*Sexually Transmitted Diseases,* April 2008, Vol. 35, No. 4, p.363–367
DOI: 10.1097/OLQ.0b013e31815e4179


# *Bacterial Vaginosis, Race, and Sexually Transmitted Infections: Does Race Modify the Association?*

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***Background:*** **There are significant racial disparities in the prevalence of sexually transmitted infections (STIs) in the United States. The purpose of this study was to evaluate whether the association of bacterial vaginosis and incident STI is modified by race even after adjustment for sexual practices and other potential confounding variables.**

***Methods:*** **We evaluated the association of bacterial vaginosis (BV) and STI acquisition in a group of 523 women at high risk for unplanned pregnancies and STI. BV was diagnosed by both Gram stain and Amsel criteria. STIs included *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, pelvic inflammatory disease, trichomoniasis, syphilis, and HIV. Cox regression estimated the associations and the synergy index assessed whether race modified the association of BV and incident STI.**

***Results:*** **Sixteen percent of participants developed an STI during the 2-year follow-up. Compared with white women without BV at baseline, the adjusted hazard ratios were as follows: white women with BV = 0.59; African American women without BV = 1.96; and African American women with BV = 2.86. The synergy index of 3.38 implies a combined association of BV and African American race with STI in excess of each factor individually.**

***Conclusions:*** **African American race modifies the association of BV and incident STI. Future research should strive to determine the relative contributions of other factors, such as biologic variation, social network or the consequences of socioeconomic position, in this disparity.**

BACTERIAL VAGINOSIS (BV) is a common condition among US women. A recent analysis of the National Health and Nutrition Examination Surveys demonstrated that almost one-third of women were positive for BV.[1] Several cross-sectional and prospective cohort studies have found that BV is associated with acquisition of both HIV and sexually transmitted infections (STIs).[2–5] Race, specifically African American, is associated with both BV and numerous sexually transmitted infections. For example, the rate of *Neisseria gonorrhoeae* infection in African American women is 19 times that of white women, and the rate of chlamydial infection is more than 7.5 times that of white women.[6]

Supported in part by grants 1RO1-HD36663, Stage-Matched Intervention to Increase Dual Method Use, and K24 HD01298, Midcareer Investigator Award in Women's Health Research, from the National Institutes of Child Health and Human Development.

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Received for publication June 20, 2007, and accepted October 12, 2007.

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Given the alarming rates of STIs in African American women, research furthering the understanding of how etiologic factors for STIs may vary by race is warranted. The high prevalence of sexually transmitted infections in communities of color is fueled by lack of access to health care, poverty, inadequate resources, distrust of health systems, disease burden in social networks, and many other social factors.[7,8] In addition, biologic and/or genetic factors may also play an important role. If the presence of BV is synergistic with race in terms of STI acquisition, then intervention studies could target women of color for therapy trials to reduce incident sexually transmitted infections.

We sought to address this issue in a longitudinal analysis of *Project PROTECT,* a randomized trial to evaluate a counseling intervention to prevent unintended pregnancy and STIs. We hypothesized that African American women with BV would be more likely to develop an STI than either white women with BV or African American women without BV, that is, the combined effect of being African American and having BV would be greater than either individual risk factor (positive synergistic effect).

## Methods

Data for the current study were derived from a longitudinal analysis of Project PROTECT, funded by the National Institute of Child Health and Human Development (NICHD). A full description of the methods of this study has been published.[9] Briefly, Project PROTECT evaluated the extent to which a computer-based individualized intervention based on the transtheoretical model of behavior change could improve dual contraceptive method use (and greater protection against incident or recurrent cases of sexually transmitted infections and unplanned pregnancies) compared with a computer-based enhanced standard care counseling approach. The transtheoretical model, a widely used model of health behavior change, posits that behavior change does not occur uniformly independent of readiness to change, instead varying with an individual's readiness to change. Levels of readiness include precontemplation (no intention to change within the next 6 months), contemplation (thinking about changing within the next 6 months), preparation (planning to change within the next 30 days), action (has changed their behavior within the past 6 months), and main-

tenance (has successfully changed their behavior 6 months ago or earlier). Before study initiation, the trial protocols were approved by both the Women and Infants' Hospital and the University of Rhode Island Institutional Review Boards.

Women eligible for Project PROTECT included those who spoke English, were between the ages of 13 and 35, and who were competent to give informed consent. Parental consent and minor assent were obtained for all participants <18 years of age. Participants were recruited from primary care, gynecology, and family planning clinics including Planned Parenthood of Rhode Island. We also advertised the study in newspapers, and local cable and radio stations, and nurse recruiters visited local high schools and colleges in the Providence area. Women had to be sexually active with a male partner in the past 6 months. Women were only included if they expressed the desire to avoid pregnancy for 24 months after randomization. High-risk women were recruited from 2 specific age groups: (a) all sexually active women aged 13–24 and (b) high-risk sexually active women aged 25–35 years. Women older than 24 years were determined to be at high risk if their history included any of the following: unplanned pregnancy, history of an STI, inconsistent use of contraception, or other factors that placed a patient at above average risk for unplanned pregnancy or STI (e.g., more than 1 sexual partner in the past 6 months or drug or alcohol abuse).

At the time of the initial baseline visit, a research clinician (physician, nurse practitioner, or nurse) took a gynecological and contraceptive history and performed a pelvic examination, including evaluation of the external genitalia, vagina, and cervix and performed tests for vaginitis, *N. gonorrhoeae*, *Chlamydia trachomatis*, and syphilis. Strand displacement nucleic acid amplification (SDA) testing (BD ProbeTec ET System, Becton-Dickenson, Spanks, MD) was completed for *C. trachomatis* and *N. gonorrhoeae*, and serologic testing was performed for syphilis and human immunodeficiency virus (HIV). BV evaluation included both a Gram stain and Amsel's clinical criteria.[10,11] For participants with an elevated vaginal pH, we performed an In-Pouch trichomonas culture. A pelvic examination was performed to evaluate for pelvic tenderness that may indicate a pelvic inflammatory disease (PID).[12] To be eligible for participation, we required that women test negative for all sexually transmitted infections or, if infected, to accept direct observed therapy and a follow-up test of cure for their STI. For the purpose of this analysis, BV was not considered a sexually transmitted disease, but rather it was an exposure (independent variable). Asymptomatic BV was not routinely treated.

At 6 and 18 months after randomization, the subjects were contacted by telephone and answered questions about sexual habits and medical and sign/symptom history. This survey also gathered information regarding any clinical outcomes they may have experienced including STIs and/or pregnancy. At 12 and 24 months after baseline, subjects were asked to return for follow-up examinations and for completion of follow-up and background surveys. At annual follow-up visits, testing was done for trichomoniasis, *N. gonorrhoeae*, and *C. trachomatis*. A serologic test for syphilis, HIV, and a sensitive urine pregnancy test were performed. Patients were examined for signs of PID. Participants were instructed to call the study team if there was any suspicion of a biologic outcome (e.g., unplanned pregnancy or incident STI). Symptoms or signs that would alert the patient to a possible biologic outcome included a late or abnormal menstrual cycle, symptoms of pregnancy, irregular bleeding, abnormal discharge, urinary symptoms, or lower abdominal pain. If a subject experienced these signs or symptoms she was invited to the study center for an interim visit for a full history, examination, and testing.

Two variables were the primary determinants of interest: (a) BV measured at baseline by Gram stain as a continuous variable,[11] but dichotomized (present = Gram stain score 7–10; absent = score <7) for group analysis of race and BV presence and (b) African American race. We also sought to estimate the extent to which being African American modified the association between BV and STI acquisition. All participants completed a self-administered questionnaire and a more extensive computer-based survey that collected demographic, reproductive, and sexual history data at the time of randomization. Patients self-identified their race/ethnicity as Hispanic versus non-Hispanic, African American (black), white, or other. For the purpose of this analysis, we grouped race/BV status as follows: white/No BV (reference group); white with BV; African American without BV; and African American with BV.

For each woman, the number of days free of an incident STI was calculated as number of days from baseline to the date of the first detected STI or censoring. Follow-up time was censored at either the last known follow-up date or 24 months from their baseline interview.

Potential confounders included variables known to be related to both exposures (race and BV) and the outcome of interest. This list included age, education, cigarette and substance use, history of STI or unplanned pregnancy, contraceptive use, and sexual history. We assessed whether these variables were associated with the exposures and outcome of interest. Potential confounders based on clinical and biologic plausibility were further assessed by putting each factor into our multivariable model to see if our effect estimate changed by 10% or more. Factors that altered the effect estimate or that have been found to be important confounders in other studies were retained in our final model.

One measure of additive interaction is synergy.[13] Synergy is calculated as follows:

$$\text{SYNERGY} = (\text{RR}_{\text{both}} - 1)/(\text{RR}_1 + \text{RR}_2 - 2) \text{ or}$$

$$\text{SYNERGY} = (\text{RR}_{(\text{African American/BV}+)} - 1)/(\text{RR}_{(\text{African American/BV}-)} + \text{RR}_{(\text{Caucasian/BV}+)} - 2)$$

$$\text{SYNERGY} = 1 \text{ when there is no departure from additivity}$$

A synergy index greater than 1 implies that the joint exposure effect is greater than that predicted by the sum of the individual effects. We used the methods described by Hosmer and Lemeshow to calculate 95% confidence intervals around the synergy index.[14] SAS (Version 9.1) and was used to perform statistical analyses.

## Results

Our final analytic sample included 523 women with known BV status at baseline. Approximately 50% were between 20 and 24 years of age, 45% were white, 26% African American, 16% Hispanic, 49% were smokers, and almost 50% of participants had experienced an STI or unplanned pregnancy. Thirty-five percent used no contraceptive method (Table 1). More than 50% had 6 or more lifetime sexual partners, and 15% had more than 1 sexual partner in the past month.

Characteristics associated with BV were race/ethnicity, lower educational level, history of STI, and unplanned pregnancy (Table 1). Race/ethnicity was associated with baseline BV in this cohort. Thirty-three percent of African Americans were found to have BV compared with 24% of whites and 31% of Hispanic women.

A total of 83 individuals were noted to have an incident STI during follow-up: 45 cases of *C. trachomatis*, 21 cases of *N. gonorrhoeae*, 21 cases of trichomoniasis, 6 episodes of PID, and 1 confirmed HIV seroconversion (Note: numbers do not add up to 83, as some patients had more than 1 STI). Factors associated with

TABLE 1. Baseline Characteristics of Study Participants by Presence of Bacterial Vaginosis

| Characteristic | Total (N = 523) | No BV at Baseline (N = 361) | BV at Baseline (N = 162) | P for Difference at Baseline |
|---|---|---|---|---|
| Age | | | | |
| <20 yr | 152 (29) | 105 (29) | 47 (29) | 0.11 |
| 20–24 yr | 263 (50) | 190 (53) | 73 (45) | |
| 25–35 yr | 108 (21) | 66 (18) | 42 (26) | |
| Race/ethnicity | | | | |
| White, non-Hispanic | 234 (45) | 177 (49) | 57 (35) | <0.01 |
| African American, non-Hispanic | 138 (26) | 92 (25) | 46 (28) | |
| Hispanic | 86 (16) | 59 (16) | 27 (17) | |
| Other | 65 (12) | 33 (9) | 32 (20) | |
| Education | | | | |
| Less than high school | 129 (25) | 78 (22) | 51 (31) | 0.06 |
| High school/GED | 193 (37) | 133 (37) | 60 (37) | |
| 2 yr degree or some college | 159 (30) | 115 (32) | 41 (25) | |
| 4 yr degree or more | 44 (8) | 34 (9) | 10 (6) | |
| Smoking status | 254 (49) | 170 (47) | 84 (52) | 0.33 |
| Substance use | 316 (60) | 220 (61) | 96 (60) | 0.69 |
| History of STD | 243 (46) | 153 (43) | 90 (56) | <0.01 |
| History of unplanned pregnancy | 251 (48) | 163 (46) | 88 (55) | 0.04 |
| No contraceptive use | 181 (35) | 116 (32) | 65 (40) | 0.07 |
| Hormonal contraceptive use | 166 (32) | 123 (34) | 43 (27) | 0.10 |
| Male condoms | 157 (30) | 115 (32) | 42 (26) | 0.17 |
| Lifetime number of sexual partners | | | | |
| 1–2 | 68 (13) | 50 (14) | 18 (11) | 0.60 |
| 3–5 | 180 (34) | 126 (35) | 54 (33) | |
| 6–10 | 124 (24) | 78 (22) | 46 (28) | |
| 11 or more | 150 (29) | 106 (29) | 44 (27) | |
| Number of sexual partners in past month | | | | |
| 0 | 70 (13) | 56 (16) | 14 (9) | 0.71 |
| 1 | 372 (71) | 255 (71) | 117 (73) | |
| 2 or more | 79 (15) | 49 (14) | 30 (19) | |
| New main partner past 6 mo | 137 (26) | 97 (27) | 40 (25) | |
| Sex after drinking | | | | |
| Never | 251 (48) | 177 (49) | 74 (46) | 0.37 |
| 1–2 times | 159 (30) | 106 (29) | 53 (33) | |
| 3 or more times | 112 (21) | 77 (21) | 35 (22) | |
| Forced to have sex in past year | 53 (10) | 37 (10) | 16 (10) | |

The values are given in percentage.

STI acquisition included African American and Hispanic race/ethnicity, lower educational level, history of sexually transmitted disease, 2 or more sexual partners in the past month, and unplanned pregnancy. Age, smoking, substance use, baseline contraceptive method (hormonal, condom, or no method), lifetime number of sexual partners, having intercourse after drinking, or report of sexual abuse in the past year were not associated with incident STI.

The incidence rate of STIs in women without baseline BV was 9.7 cases per 100 women-years compared with 15.3 cases in women with BV (Table 2). The incidence rate in African American women was 19.9 cases per 100 women-years compared with 5.5 in white women. Race/ethnicity (both African Americans (adjusted HR = 2.8) and Hispanics (adjusted HR = 2.2)), history of STI (adjusted HR = 2.3), two or more sexual partners in the past month (adjusted HR = 1.7), and vaginal Gram stain (as a continuous variable, adjusted HR = 1.1) were significantly associated with incident STI.

We then evaluated whether race modified the association of BV and incident STI (Table 3). We calculated a synergy index using the combination of white race and the absence of BV as the reference group. After adjustments for history of STI, number of sexual partners in the past month, age, education and intervention arm, the following hazard ratios were obtained: no BV/white HR = 1.0; BV/white HR = 0.59; No BV/African American HR = 1.96, and BV/African American HR = 2.86. Our calculation of synergy indicated positive synergy between African American race and the presence of BV at baseline (synergy = 1.86/0.55 = 3.38; 95% CI 2.34, 4.89). Thus, there is a threefold excess risk due to the joint effects of being African American and having BV relative to the individual effects of being African American and having BV. Interestingly, a subsequent analysis of Hispanic participants noted that there was also a positive synergistic effect of BV and Hispanic ethnicity (synergy = 2.75; 95% CI 1.72, 3.82).

## Discussion

Effect measure modification should be investigated and explained, rather than eliminated. In this study, we found that BV and African American race were associated with STI acquisition. African American race appears to modify the association of BV and incident STIs, and this interaction remains significant even after adjustment for behavioral risk factors. Race and the presence of BV appear to act synergistically to increase the risk of STI acquisition.

BV is a disruption of the normal vaginal ecosystem. Many pathogens are sensitive to acidic pH, and normal vaginal flora typically creates a pH of 4.0–4.2 with the presence of $H_2O_2$-producing lactobacilli. BV is characterized by a lack of $H_2O_2$-producing lactobacilli and an alkaline pH. These factors may limit host defenses against STI pathogens.[3,15] BV is associated with

TABLE 2. Crude Rates and Adjusted Hazard Ratios for Outcome of Incident or Recurrent STD

| Characteristic | Events | Person-Years | Rate per 100 Person-Years | Crude HRR | 95% CI | Adjusted HRR | 95% CI |
|---|---|---|---|---|---|---|---|
| Gram stain score | | | | | | | |
| <7 | 49 | 505.4 | 9.7 | Ref. | | Ref. | |
| 7 or more | 34 | 222.9 | 15.3 | 1.59 | (1.02, 2.46) | 1.26 | (0.80, 1.97) |
| Race/ethnicity | | | | | | | |
| White | 18 | 326.4 | 5.5 | Ref. | | Ref. | |
| African American | 37 | 186.3 | 19.9 | 3.56 | (2.03, 6.26) | 2.83 | (1.58, 5.07) |
| Hispanic | 17 | 119.0 | 14.3 | 2.56 | (1.32, 4.96) | 2.23 | (1.13, 4.42) |
| Other | 11 | 96.6 | 11.4 | 2.06 | (0.97, 4.35) | 1.6 | (0.73, 3.48) |
| Age | | | | | | | |
| >24 | 15 | 143.2 | 10.5 | Ref. | | Ref. | |
| 20–24 | 39 | 362.6 | 10.8 | 1.00 | (0.55, 1.82) | 1.69 | (0.91, 3.15) |
| <20 | 29 | 222.6 | 13.0 | 1.22 | (0.66, 2.28) | 1.8 | (0.91, 3.55) |
| Education | | | | | | | |
| College graduate | 2 | 60.8 | 3.3 | Ref. | | Ref. | |
| Some college | 14 | 222.0 | 6.3 | 1.97 | (0.45, 8.67) | 1.51 | (0.34, 6.70) |
| High school/GED | 31 | 276.0 | 11.2 | 3.52 | (0.84, 14.7) | 1.83 | (0.42, 7.88) |
| Less than high school | 36 | 168.0 | 21.4 | 6.73 | (1.62, 28.0) | 3.48 | (0.80, 15.1) |
| History of STD | | | | | | | |
| No | 24 | 391.4 | 6.1 | Ref. | | Ref. | |
| Yes | 58 | 331.5 | 17.5 | 2.84 | (1.77, 4.57) | 2.3 | (1.36, 3.88) |
| No. sexual partners (past month) | | | | | | | |
| <2 | 66 | 629.5 | 10.5 | Ref. | | Ref. | |
| Two or more | 17 | 98.8 | 17.2 | 1.65 | (0.97, 2.82) | 1.74 | (1.01, 3.03) |

Note: Cox proportional hazard model included age group, education, race/ethnicity, history of STD, BV gram stain, number of sexual partners, and intervention arm.

sexually transmitted infections such as *N. gonorrhoeae*, *C. trachomatis*, and PID. Among 255 women, Wiesenfeld noted that those with baseline BV were more likely to test positive for *N. gonorrhoeae* (OR = 4.1) and *C. trachomatis* (OR = 3.4), whereas subjects with $H_2O_2$-producing lactobacilli were less likely to have an infection.[5] In another study, women with a heavy growth of BV-associated microorganisms and a new sexual partner were at a ninefold increased risk of PID (adjusted RR = 8.8).[16]

African American race is an important risk factor for sexually transmitted infections.[6,17] The explanation and mechanisms for the association of African American race and STIs are not known. Even when well-known risk factors are controlled for, racial disparities remain. Social issues such as poverty, limited resources, access to health care, distrust of the health care system and providers, and multiple other social factors play a role.[7,8] Biologic and genetic factors may also be involved. Social structure (e.g., race-based income distribution, de facto residential segregation, etc.) can help explain some of these disparities.[18] When aspects of social structure are included in a multivariable model, the proportion of African Americans no longer had an effect on the rates of *N. gonorrhoeae*.[18] Unfortunately, we do not have the information in our database to deconstruct race and evaluate this hypothesis in our data set.

Biologic factors may help explain the racial differences in the rate of STIs. Our study demonstrates that race may modify the effect of BV on incident or recurrent STI. African American women with BV were at high risk for incident STI, and this risk is not fully explained by the risk factors of BV and race individually. We found that these 2 risk factors act synergistically to elevate women's risk of STI acquisition. While this study advances our understanding of one of the influences on the persistent racial disparities in STI acquisition, it is likely that the reason for the observed positive synergy is multifactorial. Studies of social networks, genetic variation in inflammation and immune response, health care access, and other health care-related factors may be important contributors.

TABLE 3. Hazard Ratios for Combinations of BV and Race Strata (N = 370)

| Characteristic | No. Events | Person-Time (yr) | IR per 100 Person-Years | Crude IRR | Crude HRR (95% CI) | Adjusted HRR (95% CI)* |
|---|---|---|---|---|---|---|
| No BV/white | 14 | 244.06 | 5.74 | Ref. | | Ref. |
| BV/white | 4 | 82.34 | 4.86 | 0.85 | 0.87 (0.29, 2.64) | 0.59 (0.19, 1.83) |
| No BV/African American | 21 | 126.44 | 16.61 | 2.90 | 2.87 (1.46, 5.63) | 1.96 (0.96, 4.00) |
| BV/African American | 16 | 59.84 | 26.74 | 4.66 | 4.75 (2.32, 9.74) | 2.86 (1.35, 6.09) |

Calculation of SYNERGY:

$SYNERGY = (HRR_{(African\ American/BV+)} - 1)/(HRR_{(African\ American/BV-)} + HRR_{(Caucasian/BV+)} - 2)$
$SYNERGY = (2.86 - 1)/(1.96 + 0.59 - 2)$
$SYNERGY = 1.86/0.55 = 3.38$

*Hazard ratios adjusted for age, education, history of STD, number sexual partners, and intervention arm.

Clinical studies evaluating risk factors and risk markers for STIs must be careful to control for the effect of potential confounding variables such as age, race/ethnicity, sexual history, and other high-risk behaviors. Investigators should use stratification and multivariate techniques to control for potential confounders and eliminate the effect of confounding. In this observational analysis, we controlled for a number of potential confounders, but we may be missing important variables that were not captured in our dataset (e.g., douching). Thus, residual confounding is always a possibility.

We were surprised by the reduced hazard rate ratio in white women with BV. When we looked into the data further, we discovered that white women with BV were more likely to use condoms than were white women without BV. This finding may partially account for the "protective" effect of BV in these women.

Strengths of our study that deserve mention include prospective assessment of a moderately large sample of high-risk women, careful and uniform ascertainment for STI acquisition, and the use of multivariable analytic techniques to adjust for important confounding variables. Limitations include the fact that this is a secondary analysis of an intervention trial and a limited population from a single urban area in the Northeast United States. We also deliberately selected high-risk women to maximize our number of outcomes. As a result, our findings may not be generalizable to all sexually active women. Additional studies of a broad range of sexually active women from a diverse geographic area would help support the generalizability of these findings. Finally, our exposure variable (baseline BV) was a static measure, and we know that BV status can change over time. It would be interesting to explore whether BV acquisition is associated with incident STIs.

In summary, our findings support our hypothesis that African American race modifies the association of baseline BV on STI acquisition. In a separate subgroup analysis, we noted that Hispanic ethnicity also modifies this association. This latter finding was not expected, and thus should be verified in additional populations. Further studies are also needed to elucidate the relative biologic, genetic, and social mechanisms responsible for this synergistic effect.

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