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Depressive Symptoms and Diabetes Management From Late Adolescence to Emerging Adulthood

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Objective: To examine changes in depressive symptoms as well as between- and within-person associations between depressive symptoms and Type 1 diabetes (T1D) management across the transition from late adolescence to emerging adulthood. ***Method:*** Beginning in the senior year of high school, 197 late adolescents with T1D (M_{age} = 17.77) reported on their student status and living situation, and completed self-report measures of depressive symptoms and adherence to the diabetes regimen, annually at 3 time points. Glycemic control was gathered from hemoglobin A1c (HbA1c) assay kits at the same time points. ***Results:*** Results of multilevel models demonstrated high depressive symptoms at baseline, with significant increases in depressive symptoms across time when participants were not living in their parental home, but no change when living with parents. Participants with higher mean levels of depressive symptoms relative to peers (between-person association) had poorer adherence and glycemic control (i.e., higher HbA1c) on average. Within-person fluctuations in depressive symptoms were significantly associated with adherence: greater increases in depressive symptoms (relative to adolescents' own average) were associated with greater deteriorations in adherence. There was not a significant within-person effect of depressive symptoms on glycemic control. ***Conclusions:*** The transition from late adolescence to emerging adulthood is particularly challenging for those with T1D. The findings that individuals with greater depressive symptoms have poorer adherence and glycemic control relative to those with lower depressive symptoms, and that increases in depressive symptoms are associated with declines in adherence, highlight the importance of screening and monitoring depressive symptoms during this life transition.

Keywords: diabetes, depression, adherence, glycemic control, adolescents

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Late adolescence is a vulnerable time during which depressive symptoms peak (Galambos, Barker, & Krahn, 2006; Saluja et al., 2004). This appears to be a particularly challenging time for those with chronic illness, as they demonstrate greater increases in depressive symptoms across adolescence, and slower decreases into emerging adulthood, relative to their physically healthy counterparts (Ferro, Gorter, & Boyle, 2015). Individuals with depressive symptoms during adolescence are more likely to experience a host of adverse outcomes 10 years later, including poor self-rated health and physical activity, recurrence of depressive episodes, and low levels of social support (Naicker, Galambos, Zeng, Senthilselvan, & Colman, 2013). Adolescents with Type 1 diabetes (T1D) report greater symptoms of depression relative to population norms (Johnson, Eiser, Young, Brierley, & Heller, 2013) and their healthy counterparts (Reynolds & Helgeson, 2011). Studies suggest that between 21% (Herzer & Hood, 2010) and 41.7% (Baucom et al., 2015) of adolescents with T1D have clinically significant symptoms of depression. Despite extensive examination of depressive symptoms in adolescents with T1D, there are few longitudinal studies focused on the transition to emerging adulthood, a time of high risk for diabetes management (Miller et al., 2015). The current study examines the degree to which both mean levels of (between-person) and fluctuations in (within-person) depressive symptoms are associated with adherence to the diabetes treatment regimen and glycemic control over the course of approximately 2 years from late adolescence to early emerging adulthood.

Diabetes management (i.e., adherence and glycemic control) deteriorates across the adolescent years (Datye, Moore, Russell, & Jaser, 2015; Miller et al., 2015), reaching a nadir during late

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adolescence and emerging adulthood (Hilliard, Herzer, Dolan, & Hood, 2011; Miller et al., 2015). The heightened risk of depressive symptoms may contribute to poor management during this developmental transition, given that individuals with greater depressive symptoms have poorer adherence and glycemic control. Greater depressive symptoms in adolescents with T1D are concurrently related to poorer adherence (Baucom et al., 2015; de Wit & Snoek, 2011; Korbel, Wiebe, Berg, & Palmer, 2007; McGrady & Hood, 2010) and glycemic control (e.g., Baucom et al., 2015; de Wit & Snoek, 2011; Johnson et al., 2013; McGrady, Laffel, Drotar, Repaske, & Hood, 2009). Further, data from a longitudinal study of 13 to 18-year-olds with T1D demonstrated between-person effects of depressive symptoms on diabetes management trajectories. Specifically, adolescents with higher levels of baseline depressive symptoms had greater likelihood of membership in a latent class with poorer adherence and glycemic control maintained over the course of several years (Hilliard, Wu, Rausch, Dolan, & Hood, 2013). Based on a wealth of research demonstrating negative associations between depressive symptoms and key diabetes outcomes across the life span, the American Diabetes Association (ADA) recommended that symptoms of depression be assessed both initially and at periodic intervals (including at a change in life circumstance) in individuals with diabetes (Young-Hyman et al., 2016).

Despite strong support that individuals with greater depressive symptoms display poorer concurrent levels of T1D management, there have been few longitudinal studies examining whether within-person fluctuations in depressive symptoms are associated with fluctuations in diabetes management across time. One study found that within-person increases in adolescents' depressive symptoms over the course of 6 months were related to concurrent declines in glycemic control (Hood, Rausch, & Dolan, 2011). However, longitudinal studies of younger adolescents and emerging adults did not demonstrate such associations across time. Specifically, neither a 5-year longitudinal study of younger adolescents with T1D (baseline age $M = 13.7$ years, $SD = 2.5$, range 13–18 years; Hood et al., 2014) nor a 5-year longitudinal study of emerging adults with T1D (Rassart et al., 2015; baseline age $M = 23.48$ years, $SD = 3.7$, range 18–30 years) found significant within-person associations between fluctuations in depressive symptoms and changes in hemoglobin A1c (HbA1c).

More important, no studies have examined associations between within-person fluctuations in depressive symptoms and diabetes management *across the transition from late adolescence to emerging adulthood*. Arnett (2000) defined emerging adulthood as the developmental period between adolescence and young adulthood (ages 18–25) that is characterized by a "high degree of demographic diversity and instability," including changes in living and educational situations. These unique experiences may contribute to fluctuations in both depressive symptoms and diabetes management during this transition (Monaghan, Helgeson, & Wiebe, 2015; Weissberg-Benchell, Wolpert, & Anderson, 2007; Wysocki, Hough, Ward, & Green, 1992). A limited number of studies have begun to examine the impact of some of these developmental experiences on depressive symptoms and diabetes management. For example, not enrolling in college was related to change from low to high levels of depressive symptoms in healthy individuals across the transition from adolescence to young adulthood (Lee, Wickrama, Kwon, Lorenz, & Oshri, 2017). In a study of emerging adults with T1D who were transitioning to college, there were decreases in clinic attendance and increases in body mass index—but no change in HbA1c or blood glucose monitoring—from precollege to college enrollment (Monaghan, King, Alvarez, Cogen, & Wang, 2016). Despite initial work in this area, there are not conclusive findings in individuals with T1D (Monaghan et al., 2015).

Heightened risk for both depressive symptoms and poor diabetes management during this developmental period, in combination with the positive long-term prognosis associated with early intervention for mood disorders (McGorry, Purcell, Goldstone, & Amminger, 2011), demonstrate the important clinical implications of a thorough longitudinal examination of the associations between depressive symptoms and diabetes management across the transition to emerging adulthood. Understanding the extent to which effects are between-versus within-person could inform future intervention research in identifying whether interventions should be targeted to those experiencing high depressive symptoms (if the effects are only between-person) or administered more broadly to late adolescents and emerging adults with T1D (if there are within-person effects).

Current Study

The present study examined changes in depressive symptoms as well as between-person and within-person associations between depressive symptoms and T1D management across approximately 2 years from late adolescence to early emerging adulthood. We used multilevel modeling to simultaneously examine within- and between-person variability in depressive symptoms as they relate to diabetes adherence and glycemic control. We expected that depressive symptoms would increase from late adolescence to emerging adulthood because of the many changes that occur during this time. Further, we expected that diabetes management would be poorer for those with higher mean levels of depressive symptoms relative to peers (between-person effect), and at times when depressive symptoms were higher than an individual's own mean (within-person effect).

Method

Participants

Participants in the current study were 197 high school seniors with T1D recruited for a 2-year longitudinal study on diabetes and self-regulation during late adolescence and emerging adulthood. Participants were recruited from three outpatient pediatric endocrinology clinics in two southwestern U.S. cities (i.e., Salt Lake City, UT and Dallas, TX) by a research assistant in clinic, or by mail and phone. Of the qualifying 507 individuals approached, 301 (59%) initially agreed to participate. Reasons for not participating included lack of interest (44.2%), being too busy in their senior year to participate (34.0%), and 21.8% declined to give a reason. Of those who agreed to participate, 247 adolescents (82%) completed the initial laboratory assessment. Data in the current study were from a subsample of late adolescents/emerging adults (66% female) who completed survey measures at each of the first three study time points. Participants who had invalid data at the initial laboratory assessment ($n = 6$) or who did not complete the survey

portion of the study at any time point ($n = 5$) were excluded from the study. Further, given the examination of within-person variability in the current study, we also excluded participants who completed only one ($n = 17$) or two ($n = 22$) of the three time points. Ten of the participants who completed two of the three time points had complete survey data at both points; therefore, we conducted sensitivity analyses (i.e., reran models described below with the addition of these 10 participants). The results were not substantively different from those reported below.

Eligibility criteria included being diagnosed with T1D for at least 1 year (years since diagnosis $M = 7.45$, $SD = 3.85$), 17 to 18 years of age ($M = 17.77$, $SD = 0.39$), enrolled in their last year of high school, living in their parental home, not planning to participate in a program or activity that restricted daily contact with parents during the study, and primary English speakers (because of requirements of cognitive testing in the larger study). Participants who did not report they were enrolled in high school were eligible if they met all other qualifying criteria ($n = 2$ [1%]). Participants' baseline glycemic control on average did not meet the recommended target for children and adolescents of HbA1c levels below 7.5% (ADA, 2017; $M = 8.13$, $SD = 1.60$); only 38.1% of participants met this target. Additionally, 43.7% of participants used an insulin pump at baseline. Consistent with the patient population at participating clinics, the sample included in the current study was 76.0% non-Hispanic White, 14.0% Hispanic, 5.7% African American, 2.0% Asian/Pacific Islander, and 2.1% American Indian. A range of parent education was reported, with 20.9% of mothers and 26.9% of fathers having no more than a high school education, 35.8% of mothers and 22.3% of fathers having some college without a bachelor's degree, and 42.9% of mothers and 46.8% of fathers having a bachelor's degree or higher.

At the second and third study time points (described in more detail below), nearly half of participants were living in their parents' home ($n = 98$ [49.7%] at Time 2 and $n = 90$ [45.7%] at Time 3) and most were full-time students at 2- or 4-year colleges or technical schools ($n = 127$ [64.5%] at Time 2 and $n = 103$ [52.3%] at Time 3). The minority of participants were working full-time at Time 2 ($n = 29$; 14.7%) and Time 3 ($n = 45$; 22.8%). Most participants reported they transitioned from pediatric to adult diabetes care after the Time 1 assessment ($n = 105$ [54.4%] before Time 2, $n = 139$ [71.6%] before Time 3).

Procedure

Data are from three waves of a longitudinal study of late adolescents with T1D. All study procedures were approved by Institutional Review Boards at the University of Utah and the University of Texas Southwestern Medical Center at Dallas where participants were recruited, and at the University of California, Merced, where follow-up assessments of the Texas sample occurred. Adolescents and their parents provided written informed assent and consent. During the initial laboratory session, as part of the larger study, adolescents completed a battery of neurocognitive and behavioral measures, as well as an HbA1c home test kit, and were trained on online survey measures to be completed at home. Data for the present primary study variables were gathered at each of three time points: during senior year of high school (Time 1; late adolescence), and on two additional occasions (Time 2 and Time 3; emerging adulthood). Measurements were approximately 1 year apart. Data analyzed in the current study included data from an online survey and HbA1c test kits completed at each time point.

Measures

Depressive symptoms. Participants completed the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), a 20-item self-report measure of symptoms of depression over the past week. Items were rated on a scale from 0 (*rarely or none of the time*) to 3 (*most or all of the time*), with higher scores indicating greater depressive symptoms. CES-D items demonstrate good internal consistency (excellent in the current sample, $\alpha = .94$ at Time 1, .91 at Time 2, and .94 at Time 3), moderate test–retest reliability, and correlations with longer clinical measures of depression (Radloff, 1977). Although we examined the CES-D as a continuous measure of depressive symptoms, the sample of adolescents included 115 (58.4%) in the minimal range (composite score of 0 to 15), 33 (16.8%) in the mild range (16 to 23), and 47 (23.9%) in the moderate/severe range (24 to 60) at Time 1 based on established categories of clinical significance (Lawrence et al., 2006). We did not have complete data on $n = 2$ participants (1%). These rates of clinically significant symptoms are substantially higher than those reported in other samples of adolescents with T1D (e.g., Herzer & Hood, 2010).

Adherence. The revised Diabetes Behavior Rating Scale (DBRS; Iannotti et al., 2006) was used to measure adherence. The DBRS is a 37-item scale that assesses adherence to the regimen of behaviors required for diabetes management, including components of problem-solving (e.g., adjusting insulin as a function of food or exercise). This measure correlated well with both interview measures of adherence and HbA1c in adolescents in the initial validation study (Iannotti et al., 2006). Consistent with previous work (Iannotti et al., 2006), DBRS scores were computed as the proportion of the maximum possible score (range = 0.06–1.00), with higher scores representing better adherence to the T1D treatment regimen. DBRS items had good internal consistency in the current study ($\alpha = .83$ at Time 1, .85 at Time 2, and .88 at Time 3).

Glycemic control. Glycemic control was measured using HbA1c assay kits obtained from and processed by CoreMedica Laboratories, which is accredited by the College of American Pathologists (www.coremedica.net). Test kit data were collected in addition to point-of-care assays in participants' medical records to allow for examination of changes in HbA1c over time for those who did not regularly attend medical appointments. At Time 1, this measure was highly correlated with HbA1c from medical records, $r = .77$, $p < .001$.

Data Analysis

Handling missing data. Items were missing on 3% of the sample of 197 adolescents at Time 1 (HbA1c test kit results from one participant, CES-D items from two participants, and DBRS items from five participants), 8% at Time 2 (HbA1c test kit results from 12 participants, CES-D items from two participants, and DBRS items from five participants), and 9.6% at Time 3 (HbA1c test kit results from 13 participants, CES-D items from 7 participants, and DBRS items from 8 participants), because of participants failing to answer all items. To account for missing data, we

generated five data sets via multiple imputations in SPSS. We included variables beyond those included in the current study during the imputation procedure to handle the "missing at random" model adequately. Data for an individual were not imputed if they were missing all data at a particular time point (see Participants for full sample of larger study and general reasons for missing data). The final reported results were pooled from the results of each imputed dataset. The lowest efficiency was .922 across all analyses, indicating that the multiple imputations adequately recovered the missing data (Rubin, 1987).

Variance partitioning. To examine between-versus within-person variance in outcomes for our hypotheses, we calculated intraclass correlation coefficients (ICCs) from variance estimates obtained from unconditional (empty) models in Hierarchical Linear and Nonlinear Modeling (HLM 7; Raudenbush, Bryk, Cheong, Congdon, & du Toit, 2011). ICCs indicated that 63% of variance in adherence, 46% of variance in HbA1c, and 48% of variance in depressive symptoms was between-persons.

Covariates. The following between-person (Level 2) covariates were included in models: gender (−0.5 = *male*, 0.5 = *female*), study site (−0.5 = *Texas*, 0.5 = *Utah*), initial pump status (−0.5 = *not using pump*, 0.5 = *using pump*), and years since T1D diagnosis (grand mean centered). Two within-person (Level 1) covariates were also included: living in parental home (−0.5 = *no*, 0.5 = *yes*) and student status (−0.5 = *not a full-time student*, 0.5 = *full-time student*).

Change in depressive symptoms over time. Change in depressive symptoms across the three time points was examined with a linear growth model computed in HLM (shown below).

Level 1 (within-person) equation:

$$\text{Depressive Symptoms}_{ti} = \pi_{0i} + \pi_{1i}(\text{Time})_{ti} + r_{ti}$$

Level 2 (between-person) equation:

$$\pi_{0i} = \beta_{00} + r_{0i}$$

$$\pi_{1i} = \beta_{10} + r_{1i}$$

We included random effects on the intercept and slope given the between-person variance estimate (ICCs) for depressive symptoms. Time was coded 0 (*Time 1*), 1 (*Time 2*), and 2 (*Time 3*), such that the intercept (β_{00}) represented mean depressive symptoms at Time 1. To account for the possibility that changes in depressive symptoms over time depended on the covariates included in the model (described above), we included interactions between time and each of the covariates in a preliminary model. The final model included only the Time × Living in Parental Home interaction (no other interactions with time were significant [*p*s > .05], so other interaction terms were dropped from the final model).

Predicting adherence and glycemic control from depressive symptoms. We estimated within- and between-person effects of depressive symptoms using a series of multilevel models in HLM. We also examined whether there were *differences* between within-person and between-person effects of depressive symptoms on outcomes by testing contextual (also known as compositional) effects (Raudenbush & Bryk, 2002) with hypothesis testing in HLM. We followed the procedures detailed in Baldwin, Wampold, and Imel (2007) with the equations below; identical models were computed for adherence and glycemic control. Data from all three time points were included in these models, with adherence and HbA1c at each time point predicted from depressive symptoms at each time point.

Level 1 (within-person) equation:

$$\text{Adherence}_{ti} = \pi_{0i} + \pi_{1i}(\text{Depressive Symptoms})_{ti} + r_{ti}$$

Level 2 (between-person) equation:

$$\pi_{0i} = \beta_{00} + \beta_{01}(\text{Mean Depressive Symptoms})_i + r_{0i}$$

$$\pi_{1i} = \beta_{10}$$

Depressive Symptoms was group (person) mean centered at Level 1, and Mean Depressive Symptoms at Level 2 represents each person's average CES-D score across the three time points, which was grand mean centered. To account for the possibility that associations between diabetes management and fluctuations in depressive symptoms depended on covariates, we included interactions of each of the covariates with fluctuations in depressive symptoms in preliminary models. As no interactions were statistically significant (*p*s > .05), the final model included only main effects of covariates.

The intercept (β_{00}) represents predicted adherence for a person with average depressive symptoms relative to peers, when the person's symptoms were at his or her mean. The slope (β_{10}) represents the within-person depressive symptoms effect (i.e., change in adherence given a one-unit increase in depressive symptoms from an individual's mean), and β_{01} represents the between-person depressive symptoms effect (i.e., change in adherence for individuals whose average depressive symptoms are one-unit higher than the sample mean). The contextual effect, which is computed by subtracting the within-person coefficient from the between-person coefficient (β_{01}–β_{10}), is the expected difference in adherence between individuals with the same depressive symptoms score but who differ by one unit in their mean depressive symptoms score (Baldwin et al., 2007). A significant contextual effect indicates that there is a significant difference between the within- and between-person effects of depressive symptoms on adherence.

Random effects were not included on the slopes in the models, as there was no evidence that slopes varied across individuals in preliminary models (variance = 0.000, $p = .354$ and variance = 0.000, $p = .357$ in glycemic control and adherence models, respectively). We also did not include a Depressive Symptoms × Mean Depressive Symptoms interaction term, as cross-level interactions were not significant in preliminary models ($B = 0.001$, $SE = 0.001$, $p = .509$ and $B = 0.001$, $SE = 0.001$, $p = .437$ in the prediction of HbA1c and adherence, respectively).

As the distribution of glycemic control was negatively skewed, we predicted log-transformed glycemic control (Tabachnik & Fidell, 2013). The pattern of results did not differ; untransformed scores were, therefore, used for ease of interpretation. The results of the log-transformed models are available in Table 1 of the online supplemental materials.

Results

Preliminary Analyses

Table 1 presents descriptive statistics and correlations among variables at each time point. As expected, greater depressive

Table 1
Descriptive Statistics and Correlations Among Variables Over Time

Variable	*M* (*SD*)	1	2	3	4	5	6	7	8	9
1. T1 HbA1c	8.13 (1.59)	—								
2. T1 adherence	.61 (.12)	−.23**	—							
3. T1 depressive symptoms	16.32 (12.26)	.21**	−.34***	—						
4. T2 HbA1c	8.96 (1.88)	.50***	−.25**	.23**	—					
5. T2 adherence	.59 (.13)	−.19**	.70***	−.25***	−.25**	—				
6. T2 depressive symptoms	15.45 (10.53)	.01	−.27***	.55***	.20**	−.31***	—			
7. T3 HbA1c	9.12 (2.00)	.48***	−.19*	.17*	.70***	−.22**	.04	—		
8. T3 adherence	.59 (.15)	−.27***	.59***	−.18*	−.26***	.69***	−.24**	−.27**	—	
9. T3 depressive symptoms	18.08 (13.49)	.05	−.24**	.44***	.22**	−.29***	.53***	.10	−.30***	—

Note. HbA1c = hemoglobin A1c; T1 = Time 1; T2 = Time 2; T3 = Time 3. All statistics are based on raw data (before imputation). *n*s range from 184 to 196 depending on the measure and time point.
* $p < .05$. ** $p < .01$. *** $p < .001$.

symptoms were related to poorer adherence and glycemic control at Time 1 and Time 2; however, correlations between depressive symptoms and glycemic control were smaller in magnitude and less consistent across time than correlations between depressive symptoms and adherence. Depressive symptoms were correlated with adherence, but not glycemic control, at Time 3.

Change in Depressive Symptoms Over Time

Table 2 presents results of the linear growth model for depressive symptoms. We conducted post hoc hypothesis tests in HLM to decompose the significant Time × Living in Parental Home interaction. When participants were not living with their parents, they demonstrated a significant increase in depressive symptoms over time ($B = 4.756$, $SE = 1.802$, $\chi^2 = 6.959$, $p = .008$), whereas when they were living with their parents, they did not demonstrate significant change in depressive symptoms ($B = -1.864$, $SE = 1.395$, $\chi^2 = 1.785$, $p = .178$). Random effects on the intercept and slope were statistically significant, indicating between-person variability in baseline levels of (intercepts) as well as changes in (slopes) depressive symptoms over time, even after including multiple covariates.

Table 2
Linear Change in Depressive Symptoms Over Time

Fixed effects	*B* (*SE*)	*p*
Intercept (β_{00})	14.564 (1.264)	<.001
Gender (β_{01})	.413 (1.503)	.563
Site (β_{02})	−3.486 (1.516)	.023
Initial pump status (β_{03})	−.886 (1.530)	.563
Years since diagnosis (β_{04})	−.073 (.185)	.692
Time (β_{10})	1.446 (.744)	.053
Parental home (β_{20})	3.438 (1.835)	.063
Student status (β_{30})	−1.001 (1.187)	.400
Time × Parent Home (β_{40})	−3.352 (1.413)	.019
Random effects	**Variance**	***p***
Intercept (r_0)	86.898	<.001
Time slope(r_1)	20.972	<.001
Level 1 residual (e)	55.070	

Note. Coding of Level 2 predictors was: Gender (−.5 = *male*, .5 = *female*), site (−.5 = *Texas*, .5 = *Utah*), Initial pump status (−.5 = *not using pump*, .5 = *using pump*), and years since diagnosis (grand mean centered). Coding of Level 1 predictors was: Time (0 = *Time 1*, 1 = *Time 2*, 2 = *Time 3*), living in parental home (−0.5 = *no*, .5 = *yes*), and student status (−.5 = *not a full-time student*, .5 = *full-time student*).

Predicting Adherence and Glycemic Control From Depressive Symptoms

Table 3 presents results of tests of within- and between-person effects of depressive symptoms on T1D outcomes. There were significant between-person effects of depressive symptoms on both adherence and HbA1c. As expected, individuals who reported more depressive symptoms demonstrated poorer glycemic control (i.e., higher HbA1c) and lower adherence relative to those who reported less depressive symptoms. There was also a significant within-person effect of depressive symptoms on adherence, such that when an individual's depressive symptoms were higher than their own average, their adherence was lower. There was not a significant association between within-person fluctuations in depressive symptoms and fluctuations in HbA1c.

We examined whether the within- and between-person effects of depressive symptoms on T1D outcomes were significantly different from one another (i.e., significant contextual effects; Baldwin et al., 2007). There was a significant contextual effect of depressive symptoms on adherence ($B = 0.003$, $SE = 0.001$, $p = .009$) but not on glycemic control ($B = 0.020$, $SE = 0.014$, $p = .162$). For two participants with the same CES-D score at a given time point, but whose mean CES-D scores differ by 1 *SD*, the difference in DBRS is 0.026 (unstandardized contextual effect regression coefficient [.003] × CES-D *SD* [9.79]), 19.4% of a *SD* in adherence. Despite the same CES-D score at a given time point, expected adherence scores are quite different. See supplemental materials for additional description and illustration of the contextual effect in the context of these results.

Discussion

This study examined depressive symptoms in individuals with T1D across the transition from late adolescence to emerging adulthood. Analysis examining linear changes in depressive symptoms across the transition revealed that depressive symptoms significantly increased across approximately 2 years, but only in adolescents who were not living with their parents. In examining the

Table 3
Longitudinal Prediction of Adherence and Glycemic Control From Depressive Symptoms

Fixed effects	Outcome: Adherence			
	B	*SE*	*p*	SC
Intercept (β_{00})	.590	.010	<.001	
Between-person depressive symptoms (β_{01})	−.004	.001	<.001	−.316
Gender (β_{02})	.016	.017	.356	.056
Site (β_{03})	.016	.016	.328	.059
Initial pump status (β_{04})	.011	.015	.432	.043
Years since diagnosis (β_{05})	−.001	.002	.923	−.005
Within-person depressive symptoms (β_{10})	−.002	.001	.001	−.162
Living in parental home (β_{20})	.014	.009	.128	.049
Student status (β_{30})	.010	.010	.336	.033
	Outcome: Glycemic control			
	B (*SE*)	*SE*	*p*	SC
Intercept (β_{00})	9.009	.131	<.001	
Between-person depressive symptoms (β_{01})	.028	.011	.009	.143
Gender (β_{02})	−.429	.233	.067	−.105
Site (β_{03})	−.166	.231	.474	−.043
Initial pump status (β_{04})	−.739	.211	<.001	−.191
Years since diagnosis (β_{05})	.029	.026	.272	.058
Within-person depressive symptoms (β_{10})	.009	.010	.366	.056
Living in parental home (β_{20})	−.484	.146	<.001	−.120
Student status (β_{30})	−.797	.197	<.001	−.186

Note. SC = standardized regression coefficient (calculated as unstandardized coefficient times *SD* of predictor over *SD* of outcome), which represents the predicted change in outcome (in *SD*s) given a 1 *SD* increase in predictor. Coding of Level 2 predictors was: Gender (−.5 = *male*, .5 = *female*), site (−.5 = *Texas*, .5 = *Utah*), initial pump status (−.5 = *not using pump*, .5 = *using pump*), years since diagnosis (grand mean centered). Coding of Level 1 predictors was: Time (0 = *Time 1*, 1 = *Time 2*, 2 = *Time 3*), living in parental home (−.5 = *no*, .5 = *yes*), and student status (−.5 = *not a full-time student*, .5 = *full-time student*).

relationship between depressive symptoms and T1D management, we simultaneously examined the effects of between-person differences and within-person fluctuations in depressive symptoms on diabetes adherence and glycemic control across this important developmental period. Consistent with hypotheses, higher levels of depressive symptoms on average (between-person) were associated with poorer adherence and glycemic control, and within-person increases in depressive symptoms were associated with decreases in adherence. There was a significant contextual effect of depressive symptoms on adherence, indicating an incremental effect on adherence of being a more depressed person over and above the within-person effect of greater-than-usual depressive symptoms.

When participants were *not* living in their parental home, CES-D scores on average increased nearly five points per year. It is possible that those living at home received more family support, which buffered against increases in depressive symptoms. Studies have found the buffering effect of parental support on depressive symptoms to be particularly strong early in the transition from adolescence to emerging adulthood (e.g., Meadows, Brown, & Elder, 2006). Alternatively, individuals who were more depressed may have been more likely to move out of their parental home. There appear to be other factors impacting trajectories of depressive symptoms in our sample, as even with a number of covariates included, random effect estimates demonstrated significant variance between individuals in both baseline levels of depressive symptoms (intercept) and change in depressive symptoms over time (slope).

Despite no significant change in depressive symptoms for emerging adults when living in their parental home, participants' baseline (Time 1) depressive symptoms were on average substantially higher than other samples of adolescents with T1D (e.g., Herzer & Hood, 2010; Hood et al., 2014), and symptoms remained high across the three time points. The average CES-D score at baseline (16.67) is above the cutoff for clinically significant depressive symptoms. Thus, although CES-D scores did not significantly increase over time when emerging adults were living with their parents, mean levels of depressive symptoms were high at baseline and remained high across this developmental transition. It is possible that the increases in depressive symptoms we expected for the sample as a whole occurred before enrollment in the study, consistent with the finding that depressive symptoms peak at age 16–17 (Ferro et al., 2015).

The hypothesis that between-person differences and within-person fluctuations in depressive symptoms would predict adherence and glycemic control was partially supported. Significant between-person effects were found across outcomes: participants with higher mean levels of depressive symptoms reported poorer adherence and had poorer glycemic control relative to peers. There was also a significant association between within-person fluctuations in depressive symptoms and adherence: across levels of mean depressive symptoms, adolescents reported poorer adherence when their depressive symptoms were higher than their average level. In contrast, there was not a significant association between within-person fluctuations in depressive symptoms and glycemic control. Despite our findings that living in the parental home moderated

changes in depressive symptoms over time, neither student status nor living in the parental home significantly interacted with mean levels of, or fluctuations in, depressive symptoms in the prediction of diabetes outcomes. Whereas previous work has demonstrated that adolescents' baseline depressive symptoms predict membership in a group with subsequent deteriorations in adherence and glycemic control (Hilliard et al., 2013), ours is the first known study to examine repeated measurements of depressive symptoms across the transition from late adolescence to emerging adulthood.

Our findings highlight the utility of current clinical recommendations for late adolescents and emerging adults with T1D in particular. The within-person effect of increases in depressive symptoms on declines in adherence supports the ADA recommendation that fluctuations in depressive symptoms be carefully monitored over major life transitions and transitions in diabetes care (Young-Hyman et al., 2016). The transition from late adolescence to emerging adulthood is a particularly high-risk time given the likelihood of increases in depressive symptoms (Galambos et al., 2006) and poor management of T1D (Miller et al., 2015). In addition to the vulnerability of this life stage, individuals with T1D often transition to adult diabetes care providers during this time as well (Weissberg-Benchell et al., 2007). The significant contextual effect (i.e., the finding that the between-person effect was significantly larger than the within-person effect) suggests that monitoring of fluctuations in depressive symptoms may be most clinically relevant for individuals who have higher average levels of depressive symptoms. For these individuals, increases in depressive symptoms above their already elevated average levels may be particularly risky for diabetes management. Consistent with this, ADA recommendations emphasize the particular importance of routine monitoring of depressive symptoms in individuals with a history of depression (Young-Hyman et al., 2016).

These results should be interpreted in the context of several limitations. First, method variance may play a role in our findings, as depressive symptoms and adherence were both measured with self-reports. Adolescents with greater depressive symptoms may have more negative views of their adherence behavior, a possible explanation for the finding that within-person fluctuations in depressive symptoms significantly predicted adherence but not glycemic control. Future work would benefit from the examination of multiple measures of adherence, including objective measures (e.g., blood glucose monitoring frequency as obtained from glucometers) that have been utilized in similar work (e.g., Hilliard et al., 2013). Second, we did not examine the mechanisms for these associations, which may be bidirectional. Work that includes biomarkers of inflammation would provide important information on the potential physiological interaction of depression and diabetes outcomes (Downs & Faulkner, 2015; Holt, de Groot, & Golden, 2014). Third, the current study could only model linear change in depressive symptoms because of the number of time points available (three); examination of these variables over four or more time points would allow for examination of quadratic change over time. Finally, the characteristics of our sample limit the generalizability of findings. Although the sample is representative of individuals with T1D in the United States in terms of ethnicity, the sample was primarily White and well-educated. These results may not generalize to populations with greater racial diversity or to other age groups. Further, although almost all participants were high school students at Time 1, they were very late in the adolescent period and were followed for only 2 years. The field would benefit from future work that enrolls participants at a younger age and follows them over a longer period of observation to fully capture the changes that occur over this transition period in both depressive symptoms and diabetes management. These future directions would be instructive for not only those with T1D, but also for those with Type 2 diabetes (T2D).

Summary and Implications

This study examined depressive symptoms, diabetes adherence, and glycemic control during a major life transition in individuals coping with T1D. The stressful transition from late adolescence to emerging adulthood is a crucial time to examine predictors of diabetes management and control as health behavior during this time sets the stage for later adult health (Wysocki et al., 1992). Given the challenges with diabetes management during this life stage (Datye et al., 2015), the finding that depressive symptoms predicted adherence and glycemic control provides strong support for careful screening and monitoring of depressive symptoms in the context of clinical diabetes care. Future technological advances in glucose monitoring may allow for the integration of monitoring of depressive symptoms and other conditions that place individuals with T1D at risk for declines in diabetes management. The field would benefit from future work that incorporates a focus on factors that protect against depression during this transition. Protective factors such as resilience (Anyan & Hjemdal, 2016) and social support (Rao, Hammen, & Poland, 2010), as well factors that may place individuals with T1D at heightened risk for depression should be examined. Diabetes-specific distress, which has longitudinal associations with depressive symptoms (Ehrmann, Kulzer, Haak, & Hermanns, 2015) and mediates concurrent associations between depressive symptoms and glycemic control (van Bastelaar et al., 2010) in adults with T1D may provide early signs of who is at risk for heightened depressive symptoms or when symptoms are likely to increase from an individual's current level of depression. Finally, given the increasing prevalence of T2D in adolescents and young adults (Lascar et al., 2018), and the higher likelihood of development of the condition in those who report clinically significant depressive symptoms in adolescence and emerging adulthood (Suglia, Demmer, Wahi, Keyes, & Koenen, 2016), it would be useful to examine associations between depressive symptoms and diabetes outcomes in those with T2D.

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