

Recurrent Bacterial Vaginosis, relapse or reinfection. The role of sexual transmission. (Mini-commentary on BJOG-20-0869.R1)

Jack Sobel¹

¹Wayne State University School of Medicine

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Epidemiologic studies performed in the Melbourne Sexual Health Center over several years have explored and emphasized the role of sexual transmission in the pathogenesis of sporadic bacterial vaginosis (BV) as well as recurrent BV (Fethers KA., et al. *Infect. Dis.* 2008; 47: 1426-1435). Some of the most definitive studies documenting details of heterosexual sexual transmission followed. There can be little doubt as to the causal role of sexual transmission in BV particularly with regard to the initial episode (Cherpes, TL., et al. *Sex. Transm. Dis* 2008; 35: 78-83). The present study adds solid molecular data to their previous epidemiologic data that recurrent BV is more likely to occur in a heterosexual woman with a single regular male partner (Ratten L., et al BJOG 2020 xxxx): Moreover, the risk is mitigated by use of an oral contraceptive and barrier contraceptives. Specifically, Ratten et al conclude that sex is associated with persistence of non-optimal, BV-associated vaginal dysbiosis following appropriate antimicrobial treatment for BV in a cohort followed prospectively, likely the result of sexual transmission from a regular partner. The key term used in the title of the study is persistence, which implies that the non-optimal vaginal microbiota fails to resolve, as opposed to future reintroduction from the same guilty partner. Persistence in this context, unfortunately, also indirectly suggests that inadequate antimicrobial treatment is currently prescribed to women, perhaps sufficient to relieve symptoms and meet diagnostic criteria of satisfactory response, but insufficient to eradicate BV pathogens. The author emphasizes needed improvement in the, so far, futile male partner therapy to prevent female reinfection, a goal that has repeatedly eluded experts to date.

The unanswered question facing patients and clinicians alike is the role of sexual reinfection as opposed to vaginal relapse in the causation and likelihood of BV recurrence. The tone of the article would indicate that reinfection is the more likely causal mechanism of BV recurrence, by emphasizing “persistence” and outweighing the role of unexplained relapse. In dealing with a symptomatic patient suffering from an episode of recurrent BV, it is currently not possible to differentiate relapse from reinfection unless the patient declares herself to be celibate, ergo relapse is the cause of recurrence. The clinical picture is identical as are Amsel or Nugent criteria. Unfortunately, molecular microbiome studies have not revealed significant differences between sequential episodes regardless of causation. We lack a “unique fingerprint” to differentiate cause or nature of the recurrent episode. Even with reinfection, sexual or otherwise, details of pathogenesis are still lacking. We know too that coitus can elicit symptoms of BV (post coital malodor) even with use of a condom. The role of receptive oral-vulvovaginal sex is also undetermined, as is the role of penile – anorectal penetration although the latter was found to be minimal in the latest study by Ratten L., et al. (BJOG 2020 xxxx): Moreover, not all longitudinal studies have revealed that heterosexual sex is a major factor in recurrence (Sobel J.D., et al. *Infect. Drug Resist.* 2019; 12; 2297-2307).

The role of sex and reinfection in causation of RBV will depend significantly upon the population studied, including biologic and behavioral differences. Determination of causation of BV recurrence in different patient populations should be personalized and acknowledged as we admit our current limitations. Will more effective male treatment help reduce BV recurrence? Hopefully but still unknown. Determining all the causes of vaginal microbiota persistence, including the role of biofilm, remains a challenge.

No disclosures: A completed disclosure of interest form is available to view online as supporting information.

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