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Family Functioning and Adherence in Youth With Type 1 Diabetes: A Latent Growth Model of Glycemic Control

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The objective of this research is to determine the direct impact of family variables on initial status of a glycosylated hemoglobin A1c test (HbA1c) and HbA1c rate of change, and to determine the indirect effects of family variables on HbA1c through adherence. Study participants were 224 children and their parents who completed baseline measures of diabetes-specific family functioning and separate parent- and child-structured adherence interviews. HbA1c assays were performed at baseline, Year 1, and Year 2. Latent growth curve modeling indicated that, together, disagreement about responsibility for diabetes regimen, critical parenting, parental guidance, and parental warmth predicted initial status of HbA1c through adherence. Individually, critical parenting indirectly predicted initial status of HbA1c. Family variables did not directly predict initial status or rate of change in HbA1c, and did not indirectly predict rate of change in HbA1c over 2 years. Risk for high HbA1c remains elevated over time for children reporting critical parents, but their trajectory does not indicate compounding risk over time.

Type 1 diabetes (T1D) is one of the most common chronic illnesses of childhood, affecting 1 out of every 400 to 600 children under age 20 (National Institute of Diabetes, 2005). Children with T1D are at risk for health complications, including ketoacidosis, heart disease, kidney disease, blindness, and gangrene (American Diabetes Association [ADA], 2006). The Diabetes Control and Complications Trial (1993) demonstrated that risk for the aforementioned complications is reduced in those who consistently have good glycemic control. Glycemic control is operationalized via a glycosylated hemoglobin A1c test (HbA1c) for which higher values are indicative of poorer glycemic control. Unfortunately, glycemic control generally becomes worse over time for many children (Jacobsen et al., 1994), highlighting the need for intervention. However, the medical management of T1D can be challenging for children and adolescents due to the varied and intrusive nature of the behaviors required (e.g., monitoring of blood glucose, insulin injections, and dietary restraint). Given these challenges, the most significant barriers to effective treatment are behavioral (Wysocki, Buckloh, Lochrie, & Antal, 2005). As many as 45% of those with T1D report some form of significant non-adherence during adolescence (Kovacs, Kass, Schnell, Goldston, & Marsh, 1989).

Patterson's (1982) coercion model offered a framework for conceptualizing non-adherence in youth with T1D. This model describes a relationship between some parents and children in which parental demands for compliance are associated with child refusal to comply, ending with parental submission. In subsequent iterations of this cycle, the demands for compliance and refusals can escalate, creating a hostile or aggressive home environment. In families characterized by such a coercive cycle, adherence to the diabetes regimen may suffer, negatively impacting glycemic control. Duke et al. (2008) supported the application of Patterson's coercion model to youths with T1D, demonstrating that adherence mediates the relations between critical parenting and glycemic control, and that it mediates the relation between youths externalizing symptoms and glycemic control.

Research testing adherence as a mediator between family variables and glycemic control also supports this model. Family variables refer to those that characterize the type or quality of interactions among family members. Miller-Johnson et al. (1994) reported that adherence fully mediated the relation between parent–child conflict and glycemic control. Lewin et al. (2006) demonstrated adherence as a partial mediator between combined family variables and glycemic control in a cross-sectional model. Others have demonstrated that family variables have a direct relation with glycemic control. For example, positive parental emotional support (e.g., relating to the child about having diabetes) and parental guidance that is not perceived by the child as coercive are associated with improved glycemic control (McKelvey et al., 1993; Waller et al., 1986). These variables may be associated with families who do not experience a coercive cycle, resulting in an optimal environment for good adherence. Similarly, parental monitoring of adolescent regimen completion and high family cohesion are associated with improved glycemic control (Cohen, Lumley, Naar-King, Partridge, & Cakan, 2004; Ellis et al., 2007). There is also evidence that family cohesion and adaptability are moderators of the relation between demographic variables (i.e., age and gender) and glycemic control (Cohen et al., 2004). Again, family cohesion may be a marker for positive relationships among family members and indicates that the coercive cycle is not occurring in those families with high adaptability and cohesion. In addition, parental behaviors specifically related to diabetes care that are perceived by the child as negative and unsupportive (e.g., coercion, threats, and criticism) are significantly related to poor glycemic control (L. C. Schafer, Glasgow, McCaul, & Dreher, 1983; L. C. Schafer, McCaul, & Glasgow, 1986). This is consistent with Patterson's (1982) coercion model, which would predict that children perceiving parents as critical are likely to be associated with refusal to comply. Parent–child disagreement about who is responsible for the implementation of the diabetes medical regimen is predictive of poor glycemic control (Anderson, Auslander, Jung, Miller, & Santiago, 1990). More recently, poor parental problem-solving skills have been related to reduced glycemic control in children (Wysocki, Iannotti, et al., 2008).

Researchers have suggested that incorporating these family variables into one model may yield stronger results than in those studies that examine family variables individually (Lewin et al., 2006; McKelvey et al., 1993). Lewin et al. (2006) investigated the following diabetes-specific family factors and their relation to glycemic control: parental warmth and caring, parental guidance and control, child perception of critical or unsupportive parents, and shared responsibility for diabetes management tasks. After controlling for demographic variables, these factors together predicted 34% of the variance in glycemic control. Individually, child perception of critical parenting ($\beta = .45$) and disagreement about responsibility for the diabetes regimen ($\beta = .17$) significantly predicted variance in glycemic control. Although a temporal relation could not be concluded, this study highlights the importance of considering multiple family dimensions related to glycemic control. A longitudinal model may more effectively be translated into a targeted intervention to improve glycemic control in children with T1D.

The majority of studies investigating the relation between family factors and glycemic control have occurred within the restrictions of cross-sectional designs, limiting the causal inferences that can be drawn. One notable exception is Jacobsen et al. (1994), who followed a cohort of 61 children with T1D across 4 years and found that encouragement of expressiveness, greater family cohesion, and reduced conflict were associated with reduced degradation of glycemic control over time. In other words, although all youths experienced a decline in glycemic control, those with greater encouragement of expressiveness, greater family cohesion, and reduced conflict did not experience as great a decline as their peers. However, no studies have investigated the combined impact of multiple family factors on glycemic control or adherence as a mediator of this relation using a longitudinal design. To determine which elements act as protective factors or require intervention to improve glycemic control, the relations among the aforementioned variables require temporal elucidation.

Prior studies of family variables in youths with T1D indicate that those variables characterizing positive relationships (e.g., child perception of parent warmth and parent guidance) are related to improved adherence and glycemic control, whereas those characterizing negative interactions (e.g., child perception of critical parenting and disagreement about diabetes care) are related to poorer adherence and glycemic control. This is consistent with Patterson's (1982) coercion model, which suggests that families characterized by negative interactions are likely to have non-compliant children. However, these relationships have not previously been tested in one model for their relation to longitudinal glycemic control. Given the lack of previous investigation in this area, this study seeks to determine the direct effect of family variables on *initial status* of HbA1c and on *rate of change* in HbA1c, and to determine the indirect effects of family variables on HbA1c through adherence. The use of a longitudinal design has two major advantages in testing the relation between family factors and glycemic control. First, the sequential order of variables in time allows greater causal inference than with a cross-sectional design (although it does not demonstrate a true causal relation as an experimental design would allow). Second, the demonstrated tendency of glycemic control to worsen over time suggests that the effects of deleterious family factors may have a cumulative effect, which has implications for intervention. Specifically, existing interventions that address family within the context of T1D, such as behavioral family systems therapy

(Wysocki et al., 2007; Wysocki et al., 2006; Wysocki, Harris, et al., 2008), may benefit from identification of family variables related to change in HbA1c over time. As previous data are entirely cross-sectional, this study represents the first examination of such a model. In this investigation, we used baseline family functioning and adherence date from participants in a prior study to predict longitudinal HbA1c. Based on Patterson's coercion model and previous studies testing the mediation potential of family variables (e.g., Lewin et al., 2006; Miller-Johnson et al., 1994), we hypothesized that baseline indexes of family functioning would have indirect effects through baseline adherence on both initial status of HbA1c and rate of change in HbA1c over 2 years.

METHOD

Participants

Participants were 224 (53.8% females) children with T1D, ranging in age from 8 to 18 years ($M = 13.82$, $SD = 2.61$). They were recruited from a university-affiliated pediatric diabetes clinic serving a largely rural community and whose patients are typically sponsored by state-funded insurance. Yearly family income ranged from \$6K to \$155K ($M =$ \$46K, $SD =$ \$29K, $Mdn =$ \$39K). Fifty-two participants (23.2%) were age 16 years or older at baseline and, thus, were followed past the age of 18 years. The ethnicity of participants included 75.9% Caucasian, 11.2% Black or African American, 9.4% Hispanic, and 3.5% representing other ethnic groups. Participants were primarily from two-parent households (70.1%). Average HbA1c at baseline was 8.84% ($SD = 1.80$), at Year 1 was 9.10% ($SD = 1.81$), and at Year 2 was 9.34% ($SD = 2.01$). Average duration since diagnosis with diabetes was 5.17 years ($SD = 3.68$), and average age at diabetes onset was 8.46 ($SD = 4.21$).

Of these participants, 109 (48.7%) were participants in a prior cross-sectional study of youths with T1D (Lewin et al., 2006), and 115 (51.3%) participants were part of a larger cross-sectional study of family variables and T1D (Duke et al., 2008; Lehmkuhl et al., 2009). Due to the cross-sectional nature of the original studies, data were unavailable for longitudinal measurement of family functioning variables or for adherence (see Table 1 for a list of measures and timing of administration). However, this investigation represents a significant departure from those studies given the addition of longitudinal HbA1c data and testing of one model that includes the relevant variables. The administration of self-report and interview measures was identical in both studies, as they were administered by the same team of trained interviewers using the same procedures, and biological assays were conducted using the same equipment and laboratory across all studies. Analysis of all variables by study membership yielded no

TABLE 1
Timing of Administration for All Measures

Measure	*Time Administered*		
	Baseline	*Year 1*	*Year 2*
Family functioning			
Critical parenting (DFBC)	X		
Parent warmth (DFBS)	X		
Parent guidance (DFBS)	X		
No responsibility for regimen (DFRQ)	X		
Adherence			
Parent rated (DSMP)	X		
Child rated (DSMP)	X		
Glycemic control (HbA1c)	X	X	X

Note. DFBC = Diabetes Family Behavior Checklist; DFBS = Diabetes Family Behavior Scale; DFRQ = Diabetes Family Responsibility Questionnaire; DSMP = Diabetes Self-Management Profile; HbA1c = glycosylated hemoglobin A1c test.

significant differences ($p > .05$). As part of the original studies, caregivers and children were recruited based on the following criteria: (a) children aged 8 to 18 years, (b) a diagnosis of T1D for at least 1 year, (c) living with and accompanied by their primary caregiver, and (d) no evidence of mental retardation.

Measures of Diabetes Specific Family Functioning (Measured at Baseline)

Diabetes Family Behavior Scale (DFBS; Waller et al., 1986). The DFBS is a child-rated measure of perceived family support specific to diabetes. For this study, only the 15-item subscales measuring parental warmth and caring (e.g., "My parent understands how I feel about having diabetes") and guidance and control (e.g., "My parent reminds me to test my blood sugar") were used. Responses to this scale are given on a 5-point scale ranging from 1 (*all of the time*) to 5 (*never*). The DFBS has shown adequate internal consistency (Cronbach's $\alpha = 0.76$–0.79) and test–retest reliability (3-week coefficients = 0.79–0.83; Waller et al., 1986). In this sample, the DFBS demonstrates adequate internal consistency (Cronbach's $\alpha = 0.77$).

Diabetes Family Behavior Checklist (DFBC; L. C. Schafer et al., 1986). The DFBC is a measure of frequency of family support behaviors related to the diabetes regimen. For this study, the child-rated version was used. In addition, only the seven-item non-supportive family behavior domain was used (e.g., "nag you about following your diet"). Responses are given on a 5-point scale ranging from 1 (*never*) to 5 (*at least once a day*). The DFBC has shown good internal consistency (Cronbach's $\alpha = 0.74$–0.82; Lewin et al., 2006; L. C. Schafer et al., 1986). In this sample, the DFBC demonstrates adequate internal consistency (Cronbach's $\alpha = 0.75$).

Diabetes Family Responsibility Questionnaire (DFRQ; Anderson et al., 1990). The DFRQ is a measure completed separately by both the parent and child, and is meant to assess family sharing of responsibilities for diabetes management. This measure consists of 17 statements concerning diabetes management tasks (e.g., remembering to take insulin), and asks the respondent to indicate which family member is responsible for that task (i.e., parent, child, or both). A "no-responsibility" score is calculated based on patterns of disagreement between the responses of the parent and that of the child. Higher scores indicate that neither the parent nor the child take responsibility for the task. The DFRQ demonstrates good internal consistency (Cronbach's $\alpha = 0.85$–0.89; Anderson et al., 1990; Lewin et al., 2006). In this sample, the DFRQ demonstrates good internal consistency (Cronbach's $\alpha = 0.85$).

Measurement of Adherence (Measured at Baseline)

Diabetes Self-Management Profile (DSMP; Harris et al., 2000). The DSMP is a 23-item, semi-structured interview. For purposes of this study, it was administered to the child without the parent present and to parents without the child present. Questions are designed to assess five areas of diabetes management: insulin administration and dose adjustment, blood glucose monitoring, exercise, diet, and management of hypoglycemia. Items are answered in an open-ended manner and coded by trained interviewers. Most items are scored on a 5-point scale relative to the content of the question (e.g., "always eats more or gives more insulin," "frequently eats more or gives less insulin," "sometimes eats more or gives less insulin," "occasionally eats more or gives less insulin," and "eats less than usual or gives more insulin"). Items from all domains are summed to produce a total adherence score, yielding a separate parent score and child score. The DSMP has shown good internal consistency (Cronbach's $\alpha = 0.69$–0.76; Harris et al., 2000; Lewin et al., 2006) and interobserver agreement (94%; Harris et al., 2000). Previous research with the DSMP has shown that it is strongly related to concurrent HbA1c (Lewin et al., 2005). In this sample, the DSMP demonstrates good internal consistency for caregivers (Cronbach's $\alpha = 0.85$) and marginal for youths (Cronbach's $\alpha = 0.68$). In this sample, child-reported ($r = -.39$, $p < .01$) and parent-reported scores on the DSMP were significantly related to baseline HbA1c ($r = -.52$, $p < .01$).

Measurement of Glycemic Control (Measured at Baseline, Year 1, and Year 2)

Glycemic control is a biological assay of health status operationalized via HbA1c. HbA1c provides an estimate of the glycemic control of a given individual over the previous 2 to 3 months (ADA, 2006). Blood samples were analyzed using a Bayer DCA 2000+ (Pittsburgh, PA). All HbA1c assays were performed by the same hospital laboratory for all participants.

Procedure

Data were collected as part of initial participation in other studies in our lab for which the institutional review board (IRB) granted approval and participants consented (Duke et al., 2008; Lewin et al., 2006). For the Lewin et al. (2006) data, a waiver of informed consent was granted by the IRB to collect additional data in the form of HbA1c levels at Years 1 and 2 past baseline. Participants in the Duke et al. study consented to allow additional data collection past the initial date of participation. In both studies, a trained investigator approached all families meeting eligibility criteria during their regularly scheduled office visits to the diabetes clinic. Measures took approximately 25 min to complete, and children and caregivers were interviewed separately. Participants were instructed to respond to all study measures based on the 3 months prior. Family functioning and adherence data were collected at baseline only. HbA1c levels at baseline, Year 1, and Year 2 were collected via a finger-stick during routine medical visits and obtained for study purposes through the hospital's computerized medical records information system.

Data Analysis

Pearson's correlations and regression analyses were used to examine demographic variables and to determine their relation to study variables for consideration as covariates to be included in the primary model. To differentiate the effects of age at baseline, age at diabetes onset, and duration of diabetes, a series of paired multiple regression analyses were conducted to examine patterns of significance in these variables while systematically controlling the others. In this situation, a single multiple regression with all predictors is inappropriate due to extreme multicollinearity. This paired regression approach, previously described by Johnson and Meltzer (2002), identifies predictors of interest among the three variables, despite each being perfectly predictable by the other two. For example, if the predictor of interest is baseline age, two models are tested: one controlling for duration of diabetes and one controlling for age at onset. Only the effects of baseline age remaining significant in both models are considered true baseline age effects. In other words, a predictor is considered to have a true effect if it remains significant across the pair of regressions.

Latent growth modeling (LGM) was used to examine the hypothesized relations among family variables (child perception of parental warmth, parental guidance, critical parenting, and discrepancy between parent and child report of responsibility for the diabetes regimen), adherence, and HbA1c over time.[1] LGM is a structural equation modeling approach, which estimates factors representing initial status (intercept; η_0) and rate of change over time (slope; η_1). This approach allows the test of both direct and indirect paths among Level 1 and Level 2 factors and associated predictors (Bollen & Curran, 2006; Duncan, Duncan, & Strycker, 2006). In the case of longitudinal data, Level 1 variables are those that are measured *within* an individual at different times. Level 2 variables are those measured at one time or estimated *between* individuals. Given that the current HbA1c data represent three points of yearly measurement, this model estimated a linear slope factor. This study examined the fit of three increasingly complex models.[2] Model 1 (see Figure 1) established the linear change trajectory necessary for conducting latent growth curve analysis through measurement of the intercept (η_0) and slope (η_1) factors of HbA1c. It also estimated a latent adherence factor, which was measured by separate parent- and child-rated adherence to obtain a common adherence construct that represented an adherence rating unbiased by joint interview. Despite the importance of Model 1 for establishing these initial values, it was expected to fit poorly in the absence of predictors. Model 2 (see Figure 1) established relations among study variables and covariates that were identified during preliminary data analysis, and provided a model for comparison against the final model. Model 3 added hypothesized paths from family variables to adherence, from adherence to intercept and slope factors, and from family variables to intercept and slope factors, testing both direct and indirect effects of family variables on the initial status and rate of change in HbA1c.

Data were analyzed in Mplus (Muthen & Muthen, 1998–2004) using maximum likelihood estimation and considering data missing completely at random (MCAR). Analysis of missing data indicated that the MCAR assumption was supported. One benefit of maximum likelihood estimation, given the assumption that data can be considered MCAR, is that it utilizes all observations in the dataset, including those with a large amount of missing data at one or more times (J. L. Schafer & Graham, 2002). In this analysis, 32.7% or participants

[1]The covariance matrix is available from Kristen M. Grabill upon request.

[2]Models were also estimated in using error terms set equal to (1 − reliability) × (variance). This approach corrects for loss of power due to low reliability of measures. However, given no significant parameter or overall model fit differences when using the approach, the model described in this investigation is one that does not correct for low reliability.

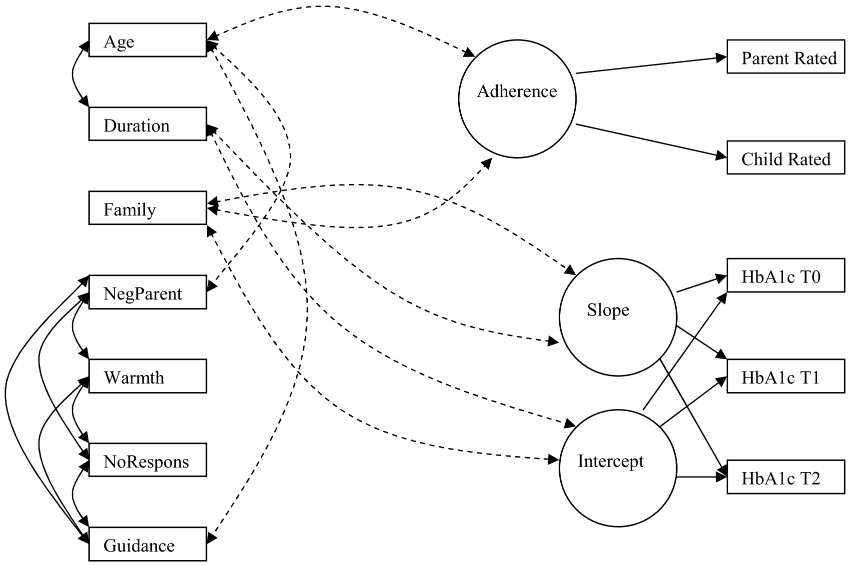

FIGURE 1 Models 1 and 2. *Note*. Solid lines represent paths in Model 1. Dashed lines represent paths added in Model 2. Age = baseline age; Duration = duration with diabetes; Family = single- or two-parent family status; NegParent = child perception that parent is critical or negative; Warmth = child perception of parental warmth; NoRespons = parent and child disagreement about responsibility for the diabetes regimen; Guidance = child perception of parent guidance or control; HbA1c = glycosylated hemoglobin A1c test.

were missing HbA1c data at Year 1, whereas 43.1% were missing data at Year 2. Model fit was evaluated using model chi-square statistic, chi-square change statistic, Tucker–Lewis Index (TLI), comparative fit index (CFI), root mean square error of approximation (RMSEA), Akaike's Information Criteria (AIC), and Bayesian Information Criteria (BIC). Suggested criteria are nonsignificant model chi-square, significant decrease in nested chi-square test, TLI $\geq$ 0.95, CFI $\geq$ 0.95, RMSEA $\leq$ 0.06, and decrease in AIC and BIC with better fitting models (Hu & Bentler, 1999). Standardized values are reported unless otherwise noted.

RESULTS

Descriptive Statistics

Initial analysis indicated that family structure (one- vs. two-parent household) was significantly related to parent-rated adherence ($r = -.20$, $p < .01$), child-rated adherence ($r = -.23$, $p < .01$), baseline HbA1c ($r = .19$, $p < .01$), and 2-year HbA1c ($r = .35$, $p < .01$), such that one-parent households were related to poorer parent- and child-rated adherence and to higher baseline and 2-year HbA1c. This is consistent with other studies relating single-parent family composition to HbA1c studies (Hanson, Henggler, Rodrigue, Burghen, & Murphy, 1988; Harris, Greco, Wysocki, Elder-Danda, & White, 1999). Results of paired multiple regression analyses indicated that duration of diabetes showed a consistent relation with HbA1c when controlling for baseline age ($\beta = .20$, $p < .01$) and age at diabetes onset ($\beta = .36$, $p < .01$). Baseline age showed a consistent relation with child perception of parent guidance and control when controlling for duration of diabetes ($\beta = -.34$, $p < .01$) and age at diabetes onset ($\beta = -.32$, $p < .01$). Baseline age, duration of diabetes, and onset age did not show consistent relations with any other study variables. Family income, ethnic status (Caucasian, African American, Hispanic, Asian, or other), and child gender were not significantly associated with any of the study variables.

Latent Growth Curve Model

Model 1 indicated that, as expected, the initial model fit the data poorly, according to chi-square and all other fit indexes (see Table 2). Average HbA1c at baseline was 8.84 ($\eta_{0\text{unstandardized}} = 8.84$, $z = 74.65$, $p < .01$). The slope factor was significant ($\eta_{1\text{unstandardized}} = .34$, $z = 4.46$, $p < .01$), indicating an average increase of .34% in HbA1c each year (e.g., from 7.00%–7.34%). Variances of intercept and slope factors indicated significant heterogeneity in initial status and rate of change in HbA1c among participants ($p < .01$). Intercept and slope factors were not significantly related, indicating that initial status is not related to rate of change in HbA1c ($r = -.11$, $p > .05$). Parent- and child-rated adherence loaded onto the adherence factor significantly ($\lambda_{\text{parent}} = .73$, $\lambda_{\text{child}} = .56$, ps $< .01$), indicating approximately equal contribution to the adherence factor. Although contribution of child-rated data was slightly smaller

TABLE 2
Model Fit Indexes for All Models

Model	*df*	χ^2	$\Delta\chi^2$	*TLI*	*CFI*	*RMSEA*	*AIC*	*BIC*
1	55	140.15*	—	.68	.76	.10	12,556.87	12,676.27
2	45	114.54*	25.61*	.76	.84	.08	12,825.35	12,978.88
3	33	35.72	78.82*	.99	.99	.01	12,770.53	12,964.35

Note. TLI = Tucker–Lewis Index; CFI = comparative fit index; RMSEA = root mean square error of approximation; AIC = Akaike's Information Criteria; BIC = Bayesian Information Criteria.
*$p < .001$.

than that of parents, this may be due to relatively lower internal consistency of the child-rated measure.

Model 2 demonstrated less than adequate fit, but demonstrated a significant improvement in chi-square, RMSEA, TLI, and CFI (see Table 2). Duration of diabetes was significantly related to the HbA1c intercept ($r = .21$, $p < .01$), as was family structure ($r = .19$, $p < .01$), such that children having longer duration and single-parent households had higher initial HbA1c. Family structure was also significantly related to adherence, such that children from single-parent households had poorer reported adherence ($r = -.34$, $p < .01$). Age was significantly related to child perception of parent guidance and control ($r = -.31$, $p < .01$), but was not related to child perception of negative parenting ($p > .05$).

Model 3 demonstrated excellent fit to the data, including significant decrease in chi-square, reduction of chi-square to nonsignificant, and improvement in RMSEA, CFI, TLI, AIC, and BIC (see Table 2). Figure 2 presents significant paths in the final model. Child perception of guidance and control and child perception of negative parenting significantly predicted adherence (ps $< .05$).

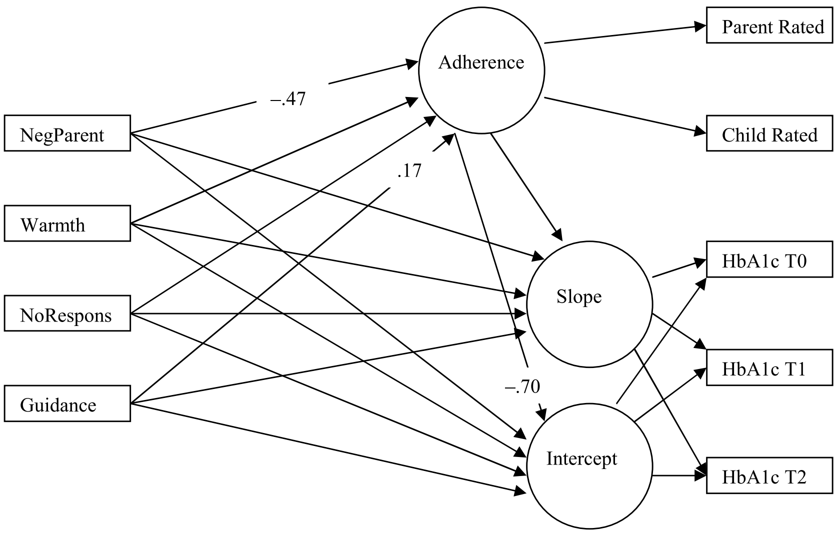

FIGURE 2 Model 3. *Note*. Age at baseline, duration of diabetes, and family structure were also included in Model 3, with relations as depicted in Models 1 and 2, but were omitted from graphic representation for parsimony. Significant paths are reported in standardized form. NegParent = child perception that parent is critical or negative; Warmth = child perception of parental warmth; NoRespons = parent and child disagreement about responsibility for the diabetes regimen; Guidance = child perception of parent guidance or control; HbA1c = glycosylated hemoglobin A1c test.

Child perception of parent warmth and parent–child disagreement about responsibility for the diabetes regimen did not predict adherence. Adherence significantly predicted the HbA1c intercept ($p < .01$), but did not predict the HbA1c slope. Family variables did not directly predict the HbA1c intercept or the HbA1c slope. Analysis of indirect effects revealed that child perception of negative parenting had a significant indirect effect on the HbA1c intercept ($r_{\text{unstandardized}} = .09$, $p < .01$), and that the combined effects of all family variables had a significant indirect effect on the HbA1c intercept ($r_{\text{unstandardized}} = .10$, $p < .05$). There were not significant indirect effects of any family variables on the HbA1c slope.

DISCUSSION

This study sought to determine whether measures of baseline family functioning indirectly affected initial status and rate of change in HbA1c via baseline adherence among youths with T1D. Examination of demographic variables revealed that one-parent households were related to worse parent- and child-rated adherence and to higher baseline and 2-year HbA1c, which is consistent with previously reported results (Hanson et al., 1988; Harris et al., 1999). This makes sense, as having one parent may be related to other variables, such as parenting stress, which could contribute to a coercive family process through parent behavior toward the child. However, contrary to findings of previous studies, our results demonstrated that family income was not related to HbA1c. Failure to find this relation may be a result of the relatively unique population from which the sample was drawn (i.e., a rural community), as data suggest that income in our sample was of average level and normally distributed (M = \$46K, SD = \$29K, Mdn = \$39K).

Hypothesized relations were examined using LGM. The overall model testing direct and indirect paths from family variables to initial status and rate of change in HbA1c demonstrated excellent fit. Consistent with our hypothesis, results indicated the combined effect of all family variables had a significant indirect effect on initial status of HbA1c. This appears to be driven by a significant individual effect of child perception of critical parenting on adherence. This is consistent with our hypothesis based on Patterson's (1982) coercion model, which would suggest that youths who perceive their parents as critical or negative may refuse to comply with adherence-related demands, resulting in higher HbA1c. Other family functioning variables (perception of parent warmth and disagreement about responsibility for diabetes regimen) did not have an individual effect on initial status of HbA1c, although the effect of child perception of parent guidance approached statistical significance. Given that these family variables have previously only been examined in cross-sectional studies, it may be that child perception of critical parenting is the most salient marker of a coercive cycle that could impact adherence behaviors over time.

Contrary to our expectations, family variables did not indirectly impact the rate of change in HbA1c. It is likely that the reason for this is that adherence was considered to be time-invariant, meaning it was only measured at baseline and could not change across time. As a potential time-varying covariate, meaning measured at multiple times, adherence may show a stronger relation with rate of change in HbA1c. Given that the biological assay measuring HbA1c tests glycemic control during the previous 2 to 3 months, it is reasonable to expect adherence to impact HbA1c greatest during that time, particularly given that adherence is known to fluctuate during adolescence (Kovacs et al., 1989). It should be noted that measurement of adherence at baseline only is not optimal for this reason, and future investigations should measure adherence at multiple times. In addition, although we hypothesized that family functioning would predict rate of change in HbA1c, the finding that all participants demonstrated similar rates of degradation highlights another important outcome. Our results demonstrated that children perceiving parents as critical had the highest initial HbA1c values. Having the same rate of change in HbA1c suggests that these children did not decrease HbA1c; and, therefore, their risk for high HbA1c remains elevated over time. Although the original hypothesis was not supported, finding that participants with high initial HbA1c did not recover glycemic control may indicate the need for intervention with families characterized by youths' perceptions of critical parenting. Future studies should continue to investigate these youths as a population that could be at risk.

It is important to note the limitations of this study. Although the study represents a significant contribution by virtue of testing relevant variables in one model that incorporates longitudinal HbA1c data, both family variables and adherence data were only measured at baseline, greatly limiting the causal inferences that can be drawn between family variables and adherence. Given that adherence fluctuates across childhood, it may be particularly important to examine this relation temporally (Kovacs et al., 1989). Analysis of adherence at multiple times would likely provide more useful guidance for intervention. Second, although effort was made to ensure participants that information was confidential and caregivers and children were interviewed alone, it is possible that responses were biased. In addition, the sample in this study was primarily Caucasian and rural, and results should be interpreted in light of this, as they may not generalize to all settings. Finally, the analyses conducted in this investigation treated observations as if they were from a single homogeneous population. Unobserved subpopulations may exist, and future research may focus on their identification and ability to predict glycemic control. Within these limitations, this investigation demonstrated the importance of baseline family variables on HbA1c through baseline adherence behaviors using a latent growth curve model.

This is the first study to test relevant variables in one model for youths with T1D. Based on the results of our study, future research should focus on further temporal elucidation of family variables and adherence and on continued intervention research targeting problematic family variables.

Implications for Practice

These findings represent the first investigation of family functioning, adherence, and longitudinal glycemic control, which incorporate variables into one model. Our results suggest that youths' perception of parents as critical and negative is related to difficulty with adherence and, further, that youths perceiving parents as critical and having poor adherence seem to start and maintain higher HbA1c without intervention. It may be that identifying these children at the level of the diabetes treatment team through careful evaluation of family variables is an important step in referring them for appropriate intervention and specialized care. However, additional research is needed to clarify the temporal relation among family functioning, adherence, and glycemic control before the mechanism for reduced glycemic control can be identified. Patterson's (1982) theory would suggest that targeting family communication skills and correcting child perception of critical parenting through breaking a coercive cycle may be important for improving adherence and glycemic control in these children. Relatedly, continued research investigating family interventions is an important step toward building the empirical knowledge necessary for treating these children. In particular, the recent investigation of behavioral family systems therapy has been shown to improve glycemic control, adherence, and family conflict (Wysocki et al., 2007; Wysocki et al., 2006; Wysocki, Harris, et al., 2008) through application of communication skills and family therapy within a diabetes-specific context. Other interventions, such as the Family Teamwork program, which emphasizes parent–child sharing of diabetes tasks and conflict avoidance, also demonstrate ability to increase glycemic control in youths (Anderson, Svoren, & Laffel, 2007). Future research is necessary to identify whether these treatments work by interrupting a coercive family cycle, and to determine whether children perceiving parents as critical truly represent a population at risk that should be referred for treatment.

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