

MATERNAL INTRUSIVE INVOLVEMENT AND ADOLESCENT  
FUNCTIONING IN YOUTH WITH TYPE 1 DIABETES

APPROVED BY SUPERVISORY COMMITTEE

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Deborah Wiebe, Ph.D.

---

Carroll Hughes, Ph.D.

---

Beth Kennard, Psy.D.

---

Richard Robinson, Ph.D.

---

Sunita Stewart, Ph.D.

## DEDICATION

I dedicate this dissertation to my husband and best friend, Jarrett Brandon Reed.

MATERNAL INTRUSIVE INVOLVEMENT AND ADOLESCENT  
FUNCTIONING IN YOUTH WITH TYPE 1 DIABETES

by

GABRIELA OROZA

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Gabriela Oroza, Ph.D.

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Supervising Professor: Deborah Wiebe, Ph.D.

One factor affecting emotional and physical well being in adolescents with type 1 diabetes is the degree of maternal involvement. Adolescents whose mothers are actively involved in the daily management of their diabetes tend to follow their regimen more consistently and are in better glycemic control. However, intrusive levels of involvement have been correlated with increased depression, decreased adherence, and poor metabolic control. In the past intrusive involvement has been seen as a consequence of innate maternal characteristics

such as trait anxiety, and as the cause of poor child functioning in adolescents with intrusively involved caregivers. More current research takes a transactional perspective in which intrusive involvement interacts with child functioning in a reciprocal manner. To investigate the current transactional perspective, the current study explored the temporal relationships between intrusive maternal involvement in adolescent diabetes management and child functioning variables including depression, adherence, and metabolic control across two time points (an average of 16 months apart) using cross-lagged panel correlation analyses and hierarchical linear regression. The current study also investigated the role of maternal trait anxiety in the development of intrusive involvement by proposing one potential transactional process and testing it in the sample. Adolescents (N = 83, 10 to 15 years of age, 53% male) with type 1 diabetes mellitus (duration of at least 1 year) completed measures of adherence, depression, and intrusive involvement, and their mothers provided relevant demographic and illness related information. Metabolic control was collected from participants' medical records. This study found no evidence to support the workings of a transactional process within mother-teen dyads for adolescents with type 1 diabetes. However, consistent with the traditional linear model, results indicated that intrusive involvement was associated with higher levels of depressive symptoms in females at Time 1, and that the effects continued to be seen over time. No association was found between intrusive involvement and depressive symptomatology for males

at either time point. These findings point to the need for interventions geared toward improving mother-daughter interactions and reducing depressive symptomatology in teenage girls with type 1 diabetes.

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## LIST OF ABBREVIATIONS

CDI	Children's Depression Inventory
HbA <sub>1c</sub>	Metabolic Control
Ill. Dur.	Illness Duration
Intrus. Inv.	Intrusive Involvement
SCIC	Self-Care Inventory - Child

# CHAPTER ONE

## Introduction

Type 1 diabetes, a challenging chronic disease, places many demands on youth with the illness and their families. A major goal of medical management for this disease is the promotion of near normal levels of glycemia, as poor glycemic control has been implicated in the development of serious health complications, such as kidney disease and blindness (Diabetes Control and Complications Trial Research Group [DCCT], 1994). Stabilizing control over blood glucose levels in diabetes requires adherence to a complex regimen of blood glucose monitoring, insulin injections, exercise and diet (Stewart et al., 2000). Unfortunately, adolescents are known for being less adherent to treatment regimens than are children (Jacobson et al., 1990) or adults (Morris et al., 1997), and research shows that patterns of nonadherence emerging in adolescence are often sustained into adulthood (Bryden et al., 2001; Kovacs, Goldston, Obrosky, & Iyenger, 1992; Patterson & Garwick, 1998).

One factor that affects diabetes management in adolescence is the degree of maternal involvement. Adolescents who identify their mothers as uninvolved exhibit poorer adherence, decreased quality of life (Wiebe et al., 2005) and reduced metabolic control (Anderson et al., 1990, 1997; Wysocki et al., 1996). However, in a study of intrusive involvement in the academic realm, Pomerantz

& Eaton found that when mothers' efforts to support their adolescent are intrusive, they may be inadvertently imparting a message of control and low competence (2000). Given the detrimental effects of sub-optimal levels of involvement, mothers coping with their adolescent's chronic illness are challenged with remaining actively supportive while at the same time fostering independence and autonomous diabetes management skills.

Achieving a healthy degree of involvement becomes increasingly difficult in adolescence as the parent's primary goal of facilitating positive health outcomes begins to conflict with the child's developing autonomy (Holmbeck et al., 2002). In children with chronic illnesses, such increased levels of parental involvement may be indicated to promote health (Holmbeck, 1997), but may also contribute to excessive, less adaptive involvement in these families. In fact, some have suggested that the highest degrees of intrusive involvement may occur in families with children who suffer from chronic illnesses such as type 1 diabetes (Anderson & Coyne, 1991, 1993; Cappelli, McGrath, MacDonald, Katsanis, & Lascelles, 1989).

Studies examining the functioning of chronically ill children and their parents have revealed a great deal of variability in the ways that families cope with the challenges of chronic illness (Thompson & Gustafson, 1996; Wallander & Thompson, 1995). In the face of these challenges, and those of adolescence, some families are unable to negotiate healthy involvement patterns. Historically,



the development of sub-optimal involvement was theorized as a manifestation of innate maternal characteristics. For example, mothers high in trait anxiety were thought to engage in higher degrees of intrusive involvement than mothers low in trait anxiety. In turn, highly trait anxious mothers were held responsible for the consequences of their intrusive involvement, including increased levels of depression (Burbach, Kashani, & Rosenberg, 1989), decreased metabolic control and poorer child adherence (Glasgow, McCaul, & Dreher, 1983).

More current research in developmental psychology, however, employs a transactional approach to parent-child relationships and child development. From this perspective, correlates of intrusive involvement such as depression, adherence, poor metabolic control and trait anxiety, which have traditionally been seen as either causes or consequences of intrusive involvement, are instead considered part of a transactional process in which they can act as both cause and effect in a dynamic fashion. For example, from a transactional perspective, trait anxious mothers may develop negative appraisals of child competence when they see their child struggling (e.g. increased depression, decreased adherence, poor metabolic control) (Dix, 1991). These negative appraisals in turn may lead mothers to increase their involvement (Cameron, Young & Wiebe, 2007), which the developing adolescent may subsequently perceive as intrusive and unwelcome. In this example, negative child functioning acts as a precursor to intrusive involvement, and not solely as an outcome of such involvement.

The present study explored this transactional perspective regarding intrusive maternal involvement in adolescent diabetes management. Aim 1 examined the temporal relationships between intrusive involvement and child functioning variables including depression, adherence, and metabolic control. By focusing on concurrent and longitudinal associations between children's perceptions of parental intrusiveness and the quality of their functioning, we began examining the extent to which each variable predicts intrusive involvement concurrently and longitudinally, as well as the extent to which intrusive involvement predicts child functioning concurrently and across time. Aim 2 examined how maternal trait anxiety plays a role in intrusive involvement by testing one potential transactional process that takes all three measures of functioning into account.

## CHAPTER TWO

### Review of Literature

#### *General Overview of Type 1 Diabetes*

Type 1 diabetes affects approximately 176,500 or 0.22% of people under the age of 20 (American Diabetes Association [ADA], 2007), making it one of the most common severe chronic diseases of childhood in the United States. Each year more than 13,000 young people are newly diagnosed with type 1 diabetes (Centers for Disease Control and Prevention [CDC], 2007). There are two forms of diabetes, type 1 diabetes (previously termed Insulin-dependent diabetes mellitus or juvenile diabetes) and type 2 diabetes (previously known as non-insulin-dependent diabetes mellitus). Type 1 diabetes is the form of the disease that generally emerges between childhood and early adulthood, and was the focus of the present study.

Type 1 diabetes is caused by beta cell destruction, which is often immune mediated. However, the etiology of this autoimmune process is not yet well understood (Rewers, Norris and Dabelea, 2004). Beta cell destruction results in the loss of insulin secretion by the pancreas and eventual absolute insulin deficiency (Olsen & Sutton, 1998). In adequate quantities, insulin provides the body with necessary energy by acting to allow glucose to permeate through cell membranes, a process essential for normal food metabolism. However, in type 1 diabetes, the relative or absolute lack of insulin prevents glucose from leaving the

bloodstream and being distributed to the cells in the body. The accumulation of glucose in the bloodstream, when reaching dangerously high levels, can result in hyperglycemia. Prolonged hyperglycemia results in an abnormal metabolic process in which cells metabolize carbohydrates, fats and proteins, depleting the body's energy reserves. When the body compensates for a lack of glucose by breaking down fat, waste products called ketones are produced and can adversely affect the chemical balance of the blood and other organ systems, resulting in a dangerous, life-threatening condition referred to as diabetic ketoacidosis (DKA).

The treatment of diabetes is designed to maintain blood glucose levels within a normal range by providing just enough insulin to metabolize glucose in the bloodstream. When blood glucose levels fall within the normal range, the potential for acute and chronic complications is lessened (Strowig & Raskin, 1992). A<sub>1c</sub> is a specific subtype of hemoglobin (Hb), the compound in red blood cells that transports oxygen. Because glucose binds slowly to Hemoglobin A and decomposes slowly, testing HbA<sub>1c</sub> allows health providers to measure a patient's average glycemia over the preceding 2 to 3 months (Sacks et al., 2002). Due to concerns regarding the risks of hypoglycemia and the potential of creating a feeling of failure in the patient and family, the American Diabetes Association (2007) recommends a target A<sub>1c</sub> of < 7.5% for adolescents ages 13 through 19, and < 8% for children ages 6 through 12.

To maintain blood glucose concentrations at an acceptable level, people with type 1 diabetes must adhere to a complex regimen of blood glucose monitoring, insulin injections, exercise and diet (Stewart et al., 2000). Prescribed treatment tasks can include monitoring of blood glucose levels at least three times a day, daily injections of one or more doses of insulin intravenously or through a subcutaneous pump, daily exercise, and a dietary plan that involves limiting consumption of sugar and monitoring carbohydrate intake (Wysocki, Greco, & Buckloh, 2003). The number and complexity of the different tasks involved in managing type 1 diabetes can be overwhelming even for the most competent and motivated patient, as people with diabetes must anticipate the impact of exercise, illness, stress, and caloric intake on their blood glucose levels and adjust their insulin dose according to these fluctuations (Stewart et al., 2003; Wysocki et al., 2003; Greening, Stoppelbein & Reeves, 2006).

The importance of good diabetes management cannot be overstated. The Diabetes Control and Complications Trial (DCCT, 1994) confirmed that improved metabolic control is significantly associated with delayed onset and progression of microvascular complications, with increasing risks as metabolic control worsens. Decreases in  $A_{1c}$  also result in a reduction in neuropathic complications and possibly macrovascular diseases (ADA, 2007). Conversely, poor metabolic control can lead to retinopathy, nephropathy, neuropathy, heart disease, limited joint mobility, and increased mortality (DCCT, 1994; La Greca et

al., 1995). In fact, type 1 diabetes is the leading cause of blindness, end-stage renal disease, amputation, and a major cause of cardiovascular disease and premature death in the general population, resulting in greater than 5 billion dollars in medical care expenditures each year (Songer, 1990). Additionally, chronic hyperglycemia in children is associated with reduced memory and learning capacity; age of onset of type 1 diabetes has a predictive negative effect on measures of intelligence, particularly visuospatial ability (Northam, Anderson, Werther, Warne & Andrews, 1999).

While good self-management dramatically extends survival, there is no cure for diabetes. As the third leading cause of death in the United States, diabetes is indirectly responsible for over 300,000 deaths annually (Davidson, 1986; Jacobson and Leibovich, 1984). The main cause of death in patients with type 1 diabetes is coronary artery disease (CAD), which accounts for a large proportion of premature morbidity and mortality in the general population. Heart disease in type 1 diabetic patients occurs earlier in life (Moss, Klein and Klein, 1991; Dorman et al., 1984) and more frequently; women with type 1 diabetes are 9 to 29 times more likely to die of CAD than women without diabetes, and the risk for men increases 4 to 9 times (Krolewski et al., 1987). Although the mortality rate of type 1 diabetes within the first 20 years is only 3-6%, it is still 12 times higher for diabetic females and 5 times higher for diabetic males when compared to the general population (Borch-Johnsen et al., 1987). However, at least half of patients

survive over 40 years and 25% of those patients have no major complications (DCCT, 1997).

### *Diabetes in Adolescence*

Adolescence complicates the already difficult task of maintaining good metabolic control. For children with diabetes, adolescence is marked by heightened family conflict, increased psychological distress, and poorer adherence to treatment regimens (Anderson et al., 1999; Delamater, Smith, Kurtz, & White, 1991; Jacobson et al., 1990; Northam et al., 1999; Wysocki, 1993). There is also a large body of research indicating that children with diabetes show a decline in glycemic control as they enter adolescence (Amiel, Sherwin, Simonson, Lauritano & Tamborlane, 1986; Anderson, Ho, Brackett, Finkelstein, & Laffel, 1997; Hanson et al., 1989; Hanson, Henggeler, & Burghen, 1987a; Jacobson et al., 1990; Johnson et al., 1992; La Greca, Follansbee, & Skyler, 1990; Mortensen & Hougaard, 1997; Stewart et al., 2003). Although this decline in metabolic control is partly attributable to the physiological aspects of puberty (Amiel et al., 1986), adolescence is also often a period of reduced self-management (Anderson, Auslander, Jung, Miller, & Santiago, 1990; Johnson et al., 1992). Specifically, teens are less adherent to diabetic treatment regimens than children (Jacobson et al., 1990) or adults (Morris et al., 1997), and research shows that patterns of

nonadherence emerging during adolescence are often sustained into adulthood (Bryden et al., 2001; Kovacs et al., 1992; Patterson & Garwick, 1998).

While intensive management that maintains near-normal blood glucose levels reduces the risk and progression of severe long-term microvascular complications (DCCT, 1994), this demanding regimen can place a tremendous burden on the developing adolescent as he or she copes with developmental challenges including heightened susceptibility to depression in girls (Nolen-Hoeksema, 1987) and increasing peer pressure (Burroughs, Pontious, & Santiago, 1993; Grossman, Brink, & Hauser, 1987). For example, adolescents with diabetes acknowledge that they have more trouble adhering to treatment protocols when in social situations (Delamater et al., 1988) and are less likely to comply with their diet plan or to check their blood glucose in the company of peers (Bearman & La Greca, 2002; Skinner, John, & Hampson, 2000).

An important aspect in the development of independence in adolescents with diabetes is the assumption of responsibility for disease management (Hanna & Guthrie, 2001). Unfortunately, the desire for increased autonomy in the formation of personal identity and independence (Kemper, Forsyth, & McCarthy, 1990), as well as the hormonal changes around the time of puberty, can result in decreased glycemic control (McConnell, Harper, Campbell, & Nelson, 2001). These developmentally appropriate changes in adolescent



autonomy demand reciprocal changes in the parent-adolescent relationship (Steinberg, 1987), particularly in the degree and quality of parental involvement.

### *Maternal Involvement*

Maternal involvement is one psychosocial variable that has consistently emerged as a significant predictor of adolescents' adherence to diabetes treatment (Anderson et al., 1990; Hanson et al., 1987a; La Greca et al., 1990, 1995; Wysocki & Gavin, 2006; Wysocki et al., 1996). Adolescents whose mothers are actively involved in the daily management of their diabetes tend to follow their regimen more consistently and are in better glycemic control (McKelvey et al., 1993; Waller et al., 1986). More specifically, child appraisals of mothers as supportive or collaborative during adolescence are associated with increased adherence and improved glycemic control (Wiebe et al., 2005). In fact, according to Wiebe et al. (2005), optimal diabetes management is more likely when mothers are identified as collaborators when problems arise in treatment. Such maternal collaboration may be particularly beneficial for diabetes management in the early to middle stages of adolescence as parents provide a scaffold to eventual autonomy by modeling optimal problem-solving strategies.

While most mothers are able to negotiate an optimal degree of involvement with their adolescent, some mothers, despite good intentions, struggle to find the middle ground between encouraging autonomy and the

parental involvement necessary for metabolic success. Mothers who are unable to flexibly respond to the challenges of diabetes in adolescence may, in time, find themselves engaged in counterproductive or detrimental patterns of involvement. For example, they may be 1) underinvolved, 2) inflexibly involved, or 3) intrusively involved. These maladaptive patterns of involvement occur to varying degrees, and are by no means the only possible reactions to the challenges posed by adolescence in this population. These particular patterns were chosen for discussion however, because they are prevalent in the literature, and because they represent extremes that are generalizable to differing degrees to many parent-adolescent dyad transactions surrounding diabetes.

*Underinvolvement.* Young teenagers' needs for privacy, peer acceptance, and autonomy can sharply change the pattern of responsibility for diabetes management and treatment (Anderson and Coyne, 1991). Because adolescents spend an increased amount of time with peers and away from home, parents often have fewer opportunities to supervise and to influence their behaviors (Drotar & Ievers, 1994; Hill & Holmbeck, 1986; Miller-Johnson et al., 1994; Steinberg, 1987). Increases in the amount of time the adolescent spends outside of the home often result in decreased maternal involvement in the child's care, ostensibly accompanied by poorer child adherence (Anderson et al., 1997; Hanson et al., 1987a; La Greca et al., 1990), decreased quality of life (Wiebe et al., 2005), and reduced glycemic control (Anderson et al., 1990, 1997; Wysocki et al., 1996).

Interventions that maintain optimal maternal involvement minimize these declines (Anderson et al., 1999).

The premature transfer of diabetes management to adolescents can also have consequences on illness management and metabolic control (Wysocki et al., 1996; Anderson et al., 1997; Hanson et al., 1987a; Wysocki et al., 1996).

Although most teens possess the necessary cognitive skills to implement the required treatment tasks for maintaining optimal glycemic control (Thomas et al., 1997), not all adolescents are equally capable or have the emotional resources to manage a complex treatment regimen while also negotiating the developmental tasks of adolescence (Wysocki et al., 2003). Teens may feel so overwhelmed and frustrated when left to manage their treatment regimen without support that they neglect or avoid important care behaviors altogether. In a study focusing on age and autonomy as cues to transfer responsibilities from parent to child, maternal under-involvement was associated with decreased HbA<sub>1c</sub> values primarily when children reported lower levels of self-reliance (Palmer et al., 2004). Therefore, the transfer of diabetes responsibility from mother to child without sufficient autonomy may be particularly detrimental to metabolic control.

*Inflexible Involvement.* As children enter adolescence and are faced with fluctuations in metabolic control, mothers may have a difficult time flexibly adjusting involvement to optimal levels and may revert to the parenting strategy they successfully employed in the past. However, approaches that were successful

in achieving metabolic control in younger children may no longer be appropriate for adolescence. A study by Wiebe et al. (2005) revealed that younger children in the 10 to 15 age range tended to have better adherence when they appraised their mothers as taking charge or controlling the management regimen. Older children in this age range, however, displayed poorer adherence when they appraised mothers as controlling their diabetes management. In other words, levels of parental involvement that were welcome and perceived as helpful by children may begin to be experienced by adolescents as intrusive and overprotective.

*Intrusive Involvement.* As adolescence approaches, metabolic control declines and parent-child relationships become less cohesive (Paikoff & Brooks-Gunn, 1991). In an effort to be helpful and involved, some well-meaning mothers may engage in what Anderson and Coyne (1991) term “miscarried helping.” Miscarried helping refers to a process by which the caretakers’ investment in being helpful and achieving a positive outcome for the child paradoxically leads to interactions that are constraining and harmful to the child’s well-being and successful adaptation. Coyne, Wortman, and Lehman (1988) described the process of miscarried helping, with reference to spousal interactions, as the failure of “well-intentioned support attempts because they are excessive, untimely, or inappropriate.” The rapid and dramatic changes that are part of adolescence, particularly those that occur in the context of changing parent-child relationships

and illness management, make such untimely or inappropriate support attempts especially likely.

*Accounting for Differences in Patterns of  
Involvement Across Families*

Given the detrimental correlates of sub-optimal involvement, it is important to understand why maladaptive degrees of involvement exist in some families and not in others.

*Gender and Maternal Involvement*

There is reason to believe that mothers engage differentially in the patterns of involvement referenced above based on the gender of their child. For example, investigators have argued that, while boys are encouraged to become self-reliant through collaborative interaction with caregivers (Ruble, Greulich, Pomerantz, and Gochberg, 1993), mothers may expect more independent diabetes care from their daughters and may respond to failures in diabetes management in girls in a more controlling way (Williams, 1999; Higgins, 1991). Additionally, gender differences, such as in self-evaluation tendencies and coping mechanisms (Ruble et al., 1993), may result in different perceptions of maternal involvement by girls versus boys. Subsequently, perceptions of maternal involvement may have different consequences on functioning in girls than in boys.

*Maternal Characteristics and Intrusive Involvement: The Linear Perspective*

In the past, clinicians and researchers have named innate maternal

characteristics such as trait anxiety as the culprit for the development of sub-optimal parental involvement (e.g. Levy, 1970). In other words, intrusive involvement was thought of as a fairly direct manifestation of anxious mothering, independent of child or illness factors. Anxiety-driven intrusive involvement was, in turn, seen to be the root of poor child functioning, including increased depression (Burbach et al., 1989; Cappelli et al., 1989; Mayes, Handford, Kowalski, & Schaefer, 1988; McFarlane, 1987), decreased adherence, and poor metabolic control (Hanson, Henggeler, and Burghen, 1987b; Schafer et al., 1983).

#### *The Transactional Perspective*

Current research on child development has moved beyond this linear manner of thinking. From the more contemporary, transactional perspective, notions of linear causality are discarded and the existence of feedback loops or reciprocal influences between child and mother are highlighted (Coyne & Holroyd, 1982). Variables such as the development of intrusive involvement are not attributed to the effects of single contributors (i.e. maternal trait anxiety) or seen as endpoints; rather they are considered as one of many contributors in a multi-faceted, reciprocal model.

While it is possible that intrusive levels of maternal involvement lead to higher levels of depression in teens with type 1 diabetes, from a transactional perspective it is also possible that mothers mistake depressive symptomatology in the child for a lack of motivation or ability to take initiative for diabetes

management (Coyne et al., 1988). In the face of such negative appraisals of child competence, mothers may increase their involvement to potentially intrusive levels (Cameron et al., 2007). This phenomenon may be more common among mothers with specific characteristics such as trait anxiety, but is unlikely to result from maternal characteristics alone. Instead, indicators of poor child functioning such as depression, poor metabolic control and decreased adherence, normally seen merely as outcomes, are examined as potential contributors to the recurring transaction between parent and child (Wiebe et al., 2006).

Considering the complex nature of parent-child interactions, the limitations of the traditional linear perspective prevent us from fully understanding the complexity of the association between intrusive involvement and child functioning. Without a more complete understanding of these dynamics, it will be difficult to design interventions to minimize the detrimental effects of sub-optimal involvement and maximize optimal involvement. The exploration of perspectives that employ a more transactional approach will be important in broadening our understanding of these relationships and in the eventual implementation of beneficial interventions.

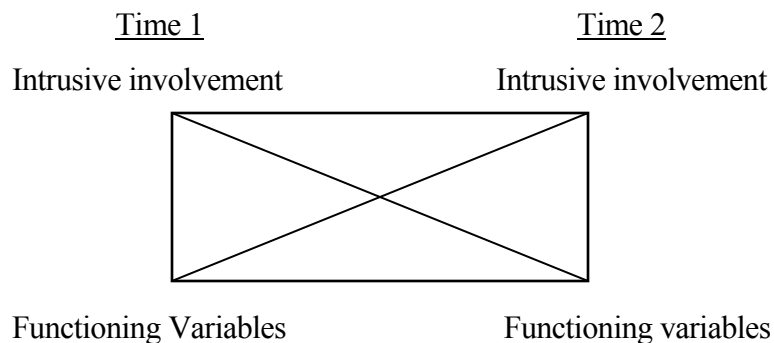
## CHAPTER THREE

### Aims, Rationale and Hypotheses of the Current Study

#### *Aim 1*

#### *Cross-lagged Panel Analyses Exploring Temporal Relationships Between Intrusive Involvement and Child Functioning*

To broaden our understanding of the complex nature of parent-adolescent relationships in the context of diabetes management, the present study examined the relationship between maternal intrusive involvement and child functioning variables (depression, adherence, or metabolic control) across time using a dataset that includes these variables at two points in time. By focusing on concurrent and longitudinal associations between children's perceptions of parental intrusiveness and the quality of their functioning, the present study explored the extent to which each variable predicts intrusive involvement concurrently and longitudinally, as well as the extent to which intrusive involvement predicts child functioning concurrently and across time (see Figure 1).



*Figure 1.* Proposed cross-lagged correlation analysis.



It was hypothesized that concurrent associations would exist at both time points such that intrusive involvement would be associated with higher levels of depressive symptoms, poorer metabolic control and decreased adherence. It was also proposed that patterns of data indicating intrusive involvement at Time 1 as predictive of decreased child functioning at Time 2, without indicating the presence of the reverse association, would lend support to the traditional linear model. Conversely, the absence of any predominant predictive pattern was proposed to indicate, (1) the presence of a spurious relationship between the variables or (2) the workings of a transactional process.

#### *Aim 2*

##### *Proposed Transactional Model Predicting Intrusive Involvement among Trait Anxious Mothers as a Function of Poor Child Functioning*

The current study also examined one potential transactional process linking maternal trait anxiety, intrusive involvement and indicators of child functioning using data from Time 2 of the longitudinal dataset.

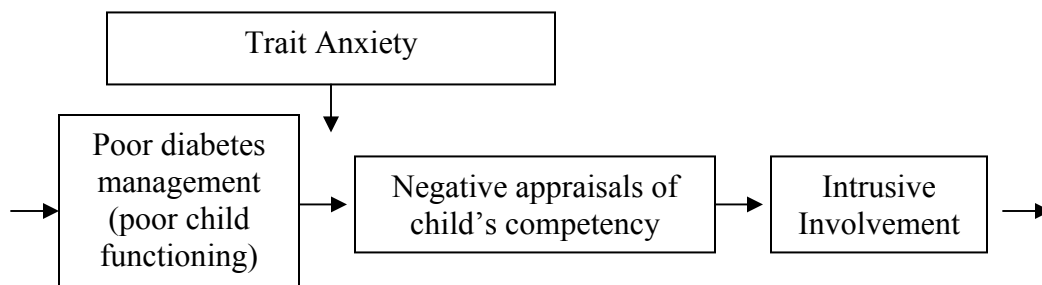
As discussed above, clinicians and researchers have historically explained the development of intrusive behaviors by resting the burden on intrinsic maternal characteristics such as trait anxiety. The term trait anxiety denotes "relatively stable individual differences in anxiety proneness" and refers to a general tendency to respond with anxiety to perceived threats in the environment

(Spielberger, Gorsuch, and Lushene, 1974). In a health context, trait anxiety is manifested in increased symptom detection sensitivity and hypervigilance to health threats (Cameron et al., 2007). Parents with high levels of trait anxiety may enter a “health vigilant” mode more easily when a child is ill, with threat cues and ambiguous events, such as poor child functioning, interpreted negatively (Thomasgard & Metz, 1995).

Within the chronically ill child and adolescent population, maternal trait anxiety has been posited to be strongly associated with intrusive maternal involvement. For example, Thomasgard & Metz (1993) found that parental trait anxiety was the best predictor of intrusive behavior, accounting for 21% of the variance in a regression model. One explanation for this association is that trait-anxious mothers are more likely to perceive their adolescents as deficient in self-care abilities (Dix, 1991), such as diabetes management. These trait anxious mothers also report taking more responsibility for diabetes management tasks and their adolescents report greater beliefs that their mothers are over-protective or intrusively involved (Cameron et al., 2007). Taken together, these findings suggest that trait-anxious mothers’ intrusive involvement may be driven in part by their negative appraisals of the adolescent’s competence in illness management activities.

From the traditional perspective, trait anxiety is considered a fairly stable characteristic that leads mothers to be intrusively involved, which in turn, results

in poor child functioning. From the transactional approach, however, trait anxiety may be one part of a complex model in which many variables play a role. In the proposed transactional model, highly trait anxious mothers who perceive their child as functioning poorly in the context of an episode of mismanagement, may develop negative appraisals of the child's competence (Dix, 1991) and intervene by becoming intrusively involved (Cameron et al., 2007), setting the stage for the following diabetes management episode (see Figure 2). Thus, it was hypothesized that maternal trait anxiety would interact with indicators of poor child functioning to influence more negative appraisals of child competence, and that these negative appraisals would mediate higher levels of intrusive involvement.



*Figure 2.* The proposed transactional model.

While the current study focused exclusively on trait anxiety, it is important to note that other maternal factors such as perceived control (Parker & Lipscombe, 1981), psychological well being (Chaney, Frank, Peterson, Mace, Kashani, and Goldstein, 1997), and social support (Coyne, Wortman & Lehman,

1988) have also been speculated to play a role in causing or exacerbating intrusive involvement and may serve as appropriate variables for future examinations from the transactional perspective.

## CHAPTER FOUR

### Methodology

This study employs a subset of data from an existing data set from the Coping with Diabetes study conducted at the University of Utah (Wiebe et al., unpublished data) as approved by both the Institutional Review Board of the University of Utah and of the University of Texas Southwestern Medical Center at Dallas.

#### *Procedure and Participants*

The existing dataset was collected at two time points per the procedure outlined below (see Palmer et al., 2004; Wiebe et al., 2005; Berg et al., 2007).

#### *Time 1*

Invitations were mailed to potential participants, who were recruited from the Diabetes Clinic at Primary Children's Medical Center and from diabetes camps. In order to be eligible, subjects had to be (a) between the ages of 10 and 15, (b) diagnosed with type 1 diabetes mellitus for at least 1 year, (c) free from any documented serious comorbid medical or psychiatric condition, and (d) able to speak, read and write English. A total of 65 males and 62 females were recruited to participate in this phase. Dyads who met eligibility criteria and who expressed interest in participating in the study were given a packet of questionnaires to be completed at home individually. Each packet contained parental consent forms and assent forms for the adolescents, which granted access

to medical records. Additionally, each dyad attended a laboratory session in which mother and adolescent were asked to complete a series of additional measures and both mother and child were compensated \$20 each for their time.

### *Time 2*

Participants from Time 1 were mailed an invitation packet to participate in a follow-up study, which occurred an average of 16.06 months later. Each packet contained a recruitment letter, consent form and another group of questionnaires, as well as a postage-paid return envelope. Mother and child were each paid \$20 for completing these questionnaires. Questionnaires and consents were returned by 65% of the original participant group.

### *The current study*

The current study was conducted on the subset of 83 children (44 male, 39 female) who completed both phases of data collection. The sample of participants who declined to participate or did not complete both phases did not differ significantly from study participants in illness duration, metabolic control, or age, indicating no apparent selection effects that might impact the generalizability of the results.

The participating sample consisted of families in which parents were predominantly Caucasian (97.6%), married (90.2%), members of the Church of Jesus Christ of Latter-day Saints (LDS or Mormon; 92.9%), middle- to upper-class socioeconomic status (85.1%), with between two and four children (74.4%).

Subjects were fairly evenly divided by gender (52.4% male, 47.6% female), had an average age of 12 years and 10 months ( $SD = 20.53$ ) at Time 1, and tended not to have any other chronic illnesses (79.3%). Within the last 6 months, most subjects had three or fewer low blood sugar reactions (58.9%), tended to miss school three or fewer times during the last 6 months because of their diabetes (71.9%), had not been to the emergency room (85.9%), and had not been hospitalized (85.4%).

Data on glycosylated hemoglobin were available for 55 of the 83 subjects. To determine if those with glycosylated hemoglobin data differed on important demographic and illness variables from those who did not have glycosylated hemoglobin data,  $t$  tests comparing the groups were conducted. Subjects with glycosylated hemoglobin readings were younger than subjects without glycosylated hemoglobin readings,  $t = 2.58$ ,  $p < .05$ . No other significant differences were found. Given the statistically significant difference in age between the two groups, age was taken into consideration in further analyses.

### *Measures*

The questionnaire measures described below are a subset of those included in the original study. Samples of all measures can be found in Appendices A through D.

#### *Intrusive Involvement (Cameron et al., 2007)*

Intrusive involvement was examined using three items from the Impact

subscale of the Diabetes Quality of Life (DQOL) measure for children and adolescents (DCCT, 1988; Cameron et al., 2006). The three items include: (1) “How often do you find that your parents are too protective of you?” (2) “How often do you feel that your parents worry too much about your diabetes?” and (3) “How often do you find that your parents act like diabetes is their disease, not yours?” At both Times 1 and 2, subjects were asked to rate the frequency of their experiences of each item on a scale ranging from (1) *never* to (5) *always* and their answers were summed to generate scores ( $\alpha = .80$  at Time 1,  $\alpha = .78$  at Time 2). Cameron et al. (2006) confirmed that these face valid items are distinct from the other DQOL impact items by conducting a series of principal components analyses with varimax rotation; each analysis included the three parental intrusive involvement items and three of the other 20 subscale items (only three additional items were used in an analysis to ensure sufficient power). The three parental intrusive involvement items loaded onto a common factor (loadings  $> .65$ ) in every analysis, while the other items loaded onto one or two separate and distinct factors.

*Maternal Appraisals of Child Competence (Wiebe, Berg, and Palmer, 2005)*

Maternal appraisals of child diabetes-management competence were assessed using a 24-item scale entitled Diabetes Management Competence. Items were developed based on maternal reports from the 127 Time 1 participants of factors they consider when deciding whether their child is competent to take



responsibility for independent diabetes management (Palmer et al., 2004). The items (e.g., “My child cannot yet adjust his/her own insulin”; “My child makes good choices about food”) were rated from 1 (*strongly disagree*) to 5 (*strongly agree*). Ratings were summed after reverse-scoring as needed, and higher scores reflected more positive beliefs about child competence. This scale shows good reliability ( $\alpha = .87$ ; Wiebe, Berg, & Palmer, 2005). Appraisals of child competence were only available at Time 2.

#### *Spielberger State-Trait Anxiety Inventory*

The Trait Form of the Spielberger State-Trait Anxiety Inventory (STAI; Spielberger, 1983) was used to measure trait anxiety. This is a 20 item self-report measure that taps into a person’s “general” feelings of anxiety. Each item is rated on a 4-point Likert scale and reverse scored as necessary to obtain a total score. The STAI has been used extensively in a variety of settings with various populations since its initial development (Britton, 2005; Huizink et al., 2004) and is one of the most commonly used instruments to assess trait anxiety (Stanley, Beck & Zebb, 1996).

#### *Measures of Child Functioning*

Indices of child functioning included measures of metabolic control obtained from medical records, as well as child-reported adherence and depressive symptomatology.

*Metabolic Control.* Metabolic control was indexed via glycosylated

hemoglobin (HbA<sub>1c</sub>) in a 55-subject subset of the larger study population. HbA<sub>1c</sub> provides information on average blood glucose levels over the preceding 12-16 weeks, and is the current standard to index whether diabetes treatment goals are being achieved. HbA<sub>1c</sub> measurement was a standard part of every clinic visit and was taken every 3-4 months on average. The present study employed the first HbA<sub>1c</sub> measure taken in the 3 to 4 months after each assessment period to ensure it reflected diabetes control subsequent to the assessment.

*Adherence.* The Self Care Inventory (SCI; Greco et al., 1990) is a 14-item self-report measure of adherence that includes all aspects of the type 1 diabetes regimen including blood glucose testing frequency, insulin administration, exercise frequency, and diet. The SCI is commonly used in research on children and adolescents with type 1 diabetes. Participants were asked to rate the extent to which they followed their recommended treatment protocol for diabetes care over the past month, on a 5-point scale ranging from (1) *never did it*, to (5) *always did this as recommended without fail*. A “*not applicable*” response was also available given the personalized nature of the diabetes regimen. Participants were instructed to complete only those items relating specifically to their prescribed care regimen. Ratings were averaged across relevant items so that higher scores reflect greater adherence.

The Self Care Inventory was developed by comparing the results of the self-report measure to diabetics’ responses to two interviews assessing adherence

over the past 24-hours. Glucose testing and exercise frequency were related to corresponding items on the SCI ( $r_s = .80, .39$  respectively,  $p_s < .05$ ). Patterns of relationships with overall regimen adherence and metabolic control were comparable for the two instruments. Mean adherence on the SCI was related to testing frequency on the 24-hour interview ( $r = .71, p < .001$ ) and HbA<sub>1c</sub> was related to testing frequency both on the 24-hour interview ( $r = -.52, p < .01$ ) and on the SCI ( $r = -.54, p < .005$ ). These findings suggest that the Self Care Inventory provides an assessment of adherence comparable to that of a 24-hour interview (Greco et al., 1990). The SCI also has adequate internal consistency (alphas  $> .76$  for adolescent and parent reports; Wysocki, Greco, Harris, Bubb, & White, 2001) and is correlated with metabolic control indices (LaGreca, Follansbee, & Skyler, 1990). This measure was available in the existing set of data at both time points and was completed by both mother and child.

*Depressive Symptomatology.* The Children's Depression Inventory (Kovacs, 1992) is a 27-item self-report symptom-oriented scale designed to appraise depression present in the two weeks prior to administration in children and adolescents between 8 and 17 years of age. Each of the 27 items that compose the CDI describes a different symptom of childhood depression. These symptoms include disturbances in mood and anhedonia, self-evaluation, psychomotor state, functions of daily living including sleep and appetite, and interpersonal behaviors. Several items also evaluate the child's ability to function in the academic

environment. Each item has three potential choices (e.g. 0, 1, or 2) indicating the symptom's absence, occasional occurrence, or frequent occurrence. Scoring involves the addition of numerical values that are assigned to each item response. Total scores reflect overall frequency of depressive symptoms and may range from 0 to 54.

Although the use of the CDI as a diagnostic instrument has yielded mixed results, the CDI is widely used for measuring emotional distress in pediatric populations. The psychometric properties of the CDI are well documented (Smucker, Craighead, Craighead, & Green, 1986). For example, the CDI demonstrates good internal consistency (Chronbach's  $\alpha = .86$ ; Kovacs, 1992), and correlates with structured diagnostic interviews (Garber, 1984). Construct and criterion related validity have both been shown to be robust (Carlson & Cantwell, 1979) and internal consistency of the CDI is adequate (.71 coefficient alpha) for research with a pediatric medical outpatient group (Kovacs, 1985). CDI scores were available in the existing data set at both time points.

#### *Demographic and Illness Factors*

Information on demographic and illness variables (gender, child-age and illness duration) as provided by the mother were considered as potential moderators and/or covariates.

*Gender.* Gender differences in medical and psychological outcomes have been noted in adolescents with diabetes, despite the fact that males and females are at a similar risk of developing the disease (Risch, Ghosh and Todd, 1993). Several

studies observe adolescent girls to evince poorer glycemic control than adolescent boys (Anderson, Miller, Auslander, & Santiago, 1981; Schafer et al., 1983; Simonds, Goldstein, Walker, & Rawlings, 1981). Poorer metabolic control observed in girls cannot be explained by inferior diabetes knowledge, problem solving, or self-care, as girls perform on par with boys in each of these areas (La Greca, Swales, Klemp, Madigan, Skyler, 1995). However, adolescent girls report more symptoms of depression than boys. In fact, both gender and metabolic control were independently and significantly associated with depressive symptomatology, such that girls and those with poor glycemic control reported greater depressive symptoms (Korbel, Wiebe, Berg, and Palmer, 2007; La Greca, Swales, Klemp, Madigan, and Skyler, 1995). Thus, gender may serve as a moderator for psychological functioning and play a role in the relationship between depressive symptomatology and intrusive involvement.

*Child Age.* Child age was also examined as it may act to moderate the effects of intrusive involvement. Research shows that parental control may have different effects in younger versus older youths. For example, parenting behaviors, including restrictiveness, physical punishment and control are not consistently linked to decreased adherence in preschool and elementary school age children (Greening et al., 2006). However, in the adolescent population, increases in maternal intrusive behaviors have been associated with poorer adherence (Gau et al., 2006; Hanson et al., 1987b; Schafer et al., 1983). Thus, parental intrusive

involvement and control may become counterproductive in supporting adherence with increasing child age (Greening et al., 2006).

## CHAPTER FIVE

### Results

All study variables (intrusive involvement, adherence, depression, and metabolic control) and demographic and illness variables (age, gender, illness duration) were evaluated to determine normalcy, and to identify outliers and violations of statistical assumptions. Table 1 contains descriptive information on study variables. Scores on the CDI were skewed; a square root transformation of the CDI at both time points was conducted to improve the normality, linearity, and homoscedasticity of residuals (Tabachnick & Fidell, 2006). All other variables were found to be within the limits of normalcy and to be without significant outliers. No significant gender differences were found for any of the variables.

Zero-order correlations among the main study variables concurrently at each time point are reported in Table 2. At Time 1, intrusive involvement was significantly correlated with poorer adherence, higher depressive symptoms, and poorer (i.e. higher) HbA<sub>1c</sub>. Additionally, all of the functioning variables (adherence, depressive symptomatology and HbA<sub>1c</sub>) were correlated with each other in expected directions but not at levels high enough to suggest the measures are redundant. At Time 2, intrusive involvement was correlated with depressive symptomatology but was no longer associated with adherence or metabolic control. There were no associations between age and any of the variables at Time

1. At Time 2, however, when the sample was older, age was associated with depressive symptomatology such that older subjects were more likely to report depressive symptoms. Gender was found to be unrelated to all variables at both time points.

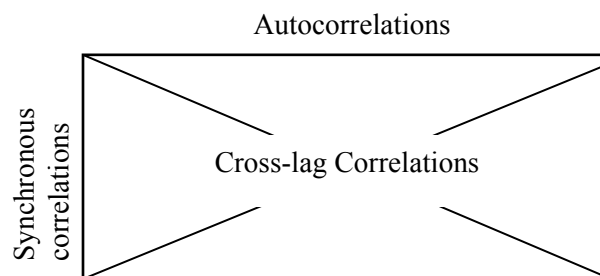
Correlations between variables across time points were also examined and can be found in Table 3. Each variable was significantly correlated with itself across time, indicating some stability in the measures. However, intrusive involvement and adherence appeared to be somewhat less stable than did the CDI or HbA<sub>1c</sub>.

Adolescents who perceived their mothers to be intrusively involved at Time 1 had higher levels of depressive symptomatology at Time 2. However, intrusive involvement at Time 2 was not found to be associated with earlier depression. Similarly, although not statistically significant (potentially a reflection of the small sample with HbA<sub>1c</sub> data at Time 2), intrusive involvement at Time 1 was associated with trends toward poorer HbA<sub>1c</sub> at Time 2 ( $p = .07$ ). Taken together, the pattern of correlations is such that intrusive involvement at Time 1 is associated with poorer well-being at Time 2, while poor functioning at Time 1 is unrelated to intrusive involvement at Time 2.



*Cross-lagged Panel Analyses Exploring Temporal Relationships Between  
Intrusive Involvement and Child Functioning*

Cross-lagged panel correlation (Calsyn, 1976; Calsyn & Kenny, 1977; Clarke-Stewart, 1973; Crano, 1977) was employed to examine the concurrent and longitudinal associations between maternal intrusive involvement and child functioning. As graphically depicted in Figure 3, cross-lagged panel correlation is a statistical technique which, considering synchronous correlations (each variable with the other at the same point in time) and autocorrelations (each variable with itself at two points in time), compares the strength and direction of cross-lags (each variable with the other at a different point in time). If the cross-lags differ significantly, this indicates that there is potentially “a causal factor in X that later causes Y” (Kenny, 1975, p. 891). Differences between cross-lagged correlation coefficients were tested using the Pearson-Filon test (Pelz & Faith, 1973), a test for comparing two non-overlapping dependent correlations. Results from this test are reported as z scores.



*Figure 3.* Graphical depiction of cross-lagged panel analysis

Cross-lagged analysis is “an exploratory approach for pointing to interesting causal hypotheses” and should be viewed as an “indicator of temporal precedence and not as proof positive of causation” (Kenny, 1975). The null hypothesis of cross-lagged panel correlation, tested by setting the cross-lags equal to each other, is that the relationship between two variables is due to an unmeasured third variable. That is, the null hypothesis states that the two variables are not causally related. If the cross-lagged differential is not zero, the null hypothesis is rejected, indicating that the cross-lags differ significantly and there is potentially “a causal factor in X that later causes Y” (Kenny, 1975).

When the null hypothesis is rejected it can also be due to the effects of a third causal variable. The existence of this hypothetical third variable can be ruled out by making two assumptions – synchronicity and stationarity. Synchronicity indicates that the two constructs X and Y are measured at the same point in time – a condition satisfied in the current study. Stationarity assumes that the structural equation for each variable is not different at both points of measurement. In other words, stationarity presumes that the causal processes did not change during the interval measured. Perfect stationarity may be implied when synchronous correlations show no change over time, proportional stationarity means that the causal coefficients of each variable change over time by the same constant, and quasi-stationarity indicates that the causal coefficients of each variable change by

a proportional constant, but each measured variable has its own unique constant (Kenny, 1975).

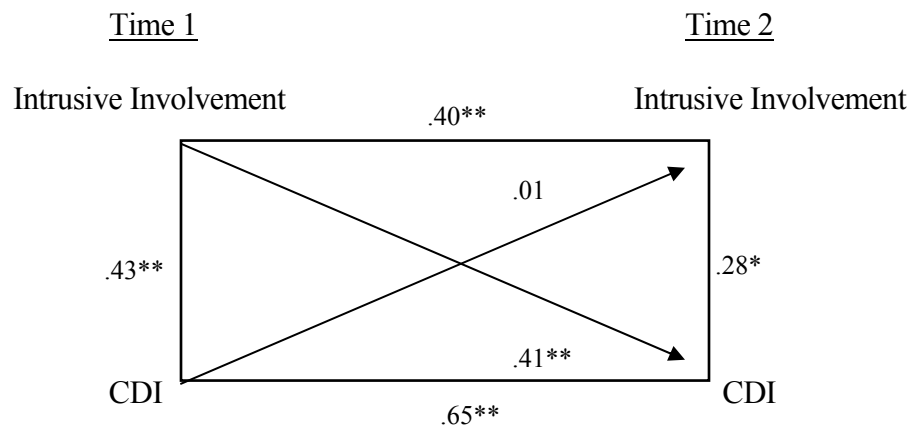
According to Kenny (1975, p. 890) “stationarity is less plausible during periods of rapid growth...for example if there were evidence that the subjects moved into a different stage over time, because a change in stage implies that the causal determinants, and therefore the causal structure, have changed over time” (Kenny, 1979). However, because the sample moves across time together it is possible that we can assume quasi-stationarity for the present study. If quasi-stationarity can be assumed, reliability ratios can be estimated and used to correct the cross-lagged correlations for changes in reliability over time (Kenny, 1975). However, in order to compute reliability ratios, at least three points of measurement are necessary. Due to the nature of the original study, in which there are only two points of measurement, it was impossible to compute reliability ratios in the current study.

In lieu of computing reliability ratios, Cronbach’s alpha coefficients were computed for each variable at each time point in order to examine the internal consistency of each variable over time (see Appendix E). We assumed that clear differences in internal consistency across time would provide evidence that stationarity was not present. The intrusive involvement scale was found to have strong internal consistency at both Time 1 and Time 2 ( $\alpha = .80$ ,  $\alpha = .78$  respectively). The CDI was also found to be internally consistent across time ( $\alpha =$

.90 at Time 1.  $\alpha = .88$  at Time 2). Finally, analysis of the internal consistency of the SCIC indicated Cronbach's  $\alpha = .80$  at Time 1 and  $\alpha = .73$  at Time 2. Given the consistent nature of the measures at both time points, we concluded that the structural equation for each variable was not significantly different at both points of measurement in order to rule out the potential existence of stationarity. However, due to low synchronous correlations for both child reported adherence and metabolic control, potentially a result of small sample sizes and/or developmental effects, results of the cross-lagged analysis for these functioning variables are tentative. Future longitudinal studies are necessary to confirm the results of this study and should include three or more measurements of intrusive involvement and child functioning variables in order to better determine the adequacy of stationarity for analysis.

#### *Associations of Intrusive Involvement with Depressive Symptomatology*

The concurrent and longitudinal associations between maternal intrusive involvement and depressive symptomatology were examined first and the results are displayed in Figure 4.



*Figure 4.* Correlation matrix for intrusive involvement and depressive symptomatology

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\*  $p < .05$ . \*\*  $p < .01$ .

There was a statistically significant difference between the two cross-lags ( $z(81) = 3.27, p < .01$ ). This indicates that intrusive involvement at Time 1 is associated with higher child reports of depressive symptomatology at Time 2, while earlier depressive symptomatology is not associated with higher intrusive involvement at Time 2. To the extent that cross-lagged panel correlations imply a causal relationship, these findings suggest that earlier intrusive involvement predicts later depressive symptoms. These findings lend support to the traditional linear perspective and are not consistent with the presence of a transactional process.

*Associations of Intrusive Involvement with Adherence and Metabolic Control*

Cross-lag correlations of intrusive involvement with child-reported adherence and with metabolic control were not significant. Both cross-lag differentials were also insignificant ( $z(81) = -1.46, p > .05$  and  $z(54) = .16, p > .05$  respectively; see Figures 5 and 6). The absence of a significant cross-lag differential for adherence and metabolic control suggests spuriousness (i.e. the association between intrusive involvement and these functioning variables could reflect an association with an unknown third variable) or might be indicative of the presence of a transactional relationship between intrusive involvement and these disease-related variables (Kenny, 1975). Given the lack of significant synchronous correlations at Time 2, which is potentially indicative of a developmental effect, research with larger sample sizes and more points of measurement is needed to further disentangle the association between these diabetes-specific variables and intrusive involvement.

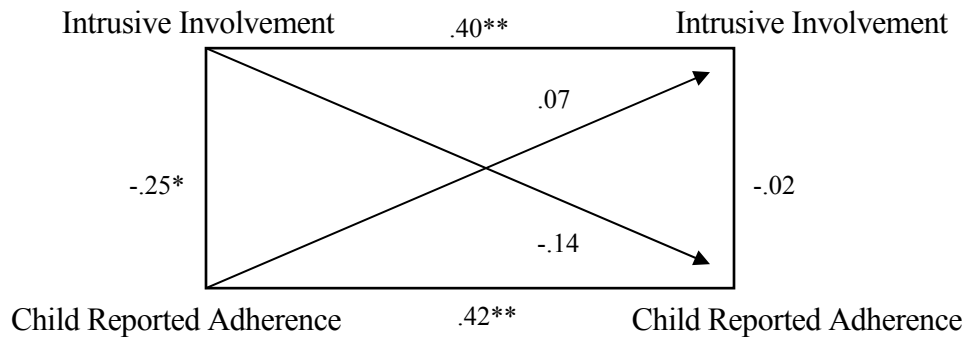


Figure 5. Correlation matrix for intrusive involvement and adherence.

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\*  $p < .05$ . \*\*  $p < .01$ .

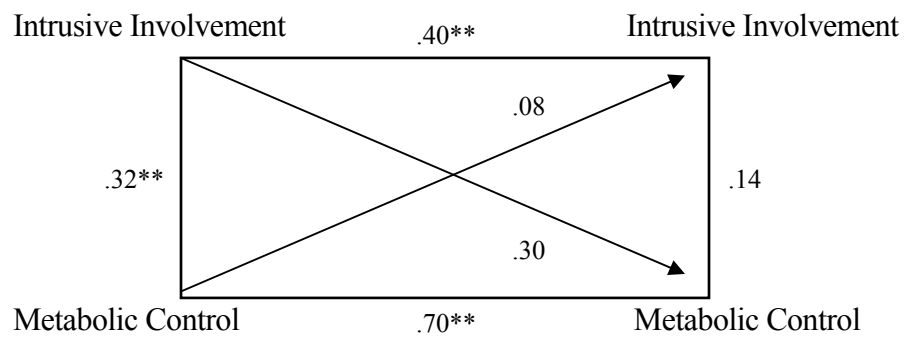


Figure 6. Correlation matrix for intrusive involvement and metabolic control.

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Note.  $N = 55$

\*  $p < .05$ . \*\*  $p < .01$ .

*Regression Analyses Exploring Temporal Relationships Between  
Intrusive Involvement and Child Functioning*

Cross-lagged panel analyses examine the differential strength of associations of two variables across time. They do not, however, examine the extent to which one variable predicts change in another variable across time, and do not lend themselves readily to statistical control of illness or demographic variables. Correlations presented above indicated illness duration was associated with adherence and metabolic control. In addition, exploratory analyses (not reported) suggested that intrusive involvement might have different effects in girls versus boys across age. Thus, in this section, regression analyses were used to examine temporal associations of intrusive involvement with functioning variables while controlling illness duration. We also examined whether associations differed by gender or age.

Three sets of hierarchical linear regression analyses were conducted for each functioning variable. Each set differed in the temporal pattern of association that was tested. First, we examined whether intrusive involvement predicted the functioning variables concurrently at Time 1 to establish the presence of gender or age differences in baseline patterns of associations while controlling for illness duration. Second, we examined whether intrusive involvement at Time 1 predicted functioning variables measured at Time 2. This established whether or not patterns of associations present at Time 1 were also present at Time 2 (i.e.



whether patterns of associations were sustained over time). Finally, we examined whether intrusive involvement at Time 1 predicted functioning variables at Time 2 while controlling for functioning at Time 1 (i.e. whether intrusive involvement is associated with changes in functioning over time).

In each analysis, Time 1 intrusive involvement, illness duration, age and gender were centered on their means (Cohen, Aiken, West & Cohen, 2002) and entered in Step 1 predicting the functioning variable of interest. All two-way interactions (or cross-products) were entered on Step 2 to discern whether patterns of associations differed by age or gender.

#### *Intrusive Involvement Predicting Depressive Symptomatology*

Results of the concurrent regression analysis (intrusive involvement predicting depression at Time1) are presented in the left-hand panel of Table 4. Analyses revealed a main effect for the association between intrusive involvement and concurrent depressive symptomatology,  $t(81) = 2.91, p < .01$ . This main effect for intrusive involvement was moderated by an interaction with gender,  $t(81) = 2.42, p < .05$ . Predicted means were computed by entering the value one standard deviation above or below the mean into the regression equation (Cohen et al., 2002). As shown in Figure 7, girls reported higher depression when they viewed mothers as intrusively involvement, while boys did not.

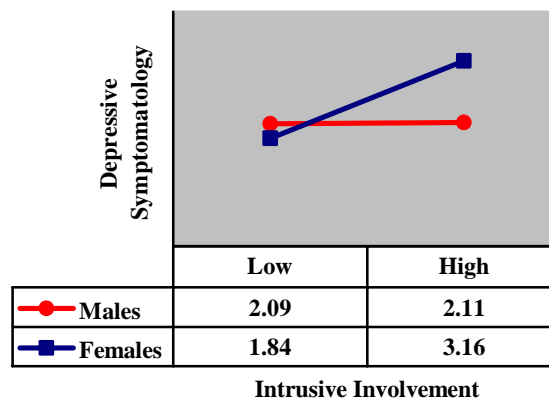
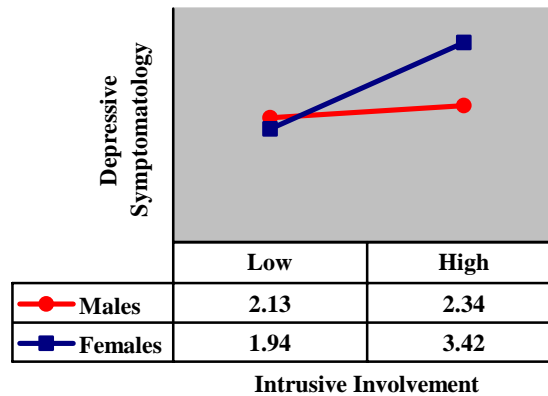


Figure 7. Predicted means for Gender X Intrusive Involvement at Time 1 predicting depressive symptomatology at Time 1

In the initial prospective analysis, as displayed in the middle panel of Table 4, intrusive involvement at Time 1 predicted depressive symptomatology at Time 2,  $t(81) = 2.90, p < .01$ , but this effect was again moderated by gender,  $t(81) = 2.17, p < .05$ . As shown in Figure 8, girls who reported higher intrusive involvement at Time 1 also reported higher depression at Time 2. Intrusive involvement was unrelated to depressive symptomatology among boys.

However, in the third analysis, found in the right-hand panel of Table 4, after covarying for depressive symptomatology at Time 1, the gender by intrusive involvement effect disappeared. This indicates that intrusive involvement is associated with poorer emotional well-being in females at Time 1, an effect that remains over time but does not become stronger or weaker. There appears to be

no association of intrusive involvement and depressive symptomatology for boys at either time point.



*Figure 8.* Predicted means for Gender X Intrusive Involvement at Time 1 predicting depressive symptomatology at Time 2

#### *Intrusive Involvement Predicting Adherence*

As reported in Table 5, concurrent analyses examining the relationship between intrusive involvement and adherence at Time 1 indicated a main effect of intrusive involvement predicting poorer adherence,  $t(81) = -2.34, p < .05$ . This effect was moderated by a marginally significant Gender X Intrusive Involvement interaction,  $t(81) = -1.83, p = .07$ , such that girls reported marginally decreased adherence with higher levels of intrusive involvement, while boys did not (see Figure 9). Neither prospective analysis revealed any predictive value of intrusive involvement at Time 1 on adherence at Time 2.

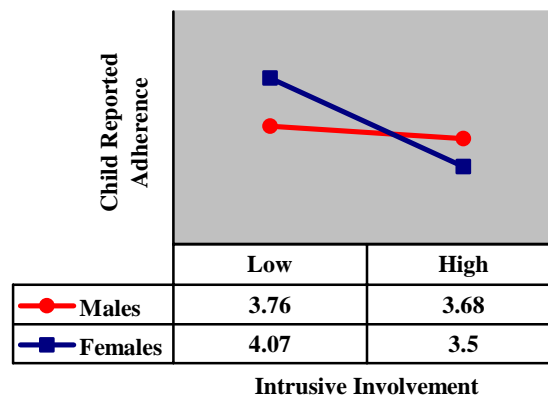


Figure 9. Predicted means for Time 1 Gender X Intrusive Involvement  
Predicting Adherence at Time 1

#### *Intrusive Involvement Predicting Metabolic Control*

Results of the concurrent regression analysis examining the association between intrusive involvement and HbA<sub>1c</sub> indicated that there was a main effect for intrusive involvement predicting concurrent metabolic control at Time 1,  $t(54) = 2.47, p < .05$  (see Table 6, left panel). However, this main effect was moderated by an interaction of intrusive involvement with age,  $t(54) = -2.35, p < .05$ . Predicted means for this unexpected effect are shown in Figure 10. Results indicate that intrusive involvement was associated with poorer metabolic control at Time 1 among younger but not older subjects. Neither prospective analysis revealed any predictive value of Time 1 intrusive involvement on metabolic control at Time 2.

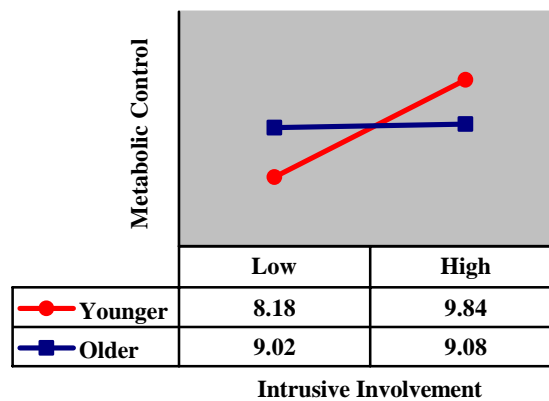


Figure 10. Predicted means for Age X Intrusive Involvement at Time 1

Predicting Metabolic Control at Time 1

*Regression Analyses of Time 1 Functioning Predicting*

*Time 2 Intrusive Involvement*

Additional regression analyses were conducted to examine the reverse pathway in which child functioning variables at Time 1 predicted change in intrusive involvement across time. Values for intrusive involvement at Time 1 were entered into the model to control for the effects of the association between intrusive involvement at Time 1 and Time 2. Parallel to analyses described above, illness duration and interactions with gender and age were also included in the models. As shown in Tables 7 through 9, there was no evidence that child

functioning at Time 1 predicted intrusive involvement at Time 2, thereby further discounting support for the transactional perspective proposed in this study.

### *Testing the Proposed Transactional Model*

Aim 2 explored one potential transactional model linking maternal trait anxiety, intrusive involvement and indicators of child functioning. It was predicted that maternal trait anxiety would interact with each of the functioning measures to predict maternal appraisals of child competence, and that these appraisals would serve as a mediator predicting intrusive involvement. For this moderated mediation effect to be present five conditions are necessary (Aiken & West, 1991; Baron & Kenny, 1986; Holmbeck, 1997; James & Brett, 1984):

- 1) Trait Anxiety X Child Functioning must predict the outcome (Intrusive Involvement).
- 2) Trait Anxiety X Child Functioning must predict the mediator (Appraisals of Competence).
- 3) The mediator (Appraisals of Competence) must predict the outcome (Intrusive Involvement).
- 4) The mediator (Appraisals of Competence) must continue to predict the outcome (Intrusive Involvement) after the interaction term (Trait Anxiety X Child Functioning) is entered into the equation.

- 5) The interaction term (Trait Anxiety X Child Functioning) does not predict the outcome (Intrusive Involvement), or its predictive effects are diminished, once the mediator (Appraisals of Competence) is entered into the equation.

To evaluate whether the Trait Anxiety X Child Functioning interaction term predicted intrusive involvement and appraisals of competence (Requirements 1 and 2), a series of regressions were conducted. In step 1, intrusive involvement was entered as the dependent variable, and duration, trait anxiety and the child functioning variable in question (all centered on their means), were entered as independent variables. In the next step, the interaction term Trait Anxiety X Child Functioning was entered. In violation of the first requirement for moderated mediation analysis, the Trait Anxiety X Child Depressive Symptomatology interaction did not predict intrusive involvement. Similar analyses were conducted looking at the interactions between trait anxiety and the other two functioning variables. None of the functioning variables interacted with maternal trait anxiety to predict intrusive involvement. Consequently, the first requirement of the proposed moderated mediation model was not met, rendering the final four requirements implausible. Results for the first three requirements of the moderated mediation analyses for each functioning variable are reported in Tables 10 through 12.

A supplemental analysis was conducted examining whether maternal appraisals of child adherence would interact with trait anxiety to predict intrusive involvement, where child appraisals of child adherence did not. No interaction predicting intrusive involvement was found. Trait anxiety and child functioning did not interact in a way that is predictive of intrusive involvement or of appraisals of competence, regardless of gender or age. Findings do not support the proposed model as descriptive of a transactional process present in study dyads.



## CHAPTER SIX

### Discussion

The current study set out to examine temporal associations between maternal intrusive involvement and child functioning with the greater goal of discerning transactional processes at work in mother-teen dyads in the context of type 1 diabetes. In contrast to the posited transactional perspective, there was no evidence that any of the child functioning variables at Time 1 predicted intrusive involvement at Time 2. In addition, the proposed transactional model between maternal trait anxiety and child functioning to predict intrusive involvement was not substantiated. However, the finding of a significant cross-lag differential between intrusive involvement and depressive symptomatology lends support to the traditional linear perspective, which views intrusive involvement as a precursor to increased depressive symptomatology.

#### *Intrusive Involvement Associations with Depressive Symptomatology*

Results from the current study suggest intrusive involvement can have detrimental effects on the emotional well-being of girls with type 1 diabetes. Intrusive involvement at Time 1 was associated with higher depressive symptomatology among girls, and these depressive symptoms were unchanged over time. Conversely, reports of intrusive involvement at Time 1 in boys were unrelated to depressive symptoms at either time point. This inconsistency may

reflect a difference in the way that mothers interact with boys versus girls.

Research has shown increased levels of negative maternal control and decreased levels of positive maternal involvement with daughters as compared to sons (Pomerantz & Ruble, 1998a, 1998b; Holmbeck & Hill, 1988). However, in the current study, no gender differences were found for intrusive involvement at either time point.

There may also be a divergence in the way girls and boys interpret intrusive maternal behavior that contributes to the gender differences found in this study. Intrusive involvement may be characterized by both the negative qualities of psychological control (Pomerantz and Eaton, 2000; Pomerantz & Ruble, 1998), and the positive qualities of behavioral control. Behavioral control can have positive consequences for children because it provides them with guidance in understanding and meeting valued standards, and conveys support (Barber, 1996). For example, when parents express worry about their child's diabetes in front of or to the child, the child may interpret this as a communication of care and concern and come to understand the importance of carrying out diabetes tasks responsibly. However, the same behavior may also carry a message that the child is not capable of managing their diabetes independently.

Girls may be more likely to infer a message of incompetence from parent-child diabetes interactions than boys. Girls possess stronger performance standards than boys (Costanzo, Miller-Johnson, & Wencel, 1995, Higgins, 1989)

and are more likely than boys to respond to stressful events by assessing whether they are meeting those standards (Hammen, 1992). In this context, girls may be more likely to interpret maternal efforts to help as an indication that they have failed in their diabetes management. Because girls evaluate themselves more negatively, and are more debilitated after failure feedback than are boys, a maladaptive self-evaluative pattern triggered by psychological control may render girls more vulnerable to depressive symptoms (Ruble, Greulich, Pomerantz & Gochberg, 1993). Nolen-Hoeksema (1987) also proposes that females are more likely to ruminate over their distress while males are more likely to distract themselves. This female style of coping may amplify and prolong depressive symptoms, also leaving females more vulnerable to depression.

Future studies with larger sample sizes will be necessary to better examine the relationship between gender, depression and intrusive involvement in adolescents with type 1 diabetes. However, it remains clear from the findings of this study that early perceptions of intrusive involvement within the diabetes realm are significantly associated with higher levels of depressive symptoms among girls, which are sustained over time. This is consistent with prior studies showing appraisals of maternal control are associated with more depression and poorer quality of life among girls versus boys (Berg, Wiebe et al., 2007; Wiebe et al., 2005).

Finally, one aim of this study was to identify transactional relationships between intrusive involvement and child functioning, however, no transactional pattern between intrusive involvement and depressive symptomatology was found in the current study. There are several possible explanations for this lack of findings. We may not see a relationship between earlier depression and later child perceptions of intrusive involvement due to the nature of the intrusive involvement measure employed in the current study. In this study teens were instructed to answer questions about their perceptions of maternal involvement only with regards to interactions in the context of diabetes management. (e.g., my parents treat my diabetes like it is their own). While adherence and metabolic control are diabetes related indices, depressive symptoms are not necessarily disease specific. When mothers see that their children have poor metabolic control or adhere poorly to their treatment regimen they may be more likely to respond by becoming intrusively involved in their child's diabetes care. Conversely, mothers may respond in different ways when they see their children becoming increasingly depressed, for instance by increasing invisible support, thereby reducing the burden on their child (Bolger, Zuckerman, and Kessler, 2000). It is also plausible that depressed children see their mothers as intrusively involved but that this involvement does not manifest itself in a disease specific way and was therefore not detected by the instrument used in this study.

*Intrusive Involvement Associations with HbA<sub>1c</sub> and Adherence*

Results of cross-lagged panel analyses examining the temporal relationship between intrusive involvement and diabetes-related child functioning (i.e. adherence and metabolic control) indicate non-significant cross-lag differentials, which could either be indicative of a spurious relationship between the variables, or due to a bi-directional association between the variables (Kenny, 1975). This pattern of findings was posited earlier as potentially indicative of a transactional relationship. However, given the lack of significant synchronous correlations at Time 2, cross-lagged panel analyses should be interpreted with caution if at all, as the presence of concurrent relationships at Time 1 not present at Time 2 could be indicative of developmental processes. For example, the goals and life tasks of subjects at Time 1 (ranging in age from 10 to 15) are likely to differ from those of subjects at Time 2 (ranging in age from 12 to 17). Consequently intrusive involvement may be perceived differently and may have different meanings for subjects at each time point. If so, the use of cross-lagged panel analyses are not appropriate and developmental considerations should be taken into account in the interpretation of this panel data.

When the data are analyzed using multiple regression, and Time 1 functioning is included in the regression model, the concurrent relationship between intrusive involvement and diabetes-related child functioning present at Time 1 is no longer present at Time 2. There are several possible explanations for

the lack of a relationship between intrusive involvement and child functioning when Time 1 functioning is taken into account. First, it is possible that the relationship between intrusive involvement and diabetes-related functioning is simply a reflection of covariation with an untested third variable, although it is difficult to conceive of a third variable to explain this pattern of findings. It is also possible that unmeasured moderating variables exist such that associations are present for some youth and their families but not others. We examined the possibilities of both gender and age as moderators and did not find that either moderated the association between intrusive involvement and diabetes-related functioning, but there are conceivably other moderating variables, such as pubertal status, that are yet to be tested.

Finally, no transactional patterns between intrusive involvement and adherence or metabolic control were found in the current study. It is possible that transactional patterns were not found for these variables because the transactional cycle of the association between intrusive involvement and adherence/metabolic control does not map on to the time period assessed by the two points of measurement in the current study. In other words, early intrusive involvement may have detrimental effects on adherence, and poor adherence may prompt action from mothers over short periods of time that are not captured by the 16-month time frame measured presently. For example, adolescents may act in response to intrusive involvement immediately or within days of perceiving

maternal behavior as such, and may therefore not report associated changes in adherence months or years later. Similarly, given the stable nature of HbA<sub>1c</sub>, changes in metabolic control may be longer in the making. Consequently assessments of metabolic control only 16 months (on average) after reports of intrusive involvement may be premature. Future research that includes repeated assessments over time and at different lengths of time may identify transactional processes that were not identified in this study due to this limitation.

*Proposed Transactional Model Predicting Intrusive Involvement in  
Trait Anxious Mothers Given Poor Child Functioning*

Aim 2 of the present study examined one specific theorized transactional process whereby poor child functioning elicits higher levels of intrusive maternal involvement among trait anxious mothers. In the proposed moderated mediation model, trait anxious mothers were hypothesized as more likely to perceive difficulties in child functioning as reflective of low child competence. These perceptions, in turn, were posited to contribute to higher levels of intrusive involvement. In contrast to this prediction, maternal trait anxiety did not interact with any of the functioning variables to predict intrusive involvement. Given that trait anxiety was only available at Time 2, and considering the lack of concurrent associations between intrusive involvement and both adherence and metabolic

control at Time 2, it is possible that this study might have yielded support for the proposed transactional model had the model been tested at Time 1.

The absence of interactions between maternal trait anxiety and child functioning to predict intrusive involvement may also be due in part to the distinction between parenting “practices” and parenting “styles” (Pomerantz & Eaton, 2001). According to Pomerantz and Eaton, practices are “strategies undertaken to achieve specific goals in specific contexts or situations” (2001, p. 175). Hart and colleagues (1997) have suggested that the relationship between parenting practices and child attributes is a reciprocal one in which parents and children are continually influencing one another. For example, parents may engage in the “practice of” intrusive involvement in specific diabetes management contexts to improve their child’s health outcomes. Styles, in contrast, are defined as “aggregates or constellations of behaviors that describe parent-child interactions over a wide range of situations and that are presumed to create a pervasive interactional climate” (Mize & Pettit, 1997, p. 312.) Parenting styles, which arise from parents’ general values and beliefs, may therefore be more stable and thus less likely to respond in a reciprocal manner to child functioning (Costanzo & Woody, 1985; Pomerantz and Ruble, 1998). In the current study, the stable nature of trait anxiety may have precluded it from interacting with any of the functioning variables in a way that predicts intrusive involvement.



### *Limitations*

The present study has several limitations that require consideration when interpreting results. The sample was largely Caucasian, of middle to upper socioeconomic status, predominantly members of the Church of Jesus Christ of Latter-day Saints, and had relatively good metabolic control. Such sample characteristics limit the generalizability of the results to other more diverse populations. Additionally, analyses of metabolic control were based on a sub-sample of the greater study sample due to limitations in data availability. This smaller subject sample may have limited our ability to detect the effects of metabolic control. In fact, patterns of data suggest that future studies with a larger and more heterogeneous subject pool may yield more powerful results.

Due to the nature of the original study, study variables were only available at two time points. To more fully test the potential transactional and evolving nature of the dyadic relationship between teens with chronic illness and their mothers, studies utilizing cross-lagged panel analysis to examine the relationship between intrusive involvement and child functioning should be replicated across at least three different time lags and in different groups of subjects. In addition, more powerful statistical methods to test such models require three or more measures of time.

There are also limitations with regards to the measures utilized in this study. Two of the measures, the intrusive involvement and maternal appraisals of

competence measures, were relatively new and have not been thoroughly studied with regards to reliability and validity. Studies to examine the psychometric properties of these measures are necessary. It is also possible that children experience intrusive involvement in different ways and in different contexts. Given the constraints of the definition of “intrusive involvement” employed in this study, future studies should examine different operationalizations of the intrusive involvement construct (Kenny, 1975).

Finally, while there are good reasons to believe that self-reports from children may be the most valid way to measure intrusive involvement (Barber, 1996), it would also be of interest to determine if maternal controlling behaviors can be observed and, if so, whether perceptions of intrusive involvement are consistent with those behavioral markers. Future research would benefit from the use of multiple informants and measures including behavioral observation of both parent-child interactions and parent behavior to assess intrusive involvement (Holmbeck et al., 2002).

### *Clinical Implications and Conclusions*

Despite the aforementioned limitations, this study contributes to the understanding of the correlates and consequences of intrusive involvement in teens with type 1 diabetes. Findings suggest that early intrusive involvement is associated with higher levels of depressive symptomatology in female adolescents with type 1 diabetes. In light of these findings, and given that intrusive involvement is seen in other chronic illnesses such as cystic fibrosis (Cappelli et al., 1989) and asthma (Parker & Lipscombe, 1979), it will be important to ascertain whether results of this study are generalizable to other chronic illness populations through future research.

In practice, providing psychoeducation to health providers of the increased risk of depression in girls with intrusively involved mothers may lead to greater identification of dyads in need of psychological intervention. This intervention could be in the form of simple psychoeducation at the lowest level, and dyad therapy in mother-daughter pairs for those most at risk. Such interventions may serve to improve mother-daughter relationships and ultimately reduce depressive symptomatology in teenage girls with type 1 diabetes.

## CHAPTER SEVEN

### Tables

Table 1

*Means and Standard Deviations of Study Variables by Gender (N = 82)*

	Sample Mean ( <i>SD</i> )	Males ( <i>N</i> = 43) Mean ( <i>SD</i> )	Females ( <i>N</i> = 38) Mean ( <i>SD</i> )	
Time 1	Child age (in months)	153.67 (20.53)	155.67 (21.12)	151.46 (19.90)
	Intrusive Involvement	7.33 (3.24)	7.24 (3.11)	7.44 (3.42)
	Depression	6.76 (6.26)	5.52 (4.25)	8.13 (7.73)
	Adherence	3.64 (.64)	3.72 (.60)	3.78 (.62)
	Metabolic Control <sup>a</sup>	8.91 (1.37)	8.75 (1.52)	9.06 (1.21)
	Illness Duration	49.13 (32.88)	48.91 (32.02)	49.37 (34.22)
	Hollingshead Index	4.20 (.86)	4.36 (.73)	4.03 (.97)
Time 2	Child age (in months)	169.91 (20.33)	171.93 (20.33)	167.68 (20.37)
	Intrusive Involvement	7.34 (2.86)	7.23 (2.71)	7.46 (3.04)
	Depression	7.59 (6.89)	6.59 (5.89)	8.72 (7.79)
	Adherence	3.72 (.59)	3.66 (.62)	3.79 (.54)
	Metabolic Control	8.63 (1.40)	8.47 (1.53)	8.79 (1.26)
	Maternal Appraisals <sup>b</sup> of Competence	3.72 (.48)	3.71 (.52)	3.74 (.44)
	Maternal Trait Anxiety	39.11 (9.83)	40.89 (10.47)	37.18 (8.82)

<sup>a</sup> Metabolic Control available on 55 participants (28 male, 27 female)

<sup>b</sup> Maternal Appraisals of Competence available on 72 participants.

\*  $p < .05$ . \*\*  $p < .01$ .



Table 3

*Correlations Between Time 1 and Time 2*

		Time 2			
		Intrusive Involvement	Adherence <sup>a</sup>	CDI	HbA <sub>1c</sub> <sup>b</sup>
Time 1	Intrusive Involvement	.40**	-.14	.41**	.30
	Adherence <sup>a</sup>	.07	.42**	-.35**	-.43**
	CDI	.01	-.33**	.65**	.23
	HbA <sub>1c</sub> <sup>b</sup>	.08	-.19	.11	.70**

<sup>a</sup> Adherence as reported by the child.

<sup>b</sup> HbA<sub>1c</sub> measured at least 3 months after the self-report variables, available on 55 participants.

\*  $p < .05.$ , \*\*  $p < .01.$

Table 4

*Regression Analyses for Intrusive Involvement Predicting Depressive Symptomatology (N = 82)*

		<i>b(SE)<sup>p</sup></i>			<i>b(SE)<sup>p</sup></i>			<i>b(SE)<sup>p</sup></i>
<b><u>Intrus. Inv. T1 → CDI T1</u></b>			<b><u>Intrus. Inv. T1 → CDI T2</u></b>			<b><u>Intrus. Inv. T1 → CDI T2</u></b>		
<b>Step 1</b>	$R^2 = .19, F = 4.36^{**}$		<b>Step 1</b>	$R^2 = .24, F = 5.84^{**}$		<b>Step 1</b>	$R^2 = .52, F = 16.53^{**}$	
<b>Ill. Dur.</b>		.01(.00)*	<b>Ill. Dur.</b>		-.00(.00)	<b>Ill. Dur.</b>		-.01(.00)**
<b>Gender</b>		.20(.13)	<b>Gender</b>		.18(.13)	<b>Gender</b>		.04(.11)
<b>Age</b>		.01(.01)	<b>Age</b>		.02(.01)**	<b>Age</b>		.01(.01)**
<b>Intrus. Inv.</b>		.12(.04)**	<b>Intrus. Inv.</b>		.12(.04)**	<b>Intrus. Inv.</b>		.05(.04)
						<b>CDI T1</b>		.64(.09)**
<b>Step 2</b>	$R^2 = .27, F = 3.98^{**}$		<b>Step 2</b>	$R^2 = .30, F = 4.47^{**}$		<b>Step 2</b>	$R^2 = .54, F = 10.38^{**}$	
<b>Gender X Age</b>		.01(.01)	<b>Gender X Age</b>		.00(.01)	<b>Gender X Age</b>		.00(.01)
<b>Gender X Intrus. Inv.</b>		.10(.04)*	<b>Gender X Intrus. Inv.</b>		.10(.05)*	<b>Gender X Intrus. Inv.</b>		.03(.04)
<b>Age X Intrus. Inv.</b>		.00(.00)	<b>Age X Intrus. Inv.</b>		-.00(.00)	<b>Age X Intrus. Inv.</b>		-.00(.00)

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

*Note.* A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .

Table 5

*Regression Analyses for Intrusive Involvement Predicting Child Reported Adherence (N = 82)*

		<i>b(SE)<sup>p</sup></i>			<i>b(SE)<sup>p</sup></i>			<i>b(SE)<sup>p</sup></i>
<b><u>Intrus. Inv. T1 → SCIC T1</u></b>			<b><u>Intrus. Inv. T1 → SCIC T2</u></b>			<b><u>Intrus. Inv. T1 → SCIC T2</u></b>		
<b>Step 1</b>	$R^2 = .19, F = 4.49^{**}$		<b>Step 1</b>	$R^2 = .06, F = 1.26$		<b>Step 1</b>	$R^2 = .24, F = 4.76^{**}$	
<b>Ill. Dur.</b>	-.01(.00) **		<b>Ill. Dur.</b>	-.00(.00)		<b>Ill. Dur.</b>	-.00(.00)	
<b>Gender</b>	.03(.06)		<b>Gender</b>	.07(.07)		<b>Gender</b>	.05(.06)	
<b>Age</b>	-.04(.04)		<b>Age</b>	-.01(.04)		<b>Age</b>	.01(.04)	
<b>Intrus. Inv.</b>	-.06(.02)**		<b>Intrus. Inv.</b>	-.03(.02)		<b>Intrus. Inv.</b>	-.00(.02)	
						<b>SCIC T1</b>	.45(.12)**	
<b>Step 2</b>	$R^2 = .25, F = 3.43^{**}$		<b>Step 2</b>	$R^2 = .07, F = .83$		<b>Step 2</b>	$R^2 = .24, F = 2.89^{**}$	
<b>Gender X Age</b>	-.03(.04)		<b>Gender X Age</b>	-.01(.04)		<b>Gender X Age</b>	.01(.04)	
<b>Gender X Intrus. Inv.</b>	-.04(.02)		<b>Gender X Intrus. Inv.</b>	-.02(.02)		<b>Gender X Intrus. Inv.</b>	-.00(.02)	
<b>Age X Intrus. Inv.</b>	-.00(.01)		<b>Age X Intrus. Inv.</b>	.00(.01)		<b>Age X Intrus. Inv.</b>	.00(.01)	

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

*Note.* A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .



Table 6

Regression Analyses for Intrusive Involvement Predicting Metabolic Control ( $N = 55$ )

$b(SE)^p$		$b(SE)^p$		$b(SE)^p$	
<b><u>Intrus. Inv. T1 → HBA<sub>1C</sub> T1</u></b>		<b><u>Intrus. Inv. T1 → HBA<sub>1C</sub> T2</u></b>		<b><u>Intrus. Inv. T1 → HBA<sub>1C</sub> T2</u></b>	
<b>Step 1</b>	$R^2 = .19, F = 3.47^*$	<b>Step 1</b>	$R^2 = .14, F = 2.04$	<b>Step 1</b>	$R^2 = .52, F = 8.94^{**}$
<b>Ill. Dur.</b>	.01(.01)*	<b>Ill. Dur.</b>	.01(.01)	<b>Ill. Dur.</b>	.01(.01)
<b>Gender</b>	.19(.16)	<b>Gender</b>	.09(.19)	<b>Gender</b>	-.04(.17)
<b>Age</b>	.00(.01)	<b>Age</b>	.00(.01)	<b>Age</b>	.01(.04)
<b>Intrus. Inv.</b>	.15(.05)**	<b>Intrus. Inv.</b>	.14(.06)	<b>Intrus. Inv.</b>	.06(.06)
---		---		<b>HBA<sub>1C</sub> T1</b>	.54(.13)**
<b>Step 2</b>	$R^2 = .28, F = 3.19^{**}$	<b>Step 2</b>	$R^2 = .15, F = 1.19$	<b>Step 2</b>	$R^2 = .73, F = 5.47^{**}$
<b>Gender X Age</b>	.01(.01)	<b>Gender X Age</b>	.00(.01)	<b>Gender X Age</b>	-.00(.01)
<b>Gender X Intrus. Inv.</b>	.04(.05)	<b>Gender X Intrus. Inv.</b>	.04(.07)	<b>Gender X Intrus. Inv.</b>	.05(.06)
<b>Age X Intrus. Inv.</b>	-.01(.00)*	<b>Age X Intrus. Inv.</b>	-.00(.00)	<b>Age X Intrus. Inv.</b>	.00(.00)

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

Note. A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .

Table 7

Regression Analyses for Depressive Symptomatology Predicting Intrusive Involvement ( $N = 82$ )

$b(SE)^p$		$b(SE)^p$		$b(SE)^p$	
<b>CDI T1 → Intrus. Inv. T1</b>		<b>CDI T1 → Intrus. Inv. T2</b>		<b>CDI T1 → Intrus. Inv. T2</b>	
<b>Step 1</b>	$R^2 = .14, F = 3.21^*$	<b>Step 1</b>	$R^2 = .12, F = 2.69^*$	<b>Step 1</b>	$R^2 = .28, F = 6.00^{**}$
<b>Ill. Dur.</b>	-.01(.01)	<b>Ill. Dur.</b>	-.03(.01)**	<b>Ill. Dur.</b>	-.03(.01)**
<b>Gender</b>	-.02(.35)	<b>Gender</b>	.11(.31)	<b>Gender</b>	.12(.28)
<b>Age</b>	.30(.21)	<b>Age</b>	.05(.18)	<b>Age</b>	.01(.01)**
<b>CDI</b>	.86(.29)**	<b>CDI</b>	.10(.26)	<b>CDI</b>	.05(.04)
				<b>Intrus. Inv. T1</b>	.38(.09)**
<b>Step 2</b>	$R^2 = .25, F = 3.55^{**}$	<b>Step 2</b>	$R^2 = .18, F = 2.31^*$	<b>Step 2</b>	$R^2 = .32, F = 4.32^{**}$
<b>Gender X Age</b>	.49(.21)*	<b>Gender X Age</b>	.42(.19)*	<b>Gender X Age</b>	.24(.18)
<b>Gender X CDI</b>	.53(.29)	<b>Gender X CDI</b>	-.19(.27)	<b>Gender X CDI</b>	-.39(.25)
<b>Age X CDI</b>	.02(.16)	<b>Gender X CDI</b>	-.10(.15)	<b>Age X CDI</b>	-.11(.14)

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

Note. A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .

Table 8

*Regression Analyses for Child Reported Adherence Predicting Intrusive Involvement*

		<i>b</i> ( <i>SE</i> ) <sup><i>p</i></sup>			<i>b</i> ( <i>SE</i> ) <sup><i>p</i></sup>			<i>b</i> ( <i>SE</i> ) <sup><i>p</i></sup>
<b><u>SCIC T1 → Intrus. Inv. T1</u></b>			<b><u>SCIC T1 → Intrus. Inv. T2</u></b>			<b><u>SCIC T1 → Intrus. Inv. T2</u></b>		
<b>Step 1</b>	$R^2 = .14, F = 3.07^*$		<b>Step 1</b>	$R^2 = .13, F = 2.82^*$		<b>Step 1</b>	$R^2 = .28, F = 5.82^{**}$	
<b>Ill. Dur.</b>	-.01(.01)		<b>Ill. Dur.</b>	-.03(.01)**		<b>Ill. Dur.</b>	-.03(.01)**	
<b>Gender</b>	.20(.34)		<b>Gender</b>	.14(.30)		<b>Gender</b>	.07(.28)	
<b>Age</b>	.30(.21)		<b>Age</b>	.04(.18)		<b>Age</b>	-.07(.17)	
<b>SCIC</b>	-.06(.02)**		<b>SCIC</b>	-.42(.53)		<b>SCIC</b>	.19(.51)	
						<b>Intrus. Inv. T1</b> 7(.09)**		
<b>Step 2</b>	$R^2 = .25, F = 3.55^{**}$		<b>Step 2</b>	$R^2 = .18, F = 2.34^*$		<b>Step 2</b>	$R^2 = .29, F = 3.69^{**}$	
<b>Gender X Age</b>	.48(.21)*		<b>Gender X Age</b>	.31(.19)		<b>Gender X Age</b>	.15(.18)	
<b>Gender X SCIC</b>	-1.02(.56)		<b>Gender X SCIC</b>	-.47(.52)		<b>Gender X SCIC</b>	-.13(.50)	
<b>Age X SCIC</b>	-.03(.29)		<b>Age X SCIC</b>	-.11(.26)		<b>Age X SCIC</b>	-.10(.25)	

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

Note. A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .

Table 9

*Regression Analyses for Metabolic Control and Intrusive Involvement (N=55)*

<i>b(SE)<sup>p</sup></i>		<i>b(SE)<sup>p</sup></i>		<i>b(SE)<sup>p</sup></i>	
<b><u>HBA<sub>1C</sub> T1 → Intrus. Inv. T1</u></b>		<b><u>HBA<sub>1C</sub> T1 → Intrus. Inv. T2</u></b>		<b><u>HBA<sub>1C</sub> T1 → Intrus. Inv. T2</u></b>	
<b>Step 1</b>	$R^2 = .40, F = 2.88^*$	<b>Step 1</b>	$R^2 = .15, F = 2.72$	<b>Step 1</b>	$R^2 = .39, F = 7.63^{**}$
<b>Ill. Dur.</b>	-.01(.01)	<b>Ill. Dur.</b>	-.04(.01)**	<b>Ill. Dur.</b>	-.03(.01)**
<b>Gender</b>	-.33(.37)	<b>Gender</b>	.07(.35)	<b>Gender</b>	.24(.30)
<b>Age</b>	.32(.21)	<b>Age</b>	.04(.19)	<b>Age</b>	-.12(.17)
<b>HBA<sub>1C</sub></b>	.80(.28)**	<b>HBA<sub>1C</sub></b>	.37(.26)	<b>HBA<sub>1C</sub></b>	-.03(.24)
				<b>Intrus. Inv. T1</b>	.49(.10)**
<b>Step 2</b>	$R^2 = .53, F = 3.11^{**}$	<b>Step 2</b>	$R^2 = .21, F = 2.15$	<b>Step 2</b>	$R^2 = .39, F = 4.62^{**}$
<b>Gender X Age</b>	.58(.23)*	<b>Gender X Age</b>	.40(.22)	<b>Gender X Age</b>	.13(.20)
<b>Gender X HBA<sub>1C</sub></b>	.32(.28)	<b>Gender X HBA<sub>1C</sub></b>	.11(.27)	<b>Gender X HBA<sub>1C</sub></b>	-.04(.24)
<b>Age X HBA<sub>1C</sub></b>	-.20(.15)	<b>Age X HBA<sub>1C</sub></b>	-.15(.15)	<b>Age X HBA<sub>1C</sub></b>	-.05(.13)

Intrus. Inv. = Intrusive Involvement, Ill. Dur. = Illness Duration

*Note.* A priori hypotheses were tested and reported as 2-tailed tests.

\* $p < .05$ . \*\* $p < .01$ .

Table 10

*Moderated Mediation Analyses for Depressive Symptomatology (N = 77)*

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
<u>Trait Anxiety X Child Depressive Symptomatology → Intrusive Involvement</u>					
Step 1				.19	5.82**
Duration	-.03	.01	-2.88**		
Trait Anxiety	.05	.03	1.70		
Child Depression	.09	.04	2.10*		
Step 2				.21	4.74**
Trait Anxiety X Child Depression	-.00	.00	-1.18		
<u>Trait Anxiety X Child Depressive Symptomatology → Appraisals of Competence</u>					
Step 1				.07	1.94
Duration	-.00	.00	-.513		
Trait Anxiety	-.01	.01	-1.82		
Child Depression	-.01	.01	-1.03		

(table continues)

Table 10 (continued)

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Step 2				.09	1.78
Trait Anxiety X Child Depression	-.00	.00	-1.13		
<u>Appraisals of Competence → Intrusive Involvement</u>					
Step 1				.13	5.45**
Duration	-.03	.01	-2.98**		
Competence Appraisals	-1.04	.65	-1.60		

*Note.* A priori hypotheses were tested and reported as 2-tailed tests.

\*  $p < .05$ . \*\* $p < .01$ .

Table 11

*Moderated Mediation Analyses for Adherence (N = 77)*

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
<u>Trait Anxiety X Adherence → Intrusive Involvement</u>					
Step 1				.16	4.59**
Duration	-.03	.01	-3.15**		
Trait Anxiety	.07	.03	2.06*		
Adherence	-.41	.55	-.75		
Step 2				.16	3.40*
Trait Anxiety X Adherence	.01	.07	.15		
<u>Trait Anxiety X Adherence → Appraisals of Competence</u>					
Step 1				.16	4.78**
Duration	.00	.00	.05		
Trait Anxiety	-.01	.01	-2.02*		
Adherence	.28	.09	3.02**		

(table continues)

Table 11 (continued)

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Step 2				.16	3.58**
Trait Anxiety X Adherence	.00	.01	.39		
<u>Appraisals of Competence → Intrusive Involvement</u>					
Step 1				.13	5.45**
Duration	-.03	.01	-2.98**		
Competence Appraisals	-1.04	.65	-1.60		

*Note.* A priori hypotheses were tested and reported as 2-tailed tests.

\*  $p < .05$ . \*\* $p < .01$ .



Table 12

*Moderated Mediation Analyses for Metabolic Control (N = 52)*

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
<u>Trait Anxiety X Metabolic Control → Intrusive Involvement</u>					
Step 1				.13	2.50
Duration	-.02	.01	-2.39*		
Trait Anxiety	.04	.03	1.26		
HbA <sub>1c</sub> <sup>a</sup>	.40	.24	1.67		
Step 2				.13	1.84
Trait Anxiety X HbA <sub>1c</sub>	.00	.02	.18		
<u>Trait Anxiety X Metabolic Control → Appraisals of Competence</u>					
Step 1				.15	2.97*
Duration	.00	.00	1.23		
Trait Anxiety	-.02	.01	-2.27*		
HbA <sub>1c</sub>	-.13	.05	-2.53*		

(table continues)

Table 12 (continued)

	<i>b</i>	<i>SE</i>	<i>t</i>	<i>R</i> <sup>2</sup>	<i>F</i>
Step 2				.17	2.40
Trait Anxiety X HbA <sub>1c</sub>	.00	.00	.86		
<u>Appraisals of Competence → Intrusive Involvement</u>					
Step 1				.13	5.45**
Duration	-.03	.01	-2.98**		
Competence Appraisals	-1.04	.65	-1.60		

*Note.* All a priori hypotheses were tested and reported as 2-tailed tests.

<sup>a</sup>HbA<sub>1c</sub> = Metabolic control measured at least 3 months after the self-report variables, available on 55 participants.

\*  $p < .05$ . \*\* $p < .01$ .

## APPENDICES

## Appendix A

*Intrusive Involvement Measure***1. How often do you find that your parents are too protective of you?**

1 = Never    2 = Sometimes    3 = Often    4 = Frequently    5 = Always

**2. How often do you feel that your parents worry too much about your diabetes?**

1 = Never    2 = Sometimes    3 = Often    4 = Frequently    5 = Always

**3. How often do you find that your parents act like diabetes is their disease, not yours?**

1 = Never    2 = Sometimes    3 = Often    4 = Frequently    5 = Always

## Appendix B

*Maternal Appraisals of Competence*

Below are statements that may or may not describe your child's current abilities and skills related to managing his/her diabetes. For each item, circle one number to indicate how much you agree or disagree with each statement now.

	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
	<b>Strongly Disagree</b>		<b>Neutral</b>		<b>Strongly Agree</b>
1. My child understands why it is important to keep blood sugars down.	1	2	3	4	5
2. My child is comfortable letting us parents be actively involved in managing his/her diabetes.	1	2	3	4	5
3. My child has the cognitive ability necessary to manage his/her diabetes.	1	2	3	4	5
4. It is too early for my child to be managing the diabetes on his/her own.	1	2	3	4	5
5. My child is mature enough to take on the responsibility of managing diabetes.	1	2	3	4	5
6. My child accurately tests his/her blood sugar.	1	2	3	4	5
7. My child remembers to take care of the diabetes without parental prodding.	1	2	3	4	5
8. My child does not understand carb counting.	1	2	3	4	5
9. My child does not yet want the responsibility of managing diabetes on his/her own.	1	2	3	4	5
10. My child cannot yet adjust his/her own insulin.	1	2	3	4	5
11. My child is not comfortable about talking to teachers	1	2	3	4	5

and friends when he/she needs help with the diabetes.

- |   |   |   |   |   |   |
|---|---|---|---|---|---|
| 12. My child is interested and willing to take on the responsibility of managing diabetes.          | 1 | 2 | 3 | 4 | 5 |
| 13. My child is able to draw and give his/her own insulin.  | 1 | 2 | 3 | 4 | 5 |
| 14. My child talks openly with friends and classmates about his/her diabetes.                       | 1 | 2 | 3 | 4 | 5 |
| 15. My child makes good choices about food.   | 1 | 2 | 3 | 4 | 5 |
| 16. My child's diabetes fits easily into his/her social activities.                                 | 1 | 2 | 3 | 4 | 5 |
| 17. My child is not good about keeping his/her blood sugar records.                                 | 1 | 2 | 3 | 4 | 5 |
| 18. My child is too immature to manage diabetes on his/her own.                                     | 1 | 2 | 3 | 4 | 5 |
| 19. My child is too dependent on his/her parents  | 1 | 2 | 3 | 4 | 5 |
| 20. My child's reasoning skills are not developed enough to manage the diabetes by himself/herself. | 1 | 2 | 3 | 4 | 5 |
| 21. My child wants more independence from his/her parents.  | 1 | 2 | 3 | 4 | 5 |
| 22. My child is unaware of complications and where they come from.                                  | 1 | 2 | 3 | 4 | 5 |
| 23. My child's diabetes is interfering with his/her social life                                     | 1 | 2 | 3 | 4 | 5 |
| 24. My child tends to make bad choices about managing diabetes.                                     | 1 | 2 | 3 | 4 | 5 |

## Appendix C

*Self Care Inventory for Children (SCIC)*

Please rate each of the items according to HOW WELL YOU FOLLOWED YOUR RECOMMENDED REGIMEN FOR DIABETES CARE in the past month. Use the following scale:

- 1 = Never did it**  
**2 = Sometimes followed recommendations; mostly not**  
**3 = Followed recommendations about 50% of the time**  
**4 = Usually did this as recommended; occasional lapses**  
**5 = Always did this as recommended without fail**  
**NA = Cannot rate this item/ Not applicable**

In the past month, how well have you followed recommendations for:

1. Glucose testing	1	2	3	4	5	NA
2. Glucose recording	1	2	3	4	5	NA
3. Ketone testing	1	2	3	4	5	NA
4. Administering correct insulin dose	1	2	3	4	5	NA
5. Administering insulin at right time	1	2	3	4	5	NA
6. Adjusting insulin intake based on blood glucose values	1	2	3	4	5	NA
7. Eating the proper foods; sticking to meal plan	1	2	3	4	5	NA
8. Eating meals on time	1	2	3	4	5	NA
9. Eating regular snacks	1	2	3	4	5	NA
10. Carrying quick-acting sugar to treat reactions	1	2	3	4	5	NA
11. Coming in for appointments	1	2	3	4	5	NA
12. Wearing a medic alert ID	1	2	3	4	5	NA
13. Exercising regularly	1	2	3	4	5	NA
14. Exercising strenuously	1	2	3	4	5	NA

## Appendix D

*The Children's Depression Inventory*

This form lists the feelings and ideas in groups. From each group of three sentences, pick one sentence that describes you best for the past two weeks. After you pick a sentence from the first group, go onto the next group.

There is no right answer or wrong answer. Just pick the sentence that *best* describes the way you have been in the past two weeks. Put a mark next to your answer.

## Item 1

- I am sad once a while.
- I am sad many times.
- I am sad all the times.

## Item 2

- Nothing will ever work out for me.
- I am not sure if things will work out for me.
- Things will work out for me O.K.

## Item 3

- I do most things O.K.
- I do many things wrong.
- I do everything wrong.

## Item 4

- I have fun in many things.
- I have fun in some things.
- Nothing is fun at all.

## Item 5

- I am bad all the time.
- I am bad many times.
- I am bad once in a while.

## Item 6

- I think about bad things happening once in a while.
- I worry that bad things will happen to me.
- I am sure that terrible things will happen to me.

## Item 7

- I hate myself.
- I do not like myself.
- I like myself.

## Item 8

- All bad things are my fault.
- Many bad things are my fault.
- Bad things are not usually my fault.



## Item 9

- I don't think about killing myself.
- I think about killing myself but I would not do it.
- I want to kill myself.

## Item 10

- I feel like crying every day.
- I feel like crying many days.
- I feel like crying once in a while.

## Item 11

- Things bother me all the time.
- Things bother me many times.
- Things bother me once in a while.

## Item 12

- I like being with people.
- I do not like being with people many times.
- I do not want to be with people

## Item 13

- I cannot make up my mind about things.
- It is hard to make up my mind about things.
- I make up my mind about things easily.

## Item 14

- I look O.K.
- There are some bad things about my looks.
- I look ugly.

## Item 15

- I have to push myself all the time to do my schoolwork.
- I have to push myself many times to do my schoolwork.
- Doing schoolwork is not a big problem.

## Item 16

- I have trouble sleeping every night.
- I have trouble sleeping many nights.
- I sleep pretty well.

## Item 17

- I am tired once in a while.
- I am tired many days.
- I am tired all the time.

## Item 18

- Most days I do not feel like eating.
- Many days I do not feel like eating.
- I eat pretty well.

## Item 19

- I do not worry about aches and pains.
- I worry about aches and pains many times.
- I worry about aches and pains all the time.

## Item 20

- I do not feel alone.
- I feel alone many times.
- I feel alone all the time.

## Item 21

- I never have fun at school.
- I have fun at school only once in a while.
- I have fun at school many times.

## Item 22

- I have plenty of friends.
- I have some friends but I wish I had more.
- I do not have any friends.

## Item 23

- My schoolwork is alright.
- My schoolwork is not as good as before.

*Item 23 continued*

- I do very badly in subjects I used to be good in.

## Item 24

- I can never be as good as other kids.
- I can be as good as other kids if I want to.
- I am just as good as other kids.

## Item 25

- Nobody really loves me.
- I am not sure if anybody loves me.
- I am sure that somebody loves me.

## Item 26

- I usually do what I am told.
- I do not do what I am told most times.
- I never do what I am told.

## Item 27

- I get along with most people.
- I get into fights many times.
- I get into fights all the time.

## Appendix E

*Means, Standard Deviations, and Cronbach's Alphas for Intrusive Involvement  
and the Three Functioning Variables at Time 1 and Time 2 (N = 82)*

	Time 1	Time 2
<b>Intrusive Involvement</b>		
Mean	7.33	7.34
Standard Deviation	3.24	2.86
Cronbach's alpha	.80	.78
<b>Depressive Symptomatology</b>		
Mean	6.76	7.59
Standard Deviation	6.26	6.89
Cronbach's alpha	.88	.90
<b>Adherence</b>		
Mean	3.64	3.72
Standard Deviation	.64	.59
Cronbach's alpha	.80	.73
<b>Metabolic Control<sup>a</sup></b>		
Mean	8.91	8.63
Standard Deviation	1.37	1.40

<sup>a</sup> Metabolic Control available on 55 participants.

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## **VITAE**

Gabriela Oroza was born in La Paz, Bolivia, on July 27th, 1981, the daughter of Maria Elizabeth Oroza Nostas and Carlos Gabriel Oroza. After graduating from Toll Gate High School, in Warwick, Rhode Island in 1999, she entered Texas A&M University at College Station, Texas. She received the degree of Bachelor of Science with a major in psychology in June, 2003. In August, 2003 she entered the Clinical Psychology Doctoral Program in the Graduate School of Basic Sciences at the University of Texas Southwestern Medical Center at Dallas where she served as Chief Resident from 2006 – 2007. In September of 2007, she married Jarrett Brandon Reed of Amarillo. She was awarded the degree of Doctor of Philosophy in December, 2007 and currently resides in Dallas. Following graduation, she will begin a Postdoctoral Fellowship with Children's Medical Center Dallas' consult-liaison psychiatry service.

Permanent Address: 3219 Trevolle Place  
Dallas, Texas 75204