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Bacterial Vaginosis and the Risk for Sexually Transmitted Infections

Steven E. Chavoustie, MD, FACOG, CCRP; Adriana Sofia Maribona, BS; Michael Hanna, PhD

Introduction

Bacterial vaginosis (BV) is a polymicrobial vaginal dysbiosis characterized by a decline in the lactic acid-producing native *Lactobacillus* species and an overgrowth of the facultative anaerobic bacteria (*Gardnerella vaginalis*) and other anaerobic bacteria (e.g., *Prevotella bivia*, *Atopobium vaginae*, and *Peptostreptococcus* species). *G. vaginalis* has been studied most in BV pathogenesis and is present in 95% to 100% of cases and is more virulent than other BV-associated bacteria.[1]

BV is the most prevalent cause of abnormal vaginal discharge, affecting over 21 million women between the ages of 15 and 44 years in the United States annually, with recurrence rates as high as 58%.[2,3] More than half of patients are asymptomatic. Symptoms include a milky, white-gray, malodorous vaginal discharge causing vulvovaginal discomfort and a "fishy" odor after sexual intercourse. Diagnosis is achieved by satisfying 3 of 4 Amsel criteria: a homogenous, thin white-gray discharge; vaginal side-wall pH of > 4.5; > 20% clue cells on microscopy; and a positive 10% potassium hydroxide whiff test. Amsel criteria have a sensitivity of 92% for making a diagnosis.[4]

Risk factors for acquiring BV include multiple sexual partners, women who have sex with women, diet, douching, cigarette smoking, uncircumcised partners, and African American/Hispanic heritage.[5] Women with BV are at an increased risk for pelvic inflammatory disease, preterm labor, endometritis, and acquiring sexually transmitted infections (STIs).[6] The epidemiology of BV robustly implies that it is acquired through sexual transmission.[1]

> BV affects over **21 million women** between the ages of **15 and 44 years** in the United States annually, with recurrence rates as high as **58%**.

This supplement discusses updated treatment guidelines for BV and reviews the available literature on the association of BV and the risk of STIs that supports the need for proper BV treatment, whether symptomatic or asymptomatic.

Treatment guidelines

Current guidelines from the American College of Obstetricians and Gynecologists (ACOG), updated in January 2020, recommend the treatment of symptomatic BV to restore the vaginal microbiome to a healthy state and reduce a woman's risk of acquiring and transmitting other STIs.[7] Although guidelines currently do not include treatment of asymptomatic BV, some prospective data show that treating asymptomatic BV may also reduce the incidence of STIs.[8]

Oral and intravaginal treatment options recommended by ACOG are shown in Table 1 and include metronidazole, clindamycin, secnidazole, and tinidazole. Because these treatments show comparable efficacy and safety, ACOG recommends that choice of treatment be individualized based on patient preference, cost, convenience, adherence, ease of use, and history of response or adverse reactions to previous treatments. Patients who experience gastrointestinal adverse events with oral metronidazole may prefer the intravaginal formulation. Secnidazole is a newer US Food and Drug Administration–approved, single-dose oral agent. In randomized clinical trials, it has been found to be superior to placebo and comparable to metronidazole.

> Patients with BV are at **increased risk of acquiring STIs** and need appropriate treatment.

Other clinical considerations for BV treatment according to ACOG guidelines include alcohol use, sexual activity, contraceptive use, and tampon use.[7] Abstaining from alcohol

Treatment guidelines *(cont.)*

during treatment and for 24 hours after completion of oral metronidazole treatment or 72 hours after oral tinidazole treatment is currently recommended by the drug manufacturers. Alternatively, in vitro studies have shown that secnidazole has no effect on aldehyde dehydrogenase activity in metabolizing alcohol. Clindamycin ovules use an oleaginous base that might weaken latex or rubber, so use of products such as condoms and vaginal contraceptives is not recommended within 72 hours after this treatment. Patients should refrain from sexual activity during BV treatment unless condoms are used. Tampons should also be avoided during treatment with intravaginal products.

TABLE 1
Guidelines from the American College of Obstetricians and Gynecologists (ACOG): Treatment options for bacterial vaginosis in nonpregnant patients

Recommended Treatment Regimens	Alternative Treatment Regimens
– Metronidazole, 500 mg orally twice daily for 7 days* or – Metronidazole gel 0.75%, one full applicator (5 g) intravaginally, once a day for 5 days* or – Clindamycin cream 2%, one full applicator (5 g) intravaginally at bedtime for 7 days	– Secnidazole, 2 g orally in a single dose or – Tinidazole, 2 g orally once daily for 2 days* or – Tinidazole, 1 g orally once daily for 5 days* or – Clindamycin, 300 mg orally twice daily for 7 days or – Clindamycin ovules, 100 mg intravaginally once at bedtime for 3 days†

*Abstaining from alcohol during treatment with nitroimidazoles and for 24 hours after completion of oral metronidazole treatment or 72 hours after treatment with oral tinidazole is currently recommended by the drug manufacturers because of a theoretical concern about a disulfiram-like reaction that may occur with the use of nitroimidazoles (Pfizer Inc. 2018. Flagyl [metronidazole] tablets. New York, NY: Available at http://labeling.pfizer.com/showlabeling.aspx?id=570, and Mission Pharmaceutical Company. 2004. Tindamax (tinidazole) tablets. San Antonio, TX. Available at https://www.accessdata.fda.gov/drugsatfda_docs/label/2007/021618s003lbl.pdf).

†Clindamycin ovules use an oleaginous base that might weaken latex or rubber products (eg, condoms and vaginal contraceptive diaphragms). Use of such products within 72 hours after treatment with clindamycin ovules is not recommended.

Adapted from Table 2, p. 6, in AGOG Practice Bulletin Number 215, Vaginitis in Nonpregnant Patients, January 2020.

Literature review

To gain a better understanding of how BV, with its concomitant vaginal dysbiosis, increases the risk for acquiring STIs, the literature on longitudinal studies was searched and summarized. Table 2 shows the risks and evidence for each of the infections discussed here.

HUMAN IMMUNODEFICIENCY VIRUS

Human immunodeficiency virus (HIV) has become rather uncommon, with an annual incidence among US women of about 7000 new diagnoses.[9] Left untreated, however, HIV can eventually start destroying the CD4 cells, which are critical to the immune system.[10]

Two high-quality meta-analyses that report the relation of BV to incident HIV have been published by the same research network from Bern, Switzerland; London, United Kingdom; and Ghent, Belgium. The first of these included eight studies that reported the effect of BV on the risk for incident HIV.[11] The studies covered a total of 10,898 African women, and there was little heterogeneity between the studies. This meta-analysis found that BV increased the risk for incident HIV, with an overall adjusted effect of 1.57 (95% confidence interval [CI], 1.26–1.94). In other words, BV raises the risk for acquiring HIV by about 57% (because the effect size is 1.57) and this result is statistically significant (because even the low end of the 95% CI is still above 1.0).

The second meta-analysis pooled the original raw data of over 8400 African women from eight published studies (none of which was included in the first meta-analysis).[12] The authors then performed two meta-analyses on disrupted vaginal flora as a possible risk for incident HIV: one for vaginal flora assessed at study baselines (N = 8452; median time to estimated HIV acquisition: 253 days), and the other for vaginal flora assessed at the follow-up visit prior to the visit when incident HIV was first diagnosed (N = 8626; median time to HIV: 53 days). The first meta-analysis found that baseline BV (Gram score 7–10 or Ison-Hay grade III) was a risk factor for incident HIV, with a hazard ratio (HR) of 1.69 (95% CI, 1.36–2.10). Baseline intermediate vaginal flora (Gram score 4–6 or Ison-Hay grade II) was also a risk factor for incident HIV, with an HR of 1.54 (95% CI, 1.20–1.97). The second meta-analysis found that BV at the follow-up visit prior to HIV detection was a risk factor for that subsequent HIV, with an HR of 1.53 (95% CI, 1.24–1.89). Intermediate vaginal flora at the follow-up visit prior to HIV was also a risk factor for that subsequent HIV, with an HR of 1.41 (95% CI, 1.12–1.79). The authors did not comment on why disrupted vaginal flora at baseline was a slightly stronger risk factor than at the follow-up visit prior to HIV detection, but the results of these two meta-analyses are essentially consistent. Importantly, all four CIs excluded the possibility that BV might not be a risk factor for incident HIV. This meta-analysis of pooled raw data was highly consistent with the previous meta-analysis of reported study outcomes from the same research network.

Another major report pooled data from four phase III randomized controlled trials (not included in the previous meta-analyses) on 7024 HIV-negative women mostly from Africa but some from India.[13] They found that baseline BV (Nugent score or Amsel criteria, dichotomized) increased the risk for incident HIV, with an HR of 1.41 (95% CI, 0.96–2.06). This is a bit lower than the two meta-analyses above (perhaps due to the definition of BV) but nonetheless is consistent with them.

So altogether, results from over 26,000 women in 20 studies have consistently shown that BV raises the hazard of incident HIV about 50%. Given the large sample size, the consistency of results, and the sufficient narrowness of the CIs, this estimate seems highly reliable and is unlikely to change much with further research.

Literature review *(cont.)*

CHLAMYDIA TRACHOMATIS

Chlamydia trachomatis (CT) is a common bacterial STI, with nearly 1.1 million reported female cases in the United States in 2018, most occurring in women age 15 to 24 years.[14] Chlamydia usually remains asymptomatic, but it can instead cause cervicitis, urethritis, and/or pelvic inflammatory disease, which can in turn lead to infertility or preterm delivery.[15]

In a systematic review published in early 2019, Tamarelle et al searched the literature through December 2016, and three longitudinal studies were identified that assessed BV as a risk factor for subsequent acquisition of CT, all of which were graded as good quality.[16] Those three studies included just over 5000 women from the United States, the United Kingdom, and South Africa, and their results appeared consistent with each other. The researchers performed a meta-analysis on these studies and calculated that BV had an effect size of 1.69 (95% CI, 1.33–2.04) on the subsequent acquisition of CT.[16]

Nonetheless, another report pooled data from two phase III randomized controlled trials (neither of which was included in the meta-analysis above) on 2700 HIV-negative women mostly from Africa but some from India. They found that BV was not a risk factor for CT, with an HR of 1.08 (95% CI, 0.76–1.55).[13]

A search of PubMed in January 2020 using the same terms from the meta-analysis by Tamarelle et al identified three new relevant articles that have been published since that literature search.[16–19] First, a sophisticated analysis of 3620 US women found that BV was not a risk factor for CT at subsequent visit, with an odds ratio (OR) of 1.1 (95% CI, 0.9–1.3).[17] The researchers provided supporting evidence and discussion that the association usually observed in other studies may be due to uncontrolled confounding by the behavioral structure of study subjects' sexual networks.

Second, a recent US-led study on 934 women, age 18 to 35 years, participating in a hormonal contraceptive clinical trial in Zimbabwe and Uganda found that women with BV (Nugent score 7–10) had a likelihood ratio (LR) of 1.67 to predict CT at the next visit, but this was not statistically significant.[18] The authors remarked that levels of CT were already high in their sample at baseline, thus limiting their statistical power to predict incident infections. If combined into the previously discussed meta-analysis, this study would increase the statistical precision of that estimate without changing the estimate itself.

Third, a matched case-control analysis of 115 women in Amsterdam reported that vaginal microbiota community state type III (dominated by *Lactobacillus iners*) increased the risk for incident CT infection compared to community state type I (dominated by *L. crispatus*), with an OR of 2.58 (95% CI, 1.01–6.61), while community states IV (dominated by non-*Lactobacillus* species) and V (dominated by *L. jensenii*) showed no statistically significant risk and community state type II was not observed.[19] This study was too small to have any influence on the overall pooled estimate of BV as a risk for CT, but it provides interesting pilot information for further research to better differentiate which vaginal bacteria species increase the risk of CT.

A recent narrative review identified another longitudinal study on BV as a risk for CT that predated the search window of the meta-analysis by Tamarelle et al.[16,20] That study, on 657 Kenyan sex workers, found nonsignificant results closer to the pooled analysis of data from the microbicide trials, with a univariate HR of 1.2 (95% CI, 0.8–1.9), for a Nugent score of 4 to 10 versus 0 to 3.[13,21]

Thus, altogether, it would seem that BV increases the risk of CT by about 40%, but some studies in the literature have not found an increase of risk.

HUMAN PAPILLOMAVIRUS

Human papillomavirus (HPV) is the most common STI, with an estimated US prevalence of 79 million people.[22] Although most often HPV is asymptomatic and resolves spontaneously without serious consequences, it can instead lead to cervical or genital cancer.

A recent, high-quality meta-analysis searched the literature until mid-June 2018 and identified four longitudinal studies on BV (diagnosed by microscopy) as a possible risk factor for HPV.[23] Three of these studies were from the United States and rated as high quality; the other one from the United Kingdom would not have changed the overall results. Altogether, they included over 5000 women and their results were very consistent. They showed that BV had a pooled relative risk (RR) for subsequent HPV acquisition of 1.33 (95% CI, 1.18–1.50). This meta-analysis also identified two further moderate-quality studies that were small but used more recent molecular techniques to assess the vaginal microbiome. Synthesizing their results (N = 96), Brusselaers et al reported that a vaginal microbiome dominated by species other than *L. crispatus* seemed to be a risk factor for incident HPV but not significantly so, with an RR of 1.85 (95% CI, 0.47–7.32).[23] These two small studies would have a negligible influence on the overall risk estimate cited, but they suggest that molecular techniques may allow more precise identification of risk profiles. This meta-analysis also provided further evidence that women with BV are at an elevated risk for HPV persistence and progression to cervical malignancy. The authors concluded that "Improved... vaginal dysbiosis prevention and management will likely reduce cervical cancer disease burden significantly."[23]

Literature review *(cont.)*

HUMAN PAPILLOMAVIRUS *(cont.)*

It might be, however, that HPV increases the risk for BV, not vice versa.

The meta-analysis by Brusselaers et al also identified one other study that they excluded—spuriously—for "insufficient data."[23] That US study assessed the temporal relationship between BV (by Amsel criteria) and HPV (by PCR for DNA) in 516 university students, tested every 4 months for up to 4 years, yielding 3792 study visits.[24] In a time-lag analysis, they found that HPV was a significant predictor of BV (statistically significant ORs of 2–3, for all time-lag intervals of HPV preceding BV by 4–20 months), but BV was never a predictor of HPV (statistically nonsignificant ORs very close to 1.0 at all time-lag intervals of BV preceding HPV by 4–20 months). The authors concluded, "Our findings also suggest that there is a temporal relationship between HPV and BV, with HPV infection generally preceding BV. Whereas treatment of BV is unlikely to have an impact on the risk of acquiring genital HPV infection, prevention of HPV infection through vaccination or behavior change may reduce the incidence of BV among sexually active young women."[24]

In January 2020, the PubMed search strategy from Brusselaers et al and the search terms from the meta-analysis by Tamarelle et al were re-used.[16,23] This identified one other new report on BV as a risk factor for incident HPV. A substudy (n = 304) on HIV-positive women, age 25 to 50 years, in South Africa assessed the influence of the vaginal microbiome on acquisition and clearance of high-risk HPV.[25] Baseline vaginal microbiome composition was neither a risk factor for acquisition of high-risk HPV nor a facilitative factor for clearance of high-risk HPV, from baseline to endline (median, 16 months later). The authors interpreted this and further results as "suggesting that high-risk HPV acquisition altered the vaginal microbiome rather than vice versa."[25] So according to the currently available evidence, it seems that BV raises the risk of HPV by about 30%, but controversy remains about the temporal relationship between the two.

BV raises the risk of **HPV** by about **30%**.

HERPES SIMPLEX VIRUS TYPE 2

Herpes simplex virus type 2 (HSV-2) is the main cause of genital herpes. The Centers for Disease Control and Prevention (CDC) estimates that HSV-2 has a prevalence of about 16% among American women age 14 to 49 years and an incidence of 776,000 new infections annually in the total population.[26] Although most infected persons remain asymptomatic or subclinical, some other infected people do have pains, aches, fever, and swollen lymph nodes, or rarely other more serious complications. HSV-2 can also be passed to a baby during birth.

A search of PubMed in January 2020 used the terms "herpes OR HSV-2" and the non-STI search terms from the meta-analysis by Tamarelle et al.[16] All restricted to the title/abstract, this search identified four longitudinal studies analyzing BV as a possible risk factor for subsequent HSV-2. The largest of these studies—a recent US-led study on 934 women, age 18 to 35 years, participating in a hormonal contraceptive clinical trial in Zimbabwe and Uganda—found that BV (Nugent score 7–10) was a statistically significant and strong predictor of incident HSV-2 (LR, 2.5), but mere vaginal dysbiosis (Nugent score 4–6) was not relevant.[18] Interestingly, the researchers reported that acquisition of BV and STIs are preceded by aberrant cervical immunity mediators in the preceding months. They suggested that the increased rates of STIs in women with BV may occur because BV alters the levels of biomarkers of the human immune system or because women with BV already have previously altered immunity. This theory of altered immunity differs substantially from the more commonly proposed explanations that certain bacterial species in the healthy vaginal microbiome provide a direct protective effect against STIs (e.g., by lowering pH, competing for metabolites and adhesion sites).

The second largest of these studies—on 670 sexually active HSV-2-negative US women—found that BV (Nugent score 7–10) was a risk factor for HSV-2 antibodies 4 months later, compared to the normal reference group (Nugent score 0–3), with an HR of 2.1 (95% CI, 1.0–4.5).[27] The study authors also calculated that the portion of HSV-2 in the population that is attributable to BV is about 21% and remarked that, "It seems likely that more-comprehensive screening and appropriate treatment of BV would reduce susceptibility to the acquisition of HSV-2 among women."[27]

By contrast, the next largest study, on almost 600 women working in the highway bar sector in Tanzania, found no relation of BV (dichotomized at an unspecified Nugent score) to incident HSV-2 at subsequent visit, with an OR of 1.02 (95% CI, 0.29–3.54).[28]

A smaller study reported on 297 HIV-negative Kenyan sex workers who were HSV-2 negative at baseline and had HSV-2 follow-up data, and who were provided with free condoms, counseling, and treatment of STIs during monthly follow-up for a median of 11 months.[29] They found that BV (tested by Gram staining, and dichotomized at an unspecified cutoff) was a risk factor for HSV-2 seroconversion (seemingly within the subsequent 60-day window), with an HR of 1.56 (95% CI, 0.96–2.55).

Based on these four studies, it seems that BV increases the risk for HSV-2, probably almost by doubling it.

MYCOPLASMA GENITALIUM

Mycoplasma genitalium (MG) is a tiny, sexually transmittable bacterium that is not new but has only recently been gaining attention as it becomes resistant to antibiotics.[30] The CDC states that it is "more common than N. gonorrhoeae but less common than *C. trachomatis*."[31] MG is usually asymptomatic, but it can cause vaginal bleeding, pelvic inflammatory disease (and thus infertility), miscarriage, or stillbirth.[30]

A recent systematic review and meta-analysis searched the literature until December 2016 and found only four articles on the association of BV and MG, only one of which contained longitudinal information on BV as a risk factor for subsequent acquisition of MG.[16] That study included an analysis of 873 sexually active students in London who provided follow-up vaginal specimens by mail at about 1-year follow-up.[32] BV was a strong risk factor for subsequent MG infection, with an RR of 6.01 (95% CI, 1.98–18.50).

A search of PubMed in January 2020 used the search terms from the meta-analysis by Tamarelle et al.[16] It yielded four new papers published since their search on BV as a risk factor for subsequent MG acquisition. First, a study of 280 sex workers in Kenya, with nearly monthly follow-up visits during a year of observation, also reported that BV (Nugent ≥ 7) was a statistically significant strong risk factor for incident MG at the next visit compared to women without BV (Nugent 0–3), with an OR of 3.49 (95% CI, 1.86–6.56).[33]

Another recent paper reported a post-hoc analysis on 221 HIV-negative sexually active women in a randomized controlled trial, mostly from Kenya but some from the United States, with follow-up testing for MG bimonthly for a year.[34] They found no relationship between BV (Nugent 7–10 vs. 0–6) and incident MG at the subsequent visit, with a univariate HR of 0.95 (95% CI, 0.50–1.81). The authors suggested that the monthly provision of health interventions (free male condoms for all, and metronidazole 750 mg and miconazole 200 mg for 5 nights per month for half the participants) might be part of the reason for the difference of results compared to the previous study.[33]

Another study of 246 women from US urban outpatient clinics, with quarterly follow-up for 1 year, found no statistically significant relation between baseline BV and incident MG, with a univariate HR of 1.3 (95% CI, 0.6–2.6).[35] Women diagnosed with BV at baseline received 2 g oral metronidazole treatment, which may have diminished any observable association with subsequent MG infection.

Finally, a study on 244 Kenyan sex workers reported that BV was not a risk factor for MG, with an HR of 1.14 (95% CI, 0.70–1.94).[36] However, this study had several deficiencies of analysis and/or reporting that make it difficult to interpret and unreliable.

Thus, the literature seems divided: two studies on 1153 women found that BV was a strong risk factor for MG, while three others on a total of 711 women found that BV was not a significant risk factor, perhaps due to treatment. So although BV should be considered a risk factor for incident MG, further research is needed and might easily change the overall estimate of risk.

TRICHOMONAS VAGINALIS

Trichomonas vaginalis (TV) is a protozoan parasite that causes trichomoniasis. It is the most common curable STI, with an estimated US prevalence of 3.7 million, and is more frequent in women than men.[6] Only about a third of infected people are symptomatic, but symptoms in women can include genital itching, burning, discomfort, soreness, and altered vaginal discharge. TV also increases the risk for preterm and low-weight childbirth. A search of PubMed identified 13 longitudinal studies on BV as a possible risk factor for TV, reporting on over 20,000 women altogether. To date, there has not been a systematic review or meta-analysis performed on these studies. The largest study (N = 3620), a sophisticated analysis published in 2018, reported that BV was a weak risk factor for incident TV with an adjusted OR of 1.3 (95% CI, 1.0–1.6).[17] By contrast, the second largest study (N = 3077), published in 2010, reported that BV was a strong risk factor for incident TV with an HR of 2.94 (95% CI, 2.27–3.81).[37] The heterogeneity between these two studies is found in the other 11 studies as well, but altogether the available literature suggests a statistically significant risk from BV for incident TV, seemingly between about 1.5 and 2.0. Although these 13 studies clearly suggest an association, a more thorough systematic review of the literature and a formal meta-analysis are still needed to reliably determine the risk estimate.

TABLE 2
Summary overview of how BV affects the risk for STIs

STI	Relative risk	Number of studies	N	Comments
HIV	**About 1.5**	20	26,000+	Literature is extensive and consistent, with high-quality meta-analyses
CT	**About 1.4**	9	13,000+	Literature is mostly consistent but has some negative studies
HPV	**1.33 (1.18–1.50)**	5	5500+	Risk based on meta-analysis of 4 studies with N > 5000 in Brusselaers et al[23]; 2 other studies (N = 820) argue instead that HPV is a risk for BV, not vice versa
HSV-2	**About 1.9**	4	2500	Literature is neither extensive nor consistent; no meta-analysis available; further research is needed
MG	**Probably strong**	5	1800+	Literature is inconsistent; further research is needed
TV	**Probably 1.5–2.0**	13	20,000+	Literature is substantial but not consistent; no meta-analysis available

BV, bacterial vaginosis; CT, *Chlamydia trachomatis*; HIV, human immunodeficiency virus; HPV, human papillomavirus; HSV-2, herpes simplex virus type 2; MG; *Mycoplasma genitalium*; STI; sexually transmitted infection; TV, *Trichomonas vaginalis*.

Discussion

Bacterial vaginosis is one of the most common vaginal infections in women. A microbiome dominated by *Lactobacillus* species, particularly *L. crispatus*, is optimal for vaginal health. Production of lactic acid by the lactobacilli has been shown to inhibit the growth of pathogenic bacteria in the vagina. Lactobacilli produce hydrogen peroxide and bacteriocins, which can kill urogenital pathogens in vitro under various conditions. These bacteria inhabiting the vagina serve as the first line of defense against vaginal infection as a result of both the competitive exclusion and direct killing of other pathogenic microbes.[5]

The several meta-analyses mentioned found a positive correlation between baseline BV and an increased risk for incident HIV. Additional studies have identified a strong correlation between baseline and recurrent BV and subsequent CT, HPV, MG, TV, and HSV-2 infection. Based on the reviewed investigations, it appears that BV increases the risk of CT by about 40%. Per the currently available evidence analyzed, BV raises the risk of HPV by about 30%. Many studies have been conducted to explore the relationship between BV and MG and TV, respectively. Regarding MG, two studies (N = 1153) found that BV was a strong risk factor, while three other studies (N = 711) found that BV was not a risk factor, perhaps due to treatment of BV. Similarly, based on several large studies, it seems that the risk of BV for incident TV is probably between 1.5 and 2.0.

In conclusion, vaginal dysbiosis, which is largely under studied and misunderstood, is a significant risk factor for acquiring STIs. Patients with a confirmed diagnosis of BV should be counseled and offered additional STI testing. Effective preventive strategies to reduce the risk of BV should be further studied and implemented. This narrative review supports the treatment of symptomatic and asymptomatic BV as a potential risk-reduction strategy for acquiring STIs.

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ABOUT THE AUTHORS

Steven E. Chavoustie, MD, FACOG, CCRP is a principal investigator at Segal Trials for Clinical Research and voluntary assistant professor of obstetrics and gynecology at University of Miami Miller School of Medicine.

Adriana Sofia Maribona received a BS in Health Sciences at Saint Louis University and is currently an MD candidate, class of 2024. She also works as a clinical research assistant at Segal Trials.

Michael Hanna, PhD, of Mercury Medical Research & Writing, Inc., searched and reviewed the literature and provided writing services on the literature review section.

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