Anxiety Symptoms in Adolescents with Type 1 Diabetes: Association with Blood Glucose Monitoring and Glycemic Control

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Objective To examine the prevalence of anxiety symptoms and their association with blood glucose monitoring (BGM) and glycemic control in adolescents with type 1 diabetes. Methods 276 adolescents and their caregivers completed measures of anxiety symptoms. Adolescents completed a measure of depressive symptoms. Demographic and family characteristics were obtained from caregiver report. Diabetes duration, regimen type, BGM frequency, and glycemic control were also collected. Results Trait anxiety symptoms that suggest further clinical assessment is needed were present in 17% of adolescents; the rate was 13% for state anxiety symptoms. Higher levels of state anxiety symptoms were associated with less frequent BGM and suboptimal glycemic control, $F(14, 261) = 6.35, p < .0001$, $R^2 = .25$, and suboptimal glycemic control, $F(15, 260) = 7.97, p < .0001$, $R^2 = .32$. State anxiety symptoms were correlates of BGM frequency and glycemic control independent of depressive symptoms. Conclusions State anxiety symptoms are associated with less frequent BGM and suboptimal glycemic control in adolescents with type 1 diabetes.

Key words adolescents; anxiety; type 1 diabetes; glycemic control.

Adolescents with type 1 diabetes are at increased risk of problematic psychological functioning. Much of the work in this area has focused on depression (Grey, Whittemore, & Tamborlane, 2002; Hood et al., 2006; Kovacs, Obrosky, Goldston, & Drash, 1997; Whittemore et al., 2002) and highlights that up to 20% of adolescents with type 1 diabetes experience elevated levels of depressive symptoms; a rate two to three times that found in the general adolescent population (Lewinsohn, Clarke, Seeley, & Rohde, 1994). Depressive symptoms have been linked to poorer disease management and glycemic control in adolescents with type 1 diabetes (Helgeson, Siminerio, Escobar, & Becker, 2008; La Greca, Swales, Klemp, & Madigan, 1995; Whittemore et al., 2002), generating concern for multidisciplinary teams providing care to these adolescents. While attention to depression in adolescents with type 1 diabetes is important, little research has focused on other areas of psychological functioning, particularly anxiety. The few studies that have examined anxiety have done so in younger children with diabetes (Grey, Cameron, Lipman, & Thurber, 1995; Kovacs, Goldston, Obrosky, & Bonar, 1997) and suggest lower rates of anxiety than depression. However, literature that suggests that rates of anxiety in the general adolescent population are often higher than rates of depression (Angold & Costello, 2001; Axelson & Birmaher, 2001; Brady & Kendall, 1992; Dierker et al., 2001) is concerning.

Prior research on the relationship between anxiety and disease management among children and adolescents with type 1 diabetes has not resulted in a clear understanding of the nature of this relationship. For example, several studies show that worries about diabetes and negative affect (e.g., trait anxiety) may negatively impact disease management and glycemic control (Mortensen, 2002; Naar-King et al., 2006; Wiebe, Alderfer, Palmer, Lindsay, & Jarrett, 1994). Research on parental anxiety has similarly shown associations with suboptimal glycemic control and...
motivation for diabetes self-care among adolescents with type 1 diabetes (Cameron, Young, & Wiebe, 2007). On the other hand, extant literature on fear of hypoglycemia suggests that a history of hypoglycemic episodes and fear of future episodes can contribute to heightened levels of stress and anxiety (Gonder-Frederick et al., 2006; Kamps, Roberts, & Varela, 2005). Regardless of the direction of the relationship, there are links between the presence of anxiety symptoms and the well-documented difficulties adolescents with type 1 diabetes experience in poor adherence (Weissberg-Benchell et al., 1995) and suboptimal glycemic control (Danne et al., 2001; Springer et al., 2006). Further, it is likely that these difficulties persist into adulthood as anxiety symptomatology in adults with type 1 diabetes is associated with less frequent blood glucose monitoring (BGM) and suboptimal glycemic control (Lloyd, Dyer, & Barnett, 2000; Mollema, Snoek, Ader, Heine, & van der Ploeg, 2001; Shaban, Fosbury, Kerr, & Cavan, 2006).

The relative lack of studies on anxiety in adolescents with type 1 diabetes also raises questions about whether the associations between anxiety symptoms, diabetes management, and glycemic control are similar to what has been documented between those variables and depressive symptoms. Studies on depression consistently show that depressive symptoms interfere with disease management and glycemic control rather than promote it (Grey et al., 2002; Kovacs Goldston, et al., 1997; McGrady, Laffel, Drotar, Repaske, & Hood, 2009); however, the role of anxiety is equivocal. In general, it has been suggested that at moderate levels, anxiety may act as a protective factor (e.g., Yerkes–Dodson Law: Yerkes & Dodson, 1908), and that if anxiety levels are either too high or too low, memory, attention, well-being, and performance can be compromised (Dellenbacher, 1994; Hanoch & Vitouch, 2004). Anxiety has been examined in the context of surveillance for early cancer detection, particularly adult breast cancer, with findings showing that elevated anxiety may promote self-exams and compliance with early surveillance (Brain, Norman, Gray, & Mansel, 1999). To our knowledge, research has not yet examined whether certain levels of anxiety can be protective or beneficial in the context of disease management in pediatric type 1 diabetes. In the context of managing type 1 diabetes, it may be that at certain levels, anxiety is associated with better adherence and glycemic control possibly due to increased vigilance to treatment.

Research examining comorbid symptoms of depression and anxiety and their combined relationship with treatment adherence and A1c levels is also lacking. In the general population of adolescents, the co-occurrence of depression and anxiety is quite high; 50% of adolescents with one diagnosis experience the other as well (Seligman & Ollendick, 1998). In addition, those who experience both symptoms of depression and anxiety are more significantly impaired than those with either disorder alone (Brady & Kendall, 1992; Kovacs & Devlin, 1998). Considered together, it is possible that the concurrent presence of elevated anxiety and depressive symptoms may be associated with even poorer adherence and glycemic control. This is the case in asthma; there are elevated rates of comorbidity (Goodwin, Fergusson, & Horwood, 2004) and the presence of anxiety and depressive symptoms is associated with greater perceived illness burden as well as functional impairment (Katon, Richardson, Russo, Lozano, & McCauley, 2006). Comorbidity may be more problematic than either anxiety or depressive symptoms alone, and may thus carry important implications for the health outcomes of adolescents with type 1 diabetes.

The purpose of the current study was to address the aforementioned gaps in the literature and to document the associations between anxiety symptoms, diabetes management, and glycemic control. Study aims were (a) to document the rates and correlates of anxiety in a large sample of adolescents with type 1 diabetes and (b) to document the magnitude of the associations between anxiety, depressive, and comorbid symptoms with diabetes management and glycemic control. We wanted to collect data on the incidence of elevated scores along both dimensions of anxiety and depression in the same large sample of adolescents with type 1 diabetes. We hypothesized that anxiety symptoms would be associated with BGM frequency and glycemic control. We also hypothesized that at moderate levels, anxiety symptoms would be associated with more frequent BGM and more optimal glycemic control. At higher levels of anxiety symptoms, lower BGM frequency and suboptimal glycemic control would occur.

**Methods**

**Participants and Procedures**

A total of 276 adolescents (ages 13–18) with type 1 diabetes and their primary caregivers participated. All adolescents had a diagnosis of type 1 diabetes according to the practice guidelines of the American Diabetes Association (ADA) (Silverstein et al., 2005) and were receiving care from a multidisciplinary team at one of two pediatric diabetes centers (Northeastern and Midwestern clinical sites). Exclusion criteria included the presence of a major psychiatric or neurocognitive disorder
that would inhibit ability to participate; a significant medical disease other than type 1 diabetes, treated thyroid disorders, or celiac disease; or the inability to read or understand English. At the Northeastern site, 126 adolescents participated. They were drawn from a sample of 173 eligible adolescents who were approached as a convenience sample about participation (agreement rate of 73%). At the Midwestern site, 150 adolescents participated from the 166 eligible adolescents similarly approached (agreement rate of 90%). All study procedures were approved by the Institutional Review Board at each clinical site. After obtaining written informed consent from caregivers and consent/assent from the youth, a research assistant administered questionnaires. All questionnaires were completed in the pediatric diabetes clinic before or after the adolescent’s clinic visit.

**Measures**

The State–Trait Anxiety Inventory (STAI) (Spielberger, 1983) was used to measure adolescent state and trait anxiety symptoms as well as caregiver trait anxiety symptoms. The STAI has 40 items with half representing present feelings (state scale) and the other half related to feelings in general (trait scale). The STAI for children/adolescents (STAIC) does not provide clinical cutoffs to denote elevated levels of anxiety suggestive of further evaluation. Of the only STAIC clinical cutpoint available (Vila et al., 1999) the false positive rate is 45%. On both scales, there was a high degree of internal consistency: adolescent (state coefficient \( \alpha = .87 \); trait coefficient \( \alpha = .87 \)) and caregiver (state coefficient \( \alpha = .93 \); trait coefficient \( \alpha = .92 \)).

Youth depressive symptoms were assessed with the Children’s Depression Inventory (CDI) (Kovacs, 2003), a self-report questionnaire consisting of 27 items rated from 0 (no symptom) to 2 (distinct symptom). Possible CDI scores range from 0 to 54 with a clinical cutoff score of 13 or higher indicative of elevated depressive symptoms and suggestive of further evaluation (Grey et al., 2002; Kovacs, 2003). Youth responses on the CDI demonstrated a high degree of internal consistency (coefficient \( \alpha = .86 \)). At the time of the clinic appointment, participants’ meters were downloaded (if available) and the daily BGM frequency was calculated across the past two weeks of data. The adolescent and caregiver also provided self-report of daily BGM frequency. In this sample, 158 adolescents provided meters for downloading. For these 158 adolescents, their meter downloaded daily frequency was highly correlated with \( r = .66, p < .0001 \) and very similar to their self-report (meter mean = 4.15 times per day, \( SD = 1.89 \); self-report mean = 4.26 times per day, \( SD = 1.27 \)). Because of this self-report inflation, the other 118 adolescents with only self-report data had their self-report value adjusted by multiplying it by 0.97 (4.15/4.26). Of note, the 118 adolescents with self-report only had a mean value of 3.97 (\( SD = 1.52 \)) prior to adjustment, which was not significantly different from the mean value with meter download. Further, there were no significant differences between those who did and did not bring their meters to the clinic visit on all sociodemographic and family variables (e.g., age, gender, insurance status, and caregiver educational level) and glycemic control. However, adolescents with meters reported slightly less trait anxiety \( t(274) = -2.32, p < .05 \).

Adolescents at the Northeastern clinical site had their glycemic control (A1c value) determined by high-performance liquid chromatography (reference range 4.0–6.0%, Tosoh 2.2; Foster City, CA, USA). Youth at the Midwestern clinical site had their A1c values measured by the DCA 2000+ (reference range 4.3–5.7%, Bayer Inc.; Tarrytown, NY, USA). Prior studies have shown that A1c values obtained from the laboratory and DCA 2000+ measurements show high agreement, \( r = .94 \) (Tamborlane et al., 2005).

Duration of diabetes and mode of insulin administration were obtained from chart review. Family demographic data were obtained from a self-report questionnaire completed by the adolescent’s caregiver during the study visit.

**Statistical Analyses**

Prior to analysis, data were double entered and cross-checked for accuracy. Descriptive statistics, frequencies, and univariate comparisons were calculated for the total sample as well as for each site. To address the first study aim of documenting the rates and correlates of anxiety symptoms in this sample of adolescents with type 1 diabetes, we examined bivariate correlations among adolescent and family variables, disease characteristics, and the psychological constructs of interest (anxiety, depression) (Table II). Our second study aim of documenting whether anxiety is associated with BGM frequency and A1c values was addressed in two ways. First, we examined the univariate relationship between anxiety symptoms, BGM frequency, and A1c values to determine if there is a point where anxiety becomes more problematic for these outcomes. To do this, we examined the presence of significant differences in BGM frequency and A1c levels in anxiety groups (low, moderate, and high levels) via an analysis of variance and by assessing the nonlinear relationship between anxiety and outcome by entering a quadratic
term into regression models. Second, we ran separate multivariate analyses in the general linear model framework to determine the factors associated with BGM frequency and A1c. These models contained sociodemographic, family, and disease covariates (age, gender, ethnicity, type 1 diabetes duration, mode of insulin delivery; caregiver education level, insurance status, and marital status); indicators of anxiety (state, trait), indicators of caregiver anxiety (trait), depressive symptoms, and comorbidity status. In addition, the clinical site and whether BGM frequency was obtained via meter download or self-report were also included in all analyses as covariates. Both state and trait anxiety were included in the models because of the relationship between these two constructs; that is, an individual’s trait anxiety manifests itself by the general tendency to perceive situations as threatening and anxiety-provoking, and to subsequently respond with state anxiety in any given situation. In addition, individuals experience different levels of state anxiety based on their level of trait anxiety (Spielberger, 1972). Caregiver trait anxiety was included because of its documented positive associations with both child anxiety and A1c (Cameron et al., 2007) and because it provides an indicator of an “anxious” family environment. Depressive symptoms and comorbidity were included to examine whether the combination of depression and anxiety places adolescents at greater risk of poor treatment BGM and A1c. Comorbidity was calculated by first dummy coding depression (above clinical cutoff of 13) and state anxiety (>1 SD above mean) values and consequently summing up these two values in order to obtain a comorbidity score of anxiety and depression (0 = no anxiety and depression, 1 = presence of anxiety or depression, or 2 = presence of comorbid anxiety and depression). Analyses were conducted in SAS v9.1 (SAS Institute, Cary, NC, USA).

### Results

#### Participant Characteristics

Table I displays characteristics of the total sample of adolescents and caregivers as well as by clinical site. The mean age for this sample was 15.63 years (SD = 1.39, range: 13.0–18.5 years), with a near equal proportion of males and females. Approximately 87% of the sample was white, not of Hispanic origin. The sample had a mean duration of diabetes of 6.61 years (SD = 4.01,
Correlations between study variables, demographic and disease characteristics can be found in Table II. Of note, adolescent state anxiety was significantly correlated with A1c values \((r = .25, p < .001)\), BGM frequency \((r = -.25, p < .001)\), and depressive symptoms \((r = .55, p < .001)\). Adolescent trait anxiety was associated with BGM frequency \((r = -.17, p = .005)\) and depressive symptoms \((r = .72, p < .001)\), but not A1c values. Effect sizes of BGM frequency and A1c values for adolescents with state and trait anxiety 1 SD above the mean versus the remainder of the sample are as follows: state anxiety (BGM frequency, Cohen’s \(d = .66\); A1c, Cohen’s \(d = .73\)), trait anxiety (BGM frequency, Cohen’s \(d = .18\); A1c, Cohen’s \(d = .28\)).

Trajectory of Anxiety on BGM Frequency and A1c

To assess whether there exists an “optimal” level of anxiety, we examined the relationship between these variables at low (<1 SD below mean), moderate (i.e., within ±1 SD around mean), and high (i.e., >1 SD above mean) levels of anxiety (state and trait). Findings revealed differences between anxiety levels on BGM frequency \([\text{state: } F(2, 273) = 8.35, p < .01 \text{ and trait: } F(2, 273) = 3.42, p < .05]\) and A1c levels \([\text{state: } F(2, 273) = 11.28, p < .01, \text{ trait: nonsignificant}]\). Adolescents with high levels of state and trait anxiety had lower BGM frequency and adolescents with high levels of state anxiety had higher A1c levels than adolescents endorsing moderate and low levels of state anxiety. There were no significant differences between levels of trait anxiety on A1c levels. When the nonlinear relationship between anxiety symptoms (examined separately for state and trait anxiety), BGM frequency, and A1c values was examined in a regression analysis, results revealed a nonsignificant quadratic term for the relationship between anxiety (state and trait) and both BGM frequency and A1c. Overall, there was no point

### Table II. Correlations of Study Variables

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BGM, blood glucose monitoring; STAI = State Trait Anxiety Inventory; CDI = Child Depression Inventory.

*p < .01;

*p < .05.

### Rates and Correlates of Anxiety

Adolescents had a mean STAI state anxiety score of 29.82 \((SD = 4.99)\) and STAI trait anxiety score of 32.15 \((SD = 7.01)\). These are comparable to published norms for children who are otherwise medically healthy (Spielberger, Edwards, Montuori, & Luchene, 1973) and for children with type 1 diabetes (Grey et al., 1995). In this sample, 13.4% and 17% of adolescents reported state and trait anxiety scores (respectively), falling 1 SD above the sample mean.1 Approximately 21% of adolescents had CDI scores ≥13 (the clinical cutoff), again consistent with published rates of depressive symptoms in adolescents with diabetes (Grey et al., 2002; Whittemore et al., 2002). With regard to caregiver anxiety, the mean on the STAI trait scale was 36.15 \((SD = 9.26)\), similar to published norms for working adults (Spielberger, 1983).

1 Since STAIC clinical cutoffs are not available, rates of state and trait anxiety do not correspond to pre-established clinically significant levels of anxiety, nor do they convey a diagnosis of an anxiety disorder.
at which anxiety was associated with optimal BGM or glycemic control.

**Multivariate Models**

With BGM frequency as the dependent variable, the following variables were entered into the model: sociodemographic, family, and disease covariates (adolescent age, gender, ethnicity, type 1 diabetes duration, mode of insulin delivery; caregiver education level, insurance status, and marital status); adolescent anxiety (state and trait) and depressive symptoms; caregiver trait anxiety; and the comorbidity index. Clinical site and availability of meter download were also included in all analyses as covariates. The overall model was significant, $F(14, 261) = 6.35, p < .0001, R^2 = .25$. Lower BGM frequency was associated with older age ($p < .0001$), lower caregiver educational attainment ($p = .008$), participation at the Midwestern site ($p = .01$), insulin delivery via injections versus CSII ($p < .001$), and higher state anxiety ($p = .03$). Depressive symptomatology alone and comorbidity were not associated with BGM frequency.

With A1c value as the dependent variable and using the same set of independent variables plus BGM frequency, the model was significant, $F(15, 260) = 7.97, p < .0001, R^2 = .32$. Higher A1c values (i.e., suboptimal glycemic control) were associated with single-caregiver marital status ($p = .01$), participation at the Northeastern site ($p = .04$), longer diabetes duration ($p = .02$), less frequent BGM ($p < .0001$), and higher state anxiety ($p = .002$). Again, there was no significant association with depressive symptoms or comorbidity.

**Discussion**

This study aimed to identify the prevalence of anxiety symptoms and their association with BGM frequency and glycemic control in adolescents with type 1 diabetes. Results indicated that 17% of the sample had trait anxiety symptoms at a level that warrant further clinical evaluation; the rate was 13% for state anxiety symptoms. These rates are slightly lower than the percentage of adolescents (21%) who met the clinical cutoff for depressive symptoms; however, state anxiety symptoms were independent correlates of BGM frequency and glycemic control above and beyond depressive symptoms. Associations between anxiety symptoms, BGM frequency, and glycemic control were similar to what has been found in depressive symptoms; higher levels of problematic psychological functioning were associated with less frequent BGM and suboptimal glycemic control. No level of anxiety was associated with more optimal BGM or glycemic control.

The statistical models used to analyze the multivariate associations in these data assumed the directional hypothesis that higher levels of anxiety symptoms contribute to poorer disease management and glycemic outcomes. Within this framework, the similarities between anxiety and depressive symptoms and their associations with BGM frequency and glycemic control may be due to shared characteristics such as negative affect, diminished ability to concentrate, and irritability (Barlow & Campbell, 2000; Seligman & Ollendick, 1998). Further, both anxiety and depression are associated with increased cognitive burden. For example, research on children and adolescents in general has shown that those who are anxious may have difficulty with concentration (Kendall & Pimentel, 2003) and problem-solving (Emerson, Mollet, & Harrison, 2004), and they may be inattentive and forgetful (Jarrett & Ollendick, 2008). Staying within this directional framework, anxiety symptoms that impose cognitive burden on the adolescent with type 1 diabetes may result in forgetting to carry out tasks (e.g., checking blood glucose levels). We also know that adolescents with type 1 diabetes who are depressed tend to experience cognitive burden and consequently experience a diminished ability to concentrate and engage in disease management (Grey et al., 1995; Hains, Berlin, Davies, Parton, & Alemzadeh, 2006). Interestingly, the combined effect of these symptoms (as measured with the comorbidity variable) did not add to the variance explained in BGM frequency and glycemic control. The lack of this effect may be due to our comorbidity designation being based on self-report questionnaires. An evaluation of actual diagnoses may have revealed an additive effect of comorbidity. Nonetheless, the overlapping characteristics of anxiety and depressive symptoms may be at the core of why the nature of the associations with BGM frequency and glycemic control, and the assumed direction, are similar.

Despite these similarities, the results indicate that one type of anxiety measured in this study, the state anxiety symptoms, have a unique association with BGM frequency and glycemic control that goes above and beyond depressive symptoms. This may be due to specific symptoms of state anxiety that differ from depressive symptoms; examples are increased heart rate and perspiration, dizziness, lightheadedness, and abdominal discomfort (Beidel, Christ, & Long, 1991; Ginsburg, Riddle, & Davies, 2006). Perhaps among adolescents with type 1 diabetes, these anxiety-induced somatic symptoms that resemble those associated with glycemic excursions (e.g., hyperglycemia) create difficulties in making treatment decisions.
For instance, adolescents may think they are experiencing lightheadedness due to hyperglycemia and then subsequently check their blood glucose level. When they discover the blood glucose level is actually not elevated, they may be less likely to check the next time they feel that way because it was a “false alarm.” This could potentially lead to fewer checks and ultimately, poorer glycemic control. This may also explain why no level of anxiety promoted optimal disease outcomes (i.e., blood glucose checks and glycemic control). Symptoms of anxiety may predominantly interfere with youths’ ability to appropriately gauge their glycemic levels rather than promote better disease management.

An equally plausible explanation assumes the opposite direction—aspects of diabetes and its management contribute to more state anxiety symptoms. Consider that all participants completed their measures at the time of their clinic visit for diabetes care. The state anxiety values obtained in this study may reflect situational nervousness about meeting with their diabetes-specific healthcare provider, what the A1c value may be, or just having had the meter downloaded to reveal infrequent BGM. In these cases, state anxiety may be a signal of problematic management and control; prior studies on related topics provide support for this interpretation. Research on fear of hypoglycemia highlights increased fear in response to the frequency and severity of hypoglycemic episodes (Irvine, Cox, & Gonder-Frederick, 1992; Green, Wysocki, & Reineck, 1990; Kamps et al., 2005), and that these fears subsequently contribute to poorer management and glycemic outcomes (Wild et al., 2007). Additionally, carrying out diabetes management tasks with awareness of the potential for long-term complications associated with the disease may also generate anxiety (Buckloh et al., 2008). Given that the results presented here are from cross-sectional data, there is no way to conclusively state which is the most accurate direction for these associations. The most likely scenario, however, may be that the exposure to diabetes, its management, and its potential negative health consequences contributes to an adolescent’s level of anxiety and then subsequently, those worries influence health behaviors and outcomes. Future longitudinal research that assesses anxiety and health behaviors at multiple time points should be able to test these hypothesized relationships and clarify the direction of these relationships.

The results on other correlates of suboptimal glycemic control highlight the link between BGM frequency and A1c values. Specifically, adolescents who engaged in less frequent BGM had higher A1c values. Longer diabetes duration was also linked to suboptimal glycemic control, possibly due to accumulating burden of disease management or direct biologic impact (Amiel, Sherwin, Simonson, Lauritano, & Tamborlane, 1986). Previous research has similarly shown that adolescents using injections had higher A1c levels than adolescents using CSII (Phillip, Battelino, Rodriguez, Danne, & Kaufman, 2007). Also, single caregiver marital status may be associated with lack of social support and greater caregiver burden which leads to diminished family involvement in diabetes management, adversely affecting diabetes self-care and resulting in suboptimal levels of glycemic control (Overstreet, Holmes, Dunlap, & Frenz, 1997). Again, longitudinal research may reveal different correlates of suboptimal glycemic control. Interestingly, caregiver anxiety was not a significant correlate of BGM frequency and glycemic control. This may be due to testing this association in an older, adolescent sample. Prior research has similarly shown that caregiver anxiety is not associated with disease outcomes in older children (Cameron et al., 2007). As adolescents become more autonomous and take on more responsibility for disease management (Anderson et al., 2002; Schilling, Knafl, & Grey, 2006), caregiver psychological health may become less salient for adolescent outcomes.

Findings from this study should be considered within the context of several limitations in addition to those noted previously about our cross-sectional data and approach to understanding the direction of these associations. First, the STAI only provides a broad measure of anxiety symptoms and does not provide a clinical cutpoint; nor convey a diagnosis of an anxiety disorder. Further, the STAI assesses generic anxiety symptoms rather than disease-specific ratings of anxiety and as a result, anxiety directly related to disease management may not have been adequately captured with the STAI. Second, even though only minor differences were found between clinical sites, this study could not control for differences inherent in clinical practice across site. Third, although availability of meter download was applied to analyses as a covariate and was not significant, combining self-report data with meter downloads may be a limitation. Self-report data is subjective and some adolescents may overestimate BGM frequency to make themselves appear more favorable. Self-reported BGM frequency could also be more exaggerated when the youth know the meter is elsewhere versus when they know that the meter is being downloaded at that same clinic visit. Youth with low BGM may selectively leave their meters at home to avoid conflict rather than bringing them to the clinic visit. In addition, this study demonstrated a level of BGM frequency that may be higher than what is seen in general pediatric samples.
In future studies, we hope to have complete BGM download data for all participants. Fourth, the adolescents in this sample were predominantly white and living in two-caregiver households. There is the possibility that the findings in this study do not generalize to samples with characteristics that are not similar to this sample.

The results of this study highlight several implications for clinical practice and clinical research. The association between anxiety symptoms, BGM frequency, and suboptimal glycemic control suggest that this may be an important clinical variable for multidisciplinary teams to consider when working with adolescents with type 1 diabetes. These teams may be able to implement brief anxiety screenings (e.g., using the STAI) and ask probing questions about anxiety, similar to what is done around depression (Delamater et al., 2001; Silverstein et al., 2005). Understanding whether anxiety symptoms are rooted in general areas or are diabetes-specific (e.g., fear of hypoglycemia) may be particularly helpful in treatment planning and delivery. It may be equally useful to determine types of anxiety (e.g., fears, phobias, obsessions/compulsions) and symptoms (e.g., difficulty concentrating, fatigue, forgetfulness) that may interfere with diabetes management and glycemic control, and to determine if anxiety symptoms are experienced after adverse health events. Cognitive–behavioral interventions are empirically supported for treating anxiety and depression in adolescents who are otherwise medically healthy (Kendall, 1993; Kendall, Hudson, Gosch, Flannery-Shroeder, & Suveg, 2008) and may be beneficial for adolescents with type 1 diabetes. However, in use with adolescents with type 1 diabetes, these interventions likely need to be tailored to address anxiety symptoms that are the result of, or result in, suboptimal diabetes management and control.

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