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Association between depressive symptoms in adolescence and birth outcomes in early adulthood using a population-based sample

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ABSTRACT

Background. Adolescent female depressive symptomatology is an unrecognized mood disorder that impairs health in adolescence or adulthood. However, the long-term effects of pre-pregnancy depressive symptoms on birth outcomes in adulthood have not been given adequate empirical assessments.

Method. In this study, we assessed the relationship between the life time duration of depressive symptoms over a 14-year period and birth outcomes (LBW and PTB) among a sample of 6023 female respondents who took part in the National Longitudinal Study of Adolescent to Adult Health (Add Health). We used the generalized estimating equation (GEE) models to assess these relationships.

Results. Exposure to elevated depressive symptoms in late adolescence, but not in adulthood, was associated with increased odds of LBW by more than 2-fold in early and young adulthoods (adjusted odds ratio [aOR] = 2.19; 95% confidence interval, CI: 1.56, 3.08). Depressive symptoms in early adulthood were independently associated with increased odds of PTB and were higher for black mothers. Maternal race modified the relationship between consistent reporting of depressive symptoms in adolescence and LBW or PTB in adulthood.

Conclusion. This study provides compelling evidence that effects of elevated depressive symptomatology on LBW or PTB appear to be linked to a specific development period in adolescence. National policies to address social inequalities and stratification particularly in health at all stages of human development, will provide an important step in reducing depressive symptoms prior to early adulthood and in pregnancy and childbirth.

Adolescent depressive symptomatology is one of the most unrecognized mood disorders that impair health in early and young adulthood (Cicchetti and Toth, 1998; Fergusson et al., 2005; Jonsson et al., 2011). Elevated depressive symptoms in adolescence strongly predict onset of depression in adulthood (Lakdawalla et al., 2007). Depression is a complex phenomena characterized by negative mood (sadness, crying spells, and unhappiness), somatic conditions (aches and pains, trouble sleeping, fatigue), and cognitive vulnerability (feelings of worthlessness, failure, loss) as well as behaviors that could disrupt an individual's social relations (Lakdawalla et al., 2007; Compas et al., 1993; Lehrer et al., 2006). The preponderance of higher depressive symptoms and major depressive episodes among females is well-established (Nolen-Hoeksema and Girgus, 1994; Williams et al., 2007; Thapar et al., 2012; Guxens et al., 2013; Naicker et al., 2013). Although most epidemiologic studies of maternal depressive symptoms on infants' health have analyzed prenatal or postnatal exposures, relatively few have evaluated pre-pregnancy symptoms (Cicchetti and Toth, 1998; Keenan-Miller et al., 2007; Giles et al., 2011; Radloff, 1977). Emerging evidence indicates early maternal exposures to depressive symptoms may be etiologically significant for later infant and maternal health outcomes, but there is a dearth of studies evaluating these relationships from pre-pregnancy periods (Keenan-Miller et al., 2007; McLearn et al., 2006; Barker, 1995) and based on cumulative exposures over time (Henriksen and Clausen, 2002). *Depressive symptoms in adolescence are a major determinant of major depressive episodes (MDE) in adulthood* (Davidson et al., 2000), *and also linked to a number of diseases including hypertension, coronary heart diseases (CHD) and myocardial infarction. Furthermore, these symptoms occurring in adolescence are a major concern because of the interaction with numerous health behaviors including increased use of psychoactive substances and other poor health sequelae in later life. Adolescents with elevated depressive symptoms have more time out of work, lower educational achievement and longer time to recovery from the diagnosis of depressive disorder* (Hankin, 2006; Haas et al., 2005). *The association between elevated depressive symptoms and low birth weight or preterm birth is biologically plausible given the mediating role of the hormone cortisol and pro-inflammatory cytokines. Prolonged maternal exposure to cortisol concentration is associated with fetal*

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programming and reduced gestational age (Rifkin-Graboi et al., 2013). *These findings indicate that focusing on addressing the prenatal needs of pregnant women might be late to modify health behaviors or treat cumulative risk exposures to the fetal environment.*

Multiple cross-sectional and prospective studies have shown that elevated prenatal depression or depressive symptoms are associated with low birth weight (LBW), preterm births (PTB), and other adverse birth outcomes among clinical and population-based samples (Lu et al., 2010; Dayan et al., 2002; Diego et al., 2009). Among longitudinal studies investigating the relationship between prenatal depressive symptoms and adverse birth outcomes, findings have been mixed (Li et al., 2009; Andersson et al., 2004; Suri et al., 2007; Evans et al., 2007) making the temporal ordering between prior prenatal depressive symptomatology and birth outcome relationships difficult to determine unambiguously (Evans et al., 2007; Gawlik et al., 2012). Although a recent meta-analysis of longitudinal studies did not find statistically significant tests among a great number of studies analyzing a prospective relationship between prenatal depressive disorders and LBW or PTB, the authors (Goedhart et al., 2010) postulated that the burden of prenatal depression is of enormous public health importance (Grote et al., 2010; Alexander et al., 2003).

LBW and PTB outcomes are directly associated with neonatal mortality, infant mortality and morbidity, and neurodevelopmental impairments in early or later adulthood (Henriksen and Clausen, 2002). In the United States (US), these outcomes constitute a major determinant of health inequalities among the racial groups (Geronimus, 1996; Colman et al., 2007). A clear understanding of the relationship between depressive symptoms occurring prior to pregnancy and modifying effects of other covariates (maternal race, age, and access to mental health services) may provide useful information about the underlying mechanisms and the natural unfolding of depression over time in determining LBW and PTB outcomes among population groups.

We used the contractual data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) from over a 14-year period to evaluate related questions that have not been adequately addressed by previous investigations. The purpose was to evaluate the relationship between pre-pregnancy depressive symptoms and birth outcomes in early adulthood.

Materials and methods

Data for this study came from the restricted National Longitudinal Study of Adolescent to Adult Health (Add Health). A comprehensive analysis of design features of the study is found in other publications (Fletcher, 2010; Harris, 2005). Add Health is a school-based representative sampling study of health and social outcomes of US adolescents in grades 7–12 in the 1994–1995 academic year, who were followed with in-home interviews until adulthood in 2008. A Wave II in-home interview (n = 14,438) was conducted approximately one year later (1995/96), and another in-home survey was conducted six years later: Wave III (n = 15,197) in 2001–2002 (aged 18–26). Wave IV was completed in 2008 and surveyed 15,355 of the original Wave I respondents, then adults in the age range 26–32.

Sample

We restricted our analyses to 6023 female in-home Wave I respondents with complete information on all variables across all four waves. The sample included 1624 non-Hispanic blacks, 2940 whites, and 1459 Hispanics. We excluded respondents with age ≥20 years in Wave I, multiple births, gestation age <20 weeks, and birth weight ≤500 g. The total samples with non-missing data on gestational age and birth weight in Wave III (mean age 21.83, SD 1.61) were 2846 and 3220, respectively. The study was approved by the Institutional Review Board of Central Michigan University, Mt Pleasant, MI.

Measures

Birth weight and gestational age

At Waves III and IV, respondents were asked about current pregnancies occurring in each wave of data collection (i.e., only the first births occurring in either Wave III or IV was selected). We selected only pregnancies resulting in live births (vaginal or caesarian) in each wave to create birth weight (BWT) and gestational age categories. Respondents indicating they had given live births (in either Wave III or Wave IV) they were also asked "What was the birth weight (pounds) at birth?" Next, respondents were asked "Was the first baby born too early—that is, after a pregnancy of less than 40 weeks?" The final question was "How many weeks early was [Baby's name] born?" We used this information to calculate gestational age by subtracting the response from 40 weeks (Harville et al., 2012). We created a two level outcome variable: PTB as <37 weeks of gestation and completed term gestation as ≥37 weeks. In a sensitivity analysis, we created a 3-level outcome variable for birth weight as LBW (<2500 g), normal birth weight (≥2500 g), and very low birth weight (<1500 g) and gestational age as: very preterm (<33 weeks gestation), moderately preterm (33–36 weeks), and normal gestation (≥37 weeks). The results were not very different from the 2-level analysis reported here.

Depressive symptoms in childhood through adulthood

At Wave I (age 11–19), respondents answered a comprehensive battery of mental and psychological health questions using a modified form of the Center for Epidemiologic Study of Depression (CES-D) scale, a widely used instrument for assessing depressive symptoms among non-psychiatric populations (Harville et al., 2012; Stommel et al., 1993). The Add Health survey used a modified version consisting of 19 items from the original 20-item CES-D scale to assess depressive symptoms in Waves I and II. In the analysis, we used a CES-D score of 24 as the cutoff point to indicate symptoms of depression among adolescent females (Giles et al., 2011; Radloff, 1977). This is known to optimize sensitivity and specificity for *Diagnostic and Statistical Manual of Mental Disorders, Revised Fourth Edition* major depressive disorders for adolescent females (Lehrer et al., 2006).

Next, we used these CES-D items in Waves I and II to create 3-level depressive symptom variables reflecting changes and cumulative exposures of depressive symptoms in childhood and adolescence (chronic depressive symptoms-reported elevated depressive symptoms in Waves I and II; moderate symptoms-reported elevated symptoms in Waves I or II; and none-did not report high depressive symptoms in either waves). Waves III and IV used a 9-item version of the CES-D scale to assess symptoms in early and young adulthood respectively. Early and young adulthoods describe respondents in the respective age waves (Wave III) and (Wave IV) and accompanying age categories. The validity of the shortened CES-D version in research is well-documented (Grywacz et al., 2006; Zhang et al., 2012). Likewise, we also used the shortened 9-item version of the original CES-D scale to assess cumulative exposures of depressive symptoms in early and young adulthood. Finally, we created comparable indicators of CES-D across Waves II and III and Waves III and IV by summing participants responses (ranging from 0 to 3), using a cutoff point of 11. We assessed preexisting comorbidity (depression diagnoses in Waves III and IV, cigarette smoking, alcohol use) and other covariates (including race, age, and access to mental health services). Access to mental health services in adolescence was used as an indication for levels of distress reported and antidepressant treatment availability (Lewinsohn et al., 2000; Naicker et al., 2013).

Statistical analysis

Each respondent was assigned a longitudinal weight factor according to variables evaluated in the study to account for the survey design,

strata, and weighting factor. Two dependent variables were evaluated with statistical models using SAS (Version 9.3; SAS Institute Inc., Cary, NC) and SAS callable SUDAAN (RTI, NC). We used generalized estimating equations (GEE) with independent covariance structure to evaluate the relationship between LBW or PTB and depressive symptoms, controlling for other confounders from different waves. The GEE is also robust to missing data and uses only non-missing pairs of data in estimating the working correlation parameters (Horton and Lipsitz, 1999). We modeled a lagged analysis with time-varying covariates, so that each outcome in Waves III or IV was determined as a function of predictors assessed from previous waves. The time lag design enables the comparison of the same individuals over different time periods, thus accounting for age and other socio-developmental influences. In this regards, we were able to examine variations in depressive symptoms with changes in other developmental factors to which individual mothers were exposed. All models controlled for age, mother's birth weight (reported at Wave I), race, prenatal care, access to mental health services, drug use, and concurrent depressive symptoms. We evaluated interaction (on the multiplicative scale) between depressive symptoms and age (adolescence vs. early adulthood; adolescence vs. young adulthood), access to mental health services, and race in fully adjusted models for both outcomes. For models of depressive symptoms, we report the odds ratios (ORs) and 95% confidence intervals (CIs) for developing LBW or PTB. A 2-sided *P*-value <0.05 was considered statistically significant.

Results

In Wave I, about 39% and 34% of adolescent girls with respective age 11–15 and 16–19 reported elevated depressive symptoms (using CES-D cut off ≥11) and the pattern increases till young adulthood. This observation correlates with CES-D cutoff ≥24. In early adulthood 10.5% (296) of babies were born preterm (<37 weeks gestation) and 18.8% babies (601) were LBW (<2500 g). Similarly, in young adulthood, of the total singleton births, 15.7% (851) babies were LBW and 9.9% (549) were preterm (Data not shown in the Table).

Table 1 shows associations among age cohorts, maternal behaviors, depressive symptoms and diagnosis of depression in Waves III and IV. In each age, the younger cohort was less likely to have access to mental health services until young adulthood (30–33 years). High alcohol consumption was prevalent among all age categories in early adolescence; this declined in older adolescence and increased somewhat in early adulthood. Likewise, prominent trends for depressive symptoms are observed across age categories.

Depressive symptoms and birth outcomes (LBW and PTB) among nulliparous women over time

Table 2 presents associations of demographic characteristics, depressive symptoms, and birth outcomes in early and young adulthood. In early adulthood, the age group 19–23 compared to 24–27 reported a higher prevalence of LBW and PTB, but this pattern was reversed in young adulthood. Across all waves, reporting of elevated depressive symptoms in adolescence was associated with a higher prevalence of LBW and PTB.

As shown in Table 3, the first set of columns presents the estimated odds of LBW and PTB in Waves III and IV respectively. In Model 1, depressive symptoms reported in older adolescence (Wave II), but not depressive symptoms in early adolescence (Wave I), was associated with LBW in adulthood (Wave III) [OR, 2.19; 95% CI: 1.56, 3.08] and Wave IV (OR, 1.40; 95% CI: 1.08, 1.82). Black mothers were at increased odds of LBW in early and young adulthoods. Similarly, depressive symptoms reported in older adolescence (Wave II), but neither depressive symptoms reported in Wave I nor Wave III, was significantly associated with LBW in Wave III (OR, 3.31 95% CI: 1.84, 5.97), LBW in Wave IV (OR, 1.63; 95% CI: 1.12, 2.38) and PTB in Wave IV (OR, 1.68; 95% CI:

Table 1
Associations between age cohorts, depressive symptoms and other selected covariates measured across the waves among participants in the National Longitudinal Study of Adolescent to Adult Health (Add Health), 1994–2008.

	Cohort age groups (in years) across different survey periods											
	11–15 16–19			12–16 17–20			19–23 24–27			25–29		30–33
	n	%[a]	%*[a]	n	%[a]	%[a]	n	%[a]	%[a]	n	%[a]	%*
Maternal race												
Black	1624	10.9	9.6	1624	11.0	9.8	1525	18.0	3.6	1624	7.2	14.2
White	2940	33.9	28.4	2940	34.7	27.4	2680	53.0	8.3	2933	21.8	38.4
Hispanic	1459	9.5	7.8	1458	9.4	7.7	1352	14.3	2.8	1459	7.3	11.1
Mental health access												
Yes	911	9.0	7.1	692	7.9	5.2	450	7.8	1.2	682	4.5	7.6
No	5106	45.2	38.2	5333	47.2	39.7	5100	77.7	13.5	5339	31.8	56.1
Alcohol use												
No alcohol	1257	9.8	14.2	2790	52.0	34.7	3359	53.4	10.2	3235	27.2	47.3
Alcohol use 1–2 weeks	334	2.5	3.5	334	3.2	4.2	157	3.0	0.4	175	1.8	1.8
Alcohol use (≥5 in a month)	3790	41.7	28.3	276	3.1	2.8	1498	28.9	4.2	840	8.0	13.9
Cigarette smoking												
No smoking	2209	44.4	30.1	3127	40.5	32.4	1561	50.8	10.8	2377	23.5	45.9
1–9	382	7.1	5.5	495	8.6	4.7	143	5.0	0.3	181	2.5	2.8
10–19	163	2.2	4.9	222	3.1	3.8	287	13.2	3.6	359	5.8	8.4
≥20	114	1.7	3.9	197	3.1	3.8	303	15.1	1.3	290	3.8	7.3
CES-D[§]												
<11	1235	15.9	10.8	1247	15.5	13.4	2169	34.4	6.0	1978	11.6	22.0
≥11	3484	39.2	34.1	3480	40.3	30.8	3391	51.0	8.7	4043	24.8	41.6
Depression diagnosis												
Yes	–	–	–	–	–		876	15.6	2.8	1402	9.6	16.5
No							4682	69.7	11.9	4619	26.8	47.2

Abbreviations: CES-D, Center for Epidemiologic Studies of Depression scale; ellipses, not available.
Statistical significance achieved at P = .0001 for all variables. Percentages may not total 100 because of missing data.
Age group categories: Wave I (11–19 years); Wave II (12–20 years); Wave III (19–27 years); Wave IV (25–33 years).
[§]CES-D scores measured in Wave III using the short version of the original CES-D instrument.
[a]Percent is weighted to account for survey design and sampling characteristics.

Table 2
Maternal characteristics according to selected baseline (Wave I) demographic and depressive symptoms reported among respondents reporting first birth in early adulthood (Waves III and IV) from the National Longitudinal Study of Adolescent to Adult Health (Add Health), 1994–2008.

Characteristic	Age category	Wave III	Wave IV[a]					
	Birth weight (g)		Gestation (weeks)		Birth weight (g)		Gestation (weeks)	
	<2500		<37		<2500		<37	
	N = 3191	%*	N = 2824	%*	N = 5425	%*	N = 5525	%*
Age (years)								
19–23	2494	15.3	2191	7.6				
24–27	541	3.1	481	2.0				
Missing	156	1.1	152	0.5				
25–29					1661	5.6	1702	3.6
30–33					3760	10.0	3816	7.2
Missing					4	0.0	7	0.0
Race								
Black	941	6.7	784	2.5	1434	4.3	1457	1.9
White	1465	10.3	1342	6.0	2692	8.7	2707	7.2
Hispanics and others	785	2.5	693	1.5	1294	2.7	1356	1.8
Missing								
CES-D[†]								
<24	1029	6.9	933	3.4	1967	5.8	1971	3.8
≥24	1493	8.4	1315	5.1	2499	6.7	2541	5.2
Missing	669	4.2	576	1.6	959	3.2	1013	1.8
Changes in symptoms								
No symptoms	385	2.5	351	1.4	769	2.1	708	1.8
Moderate symptoms	2221	13.9	1955	6.7	3794	11.3	3843	7.6
Severe symptoms	594	3.1	518	1.8	862	2.4	904	1.4
Mental health access[§]								
No	2645	16.4	2343	7.6	4586	13.5	4670	8.4
Yes	541	3.1	476	2.5	830	2.2	844	1.5
Missing	5	0.1	5	0.1	9	0.1	11	0.2

Add Health is The National Longitudinal Study of Adolescent to Adult Health.
Statistical significance achieved at P = .0001 for all variables except for mental health and gestation period (P = .39). Percentages may not total 100% because of missing data.
[†]CES-D score measured at Wave I, using a cutoff point of 24 as an indicator of depressive symptoms for adolescents.
[a]Weighted percent to reflect larger population sample characteristics in the United States.
Key study variables measured at Wave 1.
[§]P = .15 (not statistically significant).
*Percent is weighted to account for survey design and sampling characteristics.

1.05, 2.69) in age, psychoactive drugs use, earlier depressive symptoms and BMI adjusted model (Model 2). In fully adjusted models (Model 3), only depressive symptoms reported in older adolescence (Wave II) was significantly associated with PTB in Wave III (OR, 2.59; 95% CI: 1.31, 5.11). Neither depressive symptoms reported in early adolescence (Wave I) nor in adulthood (Wave III) were associated with either birth outcomes in Model 2 or fully adjusted models (Model 3).

Modifying effects of age, race, and depressive symptoms on LBW and PTB outcomes

We found significant interactions among the racial groups, pre-pregnancy reporting of depressive symptoms, access to mental health services (Wave I) and maternal age groups in determining LBW and PTB (Table 3). Although the width of the confidence interval for the interaction term between the racial groups and access to mental health in Wave I was wider in models (Model 4 Table 3) predicting LBW in adulthood (Wave III) and LBW or PTB in Wave IV. In race-stratified analyses (Table 3), access to mental health in early adolescence (Wave I) among black mothers predicted LBW (OR, 7.21; 95% CI: 1.77, 16.29), but not on other black mothers with no access to mental health in Wave I, while high depressive symptoms reported in Wave II among white mothers was associated with LBW in adulthood (Wave III) (OR, 3.24; 95% CI: 1.86, 5.67). The non-significant associations or unstable estimates found among the interaction terms and birth outcomes are likely a reflection of low power among the racial groups. Likewise, a statistically significant result was found for the association between black mothers and LBW in early adulthood among the age group 24–27 years (vs. black mothers 19–23 years) (Table 3).

Cumulative depressive symptom exposures, access to mental health services and birth outcomes over the developmental stages

To evaluate depressive symptom exposures across developmental periods (in adolescence and adulthood), we modeled changes in exposures during adolescence and adulthood (Table 4). Although the main effects of severe and moderate depressive symptoms reported in adolescence on LBW or PTB in adulthood (Wave IV) were null, these symptoms predicted LBW in Wave III in a fully adjusted models (including maternal age, race, BMI, smoking, and current alcohol use). However, depressive symptom exposures in adulthood were not associated with LBW or PTB. In adulthood (Waves III and IV), reporting severe depressive symptoms was associated with LBW (OR, 2.96; 95% CI: 1.40, 6.23) and PTB (OR, 3.76; 95% CI: 1.53, 9.26) among black mothers and Hispanics for PTB (OR, 23.31; 95% CI: 1.65, 328.95), while reporting of moderate depressive symptoms in adulthood was associated with PTB in Wave IV (OR, 30.17; 95% CI: 2.01, 453.62) among Hispanics.

For the entire sample, the adjusted ORs were statistically significant for PTB outcomes in Wave III (OR, 2.12; 95% CI: 1.26, 3.58) and Wave IV (OR, 1.58; 95% CI: 1.01, 2.47) among adolescents reporting limited access to mental health services (in Waves I and II) compared to those reporting none access to the mental health services. However, in adulthood (Waves III and IV) only respondents reporting increased access to mental health services showed marginally statistical significant odds for PTB in adulthood (Wave IV).

Table 3
The risk of low birth (LBW) and preterm birth outcomes according to maternal depressive symptoms among respondents participating in the National Longitudinal Study of Adolescent to Adult Health (Add Health), 1994–2008.

	Wave III (Adulthood)		Wave IV (Adulthood)	
	LBW	PTB	LBW	PTB
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Total sample				
Model 1				
Depressive symptoms	0.70 (0.49, 0.99)	1.41 (0.87, 2.27)	1.14 (0.80, 1.61)	
Depressive symptoms	2.19 (1.56, 3.08)	1.12 (0.74, 1.71)	1.40 (1.08, 1.82)	1.29 (0.91, 1.82)
Black	1.79 (1.31, 2.45)	1.02 (0.66, 1.57)	1.52 (1.17, 1.97)	0.70 (0.49, 0.98)
Hispanic	0.84 (0.53, 1.33)	0.82 (0.48, 1.38)	0.95 (0.69, 1.30)	0.75 (0.50, 1.11)
Model 2				
Depressive symptoms	0.53 (0.30, 0.91)	1.85 (0.89, 3.87)		1.17 (0.73, 1.86)
Depressive symptoms	3.31 (1.84, 5.97)	0.90 (0.48, 1.69)	1.63 (1.12, 2.38)	1.68 (1.05, 2.69)
Depressive symptoms	---	---	0.90 (0.64, 1.26)	0.86 (0.57, 1.30)
Black	0.87 (0.48, 1.59)	0.47 (0.23, 0.97)	1.37 (0.94, 2.00)	0.72 (0.93, 2.18)
Hispanic	0.75 (0.37, 1.53)	0.33 (0.13, 0.82)	1.18 (0.79, 1.78)	0.57 (0.33, 0.97)
24–27 years	0.45 (0.23, 0.86)	0.56 (0.25, 1.25)	---	---
Model 3				
Depressive symptoms	3.22 (1.76, 5.88)	0.68 (0.36, 1.28)	1.45 (0.54, 3.85)	2.15 (0.38, 12.08)
Depressive symptoms	1.25 (0.75, 2.08)	2.59 (1.31, 5.11)	0.94 (0.45, 1.97)	2.18 (0.69, 6.89)
Depressive symptoms			0.48 (0.20, 1.15)	1.30 (0.31, 5.43)
Black	0.78 (0.39, 1.55)	0.52 (0.23, 1..20)	2.44 (0.69, 8.65)	0.08 (0.02, 0.40)
Hispanic	1.14 (0.54, 2.38)	0.39 (0.13, 1.13)	2.21 (0.77, 6.34)	0.50 (0.11, 2.28)
Access to mental health	0.43 (0.20, 0.89)	1.20 (0.55, 2.65)	1.19 (0.29, 4.91)	0.48 (0.12, 2.02)
Model 4 (Interaction results only)				
Black[†]	7.21 (1.77, 16.29)	1.38 (0.24, 8.09)	59.46 (6.30, 560.97)[§]	1.96 (0.09, 43.70)
Hispanic[††]	0.52 (0.07, 4.18)	0.03 (0.00, 0.37)	3.48 (0.16, 76.06) [§]	24.03 (1.57, 367.20)
Black (24–27 years)	2.95 (1.21, 7.21)	1.01 (0.14, 7.41)	---	---
Hispanic (24–27 years)	0.10 (0.20, 0.53)	0.13 (0.02, 0.93)		
White x depressive symptoms[±]	3.24 (1.86, 5.67)	---	---	---

Abbreviations: CI = Confidence Interval; OR, Odds ratio; LBW, low birth weight; PTB, preterm births. -- ellipses, not available/unstable estimates.
*P < 0.05 (compared with normal gestation ≥37 weeks or birth weight at birth ≥2500 g).
Model 1 adjusted for age (at respective wave), BMI at Wave I, and depressive symptoms at Wave I.
Model 2 adjusted for age, BMI (at Wave 1), depressive symptoms (Wave 1), Wave 2 depressive symptoms, current smoking and alcohol use during pregnancy.
Model 3 adjusted for age, BMI (at Wave I), depressive symptoms (Wave I), depressive symptoms (Wave II), current smoking, access to mental healthcare (Wave I), prenatal care at the respective waves.
Model 4 is Model 3 + interaction terms as the product term (Wave 1 access to mental health + concurrent depressive symptoms).
[†]Blacks access to mental health, compared with other black population with no access to mental health services in Wave I.
[††] Compares whites with access to those with no mental health services in Wave I.
[§]Refers to the interaction between Black mother's earlier access to mental health services in adolescence and risk of LBW in later adulthood compared with other similarly Black mothers.
[ɡ] Refers to the interaction of access to mental health in Wave I and racial categories in determining LBW or PTB outcomes.
[±] Interaction of white race with depressive symptoms reported in Wave II compared with other whites with low depressive symptoms.

Discussion

Main findings

Reporting of depressive symptoms in older adolescence, maternal race as black, and cumulative exposures in adolescence were associated with increased risks of LBW or PTB outcomes in adulthood. Diagnosis of depression in early or young adulthood was not associated with increased risk of LBW or PTB. We found that maternal race is the most consistent effect modifier of the relationship between pre-pregnancy depressive symptom exposures and LBW, with black mothers experiencing higher odds. Our analysis also showed that maternal age modified the relationship between pre-pregnancy depressive symptom exposures and LBW. Future studies could investigate the extent to which childhood socioeconomic or contextual factors modify the association between pre-pregnancy depressive symptoms and birth outcomes in early or young adulthood.

Our results indicate that early exposure to elevated levels of depressive symptoms in adolescence, but not in adulthood, may increase the risk of LBW and, to some extent, PTB outcomes. Previous studies assessing the possible relationship between elevated depressive symptoms or diagnosis of depression and LBW, PTB, and other perinatal birth outcomes have been inconsistent (Orr et al., 2002; Gavin et al., 2009), likely due to limitations of selection bias, study design, and statistical power to assess modifying effects of depressive symptoms with other exposures. None of the previous studies simultaneously assessed relationships between pre-pregnancy depressive symptom exposures, access to mental health services, diagnosis of depression, and birth outcomes separately. Interestingly, the apparent inverse association between cigarette smoking, depressive symptoms and LBW or PTB is consistent with the "birth weight paradox" observed in numerous investigations on maternal, infant, and child health outcomes (Hernandez-Diaz et al., 2006; Wilcox, 2006). The paradox describes a reversal of effects (bias) when populations with increased risk exposures (black race) for perinatal outcomes report improved outcomes when stratified on those risks. Given that LBW is a shared effect of many different exposures, the reversal of smoking effects likely results from ignoring unmeasured covariates, thus adjustment of the birth weight variable through stratification or restriction might be unwarranted (Hernandez-Diaz et al., 2006; Whitcomb et al., 2009). A naïve interpretation of this finding might suggest that being black and having depressive symptoms early in adolescence improves birth outcomes, but given the biological plausibility and observed disparities in birth weight, this is unlikely. Exposure to early mental health services among black mothers appears to have less beneficial effects in determining LBW or PTB.

In the US, poor health outcomes among the black population, particularly racial disparities in birth outcomes, are the fundamental cause of

Table 4
Relationship between cumulative depressive symptom exposures and access to mental health services from adolescence to adulthood as predictors of low birth weight and preterm birth outcomes among participants in the National Longitudinal Survey of Adolescent to Adult Health, 1994–2008.

	Wave III (adulthood)		Wave IV (adulthood)	
	LBW	PTB	LBW	PTB
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Depressive symptoms				
Adolescence[§]				
Severe symptoms	3.20 (1.51, 6.76)	1.50 (0.60, 3.74)	1.16 (0.66, 2.04)	0.67 (0.51, 1.32)
Moderate symptoms	4.10 (2.18, 7.69)	1.91 (0.85, 4.28)	1.06 (0.67, 1.69)	0.87 (0.51, 1.48)
Black x severe symptoms			0.21 (0.07, 0.64)	0.24 (0.06, 0.95)
Age (30–33) x severe symptoms			0.59 (0.39, 0.89)	---
Adulthood[§]				
Severe symptoms			1.17 (0.37, 3.69)	0.56 (0.23, 1.38)
Moderate symptoms			1.06 (0.33, 3.41)	0.58 (0.23, 1.46)
Black x severe symptoms			2.96 (1.40, 6.23)	3.76 (1.53, 9.26)
Hispanic x severe depressive symptoms				23.31 (1.65, 328.95)
Hispanic x moderate depressive symptoms				30.17 (2.01, 453.62)
Access to mental health				
Adolescence				
Limited access	0.74 (0.45, 1.20)	2.12 (1.26, 3.58)	0.87 (0.57, 1.32)	1.58 (1.01, 2.47)
Increased access	1.36 (0.74, 2.50)	1.06 (0.35, 3.22)	0.94 (0.54, 1.64)	1.61 (0.86, 3.00)
Adulthood				
Limited access			0.99 (0.73, 1.33)	1.10 (0.79, 1.54)
Increased access			1.16 (0.57, 2.39)	2.08 (1.00, 4.32)

[§]Cumulative depressive symptom exposures between adolescence (Waves I and II) and adulthood (Waves III and IV). Severe depressive symptoms indicate reporting of symptoms ≥11 across both waves of data collection. Moderate depressive symptoms include reporting of symptoms ≥11 in only one wave of data collection. None suggests participant did not report depressive symptoms ≥11 in either wave. None was used as the reference category.
Access to mental health services across the waves during adolescence and adulthood was categorized as limited access (receiving mental health services during adolescence Waves I and II; or in adulthood Wave III to Wave IV) and increased access-receiving services across both waves.
— ellipses, not available/unstable estimates.

racial health inequalities. These inequalities are not biologically determined, but result from patterned social circumstances, including exposures to adverse environmental conditions that create differential distress in the health of population groups (Nkansah-Amankra et al., 2010). It is likely that depressive symptoms erode black mothers' physiological health prior to pregnancy. Geronimus has described this early deterioration typified by high allostatic load scores as weathering, and the phenomenon likely accounts for poor birth outcome disparities among US racial groups (Geronimus, 1996; Geronimus et al., 2006). Geronimus proposes that weathering is an adaptive response for black mothers, encapsulating cumulative disadvantages associated with socioeconomic factors, discrimination, and racism. Implied is the observation that advancing age among black mothers' results in a precipitous decline in physiological capacity and adverse birth outcomes (Wildsmith, 2002).

LBW represents heightened growth retardation or restriction during fetal life (Elsenbruch et al., 2007), and PTB suggests pregnancy complications of unknown etiology (Goldenberg and Culhane, 2005). Our analyses raise important issues for etiological research. First, the direct effect of adolescent depressive symptoms on increased LBW or PTB outcomes is unequivocal. Elevated depressive symptoms in adolescence "have a long reach" in adulthood to determine adverse maternal birth outcomes, even when diagnosis of depression is conditioned. It is likely that one major pathway involves neuroendocrine imbalances and dysregulation of the hypothalamic pituitary–adrenal (HPA) axis hyper action, which results in secretion of the high glucocorticoids (cortisol in humans) and elevated pro-inflammatory cytokine responses to activate the placental endothelium and other changes in the placental environment (Osborne and Monk, 2013; Ananth and Wilcox, 2001). Because placental abruption is an indication of chorionic tissue extending to placental implantation, the effect of maternal and the resulting pro-inflammatory responses likely affect later fetal developments and implantation (Ananth and VanderWeele, 2011; Pariante and Lightman, 2008) through programming in the HPA axis. Second, preterm, intrauterine growth retardation and LBW each serve as a marker of prenatal adversity before or during pregnancy. The stress vulnerability framework suggests that negative or stressful events predispose individuals to depression (Hankin, 2006). Major social and environmental factors and the individual's pathological state are viewed as threats to health and well-being. This bidirectional transactional model suggests that personality characteristics (including hostility, anger, and anxiety) and events such as isolation exacerbate depressive symptoms.

Third, because depressed women are more likely to withdraw socially, obtaining pregnancy support could be challenging. These challenges are likely to threaten the life of the fetus (Elsenbruch et al., 2007) and negatively affect the maternal-infant post-partum dyad. Women with elevated depressive symptoms are also likely to engage in behaviors that put their own and the infant's health at risk. Thus, interventions targeting girls and young women before pregnancy may profoundly affect maternal and infant health.

The overall effects of depressive symptoms or depression on health and maternal-infant child health (MICH) outcomes have been widely documented in the literature. Some inconclusive findings have been reported and are recognized in this study. Given the transient nature of depressive symptoms, the controversy has been whether pre-pregnancy depression or symptoms persist to impact on pregnancy outcomes in early or young adulthood. However, our findings add to the growing evidence that high depressive symptoms in late adolescence adversely affect MICH outcomes.

In a population-based prospective cohort study of pregnant women in the Kaiser Permanente Medical Care Program, women with a high CES-D score (≥22) had a 2-fold increased risk of preterm delivery (Li et al., 2009) as did pregnant African–American women enrolled in a four hospital-based study in Baltimore, Maryland (Orr et al., 2002). Elevated levels of pre-pregnancy symptoms predicted higher preterm delivery among the Coronary Artery Risk Development in Young Adults study population (Gavin et al., 2009) and LBW in a hospital-based sample screened with the Edinburgh Postnatal Depression Scale. A recent meta-analysis of more than 29 studies found antenatal depression and depressive symptoms among participants from varied social settings as significant risks for LBW or PTB outcomes (Grote et al., 2010).

However, most previous investigations did not find a statistically significant association between pre-pregnancy and LBW or PTB outcomes. Pre-pregnancy depressive symptoms were not associated with spontaneous preterm delivery among predominantly black mothers (Phillips et al., 2010) or in the WISH (Women and Infants Starting Healthy) Project in San Francisco (Haas et al., 2005). The Avon Longitudinal Study of Parents and Children, consisting of 14,541 women, did not find evidence to support independent effects of higher depressive symptoms on LBW infants at term (Evans et al., 2007). A plausible explanation for result differences could be the period of measurement, use of different instruments to assess depression, and populations involved (clinical vs. population-based).

This study has several strengths. Numerous exposures associated with birth outcomes were assessed at baseline and follow-up to determine the likelihood of uncontrolled confounders. In particular, depressive symptoms were measured over developmental transitions prior to the index pregnancy and in young adulthood. The prospective design enabled ascertainment of exposures before the occurrence of birth. The large population-based sample studied over a relatively long period of time prior to first births in adulthood enabled assessment of statistical interactions among covariates used. Measurement of depressive symptomatology predictors enabled modeling of changes over the life course.

Limitations of the study are notable. In clinical practice, the CES-D instrument is not used as a diagnostic tool, but to screen for possible risks for depressive disorders (Cicchetti and Toth, 1998; Liem et al., 2008). Therefore, we might have underestimated the burden of depressive symptoms among our sample, particularly in adolescence. Second, assessment of depressive disorders using CES-D was based on self-report. The extent to which other social and psychological factors potentially affected participants' willingness to recall symptoms is unclear. It is also unclear how memory recall could lead to disparities in reporting among age and racial groups. On the one hand, if memory recall was common among our respondents, this is likely to attenuate the relationship between exposure and outcome measures. On the other hand, if depressive symptoms were measured prior to pregnancy and if changes occurred throughout the pregnancy, we were likely to have misclassified the true depressive exposures and birth outcome relationships. Third, the instrument used was not specific to prenatal depression. However, given the high concordance between the Edinburgh Perinatal Depression Scale and CES-D, its validity in assessing pre-pregnancy risks for depressive disorders can be assumed. For example, an evidence report from the Agency for Healthcare Research and Quality on perinatal depression suggests that CES-D and EPDS achieve the highest standards of specificity and sensitivity for depression assessment during pregnancy (Breedlove and Fryzelka, 2011). Fourth, antecedents of PTB (preterm labor, spontaneous membrane rupture, and medically indicated) have distinct etiologies. We had no information to assess these distinct categories of PTB outcomes, so our analysis is likely to result in residual confounding bias of possible determinants of PTB in relation to depressive symptom exposures.

Fifth, *the Add Health data did not include specific pregnancy risk factors including hypertension, preeclampsia and diabetes and were not included in our analyses. Although our study controlled for important and relevant maternal risk factors, we did not include adjustment for pre-pregnancy hypertension, preeclampsia and gestational diabetes in the current pregnancy. We may have overestimated the relation between prepregnancy depressive symptoms and birth outcomes analyzed without adjustment for these covariates. In the analysis, we created categories of depressive symptoms, birth weight and gestational periods. This approach tends to reduce statistical power and therefore our data may likely underestimate the risk for the relationship between prepregnancy depressive symptoms and birth outcomes analyzed in the study*. Finally, findings show that the broader context of income inequality strongly affects women's risk of depression (Nkansah-Amankra et al., 2010, 2013). We did not include these contextual factors. Depressive symptoms assessed in this study might also be a consequence of other psycho pathology, substance use disorders (Colman et al., 2007) or psychiatric conditions (Clark et al., 2010).

National policies to address social inequalities in health will be important for reducing depressive disorders prior to early adulthood and pregnancy. However, given widening health and social inequalities, particularly in the last three decades, it is doubtful that early interventions will be adequate to improve MICH outcomes. Prenatal care and other pregnancy interventions are unlikely to modify the cumulative poor health burden prior to pregnancy, particularly among minority populations. Focusing on childhood development support systems will guide early recognition of triggers of mental health disorders. Our findings reinforce this observation: Assessing the mental health sequelae of adolescents along the life course may have a more significant impact on LBW and PTB outcomes than any other interventions in pregnancy. Applying interventions in adulthood or pregnancy to correct past "physiological insults" might be too late to correct pathological processes associated with adverse birth outcomes. Effective treatment of maternal depressive symptoms greatly benefits children's mental health (Thapar et al., 2012), and early diagnosis and treatment during adolescence and before pregnancy might prove fruitful in improving population health. In conclusion, our analysis suggests that depressive symptoms reported in adolescence might have enduring adverse effects on LBW or PTB in adulthood.

Conflict of interest statement

The authors declare that there are no conflicts of interests.

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