BARRIERS TO TREATMENT ADHERENCE AND HEMOGLOBIN A1C IN DIABETIC POPULATIONS

by

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LIST OF ABBREVIATIONS

Abbreviation	Description
ADA	American Diabetes Association
ADS	Appraisal of Diabetes Scale
CBG	Controlled Blood Glucose
CDC	Centers for Disease Control
EUROHIS-QOL-8	EUROHIS Quality of Life Index
hbA1c	Hemoglobin A1c
HPA	Hypothalamus-Pituitary Adrenal (axis)
ISEL-8	Interpersonal Support Evaluation Scale
MARS-D	Medication Adherence Rating Scale
PHQ	Patient Health Questionnaire
PSS	Perceived Stress Scale
T1D	Type 1 Diabetes
T2D	Type 2 Diabetes
UBG	Uncontrolled Blood Glucose
VAS	Visual Analogue Scale for Medication Adherence

ABSTRACT

Diabetes mellitus is a disease characterized by decreased function or complete dysfunction of islet β cells in the pancreas. Failure to properly manage the disease may result in numerous adverse health complications, which include ketoacidosis, organ failure, cataracts, neuropathy, seizures, limb amputation, pregnancy complications, and even death. With increasing prevalence rates of the disease, it is important for patients with diabetes to adhere to their recommended treatment to avoid these complications. Unfortunately, a substantial amount of diabetic patients fail to adhere to their recommended treatment, which results in elevated blood glucose, and puts them at an increased risk for adverse health effects. One of the main obstacles in evaluating diabetes treatment adherence has been measuring what factors actually reflect adherence and controlled hbA1c. Many barriers may affect adherence; however, using a holistic view of barriers that contribute to non-adherence and uncontrolled glycemic index in diabetic patients may help to identify relevant barriers that prevent adequate health. The purpose of this study is to identify barriers that prevent proper treatment adherence and controlled hbAlc levels in chronic diabetic patients. A total of eighty-five type 1 and type 2 diabetic patients completed the study survey by responding to demographic questions and completing the Medication Adherence Rating Scale (MARS-D), the Barriers to Medication Adherence Questionnaire, the Visual Analogue Scale for Medication Adherence (VAS), the Perceived Stress Scale (PSS), the Interpersonal Support Evaluation List (ISEL-12), the Appraisal of Diabetes Scale (ADS), the EUROHIS

Quality of Life Index (EUROHIS-QOL-8), and the Patient Health Questionnaires (PHQ) to assess treatment adherence and to identify potential barriers that affect said adherence. Patients were categorized having either controlled (CBG) or uncontrolled (UBG) blood glucose levels, based on reported current hbAlc levels. The results of the study showed that there were no significant differences in demographic variables. While there was no significant difference in reported treatment adherence as assessed by the patients' MARS-D and the overall VAS scores, patients with UBG had a significantly lower quality of life score as assessed by the EUROHIS-QOL-8. There were no significant differences in MARS-9, VAS, PSS, ISEL-12, or ADS scores; however, patients with UBG showed congruent trends with previous research in that they did have lower MARS-9 scores, higher PSS scores, lower ISEL-12 scores, and lower ADS scores. Patients with UBG also had a higher incidence of depression, as assessed by the PHQ. While there was no significant difference in the number of UBG patients with anxiety or somatization, there were more patients with anxiety and somatization with UBG than with CBG. Also, patients with UBG showed significantly less adherence to short-acting insulin and non-diabetic medication. There were no significant differences in adherence to long-acting/intermediate-acting insulin and oral diabetic medication. Implications of this study suggest that further research must be done to identify barriers to self-care and adherence for diabetic populations to decrease the number of patients who currently experience serious and even fatal complications of having uncontrolled diabetes.

I. INTRODUCTION

It is estimated that by the year 2050, the current prevalence of diabetes will increase from every 1 in 10 adults to every 1 in 3 (Boyle, Thompson, Gregg, Barker, & Williamson, 2010). This substantial increase is also reflected in the fact that currently 33% of Americans have prediabetes, a condition where blood sugar levels are abnormally elevated, but not high enough to be diagnosed as Type 2 diabetes (T2D). Subsequently, 15-30% of those with prediabetes develop Type 2 diabetes within 5 years (Centers for Disease Control, 2014). While Type 1 and gestational diabetes are rarer forms of the disease, the instance of complications associated with these types are just as alarming (Alberti & Zimmet, 1998; Bone, 2015; Maahs, West, Lawrence, & Mayer-Davis, 2010). Diabetes is a rapidly growing issue among the general public that can have serious, and even fatal, health complications if not appropriately treated. Long-term failure to maintain acceptable glycemic control could result in diabetic ketoacidosis, organ failure, cataracts, neuropathy, limb amputation, pregnancy complications, and even death (American Diabetes Association, 2008; Bone, 2015). It is important that patients with diabetes adhere to the recommended treatment plan provided by their healthcare provider to avoid these complications. The rapid increase in diabetes diagnoses indicate that the diabetes epidemic can affect anyone, and it is especially crucial to identify potential barriers to treatment adherence in populations particularly at risk of complications due to the disease. These populations may include the following: those with low socioeconomic status, those with a comorbid mental or physical illness, those without a stable support system, those who struggle with taking routine medication, and those who are affected by chronic stress (Martin, Haskard-Zolnierek, & DiMatteo, 2010).

While regular doctor visits are an important part of treating diabetes, self-management behaviors are a large part of managing the disease to promote glycemic control, the standard at which health care providers determine health risks and complications for patients with diabetes (Haskard & DiMatteo, 2013; American Diabetes Association, 2014). However, several barriers have been identified in previous literature to inhibit self-managing health habits for diabetics and have subjected many to major health consequences, including organ failure, diabetic ketoacidosis, neuropathy, limb amputation, and death (ADA, 2008; ADA, 2014; Maahs, West, Lawrence, & Mayer-Davis, 2010). These barriers include the following: hassle with and inconvenience of taking medication, costs associated with medication and medical equipment, dietary changes and restrictions, forgetfulness, physical pain associated with medication intake, stress, depression, anxiety, lack of social support, and poor appraisal of having diabetes. (Adisa, Alutundu, & Fakeye, 2009; Anderson, Freedland, Clouse, & Lustman, 2001; Antinori-Lent, 2013; Miller & DiMatteo, 2013; Ng, Lee, Toh, & Yu, 2014; Ramchandi et al., 2000; Rosland et al., 2008; Sultan, Epel, Sachon, Vaillant, & Hertemann-Heurtier, 2008; Walker, Smalls, Hernandez-Tejada, Campbell, & Egede, 2014). Often, previous literature identifies independent effects of a particular barrier on the disease, but it may be more important to holistically consider the combined effects of several barriers on an individual with diabetes. Identifying the impact that several barriers have on diabetes control could promote treatment adherence and glycemic control and prevent short-term and long-term diabetic complications.

II. BACKGROUND INFORMATION

Diabetes: Diagnoses and Treatments

Diabetes mellitus (diabetes) is a chronic disease characterized by elevated blood glucose, or blood sugar, as determined by an elevated hemoglobin A1c level (hbA1c), which measures the individual's average blood glucose level for the life span of a red blood cell (approximately 3-4 months) (ADA, 2008; ADA 2014). Treatments for the disease depend on the type of diabetes in question. Type 2, the most common form of diabetes, affects over 29 million people in America and accounts for approximately 90%-95% of all diabetes diagnoses (ADA, 2008; Alberti & Zimmet, 1998; Prentki & Nolan, 2006). The highest rates are among American Indians/Native Americans, non-Hispanic African Americans, and Hispanic populations (ADA, 2008; Centers for Disease Control, 2014b). T2D is notably characterized by insulin resistance – a process by which the body does not reabsorb glucose into the muscle lining, does not recognize insulin produced by the pancreas, or the pancreas itself has insufficient insulin production (ADA, 2008). When insulin cannot be reabsorbed, high levels of blood glucose remain in the blood stream and cause hyperglycemia (Prentki & Nolan, 2006). The counteracting chemical, insulin, is produced by islet β cells in the pancreas in response to excess glucose to establish homeostasis (Prentki & Nolan, 2006); however, those with diabetes do not properly counteract this excess glucose. A major contribution to this phenomenon is thought to be related to excess body fat, especially around the waist and stomach. Patients with T2D are often overweight or obese, and this excess fat can cause insulin resistance, which either creates or exacerbates diabetes symptoms (ADA, 2008). Obesity is also comorbid with other diseases that can negatively affect one's ability to maintain

controlled glucose, such as hypertension, stroke, cardiac arrest, and nephropathy. The exact mechanism behind why β cell function decreases is not well understood, but in a meta-analysis conducted by Prentki and Nolan (2006), they described T2D in terms of overnutrition (malnourishment caused by an excess of nutrients, particularly an abundance of fat) and sedentary lifestyle. Islet β cells expand in response to overnutrition. Eventually, the excess glucose causes oxidative stress to the expanded islet β cells, and result in cell dysfunction and, consequently cell failure. At this point the person is diagnosed with T2D, because there is no longer enough insulin being produced to counteract the high levels of glucose in the blood stream. In a study that identified both environmental and biological factors associated with T2D, researchers found a strong genetic link between genetics and the development of T2D, as indicated by biomarkers, which included hbA1c, body mass index, log triglycerides, and cholesterol levels (Zhou et al., 2014), suggesting that while lifestyle factors are important, those alone do not determine one's risk of developing T2D.

Type 1 diabetes (T1D), formerly referred to as juvenile-onset diabetes, is a rarer form of the disease, affecting 1.25 million in the United States and accounting for approximately 1% of all diabetics (CDC, 2014b). T1D results as an attack on islet β cell functioning in the pancreas by autoantibodies, classifying the disease as an autoimmune disorder (American Diabetes Association, 2013). It is unknown why the immune system suddenly attacks islet β cells in certain individuals, although genetic factors are thought to play a large role in the disease's incidence. It is also unclear as to how much of a role the environment (e.g. viral contact) plays on the incidence and development of the disease (Stene & Rewers, 2012). Unlike T2D, weight is not a contributing factor to the

development of the disease; however, being overweight can cause insulin resistance during the course of the disease and will affect how the patient responds to treatment (Pop et al., 2016). Physical appearance is not the only distinction between T1D and T2D; c-peptide is an amino acid also produced by the pancreas that indicates insulin production (Berger, Stenstrom & Sundkvist, 2000). Patients with T2D, even with severe insulin resistance, have higher c-peptide levels than those with T1D. It is possible to still show an existing c-peptide level along with co-existing low levels of insulin production during T1D onset, in a phenomenon called "the honeymoon phase." However, when an hbA1c level of over 7% and c-peptide level below 0.5 ng/mL are presented, the patient is then diagnosed with T1D, and must soon begin insulin therapy (Berger et al., 2000).

Treatment adherence is defined as the extent to which patients regularly and consistently maintain the treatment plan provided by their primary health care providers (Miller & DiMatteo, 2013; DiMatteo, 2004). Conversely, non-adherence describes patients who do not follow their recommended treatment regimen and engage in negative behaviors such as not taking or incorrectly taking medications, failing to fill or refill prescriptions, failing to get regular blood tests, and not engaging in positive health habits (DiMatteo, Haskard-Zolnierek, & Martin, 2012; Lai-Ming Hui et al., 2015; Magnabosco et al., 2015; Morris et al., 1997). Common treatments to control glucose levels for both T1D and T2D include checking blood sugar levels daily on a personal glucometer, regularly exercising, eating a healthy diet low in carbohydrates, seeing an optometrist and nutritionist annually, getting an adequate amount of sleep, and carbohydrate counting before meals (ADA, 2014). T2D management may include an oral liver suppressant, such as a metformin, to reduce the amount of glucose produced by the liver or the disease may

be treated with a pancreas stimulant, such as a glipizide, to increase insulin production in the pancreas to allow for insulin already produced by the pancreas to be recognized as insulin (ADA, 2014; CDC, 2014).

T1D management includes daily artificial insulin therapy administered subcutaneously through a needle and syringe, an insulin pen, or an insulin pump (ADA, 2014). Type 1 diabetics do not produce the insulin necessary for survival, and therefore must be treated with synthetic insulin throughout their entire lifetimes (ADA, 2014). Injection doses vary from person-to-person, and there is no exact formula to determine doses; because of this, type 1 diabetics are especially subjected to hypoglycemia (an abundance of insulin in the blood stream that results in low blood sugar), which can cause seizures, increased heart rate, decreased blood pressure, cognitive deficits, coma, and even death if left untreated (Evans, Pernet, Lomas, Jones, & Amiel, 2000; Fahrman, et al., 2015; Frier, Schernthaner, & Heller, 2011). Achieving balance between glucose and insulin is the ultimate goal in T1D treatment. The most commonly prescribed insulins are a long-lasting insulin and a short-acting insulin. Type 2 diabetics may also be prescribed a long-acting, short-acting, or mixed formula (intermediate-acting) insulin therapy as a form of treatment, but this is typically done at more severe instances of insulin resistance and uncontrolled blood glucose levels. Long-lasting insulin, such as insulin glargine, is administered once daily to treat basal insulin rates and can remain in the body's system for 24-48 hours (ADA, 2014). Its purpose is to treat basal insulin rates that counteract natural glucose produce by the liver. Short-acting insulin, such as insulin aspart, is given before meals to treat consumed glucose and quickly leaves the blood stream 1-4 hours after administering (ADA, 2014). Patients with T1D are initially treated with both,

however, if the patient is able to use an insulin pump, a stable amount of short-acting insulin is continuously released automatically from the device, and the patient uses the device to administer a bolus dose before meals (ADA, 2014). Failure to adhere to insulin therapy could result in hyperglycemia (high blood sugar) which is the main contributor to major organ failure, especially of the kidneys, liver, heart, eyes and brain; neuropathy; increased risk of infection and subsequent amputation of the extremities; sexual dysfunction; depression; anxiety; and an overall lessened quality of life (Groot et al., 2012; Walker et al., 2014; Boyle et al., 2010; Serrabulho, Gaspar de Matos, Nabais, & Raposo, 2013).

Medication adherence is only one aspect of treatment. As part of regular maintenance patients must see an endocrinologist every 3 months to monitor their hbA1c levels (ADA, 2014). In accordance with the ADA (2014), health care providers recommend that a person with diabetes maintain an hbA1c level of 7.0% or below (≥ 154 mg/dl). This reading helps both the doctor and patient decide what health-promoting practices the patient should engage in, until the next doctor's visit. Engaging in these health-promoting behaviors requires a great amount of independence and self-motivation on the part of the individual. Diabetics with higher self-efficacy, defined as an individual's belief that he or she is capable of performing self-motivating tasks (Bandura, 1997; Wallston, Rothman, & Cherrington, 2007) have been reported to have more controlled glucose levels, better medication adherence, and an overall better quality of life (Walker et al., 2014; Rosland et al., 2008; Miller & DiMatteo, 2013).

The Biopsychosocial Model: Stress and the Autonomic Nervous System

Previous studies have attempted to establish the positive relationship between stress and glycemic control in diabetic populations (Kramer, Ledolter, Manos, & Bayless, 2000; Ismail, Winkley, & Rabe-Hesketh, 2004). However, a holistic view of exact sources of stress has not been well established within the context of treatment adherence and glycemic control (Kramer et al., 2000). This deficit in literature seems to be due to methodological constraints or a narrowed (or conflicting) view of what stress is and from where these stressors come (Lloyd, Smith, and Weinger, 2005). This study attempts to address both the general definition of stress and sources of stress relevant to treatment adherence and glycemic control for diabetic populations.

Stress, as popularly defined by Hans Selye, is "the non-specific response of the body to any demand for change" (1956, pp. 472). As early as 1935, Walter Cannon recognized the body's need for homeostasis (Cannon, 1935). Stress, within acute contexts, is useful for the body and imperative to survival, from an evolutionary perspective. Commonly referred to as "fight-or-flight" response, when faced with an imminent threat, the body's sympathetic nervous system initiates the hypothalamic-pituitary-adrenal (HPA) axis in preparation to be able to either face or flee from the confronting threat (Tsigos & Chrousos, 2002). Signals from the stressor are sent to the hypothalamus and pituitary glands to down-regulate parasympathetic nervous system functioning, which works in opposition to the sympathetic nervous system, and to innervate glands that distribute the necessary hormones and proteins from the adrenal glands needed to provide fuel for the body's "fight-or-flight" response. In order to understand the impact of stress on the body's immune functioning, it is important to

highlight the roles the adrenal glands play in stress responses. The adrenal glands are divided into two main sections: the adrenal medulla and the adrenal cortex. The adrenal medulla is responsible for releasing the non-vital catecholamines epinephrine and norepinephrine which work to regulate the physiological symptoms of stress such as increasing oxygen levels and respiration, increasing muscle strength, constricting vessels, and inhibiting gastrointestinal function. The adrenal cortex produces glucocorticoids among which cortisol may play the largest role in a stress response. Cortisol regulates metabolism by causing glucose to be excreted from the liver during stress response. It is also important in inhibiting parasympathetic functioning and suppressing the immune system. Such processes are not vital to survival when faced with an immediate threat; therefore they are reserved for establishing homeostasis following a "fight-or-flight" response (Tsigos & Chrousos, 2002).

Unfortunately, the body is not able to differentiate the severity of threat amongst different sources of stress. Whether a person is faced with immediate threat to physical safety, or if they suffer from psychological or emotional stress, the inevitable stress response is the same (Tsigos & Chrousos, 2002). Stress causes the liver to produce excess glucose to be used as energy in an acute, stressful situation. However, if an individual experiences prolonged stress, the parasympathetic system cannot fully establish homeostasis and these stress processes that are meant for acute stress remain in effect while experiencing chronic stress. Epinephrine and norepinephrine continue to constrict blood vessels and cause high blood pressure, and cortisol continues to suppress immune function and provide excess glucose for energy (Tsigos & Chrousos, 2002). This hormonal imbalance can be harmful to anyone's health, but as expected, can be even

more detrimental to someone with diabetes. If an individual with diabetes experiences prolonged, chronic stress, glucose levels will remain elevated and can cause both serious complications due to a lack of sufficient insulin production or recognition (Lindmark, Buren, & Eriksson, 2006). Increased catecholamine levels have been observed in diabetic patients with uncontrolled glucose levels (Chiodini et al., 2007; Christensen, 1974), which may indicate a higher level of perceived stress in diabetic populations. Those with elevated cortisol or epinephrine levels can even develop diabetes, as these hormones are positively correlated with stress (ADA, 2008). In addition to dealing with daily hassles that afflict many people living in developed countries, diabetics are faced with the added stress of managing a disease that requires much self-regulation and constant attendance. It is important for individuals with diabetes to identify and resolve these acute stressors in order to prevent chronic stress, poor glycemic control, and subsequent complications.

Barriers to Treatment Adherence

Past research has demonstrated that a number of barriers are related to treatment adherence. Each patient may face a different set of barriers that impede their ability to adhere to a treatment regimen. First, stress plays a large role in treatment adherence, though stress can come from a number of sources and present itself in a number of ways for different individuals. In a controlled study, type 2 diabetic patients who underwent stress management training significantly reduced their hbA1c levels following treatment (Surwit et al., 2002). It may, however, not be so feasible for every diabetic patient to be able to participate in stress reduction treatment due to various limitations. Every diabetic will need to seek medical treatment, and this study may implicate that during the diagnosis or routine doctor's visit, it may be important to emphasize self-managing

behaviors in-between visits. Previous studies have revealed several barriers that have been shown to prevent those with diabetes from performing self-motivated health habits, which may subject them to poor glycemic control and severe diabetic complications (Baghurst & Kelley, 2014; Miller-Hagan & Janas, 2002; Ramchandi et al., 2000; Serrabulho et al., 2013).

Those with diabetes must adhere to a diet low in carbohydrates and fat to help with glycemic control and weight management. Several factors can contribute to poor eating habits including being of low socioeconomic status, stress, or having a busy or demanding lifestyle (Baker, Schootman, Barnidge, & Kelly, 2006; Richardson, Arsenault, Cates, & Muth, 2015). All of these factors can be considered stressful, especially when experienced chronically, and can affect glycemic control in patients with diabetes. In a study that assessed eating behaviors in stressed, low income women, perceived stress was positively associated with uncontrolled eating, emotional eating, and obesity (Richardson et al., 2015). In addition to poor diet, stress can affect one's motivation to get regular exercise. Chaput, Klingenberg, Astru and Sjodin (2010) conducted a meta-analysis and found that living a sedentary lifestyle contributes to overeating. It may not be enough to simply recommend a preferred diet for the patient if they participate in limited to no exercise and have uncontrolled eating in order to cope with stress.

Patients with diabetes may often feel marginalized in that the general public may not understand what it is like to live with the disease and therefore cannot relate to the burden of treatment (Griffith, Field, & Lustman, 1990; Miller & DiMatteo, 2013). Those who take injections may experience pain and physical deformities after prolonged

medication use, and turn to others who understand the everyday hassle of taking medication for advice on less painful ways of injecting, as pain has been positively correlated to hbA1c (Antinori-Lent, 2013; Tamir, Wainstein, Abadi-Korek, Horowitz, & Shemer, 2012). In an article discussing online social media groups for people with diabetes, some vented to others who suffer from diabetes to discuss coping strategies for themselves and their loved ones (Greene, Choudry, Kilabuk, & Shrank, 2011). Social support has been found to increase adherence in diabetic regimens (Miller & DiMatteo, 2013). Conflicting evidence suggests that for adolescent and young adult populations, the quality of support influences adherence more so than simply the presence of support (Rosland et al., 2008). Parents who constantly inquire about blood sugar testing and diet may negatively influence a young person who is already trying to establish independence – creating a competitive, rather than collaborative, outlook on the relationship, and ultimately, the disease itself (DiMatteo, 2004; Rosland et al., 2008). That is to say, it is possible that too much support could also result in stress. Recently, technology has been applied to social support for patients with T2D (Roblin, 2011). Patients who used a text-messaging app that reminded users to engage in self-managing health habits and communicate blood sugar outcomes to their own chosen sources of support (i.e. family, friends, other diabetic patients) were more aware of their self-managing behaviors (Roblin, 2011). Further research may shed light on the impact of a social support group that better understands the difficulties of living with diabetes and the effect such groups have on treatment adherence.

Medical expenses, without considering the average cost of living, for each person living with diabetes in the United States will average approximately between $10,000 and

$14,060 a year, including the costs of blood glucose testing, medication, and regular physicians visits, and emergency hospital visits (ADA, 2013; Miller & DiMatteo, 2013, Ng et al., 2014). In 2012, the overall cost of diabetes in the United States was $245 billion (ADA, 2013; DiMatteo, 2004; Ng et al., 2014; Tunceli, Wade, Bouchard, Aagren, & Luo, 2010). For patients with T1D, these medical expenses can be partially explained by the daily, routine medication. Patients with uncontrolled blood glucose are often prescribed a continuous glucose monitor to help reduce hbA1c, but these devices can cost hundreds of dollars a month. Insulin, insulin supplies (e.g. pen needles, syringes, etc.), and glucagon (emergency glucose injection to counteract severe hypoglycemia) can cost up to $500 depending on the insurance provider. Insulin pumps use a calibration system prescribed by a physician to dispense only short-acting insulin patients with T1D and would reduce the cost of insulin; however, the pump itself can cost between $4,000 and $6,500 without health insurance, and hundreds of dollars a month with insurance for parts that are placed subcutaneously and require weekly replacements (Rosenthal, 2014). Because genetics influence both types of diabetes, oftentimes, there are several diabetics in the household or immediate family; therefore, the financial burden of adherence will have more of an impact. Finances associated with managing diabetes affect medicine-taking behaviors, and presents itself as a barrier to treatment adherence (Ramchandani et al., 2000; Labig, Zantow, & Peterson, 2005; Walker et al., 2014).

Forgetfulness due to time management has also been reported as a possible barrier to treatment adherence in college students (Lehane & McCarthy, 2007; Ramchandani et al., 2000). Research on forgetfulness and time management in diabetic treatment adherence is limited; however, few studies do indicate that forgetfulness is a major

barrier to treatment adherence especially for those with type 2 diabetes or those with type 1 diabetes that have been diagnosed in adulthood (Adisa et al., 2009; Farsaei, Sabzghabaee, Zargarzadeh, & Amini, 2010; Rifulla, Sreedaran, Muttappallymyalil, & Basha, 2014). This may be due to the fact that one may experience difficulties adjusting to the new responsibilities of managing a chronic illness, including a drastic change in diet or the addition of a new medication.

Additionally, a person's perception of the disease may affect his or her ability to cope with the diagnosis and adhere to treatment. The Health Belief Model states that individuals may be more adherent to certain health behaviors if the patients understand how susceptible they are to developing or worsening a disease, know what the negative consequences of having the disease would be, trust their diagnosis and recommended treatment are going to be effective, and understand the difficulties of living with the disease (Martin et al., 2010). Patients who suffer from a chronic disease must be efficacious enough to commit to treatment regimens, as the disease demands self-regulatory behaviors outside of doctor's visits. They must factor in a variety of personal influences that could potentially be a barrier to managing the disease and affect their own quality of life. Research reveals that a person's appraisal of a diagnosis heavily influences the way in which the patient adheres to treatments (Martin et al., 2010). Appraisal has been described as the cognitive process that takes place when an individual evaluates how much value an event has on their life and how to interpret and react to that event (Lazaus & Folkman, 1984). Appraisal of a diabetes diagnosis has been shown to be negatively correlated with the number of diabetic symptoms, suggesting that those who poorly appraise the diagnosis are more likely to have uncontrolled diabetes (Patel, Oza,

Patel, Malhotra, & Patel, 2014; Martin et al., 2010). A negative outlook on being diagnosed with diabetes, and belief that the prescribed medications will not help with blood glucose management may also be a possible barrier to treatment adherence.

If an individual with diabetes suffers from psychopathology, treatment adherence may be affected. It has been reported that patients with T1D and T2D are almost three times more likely to have depression than those who do not have diabetes (Anderson et al., 2001). Depressive symptoms often affect one's daily functioning and for a person with diabetes, this could influence the individual's willingness to commit to the necessary adherence behaviors involved in regulating blood glucose levels. Glycemic control, treatment adherence, and stress were reported to be significantly worse in adolescents with T1D who experienced depressive symptoms (Baucom et al., 2015). It can be argued that stress and depression are higher overall in adolescents and they tend to engage in poorer health behaviors such as poor diet, less exercise, irregular sleeping, and increased rumination, particularly if the child is under stress (Baucom et al., 2015; de Vriendt et al., 2012; Dewald, Meijer, Oort, Kerkhof, & Bögels, 2014; Skitch & Abela, 2008). Unfortunately, adult patients with diabetes also tend to engage in poor health habits when presenting depressive symptoms (Anderson et al., 2001). Cienchanowski, Katon and Russo (2000) assessed the impact of depression on diabetic treatment adherence, and in their study depression severity was categorized into three groups based on severity and number of presenting depression symptoms. Being in the most severe depression symptom category significantly predicted fewer self-care behaviors, poorer diets, and having higher costs related to doctors' visits and number of hospitalizations (Cienchanowski et al., 2000). State and trait anxiety have been shown to be predictors in

determining uncontrolled hbA1c, with higher anxiety indicating more uncontrolled hbA1c (Sultan et al., 2008). Appraisal may also contribute to poor glucose control in patients with anxiety, as a negative perception of the disease may invoke a sense of insecurity.

Measuring Treatment Adherence

The purpose of this study is to identify perceived barriers to treatment adherence and glycemic control in patients diagnosed with T1 or T2 diabetes mellitus. While the recommended hbA1c level is an objective concrete measurement, one of the major limitations of previous research has been adequately measuring treatment adherence specifically for diabetic populations. For a person with diabetes, the best measure of treatment adherence would be one's hbA1c level, along with other indicators of acceptable health, such as HDL cholesterol levels. Unfortunately, these tests can be expensive and invasive when used in research studies; therefore researchers heavily rely on self-report measures. These tests also do not explain other psychosocial factors that contribute to glycemic control. Serrabulho et al. (2013) devised a 63-item survey that correlated health behaviors of adults with type 1 diabetes aged 18-35 with hbA1c levels, and found that while 51% of participants expressed that they had "reasonable health," the mean hbA1c was 8.7% (203 mmg/dl) and 86% of respondents suffered from late-stage diabetic complications (Serrabulho et al., 2013). This discrepancy highlights potential errors in the scales' validity. Other studies have attempted to measure adherence in patients with chronic illness, but there is no widely accepted scale that measures overall treatment adherence for someone with diabetes (Culig & Leppée, 2014). Based on previous literature, several facets are involved in treating diabetes, and thus each facet

must be addressed. Adherence cannot be fully established through a single self-report measure. Through having a more thorough account of not only medication history but also accounting for factors that support the patient's medication-taking habits will help to validate self-reported adherence. Because several factors affect medication-taking habits, inconsistencies can be more easily detected if medication, medication type, views concerning treatment, and barriers to treatment are addressed

III. PURPOSE AND HYPOTHESES

This study will explore the association between potential barriers to treatment adherence for people with diabetes, such as financial income, social support, depression, anxiety, somatization, common barriers such as medication difficulties and side effects, appraisal of the disease, stress level, and quality of life. Because this study's interests were in the holistic effects of many stressors on diabetic glycemic control, comparison groups were based on glycemic control as determined by hbA1c level. Predictions maintained are as follows:

1. There will be no significant demographic differences in gender, ethnicity, or diagnosis (Type 1 insulin dependent, Type 2 non-insulin dependent, Type 2 insulin dependent) between patients with uncontrolled hbA1c level (UBG) and patients with controlled hbA1c level (CBG).
2. The mean age of those in the UBG group will be significantly lower than those in the CBG group.
3. There will be a significant difference in income levels between patients with UBG and patients with CBG, with more uncontrolled patients in the lower income levels.
4. There will be a significant difference in treatment adherence scores between patients with UBG and patients with CBG. The UBG group will report less treatment adherence.
5. The extent to which patients with UBG report barriers to affect adherence will be greater than that of patients with CBG.

6. There will be a significant difference in perceived stress score. Those with UBG will have significantly higher perceived stress scores than those with CBG. There will be a significant difference in perceived social support. Those with UBG will have significantly lower perceived social support scores than those with CBG.
7. There will be a significant difference in diabetes appraisal. Those with UBG will have significantly lower diabetes appraisal scores than those with CBG.
8. There will be a significant difference in perceived quality of life. Those with UBG will have significantly lower perceived quality of life scores than those with CBG.
9. There will be a significant difference in medication adherence, with patients on insulin having worse adherence scores than patients on oral diabetic medication. Those with UBG will have lower adherence scores than those with CBG.
10. Significantly more patients with UBG will have depression than those with CBG.
11. Significantly more patients with UBG will have anxiety than those with CBG.
12. Significantly more patients with UBG will have somatization than those with CBG.

IV. METHOD

Participants

This study consisted of 85 total participants (males = 11.8%) who were at least 18-years-old and must have had a prior diagnosis of either type 1 or type 2 diabetes mellitus. Participants also had to have been recommended to follow a treatment regimen as prescribed by their primary health care provider. All participants were recruited via social networking websites or groups within a particular website (e.g. Women with Diabetes Facebook group, Students with Diabetes, Juvenile Diabetes Research Fund Facebook groups, etc.) of which the themes were diabetes maintenance or social support for people with diabetes. The study was considered to have low potential risk of trauma, as the survey included questions regarding state of mental illness (e.g. depression, anxiety, somatization, etc.), medical information regarding hemoglobin A1c, and medication use. The survey was only administered in English and was therefore only available to those who could read and write in English. Participants were informed that participation was entirely voluntary and that they could stop the survey at any point without consequence; however, they must have given informed consent to begin the study. All participants consented to participate and all information analyzed was self-reported. This study was approved by the Institutional Review Board for Texas State University.

Materials

The survey was administered online via Qualtrics at the participants' convenience. Upon giving consent, participants provided general demographics. The demographics analyzed included gender, year of birth, race, income level, last known hemoglobin A1c, the type of diabetes with which the patient had been diagnosed, and what treatment plan and medications had been prescribed by an endocrinologist or primary care physician.

Medication Adherence Rating Scale (MARS-D).

The original MARS scale consisted of 5 Likert-scale questions to assess medication-taking habits within the past 6 months, however, the 9-item MARS-D has been validated in several countries including Sweden and Germany. The MARS-D has a moderate Cronbach's α value of .60, and a Pearson's r score of .61, and includes 5-point Likert-scale questions (0 "never" – 5 "always") on how often they adhere to taking medication in the past 6 months, such as, "I decide to miss out on a dose" and "I forget to use it" (Mahler et al., 2010; Thompson, Kulkarni, & Sergejew, 2000). The scale included one reverse scored item, and adherence scores calculated as an averaged sum total.

Barriers to Medication Adherence Questionnaire.

The Barriers to Medication Adherence Questionnaire is a set of questions previously comprised by Haskard-Zolnierek and Howard in an unpublished article describing relevant barriers that may affect treatment adherence or medication-taking habits. Using an 11-point visual analogue scale, participants are asked to rate whether or not each barrier is not, sometimes is, or is a major barrier for them, using statements such as, "I forget to take my medications" and "I cannot afford my medications." Common

barriers included medication costs, medication-taking methods, alcohol consumption, and side effects. Each barrier was calculated as a single rating based on the participant's provided score.

Patient Health Questionnaire (PHQ).

The PHQ is comprised of several different facets of health. For the purposes of this study, we chose to use this tool to assess mental health functioning, as the PHQ is a widely used and well-validated tool (Spitzer, Kroenke, &Williams, 1999). Because each sub-scale can be independently scored, this study used the appropriate sub-sections to assess somatization, depression, and anxiety. Only questions over these areas were asked, as they are the most relevant to treatments widely prescribed for diabetes, as well as comorbid symptoms of diabetes. A total of 31% of those with diabetes have been diagnosed with major depression (Katon, 2008) and those with both diabetes and depression have been reported to be 66% more likely to be non-adherent to treatment regimens (Anderson et al., 2001). The PHQ used Likert-scale questions to assess experience with somatizations (e.g. headaches, back pain, fainting, etc.), depression symptoms (e.g. feeling down or depressed, having little interest in activities), and anxiety (e.g. feeling "on edge" or restless). As anxiety and somatoform symptoms are known to be highly comorbid with depression, this study addressed these symptoms as well.

Perceived Stress Scale (PSS).

The PSS asks 10 Likert-scale questions (0 "never" – 4 "very often") to assess the participants general stress levels in the past month (Cohen, Kamarack, & Mermelstein, 1983). For example, participants rated how often in the past month they "felt nervous and 'stressed'" or "found that you could not cope with all the things you had to do." Four of

the questions are reverse coded items, and each participant's PSS score was a continuous variable denoted by a sum total. Higher scores indicate higher perceived stress. This scale was included because it has been widely validated and used globally.

Visual Analogue Scale for Medication Adherence (VAS).

The VAS asks participants to describe to what extent they have taken each self-reported, prescribed medication within the past month, on a visual analogue scale from 0%-100% (Nau, Steinke, Williams, Austin, Lafata, Civine, & Pladevali, 2007). The single item was as follows: "We would be surprised if most people take %100 of their medications. Below 0% means you have not taken the medication in this past month, 50% means you have taken half of your medication this past month and 100% means you have taken every single dose this past month. Please list and provide the percent of each medication you took this month." This scale was used because it allows for diversity within the survey, and will appeal to a more visual audience. Because diabetes tends to be co-morbid with other illnesses (Piette & Kerr, 2006), this scale analyzes adherence for non-diabetic medication. The listed medications were categorized into one of 4 groups for analysis: long/intermediate-acting insulin, short-acting insulin, oral diabetic medication, or non-diabetic medication. Participants could report as many as 4 different types of medications prescribed. Adherence percentages were averaged within each medication category per participant to yield a total adherence score for each medication category.

EUROHIS Quality of Life Index (EUROHIS-QOL-8).

The EUROHIS-QOL-8 (Schmidt, Mühlan, & Powers, 2006) is derived from the 100-item World Health Quality of life survey (WHOQOL-BREF). This Likert-scale

survey has been tested and validated in over 10 countries. The index asks general questions over one's satisfaction with life, such as, "How satisfied are you with your personal relationships?" and "Do you have enough energy for everyday life?" The EUROHIS-QOL-8 score was a continuous variable calculated as the total sum of all questions, with higher scores indicating better quality of life.

Appraisal of Diabetes Scale (ADS).

Also derived from the WHOQOL-BREF, the ADS includes 7 Likert-scale questions over one's perception of their diabetes diagnosis (Patel et al., 2014). This survey assesses one's perception of the severity of the disease as well as the extent of control he/she has over managing their disease. Example questions include, "How likely is your diabetes to worsen over the next several years?" and "To what degree does your diabetes get in the way of developing your life goals?" The ADS showed a strong Cronbach's α value of .80 during development and validation (Patel et al., 2014), and was chosen for its specificity to diabetic patients. The total ADS score was a total sum of responses, with higher scores indicating worse appraisal.

Interpersonal Support Evaluation List (ISEL-12).

The ISEL-12 (Merz et al., 2014) assesses the participant's perceived social support system. Participants responded to statements to which they agreed to how definitely true to definitely false the statements were. This scale has a total Cronbach's α of .70 and has been validated in over 5 countries for both English and Spanish speakers (Merz et al., 2014). Example questions included, "If I were sick, I could easily find someone to help me with my daily chores" or "There is someone I can turn to for advice about handling problems with my family." The scale included 6 negatively worded items,

and was calculated as a total sum, with higher scores indicating more perceived social support.

Procedure

Participants were placed into comparison groups based on hbA1c level, as either having controlled blood glucose (CBG) (hbA1c $\leq$ 7.0) (n = 40) or uncontrolled blood glucose (UBG) (hbA1c > 7.0) (n = 44) in accordance to the ADA recommended average blood glucose level for patients with diabetes. Participants were given a link to complete an online survey at their convenience via Qualtrics, an online surveying tool. All participants were encouraged to complete the survey in its entirety, however due to the discretionary nature of the study, the total number of responses varied by scale. The survey began with a presenting consent form which participants must have agreed to prior to beginning the survey. The survey also included basic demographic questions as well as the following questionnaires: MARS-9, Barriers to Adherence Questionnaire, PHQ, VAS, EUROHIS-QOL-8, ADS, and ISEL-12.

Statistical Analysis

This study is exploratory in nature. Univariate tests were used to compare differences in barriers across the UBG and CBG groups. For the categorical demographic variables ethnicity, gender, and diagnosis (i.e. type 1 insulin-dependent, type 2 insulin dependent, and type 2 non-insulin dependent), Pearson chi-square tests were used to identify differences across the UBG and CBG groups. For the continuous demographic variables, income and age, independent samples t-tests were used to identify differences between UBG and CBG groups.

Independent sample t-tests were also conducted to identify differences between UBG and CBG groups for MARS-9, Barriers to Adherence Questionnaire, PSS, VAS, EUROHIS-QOL-8, ADS, and ISEL-12. For PHQ subsections, chi-square with Fischer Exact Tests were used to identify frequencies and differences between those with or without the presence of depression, anxiety, and somatization. All analyses were conducted using SPSS version 22.

V. RESULTS

Participants totaled 84 overall, however due to the voluntary nature of the survey, every participant did not answer every question. Data was screened for missing values, skewness, and outliers during analysis. In regard to hypothesis 1, there were no significant differences in gender χ^2 (1, N=84) = .026, p = .872; ethnicity χ^2 (1, N=84) = 8.803, p = .117; or diagnosis, that is type 1 insulin dependent, type 2 non-insulin dependent, type 2 insulin dependent, χ^2 (2, N=84) = .045, p = .978, between patients with uncontrolled hbA1c level (UBG) and patients with controlled hbA1c level (CBG), as expected. In regard to hypothesis 2, the mean age of those in the UBG group (34.93) was not significantly lower than the mean age of those in the CBG group (37.80), t(80) = .914, p = .363, in contrast to the hypothesis. Considering hypothesis 3, there was no significant difference in income level between patients with UBG and patients with CBG, χ^2 (4, N=82) = 6.315, p = .177, which was unexpected (see Table 1).

Table 1. Participant demographics frequencies, or means and standard deviations, based on controlled blood glucose level and uncontrolled blood glucose level comparison groups.

	Controlled N (%)	Uncontrolled N (%)	χ^2	t	df	Significance
Age (n = 84)						
Mean (SD)	37.8 (14.9)	36.3 (13.5)	--	.914	80	p = .363 (NS)
Gender (n = 84)						
% Male	5 (12.5%)	5 (11.4%)	.026	--	1	p = .872 (NS)
% Female	35 (87.5%)	39 (88.6%)				
Ethnicity (n = 84)						
Black/African American	0 (0.0%)	2 (4.5%)				
Asian	0 (0.0%)	1 (2.3%)				
Hispanic/Latino(a)	1 (2.5%)	8 (18.2%)	8.803	--	5	p = .117 (NS)
White/Caucasian	37 (92.5%)	31 (70.5%)				
Native American/Alaskan Native	1 (2.5%)	1 (2.3%)				
Other	1 (2.5%)	1 (2.3%)				
Income Groups (n = 82)						
< $20,000	7 (18.4%)	15 (34.1%)				
$20,001 - $40,000	8 (21.1%)	8 (18.2%)				
$40,001- $60,000	6 (15.8%)	11 (25.0%)	6.315	--	4	p = .177
$60,001 - $80,000	6 (15.8%)	5 (11.4%)				
> $80,000	11 (28.9%)	5 (11.4%)				
Diagnosis (n = 84)						
Type 1 (insulin dependent)	32 (80.0%)	36 (81.8%)				
Type 2 (non-insulin dependent)	4 (10.0%)	4 (9.1%)	.045	--	2	p = .978 (NS)
Type 2 (insulin dependent)	4 (10.0%)	4 (9.1%)				

With regard to hypothesis 4, there was no significant difference in MARS-D scores, t(73) = -.660, p = .511, between patients with UBG (M=7.32, SD=5.01), and patient

with CBG (M=6.65, SD=3.61), in contrast to the hypothesis. There was no significant difference in general VAS scores, t(80) = .470, p = .640, between patients with UBG (13.20) and patients with CBG (13.69) (see Table 2), in contrast to the hypothesis (see Table 2).

Table 2. Means, standard deviations and t-test results for MARS, VAS, PSS, ISEL-12, ADS, and EUROHIS-QOL-8 scores between controlled blood glucose and uncontrolled blood glucose groups.

	Controlled Mean (SD)	**Uncontrolled Mean (SD)**	**t**	**df**	**p-value**
Medication Adherence Rating Scale	6.65 (3.61)	7.32 (5.01)	-.660	73	.511 (NS)
Visual Analogue Scale for Medication Adherence	13.69 (4.75)	13.20 (4.71)	.470	80	.640 (NS)
Perceived Stress Scale	14.52 (8.44)	18.07 (7.21)	-1.724	56	.090 (NS)
Interpersonal Support Evaluation List	38.93 (7.64)	35.36 (8.99)	1.671	60	.100 (NS)
Appraisal of Diabetes Scale	7.97 (4.73)	10.00 (3.71)	-1.859	58	.068 (NS)
EUROHIS Quality of Life Index	31.50 (5.26)	27.22 (6.13)	2.883	58	.006**

*p <.05, **p<.01

Hypothesis 5 stated that the extent to which each barrier affects medication adherence would be significantly higher for the UBG group than the CBG group. Comparison results between the UBG and CBG groups for each of the twelve barriers are as follows

(see Table 3): “I cannot afford my medications.” – There was no significant difference between patients with UBG (M=2.30, SD=3.21) and patients with CBG (M=1.28, SD=2.53), t(80.447) = -1.625, p = .108, in contrast to the hypothesis. “My medication does not seem to work.” – There was no significant difference between patients with UBG (M=1.05, SD=2.19) and patients with CBG (M=.58, SD=1.43), t(82) = -1.154, p = .252. “I feel better, so I don’t need to take my medicine.” – There was no significant difference between patients with UBG (M=.70, SD=1.93) and patients with CBG (M=.28, SD=.85), t(60.076) = -1.338, p = .186, in contrast to the hypothesis. “Taking my medication is a hassle.” – As hypothesized, there was a significant difference between patients with UBG (M=2.48, SD=3.19) and patients with CBG (M=.92, SD=1.72), t(67.687) = -2.805, p = .007. “My medication causes me to gain weight.” – There was a significant difference between patients with UBG (M=2.09, SD= 3.18) and patients with CBG (M=.87, SD= 1.84), t(70.195) = -2.166, p = .034, as hypothesized. “My medication causes a loss of appetite and/or makes me lose weight.” – In contrast to the hypothesis, there was no significant difference between patients with UBG (M=.18, SD=.79) and patients with CBG (M=.18, SD=.51), t(81) = -.016, p = .987. “My medication makes me nauseous or causes stomach/digestive problems.” – There was no significant difference between patients with UBG (M=.59, SD=1.50) and patients with CBG (M=.38, SD=1.09), t(81) = -.709, p = .481, in contrast to the hypothesis. “My medication makes me dizzy, drowsy, or light-headed.” – There was no significant difference between patients with UBG (M=.50, SD=1.37) and patients with CBG (M=.31, SD=1.17), t(81) = -.682, p = .497, in contrast to the hypothesis. “I am not supposed to drink alcohol while taking this medication.” – There was no significant difference between patients with

UBG (M=.57, SD=1.48) and patients with CBG (M=.49, SD=1.17), t(81) = -.493, p = .785, in contrast to the hypothesis. "My medication is difficult to take (pills too big, tastes bad, etc.)" – There was no significant difference between patients with UBG (M=.76, SD=2.09) and patients with CBG (M=.15, SD=.43), t(45.925) = -1.880, p = .066; however, this barrier reflected the expected trend based on the hypothesis. "I have a hard time following directions for how to take my medicine (on an empty stomach or with food, etc.)" – There was no significant difference between patients with UBG (M=.50, SD=1.62) and patients with CBG (M=.10, SD=.38), t(81) = -1.494, p = .139, which was in contrast to the hypothesis. "My medication makes me anxious or jittery." – There was no significant difference between patients with UBG (M=.11, SD=.39) and patients with CBG (M=.15, SD=.43), t(81) = .448, p = .656, which was in contrast to the hypothesis.

Table 3. Means, standard deviations and t-test results for individual barriers to adherence between controlled blood glucose and uncontrolled blood glucose groups.

	Controlled Mean (SD)	**Uncontrolled Mean (SD)**	***t***	**df**	**p-value**
I cannot afford my prescriptions.	1.28 (2.53)	2.30 (3.21)	-1.625	80.447	.108 (NS)
My medication does not seem to work.	.58 (1.43)	1.05 (2.19)	-1.154	82	.252 (NS)
I feel better so I don't need to take my medicine.	.28 (.85)	.70 (1.93)	-1.338	60.076	.186 (NS)

Table 3. Continued.

Taking my medication is a hassle.	.92 (1.72)	2.48 (3.19)	-2.805	67.687	007**
My medication causes me to gain weight.	.87 (1.84)	2.09 (3.18)	-2.166	70.195	.034*
My medication causes a loss of appetite and/or makes me lose weight.	.18 (.51)	.18 (.79)	-.016	81	.987 (NS)
My medication makes me nauseous or causes stomach/digestive problems.	.38 (1.09)	.59 (1.50)	-.709	81	.481 (NS)
My medication makes me dizzy, drowsy, or light-headed.	.31 (1.17)	.50 (1.37)	-.682	81	.497 (NS)
I am not supposed to drink alcohol while taking this medication.	.49 (1.17)	.57 (1.48)	-.493	81	.785 (NS)
My medication is difficult to take (pills to big, tastes bad, etc.)	.15 (.43)	.57 (1.48)	-1.880	45.925	.066 (NS)
I have a hard time following directions for how to take my medicine (on an empty stomach or with food, etc.)	.10 (.38)	.50 (1.62)	-1.494	81	.139 (NS)

Table 3. Continued.

My medication makes me anxious or jittery.	.15 (.43)	.11 (.39)	.448	81	.656 (NS)

*p < .05

With regard to hypothesis 6, there was no significant difference in PSS scores between patients with UBG (M=18.07, SD= 7.21) and CBG (M=14.52, SD=8.44), t(56) = -1.724, p = .090; however, the trend in PSS scores did reflect the direction of the hypothesis (see Table 2). Considering hypothesis 7, there was no significant difference in ISEL-12 between patients with UBG (M=35.36, SD=8.99) and patients with CBG (M=38.93, SD=7.64), t(60) = 1.671, p = .100 (see Table 2), in contrast to the hypothesis In regard to hypothesis 8, there was no significant difference in ADS scores between patients with UBG (M=10.00, SD=3.71) and CBG (M=7.97, SD=4.73), t(58) = -1.859, p = .068; however, the trend in ADS scores did reflect the direction of the hypothesis. (see Table 2). There was a significant difference in EUROHIS-QOL-8 between patients with UBG (M=27.22, SD= 6.13) and CBG (M=31.50, SD=5.26), t(58) = 2.883, p = .006, as hypothesized (see Table 2).

With regard to hypothesis 10, there was no significant difference in VAS scores for patients taking long-acting/intermediate insulin between UBG (M=82.73, SD= 34.05) and CBG (M=99.90, SD=.31), t(7.001) = 1.456, p = .120, which was not hypothesized, but did support the hypothesis, in that adherence was worse for the UBG group. There was a significant difference in VAS scores for patients taking short-acting insulin between UBG (M=85.19, SD=22.25) and CBG (M=97.41, SD=4.40), t(24.798) = 2.641,

p = .014, as hypothesized. There was no significant difference in VAS scores for patients taking oral diabetic medication between UBG (M=89.44, SD=12.29) and CBG (M=96.54, SD=9.38), t(15) = 1.324, p = .305, in contrast to the hypothesis. There was a significant difference in VAS scores for patients taking non-diabetic medication between UBG (M=75.90, SD=26.55) and CBG (M=94.67, SD=9.79), t(23.473) = 2.993, p = .006 (see Table 4).

Table 4. Means, standard deviations, and t-values in mean VAS adherence scores between controlled blood glucose and uncontrolled blood glucose groups, amongst medication types.

	Controlled Mean (SD)	**Uncontrolled M (SD)**	**t**	**df**	**p-value**
Long-acting/Intermediate-acting Insulin	99.90 (.31)	82.37 (34.05)	1.456	7.001	.120 (NS)
Short-acting Insulin	97.41 (4.40)	85.19 (22.25)	2.641	24.798	.014*
Oral Diabetic Medication	96.54 (9.38)	89.44 (12.29)	1.324	15	.205 (NS)
Non-Diabetic Medication	94.67 (9.79)	75.90 (26.55)	2.993	23.473	.006**

*p <.05, **p <.01

Considering hypothesis 11, significantly more patients with UBG (n=13) had depression than those with CBG (n=4), χ^2 (1, N=66) = 5.051, p = .025, as hypothesized (see Table 5). In regard to hypothesis 12, there was no significant difference in the number of patients with UBG (n=7) than those with CBG (n=1) that had anxiety, χ^2 (1,

N=65) = 3.808, p = .066; however, the hypothesis was supported, in that there were more UBG patients with anxiety and scores approached significance (see Table 5). Finally, considering hypothesis 13, there was no significant difference in the number of patients with UBG (n=9) than those with CBG (n=6) that had somatization, χ^2 (1, N=66) = .233, p = .629, in contrast to the hypothesis (see Table 5).

Table 5. Chi Square and Fisher exact values and frequencies for Patient Health Questionnaire presence of somatization, anxiety, and depression among controlled blood glucose and uncontrolled blood glucose groups.

	Controlled N (%)	**Uncontrolled N (%)**	**χ^2**	**df**	**p-value**
Somatization (n = 66)					
Present	6 (20%)	9 (25%)	.233	1	.629 (NS)
Not Present	24 (80%)	27 (75%)			
Anxiety (n = 65)					
Present	1 (3.4%)	7 (19.4%)	3.808	1	.066 (NS)
Not Present	28 (96.6%)	29 (80.6%)			
Depression (n = 66)					
Present	4 (12.9%)	13 (37.1%)	5.051	1	.025*
Not Present	27 (87.1%)	22 (62.9%)			

*p <.05

VI. DISCUSSION

The purpose of this study was to identify significant barriers to treatment adherence and glycemic control for diabetic patients. It was predicted that patients with an uncontrolled hbA1c level would be significantly less adherent to treatment regimens and have more reported barriers to adherence. While many of the variables tested did not reach traditional significance levels, most variables reflected the expected trends in the literature, in that the patients with UBG were less adherent to treatment and expressed more barriers to treatment adherence. One variable that did not reflect the literature was age. It was predicted that the mean age of those in the UBG group would be younger than the mean age of the CBG group. There were no significant differences in age, which may be due to the study's small sample size. However, patients with an uncontrolled hba1c level reported they were less adherent to treatment, had higher levels of perceived stress, had less social support, and had a lower appraisal of diabetes which has been depicted in previous research, as trends in these variables reflected trends in previous literature and approached significance.

One of the significant items in the Barriers to Medication Adherence Questionnaire that was significantly related to having UBG was "Taking my medication is a hassle." As previously discussed, medication adherence requires more than simply taking the medication; the patient must also consistently fill prescriptions and take the medication according to the prescribed regimen provided by the primary healthcare physician (Miller & DiMatteo, 2013; DiMatteo, Haskard-Zolnierek, & Martin, 2012). If the patient is not able to afford or has difficulty retrieving his or her medication, they may struggle with non-adherence and loss of glycemic control. Whether the patient is on oral

medication or taking insulin subcutaneously, they must make sure they are taking the medication as prescribed. For some patients a daily oral medication (e.g. Metformin Extended Release) or a daily injection of long-acting insulin (e.g. Lantus) can be enough to control blood glucose levels, with controlled eating; however, many patients must take multiple doses any time blood sugars may be affected, which can mean any time any amount of glucose or carbohydrates is consumed. Someone with diabetes cannot mindlessly drink a beverage or eat a small snack with carbohydrates without the risk of the elevated blood sugar that may result (England, Andrews, Jago, & Thompson, 2014). This risk is even higher for patients with T1D, as there is no insulin production and without taking a shot of synthetic insulin or bolusing from an insulin pump, an elevated blood sugar level is inevitable. Patients in the UBG group did express significantly more hassles with medication than those in the CBG group. Unfortunately, the pain associated with shots or forgetfulness due to inconvenience also contributes to medication hassle and further emphasizes the importance of holistically observing multiple barriers to each facet of adherence (Antinori-Lent, 2013; Adisa et al., 2009; Lehane & McCarty, 2007).

Another significant item in the Barriers to Adherence Questionnaire was "My medication makes me gain weight." While weight gain may not be thought of as a side effect of diabetic medication, for patients with T1D, weight gain is a very common side effect when initiating insulin therapy. Without proper insulin production, glucose remains at elevated levels in the bloodstream, so naturally, the body looks for ways to eliminate the excess glucose. Glucosuria is the process of glucose being eliminated from the system through urine. For T1D patients with UBG, excess weight loss is a symptom due to the fact that the body is constantly using energy to eliminate the glucose and is losing high

volumes of nutrients and water to do so. The result is the appearance of abnormally low weight (Brown, Wijewickrama, Harlan, & Rother, 2009; The DCCT Research Group, 1988; National Institutes of Diabetes and Digestive and Kidney Diseases, 2013; Yazigi & Andreelli, 2007). Once insulin therapy begins, the body no longer has to rely on glucosuria to do insulin's job and the body "holds onto" the consumed calories as it normally would without the presence of diabetes, resulting in normal fat- and water-retention, which often looks like excessive weight gain (The DCCT Research Group, 1988). In this study, 76 of the 84 patients expressed they use insulin to control blood sugars, which explains why many may have seemed to experience weight gain from the medication.

As hypothesized, patients in the UBG group had significantly lower quality of life scores than those in the CBG group. Both long-term and short-term diabetes complications will affect one's quality of life causing physical, social, and psychological discomfort (Adisa, Alutundu, & Fakeye, 2009; Anderson, Freedland, Clouse, & Lustman, 2001; Antinori-Lent, 2013; Miller & DiMatteo, 2013; Ng, Lee, Toh, & Yu, 2014; Ramchandi et al., 2000; Rosland et al., 2008; Sultan, Epel, Sachon, Vaillant, & Hertemann-Heurtier, 2008; Walker, Smalls, Hernandez-Tejada, Campbell, & Egede, 2014). Physically, patients with UBG face fatigue, ketoacidosis, nerve damage (neuropathy), organ failure, limb amputation, and death (American Diabetes Association, 2008; Bone, 2015). Socially, patients with diabetes may feel isolated and often have difficulty covering the cost of maintaining the disease (Griffith, Field, & Lustman, 1990; Miller & DiMatteo, 2013). Patients with UBG were significantly more likely to have depression and, while not significant, the trend in the data showed the UBG group to

have an increased instance of anxiety and somatization. Psychologically, patients with diabetes are known to have higher rates of depression and anxiety (Anderson et al., 2001; Cienchanowski et al., 2000), but the direction of the relationship between psychopathology and glycemic control has not yet been established (Kongakew, Jampachaisri, Chaturongkul, & Scholfield, 2014). In a wide-scale meta-analysis conducted by Grenard at al. (2011), it was noted that depression generally does have a negative impact on adherence for chronically ill patients taking routine medications, but they excluded populations that take injectable medications from their study.

One prediction that did not follow the hypothesis was the instance of patients with UBG on insulin versus the instance of those on oral medication. Patients with UBG were overall less adherent to their medication regimens, as predicted; however, they were only significantly less adherent to short-acting insulin and non-diabetic medication. Short-acting insulin peaks only minutes to a few hours after injection, and quickly leaves the system. There may be more of a hassle associated with multiple injections as opposed to one long-acting injection (Antinori-Lent, 2013). Lower adherence scores were also observed in patients with UBG on non-diabetic medication. All participants were on medications to control glucose levels for diabetes, which means if they were taking a medication for a non-diabetic condition, they were taking multiple medications (polypharmacy). Polypharmacy has been associated with decreased adherence for patients with T2D largely due to misinformation regarding medication side effects, efficacy, and communication breakdowns between the patients and the prescribing physician (Austin, 2006; Haskard-Zolnierek & DiMatteo, 2009).

Limitations

There were limitations with this study. Many of the hypotheses tested were not significant, yet the trends in the data supported the direction of the hypotheses. These null results could be explained by the small sample size, as trends would be expected to continue to significance if the sample was larger. The sample was also overrepresented by Caucasians and by females (see Table 1), and thus may not generalize to other populations. Another limitation of this study was the differing completion rates for each of the scales in the survey. Due to the voluntary nature of the survey, participants could choose whether or not they completed each battery. Because of this, completion rates were inconsistent throughout the survey, and had an effect on the amount of information received.

Measuring adherence was also a limitation to this study. All measures were self-report; therefore, there may not have been an accurate assessment of actual medication adherence. There is no standard measure of adherence for patients with diabetes, as treatment relies heavily on self-care. Generally, this should have been addressed by the adherence scores' consistency with other measures (e.g. hbA1c) that would generally be associated with adherence; but, without an objective measure of adherence, some validity may have been lost. This also relates to the fact that different patients will have different treatment regimens depending on a number of variables, including weight, metabolism, diet, exercise, medication allergies, and a host of other factors that could affect prescribing. While there were questions – notably in the VAS – that addressed whether patients were adherent to their doctors' suggested treatment plan for them or not, this study did not address each patient's specific instructions given for treatments other than

medication doses (i.e. daily carbohydrate intake, amount/types of exercise recommended, etc.). As previously discussed, there are standard treatments for patients with each type of diabetes; however, if a patient required modifications to the standard recommended treatment, those modifications were not assessed by this study.

Clinical Implications

Diabetes is a chronic condition that affects every facet of life. Self-managing behaviors are key to glycemic control and preventing diabetic complications. Several barriers to adherence may not be addressed in clinical settings, and this can negatively affect health outcomes for diabetic patients. Because patients with UBG are seen to have more adverse biological, psychological, and social outcomes, discussing treatment options, medication side effects, medication and equipment costs, dietary changes, exercise options, reaching out for social support, and psychological well-being should be addressed in clinical settings. If patients are aware of the steps they must take to avoid costly complications, they may be more inclined to follow treatment protocols and improve their health.

Future Directions

This study has shed light on several barriers that can affect treatment adherence and glycemic control in diabetic populations. In addition to addressing the limitations to this study, future studies may include assessing barriers in specific diabetic populations, such as college students; assessing sleep disturbance as a contributor to insulin resistance in type 1 diabetics; understanding the effect of multiple diagnoses and polypharmacy on diabetic glycemic control; comparing biomarkers of chronic stress (allostatic load)

between socioeconomic groups with diabetes; or assessing food convenience/snacking in patients with diabetes.

REFERENCES

Adisa, R., Alutundu, M. B., & Fakeye, T. O. (2009). Factors contributing to nonadherence to oral hypoglycemic medications among ambulatory type 2 diabetes patients in Southwestern Nigeria. *Pharmacy Practice*, *7*(3), 163.

Alberti, K. G. M. M. & Zimmet, P. Z. (1998). Definition, diagnosis, and classification of diabetes mellitus and its complications part 1: Diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabetic Medicine*, *15*, 539-553.

American Diabetes Association (2008). Diagnosis and classification of diabetes mellitus. *Diabetes Care, 31*(1), 55-59. doi:10.2337/dc08-S055

American Diabetes Association (2013). Economic costs of diabetes in the U.S. in 2012. (2013). *Diabetes Care*, *36*(4), 1033-1046. doi:10.2337/dc12-2625

American Diabetes Association (2014). Standards of medical care in diabetes. *Diabetes Care*, *37*(Supplement 1), S14-S80.

Anderson, R. J., Freedland, K. E., Clouse, R. E., & Lustman, P. J. (2001). The prevalence of comorbid depression in adults with diabetes a meta-analysis. *Diabetes Care*, *24*(6), 1069-1078. doi:10.2337/diacare.24.6.1069

Antinori-Lent, K. (2013). Improving insulin adherence in diabetes care. *American Nurse Today*, *8*(9), 13-15.

Austin, R.P. (2006). Polypharmacy as a risk factor in the treatment of type 2 diabetes. *Diabetes Spectrum*, *19*(1), 13-16. doi: 10.2337/diaspect.19.1.13

Baghurst, T. & Kelley, B. C. (2014). An examination of stress in college students over the course of a semester. *Health Promotion Practice*, *15*(3), 438-447. doi:10.1177/1524839913510316

Baker, E. A., Schootman, M., Barnidge, E., & Kelly, C. (2006). The role of race and poverty in access to foods that enable individuals to adhere to dietary guidelines. *Preventing Chronic Disease*, *3*(3), 1-11.

Bandura, A. (1997). *Self efficacy: The exercise of control*. New York: W. H. Freeman.

Baucom, K. J. W., Queen, T. L., Wiebe, D. J., Turner, S. L., Wolfe, K. L. Godbey, E. I., … & Berg, C. A. (2015). Depressive symptoms, daily stress, and adherence in late adolescents with diabetes. *Heath Psychology*, *34*(5), 522-530. doi:10.1037/hea0000219

Berger, B., Stenstrom, G., & Sundkvist, G. (2000). Random c-peptide in the classification of diabetes. *Scandinavian Journal of Clinical and Laboratory Investigation*, *60*(8), 687-693. doi:10.1080/00365510050216411

Bone, R. (2015). Big babies: An exploration of gestational diabetes. *International Journal of Childbirth*, *30*(3), 42-46.

Brown, R. J., Wijewickrama, R. C., Harlan, D. M., & Rother, K. I. (2011). Uncoupling intensive insulin therapy from weight gain and hypoglycemia in type 1 diabetes. *Diabetes Technology & Therapeutics*, *13*(4), 457.

Boyle, J. P., Thompson, T. J., Gregg, E. W., Barker, L. E., & Williamson, D. F. (2010). Projection of the year 2050 burden of diabetes in the US adult population: Dynamic, modeling of incidence, mortality, and prediabetes prevalence. *Population Health Metrics*, 8(29), 3-12. doi:10.1186/1478-7954-8-29

Cannon, W. (1935). Stresses and the strains of homeostasis. *The American Journal of the Medical Sciences*, *189*(1), 1-14.

Centers for Disease Control (2014a). Diabetes 101. Retrieved from http://www.cdc.gov/diabetes/basics/index.html

Centers for Disease Control (2014b). The National Diabetes Statistics Report. Retrieved from http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf

Chaput, J., Klingenberg, L., Astrup, A., & Sjodin, A. (2011). Modern sedentary activities promote overconsumption of food in our current obesogenic environment. *Obesity Reviews*, *12*(5), 12-20. doi:10.1111/j.1467-789X.2010.00772.x

Chiodini, I., Adda, G., Scillitani, A., Coletti, F., Morelli, V., Di Lembo, S., ... & Ambrosi, B. (2007). Cortisol secretion in patients with type 2 diabetes relationship with chronic complications. *Diabetes Care*, *30*(1), 83-88.

Christensen, N. J. (1974). Plasma norepinephrine and epinephrine in untreated diabetics, during fasting and after insulin administration. *Diabetes*, *23*(1), 1-8.

Ciechanowski, P. S., Katon, W. J., & Russo, J. E. (2000). Depression and diabetes: Impact of depressive symptoms on adherence, function, and costs. *Archives of Internal Medicine*, *160*(21), 3278-3285.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, 24(2), 385-396.

Culig, J., & Leppée, M. (2014). From Morisky to Hill-bone; self-reports scales for measuring adherence to medication. *Collegium Antropologicum*, *38*(1), 55-62.

The DCCT Research Group (1988). Weight gain associated with intensive therapy in the diabetes control and complications trial. *Diabetes Care*, *11*(7), 567-573. doi:10.2337/diacare.11.7.567

De Vriendt, T., Clays, E., Huybrechts, I., De Bourdeaudhuij, I., Moreno, L. A., Patterson, E., & ... De Henauw, S. (2012). European adolescents' level of perceived stress is inversely related to their diet quality: the Healthy Lifestyle in Europe by Nutrition in Adolescence study. *The British Journal of Nutrition*, *108*(2), 371-380. doi:10.1017/S0007114511005708

Dewald, J. F., Meijer, A. M., Oort, F. J., Kerkhof, G. A., & Bögels, S. M. (2014). Adolescents' sleep in low-stress and high-stress (exam) times: A prospective quasi-experiment. *Behavioral Sleep Medicine*, *12*(6), 493-506. doi:10.1080/15402002.2012.670675

DiMatteo, M. (2004). Variations in patients' adherence to medical recommendations: A quantitative review of 50 years of research. *Medical Care, 42*(3), 200-209. doi:10.2147/DSMO.S36368

DiMatteo, M., Haskard-Zolnierek, K. & Martin, L. (2012). Improving patient adherence: A three-factor model to guide practice. *Health Psychology Review*, *6*(1), 74-91.

England, C. Y., Andrews, R., Jago, R., & Thompson, J. L. (2014). Changes in reported food intake in adults with type 2 diabetes in response to a nonprescriptive dietary intervention. *Journal of Human Nutrition & Dietetics*, *27*(4), 311-321. doi:10.1111/jhn.12154

Evans, M. L., Pernet, A., Lomas, J., Jones, J., & Amiel, S. A. (2000). Delay in onset of awareness of acute hypoglycemia and of restoration of cognitive performance during recovery. *Diabetes Care*, *23*(7), 893-897.

Fährmann, E. R., Adkins, L., Loader, C. J., Han, H., Rice, K. M., Denvir, J., & Driscoll, H. K. (2015). Severe hypoglycemia and coronary artery calcification during the diabetes control and complications trial/epidemiology of diabetes interventions and complications (DCCT/EDIC) study. *Diabetes Research & Clinical Practice*, *107*(2), 280-289. doi:10.1016/j.diabres.2014.10.007

Farsaei, S., Sabzghabaee, A. M., Zargarzadeh, A. H., & Amini, M. (2011). Adherence to glyburide and metformin and associated factors in type 2 diabetes in Isfahan, Iran. *Iranian Journal of Pharmaceutical Research*, *10*(4), 933-939.

Frier, B. M., Schernthaner, G., & Heller, S. R. (2011). Hypoglycemia and cardiovascular risks. *Diabetes Care*, *34*(Supplement 2), S132-S137.

Greene, J. A., Choudhry, N. K., Kilabuk, E., & Shrank, W. H. (2011). Online social networking by patients with diabetes: A qualitative evaluation of communication with Facebook. *Journal of General Internal Medicine*, *26*(3), 287-292. doi:10.1007/s11606-010-1526-3

Grenard, J., Munjas, B., Adams, J., Suttorp, M., Maglione, M., McGlynn, E., Gellad, W. (2011). Depression and mediation adherence in the treatment of chronic disease in the United States: A meta-analysis. *Journal of General Internal Medicine*, *26* (10), 1175-1182. doi:10.1007/s11606-011-1704-y

Griffith, L. S., Field, B. J., & Lustman, P. J. (1990). Life stress and social support in diabetes: Association with glycemic control. *The International Journal of Psychiatry in Medicine*, *20*(4), 365-372.

Groot, M., Doyle, T., Kushnick, M., Shubrook, J., Merrill, J., Rabideau, E., & Schwartz, F. (2012). Can lifestyle interventions do more than reduce diabetes risk?: Treating depression in adults with type 2 diabetes with exercise and cognitive behavioral therapy. *Current Diabetes Reports*, (2), 157-166.

Haskard-Zolnierek, K. B., & DiMatteo, M. R. (2009). Physician communication and patient adherence to treatment: a meta-analysis. *Medical Care*, *47*(8), 826.

Ismail, K., Winkley, K., & Rabe-Hesketh, S. (2004). Systematic review and meta-analysis of randomised controlled trials of psychological interventions to improve glycaemic control in patients with type 2 diabetes. *The Lancet*, *363*(9421), 1589-1597.

Katon, W., Fan, M. Y., Unützer, J., Taylor, J., Pincus, H., & Schoenbaum, M. (2008). Depression and diabetes: a potentially lethal combination. *Journal of General Internal Medicine*, *23*(10), 1571-1575. doi:10.1016/j.amjmed.2008.09.008

Kramer, J. R., Ledolter, J., Manos, G. N., & Bayless, M. L. (2000). Stress and metabolic control in diabetes mellitus: Methodological issues and an illustrative analysis. *Annals of Behavioral Medicine*, *22*(1), 17-28.

Kongkaew, C., Scholfield, C., Jampachaisri, K., & Chaturongkul, C. (2014). Depression and adherence to treatment in diabetic children and adolescents: A systematic review and meta-analysis of observational studies. *European Journal of Pediatrics*, *173*(2), 203-212. doi:10.1007/s00431-013-2128-y

Labig, C., Zantow, K., & Peterson, T. (2005) Factors affecting students' medicine-taking habits. *Journal of American College Health*, *54*(3), 177-183.

Lai-Ming Hui, C., Wing-Yan Poon, V., Shuk-Kuen Kwok, V., Chang, W., Kit-Wa Chan, S., Ho- Ming Lee, E., & Yu-Hai Chen, E. (2015). Prevalence and predictors of medication non-adherence among Chinese patients with first-episode psychosis. *Psychiatry Research*, 228(3), 680-687. doi:10.1016/j.psychres.2015.04.047

Lehane, E., & McCarthy, G. (2007). Intentional and unintentional medication non-adherence: A comprehensive framework for clinical research and practice? A discussion paper. *International Journal of Nursing Studies*, *44*(8), 1468-1477. doi:10.1016/j.ijnurstu.2006.07.010

Lindmark S., Buren, J., Eriksson, J. W. (2006) Insulin resistance, endocrine function and adipoines in type 2 diabetes patients at different glycaemic levels: Potential impact for glucotoxicity in vivo. *Clincial Endocrinology*, *65*(3), 301-309. doi:10.111/j.1365-2265.2006.02593.x

Maahs, D. M., West, N. A., Lawerence, J. M., & Mayer-Davis, E. J. (2010). Chapter 1: Epidemiology of type 1 diabetes. *Endocrinology and Metabolism Clinics of North America*, *39*(3), 481-497. doi:10.1016/j.ecl.2010.05.011

Magnabosco, P., Teraoka, E. C., de Oliveira, E. M., Felipe, E. A., Freitas, D., & Marchi-Alves, L. M. (2015). Comparative analysis of non-adherence to medication treatment for systemic arterial hypertension in urban and rural populations. *Revista Latino-Americana de Enfermagem*, *20*(1), 20-27. doi:10.1590/0104-1169.0144.2520

Mahler, C., Hermann, K., Horne, R., Ludt, S., Haefeli, W., Szecsenyi, J., & Jank, S. (2010). Assessing reported adherence to pharmacological treatment recommendations. Translation and evaluation of the Medication Adherence Report Scale (MARS) in Germany. *Journal of Evaluation in Clinical Practice*, *16*(3), 574-579. doi:10.1111/j.1365-2753.2009.01169.x

Martin, L. R., Haskard-Zolnierek, K. B., & DiMatteo. (2010). Evidence-based guidelines for improving healthcare. New York, New York: Oxford University Press.

Maslow, A. H. (1943). A Theory of Human Motivation. *Psychological Review, 50(4)*, 370-96.

Merz, E. L., Roesch, S. C., Malcarne, V. L., Penedo, F. J., Llabre, M. M., Weitzman, O. B., ... & Johnson, T. P. (2014). Validation of interpersonal support evaluation list-12 (ISEL-12) scores among English-and Spanish-speaking Hispanics/Latinos from the HCHS/SOL Sociocultural Ancillary Study. *Psychological Assessment*, *26*(2), 384. doi:10.1037/a0035248

Miller, T. A., & DiMatteo, M. R. (2013). Importance of family/social support and impact on adherence to diabetic therapy. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy*, *6*, 421-426. doi:10.2147/DMSO.S36368

Miller-Hagan, R. & Jana, B. (2002). Drinking perceptions and management strategies of college students with diabetes. *The Diabetes Educator*, *28*(2), 233-244. doi:10.1177/014572170202800209

Morris, A. D., Boyle, D. I., McMahon, A. D., Greene, S. A., MacDonald, T. M., Newton, R. W., & DARTS/MEMO Collaboration. (1997). Adherence to insulin treatment, glycaemic control, and ketoacidosis in insulin-dependent diabetes mellitus. *The Lancet*, *350*(9090), 1505-1510.

National Institutes of Diabetes and Digestive and Kidney Diseases. (2013). What I need to know about diabetes medicines. Retrieved from: http://www.niddk.nih.gov/health-information/health-topics/Diabetes/diabetes-medicines/Pages/index.aspx#sideeffects

Nau, D. P., Steinke, D. T., Williams, L. K., Austin, R., Lafata, J. E., Divine, G., & Pladevall, M. (2007). Adherence analysis using visual analog scale versus claims-based estimation. *Annals of Pharmacotherapy*, *41*(11), 1792-1797.

Ng, C. S., Lee, J. C., Toh, M. S., & Yu, K. (2014). Cost-of-illness studies of diabetes mellitus: A systematic review. *Diabetes Research & Clinical Practice*, *105*(2), 151-163. doi:10.1016/j.diabres.2014.03.020

Patel, B., Oza, B., Patel, K., Malhotra, S., & Patel, V. (2014). Health related quality of life in type-2 diabetic patients in Western India using World Health Organization Quality of Life - BREF and appraisal of diabetes scale. *International Journal of Diabetes in Developing Countries*, *34*(2), 100-107. doi:10.1007/s13410-013-0162-y

Piette, J. D. & Kerr, E. A. (2006). The impact of comorbid chronic condition on diabetes care. *Diabetes Care*, *29*(3), 725-731.

Pop, A., Clenciu, D., Anghel, M., Radu, S., Socea, B., Mota, E., & ... Panduru, N. (2016). Insulin resistance is associated with all chronic complications in type 1 diabetes. *Journal of Diabetes*, *8*(2), 220-228. doi:10.1111/1753-0407.12283

Prentki, M. & Nolan, C. (2006). Islet β cell failure in type 2 diabetes. *Journal of Clinical Investigation*, *116*(7). 1802-1812. doi:10.1172/JCI29103.

Ramchandani, N., Cantey-Kiser, J., Alter, C., Brink, S., Yeager, S., Tamborlane, W., & Chipkin, S. (2000). Self-reported factors that affect glycemic control in college students with type 1 diabetes. *The Diabetes Educator*, *26*(6), 656-666. doi:10.177/01457217000260413

Richardson, A. S., Arsenault, J. E., Cates, S. C., & Muth, M. K. (2015). Perceived stress, unhealthy eating behaviors, and severe obesity in low-income women. *Nutrition Journal*, 14, 1-10. doi:10.1186/s12937-015-0110-4

Rifulla, M., John, L. J., Sreedaran, J., Muttappallymyalil, J., & Basha, S. A. (2014). Patients' adherence to anti-diabetic medications in a hospital at ajman, UAE. *Malaysian Journal of Medical Sciences*, *21*(1), 44-49.

Roblin, D. W. (2011). The potential of cellular technology to mediate social networks for support of chronic disease self-management. *Journal of Health Communication*, *16,* 59-76.

Rosenthal, E. (2014). Even small medical advances can mean big jumps in bills. *The New York Times*. A1.

Rosland, A. M., Kieffer, E., Israel, B., Cofield, M., Palmisano, G., Sinco, B., … Heisler, M. (2008). When is social support important?: The association of family support and professional support with specific diabetes self-management behaviors. *Journal of General Internal Medicine*, *23*(12), 1992–1999. doi:10.1007/s11606-008-0814-7

Schmidt, S., Mühlan, H., & Power, M. (2006). The EUROHIS-QOL 8-item index: psychometric results of a cross-cultural field study. *The European Journal of Public Health*, *16*(4), 420-428. doi:10.1093/eurpub/cki155

Selye, Hans. (1956). The stress of life. New York, New York: McGraw-Hill Book Company.

Serrabulho, M. L., Gaspar de Matos, M., Nabais, J. V., & Raposo, J. F. (2014). Lifestyles and health behaviours of young adults with type 1 diabetes. *European Diabetes Nursing*, *11*(2), 49-52. doi:10.1002/edn.248

Skitch, S. A., & Abela, J. Z. (2008). Rumination in response to stress as a common vulnerability factor to depression and substance misuse in adolescence. *Journal of Abnormal Child Psychology*, *36*(7), 1029-1045. doi:10.1007/s10802-008-9233-9

Spitzer, R. L., Kroenke, K., Williams, J. B., & Patient Health Questionnaire Primary Care Study Group. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary care study. *Journal of the American Medical Association*, *282*(18), 1737-1744. doi:10.1001/jama.282.18.1737

Stene, L., & Rewers, M. (2012). Immunology in the clinic review series; focus on type 1 diabetes and viruses: The enterovirus link to type 1 diabetes: Critical review of human studies. *Clinical and Experimental Immunology*, *168*(1), 12-23. doi:10.1111/j.1365- 2249.2011.04555.x

Sultan, S., Epel, E., Sachon, C., Vaillant, G., & Hartemann-Heurtier, A. (2008). A longitudinal study of coping, anxiety and glycemic control in adults with type 1 diabetes. *Psychology & Health*, *23*(1), 73-89. doi:10.1080/14768320701205218

Surwit, R. S., Van Tilburg, M. A., Zucker, N., McCaskill, C. C., Parekh, P., Feinglos, M. N., ... & Lane, J. D. (2002). Stress management improves long-term glycemic control in type 2 diabetes. *Diabetes Care*, *25*(1), 30-34.

Tamir, O., Wainstein, J., Abadi-Korek, I., Horowitz, E., & Shemer, J. (2012). The patient-perceived difficulty in diabetes treatment (PDDT) scale identifies barriers to care. *Diabetes/Metabolism Research and Reviews*, *28*(3), 246-251. doi:10.1002/dmrr.1300

Thompson, K., Kulkarni, J., & Sergejew, A. A. (2000). Reliability and validity of a new Medication Adherence Rating Scale (MARS) for the psychoses. *Schizophrenia Research*, *42*(3), 241-247.

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, 53(4), 865-871. doi:10.1016/S0022-3999(02)00429-4

Tunceli, O., Wade, R., Gu, T., Bouchard, J. R., Aagren, M., & Luo, W. (2010). Cost of diabetes: comparison of disease-attributable and matched cohort cost estimation methods. *Current Medical Research & Opinion*, *26*(8), 1827-1834. doi:10.1185/03007995.2010.488544

Walker, R. J., Smalls, B. L., Hernandez-Tejada, M. A., Campbell, J. A., & Egede, L. E. (2014). Effect of diabetes self-efficacy on glycemic control, medication adherence, self-care behaviors, and quality of life in a predominantly low-income, minority population. *Ethnicity & Disease*, *24*(3), 349-355.

Wallston, K. A., Rothman, R. L., & Cherrington, A. (2007). Psychometric properties of the perceived diabetes self-management scale (PDSMS). *Journal of Behavioral Medicine*, *30*(5), 395-401.

Yazigi, A., & Andreelli, F. (2007). Insulin therapy and weight gain. / Insuline et prise de poids. *Cahiers De Nutrition Et De Diététique*, *42*(1), 31-35. doi:10.1016/S0007-9960(07)88697-4

Zhou, K. A., Donnelly, L. A., Morris, A. A., Franks, P. A., Jennison, C. A., Palmer, C. A., & ... Lund University, F. P. (2014). Clinical and genetic determinants of progression of type 2 diabetes: A DIRECT study. *Diabetes Care*, *37*(3), 718-724. doi:10.2337/dc13-1995