NUMBER OF COMORBIDITIES, PSYCHOSOCIAL FACTORS, AND TYPE 2 DIABETES-RELATED OUTCOMES: A MODERATED MEDIATION ANALYSIS

Emily Rose Nepacina San Diego

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NUMBER OF COMORBIDITIES, PSYCHOSOCIAL FACTORS, AND TYPE 2 DIABETES-RELATED OUTCOMES: A MODERATED MEDIATION ANALYSIS

by

Emily Rose N. San Diego

A Dissertation
Submitted in Partial Fulfillment of the
Requirements for the Degree of
Doctor of Philosophy

Major: Social and Behavioral Sciences

The University of Memphis
August 2021
DEDICATION

I dedicate this project to my mother Josephine San Diego whose resilience and selflessness inspire me every day to stay strong and continue moving forward. I am so lucky to be your daughter. I dedicate this dissertation to my brother Jan Vincent San Diego whose loving memory constantly reminds me to live my life to the absolute fullest. I also dedicate this dissertation to my father Vicente San Diego whose loving memory inspires me every day to follow my dreams. It has been a pleasure to be able to dedicate my work in your honor.
ACKNOWLEDGEMENTS

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ABSTRACT

San Diego, Emily Rose N. Ph.D. The University of Memphis. August 2021. Number of Comorbidities, Psychosocial Factors and Type 2 Diabetes-Related Outcomes: A Moderated Mediation Analysis.

Major Professor: Kenneth D. Ward, Ph.D.

Type 2 Diabetes (T2D) is an important public health concern that is associated with excess morbidity and mortality in the US. Understanding factors that influence T2D self-management and clinical outcomes is important for improving T2D-related outcomes. This dissertation used the Healthy Eating and Active Living in the Spirit (HEALS) study baseline data to examine the associations between number of comorbidities, psychosocial well-being (depressive symptoms, discrimination stress), fatalism, T2D-related health behaviors (diet, physical activity), and clinical outcomes (hemoglobin A1C [A1C], C-reactive protein [CRP], interleukin-6 [IL-6]), and the buffering effect of social support in these relationships in a sample of 106 African American adults with T2D. Moderated mediation analyses revealed that at different levels of social support, there was no significant indirect association between number of comorbidities and fatalism through psychosocial well-being, and no significant direct association between psychosocial well-being and fatalism. At different levels of social support, there was no significant indirect association between psychosocial well-being and health behaviors through fatalism, and no significant direct association between fatalism and health behaviors. There was no significant indirect association of fatalism and clinical outcomes through health behaviors at different levels of social support. There also was no significant direct association between physical activity and clinical outcomes at different levels of social support. However, greater social support was directly associated with lower CRP ($b = -61.338, p = 0.029$) and lower IL-6 ($b = -15.705, p = 0.040$). A more pro-inflammatory diet was also associated elevated IL-6 only for
individuals with low ($b = 0.247, p = 0.034$) and moderate ($b = 0.220, p = 0.049$) social support but not for individuals with high level of social support ($b = 0.193, p = 0.098$). Findings suggest improving dietary behaviors should be considered within the context of social support, as this appears to be a modifying factor for IL-6. Future studies should examine the impact of different types of social support (i.e., informal vs. formal social support; emotional support, instrumental support, informational support; positive vs. negative support, religiosity as a source of support) in these associations.
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CHAPTER 1: INTRODUCTION

Type 2 Diabetes (T2D) is a major public health concern, with race/ethnic minorities being disproportionately affected. Despite medical advances and prevention efforts, T2D remains a significant public health crisis with increasing economic burden. The disease burden is especially high for individuals with unmanaged T2D due to higher risk for developing complications (e.g., cardiovascular disease, kidney failure, hypertension, blindness, and lower limb amputation) (American Diabetes Association [ADA], 2020a; Centers for Disease Control and Prevention [CDC], 2020c). Therefore, examining strategies for improving T2D self-management may help decrease the burden of T2D in the US.

Following T2D self-management protocols can decrease or prevent the risk for developing T2D-related complications (ADA, 2020b; CDC, 2020c). Protocols for T2D self-management include blood glucose monitoring, blood pressure control, preventive care for eyes, feet, and kidneys, exercise, medication adherence, and following specific dietary recommendations (ADA, 2020b). Achieving the recommended goals for T2D clinical outcomes (i.e., hemoglobin A1C, cholesterol, blood pressure) is also important to prevent the progression of the disease and reduce the risk of complications (ADA, 2020b). However, low adherence to T2D self-management protocols as well low attainment of recommended clinical goals is prevalent among individuals with T2D. Thus, understanding factors that influence adherence as well as factors that inhibit (or enable) one from achieving the recommended clinical goals may be important for improving T2D-related outcomes.

Poor T2D-related outcomes has been associated with a greater number of comorbidities, poorer psychosocial well-being (i.e., greater depressive symptoms, higher discrimination stress), and higher fatalism (Aguilar-Zavala, Garay-Sevilla, Malacara, & Pérez-Luque, 2008; Campbell
et al., 2020; San Diego, Ward, Pichon, West, & Harmon, under review). There also is a small number of studies examining direct and indirect associations between number of comorbidities, psychosocial well-being, fatalism, and T2D-related outcomes (Egede & Osborn, 2010; Osborn & Egede, 2010; Sukkarieh-Haraty, Egede, Abi Kharma, & Bassil, 2018a; Walker, Gebregziabher, Martin-Harris, & Egede, 2014), but the underlying mechanisms remain to be clarified. Furthermore, the empirical work on fatalism and T2D self-management is relatively small and factors that increase risk for fatalism are not well understood. While social support has been shown as protective against low participation to T2D self-management and worse outcomes (Strom & Egede, 2012), its buffering effect on the associations between number of comorbidities, psychosocial well-being, fatalism, and T2D-related outcomes has never been examined.

Therefore, the overarching goal of the study is to address gaps in the literature by examining the associations between number of comorbidities, psychosocial well-being (i.e., depressive symptoms, discrimination stress), fatalism, and T2D-related outcomes (i.e., hemoglobin A1C, dietary inflammation), and the buffering effect of social support in these relationships. To provide context for this dissertation, the background and types of diabetes are discussed. Second, the burden of T2D, T2D disparities, T2D self-management protocols, T2D clinical outcomes, prevalence of adherence to T2D self-management protocols, and prevalence of achieving the recommended T2D clinical goals are discussed. Third, a review of the literature for the associations and mechanisms for number of comorbidities, depressive symptoms, stress (i.e., perceived stress, diabetes distress, discrimination stress), fatalism, and T2D self-management and clinical outcomes are provided. Fourth, the guiding theoretical model called the Stress-Buffering Effect of Social Support and a review of the literature on social support and
T2D-related outcomes are discussed. Fifth, the proposed theoretical framework, study aims and hypotheses, methodology, and analytic plan are provided. Sixth, the results are presented and discussed. Finally, the limitations, strengths, and implications of the current study are discussed.
CHAPTER 2: LITERATURE REVIEW

Diabetes

Diabetes is a common and serious chronic health condition that is associated with excess morbidity and mortality in the US (CDC, 2020c). It is a result of having high blood glucose levels in the body due to problems in the way the body metabolizes digested carbohydrates (Wood & Peters, 2018). The bloodstream carries glucose to cells throughout the body to use for energy. To help glucose get into the cells, β-cells in the pancreas secrete the hormone insulin (Wood & Peters, 2018). However, individuals with diabetes are unable to produce enough insulin or the body resists the action of insulin resulting in glucose remaining in the blood, which leads to significant damage of bodily tissues (Wood & Peters, 2018). The signs and symptoms of diabetes include being very thirsty, frequent urination, feeling very hungry, significant fatigue, losing weight without trying, sores that heal slowly, dry and itchy skin, significant fatigue, feelings of pins and needles or even loss of feeling in the feet, and blurred vision (CDC, 2020b).

There are several types of diabetes, which are all characterized by elevated blood glucose levels due to the body’s inability to produce insulin, insulin resistance, or both (Wood & Peters, 2018). Type 1 Diabetes (T1D), previously called insulin-dependent diabetes or juvenile diabetes, is an autoimmune disease wherein β-cells in the pancreas are destroyed, usually leading to an absolute insulin deficiency (ADA, 2017b). T1D is relatively rare, accounting for only 5% of all cases of diagnosed diabetes (CDC, 2020c). T1D is managed through monitoring blood glucose levels, insulin treatment through daily injections or an insulin pump, dietary management, regular physical activity, and controlling cholesterol and blood pressure levels (ADA, 2017b).

Gestational diabetes is defined as glucose intolerance with onset or first recognition during pregnancy and characterized by β-cell function that is insufficient to meet the body’s
insulin needs (ADA, 2020b; Buchanan, Xiang, Kjos, & Watanabe, 2007). Gestational diabetes accounts for 2% of all cases of diagnosed diabetes, and about 6%-9% of women develop it during pregnancy (CDC, 2018c). It is managed through regular physical activity, dietary management, and may also include daily blood glucose testing and insulin injections (ADA, 2020b). While gestational diabetes occurs only during pregnancy, remitting after the baby is born, a history of this condition is associated with a higher risk for developing Type 2 Diabetes (Daly et al., 2018).

Type 2 Diabetes (T2D), previously known as non-insulin-dependent diabetes, develops when there is an abnormal increased resistance to the action of insulin, and/or when the pancreas gradually loses its ability to secrete insulin (ADA, 2020b). A diagnosis of T2D is confirmed through different tests in a healthcare setting (doctor’s office or lab). Fasting Plasma Glucose (FPG) is a blood test to check a person’s blood glucose level that requires the patient to fast, or not have anything to eat or drink except water, for at least 8 hours before the test. An individual is diagnosed with T2D when the FPG result is ≥ 126 mg/dl (ADA, 2020b). The hemoglobin A1C (A1C) test is a blood test that measures the average blood glucose for the past two to three months and can be administered without fasting. An individual is diagnosed with T2D when the A1C test is ≥ 6.5% (ADA, 2020b). The Oral Glucose Tolerance Test (OGTT) is a blood test that helps the doctor determine how glucose is being processed in the body. The OGTT uses two blood glucose measurements – one taken prior to drinking a liquid glucose solution and one taken two hours later. An individual is diagnosed with T2D when the OGTT result is ≥ 200 mg/dl (ADA, 2020b).

T2D is the most common type of diabetes, accounting for about 95% of all diagnosed cases (CDC, 2020a). T2D develops subtly and gradually, and there are often no obvious
symptoms or immediate effects, leading many cases to go undiagnosed for years (ADA, 2009). The prevalence of T2D continues to increase yearly (CDC, 2020c), underscoring the importance of gaining a greater understanding of the disease.

**Type 2 Diabetes**

*T2D Burden*

T2D is a major public health concern in the US (CDC, 2020c). In 2018, there were approximately 34 million adults with T2D and 1.5 million are newly diagnosed each year (CDC, 2020c). It is ranked as the seventh leading cause of death, with approximately 277,000 deaths attributed to T2D each year (ADA, 2019b). Death rates in adults with T2D were about 1.3 to 2.0 times higher than the general population (Nwaneri, Cooper, & Bowen-Jones, 2013; Tancredi et al., 2015). Despite medical advances and prevention efforts, T2D is expected to remain a significant public health crisis in the US (Rowley, Bezold, Arikan, Byrne, & Krohe, 2017). By 2030, it is projected that the prevalence of T2D will increase by approximately 54% (~55 million people) and annual mortality attributed to T2D will increase by 38% (~385,800 deaths) (Rowley et al., 2017).

The economic burden of T2D in the US is alarmingly high. In 2017, there was an estimated $237 billion in direct costs from medical goods and services, and approximately $90 billion in indirect costs from lost workdays, restricted activity and premature mortality (ADA, 2019b; CDC, 2020c). In fact, people with T2D spend more than twice as much on medical costs than individuals without T2D (ADA, 2019b). The disease and economic burden is especially high for individuals with unmanaged T2D due to higher risk for developing short-term complications (e.g., hyperglycemia or very high blood glucose, hypoglycemia or very low blood glucose) and long-term complications (e.g., cardiovascular disease, kidney failure, hypertension,
blindness, and lower limb amputation) (ADA, 2020a; CDC, 2020c). Therefore, examining strategies for improving T2D self-management may help decrease the economic burden in the US.

**T2D Race/Ethnic Disparities**

Racial/ethnic minorities are systematically affected by T2D, with higher rates of T2D observed among race/ethnic minorities compared to Whites (CDC, 2020c). Among US adults aged 20 years or older, the prevalence of diagnosed T2D was highest among American Indians/Alaska Natives (15%), followed by Hispanics (13%), African Americans (12%), Asians (9%), and Whites (8%) (CDC, 2020c). Likewise, T2D-related complications are also high among race/ethnic minorities, including African Americans. Compared to Whites, African Americans have two- to four-times the rates of T2D-related complication including kidney failure, blindness, lower limb amputation, and amputation-related mortalities (ADA, 2020b; Bhattacharya, 2012; Bogner & de Vries, 2010). In addition, a study using national data from the Surveillance, Prevention, and Management of Diabetes network (N = 351,586) between 2007 – 2011 found that African American adults with T2D (n = 44,580) had consistently higher rates of severe hypoglycemia each year (significant increase in percentage points ranging from 0.7% - 1.3%) compared to their White counterpart (n = 171,995) (Karter et al., 2017). This large study also found a significant increase in annual severe hypoglycemic rates among African Americans during the 7-year observation period (Average annual percentage change: +4.3%; 95% CI: 2.1, 6.5) (Karter et al., 2017). A study using data from the National Health and Nutrition Examination Survey (NHANES) between 1999-2008 (N = 2,310) also found that the prevalence of early chronic kidney disease was significantly greater among African Americans with T2D than their White counterparts (p < 0.001) (Sinha et al., 2014).
It has been suggested that genetics play a role in the higher prevalence of insulin resistance among race/ethnic minorities, but the mechanism is unclear (Golden, Yajnik, Phatak, Hanson, & Knowler, 2019; Spanakis & Golden, 2013). However, it is important to note that “race” is not a biologically-defined construct, but rather a social and political one (Buchanan, Perez, Prinstein, & Thurston, 2020). Previous research also suggests that differences in T2D-related health behaviors (e.g., diet, physical activity, smoking) and social/environmental factors (e.g., socioeconomic status, neighborhood environment) by race/ethnicity also impact the high prevalence of T2D among race/ethnic minorities (Dagogo-Jack, 2003; Spanakis & Golden, 2013). Given the evidence of T2D race/ethnic disparities, a greater understanding of strategies for improving T2D self-management is especially important among race/ethnic minority groups.

*T2D Self-management*

T2D self-management refers to regular performance of a broad set of self-care behaviors (Gonzalez, Tanenbaum, & Commissariat, 2016). T2D self-management protocols depend on many factors, including disease severity, but include foot care, medication adherence and blood glucose monitoring (ADA, 2017a; CDC, 2018d; National Institute of Diabetes and Digestive and Kidney Disease [NIDDKD], 2016). To help control weight, blood glucose, blood pressure, and cholesterol, people with T2D also are encouraged to follow specific dietary recommendations including eating foods high in fiber, fruits and vegetables, and eating fewer foods that are high in sugar and salt (ADA, 2017a; CDC, 2020c; Colberg et al., 2016; NIDDKD, 2016). In addition, it is recommended that individuals with T2D engage in at least 150 minutes or more of moderate-to-vigorous intensity physical activity per week (ADA, 2017b). For most people with T2D, oral medications, insulin injections, or both also may be required to control their blood glucose levels (ADA, 2017a).
Hemoglobin A1C. One of the most commonly evaluated clinical outcomes in T2D self-management is A1C, or the estimated average blood glucose in the body (ADA, 2020b). For adults with T2D, achieving the recommended goal for A1C (7.0%) is important for optimal health as well as to prevent or reduce the risk of T2D-related complications (ADA, 2019a; Diabetes Control and Complications Trial Research Group, 1993; UK Prospective Diabetes Study (UKPDS) Group, 1998)

There is a large body of evidence linking adherence to T2D self-management protocols (diet, physical activity, medication management) and reduced A1C. In a meta-analysis of 13 randomized controlled trials (RCT; N = 605 adults with T2D), researchers found that compared to low-fiber diets, high-fiber diets significantly reduced A1C by 0.55%, although possible dietary confounders (e.g., total energy protein, carbohydrate fat, dietary composition) were not controlled for or reported in 12 of the 13 studies (Silva et al., 2013). A different meta-analysis of 9 RCTs (N = 1178 people with T2D) found a Mediterranean style diet (i.e., high consumption of vegetables, monounsaturated fatty acids, fruits, and legumes; low consumption of red or processed meat) showed greater improvements in A1C compared to control diets (e.g., high carbohydrate diet) (Huo et al., 2015). A RCT conducted to examine the benefits of both aerobic exercise and resistance training on A1C among individuals with T2D reported a statistically significant mean change of 0.34% (p = 0.03) at 9 months in the intervention group (n = 76) when compared to the non-exercise group (n = 41) (Church et al., 2010). A systematic review of 196 cross-sectional studies found higher medication adherence was associated with improved A1C in individuals with T2D (Capoccia, Odegard, & Letassy, 2015).
**Inflammation.** There has been an increasing interest in the role of inflammation in T2D-related outcomes in recent decades (Donath, 2013; Elimam, Abdulla, & Taha, 2019; Tsalamandris et al., 2019). Inflammation represents a naturally occurring acute immune response that helps heal wounds and fight infection (Libby, 2007). However, chronic inflammation is responsible for production of pro-inflammatory cytokines, as reflected by increased plasma levels of inflammatory biomarkers (e.g., C-reactive protein [CRP] and interleukin-6 [IL-6]) (Becker, Bromme, & Jucks, 2008; Mugabo, Li, & Renier, 2010; Pearson et al., 2003), and has been associated with the development and progression of many chronic conditions (Bastard et al., 2000; Pearson et al., 2003; Thun, Hneley, & Gansler, 2004), including T2D. For individuals with T2D, chronic inflammation has been associated elevated A1C (Elimam, Abdulla, & Taha, 2019; King, Mainous, Buchanan, & Pearson, 2003) and increased risk for developing complications such as cardiovascular disease, retinopathy, kidney failure, and mortality (Kengne, Batty, Hamer, Stamatakis, & Czernichow, 2012; Landman et al., 2016; Lowe et al., 2014).

**Diet and Inflammation**

Diet plays an important role in the regulation of chronic inflammation (Cavicchia et al., 2009; Cui et al., 2012), which is partly due to direct and indirect effects of nutrients and dietary patterns on components of the inflammatory response itself (Galland, 2010). High consumption of foods rich in saturated fatty acid (e.g., processed meats such as sausages, burgers, full-fat dairy products), trans-fatty acid (e.g., fried foods, baked goods), and high-glycemic index carbohydrates (e.g., white bread, white rice, starchy vegetables such as potato) have been associated with increased levels of inflammation, whereas consumption of foods high in magnesium (e.g., spinach, almonds), fiber (e.g., legumes), monosaturated fatty acid (e.g., nuts,
avocado), flavonoids (e.g., broccoli, kale) and carotenoids (e.g., carrots, squash) have been associated with decreased levels of inflammatory markers in serum (Galland, 2010).

In the context of T2D, studies have demonstrated the role of diet in reducing inflammation as measured by inflammatory biomarkers. In a randomized crossover trial ($N = 31$ individuals with T2D), researchers reported significantly greater reduction in CRP after switching from standard diet to the 8-week Dietary Approaches to Stop Hypertension (DASH) diet (i.e., diet high in fruits, vegetables, whole grains, low-fat dairy products, and low-sodium foods, and low in saturated fat, total fat, cholesterol, refined grains and sweets) compared to switching from the DASH to a standard diet (i.e., macronutrient composition of 50-60% carbohydrate, 15-20% protein, <30% total fat, and 5% of energy intake from simple sugars; -26.9 mg/L vs. -5.1 mg/L; $\Delta = -21.8, p = 0.02$) (Azadbakht, Surkan, Esmaillzadeh, & Willett, 2011). In a different RCT, researchers reported that CRP for participants in the Mediterranean diet group ($n = 102$) reduced by 37% at 1-year follow-up while remaining unchanged for participants in the low-fat diet ($n = 97$) group (Maiorino et al., 2016).

In addition, studies have examined the inflammatory potential of diet using the Dietary Inflammatory Index (DII®), a measure of diet that categorizes individuals’ diets on a continuum from maximally anti-inflammatory to maximally pro-inflammatory; lower scores indicate more anti-inflammatory diet while higher scores indicate more pro-inflammatory diet (Cavicchia et al., 2009). The DII was created and validated in the longitudinal data of the SEASONS study with CRP and has shown to significantly predict changes in CRP (Cavicchia et al., 2009). The literature on the DII within the context of T2D is small. In a large study of 70,991 French women, more pro-inflammatory diet (higher DII scores) was found to be associated with higher risk of T2D (HR = 0.85, 95% CI = 0.77, 0.94 to HR = 0.77, 95% CI = 0.69, 0.85) compared to a
more anti-inflammatory diet (lower DII scores) (Laouali et al., 2019). Similarly, a study of 1,174 Hispanic adults found that individuals with a more pro-inflammatory diet (higher DII scores) had greater odds of developing T2D compared to those with a more anti-inflammatory diet (lower DII scores) (OR = 3.02, 95% CI = 1.39, 6.58) (Denova-Gutiérrez et al., 2018).

*Physical Activity and Inflammation*

The anti-inflammatory action of physical activity has been examined in several studies, although the biological mechanism is not well defined (Ertek & Cicero, 2012; Geffken et al., 2001; Kasapis & Thompson, 2005). Nonetheless, there is evidence suggesting that greater levels of physical activity and exercise are associated with lower levels of inflammation in numerous populations (Geffken et al., 2001; Kasapis & Thompson, 2005; Lee, Sesso, Ridker, Mouton, & Stefanick, 2012; Nilsson, Bergens, & Kadi, 2018). In the context of T2D, a meta-analysis of fourteen RCTs (N = 824) found aerobic exercise to be associated with a significant decrease in CRP from baseline among individuals with T2D (Hayashino et al., 2014).

*Other Clinical Outcomes.* Other clinical outcomes that are important to monitor in T2D self-management include blood pressure, cholesterol, and body mass index (BMI). As with other T2D clinical outcomes, achieving the recommended goals for blood pressure (diastolic: <140 mmHg; systolic: <90 mmHg) and cholesterol (<130 mg/dL or 7.2 mmol/L) (ADA, 2020b; CDC, 2020c) as well as weight management (Wilding, 2014) are important to reduce or prevent the development of T2D-related complications.

Following T2D self-management protocols can help maintain or improve blood pressure, cholesterol, and BMI (ADA, 2020b). A meta-analysis of 9 RCTs (N = 1178 individuals with T2D) found that a Mediterranean-style diet led to greater reductions in BMI and body weight, total cholesterol and triglyceride, and systolic and diastolic blood pressure compared with control
diets (e.g., high-carbohydrate diet, low-fat diet) (Huo et al., 2015). A meta-analysis of 42 RCTs ($N = 2808$) also found that exercise (i.e., aerobic, resistance training, or both) was effective in improving systolic and diastolic blood pressure as well as high- and low-density lipoprotein cholesterol among adults with T2D (Hayashino, Jackson, Fukumori, Nakamura, & Fukuhara, 2012). Furthermore, a RCT of adults with T2D reported that, compared to participants in the control group (usual care; $n = 165$), those in the intervention group (counseling and daily mobile messages; $n = 165$) had a significant improvement in medication adherence as well as significant reductions in systolic blood pressure (135.8 mmHg to 126.2 mmHg; $p < 0.01$) and low-density lipoprotein cholesterol (104.1 mg/dL to 98.3 mg/dL; $p < 0.01$) from baseline to 6-month follow-up (Goruntla, Mallela, & Nayakanti, 2019).

**Prevalence of Adherence to T2D Self-management**

Although adherence to T2D self-management protocols is important for improved prognosis and outcomes, it often is sub-optimal (CDC, 2018d). In fact, low participation to T2D self-management is common, especially performance of self-care behaviors (i.e., diet, physical activity, medication management, preventive care for foot, eyes, and kidneys). For example, the 2020 National Diabetes Statistics Report stated that only 24% of US adults with T2D met the recommended goal for physical activity (CDC, 2020c). A study using data from NHANES ($n = 5800$) National Health Interview Survey (NHIS; $n = 48519$) and Behavioral Risk Factor Surveillance System (BRFSS; $n = 741,497$) from 1999 to 2016 reported that among US adults with T2D, adherence to saturated fat ($<10\%$ of total daily calories) and sodium ($<2300$ mg/day) recommendations decreased by 6.5 and 5.2 percentage points ($p < 0.05$ for trends), respectively (Fang, 2020).
Previous studies also indicate low adherence to T2D self-management among African Americans. A systematic review of 25 studies \((N = 2,808,199\) adults with T2D; cross-sectional: \(n = 18\); longitudinal: \(n = 7\)) found African Americans were less adherent to medication compared to Whites (Mayberry, Bergner, Chakkalakal, Elasy, & Osborn, 2016). A study using data from the 2003 BRFSS \((N = 21,459\) adults with T2D) found that African Americans were less likely to meet the recommended physical activity levels \((OR = 0.63; 95\% CI = 0.51, 0.79)\) and were less likely to engage in all four self-care behaviors (physical activity, fruits and vegetable consumption, home blood glucose testing, home foot examination; as measured by a composite score; \(OR = 0.56; 95\% CI = 0.36, 0.87\)) compared to Whites (Nwasuruba, Khan, & Egede, 2007).

**Prevalence of Achieving the Recommended Goals for T2D Clinical Outcomes**

Achieving the recommended goals for T2D clinical outcomes is important to prevent the progression of the disease and reduce the risk of complications (ADA, 2020b). However, there is evidence of low attainment of recommended T2D clinical goals. Using NHANES \(n = 3355\) and BRFSS \(n = 97,310\) data between 1999-2010, improvements in T2D clinical outcomes were observed in US adults with T2D, but 33\% to 59\% did not meet recommended goals for A1C, blood pressure, and cholesterol (Ali et al., 2013). More recently, the 2020 National Diabetes Statistics Report noted that only about 19\% of US adults with T2D met recommendations for A1C, blood pressure, and cholesterol between 2013 – 2016 (CDC, 2020c). Another large study \(N = 5,388\) of US adults with T2D reported that approximately 36\% had CRP of 1 – 3mg/L and 40\% had CRP of > 3.0 mg/L (Hwang et al., 2018), which corresponds with levels of borderline and high-risk for future a cardiovascular event, respectively (Pearson et al., 2003).
There are also race/ethnic disparities in management of clinical targets. In a study using NHANES data from 2003-2014 (N = 1,767 adults with T2D), African Americans (β = 0.51, p = 0.012) had a significantly increased mean A1C over time compared to Whites (Smalls, Ritchwood, Bishu, & Egede, 2020). In addition, a study using NHANES data between 1999 – 2008 (N = 2,310) found African American adults with T2D had higher adjusted odds ratio for CRP ≥ 0.2 mg/dL compared to their White counterpart (OR = 1.81; 95% CI = 1.19, 2.78) (Sinha et al., 2014).

Taken together, there is a clear link between decreased participation to T2D self-management and worse T2D-related outcomes. There is also evidence of decreased participation to T2D self-management protocols as well as low attainment of recommended clinical goals, especially among African Americans. Thus, understanding factors that influence adherence as well as factors that inhibit (or enable) one from achieving the recommended clinical goals is important for improving T2D self-management and related outcomes.

**Comorbidity**

Comorbidity is characterized as “the presence of more than one distinct condition in an individual” (Valderas, Starfield, Sibbald, Salisbury, & Roland, 2009). In the context of T2D, comorbidity is also described as any condition that affects people with T2D more often than age-matched people without T2D (ADA, 2017b). Comorbidities in individuals with T2D are prevalent (Lin, Kent, Winn, Cohen, & Neumann, 2015; Piette & Kerr, 2006), with the majority of patients having at least one comorbid chronic condition and approximately 86% having two or more (Iglay et al., 2016).

**Comorbidity and T2D.** Common comorbidities associated with T2D include BMIs > 95th percentile (89%), hypertension (68%), high cholesterol (44%), chronic kidney disease (37%),
and retinopathy (12%) (CDC, 2020c). A systematic review of 57 cross-sectional survey studies also reported that between 2007 – 2017, approximately 32% of individuals with T2D globally were affected by a cardiovascular disease including coronary artery disease and coronary heart disease (Einarson, Acs, Ludwig, & Panton, 2018). T2D is also associated with increased risk of cancers of the liver, pancreas, endometrium, colon and rectum, breast, and bladder (Suh & Kim, 2011), which may result from shared risk factors between T2D and cancer (i.e., older age, obesity, and physical inactivity) as well as other possible physiological mechanisms including hyperinsulinemia (excess levels of insulin in blood), hyperglycemia, and inflammation (Giovannucci et al., 2010). Other comorbidities associated with T2D include cognitive impairment/dementia, hearing impairment, obstructive sleep apnea, low testosterone in men, and disordered eating behaviors, among others (ADA, 2017b).

Comorbidity is associated with worse health outcomes, more complex disease management, increased hospitalizations, and increased healthcare costs (Li et al., 2013; Lin et al., 2015; Nowakowska et al., 2019; Parekh & Barton, 2010). For individuals with T2D, the presence of comorbidities can have a significant impact on disease management, especially performing self-care behaviors (Aga, Dunbar, & Kebede, 2019; Aga, Dunbar, Kebede, Higgins, & Gary, 2019). Comorbidity can also negatively impact T2D clinical outcomes. In a study using NHANES data, researchers compared glycemic control (as measured by A1C) of US adults with T2D between 1988 – 1994 (n = 612) and 1999-2004 (n = 608) and found greater improvement among those without comorbidities than those with comorbidities (p < 0.001) (Suh, Kim, Choi, & Plauschinat, 2008). This study also found that patients with nephropathy or renal insufficiency were 40% less likely to achieve controlled A1C (< 7%) than those without the comorbidity (Suh et al., 2008).
T2D patients with comorbidities also are more likely to have higher levels of depressive symptoms and worse outcomes. In a study of Hispanic adults with T2D, \((N = 1,274)\), depressive symptoms were significantly higher among those with cardiovascular disease as a comorbidity \((p < 0.001)\) and hypertension \((p = 0.002)\) than those without the comorbidity (Rivera-Hernandez, 2014). In addition, a cross-sectional study \((N = 325)\) of older adults with T2D \((\geq 65\) years old) reported that individuals with depression had more frequent self-reported hypoglycemic events \((17\% \text{ vs. } 6\%, p = 0.03)\) and were less likely to attain targets for HbA1C, blood pressure, and cholesterol \((0\% \text{ vs. } 16\%, p = 0.004)\) than those without depression (Fung et al., 2018).

**Presence of Comorbidity in African Americans with T2D.** Previous studies indicate high prevalence of comorbidity among African Americans with T2D. A study using NHANES data between 1999 – 2004 \((N = 11,492)\) reported 33% of African Americans with T2D reported also having hypertension, hyperlipidemia, and obesity (Suh, Choi, Plauschinat, Kwon, & Baron, 2010). A recent large study of adults with early-onset T2D found that African Americans \((n = 101,104)\) had a 17% (95% CI: 1.05, 1.31) significantly higher adjusted risk for atherosclerotic cardiovascular disease in the 18-39-year age-group compared to their White counterparts \((n = 505,336)\) (Dibato et al., 2021). This study also found those who hold privileged identities tend to have lower risk of experiencing three major adverse cardiovascular events (heart failure, myocardial infarction, or stroke) across all age-groups (Dibato et al., 2021). In addition, a different study of African Americans with T2D \((N = 241)\) found that having 1 – 3 comorbidities \((\beta = -2.03, p < 0.05)\) and having 4 – 9 comorbidities \((\beta = -2.49, p = 0.001)\) were associated with higher A1C compared to having no comorbidities (Campbell et al., 2020).
Psychosocial Factors

Psychosocial characteristics is commonly described as an individual’s psychological development related to one’s social and cultural environment (OED Online, 2021). “Psychosocial” pertains to the influence of social factors on one’s mind or behavior, and to the interrelation of behavioral and social factors (OED Online, 2021). In the context of health, psychosocial factors can be defined as the mediation of the effects of social structural factors on individual health, conditioned and modified by the social structures contexts in which they exist (Martikainen, Bartley, & Lahelma, 2002). While examinations of potential mechanisms underlying the relationship between psychosocial factors and T2D-related outcomes are continuously evolving, numerous investigations have indicated psychosocial factors such as depression, stress, and fatalism can impact T2D self-management and clinical outcomes.

Depression

Depression or depressive symptoms are common in individuals with T2D (ADA, 2020b; Khaledi, Haghihatdoost, Feizi, & Aminorroaya, 2019). It is a medical condition that causes extreme feelings of sadness lasting for a long time and interferes with one’s day-to-day functioning (ADA, 2017b).

Depression and T2D. People with T2D are 2 to 3 times more likely to have any depressive symptoms than people without T2D, but only 25% - 50% of individuals with T2D who have depression are diagnosed and treated (CDC, 2018b). In a meta-analysis of 42 cross-sectional studies (N = 21,351 adults with T2D), researchers also found that the prevalence of major depression in people with T2D was 11% and the prevalence of clinically relevant depression was 31% (Anderson, Freedland, Clouse, & Lustman, 2001).
There is also a large body of literature linking depression and low adherence to T2D self-management behaviors. For example, a systematic review of 27 studies \((n = 14 \text{ cross-sectional, } n = 6 \text{ RCT, } n = 2 \text{ longitudinal, } n = 2 \text{ mixed methods, } n = 2 \text{ prospective observational, } n = 1 \text{ descriptive, and } n = 1 \text{ quasi-experiment}; N = 7,266 \text{ adults with T2D})\) found a negative association between depression and adherence to diet and physical activity recommendations (Sumlin et al., 2014). In a large cross-sectional study of T2D patients with newly diagnosed depression \((n = 3,106)\), 52% had low adherence to medication in the year after their depression diagnosis, and depression was found to be associated with low adherence after controlling for baseline adherence and other confounders \((OR = 1.24; 95\% \text{ CI} = 1.13, 1.37)\) (Lunghi, Zongo, Moisan, Grégoire, & Guénette, 2017).

Depression is also linked with worse health outcomes in individuals with T2D. One population-based cross-sectional study \((N = 1,769)\) also reported that greater depressive symptoms were significantly associated with increased CRP \((B = 0.13, p < 0.001)\) in adults with newly diagnosed T2D (Laake et al., 2014). A large longitudinal study of veterans with T2D \((N = 11,525)\) reported that at baseline, the mean A1C for individuals with depression \((n = 696)\) was higher than those without depression \((n = 10,826)\), although the difference was not statistically significant \((\text{mean difference for A1C} = 0.08\%, p = 0.424)\) (Richardson, Egede, Mueller, Echols, & Gebregziabher, 2008). However, over a 9-year period, individuals with depression had statistically significantly higher mean A1C compared to individuals without depression \((\text{mean difference for A1C} = 0.13\%, p = 0.008)\) (Richardson et al., 2008).

In addition, T2D patients with comorbidities are more likely to have higher levels of depressive symptoms and worse outcomes. In a cross-sectional study of adults with T2D, \((N = 1,274)\), depressive symptoms were significantly higher among those with comorbidities of
cardiovascular disease ($p < 0.001$) and hypertension ($p = 0.002$) than those without these comorbidities (Rivera-Hernandez, 2014). In addition, a cross-sectional study ($N = 325$) of older adults with T2D ($\geq 65$ years old) reported that individuals with depression had more frequent self-reported hypoglycemic events (17% vs. 6%, $p = 0.03$) and were less likely to attain targets for A1C, blood pressure, and cholesterol (0% vs. 16%, $p = 0.004$) than those without depression (Fung et al., 2018).

**Depression in African Americans with T2D.** Depression is an important psychosocial concern among African Americans with T2D. A prospective study of 177 urban African American adults with T2D found the presence of major depression predicted increased A1C levels over time ($B = 0.91, p = 0.002$) (Musselman et al., 2014). Presence of comorbidity also has been linked with depression in African Americans with T2D. In a study using data from the National Survey of American Life study between 2001 – 2003 ($N = 603$), researchers found that number of comorbidities was positively associated with 12-month (OR = 1.38, 95% CI 1.18, 1.62) and lifetime (OR = 1.24, 95% CI = 1.06, 1.45) major depressive disorder among African American adults with T2D (Lankarani & Assari, 2015).

**Stress**

Stress is defined as the perception that a situation exceeds the psychological, social, or material resources for coping (Cohen, Kessler, & Gordon, 1995). Response to acute stress, particularly in healthy individuals, may be adaptive and typically does not impose serious health consequences (Schneiderman, Ironson, & Siegel, 2005). However, response to chronic stress can have long-term effects that can damage one’s health and impact health outcomes (Schneiderman et al., 2005).
Stress and T2D. Individuals with T2D have greater levels of perceived stress compared to those without the disease (Almawi et al., 2008), and perception of stress tend to increase as the disease duration lengthens (Aguilar-Zavala et al., 2008). This is especially problematic for individuals with T2D due to its association with the release of hormones such as epinephrine and cortisol that cause glucose to be secreted into the bloodstream, often resulting in elevated blood glucose levels (Surwit & Schneider, 1993). Performance of T2D self-behaviors is time consuming, which has been estimated to take up to two hours per day for an average adult with T2D (Russell, Suh, & Safford, 2005). The burden of financial cost related to healthcare visits, medications, and supplies can also contribute to the stress of managing the disease (Gonzalez et al., 2016). Thus, it is especially important to examine the influence of stress in T2D self-management given their indirect impact through performance of self-care behaviors (Walker et al., 2014; Walker, Gebregziabher, Martin-Harris, & Egede, 2014; Walker, Williams, & Egede, 2018). The relationship between stress and clinical outcomes in individuals with T2D also have been demonstrated. One study \((N = 615)\) of adults with T2D found greater perceived stress was associated with elevated A1C \((p < 0.01)\) (Walker, Smalls, & Egede, 2015).

Diabetes Distress and T2D. Diabetes-related stress, also referred to as diabetes distress\(^1\) and distinct from general perceived stress, is characterized as the emotional stress associated with the ongoing worries, burdens, and concerns related to managing T2D (Fisher, Gonzalez, & Polonsky, 2014; Polonsky et al., 2005). Similar to perceived stress, diabetes distress is a common and important psychosocial issue in individuals with T2D (Fisher, Hessler, Polonsky, & Mullan, 2012; Nicolucci et al., 2013). In meta-analysis of 43 observational and 12 experimental studies of individuals with T2D \((N = 36,998)\), researchers found an overall diabetes distress prevalence

\(^1\) Definition and constructs of diabetes distress are different from general perceived stress, which is important when evaluating associations with glycemic control and in clinical practice.
of 36% (as commonly measured across studies by the *Diabetes Distress Scale* [Example item: “Feeling that diabetes controls my life?” (Polonsky et al., 2005)] and *Problem Areas in Diabetes Scale* [Example item: “Feeling depressed when you think about living with diabetes?” (McGuire et al., 2010)] (Perrin, Davies, Robertson, Snoek, & Khunti, 2017).

Diabetes distress has been linked with decreased participation to T2D self-management and worse clinical outcomes. For example, one study of 615 adults with T2D found higher diabetes distress was associated with low adherence to dietary recommendations ($r = -0.33, p < 0.05$), lower consumption of fruits and low-fat foods ($r = -0.23; p < 0.05$), decreased exercise level ($r = -0.18, p < 0.05$), inadequate blood glucose testing ($r = -0.10, p < 0.05$), decreased adherence to medication ($r = -0.36, p < 0.05$), and elevated A1C ($r = 0.27, p < 0.05$) (Asuzu, Walker, Williams, & Egede, 2017). In addition, one study of adults with T2D ($N = 3,118$) found a greater risk of age- and sex-adjusted all-cause mortality among participants with elevated diabetes distress compared to those with low level of diabetes distress (HR = 1.57, 95% CI = 1.08, 2.27, $p = 0.017$) (Hayashino, Okamura, Tsujii, & Ishii, 2020).

**Diabetes distress in African Americans with T2D.** Diabetes distress is a prevalent psychosocial issue among African Americans with T2D. A study of 189 rural African American women with T2D found that participants with elevated diabetes distress had significantly lower mean medication adherence score (4.4 vs. 6.4, $p < 0.001$) and significantly higher A1C (9.5% vs. 8.6%, $p < 0.001$) compared to individuals with low level of diabetes distress (Cummings et al., 2014). One study of individuals with T2D ($N = 283$) found significantly higher mean diabetes-related distress among African Americans ($n = 79$) with T2D compared to their White counterparts ($n = 203$) ($M = 40.1, SD = 22.9$ vs. $M = 33.4, SD = 20.8, p = 0.02$) (Hausmann, Ren, & Sevick, 2010).
Discrimination Stress and T2D. Discrimination is broadly defined as the unfair or prejudicial treatment of people and groups based on characteristics such as race/ethnicity, gender, age, or sexual orientation (Pascoe & Richman, 2009). It is a type of stressor that surrounds negative attitudes or unfair treatment toward members of certain groups (Pascoe & Richman, 2009). Discrimination has been linked with worse health-related outcomes (i.e., psychological distress, depression, substance abuse, high blood pressure, low birth weight, inflammation (Cuevas et al., 2020)) in numerous populations (Harris et al., 2006; Marmot et al., 2008; Williams & Mohammed, 2009). The biological mechanisms underlying the effects of discrimination stress on health outcomes may be related to sympathetic nervous system activation and upregulation of the hypothalamic axis, increasing physiological ‘wear and tear’ and elevating the risk for cardiometabolic conditions (Goosby, Cheadle, & Mitchell, 2018). Thus, discriminatory experiences may operate like other stressors in that they activate physiological responses that adversely affect the maintenance of homeostasis (Cuevas et al., 2020).

In the context of T2D, several studies have demonstrated the link between discrimination and worse T2D-related outcomes. For example, a cross-sectional study of 148 older adults with T2D (age ≥ 65 years) found that higher perceived discrimination was associated with higher diabetes distress (Pearson’s $r$ ranging from 0.20 – 0.24, $p < 0.05$ for associations of the three discrimination measures with diabetes distress) (Williams, Clay, Ovalle, Atkinson, & Crowe, 2020). A different cross-sectional study of 185 community-dwelling adults with unmanaged T2D found that greater weight-specific discrimination was significantly associated with higher A1C after controlling for BMI, gender, race/ethnicity, and age ($F (8, 127) = 7.80, p = 0.006$) (Potter et al., 2015). Another study of 602 adults with T2D found that greater education-specific
discrimination was significantly associated with elevated A1C after adjusting for race/ethnicity, location, gender, marital status, duration of diabetes, number of school years, number of hours worked per week, income, and health status ($\beta = 0.47$, 95% CI $= 0.03$, 0.92, $p = 0.036$) (Brice Reynolds, Walker, Campbell, & Egede, 2015).

**Discrimination Stress in African Americans with T2D.** It is well-established that race/ethnic minorities, particularly African Americans, experience greater discrimination than other race/ethnic populations (Dolezsar, McGrath, Herzig, & Miller, 2014; Pascoe & Richman, 2009). In the context of T2D, one study found that African Americans with T2D ($n = 391$) had significantly higher mean scores on the race discrimination scale ($M = 1.44$, $SD = 0.72$ vs. $M = 1.09$, $SD = 0.30$, $p < 0.001$) compared to their White counterpart ($n = 293$) (Dawson, Walker, Campbell, & Egede, 2015). Similarly, a study of adults with T2D found that African Americans ($n = 74$) had significantly higher mean scores on the discrimination scale ($M = 5.73$, $SD = 6.21$ vs. $M = 0.46$, $SD = 1.46$, $p < 0.001$) compared to their White counterparts ($n = 74$) (Williams et al., 2020).

Several studies have demonstrated the link between discrimination and worse health-outcomes in African Americans with T2D. For example, one study of African American men with T2D ($N = 85$) found that perceived racial discrimination in healthcare was associated with elevated A1C ($b = 0.86$, 95% CI $= 0.01$, 1.73, $p < 0.01$) (Assari et al., 2017). A different study of African American adults with T2D ($N = 391$) reported that greater perceived discrimination was associated with higher systolic blood pressure ($\beta = 10.17$, 95% CI $= 1.13$, 19.22, $p = 0.028$) (Dawson et al., 2015)
Fatalism

Fatalism is broadly characterized as a set of beliefs that one’s health and health outcomes are outside of one’s personal control (Powe, 1995; Straughan & Seow, 1998). It has been operationalized in a variety of ways in the scientific literature, from “the generalized belief that events, such as the actions and occurrences that form an individual life, are determined by fate” to “the belief that events are predetermined or determined by an external event” (Abraído-Lanza et al., 2007; Espinosa De Los Monteros & Gallo, 2013). In the context of health, fatalism has been defined as a belief that some health issues are beyond human control regardless of personal action (Straughan & Seow, 1998) and the perception of an illness as unavoidable and untreatable (Powe, 1995; Powe & Johnson, 1995).

Fatalism also has been examined within the context of religiosity and health and has been referred to as “religious fatalism” in some studies to distinguish individuals whose belief in fatalism is largely connected to their religious beliefs and practices (Franklin et al., 2007; Franklin, Schlundt, & Wallston, 2008). Within this, fatalism has been described as the belief that an individual’s health outcomes are predetermined or purposed by a higher power such as God, and a person with fatalistic beliefs perceives health as being beyond one’s control and becomes dependent on chance, luck, fate, or God (Powe & Finnie, 2003; Straughan & Seow, 1998; Sukkarieh-Haraty et al., 2018a; Unantenne, Warren, Canaway, & Manderson, 2013). It is important to note, however, that religious fatalism does not automatically imply that individuals who describe themselves as “religious” are inherently fatalistic, or that fatalism has only religious components (Franklin et al., 2007), but rather reflects acceptance of and provides meaning for difficult life circumstances such as a diagnosis of a disease (Gullatte, Brawlet, Kinney, Powe, & Mooney, 2009). Several studies also suggest that fatalism and religiosity
influence health differently when viewed together rather than separately, which may be due to differences in control beliefs and vary across race/ethnicity (Friori, Brown, Cortina, & Antonucci, 2006; Neff & Hoppe, 1993).

Fatalism and Health-Related Outcomes. Studies in numerous populations have demonstrated the relationship between fatalism and health-related outcomes. For example, one large study of adults ($N = 5,313$) found that greater general health fatalism was associated with higher odds of hypertension (OR = 1.14, 95% CI = 1.02, 1.28) (Gutierrez et al., 2017). Data from a community sample of 300 Hispanic women also found that greater general health fatalism was significantly associated with greater cardiometabolic dysfunction ($r = 0.19, p < 0.05$), which attributed to decreased likelihood of engaging in health-enhancing behaviors (Espinosa De Los Monteros & Gallo, 2013). In the context of religious fatalism, one study using data from the 2010 Nashville Racial and Ethnic Approaches to Community Health study ($N = 16,200$) found that greater religious fatalism was significantly associated with increased fat-increasing behaviors (frequent fast-food consumption and adding fat to vegetables when cooking; $r = 0.006, p < 0.01$) (Franklin et al., 2007). While understanding the relationship between general fatalism and health is important, fatalism specific to certain health conditions may be more nuanced. Accordingly, there are several lines of research examining the association between disease-specific fatalism and disease-specific health outcomes.

Fatalism and Cancer. One of the largest literatures on fatalism in health has been within the cancer context. Within this, cancer fatalism has been more specifically defined as the belief that “death is inevitable when cancer is present” (Powe & Finnie, 2003). Cancer fatalism has been recognized as a potential barrier to participation in cancer screening, detection, and treatment (Morgan, Tyler, & Fogel, 2008; Powe & Finnie, 2003). In one study of 4,319 newly
diagnosed cancer patients, researchers found that cancer fatalism functions as a barrier to early cancer diagnosis, and higher cancer fatalism was associated with a slightly higher odds of stage IV cancer at the time of diagnosis (OR = 1.05, 95% CI = 1.02, 1.08) (Lyrtzopoulos, Liu Pang-Hsiang, Abel, Wardle, & Keating, 2015). In another study (N = 6,369) of adults using data from the 2003 Health Information National Trends Survey, the fatalistic belief that “everything causes cancer” was associated with lower odds of being a nonsmoker (OR = 0.67, 95% CI = 0.55, 0.82) and lower odds of eating five servings of fruits and vegetables (OR = 0.80, 95% CI = 0.66, 0.97) (Niederdeppe & Levy, 2007). In addition, the fatalistic belief that “there is not much a person can do to lower their chances of getting cancer” was associated with lower odds of engaging in a weekly exercise (OR = 0.71, 95% CI 0.59, 0.85) (Niederdeppe & Levy, 2007). A systematic review on cancer screening participation in Hispanic women found that 7 of the 11 cross-sectional studies (64%) reported that greater cancer fatalism was significantly associated with lower utilization of cancer screening services after controlling for age, socioeconomic status, and access to healthcare (Espinosa De Los Monteros & Gallo, 2011). In a different study, researchers reported that greater cancer fatalism was associated with lower clinical breast exam compliance in both Hispanic (n = 144; β = -0.22, 95% CI 0.53, 0.54) and White (n = 137; β = -0.31, p = 0.006) women (Flynn, Betancourt, & Ormseth, 2011).

Fatalism and T2D. There is a small but developing literature on fatalism within the context of T2D, with the majority of studies using Powe and Weinrich’s (1999) definition of “a complex psychological cycle characterized by perceptions of hopelessness, worthlessness, meaninglessness, powerlessness, and social despair” (Powe & Weinrich, 1999). Consistent with research on fatalism and other health outcomes (Powe, 1996; Roberts, Roberts, & Chen, 2000; Skolarus et al., 2012; Zuo, Zhang, Wen, & Zhao, 2020), inverse associations between fatalism
and T2D self-management have been reported. In a systematic review of 14 cross-sectional studies \((N = 4,619)\), researchers found that greater fatalism was associated with decreased performance of self-care behaviors (i.e., low medication adherence, decreased exercise level, unhealthful diet, decreased foot care) and worse clinical outcomes (i.e., elevated A1C) in adults with T2D, although mixed and null findings were also observed (San Diego et al., under review).

In addition, the majority of studies on fatalism within the context of T2D have examined fatalism as a predictor of T2D-related outcomes, although one cross-sectional study examined predictors of fatalistic beliefs related to T2D self-management (Sukkarieh-Haraty et al., 2018a). In this study of 280 adults with T2D, researchers found that age \((\beta = -0.14, 95\% \text{ CI} = -0.27, -0.002)\), BMI \((\beta = 0.35, 95\% \text{ CI} = 0.15, 0.54)\) and educational level \((\beta = -3.98, 95\% \text{ CI} = -9.89, -0.18)\) were significantly associated with fatalistic attitudes about T2D self-management (Sukkarieh-Haraty et al., 2018a). Interestingly, this study also found that each additional comorbidity was associated with lower levels of fatalistic beliefs about T2D self-management (5-point decrease on the *Diabetes Fatalism Scale-Arabic version* (Sukkarieh-Haraty, Egede, Abi Kharma, & Bassil, 2018b) \((\beta = -5.03; 95\% \text{ CI} = -9.89, -0.18; p = 0.42)\) (Sukkarieh-Haraty et al., 2018a), which is inconsistent with studies of other populations that found a positive relationship between number of comorbidities and fatalism (Franklin et al., 2007; Keeley, Wright, & Condit, 2009). Given findings from this study, further examination of risk factors for fatalistic attitudes related to T2D self-management is needed to provide clarification on the nuances of this construct.

There is a small literature on the link between psychosocial well-being and fatalism within the context of T2D, although findings are mixed, and the mechanism underlying these associations is unknown. In one cross-sectional study \((N = 615)\) of adults with T2D, researchers
found diabetes distress to have a direct effect on self-care behaviors ($r = -0.69$, $p < 0.001$), and fatalism ($r = 0.69$, $p < 0.001$) and depressive symptoms ($r = 0.60$, $p < 0.001$) to have indirect effects on A1C through diabetes distress (Asuzu et al., 2017). In a different study ($N = 615$) of adults with T2D, it was reported that while A1C and fatalism were not significantly related, elevated A1C and low medication adherence were independently associated with greater diabetes distress (Walker et al., 2014). Significant independent associations also were observed between greater perceived stress and decreased adherence to medication, and between greater depressive symptoms and lower levels of exercise (Walker et al., 2014).

Furthermore, the intersection of fatalism and religiosity in the context of T2D has been examined. In one study ($N = 183$) of adults with T2D, greater fatalism was significantly associated with elevated A1C ($\beta = 0.51$, $p = 0.01$), but the association was no longer significant after the inclusion of self-reported religiosity in the model ($\beta = 0.31$, $p = 0.13$) (Berardi et al., 2016). While this study demonstrated the role of fatalism in T2D self-management, a better understanding of the intersection between fatalism and religiosity in the T2D context is needed.

**Fatalism and African Americans with T2D.** Previous studies suggest the magnitude of fatalistic beliefs about health and health outcomes among African Americans is higher (Morgan, Fogel, Tyler, & Jones, 2010; Powe, Ross, & Cooper, 2007), especially when compared to Whites (Franklin et al., 2007; Powe, 1995). This could be due to historical (slavery, discrimination, segregation), cultural, and socioeconomic (substandard health care, poverty) factors that have influenced the life experiences of African Americans over time (Powe, 1996). Moreover, the impact of such factors as well as the subsequent perceptions of meaninglessness, hopelessness, and social despair worsen the perception of fatalism (Powe, 1996; Powe & Finnie, 2003).
Literature on fatalism in African Americans in the context of T2D is small. In one of the earliest studies in this area, Egede and Bonadonna (2003) qualitatively explored the construct of fatalism in 39 African American adults with T2D and found that consistent with Powe and Johnson’s (1995) conceptualization of fatalism, perceptions of hopelessness, meaninglessness, powerlessness, and social despair were prominent in the focus group sample (Egede & Bonadonna, 2003). In a different qualitative study, researchers explored psychosocial factors influencing T2D self-management among 31 African American adults with T2D and found that perceived helplessness over controlling their T2D as well as fatalistic attitudes about T2D-related health consequences were among the prominent themes in the focus groups (Bhattacharya, 2012). This study also reported that participants trusted that God is their healer and will always take care of them in their challenging life situations, and this belief in destiny reduced their self-motivation to make changes by themselves (Bhattacharya, 2012). Given the evidence of high fatalism among African Americans in other disease context (Morgan et al., 2010; Powe, 1996; Powe, Ross, Wilkerson, Brooks, & Cooper, 2007) as well as the high prevalence of T2D in this race/ethnic group (CDC, 2020c), a greater understanding of the impact of fatalism in T2D self-management in this population is needed.

In addition, research demonstrates that about 74% of African Americans endorse a belief in a higher power or God (Pew Research Center, 2020) and are also more likely to respond that religion is a significant factor in their lives and decision making (Franklin et al., 2007; Morgan et al., 2008; Taylor & Chatters, 1986a). Additionally, about 68% believe in a higher power or God who directly determines all or most of what happens in their lives, which is higher than the US adult population (46%) (Pew Research Center, 2020). Considering this, as well as the intersection between religiosity and fatalism (Franklin et al., 2007; Franklin et al., 2008; Morgan
et al., 2008), understanding this within the context of T2D is particularly relevant for improving T2D care among African Americans.

**Social Support**

Social support is a multifaceted experience that involves associations as well as formal and informal relationships with others (Holt-Lunstad & Uchino, 2015). It is characterized as the perception that certain individuals or a specific group will be available to provide support if needed or the actual provision of support by another (Holt-Lunstad & Uchino, 2015). Social support can be positive or negative and can be provided by different sources including healthcare professionals (formal support), and family members, friends, and peers (informal support) (van Dam et al., 2005). Different types of social support have been established, which includes emotional (e.g., positive feedback, encouragement), instrumental or tangible (e.g., money or transportation), companionship (e.g., social activities), and informational (e.g., advice or guidance) support (Holt-Lunstad & Uchino, 2015).

Decades of research have supported the connection between social support and health-related outcomes in numerous populations. In the context of T2D, social support is a particularly relevant psychosocial resource as it is widely recognized as being protective against low participation to T2D self-management and worse outcomes (Strom & Egede, 2012; van Dam et al., 2005). To better understand the role of social support in health outcomes, several models have been postulated and examined within the T2D context (Mazzella et al., 2010; Shor, Roelfs, & Yoge, 2013; Stringhini et al., 2012).
**Direct Effect of Social Support.** The *direct effect* model (see Figure 1) postulates that individuals with high levels of social support are generally in better health than people with low levels of social support, regardless of stress levels (Cohen & Willis, 1985).

![Figure 1. Direct effect model of social support on health-related outcomes (Cohen & Willis, 1985)](image_url)

Numerous investigations have demonstrated the direct effect of social support on T2D self-management and clinical outcomes. A systematic review of 37 studies (*n* = 18 cross-sectional studies; *n* = 2 longitudinal studies, *n* = 17 intervention studies), found that high levels of social support were associated with better adherence or adoption of T2D self-care behaviors (i.e., diet, exercise, footcare, medication adherence), improved T2D clinical outcomes (i.e., A1C, lower blood pressure level, lower cholesterol level), and decreased mortality (Strom & Egede, 2012). Another systematic review of 6 RCTs (*N* = 1,566) that examined different types of social support delivery in T2D self-management found that group consultations improved A1C and
lifestyle behaviors, internet-based peer support and telephone-based peer support increased physical activity, and social support groups improved knowledge and psychosocial functioning (van Dam et al., 2005).

Direct and indirect pathways also have been examined to demonstrate the role of social support in the relationship between psychosocial well-being and T2D-related outcomes. One cross-sectional study (N = 139) of adults with T2D found an indirect association of depressive symptoms on medication adherence through social support after adjusting for demographic variables (β = 0.01, p < 0.05) (Osborn & Egede, 2013). In a cross-sectional study of 208 Hispanic adults with T2D, participants who perceived greater support resources reported better T2D self-management (β = 0.40, p < 0.001) and less depression (β = -0.19, p < 0.01), and in turn, better T2D self-management and less depression were associated with better glycemic control (lower A1C; β = -0.17, p < 0.05 and β = 0.15, p < 0.05, respectively) (Fortmann, Gallo, & Philis-Tsimikas, 2011). Another cross-sectional study of 222 Chinese adults with T2D found an indirect association between social support and A1C through self-care behaviors (β = -0.04, p < 0.01) (Gao et al., 2013).

Similarly, direct and indirect pathways have been examined to demonstrate the role of social support in the relationship between fatalism and T2D-related outcomes, although the literature is small. One cross-sectional study (N = 126) of adults with T2D found a significant indirect association between lower fatalistic attitudes and better self-care behaviors through greater social support (r = 0.27, p < 0.001) (Egede & Osborn, 2010). Another cross-sectional study (N = 130) of adults with T2D also found that less fatalistic attitudes (r = -0.20, p < 0.05) and more social support (r = 0.27, p < 0.05) were independent, direct predictors of better self-
care behaviors, and through self-care behaviors, were significantly related to better glycemic control (lower A1C; \( r = -0.20, p < 0.05 \)) (Osborn & Egede, 2010).

**Stress-Buffering Model of Social Support.** A widely studied model of social support is the *stress-buffering* model (see Figure 2), which asserts that stressors have an adverse influence on health behaviors and health outcomes, but that appropriate support can be an important resource for minimizing such links (Cohen & Willis, 1985). It is also hypothesized that this stress buffering occurs through a cognitive appraisal process (interpretation of the situation and one’s coping resources), which can in turn weaken or “buffer” the normally robust association between stress and health-related outcomes (Cohen & Willis, 1985).

![Figure 2. Buffering effect of social support on stress and health-related outcomes (Cohen & Willis, 1985)](image)
Several studies have demonstrated the buffering (or moderating) effect of social support on the relationship between psychosocial well-being and T2D-related outcomes. A study using baseline data ($N = 290$ veterans with T2D) from a RCT found that greater diabetes distress was associated with lower A1C for individuals with a high level of social support ($b = -0.02; p = 0.007$) (Mizokami-Stout, Choi, Richardson, Piatt, & Heisler, 2021). A cross-sectional study ($N = 119$) of adults with T2D also reported that greater disease burden (as measured by insulin use [yes vs. no]) was significantly associated with elevated diabetes distress for individuals with low ($b = 1.07, p < 0.001$) and moderate ($b = 0.61, p < 0.01$) levels of social support satisfaction but not for those with high levels of social support satisfaction ($b = 0.22, p > 0.05$) (Baek, Tanenbaum, & Gonzalez, 2014). Similarly, they found that greater diabetes burden was associated with elevated distress for individuals with low ($b = 1.28, p < 0.01$) and average number ($b = 0.54, p < 0.001$) levels of social support but not for those with high levels of social support ($b = 0.20, p > 0.05$) (Baek et al., 2014). A cross-sectional study of 401 Thai adults with T2D also reported that the interaction between T2D self-management behaviors (i.e., glucose management, dietary control, physical activity, healthcare use) and social support (informal [e.g., family or friends, significant other] or formal [e.g., healthcare professional]) significantly predicted lower A1C ($\beta = 0.98, p = 0.009$) (Thojampa & Mawn, 2017).

To date, there have been no studies examining social support as a buffer or moderator of the relationship between fatalism and T2D-related outcomes. Previous research in non-clinical samples has suggested that brief interventions can diminish cancer-specific fatalism (Morgan, Fogel, Tyler, & Jones, 2010; Powe & Weinrich, 1999). Given that the modifiability of fatalism in the context of T2D management is still not well understood, examining buffers or potential
protective factors (i.e., social support) against fatalism may provide insight on strategies for diminishing T2D-specific fatalism and improve T2D-related outcomes.

**Social Support in African Americans with T2D.** Research has supported the important role social support plays in T2D self-management and related outcomes of African Americans with T2D. African American adults with T2D tend to rely more heavily than their White counterparts on their informal social networks to meet their disease management needs (Ford, Tilley, & McDonald, 1998a). In a cross-sectional study of 89 African American adults with T2D, satisfaction with support was positively correlated with improved diabetes-specific quality of life ($r = 0.58$, $p < 0.001$) and blood glucose monitoring ($r = 0.26$, $p < 0.05$) (Tang, Brown, Funnell, & Anderson, 2008). This study also found that positive support behavior from family members was a predictor for following a healthy eating plan ($r = 0.37$, $p < 0.01$), controlling carbohydrate intake throughout the day ($r = 0.28$, $p < 0.05$), and performing physical activity at least 30 minutes per day ($r = 0.30$, $p < 0.05$) while negative support behavior from family members was a predictor for low medication adherence ($r = -0.35$, $p < 0.01$) (Tang et al., 2008).

In addition, research has shown a positive association between religiosity and health, which has been attributed to social support provided by church and faith communities (Krause, 2006; Taylor & Chatters, 1988). *Church-based support* is a type of social support in which participation in religious activities provides people with access to social networks that include support from church leaders (e.g., clergy, pastors) and from other members of the religious organization (Kanu, Baker, & Brownson, 2008). Church-based support is especially important for African Americans. Research indicated that 45% of African Americans report that they receive help from church members often or sometimes (Taylor, Chatters, & Levin, 2004). In addition, 3 out of 4 African Americans surveyed reported that church members were either a *lot*
of help or some help to them (Taylor et al., 2004). Common types of support in African American faith communities include advice, encouragement, companionship, financial assistance, prayers, and help during illness (Krause, 2002; Levin, 1984; Taylor & Chatters, 1986a, 1986b; Taylor et al., 2004).

Several studies have examined the role of church-based social support in improving health outcomes among African Americans. A study using data from the Religion and Health in African Americans study (N = 2,370) found that greater religious-oriented social support was associated with greater intake of fruit and vegetable consumption (r = 0.13, p < 0.01) (Debnam, Holt, Clark, Roth, & Southward, 2012). In a study using data from the National Survey of American Life study (N = 2,991) of African American church goers, researchers found that greater frequency of contact with church members was associated with lower depressive symptoms (Incidence rate ratio = 0.95, 95% CI = 0.93, 0.98, p < 0.05) (Chatters, Nguyen, Taylor, & Hope, 2018).

To date, no studies have examined the role of church-based social support in T2D-related outcomes of African Americans with T2D. However, given churches are trusted sources of information and emotional support among African Americans (Krause, 2006), studies have examined the effect of church-based health promotion programs and interventions in improving T2D-related behaviors and outcomes. In a RCT of 201 African Americans from 24 churches, researchers reported that at 8-months post-treatment, participants in the special intervention arm (i.e., intensive phase consisting of 1 individual counseling visit with a church diabetes advisor, 12 group sessions, monthly phone contacts, and 3 encouragement postcards) had a significantly lower A1C compared to participants in the minimal intervention arm (i.e., standard educational pamphlets by mail) (7.4% vs. 7.8%, p = 0.009) (Samuel-Hodge et al., 2009). A pilot church-
based self-management education intervention study developed by health practitioners in collaboration with local pastors found that participants after 12 weeks post-treatment had significant improvements in medication adherence ($\chi^2 = 14.4, p = 0.006$), healthy eating ($\chi^2 = 26.67, p = 0.009$), and foot care adherence ($\chi^2 = 25.6, p = 0.003$) (Collins-McNeil et al., 2012).

**Summary**

Taken together, studies have demonstrated that having comorbidities, poor psychosocial well-being (i.e., greater depressive symptoms, higher perceived stress), and high levels of fatalism are associated with worse T2D-related outcomes (Aguilar-Zavala et al., 2008; Campbell et al., 2020; San Diego et al., under review; Sumlin et al., 2014). There is also evidence indicating social support is protective against low participation to T2D self-management and worse outcomes, with some studies indicating a main effect in which social support leads to improved outcomes (Strom & Egede, 2012), and also as a moderator, in which it buffers against the effects of other factors (e.g., depression) (Baek et al., 2014; Mizokami-Stout et al., 2021).

There is also a small literature examining mechanisms and pathways to explain the relationships among number of comorbidities, psychosocial well-being, fatalism, social support, and T2D-related outcomes (Egede & Osborn, 2010; Osborn & Egede, 2010; Sukkarieh-Haraty, Egede, Abi Kharma, & Bassil, 2018b; Walker et al., 2014), but mechanisms underlying these associations remain largely unknown. It is also difficult to ascertain whether psychosocial well-being is driven by one’s fatalistic attitudes about T2D management, or if psychosocial well-being is driving fatalism, indicating the need for further examination of factors that increase risk for fatalism. Considering these associations, as well as the buffering effect of social support, it is conceivable the presence of social support diminishes the relationship between number of
comorbidities, poor psychosocial well-being, high fatalism, and worse T2D-related outcomes (for a simplified conceptual model, see Figure 3).

**Current Study**

T2D is a major public health concern in the US as the rates continue to increase and economic burden remains high (CDC, 2020c). The disease burden is especially high for individuals with unmanaged T2D due to greater risk for developing complications (ADA, 2020b). While adherence to T2D self-management has been shown to improve T2D-related outcomes (Azadbakht et al., 2011; Capoccia et al., 2015; Church et al., 2010; Hayashino et al., 2012; Hayashino et al., 2014; Huo et al., 2015), performance of self-management protocols (Ponzo et al., 2017; Saleh, Mumu, Ara, Hafez, & Ali, 2014) as well as attainment of clinical targets is inadequate (Ali et al., 2013; Hwang et al., 2018). Therefore, understanding factors that influence adherence as well as factors that inhibit (or enable) achieving recommended clinical goals may be important in improving T2D self-management and related outcomes.

**Aims and Hypotheses**

The first aim of the study was to examine the relationship between number of comorbidities, psychosocial well-being (i.e., depressive symptoms, discrimination stress) and fatalism, and whether these associations were moderated by social support such that the association weakens at increased levels of social support. It was hypothesized that there was a conditional indirect effect of number of comorbidities on fatalism through psychosocial well-being at different levels of social support. It was also hypothesized that there was a direct effect of psychosocial well-being on fatalism at different levels of social support. (See also Figure 3 under Analytic Plan of the Methods section)
The second aim was to examine the relationship between psychosocial well-being (i.e., depressive symptoms, discrimination stress), fatalism, and health behaviors (i.e., diet, physical activity), and whether these associations were moderated by social support such that the association weakens at increased levels of social support. It was hypothesized that there was a conditional indirect effect of psychosocial well-being on health behavior through fatalism at different levels of social support. It was also hypothesized that there was a conditional direct effect of fatalism on health behavior at different levels of social support. (See also Figure 4 under Analytic Plan of the Methods section)

The third aim was to examine the relationship between fatalism, health behaviors (i.e., diet, physical activity), and clinical outcomes (i.e., A1C, CRP, IL-6), and whether these associations were moderated by social support such that the association weakens at increased levels of social support. It was hypothesized that there was a conditional indirect effect of fatalism on clinical outcome through health behavior at different levels of social support. It was also hypothesized that there was a conditional direct effect of health behavior on clinical outcome at different levels of social support. (See also Figure 5 under Analytic Plan of the Methods section)
CHAPTER 3: METHODS

Data Source

The current study will be a cross-sectional analysis using baseline data from the Healthy Eating and Active Living in the Spirit (HEALS) study, a NIH-funded community-based, lifestyle intervention designed to reduce inflammation in a population at high risk for diabetes, cardiovascular disease, and many types of cancer (NIMHD R24 MD002769; Principal investigator [PI]: James Hébert, ScD) (Hébert et al., 2013). The HEALS study was designed using community-based participatory research approaches to engage community partners from the African-American faith community and was comprised of two arms: (1) the 12-month lifestyle intervention arm was a healthy diet, physical activity, and stress reduction program that included materials such as cooking classes and recipes, tips for increasing physical activity level as part of one’s daily routine, suggestions for stress reduction, and assistance for tracking basic measurements such as weight and blood pressure; and (2) the delayed-intervention arm, which included churches establishing a monthly meeting sequence with the participants focusing on health issues, but not including nutrition, physical activity, or stress reduction,

Recruitment

During recruitment, each church committed to at least 24 months of participation to accommodate both the 12-month intervention and the year-delay if randomized into the control group. Churches were the units of randomization and the individual church members were the unit of measurement for all health-related outcomes. Churches were recruited in the Midlands of South Carolina. Recruitment methods included: word of mouth; media (TV and radio); and community liaisons with connections to area churches. Eligibility criteria included: aged ≥ 30 years; no reported cancer diagnosis or unstable comorbidities that might limit participation in the
intervention. Participants received small incentives throughout the study. The number of participants from each church ranged from 5 to 22 with a mean of ~12 subjects per church.

**Baseline Data Collection**

Data collection occurred at three time points (baseline, at 3-months, at 1-year), but the current analysis only used baseline data. Baseline data collection occurred between 2009 and 2012 in or near Columbia, SC. The intervention design and implementation have been described previously (Hébert et al., 2013). In brief, self-reported data were obtained via questionnaires and laboratory analyses of blood samples collected during clinic visits. Clinics were held in the respective churches, although make-up clinics were often held in the offices of the Cancer Prevention and Control Program in Columbia, SC. Baseline self-reported questionnaires and fasting blood sample were obtained after the first clinic visit. All procedures were approved by the University of South Carolina Institutional Review Board (IRB).

**Data Acquisition**

Data are not publicly available. An encrypted file containing the de-identified data was emailed to the author of this dissertation (ES). Only ES, dissertation committee members, and HEALS PI and data management team at University of South Carolina have access to the current study data. The University of Memphis IRB has determined the proposed cross-sectional analysis is exempt from their IRB oversight (see Appendix A).

**Measures**

*Sample Characteristics*

**Demographic Variables.** Self-reported demographic variables included age, sex, race, educational level, insurance information, employment, marital status, and family history of diabetes, heart disease, and kidney disease.
**Number of comorbidities.** Number of comorbidities was assessed using self-reported total number of diagnosed chronic diseases. Response options ranged from 0 to ≥ 4. Higher scores indicated greater number of comorbidities (e.g., cancer, hypertension).

**Psychosocial Factors**

**Depressive symptoms.** Depressive symptomatology was measured using the *Center for Epidemiologic Studies Short Depression Scale* (CESD-10) (Kohout, Berkman, Evans, & Cornoni-Huntley, 1993). It is a 10-item depression screening tool (not a diagnostic tool) that measures distress associated with depressive symptoms in the past week. An example of an item is “I was bothered by things that usually don’t bother me.” Participants responded using a four-point scale with response options ranging from 0 = rarely or none of the time to 3 = all of the time. Higher scores indicate greater distress; scoring for items 5 and 8 were reversed. It is also a valid tool for screening depression and has shown excellent internal consistency reliability (α = .70) and test-retest correlation coefficient of 0.64 in a sample of adults with T2D (Zhang et al., 2015).

**Discrimination Stress.** Discrimination stress was assessed using the *Experiences of Discrimination* (EOD) scale (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). It is a 21-item self-report questionnaire that measures the occurrence and frequency of acute forms of discrimination due to race/ethnicity, weight, and financial standing over a lifetime, such as not getting hired or getting a job, not receiving medical care, or not receiving service in a store or restaurant. Response options were “yes” or “no.” Higher scores indicated greater discrimination stress. Initial validation of the EOD indicated it is a valid tool for assessing discrimination stress and has shown excellent internal consistency reliability (α = .74) and test-retest correlation.
coefficient of 0.70 in a sample of working adults \((n = 100\) African Americans; \(n = 100\) Latinos) (Krieger et al., 2005)

**Fatalism.** Fatalism was assessed using the *Religious Health Fatalism Questionnaire* (RHFQ) (Franklin et al., 2008). It is a 17-item questionnaire that measures faith-related health beliefs across three dimensions (RHFQ subscales): divine provision, destiny, and helpless inevitability. An example of an item was “If I just pray to God about my health, He will work it out.” Participants responded to a five-point scale with response options ranging from 1 = strongly disagree to 5 = strongly agree. Higher scores indicated greater fatalistic beliefs (e.g., God is in control of one’s health). Initial validation of the RHFQ subscales demonstrated convergent and predictive validity [outcome: decreased fruit/vegetable intake, increased meat and fatty food intake, lower physical activity levels] an African American faith community sample (Franklin et al., 2008).

**Social support.** Social support was measured using the Social Support Questionnaire (SSQ) (Seeman, Gruenwald, Cohen, Williams, & Matthews, 2014). SSQ is comprised of 8 items that measure quality of relationships, which reflected the extent of social support (e.g., “How much do members of your family or friends really care about you?”) as well as the extent of social strains (e.g., “How much do members of your friends and family get on your nerves?”). Participants responded to a four-point scale with response options ranging from 1 = Not at all to 4 = A lot. Higher scores indicated greater social support; scoring for items 5 – 8 were reversed. The internal consistency ranged from \(\alpha = .75 - .83\) (Seeman et al., 2014).

**Health Behaviors**

**Diet.** Dietary intake was measured using a version of the National Cancer Institute *Food-Frequency Questionnaire* (FFQ) modified for use in South Carolina (Mayer-Davis et al., 1999).
FFQ was designed to obtain information on frequency and serving size of commonly consumed foods and beverages which were used to estimate nutrient intake.

The Dietary Inflammatory Index (DII) was applied to the FFQ data to assess dietary inflammation. The development and construct validation of the DII have been described previously (Shivappa, Steck, Hurley, Hussey, & Hébert, 2014; Shivappa, Steck, Hurley, Hussey, Ma, et al., 2014; Wirth et al., 2017). In brief, literature on associations between food parameters and inflammatory markers [i.e., C-reactive protein (CRP), interleukin (IL)-6] were examined. A total of 45 food parameters were found to have extensive literature related to at least one of these inflammatory markers and were used in determining an inflammatory effect score. The DII has been construct-validated with various inflammatory markers including CRP (Shivappa, Steck, Hurley, Hussey, Ma, et al., 2014; Wirth et al., 2014) and IL-6 (Wood, Shivappa, Berthon, Gibson, & Hebert, 2015), and has demonstrated usefulness in assessing the inflammatory potential of diets in African American populations (Wirth et al., 2017).

The overall DII score for each participant was the sum of the product of the centered proportion z-score and the inflammatory effect score for each food parameter. In this study, the energy-adjusted DII (E-DII™) was used, which adjusted food parameters for energy intake (Sokol et al., 2016; Tyrovolas et al., 2017; Wirth et al., 2016). Higher scores indicated a more pro-inflammatory diet while lower scores indicated a more anti-inflammatory diet.

Physical Activity. To assess physical activity, participants were provided Sensewear® armband monitors (www.bodymedia.com). Using software provided by Sensewear, participants’ age, date of birth, height and weight, current smoking habits, and dominant hand were used to calibrate each armband monitor. Participants were asked to wear the monitors for 7 days during periods of wakefulness, but not while sleeping. Instead, amount of time spent sleeping was

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measured using *Pittsburg Sleep Quality Index* (Carpenter & Andrykowski, 1998). Only participants with ≥ 4 days of monitoring based on armband usage were included in the study. For a day to “count” in achieving a minimum of 4 days of usable data, at least 20 of the 24 hours in any day needed to be accounted for by wearing the armband plus time reported spent sleeping (e.g., 15 hours of armband usage + 8 hours of sleep would be acceptable). The monitors provide valid assessments of total energy expenditure, intensity of physical activity, and bouts of physical activity (St-Onge, Mignault, Allison, & Rabasa-Lhoret, 2007; Welk, McClain, Eisenmann, & Wickel, 2007).

*Laboratory-Derived Clinical Data*

**A1C and Dietary Inflammatory Marker (CRP and IL-6).** During clinic visits, blood was collected in ethylenediaminetetraacetic acid (EDTA) vacutainers®, centrifuged at 3000 rpm for 20 minutes, and plasma specimens were aliquoted and stored at -80°C until they were analyzed for CRP and IL-6. Plasma cytokine levels were measured using an enzyme-linked immunosorbent assay kit (Quantikine kits DCRP00 [for CRP] and HS600B [for IL-6]) according to the manufacturer’s instructions. All samples were run in duplicate (CRP: CV = 3.9%, sensitivity = 0.022 ng/mL; IL-6: CV = 3.7%, sensitivity = 0.110 pg/mL).

**Analytic Plan**

Analyses were performed using Mplus Version 8.4.31 (Muthén & Muthén, 2020). Categorical variables were described as frequencies and percentage, while means and standard deviations were used to represent continuous variables. The detailed analytic procedures used to examine the study aims are described below. For all analytic procedures, number of comorbidities, depressive symptoms, perceived stress, social support, fatalism, energy-adjusted DII, physical activity A1C, CRP, and IL-6 were examined as continuous variables.
Covariates

For each aim, bivariate analyses were performed to determine which covariates should be included in the full model based on their association with the outcome. The following covariates were examined: age (continuous), sex (men, women), marital status (married, not married), educational level (≤ high school, some college; college), employment status (yes [full time], yes [part time], retired, not employed) and BMI (continuous). For each aim, covariates with statistically significant associations ($p \leq 0.05$) with the outcome were included in the moderated mediation analyses.

Missing data

For each aim, missing patterns of outcome variables were examined during preliminary analyses. For aim 1, there were 4 (4.8%) missing data points for fatalism. For aim 2, there were 9 (8.5%) missing data points for E-DII and 38 (35.8%) for physical activity. For aim 3, there were 8 (7.5%) missing data points for A1C, 4 (3.8%) for CRP, and 4 (3.8%) for IL-6. Maximum likelihood (ML) estimation was used in the moderated mediation analyses to account for missing data in the outcome variables because it uses the observed data to search for the parameters that yield the highest log likelihood (i.e., best fit to the observed data) (Graham & Coffman, 2012; Kline, 2016). Including the incomplete cases steers estimation toward a more accurate answer and ML effectively borrows information from the observed data to estimate the parameters of the incomplete variables (Kline, 2016).

For each aim, missing patterns for the predictor variables also were examined. Missing variables ranged from 5.7% to 35.8% with physical activity having the most missing data points. Little’s Missing Completely at Random (MCAR) tests were run for all independent variables and covariates in SPSS Version 27.0 (IBM Corp., 2020) to determine whether the missing values are
randomly distributed across the observations (Little, 1998; Little & Rubin, 2014). Data are assumed to be MCAR if the $p$-value is not statistically significant ($p > 0.05$) (Little, 1998). The results of Little’s MCAR test were used to further justify ML, which assumes MCAR or Missing at Random (Garson, 2015).

**Aim 1**

Using strategies by Preacher, Rucker, and Hayes (2017), a moderated mediation analysis was conducted to examine the relationship between number of comorbidities as predictor, psychosocial well-being (i.e., depressive symptoms, discrimination stress) as mediator, and fatalism as outcome while moderated by low, moderate, and high levels of social support (see Figure 3). Two separate models were run for each psychosocial well-being. Predictors were grand-mean centered for the construction of the interaction terms. To explore a significant interaction term, simple regression lines were computed using the “pick-a-point” approach at low (1 SD below the mean), medium (at the mean), and high (1 SD above the mean) levels of social support. The slopes ($b$’s) for low, medium, and high fatalism were inspected for statistical significance ($p < 0.05$).
**Figure 3.** Hypothesized model for the relationship between number of comorbidities, psychosocial well-being and fatalism while moderated by different levels of social support.

*Aim 2*

Using strategies by Preacher, Rucker, and Hayes (2017), a moderated mediation analysis was conducted to examine the model of the relationship between psychosocial well-being (i.e., depressive symptoms, discrimination stress) as predictor, fatalism as mediator, and health behavior (i.e., diet, physical activity) as outcome while moderated by low, moderate, and high levels of social support (see Figure 4). Four separate models were run for each psychosocial well-being and health behavior. Predictors were grand-mean centered for the construction of the interaction terms. To explore a significant interaction term, simple regression lines were computed using the “pick-a-point” approach at low (1 SD below the mean), medium (at the mean), and high (1 SD above the mean) levels of social support. The slopes (b’s) for low, medium, and high fatalism were inspected for statistical significance (p < 0.05).
**Figure 4.** Hypothesized model for the relationship between psychosocial well-being, fatalism, and health behavior while moderated by different levels of social support.

**Aim 3**

Using strategies by Preacher, Rucker, and Hayes (2017), a moderated mediation analysis was conducted to examine the full model of the relationship between fatalism as predictor, health behavior (i.e., diet, physical activity) as mediator, and clinical outcome (i.e., A1C, CRP, IL-6) as outcome while moderated by different levels of social support. Six separate models were run for each health behavior and clinical outcome. Predictors were grand-mean centered for the construction of the interaction terms. To explore a significant interaction term, simple regression lines were computed using the “pick-a-point” approach at low (1 SD below the mean), medium (at the mean), and high (1 SD above the mean) levels of social support. The slopes ($b$’s) for low, medium, and high fatalism were inspected for statistical significance ($p < 0.05$).
Figure 5. Hypothesized model for the relationship between fatalism, health behaviors, and clinical outcomes while moderated by different levels of social support.
CHAPTER 4: RESULTS

Sample Characteristics

Table 1 presents the descriptive analyses on the sample characteristics. The sample was comprised of 106 African Americans who self-reported as having a T2D diagnosis. Participants were on average 58.75 years old ($SD = 9.15$), and the majority were women (75%), had a college degree (42%), had medical insurance (92%), and were employed full-time (38%) or retired (42%). Most participants had a family history of diabetes (91%), high blood pressure (95%) and heart disease (64%) while only 24% had a family history of kidney disease. The majority were obese ($\geq 30$ kg/m$^2$) (81%), with an average body mass index of 35.60 ($SD = 7.24$). Most perceived their health as “good” (55%).
<table>
<thead>
<tr>
<th>Variable</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong> $M (SD)$</td>
<td>58.75 (9.15)</td>
</tr>
<tr>
<td><strong>Sex</strong> $n (%)</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>79 (75)</td>
</tr>
<tr>
<td>Men</td>
<td>27 (25)</td>
</tr>
<tr>
<td><strong>Education</strong> $n (%)</td>
<td></td>
</tr>
<tr>
<td>≤ High school</td>
<td>25 (24)</td>
</tr>
<tr>
<td>Some college</td>
<td>36 (34)</td>
</tr>
<tr>
<td>College</td>
<td>45 (42)</td>
</tr>
<tr>
<td><strong>Marital Status</strong> $n (%)</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>68 (64)</td>
</tr>
<tr>
<td>Not married</td>
<td>38 (36)</td>
</tr>
<tr>
<td><strong>Insurance Status</strong> $n (%)</td>
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</tr>
<tr>
<td>Yes (Private; Medicare; Medicaid; Other)</td>
<td>98 (92)</td>
</tr>
<tr>
<td><strong>Employment status</strong> $n (%)</td>
<td></td>
</tr>
<tr>
<td>Yes, full-time</td>
<td>40 (38)</td>
</tr>
<tr>
<td>Yes, part-time</td>
<td>11 (10)</td>
</tr>
<tr>
<td>Retired</td>
<td>44 (42)</td>
</tr>
<tr>
<td>No</td>
<td>10 (10)</td>
</tr>
<tr>
<td>**Family History of Disease (Yes) $n (%)</td>
<td></td>
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<tr>
<td>Diabetes</td>
<td>86 (91)</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>101 (95)</td>
</tr>
<tr>
<td>Heart disease</td>
<td>68 (64)</td>
</tr>
<tr>
<td>Kidney disease</td>
<td>25 (24)</td>
</tr>
<tr>
<td><strong>Body Mass Index</strong> $n (%)</td>
<td></td>
</tr>
<tr>
<td>Normal (18.5 – 24.9 kg/m²)</td>
<td>8 (8)</td>
</tr>
<tr>
<td>Overweight (25.0 – 29.9 24.9 kg/m²)</td>
<td>12 (11)</td>
</tr>
<tr>
<td>Obese (≥ 30 kg/m²)</td>
<td>86 (81)</td>
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<td><strong>Perceived Health</strong> $n (%)</td>
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<tr>
<td>Excellent</td>
<td>16 (15)</td>
</tr>
<tr>
<td>Good</td>
<td>57 (55)</td>
</tr>
<tr>
<td>Fair or poor</td>
<td>31 (29)</td>
</tr>
</tbody>
</table>

*M (SD) = Mean (standard deviation)*  
*n (%) = frequency (percentage)*  
*Married = married or living with a partner*  
*Not married = widowed, divorced, separated, single and never married, or single and not living with a partner*
Description of Number of Comorbidities, Psychosocial Factors, Health Behaviors, and Clinical Outcomes

Table 2 presents the descriptive analyses for number of comorbidities, psychosocial factors, health behaviors, and clinical outcomes. Most participants had 2 comorbidities (39%). Participants reported low mean depressive symptoms scores ($M = 5.90$, $SD = 4.43$, Range = 0.00 – 20.00; as measured by CES-D), low mean discrimination stress scores ($M = 4.63$, $SD = 3.42$, Range = 0.00 – 18.00; as measured by EOD), moderate mean religious fatalism scores ($M = 34.99$, $SD = 8.21$, Range = 19.45 – 57-62; as measured by RHFQ), and moderate mean social support scores ($M = 24.96$, $SD = 3.53$, Range = 15.00 – 32.00 as measured by SSS). On average, participants had a more anti-inflammatory diet ($M = -0.92$, $SD = 1.96$, Range, -5.22 – 3.98; as measured by E-DII). On average, time spent in moderate-intensity activity was below recommendations ($M = 39.79$ minutes/week, $SD = 40.45$, Range = 0.00 – 209.00). The Department of Health and Human Services Physical Activity Guidelines suggest that adults engage in at least 150 minutes/week of moderate-intensity physical activity (U.S. Department of Health and Human Services, 2018). In the current study, only 2.83% ($n = 3$) of the participants met this recommendation. Although the average A1C in the current sample was high ($M = 7.42\%$, $SD = 1.55$), there were more participants who met the A1C clinical recommendation of 7.0% or less (ADA, 2020b) than those who did not meet the clinical recommendation (56% vs. 44%). The average CRP ($M = 5.98$ mg/L, $SD = 7.06$) and IL-6 ($M = 2.30$ pg/mL, $SD = 1.53$) in the current sample were high, which corresponds with high-risk level for a future cardiovascular event (Pearson et al., 2003).
Table 2. Descriptive Analyses of Number of Comorbidities, Psychosocial Factors, Health Behaviors, and Clinical Outcomes

<table>
<thead>
<tr>
<th>Number of Comorbidities</th>
<th>n (%)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9 (8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>41 (39)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>34 (32)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 4</td>
<td>11 (21)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Psychosocial Factors

<table>
<thead>
<tr>
<th>Factor</th>
<th>M (SD)</th>
<th>Range (Min – Max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressive symptoms</td>
<td>5.90 (4.43)</td>
<td>0.00 – 20.00</td>
</tr>
<tr>
<td>Discrimination stress</td>
<td>4.63 (3.42)</td>
<td>0.00 – 18.00</td>
</tr>
<tr>
<td>Religious fatalism</td>
<td>34.99 (8.21)</td>
<td>19.45 – 57.62</td>
</tr>
<tr>
<td>Social support</td>
<td>24.96 (3.53)</td>
<td>15.00 – 32.00</td>
</tr>
</tbody>
</table>

Health Behaviors

<table>
<thead>
<tr>
<th>Behavior</th>
<th>M (SD)</th>
<th>Range (Min – Max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet (E-DII)</td>
<td>-0.92 (1.96)</td>
<td>-5.22 – 3.98</td>
</tr>
<tr>
<td>Physical activity (minutes/day)</td>
<td>39.79 (40.45)</td>
<td>0.00 – 209.00</td>
</tr>
</tbody>
</table>

Clinical Outcome

<table>
<thead>
<tr>
<th>Outcome</th>
<th>M (SD)</th>
<th>Range (Min – Max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1C (%)</td>
<td>7.42 (1.55)</td>
<td>5.60 – 13.60</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>5.98 (7.06)</td>
<td>0.29 – 35.21</td>
</tr>
<tr>
<td>IL-6 (pg/mL)</td>
<td>2.30 (1.53)</td>
<td>0.52 – 7.90</td>
</tr>
</tbody>
</table>

Categorical variables were described as frequency (n) and percentage (%), and range of scores/values.
Continuous variables were described as means (M) and (SD).

E-DII = Energy-adjusted Dietary Inflammatory Index; Lower scores on the E-DII indicated more anti-inflammatory diet and higher scores indicated a more pro-inflammatory diet.

PA = physical activity; A1C = hemoglobin A1C; CRP = C-reactive protein; IL-6 = interleukin 6

Covariates

**Aim 1.** Bivariate analyses revealed sex (p = 0.030) and educational level (p = 0.009) were positively associated with fatalism and were included as covariates in the adjusted models; age, marital status, employment status, and BMI were not significantly associated with fatalism and were not included as covariates for the adjusted model, ps > 0.05.

**Aim 2.** Bivariate analyses revealed age (p < 0.001), educational level (p = 0.045), and employment status (p = 0.005) had statistically significant positive associations with diet as outcome and were included as covariates in the adjusted models; sex, marital status, and BMI were not statistically significantly associated with diet (ps > 0.05) and were not included as
covariates in the adjusted model. In addition, bivariate analyses revealed sex ($p = 0.002$) and BMI ($p = 0.001$) had statistically significant positive associations with physical activity as outcome and were included as covariates in the adjusted model; age, educational level, employment status, and marital status were not significantly associated with physical activity ($ps > 0.05$) and were not included as covariates in the adjusted model.

**Aim 3.** Bivariate analyses revealed age ($p = 0.007$), employment status ($p = 0.029$), and BMI ($p = 0.022$) had statistically significant positive associations with A1C as outcome and were included as covariates in the adjusted model; sex, educational level and marital status were not statistically significantly associated with A1C ($ps > 0.05$) and were not included as covariates in the adjusted model. In addition, bivariate analyses revealed age ($p = 0.0150$), marital status ($p = 0.0024$) and BMI ($p = 0.001$) were statistically positively associated with CRP and were included in the adjusted model; sex, employment status, and educational were not statistically significantly associated with CRP ($ps > 0.05$) and were not included as covariates in the adjusted model. Bivariate analyses also revealed age ($p = 0.001$) and BMI ($p < 0.001$) were statistically positively associated with IL-6 and were included in the adjusted model; sex, employment status, educational level, and marital status were not statistically significantly associated with IL-6 ($ps > 0.05$) and were not included as covariates in the adjusted model.

**Missing data**

For Aim 1, Little’s MCAR (Missing Completely at Random) tests were not statistically significant ($p = 0.372 - 0.700$) for the two separate models, indicating the predictor variables and covariates were MCAR. For Aim 2, Little’s MCAR tests were not statistically significant ($p = 0.394 – 0.652$) for the four separate models, indicating the predictor variables and covariates were MCAR. For Aim 3, Little’s MCAR tests were not statistically significant ($p = 0.212 –$
0.958) for the six separate models, indicating the predictor variables and covariates were MCAR. Therefore, maximum likelihood estimation was used to replace missing values in the analyses.

**Aim 1: Examine the relationship between number of comorbidities (predictor), psychosocial well-being (mediator), and fatalism (outcome) while moderated by different levels of social support**

**Aim 1a: Depressive symptoms as a mediator**

Unstandardized parameter estimates revealed the direct effect of number of comorbidities on depressive symptoms ($b = 0.012, 95\% \text{ CI} = 0.000, 0.023, p = 0.095$) and fatalism ($b = 0.006, 95\% \text{ CI} = -0.011, 0.029, p = 0.427$) were not statistically significant after adjusting for covariates. The direct effect of depressive symptoms ($b = 0.159, 95\% \text{ CI} = -0.169, 0.663, p = 0.508$) and social support ($b = 0.427, 95\% \text{ CI} = -0.058, 0.967, p = 0.173$) on fatalism also were not statistically significant after adjusting for covariates. See Figure 6.

**Figure 6.** Moderated mediation analysis of the relationship between number of comorbidities, depressive symptoms, fatalism, and social support and diet after adjusting for covariates.
The conditional indirect effect of number of comorbidities on fatalism through depressive symptoms at low ($b = 0.003$, 95% CI = -0.002, 0.009, $p = 0.591$), moderate ($b = 0.003$, 95% CI = -0.002, 0.009, $p = 0.591$), and high ($b = 0.003$, 95% CI = -0.002, 0.009, $p = 0.590$) levels of social support were not statistically significant. The conditional effect of depressive symptoms on fatalism at low ($b = 0.159$, 95% CI = -0.158, 0.635, $p = 0.533$), moderate ($b = 0.159$, 95% CI = -0.158, 0.635, $p = 0.533$), and high ($b = 0.159$, 95% CI = -0.158, 0.635, $p = 0.533$) levels of social support also were not statistically significant. See Table 3

**Aim 1b: Discrimination stress as a mediator**

Unstandardized parameter estimates revealed that the direct effect of number of comorbidities on discrimination stress ($b = 0.005$, 95% CI = -0.003, 0.014, $p = 0.282$) and fatalism ($b = 0.104$, 95% CI = -0.008, 0.029, $p = 0.303$) were not statistically significant after adjusting for covariates. The direct effect of discrimination stress ($b = 0.104$, 95% CI = -0.535, 0.742, $p = 0.789$) and social support ($b = 0.541$, 95% CI = -0.021, 1.070, $p = 0.104$) on fatalism also were not statistically significant after adjusting for covariates. See Figure 7
Figure 7. Moderated mediation analysis of the relationship between number of comorbidities, discrimination stress, fatalism, and social support and diet after adjusting for covariates.

The conditional indirect effect of number of comorbidities on fatalism through discrimination stress at low ($b = 0.001$, 95% CI = -0.003, 0.006, $p = 0.844$), moderate ($b = 0.001$, 95% CI = -0.003, 0.006, $p = 0.845$), and high ($b = 0.001$, 95% CI = -0.003, 0.006, $p = 0.846$) levels of social support were not statistically significant. The conditional direct effect of discrimination stress on fatalism at low ($b = 0.105$, 95% CI = -0.534, 0.742, $p = 0.788$), moderate ($b = 0.104$, 95% CI = -0.535, 0.742, $p = 0.789$), and high ($b = 0.103$, 95% CI = -0.536, 0.742, $p = 0.779$) levels of social support also were not statistically significant. See Table 3.
Aim 2: Examine the relationship between psychosocial well-being (predictor), fatalism (mediator), and health behavior (outcome), and whether these associations were moderated by different levels of social support

Aim 2a: Depressive symptom as predictor

Diet. Unstandardized parameter estimates revealed the direct effect of depressive symptoms on fatalism ($b = 0.095, 95\% CI = -0.172, 0.417, p = 0.599$) and diet ($b = 3.867, 95\% CI = -5.138, 11.977, p = 0.465$) were not statistically significant after adjusting for covariates. The direct effect of fatalism ($b = -0.754, 95\% CI = -4.602, 2.860, p = 0.742$) and social support ($b = 5.957, 95\% CI = -7.686, 18.228, p = 0.453$) on diet also were not statistically significant after adjusting for covariates. See Figure 8.
**Figure 8.** Moderated mediation analysis of the relationship between depressive symptoms, fatalism, social support, and diet after adjusting for covariates.

The conditional indirect effect of depressive symptoms on diet through fatalism at low ($b = -0.072$, 95% CI = -0.918, 0.546, $p = 0.880$), moderate ($b = -0.072$, 95% CI = -0.917, 0.545, $p = 0.880$), and high ($b = -0.072$, 95% CI = -0.918, 0.543, $p = 0.879$) levels of social support were not statistically significant. The conditional direct effect of fatalism on diet at low ($b = -0.752$, 95% CI = -4.606, 2.868, $p = 0.743$), moderate ($b = -0.754$, 95% CI = -4.602, 2.860, $p = 0.742$), and high ($b = -0.756$, 95% CI = -4.609, 2.852, $p = 0.742$) levels of social support also were not statistically significant. See Table 4.

**Physical activity.** Unstandardized parameter estimates revealed that the direct effect of depressive symptoms on fatalism ($b = 0.098$, 95% CI = -0.166, 0.417, $p = 0.588$) and physical activity ($b = 0.794$, 95% CI = -0.984, 2.868, $p = 0.496$) were not statistically significant after adjusting for covariate. The direct effect of fatalism ($b = 1.733$, 95% CI = -0.077, 3.240, $p = 0.077$).
0.087) and social support ($b = -1.470$, 95% CI = -4.940, 0.952, $p = 0.429$) on physical activity also were not statistically significant after adjusting for covariates. See Figure 9.

Figure 9. Moderated mediation analysis of the relationship between depressive symptoms, fatalism, social support, and physical activity after adjusting for covariates.

The conditional indirect effect of depressive symptoms on physical activity through fatalism at low ($b = 0.170$, 95% CI = -0.202, 0.964, $p = 0.662$), moderate ($b = 0.170$, 95% CI = -0.202, 0.964, $p = 0.662$), and high ($b = 0.170$, 95% CI = -0.202, 0.963, $p = 0.662$) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effects of fatalism on physical activity at low ($b = 1.736$, 95% CI = -0.076, 3.243, $p = 0.017$), moderate ($b = 1.733$, 95% CI = -0.077, 3.240, $p = 0.017$), and high ($b = 1.731$, 95% CI = -0.076, 3.237, $p = 0.017$) levels of social support also were not statistically significant after adjusting for covariates. See Table 4.
Aim 2b: Discrimination stress as predictor

Diet. Unstandardized parameter estimates revealed that the direct effect of discrimination stress on fatalism ($b = -0.243$, 95% CI = -0.774, 0.196, $p = 0.415$) and diet ($b = 5.647$, 95% CI = -9.132, 17.577, $p = 0.619$) were not statistically significant after adjusting for covariates. The direct effect of fatalism ($b = -0.812$, 95% CI = -4.791, 2.848, $p = 0.727$) and social support ($b = 6.470$, 95% CI = -7.936, 19.819, $p = 0.445$) on diet also were not statistically significant after adjusting for covariates. See Figure 10.

Figure 10. Moderated mediation analysis of the relationship between discrimination stress, fatalism, social support, and diet after adjusting for covariates.

The conditional indirect effect of discrimination stress on diet through fatalism at low ($b = 0.197$, 95% CI = -1.157, 1.779, $p = 0.839$), moderate ($b = 0.197$, 95% CI = -1.158, 1.782, $p = 0.839$), and high ($b = 0.198$, 95% CI = -1.158, 1.780, $p = 0.839$) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effect of fatalism on
diet at low ($b = -0.810, 95\% \text{ CI} = -4.791, 2.852, p = 0.728$), moderate ($b = -0.812, 95\% \text{ CI} = -4.791, 2.848, p = 0.727$), and high ($b = -0.814, 95\% \text{ CI} = -4.789, 2.849, p = 0.726$) levels of social support also were not statistically significant after adjusting for covariates. See Table 4.

**Physical activity.** Unstandardized parameter estimates revealed that the direct effect of discrimination stress on fatalism ($b = -0.163, 95\% \text{ CI} = -0.688, 0.289, p = 0.581$) and physical activity ($b = -2.939, 95\% \text{ CI} = -7.335, 1.629, p = 0.280$) were not statistically significant after adjusting for covariates. The direct effect of fatalism ($b = 1.798, 95\% \text{ CI} = -0.139, 3.323, p = 0.089$) and social support ($b = -2.348, 95\% \text{ CI} = -5.808, 0.699, p = 0.231$) also were not statistically significant after adjusting for covariates. See Figure 11.

![Diagram](image_url)

**Figure 11.** Moderated mediation analysis of the relationship between discrimination stress, fatalism, social support, and physical activity after adjusting for covariates.

The conditional indirect effect of discrimination stress on physical activity through fatalism at low ($b = -0.293, 95\% \text{ CI} = -1.461, 0.455, p = 0.625$), moderate ($b = -0.292, 95\% \text{ CI} = -1.455, 0.465, p = 0.625$), and high ($b = -0.291, 95\% \text{ CI} = -1.460, 0.467, p = 0.625$) levels of social support also were not statistically significant after adjusting for covariates. See Table 4.
-1.458, 0.455, \( p = 0.625 \), and high \( (b = -0.292, 95\% \text{ CI} = -1.457, 0.454, p = 0.625) \) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effect of fatalism on physical activity at low \( (b = 1.800, 95\% \text{ CI} = -0.138, 3.326, p = 0.089) \), moderate \( (b = 1.798, 95\% \text{ CI} = -0.139, 3.323, p = 0.089) \), and high \( (b = 1.796, 95\% \text{ CI} = -0.140, 3.319, p = 0.089) \) levels of social support also were not statistically significant after adjusting for covariates. See Table 4.
### Table 3. Conditional Effects at Different Levels of Social Support with Number of Comorbidities as Predictor, Psychosocial Well-Being as Mediator and Fatalism as outcome

<table>
<thead>
<tr>
<th></th>
<th>Diet</th>
<th>Physical Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Psychosocial Well-Being $\rightarrow$ Health Behavior through Fatalism $^{a,c}$</td>
<td>Psychosocial Well-Being $\rightarrow$ Health Behavior through Fatalism $^{b,d}$</td>
</tr>
<tr>
<td></td>
<td>$b$</td>
<td>S.E.</td>
</tr>
<tr>
<td>Depressive Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>-0.072</td>
<td>0.475</td>
</tr>
<tr>
<td>Moderate</td>
<td>-0.072</td>
<td>0.475</td>
</tr>
<tr>
<td>High</td>
<td>-0.072</td>
<td>0.474</td>
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<tr>
<td>Discrimination Stress</td>
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<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.197</td>
<td>0.971</td>
</tr>
<tr>
<td>Moderate</td>
<td>0.197</td>
<td>0.970</td>
</tr>
<tr>
<td>High</td>
<td>0.198</td>
<td>0.970</td>
</tr>
</tbody>
</table>

$b = \text{unstandardized parameter estimate; } S.E. = \text{standard error; } CI = \text{confidence interval}$

Psychosocial well-being includes depressive symptoms and discrimination stress

Low, Moderate, and High indicated levels of social support

$^a$ Conditional *indirect effect* of number of comorbidities on fatalism through psychosocial well-being at different levels of social support

$^b$ Conditional *direct effect* of psychosocial well-being on fatalism at different levels of social support

$^c$ Covariates for adjusted models included sex and educational level
Aim 3: Examine the relationship between fatalism (predictor), health behavior (mediator), and clinical outcome (outcome) while moderated by different levels of social support

Aim 3a: Diet as a mediator

A1C. Unstandardized parameter estimates revealed that the direct effect of fatalism on diet ($b = 0.222, 95\% \text{ CI} = -3.827, 4.311, p = 0.929$) and A1C ($b = 2.470, 95\% \text{ CI} = -0.899, 6.642, p = 0.289$) were not statistically significant after adjusting for covariates. The direct effect of diet ($b = 0.119, 95\% \text{ CI} = -0.081, 0.343, p = 0.362$) and social support ($b = -1.123, 95\% \text{ CI} = -8.628, 6.606, p = 0.552$) on A1C also were not statistically significant after adjusting for covariates. However, the interaction term between diet and social support was statistically significant ($b = -0.062, 95\% \text{ CI} = -0.110, -0.010, p = 0.045$). See Figure 12.

Figure 12. Moderated mediation analysis of the association between fatalism, diet, social support, and A1C after adjusting for covariates. * denotes a statistically significant path ($p < 0.05$).
The conditional indirect effect of fatalism on A1C through diet at low \((b = 0.040, 95\% \text{ CI} = -0.740, 1.113, p = 0.945)\), moderate \((b = 0.026, 95\% \text{ CI} = -0.542, 0.930, p = 0.955)\) and high \((b = 0.012, 95\% \text{ CI} = -0.431, 0.803, p = 0.976)\) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effect of diet on A1C at low \((b = 0.181, 95\% \text{ CI} = -0.014, 0.398, p = 0.148)\), moderate \((b = 0.119, 95\% \text{ CI} = -0.081, 0.343, p = 0.362)\), and high \((b = 0.056, 95\% \text{ CI} = -0.162, 0.300, p = 0.692)\) levels of social support also were not statistically significant after adjusting for covariates. See Table 5.

CRP. Unstandardized parameter estimates (see Figure 19) revealed that the direct effect of fatalism on diet \((b = 0.638, 95\% \text{ CI} = -3.341, 4.678, p = 0.796)\) and CRP \((b = 6.540, 95\% \text{ CI} = -10.420, 25.155, p = 0.552)\) were not statistically significant after adjusting for covariates. The direct effect of diet \((b = 0.312, 95\% \text{ CI} = -0.483, 1.030, p = 0.499)\) and social support \((b = -6.682, 95\% \text{ CI} = -46.300, 35.143, p = 0.788)\) on CRP also were not statistically significant after adjusting for covariates. See Figure 13.
Figure 13. Moderated mediation analysis of the association between fatalism, diet, social support, and CRP after adjusting for BMI, age, and marital status.

The conditional indirect effect of fatalism on CRP through diet at low ($b = 0.331$, 95% CI $= -2.188, 3.015, p = 0.837$), moderate ($b = 0.199$, 95% CI $= -2.669, 2.310, p = 0.881$), and high ($b = 0.067$, 95% CI $= -2.097, 1.964, p = 0.959$) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effect of diet on CRP at low ($b = 0.518$, 95% CI $= -0.235, 1.201, p = 0.242$), moderate ($b = 0.312$, 95% CI $= -0.483, 1.030, p = 0.499$), and high ($b = 0.105$, 95% CI $= -0.780, 0.894, p = 0.838$) levels of social support also were not statistically significant after adjusting for covariates. See Table 4.

IL-6. Unstandardized parameter estimates revealed that the direct effect of fatalism on diet ($b = 0.638$, 95% CI $= -3.341, 4.678, p = 0.796$) and IL-6 ($b = 1.383$, 95% CI $= -2.261, 5.070$, $p = 0.540$) were not statistically significant after adjusting for covariates. The direct effect of social support on IL-6 also was not statistically significant after adjusting for covariates ($b = -2.957$, 95% CI $= -11.944, 8.037, p = 0.626$). However, the direct effect of diet on IL-6 was
statistically significant ($b = 0.220$, 95% CI = 0.035, 0.400, $p = 0.049$). A more pro-inflammatory diet was associated with elevated IL-6 such that a one-unit increase in the E-DII was associated with a 0.220 increase in IL-6. See Figure 14.

**Figure 14.** Moderated mediation analysis of the association between fatalism, diet, social support, and IL6 after adjusting for covariates. * denotes a statistically significant path ($p < 0.05$).

The conditional indirect effect of fatalism on IL-6 through diet at low ($b = 0.157$, 95% CI = -0.931, 1.238, $p = 0.810$), moderate ($b = 0.140$, 95% CI = -0.822, 1.112, $p = 0.812$), and high ($b = 0.123$, 95% CI = -0.726, 1.039, $p = 0.819$) levels of social support were not statistically significant after adjusting for covariates. However, the conditional direct effect of diet on IL-6 at low ($b = 0.247$, 95% CI = 0.051, 0.433, $p = 0.034$) and moderate levels ($b = 0.220$, 95% CI = 0.035, 0.400, $p = 0.049$) of social support remained statistically significant, but not for high level
of social support ($b = 0.193$, 95% CI = -0.002, 0.381, $p = 0.098$) after adjusting for covariates. A more pro-inflammatory diet was associated with elevated IL-6 only for individuals with low and moderate levels of social support. See Table 5

*Aim 3b: Physical Activity as a mediator*

A1C. Unstandardized parameter estimates revealed that the direct effect of fatalism ($b = 3.408$, 95% CI = -1.736, 10.404, $p = 0.361$), physical activity ($b = 0.040$, 95% CI = -0.997, 3.148, $p = 0.976$), and social support ($b = -8.019$, 95% CI = -21.474, 13.999, $p = 0.482$) on A1C were not statistically significant after adjusting for covariates. However, the direct effect of fatalism on physical activity was statistically significant ($b = 2.388$, 95% CI = 0.469, 4.030, $p = 0.029$) after adjusting for covariates. Greater fatalism was associated with greater physical activity such that a one-unit increase in the fatalism scale was associated with a 2.388 increase in minutes spent engaging in physical activity after adjusting for covariates. See Figure 15.
**Figure 15.** Moderated mediation analysis of the association between fatalism, physical activity, social support, and A1C after adjusting for covariates. * denotes a statistically significant path (p < 0.05).

The conditional indirect effect of fatalism on A1C through physical activity at low (b = 0.099, 95% CI = -2.896, 6.327, p = 0.973), moderate (b = 0.096, 95% CI = -2.904, 6.345, p = 0.974), and high (b = 0.092, 95% CI = -2.910, 6.365, p = 0.975) levels of social support were not statistically significant after adjusting for covariates. The direct effect of physical activity on A1C at low (b = 0.042, 95% CI = -0.994, 3.141, p = 0.975), moderate (b = 0.040, 95% CI = -0.997, 3.148, p = 0.976), and high (b = 0.039, 95% CI = -0.998, 3.154, p = 0.977) levels of social support also were not statistically significant after adjusting for all covariates. See Table 5.

**CRP.** Unstandardized parameter estimates revealed that the direct effect of fatalism (b = 14.699, 95% CI = -11.752, 43.814, p = 0.390) and physical activity (b = -6.800, 95% CI = -16.151, -1.284, p = 0.143) on CRP were not statistically significant after adjusting for covariates.
However, the direct effect of social support on CRP was statistically significant after adjusting for covariates \((b = -61.338, 95\% \text{ CI} = -111.052, -23.411, p = 0.029)\). Greater social support was associated with lower CRP such that a one-unit increase in the social support scale was associated with a 61.338 decrease in CRP after adjusting for covariates. The direct effect of fatalism also was statistically significant after adjusting for covariates \((b = 2.414, 95\% \text{ CI} = 0.559, 4.030, p = 0.023)\). Greater fatalism was associated with more physical activity such that a one-unit increase in the fatalism scale was associated with a 2.414 increase in minutes spent engaging in physical activity. See Figure 16.

![Diagram](image.png)

**Figure 16.** Moderated mediation analysis of the association between fatalism, physical activity, social support, and CRP after adjusting for covariates. * denotes a statistically significant path \((p < 0.05)\).

The conditional indirect effect of fatalism on CRP through physical activity at low \((b = -16.399, 95\% \text{ CI} = -41.439, -1.296, p = 0.205)\), moderate \((b = -16.418, 95\% \text{ CI} = -41.506, -1.303, p = 0.205)\), and high \((b = -16.438, 95\% \text{ CI} = -41.574, -1.297, p = 0.206)\) levels of social support
were not statistically significant after adjusting for covariates. The conditional direct effect of physical activity on CRP at low ($b = -6.792$, 95% CI = -16.127, -1.275, $p = 0.143$), moderate ($b = -6.800$, 95% CI = -16.151, -1.284, $p = 0.143$), and high ($b = -6.808$, 95% CI = -16.168, -1.282, $p = 0.143$) levels of social support were not statistically significant after adjusting for covariates. See Table 5.

**IL-6.** Unstandardized parameter estimates revealed that the direct effect of fatalism ($b = 2.387$, 95% CI = -1.215, 6.570, $p = 0.317$) and physical activity ($b = -0.929$, 95% CI = -1.584, 0.178, $p = 0.114$) on IL-6 was not statistically significant after adjusting for covariates. However, the direct effect of social support on IL-6 was statistically significant after adjusting for covariates ($b = -15.705$, 95% CI = -25.806, -1.245, $p = 0.040$). Greater social support was associated with lower IL-6 such that a one-unit increase in the social support scale was associated with a 15.705 decrease in IL-6 after adjusting for covariates. The direct effect of fatalism on physical activity also was statistically significant after adjusting for covariates ($b = 2.414$, 95% CI = 0.559, 4.030, $p = 0.023$). Greater fatalism was associated greater physical activity such that a one-unit increase on the fatalism scale was associated with a 2.414 increase in minutes spent engaging in physical activity after adjusting for covariates. See Figure 17.
Figure 17. Moderated mediation analysis of the association between fatalism, physical activity, social support, and IL-6 after adjusting for covariates. * denotes a statistically significant path ($p < 0.05$).

The conditional indirect effect of fatalism on IL-6 through physical activity at low ($b = -2.246, 95\% \text{ CI} = -4.906, 0.233, p = 0.180$), moderate ($b = -2.244, 95\% \text{ CI} = -4.903, 0.240, p = 0.181$), and high ($b = -2.242, 95\% \text{ CI} = -4.901, 0.244, p = 0.182$) levels of social support were not statistically significant after adjusting for covariates. The conditional direct effect of physical activity on IL-6 at low ($b = -0.930, 95\% \text{ CI} = -1.585, 0.176, p = 0.113$), moderate ($b = -0.929, 95\% \text{ CI} = -1.584, 0.178, p = 0.114$), and high ($b = -0.929, 95\% \text{ CI} = -1.584, 0.181, p = 0.115$) levels of social support were not statistically significant after adjusting for covariates. See Table 5.
Table 5. Conditional Effects at Different Levels of Social Support with Fatalism as Predictor, Health Behavior as Mediator and Clinical Outcome as outcome

<table>
<thead>
<tr>
<th>Diet</th>
<th>Fatalism → Clinical Outcome through Health Behavior&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Health Behavior → Clinical Outcome&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>b</strong></td>
<td><strong>S.E.</strong></td>
</tr>
<tr>
<td>A1C&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.040</td>
<td>0.578</td>
</tr>
<tr>
<td>Moderate</td>
<td>0.026</td>
<td>0.471</td>
</tr>
<tr>
<td>High</td>
<td>0.012</td>
<td>0.408</td>
</tr>
<tr>
<td>CRP&lt;sup&gt;d&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.331</td>
<td>1.610</td>
</tr>
<tr>
<td>Moderate</td>
<td>0.199</td>
<td>1.324</td>
</tr>
<tr>
<td>High</td>
<td>0.067</td>
<td>1.297</td>
</tr>
<tr>
<td>IL-6&lt;sup&gt;e&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>0.157</td>
<td>0.655</td>
</tr>
<tr>
<td>Moderate</td>
<td>0.140</td>
<td>0.589</td>
</tr>
<tr>
<td>High</td>
<td>0.123</td>
<td>0.537</td>
</tr>
</tbody>
</table>

| Physical Activity | | | | |
|------------------|-------------------------------------------------|----------------------------------|
|                 | **b** | **S.E.** | **95% CI** | **b** | **S.E.** | **95% CI** |
| A1C<sup>c</sup> | | | | | |
| Low  | 0.099 | 2.952  | -2.896, 6.327  | 0.042 | 1.308  | -0.994, 3.141 |
| Moderate | 0.096 | 2.961  | 2.904, 6.345   | 0.040 | 1.311  | -0.997, 3.148 |
| High  | 0.092 | 2.970  | -2.910, 6.365  | 0.039 | 1.314  | -0.998, 3.154 |
| CRP<sup>d</sup> | | | | | |
| Moderate | -16.418 | 12.964 | -41.506, -1.303 | -6.800 | 4.644 | -16.151, -1.284 |
| IL-6<sup>e</sup> | | | | | |
| Low  | -2.246 | 1.675  | -4.906, 0.233  | -0.930 | 0.587  | -1.585, 0.176 |
| Moderate | -2.244 | 1.677  | -4.903, 0.240  | -0.929 | 0.588  | -1.584, 0.178 |
| High  | -2.242 | 1.679  | -4.901, 0.244  | -0.929 | 0.589  | -1.584, 0.181 |

<sup>b</sup> = unstandardized parameter estimate; S.E. = standard error; CI = confidence interval

Clinical outcomes included A-hemoglobin A1C, CRP (C-reactive protein), IL-6 (Interleukin-6)

Health behaviors included diet and physical activity

Low, Moderate, and High indicate levels of social support

Bolded values indicate statistically significant associations (p < 0.05)

<sup>a</sup> Conditional indirect effect of fatalism on clinical outcome through health behavior at different levels of social support

<sup>b</sup> Conditional direct effect health behavior on clinical outcome at different levels of social support

<sup>c</sup> Covariates included age, employment status, and BMI

<sup>d</sup> Covariates included age, marital status, and BMI

<sup>e</sup> Covariates included age and BMI
CHAPTER 5: DISCUSSION

The purpose of the study was examine the associations between number of comorbidities, psychosocial well-being (i.e., depressive symptoms, discrimination stress), fatalism, health behaviors (i.e., diet, physical activity), and T2D-related outcomes (i.e., A1C, CRP, IL-6), and the buffering effect of social support on these relationships in a sample of African American adults with T2D. Examining mechanisms underlying these associations can provide insight when developing effective strategies for improving T2D self-management and related outcomes.

Description of Number of Comorbidities, Psychosocial Well-Being, Health Behaviors, and Clinical Outcomes

Number of Comorbidities. The prevalence of comorbidities in the current sample is high, with 92% reported having at least 2 comorbidities (2 comorbidities = 39%; 3 comorbidities = 32%; ≥ 4 comorbidities = 21%). This aligns with previous studies indicating high prevalence of comorbidities in individuals with T2D. For example, one study of individuals with T2D (N = 1,389,016) reported that the majority of participants had at least one comorbid chronic condition and about 86% reported having two or more (Iglay et al., 2016). Among African American adults with T2D, one study (N = 11,492) found that at least 33% reported having at least 3 comorbidities (i.e., hypertension, hyperlipidemia, obesity) (Campbell et al., 2020).

When compared to national data on US adults with T2D, the current sample had similar prevalence of obesity (86% vs. 89%) but higher prevalence of high blood pressure (90% vs. 68%) and comorbidities (CDC, 2020c). However, only 3% of the current sample reported having kidney disease as a comorbidity, which is lower than the general US adult population of adults with T2D (37%) (CDC, 2020c). Kidney function declines naturally with aging and kidney disease develops more frequently in older people (aged ≥ 65 years) (O'Hare et al., 2007), particularly among those with unmanaged T2D (Sheen & Sheu, 2014; Wasen et al., 2004). In
addition, the development and progression of kidney disease in adults with T2D increase as T2D duration lengthens (Kim, Park, Cho, & Kim, 2018). While the average age in the current sample was 58.75 years (SD = 9.15), T2D duration in the current sample was not assessed, so it is unknown whether the low prevalence of kidney disease was due to age or short T2D duration. Despite the low prevalence of kidney disease in the current sample, the high prevalence of high blood pressure and obesity as comorbidities was alarming given its association with higher risk for greater complications (e.g., cardiovascular disease) and worse health outcomes (e.g., mortality) (Barr et al., 2007; Petrie, Guzik, & Touyz, 2018).

Psychosocial Factors

Depression. The current sample reported low depressive symptoms, which is inconsistent with previous research indicating a slightly higher prevalence of depression in individuals with T2D. In a meta-analysis of 42 studies (N = 21,351 adults with T2D), researchers found the prevalence of major depression in people with T2D was 11% and the prevalence of clinically relevant depression was 31% (Anderson et al., 2001). One study also reported the prevalence of any depressive symptoms in a sample of 183 African Americans with T2D was about 30% (Gary, Crum, Cooper-Patrick, Ford, & Brancati, 2000). Low depressive symptoms in the current sample is also interesting given 92% had at least 2 comorbidities, which is contradictory to previous research indicating T2D patients with comorbidities tend to have higher levels of depressive symptoms (Fung et al., 2018; Rivera-Hernandez, 2014). In addition, low depressive symptoms in the current sample is surprising given the majority of participants were women (75%), which is inconsistent with previous findings indicating high rates of depression in women with T2D (Anderson et al., 2001).
In this study, more than half of the participants (55%) indicated their perception of general health as “good,” which is an indicator of good quality of life (CDC, 2018a). Previous studies have suggested that better quality of life is associated with lower depressive symptoms in a sample of adults with T2D (Derakhshanpour, Vakili, Farsinia, & Mirkarimi, 2015; Juárez-Rojop et al., 2018; Wexler et al., 2006), which supports the pattern observed in this current sample. However, evidence also indicate that African Americans tend to underreport depressive symptoms especially when compared to their White counterparts (Wagner, Perkins, Piette, Lipton, & Aikens, 2009). This could be due to stress and adversity they frequently experience (Barnes & Bates, 2017), making this population desensitized to effects such as depression.

**Discrimination Stress.** Discrimination stress was low in the current sample. This aligns with findings from another study that reported similar mean scores on the *Experiences of Discrimination* (EOD) scale (Krieger et al., 2005) among African Americans with T2D (Williams et al., 2020). Research suggests religion is an important social and psychological resource that can help buffer the effect of discrimination on mental health especially among African Americans, which is partly due to the social support benefits associated with one’s involvement to religious communities (Bierman, 2006). Given this study is comprised of a sample with strong religious orientation, it is possible the low discrimination stress is influenced by the buffering effects of religiosity on discrimination. It is also possible the low prevalence of discrimination stress in this sample is due to the same phenomenon that explains low prevalence of depression. That is, frequent experiences of stress and adversity related to discrimination may have made African Americans in the current sample less sensitive to the impact of discrimination, leading them to underreport their actual experiences or characterize their subjective well-being or emotions related to their experiences.
Fatalism. Fatalism in the current sample was moderate, which is similar to other studies that have used the *Religious Health Fatalism Scale* (Franklin et al., 2008) to assess fatalism in a sample of African American faith communities (Franklin et al., 2007; Franklin et al., 2008). A recent study of African American adults with T2D also reported moderate levels of fatalism (as measured by the *Diabetes Fatalism Scale (DFS)*) (Abbott, Slate, Graven, Lemacks, & Grant, 2021). However, several studies also indicated high fatalism (as measured by the DFS) in this population (Asuzu et al., 2017; Egede & Osborn, 2010; Osborn & Egede, 2010), which is more consistent with previous studies on fatalism in other disease context such as cancer (as commonly measured by the *Powe Fatalism Inventory*) indicating high fatalism among African American adults (Powe & Finnie, 2003; Powe & Johnson, 1995; Powe & Weinrich, 1999). Moderate fatalism may be due to the fatalism scale (*Religious Health Fatalism Questionnaire*) used in the current study, which captures fatalistic beliefs related to broader aspects of health. It is possible that assessments of fatalism specific to chronic disease-related outcomes make people feel they have less control over their health, which may explain the higher fatalism observed in other studies that used disease-specific fatalism scales.

Given the philosophical perspective that fatalistic perceptions develop over time as a result of social, political, geographical, and/or historical factors and may influence one’s overall life experience (Powe & Johnson, 1995), fatalism was expected to be higher in the current

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1 The *Diabetes Fatalism Scale (DFS)* is a 12-item questionnaire that measures fatalism components emotional distress, religious and spiritual coping, and self-efficacy specific to T2D self-management and outcomes (Egede & Ellis, 2009).

2 The *Powe Fatalism Inventory* is a 15-item questionnaire that measures belief in divine control and predetermination, pessimism, resignation, and the perceived inevitability of death associated with a cancer diagnosis (Powe, 1995).

3 The *Religious Health Fatalism Questionnaire* is a 17-item questionnaire that measures faith-related beliefs across three dimensions: divine provision, destiny, and helpless inevitability (Franklin et al., 2008).
sample given the average age was 58.75 years ($SD = 9.15$). In addition, previous studies indicate African Americans tend to endorse higher fatalistic beliefs about health (Morgan et al., 2010; Morgan et al., 2008; Powe, Ross, Wilkerson, Brooks, & Cooper, 2007; Powe & Johnson, 1995), especially when compared to Whites (Franklin et al., 2007; Powe & Johnson, 1995), so moderate fatalism in the current sample was somewhat unexpected.

Given the intersection between religiosity and fatalism (Franklin et al., 2007; Franklin et al., 2008), and the strong religious orientation of the current sample, fatalism was expected to be higher given fatalism was measured using a religious-specific fatalism scale. However, there is also research indicating fatalism and religiosity influence health differently when viewed together rather than separately, which may be due to differences in control beliefs (Friori et al., 2006; Neff & Hoppe, 1993). Therefore, future studies should examine fatalism (e.g., General Health Fatalism Scale (Shen, Condit, & Wright, 2009)) and religiosity (e.g., Centrality of Religiosity Scale (Huber & Huber, 2012)) as separate constructs, rather than as combined constructs, to assess if fatalism would be higher despite one’s strong religious orientation.

**Social support.** Social support was moderate in the current sample, unlike findings from a recent study of African American adults with T2D indicating high level of social support (Abbott et al., 2021). Findings on social support in the current sample was expected to be higher given participants were recruited from churches, a place where many African Americans find sources of support including advice, encouragement, companionship, financial assistance, prayers, and help during illness (Krause, 2002). Church-based social support and other informal types of social support also are particularly relevant to African Americans, a population often marginalized due to economic or cultural factors from the types of formal social support provided by healthcare professionals (Ford, Tilley, & McDonald, 1998b). However, social
support is a multifaceted experience that involves numerous factors and circumstances including satisfaction with social support received, the size of social support network, perception of the availability of social support, and socioeconomic status (Ford et al., 1998b; Holt-Lunstad & Uchino, 2015).

Health Behaviors

Diet. Participants in the current study had low E-DII scores indicating a more anti-inflammatory diet. Anti-inflammatory diets typically consist of high consumption of vegetables (e.g., dark green leafy vegetables such as kale, spinach, collard greens), legumes, nuts and seeds, more fatty fish and some lean animal protein, foods high in fiber as well as consumption of whole grains in small amounts and reduced consumption of refined carbohydrates, whereas a pro-inflammatory diet consists of high consumption of meat (i.e., red meat, processed meat, and organ meat), refined carbohydrates (e.g., white bread, white rice), sugar sweetened beverages (e.g., cola, fruit juice), and alcohol (Ricker & Haas, 2017). In the current study, findings on diet are inconsistent with the literature demonstrating a more pro-inflammatory diet among individuals with T2D. For example, while the inflammatory potential of diet was not examined, a systematic review of 11 cross sectional studies found that individuals with T2D had a dietary pattern that was more consistent with a pro-inflammatory diet (e.g., low consumption of vegetables, high consumption of meats) (Burch, Ball, Somerville, & Williams, 2018). There have not been any previous studies examining the inflammatory potential of diet among African Americans with T2D. However, a study of Hispanic adults with T2D found more T2D patients with a more pro-inflammatory diet (higher DII scores; 22.8%) than anti-inflammatory diets (lower DII scores; 6.4%) (Denova-Gutiérrez et al., 2018). It is interesting that participants in the current study had a more anti-inflammatory diet given the elevated average levels of CRP and
IL-6. However, the dietary intake was assessed using the *Food Frequency Questionnaire* (FFQ), and while this is a comprehensive measure of dietary intake (Mayer-Davis et al., 1999), the self-report nature of the FFQ increases the risk of bias (e.g., recall bias, non-response bias), which may be contributing to the current findings. In addition, the larger study (Hébert et al., 2013) was a lifestyle intervention, so participants interested in health often volunteer for these studies (Fisher et al., 2018), which may have skewed the sample to a healthier population.

**Physical Activity.** The time spent in moderate-intensity physical activity level was low ($M = 39.79$ minutes/week, $SD = 40.45$, Range = $0.00 – 2.09$) when compared to the recommended moderate-intensity physical activity level of 150 minutes/week for adults (U.S. Department of Health and Human Services, 2018). In the current study, only 2.84% ($n = 3$) met this recommendation, although it is notable that 35.85% had missing datapoints for physical activity. Nonetheless, this is consistent with findings from previous studies indicating high prevalence of inadequate physical activity among African Americans (Nwasuruba et al., 2007) as well the general US population of adults with T2D (CDC, 2020c).

Approximately 28% of US adults age ≥ 50 years are physically inactive (CDC, 2019). It is also well-established that physical activity level declines with aging due to reduction of muscle strength in both upper and lower limbs and changes in body-fat percentage, flexibility, agility, and endurance (Cvecka et al., 2015; Milanović et al., 2013), although it has been suggested that regular exercise and an active lifestyle can delay decline in physical functioning among older adults (Westerterp & Meijer, 2001). Nonetheless, the low physical activity level observed in the current sample is unsurprising given the average age was 58.75 years ($SD = 9.15$). In addition, the presence of comorbidities for people with T2D can have a significant impact on their disease management, especially performing self-care behaviors such as engaging in physical activity
(Aga, Dunbar, & Kebede, 2019; Aga, Dunbar, Kebede, et al., 2019). Therefore, it is possible that the low physical activity level in the current sample was due to high prevalence of comorbidities, limiting them performing self-care behaviors and achieving the recommended physical activity level.

**Clinical Outcomes**

Although the average A1C in the current sample was high ($M = 7.42\%$, $SD = 1.55$), there were slightly more participants who met the A1C clinical recommendation ($\leq 7.0\%$) than those who did not meet the recommendation (56% vs. 44%). This aligns with findings from a large study of US adults with T2D using NHANES ($n = 3,335$) and BRFSS ($n = 97,310$) data between 1999-2010 that reported about 33% - 59% did not meet the recommended goals for A1C (Ali et al., 2013). More recently, the 2020 National Diabetes Statistics Report noted that only about 19% of US adults with T2D met the recommendation for A1C (CDC, 2020c). Moreover, the average CRP in the current sample was high ($M = 5.98 \text{ mg/L}$, $SD = 7.06$), and approximately 22% had CRP of < 1 mg/L, 29% had CRP of 1-3 mg/L, and 49% had CRP >3.0 mg/L, which corresponds low-, borderline-, and high-risk for future cardiovascular event, respectively (Pearson et al., 2003). Furthermore, the average IL-6 was 2.30 ρg/mL ($SD = 1.53$), and approximately 23% had IL-6 above 3.0 ρg/mL, which is a cut-off point that has been associated with high-risk of cardiovascular disease and mortality (Volpato et al., 2001).

Adherence to T2D self-management protocols has been associated with better clinical outcomes in individuals with T2D. For example, several studies found that diets consistent with an anti-inflammatory diet (e.g., high fiber diet, Mediterranean diet, Dietary Approaches to Stop Hypertension (DASH) diet) can reduce A1C (Huo et al., 2015; Silva et al., 2013) and inflammatory markers (Azadbakht et al., 2011; Maiorino et al., 2016) in individuals with T2D.
Studies also found that exercise (e.g., aerobic exercise, resistance training) and increased levels of physical activity can help lower A1C (Church et al., 2010) and inflammatory markers (Hayashino et al., 2014) in individuals with T2D. Given this, it is especially important for participants to follow the dietary recommendations and increase physical activity levels for improved outcomes, and to reduce the risk for developing complications and more comorbidities.

**Does social support moderate the associations between number of comorbidities, psychosocial well-being, fatalism, health behaviors, and clinical outcomes?**

In the current study, the *Stress-Buffering Model of Social Support* (Cohen & Willis, 1985) was used as a guiding framework to explore the buffering effect of social support on potential stressors (e.g., presence of comorbidities, poor psychosocial well-being, high fatalism), health behaviors and clinical outcomes among African American adults with T2D.

*Aim 1*

The first aim examined potential risk factors and mechanisms that increase (or reduce) risk for fatalistic beliefs about health. The hypothesis that the indirect relationship between number of comorbidities and fatalism through psychosocial well-being would be influenced at different levels of social support was not supported. The hypothesis that the direct relationship between psychosocial well-being and fatalism would be influenced by different levels of social support also was not supported. While the prevalence of number of comorbidities was high and level of social support was moderate, null associations could be due to low levels of depressive symptoms and discrimination stress in the overall sample.

There has been one study that examined predictors of fatalism in the context of T2D and found that fewer number of comorbidities predicted greater fatalism (Sukkarieh-Haraty et al., 2018a), which is inconsistent with previous findings in other populations indicating a positive association between number of comorbidities and fatalism (Franklin et al., 2007; Keeley et al.,
2009). Studies in other populations also failed to report a clear connection between psychosocial well-being and fatalism. For example, a study of stroke patients found the association between fatalism and mortality was more pronounced for patients reporting no depressive symptoms (Morgenstern et al., 2011). The null findings in the current study highlight that fatalism is a complex construct that may not be easily predicted by number of comorbidities or psychosocial well-being (Leyva et al., 2014; Morgan et al., 2008). That is, a person may have fewer comorbidities, low depressive symptoms, low perception of discrimination stress, and high perception of social support, but these factors may be less important in determining whether (or not) they feel like they have control over their health and health outcomes.

Literature suggests factors related to social determinants of health such as poverty and low socioeconomic status represent important barriers to achieving optimal health (Williams & Jackson, 2005), and those potentially facing these barriers are likely to reinforce fatalistic beliefs about chronic diseases (Espinosa De Los Monteros & Gallo, 2011; Powe, 1995). In the context of T2D, one study of individuals with T2D found that lower educational level was associated with greater fatalistic beliefs about T2D self-management (Sukkarieh-Haraty et al., 2018a). Given this, more research is needed to examine the influence of social determinants of health (e.g., socioeconomic status) when understanding potential risk factors for fatalism. In addition, previous research indicates health beliefs vary by race/ethnic groups (Espinosa De Los Monteros & Gallo, 2013). Given this, more research is needed to identify the underlying components of T2D-specific fatalism that are most pertinent to African American health.

Aim 2

The second aim examined the underlying mechanism between psychosocial well-being, fatalism, and social support as predictors of health behaviors in individuals with T2D. The
hypothesis that the indirect association between psychosocial well-being and health behaviors through fatalism would be influenced by different levels of social support was not supported. The hypothesis that the direct relationship between fatalism and health behaviors would be influenced by different levels of social support also was not supported. There is a small literature linking psychosocial well-being, social support, and fatalism as predictors of health behaviors in individuals with T2D, although the buffering effect of social support has not been examined. In one study, researchers found that when depressive symptoms were included in the model, fatalism was no longer significantly associated with self-care behaviors (e.g., diet, physical activity) while significant associations were observed for social support and self-care behaviors (Egede & Osborn, 2010). Another study also found that less fatalistic attitudes and more social support were independent, direct predictors of better self-care behaviors (Egede & Osborn, 2010). Given these findings, it is possible that one’s perception of control over performing a health behavior or the actual performance of a health behavior are not impacted by psychosocial well-being or social support in the current sample. Rather, factors such as having time to engage in physical activity (Sallis & Hovell, 1990), access to a gym or fitness facility (Sallis & Hovell, 1990), neighborhood walkability (Sallis & Hovell, 1990), or access to healthy food (Gundersen, 2013) may simply be more relevant.

In addition, the Health Belief Model states that factors such as health knowledge influence individual beliefs about health, whether there are benefits or barriers to performing health behavior, and whether they feel they can perform a health behavior (self-efficacy), which in turn leads to whether (or not) they engage in a behavior (Champion, Rimer & K. Viswanath (Eds.), & 65). 2008). Previous studies have demonstrated the association between health knowledge and fatalism, although the current literature in this area is small. In a previous study
of young adults without T2D, higher diabetes knowledge was associated with healthier diet among individuals with low fatalism (San Diego & Merz, 2020). In a study of individuals with T2D, greater diabetes fatalism was associated with lower diabetes knowledge after adjusting for age, race/ethnicity, sex, education, income, and employment (Walker et al., 2012). Given this, more research is needed to examine the impact of health knowledge on one’s individual belief about health (fatalism) and its influence on performing health behaviors.

**Aim 3**

The third aim examined the associations between fatalism, social support, and health behavior as predictors of clinical outcomes. The hypothesis that the indirect association of fatalism on clinical outcomes through health behaviors would be influenced by different levels of social support was not supported. However, greater fatalism was directly associated with greater physical activity, which is inconsistent with previous studies demonstrating an inverse association between fatalism and physical activity in individuals with T2D (Asuzu et al., 2017; Osborn, Bains, & Egede, 2010; Osborn & Egede, 2010; Walker et al., 2012). The current findings on fatalism and physical activity may have been impacted by the high percentage (36%) of missing datapoints for physical activity in the current sample. However, Maximum Likelihood Ratio was used to account for missingness, which is a robust method that uses observed data to search for parameters that yield the highest log likelihood (Kline, 2016). Nonetheless, these findings should be interpreted with caution given the complexity of fatalism when predicting health behaviors. In addition, it is possible that some items in the *Religious Health Fatalism Questionnaire* (e.g., “Sometimes someone can be ill because of disobedience to God”) were interpreted as they should take care of themselves (e.g., exercise, engage in physical activity)
because it is “God’s temple”, which has been indicated in the scripture (e.g., “If anyone destroys God’s temple, God will destroy him. For God’s temple is holy, and you are that temple”).

The hypothesis that the direct association of health behaviors on clinical outcomes would be influenced by different levels of social support was partially confirmed. In this study, a more pro-inflammatory diet was associated with elevated IL-6, which is in line with previous findings on the inflammatory potential of diet in a sample of adults at high risk for T2D (Laouali et al., 2019; Maiorino et al., 2016). In addition, a more pro-inflammatory diet was also associated with elevated IL-6 only for individuals with low and moderate levels of social support, but not for individuals with a high level of social support. This is inconsistent with findings from a previous study that demonstrated the interaction of T2D-self management behaviors (i.e., glucose management, dietary control, physical activity, healthcare use) and social support predicted lower A1C, although the buffering effect of social support at different levels was not examined (Thojampa & Mawn, 2017). However, it is important to note that while the moderating effect was not statistically significant for individuals with high level of social support, some effects may be at borderline but not significantly different from each other. That is, although low and moderate levels of social support were statistically significant and high social support was not, betas may overlap considerably. Thus, although high social support was not statistically significant ($p = 0.098$), the beta was very similar to low and medium levels of social support, which were statistically significant ($p = 0.034$ and $p = 0.049$, respectively).

The current findings suggest that if individuals with T2D have a more pro-inflammatory diet, they are also likely to have elevated IL-6 regardless of the existence of social support, which is unsurprising given the direct physiological impact of diet on inflammation (Galland,
Despite this, improving dietary behaviors among individuals with T2D should be considered within the context of social support, as this appears to be a modifying factor for IL-6.

More research on the buffering effects of social support is needed as the impact of a pro-inflammatory diet on IL-6 appears to be stronger when social support is inadequate. While positive effects (i.e., social support) of religiosity through religious practice and participation in religious organizations have been demonstrated (Davis et al., 1994; Ferraro & Koch, 1994; Wilson, 2000), there is also evidence that certain practices may inhibit health enhancing behaviors leading to worse health outcomes. For example, practices of various religious groups may conflict with the recommendations of medical professionals and affect an individual’s well-being such as create conflicts and misunderstanding in patient education and lack of adherence to treatment (Gall et al., 2005; Powe, 1995). Given this, more studies are needed to examine the underlying mechanism of religiosity as a source of support and its positive and negative influences on health behaviors and clinical outcomes.

Social support has been characterized as the perception that certain individuals or a specific group will be available to provide support if needed or the actual provision of support by another (Holt-Lunstad & Uchino, 2015). Different types of social support also have been established, which includes emotional (e.g., positive feedback, encouragement), instrumental or tangible (e.g., money or transportation), companionship (e.g., social activities), and informational (e.g., advice or guidance) support (Holt-Lunstad & Uchino, 2015). In the current study, social support was assessed using the Social Support Questionnaire (Seeman et al., 2014), which measures quality of relationships and reflects the extent of social support as well as the extent of social strains. It is possible that scale used in the study does not capture aspects of social support that are more relevant when understanding its main or buffering effects on perceptions of control.
(i.e., fatalism) and health outcomes. It has been suggested that instrumental and emotional support, as well as support from family are especially important to adherence and illness management while instrumental support can help facilitate health-behavior change in a practical way by reducing stressful situation through receipt of medical care (Gallant, 2013). Therefore, scales that capture different types of social support such as emotional support, instrumental/tangible support or informational support (e.g., Interpersonal Support Evaluation List (Cohen, Mermelstein, Kamarck, & Hoberman, 1985)) may provide more insight when understanding main or buffering effects of social support on one’s perception of control over their health and health outcomes.

The role of social support on dietary behaviors is not consistently supported, with some evidence indicating social support does not always influence positively influence health behaviors (Gallant, 2003). Within the social support theory, it has been suggested that social interactions may have unintentional negative influences on self-management behaviors (Krause, 1990; Wortman & Conway, 1985). That is, some family members or friends may have misconceptions or a lack of understanding about disease management and behave in unsupportive or inappropriate ways, offer well-intentioned advice that conflicts with self-management recommendations, or directly/indirectly promote unhealthy behaviors such as preparing high-fat foods (Gallant, 2003). In the current study, it is possible that participants with high levels of social support have a social network that reinforces low adherence to dietary recommendations.

Conflicting findings on social support may also reflect the possibility that the social support-health behavior association is not the same across different types of support, nor across different types of health behaviors, and that the influence of social support depends on many
contextual and situational factors (Gallant, 2013). Therefore, social support measures specific to diet (Social Support and Eating Habits Survey (Sallis, Grossman, Pinski, Patterson, & Nader, 1987)) and physical activity (Social Support and Exercise Survey (Sallis et al., 1987)), rather than a global measure of social support, may provide more understanding on the moderating effect social support on the association between health behavior and T2D-related clinical outcomes.

A systematic review of the impact of social support on clinical outcomes in adults with T2D reported that the interpretation or receipt of social support can differ based on numerous factors, including gender, race/ethnicity, culture, or social environment (Strom & Egede, 2012). Furthermore, Strom and Egede (2012) found that there were differences in the method of delivery and the source of social support among race/ethnic minority groups as compared to their White counterparts (Strom & Egede, 2012). For example, African Americans demonstrated more variability in the modes of delivery of social support. Race/ethnic minorities also exhibited a greater propensity for support from family and friends as compared with Whites, who tended to rely less on support from family and the community and more on support from the media and healthcare professionals. Given this, it is possible that there are underlying facets in social support (i.e., modes of delivery; formal vs. informal support) that are more relevant and important to consider when examining the association of social support and clinical outcomes in African Americans with T2D.

**Limitations**

There are several limitations in the current study. The first limitation concerns some of the measurements used. Although the fatalism measure was a validated scale (Franklin et al., 2008), it has been suggested that a T2D-specific fatalism scale [e.g., 12-item Diabetes Fatalism...
Scale (Egede & Ellis, 2009)] may be more appropriate when understanding different aspects of T2D self-management that are more prone to fatalism (San Diego et al., under review). However, the use of the Religious Health Fatalism Scale (Franklin et al., 2008) is appropriate in the current study given this is a sample with a strong religious orientation. In addition, number of comorbidities and diet (Food Frequency Questionnaire (Mayer-Davis et al., 1999)) were assessed using self-report measures, which increased the possibility of recall bias due to respondents’ inaccurate knowledge of their comorbidities and dietary behaviors. It is also conceivable that there was non-response bias or social desirability when assessing one’s psychosocial well-being and dietary behaviors, which may be due to the possibility of respondents’ unwillingness to report accurate health information.

Second, the generalizability of the findings was limited in terms of its sample characteristics. Data were collected from Southeastern, US, an area with high concentration of African Americans, so psychosocial well-being, health behaviors and clinical outcomes of the current sample may differ or may not be representative of African Americans in other areas of the US. Most of the sample was comprised of women, which could have introduced bias to the psychosocial well-being and health behavior outcomes. For example, differences in social support, quality of life, and adherence to T2D self-management between men and women with T2D have been demonstrated (Chiu & Wray, 2011; Misra & Lager, 2009). Therefore, potential gender differences in psychosocial well-being and health behaviors could have impacted the overall findings. Furthermore, the larger study was a lifestyle intervention study, which could have led to a sample that was healthier given these are often the individuals who volunteer for lifestyle intervention studies (Fisher et al., 2018).
Third, the small sample size as well as high rates of missing datapoints, especially for physical activity and social support, reduced the statistical power in the current analysis. Due to high attrition rate (66 %) (Wirth et al., in preparation), the sample size also does not allow for adequate statistical power to evaluate the theoretical models at different time-points and only allowed for a cross-sectional analysis of the baseline data. Various rules-of-thumb related to sample size in structural equation modeling have been formulated, including (a) a minimum sample size of 100 or 200 (Boosma, 1982, 1985), (b) 5 or 10 observations per estimated parameter (Bentler & Chou, 1987), and (c) 10 cases per variables (Nunnally, 1967). However, these rules are not model-specific, which may lead to over- or underestimated sample size requirements (Wolf, Harrington, Clark, & Miller, 2013). Nonetheless, future studies with higher sample size ($N \geq 200$) are needed given the probability of making a Type II error decreases as power increases (VanVoorhis & Morgan, 2007).

**Strengths**

Despite the limitations, several strengths have been identified. While research on fatalism and African Americans in the cancer context is relatively large (Morgan et al., 2008; Powe & Finnie, 2003), this study contributes to the growing literature on fatalism in the context of T2D in this population. In addition, while direct and indirect effects of social support in the association between fatalism and T2D-related outcomes have been examined, this is the first study to the author’s knowledge to explore the buffering effect of social support in this relationship. In addition, this is also the first study to examine the association between discrimination stress and fatalism in the context of T2D. Moreover, the current literature on predictors of fatalism in the context of T2D self-management is small (Sukkarieh-Haraty et al., 2018a) and findings have been inconsistent (Franklin et al., 2007; Keeley et al., 2009). While no significant findings were
observed, the current study provides insight on how to better examine factors that increase (or reduce) risk for fatalism.

In this study, strengths related to several measures also were identified. This is the first to examine the associations between psychosocial well-being, fatalism, social support, and the inflammatory potential of diet (as measured by the DII) in a sample of African Americans with T2D. This study also used laboratory derived data for assessing T2D clinical outcomes (A1C, CRP, IL-6) as well as accelerometer for physical activity, which decreased the potential for recall bias.

**Implications**

Despite medical advances and prevention efforts, T2D remains a public health crisis especially for race/ethnic minorities including African Americans. Understanding factors that influence adherence as well as factors that inhibit (or enable) one from achieving recommended clinical goals is important for improving T2D-related outcomes.

Findings in the current study emphasize that fatalism is a complex construct that may be less predictable by number of comorbidities or psychosocial well-being. More research is needed to examine the influence of social determinants of health (e.g., poverty, socioeconomic status, educational level) and the role of religiosity when understanding potential risk factors for fatalism. In addition, exploring the underlying components of T2D-specific fatalism (e.g., cultural histories, structural barriers) that are most pertinent to African Americans with T2D is important to better understand the impact of fatalism in this population.

Literature on modifiability of fatalism is small. Previous research in non-clinical samples has suggested that brief interventions can diminish cancer-specific fatalism (Morgan, Fogel, Tyler, & Jones, 2010; Powe & Weinrich, 1999). In the context of T2D, a recent RCT of African
American adults with T2D found a significant decrease in fatalism scores (as measured by the *Diabetes Fatalism Scale* (Egede & Ellis, 2009)) after the 3-week session for participants in both the control (i.e., received diabetes self-care brochure) and intervention group (i.e., presented with information about diabetes types, risk factors, and symptoms of hyper- and hypoglycemia; discussion on managing A1C through diet and exercise; discussion on strategies to manage diabetes and reduce risk for complications). While evidence indicates increasing one’s awareness of T2D self-management can help increase perception of control over their health outcomes, more research is needed to examine other potential protective factors against fatalism to provide insight on other strategies for diminishing T2D-specific fatalism and improve T2D-related outcomes.

It has been suggested that fatalism may, rather than inhibit health behaviors, be a reaction to a health condition or chronic illness (Franklin et al., 2007). In this study, the second theoretical model examined whether fatalism is in the path of the association between psychosocial well-being and health behaviors while the third model examined fatalism as a predictor of clinical outcomes through health behaviors. Given the largely null findings in these causal pathways, future work in this area should examine whether health behaviors are associated with clinical outcomes through fatalism, or if health behaviors are associated with fatalism through clinical outcomes. That is, future studies should examine whether fatalism is an outcome (rather than a predictor) of poor T2D self-management and worse clinical outcomes to enhance our understanding of the modifiability of fatalism.

In addition, findings in this study indicate that one’s perception of control over performing a health behavior or the actual performance of a health behavior are not impacted by psychosocial factors. Therefore, exploring the role of potential barriers such as time to engage in
physical activity, access to a fitness facility, neighborhood walkability, access to healthy foods, and knowledge related to T2D self-management may be important when understanding the influence of fatalistic beliefs on performing health behaviors.

Lastly, findings suggest improving dietary behaviors should be considered within the context of social support, as this appears to be a modifying factor for IL-6. However, a better understanding of the buffering effect of social support on the associations between number of comorbidities, psychosocial well-being, fatalism, and T2D-related outcomes is needed. Future studies should examine the impact of different sources (formal vs. informal) and types of social support (i.e., emotional support, instrumental support, informational support; positive vs. negative; religiosity/spirituality as a source of support) in these associations.
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APPENDIX A
UNIVERSITY OF MEMPHIS INSTITUTIONAL REVIEW BOARD APPROVAL

Subject: PRO-FY2021-86 - Admin Withdrawal: Not Human Subject Research
Date: Tuesday, September 15, 2020 at 8:44:27 AM Pacific Daylight Time
From: irb@memphis.edu
To: Kenneth Daniel Ward (kdward), Emily Rose Nepacina San Diego (nsndiego)
Attachments: ATT00001.png

Institutional Review Board
Division of Research and Innovation
Office of Research Compliance
University of Memphis
315 Admin Bldg
Memphis, TN 38152-3370

September 15, 2020

PI Name: Emily San Diego
Co-Investigators:
Advisor and/or Co-PI: Kenneth Ward
Submission Type: Admin Withdrawal
Title: Number of co-morbidities, psychosocial factors, fatalism and Type 2 Diabetes-related outcomes: a moderated mediation analysis
IRB ID: PRO-FY2021-86

From the information provided on your determination review request for “Number of co-morbidities, psychosocial factors, fatalism and Type 2 Diabetes-related outcomes: a moderated mediation analysis”, the IRB has determined that your activity does not meet the Office of Human Subjects Research Protections definition of human subjects research and 45 CFR part 46 does not apply.

This study does not require IRB approval nor review. Your determination will be administratively withdrawn from Cayuse IRB and you will receive an email similar to this correspondence from irb@memphis.edu. This submission will be archived in Cayuse IRB.

Thanks,

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