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Depressive Symptoms in Adolescent Girls At-Risk for Type 2 Diabetes and Their Parents

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Abstract

Few studies have characterized the relation between parent's depression symptoms and adolescent's depression symptoms in adolescents at-risk for type 2 diabetes (T2D). We evaluated the associations of parental depression symptoms with the depression symptoms and metabolic functioning of adolescent offspring at-risk for T2D. One-hundred sixteen parents and adolescent girls with a family history of diabetes completed surveys of depression symptoms. Adolescents' degree of metabolic risk for T2D was estimated from body mass index (BMI; kg/m^2) standard score, percent adiposity from dual-energy x-ray absorptiometry scan, and whole body insulin sensitivity index determined from glucose/insulin concentrations during a two-hour oral glucose tolerance test. Parents' and adolescents' depression symptoms were significantly associated, even after accounting for race/ethnicity, age, puberty, body composition, and parental diabetes/BMI. Adjusting for similar covariates, parent depression symptoms also were positively related to adolescents' BMI standard score and had a trend-level association with adiposity. There was an inverse relation between parental depression symptoms and adolescent insulin sensitivity, which

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was entirely accounted for by adolescent body composition. The associations of parental depression symptoms with more elevated depression symptoms and higher BMI in adolescents at-risk for T2D has potential implications for interventions addressing these co-morbid health conditions.

Keywords

adolescents; parents; depression; type 2 diabetes; obesity

Type 2 diabetes (T2D) is a widespread, chronic illness that causes serious health complications and a shortened lifespan (American Diabetes Association, 2018). Approximately 40% of adults are expected to develop T2D in their lifetime, with even higher rates (>50%) among individuals who are non-Hispanic Black/African American and Hispanic/Latino (Gregg et al., 2014). Historically a disease of middle and later adulthood, there has been an alarming rise in the incidence of T2D among younger groups (age 10–19 years), which account for 20–50% of new-onset diabetes in this age group (Mayer-Davis et al., 2017). Youth-onset (<20 years) T2D may have a particularly aggressive disease course, carrying a greater risk of health comorbidities and earlier mortality as compared to adult-onset T2D (Nadeau et al., 2016). Of note, youth-onset T2D is far more common in girls than boys, and it disproportionately affects racial/ethnic minority youth (Mayer-Davis et al., 2017).

In younger individuals, having overweight or obesity (body mass index [BMI] ≥85th percentile for age and sex) and having family members with T2D each significantly increase the risk of reduced insulin sensitivity (the ability of the hormone insulin to remove blood sugar from the blood stream by causing it to enter body tissues). Low insulin sensitivity is a major precursor for T2D (Morrison, Glueck, Horn, & Wang, 2010). In addition to these traditional risk factors, there may be modifiable, psychosocial factors that play a role in the worsening of insulin sensitivity and risk for T2D. Depression symptoms, in particular, have been associated with poorer insulin sensitivity and T2D onset in adolescents and adults (Hannon, Rofey, Lee, & Arslanian, 2013; Jaser, Holl, Jefferson, & Grey, 2009; Rotella & Mannucci, 2013; Shomaker & Goodman, 2015; Shomaker et al., 2010; Suglia, Demmer, Wahi, Keyes, & Koenen, 2016). In healthy adolescents and those at-risk for T2D, cross-sectional investigations show that depression symptoms are inversely related to insulin sensitivity, even after accounting for body composition (Hannon et al., 2013; Shomaker et al., 2010). Further, depression symptoms have been associated prospectively with worsening of insulin sensitivity (Shomaker et al., 2011), higher fasting insulin and sustained hyperinsulinemia into young adulthood (Shomaker & Goodman, 2015), and a two-fold greater odds of T2D onset in young adulthood (Suglia et al., 2016). These adolescent/young adult findings are consistent with extant adult data illustrating a cross-sectional relation between depression symptoms and poor insulin sensitivity, as well as longitudinal evidence that adults' depression symptoms predict the onset of T2D (Rotella & Mannucci, 2013).

Although the association between parental depression symptoms and adolescent depression symptoms is well supported in community samples (Agerup, Lydersen, Wallander, & Sund,

2014), little research has characterized this association in families at-risk for T2D, in spite of the knowledge that depression symptoms may be a psychosocial contributor to T2D risk and management (Kong & Chan, 2015; Rotella & Mannucci, 2013). In adolescents with T2D, as well as those with type 1 diabetes, parents' elevated depression symptoms are prevalent (>20%) and positively associated with adolescent depression symptoms (Eckshtain, Ellis, Kolmodin, & Naar-King, 2010; Weinstock et al., 2015). Understanding the relation of parental depression symptoms to the depression symptoms and metabolic risk status of adolescents at-risk for T2D is needed, as such knowledge may shed light on the potential value of family-based, psychosocial approaches to T2D prevention.

The primary aim of the current study was to evaluate the associations of parental depression symptoms with the depression symptoms and degree of metabolic risk for T2D in adolescent offspring identified as at-risk for T2D. Based upon past research (Eckshtain et al., 2010; Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002; Weinstock et al., 2015), we hypothesized that there would be a significant association between parental and adolescent depression symptoms, even after accounting for potentially confounding factors including adolescent body composition, parental BMI, and parental T2D status. The second objective was to investigate if parental depression symptoms related to adolescents' metabolic risk for T2D, as reflected in higher BMI, higher adiposity, and lower insulin sensitivity. We hypothesized that parental depression symptoms would be related to adolescents' higher BMI/adiposity and poorer insulin sensitivity.

Materials and Methods

Participants

The current study is a secondary data analysis of girls ages 12–17 years taking part in the baseline phase of a T2D behavioral prevention trial (ClinicalTrials.gov: NCT01425905). Main outcomes have been published (Shomaker et al., 2016; Shomaker, Kelly, et al., 2017); there have been no prior publications with measures of parental depression from this cohort. Adolescents were included if they were female and determined to be at-risk for T2D by having a BMI ≥85th percentile for age and having ≥1 first- or second-degree family member with T2D, pre-diabetes, or gestational diabetes. They also were selected to have mild-to-moderate depression symptoms, as indicated by a Center for Epidemiological Studies-Depression Scale score ≥16 (Stockings et al., 2015). Exclusion criteria were a full-syndrome psychiatric disorder (e.g., major depressive disorder) that necessitated treatment, a major medical problem (e.g., T2D), medication use that could affect mood or insulin sensitivity, structured weight loss or psychotherapy, and pregnancy. Participants were recruited through direct mailings to Washington, D.C. area families, letters to physicians, media advertisements, distribution to school listservs, and community flyers. Phone screenings were conducted with 937 adolescents and their parents. Of adolescents determined to be potentially eligible by phone screening, 54% attended a laboratory screening visit. The majority (98%) of adolescents who were determined to be eligible at the laboratory screening visits enrolled in the study.

Procedures

All procedures were approved by the Institutional Review Boards of the *Eunice Kennedy Shriver* NICHD and USUHS. All data were from the baseline assessment phase of the study. Procedures were performed in a pediatric outpatient clinic at the NIH Clinical Research Center in Bethesda, Maryland. Parents/guardians provided written consent and adolescents provided written assent for participation. Adolescents were reimbursed for participation.

Measures

Parental Depression Symptoms—Parents completed the reliable and valid 18-item depressed/anxious scale of the Adult Self-Report (Achenbach & Rescorla, 2003). Items are rated on a Likert-scale from 0 to 2, with higher values indicating greater symptomatology. A normed T-score was used for descriptive purposes. The total raw summed score was used in analyses, with higher scores reflecting greater depressed/anxious symptomatology. This scale demonstrates adequate psychometric properties in adults, including good internal reliability (Achenbach & Rescorla, 2003).

Adolescent Depression Symptoms—Adolescent depression symptoms were assessed with the 27-item Children's Depression Inventory (Kovacs & Beck, 1977). The Children's Depression Inventory has been widely used in children and adolescents and demonstrates strong reliability and validity (Kovacs & Beck, 1977). Items are rated on a Likert-scale from 0 to 2. A total score of continuous depression symptoms was derived from the sum of all items, with higher values indicative of greater depression symptoms.

Adolescent Metabolic Risk for Type 2 Diabetes (T2D)—BMI/adiposity. Adolescents' height in triplicate by stadiometer and fasting weight by digital scale were measured to derive BMI (kg/m^2), BMI standard score (z-score), and BMI percentile for age and sex (Kuczmarski et al., 2000). BMI and BMI percentile are reported for descriptive purposes, but age- and sex-adjusted BMI-z score was used in primary analyses. A dual-energy x-ray absorptiometry scan (iDXA, GE Healthcare, Madison, WI) was used to determine percentage body fat and lean mass (kg). Adolescent insulin sensitivity. After an overnight fast, adolescents received 1.75 g/kg of glucola (maximum = 75 g). Blood samples were collected to measure insulin and glucose at fasting, 30, 60, 90 and 120 minutes after oral glucose administration. Insulin concentrations were determined using a commercially-available immunochemiluminometric assay (Diagnostic Product Corporation, Los Angeles, California). Plasma was collected in tubes containing powdered sodium fluoride and glucose was measured by the NIH Clinical Center clinical laboratory using a Hitachi 917 analyzer (Roche Diagnostics Indianapolis, Indiana). Whole body insulin sensitivity index (WBISI) was calculated to estimate insulin sensitivity (Matsuda & DeFronzo, 1999).

Physical Examination and Medical History—A nurse practitioner or endocrinologist assessed pubertal status by breast Tanner staging (Marshall & Tanner, 1969) and conducted a medical family history with the parent/guardian, including the determination of T2D, prediabetes, or gestational diabetes in first- and second-degree relatives. Parents reported their weight and height, which were used to calculate parental BMI (kg/m^2).

Analysis Plan

All analyses were performed with SPSS 23.0 (IBM Corp, 2016). Data were adjusted for outliers, which were corrected to fall within 1.5 times the interquartile range above the 75th or below the 25th percentile. Multiple imputation with 20 imputed datasets was used to handle missing data (2.0%). Pearson correlations were conducted to describe the unadjusted, bivariate associations among parental depression symptoms, adolescent age, adolescent depression symptoms, and adolescent metabolic risk for T2D (BMI-z, adiposity, insulin sensitivity). Multiple linear regressions were run to evaluate the association of parental depression symptoms to adolescent depression symptoms and metabolic risk indicators for T2D. For the dependent variable of adolescent depression symptoms, the covariates of race/ethnicity, age, puberty, body composition (percent fat, lean mass, height), parental T2D status, and parental BMI were entered in the first step, in order to ensure that any observed associations of parental depression were independent of these factors; parental depression symptoms were entered in the next step. Adolescent BMI-z/adiposity were regressed on race/ethnicity, age, puberty, parental T2D status, parental BMI, and parental depression symptoms. Insulin sensitivity was regressed on these same covariates, but also body composition, in order to determine the association of parental depression to adolescent insulin sensitivity, independent of lean mass, percent adiposity, and height.

Results

Descriptive Information

Participants were 116 girls with a mean age of 15.01 (SD = 1.58) years. The majority of participants (63%; n = 73) were non-Hispanic Black (Table 1). The parents who completed questionnaires consisted mostly of mothers (n = 106) with a minority of fathers (n = 10). The primary analyses were unchanged when we included responses only from mothers; thus, we report results for all adolescents. Fifty-seven percent (n = 66) of parents reported having obesity, and nearly one quarter of parents (n = 25) had T2D.

Table 2 presents bivariate correlations among key variables. There was a positive correlation between parental and adolescent depression symptoms (p = .01). In addition, parental depression symptoms were positively related to adolescents' BMI z-score (p = .02) and inversely related to insulin sensitivity (p = .03). Parental depression symptoms showed a trend-level association with adolescent adiposity (p = .07).

Adjusted Associations of Parental Depression Symptoms with Adolescent Depression and Metabolic Risk for T2D

Table 3 displays the regression analysis predicting adolescent depression symptoms. Accounting in step 1 for adolescent race/ethnicity, age, pubertal status, adiposity, lean mass, height, parental T2D, and parental BMI, parental depression symptoms in step 2 remained significantly associated with adolescent depression symptoms (p = .04) and accounted for a unique 4% of variance. Total variability explained in adolescent depression symptoms from all variables in the model was 14%.

The series of regression models predicting adolescent metabolic risk for T2D is shown in Table 4. Parental depression symptoms were significantly, positively related to adolescent BMI-z ($p = .02$) and explained a significant 5% of the variance in youth's BMI-z, after accounting for covariates. Similarly, there was a positive association of parental depression with adiposity, which approached significance ($p = .06$) and explained a unique 3% of variability in adolescent percent adiposity. In contrast to the correlation analysis, parental depression was no longer significantly related to adolescent insulin sensitivity after body composition was accounted for ($p = .50$). Body composition and other covariates explained 38% of the variability in insulin sensitivity, with parental depression adding a non-significant 1% of explained variance.

Discussion

The main aim of this study was to examine the relation between parental depression symptoms and adolescent depression symptoms among adolescent girls identified to be at heightened risk for T2D. In these at-risk youth, there was a significant association between parental and adolescent depression symptoms, even after accounting for potential confounds including adolescent body composition. The second aim was to investigate how parental depression symptoms related to adolescents' degree of metabolic risk for T2D, assessed as BMI-z standard score, percent adiposity, and insulin sensitivity. Our results suggested that parental depression was related to BMI-z, tended to relate to percent adiposity, and was correlated with insulin sensitivity. However, the association of parental depression with insulin sensitivity was accounted for by these factors' joint connection with adolescent body composition.

The positive association between parental depression symptoms and depression symptoms in adolescent girls at-risk for T2D is consistent with past literature in community samples (Agerup et al., 2014) and adolescent patients with type 1 diabetes and T2D (Eckshtain et al., 2010; Lawrence et al., 2006; Weinstock et al., 2015). Existing evidence in adolescents and adults suggests that depression symptoms potentially may increase the risk of worsening of insulin sensitivity, T2D onset, poor treatment adherence, and comorbidities in T2D (Kong & Chan, 2015; Rotella & Mannucci, 2013; Shomaker et al., 2011; Suglia et al., 2016). Interventions addressing depression symptoms in T2D (Baumeister, Hutter, & Bengel, 2014), or more recently, in adolescents at-risk for T2D (Shomaker, Bruggink, et al., 2017; Shomaker et al., 2016; Shomaker, Kelly, et al., 2017), have focused on adults and youth separately. The cross-sectional nature of this study precludes any determinations of directionality in the relation between parental and adolescent depression symptoms. Yet, familial aggregation of depression symptoms in families at-risk for T2D, regardless of the underlying etiology, has potential implications for interventions addressing depression symptoms in T2D prevention and management. For instance, adolescent selective depression prevention research suggests that the persistence of parental depression symptoms may undermine the efficacy of adolescent-only interventions designed to decrease elevated depression symptoms and prevent the onset of major depressive disorder (Garber et al., 2009). Thus, the potential value of family-based interventions addressing both parental and adolescent depression symptoms in families with adolescents at-risk for T2D warrants consideration.

Another study objective was to evaluate how parental depression symptoms were associated with adolescents' degree of metabolic risk for T2D. Parental depression symptoms were related to adolescent girls' higher BMI z-score, and parental depression also had a trend-level association with adiposity. In prior research, parental depression symptoms have been associated with excessive BMI gain in children (Morrissey & Dagher, 2014). Parental depression also was correlated with adolescents' diminished insulin sensitivity, although this effect appeared to be entirely explained by the overlap of body composition with both of these factors, particularly for insulin sensitivity. In prior research, having a familial risk of depression was associated with youths' diminished insulin sensitivity (Mannie et al., 2013). More research is needed to determine the influence of parental depression on metabolic risk for T2D in adolescent offspring.

Strengths of this study include the use of well-validated psychological, anthropometric, and physiological measures in racially/ethnically diverse families with adolescents at-risk for T2D. Study limitations include the relatively small sample size as well as the cross-sectional design, which limits inferences about directionality. Likewise, third variable explanations cannot be ruled out in the observed relations of parental depression symptoms with adolescent depression symptoms and metabolic risk for T2D. Furthermore, due to the highly select nature of the sample of adolescent girls with overweight/ obesity, family members with diabetes, and mild-to-moderate depression symptoms, variability in these characteristics, as well as insulin sensitivity, was therefore restricted, and caution must be exercised in generalizing the results to adolescents without these characteristics. These characteristics may have restricted our ability to detect an association of parental depression with degree of insulin sensitivity. Although the analyses accounted for parental BMI and diabetes status, these measures were self-reported. The majority of parents were mother figures, and future research on this topic would benefit from the inclusion of multiple parental figures and even the entire family unit.

In adolescent girls at-risk for T2D, parent depression symptoms related to adolescents' greater depression symptoms and higher BMI-z. Future research should determine the intervening variables that account for these linkages. Intervening variables might include genetic factors (Franic, Middeldorp, Dolan, Ligthart, & Boomsma, 2010), parenting factors (Hayden et al., 2013), and/or parent-adolescent relationship qualities such as the degree of relationship conflict (Cummings, Koss, & Davies, 2015). Possible mechanisms also may be more specific to T2D risk such as stress-related lifestyle factors (e.g., emotional eating or physical inactivity). Elucidation of mechanisms would contribute to our understanding of how to best tailor and maximize prevention and intervention efforts to ameliorate metabolic and T2D risk in at-risk adolescents and their families. Parent characteristics are often overlooked in studies of adolescent metabolic and T2D risk; findings from the current study highlight the importance of considering the role that parents' psychological conditions may play in adolescent diseases and their prevention.

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Table 1.

Descriptive information about study participants

Adolescent Variables	
	Mean (SD)
Age, years	15.01 (1.58)
BMI, kg/m^2	33.00 (6.57)
BMI z-score	1.97 (.46)
BMI percentile for age and sex	95.92 (3.25)
Adiposity, fat mass %	41.79 (5.74)
Depression symptoms	13.57 (6.09)
Fasting glucose, mg/dL	89.04 (6.84)
Fasting insulin, μU/mL	24.28 (14.02)
Insulin sensitivity, WBISI	2.51 (1.43)
	Frequency (%)
Obesity, BMI ≥ 95th percentile	85 (73.3)
Late puberty, Breast Tanner stage = 5	83 (71.6)
Race/ethnicity	
Non-Hispanic Black/African American	73 (62.9)
Non-Hispanic White	18 (15.5)
Hispanic/Latina	13 (11.2)
Asian	4 (3.4)
Parent Variables	
	Mean (SD)
Depression symptoms T-Score	52.92 (5.08)
	Frequency (%)
Obesity, BMI ≥ 30 kg/m^2	66 (56.9)
T2D, presence	25 (21.6)

Note. BMI = Body mass index. WBISI = Whole body insulin sensitivity index. T2D = Type 2 diabetes. *N* = 116.

Table 2.

Bivariate correlations among key variables

Variable	1.	2.	3.	4.	5.
1. Parent depression symptoms	--				
2. Adolescent age, years	−.10	--			
3. Adolescent BMI-z	.22*	−.09	--		
4. Adolescent adiposity	.17+	.05	.78***	--	
5. Adolescent insulin sensitivity	−.21*	.17+	−.51***	−.46***	--
6. Adolescent depression symptoms	.24**	.13	.11	.01	−.02

Note. BMI-z = Body mass index standard score for age and sex.

*** $p \leq .001$.

** $p \leq .01$.

* $p \leq .05$.

$N = 116$.

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Table 3.

Multiple linear regression predicting adolescent depression symptoms

	Adolescent Depression Symptoms			
Predictors	**B**	**SE**	***t***	**Model R^2**
Step 1. Covariates				
Adolescent race, Black	−.04	1.24	−.03	
Adolescent age, years	.38	.43	.89	
Adolescent puberty, Tanner 5	−.12	1.51	−.08	
Adolescent adiposity, % fat	−.08	.11	−.78	
Adolescent lean mass, kg	.22*	.10	2.25*	
Adolescent height, cm	−.14	.11	−1.20	
Parent T2D, presence	−2.95*	1.38	−2.14*	
Parent BMI, kg/m^2	.03	.06	.45	
				R^2=.10
				ΔR^2=.10
Step 2. Parent Depression				
Parent depression symptoms	.29*	.14	2.03*	R^2=.14*
				ΔR^2=.04*

Note. T2D = Type 2 diabetes. BMI = Body mass index.

*** $p \leq .001$.

** $p \leq .01$.

* $p \leq .05$.

$N = 116$.

Table 4.

Multiple linear regressions predicting adolescent metabolic risk indicators for T2D

	Adolescent BMI-z				Adolescent Adiposity (%)				Adolescent Insulin Sensitivity			
Predictors	**B**	**SE**	***t***	**Model R^2**	**B**	**SE**	***t***	**Model R^2**	**B**	**SE**	***t***	**Model R^2**
Step 1. Covariates												
Adolescent race, Black	.06	.09	.65		−1.35	1.18	−1.14		−.15	.25	−.59	
Adolescent age, years	−.03	.03	−.90		.27	.41	.66		.20 *	.09	2.28 *	
Adolescent puberty, Tanner 5	.02	.11	.14		−.31	1.42	−.22		.32	.30	1.07	
Adolescent adiposity, % fat	--	--	--		--	--	--		−.09 ***	.02	−4.10 ***	
Adolescent lean mass, kg	--	--	--		--	--	--		−.07 ***	.02	−3.95 ***	
Adolescent height, cm	--	--	--		--	--	--		.03	.02	1.53	
Parent T2D, presence	.09	.11	.82		.56	1.33	.42		.38	.27	1.39	
Parent BMI, kg/m^2	.01	.01	1.24		.02	.07	.29		.02	.01	1.17	
				R^2=.04				R^2=.02				R^2=.38 ***
				ΔR^2=.04				ΔR^2=.02				ΔR^2=.38 ***
Step 2. Parent Depression												
Parent depression symptoms	.03 *	.01	2.41 *		.26 +	.14	1.89 +		−.02	.03	−.68	
				R^2=.09 *				R^2=.05 +				R^2=.39 ***
				ΔR^2=.05 *				ΔR^2=.03 +				ΔR^2=.01

Note. T2D = Type 2 diabetes. BMI = Body mass index.

*** $p \leq .001$.

** $p \leq .01$.

* $p \leq .05$.

$N = 116$.