RACE AND NEIGHBORHOOD SES DIFFERENCES IN THE

DEVELOPMENTAL TRAJECTORIES OF TYPE 1 DIABETES

MANAGEMENT OF CAUCASIAN AND

ETHNIC MINORITY YOUTH

APPROVED BY SUPERVISORY COMMITTEE

_____

_____

_____

_____

_____

DEDICATION

For my parents

who came to this country with

nothing

to give their children

everything…

謝謝, 媽媽爸爸

RACE AND NEIGHBORHOOD SES DIFFERENCES IN THE DEVELOPMENTAL TRAJECTORIES OF TYPE 1 DIABETES MANAGEMENT OF CAUCASIAN AND ETHNIC MINORITY YOUTH

by

JENNY TZU-MEI WANG

DISSERTATION

Presented to the Faculty of the Graduate School of Biomedical Sciences

The University of Texas Southwestern Medical Center at Dallas

In Partial Fulfillment of the Requirements

For the Degree of

DOCTOR OF PHILOSOPHY

The University of Texas Southwestern Medical Center at Dallas

Dallas, TX

August, 2009

Copyright

by

JENNY TZU-MEI WANG, 2009

All Rights Reserved

ACKNOWLEDGEMENTS

There are not enough pages in this section to truly express my gratitude and thanks to those who have significantly contributed to my graduate training and the successful completion of this project. That being said, I am overwhelmingly grateful for the support, guidance, and insightful challenges of my dissertation committee who helped me make this project one of significance in the area of pediatric psychology. Dr. Deborah Wiebe became my mentor in my early years of graduate school and has been a source of support and direction for this project. Her willingness and encouragement to develop a project that suited my interest in health disparities allowed me to explore an area that has deep personal and intellectual meaning to me. I sincerely appreciate the early morning meetings and late-night emails that she tolerated with grace while I completed this project. Dr. Sunita Stewart is a role model that I admired even before my admission to the program. I appreciate her role in enabling me to work at CMCD and for all the knowledge that I gained from working there. Her research abroad continues to inspire me to one day use my skills in multicultural settings. I would like to thank Dr. Perrin White for the opportunity to work at the pediatric endocrinology center at CMCD that provided me with a first-hand perspective of how families cope with diabetes, which greatly informed the development of this project. I am grateful for Dr. Crista Wetherington who was my mentor throughout my time at CMCD. Her encouragement and belief in my potential

have been invaluable during my training. I would also like to thank Dr. Margaret Caughy whom I was fortunate enough to meet towards the end of my training and inspires me with her research and dedication to underserved and high-risk communities. I appreciate all of her insights that greatly contributed to this project.

Many mentors, peers, and colleagues were vital to the process of completing this project and my graduate training. Drs. Kennard, Saine, Van Hoose, Wetherington, and Hoenig have been models of the type of mentor and supervisor that I aspire to be in the future. I am also grateful for the help and support of Andrea Croom who was an efficient and willing contributor to this project. I would like to thank my classmates who taught me about life during some of the most formative years of my life. They showed me how to live and laugh without limits and to have "work-life balance" in the midst of overwhelming demands. I would particularly like to thank Kristi Baker and OJ Benitez whose help and friendship *sustained* me through these past 4 years.

It goes without saying that I would not have made it to this point without the support of my loving family and friends. I am thankful for my parents who encouraged me to step outside of the box to pursue a "non-Asian" career that brought me happiness. It has been through their support and sacrifices that this day has come to fruition. My sister, Ellen, has been my confidante, cheerleader, and consistent source of humor, which helped me laugh through the tears. To my

friends who stuck by as I knowingly neglected them in order to finish this project, I am grateful for your loyalty and understanding in this process. Lynn and I-fang, you both have been kindred spirits even before we knew what we would be when we "grew up."

I would like to thank my husband, Jason, for witnessing this process and finding the strength to encourage, challenge, and keep me grounded despite the demands of medical school and residency. I could not have found a better partner for this journey and I am so grateful that the completion of this project will allow us to be together again. Last, but certainly not least, I would like to thank my Adonai, Jesus Christ, for his redemption and for being the one constant throughout my life. I am thankful for His vision, purpose, and strength, which have carried this to completion.

RACE AND NEIGHBORHOOD SES DIFFERENCES IN THE DEVELOPMENTAL TRAJECTORIES OF TYPE 1 DIABETES MANAGEMENT OF CAUCASIAN AND ETHNIC MINORITY YOUTH

JENNY TZU-MEI WANG, Ph.D.

The University of Texas Southwestern Medical Center at Dallas, 2009

DEBORAH J. WIEBE, Ph.D., MPH

Pediatric diabetes is a chronic illness that significantly impacts the lives of children and adolescents and their families. Poor metabolic control increases the risks for severe long-term consequences with debilitating effects in adulthood. Adolescence is a particularly difficult period of time for Caucasian youth as they

evidence characteristic declines in metabolic control. There is some cross-sectional evidence that racial disparities in pediatric diabetes exist. However, only one study has evaluated racial disparities longitudinally and no studies have evaluated these differences across the critical period of adolescence. The aims of this retrospective study were to replicate age-related declines in metabolic control in a sample of African American youth and to characterize racial differences in the developmental trajectories of metabolic control between African American and Caucasian youth. This study also aimed to evaluate whether race effects remained beyond neighborhood SES. The sample consisted of 162 Caucasian and African American subjects matched on gender and age seen at the Children's Medical Center Dallas endocrinology outpatient clinic during 2007. Retrospective medical record reviews were conducted for sociodemographic information and retrospective health data. Neighborhood SES variables were obtained through publicly available census databases. This retrospective investigation revealed age-related declines in metabolic control regardless of race. African American youth had higher HbA1c levels compared to Caucasian counterparts throughout ages 10 through 18. However, African American and Caucasian youth evidenced parallel trajectories (similar rates of change) in metabolic control across this critical period. Race retained unique explanatory effects beyond that of neighborhood SES while family structure was not a predictive variable. These results suggest that African American youth

experience significant risks prior to adolescence that place them at poorer levels of metabolic control and these risks are maintained across adolescence. Adolescence may be an equally risky period for African American and Caucasian youth, highlighting the need for further research on how African American youth and their families cope with diabetes. These results provide evidence that adolescence may be a critical period for both African American and Caucasian youth and unique interventions should be developed to prevent declines in metabolic control during adolescence in both racial groups.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE

INTRODUCTION

Statement of the Problem

Type 1 diabetes management declines during adolescence presumably because adolescence changes numerous aspects of the diabetes management routines that had been previously established. Successful treatment of type 1 diabetes depends upon the effective coordination of several intricate behaviors. As such, children and adolescents and their families are expected to adjust their lifestyles, routinely administer insulin, and engage in education to promote self-management (American Diabetes Association [ADA], 2007). The developmental period of adolescence often disrupts the well-rehearsed and established routines that may have developed in early childhood. Patients typically experience significant declines in metabolic control as they begin the transition through adolescence (Auslander, Thompson, Dreitzer, White, & Santiago, 1997; Goldston, Kovacs, Obrosky, & Iyengar, 1995; Povlsen, Olsen, & Ladelund, 2005; Weissberg-Benchell et al., 1995; Wysocki et al., 1996). Presumably, the rapid changes in physiological, psychological, and relational factors such as pubertal status, autonomy development, peer relationships, and parental involvement that occur during adolescence explain these declines in metabolic control (Amiel, Sherwin, Simonson, Lauritano, & Tamborlane, 1986; Hains, Berlin, Davies,

Parton, & Alemzadeh, 2006; Mansfield, Addis, Laffel, & Anderson, 2004; Thomas, Peterson, & Goldstein, 1997; Wysocki et al., 1996).

Difficulties of diabetes management during adolescence have been documented almost exclusively in Caucasian populations, although there is reason to believe there may be racial differences in these trajectories. Current research provides evidence that significant racial differences in diabetes management exist between African American and Caucasian youth and adults. Most notably, African American youth have been shown to have poorer metabolic control compared to Caucasian counterparts in cross-sectional studies (Auslander et al., 1997; Chalew et al., 2000; Delamater, Albrecht, Postellon, & Gutai, 1991; Delamater et al., 1999). However, no studies have examined the developmental trajectories of diabetes management in African American youth across the critical period of adolescence (age 10 through 18), during which the most significant declines in metabolic control appear in Caucasian samples. As a result, it is currently unknown whether the characteristic declines in metabolic control during adolescence that are well established in Caucasian youth can be generalized to African American counterparts.

Socioeconomic position and racial status are often correlated in the United States, and both variables are associated with health and child development. Specifically, significant negative outcomes are associated with lower socioeconomic status in multiple areas of children's lives such as physical health,

emotional well-being, and cognitive development (Bradley & Corwyn, 2002). Differentiating between the independent effects of socioeconomic status and race are important given that African Americans are disproportionately represented in lower socioeconomic (United States Census Bureau [USCB], 2005) and less educated groups (USCB, 2004). Furthermore, identifying the unique effects of socioeconomic and racial status provide more refined hypotheses regarding ways in which health disparities in diabetes management may be reduced. Given the associations between racial and socioeconomic status, certain racial groups may experience disproportionate risks for disruptions in normal child developmental processes that may also disrupt the acquisition and implementation of skills required for effective diabetes management.

Growing research in developmental psychology consistently highlights the importance of understanding psychological and health factors within a developmental context. Chen (2004) suggests that the impact of socio-demographic variables on health outcomes may vary based on the developmental stage of the child or adolescent. Characterizing the developmental trajectories of diabetes management between African American and Caucasian youth provides critical information for developing plausible hypotheses about the mechanisms that may initiate and maintain health disparities between these two groups. For example, if racial disparities in metabolic control begin in childhood rather than adolescence in African American children, this trajectory would suggest the

importance of childhood pathways through which racial or socioeconomic differences in metabolic control begin and persist.

The present study utilized a medical record review technique to examine different developmental trajectories of metabolic control in African American and Caucasian youth across ages 10 through 18. These developmental trajectories allowed us to determine whether we could replicate age-related declines in metabolic control that are consistently found in Caucasian youth in a sample of African American children and adolescents. Furthermore, we aimed to evaluate whether African American and Caucasian youth entered adolescence with different levels and/or displayed different rates of change in metabolic control across ages 10 to 18. Finally, we determined whether the effects of racial status on metabolic control remained after neighborhood socioeconomic status was statistically controlled. By describing the differential trajectories of metabolic control in African American versus Caucasian youth, we aimed to identify plausible hypotheses regarding the mechanisms that explain health disparities in pediatric diabetes. Such hypotheses can guide future research, which may aid in the development and implementation of interventions at optimal time periods to reduce racial disparities and enhance health outcomes in adulthood.

CHAPTER TWO

REVIEW OF THE LITERATURE

Overview of Type 1 Diabetes

Type 1 diabetes mellitus (T1DM) is a chronic illness that results when beta cells within the pancreas are destroyed due to an autoimmune response (Atkinson & Maclaren, 1994). Once beta cells have been destroyed, the body is no longer able to produce insulin, a hormone that is responsible for the breakdown of glucose stores in the bloodstream. Persistently high levels of glucose in the bloodstream have been associated with severe long-term consequences such as retinopathy, neuropathy, and nephropathy caused by microvascular and macrovascular damage to nerves and organs (Porte & Schwartz, 1996). As a result, a main treatment goal for type 1 diabetes is to restore appropriate levels of glucose in the body through the administration of synthetic insulin.

There are several different criteria used for the diagnosis of type 1 diabetes mellitus (ADA, 2007). Patients characteristically present with acute symptoms that include excessive urination and thirst, stomach pain, dizziness, visual disturbance, and weight loss not explained by behavioral or other physiological causes (Scott, Smith, Cradock, & Pihoker, 1997). A notable feature during initial diagnosis may include diabetic ketoacidosis (DKA), as it often precipitates hospitalization. Although the exact frequency of DKA in newly diagnosed cases is unknown, it has been estimated that approximately 15-27% of new diagnoses of

TIDM in children present with some level of DKA (Soliman, Salmi, & Asfour, 1997). The clinical and physiological presentations of type 1 diabetes are often quickly recognized, as symptoms of T1DM arise acutely prior to diagnosis, but require lifelong management once identified.

Prevalence and Incidence

In 2003, 18.2 million people were estimated to have diabetes, reflecting approximately 6.3% of the United States population (Centers for Disease Control and Prevention [CDC], 2003). Approximately 300,000 to 500,000 individuals of all ages are reportedly living with type 1 diabetes (Laporte, Matsushima, & Chang, 1995) with 120,000-150,000 cases under the age of 19 (Search for Diabetes in Youth Study Group, 2007). Of the 30,000 new cases of type 1 diabetes diagnosed each year, more than 13,000 are in young people under age 19. Type 1 diabetes is still the most often diagnosed form of diabetes in children and adolescents, despite concerns of growing rates of type 2 diabetes with rising obesity rates (Dabelea et al., 2007; Search for Diabetes in Youth Study Group, 2006).

A worldwide study on type 1 diabetes demonstrated that the incidence of type 1 diabetes in 103 study centers all over the world was increasing at an average rate of 2.4% per year between 1990 through 1999 (DIAMOND Project Group, 2006), consistent with rising rates in the United States and Europe (Dabelea et al., 2007; EURODIAB ACE Study, 2000; Laporte et al., 1995).

There is evidence in the US that racial differences exist in the prevalence of T1DM, which has been hypothesized to be due to genetic and social factors influencing the disease (Lipman, Chang, & Murphy, 2002; Oldroyd, Banerjee, Heald, & Cruickshank, 2005). As overall incidence rates rise, the number of ethnic minority children with type 1 diabetes can also be expected to rise. Therefore, understanding diabetes management in ethnic minority youth should increase in importance as well.

Treatment Recommendations

The treatment of type 1 diabetes involves a collection of highly coordinated tasks that must be performed several times a day. Children and adolescents are required to monitor their blood glucose levels 3-4 times per day, inject insulin several times a day, and become cognizant of their dietary intake (ADA, 2007). Successful achievement of these tasks requires a unified effort between parents and children or adolescents (Anderson, Auslander, Jung, Miller, & Santiago, 1990). The primary treatment goal of the abovementioned behaviors is to prevent hyperglycemia and its associated acute symptoms such as dizziness, blurred vision, and abdominal pain and long-term consequences such as retinopathy, neuropathy, and nephropathy (Diabetes Control and Complications Trial Research Group [DCCT], 1993).

Blood glucose monitoring is often seen as the primary method by which children and adolescents and their parents are able to self monitor metabolic

control. The recommended target ranges for mean blood glucose in school-age children is between 90 and 180 and for adolescents between 90 and 130 before meals (ADA, 2007). Given that appropriate insulin administration prior to meals facilitates the breakdown of glucose, patients are able to exert a considerable impact on their blood glucose levels throughout the day. As such, blood glucose monitoring serves as a marker for metabolic control and identifies when patients are either entering hyperglycemia (high levels of glucose) or hypoglycemia (low levels of glucose). Blood glucose monitoring is a modifiable behavioral component in diabetes management and is often the target for interventions aiming to improve metabolic control (Ellis et al., 2007; Wysocki et al., 2006).

Another index by which families and the health care team can discern treatment effectiveness is glycosylated hemoglobin A1c (HbA1c). HbA1c reflects the amount of glucose that has bonded with plasma hemoglobin over the previous several months, forming a glycated hemoglobin (Nathan, Singer, Hurxthal, & Goodson, 1984). Individuals with poorly controlled diabetes typically have higher levels of glucose in their bloodstream, which binds with the hemoglobin, resulting in higher glycated hemoglobin levels. As such, higher HbA1c values reflect poorer metabolic control. Similar to the usefulness of blood glucose monitoring in tracking daily fluctuations in metabolic control, HbA1c allows medical teams to characterize metabolic control over the previous 2-3 months. Improvements in daily glucose monitoring have been shown to be

associated with lower HbA1c levels (Koenig et al., 1976), highlighting the utility of this assessment measure. The standards of care recommendations by the American Diabetes Association (2007) suggest that patients should receive HbA1c assessments at least twice a year or more, especially if treatment recommendations have been altered. It is recommended that children and adolescents with adequate control should have less than 7.5% to 8% HbA1c levels.

Administration of synthetic insulin is the primary treatment modality for T1DM. Insulin doses are often based on the amount of food intake for each meal, which require the child or adolescent as well as his or her parents to persistently monitor dietary carbohydrate intake. In addition, the ADA (2007) cautions that children and adolescents should be monitored for both hyperglycemia *and* hypoglycemia, as young children may not be as aware of sudden declines in blood glucose levels. Therefore appropriate insulin levels serve to combat the consequences of persistent hyperglycemia, but too high of insulin levels may trigger immediate consequences of hypoglycemia. Additional precautions should be taken as children with type 1 diabetes exhibit hypoglycemia symptoms that may vary from those found in adults (McCrimmon, Gold, Deary, Kelnar, & Frier, 1995). Consequently, physicians have the careful task of balancing the need to maintain recommended blood glucose levels as well as preventing the negative consequences of severe hypoglycemia.

Consequences of Poor Metabolic Control

Persistent hyperglycemia results in microvascular and macrovascular complications that lead to the development of severe long-term consequences. The Diabetes Control and Complications Trial (1993) demonstrated that intensive management of blood glucose levels could reduce the risk for the development of diabetes-related consequences. Complications of poor metabolic control have debilitating long-term effects for patients with type 1 diabetes such as loss of vision and amputation of limbs. Prevention of these consequences may best be initiated in childhood and adolescence, as health behaviors developed early in life have influences on health outcomes of later life (Seiffge-Krenke & Stemmler, 2003).

The Diabetes Control and Complications Trial (1993) evaluated the long-term effectiveness of intensive management, which included three or more insulin injections per day versus conventional management that required only one or two injections each day. They found that those who intensively managed their blood glucose levels had lower HbA1c and evidenced a reduction in incidence of retinopathy by 50% compared to their conventional therapy counterparts. Similar reductions in risk for neuropathy and nephropathy were found in those who administered insulin at least 3 times per day. These findings provided evidence and rationale for implementing more intensive management programs for children, teens, and adults with type 1 diabetes.

In addition to the costs related to physical wellbeing and overall quality of life, significant economic costs are associated with poorly controlled diabetes. It has been estimated that approximately $132 billion are lost each year for health care costs and reduced productivity of workers with diabetes (ADA, 2003). This amount likely underestimates overall costs, which include the psychological and emotional difficulties that accompany the illness progression. An estimated 20% of all medical expenditures in the United States are for patients with all forms of diabetes, which is a staggering amount given that people with diabetes only comprise of 6.3% of the population in the United States. These economic costs highlight the significant burdens that children, families, and adults with diabetes must face in order to maintain their physical health and well-being. Consequently, early prevention and identification of barriers to optimal metabolic control need to be researched and addressed in order to reduce the national and personal burdens associated with having diabetes.

Diabetes in Adolescence

The rapid and numerous changes that occur during adolescence complicate the difficulties inherent in coordinating multiple intricate tasks required for effective diabetes management. There is evidence that adolescents exhibit poorer adherence and metabolic control compared to younger children (Anderson, Ho, Brackett, Finkelstein, & Laffel, 1997; Auslander et al., 1997; Delamater et al., 1991; Goldston et al., 1995; Johnson et al., 1992; Weissberg-

Benchell et al., 1995; Wysocki et al., 1996). Declines in metabolic control are occasionally associated with behavioral changes such as missing insulin injections or failing to monitor blood glucose levels several times per day (Altobelli et al., 2000).

It is important to understand metabolic changes during adolescence from a developmental framework, as several salient factors during this period have been hypothesized to contribute to noticeable deteriorations in metabolic control. Firstly, adolescence is a developmental period during which teenagers experience visible and subtle physiological changes. The initiation of puberty has been associated with insulin resistance in adolescents. Teenagers occasionally continue to have high glycosylated hemoglobin levels despite receiving higher doses of insulin than younger children (Amiel et al., 1986; Bloch, Clemons, & Sperling, 1987). The onset of heightened insulin resistance during adolescence may result in increased frustration with the treatment regimen, as adolescents may feel their metabolic control is no longer contingent upon their efforts or adherence behaviors.

The initiation of puberty is also associated with psychological and emotional changes. It has been found that the onset of puberty may exert significant changes in self-perceptions, behavioral issues, and depressive symptoms (Dorn, Susman, & Ponirakis, 2003; Nolen-Hoeksema & Girgus, 1994). The greater risks for depressive symptoms during puberty, especially in females

(Graber, Brooks-Gunn, & Warren, 2006), may place adolescents at greater risk for reduced adherence and diabetes complications that require hospitalization (Korbel, Wiebe, Berg, & Palmer, 2007; Stewart, Rao, Emslie, Klein, & White, 2005). The onset of physical and psychological changes that occur in adolescence suggest that it is a critical period during which teens must learn to adapt their self-management behaviors as well as simultaneously cope with the emotional difficulties of adolescence.

Adolescence is also a time during which teenagers and their parents experience changes in their relationship dynamics. Parents who have older teenagers tend to withdraw their involvement in diabetes tasks (Mansfield et al., 2004), as they may perceive physiological changes as signals to relinquish control and perhaps prematurely give their adolescent increased autonomy (Palmer et al., 2004). However, adolescents who demonstrate high levels of autonomy do not necessarily exhibit better adherence behaviors (Wysocki et al., 1996), as they may alienate parental involvement during a time in which they may still require assistance from parental figures. Cohesive and supportive families are associated with better metabolic control (Cohen, Lumley, Naar-King, Partridge, & Cakan, 2004), suggesting that despite the needs for increased autonomy, adolescents continue to benefit from parental relationships when they remain involved and supportive.

Continued parental involvement during adolescence has been consistently found as an important factor in adherence behaviors (Anderson et al., 1997; Anderson et al., 2002), although not all styles of parental involvement are equally effective (Wiebe et al., 2005). Parental involvement has been strongly implicated to impact metabolic control such that interventions to promote involvement have been developed to reduce its decline during adolescence (Anderson, Brackett, Ho, & Laffel, 1999). In addition, child perceptions of maternal involvement may be particularly important as they may lead to negative emotions especially when parents are perceived as controlling or critical (Berg et al., 2007; Butler, Skinner, Gelfand, Berg, & Wiebe, 2007; Lewin et al., 2006). Maternal psychological characteristics may also influence metabolic control in children and adolescents (Cameron, Young, & Wiebe, 2007), highlighting the intimate and complex relationship that adolescents have with their parents especially when coping with diabetes.

Finally, peer relationships often undergo significant changes during adolescence, as they may become a higher priority for teens. These relationships become increasingly influential such that adolescents with diabetes may choose to neglect adherence behaviors in order to be accepted by peers (Thomas et al., 1997). In addition, peer perceptions may become more important during adolescence. They may spend less time with friends or become less adherent with their medical regimen because they negatively anticipate problems between peers

and their diabetes responsibilities (Hains et al., 2006). As a result, adolescents experiencing significant changes in their social relationships may inadvertently reduce their metabolic control because they want to "fit in" or be accepted by peers.

All the factors mentioned above emphasize the importance of understanding the trajectories of metabolic control in African American and Caucasian youth within the context of adolescent development, as they provide researchers with plausible hypotheses regarding mechanisms that contribute to these changes. Specifically, reductions in metabolic control during adolescence would suggest that adolescent-related mechanisms might be important in explaining the deteriorations in metabolic control. In contrast, deteriorations that occur prior to adolescence would suggest that child-related pathways might be important in explaining changes in metabolic control between the different racial groups.

Diabetes in African American Youth

Research has not documented a comparable decline in metabolic control across adolescence in African American youth, and it is possible that African American teens may exhibit differential trajectories of metabolic control across this developmental period. Cross-sectional evidence consistently supports that African American youth have poorer metabolic control compared to Caucasian counterparts. This present study built upon the only longitudinal study that

evaluated racial differences in metabolic control by examining the important effects of adolescent development on racial differences in diabetes management.

Cross-sectional Evidence

Potentially because type 1 diabetes disproportionately affects Caucasian youth, relatively few studies have examined factors associated with diabetes management for African American children and adolescents. Of the few studies conducted, however, a consistent finding is that African American youth have poorer metabolic control compared to Caucasian counterparts (Auslander et al., 1997; Chalew et al., 2000; Delamater et al., 1991; Delamater et al., 1999; Patino, Sanchez, Eidson, & Delamater, 2005). Delamater, Albrecht, Postellon, and Gutai (1991) found that, despite receiving higher insulin doses, African American children and adolescents demonstrated poorer metabolic control and more frequent hospitalization for DKA compared to Caucasian youth. Delamater et al. (1999) also found that African American youth had an almost four-fold higher risk of being classified in the poorly controlled group compared to Caucasian children and teens. Finally, there is evidence of disproportionate risks for physical complications (i.e., blindness, renal failure, death) as African American youth enter adulthood (Arfken, Reno, Santiago, & Klein, 1998; Bosnyak et al., 2005; Lipton, Good, Mikhailov, Freels, & Donoghue, 1999; Tull & Barinas, 1996).

Socioeconomic status has been hypothesized to explain racial differences in metabolic control. Auslander and colleagues (1997) found that lower parental education and financial resources partially accounted for racial differences in adherence levels among Caucasian and African American youth. The significant associations between indices of socioeconomic status and metabolic control are important for understanding racial differences, as African American families are most often categorized in groups with the lowest socioeconomic status and worst glycemic control compared to Caucasian families (Davis et al., 2001). Several studies have found that SES does not fully explain racial differences in metabolic control across African American and Caucasian groups (Chalew et al., 2000; Delamater et al., 1991; Hanson, Henggeler, & Burghen, 1987). Therefore, current understandings of the mechanisms that may explain racial differences in metabolic control are unclear and understudied in African American populations.

Racial disparities in metabolic control have also been attributed to differences in family structure (i.e., single-parent or two-parent households) found in Caucasian and African American youth. African American adults are less likely to be married compared to any ethnic group (USCB, 2003) and African American youth with diabetes more often come from families with a single parent (Auslander et al., 1997; Delamater et al., 1991; Thompson, Auslander, & White, 2001). In addition, children and adolescents from single-parent households have poorer metabolic control than those in two-parent households (Auslander et al.,

1997; de Wit et al., 2007; Hoey et al., 2001). Thompson and colleagues (2001) confirmed that single parenthood continued to predict metabolic control after controlling for race and age. These findings suggest that race and family structure have at least partially independent and potentially additive effects on risk, which need to be further evaluated to appropriately inform the development of interventions to reduce health disparities.

In sum, cross-sectional data provide evidence that African American youth have poorer metabolic control than their Caucasian counterparts. The current literature points to many complex factors that may explain racial differences in diabetes management cross-sectionally. Specifically, socioeconomic status and family structure appear to exert differential effects on diabetes management across African American and Caucasian youth. This present study aimed to extend upon the cross-sectional literature by evaluating racial differences in metabolic control across the critical period of adolescence, during which the most significant deteriorations of metabolic control occur in Caucasian youth.

Longitudinal Evidence

Differences in metabolic control between African American and Caucasian youth have been most consistently found in cross-sectional studies. To date, only one study has attempted to understand racial differences in metabolic control longitudinally (Frey, Templin, Ellis, Gutai, & Podolski, 2007). Frey and colleagues evaluated the longitudinal trajectory of racial differences in metabolic

control from time at diagnosis to five years post-diagnosis. They found that declines in metabolic control began soon after diagnosis for both African American and Caucasian youth. However, the declines in metabolic control among African American youth were almost twice as fast as those in Caucasian youth. Interestingly, the influence of racial status on metabolic control was no longer significant when family structure was entered into the analyses, suggesting a plausible mechanism for racial differences in metabolic control.

The current study built on Frey et al.'s (2007) longitudinal work by examining racial differences in metabolic control across the adolescent years. Frey et al. demonstrated how quickly racial disparities emerge after diagnosis, but did not address issues of adolescent development. Their study focused on the first five years post diagnosis, and their convenience sample consisted of 71 participants (36 Caucasian, 35 African American) between the ages of 7 and 19, which likely did not provide sufficient numbers of youth in the different age groups to adequately measure developmental changes.

Disparities in Adulthood

There are substantial and clinically significant implications for understanding the factors that may be related to the onset and maintenance of racial differences in metabolic control between African American and Caucasian youth. Racial disparities in health outcomes for type 1 diabetes extend into adulthood with grave consequences (Gary, McGuire, McCauley, & Brancati,

2004; Summerson, Konen, & Dignan, 1992; Tull & Barinas, 1996). Aside from poorer metabolic control (Summerson et al., 1992), African American adults also have a 2.5 fold increase in risk of mortality compared to Caucasian adults (Tull & Barinas, 1996), with African American females conferring the greatest risk. Commencing research on racial disparities in metabolic control during childhood and adolescence is a logical step given that health behaviors developed early in life may impact later life (Seiffge-Krenke & Stemmler, 2003) and may be a point at which risks for serious consequences can be prevented.

A Developmental Framework

The present study draws upon on a developmental framework proposed by Chen and colleagues (2004) to understand racial disparities in metabolic control across adolescence. According to this framework, evaluating how sociodemographic variables (i.e., race or socioeconomic status) are associated with health outcomes across different developmental periods provides significant information about how these variables impact health (Chen, Martin, & Matthews, 2006; Chen, Matthews, & Boyce, 2002). From a developmental framework, for example, it is important to understand whether African American and Caucasian youth show different patterns of deterioration in metabolic control across the adolescent years because such "developmental trajectories" provide information about likely mechanisms for racial disparities in diabetes management. This approach can also identify critical periods

to inform the timing of interventions that may result in optimal improvements in metabolic control and reductions in racial disparities in diabetes management.

In this section, several hypothetical developmental trajectories are proposed to demonstrate how this model can be applied in the context of diabetes management in African American adolescents. These hypothetical trajectories are not intended to be prescriptive or comprehensive; each is plausible based on existing data, and other trajectories are possible. In each case, (a) the basic pattern of associations between child age and metabolic control across African American and Caucasian youth are described, and (b) relevant research and theory that would support such a pattern are discussed.

Assumptions

In developing the following models, several assumptions were made that influenced the hypothetical developmental trajectories of metabolic control. First, HbA1c was assumed not to rise above 14%. This assumption limited how high the HbA1c levels could rise across adolescence for both African American and Caucasian youth in the hypothetical trajectories. This limit also reflected clinic standards of how HbA1c values are measured, as this clinic only measures HbA1c up to 14%, reflecting the worst level of metabolic control. Second, racial disparities were assumed to emerge shortly after diagnosis and to continue through adolescence and adulthood (Delamater et al., 1991; Frey et al., 2007; Lipton et al., 1999; Summerson et al., 1992). Consequently, African American youth were assumed in

the models to have poorer metabolic control than Caucasian youth at all ages. Third, the components of adolescent development were assumed to be similar between African American and Caucasian youth. That is, the biological and psychosocial aspects of pubertal development, autonomy development, and changing peer and parent-child relationships were assumed to influence developmental outcomes in both African American and Caucasian youth. However, these components may not begin at exactly the same time point or work in exactly the same way across different race or SES groups and may play out in different ways in the context of managing a serious illness such as type 1 diabetes.

The following hypothetical models focused on the developmental stage of adolescence between the ages of 10 through 18. This age range included the onset of puberty, captured the critical period of ages 12 through 15 during which active autonomy development occurs, and provided information on diabetes management during later adolescence when youth are more independently responsible for diabetes tasks. This age range also reflected the period during which deterioration in metabolic control has been documented in Caucasian youth (Anderson et al., 1997; Weissberg-Benchell et al., 1995; Wysocki et al., 1996). Furthermore, specific interventions have been developed for this age range to prevent declines in metabolic control during adolescence (Ellis et al., 2007; Wysocki et al., 2006).

Hypothetical Models

Childhood Risk Model. The first model proposed is the Childhood Risk Model (see Figure 1). This model suggests that African American youth enter adolescence already displaying poorer metabolic control than Caucasian youth and that persists throughout adolescence. Therefore, it is assumed that the risk factors experienced in childhood rather than adolescence place African American youth at greater risk for poorer metabolic control compared to their Caucasian counterparts.

Several childhood-related pathways could converge to support the Childhood Risk Model. Some of these pathways may include child poverty status and early diabetes education. Individuals living in poverty are most often exposed to higher risk contexts, disrupted family lives, and fewer resources such as financial, environmental, and social capital. These environmental disadvantages have detrimental consequences for health (Papas et al., 2007; Taylor, Repetti, & Seeman, 1997) and child development (Caughy, Nettles, O'Campo, & Lohrfink, 2006; Repetti, Taylor, & Seeman, 2002). Childhood poverty status has been associated with detrimental health outcomes because it places chronic stress on families and has been associated with actual biological changes (i.e., immune responses) (Chen et al., 2006). Within the context of diabetes, limited financial resources may result in chronic stress for parents leading to ineffective parental involvement and may also increase the physiological risks for poor diabetes management.

Diabetes education is a crucial part of initial diagnosis because several new and unique tasks are required of newly diagnosed children and adolescents. As a

result, parents often play an important role in learning the diabetes tasks and educating their children and adolescents at home. Most of these educational experiences are conducted in clinics or hospitals, and are highly dependent upon adequate clinic attendance by parents and children and adolescents. African American families are more likely to miss clinic appointments than Caucasian families (Delamater et al., 1991). Infrequent attendance of clinic visits may be attributed to difficulties with transportation, few resources (e.g., child care for other children, social support), and inability to taking time off from employers. As a result, newly diagnosed African American children may be at greater risk if their parents do not receive adequate diabetes education shortly after diagnosis. Compared to Caucasian families, African American families may not comparably gain adequate skills and knowledge required to effectively manage their diabetes, resulting in poor metabolic control that persists into adolescence.

The impact of socioeconomic status on this model may be particularly salient given the relationships between childhood poverty status and several areas of normal development. Furthermore, it is possible that the risks associated with childhood poverty may be exacerbated by the physical and emotional demands of chronic illnesses like diabetes. If this hypothetical model emerges in the findings, it will be particularly important to covary socioeconomic status to evaluate whether it may play an explanatory role for racial differences in metabolic control.

Differential Adolescent Risk Model. The second hypothesized model is the Differential Adolescent Risk Model (see Figure 2). This model suggests that African American and Caucasian youth both experience adolescent risks that impact metabolic control, but these occur at different periods of adolescent development. As a result, one racial group experiences a significant deterioration (rise in HbA1c) in metabolic control earlier than the other racial group. Thus, the differential adolescent risk model hypothesizes that differences in the timing of adolescent development result in differential developmental trajectories between African American and Caucasian youth.

Several factors could support a differential risk model for African American versus Caucasian youth. Literature indicates there are differences in the timing of pubertal onset between African American and Caucasian adolescents. In a national sample of 4,263 ethnic minority and Caucasian children and adolescents between the ages of 8 and 19, Sun et al. (2002) reported that African American youth entered puberty earlier than Caucasian teens. In some cases, especially in girls, pubertal onset was over a year earlier for features related to sexual maturity (i.e., pubic hair, breast and genital development), which may act as visual cues to parents, teachers, and peers that an adolescent has entered puberty.

Puberty exerts significant biological and social influences on diabetes management. A 32% decline in insulin sensitivity was found in African American and Caucasian youth transitioning from Tanner stage I to III or IV (Goran & Gower,

2001). Although there were not enough subjects to thoroughly evaluate racial differences in declines in insulin sensitivity, preliminary findings suggest that declines in insulin sensitivity may be stronger in African American youth compared to Caucasian youth. That is, earlier pubertal onset in African American adolescents compared to Caucasian youth may alter the way their bodies use insulin and may change how the illness is managed.

Visible pubertal changes such as breast development, facial hair, and growth spurts provide social signals for parents and adolescents to alter their relationships and interactions (Hauser et al., 1985; Sagrestano, McCormick, Paikoff, & Holmbeck, 1999; Steinberg, 1981). Physical changes in African American females prompt significant changes in mother-daughter interactions regarding opposite-sex contact (O'Sullivan, Meyer-Balhburg, & Watkins, 2000). These signs of physical maturity may also alter parent and child relationships in ways that lead children to assume independent responsibility for diabetes-related activities. Cues of physical maturity may encourage parents to engage in different patterns of interaction such as withdrawing involvement that result in disruptions of metabolic control (Palmer et al., 2004), as adolescents often still require parental involvement to effectively cope with diabetes (Wysocki et al., 1996). Consequently, if African American youth display signs of pubertal development earlier than their Caucasian counterparts, African American parents may prematurely encourage independence in diabetes tasks, which then may be associated with earlier deteriorations in metabolic control.

Although it is less expected that SES would play a major role in this model, it is possible from an ecological perspective that families living in high-risk environments (i.e., low SES communities) may be more susceptible to negative health outcomes moderated by increased family conflict. Low SES families typically live in "high-risk" neighborhoods with greater levels of crime, exposure to violence, and drug use. It is possible that within these high-risk contexts, adolescence becomes a particularly difficult time as teens are spending less time at home, which may prompt parents to change rules and limits to protect them from perceived environmental harm. These changes in parental limitations and rules with concurrent increases in autonomy seeking in adolescents may result in greater family conflict. Higher levels of family conflict are associated with increased risky behaviors (i.e., sexual debut) (McBride, Paikoff, & Holmbeck, 2003), which may also include risk behaviors associated with neglecting diabetes management responsibilities.

African American families may experience greater vulnerability to the negative consequences of low SES/high-risk context, resulting from heightened family conflict that may be associated with earlier deterioration of metabolic control in African American youth. African American families are disproportionately represented in low SES groups and are more likely to live in higher risk environments. African American teens begin to exhibit outward signs of puberty earlier than Caucasian youth (Sun et al., 2002), which may prompt earlier changes in parenting behaviors, as African American parents have more "hot" discussions and

conflict with early maturing versus on-time or later maturing adolescents (Sagrestano et al., 1999). Furthermore, African American parents tend to exercise more control over their children and adolescents compared to Caucasian parents (Bulcroft, Carmody, & Bulcroft, 1996), which may increase conflict in these families. Heightened family conflict at an earlier period of adolescence in African American families can partially explain why African American youth may experience a differential trajectory of diabetes management, as family conflict has been found to be associated with poor metabolic control (Anderson et al., 1999; Anderson et al., 2002; Lewin et al., 2006).

Parallel Deterioration Model. The hypothetical Parallel Deterioration Model (see Figure 3) suggests that African American youth may demonstrate comparable rates of decline in metabolic control across adolescence as do Caucasian youth, but at each point in time display poorer management (i.e., higher HbA1c) (Anderson et al., 1997; Delamater et al., 1991; Delamater et al., 1999; Weissberg-Benchell et al., 1995). If this model effectively characterizes the trajectory of African American youth, then previous research conducted on Caucasian youth regarding the disruptions of metabolic control during adolescence may be more directly relevant for African American samples. The current literature on the factors associated with deteriorations in metabolic control in Caucasian youth has been discussed earlier in detail. However, the most notable findings that may explain similar trajectories of

metabolic control in African American and Caucasian youth based on this model are briefly reviewed.

Despite findings that African American and Caucasian parents engage in different patterns of providing independence to their children (Bulcroft et al., 1996), there is evidence that variations in parenting practices between races are much smaller when socioeconomic status is controlled (Julian, McKenry, & McKelvey, 1994). Within the context of diabetes, children and adolescents are required to engage in repetitive monitoring and intimate interaction with their parents throughout the day. Furthermore adolescence is a period during which autonomy and independence are often negotiated with parents. More involved parenting has been found to be effective in younger children (Anderson et al., 1997), but may result in negative outcomes for adolescents when autonomy development is a priority. Therefore, African American and Caucasian parents must deal with the difficulties of providing their adolescents with increased independence over diabetes tasks while risking deterioration of metabolic control.

African American and Caucasian parents may enter the period of adolescence struggling to adapt their parenting practices to the needs of their adolescents (Gutman & Eccles, 2007), but they both must also deal with changes in how adolescents may perceive their behaviors during this developmental period. Literature on Caucasian youth with diabetes suggests that when children and adolescents perceive their parents' behaviors as controlling or intrusive these perceptions are associated with

negative psychological outcomes (Berg et al., 2007; Butler et al., 2007; Wiebe et al., 2005). Concurrent changes in parenting behaviors and adolescents' perceptions of these behaviors may result in increased conflicts in the home.

Parent-child conflict is a normal part of adolescent autonomy development in African American youth similar to Caucasian adolescents (Smetana & Gaines, 1999). Longitudinal evidence suggests that there is little variation in the number or frequency of parent-child conflicts in African American families across early to middle adolescence (Smetana, Daddis, and Chuang, 2003). Furthermore, African American youth tended to resolve conflicts by "giving in" to their parents across this period, which may reflect the unique collectivistic values of African American culture. Consequently, parent-child conflict appears to be a pervasive part of adolescence in both African American and Caucasian families. African American and Caucasian parents must learn to successfully adjust to increased parent-child conflicts especially within the context of diabetes, as family conflict negatively impacts adherence over time (Hauser et al., 1990).

During adolescence, teenagers reduce their time spent with parents, which may place them at greater risk for negative behaviors (Barnes, Hoffman, Welte, Farrell, & Dintcheff, 2007; Larson & Richards, 1991). Studies on Caucasian samples suggest that concerns about peer perceptions related to diabetes management increase stress, which significantly impacts metabolic control (Hains et al., 2006). Although similar concepts have not yet been explored in African American youth,

there is evidence that peer associations influence African American teens. African American adolescents' peer relationships and attachment to school are significantly related to antisocial behaviors (Joseph, 1995). In addition, African American adolescents' perceptions of peer engagement in drug use predicted their level of involvement with drugs 6 and 24 months later (Stanton et al., 2002). These findings suggest that African American and Caucasian youth may be similarly affected by significant peer influences during adolescence, which may alter their adherence behaviors and metabolic control.

The presence of psychiatric disorders has been known to be more prevalent in pediatric type 1 diabetes populations compared to the general population, with higher rates of depression during adolescence (Kovacs, Goldston, Obrosky, & Bonar, 1997). Specifically, depression has been found to be associated with reductions in adherence, higher rates of hospitalization, and diabetes complications (Korbel et al., 2007; Stewart et al., 2005). There are no known differences in prevalence of Major Depressive Disorder between African American and Caucasian populations (American Psychiatric Association, 2000). Consequently, it is possible that African American and Caucasian youth are similarly affected by the presence of diagnosable psychiatric disorders such as depression, which negatively impact metabolic control across similar developmental trajectories.

All the abovementioned factors may explain why the rate of change of metabolic control across adolescence is similar between African American and

Caucasian groups based on the Parallel Deterioration Model. However, these factors do not explain why African American youth have poorer metabolic control at each stage of development compared to Caucasian counterparts. One plausible explanation may be that SES-related differences might explain the poorer metabolic control (i.e., higher HbA1c) of African American youth compared to Caucasian teens at entry of adolescence as depicted in this model. However, once entering adolescence, African American and Caucasian adolescents experience similar adolescent risks, resulting in similar trajectories of deterioration of metabolic control for both groups across this period.

CHAPTER THREE

RATIONALE, STUDY AIMS, AND HYPOTHESES

Rationale and Aims

Current research on type 1 diabetes in African American youth provides little guidance regarding the mechanisms that may be driving racial differences in metabolic control. The primary purpose of this study was to evaluate the developmental trajectories of metabolic control across ages 10 to 18 in two racially and economically diverse groups. The three models presented above provide hypothetical examples of unique developmental trajectories of metabolic control between African American and Caucasian adolescents. Analyses were conducted to characterize the developmental trajectories of metabolic control in the full sample, to discern whether there are racial differences in these trajectories, and whether race effects remain after controlling for neighborhood SES. While we fully recognized that other unique models, not discussed above, might have emerged during our analyses, we believed that the trajectories would most likely follow one of the described hypothetical models. Based on this longitudinal perspective, we aimed to provide hypotheses on the most plausible biopsychosocial mechanisms that may explain racial disparities in metabolic control between African American and Caucasian youth.

1. The first aim was to examine whether deteriorations in metabolic control that have been documented across ages 10 to 18 in Caucasian

youth could be replicated in a racially and economically diverse sample of youth.

2. The second aim was to determine whether African American youth compared to Caucasian counterparts began adolescence with different levels of HbA1c (intercepts) and had different rates of change (slopes) in metabolic control across ages 10 to 18.

3. The third aim was to examine whether these race-related trajectories occurred independently of neighborhood SES.

Hypotheses

Hypothesis One

Caucasian and African American children would display declines in metabolic control longitudinally across ages 10 to 18.

Hypothesis Two

Caucasian and African American adolescents would enter adolescence with different HbA1c levels and rates of change of metabolic control.

Hypothesis Three

Race effects would remain independent of neighborhood SES.

CHAPTER FOUR

METHODOLOGY

This current study utilized a medical record review to obtain health and demographic data from Caucasian and African American children and adolescents at the Children's Medical Center in Dallas, Texas (CMCD) endocrinology outpatient clinic.

Procedure and Subjects

The University of Texas Southwestern Medical Center at Dallas Institutional Review Board approved the procedures and the access to the medical charts of eligible subjects for this study. The CMCD endocrinology center and finance department generated a target list of African American and Caucasian patients with type 1 diabetes. The specific inclusion criteria were African American and Caucasian subjects who were between the ages of 14 years 0 months and 18 years 11 months when they were seen at the endocrinology clinic between January 1, 2007 and December 31, 2007. All eligible subjects must have had type 1 diabetes for at least one year. The exclusion criteria included subjects with incomplete medical information or those with less than 3 clinic visits during which HbA1c was collected. A total of 136 African American subjects were identified as potentially eligible.

From the initial list of potentially eligible subjects, diagnosis of type 1 diabetes and a minimum of 3 clinic visits were verified. Subjects who were

classified as having type 2 diabetes or less than 3 clinic visits that were documented in the electronic medical charts were excluded, resulting in 81 available African American subjects. The 81 African American subjects were matched on gender and birth date to Caucasian counterparts yielding a total of 162 subjects. Medical records for all eligible African American and Caucasian subjects were reviewed. Retrospective medical reviews were conducted through age 10 (as possible) for all subjects. In this way, data across the full sample included ages 10 through 18, capturing the developmental period during which metabolic control deteriorates for Caucasian youth. All health-related and demographic data were collected from a medical record review of electronic medical charts. Neighborhood SES variables were collected from publicly available databases containing 2000 census data based on each subject's street address.

Measured Variables

The following demographic, socioeconomic, and health information were obtained through medical chart review.

Hemoglobin A1c. Glycated hemoglobin A1c (HbA1c) was the primary measure of metabolic control and the outcome variable used in modeling trajectories of diabetes management across African American and Caucasian children and adolescents. All available measurements of HbA1c from routine clinic visits (average 2-3 per year) were collected for all subjects. HbA1c has

been consistently used as an index of metabolic control given its associations with declines in blood glucose levels (Koenig et al., 1976). Increasing HbA1c levels reflect deteriorating metabolic control. Adequate control has been defined as less than 7.5 to 8% HbA1c in children and adolescents with type 1 diabetes (ADA, 2007). High HbA1c levels have been associated with long-term diabetes complications such as retinopathy and neuropathy (Diabetes Control and Complications Trial Research, 1993).

Developmental Level. Age was the primary time variable used to map trajectories of metabolic control in this sample. Documented age was computed by birth date and date at each clinic visit during which HbA1c was measured.

Race. Patient race was determined by reported race as identified in the medical chart. This was the primary race variable used to categorize African American and Caucasian groups. Given the retrospective nature of this proposed study, the only indicators of racial status were those listed on the subject's medical record. Several studies report that the accuracy of racial classifications based on medical record information may vary based on the race of the individual (West et al., 2005). The accuracy of racial classifications is typically the highest for white patients, moderate for black, and significantly poorer for Latino and Native American individuals (Blustein, 1994; Boehmer et al., 2002; Gomez, Kelsey, Glaser, Lee, & Sidney, 2005). Present findings suggest that racial classifications based on medical records are relatively more accurate for

individuals who are typically classified as white or black and were assumed sufficient for the objectives of this study.

Neighborhood Socioeconomic Status. Given the retrospective nature of this study, individually reported SES could not be obtained. Neighborhood SES was collected from census tract-based data from geocoded subject addresses and served as an estimate or proxy of individual SES for all analyses. Neighborhood SES estimates have been found to be valid alternative markers of SES when individual SES measures are not available (Krieger, 1992). In a sample of 14,240 African American and Caucasian individuals, census-based and individually reported SES measures were similarly associated to health outcome (Krieger, Chen, Waterman, Rehkopf, & Subramanian, 2003). However, caution should be exercised when evaluating the validity of these measures as proxies of SES at the lowest income groups, as individuals from this economic group were underrepresented in this study.

There is considerable evidence that neighborhood contexts have significant associations with childhood health outcomes (Burton & Jarrett, 2000; Chen & Paterson, 2006). Neighborhood SES measures have been associated with health indicators in adolescents (i.e., systolic blood pressure) (McGrath, Matthews, & Brady, 2006). Although census-based estimates of socioeconomic data may vary in accuracy across racial groups (Kwok & Yankaskas, 2001), markers of neighborhood context continue to offer valuable and unique

information for evaluating SES effects on racial differences (Braveman et al., 2005; Diez-Roux et al., 2001) and were assumed to be the most adequate measures of SES available for the purposes of this exploratory study.

Several methods of measuring SES (i.e., income, education, assets, occupation, social capital) were available to estimate various SES constructs of interest (i.e., accumulated wealth, income level). The limitations of currently used measures (i.e., income, education) call for the need to estimate SES in multiple ways to provide a more comprehensive assessment of economic status (Braveman et al., 2005). At this exploratory stage, we chose to aggregate several neighborhood measures of SES into a single index (Edith Chen, personal communication, October 15, 2008) while also retaining individual components to fully evaluate these variables.

Neighborhood estimates of SES were obtained via publicly available geographical data systems based on the subject's listed street address. For neighborhood estimates of income, percentage of census tract below the poverty line and median family and household income were obtained. To characterize neighborhood racial composition, percentage of minority population, percentage of non-Hispanic White population, and percentage of Black population were collected. Percentage of owner-occupied and renter-occupied housing units served as estimates of neighborhood assets. Additional markers of individual SES

were obtained from medical record review information, which included insurance status and parental occupation.

Descriptive Data

The following descriptive variables were considered as potential covariates in the current study as they are often associated with metabolic control: illness duration and type of insulin therapy. Longer duration of illness is associated with declines in metabolic control (Arfken et al., 1998; Chalew et al., 2000). Therefore, age at diagnosis was included as a covariate to control for the effects of illness duration on the racial differences of metabolic control in African American and Caucasian youth. Insulin pump therapy has been associated with better metabolic control and improved quality of life in children and adolescents compared to standard insulin administration (Cogen, Henderson, Hansen, & Streisand, 2007; Hanaire-Broutin, Melki, Bessieres-Lacombe, & Tauber, 2000). As such, pump status was also included as a potential covariate.

Diabetes-related emergency room and inpatient hospital visits were collected, as these visits served as additional indices regarding the quality of diabetes management. Specifically, hyperglycemia can lead to diabetic ketoacidosis (DKA), which may result from lack of insulin administration causing high blood glucose in the bloodstream (Musey et al., 1995). These complications often require hospital visits in order to resolve the medical consequences associated with a DKA episode. As a result, HbA1c levels measured during clinic

visits were corroborated by records of diabetes-related emergency room and inpatient hospital visits, which provided additional evidence of whether African American and Caucasian youth had different patterns of metabolic control across adolescence.

CHAPTER FIVE

RESULTS

Overview of Statistical Analyses

Data were entered and managed by the Statistical Package for Social Sciences (SPSS) version 16.0 (SPSS, Inc., Chicago, IL) and descriptive statistics were analyzed in SPSS. For hierarchical linear modeling analyses that were performed, data were imported from SPSS into Hierarchical Linear Modeling (HLM) version 6.04 (Scientific Software International, Inc., Chicago, IL) and analyzed with HLM software. The data were preliminarily evaluated for violations of statistical assumptions and for outliers that may impact analyses. Distribution characteristics of all of the variables were also examined. We explored whether there were differences between the two racial groups on sociodemographic and illness variables that were not included in the a priori models; variables showing differences were covaried.

Aims were analyzed via Hierarchical Linear Modeling (HLM) techniques (Bryk & Raudenbush, 1992). HLM was selected to evaluate longitudinal trajectories of metabolic control because of several advantages that it has over traditional regression analyses (Singer & Willett, 2003). First, HLM analyzes systematic variance on two different levels. The first level analyzes variables that may be contributing to variance that accounts for changes that occur across time (i.e., within-subject changes in HbA1c across time). The second level then

considers variables that may explain variability in the pattern of change across time due to individual differences between subjects (i.e., race, gender, pump therapy, etc). In our study, the model was specified such that wave of assessment or HbA1c at each clinic visit date (level 1) was nested within each person (level 2). The level 1 equation models the relationship between HbA1c and age across clinic visits, and the level 2 equations identify how the overall relationship between HbA1c and age at clinic visit depends on the participants' individual characteristics such as race or gender. Another advantage of HLM over other statistical methods is that it utilizes all the data points even if subjects do not have information at all the time points. Thus, subjects were included in these analyses, even if they did not have data across the full age range (10 to 18). We describe the models examined for each specific aim.

Aim I: To examine whether deteriorations in metabolic control that have been documented across ages 10 to 18 in Caucasian youth could be replicated in a racially and economically diverse sample of youth who have had type 1 diabetes for at least 1 year. At level 1 (within-person), estimates of the association between HbA1c and child age were examined. This generated estimates of the average HbA1c across participants (intercepts), and average change in that value across age (slopes). Steeper slopes indicated that HbA1c was changing (presumably increasing) more rapidly with age. We expected there to be a

significant within-person effect for age indicating that HbA1c increases significantly across ages 10 to 18 years in the full sample.

Aim II: To determine whether African American youth compared to Caucasian counterparts began adolescence with different levels of HbA1c (intercepts) and had different rates of change (slopes) in metabolic control across the adolescent years. The above analyses also generated measures of variability for each parameter (intercept, slope) across participants. Such variance components indicated whether there was systematic variance in the intercept and/or slope that could be examined at level 2. We predicted the above-mentioned analyses would reveal a significant variance component on the intercept, and potentially on the slope (i.e., if the Childhood Risk Model is supported). If significant, level 2 analyses were conducted to determine whether race explained such variability. We predicted a significant level 2 effects for race on the intercept, indicating that African American youth would have higher HbA1c at age 10 than Caucasian youth. We also predicted that race would exert a level 2 effect on the slope, if there was a significant variance component. This would indicate that racial groups have different rates of change or developmental trajectories across ages 10 to 18.

Aim III: To examine whether race-related trajectories occurred independently of SES. To evaluate whether any race-related effects identified above were independent of socioeconomic status, the above analyses were

examined while statistically controlling for neighborhood SES, which for this study was used as a proxy for individual SES. These analyses were particularly important for exploring both the Childhood Risk and the Parallel Deterioration models, as discussed above.

Characteristics of the Sample

A total of 162 subjects were in the final sample for data analysis. The primary analyses reported below reflect the full sample of 162 subjects when possible. Data were available on the full sample for race, gender, insulin pump status, and age at diagnosis. A reduced sample of 152 subjects was used for analyses on neighborhood SES variables, as there was no available neighborhood SES data on 10 subjects. Family structure information was missing from 16 subjects resulting in another reduced sample of 146 subjects. All analyses from the full sample, those involving race, gender, insulin pump status, and age at diagnosis, were replicated in the reduced samples (N=152 and N=146) to ensure that there were no sampling effects on analyses conducted with the reduced samples. Where possible, the full sample was retained in order to maximize the power available for the analyses. All of the following analyses indicate whether the full or reduced samples were used.

Descriptive Statistics

Descriptive data by race for demographic, health, and neighborhood SES variables and corresponding results from Chi-square and independent sample t

tests testing racial differences are summarized in Tables 1-4. As mentioned earlier, African American subjects (N=81) were matched by birth date and gender to Caucasian counterparts (N=81). Therefore, as expected, there were no group differences based on race, gender, and age. Independent sample t tests and χ^2 analyses were conducted to examine whether racial group differences were present on background and demographic variables. There were no group differences on maternal [χ^2 (1, N = 162) = 5.19, p = .268] or paternal [χ^2 (1, N = 162) = 3.10, p = .973] occupational classification (United States Office of Personnel Management, 2008), type of other family members listed in medical record [χ^2 (1, N = 162) = 8.05, p = .153], systolic blood pressure at each clinic visit, teen age at time of diagnosis, and age at each clinic visit in years.

Insurance type differed by race. Caucasian adolescents were more likely to have managed care insurance compared to African American youth who were more likely to have public or governmentally subsidized health insurance. There was a group difference on insulin pump status whereby more Caucasian adolescents utilized insulin pump therapy compared to multiple daily injection therapy (described here as no pump therapy) than African American counterparts. Regarding number of DKA-related hospital visits, 51 Caucasian subjects did not experience any DKA episodes during the data collection period while only 28 African American subjects did not experience any DKA episodes. A significant group difference emerged such that African American subjects were significantly

more likely to have one or more DKA episodes throughout the data collection period compared to Caucasian youth. A marginal trend towards significance was found for race and family structure such that African American subjects were marginally more likely to be from a single-parent home as measured presently.

Significant group differences emerged on mean HbA1c across time, with Caucasian adolescents experiencing lower mean HbA1c across the period of adolescence than African American counterparts. Furthermore, group differences were present on height, weight, BMI, and diastolic blood pressure between Caucasian and African American adolescents. Specifically, African American adolescents were taller, weighed more, and had higher BMI values and diastolic blood pressure than Caucasian counterparts.

For neighborhood SES variables (N=152), significant racial group differences emerged such that African American subjects were more likely to live in neighborhoods with lower median family income, higher percentage of tract population below the poverty line, higher percentage of tract population classified as minority, a lower percentage of non-Hispanic White population, higher percentage of Black population, lower percentage of owner-occupied housing units, and higher percentage of renter-occupied units.

Neighborhood SES Variables

Several estimates of neighborhood socioeconomic status were collected based on the street addresses listed on the face sheet of the medical record. This

address was assumed to be the most up-to-date address at the time that data collection was initiated for the subjects. These neighborhood variables were initially correlated with the "terminal" HbA1c (i.e., the most recent HbA1c) for all subjects to identify which neighborhood SES variables most strongly correlated with the concurrent measures of metabolic control. Only median family income $r(150) = -.16$, $p = .05$ and percentage of census tract classified as minority $r(150) = .16$, $p = .05$ were significantly correlated with this terminal HbA1c measure. Thus, at the most recent visit, subjects who lived in neighborhoods with higher median family incomes and lower percentage of minority population had lower HbA1c (i.e., better metabolic control).

Factor analysis was conducted on all neighborhood SES variables to determine whether the neighborhood SES variables reflected a single dimension or multiple dimensions, and whether composite neighborhood SES scores would produce a more reliable index. Principal components analyses with varimax rotation yielded two factors with Eigenvalues greater than 1. Examination of the scree plot supported this two-factor solution. The first large factor reflected variables related to minority or racial status (e.g., percentage of tract population classified as minority), while a second smaller factor suggested income or wealth variables (e.g., median family income). The racial status factor accounted for 67% of the variance, while the income factor accounted for 16.5% of the variance. Only one variable (percentage of tract below poverty line) loaded on both factors.

In sum, the factor analysis and correlational analyses revealed two distinct neighborhood variables that correlated with "terminal" HbA1c. One factor reflected information regarding neighborhood racial composition while another reflected neighborhood estimates of income from communities in which the subjects reside. As such, the two strongest and most highly correlated predictors of terminal HbA1c - median family income and percentage of minority population - were selected for primary HLM analyses. Given that we did not have a priori hypotheses for the effects of specific neighborhood SES variables, we also created an income composite score based on the dominant factor loadings to ensure that we examined neighborhood SES as fully as possible. Therefore, median family income, percentage of minority population, and the income composite score were included in the analyses to control for the best available estimates of individual SES. There is evidence that aggregate neighborhood SES variables are useful in understanding illness trajectories in children (Edith Chen, personal communication, October 15, 2008), particularly when there are not a priori hypotheses about which neighborhood characteristic is most important.

Longitudinal Growth Curve Modeling

Linear Model Specification and Analysis Plan. The initial level 1 model evaluated the variance in the data because of the linear relationship between HbA1c (Y) and age at each clinic visit (X), and did not specify any level 2 predictors. The level 1 variable age at each clinic visit was centered at the overall

group grand mean given that all subjects were matched on age. This level 1 specification produced estimations of two level 1 random effects: the intercept (representing the expected HbA1c at the overall mean age of subjects in years, M = 13.32 years) and the linear slope. The linear slope was selected as the initial model specification, as there is reason to believe that the relationship between HbA1c and age across the period of adolescence is linear (Helgeson, Siminerio, Escobar, and Becker, 2009). Level 2 variables such as gender, pump therapy status, and age at diagnosis were then added to the initial model to determine whether they should be included as covariates in analyses with race. Level 2 variables that were significant were included as covariates in all of the subsequent primary analyses examining race and neighborhood SES associations.

Results of HLM Linear Model. Analyses of HLM models are presented in Tables 5-9. The initial random coefficients model (model 1) was conducted first and only evaluated level 1 variables to determine whether there was evidence that age at clinic visit was associated with HbA1c at clinic visit. This provided a basic characterization of the average HbA1c (i.e., intercept) and the manner in which HbA1c changed over time (i.e., slope), and to determine whether there were variance components on each of those coefficients. The variance components provided information about whether there were systematic individual differences (e.g., race) as a function of the average HbA1c or in change in HbA1c across

time. Without significant variance components, there would be no basis for exploring racial effects.

The level 1 equation for the initial random coefficients model (model 1) was:

$$\text{HbA1c}_{ij} = B_{0i} + B_{1i}\,(\text{age at clinic visit}) + r_{ij}\ .$$

This equation specified that the HbA1c for subject *i* at clinic visit *j* was a function of B_{0i} (intercept), which was the HbA1c for subject *i* when age was 0 (the average age when centered at the mean), B_{1i} (slope) reflected the relationship between age and HbA1c across clinic visits for subject *i*, and r_{ij} was the measurement error. No level 2 predictors were included in the basic model (model 1). In subsequent analyses, the intercept (B_{0i}) and slope (B_{1i}) became the dependent variables predicted by level 2 variables (i.e., race, gender, pump status), as specified in the following level 2 equations:

$$B_{0i} = G_{00} + G_{01}\,(\text{level 2 predictor variables}) + u0$$

$$B_{1i} = G_{10} + G_{11}\,(\text{level 2 predictor variables}) + u1.$$

Table 5 summarizes the intercept and slope coefficients for model 1. The coefficients for both the intercept and slope were significant, and there were variance components for both intercept and slope. This meant that at the average age (i.e., 13.32 years old), subjects had an HbA1c value of 8.94% and this was statistically different from zero. In addition, for every yearly increase in age there was a .22% increase in HbA1c, and this rate of change was significant from zero.

The variance components indicated that there were systematic differences between subgroups of subjects in HbA1c observed at the average age, and in the change in HbA1c across age.

The results from this analysis addressed Aim 1, which aimed to replicate age-related declines in HbA1c (previously only witnessed in Caucasian samples) across the period of adolescence in the entire sample of Caucasian and African American youth. Significant within-person age effects emerged indicating that HbA1c increased significantly across ages 10 to 18 years in the full sample. The significant variance components provided support for further analyses to discern whether race and/or other Level 2 variables may be explaining differences in average HbA1c or in change in HbA1c across the transition into and through adolescence.

Level 2 Covariates

Potential covariates were then analyzed in order to determine whether they should be included in all subsequent analyses.

Gender (N =162). Model 2 included gender as the only level 2 predictor to evaluate possible gender effects on the intercept and slope trajectories of metabolic control across adolescence. In this model, the coefficients for intercept (B_{0i}) and for slope (B_{1i}) became the outcome variables, with gender as the predictor variable in the following level 2 equations:

$$B_{0i} = G_{00} + G_{01}\ (\text{Gender}) + u0$$

$B_{1i} = G_{10} + G_{11}$ (Gender) + u1.

As reported on Table 5 in model 2, gender did not predict either intercept or slope for the sample, and the variance components on both remained significant. Specifically, there were systematic differences in average HbA1c at the average age and the rate of change across adolescence, but gender was not predictive of these differences. As a result, gender was excluded from subsequent models.

Insulin Pump Status (N =162). Pump status was analyzed in model 3 to determine whether it emerged as a significant predictor of level 1 variance components and should be included as a covariate in subsequent analyses.
As reported on Table 5 in model 3, pump status emerged as a significant predictor of intercept and slope variance in the level 1 model. The intercept coefficient of 1.19 reflected that adolescents using the insulin pump had 1.19% lower HbA1c than children who did not use the pump. Furthermore, there was a .15% greater increase in HbA1c with every year of age among those with no insulin pump versus insulin pump therapy patients. Pump status was included as a level 2 covariate for subsequent analyses

Age at Diagnosis (N = 162). Age at diagnosis was analyzed in model 4 as the only level 2 predictor to determine whether it is a significant level 2 predictor of level 1 variance components and whether it should be included in the subsequent analyses as a covariate. As shown on Table 5 in model 4, age at diagnosis was found to be a significant level 2 predictor of HbA1c at the intercept

and the linear slope. Subjects who were diagnosed at an older age had .21% lower HbA1c at the average age and for each subsequent year later that they were diagnosed they had a .02% higher increase in HbA1c across age. Therefore, age at diagnosis was included in all subsequent analyses.

In sum, these analyses confirmed that age at diagnosis and pump status should be included in all subsequent analyses as covariates in order to control for the effects of these health variables on potential race effects on average HbA1c and change in HbA1c over time. Gender was excluded from all further analyses given that it was not a significant predictor of level 1 variance for intercept and slope.

Level 2 Predictors

Racial Status (N = 162). Model 5 evaluated the effect of racial status on average HbA1c at the average age and the rate of change in HbA1c across adolescence. This analysis included age at diagnosis and pump status as level 2 covariates given their significant effects on the level 1 variances for intercept and slope. This analysis served to address Aim 2, which aimed to determine whether African American youth compared to Caucasian counterparts had different levels of HbA1c (intercept) and different rates of change (slopes) in metabolic control across the adolescent years.

As reported on Table 6 in model 5, when race was included as a level 2 predictor, with age at diagnosis and pump status as covariates in the model, the

intercept coefficient of 1.32 was significant, while the slope coefficient was non-significant. This indicated that African American subjects at the average age had 1.32% higher HbA1c than Caucasian counterparts, but evidenced parallel trajectories in rates of change in HbA1c over time. This finding provided support for the a priori hypothesis that African American youth would have poorer HbA1c during adolescence than Caucasian youth; however, it also suggested parallel rates of change in HbA1c. Race continued to remain a significant level 2 predictor of level 1 intercept differences even when age at diagnosis and pump status were included as covariates in the analyses.

In sum, the findings indicate that while race explained racial differences in the level (intercept) of HbA1c it did not predict age-related changes (slope) in HbA1c trajectories. Specifically, African American and Caucasian subjects have different levels of HbA1c across adolescence, but they show parallel trajectories and did not differ in the rate of change in metabolic control across this time period. Pump status appeared to be a significant level 2 predictor of level 1 slope variance independently of race and age at diagnosis. Specifically, the positive relationship between HbA1c and age at clinic visit was stronger for individuals not on the insulin pump than those who were on the pump.

Re-centered Age (N=162). In Aim 2, we also proposed to evaluate whether African American adolescents began and ended the period of adolescence with different mean HbA1c compared to Caucasian subjects. In order to evaluate

these differences, the overall group mean age was re-centered to age 10 and then to age 17 in order to test group differences in intercept as the youth entered (age 10) and exited (age 17) the period of adolescence. Several previous studies evaluating the effects of adolescence on HbA1c have used age 10 as the baseline age prior to the period of adolescence (Anderson et al., 1997; Helgeson et al., 2009). Race was entered into analyses while age at diagnosis and pump status remained as covariates.

When mean clinic visit age was re-centered at age 10 and age 17, the same results were found as when the mean age was centered at the grand mean (M=13.32 years old). Specifically, race significantly predicted intercept differences, but not slope differences in HbA1c. These findings further address Aim 2 and support the a priori hypotheses that African American subjects begin adolescence with higher levels of HbA1c. Furthermore, African Americans continue to have higher HbA1c through adolescence and end this period with higher HbA1c as well. No differences in slope were found at the beginning, middle, and end of adolescence (ages 10-17). These findings suggest that although African Americans enter the period of adolescence with poorer metabolic control, this period is equally risky for African American and Caucasian youth, consistent with the Parallel Deterioration Model.

Neighborhood SES predictors (N =152). Aim 3 examined whether the impact of race on the developmental trajectories of metabolic control occurred

independently of neighborhood SES. As discussed earlier, three neighborhood SES factors median family income, percentage of minority population, and income composite scores were included as individual and aggregate neighborhood proxies of individual SES.

Median family income, percentage of minority population, and income composite scores were first analyzed in relation with HbA1c over time, independently of race. All 3 neighborhood SES variables, median family income (p = .009), percentage of minority population (p = .017), and income composite score (p = .049), significantly predicted intercept differences in HbA1c, but failed to predict the linear slope. In sum, all these neighborhood SES variables independently had the same effect on intercept and slope as race whereby they were predictive of intercept differences, but not linear slope.

Given the significant predictive value for all three neighborhood SES variables on intercept differences in HbA1c, they were further analyzed with race, age at diagnosis, and insulin pump status. As reported on Table 7, model 6 analyzed the effects of neighborhood SES on HbA1c over time by including median family income and percentage of minority with race, age at diagnosis, and insulin pump status. Race, insulin pump status, and age at diagnosis continued to significantly predict intercept differences, but not slope, although there was a marginal effect on slope from insulin pump status. While both median family income and percentage of minority population were predictive independently,

when combined, median family income emerged with unique predictive value beyond the effects of percentage of minority population.

In sum, race effects were independent of SES, which addressed Aim 3 in which we evaluated whether race retained independent effects from neighborhood SES in explaining the longitudinal trajectories of metabolic control in Caucasian and African American youth. Despite the robust effect of race, median family income appeared to capture more of the unique variance compared to all other neighborhood SES variables and remained associated with average HbA1c independent of race.

Exploratory Analyses

Family Structure (N = 146). Family structure was analyzed independently of race, then with race, pump status, and age at diagnosis. When family structure was included as a level 2 predictor independently of race (shown on Table 8 in model 7), it did not independently predict level 1 variance for intercept and slope. When race and family structure were included in model 8 along with pump status and age at diagnosis as covariates, the same results emerged as in earlier models whereby race, insulin pump status, and age at diagnosis were significant predictors of intercept differences, (but not of slope), and family structure did not predict either intercept or slope. Family structure was not further analyzed with neighborhood SES variables given that it was consistently not predictive of intercept or slope differences over time. These data provide no evidence of

effects of family structure on trajectories of HbA1c, but should be interpreted with caution given the limitations of the family structure measure in the present study.

Overall, the results indicated that African American youth had poorer HbA1c than Caucasian youth at the beginning of adolescence (age 10) and this continued through age 17. Subjects also had poorer HbA1c if they were not on the pump, had lower median family income, and were diagnosed at an earlier age. All of these effects were independent of each other suggesting that systematic individual differences on these dimensions provide important information about subjects at higher risk for poorer metabolic control. None of these variables explained the racial differences in HbA1c. While there were no statistically significant effects of these predictors on slope, insulin pump status had marginal independent effects on slope. Median family income emerged as the neighborhood SES variable that accounted for the most unique variance in intercept differences in HbA1c. Family structure did not account for any variance components for intercept or slope.

Nonlinear Model. Loess plots of overall HbA1c across adolescence by race suggested possible nonlinear changes over time. As a result, exploratory analyses were conducted for a quadratic model to evaluate potential curvilinear associations between age and HbA1c by race. In the linear models, race, insulin pump status, and age at diagnosis emerged as the most significant predictors of

HbA1c over time. Therefore, these variables were included as level 2 predictors for the quadratic model and reported on Table 9 (model 9). The results indicated that the quadratic slope was not significant, suggesting that our model was not able to capture significant quadratic changes over time. However, a significant variance component for the quadratic slope emerged, which suggested that there appeared to be systematic differences in quadratic changes over time such that some individuals showed curvilinear patterns of change while others did not. Those differences, however, were not captured by the variables obtained in the present study.

Pump status as time-varying covariate. Exploratory analyses were conducted with pump status as a level 1 time-varying covariate for all primary analyses. As reported in Table 10, pump status was analyzed as a level 1 predictor of HbA1c (model 10) and then with race and age at diagnosis shown in Table 11 (model 11). Pump status was further analyzed with race and age at diagnosis and the neighborhood SES variables (median family income and percentage minority in census tract) as reported in Table 12 (model 12). There were no differences in the primary findings when pump status was covaried as a level 1 versus level 2 variable in all the primary analyses.

CHAPTER SIX

DISCUSSION

Overview of the Study

The first aim of this study was to evaluate whether declines in metabolic control evidenced in Caucasian youth could be replicated in a racially and economically diverse sample that included African American adolescents. A second aim of this study was to evaluate whether these racial groups entered the period of adolescence with different mean levels of HbA1c (intercept) and had different rates of change (slope) in HbA1c across this period. The final aim of this study was to determine whether the effects of race on the developmental trajectories of HbA1c were independent of neighborhood SES. By characterizing the developmental illness trajectories in both Caucasian and African American youth, further research elucidating the effects of race on metabolic control can be targeted to the most critical periods of vulnerability for Caucasian and African American youth. Furthermore, it may aid in the development of interventions to reduce racial disparities in type 1 diabetes management and prevent detrimental long-term consequences in adulthood.

The results from this current study are the first to report age-related declines in metabolic control characteristically observed in Caucasian youth in a sample of gender and age-matched African American adolescents. That is, adolescents evidenced deterioration in metabolic control across adolescence

regardless of racial status. These findings extended previously well-documented age-related deteriorations across adolescence in Caucasian youth (Amiel et al., 1986; Anderson et al., 1997; Helgeson et al., 2009; Weissberg-Benchell et al., 1995; Wysocki et al., 1996) to African American youth. African American youth had higher HbA1c at every age across adolescence compared to Caucasian youth, extending previous cross-sectional evidence for racial differences (Auslander et al., 1997; Delamater et al., 1991; Delamater et al., 1999). Furthermore, these race effects were found independently of neighborhood SES. Rates of change in HbA1c across adolescence were similar for both racial groups suggesting parallel trajectories of illness. Overall, these results are the first to provide a developmental perspective of racial differences in metabolic control across the period of adolescence and are consistent with the Parallel Deterioration Model proposed in this study.

Intercept Differences

This is one of the first studies to provide evidence for race effects on intercept differences in HbA1c across adolescence, which were independent of neighborhood SES. Given that race is often correlated with SES, as it clearly was presently, it is difficult to determine whether race effects are merely proxies for underlying mechanisms such as low-income status or lack of access to health care. Our findings suggested a robust independent effect of race on average HbA1c that occurred beyond the effects of neighborhood SES. Furthermore, race

effects were not fully explained by pump status despite significant racial differences on pump status such that African American youth were less likely to receive insulin pump therapy. Eligibility for the use of insulin therapy may depend upon availability of financial resources as well as a history of adequate metabolic control. Therefore, this finding provides evidence for identifying additional mechanisms that explain HbA1c differences between African American and Caucasian youth, as racial differences in HbA1c were not merely due to racial differences in use of insulin pump therapy. It should be noted that neighborhood SES was a predictor when analyzed independently of race and was, thus, not an inactive variable. While results should be interpreted with caution, as it is possible that neighborhood SES variables were not sufficient proxies of individual SES, these are the first results to identify unique racial differences beyond neighborhood SES in the level of HbA1c across the critical period of adolescence.

Frey et al. (2007) found racial differences in level of HbA1c shortly after diagnosis suggesting that there is a period of risk shortly after diagnosis that is unique for African American youth. Our findings included the time period shortly after diagnosis and controlled for duration of illnesses and also found race effects around the time period shortly after diagnosis. These results provide additional support that racial differences in level of HbA1c may begin prior to the period of adolescence.

Several mechanisms are hypothesized to explain why African American youth had higher HbA1c before entering adolescence, although it should be noted that no direct measures for these pathways were obtained in this study. Family factors are consistently related to metabolic control (Delamater, 2007) and should be a priority for future research evaluating racial disparities in metabolic control. There is significant evidence that African American parents socialize and engage with their children differently from Caucasian parents (Kotchick & Forehand, 2002; Bulcroft et al., 1996). It is no less plausible that the context of chronic illness may exert different pressures on African American families in areas such as parenting practices. For example, African American parents tend to engage in more "no-nonsense parenting," which is hypothesized to be in response to the unique challenges that African American families face in more dangerous communities. While this style of parenting may be adaptive for healthy adolescents in dangerous environments, authoritarian parenting practices in the context of diabetes management may place African American youth at greater risks for poor control, as harsh and controlling parenting practices negatively impact metabolic control in older Caucasian youth (Wiebe et al., 2005).

No studies have evaluated the importance of family and parenting practices in African American families and its impact on metabolic control for African American children and adolescents. Furthermore, no interventions have been developed to increase or maintain parental involvement in African American

families during adolescence, while several have been effective in Caucasian families (Wysocki et al., 2003; Anderson et al., 1997). Studies with chronic illnesses more prevalent in African American children such as sickle cell disease or asthma suggest that culturally appropriate family interventions may improve disease knowledge and psychosocial functioning in families (Celano, 2006; Kaslow, 2000; Kaslow et al., 1997). Taken together, our findings provide support for unique race effects on metabolic control and current literature on pediatric chronic illnesses suggest that family functioning may explain some of these racial differences.

Another important mechanism that may explain racial differences in HbA1c is family structure, as African American youth disproportionately come from single-parent families in the United States. In this study, exploratory analyses on family structure did not yield significant effects on either average HbA1c or trajectories of HbA1c across adolescence. However, these current findings should be interpreted with caution due to measurement issues. Firstly, our measure of family structure was very indirect, based on the listed address of both parents from the most recent clinic visit available. Secondly, we did not have this indirect measure of family structure across the entire period of adolescence because it was only measured at the "terminal" visit. There was only a marginal association between race and family structure, which may partially

explain the lack of effect in HLM analyses. Furthermore, the lack of significant findings for parental structure may be related to issues of statistical power.

Frey et al. (2007) found that deteriorations in HbA1c after diagnosis could be better explained by family structure than by race. It is important for future studies to replicate these previous findings, as several studies support that adolescents living in single-parent families have poorer metabolic control than those from two-parent households (Auslander et al., 1997; Delamater et al., 1991; Thompson et al., 2001). It is hypothesized that the lack of an additional parent may reduce the level of monitoring, involvement, and resources available for optimal diabetes management. Furthermore, the lack of a parent may increase the financial burdens on families as they are struggling to meet their financial needs while coping with a child's chronic illness. While our measure of parental structure was not robust enough to capture significant differences, it remains a significant area for future research given its significance in explaining racial differences in HbA1c in previous studies.

Racial differences in interactions with medical professionals may also contribute to racial differences in HbA1c, especially during the period shortly after diagnosis because families may not engage in necessary education or attend required appointments as consistently. Racial disparities have been found in perceptions of illness and in seeking medical care (Landrine & Klonoff, 2001), which remain even if racial disparities in income and health insurance coverage

are removed (Weinick, Zuvekas, & Cohen, 2000). Minority groups may experience feelings of distrust with medical professionals that do not represent their racial background (LaViest, Nickerson, & Bowie, 2000), while racial concordance between patient and doctor improve perceived quality of health care (Saha, Komaromy, Koepsell, & Bindman, 1999). In addition, doctors are influenced by demographic or socioeconomic cues and tend to perceive African American and lower-SES individuals more negatively than White or upper class patients (van Ryn & Burke, 2000).

Consequently, racially influenced perceptions of medical care as well as doctors' negative perceptions of African American families may hinder these families from developing the relationships and skills necessary to optimally assist in their child's medical care. Negative interactions with medical staff may result in poorer adherence to medical regimens or lower attendance of clinic or diabetes education visits. While the assessment of racism or discrimination in medical settings may be controversial, experiences of prejudice are crucial to a child's development and self-identity (Garcia Coll et al., 1996). The effects of racism and discrimination on health outcomes have been documented in adults and are no less important in adolescence, as teens are beginning the process of racial and ethnic identity formation. Racial differences in interactions with medical teams occur independently of socioeconomic status and may partially explain why

African American youth have higher HbA1c than Caucasian youth before entering adolescence.

Parallel Slope

Although African American youth had higher HbA1c at every age across adolescence compared to Caucasian youth, there were no racial differences in the rate of change in HbA1c across age. That is, they evidenced parallel rates of change in metabolic control across the period of adolescence. These findings suggest that adolescence is a period of risk for both Caucasian and African American youth and these risks impact both racial groups equally. While Frey et al. (2007) found that across the first 5 years post-diagnosis African American youth had faster rates of deterioration in metabolic control than Caucasian youth, our results did not find differential rates of change in adolescence.

Our findings are not entirely contradictory to Frey et al.'s findings (2007) because the present study characterized rates of change across adolescence while their study evaluated racial differences shortly after diagnosis. In combination, these results may suggest that unique risks for African American youth emerge in childhood and are maintained across adolescence. The initial period of vulnerability shortly after diagnosis may place African American youth at higher levels of HbA1c compared to Caucasian youth before they enter adolescence. Once in adolescence, the rates of decline in metabolic control of African American youth may equalize with Caucasian counterparts, as they experience

similar adolescent risk factors, suggesting continued risks for African American adolescents.

The results from this study regarding the parallel rates of change in HbA1c between African American and Caucasian youth are the first to ever be reported to our knowledge and support the hypothesized Parallel Deterioration Model proposed in this study. The plausible mechanisms or pathways that may explain these parallel trajectories can only be speculated at this time. While this current study did not measure pathways that explain the trajectories of metabolic control, it is notable to mention that the parallel trajectories provide rationale for replicating currently researched mechanisms explaining declines in metabolic control across adolescence in Caucasian samples within African American groups.

Commonly researched explanations for age-related deteriorations in HbA1c for Caucasian youth during adolescence are briefly summarized and discussed as potential areas of research that may explain parallel trajectories of HbA1c in African American and Caucasian youth. Adolescence is a time when teens experience significant physiological, emotional and behavioral changes that invariably impact diabetes management. Insulin resistance during adolescence (Goran & Gower, 2001; Amiel et al., 1986) may explain parallel trajectories between these two groups. Research studies on how African American teens interpret the physiological impact of puberty on their metabolic control may

provide insights into reasons why they experience similar declines as Caucasian youth. Self-care behaviors that decline during adolescence (Anderson et al., 1997) may also explain parallel deteriorations in metabolic control and point towards future research evaluating the reasons why African American youth may forgo self-care behaviors. These findings can generate important information for the development of behavioral interventions for this minority group. Understanding the presence and prevalence of emotional difficulties such as depression that emerge with higher rates in children with diabetes (Hood et al., 2006; Kovacs et al., 1997) may be particularly important as few studies have evaluated the impact of depression on African American children and adolescents with diabetes.

Another possible mechanism for parallel rates of change in HbA1c in African American and Caucasian youth may be related to similar increases in autonomy seeking that emerge during adolescence. During adolescence, teens begin to claim increased self-care autonomy that result in deteriorations of metabolic control (Wysocki et al., 1996). Furthermore, parents may rely solely on chronological age to relinquish diabetes responsibilities to teens (Palmer et al., 2004), which may happen in both Caucasian and African American families. Therefore, it would be plausible that African American and Caucasian families struggling with diabetes experience similar challenges as they negotiate self-care

responsibilities as well as manage the increased conflicts associated with this developmental period.

Peer relationships are another important mechanism for explaining parallel trajectories in African American and Caucasian youth. Several studies have found that peer relationships impact diabetes management (Burroughs, Harris, Pontious, & Santiago, 1997; Hains et al., 2007), but with mixed positive and negative findings (Bearman & La Greca, 2002; Greco, Pendley, McDonell, & Reeves, 2001; Hains et al., 2006; Naar-King, Podolski, Ellis, Frey, & Templin, 2006; Pendley et al., 2002; Thomas et al., 1997). There is some preliminary evidence that peer interactions predict HbA1c over time, providing support for its long-term impact in diabetes management across adolescence (Helgeson et al., 2009).

African American communities have higher rates of juvenile delinquency and drug use compared to Caucasian groups and some of these behaviors are associated with delinquent peer affiliations (Joseph, 1995; Williams et al., 2007). It is plausible that peer relationships unique to African American youth may have significant influences on metabolic control, contributing to the parallel trajectories between African American and Caucasian youth. While some studies have included African American participants in evaluating peer influences on metabolic control, few have included large enough samples of African American youth to find significant racial differences. Innovative research investigating the

impact of potentially unique peer dynamics on metabolic control in African American groups may provide additional information on the parallel trajectories of declining metabolic control in Caucasian and African American adolescents.

All of the hypothesized mechanisms mentioned above assume that the risks associated with adolescence are the same between African American and Caucasian youth. However, it is equally plausible that different mechanisms are at work in different racial groups such that one mechanism may have a greater impact on a certain racial group over other mechanisms. For example, conflicts in parent-child relationships may be exerting the most strain on metabolic control for Caucasian youth across adolescence while negative peer influences may actually explain the deteriorations in metabolic control in African American youth across this period. These nuanced factors must be further evaluated in order to understand why and how adolescence is an equally vulnerable period of time for African American and Caucasian youth. The present data provide confidence that such evaluations will be useful for future research and interventions.

Implications of Tangential Findings

Significant variance components emerged for both the intercept and slope coefficients in all of the models, suggesting that a significant amount of variance has been left unexplained by our measured variables. It is possible that all the unmeasured variables mentioned above are potential candidates (i.e., child perceptions or parenting style) for explaining racial differences. These findings

highlight the need for further research studies that include large samples of African American youth and their families coping with diabetes. At this time, family functioning and peer influences appear to be the most promising areas of future research in pediatric diabetes.

While several studies have found that income or SES have unique effects on metabolic control independently from race, our current findings did not support these previous findings. However, median family income was the strongest predictor of HbA1c compared to other measures of neighborhood income/wealth and racial composition. These findings are consistent with research on trajectories of child health that suggest that accumulated family income is a stronger predictor of long-term trajectories of activity limitations in children than other measures of income status (Chen, Martin, & Matthews, 2007). Low-SES children have worse health with older age compared to high-SES children because of higher exposure to health risks (Currie & Stabile, 2003), suggesting that low-income status may place children at higher risk for health-related problems. While our study did not find unique predictive effects of neighborhood SES from race effects, current findings on trajectories of child health indicate that family income continues to have significant effects that may be independent of race and should be further evaluated in pediatric chronic illnesses.

Methodological Considerations

Several methodological considerations should be taken into account when interpreting the findings. Firstly, the data available were limited to retrospective health data collected during routine clinic visits for standard care. As such, while we were able to characterize broad associations between socio-demographic variables and metabolic control, we were not able to measure variables that could explain processes or mechanisms by which these illness trajectories occur. Specifically, no measures were obtained to clarify findings or mechanisms (e.g., parenting, child perceptions), which should be included in and will greatly inform future studies. No direct measures of psychological status were collected and pubertal status was not consistently available so we could not evaluate the effects of pubertal and psychological status on metabolic control. While the sample size of this study was larger than the only other study investigating racial differences in developmental trajectories of diabetes management, future studies should obtain a larger sample size in order to address issues related to statistical power. The findings of this study do not generalize to other ethnic minority groups or beyond this single site. Multi-site studies that include other minority groups would further expand on the current findings and increase overall generalizability.

We utilized neighborhood SES as a proxy for individual SES, which has been associated with trajectories of child health. While this technique has often been used when individual SES has not been available and is a recommended

solution for the lack of socioeconomic data in health databases, its use is not without limitations. Specifically, neighborhood SES remains only an estimate of the economic and racial composition of the neighborhood within which the individual resides. Therefore, there are issues associated with misclassification of individuals based on neighborhood aggregates of socioeconomic status (Hyndman et al., 1995). Furthermore, this method may be less accurate given that African Americans are more likely to live in communities with higher risk and greater disadvantages compared to Caucasian counterparts regardless of income (Diez-Roux et al., 2001). As such, the racial inequity of communities regardless of income status may confound the interpretation of results based on this technique. While these limitations make the interpretation of neighborhood proxies of individual SES data more difficult, it is likely that this technique will remain useful until national objectives to obtained individual SES with health information are achieved. It is recommended that future studies collect both neighborhood and individual SES variables to determine their overall and unique effects on metabolic control.

Another debated issue in using geocoded data to obtain aggregates of neighborhood SES as proxies of individual SES is the unit of measurement or size of geographic region from which the data are collected. It has been argued that smaller units of measurement would increase the power of findings such that moving from zip code-based to census tract to block group data would increase

the variability needed to obtain significant effects. However, there is only minimal improvement in predictive usefulness when moving to smaller units of measurement (Geronimus & Bound, 1998; Soobader, LeClere, Hadden, & Maury, 2001). Furthermore, block group data are more difficult to obtain and often do not account for less populated communities. Given that our hypothesized mechanisms focused on family and peer relationships, no significant a priori hypotheses were developed regarding which unit of measurement would obtain more significant results. However, we would assume that moving from census tract to block group units of measurement would not significantly impact the hypothesized importance of family or peer relationships on metabolic control. It has been suggested that determining the unit of measurement tends to vary between studies based on the questions being asked (Diez-Roux et al., 2001).

As discussed earlier, family structure was obtained through medical record information and was only obtained at the most recent clinic visit date. Therefore, future longitudinal studies should try to obtain multiple measures of concurrent family structure information to precisely evaluate its impact on metabolic control across adolescence.

Clinical Implications

Despite the limitations of this current study, several findings emerged that have significant implications on directions for future research and the development of appropriate interventions to address health disparities in pediatric

diabetes. Most notably, our study is the first to confirm that African American adolescents experience similar age-related declines compared to Caucasian youth. Furthermore, we found that while African American youth have poorer metabolic control across the entire period of adolescence, they have similar rates of change in metabolic control compared to Caucasian counterparts. These findings combined with Frey et al. (2007) suggest that African American youth experience risks during childhood that are maintained throughout adolescence.

The findings from this study point to the need for further research evaluating the mechanisms that may explain why African American families experience initial vulnerability that is maintained into adolescence. Specifically, adolescent pathways are at least as important for African American youth as they are for Caucasian youth. These findings highlight the importance of understanding unique factors in adolescence for African American as well as Caucasian teens that may be contributing to challenges in metabolic control. Furthermore, this study provides rationale for the research and development of interventions that target racial disparities in metabolic control in the critical period of adolescence.

Addressing racial disparities in metabolic control are particularly important given that health behaviors developed early in life remain fairly consistent throughout life. Furthermore, there is evidence for "metabolic memory," which hypothesizes that early glycemic patterns or "environments"

may be “remembered” or stored in target organs (Ceriello, Ihnat, & Thorpe, 2009). For example, the heart or eyes may “remember” the poorly controlled glycemic state from early life and may actually develop later complications despite improved or normal glycemic control in later life. This may make it more difficult for individuals to change the course of their illnesses and protect themselves from long-term complications once their bodies have become repeatedly exposed to poor glycemic conditions from earlier life. These biological effects further highlight the importance of addressing health outcomes early in life, especially in light of the disparities that emerge in childhood and adolescence.

Until we begin the often difficult, yet rewarding task of understanding how race and culture impact pediatric diabetes, racial disparities in health outcomes will likely persist and disproportionately place certain racial groups at higher morbidity and mortality for illnesses that are well-controlled and maintained in the majority racial group. Racial disparities in type 1 diabetes management for children and adolescents likely place these individuals on lifelong trajectories of poor metabolic control that persist into adulthood, as African American adults with diabetes have higher morbidity and mortality compared to Caucasian adults. Understanding racial differences from a developmental perspective can guide innovative research and interventions that

are crucial in answering the national objectives to improve quality of care and health outcomes for all minority groups.

APPENDIX A

Tables

Table 1
Descriptive Demographic Data with Summary Statistics for χ^2 Analyses by Race

Source	Mean, (SD), or Percent	χ^2, df	Significance
Demographic Data			
Race (%)			
Caucasian	50.0		
African American	50.0		
Gender (%)			
Female	50.6	.000, 1	1.00
Male	49.4		
Public Insurance (%) +			
Caucasian	19.8	8.42, 1	.004
African American	42.0		
Single-Parent Households (%) +			
Caucasian	44.4	3.10, 1	.078
African American	61.7		

Note: All of the variables included N = 162 with the exception of those otherwise noted.
+ N = 146

Table 2
Descriptive Neighborhood SES Data with Summary Statistics for Independent Samples t tests by Race

Source	Mean, (SD), or Percent	Range	T, df	Significance
*Neighborhood SES Variables**				
Median Family Income ($)				
Caucasian	76,289, (36,290)	25,283-193,016	4.39, 112	.000
African American	55,081, (20,501)	11,132-106,397		
Median Household Income ($)				
Caucasian	68,370, (30,626)	25,690-149,428	4.42, 120	.000
African American	49,852, (19,358)	10,800-96,482		
% Minority Population				
Caucasian	25.16, (18.56)	2.95-86.46	-7.49, 138	.000
African American	53.42, (27.40)	12.68-99.46		
% Tract Below Poverty Line				
Caucasian	6.55, (5.86)	.19-29.74	-3.76, 121	.000
African American	11.88, (11.02)	.80-62.19		

Note: All of the variables included N = 162 with the exception of those otherwise noted. * N = 152

Table 2 continued

Descriptive Neighborhood SES Data with Summary Statistics for Independent Samples t tests by Race

Source	Mean, (SD), or Percent	Range	T, df	Significance
*Neighborhood SES Variables**				
% non-Hispanic White population				
Caucasian	74.84, (18.56)	13.54-97.05	7.49, 138	.000
African American	46.58, (27.41)	.54-87.32		
% Black Population				
Caucasian	7.87, (9.69)	.20-60.70	-7.21, 98	.000
African American	32.11, (28.11)	1.21-96.23		
% owner-occupied units				
Caucasian	73.07, (18.64)	7.34-98.04	2.60, 150	.010
African American	64.63, (21.11)	6.56-95.70		
% renter-occupied units				
Caucasian	22.05, (17.62)	1.11-85.05	-2.60, 150	.010
African American	29.93, (19.75)	2.12-89.60		

Note: All of the variables included N = 162 with the exception of those otherwise noted. * N = 152

Table 3
Descriptive Medical Data with Summary Statistics for χ^2 and Independent Samples t tests by Race

Source	Mean, (SD), or Percent	Range	χ^2, df	T, df	Significance
Health Data					
Age at diagnosis (years)					
Caucasian	9.91, (3.27)	1.25-15.55		-.709, 160	.479
African American	10.09, (3.74)	1.08-16.57			
Number of Clinic Visits ^					
Caucasian	19.28, (8.19)	4-43		1.94, 160	.054
African American	16.49, (10.02)	3-44			
Average age across all clinic visits ^					
Caucasian	13.30, (3.00)	5.51-19.13		-.38, 2898	.706
African American	13.34, (3.09)	6.95-18.85			
Insulin Pump Therapy (%)					
Caucasian	18.5		9.00, 1		.003
African American	3.7				

Note: All of the variables included N = 162 with the exception of those otherwise noted. ^ N = 2900

Table 4
Descriptive Recurrent Health Data with Summary Statistics for χ^2 and Independent Samples t tests by Race

Source	Mean, (SD), or Percent	Range	χ^2, df	T, df	Significance
Recurrent Health Data ^					
HbA1c					
Caucasian	8.36, (1.57)	4.6-14.0		-22.36, 2356	.000
African American	9.98, (2.20)	4.9-14.0			
Number of subjects with no presence of DKA related visits (%)					
Caucasian	62.9		13.07, 1		.000
African American	34.5				
Height (cm)					
Caucasian	159.60, (13.86)	111.7-189.6		-2.35, 2531	.019
African American	160.77, (11.28)	121.5-194.5			
Weight (kg)					
Caucasian	56.84, (17.42)	22.15-112.4		-4.44, 2589	.000
African American	59.59, (14.08)	20.25-106.4			

Note: All of the variables included N = 162 with the exception of those otherwise noted. ^ N = 2878

Table 4 continued

Descriptive Recurrent Health Data with Summary Statistics for Independent Samples t tests by Race

Source	Mean, (SD), or Percent	Range	T, df	Significance
Recurrent Health Data ^				
Body Mass Index				
Caucasian	22.09, (4.21)	13.52-41.04	-4.56, 2530	.000
African American	22.82, (3.80)	13.30-35.09		
Systolic Blood Pressure				
Caucasian	117.7, (13.3)	43-183	-1.26, 2381	.209
African American	118.4, (12.4)	43-174		
Diastolic Blood Pressure				
Caucasian	65.1, (8.7)	37-108	-4.06, 2426	.000
African American	66.6, (9.0)	29-116		

Note: All of the variables included N = 162 with the exception of those otherwise noted. ^ N = 2878

Table 5
HLM Estimates of Fixed and Random Effects for the Basic Model and Covariates

	Coefficient	p	Variance Component	p
Model 1: Level 1 predictors only				
$B_{00\ (intercept)}$	8.94	.000	2.52	.000
$B_{10\ (slope)}$	.22	.000	.11	.000
Model 2: Gender as Level 2 predictor				
$G_{01\ (intercept)}$	-.38	.148	2.51	.000
$G_{11\ (slope)}$	.01	.835	.11	.000
Model 3: Pump Status as Level 2 predictor				
$G_{01\ (intercept)}$	1.19	.000	2.38	.000
$G_{11\ (slope)}$	.15	.040	.10	.000
Model 4: Age at Diagnosis as Level 2 predictor				
$G_{01\ (intercept)}$	-.20	.000	2.16	.000
$G_{11\ (slope)}$	.02	.045	.10	.000

Note: Caucasians were coded as 1 and African Americans were coded as 2. Females were coded as 1 and males as 2. Use of insulin pump was coded as 1 and no pump therapy as 2.

Table 6
HLM Estimates of Fixed and Random Effects for Race and Covariates

	Coefficient	p	Variance Component	p
Model 5: Race, Pump Status, and Age at Diagnosis as Level 2 predictors				
Intercept				
G_{01} Race	1.32	.000		
G_{02} Pump Status	.83	.007		
G_{03} Age at Diagnosis	-.21	.000		
Slope				
G_{11} Race	-.01	.921		
G_{12} Pump Status	.15	.033		
G_{13} Age at Diagnosis	.01	.133		
Intercept			1.49	.000
Slope			.11	.000

Note: Caucasians were coded as 1 and African Americans were coded as 2. Females were coded as 1 and males as 2. Use of insulin pump was coded as 1 and no pump therapy as 2.

Table 7
HLM Estimates of Fixed and Random Effects for Race and Neighborhood SES

	Coefficient	p	Variance Component	p
Model 6: Median Family Income and Percentage of Minority Population as Level 2 predictors				
Intercept				
G_{01} Race	1.34	.000		
G_{02} Median Family Income	-.00	.048		
G_{03} % Minority Population	-.00	.547		
G_{04} Pump Status	.89	.002		
G_{05} Age at Diagnosis	-.20	.000		
Slope				
G_{11} Race	.03	.656		
G_{12} Median Family Income	-.00	.265		
G_{13} % Minority Population	-.00	.242		
G_{14} Pump Status	.13	.087		
G_{15} Age at Diagnosis	.01	.182		
Intercept			1.45	.000
Slope			.11	.000

Note: Caucasians were coded as 1 and African Americans were coded as 2. Use of insulin pump was coded as 1 and no pump therapy as 2.

Table 8
HLM Estimates of Fixed and Random Effects of Race and Family Structure

	Coefficient	p	Variance Component	p
Model 7: Family Structure as only Level 2 predictor				
$G_{01\ (intercept)}$	-.37	.182	2.68	.000
$G_{11\ (slope)}$	-.10	.128	.11	.000
Model 8: Race and Family Structure as Level 2 predictors				
Intercept				
G_{01} Race	1.36	.000		
G_{02} Family Structure	-.21	.414		
Slope				
G_{11} Race	.00	.975		
G_{12} Family Structure	-.09	.168		
Intercept			2.10	.000
Slope			.11	.000

Note: Caucasians were coded as 1 and African Americans as 2. Single-parent households were coded as 1 and two-parent households as 2.

Table 9
HLM Estimates of Fixed and Random Effects for the Quadratic Model

	Coefficient	p	Variance Component	p
Model 9: Quadratic Model with Race, Pump Status, and Age at Diagnosis as Level 2 predictors				
Intercept				
B_{00}	5.44	.000		
G_{01} Race	1.29	.000		
G_{02} Pump status	.79	.012		
G_{03} Age at diagnosis	-.22	.000		
Linear Slope				
B_{10}	-.06	.622		
G_{11} Race	.00	.962		
G_{12} Pump status	.17	.019		
G_{13} Age at diagnosis	.01	.395		
Quadratic Slope				
B_{20}	-.00	.903		
G_{21} Race	-.01	.395		
G_{22} Pump status	.01	.520		
G_{23} Age at diagnosis	.00	.655		
Intercept			1.89	.000
Linear Slope			.11	.000
Quadratic Slope			.00	.000

Note: Caucasians were coded as 1 and African Americans were codes as 2. Use of insulin pump was coded as 1 and no pump therapy as 2.

Table 10
HLM Estimates of Fixed and Random Effects for Pump Status across time

	Coefficient	p	Variance Component	p
Model 10: Pump Status as Level 1 Time-varying Covariate				
$B_{00\ (intercept)}$	8.97	.000	2.52	.000
$B_{10\ (pump\ slope)}$	-.85	.000	.64	.055
$B_{20\ (slope)}$	.23	.000	.10	.000

Note: These analyses are replications of primary analyses with pump status as a level 1 predictor instead of a level 2 predictor.

Table 11
HLM Estimates of Fixed and Random Effects for Race and Covariates with Pump Status across time

	Coefficient	p	Variance Component	p
Model 11: Race and Age at Diagnosis as Level 2 predictors with Pump Status as Level 1 Time-varying Covariate				
Intercept				
B_{00}	6.77	.000		
G_{01} Race	1.44	.000		
G_{02} Age at diagnosis	-.21	.000		
Pump Slope				
B_{10}	.59	.305		
G_{11} Race	-1.06	.017		
G_{12} Age at diagnosis	.14	.069		
Slope				
B_{20}	.24	.009		
G_{21} Race	.00	.916		
G_{22} Age at diagnosis	.01	.142		
Intercept			1.50	.000
Pump Slope			.57	.024
Slope			.11	.000

Note: These analyses are replications of primary analyses with pump status as a level 1 predictor instead of a level 2 predictor.

Table 12
HLM Estimates of Fixed and Random Effects for Race and Neighborhood SES with Pump Status across time

	Coefficient	p	Variance Component	p
Model 12: Median Family Income and Percentage of Minority Population as Level 2 predictors with Pump Status as Level 1 Time-varying Covariate				
Intercept				
B_{00}	6.62	.000		
G_{01} Race	1.52	.000		
G_{02} Median family income	-.00	.056		
G_{03} Percent minority	-.01	.357		
G_{04} Age at diagnosis	-.20	.000		
Pump Slope				
B_{10}	.88	.087		
G_{11} Race	-1.27	.001		
G_{12} Median family income	.00	.294		
G_{13} Percent minority	.02	.020		
G_{14} Age at diagnosis	.11	.074		
Slope				
B_{20}	.18	.119		
G_{21} Race	.05	.538		
G_{22} Median family income	-.00	.219		
G_{23} Percent minority	-.00	.177		
G_{24} Age at diagnosis	.014	.169		
Intercept			1.50	.000
Pump Slope			.72	.019
Slope			.12	.000

Note: These analyses are replications of primary analyses with pump status as a level 1 predictor instead of a level 2 predictor.

APPENDIX B
Figures

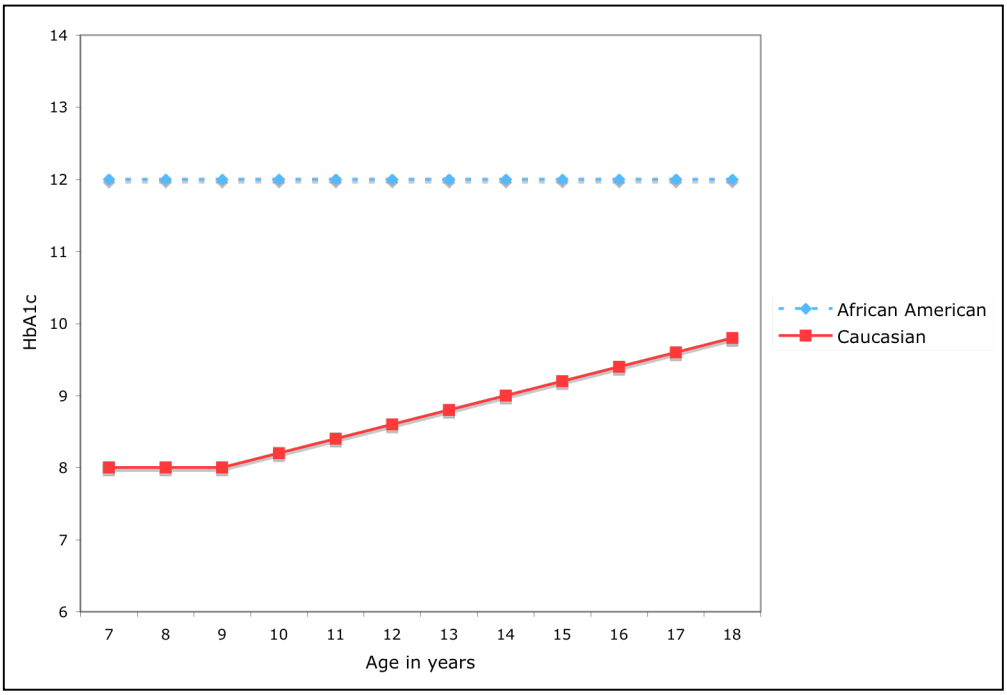

Figure 1. Hypothetical developmental trajectories of African American and Caucasian youth across adolescence based on the Childhood Risk Model.

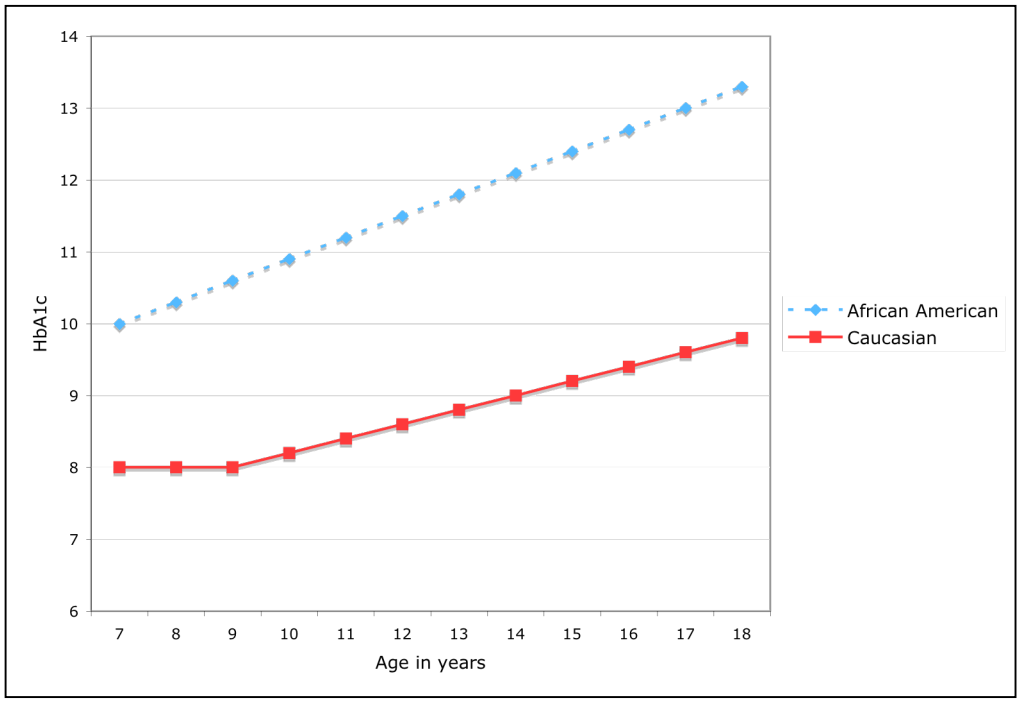

Figure 2. Hypothetical developmental trajectories of African American and Caucasian youth across adolescence based on the Differential Adolescent Risk Model.

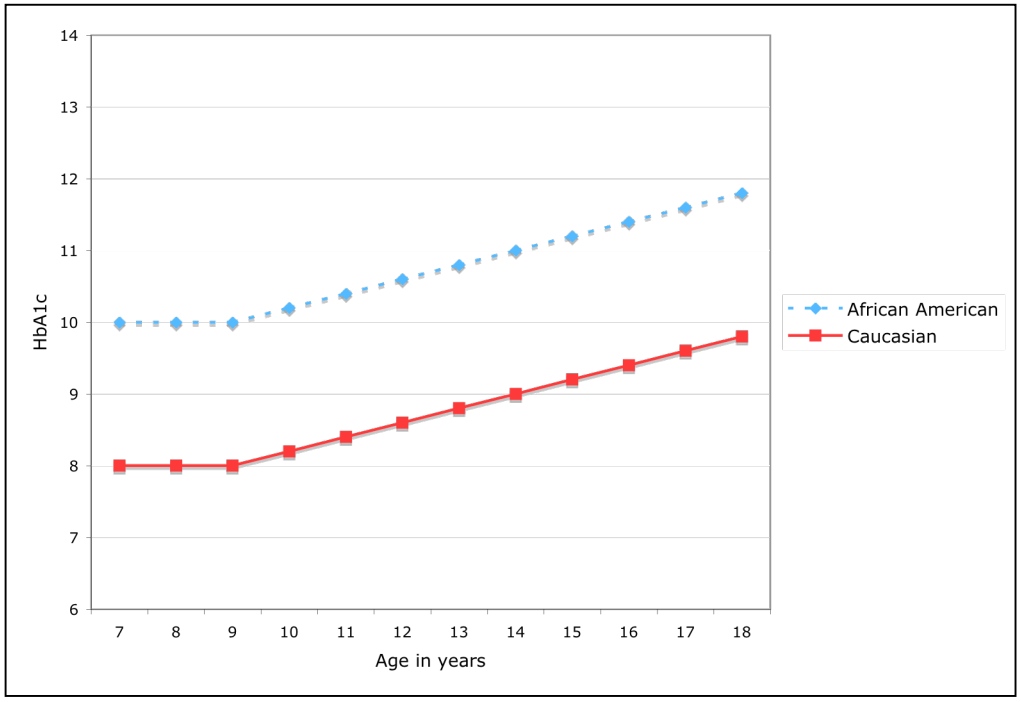

Figure 3. Hypothetical developmental trajectories of African American and Caucasian youth across adolescence based on the Parallel Deterioration Model.

REFERENCES

Altobelli, E., Valenti, M., Verrotti, A., Masedu, F., Tiberti, S., Chiarelli, F., et al. (2000). Family and disease management in young type 1 diabetic patients. *Acta Diabetologica, 37*(4), 173-178.

American Diabetes Association (ADA). (2003). Economic costs of diabetes in the U.S. in 2002. *Diabetes Care, 26*(3), 917-932.

American Diabetes Association (ADA). (2007). Standards of medical care in diabetes--2007. *Diabetes Care, 30* (suppl_1), S4-41.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. Washington, DC: American Psychiatric Association.

Amiel, S. A., Sherwin, R. S., Simonson, D. C., Lauritano, A. A., & Tamborlane, W. V. (1986). Impaired insulin action in puberty. A contributing factor to poor glycemic control in adolescents with diabetes. *N Engl J Med, 315*(4), 215-219.

Anderson, B. J., Auslander, W. F., Jung, K. C., Miller, J. P., & Santiago, J. V. (1990). Assessing family sharing of diabetes responsibilities. *J. Pediatr. Psychol., 15*(4), 477-492.

Anderson, B. J., Brackett, J., Ho, J., & Laffel, L. M. (1999). An office-based intervention to maintain parent-adolescent teamwork in diabetes

management. Impact on parent involvement, family conflict, and subsequent glycemic control. *Diabetes Care, 22*(5), 713-721.

Anderson, B., Ho, J., Brackett, J., Finkelstein, D., & Laffel, L. (1997). Parental involvement in diabetes management tasks: relationships to blood glucose monitoring adherence and metabolic control in young adolescents with insulin-dependent diabetes mellitus. *Journal of Pediatrics, 130*(2), 257-265.

Anderson, B. J., Vangsness, L., Connell, A., Butler, D., Goebel-Fabbri, A., & Laffel, L. M. (2002). Family conflict, adherence, and glycaemic control in youth with short duration Type 1 diabetes. *Diabet Med, 19*(8), 635-642.

Arfken, C. L., Reno, P. L., Santiago, J. V., & Klein, R. (1998). Development of proliferative diabetic retinopathy in African-Americans and whites with type 1 diabetes. *Diabetes Care, 21*(5), 792-795.

Atkinson, M. A., & Maclaren, N. K. (1994). The pathogenesis of insulin-dependent diabetes mellitus. *N Engl J Med, 331*(21), 1428-1436.

Auslander, W. F., Thompson, S., Dreitzer, D., White, N. H., & Santiago, J. V. (1997). Disparity in glycemic control and adherence between African-American and Caucasian youths with diabetes. Family and community contexts. *Diabetes Care, 20*(10), 1569-1575.

Barnes, G., Hoffman, J., Welte, J., Farrell, M., & Dintcheff, B. (2007). Adolescents time use: Effects on substance use, delinquency and sexual activity. *Journal of Youth and Adolescence, 36*, 697-710.

Bearman, K. J., & La Greca, A. M. (2002). Assessing friend support of adolescents' diabetes care: the diabetes social support questionnaire-friends version. *J Pediatr Psychol, 27*(5), 417-428.

Berg, C. A., Wiebe, D. J., Beveridge, R. M., Palmer, D. L., Korbel, C. D., Upchurch, R., et al. (2007). Mother-Child Appraised Involvement in Coping with Diabetes Stressors and Emotional Adjustment. *Journal of Pediatric Psychology Special Section: Broadening the Scope of Practice and Research in Pediatric Psychology, 32*(8), 995-1005.

Bloch, C. A., Clemons, P., & Sperling, M. A. (1987). Puberty decreases insulin sensitivity. *J Pediatr, 110*(3), 481-487.

Blustein, J. (1994). The reliability of racial classifications in hospital discharge abstract data. *Am J Public Health, 84*(6), 1018-1021.

Boehmer, U., Kressin, N. R., Berlowitz, D. R., Christiansen, C. L., Kazis, L. E., & Jones, J. A. (2002). Self-reported vs administrative race/ethnicity data and study results. *Am J Public Health, 92*(9), 1471-1472.

Bosnyak, Z., Nishimura, R., Hagan Hughes, M., Tajima, N., Becker, D., Tuomilehto, J., et al. (2005). Excess mortality in Black compared with

White patients with Type 1 diabetes: an examination of underlying causes. *Diabet Med, 22*(12), 1636-1641.

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annu Rev Psychol, 53*, 371-399.

Braveman, P. A., Cubbin, C., Egerter, S., Chideya, S., Marchi, K. S., Metzler, M., et al. (2005). Socioeconomic status in health research: One size does not fit all. *Jama, 294*(22), 2879-2888.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models : applications and data analysis methods*. Newbury Park: Sage Publications.

Bulcroft, K. A., Carmody, D. C., & Bulcroft, R. A. (1996). Patterns of parental independence giving to adolescents: Variations by race, age, and gender of child. *Journal of Marriage and Family, 58*(4), 866-883.

Burroughs, T. E., Harris, M. A., Pontious, S. L., & Santiago, J. V. (1997). Research on social support in adolescents with IDDM: a critical review. *Diabetes Educ, 23*(4), 438-448.

Burton, L. M., & Jarrett, R. L. (2000). In the mix, yet on the margins: The place of families in urban neighborhood and child development research. *Journal of Marriage and Family, 62*(4), 1114-1135.

Butler, J. M., Skinner, M., Gelfand, D., Berg, C. A., & Wiebe, D. J. (2007). Maternal Parenting Style and Adjustment in Adolescents with Type I Diabetes. *J Pediatr Psychol, 32*(10), 1227-1237.

Cameron, L. D., Young, M. J., & Wiebe, D. J. (2007). Maternal Trait Anxiety and Diabetes Control in Adolescents with Type 1 Diabetes. *Journal of Pediatric Psychology 32*(7), 733-744.

Caughy, M. O., Nettles, S. M., O'Campo, P. J., & Lohrfink, K. F. (2006). Neighborhood matters: Racial socialization of African American children. *Child Dev, 77*(5), 1220-1236.

Celano, M. P. (2006). Family processes in pediatric asthma. *Curr Opin Pediatr, 18*(5), 539-544.

Centers for Disease Control and Prevention (CDC). (2003). National diabetes fact sheet United States. Retrieved June 28, 2008, from http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2003.pdf.

Centers for Disease Control and Prevention (CDC). (2008). Body mass index. Retrieved August 21, 2008, from http://www.cdc.gov/nccdphp/dnpa/healthyweight/assessing/bmi/index.htm .

Ceriello, A., Ihnat, M. A., & Thorpe, J. E. (2009). Clinical review 2: The "metabolic memory": is more than just tight glucose control necessary to prevent diabetic complications? *J Clin Endocrinol Metab, 94*(2), 410-415.

Chalew, S. A., Gomez, R., Butler, A., Hempe, J., Compton, T., Mercante, D., et al. (2000). Predictors of glycemic control in children with type 1 diabetes: The importance of race. *J Diabetes Complications, 14*(2), 71-77.

Chen, E. (2004). Why socioeconomic status affects the health of children: A psychosocial perspective. *Current Directions in Psychological Science, 13*(3), 112-115.

Chen, E., Hanson, M. D., Paterson, L. Q., Griffin, M. J., Walker, H. A., & Miller, G. E. (2006). Socioeconomic status and inflammatory processes in childhood asthma: The role of psychological stress. *J Allergy Clin Immunol, 117*(5), 1014-1020.

Chen, E., Martin, A. D., & Matthews, K. A. (2006). Socioeconomic status and health: Do gradients differ within childhood and adolescence? *Soc Sci Med, 62*(9), 2161-2170.

Chen, E., Martin, A. D., & Matthews, K. A. (2007). Trajectories of Socioeconomic Status Across Children's Lifetime Predict Health. *Pediatrics, 120*(2), e297-303.

Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychol Bull, 128*(2), 295-329.

Chen, E., & Paterson, L. Q. (2006). Neighborhood, family, and subjective socioeconomic status: How do they relate to adolescent health? *Health Psychol, 25*(6), 704-714.

Christen, A., Efstathiadou, Z., Laspa, E., Johnston, D. G., & Godsland, I. F. (2007). Rate of change and instability in body mass index, insulin resistance, and lipid metabolism as predictors of atherosclerotic vascular disease. *J Clin Endocrinol Metab, 92*(10), 3780-3787.

Cogen, F. R., Henderson, C., Hansen, J. A., & Streisand, R. (2007). Pediatric quality of life in transitioning to the insulin pump: Does prior regimen make a difference? *Clin Pediatr (Phila), 46*(9), 777-779.

Cohen, D. M., Lumley, M. A., Naar-King, S., Partridge, T., & Cakan, N. (2004). Child behavior problems and family functioning as predictors of adherence and glycemic control in economically disadvantaged children with type 1 diabetes: a prospective study. *J Pediatr Psychol, 29*(3), 171-184.

Currie, J., & Stabile, M. (2003). Socioeconomic Status and Child Health: Why Is the Relationship Stronger for Older Children? *American Economic Review, 93*(5), 1813-1823.

Dabelea, D., Bell, R. A., D'Agostino, R. B., Jr., Imperatore, G., Johansen, J. M., Linder, B., et al. (2007). Incidence of diabetes in youth in the United States. *Jama, 297*(24), 2716-2724.

Davis, C. L., Delamater, A. M., Shaw, K. H., La Greca, A. M., Eidson, M. S., Perez-Rodriguez, J. E., et al. (2001). Parenting styles, regimen adherence, and glycemic control in 4- to 10-year-old children with diabetes. *J Pediatr Psychol, 26*(2), 123-129.

Delamater, A. M. (2007). Psychological care of children and adolescents with diabetes. *Pediatr Diabetes, 8*(5), 340-348.

Delamater, A. M., Albrecht, D. R., Postellon, D. C., & Gutai, J. P. (1991). Racial differences in metabolic control of children and adolescents with type I diabetes mellitus. *Diabetes Care, 14*(1), 20-25.

Delamater, A. M., Shaw, K. H., Applegate, E. B., Pratt, I. A., Eidson, M., Lancelotta, G. X., et al. (1999). Risk for metabolic control problems in minority youth with diabetes. *Diabetes Care, 22*(5), 700-705.

de Wit, M., Delemarre-van de Waal, H. A., Bokma, J. A., Haasnoot, K., Houdijk, M. C., Gemke, R. J., et al. (2007). Self-report and parent-report of physical and psychosocial well-being in Dutch adolescents with type 1 diabetes in relation to glycemic control. *Health Qual Life Outcomes, 5*, 10.

Diabetes Control and Complications Trial Research Group (DCCT). (1993). The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med, 329*(14), 977-986.

DIAMOND Project Group. (2006). Incidence and trends of childhood type 1 diabetes worldwide 1990-1999. *Diabetic Medicine, 23*(8), 857-866.

Diez-Roux, A. V., Kiefe, C. I., Jacobs, D. R., Jr., Haan, M., Jackson, S. A., Nieto, F. J., et al. (2001). Area characteristics and individual-level socioeconomic position indicators in three population-based epidemiologic studies. *Ann Epidemiol, 11*(6), 395-405.

Dorn, L. D., Susman, E. J., & Ponirakis, A. (2003). Pubertal timing and adolescent adjustment and behavior: Conclusions vary by rater. *Journal of Youth and Adolescence, 32*(3), 157-167.

Eeg-Olofsson, K., Cederholm, J., Nilsson, P. M., Gudbjornsdottir, S., & Eliasson, B. for the Steering Committee of the Swedish National Diabetes Register. (2007). Glycemic and risk factor control in Type 1 Diabetes: Results from 13,612 patients in a national diabetes register. *Diabetes Care, 30*(3), 496-502.

Ellis, D. A., Templin, T., Naar-King, S., Frey, M. A., Cunningham, P. B., Podolski, C. L., et al. (2007). Multisystemic therapy for adolescents with poorly controlled type I diabetes: Stability of treatment effects in a randomized controlled trial. *J Consult Clin Psychol, 75*(1), 168-174.

EURODIAB ACE Study Group. (2000). Variation and trends in incidence of childhood diabetes in Europe. *Lancet, 355*(9207), 873-876.

Frey, M. A., Templin, T., Ellis, D., Gutai, J., & Podolski, C. L. (2007). Predicting metabolic control in the first 5 yr after diagnosis for youths with type 1 diabetes: the role of ethnicity and family structure. *Pediatr Diabetes, 8*(4), 220-227.

Garcia Coll, C., Lamberty, G., Jenkins, R., McAdoo, H. P., Crnic, K., Wasik, B. H., et al. (1996). An integrative model for the study of developmental competencies in minority children. *Child Dev, 67*(5), 1891-1914.

Gary, T. L., McGuire, M., McCauley, J., & Brancati, F. L. (2004). Racial comparisons of health care and glycemic control for African American and white diabetic adults in an urban managed care organization. *Dis Manag, 7*(1), 25-34.

Geronimus, A. T., & Bound, J. (1998). Use of census-based aggregate variables to proxy for socioeconomic group: evidence from national samples. *Am J Epidemiol, 148*(5), 475-486.

Goldston, D. B., Kovacs, M., Obrosky, D. S., & Iyengar, S. (1995). A longitudinal study of life events and metabolic control among youths with insulin-dependent diabetes mellitus. *Health Psychol, 14*(5), 409-414.

Gomez, S. L., Kelsey, J. L., Glaser, S. L., Lee, M. M., & Sidney, S. (2005). Inconsistencies between self-reported ethnicity and ethnicity recorded in a health maintenance organization. *Annals of Epidemiology, 15*(1), 71-79.

Goran, M. I., & Gower, B. A. (2001). Longitudinal study on pubertal insulin resistance. *Diabetes, 50*(11), 2444-2450.

Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (2006). Pubertal effects on adjustment in girls: Moving from demonstrating effects to identifying pathways. *Journal of Youth and Adolescence, 35*(3), 391-401.

Greco, P., Pendley, J. S., McDonell, K., & Reeves, G. (2001). A peer group intervention for adolescents with type 1 diabetes and their best friends. *J Pediatr Psychol, 26*(8), 485-490.

Gutman, L. M., & Eccles, J. S. (2007). Stage-environment fit during adolescence: trajectories of family relations and adolescent outcomes. *Dev Psychol, 43*(2), 522-537.

Hains, A. A., Berlin, K. S., Davies, W. H., Parton, E. A., & Alemzadeh, R. (2006). Attributions of adolescents with type 1 diabetes in social situations: Relationship with expected adherence, diabetes stress, and metabolic control. *Diabetes Care, 29*(4), 818-822.

Hains, A. A., Berlin, K. S., Davies, W. H., Smothers, M. K., Sato, A. F., & Alemzadeh, R. (2007). Attributions of adolescents with type 1 diabetes related to performing diabetes care around friends and peers: the moderating role of friend support. *J Pediatr Psychol, 32*(5), 561-570.

Hanaire-Broutin, H., Melki, V., Bessieres-Lacombe, S., & Tauber, J. P. (2000). Comparison of continuous subcutaneous insulin infusion and multiple

daily injection regimens using insulin lispro in type 1 diabetic patients on intensified treatment: a randomized study. The Study Group for the Development of Pump Therapy in Diabetes. *Diabetes Care, 23*(9), 1232-1235.

Hanson, C. L., Henggeler, S. W., & Burghen, G. A. (1987). Race and sex differences in metabolic control of adolescents with IDDM: a function of psychosocial variables? *Diabetes Care, 10*(3), 313-318.

Hauser, S. T., Jacobson, A. M., Lavori, P., Wolfsdorf, J. I., Herskowitz, R. D., Milley, J. E., et al. (1990). Adherence among children and adolescents with insulin-dependent diabetes mellitus over a four-year longitudinal follow-up: II. Immediate and long-term linkages with the family milieu. *J Pediatr Psychol, 15*(4), 527-542.

Hauser, S. T., Liebman, W., Houlihan, J., Powers, S. I., Jacobson, A. M., Noam, G. G., et al. (1985). Family contexts of pubertal timing. *Journal of Youth and Adolescence, 14*(4), 1573-6601.

Helgeson, V. S., Siminerio, L., Escobar, O., & Becker, D. (2009). Predictors of metabolic control among adolescents with diabetes: a 4-year longitudinal study. *J Pediatr Psychol, 34*(3), 254-270.

Hoey, H., Aanstoot, H. J., Chiarelli, F., Daneman, D., Danne, T., Dorchy, H., et al. (2001). Good metabolic control is associated with better quality of life

in 2,101 adolescents with type 1 diabetes. *Diabetes Care, 24*(11), 1923-1928.

Hood, K. K., Butler, D., Huestis, S., Volkening, L., Maher, A., & Laffel, L. M. B. (2006). Depressive symptoms in children and adolescents with type 1 diabetes. *Diabetes Care*, 29(6), 1389-1391.

Hyndman, J. C., Holman, C. D., Hockey, R. L., Donovan, R. J., Corti, B., & Rivera, J. (1995). Misclassification of social disadvantage based on geographical areas: comparision of postcode and collector's district analyses. *Int J Epidemiol, 24*(1), 165-176.

Johnson, S. B., Kelly, M., Henretta, J. C., Cunningham, W. R., Tomer, A., & Silverstein, J. H. (1992). A longitudinal analysis of adherence and health status in childhood diabetes. *J Pediatr Psychol, 17*(5), 537-553.

Joseph, J. (1995). Juvenile delinquency among African Americans. *Journal of Black Studies, 25*(4), 475-491.

Julian, T. W., McKenry, P. C., & McKelvey, M. W. (1994). Cultural variations in parenting: Perceptions of Caucasian, African-American, Hispanic, and Asian-American parents. *Family Relations, 43*(1), 30-37.

Kaplowitz, P. B., Slora, E. J., Wasserman, R. C., Pedlow, S. E., & Herman-Giddens, M. E. (2001). Earlier onset of puberty in girls: relation to increased body mass index and race. *Pediatrics, 108*(2), 347-353.

Kaslow, N. J. (2000). The Efficacy of a Pilot Family Psychoeducational Intervention for Pediatric Sickle Cell Disease. *Families, Systems & Health: The Journal of Collaborative Family HealthCare, 18*(4), 381.

Kaslow, N. J., Collins, M. H., Loundy, M. R., Brown, F., Hollins, L. D., & Eckman, J. (1997). Empiricaly Validated Family Intervetions for Pediatric Psychology: Sickle Cell Disease as an Exemplar. *J. Pediatr. Psychol., 22*(2), 213-227.

Koenig, R. J., Peterson, C. M., Jones, R. L., Saudek, C., Lehrman, M., & Cerami, A. (1976). Correlation of glucose regulation and hemoglobin AIc in diabetes mellitus. *N Engl J Med, 295*(8), 417-420.

Korbel, C. D., Wiebe, D. J., Berg, C. A., & Palmer, D. L. (2007). Gender Differences in adherence to type 1 diabetes management across adolescence: The mediating role of depression. *Children's Health Care, 36*(1), 83-98.

Kotchick, B. A., & Forehand, R. (2002). Putting Parenting in Perspective: A Discussion of the Contextual Factors That Shape Parenting Practices. *Journal of Child & Family Studies, 11*(3), 255-269.

Kovacs, M., Goldston, D., Obrosky, D. S., & Bonar, L. K. (1997). Psychiatric disorders in youths with IDDM: Rates and risk factors. *Diabetes Care, 20*(1), 36-44.

Krieger, N. (1992). Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am J Public Health, 82*(5), 703-710.

Krieger, N., Chen, J. T., Waterman, P. D., Rehkopf, D. H., & Subramanian, S. V. (2003). Race/ethnicity, gender, and monitoring socioeconomic gradients in health: a comparison of area-based socioeconomic measures--the public health disparities geocoding project. *Am J Public Health, 93*(10), 1655-1671.

Kwok, R. K., & Yankaskas, B. C. (2001). The use of census data for determining race and education as SES indicators: a validation study. *Ann Epidemiol, 11*(3), 171-177.

Ladrine, H., & Klonoff, E. A. (2001). Cultural diversity and health psychology. In A. Baum, T. A. Revenson, & J. E. Singer (Eds.). Handbook of Health Psychology (pp. 851-891). New Jersey: Lawrence Erlbaumm Associates, Inc.

La Greca, A. M., Swales, T., Klemp, S., Madigan, S., & Skyler, J. (1995). Adolescents With Diabetes: Gender Differences in Psychosocial Functioning and Glycemic Control. *Children's Health Care, 24*(1), 61.

Laporte, R. E., Matsushima, M., & Chang, Y. (1995). Prevalence and incidence of insulin-dependent diabetes. In *Diabetes in America* (pp. 37-46): National

Institutes of Health National Institutes of Diabetes and Digestive and Kidney Disease.

Larson, R., & Richards, M. H. (1991). Daily companionship in late childhood and early adolescence: Changing developmental contexts. *Child Development, 62*(2), 284-300.

LaViest, T. A., Nickerson, K. J., & Bowie, J. V. (2000). Attitudes about racism, medical mistrust, and satisfaction with care among African American and White cardiac patients. *Medical Care Research and Review, 57, 146-161.*

Lernmark, B., Persson, B., Fisher, L., & Rydelius, P. A. (1999). Symptoms of depression are important to psychological adaptation and metabolic control in children with diabetes mellitus. *Diabet Med, 16*(1), 14-22.

Lewin, A. B., Heidgerken, A. D., Geffken, G. R., Williams, L. B., Storch, E. A., Gelfand, K. M., et al. (2006). The Relation Between Family Factors and Metabolic Control: The Role of Diabetes Adherence. *Journal of Pediatric Psychology, 31*(2), 174-183.

Lipman, T. H., Chang, Y., & Murphy, K. M. (2002). The epidemiology of type 1 diabetes in children in Philadelphia 1990-1994: Evidence of an epidemic. *Diabetes Care, 25*(11), 1969-1975.

Lipton, R., Good, G., Mikhailov, T., Freels, S., & Donoghue, E. (1999). Ethnic differences in mortality from insulin-dependent diabetes mellitus among people less than 25 years of age. *Pediatrics, 103*(5 Pt 1), 952-956.

Mansfield, A., Addis, M., Laffel, L., & Anderson, B. (2004). Gender differences in reports of self-reliance for diabetes tasks in a pediatric sample. *International Journal of Men's Health, 3*(1), 61-66.

McBride, C. K., Paikoff, R. L., & Holmbeck, G. N. (2003). Individual and familial influences on the onset of sexual intercourse among urban African American adolescents. *J Consult Clin Psychol, 71*(1), 159-167.

McCrimmon, R. J., Gold, A. E., Deary, I. J., Kelnar, C. J., & Frier, B. M. (1995). Symptoms of hypoglycemia in children with IDDM. *Diabetes Care, 18*(6), 858-861.

McGrath, J. J., Matthews, K. A., & Brady, S. S. (2006). Individual versus neighborhood socioeconomic status and race as predictors of adolescent ambulatory blood pressure and heart rate. *Soc Sci Med, 63*(6), 1442-1453.

Musey, V. C., Lee, J. K., Crawford, R., Klatka, M. A., McAdams, D., & Phillips, L. S. (1995). Diabetes in urban African-Americans. I. Cessation of insulin therapy is the major precipitating cause of diabetic ketoacidosis. *Diabetes Care, 18*(4), 483-489.

Naar-King, S., Podolski, C. L., Ellis, D. A., Frey, M. A., & Templin, T. (2006). Social ecological model of illness management in high-risk youths with type 1 diabetes. *J Consult Clin Psychol, 74*(4), 785-789.

Nathan, D. M., Singer, D. E., Hurxthal, K., & Goodson, J. D. (1984). The clinical information value of the glycosylated hemoglobin assay. *N Engl J Med, 310*(6), 341-346.

Nolen-Hoeksema, S., & Girgus, J. S. (1994). The emergence of gender differences in depression during adolescence. *Psychol Bull, 115*(3), 424-443.

Oldroyd, J., Banerjee, M., Heald, A., & Cruickshank, K. (2005). Diabetes and ethnic minorities. *Postgrad Med J, 81*(958), 486-490.

O'Sullivan, L. F., Meyer-Balhburg, H. F., & Watkins, B. X. (2000). Social cognitions associated with pubertal development in a sample of urban, low-income, African-American and Latina girls and mothers. *J Adolesc Health, 27*(4), 227-235.

Paikoff, R. L., & Brooks-Gunn, J. (1991). Do parent-child relationships change during puberty? *Psychol Bull, 110*(1), 47-66.

Palmer, D. L., Berg, C. A., Wiebe, D. J., Beveridge, R. M., Korbel, C. D., Upchurch, R., et al. (2004). The role of autonomy and pubertal status in understanding age differences in maternal involvement in diabetes responsibility across adolescence. *J Pediatr Psychol, 29*(1), 35-46.

Papas, M. A., Alberg, A. J., Ewing, R., Helzlsouer, K. J., Gary, T. L., & Klassen, A. C. (2007). The built environment and obesity. *Epidemiol Rev, 29*(1), 129-143.

Patino, A. M., Sanchez, J., Eidson, M., & Delamater, A. M. (2005). Health beliefs and regimen adherence in minority adolescents with type 1 diabetes. *J Pediatr Psychol, 30*(6), 503-512.

Pendley, J. S., Kasmen, L. J., Miller, D. L., Donze, J., Swenson, C., & Reeves, G. (2002). Peer and family support in children and adolescents with type 1 diabetes. *J Pediatr Psychol, 27*(5), 429-438.

Porte, D., Jr., & Schwartz, M. W. (1996). Diabetes complications: Why is glucose potentially toxic? *Science, 272*(5262), 699-700.

Povlsen, L., Olsen, B., & Ladelund, S. (2005). Diabetes in children and adolescents from ethnic minorities: Barriers to education, treatment and good metabolic control. *J Adv Nurs, 50*(6), 576-582.

Raudenbush, S., Bryk, A., Cheong Y., Congdon R. (2004). HLM 6: Hierarchical linear and nonlinear modeling. Chicago, IL: Scientific Software International.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychol Bull, 128*(2), 330-366.

Sagrestano, L. M., McCormick, S. H., Paikoff, R. L., & Holmbeck, G. N. (1999). Pubertal development and parent-child conflict in low-income, urban, African American adolescents. *Journal of Research on Adolescence, 9*(1), 85 - 107.

Saha, S., Komaromy, M., Koepsell, T. D., & Bindman, A. B. (1999). Patient-physician racial concordance and the perceived quality and use of health care. *Arch Intern Med, 159*(9), 997-1004.

Scott, C. R., Smith, J. M., Cradock, M. M., & Pihoker, C. (1997). Characteristics of youth-onset Noninsulin-Dependent Diabetes Mellitus and Insulin-dependent Diabetes Mellitus at diagnosis. *Pediatrics, 100*(1), 84-91.

Search for Diabetes in Youth Study Group. (2006). The burden of diabetes mellitus among US youth: Prevalence estimates from the SEARCH for diabetes in youth study. *Pediatrics, 118*(4), 1510-1518.

Search for Diabetes in Youth Study Group. (2007). Incidence of diabetes in youth in the United States. *JAMA, 297*(24), 2716-2724.

Seiffge-Krenke, I., & Stemmler, M. (2003). Coping with everyday stress and links to medical and psychosocial adaptation in diabetic adolescents. *J Adolesc Health, 33*(3), 180-188.

Singer, J. D., & Willett, J. B. (2003). Applied longitudinal data analysis. New York: Oxford University Press.

Smetana, J. G., Daddis, C., & Chuang, S. S. (2003). "Clean your Room!": A Longitudinal Investigation of Adolescent-Parent Conflict and Conflict Resolution in Middle-Class African American Families. *Journal of Adolescent Research, 18*(6), 631-650.

Smetana, J., & Gaines, C. (1999). Adolescent-Parent Conflict in Middle-Class African American Families. *Child Development, 70*(6), 1447.

Soliman, A. T., Salmi, I. A., & Asfour, M. (1997). Mode of presentation and progress of childhood diabetes mellitus in the Sultanate of Oman. *J Trop Pediatr, 43*(3), 128-132.

Soobader, M., LeClere, F. B., Hadden, W., & Maury, B. (2001). Using aggregate geographic data to proxy individual socioeconomic data: does size matter? *Am J Public Health, 91*(4), 632-636.

Stanton, B., Li, X., Pack, R., Cottrell, L., Harris, C., & Burns, J. M. (2002). Longitudinal influence of perceptions of peer and parental factors on African American adolescent risk involvement. *Journal of Urban Health: Bulletin of the New York Academy of Medicine, 79*, 536-548.

Steinberg, L. D. (1981). Transformations in family relations at puberty. *Developmental Psychology, 17*(6), 833-840.

Stewart, S. M., Rao, U., Emslie, G. J., Klein, D., & White, P. C. (2005). Depressive symptoms predict hospitalization for adolescents with type 1 diabetes mellitus. *Pediatrics, 115*(5), 1315-1319.

Summerson, J. H., Konen, J. C., & Dignan, M. B. (1992). Race-related differences in metabolic control among adults with diabetes. *South Med J, 85*(10), 953-956.

Sun, S. S., Schubert, C. M., Chumlea, W. C., Roche, A. F., Kulin, H. E., Lee, P. A., et al. (2002). National estimates of the timing of sexual maturation and racial differences among US children. *Pediatrics, 110*(5), 911-919.

Taylor, S. E., Repetti, R. L., & Seeman, T. (1997). Health psychology: What is an unhealthy environment and how does it get under the skin? *Annual Review of Psychology, 48*(1), 411-447.

Thomas, A. M., Peterson, L., & Goldstein, D. (1997). Problem solving and diabetes regimen adherence by children and adolescents with IDDM in social pressure situations: A reflection of normal development. *J. Pediatr. Psychol., 22*(4), 541-561.

Thompson, S. J., Auslander, W. F., & White, N. H. (2001). Comparison of single-mother and two-parent families on metabolic control of children with diabetes. *Diabetes Care, 24*(2), 234-238.

Tull, E. S., & Barinas, E. (1996). A twofold excess mortality among black compared with white IDDM patients in Allegheny county, Pennsylvania. Pittsburgh DERI Mortality Study Group. *Diabetes Care, 19*(12), 1344-1347.

United States Census Bureau. (2003). Marital status: 2000. Retrieved July 11, 2008, from http://www.census.gov/prod/2003pubs/c2kbr-30.pdf.

United States Census Bureau (2004). Educational attainment in the United States: 2003. Retrieved July 2, 2008 from http://www.census.gov/prod/2004pubs/p20-550.pdf.

United States Census Bureau. (2005). 2005 Annual social and economic supplement. Retrieved July 2, 2008 from http://pubdb3.census.gov/macro/032005/hhinc/new05_000.htm.

United States Office of Personnel Management (2008). Handbook of Occupational Groups and Families. Retrieved February 3, 2009 from www.opm.gov/FEDCLASS/GSHBKOCC.pdf.

van Ryn, M., & Burke, J. (2000). The effect of patient race and socio-economic status on physicians' perceptions of patients. *Soc Sci Med, 50*(6), 813-828.

Weinick, R. M., Zuvekas, S. H., & Cohen, J. W. (2000). Racial and ethnic differences in access to and use of health care services, 1977 to 1996. *Med Care Res Rev, 57 Suppl 1*, 36-54.

Weissberg-Benchell, J., Glasgow, A. M., Tynan, W. D., Wirtz, P., Turek, J., & Ward, J. (1995). Adolescent diabetes management and mismanagement. *Diabetes Care, 18*(1), 77-82.

West, C. N., Geiger, A. M., Greene, S. M., Harris, E. L., Liu, I. L., Barton, M. B., et al. (2005). Race and ethnicity: comparing medical records to self-reports. *J Natl Cancer Inst Monogr*(35), 72-74.

Wiebe, D. J., Berg, C. A., Korbel, C., Palmer, D. L., Beveridge, R. M., Upchurch, R., et al. (2005). Children's Appraisals of Maternal Involvement in Coping With Diabetes: Enhancing Our Understanding of Adherence, Metabolic Control, and Quality of Life Across Adolescence. *Journal of Pediatric Psychology, 30*(2), 167-178.

Williams, J. H., Van Dorn, R. A., Ayers, C. D., Bright, C. L., Abbott, R. D., & Hawkins, J. D. (2007). Understanding Race and Gender Differences in Delinquent Acts and Alcohol and Marijuana Use: A Developmental Analysis of Initiation. *Social Work Research, 31*(2), 71-81.

Wysocki, T., Greco, P., & Buckloh, L. M. (2003). Childhood diabetes in psychological context. In M. C. Roberts (Ed.), *Handbook of Pediatric Psychology* (3rd ed., pp. 304-320). New York: The Guilford Press.

Wysocki, T., Harris, M. A., Buckloh, L. M., Mertlich, D., Lochrie, A. S., Taylor, A., et al. (2006). Effects of behavioral family systems therapy for diabetes on adolescents' family relationships, treatment adherence, and metabolic control. *J Pediatr Psychol, 31*(9), 928-938.

Wysocki, T., Taylor, A., Hough, B. S., Linscheid, T. R., Yeates, K. O., & Naglieri, J. A. (1996). Deviation from developmentally appropriate self-care autonomy. Association with diabetes outcomes. *Diabetes Care, 19*(2), 119-125.