JOURNAL OF WOMEN'S HEALTH
Volume 25, Number 7, 2016

DOI: 10.1089/jwh.2015.5462

Original Articles

The Influence of Antenatal Partner Support on Pregnancy Outcomes

Erika R. Cheng, PhD, MPA,[1] Sheryl L. Rifas-Shiman, MPH,[2] Meghan E. Perkins, MPH,[3] Janet Wilson Rich-Edwards, ScD, MPH,[4,5] Matthew W. Gillman, MD, SM,[2] Rosalind Wright, MD, MPH,[6,*] and Elsie M. Taveras, MD, MPH[3,*]

Abstract

Background: While there has been considerable attention given to the multitude of maternal factors that contribute to perinatal conditions and poor birth outcomes, few studies have aimed to understand the impact of fathers or partners. We examined associations of antenatal partner support with psychological variables, smoking behavior, and pregnancy outcomes in two socioeconomically distinct prebirth cohorts.
Materials and Methods: Data were from 1764 women recruited from an urban–suburban group practice (Project Viva) and 877 women from urban community health centers (Project ACCESS), both in the Boston area. Antenatal partner support was assessed by the Turner Support Scale. Multivariable linear and logistic regression analyses determined the impact of low antenatal partner support on the outcomes of interest.
Results: In early pregnancy, 6.4% of Viva and 23.0% of ACCESS participants reported low partner support. After adjustment, low partner support was cross-sectionally associated with high pregnancy-related anxiety in both cohorts (Viva AOR 1.8; 95% CI: 1.0–3.4 and ACCESS AOR 1.9; 95% CI: 1.1–3.3) and with depression in ACCESS (AOR 1.9; 95% CI: 1.1–3.3). In Viva, low partner support was also related to depression mid-pregnancy (AOR 3.1; 95% CI: 1.7–5.7) and to smoking (AOR 2.2; 95% CI: 1.3–3.8). Birth weight, gestational age, and fetal growth were not associated with partner support.
Conclusions: This study of two economically and ethnically distinct cohorts in the Boston area highlights higher levels of antenatal anxiety, depression, and smoking among pregnant women who report low partner support. Partner support may be an important and potentially modifiable target for interventions to improve pregnancy outcomes.

Introduction

Nearly 50% of all infant deaths in the United States (US) are attributable to perinatal conditions, including pregnancy complications, low birth weight, and prematurity.[1] In turn, low birth weight and prematurity, which occur in 8% and 11.4% of all US births, are leading causes of immediate and chronic health problems for children[2,3] and accountable for over 5 billion dollars in annual healthcare costs.[4] While there has been considerable attention given to the multitude of maternal factors that contribute to perinatal conditions and poor birth outcomes (*e.g.*,[5]), fewer studies have aimed to understand the impact of fathers or partners on the perinatal health of their offspring. More research into this understudied area of perinatal health is needed and could have important implications for maternal, infant, and child health. For example, a protective effect of paternal involvement on birth outcomes might suggest that programs and policies could

[1]Section of Children's Health Services Research, Department of Pediatrics, Indiana University School of Medicine, Indianapolis, Indiana.
[2]Obesity Prevention Program, Department of Population Medicine, Harvard Medical School and Harvard Pilgrim Health Care Institute, Boston, Massachusetts.
[3]Division of General Academic Pediatrics, Department of Pediatrics, Massachusetts General Hospital for Children, Boston, Massachusetts.
[4]Connors Center for Women's Health and Gender Biology, Brigham and Women's Hospital, Boston, Massachusetts.
[5]Department of Epidemiology, Harvard School of Public Health, Boston, Massachusetts.
[6]Department of Pediatrics, Kravis Children's Hospital, Mindich Child Health and Development Institute, Icahn School of Medicine at Mount Sinai, New York, New York.
*Co-senior authors.

target their engagement as a key strategy for improving outcomes during the perinatal period.

Research on the role of fathers in perinatal health has tended to focus on direct pathways linking men's health to the health of their offspring. This research demonstrates that factors such as older paternal age, higher anthropometry (*e.g.*, height and weight), and fathers' own birth weight are associated with poor birth outcomes such as low birth weight.[6] Less understood are the indirect social pathways linking fathers to pregnancy outcomes,[7] but research suggests that they are likely to be influential through their impact on maternal health and well-being.[8] For example, evidence suggests that pregnant women with involved partners (*e.g.*, those who listened to the baby's heartbeat, bought things for the baby, and attended childbirth or Lamaze classes) are more likely to receive early prenatal care and to reduce cigarette smoking than women whose partners are not involved in the pregnancy.[9] Social support, a construct related to partner support or involvement, is also an important correlate of maternal well-being during pregnancy and perinatal outcomes, including birth weight.[10]

Existing research focused on father or partner involvement has generally been limited by several factors: (1) the use of surrogate measures of paternal involvement, such as marital status or the presence of the father's name on the birth certificate as indicative of support[11–14]; (2) cross-sectional models that are unable to examine temporal relationships[15]; and (3) retrospective data that are subject to long-term recall and information bias.[9,16] Furthermore, while there has been some research focus on the relationship between antenatal partner support and women's mental health[17,18] and health behaviors[15] during pregnancy, existing work has focused on a relatively narrow range of pregnancy outcomes. No studies to our knowledge have investigated the relationship of antenatal partner support to pregnancy risk factors beyond birth weight and gestational age, including gestational weight gain and fetal growth.

Therefore, we examined the influence of partner support during pregnancy on psychological variables, smoking behavior, and pregnancy risk factors using two socioeconomically distinct cohorts in Boston, Massachusetts. This study addresses the limitations of prior studies by using a longitudinal design and a direct measure of partner support administered to women during pregnancy. We hypothesized that women who report high partner support during pregnancy have better pregnancy outcomes than women with low antenatal partner support and that this relationship is independent of women's demographic, health, and socioeconomic characteristics.

Materials and Methods

This was a secondary analysis of data from Project Viva and Project ACCESS (Asthma Coalition on Community, Environment, and Social Stress), two large prebirth cohorts in the Boston area. Both studies are prospective observational cohorts developed to examine the impact of prenatal health and environmental exposures on maternal and child health outcomes. We capitalized on the fact that both studies were launched at roughly the same time in the Boston area and designed in tandem with complementary protocols and similar survey instruments, which enabled us to examine health effects across a broader range of socioeconomic position and racial/ethnic diversity. The institutional review boards of participating institutions approved the analyses for this study.

Project Viva

Project Viva participants were recruited between 1999 and 2002 at their first prenatal visit to one of eight obstetric practices of Harvard Vanguard Medical Associates, a large group practice in eastern Massachusetts. Most patients in this system are privately insured. Women were eligible to participate in Project Viva if they obtained their first prenatal visit before 22 weeks of gestation, had singleton pregnancies, were able to complete interviews and questionnaires in English, and planned to carry their pregnancy to term and deliver within the Harvard Vanguard system.

Project ACCESS

Between August 2002 and December 2009, Project ACCESS enrolled 955 English and Spanish-speaking women (>18 years old) receiving prenatal care at Brigham and Women's Hospital, Boston Medical Center, three urban community health centers, and their affiliated Women, Infants, and Children (WIC) programs in the Boston metropolitan area and its surrounding suburbs. Project ACCESS enrolled women in mid to late pregnancy (average 28.4 weeks gestation, standard deviation 7.9 weeks); we included women who delivered before September 2008.

Population for analysis

For this study, we included women enrolled in Project Viva and Project ACCESS who completed an early pregnancy questionnaire (Viva, average 10.5 weeks gestation) or a maternal stress questionnaire (ACCESS, within 2 weeks of enrollment) and who had complete data to assess partner support. Our sample of 2641 women included 1764 of 2128 enrolled Project Viva participants and 877 of 1267 enrolled Project ACCESS participants who met these study criteria. Of 2128 women who delivered a live infant in Project Viva, we excluded 364 participants with missing data to assess partner support. Compared with the 1764 mothers included in the analysis, the 364 excluded mothers were younger, less educated, and were more likely to be of nonwhite race/ethnicity. However, included and excluded participants were similar in terms of pregnancy weight gain and infant birth weight and gestation length. Project ACCESS screened 1641 women, of whom 1501 were eligible and 1267 (84% of those eligible) were initially enrolled; of these, 979 (77.2%) gave birth to a live infant and 877 had data to assess partner support.

Measures

Both projects collected data directly from the participants and from medical records. For Project Viva, we used information on partner support, pregnancy-related anxiety, smoking, and sociodemographic measures obtained from the first visit in early pregnancy (average 10.5 weeks gestation), as well as data on antenatal and prepregnancy depressive symptoms assessed during the second visit in mid-pregnancy (average 28 weeks gestation). All data for Project ACCESS were obtained from the mid-pregnancy interview (within 2

weeks of enrollment). We assessed gestational weight gain and birth outcomes by using medical record data.

Partner support. Both cohorts measured partner support by a 4-point Likert scale (0 = strongly disagree to 3 = strongly agree), which asked women to report their level of agreement with the following five statements: (1) I can count on my partner for financial assistance should I need it; (2) My partner is affectionate toward me; (3) My partner will help a lot when the baby comes; (4) My partner understands how I am feeling; and (5) I can count on my partner to be there when I need him/her. This scale has been previously validated among pregnant women in both English and Spanish with good reliability (Cronbach's alpha = 0.89 [English] and 0.94 [Spanish]).[19] Due to the non-normal distribution of responses, we categorized summed responses into groups representing low partner support (score 0–9) and high partner support (score 10–15). For all analyses, women who reported not having a partner ($N = 30$ Project Viva participants and $N = 127$ Project ACCESS participants) were categorized as having low partner support (*e.g.*, score = 0). Removing these women from the analyses had no appreciable impact on our findings (data not shown).

Psychological variables. *Pregnancy-related anxiety.* Women completed a 7-item measure of pregnancy-related anxiety.[20] Items included concern about how the baby is growing and developing and concern about having a hard or difficult labor and delivery. Possible responses were very much, moderately, somewhat, and not at all. Women who had three or more very much responses were coded as having high pregnancy-related anxiety, as consistent with previous work.[21]

Antenatal depressive symptoms. Mothers completed a 10-item Edinburgh Postpartum Depression Scale (EPDS) in pregnancy that queried current depressive symptoms. The EPDS is the only self-reported depression scale that has been validated for prenatal use.[22] Scores ranged from 0 to 30, with higher scores indicating more depressive symptoms. We used a cut point of ≥13 to denote probable depression (hereafter depressed), consistent with our previous research.[23,24]

Smoking during pregnancy. Participants were asked to report their cigarette smoking habits before and during pregnancy. In ACCESS, women reported on smoking at enrollment and in the third trimester; we classified women as prenatal smokers if they reported smoking at either visit. Project Viva queried mothers at both first and second trimester visits about their cigarette smoking habits before and during pregnancy. We compared Project Viva women who smoked during pregnancy with those who never smoked or quit before becoming pregnant.

Pregnancy risk factors. *Gestational weight gain.* We determined gestational weight gain, in kilograms, in Project Viva participants by calculating the difference between the last weight recorded in the medical record before delivery and self-reported prepregnancy weight. Self-reported prepregnancy weight has been previously validated in Project Viva.[25] Project ACCESS did not assess gestational weight gain.

Birth outcomes. We investigated (1) birth weight in grams; (2) gestational age at birth, reported in weeks as the time between the last menstrual period to the date of delivery[†]; and (3) fetal growth, calculated as a birth weight for gestational age *z*-score.[26]

Maternal demographic and socioeconomic factors. Both Project Viva and Project ACCESS collected data on maternal race/ethnicity (non-Hispanic white; non-Hispanic black; non-Hispanic other race; or Hispanic), age in years, gravidity (Project ACCESS), or parity (Project Viva) (hereafter parity: nulliparous vs. primi or multiparous), marital status (married; unmarried/cohabitating; never married; or divorced, separated, or widowed); education (high school degree or less; some college; college degree; or graduate), annual household income (<$20,000; $20,001–40,000; $40,001–70,000; or >70,000), and prepregnancy body–mass index (self-reported weight and measured height, measured in kg/m^2). Prepregnancy depression was measured by the following question: ''Before this pregnancy, was there ever a period of time when you were feeling depressed or down or when you lost interest in pleasurable activities most of the day, nearly every day, for at least two weeks?'' We coded women who endorsed this question as having prepregnancy depression.

Statistical analyses

We obtained means and percentages to describe the sample characteristics, and then used linear and logistic regression to estimate unadjusted and adjusted associations of low partner support with pregnancy outcomes. We used linear regression for continuous outcomes (gestational weight gain, birth weight, gestational age, and fetal growth) and logistic regression for dichotomous outcomes (high pregnancy-related anxiety, antenatal depression, and smoking during pregnancy). We also modeled partner support as a continuous variable, with higher scores indicating more support. The unadjusted model for gestational weight gain controlled for prepregnancy body–mass index (BMI). All adjusted models controlled for maternal race/ethnicity, age, parity, education, prepregnancy BMI, and household income. The models for antenatal depression additionally adjusted for prepregnancy depression. The models for infant birth weight and gestational age additionally adjusted for antenatal smoking, and the model for fetal growth additionally adjusted for antenatal smoking and the infant's gestational age at birth.

We also ran the analyses stratified by race/ethnicity to examine the extent to which the relationships between low partner support and pregnancy outcomes differed by maternal race/ethnicity. The stratified analyses were performed among Project Viva only, owing to small sample sizes in the Project ACCESS cohort.

Results

Table 1 presents sample characteristics of the Project Viva and Project ACCESS cohorts. Project Viva participants were mostly non-Hispanic white (72.3%), married (84.6%), and had a least a college degree (69.6%), with annual household

[†]In Project Viva, about 10% of gestational ages were based on ultrasound estimation.

Table 1. Participant Characteristics and Prevalence of Antenatal Partner Support in Project Viva (N=1764) and Project ACCESS (N=877)

	Sample characteristics		*Project Viva*		*Project ACCESS*	
			Partner support		*Partner support*	
	Viva	*ACCESS*	*High*	*Low*[a]	*High*	*Low*[a]
	N% *or mean (SD)*		N% *or mean (SD)*		N% *or mean (SD)*	
Partner support (%)						
High (score 10–15)	1651 (93.6)	675 (77.0)	—	—	—	—
Low (score 0–9)[a]	113 (6.4)	202 (23.0)	—	—	—	—
Maternal factors						
Race/ethnicity (%)						
Non-Hispanic white	1269 (72.3)	86 (9.9)	1219 (74.3)	50 (44.2)	66 (9.9)	20 (10.1)
Non-Hispanic black	211 (12.0)	284 (32.8)	180 (11.0)	31 (27.4)	211 (31.6)	73 (36.7)
Non-Hispanic other race	168 (9.6)	49 (5.7)	149 (9.1)	19 (16.8)	353 (52.9)	94 (47.2)
Hispanic	106 (6.0)	447 (51.6)	93 (5.7)	13 (11.5)	37 (5.5)	12 (6.0)
Age, years, mean (SD)	32.2 (4.9)	26.9 (5.9)	32.4 (4.8)	29.8 (6.7)	26.8 (6.0)	27.3 (5.8)
Prepregnancy BMI, kg/m^2, mean (SD)	24.6 (5.3)	28.8 (6.4)	24.5 (5.2)	25.2 (6.0)	28.6 (6.1)	29.8 (7.1)
Parity (%)						
0	880 (49.9)	250 (28.9)	832 (50.4)	48 (42.5)	192 (28.8)	58 (29.3)
1+	884 (50.1)	614 (71.1)	819 (49.6)	65 (57.5)	474 (71.2)	140 (70.7)
Marital status (%)						
Married	1484 (84.6)	244 (28.4)	1437 (87.6)	47 (41.6)	219 (33.1)	25 (12.7)
Unmarried, cohabitating	151 (8.6)	263 (30.7)	132 (8.0)	19 (16.8)	232 (35.1)	31 (15.7)
Never married	87 (5.0)	264 (30.8)	49 (3.0)	38 (33.6)	161 (24.4)	103 (52.3)
Divorced, separated, or widowed	37 (1.8)	87 (10.1)	23 (1.4)	9 (8.0)	49 (7.4)	38 (19.3)
Education (%)						
High school or less	160 (9.1)	552 (65.6)	134 (8.2)	26 (23.0)	430 (66.5)	122 (62.6)
Some college	373 (21.3)	206 (24.5)	337 (20.5)	36 (31.9)	144 (22.3)	62 (31.8)
College	660 (37.6)	65 (7.7)	628 (38.3)	32 (28.3)	58 (9.0)	7 (3.6)
Graduate	561 (32.0)	19 (2.3)	542 (33.0)	19 (16.8)	15 (2.3)	4 (2.1)
Annual household income (%)						
<$20,000	49 (3.0)	342 (67.6)	36 (2.3)	13 (13.7)	249 (64.8)	93 (76.2)
>$20,000–$40,000	156 (9.4)	113 (22.3)	134 (8.6)	22 (23.2)	87 (22.7)	26 (21.3)
>$40,000–$70,000	389 (23.5)	35 (6.9)	361 (23.2)	28 (29.5)	32 (8.3)	3 (2.5)
>$70,000	1059 (64.1)	16 (3.2)	1027 (65.9)	32 (33.7)	16 (4.2)	0 (0.0)
Pregnancy outcomes						
Pregnancy-related anxiety (%)						
Low or moderate	1493 (89.9)	637 (75.4)	1408 (90.3)	85 (83.3)	503 (77.3)	134 (69.1)
High	169 (10.2)	208 (24.6)	152 (9.7)	17 (16.7)	148 (22.7)	60 (30.9)
Depression (EPDS ≥13), (%)						
No	1379 (91.4)	668 (78.8)	1318 (92.6)	61 (72.6)	540 (83.1)	128 (64.6)
Yes	129 (8.6)	180 (21.2)	106 (7.4)	23 (27.4)	110 (16.9)	70 (35.4)
Smoking during pregnancy (%)						
No	1567 (88.8)	715 (82.8)	1483 (89.8)	84 (74.3)	556 (83.4)	159 (80.7)
Yes	197 (11.2)	149 (17.2)	168 (10.2)	29 (25.7)	111 (16.6)	38 (19.3)
Gestational weight gain, kg, mean (SD)	15.6 (5.6)	—	15.6 (5.5)	15.6 (6.0)	—	—
Infant birth weight, grams, mean (SD)	3467 (575)	3285 (623)	3476 (575)	3332 (560)	3296 (610)	3245 (668)
Gestational age at birth, weeks, mean (SD)	39.5 (1.9)	39.0 (2.3)	39.5 (1.9)	39.1 (2.2)	39.0 (2.1)	38.9 (2.7)
BW for gestational age z-score, mean (SD)	0.18 (0.95)	–0.42 (1.26)	0.19 (0.95)	–0.01 (0.95)	–0.39 (1.24)	–0.53 (1.31)

[a]Includes women who reported not having a partner.

BMI, body–mass index; EPDS, Edinburgh Postpartum Depression Scale; kg, kilograms; SD, standard deviation.

incomes greater than $70,000 (64.1%); about half were multiparous. Most Project ACCESS participants, in contrast, were non-Hispanic black (32.8%) or Hispanic (51.6%). The majority were multiparous (71.1%). Less than one-third of Project ACCESS participants had educational attainment beyond high school or annual household incomes greater than $20,000.

Factors associated with low partner support

In early pregnancy, 6.4% of Project Viva and 23.0% of Project ACCESS participants reported low antenatal partner support (Table 1). Within both cohorts, pregnant women who reported low partner support were less likely to be married and more likely to be non-Hispanic black and have lower annual household incomes than women who reported high partner support. Within Project Viva, married women experienced higher partner support than unmarried women; within Project ACCESS, married or cohabitating women reported higher partner support than other women. Education was positively associated with partner support in Project Viva; there were few differences in Project ACCESS with regard to partner support by maternal education.

Association of partner support with outcomes

In early pregnancy, high anxiety was noted among 10.2% of Project Viva and 24.6% of Project ACCESS participants (Table 1); 8.6% of Project Viva and 21.2% of Project ACCESS participants reported symptoms indicative of depression at mid- and early pregnancy, respectively. Smoking during pregnancy was more common among Project ACCESS (17.2%) than Project Viva (11.2%) mothers. There were no noticeable differences in infant birth weight or gestational age between the two cohorts.

Table 2 presents the unadjusted and adjusted associations of low partner support with psychological variables, smoking behavior, and pregnancy risk factors. Low partner support in early pregnancy was associated with increased odds of high pregnancy-related anxiety in both cohorts (odds ratio [OR] 1.9; 95% confidence interval [CI]: 1.1–3.2 and OR 1.5; 95% CI: 1.1–2.2 in Project Viva and Project ACCESS, respectively), with antenatal depression during mid-pregnancy (OR 4.7; 95% CI: 2.8–7.9) and smoking during pregnancy (OR 3.0; 95% CI: 1.9–4.8) among Project Viva participants, and cross-sectionally with antenatal depression among Project ACCESS participants (OR 2.7; 95% CI: 1.9–3.8). In Project Viva, low antenatal partner support was also associated with reduced infant birth weight (beta = −144 grams; 95% CI: −254 to −35), gestational age at birth (beta = −0.4 weeks, 95% CI: −0.8 to −0.1), and fetal growth (beta = −0.21; 95% CI: −0.39 to −0.03).

After adjusting for confounders, Project Viva participants who reported low partner support were roughly 80% more likely to have high pregnancy-related anxiety in early pregnancy (adjusted OR [AOR] 1.8; 95% CI: 1.0–3.4) and three times more likely to be depressed mid-pregnancy (AOR 3.1; 95% CI: 1.7–5.7) than Viva participants who reported high partner support. These relationships were also observed in ACCESS, such that low partner support was cross-sectionally associated with nearly double the odds of high pregnancy-related anxiety (AOR 1.9; 95% CI: 1.1–3.3) and antenatal depression (AOR 1.9; 95% CI: 1.1–3.3). In the Project Viva, but not the Project ACCESS cohort, women with low partner support were more likely to smoke during pregnancy (AOR 2.2; 95% CI: 1.3–3.8). Low partner support was not associated with birth weight, gestational age, or fetal growth in either cohort after adjustment or with smoking during pregnancy among Project ACCESS participants.

These same associations were found when assessing the continuous measure of partner support except for between partner support and gestational weight gain in Project Viva. In the adjusted model, each unit increase in the partner

Table 2. Unadjusted and Multivariable Adjusted Associations of Low (Score 0–9 vs. 10–15) Antenatal Partner Support with Pregnancy Outcomes

	Project Viva		*Project ACCESS*	
	Unadjusted	*Adjusted*[a]	*Unadjusted*	*Adjusted*[a]
	OR (95% CI)		*OR (95% CI)*	
Pregnancy outcomes				
High antenatal anxiety	1.9 (1.1, 3.2)	1.8 (1.0, 3.4)	1.5 (1.1, 2.2)	1.9 (1.1, 3.3)
Antenatal depression[b]	4.7 (2.8, 7.9)	3.1 (1.7, 5.7)	2.7 (1.9, 3.8)	1.9 (1.1, 3.3)
Smoking during pregnancy	3.0 (1.9, 4.8)	2.2 (1.3, 3.8)	1.2 (0.8, 1.8)	0.8 (0.4, 1.4)
	β (95% CI)	*β (95% CI)*	*β (95% CI)*	*β* (95% CI)
Gestational weight gain, kg[c]	0.1 (−1.0, 1.1)	0.7 (−0.4, 1.8)	—	—
Infant birth weight, grams[d]	−144 (−254, −35)	−26.2 (−116, 64)	−50.7 (−160, 58)	44.2 (−90, 179)
Gestational age at birth, weeks[e]	−0.4 (−0.8, −0.1)	−0.2 (−0.6, 0.2)	0.0 (−0.4, 0.3)	0.4 (−0.1, 0.9)
Fetal growth[d]	−0.21 (−0.39, −0.03)	−0.10 (−0.29, 0.09)	−0.14 (−0.35, 0.06)	−0.05 (−0.34, 0.24)

Fetal growth is measured as birth weight for gestational age *z* score. We used logistic regression for the following outcomes: high antenatal anxiety, antenatal depression, and smoking during pregnancy. We used linear regression for the following outcomes: infant birth weight, gestational age, and fetal growth.

[a]Adjusted for maternal race/ethnicity, age, parity, education, prepregnancy BMI, and household income.

[b]Adjusted model additionally controls for prepregnancy depression.

[c]Unadjusted model controls for prepregnancy BMI.

[d]Adjusted model additionally controls for antenatal smoking and gestational age at birth.

[e]Adjusted model additionally controls for antenatal smoking.

CI, confidence interval; GA, gestational age; OR, odds ratio.

support scale was associated with 0.14 fewer kg of weight gain during pregnancy (adjusted beta = −0.14 kg; 95% CI: −0.24 to −0.03; data available upon request).

In stratified analyses among Project Viva participants (Supplementary Table; Supplementary Data are available online at www.liebertpub.com/jwh), non-Hispanic white women with low antenatal partner support had increased odds of antenatal depression (AOR 2.7; 95% CI: 1.1–6.4) and smoking during pregnancy (AOR 3.4; 95% CI: 1.6–7.4). Non-Hispanic black women with low partner support had increased odds of antenatal depression compared with their counterparts with high partner support. Among non-Hispanic black mothers, those with low antenatal partner support in early pregnancy had a sevenfold increased likelihood of depression at mid-pregnancy compared with those with high partner support (AOR 7.0; 95% CI: 1.6–30.2).

Discussion

This study of two economically and ethnically distinct cohorts in the Boston area highlights higher levels of antenatal anxiety, depression, and smoking among pregnant women who report low partner support. Within both cohorts—one predominantly nonlow income (Project Viva) and the other predominantly low income (Project ACCESS)—women who reported low support from their partners during pregnancy had increased odds of experiencing antenatal anxiety and depression. Pregnant women within the Project Viva cohort with low partner support were also more likely to smoke, but gained less weight as the level of support from their partners increased. Our multivariable results suggest that differences were most likely not a function of women's sociodemographic factors. Furthermore, stratified analyses in Project Viva revealed significant racial and ethnic differences in these relationships, such that non-Hispanic black women with low partner support had sevenfold increased likelihood of reporting symptoms consistent with antenatal depression than their counterparts with high antenatal partner support. Although we observed a trend toward reduced infant birth weight, gestational age at birth, and fetal growth among Project Viva participants with low partner support, these effects were modestly attenuated after adjustment of maternal characteristics.

Past studies have addressed the question of whether partner or father involvement influences perinatal outcomes, with different and sometimes conflicting findings. Women whose partners are involved in their pregnancies are more likely to receive prenatal care in the first trimester and reduce cigarette smoking.[9,27] Unmarried pregnant women who report being in good relationships with the baby's father report less depressive symptoms, stress, and drug and tobacco use than unmarried pregnant women in poor relationships with the baby's father.[15] Women reporting effective prenatal partner support have lower anxiety in mid-pregnancy[17] and reduced anxiety and depressive symptoms from pregnancy to postpartum.[18,28] Lack of prenatal father involvement, assessed by the absence of paternal information on the birth certificate, is associated with higher rates of neonatal mortality,[12] very and low birth weight, preterm birth, and small for gestational age.[11] Conversely, a recent study among African American women found no association between prenatal partner support and preterm birth, low birth weight, or maternal health behaviors.[29]

Our data support the conclusion that women with low antenatal partner support have worse mental health[17,18] and health behaviors[15] than their peers who report high partner support. This is consistent with some,[29] but not all, of the available research. These differences may reflect the disparate use of indicators and definitions of partner and father involvement used across studies. In our analyses, partner support as measured by self-report and measured on a continuous scale (as opposed to the presence or absence of the fathers' first and/or last name on the birth certificate) was not associated with birth outcomes. This conflicts with findings from the UCLA Environment and Pregnancy Outcomes Study (EPOS), which noted an inverse association between partner support and the likelihood of preterm birth using a self-reported measure of support, although partner support was assessed retrospectively after the baby's birth and therefore subject to recall bias.[16] It is also possible that our analyses were underpowered to find statistically significant differences. Other socioeconomic and health factors that varied across studies may also explain the differences; future research is warranted.

We did not find strong associations for pregnancy risk factors nor with fetal growth, but low partner support was associated with higher levels of antenatal anxiety, depression, and tobacco use, even after controlling for sociodemographic factors. In Project Viva, we also found some evidence that partner support may be inversely associated with gestational weight gain, which to our knowledge has not been previously reported in the literature. There are several plausible explanations for our findings. As discussed by Misra,[7] factors such as fathers' attitudes and intentions regarding the pregnancy, own prenatal health behaviors, and the mother–father relationship likely influence maternal health and well-being during the perinatal period. Partners are one of the most important influences on women's smoking habits during pregnancy[30,31] and there is evidence that fathers' smoking intensifies the effect of mothers' smoking on infant birth weight.[32] Fathers may provide financial support to pregnant mothers, which is associated with higher rates of prenatal care and lower rates of prenatal substance use.[27] It is also possible that women experiencing high anxiety or depressive symptoms in pregnancy may be more likely to appraise lower support received from their partner. However, this would not necessarily explain our findings for tobacco use or gestational weight gain.

Low antenatal partner support among non-Hispanic black mothers in the Project Viva cohort was associated with sevenfold higher odds of antenatal depression. A previous study of Project Viva participants suggests that the increased prevalence of depressive symptoms among pregnant Black and Hispanic women relative to non-Hispanic white women may be attributed, in part, to socioeconomic factors.[23] Racial/ethnic minorities and foreign-born mothers may also be less likely than non-Hispanic white mothers to seek medical care for their emotional problems.[33] Regardless of the mechanism, this finding suggests that partner support may have salience with regard to racial/ethnic disparities in pregnancy outcomes. Targeting fathers has been proposed as a key strategy to mitigate racial and ethnic disparities in birth outcomes,[34,35] and further inquiry into the role of the partner and fathers during pregnancy on such disparities is needed.[7,11,34] However, we note that this finding should be interpreted with caution, owing to the small sample sizes for

analysis. More research is needed to replicate our work using a larger sample.

The primary strength of this study is the unusual opportunity to examine the prevalence and impact of low antenatal partner support using identical study instruments within two simultaneous and sociodemographically distinct populations in Boston. Even in these very different populations, antenatal partner support was shown to have an important impact on the anxiety and depression women identify. Our prospective design and measure of partner involvement are notable advances over existing work that has been almost exclusively cross-sectional and reliant on proxy measures (*e.g.*, birth certificates) of fathers' involvement. Importantly, we believe ours is the first study to report on the relationship between antenatal partner involvement and gestational weight gain and fetal growth. We also note several limitations. Although our analysis of partner support and some pregnancy outcomes was prospective in design, our main findings of the relationship between partner support and depressive symptoms (in ACCESS) and anxiety (in both cohorts) are cross-sectional, so we cannot infer causality. Most measures were self-reported, which may introduce bias, particularly if anxious or depressed women were more likely to report low partner support. However, we controlled for prepregnancy depression in our analysis, which may have minimized this potential bias. Self-reported prepregnancy weight has been previously validated in Project Viva[25] and Project ACCESS.[36] Partner support is difficult to define and quantify and the summary score we used may not have captured specific aspects of support relevant to perinatal health or cross-cultural differences therein. We did not evaluate differential effects of specific types of support (*e.g.*, financial vs. emotional) on our outcomes of interest. Project Viva and Project ACCESS study protocols limited participants to English or Spanish-speaking women, so our findings may not be generalizable to other more diverse samples of women. The EDPS items do not parallel diagnostic criteria used to identify depression in a clinical setting. The EDPS also does not quantify the duration or frequency of depressive episodes or whether or not depressed mothers received treatment. Finally, we had limited power to address effect modification and were unable to perform these analyses in the Project ACCESS cohort.

In conclusion, our findings add to mounting evidence that fathers and partners are influential in maternal psychological variables and smoking behavior during pregnancy. We noted similar findings across two birth cohorts with different and diverse social determinants of health, suggesting that partner support may be germane to all childbearing women. This has practical implications for feasible interventions aimed to promote optimal family wellness during childbearing transitions. It may be important to target low antenatal partner support as a potentially modifiable target for interventions to improve perinatal outcomes. Expanding women's access to evidence-based programs (*e.g.*, doulas and Centering Pregnancy group prenatal care) may also improve psychosocial and obstetric birth outcomes when partner support is lacking.

Acknowledgments

This research has was supported, in part, by federal funds from NIH/NIMHD (R01MD003963; PI: E.M.T.) and a National Research Science Award (T32HD075727-02; PI: J.A.F.).

Author Disclosure Statement

No competing financial interests exist.

References

1. Murphy SL, Xu J, Kochanek KD. Deaths: Final data for 2010. National vital statistics reports: From the Centers for Disease Control and Prevention, NCHS. NVSS 2013; 61:1–117.
2. Bhushan V, Paneth N, Kiely JL. Impact of improved survival of very low birth weight infants on recent secular trends in the prevalence of cerebral palsy. Pediatrics 1993; 91:1094–1100.
3. McCormick MC. The contribution of low birth weight to infant mortality and childhood morbidity. N Engl J Med 1985;312:82–90.
4. Russell RB, Green NS, Steiner CA, Meikle S, Howse JL, Poschman K, et al. Cost of hospitalization for preterm and low birth weight infants in the United States. Pediatrics 2007;120:e1–e9.
5. Kramer MS. Determinants of low birth weight: Methodological assessment and meta-analysis. Bull World Health Organ 1987;65:663–737.
6. Shah PS. Paternal factors and low birthweight, preterm, and small for gestational age births: A systematic review. Am J Obstet Gynecol 2010;202:103–123.
7. Misra DP, Caldwell C, Young AA, Abelson S. Do fathers matter? Paternal contributions to birth outcomes and racial disparities. Am J Obstet Gynecol 2010;202:99–100.
8. Alio AP, Bond MJ, Padilla YC, Heidelbaugh JJ, Lu M, Parker WJ. Addressing policy barriers to paternal involvement during pregnancy. Matern Child Health J 2011;15: 425–430.
9. Martin L, McNamara M, Milot A, Halle T, Hair E. The effects of father involvement during pregnancy on receipt of prenatal care and maternal smoking. Matern Child Health J 2007;11:595–602.
10. Elsenbruch S, Benson S, Rucke M, Rose M, Dudenhausen J, Pincus-Knackstedt MK, et al. Social support during pregnancy: Effects on maternal depressive symptoms, smoking and pregnancy outcome. Hum Reprod 2007;22:869–877.
11. Alio AP, Kornosky JL, Mbah AK, Marty PJ, Salihu HM. The impact of paternal involvement on feto-infant morbidity among whites, blacks and hispanics. Matern Child Health J 2010;14:735–741.
12. Alio AP, Mbah AK, Kornosky JL, Wathington D, Marty PJ, Salihu HM. Assessing the impact of paternal involvement on racial/ethnic disparities in infant mortality rates. J Commun Health 2011;36:63–68.
13. Gaudino JA, Jr, Jenkins B, Rochat RW. No fathers' names: A risk factor for infant mortality in the State of Georgia, USA. Soc Sci Med 1999;48:253–265.
14. Ngui E, Cortright A, Blair K. An investigation of paternity status and other factors associated with racial and ethnic disparities in birth outcomes in Milwaukee, Wisconsin. Matern Child Health J 2009;13:467–478.
15. Bloch JR, Webb DA, Mathews L, Dennis EF, Bennett IM, Culhane JF. Beyond marital status: The quality of the mother–father relationship and its influence on reproductive health behaviors and outcomes among unmarried low income pregnant women. Matern Child Health J 2010;14:726–734.

16. Ghosh JK, Wilhelm MH, Dunkel-Schetter C, Lombardi CA, Ritz BR. Paternal support and preterm birth, and the moderation of effects of chronic stress: A study in Los Angeles county mothers. Arch Womens Ment Health 2010; 13:327–338.
17. Rini C, Schetter CD, Hobel CJ, Glynn LM, Sandman CA. Effective social support: Antecedents and consequences of partner support during pregnancy. Pers Relationship 2006; 13:207–229.
18. Stapleton LR, Schetter CD, Westling E, Rini C, Glynn LM, Hobel CJ, et al. Perceived partner support in pregnancy predicts lower maternal and infant distress. J Fam Psychol 2012;26:453–463.
19. Turner RJ, Grindstaff CF, Phillips N. Social support and outcome in teenage pregnancy. J Health Soc Behav 1990: 43–57.
20. Rini CK, Dunkel-Schetter C, Wadhwa PD, Sandman CA. Psychological adaptation and birth outcomes: The role of personal resources, stress, and sociocultural context in pregnancy. Health Psychol 1999;18:333–345.
21. Fairlie TG, Gillman MW, Rich-Edwards J. High pregnancy-related anxiety and prenatal depressive symptoms as predictors of intention to breastfeed and breastfeeding initiation. J Womens Health (Larchmt) 2009;18:945–953.
22. Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression. Development of the 10-item Edinburgh Postnatal Depression Scale. Br J Psychiatry 1987;150:782–786.
23. Rich-Edwards J, Kleinman K, Abrams A, Harlow B, McLaughlin T, Joffe H, et al. Sociodemographic predictors of antenatal and postpartum depressive symptoms among women in a medical group practice. J Epidemiol Community Health 2006;60:221–227.
24. Rich-Edwards JW, James-Todd T, Mohllajee A, Kleinman K, Burke A, Gillman MW, et al. Lifetime maternal experiences of abuse and risk of pre-natal depression in two demographically distinct populations in Boston. Int J Epidemiol 2011;40:375–384.
25. Oken E, Taveras EM, Kleinman KP, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. Am J Obstet Gynecol 2007;196:322 e1–322 e8.
26. Oken E, Kleinman KP, Rich-Edwards JW, Gillman MW. A nearly continuous measure of birth weight for gestational age using a United States national reference. BMC Pediatr 2003;3:6.
27. Teitler JO. Father involvement, child health and maternal health behavior. Children Youth Serv Rev 2001;23:403–425.
28. Dunkel Schetter C. Psychological science on pregnancy: Stress processes, biopsychosocial models, and emerging research issues. Annu Rev Psychol 2011;62:531–558.
29. Straughen JK, Caldwell CH, Young AA, Misra DP. Partner support in a cohort of African American families and its influence on pregnancy outcomes and prenatal health behaviors. BMC Pregnancy Childbirth 2013;13:187.
30. Flemming K, Graham H, Heirs M, Fox D, Sowden A. Smoking in pregnancy: A systematic review of qualitative research of women who commence pregnancy as smokers. J Adv Nurs 2013;69:1023–1036.
31. Chamberlain C, O'Mara-Eves A, Oliver S, Caird JR, Perlen SM, Eades SJ, et al. Psychosocial interventions for supporting women to stop smoking in pregnancy. Cochrane Database Syst Rev 2013;10:CD001055.
32. Haug K, Irgens LM, Skjaerven R, Markestad T, Baste V, Schreuder P. Maternal smoking and birthweight: Effect modification of period, maternal age and paternal smoking. Acta Obstet Gynecol Scand 2000;79:485–489.
33. Huang ZJ, Wong FY, Ronzio CR, Yu SM. Depressive symptomatology and mental health help-seeking patterns of U.S.- and foreign-born mothers. Matern Child Health J 2007;11:257–267.
34. Lu MC, Jones L, Bond MJ, Wright K, Pumpuang M, Maidenberg M, et al. Where is the F in MCH? Father involvement in African American families. Ethn Dis 2010;20: S2–S49.
35. Lu MC, Kotelchuck M, Hogan V, Jones L, Wright K, Halfon N. Closing the Black-White gap in birth outcomes: A life-course approach. Ethn Dis 2010;20:S2-62-76.
36. Wright RJ, Fisher K, Chiu YH, Wright RO, Fein R, Cohen S, et al. Disrupted prenatal maternal cortisol, maternal obesity, and childhood wheeze. Insights into prenatal programming. Am J Respir Crit Care Med 2013;187:1186–1193.

Address correspondence to:
Elsie M. Taveras, MD, MPH
Division of General Academic Pediatrics
Department of Pediatrics
Massachusetts General Hospital for Children
125 Nashua Street, Suite #860
Boston, MA 02114

E-mail: elsie.taveras@mgh.harvard.edu