REVIEW ARTICLES

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Vaginal Microbiota and Pelvic Floor Dysfunction: Mechanistic Insights and Therapeutic Implications

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Pelvic floor dysfunction (PFD) encompasses a spectrum of prevalent chronic conditions, including urinary incontinence, pelvic organ prolapse, and female sexual dysfunction, arising from the impaired integrity of the pelvic floor muscles, ligaments, and connective tissues. The vaginal microbiota (VM) plays a critical role in maintaining mucosal barrier function and homeostasis within the lower reproductive tract. The integrity of the levator hiatus and urogenital hiatus maintains the closure of the pelvic floor and the stability of the VM. Emerging evidence indicates a bidirectional relationship between PFD and the VM, mediated through anatomical, hormonal-metabolic, and immunological pathways. Recent studies indicate that some of these interventions are associated with shifts in VM composition. Emerging evidence suggests that microbiota-targeted approaches, including probiotics and VM transplantation, demonstrate potential in restoring microbial balance and modulating local inflammatory responses in the lower genital tract. Although large-scale clinical trials are lacking, several studies have demonstrated that microbiota-targeted interventions, including probiotics, VM transplantation, and estriol-containing pessaries, can improve vaginal dysbiosis and modulate local inflammatory responses, with potential implications for PFD management. This review synthesizes current evidence on the interaction among the VM, pelvic floor supports, and PFD development, with a focus on underlying mechanisms and clinical associations, and discusses the potential role of microbiome-based diagnostics and therapeutics in PFD management. This article aims to review the roles and therapeutic implications of the VM and VM dysbiosis in PFD.

Keywords: Microbiota • Dysbiosis • Female Urogenital Diseases

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Introduction

Pelvic floor dysfunction (PFD) encompasses a range of conditions, including pelvic organ prolapse (POP), vaginal laxity, stress urinary incontinence, defecation dysfunction, fecal incontinence, female sexual dysfunction, and postoperative complications following pelvic floor surgery. A 2002 survey estimated that the average incidence of POP is around 40%, with age, parity, and body mass index identified as significant risk factors. A population-based cross-sectional study in China reported a prevalence of symptomatic POP at 9.6% [1,2]. In Iran, surveys have shown an overall urinary incontinence prevalence of 39.5%, with stress urinary incontinence affecting 20.6% of women [3]. Multiple studies indicate that stress urinary incontinence affects between 26% and 49% of women, significantly impacting their quality of life [4]. By 2050, the prevalence of female urinary incontinence is projected to reach 55% [5-8].

Every part of the human body hosts a complex microbial community that coexists with the host and plays vital roles in maintaining health. Numerous studies have demonstrated that gut microbiota is crucial for food digestion, immune system function, and protection against infections [9]. Similarly, the vaginal microbiome, a key focus of the Human Microbiome Project, is closely related to women's reproductive health [10]. Although various studies have been conducted on the mechanism of PFD, the relationship between vaginal microbiota (VM) and PFD, as well as the relationship between the involvement of VM and the outcome of treatment for PFD, are not fully understood.

In 2011, Ravel et al analyzed the VM of 396 asymptomatic North American women from 4 ethnic groups (White, Black, Hispanic, and Asian) using 16S rRNA gene sequencing, and found that lactobacilli were dominant in the lower reproductive tract of women [11]. Ravel et al identified 5 distinct vaginal bacterial community types, termed community state types (CSTs): CST-I is dominated by *Lactobacillus crispatus*; CST-II by *Lactobacillus gasseri*; CST-III by *Lactobacillus iners*; CST-IV is characterized by a low abundance of *Lactobacillus* and a high diversity of anaerobic bacteria such as *Gardnerella*, *Atopobium*, *Megasphaera*, and *Mobiluncus*; and CST-V is primarily composed of *Lactobacillus jensenii* [11]. The functions of the main lactobacilli in each CST are shown in **Table 1**. Beyond the VM, emerging evidence suggests that microbial communities across the female reproductive tract, including the cervix and endometrium, undergo dynamic changes in response to physiological processes. For example, Anna Bednarska-Czerwińska et al demonstrated that microbiome composition in the endometrium and uterine cervix shifts significantly during embryo implantation [15].

This article aims to review the roles and therapeutic implications of the VM and VM dysbiosis in PFD. The discussion is organized around 3 interrelated mechanistic pathways: (1) anatomical and structural interactions between pelvic floor integrity and the vaginal microenvironment; (2) hormonal and metabolic influences on microbial composition; and (3) immunological regulation at the host-microbe interface. Building on this mechanistic foundation, we further examine how established PFD interventions, such as pelvic floor muscle training, biofeedback, and electrical stimulation, are associated with shifts in VM composition, and consider their potential roles as indirect or adjunctive strategies for microbiome modulation.

Table 1. Key functions of the representative organisms of the normal vaginal microbiota by community state type (CST).

Author, year	Main organism	CST	Key functions
Ravel et al, 2011 [11]; Rizzo A et al, 2013 [32]	*Lactobacillus crispatus*	CST-I	Dominant species in the most stable CST; produces high-level D/L-lactic acid and H_2O_2, maintains vaginal pH < 4.5, strongly inhibits bacterial vaginosis-associated bacteria and pathogens; upregulates tight-junction proteins and anti-inflammatory defensins
Ravel et al, 2011 [11]; Zhang Z et al, 2025 [12]	*Lactobacillus gasseri*	CST-II	Positive correlation with interferon α and interferon β expression levels; regulate the immune response of epithelial cells
Ravel et al, 2011 [11]; Petrova MI et al, 2017 [13]	*Lactobacillus iners*	CST-III	The most abundant female genital tract species worldwide; cysteine dependence; associated with higher risk of bacterial vaginosis
Ravel et al, 2011 [11]; Nori SRC et al, 2023 [14]	*Lactobacillus jensenii*	CST-V	Produces lactic acid and bacteriocins; reduce vaginal pH; involved in cell wall synthesis, lactate and acetate metabolism

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Review Approach

This narrative review summarizes current evidence on the relationship between the VM and PFD. Relevant literature was identified through searches of major electronic databases, including PubMed, Web of Science, and Scopus, for studies published up to December 2025. Search terms included combinations of "pelvic floor dysfunction", "pelvic organ prolapse", "urinary incontinence", "vaginal microbiota", "vaginal microbiome", "urobiome", and "dysbiosis". Studies were selected based on their relevance to the topic, with priority given to original research articles, clinical studies, and recent reviews addressing microbiota composition, host-microbe interactions, and therapeutic approaches in PFD and related conditions. Additional articles were identified through manual screening of reference lists. The included studies were qualitatively synthesized, with emphasis on key themes, including microbial composition, potential mechanisms linking microbiota and pelvic floor function, and emerging microbiome-targeted interventions.

Effects of Pelvic Floor and Vaginal Anatomy on the VM

The female pelvic floor consists of muscles, ligaments, nerves, and connective tissues that together support the pelvic organs (including the bladder, uterus, and rectum) and maintain their normal function. The fibromuscular connective tissue within the pelvic ligaments, as well as the levator ani muscles, play a key role in supporting these organs. Damage to these connective tissues, ligament weakness, or a reduction in levator ani muscle volume can increase the risk of POP [16]. Women with POP often exhibit defects in the levator ani muscles and have lower vaginal closure force during maximal contraction compared with women without POP [17]. Chen et al investigated patients with Mayer-Rokitansky-Küster-Hauser syndrome who underwent laparoscopic peritoneal vaginoplasty and found that the primary microbial sources for the neovagina originated from adjacent anatomical sites, particularly the perineum and anal region. Furthermore, the evolution of the neovaginal microbiota was found to be synchronized with vaginal tissue remodeling, including squamous epithelialization and improvements in nutritional conditions [18]. The integrity of the pelvic floor helps maintain the natural closure of the vaginal canal and preserve a balance between aerobic and anaerobic bacterial communities in the vagina, limiting the entry of external pathogens. Conversely, PFD, such as cystocele or rectocele, which usually originates from or are accompanied by an enlarged urogenital hiatus, can alter the vaginal microenvironment by disrupting the balance of the VM [19].

Childbirth, a major risk factor for POP, is also closely associated with changes in the VM. Some findings predominantly derived from cross-sectional sequencing studies and small observational cohorts indicate that anatomical changes combined with the specific hormonal status during lactation in the vagina can influence microbial colonization. Zhang X et al demonstrated that the composition of the VM undergoes significant shifts from pregnancy to the postpartum period. In Chinese women, the proportion of *Lactobacillus* species decreases after delivery, leading to an increase in microbial diversity [20]. Costello et al reported that childbirth, regardless of delivery mode, is associated with a vaginal pro-inflammatory cytokine response and a loss of *Lactobacillus* dominance. Specifically, the proportion of *Lactobacillus* significantly declined from 41% before delivery to 4% in the postpartum period, and had not fully recovered 1 year later. After delivery, the decline in *Lactobacillus* abundance, a key component of a healthy VM, may be related to structural and hormonal changes in the pelvic floor. The sustained elevation of inflammatory factors may further reflect alterations in the local microenvironment due to vaginal mucosal injury or changes in pelvic floor support structures [21].

Female Hormones and Pelvic Floor Anatomy Co-Regulate the VM

Evidence linking hormonal status to VM composition is supported by clinical observational studies and mechanistic findings; however, most human data remain cross-sectional. Graham et al summarized the relationship between the VM and conditions affecting women of reproductive age, such as polycystic ovary syndrome, unexplained infertility, postpartum depression, and menopausal disorders. Collectively, these findings indicate that throughout a woman's life, the VM and steroid hormones interact closely, with both playing an important role in maintaining women's reproductive and overall health [22].

Estrogen is known to stimulate vaginal epithelial cells to increase glycogen production, which promotes the growth of *Lactobacillus* species and supports the establishment of a *Lactobacillus*-dominant vaginal microbiome [23]. Genitourinary syndrome of menopause (GSM) refers to a cluster of symptoms and signs associated with estrogen deficiency, including vaginal dryness, burning, itching, irritation, dyspareunia, urgency urinary incontinence (UUI), difficulty urinating, and recurrent urinary tract infections [24]. Zeng et al reported that 83.58% of perimenopausal and postmenopausal women experience GSM. Notably, the incidence and severity of GSM are negatively correlated with the abundance of *Lactobacillus* species. Women with GSM tend to have higher richness and diversity in their VM, further indicating that changes in the vaginal microbiome are closely associated with the development of GSM [25]. Studies have shown that the surge in steroid hormone levels during adolescence is associated with reduced microbial diversity in the vaginal microbiome, further confirming the involvement of steroid hormones in regulating the composition and stability of the VM [26]. A decline in estrogen levels, reduced muscle mass, and postpartum as well as postmenopausal microenvironmental changes may collectively lead to pelvic floor

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muscle relaxation, which is often accompanied by a reduction in *Lactobacillus* abundance and an increase in microbial diversity.

Interaction Mechanisms Between VM Metabolism and Pelvic Floor Structure-Related Metabolism

The female vaginal barrier is formed by an intact layer of vaginal epithelial cells connected by tight junctions and desmosomal proteins [27]. These epithelial cells provide a mechanical barrier and enhance immune defense by interacting with epithelial cell pattern recognition receptors and promoting the release of inflammatory cytokines, forming the first line of defense against microbial infections [28]. Delgado-Diaz et al demonstrated that vaginal *Lactobacillus* species can directly strengthen the integrity of the epithelial barrier and upregulate the expression of tight junction proteins through the production of the metabolite lactate [29]. Beneath the epithelium, the connective tissue layer of the vaginal wall is composed of a dense extracellular matrix (ECM). Fibroblasts and myofibroblasts synthesize and maintain fibrillar components, including type I, III, and V collagens and elastin, which provide structural strength to the ECM [30]. A unique feature of the female reproductive tract is its capacity for cyclic tissue remodeling, which involves periodic inflammatory clearance to maintain reproductive function. However, aging or other pathological factors can disrupt this balance, leading to chronic inflammation and fibrosis [31]. Collectively, these findings suggest that there are bidirectional interactions between the metabolic activity of the VM and the metabolic processes involved in pelvic floor connective tissue maintenance.

Mechanistic evidence in this area is largely derived from in vitro experiments, with relatively limited direct validation in human clinical studies. Furthermore, human studies linking microbial metabolites to ECM remodeling are scarce and often indirect. In a HeLa cell–based experimental model, *Lactobacillus crispatus* was shown to reduce infections by other microorganisms by enhancing epithelial cell defenses through the regulation of key immune mediators, including toll-like receptors 2/4, interleukin 8, and β-defensins 2 and 3 [32]. The fibroblast cell number and type I collagen production can be significantly impaired by *Gardnerella vaginalis*, suggesting bacterial vaginosis–associated microbes may change fibroblast function, thereby leading to the aggravation of POP [33]. Metabolites produced by *Lactobacillus spp.* may protect pelvic floor connective tissue from chronic inflammatory damage by regulating the inflammatory response of vaginal epithelial cells.

Potential Roles of the Immune System in VM Dysbiosis and Pelvic Organ Dysfunction

It is well established that microbial antigens and metabolites continuously interact with the host immune system, eliciting microbiota-specific immune responses without necessarily provoking overt inflammation. Dysregulation of this delicate immune-microbiota balance can lead to inflammation and disease [34]. All microorganisms colonizing epithelial barrier surfaces contribute to the induction and modulation of immune responses, even in the absence of direct pathogenic challenge [35].

Innate immune cells play vital roles in tissue injury, repair, and homeostasis. Among these, macrophages are key coordinators of tissue repair processes, including the activation of fibroblasts and maintenance of ECM homeostasis [36]. It has been shown that the morphology and distribution of tissue-resident macrophages in organs and tissues of older adult individuals undergo age-related changes, characterized by impaired phagocytic and autophagic capacities and reduced tissue repair function. These dysfunctions are closely associated with age-related diseases and chronic inflammation, often driven by cellular senescence and the development of a senescence-associated secretory phenotype [37]. Current evidence on immune-microbiota interactions in PFD includes a combination of single-cell sequencing studies, observational clinical research, and experimental models. In 2021, Li Y et al used single-cell sequencing to investigate the regulatory mechanisms underlying POP. Their findings highlighted the significant roles of fibroblasts and macrophages in ECM remodeling and immune dysregulation in POP. The study demonstrated that various cell types contribute to pathological ECM degradation and aberrant immune responses during the development of prolapse [38]. More recently, Miao et al examined age-related differences in the pelvic microenvironment by comparing young and old patients with POP. Their results showed that in older patients, POP is primarily associated with chronic inflammation, whereas in younger patients, ECM metabolic dysregulation is the dominant factor [39].

Correlation Study Between VM and Treatments for Pelvic Organ Dysfunction

Effects of Pelvic Floor Biofeedback and Electrical Stimulation on VM in Postpartum Women with PFD

PFD arises from multiple contributing factors, with pregnancy recognized as a major risk factor [40]. The physiological changes in pelvic floor muscles, connective tissues, and the reproductive system during pregnancy are strongly associated with fetal development and childbirth-related strain [41]. Biofeedback is a conservative treatment that enhances voluntary pelvic muscle contractions and strengthens muscle tone. Numerous studies have demonstrated that biofeedback is effective for managing various forms of PFD, including stress urinary incontinence, fecal incontinence, POP, and female sexual dysfunction [42]. Studies evaluating the effects of biofeedback

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and electrical stimulation are primarily small clinical cohorts or interventional studies with limited microbiome-specific endpoints. Li W et al evaluated the efficacy of different biofeedback protocols in postpartum women with severely weakened pelvic floor muscle strength. Their findings showed that transvaginal biofeedback electrical stimulation significantly improved voluntary pelvic muscle control and enhanced muscle strength in this population [43]. Beyond its mechanical benefits, biofeedback may also influence the vaginal microbiome. For example, studies have suggested that biofeedback can inhibit the growth of pathogens such as *Escherichia coli*, *Staphylococcus aureus*, and *Pseudomonas aeruginosa* [44]. Zhang Y et al further proposed that biofeedback can help restore and maintain a healthy VM dominated by *Lactobacillus* species, thereby reducing the risk of vaginal inflammation. Notably, biofeedback was associated with an increase in the abundance of *Lactobacillus crispatus*, which is known to strengthen mucosal defenses against infection and modulate local anti-inflammatory responses [45].

Changes in the VM Following the Use of a Pessary

Treatment strategies for female POP include conservative measures such as lifestyle modifications (eg, weight management) and pelvic floor muscle training; non-surgical devices such as vaginal pessaries; and surgical interventions [46]. For patients who undergo surgical treatment, Chen S et al retrospectively compared the VM of patients who received vaginal hysterectomy for POP with those undergoing hysterectomy for non-POP indications. Their findings indicated that the VM dysbiosis could lead to increased secretion of matrix metalloproteinase-3 in the uterosacral ligament tissue, resulting in collagen degradation, suggesting that VM dysbiosis may contribute to POP pathogenesis and offer potential avenues for novel therapeutic approaches [47]. The vaginal pessary is a widely used non-surgical treatment option for patients with POP. However, its use can be associated with complications, including vaginal bleeding, mucosal erosion, and abnormal discharge [48]. Some observational studies provide evidence for changes in VM associated with the use of pessary, indicating that pessary use can alter the vaginal microenvironment [49]. Studies have shown that most pessary-associated biofilms are deficient in *Lactobacillus* species and that their microbial composition closely resembles that of the surrounding VM [50].

In women with POP, vaginal dysbiosis is typically characterized by a decrease in *Lactobacillus* dominance and an increase in microbial diversity [51]. Comparative studies have demonstrated that patients with untreated POP exhibit the highest alpha diversity in species richness relative to pessary-treated or post-surgical groups [52]. Nevertheless, neither pessary use nor surgical intervention fully restores a healthy vaginal microbiome; shifts in vaginitis-associated bacteria often persist [53].

Improvement of the VM May Enhance the Success of Pelvic Organ Dysfunction Treatment

The VM of patients with POP is often characterized by a reduction in *Lactobacillus* abundance and increased microbial diversity, commonly classified as CST IV. This dysbiotic state highlights the importance of restoring a healthy VM following surgical repair or pessary use. Current evidence supporting microbiota-targeted interventions, such as probiotics and local estrogen therapy, is derived from a mix of small clinical trials and observational studies. Alongside anatomical correction, adjunctive therapies aimed at reestablishing a *Lactobacillus*-dominant microbiota may play a critical role in maintaining the long-term health of the female reproductive tract, especially in postmenopausal women [52]. Effective treatments for PFD, such as pelvic floor electrical stimulation, have been shown to influence the composition of the VM, indicating that part of their therapeutic effect may be mediated through modulation of the local microbial community [45].

Additionally, certain interventions, such as estriol-containing pessaries, may exert dual benefits by alleviating vaginal or intestinal dysbiosis [54]. For instance, Long et al developed a pessary capable of sustained estriol release for up to 3 months, demonstrating that targeted modulation of the vaginal environment can be integrated into comprehensive POP management [55]. Multiple studies have shown that probiotic-based modulation of the VM composition can improve the vaginal microenvironment and is a promising strategy for the prevention and treatment of vaginal inflammatory disorders [56,57].

Therapeutic Interventions Targeting Pelvic Organ Dysfunction Contribute to the Restoration of VM Homeostasis

The postpartum period is a critical window for coordinated remodeling of pelvic floor tissues, such as muscles and connective tissues and the vaginal microenvironment. A longitudinal study following women from their first pregnancy to 8 years postpartum demonstrated that the pelvic floor undergoes substantial biomechanical adaptations during gestation, which may predispose to PFD but also facilitate postpartum recovery following vaginal delivery [58]. Evidence suggests that declines in pelvic floor muscle function during the early postpartum period (typically 6-8 weeks) can disrupt VM balance. Conversely, recovery of pelvic floor muscle strength and integrity supports the reestablishment of a healthy, *Lactobacillus*-dominant vaginal microbial community [59]. Interventions targeting pelvic floor anatomy and function may therefore indirectly support VM homeostasis, while restoration of a healthy microbiota may in turn support tissue repair. These findings highlight a functional interaction between therapeutic interventions and microbiota regulation.

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Table 2. The vaginal microbiota of the lower reproductive tract in healthy women and women with pelvic floor dysfunction.

Author, year	Vaginal microbiota	Associated clinical conditions
Ravel et al, 2011 [11]	*Lactobacilli dominant* (*Lactobacillus crispatus*, *Lactobacillus gasseri*, *Lactobacillus iners*, and others)	Main vaginal microbiota in the lower reproductive tract of healthy women of childbearing age
	Community state type IV: Anaerobic bacteria dominant (*Gardnerella*, *Atopobium*, *Megasphaera*, *Mobiluncus*, and others)	More related to inflammatory diseases of the lower genital tract
Zeng Q et al, 2024 [25]	The abundance of *Lactobacillus* species decreases	Vaginal dryness, burning, itching, irritation, dyspareunia, urgency urinary incontinence, difficulty urinating, and recurrent urinary tract infections
Ashrafi et al, 2017 [44]	The abundance of *Escherichia coli*, *Staphylococcus aureus*, and *Pseudomonas aeruginosa* decreases	Biofeedback may also influence the vaginal microbiome
Zhang Y et al, 2019 [45]	The abundance of *Lactobacillus crispatus* increases	After biofeedback, strengthen mucosal defenses against infection and modulate local anti-inflammatory responses
Gottschick et al, 2017 [51]	A decrease in *Lactobacillus* dominance and an increase in microbial diversity	Pelvic organ prolapse
Karstens et al, 2016 [61]	A decrease in microbial diversity	Increased severity of urinary incontinence symptoms
Li K et al, 2022 [62]	*Lactobacillus* and *Streptococcus* (phylum *Firmicutes*), *Gardnerella* (phylum *Actinobacteria*), *Prevotella* (phylum *Bacteroidetes*), as well as *Methylobacterium*, *Acinetobacter*, and *Sphingomonas* (phylum *Proteobacteria*)	significantly associated with the severity of overactive bladder sub-symptoms

Bidirectional Interplay Between the VM and PFD: Diagnostic and Therapeutic Implications

Labor injury is an independent high-risk factor for PFD. Studies on the VM of women after childbirth have found that as the diversity and inflammation decrease during the postpartum period, the dominant position of the typical healthy symbiotic bacterium, *L. crispatus*, has not been restored [22]. Postpartum or long-term abnormal microbiota may be a confounding or promoting factor for PFD.

A growing body of evidence supports a bidirectional relationship between VM composition and PFD. A recent systematic review identified key microbial taxa, such as *Gardnerella*, *Streptococcus*, and *Prevotel*a, as important contributors to urogenital health and disease [60]. The vaginal microbiota of the lower reproductive tract in healthy women and women with PFD can be seen in **Table 2**. Alterations in vaginal microbial communities may influence local inflammation, tissue integrity, and susceptibility to PFD-related conditions. For instance, Karstens et al found significant differences in the relative abundance of bacteria (*Lactobacillus*, *Proteus*, *Prevotella*, *Bifidobacterium*, *Gardnerella*, *Escherichia coli*, *Shigella*) in female patients with urinary incontinence. It is speculated that the increased severity of urinary incontinence symptoms is related to a decrease in microbial diversity [61]. Li K et al conducted a clinical study using 16S rRNA gene sequencing technology for microbiological analysis on the urine of patients with overactive bladder. Several specific bacterial genera were found to be significantly associated with the severity of overactive bladder sub-symptoms, including *Lactobacillus* and *Streptococcus* (phylum Firmicutes), *Gardnerella* (phylum Actinobacteria), and *Prevotella* (phylum Bacteroidetes), as well as *Methylobacterium*, *Acinetobacter*, and *Sphingomonas* (phylum Proteobacteria). These results suggest that the bladder microbiota may exert a potential regulatory effect on the severity of overactive bladder symptoms [62]. Some studies also focused on the role of VM in PFD, such as urgency urinary incontinence. Research conducted by Nardos et al showed no significant discrepancies in the vaginal or bladder microbiomes between women with urgency urinary incontinence and those without it in terms of alpha and beta diversity [63]. In addition, Veit-Rubin et al reported a higher abundance of *Veillonella spp.* in the VM of patients with mesh contraction [64].

Current clinical evidence supports the therapeutic potential of vaginal microbiota transplantation in treating dysbiotic conditions such as bacterial vaginosis. Probiotics and VMT will be a

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promising intervention for restoring a *Lactobacillus*-dominant vaginal environment and suppressing the overgrowth of anaerobic bacteria [65]. If it is verified that vaginal dysbiosis leads to or promotes the formation of POP by affecting the microenvironment, future studies may explore VM transplantation as a preventive and therapeutic approach for PFDs, including POP, rather than being limited to vaginitis [66].

Future Recommendations

Future research should also expand to investigate cross-talk between the gut and urogenital microbiota. The type of PFD should be detailed. Future microbial association studies are needed to obtain more PFDs, such as sexual dysfunction disorders and vaginal laxity. In the future, the integration of structural restoration via surgical repair or pessary use, functional muscle rehabilitation such as electrical stimulation, and targeted microbial modulation, including probiotics or VM transplantation, is likely to shape the next generation of comprehensive treatment strategies for POP. In addition, it is necessary to use new technologies, such as metagenomic sequencing, to directly obtain genetic information of the entire microbiome from clinical samples to identify specific microbial species associated with different types of PFD. It can also be used to study microbial gene functions and predict antibiotic resistance.

Conclusions

The role of VM in POP is still unclear. This review points to the need for further studies on how the VM influences pelvic floor tissue repair through immune, metabolic, and signaling pathways, with robust mechanistic validation in clinical and preclinical settings.

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