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Review Article

Use of probiotics in vaginal infections

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ABSTRACT

Vaginal infections are characterized by an overgrowth of microorganisms opportunistic and decreased levels of Lactobacillus. Vaginosis is common in women of childbearing age, perimenopausal women, women with polycystic ovary syndrome, pregnant women and adolescents. Vaginosis is often underestimated or misdiagnosed. Multiple studies mention that vaginosis detection tests should be performed on all high-risk pregnancies, women with significant obstetric history such as premature abortions and those who suffer from tuberculosis, because they are a risk factor for the development of abortions, premature rupture of membranes, choriomanionitis or preterm births. A challenge that is becoming more and more common revolves around the resistance of the microorganisms that cause vaginosis to the established treatments, generating recurrent and multi-resistant infections. For the treatment of different vaginosis, antibiotics and antifungals are prescribed. Studies point out the importance of prescribing antibiotics together with probiotics and thus avoiding dysbiosis in order to avoid recurrence of vaginal infections. Probiotics must be administered in the necessary dose and time so that they can generate a positive effect. More than 80% of Lactobacillus species must be present in a probiotic to ensure adequate acid and bactericidal production to prevent microfilm formation. The results of the use of probiotics will depend on the quality of the formula and the maintenance of the cold chain.

Keywords: Multi-resistance, Probiotics, Vaginal infections, Vaginosis

INTRODUCTION

Vaginosis is one of the most common causes of abnormal vaginal discharge in women of reproductive and premenopausal age.[1] Bacterial vaginosis (BV) is the most bothersome of these, characterized by an overgrowth of opportunistic bacteria and a reduction in Lactobacillus levels. A healthy vaginal flora is dominated by Lactobacillus, which accounts for 95% of the total bacterial flora however, some studies indicate that some healthy women do not have Lactobacillus dominance. In cases of BV, gram-positive coccus microorganisms and gram-negative bacilli dominate the bacterial flora. These include Prevotella species, *Gardenella vaginalis* and Mobiluncus.[2] The prevalence of BV ranges from 15% to 30% in non-pregnant women, while in pregnant women it is more than 50%.

The prevalence of vaginal infections varies by country and continent, with those with lower economic resources suffering most frequently. A high incidence has been reported in Africa, a moderate prevalence in Asia and a low prevalence in Finland, Australia and New Zealand.[3] Cultural, social, economic and environmental factors play

a fundamental role in the presence and incidence of different vaginal infections. Additionally, there are differences in the surveillance techniques used and BV may not be a disease reported by the epidemiological system of each country. Diagnostic techniques vary by country and depend on the skills, resources and techniques implemented by each operator and laboratory. Currently, the prevalence of vaginal infections is increasing. Risk factors associated with a high prevalence of BV include unprotected sexual activity, frequent use of condoms, douching and diabetes.[4]

The approach to diagnosing and managing BV can vary. Although it is highly prevalent in different countries, it remains underdiagnosed and not taken seriously. This makes the chances of receiving adequate treatment remote, increasing morbidity and the risk of contracting other sexually transmitted infections (STIs). Observational studies have found that douching is associated with an increase in vaginal infections due to altered vaginal flora.[5]

REVIEW

This is a descriptive-exploratory literature review. The bibliographic search period ranged from 2014 to 2024 in electronic databases such as PUBMED, ELSEVIER and Web of Science, both in English and Spanish. The keywords used in the MesH search were: vaginal infections, vaginosis, probiotics, multi-resistance.

Inclusion criteria: search terms, level of evidence, abstracts and keywords; exclusion criteria: unrelated to the topic, outside the year limit, unavailable; classifications were made by year, type of study and level of evidence. Eligibility criteria included critical reading, level of evidence, documents available for analysis and relevance to the topic. A total of 20 sources were obtained for analysis and synthesis.

MECHANISM OF PROBIOTICS IN BACTERIAL VAGINAL INFECTIONS

The vaginal microbiome is essential for supporting female reproductive health, with Lactobacillus species accounting for 70-90% of the typical vaginal flora in healthy women. These helpful bacteria maintain vaginal homeostasis by generating lactic acid, hydrogen peroxide (H_2O_2) and bacteriocins, creating an acidic environment (pH 3.5-4.5) unfriendly to infections.

When this delicate balance is disturbed, women frequently develop one of two problems: bacterial vaginosis or yeast infections. Other oxygen-avoiding germs push out beneficial Lactobacillus bacteria in BV, but *Candida albicans* fungal overgrowth causes yeast infections. Antibiotics are frequently administered for these disorders, but many women are locked in a painful cycle of recurring infections after treatment. This ongoing conflict has encouraged both patients and healthcare practitioners to look more seriously at probiotics as a kinder and maybe more beneficial long-term strategy to supporting vaginal health.[1]

Probiotics provide several benefits to women. First, they make lactic acid, which creates a somewhat sour environment in which pathogenic bacteria cannot live. Many Lactobacillus strains produce hydrogen peroxide, which functions as a natural disinfectant against certain bacteria. These beneficial microorganisms even produce their own natural antibiotics, known as bacteriocins, which kill harmful pathogens directly. Lactic acid has been identified by researchers to permeate the outer surface of dangerous creatures, disturbing their interior chemistry and blocking key life activities. This natural treatment works astonishingly well against both the problematic bacteria underlying BV and the Candida yeast, which causes most vaginal fungal infections.[2]

Another important mechanism is competition for adhesion sites on vaginal epithelial cells. Lactobacilli possess surface components such as carbohydrates and glycoproteins that enable them to adhere strongly to vaginal mucosa, forming a protective biofilm that prevents pathogen colonization. This competitive exclusion mechanism is particularly important for preventing *Gardnerella vaginalis* attachment in BV and *Candida albicans* adhesion in VVC. Some probiotic strains can also displace already attached pathogens. Research has demonstrated that *Saccharomyces cerevisiae* can cause co-aggregation of Candida, preventing its adhesion to mucosal surfaces.[3]

Probiotics also help to regulate the host immune response, resulting in a more balanced inflammatory environment. Lactobacillus species affect both innate and adaptive immunity by boosting phagocyte activity and regulating cytokine production. Probiotics have been shown to decrease pro-inflammatory cytokines including IL-8, IL-1β, TNF-α and IL-6 while increasing anti-inflammatory cytokines like IL-10. *Saccharomyces cerevisiae* increases the antibacterial capabilities of polymorphonuclear cells in VVC by producing more reactive oxygen species. Probiotics, by controlling the inflammatory response, can minimize symptoms and tissue damage associated with vaginal infections.[4]

Women participating in clinical trials have experienced mixed results when using probiotics for vaginal health issues. When it comes to bacterial vaginosis, the research picture is quite encouraging - numerous studies show that women benefit from taking probiotics either by themselves or alongside traditional antibiotics. Several comprehensive reviews of multiple studies reveal that women using probiotics tend to recover more completely and experience fewer BV flare-ups than those taking dummy pills. The story changes with yeast infections though, where the evidence isn't nearly as clear-cut.

How well a woman responds to probiotic treatment can vary depending on a few important factors—like the

specific strains of bacteria she's using, whether she takes them by mouth or as vaginal suppositories, the dose she takes and how long she sticks with the treatment. Researchers are now diving into promising new types of beneficial bacteria and uncovering the intriguing link between gut health and vaginal wellness.[5]

Scientists are now searching beyond the well-known Lactobacillus bacteria to discover other beneficial microbes that might help humans stay healthy. One surprise contender is baker's yeast or *Saccharomyces cerevisiae,* the yeast used to create bread. Recent lab and animal research reveal that this common yeast might be a valuable ally. When applied in the vaginal area, it can help keep Candida under control, prevent it from becoming more aggressive, diminish its potential to cause infections and even break down the strong protective layers it forms to cover itself.

Women who received vaginal applications of *S. cerevisiae* experienced a reduction in Candida levels comparable to those taking the prescription medication fluconazole. Similarly impressive results occurred with bacterial vaginosis, where the yeast substantially decreased harmful bacteria and even cleared Gardnerella from the upper reproductive tract, suggesting it might effectively treat both yeast infections and bacterial conditions.[3]

The mode of delivery influences the effectiveness of probiotics for vaginal health. Vaginal administration is a direct route, allowing probiotics to colonize the vagina quickly. Although oral delivery is less direct, it may have systemic effects via the gut-vagina axis. According to studies, oral probiotics might affect vaginal flora by migrating from the rectum to the vagina. According to studies, both methods can be useful, with vaginal therapy having a faster effect (2-3 days) than oral administration (7 days). The administration approach should match the specific patient conditions and infection characteristics.[4]

MECHANISMS OF ACTION OF PROBIOTICS IN MIXED VAGINAL INFECTIONS

The vaginal microbiome has been the subject of study in recent years, as understanding it may lead to new therapeutic perspectives in bacterial vaginosis and various clinical-pathological conditions of the genital tract. The human vagina is colonized by diverse microorganisms, with Lactobacillus being the most frequently isolated genus. The main Lactobacillus species include *L. crispatus*, *L. gasseri*, *L. iners* and *L. jensenii*. The genital microbiome is so complex that even the predominance of different Lactobacillus species varies according to ethnicity, hormonal levels and other factors.[6]

These vaginal lactobacilli prevent pathogen invasion by keeping their population in check. When dysbiosis occurs, the probability of bacterial vaginosis, sexually transmitted infections, vulvovaginal candidiasis increases and even the chances of human immunodeficiency virus (HIV) infection rise in people with risk factors.[7] *Vaginal eubiosis* (healthy microbiome) is characterized by Lactobacillus dominance, pH≤4.5 and the presence of lactic acid. Dysbiosis (imbalance) associated with bacterial vaginosis (BV) presents lower lactic acid concentration and higher presence of short-chain fatty acids. In the transition from eubiosis to dysbiosis, lactic acid decreases dramatically (from ~110 mM to <20 mM) while acetic, propionic, butyric and succinic acids increase. Lactic acid produced by lactobacilli appears to contribute to protection against urogenital pathogens, an important characteristic for a potential vaginal probiotic. Lactobacilli are associated with a non-inflammatory vaginal environment, while polymicrobial microbiotas (as in bacterial vaginosis or BV) are linked to a pro-inflammatory environment.[8]

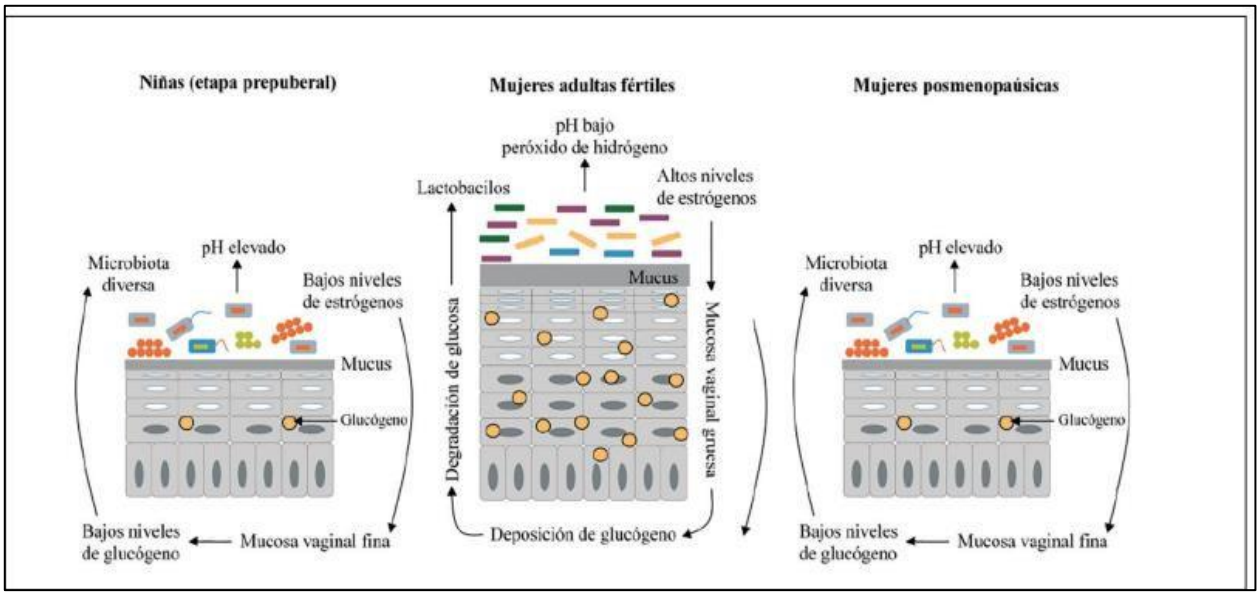

Figure 1: Vaginal microbiota at different life stages.[5]

With the decrease in Lactobacillus population, proteomic analyses of cervicovaginal fluid have linked increased inflammatory cytokines and vaginal bacterial diversity with alterations in mucin proteins, protease inhibitors and mucosal barrier proteins. A decrease in cornified envelope factors (involucrin and small proline-rich protein 1A) involved in wound healing repair is observed. This coincides with the ability of *G. vaginalis* (a bacterium associated with BV) to inhibit wound healing, something that does not occur with *L. iners*.

Certain lactobacilli reduce pro-inflammatory responses elicited by Toll-like receptor (TLR) agonists in cervicovaginal epithelial cells. Lactic acid dramatically increases the anti-inflammatory cytokine IL-1RA without significantly increasing pro-inflammatory IL-1β. Protonated lactic acid reduces the production of pro-inflammatory cytokines and chemokines stimulated by the TLR 1/2 agonist Pam3CSK4.[9] In Figure I we can observe the vaginal microbiota at different ages.

NORMAL BACTERIAL FLORA IN THE VAGINA AND CAUSES THAT ALTER ITS NORMAL MICROBIOTA

The human vagina is a complex, dynamic and carefully balanced ecosystem, composed of a diverse community of both aerobic and anaerobic bacteria. The Lactobacillus spp. genus is the predominant one and plays a key role in protecting the vaginal environment through three synergistic mechanisms: a) formation of a biofilm that blocks pathogen adhesion by covering epithelial receptors; b) generation of antimicrobial compounds: lactic acid, hydrolysis products, hydrogen peroxide and bacteriocins that contribute to maintaining an acidic vaginal pH and c) coaggregation with pathogens.

The relationship between humans and the vaginal microbiota is symbiotic in nature. While bacteria contribute to the defence of the vaginal environment against pathogens, the host supplies the nutrients necessary for its proliferation and maintenance. The community state types (CSTs) that make up the vaginal microbiota are categorized based on the amount of lactic acid they produce, a key element in the stability of the vaginal ecosystem.

These communities are made up of different species of the genus Lactobacillus, classified into five groups: CST I (*L. crispatus),* CST II (*L. gasseri*), CST III (*L. iners*), CST IV (polymicrobial microbiota composed of Lactobacillus and bacteria associated with bacterial vaginosis) and CST V (*L. jensenii*). Groups I, II and IV are the most studied and occur more frequently in healthy women, compared to types CST III and CST V.[11]

A vaginal microbiota dominated by *Lactobacillus crispatus* is considered a reliable marker of good vaginal health. In contrast, the dominant presence of *Lactobacillus iners* or *Lactobacillus jensenii* has been linked to elevated levels of proinflammatory mediators, such as colony-stimulating factor (which promotes the proliferation and phagocytic activation of monocytes and macrophages), interleukin-18, interleukin-1α and tumor necrosis factor alpha (TNF-α).

Overexpression of these elements favors an imbalance between anti-inflammatory and pro-inflammatory immune responses.[3] CST IV, in particular, has been widely associated with bacterial vaginosis and is recognized as the most common dysbiosis profile. However, its detection in women without clinical symptoms has led to scientific debates regarding its interpretation and it is still debated whether it represents a non-pathological variant of the vaginal ecosystem or an asymptomatic form of dysbiosis.[12]

Lactic acid

The vagina maintains an acidic environment with an average pH of around 3.80±0.20, thanks to the production of lactic acid by lactobacilli.[5] Lactic acid is a metabolite generated by the fermentation of carbohydrates present in the vaginal epithelium, which contributes to the acidification of the local environment. Its potent bactericidal activity against pathogens of the urogenital tract, including E. coli, N. gonorrhoeae and HSV-2, has been demonstrated; it also has the ability to slow and trap HIV-1 virions through the cervicovaginal mucus, *L. crispatus*, *L. jensenii* and *L. gasseri* have been observed to confer a protective effect against bacterial vaginosis through the production of lactic acid, unlike *L. iners*, which does not possess this capacity.[7] In a comparative study, it was observed that all the strains of *L. gasseri* and *L. crispatus* analyzed were able to eradicate Prevotella bivia, Gardnerella spp. and uropathogenic *Escherichia coli* strains associated with premature birth. This antimicrobial activity was achieved through direct cellular contact, which suggests a mechanism of physical competition or localized release of antimicrobial compounds.[17]

L. crispatus and *L. iners* exhibit substantial differences in their metabolic pathways. *L. crispatus* is capable of metabolizing both glucose and lactose and expresses dual-specific lactate dehydrogenase (LDH), allowing it to generate both lactic acid isoforms: D-lactate and L-lactate. In contrast, *L. iners* metabolizes glucose exclusively and its LDH enzyme only catalyzes the production of L-lactate. This difference in lactate isoform production has significant implications because the presence of D-lactate has been associated with increased protection against vaginal pathogens, which may explain the limited ability of *L. iners* to prevent genital infections.[13]

Bacteriocins

The epithelial mucosa of the female genital tract represents the first barrier, playing an important role in the defense against pathogens. Bacteriocins are antimicrobial peptides that exert an antagonistic action, interfering with the growth and proliferation of pathogenic bacteria. They are

classified into two categories: class I bacteriocins (lantibiotics) are small peptides containing lanthionine and act by forming pores in the cell membrane, causing the release of essential metabolites and enzymatic inhibition. Class II bacteriocins, on the other hand, lack lanthionine and act by inducing membrane permeabilization and leakage of intracellular components from target bacteria. It has been postulated that their production is the result of ecological competition between species of the genus Lactobacillus spp. and that their presence and interaction with pathogens are factors that affect their synthesis.[14]

Hydrogen peroxide

Species of the Lactobacillus genus lack the heme and cytochrome proteins required for terminal oxidation; instead, they use flavoproteins that synthesize hydrogen peroxide (H_2O_2) from the oxidation of lactate, pyruvate or NADH. The synthesis of H_2O_2 by these microorganisms is closely related to the availability of oxygen in the environment. Furthermore, it not only exerts antimicrobial effects by interacting with other microorganisms but also modulates the vaginal environment by influencing the mucosa, including epithelial and immune cells.[15]

Hydrogen peroxide has been observed to have an immunomodulatory effect, particularly through the regulation of inflammatory markers such as IL-1B, IL-6 and secretory leukoprotease inhibitor (SLPI), inflammatory markers of the vaginal mucosa.[15] Furthermore, H_2O_2 production by *Lactobacillus crispatus* has been associated with the inhibition of colonization by anaerobic bacteria. This effect is enhanced by a genomic iron regulation system that limits the availability of this essential micronutrient to vaginal pathogens.[16] The composition of the vaginal microbiota is modulated by a variety of factors that contribute to a dynamic and sensitive balance. Internal factors that influence it include hormonal fluctuations, pregnancy, the menstrual cycle and more. External factors such as contraceptive use, diet, douching and having multiple sexual partners also play a determining role in shaping the vaginal ecosystem. Alterations in these factors can lead to a deficient Lactobacillus spp. and the subsequent proliferation of anaerobes such as Gardnerella spp., Prevotella and Mobiluncus or aerobic species, causing two dysbiotic states: bacterial vaginosis and aerobic vaginitis. A close relationship between diet and the vaginal microbiota has been observed; a diet high in fat and deficient in micronutrients such as vitamins A, C, E, D, carotenes, folate and calcium increases the risk of bacterial vaginosis.

The relationship between the microbiota of obese and non-obese women was also analyzed, observing that obese women were more likely to have a vaginal microbiome with decreased Lactobacillus and high levels of local cytokines.[16] There is a significant and independent association between psychosocial stress and the onset and persistence of bacterial vaginosis in pregnant and non-pregnant women. The factors most frequently identified were: unfavourable living conditions, limited income, inadequate living conditions, unsafe social environment, malnutrition and interpersonal conflicts. Smoking also plays a key role in the behavior of the vaginal microbiota, revealing that smoking patients have a higher prevalence of bacterial vaginosis and premature birth compared to non-smokers.[17-20]

Table 1: Some of the studies and their results.

Autor	Study	Results
Jeng et al	30 studies	Probiotics have a significant short-term effect in the treatment of common vaginal infections in non-pregnant women. Probiotic interventions were significantly associated with a lower rate of vaginitis recurrence.
Li et al	13 studies, 1258 patients	Probiotics may have a positive effect on the treatment of BV. More data are needed.
Liu et al	18 studies 1651 patients	Compared with short-term probiotic treatment (<1 month), long-term probiotic treatment (1–3 months) produces superior beneficial results and efficacy in the treatment of BV. Compared with antibiotics, probiotics significantly decreased the recurrence rate of BV.
Chen et al	14 studies	Probiotics may play a positive role in the treatment of BV, but more robust evidence is needed.

DISCUSSION

Fungal and antibiotic resistance

Bacterial vaginosis is the most common cause of bacterial vaginosis in women of reproductive age and is associated with adverse reproductive health consequences, such as preterm birth, pelvic inflammatory disease and an increased risk of sexually transmitted infections. Although treatment is based on antibiotics such as metronidazole and clindamycin, these have high recurrence rates, attributable to the presence of biofilms, antimicrobial resistance and failure of vaginal recolonization by protective Lactobacillus species, especially *Lactobacillus crisapatus*.

In the absence of an effective long-term cure, alternative therapeutic strategies are being investigated, including the use of probiotics. Likewise, behavioral factors such as the use of hormonal contraceptives, smoking and improved diet could play a complementary role in preventing

recurrences. However, most of these interventions require scientific evidence to allow for their systematic implementation in clinical practice.[18] Vulvovaginal candidiasis and its recurrent form represent a growing therapeutic challenge, largely due to the ability of Candida, particularly *C. albicans* and *C. glabrata,* to generate biofilms that confer resistance to conventional antifungals. This article reviews the use of probiotics as emerging strategies in the prevention and treatment of these infections. It is highlighted that metabolites derived from specific Lactobacillus strains possess antimicrobial and antibiofilm properties, capable of modulating the vaginal microbiota and reinforcing the mucosal barrier against fungal colonization. Despite promising results in scientific research, therapeutic efficacy still depends on factors such as the route of administration, dose, strain used and individual characteristics of the host.[19]

Vaginal infections represent a therapeutic challenge due to the increasing resistance to fluconazole, especially in *Candida albicans* and non-albicans species such as C. glabrata and intrinsically resistant ones such as *C. krusei*. Recent studies highlight the efficacy of alternatives such as boric acid (77% success rate), oteseconazole (88%) and ibrexafungerp (70%). Resistance varies geographically, with rates ranging from 2% in Ethiopia to 52% in the USA under low pH conditions, necessitating regional strategies. Furthermore, the overuse of antifungals and antibiotics alters the vaginal microbiota, exacerbating the problem. Topical therapies, probiotics with Lactobacillus and novel antifungals (fosmanogepix, rezafungin) are emerging as promising options, although more evidence is required for their standardization. Medical education and accurate diagnosis are key to combating resistance.[20]

Use of probiotics to regenerate the vaginal microbiota. The effect of probiotics for the treatment of vaginal infections shows multiple positive evidence. Some of the studies and their results are shown in Table 1.

CONCLUSION

Vaginal infections are very common conditions that all women around the world will experience at least once in their lives. In recent years, their incidence and prevalence have increased due to multiple causes, including excessive use of vaginal douches, use of contraceptive methods such as condoms, tampons or vaginal cups and sexual practices with multiple partners. The use of antibiotics without specific criteria or diagnosis increases bacterial resistance and even plays a role in the risk of vaginal infections due to changes in the vaginal microbiota.

Probiotics have very good evidence supporting the incidence of vaginal infections. They can be administered concomitantly, before or after antibiotic treatment without reporting adverse effects and even showing significant benefits, including the reduction of bacterial vaginosis, helping the vaginal microbiota maintain its normal flora and low pH.

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