DEVELOPMENT OF THE DIABETES FAMILY ASSESSMENT MEASURE (D-FAM) FOR CHILDREN AND ADOLESCENTS

By

ADAM BENJAMIN LEWIN

A DISSERTATION PRESENTED TO THE GRADUATE SCHOOL OF THE UNIVERSITY OF FLORIDA IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

UNIVERSITY OF FLORIDA

2007
To my grandmother, Gwen Lewin
ACKNOWLEDGMENTS

I thank my mentor and chair Gary R. Geffken, Ph.D. for his support and guidance throughout my training. Not only has Dr. Geffken has been instrumental in my research and clinical training, but he has contributed to my personal and professional development in countless ways. I am extremely grateful for his countless hours, incessant availability, and passion for mentorship! I also thank members of my doctoral committee for their many hours: James H. Johnson, Ph.D., M. Jeffrey Farrar, Ph.D., and Samuel Sears, Ph.D. In addition, I thank Janet H. Silverstein (Division Chief, Pediatric Endocrinology) and her staff for their collaboration with this project. Dr. Silverstein has been a professional and personal mentor for over 16 years – a friend and colleague for whom I have the utmost respect and appreciation. In addition, I thank Danny Duke, M.S. and our team of research assistants for their hard work, especially while I was doing my residency at UCLA. I acknowledge other members of our research team, in addition to drs. Geffken and Silverstein and Mr. Duke: Drs. Laura B. Williams and Eric Storch. Last, but certainly not least, my thanks and love to my wife, parents, and family for their never-ending support and encouragement throughout six years of graduate school.
# TABLE OF CONTENTS

| ACKNOWLEDGMENTS | .................................................. | 4 |
| LIST OF TABLES | .................................................. | 7 |
| LIST OF FIGURES | .................................................. | 8 |
| ABSTRACT | .................................................. | 9 |

## CHAPTER

1 **INTRODUCTION** .................................................. 10

- Overview of Diabetes .................................................. 10
- Treatment and the DCCT .................................................. 13
- Non-Adherence to the TID Regimen .................................................. 16
- Relations between Adherence and Metabolic Control .................................................. 21
- Family Functioning, Adherence, and Metabolic Control .................................................. 23
- Rationale and Purpose of Study .................................................. 31

**Specific Aims and Analyses** .................................................. 33

- Scale Development .................................................. 33
- Preliminary Operations/Optimizing Scale Length .................................................. 34
- Confirmatory Models .................................................. 35
- Explanatory Factory Analysis .................................................. 37
- Reliability .................................................. 37
- Validity .................................................. 37
- Demographic Effects .................................................. 38

2 **METHOD** .................................................. 40

- Phase 1: Item Generation and Selection .................................................. 40
- Phase 2: Measure Evaluation (Including Psychometrics, and Hypothesis Testing) .................................................. 41
  - Participants and Procedure .................................................. 41
  - Measures .................................................. 42

3 **RESULTS** .................................................. 47

- Descriptive Analysis .................................................. 47
- Scale Development .................................................. 48
- Factor Analysis .................................................. 51
  - Confirmatory Models .................................................. 51
  - Explanatory Model .................................................. 53
- Analysis of Validity and Reliability .................................................. 54
- Supplemental Analysis .................................................. 57
  - Descriptive Analysis with the D-FAM .................................................. 57
Development and Analysis of Validity/Social Desirability Scale
Exploratory Analysis with Experimental Items

4 DISCUSSION

Limitations
Future Directions

LIST OF REFERENCES

BIOGRAPHICAL SKETCH
# LIST OF TABLES

<table>
<thead>
<tr>
<th>Table</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-1</td>
<td>Sample characteristics .................................................................46</td>
</tr>
<tr>
<td>3-1</td>
<td>Exploratory Factor Analysis of the Diabetes Family Adherence Measure (D-FAM) ......62</td>
</tr>
<tr>
<td>3-2</td>
<td>Item to Total Correlations and Cronbach’s a coefficient among D-FAM Scores. ..........62</td>
</tr>
<tr>
<td>3-3</td>
<td>Pearson Product Moment Correlations among D-FAM Scores ...............................63</td>
</tr>
<tr>
<td>3-4</td>
<td>Pearson Product Moment Correlations among D-FAM Scores and Child Age, Years with T1D. .................................................................63</td>
</tr>
<tr>
<td>3-5</td>
<td>Pearson Product Moment between D-FAM Scores, Metabolic Control, DKA, and Parent/Child Measures of Regimen Adherence.................................................................64</td>
</tr>
<tr>
<td>3-6</td>
<td>Pearson Product Moment between D-FAM Scores, Extant Measures of Family Functioning, and Externalizing Behavior Problems .................................................................65</td>
</tr>
<tr>
<td>3-7</td>
<td>Pearson Product Moment Correlations among D-FAM Scores and Test-Retest Scores ...66</td>
</tr>
<tr>
<td>3-8</td>
<td>Exploratory Factor Analysis of the D-FAM with Experimental Items ........................67</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>3-1</td>
<td>D-FAM Items Retained</td>
<td>68</td>
</tr>
<tr>
<td>3-2</td>
<td>Scree Plot for Exploratory Factor Analysis of D-FAM</td>
<td>69</td>
</tr>
<tr>
<td>3-3</td>
<td>Exploratory Four-Factor Model</td>
<td>70</td>
</tr>
<tr>
<td>3-4</td>
<td>Scree Plot for Exploratory Factor Analysis of D-FAM with Experimental Items</td>
<td>71</td>
</tr>
<tr>
<td>3-5</td>
<td>Final D-FAM with questions randomized.</td>
<td>72</td>
</tr>
<tr>
<td>3-6</td>
<td>Confirmatory Factor Analysis (CFA) Diagram for Final D-FAM 2-Factor Model.</td>
<td>73</td>
</tr>
</tbody>
</table>
My research developed and validated the Diabetes Family Adherence Measure (D-FAM), a comprehensive tool designed for the assessment of adherence-related parenting processes for children with type 1 diabetes (T1D). Despite the existence of other psychometrically-sound measures of diabetes family functioning, none capture multiple aspects of family functioning behaviors specific to diabetes management. Our development and evaluation of the D-FAM followed an empirical approach for scale design. One-hundred sixty-five children with type 1 diabetes and their families participated. A parsimonious and current assessment tool, consisting of 14-items (as well as 4 validity items) resulted. Scales of diabetes specific family functioning on the D-FAM were associated with both adherence and health status (HbA1c) as well as with extant scales of family functioning. Research suggests that improving adherence and metabolic control in children and adolescents with T1D must involve changes within the family system – our research suggests it to be a useful tool in evaluating family functioning related to adherence/metabolic control.
CHAPTER 1
INTRODUCTION

Overview of Diabetes

**Epidemiology.** Type 1 diabetes (T1D), often referred to as juvenile-onset or insulin-dependent diabetes, is a serious and costly chronic metabolic disorder. One of the most common chronic illness in children in the United States, T1D is more prevalent than all other childhood diseases combined (LaPorte, Matsushima, & Chang, 1995). Data from the 1960s to 1980s indicate that the prevalence of T1D in children less than 19 years of age averaged around 1.7 cases per 1000 (Cruickshanks et al., 1985; LaPorte et al., 1995). Laporte and colleagues estimated that over 123,000 children in the United States under the age of twenty have T1D (LaPorte et al., 1995). Further, the prevalence of T1D is increasing; approximately 30,000 new cases of T1D are diagnosed each year, and about 40% of those cases are under 20 years of age (Engelgau et al., 2004; Engelgau & Geiss, 2000).

Peak incidence occurs at puberty (Feingold & Funk, 1997). The incidence of T1D varies considerably across ethnicities. Overall, higher rates of T1D among Caucasian Americans compared to African, Asian, and Native Americans is documented (Kaufman, 1997; National Institutes of Health, 1995). Considerable geographical variation is also reported. The annual incidence in Shanghai, China is 0.7 per 100,000 whereas the incidence in Finland is fifty times greater (35.3 per 100,000) (LaPorte et al., 1995). The incidence in a comparable sample from the United States is 18.2 per 100,000 (Alleghany County, Pennsylvania; twenty-six times the annual incidence of Shanghai) (National Institutes of Health, 1995). Although age-of-onset varies, a pre-pubertal peak is well-documented (Engelgau et al., 2000).

**Etiology.** T1D is characterized by destruction of the insulin-producing pancreatic beta cells (β-cells) in the islets of Langerhans, leading to absolute insulin deficiency (Atkinson &
Maclaren, 1994). This is usually due to complete autoimmune destruction of the pancreatic beta cells (type 1A). The presence of islet-cell antigens or other autoantibodies (e.g., antibodies to insulin) in serum may be helpful if establishing if autoimmune processes are present (Roll, Christie, Standl, & Ziegler, 1994; Schatz et al., 1994). Idiopathic, or type 1B, refers to patients with no evidence of autoimmunity or other known cause for β-cell destruction (Libman, Pietropaolo, Arslanian, LaPorte, & Becker, 2003). Both genetic susceptibility and environmental factors are hypothesized to be involved with the pathogenesis of T1D (Kaufman, 1997). For example, the concordance rate of T1D development in monozygotic twins is 20-55%, with a 2-8% rate for a first-degree relative (parent or sibling) (Kyvik, Green, & Beck-Nielsen, 1995; LaPorte et al., 1995). However, 80-85% of individuals with T1D have no other affected family member (Dahlquist et al., 1985).

Researchers speculate that one or more environmental factors may trigger the onset of autoimmune or other destructive processes of the β-cells, including viral exposure and environmental toxins (Kaufman, 1997). This process likely occurs in genetically susceptible subjects and usually progresses over many months or years during which the subject may be asymptomatic and euglycemic. This long latent period is a reflection of the large number of functioning beta-cells that must be lost before hyperglycemia (high blood-glucose; one of the primary symptoms of T1D) occurs.

While T1D accounts for 5-10% of all diagnosed diabetes (Engelgau et al., 2000), type 2 diabetes is by far the most common type of diabetes (90%; National Institutes of Health, 1995) and is characterized by variable degrees of insulin deficiency and resistance, hyperglycemia, and relative impairment in insulin secretion. The prevalence of type 2 diabetes rises markedly with increasing degrees of obesity (Harris, 1989).
Diagnosis, clinical features, and adverse effects. Genetic markers for T1D are present from birth (Baughcum et al., 2005), immune markers are detectable after the onset of the autoimmune process (Schatz et al., 1994), and metabolic markers can be detected ensuing substantial β-cell damage, but before the onset of symptomatic hyperglycemia (McCulloch & Palmer, 1991). The diagnosis of T1D is typically straightforward. Most patients present with classic symptoms of hyperglycemia (polydipsia, polyuria, weight loss, ketonuria, ketonemia, visual blurring) (Silverstein et al., 2005). American Diabetes Association diagnostic criteria include a fasting blood glucose concentration of 126 mg/dL (7.0 mmol/L) or higher, or a random value of 200 mg/dL (11.1 mmol/L) or higher confirmed on another occasion (Alberti & Zimmet, 1998; American Diabetes Association, 2004; National Diabetes Data Group, 1979; Silverstein et al., 2005).

The absence of endogenous insulin, the hallmark of T1D, results in the inability of cells to metabolize sugars present in the blood stream. A result is marked elevation in blood-glucose concentration called hyperglycemia. In children, the most severe disease related risk of diabetes is diabetic ketoacidosis (DKA), a life-threatening event resulting from severe insulin deficiency/hyperglycemia and an accumulation of ketones in the blood (Feingold et al., 1997). Currently, DKA represents the most common cause of hospitalization and death in children with diabetes (Rosenbloom, 1990). For patients with T1D, DKA is responsible for approximately 10% of all diabetes-related deaths in individuals under the age of 45 (American Diabetes Association, 2001); the mortality rate is 1-3% of children with DKA in the United States (Scibilia, Finegold, Dorman, Becker, & Drash, 1986) – a high percentage given that DKA is almost always avoidable with sufficient exogenous insulin. Cerebral edema, an important factor with all DKA associated deaths and 20% of all diabetes-related mortality in childhood, is most
common among pediatric patients with DKA-related symptomology (e.g., severely dehydration, acidosis, and hyperosmolar) (Glaser et al., 2001; Silverstein et al., 2005). Recurrent DKA is extremely problematic and is often associated with the presence of psychopathology (Liss et al., 1998). In addition to DKA and other immediate adverse effects, chronic hyperglycemia can also occasion long term damage to multiple organ symptoms, including microvascular and macrovascular disease. Complications include retinopathy, hypertension, neuropathy, nephropathy, and cardiovascular disease (American Diabetes Association 2005a; American Diabetes Association 2005b).

Differentiating newly diagnosed T1D from type 2 diabetes has become more difficult as the incidence of type 2 diabetes has markedly increased among pediatric patients (Silverstein et al., 2005). In overweight children and adolescents, measures of islet autoantibody levels or plasma C-peptide levels may be useful for differential diagnosis (Silverstein et al., 2005). Clinically, patients with type 2 diabetes typically present with hyperglycemia but not DKA although some patients with type 2 diabetes develop DKA under certain circumstances (American Diabetes Association, 2004).

**Treatment and the DCCT**

Clinical practice recommendations state that the primary goal for treatment of T1D is to maintain near-normal (non-diabetic) blood-glucose levels (70-120 mg/100 mL) in order to avoid severe or frequent hyperglycemia or hypoglycemia (low blood sugar) (American Diabetes Association, 2005b). Achieving near normal blood-glucose levels requires insulin injections, frequent blood-glucose monitoring, regular exercise, and dietary adjustments. Given the inability to produce insulin, individuals with T1D require insulin via subcutaneous injections. Injected or infused insulin (i.e., via the insulin pump, a device that supplies insulin via a small dermal catheter) allows for the uptake of glucose from the blood-stream for use in cellular metabolic
processes. Presently, several different types of insulin are available; variants include approximate time of onset, peak activity, and duration of action. Typically, the basic requirements are a stable baseline dose of insulin (basal insulin; an intermediate or long-acting insulin or given via continuous subcutaneous insulin infusion) plus adjustable doses of pre-meal rapid-acting/very-rapid-acting insulin (American Diabetes Association, 2005b). Excess injected insulin can result in hypoglycemic reactions, a condition that can lead to mental confusion, and if left untreated, disorientation, coma, seizures, or death. Mild hypoglycemia is typically managed by oral glucose/food, whereas more severe cases may require intravenous dextrose solution or injected glucagon (a protein hormone, usually secreted by the pancreas, to stimulate the liver to produce glucose/increase the level of glucose in the blood). In addition to insulin, maintaining recommended blood-glucose levels requires frequent blood glucose monitoring (conducted via a finger-stick) as well daily exercise, dietary management (e.g., carbohydrate counting), and weight management (American Diabetes Association, 2005b).

These clinical practice recommendations are supported by extensive longitudinal research. Specifically, the Diabetes Control and Complications Trial (DCCT) established an empirical relationship between intensive insulin therapy and reduced complications (Diabetes Control and Complications Trial Research Team, 1986; Diabetes Control and Complications Trial Research Team, 1993; Diabetes Control and Complications Trial Research Team, 1994). This randomized, multisite, controlled clinical trial demonstrated that for patients with T1D, the risk of development or progression of diabetes related complications (including retinopathy, nephropathy, and neuropathy) is reduced 50-75% by intensive treatment regimens when compared to conventional treatment regimens (Zhang, Krzentowski, Albert, & Lefebvre, 2003). For example, Zhang and colleagues found that, among patients in the DCCT (ages 13-39) with
mean Hemoglobin A1c (HbA1c) \(^1\) \(\leq 6.87\%\) (‘good control’), 90% remained free of retinopathy (Zhang, Krzentowski, Albert, & Lefebvre, 2001). However, patients with a mean HbA1c \(\geq 9.49\%\) (‘poor control’), the complications developed in 57% of the patients. In a study of 195 adolescents, participants who were assigned an intensive therapy regimen (required individuals to: administer three or more injections per day or use of an insulin pump, adjust insulin based on blood-glucose levels, at least four blood-glucose tests per day, diet, and exercise) showed delayed onset and slower progression of retinopathy compared to individuals in a conventional therapy (one or two insulin injections and self-monitoring of blood glucose at least twice per day). Additionally, intensive therapy also reduced the risk of the development of microalbuminuria which is a precursor to the development of diabetes-related nephropathy (Diabetes Control and Complications Trial Research Team, 1994). Overall, a reduction in HbA1c by 1% was associated with a 15 to 30% decrease in the risk of microvascular and neuropathic complications of diabetes, highlighting the clinical significance of this group difference (American Diabetes Association, 2003).

In order to prevent long-term complications, the DCCT research group recommended intensive insulin and monitoring therapy for adults and adolescents as young as 13 years old (Diabetes Control and Complications Trial Research Team, 1993; Diabetes Control and Complications Trial Research Team, 1994). While intensive therapy reduces the risk of diabetes-related complications and HbA1c levels, it is a complex and demanding regimen for children and their families. Physicians prescribe complex daily regimens including insulin injections, blood-glucose monitoring, diet and exercise. These behavioral adjustments must be integrated into the patient’s daily routine.

---

\(^1\) Hemoglobin A1c (HbA1c) is an index of mean blood glucose over the past 2-3 months. Lower numbers indicate better metabolic control. Non-diabetic range is 4-6%; Values > 8% suggest poor control (Rohlfing et al., 2002).
Non-Adherence to the TID Regimen

Despite the omnibus findings of the DCCT, data support that children and adolescents are often noncompliant with many of these recommendations (Johnson, Silverstein, Rosenbloom, Carter, & Cunningham, 1986; La Greca, 1990). It is estimated that on average, 50 to 55% of chronically ill patients are non-adherent with their prescribed medical regimen (Gowers, Jones, Kiana, North, & Price, 1995; Rapoff, 1999; Rapoff & Bernard, 1991). However, the extent of nonadherence to the diabetes regimen is variable, ranging from 20-93% (McNabb, 1997). It has been established in a 9-year follow-up study by Kovacs and colleagues (1992) that 45% of adolescents engage in some significant form of noncompliance during adolescence (Kovacs, Goldston, Obrosky, & Iyengar, 1992). Other studies have found that 25% of a sample of 144 adolescents with T1D reported regularly missing daily injections (Weissberg-Benchell et al., 1995), the most important single component of the treatment regimen. Additionally, in an analysis of ninety-one psychological consultations received from a pediatric diabetes clinic, 62% were requested to address concerns related to poor adherence (Gelfand et al., 2004).

Each year, nonadherence with the treatment regimen leads to medical complications, hospitalizations and consequently higher healthcare costs (Cahill, Etzwiler, & Freinkel, 1976; Rapoff et al., 1991). The societal impact of medical non-adherence is substantial with estimated costs of non-adherence in the United States exceeding $100 billion per year (Rapoff, 1999). Further, poor adherence results in increased morbidity and mortality, as well as problematic medication use and excessive use of health care services (Lemanek, Kamps, & Chung, 2001; Quittner, Espelage, Ievers-Landis, & Drotar, 2002). Despite breakthroughs in diabetes treatment technology (e.g., insulin pumps, electronic blood-glucose meters) and empirical support for intensive insulin regimens, these advances are negated when recommendations are not followed. For instance, non-adherence can negatively impact clinical decisions made by health care
providers such as prescribing incorrect insulin doses. Thus as children and adolescents receive increasingly intensive and complex medical regimens, there is a growing significance of understanding adherence.

**Defining adherence.** Defining adherence (or compliance) with a medical regimen is difficult due to the multitude and complexity of the regimen behaviors (Johnson, 1993). In perhaps the best cited attempt, Haynes (1979, pp. 1-2) defined adherence/compliance as “the extent to which a person’s behavior (in terms of taking medication, following diets, or executing lifestyle changes) coincides with medical or health advice,” (Haynes, 1979). While the term compliance implies patient subservience to an external authority (i.e., what the doctor says), adherence, the preferred term, places greater emphasis on the patient’s role in choosing whether (or not) to follow a particular regimen (Johnson, 1993). The term self-management is also commonly used in discussion of medical regimen behaviors; the connotation of the term is what behaviors are actually done (La Greca, 1990). Regardless of the semantics, there are several implications of the Haynes’ definition of adherence including: (1) a focus on specific behaviors as a basis for the measurement of adherence, (2) the implication that adherence should be measured on a continuum rather than with the use of discreet categories (e.g., dichotomizing adherent vs. non-adherent), and (3) that adherence behaviors must be measured against a standard, such as general medical advice or specific advice provided to a patient by a medical provider (Rapoff, 1999).

**Measurement of adherence.** There is no universal agreement on explicit standards for measuring adherence; measures of adherence range on a continuum from direct to indirect, each offering several advantages and limitations (La Greca, 1990). Direct observation of regimen behaviors (behavioral observation) is a highly specific, unconfounded method of assessment.
Skinner (1938, p. 6) defined a behavior as “what an [individual] is doing or more accurately what is observed by another organism to be doing,” (Skinner, 1938). Adherence, and non-adherence, are behavioral components of diabetes management. Intuitively, the most accurate method of assessment should be observation of diabetes care behaviors. However, the behavioral observation approach is often unobtainable and impractical given that the complexity of the diabetes treatment regimen requires scrutiny of numerous behaviors over extended, laborious periods of observation. This method would be expensive, time-consuming, and relatively invasive to the family.

Indirect measures of adherence behaviors offer an alternative method of assessment. For example, health status indicators (e.g., HbA1c) are often used to quantify adherence. However, this approach does not involve any direct assessment of regimen behaviors. Instead, adherence is inferred based on a single laboratory value. This method confounds adherence and health status and provides no useful information regarding what the patient is doing that is consistent with the prescribed regimen (Johnson, 1993). Physician ratings of adherence are also problematic. First, physicians often have knowledge of the patient’s health-status indicators (e.g., HbA1c) and thus may also confound adherence and metabolic control. Second, the physician ratings of adherence are often global; this is problematic because diabetes adherence is likely a multivariate construct (McNabb, 1997; Reynolds, Johnson, & Silverstein, 1990). Additionally, physicians may be subject to biases (e.g., history of disease management, relative improvements) (La Greca, 1990).

Permanent product counts offer another indirect method of assessing adherence (e.g., drug assays, prescription recall, bottle/strip counts, blood-glucose meter downloads). However, filling a prescription or using a test-strip/syringe is not necessarily equivalent to following the regimen. For example, a child can inject his/her mattress with insulin (rather than his/herself), use diluted
urine on ketone strips, test a friend’s blood-glucose, etc. Further, many diabetes self-
management behaviors are not associated with permanent products.

A more commonly applied indirect approach involves obtaining patient self-reports or
parental reports of adherence, via paper-and-pencil or semi-structured interview (Anderson,
Auslander, Jung, Miller, & Santiago, 1990). However, La Greca (1990) cautions against making
generalizations from self-reports due to extensive variability in their detail and
comprehensiveness (La Greca, 1990). Thus reports from multiple informants (e.g., parents)
should be obtained in addition to patient self-reports. Problems with self- and parent-reports
include establishing an accurate time-frame, participant honesty (patients and parents are often
biased in the direction of over-reporting adherence), comprehension, and accuracy (Gonder-
(1986) developed a 24-hour recall interview to quantify adherence behaviors (Johnson et al.,
1986). Interviewers ask patients and parents to describe diabetes-management behaviors during
the previous 24-hour period over a series of three 20-minute structured phone interviews.
Thirteen adherence indices are obtained, several of which include glucose testing, insulin
administration, diet, and exercise constructs. Significant correlations between parent and child
reports have been documented in separate analyses with correlations ranging from .09 to .94
(Freund, Johnson, Silverstein, & Thomas, 1991; Reynolds et al., 1990; Spevack et al., 1987).
Although there are advantages to this interview (e.g., minimization of recall errors, high
reliability, and the ability to capture a non-reactive example of the child’s adherence), the 24-
hour recall interview is difficult to implement in a clinical setting, due to its reliance on multiple
interviews (3 child, 3 parent) and complex scoring system (McNabb, 1997). Required resources
to conduct interviews and analyze data (e.g. time and labor) are often not available to healthcare
providers. Additionally, despite the comprehensiveness of the 24-hour recall interview, strong associations between adherence and HbA1c were not supported in a longitudinal analysis of 186 children using the measure (Johnson, 1992).

Given the problems with laborious assessments such as the 24-hour recall approach, cost-effective/efficient methods for assessing adherence are necessary. The Self-Care Adherence Inventory (SCAI) (Hanson, Henggeler, & Burghen, 1987), a semi-structured clinician-rated interview to assess adherence with the T1D treatment regimen, is an alternative to the 24-hour recall interview (Hanson et al., 1996; Hanson et al., 1987). The interview is administered to the patient by an individual familiar with the requirements of the diabetes regimen. The SCAI content areas include glucose testing, dietary behaviors, insulin adjustment, and hypoglycemia preparedness. The authors found that the SCAI related to HbA1c all three times the measure was administered ($r_p = -0.28$, $-0.25$, and $-0.20$, $p < 0.001$). Harris and colleagues (2000) refined the SCAI and developed the Diabetes Self Management Profile (DSMP) (Harris et al., 2000). The DSMP is a 23-item structured interview that assesses five areas of diabetes management, including: insulin administration/dose adjustment, blood-glucose monitoring, exercise, diet, and management of hypoglycemia. The DSMP considers recent advances in diabetes treatment, such as rapid acting insulin and carbohydrate management. The DSMP is designed to assess diabetes self-management over the preceding three months (consistent with the latest HbA1c). The authors examined predictive validity by correlating the DSMP Total Score with HbA1c. Pearson’s correlations between HbA1c and the DSMP Total score were significant ($r = -0.28$, $p < 0.01$). Lewin and colleagues (2005) also found strong relations between the DSMP Total score and HbA1c ($r = -0.60$, $p < 0.01$, $r = -0.54$, $p < 0.01$ for child and parent report of adherence, respectively) (Lewin et al., 2005b).
A inherent confound in all of the aforementioned measurement techniques for adherence is that assessment of adherence often does not measure an individual’s behavior in comparison with his prescribed regimen but rather with an “ideal regimen” (La Greca, 1990). Unfortunately, ideal treatment standards are rarely conveyed adequately to the patient. For example, physician documentation of the regimen is often poor (Glasgow, Wilson, & McCaul, 1985; Johnson, 1993). If a patient’s prescribed medical regimen is not effective, even perfect adherence is not expected to link with good metabolic control. Plausible problems include: the ideal medical advise was not given, the family did not understand or remember the medical advise (as a function of poor communication/documentation), or the family/child chose not to follow any/all of the advise. Currently, there is no specific, standardized set of self-care behaviors applicable to all children with diabetes. It is difficult to measure adherence of behaviors that are not uniformly defined in operational terms (McNabb, 1997).

**Relations between Adherence and Metabolic Control**

The DCCT demonstrated that good metabolic control (via adherence to a strict insulin-injection and blood-glucose testing regimen) can delay or prevent the progression of diabetes related complications (Diabetes Control and Complications Trial Research Team, 1994). As medical practice focused on improving metabolic control, psychological researchers began using this test (HbA1c) more frequently as an outcome measure and began equating metabolic control with diabetes adherence. Given the intuitive relationship between adherence and health status (metabolic control), pediatric psychologists focus on changing health behaviors in patients with a history of poor metabolic control. Nevertheless, research has consistently failed to document a perfect relationship between metabolic control and adherence (Johnson et al., 1992; Kurtz, 1990). Johnson (1994) indicated that a perfect relation between health and behavior cannot be assumed (Johnson, 1994). For example, children reporting good adherence can be in poor
metabolic control and children reporting poor adherence can be in good metabolic control. However, 58% fell into the expected cells (reported good adherence/good metabolic control \([\text{HbA1c } \leq 7.7]\) and reported poor adherence/poor metabolic control \([\text{HbA1c } \geq 10.1]\)). Hence, the difficulty is explaining the “other” 42% that do not fit into the expected cells of the matrix (eighteen percent reported good adherence/poor metabolic control \([\text{HbA1c } \geq 10.1]\); 24% reported poor adherence/good control \([\text{HbA1c } \leq 7.7]\)). The “honeymoon” period (i.e., the period of time shortly after the diagnosis of T1D during which there is some residual insulin production and the blood sugar levels improve to normal, or near-normal, levels) is one plausible explanation for patients in good metabolic control despite poor adherence.

Other biological factors such as stress or pubertal status have also been postulated as potential mediators of the imperfect relationship between adherence and metabolic control (Johnson, 1994). Even harder to explain are children reported good adherence who are in poor metabolic control. Although factors such as dishonesty (see above) may explain this finding, other possibilities include inadvertent non-adherence (Johnson, 1994), i.e., the family misunderstands prescribed regimen behaviors/perform them incorrectly (inadvertently). Additionally, if a patient’s prescribed treatment regimen is not effective (e.g., incorrect insulin dosage or dietary plan), even perfect adherence (with a faulty regimen) should not favorably impact that individual’s metabolic control (Johnson, 1992). Nevertheless, clinicians hypothesize that adherence is a significant predictor of metabolic control. For example, data from a residential treatment program for youngsters in poor metabolic control demonstrate a significant mean reduction in HbA1c when the treatment regimen is delivered in a controlled environment (Geffken et al., 1997). Pilot data from treatment studies using telemedicine also suggest an
adherence-metabolic control relation (Gelfand, Geffken, Halsey-Lyda, Muir, & Malasanos, 2003; Heidgerken et al., 2005).

One explanation for this discrepancy might result from the measurement of adherence. As discussed above, most studies of adherence measure an individual’s behavior in relation to an ideal treatment regimen, failing to examine individualized prescriptions (La Greca, 1990; La Greca et al., 1995). Alternatively, Glascow et al. (1987) suggested that the relationship between adherence and metabolic control is not always straightforward, and regimen adherence must be examined in the context of other factors (Glasgow, McCaul, & Schafer, 1987). Research thus far has identified several variables associated with nonadherence, including regimen characteristics, disease characteristics, and family variables (La Greca et al., 1995; Rapoff et al., 1991). While regimen and disease characteristics have received much attention in the medical literature, family variables have received less focus. This is surprising given that children with diabetes identify parents as their most significant source of support (La Greca et al., 1995). Often models of adherence do not address regimen behaviors in the context of the family. However, experts agree that managing a pediatric chronic illness is not simply a matter of what the individual does – rather, it is a function of relationships within the family (Anderson & Coyne, 1993). When considering the complexity of diabetes management, it is intuitive to consider the familial environment in which adherence behaviors occur (Lewin et al., 2006).

**Family Functioning, Adherence, and Metabolic Control**

**General family functioning.** A body of research has found associations between general measures of family functioning (e.g., support, conflict, communication, and caring behaviors not specific to diabetes care) and metabolic control and adherence. Hauser and colleagues (1990) found that children’s perceptions of family conflict, measured by the Family Environmental Scale (FES; (Moos & Moos, 1981)), was the strongest predictor of poor adherence with multiple
diabetes treatment regimen components \((r = 0.50, p < 0.001)\) (Hauser et al., 1990). Likewise, reports of parental restrictiveness correlated with worse HbA1c (using the Parent Dimensions Inventory, a general measure of parenting style (Slater & Power, 1987)) (Davis et al., 2001). Another study found that child reports of parent-child conflict (measured by the Parent-Child Scales (Hetherington & Clingempeel, 1992)) contributes to the unique variance in predicting both adherence \((r = 0.50, p < 0.001)\) and metabolic control \((r = 0.31, p < 0.05)\) (Miller-Johnson et al., 1994). Conversely, the study suggested that both child and parent perceptions of family cohesion were related to improved adherence. Cohen et al. (2004) also found that better adherence and metabolic control linked to high levels of overall family cohesion, as measured using the Family Adaptability and Cohesion Evaluation Scales (FACES-III, (Olson, Portner, & Lavee, 1985)) (Cohen, Lumley, Naar-King, Partridge, & Cakan, 2004). Parental reports of warmth (using the Parent Dimensions Inventory) have been associated with better adherence (explained 27% of the variance in their children’s adherence) (Davis et al., 2001). Longitudinal data lends preliminary support for predictive validity: parent-child conflict, cohesion, and organization all related to adherence after one-year (Hauser et al., 1990). Data from a longitudinal study supports an association between family communication (using the FES) and improved metabolic control (Jacobson et al., 1990). Similarly, a child’s adjustment, as measured by an index on the Child Behavior Checklist (CBCL parent/child; (Achenbach, 1994)) related to adherence over a 4-year period (accounting for 47% of the variance in adherence) (Jacobson et al., 1990).

Although some researchers have found that found that both disease-specific and general family factors related to treatment adherence (Hanson, De Guire, Schinkel, Henggeler, & Burghen, 1992; McKelvey et al., 1989), many believe that family functioning behaviors that are
specifically related to diabetes care may be more strongly associated with adherence and metabolic control. Comparisons of the impact of illness-specific and general family functioning in the literature have shown inconclusive results although many experts believe that diabetes-specific family functioning is more strongly and consistently associated with adherence and metabolic control. For example, Liss et al. (1998) found that both children hospitalized with DKA and their parents reported less family support (specific to the diabetes regimen) than did a clinic control group (Liss et al., 1998). However, the Family Assessment Measure (Skinner, Steinhauser, & Santa-Barbara, 1983), a non-diabetes specific measure of family functioning, detected no differences in family functioning between the DKA and clinic control groups. Other studies (Dashiff, Bartolucci, Wallander, & Abdullatif, 2005; Gowers et al., 1995) have found no relation between HbA1c and general family functioning (e.g., using the McMaster Family Assessment Device (Epstein, Baldwin, & Bishop, 1983)). Moreover, Schafer et al. (1983) found that diabetes-specific family measures were more predictive of adherence than were more general measures of family interaction (mean correlation between general measures [e.g., the FES] of family interaction and adherence $M < 0.10$; mean correlation between diabetes specific measures and adherence $M = 0.18$) (Schafer, Glasgow, McCaul, & Dreher, 1983). Measures of family functioning that are specifically designed for families of children with diabetes (e.g., family support specifically related to diabetes care) may be more successful in detecting possible relationships between family functioning and metabolic control.

**Diabetes-specific family functioning.** Associations between aspects of family functioning (specific to diabetes care) and metabolic control are well-documented (Anderson & Laffel, 1997; McKelvey et al., 1993; Schafer et al., 1983; Waller et al., 1986). For example, positive parental emotional support (e.g., expressing understanding regarding the difficulties of
living with diabetes and the treatment regimen, relating to their child about having diabetes) is associated with improved metabolic control (Lewin et al., 2005a; Lewin et al., 2006; McKelvey et al., 1989; McKelvey et al., 1993). La Greca and colleagues (La Greca et al., 1995) found that diabetes-related parental supportive behaviors related to adherence with all components of the diabetes care regimen. Data from a longitudinal study also supports an association between family communication and improved metabolic control (Jacobson et al., 1994). Additionally, sufficient (but non-coercive) parental guidance with diabetes-related care tasks is positively correlated with improved diabetes health status indicators such as metabolic control (Lewin et al., 2005a; McKelvey et al., 1993; Waller et al., 1986).

Studies have also found that patients experiencing high levels of family conflict display poorer adherence or worse metabolic control (Klemp & La Greca, 1987; Miller-Johnson et al., 1994). More specifically, negative and unsupportive parental behavior patterns related to diabetes care behaviors (e.g., coercion, nagging, threats, criticism, and scolding) are correlated with both metabolic control and regimen adherence (Lewin et al., 2006; Schafer et al., 1983; Schafer, McCaul, & Glasgow, 1986). Further, in a study evaluating a parent-adolescent teamwork approach to diabetes management, families in the intervention group reported significantly less parent-child conflict related to diabetes management and were in better metabolic control (Anderson, Brackett, Ho, & Laffel, 1999). Family conflict is associated with worse metabolic control for both boys (Jacobson et al., 1994) and girls (Bobrow, AvRuskin, & Siller, 1985). It has been suggested that these factors be considered in selecting patients for insulin pump therapy (Williams, Storch, Lewin, Geffken, & Silverstein, 2005). Overall, family conflict specific to diabetes-care has been the factor most strongly related to both metabolic
Finally, appropriate parental supervision of diabetes care tasks is closely related to adherence and metabolic control. For example, Anderson and colleagues (Anderson et al., 1990) found that disagreements between parents and children regarding responsibility for diabetes related tasks predicted poor metabolic control. In addition, these researchers found that poorer metabolic control was positively correlated with families in which neither the parent nor child assumed responsibility for diabetes related tasks. Wysocki and colleagues (Wysocki et al., 1996) also examined families’ diabetes responsibility relative to the child’s developmental level. Results indicated that children reporting more diabetes management responsibilities demonstrated less adherence and worse metabolic control. Parents who are less involved have children who are less adherent with their treatment regimen, have children who make more mistakes in self-care, and have poorer metabolic control than children whose parents are involved in a developmentally appropriate style (Weissberg-Benchell et al., 1995; Wysocki et al., 1996). More recently, a study of 127 adolescents found that maternal un-involvement with a child’s treatment regimen was associated with poorer adherence and worse quality of life (Wiebe et al., 2005).

Consistent with the extant literature, studies have found that parental involvement was important across all ages (with regard to maximizing adherence) although the optimal level of involvement varies with age/developmental level (Anderson, Ho, Brackett, Finkelstein, & Laffel, 1997; Anderson et al., 1997). One major challenge is matching the child's emerging developmental desire for independence with appropriate parental supervision for disease related tasks. Research suggests that in T1D regimen nonadherence peaks during middle adolescence.
(Kovacs et al., 1992). Indeed, some evidence suggests that nonadherence may arise due to inappropriate transfer of disease related control from parent to adolescent (Gowers et al., 1995). Moreover, parents must gauge how to turn over responsibility to the adolescent during this transition. Although there is agreement in the literature that shifts of responsibility should be gradual, adolescents and parents are likely to approach the transferring of responsibility from different perspectives (Dashiff et al., 2005). Because adolescents desire more freedom and autonomy, whereas parents prefer more conventional perspectives related to compliance, these differing perspectives can result in diabetes-related parent-child conflict (Dashiff et al., 2005). Overall, better adherence has been documented when parents were seen as collaborating, not controlling when dealing with their adolescent’s diabetes-related problems (Wiebe et al., 2005).

Overall, these findings linking metabolic control to diabetes-specific family processes (such as parental involvement in diabetes tasks and the child’s perception of the valence of diabetes-related parental behaviors and support) highlight the importance of these constructs. Anderson and Laffel (Anderson et al., 1997) described diabetes-specific family functioning as critical constructs for assessment in order to optimize metabolic control and adherence outcomes. Although relations between individual family processes (e.g., responsibility, parental warmth, etc.) with metabolic control are relatively small, experts suggest that incorporating multiple diabetes-related family constructs might demonstrate a stronger connection with metabolic control (McKelvey et al., 1993). This premise was strongly supported. In a multivariate analysis, diabetes-specific family functioning (conflict, warmth, guidance/support, and parental responsibility) and adherence accounted for 49% of the variance in metabolic control (Lewin et al., 2006). This was the first study to examine all of these diabetes-related family processes concurrently. When combined, these diabetes family processes delineated a separate construct,
related to adherence, which indirectly influences metabolic control. Relations between diabetes-specific family factors and episodes of DKA have also been examined. Children in families with higher degrees of warmth-caring and less parental negativity were less likely to experience a DKA episode (Walker et al., 2004).

In the past, experts theorized that diabetes-specific family processes influence metabolic control indirectly through diabetes self-management (Hanson, De Guire, Schinkel, & Kolterman, 1995). Researchers suggested that family behaviors associated with adherence might mediate the relationship between family functioning and metabolic control (Kovacs, Kass, Schnell, Goldston, & Marsh, 1989). In other words, family behaviors related to diabetes management may facilitate or impede adherence with prescribed treatment and consequently affect metabolic control.

Research by Miller-Johnson et al. (Miller-Johnson et al., 1994) supported this premise; the study identified that ratings of parent and child conflict failed to contribute unique variance in metabolic control beyond that accounted for by adherence. Cohen and colleagues (Cohen et al., 2004) also suggested that family dysfunction may affect metabolic control indirectly, via effects on adherence behaviors. Although, data from this study did not support mediation, it is noteworthy that the measured family functioning processes in Cohen et al. (2004) (e.g., adaptability, cohesion) were general – not specific to diabetes. These findings suggest that further research is needed to verify mediation and to extend finding (e.g., Miller-Johnson et al., 1994) to other family processes related to diabetes adherence. An empirical study failed to document a mediating effect of adherence between general family functioning and metabolic control (Cohen et al., 2004). However, adherence was found to mediate associations between appraised collaboration (specific to diabetes) and metabolic control (HbA1c) (Wiebe et al., 2005). Moreover, adherence was found to mediate the relation between several diabetes-related
parenting behaviors (e.g., warmth, guidance, responsibility, and coerciveness) and health status (Lewin et al., 2006). In other words, the authors found that family-related diabetes management behaviors facilitated or impeded adherence with prescribed treatment and consequently affected metabolic control.

Finally, family and child stress should be considered when discussing family factors associated with adherence. Higher levels of stress and role-strain are consistently documented in children and parents of children with chronic illness relative to healthy children (Hauenstein, Marvin, Snyder, & Clarke, 1989; Quittner et al., 1998; Wagner, Cook, Chung, & Messig, 2003; Wiener, Vasquez, & Battles, 2001; Wysocki, Huxtable, Linscheid, & Wayne, 1989). A recent study of a psychology consult service for children with T1D in a tertiary clinic documented a high incidence of distress/internalizing disorders (Gelfand et al., 2004). Similar data were reported in a longitudinal study of youth with T1D (Kovacs et al., 1990b). Moreover, a study found that child-stress (assessed by a measure of life-changes) was linked to an index of health status (Brand, Johnson, & Johnson, 1986).

More recently, studies have focused on parenting stress associated with having a child with type 1 diabetes (Hauenstein et al. 1989; Streistand et al. 2006). Kovacs and colleagues (Kovacs et al., 1990a) document significant distress among mothers of youth with T1D in another longitudinal analysis. Additionally, for mothers of children with T1D, diabetes-specific stress also appears to be closely linked with child behavior problems. A recent study found that mothers of children with T1D, who also report externalizing behavior problems occurring with the child, report higher levels of disease-related stress (Lewin et al., 2005c). As with the assessment of family functioning, the importance of understanding stress specific to diabetes is being emphasized. Parenting stress assessment instruments used within pediatric psychology
Research are typically designed for children without chronic diseases (e.g., the Parenting Stress Index; PSI) (Abidin, 1995). Thus, normative data generated from parents of children generally free of medical illnesses are used to make inferences on distress levels of parents of medically ill children, which can be problematic. To address this limitation, Streisand and colleagues (Streisand, Braniecki, Tercyak, & Kazak, 2001) developed a measure of parental stress related to having a child with a chronic illness, the Pediatric Inventory for Parents (PIP). Understandably, parenting stress was found to be directly proportional to increased parental responsibility associated with parenting a child with a chronic illness (Streisand, Swift, Wickmark, Chen, & Holmes, 2005). However, there is a relative paucity in the literature regarding associations between parenting stress, family functioning, and adherence; future studies are needed to elucidate interrelations between these domains.

**Rationale and Purpose of Study**

Management of T1D requires a complex regimen of blood-glucose monitoring, insulin injections, dietary management, and appropriate exercise (American Diabetes Association, 2003). Further, the Diabetes Control and Complications Trial identified that good metabolic control (via adherence to a strict insulin-injection and blood-glucose testing regimen) can delay or prevent the progression of diabetes related complications such as including retinopathy, hypertension, neuropathy, nephropathy, and cardiovascular disease (Diabetes Control and Complications Trial Research Team, 1994). However, nonadherence with prescribed treatment is commonplace among youth with T1D (Weissberg-Benchell et al., 1995). The extant literature has demonstrated the strong association between diabetes-specific family function constructs and both adherence/non-adherence with the T1D regimen and health status (Abidin, 1995; Anderson et al., 1997; McKelvey et al., 1993; Miller-Johnson et al., 1994; Schafer et al., 1986; Waller et al., 1986).
Despite the existence of several measures of individual aspects of diabetes-related parenting (e.g., conflict, responsibilities, support-behaviors), there are no instruments designed to assess multiple domains simultaneously. Given the finding that as much as 49% of the variance in metabolic control (Lewin et al., 2006) and 44% of the variance in DKA (Walker et al., 2004) can be accounted for by assessing multiple parenting/family aspects, the development of a more practical, single assessment instrument appears warranted. This research proposes to develop a valid and reliable protocol for assessing disease-regimen related processes that may predict diabetes adherence and consequently metabolic control and DKA.

The objective of this research is to develop and validate the Diabetes Family Adherence Measure (D-FAM), a comprehensive measure of adherence-related parenting and family processes for youth with T1D. This measure will provide a tool for clinicians to assess family behaviors which (1) correspond with adherence to the diabetes treatment regimen, and (2) have been linked to diabetes-related health status (e.g., HbA1c and DKA). The technology of diabetes treatment regimen has increased exponentially over the past decade with the advent of the insulin pump, intensive insulin therapy, glargine and very rapid acting insulin, blood-glucose meters, carbohydrate counting, etc. It comes to reason that the resources of the family to cope with the increased complexity have become increasingly taxed. Given the complex, challenging, and often stress-inducing nature of diabetes treatment regimen, there is a clear role of psychology in helping family members understand and adjust behaviorally to the requirements of the child's diabetes regimen. Information gathered from a comprehensive assessment of family functioning (as assessed by the D-FAM) would be useful in treatment planning including examination of current perceptions of the child as well as changes over time. Further, learning about the psychometric properties of this measure, with respect to relations with measures of disease-
specific family processes and externalizing behaviors can lead to an understanding of the interrelations between these processes. Additionally, this instrument will have utility for evaluating treatment response to family systems-based intervention approaches for poorly controlled diabetes (Harris et al., 2000; Harris & Mertlich, 2003; Wysocki, Greco, Harris, Bubb, & White, 2001).

This study will examine the psychometric properties including reliability, factor structure, and validity of the D-FAM. Extant measures of diabetes-specific family functioning will administered for analysis of construct validity; these measures are selected based on previous empirical studies validating their utility.

**Specific Aims and Analyses**

**Scale Development**

There are various strategies employed in measure construction including logical/rational, theoretical, and empirical (Friedenberg, 1995; Worthington & Whittaker, 2006). The logical/rational approach involves the scale developer rationally deriving items that appear face-valid and obviously relate to the construct being measured. The theoretical approach applies extant psychological theories to generate scale items. However, both the aforementioned approaches have been superceded by a statistically rigorous, empirical approach where items are selected based on predictive utility for a criterion group and/or homogenous item groupings (Worthington et al., 2006).

Although the empirical approach to scale development generally includes the following steps, specific procedures and sequences may vary. No single model or sets of guidelines for questionnaire design “reflect a unitary best practice” (Worthington et al., 2006, pg. 811). However, experts agree on the following general procedural components: (1) determine the construct of interest, (2) determine the format of the measure, (3) generate a pool of items, (4)
have experts review the initial item pool, (5) consider inclusion of validation items, (6) administer items to a sample, (7) optimize scale length and, (8) evaluate items and evaluate scale(s) (Anastasi, 1988; Dawis, 1987; DeVellis, 2003; Worthington et al., 2006).

In the development of the D-FAM, the above guidelines for empirical scale development were employed. First, the construct of interest for the D-FAM, family adherence-related behaviors specific to T1D, is discussed at length in the preceding sections of this dissertation. Second, a five-response Likert scale format was chosen for the D-FAM based on similarity with previous measures with strong psychometric properties of disease-specific and general family functioning. Five response choices allow for sufficient variability to allow for correlation-based analyses without limiting meaningful discrimination between response-alternatives (Clark & Watson, 1995). Clark et al. (1995) recommends multiple-item Likert formats with an odd number of alternatives to allow a middle option. Third, experts in our research program developed the initial pool of items, and fourth, a nationwide panel of reviewers evaluated these items (see Methods below for a detailed procedure). Age-appropriateness of language and content was considered and less straightforward/confusing items were removed. Fifth, validation items were included to evaluate for uncommon response sets (evaluation procedures are discussed below in Results). Sixth, the D-FAM was administered to a relatively large sample of 165 children with T1D. The general statistical procedures are described below for steps seven and eight. Additional details and rationale are provided throughout the Methods and Results sections.

**Preliminary Operations/Optimizing Scale Length**

Commonly, three to four times the number of questions that will eventually be included on the final instrument are administered to the initial sample (Worthington et al., 2006). Consequently, item-to-total and inter-item correlations were calculated for each D-FAM item. In
general, items that did not correlate with the total score were evaluated and most were removed from subsequent analyses (DeVellis, 2003; Floyd & Widaman, 1995). Additionally, response variability of individual items was examined. Items with minimal response variation were withheld as these items are not likely to detect differences in study constructs (Clark et al., 1995; DeVellis, 2003; Floyd et al., 1995). (Please refer to a detailed discussion of criteria for item elimination in the Results section below.)

**Confirmatory Models**

The data for the D-FAM were examined for outliers and violations to the assumptions of multivariate normality. Although both confirmatory and exploratory factor analysis are relatively robust to violations of multivariate normality (Floyd et al., 1995), univariate normality was assessed for skewness and kurtosis (Kline, 1998). Confirmatory factor analyses (CFA) was computed for the D-FAM (based on *a priori* models) to examine the factor structure. The CFA allowed for examination of whether the data conformed with our pre-established theory specifically (specifically, evaluation of the number of factors and the loadings of measured variables on these factors). CFA allows for more flexibility (e.g., specifying that specific factors may correlate while not allowing others to correlate) than EFA; it is the preferred technique for the development of new scales and the reexamination of established measures (Klem, 2000). The models proposed for this study were as follows for the D-FAM: First, we examined a two-factor model based on research by Schafer and colleagues (recently replicated by our research program). This model poses distinct but related supportive and non-supportive diabetes-specific family-adherence factors. In the second model, it was hypothesized that the three latent constructs (parental warmth/caring/guidance, responsibility, and coercive parenting/conflict) would adequately define the general factor, Diabetes-Related Family Processes. This model was based on our previous research (Lewin et al., 2006) as well as data from other empirical studies.
(see above for a review). Additionally, a model consisting of a single, general latent factor of
diabetes-related parenting/family processes (measurement model) was evaluated using CFA.

Maximum likelihood estimation (MLE) was be used in order to maximize the probability
that the coefficient estimates have the greatest chance of reproducing the observed data. The fit
of each model was evaluated based on the following goodness-of-fit indices. The models were
evaluated with the following fit indices: $\chi^2$, the Jöreskog-Sörbom Goodness of Fit Index (GFI),
Adjusted Goodness of Fit Index (AGFI) the Bentler Comparative Fit Index (CFI), Bentler-Bonett
Normed Fit Index (NFI), and the Root Mean Square Error of Approximation (RMSEA)
(Bentler, 1990; Bentler & Bonett, 1980; Jöreskog & Sörbom, 1984; Jöreskog & Sörbom, 1993;
Marsh, Balla, & McDonald, 1988). The chi-square evaluates discrepancies between the
correlation matrix specified in the theoretical factor model(s) with correlation from the original
data matrix – better fit is suggested if any discrepancies in the model are small and occurred by
chance (implied in a non-significant chi-square) (Klem, 2000). Chi-square estimates that are
non-significant and/or less than twice the degrees of freedom, suggest adequate fit (Akaike,
1987; Bryant & Yarnold, 1998). GFI, AGFI, CFI, and NFI fit indices range from 0 to 1, with
values of .95 or higher indicating an adequate fit between the observed model and the theoretical
model (Bentler, 1990; Bryant et al., 1998; Chou & Bentler, 1993; Jöreskog et al., 1984;
Thompson, 2000). These indices utilize a variety of algorithms to evaluate how well the
proposed solution fits the data relative to a null model (where error explains item covariances)
(Bryant et al., 1998). Essentially, fit indices reflect the ability of the model to reproduce the
variance-covariance model (Thompson, 2000). For the RMSEA, values below .05 indicate a
good fit and values as high as .08 represent an adequate fit (Thompson, 2000).
Explanatory Factory Analysis

If the theorized models are not found to fit the data, exploratory factor analyses would be conducted to examine the factor structure of the data. Principal components analysis with promax/oblique rotation will be implemented in order to allow the factors to correlate; criteria for identifying the factors will be based on (1) Glorfeld’s (Glorfeld, 1995) version of parallel analysis; (2) the Minimum Average Partials (MAP) method (Velicer, 1976); and (3) the scree plot. This offers a more conservative estimation of retaining factors than the Kaiser Criterion/scree plot (O'Connor, 2000). Orthogonal and oblique (promax) solutions may be contrasted to account for the maximal percentage of the variance, although it is our expectations that the best solution will allow correlation between family factors (on the D-FAM). Subsequent CFA (see above) will be performed to determine the adequacy of fit of any exploratory model(s) generated in this step.

Reliability

To examine the reliability of the D-FAM, coefficient alpha was calculated for the total and subscale scores. Item-to-total correlations were examined for each subscale. Test-retest reliability was obtained for approximately 10-15% of the sample after two weeks.

Validity

Content validity for the D-FAM was assessed by a group of expert raters (see METHODS below). Items on which there was disagreement were discarded.

Concurrent validity (using Pearson product-moment correlations) was evaluated using previously validated measures of related constructs (e.g., the DFBC, DFBS, and DFRQ). Specific scales from the D-FAM cannot are not discussed until after the factor analyses. However, in general, we expected that higher levels of family functioning (on the D-FAM and/or subscales if applicable) will directly correlate with reports of parental warmth/caring and
guidance/control (on the DFBS) and will inversely correlate with reports of conflict (on the DFBC) and no-responsibility (on the DFRQ). Construct validity of the D-FAM was explored by examining the relations between the D-FAM and (1) HbA1c, (2) DSMP and SCAI (adherence reports), and (3) frequency of DKA. We expected that better family functioning would relate to better adherence and metabolic control. Similarly, relations between CBCL externalizing behaviors and the D-FAM were be evaluated given that externalizing behavior on the CBCL has been associated with worse metabolic control (Cohen et al., 2004).

Demographic Effects

Age-variations were examined on the D-FAM. As discussed above, we expected developmental changes in diabetes-specific family processes. For example, several analyses suggested that the relation between diabetes family functioning and adherence (Anderson & Laffel, 1997; Waller et al., 1986) and metabolic control (Lewin et al., 2006) varies with age. The relation between parental guidance/control and HbA1c has been found to be weaker in adolescents than in younger children (McKelvey et al., 1993; Waller et al., 1986). Conversely, age moderates the relation between parental coercive behaviors and HbA1c (with a strong association in adolescence) (Lewin et al., 2006). Thus, increased parent-child conflict (Wysocki et al., 2000) and decreased parental involvement (Anderson & Laffel, 1997) in diabetes self-care activities during adolescence was expected in this sample. Differences in family processes between adolescents and pre-adolescents (below age 13 years) was examined using t-tests. Similarly, both adherence and metabolic control relations with age are well documented (Johnson, 1992). Therefore, age variations in family functioning (as measured by the D-FAM)/metabolic control relations were also assessed, provided that age-D-FAM relations are identified. Baron and Kenny (1986) criteria for moderation will be applied. Although not
expected, gender and ethnicity differences on the D-FAM were also evaluated using paired t-tests and ANOVA respectively.
CHAPTER 2
METHOD

Phase 1: Item Generation and Selection

Diabetes and family adherence measure (D-FAM). One hundred items were generated by the author; approximately thirty questions in each of the three domains of diabetes-related parenting/family functioning that are most associated with adherence and metabolic control in the extant literature (conflict, support/guidance, and responsibility). A 5-point Likert scale is utilized with anchors from “never” to “always.” Fifty percent of the items were negatively phrased to discourage response sets (Comrey, 1988). Scoring was later reversed on these items for consistency. Items cover a three-month period of time, consistent with the laboratory HbA1c measurement (which provides an estimate of metabolic control over the preceding three months).

Subsequently, a panel of nine pediatric psychologists and three postdoctoral fellows (with expertise in diabetes), six pediatric endocrinologists/fellows, and ten diabetes nurse educators reviewed items for their appropriateness and content area. These professionals are employed at various academic medical centers across the country with affiliations/appointments in the respective diabetes programs, including: the University of Florida Health Science Center, Children’s Hospital of New Orleans, Vanderbilt University Children’s Hospital, Nemours Children’s Hospital (Jacksonville, FL), Northwestern University Medical Center, University of Maryland Medical Center (Baltimore), University of Alabama-Birmingham, and the Mount Washington Pediatric Hospital (Baltimore, Maryland). Written feedback was provided from the reviewers. Based on comments from these expert reviewers, the original 100 items were reduced and a 70-item D-FAM was developed. Included in this version are six items designed to assess careful responding/desire to appear favorably. Items include: “I check for ketones 6 times every
day;” I like having diabetes;” and “My blood sugar is always between 80 and 120.” Subsequently, the order of the questions was randomized.

**Phase 2: Measure Evaluation (Including Psychometrics, and Hypothesis Testing)**

**Participants and Procedure.**

A total of 165 pediatric patients with T1D (ages 8-18) and their primary caregivers were recruited from the University of Florida Pediatric Endocrinology Clinic in Gainesville, Florida. Families were compensated with a ten dollar gift-card (from Target, Wal-Mart or Blockbuster Video) for their participation (approximately 30-60 minutes of questionnaires). Medical staff approached eligible participants regularly scheduled clinic appointments. If the patient and his/her legal guardian provided assent, the investigator or his designee obtained informed consent from the parent/legal guardian (and the patient if age 18) and assent from the child participant (See UF IRB-01 Informed Consent, Appendix 1). Participation rate was approximately 92%. The modal indication for non-participation (among eligible patients; see criteria below) was time restriction; most of these families agreed to participate at a future appointment. The study principal investigator, co-investigators, or trained research assistants administered study measures to children and parents individually. Medical data such as HbA1c results and frequency of DKA will be obtained from the medical chart. Inclusion criteria were as follows: (a) ages 8 to 18 years, (b) a diagnosis of T1D for at least 1 year, (c) living with and accompanied by their primary caregiver, (d) ability to read and comprehend study materials and (e) no evidence of mental retardation or psychosis. Signed informed consent and assent, approved by the institutional review board, was obtained from each participant.

---

2 Funding made possible by a grant from the Children’s Miracle Network, the Geoffrey Clark Ryan Award, and the Society of Pediatric Psychology Routh Dissertation Honorable Mention awarded to Mr. Lewin.
The current sample consisted of 66 boys and 99 girls ages 8.0 to 18.75 years ($M = 13.5$, $SD = 3.0$). The ethnic distribution was 72.1% Caucasian, 16.4% African American, 9.1% Hispanic, and 2.4% representing other ethnic groups. Children participating in this study were from predominantly two-parent families (65.8%). More mothers (76.4%) participated in the study compared to fathers (15.2%) and other caregivers (8.4%). On average, participants had been diagnosed with diabetes for 4.8 years ($SD = 3.7$; range: 0.5-18 years). Mean HbA1c was 8.9% ($SD = 1.9$; range = 5-14%). Sixty-four and one-third percent of families reported that their child had at least one episode of DKA (mean = 1.3, $SD = 1.8$, range = 0-8). Twenty-five and one-half percent of the total sample reported multiple episodes of DKA; less than 6% reported three or more episodes. Data are presented in Table 2-1.

**Measures**

**Diabetes family adherence measure (D-FAM).** The DFAM is a 70-item (5-point Likert Scale) comprehensive measure of type 1 diabetes (T1D) adherence-related parenting and family processes (under development and evaluation in this study).

**Demographic information form.** This form obtains information such as age, sex, socioeconomic status (parental education, occupation, and income), duration of diabetes, frequency of DKA (Diabetes Ketoacidosis) and family composition.

**Diabetes family behavior scale (DFBS).** The DFBS is a measure of perceived family support completed by youth with T1D, Waller et al., 1986). Only the 15-item warmth/caring (e.g., “my parent understands how I feel about having diabetes”) and guidance/control (e.g., “my parent reminds me to test my blood sugar”) subscales were used due to the aims of this study. Participants responded to statements on a 5-point scale anchored by “all of the time” and “never.” Waller et al. (1986) reported good internal consistency ($a = .82$ for both scales) and
promising reliability (three-week test-retest reliability coefficients for the warmth/caring and guidance/control subscales were .79 and .83).

**Diabetes family behavior checklist (DFBC).** The DFBC is a child-rated measure of family support of the child’s diabetes self-care regimen (Schafer et al., 1986). Only the seven-item non-supportive family behavior domain was used. Children rated their parents on items such as how often does he/she “nag you about following your diet.” Items are scored on a 5-point Likert scale ranging from “never” to “at least once a day.” Schafer et al. (Schafer et al., 1986) reported acceptable internal consistency for this scale (α = .60). In a more recent study of 133 children with T1D, internal consistency for children (α = .79) and parents (α = .74) were acceptable (Lewin et al., 2005a).

**Diabetes family responsibility questionnaire (DFRQ).** The DFRQ assesses the family sharing of responsibilities concerning diabetes treatment (Anderson et al., 1990). Both the parent and child completed this measure individually by reading 17 statements concerning diabetes management tasks and indicating which family member accepts responsibility for that specific task (i.e., parent, child, or both). A parent-child dyadic no-responsibility score is calculated based on patterns of agreement and disagreement within the pair. Higher no-responsibility scores suggest that neither the parent nor child take responsibility for aspects of diabetes care. For example, if the child indicates that the parent is responsible for a care task (e.g., remembering to take insulin) while the parent indicates that the child is responsible, this item is scored on the No-Responsibility Taken index. Anderson et al. (1990) reported good internal consistency for the DFRQ (α = .85).

**The diabetes self-management profile (DSMP).** The DSMP is a 23-item structured interview with an administration time of approximately 15 minutes (Harris et al., 2000).
Questions assess five areas of diabetes management: insulin administration/dose adjustment, blood-glucose monitoring, exercise, diet, and management of hypoglycemia. Items were responded to in an open-ended manner and were coded by trained interviewers. The majority of the items are scored on a 5-point scale derived from the specific domain of the question (e.g., “always eats more or gives less insulin, frequently eats more or gives less insulin, sometimes eats more or gives less insulin, occasionally eats more or gives less insulin, eats less than usual or gives more insulin”). All items summed to produce a total adherence score. While there are no direct comparisons between the DSMP and other adherence measures in the extant literature, the predictive validity ($r = -.28$, $p < 0.01$; Harris et al., 2000) indicates that the DSMP accounts for a similar amount of variance in HbA1c compared to written self- and parent-report measures of adherence (e.g., the SCAI; Hanson, Henggeler, & Burghen, 1987). Additionally, a recent analysis found that child ($r = -.60$) and parent ($r = -.54$) reports of adherence using the DSMP were strongly correlated with HbA1c (Lewin et al., 2005b). Advantages of the DSMP interview over available self-report measures of adherence include assessment of up-to-date aspects of diabetes care such as carbohydrate counting. Harris et al. (2000) found good internal consistency ($a = .76$) and inter-observer agreement (94%).

**Self-care inventory (SCI).** The SCI is a 14-item self-report measure, assessing patients’ perceptions of their adherence to diabetes self-care recommendations over the previous 1–2 months (Greco et al., 1990; La Greca, Swales, Klemp, & Madigan, 1988). There are identical versions for adolescents and their parents. The authors report good internal consistencies and associations with metabolic control (Greco et al., 1990; La Greca et al., 1988).

**Child behavior checklist (CBCL).** Parent ratings of childhood affective and behavioral problems were assessed using the CBCL (113 item version for children ages 4-18), a widely used
measure of childhood externalizing and internalizing problems (Achenbach, 1994). Parents rate a series of behaviors on a 3 point scale (0 = not true, 1 = sometimes true, 2 = very true). Sound psychometric properties are available for this measure (Achenbach, 1994).

**Measurement of Metabolic Control.** While adherence is a behavioral construct, metabolic control is a biological assay of health status. The gold standard of measurement for diabetes metabolic control is the glycated hemoglobin A1c test (HbA1c). This HbA1c is a measure of glucose bound to the hemoglobin molecules within red blood cells (RBCs). The amount of glucose that is bound to hemoglobin is directly proportional to the concentration of glucose in the blood. Therefore, HbA1c provides an estimate of the average blood glucose concentration over the previous 8-12 weeks (RBCs live for approximately 90 days). Thus, HbA1c provides an estimate of metabolic (glycemic) control over the previous 2 to 3 months (American Diabetes Association, 2003). For youths with T1DM, findings from the DCCT suggest that the target range for good metabolic control is an A1c of less than 7.0% (American Diabetes Association, 2005a). Blood samples were analyzed using a Bayer DCA 2000+ and were collected as part of the patient’s routine medical care.
Table 2-1. Sample characteristics

<table>
<thead>
<tr>
<th>Characteristic (sample size = 165)</th>
<th>Value</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child Age (Mean + Standard Deviation)</td>
<td>13.5 +/- 3.0</td>
<td>8-18</td>
</tr>
<tr>
<td>Years since diagnosis (Mean + SD)</td>
<td>4.8 +/- 3.7</td>
<td>0.5-18</td>
</tr>
<tr>
<td>Child A1c (Mean + SD)</td>
<td>8.9 +/- 1.9</td>
<td>5-14</td>
</tr>
<tr>
<td>Child Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>66 (40%)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>99 (60%)</td>
<td></td>
</tr>
<tr>
<td>Child Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>119 (72.1%)</td>
<td></td>
</tr>
<tr>
<td>African American/African Descended</td>
<td>27 (16.4%)</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>15 (9.1%)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>4 (2.4%)</td>
<td></td>
</tr>
<tr>
<td>Parent/Guardian Relations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>126 (76.4%)</td>
<td></td>
</tr>
<tr>
<td>Father</td>
<td>25 (15.2%)</td>
<td></td>
</tr>
<tr>
<td>Grandmother</td>
<td>8 (4.8%)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>6 (3.6%)</td>
<td></td>
</tr>
<tr>
<td>Parent Marital Status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parents married</td>
<td>90 (54.5%)</td>
<td></td>
</tr>
<tr>
<td>Parent remarried</td>
<td>17 (10.3%)</td>
<td></td>
</tr>
<tr>
<td>Divorced/separated</td>
<td>43 (26.0%)</td>
<td></td>
</tr>
<tr>
<td>Single/never married</td>
<td>15 (9.2%)</td>
<td></td>
</tr>
<tr>
<td>Annual Family Income (Mean +/- SD)</td>
<td>46752 +/- 32377</td>
<td>$1200-$200000</td>
</tr>
<tr>
<td>Less than $25000 ( % of families)</td>
<td>26.2%</td>
<td></td>
</tr>
<tr>
<td>$25,000 - $50,000</td>
<td>32.7%</td>
<td></td>
</tr>
<tr>
<td>$50,000 - $75,000</td>
<td>27.6%</td>
<td></td>
</tr>
<tr>
<td>$75,000 - $100,000</td>
<td>10%</td>
<td></td>
</tr>
<tr>
<td>Over $100,000</td>
<td>3.5%</td>
<td></td>
</tr>
<tr>
<td>Education (mother)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some high school or less</td>
<td>19 (12%)</td>
<td></td>
</tr>
<tr>
<td>Completed high school</td>
<td>52 (32.9%)</td>
<td></td>
</tr>
<tr>
<td>Some college/trade school</td>
<td>39 (24.5%)</td>
<td></td>
</tr>
<tr>
<td>College degree or beyond</td>
<td>48 (18.2%)</td>
<td></td>
</tr>
<tr>
<td>Education (father)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Some high school or less</td>
<td>21 (14.9%)</td>
<td></td>
</tr>
<tr>
<td>Completed high school</td>
<td>47 (29.6%)</td>
<td></td>
</tr>
<tr>
<td>Some college/trade school</td>
<td>41 (25.8%)</td>
<td></td>
</tr>
<tr>
<td>College degree or beyond</td>
<td>32 (20.2%)</td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER 3
RESULTS

Confirmatory Factor Analysis was conducted using AMOS 5.0 (Arbuckle & Wothke, 2006) statistical software package. All other analyses were conducted using SPSS Version 14.0 (SPSS, 2006). The findings are presented as follows: (1) descriptive/sample characteristics; (2) Scale Development; (3) Factor Analysis; (4) Analysis of Reliability and Validity; and (4) Supplemental Analyses (D-FAM Demographics, Validity/Social Desirability Scale, and Experimental Item Analysis).

Descriptive Analysis

Demographic data are presented above and in Table 2-1. For this sample, mean HbA1c = 8.9 (SD = 1.9; range 5.0 – 14.0). On average, children in this sample had 1.3 episodes of DKA (SD = 1.8; range 0-10). Not surprisingly, worse metabolic control at the time of this study correlated with previous DKA ($r = .236, p < .001$). Similar to previous findings (Johnson et al., 1992), longer duration of T1D was linked to higher HbA1c ($r = .225, p < .001$). Additionally, greater age of the child was liked to more frequent DKA ($r = .19, p < .01$). Surprisingly, age was not associated with metabolic control in this sample ($r = .1, p = .19$). However, lower family income related to worse metabolic control ($r = -.2, p < .01$) and frequency of DKA ($r = -.17, p < .05$). Children residing in two-parent families (M = 8.5, SD = 1.9) had children with lower HbA1c’s than children in single parent families (M=9.4, SD = 1.9; $t(154) = 2.37, p < .01$). Parent marital status was not predictive of group differences in frequency of DKA, $t(154) = .9, p = .33$, child externalizing behavior, $t(154) = 1.1, p = .24$, or child report of adherence (adherence as assessed by the DSMP, child interview) $t(154) = -1.2, p = .22$). Parents from dual-parent families reported slightly greater adherence compared to single parent families $t(154) = -2.1, p < .04$ (adherence as assessed by the DSMP, parent interview). Not surprisingly, marital status was
also predictive of income; single parent families reported lower annual income (M = $31871, SD = 22341) than two-parent families (M = $54200, SD = 34142); t(163) = 4.2, p < .001. Consistent with previous data (Harris et al., 2000), metabolic control was inversely correlated with both parent (r = -.56, p < .001) and child (r = .37, p < .001) reports of adherence as assessed by the DSMP. Similarly, HbA1c was inversely correlated with both parent (r = -.41, p < .001) and child (r = .36, p < .001) reports of adherence as assessed by the SCAI. Externalizing behavioral problems (parent rated on the CBCL) was positively correlated with HbA1c (r = .25, p < .01) and negatively correlated with both parent (r = -.34, p < .001) and child (r = -.27, p < .01) reports of adherence as assessed by the DSMP. Gender and ethnicity were not predictive of differences in HbA1c, adherence, or DKA.

**Scale Development**

The procedure for content validity/selecting appropriate items for the D-FAM was described above (see: Method: *Item Generation and Selection*). Although the D-FAM questionnaire administered to this study sample was initially seventy questions, eight questions were designed as potential validity/social desirability indicators (Strahan & Gerbasi, 1972) and were excluded from factor analytical procedures. Further, the majority of the remaining items included multiple phrasing of similar content. Often, 3-4 times the number of questions that will be included in the final measure are administered to the initial sample (Worthington et al., 2006). During scale development, redundancy of items and over-inclusiveness is preferred to parsimony (DeVellis, 2003). Given that reliability can vary as a function of the number of items (Raykov & Marcoulides, 2000), we are hoping to gather the redundant content of interest from a pool of items (i.e., the construct) while their irrelevant idiosyncrasies (i.e., error) will be eliminated (DeVellis, 2003). Additionally, the redundancy of content allows for similar items to be evaluated for ease of comprehension, grammar, vocabulary, and other syntactic/semantic
dimensions (DeVellis, 2003). However, despite the benefits of over-inclusiveness during initial testing, the D-FAM is developed to be a succinct and valid (15-20 items) tool for clinical and research applications. Consequently, further item reduction was needed.

Statistical methodology further suggests the need for parsimony. Although some suggest that sample size for structural equation modeling (SEM) based approaches to confirmatory factor analysis (CFA; e.g., AMOS) requires at least 200 subjects (Baldwin, 1989), other experts suggest less stringent criteria. For example, Lomax (Lomax, 1998) suggested a sample size of at least 100 subjects; DeVellis (2003) suggested 150. Others suggest a 10:1 subject to measured/observed parameter (Mueller, 1997). Thompson (Thompson, 2000) suggested either 100 subjects or a ratio of at least 10:1 subjects to measured variables. Even less stringent, Bryant & Yarnold (1998) and Floyd & Widaman (1995) noted that 5-10:1 is a rough guideline for CFA (Bryant et al., 1998; Floyd et al., 1995). Consequently, with our sample size of 165, CFA with approximately 12-16 measured variables (questionnaire items) appeared consistent with both criteria.

Several considerations were included in eliminating items, such as redundancy, item-to-total correlations, item means, and reverse scoring. Given that items with means too near to an extreme of the response range on the Likert scale will have low variance, items with a mean closer to ‘3’ were preferred to items with means approaching ‘1’ or ‘5’ (DeVellis, 2003). Ten redundant items were removed based on these guidelines. Next, in order to discriminate among individuals completing the D-FAM, a degree of variance on each item is desirable (DeVellis, 2003). Highly unbalanced items (1) convey minimal information because most individuals answer the same and (2) their limited variability weakens correlations and thus factor analyses.
Consequently, items with minimal variability (greater than 75-95% responding on a single choice) were not retained (15 items) (Clark et al., 1995; DeVellis, 2003).

According to DeVellis (2004), the primary quality sought in each item is a high correlation with the latent construct(s). Although we cannot directly assess the relationship between each variable and the latent variable prior to CFA, we can assess inter-item correlations – the more reliable the individual items are to each other, the more related they are to the true score (DeVellis, 2003). In addition to examination of inter-item correlations, item-to-total correlations were examined (DeVellis, 2003). Items with weak (r < .2) (Cohen, 1977; Floyd et al., 1995) or non-significant inter-item and item-to-total correlations were removed (18 items). Similarly worded items (e.g., with highly overlapping content) were compared and those with lower item-to-total correlations were eliminated (DeVellis, 2003). Finally, in cases of duplication of content, negatively phrased items were retained to discourage response sets (Comrey, 1988). Five items with either borderline variability or inter-item/item-to-total correlations (that strongly matched the other criterion) were retained on the D-FAM as experimental items. Given that these items did not meet recommended criteria for CFA, they were excluded from these analyses.

Data for the remaining pool of 14 items were examined for outliers and violations to the assumptions of multivariate normality. Univariate normality was assessed through SPSS, which yielded measures of skewness ranging from -1.85 to 1.5 and measures of kurtosis, which ranged from -1.36 to 2.44. These values meet standards of univariate normality as outlined by (Kline, 1998). The data did not meet multivariate normality as assessed through AMOS procedures (kurtosis = 11.07, c.r. = 3.9). Given that confirmatory factor analysis is relatively robust to violations of multivariate normality, we proceeded with a maximum likelihood solution (Floyd et al., 1995). EFA will be completed subsequent to CFA to verify findings and/or identify
alternative factor structures (Worthington et al., 2006), especially multivariate normality assumptions.

Factor Analysis

Confirmatory Models

The relations between items were examined at the latent level using SEM based confirmatory factor analysis (CFA) in Amos 5.0 (Arbuckle et al., 2006; Osterlind & Tabachnick, 2000). As proposed above, three preexisting models were tested based on a priori theory to confirm or reject the existence of the specified factor structures. CFA allows for the investigator to: (1) accept or reject a single-formulated model based on a series of goodness-of-fit indices (discussed below); (2) select the strongest model from a series of alternative, theory-based models; or (3) a model is specified and repeatedly tested until acceptable fit is obtained (Joreskog & Sorbom, 1993; Raykov et al., 2000).

Two-factor model. The hypothesized two-factor model was specified representing an established theory of supportive and non-supportive parental behaviors (Lewin et al., 2005a; Schafer et al., 1986). The non-supportive and supportive factors were derived from the theorized factor structure of the measure. The two-factor structure resulted in excellent fit \( \chi^2 (df = 72, N = 165) = 61.4, p = .81; \) Jöreskog-Sörbom Goodness of Fit Index (GFI) = .95; Adjusted Goodness of Fit (AGFI) = .93; Bentler Comparative Fit Index (CFI) = .99, Bentler-Bonett Normed Fit Index (NFI) = .92; Root Mean Square Error of Approximation (RMSEA) = .01] (Bentler, 1990; Bentler et al., 1980; Jöreskog et al., 1984; Jöreskog et al., 1993; Marsh et al., 1988). The chi-square evaluates discrepancies between the matrix of correlations implied in the theoretical factor model with original data matrix – better fit is suggested if any discrepancies in the model are small and occurred by chance (implied in a non-significant chi-square) (Klem, 2000). For our two-factor solution, the chi-square estimate was both non-significant and less than twice the
degrees of freedom, suggesting adequate fit and the appropriateness of examining the goodness-of-fit estimates (Akaike, 1987; Bryant et al., 1998). Goodness-of-fit indices such as GFI, AGFI, NFI and CFI use a variety of algorithms to evaluate how well the proposed solution fits the data relative a null model, where error alone explains item covariances (Bryant et al., 1998). Essentially, fit indices reflect the ability of the model to reproduce the covariance matrix of the actual data (Thompson, 2000). Values above .90 are expected for correctly specified models (values range from 0-1 with 1 suggesting perfect fit) (Bentler, 1990; Bryant et al., 1998; Chou et al., 1993; Jöreskog et al., 1984; Thompson, 2000). Finally, the RMSEA suggests a strong fit. Values approaching zero are desired and a value less than or equal to .08 suggest reasonable error of approximation (Thompson, 2000).

**Single-factor model.** A single latent factor model was tested, to determine whether supportive/non-supportive behaviors were two extremes on a single continuum. CFA for this parsimonious model resulted in poor fit $[\chi^2 (df = 73, N = 165) = 222.1, p < .0001; \text{Jöreskog-Sörbom Goodness of Fit Index (GFI)} = .77; \text{Adjusted Goodness of Fit (AGFI)} = .67; \text{Bentler Comparative Fit Index (CFI)} = .75, \text{Bentler-Bonett Normed Fit Index (NFI)} = .64; \text{Root Mean Square Error of Approximation (RMSEA)} = .11]$ (Bentler, 1990; Bentler et al., 1980; Jöreskog et al., 1984; Jöreskog et al., 1993; Marsh et al., 1988).

**Three-factor model.** Finally, a three-factor model was tested, based on a priori theory that supportive, non-supportive, and parent-involvement/responsibility represent separate family factors related to adherence (Anderson et al., 1990; Lewin et al., 2006; McKelvey et al., 1993; Walker et al., 2004; Waller et al., 1986). CFA for the three factor model resulted in modest fit $[\chi^2 (df = 84, N = 160) = 94.81, p < .19; \text{Jöreskog-Sörbom Goodness of Fit Index (GFI)} = .92; \text{Adjusted Goodness of Fit (AGFI)} = .89; \text{Bentler Comparative Fit Index (CFI)} = .89, \text{Bentler-
Bonett Normed Fit Index (NFI) = .87; Root Mean Square Error of Approximation (RMSEA) = .07] (Bentler, 1990; Bentler et al., 1980; Jöreskog et al., 1984; Jöreskog et al., 1993; Marsh et al., 1988).

**Model selection.** Data from CFA support a 2-factor structure for the D-FAM (Supportive and Non-Supportive Scales). There is inadequate support for either single factor or three-factor models. Please see Figure 3-1 for scale items and Figure 3-6 for CFA diagram.

**Explanatory Model**

An exploratory factor analysis (EFA) was for supplemental support of the identified model (Worthington & Whittaker, 2006). A principal component factor analysis with promax rotation was performed to determine the optimal factor structure for this sample. Using a promax rotation allowed the factors to correlate with each other. Criteria for identifying the factors were based on (1) Glorfeld’s version of parallel analysis with a sample size of \( N = 165 \) and \( k = 14 \) variables (eigenvalues must be greater than 4.5 eigenvalues for the first component, 1.9 for the second component for the D-FAM, using the 95\(^{th}\) percentile and 1000 replications); (2) the Minimum Average Partials (MAP) method (Velicer, 1976); and (3) the scree plot (see Figure 3-2) (Zwick & Velicer, 1986). The minimum average partials calculation provides a more conservative criteria for determining the number of factors as compared to the Kaiser criteria/scree plot estimation (O'Connor, 2000). Syntax for Velicer’s MAP test (O'Connor, 2000) indicated retainment of two components. EFA identified a two-factor solution accounting for 48% of the variance (eigenvalues were 4.6 and 2.0). Seven items loaded on the first factor and seven items loaded on the second factor (see Table 3-1). To maximize reliability of factors, loading greater than .3 were required, although most item loadings exceeded .6 (Floyd et al., 1995; Zwick et al., 1986). On the Non-Supportive scale, factor loading range from .382-.822 with only one value
less than .60. On the Supportive scale, factor loadings range from .364-.812 with only one value less than .50. These factor loadings suggest stability of the scales. Specifically, Floyd & Widaman (1995) described factor loadings of .80 solutions to be highly stable across replicated samples, regardless of the number of subjects; factor loadings of .60 were highly stable if obtained from sample sizes of 150 or greater. Given (1) our sample size of 165 children combined with (2) most factor loadings being greater than .60 (with several over .80), this factor solution is likely to be stable. Communalities (i.e., the variance each item shares with the latent variable) are adequate for this model (.2-.4 are adequate; .6 or higher are excellent) (Cohen & Cohen, 1983; MacCallum, Widaman, Zhang, & Hong, 1999). Consistent with rationally derived scales, tested in the CFA, EFA provides strong support for the \textit{a priori} 2-factor model.

\textbf{Analysis of Validity and Reliability}

Please see Figure 3-3 for the final items. Only the fourteen items in the Supportive and Non-Supportive scales are included in subsequent analyses unless clearly noted (e.g., validity/social desirability scale). Data are recoded such that higher scores reflect more supportive behavior on the Supportive Scale and less non-supportive behavior on the Non-Supportive Scale. Despite the relatively poor fit of a single factor model, a total score is included, for comprehensiveness of reporting, in the analyses below (higher scores suggest more support).

\textbf{Internal Consistency.} The internal consistency of the D-FAM scores was evaluated using Cronbach's \(a\) coefficient (Cronbach, 1951). Alpha’s of .80-.90 are considered excellent; .60’s and .70’s are characterized as good or adequate (Clark et al., 1995). Good internal consistency was found for the D-FAM total score \((a = .85)\). Similarly, adequate internal consistency was also found for D-FAM non-supportive \((a = .82)\), and supportive scales \((a = .79)\).
**Convergent Validity.** Table 3-3 presents inter-correlations between D-FAM subscale scores. The resulting correlation coefficient (r = .43, p < .01) suggests separate yet related constructs. Pearson’s product-moment correlations between D-FAM scores and extant measures of diabetes-specific family functioning are presented in Table 3-6. Please note: data on the D-FAM Non-Supportive scale are recoded such that higher scores reflect lower reports of parental negativity – thus higher scores on the D-FAM total score may (in part) also be accounted for by lower negativity. The interscale correlation between the Non-Supportive and Supportive scales was .43 (p < .001), suggesting that the scales represent unique yet related dimensions. The interscale correlations between the Non-Supportive scale and the D-FAM total score and Supportive scale and the D-FAM total score were .86 (p < .001) and .83 (p < .001) respectively. Table 3-6 also presents correlations between the D-FAM and subscales with the DFBC Negativity Scale (child and parent reports), the DFBS Warmth/Caring and Guidance/Control subscales (child report), the DFRQ No-Responsibility Index (parent-child dyad score), and the CBCL Externalizing Scale (parent report).

Strong and significant associations were identified between the D-FAM and the DFBC. More family negativity reported on the D-FAM Non-Supportive Scale was strongly associated with parental negativity reported on the DFBC (both Child and Parent Reports). Similarly, reports of more supportive family behaviors on the D-FAM Supportive Scale were associated with less parental negativity on the DFBC (both Child and Parent Reports). Relations between the D-FAM and the DFBC were stronger for the child report of the DFBC. Weak, yet significant correlations were identified between the D-FAM Non-Supportive Scale and the DFBS Warmth/Caring and Guidance and Control Scale (less parental negativity related to more parental Warmth/Caring and more Guidance/Control). Stronger, positive associations were
found between the D-FAM Supportive scale and the DFBS Warmth/Caring Scale. More family negativity on the D-FAM Non-Supportive scale related to higher reports of no-one assuming responsibility for diabetes care tasks on the DFRQ. Finally, we identified a moderate association between higher reports of family negativity of the D-FAM Non-Supportive scale and child externalizing behavior problems reported by the parent on the CBCL.

**Construct Validity.** Table 3-5 presents correlations between the D-FAM, metabolic control (HbA1c), frequency of diabetes ketoacidosis (DKA) and parent/child reports of adherence (DSMP interview-format and SCAI self/parent-report format). Increased negative family behaviors on the D-FAM Non-Supportive scale related to worse metabolic control and increased episodes of DKA. Similarly, higher supportive family behaviors reported on the D-FAM Supportive scale correlated with better metabolic control. The D-FAM Supportive scale did not correlate with DKA. Both D-FAM Non-Supportive and Supportive scales were associated with parent and child reports of adherence on both individual interviews (DSMP Parent and Child) and self/parent report questionnaires (SCAI Parent and Child). Of note, D-FAM scores were more strongly associated with parent reports of adherence as compared to child reports of adherence. However, Fisher’s r to z tests (used to compare the magnitude of correlations) (Cohen et al., 1983) showed the only significant difference in the magnitude of the correlations between D-FAM and adherence measures occurred between the D-FAM Non-Supportive scale with child report of adherence (DSMP) and D-FAM Non-Supportive scale with parent report of adherence (DSMP), z = -1.3, p < .05.

**Test-Retest Reliability.** Two-week test-retest data were collected for 17.5% (N = 28) of subjects participating in this research. Approximately 30% of participants were asked to complete test-retest data. Two refused, citing time constraints as their reason. Thus,
approximately 60% of participants whom were asked to complete test-retest data and agreed returned the measures within the two-week period. Data postmarked after the two-week period were excluded to minimized error. MANOVA procedures were then used to analyze differences between subjects completing test-retest data and the overall sample. The resulting MANOVA was non-significant for group difference with a Wilk’s Lambda of $F(52, 165) = .93, p = .65$ indicating that completers of the test-retest data did not differ, relative to the overall sample, on HbA1c $[F(13, 165) = .01, p = .94]$, age $[F(13, 165) = .16, p = .89]$, DKA $[F(13, 165) = 2.5, p = .15]$, duration with diabetes $[F(13, 165) = .45, p = .5]$, family income $[F(13, 165) = 1.2, p = .26]$, child report of adherence (DSMP) $[F(13, 165) = .04, p = .85]$, parent report of adherence (DSMP) $[F(13, 165) = 1.2, p = .27]$, D-FAM Non Supportive score $[F(13, 165) = 1.3, p = .30]$, D-FAM Supportive score $[F(13, 165) = 2.0, p = .16]$ and D-FAM Total Score $[F(13, 165) = 2.1, p = .15]$. Strong, positive correlations between the original administration and the retest administration were identified for each D-FAM scale as well as for the total score (see Table 3-7). Similar to the initial administration, good internal consistency (Cronbach's a coefficient) of D-FAM scores was identified evaluated (Cronbach, 1951): D-FAM total score ($a = .82$); D-FAM Non-Supportive ($a = .87$); and D-FAM Supportive scales ($a = .75$).

Supplemental Analysis

Descriptive Analysis with the D-FAM

Age was negatively associated with family support on the D-FAM (see Table 3-4). Older children reported lower support and more non-supportive family behaviors. T-tests identified significantly higher D-FAM total scores for preteens (M=54.8, SD=11.2) compared to teenagers (M=49.2, SD=9.5), $t(165) = 3.5, p<.001$, higher D-FAM Non-Supportive scale scores for preteens (M=27.2, SD=6.9) compared to teenagers (M=24.4, SD=6.1), $t(165) = 2.9, p<.003$, and higher D-FAM Supportive scale scores for preteens (M=27.6, SD=6.5) compared to teenagers.
(M=24.8, SD=5.6), t(165) = 2.8, p<.006. Similarly, children with a longer duration of T1D reported more non-supportive family behavior (see Table 3-4). Gender differences on D-FAM scales were not identified using t-tests. Similarly, ANOVA did not identify differences based on ethnicity in D-FAM scores. The D-FAM Total Score was weakly correlated with family income (r=.2, p<.05); subscale scores were not correlated with income. D-FAM Positive scores were lower for children of single parent families (M = 24, SD = 7.3) than two-parent families (M = 27 SD = 5.4); t(163) = 2.6, p < .05. D-FAM Non-Supportive scores were not significantly lower (at the alpha = .05 level) among children of single parent families (M = 24, SD = 6.5) compared to children of two-parent families (M = 26, SD = 6.5); t(163) = 1.7, p = .07. Family income was not correlated with D-FAM scores.

Baron and Kenny’s (1986) guidelines for moderation were followed to examine whether child’s age moderated the relation between D-FAM Supportive and Non-Supportive Scores and metabolic control. According to these criteria, moderation is met if there is a significant interaction between the moderator (i.e., age) and a family functioning variable (e.g., D-FAM Support) after the effects of both the moderator and family functioning variables are controlled (Baron & Kenny, 1986). Moderation was tested separately for both D-FAM scales using hierarchical regression with metabolic control as the dependent variable. Child age and a family variable were added next as predictors into the regression, and the age by family functioning interaction was added into the final block. Neither family functioning by age interaction was significant, suggesting that age does not moderate metabolic control-D-FAM relations with this sample; Non-Supportive \( F (1,165) = .45, p = .5; \beta = -.28, p = .5 \) and Supportive \( F (1,165) = .12, p = .7; \beta = .01, p = .7 \). In fact, when the sample was dichotomized, strong correlations were found between HbA1c and D-FAM Scores in both groups, teenagers (age 13 and above, \( N = 92 \)
and preteens (under age 13, $N = 73$). Specifically, for D-FAM Non-Supportive scores, $r = .3, p < .01$ for teenagers and $r = -.5, p < .01$ for preteens. For D-FAM Supportive $r = -.33, p < .01$ for teenagers and $r = -.53, p < .01$ for preteens scores. Fisher's $r$ to $z$ tests failed to show significant differences in the magnitude of the correlations ($p = .13$ and .12 for Non-Supportive and Supportive scales respectively). Age 13 was selected as a cutoff for the moderator analysis in this sample was based trends from the extant literature (see Johnson et al., 1992) and clinical expectations. For example, Wysocki and colleagues described this age to be when parent-child conflict often increases (Wysocki et al., 2000). Further, Anderson and Laffel (1997) suggested that diabetes management responsibilities shift from the parent to adolescent at this age.

Development and Analysis of Validity/Social Desirability Scale

Eight original questions were designed as potential validity/social desirability indicators (Strahan et al., 1972). Unlike scale items, validity items were selected based on non-normal distribution patterns (Devellis, 2003). Specifically, items with less than 10% of our sample responding on the high-end anchor were retained (four items). The items are as follows (percentage of children endorsing “Always” is listed in parentheses): I always eat exactly what I should every time (7.9%); I’m glad I have diabetes (7.3%); My blood sugar is always between 80 and 120 (3.6%); and I check ketones 6 times every day (1.8%). Only 4.24% percent of the sample endorsed “Always” on two of these four items. Coefficient alpha was low for this scale (Cronbach’s $a = .38$), which is not surprising given the short length of the scale, variability of content, skewed distribution of responses, and low overall responses. Additionally, test-retest reliability was low ($N = 28; r = .21, p = .5$) which is also not surprising given that endorsing “Always” on these items was markedly atypical. It is noteworthy that only one child who endorsed “Always” on two Validity/Social Desirability scale items completed test-retest data; only one item was endorsed on the second administration.
While there is insufficient power to compare group differences between children who endorse atypical items versus the remainder of the sample, qualitative differences emerge. Specifically, HbA1c is worse (Mean = 9.5 compared to 8.8); more externalizing behavioral problems are reported (Mean = 12 compared to 8); and parents report lower adherence on interview (Mean = 48 compared to 59). As expected, children who respond in an atypically favorable pattern on D-FAM validity/socially desirability items report better family functioning across the D-FAM (Supportive: M=34.3 compared to 25; Non-Supportive: M=34.5 compared to 26; Total Score: M=69 compared to 51.7). See Figure 3-5 for complete D-FAM with validity and experimental items.

**Exploratory Analysis with Experimental Items**

Although beyond the scope of the proposed dissertation, an EFA was conducted with the 14 D-FAM items as well as the four experimental items that were not included in any of the analyses above (see Figure 3-1). As reported above, these items barely missed criteria for inclusion. Thus, this preliminary analysis was conducted to identify their influence on the extant factor structure. A promax/oblique rotation allowed the factors to correlate with each other. Criteria for identifying the factors were based on (1) Glorfeld’s version of parallel analysis with a sample size of \(N = 165\) and \(k = 18\) variables (eigenvalues must be greater than 5.0 eigenvalues for the first component, 2.5 for the second component, 1.3 for the third component and 1.0 for the fourth component using the 95\(^{th}\) percentile and 1000 replications); (2) the Minimum Average Partials (MAP) method (Velicer, 1976); and (3) the scree plot (see Figure 3-4) (Zwick et al., 1986). Syntax for Velicer’s MAP test (O’Connor, 2000) indicated four components. EFA identified a four-factor solution accounting for 63\% of the variance (eigenvalues were 5.1, 2.5, 1.4, and 1.3). Seven items loaded on the first factor, five items loaded on the second factor, four items on the third factor, and three items on the fourth factor (see Table 3-8 and Figure 3-3).
first factor was identical to the Non-Supportive scale validated in the CFA and EFA above. Factor loading ranged from .34-.88 with only two values less than .5. The second item contained five items on the D-FAM Supportive scale described in the sections above. Factor loadings range from .671-.881. The next factor contained items that can be described as Parental Control (e.g., my parents decide how much insulin I take or they do the carb counting for me). Factor loading were high, ranging from .754 to .812. The final factor contained items that are described as Parental Monitoring (e.g., my parents watch me draw up my insulin, my parent looks at my ketone strip). Loadings were also high for this factor, ranging from .525 to .928 (Floyd et al., 1995). Communalities are strong for this model (.2-.4 are adequate; .6 or higher are excellent) (Cohen et al., 1983; MacCallum et al., 1999). Regardless of EFA findings, subsequent CFA did not support this factor structure. CFA for this resulted in marginal fit $\chi^2 (df = 113, N = 165) = 122.9, p = .25$; Jöreskog-Sörbom Goodness of Fit Index ($GFI$) = .92; Adjusted Goodness of Fit ($AGFI$) = .89; Bentler Comparative Fit Index ($CFI$) = .98, Bentler-Bonett Normed Fit Index ($NFI$) = .87; Root Mean Square Error of Approximation ($RMSEA$) = .02] (Bentler, 1990; Bentler et al., 1980; Jöreskog et al., 1984; Jöreskog et al., 1993; Marsh et al., 1988). It is noteworthy that despite a strong EFA and conceptual support for this four-factor structure, additional subjects are necessary for validation. Nevertheless, it is presented as a potential direction for future analysis with the D-FAM.
Table 3-1. Exploratory Factor Analysis of the D-FAM

<table>
<thead>
<tr>
<th>Item</th>
<th>Factor Loading Non-Supportive</th>
<th>Factor Loading Supportive</th>
<th>Communalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>I get yelled at if I forget my insulin</td>
<td>.765</td>
<td>-.060</td>
<td>.64</td>
</tr>
<tr>
<td>I’m afraid to talk about diabetes with my parents</td>
<td>.382</td>
<td>.140</td>
<td>.26</td>
</tr>
<tr>
<td>My parents and I argue about diabetes</td>
<td>.621</td>
<td>.103</td>
<td>.45</td>
</tr>
<tr>
<td>My parents nag me to test my blood sugar</td>
<td>.707</td>
<td>-.057</td>
<td>.47</td>
</tr>
<tr>
<td>My parents get mad if I forget my insulin</td>
<td>.822</td>
<td>-.054</td>
<td>.64</td>
</tr>
<tr>
<td>My parents blame me when my blood sugar is too high</td>
<td>.731</td>
<td>.005</td>
<td>.57</td>
</tr>
<tr>
<td>My parents nag me for not taking care of my diabetes</td>
<td>.685</td>
<td>.001</td>
<td>.50</td>
</tr>
<tr>
<td>My parents do the carbs counting for me</td>
<td>-.061</td>
<td>.364</td>
<td>.20</td>
</tr>
<tr>
<td>My parents look at my meter’s log to see what my blood sugars were</td>
<td>.121</td>
<td>.503</td>
<td>.31</td>
</tr>
<tr>
<td>My parents tell me when I do a good job taking care of my diabetes</td>
<td>.181</td>
<td>.510</td>
<td>.36</td>
</tr>
<tr>
<td>I can talk to my parents about having diabetes</td>
<td>.112</td>
<td>.748</td>
<td>.64</td>
</tr>
<tr>
<td>My parents pay attention when I ask for help with diabetes</td>
<td>-.042</td>
<td>.812</td>
<td>.63</td>
</tr>
<tr>
<td>If I have a problem taking care of my diabetes, my parents will help</td>
<td>-.086</td>
<td>.802</td>
<td>.62</td>
</tr>
<tr>
<td>My parents explain diabetes to my teachers</td>
<td>-.077</td>
<td>.779</td>
<td>.61</td>
</tr>
</tbody>
</table>

Extraction Method: Principal Component Analysis. Rotation Method: Promax

Table 3-2. Item to Total Correlations and Cronbach’s a coefficient among D-FAM Scores.

<table>
<thead>
<tr>
<th></th>
<th>Item-to-total Correlation Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM Non-Supportive</td>
<td>.82</td>
</tr>
<tr>
<td>(2) D-FAM Supportive</td>
<td>.79</td>
</tr>
<tr>
<td>(3) D-FAM Total</td>
<td>.85</td>
</tr>
</tbody>
</table>

Note: D-FAM = Diabetes family adherence measure

***p < .001 for all correlations
Table 3-3. Pearson Product Moment Correlations among D-FAM Scores.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM Non-Supportive</td>
<td>1.00</td>
<td>.43***</td>
<td>.86***</td>
</tr>
<tr>
<td>(2) D-FAM Supportive</td>
<td>1.00</td>
<td>.83***</td>
<td></td>
</tr>
<tr>
<td>(3) D-FAM Total</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mean (Standard deviation) 25.6 (6.6) 26.1 (6.1) 51.7 (10.7)
Range 7-35 7-35 19-70

Note: D-FAM = Diabetes family adherence measure
* p < .05 level; ** p < .01 level; ***p < .001 level (2-tailed)

Table 3-4. Pearson Product Moment Correlations among D-FAM Scores and Child Age, Years with T1D.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM Non-Supportive</td>
<td>1.00</td>
<td>.43***</td>
<td>.86***</td>
<td>-.21**</td>
<td>-.21**</td>
</tr>
<tr>
<td>(2) D-FAM Supportive</td>
<td>1.00</td>
<td>.83***</td>
<td>-.22**</td>
<td>-.04</td>
<td></td>
</tr>
<tr>
<td>(3) D-FAM Total</td>
<td>1.00</td>
<td>-.25**</td>
<td>-.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) Child Age (years)</td>
<td>1.00</td>
<td></td>
<td>.18*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5) Duration of Diabetes (years)</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mean (Standard deviation) 25.6 (6.6) 26.1 (6.1) 51.7 13.5 (3.0) 4.8 (3.7)

Note: D-FAM = Diabetes family adherence measure
* p < .05 level; ** p < .01 level; ***p < .001 level (2-tailed)
Table 3-5. Pearson Product Moment between D-FAM Scores, Metabolic Control, DKA, and Parent/Child Measures of Regimen Adherence.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>(7)</th>
<th>(8)</th>
<th>(9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM</td>
<td>1.00</td>
<td>.43***</td>
<td>.86***</td>
<td>-.37***</td>
<td>-.26**</td>
<td>.23**</td>
<td>.37***</td>
<td>.41***</td>
<td>.38***</td>
</tr>
<tr>
<td>Non-Supportive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2) D-FAM</td>
<td>1.00</td>
<td>.83***</td>
<td>-.42***</td>
<td>-.14</td>
<td>.33***</td>
<td>.37***</td>
<td>.42***</td>
<td>.32***</td>
<td></td>
</tr>
<tr>
<td>Supportive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(3) D-FAM</td>
<td>1.00</td>
<td>-.47***</td>
<td>-.24**</td>
<td>.33***</td>
<td>.44***</td>
<td>.51***</td>
<td>.43***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) HbA1c</td>
<td>1.00</td>
<td>.26**</td>
<td>-.37***</td>
<td>-.56***</td>
<td>-.38***</td>
<td>-.41***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5) Frequency</td>
<td>1.00</td>
<td>.03</td>
<td>-.24**</td>
<td>-.16</td>
<td>-.28**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of DKA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(6) DSMP-Child</td>
<td>1.00</td>
<td>.46***</td>
<td>.39***</td>
<td>.41***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(7) DSMP-Parent</td>
<td>1.00</td>
<td>.47***</td>
<td>.62***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(8) SCAI-Child</td>
<td>1.00</td>
<td>.53***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(9) SCAI-Parent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
</tbody>
</table>

Mean (Standard deviation) 25.6 (6.6) 26.1 (6.1) 51.7 (10.7) 8.9 (1.9) 1.3 (1.8) 58.0 (9.8) 57.1 (11.6) 52.7 (10.1) 53.0 (9.8)
Table 3-6. Pearson Product Moment between D-FAM Scores, Extant Measures of Family Functioning, and Externalizing Behavior Problems

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>(7)</th>
<th>(8)</th>
<th>(9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM Non-Supportive</td>
<td>1.00</td>
<td>.43***</td>
<td>.86***</td>
<td>-.62***</td>
<td>-.45***</td>
<td>.18*</td>
<td>.17*</td>
<td>-.24**</td>
<td>-.30***</td>
</tr>
<tr>
<td>(2) D-FAM Supportive</td>
<td>1.00</td>
<td>.83***</td>
<td>-.37***</td>
<td>-.23**</td>
<td>.28***</td>
<td>.16*</td>
<td>-.12</td>
<td>-.18*</td>
<td></td>
</tr>
<tr>
<td>(3) D-FAM Total</td>
<td>1.00</td>
<td>-.61***</td>
<td>-.41***</td>
<td>.28***</td>
<td>.20**</td>
<td>-.22**</td>
<td>-.30**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) DFBC-Child Negative</td>
<td>1.00</td>
<td>.44***</td>
<td>-.18*</td>
<td>-.07</td>
<td>.19*</td>
<td>.30***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5) DFBC-Parent Negative</td>
<td>1.00</td>
<td>-.13</td>
<td>-.04</td>
<td>.12</td>
<td>.38***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(6) DFBS-Warmth Caring</td>
<td>1.00</td>
<td>.06</td>
<td>-.10</td>
<td>-.18*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(7) DFBS-Guidance Control</td>
<td>1.00</td>
<td>.10</td>
<td>-.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(8) DFRQ-No Responsibility</td>
<td>1.00</td>
<td>.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(9) CBCL-Externalizing Mean (Standard deviation)</td>
<td>25.6 (6.6)</td>
<td>26.1 (6.1)</td>
<td>51.7 (10.7)</td>
<td>15.2 (5.5)</td>
<td>16.4 (5.8)</td>
<td>52.9 (10.3)</td>
<td>55.1 (8.9)</td>
<td>2.3 (2.3)</td>
<td>8.1 (8.0)</td>
</tr>
</tbody>
</table>
Table 3-7. Pearson Product Moment Correlations among D-FAM Scores and Test-Retest Scores

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
<th>a</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) D-FAM Non-Supportive (RETEST)</td>
<td>1.00</td>
<td>.25</td>
<td>.82***</td>
<td>.83***</td>
<td>.15</td>
<td>.67***</td>
<td>.87</td>
</tr>
<tr>
<td>(2) D-FAM Supportive (RETEST)</td>
<td>1.00</td>
<td></td>
<td>.76***</td>
<td>.31</td>
<td>.85***</td>
<td>.71***</td>
<td>.75</td>
</tr>
<tr>
<td>(3) D-FAM Total (RETEST)</td>
<td>1.00</td>
<td></td>
<td>.74***</td>
<td>.60**</td>
<td>.87***</td>
<td>.82</td>
<td></td>
</tr>
<tr>
<td>(4) D-FAM Non-Supportive (INITIAL)</td>
<td></td>
<td></td>
<td>1.00</td>
<td>.43***</td>
<td>.86***</td>
<td>.82</td>
<td></td>
</tr>
<tr>
<td>(5) D-FAM Supportive (INITIAL)</td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
<td>.83***</td>
<td>.79</td>
<td></td>
</tr>
<tr>
<td>(6) D-FAM Total (INITIAL)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
<td>.85</td>
<td></td>
</tr>
<tr>
<td>Items</td>
<td>Non-Supportive</td>
<td>Supportive</td>
<td>Parental control</td>
<td>Parental monitoring</td>
<td>Communalities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
<td>----------------</td>
<td>------------</td>
<td>------------------</td>
<td>---------------------</td>
<td>---------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I get yelled at if I forget my insulin</td>
<td>.874</td>
<td>.061</td>
<td>-.047</td>
<td>-.041</td>
<td>.686</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I’m afraid to talk about diabetes with my parents</td>
<td>.341</td>
<td>.060</td>
<td>-.029</td>
<td>.032</td>
<td>.729</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents and I argue about diabetes</td>
<td>.339</td>
<td>.177</td>
<td>-.095</td>
<td>-.083</td>
<td>.592</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents nag me to test my blood sugar</td>
<td>.748</td>
<td>-.024</td>
<td>-.050</td>
<td>.019</td>
<td>.527</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents get mad if I forget my insulin</td>
<td>.865</td>
<td>-.001</td>
<td>.023</td>
<td>-.044</td>
<td>.685</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents blame me when my blood sugar is too high</td>
<td>.618</td>
<td>-.097</td>
<td>.043</td>
<td>.024</td>
<td>.556</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents nag me for not taking care of my diabetes</td>
<td>.505</td>
<td>-.125</td>
<td>.127</td>
<td>.091</td>
<td>.518</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents pay attention when I ask for help with diabetes</td>
<td>.037</td>
<td>.844</td>
<td>-.050</td>
<td>-.030</td>
<td>.678</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents explain diabetes to my teachers</td>
<td>-.045</td>
<td>.694</td>
<td>.210</td>
<td>.027</td>
<td>.634</td>
<td></td>
<td></td>
</tr>
<tr>
<td>If I have a problem taking care of my diabetes, my parents will help</td>
<td>-.062</td>
<td>.888</td>
<td>-.081</td>
<td>-.039</td>
<td>.688</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I can talk to my parents about having diabetes</td>
<td>-.002</td>
<td>.771</td>
<td>.007</td>
<td>.019</td>
<td>.710</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents tell me when I do a good job taking care of my diabetes</td>
<td>.050</td>
<td>.671</td>
<td>.010</td>
<td>.033</td>
<td>.724</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents decide how much insulin I take</td>
<td>.060</td>
<td>.124</td>
<td>.754</td>
<td>.074</td>
<td>.550</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents are responsible for reminding me to take my insulin</td>
<td>.042</td>
<td>-.040</td>
<td>.757</td>
<td>-.029</td>
<td>.585</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents do the carb counting for me</td>
<td>-.083</td>
<td>-.046</td>
<td>.812</td>
<td>-.082</td>
<td>.728</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents watch me draw-up my insulin (or watch me set my pump)</td>
<td>-.047</td>
<td>-.026</td>
<td>.065</td>
<td>.845</td>
<td>.728</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents watch me test my blood sugar</td>
<td>-.022</td>
<td>-.102</td>
<td>-.118</td>
<td>.928</td>
<td>.511</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents look at my ketone strip</td>
<td>.128</td>
<td>.242</td>
<td>-.075</td>
<td>.525</td>
<td>.572</td>
<td></td>
<td></td>
</tr>
<tr>
<td>My parents look at my meter’s log to see what my blood sugars were</td>
<td>-.010</td>
<td>.037</td>
<td>.068</td>
<td>.658</td>
<td>.686</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Supportive Scale Items

- My parents do the carb counting for me
- If I have a problem taking care of my diabetes, my parents will help
- My parents look at my meter’s log to see what my blood sugars were
- My parents tell me when I do a good job taking care of my diabetes
- I can talk to my parents about having diabetes
- My parents pay attention when I ask for help with diabetes
- My parents explain diabetes to my teachers

### Non-Supportive Scale Items

- I get yelled at if I forget my insulin
- I’m afraid to talk about diabetes with my parents
- My parents and I argue about diabetes
- My parents nag me to check my blood sugar
- My parents get mad if I forget to exercise
- My parents blame me when my blood sugar is too high
- My parents nag me about not taking care of my diabetes

### Validity Items

- My blood sugar is always between 80 and 120
- I check ketones 6 times every day
- I’m glad I have diabetes
- I eat exactly what I should every time

### Experimental Items

- My parents decide how much insulin I take
- My parents watch me test my blood sugar
- My parents look at my ketone strip
- My parents watch me draw-up my insulin (or watch me set my pump)
- My parents are responsible for reminding me to take my insulin

**NOTE:** Supportive and Non-Supportive Scales above based on 2-Factor Model. Experimental and validity items are not included in factor analyses.

Figure 3-1. D-FAM Items Retained
Figure 3-2. Scree Plot for Exploratory Factor Analysis of D-FAM
**Supportive Scale Items**

- My parents tell me when I do a good job taking care of my diabetes
- If I have a problem taking care of my diabetes, my parents will help
- My parents pay attention when I ask for help with diabetes
- I can talk to my parents about having diabetes
- My parents explain diabetes to my teachers

**Non-Supportive Scale Items**

- My parents nag me about not taking care of my diabetes
- I’m afraid to talk about diabetes with my parents
- My parents nag me to check my blood sugar
- My parents and I argue about diabetes
- My parents blame me when my blood sugar is too high

**Parental Control**

- My parents decide how much insulin I take
- My parents are responsible for reminding me to take my insulin
- My parents do the carb counting for me

**Parental Monitoring**

- I get yelled at if I forget my insulin

**Validity Items**

- My blood sugar is always between 80 and 120
- I check ketones 6 times every day
- I’m glad I have diabetes
- I eat exactly what I should every time

- My parents look at my meter’s log to see what my blood sugars were
- My parents watch me test my blood sugar
- My parents look at my ketone strip
- My parents watch me draw-up my insulin (or watch me set my pump)

**NOTE:** Experimental and validity items are not included in factor analyses. This four-factor model is an experimental future direction and requires additional subjects prior to confirmation.

**Figure 3-3. Exploratory Four-Factor Model**
Figure 3-4. Scree Plot for Exploratory Factor Analysis of D-FAM with Experimental Items
<table>
<thead>
<tr>
<th>D-FAM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>7</td>
</tr>
<tr>
<td>8</td>
</tr>
<tr>
<td>9</td>
</tr>
<tr>
<td>10</td>
</tr>
<tr>
<td>11</td>
</tr>
<tr>
<td>12</td>
</tr>
<tr>
<td>13</td>
</tr>
<tr>
<td>14</td>
</tr>
<tr>
<td>15</td>
</tr>
<tr>
<td>16</td>
</tr>
<tr>
<td>17</td>
</tr>
<tr>
<td>18</td>
</tr>
<tr>
<td>19</td>
</tr>
<tr>
<td>20</td>
</tr>
<tr>
<td>21</td>
</tr>
<tr>
<td>22</td>
</tr>
</tbody>
</table>

Figure 3-5. Final D-FAM with questions randomized.
Figure 3-6. Confirmatory Factor Analysis (CFA) Diagram for Final D-FAM 2-Factor Model.
CHAPTER 4
DISCUSSION

This dissertation research has addressed the development and validation the Diabetes Family Adherence Measure (D-FAM), a comprehensive tool for the assessment of adherence-related parenting processes for children with type 1 diabetes. Despite the existence of other psychometrically-sound measures of diabetes family functioning, none capture multiple aspects of family functioning behaviors specific to diabetes management. Recently, our research program conducted the first analysis of multiple adherence-related family factors specific to T1D (Lewin et al., 2006; Walker et al., 2004). However, in the previous study, multiple questionnaires were required. Additionally, the extant measures did not include more recent facets of diabetes management (e.g., carbohydrate counting, insulin pumps). For some time, researchers and clinicians posited that improving adherence and metabolic control in children and adolescents with T1D must involve changes within the family system; recent controlled investigations are targeting these factors (Heidgerken et al., 2006; Wysocki et al., 2000). Consequently, practical and valid measurement of these family processes is indicated. The D-FAM satisfies this need by providing a parsimonious, up-to-date assessment tool for identifying family factors associated with adherence and health status.

The development and evaluation of the D-FAM followed an empirical approach for scale design (Friedenberg, 1995; Worthington et al., 2006). Although there is no universal set of guidelines for measurement development, we followed expert consensus for general procedures including determining a specific construct of interest, developing a format for the measure, generating a pool of items, expert-review the initial item pool, consideration of validation items, initial administration, optimizing scale length, and evaluation of items/scales (Anastasi, 1988; Dawis, 1987; DeVellis, 2003; Worthington et al., 2006). Although our sample size (N=165) may
be considered borderline for CFA, many experts suggest the utility of CFA over EFA even with relatively small samples, especially with theoretically-based models of the factor structure (Bryant et al., 1998; DeVellis, 2003). With over 150 subjects and more than 10 subjects per variable, our dataset meet all but the most stringent criteria for CFA and EFA. Convergent, favorable findings with both CFA and EFA bolster our conclusions.

Confirmatory factor analysis supported a two-factor structure for the D-FAM: a Supportive scale and a Non-Supportive scale. The Supportive scale contained parental behaviors that are emotionally supportive of the child with diabetes, are instrumentally supportive with his/her regimens, and indicate appropriate involvement. More specifically, items suggest emotional support specific to diabetes (e.g., I can talk to my parents about having diabetes); assisting with diabetes-specific responsibilities/instrumental support (e.g., My parents do the carb counting for me); and monitoring regimen behaviors (e.g., My parents look at my meter’s log to see what my blood sugars were). The Non-Supportive scale includes critical, negative, and coercive parental behaviors surrounding the diabetes care regimen (e.g., I get yelled at if I forget my insulin, I’m afraid to talk about diabetes with my parents, My parents blame me when my blood sugar is too high). Exploratory factor analysis provided additional support for the two-factor model. High factor loadings and the large sample suggest replicablity of the factor structure (Floyd et al., 1995).

Internal consistency and test-retest reliability were used as indices of reliability. Acceptable internal consistency was found for D-FAM non-supportive (α = .82) and supportive scales (α = .79), suggesting that items within each scale contribute to the overall scale’s score. Strong test-retest correlations were found between Supportive (.85) and Non-Supportive scales (.83). High associations between initial reports and two-week retests are promising statistics
given that family functioning is believed to be a relatively stable construct. Correlations were strong and significant, despite the low number of children completing the retest administration. Further, no differences in demographic variables or adherence, metabolic control, or extant family functioning measures were found between re-test completers (17.5% of the sample) and the remainder of the sample, suggesting that children who were re-tested were representative of the overall sample. Overall, data from our sample support reliability of the D-FAM and the two-factor structure.

A strong, initial analysis of the D-FAM’s validity was also conducted. First, concurrent validity was assessed via comparisons with extant measures of diabetes specific family functioning. As expected, DFBC scores reflecting child reports of more parental negativity were associated with more parental negativity on the D-FAM Non-Supportive scale and less parental support on the D-FAM Supportive scale. Similarly, DFBS scores reflecting more parental warmth and caring was positively correlated with D-FAM Supportive scale scores. Additionally, parental negativity on the D-FAM was inversely correlated with DFRQ scores reflecting family responsibility for diabetes care. Parent report of negativity on the DFBC was also associated with less support on the D-FAM. Interestingly, associations between the DFBC (parent report) and D-FAM (child report only) were weaker than between the DFBC (child report) and the D-FAM. This discrimination lends further support for the construct-validity of the D-FAM given that it is reasonable that two child-report measures of family support should relate more strongly than a parent-report compared to a child-report. Stronger relations between the D-FAM and the DFBC than between the D-FAM and the CBCL (child externalizing behavior) supports discriminate validity. Construct validity of the D-FAM is also promising. Moderate to strong
correlations were found between the D-FAM and HbA1c and child/parent reports of regimen adherence. Significant correlations were also found with the frequency of prior DKA.

The addition of the validity/social desirability scale adds to the utility of interpreting D-FAM scores. Rationally derived, the four items on this scale were selected based on low endorsement of high-anchor responses (i.e., “Always”). Psychometric properties of these items were examined. Unlike D-FAM content items, validity items were sought based on a mean of less than 2 (“Rarely”), mode of 1, and skewed right distribution. Given that (1) the content validity/social desirability items were not related and (2) the overall low frequency of endorsement, poor internal consistency was not unexpected. In other words, these items were not designed to congregate statistically as a scale. Rather, they are included to provide indication to researchers and or clinicians that other responses may be atypical. Trends in our data support this premise. Specifically, qualitative analysis (i.e., visual inspection of data) suggests that children who endorse atypical responses on the validity/social desirability items have worse metabolic control, lower adherence, and more externalizing behavior problems. Data trends also suggest that overall D-FAM subscale scores are higher among children who endorsed validity/social desirability items, suggesting inflation of D-FAM scores among children who endorse validity items. Although too few participants endorsed atypical responses on the validity items for quantitative/statistical analysis, observable trends suggest the utility of these items in interpreting the D-FAM. Unfortunately, of the 17.5% of children completing test-retest data, only one child endorsed “Always” on two validity items during the initial administration. Ideally, a larger test-retest sample would allow us to evaluate whether validity items predict test-retest performance. For the present time, D-FAM reports with children endorsing “5” or
“always” on 2 or more validity items should be interpreted with caution. Scores may reflect attempts to respond favorably, careless responding, or poor comprehension of the material.

For ease of interpretation, items were recoded prior to analysis (for ease of interpretation) such that higher scores reflect more positivity on the D-FAM Supportive scale and less negativity on the D-FAM Non-Supportive scale. In other words, higher scores are desirable. A D-FAM total score is also provided (the sum of the two subscale scores). Given the poor fit of a single-factor model, interpretation of the D-FAM total score is not recommended. Although the recoded data suggests higher D-FAM total scores correlate with increased diabetes-specific family/parenting support, we caution that Support and Non-Support are not mutually exclusive. Thus, the D-FAM total score should be considered experimental, despite inclusion in the analyses above.

Consistent with previous research, this study suggests age-related changes in family-functioning as measured by the D-FAM. Specifically, older children report less supportive parental behaviors and more non-supportive parental behaviors. Similarly, children who have had diabetes longer report more negative diabetes-specific interactions with their parents on the D-FAM. Previous studies clearly document the developmental changes in family diabetes management responsibility (e.g., the shift of responsibilities from the parent to the adolescent (Anderson & Laffel, 1997). However, independent of age/developmental level, the overall dyadic lack of responsibility is predictive of poor health status (Anderson, 1990). Previous studies have also found more supportive parental behaviors among younger children compared to older children (Waller et al., 1986). In the current study sample, age was not found to moderate the relation between D-FAM scores and HbA1c. In prior studies, age-moderating effects were found between HbA1c and extant measures of diabetes-specific family functioning (Lewin et al.,
The reasons why moderation was not replicated in this study are unclear (they may include abnormalities in the sample [see below] or the strength of the measure). However, the absence of age moderation may be advantageous: HbA1c-D-FAM scores were significantly correlated across ages, suggesting utility of the D-FAM across developmental groups. Although this study offers the largest cross-sectional dataset of diabetes adherence-related family factors, there are no longitudinal analyses of changes in diabetes-specific family functioning with age. Give that recent, unpublished data from our research group found that diabetes-specific family functioning predicted metabolic control at two-year follow-up, longitudinal analysis of diabetes-specific family functioning appears warranted.

Descriptively, D-FAM scores did not relate to gender, ethnicity, or family income. Modest group differences were found between single-parent (lower) and two-parent families on the D-FAM Supportive scale. Perhaps single parents are more burdened with parenting, domestic, and workplace duties to provide the same level of support as parents with a partner. Although ANOVA may have been unable to detect differences in ethnicity due to low number of subjects in each group, t-tests found no differences between Caucasians and Non-Caucasians on D-FAM scores. The overall lack of demographic covariates is encouraging and may suggest that D-FAM/HbA1c/adherence relations are less confounded.

Limitations

There are a number of limitations regarding this research. First, sample size limited the inclusion of additional items and possibly the identification of additional factors at this time. For example, exploratory factor analysis identified a four-factor model when the five experimental items were included. Confirmatory factor analysis failed to verify this factor structure, likely due to insufficient cases. Although our sample was adequately sized for our initial factor analyses (14 items, 1-3 factors), more detailed analysis would be possible with a larger sample.
Additional participants might also allow for a more empirical evaluation of the validity/social desirability scale. At present, only 4.24% of children endorsed “Always” on two items of the validity scale. Consequently, inferential statistics could not be used to evaluate group differences between the overall sample and children who responded atypically from the remainder of the sample. A larger sample might also allow the inclusion of redundant items (i.e., items with similar content but different wording) to assess for consistency in responses (e.g., including positive and negative phrasing of items with similar content). Additionally, a parent report version would allow for parent-child comparisons and provide a potential indicator for invalid responding.

Characteristics of the present sample may also limit generalization of findings. First, over fifty percent of families reported less that $50,000 total annual income and over 25% report earning less than $25,000. Increased financial demands and single parent status may alter family processes related to diabetes (e.g., added responsibility placed on the child with T1D or less time for emotional availability). Second, and even more striking, is the frequency of DKA among our sample. Over 26% of children have had multiple episodes of DKA. This is alarming given that the mortality rate is 1-3% of children with DKA in the United States (Scibilia et al., 1986). Further, recurrent DKA has been associated with the presence of child psychopathology (Liss et al., 1998). If psychopathology is over-represented in this sample, reports of family functioning may not be comparable for a less medically and psychiatrically complex/severe population. Although the CBCL externalizing scale did not suggest a disproportionate percentage of children with externalizing behavioral disorders (e.g., symptoms associated with Attention Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, Conduct Disorder), no screening for internalizing disorders was administered (e.g., depression, anxiety). Finally, inclusion of a
measure of general family functioning/parental psychopathology would have been useful (although these items would have substantially lengthened a long battery of study measures) to identify the presence of more global familial dysfunction, beyond the scope of diabetes-care. In our sample, HbA1c was not correlated with age (contrary to prior findings), with may be due to a restriction-of-range given the severity of the population. This phenomenon (i.e., the potentially atypical sample) may limit the generalization of the current findings given the study sample. In sum, replication of these findings in other samples is desirable (i.e., cross-validation; see below).

**Future Directions**

There are several future considerations for this research. First, as discussed above, reevaluation of the D-FAM with a larger, more diverse sample could provide further support to validity and reliability. Experts recommend reexamination of new scales with a secondary sample after initial development (DeVellis, 2003). Second, longitudinal analysis of the D-FAM could provide long-term stability estimates for the D-FAM as well as tracing developmental changes in diabetes-related family functioning. Additionally, prospective collection of DKA and HbA1c would allow for true assessment of predictive validity of the D-FAM. Third, including the D-FAM in trials of behavioral family treatment for children with T1D and poor metabolic control could: (1) evaluate whether family functioning changes with psychological treatment and (2) whether changes in family functioning predict behavioral/adherence changes (or changes in metabolic control). Fourth, development of a parent version of the D-FAM may provide additional data about family processes related to adherence. This parallel parental version of the D-FAM would allow researchers to monitor parental perception of support and could provide contrasts with child reports. Of particular interest would be cases were a child reports poor support and a parent reports strong support. Perhaps parent-child disagreement would predict worse medical/behavioral outcomes.
Fifth, continued evaluation of the D-FAM with the experimental items appears warranted. EFA suggests support for a multidimensional construct of family-support, additional subjects may justify repeating CFA. Viewing diabetes-specific family support as a multidimensional construct has been proposed in prior research. For example, Waller and colleagues (1986) suggested emotional support and guidance/behavioral support. Unfortunately, a relatively small sample likely prevented empirical derivation/validation of scales (i.e., factor analysis). Our experimental model with multiple dimensions of support (emotional support, control, and monitoring) is consistent with the conceptualization by Waller et al. (1986). It is reasonable that a parent could be emotionally supportive while failing to monitor care activities. Conversely, a parent may be hypervigilant with monitoring and regulating/controlling but lacking in emotional availability regarding diabetes. For example, data with our two-factor model suggest that some parents can be perceived as both supportive and non-supportive/critical. Anderson and colleagues (1997) suggest developmental shifts in responsibility and parental-care behaviors specific to diabetes. Thus, future research with the D-FAM could track shifts in these multiple dimensions of parental supportive behaviors (e.g., age variations, interrelations with adherence/metabolic control). Although it was beyond the scope of this dissertation, evaluation of validity and reliability of the four factor D-FAM structure should be examined, pending stability of the factor solution with more subjects.

Finally, future data collection with a broad, multi-site sample could allow for normative data to be obtained for the D-FAM. Ideally, normative data could be stratified by age with a minimum breakdown of pre-teens, younger adolescents and older adolescents. These data would improve cross-sectional, developmental comparisons and allow for researchers to examine whether low/high D-FAM scores correspond with extreme HbA1c values.
The D-FAM provides a clinical and research tool for the assessment of family behaviors that correspond with adherence to the diabetes treatment regimen and have been linked to diabetes-related health status. Despite the utility of extant measures of family functioning, the technology of diabetes treatment regimen has increased exponentially over the past decade with the advent of the insulin pump, intensive insulin therapy, insulin-glargine and very rapid Acting insulin, blood-glucose meters, carbohydrate counting, etc. It comes to reason that the resources of the family to cope with the increased complexity have become increasingly taxed. Given the complex, challenging, and often stress-inducing nature of diabetes treatment regimen, there is a clear role of psychology in helping family members understand and adjust behaviorally to the requirements of the child's diabetes regimen. Information gathered from a comprehensive assessment of family functioning (as assessed by the D-FAM) would be useful in both research evaluation and clinical treatment planning including examination of current perceptions of the child as well as changes over time.
LIST OF REFERENCES


BIOGRAPHICAL SKETCH

Adam Benjamin Lewin, Ph.D. completed his graduate training in the Department of Clinical and Health Psychology at the University of Florida Health Science Center in 2007. Subsequently, he completed his Residency/Internship in Clinical Psychology in the Department of Psychiatry at the Stewart and Linda Resnick Neuropsychiatric Hospital/Semel Neuropsychiatric Institute at the University of California, Los Angeles’ (UCLA) David Geffen School of Medicine. He is currently an NIH-sponsored (T-32) Postdoctoral Fellow in clinical psychology, also at UCLA’s Department of Psychiatry. His research focus is on psychophysiological and neurobiological mechanisms of pediatric Obsessive Compulsive Disorder.

Adam Lewin graduated from Eastside High School’s International Baccalaureate Program (Gainesville, FL) in 1994. Next, he completed his bachelor of science (psychology) at the University of Florida’s College of Liberal Arts and Sciences in 1998. Following matriculation, he worked as a research therapist for the University of Florida Center for Self-Injury. Subsequently, he worked as a therapist and research assistant in the area of autism and developmental disabilities for two years at the Johns Hopkins University and Kennedy Krieger Institute in Baltimore, Maryland. Adam Lewin returned to Gainesville in 2001 to pursue his doctorate in Clinical Psychology at the University of Florida. He received his Masters in Clinical Psychology in 2003. His thesis focused on family functioning among youth with type 1 diabetes. Dr. Lewin’s current research interests include pediatric psychology, diabetes psychology, and anxiety/OCD/tic disorders.

In addition to his professional interests, Dr. Lewin is a board member and 16-plus year volunteer at Florida’s Diabetes Camp (a not-for-profit camp for youth with type 1 diabetes). Dr. Lewin enjoys outdoor recreation including hiking, sailing, national park travel, and camping.
Travel, photography, dining, exercise/fitness, and watching sports are other longstanding interests. More specifically, Dr. Lewin is a dedicated Gator Fan who is proud to have been a UF student for the 1996 and 2006 Football National Championships as well as the Back-to-Back 2006 and 2007 Basketball National Championships. Above all, Dr. Lewin enjoys time with his family, friends and pets as well as visits to his beloved hometown of Gainesville, FL for visits to Godfathers Pizza, Burrito Brothers, Satchel’s Pizza, and Caribbean Spice.