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Identification and Prediction of Group-Based Glycemic Control Trajectories during the Transition to Adolescence

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Abstract

Objective—To identify trajectories of glycemic control over a period of three years in a pediatric sample of youth diagnosed with type 1 diabetes transitioning to adolescence. A second aim was to examine a set of modifiable individual and family-level baseline predictors of glycemic control group membership.

Methods—This multisite, prospective study included 239 children and adolescents (ages 9–11 years at baseline) diagnosed with type 1 diabetes and their caregivers. Glycemic control was based on hemoglobin A1c (HbA1c) collected at six month intervals over a period of three years. Predictors of glycemic control membership included baseline global executive functioning, diabetes self-management, diabetes-specific family conflict, blood glucose monitoring frequency, and relevant individual and family level covariates.

Results—Group-based trajectory analyses were used to describe patterns of glycemic control from baseline to 36 months and three trajectories were identified: low risk (42.9%), elevated risk (44.6%), and high risk (12.1%) subgroups. Baseline maternal-reported family conflict, blood glucose monitoring frequency, and gender were significant predictors of glycemic control group membership. Higher levels of baseline family conflict, lower frequency of blood glucose monitoring, and female gender were associated with elevated and high risk group membership.

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Conclusions—These findings underscore the importance of examining trajectories of HbA1c across time. These results suggest that problematic trajectories of glycemic control are evident during the transition to adolescence. Furthermore, there are modifiable individual and family-level characteristics that predict group membership and hence could be targeted in interventions to ensure adequate glycemic control is maintained over time and that risks for diabetes-related complications are reduced.

Keywords

Glycemic control; trajectories; individual factors; family factors

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The primary goal of clinical management in pediatric type 1 diabetes is to maintain optimal glycemic control over time (American Diabetes Association, 2010). Despite American Diabetes Association (ADA) glycemic control recommendations for pediatrics (e.g., below 8% for children ages 6–12 years; below 7.5% for adolescents ages 13–19 years) (Silverstein et al., 2005), glycemic control often begins to deteriorate during early adolescence (Moran, 2002) and significantly declines throughout adolescence and young adulthood (Helgeson et al., 2010). Large diabetes centers that have implemented intensive, comprehensive insulin therapy regimens have found that it is very challenging for adolescents to achieve optimal glycemic control (Danne et al., 2001).

The established relationship between less adequate glucose control in adolescence and higher rates of a wide range of diabetes-related complications in adulthood (Danne et al., 2001; DCCT Research Group, 1994; Krolewski, Laffel, Krolewski, Quinn, & Warram, 1995; Nathan et al., 2005; Silverstein et al., 2005) heightens the importance of achieving optimal glycemic control. However, not everyone is equally vulnerable, and there is considerable intra-group variability (Keough, Sullivan-Bolyai, Crawford, Schilling, & Dixon, 2011; Rohan et al., 2011; Schneider et al., 2007). Relatively little is known about the specific distribution of trajectories of glycemic control among early adolescents with type 1 diabetes and the factors that predict them. Most studies have focused on description and prediction of average levels of glycemic control, often over limited periods (e.g., one year) (Butler et al., 2008; Drotar et al., 2013; Helgeson & Palladino, 2012; Ingerski, Anderson, Dolan, & Hood, 2010).

Three studies have described subgroups or trajectories of glycemic control in adolescents with type 1 diabetes and relevant predictors. Luyckx and Seiffge-Krenke (2009) studied 72 adolescents across eight time points (e.g., from ages 14–17 and then again at ages 21–25 years). Based on latent class growth analyses, 3 subgroups were identified that described glycemic control patterns from ages 14 – 25 years: 1) optimal glycemic control (N = 10; *Mean HbA1c* = 5.9 – 7.4); 2) moderate glycemic control (N = 51; M = 7.4 – 8.5); and, 3) deteriorating glycemic control (N = 11; M = 6.7 – 9.7). The optimal glycemic control subgroup reported a more cohesive family climate (i.e., increased family control and organization) and higher positive self-concept compared to the deteriorating subgroup (Luyckx & Seiffge-Krenke, 2009).

Helgeson and colleagues (2010) studied a sample of 132 adolescents (M age at baseline = 12 years) across five years. Two glycemic control subgroups were identified: 1) stable, good

glycemic control (*N* = 83; *M HbA1c* = 7.8 – 8.0); and, 2) a deteriorating glycemic control subgroup (*N* = 46; *M HbA1c* = 9.0 – 10.5). Individuals in the deteriorating glycemic control subgroup were characterized by higher peer conflict, more negative diabetes emotions, fewer blood glucose tests, and more missed clinic appointments (Helgeson et al., 2010).

King and colleagues (2012) studied a sample of 252 adolescents across two years (*M age* = 12.5 years) and also identified two glycemic control trajectories using latent curve analysis: 1) moderate glycemic control group (*N* = 231), whose average glycemic control was 8.2% at baseline and increased at a rate of 0.07% per year; and, 2) poor glycemic control group (*N* = 21), whose average glycemic control of 12.1% at baseline increased rapidly at a rate of 0.32% per year. Compared to the optimal glycemic control subgroup, the poor glycemic control subgroup reported less paternal involvement and monitoring of daily diabetes-related management, as well as, lower functional autonomy and self-control. In addition, those in the poor glycemic control group reported sacrificing their own personal goals to gain the approval of their friends more often than the optimal glycemic control group (King et al., 2012).

Hilliard and colleagues (2013) studied a sample of 150 adolescent and parent dyads across 18 to 24 months and identified three stable glycemic control trajectories using latent group-based trajectory modeling: 1) “meeting treatment targets” (40%), whose glycemic control across time averaged about 7.4%; 2) “normatively similar” (40%), whose glycemic control across time averaged about 9.2%; and, 3) “high risk” (20%), whose glycemic control across time averaged about 11.2%. Adolescents who demonstrated poorer diabetes management and control were of older age, had longer diabetes duration, identified with an ethnic minority status, had an unmarried caregiver, used injection treatment regimens, reported greater depressive symptoms, more negative affect about blood glucose monitoring, and higher diabetes-specific family conflict (Hilliard, Wu, Rausch, Dolan, & Hood, 2013).

Each of these previous studies had limitations, many of which are addressed in the current study design. Luyckx and Seiffge-Krenke’s (2009), Helegon et al.’s (2010), and Hilliard et al.’s findings are limited by relatively small sample sizes (*N*s = 72 132, and 150 respectively). Ideally, group-based trajectory or latent class growth curve analyses require larger sample sizes for reliable results (Nagin, 2005). Furthermore, Luyckx and Seiffge-Krenke’s (2009) research did not study trajectories of glycemic control across the entire age span of the sample: a gap of four years was not accounted for in their analyses. Helgeson et al. (2010) used a self-report measure of pubertal status and studied a very homogenous sample in race and ethnicity (93% of patients were white). Similarly, King and colleagues (2012) studied a sample of mostly Caucasian adolescents (92.8%) and did not examine covariates such as gender, diabetes duration, insulin regimen, or tanner stage, which are known to influence glycemic control (Amiel, 1986; Helgeson et al., 2010; Moran, 2002)

One salient contribution of the present study was the prospective measurement of trajectories of glycemic control across three years (seven time points collected at six month intervals) starting at the transitional period of late childhood and continuing through early adolescence using group-based trajectory modeling. Glycemic control was selected as the primary outcome because it is a well-recognized biomarker of health status that predicts risk

for future diabetes-related complications and is a primary target of clinical management (DCCT Research Group, 1994; Krolewski et al., 1995). We recruited a relatively homogenous sample of youth with type 1 diabetes at the cusp of adolescence across three sites, the majority of whom were pre-pubertal, in order to identify potential risk factors for problematic glycemic control as these youth transitioned through adolescence. The age group was selected for study because: 1) it is well documented that glycemic control deteriorates after the onset of adolescence (Helgeson et al., 2010; Moran, 2002), and 2) preventive interventions delivered at this age may reduce exposure to chronic poor glycemic control through adolescence and into young adulthood (Fogel & Weissberg-Benchell, 2010). It was hypothesized that three subgroups representing different clinically relevant trajectories of glycemic control would be identified: 1) low risk group with optimal glycemic control, 2) elevated risk group with problematic patterns of glycemic control, and 3) a high risk group with consistently very high levels of glycemic control over time.

A second relevant scientific-value added contribution of our study involved the prediction of membership in subgroups of clinically-relevant trajectories of glycemic control. Our selection of predictor variables was based on the following factors: 1) inclusion of individual psychological and family variables in a single predictive model; and 2) focus on clinically-relevant, potentially modifiable variables that could be targets of intervention in the event they were shown to be significant. We tested the predictive value of these variables in a single model that also controlled for covariates (e.g., pubertal status, age, gender, etc.) that reflected non-modifiable influences that would also be expected to affect glycemic control based on previous research.

Executive functioning refers to meta-cognition and behavioral regulation and has been shown to predict glycemic control (Bagner, Williams, Geffken, Silverstein, & Storch, 2007; McNally, Rohan, Shroff Pendley, Delamater, & Drotar, 2010; Miller et al., 2013). Diabetes self-management refers to behaviors across multiple domains (e.g., diet, physical activity, insulin management, glucose monitoring) that a patient and/or family engage in to promote optimal glycemic control (Harris et al., 2000; Keough et al., 2011; Kichler, Kaugars, Ellis, & Alemzadeh, 2010; Rohan et al., 2011; Schneider et al., 2007). Blood glucose monitoring frequency, which measures how well a patient/family follows the treatment regimen for blood glucose testing has been used to describe adherence, and is known to be associated with glycemic control (DCCT Research Group, 1994; Harris et al., 2000; Helgeson, Honcharuk, Becker, Escobar, & Siminerio, 2011; Keough et al., 2011; Kichler et al., 2010; Levine et al., 2001). Investigators have used data from BGM frequency to validate a measure of combined risk for hyperglycemia and hypoglycemia episodes referred to as the average daily risk range (ADRR) (Kovatchev, Otto, Cox, Gonder-Frederick, & Clarke, 2006; Patton, Midyett, Dolan, & Powers, 2012) . However, in contrast to the frequency of BGM, the ADRR is not used in routine follow-up of pediatric diabetes. Finally, family factors, especially diabetes-specific family conflict has been shown to negatively influence self-management, treatment adherence to blood glucose monitoring, and glycemic control (Helgeson & Palladino, 2012; Hilliard et al., 2012; Hood, 2007; Ingerski et al., 2010; Levine et al., 2001). We hypothesized that those in the optimal glycemic control group would report better executive functioning, less family conflict, better self-management skills, and higher

frequency of blood glucose monitoring at baseline compared to the groups with more problematic glycemic control over time.

Methods

Participants and Procedures: Baseline

Participants were youth with type 1 diabetes and their maternal caregivers who were followed at pediatric diabetes clinics at three university affiliated medical centers in the United States. Each site's Institutional Review Board approved the study. Data were collected as part of an ongoing, three-year longitudinal study. Reports of longitudinal data from this study have been previously described in Drotar et al. (2013), Rausch et al. (2012), and Rohan et al. (2013). This is the first report from this study that has focused on identification of trajectories of glycemic control across time and the prediction of glycemic control group membership over three years.

Caregivers and children were recruited during a routine outpatient clinic visit. Potentially eligible participants were identified by clinic staff and then approached by research staff who explained the study procedures and verified eligibility. Inclusion criteria included duration of type 1 diabetes for at least one year, ages 9–11 at the time of recruitment, English speaking, no known plans to move out of the area during the duration of the study, and absence of secondary causes of a type 1 diabetes diagnosis (e.g., cystic fibrosis). Exclusionary criteria included current involvement in foster care, presence of severe psychiatric disorders or comorbid chronic conditions that required burdensome, ongoing treatment regimens, or diagnosis of intellectual and/or developmental disabilities.

Of the 361 eligible participants who were approached, 240 (66.5%) consented and participated. Reasons for not participating included being too busy (n = 54), no transportation (n = 3), and other (n = 64; not interested in research, did not return recruitment phone calls, did not attend clinic regularly, etc.). Signed informed consent was obtained from a parent or legal guardian, written assent from children 11 years old, and verbal assent from children less than 11 years according to the guidelines established by each sites Institutional Review Boards. After enrollment, one child was diagnosed with monogenic diabetes of the young (MODY) (Gardner & Tai, 2012), no longer treated with insulin, and hence removed from the study and subsequent data analysis.

The three-year follow-up yielded a sample of 222 youth (ages 11.9 – 15.6 years) with type 1 diabetes and their maternal caregivers. Overall attrition from baseline to three years was 4.2% (n = 10). Reasons for discontinuing participation included: child and/or family no longer interested in research or too busy to participate (n = 3), family moving out of the area (n = 1), patient changed endocrinologists and the doctor was not affiliated with the hospital (n = 1), family was too overwhelmed to participate in research (n = 1), and families would not schedule research visit or were lost to follow-up and were dropped from the study (n = 4). In addition to attrition, missing data also included non-completion of visits at one year (n = 13), two years (n = 14), and three years (n = 7). There were no significant differences (p > 0.05) between those who participated in the one, two, and three year follow-ups and those who did not complete the one, two, and/or three year study visit with respect to baseline

disease duration, age, ethnicity and race, income, household composition (one versus two-parent), child's gender, insulin delivery method from baseline to 36 months, or HbA1c obtained at six month intervals.

Sample Characteristics: Baseline to Three Years

The demographic and medical characteristics of our sample from baseline through three year follow-up are shown in Table 1. At three years, the sample (mean age of 13.62 years) had a comparable percentage of males (45.5%) and females (54.5%) and included a majority of non-Hispanic Caucasian youth (76.6%), but higher than typical percentages of Hispanic Caucasian youth (13.1%) in studies of adolescents with type 1 diabetes (Helgeson et al., 2011; Ingerski et al., 2010). The majority of the sample (68.9%) received insulin via subcutaneous insulin infusion (i.e., insulin pump or pod).

Measures: Primary Outcome

Glycemic Control: Hemoglobin HbA1c—Blood samples were obtained at six month intervals from baseline to three years post baseline by a finger stick during the study visit. Samples from each study site were analyzed using the TOSOH-G7 method (reference range 4.0 – 6.0%) by one central laboratory to ensure standardization of results across sites.

Measures: Predictors of Trajectories of Glycemic Control

Global Executive Functioning—Participants' executive functioning was measured using the Behavior Rating Inventory of Executive Functioning (BRIEF), an 86-item parent report measure (Gioia, Isquith, Guy, & Kenworthy, 2000). Higher scores mean less adequate executive functioning. The composite raw score for the Global Executive Composite (GEC) includes the Behavioral Regulation Index (e.g. the child's ability to shift cognitive set and moderate emotions and behaviors via emotional control) and Metacognition Index (e.g. the child's ability to monitor, initiate, plan, organize, and sustain future oriented problem solving and working memory). A T score of 65 or higher on any of the scales of the BRIEF is considered an elevated score (e.g., clinically significant). Reliability of the BRIEF has been established (α =.80 – .98) for both clinical and normative samples (parent and teacher forms), and validity has been documented with other measures of behavioral and attentional functioning (Gioia et al., 2000). Standardized scores were used in the analyses. In our sample, the maternal reported (N = 236) Global Executive Composite internal consistency was assessed using Cronbach's α: .97 at baseline.

Family Conflict—The Diabetes Family Conflict Scale – Revised (DFCS-R) (Hood, 2007) is a 19 item self-report measure that was completed by youth and parents and reflects the level of conflict within the family as a whole regarding specific tasks such as taking more or less insulin depending on results, remembering to check blood sugars. Total possible scores on the measure range from 19 to 57 with higher scores representing higher levels of diabetes-specific conflict in the family. This measure has demonstrated good internal consistency for youth (α = .85) and for parents (α = .81) (Hood, 2007). In the present sample, internal consistency (α) at baseline was .87 for the youth DFCS-R and .85 for the maternal caregiver DFCS-R.

Blood Glucose Monitoring Frequency—Blood glucose monitoring frequency (BGM) results were obtained from the child's blood glucose meter(s) for the previous two weeks starting with the day prior to the assessment visit. If one or more of the meters (e.g., a school meter) were not available at the time of the study visit, the same information was obtained from the child's logbook.

Diabetes Self-Management—The Diabetes Self-Management Profile (DSMP) is a 25-item structured interview administered independently to children and their caregivers to assess diabetes-related management behaviors during the previous 3 months (Harris et al., 2000). Open-ended questions addressed the following domains: exercise, hypoglycemia management, diet, blood glucose monitoring, and insulin administration. A total self-management score was calculated by summing all items, and subscale scores were obtained by summing items for each appropriate scale. Higher scores reflected better self-management behaviors. The DSMP total score has demonstrated good internal consistency (r = .76), moderate agreement between parent and youth reporters (r = .61), and strong inter-rater agreement (r = .94) (Harris et al., 2000). This measure also has demonstrated good predictive validity between parent and child reported self-management behaviors and glycemic control (Harris et al., 2000). Internal consistency for the present sample at baseline was .66 for the maternal DSMP and .60 for the youth DSMP.

Measures: Covariates

We chose to include a comprehensive set of baseline covariates in our model based on theory and previous research in pediatric type 1 diabetes. Pubertal status (as measured by Tanner Stage based on provider exam) was included based on research that has consistently underscored the impact of puberty in triggering insulin resistance (Amiel, 1986; Moran, 2002), which would be expected to reduce the level of glycemic control. We also included the following demographic and medical characteristics based on their potential influence on glycemic control and hence trajectory group membership (Helgeson et al., 2010): site; child gender; child ethnicity and race (Non-Hispanic, Caucasian; Non-Hispanic, Other; or Hispanic); maternal education (i.e., did not finish high school; obtained high school diploma or equivalent; obtained some college or college degree); household composition (i.e., one or two caregiver involvement); insulin delivery method (i.e., pump/pod versus injections); type 1 diabetes duration in years; and, child age.

Approach to Statistical Analysis

Group-based trajectory modeling was used to model patterns of change over time and identify specific subgroups (Nagin, 2005). Methods such as growth curve modeling, which has been used in previous studies of adolescents with type 1 diabetes (Miller et al., 2013) describes change over time at the individual level as well as for the population as a whole but does not provide information regarding the heterogeneity of the population being modeled or distinct, specific subgroups that follow similar patterns over time (Nagin, 2005).

We used the SAS TRAJ Procedure to identify trajectories of subgroups of glycemic control (HbA1c) based on latent group-based trajectory modeling (LGTM) (Nagin, 2005). When using LGTM, each person is assumed to belong to a single subgroup for the duration of the

observational period. We used the normal distribution option in the TRAJ procedure to model HbA1c over time. We examined solutions with 2,3,4,5 and 6 subgroups and allowed for quadratic trajectories within each subgroup; however, models with linear trajectories were ultimately chosen because quadratic terms were unnecessary. Our statistical criteria for selecting the best model was the Bayesian Information Criterion (BIC) and a subgroup proportion of at least .10, the latter to ensure the group size was large enough to be of practical utility. Ultimately, this left the two and three subgroup solutions as possible candidates, and of these two, the Bayesian Information Criterion (BIC) was substantially better for the three group solution (−2249.2 for the three group solution vs. −2379.5 for the two group solution). The final three subgroup solution was chosen based on clinical considerations, in addition to being the model that had the best statistical fit based on the BIC. For example, the two group model combined the low risk and moderate risk trajectories into a single trajectory. However, previous research has provided evidence that there is an association between moderate levels of elevated or moderate risk levels of glycemic control and risk for future health complications (DCCT Research Group, 1994; Levine et al., 2001). Thus, the model with three subgroups seemed reasonable given the percentage of adolescents with problematic glycemic control as defined by the American Diabetes Association (2010) and based on data from the Diabetes Control and Complications Trial indicating risk for future health complications (DCCT Research Group, 1994; Nathan et al., 2005; Nathan et al., 2009). The adequacy of the final model was evaluated using statistical diagnostics (see Supplemental Table) recommended by Nagin (2005), including the model estimate of group probability, average posterior probability, and odds correct classification. It was recommended that if the average posterior probability was greater than 0.7 and the odds correct classification was greater than 5 for all of the groups in the model, the trajectory model represented a high level of accuracy in classifying individuals into their specific trajectory assignment (Nagin, 2005).

Univariate ordinal logistic regression models were run to separately examine individual correlates and predictors of glycemic control trajectory group membership to determine the set of individual and family-level factors that should be included in a final, multivariate predictive ordinal logistic regression model. In the multivariate ordinal logistic regression model, we identified the set of individual and family-level factors that predicted membership in each subgroup, where membership was obtained using the maximum posterior probability rule, using ordinal logistic regression in SAS with the LOGISTIC procedure where all the predictors were entered simultaneously. Wald chi-squared values and odds ratios are presented for statistically significant predictors. Statistical significance was defined as $p < .05$.

Results

Hypothesis 1. Identification of Glycemic Control Trajectories

As hypothesized, three subgroups or trajectories of glycemic control were identified for the present sample (Figure 1) based on LGTM: 1) low risk for future diabetes-related complications ($n = 103$, 42.9% of patients) with an average HbA1c at baseline that was at ADA (2010) recommendations for the school age group ($M = 7.28$) and remained relatively

stable over time; 2) elevated risk for future diabetes-related complications (n = 107, 44.6% of patients) with an average HbA1c at baseline that was ~ 1% above ADA (2010) recommendations for the school age group (M = 8.58) and increased over time to 9.26% at three years; and, 3) high risk for future diabetes-related complications (n = 29, 12.1%) with an average HbA1c at baseline that was significantly above ADA recommendations (M = 10.02) and fluctuated over time but still remained high at three years (M = 11.4). The supplemental table presents the parameter estimates obtained for each of the trajectories as well as the diagnostic criteria established by Nagin (2005) for evaluating the fit of a given LGTM. As shown here, our final model fits the data well based on these criteria and model diagnostics indicated that individuals in the current sample were likely assigned to the appropriate glycemic control trajectory.

Hypothesis 2. Determination of Correlates and Predictors of Glycemic Control Trajectories

The descriptive statistics for each predictor variable based on glycemic control trajectory group membership as well as the results of the univariate and multivariate ordinal logistic regression models for determining baseline predictors of HbA1c trajectory groups are shown in Table 2. Univariate ordinal logistic regression models were conducted to identify which baseline predictor and covariates should be included in our multivariate logistic model. These analyses indicated that the potentially modifiable baseline individual and family-level factors that independently predicted glycemic control trajectory group membership, included: global executive functioning [Odds Ratio (OR) = 0.97, 95% CI: (0.95,1.00)], youth [OR=1.04, 95% CI: (1.01,1.08)] and maternal-reported diabetes self-management [OR=1.09, 95% CI: (1.06,1.12)], maternal-reported conflict [OR=0.86, 95% CI: (0.84,0.94)], and blood glucose monitoring frequency [OR=1.41, 95% CI: (1.22,1.64)]. Covariates that significantly predicted glycemic control trajectory group membership were gender [OR=1.99, 95% CI: (0.1.22,3.26)], site [OR site 1 versus 3=2.23, 95% CI: (1.15,4.31); OR site 2 versus 3=1.47, 95% CI: (0.75,2.91)], insulin therapy regimen [OR=0.38, 95% CI: (0.23,0.63)], one versus two parent households [OR=2.48, 95% CI: (1.36,4.53)]), maternal education level [OR=2.22, 95% CI: (1.42,3.46)], ethnicity and race [OR non-Hispanic Caucasian versus Hispanic individuals =2.43, 95% CI: (1.19,4.98); non-Hispanic minority versus Hispanic individuals =0.70, 95% CI: (0.26,1.85)], youth age [OR=0.67, 95% CI: (0.51,0.87)], and pubertal status [OR=0.62, 95% CI: (0.47,0.82)]. As shown in Table 2, those individuals who were identified as having low risk for future diabetes-related complications based on glycemic control trajectories over time also demonstrated higher levels of executive functioning, better diabetes self-management, reduced diabetes-related family conflict, and/or more frequent blood glucose testing compared to those identified as having an elevated or high risk for future complications.

A multivariate, ordinal, simultaneous logistic regression model was examined to determine the set of individual and family-level factors that significantly predicted glycemic control group membership over and above that of the other predictors. The univariate logistic regression model for adolescent-reported diabetes specific family conflict was not significant, and thus adolescent-reported conflict was not included in our final model. Similarly, youth and maternal-reported self-management behaviors at baseline were not significantly related to glycemic control group membership in the final model, and hence not

included in our final model given potential shared variance with blood glucose monitoring frequency. A test of the full model with all of the individual and family-level factors included was statistically significant, χ^2 (14, N = 216) = 60.35, p < .0001; Cramer's D = 0.54, which suggests 29% shared variance between glycemic control trajectory group membership and the set of predictors.

As expected, in the multivariate model, mother's report of family conflict [OR=0.89, 95% CI: (0.82,0.96)], blood glucose monitoring frequency [OR=1.20, 95% CI: (1.02,1.43)], and gender [OR=2.45, 95% CI: (1.32,4.58)] were all statistically significant predictors of glycemic control trajectory membership when controlling for the other variables. Mothers in the elevated risk (M = 25.13) and high risk group (M = 26.68) reported higher levels of diabetes-specific family conflict compared to those in the low risk group (M = 23.52). Treatment adherence as measured by blood glucose monitoring frequency was higher in the low risk glycemic control group (M = 5.5) compared to the elevated risk (M = 4.8) and high risk groups (M = 3.8). Adolescent girls were more likely to be in the elevated risk (55.1%) and very high risk groups (79.3%) compared to adolescent boys. However, adolescents' global executive functioning (based on maternal report) was not significantly related to glycemic control group membership in the multivariate logistic model, which was unexpected.

Discussion

Our primary descriptive finding was that a group-based trajectory analysis yielded three longitudinal glycemic control trajectories. Two of these subgroups (56.7% of the sample) had average HbA1c values across the three years that were higher than that recommended by the ADA for school age children and adolescents (i.e., HbA1c targets of 7.5–8.0%) (American Diabetes Association, 2010; Silverstein et al., 2005): 1) *high risk glycemic control subgroup* with average glycemic control of 10% at baseline; and, 2) *elevated risk glycemic control subgroup* (44.6%) with a mean baseline HbA1c value of 8.58%, which steadily increased over the three year period. The present study replicated findings reported by Luyckx and Seiffge-Krenke (2009) and Hilliard and colleagues (2013) such that three subgroups representing different levels of glycemic control over time were obtained: low risk, elevated risk, and high risk subgroups.

One contribution of our findings is the identification of trajectories of glycemic control at the onset of adolescence that continued through mid-adolescence. The combination of increased degree and duration of exposure to problematic glycemic control puts individuals at an increased risk for developing a wide range of diabetes-related complications in adulthood (DCCT Research Group, 1994; Nathan et al., 2005; Nathan et al., 2009; Silverstein et al., 2005). Research conducted by the Diabetes Control and Complications Trial and its follow-up studies (e.g., Epidemiology of Diabetes Interventions and Complications) suggested that there is a strong exponential relationship between the risk for retinopathy and neuropathy and mean HbA1c over time: a 10% decrease in HbA1c (e.g., 9.0% to 8.1%) was associated with a 39% risk reduction for retinopathy and a 25% risk reduction for microalbuminuria (i.e., neuropathy) (Diabetes Control and Complications Trial, 2002).

Univariate logistic regression analyses suggested that a number of potentially modifiable individual and family-level factors at baseline, including several individual and family-level covariates, significantly predicted glycemic control trajectory group membership when these variables were examined in isolation. Our findings were consistent with previous research such that less adaptive diabetes self-management (youth, maternal caregivers), deficits in executive functioning (maternal reporters), increased diabetes-related family conflict (maternal caregivers), and less frequent blood glucose monitoring were related to elevated and high risk glycemic control trajectory group membership (see Table 2).

However, our findings extended previous research by identifying two potentially modifiable variables in a multivariate predictive model: one individual level variable (i.e., blood glucose monitoring frequency) and one family-level variable (diabetes-related family conflict as perceived by maternal caregivers) as predictors of membership in clinically-relevant trajectories of glycemic control when controlling for a number of other individual and family-level predictors and covariates. The importance of BGM in glycemic control may stem from its multifaceted utility: The more frequent the blood testing, the more precisely decisions regarding insulin management can be accomplished. Frequency of blood glucose testing is especially important given the variability of blood glucose and the need for feedback throughout the day in order to adjust insulin (Patton et al., 2012).

Higher levels of maternal-reported family conflict were also associated with more problematic trajectories of glycemic control. Family conflict may have a multifaceted impact on glycemic control by interfering with the quality of diabetes management and treatment adherence and/or by increasing level of stress on the child and parents (Drotar et al., 2013). In addition, family conflict could also be representative of parent-child relationship quality, which has been known to be predictive of glycemic control (Miller-Johnson et al., 1994). However, it is notable that in contrast to previous research (Hilliard et al., 2012; Ingerski et al., 2010), adolescent reports of family conflict were not predictive of glycemic control group membership We studied the present sample at the cusp of adolescence (*M age* = 10.5) and it is possible that these young adolescents do not yet perceive their parental requests to manage their diabetes as conflictual but instead perceive these requests as normal parenting behavior. Finally, one non-modifiable covariate, gender also had a significant relationship with group trajectory status: girls were more likely to demonstrate trajectories of glycemic control reflecting risk status than boys. One factor that may account for this difference is the higher risk for eating disorders in girls or blood glucose dysregulation associated with menstrual cycles (Maharaj, Rodin, Olmsted, Connolly, & Daneman, 2003).

Our findings have important implications for both clinical management and research that focuses on preventive interventions to reduce the levels of risk for problematic trajectories of glycemic control. First, the different levels of glycemic control and risk for future health complications that are experienced by the different trajectory subgroups provide a logical basis for tailoring more intensive psychological interventions to young adolescents with the highest risk for future health complications. Our findings suggest that both the elevated risk and the high risk groups are in need of earlier identification and comprehensive medical and psychological intervention, such as more frequent medical visits and family-centered

interventions (Wysocki et al., 2008). On the other hand, the group who is maintaining glycemic control within target levels would benefit from continuing support, medical follow-up, and reinforcement from their providers. It should be noted that the subgroups of trajectories that reflected very different categories of risk status were relatively stable over the course of the three year follow-up. This finding suggests that it may also be advantageous to provide preventative adherence promotion interventions to the subgroups who are at elevated and very high risk prior to adolescence.

Because pediatric diabetes centers routinely collect information regarding glycemic control over time as well as frequency of blood glucose monitoring, they could identify pre-adolescents with trajectories of glycemic control over time that reflect higher risk and target key modifiable individual and family factors (e.g., family conflict and treatment adherence) in preventative interventions. Depending on the response to intervention, additional intervention or booster sessions might also be given during adolescence to enhance glycemic control that remains below target levels.

Previous research has provided strong evidence that family-based interventions that target both youth and parents improve treatment adherence and ameliorate family conflict (Butler et al., 2008; Harris et al., 2008; Wysocki et al., 2008). These interventions seem to be most powerful at improving glycemic control when they target specific areas of problem-solving (e.g., how to respond to low, high, and variable blood sugar values; identification of barriers and facilitators of treatment adherence (Butler et al., 2008; Harris et al., 2008; Wysocki et al., 2008). Anderson and colleagues (1999) suggested that a low-intensity intervention delivered during routine diabetes medical visits could positively influence diabetes management, enhance parent-adolescent communication and increase parental involvement in diabetes care while maintaining and promoting adolescent involvement in their own care, and decreasing diabetes-related family conflict. Such an intervention might be most appropriate and realistic for those adolescents who demonstrate trajectories of control consistent with elevated risk. Those at highest risk may require more intensive interventions such as multisystemic therapy (Ellis et al., 2012).

The present study has several limitations that should be considered in interpreting these findings. For example, executive functioning was measured by parental report. Parents may not be fully aware of their adolescents' executive functioning behaviors (e.g., planning, working memory, problem-solving, and moderation of emotions). Self-management was also measured via parental report and youth report and may have been biased due to social desirability. Other limitations include: the follow-up period of only three years and fact that other relevant factors may have influenced glycemic control trajectories (e.g., anxiety, depression, peer relationships, diabetes knowledge, parent-child relationship quality, parental involvement, child's maturity level, etc.) that were not included in our predictive model.

Future research is needed to address these limitations. It will be important to determine whether the subgroups of trajectories that were identified in this sample of early to mid-adolescents are sustained during later adolescence and young adulthood. Such data will be critical in planning for the transition from pediatric diabetes centers to adult care. If these

glycemic control subgroups are sustained through late adolescence, this would necessitate very different levels of intensity of transitional planning and services to facilitate optimal health outcomes. In addition, the efficacy of interventions that are tailored to different trajectories of glycemic control should be evaluated to identify those interventions that result in the greatest and most long-lasting reductions in glycemic control and minimize the risk for diabetes-related complications in adulthood.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

American Diabetes Association. Standards of medical care in diabetes. Diabetes Care. 2010; 33:S11–S61. [PubMed: 20042772]

Amiel SA, Sherwin RS, Simonson DC, Lauritano AA, Tamborlane WV. Impaired insulin action in puberty: A contributing factor to poor glycemic control in adolescents with diabetes. New England Journal of Medicine. 1986; 315:215–219. [PubMed: 3523245]

Anderson BJ, Brackett J, Ho J, Laffel L. An office-based intervention to maintain parent-adolescent teamwork in diabetes management. Impact on parent involvement, family conflict, and subsequent glycemic control. Diabetes Care. 1999; 22:713–721. [PubMed: 10332671]

Bagner DM, Williams LB, Geffken GR, Silverstein JH, Storch EA. Type 1 diabetes in youth: The relationship between adherence and executive functioning. Children's Health Care. 2007; 36:169–179.

Butler DA, Zuehlke JB, Tovar A, Volkening LK, Anderson BJ, Laffel L. The impact of modifiable family factors on glycemic control among youth with type 1 diabetes. Pediatric Diabetes. 2008; 9:373–381. [PubMed: 18774997]

Danne T, Mortensen HB, Hougaard P, Lynggaard H, Aanstoot HJ, Chiarelli F, Greene SA. Persistent differences among centers over 3 years in glycemic control and hypoglycemia in a study of 3,805 children and adolescents with type 1 diabetes from the hvidøre study group. Diabetes Care. 2001; 24:1342–1347. [PubMed: 11473067]

DCCT Research Group. Effect of intensive diabetes treatment on the development and progression of long-term complications in adolescents with insulin-dependent diabetes mellitus: Diabetes control and complications trial. Journal of Pediatrics. 1994; 125:177–188. [PubMed: 8040759]

Diabetes Control and Complications Trial. Effect of intensive therapy on the microvascular complications of type 1 diabetes mellitus. JAMA. 2002; 287:2563–2569. [PubMed: 12020338]

Drotar D, Ittenbach R, Rohan JM, Gupta R, Pendley JS, Delamater A. Diabetes management and glycemic control in youth with type 1 diabetes: Test of a predictive model. Journal of Behavioral Medicine. 2013; 36:234–245. [PubMed: 22569775]

Ellis DA, Naar-King S, Chen X, Moltz K, Cunningham PB, Idalski-Carcone A. Multisystemic therapy compared to telephone support for youth with poorly controlled diabetes: Findings from a randomized controlled trial. Annals of Behavioral Medicine. 2012:1–9.

Fogel NR, Weissberg-Benchell J. Preventing poor psychological and health outcomes in pediatric type 1 diabetes. Current Diabetes Reports. 2010; 10:436–443. [PubMed: 20835901]

Gardner DSL, Tai ES. Clinical features and treatment of maturity onset diabetes of the young (mody). Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy. 2012; 5:101.

Gioia GA, Isquith PK, Guy SC, Kenworthy L. Behavior rating inventory of executive function. Child Neuropsychol. 2000; 6:235–238. [PubMed: 11419452]

Harris MA, Antal H, Oelbaum R, Burkloh LM, White NH, Wysocki T. Good intentions gone awry: Assessing parental miscarried helping in diabetes. Families, Systems, & Health. 2008; 26:393–403.

Harris MA, Wysocki T, Sadler M, Wilkinson K, Harvey LM, Buckloh LM, White NH. Validation of a structured interview for the assessment of diabetes self-management. Diabetes Care. 2000; 23:1301–1304. [PubMed: 10977022]

Helgeson VS, Honcharuk E, Becker D, Escobar O, Siminerio L. A focus on blood glucose monitoring: Relation to glycemic control and determinants of frequency. Pediatric Diabetes. 2011; 12:25–30. [PubMed: 20522169]

Helgeson VS, Palladino DK. Implications of psychosocial factors for diabetes outcomes among children with type 1 diabetes: A review. Social and Personality Psychology Compass. 2012; 6:228–242.

Helgeson VS, Snyder PR, Seltman H, Escobar O, Becker D, Siminerio L. Brief report: Trajectories of glycemic control over early to middle adolescence. Journal of Pediatric Psychology. 2010; 35:1161–1167. [PubMed: 20189951]

Hilliard ME, Holmes CS, Chen R, Maher K, Robinson E, Streisand R. Disentangling the roles of parental monitoring and family conflict in adolescents' management of type 1 diabetes. 2012

Hood KK, Butler DA, Anderson BJ, Laffel LMB. Updated and revised diabetes family conflict scale. Diabetes Care. 2007; 30:1764–1769. [PubMed: 17372149]

Ingerski LM, Anderson BJ, Dolan LM, Hood KK. Blood glucose monitoring and glycemic control in adolescence: Contribution of diabetes-specific responsibility and family conflict. Journal of Adolescent Health. 2010; 47:191–197. [PubMed: 20638012]

Keough L, Sullivan-Bolyai S, Crawford S, Schilling L, Dixon J. Self-management of type 1 diabetes across adolescence. The Diabetes Educator. 2011; 37:486–500. [PubMed: 21602489]

Kichler JC, Kaugars AS, Ellis J, Alemzadeh R. Exploring self-management characteristics in youths with type 1 diabetes mellitus: Does membership in a glycemic control category matter?†. Pediatric Diabetes. 2010; 11:536–543. [PubMed: 20144180]

King PS, Berg CA, Butner J, Drew LM, Foster C, Donaldson D, Wiebe DJ. Longitudinal trajectories of metabolic control across adolescence: Associations with parental involvement, adolescents' psychosocial maturity, and health care utilization. Journal of Adolescent Health. 2012; 50:491–496. [PubMed: 22525113]

Kovatchev BP, Otto E, Cox D, Gonder-Frederick L, Clarke W. Evaluation of a new measure of blood glucose variability in diabetes. Diabetes Care. 2006; 29:2433–2438. [PubMed: 17065680]

Krolewski AS, Laffel LMB, Krolewski M, Quinn M, Warram JH. Glycosylated hemoglobin and the risk of microalbuminuria in patients with insulin-dependent diabetes mellitus. New England Journal of Medicine. 1995; 332:1251–1255. [PubMed: 7708068]

Levine BS, Anderson BJ, Butler DA, Antisdel JE, Brackett J, Laffel L. Predictors of glycemic control and short-term adverse outcomes in youth with type 1 diabetes. The Journal of pediatrics. 2001; 139:197–203. [PubMed: 11487743]

Luyckx K, Seiffge-Krenke I. Continuity and change in glycemic control trajectories from adolescence to emerging adulthood relationships with family climate and self-concept in type 1 diabetes. Diabetes Care. 2009; 32:797–801. [PubMed: 19228859]

Maharaj S, Rodin G, Olmsted M, Connolly J, Daneman D. Eating disturbances in girls with diabetes: The contribution of adolescent self-concept, maternal weight and shape concerns and mother-daughter relationships. Psychological Medicine. 2003; 33:525–539. [PubMed: 12701673]

McNally K, Rohan JM, Shroff Pendley J, Delamater A, Drotar D. Executive functioning, treatment adherence, and glycemic control in children with type 1 diabetes. Diabetes Care. 2010; 33:1159–1162. [PubMed: 20215458]

Miller-Johnson S, Emery RE, Marvin RS, Clarke W, Lovinger R, Martin M. Parent-child relationships and the management of insulin-dependent diabetes mellitus. Journal of Consulting and Clinical Psychology. 1994; 62:603–610. [PubMed: 8063987]

Miller M, Rohan JM, Delamater A, Pendley JS, Dolan L, Reeves G, Drotar D. Changes in executive functioning and self-management in adolescents with type 1 diabetes: A growth curve analysis. Journal of Pediatric Psychology. 2013; 38:18–29. [PubMed: 23027720]

Moran A, Jacobs DR Jr, Steinberger J, Cohen P, Hong CP, Prineas R, Sinaiko AR. Association between the insulin resistance of puberty and the insulin-like growth factor-i/growth hormone axis. Journal of Clinical Endocrinology and Metabolism. 2002; 87:4817–4820. [PubMed: 12364479]

Nagin, DS. Group-based modeling of development. Cambridge, MA: Harvard University Press; 2005.

Nathan DM, Cleary PA, Backlund J, Genuth SM, Lachin JM, Orchard TJ, Zinman B. Diabetes control and complications trial/epidemiology of diabetes interventions and complications (dcct/edic) study research group. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. New England Journal of Medicine. 2005; 353:2643–2653. [PubMed: 16371630]

Nathan DM, Zinman B, Cleary PA, Backlund JYC, Genuth S, Miller R, Orchard TJ. Modern-day clinical course of type 1 diabetes mellitus after 30 years' duration: The diabetes control and complications trial/epidemiology of diabetes interventions and complications and pittsburgh epidemiology of diabetes complications experience (1983–2005). Archives of Internal Medicine. 2009; 169:1307. [PubMed: 19636033]

Patton SR, Midyett LK, Dolan LM, Powers SW. A comparison of average daily risk range scores for young children with type 1 diabetes mellitus using continuous glucose monitoring and self-monitoring data. Diabetes Technology and Therapeutics. 2012; 14:239–243. [PubMed: 22047051]

Rausch JR, Hood KK, Delamater A, Shroff Pendley J, Rohan JM, Reeves G, Drotar D. Changes in treatment adherence and glycemic control during the transition to adolescence in type 1 diabetes. Diabetes Care. 2012; 35:1219–1224. [PubMed: 22474040]

Rohan JM, Delamater A, Pendley JS, Dolan L, Reeves G, Drotar D. Identification of self-management patterns in pediatric type 1 diabetes using cluster analysis. Pediatric Diabetes. 2011; 12:611–618. [PubMed: 21446925]

Rohan JM, Pendley JS, Delamater A, Dolan L, Reeves G, Drotar D. Patterns of self-management in pediatric type 1 diabetes predict level of glycemic control 2 years later. Journal of Developmental and Behavioral Pediatrics. 2013; 34:186–196. [PubMed: 23572169]

Schneider S, Iannotti RJ, Nansel TR, Haynie DL, Simons-Morton B, Sobel DO, Plotnick LP. Identification of distinct self-management styles of adolescents with type 1 diabetes. Diabetes Care. 2007; 30:1107–1112. [PubMed: 17322481]

Silverstein J, Klingensmith G, Copeland K, Plotnick L, Kaufman F, Laffel L, Holzmeister LA. Care of children and adolescents with type 1 diabetes a statement of the american diabetes association. Diabetes Care. 2005; 28:186–212. [PubMed: 15616254]

Wysocki T, Harris MA, Buckloh LM, Mertlich D, Lochrie AS, Taylor A, White NH. Randomized, controlled trial of behavioral family systems therapy for diabetes: Maintenance and generalization of effects on parent-adolescent communication. Behavior Therapy. 2008; 39:33–46. [PubMed: 18328868]

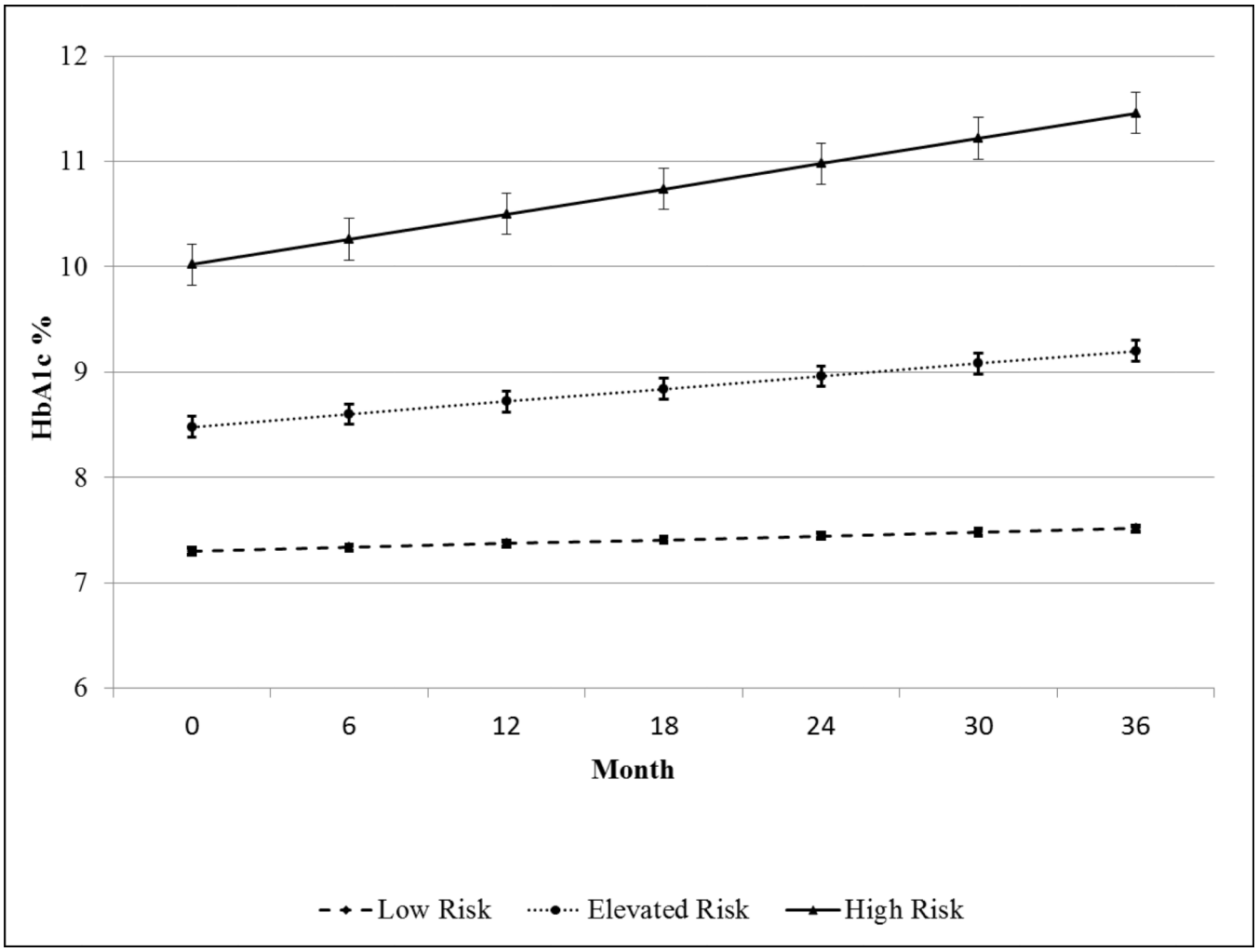

Figure 1.
Model-Based HbA1c Trajectories for Subgroups from Final Latent Group-based Trajectory Modeling Solution.

Table 1

Demographics of Sample from Baseline to Three Years

	Baseline	One Year	Two Year	Three Year
Child Age (years)* (mean ± SD; Range)	10.54 ± .94; 9.0–12.09	11.59 ± .97; 9.86–13.22	12.62 ± .96; 10.95–14.39	13.62 ± .97; 11.9–15.6
Duration of diabetes (years) (mean ± SD; Range)	4.41 ± 2.46; 1–11	5.43 ± 2.49; 2–12	6.46 ± 2.43; 3–13	7.48 ± 2.43; 4–14
HbA1c (mean ± SD; Range)	8.20 ± 1.37; 5.7–16.8	8.31 ± 1.38; 5.6–14.5	8.51 ± 1.41; 5.7–13.4	8.77 ± 1.60; 5.9–15.8
Child Gender				
Male (n, %)	109 (45.61)	103 (45.6)	104 (46.2)	101 (45.5)
Female (n, %)	130 (54.39)	123 (54.4)	121 (53.8)	121 (54.5)
Child Ethnicity/Race				
Non-Hispanic, Caucasian (n, %)	179 (74.9)	171 (75.7)	170 (75.6)	170 (76.6)
Non-Hispanic, Other (n, %)	27 (11.3)	26 (11.5)	25 (11.1)	23 (10.4)
Hispanic (n, %)	33 (13.8)	29 (12.8)	30 (13.3)	29 (13.1)
Insulin Regimen				
Injection (n, %)	109 (45.6)	76 (33.6)	67 (29.8)	68 (30.6)
Pump/Pod (n, %)	130 (54.4)	150 (66.4)	154 (68.4)	153 (68.9)
Insurance Type				
Private (n, %)	179 (74.9)	Not assessed	Not assessed	Not assessed
Public (Medicare/Medicaid) (n, %)	45 (18.8)	Not assessed	Not assessed	Not assessed
No insurance (n, %)	1 (0.4)	Not assessed	Not assessed	Not assessed
Unknown (n, %)	14 (5.9)	Not assessed	Not assessed	Not assessed
Household Composition				
One-Parent Household (n, %)	51 (21.3)	46 (20.4)	47 (20.9)	46 (20.7)
Two-Parent Household (n, %)	188 (78.7)	180 (79.6)	178 (79.1)	176 (79.3)
Maternal Education				
Did not finish high school (n, %)	9 (3.8)	6 (2.7)	7 (3.1)	7 (3.2)
Obtained high school diploma/equivalent (n, %)	70 (29.3)	68 (30.1)	65 (28.9)	66 (29.7)
Obtained some college/college degree (n, %)	159 (66.5)	151 (66.8)	152 (67.6)	148 (66.7)
Annual Household Income	$49,000–$72,999	Not assessed	Not assessed	Not assessed

Table 2

Univariate and Multivariate Ordinal Logistic Regressions: Predictors of HbA1c Trajectory Group Status

	Descriptive Statistics for Trajectories			*Univariate Model*			*Multivariate Model*		
Predictor Variable**	**Low Risk M ± SD or (%)**	**Elevated Risk M ± SD or (%)**	**High Risk M ± SD or (%)**	***df****	**Wald χ^2**	***p****	***df****	**Wald χ^2**	***p****
Executive Functioning (maternal)	50.89 ± 10.97	52.52 ± 11.27	57.39 ± 11.54	**1**	**5.15**	**.023**	1	0.19	.66
Diabetes Self-Management (youth)	61.81 ± 7.76	60.70 ± 7.79	58.56 ± 9.10	**1**	**7.15**	**.008**	N/A	N/A	N/A
Diabetes Self-Management (maternal)	67.84 ± 8.15	63.98 ± 8.39	58.56 ± 8.17	**1**	**30.59**	**< .001**	N/A	N/A	N/A
Family Conflict (youth)	25.35 ± 5.03	26.87 ± 6.33	25.76 ± 4.14	1	2.47	.116	N/A	N/A	N/A
Family Conflict (maternal)	23.52 ± 4.79	25.13 ± 4.23	26.68 ± 4.17	**1**	**16.91**	**< .001**	**1**	**8.18**	**.004**
BGMF	5.52 ± 1.78	4.87 ± 1.80	3.77 ± 1.55	**1**	**20.51**	**< .001**	**1**	**4.63**	**< .05**
Gender	Male (53.4%) Female (46.6%)	Male (44.9%) Female (55.1%)	Male (20.7%) Female (79.3%)	**1**	**7.49**	**.006**	**1**	**7.97**	**.005**
Site	Site 1 (49.5%) Site 2 (35.9%) Site 3 (14.6%)	Site 1 (49.5%) Site 2 (29.0%) Site 3 (21.5%)	Site 1 (13.8%) Site 2 (55.2%) Site 3 (31.0%)	**2**	**6.10**	**.047**	2	2.35	.31
Diabetes Duration	4.36 ± 2.46	4.66 ± 2.53	4.00 ± 2.55	1	0.002	.96	1	1.99	.16
Insulin Regimen	Injection (35%) Pump (65.0%)	Injection (46.7%) Pump (53.3%)	Injection (79.3%) Pump (20.7%)	**1**	**14.13**	**< .001**	1	1.12	.29
Household, Single versus Two Parent	Single (12.6%) Two (87.4%)	Single (26.2%) Two (73.8%)	Single (34.5%) Two (65.5%)	**1**	**8.84**	**.003**	1	1.76	.18
Maternal Education, % college education	(79.4%)	(59.8%)	(48.3%)	**1**	**12.15**	**.001**	1	3.63	.06
Ethnicity/Race, % Non-Hispanic, Caucasian	(84.5%)	(72.0%)	(51.7%)	**2**	**13.35**	**.001**	2	2.09	.35
Age	10.33 ± 0.94	10.63 ± 0.93	10.86 ± 0.81	**1**	**9.07**	**.003**	1	3.69	.055
Tanner Stage	1.58 ± .78	1.74 ± .90	2.32 ± 1.21	**1**	**11.09**	**.001**	1	0.28	.60

* *df* - degrees of freedom; statistically significant predictors are bold.

** Executive functioning: higher scores represent more self-reported deficits in executive functioning; Diabetes self-management: higher scores indicate better self-management patterns; Family conflict: higher scores suggest more diabetes-related family conflict