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Full Length Research

Antimicrobial resistance patterns among bacterial isolates from high vaginal swabs in a tertiary hospital in Southern Nigeria: Implications for empirical therapy

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ABSTRACT: Antimicrobial resistance (AMR) has emerged as one of the most significant global public health challenges of the twenty-first century. The burden is particularly severe in low- and middle-income countries, where antibiotics are frequently available without prescription and empirical treatment is common practice. In many healthcare settings, vaginal infections are often managed syndromically without microbiological confirmation. Such practices may contribute to the inappropriate selection of antibiotics and the subsequent emergence and spread of resistant bacteria. Consequently, the effectiveness of commonly used antimicrobial agents is increasingly threatened, complicating the management of routine gynaecological infections. This study aimed to characterise the spectrum of bacterial isolates recovered from high vaginal swabs and determine their antimicrobial susceptibility profiles in a tertiary healthcare facility in Southern Nigeria to inform evidence-based therapeutic decisions. A laboratory-based retrospective cross-sectional study design was employed. A total of 100 non-duplicate bacterial isolates obtained from high vaginal swab specimens submitted for routine diagnostic evaluation were analysed. Bacterial identification was carried out using standard microbiological methods, including Gram staining, colony morphology, and conventional biochemical tests. Antimicrobial susceptibility testing was performed using the Kirby–Bauer disc diffusion method on appropriate culture media. Inhibition zone diameters were measured and interpreted in accordance with the laboratory's standard operating procedures. Data were analysed using descriptive statistics and presented as frequencies and percentages. Five principal bacterial species were identified: *Staphylococcus aureus* (30%), *Pseudomonas* species (23%), *Escherichia coli* (20%), *Klebsiella* species (15%), and *Proteus* species (12%). Overall antimicrobial susceptibility was highest to ertapenem (99%) and ciprofloxacin (93%). In contrast, cefepime demonstrated the highest resistance rate (94%), with cephalosporin resistance elevated across several species. The findings highlight the need for routine culture and susceptibility testing, strengthened antimicrobial stewardship programmes, rational prescribing practices, and continuous surveillance to curb the escalation of antimicrobial resistance.

Keywords: Antimicrobial resistance, antimicrobial stewardship, antimicrobial susceptibility, bacterial isolates, cephalosporin resistance, high vaginal swab, testing.

INTRODUCTION

Vaginal discharge is one of the most frequent presenting complaints in gynaecological and primary care practice and represents a significant proportion of outpatient consultations (Paladine and Desai, 2018; Sim *et al*., 2020). It may arise from physiological processes, such as cyclical hormonal variations, or from infectious and inflammatory conditions affecting the lower genital tract. Differentiating between normal and pathological discharge is clinically important, as an inappropriate diagnosis may lead to unnecessary antimicrobial use. Infectious causes are commonly associated with alterations in the composition of the vaginal microbiota, resulting in symptomatic discomfort, pruritus, odour and abnormal secretions.

The healthy vaginal microbiome is predominantly

composed of *Lactobacillus* species, which play a crucial protective role by producing lactic acid, hydrogen peroxide and bacteriocins that maintain an acidic vaginal pH and inhibit the proliferation of pathogenic bacteria (Hiller *et al*., 1992; Velraeds *et al*., 1996). This stable ecological environment forms an essential component of innate mucosal immunity. Disruption of this balance—whether due to antibiotic exposure, hormonal fluctuations, sexual activity, poor hygiene practices or underlying systemic illness—predisposes individuals to colonisation and infection by opportunistic and potentially pathogenic bacteria. Frequently implicated bacteria include *Staphylococcus aureus*, *Escherichia coli*, *Klebsiella* species, *Proteus* species and *Pseudomonas* species, all of which may be associated with symptomatic genital tract infections and adverse reproductive health outcomes.

The advent of antibiotics revolutionised the treatment of infectious diseases and markedly reduced morbidity and mortality worldwide (Aminov, 2010; Gould, 2016). Nevertheless, the success of antimicrobial therapy has been undermined by the widespread misuse and overuse of these agents in both human medicine and agriculture (Alanis, 2005; Levy, 1993; Spellberg, 2014). In many settings, empirical prescribing without laboratory confirmation, incomplete treatment courses, self-medication and the availability of antibiotics without prescription have accelerated the development and dissemination of antimicrobial resistance (AMR) (Pulcini *et al*., 2011; Kheder, 2013). The World Health Organisation has identified AMR as one of the foremost global public health threats, warning of a potential post-antibiotic era in which common infections may once again become difficult to treat (WHO, 2014; WHO, 2023). The burden of resistance is particularly severe in low-income and resource-limited settings, where regulatory enforcement may be weak and diagnostic infrastructure limited (Vila and Pal, 2010; Vincent, 2003).

At the molecular level, bacterial resistance arises through diverse mechanisms. These include modification of antimicrobial targets, enzymatic inactivation of drugs (such as beta-lactamase production), activation of efflux pumps that expel antibiotics from the cell, and alterations in membrane permeability that reduce intracellular drug accumulation (Chopra and Roberts, 2001; Kapoor *et al*., 2017). Horizontal gene transfer via plasmids, transposons and integrons further facilitates the rapid spread of resistance determinants among bacterial populations. Compounding this challenge is the diminishing pipeline of novel antimicrobial agents, which limits therapeutic options and increases reliance on last-resort drugs (Piddock, 2012; Gould and Bal, 2013).

In the context of vaginal infections, treatment is frequently initiated empirically, particularly in busy clinical settings where immediate laboratory confirmation may not be readily available. While syndromic management can provide prompt relief, it may also contribute to inappropriate antibiotic selection and selective pressure favouring resistant strains. Surveillance of local antimicrobial susceptibility patterns is therefore essential to guide rational therapy and inform antimicrobial stewardship strategies. Although studies have documented susceptibility profiles of vaginal isolates in other geographical regions (Ayesha *et al*., 2014; González-Reyes *et al.,* 2024), there remains a paucity of comprehensive and up-to-date data from Southern Nigeria.

This study was therefore undertaken to determine the bacterial profile of high vaginal swab isolates and to evaluate their antimicrobial susceptibility patterns in a tertiary hospital in Southern Nigeria. Generating local evidence is vital for optimising empirical treatment guidelines, supporting stewardship interventions and mitigating the continued emergence of antimicrobial resistance within the region.

MATERIALS AND METHODS

Study design

This investigation was designed as a retrospective, laboratory-based cross-sectional study. It involved the analysis of 100 non-duplicate bacterial isolates recovered from high vaginal swab specimens obtained from symptomatic women attending a tertiary healthcare facility in Southern Nigeria. Only one isolate per patient was included to prevent duplication and ensure independence of observations. Laboratory records were reviewed over the defined study period, and relevant microbiological data were extracted for analysis.

Sample size determination

The required sample size was calculated using the single-proportion formula commonly applied in cross-sectional studies:

$$\mathrm{n} = \frac{Z^2 P(1-P)}{d^2}$$

Where *n* represents the minimum sample size, *Z* corresponds to the standard normal deviate at a 95% confidence level (1.96), *P* denotes the anticipated prevalence of antimicrobial resistance, and *d* indicates the desired margin of error (precision). In the absence of reliable local prevalence data, a conservative estimate of 50% resistance was assumed to maximise sample size and enhance statistical robustness. With a precision level of 10%, the calculated minimum sample size was 96 isolates. To compensate for possible incomplete records or exclusion during data validation, the sample size was rounded up, and 100 eligible isolates were ultimately included in the final analysis.

Identification procedures

Bacterial identification was carried out using standard microbiological techniques in accordance with established laboratory protocols (Reller *et al*., 2009). Initial differentiation was performed by Gram staining to determine Gram reaction and cellular morphology. Colony characteristics, including size, pigmentation, haemolytic properties and growth patterns on routine culture media, were also assessed. Biochemical confirmation tests (catalase test (tube method), coagulase test (slide method), indole test, oxidase test and sugar fermentation test) appropriate to each bacterium were subsequently conducted to achieve species-level identification. These procedures ensured accurate characterisation of isolates before antimicrobial susceptibility testing.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed using the Kirby–Bauer disc diffusion method on appropriate culture media, following recognised standard methodology (Graham *et al*., 1985). Briefly, bacterial suspensions were standardised to match the turbidity of a 0.5 McFarland standard before inoculation onto agar plates. Antibiotic-impregnated discs were placed on the inoculated surface, and plates were incubated under appropriate conditions. The antibiotics tested included ciprofloxacin, ertapenem, cefuroxime, cefalexin and cefepime, representing commonly prescribed agents within the study setting. After incubation, inhibition zone diameters were measured in millimetres using a calibrated ruler. Results were interpreted as susceptible, intermediate or resistant in accordance with the laboratory's established standard operating procedures for routine diagnostic antimicrobial susceptibility testing.

Statistical analysis

Data obtained from laboratory records were entered into a structured data sheet and analysed using manual descriptive statistical methods. Categorical variables were summarised as frequencies and percentages to illustrate the distribution of bacterial species and their corresponding susceptibility profiles. Ninety-five per cent confidence intervals were calculated using standard binomial approximation techniques to provide estimates of precision around key proportions. The results were presented in tabular and narrative formats to facilitate clarity and interpretation.

Ethics statement

This study was based on the retrospective analysis of anonymised bacterial isolates obtained as part of routine diagnostic laboratory procedures. No direct patient contact occurred during the course of the study, and no identifiable personal or clinical information was accessed or recorded. All data were handled in accordance with established institutional policies governing confidentiality and data protection. In line with institutional guidelines for laboratory audit and quality assurance studies involving de-identified specimens, formal ethical approval and informed consent were not required. The study was conducted in accordance with accepted ethical standards for research involving human-derived laboratory materials.

RESULTS

Distribution of bacterial isolates from high vaginal swabs

As seen in Table 1, a total of 100 bacterial isolates were analysed, and five distinct bacterial groups were identified. *Staphylococcus aureus* was the most frequently isolated bacterium, accounting for 30% of all isolates. This was followed by *Pseudomonas* species (23%), *Escherichia coli* (20%), *Klebsiella* species (15%) and *Proteus* species (12%). The distribution indicates a predominance of both Gram-positive and Gram-negative bacteria among high vaginal swab samples in the study population.

Antibiotic susceptibility patterns

The antibiotic susceptibility profiles of the isolates are summarised in Tables 2 to 7. The analysis of antimicrobial susceptibility patterns demonstrated considerable variation across the tested bacteria. Ertapenem exhibited the highest overall effectiveness, with 99% of isolates classified as susceptible. Ciprofloxacin also demonstrated substantial activity, with an overall susceptibility rate of 93%. In contrast, cefepime showed the highest level of resistance, with 94% of isolates exhibiting non-susceptibility. These findings reflect a pattern of preserved carbapenem efficacy alongside marked resistance to certain cephalosporins within the study setting.

DISCUSSION

The findings of this study demonstrate a predominance of Gram-negative bacteria among isolates recovered from high vaginal swab specimens. This observation is consistent with reports from similar settings, where Gram-negative enteric bacteria have been frequently implicated in genital tract infections (Ayesha *et al*., 2014; González-Reyes *et al.,* 2024). The notable presence of bacteria such as *Escherichia coli*, *Klebsiella* species, *Proteus* species and *Pseudomonas* species, as seen in Table 1, may reflect

Table 1. Distribution of bacterial isolates from high vaginal swabs.

Bacterial Species	Number of Isolates (n)	Percentage (%)
Staphylococcus aureus	30	30
Pseudomonas species	23	23
Escherichia coli	20	20
Klebsiella species	15	15
Proteus species	12	12

Table 2. Antibiotic susceptibility pattern of *Staphylococcus aureus*

S/N	CIP (mm)	ETP (mm)	CXM (mm)	CFX (mm)	FEP (mm)
1	21	28	4	20	0
2	21	28	0	8	0
3	0	27	9	23	2
4	12	22	2	0	12
5	0	23	0	0	0
6	35	27	12	10	0
7	21	20	0	13	0
8	0	23	0	0	0
9	28	24	0	14	0
10	0	21	0	0	0
11	26	22	10	0	0
12	22	20	0	0	0
13	26	34	15	18	0
14	17	28	20	16	0
15	30	21	12	13	4
16	21	23	2	12	0
17	30	21	12	14	0
18	28	20	12	0	0
19	22	18	13	0	0
20	21	26	8	13	0
21	27	22	0	13	0
22	25	27	11	10	0
23	30	22	10	13	0
24	27	24	14	10	0
25	30	27	8	4	0
26	35	28	9	0	0
27	26	30	18	16	0
28	26	26	0	17	0
29	28	25	2	19	0
30	26	28	8	10	0

faecal contamination, anatomical proximity of the perineum to the gastrointestinal tract, and behavioural or hygiene-related factors. The identification of *Staphylococcus aureus* as a leading isolate further underscores the diverse microbial aetiology associated with symptomatic vaginal discharge and highlights the need for accurate microbiological diagnosis rather than reliance solely on syndromic management.

The high level of susceptibility observed with ertapenem is encouraging and is biologically plausible, given the structural stability of carbapenems against many β-lactamases produced by Gram-negative bacteria (Kahne *et al*., 2005). Carbapenems are widely regarded as highly effective broad-spectrum agents, often reserved for the treatment of multidrug-resistant infections. However, their preserved activity in this study should be interpreted with caution. Increased or indiscriminate reliance on carbapenems may exert selective pressure favouring the emergence and dissemination of carbapenem-resistant strains, including those harbouring carbapenemase

Table 3. Antibiotic susceptibility pattern of *Klebsiella species.*

S/N	CIP (mm)	ETP (mm)	CXM (mm)	CFX (mm)	FEP (mm)
1	19	24	12	9	0
2	24	30	28	28	29
3	25	28	10	20	0
4	20	22	0	0	2
5	17	20	0	0	0
6	26	26	2	12	0
7	26	26	0	10	0
8	40	22	2	19	0
9	22	29	2	10	0
10	22	25	0	10	0
11	22	26	2	12	0
12	20	26	2	12	0
13	25	25	0	0	0
14	22	23	10	12	0
15	23	25	8	0	0

Table 4. Antibiotic susceptibility pattern of *Escherichia coli.*

S/N	CIP (mm)	ETP (mm)	CXM (mm)	CFX (mm)	FEP (mm)
1	25	28	10	29	0
2	0	29	0	0	10
3	25	28	10	28	0
4	29	29	10	25	0
5	35	27	12	10	0
6	0	31	15	10	10
7	25	18	2	17	0
8	2	10	16	0	0
9	29	29	10	25	0
10	0	30	0	0	10
11	20	25	0	17	0
12	24	28	4	0	2
13	20	24	2	14	0
14	27	29	0	0	0
15	28	30	2	13	0
16	22	26	0	10	0
17	20	25	12	20	0
18	23	29	2	10	0
19	22	25	0	0	0
20	24	25	0	0	0

Table 5. Antibiotic susceptibility pattern of *Proteus species.*

S/N	CIP (mm)	ETP (mm)	CXM (mm)	CFX (mm)	FEP (mm)
1	30	32	28	28	29
2	25	23	23	30	0
3	30	32	28	28	29
4	25	23	23	30	3
5	23	28	18	12	0
6	25	31	22	22	22
7	20	30	0	0	0
8	22	27	12	10	0
9	24	28	0	2	0
10	22	27	14	10	0
11	14	26	0	10	0
12	24	25	2	12	0

Table 6. Antibiotic susceptibility pattern of *Pseudomonas species.*

S/N	CIP (mm)	ETP (mm)	CXM (mm)	CFX (mm)	FEP (mm)
1	0	30	19	14	18
2	35	30	0	0	0
3	30	30	10	9	0
4	35	30	2	0	0
5	23	28	18	12	0
6	26	28	2	0	0
7	23	28	0	0	0
8	20	27	10	0	0
9	25	29	14	0	0
10	27	31	15	4	4
11	24	30	13	10	0
12	23	28	10	13	2
13	22	28	13	0	0
14	26	27	10	11	0
15	24	27	0	9	0
16	20	25	0	8	0
17	23	24	10	2	0
18	23	26	0	0	0
19	21	27	14	13	0
20	26	29	17	8	0
21	25	24	12	2	0
22	25	26	10	0	0
23	23	28	4	0	0

Table 7. Frequency of bacterial isolates and percentage of antibiotic susceptibility against bacterial isolates

Frequency	Bacteria	CIP (S)	CIP (R)	ETP (S)	ETP (R)	CXM (S)	CXM (R)	CFX (S)	CFX (R)	FEP (S)	FEP (R)
30	*Staphylococcus aureus*	30	0	30	0	4	26	7	23	1	29
15	*Klebsiella spp.*	15	0	15	0	1	14	4	11	1	14
20	*Escherichia coli*	15	5	19	1	2	18	7	13	0	20
12	*Proteus spp.*	11	1	12	0	6	6	5	7	3	9
23	*Pseudomonas spp.*	22	1	23	0	4	19	0	23	1	22
	Overall %	93	7	99	1	17	83	23	77	6	94

Key: S = Sensitive, R= Resistant, Cip = Ciprofloxacin, Etp = Ertapenem, Cxm = Cefuroxime, Cfx= Cefalexin, Fep = Cefepime.

enzymes (Imamovic and Sommer, 2013). The global rise of carbapenem-resistant Enterobacteriaceae illustrates the potential consequences of overdependence on this class of antibiotics, particularly in resource-limited settings where infection prevention and control measures may be suboptimal.

The markedly high resistance to cefepime (94%) is a cause for concern and may indicate diminished clinical utility of certain fourth-generation cephalosporins within the study environment (Qavi *et al*., 2005). Cephalosporins are frequently prescribed for a variety of community- and hospital-acquired infections due to their broad spectrum and favourable safety profile. However, extensive use over time can select for bacteria producing extended-spectrum β-lactamases (ESBLs) or other resistance determinants, thereby reducing therapeutic effectiveness. The elevated resistance observed in this study suggests that empirical use of cephalosporins for vaginal infections may warrant reconsideration pending susceptibility results.

Ciprofloxacin demonstrated relatively high levels of retained activity, which aligns with earlier reports documenting its effectiveness against common urogenital pathogens (Hoge *et al*., 1998). As a fluoroquinolone, ciprofloxacin offers the advantages of oral bioavailability and broad antimicrobial coverage. Nevertheless, caution remains necessary, as global surveillance data indicate increasing resistance to fluoroquinolones in both community and hospital settings (Cars and Nordberg, 2005; WHO, 2011). Continued monitoring of susceptibility trends is therefore essential to prevent erosion of its therapeutic value.

Although limited in scope, this study contributes valuable baseline data on antimicrobial susceptibility patterns in Southern Nigeria, a region where structured and continuous

resistance surveillance remains insufficient. Local epidemiological data are indispensable for developing evidence-based empirical treatment guidelines tailored to prevailing resistance profiles. Furthermore, such information supports antimicrobial stewardship initiatives aimed at optimising antibiotic selection, minimising unnecessary exposure, and slowing the progression of resistance.

Several limitations should be acknowledged. The study did not incorporate molecular methods to characterise specific resistance genes or mechanisms, which would have provided deeper insight into the genetic determinants underlying observed phenotypic resistance patterns. In addition, inferential statistical analyses were not performed, thereby limiting the ability to explore associations or determine statistical significance between variables. The single-centre, retrospective design may also restrict generalisability to other healthcare facilities or regions. Despite these constraints, the findings offer a useful foundation for future prospective, multi-centre investigations

Conclusion

The findings of this study demonstrate a notable level of resistance to cephalosporins among bacterial isolates recovered from high vaginal swab specimens, raising concerns regarding the continued empirical use of this class of antibiotics in the management of vaginal infections within the study setting. In contrast, carbapenems and fluoroquinolones, particularly ertapenem and ciprofloxacin, retained comparatively high levels of activity against the majority of isolates. While this preserved susceptibility is encouraging, cautious and judicious use of these agents is imperative to prevent the emergence of resistance to last-line therapies.

The observed resistance patterns underscore the importance of routine microbiological culture and antimicrobial susceptibility testing in guiding treatment decisions. Reliance on syndromic management without laboratory confirmation may contribute to inappropriate antibiotic selection and accelerate resistance development. Strengthening antimicrobial stewardship programmes, promoting rational prescribing practices, and enhancing continuous local surveillance of resistance trends are critical steps towards mitigating further escalation of antimicrobial resistance. In addition, investment in diagnostic capacity and ongoing professional education for healthcare providers will support evidence-based management and improve patient outcomes.

CONFLICT OF INTEREST

The authors declare that there are no financial, professional or personal relationships that could be construed as potential conflicts of interest in relation to this study. The research was conducted independently, and the findings presented are solely those of the authors.

REFERENCES

Alanis, A. J. (2005). Resistance to antibiotics: are we in the post-antibiotic era? *Archives of Medical Research*, *36*(6), 697-705.

Aminov, R. I. (2010). A brief history of the antibiotic era: lessons learned and challenges for the future. *Frontiers in Microbiology*, *1*, 134.

Ayesha, B., Jabeen, S., Ismail, M., Salman, S., Ullah, S., Niaz, Z., & Ahmad, T. (2014). Isolation, identification and antibiotic susceptibility testing of microorganisms from female patients of Ayub medical complex through high vaginal swab. Science International 26(4), 1581-1586.

Cars, O., & Nordberg, P. (2005). Antibiotic resistance–The faceless threat. *International Journal of Risk & Safety in Medicine*, *17*(3-4), 103-110.

Chopra, I., & Roberts, M. (2001). Tetracycline antibiotics: mode of action, applications, molecular biology, and epidemiology of bacterial resistance. *Microbiology and Molecular Biology Reviews*, *65*(2), 232-260.

González-Reyes, C., Rosas-Partida, G., Ramos-Ramírez, L. C., Arvizu-Gómez, J. L., Martínez-Rubio, L. N. R., Becerra-Verdín, E. M., & Rodríguez-Ocampo, A. N. (2024). Prevalence and susceptibility of microorganisms in vaginal isolates. *World Journal of Biology Pharmacy and Health Sciences*, *19*(01), 210–217.

Gould, I. M., & Bal, A. M. (2013). New antibiotic agents in the pipeline and how they can help overcome microbial resistance. *Virulence*, *4*(2), 185-191.

Gould, K. (2016). Antibiotics: from prehistory to the present day. *Journal of Antimicrobial Chemotherapy*, *71*(3), 572-575.

Graham, D. R., Dixon, R. E., Hughes, J. M., & Thornsberry, C. (1985). Disk diffusion antimicrobial susceptibility testing for clinical and epidemiologic purposes. *American Journal of Infection Control*, *13*(6), 241-249.

Hiller, S. L., Krohn, M. A., Klebanoff, S. J., Eschenbach, D. A. (1992). The relationship of hydrogen peroxide-producing lactobacilli to bacterial vaginosis and genital microflora in pregnant women. *Obstet Gynecol*, *79*, 369-373.

Hoge, C. W., Gambel, J. M., Srijan, A., Pitarangsi, C., & Echeverria, P. (1998). Trends in antibiotic resistance among diarrheal pathogens isolated in Thailand over 15 years. *Clinical Infectious Diseases*, *26*(2), 341-345.

Imamovic, L., & Sommer, M. O. (2013). Use of collateral sensitivity networks to design drug cycling protocols that avoid resistance development. *Science Translational Medicine, 5*(204), 204ra132.

Kahne, D., Leimkuhler, C., Lu, W., & Walsh, C. (2005). Glycopeptide and lipoglycopeptide antibiotics. *Chemical Reviews*, *105*(2), 425-448.

Kapoor, G., Saigal, S., & Elongavan, A. (2017). Action and resistance mechanisms of antibiotics: A guide for clinicians. *Journal of Anaesthesiology Clinical Pharmacology*, *33*(3), 300-305.

Kheder, S. I. (2013). Physicians' knowledge and perception of antimicrobial resistance: a survey in Khartoum state hospital settings. *British Journal of Pharmaceutical Research*, *3*(3), 347-362.

Levy, S. B. (2013). The antibiotic paradox. How miracle drugs are destroying the miracle. *JAMA, 270*(3), 384-385.

Neal, C. M., Kus, L. H., Eckert, L. O., & Peipert, J. F. (2020). Noncandidal vaginitis: a comprehensive approach to diagnosis and management. *American Journal of Obstetrics and Gynecology*, *222*(2), 114-122.

Paladine, H. L., & Desai, U. A. (2018). Vaginitis: diagnosis and treatment. *American Family Physician*, *97*(5), 321-329.

Piddock, L. J. (2012). The crisis of no new antibiotics—what is the way forward? *The Lancet infectious diseases*, *12*(3), 249-253.

Pulcini, C., Williams, F., Molinari, N., Davey, P., & Nathwani, D. (2011). Junior doctors' knowledge and perceptions of antibiotic resistance and prescribing: a survey in France and Scotland. *Clinical microbiology and infection*, *17*(1), 80-87.

Qavi, A., Segal-Maurer, S., Mariano, N., Urban, C., Rosenberg, C., Burns, J., Chiang, T., Maurer, J., & Rahal, J. J. (2005). Increased mortality associated with a clonal outbreak of ceftazidime-resistant Klebsiella pneumoniae: a case–control study. *Infection Control & Hospital Epidemiology*, *26*(1), 63-68.

Reller, L. B., Weinstein, M., Jorgensen, J. H., & Ferraro, M. J. (2009). Antimicrobial susceptibility testing: A review of general principles and contemporary practices. *Clinical infectious diseases*, *49*(11), 1749-1755.

Sim, M., Logan, S., & Goh, L. H. (2020). Vaginal discharge: evaluation and management in primary care. *Singapore Medical Journal*, *61*(6), 297-301.

Spellberg, B. (2014). The future of antibiotics. *Critical Care*, *18*(3), 228.

Velraeds, M. M., van der Mei, H. C., Reid, G., & Busscher, H. J. (1996). Physicochemical and biochemical characterization of biosurfactants released by Lactobacillus strains. *Colloids and Surfaces B: Biointerfaces*, *8*(1-2), 51-61.

Vila, J., & Pal, T. (2010). Update on antibacterial resistance in low-income countries: factors favoring the emergence of resistance. *The Open Infectious Diseases Journal, 4*, 38-54.

Vincent, J. L. (2003). Nosocomial infections in adult intensive-care units. *The Lancet*, *361*(9374), 2068-2077.

WHO (2001). Global strategy for containment of antimicrobial resistance. WHO, Geneva. Retrieved from https://www.who.int/publications/i/item/who-global-strategy-for-containment-of-antimicrobial-resistance.

WHO (2011). World Health Day 2011: Combating drug resistance, 7 April. WHO, Geneva. Retrieved from https://www.emro.who.int/pakistan-press-releases/2011/whd-2011.html.

WHO (2014). Antimicrobial resistance: Global report on surveillance. WHO, Geneva. Retrieved from https://www.who.int/publications/i/item/9789241564748.

WHO (2023). Antibiotic resistance fact sheet. WHO, Geneva. Retrieved from https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance.