HHS Public Access

Author manuscript

J Dev Behav Pediatr. Author manuscript; available in PMC 2017 January 01.

Published in final edited form as:

J Dev Behav Pediatr. 2016 January ; 37(1): 33–42. doi:10.1097/DBP.0000000000000233.

Postnatal Depressive Symptoms among Mothers and Fathers of Infants Born Preterm: Prevalence and Impacts on Children's Early Cognitive Function

Erika R. Cheng, PhD, MPA[1], **Milton Kotelchuck, PhD, MPH**[2], **Emily D. Gerstein, PhD**[3], **Elsie M. Taveras, MD, MPH**[2], and **Julie Poehlmann-Tynan, PhD**[4]

[1]Indiana University School of Medicine, Department of Pediatrics, Section of Children's Health Services Research, Indianapolis, Indiana

[2]Harvard Medical School and the Massachusetts General Hospital for Children, Division of General Academic Pediatrics, Boston, MA

[3]Department of Psychological Sciences, University of Missouri-St. Louis, St. Louis, Missouri

[4]Department of Human Development and Family Studies, Waisman Center, University of Wisconsin, Madison, Wisconsin

Abstract

Objective—Preterm birth is associated with lower cognitive functioning. One potential pathway is postnatal parental depression. We assessed depressive symptoms in mothers and fathers after preterm birth, and identified the impacts of both prematurity and parental depressive symptoms on children's early cognitive function.

Method—Data were from the nationally-representative *Early Childhood Longitudinal Study-Birth Cohort* (n=5,350). Depressive symptoms at 9 months were assessed by the *Center for Epidemiologic Studies Depression Scale* (CESD) and children's cognitive function at 24 months by the *Bayley Short Form-Research Edition*. Weighted generalized estimating equation models examined the extent to which preterm birth and mothers' and fathers' postnatal depressive symptoms impacted children's cognitive function at 24 months and whether the association between preterm birth and 24-month cognitive function was mediated by parental depressive symptoms.

Results—At 9 months, fathers of very preterm (<32 weeks gestation) and moderate/late preterm (32–37 weeks gestation) infants had higher CESD scores than fathers of term-born (≥37 weeks gestation) infants (p-value=0.02); preterm birth was not associated with maternal depressive symptoms. In multivariable analyses, preterm birth was associated with lower cognitive function at 24 months; this association was unaffected by adjustment for parental depressive symptoms. Fathers', but not mothers', postnatal depressive symptoms predicted lower cognitive function in the fully-adjusted model (β=−0.11, 95% CI: −0.18, −0.03).

Corresponding Author: Erika R. Cheng, PhD, MPA, Department of Pediatrics, Section of Children's Health Services Research, Indiana University School of Medicine, 410 West 10th Street, Suite 1000; Indianapolis, Indiana, 46202; Tel: (317) 278-0505; echeng@iu.edu.

Disclosure: The authors have no conflicts of interest to disclose.

Conclusions—Fathers of preterm infants have more postnatal depressive symptomology than fathers of term-born infants. Fathers' depressive symptoms also negatively impact children's early cognitive function. Our national findings support early identification and treatment of fathers of preterm infants with depressive symptoms.

INTRODUCTION

Preterm birth, defined as gestational age at birth less than 37 weeks, affects approximately 11% of all infants born in the US.[1] Preterm neonates are at risk for neurodevelopmental difficulties and low cognitive function,[2] due to both perinatal complications[2,3] and social risks such as parental education, socioeconomic status, and family well-being.[3,4] Children's cognitive difficulties in infancy and early childhood are correlated with later cognitive and academic outcomes,[5–8] making it especially important to understand the biologic and social contributions to children's development during this critical phase of the life course.

One potential pathway linking prematurity with lower cognitive outcomes is parental postnatal depression. Depression in mothers is a common condition in the postnatal period[9,10] and a documented correlate of a variety of poor developmental outcomes, including impairments in children's cognitive functioning.[11–14] Preterm infants and their families may be more vulnerable to these associations. For example, evidence shows that mothers of preterm infants have a higher risk for psychological stress and depression than mothers of term born children,[15–19] and in turn, early exposure to maternal depressive symptoms appears to have a negative influence on preterm children's later cognitive function.[20]

Less well studied are the bidirectional relationships among prematurity, fathers' postnatal depression, and children's early cognitive function. Like mothers, men undergo significant transitions during the periods of pregnancy and childbirth and there is indication that they too are at risk for depression during the postnatal[21,22] and early childhood periods.[23] A small, but growing body of research also illustrates the connections between varied aspects of father involvement (e.g., active and regular engagement, warmth, participation in caregiving activities, and positive parental control) and enhanced cognitive development of their offspring,[24–26] with some evidence linking father involvement to better cognitive outcomes within the preterm population.[27] Preterm birth is an emotionally traumatizing event that could result in a stressful transition to parenthood and psychosocial stress related to loss of control[28] or fear about the infant's health, development or survival.[29,30] These emotions likely affect fathers' mental health and well-being as well as mothers'; however both the extent to which fathers experience postnatal depressive symptoms after preterm birth and whether their depressive symptoms in turn impact children's early cognitive function are not well understood.

Our aims were to assess depressive symptoms in mothers and fathers after preterm birth, and to identify the impacts of both prematurity and parental depressive symptoms on children's early cognitive function. We postulated that mothers and fathers of infants born preterm would have higher levels of depressive symptoms in the postnatal period than parents of term-born infants, and that these depressive symptoms would in turn be associated with

lower cognitive functioning in children aged 24 months. In view of previous work, we further hypothesized that parental depressive symptoms would mediate the relation between children's preterm status and subsequent cognition, such that in the presence of parental postnatal depressive symptoms, children's preterm status would have a less adverse influence on their cognitive function.

METHODS

Data Source

Data were from the nationally-representative *Early Childhood Longitudinal Study, Birth Cohort* (ECLS-B), a longitudinal, population-based cohort of nearly 10,700 children born in 2001 and their parents. The ECLS-B selected a probability sample of the approximately four million children born in 2001, with oversampling of minority groups, twins and those born at low and very low birthweights, from births registered in the National Center for Health Statistics vital statistics system.[31] The sampling frame excluded births to mothers under 15 years of age and children who were adopted or deceased before the initial data collection wave. The ECLS-B initially sampled more than 14,000 births, and formed the final study cohort (consisting of completed 9 month interviews) of 10,700 when the children were aged approximately 9 months. The weighted response rate for the 24 month data collection wave was 93.1%. The ECLS-B data file provides analytic weights that adjust for disproportionate sampling, survey nonresponse and under-coverage; all analyses for this study were weighted and are representative of US children born in 2001. We obtained restricted data from the US Department of Education, National Center for Education Statistics (NCES) and report all unweighted sample sizes as rounded to the nearest 50 to comply with NCES guidelines. The Partners Human Research Committee at the Massachusetts General Hospital for Children considered this study exempt from review.

Data for this study are from the children's birth certificates and the first two waves of data collection, which occurred when children were approximately 9 and 24 months of age. During these waves, the ECLS-B collected data from the children through direct observation and from their biological mothers and resident and non-resident biological fathers through interviews and self-reported questionnaires. Non-resident biological fathers (e.g., those not currently residing the household with the child) were eligible to participate only if the mother consented to their involvement and reported that they had recent contact themselves or with the child.

Because of the nature of our research question, we limited our sample to only those children with complete data to assess mother and biological father depressive symptoms. Of the 10,700 completed maternal interviews, 9,450 had complete depressive symptom data available at 9 months. Of these, we included only those cases where biological mothers were living with the index child (n = 9,300). We further restricted the sample to include only those mothers with corresponding biological resident or biological non-resident father data, resulting in 6,300 cases. Finally, we excluded 200 cases with missing father depressive symptom data (for 150 resident and 50 non-resident fathers), 100 cases with missing birth certificate information, and 650 cases where the child did not complete the 24 month assessment, yielding a final sample size of 5,350. Mothers and fathers who did not provide

data on depressive symptoms were more likely to be Hispanic, younger, and less educated than parents who completed the instrument so our findings may not be generalizable to these populations.

Measures

Preterm Birth—We extracted gestational age, reported in continuous weeks, from the birth certificate and categorized children's births as very preterm (<32 weeks gestation), moderate/late preterm (32–37 weeks gestation), and term (≥37 weeks gestation).

Depressive Symptoms—At 9 months, mothers and fathers completed the abbreviated *Center for Epidemiologic Studies Depression Scale* (CESD),[32] a 12-item self-administered scale that assesses the frequency of depressive symptoms felt during the previous week. Items included events such as "I felt lonely," and "I could not get going." Each item was coded on a Likert scale between 0 (never) and 3 (often). We combined responses to individual CESD items to create a raw symptom score (range 0–36) with higher scores representing more depressive symptoms. Although not indicative of a diagnosis of clinical depression, scores of 9 or higher (comparable to a score of ≥16 on the full CESD) have been shown to identify major depressive disorder in adults. Hereafter, we refer to scores above this cut point as "high depressive symptoms" or "HDS."[33]

Child Cognition—Cognitive function was assessed at age 24 months using the mental scale of the *Bayley Short Form-Research Edition* (BSF-R),[31] a screening instrument that comprised a subset of items from the revised *Bayley Scales of Infant Development* (BSID-II).[34] The BSF-R mental scale consists of 19 core items, which measured children's early communication skills, memory, expressive and receptive vocabulary, comprehension, and problem-solving abilities.[31,35] BSF-R items were selected from the BSID-II using Item Response Theory (IRT) modeling to approximate full BSID-II results and to facilitate comparisons of BSF-R and BSID-II scores. Selected items were extensively tested to ensure that the psychometric properties of the BSID-II were maintained and that they accurately measured children's performance over the entire ability distribution.

The ECLS-B data file included estimated BSID-II scores (e.g., the predicted number of items a child would have answered correctly were he/she administered the full set of BSID-II), derived from the BSF-R. Higher scores reflect higher cognitive functioning. In theory, the BSF-R mental scores could range from 0–178 (the total number of mental items on the full BSID-II), but all scores fell within these intervals since the items were designed for children from birth through 42 months. At 24 months, the BSF-R mental scale scores for the entire sample ranged from 92 to 174. The range for our sample was 92.6–173.3 (mean=128.3). The IRT reliability coefficient was 0.88.[36]

Covariates—Birth certificates provided the child's sex and plurality status (e.g., singleton versus twin or triplet). We created a flag to identify children whose mothers reported that they had been hospitalized in the neonatal intensive care unit (NICU) after birth because of medical problems. Family factors assessed at 9 months included mothers' and fathers' relationship to each other (married co-resident; not married co-resident; or non-resident

biological father), age in years, and race/ethnicity (non-Hispanic white; non-Hispanic black; non-Hispanic other race; or Hispanic). Household SES was defined by using a composite index generated by NCES that incorporated parental education, income, and occupation;[36] increasing scores reflect higher household SES (range −2.13, 2.18). BSF-R scores were not age normed, so we also measured the child's age at the 24-month assessment which for preterm children was recorded as their chronological age minus the number of weeks preterm.

Analytic Approach

All analyses were conducted using SAS v9.3 and applied appropriate weights to account for the complex sampling design. Study variables, overall and by preterm status, were summarized using descriptive statistics. We used t-tests and chi-squared tests to examine differences in mean continuous parental CESD scores and the dichotomized measure of parental depressive symptoms by child and family factors.

A series of generalized estimating equation models with an independent correlation structure were then used to examine the extent to which preterm birth and mothers' and fathers' depressive symptoms at 9 months were associated with children's cognitive function at 24 months, and whether the association between children's preterm status and 24-month cognitive function was mediated by parental depressive symptoms. CESD scores were treated as a continuous variable for these analyses. We also estimated the models using the dichotomized CESD measure, which yielded similar, but somewhat weaker results (data available upon request).

The first model tested the association between children's preterm status and their cognitive function at 24 months, controlling for child sex, plurality, postnatal hospitalization, the mother-father relationship, maternal age (continuous years), maternal race/ethnicity, household SES, and the child's age at the 24 month assessment. Preliminary analyses revealed that maternal race/ethnicity (r=0.67) and maternal age (r=0.74) were highly collinear with paternal race/ethnicity and paternal age, so we included only maternal factors in these analyses. The second model added both maternal and paternal 9 month CESD scores to test if the effect of preterm status on children's cognitive function was altered after accounting parental postnatal depressive symptoms (i.e., mediation). The relation between preterm status and cognitive function was determined to be mediated by parental depressive symptoms if the regression coefficient for preterm status was attenuated. We adjusted for parental CESD scores simultaneously, as including mothers' and fathers' CESD scores separately yielded similar results (data not shown).

Beta estimates are reported as the mean difference (and 95% confidence interval) in children's cognitive function for each covariate. All models were weighted and accounted for any clustering of twins within families.

RESULTS

Sample characteristics are presented in Table 1. The sample consisted of 5,350 families, 78.6% of which were children living with married parents; we also had data for 500 non-

resident biological fathers (8.3% of the sample). Slightly more than 1 in 10 children were born preterm; of which 1.5% were born very preterm and 8.9% were born moderate/late preterm.

The preterm sample was disproportionately comprised of multiples and infants whose parents were non-Hispanic black race/ethnicity and of lower SES. Post-neonatal hospitalizations were common among the preterm population, reported for 81.6% of children born very preterm and for 39.4% of children born moderate/late preterm, compared to 7.7% of term-born children (p<.0001).

The mean BSF-R score at 24 months among all children was 128.3 (SD=18.9). BSF-R scores were lower among children born very preterm (mean 119.9, SD=23.4) and moderate/late preterm (mean 126.1, SD=18.3) than among term-born children (mean 128.6, SD=17.1; overall p-value <0.001).

Parent Depressive Symptoms at 9 months

Among all parent respondents at child age 9 months, 14.3% of mothers and 11.9% of fathers reported HDS (Table 2). On the continuous CESD measure, mothers had a mean score of 4.5 (SD=7.2) and fathers had a mean score of 4.0 (SD=6.0). Mothers' and fathers' CESD scores showed a moderate positive correlation (Spearman r=0.24, p<.0001), with both parents reporting HDS in 2.7% of the households (data not shown). Fathers' rates of HDS were twice as high when mothers had HDS than when mothers did not have HDS (19.3% versus 9.4%, p<.0001); similarly, twice as many mothers reported HDS when fathers had HDS than when fathers did not have HDS (25.4% versus 12.9%, p<.0001).

Among both parents, the highest rates of HDS were observed among both mothers who identified a non-resident father (26.7%) and the non-resident fathers themselves (29.8%). In both mothers and fathers, higher mean CESD scores were reported when children were hospitalized after birth and among parents who were non-Hispanic black, younger, and of lower SES. Similar associations were noted for the dichotomized CESD measure (Table 2), except that mothers, but not fathers, were more likely to report HDS if their child was a multiple (17.7%) versus singleton (14.2%, p=0.04), and fathers, but not mothers, were more likely to report HDS if their child was hospitalized after birth (15.0% versus 11.5%, p=0.04).

Fathers, but not mothers, also reported more depressive symptoms at 9 months if their child was born preterm (mean CESD score 5.1 [SD=11.3] among fathers of very preterm infants; mean CESD score 4.2 [SD=7.3] among fathers of moderate/late preterm infants; versus mean CESD score 3.9 [SD=6.8] among fathers of term infants, overall p-value=0.02); 21.1% of fathers of very preterm infants and 13.1% of fathers of moderate/late preterm infants reported HDS compared to 11.6% of fathers of term-born infants (overall p-value=0.07). Sub-analyses revealed that within the preterm population (<37 weeks gestation) nearly 40% of non-resident biological fathers had HDS compared to roughly 8% of co-resident, married fathers (data not shown).

Impact of Prematurity and Parent Depressive Symptoms on Cognitive Function

In Table 3, Model 1 showed that being born very preterm was associated with lower cognitive function at 24 months (mean difference= −5.25; 95% CI: −8.05, −2.45), conditional on a host of child and family characteristics. Children who were male (mean difference=−3.84; 95% CI: −4.50, −3.18), multiple births (mean difference=−3.82; 95% CI: −5.10, −2.53), hospitalized after birth (mean difference=−1.53; 95% CI: −2.67, −0.39), lived with unmarried parents (mean difference=−1.14; 95% CI: −2.21, −0.07), and who were racial/ethnic minorities had lower cognitive function than their counterparts, while increasing levels of household SES predicted higher cognitive functioning (mean difference per unit increase of SES=3.17; 95% CI: 2.62, 3.73).

Model 2 additionally included both mothers and fathers' depressive symptoms. Maternal depressive symptoms at 9 months were not significantly associated with cognitive function at 24 months (mean difference=−0.06 per point increase in CESD score; 95% CI: −0.13, 0.02), but paternal CESD scores were inversely associated (mean difference per point increase in CESD score =−0.11; 95% CI: −0.18, −0.03; mean difference for HDS [data not shown] =−1.2; 95% CI: −2.23, −0.06). The addition of parental depressive scores did not attenuate the effect of prematurity, indicating no mediation. That is, both prematurity and fathers' depressive symptoms were independently associated with lower cognitive function in the fully adjusted model.

DISCUSSION

The findings from this national study confirm that depressive symptoms are present in new parents,[37,38] with 14% of mothers and 12% of fathers endorsing clinically significant symptoms 9 months post-term. Although fathers reported lower levels of symptomology than mothers in the overall population, they had higher symptoms following preterm birth, with more than 1 in 5 reporting high depressive symptoms when their children were born before 32 weeks. In turn, children of fathers with higher depressive symptoms had lower cognitive function at age 24 months, even after adjustment for maternal depressive symptoms and children's biologic and social risk factors. These associations were not found for mothers.

The levels of depressive symptoms reported by fathers of preterm infants are consistent with studies indicating that the birth and care of a preterm infant is distressing for fathers.[39–44] Qualitative evidence shows that fathers experience stress, fear, anger, insecurity, and anxiety following preterm birth[40,42] and find it difficult to come to terms with the experience.[41] Fathers of preterm infants may experience more depressive symptoms related to the stressors and challenges associated with the transition to fatherhood (e.g., parental role attainment, competency, prioritizing the roles of partner, father and financial provider), which may be exacerbated in preterm birth.[44]

The opportunity to extend these analyses to a cohort of non-resident fathers – a notable strength of this study – revealed a strikingly high rate (40%) of high depressive symptoms among non-resident fathers following preterm birth. Research shows that having a baby admitted to the NICU, a common occurrence with preterm birth, is a pivotal moment when

fathers prioritize parenthood;[45] this experience may be doubly stressful for non-resident fathers who also face personal and societal expectations about fatherhood that emphasize direct child care and involvement with family life.[46] Non-resident fathers may also be less able to forge physical and emotional relationships with their infants. Regardless of the mechanism, these results indicate that both non-resident and resident fathers of preterm infants may be vulnerable to depressive symptoms and could thus be in need of intervention.

In contrast to fathers, mothers of preterm infants did not have elevated postnatal depressive symptoms as compared to mothers of term-born infants. It is possible that their depressive symptoms resolved prior to the 9 month data collection wave. While several studies document high rates of maternal depressive symptomology and psychological distress immediately following preterm birth,[17,19,43,47] longitudinal analyses confirm that symptoms decrease over time[16,17,29,48,49] with few differences from mothers of term-born infants observed by 8 and 12 months.[19] The availability of postpartum depression screening and referral could play a role. Maternal depression screening is commonly conducted at postpartum visits by pediatricians and treatment may have been recommended long before the CESD was administered at 9 months. Fathers, on the other hand, are rarely the target of postpartum depression referral efforts, which may have contributed to the differences between maternal and paternal depressive symptomology we found here. Fathers also report putting their own needs aside to be supportive of their partners and to provide for their children,[50] as well as difficulties finding counseling support;[45] they might also lack social resources that are known to positively impact maternal depression trajectories.[16] The ECLS-B does not have data to explicitly examine these hypotheses.

We also sought to identify the impacts of both prematurity and postnatal parental depressive symptoms on children's early cognitive function. Not surprisingly, 24-month old children born preterm had worse cognitive function that their term born peers, even after accounting for a wide-range of child and family characteristics. Although we anticipated that mothers' and fathers' postnatal depressive symptoms would each mediate the association between children's preterm status and their later cognitive function, we found no evidence of this. Rather, our findings highlight the importance of understanding the contributions of both prematurity and postnatal paternal depressive symptoms to children's cognitive function.

Many studies suggest that children of mothers who suffer from postpartum depression have worse cognitive function than children of non-depressed mothers, but we found a null effect. However previous associations have been mostly observed in high-risk samples where postnatal depression is accompanied by other family and social problems, suggesting that social risk factors are more influential to children's developmental outcomes than maternal depression per se. For example, Kurstjens and Wolke reported long-term effects of maternal postnatal depression on children's cognitive outcomes only among low SES boys whose mothers were chronically depressed.[51] McManus and Poehlmann found that maternal postnatal depression at 4 months predicted lower cognitive function at 16 months among a sample of preterm infants from Wisconsin, but only for infants whose mothers reported low levels of perceived social support.[20] Tse et al similarly found that the association between maternal depressive symptoms (measured prenatally) and children's cognitive function was largely explained by family sociodemographic characteristics,[52] providing further evidence

for this interpretation. We accounted for several social risk factors in our multivariable model. Another possibility is that the effect of maternal depression on children's cognitive function is more strongly influenced by the chronicity, rather than the timing, of symptoms.[11,51] The CESD was not administered prenatally or during the 24 month data collection wave so we could not account for any timing- or pattern-specific effects. Future longitudinal research should focus on the variable course of maternal depressive symptoms (e.g., timing, chronicity, and severity) to determine their differential consequences for children's cognitive function.

Paternal postnatal depressive symptoms, on the other hand, were more strongly associated with children's subsequent cognitive function than maternal depressive symptoms, further suggesting that the pathways by which postnatal depression influences children's development may differ between mothers and fathers.[53] Although effects were small, the impact of paternal HDS to children's cognitive function was similar in magnitude to having unmarried parents, often viewed as a significant risk factor for children's development. The relation of postnatal depressive symptoms in fathers to children's cognitive function is not entirely understood, but several mechanisms are possible. First, depressive symptoms might have a direct impact on how fathers interact with their children. Depressive symptoms including low mood, irritability, and feelings of hopelessness likely interfere with the ability to provide responsive parenting, which may contribute to lower cognitive function. Studies report that depressed fathers of young children are less likely to engage with their children,[38] are more likely to use aggressive or harsh discipline,[54] and participate in less parent-to-child reading[37] than non-depressed fathers. In turn, markers of father involvement (e.g., reading and play) have been associated with young children's cognitive outcomes, both within the general population[37] and among those born preterm.[27] Indirect pathways are also plausible, for example if depression in fathers leads to marital conflict or compromises maternal affect or involvement.[53] Further, while this study focused on the effects of parental depression on child cognitive skills in children born preterm, future studies should examine the reciprocal or transactional relations between these dyads.[55] Studies into bidirectional relations have historically focused on maternal depression and child behavior,[56,57] but over time, paternal depression may also be influenced by their child's health and cognitive capabilities. It is also important to note that paternal depressive symptoms, while significant, were far less influential to children's cognitive function than their preterm status and certain aspects of their social environment. Efforts to improve children's cognitive function should thus continue to focus on these factors in addition to fathers' postnatal well-being.

Our study has several strengths. We report data from a large, national sample using longitudinal data that are ideal for examining life course transitions for children and their families. In addition to providing a large cohort of children born preterm, the ECLS-B contains widely-used, well-validated measures of depressive symptoms and child development, and also has the advantage of being among the few national data sets available to address research questions regarding fathers.[58] We also acknowledge the following limitations. The CESD items do not parallel diagnostic criteria used to identify depression in a clinical setting, nor do they quantify the duration or frequency of depressive episodes or whether or not depressed parents received treatment. The initial data collection wave, at 9 months, likely missed cases of depression that resolved in the early postnatal period; we did

not have data to ascertain whether depressive symptoms were present before or during pregnancy, which may have biased our findings, particularly if prenatal depressive symptoms have a causal relationship with postnatal depressive symptoms[10] or children's cognitive function.[59] Children's early health problems (e.g., those present before 9 months of age) could have increased the risk of depression in their parents and also played a role in their later cognitive function. However, we accounted for children's postnatal hospitalizations, a proxy for early health and developmental difficulties, which may have minimized this bias. As a sensitivity analyses, we also reran our analyses adjusting for congenital abnormalities, yielding nearly identical results. Birth certificate data are not as precise as medical record data.[60] Finally, the ECLS-B study design called for the child's biological mother to be the respondent for the parent instruments whenever possible; more research is needed to explicitly target fathers who are the primary caregivers of their children.

Clinically, our findings underscore the need to identify and manage postnatal depressive symptoms in both parents, and especially for non-resident fathers and fathers of infants born preterm who may be experiencing clinical levels of depressive symptoms beyond the initial phase following their child's birth. While there has been considerable attention paid to the identification and treatment of postnatal maternal depression,[61–63] little is known about the frequency of efforts to address depression or other health problems among new fathers. However early identification and referral seem justified, particularly in light of our findings that postnatal paternal depressive symptoms may be one factor that delays children's cognitive development.

To our knowledge, the present study is the first to examine the relations among preterm birth, parental postnatal depressive symptoms, and cognitive function using a national cohort of US families that includes data for preterm infants and their resident and non-resident biological fathers. This study builds upon existing research demonstrating negative impacts of fathers' depressive symptoms on children's developmental outcomes[24–26] by offering new findings with respect to depression in fathers of preterm children, as well as among non-resident fathers. Our findings support early identification and treatment of fathers of preterm infants with depressive symptoms.

Acknowledgments

No funder or sponsor had a role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review or approval of the manuscript; or decision to submit the manuscript for publication. This research has been approved by the Partners Human Research Committee at the Massachusetts General Hospital for Children and was supported in part by federal funds from a National Research Service Award institutional training grant (T32HD075727-02; PI: JA Finkelstein).

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J Dev Behav Pediatr. Author manuscript; available in PMC 2017 January 01.

Table 1

Distribution of Study Variables, Overall and by Children's Preterm Status, National Estimates from the Early Childhood Longitudinal Study, Birth Cohort (N=5,350)

		Preterm Status			
	Total N (%)	**Very Preterm**	**Moderate/Late Preterm**	**Term**	**p-value**
Total	5350 (100)	450	900	4000	
Child Factors			%		
Preterm Status					
Very Preterm	450 (1.5)				
Moderate/Late Preterm	900 (8.9)				
Term	4000 (89.6)				
Male Sex	2750 (51.5)	48.3	53.4	51.3	0.60
Twin/Triplet	950 (3.2)	23.0	16.9	1.6	<.0001
Postnatal Hospitalization	950 (7.0)	81.6	39.4	7.7	<.0001
Family Factors					
Father-Mother Relationship					
Co-resident, married	4150 (78.6)	70.9	76.5	79.0	0.25
Co-resident, not married	700 (13.1)	17.4	13.5	13.0	
Nonresident father	500 (8.3)	11.7	9.9	7.9	
Maternal Race/Ethnicity					
Non-Hispanic White	3000 (68.1)	61.4	60.9	68.9	<.0001
Non-Hispanic Black	600 (8.9)	18.0	14.4	8.0	
Non-Hispanic Other Race	1000 (5.6)	3.8	6.4	5.5	
Hispanic	750 (17.4)	16.9	18.3	17.5	
Paternal Race/Ethnicity					
Non-Hispanic White	3050 (67.5)	56.7	62.2	68.2	<.0001
Non-Hispanic Black	550 (8.6)	21.8	14.2	7.7	
Non-Hispanic Other Race	900 (4.5)	2.6	3.7	4.6	
Hispanic	900 (19.4)	18.9	19.9	19.5	
Household Socioeconomic Status					
First quintile (lowest)	650 (11.6)	15.2	15.3	11.1	0.05

	Total N (%)	Preterm Status: Very Preterm	Moderate/Late Preterm	Term	p-value
Second quintile	900 (16.1)	24.7	16.1	15.9	
Third quintile	1100 (20.3)	22.5	19.1	20.4	
Fourth quintile	1200 (25.0)	18.1	24.4	25.2	
Fifth quintile (highest)	1550 (27.0)	19.5	25.0	27.4	
		Mean (SD)			
Child Age, months	24.3 (2.1)	24.4 (3.6)	24.3 (1.9)	24.3 (1.8)	0.42
Maternal Age, years	29.1 (10.1)	29.3 (11.3)	29.6 (8.8)	29.0 (10.0)	0.24
Paternal Age, years	31.3 (10.8)	31.6 (10.7)	31.7 (10.1)	31.3 (10.0)	0.33
Household Socioeconomic Status	0.14 (0.83)	-0.07 (0.84)	0.07 (0.82)	0.15 (0.79)	<.0001
Child's Cognitive Function, 24 months	128.3 (18.9)	119.9 (23.4)	126.1 (18.3)	128.6 (17.1)	<.0001

Notes. Weighted estimates. Percentages may not sum to 100 due to rounding. Unweighted sample sizes were rounded to the nearest 50 in accordance with NCES guidelines. Children's cognitive function at 24 months was measured by the *Bayley Short Form, Research Edition*. SD – standard deviation.

Table 2

Factors Associated with Mothers' and Fathers' Depressive Symptoms at 9 months, National Estimates from the Early Childhood Longitudinal Study, Birth Cohort (N=5,350)

	Maternal CESD Scores				Paternal CESD Scores			
	Mean (SD)	**p-value**	**HDS, %**	**p-value**	**Mean (SD)**	**p-value**	**HDS, %**	**p-value**
Total	4.5 (7.2)		14.2		4.0 (6.0)		11.9	
Child Factors								
Preterm Status								
Very Preterm	4.9 (8.1)	0.16	14.9	0.96	5.1 (11.3)	0.02	21.1	0.07
Moderate/Late Preterm	4.8 (7.2)		13.9		4.2 (7.3)		13.1	
Term	4.5 (7.0)		14.3		3.9 (6.8)		11.6	
Sex								
Male	4.7 (7.1)	0.04	14.8	0.37	3.9 (5.9)	0.87	11.8	0.84
Female	4.0 (6.6)		13.8		3.9 (6.9)		12.0	
Plurality								
Singleton	4.5 (6.8)	0.21	14.2	0.04	3.9 (6.7)	0.94	11.9	0.53
Twin/Triplet	5.0 (7.9)		17.7		3.9 (6.6)		10.8	
Postnatal Hospitalization								
Yes	5.0 (8.5)	0.01	13.7	0.06	4.5 (8.0)	0.002	15.0	0.04
No	4.5 (6.8)		14.4		3.8 (6.9)		11.5	
Family Factors								
Father-Mother Relationship								
Co-resident, married	4.1 (7.8)	<.0001	11.6	<.0001	3.4 (6.0)	<.0001	8.5	<.0001
Co-resident, not married	5.7 (8.0)		22.8		4.4 (7.0)		14.3	
Nonresident father	6.8 (8.7)		26.7		6.6 (7.3)		29.8	
Maternal HDS								
Yes	14.3 (5.0)	<.0001	-		5.8 (8.6)	<.0001	19.3	<.0001
No	2.9 (3.8)		-		3.6 (6.7)		9.4	
Paternal HDS								
Yes	6.8 (8.1)	<.0001	25.4	<.0001	14.2 (3.6)	<.0001	-	
No	4.3 (6.9)		12.9		2.5 (6.2)		-	
Mother/Father Race/Ethnicity								
Non-Hispanic White	4.5 (7.4)	<.0001	14.0	0.02	3.8 (6.4)	<.0001	10.9	0.03
Non-Hispanic Black	5.5 (5.8)		18.7		4.9 (7.6)		16.4	
Non-Hispanic Other Race	4.8 (9.8)		16.8		3.8 (6.8)		9.6	
Hispanic	4.0 (5.0)		12.5		3.8 (6.8)		13	
Mother/Father Age								
15–19	6.5 (8.7)	<.0001	26.4	<.0001	5.3 (7.7)	<.0001	22.5	<.0001
20–24	5.5 (6.2)		19.2		5.0 (6.9)		17.3	
25–29	4.6 (5.6)		14.6		3.9 (6.9)		12.2	
30–34	3.8 (5.9)		10.2		3.2 (5.9)		7.2	
35+	4.1 (7.6)		12.0		3.6 (7.9)		12.0	

	Maternal CESD Scores				Paternal CESD Scores			
	Mean (SD)	**p-value**	**HDS, %**	**p-value**	**Mean (SD)**	**p-value**	**HDS, %**	**p-value**
Household SES								
First quintile (lowest)	5.8 (8.0)	<.0001	22.7	<.0001	4.4 (7.4)	<.0001	17.0	<.0001
Second quintile	5.4 (7.0)		19.7		4.4 (6.5)		14.9	
Third quintile	5.0 (7.5)		15.7		4.2 (5.8)		12.6	
Fourth quintile	4.2 (5.6)		12.1		3.8 (6.8)		10.3	
Fifth quintile (highest)	3.5 (5.2)		8.5		3.1 (5.8)		7.5	

Notes. Weighted estimates. P-values, which indicate statistical significance of differences in child and family factors by maternal and paternal depressive outcomes, are based on t-tests for continuous CESD scores and chi-square tests for the prevalence high depressive symptoms. High depressive symptoms were defined by CESD score ≥9. HDS – high depressive symptoms; CESD – Center for Epidemiologic Studies Depression Scale; SES – socioeconomic status.

Table 3

Mean Difference in Children's 24-month Cognitive Function from Adjusted Regression Models, National Estimates from the Early Childhood Longitudinal Study, Birth Cohort

	Mean Difference in Cognitive Function (95% Confidence Interval)	
	Model 1	**Model 2**
Intercept	88.6 (78.5, 98.8)	89.0 (78.9, 99.1)
Child Factors		
Preterm Status		
Very Preterm	−5.25 (−8.05, −2.45)	−5.24 (−8.04, −2.43)
Moderate/Late Preterm	−0.80 (−1.94, 0.33)	−0.79 (−1.93, 0.35)
Term	Reference	Reference
Sex		
Male	−3.84 (−4.50, −3.18)	−3.82 (−4.48, −3.16)
Female	Reference	Reference
Plurality		
Singleton	Reference	Reference
Twin/Triplet	−3.82 (−5.10, −2.53)	−3.79 (−5.07, −2.51)
Postnatal Hospitalization		
Yes	−1.53 (−2.67, −0.39)	−1.46 (−2.60, −0.32)
No	Reference	Reference
Family Factors		
Father-Mother Relationship		
Co-resident, married	Reference	Reference
Co-resident, not married	−1.14 (−2.21, −0.07)	−1.00 (−2.07, 0.08)
Nonresident father	−0.47 (−1.82, 0.87)	−0.07 (−1.43, 1.29)
Maternal Race/Ethnicity		
Non-Hispanic White	Reference	Reference
Non-Hispanic Black	−3.18 (−4.39, −1.98)	−3.20 (−4.40, −1.99)
Non-Hispanic Other Race	−3.53 (−4.55, −2.50)	−3.51 (−4.53, −2.49)
Hispanic	−4.18 (−5.14, −3.21)	−4.36 (−5.33, −3.40)
Maternal Age	−0.07 (−0.13, 0.00)	−0.07 (−0.14, −0.01)
Household SES	3.17 (2.62, 3.73)	3.10 (2.55, 3.65)
Maternal CESD Scores		−0.06 (−0.13, 0.02)
Paternal CESD Scores		−0.11 (−0.18, −0.03)

Notes. Weighted estimates. Children's cognitive function at 24 months was measured by the Bayley Short Form, Research Edition; CESD – Center for Epidemiologic Studies Depression Scale; SES – socioeconomic status.