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Metabolic Control and Academic Achievement over Time among Adolescents with Type 1 Diabetes

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Abstract

The relation between metabolic control (HbA1c) and achievement (grade point average [GPA]) was examined over a period of 2.5 years (every 6 months) employing a dynamical systems approach that allowed for the examination of whether HbA1c was associated with change in subsequent GPA and vice versa. Metabolic control tends to deteriorate (i.e., with higher HbA1c reflecting poorer metabolic control) during adolescence. It was hypothesized that these higher levels of HbA1c would limit subsequent increases in GPA. The sample included 252 adolescents $(M_{baseline age} = 12.49 \text{ years}, SD = 1.53; 53.6\% \text{ female})$ with type 1 diabetes. Mothers' report and school records provided information on relevant demographics and GPA; medical records provided values of HbA1c. Two simultaneous coupled change equations (i.e., examining current values in one variable associated with changes in the other) controlling relevant risk indicators (i.e., age, sex, disease duration, insulin delivery method, IQ) revealed higher levels of HbA1c limited increases in GPA. Higher levels of GPA, however, were not associated with change in HbA1c except for two instances where moderation existed by disease duration and IQ. Higher GPA was associated with slower increases in HbA1c over time for youth with shorter disease duration and lower IQ. These results affirm the importance of maintaining good metabolic control to facilitate adequate school performance across the adolescent years. Further, the results suggest that factors related to school achievement may protect adolescents who are newly diagnosed or who have low cognitive ability from subsequent deterioration in metabolic control.

Keywords

academic performance; adolescence; diabetes; HbA1c; metabolic control

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Type 1 diabetes is a common chronic health condition occurring in children and adolescents (National Diabetes Education Program [NDEP], 2010). Based on data from 2009, the prevalence of diabetes is 1 in 433 among school-aged youth within the U.S. (<20 years; Pettitt et al., 2014). Youth diagnosed with type 1 diabetes are at-risk for cognitive impairments, learning problems, and poor academic performance (Gaudieri, Chen, Greer, & Holmes, 2008; Kent, Chen, Kumar, & Holmes, 2010; Lin, Northam, Rankins, Werther, & Cameron, 2010; Naguib, Kulinskaya, Lomax, & Garralda, 2009), and score lower on achievement assessments than healthy peers (Gaudieri et al., 2008). Further, higher HbA1c (the chemical assay that reflects 2–3 months of glucose concentration in an individual's blood stream with higher scores reflecting poorer metabolic control; Chiang, Kirkman, Laffel, & Peters, 2014) is related to poorer academic achievement for youth with type 1 diabetes (Kaufman, Epport, Engilman, & Halvorson, 1999; McCarthy, Lindgren, Mengeling, Tsalikian, & Engvall, 2003; Parent, Wodrich, & Hasan, 2009). However, what is less clear from the current literature is whether metabolic control limits subsequent student academic achievement or whether deficits in academic achievement reflecting lesser scholastic competencies or contextual supports also limit subsequent diabetes metabolic control (Kaufman et al., 1999, McCarthy et al., 2003). In the current study, we examined coupled relationships between metabolic control and academic achievement to address the possible dynamic associations between metabolic control and academic achievement during the crucial adolescent years.

A meta-analysis conducted by Pinquart and Teubert (2011) involving 954 studies examining outcomes for children with chronic health conditions revealed a moderate effect size of -.35 (p < .001) representing the impact of diabetes on the academic functioning of youth. Moreover, Kaufman et al. (1999) reported significant moderate negative correlations between metabolic control (HbA1c) and scores from the Woodcock-Johnson Tests of Achievement for reading, writing, and mathematics that ranged from -.32 to -.33. Beyond performance on standardized assessments, full participation in classroom instruction can be inhibited due to daily fluctuations in glucose levels outside of the recommended range. These fluctuation in glucose levels lead to transitory impairments in attention (Parent et al., 2009) and reaction time (Gonder-Fredrick et al., 2009) along with a range of observable symptoms (e.g., irritability, difficulty concentrating, fatigue, disorientation) and the lifethreatening condition hypoglycemia that requires immediate intervention (NDEP, 2010). Taken together, there is support for the idea that poor metabolic control could limit academic achievement. Still, it has also been proposed that poorer academic achievement scores might signal difficulties in subsequent diabetes metabolic control (Kaufman et al., 1999, McCarthy et al., 2003). To illustrate, limited academic achievement and the scholastic competencies that are involved (problem solving, organizing, planning ahead) might limit an adolescent's ability to adequately complete the difficult treatment regimen required to maintain recommended levels of metabolic control (Kaufman et al., 1999, McCarthy et al., 2003).

At present, there appear to be inconsistent results between the findings of concurrent and longitudinal studies regarding the direction of influence between metabolic control and academic achievement. Concurrent studies have reported that higher HbA1c levels are associated with lower achievement for youth with type 1 diabetes and have largely employed cross-sectional (e.g., McCarthy et al., 2003) or case control designs (e.g., Kaufman et al.,

1999). In longitudinal studies, although progressive decline in achievement has been reported (Kovacs, Goldston, & Iyengar, 1992), metabolic control has not been identified as a meaningful predictor of subsequent learning or achievement (Fox, Chen, & Holmes, 2003; Kovacs et al., 1992). In contrast to conventional longitudinal analyses (e.g., repeated measure, multiple regression) previously used to examine this phenomenon, Kent et al. (2010) examined cognition amongst youth with type 1 diabetes employing Latent Growth Modeling (LGM; allowing one to capture individual differences in rates of change) and reported that as HbA1c trajectories increased over a 3-year period, visual memory scores correspondingly decreased. Such results are important as previous LGM studies have indicated metabolic control tends to deteriorate during adolescence for some youth with type 1 diabetes (Helgeson et al., 2010; King et al., 2012; Luyckx & Seiffge-Krenke, 2009; Miller et al., 2013; Rausch et al., 2012). Thus, given the propensity for HbA1c to increase during adolescence, a negative correlation between HbA1c and academic performance (Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009) might be indicative of adolescents with type 1 diabetes being at-risk for poor academic performance.

LGM might also present limitations in examining this association if the relation between HbA1c and achievement (or learning) is not a linear form of change across time (which might explain some of the incongruences in the findings of previous studies). For instance, a marker of academic performance such as grade point average (GPA) might increase or decrease from semester to semester for students dependent upon various contextual factors. Accordingly, examining emergent temporal patterns of how variables interact with one another in response to multiple contextual variables might be a more representative model of change over time for these variables (Butner, Berg, Baucom, & Wiebe, 2014; Butner, Gagnon, Geuss, Lessard, & Story, 2014). As the dynamic associations of change between our variables of interest might not be linear, employing a dynamical systems framework will allow the examination of change over time that does not require an assumption of linear change.

A dynamical systems approach allows for the examination of how change in both metabolic control and academic achievement affects the other as they change through time (Butner, Berg, et al., 2014; Butner, Gagnon, et al., 2014). This coordination or movement through time together is referred to as coupling. Systems models assume a multidirectional causal relationship, but coupling can be used to examine for asymmetries in the predictive nature of variables interacting through time. Coupling relationships in the present paper are referred to as the relationships between GPA and subsequent changes in HbA1c and vice versa. A dynamical systems approach allows the user to observe perturbations of variables on one another observed in this model along with perturbations influencing the system that are not modeled. In essence, the advantage of a dynamical systems approach is that coupling allows the observation of change over time in the presence of constant perturbations due to multiple contextual factors that might influence the system. In short, a dynamical systems approach allows for an examination of the coordination between academic achievement and metabolic control as they affect each other through time. Importantly, within the context of this paper, as causality is multidirectional, it cannot be tested directly. In the present study, we examined simultaneously how metabolic control may predict changes in subsequent

In addition, such coupling relationships could be moderated through contextual variables relevant to academic achievement and metabolic control. Covariates and moderators might strengthen or even uncouple the association between two variables as they change through time. Factors regarding the disease progression, such as disease duration might affect the coupling relationship. Disease duration is associated with poorer metabolic control (Hilliard, Wu, Rausch, Dolan, & Hood, 2013) as well as poorer achievement (Fox et al., 2003; Kovacs et al., 1992). We also examined other contextually relevant variables (e.g., age, insulin delivery system, sex) that might also moderate the dynamic relation or function of covariates between metabolic control and academic performance.

Purpose of the Current Study

The purpose of this study was to examine the dynamic relation between metabolic control fluctuation and academic performance of adolescents diagnosed with type 1 diabetes over a 2.5-year period. Specifically, a model of two simultaneously coupled change equations was employed to examine the dynamic associations between adolescents' (aged 10–14 years of age at study initiation) metabolic control and academic achievement sampled every six months. Overall, poor metabolic control was expected to be a limiting factor for academic performance over time and GPA (a multiply determined variable possibly reflecting overall cognitive ability, self-regulatory capacity, and parental involvement in addition to academic performance) was expected to limit deterioration in metabolic control during adolescence.

Methods

Participants

Participants included 252 adolescents diagnosed with type 1 diabetes ($M_{baseline age} = 12.49$ years, SD = 1.53; 53.6% female) recruited from a university outpatient diabetes clinic and a second private practice clinic conducted by a pediatric endocrinologist following similar treatment procedures as part of a larger longitudinal study of adolescent diabetes management.. Individuals were recruited by a research assistant at their clinic appointment. Criteria for eligibility included youth that were between 10-14 years of age at baseline, literate in either English or Spanish, and were diagnosed with type 1 diabetes for 1 year or more. Sixty-six percent of participants approached that met inclusion criteria agreed to participate. The following reasons were cited for not participating: not interested (30%), too busy (21%), distance (18%), uncomfortable with being studied (14%), and time commitments (5%). Rates of participants skipping data collection time points or dropping out of the study follow: Time 2 (20 skipped, 17 dropped), Time 3 (28 skipped, 11 dropped), Time 4 (30 skipped, 11 dropped), Time 5 (18 skipped, 9 dropped), and Time 6 (5 skipped, 1 dropped). Overall, an 81% retention rate (N = 203) for the study was maintained. At baseline, parents' report indicated that participants had been diagnosed with type 1 diabetes for an average of 4.74 years (SD = 2.96). Approximately half of participants (50.8%) were on Continuous Subcutaneous Insulin Infusion (CSII; i.e., insulin pump therapy) at baseline. In regard to ethnicity, 90.9% of mothers identified youth as non-Hispanic White.

Additionally, 63.5% of mothers reported annual household income > \$50,000 and 81% reported some college education or higher.

Measures

HbA1C—HbA1c was used to determine levels of average glucose concentration in the blood stream over a period of 2–3 months (Chiang et al., 2014; Centers for Disease Control, 2008). HbA1c levels less than 7.5% are recommended for all youth with diabetes (Chiang et al., 2014). HbA1c was assessed every 6 months for each participant by clinic staff employing the Bayer DCA 2000. The mean values of HbA1c at baseline ($M_{baselineHbA1c} = 8.37$), final assessment ($M_{finalHbA1c} = 8.84$), and across all time points for this sample ($M_{HbA1c} = 8.65$) were above the American Diabetes Association's (ADA; Chiang et al., 2014) recommended threshold for ideal metabolic control.

GPA—In the current study, participants' most recent GPA score was reported by participants' mothers during each 6-month clinic visit. At Time 6, mothers' reports of GPA correlated highly with GPA in adolescents' school records (r =.84, p < .001), lending validity to mothers' reports of GPA. In the current study, GPA at Time 6 was taken from the student's school record. At Time 6, mothers tended to overestimate GPA scores (3.43, SD = . 57) on average compared to mean GPA pulled from the school records (3.34, SD = .69). The mean values of GPA at baseline, final assessment, and across all time points for this sample were 3.40, 3.42, and 3.42, respectively.

Covariates and moderators—The rationale for selecting covariates and moderators was based upon prior research and the demographics of the current sample. Covariates and moderators assessed at baseline that were considered to be meaningful predictors of achievement, learning, or memory included youth's IQ (Hannonen et al., 2012; Kovacs et al., 1992), duration of diabetes (Fox et al., 2003; Gaudieri et al., 2008; Kovacs et al., 1992;), age (Kent et al., 2010), and sex (Fox et al., 2003; Kent et al., 2010). Baseline predictors of metabolic control included youth's cognitive ability score (Kaufman et al., 1999; McCarthy et al., 2003) represented by scores from the Kaufman Brief Intelligence Test, Second Edition (Kaufman & Kaufman, 2004), insulin delivery method (e.g., insulin pump; Hilliard et al., 2013; Rausch et al., 2012), age and disease duration (Hilliard et al., 2013), and sex (Luyckx & Seiffge-Krenke, 2009).

Kaufman Brief Intelligence Test, Second Edition (KBIT-2)—Teens completed the Kaufman Brief Intelligence Test, Second Edition (Kaufman & Kaufman, 2004), which consists of two subtests, Vocabulary and Matrices, and provides a composite IQ score (Bain & Jaspers, 2010) that was used in the present study. The mean for the test-retest reliability coefficient was .90 and the mean for the internal consistency coefficient was .93 for the KBIT-2 IQ Composite (Bain & Jaspers, 2010) indicating high reliability. Mean cognitive scores for this sample of youth with type 1 diabetes fell within the average range.

Data Analysis

Missing data—A missing value analysis (MVA) revealed >10% missing data for key outcome variables (GPA Times 1–6, HbA1c Times 4–6)¹. Given the number of participants

lost to follow-up across the study (49), three values are reported to examine the issue of missing data across time points. The first value represents the percentage of total missing data (dropped, skipped, and missing) per round for each key variable. The second value represents the frequency of participants that dropped out of the study (summing across previous and current rounds) whereas the third value represents the total number of missing data points per variable for that round. The percentages and frequencies of missing data for key variables follow: GPA Time 1 (32%, 0/81), GPA Time 2 (41%, 17/103), GPA Time 3 (35%, 28/89), GPA Time 4 (42%, 39/107), GPA Time 5 (37%, 48/92), GPA Time 6 (36%, 49/91), HbA1c Time 4 (18%, 39/45), HbA1c Time 5 (21%, 48/54), and HbA1c Time 6 (23%, 49/57). The MVA also revealed that although the overall total of missing data values totaled 19%, the use of listwise deletion would result in retention of only 55 (22%) cases.

totaled 19%, the use of listwise deletion would result in retention of only 55 (22%) cases. Multilevel modeling utilizes Maximum Likelihood (ML) Estimation, which allows for the inclusion of cases with missing data under the assumption of data missing at random and normality (Baraldi & Enders, 2010; Graham, 2009; Schafer & Graham, 2002). In addition, we used Restricted ML Estimation (REML), which tends to perform well in small and moderate sample sizes by restricting ML estimation to random effects.

Data preparation—Univariate analyses did not reveal extreme absolute values of skewness (< 3.0) or kurtosis (< 8.0; Kline, 2005). Nonetheless, univariate skewness and kurtosis values indicated moderate nonnormality. Correlations, means, standard deviation values, and skewness and kurtosis values are presented in Table 1. Mardia's test of multivariate kurtosis (DeCarlo, 1997) revealed some degree of non-normal data distribution; however, multilevel modeling is robust to reasonable amounts of non-normality since the assumptions are more related to the residuals than the observed variables.

Analytic strategy—The analyses were conducted using IBM SPSS 19 Mixed Model employing REML. For each time series we separately created difference scores of consecutive time points of HbA1c and GPA by calculating the future value (t+1) minus the current value (t). These were treated as simultaneous dependent variables predicted by current HbA1c and GPA alone. Such models are comparable to the actor-partner multilevel model (Raudenbush, Breennan, & Barnett, 1995) where one partner (or in this case one variable GPA or HbA1c) is affecting the other and vice versa. As such, the multivariate multilevel model is to be completed through a single model as described in Butner, Berg, et al., (2014) and Butner, Gagnon, et al., (2014). To be clear, the first level of the general model examined the general pattern of association between change in HbA1c and GPA over time, whereas the impact of risk-related variables on the system was examined at level 2. Age, disease duration, KBIT-2, pump status, and sex were included as covariates. Level 1 equations examined how one variable predicted the other variable's change in a dynamic coupling relationship translating into how HbA1c and GPA were predicting change in each other through time. Finally, we added interaction terms between the coupling relationships and the various risk indicators (i.e., age, disease duration, KBIT-2, pump status, sex) to see which of these terms acted as moderators between the linkages between HbA1c and GPA.

¹HbA1c Times 1–3 had 10% missing data.

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To account for the simultaneous equations, each change was allowed to have a unique error variance and a shared error covariance.

The two simultaneous Level 1 equations follow below:

Level 1:

$$(A1C_{t+1} - A1C_t)_i = \beta_{0i} + \beta_{1i} * A1C_{ti} + \beta_{2i} * GPA_{ti} + e_{ti} (GPA_{t+1} - GPA_t)_i = \beta_{3i} + \beta_{4i} * GPA_{ti} + \beta_{5i} * A1C_{ti} + e_{ti}$$

At Level 2, all equations included a Level 2 intercept. Only the Level 1 intercepts (β_{0i}, β_{3i}) and coupling relationships (β_{2i}, β_{5i}) included additional predictors. The Level 2 equations follow below:

Level 2:

In the final two equations, we systematically replaced the moderator_i term with each risk factor (age, disease duration, KBIT-2, pump status, sex) one at a time to test which if any moderated the coupling relationships. Although each moderator was tested individually, we cannot explicitly compare one moderated model to another as the models are not nested. Nonetheless, a significant moderator signals which factors should be considered meaningful.

Random effects were excluded at Level 2 from the model because the sheer number of random effects generated through these simultaneous equations would have been too demanding for the six time points (or five differences). Notably, both age and GPA were grand centered, and KBIT-2 was centered at 100 – representing the score average for the population. Moreover, scaling of the other variables included dummy coding for pump status and sex, while age and disease duration remained continuous.

Results

Coupling of HbA1c and GPA

A model treating the risk factors only as covariates (no moderators) generated the average pattern. This model included 18 parameters. All coupling results were interpreted in relation to the average positive trends in GPA and HbA1c for this sample. On average, a coupling relationship was observed before the moderators were added. HbA1c predicted change in GPA (coupling term = -.035, SE = .012, df = 540.774, t = -2.894, p = .004, 95% CI [-.058, -.011]), yet GPA did not predict the change in HbA1c (coupling term = -.103, SE = .089, df = 651.257, t = -1.160, p = .246, 95% CI [-.278, .072]). Specifically, higher levels of HbA1c were associated with smaller subsequent increases in GPA. However, higher GPA was not in general associated with subsequent change in HbA1c.

Moderated coupling

Table 2 contains all of the estimated moderation coefficients and their 95% confidence intervals. There was no moderation of how HbA1c predicted change in GPA. However, both disease duration and KBIT-2 moderated how GPA predicted change in HbA1c. For disease duration (M = 4.7 years), those who had the disease for a briefer period of time or one standard deviation (SD = 3 years) below average (1.7 years; coupling term = -.281, df = 651.513, t = -2.537, p = .001), showed stronger coupling such that higher GPA was related to a slower increase in HbA1c. In contrast, coupling became nonsignificant for participants having had the disease for the group average of 4.7 years (coupling term = -.101, df = 650.221, t = -1.137, p = .256) or one *SD* above the average (7.7 years, coupling term = . 079, df = 651.512, t = .709, p = .479). In short, coupling remained significant only for adolescents with disease duration of 1.7 years or less suggesting that higher GPA scores attenuated increases in HbA1c levels for adolescents with average or above average disease durations of the disease. Conversely, HbA1c levels for adolescents with average or above average disease duration periods did not evidence any coupling between GPA and HbA1c.

For KBIT-2, adolescents with a lower IQ score showed stronger coupling such that higher GPA was related to a slower HbA1c increase. For adolescents with an IQ score on the KBIT-2 one *SD* below average (85; coupling term = -.287, df = 652.380, t = -2.257, p = . 024), coupling remained significant, but not when average (100; coupling term = -.125, df = 650.366, t = -1.393, p = .164) or one *SD* above average (115; coupling term = .038, df = 652.173, t = .334, p = .739). Overall, coupling remained significant only for adolescents with KBIT-2 scores one *SD* below average or lower. Further, age, pump status, and sex did not moderate the coupling relationship in either case.

Discussion

Two simultaneous coupled change equations were conducted to examine how poor metabolic control (i.e., higher HbA1c) may limit changes in GPA over time as well as whether GPA limited changes in HbA1c. On average, HbA1c was a limiting factor for GPA while controlling for age, disease duration, KBIT-2 score, pump status, and sex, at study entry, whereas GPA was not associated with changes in HbA1c. The primary results of this study align with previous findings suggesting youth with type 1 diabetes with poor metabolic control (higher blood glucose levels) are at-risk for academic performance or learning difficulties (Kaufman et al., 1999; Kent et al., 2010; McCarthy et al., 2003; Parent et al., 2009). A possible mechanism for this association is the impact of poor metabolic control on both long- (Kent et al., 2010; Lin et al., 2010) and short-term cognitive functioning (Gonder-Fredrick et al., 2009). Long-term cognitive impairment is likely the result of microvascular complications stemming from the toxic effects of chronic hyperglycemia (high blood glucose levels) on the brain and might even lead to structural abnormalities during critical stages of brain development (Biessels, Deary, & Ryan., 2008; Ryan, 2006). As the brain relies on glucose as its sole energy source, short-term or transitory cognitive impairment can result from rapid fluctuations in blood glucose levels which also impact skills required for academic performance and learning such as reaction time, accuracy, and attention (Gonder-Fredrick et al., 2009; Parent et al., 2009) not to mention

behavior (NDEP, 2010). Unfortunately this mechanism could not be examined in the present study given that cognitive function was only measured at baseline.

Results provided through a dynamical systems approach were supportive of previous work evidencing a limiting effect of poor metabolic control on academic achievement and learning in studies employing concurrent (Kaufman et al., 1999; McCarthy et al., 2003; Parent et al., 2009) and prospective analyses (Kent et al., 2010). A dynamical systems framework revealed the emergence of a coordinated pattern reflective of the consistency of the covariation of the variables through time (Butner, Berg, et al., 2014). In contrast, previous studies employing analyses that required an assumption of linear change (e.g., LGM) might not have been able to reveal the dynamic association between metabolic control and achievement as each perturbs the value of the other through time.

Moreover, GPA scores on average did not predict change in HbA1c while controlling for age, disease duration, KBIT-2 score, pump status, and sex. A moderation effect was evidenced, however, for youth earlier in the disease or with lower KBIT-2 scores. For adolescents with shorter disease duration or lower cognitive ability, higher GPA scores appeared to attenuate increases in HbA1c levels. GPA is a variable that is multiply determined including factors of overall cognitive ability, self-regulatory capacity, and parental involvement (Duckworth, Quinn, & Tsukayama, 2012; Duckworth & Seligman, 2005, Tangney, Baumeister, & Boone, 2004; Wolfe & Johnson, 1995), and such factors could moderate associations between GPA and subsequent metabolic control.

As duration increased for this sample, coupling for this moderation effect went to zero. Adolescents with higher GPA who have been recently diagnosed may be able to apply the skills gained in their academic work to the context of diabetes management (e.g., breaking a task down into lower order goals, good problem solving, organizing, planning ahead). In this case, GPA might represent an indicator of self-regulatory capacities (Duckworth et al., 2012; Duckworth & Seligman, 2005, Tangney et al., 2004; Wolfe & Johnson, 1995) serving as a protective factor for adolescents who have been newly diagnosed when managing the disease. In contrast, adolescents with a prolonged course of the disease might evidence impairments in self-regulatory capacities likely necessary for achieving adequate metabolic control. At the same time, poorer academic achievement might also reflect the degree of parental involvement across contexts or even neurocognitive insult related to complications of diabetes that might make treatment adherence all the more difficult (Kaufman et al., 1999, McCarthy et al., 2003).

Further, higher GPA values muted increases in HbA1c for adolescents with IQ scores one *SD* below average or lower, whereas adolescents with average or one *SD* above average KBIT-2 scores did not experience any attenuation in HbA1c increases over time. Those adolescents who have a high GPA with lower intelligence may have parents highly involved in the management of their care (Kaufman et al., 1999). Parents who are more involved in their schoolwork (and by extension their diabetes as well; Kaufman et al., 1999), may help compensate for the adolescent's lower intelligence. Parental involvement (Berg et al., 2008, 2011; Palmer et al., 2011) is associated with adherence and metabolic control and might allow an adolescent with a low KBIT-2 score to still achieve adequate metabolic control.

The results should be interpreted within the context of some limitations. First, GPA was measured with mothers' reports of GPA, which were unstandardized. Although mothers' reports were highly correlated with verified report cards at Time 6, future research should include GPA from reports cards. However, even report card GPAs are unstandardized and depend upon the school or teacher that assigned the score. As such, the use of a brief reading or mathematics subtest from a standardized achievement assessment such as the Woodcock-Johnson Tests of Achievement (Woodcock, McGrew, & Mather, 2001) might be a reasonable alternative for future studies. Likewise, the use of a brief standardized cognitive assessment at every sampling round in future studies might provide more insight into the deleterious impact of metabolic deterioration on cognition and subsequent achievement. Second, the impact of inconsistent academic performance might not be reflected in summative or global GPA scores. The benefit of instruments designed for repeated measure that are sensitive to change over time such as Curriculum-Based Measures might also be considered for future studies. Third, from a demographic perspective, the sample was relatively homogenous limiting the generalizability of the results. Results from this study evidenced deterioration in metabolic control as a limiting factor for achievement in this sample comprised of mostly white, higher SES, high achieving students, with average cognitive ability levels. Indeed, this vulnerability might potentially be even more detrimental for youth with type 1 diabetes with additional risk factors (e.g., impoverished families, already low achieving students). Moreover, future studies might want to determine whether these findings can be replicated in more diverse samples. Lastly, both HbA1c and GPA require a high level of investment and as such may be influenced both by a broad array of intrapersonal (self-regulation, motivation) as well as interpersonal factors (parental involvement). Accordingly, the association between GPA and HbA1c might also reflect an association with some third variable or set of variables (i.e., self-regulation, resources available to the student such as parent involvement) that is influencing the system. Additional research is needed to understand how these factors affect the HbA1c and GPA links.

Clinical and School-Based Implications

The results of this study revealed that poor metabolic control may be a limiting factor in student academic achievement and point toward the importance of maintaining good diabetes management for school success. To support diabetes management within the school context, school psychologists have the training and capacity to coordinate and consult with educational and health professionals and to provide behaviorally focused interventions targeting treatment adherence in conjunction with appropriate therapeutic interventions (Kucera & Sullivan, 2011; Schmitt, Wodrich, & Lazar; 2010; Power, DuPaul, Shapiro, & Parrish, 1995). Evidence-based interventions such as behavioral strategies including positive reinforcement may be used to help youth adhere to a treatment protocol and engage in more adaptive behaviors (Stoeckel & Duke, 2015; Wysocki, 2006) while at school. The importance of the role of the school psychologist in supporting the well-being of youth with chronic health conditions cannot be overstated as the interaction between educational and health professionals continues to increase due to the growing presence of integrated care within the school context (Power et al., 1995).

To support good metabolic control, educators, administrators, and health care professionals should work toward facilitating the successful implementation of an intensive treatment regimen as defined by the ADA (2012; Silverstein et al., 2005). Both the ADA (2012) and JDRF (2013) recommend a written care plan including an emergency plan be developed with the Local Education Agency for all youth with diabetes and enforced under a relevant federal law such as Section 504 of the Rehabilitation Act of 1973 or the Individuals with Disabilities Education Improvement Act (2004). Written care plans identify goals and define measurable objectives for diabetes care in school including ensuring the youth's safety, immediate and long-term health, optimal academic performance, and full participation in school related activities (NDEP, 2010). As the appropriateness of selecting a 504 Plan or an Individualized Education Program can vary on a case-to-case basis and is beyond the scope of this article, please see Schmitt et al. (2010) for an in depth examination on the topic. Although health care professionals will generate the medical components of a youth's written care plan, school psychologists will be truly essential in identifying the most appropriate federal law to ensure the enforcement of the written care plan along with any behavioral or therapeutic treatment plans.

Involvement of caregivers is also important in supporting treatment adherence and metabolic control (Berg et al., 2008, 2011; Palmer et al., 2011). Nevertheless, most parents are unaware of relevant federal laws related to diabetes care in school and nearly half of students with diabetes do not have written medical care plans (Jacquez et al., 2008). Additionally, parental reports suggest most school personnel do not have adequate training in diabetes management (Wagner, Heapy, James, & Abbott, 2006). This is important as the omission of even a single daily blood glucose test per day was predictive of an increase in HbAlc levels of 1.26% in a 2-year longitudinal study of adolescents (Rausch et al., 2012). Again, advocacy and facilitation between educational and health professionals in supporting youth with diabetes is clearly needed (Kucera & Sullivan, 2011; Schmitt et al.; 2010; Power et al., 1995).

Further, as higher GPA scores appeared to slow the deterioration of metabolic control for youth with a shorter duration of the disease, evidence-based interventions for increasing skills associated with GPA appear warranted. Self-control has been evidenced as an influential factor in student achievement (Duckworth et al., 2012; Duckworth & Seligman, 2005, Tangney et al., 2004; Wolfe & Johnson, 1995). As such, evidence-based interventions focusing on increasing self-regulatory capacity appear appropriate and are recommended to improve academic achievement (Duckworth & Seligman, 2005) as well as adherence to diabetes treatment plans (Berg et al., 2014; Hughes, Berg, & Wiebe, 2012; Lansing & Berg, 2014).

Conclusion

Metabolic control tends to deteriorate for adolescents with type 1 diabetes (Helgeson et al., 2010; King et al., 2012; Miller et al., 2013; Rausch et al., 2012) placing youth at risk for academic problems (Kaufman et al., 1999; Kent et al., 2010; McCarthy et al., 2003; Parent et al., 2009). Our results revealed that increasing HbA1c during adolescence was a limiting factor for achievement over a 2.5-year period. GPA scores, however, did not attenuate the rise of HbA1c levels save for two instances of moderation. Youth with shorter disease

duration or lower IQ experienced slower increases in HbA1c over time if higher GPA scores were evidenced. Factors associated with higher GPA scores may protect recently diagnosed adolescents or adolescents with low cognitive ability from later deterioration in metabolic control. Thus, the efforts of school psychologists to facilitate good diabetes management within the school context affects not only the immediate health and safety, but also the long-term health and academic achievement of this vulnerable population.

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Table 1

Univariate Correlations, Means, and Standard Deviations for a Multilevel Model of HbA1c (A1C) Values and Grade Point Average (GPA) over 2.5 Years

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Variables	1	2	3	4	5	9	7	8	6	10	11	12	13	14	15	16	17
1. T1_A1C	I																
2. T2_A1C	.74 **	I															
3. T3_A1C	.71 **	.64	I														
4. T4_A1C	.58**	.62**	.64 **	I													
5. T5_A1C	.52 **	.52 **	.59 **	.60 ^{**}	I												
6. T6_A1C	.47 **	.35 **	.55 **	.45 **	.62	I											
7. T1_GPA	43 **		17*	22 **	30 **	22 **	I										
8. T2_GPA	35 **		12	37 **	29 **	23 **	.81 **	I									
9. T3_GPA	42 **	32 **	30 **		23 **	22 **	** TT.	.73 **	I								
10. T4_GPA	46 **	44 **	25 **	36 **	28 **	18*	.73 **	.84	.89 **	I							
11. T5_GPA	44 **			35 **	28	25 **	.64	.65 **	.63 **	.76**	I						
12. T6_GPA	44 **		30 **	40	32 **	38	.64	.55 **	.62	.70**	.64	I					
13. Duration	.12	60.	.10	.03	02	03	02	.07	02	90.	90.	.12	I				
14. Sex	06	.01	00	03	.01	02	.21 **	.24 **	.23 **	.29 **	.17*	.13	04	I			
15. No Pump	.28**	.25 **	.17*	.24 **	.18*	.18*	20 **	15	24 **	17*	15	18^{*}	14 *	06	I		
16. Age	.15*	60.	90.	.05	.06	.02	30 **	24 **	32 **	26 **	22 ^{**}	15	.07	10	.01	I	
17. IQ_KBIT	26 **	25 **	17*	25 **	25 **	19**	.40 **	.37 **	.22	.28 **	.27 **	.37 **	05	06	03	04	I
Mean ^b	8.37	8.45	8.60	8.71	8.92	8.84	3.40	3.47	3.33	3.41	3.46	3.43	4.74	.54	.48	12.49	103.54
SD	1.58	1.54	1.75	1.72	1.64	1.59	.62	.59	.70	.67	.62	.57	2.96	.50	.50	1.53	13.36
Skewness	1.22	1.31	1.53	1.49	1.17	1.53	-1.41	-1.77	-1.73	-1.98	-1.91	-1.52	99.	16	.05	11	05
Kurtosis	2.24	2.59	2.45	2.04	1.29	2.68	1.91	3.81	3.98	4.90	3.96	2.62	63	-1.99	-2.01	-1.26	07

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Im Likelihood (REML) Moderated Multilevel Model of Changes in HbA1c (A1C; Dependent Variable [DV]; Intercept) Values and	ge (GPA; Dependent Variable [DV]. Intercept) over 2.5 Years for Adolescents with Tvpe 1 Diabetes
Restricted Maximum Likelihood (1	Grade Point Average (GPA: Depen

Lower Lower Upper $.06(.02)^{**}$ $.02$ $.11$ $.06(.02)^{**}$ $.02$ $.11$ $.13(.16)$ 20 $.45$ $.13(.16)$ 20 $.45$ $.13(.16)$ 20 $.45$ $.13(.16)$ 20 $.45$ $.13(.16)$ 20 $.45$ $.08(.06)$ 03 $.20$ $.08(.06)$ 03 $.20$ $.08(.06)$ 03 $.00$ $.08(.06)$ 03 $.02$ $.01(.01)^{*}$ $.00$ $.02$ $.01(.01)^{*}$ $.00$ $.02$ $.02(.02)$ $.02$ $.05$ $.02(.02)$ 02 $.05$ $.06(.10)$ 15 $.26$ $.06(.10)^{*}$ $.06$ $.46$ $.05(.03)$ 12 $.02$ $.05(.03)$ 12 $.02$	INTONCI GLOI	DV Change in HbA1c (SE)	95% Confidence Interval	ence Interval	DV Change in GPA (SE)	95% Confid	95% Confidence Interval
$ \begin{array}{l lllllllllllllllllllllllllllllllllll$			Lower	Upper		Lower	Upper
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	Duration	.06 (.02) **	.02	.11	(00.) 00.	01	.01
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $	Sex	.13 (.16)	20	.45	.04 (.02)	01	.08
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	No Pump	18 (.16)	50	.14	.03 (.02)	02	.07
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Age	.08 (.06)	03	.20	.00 (.01)	02	.02
$ \begin{array}{ c c c c c c } \hline \mbox{Moderation at HbA1c Intercept (SE)} \\ \hline \mbox{Moderation at HbA1c Intercept (SE)} \\ \hline \mbox{.02 (02)} &02 & 05 & .01 (.01) & .00 \\ \hline \mbox{.06 (.10)} &15 & .26 & .07 (.03) & .01 \\ \hline \mbox{.06 (.10)} &12 & .06 & .04 (.03) &11 \\ \hline \mbox{.07 (.03)} &01 & .00 & .00 (.01) & ** &06 \\ \hline \mbox{.01 (.00)} &01 & .00 & .00 (.00) & ** & .00 \\ \hline \mbox{.00 (.00)} & ** & .00 \\ \hline \end{array} $	IQ_KBIT	.01 (.01) *	00	.02	.00(00)	00.	00.
.02 (.02) 02 .05 $.01 (.01)^*$.00.06 (.10) 15 .26 $.07 (.03)^*$.01.26 (.10)^*.06.46 $04 (.03)$ 11 $05 (.03)$ 12 .02 $04 (.01)^{**}$ 06 $01 (.00)$ 01 .00.00 $.00^{.00}^{**}$.00	Moderator	Moderation at HbA1c Intercept (SE)			Moderation at GPA Intercept (SE)		
.06 (.10) 15 .26 $.07 (.03)^*$.01.26 (.10)^*.06.46 $04 (.03)$ 11 $05 (.03)$ 12 .02 $04 (.01)^{**}$ 06 $01 (.00)$ 01 .00.00 $.00 (.00)^{**}$.00	Duration	.02 (.02)	02	.05	.01 (.01)*	.00	.02
$.26(.10)^*$ $.06$ $.46$ $04(.03)$ 11 $05(.03)$ 12 $.02$ $04(.01)^{**}$ 06 $01(.00)$ 01 $.00$ $.00(.00)^{**}$ $.00$	Sex	.06 (.10)	15	.26	.07 (.03)*	.01	.14
05 (.03) 12 $.02$ 04 (.01) ** 06 01 (.00) 01 $.00$ $.00$ (.00) ** $.00$	No Pump	.26 (.10) *	.06	.46	04 (.03)	11	.02
01 (.00) 01 .00 $.00$ $.00$ $.00$ $.00$	Age	05 (.03)	12	.02	04 (.01)**	06	02
	IQ_KBIT	01 (.00)	01	00.	$.00(.00)^{**}$	00.	.01
	$_{p < .05, *}^{*}$						
p < .05,	** n / 01						