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Rectal Colonization by Group B *Streptococcus* as a Predictor of Vaginal Colonization

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Abstract

Objective—To describe rectal colonization by Group B *Streptococcus* (GBS) and its role in predicting vaginal colonization.

Study Design—In this prospective cohort of 1248 nonpregnant women, vaginal and rectal swabs for GBS culture were obtained at enrollment and three 4-month intervals. Generalized estimating equations were used to identify factors associated with colonization.

Results—Eight hundred fourteen (65%) women were colonized by GBS sometime during the observation period. Rectal GBS colonization was the strongest predictor of vaginal colonization (adjusted odds ratio = 14.3; 95% confidence interval: 11.9 to 17.1). Recent sexual intercourse, vaginal colonization with yeast, and a vaginal Nugent score ≥ 4 were also independent determinants of vaginal GBS colonization. Antimicrobial use decreased vaginal GBS colonization only among women lacking rectal colonization.

Conclusions—GBS in the gastrointestinal tract is a risk factor for vaginal GBS. Sexual activity and abnormal vaginal microflora are independent determinants of vaginal GBS colonization.

Keywords

Group B *Streptococcus*; streptococcal infection/epidemiology; vaginal microflora; sexual behavior

INTRODUCTION

Group B *Streptococcus* (GBS) has been isolated from genital or rectal cultures from women at rates ranging between 5 and 40%, depending on the population studied, number of anatomical sites sampled, and method of detection (1). The gastrointestinal tract is considered to be the reservoir for GBS based on studies reporting higher carriage rates in the rectum than in the vagina (2–5). Some authors posit that sexual transmission may also play a role in GBS

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While rectal colonization by GBS is the greatest predictor of vaginal GBS, recent sexual activity and altered vaginal flora also increase vaginal GBS colonization

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colonization of the genital tract, since 45–91% of male partners of GBS positive women are colonized with the identical serotype and/or strain (6–8). However, few studies have explored site-specific risk factors for GBS colonization. A study of 241 young female college dormitory residents reported that a history of yeast infection, infrequent hand-washing, and pork consumption are risk factors for GBS colonization (9). In a cross-sectional study of 1248 young, nonpregnant women, black race, concurrent vaginal yeast colonization, intermediate Nugent score, and recent sexual intercourse were associated with vaginal colonization irrespective of rectal GBS colonization status (10). The purpose of this secondary longitudinal analysis of the same cohort was to describe the risk factors for rectal GBS colonization over time and the role of rectal GBS in predicting vaginal colonization in young, nonpregnant women. A secondary objective of this study was to determine whether vaginal colonization with GBS was associated with self-reported urogenital symptoms.

MATERIALS AND METHODS

Nonpregnant women between the ages of 18 and 30 years, who were able to provide written informed consent and willing to return for scheduled follow-up visits, were recruited from three clinics in Pittsburgh, PA; University of Pittsburgh Student Health Clinic, Allegheny County Health Department Clinic, and Family Health Council Clinic of Aliquippa. Exclusion criteria included current vaginal bleeding, current use of systemic antimicrobials active against GBS (penicillins, cephalosporins, or macrolides), use of an intravaginal product in the past 24 hours (douche, antifungal products, or spermicides), or conditions that would prevent participation in scheduled follow-up visits. From 1998–2000, 1248 women were enrolled. The Institutional Review Board of Magee-Womens Hospital in Pittsburgh, PA approved the study.

Vaginal and rectal swabs for culture detection of GBS, a vaginal smear, and demographic and behavioral interview data were obtained from the women at enrollment and at 4-month intervals for 12 months. The interviews were administered by trained research personnel and consisted of closed format questions pertaining to demography, cigarette, alcohol, and marijuana use, vaginal product and systemic antimicrobial use, contraception, sexual activity, and urogenital symptoms during the past 4 months.

The vaginal and rectal swabs were processed in a single laboratory for detection of GBS as previously described (10,11). GBS was identified on the basis of colony morphology, beta hemolysis, catalase reaction, and group typing as previously described (10). Growth of GBS was reported as negative, broth only, and 1 to 4+. Yeast was detected on Sabouraud Dextrose agar plates (Gibson Laboratories, Inc., Lexington, KY). *Lactobacillus* and *E. coli* were identified by colony and Gram stain morphology. The vaginal smear was Gram-stained and evaluated for bacterial vaginosis according to the Nugent criteria (12). Normal vaginal flora received a score from 0–3, intermediate flora 4–6, and bacterial vaginosis 7–10. GBS colonization was defined as detection of growth from agar or broth culture. All statistical analyses were performed using Stata statistical software release 9.1 (Stata Corp., College Station, TX). All statistical tests were evaluated at the 0.05 significance level. Generalized estimating equations (GEE) were used to identify factors that were associated with the marginal prevalence of GBS colonization. GEE was also used to identify microorganisms associated with the marginal prevalence of self-reported urogenital symptoms during the past 4 months (dysuria, pruritus, vaginal burning/pain, abnormal vaginal odor, abnormal vaginal discharge amount and consistency). GEE is a regression method that models the marginal expectation of repeated, correlated outcomes. This method was appropriate for these data since the GBS colonization status in these women during the period of observation was transient and highly variable. It was not possible to distinguish between a new, continuing, or recurrent colonization at each 4-month interval. An exchangeable working correlation matrix was specified and modified sandwich estimates of the variance were calculated. Statistical inference was based

on the generalized Wald test statistic (13). Multivariable GEE models were developed using forward stepwise regression. No significant variable interactions were found. The unadjusted and adjusted population averaged odds ratios derived from the main effects GEE models are presented along with the 95% confidence intervals and p-values.

Missing visits were excluded from the analysis. At least one follow-up visit was obtained for 1,089 women (87%) and 710 (57%) completed all four scheduled visits. Women with missed visits were younger, more likely to report tobacco and marijuana use, and more likely to have more sexual partners and bacterial vaginosis at enrollment ($p < 0.05$). Incomplete follow-up was not associated with GBS colonization status at enrollment or with history of sexually transmitted infections ($p \geq 0.2$). Since marijuana use, sexual activity, and abnormal vaginal microflora were found to be associated with GBS colonization in this study, the loss of data from missed visits may have had the effect of underestimating the odds ratios for GBS colonization.

RESULTS

Of the 1248 women enrolled, 764 (61%) were white, 434 (35%) were black, and 50 (4%) were Hispanic, Asian, American Indian/Alaskan Native, or multiethnic based on self-report. The median age was 21 years, 878 (70%) had more than 12 years of education, and 1,157 (93%) were sexually active. From 4010 completed visits, 1333 (33%) rectal samples were positive for GBS. The unadjusted and adjusted associations of demographic and behavioral characteristics with rectal GBS colonization are shown in Table 1. Age > 21 years, black race, and self-reported marijuana use during the past 4 months were the only independent determinants of rectal GBS colonization. Concurrent vaginal GBS colonization was detected at 938 (70%) of rectal positive visits and was the factor that was most strongly associated with rectal colonization (adjusted odds ratio (OR) = 12.3; 95% confidence interval (CI): 10.4 – 14.6).

Concurrent rectal GBS colonization was the factor associated with the highest risk of vaginal GBS colonization (adjusted OR = 14.3; 95% CI: 11.9 to 17.1, Table 2). Other factors that were independently associated with vaginal colonization over time were concurrent vaginal colonization by yeast or *E. coli*, a Nugent score ≥ 4 , and vaginal sexual intercourse in the past 6 days, (Table 2). Hormonal contraception, use of spermicides or condoms containing nonoxynol-9, and other markers of sexual activity in the past 4 months were not associated with vaginal or rectal GBS colonization including: new, female, or uncircumcised partner; oral sex; sexual intercourse during menses, or vaginal intercourse after anal intercourse. History of or diagnosis within the past 4 months of sexually transmitted infections (*Chlamydia trachomatis*, *Neisseria gonorrhoeae*, *Trichomonas vaginalis*) were not independent predictors of vaginal GBS colonization (data not shown).

In order to determine which factors were associated with vaginal GBS colonization in the absence of rectal GBS colonization, a multivariable GEE model was developed for only the woman-visits where GBS was not isolated from the rectum (Table 3). Sexual intercourse in the past 6 days, concurrent vaginal yeast colonization, and a Nugent score ≥ 4 were associated with an adjusted 30 – 80% increased risk of vaginal GBS colonization while the use of systemic antimicrobials active against GBS during the past 4 months was associated with a 50% decreased risk of GBS (Table 3). This analysis was repeated for those woman-visits where GBS was detected in the rectum. Recent sexual intercourse, concurrent vaginal yeast colonization, and a Nugent score ≥ 4 were again independently associated with vaginal GBS colonization. In addition, concurrent vaginal *E. coli* colonization was associated with a 40% increased risk. The use of systemic antimicrobials active against GBS during the past 4 months did not affect vaginal colonization when the rectum was GBS colonized. Thus, sexual behavior

and the health of the vaginal ecosystem were independently associated with vaginal GBS colonization.

Symptoms were elicited at each visit by interview and evaluated for frequency among women with and without vaginal GBS colonization (Table 4). While there was an unadjusted association between vaginal GBS colonization, pruritus and vaginal burning/pain, the association was not significant after adjustment for urinary tract, yeast, and herpes simplex virus 2 infections during the past 4 months. Vaginal GBS colonization was not associated with abnormal vaginal odor, abnormal vaginal discharge, or abnormal vaginal discharge amount and consistency. Further, high-density vaginal GBS colonization (greater than 10^5 colony forming units per gram of vaginal fluid) was not linked with any vaginal symptom (data not shown). Colonization with vaginal GBS was independently associated with self-reported dysuria during the past 4 months, even after adjusting for clinician-diagnosed urinary tract, yeast, and herpes simplex virus 2 infections during the past 4 months (OR = 1.6; 95% CI: 1.1 – 2.3). Concurrent vaginal colonization with yeast or *E. coli* and Nugent score were not independently associated with dysuria.

COMMENT

The present study which included over 4000 visits on 1248 women confirms previous studies documenting an increased prevalence of colonization by GBS among black women (10,14–17), and has documented that the increased rate of GBS in black women is attributable to an enhanced rate of rectal rather than vaginal colonization. Black women are more likely to harbor GBS in their gastrointestinal tract, which, in turn increases their likelihood of becoming vaginally colonized and increases the risk of neonatal GBS disease (18,19).

Marijuana use has been linked with sexually transmitted infections among adolescents, and has been attributed to more frequent risky sexual behavior among marijuana users (20,21). In our study, marijuana use was associated with a 30% increased risk of rectal, but not vaginal, GBS colonization independent of sexual activity. Recent studies have shown that cannabinoids modulate several functions of the gastrointestinal tract, including gastric secretion, gastric emptying, and intestinal motility (22,23). Thus, marijuana usage may alter the gastrointestinal environment in a way that increases GBS colonization of the rectum.

Rectal GBS was the single strongest determinant of vaginal colonization in this study. This is not surprising since numerous studies have reported a prevalence of rectal GBS colonization that is consistently higher than the prevalence of vaginal GBS colonization (2–5). In addition, a study of 754 pregnant women with sequential cultures in the second and third trimesters, found that persistent GBS carriage was linked with second trimester rectal colonization (4).

Recent sexual intercourse was associated with an increased risk of vaginal GBS colonization in this study, both in the presence and absence of rectal colonization. However, there was no association with increased numbers of sexual partners or having a new sexual partner. This agrees with a recent study which reported an association between GBS incidence and increasing frequency of vaginal intercourse and other measures of sexual activity (24). Sexual activity has been linked with increased vaginal colonization by GBS in several studies (16,17,24–26) while two studies reported no association between sexual activity and GBS colonization (27, 28). Male sexual partners of women with GBS have shown to be colonized by identical strains or serotypes suggesting sexual transmission of GBS may occur (6–8). Sexual activity may transmit GBS from men to women, may alter the microenvironment of the vagina in a manner that increases the persistence of GBS and/or it may enhance the transfer of microorganisms from the perineum or rectum to the vagina.

The use of antibiotics active against GBS was associated with a decreased rate of vaginal GBS colonization only when the rectum was not colonized by GBS. This suggests that antibiotic treatment is ineffective at eradicating rectal colonization by GBS, and that antibiotics would not be effective at eradicating vaginal GBS among women who are rectally colonized. Studies of pregnant women and infants have reported persistence of GBS colonization with the identical serotype after using appropriate antibiotic therapy (29).

An association between symptoms of vaginitis and GBS colonization in the absence of other known causes has been reported in some studies (30,31). In the present study, vaginal colonization by GBS was not independently associated with any genital symptom except dysuria. Our findings are in agreement with a previous study which found no association between GBS colonization and vulvovaginal symptoms (32). However, a limitation of this study was that it relied upon cervical and urethral swabs for the detection of GBS and did not include selective broth enrichment media, which decreased the sensitivity of GBS detection. A limitation of our study is that samples for GBS were obtained at scheduled intervals, and not necessarily when symptoms were present. However, the consistency in the two studies suggests that symptoms associated with GBS are likely due to concurrent yeast, bacterial vaginosis, or urinary tract infection since the association between symptoms and GBS were no longer statistically significant following adjustment for these co-infections.

Colonization with GBS is more common than previously reported with 65% of women being colonized at least once during one year of observation. In this study, black race and marijuana use increased rectal GBS colonization. The fact that rectal GBS colonization was the most significant predictor of vaginal colonization suggests that the vagina becomes colonized with GBS as a result of transfer of the organism from the rectum into the vagina. Results from this study also suggest that sexual activity likely enhances the transit of GBS from the perineum or rectum to the vagina, while altered vaginal flora enhances the capacity of the organism to establish vaginal colonization. Since vaginal GBS colonization and the factors that influence it are dynamic, it is not possible to adequately predict who will be colonized with GBS at delivery. Reducing vaginal colonization by GBS during pregnancy would decrease the risk of pregnancy complications including pregnancy loss, intraamniotic infection, and neonatal sepsis. Antibiotics administered during delivery can interrupt transmission of GBS from colonized women to their neonates, but a vaccine active against GBS may be a more effective long term strategy to prevent GBS disease.

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References

1. Edwards, MS.; Baker, CJ. *Streptococcus agalactiae* (Group B *Streptococcus*). In: Mandell, GL.; Bennett, JE.; Dolin, R., editors. *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*. Philadelphia: Churchill Livingstone; 2000. p. 2156-2167.
2. Badri MS, Zawaneh S, Cruz AC, et al. Rectal colonization with group B streptococcus: relation to vaginal colonization of pregnant women. *J Infect Dis* 1977;135:308–12. [PubMed: 320278]

3. Boyer KM, Gadzala CA, Kelly PD, Burd LI, Gotoff SP. Selective intrapartum chemoprophylaxis of neonatal group B streptococcal early-onset disease. II. Predictive value of prenatal cultures. *J Infect Dis* 1983;148:802–9. [PubMed: 6355317]
4. Dillon HC Jr, Gray E, Pass MA, Gray BM. Anorectal and vaginal carriage of group B streptococci during pregnancy. *J Infect Dis* 1982;145:794–9. [PubMed: 7045248]
5. Maurer M, Thirumoorthi MC, Dajani AS. Group B streptococcal colonization in prepubertal children. *Pediatrics* 1979;64:65–7. [PubMed: 377205]
6. Franciosi RA, Knostman JD, Zimmerman RA. Group B streptococcal neonatal and infant infections. *J Pediatr* 1973;82:707–18. [PubMed: 4572747]
7. Manning SD, Tallman P, Baker CJ, Gillespie B, Marrs CF, Foxman B. Determinants of co-colonization with group B streptococcus among heterosexual college couples. *Epidemiology* 2002;13:533–9. [PubMed: 12192222]
8. Yamamoto T, Nagasawa I, Nojima M, Yoshida K, Kuwabara Y. Sexual transmission and reinfection of group B streptococci between spouses. *J Obstet Gynaecol Res* 1999;25:215–9. [PubMed: 10467796]
9. Manning SD, Neighbors K, Tallman PA, et al. Prevalence of group B streptococcus colonization and potential for transmission by casual contact in healthy young men and women. *Clin Infect Dis* 2004;39:380–8. [PubMed: 15307006]
10. Meyn LA, Moore DM, Hillier SL, Krohn MA. Association of sexual activity with colonization and vaginal acquisition of group B Streptococcus in nonpregnant women. *Am J Epidemiol* 2002;155:949–57. [PubMed: 11994235]
11. Gentry YM, Hillier SL, Eschenbach DA. Evaluation of a rapid enzyme immunoassay test for detection of group B Streptococcus. *Obstet Gynecol* 1991;78:397–401. [PubMed: 1876373]
12. Nugent RP, Krohn MA, Hillier SL. Reliability of diagnosing bacterial vaginosis is improved by a standardized method of gram stain interpretation. *J Clin Microbiol* 1991;29:297–301. [PubMed: 1706728]
13. Hardin, JW.; Hilbe, JM. Generalized Estimating Equations. Boca Raton: Chapman & Hall/CRC; 2003.
14. Campbell JR, Hillier SL, Krohn MA, Ferrieri P, Zaleznik DF, Baker CJ. Group B streptococcal colonization and serotype-specific immunity in pregnant women at delivery. *Obstet Gynecol* 2000;96:498–503. [PubMed: 11004347]
15. Hickman ME, Rench MA, Ferrieri P, Baker CJ. Changing epidemiology of group B streptococcal colonization. *Pediatrics* 1999;104:203–9. [PubMed: 10428995]
16. Newton ER, Piper JM, Shain RN, Perdue ST, Peairs W. Predictors of the vaginal microflora. *Am J Obstet Gynecol* 2001;184:845–53. [PubMed: 11303191]discussion 853–5
17. Regan JA, Klebanoff MA, Nugent RP. The epidemiology of group B streptococcal colonization in pregnancy. Vaginal Infections and Prematurity Study Group. *Obstet Gynecol* 1991;77:604–10. [PubMed: 2002986]
18. Phares CR, Lynfield R, Farley MM, Mohle-Boetani J, et al. Active Bacterial Core surveillance/ Emerging Infections Program Network. Epidemiology of invasive group B streptococcal disease in the United States, 1999–2005. *JAMA* 2008;299:2056–65. [PubMed: 18460666]
19. Zaleznik DF, Rench MA, Hillier S, et al. Invasive disease due to group B Streptococcus in pregnant women and neonates from diverse population groups. *Clin Infect Dis* 2000;30:276–81. [PubMed: 10671328]
20. Crosby R, DiClemente RJ, Wingood GM, et al. Predictors of infection with *Trichomonas vaginalis*: a prospective study of low income African-American adolescent females. *Sex Transm Infect* 2002;78:360–4. [PubMed: 12407241]
21. Liao A, DiClemente RJ, Wingood GM, et al. Associations between biologically confirmed marijuana use and laboratory-confirmed sexually transmitted diseases among African American adolescent females. *Sex Transm Dis* 2002;29:387–90. [PubMed: 12170126]
22. Di Carlo G, Izzo AA. Cannabinoids for gastrointestinal diseases: potential therapeutic applications. *Expert Opin Investig Drugs* 2003;12:39–49.
23. Massa F, Storr M, Lutz B. The endocannabinoid system in the physiology and pathophysiology of the gastrointestinal tract. *J Mol Med* 2005;83:944–54. [PubMed: 16133420]

24. Foxman B, Gillespie BW, Manning SD, Marrs CF. Risk factors for group B streptococcal colonization: potential for different transmission systems by capsular type. *Ann Epidemiol* 2007;17:854–62. [PubMed: 17689259]
25. Baker CJ, Goroff DK, Alpert S, et al. Vaginal colonization with group B streptococcus: a study in college women. *J Infect Dis* 1977;135:392–7. [PubMed: 321702]
26. Newton ER, Butler MC, Shain RN. Sexual behavior and vaginal colonization by group B streptococcus among minority women. *Obstet Gynecol* 1996;88:577–82. [PubMed: 8841222]
27. Shafer MA, Sweet RL, Ohm-Smith MJ, Shalwitz J, Beck A, Schachter J. Microbiology of the lower genital tract in postmenarchal adolescent girls: differences by sexual activity, contraception, and presence of nonspecific vaginitis. *J Pediatr* 1985;107:974–81. [PubMed: 3877803]
28. Wallin J, Forsgren A. Group B streptococci in venereal disease clinic patients. *Br J Vener Dis* 1975;51:401–4. [PubMed: 130184]
29. Gardner SE, Yow MD, Leeds LJ, Thompson PK, Mason EO Jr, Clark DJ. Failure of penicillin to eradicate group B streptococcal colonization in the pregnant woman. A couple study. *Am J Obstet Gynecol* 1979;135:1062–5. [PubMed: 391044]
30. Honig E, Mouton JW, van der Meijden WI. Can group B streptococci cause symptomatic vaginitis? *Infect Dis Obstet Gynecol* 1999;7:206–9. [PubMed: 10449271]
31. Maniatis AN, Palermos J, Kantzanou M, Maniatis NA, Christodoulou C, Legakis NJ. Streptococcus agalactiae: a vaginal pathogen? *J Med Microbiol* 1996;44:199–202. [PubMed: 8636937]
32. Shaw C, Mason M, Scoular A. Group B streptococcus carriage and vulvovaginal symptoms: causal or casual? A case-control study in a GUM clinic population. *Sex Transm Infect* 2003;79:246–8. [PubMed: 12794214]

TABLE 1

Association of Demographic and Behavioral Characteristics with Rectal Group B Streptococcus (GBS) Colonization

Variable	# Total Visits* (n=4010)	# (%) Rectal GBS Positive Visits (n=1333)	OR	Unadjusted 95% CI	P-value	OR	Adjusted [†] 95% CI	P-value
Race								
White	2496	724 (29%)	1.0	Referent		1.0	Referent	
Black	1361	568 (42%)	1.8	1.5 – 2.1	<0.001	1.5	1.2 – 1.8	<0.001
Other	153	41 (27%)	0.9	0.6 – 1.4	0.6	0.9	0.5 – 1.4	0.6
Age								
18–21 years	2262	711 (31%)	1.0	Referent		1.0	Referent	
22–30 years	1748	622 (36%)	1.2	1.02 – 1.5	0.03	1.2	1.01 – 1.4	0.04
Education								
13 or more years	2875	899 (31%)	1.0	Referent		1.0	Referent	
12 or fewer years	1128	431 (38%)	1.3	1.1 – 1.6	0.003	1.3	1.1 – 1.5	0.003
Marijuana use past 4 months								
No	2860	885 (31%)	1.0	Referent		1.0	Referent	
Yes	1146	447 (39%)	1.3	1.1 – 1.5	<0.001	1.3	1.1 – 1.5	0.003
Use of antimicrobials active against GBS past 4 months								
No	3521	1198 (34%)	1.0	Referent		1.0	Referent	
Yes	489	135 (28%)	0.8	0.7 – 0.98	0.03	0.8	0.7 – 0.98	0.03
Concurrent Vaginal Group B Streptococcus Colonization								
Absent	2725	394 (14%)	1.0	Referent		1.0	Referent	
Present	1284	938 (73%)	12.5	10.5 – 14.8	<0.001	12.3	10.4 – 14.6	<0.001
Any sexual intercourse past 4 months								
No	608	152 (25%)	1.0	Referent		1.0	Referent	
Yes	3402	1181 (35%)	1.4	1.1 – 1.8	0.002	1.4	1.1 – 1.8	0.002
No. of sexual partners past 4 months								
None	616	154 (25%)	1.0	Referent		1.0	Referent	
One	2787	951 (34%)	1.4	1.1 – 1.7	0.003	1.4	1.1 – 1.7	0.003
Two or more	604	227 (38%)	1.6	1.2 – 2.1	0.001	1.6	1.2 – 2.1	0.001
Frequency of sexual intercourse past 4 months								
None	612	154 (25%)	1.0	Referent		1.0	Referent	
Less than 3 times/week	2577	883 (34%)	1.4	1.1 – 1.7	0.004	1.4	1.1 – 1.7	0.004
Three or more times/week	819	296 (36%)	1.5	1.2 – 2.0	0.001	1.5	1.2 – 2.0	0.001
Time from last sexual intercourse to GBS culture								
Sexual abstinence	609	152 (25%)	1.0	Referent		1.0	Referent	
7 or more days	1654	521 (32%)	1.3	1.0 – 1.6	0.051	1.3	1.0 – 1.6	0.051
Within 6 days	1746	658 (38%)	1.6	1.3 – 2.0	<0.001	1.6	1.3 – 2.0	<0.001

OR, odds ratio; CI, confidence interval

* Some numbers do not add up to total (N = 4010 visits) because of missing values

[†] Adjusted for race, age, marijuana use, and concurrent vaginal GBS colonization.

TABLE 2

Association of Demographic, Behavioral Characteristics, and Vaginal Microflora with Vaginal Group B Streptococcus (GBS) Colonization

Variable	# Total Visits [*] (n=4010)	# (%) Vaginal GBS Positive Visits(n=1284)	OR	Unadjusted 95% CI	P-value	OR	Adjusted [†] 95% CI	P-value
Race								
White	2496	705 (28%)	1.0	Referent				
Black	1361	538 (40%)	1.7	1.4 – 2.0	<0.001			
Other	153	41 (27%)	0.9	0.6 – 1.5	0.7			
Age								
18–21 years	1785	545 (31%)	1.0	Referent				
22–30 years	1569	528 (34%)	1.1	0.9 – 1.3	0.3			
Education								
13 or more years	2875	873 (30%)	1.0	Referent				
12 or fewer years	1128	409 (36%)	1.3	1.1 – 1.6	0.005			
Marijuana use past 4 months								
No	2860	867 (30%)	1.0	Referent				
Yes	1146	414 (36%)	1.3	1.1 – 1.5	0.001			
Use of antimicrobials active against GBS past 4 months								
No	3521	1156 (33%)	1.0	Referent				
Yes	489	128 (26%)	0.8	0.7 – 0.98	0.03			
Concurrent Rectal Group B Streptococcus Colonization								
Absent	2677	346 (13%)	1.0	Referent		1.0	Referent	
Present	1333	938 (70%)	14.1	11.9 – 16.8	<0.001	14.3	11.9 – 17.1	<0.001
Concurrent Vaginal Yeast Colonization								
Absent	2795	827 (30%)	1.0	Referent		1.0	Referent	
Present	1213	457 (38%)	1.4	1.2 – 1.6	<0.001	1.3	1.1 – 1.6	0.001
Concurrent Vaginal E. coli Colonization								
Absent	3017	927 (31%)	1.0	Referent		1.0	Referent	
Present	992	357 (36%)	0.8	0.7 – 0.96	0.01	1.2	1.01 – 1.5	0.04
Concurrent Vaginal Nugent Score								
Normal (0–3)	2115	547 (26%)	1.0	Referent		1.0	Referent	
Intermediate (4–6)	772	324 (42%)	2.0	1.7 – 2.3	<0.001	2.1	1.7 – 2.6	<0.001
Bacterial Vaginosis (7–10)	1099	404 (37%)	1.5	1.3 – 1.8	<0.001	1.7	1.4 – 2.0	<0.001
Any sexual intercourse past 4 months								
No	608	149 (25%)	1.0	Referent				
Yes	3402	1135 (33%)	1.5	1.2 – 1.9	0.001			
No. of sexual partners past 4 months								
None	616	152 (25%)	1.0	Referent				
One	2787	912 (33%)	1.4	1.1 – 1.8	0.002			
Two or more	604	219 (36%)	1.6	1.2 – 2.1	0.001			
Frequency of sexual intercourse past 4 months								
None	612	150 (25%)	1.0	Referent				
Less than 3 times/week	2577	837 (32%)	1.4	1.1 – 1.8	0.003			
Three or more times/week	819	297 (36%)	1.7	1.3 – 2.2	<0.001			
Time from last sexual intercourse to GBS culture								
Sexual abstinence	609	149 (24%)	1.0	Referent		1.0	Referent	
7 or more days	1654	464 (28%)	1.2	0.9 – 1.5	0.1	1.0	0.7 – 1.2	0.7
Within 6 days	1746	670 (38%)	1.8	1.4 – 2.3	<0.001	1.4	1.1 – 1.8	0.008

OR, odds ratio; CI, confidence interval

^{*} Some numbers do not add up to total (N = 4010 visits) because of missing values.[†] Adjusted for concurrent rectal GBS colonization, concurrent vaginal yeast colonization, concurrent vaginal E coli colonization, concurrent vaginal Nugent score, and time since last sexual intercourse.

TABLE 3

Independent Risk Factors for Vaginal Group B Streptococcus (GBS) Colonization – Multivariable Generalized Estimating Equations Model

Factor	Adjusted* OR	95% CI	P-value
1) In Absence of Rectal Group B Streptococcus (GBS) Colonization:			
Use of Antimicrobials Active Against GBS Past 4 months	0.5	0.3 – 0.8	0.001
Sexual intercourse within 6 days of GBS culture	1.6	1.2 – 2.0	<0.001
Concurrent Vaginal Yeast Colonization	1.3	1.02 – 1.7	0.03
Concurrent Vaginal Nugent Score			
Normal (0 – 3)	1.0	Referent	
Intermediate (4 – 6)	1.8	1.4 – 2.4	<0.001
Bacterial Vaginosis (7 – 10)	1.5	1.1 – 2.0	0.005
2) In Presence of Rectal Group B Streptococcus (GBS) Colonization:			
Use of Antimicrobials Active Against GBS Past 4 months	1.3	0.9 – 2.0	0.2
Sexual intercourse within 6 days of GBS culture	1.4	1.1 – 1.8	0.008
Concurrent Vaginal Yeast Colonization	1.3	1.02 – 1.8	0.04
Concurrent Vaginal E. coli Colonization	1.4	1.03 – 1.8	0.03
Concurrent Vaginal Nugent Score			
Normal (0 – 3)	1.0	Referent	
Intermediate (4 – 6)	2.8	2.0 – 3.9	<0.001
Bacterial Vaginosis (7 – 10)	1.9	1.4 – 2.5	<0.001

OR, odds ratio; CI, confidence interval

* Odds ratios adjusted for 1) antimicrobial use, time since last sexual intercourse, concurrent vaginal yeast colonization, and concurrent vaginal Nugent score and 2) antimicrobial use, time since last sexual intercourse, concurrent vaginal yeast colonization, concurrent vaginal E. coli colonization, and concurrent vaginal Nugent score.

TABLE 4

Association of Self-reported Vaginal and Urethral Symptoms Experienced in the Past 4 Months with Vaginal Group B Streptococcus (GBS) Colonization

Symptom	# Total Visits*	# (%) Vaginal GBS Positive Visits	OR	95% CI	P-value
Dysuria					
No	3741	1176 (31%)	1.0	Referent	
Yes	267	108 (40%)	1.4	1.1 – 1.9	0.008
Pruritus					
No	3289	1020 (31%)	1.0	Referent	
Yes	720	264 (37%)	1.2	1.04 – 1.5	0.02
Vaginal Burning or Pain					
No	3679	1153 (31%)	1.0	Referent	
Yes	330	131 (40%)	1.4	1.1 – 1.8	0.005
Abnormal Vaginal Odor					
No	3424	1071 (31%)	1.0	Referent	
Yes	582	211 (36%)	1.1	0.9 – 1.3	0.5
Abnormal Vaginal Discharge					
No	3141	983 (31%)	1.0	Referent	
Yes	866	300 (35%)	1.0	0.8 – 1.2	0.9

* Some numbers do not add up to total (N = 4010 visits) because of missing values.