ETHNIC EFFECTS ON BIOLOGICAL, PSYCHOSOCIAL AND DISEASE CARE FACTORS IN AFRICAN-AMERICAN YOUTH WITH TYPE 1 DIABETES

Priscilla Powell
Virginia Commonwealth University

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ETHNIC EFFECTS ON BIOLOGICAL, PSYCHOSOCIAL AND DISEASE CARE FACTORS IN AFRICAN-AMERICAN YOUTH WITH TYPE 1 DIABETES

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

by

PRISCILLA WYATT POWELL
Bachelor of Arts, Washington & Lee University, 2005

Director: Clarissa S. Holmes, Ph.D.
Professor, Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
December, 2009
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Abstract

ETHNIC EFFECTS ON BIOLOGICAL, PSYCHOSOCIAL AND DISEASE CARE FACTORS IN AFRICAN-AMERICAN YOUTH WITH TYPE 1 DIABETES

By Priscilla W. Powell, B.A.

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

Virginia Commonwealth University, 2009.

Major Director: Clarissa S. Holmes, Ph.D.
Professor, Department of Psychology

Person-oriented and variable-oriented analyses were conducted to investigate sociodemographic differences in biological, psychosocial and disease care factors in youth with type 1 diabetes. Additionally, diabetes knowledge was evaluated as a potential mediator of SES effects on HbA1c and disease care. The sample included 349 youth, age 9-17 years (79.9% Caucasian, 71.3% lived with two biological parents, M SES = 46.24). Person-oriented t-tests confirmed commonly reported ethnic differences in HbA1c and disease care behaviors. However, variable-oriented analyses controlling for confounding sociodemographic influences showed most disease care effects attributed to ethnicity were better explained by SES. While diabetes knowledge was not a significant mediator of meal composition, it appeared to suppress the effect of social class on carbohydrate and fat consumption, such that more diabetes knowledge minimized the
negative effect of lower SES on optimal carbohydrate and fat consumption. Results may inform future interventions for youth at risk of poor metabolic control.
Introduction

Type 1 diabetes (T1D) is an autoimmune disease that prevents the body from producing insulin, a hormone necessary to allow sugar absorption. When insulin-producing beta cells are destroyed by the immune system, the body is no longer able to make insulin and therefore cannot absorb appropriate nutrients from food. T1D affects 1 in every 400 to 600 children and adolescents in United States (American Diabetes Association, 2008b). While prevalence rates of T1D are higher among Caucasian youth, African-American youth carry a greater disease burden in morbidity or disease complications (Chalew, Gomez, Butler, Hempe, Compton, Mercante, Rao, & Vargas, 2000; Delamater, Shaw, Applegate, Pratt, Eidson, Lancelotta, Gonzalez-Mendoza, & Richton, 1999). Current literature has established remarkable health disparities between African-American children and Caucasian children with T1D. Disparities in ethnic morbidity are evident across multiple disease domains associated with T1D including: metabolic control, acute and chronic disease complications, and mortality rates.

Metabolic Control

Metabolic control for youth with T1D is measured by glycosylated hemoglobin (HbA1c) levels. HbA1c levels provide an estimate of average blood glucose concentration during the previous three-month period. Lower HbA1c percentages indicate better metabolic control and inversely, higher HbA1c percentages indicate
poorer metabolic control. According to the American Diabetes Association (2008a), the target HbA1c level for children with T1D is less than 8.0% and less than 7.5% for adolescents with T1D.

Extant research demonstrates that African-American children and adolescents with T1D have poorer metabolic control than Caucasian children and adolescents with T1D (Auslander, Anderson, Bubb, Jung, & Santiago, 1990; Auslander, Thompson, Dreitzer, White, & Santiago, 1997; Chalew et al., 2000; Delamater, Albrecht, Postellon, & Gutai, 1991; Delamater et al., 1999; Arfken, Reno, Santiago, & Klein, 1998).

Auslander et al. (1997) reported an ethnic difference in HbA1c of 1.5%, indicating higher HbA1c levels for African-American children than Caucasian children. This difference is both statistically and clinically significant as it is associated with approximately a 53-70% higher risk of secondary disease complications in adolescents, particularly eye damage (Diabetes Control and Complications Trial Research Group, 1994). In a comparison with other minority populations, African-American children have significantly higher HbA1c levels than Hispanic children, and the average HbA1c level for both minority groups exceeds the target metabolic range for youth with T1D (Patino, Sanchez, Eidson, & Delamater, 2005). Ethnic differences in HbA1c levels persist through adulthood as detected by a meta-analysis of 78 studies comparing African-American and Caucasian adults with T1D (Kirk, D’Agostino, Bell, Passmore, Bonds, Karter, & Narayan, 2006). If better metabolic control leads to fewer acute and chronic health issues (Silverstein, Klingensmith, Copeland, Plotnick, Kaufman, Laffel, Deeb, Grey, Anderson, Holzmeister, & Clark, 2005; Epidemiology of Diabetes Interventions
and Complications Study, 2003), then it is no surprise that poorer metabolic control in African-American children is related to poorer acute and chronic diabetes complications.

**Disease Complications**

The most common acute complication of T1D is diabetic ketoacidosis, or DKA. DKA occurs secondary to relative insulin depletion which leads to an accumulation of glucose in the bloodstream. Increases in blood glucose levels signify the biounavailability of glucose at a cellular level such that the body must utilize stored fat nutrients, causing acidic ketones to accumulate in the bloodstream. African-American children are hospitalized more frequently for DKA than Caucasian children (Delamater et al., 1991). Additionally, African-American youth are at higher risk for readmission to the hospital after poorer treatment adherence following an initial episode of DKA than Caucasian youth (Glasgow, Weissburg-Benchell, & Tynan, 1991).

Chronic complications of T1D include retinopathy (damage to the retina), neuropathy (nerve damage), nephropathy (kidney damage), and angiopathy (damage to the blood vessels) (Silverstein et al., 2005). African-American children with T1D also are more likely to experience long-term diabetes complications during adulthood than Caucasian children (Chalew et al., 2000). The prevalence rates of retinopathy (Arfken et al., 1998) and nephropathy (Cowie, 1993), are higher among African-American adults relative to Caucasian adults and are secondary to poorer glycemic control. Increased morbidity rates of African-American adults with T1D may be due to biological factors (Arfken et al., 1998) as well as the indirect influence of poorer diabetes care behaviors (Cowie, Port, Wolfe, Savage, Moll, & Hawthorne, 1989).
The higher mortality rates of African-Americans with T1D further demonstrate the pervasive health disparities of this disease. A 20-year follow-up of adults in an epidemiological study by Laporte, Tajima, Dorman, Cruickshanks, Eberhardt, Rabin, Atchison, Wagener, Becker, Orchard, Songer, Slemenda, Kuller, and Drash (1986) revealed that 26% of the sample’s African-American participants had died, versus 11% of the sample’s Caucasian participants (as cited in Auslander et al., 1997). In another retrospective study, African-American youth were nine times more likely to die from diabetes-related complications than Caucasian youth ages 1 to 24 years-old (Lipton, Good, Mikhailov, Freels, & Donoghue, 1999).

The Diabetes Control and Complications Trial (DCCT) conclusively demonstrates that diabetes complications result from poorer metabolic control (1993). Considering the remarkable ethnic disparity in metabolic control, some researchers conclude that African-American youth may have a biological predisposition to higher HbA1c levels than other ethnic groups. In fact, the 3rd National Health and Nutrition Examination Survey reports ethnic discrepancies in HbA1c levels among healthy populations. Healthy African-American youth had higher HbA1c levels than Caucasian or Hispanic youth regardless of age, sex, body mass index (BMI) and education level (Saaddine, Fagot-Campagna, Rolka, Narayan, Geiss, Eberhardt, & Flegal, 2002).

Ethnic differences in metabolic control also may be attributed to non-biological factors such as poorer diabetes care behaviors, as well as sociodemographic and psychosocial factors. Socioeconomic Status (SES) and parental marital status are two sociodemographic factors often confounded with ethnicity, yet independently relate to
metabolic control and disease care behaviors. Psychosocial factors to consider are the mediating effects of diabetes knowledge, family conflict and cohesion, and parental monitoring in conjunction with distribution of responsibility for diabetes management.

*Diabetes Care Behaviors*

Diabetes care behaviors are thought to have a relation to both acute and chronic health complications as mediated by poorer metabolic control. The concept of disease management is defined as “an active, daily, and flexible process in which youth and their parents share responsibility and decision-making for achieving disease control, health, and well-being through a wide range of illness-related activities” (Schilling, Grey, & Knafl, 2002a, pp 92). Specific disease care behaviors for youth with T1D include: insulin administration, blood glucose checks, nutritional guidelines, and physical activity.

*Insulin regimen.* Youth with T1D are unable to produce insulin independently and require daily insulin injections or an insulin pump, a continuous subcutaneous insulin infusion. Insulin regimens are categorized by the number of insulin injections administered per day. A conventional treatment regimen for children with T1D consists of 1 or 2 daily insulin injections. Youth prescribed an intensive treatment regimen administer 3 or more insulin injections per day or rely on an insulin pump which supplies a continuous basal of insulin along with prescribed boluses. The DCCT examined the difference in effectiveness between conventional and intensive treatment regimens in relation to metabolic control and diabetes complications (1994). DCCT participants receiving intensive treatment had an average HbA1c level of 8.06%, which was lower than the conventional treatment group participants who had HbA1c levels of 9.76%. The
6.5 year follow-up of the DCCT concluded that intensive treatment prevents the onset and delays the progression of diabetes complications as compared to conventional treatment. Specifically, the onset of retinopathy was reduced by 53% and progression of this complication was delayed by 70% (DCCT, 1994).

*Insulin regimens of African-Americans.* When reporting sample characteristics, few investigators indicate ethnic differences in prescription rates for intensive versus conventional insulin regimens. In a study of quality of life related to insulin regimens in youth with T1D, Valenzuela, Patino, McCullough, Ring, Sanchez, Eidson, Nemery, and Delamater (2006) revealed significant ethnic differences in prescribed treatment regimens of injections versus an insulin pump. Of the African-Americans in this sample, 89% were on insulin injections and 11% were on an insulin pump, whereas 64% of Caucasian sample injected insulin and 36% received insulin via an insulin pump. In fact, Caucasian adolescents were more likely to be prescribed insulin pump therapy than all minority groups. Importantly, Valenzuela et al. (2006) found minority groups differed unfavorably from Caucasians in parents’ education level, disease duration, and HbA1c such that social class or disease characteristics may account for at least some of the ethnic disparities in treatment regimens.

Beyond a possible direct association between social class and poorer health status, Delamater et al. (1999) found that African-American youth had 4 times the risk of poorer metabolic control than Caucasian youth, even though no ethnic differences existed in conventional versus intensive treatment regimens. The authors noted the demographic composition of their sample differed from the DCCT sample and that intensive treatment
regimens were prescribed less frequently than during the DCCT (Delamater et al., 1999). Delamater et al. (1999) described their sample as low SES, classified by a participant’s health insurance status: 13% of African-American participants versus 66% of Caucasian participants held private health insurance. Their assessment of SES did not account for potential disparities in parental educational levels, which may relate to an indirect effect of their SES proxy on health status via a poorer understanding of daily disease management issues.

According to the DCCT findings, insulin regimen is the single most important disease management factor that explains differences in HbA1c levels (1993). Access to intensive or optimal treatment is a classic disparity in medical treatment for African-Americans who may be less likely to be prescribed optimal disease management strategies (Institute of Health, 2003; Elster, Jarosik, VanGeest, & Fleming, 2003). This situation could result from either direct or indirect SES effects, (i.e., via limited access to regular medical care secondary to life hardships) or more indirect effects related to parents’ educational achievement or both. If African-American youth with T1D do not have access to the single most important disease management factor, then unfortunately this population is likely to be in poorer metabolic control and to have a higher prevalence of acute and chronic disease complications.

**Blood glucose monitoring.** The ADA recommends that children and adolescents with T1D test blood glucose levels four or more times per day (ADA, 2008a; Silverstein et al., 2005). Extant research demonstrates a strong correlation between monitoring frequency and overall metabolic control (Anderson, Ho, Brackett, Finkelstein, & Laffel,
Children who test their blood glucose levels more often are likely to have lower HbA1c levels than children who test infrequently (Holmes, Chen, Streisand, Marschall, Souter, Swift, & Peterson, 2006; Swift, Chen, Hershberger, & Holmes, 2006; Stewart, Emslie, Klein, Haus, & White, 2005; Hanson, De Guire, Schinkel, Kolterman, Goodman, & Buckingham, 1996; Johnson, Kelly, Henretta, Cunningham, Tomer, & Silverstein, 1992).

Blood glucose monitoring of African-Americans. Auslander et al. (1997) found that mothers of African-American children with T1D reported lower levels of adherence to blood glucose testing and diet regimen than mothers of Caucasian children with T1D. Adherence was measured through a semi-structured interview with participant mothers. The interview consisted of adherence questions related to recommended testing frequency for blood glucose and urine, recommended diet, and overall T1D adherence. However, Auslander et al. (1997) do not provide specific questions nor do they report the time-frame for the adherence measure. They report sociodemographic disparities between the African-American and Caucasian participants in their sample. Specifically, African-American mothers reported lower education levels and lower SES than Caucasian mothers. Also, African-American participants were more likely to live in single-parent families and receive public health insurance than Caucasian participants (Auslander et al., 1997). Auslander et al. (1997) indicate that parental marital status, SES, and education level significantly contribute to the ethnic differences in metabolic control and disease care behaviors such as blood glucose monitoring.
Patino et al. (2005) reported significant rates of “nonadherence” among a lower-middle SES, minority sample comprised of African-American and Hispanic children with T1D. Conclusions were based on the Self Care Inventory (SCI); a 14 item parent- and child-report questionnaire of adherence behavior over a one month period. Adherence behavior was measured on a 5-point Likert scale ranging from complete nonadherence to complete adherence. Almost 75% of their sample reported nonadherence to blood glucose monitoring in the past month (Patino et al., 2005). A second study of a mixed-minority sample examined the effects of T1D in urban youth through a global structured interview (Lipton, Drum, Burnet, Mencarini, Cooper, & Rich, 2003). Ten percent of the African-American participants reported checking their blood glucose level less than once a day. Nonadherence rates of African-American participants were higher than other ethnic groups as 8% of Hispanic youth and 0% of non-Hispanic white participants reported less than one blood glucose check per day (Lipton et al., 2003).

**Nutrition.** Nutrition recommendations are based on guidelines “for all healthy children” rather than disease specific requirements. However, research indicates that children and adolescents with T1D adhere to published general nutritional guidelines more closely than children without T1D (Franz, Bantle, Beebe, Brunzell, Chiasson, Garg, Holzmeister, Hoogwerf, Mayer-Davis, Mooradian, Purnell, & Wheeler, 2002). The Dietary Reference Intakes (DRIs) published by the Institute of Medicine’s Food and Nutrition Board recommends that for children ages 4-18 years old, 45-65% of calories consumed per day should come from carbohydrates and 25-35% of daily calories should come from fats (American Dietetic Association, 2004). In addition to dietary
composition, eating frequency is an important health factor for youth with T1D. Smaller, more frequent meals and snacks are recommended for youth with T1D, up to six meals or snacks per day (Johnson, Silverstein, Rosenbloom, Carter, & Cunningham, 1986; Freund, Johnson, Silverstein, & Thomas, 1991).

**Nutrition of African-Americans.** There are no available studies that measure dietary composition or meal content of carbohydrate and fat consumption among T1D samples of minority youth. However, Lowry, Kann, Colin, and Kolbe, (1996) reported that healthy African-American youth consume more foods high in fat and eat fewer complex carbohydrates, consisting of fruits and vegetables, than healthy Caucasian youth. Among a sample of 6,321 healthy youth ages 12 - 17 years-old, 89.3% of African-Americans versus 85.5% of Caucasians reported eating less than 5 fruits and vegetables during the previous day. Additionally, 38.1% African-Americans versus 33.2% Caucasians reported eating more than 2 high-fat foods the during previous day, such as french fries, cookies or doughnuts (Lowry et al., 1996). It is important to note these ethnic differences were not detected when Lowry et al. (1996) controlled for SES and parental education level. These confounded sociodemographic factors will be discussed further in the confounding variables section of this paper.

Cullen, Baranowski, Owens, de Moor, Rittenberry, Olvera, and Resnicow (2002) reported healthy Caucasian students grades 4 - 6 consume more fruits and vegetables than African-American students. Specifically, healthy Caucasian children ate 0.32 more servings of fruit and 0.25 more servings of vegetables than healthy African American children (Cullen, Baranowski, Rittenberry, Cosart, Owens, Hebert, & de Moor, 2000, as
cited in Cullen et al., 2002). Given that so little is known about ethnic differences, Cullen et al. (2002) did a follow-up study about ethnic differences in peer normative beliefs about nutrition. African-American children perceived their same ethnicity peers felt more positively about eating fruits and vegetables than Caucasian youth. However, African-American children also reported more permissive parental attitudes toward food consumption. African-American youths felt their parents let them eat what they chose more so than Caucasian children which may explain ethnic differences in fruit and vegetable consumption (Cullen et al., 2002). As previously noted, children and adolescents with T1D generally adhere more closely to nutritional recommendations compared to their healthy counterparts (Franz et al., 2002). Nevertheless, similar ethnic differences in dietary consumption may exist within the T1D population as in the healthy population.

*Exercise.* While exercise guidelines are not designed specifically for youth with T1D, the ADA concurs with the frequency and duration recommendations of the American Academy of Sports Medicine and the Center for Disease Control that youth should participate in a minimum of 30 to 60 minutes of moderate physical activity each day (Silverstein et al., 2005; ADA, 2008a). Similar to trends in nutrition, youth with T1D engage in more physical activity than their healthy counterparts (Raile, Kapellen, Schweiger, Hunkert, Nietzschmann, Dost, & Kiess, 1999).

Recent studies have reported mixed results regarding the relation between exercise and metabolic control (Silverstein et al., 2005, Raile et al., 1999; Stewart et al., 2005; & Streisand, Repess, Overstreet, de Pijem, Chen, & Holmes, 2002). Streisand et
al. (2002) reported an association between exercise behaviors and metabolic control in a sample of Puerto Rican youth with T1D. Specifically, longer exercise duration and more frequent exercise was associated with lower HbA1c levels in a primarily lower income sample, as measured by the 24-hour Diabetes Care Interview (Streisand et al., 2002). However, Hanson et al. (1996) utilized a different methodology to measure exercise and did not find a consistent association between exercise and HbA1c levels. They used two methods to assess exercise frequency and intensity; a 7-day physical activity interview and a 3-day physical activity log completed by both parent and child. Hanson et al. (1996) reported that exercise was not related to metabolic control, even when accounting for varying degrees of intensity and activity type. Stewart et al. (2005) also failed to find a relation between exercise and HbA1c in child and parent reports on a global rating scale from 20-100. Stewart et al. explain, “The behaviors most closely related to HbA1c levels were those that are most proximal in influencing glucose metabolism” (2005, pp 246). Proximal factors of diabetes self-care include blood glucose testing and insulin injections, whereas physical activity and adherence to nutrition plans act as distal influences of metabolic control. While exercise may be beneficial in preventing long-term health complications for youth with T1D, the benefits of exercise may not translate into immediate improvements in metabolic control (Stewart et al., 2005). Additional blood glucose testing also may be necessary before and after physical activity in addition to increased meal frequency, as snacks are often necessary to prevent or treat low blood glucose levels due to exercise (Silverstein et al., 2005).
Exercise patterns of African-Americans. Patino et al. (2005) report a 36% adherence rate to exercise recommendations among a minority sample of African-American and Hispanic youth with T1D based on parent and child-reports of adherence. Lowry et al. (1996) report African-American without T1D adolescents are more sedentary than their Caucasian peers without T1D. Specifically, 40.6% of African-Americans versus 33.4% of Caucasian youth reported exercising less than 3 times during the previous 7 days. Similarly, the 3rd National Health and Nutrition Examination Survey found that African-American and Hispanic females reported lower exercise frequency than Caucasian females or males from all ethnic groups (Andersen, Crepso, Bartlett, Cheskin, & Pratt, 1998). While these findings from healthy youth must be generalized with caution, they suggest that ethnic differences may be found in exercise patterns.

Confounding Sociodemographic Variables

Evidence suggests some ethnic health disparities may be due to differences in socioeconomic status (SES) and parental marital status, variables that often are not measured in studies that evaluate ethnic health differences (Overstreet, Holmes, Dunlab, & Frentz, 1997a; Swift et al., 2006). In most early studies, these three factors are confounded, and therefore ethnicity may not be a significant factor in metabolic control or diabetes care behaviors. However, recent studies better account for potentially confounded variables. For example, Harris, Greco, Wysocki, Elder-Danda, and White (1999) found that African-American youth raised in single-parent families have higher HbA1c levels than African-American youth from two-parent families or than Caucasian youth from both single- and two-parent families.
Swift et al. (2006) attempted to disentangle the statistical overlap of SES, ethnicity and parent marital status through a complex series of path analyses and structural equation models. Results indicated that SES and parental marital status are each significantly related to metabolic control of children with T1D. Children of two-parent families had lower HbA1c levels than children of single-parent or blended families (Swift et al., 2006). Children from two-parent families also reported higher adherence rates to several diabetes care behaviors such as more frequent meals and more blood glucose tests than children from single-parent or blended families. Swift et al. (2006) suggests that ethnicity affects metabolic control through specific diabetes care behaviors as indicated by the relationship with exercise duration and blood glucose monitoring frequency. Specifically, African-American youth reported briefer exercise duration and less frequent blood glucose monitoring than Caucasian youth (Swift et al., 2006).

While it is informative to parse the effects of ethnicity, SES and parental marital status, census data indicates that these three factors frequently overlap significantly in the population. Single-mother families are five times more likely to have lower SES (i.e., poverty level) than two-parent families (Lugaila & Overturt, 2004). Specifically, 39% of single-mother families versus 8% of two-parent families live at or below the poverty line (Lugaila & Overturt, 2004). In the general population, 23% of all children live in single-mother households (Federal Interagency Forum on Child and Family Statistics, 2007). Almost two-thirds of African-American children live with a single-parent or alternate family arrangement as only 35% of African-American children live with two parents (Federal Interagency Forum on Child and Family Statistics, 2007). In essence, an
African-American child with T1D is more likely to live in a low SES, single-mother household than any other combination of demographic characteristics.

This overlap in SES and parental marital status are reflected in current diabetes literature. Thompson, Auslander, and White (2001) found that children with T1D who live with single-mothers are in poorer metabolic control than those who live in two-parent families. Single-mothers reported that their children had poorer regimen adherence than mothers of two-parent families, as defined by a summary score on the Adherence and IDDM Questionnaire-R; a 15-item structured interview containing questions about how well a child adheres to prescribed medical regimens concerning blood glucose monitoring, urine testing, and treatment of hypoglycemia (Thomson et al., 2001). However, 67% of the sample of single-mother families was comprised of African-American families whereas only 17% of the two-parent families were African-American (Thompson et al., 2001). While it is not appropriate to conclude that African-American children with T1D have poorer metabolic control and adherence than Caucasian children with T1D based on these findings alone it is evident that these confounding variables must be considered simultaneously, just as they often present together in the population.

*Diabetes Knowledge and Disease Care Behaviors*

Mixed evidence exists regarding the role of diabetes knowledge in relation to metabolic control and disease care adherence. In general, greater diabetes knowledge is associated with better adherence to disease care behaviors while poorer diabetes knowledge is associated with poorer adherence to disease care behaviors (Holmes et al.,
For example, youth with greater disease knowledge exhibit higher levels of self-efficacy, which is in turn related to better nutritional care behaviors (i.e., a higher percentage of carbohydrates and smaller percentage of fats in daily calorie consumption) (Holmes et al., 2006). Chisholm, Atkinson, Donaldson, Noyes, Payne, and Kelnar (2007) found that greater maternal diabetes knowledge was associated with lower HbA1c levels and greater adherence to diabetes care behaviors for young children, age 2 - 8 years old. Specifically, greater maternal diabetes knowledge related to more frequent blood glucose monitoring and a lower percentage of calories from “extrinsic sugars”, as recommended by the guidelines from the International Society for Paediatric and Adolescent Diabetes (Chisholm et al., 2007). Maternal diabetes knowledge was measured with a 53-item Diabetes Knowledge Questionnaire (DKQ) and individual disease care behaviors were assessed with the 24-hr Diabetes Care Interview. La Greca, Follansbee, & Skyler (1990) reported similar findings. Mothers of younger children with greater diabetes knowledge had children with lower HbA1c levels. However, youth knowledge, rather than maternal knowledge, was related to metabolic control for adolescents with T1D (La Greca et al., 1990b). This finding provides a glimpse of the redistribution of diabetes responsibilities that occurs during adolescence, a factor discussed in the following section on responsibility and diabetes care behaviors.

Stallwood (2006) reported that more parent diabetes knowledge is associated with lower HbA1c levels, greater financial income, and marital status. Specifically, currently married caregivers scored higher on the Michigan Diabetes Research and Training Center Diabetes Knowledge Test (MDRTC) than unmarried caregivers (i.e., divorced or never
married) (Stallwood, 2006). However, there was no association between marital status and metabolic control. Caregivers of children with HbA1c levels between 7% and 9% had more diabetes knowledge and reported higher income levels than caregivers of children with HbA1c levels above 9%. According to Stallwood (2006), these associations suggest that lower SES caregivers may not benefit fully from diabetes information provided by their child’s medical team. Essentially, lower SES parents may have difficulty comprehending diabetes related materials and thus their diabetes knowledge may not increase. This factor that has been shown to greatly influence children’s metabolic control, as well as a caregiver’s ability to implement diabetes knowledge when assisting with disease care behaviors (Stallwood, 2006).

**Responsibility and Disease Care Behaviors**

When youth with T1D are given greater responsibility for their disease management, they are likely to adhere less with their diabetes care behaviors. Inversely, youth with T1D whose parents maintain more diabetes responsibility adhere more with disease care behaviors and have better metabolic control (Palmer, Berg, Wiebe, Beveridge, Korbel, Upchurch, Swinyard, Lindsay, & Donaldson, 2004; La Greca et al., 1990; Holmes et al., 2006). Holmes et al. (2006) reported that higher youth responsibility was associated with lower frequency and shorter exercise duration, fewer daily blood glucose tests, and less frequent meals. This discrepancy in disease care also was reflected in metabolic control as children with greater responsibility had poorer metabolic control indicated by higher HbA1c levels than children who had less disease responsibility (Holmes et al., 2006). However, youth responsibility was not associated with nutrition
measures including percentage of calories from carbohydrates and fats (Holmes et al., 2006).

Helgeson, Reynolds, Siminerio, Escobar, and Becker (2008) also assessed diabetes responsibility with the Diabetes Family Responsibility Questionnaire (DFRQ). Consistent with the findings reported by Holmes et al. (2006), youth who reported greater self responsibility demonstrated declines in metabolic control. When parents and youth perceived shared responsibility, youth adhered better with diabetes care behaviors and had better metabolic control, especially older adolescents (Helgeson et al., 2008). However, youth who reported greater parent responsibility demonstrated similar self-care behaviors as youth with parents who reported shared responsibility (Helgeson et al., 2008). This finding may suggest that parental and shared responsibility are related to similar outcomes in metabolic control, or alternately, children may over-report a parent’s responsibility in their daily disease care.

**Parental Monitoring and Disease Care Behaviors**

Previous research has established that higher parental involvement is associated with greater adherence to disease care behaviors and is indirectly associated with better metabolic control via regimen adherence (Wiebe, Berg, Korbel, Palmer, Beveridge, Upchurch, Lindsay, Swinyard, & Donaldson, 2005). Similar to the trends reported in parental responsibility, when parents are uninvolved in diabetes management, youth are at risk for poorer adherence and higher HbA1c levels than when parents remain involved in disease care. Weibe et al. (2005) classified parental involvement into three categories: uninvolved, controlling and collaborative. They found that children with uninvolved
parents had poorer adherence to diabetes care behaviors, whereas children with collaborative parents had both better adherence and better metabolic control (Weibe et al., 2005). Older children of controlling parents reported poorer diabetes care behaviors, yet this was not reported in younger children of controlling parents.

Parental monitoring is one aspect of parental involvement which refers to direct supervision of diabetes care behaviors. Higher levels of diabetes-specific monitoring are associated with greater adherence to diabetes care behaviors (Ellis, Podolski, Frey, Naar-King, Wang, & Moltz, 2007). Ellis, Templin, Podolski, Frey, Naar-King, and Moltz (2008) recently developed the Parental Monitoring of Diabetes Care Scale (PMDC), a 19-item parent-report measure of parental monitoring of daily diabetes care. They report a strong association between parental monitoring and adherence to diabetes care behaviors, which was indirectly associated with metabolic control via regimen adherence. Specifically, higher parental monitoring related to better adherence and lower HbA1c levels. Single- and married- parents reported equivalent levels of diabetes-specific monitoring, yet parents of minority youth reported lower levels of diabetes-specific monitoring than parents of Caucasian youth (Ellis et al., 2008). Ellis et al. (2008) suggest that this ethnic difference may be due to “differences in beliefs in the value of early emancipation of adolescents” (pp 152). Chilcoat, Breslau, and Anthony (1996) found that single-mothers of youth with T1D reported lower levels of general child monitoring than married mothers, as measured by a 10-item telephone interview addressing a variety of parenting behaviors such as rule establishment, supervision, and knowledge of a child’s whereabouts. Together, these results indicate that sociodemographic factors such
as parent marital status and ethnicity may differentially influence general parental monitoring in comparison to diabetes-specific monitoring.

*Family Environment and Disease Care Behaviors*

Family environment may influence a family’s ability to work together to help youth adhere to diabetes care and achieve good metabolic control. A cohesive family environment is characterized by better communication and higher levels of commitment to help and support other family members. Families high in conflict express anger more frequently, demonstrate lower levels of cooperation and provide less emotional support (Swift et al., 2006). Lower conflict and higher cohesion are each related to better disease care and metabolic control. Inversely, higher family conflict and lower cohesion are associated with poorer adherence to diabetes regimen and higher HbA1c levels (Swift et al., 2006; Jacobson, Hauser, Lavori, Willett, Cole, Wolfsdorf, Sumont, & Wertlieb, 1994; Overstreet, Goins, Chen, Holmes, Greer, Dunlap, & Frentz, 1995; Auslander et al., 1997).

Evidence suggests that parental marital status may mediate the relation between family environment, disease care behavior, and metabolic control. For example, youth from single-parent families reported lower levels of cohesion and poorer metabolic control than youth from two-parent families (Overstreet et al., 1995). Swift et al. (2006) found that under similar family environments of low conflict and high cohesion, youth from two-parent biological families are in better metabolic control than youth from single-parent families. However, two-parent marital status is no longer a protective factor for unfavorable family environments of high conflict and low cohesion. These youth have similar HbA1c levels as those from single-parent families, suggesting that
parent marital status and family environment act independently on health outcomes (Swift et al., 2006).

As previously described, Harris et al. (1999) found that African-American youth from single-parent families had higher HbA1c levels than Caucasian youth from single-parent families and both African-American and Caucasian youth from two-parent families. However, they did not detect ethnic differences in diabetes care or in adolescent-reported diabetes conflict, as measured by a 15-item youth report questionnaire which assessed conflict relating to diabetes tasks during the previous month. Mother-reported diabetes conflict did not differ by ethnicity, yet single mothers reported higher levels of diabetes-related conflict than mothers of two-parent families. Harris et al. (1999) suggest that ethnicity and parental marital status together mediate the relation between family environment and metabolic control although marital status and ethnicity variables are confounded in this study such that clear conclusions cannot be drawn about the effects of one versus the other.

Psychosocial factors may also mediate the relations between family environment, adherence to diabetes care, and metabolic control. For example, favorable family environments of low conflict and high cohesion related directly to greater maternal diabetes knowledge which then related to better disease care adherence and metabolic control (Chisholm et al., 2007). Contrary to their hypothesis, Holmes et al. (2006) did not detect a relation between optimal environmental factors and diabetes care behaviors or metabolic control via self-efficacy. However, they concluded that environmental factors remain pertinent to successful intervention based on established relations between
youth behavioral problems and family environments of high conflict and low cohesion (Holmes et al., 2006).

**Statement of the Problem**

Previous studies have examined differences in demographic factors and differences in metabolic control, but have not examined differences in actual diabetes care behaviors between African-American and Caucasian children with T1D. Essentially, prior studies report relations among demographic independent variables on a *distal* biomedical dependent variable, primarily metabolic control. By neglecting to account for relations associated with proximal or *immediate* disease care dependent variables, several fundamental questions remain unanswered. Do African-American youth and their parents manage diabetes differently than Caucasian youth? If so, how? Is it possible to link potential differences in disease care to different outcomes in a primary biomedical outcome of HbA1c levels?

A major reason for this gap in the literature is the difficult and time-consuming process of accurately measuring diabetes care behaviors, as well as the difficulty for any one study site to obtain a large enough sample of minority youth. While many researchers acknowledge the importance and necessity of this information, very few experimenters have pursued this line of research. Or they have done so through global disease care measures, which are not designed to assess actual disease care behaviors, but global impressions of disease management. The present study attempts to measure the correlates of ethnicity as a factor related to diabetes care behaviors.
A unique factor of this study is the ethnic and SES diversity of the participant sample. Research that has included a substantial minority sample often compares low-income African-American with upper-middle class Caucasian youth, which is a limiting factor in current literature. The sample of the proposed study is comprised of participant groups of African-American and Caucasian children with T1D, which are more comparable on SES and parent marital status in comparison to highly discrepant samples from existing research.

In addition to providing a clearer picture of group differences in disease care behaviors, the present study proposes an additional contribution to the literature by evaluating some of the substrates or mediators that might underlie ethnic differences that remain after statistical treatment of covariates. Ethnic differences in disease knowledge, responsibility, parental monitoring, and family environment potentially are the proximal processes by which more distal factors like single-parent status and lower SES operate on children’s disease care. If disease management behaviors differ by ethnicity, is this discrepancy related to different levels of parent and child knowledge about diabetes? Alternately, is this discrepancy the result of different levels of youth responsibility and parental monitoring in diabetes care behaviors? Or do family environment factors such as conflict and cohesion mediate disease care? Finally, of the broad array of disease care variables which are examined, which are most important in predicting the metabolic control of African-American versus Caucasian youth?


**Hypotheses**

Biomedical and demographic hypotheses:

1) There may be ethnic differences in HbA1c, SES distribution and parental marital status.

Diabetes-care hypotheses:

2) There may be ethnic differences in insulin regimens, blood glucose monitoring frequency, nutritional intake, and exercise frequency.

Psychosocial hypotheses:

3) There may be ethnic differences in diabetes knowledge, disease responsibility, parental monitoring, and family conflict and cohesion.

Mediational hypotheses:

4) Potential ethnic differences in psychosocial factors will be evaluated as potential mediators in disease care behaviors and HbA1c.

![Proposed Psychosocial Mediation Model](image)

*Figure 1. Proposed Psychosocial Mediation Model for the Relationship between Ethnicity and Disease Care Factors.*
Method

Participants

Participants included 349 children age 9 to 17 years old, and a parent from two different data bases. Children were patients at one of two pediatric endocrinology clinics located within metropolitan children’s hospitals in Richmond, Virginia and Washington, DC. Inclusion criteria required that children were diagnosed with T1D and without any other major chronic illness or injury.

Procedure

Potential participants and their families received a recruitment letter for one of two studies. The first was an evaluation of memory and learning skills on disease management; the second was a randomized clinical trial (RCT) designed to prevent deterioration in youth disease care behaviors. Only descriptive baseline data were utilized from the second study. After receiving a letter, parents were contacted by phone and invited to participate. For those families who agreed to participate, assessments were scheduled in conjunction with a child’s upcoming medical appointment. After obtaining written informed parental consent and youth assent, a trained research assistant interviewed each parent and child separately in a clinic exam room and then administered the test battery or distributed questionnaire packets to both parents and children. Upon completion of the study tasks, each child participant received $25 for their participation.
Measures

Demographic information. Participant demographic information was obtained through questionnaires completed by a parent who accompanied each participant to a medical appointment. Information was obtained about ethnicity, parental marital status, age of disease onset, disease duration, and socioeconomic status.

Metabolic control. Metabolic control was measured by glycosylated hemoglobin (HbA1c) levels at time of a youth’s medical appointment. HbA1c provides an estimate of average blood glucose concentration for the previous three-month period. According to the American Diabetes Association (2008a), recommended HbA1c levels for children with T1D are < 8.0% and for adolescents < 7.5%. Poorer metabolic control is indicated by higher HbA1c levels. HbA1c levels were obtained by reviewing each participant’s medical chart.

Diabetes care behaviors. Diabetes care behaviors were measured with the 24-hour Diabetes Care Interview (Holmes et al., 2006; Johnson et al., 1986). During individual interviews, research assistants asked parents and children to report all diabetes-relevant behaviors from the previous 24-hour period in temporal order including each instance of blood glucose testing, insulin injection or bolus, nutritional intake and physical activity. In the event that a parent or child omitted diabetes-care information, research assistants were instructed to prompt with specific, non-judgmental questions to obtain the most complete and accurate report possible. Data reported by each parent and child was analyzed according to a specific set of decision rules (Johnson et al., 1986), yielding a combined profile of seven quantifiable disease care behaviors: (1) frequency of
blood glucose monitoring (2) meal/snack frequency (3) percentage of daily calories from fats (4) and carbohydrates, (5) exercise duration (6) exercise frequency, and (7) insulin regimen.

Previous research has established that the 24-hour Diabetes Care Interview provides a reliable and valid report of disease care behaviors. Though decisions rules assist researchers in resolving discrepancies between parent and child reports, the significant Person product-moment correlations (p < .0001) for each measured variable suggests acceptable levels of agreement among parent-child dyads (Johnson et al., 1986). The test-retest reliability over a three-month interval varies by diabetes care behavior (i.e., Blood glucose monitoring, \( r = .72 \) to \( .76 \); Diet behaviors, \( r = .45 \) to \( .77 \); Exercise behaviors, \( r = .37 \)), indicating generally appropriate temporal stability (Freund et al., 1991).

*Diabetes knowledge.* The Test of Diabetes Knowledge (TDK) was used to assess parent and child diabetes knowledge (Johnson, Pollak, Silverstein, Rosenbloom, Spillar, & McCallum, 1982) in the Study 1 sample database. The TDK is not available in the Study 2 sample. The TDK consists of 75 multiple-choice questions relating to several factors of diabetes knowledge and disease care behaviors. Thirty-nine items constitute the diabetes Problem-Solving subscale and 36 items comprise the General Diabetes Information subscale. The TDK has established appropriate levels of internal consistency for both the General Information (\( \alpha = .71 \)) and Problem Solving subscales (\( \alpha = .80 \)) (Johnson et al., 1992).
Responsibility for diabetes management. Parent and child report of diabetes responsibility was measured by the Diabetes Family Responsibility Questionnaire (DFRQ; Anderson, Auslander, Jung, Miller, & Santiago, 1990). The DFRQ consists of 19 items relating to responsibility for specific diabetes care behaviors. Parents and children indicate their perceived level of responsibility for each task on a 3-point Likert scale. Response choices for each item include: “parent takes or initiates responsibility for this almost all of the time”, “parent and child share responsibility for this about equally”, “child takes or initiates responsibility almost all of the time”. Each item contributes to 1 of 4 different subscales: General, Communication, Insulin Frequency, and Hypoglycemia. Scores range from 19 - 57, with a higher score indicating greater youth responsibility. Recent studies with the DFRQ have established concurrent and construct validity with a family environment measurement (Streisand, Swift, Wickmark, Chen, & Holmes, 2005). Appropriate levels of internal consistency have been established for the original 17-item version of the DFRQ (.69 - .85; Auslander et al., 1990).

Parental monitoring in diabetes care. The Parent Monitoring of Diabetes Scale was used to measure parental monitoring of children’s daily diabetes care behaviors in the second participant sample (PMDS; Ellis et al., 2008; Ellis et al., 2007). The PMDS is comprised of 19 items; 18 items are presented on a 5-point Likert scale and 1 open-ended item asks the parent to indicate additional diabetes-care tasks he or she routinely monitors. The most recent set of subscales established by Ellis et al. (2008) includes: Supervision of the availability of medical supplies/devices, Monitoring of blood glucose testing, Oversight of diet, Monitoring of nonadherence, and parent report of Direct
oversight of diabetes management behaviors. Several items require reverse scoring and total scores range from 18 - 90, with higher scores indicating higher levels of parental monitoring. The PMDS has established adequate internal consistency ($\alpha = .81$) and good temporal stability over a 2-week interval (ICC = .80).

Family environment. The Conflict and Cohesion subscales of the Family Environment Scale (FES) were used to measure perceived levels of conflict and support in the home environment (Moos & Moos, 2002). The FES contains 18 items and participants indicate whether statements are true or false regarding their perceived family environment. Each subscale is comprised of 9 different items and subscale scores range from 0-9, with higher scores indicating higher levels of conflict or cohesion. The FES has demonstrated sufficient levels of internal consistency for both subscales: Conflict ($\alpha = .75$), and Cohesion ($\alpha = .78$) (Moos & Moos, 2002). This measure also has adequate temporal stability as indicated by test-retest reliability at 2 and 4 month intervals for both subscale: Conflict (.85, .66), and Cohesion (.86, .72).

Data Analysis Plan

Descriptive statistics and univariate analyses were performed on all hypothesized variables of interest. Pearson’s correlation coefficients evaluated relations between sociodemographic, disease care, and psychosocial variables and results were used to inform subsequent analyses. Independent samples $t$-tests and chi-square tests for independence were conducted, consistent with the literature, to detect unique ethnic group differences in biomedical and demographic factors, disease care behaviors, and
psychosocial correlates of diabetes knowledge, responsibility, parental monitoring and family environment.

However, the goal of the current study is to identify ethnic differences that may exist above and beyond the frequent confounds of SES and parental marital status. SES and parent marital status were entered into the first step of each regression to control for these potential confounds prior to entering ethnicity as a predictor. The final aim of the current study is to examine potential psychosocial mediators of sociodemographic effects on disease care variables. A series of hierarchical regression analyses were conducted to demonstrate these relations. According to Baron and Kenny (1986), there are four conditions which must be met to support a mediation model. First, there must be a significant relation between the predictor (sociodemographic variable) and the proposed mediator (psychosocial variable) (Path a in Figure 1). Second, the predictor (sociodemographic variable) must be significantly related to the outcome variable (disease care variable) to demonstrate an existing effect and merit further investigation of mediators (Path c in Figure 1). Third, the proposed mediator (psychosocial variable) must be significantly related to the outcome variable (disease care variable) while controlling for the predictor variable (sociodemographic variable) (Path b in Figure 1). And fourth, the relation between the predictor (sociodemographic variable) and outcome variable (disease care variable) must decrease in magnitude after entering a proposed mediator (psychosocial variable) (Path c versus Path c’ in Figure 1). The strength of a mediator is determined by the magnitude of change in the relation between the predictor and the outcome variable. A full mediation effect is demonstrated when the significance
of a predictor is reduced to a non-significant level after accounting for the mediator, whereas a partial mediation is demonstrated when a predictor remains significant, yet the level is reduced after accounting for the mediator (Frazier, Tix, & Barron, 2004; Holmbeck, 1997). In order to reduce type 1 error, mediator and outcome variables entered into the analyses were limited to those significantly intercorrelated as indicated by Pearson’s correlation coefficients. Additionally, the mediator and predictor variables were entered simultaneously to control for the influence of both the mediator and outcome variables (while inversely controlling for each) (Holmbeck, 1997; Baron & Kenny, 1986).
Results

Descriptive Results

Descriptive statistics for participant characteristics are presented in Table 1. The sample included 349 youth: 200 (57.3%) participants from Study 1 completed an evaluation of memory and learning effects on disease management and 149 (42.7%) participants from study 2 participated in baseline assessment of a randomized clinical trial to prevent deterioration of disease management. The mean age of the combined sample was 13.03 years ($SD = 1.63$) and mean disease duration was 4.57 years ($SD = 3.30$). Ethnicity was self-reported as Caucasian (79.9%) or African-American (20.1%), consistent with T1D prevalence rates among minority populations in metropolitan areas (Delamater et al., 1999). The majority of participants lived with their married biological parents (71.3%) in average middle-class households (mean SES = 46.24, $SD = 11.53$), consistent with literature on families of children with chronic illness (Shudy, de Almeida, Ly, Landon, Groft, & Jenkins, 2006).
Table 1

*Participant Characteristics*

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combined Study N</td>
<td>349</td>
</tr>
<tr>
<td>1&lt;sup&gt;st&lt;/sup&gt; study cohort</td>
<td>200 (57.3%)</td>
</tr>
<tr>
<td>DC site</td>
<td>198 (56.7%)</td>
</tr>
<tr>
<td>Female</td>
<td>175 (50.1%)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>279 (79.9%)</td>
</tr>
<tr>
<td>Married biological</td>
<td>249 (71.3%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>M (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>SES&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.24 (11.53)</td>
<td>11.50 – 66.00</td>
</tr>
<tr>
<td>Youth age</td>
<td>13.03 (1.63)</td>
<td>9.17 – 17.80</td>
</tr>
<tr>
<td>T1D duration</td>
<td>4.57 (3.30)</td>
<td>.05 - 13.63</td>
</tr>
</tbody>
</table>

<sup>a</sup>SES assessed by Hollingshead Index, lower scores indicate lower SES, higher scores indicate higher SES.

Disease care behaviors and psychosocial characteristics are presented in Table 2. The mean HbA1c level for this sample was 8.36% (SD = 1.52%), which is mildly elevated in comparison to current levels recommended by the ADA (< 7.5% for adolescents and < 8.0% for children; ADA, 2008a). Overall, participants’ disease care behaviors were consistent with broad ADA recommendations. Youth tested blood glucose levels 3 - 4 times per day (M = 3.72, SD = 1.29) and ate 4 meals or snacks per
day ($M = 4.24, SD = .88$). Youth consumed 47.26% ($SD = 9.30\%$) of daily calories from carbohydrates and 35.11% ($SD = 8.19\%$) from fats. Participants engaged in daily physical activity ($M = 1.19, SD = .74$) for approximately 25 minutes per day ($M = 24.23, SD = 38.06$).
Table 2

*Disease Care and Psychosocial Characteristics*

<table>
<thead>
<tr>
<th></th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin pump</td>
<td>95 (28.0%)</td>
</tr>
<tr>
<td>Intensive insulin</td>
<td>224 (66.1%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>M (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c</td>
<td>8.36 (1.52)</td>
<td>5.20 - 13.30</td>
</tr>
<tr>
<td>Blood glucose freq</td>
<td>3.72 (1.29)</td>
<td>.75 – 7.75</td>
</tr>
<tr>
<td>Eating freq</td>
<td>4.24 (.88)</td>
<td>2.00 - 6.00</td>
</tr>
<tr>
<td>% Calories- carbs</td>
<td>47.26 (9.30)</td>
<td>16.77 – 72.88</td>
</tr>
<tr>
<td>% Calories- fats</td>
<td>35.11 (8.19)</td>
<td>10.08 - 54.96</td>
</tr>
<tr>
<td>Exercise freq</td>
<td>1.19 (.74)</td>
<td>.00 - 3.25</td>
</tr>
<tr>
<td>Exercise duration</td>
<td>24.23 (38.06)</td>
<td>.00 - 150.00</td>
</tr>
<tr>
<td>TDK(^a)</td>
<td>56.77 (6.53)</td>
<td>38.00 – 68.50</td>
</tr>
<tr>
<td>DFRQ(^b)</td>
<td>36.47 (5.49)</td>
<td>21.00 – 49.00</td>
</tr>
<tr>
<td>PMDS(^c) (parent-report)</td>
<td>77.45 (7.91)</td>
<td>55.00 – 90.00</td>
</tr>
<tr>
<td>FES(^d)</td>
<td>13.21 (2.93)</td>
<td>4.00 – 18.00</td>
</tr>
</tbody>
</table>

\(^a\)Test of Diabetes Knowledge: higher scores indicate more diabetes knowledge. \(^b\)Diabetes Family Responsibility Questionnaire: lower scores indicate more parent responsibility, higher scores indicate more child responsibility. \(^c\)Parent Monitoring of Diabetes Scale: higher scores indicate more parental monitoring. \(^d\)Family Environment Scale: lower scores indicate low cohesion/high conflict, higher scores indicate high cohesion/low conflict.
Univariate correlation analyses were conducted for all sociodemographic, disease care and psychosocial variables of hypothesized interest. Pearson’s correlation coefficients for the total sample are presented in Table 3. As predicted, ethnicity was significantly correlated with sociodemographic factors of SES and parent marital status. Disease care factors correlated with ethnicity include: HbA1c levels, blood glucose frequency, insulin regimen (pump vs. injection and intensive vs. conventional), and percentage of daily calories from carbohydrates and fats. Of the four psychosocial factors, only diabetes knowledge was significantly correlated with ethnicity.
Table 3

Correlations between Sociodemographic, Disease Care and Psychosocial Variables from Combined Dataset

|---------------|-------------------|---------------------------|-------------------|----------|------------|--------------------------|--------------------------|-------------|----------|-----------|-------------|-------------|----------------------|----------------------|---------------------|******************|
| 1. Eth<sup>a</sup> |       |                           |                   |          |            |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 2. SES        | -.303 ***         |                           |                   |          |            |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 3. PMS<sup>b</sup> | -.268 ***        | .290 ***                  |                   |          |            |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 4. HbA1c      | .154 **           | -.207 ***                 | -.290 ***        |          |            |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 5. BG freq    | -.140 *           | .240 **                   | .148 **          | -.107 ***|            |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 6. Ins pump<sup>c</sup> | -.181 **         | .246 ***                  | .232 ***        | -.070    | .405 ***   |                          |                          |             |          |            |             |             |                      |                      |                     |                       |
| 7. Int insulin<sup>d</sup> | -.155 **         | .165 ***                  | .061 ***        | .064     | .432 ***   | .447 ***                 |                          |             |          |            |             |             |                      |                      |                     |                       |
| 8. Eat freq   | -.063 *           | -.026 **                  | .098 **         | -.175 ** | .121 *     | .150 *                  | .133 ***                 |             |          |            |             |             |                      |                      |                     |                       |
| 9. Carbs      | -.156 **         | .176 ***                  | .152 ***        | -.171 ** | .081 **    | .115 **                 | .111 ***                 | .007 ***    |          |            |             |             |                      |                      |                     |                       |
| 10. Fats      | .111 *           | -.218 **                  | -.049 **        | .048     | -.193 **   | .113 **                 | .233 **                 | -.003 **    | -.295 ***|            |             |             |                      |                      |                     |                       |
| 11. Ex freq   | .041 **          | .060 **                   | .022 **         | -.074 ** | .051 **    | -.067 **                | -.108 **                | .173 *    | .040     | -.051 ***|             |             |                      |                      |                     |                       |
| 12. Ex dur    | -.003 *          | .144 **                   | -.012 **        | -.022 ** | .459 **    | .335 **                 | .403 **                 | -.110 **   | -.021 ** | -.062 ***| -.026 ***  |             |                      |                      |                     |                       |
| 13. TDK<sup>e</sup> | -.381 ***        | .373 ***                  | .105 **         | -.232 ** | .246 **    | .273 **                 | .237 **                 | .003 **    | .221 **  | -.225 **  | -.068 **    | .073 **     |                      |                      |                     |                       |
| 14. DFRQ<sup>f</sup> | .016 *           | .129 *                    | -.066 **        | .039     | .240 **    | .250 **                 | .300 **                 | -.231 **   | -.094 ** | -.052 **  | -.200 **    | .461 **     | .162 ***               |                      |                     |                       |
| 15. PMDS<sup>g</sup> | -.016 *          | .000 *                    | .144 **         | -.168 ** | .220 **    | .061 **                 | -.076 **                | .284 **    | -.059 ** | .009 **   | .136 **     | .156 **     | n/a                   | -.128 ***               |                     |                       |
| 16. FES<sup>h</sup> | -.022 **         | .069 **                   | .243 **         | -.106 ** | .273 **    | .140 **                 | .053 **                 | .093 **    | .070 **  | -.133 **  | -.047 **    | .062 **     | .016 **                | .019 **                | .291 **            |                       |

Note. *p < .05, **p < .01, ***p < .001. Eth: 1 = Cau, 2 = AA. PMS: 1 = married biological parents, 0 = other arrangement. Ins pump: 1 = injections, 2 = pump. Int insulin: 1 = conventional, 2 = intensive. TDK: higher scores indicate greater diabetes knowledge. DFRQ: lower scores indicate greater parent responsibility, higher scores indicate greater child responsibility. PMDS: higher scores indicate greater parental monitoring. FES: lower scores indicate low cohesion/high conflict, higher scores indicate high cohesion/low conflict.
*Hypothesis 1: Ethnic Differences in Sociodemographic Variables and Metabolic Control*

Independent samples $t$-tests and chi-square analyses evaluated ethnic differences in SES, parent marital status and metabolic control between Caucasian and African-American youth. In order to reduce the likelihood of type 1 error given the large number of ethnic comparisons for these exploratory hypotheses, a sequentially selective Bonferroni adjustment was applied (Holm, 1979). Similarly, the Yates’ Correction for Continuity was applied to all chi-square analyses to account for the overestimation of chi-square values associated with $2 \times 2$ designs.

Ethnic group differences are presented in Table 4. Parents of Caucasian youth reported significantly higher SES levels ($M = 47.98$, $SD = 11.07$) than parents of African-American youth ($M = 39.29$, $SD = 10.73$); $t(347) = 5.91$, $p < .001$. Analyses revealed a moderate effect size for this ethnic group difference ($\eta^2 = .091$). A chi-square test for independence with Yates’ Continuity Correction revealed that a greater percentage of Caucasian youth lived with their married biological parents (77.4%) than African-American youth (47.1%). This ethnic group difference was significant; $\chi^2 (1, N = 349) = 23.63$, $p < .001$, and the effect size was moderate ($\phi = -.268$, $p < .001$).

Additionally, African-American youth had significantly higher HbA1c levels ($M = 8.84\%$, $SD = 1.96$) compared with Caucasian youth ($M = 8.25\%$, $SD = 1.37$); $t(76.16) = -2.24$, $p < .05$, though the effect size was small ($\eta^2 = .016$).
Table 4

**Sociodemographic, Disease Care and Psychosocial Characteristics by Ethnicity**

<table>
<thead>
<tr>
<th></th>
<th>Caucasian (N = 279)</th>
<th>African-American (N = 70)</th>
<th>χ²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Married biological</td>
<td>216 (77.4%)</td>
<td>33 (47.1%)</td>
<td>23.63***</td>
</tr>
<tr>
<td>Insulin pump</td>
<td>87 (32.1%)</td>
<td>8 (11.8%)</td>
<td>10.16**</td>
</tr>
<tr>
<td>Intensive insulin</td>
<td>189 (69.7%)</td>
<td>35 (51.5%)</td>
<td>7.30**</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
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<td></td>
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<tr>
<td>SES</td>
<td>47.98 (11.07)</td>
<td>39.29 (10.73)</td>
<td>5.91***</td>
</tr>
<tr>
<td>HbA1c</td>
<td>8.25 (1.37)</td>
<td>8.84 (1.96)</td>
<td>-2.24*</td>
</tr>
<tr>
<td>Blood glucose freq</td>
<td>3.81 (1.30)</td>
<td>3.35 (1.21)</td>
<td>2.59*</td>
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<tr>
<td>Eating freq</td>
<td>4.27 (.88)</td>
<td>4.13 (.87)</td>
<td>1.16</td>
</tr>
<tr>
<td>% Calories- carbs</td>
<td>47.97 (9.15)</td>
<td>44.34 (9.42)</td>
<td>2.88**</td>
</tr>
<tr>
<td>% Calories- fats</td>
<td>34.66 (8.23)</td>
<td>36.93 (7.82)</td>
<td>-2.06*</td>
</tr>
<tr>
<td>Exercise freq</td>
<td>1.18 (.71)</td>
<td>1.25 (.83)</td>
<td>-.72</td>
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<tr>
<td>Exercise duration</td>
<td>24.29 (38.17)</td>
<td>23.97 (37.90)</td>
<td>.06</td>
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<tr>
<td>TDK</td>
<td>57.91 (5.82)</td>
<td>51.37 (7.10)</td>
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<tr>
<td>DFRQ</td>
<td>36.43 (5.47)</td>
<td>36.66 (5.65)</td>
<td>-2.80</td>
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<tr>
<td>PMDS (parent-report)</td>
<td>77.51 (7.74)</td>
<td>77.19 (8.73)</td>
<td>.19</td>
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<tr>
<td>FES</td>
<td>13.24 (2.93)</td>
<td>13.08 (2.96)</td>
<td>.39</td>
</tr>
</tbody>
</table>

*Note. *p < .05. **p < .01. ***p < .001. Italics: t-tests remain statistically significant after Sequentially Selective Bonferroni Correction or Yates’ Continuity Correction.*
Hypothesis 2: Ethnic Differences in Disease Care Variables

Independent samples t-tests evaluated ethnic differences in disease care behaviors related to insulin regimen, blood glucose monitoring, nutritional intake, and physical activity (See Table 4).

Insulin regimen by ethnicity. A comparison of insulin regimens revealed a larger percentage of Caucasian youth (32.1%) were prescribed an insulin pump (versus insulin injections) in comparison to African-American youth (11.8%); $\chi^2 (1, N = 339) = 10.16, p < .01$, though the effect size was small ($\phi = -.181, p < .01$). Further comparisons revealed intensive insulin treatments (insulin pump or ≥ 3 insulin shots per day) were more frequently prescribed to Caucasian youth (69.7%) than to African American youth (51.5%); $\chi^2 (1, N = 339) = 7.30, p < .01$, with a small effect size ($\phi = -.155, p < .01$).

Blood glucose monitoring by ethnicity. Combined parent- and child-report scores revealed that Caucasian youth ($M = 3.81, SD = 1.30$) tested blood glucose levels more frequently than African-American youth ($M = 3.35, SD = 1.21$); $t (335) = 2.59, p < .05$. Though this difference was significant, the effect size was small ($\eta^2 = .020$).

Nutritional intake by ethnicity. Analyses of nutritional intake revealed Caucasian youth adhered more closely with ADA’s recommended dietary guidelines by consuming a greater percentage of daily calories from carbohydrates ($M = 47.97\%, SD = 9.15$) and a lower percentage of calories from fats ($M = 34.66\%, SD = 8.23$) than African-American youth ($M = 44.34\%, SD = 9.42; M = 36.93\%, SD = 7.82$ respectively). The difference in carbohydrate consumption was significant, $t (333) = 2.88, p < .01$, yet the effect size was small ($\eta^2 = .024$). Similarly, the ethnic difference in fat consumption was
significant, $t(334) = -2.06, p < .05$, yet the effect size was small ($\eta^2 = .013$).

Contrary to the hypothesis, Caucasian and African-American youth did not adhere differentially to other disease care behaviors of eating frequency, exercise frequency, or exercise duration.

**Hypothesis 3: Ethnic Differences in Psychosocial Variables**

Independent samples $t$-tests were used to detect ethnic group differences in psychosocial measures of diabetes knowledge, disease responsibility, parental monitoring, and family environment (See Table 4). Consistent with existing literature, ethnicity was significantly related to diabetes knowledge. Caucasian parents and children ($M = 57.91, SD = 5.82$) scored significantly higher on the Test of Diabetes Knowledge (TDK) than African-American parents and children ($M = 51.37, SD = 7.10$); $t(153) = 5.10, p < .001$. Contrary to the hypothesis, there were no ethnic differences in division of diabetes responsibility, parental monitoring of diabetes management, or family environment.

**Unique Ethnic Differences in Psychosocial and Disease Care Variables**

Hierarchical regression analyses were conducted to determine whether ethnic differences remained after controlling for the confounded effects of SES and parent marital status. To reduce type 1 error, psychosocial and disease care variables were limited to those found to differ by ethnicity in the independent samples $t$-tests: 1) HbA1c, 2) insulin regimen, 3) blood glucose testing frequency, 4) dietary composition, and 5) diabetes knowledge.
HbA1c. Ethnicity was evaluated as a predictor of HbA1c, after controlling for SES and parent marital status. While the overall model was significant, $F (3, 312) = 11.97, p < .001$, $R^2 = .103$, ethnicity did not explain any additional variance beyond the control variables, $F$ change $(1, 312) = .847, R^2$ change $= .002$. Though there was no association between ethnicity ($\beta = .053, p = .358$) and HbA1c, both parent marital status ($\beta = -.241, p < .001$) and SES ($\beta = -.122, p < .05$) were significant predictors of HbA1c.

Insulin regimen. Although the model was significant, $F (3, 335) = 11.761, p < .001$, $R^2 = .095$, ethnicity did not explain any additional variance beyond SES and parent marital status, $F$ change $(1, 336) = 2.398, p = .122, R^2$ change $= .006$. Both SES ($\beta = .175, p < .01$) and parent marital status ($\beta = .158, p < .01$) emerged as significant individual predictors of insulin regimen, but not ethnicity ($\beta = -.086, p = .122$).

Blood glucose frequency. Ethnicity was evaluated as a predictor of blood glucose testing frequency, after controlling for SES and parent marital status. Although the overall model was significant, $F (3, 333) = 8.046, p < .001$, $R^2 = .068$, ethnicity did not explain a significant portion of unique variance in blood glucose frequency beyond the effects of SES and parent marital status, $F$ change $(1, 333) = 1.097, p = .127, R^2$ change $= .003$. Neither ethnicity ($\beta = -.059, p = .296$) nor parent marital status ($\beta = .074, p = .191$) were significant individual predictors of blood glucose monitoring, although SES was significant ($\beta = .201, p < .001$).

Carbohydrate consumption. Although the overall model was significant, $F (3, 331) = 5.813, p < .01$, $R^2 = .050$, ethnicity did not explain a significant portion of unique variance in carbohydrate consumption beyond the effects of SES and parent marital
status, $F$ change (1, 331) = 2.720, $p$ = .10, $R^2$ change = .008. Neither ethnicity ($\beta$ = -.095, $p$ = .100) nor parent marital status ($\beta$ = .092, $p$ = .109) were significant individual predictors of carbohydrate consumption, though SES was significant ($\beta$ = .121, $p$ < .05).

Fat consumption. Ethnicity was evaluated as a predictor of fat consumption, after controlling for SES and parent marital status. The model was significant, $F$ (3, 332) = 5.873, $p$ < .01; $R^2$ = .050, yet ethnicity did not explain any additional variance beyond the control variables, $F$ change (1, 332) = .921, $p$ = .338, $R^2$ change = .003. Neither ethnicity ($\beta$ = .055, $p$ = .338) nor parent marital status ($\beta$ = .026, $p$ = .647) were significant individual predictors of fat consumption, though SES was significant ($\beta$ = -.209, $p$ < .001).

Diabetes knowledge. Ethnicity explained an additional 8.3% of variance in diabetes knowledge, beyond the effects of SES and parent marital status, $F$ (3, 151) = 14.37, $p$ < .001; $R^2$ = .222. Both ethnicity ($\beta$ = -.308, $p$ < .001) and SES ($\beta$ = .298, $p$ < .001), but not parent marital status ($\beta$ = -.065, $p$ = .40) were significant individual predictors of diabetes knowledge.

*Hypothesis 4: Psychosocial Mediators of Ethnic Differences in Metabolic Control and Disease Care*

A series of hierarchical multiple regressions were planned originally to evaluate diabetes knowledge as a potential psychosocial mediator of significant ethnic differences. However, since multiple regressions indicated ethnic differences were in fact attributable to SES differences, a *post hoc* data analysis plan explored mediators of the effect of SES...
on group differences, while controlling for the effects of ethnicity and parent marital status (See Figure 2 for revised mediation model).

Figure 2. Proposed Model for Mediating Effects of Diabetes Knowledge on the Relationship between SES and Disease Care Factors.

Further, because diabetes knowledge was assessed only in Study 1, regression analyses were conducted exclusively with this dataset. In order to demonstrate participants from Study 1 were representative of participants in Study 2 and that study results should generalize to all participants in the combined dataset, independent sample t-tests and chi-square analyses were used to detect significant differences between data from Study 1 and Study 2 in the variables of interest in Table 5. After applying a sequentially selective Bonferroni adjustment to minimize Type 1 error (Holm, 1979) significant study differences remained for the following variables: insulin regimen, blood glucose testing frequency, and diabetes responsibility. Reflecting trends over time toward intensified insulin regimens, there was a significant difference in the percentage
of participants prescribed an insulin pump in Study 1 (12.6%) and Study 2 (48.0%), $\chi^2 (1, N = 339) = 50.09$, $p < .001$, and the effect size was moderate ($\phi = .391$, $p < .001$).

Similarly, a larger percentage of participants were prescribed an intensive insulin regimen in Study 2 (93.2%) than Study 1 (45.0%), $\chi^2 (1, N = 339) = 84.35$, $p < .001$, and again the effect size was moderate ($\phi = .505$, $p < .001$). Study 1 participants ($M = 3.24$, $SD = .94$) reported significantly fewer daily blood glucose checks in comparison to Study 2 participants ($M = 4.36$, $SD = 1.41$); $t (234.63) = -8.291$, $p < .001$. Finally, participants from Study 2 ($M = 39.98$, $SD = 4.20$) reported more child diabetes responsibility than Study 1 participants ($M = 33.14$, $SD = 4.39$); $t (304) = -13.908$, $p < .001$. Differences between the Study 1 and Study 2 generally reflect changes in standards of care over time with greater use of intensified insulin regimens and increased blood glucose monitoring.

Most importantly, study groups did not differ in sociodemographic features over time which indicates the meditational effects described in the Study 1 subsample should generalize to the combined sample as a whole.
Table 5

**Sociodemographic, Disease Care and Psychosocial Characteristics by Study Cohort**

<table>
<thead>
<tr>
<th></th>
<th>Total Sample (N = 349)</th>
<th>Study 1 (N = 200)</th>
<th>Study 2 (N = 149)</th>
<th>( \chi^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%</td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
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</tr>
<tr>
<td>Married biological</td>
<td>249 (71.3%)</td>
<td>145 (72.5%)</td>
<td>104 (69.8%)</td>
<td>.187</td>
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<tr>
<td>Insulin pump</td>
<td>95 (28.0%)</td>
<td>24 (12.6%)</td>
<td>71 (48.0%)</td>
<td>50.088***</td>
</tr>
<tr>
<td>Intensive insulin</td>
<td>224 (66.1%)</td>
<td>86 (45.0%)</td>
<td>138 (93.2%)</td>
<td>84.349***</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td>t</td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td>46.24 (11.53)</td>
<td>45.43 (11.48)</td>
<td>47.33 (11.54)</td>
<td>-1.528</td>
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<tr>
<td>HbA1c</td>
<td>8.36 (1.52)</td>
<td>8.30 (1.58)</td>
<td>8.46 (1.41)</td>
<td>-.906</td>
</tr>
<tr>
<td>Blood glucose freq</td>
<td>3.72 (1.29)</td>
<td>3.24 (.94)</td>
<td>4.36 (1.41)</td>
<td>-8.291***</td>
</tr>
<tr>
<td>Eating freq</td>
<td>4.24 (.88)</td>
<td>4.34 (.87)</td>
<td>4.11 (.88)</td>
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<tr>
<td>% Calories- carbs</td>
<td>47.26 (9.30)</td>
<td>48.42 (7.49)</td>
<td>45.69 (11.12)</td>
<td>2.536*</td>
</tr>
<tr>
<td>% Calories- fats</td>
<td>35.11 (8.19)</td>
<td>35.79 (6.54)</td>
<td>34.20 (9.94)</td>
<td>1.659</td>
</tr>
<tr>
<td>Exercise freq</td>
<td>1.19 (.74)</td>
<td>1.26 (.76)</td>
<td>1.10 (.69)</td>
<td>1.893</td>
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<tr>
<td>DFRQ</td>
<td>36.47 (5.49)</td>
<td>33.14 (4.39)</td>
<td>39.98 (4.20)</td>
<td>-13.908***</td>
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<tr>
<td>FES</td>
<td>13.21 (2.93)</td>
<td>13.07 (2.65)</td>
<td>13.36 (3.21)</td>
<td>-.869</td>
</tr>
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</table>

*Note. *p < .05. **p < .01. ***p < .001. Italics: t-tests remain statistically significant after Sequentially Selective Bonferroni Correction or Yates’ Continuity Correction.*

In order to reduce type 1 error, mediator and outcome variables entered into the analyses were limited to those significantly intercorrelated as indicated by Pearson’s correlation coefficients conducted on Study 1 data (See Table 6). Accordingly, diabetes
knowledge emerged as a potential mediator on the relation between SES and four disease care outcome variables: 1) HbA1c, 2) blood glucose frequency, 3) percentage of calories from carbohydrates, and 4) percentage of calories from fats. At each step, ethnicity and parent marital status were entered first, to control for these potential confounds.
Table 6

**Correlations between Sociodemographic, Disease Care and Psychosocial Variables from Study 1**

<table>
<thead>
<tr>
<th></th>
<th>1.</th>
<th>2.</th>
<th>3.</th>
<th>4.</th>
<th>5.</th>
<th>6.</th>
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<th>9.</th>
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<th>11.</th>
<th>12.</th>
<th>13.</th>
<th>14.</th>
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</thead>
<tbody>
<tr>
<td>1. Eth(^a)</td>
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<tr>
<td>2. SES</td>
<td>-0.253</td>
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<td>3. PMS(^b)</td>
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<td>4. HbA1c</td>
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<tr>
<td>5. BG freq</td>
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<td>6. Ins pump(^c)</td>
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<td>7. Int ins(^d)</td>
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<td>8. Eat freq</td>
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<td>9. Carbs</td>
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<td>10. Fats</td>
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<td>11. Ex freq</td>
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<td>12. Ex dur</td>
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<td>13. TDK(^e)</td>
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<td>14. DFRQ(^f)</td>
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<td>15. FES(^g)</td>
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</table>

**Note.** *p < .05. **p < .01. ***p < .001. \(^a\)Eth: 1 = Cauc, 2 = AA. \(^b\)PMS: 1 = married biological parents, 0 = other arrangement. \(^c\)Ins pump: 1 = injections, 2 = pump. \(^d\)Int insulin: 1 = conventional, 2 = intensive. \(^e\)TDK: higher scores indicate greater diabetes knowledge. \(^f\)DFRQ: lower scores indicate greater parent responsibility, higher scores indicate greater child responsibility. \(^g\)FES: lower scores indicate low cohesion/high conflict, higher scores indicate high cohesion/low conflict.
HbA1c. The first model proposed diabetes knowledge as a mediator of the effects of SES on HbA1c, beyond the effects of ethnicity and parent marital status (See Figure 3). In the first regression equation, SES was evaluated as a predictor of diabetes knowledge, after controlling for ethnicity and parent marital status (Path a). The model was significant, $F(3, 151) = 14.795, p < .001; R^2 = .227$, demonstrating the first condition of mediation (Path a). SES explained an additional 8.0% of variance in diabetes knowledge, $F$ change $(1, 151) = 15.650, p < .001, R^2$ change $= .080$. Both SES ($\beta = .297, p < .001$) and ethnicity ($\beta = -.308, p < .001$), but not parent marital status ($\beta = -.010, p = .894$) were significant individual predictors of diabetes knowledge.

In the second regression equation, SES was entered to predict HbA1c, after controlling for ethnicity and parent marital status (Path c). Although the overall model was significant, $F(3, 194) = 9.977, p < .001, R^2 = .134$, SES did not explain any additional variance, $F$ change $(1, 194) = 2.804, p = .096, R^2$ change $= .013$. Though SES ($\beta = -.118, p = .096$) and ethnicity ($\beta = .067, p = .334$) were not significant, parent marital status ($\beta = -.299, p < .001$) was a significant predictor of HbA1c. No further analyses were performed because the results failed to demonstrate the second condition of mediation (Path c).
Figure 3. HbA1c as Predicted by SES and Mediated by Diabetes Knowledge.

**Blood glucose testing frequency.** The second mediation model proposed diabetes knowledge as a mediator for the effects of SES on blood glucose frequency, after controlling for ethnicity and parent marital status (See Figure 4). The significant association between SES and diabetes knowledge (Path a) was demonstrated in the previous mediation model and will therefore be assumed for all subsequent models.

In the second regression equation, SES was entered to predict blood glucose frequency, beyond the effects of control variables (Path c), which yielded a significant model, $F(3, 189) = 3.758, p < .05, R^2 = .056$. However, SES did not explain a significant portion of unique variance, $F$ change (1, 189) = 1.295, $p = .257$, $R^2$ change = .006. Neither SES ($\beta = .084, p = .257$) nor parent marital status ($\beta = .049, p = .505$) were significant individual predictors of blood glucose monitoring, although ethnicity was significant ($\beta = -.185, p < .05$). No further analyses were performed because the results failed to demonstrate the second condition of mediation (Path c).
Carbohydrate consumption. The third mediation model proposed diabetes knowledge as a mediator for the effects of SES on carbohydrate consumption, beyond the effects of ethnicity and parent marital status (See Figure 5). After demonstrating the first condition of mediation (Path a), the second regression equation was performed to determine whether SES predicted percentage of calories from carbohydrates after controlling for ethnicity and parent marital status (Path c). The model was significant, $F(3, 188) = 4.088, p < .01, R^2 = .061$, and SES explained a portion of unique variance beyond the effects of ethnicity and parent marital status, $F$ change $(1, 188) = 8.486, p = .01, R^2$ change $= .042$. SES emerged as a significant individual predictor of carbohydrate consumption ($\beta = .216, p = .01$), demonstrating the second condition of mediation (Path c). However, parent marital status ($\beta = .016, p = .828$), and ethnicity ($\beta = -.070, p = .345$) were not significant predictors.
In the third regression analysis, SES was entered simultaneously with diabetes knowledge to predict carbohydrate consumption after controlling for ethnicity and parent marital status. The model was significant, $F(4, 146) = 3.071, p < .05, R^2 = .078$. SES and diabetes knowledge explained an additional portion of unique variance, $F$ change (2, 146) = 4.648, $p < .05, R^2$ change = .059. In the final model, diabetes knowledge was not a significant predictor of carbohydrate consumption ($\beta = .145, p = .110$), indicating no mediational effect according to Baron and Kenny (1986). However, the significant effect of SES was reduced ($\beta = .173, p = .50$), indicating that diabetes knowledge accounted for additional variance by suppressing the effect of social class on carbohydrate consumption.

Figure 5. Carbohydrate Consumption as Predicted by SES and Mediated by Diabetes Knowledge.

**Fat consumption.** The fourth mediation model proposed diabetes knowledge as a mediator for the effects of SES on fat consumption, beyond the effects of ethnicity and
parent marital status (See Figure 6). In the second regression equation, SES was entered to predict fat consumption, while controlling for ethnicity and parent marital status (Path c). The model was significant, $F (3, 189) = 4.447, p < .01$, $R^2 = .066$, and SES explained an additional portion of unique variance beyond the effects of ethnicity and parent marital status, $F \text{ change } (1, 189) = 7.380, p < .01$, $R^2 \text{ change } = .036$. SES emerged as a significant individual predictor of fat consumption ($\beta = -.201, p < .01$), demonstrating the second condition of mediation (Path c). However, parent marital status ($\beta = -.026, p = .720$) and ethnicity ($\beta = .106, p = .150$) were not significant predictors.

In the third regression analysis, SES was entered simultaneously with diabetes knowledge to predict fat consumption after controlling for ethnicity and parent marital status. The model was significant, $F (4, 147) = 3.232, p < .05$, $R^2 = .081$. SES and diabetes knowledge explained an additional portion of unique variance beyond the effects of control variables, $F \text{ change } (2, 147) = 4.107, p < .05$, $R^2 \text{ change } = .051$. In the final model, diabetes knowledge was not a significant predictor of fat consumption ($\beta = -.139, p = .125$), indicating no mediational effect according to Baron and Kenny (1986). However, the effect of SES was reduced to non-significance ($\beta = -.159, p = .70$), indicating that diabetes knowledge accounted for additional variance by suppressing the effect of social class on percentage of calories from fats.
Figure 6. Fat Consumption as Predicted by SES and Mediated by Diabetes Knowledge.
Discussion

Little study of ethnic differences in disease outcomes exists for youth with T1D, and available evidence rarely accounts for frequently confounded sociodemographic factors. Additionally, small sample sizes of African-American youth prevent researchers from adequately investigating individual self-care behaviors and potential psychosocial mediators of ethnic differences. The current study sought rigorous evaluation and extension of reported ethnic differences through a large-scale investigation with a sample of 349 youth with T1D.

Person-oriented group comparisons found ethnic differences in sociodemographic status and disease care, consistent with the available literature. However, a variable-oriented approach which controlled for the confounding influences of SES and parental marital status on ethnicity shows most disease care effects attributed to ethnicity are in fact better explained by social class. Finally, diabetes knowledge is evaluated as a potential mediator of social class effects.

*Person-Oriented Ethnic Differences*

As hypothesized, ethnic differences in SES and parental marital status were detected in the current large-scale study, mirroring patterns found in the US population and those reported in previous pediatric literature (Delamater et al., 1999; Shudy et al., 2006, Thompson et al., 2001).
As hypothesized, ethnic differences were found in metabolic control, insulin regimen, blood glucose monitoring frequency and meal composition. African-American youth were in poorer metabolic control than Caucasian youth, as indicated by a .59% difference in HbA1c levels. Though this discrepancy was not as pronounced as current literature which reports ethnic differences in HbA1c levels as high as 1.5% (Auslander et al., 1997; Chalew et al., 2000; Delamater et al., 1999), this discrepancy remains clinically significant and likely to be associated with clinically significant differences in serious chronic disease complications with longer disease duration.

Ethnic differences in metabolic control may be attributed to discrepancies in prescribed insulin regimens in the current study which reflect regimen differences reported by Valenzuela and colleagues (2006) for both African-American and Caucasian samples. Ethnic differences in type of insulin regimen may be attributed to health disparities in access to insulin pump regimens. The prevalence of insulin pump regimens among African-American youth is considerably lower than Caucasian youth, especially given the current effort to transition to intensive treatment regimens after the DCCT follow-up report (DCCT, 1994). However, it is important to note that this health disparity may partially reflect the results of Study 1, when data collection occurred during a time that insulin pumps were first adopted in pediatric populations (1998-2005).

Consistent with this hypothesis, the rates of insulin pump usage increased across time from Study 1 to Study 2 for both African-American and Caucasian youth (See Table 5). Though costs have decreased considerably, insulin pumps remain expensive,
complicated, and may not always covered by health insurance, especially state or public health insurance.

As hypothesized, African-American families reported less frequent blood glucose testing than Caucasians by approximately one-half check per day. This ethnic difference is consistent with the trend reported in current literature (Auslander et al., 1997; Patino et al., 2005; Lipton et al., 2003). Greater blood glucose monitoring frequency relates to more intensified insulin regimens and has been shown to independently predict HbA1c levels (Holmes, et al., 2006). Additionally, ethnic differences were detected in meal composition for youth with T1D. African-American youth consumed fewer carbohydrates and a greater percentage of calories from fats than Caucasian youth. While dietary differences are statistically significant they may not be clinically significant. Compared to recommendations from the American Dietetic Association, Caucasian youth consumed only slightly more than the minimum percentage of carbohydrates and only slightly less than maximum percentage of fats, while African-American youth consumed slightly under the minimum percentage of carbohydrates and slightly exceeded the maximum percentage of fats (ADA, 2004).

**Ethnic differences in psychosocial factors.** Of all the psychosocial variables of interest, ethnic differences emerged only for diabetes knowledge. Caucasian parents and youth exhibited greater diabetes-related knowledge in comparison to their African-American counterparts. The ethnic difference in disease knowledge is both statistically and clinically significant, given the strong relation between a caregiver’s ability to comprehend and implement diabetes knowledge in a child’s disease care, as well as
overall adherence to medical regimen and metabolic control (Stallwood, 2006; Chisholm et al., 2007). However, Caucasian and African-American families reported comparable levels of diabetes responsibility, parental monitoring and family environment.

The absence of significant ethnic differences in psychosocial factors is surprising in light of the detected differences in disease care behaviors including blood glucose monitoring, meal composition and insulin regimen. For instance, discrepancies in adherence comparable to those detected in the current study often are attributed to differences in parental involvement, responsibility, or direct observation of daily disease care (Palmer et al., 2004, Helgeson et al., 2008; Weibe et al., 2005; Ellis et al., 2007; Holmes et al., 2006). However, many previous investigations with significant psychosocial differences have compared upper-middle SES Caucasian families to lower SES African-American families. The current study’s sample is comprised of ethnic groups that differ significantly in SES, yet are still largely in the middle class range. Therefore, the present lack of significant psychosocial effects may be an artifact of the relatively similar SES status of both ethnic groups. Conversely, parental responsibility and monitoring greatly influences youths’ food selection and is associated with meal composition for populations without T1D (Cullen et al., 2002). Hence, one might expect to find a similar pattern in psychosocial factors given the ethnic differences in carbohydrate and fat consumption found in the current study.

**Variable-Oriented Ethnic Differences**

Persistent sociodemographic and ethnic confounds suggest the need to disentangle the individual influences of co-occurring SES and parent marital status to more
accurately ascribe disease characteristics to pertinent sociodemographic factors. More accurate description may facilitate appropriate interventions to improve disease care and metabolic control. Importantly, although the person-oriented or group difference approach revealed ethnic differences consistent with the literature, the variable-oriented approach failed to validate ethnic differences once the confounding influence of SES and parent marital status were statistically removed. In fact, the only significant ethnic difference that remained was a difference in diabetes knowledge. Poorer metabolic control or higher HbA1c levels and disease care behaviors previously attributed to ethnicity, are better accounted for by lower SES and in the case of HbA1c and insulin regimen, by nontraditional parent marital status. Thus, the mediational data analysis plan was adjusted post hoc to reflect the predominant role of SES, not ethnicity, as the sociodemographic factor of import.

Results from the current study deviate slightly from Swift et al. (2006), which implicated parent marital status as the sole predictor of metabolic control. Variable-oriented analyses reveal both parent marital status and SES as significant predictors of HbA1c levels. The larger sample size in the current study (N = 349) may explain this difference compared to the Swift et al. study (N = 211). Additionally, differences may be attributed to methodology, as Structural Equation Modeling also simultaneously considers many other factors, including individual disease care behaviors compared to multiple regression analyses used in the current study. However, in the first mediation model in the current study, only parent marital status emerges as a significant predictor of HbA1c levels (Figure 3), whereas SES and ethnicity do not remain significant.
Regardless of the varying role of SES in HbA1c levels, the two studies present the consistent finding that ethnicity is relatively unimportant as a differential predictor of disease care behaviors and metabolic control outcomes.

*Diabetes Knowledge as a Mediator*

Diabetes knowledge did not mediate SES differences in HbA1c or blood glucose monitoring. In other words, SES effects were equally influential on metabolic control and blood glucose monitoring, regardless of level of disease knowledge. The absence of mediational effects is somewhat surprising, but the implications of these models remain important in disentangling sociodemographic factors. When considering the overlap of minority status, low SES, and single-parent marital status, an individual presenting with these combined sociodemographic characteristics may likely face the compounded risk of poorer metabolic control and associated long-term adverse health outcomes.

While diabetes knowledge was not a significant mediator of meal composition, it appeared to suppress the effect of social class on the percentage of daily calories from carbohydrates and fats (See Figure 5 & 6). A youth’s SES level is less important in predicting carbohydrate consumption when disease knowledge is taken into account. Similarly, while diabetes knowledge was not independently related to fat consumption, the inclusion of this factor negated the effect of SES on percentage of daily calories from fats. With increased diabetes knowledge, low SES youth may make healthier dietary decisions and food selections, despite the negative influence of low SES (e.g., lower cost of high fat foods, lack of healthier, more expensive, more complex carbohydrate food choices).
Strengths, Limitations and Future Directions

One of the major contributions of the current study is the direct comparison of a person-oriented approach, which compares two ethnic groups, versus the variable-oriented approach, which statistically controls for confounded demographic characteristics. Person-oriented t-test findings contribute to an understanding of the constellation of factors that an individual youth is likely to bring into the pediatrician’s office when seen for diabetes care. According to population statistics, an African-American child is more likely to live in a single-parent, lower SES household than any other sociodemographic combination (Lugaila & Overturt, 2004; Federal Interagency Forum on Child and Family Statistics, 2007). Additionally, results from the person-oriented analyses extend previous findings through in-depth evaluation of daily disease care behaviors rather than a comparison of an overall global “adherence” construct. However, person-oriented analyses pose risk of potential bias by not accounting for confounded sociodemographic influences. Alternately, variable-oriented meditational analyses conducted to reduce potential bias, implicate SES as the predominant sociodemographic feature that affects disease care via level of diabetes knowledge.

More importantly, results from the person-oriented analyses, in conjunction with results from the variable-oriented analyses suggest that differences in disease care and HbA1c that are frequently attributed to ethnicity may more accurately be attributed to persistent SES differences in ethnicity. These hypotheses may be better evaluated with a matched-pairs approach to determine whether ethnic differences persist within matched levels of SES, a method used in previous research with pediatric T1D samples.
(Overstreet et al., 1995). By using a stratified match procedure, future investigations may also match groups for parent marital status. Though this method would enable researchers to identify unique ethnic effects, this method also has limitations to consider such as how SES and marital status may have differential influences among ethnic groups.

A unique strength of the current study is the ethnic and SES diversity of the participant sample. Previously, researchers have neglected to account for the intercorrelation of sociodemographic factors in evaluation of ethnic differences, often comparing upper-middle class Caucasian families to lower income minority families (e.g., Auslander et al., 1997). The present sample is comprised of a large number of African-American youth with overall average socioeconomic status as well as a more diverse range of SES among the Caucasian and African-American families.

A discussion of limitations may inform future investigation of sociodemographic influences on disease outcomes. One major limitation is the use of cross-sectional data to demonstrate relations among sociodemographic, psychosocial and disease care variables. While cross-sectional data may provide evidence for these relations, longitudinal data are necessary and more powerful to demonstrate causal effects (Kazdin, 2003).

An additional methodological limitation is the use of multiple regression analyses rather than Structural Equation Modeling (SEM). SEM may be a more appropriate, powerful statistical method (Frazier et al., 2004), however, this technique requires a substantial sample that is difficult to acquire when working with pediatric populations in comparison to larger child clinical populations. As with many pediatric populations,
sample sizes are limited and therefore multiple regression is more commonly accepted in this field than in general child psychology research (Holmbeck, 1997). Future investigations in this area may remedy these methodological limitations, increase sensitivity, and optimize effect sizes by oversampling minority youth. Additionally, the inclusion of multiple ethnic minority groups may further increase generalizability.

The results from the current investigation, in conjunction with previous literature have implications in future interventions for youth at risk of poor metabolic control. Specifically, lower SES families may benefit from comprehensive diabetes education programs to facilitate successful implementation of diabetes care (e.g., nutrition guidelines relating to carbohydrates and fat consumption). Given the correlation between insulin pump regimens and higher diabetes knowledge, it may be important to ensure parents are both knowledgeable and competent when youth are prescribed insulin pump regimens in order to optimize treatment outcomes. While ethnic differences in parental responsibility and monitoring did not emerge in the current study, these results, in conjunction with the findings of Cullen et al. (2002) which indicated the importance of youth’s perception of parental permissiveness in food choice, may provide and interesting avenue for future intervention in nutrition education and food selection. Once a comprehensive set of descriptive effects are localized to a particular sociodemographic characteristic, future investigation of psychosocial mediators may reveal additional appropriate targets for effective interventions for youth with T1D across ethnicity, SES and parent marital status groups.
References
References


Vita

Priscilla Wyatt Powell was born on August 3, 1982, in Richmond, Virginia. She graduated from Collegiate School in Richmond, Virginia in 2001. She received her Bachelor of Arts in Psychology from Washington & Lee University, in Lexington, Virginia in 2005.