



Published in final edited form as:

*Diabet Med.* 2009 September ; 26(9): 908–914. doi:10.1111/j.1464-5491.2009.02794.x.

## Original Article: Education and Psychological Aspects Diabetes-specific family conflict and psychological distress in paediatric Type 1 diabetes

L. B. Williams\*, L. M. B. Laffel†, and K. K. Hood\*‡

\*Center for Treatment Adherence, Division of Behavioral Medicine and Clinical Psychology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

†Pediatric, Adolescent, and Young Adult Section, Genetics and Epidemiology Section, Joslin Diabetes Center, Harvard Medical School, Boston, MA

‡Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, OH, USA

### Abstract

**Aims**—Diabetes-specific family conflict is associated with suboptimal adherence and glycaemic control. Little is known about the individual and family factors associated with diabetes-specific family conflict. The purpose of this study was to examine whether background factors (e.g. age, gender), diabetes variables (e.g. duration of diabetes, adherence, glycaemic control) and psychological distress (i.e. depression and anxiety) in parents and children and adolescents were associated with diabetes-specific family conflict.

**Methods**—Participants were 187 children and adolescents with Type 1 diabetes and their parents. Study measures assessed diabetes-specific family conflict, youth depression and parent depression and anxiety. Demographic and disease-specific data (adherence, glycaemic control) were also collected.

**Results**—Findings suggested a close link between psychological distress in parents and children and adolescents and reports of increased diabetes-specific family conflict. In the presence of suboptimal glycaemic control, children and adolescents and parents reported more family conflict. Adherence was not significantly associated with family conflict.

**Conclusions**—This study highlights the importance of considering the impact of individual psychological functioning on family conflict and also suggests a bidirectional relationship between conflict and glycaemic control.

### Keywords

anxiety; depression; distress; family management; Type 1 diabetes

---

Correspondence to: Korey K. Hood, PhD, Cincinnati Children's Hospital Medical Center, Division of Behavioral Medicine, 3333 Burnet Ave—MLC 7039, Cincinnati, OH 45229, USA. Korey.hood@cchmc.org.

Competing interests Nothing to declare.

## Introduction

Paediatric Type 1 diabetes is often characterized as a ‘family disease’ because family interactions, communication styles and supervisory roles of parents contribute to diabetes management [1–3]. More positive interactions, such as parental warmth and caring and parental responsibility taking for diabetes tasks, are associated with optimal diabetes management [4–6]. Positive family interactions are also associated with the prevention of deteriorating adherence following the diagnosis of Type 1 diabetes [7,8]. Conversely, when family interaction patterns include diabetes-specific conflict about treatment tasks, children and adolescents tend to evidence poorer adherence to their diabetes regimen and poorer health outcomes [9–11]. Indeed, effective management of paediatric Type 1 diabetes is directly linked to effective functioning of the family.

Within the context of family management of Type 1 diabetes, individual psychological factors, such as depression and anxiety, are also influential. For example, children and adolescents may feel burdened by diabetes management over time and depressive symptoms can develop, subsequently impacting on diabetes specific health behaviours and outcomes. There are compelling findings that children and adolescents with Type 1 diabetes experience depression at two to three times the rate of depression in the general population of children and adolescents [12–14] and depression has been linked to poorer diabetes-specific health outcomes [15,16].

Less has been documented about the prevalence of psychological distress in parents of children and adolescents with Type 1 diabetes, although several studies do indicate these individuals are at increased risk [14,17,18]. In addition, within paediatric diabetes populations, the experience of parental distress can negatively affect the psychological and health-related outcomes of children and adolescents [18,19]. It may be that distressed parents are less able to provide adequate support for children and consequently their diabetes management suffers. Indeed, parent depression, particularly in mothers, has long been implicated as influencing the outcomes of children who are otherwise medically healthy [20–22]. For example, children with depressed parents are more likely to become depressed as adults, which is likely because of the negative environment that is often present when parents are depressed [21]. There is also evidence to suggest that, in families of healthy children, maternal depression can persist for longer periods of time within the context of high family conflict [23], such that these negative states may serve to reinforce one another. Although parental depression has been linked to child outcomes in healthy populations, and to a lesser degree in paediatric diabetes populations, the role of other forms of psychological distress, such as parental anxiety, has not been as well examined. Furthermore, the linkage between parental distress and family-level distress (i.e. conflict) has not been examined fully in children and adolescents with diabetes.

For children and adolescents with Type 1 diabetes and their parents, diabetes-specific family conflict and psychological distress often occur in the context of one another and may serve inadvertently to reinforce each other. However, we are unaware of any examinations of the potential relationship between psychological distress and diabetes-specific family conflict. An attempt to integrate these areas may prove beneficial to better explain the nature of

diabetes-specific family conflict. Therefore, the first aim of this study is to document rates of psychological distress in paediatric patients and their parents and to examine the association with diabetes-specific family conflict. We hypothesized that higher rates of family conflict reported by children and adolescents and their parents would be associated with indicators of psychological distress in these individuals.

Further, the extant literature in paediatric Type 1 diabetes has examined diabetes-specific family conflict exclusively as a variable that promotes poor adherence and suboptimal glycaemic control [9,24]. In other words, family conflict is typically investigated as a predictor of health behaviours and outcomes. While there is certainly benefit in examining family conflict in this way, bidirectional relationships among these variables are likely. In fact, it is plausible that diabetes-specific family conflict may develop in response to learning the child or adolescent's current glycated haemoglobin (HbA<sub>1c</sub>) value (i.e. higher HbA<sub>1c</sub> leads to more family conflict). Considering this, the second aim of this study was to test a model of influential variables on diabetes-specific family conflict. Specifically, background factors (e.g. age, gender, ethnicity), diabetes-specific variables (e.g. diabetes duration, adherence, glycaemic control) and psychological factors (depression and anxiety) are hypothesized to be factors associated with the occurrence of diabetes-specific family conflict.

## Patients and methods

### Patients and procedures

Study participants included a convenience sample of 187 children and adolescents with Type 1 diabetes and their primary caregiver receiving care at a tertiary paediatric diabetes centre. All participants had Type 1 diabetes diagnosed according to American Diabetes Association (ADA) practice guidelines [25] and were between 10 and 17 years of age, inclusive. If more than one parent or guardian accompanied the child or adolescent to their clinic visit, data were collected on the 'primary caregiver' as designated by the family. Exclusion criteria included a major psychiatric or neurocognitive disorder that would limit the child or adolescent's ability to complete surveys (e.g. cognitive impairment), significant medical disease other than Type 1 diabetes or treated thyroid disorders or coeliac disease or present participation in a psychosocial intervention study. During the recruitment period, we approached 270 families and 196 (73%) agreed to participate. Of the families that declined participation, most did so because of lack of time or interest in study participation. We subsequently excluded nine families from data analysis because of substantial missing or incomplete data, giving a final study sample of 187 children and adolescents and their parent.

Prior to implementing the study procedures, the Institutional Committee on Human Studies approved the protocol. Research assistant obtained written informed consent from the participating parent and assent from the child or adolescent and then administered the questionnaires in the waiting room of the paediatric and adolescent clinic. Family demographic data were obtained during the clinic visit via self-report questionnaire.

## Measures

**Diabetes-specific family functioning**—Diabetes-specific family conflict was assessed using the revised Diabetes Family Conflict Scale (DFCS) [10]. This version of the original DFCS [26] contains updated language and additional items related to present diabetes management (e.g. new technologies). Each child or adolescent and their parent completed this scale independently. The level of diabetes specific family conflict is rated on a 3-point scale (1 = never argue, 2 = sometimes argue, 3 = always argue) across 19 diabetes management tasks such as insulin administration, checking blood glucose values and telling others about diabetes. A total score, ranging from 19 to 57, is calculated for each child or adolescent and parent, with higher scores indicating more conflict. Internal consistency for the DFCS in this study sample was high (youth coefficient  $\alpha = 0.87$ ; parent coefficient  $\alpha = 0.90$ ).

**Parental distress**—The 20-item Center for Epidemiologic Studies–Depression (CES–D) scale [27] was used to assess depressive symptoms in parents. This measure is widely used and large sample normative data and clinical cut-off scores ( $> 16$ ) are available for the CES–D. Parents respond to each item by endorsing 0 (not experiencing that symptom) to 3 (experiencing that symptom all the time) over the past week. There was a high level of internal consistency in this sample (coefficient  $\alpha = 0.91$ ). The State-Trait Anxiety Inventory (STAI) [28] was used to measure parental anxiety symptoms. The STAI has 40 items, with half representing present feelings (state scale) and the other half related to feelings in general (trait scale). On both scales, there was a high degree of internal consistency (state coefficient  $\alpha = 0.93$ ; trait coefficient  $\alpha = 0.92$ ). A clinical cut-off of one standard deviation above the mean of a normative sample was used to classify participants with increased symptoms of anxiety [28].

**Depression in children and adolescents**—Child and adolescent depressive symptoms were assessed with the Children’s Depression Inventory (CDI) [29], a self-report questionnaire consisting of 27 items rated from 0 (no symptom) to 2 (distinct symptom). CDI scores range from 0 to 54 with a clinical cut-off score of 13 or higher indicative of elevated depressive symptoms and suggestive of the need for further investigation [12, 14]. Responses on the CDI demonstrated a high degree of internal consistency (coefficient  $\alpha = 0.83$ ).

**Diabetes-specific variables**—Each child or adolescent provided blood for HbA<sub>1c</sub> measurement, by high-performance liquid chromatography (reference range 4.0–6.0%, Tosoh Bioscience, Tosoh 2.2; Foster City, CA, USA). HbA<sub>1c</sub> results were not available prior to participants completing study measures. Adherence to the diabetes regimen was obtained by calculating the daily frequency of blood glucose monitoring based on meter downloads, which was strongly correlated with HbA<sub>1c</sub> ( $r = -0.040$ ,  $P = 0.0001$ ). Duration of diabetes was obtained by reviewing the child or adolescent’s clinic visit note.

## Analysis plan

Three levels of analysis were conducted. First, descriptive statistics and frequencies for child and family characteristics, as well as the proportions of the sample that met clinical cut-offs

on measures of psychological distress, were calculated. Second, means, standard deviations and effect sizes for diabetes-specific family conflict were calculated in relation to cut-offs on measures of parental psychological distress. This was performed to illustrate the magnitude of differences in reported diabetes-specific family conflict when the clinical cut-off was met on distress measures. Third, hierarchical linear regression was conducted to determine factors associated with family conflict. We entered blocks of variables to illustrate the contribution of each block to family conflict. Block 1 contained background factors (child age, gender, race/ethnicity, parental level of education and family structure). Block 2 included diabetes-specific variables (diabetes duration, insulin delivery method, adherence and HbA<sub>1c</sub>). Block 3 contained psychological status of the child or adolescent (CDI score). Block 4 included the psychological status of the parent (CES-D and STAI scales scores). Block 5 included psychological distress by HbA<sub>1c</sub> interaction terms (CDI × HbA<sub>1c</sub>, CES-D × HbA<sub>1c</sub> and STAI scales × HbA<sub>1c</sub>). Regression coefficients, total  $R^2$  for the model,  $R^2$  change for each new block entry and change in the  $F$  value with each new block were calculated. Two regression models were run: one for family conflict reported by parents and one for family conflict reported by children and adolescents. All analyses were performed in sas 9.1 (SAS Institute, Cary, NC, USA).

## Results

### Participant characteristics

Table 1 provides complete details on the characteristics of the study participants. In brief, the 187 children and adolescents in this study had a mean age of  $14.4 \pm 2.4$  years, were predominantly white (87%) and the majority (80%) resided in two-caregiver families. These children and adolescents had a mean duration of Type 1 diabetes of  $6.5 \pm 3.9$  years and a mean HbA<sub>1c</sub> of  $9.0 \pm 1.5\%$ . Nearly 78% of children and adolescents monitored blood glucose levels four or more times daily and 47% used an insulin pump. Parents included 153 mothers (82%), 27 fathers (14%) and seven who identified themselves as ‘other caregiver’ (4%). Pearson correlations were used to examine the univariate associations among continuous demographic variables and disease variables in the study. Child age was negatively correlated with diabetes adherence ( $r = -0.37$ ,  $P = 0.0001$ ), but all other relationships were not significant.

### Psychosocial variables

The rates of diabetes-specific family conflict were nearly identical to a published sample [10] for both children and adolescents and parents. Parents had a group mean score of  $24.7 \pm 5.3$  and children and adolescents had a group mean score of  $24.9 \pm 6.9$ , indicating general agreement about the amount of conflict in their families around diabetes. Regarding depressive symptoms, of the 187 children and adolescents in this sample, 30 (16%) scored at or above the clinical cut-off on the CDI. With regard to parents, 23% ( $n = 42$ ) of parents reported significant symptoms of depression based on the CES-D clinical cut-off score. Fifteen per cent of parents ( $n = 28$ ) reported significant symptoms of state anxiety based on a cut-off score of 46 on the STAI-State (1 sd above the normative mean) and 18% ( $n = 33$ ) met criteria for significant symptoms on the STAI-Trait based on a cut-off score of 44 [24]. Overall, 24% of parents ( $n = 45$ ) endorsed symptoms of state and/or trait anxiety above the

cut-off. Table 2 displays the mean and effect size differences in diabetes-specific family conflict for those parents who scored at or above the clinical cut-off for distress (i.e. CES-D, STAI) and those who did not. On all three scales, distressed parents endorsed significantly higher rates of family conflict, with Cohen's *d* effect sizes ranging from 0.38 to 0.62.

### Factors associated with family conflict

Results from the first hierarchical linear regression on parent-reported diabetes-specific family conflict are presented in Table 3. As noted in the Analysis Plan, demographic, disease-specific, child distress and parent distress variables were entered into this regression. After the final block was entered, the overall model was significant,  $F_{(12,186)} = 8.49$ ,  $P = 0.0001$ ,  $R^2 = 0.37$ . The significant variables in this final model were glycaemic control ( $P = 0.0001$ ), state anxiety from the STAI ( $P = 0.05$ ) and trait anxiety from the STAI ( $P = 0.01$ ). More diabetes-specific family conflict was reported by parents when the child or adolescent's HbA<sub>1c</sub> was higher and when the parent experienced higher levels of anxiety. With the addition of Block 5 (psychological distress  $\times$  HbA<sub>1c</sub> interaction terms) the overall model remained significant,  $F_{(13,186)} = 9.72$ ,  $P = 0.0001$ ,  $R^2 = 0.42$ . All interaction terms entered in Block 5 were non-significant, with the exception of the STAI-Trait by HbA<sub>1c</sub> interaction (Block 5  $F$  change = 1.23,  $\beta = 0.12$ ,  $P = 0.0001$ ).

A second regression was conducted to predict family conflict reported by children and adolescents (results presented in Table 4). Blocks of variables were entered in the same manner. The psychological distress by HbA<sub>1c</sub> interaction terms entered in Block 5 were not significant. Thus the four-block model is presented and this final model was significant,  $F_{(12,185)} = 4.33$ ,  $P = 0.0001$ ,  $R^2 = 0.23$ . Only glycaemic control and the child or adolescent's report of depressive symptoms were related to diabetes-specific family conflict. More family conflict was reported by the children and adolescents when HbA<sub>1c</sub> values and report of depressive symptoms were higher.

## Discussion

The overarching goal of the current study was to examine the influence of background factors, diabetes factors and individual psychological factors on diabetes-specific family conflict. The results revealed two main conclusions: (i) diabetes-specific family conflict is associated with the occurrence of psychological distress in both parents and children and adolescents and (ii) the level of glycaemic control relates to the level of diabetes-specific family conflict. Consistent with our conceptual framework, these variables produced unique and significant contributions to diabetes-specific family conflict. To our knowledge, the approach taken in the current manuscript is unique in that it elucidates correlates of diabetes-specific family conflict by examining associations between individual psychological distress and disease factors and diabetes-specific family conflict.

With regard to the first conclusion, we found that increased psychological distress of parents and children and adolescents is associated with the occurrence of more diabetes-specific family conflict. It may be that distress contributes to conflicting interactions as a result of problems communicating around diabetes or because of competing emotional needs. Our findings are consistent with research in families with healthy children, which suggests that

parental depression and family conflict often occur concurrently and are transactional [23]. In this study, levels of parental anxiety were related to family conflict over and above parental depressive symptoms. This is further highlighted by the finding that the HbA<sub>1c</sub> by parental trait anxiety interaction term significantly predicted parental reports of family conflict. These results may be as a result of differences in the manifestation of anxiety vs. depression and suggest that parental anxiety interacts with glycaemic control to predict family conflict. For example, an anxious parent may engage in behaviours such as nagging and arguing about the diabetes regimen in efforts to reduce their anxiety, particularly when a child or adolescent's glycaemic control is poor. In this way, anxiety symptoms may be expressed in amore external manner compared with a parent experiencing depression, which often has a more internal focus. While depressed individuals may at times engage in more arguments as a result of the irritability associated with this condition, they may also be less reactive and less engaged in family interactions given their depressive symptomatology.

In the current sample, 23% of parents reported clinically elevated depressive symptoms and 24% reported elevated symptoms of anxiety. For children and adolescents, 16% reported elevated symptoms of depression. These rates of psychological distress are concerning and are much higher than those that are found in the general population. Given that our current findings suggest that psychological distress is associated with increased diabetes-specific family conflict, these distressed individuals probably face the added burden of negative family interactions. While the relationship between parent psychological distress and increased family conflict has been elucidated in the general child development literature, this is the first study to link these constructs within a paediatric diabetes population.

The second major conclusion from this study deals with glycaemic control. A body of prior research [9–11,24] examined the impact of diabetes-specific family conflict on glycaemic control and adherence. We found that HbA<sub>1c</sub> was associated with reports of diabetes-specific family conflict by both children and adolescents and their parents. This effect across both informants suggests that families have a constant eye on overall diabetes control and that an elevated HbA<sub>1c</sub> is associated with diabetes-specific family conflict from both youth and parent perspectives. Somewhat surprisingly, regimen adherence, measured via blood glucose meter downloads, was not associated with diabetes conflict in the overall model. This finding may indicate that families focus more on the HbA<sub>1c</sub> value than on behavioural components of adherence, such as blood glucose monitoring. For example, when glycaemic control is suboptimal, parents may initiate interactions that end up being negative given their frustrations with not achieving more optimal control. This seems to be particularly true when parents report symptoms of trait anxiety. Likewise, children and adolescents may be embarrassed or disappointed when they have a high HbA<sub>1c</sub> and may respond negatively to suggestions for improvements in their diabetes care. Because of the cross-sectional nature of this study, we cannot speak to the temporal relations of these variables. However, prior studies [9–11] suggest that increased diabetes-specific family conflict predicts suboptimal glycaemic control and the current study now provides preliminary evidence that it may be worthwhile to examine whether this relationship is bidirectional in nature in future longitudinal studies.

Considering these findings, there are important implications for researchers and clinicians. First, the nature of diabetes-specific family conflict appears to be made up of both general psychological constructs (individual depression) and diabetes-specific variables (glycaemic control). This lends support for continuing to examine ways to reduce diabetes-specific conflict through the integration of efforts to promote positive family communication related to diabetes management and sharing of responsibilities for specific diabetes tasks. These findings further suggest that it may be important to target the distress of both parents and children and adolescents in interventions. In fact, the negatively reinforcing nature of psychological distress and diabetes-specific family conflict may require phased treatment components to offer the best chance for reduction in both areas. For instance, it may be useful to address parental symptoms of distress such as anxiety and depression prior to working on diabetes-specific interactions in order to maximize the success of the family-based interventions. Additionally, within clinical settings it may be important to consider the negative impact that suboptimal glycaemic control may have on family interactions. When families of children and adolescents with diabetes are presented with negative feedback about glycaemic control, it may be useful to provide them with guidance or interventions to minimize the potentially destructive impact within the family as well as to promote more optimal management. For example, problem solving and strategies to improve communication may be useful to prevent or reduce family conflict in the face of suboptimal diabetes outcomes. As interventions are developed or modified to address these areas, it is desirable for randomized controlled trials to be conducted to examine the efficacy of such treatments.

The current study has several limitations worth noting. First, the data used in this study are cross-sectional. This makes it impossible to determine the direction of associations. However, it is likely that these associations are bidirectional and the purpose of this study was to describe more clearly the nature of diabetes-specific family conflict. Longitudinal studies are underway that assess all of these variables over time. Another limitation is the reliance on self-report data. It is possible that some of the associations elucidated are as a result of shared method variance (e.g. parental distress was related to parent report of diabetes-specific family conflict). Observational measures of diabetes-specific family conflict or structured interviews assessing depression and anxiety in participants would provide objective data to address this limitation. Additionally, this study utilized blood glucose monitoring as the sole indicator of adherence to the diabetes regimen. While blood glucose monitoring is strongly related to glycaemic control, this measure does not take into account adherence to other components of the diabetes regimen, such as insulin administration, diet or exercise. Data on adherence to other regimen components would provide a more thorough understanding of this construct. The generalizability of the current study is also limited given that the sample was predominately Caucasian, middle to upper class and given that a large portion of the participating children and adolescents used insulin pumps and demonstrated relatively good adherence to the diabetes regimen. Future examination of these constructs in more diverse samples is warranted.

In conclusion, for families of children and adolescents with Type 1 diabetes, this study highlights the importance of psychological functioning and diseases-specific variables as correlates of diabetes-specific family conflict. These findings suggest that interventions to



improve functioning in these families should focus on individual and disease characteristics in addition to family interactions.

## Acknowledgments

Funding for this study comes from NIH (K23 DK 073340; PI - Hood).

## Abbreviations

<b>CDI</b>	Children's Depression Inventory
<b>CES-D</b>	Center for Epidemiologic Studies–Depression
<b>DFCS</b>	Diabetes Family Conflict Scale
<b>HbA<sub>1c</sub></b>	glycated haemoglobin
<b>STAI</b>	State-Trait Anxiety Inventory

## References

1. Anderson B, Ho J, Brackett J, Finkelstein D, Laffel L. Parental involvement in diabetes management tasks: relationships to blood glucose monitoring adherence and metabolic control in young adolescents with insulin-dependent diabetes mellitus. *J Pediatr*. 1997; 130:257–265. [PubMed: 9042129]
2. Hanson CL, Henggeler SW, Harris MA, Burghen GA, Moore M. Family system variables and the health status of adolescents with insulin-dependent diabetes mellitus. *Health Psychol*. 1989; 8:239–253. [PubMed: 2737175]
3. La Greca AM, Auslander WF, Greco P, Spetter D, Fisher EB, Santiago JV. I get by with a little help from my family and friends: adolescents' support for diabetes care. *J Pediatr Psychol*. 1995; 20:449–476. [PubMed: 7666288]
4. Grey M, Boland EA, Yu C, Sullivan-Bolyai S, Tamborlane WV. Personal and family factors associated with quality of life in adolescents with diabetes. *Diabetes Care*. 1998; 21:909–914. [PubMed: 9614606]
5. Anderson BJ, Auslander WF, Jung KC, Miller JP, Santiago JV. Assessing family sharing of diabetes responsibilities. *J Pediatr Psychol*. 1990; 15:477–492. [PubMed: 2258796]
6. McKelvey J, Waller DA, North AJ, Marks JF, Schreiner B, Travis LB, et al. Reliability and validity of the Diabetes Family Behavior Scale (DFBS). *Diabetes Educ*. 1993; 19:125–132. [PubMed: 8458308]
7. Jacobson AM, Hauser ST, Lavori P, Willett JB, Cole CF, Wolfsdorf JI, et al. Family environment and glycemic control: a four-year prospective study of children and adolescents with insulin-dependent diabetes mellitus. *Psychosom Med*. 1994; 56:401–409. [PubMed: 7809339]
8. Hauser ST, Jacobson AM, Lavori P, Wolfsdorf JI, Herskowitz RD, Milley JE, et al. Adherence among children and adolescents with insulin-dependent diabetes mellitus over a four-year longitudinal follow-up: II. Immediate and long-term linkages with the family milieu. *J Pediatr Psychol*. 1990; 15:527–542. [PubMed: 2258799]
9. Anderson BJ, Vangsness L, Connell A, Butler D, Goebel-Fabbri A, Laffel LM. Family conflict, adherence, and glycaemic control in youth with short duration Type 1 diabetes. *Diabet Med*. 2002; 19:635–642. [PubMed: 12147143]
10. Hood KK, Butler DA, Anderson BJ, Laffel LM. Updated and revised Diabetes Family Conflict Scale. *Diabetes Care*. 2007; 30:1764–1769. [PubMed: 17372149]
11. Lewin AB, Heidgerken AD, Geffken GR, Williams LB, Storch EA, Gelfand KM, et al. The relation between family factors and metabolic control: the role of diabetes adherence. *J Pediatr Psychol*. 2006; 31:174–183. [PubMed: 16467317]

12. Grey M, Whittemore R, Tamborlane W. Depression in type 1 diabetes in children: natural history and correlates. *J Psychosom Res.* 2002; 53:907–911. [PubMed: 12377302]
13. Hood KK, Huestis S, Maher A, Butler D, Volkening L, Laffel LM. Depressive symptoms in children and adolescents with type 1 diabetes: association with diabetes-specific characteristics. *Diabetes Care.* 2006; 29:1389–1391. [PubMed: 16732028]
14. Kovacs M, Goldston D, Obrosky DS, Bonar LK. Psychiatric disorders in youths with IDDM: rates and risk factors. *Diabetes Care.* 1997; 20:36–44. [PubMed: 9028691]
15. Kokkonen J, Taanila A, Kokkonen E. Diabetes in adolescence: the effect of family and psychologic factors on metabolic control. *Nord J Psychiatry.* 1997; 51:165–172.
16. Stewart SM, Rao U, Emslie GJ, Klein D, White PC. Depressive symptoms predict hospitalization for adolescents with type 1 diabetes mellitus. *Pediatrics.* 2005; 115:1315–1319. [PubMed: 15867041]
17. Whittemore R, Urban AD, Tamborlane WV, Grey M. Quality of life in school-aged children with type 1 diabetes on intensive treatment and their parents. *Diabetes Educ.* 2003; 29:847–854. [PubMed: 14603873]
18. Jaser SS, Whittemore R, Ambrosino JM, Lindemann E, Grey M. Mediators of depressive symptoms in children with type 1 diabetes and their mothers. *J Pediatr Psychol.* 2008; 33:509–519. [PubMed: 17991690]
19. Hassan K, Loar R, Anderson BJ, Heptulla RA. The role of socioeconomic status, depression, quality of life, and glycemic control in type 1 diabetes mellitus. *J Pediatr.* 2006; 149:526–531. [PubMed: 17011326]
20. Cicchetti D, Rogosch FA, Toth SL. Maternal depressive disorder and contextual risk: contributions to the development of attachment insecurity and behavior problems in toddlerhood. *Dev Psychopathol.* 1998; 10:283–300. [PubMed: 9635225]
21. Orvaschel H, Weissman MM, Kidd KK. Children and depression—the children of depressed parents; the childhood of depressed patients; depression in children. *J Affect Disord.* 1980; 2:1–16. [PubMed: 6448876]
22. Radke-Yarrow M, Zahn-Waxler C, Richardson DT, Susman A, Martinez P. Caring behavior in children of clinically depressed and well mothers. *Child Dev.* 1994; 65:1405–1414. [PubMed: 7982358]
23. Horwitz SM, Briggs-Gowan MJ, Storfer-Isser A, Carter AS. Prevalence, correlates, and persistence of maternal depression. *J Womens Health.* 2007; 16:678–691.
24. Wysocki T, Harris MA, Buckloh LM, Mertlich D, Lochrie AS, Taylor A, et al. Effects of behavioral family systems therapy for diabetes on adolescents' family relationships, treatment adherence, and metabolic control. *J Pediatr Psychol.* 2006; 31:928–938. [PubMed: 16401678]
25. Silverstein JH, Klingensmith G, Copeland K, Plotnick L, Kaufman F, Laffel L, et al. Care of children and adolescents with type 1 diabetes: a statement of the American Diabetes Association. *Diabetes Care.* 2005; 28:186–212. [PubMed: 15616254]
26. Rubin RR, Young-Hyman D, Peyrot M. Parent–child responsibility and conflict in diabetes care. *Diabetes.* 1989; 38:28.
27. Radloff LS. The CES–D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977; 1:385–401.
28. Spielberger, CD. *State-Trait Anxiety Inventory for Adults.* Redwood City, CA: Mind Garden; 1983.
29. Kovacs, M. *The Children's Depression Inventory (CDI): Technical Manual.* North Tonawanda, NY: Multi-Health Systems; 2003.

**Table 1**

## Participant characteristics

Characteristic	Youth ( <i>n</i> = 187)	Range
Age (years)	14.4 ± 2.4	10.0–17.9
Sex (% female)	45%	
Ethnicity <i>n</i> (%)		
White, not of Hispanic origin	162 (87%)	
Black/African-American	11 (6%)	
Hispanic/Latino	9 (5%)	
Asian/Pacific Islander	5 (< 3%)	
Education level of primary caregiver [(%) with at least college degree]	58%	
Insurance status		
Private insurance	158 (85%)	
Public insurance	29 (15%)	
Family composition (% with two caregivers in home)	80%	
T1D duration (years)	6.5 ± 3.9	0.2–15.2
HbA <sub>1c</sub> (%)	9.0 ± 1.5	6.2–16.1
Blood glucose monitoring (number per day)	4.8 ± 1.9	0–9+
3 or less times per day (%)	23%	
4 times per day (%)	29%	
5 or more times per day (%)	48%	
Method of insulin delivery		
CSII	87 (47%)	
3 daily injections	60 (32%)	
4 daily injections	40 (21%)	

Scores shown as mean ± sd.

CSII, continuous subcutaneous insulin infusion; HbA<sub>1c</sub>, glycated haemoglobin; T1D, Type 1 diabetes.

**Table 2**

Diabetes-specific conflict based on parental distress groups

Construct	Group by cut-off score	Mean parent DFCS (overall mean = 25 ± 6.0)	Effect size Cohen's <i>d</i>
Parent CES–D score	Above cut-off CES–D 16 ( <i>n</i> = 42)	28 ± 8.0 *	0.62
	Below cut-off CES–D < 16 ( <i>n</i> = 145)	24 ± 5.0 *	
Parent STAI—state score	Above cut-off STAI-state 46 ( <i>n</i> = 31)	29 ± 7.8 *	0.38
	Below cut-off STAI-state < 46 ( <i>n</i> = 156)	24 ± 5.4 *	
Parent STAI—trait score	Above cut-off STAI-trait 44 ( <i>n</i> = 36)	29 ± 7.4 *	0.39
	Below cut-off STAI-trait < 44 ( <i>n</i> = 151)	24 ± 5.3 *	

\* *P* < 0.0001.

CES–D, Center for Epidemiologic Studies–Depression; DFCS, Diabetes Family Conflict Scale; STAI, State-Trait Anxiety Inventory.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 3

Hierarchical linear regression results—parent report of family conflict

Variable	R <sup>2</sup>	R <sup>2</sup>	F	β	CI
Block 1—Socio-demographic characteristics	0.08	0.08*	3.28 <sup>†</sup>		
Child age (years)				0.32	-0.04 to 0.67
Child gender (male)				0.38	-1.12 to 1.88
Child race/ethnicity (white)				-1.65	-3.90 to 0.59
Family status (two caregivers in home)				1.27	-0.64 to 3.18
Parent education (< college degree)				-0.27	-1.87 to 1.33
Block 2—Diabetes variables	0.18	0.10*	4.41 <sup>†</sup>		
Type 1 diabetes duration (years)				-0.01	-0.21 to 0.19
Method of insulin delivery (pump)				0.70	-0.99 to 2.39
Adherence (BG monitoring frequency)				0.29	-0.19 to 0.77
Glycaemic control (HbA <sub>1c</sub> )				1.49 <sup>‡</sup>	0.93 to 2.05
Block 3—Youth depressive symptoms	0.19	0.01	4.17		
CDI score				0.02	-0.13 to 0.16
Block 4—Parent depression/anxiety	0.37	0.18*	7.88 <sup>†</sup>		
CES-D score				-0.01	-0.14 to 0.12
STAI state score				0.10	-0.00 to 0.20
STAI trait score				0.20 <sup>‡</sup>	0.05 to 0.35

Race/ethnicity: 0, white, not of Hispanic origin; 1, non-white.

Education: 0, less than a college degree; 1, at least a college degree.

Method of insulin delivery: 0, injections; 1, pump.

BG, blood glucose; CDI, Children's Depression Inventory; CES-D, Center for Epidemiologic Studies-Depression Scale; CI, confidence interval; HbA<sub>1c</sub>, glycated haemoglobin; STAI, State Trait Anxiety Inventory.

Regression coefficients presented in this table were derived from final model (i.e. all of the variables).

\* R<sup>2</sup> significant at  $P < 0.05$ .<sup>†</sup> Significant  $F$ -value at  $P < 0.0001$ .<sup>‡</sup> Significant regression coefficients at  $P < 0.01$ .

Table 4

Hierarchical linear regression results—youth report of family conflict

Variable	R <sup>2</sup>	R <sup>2</sup>	F	β	95% CI
Block 1—Socio-demographic characteristics	0.03	0.03	1.13		
Child age (years)				0.06	-0.29 to 0.40
Child gender (male)				0.20	-1.26 to 1.67
Child race/ethnicity (white)				-1.05	-3.26 to 1.16
Family status (two caregivers in home)				0.37	-1.50 to 2.24
Parent education (< college degree)				-0.82	-2.38 to 0.73
Block 2—Diabetes variables	0.07	0.04	1.47		
Type 1 diabetes duration (years)				-0.12	-0.31 to 0.08
Method of insulin delivery (pump)				-0.18	-1.83 to 1.46
Adherence (BG monitoring frequency)				-0.04	-0.43 to 0.51
Glycaemic control (HbA <sub>1c</sub> )				0.79 <sup>‡</sup>	0.25 to 1.34
Block 3—Youth depressive symptoms	0.22	0.15 <sup>*</sup>	4.80 <sup>‡</sup>		
CDI score				0.38 <sup>‡</sup>	0.24 to 0.52
Block 4—Parent depression/anxiety	0.23	0.01	3.98 <sup>‡</sup>		
CES-D score				-0.04	-0.17 to 0.09
STAI state score				0.01	-0.09 to 0.11
STAI trait score				0.09	-0.05 to 0.24

Race/ethnicity: 0, white, not of Hispanic origin; 1, non-white.

Education: 0, less than a college degree; 1, at least a college degree.

Method of insulin delivery: 0, injections; 1, pump.

BG, blood glucose; CDI, Children's Depression Inventory; CES-D, Center for Epidemiologic Studies-Depression Scale; CI, confidence interval; HbA<sub>1c</sub>, glycated haemoglobin; STAI, State Trait Anxiety Inventory.

Regression coefficients presented in this table were derived from final model (i.e. all of the variables).

<sup>\*</sup> R<sup>2</sup> significant at  $P < 0.05$ .<sup>‡</sup> Significant  $F$ -value at  $P < 0.05$ .<sup>‡</sup> Significant regression coefficients at  $P < 0.05$ .