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Antimicrobial Resistance Profile of Uro-Genital Bacterial Isolates in Southern Libreville, Gabon: A Cross-Sectional Study Based on Minimum Inhibitory Concentrations (2025)

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Abstract

Background: Antimicrobial resistance (AMR) is an escalating public health threat in sub-Saharan Africa, where local surveillance data remain limited, including in Gabon. This study aimed to describe the antimicrobial resistance profile of bacterial isolates from uro-genital infections in the southern Libreville region over the year 2025. **Methods:** Retrospective cross-sectional study of 477 clinical specimens processed with an automated identification and susceptibility testing system. After excluding yeasts (n = 199), 278 bacterial isolates were included. Resistance status per antibiotic class was determined from resistance phenotypes and raw minimum inhibitory concentrations (MICs); MIC50 and MIC90 values were calculated for key antibiotics in *Escherichia coli*, *Klebsiella pneumoniae*, and *Staphylococcus* spp. Proportions are reported with 95% confidence intervals (Wilson method). **Results:** The population was predominantly female (76.3%), with urine cytobacteriological examination (43.8%) and combined genito-urinary specimens (40.7%) as the leading specimen types. *Escherichia coli* (35.6%) and *Klebsiella pneumoniae* (14.7%) were the most frequent bacterial isolates. High resistance was observed for trimethoprim-sulfamethoxazole (98.3%) and beta-lactams (79.3%), with an MIC90 for cefotaxime and ceftriaxone ≥ 64 mg/L in both *E. coli* and *K. pneumoniae*, suggestive of a high prevalence of ESBL production. More than half of all bacterial isolates (56.5%) were resistant to at least three antibiotic classes, meeting a standard multidrug-

resistance (MDR) definition. Direct analysis of vancomycin MICs (MIC50 0.75 - 1 mg/L, MIC90 1 mg/L) revealed no glycopeptide resistance among *Staphylococcus* spp., contradicting an initially discordant automated derived indicator. **Conclusion:** These data confirm a high level of resistance to first-line antibiotics among uropathogens in the southern Libreville region, consistent with published data from other Gabonese and sub-Saharan African sites, and highlight fosfomycin, nitrofurantoin, and carbapenems as valuable therapeutic options of last resort.

Subject Areas

Microbiology

Keywords

Antimicrobial Resistance, Minimum Inhibitory Concentration, *Escherichia coli*, *Klebsiella Pneumoniae*, Urinary Tract Infection, Gabon, Sub-Saharan Africa

1. Background

Antimicrobial resistance (AMR) has emerged as one of the defining public health challenges of the twenty-first century. According to PubMed, the most comprehensive assessment of this burden to date, led by Murray and colleagues and funded in part by the UK Department of Health and Social Care through the Fleming Fund, estimated that bacterial AMR was associated with 4.95 million deaths worldwide in 2019, of which 1.27 million were directly attributable to resistant infections [1]. Strikingly, the same analysis found that western sub-Saharan Africa carried the heaviest burden of any region studied, with a death rate attributable to resistance of 27.3 per 100,000 population, more than four times the rate observed in Australasia [1]. This stark geographical disparity is precisely why locally generated surveillance data from African settings, where the human cost of AMR is greatest yet historically least documented, are so urgently needed to inform both national policy and the global response [2].

Central Africa illustrates this evidence gap particularly well, even as the picture there has improved considerably over the past decade [3] [4]. Early hospital-based surveys from Gabon, including a retrospective analysis by Alabi and colleagues at a mission hospital in Lambaréné, described a bacterial spectrum dominated by *Staphylococcus aureus* and *Escherichia coli* with emerging extended-spectrum beta-lactamase (ESBL) production [5] [6], while Scherbaum and co-workers characterised nosocomial infection patterns and resistance at the same rural facility the following year [7] [8]. More recent work from southeastern Gabon has considerably deepened this picture. According to the work of Mouanga-Ndzime and colleagues, in a series of studies conducted around Franceville, reported that community-acquired *E. coli* and *K. pneumoniae* urinary isolates carried ESBL genes in roughly a third of cases, with resistance to beta-lactams, fluoroquinolones and

cotrimoxazole frequently exceeding 50%, and with resistance significantly more common in men and in children under five years of age [3] [4] [6]. A parallel paediatric cohort from the same research group linked multidrug-resistant ESKAPE uropathogens, and cotrimoxazole resistance specifically, to recurrent urinary tract infection and to male sex [5]. Closer still to the setting of the present study, Yala and colleagues, working at the Omar Bongo Ondimba Army Training Hospital in Libreville, found that 74.1% of uropathogenic *Enterobacterales* produced a broad-spectrum beta-lactamase, with resistance to first-line agents reaching 81.6% [2] figures that anticipate, almost precisely, the pattern observed several years later in the present dataset.

A comparable, and in some respects even more concerning, trajectory has been documented in neighbouring Cameroon. Sonkoue Lambou and colleagues, working in Douala, showed that multidrug-resistant *E. coli* disproportionately affected patients with poorly controlled type 2 diabetes, pointing to metabolic disease as an additional, under-recognised driver of AMR in the region [9]. More recent molecular studies led by Founou, Koudoum, Guemkam and co-authors a collaboration spanning the University of Dschang in Cameroon, the Antimicrobial Research Unit at the University of KwaZulu-Natal in South Africa, and the Infection & Global Health Division at the University of St Andrews in the United Kingdom found ESBL prevalence of 64% among *Enterobacterales* in Douala health facilities, with more than 80% of ESBL producers also resistant to fluoroquinolones [10], and traced matching CTX-M-type resistance genes across hospitalised patients, hospital surfaces and wastewater in Yaoundé, providing direct genomic evidence of both nosocomial and environmental transmission [11]. A further study from the West region of Cameroon reported an almost identical ESBL prevalence of 64.7% among urinary *E. coli* and *K. pneumoniae* isolates, with 82% of these meeting standard criteria for multidrug resistance [12]. This work, deliberately bridging Central African, southern African and European institutions, exemplifies the kind of collaborative, genomically informed surveillance that the present study seeks to complement at the phenotypic and MIC level [10].

Beyond Central Africa, comparable patterns have been reported across the continent, even where the predominant pathogens and antibiotic classes of concern differ. In South Africa, Teixeira and colleagues' four-year retrospective analysis at a regional hospital in North West province found that first-line agents recommended by the National Essential Medicines List, including ciprofloxacin and amoxicillin-clavulanate, achieved susceptibility rates below 80% against the leading uropathogens, prompting the authors to call directly for a revision of empirical prescribing guidelines [13]. In Togo, a large multicentre study by Bouyo and colleagues characterised around 409 Klebsiella isolates from urinary and wound infections and found that 92.4% were multidrug-resistant, with ESBL production in 38.1% [14]. In Mali, a nearly two-decade longitudinal analysis at a paediatric referral hospital in Bamako, conducted in partnership with the University of Cambridge, documented a rise in third-generation cephalosporin resistance among

Gram-negative *Enterobacterales* from 30% in 2005-2009 to 93% in 2021-2023, alongside correspondingly higher mortality [15]. In the Democratic Republic of the Congo, Mulinganya and colleagues found that nearly all Enterobacter cloacae isolates and the great majority of *Klebsiella pneumoniae* isolates causing neonatal sepsis in Bukavu displayed an ESBL phenotype and were resistant to the World Health Organization's recommended first-line regimen [16]. In Angola, Avelino and colleagues linked multidrug-resistant pneumococcal disease directly to in-hospital mortality among children under five in Luanda [17]. Taken together, these studies spanning Gabon, Cameroon, South Africa, Togo, Mali, the Democratic Republic of the Congo and Angola depict a sub-Saharan region in which resistance to first-line antibacterial agents is now the rule rather than the exception in common bacterial infections, even as considerable heterogeneity persists between countries, specimen types and patient populations.

Data specific to the Libreville region Gabon's capital and largest urban centre nonetheless remain limited outside the single 2019 study cited above [2]. The present study addresses this gap by describing, for the year 2025, the antimicrobial resistance profile of bacterial isolates from uro-genital and systemic specimens processed in the southern Libreville region, drawing on both antibiotic-class resistance phenotypes and raw minimum inhibitory concentrations to provide a locally grounded, quantitatively detailed contribution to the regional evidence base outlined above.

2. Patients and Methods

2.1. Study Design and Setting

This was a retrospective, descriptive cross-sectional study of all bacterial and fungal isolates identified from clinical specimens received from patients who came to have a checkup at the Department of Bacteriology and Virology of University of Health Sciences in the southern Libreville region, Gabon, between 03 January and 31 December 2025.

2.2. Population and Specimens

All specimens yielding a positive microbiological identification during the study period were included (n = 477), with no a priori exclusion criteria based on patient age or sex. Specimen types included urine cytobacteriological examination (ECBU, n = 209; 43.8%), combined genito-urinary specimens (PV/PU, n = 194; 40.7%), urine specimens alone (PU, n = 28; 5.9%), semen cultures (n = 14; 2.9%), vaginal specimens alone (PV, n = 13; 2.7%), blood cultures (n = 10; 2.1%), stool cultures (n = 8; 1.7%), and one puncture fluid specimen (n = 1; 0.2%). Precise geographic location and patient age were not recorded in an analyzable format in the source database; demographic description is therefore limited to sex.

2.3. Identification and Susceptibility Testing

Microorganism identification and determination of minimum inhibitory concen-

trations (MICs) were performed using an automated identification and antimicrobial susceptibility testing system Vitek 2 Compac (bioMérieux), using identification and susceptibility cards appropriate to each microorganism type. Clinical categories (susceptible/intermediate/resistant) and mechanism-based resistance phenotypes (e.g., extended-spectrum beta-lactamase, efflux-mediated resistance, wild-type phenotype) were generated by the instrument's expert software.

2.4. Data Extraction and Processing

Yeast isolates (*Candida* spp. and related organisms, n = 199) were excluded from antimicrobial resistance analyses, as these organisms are not tested against the antibacterial classes considered here; they are described only in the overall population characterization. Resistance status per antibiotic class was determined from the resistance-phenotype fields generated by the analyzer (wild-type phenotype isolate classified as susceptible for that class; any named acquired resistance mechanism isolate classified as resistant). This approach was preferred over pre-computed summary variables available in the source file, cross-validation of which against raw MIC values revealed systematic inconsistencies (e.g., isolates classified as "resistant" to ciprofloxacin or ceftriaxone despite MIC values well below susceptibility breakpoints). For the glycopeptide class, whose derived phenotype classification remained discordant with the observed vancomycin MICs, interpretation was instead based directly on these MIC values, applying standard clinical breakpoints (MIC ≤ 2 mg/L: susceptible; MIC ≥ 16 mg/L: resistant, per current CLSI/EUCAST recommendations for *Staphylococcus* spp.) reference and edition used by the laboratory, CLSI or EUCAST over the worldwide and European bacteriological laboratories.

Minimum inhibitory concentrations were extracted for the principal antibiotics tested against the most frequently isolated species. Censored values ("≤X" or ">X") were converted to numeric values by retaining the reported bound for "≤X" values, and doubling the reported value for ">X" values, consistent with standard two-fold dilution series conventions. MIC50 and MIC90 values (the MIC inhibiting 50% and 90% of tested isolates, respectively) were calculated as the 50^{th} and 90^{th} percentiles of the MIC distribution for each organism-antibiotic pair with at least 5 evaluable values. Isolates resistant to at least three antibiotic classes were additionally classified as multidrug-resistant (MDR), consistent with the definition used in the comparator studies discussed below [10] [12] [14].

2.5. Statistical Analysis

Resistance proportions were calculated with 95% confidence intervals using the Wilson method. Comparisons of resistance rates by specimen type, sex, and time period of 2025 are presented descriptively with confidence intervals; no formal hypothesis testing was applied given the limited sample size in several subgroups, an explicitly acknowledged limitation (see Discussion). Analyses were performed in Excel 2016, GraphPad 11.

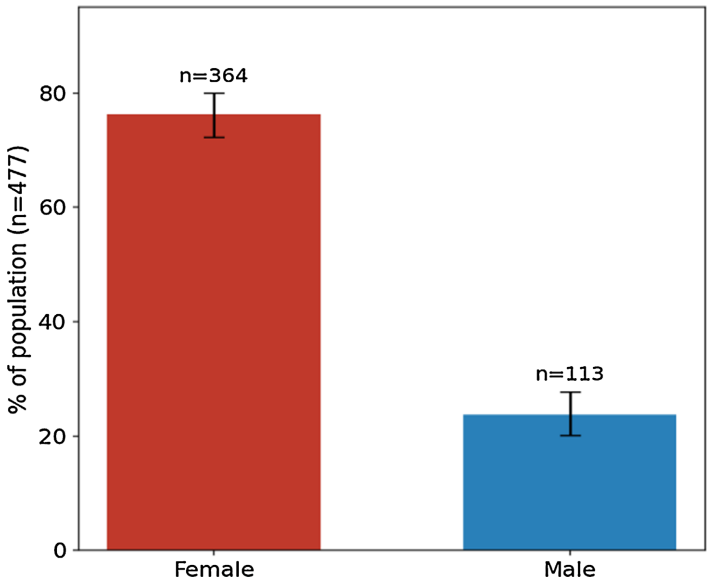
2.6. Ethical Considerations

This study was approved by an authorization from the Director of Medical Affairs of CHU of Libreville after presentation of the research project. For all patients who participated in the study, an informed consent was signed by all patients, both male and female, who agreed to take part in the study after receiving explanations about their participation. All tests were free of charge, and the results were also sent to the department's doctors for appropriate patient management, in line with national recommendations.

3. Results

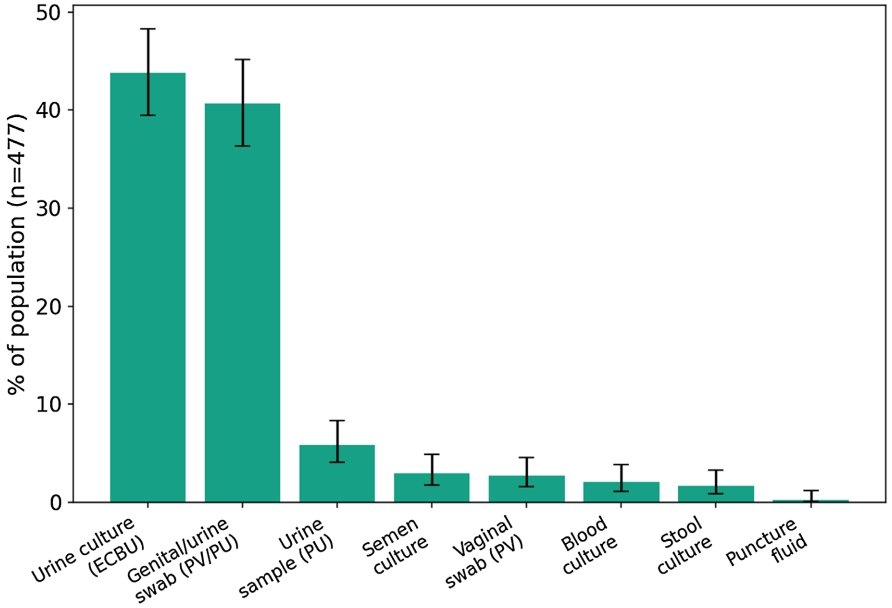
3.1. Population Characteristics

Based on Figure 1, 477 isolates were included, 364 were from female patients (76.3%), and 113 were from male patients (23.7%). *Candida* yeasts accounted for 199 isolates (41.7%), dominated by *Candida albicans* (n = 129, 27.0% of all isolates), followed by *C. glabrata* (n = 27, 5.7%) and *C. tropicalis* (n = 18, 3.8%) based on Figure 2, Among the 278 bacterial isolates (58.3%), *Escherichia coli* was the most frequent species (n = 99, 35.6%), followed by *Klebsiella pneumoniae* (n = 41, 14.7%), *Staphylococcus haemolyticus* (n = 31, 11.2%), *Staphylococcus aureus* (n = 14, 5.0%), *Streptococcus agalactiae* (n = 10, 3.6%), *Staphylococcus saprophyticus* and *Enterobacter cloacae* complex (n = 9 each, 3.2%), and *Pseudomonas aeruginosa* and *Enterococcus faecalis* (n = 8 each, 2.9%) in Figure 3. A further nineteen species were represented by fewer than five isolates each, including *Proteus mirabilis*, *Acinetobacter baumannii* complex, *Citrobacter freundii*, and *Salmonella* spp., reflecting the broad but long-tailed diversity typical of routine clinical bacteriology.



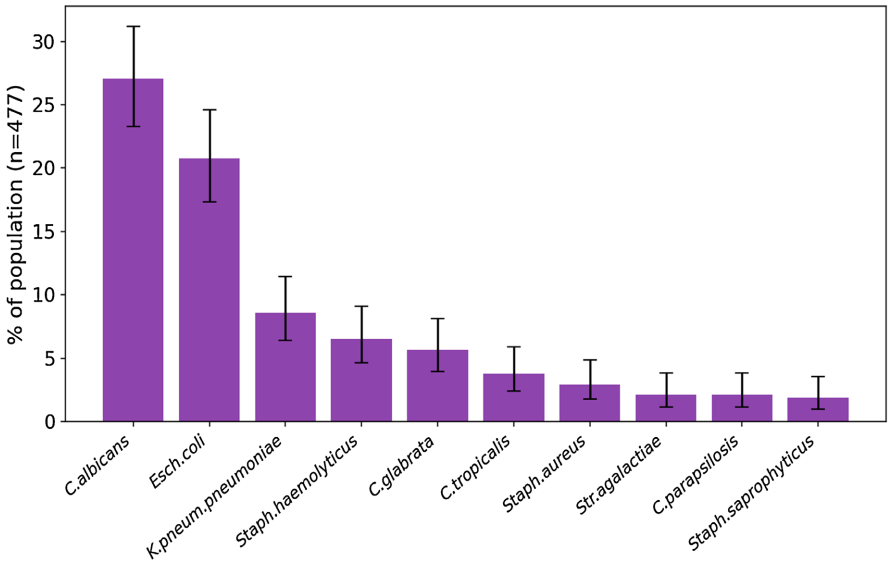
Error bars: 95% confidence interval (Wilson method). n = 477.

Figure 1. Distribution of the study population by sex.



Error bars: 95% confidence interval (Wilson method). n = 477.

Figure 2. Distribution of the study population by specimen type.



Ten most frequent microorganisms overall (bacteria and yeasts). Error bars: 95% confidence interval (Wilson method). n = 477.

Figure 3. Most frequent microorganisms in the study population.

3.2. Overall Resistance by Antibiotic Class

Among the 278 bacterial isolates, resistance was particularly high for trimethoprim-sulfamethoxazole (98.3%, n = 238) and beta-lactams (79.3%, n = 261). Intermediate resistance was observed for tetracyclines (47.0%, n = 83), aminoglycosides (43.5%, n = 253), and fluoroquinolones (34.0%, n = 259). Resistance to nitrofurans remained more limited (21.0%, n = 233), as did resistance to fosfomycin (5.9%, n = 51) and oxazolidinones (1.2%, n = 83) in **Figure 4**. Applying a

standard multidrug-resistance (MDR) definition non-susceptibility to at least one agent in three or more antibiotic classes 56.5% (157/278) of bacterial isolates qualified as MDR, a figure that, while substantial, remains below the 82% - 99.5% MDR rates reported among *Enterobacterales* in comparable Cameroonian series [10]-[12] and the 92.4% reported among Klebsiella isolates in Togo [14], possibly reflecting the broader taxonomic mix of organisms (including less resistant Gram-positive cocci) included in the present denominator. As detailed below (MIC section), the initial automated phenotype classification suggesting 51.9% glycopeptide resistance was not confirmed by direct analysis of vancomycin MICs and is therefore not retained as a primary finding.

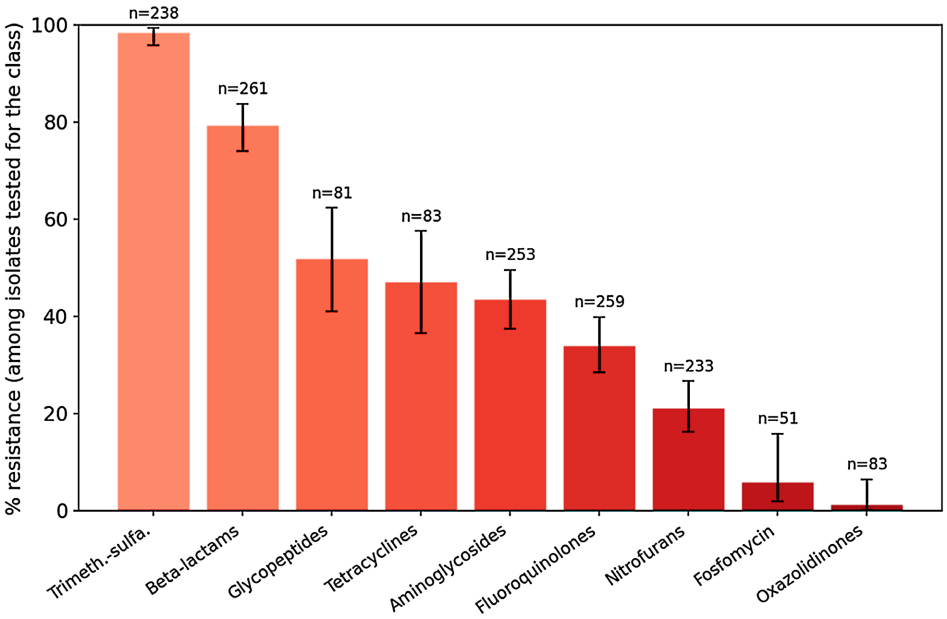
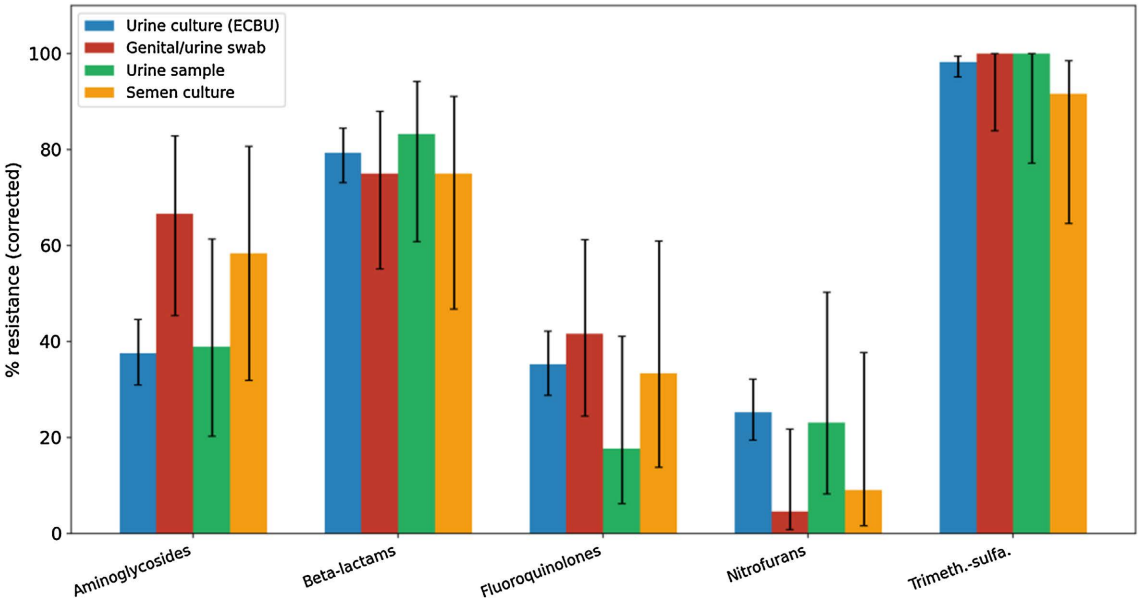


Figure 4. Resistance rate by antibiotic class.

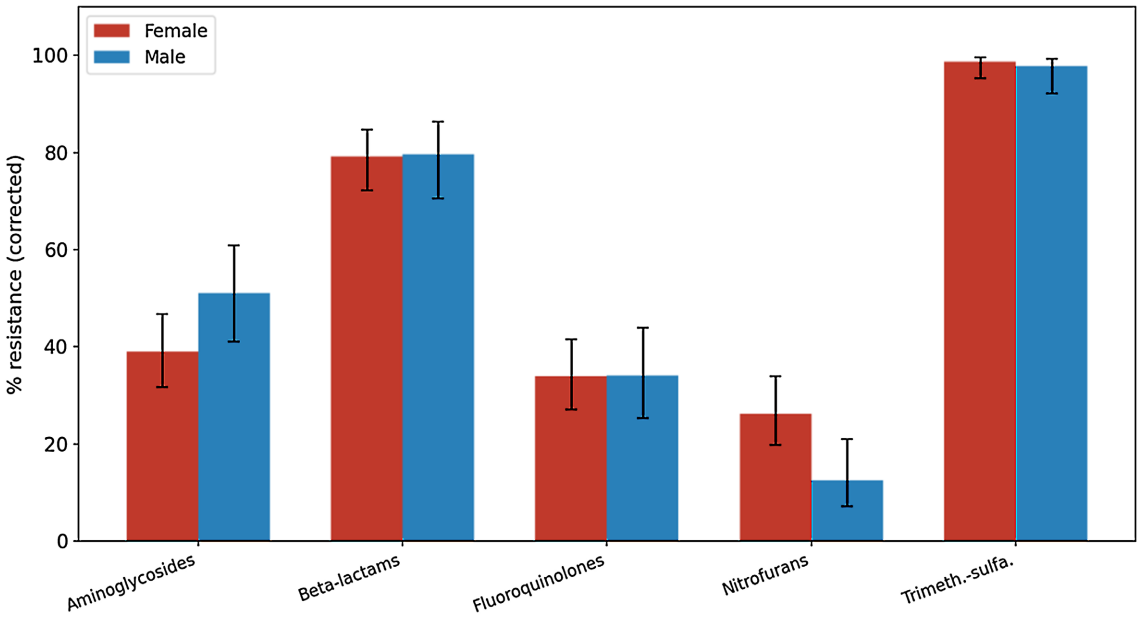
3.3. Resistance Profile by Specimen Type and Sex

Resistance to the main antibiotic classes in **Figure 5** varied by specimen type, with no difference considered clinically major between ECBU, PV/PU, PU, and semen culture for most classes tested, confidence intervals overlapping substantially given the available sample sizes. Beta-lactam resistance ranged from 74.8% (PV/PU) to 83.3% (PU), aminoglycoside resistance from 38.9% (PU) to 66.5% (PV/PU), and fluoroquinolone resistance from 17.5% (PU) to 41.7% (PV/PU), the genital/urinary swab category consistently showing numerically, though not always statistically distinguishable, higher resistance across classes as in **Figure 6**. Similarly, in **Figure 6**, we cannot mark a difference was observed between sexes in resistance to the major classes, sex likely acting more as a proxy for specimen type (semen culture in men, PV/PU in women) than as a resistance determinant in itself; aminoglycoside resistance was nominally higher in men (51.1% vs 39.1% in women) but with overlapping confidence intervals given the smaller male sample size (n = 113 overall).



Bacterial isolates, 95% CI. ECBU: urine cytobacteriological examination; PV/PU: combined genital/urine swab; PU: urine sample only.

Figure 5. Resistance rate by antibiotic class and specimen type.



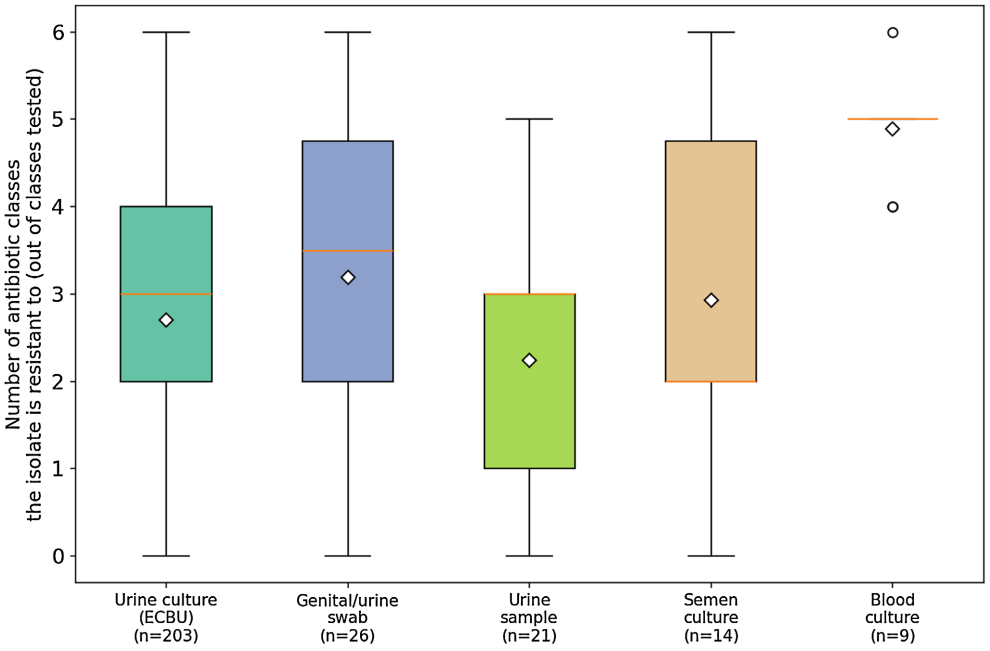
Bacterial isolates, 95% CI.

Figure 6. Resistance rate by antibiotic class and sex.

3.4. Distribution of Resistance Burden per Isolate

Among the 278 bacterial isolates, only 19 (6.8%) remained susceptible to all antibiotic classes tested ("normal" antibiogram), while 259 (93.2%) exhibited resistance to at least one class, and, as noted above, 157 (56.5%) met the multidrug-resistance threshold of three or more classes has mentioned in Figure 7. The median number of classes to which an isolate was resistant ranged from 2 (urine-only specimens)

to 5 (blood cultures), the latter warranting cautious interpretation given the small sample size (n = 9); by species, the highest median resistance burden was observed in *Klebsiella pneumoniae* and in coagulase-negative staphylococci (*S. haemolyticus*, *S. hominis*, *S. epidermidis*), each with a median of 4 - 5 resistant classes, compared with a median of 3 for *E. coli* seen.



Box-and-whisker plot; diamond marker = mean. Bacterial isolates.

Figure 7. Distribution of the number of resistant antibiotic classes per isolate, by specimen type.

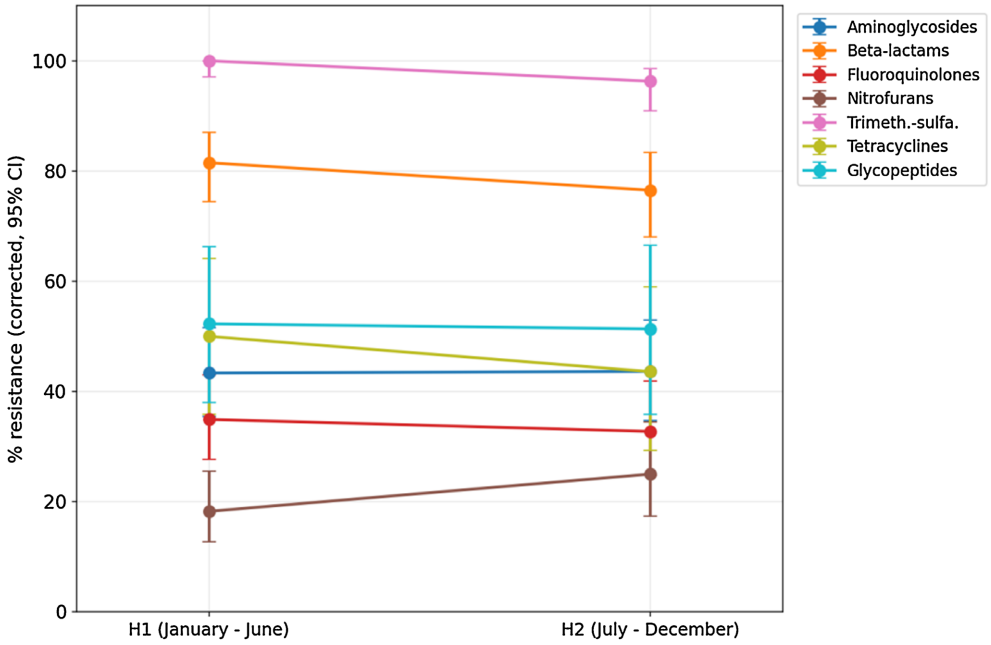
3.5. Temporal Trends

In **Figure 8**, across the four quarters of 2025 (Q1: n = 76; Q2: n = 80; Q3: n = 50; Q4: n = 72 bacterial isolates) and across the two half-years (H1: n = 156; H2: n = 122), no clear directional trend was observed for the major antibiotic classes, with observed fluctuations remaining consistent with sampling variability given the sample sizes per period. Beta-lactam resistance ranged narrowly between 66.7% and 83.3% across quarters, and trimethoprim-sulfamethoxazole resistance remained above 95% in every quarter, suggesting that, unlike the multi-year upward trend documented in Mali over 2005-2023 [15], resistance in this single-year Libreville sample was already at, or close to, a high plateau rather than in active transition.

3.6. Minimum Inhibitory Concentrations

Enterobacterales

In the present study, MIC50 and MIC90 values for the principal antibiotics tested in *Escherichia coli* (n up to 99) and *Klebsiella pneumoniae* (n up to 41). MIC90 values for cefotaxime and ceftriaxone reached the highest tested value (≥64 mg/L) in both species, while the MIC50 remained low (1 mg/L), suggesting



Bacterial isolates, 95% CI. H1: January-June 2025; H2: July-December 2025.

Figure 8. Half-yearly evolution of resistance rate by major antibiotic class.

a bimodal distribution consistent with the coexistence of wild-type strains and ESBL-producing strains within the same population a pattern quantitatively very similar to the roughly one-third ESBL prevalence reported among community-acquired *E. coli* and *K. pneumoniae* in southeastern Gabon [3]. The MIC90 of trimethoprim-sulfamethoxazole (320 mg/L, the maximum value tested) confirms the near-universal resistance observed at the phenotypic level, and mirrors the very high cotrimoxazole resistance repeatedly reported elsewhere in the sub-region [4] [15] (see **Table 1**). Conversely, ertapenem and imipenem retained low MIC50/MIC90 values (≤0.5 mg/L) in both species, consistent with the absence of detectable carbapenem resistance in this series and with the preserved carbapenem susceptibility reported in Togo and Mali [14] [15]. Fosfomycin and nitrofurantoin also showed MIC50 values within the usual susceptibility range for *E. coli*, reinforcing their potential role as oral step-down options for uncomplicated lower urinary tract infection in this setting (see **Table 2**).

3.7. Minimum Inhibitory Concentrations of *Staphylococcus* spp.

In *Staphylococcus aureus* (n = 14) and *Staphylococcus haemolyticus* (n = 31), vancomycin MIC50 and MIC90 were 0.75 - 1.0 mg/L and 1.0 mg/L, respectively, well below the usual resistance threshold (≥16 mg/L) and consistent with preserved vancomycin susceptibility across all tested isolates directly contradicting the initial automated classification suggesting glycopeptide resistance in roughly half of the isolates in this group, as we see in **Table 3** and **Table 4**. Oxacillin MIC50 was 4 mg/L for both species, at the resistance threshold, suggesting a notable proportion of methicillin-resistant strains in this series, to be confirmed with a dedicated

Table 1. Resistance rate by antibiotic class (bacterial isolates, n = 278).

Antibiotic class	n tested	n resistant	% resistance	95% CI
Trimethoprim-sulfamethoxazole	238	234	98.3	95.3 - 99.4
Beta-lactams	261	207	79.3	74.0 - 83.8
Tetracyclines	83	39	47.0	36.6 - 57.6
Aminoglycosides	253	110	43.5	37.5 - 49.6
Fluoroquinolones	259	88	34.0	28.4 - 40.0
Nitrofurans	233	49	21.0	16.2 - 26.7
Fosfomycin	51	3	5.9	2.0 - 15.9
Oxazolidinones	83	1	1.2	0.2 - 6.5
Glycopeptides†	81	0†	0†	0 - 4.5†

Bacterial isolates, 95% CI. *Glycopeptide resistance shown here is based on the automated phenotype flag; see main text direct MIC analysis found no resistance for this class as describe in **Table 1**.

Table 2. MIC50/MIC90 (mg/L), *Staphylococcus aureus* and *S. haemolyticus*.

Antibiotic	Species	n	MIC min	MIC50	MIC90	MIC max
Oxacillin	*S. aureus*	14	0.25	4.0	4.0	4.0
Oxacillin	*S. haemolyticus*	31	0.25	4.0	4.0	4.0
Vancomycin	*S. aureus*	14	0.50	0.75	1.0	1.0
Vancomycin	*S. haemolyticus*	31	0.50	1.0	1.0	1.0
Gentamicin	*S. aureus*	14	0.50	0.50	0.50	0.50
Gentamicin	*S. haemolyticus*	31	0.50	2.0	4.0	16.0
Erythromycin	*S. aureus*	14	0.25	8.0	8.0	8.0
Erythromycin	*S. haemolyticus*	31	0.25	8.0	8.0	8.0
Clindamycin	*S. aureus*	14	0.12	0.25	0.25	0.25
Clindamycin	*S. haemolyticus*	31	0.12	0.25	0.25	8.0
Linezolid	*S. aureus*	6	2.0	2.0	2.0	2.0
Linezolid	*S. haemolyticus*	16	1.0	2.0	2.0	2.0
Tetracycline	*S. aureus*	14	1.0	1.0	1.0	16.0
Tetracycline	*S. haemolyticus*	31	1.0	16.0	16.0	16.0
Trimethoprim-sulfamethoxazole	*S. aureus*	14	10.0	10.0	227.0	320.0
Trimethoprim-sulfamethoxazole	*S. haemolyticus*	31	10.0	80.0	320.0	320.0

All antibiotics has noticed in **Table 2**, tested on *Staphylococcus aureus* and *Staphylococcus haemolyticus* have MIC (minimum inhibitory concentration). ">X" values. Their presence at the MIC90/MIC max level signals high-level resistance rather than an exact quantitative value.

Table 3. MIC50/MIC90 (mg/L), *Escherichia coli*.

Antibiotic	n	MIC min	MIC50	MIC90	MIC max
Ampicillin	98	2.0	32.0	32.0	32.0
Amikacin	99	1.0	2.0	4.0	16.0
Ceftazidime	99	0.12	1.0	16.0	64.0
Ceftriaxone	49	1.0	1.0	64.0	64.0
Cefotaxime	49	0.25	1.0	64.0	64.0
Cefoxitin	98	4.0	4.0	10.4	64.0
Ciprofloxacin	50	0.06	0.75	4.0	4.0
Ofloxacin	88	0.25	2.0	8.0	8.0
Ertapenem	98	0.12	0.12	0.50	8.0
Imipenem	51	0.25	0.25	0.25	16.0
Gentamicin	98	1.0	1.0	16.0	16.0
Tobramycin	50	1.0	1.0	16.0	16.0
Trimethoprim-sulfamethoxazole	99	20.0	320.0	320.0	320.0
Piperacillin-tazobactam	98	4.0	4.0	64.0	128.0
Ticarcillin	98	8.0	128.0	128.0	128.0
Nitrofurantoin	89	16.0	16.0	16.0	32.0
Fosfomycin	49	16.0	16.0	16.0	256.0

Based on direct interpretation of vancomycin MICs (see MIC section below) rather than the automated derived phenotype variable, *Escherichia coli* has discordant with the observed MICs in Table 3.

Table 4. MIC50/MIC90 (mg/L), *Klebsiella pneumoniae*.

Antibiotic	n	MIC min	MIC50	MIC90	MIC max
Ampicillin	41	2.0	32.0	32.0	32.0
Amikacin	41	1.0	2.0	4.0	16.0
Ceftazidime	41	0.12	1.0	16.0	64.0
Ceftriaxone	21	1.0	1.0	64.0	64.0
Cefotaxime	20	0.25	1.0	64.0	64.0
Cefoxitin	41	4.0	4.0	64.0	64.0
Ciprofloxacin	20	0.06	0.25	1.3	4.0
Ofloxacin	39	0.25	0.25	8.0	8.0
Ertapenem	41	0.12	0.25	0.50	0.50
Imipenem	20	0.25	0.25	0.55	1.0
Gentamicin	41	1.0	1.0	16.0	16.0
Tobramycin	20	1.0	1.0	8.0	16.0

Continued

Trimethoprim-sulfamethoxazole	41	20.0	20.0	320.0	320.0
Piperacillin-tazobactam	40	4.0	4.0	17.6	128.0
Ticarcillin	41	8.0	128.0	128.0	128.0
Nitrofurantoin	39	16.0	64.0	128.0	128.0

MIC: minimum inhibitory concentration. ">X" values were coded as 2 × X per the two-fold dilution convention (see Methods); their presence at the MIC90/MIC max level signals high-level resistance rather than an exact quantitative value has noticed in **Table 4**.

cefoxitin screening test. Tetracycline MIC50 differed markedly between *S. aureus* (1 mg/L, susceptible) and *S. haemolyticus* (16 mg/L, resistant), accounting for most of the overall tetracycline resistance reported above, while linezolid MIC50/MIC90 values of 2 mg/L in both species confirmed the very low oxazolidinone resistance observed at the phenotypic level.

4. Discussion

This study describes a high level of resistance to first-line antibiotics beta-lactams and trimethoprim-sulfamethoxazole in particular, among urogenital bacterial isolates from the southern Libreville region in 2025, with an MIC90 for cefotaxime and ceftriaxone reaching the upper tested limit in both *E. coli* and *K. pneumoniae*, strongly suggestive of a high prevalence of extended-spectrum beta-lactamases (ESBLs), and with 56.5% of all bacterial isolates meeting standard criteria for multidrug resistance. These findings closely mirror those reported at the Omar Bongo Ondimba Army Training Hospital in Libreville, where Yala and colleagues found ESBL production in 74.1% of uropathogenic *Enterobacterales* and resistance to first-line agents of 81.6% [2], and are consistent with the broader Gabonese literature from Franceville and Lambaréné [3]-[6] [8].

The pattern observed here also aligns closely with recent Cameroonian data. The ESBL prevalence of 64% (Douala) and 64.7% (West region) reported by Guemkam and colleagues and by Bayaba and colleagues, respectively [10] [12], sits only slightly below the ESBL signal inferred from the MIC90 values in the present study, while the near-universal fluoroquinolone co-resistance among ESBL producers described by Guemkam *et al.* (81.1%) [10] parallels the 34.0% overall fluoroquinolone resistance, and the markedly elevated MIC90 values for ciprofloxacin, observed here among presumptively ESBL-producing isolates. The molecular tracing of identical CTX-M-type resistance genes across patients, hospital surfaces and wastewater by Koudoum and colleagues in Yaoundé [11] offers a plausible mechanistic explanation applicable equally to the Libreville setting for how such resistance becomes entrenched at the community level rather than remaining confined to hospitalised patients.

Further afield, the trajectory is strikingly similar. In Togo, Bouyo and colleagues reported that 92.4% of 409 Klebsiella isolates from urinary and wound infections were multidrug-resistant, with high-level resistance to cephalosporins (85.8%)

and fluoroquinolones (81.4%) but preserved susceptibility to carbapenems and amikacin (<6%) [14] a resistance hierarchy essentially identical to the one described here, where carbapenems and fosfomycin remained the most reliable options based on MIC data. In Mali, the nearly two-decade Bamako cohort analysed by Cherukumilli and colleagues, in collaboration with the University of Cambridge, documented an increase in third-generation cephalosporin resistance among paediatric Gram-negative *Enterobacterales* from 30% to 93% over roughly fifteen years [15]; if a comparable trajectory has occurred in Gabon, the already high resistance levels recorded in this single-year cross-sectional dataset may represent a point on a still-rising curve rather than a stable plateau, an important caveat for interpreting the temporal analysis presented above.

In the Democratic Republic of the Congo, Mulinganya and colleagues reported that virtually all Enterobacter cloacae isolates and 89% of *Klebsiella pneumoniae* isolates causing neonatal sepsis in Bukavu displayed an ESBL phenotype [16], a rate even higher than that inferred among the *Enterobacterales* in the present series, and a sobering reminder that vulnerable paediatric populations, not specifically captured in the present adult-predominant dataset, may bear a disproportionate share of the regional AMR burden. In Angola, Avelino and colleagues showed that multidrug-resistant pneumococcal disease was an independent predictor of in-hospital mortality among children under five in Luanda [17], reinforcing the broader point also evident in the present dataset's substantial resistance burden among bloodstream as well as urinary isolates that AMR in this part of Africa is not confined to any single specimen type or pathogen. In South Africa, Teixeira and colleagues' four-year review at a North West province hospital found that ciprofloxacin, nitrofurantoin and amoxicillin-clavulanate susceptibility among uropathogens had all fallen below the thresholds assumed by national treatment guidelines [13], echoing the present study's finding that nitrofuran resistance, while lower than for beta-lactams or cotrimoxazole, was nonetheless present in a fifth of tested isolates and therefore cannot be assumed reliable for empirical therapy without local confirmation. Notably, no peer-reviewed surveillance data specific to Chad were identified in PubMed at the time of writing, a gap that is itself consistent with the observation by Murray and colleagues that data scarcity remains most acute in precisely the low-resource settings where the burden of AMR is highest [1]; this absence should be read as a call for expanded microbiological surveillance capacity in Chad, rather than as evidence of a lower regional burden there.

A noteworthy methodological finding of this study concerns the glycopeptide class: the resistance variable derived automatically by the laboratory information system indicated vancomycin resistance in roughly half of the tested *Staphylococcus* spp. isolates, a rate with no equivalent in the available literature, regional or international. According to PubMed, a recent Ethiopian study of 151 S. aureus isolates detected no vancomycin resistance whatsoever (MIC ≤ 2 mg/L for all isolates) [18], and a recent international review reports an average vancomycin resistance rate among human-source bacteria of approximately 0.5% worldwide

[19], with a further Ethiopian study finding only 2.8% vancomycin-resistant Enterococcus among cancer patients [20]. Direct analysis of vancomycin MICs in the present study (MIC50 0.75 - 1 mg/L, MIC90 1 mg/L) confirms preserved susceptibility, in agreement with this international evidence, and invalidates the automated derived variable for this antibiotic class. This finding underscores the importance of systematically cross-checking pre-computed resistance indicators generated by laboratory information systems against raw quantitative data before epidemiological use or publication a precaution that may be equally relevant to other laboratories across the region using similar automated expert systems, including those described in the Cameroonian and Malian studies cited above [10] [11] [15].

Collectively, this regional comparison-spanning Gabon, Cameroon, Togo, Mali, the Democratic Republic of the Congo, Angola and South Africa situate the present findings squarely within a well-documented, continent-wide pattern of high, and in several settings apparently still-rising, resistance to first-line antibacterial agents, tempered by consistently preserved susceptibility to carbapenems, fosfomycin and, once the derived-variable artefact is corrected for, glycopeptides. The consistency of this pattern across countries with markedly different healthcare infrastructures and antibiotic-use practices suggests that shared regional drivers including over-the-counter antibiotic availability, limited diagnostic capacity, and the cross-border circulation of mobile resistance elements such as those characterised by Koudoum and colleagues [11] may be at least as important as any single national policy environment, an interpretation consistent with, though not directly tested by, the present cross-sectional dataset.

5. Limitations

- Single-centre, retrospective design, limiting representativeness for the whole southern Libreville region and precluding causal inference.
- Lack of data on patient age and clinical context (community-acquired vs. nosocomial, prior antibiotic exposure), which would have allowed a more detailed analysis of factors associated with resistance, as performed in several of the comparator studies cited above [3] [5] [15].
- Non-uniform antibiotic testing panel across isolates (dependent on the automated cards used), limiting direct comparability of some class-level resistance rates and reducing statistical power for certain subgroup analyses (specimen type, sex, time period).
- Limited sample sizes for several bacterial species and for certain time periods, warranting cautious interpretation of subgroup comparisons.
- Absence of molecular confirmation (detection of resistance genes, e.g., blaCTX-M, mecA) to confirm the mechanisms suggested by phenotypes and MICs, unlike the genomic approaches used in the Cameroonian and Malian studies discussed above [10] [11] [15].
- The discovery of a discrepancy in the glycopeptide resistance variable warrants

a broader review of all derived variables from the laboratory information system before their use in future analyses.

6. Conclusion

This study confirms, using local 2025 data, a high level of resistance to first-line antibiotics (beta-lactams, trimethoprim-sulfamethoxazole) among the leading uropathogens in the southern Libreville region, consistent with data available from other Gabonese sites and, more broadly, from Cameroon, Togo, Mali, the Democratic Republic of the Congo, Angola and South Africa. Fosfomycin, nitrofurantoin, and carbapenems retain high activity and represent valuable therapeutic options of last resort pending antibiogram results. Strengthened local surveillance, including systematic recording of patient age and clinical context, molecular confirmation of resistance mechanisms, and rigorous validation of laboratory information system outputs, is recommended to improve the quality of antimicrobial resistance surveillance data in the region, alongside expanded microbiological capacity in currently under-represented settings such as Chad.

Data Availability Statement

The data generated in the study were provided in the tables and figures of the manuscript.

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Author Contributions

Farel-Constant Boumas Retiga collected the patient samples, performed the biological analyses, and contributed to writing the article with Guy Judicaël Ella Ndong and Gace Pathy Goulabou Mbandza. Hardy Roger Sibi Matotou and Luice Aurtin Joël James created the database. Marie Andrée N'negue ép. Mezui-Mbeng made substantive and formal revisions to the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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