Association between obesity and bacterial vaginosis as assessed by Nugent score

Rita T. BROOKHEART, PhD, Warren G. LEWIS, PhD, Jeffrey F. PEIPERT, MD, PhD, Amanda L. LEWIS, PhD, Jenifer E. ALLSWORTH, PhD

Author information: From the Department of Medicine (Dr. Brookheart), Center for Women's Infectious Disease Research (Drs. Lewis and Lewis), and the Departments of Molecular Microbiology (Drs. Lewis and Lewis), and Obstetrics and Gynecology (Dr. A.L. Lewis), Washington University School of Medicine; Department of Obstetrics and Gynecology (Dr. Peipert), Indiana University School of Medicine; Department of Biomedical and Health Informatics (Dr. Allsworth), University of Missouri, Kansas City School of Medicine

Corresponding Authors: Jenifer E. Allsworth, Department of Biomedical and Health Informatics, University of Missouri-Kansas City School of Medicine, 2411 Holmes Street, Kansas City, MO 64108, USA. Phone: (816) 235-1781. E-mail: allsworthj@umkc.edu

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Condensation: Obese and overweight women exhibit higher Nugent scores and an increased prevalence of bacterial vaginosis than lean women.

Short Title: Obesity and the prevalence of bacterial vaginosis

AJOG at a Glance:

A. Although several risk factors for bacterial vaginosis have been identified, whether obesity/overweight is a risk factor for bacterial vaginosis is not clear. This study was conducted to determine whether an association between obesity/overweight and prevalence of bacterial vaginosis exists and to examine the role of race in this context.

B. Key findings of this study are that obese and overweight women have higher Nugent scores and increased prevalence of bacterial vaginosis. We also show that race is an effect modifier of the relationship between body mass index and prevalence of bacterial vaginosis.

C. This study uncovers an association between obesity/overweight and frequency of bacterial vaginosis, as well as demonstrating that, unlike white women, black women exhibit higher Nugent scores and increased prevalence of bacterial vaginosis regardless of body mass index.

Abstract

BACKGROUND: Bacterial vaginosis is one of the most common vaginal conditions in the U.S. Recent studies have suggested obese women have an abnormal microbiota reminiscent of BV; however, few studies have investigated the prevalence of bacterial vaginosis in overweight and obese populations. Moreover, despite the increased prevalence of obesity and bacterial vaginosis in black women, it is not known whether racial disparities exist in the relationship between obesity and bacterial vaginosis.

OBJECTIVE: The objective of this study was to examine the relationship between body mass index and bacterial vaginosis as determined by Nugent score and to determine the influence of race in this context.

STUDY DESIGN: We performed a cross-sectional study using patient data and vaginal smears from 5,918 participants of the Contraceptive CHOICE Project. Gram stained vaginal smears were scored using the Nugent method and categorized as BV-negative (Nugent score 0-3), BV-intermediate (Nugent score 4-6), or BV-positive (Nugent score 7-10). Body mass index was determined using Centers for Disease Control and Prevention guidelines and obese individuals were categorized as Class I, II, or III obese based on NIH and World Health Organization body mass index parameters. Linear regression was used to model mean differences in Nugent scores and Poisson regression with robust error variance was used to model prevalence of bacterial vaginosis.

RESULTS: In our cohort, 50.7% of participants were black, 41.5% were white, and 5.1% were of Hispanic ethnicity with an average age of 25.3 years old. Overall, 28.1% of participants were bacterial vaginosis-positive. Bacterial vaginosis was prevalent in 21.3% of lean, 30.4% of

overweight, and 34.5% of obese women (p<0.001). The distribution of bacterial vaginosis-intermediate individuals was similar across all body mass index categories. Compared to lean women, Nugent scores were highest among overweight and obese Class I women (adjusted mean difference; overweight 0.33 [95% CI 0.14, 0.51] and Class I obese 0.51 [95% CI 0.29, 0.72]). Consistent with this, overweight and obese women had a higher frequency of bacterial vaginosis compared to lean women, even after adjusting for variables including race. Among white women, the prevalence of BV was higher for overweight and Class I and Class II/III obese white women compared to lean white women, a phenomenon not observed among black women, suggesting an effect modification.

CONCLUSION: Overweight and obese women have higher Nugent scores and a greater occurrence of bacterial vaginosis compared to lean women. Black women have a greater prevalence of bacterial vaginosis independent of their body mass index compared to white women.

KEYWORDS: bacterial vaginosis, obesity, body mass index, Nugent score, race, overweight, microbiome

Introduction

Bacterial vaginosis (BV) is one of the most common vaginal conditions in the U.S. and is present in approximately one out of every three women.[1] BV is characterized by lower levels of beneficial *Lactobacilli* and an overgrowth of fastidious anaerobic bacteria such as *Gardnerella vaginalis*, *Atopobium vaginae* and species of *Prevotella* and *Mobiluncus*.[2] Women with BV are at an increased risk for sexually transmitted infections (STIs; e.g., gonorrhea, chlamydia, HIV, and trichomoniasis), urinary tract infection, pelvic inflammatory disease, and adverse pregnancy outcomes including preterm birth.[3–13]

Nugent scoring is the gold standard for laboratory-based BV diagnosis and uses morphotype evaluation of Gram-stained slides to quantify the representation of Gram-positive (*Lactobacillus*), small Gram-negative or -variable (*Gardnerella, Bacteroides*), and curved organisms (such as *Mobiluncus*) in vaginal fluid smears.[14] These measurements are reported as a score ranging from 0 to 10, with scores 0-3 indicative of a "normal" *Lactobacillus*-dominant microbiota and 7-10 indicating a positive BV diagnosis. Women with a score of 4 to 6 have an "intermediate" microbiota, and, similar to BV-positive individuals, may be at greater risk for acquiring STIs compared to women with a "normal" *Lactobacillus*-dominant microbiota.[8,15–17] Although the pathologic significance of BV-intermediate status is still not clear in all situations, this type of vaginal microbiota is often considered along with BV as an "abnormal microbiota".[8,18,19] It is known that several factors including menstruation, [20,21] douching,[1,22,23] and high numbers of sexual partners[24] are associated with disruptions of the vaginal microbiota. Many questions still remain about how BV negatively influences women's reproductive health. Unfortunately, there is little mechanistic information about how the dysbiotic BV microbiome

develops or how individual bacteria interact with the host to produce disease. However, recent studies in mouse models have further implicated *Gardnerella vaginalis* as a cause of features related to BV. [25,26] These unknowns and the fact that BV is a common condition in the U.S. underscore the importance of identifying BV-associated risk factors to identify women at high risk for adverse gynecologic and obstetric outcomes and to design more effective treatments and prevention strategies.

While a relationship between increased body mass index (BMI) and gut dysbiosis has been widely studied, [27–32] little is known about the relationship between BMI and BV prevalence. Most recently, it has been reported that the vaginal microbiota of overweight and obese Korean women exhibited a larger proportion of *Lactobacillus iners* and *Prevotella* compared to lean women.[33,34] This is of interest since both of these taxa have been previously associated with BV.[35,36] While these studies suggest there may be an increased prevalence of BV in overweight/obese women, participant BV status was not reported.[33,34] One study conducted among U.S. women reported a positive correlation between high BMI and BV; however, after multivariable modeling, this study showed BMI was not independently associated with BV.[37] This study had several caveats including that less than one third of the women examined were black, and it did not examine the relationship between BMI and women with an “intermediate” microbiota (Nugent score 4-6). Moreover, all obese women were categorized into a single BMI group regardless of the subclass of obesity. Both NIH and WHO categorize obese individuals into three subclasses based on BMI: Class I (30-34.9 kg/m^2), Class II (35-39.9 kg/m^2) and Class III (≥40 kg/m^2),[38,39] and reports have shown an association between obesity class level and an increased prevalence of disease.[40,41] Given the racial disparities among overweight and obese

women, and the higher prevalence of BV in black women, understanding the relationship between BV and BMI, and the role of race, is highly warranted.[1,42–44]

To increase our understanding of the vaginal microbiota among overweight/obese women, and the extent to which this association may be influenced by race, we examined the correlation between BMI, Nugent score, and BV prevalence among women in the St. Louis region. Specifically, we examined whether BMI positively correlated with higher Nugent scores and increased BV prevalence. To test whether factors such as race influenced the proposed relationships, we performed multivariable modeling using information gathered from 5,918 reproductive aged women, of whom 50.7% were black.

Materials and Methods

Study design

We conducted a cross-sectional sub-study of participants from the Contraceptive CHOICE Project (CHOICE).[45] CHOICE obtained written informed consent from all participants before enrollment in accordance with its approved IRB protocol from Washington University in St. Louis. CHOICE participants consented to the use of questionnaire data and stored vaginal samples by future sub-studies. The current sub-study obtained IRB approval (ID# 201108155) from Washington University in St. Louis and followed the principles outlined in the Declaration of Helsinki for human research.

Over a 4-year period, CHOICE enrolled 9,256 women from the St. Louis region and provided FDA-approved reversible contraceptive methods at no-cost.[45] Eligibility criteria included women

14 to 45 years of age, self-reported sexual activity in the past 6 months or plans to become sexually active with a male partner, and a desire to prevent pregnancy through the use of a reversible contraceptive method. Participants with a history of tubal ligation or hysterectomy were excluded from the study. The CHOICE cohort predominantly consisted of black and white participants, which is representative of the racial make-up of the St. Louis region. The current sub-study only included women with a complete baseline questionnaire survey, BMI measurement, and Nugent score (n= 5,918). The baseline questionnaire included age, self-reported race and ethnicity, highest level of education obtained, monthly income, receipt of public assistance, difficulty paying for basic necessities, tobacco history, number of sexual partners, history of douching in last 30 and 180 days, history of STIs or positive for an STI at enrollment. Menstrual status was estimated as last menstrual period within 6 days of enrollment and a flag for recent hormonal contraceptive method use was created for those who reported contraceptive pills, patch, ring or injection, the levonorgestrel intrauterine system or subdermal implant. History of STI was defined as ever told by a healthcare provider that had one of the following sexually transmitted infections: chlamydia, gonorrhea, trichomoniasis, syphilis, human papillomavirus or genital warts, human immunodeficiency virus or herpes; current STI was defined as positive test for *Chlamydia trachomatis*, *Neisseria gonorrhoeae* or *Trichomonas vaginalis* at enrollment.

Assessment of Bacterial Vaginosis

At the time of CHOICE enrollment and prior to LARC method insertion, participants were instructed by a medical professional for self-collection of vaginal fluid from a mid-vaginal site (approximately 2 inches into the vagina) using a double-headed rayon swab (Starplex Scientific

Inc., Etobicoke, Ontario, Canada). Vaginal swabs were immediately rolled onto glass slides to create vaginal smears, which were Gram-stained and scored using the Nugent method.[14] The Nugent method consisted of microscopic evaluation of bacterial morphotypes to score the overall character of the vaginal flora.[14] Nugent scores range from 0 to 10 based on the prevalence of three bacterial morphotypes that roughly correspond to *Lactobacillus*, *Gardnerella vaginalis* or *Bacteroides*, and *Mobiluncus*. The number of long rod-shaped Gram-positive bacilli are scored 0-4, where 0 indicates high numbers of *Lactobacillus*; small Gram-negative and Gram-variable rods and coccobacilli (*Bacteroides* and *G. vaginalis*) scored 0-4, with 4 denoting the highest observed number of these bacteria; and curved rods (e.g. *Mobiluncus* spp.) scored 0-2, where 2 indicates the highest observed numbers. To ensure consistency in the amount of vaginal fluid on each slide and Gram-staining and Nugent scoring, all swabs were rolled by the same technician and all slides were stained and scored by the same technician. To assess the reliability of our scoring, a subset of smears we scored were also scored by the laboratory of Dr. Sharon Hillier (who established the Nugent score method[14]) at the Magee-Womens Research Institute, University of Pittsburgh and was reproducible between both research groups. Samples were categorized as BV-negative (score 0-3), BV-intermediate (score 4-6), or BV-positive (score 7-10).

BMI determination

Weight and height of participants were measured at the clinics by research personnel using a standardized protocol at the time of enrollment. Weight was recorded in pounds and height in feet and inches. Participants removed shoes and heavy outer clothing before being measured. This data was converted to BMI using the formula published by the Centers for Disease Control

and Prevention:[46] (weight (lb)/[height(in)]^2) x 703. Women were categorized by BMI based on NIH and WHO recommendations: underweight (<18.5 kg/m^2), lean (18.5-24.9, overweight (25-29.9 kg/m^2), and Class I obese (30-34.9 kg/m^2), Class II (35-39.9 kg/m^2) obese and Class III (≥40 kg/m^2) obese.[38,39]

Statistical analysis

Participant characteristics were described for all women and among strata of BMI categories. P-values for these comparisons were estimated using chi-square tests (all categorical variables) or linear regression (age). We examined multiple metrics of BV in relation to BMI: Nugent score category (including intermediate), Nugent-defined bacterial vaginosis, and symptomatic BV (report of discharge, itching, odor or pain during urination[47] during the 7 days prior to the clinic visit and sample collection).

Crude and adjusted mean differences and 95% confidence intervals were estimated using linear regression stratified by BMI among all participants and by self-identified race group (black or white). Potential confounders (listed in Table 1) were evaluated for association with body mass index and Nugent score. All variables that were significant at the alpha < 0.05 level were retained for inclusion in the fully adjusted model. Hispanic ethnicity and ever use of tobacco were not associated with Nugent score and were excluded. Variables that were significant in the fully adjusted model (public assistance, education, current smoker, douching in the last 30 days, sexually transmitted infection at baseline, and current hormonal contraception) were included in the final adjusted model. The All Participant models were also adjusted for race. Prevalence ratios of BV were estimated using Poisson regression with robust error variance. This approach provides an unbiased estimate of the prevalence ratio in the instance of a common binary

outcome. The p-value for the interaction term for BMI and race served as an indicator of effect modification. P-values for two-tailed tests less than alpha = 0.05 were considered statistically significant. All analyses were conducted in Stata 13.0 (StataCorp LP, College Station, TX).

Results

Participant characteristics

Of the 9,256 CHOICE participants, 6,022 (65.1%) had a baseline questionnaire survey, BMI measurement, and Nugent score. The main reason for missingness (N=2,417, 26.1%) was absence of a vaginal smear for Nugent scoring, an element added to the protocol after enrollment began. Of the 6,022 eligible participants, 5,918 (98.3%) had complete data and were included in the current analysis. Participant data and vaginal specimens were obtained at the time of enrollment. Participants averaged 25.3 years old, and 50.7% self-identified as black (Table 1). Over half of participants (52.9%) reported a monthly income of $800 or less and 38.1% reported some form of public assistance at enrollment. One third of participants (33.9%) reported a high school diploma as the highest degree obtained. Most women reported multiple lifetime sexual partners (median=3); 27.5% of participants reported 2-4 partners, 29.2% reported 5-7, 14.2% reported 8-12, and 19.7% reported 13 or more lifetime sexual partners. Forty-six percent had a history of smoking, with 23.1% self-reporting as current smokers at the time of enrollment.

In this cohort, 27.3% of women were BV-intermediate and 28.1% were BV-positive (Table 2). Of the women diagnosed as BV-positive, 17.2% reported symptoms associated with BV (i.e., abnormal discharge, foul odor, and vaginal itching[47]) at the time of enrollment.

BV prevalence by BMI category

Of the 5,918 study participants, 2.9% were underweight (BMI <18.5 kg/m^2), 39.1% were lean (BMI 18.5-24.9 kg/m^2), 26% were overweight (BMI 25-29.9 kg/m^2), and 32% were obese (BMI ≥30 kg/m^2) (Table 1). As shown in Table 2, 34.5% of obese, 30.4% of overweight, and 21.3% of lean women were BV-positive. Given that we observed no relationship between BMI and BV-intermediate scores in this cohort, we examined the number of women below the threshold of BV (BV-negative and -intermediate) and found it to be highest among lean women (78.7%) and lowest among obese women (65.5%) (Table 2).

We next examined whether a relationship existed between obesity class and BV prevalence. Due to the limited number of Class II and III obese individuals in this cohort, members of these two classes (BMI ≥35 kg/m^2) were grouped together (n=958) and members of Class I (n=934) remained separate. Nugent scores were higher in overweight (0.33 [95% CI 0.14, 0.51]), Class I obese (0.51 [95% CI 0.29, 0.72]), and Class II/III obese groups (0.37 [95% CI 0.16, 0.59]) compared to lean women (Table 3). Consistent with this observation, the adjusted prevalence ratio of BV was 1.25 (95% CI 1.12, 1.39) for overweight, 1.31 (95% CI 1.16, 1.47) for Class I obese, and 1.25 (95% CI 1.11, 1.41) for Class II/III obese women compared to lean women (Table 4, 5th column).

The role of race in the BMI-BV relationship

To determine whether the relationship between BMI and BV was influenced by race, we performed a within race analysis of the mean difference in Nugent scores and the prevalence ratio of BV among black women (n=3,001) in each BMI category. Adjusted Nugent scores were

higher in overweight (0.30 [95% CI 0.01, 0.58)] and Class I obese (0.41 [95% CI 0.10, 0.73]) black women, compared to lean black women (Table 3). However, the adjusted Nugent scores of Class II/III obese black women were not significantly different compared to lean counterparts. Among white women (n=2,457), Nugent scores were higher for Class I (0.56 [95% CI 0.23, 0.89]) and Class II/III (0.58 [95% CI 0.21, 0.95]) obese white women compared to lean white women. We observed no significant difference in Nugent scores for overweight white women compared to lean white women (Table 3).

We next examined the adjusted prevalence ratio of BV for black women across all BMI categories. We observed that only Class I obese black women had an increased occurrence of BV (1.14 [95% CI 1.00, 1.31]) compared to lean black women, while the prevalence of BV for overweight and Class II/III obese black women was not statistically different than lean black women (Table 4). Among white women, the adjusted prevalence ratio of BV was greater in overweight (1.44 [95% CI 1.16, 1.79), Class I (1.73 [95% CI 1.35, 2.22]), and Class II/III (1.63 [95% CI 1.23, 2.15]) obese white women compared to lean white women (Table 4). We next examined the effect modification of race on the BMI-BV relationship. The statistical interaction of increasing BMI and race in relation to BV prevalence was significant for overweight (p =0.024) and obese (class I, p = 0.001 and class II/III, p = 0.002) women (Table 4). No interaction of race was observed in the association of BMI and Nugent score (Table 3).

Comment

We report that Nugent scores were higher in overweight (4.53) and obese (class I - 4.87, and class II/III - 4.93) women compared to lean (3.90) women. Overweight and obese women also

had a higher frequency of BV (overweight - 25%, and obese class I - 31% and class II/III - 25%; adjusted). Because black race is a risk factor for both BV and obesity in women, [1,44–46] we examined the relationship between BMI and BV by race. Among white women, Nugent scores were higher in obese (class I - 3.99 and class II/III - 4.08) women than in lean (3.21) women. White overweight (19.9%) and obese (class I - 24.7% and class II/III - 24.2%) women had a higher prevalence of BV compared to lean (12.5%) white women. However, among black women, this phenomenon was not present, suggesting that BV occurrence in black women is independent of their BMI. We observed a significant interaction of race and increasing BMI in relation to BV prevalence for overweight ($p = 0.024$) and obese (class I $p = 0.001$ and class II/II $p = 0.002$) women, suggesting race is an effect modifier of the association of increasing BMI and BV prevalence. While the interaction of race on the BMI-BV relationship has not been previously reported, studies have shown obese white women exhibit a higher avoidance of female preventative health care services (e.g., Papanicolau test and breast cancer screening), a phenomenon not observed in obese black women.[48,49] Multiple factors likely contribute to the significant interaction between race, BMI, and BV in our study; the previously observed higher level of delay and avoidance toward preventative genital health services among obese white women may be one factor.[50]

Few studies have explored the relationship between BMI and BV prevalence, and a consensus on whether BMI is a risk factor for BV has not been reached. In one study of 2,906 U.S. women, of which 26.2% were black, 36% of obese women were BV positive; however, after adjusting for confounders, there was no relationship between BMI and BV.[37] This apparent discrepancy may be due to our larger sample size (n=5,918), a larger representation of black women (50.7%), and

potential differences in the differential control of confounders and levels of residual confounding between our study and Koumans *et al*. A recent longitudinal study reported obesity was associated with nearly a 20% decrease of BV risk in a cohort of 1,946 Kenyan female sex-workers.[51] The longitudinal Kenyan study measured relative risk of BV in obese populations while our cross sectional study measured prevalence (e.g., one infers a causal relationship while the other offers association). Differences in the characteristics of the Kenyan cohort and our cohort may also account for the discrepancy between the two studies, for example, our larger sample size (n=5,918 total and n=3,001 black women versus their n=1,946). Additionally, their cohort consisted of only African women, while our analysis included women of white (41.5%), black (50.7%), and other (7.8%) races. This difference may be important since African and black women exhibit a higher incidence of vaginal microbiota disruption compared to white women,[52,53] thus results of one race may vary from results of other races. Expanding on this point, our within race analyses (Tables 3-4) show that in white women, increasing BMI is associated with a higher incidence of a disrupted vaginal microbiota and increased prevalence of BV; however, for black women, the same comparison did not reach statistical significance. Other differences include a high HIV prevalence (41.8%) and the women studied were sex workers; the obese women in the study also appeared to be more likely to have high CD4 counts compared to normal women. Whether these characteristics influenced BV risk in the Kenyan population was not explored. Additional studies are needed to fully understand the relationship between BMI and BV prevalence in different geographic populations.

Given the complex nature of obesity, mechanisms contributing to the increased occurrence of BV in obese women are expected to be multifactorial. While reports have shown a positive

correlation between overweight/obese women and the presence of BV-associated microbiota,[33,34] the mechanisms at play remain unknown. Obesity may generate a favorable environment for BV through disturbances in host hormonal, metabolic, and/or immune functions. Diet may also influence the BMI-BV relationship, since certain dietary habits have been associated with BV.[54,55] A potential role for the gut microbiota in BV is also plausible, since the gut microbiota has been suggested to influence the composition of the vaginal microbiota by serving as an extravaginal reservoir of bacteria.[56] In addition, given the higher prevalence of menstrual irregularity in obese women, the presence of blood may alter vaginal flora. The role of douching in the BMI-BV relationship should also be considered, since douching is associated with BV and was found in one study to be practiced more often among obese women.[37] The mechanisms that contribute to the BMI-BV relationship may best be explored via established animal models of obesity and BV,[25] which would allow for a causal analysis of the role of specific factors such as obesity-associated hormonal and metabolic dysfunctions, dietary habits, the gut microbiota, and the synergistic effects these factors may exhibit.

This study had both strengths and limitations. Our 5,918 cohort represented a diverse group of women socioeconomically and racially. BMI and Nugent score were determined for each participant by trained clinical staff using universally approved and established guidelines.[14,46] Reproducibility of our Nugent scoring was verified by Dr. Sharon Hillier's laboratory (developer of the Nugent scoring method [14]), for a sample of specimens. In this cohort, 28.1% of women were BV-positive, a figure similar to estimates from a representative sample of U.S. reproductive aged women (29%), [57] and at the time of enrollment, 17.2% of BV-positive women reported symptoms associated with BV, a percentage consistent with another report (15.7%), [37] thus

underscoring the commonly asymptomatic nature of BV from the patient perspective. Limitations in our study included small numbers of underweight and Class II and III obese women, a cross-sectional design, and a lack of information on recent antibiotic use. Also, our study focuses on two races, black and white, and does not focus on the relationship between BMI and BV in other racial populations, since the sample size of other races in our cohort was small.

Obesity and BV pose serious threats to women's health and black race is a risk factor for both of these conditions. Our study demonstrates overweight and obesity are associated with higher Nugent scores and increased prevalence of BV, and the relationship between BMI and BV prevalence varies between black and white women. Our observations indicate additional efforts to understand the relationship between obesity and BV and the influence of BMI on the vaginal microbiome in racially diverse cohorts are highly warranted.

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References

1. Allsworth JE, Peipert JF. Prevalence of bacterial vaginosis: 2001-2004 National Health and Nutrition Examination Survey data. *Obstet Gynecol*. 2007;109(1):114-120. doi:10.1097/01.AOG.0000247627.84791.91.
2. Allsworth JE, Lewis VA, Peipert JF. Viral sexually transmitted infections and bacterial vaginosis: 2001-2004 National Health and Nutrition Examination Survey data. *Sex Transm Dis*. 2008;35(9):791-796. doi:10.1097/OLQ.0b013e3181788301.
3. Wiesenfeld HC, Hillier SL, Krohn MA, Landers D V, Sweet RL. Bacterial vaginosis is a strong predictor of Neisseria gonorrhoeae and Chlamydia trachomatis infection. *Clin Infect Dis*. 2003;36(5):663-668. doi:10.1086/367658.
4. Wiesenfeld HC, Hillier SL, Krohn MA, et al. Lower genital tract infection and endometritis: Insight into subclinical pelvic inflammatory disease. *Obstet Gynecol*. 2002;100(3):456-463. doi:10.1016/S0029-7844(02)02118-X.
5. Kline KA, Lewis AL. Gram-Positive Uropathogens, Polymicrobial Urinary Tract Infection, and the Emerging Microbiota of the Urinary Tract. *Microbiol Spectr*. 2016;4(2). doi:10.1128/microbiolspec.UTI-0012-2012.
6. Peipert JF, Lapane KL, Allsworth JE, Redding CA, Blume JD, Stein MD. Bacterial vaginosis, race, and sexually transmitted infections: does race modify the association? *Sex Transm Dis*. 2008;35(4):363-367. doi:10.1097/OLQ.0b013e31815e4179.
7. Allsworth JE, Peipert JF. Severity of bacterial vaginosis and the risk of sexually transmitted infection. *Am J Obstet Gynecol*. 2011;205(2):113.e1-113.e6. doi:10.1016/j.ajog.2011.02.060.
8. Brotman RM, Klebanoff MA, Nansel TR, et al. Bacterial vaginosis assessed by gram stain and diminished colonization resistance to incident gonococcal, chlamydial, and trichomonal genital infection. *J Infect Dis*. 2010;202(12):1907-1915. doi:10.1086/657320.
9. Rezeberga D, Lazdane G, Kroica J, Sokolova L, Donders GGG. Placental histological inflammation and reproductive tract infections in a low risk pregnant population in Latvia. *Acta Obstet Gynecol Scand*. 2008;87(3):360-365. doi:10.1080/00016340801936487.
10. Zhang X, Xu X, Li J, Li N, Yan T, Ju X. [Relationship between vaginal sialidase bacteria vaginosis and chorioammionitis]. *Zhonghua Fu Chan Ke Za Zhi*. 2002;37(10):588-590. http://www.ncbi.nlm.nih.gov/pubmed/12487930. Accessed July 12, 2017.
11. Flynn CA, Helwig AL, Meurer LN. Bacterial vaginosis in pregnancy and the risk of prematurity: a meta-analysis. *J Fam Pract*. 1999;48(11):885-892. http://www.ncbi.nlm.nih.gov/pubmed/10907626. Accessed July 12, 2017.
12. Holst E, Goffeng AR, Andersch B. Bacterial vaginosis and vaginal microorganisms in idiopathic premature labor and association with pregnancy outcome. *J Clin Microbiol*. 1994;32(1):176-186. http://www.ncbi.nlm.nih.gov/pubmed/8126176. Accessed July 12, 2017.
13. McGregor JA, French JI, Jones W, et al. Bacterial vaginosis is associated with prematurity and vaginal fluid mucinase and sialidase: results of a controlled trial of topical clindamycin cream. *Am J Obstet Gynecol*. 1994;170(4):1048-59; discussion 1059-60. http://www.ncbi.nlm.nih.gov/pubmed/8166188. Accessed July 12, 2017.
14. 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(2):297-301. doi:10.1136/sti.2003.006106.
15. Balkus JE, Richardson BA, Rabe LK, et al. Bacterial vaginosis and the risk of

trichomonas vaginalis acquisition among HIV-1-negative women. *Sex Transm Dis*. 2014;41(2):123-128. doi:10.1097/OLQ.0000000000000075.

16. Guédou FA, Van Damme L, Mirembe F, et al. Intermediate vaginal flora is associated with HIV prevalence as strongly as bacterial vaginosis in a cross-sectional study of participants screened for a randomised controlled trial. *Sex Transm Infect*. 2012;88(7):545-551. doi:10.1136/sextrans-2011-050319.
17. Guédou FA, Van Damme L, Deese J, et al. Intermediate vaginal flora and bacterial vaginosis are associated with the same factors: findings from an exploratory analysis among female sex workers in Africa and India. *Sex Transm Infect*. 2014;90(2):161-164. doi:10.1136/sextrans-2012-050896.
18. Donders GGG, Vereecken A, Bosmans E, Dekeersmaecker A, Salembier G, Spitz B. Definition of a type of abnormal vaginal flora that is distinct from bacterial vaginosis: aerobic vaginitis. *BJOG*. 2002;109(1):34-43. http://www.ncbi.nlm.nih.gov/pubmed/11845812. Accessed July 12, 2017.
19. Meyn LA, Krohn MA, Hillier SL. Rectal colonization by group B Streptococcus as a predictor of vaginal colonization. *Am J Obstet Gynecol*. 2009;201(1):76.e1-7. doi:10.1016/j.ajog.2009.02.011.
20. Srinivasan S, Liu C, Mitchell CM, et al. Temporal variability of human vaginal bacteria and relationship with bacterial vaginosis. Ratner AJ, ed. *PLoS One*. 2010;5(4):e10197. doi:10.1371/journal.pone.0010197.
21. Gajer P, Brotman RM, Bai G, et al. Temporal dynamics of the human vaginal microbiota. *Sci Transl Med*. 2012;4(132):132ra52. doi:10.1126/scitranslmed.3003605.
22. Ness RB, Hillier SL, Richter HE, et al. Douching in relation to bacterial vaginosis, lactobacilli, and facultative bacteria in the vagina. *Obstet Gynecol*. 2002;100(4):765-772. doi:10.1016/S0029-7844(02)02184-1.
23. Ocelík V, Furár I, De Hosson JTM. Microstructure and properties of laser clad coatings studied by orientation imaging microscopy. *Acta Mater*. 2010;58(20):6763-6772. doi:10.1016/j.actamat.2010.09.002.
24. Schwebke JR, Richey CM, Weiss HL. Correlation of Behaviors with Microbiological Changes in Vaginal Flora. *J Infect Dis*. 1999;180(5):1632-1636. doi:10.1086/315065.
25. Gilbert NM, Lewis WG, Lewis AL. Clinical Features of Bacterial Vaginosis in a Murine Model of Vaginal Infection with Gardnerella vaginalis. Ratner AJ, ed. *PLoS One*. 2013;8(3):e59539. doi:10.1371/journal.pone.0059539.
26. Gilbert NM, O'Brien VP, Lewis AL. Transient microbiota exposures activate dormant Escherichia coli infection in the bladder and drive severe outcomes of recurrent disease. Mobley HLT, ed. *PLOS Pathog*. 2017;13(3):e1006238. doi:10.1371/journal.ppat.1006238.
27. Turnbaugh PJ, Hamady M, Yatsunenko T, et al. A core gut microbiome in obese and lean twins. *Nature*. 2009;457(7228):480-484. doi:10.1038/nature07540.
28. Xu X, Grijalva A, Skowronski A, van Eijk M, Serlie MJ, Ferrante AW. Obesity Activates a Program of Lysosomal-Dependent Lipid Metabolism in Adipose Tissue Macrophages Independently of Classic Activation. *Cell Metab*. 2013;18(6):816-830. doi:10.1016/j.cmet.2013.11.001.
29. Gregor MF, Hotamisligil GS. Inflammatory mechanisms in obesity. *Annu Rev Immunol*. 2011;29:415-445. doi:10.1146/annurev-immunol-031210-101322.
30. Ding S, Chi MM, Scull BP, et al. High-fat diet: bacteria interactions promote intestinal inflammation which precedes and correlates with obesity and insulin resistance in mouse.

Gaetani S, ed. *PLoS One*. 2010;5(8):e12191. doi:10.1371/journal.pone.0012191.

31. Kim K-A, Gu W, Lee I-A, Joh E-H, Kim D-H. High fat diet-induced gut microbiota exacerbates inflammation and obesity in mice via the TLR4 signaling pathway. Chamaillard M, ed. *PLoS One*. 2012;7(10):e47713. doi:10.1371/journal.pone.0047713.

32. Cani PD, Amar J, Iglesias MA, et al. Metabolic Endotoxemia Initiates Obesity and Insulin Resistance. *Diabetes*. 2007;56(7):1761-1772. doi:10.2337/db06-1491.

33. Oh HY, Seo S-S, Kong J-S, Lee J-K, Kim MK. Association between Obesity and Cervical Microflora Dominated by Lactobacillus iners in Korean Women. Munson E, ed. *J Clin Microbiol*. 2015;53(10):3304-3309. doi:10.1128/JCM.01387-15.

34. Si J, You HJ, Yu J, Sung J, Ko G. *Prevotella as a Hub for Vaginal Microbiota under the Influence of Host Genetics and Their Association with Obesity*. Vol 21. 2017. doi:10.1016/j.chom.2016.11.010.

35. Hillier SL, Krohn MA, Rabe LK, Klebanoff SJ, Eschenbach DA. The normal vaginal flora, H2O2-producing lactobacilli, and bacterial vaginosis in pregnant women. *Clin Infect Dis*. 1993;16 Suppl 4:S273-81. http://www.ncbi.nlm.nih.gov/pubmed/8324131. Accessed August 2, 2018.

36. Fredricks DN, Fiedler TL, Marrazzo JM. Molecular Identification of Bacteria Associated with Bacterial Vaginosis. *N Engl J Med*. 2005;353(18):1899-1911. doi:10.1056/NEJMoa043802.

37. Koumans EH, Sternberg M, Bruce C, et al. The prevalence of bacterial vaginosis in the United States, 2001-2004; associations with symptoms, sexual behaviors, and reproductive health. *Sex Transm Dis*. 2007;34(11):864-869. doi:10.1097/OLQ.0b013e318074e565.

38. Flegal KM, Carroll MD, Kit BK, Ogden CL. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. *Arch Intern Med*. 1998;158(17):1855-1867. doi:10.1001/jama.2012.39.

39. Bjorntorp P, Bray GA, Carroll KK, et al. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser*. 2000;894:i-xii, 1-253. doi:ISBN 92 4 120894 5.

40. Nguyen NT, Magno CP, Lane KT, Hinojosa MW, Lane JS. Association of Hypertension, Diabetes, Dyslipidemia, and Metabolic Syndrome with Obesity: Findings from the National Health and Nutrition Examination Survey, 1999 to 2004. *J Am Coll Surg*. 2008;207(6):928-934. doi:10.1016/J.JAMCOLLSURG.2008.08.022.

41. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The Disease Burden Associated With Overweight and Obesity. *JAMA*. 1999;282(16):1523. doi:10.1001/jama.282.16.1523.

42. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of Overweight and Obesity Among US Children, Adolescents, and Adults, 1999-2002. *JAMA*. 2004;291(23):2847. doi:10.1001/jama.291.23.2847.

43. 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(3):201-212. http://www.ncbi.nlm.nih.gov/pubmed/12749680. Accessed July 3, 2018.

44. Goldenberg RL, Klebanoff MA, Nugent R, Krohn MA, Hillier S, Andrews WW. Bacterial colonization of the vagina during pregnancy in four ethnic groups. Vaginal Infections and Prematurity Study Group. *Am J Obstet Gynecol*. 1996;174(5):1618-1621. http://www.ncbi.nlm.nih.gov/pubmed/9065140. Accessed April 15, 2017.

45. Secura GM, Allsworth JE, Madden T, Mullersman JL, Peipert JF. The Contraceptive CHOICE Project: reducing barriers to long-acting reversible contraception. *Am J Obstet Gynecol*. 2010;203(2):115.e1-115.e7. doi:10.1016/j.ajog.2010.04.017.
46. Prevention C for DC and. About Adult BMI. https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html. Accessed January 1, 2017.
47. Centers for Disease Control. Bacterial Vaginosis - CDC Fact Sheet. 2017. https://www.cdc.gov/std/bv/stdfact-bacterial-vaginosis.htm. Accessed December 12, 2018.
48. Maruthur NM, Bolen SD, Brancati FL, Clark JM. The asociation of obesity and cervical cancer screening: A systematic review and meta-analysis. *Obesity*. 2009;17(2):375-381. doi:10.1038/oby.2008.480.
49. Wee CC, McCarthy EP, Davis RB, Phillips RS. Obesity and breast cancer screening: The influence of race, illness burden, and other factors. *J Gen Intern Med*. 2004;19(4):324-331. doi:10.1111/j.1525-1497.2004.30354.x.
50. Wee CC, Phillips RS, McCarthy EP. BMI and Cervical Cancer Screening among White, African-American, and Hispanic Women in the United States. *Obes Res*. 2005;13(7):1275-1280. doi:10.1038/oby.2005.152.
51. Lokken EM, Richardson BA, Kinuthia J, et al. A prospective cohort study of the association between body mass index and incident bacterial vaginosis . *Sex Transm Dis*. 2018;(5). doi:10.1097/OLQ.0000000000000905.
52. Ravel J, Gajer P, Abdo Z, et al. Vaginal microbiome of reproductive-age women. *Proc Natl Acad Sci*. 2011;108(Supplement_1):4680-4687. doi:10.1073/pnas.1002611107.
53. Kenyon C, Colebunders R, Crucitti T. The global epidemiology of bacterial vaginosis: a systematic review. *Am J Obstet Gynecol*. 2013;209(6):505-523. doi:10.1016/j.ajog.2013.05.006.
54. Neggers YH, Nansel TR, Andrews WW, et al. Dietary intake of selected nutrients affects bacterial vaginosis in women. *J Nutr*. 2007;137(9):2128-2133. http://www.ncbi.nlm.nih.gov/pubmed/17709453. Accessed December 6, 2017.
55. Thoma ME, Klebanoff MA, Rovner AJ, et al. Bacterial Vaginosis Is Associated with Variation in Dietary Indices. *J Nutr*. 2011;141(9):1698-1704. doi:10.3945/jn.111.140541.
56. Marrazzo JM, Fiedler TL, Srinivasan S, et al. Extravaginal reservoirs of vaginal bacteria as risk factors for incident bacterial vaginosis. *J Infect Dis*. 2012;205(10):1580-1588. doi:10.1093/infdis/jis242.
57. Allsworth JE, Peipert JF. Prevalence of Bacterial Vaginosis. *Obstet Gynecol*. 2007;109(1):114-120. doi:10.1097/01.AOG.0000247627.84791.91.

Table 1. Demographics of CHOICE Participants by BMI Category, N=5,918

	All Participants	**Participants by BMI Category (kg/m^2)**					
		Underweight	*Lean*	*Overweight*	*Class I Obese*	*Class II/III Obese*	
		< 18.5	18.5-24.9	25-29.9	30-34.9	≥ 35	
	N=5918	N=174	N=2,312	N=1,540	N=934	N=958	p-value*
Age, mean(SD)	25.3 (5.9)	23.2 (4.7)	24.1 (5.4)	25.6 (6.0)	26.1 (6.2)	26.9 (6.1)	<0.001
Race							
Black	3001 (50.7)	72 (41.4)	870 (37.6)	809 (52.5)	570 (61.0)	680 (71.0)	<0.001
White	2457 (41.5)	84 (48.3)	1250 (54.1)	604 (39.2)	296 (31.7)	223 (23.3)	
Other	460 (7.8)	18 (10.3)	192 (8.3)	127 (8.3)	68 (7.3)	55 (5.7)	
Hispanic	300 (5.1)	9 (5.2)	105 (4.5)	99 (6.4)	53 (5.7)	34 (3.6)	0.014
Monthly income							
None	1226 (20.8)	35 (20.1)	524 (22.7)	304 (19.8)	187 (20.1)	176 (18.4)	<0.001
$1-800	1903 (32.3)	75 (43.1)	780 (33.9)	494 (32.1)	258 (27.7)	296 (31.0)	
$801-1600	1666 (28.2)	45 (25.9)	587 (25.5)	436 (28.4)	295 (31.7)	303 (31.7)	
$1601+	1106 (18.7)	19 (10.9)	413 (17.9)	304 (19.8)	190 (20.4)	180 (18.9)	
Receiving public assistance	2250 (38.1)	48 (27.8)	639 (27.7)	625 (40.6)	445 (47.7)	493 (51.5)	<0.001
Trouble paying for basic necessities	2393 (40.5)	62 (35.6)	828 (35.9)	625 (40.6)	433 (46.4)	445 (46.5)	<0.001
Education							
≤ High school	2007 (33.9)	71 (40.8)	734 (31.8)	535 (34.8)	345 (37.0)	322 (33.6)	<0.001
Some college	2512 (42.4)	67 (38.5)	895 (38.7)	670 (43.5)	408 (43.8)	472 (49.3)	
College graduate	1396 (23.6)	36 (20.7)	683 (29.5)	334 (21.7)	179 (19.2)	164 (17.1)	
Ever smoking	2765 (46.7)	79 (45.4)	1123 (48.6)	731 (47.5)	514 (55.0)	546 (57.0)	0.037
Current smoking	1367 (23.1)	48 (27.6)	550 (23.8)	374 (24.3)	199 (21.3)	196 (20.5)	0.044
Sexual partners last 30 days							
None	1125 (19.2)	21 (12.4)	390 (17.1)	316 (20.7)	191 (20.7)	207 (21.8)	0.004
One	4356 (74.5)	136 (80.0)	1750 (76.8)	1124 (73.6)	673 (72.8)	673 (70.9)	
2 or more	370 (6.3)	13 (7.7)	139 (6.1)	88 (5.8)	61 (6.6)	69 (7.3)	
Lifetime sexual partners							
None	39 (0.7)	0	12 (0.5)	14 (0.9)	4 (0.4)	9 (1.0)	<0.001
One	516 (8.7)	14 (8.1)	253 (10.9)	128 (8.3)	72 (7.7)	49 (5.1)	
2-4	1630 (27.5)	56 (32.2)	680 (29.4)	433 (28.1)	231 (24.7)	230 (24.0)	

5-7	1727 (29.2)	56 (32.2)	646 (27.9)	428 (27.8)	303 (32.4)	294 (30.7)	
8-12	839 (14.2)	15 (8.6)	308 (13.3)	225 (14.6)	136 (14.6)	155 (16.2)	
13 or more	1167 (19.7)	33 (19.0)	413 (17.9)	312 (20.3)	188 (20.1)	221 (23.1)	
Douching in the past 180 days	1340 (22.7)	32 (18.4)	407 (17.6)	354 (23.0)	248 (26.6)	299 (31.2)	<0.001
Douching in the past 30 days	590 (10.0)	19 (10.9)	168 (7.3)	162 (10.6)	99 (10.6)	142 (14.9)	<0.001
Past sexually transmitted infection	2461 (41.6)	63 (36.2)	801 (34.7)	660 (42.9)	441 (47.2)	496 (51.8)	<0.001
Sexually transmitted infection at baseline	518 (8.8)	17 (9.8)	170 (7.4)	132 (8.6)	85 (9.1)	114 (11.9)	0.001
Current menstruation flag	856 (14.5)	19 (10.9)	342 (14.8)	216 (14.0)	129 (13.8)	150 (15.7)	0.458
Current hormonal contraceptive method prior to enrollment	1520 (25.7)	38 (21.8)	636 (27.5)	412 (26.8)	199 (21.3)	235 (24.5)	0.003

Except for age, all demographics are reported as N (%). SD – standard deviation; BMI – body mass index

*p-values were determined using chi-square test (all categorical variables) or linear regression (age). For categorical variables, p-values represent the distribution of a given categorical variable for All Participants and within a specific BMI category, as shown.

Table 2. Nugent Score and Prevalence of BV by BMI Category

	All Participants	**Participants by BMI Category (kg/m^2)**					
		Underweight	*Lean*	*Overweight*	*Class I Obese*	*Class II/III Obese*	
		< 18.5	18.5-24.9	25-29.9	30-34.9	≥ 35	
Nugent score - BV status	N=5918	N=174	N=2,312	N=1,540	N=934	N=958	p-value*
Nugent score							
0-3	2639 (44.6)	78 (44.8)	1170 (50.6)	657 (42.7)	370 (39.6)	364 (38.0)	<0.001
4-6	1618 (27.3)	48 (27.6)	649 (28.1)	415 (27.0)	247 (26.5)	259 (27.0)	
7-10	1661 (28.1)	48 (27.6)	493 (21.3)	468 (30.4)	317 (33.9)	335 (35.0)	
Bacterial vaginosis							
No	4257 (71.9)	126 (72.4)	1819 (78.7)	1072 (69.6)	617 (66.1)	623 (65.0)	<0.001
Yes	1661 (28.1)	48 (27.6)	493 (21.3)	468 (30.4)	317 (33.9)	335 (35.0)	
Symptomatic BV							
No	1376 (82.8)	41 (85.4)	406 (82.4)	379 (81.0)	261 (82.3)	289 (86.3)	0.371
Yes	285 (17.2)	7 (14.6)	87 (17.7)	89 (19.0)	56 (17.7)	46 (13.7)	

All variables are reported as N (%). BV – bacterial vaginosis; BMI – body mass index

*p-values were determined using chi-square test for categorical variables. p-values represent the distribution of a given categorical variable for All Participants and within a specific BMI category, as shown.

Table 3. Mean Difference in Nugent Score by BMI Category Overall and Within Each Race

BMI Category (kg/m^2)	Mean Nugent Score (SD)	Mean Difference in Nugent Score (95% Confidence Interval) Crude	Fully Adjusted*	Final Adjusted**	Black v. White Interaction p-value
All Women†					
< 18.5	4.27 (3.01)	0.30 (-0.14, 0.73)	0.15 (-0.29, 0.58)	0.19 (-0.24, 0.62)	0.557
18.5-24.9	3.90 (2.85)	Referent	Referent	Referent	Referent
25-29.9	4.53 (2.94)	**0.40 (0.22, 0.59)**	**0.29 (0.11, 0.48)**	**0.33 (0.14, 0.51)**	0.891
30-34.9	4.87 (2.99)	**0.61 (0.39, 0.83)**	**0.44 (0.23, 0.66)**	**0.51 (0.29, 0.72)**	0.401
≥ 35	4.93 (2.96)	**0.53 (0.31, 0.75)**	**0.28 (0.07, 0.50)**	**0.37 (0.16, 0.59)**	0.064
Black Women					
< 18.5	5.08 (3.02)	0.10 (-0.62, 0.83)	0.00 (-0.72, 0.72)	0.00 (-0.72, 0.71)	
18.5-24.9	4.98 (3.01)	Referent	Referent	Referent	
25-29.9	5.24 (3.01)	0.26 (-0.03, 0.55)	0.23 (-0.06, 0.52)	**0.30 (0.01, 0.58)**	
30-34.9	5.37 (3.06)	**0.39 (0.07, 0.71)**	**0.34 (0.02, 0.66)**	**0.41 (0.10, 0.73)**	
≥ 35	5.19 (3.00)	0.21 (-0.09, 0.51)	0.07 (-0.23, 0.38)	0.18 (-0.12, 0.48)	
White Women					
< 18.5	3.63 (2.79)	0.43 (-0.15, 1.01)	0.23 (-0.34, 0.81)	0.30 (-0.28, 0.87)	
18.5-24.9	3.21 (2.51)	Referent	Referent	Referent	
25-29.9	3.62 (2.70)	**0.42 (0.16, 0.67)**	0.24 (-0.02, 0.49)	0.24 (-0.01, 0.49)	
30-34.9	3.99 (2.78)	**0.78 (0.45, 1.11)**	**0.51 (0.18, 0.84)**	**0.56 (0.23, 0.89)**	
≥ 35	4.08 (2.71)	**0.88 (0.50, 1.25)**	**0.51 (0.13, 0.88)**	**0.58 (0.21, 0.95)**	

BMI – body mass index; SD – standard deviation; statistically significant values are in bold.
* Fully adjusted model included income, public assistance, trouble paying for basics, education, number of sex partners in the last 30 days, lifetime number of sex partners, current tobacco use, douching in last 30 days, douching in last 180 days, history of sexually transmitted infection, current sexually transmitted infection.
** Final model adjusted for public assistance, education, current smoker, douching in the last 30 days and sexually transmitted infection at baseline.
† The All Women model was also adjusted for race.

Table 4. Prevalence Ratio of BV by BMI Category Overall and Within Each Race

BMI Category (kg/m^2)	BV Prevalence	Prevalence Ratio (95% Confidence Interval) Crude	Fully Adjusted*	Final Adjusted**	Black v. White Interaction p-value
All Women†					
< 18.5	27.6%	1.25 (0.98, 1.60)	1.18 (0.92, 1.51)	1.20 (0.94, 1.54)	0.314
18.5-24.9	21.3%	Referent	Referent	Referent	Referent
25-29.9	30.4%	**1.28 (1.15, 1.43)**	**1.23 (1.10, 1.36)**	**1.25 (1.12, 1.39)**	**0.024**
30-34.9	33.9%	**1.36 (1.20, 1.53)**	**1.26 (1.12, 1.42)**	**1.31 (1.16, 1.47)**	**0.001**
≥ 35	35.0%	**1.31 (1.16, 1.48)**	**1.20 (1.07, 1.35)**	**1.25 (1.11, 1.41)**	**0.002**
Black Women					
< 18.5	38.9%	1.11 (0.82, 1.50)	1.08 (0.80, 1.46)	1.07 (0.79, 1.45)	
18.5-24.9	35.2%	Referent	Referent	Referent	
25-29.9	39.1%	1.11 (0.98, 1.26)	1.09 (0.97, 1.24)	1.12 (0.99, 1.27)	
30-34.9	39.8%	1.13 (0.99, 1.30)	1.11 (0.97, 1.27)	**1.14 (1.00, 1.31)**	
≥ 35	37.8%	1.07 (0.94, 1.23)	1.03 (0.90, 1.17)	1.07 (0.98, 1.18)	
White Women					
< 18.5	19.1%	1.53 (0.96, 2.43)	1.37 (0.86, 2.18)	1.44 (0.92, 2.25)	
18.5-24.9	12.5%	Referent	Referent	Referent	
25-29.9	19.9%	**1.59 (1.28, 1.98)**	**1.42 (1.14, 1.76)**	**1.44 (1.16, 1.79)**	
30-34.9	24.7%	**1.98 (1.54, 2.53)**	**1.69 (1.31, 2.17)**	**1.73 (1.35, 2.22)**	
≥ 35	24.2%	**1.94 (1.47, 2.55)**	**1.56 (1.18, 2.07)**	**1.63 (1.23, 2.15)**	

BV – bacterial vaginosis; BMI – body mass index; statistically significant values are in bold.
* Fully adjusted model included income, public assistance, trouble paying for basics, education, number of sex partners in the last 30 days, lifetime number of sex partners, current tobacco use, douching in last 30 days, douching in last 180 days, history of sexually transmitted infection, current sexually transmitted infection.
** Final model adjusted for public assistance, education, current smoker, douching in the last 30 days and sexually transmitted infection at baseline.
† The All Women models also adjusted for race.