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# Depressive Symptoms and Glycemic Control in Adolescents With Type 1 Diabetes

## Mediational role of blood glucose monitoring

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**OBJECTIVE**— To determine whether the association between depressive symptoms and glycemic control is mediated by blood glucose monitoring (BGM).

**RESEARCH DESIGN AND METHODS**— A total of 276 adolescents with type 1 diabetes (mean age  $\pm$  SD, 15.6  $\pm$  1.4 years) completed a measure of depressive symptoms. Socio-demographic and family characteristics were obtained from caregivers. BGM frequency and glycemic control were obtained at a clinic visit.

**RESULTS**— Separate regression analyses revealed that depressive symptoms were associated with lower BGM frequency ( $B = -0.03$ ;  $P = 0.04$ ) and higher A1C ( $B = 0.03$ ;  $P = 0.05$ ) and that lower BGM frequency was associated with higher A1C ( $B = -0.39$ ;  $P < 0.001$ ). With depressive symptoms and BGM frequency included together, only BGM frequency was associated with A1C and depressive symptoms became nonsignificant ( $B = 0.02$ ;  $P = 0.19$ ). The Sobel test was significant ( $Z = 1.96$ ;  $P < 0.05$ ) and showed that 38% of the depression-A1C link can be explained by BGM.

**CONCLUSIONS**— BGM is a mediator between depressive symptoms and glycemic control in adolescents with type 1 diabetes.

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Adolescents with type 1 diabetes have elevated risk for poor blood glucose monitoring (BGM) adherence and suboptimal glycemic control (1,2). Adolescents also experience increased risk for depressive symptoms (3–5), which are associated with higher A1C values (4,5). Although BGM nonadherence and depressive symptoms both contribute to higher A1C values, little is known about their collective association with glycemic control. Previous studies in adults have tested the mediational role of adherence (6). We aimed

to evaluate whether the depressive symptoms–glycemic control link is mediated by BGM.

### RESEARCH DESIGN AND METHODS

Adolescents with type 1 diabetes were eligible for this study if they did not have a major psychiatric/neurocognitive disorder that would inhibit ability to participate, a significant medical disease other than type 1 diabetes, or the inability to read or understand English. At the northeastern clinical site, 173 eligible adolescents were approached

and 126 participated. At the midwestern site, 166 eligible adolescents were approached and 150 participated. All study procedures were approved by the institutional review board at each site. Written informed consent from caregivers and consent/assent from adolescents were obtained.

### Measures

Youth depressive symptoms were assessed using the Children's Depression Inventory (CDI) (7), a self-report questionnaire consisting of 27 items. Higher scores indicate more depressive symptomatology (4,7). There was a high degree of internal consistency in this sample (coefficient  $\alpha = 0.86$ ).

BGM frequency was obtained via meter download or self-report. Of the 276 adolescents, 158 provided meters for downloading; their daily BGM frequency was correlated with ( $r = 0.66$ ;  $P < 0.0001$ ) and similar to their self-report (daily meter mean 4.15 and self-report mean 4.26). Because of this self-report inflation, 118 adolescents with only self-report data had their values adjusted by multiplying them by 0.97 (4.15/4.26). There were no differences between adolescents with or without meter download on A1C or CDI scores ( $P$  values  $> 0.05$ ).

Adolescents at the northeastern clinical site had A1C determined by high-performance liquid chromatography (reference range 4.0–6.0%) (Tosoh 2.2; Tosoh, Foster City, CA). Adolescents at the midwestern clinical site had A1C measured by DCA 2000+ (4.3–5.7%) (Bayer, Tarrytown, NY). A1C values obtained from the laboratory and by DCA 2000+ have shown high agreement (8).

Duration of diabetes and mode of insulin administration were obtained from chart review. Family demographic data were obtained from the caregiver.

### Statistical analyses

We conducted multivariate analyses (9) using general linear modeling to test the hypothesis that BGM would mediate the

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depressive symptoms—glycemic control link. First, the effect of depressive symptoms on BGM frequency was tested. Second, the effect of depressive symptoms on A1C values was tested. Third, BGM frequency was added to test its mediational role. The Sobel test examined the significance of the mediational effect, and a post hoc model of interactions was tested. Covariates (age, sex, ethnicity, diabetes duration, and mode of insulin delivery; caregiver education level, insurance status, and marital status; site; and availability of meter download) were included in all models. Analyses were conducted using SAS (version 9.1; SAS Institute, Cary, NC).

**RESULTS**— Table 1 displays characteristics of the total sample by site. The data for the first model (depressive symptoms plus covariates → BGM) were significant. Lower levels of BGM frequency were associated with more depressive symptoms ( $B = -0.03$ ;  $P = 0.02$ ), insulin delivery via injections ( $B = 0.85$ ;  $P < 0.001$ ), less caregiver education ( $B = -0.53$ ;  $P = 0.01$ ), participation at the midwestern site ( $B = 0.85$ ;  $P = 0.005$ ), and older age ( $B = -0.34$ ;  $P < 0.001$ ).

The data for the second model (depressive symptoms plus covariates → glycemic control) were significant. Higher A1C values were associated with more depressive symptoms ( $B = 0.03$ ;  $P = 0.05$ ), longer diabetes duration ( $B = 0.07$ ;  $P = 0.007$ ), insulin delivery via injections ( $B = 0.74$ ;  $P = 0.001$ ), and single caregiver marital status ( $B = 0.63$ ;  $P = 0.02$ ).

The data for the third model (depressive symptoms plus BGM frequency plus covariates → glycemic control) were significant; however, with depressive symptoms and BGM frequency in the model, the effect of depressive symptoms became nonsignificant ( $B = 0.02$ ;  $P = 0.19$ ). Higher A1C values were associated with lower levels of BGM frequency ( $B = -0.39$ ;  $P < 0.001$ ), longer duration of diabetes ( $B = 0.05$ ;  $P = 0.03$ ), single caregiver marital status ( $B = 0.58$ ;  $P = 0.02$ ), and participation at the northeastern site ( $B = 0.61$ ;  $P = 0.05$ ). The Sobel test, evaluating the magnitude of mediation, was significant ( $Z = 1.96$ ;  $P < 0.05$ ); 37.5% of the depressive symptoms—glycemic control link was explained by BGM. A post hoc model included interactions between significant covariates and BGM frequency; however, none were significant, indicating that covariates were directly associated with glycemic control.

**Table 1—Participant characteristics**

	Total sample	Northeast	Midwest
<i>n</i>	276	126	150
Age (years)	15.6 ± 1.4	15.8 ± 1.4	15.5 ± 1.4
Female sex	47.5	42.9	51.3
Ethnicity (non-Hispanic white)	87.3	88.9	86.0
Caregiver marital status (married)	80.4	84.1	77.3
Primary caregiver (mother)	82.6	77.8	86.7
Education level of primary caregiver (at least a college degree)	54.0	62.7	46.7
Insurance status (private)	84.4	85.7	83.3
Type 1 diabetes duration (years)	6.6 ± 1.81	7.3 ± 4.0	6.0 ± 3.9
A1C (%)	8.9 ± 1.8	9.0 ± 1.7	8.8 ± 1.9
BGM frequency (times daily)	3.83 ± 1.45	4.08 ± 1.38	3.61 ± 1.48
Method of insulin delivery			
Multiple daily injections	44.9	54.8	36.7
CSII	55.1	45.2	63.3
CDI score	7.3 ± 6.4	6.5 ± 5.4	8.0 ± 7.1

Data are means ± SD or % unless otherwise indicated. CSII, continuous subcutaneous insulin infusion.

**CONCLUSIONS**— Results from this cross-sectional analysis indicate that the depressive symptoms—glycemic control link is partially explained by BGM. Previous research highlights that depressive symptoms are associated with lower self-efficacy, negative attributions, and diminished ability to concentrate (10,11). Adolescents with type 1 diabetes and elevated depressive symptoms may have trouble initiating tasks for diabetes management, carrying them out, and believing they will be effective.

The primary implication is that careful monitoring of depressive symptoms is warranted. This recommendation has been advocated previously in individual studies (4,5) and by the American Diabetes Association task force (12); however, these findings highlight the potential for negative consequences with regard to diabetes management and outcomes when elevated depressive symptoms exist. Second, once identified, depressive symptoms need to be treated. There is strong empirical evidence that cognitive-behavioral treatments are effective in reducing depressive symptoms in adolescents (13); thus, this appears to be a viable option, especially if the diabetes-specific context is considered. Third, straightforward attempts to promote BGM adherence may prove ineffective. Phased interventions may be most appropriate; one should attempt to reduce depressive symptoms before attempting to promote BGM adherence.

Limitations include the inability to rule out bidirectional relationships be-

tween depressive symptoms and glycemic control in these cross-sectional data. Depressive symptoms were measured by self-report using CDI, providing an indication of clinically significant levels of depressive symptoms and not a diagnosis. Adolescents were predominantly white with married caregivers; findings may not generalize to more diverse families. Finally, we only examined BGM adherence. While previous studies highlight the importance of BGM frequency as an indicator of overall adherence and glycemic outcomes (14,15), future studies should examine multiple dimensions of adherence.

In sum, adolescents with type 1 diabetes who experience elevated depressive symptoms are also likely to experience problems with BGM. When that occurs, suboptimal glycemic control likely results. Continued surveillance of depressive symptoms is suggested, and targeted interventions for these adolescents within a diabetes-specific framework appear warranted.

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