EFFECT OF LONGITUDINAL GROWTH TRAJECTORIES OF METABOLIC CONTROL ON ACADEMIC ACHIEVEMENT OF ADOLESCENTS WITH TYPE 1 DIABETES

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Abstract

A latent growth model was employed to examine how deteriorating metabolic control may be a limiting factor related to the academic performance of adolescents diagnosed with type 1 diabetes at the end of a three year study. HbA1c was assessed by medical clinic staff every 6 months and final Grade Point Average (GPA) scores were collected from school records for a sample of 217 adolescents (\(M_{\text{baseline age}} = 12.61\) years). Results indicated that worse initial adolescent HbA1c was a significant and meaningful limiting factor of final GPA scores. Metabolic control deteriorated for youth with type 1 during adolescence as HbA1c increased from 8.3 to 8.9%. The deterioration of metabolic control occurred at a faster rate for adolescents with higher initial HbA1c. Metabolic deterioration over time was evidenced; however, deterioration over time was not a meaningful predictor of final GPA scores. Implications include the importance of early health intervention to decrease the risk of disease complications related to poor metabolic control that might impact future academic success.

Keywords: academic performance; adolescence; diabetes; GPA; HbA1c; metabolic control
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CHAPTER 1: INTRODUCTION AND LITERATURE REVIEW

The purpose of this study was to examine how poor metabolic control may be a limiting factor related to the academic performance of adolescents diagnosed with type 1 diabetes at the sampling final time point by employing a latent growth model. Although latent growth models have been conducted to examine health behaviors related to metabolic control (e.g., Rausch et al., 2012), a dearth of longitudinal and latent growth modeling research exists concerning achievement outcomes related to metabolic control for adolescents with type 1 living in the United States (US). The majority of research articles addressing achievement outcomes related to metabolic control for children and adolescents with diabetes have employed a cross-sectional (e.g., McCarthy, Lindgren, Mengeling, Tsalikian, & Engvall, 2003) or case control designs (e.g., Hannonen et al., 2012). More sophisticated studies by McCarthy, Lindgren, Mengeling, Tsalikian, and Engvall, (2002) applied a latent growth model, and Kovacs, Goldston, and Iyengar, 1992 employed a repeated measures design to examine academic trajectories of adolescents living with type 1, but each study had limitations. McCarthy et al. only contrasted achievement score trajectories for youth with type 1 with healthy controls rather than examining changes in academic performance occurring over time for youth with type 1 related to changes in metabolic control. The Kovacs et al. study excluded metabolic control as a predictor as the result of model trimming. Moreover, Kent, Chen, Kumand and Holmes (2010) examined cognition amongst this population and employed a latent growth model. Kent et al. reported a significant effect and an inverse relation between metabolic control trajectories and visual memory scores over a 3-year period. Thus,
studies exploring the impact of metabolic control trajectories on achievement trajectories for youth with type 1 diabetes appear warranted.

Previous research has indicated that adolescents with type 1 diabetes are at-risk for cognitive impairments, learning problems, and poor academic performance (Dalenquest & Kallen, 2007; Fox, Chen, & Holmes, 2003; Gaudieri, Chen, Greer, & Holmes, 2008; Hannonen et al., 2010; Hannonen et al., 2012; Holmes, O’Brien, & Greer, 1995; Kent, Chen, Kumar, & Holmes, 2010; Lin, Northam, Rankins, Werther, & Cameron, 2010; McCarthy et al., 2003; Naguib, Kulinskaya, Lomax, & Garralda, 2009; Parent, Wodrich, & Hasan, 2009; Wodrich, Hasan, & Parent, 2011). Based on 2002-2005 data, 15,600 new cases of type 1 were diagnosed among school-aged youth annually (Centers for Disease Control and Prevention [CDC], 2011). Consequently, there is a high likelihood that educators and school administrators will encounter students and families living with type 1 diabetes during his or her careers.

Accordingly, educators and school administrators need to be aware of challenges faced by students with type 1 diabetes and to receive training in best practices in serving this population. Both educational and health professionals should also be aware that discordance exists within the current literature concerning the impact of diabetes-related complications on student academic achievement. For example, some studies have reported an inverse relation between HbA1c level, a metric for metabolic control, and achievement (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009). Conversely, mean HbA1c across time points was not found to be a significant predictor of learning or achievement in longitudinal regression models (Fox et al., 2003; Hannonen et al., 2012). Unfortunately, predictors such as mean HbA1c or
HbAlc from a single time point might not reveal the magnitude of how fluctuations in metabolic control over time might impact academic performance. This discordance indicates that more research is needed to better understand the nature of the relation between metabolic control and academic outcomes in order to better serve this at-risk student population from both educational and health perspectives.

Further, previous longitudinal studies have indicated metabolic control tends to deteriorate during adolescence for individuals diagnosed with type 1 diabetes (e.g., Rausch et al., 2012). A negative correlation between metabolic control and academic performance might make adolescents with type 1 a particularly vulnerable student population. Thus, examination of achievement trajectories amongst adolescents with type 1 with fluctuating levels of metabolic control might reveal the magnitude of poor metabolic control as a limiting factor related to academic performance.

This literature review is organized based upon the aims of the study and follows a two chapter model. In the first chapter, diabetes will be defined and etiology, physiology, and treatments will be discussed for types 1 and 2. Additionally, rationale will be presented for focusing only on outcomes for youth with type 1. Specifically, (a) the majority of youth diagnosed with diabetes are living with type 1 and (b) failing to separately examine outcomes for youth living with diabetes based upon type potentially limits the generalizability of the findings and makes the interpretation of the results suspect. Finally, the link between poor metabolic control and achievement will be addressed. In the second chapter, methodology of previous studies will be discussed and rationale will be provided for examining the metabolic trajectory and academic outcomes of adolescents with type 1 diabetes.
Diabetes

Diabetes is a disease that occurs when an insufficient amount of insulin is being produced to meet the metabolic demands of the body (American Diabetes Association [ADA], 2012, 2000; Beaser, 2007a; CDC, 2011). Insulin is a critical hormone produced by the Beta cells within the pancreas and regulates blood glucose levels. This hormone signals the body’s cells to allow glucose to enter into the cells to be metabolized for energy (Beaser, 2007a; National Diabetes Education Program [NDEP], 2010).

In a healthy individual, Beta cells respond to any rise in blood glucose levels (e.g., resulting from the digestion of food) with a release of insulin proportional to the body’s needs (Beaser, 2007a). If the Beta cells cannot meet the body’s demand for insulin, the resulting sustained high-levels of blood glucose concentration will lead to deleterious and eventually fatal health effects (CDC, 2011). Sustained high levels of glucose in the blood, termed hyperglycemia, can lead to mild to severe complications including heart disease, hypertension, retinopathy (resulting in severe vision loss), nephropathy (resulting in kidney damage), neuropathy (resulting in damage to the nervous system), periodontal disease; and diabetic ketoacidosis (DKA) which can result in coma and even death (CDC, 2011). Over time, poorly managed diabetes also dramatically increases the risk of stroke, blindness, kidney disease or failure, and nontraumatic lower-limb amputations (CDC, 2011).

Prevalence of Diabetes in Youth
Estimates based on 2010 data indicated that 25.8 million people (8.3% of the population in the US) had diabetes (CDC, 2011), with type 1 comprising 5% of the cases in the total US population (CDC, 2011). A few rarer forms of the condition such as gestational diabetes and Maturity Onset Diabetes in Youth (MODY) have been identified; however, as these are not the focus of the study they will not be described further.

Based on 2010 data, the CDC (2011) estimated that 215,000 youth (< 20 years of age) had either type 1 or 2 diabetes in the US, representing .26% of this age group. The combined prevalence rate for diabetes (types 1 and 2) in the US based on 2001 data is estimated at approximately 1.8 out of every 1,000 youth (SEARCH for Diabetes in Youth Study Group [SEARCH], 2006). When the prevalence of diabetes is examined among the entire population in the US, it appears that type 2 diabetes is most prevalent; however, these proportions are not consistent when examining prevalence rates for youth (SEARCH, 2006). Data from 2002-05 revealed 15,600 new diagnoses of type 1 and 3,600 new diagnoses of type 2 cases among youth annually (CDC, 2011). This finding is consistent with other demographic data (SEARCH, 2006) indicating that type 1 diabetes is the most prevalent form of diabetes in youth.

Although type 1 diabetes was the most common form of diabetes diagnosed in youth overall, health disparities were present in the occurrence of type 2 diabetes amongst youth from ethnic minority groups in the US during mid-puberty (SEARCH, 2006). Specifically, based on 2001 data from the SEARCH group, type 1 accounted for ≥80% of youth 0-9 years of age with diabetes across all ethnic groups (American Indian [AI], Asian/Pacific Islander [API], Hispanic, non-Hispanic black [NHB], non-Hispanic
white [NHW]). In contrast, for youth between the ages of 10-19, type 2 diabetes rates were more variable across ethnic groups (AI = 76%, API = 40%, NHB = 33%, Hispanic = 22%, NHW = 6%). Certainly a high likelihood exists that the rise in incidence of type 2 is related to increasing rates of obesity in youth (ADA; CDC; Rosenbloom & Silverstein).

Additionally, in youth 0-9 years of age, prevalence rates for type 1 and type 2 diabetes for males and females appear equal (SEARCH, 2006). Conversely, in youth 10-19 years of age, NHB and AI females had a higher probability of developing type 2 diabetes than their male counterparts (SEARCH, 2006). Rosenbloom and Silverstein (2003) reported that the sex ratio for type 1 was equal, whereas the sex ratio for type 2 was variable. Specifically, female youth from ethnic minority groups including native North Americans, African-Americans, and Mexican-Americans were at higher risk for developing type 2 diabetes than males (Rosenbloom & Silverstein).

Although differences in prevalence rates between type 1 and type 2 diabetes amongst youth are important to consider, this study will only focus on outcomes for youth with type 1. Rationale for this decision is two pronged. First, as previously described, the majority of youth diagnosed with diabetes are living with type 1 (SEARCH, 2006). Second, studies that attempt to aggregate outcome data and interpret results for combined samples of individuals living with type 1 or type 2 appear suspect. Specifically, given the substantial differences in etiology, physiology, and treatment modalities between type 1 and type 2, failing to differentiate outcomes for participants with type 1 or type 2 potentially limits the generalizability of the findings. Thus, as the majority of youth living with diabetes are living with type 1 (CDC), outcomes for youth
with type 1 will be the focus of this study. Nonetheless, the diversity between the physiologies, etiologies, and treatments of type 1 and type 2 will be addressed in this study for the benefit of the reader.

**Type 1 Diabetes**

**Physiology of type 1.** Type 1 diabetes, formerly referred to as insulin-dependent diabetes mellitus (IDDM) or juvenile-onset diabetes, is an autoimmune disorder that primarily occurs in children and young adults (CDC, 2011). Type 1 occurs when antibodies trigger the immune system to destroy the insulin-producing Beta cells of the pancreas. The destruction of the Beta cells results in the absolute absence of endogenous insulin (ADA, 2012b; ADA, 2000; Beaser, 2007a). Exogenous forms of insulin must be injected for survival (CDC; Beaser). While insulin use can improve health and extend life, insulin is not a cure but a form of treatment that has the potential to lower blood glucose levels to life-threateningly low levels (ADA, 2012a).

**Etiology of type 1.** Type 1 diabetes is considered to be idiopathic (Beaser, 2007a; Rosenbloom & Silverstein, 2003); no cause has been identified and its development appears spontaneous. The most current theory amongst diabetic researchers involves a polygenetic basis that results in a predisposition to develop the disease. An environmental trigger, such as a common virus (e.g., coxsackie), is considered the most likely cause for the biological cascade that takes place in the body that leads to the destruction of the Beta cells and the resulting absence of endogenous insulin (Beaser, 2007a). Although genetics can predispose an individual to developing type 1, only 5-10% of individuals with type 1 have a first- or second-degree relative with type 1
(Rosenbloom & Silverstein, 2003). Thus, the development of type 1 does not follow traditional Mendelian genetics (Rosenbloom & Silverstein).

Further, out of all individuals with a high genetic risk of developing type 1 diabetes (i.e., human leukocyte antigen [HLA] conferred susceptibility), less than 10% manifest the disease which supports the hypothesis that a potential interaction involving an unidentified exogenous factor might trigger the development of type 1 (Knip, Veijola, Virtanen, Hyoty, Vaarala, & Akerblom, 2005). The interaction of more than one exogenous factor might also trigger the development of diabetes in susceptible individuals. One such potential interaction might include a virus and perhaps a dietary agent containing foreign proteins such as cow’s milk (Knip et al.). Given the complexity of the issue, several major longitudinal epidemiological studies such as the Diabetes Autoimmunity Study in the Young (DAISY; Lamb et al., 2009) are being conducted to identify possible exogenous triggers.

**Treatment of type 1.** Intensive insulin therapy and glycemic control are the current recommended standard of care for the treatment of adults and youth with type 1 diabetes (ADA, 2012b; Silverstein et al., 2005). The recommendations are based on the results of the landmark study, the Diabetes Control and Complications Trial (DCCT; 1993). The DCCT was a longitudinal, randomized control trial (RCT) with an average follow-up of 6.5 years that examined the impact of intensive treatment versus conventional treatment in a population of 1,441 individuals with type 1 who were ages 13-39 years at start of the trial. Results from the DCCT revealed that participants from the intensive treatment method substantially reduced the risk of diabetes complications including eye, nerve, and kidney diseases by 76%, 60%, and 50%, respectively (CDC,
Thus, the intensive treatment methods used in the DCCT have become the basis for the standards of care in treating diabetes (ADA, 2012b). For the purposes of this study, treatment recommendations for only youth with diabetes will be examined.

Based upon the recommendations of the ADA (2012b; Silverstein et al., 2005), intensive insulin therapy and glycemic control is the standard of care for youth with type 1 diabetes. Upon initial diagnosis core members of a youth’s diabetic care team should consist of an endocrinologist, diabetes nurse educator, dietitian, and mental health professional. Additional referrals and screenings may also involve specialists such as an ophthalmologist or behavioral specialist. Treatment also includes an exercise plan and adherence to a diet. Individualized treatment plans are developed by a medical team and tailored to the characteristics and needs of the individual (e.g., age, weight, pubertal status) and family (Silverstein et al., 2005). In summary, the components of the intensive treatment method include: (a) intensive insulin therapy, also termed basal/bolus insulin therapy; (b) intensive blood glucose monitoring; (c) insulin doses adjusted based on exercise and food intake; (d) adherence to exercise and diet plan developed through medical team; and (e) regularly scheduled visits with diabetic care team including a physician, diabetes nurse educator, and any other appropriate specialist (e.g., dietitian, behavioral therapist, ophthalmologist; CDC, 2008; Silverstein et al. 2005).

**Intensive insulin therapy.** Intensive insulin therapy, also referred to as basal/bolus insulin therapy, requires four or more multiple daily injections (MDI) of exogenous insulin or insulin pump therapy for optimal care (as compared to the previous conventional treatment of two injections per day; Silverstein et al., 2005). In basal/bolus insulin therapy, the basal insulin or long-acting insulin maintains glycemic levels over the
course of 24 hours, while the bolus insulin or rapid- or short-acting insulin is used to compensate for the consumption of carbohydrates or elevated blood glucose levels.

Depending on an individual’s specific treatment regimen and the number of meals or snacks per day, MDI can range upwards of 6-7 (Silverstein et al.). MDI allows more flexibility in the lives of youth with type 1 allowing more adjustments to be made in insulin dosages based on an insulin-to-carbohydrate ratio (i.e., insulin delivered proportional to carbohydrates consumed) and amount of physical activity. In contrast, the former conventional treatment consisted of fewer injections, but an individual was on a fixed insulin regimen and would have to eat at specific times and specified quantities of food in accordance with the onset, peak, and duration of insulin action.

Highly restrictive diet plans were previously part of standard diabetes treatment; however, the ADA currently discourages making a youth eat when he or she is not hungry or depriving a youth of food as a method of controlling blood glucose ranges (Silverstein et al., 2005). Alternatively, individuals following a basal/bolus insulin plan do not have to eat a prescribed amount of carbohydrates during a meal or snack. Instead, rapid- or short-acting insulin can be administered proportional to the carbohydrates that were consumed (NDEP, 2010). It should be noted that while youth are not deprived of food in the current treatment paradigm, individualized meal plans are developed through medical nutrition training to develop healthy eating habits to ensure caloric and nutritional requirements for typical growth and development (Silverstein et al.).

The development of more advanced insulin analogues (e.g., rapid- or short-acting, intermediate-acting, long-acting) and delivery devices, such as an insulin pen or pump, have impacted treatment regimens (NDEP, 2010; Silverstein et al., 2005). The traditional
method of insulin injection is through a syringe. Two advancements in delivery device technology include the insulin pen and pump (Silverstein et al.). An insulin pen is a device that holds a cartridge of insulin and has a dial for selecting the prescribed dose of insulin to be administered (NDEP, 2010). The procedure for using an insulin pen includes fixing a single-use needle to the tip of the cartridge, using the dial to set the appropriate dose of insulin, and then injecting the insulin.

The insulin pump, also termed Continuous Subcutaneous Insulin Infusion (CSII), is a computerized device that is programmed to provide small increments of rapid- or short-acting insulin as the basal rate (NDEP, 2010). Depending on the youths’ level of maturity, either an adult (e.g., caregiver, school nurse) or the youth themselves can program additional bolus doses in response to carbohydrates consumed or high blood glucose levels. Notably, the current insulin pump technology available still requires the user to test his or her blood glucose frequently throughout the day and to make adjustments (e.g., bolus/basal) accordingly.

One type of insulin pump currently available is the size and shape of a pager (NDEP, 2010). A youth may wear this style of pump on his or her waistband, belt, or inside a pocket. The insulin is stored inside of the pump in a reservoir. The reservoir is attached to an infusion set comprised of a thin flexible plastic tube (of varying lengths) and a small needle or soft plastic cannula that remains under the youth’s skin. The infusion set is disposable and is changed every 2 or 3 days or in the instances of a failure in insulin delivery. Insulin pump site changes are conducted typically by the family in the home (NDEP). Youth using pager-type pumps can disconnect from the insulin pump temporarily for certain activities (e.g., sports activities). If youth keep the insulin pump
on during exercise, the youth or an adult may set a reduced basal rate or suspend the
delivery of insulin temporarily.

**Intensive glycemic control.** Blood glucose monitoring is essential for optimal
glycemic control. Four or more tests per day are recommended for youth with type 1
diabetes (Silverstein et al., 2005). Fluctuations in blood glucose levels are monitored
through the use of a small, portable blood glucose meter (NDEP, 2010). A lancet device
is used to puncture the skin of a finger or an alternative testing site (e.g., forearm) in
order to procure a drop of blood. The drop of blood is then transferred to a disposable
plastic strip that is inserted into the blood glucose meter. Within seconds, a number will
be displayed on the screen of the meter indicating the blood glucose level. It should be
noted that measurements taken from capillary blood procured from finger sticks may be
more accurate than blood from alternate-sites (Silverstein et al., 2005).

Further, newer technologies such as continuous glucose monitors (CGM) are used
to examine glucose fluctuations throughout the day (Silverstein et al., 2005). CGM
measure glucose levels in interstitial fluid through a sensor inserted underneath the skin
(NDEP, 2010). CGM is employed for identifying glucose level trends and is used in
combination with a blood glucose meter (NDEP). Improvements to CGM devices are
currently in development (Silverstein et al.). Notably, while CGM devices are in
development, Hood, Peterson, Rohan, and Drotar (2009) suggested that benefits of CGM
have not yet been evidenced for youth due to the likelihood that real-time data provided
by the CGM might be “too complex, demanding, or overwhelming for most youth with
type 1 diabetes and their families” p. e1177. Additionally, Hood et al. stated that the
benefits of CGM might not be apparent in the research literature because youth and
families that have participated in CGM studies are typically already doing well managing the youth’s diabetes care.

Target ranges for blood glucose levels depend upon the age of the youth and time of day (Silverstein et al., 2005). Differing target ranges are suggested for youth to mitigate the high risk of hypoglycemia, or blood glucose levels lower than the target range (≤70-80mg/dL; NDEP, 2010). Accordingly, pre-meal (i.e., preprandial) blood glucose target ranges vary for toddlers and preschoolers (<6 years; 100-180 mg/dL), school age children (6-12 years; 90-180 mg/dL), and adolescents and young adults (13-19 years; 90-130 mg/dL; Silverstein et al.). Bedtime and overnight ranges also vary across toddlers and preschoolers (110-200 mg/dL), school age children (100-180 mg/dL), and adolescents and young adults (90-150 mg/dL; Silverstein et al.).

**Metric for control.** One of the important consequences of the DCCT (1993) was in establishing a metric for metabolic control: hemoglobin A1C (HbA1c; Beaser, 2007c; DCCT, 1999, 1987). HbA1c, also referred to as A1C, is used to determine levels of average glucose concentration in the blood stream over a period of 2-3 months (ADA, 2012; Beaser; CDC, 2008). The ADA recommends that individuals with type 1 diabetes have HbA1c levels checked four times a year. HbA1c levels less than 7% are recommended for nonpregnant adults while levels for children vary based upon age (toddlers and preschoolers [0-6], HbA1c <8.5%; school age [6-12], HbA1c <8%; adolescents and young adults [13-19], HbA1c <7.5%; ADA, 2012). These recommendations were based on the relation between HbA1c level and the development of diabetes-related complications. Specifically, lower HbA1c levels resulted in a lower risk of developing complications.
Acute symptoms of hyper- or hypoglycemia. Hyperglycemia (blood glucose levels above the target range) and hypoglycemia (blood glucose levels below the target range) both result in acute physiological symptoms (NDEP, 2010). Acute symptoms of hyperglycemia include thirst, frequent urination, change in appetite, nausea, blurry vision, and fatigue (NDEP). Sustained high blood glucose levels lead to an increased risk of long-term complications such as heart disease, stroke, blindness, kidney failure, nerve disease, gum disease, and amputations (NDEP). Hyperglycemia can be caused by insufficient insulin present in the body, carbohydrate consumption not accounted for by the most recent insulin dosage, decreased physical activity, illness, infection, injury, and intense physical or emotional stress (NDEP).

Acute hyperglycemia does not typically lead to a medical emergency; however, urine should be tested for ketones. Ketones are chemicals the body produces during the break down of fat for energy in the absence of insulin (NDEP, 2010). Prolonged hyperglycemia will result in DKA that can lead to sleepiness, lethargy, and depressed level of consciousness. The buildup of ketones in the blood with ensuing metabolic acidosis can become life threatening (Silverstein et al., 2005). Acute symptoms of DKA include vomiting, sleepiness, fruity smelling breath, and breathing difficulty (NDEP). If the youth is not treated or hospitalized, coma or death may result (NDEP). DKA most commonly occurs at initial diagnosis of type 1 or insulin omission. Silverstein et al. (2005) reported that psychological problems (e.g., eating disorders, depression, anxiety) and lack of financial resources are the most common causes of DKA resulting from insulin omission.
While acute hyperglycemia does not usually result in a medical emergency, hypoglycemia is the most immediate risk to a youth and can lead to unconsciousness, coma, or even death if not treated immediately. Hypoglycemia (≤70-80 mg/dL; NDEP, 2010) usually occurs due to administering more insulin than what is required, skipping or postponing meals or snacks, not consuming enough carbohydrates at meal or snack times, or engaging in additional, strenuous, or unscheduled physical activity (NDEP). Acute symptoms of mild to moderate hypoglycemia include shakiness, sweating, hunger, paleness, headache, blurry vision, sleepiness, lethargy, weakness, dizziness, confusion, disorientation, lack of coordination, irritability, nervousness, changed personality, changed behavior (e.g., argumentative, combative), and inability to concentrate. Mild hypoglycemia can be treated with 15 grams of quickly absorbed carbohydrate (e.g., 3 or 4 glucose tabs, or 4 ounces of fruit juice; NDEP, Silverstein et al., 2005). Moderate hypoglycemia can typically be treated with 20-30 grams of a quickly absorbed carbohydrate to raise blood glucose levels (>80mg/dL; Silverstein et al.).

Manifestations of symptoms related to diabetes in the classroom, such as symptoms caused by hypoglycemia, are sometimes misidentified as problem behaviors unrelated to the youth’s chronic health condition (NDEP; Wodrich, 2005). In addition, persistent effects of hypoglycemia can cause transitive cognitive impairment on choice reaction-time tasks, even after blood glucose levels return to the normal range (Evans, Pernet, Lomas, Jones, & Amiel, 2000; Ryan et al, 1990).

Acute symptoms for severe hypoglycemia include the inability to eat or drink, unconsciousness, unresponsiveness, and seizure activity or convulsions (NDEP; 2010). The treatment for severe hypoglycemia is the administration of an injection of glucagon,
a hormone that triggers the release of glycogen stored in the liver that subsequently raises blood glucose levels. Administration of glucagon may result in nausea and vomiting once consciousness is regained (Silverstein et al., 2005). If this life-saving treatment is not delivered immediately, unconsciousness, coma, or even death can result. Thus, the fear of hypoglycemia can be a barrier to target levels of glycemic control (Silverstein et al.).

While acute cognitive impairment due to hypoglycemia is readily apparent (Silverstein et al.) and well established in the literature (e.g., Gonder-Frederick et al., 2009), the development of long-term cognitive deficits associated with episodes of severe hypoglycemia is disputed. For instance Northam et al. (2001) concluded that neuropsychological deficits were most likely caused by severe episodes of hypoglycemia. In contrast, The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study Research Group (DCCT/EDIC; 2007) reported that recurrent episodes of severe hypoglycemia were not related to declines in long-term cognitive functioning. Cognitive impairment related to type 1 diabetes will be discussed in depth later in this chapter.

Although exercise can lead to hypoglycemia, the ADA (Silverstein et al., 2005) recommends that youth engage in a minimum of 30-60 minutes of moderate physical activity daily to control weight and maintain physical fitness and overall health. Further, blood glucose levels should be monitored before physical activity. If blood glucose levels are below the target range, 15 grams of carbohydrates should be consumed with an additional 15 grams consumed for strenuous physical activity lasting longer than 30 minutes. Blood glucose tests should also occur following exercise, or once an hour for
prolonged strenuous exercise. Recommendations also favor decreasing insulin dosages for planned exercise rather than increasing caloric intake as part of a weight management program for youth (Silverstein et al.).

In summary, both hyper- and hypoglycemia lead to symptoms that will likely impair behavior and cognition (NDEP, 2010). The authors of the DCCT (1993) acknowledged that individuals following an intensive therapy regimen were two to three times more likely to experience hypoglycemia; however, the benefits of treatment outweighed the deleterious costs to health due to high blood glucose levels. Thus, while the intensive therapy regimen leads to better health outcomes for individuals with type 1 diabetes, the adults providing care to the youth (e.g., family unit, school nurse), and the youth themselves, must take on a great deal of responsibility including training to complete this complex regimen (NDEP, 2010; Silverstein et al., 2005).

**Diabetes education.** Intensive training and diligence are required to implement the recommended standards of care for type 1 diabetes management (ADA, 2012b; Silverstein et al., 2005). Upon initial diagnosis, a diabetes team comprised of certified professionals (e.g., pediatric endocrinologist, diabetes nurse educator, dietitian, mental health professional) able to provide current “pediatric-specific education and support” should evaluate the youth (Silverstein et al., p. 187). Critical skills essential for management, also referred to as “survival skills”, should be provided immediately after diagnosis (Silverstein et al.). Continuing education should also be provided to the youth and primary caregiver(s). Positive outcomes such as fewer hospitalizations and a reduction in medical costs are associated with patient and family education, intense diabetes case management, and telephone availability (as cited in Silverstein et al.).
The education program should be tailored to the needs of the youth, primary caregiver(s), and family (Silverstein et al., 2005). Accordingly, the training and information provided should be appropriate for the age and developmental level of the youth. For example, instruction and training in management skills would be directed toward the primary caregiver(s) of a toddler. For an adolescent, instruction and training would be directed toward the adolescent while also including the primary caregiver(s). In general, youth should not be totally independent providers of her or his diabetes management care (Silverstein et al.); however, responsibilities should be gradually transferred with the goal of achieving greater independence. For example, adolescents will likely be able to implement self-monitoring of blood glucose (SMBG) and self-administration of insulin; although, he or she will likely still need assistance in making decisions regarding the dosage of insulin to be administered (Silverstein et al.). The ADA (Silverstein et al.) provides guidelines for management priorities based on corresponding developmental stages that will not be detailed in this paper.

Palmer et al. (2009) reported that level of parental responsibility declined with adolescent age, and parental perceptions of both pubertal status and adolescent efficacy. Notably, poorer glycemic control occurred when responsibilities were transferred to an adolescent based upon age rather than parental ratings of adolescents’ efficacy. Thus, age alone may not be the best indicator of when to transfer responsibility for diabetes management to an adolescent. Consequently, parental perceptions of adolescents’ level of efficacy show promise as a factor to consider when deciding how much responsibility related to diabetes management to transfer to an adolescent.

**Type 2 Diabetes**
Physiology of type 2. Type 2 diabetes, formerly referred to as non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes, results from two factors: (a) insulin resistance and (b) insufficient levels of insulin being produced (ADA, 2000; Beaser, 2007a; Rosenbloom & Silverstein, 2003). Insulin resistance occurs in the liver, muscle, and adipose (i.e., fat) tissues in the body (Beaser). An individual experiencing insulin resistance might even have levels of insulin circulating in the body that far exceed levels found in healthy individuals; however, if the amount of insulin is not meeting the demands of the body due to insulin resistance, the amount is still considered to be insufficient (Beaser). Eventually, the Beta cells in the pancreas are no longer able to produce insulin to meet the demands of the body, resulting in Beta cell failure from hypersecretion (ADA, 2000; CDC, 2011).

Etiology of type 2. Risk factors associated with developing type 2 diabetes include obesity, physical inactivity, family history of diabetes, and ethnicity (CDC, 2011). Rates of type 2 are increasing among children and adolescents in the US (ADA, 2000). The increasing rate of type 2 in the US is theorized to be related to the increasing rate of obesity and the declining physical activity levels in youth (ADA). Further, 85% of youth diagnosed with type 2 are either overweight or obese (ADA). Family history also plays a role in the development of type 2. Seventy-four to one hundred percent of individuals diagnosed with type 2 have a first- or second-degree relative who also has type 2 (ADA). Finally, as mentioned previously, AI, API, Hispanic, and NHB youth (ages 10-19) are at higher risk for developing type 2 (CDC, 2011).

Prevention and treatment of type 2. Unlike type 1, the development of type 2 diabetes can be delayed or prevented entirely. The Diabetes Prevention Program (DPP)
was a RCT where a lifestyle intervention and a pharmacological intervention both separately reduced the development of metabolic syndrome, a precursor to type 2, in a group of individuals at high risk for developing type 2 over the course of 3 years (Orchard et al., 2005). The lifestyle intervention included weight loss of at least 7% clinical body weight through a healthy diet and 150 minutes of physical activity of moderate intensity (e.g., walking) per week. The rate of metabolic syndrome was reduced by 41% ($p < .001$) for individuals involved in the lifestyle intervention compared to the control group. Additionally, of those individuals identified as having metabolic syndrome at the start of the study, 38% of those involved in the lifestyle intervention no longer had the syndrome at the end of 3 years. In regard to the pharmacological intervention (i.e., metformin), there was a 17% ($p = .03$) reduction in the rate of metabolic syndrome compared to placebo. Metabolic syndrome was defined as three or more the following conditions: waist circumference greater than 102 cm for men, 88 cm for women; serum triglyceride of at least $\geq$ 150 mg/dL; high-density lipoprotein (HDL) cholesterol level $< 40$ mg/dL in men, $< 50$ mg/dL in women; blood pressure of $\geq 130/85$ mm/Hg; and, fasting plasma glucose level of 110 mg/dL (Orchard et al.). Thus, many participants were able to prevent or delay the development of type 2 diabetes.

Upon diagnosis with type 2, the treatment regimen includes medical nutrition therapy (i.e., diet) and exercise (ADA, 2000). Depending on the severity of the condition, a glucose lowering oral medication might also be prescribed. Deterioration of glycemic control, even with proper adherence to healthy lifestyle changes, may result in the necessity of one or more oral medications and eventually the addition of exogenous insulin administrations (ADA). In contrast to individuals with type 1, hypoglycemia is
not common for individuals with type 2 that are treated with insulin (Rosenbloom & Silverstein, 2003). If youth present with comorbid conditions such as hypertension and hyperlipidemia (i.e., high cholesterol), medications might also be prescribed in an effort to prevent future complications (ADA).

**Role of Technology in Diabetes Care**

Before the results of the DCCT (1993), fatalistic attitudes were common in the delivery of healthcare services in the US (Beaser, 2007b). Indeed, prior to the discovery of insulin in 1921, type 1 diabetes was unequivocally a death sentence (Beaser, 2007d). Without exogenous insulin, there were no means of extending life for individuals with type 1 beyond a few months after diagnosis (Beaser).

Globally, type 1 diabetes is still a death sentence in some regions. Based on 2008 data, the lack of access to insulin was the leading cause of death among individuals with type 1 (Daneman, 2009). For example, in some poorer countries, the costs of expensive testing equipment and exogenous insulin for a single child might result in a lack of other resources for the whole family. Thus, some parents must choose between starvation for all children or the death of one. To put the dire reality of this circumstance in perspective, in rural Mozambique the life expectancy for a youth with type 1 is approximately 7 months (Daneman).

While fatalistic attitudes toward diabetes might seem foreign in the US at present, Beaser (2007d) stated that before the results of the DCCT (1993), some medical professionals were “polarized” into camps that championed or condemned intensive treatment modalities. Some physicians believed that complications from diabetes were an inevitability unrelated to the type of treatment prescribed and some professionals held
fatalistic attitudes concerning the health outcomes for individuals living with diabetes even in to the early 1980’s. Beaser (2007b; 2007d) stated that some healthcare professionals would recommend less aggressive forms of treatment, employing the misguided rationale that patients should enjoy the remaining good years of their lives unburdened by aggressive therapies. Thus, the magnitude of the DCCT is that it provided incontrovertible evidence of the health benefits related to intensive diabetes control.

Not all physicians subscribed to a fatalistic paradigm before the DCCT. Dr. Elliott P. Joslin championed aggressive therapies to improve metabolic control and patient responsibility for individuals with diabetes in the early 1900’s (Beaser, 2007a). After exogenous insulin first became available, the Joslin Clinic created medals to commemorate the transition from a “rare fatal disease to a condition in which prolonged survival was possible…(and) the prevailing uncertainty as to the future of children whose lives depended on insulin” (Gale, 2002, p. 3354). On the medal, the image depicted “a small boy and his dog in an open boat with the sun rising beside them, and is entitled ‘explorers of uncharted seas’” (Gale, p. 3354).

Advancements in technology. The technology used to determine HbA1c levels and conduct Daily Blood Glucose Monitoring (DBGM) were not available until the early 1980s (Beaser, 2007c). Before that time, urine testing was the standard of care to assess glucose levels in the body. In fact, with the development of blood glucose meters, the protocols in the DCCT were changed from assessing glucose levels from urine samples to blood samples to accommodate the new technology (DCCT, 1993). Additionally, only pork or bovine insulin was available before the production of human analog insulins in the early 1980s (Beaser, 2007d). Thus, technological advancements have substantially
changed the assessment of metabolic control and treatment modalities. Through extension, the ability to analyze complex relations amongst variables over time, such as statistical programs employing structural equation modeling, is also another form of technology that has shown promise as a tool in studying diabetes management (e.g., Rausch et al, 2012).

**Impact on Education**

**Impact of type 1 diabetes on cognition.** Previous studies have indicated that youth with type 1 typically score within the average range on cognitive assessments (Holmes, O’Brien, & Greer, 1995; Kaufman, Epport, Engilman, & Halvorson, 1999); however, other research has also evidenced that youth with type 1 perform lower than healthy controls on assessments of intelligence, attention, processing speed, long-term memory, and executive skills putting youth with type 1 at greater risk for academic problems (Northam et al. 2001, Holmes et al.). Previous researchers concluded that early onset of type 1 was associated with lower Performance IQ (PIQ) scores, while late onset of type 1 was associated with lower Verbal IQ (VIQ) scores (Desrocher & Rovet, 2004; Holmes et al.). Differential PIQ and VIQ scores, ranging up to 10 points, were considered to be clinically meaningful as they might represent a unique pattern of impairment that could lead to learning problems (Holmes et al.). Further, Desrocher and Rovet proposed a theoretical model based on the findings of previous studies wherein early age of onset increases the risk of motor and visuospatial impairments, both acute (severe) and chronic (mild) hypoglycemia increases the risk of attention and memory deficits, hyperglycemia increases the risk of impairment in the areas of verbal and executive functioning, and puberty increases the risk of impairment in executive function.
In contrast, Northam et al. reported that early onset of type 1 was associated with lower scores in the areas of attention, processing speed, and executive skills, whereas severe episodes of hypoglycemia increased the risk of lower verbal and full-scale IQ scores. Moreover, Kent, Chen, Kumar, and Holmes (2010) reported no statistically significant effects of disease duration, age of disease onset, or severe hypoglycemia associated with performance on visual or verbal memory assessments. Only poor metabolic control (i.e., hyperglycemia) resulted in a significant impairment in visual memory. Thus, notable differential cognitive weaknesses associated with differing factors associated with type 1 are present in the literature (Desrocher & Rovet; Holmes et al.) along with discordance related to the magnitude of corresponding impairment.

Alternatively, McCarthy, Lindgren, Mengeling, Tsalikian, and Engvall (2002) suggested that fluctuations in blood glucose levels might exacerbate preexisting cognitive deficits that might explain why verbal impairments are reported in some studies and non-verbal or visuospatial and motor impairments are reported in others. As such, previous studies examining cognitive impairment in youth with type 1 have resulted in (a) a discordance in the literature concerning the magnitude of cognitive impairment related to type 1, (b) difficulty interpreting the impact of type 1 on cognition across studies due to the examination of different constructs related to cognition, and (c) differing conclusions concerning the level of risk of cognitive impairment presented by factors related to a youth’s medical history such as the age of onset of type 1 and metabolic control, as well as a history of DKA or severe episodes of hypoglycemia (Desrocher & Rovet; Gaudieri, Chen, Greer, & Holmes, 2008; Naguib, Kulinskaya, Lomax, & Garralda, 2009).
Desrocher and Rovet (2004) posited that some potential problems in comparing results between studies include differences in sample characteristics and study designs (e.g., cross-sectional rather than prospective). Additionally, Desrocher and Rovet suspected that researchers might tend to select assessments that are accessible rather than based on theoretical constructs, therefore, making comparisons between studies difficult. Further, differing magnitudes of cognitive impairment across studies might also be related to advancements in treatment modalities (e.g., DCCT) making examination of recent research paramount (Holmes et al., 1995; Naguib et al., 2009). Two relatively recent meta-analyses (Gaudieri et al., 2008; Naguib et al., 2009) have been useful in examining the impact of type 1 diabetes on cognition and risk factors related to medical history.

**Cognitive scores of youth with type 1 diabetes compared to controls.** Naguib et al. (2009) examined 24 studies published from 1980 to 2005. The aims of the meta-analysis were (a) to determine the impact of diabetes-related cognitive impairments in youth (≤ 19 years of age) with type 1 compared to controls, and (b) to examine the impact of disease-related variables such as age of onset and severe hypoglycemic episodes. Only studies with a case control design and standardized neuropsychological assessments that measured one of seven cognitive domains such as intelligence, visuospatial ability, language and education, memory and learning, psychomotor activity, attention, and executive function were included. Across the studies used in the Naguib et al. meta-analysis, participants included 894 youth with type 1 and 758 controls.

When scores were compared between youth with type 1 diabetes and controls, the Naguib et al. (2009) meta-analysis revealed statistically significant and small effect sizes
(Cohen’s $d$) indicating lower scores for youth with type 1 in the cognitive domains of visuospatial ability ($d = -.29$, $p = .01$), language-reading ($d = -.23$, $p = .03$), language-written ($d = -.28$, $p \leq .000$), motor speed ($d = -.26$, $p = .03$), and sustained attention ($d = -.22$, $p = .02$). Absolute values of effect sizes are typically interpreted using Cohen’s $d$ criteria as small ($d \geq 0.2$), moderate ($d \geq 0.5$) and large ($d \geq 0.8$; Cohen, 1988).

Statistically significant effect sizes also indicated lower scores for youth with type 1 compared to controls in the areas of overall IQ score ($d = -.14$, $p = .03$), verbal IQ ($d = -.15$, $p = .05$), and performance IQ ($d = -.18$, $p = .04$; Naguib et al.). Naguib et al. concluded that the results indicated that youth with type 1 demonstrated mild cognitive impairments and slightly diminished overall intellectual functioning. Naguib et al. stated that although the small effect sizes related to differences in cognitive scores between youth with type 1 and controls might not be clinically significant, youth with type 1 might be an at-risk group of students compared to peers.

Gaudieri et al. (2008) examined 19 studies with publication dates ranging from 1985 to 2008. The objectives of this meta-analysis were (a) to assess the impact of cognitive impairments related to diabetes in youth with type 1 diabetes compared to controls and (b) to examine factors related to cognitive impairment including age of onset and episodes of severe hypoglycemia. Studies were included if they utilized a case control design, youth were diagnosed before the age of 18, and at least one of six broad cognitive domains were assessed including intelligence, learning and memory, psychomotor activity and speed of information processing, attention/executive function, academic achievement, and visual motor integration. Across the studies used in the
Gaudieri et al. meta-analysis, participants included 1,393 youth with type 1 and 751 controls.

In the Gaudieri et al. (2008) meta-analysis, youth with type 1 diabetes scored lower on cognitive assessments when compared to controls in the cognitive domains of psychomotor efficiency ($d = -0.10, p = .01$), motor speed ($d = -0.16, p = .00$), attention/executive function ($d = -0.10, p = .04$), academic achievement ($d = -0.13, p = .01$), and visual motor integration ($d = -0.18, p = .02$). Youth with type 1 diabetes also scored slightly lower compared to controls on assessments of overall cognition ($d = -0.13, p = .00$), crystallized intelligence ($d = -0.18, p = .00$), and fluid intelligence ($d = -0.15, p = .00$). No statistically significant differences in scores between youth with type 1 and controls were found in the areas of learning and memory. Gaudieri et al. determined that the small effect sizes related to the difference in overall cognitive scores translates as a total IQ score decrease of 1 to 2 points on average for youth with type 1 compared to controls. Thus, analogous to previous studies (e.g., Holmes et al.), Gaudieri et al. concluded that the overall cognitive scores for youth with type 1 are slightly lower, but typically age appropriate. Further, Gaudieri et al. deduced that a difference of 1 or 2 points in overall IQ score is unlikely to be clinically significant. Accordingly, Gaudieri et al. suggested that while their results revealed generally age-appropriate group scores, the variability in cognitive scores might include students with no impairment to substantial cognitive impairment.

In summary, both Gaudieri et al. (2008) and Naguib et al. (2009) concluded that overall cognitive scores for youth with type 1 are subtly lower than controls without type 1 diabetes. Gaudieri et al. reported an effect size ($d = -0.13, p = .00$) comparable to that of
Naguib et al. \((d = -0.14, p = 0.03)\) when examining overall cognitive score differences between youth with type 1 diabetes and controls. Gaudieri et al. concluded that this small effect can be quantified as a decrease in overall cognitive score of 1 or 2 points on average for youth with type 1 diabetes when compared to controls. Based on the small effect size related to differences in overall cognitive performance for youth with type 1 versus controls, Gaudieri et al. concluded that the results were not clinically significant. Likewise, Naguib et al. suggested the differences in overall cognitive scores between youth with type 1 and controls were unlikely to be clinically significant. Nonetheless, both research groups stated that youth with diabetes are still at-risk for cognitive impairments and the magnitude of impairment might vary among youth.

**Age of onset.** Gaudieri et al. reported that youth with early onset type 1 diabetes (before 7 years of age) earned lower scores on overall IQ \((d = -0.20, p = 0.00)\) when compared to youth with later onset type 1. Previous research suggested differential risk for cognitive impairment was dependent on age of disease onset whereby early age of onset of the disease was associated with lower PIQ scores, while late age of onset was associated with lower VIQ scores (Desrocher & Rovet, 2004; Holmes et al., 1995). Conversely, in the Gaudieri et al. meta-analysis youth with early onset of type 1 scored lower than youth with late onset in the cognitive domains of crystallized intelligence \((d = -0.15, p = 0.03)\), verbal learning \((d = -0.26, p = 0.00)\), verbal learning and memory \((d = -0.28, p = 0.00)\), visual learning and memory \((d = -0.25, p = 0.04)\), attention/executive function \((d = -0.27, p = 0.01)\), and academic achievement \((d = -0.19, p = 0.00)\). Nevertheless, all of the aforementioned effect sizes in the Gaudieri et al. meta-analysis were small.
Further, when compared to controls, youth with early onset type 1 diabetes evidenced lower overall cognitive scores ($d = -.29$) compared to youth with later onset of type 1 diabetes ($d = -.20$). Notably, when compared to controls, meaningful effect sizes were evidenced for youth with early onset type 1 demonstrating markedly lower scores in the cognitive domains of verbal ($d = -.49$) and visual ($d = -.44$) learning and memory, attention/executive function ($d = -.39$), and crystallized ($d = -.35$) and fluid ($d = -.28$) intelligence. Gaudieri et al. concluded that the aforementioned moderate effect sizes in domains of verbal and visual learning and memory abilities and attention/executive function might be clinically significant as they represent overall score decreases of 6 to 7 points on average. In summary, children with early onset type 1 appear to be differentially at risk for impairments in verbal and visual learning and memory abilities, and attention/executive function than children with late onset of the disease (Gaudieri et al.).

In summary, early age of onset appeared to be a substantive risk factor for cognitive impairments most apparent in the areas of verbal ($d = -.49$) and visual ($d = -.44$) learning and memory when compared to controls in the Gaudieri et al. meta-analysis. Overall cognition was differentially impacted for youth with early onset type 1 diabetes ($d = -.29$) than youth with late onset diabetes ($d = -.20$) when compared to controls (Gaudieri et al.). Interestingly, a decline in cognitive scores has been reported to occur in youth (3-14 years of age) as soon as two years after diagnosis with type 1 (Northam et al., 1999). In contrast, Naguib et al. (2009) did not report any statistically significant effects related to differences in cognitive scores between youth with early age of onset and late age of onset of the disease (Naguib et al., 2009). A recently published longitudinal study
revealed evidence to suggest cognitive impairments associated with early onset of the disease similar to those reported in Gaudieri et al. Lin, Northam, Rankins, Werther, and Cameron (2010) followed youth with type 1 for 12 years after disease onset found that youth with early onset of the disease demonstrated lower scores compared to youth with late onset of the disease in the cognitive domains of mental efficiency ($p < .05$), new learning ($p < .05$), divided attention ($p = .001$), and sustained attention ($p < .001$).

**History of severe episodes of hypoglycemia.** In the Gaudieri et al. meta-analysis, severe hypoglycemic episodes were defined in accordance with EDIC as a history of at least one seizure. The meta-analysis included 310 youth identified as having had at least one seizure and 590 youth were identified as never having had a seizure. Also included in the meta-analysis were two studies that defined a hypoglycemic episode as severe if youth required an administration of glucagon or the assistance of others in treating hypoglycemia. A negligible effect size was evidenced for differences in overall cognition scores ($d = -.06$) between youth with and without a history of severe hypoglycemic episodes. Unexpectedly, youth with a history of severe hypoglycemic episodes performed better than youth that have not experienced severe hypoglycemic episodes in the areas of visual memory ($d = .13$) and learning ($d = 0.12$). The authors speculated that the higher scores in visual memory and learning may have occurred due to the protective factors of better metabolic control, based on the rationale that tighter control increases the likelihood of severe hypoglycemic episodes (DCCT, 1993).

Overall, the effect of episodes of severe hypoglycemia on cognition was benign (Naguib et al., 2009; Gaudieri et al., 2008); however, some inconsistent performances in cognitive domains were noted. Naguib et al. did report a statistically significant, albeit
small, effect size for youth with type 1 diabetes with a history of episodes of severe hypoglycemia scoring lower than youth with type 1 without a history of severe hypoglycemic episodes on assessments of short-term verbal memory \((d = -.14, p = .04)\); however, no significant effect was evidenced for any impairment in overall cognition related to history of severe hypoglycemic episodes. Likewise, Gaudieri et al. reported a negligible effect size for differences in overall cognition scores \((d = -0.06)\) between youth with and without a history of severe hypoglycemic episodes. A protective factor related to better metabolic control may have been observed as youth with a history of severe hypoglycemic episodes performed better than youth that have not experienced severe hypoglycemic episodes in the areas of visual memory \((d = .13)\) and learning \((d = 0.12)\). Thus, the impact of severe hypoglycemic episodes on overall cognition appears negligible for youth with type 1 diabetes in two meta-analyses.

The discordance in the literature related to the impact of severe episodes of hypoglycemia on cognition might be related to varying sample characteristics and sample sizes across studies. In a nationally representative follow-up study to the DCCT (DCCT/EDIC, 2007), there was no evidence that high rates of recurrent severe hypoglycemia substantially impacted long-term cognitive functioning of the 453 participants \((N = 1144)\) that experienced one or more severe hypoglycemic events that lead to coma or seizure. In contrast, Kaufman et al. (1999) reported youth with type 1 diabetes from Los Angeles with a history of hypoglycemia related seizures \((n = 8)\) scored lower on the Woodcock-Johnson Short-Term Memory \((p < .03)\) and Memory for Words subtest \((p < .03)\) than youth without a history of hypoglycemia related seizures \((n = 10)\). Thus, results from a large and nationally representative sample as demonstrated in the
DCCT/EDIC study appear to support the conclusion that the overall effects of hypoglycemia on cognition are benign (Naguib et al., 2009; Gaudieri et al., 2008).

Notwithstanding, authors of both the DCCT/EDIC (2007) and Kaufman et al. (1999) recognized that seizures due to profound hypoglycemia can negatively impact the brain. Additionally, Ryan, Gurtunca, and Becker (2005) reported physiological changes to the brain including evidence of neuronal necrosis following profound and extended episodes of severe hypoglycemia. As such, the magnitude of said impact remains uncertain in the literature. Accordingly, Gaudieri et al. stated that it was unclear in the meta-analysis if (a) potentially negative cognitive impairments related to seizures are due to possible synergistic effects with early onset of the disease, or (b) whether children with poorer metabolic control are impacted more profoundly by severe hypoglycemic episodes in comparison to children with better metabolic control. Gaudieri et al. suggested that future studies might find that the interaction of disease variables (e.g., severe episode of hypoglycemia, poor control) might be predictive of cognitive impairment rather than subgroup membership (e.g., early onset).

A developing brain at risk. Ryan (2006) reported that it was previously believed by most researchers that the cumulative effect of episodes of severe hypoglycemia resulted in cognitive impairment for youth with type 1 diabetes. For example, Northam et al. (2001) followed 60 youth (6-17 years of age) with type 1 for 6 years after diagnosis and determined that neuropsychological deficits in verbal and overall IQ performance were most likely attributable to episodes of severe hypoglycemia even in the absence of seizures. In contrast to Northam et al.’s previous assertion, that same research group stated that chronic hyperglycemia might also impact the developing brain of a small child
during critical developmental periods and recommended more research exploring these effects. Interestingly, results from two meta-analyses (Gaudieri et al., 2008; Naguib et al., 2009) and more recent studies (e.g., Kent et al., 2010) indicated the impact of severe hypoglycemic episodes on cognitive functioning as benign. In regard to the deleterious effects of hyperglycemia, Kent, et al. reported that the results of growth curve modeling analysis revealed an inverse relation between metabolic control and visual memory, but no significant effects of severe hypoglycemia on visual or verbal memory scores of youth (9-17 years of age) with type 1 over a 3-year period. Ryan (2006) addressed the discordance in the literature regarding the magnitude of the impact of severe hypoglycemic events on cognition for youth that develop type 1 early in life. Ryan proposed a Diathesis Hypothesis that states that (a) youth with early onset diabetes are at substantial risk for neurocognitive dysfunction resulting from structural and functional changes in the central nervous system (CNS) caused by poor metabolic control (i.e., chronic hyperglycemia) during early brain development (first 5-6 years of life) and (b) a synergistic effect occurs between the vulnerability caused by the resulting changes to the CNS early in life and the occurrence of severe hypoglycemia at any age. Thus, youth with type 1 diabetes with abnormal brain development stemming from hyperglycemia will be more vulnerable and experience a greater degree of cognitive impairment resulting from severe episodes of hypoglycemia.

Ryan (2006) postulated that recent evidence suggested that neurocognitive dysfunction was unlikely to occur as a result of severe hypoglycemic episodes unless other concomitant factors are present. Microvascular complications associated with poor metabolic control and the toxic effects of hyperglycemia on the CNS have both been
associated with cognitive impairment (Biessels, Deary, & Ryan, 2008). Consequently, sequelae related to poor metabolic control affecting the CNS not only negatively impacts the developing brain of a young child but also puts young children at risk for greater cognitive complications following a severe hypoglycemic episode (Ryan). Similarly, Lin et al. (2010) reported that a synergistic effect between risk factors related to type 1 diabetes was evidenced on cognitive assessment scores. Specifically, youth with type 1 with a combination of 2 or 3 risk factors such as early onset diabetes, hypoglycemia, and hyperglycemia scored lower than healthy controls and youth with type 1 with only one or no risk factors on assessments of verbal abilities, working memory, and mental efficiency (Lin et al.). Thus, it appears that a synergetic effect of multiple risk factors may impact cognition (Ryan; Lin et al.).

Ryan (2006) proposed that while health care professionals may suggest higher blood glucose target ranges for young children to avoid the occurrence of severe hypoglycemic episodes, an individualized intensive treatment regimen with lower blood glucose target ranges might be more helpful in protecting the developing brain from insult related to hyperglycemia. Analogous to Ryan’s proposal, Kent et al. (2010) stated that the innocuous effects of severe hypoglycemic episodes on the trajectory of memory scores might indicate that although tighter metabolic control increases the risk of severe hypoglycemic episodes, this higher risk might be acceptable to prevent the deleterious effects of poorer metabolic control on memory.

**Poor metabolic control as a risk factor for cognitive impairment.** Although overall metabolic control was predominately poor across the studies used in the Naguib et al. (2009) meta-analysis, the relation between metabolic control and cognitive
impairment remained unclear. Similarly, Northam et al. (2001) reported no significant
relation between poor metabolic control and neuropsychological impairment.
Conversely, research studies have been published that suggest a relation exists between
metabolic control and cognitive impairment (e.g., Kaufman et al., 1999; Kent et al., 2010;
Lin et al., 2010). As stated previously, researchers have proposed that a young child’s
developing brain is susceptible to insult related to metabolic fluctuations (Biessels,
Deary, & Ryan, 2008; Ryan, 2006). Thus, chronic hyperglycemia is toxic to the brain
throughout life and may lead to structural abnormalities during development (Biessels et
al., 2008; Ryan, 2006).

Moreover, a follow-up study to the DCCT (DCCT/Epidemiology of Diabetes
Interventions and Complications Study Research Group [DCCT/EDIC], 2007) revealed
that poorer metabolic control was associated with moderate declines in the cognitive
domains of motor speed ($p = .001$) and psychomotor efficiency ($p < .001$). Additionally,
Kent et al. (2010) reported growth curve modeling analysis revealed an inverse relation
between metabolic control (i.e., HbAlc) and visual memory scores ($p < .01$) over the
course of 3 years for youth (9-17 years of age) with type 1 diabetes. In other words,
youth with poorer metabolic control (i.e., higher HbAlc levels) scored lower on visual
memory assessments over time. Likewise, Lin et al. (2010) reported that 12 years after
onset of type 1, youth with type 1 with poor metabolic control (i.e., chronic
hyperglycemia) scored significantly lower on a working memory assessment than
controls and yet while no statistically significant differences emerged between youth with
type 1 with good metabolic control and healthy controls concerning working memory.
Further, Parent, Wodrich, and Hasan (2009) reported a statistically significant and
meaningful relationship between teacher ratings of classroom attention for youth with type 1 and metabolic control ($r = .53, p < .001$). While discordance in the literature is present, previous research has provided evidence of an association between metabolic control and cognitive impairment.

**Short-term cognitive implications.** Short-term or transitory cognitive impairment has been reported in the literature (Gonder-Fredrick et al., 2009). Gonder-Fredrick et al. (2009) followed 61 youth with type 1 diabetes (6-11 years of age) over 4-6 weeks. Both completion time and scores on mental math and choice reaction time tasks, administered through personal digital assistant (PDA) devices, were correlated with glucose levels directly after testing. Transitory cognitive impairment impacting only completion time was evidenced during episodes of both hypoglycemia and hyperglycemia. Specifically, participants took significantly longer to complete mental math tasks during episodes of both hypoglycemia ($p = .0017$) and hyperglycemia ($p = .0001$) when compared to performance during euglycemia (i.e., within target range glucose levels). The completion of choice reaction time tasks was significantly slower during only hypoglycemia ($p = .01$). Gonder-Fredrick et al. quantified the observed transitory cognitive impairment as an approximate 20% speed reduction in completing mental math problems. The authors concluded that this decrease in mental efficiency was clinically meaningful. Further, examination of individual impairment scores revealed that during both hypoglycemia and hyperglycemia performance was reduced >1 standard deviation for >20% of the participants.

Notably, Ryan et al. (1990) reported that transitive cognitive impairment due to hypoglycemia in choice reaction-time tasks persisted for a period even after blood
glucose levels in youth (11-18 years of age) returned to the normal range. To quantify the length of transitory impairment associated with hypoglycemia, Evans et al. (2000) induced hypoglycemia in eight male volunteers (mean age 28.5 ± 3.6 years) without diabetes and discovered it took 20 minutes after blood glucose levels returned to normal before post-test scores were comparable to pre-test scores on Stroop word and color-word subtests. In comparison, 4-choice reaction time performance remained significantly impaired 20 minutes after blood glucose levels were returned to normal (Evans et al.). Thus, previous research has evidenced a clinically meaningful impact of transitory cognitive impairment on mental efficiency.

*Conclusions related to impact of type 1 diabetes on cognition.* Both long-term (Lin et al., 2010; Northam et al., 2001) and short-term cognitive impairments (Gonder-Frederick et al., 2009) were associated with chronic and acute metabolic insult related to type 1. Discordance is present in the literature related to differential cognitive weaknesses associated with varying factors of type 1 and the magnitude of the impact on cognition possibly due to methodological differences across studies (e.g., sample characteristics, research design, examination of different constructs of cognition), as well as, advancements in treatment modalities (Desrocher & Rovet, 2004; Gaudieri et al., 2008; Holmes et al., 1995; Naguib et al., 2009). A review of two meta-analyses (Gaudieri et al.; Naguib et al.) and an examination of more recent publications evidenced an increased risk for cognitive impairment associated with early age of onset (Gaudieri et al.; Lin et al.) and poor metabolic control (DCCT/EDIC, 2007; Kent et al., 2010; Lin et al.). Early age of onset was associated with greater risk for impairments in verbal and visual learning and memory abilities, attention, and executive functioning, when
compared to youth with late onset of the disease (Gaudieri et al.; Lin et al.). Poor metabolic control increased the risk of cognitive impairment in the areas of motor speed (DCCT/EDIC, 2007), psychomotor efficiency (DCCT/EDIC), visual memory (Kent et al.), working memory (Lin et al.), long-term retrieval (i.e., memory; Kaufman et al.), and attention (Kaufman et al.; Parent et al., 2009). Further, in contrast to previous studies (Northam et al.; Rovet & Ehrlich, 1999) the risk associated with severe episodes of hypoglycemia appeared benign (Gaudieri et al.; Naguib et al.); however, a recent study (Lin et al.) provided evidence of a synergistic effect (Ryan, 2006) of multiple type 1 diabetes risk factors including early onset diabetes, hypoglycemia, and hyperglycemia associated with poorer performance on assessments of verbal abilities, working memory, and mental efficiency. Overall, previous research has indicated that mean cognitive scores for youth with type 1 fall within the average range (Holmes et al., 1995; Kaufman et al., 1999) and differences in overall cognitive scores between youth with type 1 and controls are slight; however, the negative impact of even subtle decrements in cognition may impact acquisition of academic skills and achievement (Gaudieri et al.; Holmes et al.; Lin et al.).

**Impact of type 1 diabetes on achievement.** While mean cognitive scores appear age appropriate for youth with type 1, despite some differences, the variability in overall cognitive scores as well as across specific cognitive domains may range from negligible to clinically significant cognitive impairment (Gaudieri et al., 2008). Moreover, transient cognitive impairment associated with hour-to-hour blood glucose fluctuations (Gonder-Fredrick et al., 2009) might impact classroom performance and academic skill acquisition (Hannonen et al., 2012). Thus, previous research has indicated that youth diagnosed with
type 1 are at-risk for cognitive impairments, learning problems, and poorer academic performance (Dalenquest & Kallen, 2007; Fox, Chen, and Holmes, 2003; Gaudieri et al.; Hannonen, Komulainen, Eklund, Tolvanen, Riikonen, & Ahonen, 2010; Hannonen et al., 2012; Holmes, et al., 1995; Kent et al., 2010; Lin et al., 2010; McCarthy, Lindgren, Mengeling, Tsalikian, & Engvall, 2003; Naguib et al., 2009; Parent, Wodrich, & Hasan, 2009; Wodrich, Hasan, & Parent, 2011). Accordingly, youth with type 1 reportedly scored lower on achievement assessments ($d = -0.13$, $p = .01$; Gaudieri et al.) and performed worse academically (Dalenquest & Kallen) when compared to healthy peers without type 1. Nevertheless, mean achievement scores for youth with type 1 tend to fall within the average range (Holmes et al., Kaufman et al., McCarthy, Lindgren, Mengeling, Tsalikian, & Engvall, 2002). An age appropriate mean score, however, may not reveal the differential range of impairment across youth with type 1 (Gaudieri et al.; Northam et al., 1999). Thus, examination of medical factors associated with type 1 related to achievement appears warranted. Medical factors related to achievement in youth with type 1 to be explored in this section will include metabolic control, age of onset, and history of severe episodes of hypoglycemia.

**Poor metabolic control as a risk factor for poor achievement.** The majority of previous studies examining the impact of metabolic control on achievement have reported an inverse relation between metabolic control and achievement (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009); conversely, some studies have not found this relation (Fox et al., 2003; Kovacs et al., 1992). Kaufman et al. employed a cross-sectional design and assessed 55 children, 7.9 ± 1.6 years of age, diagnosed with type 1 diabetes before the age of 10 using the Woodcock-Johnson Tests
of Achievement (WJ-ACH). Kaufman et al. reported a negative correlation between poor metabolic control (i.e., higher HbAlc) and scores on achievement domains of the WJ-ACH including basic academic skills ($r = -.37$, $p < .006$), WJ Reading ($r = -.32$, $p < .019$), WJ Mathematics ($r = -.33$, $p < .014$), and WJ Written Language ($r = -.32$, $p < .017$). Kaufman et al. concluded that better glucose control might impact cognitive ability. Alternatively, Kaufman et al. also stated that better metabolic control might be related to the cognitive skills of the youth with type 1 or represent the cognitive skills of the parents managing the diabetes care for the youth.

McCarthy et al. (2002) examined achievement outcomes for youth with type 1 diabetes ($n = 244$), 8-18 years of age, using the Iowa Tests of Basic Skills (ITBS) for grades 3-8, Iowa Tests of Educational Development (ITED) for grades 9-12, and grade point averages (GPA). Both cross-sectional and hierarchical linear modeling (HLM) designs were employed to assess academic outcomes. The cross-sectional analysis examined current academic performance and indicated that youth with poorer metabolic control scored lower on achievement assessments when compared to youth with better metabolic control. It should be noted, however, that the methodology of this study analyzed youth with type 1 based on three levels of metabolic control (i.e., HbAlc < 8%, HbAlc 8-10%, HbAlc > 10%). Mean achievement scores of each of the three levels of metabolic control of youth with type 1 were compared to mean achievement scores of healthy sibling controls and matched classmate controls, but not across levels of metabolic control within the sample of youth with type 1. Notably, based on averaged performance scores of youth with type 1 that were generated when matched comparisons were examined with siblings, achievement scores for youth with type 1 were higher based
on better metabolic control across the areas of ITBS/ITED reading (HbAlc < 8% = 115.4, HbAlc > 10% = 107.4), mathematics (HbAlc < 8% = 121.8, HbAlc > 10% = 109.4), and core total (HbAlc < 8% = 119.5, HbAlc > 10% = 108.8). Unfortunately, the impact of better metabolic control was not examined through a statistical analysis; only comparisons between youth with type 1 and control groups were examined. McCarthy et al. did address this trend by suggesting that youth with higher cognitive ability might be better able to manage this disease, rather than HbAlc impacting achievement outcomes. When compared to siblings, no statistically significant difference was reported between mean GPA scores for youth with type 1 (3.1) compared to matched siblings (3.2). In regard to HLM, achievement growth curves for the ITBS/ITED scores did not reveal any differences between the performance of youth with type 1 and healthy sibling controls (n = 110) or healthy classmate controls (n = 209). A limitation of the study that should be discussed concerns matched comparisons decreasing the sample size across analyses. For example, sample size was decreased when ITBS/ITED scores for youth with type 1 and healthy sibling controls were matched for reading (86), mathematics (86), and core total (83).

In a follow-up study, McCarthy et al. (2003) examined the same sample of youth with type 1 diabetes (n = 244), 8-18 years of age, using t-tests, ANOVAs, and regression analyses to explore differences between differing levels of metabolic control. Subgroups were identified as good control (HbAlc < 8%), average control (HbAlc 8-10%), and poor control (HbAlc > 10%). Results indicated that on average, youth with type 1 in the poor metabolic control group scored lower on ITBS/ITED assessments of reading (105.7 vs. 112.1) and core total (106. vs. 113.1), and GPA (2.8 vs. 3.1) at a statistically significant
level ($p < .05$) compared to youth in the good control group. Further, compared to youth with average control, mean scores for youth with poor control were lower on the ITBS/ITED reading assessment (105.7 vs. 110.1) and GPA (2.8 vs. 3.1) at a statistically significant level ($p < .05$). In comparison to the results of the McCarthy et al. (2002) study where average GPA between youth with type 1 (3.1) and healthy sibling controls (3.2) revealed no statistical differences, further examination within the type 1 group revealed a statistically significant difference between the poor control group mean GPA (2.8) and the average (3.1) and good control (3.1) groups. Thus, this in-depth examination of GPA among youth with vary degrees of metabolic control (McCarthy et al., 2003) is an example of how using only mean ratings or scores for all youth with type 1 in a sample might (McCarthy et al., 2002) not reveal the magnitude of factors such as metabolic control that may negatively impact achievement.

Regression analyses were also employed in the McCarthy et al. (2003) study to examine the predictive power of health related variables on the achievement outcomes of core total on the ITBS/ITED and GPA. HbA1c was negatively correlated with ITBS/ITED core total score ($r = -.21, p < .05$). Model trimming was employed with ITBS/ITED core total score as the dependent variable. Independent variables included background, medical, and behavioral predictors. The final model ($R^2 = .38$) used to predict core total score revealed three statistically significant ($p < .01$) predictors including parental assessments of fatigue ($\beta = 9.55$) and learning ($\beta = -24.75$) from the Pediatric Behavior Scale (PBS-50d), and socioeconomic status (SES; $\beta = -.54$). In regard to GPA, bivariate correlations revealed statistically significant ($p < .05$) inverse relations with HbA1c ($r = -.21$), SES ($r = -.30$), school absences ($r = -.27$), and hospitalizations due
to hypoglycemia ($r = -.15$), and all eight PBS-50d scales (-.27 to -.61). Model trimming was also employed with GPA as the dependent variable regressed upon background, medical, and behavioral predictors. Only SES ($\beta = -.01$) and a parental assessment of learning from the PBS-50d ($\beta = -.70$) had statistically significant regression coefficients ($R^2 = .44$, $p < .001$). Thus, while correlations were negative and significant between HbAlc and GPA or ITBS/ITED core total score, SES and behavioral variables (e.g., PBS-50d) were the only significant coefficients in the regression models predicting GPA and ITBS/ITED core total score.

A second regression analysis conducted by McCarthy et al. (2003) included a quadratic term to test a proposed inverse “U-shaped” relation between frequent hypo- and hyperglycemia and lower achievement scores (p. 116). In other words, youth with type 1 diabetes with frequent fluctuations to extreme ends of blood glucose ranges (e.g., good control, poor control) were expected to perform worse academically than youth with average control. The proposed inverse U-shaped model was not supported. As previously discussed, youth with type 1 with poor metabolic control (HbAlc > 10%) demonstrated lower mean GPA and scores on standardized academic assessments (reading and core total) than youth with good control (HbAlc < 8%; $p < .05$) and lower mean GPA and scores on a standardized reading assessment than youth with average control (HbAlc = 8-10%, $p < .05$). Thus, it appears that youth with poor metabolic control demonstrate more impairment related to achievement than youth with better metabolic control. Additionally, there was no evidence to support a negative effect on academics related to tight control or, alternatively stated, more time spent in a lower average glucose range (HbAlc < 8%).
A limitation of the McCarthy et al. (2003) regression was that it was cross-sectional and not longitudinal. Thus, individual achievement performance over time related to metabolic fluctuation was not examined. Additionally, the HLM used to examine growth curves used matched pairs, which limited the sample size in some years, and contrasted youth with type 1 with healthy controls rather than examining changes in academic performance occurring over time for youth with type 1 related to changes in metabolic control.

More recent studies have also supported an inverse relation between metabolic control and achievement. In a study by Parent et al. (2009), teachers were asked to rate 95 youth with type 1 diabetes ($M_{age} = 11.8; SD = 3.0$) and 95 healthy sibling controls in the areas of academic skills and classroom attention. Academic skills were rated on a scale of 1 to 5 with higher scores representing better academic skills in the areas of reading, mathematics, and writing. An inverse relation between HbA1c values and academic performance was evidenced through moderate and statistically significant negative correlation coefficients. Youth with type 1 with higher HbA1c values (i.e., poorer metabolic control) earned lower academic performance ratings in the areas of reading ($r = -.37; R^2 = .139; p < .001$), mathematics ($r = -.34; R^2 = .116; p = .002$), and writing ($r = -.34; R^2 = .119; p = .001$). Additionally, ratings of classroom attention were also completed where higher scores represented greater inattention. A large effect was reported as youth with type 1 demonstrated more attention difficulties with worsening metabolic control ($r = .53; R^2 = .279; p < .001$). Parent et al. concluded that better metabolic control is essential for academic skill development and optimal classroom
attention levels. Further, the authors recommended that health care professionals and educators monitor academic skill areas and promote metabolic control.

Hannonen et al. (2012) examined academic skills in the areas of reading, mathematics, and spelling accuracy in 63 youth with type 1 \( (M_{age} = 9\text{ years, 11 months}; SD = 4\text{ months}) \) diagnosed before the age of 5 (i.e., early onset) and 92 healthy controls in Finland. All assessments were conducted in Finish. Covariates in this cross-sectional, multivariate General Linear Model (GLM) analyses included participant’s sex, parents’ education level, familial risk for dyslexia, and the participant’s IQ. Participant IQ was the only statistically significant predictor of reading accuracy, spelling accuracy, and mathematics skills. Interestingly, HbAlc level during the first year following diagnosis (approximately 5 years of age) was a meaningful and statistically significant predictor of current (approximately 9-10 years of age) spelling accuracy \( (F_{1,52} = 6.63; \eta^2_p = .11; p = .013) \); however, mean HbAlc and current HbAlc values were not significant coefficients. Hannonen et al. concluded that the impact of early HbAlc levels on spelling accuracy was clinically significant. Accordingly, the authors championed Ryan’s Diathesis Hypothesis (2006) and suggested that poor metabolic control (i.e., hyperglycemia) earlier in life can impact cognitive development and consequently later academic performance. Although neither McCarthy et al. (2003) or Hannonen et al. found current HbAlc levels to be significant coefficients, Hannonen et al. found HbAlc levels a year following diagnosis as a clinically significant predictor of future performance in spelling accuracy. Thus, the use of HbAlc as a predictor appears to include assessments a year after disease onset, HbAlc levels averaged over all time points, or current HbAlc levels. Predictors that are
averaged over time or selected from a single time point might not reveal the magnitude of how fluctuations over time might impact academic performance.

Other studies have not found an inverse relation between HbAlc and achievement. Prior to Kaufman et al.’s (1999) work, Kovacs, Goldston, and Iyengar (1992) employed a repeated measures analysis for a 6-year longitudinal study following youth with type 1 diabetes \((n = 87; 8.2 – 13.8 \text{ years of age})\) and determined that metabolic control (i.e., HbAlc) did not provide any explanatory power over and above duration of type 1 \((F = 29.53, p < .005)\) on Weschler Intelligence Scale for Children – Revised (WISC-R) Vocabulary scaled scores. Based on this logic, Kovacs et al. omitted metabolic control as a predictor in the final model examining academic outcomes (i.e., GPA). Kovacs et al. concluded that although a progressive decline in school grades was evidenced \((t [60] = 2.15, p = .003)\) for youth with type 1, it was the duration of type 1 rather than poor metabolic control that impacted academic performance. Notably, it was unclear from the study if mean HbA1c values across all time points or HbA1c values from a single time point were used as predictors. The appropriate author identified in the Kovacs et al. study was contacted regarding this matter, but no response was provided. In contrast to the Kovacs et al. study, Kaufman et al. reported finding no association between duration of type 1 and achievement.

Fox, Chen, and Holmes (2003) examined memory and learning assessment scores of youth with type 1 diabetes \((n = 95, \text{ age range } = 7-9 \text{ years})\) and healthy controls \((N = 100)\) 4 years after an initial evaluation. Mean HbAlc was not found to be a statistically significant regression coefficient \((\beta = -.03; p = .7768)\) when predicting learning scores 4 years later; however, gender \((\beta = -.23; p = .0315)\) and disease duration \((\beta = -.4; p = \)
.0086) were found to be significant coefficients. In regard to gender, being a male youth with type 1 predicted worse performance on the percentage of words learned compared to female youth with type 1 at follow-up.

In summary, the Hannonen et al. (2012), Kaufman et al. (1999), McCarthy et al. (2003), and Parent et al. (2009) studies revealed that the examination of HbAlc at a single time point (e.g., year after diagnosis, most recent HbAlc) revealed an inverse relation between HbAlc and current achievement. Conversely, mean HbAlc across time points or HbAlc from a single time point were not found to be significant predictors in regression models (Fox et al., 2003; Hannonen et al., 2012; Kovacs et al., 1992; McCarthy et al., 2003), the only exception being Hannonen et al. reported HbAlc levels one year after diagnosis as a significant predictor of future performance in spelling accuracy. Thus, the use of either mean HbAlc or HbAlc from a single time point, might not reveal the magnitude of how fluctuations in metabolic control over time might impact academic performance. To date, latent growth modeling has only been used to compare achievement trajectories for youth with type 1 diabetes to healthy controls (McCarthy et al., 2002), rather than examining the impact of metabolic control trajectories on achievement scores of youth with type 1 exclusively. Alternatively, studies examining cognition in youth with type 1 have employed latent growth modeling to study the impact of metabolic control on cognitive impairment. Kent et al. (2010) examined the trajectories of memory scores over the course of 3 years and found a significant effect and an inverse relation between metabolic control (i.e., HbAlc) and visual memory scores ($t = -2.78, p < .01$), while negative effects of disease duration, age of onset, and
severe hypoglycemia were not evidenced. Thus, studies examining the impact of trajectories of metabolic control on achievement appear warranted.

**Age of onset.** In the Gaudieri et al. (2008) meta-analysis youth with early onset of type 1 diabetes evidenced lower overall academic achievement scores ($d = -.19, p = .00$) than youth with late onset type 1. In contrast, Fox et al. (2003), Kaufman et al. (1999), McCarthy et al. (2003), and Parent et al. (2009) reported finding no significant association between age of onset and achievement scores. Thus, while the some studies indicate that early age on onset is a risk factor for lower achievement scores, discordance remains in the literature.

**History of severe episodes of hypoglycemia.** Unlike some authors of cognitive studies that suggested a history of severe episodes of hypoglycemia was related to impairment (e.g., Northam et al., 2001), one study that examined achievement found that students with a history of severe episodes of hypoglycemia scored higher than participants with fewer or no episodes of hypoglycemia (Hannonen et al., 2012). Similarly, in a recent meta-analysis conducted by Gaudieri et al. (2008), the authors found that youth with a history of severe hypoglycemic episodes performed better than youth that did not experience severe hypoglycemic episodes in the areas of visual memory ($d = .13$) and learning ($d = 0.12$). Gaudieri et al. proposed that better metabolic control, associated with a 2 to 3-fold increase in hypoglycemic episodes (e.g., DCCT, 1993), likely provided a protective factor against microvascular complications associated with cognitive impairment.

In regard to achievement, Hannonen et al. (2012) reported that overall, severe episodes of hypoglycemia did not appear to impact academic skills. Differences in
mathematic achievement ($F_{2,151} = 4.50, p = .013$), however, were reported when scores of youth with type 1 diabetes were compared to controls. Only youth with type 1 without a history of severe episodes of hypoglycemia performed worse on mathematic assessments compared to healthy controls at a statically significant level ($p = .003$). Specifically, 31% of youth with type 1 without a history of severe episodes of hypoglycemia demonstrated problems with mathematics in comparison to 19% of youth with type 1 with a history of severe episodes of hypoglycemia and 10% of the healthy controls (Hannonen et al.). Thus, while youth without a history of severe episodes of hypoglycemia demonstrated more problems in mathematics than controls, all youth with type 1 evidenced more problems in mathematics than controls.

In summary, although a review of the literature suggested that episodes of severe hypoglycemia may not impact achievement overall (Kaufman et al., 1999; Hannonen et al., 2012), some authors (Desrocher & Rovet, 2004; Hannonen et al.; Holmes et al., 1995; Kaufman et al.; McCarthy et al., 2003) stated that it still posed a risk for cognitive impairment for youth with type 1 diabetes. Specifically, severe episodes of hypoglycemia are related to physiological changes in the brain, such as neuronal necrosis following profound and extended periods of severe hypoglycemia (Ryan, Gurtunca, & Becker, 2005). Other authors (Ryan, 2006; Lin et al., 2010) have suggested that it is not severe episodes of hypoglycemia alone, but rather a synergetic effect of multiple risk factors may impact cognition including early age of onset and hyperglycemia as well. While the majority of studies suggested that the impact of severe episodes of hypoglycemia on cognition and learning was benign (Gaudieri et al., 2008; Kent et al., 2010; Naguib et al., 2009), discordance persists in the literature. In regard to
achievement, Hannonen et al. reported that youth with a history of severe episodes of hypoglycemia actually performed better than youth that did not experience any severe episodes of hypoglycemia when compared to controls. This finding might not be unexpected considering authors such as Gaudieri et al. proposed that the frequency of severe episodes of hypoglycemia might indicate overall better metabolic control. According to that logic, youth would be better protected against hyperglycemia related neurotoxicity and microvascular complications associated with cognitive impairment. Support for this position can be found in Kaufman et al.’s study (1999) that reported participants with 10 or more blood glucose readings <70mg/dL per month (not identified as severe hypoglycemia) scored higher than participants with less than 10 blood glucose readings <70mg/dL per month on the WJ-ACH in the areas of Broad Reading (104 ± 18 vs. 93 ± 21; p < .03) and Passage Comprehension (106 ± 18 vs. 92 ± 20; p < .008).

Moreover, severe episodes of hypoglycemia might be part of a complex interaction with other risk factors (Ryan; Lin et al.) and might not reveal a substantive impact on achievement alone. Thus, a participant’s history of episodes of severe hypoglycemia does not appear to be a substantive predictor of achievement outcomes when examining outcomes amongst only participants with type 1 diabetes.

**Conclusions related to impact of type 1 diabetes on achievement.** Discordance is present in the literature regarding the impact of medical factors (e.g., age of disease onset, history of severe episodes of hypoglycemia, poor metabolic control) related to type 1 on achievement. Thus, further examination of the magnitude of the impact of factors related to type 1 on achievement appears warranted given the discordance in the literature. Nonetheless, evidence suggesting early onset of the disease (Gaudieri et al., 2008) and
poor metabolic control (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009) are risk factors for poor achievement can be found in the current literature. It should be noted that the aforementioned conclusions were also championed by Taras and Potts-Datema (2005) and Kucera and Sullivan (2011), although their literature review did not include all of the studies presented in this literature review.

In the context of the school environment, school administrators, faculty, nurses, and staff are responsible for ensuring a safe educational environment and providing support or assistance in completing daily medical procedures when appropriate (ADA, 2012a; Silverstein et al., 2009). When considering the aforementioned medical factors and the associated impact on achievement, the only factor a school can likely influence is metabolic control through appropriate support or assistance (Taras & Potts-Datema, 2005). Thus, the role of the school or Local Education Agency (LEA) in type 1 diabetes care in the school environment will be explored with an emphasis on metabolic control.

**Role of the Local Education Agency (LEA) in diabetes care.** Diabetes education is paramount for the youth, primary caregiver(s), and family to ensure best practices are implemented in diabetes management (ADA, 2012b; NDEP, 2010; Silverstein et al., 2005). Through extension, education is also necessary for any organization or individual that assumes responsibility for the youth such as the youth’s LEA (ADA, 2012a; NDEP). As stated in Silverstein et al., “…school can present significant challenges or be a source of support to the child with diabetes” (p. 190).

The NDEP (2010) identified four goals of diabetes management in the educational environment to include (a) the youth’s immediate safety, (b) safeguarding the youth’s long-term health, (c) ensuring optimal academic performance and full
participation in school related activities, and (d) decreasing the probability of diabetes-related classroom disruptions. In addition, Silverstein et al. (2009) stated that goals might also include the promotion of normal growth, development, and socialization for youth with diabetes. In order to meet the aforementioned goals, both the ADA (2012a) and the NDEP (2010) recommend generating individualized Diabetes Medical Management Plans (DMMP) for youth with diabetes in school.

**Diabetes care plans.** Both the ADA (2012a) and the NDEP (2010) have provided general guidelines regarding diabetes care in school and templates publicly accessible at no charge for generating individualized DMMP, Individualized Health Care Plans (IHP), Emergency Care Plans for Hypo- and Hyperglycemia. The NDEP has also provided a website for school personnel, parents, and youth with resources that can be downloaded for free ([http://ndep.nih.gov/hcp-businesses-and-schools/Schools.aspx](http://ndep.nih.gov/hcp-businesses-and-schools/Schools.aspx)). Resources include a guide for school personnel ([http://ndep.nih.gov/publications/PublicationDetail.aspx?PubId=97#main](http://ndep.nih.gov/publications/PublicationDetail.aspx?PubId=97#main)) consisting of a diabetes primer for school personnel, identified actions for specific personnel, and the aforementioned templates for medical plans. Health care professionals and caregiver(s), as well as the youth, if applicable, are responsible for completing the DMMP (NDEP). The school nurse is responsible for integrating the information provided in the DMMP to generate the IHP and emergency care plans that will be implemented in the school (NDEP). An IHP will likely identify (a) the youth’s diabetes care plan (e.g., target blood glucose range, blood glucose monitoring schedule, insulin administration guidelines and schedule, treatment for hypo- and hyperglycemia, dietary plan, strategies for managing glucose levels during exercise); (b) level of permission to treat hypoglycemia (e.g., eating
snacks in classroom); (c) access to drinking water and bathroom, (d) plan for school-related actives (e.g., field trips); (e) level of permission to contact caregiver(s) or health care professional (e.g., cell phone, office phone); (f) schedule of training for school personnel; (g) dates for review of youth’s school-related outcomes; (h) services provided during the school day (e.g., nurse); (i) accommodations (e.g., school nurse’s office for monitoring blood glucose or administering insulin); (j) the reservation of penalty for absences related to illness or medical appointments; (k) diabetes management tasks that can be completed independently by the youth and subsequent permission to complete said tasks and possess required equipment; (l) location, storage, and disposal of diabetes care supplies (e.g., insulin, syringes, lancets, glucagon emergency kit); (m) nutritional information concerning cafeteria food; and, (n) the youth’s right to privacy (ADA, 2012a; NDEP). An emergency care plan will include the identification and treatment of severe episodes of hypo- or hyperglycemia (NDEP). Where the ADA and NDEP differ was in the extent of coverage of the medical plans. Specifically, the ADA (2012a) recommended that the aforementioned diabetes medical plans should be developed under an applicable federal law, while the NDEP stated that a diabetes medical plan should be generated at a minimum. The NDEP did recognize, however, that while a DMMP could be used assist in generating a Section 504 Plan or an Individualized Education Program (IEP), it was not a substitute for coverage under a federal law.

**Applicable federal laws.** Section 504 of the Rehabilitation Act of 1973, the Individuals with Disability Education Improvement Act (IDEA), and the Americans with Disabilities Act of 1990 were designed to prevent discrimination based on disability and recognized youth with diabetes as disabled (ADA, 2012a; Jacob & Hartshorne, 2007;
NDEP, 2010). The Office of Civil Rights (OCR) enforces the aforementioned federal laws (NDEP). Under federal law, students considered to have a disability are guaranteed a free appropriate public education in the least restrictive environment (Jacob & Hartshorne, 2007). A Section 504 Plan ensures students with a disability are provided with reasonable accommodations to participate in and benefit from school programs and activities alongside non-disabled peers. A Section 504 Plan may be generated to ensure equal opportunities and appropriate medical care for youth with diabetes; however, coverage under IDEA might be warranted if a student’s educational performance is adversely impacted by diabetes sequelae (e.g., inattention, difficulty concentrating; NDEP). If evidence could be provided for the illness adversely impacting educational performance, youth with diabetes could potentially be recognized as meeting definitional criteria for a disability under the Other Health Impairment category of IDEA (NDEP). Consequently, an IEP could then be generated for the student. In short, while DMMP are recommended for youth with diabetes at a minimum (NDEP), a diabetes care plan written under an applicable federal law (e.g., 504 Plan, IEP) and enforced by the OCR (ADA, 2012a) would likely ensure said medical plan was followed during the school day.

**ADA recommendations and discordance with actual care provided.** Previous studies have indicated the need for identifying youth with type 1 diabetes as an at-risk population (e.g., Holmes, Dunlap, Chen, & Cornwell, 1992), discussed the importance of school personnel and student trainings (e.g., Wagner et al., 2006), and revealed some alarming statistics concerning youth with type 1 that do not have medical care plans in school (e.g., Jacquez et al., 2008). Holmes et al. concluded that youth with type 1 were more likely to experience learning problems as 24% of youth with type 1 received special
education services compared to 13% of the control group ($F_{1,188} = 4.06, p = .0453$).

Additionally, it was reported that more males with type 1 (29%) received remedial or resource room instruction than females with type 1 (11%) or healthy male (5%) or female (6%) controls ($F_{1,188} = 4.65, p = .0323$; Holmes et al., 1992). Also, parent report indicated that more males with type 1 (21%) had repeated a grade than females with type 1 (4%) or healthy controls (5%; $F_{1,188} = 8.58, p = .0038$; Holmes et al., 1992). Further, more behavioral problems at school were reported for youth with type 1 (16%) compared to healthy controls (5%; $F_{1,188} = 5.93, p = .0158$; Holmes et al., 1992).

In regard to the importance of school personnel and student trainings, the ADA (2012a) has published a position statement wherein school personnel are to receive varying degrees of training from the school nurse or another health professional concerning diabetes care and emergency management dependent upon the level of the interaction with the student. For example, all personnel at the school should receive basic training in diabetes care, while a select few should receive second or third tier training to provide appropriate assistance to the youth (ADA, 2012a; NDEP, 2010). For instance, a classroom teacher would likely receive second tier training to know how to respond to an emergency. In addition, personnel with third tier training would be able to respond to emergencies and assist youth that demonstrate a need for help with regular diabetes care tasks such as administering insulin or blood glucose monitoring. The ADA (2012a) recommends that at a minimum, one adult should be present to assist the youth at all times whether that be the school nurse, who is the principle provider and coordinator of care, or an adult trained by the school nurse or other health professional. In contrast to the recommendations of the ADA, Wagner et al. (2006) indicated from a sample of
parents \((n = 58)\) of youth with type 1 diabetes, only 58% reported that school personnel had received any training in diabetes care. When training had been provided, 48% of trainings had been provided by parents for school personnel. Training quality reportedly varied from informal conversations to consultation with a healthcare professional. Notably, mean HbA1c levels for youth with trained school personnel were significantly lower (7.7%) than mean HbA1c levels for youth with untrained school personnel (8.4%; \(F_{3,41} = 5.12, p < .05\)). Further, youth with type 1 reported higher scores on a diabetes-related quality of life assessment when classmates had been provided with an informal presentation conducted by a parent or school nurse concerning basic diabetes information and identifying and treating hypoglycemia. Thus, this study identified the substantial impact school personnel and peer trainings can have on improving metabolic control and higher diabetes-related quality of life ratings for youth with type 1 diabetes.

Jacquez et al. (2008) examined parent report \((n = 302)\) of diabetes management and awareness of federal laws related to diabetes care in schools of a diverse sample in terms of ethnicity (youth identified by parents as 61% Hispanic white, 19% non-Hispanic white, 19% African or Caribbean American, 1% other) and SES. The sample predominately represented youth with type 1 diabetes (88%), along with smaller proportions of youth with type 2 (9%) and atypical (3%) diabetes. Overall, nearly half of youth with diabetes (45%) did not have a written diabetes care plan or a nurse (45%) at school. Racial disparities were evidenced in the percentages of youth without written care plans or a school nurse. Specifically, youth with diabetes identified as non-Hispanic white were more likely to have a written care plan (74%) than Hispanic (52%) or Black (46%) peers at a statistically significant level \((p < .05)\). Additionally, youth with diabetes
identified as non-Hispanic white had a greater likelihood of having a school nurse (78%, \( p < .05 \)) than Hispanic (48%) or Black (54%) peers. Further, only 49% of parents reported that their child had a glucagon emergency kit at school. Again, significant racial disparities were reported as non-Hispanic white youth (70%) were more likely to have a glucagon emergency kit at school than Hispanic (45%) or Black (43%) peers.

Parents from the Jacquez et al. (2008) study reported that most youth were not permitted to perform diabetes management tasks in the classroom such as blood glucose monitoring (52%) or administration of insulin (79%). Shockingly, only 46% of parents reported that insulin injections were allowed on campus in specially designated areas. Similarly, only 50% of parents reported schools allowing blood glucose checks in specially designated areas.

In regard to permission to access snacks or drinking water or a bathroom, most youth were allowed to eat snacks when required (84%) and access bathroom when required (81%). Racial disparities, however, were revealed in these areas. For example, youth identified as Black had a significantly higher risk of being denied access to snacks, drinking water, and a bathroom than non-Hispanic white or Hispanic peers.

A lack of awareness concerning applicable federal laws amongst parents of youth with diabetes was evidenced in the Jacquez et al. (2008) study. For instance, while parents of youth with type 1 diabetes reported higher awareness of federal laws, overall, parents’ reported awareness of federal laws including ADA, Section 504, and IDEA were 36%, 33%, and 13%, respectively. Racial and SES disparities regarding unawareness of federal laws were notable. Parents identified with high SES levels reported significantly higher levels of awareness of Section 504 and ADA compared to parents identified with
middle or low SES levels. Parents of non-Hispanic white youth reported significantly more awareness of Section 504 and ADA than parents of Hispanic or Black youth. Most parents (87%), regardless of ethnicity or SES level, reported unawareness of IDEA. Not surprisingly, the majority of parents surveyed indicated concern regarding the school’s ability to meet their child’s diabetes care needs. Jacquez et al. (2008) concluded that increased advocacy for youth with diabetes in school and applicable federal laws that necessitate written diabetes care plans was evidenced.

In contrast to the ADA (2012a) recommendations for allowing responsible youth to perform necessary, daily diabetes care tasks in the classroom, 56% of youth in one study reported having to leave the classroom to monitor blood glucose levels, treat hypoglycemia, or inject insulin (Wagner et al., 2006). Similarly, Jacquez et al. (2008) reported that most youth with diabetes were not permitted to monitor blood glucose levels (52%) or inject insulin (79%) in the classroom. To quantify the importance of responsible youth being able to check his or her blood glucose levels in class when needed, Wagner et al. reported that mean HbA1c levels were poorer for youth that reported having to leave the classroom to attended to diabetes-related care (8.4%) than mean HbA1c levels for youth allowed to remain in class (7.5%) at a statistically significant level \( F_{4,48} = 17.31, p < .001 \). Moreover, Rausch et al. (2012) reported in a 2-year longitudinal study of adolescents that the loss of even one daily blood glucose check predicted an increase in HbA1c of 1.26%. Further, DCCTEDIC Research Group (2001) discussed the deleterious effect poor metabolic control can have on future health outcomes. Outcomes were compared for adolescents concerning the prevention of microvascular complications in both the conventional and intensive treatment groups of
the DCCT using data from the follow-up study to the DCCT, the EDIC. While mean HbAlc levels were similar for adolescents in the intensive therapy (8.38%) versus conventional (8.45%) therapy groups 4 years after the DCCT, it appeared that the impact of the significantly lower ($p < .0001$) mean HbAlc level for the intensive therapy group (8.06%) as compared to the conventional therapy group (9.76%) at the end of the DCCT was a protective factor against microvascular complications. Specifically, lower HbAlc levels at the end of the DCCT reduced the risk of progression to proliferative retinopathy by 74% ($p < .001$) or severe nonproliferative retinopathy by 78% ($p < .007$) for the adolescents formerly in the intensive therapy group when compared to their peers in the conventional therapy group only 4 years later in the DCCT/EDIC study. The authors suggested that this finding provided evidence that intensive therapy should be implemented in adolescence as a protective factor against the development of microvascular complications later in life, or in this case of this study, only 4 years later. Intensive treatment involves testing blood glucose levels 4 or more times per day (Silverstein et al., 2005). Further, Wodrich et al. (2011) suggested that having to leave a classroom to check one’s blood glucose level not only delayed treatment, but also increased the amount of instructional time missed by the student. Thus, the absence of a medical care plan or a school climate that does not support the responsible youth in conducting essential, daily diabetes care tasks including blood glucose monitoring in class will likely lead to fewer blood glucose checks per day, potentially missing additional instructional time, and consequently poorer metabolic control.

Conclusions related to the role of the LEA in diabetes care. Researchers recommend promoting better metabolic control in school with the aim of removing
barriers to enable students to perform to the best of his or her ability level in school (Holmes et al., 1999; Parent et al., 2009). From a health perspective, researchers have also recommended facilitating an intensive treatment regimen for adolescents to improve glycemic control in an effort to reduce the risk of developing future microvascular complications (DCCT/EDIC, 2001). Additionally, the ADA recommended that medical care plans should be developed for youth with diabetes under applicable federal laws to ensure that essential diabetes related care is provided throughout the school day and an emergency plan is in place (ADA, 2012a). Further, educating teachers, administrators, classmates, and appropriate school staff might also help to improve the management of the disease in school and quality of life for youth with type 1 diabetes (Wagner et al., 2006). Thus, given that school-age youth spend a substantial portion of their day in an educational setting, it would behoove educators, administrators, and health care professionals to work toward facilitating the successful implementation of an intensive treatment regimen as defined by the ADA (Silverstein et al., 2005) for youth with type 1 in the school setting under an applicable federal law (ADA, 2012a).

In conclusion, four critical goals for diabetes care in school can be summarized to include ensuring the youth’s (a) safety, (b) immediate and long-term health, (c) optimal academic performance, and (d) full participation in school related activities (NDEP, 2010). All of these goals identified through written diabetes care plans under applicable federal laws (ADA, 2012a; NDEP, 2010). Unfortunately, according to the literature, most parents are unaware of applicable federal laws related to diabetes care in school (Jacquez et al., 2008), nearly half of students with diabetes do not have written medical care plans (Jacquez et al.), and most school personnel do not have adequate training in
diabetes management (Wagner et al., 2006). The ADA (2012a) recommends using an applicable federal law to ensure a written plan is developed for all youth with diabetes. Federal laws are paramount in preventing discrimination based on disability and ensuring a free, appropriate public education in the least restrictive environment (Jacob & Hartshorne, 2007). Moreover, personnel trainings, appropriate support staff (e.g., nurse), and additional academic support could be included in a Section 504 Plan or an IEP under IDEA. Further, previous studies have evidenced an inverse relation between HbA1c and achievement and have accordingly recommended better metabolic control in school to optimize school performance (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009). Thus, further research examining the relation between metabolic control and achievement might convey the importance of developing a diabetes care plan to educators as some authors suggest (Holmes et al., 1999; Parent et al., 2009, Taras & Potts-Datema, 2005) that metabolic control should be attended to in school.
CHAPTER II: THE CURRENT STUDY

The Current Study

Type 1 diabetes is a common chronic health condition occurring in children and adolescents (National Diabetes Education Program [NDEP], 2010). Based on data from 2002-05, 15,600 new cases of type 1 were diagnosed among school-aged youth annually (Centers for Disease Control and Prevention [CDC], 2011). Youth diagnosed with type 1 are at-risk for cognitive impairments, learning problems, and poor academic performance (Gaudieri, Chen, Greer, & Holmes, 2008; Kent, Chen, Kumar, & Holmes, 2010; Lin, Northam, Rankins, Werther, & Cameron, 2010; Naguib, Kulinskaya, Lomax, & Garralda, 2009). Accordingly, a meta-analysis (Gaudieri et al.) revealed that youth with type 1 scored lower on achievement assessments than healthy peers, evidencing a small effect ($d = -.13$). Further, evidence in the literature suggests an inverse relation between metabolic control and current achievement for youth with type 1 (Hannonen et al., 2012; Kaufman, Epport, Engilman, & Halvorson, 1999; McCarthy, Lindgren, Mengeling, Tsalikian, & Engvall, 2003; Parent, Wodrich, & Hasan, 2009).

Nevertheless, the current literature is equivocal as to the relationship between metabolic control and student academic achievement. For instance, some studies have reported that higher HbA1c (indicating poorer metabolic control) is associated with poorer concurrent academic achievement (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009). In contrast, Fox et al. (2003) found mean HbA1c level collected over the course of the study was not a significant predictor of subsequent learning or achievement measured 4 years later as analyzed in a longitudinal regression model (Fox et al., 2003). Predictors such as mean HbA1c or HbA1c from a
single time point might not reveal how the range of changes in metabolic control over time might relate to academic performance for youth with type 1. This discordance indicates that more research is needed to better understand the nature of the relation between longer term metabolic control and academic outcomes in order to better serve this at-risk student population.

Although latent growth models (LGM) have been successfully applied to examine health behaviors related to metabolic control (e.g., Rausch et al., 2012), there is a dearth of LGM research concerning achievement outcomes related to metabolic control for adolescents with type 1 diabetes. The majority of research articles addressing achievement outcomes related to metabolic control for youth with type 1 have employed a cross-sectional (e.g., McCarthy et al., 2003) or case control design (e.g., Hannonen et al., 2012). While evidence suggests the existence of an inverse relation between metabolic control and achievement (e.g., McCarthy et al., 2003), no studies to date have explored a possible link between longitudinal changes in metabolic control and academic achievement.

Two studies (Kent, Chen, Kumar, & Holmes, 2010; Kovacs et al., 1992) provide evidence that suggests a potential link between a decline in achievement or visual memory scores overtime and diabetes-related factors. Kovacs et al. (1992) employed a repeated measures analysis for a 6-year longitudinal study following youth with type 1 diabetes. The authors determined that metabolic control did not provide any explanatory power over and above disease duration when predicting standardized vocabulary scores. Consequently, metabolic control was excluded as a predictor through model trimming. Kovacs et al. concluded that although a progressive decline in school grades was
evidenced for youth with type 1, it was the disease duration rather than poor metabolic control that impacted academic performance. In contrast, Kaufman et al. (1999) reported finding no association between duration of type 1 and achievement. Notably, a limitation of a repeated measures design is that variation amongst the growth trajectories of individual cases is interpreted as error variance (Kline, 2005, p. 278). Conversely, variation in individual growth trajectories over time is not viewed as error variance in LGM, but rather is the focus of the investigation (Kline).

Supportive of the value of taking a LGM approach, Kent and colleagues (Kent et al., 2010) examined cognition amongst youth with type 1 employing a LGM and reported that as HbA1c trajectories increased over a 3-year period, visual memory scores correspondingly decreased. As such, Kent et al.’s work is suggestive of the possible link between longitudinal changes in metabolic control and academic achievement examined in the present study. Such results are important as previous LGM studies have indicated metabolic control tends to deteriorate during adolescence for some youth with type 1 diabetes (Helgeson et al., 2010; King et al., 2012; Luyckx & Seiffge-Krenke, 2009; Miller et al., 2013; Rausch et al., 2012). Specifically, Rausch et al. and Miller et al. reported adolescent HbA1c levels increased significantly at each sampling round by .2 (baseline, 6 months, 18 months) -.24 (baseline, 12 months, 24 months) units, respectively. Thus, given the propensity for metabolic control to perhaps worsen during adolescence, a negative correlation between metabolic control and academic performance (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009) might make adolescents with type 1 a particularly vulnerable student population.
Although there is discordance in the current literature, predictors of academic outcomes need to be identified in order to examine the unique effects of changes in metabolic control over time on academic performance in youth with type 1. Meaningful predictors of achievement, learning, or memory in previous studies include participant age (Kent et al.; DiPerna, Lei, Reid, 2007) and sex (Fox et al., 2003; Kent et al.); disease duration (Kovacs et al., 1992; Fox et al., 2003); cognitive ability score (Hannon et al., 2012; Kovacs et al., 1992); and, prior achievement (Volpe et al., 2006). Notably, the effects of frequency of hypoglycemia on cognition have been reported as benign (Gaudieri et al.; Kent et al., 2010; Naguib et al., 2009). Likewise, Hannonen et al. reported that youth with a history of severe episodes of hypoglycemia actually performed better than youth that did not experience any severe episodes of hypoglycemia when compared to controls on mathematics assessments. Further, recent studies have revealed the effects of school absences on achievement appear negligible for youth with type 1 (McCarthy et al., 2003).

In regard to metabolic change over time, evidence is present to warrant the examination of cognitive ability score (derived from a standardized assessment), age, sex, duration of disease, and initial HbA1c level of the participants as relevant predictors. Previous investigators have postulated that the participant’s cognitive ability level might influence metabolic control (Kaufman et al., 1999; McCarthy et al., 2002, 2003). In contrast, Miller et al. (2013) reported that teens’ executive functioning based on maternal caregiver scores on the Behavior Rating Inventory of Executive Function (BRIEF) did not predict the rate of change in metabolic control. Additionally, participant age and disease duration have been identified as significant predictors of metabolic control.
trajectories (Hilliard, Wu, Rausch, Dolan, & Hood, 2013). On the contrary, Helgeson et al. (2010) reported neither participant age nor disease duration were significant predictors of trajectories of metabolic control. Further, some studies indicated that sex was not a significant predictor of metabolic change (Helgeson et al., Hilliard et al.), while adolescent girls were overrepresented in a deteriorating metabolic control trajectory (latent) class in a study by Luyckx & Seiffge-Krenke (2009). Finally, insulin delivery method at the start of one study was no longer a significant predictor of metabolic control trajectories once initial HbA1c level was included in the model (Helgeson et al.). Rather, initial HbA1c level was a significant predictor of metabolic control trajectories (Helgeson et al.). Thus, although discordance is present in the literature, the aforementioned relevant predictors will be included in the analysis.

**Rationale and Purpose.**

The purpose of this study was to examine the effect of metabolic control fluctuation as a limiting factor related to the academic performance of adolescents diagnosed with type 1 diabetes over a 3-year period. Specifically, a LGM was employed to examine the limiting effect of the growth curve of adolescents' (aged 10-14 years of age at study initiation) metabolic control sampled every six months on academic achievement at the end of six sampling rounds. Four hypotheses were tested through this analysis: (a) higher initial adolescent HbA1c will result in lower GPA scores at the end of 3 years, (b) growth will be positive for HbA1c indicating deterioration of metabolic control during adolescence, (c) a greater increase in adolescent HbA1c across sampling rounds will result in a lower final GPA score, and (d) a positive covariance will emerge between HbA1c intercept and the HbA1c slope (i.e., higher initial HbA1c levels will lead
to faster increases in HbA1c levels across the sampling rounds). Overall, poor metabolic control was expected to be a limiting factor concerning academic performance at the final time point.
CHAPTER III: METHODS, RESULTS, AND DISCUSSION

Methods

Participants

Participants included 217 adolescents diagnosed with type 1 diabetes ($M_{baseline\ age} = 12.61\ years,\ SD = 1.49;\ 54.8\%\ female$). Criteria for eligibility included youth that were between 10-15 years of age at baseline, literate in either English or Spanish, and were diagnosed with type 1 for 1 year or more. At baseline, parents’ report indicated that participants had been diagnosed with type 1 for an average of 4.65 years ($SD = 2.97$). Approximately half of participants (53%) were on Continuous Subcutaneous Insulin Infusion (CSII; i.e., insulin pump therapy) at baseline. Demographically, this sample was relatively homogenous. In regard to ethnicity, 94.5% of mothers identified youth as non-Hispanic White. Additionally, 67.3% of mothers reported annual household income $\geq$ $50,000$ and 84.79% reported some college education or higher. Participants and their families were recruited as part of a larger study funded through R01 DK063044-01A1 from the National Institute of Diabetes and Digestive Kidney Diseases (NIDDK).

Measures

**HbA1C.** HbA1c was used to determine levels of average glucose concentration in the blood stream over a period of 2-3 months (ADA, 2012; Beaser; CDC, 2008). HbA1c levels less than 7.5% are recommended for adolescents and young adults [13-19] ADA, 2012). HbA1c was assessed every 6 months for each participant by clinic staff employing the Bayer DCA 2000.

**GPA.** Previous studies employing structural equation modeling (SEM) have provided evidence for the utility of including prior achievement as a predictor of
achievement (Byrnes & Miller; 2007; Volpe et al., 2006). In the current study, the participants’ most recent GPA score was reported by the participants’ mothers during each 6-month clinic visit. At Time 6, mothers’ reports of GPA correlated highly with GPA in adolescents’ school records ($r = .89, p < .001$), lending validity to mothers’ reports of GPA. In the current study, GPA at Time 6 was taken from the student’s school record.

**Covariates.** Rationale for selecting covariates was based upon prior research and the demographics of the current sample. Covariates assessed at baseline considered as meaningful predictors of achievement, learning, or memory included youth’s: IQ (Hannon et al., 2012; Kovacs et al., 1992), prior achievement (Byrnes & Miller, 2007; Volpe et al., 2006), duration of diabetes (Gaudieri et al., 2008; Kovacs et al., 1992; Fox et al., 2003), age (Kent et al.), and sex (Fox et al., 2003; Kent et al.). Additionally, covariates assessed at baseline considered to be meaningful predictors of metabolic control included youth’s cognitive ability score (Kaufman et al., 1999; McCarthy et al., 2002, 2003), insulin delivery method (e.g., insulin pump; Hilliard et al., 2013; Rausch et al., 2012), and age and disease duration (Hilliard et al.). Further, rationale for examining sex as a predictor for metabolic control has also been found in the literature (e.g., Luyckx & Seiffge-Krenke, 2009).

**Kaufman Brief Intelligence Test (K-BIT).** The Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990) was used as a measure of IQ. The K-BIT can be administered in 15-30 minutes and consists of two subtests, Vocabulary and Matrices (Canivez, Neitzel, & Martin, 2005). Previous research has provided evidence supporting the construct validity of the K-BIT as well as the utility of using K-BIT scores to predict
achievement scores (Canivez et al.). Specifically, the K-BIT IQ Composite and Full-Scale IQ (FSIQ) scores of the Wechsler Intelligence Scale for Children, Third Edition (WISC-III; Wechsler, 1991) were highly correlated and statistically significant ($r = .89$) indicating both scales are measuring the same construct (i.e., intelligence; Canivez et al.). Additionally, K-BIT IQ Composite scores were significantly correlated with standardized achievement scores (e.g., Woodcock-Johnson series; Woodcock, McGrew, & Mather, 2001) in the areas of basic reading ($r = .60$), reading comprehension ($r = .66$), mathematics calculation ($r = .61$), mathematics reasoning ($r = .72$), and written expression ($r = .55$; Canivez et al.). Further, test-retest reliability coefficients for K-BIT IQ Composite scores range from .92 to .95 and internal consistency coefficients range from .88 to .98 indicating high reliability (Sattler, 2001). Rationale for including cognitive ability score as a covariate stems from previous researchers postulating that better metabolic control might be related to the cognitive ability level of the youth (Kaufman et al., 1999; McCarthy et al., 2002, 2003).

**Design and Data Analysis**

**Data preparation.** Multivariate normality, linearity, and homoscedasticity were examined (Kline, 2005). Univariate and multivariate normality were examined using 9.1 of LISREL for Windows (Joreskog & Sorbom, 2012). Univariate analyses did not reveal extreme absolute values of skewness ($< 3.0$) or kurtosis ($< 8.0$; Kline, 2005). Nonetheless, univariate skewness and kurtosis values indicated moderate nonnormality. Skewness and kurtosis values are presented in Table 1. Standardized residual plots provided by LISREL indicated that the assumptions of linearity and homoscedasticity were not violated.
Results from a Mahalanobis distance test employing pairwise deletion revealed significant chi-square values \((p < .001)\) for four cases (75, 231, 108, 109) that were retained for the analyses. Two cases (234, 246) were removed due to missing data across all time points. One case (165) was removed due to a missing KBIT score. Further, 34 cases were removed due to missing data for GPA at both Time 1 and Time 6. Cases missing data only at GPA at Times 1 or 6 were retained. In total, 37 cases were removed, reducing the original sample size of 254 to 217. Fortunately, a sample size of 217 remains above the recommended sample size of at least 200 cases required for SEM (Kline, 2005; p. 111).

Missing data. A missing value analysis (MVA), conducted using SPSS Version 18.0, revealed >10% missing data for key outcome variables (GPA rounds 1 and 6, HbA1c round 4-6). The percent of missing data for key variables follows: GPA round 1 (20.3%), GPA round 6 (29.5%), A1C round 4 (13.8%), A1C round 5 (18.4%), and A1C round 6 (20.3%). The MVA also revealed that while the overall total of missing data values totaled 8.72%, the use of listwise deletion would result in retention of only 95 (43.78%) cases. Moreover, a statistically significant result from Little’s Missing Completely at Random (MCAR) test suggested that MCAR could not be inferred. Although MCAR cannot be presumed, the assumption of missing at random (MAR) cannot be tested directly (Schafer & Graham, 2002). For this reason, indicators of MAR were examined. Specifically, results from independent samples \(t\) tests, using A1C and GPA at round 6 as grouping variables to determine if a pattern of missing data was present, did not suggest MCAR. Further, examination of the data collection procedures and participant responses did not provide any evidence to suggest a systematic pattern of
missing data. Thus, the assumption of MAR might not have been violated and the missing data mechanism was interpreted as ignorable.

**Full Information Maximum Likelihood (FIML) Estimation.** Given the amount of missing data, Full Information Maximum Likelihood (FIML) estimation was considered. In FIML, parameters are estimated based upon all available data in contrast to traditional methods of dealing with missing data such as listwise deletion where cases with any missing data are removed from the analysis (Baraldi & Enders, 2010; Enders, 2001; Enders & Bandalos, 2001). Consequently, all cases, even those with missing data, are retained when employing FIML. Moreover, assumptions for FIML include normally distributed data and MAR or MCAR (Baraldi & Enders, 2010; Raykov, 2005; Schafer & Graham, 2002). With potential deviations from normality, FIML parameter estimates have been found to be less biased and more efficient than traditional methods of dealing with missing data when analyzing both normal (Enders & Bandalos, 2001) and nonnormal (Enders, 2001; Raykov, 2005) data distributions in SEM. Further, nonnormal data distributions are unlikely to substantively impact analyses of large samples \( n \geq 200 \) or consequently the generalizability of the results (Tabachnick & Fidell, 2007). In general, maximum likelihood estimation is robust concerning departures from normality (McDonald & Ho, 2002). Accordingly, FIML estimation was deemed appropriate for the analyses given the aforementioned moderate nonnormality of the data and the nonsystematically missing data.

As such, the covariance matrix and model parameters were estimated using FIML estimation. FIML was employed through version 9.1 of LISREL for Windows (Joreskog
& Sorbom, 2012). The use of FIML ensured the recommended sample size of at least 200 cases was retained for SEM (Kline, 2005; p. 111).

**Analysis.** The linearity and fit of the growth model for HbA1c values were assessed before constructing the full model based on Kline’s (2005) recommendations. Kline suggested a two-step analysis where the repeated measures variables are analyzed first as a change model. If the change model is acceptable, the second step is comprised of adding predictors to the model. The first step of this analysis is represented as a change model for HbA1c values as presented in Figure 1. The acceptable LGM for HbA1c along with the covariates were then combined to form the full model to predict GPA at Time 6 as presented in Figure 2.

The chi-square test statistic and the root mean square error of approximation (RMSEA; < .06) were used to evaluate the model fit to the data (Byrne, 1998; Enders, 2001; Kline, 2005; Hu & Bentler, 1999; Lei & Wu, 2007; Schafer & Graham, 2002). FIML provides a chi-square test statistic and a RMSEA value; however, fit indices such as the Comparative Fit Index (CFI) or Goodness-of-fit index (GFI) cannot be generated due to the mean structure being specified (Enders, 2001). A statistically significant chi-square test would indicate that the restrictions imposed by the proposed model (H₁) might be a poor fit to the data because the specifications may not hold in the population compared to a less restricted model (H₀; Enders, 2001; Raykov, 2005; Schafer & Graham, 2002). Notably, the chi-square test statistic is an extremely sensitive test that may indicate statistically significant results even when the proposed model may fit the data adequately (Lei & Wu, 2007). Additionally, nonnormality of the data may also lead to inflated rejection rates of the model and negatively biased standard errors (Enders,
Moreover, the chi-square test statistic assumes the model to fit the population perfectly, while the RMSEA assumes that the model is an approximation of reality and assesses error of the approximation of model fit to the population covariance matrix (Kline). Further, the RMSEA can account for model complexity whereas more parsimonious models will have lower values (Kline). Accordingly, the RMSEA will likely be the more informative indicator of model fit given the moderately nonnormal data distribution.

Correlations, means, and standard deviation values are presented in Table 1. Based upon the recommendations of McDonald and Ho (2002), parameter (standardized and unstandardized) and standard error estimates for the LGM and the full model are presented in Tables 2 and 3. Statistically significant unstandardized and standardized parameter estimates (absolute value ≥ 1.96), as well as standard error estimates, were interpreted (Lei & Wu, 2007). Standard errors (SE) of varying magnitudes were considered indicators of potential model misfit to the data (Lei & Wu). Appropriate interpretations for beta weights in educational research have been identified as small (β > .05), moderate (β > .10), and large (β > .25; Keith, 2005, p. 62).

Results

Latent Growth Model of Change in HbA1c (A1C) Values

In the change model, each value for HbA1c across the six sampling rounds (T1_A1C – T6_A1C) is an indicator for the two latent growth factors. The Initial Status (IS) latent factor is comparable to an intercept of a regression analysis. Likewise, the Linear Change (LC) latent factor is comparable to a slope. The IS loading factors were fixed to 1 and first loading on the LC factor was fixed to -3 to identify Time 4 (T4_A1C).
as the initial level (See Figure 1). The factor loadings of the LC factor for the
aforementioned sampling rounds were fixed at increasing increments of 1-point (-3, -2, -
1, 0, 1, 2) to correspond to the equal spacing between times of measurement: every 6
months (Kline, 2005, p. 276). The LC was centered at Time 4 as the average age for the
entire sample and at Time 4 was 14 years of age. Standard errors appeared to be of a
similar magnitude and none exceeded a value of 1.00 (Lei & Wu, 2007).

The model converged in 7 iterations. The RMSEA value of .067 indicated
reasonable error of approximation (Browne & Cudeck, 1993; Byrne, 1998; Kline, 2005).
The 90% confidence interval for the RMSEA value (0.030, 0.102) indicated good to
mediocre error of approximation (Browne & Cudeck, 1993; Byrne, 1998; Kline, 2005).
Additionally, the p-value for the Test of Close Fit for RMSEA was .195. As such, the
null hypothesis of close approximate fit was not rejected (Kline, 2005; p. 139). The chi-
square test statistic $X^2_{FIML}(15, N = 217) = 29.629, p = .013$, indicated that the restrictions
imposed by the proposed model ($H_1$) was likely an adequate fit to the population as the p-
value was not < .01 (Raykov, 2005).

The means were estimated through the LGM and were adjusted for measurement
error (Kline, 2005). A positive linear trend was evidenced by the estimated or fitted
HbA1c means across the 6 sampling rounds (See Figure 3). The estimated mean for LC
was .139 ($SE = .021, p < .05$), representing the average increase in HbA1c values every 6
months. The estimated mean for IS was 8.731 ($SE = .092, p < .05$), indicating the
average HbA1c value at time 4. An alternative model was tested without T6_A1C, and
the estimated average increase in HbA1C increased slightly (.176) and the fit improved
$X_{FIML}^2(10, N = 217) = 11.985, p = .286, \text{RMSEA} = .030$; however, the LGM incorporating all six time points was ultimately retained.

Variances for the two latent factors and six measurement errors were free parameters in the change models, while the measurement error covariances were fixed parameters. The statistically significant variances for IS (1.575) and LC (.061) indicated that participants’ starting HbA1c values and changes over time were heterogenous and highly variable (Kline, 2005). The error covariance between T1_A1C and T6_A1C was set free based upon modification indices and expected parameter change values (Byrne, 1998). The standardized error covariance between T1_A1C and T6_A1C was substantively meaningful (.096, $p < .05$) and the positive sign was expected based upon the positive linear growth trajectory.

Additionally, the IS and LC factors were allowed to covary to reveal the influence of IS on LC. While not statistically significant, the positive estimated covariance (.035) and the standardized factor correlation (.113) between the latent factors suggested that higher initial HbA1c values led to faster increases in HbA1c values over time. Alternatively stated, this positive covariance also suggested that lower initial HbA1c values led to slower increases in HbA1c values overtime.

**Full Model**

In accordance with Kline’s (2005) suggestion of a two-step analysis, the covariates were added to the acceptable LGM for HbA1c to form the full model used to predict GPA scores Time 6 (T6_GPA) while controlling for GPA scores at Time 1 (T1_GPA) as presented in Figure 2. Notably, the inclusion of GPA at Time 1 (baseline) as a covariate transformed GPA at Time 6 into a residualized change score (Lance, 1988;
MacKinnon, 2008, p. 199). As baseline is taken into account, results from analyses employing residualized change scores are interpreted as equivalent to an analysis of covariance (MacKinnon, 2008). As such, pathways were interpreted according to Kline’s recommendations.

The number of observations was greater than the number of parameters in the initial model. As a result, the initial model was over identified. Two pathways are of primary interest in the full model: (a) the relation between initial HbA1c values (IS) and GPA at Time 6 (T6_GPA), and (b) the relation between linear change in HbA1c values over time (LC) and GPA at Time 6 (T6_GPA). Pathways between the covariates and GPA at Time 6 (T6_GPA) as well as the latent factors were also interpreted. Unstandardized and standardized parameter estimates as well as standard errors derived from the full model are presented in Tables 2 and 3. The error variances for the covariates (T1_GPA, IQ_KBIT, Duration, Age, Sex, Insulin pump status) were fixed to zero. Standard errors appeared to be of a similar magnitude and none exceeded a value of 1.00 save for 3 of the parameters (See Tables 2 and 3; Lei & Wu, 2007).

The results for the full model are presented in Tables 2 and 3 and the model is depicted in Figure 2. The model converged in 11 iterations. The RMSEA value of .057 indicated that the model demonstrated close approximate fit to the population covariance matrix. The 90% confidence interval for the RMSEA value (.035, .078) indicated good to reasonable error of approximation. Additionally, the p-value for the Test of Close Fit for RMSEA was .269. Accordingly, the null hypothesis of close approximate fit was not rejected (Kline, 2005). The chi-square test statistic $X^{2}_{FIML}$($47, N = 217$) = 80.571 ($p = .002$) indicated that the fit of the proposed model ($H_1$) was not probable in the population.
compared to a less restricted model (H₀; Raykov, 2005). As mentioned previously, although the null hypothesis was rejected, the extreme sensitivity of the chi-square test makes RMSEA the more informative indicator of model fit (Lei & Wu, 2007). Overall, the squared multiple correlation value for T6_GPA indicated that the full model accounted for 40.4% of the variance of T6_GPA.

IS for HbA1c was a statistically significant and meaningful predictor (β = -.178) of final GPA (T6_GPA) even after controlling for previous achievement (T1_GPA), cognitive ability (IQ_KBIT), duration, age, sex, insulin pump status, and LC. The beta weight for the path from IS for HbA1c to T6_GPA was interpreted as moderate. The negative beta weight for the aforementioned pathway indicated that for every 1 standard deviation unit increase in IS (T4_HbA1c; SD = 1.71), there was a .18 standard deviation decrease in T6_GPA (SD = .69) equaling a decrease in T6_GPA by .12 points.

Conversely, LC for HbA1c was not a statistically significant or meaningful predictor (β = .075) of final GPA (T6_GPA). Although the sign was positive for the pathway from LC to T6_GPA, the large SD of .240 caused the value to cross zero and the non-significant result made this value indistinguishable from zero. An alternative model including all of the covariates was tested where the LGM for HbA1c was centered at time 1 to examine the relation between IS and T6_GPA. When the LGM was centered at time 1 (i.e., 0, 1, 2, 3, 4, 5), the relation between intercept and T6_GPA was commensurate (β = -.20, p < .05) and fit indices were identical to the final model.

Amongst the covariates, T1_GPA produced a statistically significant pathway to final GPA (T6_GPA) whereas cognitive ability (IQ_KBIT), disease duration, age, and sex did not. The path from T1_GPA to T6_GPA produced a statistically and practically
significant beta weight ($\beta = .540$) that was interpreted as large. The positive beta weight for the aforementioned pathway indicated that for every 1 standard deviation unit increase in T1_GPA ($SD = .62$) there was a .54 standard deviation increase in T6_GPA ($SD = .69$) equaling an increase in T6_GPA by .37 points. Further, an alternative model was tested by removing T1_GPA. In the alternative model, although the signs of the pathways from IS to T6_GPA and LC to GPA remained the same, neither pathway was significant and the model fit worsened $X_{FIML}^2 (41, N = 217) = 74.884 (p = .001)$, RMSEA = .062.

In regard to the covariates for IS and LC of metabolic control, cognitive ability (IQ_KBIT) and insulin pump status were meaningful predictors of IS; whereas, only duration was a meaningful predictor of LC. The path from IQ-KBIT to IS produced a statistically and practically significant beta weight ($\beta = -.321$) that was interpreted as large. The negative beta weight for the aforementioned pathway indicated that for every 1 standard deviation unit increase in IQ-KBIT ($SD = 13.35$) there was a .32 standard deviation decrease in IS or T4_A1C ($SD = 1.71$) equaling a decrease in IS by .55 points. The path from insulin pump status to IS produced a statistically and practically significant beta weight ($\beta = .295$) that was interpreted as large. The positive beta weight for the aforementioned pathway indicated that for every 1 standard deviation unit increase in insulin pump status, which based on the coding reflects not employing insulin pump therapy ($SD = .50$), there was a .30 standard deviation increase in IS or T4_A1C ($SD = 1.71$) equaling an increase in IS by .51 points. In short, participants with higher cognitive ability scores had lower initial starting values for metabolic control. Conversely, participants that were not on insulin pump therapy had higher initial starting
values for metabolic control. Moreover, the path from duration to LC produced a statistically and practically significant beta weight ($\beta = -0.224$) that was interpreted as moderate. The negative beta weight for the aforementioned pathway indicated a slower rate of metabolic deterioration for participants that had type 1 for a longer period of time.

**Discussion**

This study examined the role of poor metabolic control among adolescents diagnosed with type 1 diabetes as a limiting factor related to academic performance. The findings revealed that (a) higher initial levels of adolescent HbA1c resulted in lower final GPA scores, (b) positive linear growth of HbA1c values indicated the deterioration of metabolic control for youth with type 1 diabetes during adolescence, and (c) a positive covariance occurred between the IS and LC of HbA1c indicating a faster deterioration in metabolic control for adolescents with higher starting values. Namely, a statistically significant and meaningful inverse relation was observed between prior HbA1c levels and final GPA scores after 3 years for adolescents with type 1. While previous studies have demonstrated a significant correlation between mean scores of HbA1c and concurrent achievement (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009), only one other study has demonstrated a statistically significant prediction of future achievement based on prior HbA1c level from one time point (Hannon et al., 2012). Thus, early identification of high HbA1c may reveal a need for targeted early intervention concerning metabolic control to mitigate subsequent effects on academic success.

In regard to the second hypothesis, positive linear growth was observed during the sampling rounds that occurred every 6 months. The linear trend was similar to that of
previous studies identifying adolescence as a time where metabolic control deteriorates for youth with type 1 (Helgeson et al., 2010; King et al., 2012; Luyckx & Seiffge-Krenke, 2009; Miller et al., 2013; Rausch et al., 2012). For instance, Miller et al. (2012) reported adolescent HbA1c levels increased significantly at each sampling round by .24 units every 12 months. In the current study, the average rates of increase in HbA1c values was .14 units every 6-months, which appears comparable.

The third hypothesis was not supported as the positive linear growth of HbA1c did not appear to be a limiting factor of final GPA scores. In contrast to the current findings, Kent et al. (2010) reported that a deterioration of HbA1c over a 3-year period was a significant limiting factor in visual memory scores. Notably, Kent et al. employed a standardized assessment with high reliability (.90) to assess learning ability. Comparatively, while GPA scores at Time 6 were taken from the school record, GPA scores are not standardized. Consequently, GPA scores are not as reliable as scores from standardized achievement assessments. Although the reliability of GPA scores might be a possible confound, the first hypothesis was ultimately supported.

Alternatively, regarding the third hypothesis, increasing levels of HbA1c might not have demonstrated a linear effect on final GPA as HbA1c values might instead oscillate over time with GPA. If indeed specific periods of HbA1c levels impact specific periods of GPA scores, the oscillation between HbA1c and GPA across time would best be examined through a parallel process model. For instance, HbA1c levels might only be a limiting factor for GPA scores when they exceed the recommended HbA1c range for adolescents (< 7.5). Accordingly, a parallel process model was attempted in this study, but was not supported due to poor model fit (RMSEA > .1). Notwithstanding, future
studies might want to employ parallel process models to examine whether HbA1c levels and GPA scores do indeed oscillate through time. The aims of employing a parallel process model might include determining a) if specific periods of HbA1c levels demonstrate a greater impact on specific periods of GPA scores and b) the utility of such findings.

Evidence was provided to support the final hypothesis as a positive, non-statistically significant covariance was observed between initial status and linear change of HbA1c. A positive covariance suggested that higher initial HbA1c values led to faster increases in deterioration over time. Alternatively, this also meant that lower initial values of HbA1c resulted in slower rates of deterioration over a 3-year period. Two different latent class growth trajectories for adolescents with type 1 have been reported in the literature that would align with a positive covariance between initial status and linear change of HbA1c: (a) adolescents with poor metabolic control evidencing rapid deterioration of control and (b) adolescents with moderate or good metabolic control evidencing slow deterioration (Helgeson et al., 2010; King et al., 2012). The aforementioned findings cannot be compared to results from other studies as no previous studies examining adolescent trajectories of metabolic control have been identified that provide a covariance value (Helgeson et al., 2010; King et al., 2012; Luyckx & Seiffge-Krenke, 2009; Miller et al., 2013; Rausch et al., 2012). Future studies should examine whether academic outcomes differ for the two identified latent class trajectories. In regard to the current study, the covariance between initial status and linear change of HbA1c should be examined in future studies to determine if this finding can be replicated across different samples.
Amongst the covariates of final GPA, prior achievement was a meaningful and statistically significant predictor of future achievement as identified in previous studies (Byrnes & Miller, 2007; Volpe et al., 2006). Conversely, disease duration, sex, age, and cognitive ability were not statistically significant predictors of achievement after 3 years. In contrast to previous studies indicating longer disease duration resulted in poorer achievement or learning (Fox, Chen, & Holmes, 2003; Kovacs et al., 1992), the results of the current study align with studies (Kaufman et al, 1999; Kent et al., 2010) that indicated disease duration was not a statistically significant predictor of achievement or learning. Advancements in treatment modalities might be a possible explanation regarding the differences observed concerning factors such as disease duration, in turn, demonstrating a need for continual research (Naguib et al., 2009; Wodrich, Hasan, & Parent, 2011). Similarly, while previous studies identified sex (Fox et al, Kent et al) as a meaningful predictor of achievement or learning, results from the current study are comparable to prior studies that have determined sex was not a meaningful predictor of achievement (Hannon et al., 2012) or GPA (McCarthy et al., 2003). Unlike previous studies (Kent et al., DiPerna, Lei, & Reid, 2007), age was not identified as a meaningful predictor of achievement for this sample. Notably, Kent et al. and DiPerna et al. used outcomes measures that were standardized.

Although cognitive ability at Time 1 was not a meaningful predictor of final achievement, the positive pathway was in the expected direction. Previous studies (Hannon et al., 2012; Kovacs et al., 1992) identified cognitive ability score as a statistically significant predictor of achievement within this population. Nonetheless, cognitive ability scores from measures with adequate psychometric properties (i.e.,
reliability, validity; Canivez et al., 2005) are typically used to predict standardized achievement scores. As stated previously, GPA scores are not as reliable as standardized achievement assessments. Moreover, the cognitive assessment used during data collection (KBIT) was a brief rather than a comprehensive assessment of cognitive ability. Limitations concerning the use of GPA as an outcome measure are addressed further in the limitations section.

Previous research has indicated that cognitive scores from youth with type 1 typically fall within the average range (Gaudieri et al., 2008; Holmes et al., 1995; Kaufman et al., 1999). Similarly, mean cognitive scores for this sample of youth with type 1 fell within the average to high average range. Notably, mean IQ scores were derived from a brief measure of intelligence (KBIT) administered at the beginning of the current study. Notwithstanding, youth with diabetes are considered at-risk for cognitive impairments (e.g., visuospatial ability, reading, writing, motor speed, sustained attention) that might not be revealed through a composite score (Gaudieri et al; Naguib et al., 2009).

In regard to covariates of metabolic control, cognitive ability (IQ_KBIT) and insulin pump status were meaningful predictors of initial status. Higher levels of cognitive ability led to lower initial starting values of HbA1c. Other authors (Kaufman et al., 1999; McCarthy et al., 2002, 2003) have speculated that cognitive ability might influence metabolic control, but cognitive ability has only been controlled as a covariate in one other study (Hughes et al., Manuscript submitted for review) besides the present study. Additionally, those individuals not receiving insulin pump therapy had worse metabolic control (higher initial starting values of HbA1c), a finding that was expected based upon the literature (Hilliard et al, 2013; Rausch et al., 2012).
The only meaningful predictor of linear change of HbA1c was disease duration. Unexpectedly, participants that had type 1 for a longer period of time evidenced a slower rate of metabolic deterioration. Previous studies indicated that longer disease duration was associated with worse metabolic control (Hilliard et al., 2013). Social factors such as family support and a reasonable peer orientation (not sacrificing treatment adherence to fit in with peers), however, might have served as protective factors to slow the deterioration of metabolic control (Berg et al., 2011; Drew, Berg, & Wiebe, 2010; Palmer et al., 2011).

**Limitations.**

First, one limitation of the study was the use of GPA which is unstandardized. Although this may be the case, in practice, prior GPA has been identified as a predictor of high school grades (Byrnes & Miller, 2007) and college retention rates (Rohr, 2013). Additionally, high school GPA may be used by colleges and universities to assess class standing, scholarship eligibility, and entry into certain fields of study. While the correlation between GPA at Times 1 and 6 was moderate and statistically significant, reliability of this score might truly depend upon the school or teacher that assigned the score. For example, GPA might not be comparable across different schools or even from semester to semester due to factors such as different courses being offered or selected; instructor; or a student’s level of interest in the coursework. As such, the use of a brief reading or mathematics subtest from a standardized achievement assessment such as the Woodcock-Johnson Tests of Achievement might be a reasonable alternative for future studies. Likewise, the use of a brief standardized cognitive assessment at every sampling
round in future studies might provide more insight into the deleterious impact of metabolic deterioration on cognition and subsequent achievement.

Second, missing data in the analyses were another limitation that was necessarily addressed through FIML. While a complete data set is preferred, FIML was deemed sufficient to estimate parameters and to assess the fit of the model (Enders & Bandalos, 2001). In short, acceptable model fit as well as reasonable parameter estimates and standard errors generated through FIML suggested a tenable solution.

Third, from a demographic perspective, the sample was relatively homogenous limiting the generalizability of the results. The majority of participants were identified as non-Hispanic white from homes with mothers reporting some college education and annual household income \( \geq \$50,000 \). Future studies might want to determine whether the solution can be replicated in more diverse samples. Notably, Latino youth and families have been inadequately represented in studies of youth with type 1 (Miller et al., 2010).

Lastly, neither time of HbA1c assessment nor participants’ pubertal status were taken into consideration in this study. Time of assessment (i.e., period of the year) might have had an influence on HbA1c values represented in this study as children might have been less active during the winter months. As exercise is part of the ADA (2012b) recommended treatment plan, less physical activity related or unrelated to weather might have resulted in worse HbA1c values. Nevertheless, this relation was not addressed in this study and a hypothesis was not tested. Subsequently, no conclusions can be drawn from this proposition. Notwithstanding, future studies may want to consider taking time of HbA1c assessment into account. Additionally, evidence is present in the literature that suggests pubertal status may be a significant predictor of HbA1c trajectories (Rausch et
al., 2012); however, pubertal status was not included as a covariate in the analyses. Future studies might also want to consider including pubertal status as a covariate to examine HbA1c change over time.

**Clinical and School-Based Implications**

The results from this study indicate that the status of early HbA1c values might be more important in predicting future achievement than deteriorating metabolic control throughout adolescence. The importance of early HbA1c values in this study rather than deterioration over time might reflect the impact of poor metabolic control (i.e., chronic hyperglycemia) on cognition and learning during a critical period of brain developmental in childhood (before the age of 7; Biessels, Deary, & Ryan, 2008; Ryan, 2006) that might extend across the lifespan. Accordingly, a focus might need to be placed on early assistance and intervention in primary grades in concert with interventions throughout adolescence with the aim of maintaining recommended treatment goals for metabolic control for children with type 1. It is important to keep in mind that while linear change in HbA1c values did not predict future achievement, the deleterious effects of out-of-range blood glucose levels on health have been well documented in the literature (DCCT/EDIC, 2001). Researchers have recommended facilitating an intensive treatment regimen for adolescents to improve glycemic control in an effort to reduce the risk of developing future microvascular complications (DCCT/EDIC) and to enable students to perform to the best of his or her ability level in school (Holmes et al., 1999; Parent et al., 2009). Given that school-age youth spend a substantial portion of their day in an educational setting, educators, administrators, and health care professionals should work
toward facilitating the successful implementation of an intensive treatment regimen as defined by the American Diabetes Association (ADA; Silverstein et al., 2005).

The ADA (2012a) recommended that an applicable federal law (e.g., Section 504 of the Rehabilitation Act of 1973, Individuals with Disability Education Act [IDEA]) should be employed in the development of a written care plan including an emergency plan with the Local Education Agency (LEA) for all youth with diabetes. Federal laws are paramount in preventing discrimination based on disability and ensuring a free, appropriate public education in the least restrictive environment for youth with chronic health conditions such as type 1 (Jacob & Hartshorne, 2007). Written care plans identify goals and define measurable objectives for diabetes care in school including ensuring the youth’s safety, immediate and long-term health, optimal academic performance, and full participation in school related activities (NDEP, 2010). Additionally, personnel trainings, appropriate support staff (e.g., school nurse), and additional academic support could be included in a Section 504 Plan or an IEP under IDEA. Moreover, metabolic control improves when youth receive support at home and treatment regimens are followed rather than ignored in an attempt to fit in with peers (Berg et al., 2008, 2011; Drew et al., 2010; Palmer et al., 2011). With this in mind, educating teachers, administrators, classmates, and appropriate school staff concerning best practices of diabetes management might also help to improve the management of the disease in school and quality of life for youth with type 1 (Wagner et al., 2006).

**Role of the school psychologist.** Out of all of the related national organizations, only the NDEP (2010) provided a list of recommended actions or best practices for school psychologists regarding type 1. Recommendations include but are not limited to:
(a) understanding and promoting the use of federally applicable laws; (b) implementing the youth’s health care and educational plans; (c) attending trainings identified by the school nurse and principal; (d) being prepared to implement his or her role in emergency care plan; (e) communicating with school health team and parents and identify concerns related to student progress; (f) promoting a supportive learning environment; (g) being prepared to respond to emotional needs of the youth; and (h) practicing confidentiality and maintaining privacy related to health information (NDEP, 2010, pp. 91-92). Overall, school psychologists may provide the most assistance to youth with chronic health conditions primarily as advocates for student health (Kucera & Sullivan, 2011; Power, McGoe, Heathfield, & Blum, 1999; Schmitt, Wodrich, & Lazar, 2010). Further, Power et al. suggested that the role of school psychologists might include intervention, consultation, program development, training of educational professionals, and applied research in the context of assisting children with chronic health conditions. In general, school psychologists are ideal advocates for ensuring the correct and successful implementation of federally applicable laws in schools to safeguard the rights and well-being of children with chronic health conditions and disabilities.

**Conclusion**

The purpose of this study was to examine metabolic control (HbA1c) as a limiting factor for achievement of adolescents with type 1 diabetes. Previous studies used the mean value of HbA1c over the course of the respective studies or HbA1c values at single time points to predict achievement scores. Moreover, using mean scores across sampling rounds does not account for the individual differences in participants over time that latent growth modeling can assess. As such, future studies might want to examine HbA1c
levels at specific time points or through LGM rather than averaging HbA1c levels across an entire study to use as a single predictor of future achievement or cognitive functioning. Further, most studies examining achievement outcomes related to metabolic control for youth with type 1 have employed a cross-sectional (e.g., McCarthy et al., 2003) or case control design (e.g., Hannonen et al., 2012). Only one other study used a LGM design to assess the impact of metabolic control on learning over a 3-year period (Kent et al., 2010).

The current study employed a LGM to examine HbA1c values over 3 years to predict achievement. Results evidenced a strong and meaningful inverse relation between prior HbA1c values and final achievement (GPA). Conversely, while linear change of HbA1c values was indicative of deterioration in metabolic control over a 3-year period, the relation between linear change and final achievement was negligible. Thus, a focus on interventions targeting metabolic control beginning in the primary grades during critical periods of brain development (e.g., before the age of 7; Biessels, Deary, & Ryan, 2008; Ryan, 2006) and continuing throughout adolescence, such as a written care plan, might be warranted to prevent possible negative impacts on future learning and achievement. If earlier A1C values are meaningful predictors of future achievement, it might be advantageous to ensure that youth enter adolescence with A1C values within the recommended range (< 7.5) as metabolic control will deteriorate over time for all youth with type 1. Early intervention programs have even been recommended for children with type 1 beginning in preschool (Holmes et al., 1995). Appropriate interventions might include a written care plan as defined by NDEP (2010) developed under an applicable federal law (APA, 2012a).
Unfortunately, according to the literature, most parents are unaware of applicable federal laws related to diabetes care in school (Jacquez et al., 2008), nearly half of students with diabetes do not have written medical care plans (Jacquez et al.), and parental reports have indicated that most school personnel do not have adequate training in diabetes management (Wagner et al., 2006). Moreover, racial disparities have been reported in the literature regarding the presence of a written diabetes care plan, youths’ access to a school nurse, and parental awareness of applicable federal laws (Jacquez et al.). Youth from impoverished families and youth identified as Black are particularly at-risk (Jacquez et al.). With this in mind, the omission of even a single daily blood glucose test predicted an increase in HbA1c levels of 1.26% in a 2-year longitudinal study of adolescents (Rausch et al., 2012). To put this into context, the risk of microvascular complications including retinopathy, neuropathy, and nephropathy increases by approximately 40% for each single percentage point increase in HbA1c values (i.e., 1.0%; CDC, 2011).

In conclusion, previous studies that evidenced an inverse relation between HbA1c and achievement have accordingly recommended better metabolic control in school to optimize academic performance (Hannonen et al., 2012; Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009). Especially amongst at-risk populations, parent education regarding federally applicable laws to address the needs of students with type 1 is paramount. Further, continuing research related to the impact of metabolic control on cognition or achievement is recommended due to continual advancements in treatment modalities for type 1 (Naguib et al., 2009; Wodrich et al., 2011). Thus, further research examining the relation between metabolic control and achievement might convey the
importance of championing the development and implementation of a written diabetes care plan in school to prioritize metabolic control during the school day to maximize opportunities for learning and achievement.
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Table 1

**Full Information Maximum Likelihood (FIML) Univariate Correlations, Covariances, Means, and Standard Deviations for a Latent Growth Model of HbA1c (A1C) Values and Prediction of GPA over 3 Years for Adolescents with Type 1 Diabetes**

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. T1_A1C</td>
<td>–</td>
<td>1.65</td>
<td>1.67</td>
<td>1.27</td>
<td>1.13</td>
<td>1.07</td>
<td>-.42</td>
<td>-.33</td>
<td>-6.13</td>
<td>.78</td>
<td>.48</td>
<td>.40</td>
<td>-.03</td>
</tr>
<tr>
<td>2. T2_A1C</td>
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<td>–</td>
<td>1.46</td>
<td>1.35</td>
<td>1.12</td>
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<td>-.40</td>
<td>-.24</td>
<td>-5.14</td>
<td>.24</td>
<td>.34</td>
<td>.31</td>
<td>.03</td>
</tr>
<tr>
<td>3. T3_A1C</td>
<td>.64</td>
<td>.56</td>
<td>–</td>
<td>1.82</td>
<td>1.66</td>
<td>1.60</td>
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<td>-.16</td>
<td>-3.94</td>
<td>.58</td>
<td>.19</td>
<td>.01</td>
<td>.13</td>
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<tr>
<td>4. T4_A1C</td>
<td>.50</td>
<td>.53</td>
<td>.61</td>
<td>–</td>
<td>1.52</td>
<td>1.22</td>
<td>-.22</td>
<td>-.34</td>
<td>-5.53</td>
<td>.13</td>
<td>.17</td>
<td>-.02</td>
<td>.20</td>
</tr>
<tr>
<td>5. T5_A1C</td>
<td>.47</td>
<td>.46</td>
<td>.59</td>
<td>.54</td>
<td>–</td>
<td>1.71</td>
<td>-.30</td>
<td>-.29</td>
<td>-6.54</td>
<td>-2.22</td>
<td>.19</td>
<td>-.01</td>
<td>.16</td>
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<td>.58</td>
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<td>-4.06</td>
<td>-.34</td>
<td>.01</td>
<td>-.03</td>
<td>.12</td>
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<td>7. T1_GPA</td>
<td>-.46</td>
<td>-.43</td>
<td>-.17</td>
<td>-.21</td>
<td>-.30</td>
<td>-.20</td>
<td>–</td>
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<td>-.03</td>
<td>-.26</td>
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<td>8. T6_GPA</td>
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<td>-.28</td>
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<td>.47</td>
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<td>-.24</td>
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<td>.40</td>
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<td>-.43</td>
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<td>-.05</td>
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<td>10. Duration</td>
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<td>.11</td>
<td>.03</td>
<td>-.05</td>
<td>-.07</td>
<td>-.02</td>
<td>.13</td>
<td>-.01</td>
<td>–</td>
<td>.52</td>
<td>-.04</td>
<td>-.21</td>
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<td>.14</td>
<td>.07</td>
<td>.07</td>
<td>.08</td>
<td>.00</td>
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<td>-.10</td>
<td>.12</td>
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<td>-.04</td>
</tr>
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<td>.03</td>
<td>.02</td>
<td>-.02</td>
<td>-.01</td>
<td>-.03</td>
<td>.21</td>
<td>.11</td>
<td>-.05</td>
<td>-.03</td>
<td>-.09</td>
<td>–</td>
<td>-.02</td>
</tr>
<tr>
<td>13. No Pump</td>
<td>.23</td>
<td>.25</td>
<td>.15</td>
<td>.23</td>
<td>.19</td>
<td>.16</td>
<td>-.21</td>
<td>-.11</td>
<td>-.01</td>
<td>-.14</td>
<td>-.05</td>
<td>-.07</td>
<td>–</td>
</tr>
<tr>
<td>Mean&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.29</td>
<td>8.40</td>
<td>8.58</td>
<td>8.67</td>
<td>8.90</td>
<td>8.85</td>
<td>3.40</td>
<td>3.33</td>
<td>104.46</td>
<td>4.65</td>
<td>12.61</td>
<td>.55</td>
<td>.47</td>
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<tr>
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<td>8.45</td>
<td>8.59</td>
<td>8.73</td>
<td>8.88</td>
<td>9.02</td>
<td>3.41</td>
<td>2.33</td>
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<td>4.65</td>
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<td>1.49</td>
<td>1.50</td>
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<td>1.63</td>
<td>1.59</td>
<td>.62</td>
<td>.69</td>
<td>13.35</td>
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<td>-.05</td>
<td>.75</td>
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<td>-.20</td>
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<td>Kurtosis</td>
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<td>1.65</td>
<td>2.67</td>
<td>1.91</td>
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<td>-.13</td>
<td>-.51</td>
<td>-1.17</td>
<td>-1.98</td>
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</table>

*Note. n = 217. Mean<sup>a</sup> = Observed means. Mean<sup>b</sup> = Fitted means. Correlations and covariances are presented below and above the diagonal, respectively.*
### Table 2

**Full Information Maximum Likelihood (FIML) Parameter Estimates for a Latent Growth Model of HbA1c (A1C) Values and Prediction of GPA over 3 Years for Adolescents with Type 1 Diabetes**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unstandardized</th>
<th>SE</th>
<th>Standardized</th>
<th>Parameter</th>
<th>Unstandardized</th>
<th>SE</th>
<th>Standardized</th>
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<td><strong>Covariances</strong></td>
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<td></td>
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<tr>
<td>IS → T6_GPA</td>
<td>-.100</td>
<td>.033</td>
<td>-.178*</td>
<td>IS ↔ LC</td>
<td>.046</td>
<td>.027</td>
<td>.147</td>
</tr>
<tr>
<td>LC → T6_GPA</td>
<td>.218</td>
<td>.240</td>
<td>.075</td>
<td>T1_GPA ↔ IQ_KBIT</td>
<td>.018</td>
<td>.134</td>
<td>-.01</td>
</tr>
<tr>
<td>T1_GPA → T6_GPA</td>
<td>.615</td>
<td>.089</td>
<td>.540*</td>
<td>T1_GPA ↔ Duration</td>
<td>-.018</td>
<td>.070</td>
<td>-.30*</td>
</tr>
<tr>
<td>IQ_KBIT → T6_GPA</td>
<td>.007</td>
<td>.004</td>
<td>.079</td>
<td>T1_GPA ↔ Age</td>
<td>-.279</td>
<td>.070</td>
<td>-.08*</td>
</tr>
<tr>
<td>Duration → T6_GPA</td>
<td>.020</td>
<td>.016</td>
<td>.102</td>
<td>T1_GPA ↔ Sex</td>
<td>.067</td>
<td>.023</td>
<td>.22*</td>
</tr>
<tr>
<td>Age → T6_GPA</td>
<td>.029</td>
<td>.026</td>
<td>.089</td>
<td>T1_GPA ↔ No Pump</td>
<td>-.068</td>
<td>.023</td>
<td>-.22*</td>
</tr>
<tr>
<td>Sex → T6_GPA</td>
<td>-.014</td>
<td>.098</td>
<td>-.009</td>
<td>IQ_KBIT ↔ Duration</td>
<td>-.430</td>
<td>2.689</td>
<td>-.01</td>
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<tr>
<td>IQ_KBIT → IS</td>
<td>-.030</td>
<td>.006</td>
<td>-.321*</td>
<td>IQ_KBIT ↔ Age</td>
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<td>1.358</td>
<td>-.10</td>
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<tr>
<td>Age → IS</td>
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<td>.057</td>
<td>.092</td>
<td>IQ_KBIT ↔ Sex</td>
<td>-.313</td>
<td>.452</td>
<td>-.05</td>
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<tr>
<td>Sex → IS</td>
<td>.052</td>
<td>.171</td>
<td>.020</td>
<td>IQ_KBIT ↔ No Pump</td>
<td>-.051</td>
<td>.452</td>
<td>-.01</td>
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<tr>
<td>Duration → IS</td>
<td>.008</td>
<td>.029</td>
<td>.019</td>
<td>Duration ↔ Sex</td>
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<td>.171</td>
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<td>Sex → LC</td>
<td>.016</td>
<td>.041</td>
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<td>.051</td>
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<tr>
<td>Duration → LC</td>
<td>-.019</td>
<td>.007</td>
<td>-.224*</td>
<td>Error Covariances</td>
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<tr>
<td>No Pump → LC</td>
<td>-.012</td>
<td>.042</td>
<td>-.025</td>
<td>T1_A1C ↔ T6_A1C</td>
<td>.228</td>
<td>.046</td>
<td>.09*</td>
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</table>

*Note.* IS = Initial status; LC = Linear change; T1_GPA = GPA measured at Time 1; T6_GPA = GPA measured at Time 6; IQ_KBIT = IQ score measured at Time 1 using the Kaufman Brief Intelligence Test; T1_A1C – T6_A1C = HbA1c values measured at Times 1 – 6.

*p < .05.
Table 3

**Full Information Maximum Likelihood (FIML) Parameter Estimates for a Latent Growth Model of HbA1c (A1C) Values and Prediction of GPA over 3 Years for Adolescents with Type 1 Diabetes**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unstandardized</th>
<th>SE</th>
<th>Standardized</th>
<th>Parameter</th>
<th>Unstandardized</th>
<th>SE</th>
<th>Standardized</th>
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<td>IS</td>
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<td>.84</td>
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<tr>
<td>LC</td>
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<td>.011</td>
<td>1.00*</td>
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<td>T6_GPA</td>
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<td>1.00*</td>
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<td>.041</td>
<td>1.00*</td>
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<td>.73</td>
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<td>IQ_KBIT</td>
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<td>17.076</td>
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<td>Duration</td>
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<td>Age</td>
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<td>-.31</td>
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<tr>
<td>No Pump</td>
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<td>.024</td>
<td>1.00*</td>
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<td>.32*</td>
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<td>.165</td>
<td>.32*</td>
<td></td>
<td></td>
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<td>.32*</td>
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</table>

Note. IS = Initial status; LC = Linear change; T1_GPA = GPA measured at Time 1; T6_GPA = GPA measured at Time 6; IQ_KBIT = IQ score measured at Time 1 using the Kaufman Brief Intelligence Test; T1_A1C – T6_A1C = HbA1c values measured at Times 1 – 6.  
<sup>a</sup>Indicates the pathway was fixed.  
<sup>*p < .05.</sup>
Figure 1. Estimate Parameters for a Latent Growth Model of HbA1c (A1C) Values over 3 Years for Adolescents with Type 1 Diabetes. T1_A1C – T6_A1C = HbA1c values measured at Times 1 – 6.

*Indicates the pathway was fixed.

*Note. Standardized value provided for statistically significant error covariance. Unstandardized values provided for loading factors.
Figure 2. Estimated Parameters for a Latent Growth Model of HbA1c (A1C) Values and Prediction of GPA over 3 Years for Adolescents with Type 1 Diabetes. Solid pathways are significant at \( p < .05 \) (two-tailed). T1_GPA = GPA measured at Time 1; T6_GPA = GPA measured at Time 6; IQ_KBIT = IQ score measured at Time 1 using the Kaufman Brief Intelligence Test; T1_A1C – T6_A1C = HbA1c values measured at Times 1 – 6.

\( \text{\textsuperscript{a}} \)Indicates the pathway was fixed.

Note. Standardized values provided only for statistically significant pathways. Unstandardized values provided only for loading factors. Not all covariance pathways are represented in model.
Figure 3. Average Growth Curve for HbA1c (A1C) Values over 3 Years for Adolescents with Type 1 Diabetes. T1_A1C – T6_A1C = HbA1c values measured at Times 1 – 6.
### Appendix

#### List of Acronyms

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
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<tr>
<td>ADA</td>
<td>American Diabetes Association</td>
</tr>
<tr>
<td>ADA</td>
<td>Americans with Disabilities Act of 1990</td>
</tr>
<tr>
<td>AI</td>
<td>American Indian</td>
</tr>
<tr>
<td>API</td>
<td>Asian/Pacific Islander</td>
</tr>
<tr>
<td>CDC</td>
<td>Centers for Disease Control</td>
</tr>
<tr>
<td>CGM</td>
<td>Continuous glucose monitoring</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>CSII</td>
<td>Continuous Subcutaneous Insulin Infusion</td>
</tr>
<tr>
<td>DCCT</td>
<td>Diabetes Control and Complications Trial</td>
</tr>
<tr>
<td>DCCT/EDIC</td>
<td>The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study Research Group</td>
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<tr>
<td>DKA</td>
<td>Diabetic Ketoacidosis</td>
</tr>
<tr>
<td>DMMP</td>
<td>Diabetes Medical Management Plan</td>
</tr>
<tr>
<td>DPP</td>
<td>Diabetes Prevention Program</td>
</tr>
<tr>
<td>GLM</td>
<td>General Linear Model</td>
</tr>
<tr>
<td>GPA</td>
<td>Grade Point Average</td>
</tr>
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<td>HbA1c</td>
<td>Hemoglobin A1C</td>
</tr>
<tr>
<td>HLM</td>
<td>Hierarchical Linear Modeling</td>
</tr>
<tr>
<td>IEP</td>
<td>Individualized Education Program</td>
</tr>
<tr>
<td>IHP</td>
<td>Individualized Health Care Plan</td>
</tr>
<tr>
<td>ITBS</td>
<td>Iowa Tests of Basic Skills</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Full Form</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
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<tr>
<td>ITED</td>
<td>Iowa Tests of Educational Development</td>
</tr>
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<td>IDDM</td>
<td>Insulin-dependent Diabetes Mellitus</td>
</tr>
<tr>
<td>IDEA</td>
<td>Individuals with Disabilities Education Improvement Act</td>
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<tr>
<td>IQ</td>
<td>Intelligence Quotient</td>
</tr>
<tr>
<td>K-BIT</td>
<td>Kaufman Brief Intelligence Test</td>
</tr>
<tr>
<td>LEA</td>
<td>Local Education Agency</td>
</tr>
<tr>
<td>LGM</td>
<td>Latent Growth Model or Linear Growth Model</td>
</tr>
<tr>
<td>MDI</td>
<td>Multiple daily injections</td>
</tr>
<tr>
<td>MODY</td>
<td>Maturity Onset Diabetes in Youth</td>
</tr>
<tr>
<td>NDEP</td>
<td>National Diabetes Education Program</td>
</tr>
<tr>
<td>NHB</td>
<td>Non-Hispanic Black</td>
</tr>
<tr>
<td>NHW</td>
<td>Non-Hispanic White</td>
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<td>NIDDM</td>
<td>Non-insulin-dependent Diabetes Mellitus</td>
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<td>OCR</td>
<td>Office of Civil Rights</td>
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<td>PBS</td>
<td>Pediatric Behavior Scale</td>
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<tr>
<td>PDA</td>
<td>Personal Digital Assistant</td>
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<tr>
<td>PIQ</td>
<td>Performance Intelligence Quotient</td>
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<td>RCT</td>
<td>Randomized Control Trial</td>
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<td>SEARCH for Diabetes in Youth Study Group</td>
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<td>Verbal Intelligence Quotient</td>
</tr>
<tr>
<td>WISC-R</td>
<td>Weschler Intelligence Scale for Children – Revised</td>
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</table>
Joel B. Winnick – Curriculum Vitae

Personal Information
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2012 – 2013
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