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A Longitudinal Investigation of Cognitive Predictors of Self-care Behaviors in Youth with Type I Diabetes

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A Longitudinal Investigation of Cognitive Predictors of Diabetes Care Behaviors in Youth with Type 1 Diabetes

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

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Abstract

A LONGITUDINAL INVESTIGATION OF COGNITIVE PREDICTORS OF SELF-CARE BEHAVIORS IN YOUTH WITH TYPE I DIABETES

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

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Cross-sectional research of youth with type I diabetes has demonstrated that rote and working memory predict blood glucose monitoring (BGM) and carbohydrate consumption, respectively; however, to date, no longitudinal follow-up studies exist. Rote and working memory subtests from well-standardized memory measures, along with a problem-solving and executive functioning measures were administered to 118 youth with type I diabetes, aged 9-16 in two waves of data collection (mean interval = 2.07 years). Diabetes care behaviors were assessed through the 24-hour Diabetes Interview. This study was the first to document longitudinal prediction of BGM by rote memory and fat consumption by working memory. Extant cross-sectional literature was replicated; rote memory was concurrently associated with BGM and working memory with carbohydrate consumption. Memory was a stronger predictor of disease care behavior than disease responsibility or age. Problem solving and executive functioning failed to significantly predict disease care behaviors.

Introduction

Type I diabetes mellitus (T1D) is one of the most common pediatric chronic diseases in North America, affecting approximately 1 in 500-600 youth under the age of eighteen (Sperling, 1990). Disease onset for T1D peaks during middle childhood, but can be diagnosed as late as middle adulthood (Wysocki, Greco & Buckloh, 2003). T1D is the result of autoimmune destruction of the pancreatic islet cells, or beta cells, that produce the hormone insulin (Atkinson & MacLaren, 1990). Destruction of the beta cells ultimately results in permanent and irreversible inability to produce endogenous insulin. Insulin is necessary because it allows glucose to cross the cell membrane and be metabolized, or utilized by the body. Glucose is the body's main source of energy, it plays a crucial role in growth, and brain and organ functioning (Wysocki, Greco & Buckloh, 2003, pg. 304). Thus, youth with T1D need replacement of their endogenous insulin to survive.

Conventional treatment of T1D has traditionally consisted of one or two daily insulin injections. In the past fifteen years, intensive treatment of T1D, defined as three or more daily injections, or use of a constant subcutaneous insulin infusion (through a device known as a "pump") has greatly increased in popularity. This notable shift toward intensive insulin therapy is largely attributable to the 1993 publication of the DCCT which markedly demonstrated the long-term advantages of intensive treatment over the conventional treatment (DCCT, 1993).

Although the goal of insulin therapy is to approximate, as closely as possible, the body's physiological production of insulin, neither conventional nor intensive treatment exactly mimics an endogenous supply of insulin. Individuals with T1D must also engage in a variety of complex diabetes care behaviors (i.e. blood glucose testing, dietary regulations and restrictions, and exercise) to better approximate normal glucose metabolism. Despite the combination of insulin therapy and behavioral adaptation, approximation of natural insulin levels remains difficult and subsequently leads to imperfect blood glucose metabolism. Inconsistencies in blood glucose metabolism cause blood glucose levels to recurrently deviate from outside of the normal range (70-120 mg/dl). (Wysocki et al., 2003, pg. 304-305).

Hypoglycemia (low blood glucose levels) and hyperglycemia (high blood glucose levels) frequently occur in youth with T1D and must be identified and corrected. Hypoglycemia can be caused by too much insulin, excessive exercise, under-eating, and results in sweating, tremor, weakness, dizziness, nausea and confusion (Mooradian, 1988). Alternatively, hyperglycemia can be caused by too little insulin, over-eating, stress, illness and infection, and results in increased urination, dehydration, electrolyte imbalance and an increased heart rate (Mooradian, 1988).

Episodes of hyper- and hypoglycemia also impact the central nervous system. Given that the brain is unable to store its own glucose supply, it relies heavily on circulating blood glucose as a source of energy necessary to maintain metabolism. Thus, episodes of hyper- and hypoglycemia effect neurocognitive functioning. Animal studies have demonstrated that cerebral neurons endure some damage as quickly as four to five

minutes after onset of a hypoglycemic episode (Auer & Siesjo, 1988). Neurological damage appears to be limited to parts of the brain where large numbers of insulin receptors are found, such as the hippocampus (Auer & Siesjo, 1988). The hippocampus is the brain structure involved with memory and attention; as would be expected, recurrent and severe hypoglycemia appears to be related to memory and attention difficulties in youth with T1D (Desrocher & Rovet, 2004; Northam, Anderson, Werther, Warne & Andrewes, 1999; Northam et al., 2001).

Hyperglycemia has a variety of neurocognitive sequelae including slowed nerve conduction due to de-myelination, destruction of ganglion cells, and general neuronal loss (Desrocher & Rovet, 2004; Mooradian, 1988). Unlike the localized effects of hypoglycemia, the neuronal changes associated with hyperglycemia appear to be diffuse. A comprehensive review of the literature suggests that among children with T1D, memory and executive functioning difficulties are associated with frequent and severe episodes of hyperglycemia (Desrocher & Rovet, 2004; Northam et al., 1999; Northam et al., 2001).

The extant literature on memory and cognitive performance in children has indicated that neurocognitive correlates of T1D exist and that cognitive disruptions become increasingly significant over time (Desrocher & Rovet, 2004). Certain disease risk factors have been consistently associated with a higher incidence of neurocognitive problems. A review of the literature identifies four principal relations between risk factors and outcome: early age of onset and motor and visuospatial difficulties, severe and/or chronic hypoglycemia and attention and memory problems, hyperglycemia and

verbal and executive functioning dysfunction, and puberty and compromised executive functioning (Desrocher & Rovet, 2004). While the chronicity of these four primary risk factors suggests that these relations would best be addressed with longitudinal designs, the majority of studies investigating neurocognitive outcomes have been cross-sectional in nature. Overall, cross-sectional studies of memory and executive functioning have yielded inconsistent, ambiguous results. This is likely due to the discrepancies in measurement of various memory constructs, the complicated nature of memory, and the exploratory nature of many studies (Desrocher & Rovet, 2004).

Despite these methodological shortcomings, several reliable trends have been found. A meta-analytic review conducted by Greer and Holmes (1996) revealed that youth with T1D consistently demonstrated compromised verbal short-term memory as assessed by the Digit Span subtest of the WISC-R. A more recent literature review of neurocognitive sequelae (Desrocher & Rovet, 2004), demonstrated considerable evidence that youth with chronic hypoglycemia have a difficult time with attention to detail, specifically, with the selective, focused, and inhibition facets of attention. The review also noted the tendency for youth with chronic hypoglycemia to perform poorly on tasks of executive functioning, decision-making, planning, verbal memory, and to a lesser extent, spatial memory tasks.

A number of longitudinal studies have investigated memory and cognitive functioning over time in children and adolescents with T1D. (Fox, Holmes & Chen, 2003; Kovacs, Goldston & Iyengar, 1992; Kovacs, Ryan & Obrosky, 1994; McCarthy, Lindgren, Mengeling, Tsalikian & Engvall, 2002; Northam et al., 2001; Northam,

Anderson, Werther, Warne & Andrews, 1999; Rovet & Ehrlich, 1999; Rovet, Ehrlich & Czuchta, 1990). Disruptions appear to manifest themselves in compromised verbal cognitive skills rather than nonverbal or performance cognitive abilities (Kovacs et al., 1992; Northam et al., 2001; Rovet & Alvarez, 1997; Rovet & Ehrlich, 1999), although the latter has only been infrequently assessed, and the ages of study samples may preclude the detection of early onset or long duration nonverbal difficulties.

Longitudinal Cognitive Studies

Not surprisingly, at disease onset children and adolescents with T1D do not demonstrate any difficulties in general intelligence, verbal abilities, spatial abilities, memory or academic achievement (Rovet et al., 1990). Correspondingly, one year post-diagnosis, children and adolescents with T1D continue to perform similarly to non-diabetic controls on measures of verbal and nonverbal cognitive functioning (Kovacs et al., 1992; Rovet et al., 1990). However, several studies have noted that two-years post-diagnosis a failure to achieve developmentally normative verbal cognitive gains is found (Fox et al., 2003; Kovacs et al., 1991; Kovacs et al., 1994; Northam et al. 2001; Rovet & Ehrlich, 1999).

Kovacs, Goldston and Iyengar (1992) annually assessed the intellectual and academic functioning of 87 youth (mean age 11.1 years) from T1D onset to six years post-diagnosis and found that while long-term verbal memory abilities and school grades declined over time, nonverbal problem solving skills improved. However, these conclusions are based on only two tests (one verbal, one non verbal) from the Wechsler Intelligence Scale for Children-Revised (WISC-R). The Vocabulary subtest assessed

general verbal intelligence, academic progress, overall verbal skills, language development and long-term memory (Sattler, 2001). Additionally, the Block Design subtest provided a measure of nonverbal problem solving, perceptual organization, and abstract conceptualization (Sattler, 2001). Disease duration predicted a decline in the WISC Vocabulary subtest scores such that by six years post-diagnosis a decline of almost one full standard deviation was found. Contrary to the deterioration of verbal cognitive functioning, nonverbal problems solving skills appeared to improve over time; Block Design scaled scores increased by almost one standard deviation over the six-year interval. However, improvement may be spurious and due to the practice effects which are particularly prominent on the nonverbal subtests of the WISC that heavily rely on novelty. Alternatively, if this increase in Block Design scores test is indicative of improved problem solving skills, older youth might be better able to adapt to their treatment regimen. In adults, cognitive flexibility, as measured by problem solving, aided in aspects of the treatment regimen such as carbohydrate counting, and adjustment of insulin based on unexpected exercise or correcting for hyper- or hypoglycemia (Hill-Briggs, 2003). Kovacs et al.'s (1992) findings warrant replication and extension to accurately interpret the clinical significance of their noted increase in Block Design scores.

While Kovacs and colleagues followed their original longitudinal sample (Kovacs et al., 1992) another two years (Kovacs, Ryan & Obrosky, 1994), they neither re-assessed for further changes in Block Design scores, nor investigated non verbal problem solving skills via an alternate measure. Instead, Kovacs and colleagues followed fifty-seven

adolescents with T1D from their original 1992 study in an attempt to establish a relation between short and long-term verbal memory and verbal intellectual performance (Kovacs, Ryan & Obrosky, 1994). Youth (mean age at follow-up = 18.90 years; mean interval between study entry and memory testing = 7.95 years) performed slightly, but not significantly, poorer than controls on a test of verbal working memory. Interestingly, those adolescents who performed poorly on the short-term verbal working memory task also experienced the greatest deterioration of long-term verbal memory and verbal intellectual performance.

Rovet and Ehrlich's (1999) findings both support and extend the work of Kovacs and associates. The verbal and memory skills of children and adolescents with T1D (mean age = 12.1 years) were tracked from disease onset to seven years post-diagnosis. Again, the Vocabulary subtest of the WISC was used as a measure of overall verbal skills. Additionally, the Digit Span subtest of the WISC was administered to assess short-term sequential auditory memory, attention and working memory (Sattler, 2001). Adolescents' scores on both the Vocabulary and Digit Span subtests significantly declined between three and seven years post-diagnosis and support previous findings that both long-term verbal and working memory do not improve with age as expected during adolescence. Compromised working memory is particularly worrisome given the role that working memory likely plays in self-care. Innovative research has shown that working memory plays an important role in diabetes behaviors such as calculating carbohydrate calories consumed (Souter, Chen, Streisand, Kaplowitz & Holmes, 2004).

In corroboration with Rovet and Ehrlich's findings, Northam et al. (2001) assessed long-term memory of 90 children and adolescents (9-17 years) from disease onset through six years post-diagnosis compared to the memory scores of controls. Northam et al. (2001) also assessed executive functioning, an understudied ability that is probably crucial to an intensive diabetes regimen, and found diminished executive functioning on the Complex Figure Test (CFT), a design-copying task that measures cognitive flexibility, planning and organization of visual information (Lezak, Howeison & Loring, 2004). This finding is particularly interesting given that executive functioning was tested in the visual modality; thus far, most investigations of neurocognitive sequelae of T1D have focused on verbal and nonverbal cognitive skills. Despite the differences found in long-term memory and executive functioning, participants with diabetes did not perform differently on nonverbal problem solving tasks compared to controls. Youth with T1D obtained similar WISC Performance Intelligence Quotient (PIQ) scores as controls; the PIQ represents fluid reasoning, or the ability to manipulate, plan, or solve novel problems (Sattler, 2002). Finally, both groups performed equivalently on the Wide Range Assessment of Memory and Learning (WRAML) Story Memory task, a measure of verbal rote memory. This finding is potentially important given that rote memory, similar to working memory is hypothesized to be a beneficial skill for treatment adherence; rote memory is involved in frequency of blood glucose testing (Souter et al., 2004).

Taken together, these longitudinal cognitive studies suggest that while rote memory appears variable affected over time, higher-level organizational skills and

strategies, such as executive functioning and working memory demonstrate more consistent deterioration. Similar to the conclusion regarding rote memory, the status of nonverbal conceptual abilities over time is also equivocal. While one study has indicated that nonverbal conceptual abilities, thought to reflect problem solving, improved over time, this finding is likely due to practice effects (Kovacs et al., 1992). Yet, another study suggests that problem solving skills are resistant to the neurocognitive sequelae associated with diabetes (Northam et al., 2001), consistent with the general belief that diabetes affects verbal cognitive skills rather than nonverbal performance skills (Kovacs et al., 1992; Northam et al., 2001; Rovet & Alvarez, 1997; Rovet & Ehrlich, 1999).

Diabetes Care Behaviors

The overarching goal of the diabetes treatment regimen is to maintain near normal blood glucose levels, and in doing so, to minimize the risk of long-term disease-related complications such as nephropathy (kidney damage), retinopathy (eye damage that may result in blindness) and neuropathy (nerve damage especially in appendages such as fingers, toes). Disease management in youth with T1D is traditionally measured both physiologically, through glycoslyated hemoglobin (HbA1c) assays, and behaviorally, though adherence, or, diabetes care behavior measures. Glycoslyated hemoglobin assays (HbA1c) represent an individual's average level of metabolic control by measuring the percentage of glucose molecules bound to red blood cells over the past six to eight weeks (Blanc, Barnett, Gleason, Dunn, & Soeldener, 1981). Traditionally, higher HbA1c levels indicate poorer metabolic control and are associated with a higher risk of long-term diabetes complications (DCCT, 1993).

Diabetes care behaviors can be "viewed as: 1) categorical versus continuous and 2) unitary versus multidimensional" (La Greca & Bearman, 2003, pg. 120). Logically, the wide variety of dissimilar behaviors associated with the treatment regimen lends support to a multidimensional approach to this construct (i.e. insulin injections, insulin injecting timing, eating frequency, diet type, exercise type and frequency, blood glucose testing frequency and recording). For example, a youth's blood glucose testing conceivably has little, if any, correlation to exercise type and frequency; a youth who tests blood sugar regularly may rarely exercise. In fact, empirical evidence supports a multidimensional approach to the study of diabetes care behaviors (Freund, Johnson, Silverstein, & Thomas, 1991; Johnson et al., 1986; Johnson et al.,1992). Thirteen independent diabetes regimen behaviors have been identified, which factor analyze into five distinct factors (Johnson et al., 1986). The five factors are: Injection Factor (regularity, interval, meal timing, and regularity of meal timing), Exercise Factor (frequency, duration, and type), Diet Type Factor (percentage of calories from fats and percentage of calories from carbohydrates), Frequency Factor (blood glucose testing, frequency of meals/snacks), and Diet Amount Factor (calories consumed and concentrated sweets).

The 24-hour diabetes interview, a multidimensional assessment of behavior, is arguably the most empirically validated and well-respected diabetes care measure. Johnson et al. (1986) adapted a 24-hour diabetes interview method from a similar nutrition measure to evaluate diabetes care behaviors. The 24-hour diabetes interview consists of an individual's report of their behavior over the previous 24-hours in temporal order from awakening through bed time. Selective prompts from the interviewer query

for the presence or absence of a behavior and the circumstances that surround it, if a behavior is not spontaneously mentioned.

Beyond the multidimensional nature of self-care, the interview has several noteworthy strengths. The report of actual, specific behaviors from the previous 24 hours reduces problems associated with self-report questionnaires that typically estimate vague, global behavior over long intervals of time (i.e. one week to three months). Interviewees are asked to only report their diabetes-related behaviors for the previous 24 hours; review of such a short period of time tends to reduce over- or under-reporting. Interviewers set a non-judgmental tone and both youth and one parent are interviewed separately to ensure multi-informant data and to provide corroboration.

Twelve of the thirteen diabetes care behaviors from the 24-hour diabetes interview have significant stability over three months, but some diabetes care behaviors display more consistency than others (Freund et al., 1991). Blood glucose testing (reliability coefficients range from .72-.76), dietary behaviors (coefficients ranging from .50-.77), and injection-meal timing (coefficients ranging from .58-.71) exhibited the greatest stability over three months. Less stable were injection regularity (coefficients ranging from .06-.35), injection interval (coefficients ranging from .38-.49) and exercise type (coefficients ranging from .37-.48), which had the lowest reliabilities.

Johnson et al. (1992) investigated stability of diabetes care behaviors in a more powerful, two-year longitudinal study. Despite the fact that all diabetes care behaviors declined as youths matured, injection-meal timing, dietary behaviors (calories consumed, concentrated sweets consumed, percentage carbohydrates and fats consumed) and the

Frequency factor (blood glucose testing and eating frequency) demonstrated significant stability across time. Interestingly, injection regularity and exercise frequency and type, two diabetes care behaviors found to be less stable in Freund et al.'s (1991) initial study of diabetes care reliability, displayed significant stability across almost two years.

The specificity and stability of the Frequency factor, dietary behaviors, and exercise frequency, over a two-year period, provide empirical support for their use as outcome variables for cognitive predictors. Given the complexity of the diabetes treatment regimen, it seems logical that rote memory, working memory, problem solving and executive skills could be instrumental in the prediction of diabetes care behaviors.

Developmental Risk Factors to Diabetes Care Behavior and Metabolic Control

The relations between cognitive skills and diabetes care behaviors in youth are complicated by developmental changes. Cognitive predictors are only likely to predict diabetes care behaviors to the extent that youth are old enough and/or have primary responsibility for diabetes care behavior. As youth with T1D age and transition from preadolescence to adolescence (between 12- and 14-years of age) performance of diabetes care skills associated with treatment markedly deteriorates along with metabolic control (Hanson, Henggeler, & Burghen, 1987a; Jacobson et al., 1987; Johnson et al., 1986; Johnson et al., 1992; La Greca, Follansbee & Skyler, 1990).

As youth move towards adolescence, disease management responsibility typically transfers from primary caretakers to youth; this transition adversely affects both treatment behaviors and metabolic control (Anderson, Auslander, Jung, Miller, & Santiago, 1990; Ingersoll, Orr, Herrold, & Golden, 1986; La Greca et al., 1990; Wysocki, Meinhold, Cox

& Clarke, 1990; Wysocki et al., 1992). When disease responsibility is ambiguously transferred, transferred prematurely to preadolescents, or transferred to cognitively immature youth, treatment care and metabolic control are both compromised (Anderson et al., 1990; Ingersoll et al., 1986; La Greca et al., 1990). Puberty and its biological sequelae also contribute to decreased diabetes care behaviors and metabolic control; the onset of puberty is frequently accompanied by a decrease in insulin sensitivity and an increase in triglycerides in both adolescents with and without T1D (Amiel, Sherwin, Simonson, Lauritano & Tamborland, 1986; Cruikshanks, Orchard & Becker, 1985). Decreased insulin sensitivity and alteration in lipid metabolism result in unstable metabolic control (Bloch, Clemmons, & Sperling, 1987; Amiel et al., 1986).

Age. Numerous developmental transitions associated with adolescence have been hypothesized to contribute to the decline in diabetes care behavior (La Greca & Bearman, 2003, pg. 124), specifically: strivings from autonomy, self-identity, social competence, parental support (Hanson et al., 1987a), peer pressure, conformity, increasing independence (Hanson, Henggeler & Burghen, 1987b; Jacobson et al., 1987), and feelings of invulnerability and invincibility (Paikoff & Brooks-Gunn, 1991 as cited by Seiffege-Krenke, 1998; Silverstein, 2005). The variety of processes hypothesized to contribute to the deterioration of diabetes care behaviors in adolescence underscores the magnitude of this phenomenon, and highlights the importance of the inclusion of age in studies of self-care.

Johnson and colleagues (1986) obtained parent and adolescent self-reports via the 24-hour interview and assessed age effects on five diabetes care dimensions of behavior;

exercise, injections, diet type, testing/eating frequency, and diet amount. Age effects were found for four of the five diabetes care behaviors; preadolescents (6- to 12-years old) and younger adolescents (13- to 15-years old) were found to exercise, test blood glucose levels, and eat more frequently than the older adolescents (16-to 19-years old). Similarly, preadolescents were significantly more adherent in both their injection behaviors and their recommended diet than either younger (13- to 15-years old) or older adolescents (16- to 19- years old). The 24-hour interview also has been used longitudinally to assess the effect of youths' age on diabetes care behaviors and metabolic control (Johnson et al., 1992). Using structural equation modeling, age was a significant predictor of 5 of 6 constructs (injection, exercise, diet type, testing/eating frequency, calories consumed and concentrated sweets) such that increasing age was related to increasingly poorer diabetes care behavior.

A study conducted by Jacobson et al. (1987) corroborated this finding with a different methodology. Jacobson et al. (1987) assessed treatment compliance with interviews of youth conducted by health care providers during medical appointments; these interviews produced a unitary compliance score. Although the care providers were explicitly instructed to only use interview data in their calculation of compliance scores, the providers had access to youth's previous HbA1c scores. It is important to note that due to this study's compliance measure and methodology, youth's compliance scores were probably confounded by their medical history of metabolic control.

Preadolescents (aged 9-12) obtained higher overall compliance scores than adolescents (13-15). Specifically, preadolescents were more compliant to a prescribed

diet and monitored their blood glucose more frequently than adolescents. However, both age groups demonstrated similar adherence to insulin usage. This pattern of compliance behavior suggests that older adolescents may be less likely to perform diabetes care behaviors that are salient in social settings. A restrained diet and frequent blood glucose testing are typically socially conspicuous, whereas insulin can be injected in a private location. Given adolescents' desire for peer approval, they may be less compliant regarding diabetes care behaviors perceived as impediments to social acceptance. Interestingly, this study found that youth with higher self-reported self-esteem and parent-reported social functioning adhered more strongly to their diabetes regimen. It is possible that the struggle with self-esteem and desire for social acceptance characteristic of adolescence may be potential mediators or moderators of the decline in diabetes care behaviors noted in adolescence.

Hanson et al. (1987a) extended the findings of Jacobson et al. (1987) and assessed psychosocial mediators of the relation between age and diabetes care behaviors. Congruent with previous literature, Hanson et al. (1987a) found a negative correlation between age and a unitary adherence score that consisted of both a combined measure of youths' self-report and parental observation. Additionally, age was negatively correlated with youth report of parental support. Multiple regression analyses revealed age had an indirect effect on adherence though its relation with parental support. Age mediated the relation between parental support and adherence such that preadolescents received more parental support than adolescents and concurrently, preadolescents demonstrated greater treatment adherence.

Hanson et al. (1987a) hypothesized that these findings might reflect older youths' experimentation with individuation from their parents and desire to handle both disease-specific and general life stressors independently. Although this study did not directly measure diabetes treatment responsibility, it is likely that the transfer of responsibility, a developmental process strongly associated with parental support, influenced Hanson et al.'s (1987a) findings. Disease responsibility among preadolescents with high parental support is likely placed on the primary caretaker. Once transfer of disease responsibility occurs, then the negative correlation between age and diabetes care behavior is found.

Diabetes Responsibility. Implicit in the association between older youth age and poorer diabetes care is the assumption that older youth have increasing self-care responsibility. However, documentation of this association is important, particularly for different diabetes care behaviors and because different families transfer responsibility for each aspect of diabetes care at different ages.

Wysocki and associates conducted two studies to evaluate the ages at which professionals and parents report adolescents are capable and responsible for different aspects of their self-care. Diabetes health care professionals reported that by age fourteen, 50% of "typical" children and adolescence with T1D had mastered all self-care skills necessary for responsibility of their treatment regimen (Wysocki et al., 1990). This study divided Johnson et al.'s (1986) 13 aforementioned, well-respected diabetes care behaviors into 38 component subskills (i.e. shakes insulin in bottle, correctly draws insulin in a syringe). In the follow-up study, parents of adolescents with T1D estimated that by age 12, their child had mastered all self-care skills (Wysocki et al., 1992). Despite

the fact that the median age provided by parents was approximately two years younger than the median age of self-care skill mastery reported by professionals, the parent's sequence of skills mastered closely followed the sequence of skill mastery provided by the health care professionals. Although this study provides information about approximate ages of responsibility transfer, several methodological shortcomings exist that warrant caution in the interpretation of results. Health care professionals in the first study were asked to estimate the age that 50% of "typical" children mastered self-care skills and this average does not capture all children. Parents in the second study were instructed to answer questions about skill mastery only as it pertained to their child. These responses may reflect reality for their children but may not be optimal ages for transfer of responsibility to occur, and of course, these scores also are averaged and do not reflect the responsibilities of an individual child. Nevertheless, there was consensus in the sequence of self-care acquisition between parents and professionals.

Other research has revealed correlations between age and responsibility, such that as age increases, disease responsibilities increase (Anderson et al., 1990; Ingersoll et al., 1986). Ingersoll et al. (1986) noted that after controlling for disease duration, youth age was negatively correlated with parental participation in insulin adjustment; as youth matured, parents' self-reported adjustment of their offsprings' insulin decreased. This finding highlights the significant impact of age on responsibility, independent of disease duration. Similarly, Anderson et al. (1990) found that youth age was positively correlated with the youth responsibility as assessed by the Diabetes Family Responsibility Questionnaire (DFRQ; a self-report of both youth and parental disease responsibility).

Anderson et al. (1990) also found that both disease duration and age significantly predicted disease responsibility (total DFRQ scores). Together, these results suggest that future studies of diabetes care behavior would benefit from analyses of both age and responsibility as the inclusion of both variables would likely provide a more comprehensive, thorough study.

Further analyses by Ingersoll et al. (1986) suggested that cognitive maturity, or certain youth cognitive skills, influenced transfer of disease responsibility. Among adolescents who had not yet mastered abstract thinking, responsibility for insulin injection (i.e. diabetes responsibility) did not always lie with the adolescents, despite the fact that mothers ceased their participation in their adolescents' insulin administration. This suggests ambiguous responsibility transfer may contribute to deterioration of diabetes care behaviors in youth, in that it is unclear who is responsible for an aspect of disease management.

Anderson et al. (1990) investigated discrepancies in parent and child reports of responsibility; dyads in which neither the parent nor the child endorsed responsibility for a diabetes care behavior were considered failures in responsibility transfer. Not surprisingly, discrepant reports of responsibility were found to be predictive of poorer metabolic control.

La Greca, Follansbee & Skyler (1990) also found that adolescents (ages 12-17, mean age = 13.00), compared to preadolescents (ages 7-11, mean age = 9.50), assumed more self-care responsibility, yet were less adherent to certain aspects of the treatment regimen. More specifically, adolescents were less likely than preadolescents to follow a

proper diet or to carry proper snacks to treat episodes of hypoglycemia. Preadolescents with treatment responsibility for blood glucose testing and measuring insulin were in poorer metabolic control than preadolescents whose families remained engaged in their treatment regimen. La Greca et al. (1990) found that while older youth assumed more disease responsibility, the relation between responsibility and metabolic control was not completely mediated by age; after age, disease duration, and treatment adherence were controlled, youth who assumed more responsibility for eating on time and charting their blood glucose results were in poorer metabolic control. These findings again emphasize that future studies should include both age and responsibility as variables, however, La Greca et al. (1990) advocates that the two variables should be included and analyzed separately.

Thus, it appears that for many adolescents, more disease responsibility comes at the cost of better diabetes care behavior and metabolic control. Interestingly, qualitative interviews have shown that both parents and adolescents alike perceive the "burden of responsibility" of treatment adherence to be an obstacle in adolescent disease related self-management (Hanna & Guthrie, 2000a; Hanna & Guthrie, 2000b).

Cross-sectional Cognitive Predictors of Diabetes Care Behaviors

Memory. Several studies reviewed earlier (Kovacs et al., 1992; Northam et al., 2001) have tried to find a relation between memory and metabolic control, yet were unable to establish a clear relation between the two variables. Only one study has investigated the next step; the relation between memory and diabetes care behaviors (Souter et al., 2004).

Souter et al. (2004) revealed that verbal rote and quantitative working memory did in fact predict diabetes specific diabetes care behaviors in 224 youths with TID (range 9-17; mean age = 12.9). Rote verbal memory, as measured by the Verbal Memory Index of the Wide Range Assessment of Memory and Learning (WRAML), significantly predicted blood glucose monitoring, but only for adolescents. Youth were dichotomously split into adolescents (youth aged > 12.5 years) and preadolescents (younger than 12.5 years of age). Among adolescents, better memory predicted more blood glucose as assessed with the 24-hour diabetes interview methodology. Quantitative verbal working memory, as measured by the Arithmetic subtest of the WISC, significantly predicted favorable levels of carbohydrates, such that those youths with better verbal working memory consumed a higher percentage of calories from carbohydrates. Again, working memory only predicted carbohydrate ingestion for older adolescents (youth aged > 14.8 years). Among older adolescents working memory alone predicted almost 10% of the variance in calories consumed from carbohydrates. In order to rule out that memory served as a proxy variable for underlying intellectual ability in the prediction of diabetes care behaviors, general conceptual intelligence (measured by the Similarities WISC subtest) also was evaluated as a predictor of blood glucose testing and carbohydrate ingestion. Conceptual ability did not predict blood glucose testing frequency or calories consumed from carbohydrates beyond the effect of memory. Finally, blood glucose testing frequency was found to be a significant predictor of variability in metabolic control. This relation again was moderated by age; for preadolescents only, more daily blood glucose tests predicted less variable metabolic control. The moderating role of age

Peterson & Goldstein, 1997). In fact, a more detailed analysis of the results of these studies reveals a noteworthy trend. It appears that while problem solving skills are positively correlated with the performance of discrete, specific diabetes care behaviors, they are not associated with global measures of self-care.

Thomas et al. (1997) investigated the relation between diet- and glucose testing-specific problem solving and a global measure of diabetes compliance. This study failed to reveal a relation between problem solving and a global diabetes compliance measure. In contrast, Cook et al. (2001), Johnson et al. (1982), and McCaul et al. (1987), assessed for the relation between problem solving and the performance of specific diabetes care behaviors; each detected a significant positive association between problem solving and self-care. Specifically, positive relations were found between problem solving and the following diabetes care behaviors; urine testing (Johnson et al., 1982), insulin injection (Johnson et al., 1982; McCaul et al., 1987), and blood glucose testing (McCaul et al., 1987). These three diabetes care behaviors all involve performance of a behavior with a particular frequency. A more recent study directly investigated the relation between problem solving skills and diabetes care behavior frequency and noted a significantly positive correlation (Cook et al., 2001).

In addition to the relation between problem solving and diabetes care behaviors, several descriptive studies have researched the association between problem solving and metabolic control, as assessed by HbA1c assay. Thus far, one study has revealed a significant positive association between problem solving and HbA1c (Cook et al., 2001) while two have failed to discover a relation between problem solving and metabolic

control, as assessed by HbA1c (Auslander, Haire-Joshu, Rogge & Santiago, 1991; Thomas et al., 1997).

As noted by Hill-Briggs, the relation between problem solving and diabetes care and metabolic control in youth with T1D has also been investigated in a number of intervention studies. Two intervention studies reviewed by Hill-Briggs (2003) detected improvements in problem-solving skills, however, these gains were associated with either no improvement in HbA1c levels, or elevated HbA1c levels (Lucey & Wing, 1985 as cited by Hill-Briggs, 2003; Kaplan, Chadwik, & Schmmel, 1985 as cited by Hill-Briggs, 2003).

More recent problem solving intervention studies aimed at improving diabetes care behaviors have generated ambiguous results. Schundt et al.'s (1999 as cited by Hill-Briggs, 2003) problem solving intervention demonstrated an improvement in problem solving skills, however, this improvement was not associated with improvement in diabetes care behaviors. In contrast, Silverman et al. (2003) used a multiple baseline design to reveal significant diabetes care improvements in six youth who received a cognitive behavioral problem solving intervention. Five of the six youths experienced improvement on at least one diabetes care behavior. These results should be interpreted carefully as the study was comprised of only six participants and failed to include an attention control group. Despite these methodological shortcomings, this study extended greatly upon previous problem solving intervention literature; Silverman et al. (2003) measured diabetes care behavioral outcome with the reliable 24-hour diabetes interview.

This study's use of the 24-hour diabetes interview may set precedence in this area of research and encourage the use of well-known, reliable outcome measures.

The relation between improved problem solving skills and improved diabetes care behaviors found by both descriptive and intervention studies (Hill-Briggs, 2003; Silverman et al., 2003), warrants a more comprehensive study because of its potential importance. Additionally, given the involvement of the prefrontal cortex in both problem solving and memory (Lezak, pgs.80-82), the two are the likely positively correlated. This probable correlation underscores the inclusion of both cognitive skills in the same study to see if each provides unique contribution.

Executive Functioning. Thus far, executive functioning has not yet been investigated as a cognitive skill that might influence diabetes care behaviors. Executive functioning is considered a cognitive pre-requisite for "independent, constructively self-serving, and productive" thought and functioning (Lezak, pg. 35). Furthermore, comprised executive functioning has been shown to affect "strategies to approaching, planning, or carrying out cognitive tasks, or in defective monitoring of performance" (Lezak, pg. 35). Executive functioning is therefore logically implicated in the deployment of memory and problem solving ability for tasks and may supercede each. The inclusion of higher-level cognitive skills such as executive functioning and problem solving, in addition to relatively more rudimentary skills such as verbal working and rote memory, would provide a more comprehensive description of cognitive functioning required for optimal diabetes care behaviors.

Statement of the Problem

Recent research indicates that verbal rote and working memory predict diabetes-specific diabetes care behaviors (Souter et al., 2004). However, given that these same cognitive skills may be compromised by recurrent episodes of hyper- and hypoglycemia (Desrocher & Rovet, 2004), their relations to diabetes care behaviors are likely to be fluid over time and dynamic. Ideally, the ability of cognitive skills to predict diabetes management should be investigated longitudinally so that the literature may determine if, given their stability, cognitive abilities are able to predict diabetes management over time.

A longitudinal investigation of cognitive predictors of diabetes care behavior is proposed. Specifically, rote verbal memory, quantitative verbal memory, problem solving and executive functioning will be examined to replicate and to extend this newly emerging research area. Importantly, specific diabetes care behaviors from the 24-hour diabetes interview have demonstrated sufficient stability and validation across time to serve as appropriate outcome variables (Freund et al., 1991; Johnson et al., 1986; Johnson et al., 1991).

In contrast, verbal rote memory ability has been variously shown to remain stable over time (Northam et al. 2001), to plateau, or to decline (Fox et al., 2003; Kovacs et al., 1991; Kovacs et al., 1994; Northam et al. 2001; Rovet & Ehrlich 1999). Given that verbal rote memory has been found to predict blood glucose testing in adolescents, the stability of this cognitive skill and its longitudinal predictive capacity warrants longitudinal investigation.

Similarly, quantitative verbal working memory has been shown to decline slightly over a two-year interval (Rovet & Ehrlich, 1999) and to predict percentage of carbohydrates consumed by adolescents (Souter et al., 2004). Thus, given quantitative verbal working memory's dynamic nature over time, the stability and predictive capacity of this memory will be assessed.

General, nonverbal problem solving skills and perceptual organization have equivocally been found to both increase (Kovacs et al., 1992) and remain constant (Northam et al., 2001) over time in youth with T1D. The stability of diabetes-specific problem solving skills over time is largely unknown. Several cross-sectional studies (Cook et al., 2001; Johnson et al., 1982; McCaul et al.,1987) have found diabetes specific problem solving skills to be positively associated with a number of specific diabetes care behaviors and the frequency of diabetes care behaviors. Additionally, a recent pilot intervention study demonstrated an improvement in diabetes care behavior associated with diabetes specific problem solving training (Silverman et al., 2003). The inconclusive understanding of general problem solving skills, combined with the probable relation between diabetes specific problem solving and diabetes care behaviors, suggests that a predictive, longitudinal study will provide a more complete understanding of the nature of this set of cognitive skills.

Finally, executive functioning has been found to decrease over time in youth T1D (Northam et al., 2001). Executive functioning, defined as the "the ability to respond in an adaptive manner to novel situations" (Lezak, pg. 611) would likely play a large role in performing complex diabetes care behaviors. However, the role of executive functioning

as a predictor of diabetes care behaviors is yet unknown. A longitudinal investigation of executive functioning as a predictor of complex diabetes care behaviors will serve to extend the extant literature regarding cognitive predictors of diabetes care behaviors.

In addition to the neurocognitive sequelae of hyper-and hypoglycemia, youth with T1D also experience developmental processes that can negatively affect diabetes care behavior. The age transition between preadolescence to adolescence is associated with more diabetes care responsibility (Anderson et al., 1990; Ingersoll et al., 1986; La Greca et al., 1990; Wysocki et al., 1990; Wysocki et al., 1992), with poorer diabetes care behaviors (Hanson et al., 1987; Jacobson et al., 1987; Johnson et al., 1986; Johnson et al., 1992; La Greca et al., 1990) and with poorer metabolic control (Amiel et al., 1986; Anderson, 1990; Bloch, Clemmons, & Sperling, 1987; La Greca et al., 1990). Youth responsibility will be entered as a control variable, and age above and below 12 years will be examined as a moderator of the predictive effects of cognitive skills on diabetes care behaviors.

Hypotheses:

The following relations will be moderated by age after disease responsibility is entered as a covariate, such that, after 12 years of age memory, problem solving and executive functioning will significantly predict diabetes care behaviors. Additionally, these relations will change for younger children who age and mature from preadolescence to adolescence.

1) Rote memory at time 1 will predict blood glucose frequency and eating frequency at time 2 and consequently metabolic control.

2) Quantitative verbal working memory at time 1 will predict percentage of calories from fat and carbohydrates at time 2, and it ultimately may predict metabolic control.

Exploratory Hypotheses:

3) Problem Solving likely correlates positively with memory (Lezak, pgs.80-82) and thus, problem solving (at time 1) will predict the same diabetes care behaviors (at time 2) as working memory. As a more complex cognitive skill, problem solving, at time 1, will also predict complicated diabetes care behaviors such as total calories consumed, and exercise frequency at time 2.
4) Executive functioning (at time 1), as a cognitive skill that supersedes all others will also predict the same diabetes care behaviors (at time 2) as the other cognitive predictors.

Method

Selection of Participants

Participants were recruited through several pediatric endocrinology clinics in two metropolitan areas. At time of initial recruitment, youth with T1D and their primary caregivers were mailed a letter to explain the study prior to their medical appointment. Prospective participants later received more information via a telephone call.

Participants were assessed annually; typically assessment occurred within two weeks of a medical appointment at a participating endocrine clinic. Most participants scheduled their assessment on the same day of their medical appointment. Once the primary caretaker provided written informed consent, and youth assent was obtained, a trained psychological examiner initiated assessment, which typically lasted approximately two hours.

Youth included in this study met the following criteria: not diagnosed with another chronic medical condition; no history of traumatic brain injury; and were not prescribed medicine affecting the central nervous system, besides insulin. Youth were between 9 and 15 years of age at initial evaluation, past six months post-diagnosis at enrollment, and had at least two complete memory assessments. One-hundred and eighteen youth (118) met the aforementioned criteria and thus comprised this study's sample.

Power Analyses

Power analyses were conducted for hypotheses' complete multiple regression models. Given that each hypothesis' model includes the same number of predictors and pertains to the same group of participants, the following power analyses are applicable to each hypothesis. Analyses determined the number of participants necessary to reveal small (.02), medium (.13), and large (.26) effect sizes for multiple correlation coefficients (R^2; Cohen, Cohen, West & Aiken, 2003, pg. 93) with a power level of 80% and a .05 confidence level. The analyses revealed that with 118 participants, this study's sample size is large enough to detect medium (.13) and large (.26) effect sizes at a power level of 80% and .05 confidence interval. In contrast, analyses determined that 590 participants would be necessary to reveal a small effect size (.02) at a power level of 80% and a .05 confidence interval.

Sample Characteristics

The sample consisted of 118 youth with type I diabetes. Please see Table 1 (pg. 31) for demographic information and disease characteristics. Overall, participants were in moderate metabolic control, resided with both biological parents and were from middle-class families. The self-identified race/ethnicity distribution of this sample (21.2% minority) is reflective of the lower T1D prevalence rates found in ethnic minority groups (Delamater et al., 1999) and is similar to previously reported ethnicity distributions of metropolitan diabetes clinics (Glasgow et al. 1991).

Table 1

Sample Characteristics

	Percent	N	
Male	51.7	61	
Biological Parents' Married	79.1	91	
Caucasian	78.8	93	
	Mean	SD	Range
Follow-up Interval (years)	2.07	.36	1.01 - 3.82
Age at Time 1 (years)	12.66	1.66	9.28-15.72
Age at Time 2 (years)	14.71	1.76	11.27-18.08
Age at Onset (years)	8.07	3.73	.6 - 14.85
Duration at Time 1(years)	4.61	3.31	.5 - 13.19
Duration at Time 2 (years)	6.66	3.31	1.49-15.08
SES score at Time 1(Hollingshead[a])	47.88	11.60	11.50 - 70.00
SES score at Time 2 (Hollingshead[a])	48.38	11.04	11.50-70.00
HbA1c at Time 1 (%)	8.17	1.50	5.75 - 14.00
HbA1c at Time 2 (%)	8.34	1.28	5.80-12.70

[a] lower scores on the Hollingshead scale signify lower SES, higher scores signify higher SES

Measures/Materials

Memory measures were administered bi-annually to avoid practice effects.

Rote Verbal Memory. Rote verbal memory was measured bi-annually with the Verbal Memory Index of the Wide Range Assessment of Memory and Learning (WRAML; Shelow & Adams, 1990). The WRAML, normed for youth ages 5-17, is a well-standardized instrument designed to assess memory and learning abilities across

both visual and verbal modalities. Criterion-referenced validity for the WRAML has been demonstrated through significant correlations with both the Stanford-Binet-4th Edition Short Memory Test and the Wechsler Memory Scale-Revised (Sheslow & Adams, 1990).

The Verbal Memory Index is one of 4 indices derived from the WRAML. The Verbal Memory Index is comprised of 3 subtests and provides a measure of verbal rote memory. Scores on the Verbal Memory Index range from 45-155 ($M = 100$, $SD = 15$). The Verbal Memory Index demonstrates good internal reliability (coefficient alpha = .93) and test-retest reliability ($r_{xx} = .82$; Sheslow & Adams, 1990).

Quantitative Verbal Working Memory. Quantitative verbal working memory was measured with the Arithmetic subtest of the WISC-III (Sattler, 2001). The Arithmetic subtest is comprised of 24 problems; the first set of 5 items are presented visually on picture cards, the next 14 are presented orally, and the last 6 items are presented in written form. Problems increase in difficulty; initial problems require a simple knowledge of mathematical operation and later problems call for a more advanced knowledge of reasoning operations. Youth are instructed to solve these problems without the use of a pencil or paper. This stipulation requires youth to attend to verbal information, and retain and manipulate numerical symbols and operations in their working memory.

The Arithmetic subtest was normed as part of the WISC-III standardization in 1991 on 2,200 children aged 6-16 (Sattler, 2001). Scaled scores on the Arithmetic subtest range from 1-19 ($M = 10$, $SD = 3$). The Arithmetic subtest demonstrates good test-retest reliability ($r_{xx} = .78$; Sattler, 2001).

Problem Solving. The Test of Diabetes Knowledge (TDK; Johnson et al., 1982) is comprised of two subtests, General Information (39 multiple-choice items) and Problem-Solving (36 multiple-choice items). The Problem-Solving subtest is composed of items that describe challenging, but regularly occurring situations regarding diabetes care behaviors and corrections that youth with T1D commonly face (i.e., You are trying out for the swimming team and practice is mid-afternoon. Your urine tests are usually negative before lunch and in mid-afternoon. You should?). Content validity of the TDK was established by asking two physicians and one nurse practitioner to independently review all items. Items that were unanimously answered correctly were kept and included in the finalized version of the TDK. Johnson and associates administered the final version of the TDK to 151 youth with T1D (6-18 years old), the Spearman-Brown reliability coefficient was calculated and yielded an internal reliability estimate of .84 ($p < .0001$). The TDK provides a valid and reliable estimate of diabetes specific problem solving ability for youths aged 6 to 18 years old. The TDK was administered yearly to youth as part of self-report questionnaire battery assessing for psychosocial functioning.

Executive Functioning. Executive functioning, or cognitive flexibility, was assessed annually with the Trail Making Test (TMT, 1944, as cited by Lezak, Howieson & Loring, 2004). The TMT is composed of two parts, Trial Making Tests A and B. TMT-A requires participants to draw lines connecting consecutive numbers that are scrambled and out of order on a worksheet; participants are instructed to work as quickly as they can without lifting the pencil from the sheet of paper. TMT-B includes an alphabet sequence in addition to the number sequence described in TMT-A; participants must alternate

between the two sequences and draw lines connecting consecutive numbers and letters (i.e. A-1-B-2-C-3). Again, participants are told to work as quickly as possible without lifting the pencil from the page. The completion time and number of errors are recorded for both TMT-A and B. Executive functioning score are calculated by subtracting TMT-A from TMT-B; this practice is thought to reduce variability associated with psychomotor speed (Strauss, Sherman & Spreen, 2006). Test re-test reliabilities of .41 for TMT-A and .65 for TMT-B have been calculated for youth aged 9 to 14 (Strauss, Sherman & Spreen, 2006). Unfortunately no data regarding the reliability of TMT-B-A difference scores exists (Strauss, Sherman & Spreen, 2006).

Diabetes Responsibility. The Diabetes Family Responsibility Questionnaire (DFRQ; Anderson et al., 1990) consists of 17 items reflecting everyday situations or tasks relating to diabetes management (e.g., remembering to take morning or evening injections, remembering times when blood sugar or urine should be tested). Item content was determined through pilot interviews conducted with both professional health care providers and families with at least one child with T1D. For each item, the respondent (can be either the youth or the primary caretaker) assigns a value of 0 if neither the parent nor the child assumes responsibility, 1 if the parent takes or initiates responsibility for this situation almost all of the time, a 2 if the parent and child share responsibility for this situation about equally, or a 3 if the child takes or initiates responsibility for this situation almost all of the time. The DFRQ is a reliable estimate of self-care responsibility for youth 6-21 years of age; the scale has an internal reliability estimate of .85 (alpha coefficient). Concurrent validity for the DFRQ was established with the Moos Family

Environment Scale (FES, Moos, 1986 as cited by Anderson et al., 1990). Higher scores on the Independence subscale of the FES (higher scores indicate independence as a priority for individual family members) were associated with higher total scores on the DFRQ ($r = .21, p < .05$). The DFRQ was administered annually to both youth and their primary caretakers separately as part of the psychosocial questionnaire battery.

Diabetes Care Behaviors. Diabetes care behaviors were assessed annually through the 24-hour diabetes interview (Johnson et al., 1992). Primary caretakers (most often biological mothers) and youths participated in a series of two interviews conducted by a trained psychological examiner. Typically, the first of two interviews was performed at the endocrine clinic along with the neuropsychological assessments. The second follow-up interview was conducted approximately ten days later by telephone. The 24-hour diabetes interview requires children and their primary caretakers to temporally report the event of the previous day, in a detailed manner, beginning at the time the youth first awoke to the time the youth went to sleep. Each interview takes between 10-15 minutes to complete. Interviewers prompt respondents with certain questions if respondents do not report diabetes care behaviors (diet, blood glucose testing, insulin injections and exercise). Interviewers are trained to conduct the interview in a non-judgmental manner regardless of the interviewee's response (e.g., not sound surprised if the youth did not test blood glucose levels all day). This objective tone encourages interviewees to honestly report typical diabetes care behaviors and not respond according what they should have done to ideally manage their diabetes. The decision rules outlined

in Johnson et al. (1986) were used to settle discrepancies between primary caretaker and youth reports and to combine data from the two sources for analysis.

Reliability for the 24-hour diabetes interview has been established through significant correlations between parent and child report (Freund et al., 1991; Johnson et al., 1986). Test-retest reliability of the 24-hour diabetes interview has also been established; estimates of parent-child concordance have demonstrated stability over periods ranging from 3-months, (Freund et al., 1991) to 2 years (Johnson et al., 1992). Furthermore, the validity of the 24-hour diabetes interview was established with excellent concurrence rates between youths' (aged 7-12 years) 24-hour diabetes interviews and observers' reports of behavior occurrence and non-occurrence (Reynolds, Johnson & Silverstein, 1990).

Glycosylated Hemoglobin. The HbA1c assay provides an average glycoslyated hemoglobin level corresponding to the past 2-3 months (Blanc et al., 1981), thus, it provides an estimate of current metabolic control. The normative reference range of HbA1c spans 4-6% of hemoglobin; higher percentages indicate higher blood glucose levels and therefore, poorer metabolic control. The American Diabetes Association (ADA) has recommended assay percentages of < 8% for children and pre-adolescents (6-12) and 7.5% from adolescents and young adults (13-19). HbA1c assay percentages were gathered annually through participant's medical records. Given that HbA1c percentages inherently provide an average estimate of metabolic control, an index of HbA1c variability will be calculated in correspondence to each wave of WRAML cognitive data. Variability will be represented by the standard deviation of three HbA1c values (six

months prior to cognitive assessment, three months prior to cognitive assessment, and assay nearest to the cognitive assessment). HbA1c variability indices are hypothesized to provide more sensitive estimates of metabolic control and to correspond more accurately with diabetes care behaviors over time (Johnson et al., 1992).

Procedure

As part of the larger study, youth with T1D and their primary caretakers annually participated in a two-hour assessment; each year, a trained graduate student administered a series of neuropsychological tests, psychosocial questionnaires, medical history forms, and the 24-hour diabetes interview. Youth were administered the WRAML, WISC-III subtests, Trails A & B in years 1 and 3 of the study, while their primary caretakers completed psychosocial questionnaires (including the DFRQ) and a medical history form. Upon completion of the neuropsychological battery, youth also completed several psychosocial questionnaires (e.g. TDK, DFRQ, etc). Finally, research assistants and graduate student conducted 24-hour diabetes interviews with the youth and their caretakers, separately. Approximately ten days after the assessment session, a trained graduate student conducted the follow-up 24-hour diabetes interview of the child and the parent, separately, via the telephone.

Data Analysis Plan

Diabetes care outcome variables were measured with reference to the most recent American Diabetes Association (ADA) recommendations for children and adolescents with T1D.

Blood testing frequency. The ADA recommends testing blood glucose levels at least 4 times each day (Silverstein, 2005). Blood glucose testing frequency will be measured through the 24-hour diabetes interview. The ADA recommends 4 or more tests per day although some children test less frequently.

Eating frequency and carbohydrate and fat consumption. Thus far, dietary and nutritional recommendations for youth with TID remain congruent with national dietary guidelines created for all children and adolescents (Silverman et al., 2005). The ideal number of meals varies for each child given their height, weight, and activity level. The ADA discourages skipping meals, or encouraging children to "eat consistently without an appetite in an effort control blood glucose" (Silverman, 2005, pg. 196) and therefore does not offer a universal recommended number of meals, or eating frequency.

For adults with T1D, the ADA has recommended that less than 30% of calories should be from fat and that approximately 50-60% of calories be from carbohydrates (Franz et al., 2002). The ADA has not yet identified recommended fat and carbohydrate percentages specific to youth with T1D (Silverstein, 2005). Fat and carbohydrate consumption were calculated for each individual as a percentage of total calories ingested.

Exercise frequency and duration. In accordance with the Center for Disease Control and the American Academy of Sports Medicine, the ADA recommends a minimum of 30-60 minutes of moderate physical activity each day (Silverstein et al., 2005). In it most recent position paper for youth with TID, the ADA did not provide any recommendations regarding the number of exercise activities per day, rather the ADA

simply recommended a total minimum of 30-60 minutes per day (Silverstein et al., 2005). Thus, exercise duration, or length of exercise activity, and exercise frequency, or number of exercise activities per day, was measured with the 24-hour recall interview.

Hierarchical Regression Analyses. Multiple regression analyses (Cohen et al. 2003) were used to analyze cognitive predictors of diabetes care behaviors. A hierarchical regression model determined how cognitive variables at time 1 (baseline) predicted variability of diabetes care behaviors at time 2 (follow-up).

Table 2

Correlations of Sample Characteristics, Cognitive Skills (T1) and Diabetes Care Behaviors, HbA1c (T2)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Gender																	
2. Race/Ethnicity	.12																
3. Age	-.03	.05															
4. Duration	-.09	.02	-.00														
5. SES	**-.22 *[a]**	**-.28 **[a]**	-.04	.13													
6. DFRQ	.08	-.08	**.43 **[a]**	-.07	.13												
7. Rote Memory	.17	-.08	.01	.14	**.19 ***	**.18 *[a]**											
8. Working Memory	-.07	**-.24 ****	-.07	-.02	**.29 **[a]**	.10	**.50 **[a]**										
9. Problem-Solving	-.01	-.12	**.27 **[a]**	**-.22 ***	.15	**.24 **[a]**	**.23 ***	**.28 **[a]**									
10. Executive Functioning	-.03	.01	**-.22 ***	.02	-.09	.00	-.18	**-.28 **[a]**	**-.27 **[a]**								
11. BGM	-.01	**-.30 **[a]**	-.11	.10	-.00	-.04	**.20 ***	.16	.00	-.06							
12.Eating Frequency	.01	-.15	**-.20 ***	.04	-.09	-.11	.03	.06	-.07	.01	**.26 ****						
13.Exercise Frequency	**-.23 ****	.03	-.02	.05	.11	-.16	.04	-.01	-.15	.01	.13	**.25 ****					
14. % Calories Fat	.06	-.07	-.01	-.07	-.11	-.02	.01	**-.19 ***	-.12	.09	-.04	-.10	-.12				
15. % Calories Carbohydrates	-.06	.02	-.08	.11	.14	-.02	-.02	.14	.05	-.07	.01	**.18 ***	.10	**-.90 *[a]**			
16. HbA1c Variability	.04	.13	.18	.02	-.01	-.10	.10	-.01	.13	-.03	-.06	-.09	.12	-.14	.03		
17. Hba1C Mean score	.12	.14	.02	.02	**-.26 ****	-.10	-.03	-.12	-.06	.04	-.00	-.03	-.08	.08	-.12	**.21 ***	

Note: Gender and race dichotomously coded: 0 represents male and Caucasian, respectively. * $p < .05$, ** $p < .01$. [a] significant after the multistage Bonferroni correction

Table 3

Correlations of Sample Characteristics, Cognitive, Diabetes Care Behaviors and HbA1c (T2)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Gender																	
2. Race/Ethnicity	.12																
3. Age	-.06	.06															
4. Duration	-.10	.03	.02														
5. SES	**-.26** *	**-.26** **	-.05	.15													
6. DFRQ	.03	.00	**.34** ** [a]	-.06	**.27** **												
7. Rote Memory	.12	-.11	.14	**.22** *	**.26** **	.12											
8. Working Memory	.08	-.12	-.03	.03	**.32** ** [a]	**.23** *	**.58** ** [a]										
9. Problem-Solving	.09	-.18	.16	-.05	**.28** **	**.23** *	**.24** **	**.28** ** [a]									
10. Executive Functioning	-.12	.09	**-.20** *	-.01	**-.19** *	**-.20** *	**-.27** **	**-.33** ** [a]	**-.23** *								
11. BGM	-.01	**-.30** ** [a]	-.15	.08	.03	.00	**.20** *	.11	.15	-.09							
12.Eating Frequency	.01	-.15	**-.22** *	.02	-.03	-.04	.09	.09	-.05	.05	**.26** **						
13.Exercise Frequency	**-.23** **	.03	-.03	.05	.15	.06	.03	-.05	-.11	.07	.13	**.25** **					
14. % Calories Fat	.06	-.07	-.01	-.07	-.11	.06	-.13	-.16	-.09	.01	-.04	-.10	-.12				
15. % Calories Carbohydrates	-.06	.02	-.09	.10	.11	-.04	.07	**.18** *	.10	-.02	.01	**.18** *	.10	**-.90** * [a]			
16. HbA1c Variability	.04	**.19** *	.16	-.02	.01	.10	.08	.08	.02	.10	-.06	-.09	.12	-.14	.03		
17. Hba1C Mean score	.12	.14	.03	.02	**-.27** **	-.08	-.00	-.04	**-.22** *	-.03	-.00	-.03	-.08	.08	-.12	**.21** *	

Note: Gender and race dichotomously coded: 0 represents male and Caucasian, respectively. * $p < .05$, ** $p < .01$. [a] significant after the multistage Bonferroni correction

Results

Descriptive Results

Developmental status. Youth ranged in age from 9.17 years (pre-adolescence) to 15.72 years (older adolescence) at time 1. The mean age of the sample at time 1 was 12.64 years (SD = 1.69), an age associated with young adolescence. More than half of the participants, 61.9% (N = 73) of youth, were older than 12 years of age and thus considered adolescents. Despite this sample's proportion of adolescents at time 1, the mean DFRQ score at time 1 was 11.94 (SD = 1.54), a score associated with a considerable amount of parental responsibility and ambiguous diabetes care responsibility. At time 2, after a follow-up interval of approximately 2 years (M = 2.07, SD = .36), youth ranged in age from 11.27 years to 18.08 years. The mean age of the sample at time 2 was 14.71 years (SD = 1.76). At follow-up, the majority of youth, 94.1% (N = 112), were older than 12 years of age. The average DFRQ score at time 2 was 12.54 (SD = 2.30), a score reflective of a significant increase in disease responsibility when compared to the average DRFQ score at time 1. Table 4 shows descriptive statistics for developmental status of the sample at times 1 (baseline) and 2 (follow-up).

Cognitive skills at time 1. The sample obtained Verbal Memory Index scores and Arithmetic scores comparable, respectively, to WRAML and WISC national standardized averages. The mean rote memory score was 96.87 (SD = 13.20), consistent with WRAML's standardized VMI mean score of 100.00 (SD = 15.00). Similarly, the mean

working memory score was 11.26 (*SD* = 3.57), similar to the WISC's standardized Arithmetic mean score of 10 (*SD* = 3.00).

Youth's mean TDK-PS score was 25.33 (*SD* = 4.51); on average, youth in this study correctly answered 25, from a total of 36, diabetes-specific problem solving items.

Additionally, youth's mean completion time for the TMT-B-TMT-A was 41.29 seconds (*SD* = 24.28). Notably, executive functioning was measured by the timed difference between two separate tasks. Lower scores indicate shorter time differences, and thus reflect more advanced executive functioning.

Diabetes care behaviors at time 2. Diabetes care variables at time 2 were measured and compared to ADA's most recent diabetes care behavior recommendations (Silverstein et al., 2005). As previously mentioned, the ADA recommends that children monitor their blood glucose levels at least four times each day. On average, youth in our sample tested their blood glucose levels 2.95 (*SD* = 1.23) times each day. Approximately thirty-five percent (34.7%; *N* = 41) of the sample monitored their blood glucose levels four, or more times each day, in accordance with ADA recommendations (Silverstein et al., 2005). See Table 4 for descriptive statistics for blood glucose monitoring and all other diabetes care behaviors.

As described earlier, the ADA adopted the national dietary individualized guidelines; recommendations for number of meals and exercise will vary for youth depending on their height, weight, age and activity level. Despite this largely individualized approach, the ADA has advised that calories from fats comprise less than 30% of daily caloric intake and that calories from carbohydrates comprise 50-60% of

daily caloric intake for adults with T1D (Franz et al., 2002). The majority of our sample (68.1%, $N = 73$; $M = 1.04$, $SD = .67$) exercised at least once daily, for an average of 1.50 hours ($SD = 1.04$). Youth also ate an average of 3.88 times each day ($SD = .79$) and consumed an average of 2194.73 calories ($SD = 718.34$). Less than half of the sample, 24.7% ($N = 30$), consumed 30.00% or less of their calories from fats, as recommended by ADA ($M = 34.64\%$, $SD = 7.76$). Additionally, 33.1% of youth ($N = 40$) met ADA's recommended percentage of carbohydrate consumption ($M = 49.60\%$, $SD = 8.56$).

Metabolic control at time 2. The ADA has stated that children and pre-adolescents (defined by ADA as 9-12 years of age) should strive for HbA1c assays of less than 8 percent (Silverstein et al., 2005). Adolescents and young adults (defined by ADA as 13-19 years of age) have been advised to achieve assays of 7.5 % (Silverstein et al., 2005). Of youth defined as children and pre-adolescents at time 1($N = 67$), 43.3% ($N = 29$) obtained HbA1c assays of less than 8% at time 2 ($M = 8.34$, $SD = 1.17$). Among youth labeled as adolescents and young adults at time 1 ($N = 51$), 33.33% ($N = 17$) obtained HbA1c assays of less than 7.5% at time 2 ($M = 8.35$, $SD = 1.41$). HbA1c variability values are also reported in Table 4. As previously stated, these variability values reflect the variance of 3 HbA1c assays calculated within 1.5 years of cognitive testing.

Test-Retest Reliability and Change in Predictors from Time 1 to Time 2

In addition to descriptive data, Table 4 also reveals the stability and mean differences of variables from time 1 to time 2. Pearson correlation coefficients demonstrate the test-retest reliability *(rxx)* from time 1 to time 2. Paired-sample t-tests

denote whether or not a variable has significantly increased or decreased from time 1 to time 2.

Stability of cognitive skills. A comparison of the test-retest reliabilities reveals that stability decreases as the cognitive skill becomes more advanced and complex. See Table 4. Rote memory, the most basic of the four cognitive predictors, at time 1 accounts for 62% of the variance in rote memory at time 2, and is most reliable (*rxx* = .79). Alternatively, executive functioning, the most advanced of the four cognitive predictors, at time 1 only accounts for 5% of the variance in executive functioning at time 2, and is the least stable of the four cognitive predictors (*rxx* = .22). Rote memory also appears more stable than working memory (*rxx* = .61), a relatively similar cognitive skill. In fact, the magnitude of the discrepancy may be an artifact of the study's methodology. Rote memory was measured by the Verbal Memory Index, an aggregated index comprised of several rote memory subtests, while working memory was measured by a single instrument, the Arithmetic subtest of the WISC.

Regardless, it appears that in this sample, rote and verbal memory measures are notably more stable than the higher level skills of problem solving and executive functioning. These differences could reflect either the poorer reliabilities of these measures, or lack of developmental progression of the sample over time, or a combination of both factors.

Mean differences in cognitive skills. Youth's rote memory, problem-solving, and executive functioning scores significantly improved from time 1 to time 2, although working memory remained the same over time. See Table 4. While improvements in rote

memory and problem-solving were statistically significant, they are not likely to be clinically significant; furthermore, practice effects may have contributed to these improved scores. Alternatively, the 7.84 second improvement on the Trail Making Test may be indicative of a clinically significant improvement on this measure; again, it is possible that practice effects may have contributed to this improvement. The poor stability of the executive functioning measure also may have influenced this sizeable improvement; given that only 6% of the variance in executive functioning at time 1 is attributable to executive functioning at time 2, this improvement is likely influenced by a number of unknown factors.

Test-retest reliability and change in diabetes care behaviors. With the exception of executive functioning, cognitive predictors were more stable than diabetes care behaviors. See Table 4. Blood glucose monitoring was the most reliable diabetes care behavior (*rxx* = .34), blood glucose monitoring at time 1 accounted for 11% of the variance in monitoring at time 2. Fat consumption was the least reliable predictor (β = .20), fats consumed at time 1 accounted for only 4% of the variance in monitoring at time 2.

Frequency-related diabetes care behaviors, that is, blood glucose monitoring, eating frequency and exercise frequency, significantly decreased from time 1 to time 2. In contrast, dietary diabetes care behaviors, carbohydrates and fats consumed, did not significantly change over time.

Test-retest reliability and change in metabolic control. Mean HbA1c assays (β = .54) were significantly more reliable than HbA1c variability (β = -.01). Mean HbA1c at

time 1 accounted for 29% of the variance in mean HbA1c at time 2, while HbA1c variability at time 1 accounted for a negligible amount of variance (0%) in HbA1c variability at time 2. See Table 4. Neither mean HbA1c nor HbA1c variability significantly changed over time. Given the low stability of HbA1c variability it is noteworthy that this variable did not significantly change from time 1 to time 2.

Table 4

Test-retest reliability from Time 1(Baseline) to Time 2 (Follow-up) and Mean Differences (T-tests): Developmental Status, Cognitive Skills, Diabetes Care Behaviors and Metabolic Control of the Total Sample

$N = 118$	rxx[a]	R^2	Mean T1	SD T1	Range T1	Mean T2	SD T2	Range T2	T-test	p
Developmental Status										
Age (years)			12.64	1.69	9.17-15.72	14.71	1.76	11.27-18.08	-62.89	.00**
Disease Responsibility (DFRQ)	.42**	.17	11.94	1.54	8.00-19.00	12.56	2.30	4.50-18.00	-3.08	.00**
Cognitive Skills										
Rote Memory (WRAML-VMI)	.79**	.62	96.87	13.21	68.00-132.00	99.12	13.56	66.00-126.00	-2.79	.01**
Working Memory (WISC-Arithmetic)	.61**	.37	11.26	3.57	4.00-19.00	11.26	3.78	1.00-19.00	0.00	1.00
Problem Solving (TDK-PS)	.40**	.16	25.33	4.51	12.00-34.00	26.77	3.61	11.00-35.00	-3.46	.00**
Executive Functioning (TMT B-A; seconds)	.22*	.05	41.29	24.28	0.00-151.00	33.45	23.64	0.00-164.00	2.81	.01**
Diabetes Care Behaviors										
Blood glucose monitoring (#/day)	.34**	.11	3.40	.91	1.00-5.00	2.95	1.23	0.00-5.00	3.86	.00**
Eating frequency (#/day)	.27**	.07	4.42	.83	2.50-6.00	3.88	.79	1.50-6.00	6.00	.00**
Exercising frequency (#/day)	.24*	.06	1.34	.71	0.00-3.50	1.04	.67	0.00-4.00	3.84	.00**
Fat calories (% of total)	.20*	.04	34.70	6.73	18.00-66.25	34.64	7.76	16.00-54.50	.07	.94
Carbohydrate calories (% of total)	.25*	.06	49.49	7.62	23.25-66.75	49.60	8.56	27.00-73.00	-.12	.91
Metabolic Control										
HbA1c variability	-.01	.00	.58	.69	0.00-3.00	.47	.58	0.00-2.65	1.43	.16
HbA1c mean score	.54**	.29	8.17	1.49	5.75-14.00	8.34	1.28	5.80-12.70	-1.39	.17

Note: * indicates $p < .05$, ** indicates $p < .01$, t-test refers to a paired samples t-test of each variable at t1 and t2; [a] represents the Pearson product coefficient, or the extent to which each variable at t1 predicts the same variable at t2. All findings remained significant after multistage Bonferroni correction.

Hypothesized Analyses

Within each of the study's four main hypotheses, separate regressions were calculated for each diabetes care behavior hypothesized to be predicted by cognitive skill, and developmental risk factors. Regressions were analyzed with a traditional hierarchical regression model; hierarchical regressions consisted of five steps. Diabetes care behaviors at time 2 were entered as the criterion variables, developmental risk factors (disease responsibility and age), cognitive skills, a cognitive skill by age interaction (product) term, and diabetes care behavior at time 1, were entered as predictor variables.

Steps 2-4 of this model investigate the predictive ability of cognitive skills at time 1, after disease responsibility, age and the age by cognitive skill interaction term have been accounted for, but before diabetes care at time 1 is included in the model. As such, steps 2-4 provide an exploratory investigation of cognitive skills as predictors of diabetes care skills at time 2. Given the aforementioned low power, in conjunction with the novelty of this area of literature, these exploratory results will still provide valuable information. Step 5 provides a more rigorous investigation of the predictive ability of cognitive skills because it evaluates the predictive capacity of the cognitive skill after controlling for the baseline level of a diabetes care behavior (at time 1) in the prediction of that same diabetes care behavior two years later (at follow-up).

All regression analyses that failed to reach overall significance, or failed to reveal any significant individual cognitive or developmental predictors, are reported in the Appendices. Appendix I is comprised of nonsignificant regressions from the study's four main hypotheses. Appendix II is comprised of regressions from diabetes care mediational

analyses, and Appendix III is comprised of exploratory regressions from *post hoc* regression analyses.

Hypothesis 1: Rote Memory at Time 1 Will Predict Blood Glucose Frequency and Eating Frequency at Time 2

Rote memory and blood glucose monitoring. The full model was significant, $R^2 = .14$, $F(5, 112) = 176.47$, $p < .05$. See Table 5. Additionally, rote verbal memory at time 1 significantly predicted blood glucose testing at time 2 in steps 2, $\beta = .21$, $t(116) = 2.26$, $p < .05$, and 3, $\beta = .21$, $t(116) = 2.28$, $p < .05$ of the hierarchical regression. Rote memory and blood glucose monitoring were positively related, as rote memory improved, youth tested blood glucose levels more frequently. In step 2 of the model, rote verbal memory at time 1 uniquely accounted for 4.24 %, $pr(116) = .206$, of the variance in blood glucose testing at time 2. In step 3 of the model, rote verbal memory at time 1 uniquely accounted for 4.32%, $pr(116) = .208$, of the variance in blood glucose testing at time 2. Given that rote memory did not remain a significant predictor in final step of the model after diabetes care at time 1 was included, rote memory's predictive ability at steps 2 and 3 should be considered exploratory in nature.

The construct stability, or, reliability of blood glucose testing was also revealed; blood glucose testing at time 1 significantly predicted blood glucose testing at time 2, $\beta = .31$, $t(116) = 3.39$. Overall, blood glucose monitoring at time 1 was the strongest predictor of blood glucose monitoring at time 2, and accounted for the most variance 9.3%, $pr(116) = .31$ in blood glucose monitoring at time 2.

Table 5

Blood Glucose Monitoring at Time 2 Predicted by Rote Memory at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.03	.07	-.04	.00	
Step 2					
DFRQ	-.06	.07	-.08	.04	.04*
Rote Memory	.02	.01	.21*		
Step 3					
DFRQ	-.05	.08	-.06	.05	.00
Rote Memory	.02	.01	.21*		
Age	-.13	.26	-.05		
Step 4					
DFRQ	-.05	.08	-.06	.05	.00
Rote Memory	.01	.02	.15		
Age	-.11	.26	-.04		
AgeXRote Memory	.01	.02	.07		
Step 5					
DFRQ	-.05	.08	-.06	.14**	.09**
Rote Memory	.01	.02	.10		
Age	-.00	.25	-.00		
AgeXRote Memory	.01	.02	.06		
BG Monitoring T1	.42	.12	.31**		

Note: * $p < .05$, ** $p < .01$

Rote memory and eating frequency. Again, the full model was significant, $R^2 = .10$, $F(5, 112) = 2.32$, $p < .05$. See Table 6. However, rote verbal memory did not significantly predict eating frequency at any step of the hierarchical model. In contrast, age at time 1, dichotomously coded as younger than 12 and older than 12, did significantly predict eating frequency at time 2 in steps 3, $\beta = -.22$, $t(116) = -2.03$, $p < .05$, and 4, $\beta = -.21$, $t(116) = -1.94$, $p = .055$. Age and eating frequency were negatively related, as children aged and transitioned from pre-adolescence to adolescence, they ate

less frequently. In step 3 of the model, age at time 1 uniquely accounted for 3.84%, *pr* (116) = -.20, of the variance in eating frequency at time 2. In step 4, age at time 1 uniquely accounted for 3.57%, *pr* (116) = -.19, of the variance in eating frequency at time 2. Age did not remain a significant predictor in step 5 of the model and thus the aforementioned findings should be considered exploratory in nature.

Eating frequency at time 1 also significantly predicted eating frequency at time 2, $\beta = .23$, t (116) = 2.24 and thus demonstrated the reliability of this diabetes care skill. Overall, eating frequency at time 1 was the strongest predictor of eating frequency at time 2 and uniquely accounted for 4.84%, *pr* (116) = .22, of the variance in eating frequency at time 2, only slightly more than age at time 1.

Table 6

Eating Frequency at T2 Predicted by Rote Memory at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.06	.05	-.12	.01	
Step 2					
DFRQ	-.07	.05	-.12	.02	.00
Rote Memory	.00	.01	.05		
Step 3					
DFRQ	-.01	.06	-.03	.05	.04*
Rote Memory	.00	.01	.06		
Age	-.37	.18	-.22*		
Step 4					
DFRQ	-.02	.06	-.03	.06	.01
Rote Memory	-.00	.01	-.04		
Age	-.36	.18	-.21		
AgeXRote Memory	.01	.01	.12		
Step 5					
DFRQ	.00	.06	.01	.10*	.05*
Rote Memory	-.00	.01	-.01		
Age	-.26	.19	-.15		
AgeXRote Memory	.01	.01	.08		
Eating Frequency T1	.22	.10	.23*		

Note: * $p < .05$, ** $p < .01$

Hypothesis 2: Quantitative Verbal Working Memory at Time 1 Will Predict Carbohydrate and Fat Consumption at Time 2

Working memory and carbohydrate consumption. Working memory at time 1 failed to predict carbohydrate consumption at time 2. The full model was significant, but failed to reveal any significant cognitive or developmental predictors of carbohydrates. See Appendix I, Table 1.

Working memory and fat consumption. The overall model was not significant, $R^2 = .07$, $F(5, 112) = 2.30$, $p > .05$. Despite the failure of the overall model to attain significance, working memory at time 1 was a significant individual predictor of fat consumption at time 2 in steps 2, $\beta = -.19$, $t(116) = -2.04$, $p < .05$, and 3, $\beta = -.19$, $t(116) = -2.04$, $p < .05$. Better working memory was associated with lower fat consumption. The magnitude and direction of these regression coefficients are congruent with the Pearson correlation coefficient calculated for the same two variables reported in Table 2, $r(116) = -.19$, $p < .05$. In both steps 2 and 3 of the regression, working memory significantly accounted for 3.50% of the variance in fat consumption, $pr(116) = -.19$. The nonsignificant overall model combined with the failure of working memory to emerge as a significant predictor in the final step of the model, suggests that these findings should be considered exploratory and interpreted with caution.

Construct reliability of fat consumption was demonstrated; fats consumed at time 1 significantly predicted fats consumed at time 2, $\beta = .19$, $t(116) = 2.04$, $p < .05$. See Appendix I, Table 2, for the complete results.

Hypothesis 3: Problem Solving at Time 1 Will Predict Carbohydrate and Fat Consumption, in Addition to Exercise Frequency at Time 2

Problem solving at time 1 failed to predict carbohydrate consumption, fat consumption and exercise frequency at time 2. The overall models for both carbohydrate and fat consumption were nonsignificant. See Appendix I, Tables 3 and 4. The overall model for exercise frequency achieved significance; however, none of the individual predictors, problem solving, age, or disease responsibility, at time 1 significantly predicted exercise frequency at time 2. See Appendix I, Table 5 for detailed results.

Hypothesis 4: Executive Functioning at Time 1, a Superordinate Cognitive Skill, Will Predict the Same Diabetes Care Behaviors at Time 2 as the Other Cognitive Predictors

As previously discussed in the Method, executive functioning was calculated with a difference score quantified in seconds. Therefore, lower scores reflect faster speed of cognitive processing and thus are indicative of better executive functioning. Performance was error-free because the task allows for correction of mistakes.

Executive functioning failed to predict blood glucose monitoring (Appendix I, Table 6) eating frequency (Table 7), carbohydrate (Appendix I, Table 7) and fat (Appendix I, Table 8) consumption, and exercise frequency (Tables 8-10). While executive functioning failed to significantly predict eating and exercise frequency, significant developmental predictors of eating and exercise functioning emerged; these findings are summarized below.

Executive functioning and eating frequency. The full model was significant, $R^2 = .09$; $F(5, 112) = 2.32$, $p < .05$. See Table 7. Congruent with the rote memory and eating frequency regression analysis, age, dichotomously coded as younger than 12, and older than 12 years of age, significantly predicted eating frequency. As youth transitioned from pre-adolescence to adolescence, they ate less frequently. Standardized regression coefficients of age as a predictor of eating frequency are similar with either rote memory as a predictor, $\beta = -.21$, $t(116) = -1.94$, $p = .055$, or executive functioning as a predictor, $\beta = .-22$, $t(116) = -2.11$, $p < .05$ (see Tables 6 and 7). However, it should be noted that in both of the aforementioned regressions, age failed to achieve significance in step 5 of the model and thus, these findings should be considered exploratory in nature. Moreover, in

the final step of both regressions, eating frequency at time 1 emerged as the strongest predictor of eating frequency a time 2.

Table 7

Eating Frequency at T2 Predicted by Executive Functioning at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.06	.05	-.11	.01	
Step 2					
DFRQ	-.06	.05	-.11	.01	.00
Exec. Functioning	-.00	.00	.01		
Step 3					
DFRQ	-.01	.05	-.02	.05	.04*
Exec. Functioning	-.00	.00	-.04		
Age	-.35	.17	-.21*		
Step 4					
DFRQ	-.01	.05	-.02	.05	.00
Exec. Functioning	-.00	.01	-.11		
Age	-.36	.17	-.22*		
AgeXExec.Functioning	.00	.01	.10		
Step 5					
DFRQ	.01	.05	.01	.09*	.04*
Exec. Functioning	-.00	.01	-.08		
Age	-.25	.17	-.16		
AgeXExec. Functioning	.00	.01	.07		
Eating Frequency T1	.21	.09	.22*		

Note: * $p < .05$, ** $p < .01$

Executive functioning and exercise frequency. The full model was significant, $R^2 = .13$; $F(5, 112) = 3.21$, $p < .05$. See Table 8. At the final step of the model, disease responsibility, $\beta = -.21$, $t(116) = -2.09$, $p < .05$, and exercise frequency at time 1, $\beta = .31$,

t (116) = 3.18, $p < .05$, were significant predictors of exercise frequency at time 2. Disease responsibility uniquely accounted for 3.76% of the variance in exercise frequency, pr (116) = .19, $p < .05$, and was negatively related to exercise frequency; as youth were more responsible for their disease management, they exercised less frequently. Additionally, the executive functioning and age interaction (product) term also was significant, β = .27, t (116) = 1.97, $p < .05$.

Table 8

Exercise Frequency at T2 Predicted by Executive Functioning at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.07	.04	-.16	.03	
Step 2					
DFRQ	-.07	.04	-.16	.03	.00
Exec. Functioning	.00	.00	.01		
Step 3					
DFRQ	-.08	.05	-.18	.03	.00
Exec. Functioning	.00	.00	.02		
Age	.06	.15	.04		
Step 4					
DFRQ	-.08	.05	-.18	.05	.02
Exec. Functioning	-.00	.00	-.14		
Age	.04	.15	.03		
AgeXExec.Functioning	.01	.01	.21		
Step 5					
DFRQ	-.09	.04	-.21*	.13**	.08**
Exec. Functioning	-.01	.04	-.21		
Age	.22	.15	.16		
AgeXExec. Funct.	.01	.01	.27*		
Exercise Frequency T1	.29	.09	.31*		

Note: * $p < .05$, ** $p < .01$

Two additional regression analyses were calculated to further investigate the interaction term. Participants were divided into preadolescents, youth younger than 12 years of age, and adolescents, youth 12 years of age or older; identical regressions were calculated separately for each age group. Disease responsibility, executive functioning at time 1, and exercise frequency at time 1 were entered as predictors of exercise frequency at time 2. The aforementioned predictors were selected for entry due to their significance in the full model, Table 8, with all participants included. Among adolescents, the only significant predictor of exercise frequency at time 2, was exercise frequency at time 1; neither executive functioning nor disease responsibility significantly predicted exercise frequency in this age group. See Appendix I, Table 9. Congruent with the adolescent subgroup, the only significant predictor of exercise frequency at time 2 was exercise frequency at time 1. See Appendix I, Table 10. Thus, while the executive functioning by age interaction term achieved significance, executive functioning did not predict exercise frequency in either age group. The reason for this counterintuitive result is unclear.

Diabetes Care Behaviors as Mediators of the Relations between Cognitive Skills and HbA1c

Preliminary correlation tables 2 and 3 were examined for significant relations requisite for mediational analyses. Baron and Kenny's (1986) four steps for establishing mediation were employed as guidelines for these tests of mediation.

While the study initially hypothesized that diabetes care behaviors at time 2 (follow-up) would mediate the relation between cognitive skills at time 1 (baseline) and HbA1c variability at time 2 (follow-up), this study explored for the presence of additional

mediations. Given that longitudinal effects or trends are typically found after similar effects or trends are first demonstrated in cross-sectional data, this study also tested the cross-sectional mediational effect of diabetes care behavior at time 2 on the relation between cognitive skills at time 2 and HbA1c variability at time 2. Additionally, this study investigated cross-sectional and longitudinal mediation of diabetes care behaviors on the relation between cognitive skills and HbA1c mean values. Despite the fact that HbA1c variability is suggested in the literature to be a more valid measure of glycemic control than average metabolic control (Johnson et al., 1992), HbA1c variability demonstrated poor stability in this sample. Thus, HbA1c mean levels were also investigated. Given the significant correlation between SES and Hb1c mean levels noted in Table 2, SES was evaluated as a covariate of the relation between cognitive skill and mean HbA1c. Additional exploratory regression analyses were conducted to examine the effect of entered SES as a covariate. See Appendix II, Tables 1-8.

Steps 1& 2 (Baron & Kenny, 1986): Demonstrate that the initial variables (cognitive skills at time 1) are correlated with both the outcome variable (HbA1c variability at time 2) and the mediator variables (diabetes care behaviors at time 2).

Baron and Kenny (1986) argue that prior to the meditional analyses, significant relations should be established between the initial variables (here, cognitive skills) and both the mediator (diabetes care behaviors) and outcome variable (HbA1c). None of the cognitive skills at time 1 or 2 were significantly associated with HbA1c variability at time 1 or 2 (see Tables 2 and 3). Problem solving at time 2 was the only cognitive skill at either time 1 or 2 that significantly correlated with mean HbA1c at times 1 or 2, r (118) -.22, $p < .05$. However, problem solving at time 2 did not significantly relate to any diabetes care

behavior, this precluded further analyses of diabetes care behavior as a mediator of the relation between problem solving and mean HbA1c (Baron & Kenny, 1986). Despite the significant correlations between SES and mean HbA1c, SES entered as a covariate did not yield any relations that allowed further investigation for diabetes care mediation effects. See Appendix II: Tables 1-8. Therefore, these analyses failed to meet the prerequisite conditions for mediational analyses set forth by Baron & Kenny (1986). Contrary to hypotheses, diabetes care behaviors did not mediate the relation between cognitive skills and metabolic control.

Post-hoc analyses

Cognitive skills at T2 as predictors of diabetes care at T2. As previously mentioned, longitudinal effects or trends are typically investigated after cross-sectional effects have been noted. *Post hoc* cross-sectional analyses at follow-up were calculated to replicate the cross-sectional baseline analyses conducted by Souter et al. (2004) with a larger sample ($N = 224$) that included the present study's sample ($N = 118$). Thus, the present study's cross-sectional analyses at time 2 were conducted with a partial subsample from the Souter et al. study that continued to follow-up. Cognitive skills at time 2 that significantly correlated with diabetes care behaviors at time 2 (Table 3), along with significant longitudinal cognitive predictors, were analyzed cross-sectionally. Traditional hierarchical analyses assessed disease responsibility, cognitive skill, age and a cognitive skill by age interaction term, as predictors of diabetes care behaviors. Specifically, the following relations were tested in the context of the aforementioned hierarchical regressions; rote memory as a predictor of blood glucose monitoring, and

working memory as a predictor of carbohydrates and fats consumed. None of the overall model hierarchical regressions were significant. See Appendix III; Tables 1-3. However, rote memory at time 2 (follow-up) was significantly associated with blood glucose monitoring at time 2 (follow-up), $\beta = .21$, $t\ (116) = 2.28$, $p < .05$ (Appendix III; Table 1), and working memory at follow-up was significantly related to carbohydrate consumption at follow-up $\beta = .21$, $t\ (116) = 2.17$, $p < .05$ (Appendix III; Table 3).

Discussion

This study sought to replicate and extend the literature of cognitive predictors of diabetes care behaviors. For the first time, the cognitive abilities of rote memory, working memory, problem solving and executive functioning were examined in an exploratory investigation of longitudinal predictors of diabetes care behaviors. Additionally, this study investigated the possible mediational effects of diabetes care behaviors between cognitive skills and metabolic control. Finally, the concurrent relations between cognitive predictors and diabetes care behaviors were examined at follow-up.

Rote Memory and Blood Glucose Monitoring

To our knowledge, this study's exploratory analyses were the first to demonstrate that rote memory at baseline predicts blood glucose monitoring two years later. The present longitudinal subset from Souter et al.'s (2004) baseline cross-sectional investigation demonstrated better rote memory at baseline is associated with increased blood glucose monitoring at two-year follow-up. Rote memory at initial testing uniquely accounts for 4.32% of the variance in blood glucose testing two years later. See Table 5. The ability of rote memory to predict 4.32% of the variance in blood glucose monitoring 2 years after cognitive testing is noteworthy, given the modest stability of blood glucose monitoring over time ($rxx = .34$). In comparison, Souter et al.'s (2004) cross-sectional analyses of the baseline data determined that better rote memory was related to more frequent blood glucose testing and revealed that rote memory accounted for 5.5% of the variance in blood glucose testing.

However, rote memory at baseline did not remain a significant predictor of blood glucose monitoring at follow-up after blood glucose monitoring at baseline was included in the statistical model; overall, blood glucose monitoring at baseline was the stronger predictor of the same diabetes care behavior at follow-up. That is, when compared to blood glucose monitoring at baseline, rote memory at baseline was not a meaningful predictor of blood glucose monitoring at follow-up. As such, the aforementioned longitudinal findings should be considered exploratory. These exploratory findings may not be considered trivial as the corroboration in direction and magnitude of effects between the longitudinal and cross-sectional studies may indicate a robust, positive relation between this cognitive skill and diabetes care behavior. Additionally, it is possible that the restricted range of the rote memory factor in the present sample may have played a role in these exploratory findings. Perhaps rote memory would emerge as an increasingly meaningful predictor of blood glucose monitoring over time in youth with increasingly poor memory; this relation may only be salient and consequential among youth who are lacking memory skills necessary for diabetes care. Only 15.3% ($N = 18$) of the longitudinal sample obtained rote memory scores more than one standard deviation below the national mean. A future study may seek to over-sample youth with memory problems in order to determine if the relationship between this cognitive skill and blood glucose monitoring is affected by memory ability level.

In addition to exploratory longitudinal confirmation of Souter et al.'s (2004) cross-sectional results, the present study replicates Souter et al.'s (2004) cross-sectional findings with group performance at follow-up; rote memory at follow-up relates to

concurrent blood glucose monitoring at follow-up. See Table 3, and Appendix III; Table 1. While the overall follow-up model was non-significant, rote memory was a significant individual predictor of blood glucose monitoring in steps 2 and 3. Additionally, Pearson product correlations (See Table 3), which are theoretically identical to bivariate regressions, reveal a significant, positive relation between rote memory at baseline and blood glucose monitoring at baseline. Congruent with Souter et al.'s (2004) study, rote memory uniquely accounted for 4.20% of variance in more frequent blood glucose monitoring. Despite the corroboration between the bivariate regression and the individual predictor in the multivariate regression, the overall multivariate model was not significant and thus these results should be interpreted as exploratory. However, the exploratory nature of these findings is not inherently indicative of a weak or inconsequential relation between rote memory and blood glucose monitoring. It is possible that the overall cross-sectional regression model failed to reach significance as in Souter et al. ($N = 244$; 2004), because the present cross-sectional findings had nearly half the participants ($N = 118$) of Souter et al. (2004) and hence had reduced statistical power. Reduced statistical power was not a problem in the present longitudinal analyses because the repeated measure, in essence, doubled the number of subjects and simultaneously reduced inter-participant variability.

The present exploratory longitudinal results provide confirmation of the capacity of rote memory to predict blood glucose monitoring. Given that an individual's rote memory can be supplemented and bolstered by alarms, timers, or notepads unlike other

unalterable predictors of diabetes care behavior (i.e., age, developmental status and SES), these findings call for further replication and extension.

Working Memory and Fats and Carbohydrates Consumed

For the first time, an exploratory bivariate longitudinal regression analysis reveals that working memory at study enrollment predicts fat consumption at follow-up. Better working memory is associated with lower fat consumption two years later; the direction of this relation is important as it supports the ADA's recommendation that less than 30% of calories should be from fats (Franz et al., 2002). Working memory accounts for 3.53% of the variance in fat consumption at follow-up. To place this finding in context, the reliability of fat consumption over the two-year interval of the study is relatively low (*rxx* = .20), such that fat consumption at baseline accounts for only 3.57% of the variance in its own prediction at follow-up. Its low stability is likely due to a multitude of factors, and the ability of working memory to account for 3.53% of the variance in this diabetes care behavior over time could be considered particularly noteworthy.

Although working memory emerges as a significant, individual predictor of fat consumption, the overall model failed to reach significance and thus should be considered exploratory in nature. Typically, interpretation of a significant individual predictor in a non-significant overall model poses problems with type I error. However, a relation of similar magnitude between working memory and fat consumption is revealed in the preliminary correlational analyses (r = -.19). Given that a Pearson product-moment correlation is in essence a bivariate regression (β = -.19), this cross corroboration suggests that a genuine relation may exist between memory and fat

consumption, and the full model likely failed to reach significance because of the nonsignificance of the additional predictors included in the model and the aforementioned low stability in fats consumed from baseline to follow-up.

In contrast, this study failed to find a significant longitudinal relation between working memory at baseline and carbohydrate consumption at follow-up. While working memory was hypothesized to predict both fat and carbohydrate consumption over time, extant literature has only shown a cross-sectional relation of working memory and carbohydrates (Souter et al., 2004). Cross-sectional exploratory *post hoc* regressions were conducted to establish cross-sectional corroboration with the findings of Souter et al. (2004). See Appendix III; Table 3. Consistent with their results, working memory at follow-up relates to carbohydrates at follow-up, but not fats at follow-up. However, congruent with the longitudinal results, once again, neither of the cross-sectional *post hoc* overall models reached significance, and the relation between working memory at follow-up and carbohydrates at follow-up should be considered exploratory. The failure of the overall model to achieve significance is likely an artifact of the low stability of dietary behaviors and the present study's small cross-sectional sample size and low power compared to Souter et al. (2004).

To summarize the dietary findings, longitudinally, working memory predicts fat consumption, while cross-sectionally, working memory relates to carbohydrate consumption. These findings may reflect youth's more proximal, immediate attention to carbohydrates, and distal thought related to fats. Souter et al. (2004) postulated that working memory is cross-sectionally associated with carbohydrates, but not fats, because

of the increased popularity of continuous subcutaneous insulin infusion (CSII), the insulin pump, and its emphasis on carbohydrate counting to determine insulin dosages (Gillepsie, Kulkarni & Daly, 1998). Thus, skills and techniques associated with working memory (Strauss, Sherman & Spleen, 2006) are frequently used by youth with insulin pumps to calculate carbohydrates consumed, but not fats. Alternatively, youths' concerns and cognitions surrounding fat consumption may be related to more distal events such as risk of long-term disease complications like cardiovascular disease, or obesity, and thus are detected in longitudinal analyses.

The relatively low stability of the diet behaviors over time undoubtedly contributed to the inability of the overall regression results to achieve significance. Nevertheless, the detection of a relation between working memory and diet composition in the bivariate analyses indicates that relations exist, as hypothesized, but that the other predictors included in the regressions were not relevant to carbohydrate and fat consumption. It is possible that with a more relevant set of predictors than those used, working memory would emerge as an even stronger predictor of carbohydrates and fats consumed. Further, the tendency for children to underestimate or incorrectly report dietary behavior due to social desirability has been established in the literature (Baxter, Smith & Litaker, 2004). Perhaps an observational study of dietary ingestion would provide a more stable, valid measure of carbohydrate and fat consumption.

Problem Solving as a Predictor of Diabetes Care Behavior

Problem solving did not significantly predict any hypothesized diabetes care behaviors. While some literature has established positive relations between diabetes-

specific problem solving and specific diabetes care behaviors, (i.e., urine testing, insulin injection, and blood glucose testing; Johnson et al., 1982; McCaul et al., 1987) another study failed to discover a link between problem solving and diet or exercise (McCaul et al. 1987). Hill-Briggs (2003) posited that the methodology and the conceptualization of diabetes-specific problem solving have serious limitations that may contribute to this literature's ambiguous, and weak, findings. Hill-Briggs (2003) notes that most investigations of problem solving use different questionnaires, moreover, a large number of studies create their own measure. In short, a consensus has not yet been reached as to the best measure of problem solving, i.e., there is no "gold standard" measure (Hill Briggs, 2003). The tendency for each study to generate a new, different measure precludes an understanding and refinement of the psychometric properties of these measures. The Test of Diabetes Knowledge Problem Solving subscale (TDK-PS) used in the present study has an advantage over other questionnaires as one whose psychometric properties has been reported. However, only the Spearman-Brown internal reliability coefficient of the TDK has been calculated and published (r = .84), and the test re-test reliabilities and validities are unknown. This study calculated test-retest reliability of the TDK-PS from baseline to follow-up and the reliability of this measure was relatively low (rxx = .40), compared to the reliability calculated for the other cognitive predictors (i.e., rote memory, rxx = .79, working memory, rxx = .61). The paucity of psychometric data of the TDK-PS combined with its low stability in the present study confound the interpretation of the nonsignificant findings.

Beyond its psychometric issues, the conceptualization of disease-specific problem solving also is problematic. The diabetes specific nature of many of these questionnaires inherently confound problem solving skills and diabetes knowledge. For example, a question from the TDK-PS reads, "you are at a school football game and being to feel dizzy, shaky, and faint. What should you do?" While this question clearly addresses problem solving skills, it also assesses general disease knowledge. The inherent confound associated with many disease specific problem solving items indicates that a measure of general, neurocognitive problem solving ability in addition to disease specific skills could be useful to include in future studies.

Executive Functioning as a Predictor of Diabetes Care Behavior

For the first time, executive functioning was investigated as a predictor of diabetes care behaviors. Contrary to hypothesized results, executive functioning did not predict diabetes care behaviors. The low stability of the measure used to quantify executive functioning may have contributed to the failure to reveal significant relations. The executive functioning measure had the lowest test-retest reliability of all of the cognitive predictors (*rxx* = .22). On average, youth were able to complete the Trail-Making Test (Trails) approximately 7 seconds faster at follow-up as compared with their time at baseline. A review of the literature of Trails difference scores fails to reveal a highly stable measure with established psychometric properties. No data currently exists regarding the reliability or validity of Trails difference scores. Unfortunately, studies that have investigated Trails-Form-B and/or Trails-Form-A reliability among children have yielded ambiguous results; test –retest reliability coefficients range from "low" (Trails,

Form-A, r = .41;Strauss, Sherman & Spreen, 2006) to "good" (Trails, Form-B, exact coefficient not provided; Strauss, Sherman & Spreen, 2006). Additionally, investigations of practice effects, conducted over 2-month intervals, have established demonstrable changes in scores, especially in youth (Strauss, Sherman & Spreen, 2006). However, given the average two year interval between cognitive assessments in this study, it is unlikely that practice effects contributed to the low Trails difference score reliability in this study.

In addition to its psychometric properties, youths' scores on the Trails may have been influenced by developmental processes. Executive functioning is characterized by formal operations, abstract thinking and higher-level cognitive processes that are not completely developed until mid-adolescence (Sattler, 2001). Thus, it is possible that the significant change in scores is reflective of better developed, more advanced executive functioning. Future studies may benefit from using alternative measures of executive functioning such as the Wisconsin Card Sorting Task, or an aggregated or composite measure of executive functioning to provide better test stability. Additionally, an investigation of executive functioning as a predictor of diabetes care skills in adults with T1D may provide valuable information as it would eliminate possible developmental confounds.

Diabetes Care as a Mediator of the Relation between Cognitive Skill and Metabolic Control

Diabetes care was not as a mediator of any relation between cognitive skill and metabolic control. Despite the intuitive, expected relation between diabetes care and HbA1c, several previous studies have also failed to produce significant relations between

these two variables (Johnson et al. 1992; Johnson et al., 1994). Johnson et al. (1994) hypothesized that one major problem was an over-reliance on the HbA1c assays as a "gold standard" measure of metabolic control. Johnson et al (1994) noted that these assays provide estimates of average blood glucose levels over 2-3 months and therefore do not take into account blood glucose variabilities, how many times blood glucose levels approximated euglycemia, or how many times individuals experienced hypoglycemia. Her study (Johnson et al., 1994) concluded that HbA1c variability levels would better represent youth's metabolic control. Although this study calculated HbA1c variability levels at baseline and follow-up, this variable had poor reliability (*rxx* = .04), and likely did not produce the expected conceptual and methodological advantage over mean HbA1c assays. Additionally, puberty is associated with insulin resistance and poorer HbA1c assays in youth with T1D (Amiel et al., 1986; Wysocki, Greco & Buckloh, 2003). Pubertal status and its effect on the HbA1c assays may have further masked potential relations between diabetes care behaviors and metabolic control in this study's youth.

Summary, Limitations and Future Directions

In sum, this study was the first to document exploratory longitudinal prediction of blood glucose monitoring with rote memory and fat consumption with working memory. The study's exploratory results also corroborated previous research in its cross-sectional analyses; working memory was associated with carbohydrate consumption and rote memory with blood glucose monitoring. These results extend the knowledge of cognitive predictors of diabetes care behaviors and call for future replication with a larger sample that includes participants with a wider range of cognitive skills. Future extensions

may include more sophisticated statistical analyses that may investigate how the change in memory abilities predicts change in diabetes care over time.

Given the dearth of studies in the area of cognitive predictors of diabetes care behaviors, several *post hoc* exploratory analyses were conducted in order to maximize the information gleaned from this study. The number of hierarchical and *post hoc* analyses calculated in this study suggest that experiment-wise type I error may be a concern (Cohen et al., 2003). However, the hierarchical nature of the regressions inherently protects against type I error (Cohen et al., 2003) because individual predictors are typically only interpreted if the overall model is significant. It is possible that some of the study's significant findings did not reflect a genuine association of variables. However, this danger is mitigated by the distinct pattern of results. Rote memory relates to blood glucose monitoring longitudinally and concurrently, in a direction and magnitude consistent with previous literature (Souter et al., 2004). Additionally, working memory relates to carbohydrates cross-sectionally in a direction that corroborates previous research (Souter et al., 2004). Moreover, individual predictor significance of working memory correspond in size and magnitude to correlation coefficients obtained.

Future longitudinal, correlational studies of cognitive predictors of diabetes care behaviors may further advance the field through use of aggregated cognitive measures, particularly for more complex cognitive skills such as problem solving and executive functioning, to increase stability and reliability of these abilities. Alternatively, future work might benefit from an investigation of diabetes care behaviors related to medical outcomes other than HbA1c, or HbA1c variability. Several studies have failed to relate

diabetes care to the HbA1c medical outcome (Johnson et al. 1992; Johnson et al., 1994). Perhaps the relations between diabetes care behaviors and triglycerides, hyperlipidemia, hypertension and other medical correlates of long-term disease complications might better elucidate the mechanisms through which diabetes care affects medical outcomes and relates to disease complications.

A more thorough understanding of the associations among cognitive predictors, diabetes care behaviors and medical outcomes could be utilized in innovative intervention techniques. Specifically, interventions could improve behaviors by enhancing precursory memory skills and strategies, techniques, or devices. These tangible, concrete intervention techniques may prove to be more successful and cost effective than interventions aimed at motivation, or alteration of family structure and dynamic.

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Appendix I: Primary Hypothesized Regressions

Hypothesis 2: Quantitative working memory at T1 and dietary factors at T2
Working memory and carbohydrate consumption. The full model was significant, $R^2 = .09$, $F(5, 112) = 2.30$, $p < .05$. See Table 1. Carbohydrate consumption at time 1 was the only significant predictor of carbohydrate consumption at time 2, $\beta = .25$, $t(116) = 2.77$. Carbohydrates consumed by youth at time 1 uniquely accounted for 6.40%, $pr(116) = .253$ of the variance in carbohydrates consumed by youth at time 2.

Table 1

Carbohydrates Consumption at T2 Predicted by Working Memory at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	ΔR^2
Step 1					
DFRQ	-.13	.52	-.02	.00	
Step 2					
DFRQ	-.21	.52	-.04	.02	.02
Working Memory	.33	.22	.14		
Step 3					
DFRQ	.01	.57	.00	.03	.01
Working Memory	.33	.22	.14		
Age	-1.61	1.79	-.09		
Step 4					
DFRQ	-.06	.58	-.01	.03	.00
Working Memory	.10	.38	.04		
Age	-1.51	1.80	-.09		
AgeXWorking Mem.	.34	.48	.12		
Step 5					
DFRQ	-.02	.56	-.00	.09*	.06**
Working Memory	.07	.37	.03		
Age	-1.86	1.75	-.11		
AgeXWorking Mem.	.33	.46	.11		
Carbohydrates T1	.28	.10	.25*		

Note: * $p < .05$, ** $p < .01$

Working memory and fat consumption. The overall model was nonsignificant. See Table 2.

Table 2

Fat Consumption at T2 Predicted by for Working Memory at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.08	.47	-.02	.00	
Step 2					
DFRQ	.01	.46	.00	.04	.04*
Working Memory	-.41	.20	-.19*		
Step 3					
DFRQ	.03	.51	.01	.04	.00
Working Memory	-.41	.20	-.19*		
Age	-.15	1.62	-.01		
Step 4					
DFRQ	.07	.52	.01	.04	.00
Working Memory	-.27	.35	-.13		
Age	-.21	1.63	-.01		
AgeXWorking Mem.	-.21	.43	-.08		
Step 5					
DFRQ	.12	.52	.02	.07	.03*
Working Memory	-.23	.34	-.10		
Age	.17	1.61	.01		
AgeXWorking Mem.	-.22	.42	-.08		
Fats	.22	.11	.19*		

Note: * $p < .05$, ** $p < .01$

Hypothesis 3: Problem solving at time 1 will predict carbohydrate and fat consumption in addition to exercise frequency at time 2

Problem solving and carbohydrate consumption. The overall model was nonsignificant. See Table 3.

Table 3

Carbohydrate Consumption at T2 Predicted by Problem Solving at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.13	.52	-.02	.00	
Step 2					
DFRQ	-.21	.53	-.04	.00	.00
Problem Solving	.11	.18	.05		
Step 3					
DFRQ	.03	.57	.01	.02	.01
Problem Solving	.17	.19	.09		
Age	-2.14	1.87	-.12		
Step 4					
DFRQ	.04	.57	.01	.02	.01
Problem Solving	-.07	.33	-.04		
Age	-1.82	1.90	-.10		
AgeXProb.Solving	.35	.40	.15		
Step 5					
DFRQ	.09	.56	.02	.08	.06**
Problem Solving	-.11	.32	-.06		
Age	-2.03	1.85	-.12		
AgeXProb.Solving	.33	.39	.14		
Carbohydrates .28	.10	.25*			

Note: * $p < .05$, ** $p < .01$

Problem solving and fat consumption. The overall model was nonsignificant. See Table 4.

Table 4

Fat Consumption T2 Predicted by Problem Solving at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.08	.47	-.02	.00	
Step 2					
DFRQ	.07	.48	.01	.02	.02
Problem Solving	-.22	.16	-.13		
Step 3					
DFRQ	.01	.52	.00	.02	.00
Problem Solving	-.23	.17	-.13		
Age	.57	1.69	.04		
Step 4					
DFRQ	.00	.52	.00	.03	.01
Problem Solving	.05	.30	.03		
Age	.20	1.72	.01		
AgeXProb,Solving	-.42	.36	-.19		
Step 5					
DFRQ	.04	.51	.01	.06	.04*
Problem Solving	.12	.30	.07		
Age	.43	1.70	.03		
AgeXProb.Solving	-.44	.36	-.20		
Fats	.23	.11	.20*		

Note: * $p < .05$, ** $p < .01$

Problem solving and exercise frequency. As hypothesized, this overall model was significant, $R^2 = .10$; $F(5, 112) = 2.57, p < .05$. See Table 5. Exercise frequency at time 1 uniquely accounted for 6.25% of the variance in exercise frequency at time 2, $pr(116) = .250$, and was the only significant predictor of exercise frequency at time 2, $\beta = .27$, $t(116) = 2.73, p < .05$.

Table 5

Exercise Frequency at T2 Predicted by Problem Solving at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.07	.04	-.16	.03	
Step 2					
DFRQ	-.06	.04	-.13	.04	.01
Problem Solving	-.02	.01	-.12		
Step 3					
DFRQ	-.07	.04	-.16	.04	.00
Problem Solving	-.02	.02	-.14		
Age	.11	.15	.08		
Step 4					
DFRQ	-.07	.05	-.16	.04	.00
Problem Solving	-.02	.03	-.16		
Age	.11	.15	.08		
AgeXProb.Solving	.00	.03	.02		
Step 5					
DFRQ	-.08	.04	-.19	.10*	.06**
Problem Solving	-.01	.03	-.08		
Age	.24	.15	.17		
AgeXProb.Solving	-.01	.03	-.03		
Exercise Frequency T1	.25	.09	.27*		

Note: * $p < .05$, ** $p < .01$

Hypothesis 4: Executive functioning at time 1 will predict the same diabetes care behaviors as the other cognitive predictors

Executive functioning and blood glucose monitoring. The full model achieved significance, $R^2 = .09$; $F(5, 112) = 2.21$, $p = .06$. See Table 6. Blood glucose monitoring at time 1 was the only significant predictor of blood glucose monitoring at time 2, $\beta = .34$, $t(116) = 3.74$, $p < .05$.

Table 6

Blood Glucose Monitoring at T2 Predicted by Executive Functioning at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.03	.07	-.04	.00	
Step 2					
DFRQ	-.03	.07	-.04	.01	.00
Exec. Functioning	-.00	.01	-.07		
Step 3					
DFRQ	-.01	.08	-.02	.01	.00
Exec. Functioning	-.00	.01	-.07		
Age	-.14	.27	-.06		
Step 4					
DFRQ	-.01	.08	-.02	.01	.00
Exec. Functioning	-.00	.01	-.09		
Age	-.14	.27	-.06		
AgeXExec.Functioning	.00	.01	.01		
Step 5					
DFRQ	-.01	.08	-.02	.12*	.11**
Exec. Functioning	-.01	.01	-.11		
Age	-.02	.26	-.01		
AgeXExec. Functioning	.01	.01	.08		
BG Monitoring T1	.46	.12	.34**		

Note: * $p < .05$, ** $p < .01$

Executive functioning and carbohydrates consumed. The full model approached significance, $R^2 = .09$; $F(5, 112) = 2.21$, $p = .06$. See Table 7.

Table 7

CLHR (Part 1) Carbohydrates consumed at T2 Predicted by Executive Functioning at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	ΔR^2
Step 1					
DFRQ	-.13	.52	-.02	.00	
Step 2					
DFRQ	-.13	.52	-.02	.01	.00
Exec. Functioning	-.03	.03	-.07		
Step 3					
DFRQ	.16	.57	.03	.02	.01
Exec. Functioning	-.04	.03	-.10		
Age	-2.16	1.85	-.12		
Step 4					
DFRQ	.15	.57	.03	.02	.00
Exec. Functioning	-.01	.05	-.03		
Age	-2.06	1.86	-.12		
AgeXExec.Functioning	-.04	.07	-.09		
Step 5					
DFRQ	.19	.56	.04	.09	.07**
Exec. Functioning	-.02	.05	-.06		
Age	-2.51	1.81	-.14		
AgeXExec. Funct.	-.03	.07	-.07		
Carbohydrates	.30	.10	.26*		

Note: * $p < .05$, ** $p < .01$

Executive functioning and fats consumed. The full model was not significant, $R^2 = .06$; $F(5, 112) = 1.30, p > .05$. See Table 8.

Table 8

Fats consumed at T2 Predicted by Executive Functioning at T1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.08	.47	-.02	.00	
Step 2					
DFRQ	-.08	.47	-.02	.01	.01
Exec. Functioning	.03	.03	.09		
Step 3					
DFRQ	-.14	.52	-.03	.01	.00
Exec. Functioning	.03	.03	.10		
Age	.39	1.69	.03		
Step 4					
DFRQ	-.13	.52	-.03	.02	.01
Exec. Functioning	.01	.05	.01		
Age	.29	1.69	.02		
AgeXExec.Functioning	.05	.06	.11		
Step 5					
DFRQ	-.07	.51	-.02	.06	.04*
Exec. Functioning	.01	.05	.05		
Age	.74	1.68	.05		
AgeXExec. Funct.	.04	.06	.08		
Fats	.24	.11	.20*		

Note: * $p < .05$, ** $p < .01$

Executive functioning and exercise frequency by age group. The full model for adolescents, youth 12 years of age or older, approaches significance. Among adolescents, disease responsibility, exercise at time 1 and executive functioning at time 1 predict 9.9%

of the variance in exercise frequency at time 2, $R^2 = .10$; $F(3, 69) = 2.54, p < .06$. See Table 9. However, the only significant predictor of exercise frequency at time 2 was exercise frequency at time 1, $\beta = .27, t(71) = 2.31, p < .05$.

Table 9

Exercise Frequency at Time 2 Predicted by Executive Functioning at Time 1 for Older Youth (≥ 12 years of age; N = 45)

Variable	*B*	SE *B*	β	R^2
Step 1				
DFRQ	-.07	.05	-.16	.10
Exercise T1	.30	.13	.27*	
Executive Funct. T1	.01	.00	.17	

Note: * $p < .05$, ** $p < .01$

The full model for pre-adolecesents, youth younger than 12 years of age, is significant. Disease responsibility, exercise at time 1 and executive functioning at time 1 predict 18.0% of the variance in exercise frequency at time 2, $R^2 = .18$; $F(3, 41) = 2.54, p < .05$. See Table 10. Congruent with adolescents, the only significant predictor of exercise frequency at time 2 was exercise frequency at time 1, $\beta = .27, t(71) = 2.31, p < .05$.

Table 10

Exercise Frequency at Time 2 Predicted by Executive Functioning at Time 1 for Younger Youth (< 12 years of age; N = 45)

Variable	*B*	SE *B*	β	R^2
Step 1				
DFRQ	-.15	.09	-.26	.18*
Exercise T1	.27	.13	.31*	
Executive Funct. T1	-.00	.00	-.17	

Note: * $p < .05$, ** $p < .01$

Appendix II: Mediational Analyses

Diabetes care behaviors at time 2 as mediators of the relations between cognitive skills at time 1 and HbA1c at time 2

Steps 1& 2: Demonstrate that the initial variables (cognitive skills at time 1) are correlated with both the outcome variable (HbA1c at time 2) and the mediator variables (diabetes care behaviors at time 2).

Table 1

HbA1c at Time 2 Predicted by Rote Memory and SES at Time 1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.26**	.07**	
Step 2					
SES	-.03	.01	-.26**	.07*	.00
Rote Memory	.00	.01	.02		

Note: * $p < .05$, ** $p < .01$

Table 2

HbA1c at Time 2 Predicted by Rote Memory and SES at Time 2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.27**	.07**	
Step 2					
SES	-.03	.01	-.29**	.08**	.01
Rote Memory	.01	.01	.07		

Note: * $p < .05$, ** $p < .01$

Table 3

HbA1c at Time 2 Predicted by Working Memory and SES at Time 1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.26**	.07**	
Step 2					
SES	-.03	.01	-.25**	.07*	.00
Working Memory	-.02	.03	-.05		

Note: * $p < .05$, ** $p < .01$

Table 4

HbA1c at Time 2 Predicted by Working Memory and SES at Time 2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.27**	.07**	
Step 2					
SES	-.03	.01	-.28**	.07*	.00
Working Memory	.02	.03	.05		

Note: * $p < .05$, ** $p < .01$

Table 5

HbA1c at Time 2 Predicted by Problem Solving and SES at Time 1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.26**	.07**	
Step 2					
SES	-.03	.01	-.26**	.07*	.00
Problem Solving	-.01	.03	-.02		

Note: * $p < .05$, ** $p < .01$

Table 6

HbA1c at Time 2 Predicted by Problem Solving and SES at Time 2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.27**	.07**	
Step 2					
SES	-.03	.01	-.22*	.09**	.02
Problem Solving	-.05	.03	-.15		

Note: * $p < .05$, ** $p < .01$

Table 7

HbA1c at Time 2 Predicted by Executive Functioning and SES at Time 1 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.26**	.07**	
Step 2					
SES	-.03	.01	-.26**	.07*	.00
Executive Functioning	.00	.01	.02		

Note: * $p < .05$, ** $p < .01$

Table 8

HbA1c at Time 2 Predicted by Executive Functioning and SES at Time 2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
SES	-.03	.01	-.27**	.07**	
Step 2					
SES	-.03	.01	-.28**	.07* *	.01
Executive Functioning	.00	.01	-.01		

Note: * $p < .05$, ** $p < .01$

Appendix III: *Post hoc* Analyses

Cognitive skills at T2 as predictors of diabetes care at T2. The overall model was nonsignificant.

Table 1

BGM at T2 Predicted by Rote Memory at T2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	.02	.05	.00	.00	
Step 2					
DFRQ	-.01	.05	-.02	.04	.04*
Rote Memory	.02	.01	.21*		
Step 3					
DFRQ	.02	.05	.00	.06*[a]	.02
Rote Memory	.02	.01	.21*		
Age	-.84	.51	-.15		
Step 4					
DRFQ	.00	.05	.00	.07	.00
Rote Memory	.06	.09	.64		
Age	3.01	8.39	.54		
AgeXRote Memory	-.04	.09	-.83		

Note: *[a] $p < .06$, * $p < .05$, ** $p < .01$

Cognitive skills at T2 as predictors of diabetes care at T2. The overall model was nonsignificant.

Table 2

Fats Consumed at T2 Predicted by Working Memory at T2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	.19	.31	.06	.00	
Step 2					
DFRQ	.33	.32	.10	.03	.03
Working Memory	-.37	.19	-.18		
Step 3					
DFRQ	.37	.32	.11	.04	.00
Working Memory	-.38	.19	-.18		
Age	-2.64	3.28	-.08		
Step 4					
DRFQ	.36	.33	.11	.04	.00
Working Memory	-.13	.77	-.06		
Age	.46	9.81	.01		
AgeXWork. Memory	-.27	.79	-.15		

Note: * $p < .05$, ** $p < .01$

Cognitive skills at T2 as predictors of diabetes care at T2. The overall model was nonsignificant.

Table 3

Carbohydrates Consumed at T2 Predicted by Working Memory at T2 (N = 118)

Variable	*B*	SE *B*	β	R^2	Δ R^2
Step 1					
DFRQ	-.14	.35	-.04	.00	
Step 2					
DFRQ	-.31	.35	-.08	.04	.04*
Working Memory	.46	.21	.20*		
Step 3					
DFRQ	-.34	.36	-.09	.04	.00
Working Memory	.46	.21	.21*		
Age	1.95	3.62	.05		
Step 4					
DRFQ	-.34	.36	-.09	.04	.00
Working Memory	.35	.85	.15		
Age	.51	10.80	.01		
AgeXWork. Memory	.12	.87	.06		

Note: * $p < .05$, ** $p < .01$

Vita

Michelle Marie Greene as born on April 1, 1982, in Des Plaines, Illinois and is an American Citizen. She graduated from Lake Park High School in Roselle, Illinois in 2000. She received her Bachelor of Arts in Psychology with a minor concentration in Hispanic Studies from Northwestern University, Evanston, Illinois in 2004.