Published in final edited form as:
Am J Obstet Gynecol. 2011 May ; 204(5): 431.e1–431.e8. doi:10.1016/j.ajog.2011.01.054.

The association between early life adversity and bacterial vaginosis during pregnancy

Alison L. Cammack, MPH[a],[b], **Claudia Buss, PhD**[a], **Sonja Entringer, PhD**[a], **Carol J. Hogue, PhD**[b], **Calvin J. Hobel, MD**[c], and **Pathik D. Wadhwa, MD, PhD**[a],[d],[e]

[a] Department of Psychiatry and Human Behavior, School of Medicine University of California, Irvine

[b] Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, GA

[c] Division of Maternal-Fetal Medicine, Cedars-Sinai Medical Center, Los Angeles, CA

[d] Department of Obstetrics and Gynecology, School of Medicine University of California, Irvine

[e] Department of Pediatrics, School of Medicine University of California, Irvine

Abstract

Objective—The purpose of this study was to examine associations between chronic preconception psychosocial and socioeconomic stress with bacterial vaginosis (BV) during pregnancy.

Study Design—Using univariate and multivariate logistic regression, childhood abuse and neglect, chronic discrimination, childhood socioeconomic status, potential confounders, and BV were assessed at 14-16 and 19-22 weeks gestation in a cohort of 312 pregnant women.

Results—Persistent BV (BV positive at both time points versus no BV at either time point) was associated with childhood sexual abuse (CSA), chronic discrimination, and lack of parental home ownership. These associations were still present after covarying for current perceived stress, socioeconomic status, and other potential confounders.

Conclusion—There is evidence that BV during pregnancy is independently linked with early life psychosocial adversity, suggesting that a life course perspective may be important in elucidating determinants of perinatal outcomes.

Keywords

bacterial vaginosis; child abuse; life course; pregnancy; psychosocial stress

Corresponding author: Pathik D. Wadhwa, MD, PhD., UC Irvine Development, Health and Disease Research Program, University of California, Irvine, School of Medicine, 3117 Gillespie Neuroscience Research Facility, Zot Code 4260, Irvine, CA 92697, Tel: (949) 824-8238, Fax: (949) 824-8218, pwadhwa@uci.edu.

Presented, in part, at the 22nd annual meeting of the Society for Pediatric and Perinatal Epidemiologic Research, Anaheim, CA, June 22-23, 2009

Introduction

Bacterial vaginosis (BV) is the most common reproductive tract infection in women of childbearing age, with a prevalence of 10-40% during pregnancy.[1] Although various risk factors have been identified, including African American race, marital status, low socioeconomic status (SES), young age, douching, substance use, and sexual practices,[2] a large proportion of the variance remains unexplained.[3] Studies have consistently shown that BV is associated with a two-fold increased risk of preterm birth and higher risks associated with early preterm birth.[2] Because preterm birth is the leading cause of infant mortality and childhood disability, research pertaining to mediators such as BV is a public health priority.[4] Such research is especially important since antibiotic trials have not shown consistent reductions for the risk of preterm birth.[5]

A potential risk factor that is a biologically plausible cause of BV is stress. A large body of literature from animal and human studies has established that psychosocial stress impacts immune and endocrine function and can increase susceptibility to infection.[6,7] Although pregnancy-specific evidence is more limited, some studies report that women with increased levels of psychosocial stress have elevated levels of serum pro-inflammatory cytokines and cortisol.[8-11] Further, behavioral sequelae of psychosocial stress, such as smoking, are risk factors for BV. Other stressors, such as those of a socioeconomic nature, have also been consistently associated with BV.[2] The stress-BV association is of particular interest, as stress is an increasingly recognized risk factor for preterm birth, and it has been postulated that infection and/or immune pathways may mediate this association.[12]

Despite the plausibility of a relationship between stress and BV, pregnancy-specific literature remains inconclusive. Among non-pregnant women, perceived stress has been associated with incident BV.[13] However, no studies among pregnant women have clearly established a temporal relationship between various measures of stress and the development of BV, and the results of cross-sectional studies are mixed. Regarding perceived stress, one study reported a significant positive association,[3] whereas two other studies did not replicate this finding.[14,15] Other stress-related constructs, including state and trait anxiety, life events, social support, and hassles have not been independently associated with BV.[14,16] Few studies have explicitly examined stressors of a chronic nature, although Culhane [17] examined community stressors and reported that residing in neighborhoods with high homelessness rates was associated with BV.

In interpreting this literature, it may be relevant to consider that the timing and chronicity of a given stressor may influence the strength of its relationship to perinatal outcomes.[11,12,18] Regarding timing, psychological and physiological reactivity to stressors has been shown to progressively decrease as gestation advances.[19,20,21] This implies that stress exposures before pregnancy or early in pregnancy may exert greater effects than those later in gestation. Also, childhood SES may make unique contributions to or interact with adversity during adulthood to explain adult health outcomes, including perinatal outcomes.[22] Regarding chronicity, there is evidence that immune and endocrine alterations in the context of chronic stress are different than those of acute stress, specifically that acute stress may lead to heightened immune responses, whereas chronic stress may lead to immune suppression.[23]

Given the limited literature specifically concerning chronic stressors, particularly those pertaining to the preconception period, the goal of this study was to assess their association with BV during pregnancy. It was hypothesized that stressors of a chronic nature with an onset in early life are more strongly associated with bacterial vaginosis than acute psychosocial or socioeconomic stress during pregnancy.

Materials and Methods

Participants

Participants were self-identified African American, Hispanic, or non-Hispanic White women ≥ 16 years old with singleton pregnancies recruited from two sites in Southern California: the University of California, Irvine Medical Center in Orange, CA and Cedars-Sinai Medical Center in Los Angeles, CA, between September 2004 and July 2007. All participants provided written informed consent and study procedures were approved by the Institutional Review Boards of the two respective institutions. Women with cord, placental, or uterine anomalies, fetal congenital malformations, conditions that may dysregulate neuroendocrine and/or immune function, those who had received corticosteroid therapy in the three months preceding enrollment, or those whose pregnancies ended in a spontaneous abortion were ineligible for the study.

Procedures

Study visits occurred at approximately 14-16 (T1: Mean GA=15.2 weeks, SD= .81) and 19-22 (T2: Mean GA= 20.5 weeks, SD= .84) weeks of gestation. Study assessments included structured psychosocial and medical interviews, questionnaire administration, fetal biometry ultrasound, a speculum examination for collection of vaginal fluid and BV assessment, and the collection of venous blood, saliva and urine. Gestational age was determined through a combination of last menstrual period and obstetric ultrasonographic biometry before 16 weeks GA, using standard clinical criteria.[24]

Measures

BV Measure—BV status was assessed by microscopic evaluation of air dried vaginal fluid slides, using Nugent scoring,[25] the epidemiologic gold standard for assessing BV. All slides were read by a single expert reviewer (Janice French, CNM, MSN), who was blinded to participant characteristics. For these analyses, BV was dichotomized into present (score 7-10) versus absent (score 0-6). Exclusion of intermediate scores (score 4-6) from the absent category yielded similar results. Because there was greater *a priori* interest in chronic exposures rather than acute exposures, the outcome of interest was persistent BV, which compares women who were BV+ at both time points versus those who were BV-at the two assessments. Further, very few women changed BV status between time points, limiting analyses of incident and remissive BV.

Psychosocial and Socioeconomic Measures—Various measures were used to capture the domains of psychosocial and socioeconomic stress. First, childhood trauma was measured with the Childhood Trauma Questionnaire Short-Form.[26] This widely utilized scale assesses five domains of childhood trauma: physical abuse, emotional abuse, sexual abuse, physical neglect, and emotional neglect. Each item is measured on a five- point likert scale, measuring the frequency or severity of each exposure. Each trauma type was dichotomized into present versus absent based on cut points suggested to minimize the false identification of trauma.[26] Another measure of chronic stress was operationalized as discrimination, based on the Everyday Discrimination Scale.[27] This nine-item scale measures the frequency of day-to-day experiences of unfair treatment on a six-point likert scale (range: 0=never to 6=almost everyday). Questions are asked without reference to race or ethnicity; rather the measure assesses generalized unfair treatment. Although commonly used in homogeneous minority samples, this scale has also been used in ethnically diverse studies.[28] A contrasting measure of more recently-experienced stress was measured with the fourteen-item version of Cohen's Perceived Stress Scale (PSS)[29] at both study assessments, which assesses the degree to which situations in the previous month are viewed as stressful on a five-point likert scale (range: 1=never to 5=very often). All scales demonstrated good

internal validity (Cronbach's alpha: T1 PSS: .86, T2 PSS: .86; discrimination: .85, childhood emotional abuse: .85, childhood physical abuse: .85, childhood sexual abuse: .94, childhood emotional neglect: .90, childhood physical neglect: .71). Scales other than the childhood trauma questionnaire were examined as both continuous and dichotomous exposures, where the upper 25th percentile was chosen to distinguish between elevated versus low/normal levels. Childhood SES was measured as parental home ownership before age 16 (yes/no). This specific measure was chosen because compared to other retrospective assessments of SES such as income, adults are generally able to recall whether their parents owned homes when they were growing up.[30] Current SES was assessed with household income in the last year, measured on an eleven-point ordinal scale ranging from less than $5000 to $100,000 or more; adjusting for the number of persons in the household yielded similar point estimates. Current SES was also measured as education, categorized as less than high school, high school degree with or without vocational education, some college, and bachelor's degree or higher.

Covariates—Covariates and potential confounders were identified from a literature review. Measures collected through maternal self-report included race/ethnicity, age, marital status, parity, illicit drug use during pregnancy, lifetime number of sexual partners, age at first sexual intercourse, frequency of sexual intercourse during pregnancy, and antibiotic use from the beginning of pregnancy through the second study assessment. Current maternal SES (i.e., education and income as described previously) was also considered as a covariate/potential confounder, in addition to a main effect. Smoking status during pregnancy was determined by self-report and cotinine assays.[31] Prenatal antibiotic use was ascertained through self-report and prenatal medical record abstraction. Continuous and categorical classifications based on literature-defined cut points were considered; also the distribution of each variable and collinearity considerations influenced categorizations for multivariate modeling.

Statistical Analyses—The modeling strategy employed several steps. First, the distribution of each variable was examined and values were checked for normality and plausibility. Second, relationships between persistent BV, stressors, and covariates were examined using chi-square and Fisher's exact tests as appropriate. Crude and adjusted associations between persistent BV and stressors were quantified as odds ratios (OR) with 95% confidence intervals (CI) using multivariate unconditional logistic regression; statistical significance was inferred for CIs not containing the value 1. Due to the relatively large number of considered covariates, variable screening was employed by examining the relationship between each individual covariate/confounder and persistent BV. Covariates associated with persistent BV at $p<.15$ were considered for inclusion in multivariate models. Further, covariates not significantly associated with BV were added to models to see if they changed the point estimate by more than 10%. Collinearity was assessed by computation of condition indices and variance decomposition proportions (VDPs), using condition indices >20 and VDPs >.5 as an indication of a collinearity problem. Goodness-of-fit was assessed with the Hosmer-Lemeshow test. Analyses were conducted in SAS 9.2 and SPSS 15.0.

Results

BV was slightly more prevalent at T1 than T2 (17.3% vs. 15.9%, respectively; difference not statistically significant). Among those who had BV data from both visits, BV status remained highly stable. Thirty-seven women (13.0%) were BV+ at T1 and T2 and 229 women (80.4%) were BV- at both assessments. Only 8 women acquired BV from T1 to T2, and 11 women experienced remission of BV. Participants who were missing BV data at either time point (n=27) did not have significantly different baseline characteristics from those with BV data at both time points.

The distribution of exposures and a description of the study population are included in Tables 1 and 2, respectively. The largest proportion of participants was Hispanic, followed by non-Hispanic whites, then African Americans. Approximately half were married, with nearly equal numbers of unmarried participants in the two cohabitation categories. Participants averaged 28.8 years of age (SD=5.9), were mostly parous, and there were few substance users. PSS scores were similar for both study assessments. A substantial minority of participant's parents did not own a home and reported exposure to each of the five categories of childhood trauma.

Table 2 also shows bivariate relationships between covariates/potential confounders and persistent BV. Persistent BV was significantly more common among women who were younger, African American or Hispanic (relative to non-Hispanic white), not cohabitating with the baby's father, earlier age at first sexual intercourse, and drug users. There was a trend among women who smoked during pregnancy (Fisher's exact test p=.11)

Results of the crude and adjusted relationships between individual psychosocial and socioeconomic stress relationships are displayed in Table 3. All presented models showed good fit (Hosmer-Lemeshow chi-square p>.05). Continuous discrimination scores were significantly positively associated with persistent BV and adjustment for covariates/ confounders did not alter the point estimate. Similarly, there was a marginal association for elevated discrimination scores that was not attenuated in the multivariate model. Among the childhood trauma measures, only childhood sexual abuse (CSA) was significantly positively associated with persistent BV; this association was found in crude and adjusted models. By contrast, continuous and elevated current perceived stress at T1 and T2 was marginally associated with persistent BV in crude models, but the association was appreciably attenuated and not statistically significant in multivariate models. Regarding socioeconomic adversity, current household income, education, and lack of parental homeownership were all significantly associated with persistent BV in crude models, but the associations were attenuated and only lack of parental home ownership remained statistically significant in adjusted models. The magnitude of some associations appeared different when stratified on race, including CSA (OR for blacks=1.65, OR for non-blacks= 2.93), elevated perceived discrimination (OR for blacks=2.36, OR for non-blacks=1.52), and most notably, lack of parental home ownership (OR for blacks=2.72, OR for non-blacks=12.97).

Models examining independent effects of stressors that were individually associated with persistent BV were also considered (i.e., all constructs associated with persistent BV in individual models were simultaneously entered as independent variables in a single model). Table 4 displays these adjusted associations for one model. Models with alternate categorizations and/or time points for PSS and discrimination yielded similar results. In comparison to crude associations, point estimates for childhood homeownership and current income were reduced, but still remained highly statistically significant. Associations with CSA and discrimination were slightly strengthened. Effects for PSS and education were strongly attenuated and no longer statistically significant. Lack of parental home ownership showed the strongest effect sizes (OR range: 6.8-8.0, depending on categorizations/time point for PSS and discrimination).

Comment

These data support the hypothesis that early life adversity is associated with bacterial vaginosis in pregnancy. This is the first study to report associations with childhood trauma, chronic discrimination, and childhood socioeconomic status. Three mechanisms may explain these findings: early exposure to adversity programs physiology in a way that increases susceptibility to BV (i.e., early life programming[32]), insults in early life lead to repeated

exposure to stress and chronic activation of the neuroendocrine stress responses, leading to physiological wear and tear (i.e., allostatic load [23]), or unmeasured and/or residual confounding.

The associations between individual stressors and persistent BV are plausible given other reported findings. First, regarding CSA, there is a substantial literature linking it with distant psychopathologies,[33,34] including studies involving pregnant women.[35,36] CSA has also been associated with physical adult outcomes, including stress-sensitive conditions, such as obesity, gastrointestinal disorders, and chronic fatigue syndrome,[37-39] and timing of reproductive milestones, such as age at menarche.[40] Further, CSA has been associated with preterm delivery;[41] BV may serve as a mediator of this relationship. Also, the sole positive association between CSA and BV makes sense in light of studies reporting stronger effects of CSA, relative to other traumas, on adolescent and young adult psychopathology[42] and evidence showing that rape and childhood molestation are the traumas most likely to cause post-traumatic stress disorder in adult women.[43]

Regarding chronic perceived discrimination, the present findings are in line with other studies reporting associations with other health conditions, including abdominal obesity,[28] cardiovascular disease,[44] and depression and anxiety.[45] Concerning preterm birth, most studies have focused on racism and report positive associations.[46] When reported in exclusively African American populations or among studies stratified by race, associations were limited to African Americans, which is in line with our findings. Unfortunately, we did not have information on the source of discrimination, precluding a more direct comparison of racism.

In addition to evidence of childhood SES as a risk factor for various health outcomes, it has been associated with infection and inflammation. Lack of parental homeownership, the specific measure utilized in our study, has been linked with subsequent susceptibility to infection and epigenetic inflammatory processes independent of adult SES.[30,47] Further, lack of parental homeownership, controlling for other SES indicators, has been associated with child behavioral and emotional problems,[48] suggesting another pathway through which home ownership may be related to future disease status.

Although there was evidence of a relationship between current perceived stress and persistent BV, it was weaker than other stressors examined. Further, the association did not persist after adjustment for potential confounders, similar to the majority of other studies. It is possible that transient elevations in perceived stress do not increase susceptibility to BV or that reporting of perceived stress is relatively more subject to non-differential misclassification, which tends to bias results towards the null. However, it is also important to acknowledge that an understanding of how risk factors are related to BV is not well established.[49] It is plausible that demographic factors are causes of perceived stress and behaviors such as smoking are intermediates on the causal pathway between stress and BV. In either scenario, these variables are not confounders and adjusting for them would inappropriately bias the perceived stress-BV association.

This study has noteworthy strengths. Study visits were conducted relatively early in gestation, nearly eliminating preterm delivery bias. The elucidation of factors associated with BV in early-to-mid gestation is arguably of greater importance than in late pregnancy, given that BV is more strongly associated with early preterm delivery than near term deliveries.[2] Also, differential recall bias is unlikely, as participants were blinded to their BV status.

This study also has important limitations. First, it is not possible to gauge the external validity of this study since it was a convenience sample. Second, because there were so few

women with incident or remissive BV, these analyses were cross-sectional in nature. Thus, with the exception of childhood trauma and home ownership, a temporal relationship between stress measures and BV cannot be definitively inferred. Third, information on one established risk factor, douching, was missing for most participants and therefore not included in these analyses. However, in stress-BV studies that covaried for douching, the point estimate was not substantially affected.[3,13] Fourth, refinement of the timing of exposure to childhood adversity and chronic discrimination may be relevant. Last, the sample size was relatively small, producing wide confidence intervals and limiting our statistical power in performing stratified analyses. Moreover, due to the small number of non-Hispanic white women with persistent BV, these findings are primarily applicable to African American and Hispanic women.

Although more studies are needed to establish a clear causal link, stress-related interventions adopting a life course perspective may be influential in countering bacterial vaginosis and its consequences during pregnancy. These findings may have especially important implications for health disparities, as lifetime chronic stress experienced by African American women has been proposed as a cause of their increased susceptibility to preterm delivery and mediators such as BV.[18,50,51] Future studies should further explore this topic, as well as other common and modifiable types of early life stress for maximum public health impact.

Acknowledgments

Supported, in part, by US PHS (NIH) grants RO1 HD 041696 and PO1 HD 047609 to PDW. ALC received support from T32 HD 052460. CJH received support from Miriam Jacobs Endowed Chair.

References

1. French, JI.; McGregor, JA. Bacterial vaginosis: history, epidemiology, microbiology, sequelae, diagnosis, and treatment. Boca Raton, FL: CRC Press; Number of pages
2. Nelson DB, Macones G. Bacterial vaginosis in pregnancy: current findings and future directions. Epidemiol Rev. 2002; 24:102–8. [PubMed: 12762086]
3. Culhane JF, Rauh V, McCollum KF, Hogan VK, Agnew K, Wadhwa PD. Maternal stress is associated with bacterial vaginosis in human pregnancy. Matern Child Health J. 2001; 5:127–34. [PubMed: 11573838]
4. Green NS, Damus K, Simpson JL, et al. Research agenda for preterm birth: recommendations from the March of Dimes. Am J Obstet Gynecol. 2005; 193:626–35. [PubMed: 16150253]
5. McDonald HM, Brocklehurst P, Gordon A. Antibiotics for treating bacterial vaginosis in pregnancy. Cochrane Database Syst Rev. 2007:CD000262. [PubMed: 17253447]
6. Chrousos GP. The hypothalamic-pituitary-adrenal axis and immune-mediated inflammation. N Engl J Med. 1995; 332:1351–62. [PubMed: 7715646]
7. Kiecolt-Glaser JK, McGuire L, Robles TF, Glaser R. Psychoneuroimmunology and psychosomatic medicine: back to the future. Psychosom Med. 2002; 64:15–28. [PubMed: 11818582]
8. Coussons-Read ME, Okun ML, Schmitt MP, Giese S. Prenatal stress alters cytokine levels in a manner that may endanger human pregnancy. Psychosom Med. 2005; 67:625–31. [PubMed: 16046378]
9. Coussons-Read ME, Okun ML, Nettles CD. Psychosocial stress increases inflammatory markers and alters cytokine production across pregnancy. Brain Behav Immun. 2007; 21:343–50. [PubMed: 17029703]
10. Wadhwa PD, Dunkel-Schetter C, Chicz-DeMet A, Porto M, Sandman CA. Prenatal psychosocial factors and the neuroendocrine axis in human pregnancy. Psychosom Med. 1996; 58:432–46. [PubMed: 8902895]
11. Wadhwa PD. Psychoneuroendocrine processes in human pregnancy influence fetal development and health. Psychoneuroendocrinology. 2005; 30:724–43. [PubMed: 15919579]

12. Wadhwa PD, Culhane JF, Rauh V, et al. Stress, infection and preterm birth: a biobehavioural perspective. Paediatr Perinat Epidemiol. 2001; 15 2:17–29. [PubMed: 11520397]
13. Nansel TR, Riggs MA, Yu KF, Andrews WW, Schwebke JR, Klebanoff MA. The association of psychosocial stress and bacterial vaginosis in a longitudinal cohort. Am J Obstet Gynecol. 2006; 194:381–6. [PubMed: 16458633]
14. Harville EW, Savitz DA, Dole N, Thorp JM Jr, Herring AH. Psychological and biological markers of stress and bacterial vaginosis in pregnant women. Bjog. 2007; 114:216–23. [PubMed: 17305894]
15. Uscher-Pines L, Hanlon AL, Nelson DB. Racial differences in bacterial vaginosis among pregnant women: the relationship between demographic and behavioral predictors and individual BV-related microorganism levels. Matern Child Health J. 2009; 13:512–9. [PubMed: 18543090]
16. Trabert B, Misra DP. Risk factors for bacterial vaginosis during pregnancy among African American women. Am J Obstet Gynecol. 2007; 197:477 e1–8. [PubMed: 17980180]
17. Culhane JF, Rauh V, McCollum KF, Elo IT, Hogan V. Exposure to chronic stress and ethnic differences in rates of bacterial vaginosis among pregnant women. Am J Obstet Gynecol. 2002; 187:1272–6. [PubMed: 12439519]
18. Hogue CJ, Bremner JD. Stress model for research into preterm delivery among black women. Am J Obstet Gynecol. 2005; 192:S47–55. [PubMed: 15891712]
19. de Weerth C, Buitelaar JK. Physiological stress reactivity in human pregnancy--a review. Neurosci Biobehav Rev. 2005; 29:295–312. [PubMed: 15811500]
20. Entringer S, Buss C, Shirtcliff EA, et al. Attenuation of maternal psychophysiological stress responses and the maternal cortisol awakening response over the course of human pregnancy. Stress. 13:258–68. [PubMed: 20067400]
21. Glynn LM, Schetter CD, Wadhwa PD, Sandman CA. Pregnancy affects appraisal of negative life events. J Psychosom Res. 2004; 56:47–52. [PubMed: 14987963]
22. Astone NM, Misra D, Lynch C. The effect of maternal socio-economic status throughout the lifespan on infant birthweight. Paediatr Perinat Epidemiol. 2007; 21:310–8. [PubMed: 17564587]
23. McEwen BS. Protective and damaging effects of stress mediators. N Engl J Med. 1998; 338:171–9. [PubMed: 9428819]
24. American College Of Obstetricans And Gynecologists. ACOG practice bulletin, clinical management guidelines for obstetrician-gynecologists—ultrasonography in pregnancy. Obstet Gynecol. 2009; 113:451–61. [PubMed: 19155920]
25. Nugent RP, Krohn MA, Hillier SL. Reliability of diagnosing bacterial vaginosis is improved by a standardized method of gram stain interpretation. J Clin Microbiol. 1991; 29:297–301. [PubMed: 1706728]
26. Bernstein, DP.; Fink, L. Childhood Trauma Questionnaire: A retrospective self-report manual. San Antonio, TX: The Psychological Corporation; Number of pages
27. Williams D, Yu Y, Jackson J, Anderson N. Racial differences in physical and mental health: socioeconomic status, stress, and discrimination. Journal of Health Psychology. 1997; 2:335–51.
28. Hunte HE, Williams DR. The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. Am J Public Health. 2009; 99:1285–92. [PubMed: 18923119]
29. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. J Health Soc Behav. 1983; 24:385–96. [PubMed: 6668417]
30. Cohen S, Doyle WJ, Turner RB, Alper CM, Skoner DP. Childhood socioeconomic status and host resistance to infectious illness in adulthood. Psychosom Med. 2004; 66:553–8. [PubMed: 15272102]
31. Gariti P, Rosenthal DI, Lindell K, et al. Validating a dipstick method for detecting recent smoking. Cancer Epidemiol Biomarkers Prev. 2002; 11:1123–5. [PubMed: 12376520]
32. Barker DJ, Osmond C, Golding J, Kuh D, Wadsworth ME. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. Bmj. 1989; 298:564–7. [PubMed: 2495113]
33. Browne A, Finkelhor D. Impact of child sexual abuse: a review of the research. Psychol Bull. 1986; 99:66–77. [PubMed: 3704036]

34. Weiss EL, Longhurst JG, Mazure CM. Childhood sexual abuse as a risk factor for depression in women: psychosocial and neurobiological correlates. Am J Psychiatry. 1999; 156:816–28. [PubMed: 10360118]
35. Benedict MI, Paine LL, Paine LA, Brandt D, Stallings R. The association of childhood sexual abuse with depressive symptoms during pregnancy, and selected pregnancy outcomes. Child Abuse Negl. 1999; 23:659–70. [PubMed: 10442831]
36. Chung EK, Mathew L, Elo IT, Coyne JC, Culhane JF. Depressive symptoms in disadvantaged women receiving prenatal care: the influence of adverse and positive childhood experiences. Ambul Pediatr. 2008; 8:109–16. [PubMed: 18355740]
37. Heim C, Wagner D, Maloney E, et al. Early adverse experience and risk for chronic fatigue syndrome: results from a population-based study. Arch Gen Psychiatry. 2006; 63:1258–66. [PubMed: 17088506]
38. Leserman J. Sexual abuse history: prevalence, health effects, mediators, and psychological treatment. Psychosom Med. 2005; 67:906–15. [PubMed: 16314595]
39. Noll JG, Zeller MH, Trickett PK, Putnam FW. Obesity risk for female victims of childhood sexual abuse: a prospective study. Pediatrics. 2007; 120:e61–7. [PubMed: 17606550]
40. Wise LA, Palmer JR, Rothman EF, Rosenberg L. Childhood abuse and early menarche: findings from the black women's health study. Am J Public Health. 2009; 99 2:S460–6. [PubMed: 19443822]
41. Noll JG, Schulkin J, Trickett PK, Susman EJ, Breech L, Putnam FW. Differential pathways to preterm delivery for sexually abused and comparison women. J Pediatr Psychol. 2007; 32:1238–48. [PubMed: 17569710]
42. Noll JG. Sexual abuse of children--unique in its effects on development? Child Abuse Negl. 2008; 32:603–5. [PubMed: 18562000]
43. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry. 1995; 52:1048–60. [PubMed: 7492257]
44. Lewis TT, Everson-Rose SA, Powell LH, et al. Chronic exposure to everyday discrimination and coronary artery calcification in African-American women: the SWAN Heart Study. Psychosom Med. 2006; 68:362–8. [PubMed: 16738065]
45. Kessler RC, Mickelson KD, Williams DR. The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. J Health Soc Behav. 1999; 40:208–30. [PubMed: 10513145]
46. Giscombe CL, Lobel M. Explaining disproportionately high rates of adverse birth outcomes among African Americans: the impact of stress, racism, and related factors in pregnancy. Psychol Bull. 2005; 131:662–83. [PubMed: 16187853]
47. Miller G, Chen E. Unfavorable socioeconomic conditions in early life presage expression of proinflammatory phenotype in adolescence. Psychosom Med. 2007; 69:402–9. [PubMed: 17556642]
48. Boyle MH. Home ownership and the emotional and behavioral problems of children and youth. Child Dev. 2002; 73:883–92. [PubMed: 12038558]
49. Ness RB, Hillier S, Richter HE, et al. Can known risk factors explain racial differences in the occurrence of bacterial vaginosis? J Natl Med Assoc. 2003; 95:201–12. [PubMed: 12749680]
50. Lu MC, Halfon N. Racial and ethnic disparities in birth outcomes: a life-course perspective. Matern Child Health J. 2003; 7:13–30. [PubMed: 12710797]
51. Rich-Edwards JW, Grizzard TA. Psychosocial stress and neuroendocrine mechanisms in preterm delivery. Am J Obstet Gynecol. 2005; 192:S30–5. [PubMed: 15891710]

Table 1

Distribution of Exposures [a]

Exposure	N/ Mean	% Exposed / Standard Deviation
Discrimination Score	Mean=6.0	SD=6.2
Childhood Sexual Abuse [b]	54	18.7%
Childhood Physical Abuse	49	16.9%
Childhood Emotional Abuse	46	15.8%
Childhood Physical Neglect	46	15.8%
Childhood Emotional Neglect	52	17.9%
Parental Home Ownership	181	61.1%
T1 PSS Score	Mean=34.0	SD=8.1
T2 PSS Score	Mean=34.2	SD=8.2
Yearly Household Income	Median=$40-50,000 per year	
Education		
Less than High School	30	9.7%
High School or Vocational School	88	28.5%
Some College	101	32.7%
Bachelor's Degree or Above	90	29.1%

[a] Sample sizes may not sum to 312 (total number of subjects with BV data), due to missing data

[b] 18 subjects were missing data on all childhood abuse/neglect exposures.

Table 2

Overall Distribution of Covariates and Bivariate Associations Between Covariates and Persistent BV

Variable	N (%) [a]	N with Persistent BV (%) [b]	Chi-Square Test Statistic
Race/ Ethnicity			29.8 [c,d]
non-Hispanic White	113 (36.2%)	4 / 101 (4%)	
African American	73 (23.4%)	19 / 53 (35.9%)	
Hispanic	126 (40.4%)	14 / 112 (12.5%)	
Parity			0.25
0	108 (34.6%)	13 / 93 (14%)	
1	115 (36.9%)	15 / 100 (15%)	
2+	89 (28.5%)	9 / 73 (12.3%)	
Age			10.7 [c]
≤ 29 years (Median)	165 (52.9%)	28 / 135(20.7%)	
>29 years	147 (47.1%)	9 / 131 (6.9%)	
Cohabitation Status			25.2 [c]
Married or Cohabiting	243 (78.4%)	18 / 213 (8.4%)	
Not Married, not Cohabiting	67 (21.6%)	18 / 51 (35.3%)	
Smoking Status During Pregnancy			
Smoker	25 (8.4%)	6 / 23 (26.1%)	3.2
Non-Smoker	271 (91.6%)	29 / 230 (12.6%)	
Drug Use During Pregnancy			
Drug Use	11 (3.6%)	4 / 8 (50%)	9.1 [c,d]
No Drug Use	293 (96.4%)	32 / 253(12.7%)	
Antibiotic Use During Pregnancy			
Antibiotic Use Since Beginning of Pregnancy	61 (20.8%)	5 / 52 (9.6%)	0.9
No Antibiotic Use Since Beginning of Pregnancy	233 (79.2%)	31 / 210 (14.8%)	
Lifetime Sexual Partners			1.8
< 4 (Median)	142 (49.3%)	15 / 132 (11.4%)	
4+	146 (50.7%)	22 / 128 (17.2%)	
Age at First Sexual Intercourse			7.8 [e,f]
<14 years	17 (5.8%)	5 / 14 (35.7%)	
14-17 years	167 (57.0%)	23 / 149 (15.4%)	
18+ years	109 (37.2%)	9 / 100 (9%)	
Frequency of Sexual Intercourse			1.9
During Pregnancy			
Less than once a month	49 (17.2%)	8 / 43 (18.60 %)	
1-3 times per month	80 (28.1%)	11 / 74 (14.9%)	
1-2 times per week	109 (38.2%)	10 / 95 (10.5%)	
More than 2 times per week	47 (16.5%)	5 / 43 (11.6%)	

[a] Sample sizes may not sum to 312 (total number of subjects with BV data), due to missing data; distribution for overall study population (regardless of missing data for persistent BV).

[b] Percentage of subjects with persistent BV, given specified covariate

[c] Chi-square-statistic, p <.01

[d] Fisher's exact test (performed due to cell sizes less than or equal to five subjects), p <.01

[e] Chi-square-statistic, p <.05

[f] Fisher's exact test (performed due to cell sizes less than or equal to five subjects), p <.05

Table 3
Associations of Psychosocial and Socioeconomic Stress Exposures with Persistent BV

Exposure	Crude		Adjusted [a]	
	OR	95% CI	OR	95% CI
Discrimination				
Discrimination Score (continuous)	1.05	1.00, 1.11	1.07	1.00, 1.14
Elevated Discrimination Score	2.08	0.99, 4.41	2.33	0.90, 6.02
Childhood Trauma				
Childhood Sexual Abuse	2.51	1.15, 5.47	2.75	1.00, 7.58
Childhood Physical Abuse	0.72	0.26, 1.95	0.76	0.23, 2.49
Childhood Emotional Abuse	0.88	0.32, 2.42	0.51	0.14, 1.88
Childhood Physical Neglect	0.94	0.34, 2.60	0.91	0.26, 3.13
Childhood Emotional Neglect	1.23	0.50, 3.01	0.68	0.21, 2.15
Perceived Stress				
T1 PSS Score (continuous)	1.04	0.99, 1.08	0.98	0.92, 1.03
T1 Elevated PSS Score	1.99	0.92, 4.29	0.77	0.28, 2.13
T2 PSS Score (continuous)	1.05	1.00, 1.10	1.01	0.95, 1.07
T2 Elevated PSS Score	1.96	0.91, 4.21	0.79	0.29, 2.15
Childhood SES				
Lack of Parental Home Ownership	9.00	3.75, 21.61	3.27	1.05, 10.17
Income				
Yearly Household Income (ordinal)	0.72	0.61, 0.84	0.83	0.67, 1.02
Education				
Less than High School	5.47	1.13, 26.54	1.84	0.24, 14.12
High School or Vocational	6.96	1.92, 25.20	1.27	0.26, 6.24
Some College	4.92	1.36, 17.81	1.08	0.24, 4.84
Bachelor's Degree and Above	1.00	Referent	1.00	Referent

[a] Adjusted for age, race/ethnicity, cohabitation status, income, education, drug use, smoking, and age at first sexual intercourse, using categorizations as presented in Table 2.

Table 4
Independent Effects of Exposures Associated With Persistent BV

Exposure	Crude		Adjusted [a]	
	OR	**95% CI**	**OR**	**95% CI**
Discrimination Score (continuous)	1.05	1.00, 1.11	1.07	0.99, 1.15
Childhood Sexual Abuse	2.51	1.15, 5.47	3.07	1.09, 8.65
T2 PSS Score (continuous)	1.05	1.00, 1.10	1.00	0.94, 1.07
Lack of Parental Home Ownership	9.00	3.75, 21.61	8.03	2.84, 22.73
Yearly Household Income (ordinal)	0.72	0.61, 0.84	0.74	0.61, 0.90
Education				
Less than High School	5.47	1.13, 26.54	0.46	0.05, 4.30
High School or Vocational	6.96	1.92, 25.20	0.80	0.17, 3.81
Some College	4.92	1.36, 17.81	1.03	0.23, 4.60

[a] Adjusted for all other exposures listed in the table.