PREDICTORS OF METABOLIC CONTROL IN YOUTH WITH TYPE 1 DIABETES: EXAMINING RACIAL DISPARITIES IN THE RELATIONSHIP BETWEEN DEPRESSIVE SYMPTOMS AND ADHERENCE

AN ABSTRACT
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TO THE DEPARTMENT OF PSYCHOLOGY
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BY

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Poor metabolic control is a major health concern for children and adolescents with type 1 diabetes, particularly for African American youth. The aims of this study were to test the mediating relationship between two variables consistently related to metabolic control, depressive symptoms and adherence, as well as to attempt to explain racial disparities in metabolic control. The study sample consisted of 53 European American youth and 33 African American youth ages 5 to 20 ($M = 13.59, SD = 3.49$) with type 1 diabetes. Information on depressive symptoms, adherence, and HbA1c was collected during routine outpatient clinic visits. Significant associations were found between depressive symptoms and metabolic control, depressive symptoms and adherence, and adherence and metabolic control. When included together in a regression model, adherence mediated the relationship between depressive symptoms and metabolic control. This mediation pathway did not significantly differ between African American youth and European American youth; however, African American youth had significantly higher HbA1c levels. These findings indicate the importance of considering depressive symptoms during treatment for type 1 diabetes. This study also supports previous research findings of racial disparities in metabolic control among youth with type 1 diabetes. Future studies should further examine mechanisms by which these racial disparities emerge.
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Predictors of Metabolic Control in Youth with Type 1 Diabetes: Examining Racial Disparities in the Relationship between Depressive Symptoms and Adherence

Type 1 diabetes is a serious and debilitating chronic illness that requires adherence to a strict treatment regimen in order to obtain optimal metabolic control and avoid severe and long-term health complications (Gillibrand & Stevenson, 2006). Poor metabolic control is a risk factor for negative health outcomes in individuals with diabetes (Hood, Rausch, & Dolan, 2011). The main clinical measurement of metabolic control is the hemoglobin A1c (HbA1c) test which is used as an average measure of blood glucose over the past two to three months. High HbA1c levels have been linked with increased rates of hospitalization, increased risk for cardiovascular disease, increased mortality and a decrease in cognitive function (Cukierman-Yaffe et al., 2009; Levine et al., 2001). Therefore, identifying factors related to poor metabolic control has major implications in the study of diabetes outcomes.

One potential risk factor for poor metabolic control is elevated depressive symptoms (Bernstein, Stockwell, Gallagher, Rosenthal, & Soren, 2013; Grey, Davidson, Boland, & Tamborlane, 2001; Hislop, Fegan, Schlaeppi, Duck, & Yeap, 2007; Hood et al., 2011; Johnson, Eiser, Young, Brierley, & Heller, 2013; Santos, Bernardo, Gabbay, Dib, & Sigulem, 2013). Specifically, researchers suggest that youth with depressive symptoms have a harder time maintaining adherence to their treatment regimen, resulting in poorer metabolic control (Hood et al., 2006; Korbel, Wiebe, Berg, & Palmer, 2007). However, only one study to date has empirically examined adherence as a mediator of the
relationship between depressive symptoms and metabolic control in youth with type 1 diabetes (McGrady, Laffel, Drotar, Repaske, & Hood, 2009). Therefore, one goal of the current study was to examine the mediating role of adherence on the depressive symptoms-metabolic control link.

A second goal of the current study was to examine whether this mediational pathway is stronger for African American youth than for European American youth. African American youth experiencing depression report less utilization of mental health care services (Cummings & Druss, 2011; Sen, 2004). This may be due to various factors, including disparities in access to care, insurance, income, and cultural differences (Primm et al., 2010; Sussman, Robins, & Earls, 1987; Zito, Safer, Zuckerman, Gardner, & Soeken, 2005). However, if African American youth are more likely to suffer from depressive symptoms without receiving the necessary treatment, depressive symptoms may have a more negative impact globally for these youth, including greater and more persistent obstacles to adherence. Although a number of studies have reported higher HbA1c among African American youth (Delamater et al., 1999; Hassan, Loar, Anderson, & Heptulla, 2006; Hilliard, Wu, Rausch, Dolan, & Hood, 2013; Kamps, Hempe, & Chalew, 2010; Naar-King et al., 2006), few have examined the potential reasons such health disparities may exist. The current study proposes that the depressive symptoms-adherence pathway is moderated by race, such that this mediation pathway is stronger for African American youth, explaining racial disparities in metabolic control.

**Type 1 Diabetes**

Type 1 diabetes is an autoimmune disorder of the pancreas that affects as many as three million Americans and costs the health care system $14.9 billion each year.
Type 1 diabetes is caused by the immune system attacking and destroying beta cells, which are endocrine producing cells located within the islets of Langerhans in the pancreas. Beta cells are responsible for the secretion of insulin, a hormone that promotes the conversion of sugar, starches, and other foods, into daily energy required for the body's survival. Beta cells respond to escalated blood glucose levels by releasing insulin, which then converts glucose into usable energy for the body, thus restoring blood glucose concentration to a healthy level. However, in individuals with type 1 diabetes, the immune system has attacked and destroyed beta cells resulting in elevated levels of blood glucose. Instead of converting blood glucose into energy, the lack of insulin causes glucose to remain stored in the blood, which eventually leads to serious damage to the body if left untreated (American Diabetes Association [ADA], 2014; JDRF, 2014).

In order to survive, individuals with type 1 diabetes must receive insulin injections daily. Administration of insulin to the body occurs through either insulin pens and syringes, or an insulin pump. Strict management and care is crucial for individuals living with type 1 diabetes to achieve positive health outcomes. Individuals with type 1 diabetes must closely monitor their blood glucose levels and administer insulin as needed in order to achieve the ADA recommended target range for glucose levels (70-130 mg/dl; ADA, 2014). However, adherence to the treatment regimen is difficult for many individuals with type 1 diabetes to achieve, particularly children and adolescents (Helgelson & Palladino, 2012). Poor monitoring of glucose levels can lead to periods of both hypoglycemia and hyperglycemia. Hypoglycemia, or low blood sugar, occurs when blood glucose levels drop below 70 mg/dl. Symptoms of hypoglycemia include fatigue,
dizziness, sweating, and confusion. Severe hypoglycemia can lead to seizures and loss of consciousness. Many negative symptoms and health complications also accompany hyperglycemia, or high blood sugar (ADA, 2014). Notably, prolonged hyperglycemia is linked with microvascular complications, including diabetic nephropathy, neuropathy, and retinopathy, and macrovascular complications, including coronary artery disease and stroke, as well as a decline in cognitive functioning (Cukierman-Yaffe et al., 2009; Fowler, 2008).

**Impact of Depressive Symptoms on Metabolic Control**

Managing a rigorous chronic illness such as diabetes imposes immense stress onto youth, including pressure to follow a strict treatment regimen while trying to fit in with peers and worrying about their future health (Hains, Berlin, Davies, Parton, & Alemzadeh, 2006). This stress has major psychological consequences for youth, increasing the likelihood of mental health problems (Eiser, 1990). In fact, a review of the literature reports children and adolescents with type 1 diabetes are at an elevated risk for developing Major Depressive Disorder (MDD; Dantzer, Swendsen, Maurice-Tison, & Salamon, 2003). The presence of mental health problems, in turn, can impact diabetes management, control, and outcomes. A review of the literature by Greydanus and Hofmann (1979) concluded that psychological factors strongly influenced the course of diabetes, including metabolic control and management adherence. However, despite the high incidence of depression in youth with type 1 diabetes and the negative implications of depressive symptoms for metabolic control and adherence, the underlying cause of this relationship has yet to be determined.
Depressive symptoms are a significant concern in youth with type 1 diabetes because depression is associated with poor quality of life and can result in inferior overall functioning, including worse diabetes related outcomes and metabolic control (Bernstein et al., 2013; Grey, Whittemore, & Tamborlane, 2002). Specifically, a review by Johnson et al. (2013) found a significant association between depressive symptoms and higher HbA1c levels in 14 of 15 studies examining the relationship. Grey et al. (2002) further elucidates this finding in a review of the literature examining correlates of depression, in which the authors found that approximately 20% of the variance in metabolic control was accounted for by depression in youth.

Santos and colleagues (2013) highlighted this relationship between depression and metabolic control in a recent study. This cross-sectional study evaluated the relationships between metabolic control and knowledge about diabetes, resilience, depression and anxiety among 85 adolescents and young adults aged 11 to 22 years with type 1 diabetes. Depression, measured using the Hospital Anxiety and Depression Scale (HADS), was the only variable that maintained a significant association with HbA1c after including all the other variables in a linear regression analysis (Santos et al., 2013).

Although there is overwhelming empirical evidence that indicates depressive symptoms are related to metabolic control, no study has identified how depressive symptoms act on metabolic control. One theory that explains this depressive symptom-metabolic control link focuses on the role of adherence as a mediator of the relationship.

**Adherence as a Mediator**

Many researchers theorize that depressive symptoms are associated with metabolic control through an influence on adherence behaviors. Common symptoms of
depression include fatigue, loss of appetite, and decrease in physical activity and motivation (American Psychiatric Association, 2013). These symptoms have important implications for individuals with type 1 diabetes. Depressive symptoms may interfere with daily treatment and adherence behaviors through the lack of energy to maintain a healthy lifestyle, and decreased motivation and attention toward treatment goals (Bernstein et al., 2013). Individuals struggling with depressive symptoms may have difficulty with self-care tasks, resulting in neglect of adherence behaviors, which in turn affects metabolic control. Therefore, one theory proposes a relationship between depressive symptoms and adherence such that adherence mediates the relationship between depressive symptoms and HbA1c. Although the mediating role of adherence has not received much attention in the literature, research does show a strong relationship between depressive symptoms and adherence, suggesting the important role of adherence in the depressive symptoms-metabolic control link.

Increased depressive symptoms in youth with type 1 diabetes have been associated with worse adherence in the literature (Hood et al., 2006; Korbel et al., 2007). Hood and colleagues (2006) examined the relationship between depressive symptoms and adherence behaviors in a cross-sectional study with a majority European American sample of 145 youth aged 10 to 18 with type 1 diabetes. Depressive symptoms were assessed using the Children's Depression Inventory (CDI) and adherence was measured based on blood glucose monitoring (BGM) frequency from meter downloads. Youth with elevated depressive symptoms were more likely to have lower BGM frequency and have higher HbA1c values (Hood et al., 2006). These findings indicate support for two of the four criteria for mediation; with significant relationships between the predictor
variable, or depressive symptoms, and both the dependent variable, or HbA1c, and the theorized mediator, or BGM frequency (Baron & Kenny, 1986). Although mediation was not explicitly tested, these findings highlight the importance of examining a mediation model to test for the role of adherence in the depressive symptoms-HbA1c link.

Another study by Hood and his colleagues (2011) highlights the central role of adherence in the depressive symptoms-metabolic control link, particularly for youth at optimal metabolic control. The researchers conducted a six-month longitudinal study with a sample of 145 majority European American youth aged 13 to 18 with type 1 diabetes. Results showed the powerful impact of adherence on the depressive symptoms-metabolic control link for youth with optimal metabolic control at baseline. For these youth, adherence levels significantly predicted change in HbA1c such that youth with increasing depressive symptoms and low adherence were in the worst metabolic control at follow-up, whereas for youth with high adherence levels, increasing depressive symptoms did not impact metabolic control at follow-up. This relationship did not hold true for adolescents in poor control at baseline. For the adolescents in poor control, increasing depressive symptoms resulted in worse metabolic control regardless of adherence levels, suggesting poor metabolic control is a predictor for worse outcomes independently. Therefore, the results of this study provide further support for adherence as an important factor in the depressive symptoms-HbA1c relationship (Hood et al. 2011).

In the only study directly examining a mediational model, McGrady et al. (2009) examined a sample of 276 adolescents with type 1 diabetes (87% European American) to
determine whether the association between total depressive symptoms and metabolic control was mediated by BGM frequency. Results of a regression analysis found depressive symptoms to be associated with lower BGM frequency and higher HbA1c. Lower BGM frequency was associated with higher HbA1c. When including depressive symptoms and BGM frequency together, only BGM frequency remained associated with HbA1c. These results provide the first empirical evidence that depressive symptoms have their effect on metabolic control indirectly, reducing adherence behaviors. One goal of the current study is to replicate this finding, taking a broader look at adherence behaviors beyond BGM frequency.

**Race as Moderator of the Mediation Pathway**

A second goal of the current study is to examine the significance of this mediation model in explaining group differences to account for racial disparities in metabolic control. A number of studies have found a relationship between race and metabolic control with higher HbA1c levels reported among African American youth with type 1 diabetes (Delamater et al., 1999; Hassan et al., 2006; Hilliard et al., 2013; Kamps et al., 2010; Naar-King et al., 2006). Several possible explanations for this difference have been proposed, including racial differences in SES, parental education level, caregiver marital status, and insulin treatment plan, but no definitive explanations have emerged (Hassan et al., 2006; Hilliard et al., 2013). To date, no studies have reported significant racial differences in the prevalence of depressive symptoms in youth with type 1 diabetes, however, there are many findings indicating that the course and impact of depressive symptoms may be different among African Americans.
Studies have found mixed results in the prevalence of depression in African American and European American youth. Some studies have reported higher rates of depressive symptoms among African American youth (Kistner, David-Ferdon, Lopez, & Dunkel, 2007; Roberts, Roberts, & Chen, 1997) while others have reported no differences between rates for African Americans and European Americans (Schraedley, Gotlib & Hayward, 1999; Siegal, Aneshensel, Taub, Cantwell, & Driscoll, 1998). Explanations for this discrepancy across studies are partly due to the differences in regions where samples are drawn, age of participants, and cultural validity of assessment methods, which make conclusions about racial differences in the prevalence of depression difficult to determine. For example, a study by Wright, Aneshensel, Botticello, and Sepulveda (2005) found significantly higher depressive symptoms only for African American adolescents living within predominantly European American neighborhoods. In addition, differences may be explained by risk and protective factors beyond race, including parent education level, household income, exposure to violence, and insurance status (Kennard et al., 2006). Therefore, the complex relationship between these variables makes it difficult to tease apart and understand true differences in prevalence.

Despite the discrepant results in the prevalence of depression among African American youth, racial disparities clearly exist in access to and utilization of mental health services. Racial minorities are disproportionately affected by barriers to care including stigma, cost, comorbidity of mental illnesses, cultural competence of health care providers, and insurance status (Primm et al., 2010; Zito et al., 2005). For example, major disparities exist across access to health insurance, with African Americans disproportionately represented in Medicaid populations (Zito et al., 2005)
However, studies examining racial disparities in access to and utilization of mental health services consistently show poorer access, utilization, and quality of mental health services for African American youth even after adjusting for socioeconomic, family, and regional factors (Anderson & Mayes, 2010; Cuffe, Waller, Cuccaro, Pumariega, & Garrison, 1995; Cunningham & Freiman, 1996; Flores & Tomany-Korman, 2008; Zahner & Daslakis, 1997). The National Medical Expenditure Survey, a large community survey of 6,216 youth, found African American children and adolescents were significantly less likely than European American children and adolescents to have made a mental health outpatient visit in the previous year (Cunningham & Freiman, 1996). These differences were not attributable to SES, family-related, or regional differences between groups. A study by Cook, Barry, and Busch (2013) found significant racial disparities, defined as any difference in health care not explained by clinical appropriateness, need, or patient preferences, in overall mental health care and psychotropic drug use in youth ages 5 to 21. Results of the study found European American youth were twice as likely as African American youth to initiate treatment for mental health even after controlling for education, income, and insurance.

Studies examining utilization of mental health services for the treatment of depression have found similar racial disparities. A study of school-aged children and adolescents by Sen (2004) found that African American youth were significantly less likely to seek help for depressive symptoms than European American youth. Similarly, Cummings and Druss (2011) found that African American adolescents aged 12 to 17 diagnosed with MDD in the previous year were significantly less likely to receive any form of treatment for MDD than European American adolescents including
antidepressant medication and outpatient treatment from a mental health specialist or medical provider. These differences in treatment persisted after adjusting for family income and insurance status.

One explanation this racial disparity persists beyond financial factors may be cultural differences in the stigma towards mental health and a history of distrust in the health care system. For example, a study by Sussman et al. (1987) found the proportion of African Americans who feared mental health treatment was 2.5 times greater than the proportion of European Americans. Further, in a study by Cooper-Patrick et al. (1997) African Americans were more likely to seek informal help or no professional help at all for mental health concerns with the greatest reasons for resisting treatment including perceived community stigma towards mental illness and distrust in the health professionals.

Taken together, these findings indicate that depressive symptoms may have more debilitating and long-term effects for African American youth due to less access to care, less utilization of mental health services, and poorer quality of care. Therefore, symptoms of depression are more prominent in the lives of African American youth such that daily diabetes-related tasks and adherence to the treatment regimen becomes more difficult to manage. Therefore, these differences will amplify the depressive symptoms-adherence pathway, with depressive symptoms having more of an impact on adherence in African American youth. Although examining the history of access to mental health services and attitudes towards psychological disorders is out of the scope of the current study, based on the previous literature, it is hypothesized that depressive symptoms will pose more of a burden for African American youth due to less care and support for their
symptoms. If this is the case, depressive symptoms in African American youth may have a greater effect on adherence, and ultimately metabolic control.

**Current Study**

Past literature supports the negative impact of depressive symptoms on metabolic control and diabetes outcomes (Bernstein et al., 2013; Grey et al., 2001; Hislop et al., 2007; Hood et al., 2011; Johnson et al., 2013; Santos et al., 2013). However, the mechanism driving these poor outcomes and explaining the depressive symptoms-metabolic control link have yet to be determined. Depressive symptoms have been linked to worse adherence, which is a main factor leading to poor metabolic control (Hood et al., 2006; Korbel et al., 2007). However, only one study to date has directly examined adherence as a mediator of the depressive symptoms-metabolic control link (McGrady et al., 2009). Therefore, the current study expands on the literature by directly testing the mediational role of adherence. The current study also extends the literature with the inclusion of a self-report measure of adherence collected from both parent and child in addition to the standard measure of adherence, BGM frequency. Although BGM frequency has been shown to be a strong predictor of metabolic control (Hood, Peterson, Rohan, & Drotar, 2009), this measurement is limited by the assumption that BGM frequency is representative of all self-care behaviors. However, many other self-care behaviors make up good adherence to the treatment regimen, including exercise, diet, and insulin administration, and are not captured measuring only BGM (Helgelson & Palladino, 2012). For example, a study by Helgeson, Honcharuk, Becker, Escobar, and Siminerio (2011) examined the separate effects of frequency of BGM and self-reported adherence, using the Self-Care Inventory, on metabolic control. Results found that both
BGM frequency and self-care indexes were significant, independent predictors of HbA1c. Therefore, the current study examined adherence beyond BGM frequency to capture a more complete range of self-care practices on diabetes outcomes.

In addition, the current study examined group differences between African American and European American youth to account for differences in metabolic control. Research has long established an association between race and metabolic control, with African American youth consistently appearing in worse control (Delamater et al., 1999; Hassan et al., 2006; Hilliard et al., 2013; Kamps et al., 2010; Naar-King et al., 2006).

However, no study has elucidated a cause for this racial disparity. The current study is the first to test for group differences within the depressive symptoms-adherence link, to account for racial differences in metabolic control (see figure 1).

**Hypotheses**

**Goal 1:** To examine the unique relations among study variables.

- **Hypothesis 1:** Depressive symptoms will be correlated with HbA1c such that youth with more depressive symptoms will display higher, and worse, HbA1c levels.
- **Hypothesis 2:** Depressive symptoms will be correlated with adherence such that youth with more depressive symptoms will have lower adherence composite scores.
- **Hypothesis 3:** Adherence will be correlated with HbA1c such that lower adherence composite scores will be associated with higher HbA1c levels.

**Goal 2:** To examine the mediational role of adherence in the relationship between depressive symptoms and HbA1c.
• Hypothesis 1: Adherence will mediate the relationship between depressive symptoms and HbA1c.

Goal 3: To examine the role of ethnicity in the mediation model.

• Hypothesis 1: Race will be correlated with HbA1c such that African American youth will display higher HbA1c levels.

• Hypothesis 2: The mediating role of adherence in the relationship between depressive symptoms and HbA1c will depend on race, such that there will be a stronger association between depressive symptoms and adherence among African American youth than among European American youth.
Method

Participants

One hundred youth with type 1 diabetes with standing appointments at an outpatient endocrinology clinic located within New Orleans Children's Hospital were recruited and enrolled for participation in the study. Individuals were eligible for participation if they ranged in age from 5 to 20 years old and had been diagnosed with type 1 diabetes at least one year before enrollment. Exclusion criteria for participation were as follows: patients with type 2 diabetes; patients under 18 without a parent or legal guardian present at the time of the appointment to give consent for participation; patients who did not receive lab A1c draws that day or who received their lab work outside of the hospital; and patients who did not have their blood glucose meters in clinic at the time of enrollment.

Of the 100 youth recruited for participation, 86 were included in the final analysis. Twelve participants were excluded from analysis for the current study based on self-identified race as other than European American or African American since the current study focused on disparities between African American and European American youth. One participant was mistakenly enrolled into the study at two time points, and one participant had type 2 diabetes. Table 1 describes the demographic and clinic characteristics of the 86 participants collectively and for each racial group. Participants ranged in age from 5 to 20 (\( M = 13.59, \ SD = 3.49 \)). Participants were 51.2% female (\( n = \))
44), 61.6% European American ($n = 53$), with 55.8% receiving Medicaid or government assistance ($n = 48$). Calculated Federal Poverty Levels (FPL) ranged from 18% below to 747% above the federal criteria of poverty with the average participant 172% above criteria for federal poverty ($M = 272.87$, $SD = 198.80$). FPL was determined using calculations from the 2015 federal guidelines for poverty based on the number of individuals living in the household and household income (U.S. Department of Health and Human Services, 2015). The average household had 4.17 ($SD = 1.46$) members. Seventy-three percent ($n = 63$) of mother's self-reported their highest level of education as some college completed or less.

There were significant group differences between African American participants and European American participants, with medium to large effects observed for all relationships (Cohen, 1998). The families of African American participants were more likely to report lower income, resulting in significant differences in the percent below and above criteria for federal poverty, $X^2 = 31.22$, $p < .001$, and receiving Medicaid or government assistance, $X^2 = 18.30$, $p < .001$. Such that 3.8% of European American families were at or below the FPL level with 37.7% receiving Medicaid or government assistance, and 58.1% of African American families were at or below the FPL level with 84.8% receiving Medicaid or government assistance. Significant differences were also observed for educational status, $X^2 = 5.84$, $p = .02$, such that 71.7% of European American mothers reported their highest education at some college or higher, and 45.2% of African American mothers reported some college or higher. African American youth were also significantly underrepresented in the use of insulin pump therapy, $X^2 = 26.37$, $p$
< .001, such that 64.2% of European American youth were on pump treatment, whereas
9.1% of African American youth were currently on pump treatment.

Recruitment and Procedure

This study received approval from the Louisiana State University Health Sciences
Center and Children's Hospital combined IRB review board. Participants were recruited
weekly during scheduled appointments at the hospital's outpatient diabetes clinic from
March 2014 to October 2014. A nurse coordinator first approached the child and parent
pair, provided a brief overview of the study and the study requirements, and asked for
interest in participating. If both parent and child agreed to participate, the hospital's
clinical trial coordinator then distributed informed consent forms to both parent and child
and provided a further detailed description of the study. The consent forms provided a
comprehensive explanation of the study goals and procedures, as well as contact
information for study coordinators and lead investigators. Once consent forms were
completed, a research assistant then administered the demographic questionnaire to the
caregiver and all appropriate psychosocial forms to the caregiver and child. The research
assistant remained in the room while all measures were completed to provide assistance
as needed. The research assistant also completed an enrollment and clinic visit form for
each participant, which provided routine clinic data.

Measures

Demographic and clinic characteristics. Demographic questionnaires provided
information on age, gender, self-identified race, family make up, parental education and
employment status, insurance status, and household income. Clinic characteristics
reported on were collected from each patient's medical chart at the time of the clinic visit
including height, weight, blood pressure, and treatment type. Several variables were created from this information.

Race was coded based on racial identification options on the demographic questionnaire as Non-Hispanic white, Non-Hispanic black, Hispanic, Native American or Alaska Native, Asian/Pacific Islander, or Other. As noted above, only African American and European American participants were included in analyses; African American was coded as 1 and European American as 0.

Treatment type identified the current insulin treatment method for each participant, with participants either using an insulin pump, multiple daily injections (MDI), or conventional insulin treatment (2DI). Multiple daily injections identified participants receiving 4-5 insulin injections per day with corrective basal injections. Conventional insulin treatment identified patients receiving two daily insulin injections without correction. Treatment type was expressed as a categorical variable measured for intensity, with pump treatment being regarded as superior, 2DI was coded 1, MDI was coded 2, and pump was coded 3.

The decision to create a composite variable to represent participant's socio-economic status (SES) was made in order to preserve study power and prevent losing degrees of freedom when entering multiple covariate variables in analyses. As noted above, family poverty level was determined using federal calculations of FPL based on the number of individuals living in the household and household income, using the midpoint of each categorical income range. Maternal education was expressed as a categorical variable coded from 1 to 6 with higher values representing more education. FPL and maternal education were standardized and included in a factor analysis with
other standardized variables (i.e., insurance status, father education status) to determine the best indicators of SES. These variables were chosen to represent SES using principle axis factoring in which maternal education and FPL received the highest loadings within the one-factor solution (Jolliffe, 2002). The composite variable using the average of the standardized variables was chosen instead of the factor score to preserve the sample size from the values lost in the factor score. Very strong correlations between the averaged composite and the latent variable supported this decision.

**HbA1c.** Hemoglobin A1c (HbA1c) was measured during each participant's clinic visit at the time of study enrollment by the Children's Hospital laboratory using a commercial immunoassay with results reported in National Glycohemoglobin Standardization Program (NGSP) equivalents (Little et al., 2001). Subjects with HbA1c values greater than or equal to 16% are above the upper reporting limit of the assay and were omitted from analyses.

**Depressive symptoms.** All participants eight years of age or older completed the Center for Epidemiological Studies Depression Scale for Children (CES-DC). Due to the age restrictions of the CES-DC, four participants under the age of eight did not complete the measure, and therefore do not have depressive symptom scores included in analyses. The CES-DC is a 20-item self-report depression inventory, adapted from the Center for Epidemiological Studies Depression Scale (CES-D) for adults, to assess levels of depression in children and adolescents (Weissman, Orvaschel, & Padian, 1980). Reliability for the CES-DC in samples ranging 8 to 17 years old was reported in a representative standardization sample consisting of 148 inpatient children and adolescents including 58 African Americans showing acceptable internal consistency (α
20

= .84) as well as acceptable concurrent validity with the Children's Depression Inventory
(r = .44; Faulstich, Carey, & Ruggiero, 1986). However, support for the measure for use
in up to 23 year olds has been found (Fendrich, Weissman, & Warner, 1990). Internal
consistency was high within the sample for the current study (α = .90).

The CES-DC measures depressive symptoms experienced over the previous
week. Questions are formatted in declarative statements, such as "I was bothered by
things that usually don't bother me" and "I felt down and unhappy" and responses are
rated on a four point Likert scale ranging from "not at all" (0) to "a lot" (4). Less than
one percent of data was missing on the CES-DC. To correct for missing data, each
participant's mean score was calculated, and mean scores were entered into cells with
missing data. A total depressive symptoms score was obtained by summing all item
ratings, with items 4, 8, 12, and 16 reverse coded. Possible total scores range from 0 to 60
with higher scores indicating more severe depressive symptoms. A cutoff score of 15
indicates clinically significant levels of depression (Weissman et al., 1980).

Adherence. Two measures of adherence to type 1 diabetes management
behaviors were collected. The first measure was the Diabetes Self Management Profile-
Self Report (DSMP-SR), designed to assess levels of adherence to the diabetes treatment
regimen. The DSMP-SR is an adapted version of the diabetes self-management profile
(DSMP) to be administered in situations with time restraints (Wysocki, Buckloh, Antal,
Lochrie & Taylor, 2012). The DSMP-SR consists of 24 items assessing compliance with
various daily adherence activities (i.e., diet, exercise, insulin administration, blood
glucose monitoring). The DSMP-SR is structured in two versions, flexible regimen and
conventional regimen, in order to accurately assess adherence for patients on all
treatment methods. The flexible regimen version of the DSMP-SR was administered to all patients on insulin pump therapy or MDI injections, and the conventional version was administered to all patients receiving conventional long acting insulin.

The DSMP-SR consists of both parent and youth report versions. All children over the age of eleven completed the youth version of the DSMP. All parents completed the parent version of the DSMP. For children under the age of eleven, only parent report was collected. All available scores were used in the final adherence composite score, such that for participants 11 years old and older composite scores consisted of parent report, self-report, and BGM frequency, and for participants younger than 11 years composite scores consisted of parent report and BGM frequency. Within the current sample, the DSMP-SR showed acceptable internal consistency for parent ($\alpha = .81$) and youth ($\alpha = .78$) report with good agreement between parent and youth report ($r = .59, p < .001$). Less than one percent of data was missing on both versions of the DSMP-SR. To correct for missing data, each participant's mean score was calculated, and mean scores were entered into cells with missing data. A total adherence score was obtained by summing all item scores. Possible scores range from 0-86, with higher scores indicating better adherence to type 1 care.

Second, BGM frequency calculated as a measure of adherence through the collection of each participant's blood glucose meter printouts. Meter downloads have been found to be significantly more reliable measures of adherence among adolescents with type 1 diabetes than both adolescent self-report of blood glucose monitoring frequency and care-giver report of blood glucose monitoring frequency (Guilfoyle, Crimmins, & Hood, 2011). Each patient's printout was collected and entered into a
database storing up to 10 daily blood glucose checks for 3 months. Blood glucose data was then used to calculate the average number of checks per day across all days reported for a three-month span. This average represents BGM frequency.

Scores on the DSMP-SR parent and child report and BGM frequency were standardized and a composite adherence score was calculated by averaging the scores following the same procedure as previously described for the created SES composite variable. This decision was supported by the very strong correlation between the latent variable and the averaged composite.

**Planned Analyses**

Correlation analyses were conducted between all variables derived from demographic and clinic data and variables of primary interest (i.e., HbA1c, depressive symptoms, adherence) to determine any demographic variables to be covaried in analyses. Covariates controlled for in analyses were determined based on simple correlations with outcome variables. Based on correlations, age, treatment type, and SES were entered as covariates in the general linear models. Due to the small sample size and the relatively small effects anticipated, marginal p values of < .10 were interpreted as significant to avoid making Type II errors and increase the power necessary to detect small but practical effects (Kirk, 2001).

**Goal 1:** To examine the unique relations among study variables.

- **Hypothesis 1:** Depressive symptoms will be correlated with HbA1c such that youth with more depressive symptoms will display higher, and worse, HbA1c levels.
Planned Analysis: First, a general linear regression will be calculated between CES-DC scores HbA1c with HbA1c entered as the dependent variable and CES-DC scores entered as the predictor variable. Second, a general linear regression will be calculated controlling for covariates predicting HbA1c, with all covariates entered in step one, and CES-DC entered in step two.

- **Hypothesis 2:** Depressive symptoms will be correlated with adherence such that youth with more depressive symptoms will have lower adherence composite scores.

  - Planned Analysis: First, a general linear regression will be calculated between CES-DC scores and adherence composite scores with adherence entered as the dependent variable and CES-DC scores entered as the predictor variable. Second, a general linear regression will be calculated controlling for covariates predicting adherence, with all covariates entered in step one, and CES-DC entered in step two.

- **Hypothesis 3:** Adherence will be correlated with HbA1c such that lower adherence composite scores will be associated with higher HbA1c levels.

  - Planned Analysis: First, a general linear model will be calculated between adherence composite scores and HbA1c with HbA1c entered as the dependent variable, and adherence entered as the predictor variable. Second, a general linear regression will be calculated controlling for covariates predicting HbA1c, with all covariates entered in step one, and adherence composite entered in step two.
**Goal 2:** To examine the mediational role of adherence in the relationship between depressive symptoms and HbA1c.

- **Hypothesis 1:** Adherence will mediate the relationship between depressive symptoms and HbA1c.
  - Planned Analysis: A mediation analysis will be used provided that significant results are found for Hypotheses 1 through 3 of Goal 1, as required for mediation by Baron and Kenny (1986). Multivariate analyses using general linear modeling will be conducted using SPSS with and without covariates. First, the effect of depressive symptoms on metabolic control will be tested. Second, the effect of depressive symptoms on adherence will be tested. Third, adherence and depressive symptoms will be entered together to test the mediational role of adherence. A Sobel test of significance will be calculated to determine the significance of the mediation (Sobel, 1982).

**Goal 3:** To examine the role of ethnicity in the mediation model.

- **Hypothesis 1:** Race will be correlated with HbA1c such that African American youth will display higher HbA1c levels.
  - Planned Analysis: First, a general linear regression will be calculated between race and HbA1c with HbA1c entered as the dependent variable, and race as the predictor variable. Second, a general linear regression will be calculated controlling for covariates predicting HbA1c, with all covariates entered in step one, and race entered in step two.
Hypothesis 2: The mediating role of adherence in the relationship between depressive symptoms and HbA1c will depend on race, such that there will be a stronger association between depressive symptoms and adherence among African American youth than among European American youth.

Planned Analysis: The mediation model tested in Goal Two will be evaluated with the addition of race as a moderator of the relationship between depressive symptoms and adherence. By fitting moderated mediation, using Mplus (Muthén & Muthén, 1998), the pathway of the race by depressive symptoms term as it predicts adherence will be evaluated. The difference in scores associated with the comparison of the indirect mediation path tested in Goal Two and the moderated mediation path tested in Goal Three will also be evaluated to confirm the role of race as a moderator. The model will include the covariates
Results

Descriptive Statistics on Study Variables

Descriptive statistics are presented in Table 1 including all variables used to create composite variables used in analyses (i.e., SES and adherence). Overall, the sample presented in poor metabolic control with the average HbA1c of the sample meeting the at-risk criteria (≥ 9.5%) for future diabetes-related complications (National Institute for Health and Care Excellence, 2004). Mean clinic BG of the sample was 194.02 (SD = 70.46) and mean HbA1c was 9.49% (SD = 1.76%). Although this was a community sample, 35.4% (n = 29) of youth met criteria for clinical levels of depressive symptoms based on the CES-DC cut off of scores ≥ 15. Overall, the sample reported fair adherence as indicated by about normative levels of self-report adherence (Lewin et al., 2010) on both the parent (M = 60.21, SD = 11.37) and child DSMP-SR (M = 53.80, SD = 11.72). The frequency of BGM per day ranged from 0 to 10 (M = 3.75, SD = 2.65), with mean levels just below the ADA recommended frequency of 4-5 BG checks per day (ADA, 2014).

Table 2 provides means, standard deviations, and intercorrelations between each study variable as well as demographic variables of interest. Interrelationships between variables were in the expected direction and replicated those found in the literature. For example, the only gender-related association observed was with depressive symptoms, r = .20, p = .07. Girls tended to report higher levels of depressive symptoms, consistent
with prior research (Delamater et al., 1999; Hilliard et al., 2013; Hood et al., 2011). As expected, age was significantly associated with adherence levels, $r = -.46, p < .001$, with adherence decreasing with older age (Helgelson & Pallidino, 2012). In addition, African American youth were more likely to be in worse metabolic control, $r = .42, p < .001$, and have lower adherence levels, $r = -.22, p = .04$. However, no difference in depressive symptoms was observed between African American youth and European American youth. Treatment type was associated with SES, $r = .27, p = .01$, race, $r = -.54, p < .001$, and adherence, $r = .27, p = .01$, with participants on conventional insulin regimens more likely to be African American, lower SES, and have worse adherence levels.

**Goal 1: To Examine the Unique Relations Among Study Variables**

The first goal of the study was to examine the associations among all study variables in order to establish relationships previously found in the literature and to provide support for the first three statistical requirements of mediation (Goal 2; Baron & Kenny, 1986). For mediation to be present, first, the independent variable, in this case depressive symptoms, must be significantly associated with the dependent variable, in this case HbA1c. Results indicate that depressive symptoms account for a significant proportion of variance in HbA1c, $B = .04, SE = .02, p = .07$, with depressive symptoms explaining 3.9% of the variance in HbA1c. Second, the independent variable must be significantly associated with the hypothesized mediator, in this case adherence. Results indicate that depressive symptoms account for a significant proportion of variance in adherence levels, $B = -.03, SE = .01, p = .004$, with depressive symptoms explaining 9.8% of the variance in adherence. Third, the hypothesized mediator must be
significantly associated with the dependent variable. Results indicated that adherence accounts for a significant proportion of the variance in HbA1c, $B = -0.98$, $SE = 0.21$, $p < 0.001$, with adherence explaining 21.3% of the variance in HbA1c. These associations provide support for the first three statistical requirements of mediation (tested in Goal 2).

When the covariates of age, treatment type, and SES were included in the first step of the regression equations, the association between depressive symptoms and HbA1c was no longer significant, $B = 0.03$, $SE = 0.02$, $p = 0.13$, but the other associations remained significant. Specifically, depressive symptoms predicted adherence, $B = -0.02$, $SE = 0.01$, $p = 0.011$, with depressive symptoms explaining 5.8% of the variance in adherence beyond covariates, and adherence predicted HbA1c, $B = -0.92$, $SE = 0.23$, $p < 0.001$, with adherence continuing to explain 12.8% of the variance in HbA1c beyond covariates.

**Goal 2: To Examine the Mediation Role of Adherence in the Relationship Between Depressive Symptoms and HbA1c**

The second goal of the study was to test adherence as a mediator of the relationship between depressive symptoms and HbA1c. Based on the significant results found in Goal 1, the mediation model was tested without covariates. The significant results found in Goal One were used to support criteria one through three of mediation. The fourth statistical criterion of mediation is that the association between the independent variable and the dependent variable must be reduced after controlling for the hypothesized mediator (Baron & Kenny, 1986). To test this, HbA1c was regressed on adherence and depressive symptoms together. In this model, adherence remained a significant predictor of HbA1c, $B = -0.96$, $SE = 0.22$, $p < 0.001$, and the effect of depressive symptoms
symptoms was no longer significant, $B = .01$, $SE = .02$, $p = .59$, satisfying the final
criterion for mediation and producing a medium sized indirect effect of .14 (Cohen, 1988;
see figure 2). A Sobel test (Sobel, 1982) evaluating the magnitude of the indirect effect
of depressive symptoms on HbA1c through adherence was significant, $z = 2.40$, $SE = .01$,
$p = .02$. In line with hypotheses, the results suggest the relationship between depressive
symptoms and metabolic control is mediated by adherence. As noted above, the
relationship between depressive symptoms and HbA1c did not hold once covariates were
controlled; therefore, mediation for this model using covariates was not evaluated.

**Goal 3: To Examine the Role of Race in the Mediation Model**

To examine race as a potential moderator to the mediation pathway, two
hypotheses were tested. First, hypothesis one, stating that race would be associated with
metabolic control such that African American youth would have higher HbA1c levels,
was examined using general linear modeling with and without controlling for covariates.
Race accounted for a significant proportion of variance in HbA1c, $B = 1.50$, $SE = .36$, $p<.001$, with African American youth displaying worse metabolic control than European
American youth and race explaining 17.4% of the variance in HbA1c. This relationship
remained significant after controlling for SES, age, and treatment type, $B = .97$, $SE = .47$,
$p = .043$, however, only 3.9% of the variance in HbA1c was explained by race beyond the
effects of covariates.

Next, moderated mediation was used to evaluate the role of race as a moderator of
the relationship between depressive symptoms and adherence in the mediation model.
Due to the conceptual significance of covariates, particularly SES, in analyses examining
effects by race, the model was tested controlling for age, SES, and treatment type. For
moderation to be present, the pathway of the interaction term between the independent variable, depressive symptoms, and hypothesized moderator, race, must be a significant predictor of the mediator, adherence. The hypothesis was not supported. Although the paths modeled demonstrate relationships in the predicted direction (see Figure 3), neither the pathway, posterior median, (posterior median = .004, posterior SD = .02, p = .40), nor the difference in scores between the mediation and the moderated mediation models, Z = .003, SE = .02, p = .40, were statistically significant.
**Discussion**

The current study set to explore three interrelated goals. The first goal of the study was to understand the unique relationships between depressive symptoms, adherence, and metabolic control in youth with type 1 diabetes. The second goal examined the mediating role of adherence in the depressive symptoms-metabolic control link. The final goal examined the moderating role of race on the depressive symptoms-adherence link. The small sample size reduced the power of the study to detect effects using traditional significance levels ($p < .05$). To reduce the chance of Type II error, a more liberal significance level of $p < .10$ was used to identify significant effects. Despite this non-traditional approach, modest effect sizes were found (Cohen, 1988), therefore supporting the practical and meaningful significance of results (Kirk, 2001). However, based on the recommendations from Jaccard, Guilamo-Ramos, Johansson, and Bouris (2006), concrete conclusions should not be drawn until replication of the study design has occurred.

Despite sample size limitations, the design and methodology were major strengths of the current study, expanding on previous literature in many important ways. First, the current study extended existing literature by explicitly testing the mediational role of adherence in the relationship between depressive symptoms and metabolic control. The current study also adds to the literature examining racial disparities in metabolic control by testing the moderating effects of race on the depressive symptoms-adherence. In addition, the current study included key covariates necessary when examining racial
differences in order to eliminate alternate causal factors, including SES and treatment type. SES is a crucial factor to examine in addition to race in order to distinguish the biological from the social dimensions and understand the confounding and interacting relationships of these variables (Committee on Pediatric Research, 2000).

**Examining the Mediating Role of Adherence**

Findings of the study indicate that adherence served as a mediator in the relationship between depressive symptoms and metabolic control. Although depressive symptoms had a small but significant direct effect on HbA1c, results of the study show the effects of depressive symptoms manifest on metabolic control through an indirect pathway, with adherence serving as a mechanism through which depressive symptoms contribute to metabolic control. Multiple studies have demonstrated the relationship between depressive symptoms and adherence (Hood et al., 2006; Korbel et al., 2007) and depressive symptoms and metabolic control (Grey et al., 2002; Johnson et al., 2013; Santos et al., 2013) without examining the nature of the relationships between these variables. The results of this study show that adherence levels have a direct role in the depressive symptoms-metabolic control link, with the behavioral expressions of depression negatively impacting adherence.

Higher depressive symptoms have been associated with lower self-efficacy, negative affect surrounding BGM, loss of energy, and trouble concentrating (Hood et al., 2006; Stewart, Rao, & White, 2005). Youth with type 1 diabetes and elevated depressive symptoms may have trouble initiating tasks for diabetes management and believing they will be effective. For example, a common symptom of depression is a lack of energy and motivation to complete daily tasks (Hood et al., 2011). This can conflict with key
adherence practices due to decreased motivation to perform complex self-care practices, including frequently checking blood sugar levels and correctly administering insulin.

The current study supports and expands on the results originally found by McGrady et al. (2009) identifying adherence as a mediator. This study used a composite measure of adherence consisting of three distinct measurements of adherence to the treatment regimen while McGrady et al. (2009) used BGM frequency as the sole illustration of adherence levels. However, relying on BGM frequency alone does not provide information on the entire scope of diabetes self-care behaviors. Therefore, the additional use of the DSMP-SR, which captures a range of daily adherence behaviors beyond BGM, strengthened the results of this study and the support for mediation. Future studies should further test how different aspects of adherence may affect this mediation pathway, examining which self-care behaviors are responsible for this relationship. In addition, the study by McGrady et al. (2009) sampled a majority of European American adolescents with a mean age of 15.6. Therefore, the use of a racially diverse sample in the current study, ranging in age from 5 to 20, help to generalize the findings to younger and minority children.

The results of the current study suggest that treating depressive symptoms in youth may be an effective way to increase adherence rates, and ultimately metabolic control. Therefore, interventions aimed to improve metabolic control through increasing adherence rates must first address depression and the negative impact depressive symptoms play on the ability to achieve ideal management of diabetes. Inability to do so will result in less effective interventions that do not address the entire scope of the problem. However, interventions that first address and target depressive symptoms in
adolescents with type 1 diabetes may be more successful in improving adherence and metabolic control. A first step in this process should be routine depression screening during regular clinic visits. The addition of this simple step has the ability to improve diabetes-related health outcomes and quality of life among children and adolescents with type 1 diabetes (Bernstein et al., 2013).

Although the mediational findings did not hold when covariates were included in the model, the pattern of effects remained the same suggesting that the non-significant finding resulted from a further lack of power with the addition of more variables in the model. Given the power issues of the current study, as well as the strong relationships between depressive symptoms and metabolic control previously found in the literature (Bernstein et al., 2013; Grey et al., 2002), future studies need to replicate the current design with larger samples to determine the veracity of these findings.

In addition, the cross-sectional design of the study limits the ability to make causal conclusions between study variables. Therefore, it cannot be determined if higher depressive symptoms result in worse adherence behaviors among children and adolescents, or if struggles with adherence lead to depressive symptoms. Youth having difficulty adhering could view themselves as a failure, feel helpless at managing their illness, and receive negative feedback from their parents and doctors, which may contribute to elevated depressive symptoms. Therefore, future longitudinal studies must be conducted to tease apart these directional relationships. In addition, the broad age range of participants included in the current study may limit results. Developmental differences in the expression and prevalence of depression and challenges with adherence may change the nature of this mediation model over time. Adolescents experience a
decline in adherence and report the most difficulties with maintaining good self-care practices (Helgeson & Palladino, 2012). In addition, the risk for depression increases in adolescence with the onset of puberty (Son & Kirchner, 2000), with adolescents with chronic illnesses particularly vulnerable to experiencing elevated depressive symptoms (Ghandour, Kogan, Blumberg, & Perry, 2010). Therefore, future research would benefit from stratifying across developmental periods in order to examine these relationships throughout different stages of development.

Furthermore, measuring depressive symptoms exclusively may have resulted in an issue of specificity overlooked in previous research studies (Grey et al., 2002), but which was highlighted in the current study due to the small sample size. For example, depressive symptoms in children and adolescents with type 1 diabetes may be better explained by the daily distress associated with managing a chronic illness. Diabetes-related distress refers to feeling overwhelmed or frustrated due to diabetes-related factors, including managing optimal diabetes outcomes, and can lead to poor coping and problem solving skills, key factors predictive of optimal self-care (Balfe et al., 2013). This lack of perceived control over one's illness can lead to elevated depressive symptoms; however, diabetes-related distress is characterized by negative emotions and behaviors not fully captured in depression scales, including fear of hypoglycemia and negative affect surrounding BGM (Shaban, Fosbury, Cavan, Kerr, & Skinner, 2009). Therefore, measuring only depressive symptoms may miss the effects of these other aspects of distress typical among individuals with type 1 diabetes. For example, a study by Hilliard et al. (2012) examined the link between depressive symptoms, diabetes-related distress, and HbA1c. High diabetes-related distress predicted membership in the high-risk
subgroup, characterized by poor metabolic control and diabetes management, whereas high levels of depressive symptoms did not. The results of this study suggest diabetes specific distress may be more salient to adolescents with type 1 diabetes than general emotional distress or depressive symptoms. Therefore, future studies should examine the unique contribution of diabetes related distress on adherence related behaviors and metabolic control.

**Examining Racial Disparities and Race as a Moderator**

The results of this study found African American youth had lower adherence to their treatment regimen and were in significantly worse metabolic control than European American youth, consistent with previous literature (Delamater et al., 1999; Hassan et al., 2006; Hilliard et al., 2013; Kamps et al., 2010; Naar-King et al., 2006). These relationships held even when controlling for SES, treatment type, and age, however, the findings remain inconclusive regarding the role of race distinctively on metabolic control. African American youth were significantly more disadvantaged than European American youth in terms of SES and treatment type with African American youth more likely to be from lower SES households and on conventional insulin treatment. Although statistical control is a simple way to address these confounds, this approach of removing the variance associated with the variables does not help explain the role these variables play separately and together in influencing metabolic control. In fact, in one of the only studies to stratify racial samples by SES, there were no differences in metabolic control across race; instead, only SES and family structure predicted metabolic control (Overstreet, Holmes, Dunlap, & Frentz, 1997). Therefore, the study design must be
replicated with equal representation of African American and European American youth at each level of SES and treatment type to determine the authenticity of these results.

In order to understand the mechanisms driving racial disparities in metabolic control, it is necessary to broaden the focus beyond demographic factors to include biological mechanisms. For example, the hemoglobin glycation Index (HGI) was developed as an alternative measure of glycated hemoglobin independent of mean blood glucose levels (Hempe, Gomez, McCarter, & Chalew, 2002). Similar to results of HbA1c alone, studies examining HGI in African American and European American populations have reported significantly higher HGI among African American adults and children with type 1 diabetes (Kamps, Hempe, & Chalew, 2010). Therefore, it is possible that African American youth are predisposed to biological differences impacting the metabolizing of glucose, resulting in different glycated hemoglobin levels.

Psychosocial factors might also play a role in the understanding of racial disparities. The current study sought to determine whether racial differences in metabolic control could be explained via depressive symptoms. Specifically, because depression presents more of a burden for African American youth than European American youth (Cummings & Druss, 2011; Sen, 2004), it was anticipated that the relationship between depressive symptoms and adherence would be stronger for African American youth. However, findings did not support race as a moderator of the depressive symptoms-adherence path within the larger model linking depressive symptoms to metabolic control. Still, many methodological issues may explain this finding and are important to consider for future research.
One probable reason for the results was the use of the CES-DC to report levels of depressive symptoms. Depressive symptoms did not differ by race, therefore, the use of this measurement when examining the depression-adherence pathway may have reduced potential effects. Instead, a measurement capturing the clinical significance of elevated depressive symptoms on daily functioning or quality of life may have found the theorized relationship between depressive symptoms and adherence. In addition, past studies examining racial differences show greater disparities in the access to and utilization of mental health services (Anderson & Mayes, 2010; Cuffe et al., 1995; Cunningham & Freiman, 1996; Flores & Tomany-Korman, 2008; Zahnner & Daslakis, 1997). Therefore, the current study would have benefited from a measure of past and present utilization of mental health services in order to capture the impact of treatment separately and in combination with elevated depressive symptoms in predicting adherence and metabolic control.

**Summary and Future Directions**

Overall, this study contributes to the literature providing insight into important factors that impact metabolic control in children and adolescents with type 1 diabetes. The current study supports the role of adherence as a mediator to the depressive symptoms-metabolic control link. This finding provides meaningful understanding to the relationships between depression, adherence, and metabolic control previously found in the literature, suggesting adherence acts a mechanism driving these associations. In addition, the results suggest the depressive symptoms-adherence pathway is not moderated by race, and therefore eliminates this pathway as responsible for the racial disparities in metabolic control. Future research should address the methodological
issues of the current study as well as use longitudinal designs to determine the causality of relationships between study variables.
## Appendix

### Table 1

**Participant Demographics, Clinic Characteristics, and Study Variables**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample (n = 86)</th>
<th>AA (n = 33)</th>
<th>EA (n = 56)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Study Outcomes</strong></td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>13.39 (3.49)</td>
<td>15.40 (10.82)</td>
<td>12.23 (9.62)</td>
</tr>
<tr>
<td>BGM frequency</td>
<td>3.75 (2.65)</td>
<td>2.60 (1.66)</td>
<td>4.47 (2.90)</td>
</tr>
<tr>
<td>Self-report adherence</td>
<td>53.80 (11.72)</td>
<td>52.84 (12.95)</td>
<td>54.30 (11.16)</td>
</tr>
<tr>
<td>Parent-report adherence</td>
<td>60.21 (11.37)</td>
<td>58.61 (12.44)</td>
<td>61.22 (10.64)</td>
</tr>
<tr>
<td><strong>Demographic and clinic</strong></td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
<td>(M) (SD)</td>
</tr>
<tr>
<td>HbA1C (%)</td>
<td>9.49 (1.76)</td>
<td>10.42 (2.01)</td>
<td>8.92 (1.25)</td>
</tr>
<tr>
<td># People in Household</td>
<td>4.17 (2.93)</td>
<td>4.33 (1.85)</td>
<td>4.10 (1.16)</td>
</tr>
<tr>
<td>Age</td>
<td>13.59 (3.49)</td>
<td>12.76 (3.64)</td>
<td>14.16 (3.32)</td>
</tr>
<tr>
<td>Gender (n, %)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>44 (51.20)</td>
<td>19 (57.60)</td>
<td>23 (43.40)</td>
</tr>
<tr>
<td>Male</td>
<td>42 (48.80)</td>
<td>14 (42.40)</td>
<td>30 (56.60)</td>
</tr>
<tr>
<td>Treatment Type (n, %)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Daily Insulin Injections</td>
<td>28 (32.60)</td>
<td>19 (57.6)</td>
<td>9 (17.00)</td>
</tr>
<tr>
<td>Multiple Daily Injections</td>
<td>21 (24.40)</td>
<td>11 (33.3)</td>
<td>10 (18.90)</td>
</tr>
<tr>
<td>Pump</td>
<td>37 (43.00)</td>
<td>3 (9.1)</td>
<td>34 (64.20)</td>
</tr>
<tr>
<td><strong>Household Characteristics</strong></td>
<td>(n) (%)</td>
<td>(n) (%)</td>
<td>(n) (%)</td>
</tr>
<tr>
<td>Maternal education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than high school</td>
<td>6 (7.00)</td>
<td>4 (12.10)</td>
<td>2 (3.80)</td>
</tr>
<tr>
<td>High school or vocational</td>
<td>26 (30.30)</td>
<td>13 (39.40)</td>
<td>13 (24.60)</td>
</tr>
<tr>
<td>Some college</td>
<td>31 (36.00)</td>
<td>13 (39.40)</td>
<td>18 (34.00)</td>
</tr>
<tr>
<td>College or graduate degree</td>
<td>21 (24.40)</td>
<td>1 (3.00)</td>
<td>20 (37.00)</td>
</tr>
<tr>
<td>Not Answered</td>
<td>2 (2.30)</td>
<td>2 (6.10)</td>
<td>---</td>
</tr>
<tr>
<td>Household Income ($)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under 10,000</td>
<td>5 (5.80)</td>
<td>5 (15.20)</td>
<td>---</td>
</tr>
<tr>
<td>10,000-14,999</td>
<td>5 (5.80)</td>
<td>4 (12.10)</td>
<td>1 (1.90)</td>
</tr>
<tr>
<td>15,000-19,999</td>
<td>4 (4.70)</td>
<td>4 (12.10)</td>
<td>---</td>
</tr>
<tr>
<td>20,000-24,999</td>
<td>6 (7.00)</td>
<td>6 (18.20)</td>
<td>---</td>
</tr>
<tr>
<td>25,000-34,999</td>
<td>10 (11.60)</td>
<td>5 (15.20)</td>
<td>5 (9.40)</td>
</tr>
<tr>
<td>35,000-49,999</td>
<td>13 (15.10)</td>
<td>4 (12.10)</td>
<td>9 (17.00)</td>
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<td>50,000-74,999</td>
<td>9 (10.50)</td>
<td>2 (6.10)</td>
<td>7 (13.20)</td>
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<td>75,000-99,999</td>
<td>9 (10.50)</td>
<td>1 (3.00)</td>
<td>8 (15.10)</td>
</tr>
<tr>
<td>100,000-149,999</td>
<td>12 (14.00)</td>
<td>---</td>
<td>12 (22.60)</td>
</tr>
<tr>
<td>Over 150,000</td>
<td>10 (11.60)</td>
<td>---</td>
<td>10 (18.90)</td>
</tr>
<tr>
<td>Not Answered</td>
<td>3 (3.50)</td>
<td>2 (6.10)</td>
<td>1 (1.90)</td>
</tr>
<tr>
<td>Insurance Status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Union or employer</td>
<td>26 (30.20)</td>
<td>5 (15.20)</td>
<td>21 (39.60)</td>
</tr>
<tr>
<td>Category</td>
<td>Count</td>
<td>BGM Frequency</td>
<td>Count</td>
</tr>
<tr>
<td>----------------------</td>
<td>-------</td>
<td>---------------</td>
<td>-------</td>
</tr>
<tr>
<td>Private</td>
<td>9</td>
<td>10.50</td>
<td>---</td>
</tr>
<tr>
<td>Government assistance</td>
<td>48</td>
<td>55.80</td>
<td>28</td>
</tr>
<tr>
<td>TRICARE/Military</td>
<td>2</td>
<td>2.30</td>
<td>---</td>
</tr>
<tr>
<td>Other</td>
<td>1</td>
<td>1.20</td>
<td>---</td>
</tr>
</tbody>
</table>

*Note. AA = African American, EA = European American. BGM Frequency = blood glucose monitoring frequency. Dashes used when no participants found for a category.*
Table 2

Descriptive Statistics and Intercorrelations between Demographic and Study Variables

<table>
<thead>
<tr>
<th>Variable (n = 86)</th>
<th>(M, SD)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. HbA1c</td>
<td>(9.49, 1.76)</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Gender</td>
<td>(0.49, 0.50)</td>
<td>0.02</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Age</td>
<td>(13.59, 3.49)</td>
<td>0.12</td>
<td>-0.10</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Treatment Type</td>
<td>(2.11, 0.87)</td>
<td>-0.35**</td>
<td>0.10</td>
<td>0.04</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. SES</td>
<td>(-.003, 0.86)</td>
<td>-0.36**</td>
<td>-0.11</td>
<td>0.05</td>
<td>0.27*</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Race</td>
<td>(0.38, 0.49)</td>
<td>0.42**</td>
<td>0.14</td>
<td>-0.19†</td>
<td>-0.54***</td>
<td>-0.50***</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Depressive Symptoms(^a)</td>
<td>(13.39, 10.13)</td>
<td>0.20†</td>
<td>0.20†</td>
<td>0.14</td>
<td>-0.01</td>
<td>-0.07</td>
<td>0.15</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>8. Adherence</td>
<td>(0.03, 0.82)</td>
<td>-0.46**</td>
<td>0.12</td>
<td>-0.46**</td>
<td>0.27*</td>
<td>0.07</td>
<td>-0.22*</td>
<td>-0.31**</td>
<td>---</td>
</tr>
</tbody>
</table>

Notes. M = mean, SD = standard deviation. For gender, 0 = male, 1 = female. For race, 0 = European American, 1 = African American. For treatment type, 1 = 2DI, 2 = MDI, 3 = pump. SES = socioeconomic status. Standardized means and standard deviations reported for SES and Adherence.

\(^a\)n = 82

† p < .10, *p < .05, **p < .01, ***p < .001.
Figure 1. Theorized model of the main study hypotheses. Adherence as a mediator of the relationship between depression and HbA1c and race as a moderator of the relationship between depression and adherence after controlling for the effects of age, treatment type, and SES on HbA1c.

Note. SES = Socioeconomic status.
Figure 2. Result of the mediation analysis testing adherence as a mediator of the effect of depressive symptoms on HbA1c. The regression coefficient between depressive symptoms and HbA1c after controlling for adherence is given in parentheses. When adherence is included, the effects of depressive symptoms on HbA1c are no longer significant, indicating mediation.

*Note. All coefficients are reported as unstandardized regression coefficients. Model does not include covariates.*

\[ \text{Depressive Symptoms} \rightarrow \text{Adherence} \rightarrow \text{HbA1c} \]

-0.03\(**\)

.04*(.01)

-.96\(***\)

\[ p < .10, **p < .01, ***p < .001 \]
Figure 3. Unstandardized estimates for the moderated mediation model, testing the indirect effect of the race by depressive symptoms interaction term as a moderator of the mediation model through the depressive symptoms-adherence pathway.

Note. For race, 0 = European American, 1 = African American. For treatment type, 1 = 2DI, 2 = MDI, 3 = pump. SES = socioeconomic status.

†p < .10, *p < .05, **p < .01, ***p < .001
List Of References


Primm, A.B., Vasquez, M. J., Mays, R. A., Sammons-Posey, D., McKnight-Eily, L. R., Presley-Cantrell, L. R.,... Perry, G. S. (2010). The role of public health in addressing racial and ethnic disparities in mental health and mental illness. *Preventing Chronic Disease, 7*(1), A20.


Biography

Brittney Jurgen was raised in Somers, Connecticut before moving to New Orleans to attend Tulane University. She first received a Bachelor of Science in psychology from Tulane University in 2013. She will graduate in 2015 with a Master of Science in psychology as a part of the 4+1 behavioral health program at Tulane University. After graduation, Brittney plans to further her passion for research before pursuing her Ph.D. in clinical psychology.