Reply to Hickey and Forney

To the Editor—We are pleased that our article prompted discussion on the pathogenesis of bacterial vaginosis (BV) [1]. As a conceptual model with testable hypotheses, this was exactly what we had hoped for, to continue the dialogue on an important topic that has been stagnant until the recent emergence of new work on *Gardnerella vaginalis* from various investigators. As Hickey and Forney point out, there remain many questions about BV. Much of the discussion and debate, however, is based on semantics, for which a history of caring for thousands of patients with BV helps provide insight.

First, let us reiterate that the title of our article states that it is a conceptual model that was put forth to stimulate discussion and further scientific investigation. Hickey and Forney point out that there are published data stating that *G. vaginalis* is found in healthy women [2]. Unfortunately, there is no definition of “healthy” vaginal flora. One logical definition might be that normal vaginal flora is acquired at birth, before the onset of any type of sexual activity that could transmit infectious agents to the vagina. The studies that are
cited by Hickey and Forney either include no sexual history data [3] or include sexual activity data based on self-report [4–6]. Despite valiant efforts to improve the quality of such data, in the end it is still self-reported. Thus, it is impossible to state whether G. vaginalis was part of the woman’s indigenous “healthy” vaginal flora or introduced via sexual activity, just as is the case with other infectious agents, such as carcinogenic human papillomavirus genotypes. As for our study [7], we were also limited by the lack of a more specific way to define normal flora and, thus, were forced to use a surrogate—a Nugent score with only lactobacillus morphotypes present. Of note, all of the women but one in our study were sexually experienced, and the woman without sexual experience was in the group of women in whom no G. vaginalis was detected.

In terms of Koch’s postulates, as discussed above, we disagree that G. vaginalis is part of the “healthy” vaginal flora. In support of the third and fourth postulates, it should be noted that only 9 women were inoculated with the 12-hour-growth culture (logarithmic phase), as opposed to the other 20 women, who received the 24-hour-growth culture (stationary phase). Of the 9 women who received G. vaginalis in the logarithmic phase of growth, 5 developed BV and G. vaginalis was reisolated from their vaginal flora [8].

We agree that important research questions remain, including why Nugent scores for some women who are colonized or infected with G. vaginalis remain in normal to intermediate, whereas others progress to BV. We concur that both host and G. vaginalis strain differences should be investigated to answer this question.

In summary, although we agree that the symptoms of BV are caused by a polymicrobial community of G. vaginalis and anaerobes, we feel that the existing data strongly support a role for G. vaginalis as the founder organism that is necessary, but not sufficient, for the symptoms of BV. Therefore, healthy vaginal flora should be defined as the absence of G. vaginalis in the setting of a robust lactobacillus-based flora.

Note

Potential conflicts of interest. All authors: No reported conflicts.

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References


