat intake. The highest fat intake group (Tertile 1) had the highest time in range: 59.19%, and the the lowest mean glucose:

138.61 mg/dL. These results counter my initial assumptions and challenge the idea that lower intake of fat corresponds to better diet quality or better glycemic control.

Higher fat intake is usually associated with the increased risk of heart disease. When doing the research into the literature surrounding this topic there was little to no research on the long-term impact of someone with T1D consuming a high fat diet long-term. While fats are a critical factor in overall health having a diet heavy in saturated fats can lead to an increase in LDL cholesterol. With this increase there is a greater risk of heart disease and other chronic cardiovascular diseases. While the results showed that the higher fat diet led to better glycemic outcomes there is the long-term risk of developing heart disease due to the higher consumption of fat.

5.2 Strengths and Limitations

The uses of both participant-level and day-level analyses increase robustness and allows for a decent sample size to be utilized. Good technology was used in the process of capturing the data. This included a custom smart phone application, which allowed participants to enter information food intake, exercise intensity, and meal timing. This study may be limited by how it is just one snapshot of time, missing data, and differences in days between one participant.

5.3 Implications

Insulin management should remain the primary driver of short term glycemic control. In this dataset, the percent of calories form fat was not a significant predictor of day to day glycemic outcomes once insulin on board was included in the mixed-effects models. There needs to be more research completed on the long-term impact of a high fat diet in this

demographic and if heart disease should be a risk factor that is considered when using this approach for glycemic control.

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