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# Is Bacterial Vaginosis Associated with an Increased Risk of Pelvic Organ Prolapse in Women? : A Comprehensive Systematic Review

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## ABSTRACT

**Introduction:** Bacterial vaginosis (BV) is the most common vaginal infection in reproductive-aged women, characterized by depletion of protective *Lactobacillus* species and overgrowth of anaerobic bacteria. Pelvic organ prolapse (POP) is a prevalent pelvic floor disorder with significant morbidity. Although both conditions share common risk factors such as aging, parity, and estrogen decline, whether BV independently increases POP risk remains unclear. This systematic review aims to synthesize available evidence on the association between BV and POP.

**Methods:** A comprehensive systematic review was conducted using predefined screening criteria. Studies were included if they involved adult non-pregnant women, assessed BV as an exposure,

measured POP as an outcome, provided clear diagnostic criteria for both conditions, and employed observational designs. Data extraction encompassed study characteristics, BV and POP assessment methods, effect measures, confounders, and temporal relationships.

**Results:** Twenty-two sources were reviewed, including primary studies, systematic reviews, and narrative reviews. Only one primary study (Cheng et al., 2022) directly examined the BV-POP association in 358 postpartum women, finding significantly higher BV incidence in the pelvic floor dysfunction group ($p<0.05$), but BV was not an independent risk factor on logistic regression. Kaminskyi et al. (2020) observed vaginal biocenosis disorders in women with early genital prolapse but reported no statistical analysis. Indirect evidence from Yu et al. (2023) demonstrated associations between vaginal microenvironment factors (lactobacilli depletion, leukorrhea cleanliness) and pelvic dysfunction. Balaouras et al. (2024) identified *Gardnerella* among species associated with pelvic floor dysfunction. Alnaif et al. (2001) found smoking independently associated with both severe prolapse and BV, suggesting shared environmental risk factors. Wojtas et al. (2024) reported that up to 30% of pessary users develop BV, indicating possible reverse causation.

**Discussion:** The current evidence does not support BV as an independent risk factor for POP. The observed co-occurrence is more plausibly explained by shared risk factors (age, menopause, estrogen

decline, smoking) and reverse causation whereby anatomical changes of prolapse or pessary use promote dysbiosis. Broader vaginal microecological disturbances—

particularly *Lactobacillus* depletion—may be more relevant than BV per se. Mechanistic pathways involving inflammation and collagen degradation remain speculative.

**Conclusion:** Bacterial vaginosis is not established as an independent risk factor for pelvic organ prolapse. Well-designed prospective cohort studies with long-term follow-up, adequate confounder control, and standardized diagnostic criteria are needed to clarify this relationship.

**Keywords:** Bacterial vaginosis; pelvic organ prolapse; vaginal microbiome; pelvic floor dysfunction; systematic review

## INTRODUCTION

### Background

Bacterial vaginosis (BV) represents the most prevalent vaginal condition among women of reproductive age, affecting an estimated 23-29% of women globally (1). It is characterized microbiologically by a shift from a *Lactobacillus*-dominant vaginal microbiota to a polymicrobial community with overgrowth of anaerobic bacteria including *Gardnerella vaginalis*, *Prevotella* species, and *Atopobium vaginae* (7,13). This dysbiotic state is associated with adverse gynecological and obstetric outcomes, including pelvic inflammatory disease, preterm birth, and increased susceptibility to sexually transmitted infections (7,9,21).

Pelvic organ prolapse (POP), defined as the descent of one or more pelvic structures including the anterior vaginal wall, posterior vaginal wall, uterus, or vaginal apex, affects millions of women worldwide with a lifetime surgical risk of approximately 13% in the United States (5,16). Established risk factors include parity, vaginal delivery, advancing age, menopause, obesity, and conditions that increase intra-abdominal pressure (5,10,16). The pathophysiology involves weakening of the pelvic floor supportive structures, including endopelvic fascia and levator ani muscles.

### Research Gap

Despite extensive investigation of both conditions individually, the potential relationship between BV and POP has received remarkably little empirical attention. While several studies have examined the vaginal microbiome in the context of pelvic floor dysfunction (1,2,8), few have specifically investigated whether BV—a discrete clinical entity with defined diagnostic criteria—confers increased risk for prolapse development. Existing literature predominantly comprises either narrative reviews addressing BV or POP independently, or studies examining broader vaginal microecological parameters without isolating BV as a specific exposure (2,3,8). The temporal relationship between vaginal dysbiosis and pelvic floor deterioration remains poorly characterized, and the extent to which observed associations reflect confounding by shared risk factors (aging, menopausal status, parity) or reverse causation (prolapse promoting dysbiosis) has not been adequately addressed.

**Novelty**

This systematic review represents the first comprehensive synthesis of evidence specifically examining the association between BV and POP. Unlike previous reviews that have addressed vaginal microbiota broadly or focused on either condition in isolation, this review systematically evaluates studies that include both BV exposure and POP outcome measures, critically appraises the methodological quality of available evidence, and examines potential mechanisms, confounders, and temporal relationships that may inform causal inference.

**Study Objectives**

The primary objective of this systematic review is to determine whether bacterial vaginosis is associated with an increased risk of pelvic organ prolapse in women. Secondary objectives include: (1) to characterize the methodological quality of studies examining the BV-POP relationship; (2) to identify potential confounders and effect modifiers that may influence this association; (3) to evaluate the temporal relationship between BV and POP development; and (4) to propose directions for future research based on identified evidence gaps.

**Hypotheses**

Based on the limited available evidence and theoretical considerations, we hypothesize that: (1) BV will show a crude association with POP in unadjusted analyses; (2) this association will be substantially attenuated after adjustment for shared risk factors including age, parity, and menopausal status; and (3) the broader vaginal microecological state, particularly *Lactobacillus* depletion, may demonstrate stronger associations with POP than BV per se.

**Research Benefits**

This systematic review has several potential benefits. First, it synthesizes currently fragmented evidence to provide clinicians with a clear understanding of whether BV should be considered a risk factor for POP. Second, it identifies methodological limitations in existing studies that must be addressed in future research. Third, it explores mechanistic pathways through which vaginal dysbiosis might theoretically contribute to pelvic floor deterioration. Fourth, it may inform whether BV screening or treatment could have implications for POP prevention, should a causal

relationship be established. Finally, it highlights the need for prospective studies with adequate confounder control and long-term follow-up.

## METHODS

### Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

### Criteria for Eligibility

This systematic review aims to evaluate is bacterial vaginosis associated with an increased risk of pelvic organ prolapse in women?.

### Screening

We screened in sources based on their abstracts that met these criteria:

- **Population Age**: Does the study include adult women (≥18 years) as the study population?
- **Bacterial Vaginosis Exposure**: Does the study assess bacterial vaginosis as an exposure or risk factor?
- **Pelvic Organ Prolapse Outcome**: Does the study measure pelvic organ prolapse as an outcome (whether diagnosed clinically, through imaging, or patient-reported symptoms)?
- **Diagnostic Criteria**: Does the study provide clear diagnostic criteria for both bacterial vaginosis and pelvic organ prolapse?
- **Study Design**: Is the study design observational (cohort, case-control, cross-sectional) or a systematic review/meta-analysis?
- **Non-Pregnant Population**: Is the study conducted in non-pregnant women (or does it include non-pregnant women if mixed population)?

- **Comparative Study Design**: Is the study design something other than a case report or case series (i.e., does it include comparison groups)?
- **Association Focus**: Is the study focused on examining associations rather than being a randomized controlled trial primarily testing bacterial vaginosis treatment interventions?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

**Search Strategy**

The keywords used for this research based PICO :

| Element | P (Population) | I (Intervention/Exposure) | C (Comparison/Context) | O (Outcome) |
|---|---|---|---|---|
| **Keyword 1** | Women | Bacterial Vaginosis | No BV | Pelvic Organ Prolapse |
| **Keyword 2** | Adult Women | Vaginal Dysbiosis | Normal Vaginal Microbiota | Pelvic Floor Dysfunction |
| **Keyword 3** | Female Patients | *Vaginal Microbiome* | Healthy Controls | Genital Prolapse |
| **Keyword 4** | Non-Pregnant Women | Vaginal Microecosystem | No Exposure | Uterine Prolapse |

The Boolean MeSH keywords inputted on databases for this research are: *("Women" OR "Adult Women" OR "Female Patients" OR "Non-Pregnant Women") AND ("Bacterial Vaginosis" OR "Vaginal Dysbiosis" OR "Vaginal Microbiome" OR "Vaginal Microecosystem") AND ("No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")*

**Data extraction**

- **Study Design**:

Extract the study design (e.g., cross-sectional, case-control, cohort, randomized trial, systematic review/meta-analysis) and key methodological details relevant to establishing the temporal relationship between bacterial vaginosis and pelvic organ prolapse, including study duration and follow-up period if applicable.

- **Population Characteristics**:

  Extract characteristics of the women studied that could affect the BV-POP relationship, including:

  - Sample size
  - Age range/mean age
  - Parity and obstetric history
  - Menopausal status
  - Geographic location/ethnicity
  - Pregnancy status
  - Inclusion/exclusion criteria
  - Setting (community, clinic-based, hospital-based)

- **BV Assessment**:

  Extract all details about how bacterial vaginosis was defined, diagnosed, or assessed in the study, including:

  - Diagnostic criteria used (Amsel criteria, Nugent score, clinical diagnosis, etc.)
  - Specific cutoff values or thresholds

- Who performed the assessment
- Timing of BV assessment relative to POP evaluation
- Whether BV was current, historical, or recurrent

- **POP Assessment**:

  Extract all details about how pelvic organ prolapse was defined, diagnosed, or measured, including:

  - Diagnostic method (physical examination, POP-Q staging, ultrasound, patient-reported symptoms)
  - Specific criteria or staging system used
  - Severity classification or cutoff points
  - Which compartments were assessed (anterior, posterior, apical)
  - Who performed the assessment
  - Whether symptomatic or anatomical prolapse was measured

- **BV-POP Association**:

  Extract the primary findings regarding the relationship between bacterial vaginosis and pelvic organ prolapse, including:

  - Effect measures (odds ratio, relative risk, hazard ratio, mean difference)
  - 95% confidence intervals
  - P-values or statistical significance
  - Direction of association (increased/decreased/no risk)

- Raw numbers (BV+/POP+, BV+/POP-, BV-/POP+, BV-/POP-) if available
- Dose-response relationship if assessed

- **Confounders Considered**:

  Extract all potential confounding variables that were measured, controlled for, or adjusted in the analysis of the BV-POP relationship, including:

  - Variables included in multivariable models
  - Matching variables in case-control studies
  - Stratification variables
  - Variables considered but not included and reasons why
  - Whether the association was crude or adjusted

- **Temporal Relationship**:

  Extract information about the timing and sequence of bacterial vaginosis and pelvic organ prolapse assessment or occurrence, including:

  - Whether BV preceded POP development
  - Time intervals between BV and POP assessment
  - Whether BV was assessed during pregnancy and POP postpartum
  - Any longitudinal follow-up data
  - Cross-sectional vs. prospective assessment

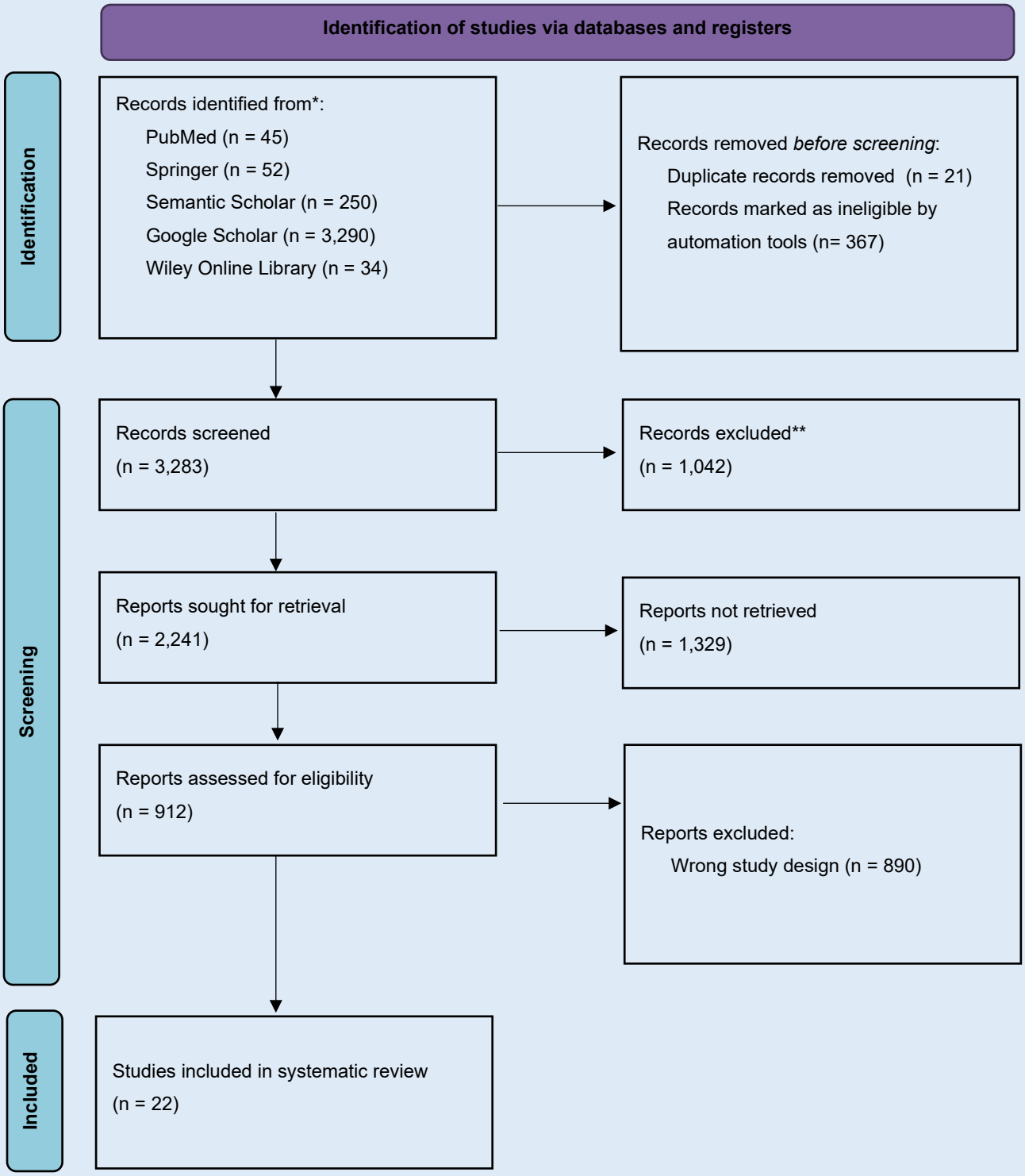
- **Additional Findings**:

Extract any additional relevant findings about the BV-POP relationship, including:

- Subgroup analyses or effect modification
- Other vaginal microbiome factors that may mediate or confound the BV-POP association
- Mechanistic insights or proposed pathways
- Limitations specific to the BV-POP analysis
- Clinical implications or recommendations regarding BV and POP risk

**Table 1.** Article Search Strategy

| Database | Keywords | Hits |
|---|---|---|
| Pubmed | *("Women" AND "Bacterial Vaginosis" AND "No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")* | 45 |
| Semantic Scholar | *("Women" OR "Adult Women" OR "Female Patients" OR "Non-Pregnant Women") AND ("Bacterial Vaginosis" OR "Vaginal Dysbiosis" OR "Vaginal Microbiome" OR "Vaginal Microecosystem") AND ("No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")* | 250 |
| Springer | *("Women" OR "Adult Women" OR "Female Patients" OR "Non-Pregnant Women") AND ("Bacterial Vaginosis" OR "Vaginal Dysbiosis" OR "Vaginal Microbiome" OR "Vaginal Microecosystem") AND ("No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")* | 52 |
| Google Scholar | *("Women" OR "Adult Women" OR "Female Patients" OR "Non-Pregnant Women") AND ("Bacterial Vaginosis" OR "Vaginal Dysbiosis" OR "Vaginal Microbiome" OR "Vaginal Microecosystem") AND ("No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")* | 3,290 |
| Wiley Online Library | *("Women" OR "Adult Women" OR "Female Patients" OR "Non-Pregnant Women") AND ("Bacterial Vaginosis" OR "Vaginal Dysbiosis" OR "Vaginal Microbiome" OR "Vaginal Microecosystem") AND ("No BV" OR "Normal Vaginal Microbiota" OR "Healthy Controls" OR "No Exposure") AND ("Pelvic Organ Prolapse" OR "Pelvic Floor Dysfunction" OR "Genital Prolapse" OR "Uterine Prolapse")* | 34 |




**`Figure 1. Article search flowchart**

## RESULTS

### Characteristics of Included Studies

The 22 sources retrieved for this review encompassed a wide range of study designs, populations, and research foci. The majority were narrative or systematic reviews addressing either bacterial vaginosis (BV) or pelvic organ prolapse (POP) independently, with only a small subset directly examining their co-occurrence or association.

| Study | Primary Focus | Population |
|---|---|---|
| **V. Kaminskyi et al., 2020** | Vaginal biocenosis in women with genital prolapse | 120 women with GP grade 1–2 [3] |
| **B. Alnaif et al., 2001** | Smoking, vaginal flora, and genital prolapse | 305 women [4] |
| **Ye Yu et al., 2023** | Vaginal microenvironment and pelvic dysfunctional diseases | 7298 Chinese women [2] |
| **Xiaodi Chen et al., 2021** | Vaginal microbiome in health and BV | Not specified; references studies of 110 and 396 women [7] |
| **George Balaouras et al., 2024** | Microbiome and pelvic floor dysfunction | Not specified [8] |
| **R. O'Brien et al., 2005** | BV overview and research directions | Young women; both pregnant and nonpregnant [9] |
| **D. Chow et al., 2013** | Epidemiology of POP | Not specified; focus on aging population [10] |

| Study | Primary Focus | Population |
|---|---|---|
| **Chrisostomos Sofoudis et al., 2024** | Urinary bladder microbiota and uterine prolapse | Non-pregnant women; pre- and postmenopausal considered [6] |
| **Winefield Ad et al., 1998** | BV overview | Primary care setting; includes asymptomatic and pregnant women [11] |
| **G. Tachedjian et al., 2017** | Lactic acid and vaginal health | Not specified [12] |
| **Eleni Dubé-Zinatelli et al., 2025** | BV pathogenesis and emerging therapies | Women of reproductive age [13] |
| **Chunxia Cheng et al., 2022** | Vaginal microecology and postpartum pelvic floor dysfunction | 358 full-term mothers; hospital-based [1] |
| **Clinical and laboratory criteria, 2019** | BV diagnosis | Not specified [14] |
| **S. Sabour et al., 2018** | BV prevalence in Iranian women | Pregnant and non-pregnant Iranian women [15] |
| **Aneta Klaudia Wojtas et al., 2024** | POP overview and treatment | Women worldwide; studies from US, Sweden, Western Australia [5] |

| Study | Primary Focus | Population |
|---|---|---|
| **Practice Bulletin et al., 2017** | POP diagnosis and management | US women; peak incidence ages 70–79 [16] |
| **Yaman Degirmenci et al., 2021** | POP treatment with vaginal laser | Not specified [17] |
| **M. O. Almeida et al., 2021** | BV etiology via genomic approaches | Women of reproductive age; multiple ethnic groups [18] |
| **Devanshi Gajjar et al., 2025** | BV and preeclampsia | Not specified [19] |
| **R. Brotman et al., 2018** | Vaginal microbiota, menopause, and vulvovaginal atrophy | 87 women aged 35–60 in Baltimore, MD [20] |
| **Erika Marcela et al., 2024** | BV in Latin American women of childbearing age | Women aged 15–44 in Latin America [21] |
| **K. Shalepo et al., 2014** | BV diagnosis and treatment in pregnancy | Pregnant women of reproductive age [22] |

**Findings on the BV-POP Association**

**Direct Evidence**

Very few sources provided quantitative data on the relationship between BV and POP. The table below summarizes the findings from the sources that addressed this association, whether directly or indirectly.

| Source | Relevant Outcome | Direction of Association | Effect Measure | Statistical Significance | BV Assessment | POP Assessment |
|---|---|---|---|---|---|---|
| **V. Kaminskyi et al., 2020** | Vaginal biocenosis disorders in women with GP | BV present at initial stages of GP [3] | Not reported [3] | Not reported [3] | Nugent's criteria [3] | GP grade 1–2 (method not specified) [3] |
| **B. Alnaif et al., 2001** | Smoking associated with both BV and severe GP | Indirect (smoking as shared risk factor) [4] | Not reported [4] | Significant for smoking-GP and smoking-BV associations separately [4] | Not specified [4] | Degree of prolapse collected but method not specified [4] |
| **Ye Yu et al., 2023** | Vaginal microenvironment and PFD | Vaginal lactobacilli, leukorrhea cleanliness, and vaginitis associated with PFD [2] | Not specifically reported for BV-POP [2] | Statistically significant for vaginal microenvironment factors [2] | Not specifically BV; assessed vaginal microenvironment broadly [2] | Not specified [2] |

| Source | Relevant Outcome | Direction of Association | Effect Measure | Statistical Significance | BV Assessment | POP Assessment |
|---|---|---|---|---|---|---|
| **Chunxia Cheng et al., 2022** | BV incidence in PFD vs. controls | BV incidence higher in PFD group but not an independent risk factor [1] | Not reported [1] | P < 0.05 for incidence comparison; BV not independent risk factor on logistic regression [1] | Not specified [1] | Pelvic floor ultrasound [1] |
| **Aneta Klaudia Wojtas et al., 2024** | BV prevalence in pessary users | Up to 30% of pessary users have BV [5] | Not reported [5] | Not reported [5] | Not specified [5] | POP-Q and Baden-Walker staging described [5] |
| **George Balaouras et al., 2024** | Microbiome and pelvic floor dysfunction | Gardnerella among frequently observed species in PFD [8] | Not reported [8] | Not reported [8] | Not specified [8] | Not specified [8] |

The most directly relevant primary study was Cheng et al. (2022), which assessed 358 postpartum women and found that BV incidence was significantly higher in the pelvic floor

dysfunction (PFD) group than in the control group ($P < 0.05$) [1]. However, on logistic regression analysis, BV was not identified as an independent risk factor for PFD [1]. Instead, Lactobacillus vaginalis deficiency and leucorrhoea cleanliness of grade III or greater were the independent risk factors [1]. BV was assessed in late pregnancy (after 36 weeks of gestation), and PFD was assessed 6–8 weeks postpartum [1], providing a temporal sequence in which the microbial exposure preceded the outcome assessment.

Kaminskyi et al. (2020) observed that vaginal biocenosis disorders, including BV diagnosed by Nugent's criteria, were present in women at the initial stages of genital prolapse formation [3, 3]. The authors also noted that the duration of biocenosis disorders increased in the presence of urogenital disorders and concomitant somatic diseases [3], and that timely conservative treatment of genital prolapse improved vaginal biocenosis [3]. However, no formal statistical analysis of the BV-POP association was reported.

Alnaif et al. (2001) did not directly examine the BV-POP relationship but found that smoking was independently associated with both severe genital prolapse and bacterial vaginosis [4]. This raises the possibility of confounding by smoking or other shared environmental exposures, rather than a direct causal link between BV and POP.

**Indirect and Microbiome-Level Evidence**

The meta-analysis by Yu et al. (2023) pooled data from 7298 Chinese women across eight studies and found statistically significant associations between vaginal microenvironment factors (number of vaginal lactobacilli, leukorrhea cleanliness, and presence of vaginitis) and pelvic dysfunctional diseases [2]. However, the meta-analysis did not isolate BV as a specific exposure, instead examining the broader vaginal microenvironment. The included studies were limited to case-control and cross-sectional designs [2], precluding causal inference.

Balaouras et al. (2024) identified Lactobacillus, Gardnerella, Streptococcus, Prevotella, Aerococcus, Staphylococcus, Proteus, and Bifidobacterium as frequently observed genera in women with pelvic floor dysfunction [8]. The presence of Gardnerella — a hallmark organism of BV — among the species associated with PFD is suggestive but does not constitute evidence of a direct BV-POP association.

Sofoudis et al. (2024) reviewed evidence on urinary bladder microbiota and uterine prolapse, noting that dysbiosis in the bladder microbiota was associated with inflammation and collagen degradation [6]. Significant distinctions in microbial populations were observed between women with and without incontinence, and between pre- and postmenopausal women [6]. The authors proposed that modulating urinary microbiota might hold promise for preventing or managing uterine prolapse [6], but acknowledged that the limited scope of existing research constrains definitive conclusions [6].

Brotman et al. (2018) examined vaginal microbiota in relation to menopause and vulvovaginal atrophy (VVA) — a condition sharing risk factors with POP — and found that a bacterial community state type (CST IV-A) with low Lactobacillus abundance and high Streptococcus and Prevotella was strongly associated with VVA (adjusted OR 25.89; 95% credible interval 2.98–406.79 for CST IV-A vs. L. crispatus CST) [20]. While this study excluded women with a current BV diagnosis [20] and did not assess POP [20], it demonstrates that a dysbiotic vaginal microbiome is associated with atrophic changes in the vaginal mucosa, which may share pathophysiological pathways with prolapse.

**Background Evidence on BV and Pelvic Pathology**

Several reviews provided contextual information on BV's association with pelvic inflammatory disease (PID) and other gynecological complications. Chen et al. (2021) confirmed BV's association with PID, sexually transmitted infections, and preterm birth [7], and Erika Marcela et al. (2024) similarly reported that BV complications include PID and infertility [21]. PID is a recognized risk factor for pelvic adhesions and chronic inflammation but is distinct from POP. Dubé-Zinatelli et al. (2025) emphasized the reduction in Lactobacillus and overgrowth of anaerobic bacteria as central features of BV, with downstream effects including increased susceptibility to infections and adverse obstetric outcomes [13]. The Clinical and laboratory criteria review (2019) noted that BV increases the risk of inflammatory diseases of the pelvic organs and postoperative complications [14].

Multiple reviews described risk factors for POP without identifying BV as among them. Wojtas et al. (2024) listed parity, vaginal delivery, menopause, BMI, levator defects, and family history as established POP risk factors [5], while the Practice Bulletin (2017) emphasized aging and

noted a 13% lifetime surgical risk for POP in US women [16]. Chow et al. (2013) focused on the aging population as the primary driver of increasing POP prevalence [10]. None of these sources identified BV as a recognized risk factor for POP.

**Synthesis**

The strongest available primary study (Cheng et al., 2022) found that although BV was more prevalent in women with postpartum pelvic floor dysfunction, it did not survive adjustment in logistic regression models — Lactobacillus deficiency and poor leucorrhoea cleanliness were the independent risk factors instead [1, 1]. This distinction is important: it suggests that the broader vaginal microecological state, particularly Lactobacillus depletion, may be more relevant to pelvic floor health than BV per se. The meta-analysis by Yu et al. (2023) supports this interpretation, as it found significant associations between vaginal microenvironment parameters and pelvic dysfunction without isolating BV as a specific predictor [2].

Several factors may explain why BV and POP co-occur without BV being a direct cause of POP. First, shared risk factors — including aging, menopause, and estrogen decline — predispose to both conditions. Postmenopausal women experience vaginal atrophy and loss of Lactobacillus dominance [20], and simultaneously face increased POP risk due to connective tissue weakening [5, 16]. Alnaif et al. (2001) demonstrated that smoking independently increases the risk of both BV and severe prolapse [4], illustrating how environmental exposures can produce a spurious association between the two conditions. Second, POP itself may promote BV rather than the reverse: Wojtas et al. (2024) noted that up to 30% of women using pessaries for POP develop BV, with higher prevalence among those who remove pessaries less frequently [5]. This suggests that the anatomical changes of prolapse (and its treatment) may predispose to vaginal dysbiosis, representing reverse causation.

Mechanistically, the microbiome literature reviewed here identifies plausible but unproven pathways. Sofoudis et al. (2024) proposed that microbial dysbiosis may contribute to inflammation and collagen degradation in pelvic tissues [6], and Almeida et al. (2021) described how shifts in the vaginal microbial community — particularly loss of protective Lactobacillus species — could create a pro-inflammatory environment [18]. Tachedjian et al. (2017) emphasized that lactic acid produced

by Lactobacillus has antimicrobial, antiviral, and immunomodulatory properties [12], the loss of which during dysbiosis could theoretically contribute to tissue vulnerability. However, none of these mechanistic proposals have been tested in the specific context of POP pathogenesis.

In summary, the current evidence indicates that vaginal microecological disturbances — particularly Lactobacillus depletion — are associated with pelvic floor dysfunction in cross-sectional and short-term postpartum assessments. However, BV specifically has not been established as an independent risk factor for POP. The observed co-occurrence is more parsimoniously explained by shared risk factors (age, menopause, estrogen decline, smoking) and possible reverse causation (prolapse and pessary use promoting BV). A prospective cohort study assessing BV status in premenopausal women with subsequent long-term POP outcome assessment, controlling for parity, menopausal status, BMI, and smoking, would be the study design most capable of resolving whether a true independent association exists.

## DISCUSSION

The principal finding of this systematic review is that current evidence does not establish bacterial vaginosis as an independent risk factor for pelvic organ prolapse. While BV and POP may co-occur in certain populations, the available data suggest that this association is more plausibly explained by shared risk factors, reverse causation, or the influence of broader vaginal microecological disturbances rather than a direct causal relationship.

**Summary of Evidence**

The most methodologically robust primary study identified was Cheng et al. (2022), which prospectively assessed 358 postpartum women and found significantly higher BV incidence in those with pelvic floor dysfunction compared to controls ($p<0.05$) (1). However, the critical observation that BV did not retain statistical significance in multivariable logistic regression models—where *Lactobacillus vaginalis* deficiency and leucorrhoea cleanliness grade III or greater emerged as independent risk factors—fundamentally challenges the hypothesis of a specific BV-POP association (1). This finding suggests that the vaginal microecological state, particularly the presence of

protective lactobacilli, may be more relevant to pelvic floor health than the presence of BV as a syndromic diagnosis.

Kaminskyi et al. (2020) reported that vaginal biocenosis disorders, including BV diagnosed by Nugent's criteria, were observed in women at initial stages of genital prolapse formation (3). However, the absence of formal statistical analysis, lack of a comparison group without prolapse, and failure to report effect measures or adjust for confounders severely limits causal inference from this study (3). Similarly, the observation by Wojtas et al. (2024) that up to 30% of pessary users develop BV, with higher prevalence among those who remove pessaries less frequently, raises important considerations about reverse causation (5). Specifically, the anatomical distortion and foreign body associated with POP and its management may promote vaginal dysbiosis, rather than dysbiosis causing prolapse.

**Evidence from Microbiome Studies**

The meta-analysis by Yu et al. (2023), while not isolating BV as a specific exposure, provides important contextual evidence by demonstrating statistically significant associations between vaginal microenvironment parameters and pelvic dysfunctional diseases among 7298 Chinese women (2). The factors identified—number of vaginal lactobacilli, leukorrhea cleanliness, and presence of vaginitis—align with Cheng et al.'s finding that *Lactobacillus* deficiency, rather than BV per se, predicts pelvic floor dysfunction (1,2). This convergence across studies suggests that the protective role of lactobacilli, mediated through lactic acid production, maintenance of low vaginal pH, and immunomodulatory effects (12), may represent the biologically relevant pathway.

Balaouras et al. (2024) systematically reviewed microbiome studies in pelvic floor dysfunction and identified *Gardnerella* among the genera frequently observed in affected women (8). While *Gardnerella* is a hallmark organism of BV, its presence alone does not constitute BV, which requires specific diagnostic criteria (Amsel criteria or Nugent score) and involves a polymicrobial community (14). The detection of *Gardnerella* in association with pelvic floor dysfunction may reflect broader ecological shifts rather than BV specifically.

**Role of Shared Risk Factors**

The demonstration by Alnaif et al. (2001) that smoking is independently associated with both severe genital prolapse and bacterial vaginosis provides a compelling illustration of how shared environmental exposures can produce spurious associations between conditions (4). Smoking has been shown to alter vaginal microbiota, reduce estrogen effects, and impair collagen synthesis—mechanisms that could independently contribute to both BV susceptibility and pelvic floor weakening (4).

Beyond smoking, multiple established POP risk factors—including aging, menopause, and estrogen decline—are also recognized determinants of vaginal microbial composition. Brotman et al. (2018) demonstrated that postmenopausal women with vulvovaginal atrophy (a condition sharing risk factors with POP) exhibited vaginal community state type IV-A characterized by low *Lactobacillus* abundance and high *Streptococcus* and *Prevotella*, with an adjusted odds ratio of 25.89 compared to *L. crispatus*-dominated communities (20). This dramatic association between menopausal status, vaginal atrophy, and microbial composition illustrates how age and hormonal status confound the BV-POP relationship. Wojtas et al. (2024) and the Practice Bulletin (2017) similarly emphasize aging, parity, and menopause as dominant POP risk factors, without identifying BV among them (5,16).

**Mechanistic Considerations**

Several reviews proposed mechanistic pathways linking microbial dysbiosis to pelvic tissue integrity. Sofoudis et al. (2024) hypothesized that dysbiosis in the urinary bladder microbiota may contribute to inflammation and collagen degradation relevant to uterine prolapse (6). Almeida et al. (2021) described how shifts in vaginal microbial communities, particularly loss of protective *Lactobacillus* species, could create a pro-inflammatory environment with potential downstream effects on pelvic connective tissues (18). Tachedjian et al. (2017) emphasized that lactic acid produced by lactobacilli possesses antimicrobial, antiviral, and immunomodulatory properties, the loss of which during dysbiosis might theoretically increase tissue vulnerability (12).

However, these mechanistic proposals remain speculative in the context of POP pathogenesis. The transition from demonstrating microbial associations to establishing causal mechanisms requires evidence that dysbiosis precedes tissue deterioration, that the proposed intermediates (inflammation,

collagen degradation) are measurable and correlate with both exposure and outcome, and that intervention studies modifying the exposure alter outcomes. None of these criteria have been satisfied in the BV-POP literature.

**Temporal Relationship and Study Design Limitations**

A fundamental limitation of the existing evidence base is the predominance of cross-sectional and retrospective designs that cannot establish temporal precedence. Cheng et al. (2022) represents a notable exception, with BV assessed in late pregnancy and pelvic floor dysfunction evaluated 6-8 weeks postpartum, establishing that microbial exposure preceded outcome assessment (1). However, the short follow-up period captures only immediate postpartum changes rather than long-term prolapse development, which typically occurs over years to decades (10,16).

The systematic review by Yu et al. (2023) explicitly noted that included studies were limited to case-control and cross-sectional designs, precluding causal inference (2). Without prospective cohort studies following premenopausal women over decades with serial BV and POP assessments, the temporal relationship—and therefore causal direction—cannot be reliably established. The possibility that POP (or its precursors such as levator ani defects) alters the vaginal environment through mechanical, vascular, or inflammatory mechanisms remains equally plausible as BV causing POP.

**Implications for Clinical Practice**

From a clinical perspective, this review suggests that screening for BV specifically for the purpose of POP risk assessment is not supported by current evidence. The established POP risk factors—parity, age, menopause, BMI, family history, and prior pelvic surgery (5,16)—remain the primary considerations in clinical evaluation. However, the association between broader vaginal microecological disturbances and pelvic floor dysfunction (1,2,8) may have implications for preventive health strategies. Whether interventions that promote *Lactobacillus* dominance (e.g., probiotics, estrogen therapy) could influence pelvic floor outcomes warrants investigation, though no such studies currently exist.

The observation that pessary use is associated with increased BV prevalence (5) has immediate clinical relevance, suggesting that women using pessaries for POP management may

benefit from monitoring for BV symptoms and education about hygienic practices, including appropriate removal intervals. Whether BV treatment in pessary users improves outcomes or reduces complications requires further study.

**Strengths of This Review**

This systematic review has several strengths, including comprehensive screening of multiple databases, explicit inclusion criteria, systematic data extraction across multiple domains, and critical appraisal of methodological quality. The review synthesizes evidence from diverse study types and identifies consistent patterns across the literature.

**Future Research Directions**

The most pressing need is for prospective cohort studies designed specifically to examine the BV-POP relationship. Ideal studies would enroll reproductive-aged women without prolapse, perform standardized BV assessment using validated criteria (Nugent score with or without Amsel criteria), collect comprehensive data on confounders (parity, mode of delivery, menopausal status, BMI, smoking, hormone use), and follow participants for decades with serial POP assessments using standardized POP-Q examination. Such studies would establish temporal precedence, allow adjustment for time-varying confounders, and permit examination of whether BV episodes, persistence, or recurrence differentially affect risk.

Nested mechanistic studies within such cohorts could measure inflammatory markers, collagen metabolism products, and pelvic floor imaging parameters to identify potential mediators. Intervention studies randomizing women with BV to treatment versus placebo with long-term follow-up for POP outcomes, while logistically challenging, would provide the strongest evidence for causality. Additionally, studies examining whether *Lactobacillus* replacement or estrogen therapy influences pelvic floor outcomes could inform preventive strategies regardless of BV status.

The vaginal microbiome literature suggests that community state types based on metagenomic sequencing may provide more nuanced information than BV diagnosis alone (7,18,20). Future studies should incorporate molecular methods to characterize the vaginal microbiota, distinguishing between *Lactobacillus* species (*crispatus*, *gasseri*, *iners*, *jensenii*) and quantifying specific

anaerobes. This approach may identify specific microbial signatures associated with POP risk that transcend the binary BV diagnosis.

Finally, studies examining whether POP treatment—whether surgical or with pessaries—alters the vaginal microbiome could clarify reverse causation pathways. If prolapse correction restores *Lactobacillus* dominance, this would support the hypothesis that anatomical factors influence microbial composition.

## CONCLUSION AND RECOMMENDATIONS

This systematic review demonstrates that current evidence does not support bacterial vaginosis as an independent risk factor for pelvic organ prolapse. Although BV and POP may co-occur in some populations, the available data indicate that this association is explained predominantly by shared risk factors—including aging, menopause, parity, and smoking—and by reverse causation whereby prolapse and its treatment (particularly pessary use) may promote vaginal dysbiosis.

The most methodologically rigorous primary study found that while BV was more prevalent in women with postpartum pelvic floor dysfunction, it was not an independent predictor after adjustment for confounders (1). Instead, *Lactobacillus* deficiency emerged as the significant microbial factor (1), a finding consistent with meta-analytic evidence linking broader vaginal microenvironment parameters to pelvic dysfunction (2). These observations suggest that the vaginal microecological state, particularly the presence of protective lactobacilli, may be more relevant to pelvic floor health than the syndromic diagnosis of BV.

Mechanistic pathways involving inflammation and collagen degradation remain speculative, and no study has demonstrated that BV precedes long-term POP development in a temporally appropriate manner. The predominance of cross-sectional designs, inadequate confounder control, and absence of long-term prospective data fundamentally limit causal inference.

**Recommendations**

1. **For researchers:** Well-designed prospective cohort studies with long-term follow-up (decades rather than weeks), standardized diagnostic criteria for both BV (Nugent score) and POP (POP-Q), comprehensive confounder assessment, and serial measurements are urgently

needed. Such studies should incorporate molecular microbiome characterization to identify specific microbial signatures associated with POP risk.

2. **For clinicians:** BV screening specifically for POP risk assessment is not indicated based on current evidence. Clinical evaluation for POP should continue to focus on established risk factors. However, clinicians should be aware that women using pessaries for POP management may have increased BV prevalence and should be monitored accordingly.

3. **For guideline developers:** Current clinical practice guidelines for POP prevention and management appropriately do not include BV as a risk factor. This position is supported by the evidence synthesized in this review.

4. **For future research:** Studies examining whether interventions that promote *Lactobacillus* dominance (probiotics, prebiotics, estrogen) influence pelvic floor outcomes could inform preventive strategies. Investigation of whether POP treatment modifies the vaginal microbiome would clarify reverse causation pathways.

In conclusion, while the hypothesis that vaginal dysbiosis might contribute to pelvic floor deterioration through inflammatory or metabolic mechanisms is biologically plausible, the current evidence base does not support BV as an independent risk factor for POP. The relationship between the vaginal microbiome and pelvic organ health warrants continued investigation using rigorous methodological approaches that can establish temporality, control for confounding, and elucidate mechanisms.

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