

Published in final edited form as:

J Nutr. 2007 September ; 137(9): 2128–2133.

Dietary Intake of Selected Nutrients Affects Bacterial Vaginosis in Women^{1,2,3}

Yasmin H. Neggers^{4,*}, Tonja R. Nansel⁷, William W. Andrews⁵, Jane R. Schwebke⁶, Kai-fun Yu⁷, Robert L. Goldenberg⁸, and Mark A. Klebanoff⁷

⁴ Department of Human Nutrition, University of Alabama, Tuscaloosa, AL 35487 ⁵ Department of Obstetrics and Gynecology, University of Alabama, Birmingham, Alabama 35233 ⁶ Department of Medicine, University of Alabama, Birmingham, Alabama 35233 ⁷ Division of Epidemiology, Statistics and Prevention Research, National Institute of Child Health and Human Development, NIH, Department of Health and Human Services, Bethesda, MD 20892 and ⁸ Department of Obstetrics and Gynecology, Drexel College of Medicine, Philadelphia, PA 19102

Abstract

Bacterial vaginosis (BV), a condition of altered vaginal flora, is associated with various adverse reproductive health outcomes. We evaluated the association between diet and the presence of BV in a subset of 1521 women (86% African-American) from a larger study of vaginal flora. Participants completed the Block Food Questionnaire and clinical assessments and self-report measures of sexual and hygiene behavior. A total of 42% of the women were classified as having BV (Nugent score ≥ 7). Severe BV (Nugent score ≥ 9 and vaginal pH ≥ 5) was present in 14.9% of the women. BV was associated [adjusted OR (AOR)] with increased dietary fat (1.5, 1.1–2.4) after adjusting for other energy nutrients and behavioral and demographic covariates. Severe BV was associated with total fat (2.3, 1.3–4.3), saturated fat (2.1, 1.2–3.9), and monounsaturated fat (2.2, 1.2–4.1). Energy intake was only marginally associated ($P = 0.05$) with BV (1.4, 1.0–1.8). There were significant inverse associations between severe BV and intakes of folate (0.4, 0.2–0.8), vitamin E (0.4, 0.2–0.8), and calcium (0.4, 0.3–0.7). We conclude that increased dietary fat intake is associated with increased risk of BV and severe BV, whereas increased intake of folate, vitamin A, and calcium may decrease the risk of severe BV.

Introduction

Bacterial vaginosis (BV)⁹ is a common vaginal infection in women of reproductive age. It is a condition of altered vaginal flora in which the quantity of hydrogen peroxide producing lactobacilli decreases and the concentration of anaerobic bacteria increases (1,2). BV is associated with spontaneous preterm birth and other adverse pregnancy outcomes (3,4) as well as acquisition of other genital tract infections and HIV-1 (2). Although factors such as race (the rate of BV in African-American women is higher compared with other ethnic groups), smoking,

¹Supported by contract N01-HD-8-3293 and by the Intramural Research Program of the National Institute of Child Health and Human Development, NIH.

²Author disclosures: Y. H. Neggers, T. R. Nansel, W. W. Andrews, J. R. Schwebke, K. Yu, R. L. Goldenberg, and M. A. Klebanoff, no conflicts of interest.

³Supplemental Table 1 is available with the online posting of this paper at jn.nutrition.org.

*To whom correspondence should be addressed. E-mail: yneggers@ches.ua.edu.

⁹Abbreviations used: AI, Adequate intake; AOR, adjusted OR; BV, bacterial vaginosis; RDA, recommended daily allowance.

chronic stress, vaginal douching, and contraceptive pill use are associated with BV, the etiology of BV is not well understood (4).

Deficiencies in local immunity may predispose women to BV. Women who are chronically stressed have a higher prevalence of BV (4). BV was found to be more common among women with inflammatory response gene polymorphisms that cause reduced immune function (5). Protein-energy malnutrition and intake of several nutrients such as vitamins A and C, iron, and zinc have been reported to affect the immune system, particularly local immunity (6). Even marginal deficiencies of micronutrients, which in Western countries are more common than gross deficiencies, are associated with depressed immune response (7). Furthermore, iron deficiency has been shown to affect the host response against bacterial colonization (8). It is therefore plausible that certain nutrient intakes may be associated with BV.

Very few studies have evaluated the role of nutritional status as a risk factor for BV in pregnant or nonpregnant women (8–10). To our knowledge, no published studies have examined the relationship between BV and nutritional status as measured by energy, macro, and micronutrient intake. This study was conducted to assess the relationship between dietary intake (energy and nutrients) and presence of BV in a sample of non-pregnant women.

Material and Methods

Subjects and procedures

The subjects of this study were drawn from women who participated in the Longitudinal Study of Vaginal Flora, which has been previously described (4). Nonpregnant women 15–45 y old were enrolled from August 1999 to February 2002 when presenting for routine health care at 1 of 12 clinics in the Birmingham, Alabama area. Women were assessed at baseline and quarterly for a year for a total of up to 5 visits. Exclusion criteria included major medical or gynecological conditions, receipt of chronic antibiotics, planning to move from the area in the next 12 mo, nonfluency in English, and inability to provide informed consent (4). Written informed consent was obtained from all participants. The protocol was approved by the institutional Review Boards of the Jefferson County Department of Health, the University of Alabama at Birmingham, and the National Institute of Child Health and Human Development.

At each visit, participants completed a questionnaire and underwent a standardized pelvic examination. Vaginal flora was evaluated by Gram stain according to Nugent criteria (11). BV was defined as a Nugent score of ≥ 7 and severe BV was defined as Nugent score ≥ 9 and vaginal pH ≥ 5 . Because of the extensive nature of the first visit assessment ($n = 3620$), the Block 98 FFQ (12) was administered at the next study visit 3 mo following the first visit. This validated, 100-item questionnaire, which measures long-term nutrient intake, was administered by trained interviewers unaware of participants' BV status. The interviewers received training on administration of the interview and were provided an instruction booklet published by the Block Dietary Data Systems (12). Due to the time required to complete the questionnaire and a determination that the accrued sample provided adequate power, the measure was discontinued after being administered to the first 2005 consecutive study participants who completed visit 2 (from a total of 2751 women completing the visit). Demographic and behavioral factors were not significantly different for all the subjects and those who provided dietary information. Three months later, participants completed visit 3 ($n = 2458$) during which vaginal flora was again evaluated.

Dietary analysis of the questionnaires was conducted by the Block Dietary Data Systems (12), which analyzes for energy, energy nutrients, and various vitamin and mineral intakes. Questionnaires of 178 women contained severe errors (e.g. too many or too few consumed or all the serving sizes were identical, etc.). As recommended by Block et al. (12), nutrient values

of these subjects were not used in further analyses. Missing data on demographic, laboratory, or health behavior variables reduced the sample for multivariate analyses to 1520.

Statistical analyses

Both cross-sectional and prospective analyses were conducted. Descriptive statistics were used to assess the overall energy and nutrient intake of participants. The relationship of potential covariates to BV and severe BV was assessed using logistic regression; those variables demonstrating significant relations ($P < 0.05$) were entered as covariates in later adjusted models. Energy and nutrient variables were divided into quartiles and analyzed as discrete variables. Logistic regression was used to assess the relation of each energy and nutrient variable to BV and severe BV. The measure of association reported in this study is adjusted OR (AOR). Both unadjusted and adjusted models were analyzed. Multivariate models were adjusted for those demographic and health practices variables that were significantly associated with BV classifications (**Supplemental Table 1**). In adjusted models, one model was fitted for total energy intake, adjusting for age, race, education, marital status, BMI, douching, oral contraceptive use, and alcohol use. Separate models were fitted for each macronutrient (protein, carbohydrate, and fat), including the same behavioral and demographic covariates as mentioned above. For each energy nutrient, total energy intake was adjusted by including all 3 energy nutrients simultaneously in the model (Table 2), an accepted method of energy adjustment (13). Finally, individual models were fitted for each type of fat (saturated, polyunsaturated, monounsaturated, and cholesterol) and micronutrients, adjusting for protein, carbohydrate, and behavioral and demographic covariates. These covariates included, age, race, BMI (BV only), income, education (BV only), douching contraceptive pill use, and alcohol intake (BV only) (Tables 2 and 3).

In the prospective analyses, women were classified into 1 of 4 groups based on their BV status at both visits 2 and 3 using a Nugent score cutoff of < 7 as BV negative and ≥ 7 as BV positive. In BV negative women at visit 2, those BV negative at visit 3 were classified as “maintaining BV negative” and those BV positive at visit 3 as “incident BV.” In those BV positive at visit 2, those BV positive at visit 3 were classified as “persistent BV” and those BV negative at visit 3 as “remitting BV.” We used logistic models (with the same covariates as above) to assess the relationship of the various dietary components with maintaining BV negative vs. incident BV and with persistent BV vs. remitting BV.

Analyses were conducted by SAS statistical software (version 9, SAS Institute). Values in the text are means \pm SD unless otherwise noted.

Results

This sample consisted predominantly of African-American (86%), unmarried women (71.6%). Their age was 25.0 ± 6.7 y and they had completed 12.1 ± 3.4 y of education. Their BMI was high (29.6 ± 8.5) and 45% were classified as obese. The prevalence of BV was 41.8% of severe BV, 14.9%. Both BV and severe BV were significantly more prevalent in African-Americans than Caucasians (45.2 vs. 27.1%, and 16.4 vs. 3.5%, respectively). We conducted analyses stratified by race; associations between macro- and micronutrients, BV, and severe BV remained essentially the same as in the combined sample (data not shown). Race was adjusted for in all analyses evaluating the relationship between both classifications of BV and nutrients.

There was a wide variation in the energy intake in this sample and the mean (603.2 ± 220.7 kJ) exceeded the recommended daily allowance (RDA) (14) for adult women (Table 1). Also, the energy, fat, and carbohydrate intakes of the women were considerably higher than those reported in NHANES (1999–2000) for women of comparable age (15). The mean intakes of total energy, fat, and protein were higher ($P < 0.01$) in African-American women than

Caucasians (604.4 ± 217.2 vs. 530.9 ± 166.0 kJ, 110.2 ± 46.8 vs. 87.5 ± 36.7 g and 78.7 ± 31.3 vs. 67.6 ± 23.0 g, respectively). A large percentage of subjects had intakes < two-thirds RDA/adequate intake (AI) (14) for calcium, potassium, iron, vitamin E, and folate. Fiber intake of all the subjects was < two-thirds AI.

Some demographic and behavioral characteristics of the women were associated with BV and severe BV (Supplemental Table 1). Age, race, income, education, BMI, douching, current birth control pill use, and alcohol use were significantly associated with BV. Severe BV was significantly associated with age, race, education, douching, and current use of birth control pills.

In unadjusted analyses, energy (OR = 1.6, 95% CI = 1.2–2.1), fat (OR = 1.6, 95% CI = 1.2–2.0), carbohydrate (OR = 1.5, 95% CI = 1.1–1.9), and protein (OR = 1.5, 95% CI = 1.1–1.9) intakes (highest vs. lowest quartile) were significantly associated with BV. Severe BV was significantly associated only with fat intake (OR = 1.5, 95% CI = 1.1–2.1). There was no significant association between any micronutrient intake and BV or severe BV.

After adjusting for demographic and behavioral covariates, total energy intake was only marginally ($P = 0.05$) associated with BV (AOR = 1.4, 95% CI = 1.0–1.9) (Table 2). Among the macronutrients, only total fat intake was significantly associated with the BV (AOR = 1.5, 95% CI = 1.1–2.4). Total fat (AOR = 2.3, 95% CI = 1.3–4.3), saturated fat (AOR = 2.3, 95% CI = 1.2–3.9), and monounsaturated fat (AOR = 2.3, 95% CI = 1.2–4.1) intakes were significantly associated with severe BV. Protein intake was significantly inversely associated with severe BV (AOR = 0.5, 95% CI = 0.3–0.9).

There were significant inverse associations between severe BV and the intakes of the micronutrients folate (AOR = 0.4, 95% CI = 0.2–0.8), vitamin E (AOR = 0.41, 95% CI = 0.22–0.79), and calcium intake (AOR = 0.4, 95% CI = 0.3–0.7) after adjusting for energy nutrients and other covariates (Table 3). Neither BV nor severe BV was associated with the intake of any other micronutrients (data not shown).

A total of 1319 of the participants completed visit 3; 727 (55.1%) were classified as maintaining negative BV, 166 (12.6%) as incident BV, 225 (17.6%) as persistent BV, and 201 (15.2%) as remitting BV. In unadjusted analyses, a marginally significant association ($P = 0.05$) between incident BV and total energy (OR = 1.6, 95% CI = 1.0–2.6) and fat intake (OR = 1.6, 95% CI = 1.0–2.6) was observed. After adjusting for demographic and behavioral covariates, total energy intake remained marginally significant (OR = 1.7, 95% CI = 1.0–2.9) ($P = 0.05$). When persistent BV was compared with remitting BV, none of the nutrients (macro- or micronutrients) or their components (specific types of fats) were significantly associated.

Discussion

To our knowledge, this is the first study to evaluate the relationship between BV and total nutritional intake. After adjusting for demographic and behavioral covariates, the risk of BV was 50% higher in women who had high energy intake compared with those with low energy intake, but this association was only marginally significant ($P = 0.05$). However, energy-adjusted total fat was a significant predictor of BV. The risk of severe BV was more than twice as high in women with total fat, saturated fat, and monounsaturated fat intakes in the highest quartile compared with those in the lowest quartile. There was significantly lower risk of severe BV in women with high intakes of folate, vitamin E, and calcium.

A few studies have evaluated the relationship between BV and specific micronutrient intakes. Westney et al. (9) evaluated the association between dietary intakes of protein and vitamins C and A, and the prevalence of genital tract infections in a cohort of pregnant, low-income

African- American women. Energy and fat intake was not measured. Similar to our findings, there was no significant association between prevalence of genital tract infections and the nutrients measured in that study. In another case-control study, no association between BV and routine biochemical indices of iron status was observed in well-nourished pregnant women (8). However, subclinical iron deficiency (measured by soluble transferrin receptor levels) was a significant predictor of BV. Because the mean nutrient intakes in both our study and that of Westney et al. (9) were more than adequate, it is possible that an association with BV might have been obscured due to a lack of substantial variation in the micronutrient intakes.

In 2 studies conducted in women with overall poor nutritional status, there was no significant association between BV and nutritional status. In a study of vitamin A-deficient, nonpregnant women in the Central African Republic, BV was not significantly associated with blood levels of vitamin A (10). In a study of low socio-economic status, nonpregnant Indian women (5), the incidence of BV was not significantly different between well-nourished (BMI ≥ 18.5) and undernourished women (BMI < 18.5) but was higher (72%) in severely malnourished women (BMI < 16). Results of these studies seem to indicate that in both well-nourished and malnourished women, nutritional status as measured by BMI or specific micronutrient intakes is not associated with BV.

In our study, total fat intake adjusted for energy and other covariates was a significant predictor of both BV and severe BV. Saturated fat intake was a significant predictor of severe BV. The marginally significant association between energy intake and BV could be due to confounding by a high fat intake (39% of energy from fat) in this cohort. Because there was no association between BV and either carbohydrates or protein intake, this indicates that a high fat intake is a predictor of BV independent of energy intake. To our knowledge, no other studies have evaluated the relationship between micronutrients and risk of BV. Our results indicate that high intakes of folate, vitamin E, and calcium are associated with decreased risk of severe BV.

Vitamin E is a strong antioxidant and studies have indicated an association between vitamin E status and immune response (16–18). Studies in elderly patients have reported reduced infection rates and respiratory problems with vitamin E supplementation (16,17). Folate intake is linked with decreased risk of certain cancers, but human and animal studies of its effect on immune response have reported contradictory results (19,20). However, it is plausible that intakes of folate and vitamin E may be improve immune functioning, thereby decreasing the risk of severe BV (Nugent score ≥ 9 ; pH ≥ 5). The role of calcium intake in decreasing the risk of severe BV is not clear. To our knowledge, calcium intake has not been associated with infections or immune response in humans.

The mechanism for an association between fat intake and BV is unclear. High fat intake, particularly saturated fat, may increase vaginal pH, thereby increasing the risk of BV. Some of the factors associated with high vaginal pH are age, race, frequency of douching, and menopausal status (2,21,22). This study consisted of predominantly African-American women who had both a high fat intake (106 ± 48 g) and a high vaginal pH (5.1 ± 0.80). There was a small but significant association between dietary fat intake and vaginal pH ($r = 0.10$; $P < 0.001$). Thus, it is possible that a high fat intake may alter the vaginal microflora, which may increase the vaginal pH and increase the risk of BV.

Another plausible mechanism for the relationship between high fat intake and BV may be related to the role of dietary fat as a stimulator of immunological functions of intestinal mucosa. Fat intake is a known modulator of the immune system (23). The mucosal immune system, which functions independently of the systemic immune system, is regulated by gut-associated lymphoid tissue (24). Studies have indicated that both the amount and type of dietary fat intake affect lymphoid cell subsets, proliferation response to mitogens, and cytokine production

(25). Thus, it is plausible that a high dietary fat intake of subjects in this study may affect the mucosal immune system and thereby increase the risk of bacterial infections related to BV.

Strengths of our study include a large sample across a broad age range, recruitment at routine health care visits, and use of a validated instrument measuring long-term dietary intake. Rates of BV were similar to published national estimates. However, the sample consisted primarily of lower socioeconomic status African-American women drawn from 1 geographic area. This may have caused limited variance in dietary intake; for example, dietary fiber was uniformly low, suggesting limited consumption of health-protective foods such as fruits and vegetables. As such, generalization of findings should be approached with caution.

This study suggests further research to clarify the mechanism of relationship between dietary fat intake and increased risk of BV and confirm the observed inverse association between folate, vitamin E, and calcium intakes and severe BV. In addition, studies addressing overall dietary patterns in addition to specific nutrient intake may be useful. Although further research is needed, findings from this study suggest that dietary practices may be associated with maintenance of normal vaginal flora.

Literature Cited

1. Nelson DB, Macones G. Bacterial vaginosis in pregnancy: current findings and future directions. *Epidemiol Rev* 2002;24:102–08. [PubMed: 12762086]
2. Morris MC, Rogers PA, Kinghorn GR. Is bacterial vaginosis a sexually transmitted disease? *Sex Transm Infect* 2001;77:63–8. [PubMed: 11158694]
3. Holzman C, Leventhal JM, Qiu H, Jones NM, Wang J. the BV Study Group. Factors linked to bacterial vaginosis in nonpregnant women. *Am J Public Health* 2001;91:1664–70. [PubMed: 11574333]
4. Nansel TR, Riggs MA, Yu K, Andrews W, Schwebke JR, Klebanoff MA. The association of psychological stress and bacterial vaginosis in a longitudinal cohort. *Am J Obstet Gynecol* 2006;194:381–86. [PubMed: 16458633]
5. Yasodhara P, Raghunath M, Sreeramulu D, Venu L, Hemlatha R, Krishna TP. Local immunity in Indian women with bacterial vaginosis. *J Reprod Immunol* 2006;70:133–41. [PubMed: 16406057]
6. Bhaskaram P. Immunology of mild micronutrient deficiencies. *Br J Nutr* 2001;85(Suppl 2):S75–80. [PubMed: 11509093]
7. Bendich A. Micronutrients in women's health and immune function. *Nutrition* 2001;17:858–67. [PubMed: 11684393]
8. Verstraelen H, Delanhe J, Roelens K, Blot S, Claeys G, Temmerman M. Subclinical iron deficiency is a strong predictor of bacterial vaginosis in early pregnancy. *BMC Infect Dis* 2005;5:55–65. [PubMed: 16000177]
9. Westney OE, Westney LS, Johnson AA, Knight EM, Oyemade UJ, Cole OJ, Laryea H, Spurlock B, Manning M. Nutrition, genital tract infections, hematologic values, and premature rupture of membranes among African American women. *J Nutr* 1994;124:S987–93.
10. Belec L, Mbopi-Keou F-X, Roubache J-F, Mayaud P, Paul JL, Gresnquet G. Vitamin A deficiency and genital tract infections in women living in Central Africa. *J Acquir Immune Defic Syndr* 2002;29:203–09. [PubMed: 11832693]
11. 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]
12. Block Dietary Data System. Berkley (CA): Nutritionquest;
13. Howe GR, Miller AB, Jain M. Total energy intake: implications for epidemiologic analyses. *Am J Epidemiol* 1986;124:157–59. [PubMed: 3013002]
14. Institute of Medicine, Food and Nutrition Board. Dietary reference intakes. Washington: National Academy Press; 2005.
15. Morbidity and Mortality Weekly Report. Trends in intake of energy and macronutrients: United States, 1971–2000. 2004;53:80–86.

16. Meydani SN, Leka LS, Fine BC, Dallal GE, Keusch GT, Singh MF, Hamer DH. Vitamin E and respiratory tract infections in elderly nursing home residents: a randomized controlled trial. *JAMA* 2004;292:828–36. [PubMed: 15315997]
17. Bunout D, Barrera G, Hircg S, Gattas V, de la Maza MP, Haschke F, Steenhout P, Klassen P, Hager C. Effects of a nutritional supplement on the immune response and cytokine production in free-living Chilean elderly. *JPEN J Parenter Enteral Nutr* 2004;28:348–54. [PubMed: 15449576]
18. Nathens AB, Neff MJ, Jurkovich GJ, Klotz P, Farver K, Ruzinski JT, Radella F, Garcia I, Maier RV. Randomized prospective trial of anti-oxidant supplementation in critically ill surgical patients. *Ann Surg* 2002;236:814–22. [PubMed: 12454520]
19. Larsson SC, Giovannucci E. Folate and risk of breast cancer: a meta-analysis. *J Natl Cancer Inst* 2007;99:64–76. [PubMed: 17202114]
20. Kim YI, Hayek M, Mason JB, Meydani SN. Severe folate deficiency impairs natural killer cell-mediated cytotoxicity in rats. *J Nutr* 2002;132:1361–7. [PubMed: 12042459]
21. Fiscella K, Klebanoff MA. Are racial differences in vaginal pH explained by vaginal flora? *Am J Obstet Gynecol* 2004;191:747–50. [PubMed: 15467534]
22. Garcia-Closas M, Herrero R, Bratti C, Hildersheim A, Sherman ME, Morera LA, Schffiman M. Epidemiologic determinants of vaginal pH. *Am J Obstet Gynecol* 1999;180:1060–6. [PubMed: 10329856]
23. Kelley DS. Modulation of human immune and inflammatory responses by dietary fatty acids. *Nutrition* 2001;17:669–73. [PubMed: 11448594]
24. Miura S, Tsuzuki Y, Hokari R, Ishii H. Modulation of intestinal immune system by dietary fat intake: relevance to Crohn's disease. *J Gastroenterol Hepatol* 1998;13:1183–90. [PubMed: 9918423]
25. Meksawan K, Venkatraman JT, Awad AB, Pendergast DR. Effect of dietary fat intake and exercise on inflammatory mediators of the immune system in sedentary men and women. *J Am Coll Nutr* 2004;23:331–40. [PubMed: 15310737]

TABLE 1

Daily energy and nutrient intakes of women ($n = 1827$)

Nutrient	Means \pm SD	Median	25th Percentile	75th Percentile	Percentage < two-thirds RDA or AI
Energy, <i>kJ</i>	603.2 \pm 220.7	548.6	459.4	718.5	11.8
Protein, <i>g</i>	78.5 \pm 31.8	72.6	56.6	94.4	1.9
Fat, <i>g</i>	109.6 \pm 47.8	100	75.4	134.8	—
Carbohydrates, <i>g</i>	307.6 \pm 113.6	286.2	229.8	364.2	0.16
Dietary fiber, <i>g</i>	14.7 \pm 6.0	13.8	10.3	18	100.0
Saturated fat, <i>g</i>	34.2 \pm 14.9	31.3	23.5	42.7	—
Monounsaturated fat, <i>g</i>	40.3 \pm 18.2	36.7	27.6	50.0	—
Polyunsaturated fat, <i>g</i>	27.1 \pm 13.7	24.2	17.5	33.7	—
Cholesterol, <i>mg</i>	323.3 \pm 176.4	286.3	194.1	412.3	—
Calcium, <i>mg</i>	830.2 \pm 400.3	748.6	535.2	1041.1	41.0
Sodium, <i>mg</i>	3,263.3 \pm 1,358	3011.4	2289.7	3978.7	0.0
Potassium, <i>mg</i>	2,830.7 \pm 1,010	2659.1	2129.1	3384.9	68.3
Zinc, <i>mg</i>	10.4 \pm 4.4	9.4	7.3	12.5	8.9
Iron, <i>mg</i>	14.6 \pm 5.9	13.6	10.6	17.6	37.6
Vitamin E, <i>α-TE</i>	10.7 \pm 4.8	9.7	7.5	13.1	53.4
Vitamin A, <i>retinol equivalent</i>	954.8 \pm 613	801.8	570.8	1173.1	14.3
β -Carotene, μ g	2,189.5 \pm 1,749	1682.8	1049.0	2739.5	—
Vitamin D, μ g	3.6 \pm 3.0	2.0	1.0	2.2	13.1
Lycopene, μ g	6,204.2 \pm 6,0992	4287.8	2489.6	7060.3	—
Vitamin C, <i>mg</i>	176.5 \pm 107.5	152.9	92.6	243.4	8.00
Folate, μ g	382.8 \pm 159.2	353.6	273.2	463.0	23.5

TABLE 2
 Association of energy, lipids, and carbohydrate and protein intakes with BV and severe BV in women determined by AOR

	BV (Nugent ≥ 7), $n = 1521$			Severe BV (Nugent ≥ 9 and pH > 5), $n = 1521$		
	AOR	95% CI	P^2	AOR	95% CI	P^1
Total energy ²						
Fourth quartile	1.4	1.0–1.9	0.05	1.5	0.97–2.3	0.22
Third quartile	0.971	0.71–1.3		1.0	0.6–1.5	
Second quartile	1.0	0.7–1.3		1.1	0.8–1.7	
First quartile	(referent)	(referent)		(referent)	(referent)	
Macronutrients ³						
Total fat	1.5	1.1–2.4	0.04	2.3	1.3–4.3	0.04
Fourth quartile	1.4	1.0–2.0		1.7	1.0–2.8	
Third quartile	1.2	0.9–1.6		1.0	0.6–1.6	
Second quartile	(referent)	(referent)		(referent)	(referent)	
First quartile						
Protein						
Fourth quartile	0.8	0.5–1.3	0.43	0.5	0.3–0.9	0.03
Third quartile	0.8	0.6–1.1		0.7	0.4–1.2	
Second quartile	0.9	0.6–1.2		0.7	0.4–1.1	
First quartile	(referent)	(referent)		(referent)	(referent)	
Carbohydrate						
Fourth quartile	1.1	0.8–1.5	0.41	0.8	0.5–1.3	0.86
Third quartile	1.1	0.8–1.5		0.9	0.6–1.4	
Second quartile	1.0	0.8–1.4		0.9	0.6–1.4	
First quartile	(referent)	(referent)		(referent)	(referent)	
Type of fat ⁴						
Saturated fat						
Fourth quartile	1.03	0.60–1.7	0.89	2.3	1.2–4.5	0.04
Third quartile	1.2	0.8–1.7		1.6	1.0–2.7	
Second quartile	1.4	1.0–1.9		1.0	0.6–1.6	
First quartile	(referent)	(referent)		(referent)	(referent)	
Polysaturated fat						

	BV (Nugent ≥ 7), $n = 1521$			Severe BV (Nugent ≥ 9 and pH > 5), $n = 1521$		
	AOR	95% CI	P^2	AOR	95% CI	P^1
Fourth quartile	1.4	0.8 – 1.9	0.13	1.2	0.7–2.2	0.32
Third quartile	1.2	0.9–1.6		1.2	0.8–1.8	
Second quartile	0.9	0.6–1.2		0.8	0.5–1.2	
First quartile	(referent)	(referent)		(referent)	(referent)	
Monounsaturated fat						
Fourth quartile	1.4	0.9–1.6	0.32	2.3	1.3 – 4.1	0.001
Third quartile	1.1	0.8–1.6		1.5	0.9–2.6	
Second quartile	1.1	0.8–1.5		0.9	0.6–1.5	
First quartile	(referent)	(referent)		(referent)	(referent)	

¹ A P -value ≥ 0.05 indicates a nonsignificant trend for association between a nutrient and BV categories.

² Logistic regression model fitted for energy (kJ) and adjusted for age, race, BMI (BV only), income, education (BV only), douching, contraceptive pill use, and alcohol intake (BV only).

³ Logistic regression model fitted for each energy nutrients included fat, protein, and carbohydrate, age, race, BMI (BV only), income, education (BV only), douching, contraceptive pill use, and alcohol intake (BV only).

⁴ Logistic regression models fitted separately for each type of fat, and adjusted for protein, carbohydrate, age, race, BMI (BV only), income, education (BV only), douching, contraceptive pill use, and alcohol intake (BV only).

TABLE 3
The association between micronutrients, BV, and severe BV in women determined by AOR^{1,2}

	BV (Nugent ≥ 7), $n = 1520$			Severe BV (Nugent ≥ 9 and pH > 5), $n = 1520$		
	AOR	95% CI	P^3	AOR	95% CI	P^3
Folate						
Fourth quartile	0.8	0.5–1.1	0.49	0.4	0.2–0.8	0.01
Third quartile	0.8	0.5–1.1		0.6	0.3–1.0	
Second quartile	0.8	0.6–1.1		0.5	0.3–0.8	
First quartile	(referent)			(referent)		
Vitamin E						
Fourth quartile	0.7	0.5–1.2	0.26	0.4	0.2–0.8	0.001
Third quartile	0.9	0.6–1.3		0.7	0.4–1.1	
Second quartile	0.8	0.6–1.1		0.5	0.3–0.99	
First quartile	(referent)	(referent)		(referent)		
Calcium						
Fourth quartile	0.7	0.5–1.0	0.16	0.4	0.3–0.7	0.003
Third quartile	0.7	0.5–1.1		0.5	0.3–0.8	
Second quartile	0.8	0.6–1.1		0.5	0.3–0.8	
First quartile	(referent)			(referent)		

¹ Logistic regression models were fitted separately for each nutrient and adjusted for fat, carbohydrate, protein, age, race, BMI (BV only), income, education (BV only), douching, contraceptive pill use, and alcohol intake (BV only).

² Intakes of vitamins A, C, D, β -carotene, lycopene, fiber, zinc, and iron were not associated with BV or severe BV (data not shown).

³ A P -value ≥ 0.05 indicates a nonsignificant trend for association between a nutrient and BV categories.