From Sexual Trauma to Vaginal Dysbiosis: A Proposed Stress-Mediated Pathway

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INTRODUCTION AND OBJECTIVE

The vaginal microbiome shapes reproductive health; bacterial vaginosis (BV), a shift from a *Lactobacillus*-dominant to a diverse anaerobic community, is linked to adverse reproductive outcomes.[1] Psychosocial stress is associated with increased BV prevalence/incidence; trauma and PTSD are independently linked to neuroendocrine dysregulation and altered genital immune/mucosal homeostasis.[2–4] Given that 45.1% of United States women report lifetime contact sexual violence and 21.0% report completed/attempted rape, a confirmed pathway linking trauma to BV would have broad reproductive health relevance.[5] The goal of this work is to synthesize evidence for a stress-mediated pathway linking sexual trauma to vaginal dysbiosis/BV, given that this pathway remains poorly characterized, and to identify gaps for future investigation.

METHODS

- PubMed search (April–August 2026) combining terms for sexual violence, PTSD/stress, vaginal dysbiosis/microbiota, cortisol/HPA-axis, inflammation, and immune markers.
- ~150 citations screened by title/abstract; primary studies most directly addressing the proposed pathway were prioritized, and reviews were used to identify additional sources.
- Studies with limited relevance to the pathway were excluded. Narrative synthesis, not a systematic review.

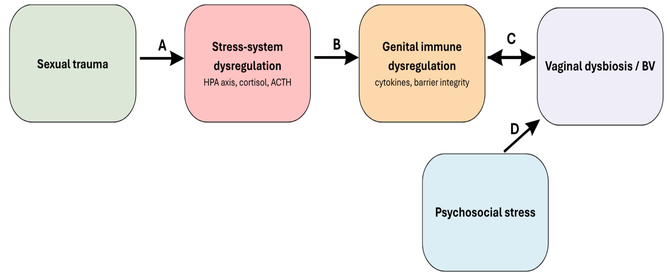

Figure 1. Existing literature supports several independently studied relationships that, when integrated, suggest a plausible stress-mediated pathway linking sexual trauma with vaginal dysbiosis. The proposed model below summarizes this conceptual framework. Proposed stress-mediated pathway linking sexual trauma to vaginal dysbiosis, integrating stress physiology (A), genital immune/mucosal changes (B), microbiome interactions (C), and population-level psychosocial stress evidence (D).

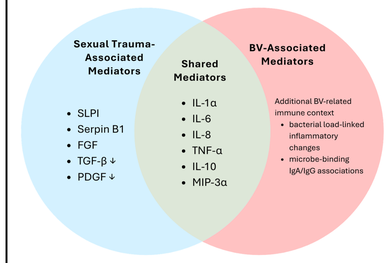

Figure 2. Overlapping inflammatory mediators identified in trauma-associated and microbiome-associated genital immune studies, supporting a shared biological interface.

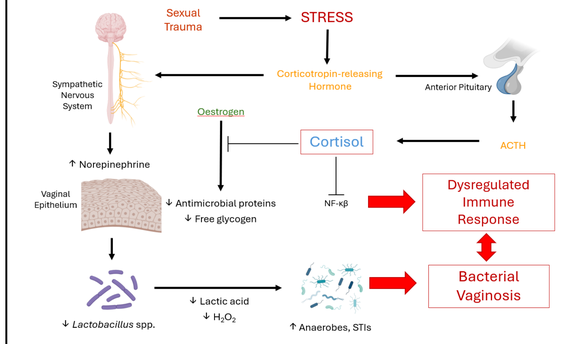

Figure 3. Proposed mechanisms linking psychosocial stress to vaginal dysbiosis through HPA- and sympathetic-axis signaling, altered immune responses, and loss of *Lactobacillus* dominance. Adapted from Amabebe and Anumba.[18]

A. Sexual Trauma → Stress-System Dysregulation

- PTSD has been shown to be linked to higher ACTH and altered cortisol vs. controls; the same cohort showed persistent cortisol/inflammatory elevation (IL-1β, MCP-1, TNF-α, CRP) at 1 year despite symptom improvement.[3,6]
- Adolescent rape survivors with established, chronic PTSD showed lower cortisol and DHEA levels vs. controls, consistent with altered HPA-axis activity.[7]
- Prior assault history predicted a blunted acute cortisol response and worse PTSD trajectories after a new assault.[8]
- Women with sexual-trauma histories showed blunted cortisol reactivity and greater anxiety during an experimental intimacy stressor vs. controls. [9]

Sexual trauma and trauma-related PTSD are associated with persistent HPA-axis dysregulation, with cortisol patterns and reactivity varying by timing, prior trauma exposure, and clinical context.

B. Trauma/Stress → Genital Immune & Mucosal Dysregulation

- Women with greater lifetime violence exposure show altered genital immune-cell profiles, epithelial changes, and reduced barrier integrity, independent of BV status.[4]
- Chronic sexual-abuse history increases IL-1α, MIP-3α, IP-10, Serpin B1, and FGF, while decreasing TGF-β, MCP-1, and PDGF in cervicovaginal secretions.[10]
- Forced-sex survivors show higher TNF-α and SLPI; PTSD symptoms correlate with genital IL-1α.[11]
- Norepinephrine has been reported to bind vaginal epithelial cells and potentiate pro-inflammatory responses.[12]

Trauma exposure is associated with altered genital immune signaling and epithelial barrier integrity across studies of lifetime violence, chronic sexual abuse, and recent forced sex.

C. Genital Immune/Mucosal Environment ↔ Vaginal Microbiome

- In cervicovaginal secretions from 196 women (validated in a second cohort), diverse BV-type communities carried higher bacterial loads than *Lactobacillus*-dominant communities; higher load correlated with elevated IL-1α, while *L. crispatus* dominance did not show this inflammatory rise even at high bacterial load.[13]
- In 200 women, IgA/IgG Ab binding to *G. vaginalis, P. bivia, L. iners, and L. crispatus* was associated with inflammatory mediators (IL-6, TNF, IP-10, MIP-3α, MCP-1, IL-8), linking local immune recognition of specific vaginal organisms to inflammation.[14]
- High-diversity, *Lactobacillus*-low vaginal communities were strongly associated with increased genital pro-inflammatory cytokines and activation of TLR4/NF-κB signaling.[15]

Vaginal microbiome composition and bacterial load are linked to immune activation, with *Lactobacillus*-depleted, high-diversity communities exhibiting inflammatory signaling and mediators identified in trauma-associated studies.

D. Psychosocial Stress → Vaginal Dysbiosis

- In a 572-woman longitudinal study, each 5-unit increase in perceived stress increased risk of molecular BV onset by 40%, *L. iners* to BV transition by 26%, and BV persistence by 23%.[16]
- In 3,614 women, higher stress predicted greater BV prevalence and incidence independent of behavioral and demographic factors.[2]
- Higher lifetime trauma was associated with ~2.5-fold greater odds of molecular BV in American Indian women.[17]
- Chronic stress hormones may reduce vaginal glycogen and antimicrobial defenses, favoring loss of *Lactobacillus* dominance and dysbiosis.[18]

Across studies, psychosocial stress and lifetime trauma are consistently linked to BV development, persistence, and impaired recovery toward *Lactobacillus*-dominant communities.

CONCLUSION & FUTURE DIRECTIONS

- Independent evidence supports each component of a stress-mediated route from sexual trauma to vaginal dysbiosis.
- Trauma and microbiome studies share overlapping inflammatory mediators (IL-1α, IL-6, IL-8, TNF-α, IP-10, MIP-3α), pointing to genital immune signaling as a biological interface.
- This convergence does not establish causality — no single study has tested the full model. Prospective studies linking trauma exposure, stress physiology, genital immune markers, and microbiome outcomes are needed to establish temporality and mediation.
- Mechanistic models (e.g., cortisol/inflammatory effects on vaginal epithelial and microbial systems) could further test the proposed mechanism.
- If confirmed, trauma-informed care may be an underrecognized avenue for reducing BV risk in trauma-exposed women.

LIMITATIONS & ALTERNATIVE PATHWAYS

- Evidence derives from separate studies of individual pathway segments; no single cohort has tested the complete proposed pathway, and observational designs cannot establish causality or temporal ordering.
- Study populations vary in trauma type, chronicity, demographics, and measurement approaches, while potential confounders were not consistently assessed across studies. Sexual trauma may also influence BV through pathways independent of chronic stress, including direct sexual/microbial exposure, genital injury, and behavioral or contextual factors.
- Genital immune–microbiome relationships may be bidirectional, limiting inference regarding which changes occur first.
- This single-reviewer narrative synthesis was designed to generate and integrate hypotheses rather than provide a systematic assessment of all available evidence.

ACKNOWLEDGEMENTS & REFERENCES

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- Full reference list available via QR code