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Association of Family Structure and Glycemic Control in Adolescents with Type 1 Diabetes: Risk and Protective Factors

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ASSOCIATION OF FAMILY STRUCTURE AND GLYCEMIC CONTROL IN ADOLESCENTS WITH TYPE 1 DIABETES: RISK AND PROTECTIVE FACTORS

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

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Abstract

ASSOCIATION OF FAMILY STRUCTURE AND GLYCEMIC CONTROL IN ADOLESCENTS WITH TYPE 1 DIABETES: RISK AND PROTECTIVE FACTORS

By Laura Jean Caccavale, B.A.
A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University
Virginia Commonwealth University, 2013

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Professor of Psychology
Departments of Psychology, Pediatrics and Psychiatry

Youth with type 1 diabetes (T1D) from single-parent families are more likely to be in poorer glycemic control (HbA1c). Demographic trends indicate more households are composed of unmarried adults and fewer youths. Family density, or a youth: adult ratio, may be a more salient factor than single-parent status in the association with glycemic control. Data from 257 adolescents aged 11-14 years ($M = 12.84$) at two different sites were collected as part of a randomized control trial of a treatment intervention designed to increase parent involvement and prevent deterioration of adolescent diabetes disease care. Single-parent status was determined by parental report of a sole caregiving adult in a youth’s household. A family density ratio was calculated via parental report of the number of youths to adults in a home. A youth: adult ratio greater than two was considered “high family density” (Liaw & Brooks-Gun, 1994). Diabetes-related risk and protective factors of parental monitoring, youth adherence to disease care behaviors, parental stress, and diabetes-related conflict were measured using parent and youth report questionnaires. Glycemic control was determined via a DCA2000 analyzer with results abstracted from medical chart review.
Consistent with the literature, single-parent status was correlated with higher HbA1c ($r = .19, p = .01$) or poorer glycemic control. Similarly, higher family density also was related to higher HbA1c ($r = .32, p < .001$). An overall multiple regression model including family structure constructs (single-parent status and density), socioeconomic status, and ethnicity accounted for 18% of the variance in glycemic control. However, family density, $\beta = .22$, and SES, $\beta = -.29$, were the only significant correlates of glycemic control in the model when considered simultaneously with single-parent status and ethnicity. Although single-parent families have youths in poorer metabolic control, higher family density appears to be a more potent correlate of youth glycemic control perhaps because it might be a more sensitive indicator of available parental time and resources. Family density is significantly related to poorer adherence and greater diabetes-related conflict. Further, poorer adherence and more diabetes-related conflict partially explained the relation between high family density and poorer glycemic control. Family density appears to be an important family structure factor for adolescents with T1D and the identification of risk factors for poorer glycemic control has both clinical and research implications.
Association of Family Structure and Glycemic Control in Adolescents with Type 1 Diabetes: Risk and Protective Factors

Type 1 diabetes (T1D) is a complex chronic illness that affects approximately 1 in 400 to 600 youth in the United States (American Diabetes Association [ADA], 2009). Management of T1D is multifaceted and requires adherence to numerous disease care behaviors which include frequent blood glucose monitoring, insulin administration, and proper nutrition and exercise. The developmental changes associated with adolescence often result in poorer disease care behaviors and subsequently, poorer glycemic control. Thus, it is crucial to study additional factors that may be related to this decline. Family structure has been identified as an important factor related to disease care and glycemic control (Brown et al., 2008). Two distinct factors of family structure, single-parent status and higher family density, will be explored in the current study for their relation to glycemic control among adolescents with T1D.

Adolescents from single-parent families have poorer glycemic control compared to those in two-parent families (Harris, Greco, Wysocki, Elder-Danda & White, 1999; Thompson, Auslander & White, 2001; Thompson, Auslander & White, 2001b). The percentage of American children living in two-parent families has decreased from 85% in 1976 to 69% in 2006; approximately three in ten children live in single-parent homes (Shudy et al., 2006). Mothers account for the majority of single-parents (approximately 85%; U.S. Census Bureau, 2011). Despite this changing demographic, little psychological research on family structure has been conducted. Although marital status is the traditional measure of family structure, in the context of changing family demographics in the United States, marital status may not be the best indicator of parental
time and resources available to allocate to youth with a chronic illness. Higher family density, or a ratio of children to adults that is greater than two to one, is an alternative way to conceptualize the functional association among family structure, disease care management and ultimately, glycemic control. Inclusion of a family density factor may be particularly important for single-parent families of adolescents with TID because parenting multiple children may impact a parent’s ability to be involved in disease care behaviors.

In the current study, single-parent status and family density were examined to determine which better describes the relation of family structure to glycemic control or whether a combination of these two constructs is more descriptive. This report will first describe background information about type 1 diabetes, glycemic control, and disease care. Next, family structure will be introduced along with challenges related to its conceptualization in youth with chronic illnesses. Then, psychosocial factors related to family structure and glycemic control will be evaluated. Parental monitoring, youth adherence to disease care behaviors, parental stress, and diabetes-related conflict may mediate or explain the relation between family structure and glycemic control. Identification of protective factors, such as parental monitoring and better adherence, as well as risk factors, like parental stress and diabetes-related conflict, may determine areas of intervention to assist adolescents and their parents with diabetes management.

**Type 1 Diabetes**

Type 1 Diabetes (TID) is one of the most common chronic illnesses in children and adolescents in the United States. The Centers for Disease Control and Prevention (CDC) estimate that each year, more than 13,000 young people are diagnosed with T1D
Risk factors for the development of T1D include autoimmune, genetic, or environmental factors (CDCP, 2012). Currently no known prevention exists (CDC, 2012). In T1D, the immune system destroys the insulin-producing beta cells of the pancreas. When insulin is no longer produced, the body is unable to regulate blood glucose levels and excess glucose results in the blood (ADA, 2010). Insulin must be delivered by an injection or a pump with adjustments made based on blood glucose levels, which fluctuate with physical activity and eating patterns.

Although the long-term survival of individuals with T1D has dramatically improved in the last 30 years, considerable short- and long-term complications exist (CDC, 2012). Hypoglycemia, or low blood glucose levels, and hyperglycemia, or high blood sugar, result in acute complications such as dizziness, confusion, weakness, hunger, thirst, irritability, nausea, and blurring of vision. Significant chronic complications include retinopathy, neuropathy, and nephropathy (Silverstein et al., 2005). Adolescents with T1D must strike a balance between the undesired extremes of hypoglycemia and hyperglycemia. Adherence to the disease care regimen may lessen the risk of acute and long-term complications.

**Adherence to Disease Care Behaviors**

Adherence to an adolescent’s prescribed medical regimen is a crucial part of managing T1D. Adherence is generally understood as the degree to which a person’s behavior corresponds to health advice (Hood, Peterson, Rohan, & Drotar, 2009). For an adolescent with T1D, a prescribed set of health behaviors includes daily insulin administration, blood glucose monitoring, nutrition, and exercise. Improved adherence to disease care behaviors usually leads to better glycemic control and reduces the risk of
complications. Despite evidence of both short- and long-term benefits of better glycemic control, optimal control is difficult to achieve and adolescents with T1D often struggle to keep HbA1c values within the recommended range (Hood et al., 2009; Silverstein et al., 2005; Springer et al., 2006).

**Glycemic Control**

Glycemic control is assessed via glycosylated hemoglobin (HbA1c). HbA1c is a more useful measure of chronic glycemic control than a single blood glucose reading because it is a composite index of blood glucose readings from the previous two to three month period (Clarke, Snyder, & Nowacek, 1985). HbA1c levels can range from 6% to 14% in adolescents with T1D; lower HbA1c values indicate better glycemic control. The American Diabetes Association (ADA) recommends HbA1c levels below 8% for children ages 6 to 12 and below 7.5% for adolescents ages 13 to 19 (ADA, 2010). A meta-analysis of 21 studies showed that poorer adherence to disease care behaviors resulted in higher HbA1c levels (Hood et al., 2009). Glycemic control and diabetes management become more difficult as adolescents seek greater control and independence in diabetes management (Silverstein et al., 2005).

**Insulin administration.** Individuals with TID must inject exogenous insulin to survive. Insulin is administered via various methods including continuous subcutaneous insulin infusion (CSII), basal/bolus regimens, or multiple daily injections (MDI). In order to maintain better glycemic control and reduce the impact of long-term disease complications, intensive insulin therapy of three or more injections per day or CSII is recommended (DCCT, 1993). A meta-analysis of insulin therapy regimens revealed significantly lower HbA1c levels with continuous subcutaneous insulin infusion (CSII)
compared to multiple daily injection (MDI) therapy or conventional insulin therapy (Weissberg-Benchell, Antisdel-Lomaglio & Seshardi, 2003).

**Blood glucose monitoring.** Monitoring of blood glucose levels provides data on current glucose concentrations and helps determine insulin requirements and guides insulin adjustments to avoid harmful blood glucose fluctuations (Rewers et al., 2007). Blood glucose monitoring is usually completed with a finger prick to draw a drop of blood for a test strip that is read by a blood glucose meter. Self-monitoring of blood glucose is important to try to keep blood glucose levels in the normal range of 80-120 mg/dl (ADA, 2009). If blood glucose values are out of range, corrective action is required such as administration of an insulin injection/bolus or ingestion of food. Blood glucose monitoring allows detection of hypoglycemia and hyperglycemia. The ADA recommends four or more daily blood glucose tests for youth with T1D (ADA, 2010). More frequent monitoring is associated with better glycemic control for adolescents (Anderson, Ho, Brackett, Finkelstein, & Laffel, 1997).

**Nutrition.** Nutritional recommendations for adolescents with T1D are based on general health requirements to promote healthful consumption of essential vitamins and minerals and reduce ingestion of excess fat, particularly saturated fat (American Dietetic Association, 2004). Adolescents with TID also may require individualized meal plans, flexible insulin regimens and algorithms, or nutrition therapy to learn to count carbohydrates. They must monitor nutrition, especially carbohydrate intake to determine insulin needs and to maintain blood glucose goals (Rewers et al., 2007). Little research exists on the relation between nutritional status and glycemic control in adolescents with
T1D. Preliminary evidence suggests a low-glycemic diet reduces hyperglycemia episodes (Rovner, Nansel, & Gellar, 2009).

**Exercise.** The exercise recommendation for all adolescents is 60 minutes of physical activity a day (CDC, 2011). Benefits for adolescents with T1D are similar to those for all individuals, such as a greater sense of well-being, better weight control, improved physical and cardiovascular fitness, and lower blood pressure (Silverstein et al., 2005; Wasserman & Zinman, 1994). Adolescents with T1D should monitor blood glucose levels before, during, and after exercise and adjust insulin and food intake as needed. Hypoglycemia also may occur during physical activity, especially when exercise is greater than usual in frequency, duration, and intensity. Parents should make school teachers, staff, and coaches aware of the risk of hypoglycemia after exercise, the associated symptoms, and the use of emergency resources for treatment (Silverstein et al., 2005).

**Adolescence and T1D**

T1D management often is difficult in adolescence due to biological, physical, cognitive, and emotional changes. Significant deterioration in glycemic control often occurs throughout adolescence (Helgeson, Siminerio, Escobar, & Becker, 2009). Increased insulin resistance during puberty (Ameil et al., 1986) and hormonal changes make it difficult to manage changing insulin requirements (Helgeson et al., 2009).

Developmental factors also may impact adolescent diabetes disease care. Although adolescents may have the cognitive skills to complete diabetes management tasks, adherence to disease care behaviors may be difficult with more attention given to school, extracurricular activities and peers, rather than diabetes management. With
increased independence, parents may have fewer opportunities to interact with their adolescent and impact behavior. Parents may transfer diabetes management to adolescents to decrease family stress (Carroll & Marrero, 2006) such that parental involvement in disease care behaviors may lessen during adolescence (Rubin, Young-Hyman, & Peyrot, 1989).

**Parental monitoring.** During childhood, parents assume the majority of responsibility for disease care management. Parental monitoring and responsibility are related but differ. Monitoring entails regular contact with an adolescent about diabetes care behaviors and knowledge about completion of tasks. Responsibility assumes that a parent assists in task completion (Berg et al., 2008; Dishion & McMahon, 1998). As children get older, parents typically decrease responsibility and switch to greater parental monitoring. Transfer of responsibility and reduction in parental involvement may lead to decreased adherence to disease care behaviors (Holmes, 2006; Johnson & Melzer, 2002; Rubin et al., 1989). Less parental supervision is associated with less frequent blood glucose monitoring, poorer diet, poorer quality of life, and poorer glycemic control (Ellis et al., 2004; Wiebe et al., 2005). Adolescents with more involved parents exhibit better glycemic control (Anderson et al., 2009; Silverstein et al., 2005). Thus, consistent parental monitoring is crucial to maintain good glycemic control through adolescence.

Encouragement of parental involvement is important, but the type of involvement should be adjusted based on a child’s level of development. For older children and adolescents, parents who collaborate with, instead of control, disease care management have youth with better adherence behaviors (Wiebe et al., 2005). Adolescents typically have the skills to perform daily management tasks but may not be capable of certain tasks
such as decision-making about insulin adjustments (Iannotti & Bush, 1993). Parents can provide behavioral assistance with daily tasks and demonstrate problem-solving steps to handle high or low blood glucose levels (Greening, Stoppelbein, & Reeves, 2006). Current recommendations highlight continued appropriate parental monitoring, with shared diabetes management and responsibility (Silverstein et al., 2005).

**Impact of Chronic Illnesses and TID on Families**

Approximately 20-30% of children and adolescents in the United States have a chronic illness such as TID (Brown et al., 2008). Childhood chronic illness influences a parent-child relationship. Parents experience a wide variety of stressors, which include financial worries, physical separations, and adjustments to various components of the medical regimen. Additionally, an entire family may experience interruptions in daily routines, plans for the future and feel general uncertainty with regard to a child’s prognosis (Brown et al., 2008). In turn, these stressors may lead directly or indirectly to parental anxiety, depression, posttraumatic stress, hopelessness, and feelings of loss of control (Kazak et al., 2003). Families are clearly impacted by a child or adolescent’s chronic illness. The burden on single-parent families may be even greater, given the reliance upon a sole caregiver and provider. Family structure can be conceptualized in a number of different ways, most commonly based on parental marital status. Single parent status will first be considered as a traditional conceptualization of family structure along with its financial ramifications, followed by a discussion of family density as a novel construct.

**Family Structure**
**Single-parent families.** Family structure is an increasingly important factor to consider in developmental research, particularly since the landscape of family demographics has changed in the United States. The number of children living in two-parent families has decreased from 85% to 69% from 1976 to 2006, with approximately three in 10 children living in single-parent homes (Shudy et al., 2006). In 2010, 24% of all children in the United States lived in single-mother households (Mather, 2010) and an estimated 59-70% of all children are likely to live in a single-parent household at some point in their lives (Ellwood & Crane, 1990). Part of this demographic shift may be attributable to women under the age of 30, 57% of whom gave birth outside of marriage in 2009 (DeParle & Tavernise, 2012). Although single-mother homes are most common, single-father homes also are increasing (Garasky & Meyer, 1996). Due to these changing demographic trends, better understanding of family functioning in pediatric diabetes necessarily entails an examination of single-parent families.

**Financial Resources.** Single-parent families have on average 55% fewer financial resources than married-parent families (Thomas & Sawhill, 2005). Children in single-parent families are four times more likely to be living in poverty than children in married-parent families, even after controlling for race and ethnicity (Thomas & Sawhill, 2005). However, single-parenthood and poverty are not synonymous. Of all female-headed households, 28% are at or below the poverty line, compared to only 5.5% of married couple households (U.S. Census Bureau, 2005). Although improved employment opportunities are available for women, single-mothers versus married mothers may face challenges with time management, child care, and physical exhaustion.
However, these challenges of single-parenting should not be presumed to exist in every family.

Families of a child with a chronic illness also experience increased financial concerns from health-care costs, medical equipment, travel expenses, and time off from work, each of which may impact socioeconomic status (SES; Montgomery, Oliver, Reisner, & Fallat, 2002; Winthrop, et al., 2005). With a chronic illness in the family, lower SES is related to lower health-related quality of life for children and poorer physical and psychological health for caregivers (Phillips, Dunavant, Lensing, & Rai, 2002; Raina et al., 2005). Single-parents must often deal with the additional financial stressors of a chronic illness without the benefit of another wage earner in the home (Mullins et al., 2010).

As the only caregiver in a household, a single-parent must schedule health care appointments, monitor illness status, administer medications and treatment, in addition to standard household tasks such as cleaning and cooking (Brown et al., 2008). Single-mothers in low income homes suffer from higher rates of distress and depression, often related to employment problems, housing, and discrimination (APA Task Force on Socioeconomic Status, 2007). Higher psychological stress can impact maternal physical health and single-mothers with high amounts of perceived stress are at higher risk for cardiovascular disease and higher cellular oxidative stress (Epel et al., 2004). Additionally, the prevalence of moderate to severe mental disability is higher in single mothers (28.7%) compared to partnered mothers (15.7%; Crosier, Butterworth, & Rodgers, 2007). Sociodemographic factors, household income, financial difficulties, and social support accounted for 94% of the association between single mother status and
poor mental health, with financial difficulties and social support as the strongest predictors (Crosier et al., 2007).

Correspondingly, children in single-parent families may be at greater risk for variety of negative outcomes such as behavioral, social, and emotional problems (Lidner, Hagan & Brown, 1992; Thompson et al., 2001). Children living in single-mother or grandparent-only families tend to have poorer physical and mental health than children living with two biological parents. This relation was not supported for single-father families (Bramlett & Blumberg, 2007), although the small number of single-father families studied to date precludes reliable generalizations at this time.

**Family density.** Family density is an alternative, potentially more comprehensive, way to conceptualize family structure. Density is defined as a family’s total child to adult ratio. A high risk ratio is described as greater than two, and is an identified risk factor for diminished cognitive and behavioral development in premature infants (Liaw & Brooks-Gun, 1994). Thought to reflect the availability of adult resources in the home (Liaw & Brooks-Gun, 1994), no research yet exists in families with T1D youth. With the increase in single-parent families, family density may be another useful measure of available parental resources and time. For example, fewer adults per child in the home may be a significant risk factor for less parental monitoring, which is detrimental for youth with T1D (Ellis et al., 2004; Wiebe et al., 2005). Prior research has not considered family density as a factor related to glycemic control. However, based on literature looking at adolescents with T1D in single-parent families, adolescents in higher density families are likely to have fewer parental and familial resources. Therefore, these adolescents might
experience poorer glycemic control compared to adolescents in lower density families, making this population an important target for research and intervention.

**Sociodemographic Factors, Family Structure, and Glycemic Control**

**Socioeconomic status.** In addition to family structure, SES and ethnicity also should be considered in diabetes research because of the associations among these variables and glycemic control (Mullins et al., 2011). Lower SES may account for a significant part of the high risk associated with single-parent status, perhaps because of limited financial and educational resources. For example, income explained the relations among single-parent status, perceived vulnerability and stress in families of children with chronic illnesses (Mullins et al., 2011). The management of diabetes, which includes the purchase of blood glucose meters and preparation and cost of foods such as vegetables and low fat meats, can be expensive and time consuming. Overall, the care of a child with a chronic illness can add financial concerns for a family because of health-care costs, medical equipment, travel costs, and potential parent time-off from work (Mullins et al., 2011; Winthrop et al., 2005). Management of a child’s chronic illness also requires planning and organization. The relative importance of familial organization in family activities and responsibilities is central to better diabetes management since management is complex and involves daily insulin administration, blood glucose monitoring, nutrition, and exercise (Herge et al., 2012). Single-parents report less family organization (Overstreet et al., 1995), perhaps due to the extra time restraints of being a sole-caregiver.

Further, since SES is calculated using parental education and occupation, lower SES is related to lower parental educational attainment (Hollingshead, 1975). Diabetes is a relatively complex illness to understand and less diabetes knowledge is associated with
poorer adherence (Chisholm et al., 2007). Overall, diabetes management may be more difficult in lower SES families because of fewer resources and less time, poorer family organization, and less education and diabetes knowledge; multiple factors which likely explain the strong relation between lower SES and poorer glycemic control (Auslander, Thompson, Dreitzer, White & Santiago, 1997; Delamater, Albrecht, Postellon & Gutai, 1991; Delamater et al., 1999; Overstreet et al., 1995).

**Ethnicity.** Approximately 66% of African American children, compared to 35% of Caucasian children, live in single-parent, low-income families (Mather, 2010). This sociodemographic confound exists in many diabetes samples, which makes ethnic disparities in glycemic control difficult to untangle. Extensive research shows that Caucasian youth demonstrate better glycemic control than African American youth (Bell et al., 2009; Chalew et al., 2000; Delamater et al., 1999; Mayer-Davis et al., 2009) with differences in HbA1c as great 1.5% (Chalew et al., 2000). However, ethnic differences in glycemic control often are confounded with SES and single-parent status. For example, Thompson and colleagues (2001) found that children of single-parents had lower levels of disease-care adherence compared to children in two-parent families, however, in this prior study, 67% of single-mother families were African American, whereas only 17% of two-parent families were African American. Poorer glycemic control typically attributed to ethnicity may be better explained by lower SES and single-parent status (Frey, Templin, Ellis, Gutai, & Podolski, 2007; Overstreet et al., 1995; Powell, Chen, Streisand, & Holmes, 2012; Swift, Chen, Hershberger, & Holmes, 2006).

**Single-parent status.** For adolescents with TID, living with married biological parents is related to better glycemic control than living with parents in other marital status
arrangements such as single-parent or blended families (Auslander Anderson, Bubb, Jung, & Santiago, 1990; Auslander, Thompson, Dreitzer, & Santiago, 1997; Harris et al., 1999; Thompson et al., 2001), even after statistically controlling factors such as SES and ethnicity (Swift et al., 2006). Youth in single-parent homes often experience dramatic declines in glycemic control five years after diagnosis, at a rate of 0.11% per month (Frey et al., 2007). A year after diagnosis, youth with an average age of 13 in single-parent homes had a 1.34% higher HbA1c compared to youth in two-parent homes and 3.86% higher HbA1c five years after diagnosis (Frey et al., 2007). Even a small reduction in HbA1c is associated with reduced risk of microvascular complications, highlighting the importance of better HbA1c levels (DCCT, 1993). When other factors such as age, pubertal status and Body Mass Index are statistically controlled, single-parent status emerges as the best predictor of glycemic control (Frey et al., 2007).

**Risk and Protective Factors associated with Family Structure**

While the relation between family structure and glycemic control is well documented, less understood are relative risk and protective factors for disease outcomes and whether these factors explain the link between single-parent status and poorer glycemic control. Furthermore, research has not yet considered the construct of family density and its potential contribution to family structure effects. For higher risk family structures, such as single-parent and/or higher density families, important protective factors of more parental monitoring and/or better youth adherence to diabetes care behaviors should be related to better glycemic control. Risk factors for this group may include high levels of parental stress and/or family conflict and should relate to poorer
youth glycemic control. Protective and risk factors may be naturally present or modifiable attributes that are discussed below.

**Parental monitoring and family structure.** Parental monitoring is related to better disease care behaviors and glycemic control (Berg et al., 2008; Green, Mandleco, Roper, Marshall, & Dyches, 2010; Palmer et al., 2010). Mothers typically accompany youth to clinic appointments and are primarily involved in youth disease care (92%; Hilliard et al., 2012). Greater maternal support and monitoring are associated with better youth adherence and glycemic control (Berg, et al., 2008; Hilliard et al., 2012). The complex diabetes medical regimen, acute disease complications, and time demands of a chronic illness may be more taxing for single-parent or higher density families so level of parental monitoring may less in these families.

To date, no studies have examined the level of parental monitoring or involvement in single-parent or high density families of youth with T1D. In the general developmental literature, single-parent families generally show less parental involvement and supervision compared to two-parent families (McLanahan & Sandefur, 1994). Lower monitoring may be a result of single-parents having less time and limited resources compared to families with two-parents who perhaps share parental responsibilities (Auslander et al., 1997). With less parental monitoring, children and adolescents with diabetes are at greater risk for poorer disease care behaviors and negative health consequences.

**Youth adherence and family structure.** Better adherence to disease care behaviors is associated with better glycemic control and reduced long-term complications (DCCT, 1993; Morris et al., 1997). A recent meta-analysis of pediatric T1D found that
while better adherence is linked to better glycemic outcomes, less than half the studies reported the family structure or SES of participants and thus these factors could not be analyzed (Hood et al., 2009). For adolescents with chronic illness, adherence to disease care behaviors is generally poor with adherence levels as low as 50% across different illnesses (Quittner, Espelage, Ievers-Landis, & Drotar, 2000).

Against this background of generally low levels of pediatric adherence in most chronic illnesses, youth with diabetes who live in nontraditional or single-parent families eat less frequent meals and snacks and test their blood glucose levels less often than children in two-parent families (Swift et al., 2006). For single-parent families, better youth adherence appears a likely avenue to improve glycemic control. The relation between higher density families and adherence has not previously been studied but similar associations are hypothesized.

**Parental stress and family structure.** Parents of youth with a chronic illness, such as T1D, often experience stress related to management of their child’s health. Parents report personal stress related to social disruption, emotional strain, and financial strain, especially when their child experiences unpredictable symptoms (Dodgson et al., 2000). Parents with greater responsibility for their child’s diabetes management and greater fear of hypoglycemia report more disease-management stress (Streisand, Swift, Wickmark, Chen, & Holmes, 2005). Increased parental stress can impact both a parent and a child. Parental stress, secondary to caring for a child with diabetes, is associated with an increased risk for poorer parental mental health, such as depressive symptoms and fear (Helgeson, Becker, Escobar, & Siminerio, 2012; Kovacs et al., 1985; Patton, Dolan, Smith, Thomas & Powers, 2011). Parental stress also is related to impairment in
parents’ capability to learn illness-management skills (Gillis, 1993). Higher parental stress, specifically parental anxiety, is related to increased stress for the child or adolescent in a medical setting (Melamed & Ridley-Johnson, 1988) and to poorer adherence to disease care behaviors (Auslander, Thompson, Dreitzer, & Santiago, 1997).

Higher levels of family stress and neighborhood stressors are present in lower income single minority mothers compared to mothers in two-parent families (Thompson et al., 2001). Single-parent families may experience more stress than two-parent families because of financial difficulties, less companionship for the parent, and fewer support networks (McLanahan, 1983). Frequency of parenting stress is higher in single-parents, those with younger children, and those with lower family socioeconomic status (Streisand et al., 2005). To our knowledge, no research has examined the association of family density with parental stress in families of adolescents with T1D.

**Diabetes-related conflict and family structure.** Family conflict is associated with poorer adherence and glycemic control in adolescents with T1D (Wysocki, 1993). Adolescents often assert independence and desire more control over their diabetes management, which can result in greater conflict (Schilling, Knafl, & Grey, 2006). Disagreements between mothers and adolescents over who is responsible for diabetes management and supervision are significant predictors of poorer glycemic control for adolescents (Anderson, Auslander, Jung, Miller & Santiago, 1990). Family interventions that reduce family conflict demonstrate improved disease-related outcomes (Anderson et al., 1997; Wysocki et al., 2006).

The association between diabetes-related conflict and poorer adherence and glycemic control is well documented in the literature, and without intervention, may
persist (Anderson, 2004; Hilliard, Guilfoyle, Dolan & Hood, 2011; Hilliard et al., 2012; Hood, Butler, Anderson & Laffel, 2007). Typical family arguments, as well as conflict over diabetes management, may negatively impact adolescent adherence. Diabetes-specific family conflict predicts poorer adolescent adherence six months later and poorer glycemic control twelve months later (Hilliard et al., 2011). Family conflict is positively associated with poorer glycemic control and parental monitoring and conflict are inversely correlated (Hilliard et al., 2012).

Adolescents from single-parent families and blended families reported more conflict and less cohesion than adolescents from two-parent families (Overstreet et al., 1995). Cohesive family relationships, characterized by better communication, cooperation, and social reinforcement among members, could reduce difficulties that a single-parent may have in monitoring an adolescent’s disease care behaviors. In contrast, higher levels of diabetes-related conflict likely increase stressors associated with single-parenthood (Swift et al., 2006).

**Statement of the Problem**

As the number of single-parent families increases in America, study of the role of family structure on daily family life is warranted. T1D is one of the most common pediatric chronic illnesses and if well managed, better glycemic control relates to better health and prevention of disease complications. The present study evaluated two different family structure constructs, single-parent status and family density, to identify the most sensitive demographic descriptor or combination of descriptors that best capture the association between number of caregivers and glycemic control in adolescents. Single-parent status and poorer glycemic control often co-occur but in light of changing
demographic trends in family structure, examination of the ratio of children to adults, i.e., family density, may better capture the risk and protective factors related to daily disease management and ultimately to glycemic control. Alternatively, the two measures of family structure, single-parent status and family density, may be more powerful when viewed in tandem.

For adolescents with T1D, parental involvement in disease management and adherence to the diabetes regimen are protective factors related to better glycemic control. Although age-related deterioration in HbA1c often occurs during adolescence, in part related to hormonal fluctuations, behavioral factors also contribute to poorer glycemic control. Youth may assert independence from parents and parental monitoring often diminishes, which could result in poorer compliance with diabetes-related tasks. Adolescents have better glycemic control when parents stay involved in diabetes management. Adolescents in single-parent families have poorer adherence compared to adolescents in two-parent homes perhaps because single-parents are less involved in diabetes care. The relations among family structure, parental monitoring, and diabetes adherence were further examined in this study. Potential risk factors for single-parent families include greater parental stress and diabetes-related conflict, either one of which may relate to less parental monitoring in youth disease care and to poorer youth adherence.

HYPOTHESES

Exploration of Family Structure Constructs
1. Single-parent status and higher family density alone and in combination will be associated with poorer glycemic control. However, family density will be a stronger correlate of glycemic control.

**Family Structure and Protective and Risk Factors**

2. Single-parent and higher density family structure will be significant correlates of less parental monitoring, lower adherence, greater parental stress, and diabetes-related conflict after controlling the effects of socioeconomic status (SES) and ethnicity.

**Mediators of Family Structure and Disease Outcomes**

3. Levels of parental monitoring, youth adherence, parental stress, and diabetes-related conflict will each mediate the relation between family structure and glycemic control.

**Method**

**Participants**

Participants are 257 adolescents between ages 11 and 14 and a parent or primary caregiver recruited from two metropolitan pediatric endocrinology clinics. Eligibility criteria included a diagnosis of T1D for at least one year prior to enrollment in the study, no other chronic illness or injury, and fluency in reading and writing English. Participant data were previously collected as part of a longitudinal randomized clinical trial (RCT) of a treatment program designed to increase parent involvement and prevent deterioration in adolescent diabetes disease care. All data used are from baseline evaluation. This study was approved by all appropriate Institutional Review Boards. See Table 1 for demographic characteristics.
**Procedure**

Families of eligible participants were identified from clinic schedules during a two-and-a-half year recruitment period and were sent a recruitment letter detailing study involvement. Families were then contacted by phone and invited to participate. If a parent and adolescent agreed, an assessment was scheduled concurrently with the next upcoming medical appointment. After written informed parental consent and youth assent were obtained, trained research staff interviewed an adolescent and parent separately via interview and administered a battery of questionnaires. After completion of the baseline assessment, families received a $25 gift card.

**Measures**

**Demographic information.** Demographic and medical questionnaires included information such as ethnicity, age, marital status, age of disease onset, disease duration, household composition, and SES. A parent reported the number of parents and children in the household and marital status. SES was measured using the Hollingshead Index of Socio-Economic Status (Hollingshead, 1975). A SES score was calculated for each family (ranging from 8-66) from reported parental education level and occupation, with higher scores indicating higher SES.

**Family Structure.** Single-parent status was measured via self-report on a parent-completed demographic questionnaire that asked about marital status. Single-parent status was determined if the parent reported being separated, divorced, widowed or never married. Family density was measured based on parent-report of the number of parents in the home and number of other children beside the youth with diabetes that lives in the household. A ratio of children to parents in the household was created. A child-parent
ratio greater than two is considered “higher family density” to reflect both family size and indirectly the availability of human resources in the home (Liaw & Brooks-Gun, 1994).

**Parental Monitoring.** The Parental Monitoring of Diabetes Care Scale (PMDS; Ellis et al., 2008) is a self-report questionnaire that measures parental monitoring in adolescent daily diabetes management and care. There are 18 items rated on a five point Likert Scale (1 = *more than once a day* to 5 = *less than once a week*) and a final 19th item that is open-ended. Subscales include Supervision of the Availability of Medical Supplies/Devices, Monitoring of Blood Glucose Checking, Oversight of Diet, Monitoring of Nonadherence, and Direct Oversight of Diabetes Management Behaviors. Scores range from 18 to 90 with higher scores indicating greater parental monitoring and involvement. Parents and adolescents completed the questionnaire separately and their responses are averaged for a composite parental involvement score. The PMDS has acceptable normative internal consistency (α = .81) and test-retest reliability (ICC = .80; Ellis et al., 2008).

**Adherence.** Disease care adherence was assessed with the Diabetes Behavioral Rating Scale (DBRS; Iannotti et al., 2006), which was administered to parents and adolescents separately. The DBRS was developed as a self-report measure of disease care for parent and youth completion. The four subscales are: Daily Prevention Behaviors (0 = *never* to 4 = *always*), Modification of Diabetes Care Plan (0 = *never* to 5 = *five times*), Intervention Behaviors (0 = *none* to 5 = *five times*), and Other Diabetes Care Practices (0 = *never* to 5 = *five times*). The pump version of the measure consists of 37 items with a maximum score of 161 and the non-pump version consists of 36 items with a maximum score of 157, with higher scores reflecting greater adherence (normative $M = 75$, $SD =$
The DBRS shows satisfactory normative internal consistency (.84), test-retest reliability (ICC = .71), and parent-adolescent agreement (.48; Iannotti et al., 2006).

**Parenting Stress.** Parental stress was self-reported with the Pediatric Inventory for Parents (PIP; Streisand, Braniecki, Tercyak, & Kazak, 2001), designed to measure frequency and difficulty of stress experienced by parents of youth with chronic or acute illness. The scale includes 42 items for which participants indicate “frequency” and “difficulty of each item on a scale from one to five (1 = never or not at all to 5 = very often or extremely). A total score for frequency and for difficulty is comprised of four domains: communication, emotional distress, medical care and role function. Scores from the test standardization data range from 42 to 210. The Cronbach’s alpha coefficient was .95 (Lewin et al., 2005).

**Diabetes-related conflict.** The Diabetes Family Conflict Scale- Revised (DFCS-R; Hood et al., 2007) was completed by parents and youth separately to measure diabetes-related conflict. The DFCS-R consists of 19 items that ask the frequency of conflict surrounding diabetes-related management tasks, using a 3-point Likert-type scale from ‘never’ to ‘almost never’ (scale range of 19, no conflict, to 57, high conflict). The DFCS-R includes two subscales: direct management and indirect management. Responses are averaged for a final diabetes-related conflict score. The DFCS-R has appropriate construct validity, normative internal consistency, concurrent validity, and predictive validity (Hood et al., 2007).

**Glycemic Control.** Glycemic control was determined via medical chart reviews that coincided with the time of assessment. A Bayer DCA 2000 Analyzer was used to measure the HbA1c level at the time of the youth’s appointment. The HbA1c results
from the DCA 2000 Analyzer are strongly correlated with the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications central laboratory values, \( r = .94, p < 0.001 \), which are a standard comparison to other assays (Tamborlane et al., 2005). HbA1c levels indicate average blood glucose concentration over the previous three months. A higher HbA1c value suggests poorer glycemic control. The recommended HbA1c level for adolescents is below 7.5% (ADA, 2010).

**Results**

**Preliminary Analyses**

Analyses were performed in order to determine the sample size needed to adequately power the current study. The following analyses had a total of nine predictor variables, including confounded variables. Using 10:1 cases to predictor ratio, a sample size of 157 is sufficient for the proposed analyses. Power analyses were further calculated using G*Power 3 software. With desired level of power set at .80, alpha level at .05, and a small expected effect size at .3, there will be adequate power with a sample size of 64 (Cohen, 1988). Thus the sample size of 257 is appropriate to power the current study.

All variables were assessed for univariate normality and multivariate outliers. None of the continuous predictor variables revealed skewness or kurtosis values above +/- 1.5 or standard values greater than +/- 3.43.

**Descriptive Results**

Participants were 257 adolescents (51% male) aged 11 to 14 \( (M = 12.84, SD = 1.24) \) with TID and a primary caregiver. The majority of the adolescents were Caucasian (69.9%) and from middle socioeconomic status families (77.5% Middle or Upper-
middle SES status; \( M = 46.61, SD = 11.73 \). The average duration of T1D was 5.12 years (SD = 3.06) and the average HbA1c was 8.80 % (SD = 1.61). Almost half of the adolescents reported use of an insulin pump (44.0 %). Evaluation of family structure revealed 23 % of adolescents were from single-parent families and 10.5 % were in higher (ratio of children to adults > 2) density families. Participant demographic and disease characteristics are included in Table 1. Means and standard deviations for study measures of parental monitoring, adherence, parental stress, and diabetes-related conflict are included in Table 2.

Table 1.

**Sociodemographic and Disease Characteristics of Participants (N = 257)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>130 (50.6 %)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>179 (69.6 %)</td>
</tr>
<tr>
<td>African American</td>
<td>49 (19.1 %)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>14 (5.4 %)</td>
</tr>
<tr>
<td>Asian/Asian American</td>
<td>5 (1.9 %)</td>
</tr>
<tr>
<td>Other</td>
<td>10 (3.9 %)</td>
</tr>
<tr>
<td>Hollingshead Index of SES</td>
<td></td>
</tr>
<tr>
<td>Upper 60-69</td>
<td>30 (11.7 %)</td>
</tr>
<tr>
<td>Upper-middle 48-59</td>
<td>103 (40.1 %)</td>
</tr>
<tr>
<td>Middle 29-47</td>
<td>96 (37.4 %)</td>
</tr>
<tr>
<td>Lower-middle 18-28</td>
<td>12 (4.7 %)</td>
</tr>
<tr>
<td>Lower 8-17</td>
<td>7 (2.7 %)</td>
</tr>
<tr>
<td>Insulin Regimen</td>
<td></td>
</tr>
<tr>
<td>CSII</td>
<td>113 (44.0 %)</td>
</tr>
<tr>
<td>BB injections</td>
<td>52 (20.2 %)</td>
</tr>
<tr>
<td>Conventional 2-3 injections</td>
<td>90 (35.0 %)</td>
</tr>
<tr>
<td>Single Parent Families</td>
<td>58 (23 %)</td>
</tr>
<tr>
<td>Higher Density Families</td>
<td>27 (11 %)</td>
</tr>
<tr>
<td>Variable</td>
<td>$M (SD)$</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------</td>
</tr>
<tr>
<td>SES</td>
<td>46.61 (11.73)</td>
</tr>
<tr>
<td>Youth age</td>
<td>12.84 (1.24)</td>
</tr>
<tr>
<td>T1D duration</td>
<td>5.12 (3.06)</td>
</tr>
<tr>
<td>HbA1c</td>
<td>8.80 (1.61)</td>
</tr>
</tbody>
</table>

*Note. SES = socioeconomic status, higher scores indicate higher SES
CSII = Continuous subcutaneous insulin infusion, a type of continuous blood glucose injection; BB injections = basal-bolus injection regimen, a type of multiple daily insulin injection therapy
HbA1c = Glycated hemoglobin, a measure of glycemic control

Table 2.

*Descriptive Data for Diabetes Factors*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$M (SD)$</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parental Monitoring</td>
<td>77.96 (6.70)</td>
<td>55.50 - 89.50</td>
</tr>
<tr>
<td>Youth Adherence</td>
<td>0.65 (0.10)</td>
<td>0.35 – 0.92</td>
</tr>
<tr>
<td>Parental Stress</td>
<td>86.97 (24.06)</td>
<td>42.50 – 159.15</td>
</tr>
<tr>
<td>Diabetes-related conflict</td>
<td>27.07 (7.12)</td>
<td>19.00 – 51.50</td>
</tr>
</tbody>
</table>

Exploration of Family Structure Constructs: Hypothesis 1

A chi-square test of independence revealed a significant relation between single-parent status and family density, $X^2 (2) = 47.53, p < .001; N = 244$. Adolescents in single-parent families were more likely to live in higher density families. However, only 8.2%
(n = 20) of adolescents lived in families that were both single-parent and higher density. The majority of adolescents resided in two-parent, lower density families (75%, n = 183).

Next, Pearson’s correlations (r) and biserial correlations were calculated among single-parent status, family density, ethnicity, SES, and HbA1c to determine if family structure factors were confounded and to determine which provides a stronger association with glycemic control. Single-parent status and family density were each significantly related to ethnicity (r = -.29, p < .001; r = .23 and p < .001, respectively), such that White race/ethnicity (compared to other ethnic groups) was associated with two-parent status and lower family density. Similarly, single-parent status and family density were both related to SES (r = .36, p < .001 and r = -.19, p = .004, respectively), with higher SES associated with two-parent status and lower family density. Additionally, both family structure factors were significant correlates of lower HbA1c such that single-parent status (r = -.21, p = .001) and higher family density (r = .29, p < .001) were each associated with poorer glycemic control (see Table 3).

Table 3.

Correlation Matrix with Primary Study Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.    Single-Parent Status</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.    Family Density</td>
<td>-.44***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.    SES</td>
<td></td>
<td>.36***</td>
<td>-.19**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.    Ethnicity</td>
<td>-.29***</td>
<td>.23***</td>
<td>-.33***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.    HbA1c</td>
<td>-.21***</td>
<td>.29***</td>
<td>-.35***</td>
<td>.23***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.    Parental Monitoring</td>
<td>.10</td>
<td>-.12</td>
<td>.07</td>
<td>-.15*</td>
<td>-.21**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.    Adherence</td>
<td>.14*</td>
<td>-.20**</td>
<td>.31***</td>
<td>-.36***</td>
<td>-.27***</td>
<td>.31***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.    Parental Stress</td>
<td>-.16**</td>
<td>.11</td>
<td>-.21***</td>
<td>.18**</td>
<td>.15</td>
<td>-.17</td>
<td>-.18**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.    Diabetes Related Family Conflict</td>
<td>-.15*</td>
<td>.17**</td>
<td>-.30***</td>
<td>.23***</td>
<td>.31***</td>
<td>-.20</td>
<td>-.25***</td>
<td>.32***</td>
<td></td>
</tr>
</tbody>
</table>

---
In order to determine the best conceptualization of the family structure factor for mediation analyses, multiple regressions were used to determine the unique contribution provided by each factor of family structure to HbA1c. First, only single-parent status and family density were included as independent variables in the model. Together, these family structure constructs were significantly related to HbA1c, $F(2, 241) = 12.63, p < .001$, $R^2 = .095$ and accounted for 9.5% of the variance in HbA1c. In this model, family density was the only significant correlate, $t(241) = 3.58, p < .001, \beta = .24$; single-parent status was not a significant correlate once the effects of family density were considered $t(241) = -1.59, p = .114, \beta = -.12$. A multiple regression was then conducted to examine the family structure factors with ethnicity and SES included as covariates. The overall model with single-parent status, family density, ethnicity, and SES was significant, $F(4, 230) = 12.61, p < .001$, $R^2 = .18$. Regression analysis revealed that 18% of the variance in HbA1c was explained by the model. Family density ($t(230) = 3.26, p = .001$) and SES ($t(230) = 4.40, p < .001$) were significant predictors of HbA1c; however, neither single-parent status ($t(230) = .18, p = .858$) nor ethnicity ($t(230) = -1.25, p = .212$) were significant correlates in this model. SES had a larger effect size ($\beta = -.29, p < .001$) compared to family density ($\beta = .22, p = .001$) in the regression analyses.

A two-way analysis of variance (ANOVA) was performed to compare HbA1c levels of adolescents living in single-parent, two-parent, high density and low density ethnicities.
families. Results indicated a main effect of family density \((F(1, 240) = 11.50, p = .001)\). Adolescents living in lower density families had significantly lower HbA1c values \((M = 8.63\%, SD = 1.52)\) compared to adolescents in higher density families \((M = 10.16\%, SD = 2.0)\). There was no main effect of single-parent status \((F(1, 240) = 1.37, p = .243)\). Although the mean HbA1c of adolescents living in single-parent, high density families was greater than other groups \((M = 10.29\%, SD = 2.05)\), the interaction effect of family density and single-parent status was not significant \((F(1, 240) = .002, p = .967)\).

A new variable, family structure combined, was created to look at the effect of single-parent status and family density simultaneously. Family structure combined was constructed from an interaction between single-parent status and family density with higher values representing single-parent higher density families. As expected, regression analyses revealed that family structure combined was significantly associated with glycemic control \(t(242) = 4.82, p < .001, \beta = .296\). However, the magnitude of this association was similar to the magnitude of the relation between family density and glycemic control \(t(242) = 4.75, p < .001, \beta = .292\). These comparable results suggest that although the combination term is a significant correlate of glycemic control, it may not be a more meaningful way to understand family structure over the simpler construct of family density.

These results provide evidence that single-parent status and higher family density are each associated with poorer glycemic control, thus supporting the first hypothesis. However, family density appears to be a more sensitive correlate of glycemic control. Perhaps given the small number of adolescents who live in both single-parent and high density families \((n = 20)\), no significant effect was found for the interaction term of
single-parent status and family density. Family density will be used as the family
structure indicator in the mediation analyses of Hypothesis 3.

**Family Structure and Protective and Risk Factors: Hypothesis 2**

**Family density and diabetes care factors.** Multiple regressions were performed
next with family density as the measure of family structure as the independent variable
and each risk and protective factor as the dependent variable. First, family density was
entered as the only independent variable, and then SES and ethnicity, which were
included as covariates.

**Parental Monitoring.** Greater family density alone was not a significant correlate
of greater parental monitoring ($t(166) = 2.53, p = .113, \beta = -.123$). When SES and
ethnicity were included in the regression, the overall model also was not significant ($F(3,\
158) = 2.43, p = .068, R^2 = .044$).

**Adherence.** Greater family density was associated with poorer adherence ($t(240)\
= -3.17, p = .002, \beta = -.20$). A model including SES and ethnicity was significant ($F(3,\
229) = 15.86, p < .001, R^2 = .172$); however only SES ($t(229) = 2.62, p = .01, \beta = .17$)
and ethnicity ($t(229) = -4.43, p < .001, \beta = -.287$) were significantly related to adherence.
Family density was no longer a significant correlate of adherence ($t(229) = -1.67, p =\
.097, \beta = -.103$) after controlling for SES and ethnicity.

**Parental Stress.** Family density was not significantly related to parental stress
($t(237) = 1.71, p = .089, \beta = .11$). However, a model including family density, SES, and
ethnicity was significant ($F(3, 227) = 5.96, p = .001, R^2 = .073$). In this model, only SES
($t(227) = -2.53, p = .012, \beta = -.172$) and ethnicity ($t(227) = 2.02, p = .045, \beta = .138$) were
significant correlates of parental stress.
**Diabetes-related Conflict.** Greater family density was associated with greater diabetes-related conflict, when modeled in isolation, \((t(237) = 2.69, p = .008, \beta = .172)\). A second model with SES and ethnicity covariates was significant \((F(3, 227) = 10.15, p < .001, R^2 = .118)\); however only SES \((t(227) = -3.32, p = .001, \beta = -.221)\) and ethnicity \((t(227) = 2.17, p = .031, \beta = .145)\) were significant correlates of diabetes-related conflict.

In summary, the second hypothesis was partially supported for the family density construct of family structure. Higher family density was associated with lower adherence and more diabetes-related conflict, but was not associated with parental monitoring or parental stress. However, when the effects of socioeconomic status and ethnicity were included in the models, family density was no longer a significant correlate in any of the models.

**Single-parent status and diabetes care factors.** Multiple regressions were performed with single-parent status (as the measure of family structure) and each diabetes risk and protective factor separately. First, single-parent was tested in a separate regression model, and then the covariates of SES and ethnicity were included in a second model.

**Parental Monitoring.** Single-parent status alone was not a significant correlate of parental monitoring \((t(173) = 1.33, p = .185, \beta = .101)\). When SES and ethnicity were included in the regression, the overall model was similarly unrelated to parental monitoring \((F(3, 165) = 1.97, p = .120, R^2 = .035)\).

**Adherence.** Single-parent status was associated with poorer adherence \((t(252) = 2.20, p = .029, \beta = .137)\). A full model that included SES and ethnicity covariates also was significant \((F(3, 241) = 17.22, p < .001, R^2 = .177)\); however, in the second model,
only SES ($t(241) = 3.35, p = .001, \beta = .217$) and ethnicity ($t(241) = -4.87, p < .001, \beta = -.307$) were significantly related to adherence. Single-parent status was no longer a significant correlate of adherence ($t(241) = -.48, p = .629, \beta = -.031$) after controlling for SES and ethnicity.

**Parental Stress.** Single-parent status was significantly related to parental stress ($t(248) = -2.47, p = .014, \beta = -.155$). When SES and ethnicity were included as covariates in a full model, the overall model was significant ($F(3, 248) = 5.43, p = .001, R^2 = .064$) but only SES was a significant correlate of parent stress ($t(248) = -2.13, p = .034, \beta = -.148$). Neither single-parent status ($t(248) = -1.03, p = .304, \beta = -.071$) or ethnicity ($t(248) = 1.74, p = .083, \beta = .117$) were significant correlates of parental stress after accounting for the effects of SES.

**Diabetes-Related Conflict.** Single-parent status was associated with greater diabetes-related conflict in a separate regression model, ($t(249) = -2.45, p = .015, \beta = -.153$). A full model that included SES and ethnicity covariates was significant ($F(3, 239) = 9.44, p < .001, R^2 = .106$); however only SES ($t(239) = -3.65, p < .001, \beta = -.249$) and ethnicity ($t(239) = 2.04, p = .042, \beta = .135$) were significant correlates of diabetes-related conflict in the full model.

In summary, single-parent status was associated with three of four hypothesized diabetes factors. Single-parent status was associated with lower adherence, more parental stress, and more diabetes-related conflict, but not parental monitoring. Thus, the second hypothesis was again partially supported. However, when the effects of SES and ethnicity were included in the models, single-parent status, similar to family density, was no longer a significant correlate of glycemic control.
Mediators of Family Structure and Disease Outcomes: Hypothesis 3

Final multiple regressions were performed with family structure (represented by family density) as the independent variable, glycemic control as the dependent variable, and parental monitoring, adherence, parental stress and then diabetes-related conflict as mediating variables. Family density was used as the measure of family structure for these mediation analyses based on the outcome of the analyses for Hypothesis 1. Of the family structure variables, only family density was significantly correlated with glycemic control once covariates of SES and ethnicity were considered. This finding suggests family density is a more sensitive correlate of glycemic control than single-parent status.

The Baron and Kenny (1986) method for testing mediation was used for the following analyses. According to this process of testing mediation, a significant relation between the independent (family density) and dependent variable (HbA1c) and then the independent (family density) and mediating variable (risk or protective factors) is first needed. Then, in order to demonstrate mediation, both family density and the mediating variable are included in the regression. The effect size of the relation between family density and glycemic control needs to become significantly less strong compared to the original relation between family density and glycemic control.

**Parental Monitoring.** Family density was associated with HbA1c ($t(242) = 4.75, p < .001, \beta = .292$) but not with parental monitoring ($t(166) = -1.59, p = .113, \beta = -.123$). Since family density was not significantly related to parental monitoring, it was not considered further as a possible mediating variable.

**Adherence.** First, significant relations with HbA1c for family density ($t(242) = 4.75, p < .001, \beta = .292$) and adherence ($t(240) = -3.17, p = .002, \beta = -.20$) were
established. When both family density and adherence were included in the third regression model, the overall model was significant ($F(2, 239) = 19.69, p < .001, R^2 = .141$) and both adherence ($t(239) = -3.88, p < .001, \beta = -.237$) and family density ($t(239) = 4.06, p < .001, \beta = .248$) were significant correlates. Since the association between family density and HbA1c was still significant with adherence in the model, adherence did not fully mediate the relation. However, the relation between family density and HbA1c was smaller with adherence in the model than the original association. Using the Sobel test, the magnitude of the relation between family density and HbA1c was found to decrease significantly when adherence was included ($z = 2.44, p = .015$). Thus, adherence partially mediated the effect of family density on HbA1c (See Figure 1).

![Figure 1](image-url)

**Figure 1.** Adherence tested as a mediator of the relation between family density and glycemic control. Values in parentheses represent the standardized relation of family density to glycemic control after controlling for adherence.

**Parental Stress.** Family density was associated with HbA1c ($t(242) = 4.75, p < .001, \beta = .292$) but not parental stress ($t(237) = 1.71, p = .089, \beta = .11$). Since family
density was not significantly related to parental stress, it was not considered a possible mediating variable.

**Diabetes-related Conflict.** The last proposed analysis was the mediating effect of diabetes-related conflict on the relation between family density and HbA1c. First significant associations were demonstrated with HbA1c for family density ($t(242) = 4.75, p < .001, \beta = .292$) and then diabetes related conflict ($t(237) = 2.69, p = .008, \beta = .172$).

When both family density and diabetes-related conflict were included in the third regression model, the overall model was significant ($F(2, 236) = 23.17, p < .001, R^2 = .164$). The relation of diabetes-related conflict to HbA1c remained significant ($t(236) = 4.65, p < .001, \beta = .281$), and the relation of family density to HbA1c was also significant, but smaller than the original association ($t(236) = 4.10, p < .001, \beta = .247$). Using the Sobel test, the magnitude of the relation between family density and HbA1c decreased significantly when diabetes-related conflict was included in the model ($z = 2.34, p = .019$). Thus, diabetes-related conflict partially mediated the effect of family density on glycemic control (See Figure 2).
Results partially support the third hypothesis that parent monitoring, youth adherence, parental stress, and diabetes-related conflict will each mediate the relation between family structure, as measured by family density, and glycemic control. Poorer adherence and more diabetes-related conflict both partially mediated the relation between higher family density and poorer glycemic control, such that the relations between family structure, specifically family density, and HbA1c is partially explained by these two risk factors. Adherence and diabetes-related conflict were partial mediators of this relationship even after controlling for the effects of SES and ethnicity (See Appendix). Neither parental monitoring nor parental stress was examined as mediators because they failed to meet the initial criteria of mediation.

**Discussion**

Single-parent family structure is consistently identified in the literature as a key sociodemographic factor related to poorer diabetes care and glycemic control in adolescents with type 1 diabetes (Harris et al., 1999; Thompson et al., 2001; Thompson et al., 2001b). However, other family structure measures and their relation with risk and protective factors have remained unstudied. As expected, single-parent status was associated with poorer glycemic control among adolescents with T1D. Higher family density, another measure of family structure based on the ratio of children to adults in a household, also was related to poorer glycemic control. Although both single-parent families and higher density families were more likely to have adolescents in poorer
glycemic control than those from two-parent or lower density families, respectively, family density appears to be a more potent factor associated with youth glycemic control, based on average differences in HbA1c levels.

Despite long-standing use of marital status as a traditional measure of family structure, current results indicate that family density may be a better correlate of glycemic status, probably secondary to consideration of parental time and resources. Although higher family density is a risk factor for poorer family functioning in low birth-weight populations (Liaw & Brooks-Gun, 1994), family density has not previously been studied in families of adolescents with TID. Family density incorporates siblings into the family structure composite, which may be particularly important in families of an adolescent with TID. With multiple children in a household, a parent’s ability to be involved in diabetes disease care behaviors could be more limited. This study suggests that high family density may be a clinically relevant factor associated with youth glycemic control. Adolescents in high density families were at greater risk for poorer glycemic control as demonstrated by HbA1c values that were two percentage points poorer than adolescents from low density families. Based on results from the Diabetes Control and Complications Trial Research Group, if these differences persist, a glycemic difference of this magnitude will be related to a 57% greater risk of retinopathy over the course of nine years along with a higher rate of other disease complications (DCCT, 1993).

Family structure variables were predicted to relate to diabetes factors of parental monitoring, adherence, parental stress, and diabetes-related conflict. Single-parent status was associated with lower adherence, and greater parental stress and diabetes-related conflict. Better adherence to disease care behaviors is associated with improved
glycemic control (DCCT, 1993; Morris et al., 1997); however, few of the existing studies report family structure (Hood et al., 2009). Since adolescents who live in nontraditional or single-parent families are more likely to eat less frequent meals and snacks and test their blood glucose levels less often than children in two-parent families (Swift et al., 2006), a focus on enhanced adherence for this population is warranted. Additionally, single-parent status is related to parental stress. The relation between single-parent status and stress is notable since parental stress is associated with a number of risk factors including poorer parental mental health (Helgeson et al., 2012; Kovacs et al., 1985; Patton et al., 2011) as well as poorer adherence to disease care behaviors (Auslander et al., 1997). Finally, as predicted, single-parent status was associated with greater diabetes-related conflict. Since diabetes-related conflict is related to poorer adherence and glycemic control in youth with type 1 diabetes (Anderson, 2004; Hilliard et al., 2011; Hilliard et al., 2012; Hood et al., 2007), a further examination of this construct is justified. This finding also adds to the literature on general family functioning and family organization and suggests that diabetes-related conflict may be an important factor related to glycemic control.

Interestingly, single-parent status was not related to parental monitoring. No studies to date have examined level of parental monitoring or involvement in single-parent families of adolescents with T1D. Although the general developmental literature suggests that single-parent families show less parental involvement and supervision compared to two-parent families (McLanahan & Sandefur, 1994), this relation may not be true for families of adolescents with T1D where supervision of disease care behaviors
is essential. A parent in these family structures may be extra vigilant about supervision because they are the sole caregiver.

Higher family density was associated with poorer adherence and more diabetes-related conflict. This is a significant finding, given that the association between family density and adherence has not previously been examined. Results suggest that higher family density may be a factor related to poorer adherence in this population. High density families may be at a greater risk for diabetes-related conflict, perhaps because of stressors related to limited parental time or resources. Additionally, since high density families are likely to encompass blended families, there may be greater conflict that exists from multiple family members and different parenting styles.

High family density was not associated with parental monitoring or stress. The relation between these constructs and family density has not been previously been studied in a population of adolescents with T1D. Higher density families may have other family members that assist with family responsibilities thus allowing for parental monitoring of disease care behaviors. In addition, since high density families may have two parents to help with diabetes management and parental responsibilities, parents in high density families may not experience as much parental stress compared to single-parents. These differences in family structure present an area for future research.

In sum, single-parent status and higher family density each were related to poorer adherence and more diabetes-related conflict. Additionally, neither was a correlate of parental monitoring. However, these family structure variables differed in their relation to parental stress; only single-parent status was associated with parental stress. This difference may be due to a greater number of family members in higher density families,
as compared to single-parent families. A larger family may allow for more individuals to contribute to household tasks, reducing parental stress. Overall, when SES was included as a covariate, family structure variables were no longer significant which underscores the importance of this demographic factor. Although SES can explain much of the variance in glycemic control, higher family density, when considered without SES, was related to poorer adherence and more diabetes-related conflict. Since family density has never been studied in this population, the association of density to these diabetes factors is notable.

Although family density is an important family structure factor to consider, SES still remains a potent correlate of glycemic control for adolescents with type 1 diabetes. SES appears to account for most of the high risk related to single-parent status and high family density because of potentially limited financial and educational resources. Income is identified as a mediator of the relation between single-parent status and perceived vulnerability and stress in families of children with chronic illnesses (Mullins et al., 2011). Specifically for families of adolescents with TID, disease management can be expensive and time consuming, and within a high density family, resources and time may be more limited. Family structure factors of single-parent status and density are highly correlated with SES, perhaps because of the risk related to less time and resources that is captured by SES. In sum, SES is the most potent sociodemographic factor that relates to glycemic control.

Poorer adherence and greater diabetes-related conflict each were identified as links to the relation between high family density and poorer glycemic control and suggest an avenue to better glycemic control for these families. Better adherence and less
diabetes-related conflict relate to better glycemic control for adolescents with T1D who live in high density families. Additionally, family density was a potent enough correlate of glycemic status that it was possible to identify mediation mechanisms. While family structure variables, such as high family density, represent risk factors for poorer glycemic control, the identification of poorer adherence and more diabetes-related conflict as mediators provides information about the mechanisms associated with poorer glycemic control. Thus, poorer adherence and greater diabetes-related conflict emerge as important risk factors for poorer glycemic status and targets for intervention with adolescents in high density families.

Although potential risk factors for poorer glycemic control were identified, based on the cross-sectional nature of the data, no causal relations can be inferred. Future research should examine these constructs longitudinally and with the use of structural equation modeling in order to examine multiple mediating factors simultaneously. Additionally, families in this sample were recruited to be part of an 18-month treatment study with multiple assessments and phone follow-up visits, which may suggest that this sample is not representative of the general population of adolescents with T1D. Despite these limitations, this study contributes to research on adolescents with T1D which is imperative given the risk for deterioration in glycemic control in this age group. Additionally, this sample was relatively diverse in ethnicity and SES compared to similar research in this population. Likewise, some of the research looking at psychological factors related to glycemic control overlooks the effects of SES. The significance of SES as a factor associated with glycemic control makes this a critical variable to consider in treatment and research. The use of multi-source youth and parent report of parental
monitoring, adherence, and diabetes-related conflict provides data from multiple perspectives, which may lead to a more accurate portrayal of overall family functioning. Lastly, family density has never been studied in adolescents with type 1 diabetes.

The association of family density to poorer glycemic control and the existence of adherence and diabetes-related conflict as partial mediators emphasizes the importance of these constructs for adolescents with type 1 diabetes. Clinicians should be aware that certain family structures, such as high density, may present a risk for poorer adherence or greater diabetes-related conflict which in turn relates to an adolescent’s glycemic control. Additionally, adherence and diabetes-related conflict should continue to be a focus in research and a target for intervention in clinical work. Low SES presents as a risk factor for glycemic control as well as related disease care behaviors. Given the relation between SES and family structure, clinical and research attention should be focused on prevention of disease deterioration for adolescents presenting with these risk factors. Future research should continue to examine different types of family structures as well as the involvement of other family members, such as fathers or extended family members. Lastly, research should focus on interventions targeted for families of adolescents with type 1 diabetes in high risk family structures in order to prevent deterioration in disease care behaviors.
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Appendix

Figure 1. Adherence tested as a mediator of the relation between family density and glycemic control controlling for SES and ethnicity. Values in parentheses represent the standardized relation of family density to glycemic control after controlling for adherence, SES, and ethnicity.

![Diagram of Figure 1]

β = -.20
p = .002

β = .292
p < .001

β = .204
p = .001

β = .292
p < .001

β = .237
p < .001

β = .259
p < .001

β = .023
p = .724

SES

YOUTH ADHERENCE

GLYCEMIC CONTROL

DIABETES-RELATED FAMILY CONFLICT

FAMILY DENSITY

β = .172
p = .008

β = .292
p < .001

β = .195
p = .002

β = .281
p < .001

β = .232
p < .001

β = .055
p = .393

SES

ETHNICITY

Figure 2. Diabetes-related conflict tested as a mediator of the relation between family density and glycemic control controlling for SES and ethnicity. Values in parentheses represent the standardized relation of family density to glycemic control after controlling for diabetes-related conflict, SES, and ethnicity.

β = .195
p = .002

β = .281
p < .001

β = .232
p < .001

β = .055
p = .393

SES

ETHNICITY
Vita

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