



# Disordered Eating Behaviors Are Not Increased by an Intervention to Improve Diet Quality but Are Associated With Poorer Glycemic Control Among Youth With Type 1 Diabetes

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## OBJECTIVE

This study examines whether participation in an 18-month behavioral intervention shown previously to improve overall diet quality inadvertently increases disordered eating behaviors (DEBs) in youth with type 1 diabetes and investigates the association of DEB with multiple measures of glycemic control and variability.

## RESEARCH DESIGN AND METHODS

Participants reported DEB and diabetes management at baseline and 6, 12, and 18 months; masked continuous glucose monitoring, HbA<sub>1c</sub>, and 1,5-anhydroglucitol (1,5-AG) were obtained concurrently. Linear mixed models estimated the intervention effect on DEB, the association of DEB with diabetes adherence and measures of glycemic control and variability, and whether DEB modified glycemic trajectories.

## RESULTS

There was no intervention effect on DEB ( $P = 0.84$ ). DEB was associated with higher HbA<sub>1c</sub> ( $P = 0.001$ ), mean sensor glucose ( $P = 0.001$ ), and percent sensor glucose values  $>180$  mg/dL ( $P = <0.001$ ); with lower 1,5-AG ( $P = 0.01$ ); and with worse diabetes adherence ( $P = 0.03$ ). DEB was not associated with percent sensor glucose values  $<70$  mg/dL or any measures of glycemic variability. There was a significant DEB  $\times$  time interaction effect for mean sensor glucose ( $P = 0.05$ ) and percent sensor glucose values  $>180$  mg/dL ( $P = 0.04$ ). Participants reporting less DEB had a developmentally expected deterioration in glycemic control throughout the study. Participants reporting more DEB had poor glycemic control at baseline that remained poor throughout the study.

## CONCLUSIONS

Findings show a potential to improve diet quality without increasing DEB and indicate an association of DEB with persistent hyperglycemia but not hypoglycemia or glycemic variability.

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Adolescents with type 1 diabetes may be more susceptible to disordered eating behaviors (DEBs) than their peers without diabetes owing to the focus on diet and carbohydrate intake necessitated by diabetes management and the risk of insulin-related weight gain (1,2). DEBs are unhealthy weight-management behaviors, such as skipping meals or purging, that are not severe or frequent enough to warrant classification as diagnosable psychiatric eating disorders (3). In the context of type 1 diabetes, DEBs may also include insulin restriction or omission to induce weight loss.

Evidence from cross-sectional studies suggests that DEBs are associated with higher glycated hemoglobin (HbA<sub>1c</sub>) (2,4–6), but important research gaps remain regarding the prospective association of DEB with multiple measures of glycemic control including hyperglycemia and glycemic variability. We found only two prospective studies examining the effects of DEB in youth with type 1 diabetes; results showed an association of DEB with poorer HbA<sub>1c</sub> 3 years later (7) and with increased retinopathy 4–5 years later (8). More prospective research during adolescence would be particularly useful, given the developmentally expected decline in glycemic control already observed in this age-group. Research examining the associations of DEB with hyperglycemia and glycemic variability is also warranted, as these are independently associated with long-term diabetes complications such as retinopathy, renal disease, and cardiovascular events (9–14). In addition, much of the previous research on DEB in patients with type 1 diabetes is limited by reliance on assessments of DEB that were developed for and validated in populations without diabetes, which are not appropriate for patients with diabetes (2,4). These measures often fail to identify DEB unique to type 1 diabetes—such as insulin omission—and risk inflating measurement of DEB by identifying some behaviors important for diabetes management as disordered, such as vigilance as to the amount of carbohydrate consumed. Thus, studies that examine prospectively the degree to which DEB modifies the developmentally expected decline in hyperglycemia, hypoglycemia, and glycemic variability using DEB measures designed for populations with diabetes have potential to advance understanding of the adverse effects of DEB in youth with type 1 diabetes.

In the general population, dietary restriction is associated with DEB (15–18), and there is concern that behavioral nutrition interventions may unintentionally increase risk of DEB through increased attention to dietary intake (19). DEB risk may be exacerbated in patients with type 1 diabetes owing to the increased attention to dietary intake for the purpose of disease management such as is seen with other chronic illnesses (20–22). Concurrently, there is a well-documented need for improved diet quality in adolescents with type 1 diabetes (23), but it is unknown whether behavioral interventions to improve diet quality adversely impact DEB, which could inhibit efforts to improve health outcomes in this population.

We previously reported that participation in a behavioral nutrition intervention successfully improved overall diet quality in youth with type 1 diabetes (24). The intervention included various components that could be expected to increase attention to dietary intake such as dietary monitoring, meal-planning strategies, recipe books, and sessions focused on intake of whole plant foods and attention to the dietary quality of carbohydrates at each meal. The two main objectives of the current study are to examine whether treatment assignment is associated with increased DEB and to examine associations of DEB with biomarkers of glycemic control and variability in treatment and control participants over 18 months. We further examine whether DEB modifies the degree of developmentally expected decline in glycemic control.

## RESEARCH DESIGN AND METHODS

### Participants, Design, and Procedure

This is a secondary data analysis of a randomized clinical trial of a family-based behavioral nutrition intervention among youth with type 1 diabetes ( $n = 136$ ). The study was conducted from August 2010 through May 2013 at an outpatient, free-standing, multidisciplinary tertiary diabetes center in Boston, MA. Eligibility criteria included age 8.0–16.9 years, diagnosis of type 1 diabetes for  $\geq 1$  year, daily insulin dose  $\geq 0.5$  units/kg, most recent HbA<sub>1c</sub> 6.5–10.0%, insulin regimen of three or more injections daily or use of insulin pump, at least one clinic visit in the past year, and ability to communicate in English. Exclusion criteria included daily use of premixed insulin, transition to

insulin pump in the last 3 months, real-time continuous glucose monitoring (CGM) use in the last 3 months, participation in another intervention study in the last 6 months, or presence of gastrointestinal disease (24). Of 622 youth invited, 148 (24%) agreed to participate in the main trial. Of these, nine did not complete the baseline assessment, and data from one sibling of three sibling pairs were excluded. Further, this analysis includes only those participants who completed the DEB questionnaire at any time during the study, which was administered only to participants age 13 years or older ( $n = 90$ ). Assessments were conducted at baseline and 6 (near the end of the core intervention sessions), 12 (during booster sessions), and 18 months (follow up).

### Intervention

The intervention comprised nine in-person sessions led by trained research personnel and included behavioral techniques and educational content to promote increased consumption of fruits, vegetables, whole grains, and legumes. Sessions included motivational interviewing, educational content, learning activities, and an applied goal-setting and problem-solving process focused on the target food groups. The first six sessions occurred approximately monthly and comprised the “core” sessions. After an initial overview session, each focused on a specific eating occasion (breakfast, lunch, dinner, snacks, and eating out). The final three sessions comprised the “booster” sessions and occurred at months 9, 10, and 15. These addressed challenges specific to social eating, meal planning, and the food environment. The control group was designed to match on potentially important aspects of research contact that could influence health outcomes but were not the focus of the dietary intervention such as frequency of contacts with research staff, completing food records concurrently with CGM, and receiving feedback on CGM data. The intervention did not specifically target weight loss or disordered eating. DEBs were assessed to allow examination of a potential unintended adverse effect. The intervention improved overall diet quality but did not influence glycemic control (24). Study methods and primary outcomes have previously been described in detail (24,25). Study procedures were approved by the Eunice Kennedy Shriver National

Institute of Child Health and Human Development Institutional Review Board and the Joslin Diabetes Center Committee on Human Subjects.

## Measures

### Glycemic Control

All measures were obtained at baseline and 6, 12, and 18 months. HbA<sub>1c</sub> was measured using a laboratory assay standardized to the Diabetes Control and Complications Trial (reference range: 4–6% [20–42 mmol/mol]). 1,5-Anhydroglucitol (1,5-AG) was assessed using an enzymatic (glucokinase) assay (GlycoMark, New York, NY).

Three-day masked CGM data were obtained using the Medtronic iPro Continuous Glucose Monitoring System. The GlyCulator glycemic variability calculation tool (26) calculated summary indexes including mean sensor glucose, SD of sensor glucose values (overall glucose variability), mean amplitude of glycemic excursions (MAGE) (indicator of the magnitude of glucose fluctuations), percent of sensor glucose values >180 mg/dL (hyperglycemia), and percent of sensor glucose values <70 mg/dL (hypoglycemia).

### Self-Reported Measures

#### Diabetes Management Questionnaire

This 20-item measure assessed adherence to diabetes management tasks over the previous month; higher scores indicate greater adherence. Response options range from 0 (almost never) to 4 (almost always); the total score was transformed by multiplying the mean of all completed items by 25, resulting in a possible range of 0–100 (27).

#### DEBs

The Diabetes Eating Problem Survey–Revised (DEPS-R) includes 16 items assessing DEB among youth with type 1 diabetes. Response options range from 0 (never) to 5 (always) and address general DEB (e.g., “I feel that my eating is out of control”) and diabetes-specific DEB (e.g., “I try to keep my blood sugar high so that I will lose weight”). Scores range from 0 to 80, and higher scores indicate greater endorsement of DEB. This measure has previously demonstrated good internal consistency ( $\alpha = 0.86$ ) and construct validity. It is validated for use among adolescents age  $\geq 13$  years (28).

#### Demographic and Biomedical Data

Youth height, weight, sex, Tanner stage, and age were collected through medical

record review, and BMI percentiles were calculated for classifying weight status according to the Centers for Disease Control and Prevention sex- and age-adjusted cutoffs (29). BMI was calculated weight in kilograms divided by the square of height in meters. Parents reported information on education level, household income, and composition. The income-to-poverty ratio was calculated as reported household income divided by the 2008 U.S. census poverty threshold for household size and composition adjusted for inflation (30). Higher values indicate greater income relative to the poverty threshold.

### Statistical Analysis

Differences in DEPS-R between the intervention and control group at each study visit (baseline and 6, 12, and 18 months) were tested using independent samples *t* tests. A Box-Cox transformation (raised to the 0.2 as the optimal  $\lambda$ ) using the SAS TRANSREG procedure was performed on the DEPS-R to satisfy the normality assumption in linear mixed-effects models. The effect of the behavioral nutrition intervention on disordered eating over the course of the study was estimated using linear mixed-effects models specifying for a random intercept with the transformed DEPS-R scores. There were no differences between treatment groups on any of the demographic variables ( $\alpha = 0.05$ ), suggesting successful randomization, so the only covariate included in this model was time.

The associations of time-varying DEPS-R with time-varying diabetes management and indicators of glycemic control (HbA<sub>1c</sub>, 1,5-AG, and the CGM variables) were evaluated using separate linear mixed-effects models controlling for treatment group, time, age, sex, parent education, Tanner stage, race/ethnicity, BMI, height, and family income. Non-transformed, continuous DEPS-R was used in these and subsequent analyses because DEPS-R was the independent variable and, thus, not necessitating normal distribution. A DEPS-R  $\times$  time interaction term was then included in the model to examine the extent to which DEPS-R (as a continuous variable) modified the outcome trajectories; all continuous covariates were mean centered to facilitate interpretation. For interpretation of any significant interactions, average DEPS-R scores across time were calculated for each participant and a median split was

conducted based on those scores. The association of time with glycemic control was then plotted for participants with DEPS-R scores higher than the median score (higher DEB) and equal to or lower than the median DEPS-R score (lower DEB).

## RESULTS

Only participants who were at least 13 years old at some point in the study completed the DEPS-R ( $n = 90$ ) and were included in analyses; among those, one did not complete the study. Participants (51.1% of whom were female and 91% white) were a mean age of 13.7 years and had a mean DEPS-R score of  $12.4 \pm 10.1$  (out of 80 [scores ranged from 0 to 56]) (Table 1).

### Effect of the Intervention on DEB

DEPS-R scores for intervention and control groups at each assessment period (baseline and 6, 12, and 18 months) are shown in Fig. 1. There were no differences in DEPS-R scores between intervention and control groups at any assessment period (all *P* values >0.07). Further, there were no within-group differences in DEPS-R scores from baseline to any assessment period (all *P* values >0.07). Linear mixed models showed no significant treatment effect (time  $\times$  intervention interaction) on DEB over the 18-month study ( $\beta \pm$  SE;  $B = 0.0001 \pm 0.0006$ ;  $P = 0.84$ ).

### Association of Time-Varying DEB With Time-Varying Glycemic Control and Diabetes Adherence

Higher DEPS-R was associated with poorer glycemic control over time as indicated by higher HbA<sub>1c</sub> ( $B = 0.03 \pm 0.01$ ;  $P = 0.001$ ) and lower 1,5-AG ( $B = -0.04 \pm 0.01$ ;  $P = 0.01$ ). Higher DEPS-R was also associated with higher mean sensor glucose ( $B = 1.17 \pm 0.33$ ;  $P = 0.001$ ) and percent of sensor glucose values >180 mg/dL ( $B = 0.58 \pm 0.16$ ;  $P < 0.001$ ). DEPS-R was not associated with SD of the mean sensor glucose ( $B = 0.17 \pm 0.16$ ;  $P = 0.27$ ), percent of sensor glucose values <70 mg/dL ( $B = -0.06 \pm 0.06$ ;  $P = 0.32$ ), or MAGE ( $B = 0.48 \pm 0.43$ ;  $P = 0.26$ ). Higher DEPS-R was also associated with poorer diabetes management ( $B = -0.20 \pm 0.09$ ;  $P = 0.03$ ). There was a significant DEB  $\times$  time interaction effect for mean sensor glucose ( $B = 0.07 \pm 0.04$ ;  $P = 0.04$ ) and percent of sensor glucose values >180 mg/dL ( $B = -0.04 \pm 0.02$ ;  $P = 0.05$ ). Both mean sensor glucose and

**Table 1—Baseline sample characteristics**

Demographics	All participants	Treatment (N = 42)	Control (N = 48)
Age (years)	13.8 ± 1.6	13.8 ± 1.5	13.8 ± 1.7
BMI (kg/m <sup>2</sup> ) <sup>1</sup>			
Normal weight	59 (65.6)	25 (59.5)	34 (70.8)
Overweight	20 (22.2)	13 (31.0)	7 (14.6)
Obese	11 (12.2)	4 (9.5)	7 (14.6)
Sex			
Male	44 (48.9)	24 (57.1)	20 (41.7)
Female	46 (51.1)	18 (42.9)	28 (58.3)
Race/ethnicity			
White, non-Hispanic	82 (91.1)	35 (83.3)	47 (97.7)
Hispanic	7.0 (7.8)	6 (14.3)	1 (2.1)
Black, non-Hispanic	1.0 (1.1)	1.0 (2.4)	0 (0.0)
Highest parent education level			
High school or equivalent	6.0 (6.7)	4 (9.5)	2 (4.2)
Junior college, technical, or some college	15 (16.6)	5 (11.9)	10 (20.9)
College degree	33 (36.7)	14 (33.3)	19 (39.6)
Graduate education	36 (40.0)	19 (45.2)	17 (35.4)
Family income-to-poverty ratio <sup>2</sup>	5.6 ± 3.3	5.8 ± 3.4	5.4 ± 3.2
DEBs (DEPS-R) <sup>3</sup>	12.4 ± 10.1	10.1 ± 7.6	14.59 ± 11.7
Diabetes adherence <sup>4</sup>	77.9 ± 11.0	79.1 ± 9.5	76.8 ± 12.2
Diabetes and health-related characteristics			
Duration of diabetes (years)	6.7 ± 3.34	6.0 ± 2.8	7.2 ± 3.7
Regimen			
Pump or both	57 (63.3)	27 (64.3)	30 (62.5)
Injections	33 (6.7)	15 (35.7)	18 (37.5)
Frequency of blood glucose monitoring (times/day)	4.9 ± 2.4	4.9 ± 2.3	4.9 ± 2.5
Tanner stage <sup>5</sup>	3.2 ± 1.3	3.2 ± 1.2	3.2 ± 1.4
Glycemic control			
HbA <sub>1c</sub> (%)	8.2 ± 1.1	8.2 ± 1.1	8.3 ± 1.1
1,5-AG (μg/mL)	2.9 ± 1.7	2.9 ± 1.5	2.9 ± 1.9
Mean sensor glucose	175.5 ± 39.7	168.7 ± 43.6	181.6 ± 35.3
SD of sensor glucose	70.67 ± 17.3	69.7 ± 17.0	71.6 ± 17.7
Mean amplitude of glycemic excursions	206.8 ± 47.7	203.7 ± 48.9	209.5 ± 47.0
% Sensor glucose values >180 mg/dL	42.0 ± 19.6	39.3 ± 20.84	44.5 ± 18.27
% Sensor glucose values <70 mg/dL**	6.9 ± 9.5	10.0 ± 12.3	4.1 ± 4.7

Data are mean ± SD or N (%). \*\**P* < 0.01. <sup>1</sup>Based on Centers for Disease Control and Prevention BMI percentiles. <sup>2</sup>Income-to-poverty ratio calculated as reported household income divided by the 2008 U.S. census poverty threshold for household size and composition adjusted for inflation. Higher values indicate greater income relative to the poverty threshold. Missing data from two participants on family income. <sup>3</sup>Assesses general and diabetes-specific DEBs. Higher scores indicate greater endorsement of DEBs, and scores can range from 0 to 80. DEPS-R data are missing for 22 participants who were not yet age 13 years or older at baseline. <sup>4</sup>Assesses adherence to diabetes management tasks over the previous month. Higher scores indicate greater adherence; scores can range from 0 to 100. <sup>5</sup>Scores range from 1 to 5; higher scores indicate more physical development based on external primary and secondary sex characteristics.

percent of sensor glucose values >180 mg/dL were higher at baseline in the higher DEPS-R group compared with the lower DEPS-R group. However, mean sensor glucose and percent sensor glucose values >180 mg/dL increased over time in the lower DEPS-R group (Fig. 2) but did not change over time in the higher DEPS-R group.

## CONCLUSIONS

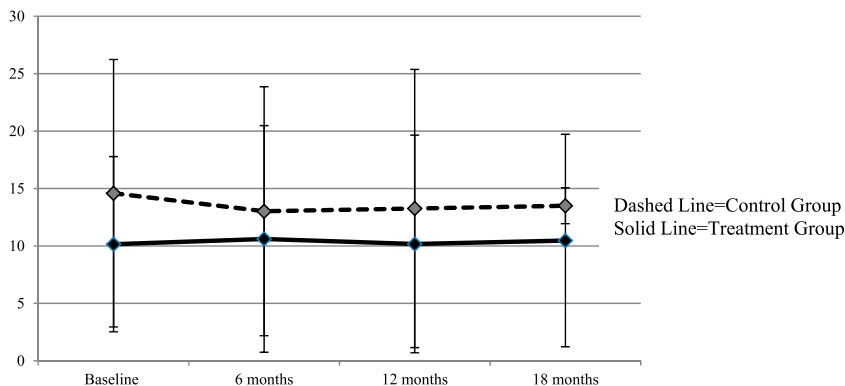
These findings indicate that participating in an effective family-based behavioral nutrition intervention to improve diet quality had no adverse effect on DEB in youth with type 1 diabetes. Overall, DEB was positively associated with indicators

of hyperglycemia, including higher HbA<sub>1c</sub>, mean sensor glucose, and percent of sensor glucose values >180 mg/dL and lower 1,5-AG. However, DEB was not associated with indicators of hypoglycemia or glycemic variability (SD of CGM values and MAGE). DEB was also associated with poorer diabetes management.

Findings from this study do not support concerns that the focus on diet inherent in diabetes management increases risk for DEB (2,31,32), which could inhibit health care providers from undertaking efforts to improve diet quality in this population. The intervention aimed to facilitate a positive approach to healthful eating by encouraging increased whole

plant food intake rather than dietary restriction and by focusing on general benefits of healthful eating rather than weight-related issues. Additionally, the family-based approach may have provided a stronger support system and more family connectedness, both of which are documented protective factors associated with DEB among adolescents (33).

The association of DEB with chronic hyperglycemia but not glycemic variability suggests that DEB may be related to intentional insulin omission to increase glycosuria and facilitate unsafe weight loss. It also suggests that hyperglycemia, not hypoglycemia or glycemic variability,



**Figure 1**—Nontransformed DEB for the treatment and control groups over the 18 months. DEB possible scores range from 0 to 80; higher scores indicate greater disordered eating. Error bars represent SDs of the DEPS-R mean scores from each time point. Results showed no significant differences using independent samples *t* tests on scores at baseline ( $n = 66$ ;  $t(64) = -1.34$ ,  $P = 0.18$ ), 6 months ( $n = 70$ ;  $t(68) = -0.48$ ,  $P = 0.64$ ), 12 months ( $n = 77$ ;  $t(75) = -1.57$ ,  $P = 0.12$ ), and 18 months ( $n = 81$ ;  $t(79) = -1.22$ ,  $P = 0.23$ ).

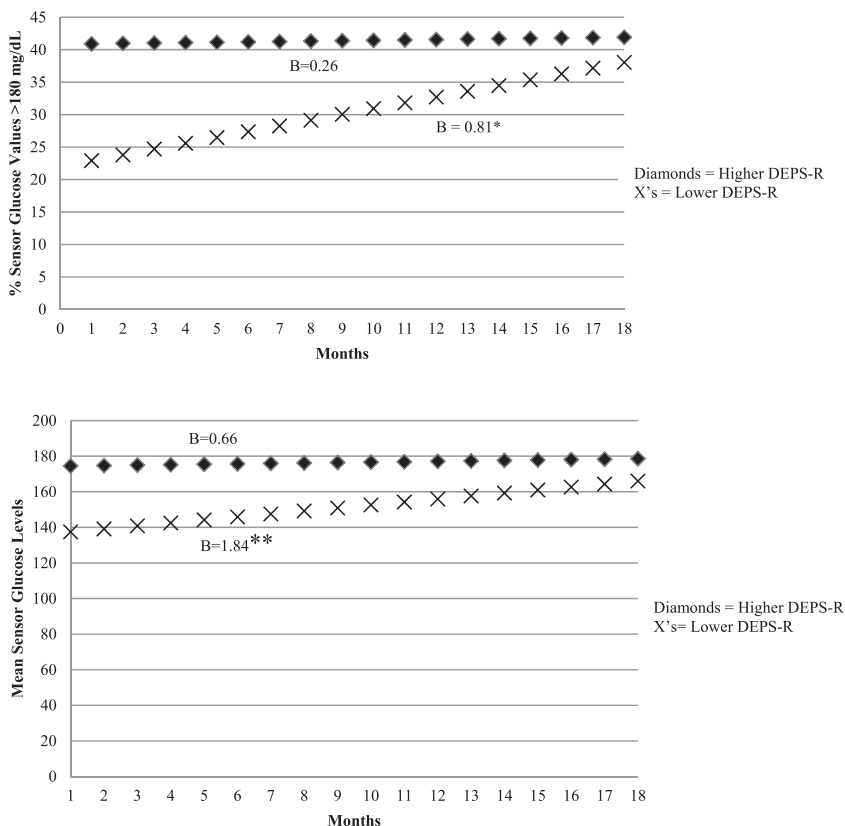
may be a better indicator of DEB or DEB risk, as has been suggested previously (34). This finding emphasizes the importance of using DEB measures tailored specifically to adolescents with type 1 diabetes, which capture DEBs related to insulin under dosing (4) rather than only

DEBs that are relevant to the general population, such as skipping meals or purging, which could result in hypoglycemia.

DEB may accelerate the deterioration in glycemic control commonly observed during adolescence (35,36). Participants with lower DEB scores

showed a developmentally expected increase in mean glucose across the 18-month study. However, mean sensor glucose for those with higher DEBs were already at hyperglycemic levels at baseline and remained so across the study. The finding that, even at the modest levels seen in this sample, DEB is associated with poorer glycemic control has important long-term clinical ramifications, since poor glycemic control in adolescence is associated with poor glycemic control in young adulthood (8) and an accelerated onset of long-term health complications (37). As such, identifying and reducing DEB in this population at a younger age may mitigate its adverse effects. Data from this sample show that among adolescents with type 1 diabetes, controlled motivation and low self-efficacy are risk factors for DEB (38), suggesting that autonomy-supportive care (i.e., emphasizing choice and rationale for action over pressure and coercive messages), which reduces controlled motivation and increases self-efficacy, could be effective in reducing DEB risk (39).

The findings should be interpreted in the context of study limitations. The sample excluded adolescents with very poor glycemic control and, therefore, may have excluded some youth with more severe DEBs. Additionally, participants in the sample were primarily white, drawn from a single institution, and only a limited number of participants were from low-socioeconomic status families; however, the sample included participants from all income levels, and the racial/ethnic breakdown was comparable with that of all patients with type 1 diabetes (40). Nonetheless, the highly selective sample, 24% participation rate, and family-based and highly resourced nature of the behavioral nutrition intervention limit generalizability. However, we are unaware of any evidence to suggest that sociodemographic characteristics would impact the association of DEB with glycemic control and variability or the impact of treatment assignment on DEB. Future studies could replicate the observational analyses in clinical samples that are not participating in a behavioral nutrition intervention. These factors, along with the 24% participation rate, limit generalizability. Another important limitation is that this intervention did not target DEB directly, so associations of DEB with glycemic outcomes are presented as



**Figure 2**—The degree of engagement in DEBs modifies the change in mean glucose levels and the percentage of blood glucose values >180 mg/dL. DEPS-R is an indicator of DEBs. Higher scores indicate more engagement in DEBs. \* $P < 0.05$ ; \*\* $P < 0.01$ .

observational analyses, which are susceptible to unmeasured confounding. Notable strengths include the prospective study design, which allows for the examination of the intervention effect on DEB and increases statistical power to test the effect of DEB on glycemic control. The use of a diabetes-specific DEB measure and the inclusion of multiple measures of glycemic control strengthen the internal validity of the findings. Examination of these questions in samples of patients with type 1 diabetes with more severe DEBs and/or poorer glycemic control would be useful. Future research could also examine which DEBs are most strongly associated with poorer glycemic control and whether individual intervention components are differentially associated with DEBs. Qualitative studies could be beneficial to obtain a more in-depth understanding of why youth with type 1 diabetes engage in DEBs.

This study demonstrated the feasibility of improving diet quality without adversely impacting DEB among adolescents with type 1 diabetes. Findings also showed an association of DEB with sustained hyperglycemia rather than glycemic variability or hypoglycemia. Finally, results demonstrate that even at the subclinical levels seen in this sample, DEB can hinder glycemic control during adolescence, emphasizing the importance of further research and early interventions to reduce these behaviors.

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**Author Contributions.** M.H.E.C. conceptualized and wrote the manuscript, ran the data analyses, and researched the literature. V.M.Q. researched the literature, contributed to the manuscript, and reviewed and edited the manuscript. L.M.L. contributed to the conceptualization of the manuscript and reviewed and edited the manuscript. K.W.D. researched the literature, contributed to the conceptualization of the manuscript, and reviewed and edited the manuscript. A.L. supervised and reviewed the statistical analyses and reviewed and edited the manuscript. L.M.B.L. collected the data and reviewed and edited the manuscript. S.N.M. collected the data and

reviewed and edited the manuscript. T.R.N. contributed to the conceptualization of the manuscript, oversaw data collection, and reviewed and edited the manuscript. T.R.N. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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