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Research Article

Antimicrobial Susceptibility Patterns of Vaginal Bacteria and *Lactobacillus* in Comparison Study

Inas Khalid Kadhim[1], Mohanad Mohsin Ahmed[2], Zuhair Hameed Abood[1]

[1] *Department of Biology, College of Science, University of Kerbala, Iraq.*

[2]*Department of Microbiology, College of Medicine, University of Kerbala, Iraq.*

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Correspondence: Inas khalid kadhim

Email: enaasalkhalde@gmail.com

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Abstract

Background: The vaginal microbiota, dominated by *Lactobacillus* species, plays a critical role in preventing infections by maintaining a low pH and inhibiting pathogens. However, antimicrobial therapies for vaginitis may inadvertently disrupt this protective flora. The study aimed to compare the antimicrobial susceptibility profiles of bacterial pathogens isolated from vaginal swabs with those of *Lactobacillus* strains from vaginal and extra-vaginal sources.

Methods: Women with vaginal discharges attending Al-Kafeel Specialized Hospital in Kerbala from March 2023 to April 2024 were enrolled. High vaginal swabs were collected from vaginitis patients. Bacterial isolates were identified using standard culture techniques and the Vitek 2 system. Eight *Lactobacillus* isolates were obtained from fermented dairy products (n=4), healthy women (n=3), and a probiotic suppository (n=1). Antimicrobial susceptibility testing was performed on bacterial and *Lactobacillus* isolates using standard methods. A side-by-side comparison of their antimicrobial resistance patterns was conducted.

Results: This study of 200 vaginal isolates revealed striking resistance disparities: while pathogenic *Staphylococci* exhibited >70% macrolide resistance (reaching 90% in Coagulase-negative *Staphylococcus*) and *Streptococci* showed 60-83.3% resistance, commensal *Lactobacilli* maintained 100% susceptibility to levofloxacin, vancomycin and tetracycline, though demonstrating complete resistance to cefoxitin (100%) and high-level aminoglycoside resistance (75-87.5%). Gram-negative pathogens displayed concerning carbapenem resistance (up to 66.7% in *Klebsiella pneumoniae*), contrasting sharply with *Lactobacilli* preserved susceptibility profile, highlighting the critical need for microbiome-conscious antibiotic selection in vaginal infections.

Conclusions: These findings highlight the potential collateral damage of antimicrobial therapy on vaginal *Lactobacilli*. Tailored antibiotic selection or probiotic adjuvants may be needed to preserve the vaginal microbiome while treating infections.

Keywords: Vaginal microbiota, Antimicrobial susceptibility, Vaginitis, Dysbiosis

Introduction

Vaginitis is a common gynecological condition affecting millions of women worldwide, frequently caused by an imbalance of vaginal microbiota (e.g., *Gardnerella vaginalis* in bacterial vaginosis), *Candida* spp. in vulvovaginal candidiasis, or *Trichomonas vaginalis* in trichomoniasis [1]. The vaginal microbiota, predominantly composed of *Lactobacillus* species, plays a crucial role in maintaining vaginal health by producing lactic acid, hydrogen peroxide, and bacteriocins, which inhibit pathogenic overgrowth [2]. However, antimicrobial agents used to treat vaginitis may inadvertently disrupt these beneficial *Lactobacilli*, potentially leading to dysbiosis and recurrent infections [3].

While antibiotics such as metronidazole, clindamycin, and antifungals like fluconazole are effective against pathogens, their impact on commensal *Lactobacillus* populations remains poorly understood [4]. Some studies suggest that certain antimicrobials may have varying degrees of *Lactobacilli* susceptibility, potentially altering vaginal microbiota composition and increasing the risk of recurrence [5]. While resistance patterns of

vaginal pathogens are well-documented, data on the susceptibility of commensal *Lactobacillus* to these drugs remain limited, especially for strains from non-vaginal niches that might serve as probiotics. Given the rising concerns over antimicrobial resistance and the importance of maintaining a healthy vaginal microbiome, a comparative assessment of the susceptibility of vaginal *Lactobacilli* to commonly prescribed antimicrobials is essential.

Recent studies highlight *Lactobacillus* species' crucial role in maintaining microbial balance through competitive exclusion and pathogen inhibition. For instance, *Lactobacillus crispatus* secretes biosurfactants that disrupt *Gardnerella vaginalis* biofilms [6]. These beneficial bacteria also modulate immune responses by downregulating pathogen-induced inflammation [7]. However, antimicrobial treatments may inadvertently disrupt these protective mechanisms, as evidenced by clindamycin exposure inducing resistance genes in *Lactobacillus jensenii* that could potentially transfer to pathogens like *Streptococcus agalactiae* [8].

This study aimed to compare the antimicrobial susceptibility profiles of bacterial pathogens isolated from vaginal swabs with those of *Lactobacillus* strains from vaginal and extra-vaginal sources. By identifying agents with minimal detrimental effects on *Lactobacilli*, this research may guide more microbiome-friendly therapeutic strategies, ultimately improving treatment outcomes and reducing recurrence rates.

Materials and Methods

Sampling collection

A total of 200 swabs were collected from female patients with vaginitis attending Al-Kafeel Specialized Hospital in Kerbala in Iraq, between March 2023 and April 2024. Within age of 18-45 years, high vaginal swabs were collected using sterile cotton swab (Himedia, India) by a nurse under the supervision of the attending Gynecologist. The swabs were immediately placed into Stuart's transport media (Himedia, India) and transported to the laboratory at room temperature within 5-6 hours. The swabs, then, were processed for the isolation and identification of bacteria using standard culture techniques and the VITEK 2 compact system. Additionally, eight *Lactobacillus* isolates were obtained from fermented dairy products (n=4), healthy women (n=3), and a probiotic suppository (n=1).

Inclusion criteria

The study population comprised female patients aged 18-45 years (reproductive age group) exhibiting clinical symptoms of vaginitis, including abnormal vaginal discharge, pruritus, burning sensation, malodorous discharge, or dysuria. Eligible participants had no history of antibiotic, antifungal, or topical vaginal treatment within four weeks before enrollment.

Exclusion criteria

Participants were excluded if they were pregnant, immunocompromised, or if specimens were improperly handled (transport delay exceeding six hours or improper storage conditions). For the healthy control group (sources of *Lactobacillus* isolates), exclusion criteria included: recent vaginal infections, antibiotic use within four weeks, menopausal status, and current use of hormonal contraceptives.

Isolation of *Lactobacillus* spp. from fermented dairy

Lactobacillus species (*Lactobacillus casei, Lactobacillus plantarum, Lactobacillus helveticus, and Lactobacillus paracasei*) were isolated from fermented milk by preparing serial dilutions (10^{-1} to 10^{-7}) in sterile distilled water. A 0.1 mL aliquot from the final dilutions was spread on pre-dried MRS agar (HiMedia, India) and incubated anaerobically at 37°C for 48 hours. Distinct colonies were purified by streaking on fresh MRS agar and re-incubated under the same conditions [9].

Isolation of vaginal *Lactobacillus* spp. from healthy women

Mid-vaginal swabs from pre-menopausal women (18–45 years, no infections/antibiotic use) were plated on MRS agar with 0.1% cysteine. Incubation was carried out at 28–30°C for 24–48 hours under anaerobic conditions (*Lactobacillus rhamnosus, Lactobacillus acidophilus, Lactobacillus reuteri*) [10].

Isolation of *Lactobacillus* spp. from vaginal probiotic suppositories

Probiotic suppositories (*Lactobacillus crispatus*) were dissolved in sterile saline, serially diluted, and plated on MRS agar. After anaerobic incubation (37°C, 24–48 hours), colonies were purified via streaking [11]. Preliminary identification involves performing Gram staining, where *Lactobacillus* spp. appear as Gram-positive rods. Additionally, a catalase test is conducted, and a negative result confirms the presence of *Lactobacillus* spp. [12]. For confirmation, the VITEK 2 ANC system card allows for identifying Lactobacilli [11].

Antimicrobial susceptibility test

The antimicrobial susceptibility testing was conducted using the Kirby-Bauer disk diffusion method following CLSI (2024) guidelines. Mueller-Hinton agar plates were prepared as per the manufacturer's instructions (Himedia, India). Bacterial inocula were adjusted to a 0.5 McFarland standard (1.5×10^8 CFU/ml) using normal saline and applied to the plates within 30 minutes. A sterile swab was dipped into the inoculum, excess liquid was removed, and the agar surface was evenly swabbed in three directions, including the edges, to ensure uniform bacterial growth. After allowing the inoculum to dry, antibiotic disks were placed on the agar using sterile forceps and gently pressed down. The plates were then incubated at 37°C for 18–24 hours. Following incubation, the zones of inhibition were measured with a ruler, and the results were interpreted as resistant, intermediate, or sensitive based on CLSI (2024) breakpoints [9].

Susceptibility of *Lactobacillus* spp. to antibiotics

The antibiotic susceptibility profiles of the Lactobacillus strains were determined by using the modified agar diffusion method of Clinical and Laboratory Standards Institute [14]. Lactobacilli were grown in MRS broth for 18 h at 37°C in an anaerobic jar and then centrifuged (8000 × g, 10 min). The cell pellets were washed twice with 0.9% saline solution (w/v) and adjusted to 0.5 MacFarland. Ten, 100 µL of this suspension was spread on MRS agar plates, and antibiotic disks were placed on the plates. The plates were incubated at 37 °C for 24 hr., and then the diameter of the zone of inhibition surrounding each disk was measured and classified as sensitive (S), intermediate (I), or resistant (R), according to [14-15].

Antibacterial disks were purchased from different manufacturers such as Oxoid (UK/USA), BD BBL (USA), BioMérieux (France), HiMedia (India), or Liofilchem (Italy). These disks, including clindamycin (CLI-2µg), linezolid (LZD-30µg), doxycycline (DOX-30µg), erythromycin (ERY-15µg), cefoxitin (FOX-30µg), azithromycin (AZM-30µg), clarithromycin (CLR-15µg), ciprofloxacin (CIP-5µg), gentamicin (GEN-10µg), trimethoprim-sulfamethoxazole (SXT-25µg), levofloxacin (LVX-5µg), penicillin (PEN-10U), tetracycline (TET-30µg), ofloxacin (OFX-5µg), vancomycin (VAN-5µg), amikacin (AMK-30µg), teicoplanin (TEC-30µg), amoxicillin-clavulanic acid (AMC-30µg), fosfomycin (FOF-200µg), ceftriaxone (CRO-30µg), imipenem (IPM-10µg), ampicillin (AMP-25µg), piperacillin-tazobactam (TZP-110µg), meropenem (MEM-10µg), cefepime (FEP-30µg), chloramphenicol (CHL-30µg), ceftazidime (CAZ-30µg), cefixime (CFM-5µg), and cefotaxime (CTX-30µg),

Ethical approval

This research was subjected to ethical considerations, and the research was approved by the Committee of Ethical Standards in the College of Science, University of Kerbala. (Approval number 005CSE and dated 24 Sept. 2024), and Karbala Health Directorate on number 44 and dated 7 February 2023.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS 23.0) was used to enter and evaluate the data from the current study. The Fisher's exact test was used for statistical comparison of groups; values < 0.05 were regarded as significant [16].

Results

A total of 239 microbial isolates were recovered in this study. The distribution of bacterial isolates from vaginal samples showed *Staphylococcus aureus* (26.8%), *Escherichia coli* (17.6%), and coagulase-negative *Staphylococci* (17.6%) as the most prevalent pathogens. Other notable isolates included *Streptococcus agalactiae* (8.4%), *Enterococcus faecalis* (7.5%), and *Klebsiella pneumoniae* (6.3%). Less common but clinically relevant organisms such as *Pseudomonas aeruginosa* (2.5%), *Acinetobacter* spp. (2.1%), and *Gardnerella vaginalis* (0.8%) were also identified (Table 1).

Table 2 showed the antibiotic susceptibility patterns of *Staphylococcus aureus* (n=64) and coagulase-negative *Staphylococcus* (CoNS, n=42) isolates, revealing concerning resistance trends. Both groups exhibited alarmingly high resistance to macrolides, while *Staphylococcus aureus* showed 89.1% resistance to erythromycin and 90.6% to azithromycin, and the CoNS demonstrated even higher resistance (90.5% and 95.2%, respectively). Clindamycin resistance was prevalent in both *Staphylococcus aureus* (57.8%) and CoNS (71.42%), while clarithromycin resistance was slightly lower (78.1% and 69%, respectively). Fluoroquinolone resistance was substantial, with *Staphylococcus aureus* displaying 51.6% resistance to ciprofloxacin and 65.6% to ofloxacin, whereas CoNS isolates showed even higher resistance (76.2% and 76.2%, respectively). Penicillin resistance was notably high in both groups (68.75% in *Staphylococcus aureus* and 88% in CoNS), while tetracycline maintained relatively better activity (37.5% and 35.7% resistance, respectively). Trimethoprim-

sulfamethoxazole (SXT) resistance was observed in 70.31% of *Staphylococcus aureus* and 78.6% of CoNS isolates. Glycopeptides (vancomycin and teicoplanin) remained the most effective, with susceptibility rates exceeding 59% in both groups, though vancomycin resistance reached 25% in *Staphylococcus aureus* and 35.7% in CoNS.

Table 3 showed the antibiotic susceptibility profiles of various *Streptococcal* species (n=40) and *Enterococcus faecalis* (n=18), which revealed significant variations in resistance patterns. Among β-hemolytic *Streptococci*, *Streptococcus agalactiae* (n=20) showed particularly high resistance to macrolide antibiotics, with clarithromycin showing the highest resistance rate (75%), followed by azithromycin (65%) and erythromycin (60%). Clindamycin resistance mirrored erythromycin at 60%. The isolates showed substantial resistance to penicillin (60%) and tetracycline (75%), which is concerning as these are traditionally first-line treatments for GBS infections. Trimethoprim-sulfamethoxazole (SXT) displayed moderate activity (50% resistance), while fluoroquinolones showed variable efficacy. Ofloxacin maintained the best activity (45% resistance) compared to levofloxacin (50% resistance). Teicoplanin demonstrated the highest susceptibility (60% susceptible), though 40% resistance still presents clinical concerns.

The antibiotic susceptibility testing of viridans group *Streptococci* revealed significant species-specific resistance patterns, with *Streptococcus salivarius* (n=4) demonstrating the highest resistance rates (75% to macrolides, fluoroquinolones, and tetracycline), followed by *Streptococcus gordonii* (n=3; 66.7% resistance to most agents), while *Streptococcus sanguinis* (n=4) maintained relatively better susceptibility (50% resistance to key antibiotics). All three species showed complete concordance in their resistance profiles across erythromycin, azithromycin, and clarithromycin (50-75% resistance). Notably, penicillin susceptibility varied substantially between species (25-75% resistance), with *Streptococcus sanguinis* remaining most susceptible (50%) and *Streptococcus gordonii* most resistant (66.7%). The isolates displayed particularly concerning resistance to levofloxacin (*Streptococcus salivarius* 75%, *Streptococcus gordonii* 33.3%, *Streptococcus sanguinis* 50%) and tetracycline (*Streptococcus salivarius* 75%, others 50-66.7%), while trimethoprim-sulfamethoxazole showed moderate activity across all species (50-66.7% resistance).

The analysis of *Streptococcus alactolyticus* (n=3) and other *Streptococcus* spp. (n=6) revealed significant resistance patterns. Both groups showed high macrolide resistance (66.7-83.3%), with *Streptococcus alactolyticus* exhibiting universal levofloxacin resistance (100%) versus 66.7% in other strains. Penicillin resistance was consistently high (66.7% in both groups), while trimethoprim-sulfamethoxazole showed better activity against non-alactolyticus strains (33.3% vs 66.7% resistance). Tetracycline resistance was higher in *Streptococcus alactolyticus* (66.7% vs 50%). These findings highlight concerning resistance trends among vaginal *Streptococci*.

Enterococcus faecalis isolates demonstrated particularly high resistance to erythromycin (83.3%), levofloxacin (66.7%), and ciprofloxacin (72.2%), while vancomycin maintained good activity against *Enterococcus faecalis* (61.1% susceptible); resistance was still notable (11.1%). Fosfomycin showed limited effectiveness against *Enterococcus faecalis*, with only 22.2% susceptibility.

Table 4 reveals concerning resistance patterns in key vaginal Enterobacteraceae, with *Escherichia coli* showing high resistance to trimethoprim-sulfamethoxazole and cefixime (54.8% each), *Klebsiella pneumoniae* exhibiting alarming carbapenem resistance (66.7% to imipenem) despite meropenem susceptibility (93%), and *Proteus mirabilis* demonstrating universal tetracycline resistance but retained sensitivity to piperacillin-tazobactam and ceftriaxone (100%). While the lone *Enterobacter* spp. isolate resisted β-lactams, it remained susceptible to carbapenems, fluoroquinolones, and aminoglycosides, underscoring both regional resistance threats and remaining therapeutic options for Gram-negative vaginal infections.

Table 5 showed the antibiotic susceptibility profiles of *Pseudomonas aeruginosa* (n=6) and *Acinetobacter* spp. (n=5), which revealed differences in resistance patterns. *Pseudomonas aeruginosa* showed excellent susceptibility to meropenem and piperacillin-tazobactam (100% each), while demonstrating moderate susceptibility to ciprofloxacin (66.7%) and levofloxacin (83.3%). In contrast, *Acinetobacter* spp. exhibited universal susceptibility to ciprofloxacin, levofloxacin, and trimethoprim-sulfamethoxazole (100% each), but concerning resistance to gentamicin (60%), ceftazidime (60%), and tetracycline (60%). Both pathogens maintained good carbapenem susceptibility (80-100%), though *Acinetobacter*

spp. showed emerging resistance to cephalosporins (40-60% resistance).

Table 1: Type and frequency of isolated microorganisms.

Types of bacteria	Number	Percentage
Staphylococcus aureus	64	26.8%
Escherichia coli	42	17.6%
Coagulase-negative *staphylococcus*	42	17.6%
Streptococcus agalactiae	20	8.4%
Enterococcus faecalis	18	7.5%
Klebsellia pneumoniae	15	6.3%
Psedomonas aeruginosa	6	2.5%
***Streptococcus* spp.**	6	2.5%
***Acinetobacter* spp.**	5	2.1%
Streptococcus salivaris	4	1.7%
Streptococcus sanguinis	4	1.7%
Proteus mirablis	4	1.7%
Strptococcus alactolyticus	3	1.3%
Streptococcus gordonii	3	1.3%
Gardenella vaginosis	2	0.8%
***Enterbacter* spp.**	1	0.4%
Total No.	239	100%

Table 2: Antibiotic susceptibility pattern of *Staphylococcus aureus* and coagulase-negative *Staphylococcus*

Antibiotic tested	Concentration (µg)	*S. aureu* (n=64)			*CoN-Staphylococci* (n=42)		
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)
Clindamycin (CLI)	2	27 (42.2%)	0 (0%)	37 (57.8%)	11 (26.2%)	1 (2.4%)	30 (71.42%)
Erythromycin (ERY)	15	6 (9.4%)	1 (1.6%)	57 (89.1%)	0 (0%)	4 (9.5%)	38 (90.5%)
Azitromycin (AZM)	15	6 (9.4%)	0 (0%)	58 (90.6%)	0 (0%)	2 (4.8%)	40 (95.2%)
Clarithomycin (CLR)	15	11 (17.2%)	3 (4.7%)	50 (78.1%)	7 (16.7%)	6 (14.3%)	29 (69%)
Ciprofloacin (CIP)	5	26 (40.6%)	5 (7.8%)	33 (51.6%)	8 (19%)	2 (4.8%)	32 (76.2%)
Gentamicin (GEN)	10	30 (46.9%)	5 (7.8%)	29 (45.3%)	12 (58.6%)	2 (4.8%)	28 (66.7%)
Trimethoprim-sulfamethoazole (SXT)	25	18 (28.1%)	1 (1.6%)	45 (70.31%)	8 (19%)	1 (2.4%)	33 (78.6%)
Levofloxacin (LVX)	5	31 (48.4%)	8 (12.5%)	25 (39.1%)	13 (30.9%)	4 (9.5%)	25 (59.5%)
Penicillin (PEN)	10 unit	20 (31.3%)	0 (0%)	44 (68.75%)	5 (11.9%)	0 (0%)	37 (88%)
Tetracycline (TET)	30	33 (51.6%)	7 (10.9%)	24 (37.5%)	19 (45.2%)	8 (19%)	15 (35.7%)
Ofloxacin (OFX)	5	22 (34.4%)	0 (0%)	42 (65.6%)	9 (21.4%)	1 (2.4%)	32 (76.2%)
Vancomycin (VAN)	MIC	42 (65.6%)	6 (9.4%)	16 (25%)	25 (59.5%)	2 (4.8%)	15 (35.7%)
Teicoplanin (TEC)	MIC	38 (59.4%)	8 (12.5%)	18 (28.1%)	26 (62%)	3 (7.14%)	13 (30.9%)

S: Sensitive, I: intermediate sensitivity, R: resistance

Table 6 showed the antibiotic susceptibility testing of *Lactobacillus* isolates from fermented dairy (n=4), healthy vaginal samples (n=3), and probiotic suppositories (n=1), which revealed that all strains (100%) were susceptible to levofloxacin, vancomycin, tetracycline, ampicillin, and chloramphenicol, making these antibiotics optimal for preserving beneficial flora. However, all isolates demonstrated complete resistance (100%) to cefoxitin and high resistance to aminoglycosides (amikacin 87.5%, gentamicin 75%), while showing variable susceptibility to erythromycin (50% susceptible, 50% intermediate) and clindamycin (25% susceptible, 37.5% intermediate/resistant).

Table 3: Antibiotic susceptibility pattern of *Streptococcus* spp. and *Enterococcus faecalis*.

Antibiotic tested	Disc conc. (µg)	β-hemolytic group			*Viridians -group*									*Non enterococcus*			*Streptococcus* spp (n=6)			*E. Feacalis* (n=18)		
		S. agalactiae (n=20)			*S. sanguinis* (n=4)			*S. gordonii* (n=3)			*S. salivarins* (n=4)			*S. alactolyticus* (n=3)								
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R(%)	S (%)	I (%)	R(%)	S (%)	I (%)	R(%)	S (%)	I (%)	R(%)	S (%)	I (%)	R(%)
Clindamycin (CLI)	2	5 (25%)	3 (15%)	12 (60%)	2 (50%)	1 (25%)	1 (25%)	2 (66.7%)	0 (0%)	1 (33.3%)	1 (25%)	0 (0%)	3 (75%)	1 (33.3%)	0 (0%)	2 (66.7%)	2 (33.3%)	0 (0%)	4 (66.7%)	---	---	---
Erythromycin (ERY)	15	5 (25%)	3 (15%)	12 (60%)	1 (25%)	1 (25%)	2 (50%)	1 (33.3%)	0 (0%)	2 (66.7%)	1 (25%)	0 (0%)	3 (75%)	1(33.3%)	1(33.3%)	1 (33.3%)	1(16.7%)	0 (0%)	5(83.3%)	2(11.1%)	1(5.6%)	15(83.3%)
Azitromycin (AZM)	15	7 (35%)	0 (0%)	13 (65%)	1(25%)	1(25%)	2 (50%)	1(33.3%)	0 (0%)	2 (66.7%)	1(25%)	0 (0%)	3 (75%)	1(33.3%)	0 (0%)	2 (66.7%)	1(16.7%)	0 (0%)	5 (83.3%)	---	---	---
Clarithomycin(CLR)	15	5 (25%)	0 (0%)	15 (75%)	1 (25%)	1 (25%)	2 (50%)	1 (33.3%)	0 (0%)	2 (66.7%)	1 (25%)	0 (0%)	3 (75%)	0 (0%)	1 (33.3%)	2 (66.7%)	1 (16.7%)	1 (16.7%)	4 (66.7%)	---	---	---
Trimethoprimsulfamethoazole (SXT)	25	6 (30%)	4 (20%)	10 (50%)	2 (50%)	0 (0%)	2 (50%)	1 (33.3%)	0 (0%)	2 (66.7%)	2 (50%)	0 (0%)	2(50%)	1 (33.3%)	0 (0%)	2 (66.7%)	2 (33.3%)	2 (33.3%)	2(33.3%)	---	---	---
Levofloxacin(LVX)	5	8 (40%)	2 (10%)	10 (50%)	2 (50%)	0 (0%)	2 (50%)	2 (66.7%)	0 (0%)	1 (33.3%)	1 (25%)	0 (0%)	3 (75%)	0 (0%)	0 (0%)	3 (100%)	2 (33.3%)	0 (0%)	4 (66.7%)	5(27.8%)	1(5.6%)	12(66.7%)
Penicillin (PEN)	MIC	8 (40%)	0 (0%)	12 (60%)	2 (50%)	1 (25%)	1 (25%)	1(33.3%)	0 (0%)	2 (66.7%)	3 (75%)	0 (0%)	1 (25%)	1 (33.3%)	0 (0%)	2 (66.7%)	2 (33.3%)	0 (0%)	4 (66.7%)	8(88.4%)	0 (0%)	10(55.5%)
Tetracycline (TET)	30	5 (25%)	0 (0%)	15(75%)	2 (50%)	0 (0%)	2 (50%)	1 (33.3%)	0 (0%)	2(66.7%)	0 (0%)	1 (25%)	3 (75%)	0 (0%)	1 (33.3%)	2 (66.7%)	2 (33.3%)	1 (16.7%)	3 (50%)	0(0.0%)	8(88.4%)	10(55.5%)
Ofloxacin (OFX)	5	8 (40%)	3 (15%)	9 (45%)	1(25%)	1 (25%)	2 (50%)	1 (33.3%)	0 (0%)	2 (66.7%)	0 (0%)	1 (25%)	3 (75%)	1 (33.3%)	0 (0%)	2 (66.7%)	1 (16.7%)	1 (16.7%)	4 (66.7%)	---	---	---
Teicoplanin (TEC)	MIC	12 (60%)	0 (0%)	8 (40%)	1 (25%)	1 (25%)	2 (50%)	---	---	---	---	---	---	0 (0%)	1 (33.3%)	2 (66.7%)	---	---	---	11(61.1%)	2(11.1%)	5(27.8%)
Ciprofloacin (CIP)	5	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	3 (16.7%)	2 (11.1%)	13(72.2%)
Vancomycin(VAN)	5	---	---	---	---	---		---	---	---	---	---	---	---	---	---	---	---	---	11(61.1%)	5 (27.8%)	2 (11.1%)
Fosfomycin(FOF)	200	---	---	---	---	---		---	---	---	---	---	---	---	---	---	---	---	---	4(22.2%)	6(33.3%)	8 (44.4%)

S: Sensitive, I: intermediate sensitivity, R: resistance

Table 4: Antibiotic susceptibility pattern of Enterobacteraceae.

Antibiotic tested	Disk conc. (µg)	*E. coli* (n=42)			*Klebsellia pneumoniae* (n=15)			*Proteus mirabilis* (n=4)			*Enterobacter* spp. (n=1)		
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R(%)
Ciprofloacin (CIP)	5	26 (61.9%)	2 (4.8%)	14 (33.3%)	6 (40%)	4 (26.7%)	5 (33.3%)	2 (50%)	0 (0%)	2 (50%)	0 (0%)	1 (100%)	0 (0%)
Gentamicin (GEN)	10	23 (54.8%)	7 (16.7%)	12 (28.6%)	9 (60%)	1 (6.7%)	5 (33.3%)	1 (25%)	2 (50%)	1 (25%)	0 (0%)	1 (100%)	0 (0%)
Trimethoprim-sulfamethoazole (SXT)	25	17 (40.5%)	2 (4.8%)	23 (54.8%)	3 (20%)	0 (0%)	12 (80%)	1 (25%)	0 (0%)	3 (75%)	1 (100%)	0 (0%)	0 (0%)
Levofloxacin (LVX)	5	25 (59.5%)	1 (2.4%)	16 (38.1%)	12 (80%)	1 (6.7%)	2 (13.3%)	2 (50%)	2 (50%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)
Tetracycline (TET)	30	15 (35.7%)	5 (11.9%)	22 (52.4%)	9 (60%)	0 (0%)	6 (40%)	0 (0%)	0 (0%)	4 (100%)	1 (100%)	0 (0%)	0 (0%)
Ofloxacin (OFX)	5	16 (38.1%)	5 (11.9%)	21 (50%)	10(66.7%)	0 (0%)	5(33.3%)	1 (25%)	0 (0%)	3 (75%)	1 (100%)	0 (0%)	0 (0%)
Amikacin(AMK)	30	25 (59.5%)	8 (19%)	9 (21%)	5 (33.3%)	6 (40%)	4 (26.7%)	2 (50%)	1 (25%)	1 (25%)	0 (0%)	1 (100%)	0 (0%)
Amoxicillin-clavulanic acid(AMC)	30	20 (47.6%)	5 (11.9%)	17 (40.5%)	8 (53%)	1 (6.7%)	6 (40%)	3 (75%)	0 (0%)	1 (25%)	0 (0%)	0 (0%)	1 (100%)
Fosfomycin (FOF)	200	22(52.38%)	6 (14.3%)	14 (33.33%)	---	---	---	2 (50%)	0 (0%)	2 (50%)	0 (0%)	0 (0%)	
Ceftriaone (CRO)	30	22(52.4%)	3 (7.1%)	17 (40.5%)	8 (53%)	0 (0%)	7 (46.7%)	4(100%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)
Cefixime(CFM)	5	15 (35.7%)	4 (9.5%)	23 (54.8%)	6 (40%)	0 (0%)	9 (60%)	3 (75%)	0 (0%)	1 (25%)	0 (0%)	1 (100%)	0 (0%)

Antibiotic tested	Disk conc. (µg)	*E. coli* (n=42)			*Klebsellia pneumoniae* (n=15)			*Proteus mirabilis* (n=4)			*Enterobacter* spp. (n=1)		
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R(%)
Cefotaime (CTX)	30	17 (40.5%)	0 (0%)	25 (59.5%)	3 (20%)	2 (13.3%)	10 (66.7%)	3 (75%)	0 (0%)	1 (25%)	0 (0%)	1 (100%)	0 (0%)
Cefepime (FEP)	30	15 (35.7%)	7 (16.7%)	20 (47.6%)	2 (13.3%)	4 (26.7%)	9 (60%)	2 (50%)	0 (0%)	2 (50%)	0 (0%)	1 (100%)	0 (0%)
Imipenem (IPM)	10	25 (59.5%)	6 (14.3%)	11 (26.2%)	4 (26.7%)	1 (6.7%)	10 (66.7%)	2 (50%)	0 (0%)	2 (50%)	0 (0%)	0 (0%)	0 (0%)
Ceftrazidime (CAZ)	30	15(35.7%)	1 (2.4%)	26 (62%)	3 (20%)	3 (20%)	9 (60%)	4(100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Meropenem (MEM)	10	38 (90.5%)	0 (0%)	4 (9.5%)	14 (93%)	0 (0%)	1 (6.7%)	3 (75%)	0 (0%)	1 (25%)	1 (100%)	0 (0%)	0 (0%)
Chloramphenicol (CHL)	30	22 (52.4%)	8 (19%)	12 (28.6%)	---	---	---	1 (25%)	0 (0%)	3 (75%)	0 (0%)	0 (0%)	0 (0%)
Piperacillin - Tazobactam (TZP)	110	24 (57%)	8 (19%)	10 (23.8%)	0 (0%)	7 (46.7%)	8(53%)	4(100%)	0 (0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)
Doycycline (DOX)	30	---	---	---	---	---	---	---	---	---	1 (100%)	0 (0%)	0 (0%)

S: Sensitive, I: intermediate sensitivity, R: resistance

Table 5: Antibiotic susceptibility pattern of *Pseudomonas aeruginosa* and *Acinetobacter* spp.

Antibiotic tested	Disc conc. (µg)	*Pseudomonas aeruginosa* (n=6)			*Acinetobacter* spp. (n=5)		
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)
Ciprofloacin (CIP)	5	4(66.7%)	0 (0%)	2(33.3%)	5(100%)	0 (0%)	0 (0%)
Gentamicin (GEN)	10	3(50%)	1(16.7%)	2(33.3%)	1(20%)	1(20%)	3(60%)
Levofloxacin (LVX)	5	5(83.3%)	1(16.7%)	0 (0%)	5(100%)	0 (0%)	0 (0%)
Ofloxacin(OFX)	5	2(33.3%)	0 (0%)	4(66.7%)	---	---	---
Amikacin(AMK)	30	4(66.7%)	0 (0%)	2(33.3%)	4(80%)	0 (0%)	1(20%)
Cefepime(FEP)	30	3(50%)	1(16.7%)	2(33.3%)	2(40%)	1(20%)	2(40%)
Impenem(IPM)	10	4(66.7%)	0 (0%)	2(33.3%)	4(80%)	0 (0%)	1(20%)
Ceftrazidime (CAZ)	30	3(50%)	1(16.7%)	2(33.3%)	2(40%)	0 (0%)	3(60%)
Meropenem (MEM)	10	6(100%)	0 (0%)	0 (0%)	4(80%)	0 (0%)	1(20%)
Piperacillin-Tazobactam (TZP)	110	6(100%)	0 (0%)	0 (0%)	4(80%)	0 (0%)	1(20%)
Trimethoprim-sulfamethoazole (SXT)	25	---	---	---	5(100%)	0 (0%)	0 (0%)
Tetracycline (TET)	30	---	---	---	2(40%)	0 (0%)	3(60%)
Ceftriaxone (CRO)	30	---	---	---	3(60%)	0 (0%)	2(40%)
Cefotaxime (CTX)	30	---	---	---	2(40%)	0 (0%)	3(60%)

S: Sensitive, I: intermediate sensitivity, R: resistance

The probiotic *Lactobacillus crispatus* strain displayed broader antibiotic susceptibility compared to other isolates, while dairy-derived *Lactobacillus plantarum* and *Lactobacillus paracasei* showed particular resistance to clindamycin. These findings demonstrate that fluoroquinolones, glycopeptides, and tetracyclines are the safest choices when aiming to protect lactobacilli during antimicrobial therapy, whereas cephalosporins and aminoglycosides should be avoided due to their detrimental effects on these beneficial microorganisms. The data provides crucial guidance for selecting antibiotics that effectively target pathogens while minimizing disruption to protective vaginal and intestinal microbiota.

This study reveals significant differences in antibiotic susceptibility patterns between clinical (vaginal and probiotic) and environmental (fermented dairy) *Lactobacillus* strains. While all strains showed universal susceptibility to levofloxacin, vancomycin, tetracycline, ampicillin, and chloramphenicol, clinical isolates exhibited greater resistance variability compared to environmental strains.

Table 7 showed the comparative analysis reveals key differences in antibiotic susceptibility between vaginal pathogens and protective *Lactobacilli*, guiding microbiome-sparing therapy. Gram-positive pathogens (*Staphylococcus aureus,* Coagulase-negative *Staphylococcus, Streptococcus agalactiae, Enterococcus faecalis, Streptococcus salivaris, Streptococcus sanguinis, Strptococcus alactolyticus, Streptococcus gordonii, Gardenella vaginosis*) showed critical resistance to erythromycin (82.31%), clindamycin (62.65%), and trimethoprim-sulfamethoxazole (66.46%), while Gram-negatives (*Escherichia coli, Klebsellia pneumonia, Psuedomonas aeruginosa, Acinetobacter* spp*., Proteus mirablis, Enterbacter* spp.) were resisted to cephalosporins (cefixime 53.42%, cefotaxime 56.16%) and tetracycline (53.42%). Crucially, *Lactobacilli* maintain 100% susceptibility to levofloxacin, vancomycin, tetracycline, ampicillin, and chloramphenicol, but exhibit complete resistance to cefoxitin and high resistance to aminoglycosides (amikacin 87.5%, gentamicin 75%).

Table 6: The antimicrobial susceptibility test (AST) of *Lactobacillus* species against various antibiotics.

Isolated sources		**fermented dairy(n=4)**				**Vaginal Healthy Women(n=3)**			**Probiotic Suppositories(n=1)**	**NO. of sensitive**	**NO. of resistance**	**NO .of intermediate**
Antibiotic tested	**Disc conc (µg)**	***L.casei***	***L.plantaurm***	***L.helyticus***	***L.paracasei***	***L.rhmanosus***	***L.reuteri***	***L.acidophilus***	***L.crispatus***			
Ciprofloxacin (CIP)	5	I	S	S	S	S	S	S	S	7 (87.5%)	0 (0%)	1 (12.5%)
Clindamycin (CLI)	2	S	R	S	R	I	I	R	I	2 (25%)	3 (37.5%)	3 (37.5%)
Amikacin (AMK)	30	R	R	R	R	R	R	R	S	1 (12.5%)	7 (87.5%)	1 (12.5%)
Gentamicin (GEN)	10	S	R	R	R	R	R	R	S	2(25%)	6 (75%)	0 (0%)
Levofloxacin (LVX)	5	S	S	S	S	S	S	S	S	8 (100%)	0 (0%)	0 (0%)
Amoxicillin-clavulanate (AMC)	30	S	S	I	S	S	I	I	S	5 (62.5%)	0 (0%)	3 (37.5%)
Vancomycin (VAN)	30	S	S	S	S	S	S	S	S	8 (100%)	0 (0%)	0 (0%)
Trimethoprim-sulfamethoxazole (SXT)	25	R	S	S	S	S	S	S	S	7 (87.5%)	1 (12.5%)	0 (0%)
Tetracycline (TET)	30	S	S	S	S	S	S	S	S	8 (100%)	0 (0%)	0 (0%)
Erythromycin (ERY)	15	S	S	S	I	I	I	I	S	4(50%)	0 (0%)	4 (50%)
Ampicillin (AMP)	10	S	S	S	S	S	S	S	S	8 (100%)	0 (0%)	0 (0%)
Cefixime (CFM)	5	R	S	R	R	R	I	R	S	2 (25%)	5 (62.5%)	1 (12.5%)
Chloramphenicol (CHL)	30	S	S	S	S	S	S	S	S	8 (100%)	0 (0%)	0 (0%)
Ceftriaxone (CRO)	30	I	S	R	R	I	I	I	I	1 (12.5%)	2 (25%)	5 (62.5%)
Cefotaxime (CTX)	30	I	S	I	R	R	R	R	S	2	4	2

										(25%)	(50%)	(25%)
Cefoxitin (FOX)	30	R	R	R	R	R	R	R	R	0 (0%)	8 (100%)	0 (0%)

S: Sensitive, I: intermediate sensitivity, R: resistance

The antibiotic susceptibility analysis revealed striking differences between *Lactobacilli* and pathogenic bacteria, with *Lactobacilli* demonstrating significantly higher susceptibility to antibiotics including levofloxacin (100% vs 68.49% in G^{-ve} pathogens), vancomycin (100% vs 65.85%in G^{+ve} pathogens), and ciprofloxacin (87.5% vs 58.90% in G^{-ve} pathogens), Amoxicillin-clavulanate (62.5% vs 100% in G^{+ve},47.94% in G^{-ve}), while Chloramphenicol (100% vs 52.63% in G^{-ve} pathogens).

All tested antibiotics showed statistically significant differences in resistance patterns ($p<0.05$) between compared groups (G^{+ve}, G^{-ve}, and *Lactobacilli*) as determined by Fisher's exact test. The extremely low p-values (<0.0001 for most comparisons) indicate these resistance profile differences are highly significant and unlikely due to chance.

Table 7: Comparative antimicrobial susceptibility of vaginal pathogens vs. Lactobacilli: implications for microbiome-sparing therapy.

Antibiotic (Disc- conc .µg)	**G+ve (n=164)**			**G-ve (n=73)**			***Lactobacilli* (n=8)**			**Groups Compared**	**p-value (Fisher's Test)**
	S (%)	**I (%)**	**R (%)**	**S (%)**	**I (%)**	**R (%)**	**S (%)**	**I (%)**	**R (%)**		
Ciprofloxacin (CIP-5)	53 (32.31%)	11 (6.70%)	100 (60.97%)	43 (58.90%)	7 (9.58%)	23 (31.50%)	7 (87.5%)	1 (12.5%)	0 (0%)	G^{+ve}, G^{-ve}, Lactob.	< 0.0001
Clindamycin (CLI-2)	55 (33.13%)	7 (4.21%)	104 (62.65%)	---	---	---	2 (25%)	3 (37.5%)	3 (37.5%)	G^{+ve}, Lactob.	0.0002
Amikacin (AMK-30)	80 (48.70%)	17 (10.36%)	67 (40.85%)	40 (54.79%)	16 (21.91%)	17 (23.28%)	1 (12.5%)	1 (12.5%)	7 (87.5%)	G^{+ve}, G^{-ve}, Lactob.	< 0.0001
Gentamicin (GEN-10)	65 (39.63%)	9 (5.48%)	90 (54.87%)	37 (50.68%)	13 (17.80%)	23 (31.50%)	2 (25%)	0 (0%)	6 (75%)	G^{+ve}, G^{-ve}, Lactob.	< 0.0001
Levofloxacin (LVX-5)	64 (39.02%)	15 (9.14%)	85 (51.82%)	50 (68.49%)	5 (6.84%)	18 (34.65%)	8 (100%)	0 (0%)	0 (0%)	G^{+ve}, G^{-ve}, Lactob.	0.0003
Amoxicillin-clavulanate (AMC-30)	100 (65.78%)	10 (6.57%)	42 (27.63%)	35 (47.94%)	7 (9.58%)	31 (42.46%)	5 (62.5%)	3 (37.5%)	0 (0%)	G^{+ve}, G^{-ve}, Lactob.	< 0.0001
Vancomycin (VAN-30)	108 (65.85%)	15 (9.14%)	41 (25%)	---	---	---	8 (100%)	0 (0%)	0 (0%)	G^{+ve}, Lactobacilli	0.001

Antibiotic (Disc- conc .µg)		G+ve (n=164)			G-ve (n=73)			*Lactobacilli* (n=8)			Groups Compared	p-value (Fisher's Test)
		S (%)	I (%)	R (%)	S (%)	I (%)	R (%)	S (%)	I (%)	R (%)		
Trimethoprim-sulfamethox		43 (26.21%)	12 (7.31%)	109 (66.46%)	30 (41.09%)	3 (4.10%)	40 (54.79%)	7 (87.5%)	0 (0%)	1 (12.5%)	G^{+ve}, G^{-ve}, Lactob.	0.0004
Tetracycline (TET-30)		62 (37.80%)	26 (15.85%)	76 (46.34%)	29 (39.72%)	5 (6.84%)	39 (53.42%)	8 (100%)	0 (0%)	0 (0%)	G^{+ve}, G^{-ve}, Lactob.	< 0.0001
Erythromycin (ERY-15)		18 (10.97%)	11 (6.70%)	135 (82.31%)	---	---	---	4 (50%)	4 (50%)	0 (0%)	G^{+ve}, Lactob.	0.002
Ampicillin (AMP-10)		---	---	---	---	---	---	8 (100%)	0 (0%)	0 (0%)		
Cefixime (CFM-5)		---	---	---	28 (38.35%)	6 (8.21%)	39 (53.42%)	2 (25%)	1 (12.5%)	5 (62.5%)	G^{-ve}, Lactob.	0.04
Chloramphenicol (CHL-30)		---	---	---	30 (52.63%)	8 (14.03%)	19 (33.33%)	8 (100%)	0 (0%)	0 (0%)	G^{-ve}, Lactob.	0.001
Ceftriaxone (CRO-30)		---	---	---	40 (54.7%)	4 (5.47%)	29 (39.72%)	1 (12.5%)	5 (62.5%)	2 (25%)	G^{-ve}, Lactob.	0.02
Cefotaxime (CTX-30)		---	---	---	28 (38.35%)	4 (5.47%)	41 (56.16%)	2 (25%)	2 (25%)	4 (50%)	G^{-ve}, Lactob.	0.03
Cefoxitin (FOX-30)		---	---	---	---	---	---	0 (0%)	0 (0%)	8 (100%)		

S: Sensitive, I: intermediate sensitivity, R: resistance

Fisher's exact test was appropriate due to: Small sample size in *Lactobacilli* group (n=8), Low expected frequencies in some cells (<5).

Discussion

This study was designed to determine the possible incidence and diversity of antimicrobial resistance profiles of bacteria colonizing the cervico-vaginal epithelium of women. The findings of this study, where *Staphylococcus aureus*, *Escherichia coli*, and coagulase-negative *Staphylococci* (CoNS) were the most prevalent vaginal isolates, align with trends observed in other recent studies [17-18]. For

instance, similar dominance of *Staphylococcus aureus* in vaginal infections has been reported [17]. The high prevalence of *Escherichia coli* and Coagulase-negative *Staphylococcus* is consistent with findings suggesting possible colonization from the gastrointestinal tract or skin microbiota [18]. The detection of *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* is notable, as these pathogens are increasingly associated with antimicrobial resistance (AMR) in urogenital infections [19]. *Streptococcus agalactiae* remains a critical pathogen due to its association with neonatal infections, reinforcing the need for routine screening [20]. The low prevalence of *Gardnerella vaginalis* contrasts with some studies where it dominates bacterial vaginosis cases, possibly due to differences in diagnostic methods or population characteristics [21].

The findings from this study demonstrate alarmingly high resistance rates among vaginal *Staphylococcal* isolates, with macrolide resistance exceeding 70% (reaching >90% for Coagulase-negative *Staphylococci* against erythromycin/azithromycin) and penicillin resistance, consistent with recent global surveillance data showing similar resistance patterns in both hospital and community settings [22]. While glycopeptides remain relatively effective, the substantial resistance to fluoroquinolones and clindamycin mirrors concerning trends reported by [23], particularly for Gram-positive urogenital pathogens. These resistance patterns, likely driven by *erm* gene-mediated macrolide resistance, *blaZ* β-lactamase production, and *gyrA*/topoisomerase mutations [13], emphasize the critical need for routine susceptibility testing and antimicrobial stewardship programs in gynecological care. The relatively preserved tetracycline susceptibility may offer alternative treatment options, though emerging resistance mechanisms warrant ongoing surveillance as highlighted in recent studies on vaginal microbiome dynamics [24].

The antibiotic resistance profiles observed in this study reveal significant challenges in managing vaginal infections. For *Streptococcus* spp., the high macrolide resistance aligns with global reports of increasing *erm* gene dissemination [25], while the species-specific variations (e.g., 100% levofloxacin resistance in *Streptococcus alactolyticus*) underscore the need for precise identification and susceptibility testing. The concerning teicoplanin non-susceptibility in some species parallels emerging glycopeptide resistance trends in Europe [26]. Among Gram-negative isolates, the high resistance in *Escherichia coli* and carbapenem-resistant *Klebsiella pneumoniae* mirrors the WHO report [23]. The preserved *Proteus mirablis* susceptibility to β-lactam/β-lactamase inhibitors contrasts with recent Asian studies showing rising ESBL production [21], suggesting regional variability. Notably, the divergent resistance between *Pseudomonas aeruginosa* and *Acinetobacter* spp. reflects their distinct resistance mechanisms. While *Pseudomonas* maintains relative susceptibility, *Acinetobacter* spp. cephalosporin resistance aligns with global reports of *AmpC* overexpression [13].

Lactobacillus strains in this study revealed important considerations for preserving beneficial microbiota during antibiotic therapy. The universal susceptibility to levofloxacin, vancomycin, tetracycline, ampicillin, and chloramphenicol aligns with recent findings in a study of vaginal probiotics' antibiotic resistance patterns [28]. However, the complete resistance to cefoxitin and high resistance to aminoglycosides (amikacin 87.5%, gentamicin 75%) [23], which caution against these classes for vaginal microbiota preservation. The variable susceptibility to clindamycin and erythromycin matches observations that *Lactobacillus* spp. resistance to these agents is strain-dependent [29]. The poor cephalosporin activity contrasts with better performance of trimethoprim-sulfamethoxazole and ciprofloxacin, suggesting these may be preferable options when *Lactobacillus* spp. preservation is desired [30].

The striking divergence in antibiotic susceptibility between vaginal pathogens and protective *Lactobacillus* species revealed in this study has critical implications for microbiome-sparing therapy. The high resistance of Gram-positive pathogens to erythromycin and clindamycin contrasts sharply with *Lactobacillus* retained susceptibility to cell-wall active agents (vancomycin/ampicillin), mirroring findings from recent multisite studies [31]. This dichotomy supports current clinical guidelines recommending vancomycin or ampicillin as preferred options when vaginal microbiota preservation is prioritized [32]. For Gram-negative pathogens, resistance to cephalosporins (cefixime 53.42%) and tetracycline (53.42%) [23]. However, the maintained *Lactobacillus* susceptibility to tetracycline (100%) and fluoroquinolones (levofloxacin 100%, ciprofloxacin 87.5%) suggests these may be preferable to β-lactams for mixed infections, as noted in established laboratory standards [29].

The statistical analysis revealed highly significant differences in antibiotic resistance patterns among bacterial groups, with Gram-positive isolates showing particularly high resistance to erythromycin and trimethoprim-sulfamethoxazole, while maintaining greater susceptibility to vancomycin (65.85% susceptible, 25% resistant), consistent with a recent study [23]. These findings align with global trends demonstrating increasing resistance among Gram-positive pathogens, particularly to macrolides and sulfonamides, while highlighting vancomycin's continued importance as a last-line agent, though emerging intermediate resistance (9.14%) warrants close monitoring [33]. The distinct resistance profile of *Lactobacilli*, including intrinsic resistance to aminoglycosides (gentamicin 75%, amikacin 87.5%) but universal vancomycin susceptibility (100%), corroborates probiotic safety studies [34] while emphasizing the need for species-specific susceptibility testing in clinical practice [35]. The superior activity of levofloxacin against Gram-negative isolates (68.49% susceptible) compared to Gram-positive organisms (51.82% resistant) reflects well-documented differences in quinolone resistance mechanisms between these groups [36].

The findings of this study align with and expand upon contemporary research on antibiotic susceptibility patterns in *Lactobacillus* spp. versus pathogenic bacteria. The significantly higher susceptibility of *Lactobacillus* spp. to fluoroquinolones compared to Gram-negative pathogens is consistent with recent reports by the study, which demonstrated that *Lactobacillus* species generally maintain high sensitivity to fluoroquinolones due to their limited exposure to these antibiotics in the gut and vaginal microbiota [37]. This contrasts with Gram-negative pathogens, which frequently develop resistance via plasmid-mediated *qnr* genes and efflux pump upregulation [23]. The universal susceptibility of *Lactobacillus* spp. to vancomycin compared to Gram-positive pathogens supports the findings of another study, which noted that most commensal *lactobacilli* lack the *vanA/vanB* resistance genes commonly found in *Enterococcus* and *Staphylococcus* species [38]. However, emerging reports suggest that prolonged vancomycin use in clinical settings may still disrupt *Lactobacillus*-dominant microbiota [39], warranting cautious use even with favorable susceptibility profiles. The superior sensitivity of *Lactobacillus* spp. to chloramphenicol was proven [39]. However, systemic toxicity of chloramphenicol limits its clinical utility despite these findings.

Conclusions

These findings highlight the potential collateral damage of antimicrobial therapy on vaginal *Lactobacillus* species. Tailored antibiotic selection or probiotic adjuvants may be needed to preserve the vaginal microbiome while treating infections.

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