

Bloodstream Infections Caused by *Klebsiella-Enterobacter-Serratia* Group Enterobacterales at Dalal Jamm National Teaching Hospital (Dakar, Senegal) in 2024: Resistance Profiles and β -Lactam Resistance Phenotypes

Awa Ba Diallo^{1,2*}, Elhadji Bambo Diakhaby³, Harouna Diallo², Fahamia Cheha², Djibril Diop², Fatou Edwige Sall², Mariama Diallo², Omar Gueye², Alioune Tine², Adja Tacko Mane Diallo², Fatou Diagne², Habsa Diagne², Awa Soumare², Dieynaba Barry², Roughyatou Ka⁴, Ndeye Coumba Toure Kane², Makhtar Camara¹

¹Bacteriology-Virology Laboratory, Biological Sciences Department of Pharmacy Cheikh Anta Diop University, Guédiawaye, Senegal

²Bacteriology-Virology Laboratory, Dalal Jamm National Teaching Hospital, Guediawaye, Senegal

³Bacteriology-Virology Laboratory at UFR Health Sciences of Gaston Berger University, Saint Louis, Senegal

⁴Bacteriology-Virology Laboratory at UFR Health Sciences of Iba Der Thiam University, Thies, Senegal

Email: *awa1.diallo@ucad.edu.sn

How to cite this paper: Diallo, A.B., Diakhaby, E.B., Diallo, H., Cheha, F., Diop, D., Sall, F.E., Diallo, M., Gueye, O., Tine, A., Diallo, A.T.M., Diagne, F., Diagne, H., Soumare, A., Barry, D., Ka, R., Kane, N.C.T. and Camara, M. (2026) Bloodstream Infections Caused by *Klebsiella-Enterobacter-Serratia* Group Enterobacterales at Dalal Jamm National Teaching Hospital (Dakar, Senegal) in 2024: Resistance Profiles and β -Lactam Resistance Phenotypes. *Advances in Microbiology*, 16, 439-458. <https://doi.org/10.4236/aim.2026.1610025>

Received: June 30, 2026

Accepted: September 27, 2026

Published: September 30, 2026

Abstract

Background: Enterobacterales of the *Klebsiella-Enterobacter-Serratia* (KES) group are among the leading causes of healthcare-associated bloodstream infections and concentrate resistance mechanisms that compromise empirical therapy. Blood culture-based data from Senegal remain scarce. We aimed to describe the frequency, susceptibility profiles and β -lactam resistance phenotypes of KES-group bloodstream infections at Dalal Jamm National Teaching Hospital. **Methods:** We conducted a retrospective descriptive study of all blood cultures processed in the Bacteriology-Virology laboratory of Dalal Jamm National Teaching Hospital between January and December 2024. The unit of microbiological analysis was the isolate; the unit of clinical description was the blood culture episode, defined as all bottles drawn from one patient within a 14-day window. Isolates were identified using API 20E strips and the Vitek 2 system; antimicrobial susceptibility testing was performed on Vitek 2 and interpreted according to CA-SFM/EUCAST breakpoints. Non-suscepti-



bility combined the intermediate and resistant categories and is reported both by individual agent and by class. β -lactam resistance phenotypes were inferred from antibiogram profiles using a pre-specified algorithm that required a cefoxitin result; isolates without one were reported as not classifiable. Multidrug resistance was defined according to Magiorakos *et al.*, after exclusion of species-specific intrinsic resistance. **Results:** Of 1844 blood cultures processed, 694 (37.6%) were positive and 589 isolates were considered significant after exclusion of 105 contaminants. Gram-negative bacilli accounted for 386 isolates (65.5%), of which 117 belonged to the KES group, representing 30.3% of Gram-negative bacilli and 19.9% of all significant isolates. Four isolates had no analysable antibiogram, leaving 113 isolates recovered from 110 blood culture episodes in at least 103 patients. *Enterobacter* spp. predominated ($n = 53$; 46.9%), closely followed by *Klebsiella pneumoniae* ($n = 51$; 45.1%) and then *Serratia* spp. ($n = 9$; 8.0%); only 10 of the 53 *Enterobacter* isolates (18.9%) carried a species-level identification. Paediatric units (neonatology, general paediatrics and paediatric oncology) accounted for 39.1% of episodes, ahead of the intensive care unit (14.5%) and clinical haematology (12.7%); 40.9% of episodes concerned children aged 10 years or younger. Median time to positivity was 1 day (IQR 1 - 2). Non-susceptibility to third-generation cephalosporins reached 94.1% in *K. pneumoniae*, 66.0% in *Enterobacter* spp. and 33.3% in *Serratia* spp. Carbapenem non-susceptibility was detected in 21 of 108 isolates tested (19.4%). Class-level aminoglycoside non-susceptibility (69.4% in *K. pneumoniae*) concealed a marked within-class divergence: amikacin was active against all 28 *K. pneumoniae* isolates tested (0% non-susceptible), and only 4 of 31 *Enterobacter* isolates were non-susceptible (12.9%), whereas gentamicin and tobramycin were non-susceptible in 58% - 75%. An ESBL-compatible phenotype was retained in 18 *K. pneumoniae* (35.3%) and 4 *Enterobacter* isolates (7.5%), with a further 35.3% and 20.8% respectively not classifiable because cefoxitin had not been tested. Multidrug resistance affected 94.1% of *K. pneumoniae*, 69.8% of *Enterobacter* spp. and 22.2% of *Serratia* spp. **Conclusion:** KES-group bloodstream infections are frequent at Djal Jamm hospital, concentrate in paediatric and critical care units, and are dominated by multidrug-resistant isolates. The observed level of resistance to third-generation cephalosporins makes their empirical use inappropriate in this hospital, while the preserved activity of amikacin identifies a carbapenem-sparing option that deserves formal evaluation. The emergence of carbapenem non-susceptible isolates calls for a coordinated strengthening of microbiological surveillance, infection prevention and control, and antimicrobial stewardship, together with routine cefoxitin testing and molecular confirmation of resistance mechanisms.

Keywords

Bloodstream Infection, Blood Culture, *Klebsiella pneumoniae*, *Enterobacter*, *Serratia*, Extended-Spectrum β -Lactamase, Carbapenems, MDR, Senegal

1. Introduction

Bloodstream infections are among the most severe infections encountered in hospital settings. They constitute a diagnostic and therapeutic emergency, and their outcome depends closely on how rapidly and how appropriately antimicrobial therapy is initiated [1] [2]. Their diagnosis rests on the isolation of a micro-organism from blood cultures, which remains the reference method for documenting sepsis and guiding treatment [3].

Among the causative agents, Enterobacterales occupy a major place. The *Klebsiella-Enterobacter-Serratia* (KES) group brings together opportunistic species frequently implicated in healthcare-associated infections, particularly in patients with invasive devices, immunocompromised patients and those admitted to critical care units [4] [5]. *Klebsiella pneumoniae* is now recognised as one of the principal global vehicles for the dissemination of antimicrobial resistance, owing to its capacity to acquire and spread resistance plasmids [6]. Species of the *Enterobacter cloacae* complex are characterised by an inducible chromosomal AmpC cephalosporinase, whose derepression under selective pressure compromises the efficacy of third-generation cephalosporins (3GC) [7] [8]. *Serratia marcescens*, although less frequent, retains clinical importance because of its persistence in the hospital environment and its association with device-related infections [9].

The emergence of extended-spectrum β -lactamases (ESBLs) and subsequently of carbapenemases has profoundly changed the management of these infections [10] [11]. The worldwide dissemination of these mechanisms is now documented by international surveillance systems [12] [13]. It is of particular concern in sub-Saharan Africa, where access to alternative therapeutic options and to molecular confirmation techniques remains limited and where resistance bears directly on the survival of bacteraemic patients. In Senegal, ESBL-producing Enterobacterales have been documented in hospital settings for more than a decade [14], but recent data derived specifically from blood cultures remain scarce.

Dalal Jamm National Teaching Hospital in Guédiawaye is a referral facility serving the Dakar suburbs, with intensive care, clinical haematology, nephrology and several paediatric units caring for patients at high infectious risk. We therefore undertook this work to describe the burden of bloodstream infections caused by KES-group Enterobacterales diagnosed in this hospital during 2024, to analyse the antimicrobial susceptibility profiles of the isolates and to determine the main β -lactam resistance phenotypes.

2. Materials and Methods

2.1. Study Setting

The study was conducted in the Bacteriology-Virology laboratory of the Department of Medical Biology at Dalal Jamm National Teaching Hospital, located at Ancien Parcours du Golf, Guédiawaye (Dakar region, Senegal). This teaching hospital comprises a general intensive care unit, clinical haematology, nephrology and haemodialysis, cardiology and infectious diseases departments, as well as sev-

eral paediatric units (neonatology, general paediatrics, paediatric oncology and paediatric surgery). The laboratory provides diagnostic bacteriology services for all of these departments.

2.2. Study Design, Population and Eligibility Criteria

This was a retrospective descriptive study covering the period from 1 January to 31 December 2024. All patients admitted to Dalal Jamm National Teaching Hospital, irrespective of age or sex, from whom at least one blood culture was drawn during the study period and in whom a KES-group organism was isolated, were included. Specimens other than blood cultures were excluded. Blood cultures whose isolate was judged to be a contaminant according to routine laboratory criteria (commensal skin flora recovered from a single bottle in the absence of supporting clinical evidence) were not counted in the denominator of significant isolates.

Units of analysis were defined a priori and are used consistently throughout. A blood culture bottle is a single vial; a blood culture set is the group of bottles filled at one venepuncture; a blood culture request (the unit recorded by the laboratory information system) covers the set or sets received under one accession number, and is what the laboratory reports as a single positive or negative culture. An isolate is one organism recovered from one request. A blood culture episode was defined as all requests from the same patient within a 14-day window, the first positive request marking the start of the episode; a new positive request beyond that window was counted as a new episode.

Because the laboratory information system in use over the study period records an accession number rather than a unique patient identifier, de-duplication could be performed reliably at the level of the request but not at the level of the patient. Requests recorded twice under the same accession number were merged before analysis. Patient and episode characteristics are therefore reported per episode, and microbiological results per isolate; the two denominators differ because a single episode may yield more than one KES organism. The number of distinct patients is reported as a range, bounded below by grouping episodes that shared an identical requesting ward, age and sex within 14 days.

2.3. Specimens and Microbiological Methods

Blood was drawn at the bedside under aseptic conditions, distributed into adult or paediatric blood culture bottles, and incubated in an automated system (BACTEC or BACT/ALERT) at 35 - 37°C under aerobic conditions. Any detected growth prompted microscopic examination after Gram staining and subculture onto chocolate agar, MacConkey agar and EMB agar.

Bacterial identification was performed using API 20E biochemical strips or the automated Vitek 2 system (GN cards). Antimicrobial susceptibility testing was carried out on Vitek 2 (AST-N cards), supplemented where appropriate by disc diffusion. Results were categorised as susceptible (S), intermediate (I) or resistant

(R) according to the prevailing recommendations of the Antibiogram Committee of the French Society for Microbiology (CA-SFM/EUCAST) [15]. The agents actually reported over the study period were: aminopenicillins (amoxicillin, ampicillin); amoxicillin-clavulanate; ticarcillin and ticarcillin-clavulanate; piperacillin and piperacillin-tazobactam; the cephamycin cefoxitin; third-generation cephalosporins (cefotaxime, ceftriaxone, ceftazidime, cefixime); the fourth-generation cephalosporin cefepime; the monobactam aztreonam; carbapenems (imipenem, meropenem, ertapenem); aminoglycosides (gentamicin, tobramycin, amikacin, kanamycin); fluoroquinolones (ciprofloxacin, levofloxacin, ofloxacin, norfloxacin, pefloxacin, moxifloxacin); co-trimoxazole and trimethoprim; fosfomycin; nitrofurantoin; tetracyclines (doxycycline, tetracycline); and chloramphenicol. Panels were not uniform across the study period, so the set of agents tested varies between isolates. Time to positivity was recorded by the automated system as the number of days elapsed between bottle loading and detection of bacterial growth; a value of 0 or 1 day was taken as positivity within 24 hours and a value of 2 days as positivity between 25 and 48 hours.

2.4. Data Collection and Analysis

Data were extracted from the laboratory information system (FileMaker Pro version 21) and exported to a spreadsheet for analysis. The variables collected were: anonymised specimen identifier, age, sex, requesting department, clinical indication for the request, sampling date, time to positivity, isolated species and detailed susceptibility results.

Results were analysed by individual antimicrobial agent, by antimicrobial class and by species. In line with standard practice in resistance epidemiology, the intermediate and resistant categories were combined under the term non-susceptibility (non-S), and percentages were calculated using the number of isolates actually tested for each agent or class as the denominator. For class-level results an isolate was considered non-susceptible as soon as at least one agent of that class was categorised as I or R. This convention is deliberately conservative and, where the agents of a class do not behave uniformly, it overstates resistance to the class as a whole; class-level results are therefore accompanied by the corresponding agent-level results, and the proportion of isolates showing discordant categorisations within a class is reported. Categorical variables are expressed as counts and percentages, and continuous variables as median and interquartile range (IQR). Denominators are stated for every proportion, and isolates for which a given agent or variable was not documented are excluded from that denominator and reported separately rather than counted as susceptible.

2.5. Definition of Resistance Phenotypes and Multidrug Resistance

β -lactam resistance phenotypes were inferred from antibiogram profiles using the following hierarchical algorithm, applied in the order shown:

- carbapenem non-susceptibility: at least one carbapenem (imipenem, meropenem or ertapenem) categorised as I or R; this category takes precedence over the others;
- ESBL-type phenotype: non-susceptibility to at least one third-generation cephalosporin, with a ceftazidime result of S and no carbapenem non-susceptibility;
- AmpC-type phenotype ($\pm$ associated ESBL): non-susceptibility to at least one third-generation cephalosporin, with a ceftazidime result of I or R and no carbapenem non-susceptibility;
- wild-type phenotype: susceptibility preserved to all third-generation cephalosporins tested;
- not classifiable: non-susceptibility to at least one third-generation cephalosporin without a documented ceftazidime result, or no third-generation cephalosporin tested. Isolates in this category were reported separately and were not redistributed among the other phenotypes.

These phenotypes are reported as probable, given the absence of systematic synergy testing and of molecular confirmation.

Because *Enterobacter* spp. and *Serratia* spp. possess an inducible chromosomal AmpC cephalosporinase, ceftazidime non-susceptibility in these genera does not discriminate between a derepressed cephalosporinase and an associated ESBL. The ESBL-type and AmpC-type labels are therefore mechanistically informative only for *K. pneumoniae*, which lacks a chromosomal AmpC, and are reported for the other two genera as a descriptive summary of the antibiogram.

Multidrug resistance (MDR) was defined according to Magiorakos *et al.* as non-susceptibility to at least one agent in at least three antimicrobial categories, after exclusion of species-specific intrinsic resistance (aminopenicillins for *K. pneumoniae*; aminopenicillins, penicillin-inhibitor combinations and cephamycins for *Enterobacter* spp.; with the addition of polymyxins, nitrofurantoin and tetracyclines for *Serratia* spp.) [16] [17].

2.6. Ethical Considerations

The study relied exclusively on the retrospective use of laboratory data generated during routine care, with no additional intervention on patients. Data were anonymised before analysis and handled confidentially. The protocol was submitted to and authorised by the management of Dalal Jamm National Teaching Hospital. Given the retrospective and anonymised nature of the study, individual informed consent was not required.

3. Results

3.1. Overview of Blood Cultures

During 2024, the Bacteriology-Virology laboratory of Dalal Jamm National Teaching Hospital processed 1844 blood cultures, of which 694 (37.6%) were positive and 1150 (62.4%) negatives. After exclusion of 105 isolates considered to be contaminants (15.1% of positive blood cultures), 589 isolates were retained as sig-

nificant: 386 Gram-negative bacilli (65.5%), 201 Gram-positive cocci (34.1%) and 2 yeasts (0.3%).

KES-group Enterobacterales accounted for 117 isolates, that is 30.3% of Gram-negative bacilli and 19.9% of all significant isolates. Four of these isolates had no analysable antibiogram and were excluded, leaving 113 isolates that constituted the study population. These 113 isolates were recovered from 110 blood culture episodes: three episodes yielded two distinct KES organisms each, and a further three episodes yielded a non-KES co-isolate (*Acinetobacter baumannii*, *Escherichia coli* and *Sphingomonas paucimobilis*, one each) that was not analysed here. Because the laboratory information system does not carry a unique patient identifier, episodes were de-duplicated at the level of the blood culture request; six groups of episodes sharing an identical ward, age and sex fell within 14 days of one another, so the 110 episodes correspond to at least 103 and at most 110 distinct patients (Figure 1).

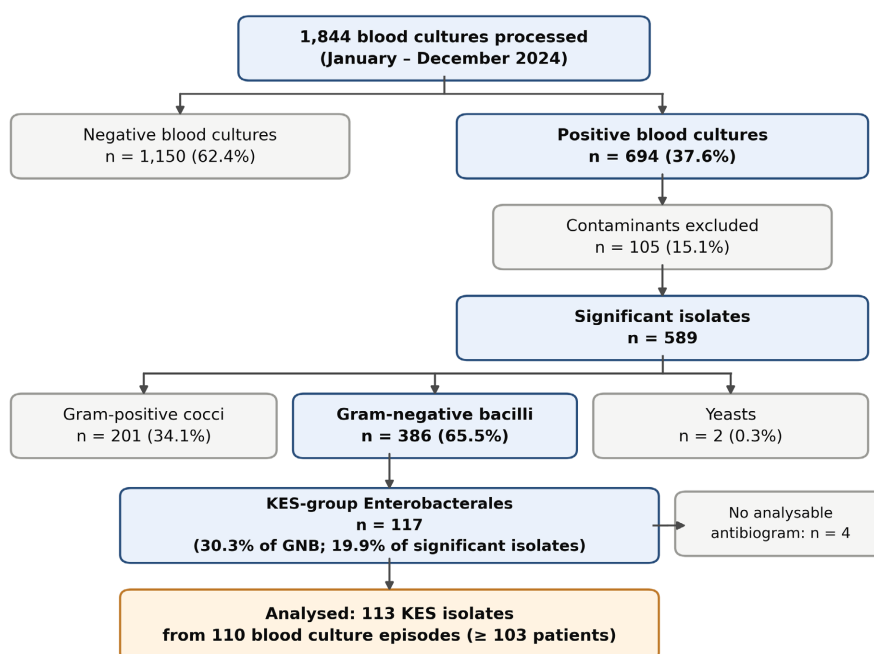


Figure 1. Flow diagram of blood cultures processed in the Bacteriology-Virology laboratory of Dalal Jamm National Teaching Hospital, January-December 2024. KES: *Klebsiella-Enterobacter-Serratia* group; GNB: Gram-negative bacilli. Percentages of Gram-negative bacilli and of significant isolates refer to the 117 KES isolates identified.

3.2. Patient and Episode Characteristics

Characteristics of the 110 blood culture episodes and of the 113 isolates they yielded are presented in Table 1. A slight female predominance was observed (59 episodes in female patients; 53.6%), giving a male-to-female ratio of 0.86. Median age was 20 years (IQR 0.4 - 50; range 1 day - 96 years). Children aged 10 years or younger accounted for 40.9% of episodes (n = 45), including 28 infants under one year of age (25.5% of the total); patients older than 60 years accounted for 12.7%

of episodes.

Paediatric units taken together (neonatology, general paediatrics and paediatric oncology) accounted for 43 episodes, that is 39.1% of cases, with neonatology alone contributing 23 episodes (20.9%). Next came the intensive care unit (n = 16; 14.5%), clinical haematology (n = 14; 12.7%), general paediatrics (n = 12; 10.9%), cardiology (n = 10; 9.1%) and nephrology (n = 9; 8.2%). Febrile infectious syndrome was the leading reason for requesting a blood culture, followed by neonatal infections and febrile episodes occurring in haematology patients.

Time to positivity was documented for 106 of the 110 episodes (96.4%). Among these, the median was 1 day (IQR 1 - 2; range 0 - 7), and 89 of 106 bottles (84.0%) flagged positive within the first 48 hours of incubation.

Table 1. Characteristics of patients and of KES-group isolates recovered from blood cultures (n = 113) in 2024.

Characteristic	n	%
Sex (per episode, n = 110)		
Female	59	53.6
Male	51	46.4
Age (years, per episode, n = 110)		
<1	28	25.5
1 - 10	17	15.5
11 - 20	11	10.0
21 - 30	12	10.9
31 - 40	3	2.7
41 - 50	12	10.9
51 - 60	13	11.8
>60	14	12.7
Requesting department (per episode, n = 110)		
Neonatology	23	20.9
Intensive care	16	14.5
Clinical haematology	14	12.7
General paediatrics	12	10.9
Cardiology	10	9.1
Nephrology	9	8.2
Paediatric oncology	8	7.3
Infectious diseases	4	3.6
Internal medicine	4	3.6
Emergency department	3	2.7
Other departments a	3	2.7
Not specified	4	3.6

Continued

Species isolated (per isolate, n = 113)		
<i>Enterobacter</i> spp. (genus level only)	43	38.1
<i>Enterobacter cloacae</i>	10	8.8
<i>Klebsiella pneumoniae</i>	51	45.1
<i>Serratia marcescens</i>	8	7.1
<i>Serratia plymuthica</i>	1	0.9
Time to positivity (per episode)		
≤24 h	73	68.9 b
25 - 48 h	16	15.1 b
>48 h	17	16.0 b
Documented, total	106	100 b
Not documented	4	-
All episodes	110	

a. Dermatology, gynaecology and orthopaedics (1 case each). b. Percentages calculated on the 106 episodes for which time to positivity was documented; the 4 episodes without a documented value are shown separately and are not included in the denominator. Sex, age and requesting department are reported per episode (n = 110); the species distribution is reported per isolate (n = 113), since three episodes yielded two KES isolates each.

3.3. Species Distribution

Enterobacter spp. was the most frequently isolated group (n = 53; 46.9%), very closely followed by *Klebsiella pneumoniae* (n = 51; 45.1%) and then by *Serratia* spp. (n = 9; 8.0%). Together, the first two accounted for 92% of isolates in the group. Identification was resolved to species level for only 10 of the 53 *Enterobacter* isolates (18.9%), all reported as *Enterobacter cloacae*; the remaining 43 (81.1%) were reported at genus level as *Enterobacter* spp., and no isolate was assigned to a species complex. Among the *Serratia* isolates, 8 were *Serratia marcescens* and 1 was *Serratia plymuthica*.

3.4. Antimicrobial Susceptibility Profiles

Non-susceptibility rates by antimicrobial class and species are detailed in **Table 2** and illustrated in **Figure 2**.

Non-susceptibility to third-generation cephalosporins was very high in *K. pneumoniae* (48/51; 94.1%) and frequent in *Enterobacter* spp. (35/53; 66.0%), whereas it remained lower in *Serratia* spp. (3/9; 33.3%). These class-level figures were robust: among the 100 isolates for which at least two third-generation cephalosporins were tested, only 4 (4.0%) gave discordant categorisations, and non-susceptibility rested on a resistant rather than an intermediate result in 84 of the 86 non-susceptible isolates. Cefepime offered no decisive advantage in *K. pneumoniae* (20 of 22 isolates non-susceptible; 90.9%). Non-susceptibility to cefoxitin clearly separated the two main groups: 85.7% in *Enterobacter* spp. (30/35) versus

20.7% in *K. pneumoniae* (6/29), consistent with expression of a chromosomal cephalosporinase in the former. Piperacillin-tazobactam was inactive against 70.3% of *K. pneumoniae* and 60.0% of *Enterobacter* spp.

Carbapenems remained the most active β -lactams, although non-susceptibility was found in 21 of 108 tested isolates (19.4%), distributed comparably across *K. pneumoniae* (10/49; 20.4%), *Enterobacter* spp. (9/50; 18.0%) and *Serratia* spp. (2/9; 22.2%). This figure should be read with caution: 14 of the 21 non-susceptible isolates were classified on the basis of a single carbapenem, in 6 cases ertapenem alone, and 4 were categorised intermediate rather than resistant. Only 2 of the 44 isolates tested against at least two carbapenems gave discordant results.

Among non- β -lactam agents, class-level non-susceptibility to fluoroquinolones reached 85.7% in *K. pneumoniae* (42/49) versus 41.5% in *Enterobacter* spp. (22/53), and class-level non-susceptibility to aminoglycosides 69.4% (34/49) and 55.1% (27/49) respectively. The aminoglycoside figure is, however, the clearest illustration of the limits of a class-level rule: 38 of the 77 isolates tested against at least two aminoglycosides (49.4%) gave discordant results within the class. Agent by agent, amikacin was non-susceptible in none of the 28 *K. pneumoniae* isolates tested (0%) and in only 4 of 31 *Enterobacter* isolates (12.9%), whereas gentamicin was non-susceptible in 25/39 *K. pneumoniae* (64.1%) and 21/36 *Enterobacter* (58.3%), and tobramycin in 21/28 (75.0%) and 15/25 (60.0%) respectively. Cotrimoxazole was inactive against 85.7% of *K. pneumoniae* (24/28). Oral alternatives offered little fallback: non-susceptibility to fosfomycin exceeded 77% in both main groups, and non-susceptibility to tetracyclines exceeded 63%. Nitrofurantoin retained activity against a majority of *K. pneumoniae* isolates (10 of 17 susceptible), without clinical relevance in the context of systemic infection.

Agent-level results for the classes whose members did not behave uniformly are given in the lower part of **Table 2**.

Table 2. Non-susceptibility (intermediate and resistant categories combined) of KES-group isolates by antimicrobial class and species.

Antimicrobial class or agent	<i>K. pneumoniae</i> (n = 51)	<i>Enterobacter</i> spp. (n = 53)	<i>Serratia</i> spp. (n = 9)	All isolates (n = 113)
By class				
Aminopenicillins	45/45 (100.0)	43/43 (100.0)	5/6 (83.3)	93/94 (98.9)
Amoxicillin-clavulanate	27/46 (58.7)	27/43 (62.8)	5/6 (83.3)	59/95 (62.1)
Ticarcillin	47/47 (100.0)	31/45 (68.9)	2/8 (25.0)	80/100 (80.0)
Piperacillin	11/11 (100.0)	15/19 (78.9)	0/1 (0.0)	26/31 (83.9)
Piperacillin-tazobactam	26/37 (70.3)	15/25 (60.0)	2/7 (28.6)	43/69 (62.3)
Cefoxitin	6/29 (20.7)	30/35 (85.7)	0/1 (0.0)	36/65 (55.4)
Third-generation cephalosporins	48/51 (94.1)	35/53 (66.0)	3/9 (33.3)	86/113 (76.1)
Cefepime	20/22 (90.9)	14/26 (53.8)	0/2 (0.0)	34/50 (68.0)
Aztreonam	18/19 (94.7)	8/13 (61.5)	0/1 (0.0)	26/33 (78.8)

Continued

Carbapenems	10/49 (20.4)	9/50 (18.0)	2/9 (22.2)	21/108 (19.4)
Aminoglycosides	34/49 (69.4)	27/49 (55.1)	7/8 (87.5)	68/106 (64.2)
Fluoroquinolones	42/49 (85.7)	22/53 (41.5)	2/9 (22.2)	66/111 (59.5)
Co-trimoxazole/trimethoprim	24/28 (85.7)	17/25 (68.0)	1/8 (12.5)	42/61 (68.9)
Fosfomycin	7/9 (77.8)	15/18 (83.3)	1/1 (100.0)	23/28 (82.1)
Nitrofurantoin	7/17 (41.2)	4/7 (57.1)	6/7 (85.7)	17/31 (54.8)
Tetracyclines	7/11 (63.6)	13/18 (72.2)	-	20/29 (69.0)
Chloramphenicol	3/7 (42.9)	4/13 (30.8)	-	7/20 (35.0)
By agent, for classes with non-uniform behaviour				
<i>Third-generation cephalosporins</i>				
Cefotaxime	36/39 (92.3)	22/39 (56.4)	1/6 (16.7)	59/84 (70.2)
Ceftriaxone	13/13 (100.0)	13/17 (76.5)	0/2 (0.0)	26/32 (81.2)
Ceftazidime	34/37 (91.9)	23/36 (63.9)	2/8 (25.0)	59/81 (72.8)
Cefixime	8/9 (88.9)	9/18 (50.0)	1/2 (50.0)	18/29 (62.1)
<i>Carbapenems</i>				
Imipenem	6/31 (19.4)	5/23 (21.7)	1/1 (100.0)	12/55 (21.8)
Meropenem	1/16 (6.2)	1/23 (4.3)	0/1 (0.0)	2/40 (5.0)
Ertapenem	6/26 (23.1)	5/23 (21.7)	1/8 (12.5)	12/57 (21.1)
<i>Aminoglycosides</i>				
Gentamicin	25/39 (64.1)	21/36 (58.3)	1/7 (14.3)	47/82 (57.3)
Tobramycin	21/28 (75.0)	15/25 (60.0)	6/7 (85.7)	42/60 (70.0)
Amikacin	0/28 (0.0)	4/31 (12.9)	6/7 (85.7)	10/66 (15.2)
Kanamycin	6/11 (54.5)	1/6 (16.7)	-	7/17 (41.2)
<i>Fluoroquinolones</i>				
Ciprofloxacin	31/37 (83.8)	14/30 (46.7)	1/8 (12.5)	46/75 (61.3)
Levofloxacin	12/18 (66.7)	9/21 (42.9)	0/3 (0.0)	21/42 (50.0)
Ofloxacin	19/21 (90.5)	9/16 (56.2)	2/6 (33.3)	30/43 (69.8)
Norfloxacin	8/13 (61.5)	2/16 (12.5)	-	10/29 (34.5)
Pefloxacin	2/4 (50.0)	2/13 (15.4)	-	4/17 (23.5)
Moxifloxacin	1/1 (100.0)	2/3 (66.7)	0/1 (0.0)	3/5 (60.0)

Results are expressed as number of non-susceptible isolates/number of isolates tested (percentage). An isolate is considered non-susceptible to a class as soon as at least one agent of that class is categorised as I or R; agent-level results are given for the classes in which the agents did not behave uniformly. Denominators differ between rows because susceptibility panels were not uniform over the study period. -: no isolate tested.

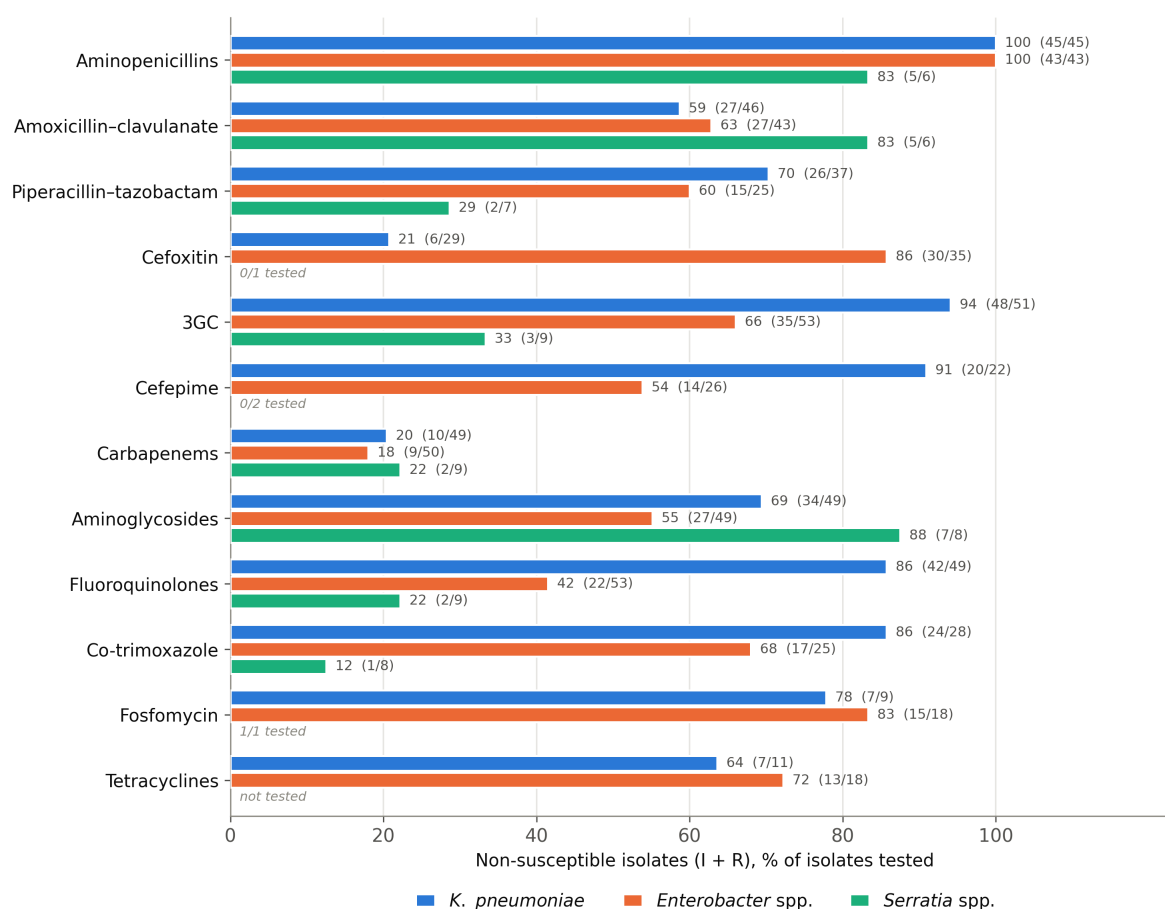


Figure 2. Non-susceptibility rates to the main antimicrobial classes by species, KES-group blood culture isolates (n = 113). 3GC: third-generation cephalosporins. Denominators vary according to the agents tested (see Table 2); combinations tested on fewer than five isolates are shown as a fraction rather than as a percentage.

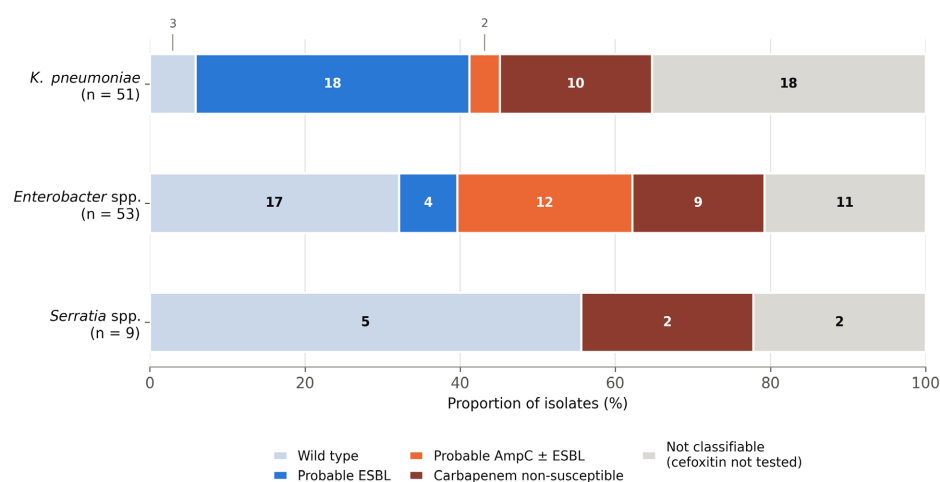
3.5. β -Lactam Resistance Phenotypes

The distribution of β -lactam resistance phenotypes is presented in Table 3 and Figure 3. In *K. pneumoniae*, 18 isolates (35.3%) met the criteria for an ESBL-type phenotype and 2 (3.9%) those for an AmpC-type phenotype $\pm$ ESBL; 10 isolates (19.6%) were carbapenem non-susceptible and 3 (5.9%) were wild type. The remaining 18 isolates (35.3%) could not be classified because cefoxitin had not been tested, even though all 18 were non-susceptible to third-generation cephalosporins. Restricted to the 20 *K. pneumoniae* isolates for which the ESBL/AmpC distinction could actually be made, an ESBL-type phenotype accounted for 18 (90.0%). In *Enterobacter* spp., the distribution was more balanced: 17 wild-type isolates (32.1%), 12 AmpC-type phenotypes $\pm$ ESBL (22.6%), 11 not classifiable for want of a cefoxitin result (20.8%), 9 carbapenem non-susceptible isolates (17.0%) and 4 ESBL-type phenotypes (7.5%). Among *Serratia* spp., 5 of 9 isolates (55.6%) displayed a wild-type phenotype, 2 were carbapenem non-susceptible and 2 were not classifiable. Overall, cefoxitin was not tested in 20 of the 48 third-generation-cephalosporin non-susceptible *K. pneumoniae* isolates (41.7%), in 13 of 35 such *Enterobacter* isolates (37.1%) and in all 3 such *Serratia* isolates.

Table 3. Distribution of probable β -lactam resistance phenotypes by species, n (%).

Phenotype	<i>K. pneumoniae</i> (n = 51)	<i>Enterobacter</i> spp. (n = 53)	<i>Serratia</i> spp. (n = 9)
Wild type	3 (5.9)	17 (32.1)	5 (55.6)
Probable ESBL	18 (35.3)	4 (7.5)	0 (0.0)
Probable AmpC $\pm$ ESBL	2 (3.9)	12 (22.6)	0 (0.0)
Carbapenem non-susceptible	10 (19.6)	9 (17.0)	2 (22.2)
Not classifiable (cefotaxime not tested) a	18 (35.3)	11 (20.8)	2 (22.2)
Total	51	53	9

ESBL: extended-spectrum β -lactamase. Phenotypes were assigned by the hierarchical algorithm defined in the Methods, in which carbapenem non-susceptibility takes precedence and an ESBL-type or AmpC-type assignment requires a documented cefotaxime result. a. Isolates non-susceptible to at least one third-generation cephalosporin, not carbapenem non-susceptible, for which cefotaxime was not tested. In *Enterobacter* spp. and *Serratia* spp., which possess an inducible chromosomal AmpC cephalosporinase, cefotaxime non-susceptibility does not distinguish a derepressed cephalosporinase from an associated ESBL; the phenotypes shown for these genera should be read as descriptive only.

**Figure 3.** Distribution of probable β -lactam resistance phenotypes by species. Carbapenem non-S: non-susceptibility to at least one carbapenem. Not classifiable: cefotaxime not tested.

3.6. Multidrug Resistance

The proportion of multidrug-resistant isolates as defined by Magiorakos *et al.* was 94.1% in *K. pneumoniae* (48/51), 69.8% in *Enterobacter* spp. (37/53) and 22.2% in *Serratia* spp. (2/9), amounting to 77.0% of all KES-group isolates (87/113) (**Table 4**).

Table 4. Frequency of multidrug resistance among KES-group blood culture isolates.

Species	Isolates analysed (n)	Multidrug-resistant isolates n (%)
<i>Klebsiella pneumoniae</i>	51	48 (94.1)
<i>Enterobacter</i> spp.	53	37 (69.8)
<i>Serratia</i> spp.	9	2 (22.2)
All KES-group isolates	113	87 (77.0)

4. Discussion

This study, covering a full year of blood culture activity in a Senegalese national hospital, highlights four main findings: the substantial share of the KES group among documented bloodstream infections, the concentration of cases in paediatric and critical care units, a level of β -lactam resistance that calls usual empirical regimens into question, and the preserved activity of amikacin in a setting where almost every other first-line option has been eroded.

KES-group Enterobacterales accounted for 30.3% of Gram-negative bacilli and close to one in five significant isolates. This proportion is consistent with data reported from other African hospital settings, where Gram-negative bacilli dominate the ecology of nosocomial infection [18] [19]. In Indonesia, Enterobacterales were responsible for 61.9% of documented bloodstream infections in a burn unit [20]. The near numerical equivalence between *Enterobacter* spp. and *K. pneumoniae* observed here is, by contrast, less usual: in most series *K. pneumoniae* clearly outnumbers *Enterobacter* spp. This feature could reflect an in-hospital circulation specific to the institution and would warrant investigation by molecular typing. It must, however, be interpreted with caution, since 81.1% of our *Enterobacter* isolates were identified only to genus level; a genus-level label may group together members of the *Enterobacter cloacae* complex and neighbouring genera that current taxonomy separates, and species-level or complex-level identification would be needed before drawing epidemiological conclusions from this balance.

The strong representation of paediatric units (39.1% of episodes, of which 20.9% from neonatology alone) and the high proportion of infants under one year of age (25.5%) constitute the most striking finding of this series. It is most plausibly explained by the combination of neonatal immune immaturity, the frequency of catheters and intravenous lines, prematurity, and the density of paediatric provision within the hospital. This pattern is not peculiar to our institution. In a paediatric hospital in Dakar, hospital-acquired Enterobacterales bloodstream infections were dominated by ESBL-producing organisms, with ESBL rates of 82% in *Klebsiella* spp. and 88% in *Enterobacter* spp. and a case fatality of 54.8% in ESBL cases [21]; and in a recent Gambian teaching-hospital series, neonates and children accounted for 56% of all bloodstream infections, with 90% of *Klebsiella* isolates producing an ESBL [22]. Multicountry neonatal sepsis studies place *K. pneumoniae* first among causative organisms across Africa and South Asia [23]. Our results are therefore best read as a local expression of a regional pattern, amplified by this hospital's case mix, rather than as an isolated observation. The distinction between community-onset neonatal bacteraemia and healthcare-associated infection could not be established from laboratory data alone; it constitutes a priority question for future work, since preventive measures differ radically between the two.

The most concerning finding is resistance to third-generation cephalosporins, affecting 94.1% of *K. pneumoniae* and 66.0% of *Enterobacter* spp. In *K. pneumoniae*, an ESBL-type phenotype accounted for 90.0% of the isolates in which the

ESBL/AmpC distinction could be made, reflecting a broad dissemination of these enzymes within the hospital and echoing observations already made in Senegal on ESBL-producing Enterobacterales in hospital settings [14] [21], the interpretive reading of such profiles resting on well-established criteria [24]. That distinction could nonetheless be made in only 20 of 51 *K. pneumoniae* isolates, because cefoxitin was omitted from the panel in 41.7% of the third-generation-cephalosporin non-susceptible isolates of this species; the ESBL burden reported here is therefore a floor rather than an estimate, and systematic cefoxitin testing is the single cheapest corrective available to the laboratory. In *Enterobacter* spp., combined non-susceptibility to third-generation cephalosporins and cefoxitin in 85.7% of tested isolates suggests derepressed expression of the chromosomal AmpC cephalosporinase, a mechanism intrinsic to this genus and selected by β -lactam exposure [7] [8]; in this genus, however, cefoxitin non-susceptibility cannot separate a derepressed cephalosporinase from an associated ESBL, so the phenotypes we report for *Enterobacter* spp. and *Serratia* spp. are descriptive rather than mechanistic. This distinction has direct therapeutic consequences: in infections caused by AmpC-producing Enterobacterales, use of third-generation cephalosporins carries a risk of treatment failure through emergence of derepressed mutants during therapy, and cefepime or a carbapenem should be preferred according to severity [8]. In our series, however, non-susceptibility to cefepime remained high in *K. pneumoniae* (90.9%), which severely limits its value as a carbapenem-sparing option in this species.

Roughly one isolate in five was non-susceptible to at least one carbapenem. This level constitutes a major warning signal in a setting where alternative drugs such as ceftazidime-avibactam, meropenem-vaborbactam and cefiderocol are scarcely or not available and where carbapenemase confirmation is not performed routinely. The underlying mechanisms may combine carbapenemase production with the coexistence of an ESBL or an AmpC together with impermeability through porin loss [10] [11]; the six isolates classified on ertapenem alone are precisely those in which porin loss combined with an ESBL or AmpC, rather than a carbapenemase, is the more likely explanation. Because two thirds of our non-susceptible isolates rested on a single carbapenem result and a fifth on an intermediate category, this proportion should be regarded as a screening signal requiring confirmation rather than as a measured carbapenemase prevalence. Molecular characterisation of these isolates, including screening for *blaOXA-48*, *blaNDM* and *blaKPC* genes, should become a priority for the institution, both to guide therapy and to allow early detection of outbreaks [25].

The high class-level rates of non-susceptibility to fluoroquinolones and co-trimoxazole, particularly in *K. pneumoniae*, reflect frequent co-resistance, consistent with the carriage of multidrug resistance plasmids described in this species [6]. The aminoglycosides are the important exception, and the reason we report agent-level results alongside class-level ones. Read as a class, aminoglycosides appear compromised in 69.4% of *K. pneumoniae*; read agent by agent, that figure is

driven entirely by gentamicin and tobramycin, while amikacin was susceptible in all 28 *K. pneumoniae* isolates tested and in 27 of the 31 *Enterobacter* isolates tested (4 non-susceptible; 12.9%). A class-level summary would have concealed the one widely available, affordable and parenteral agent that retained near-complete activity in this series. Subject to confirmation on a larger number of isolates and to the usual constraints on aminoglycoside use in neonates and in renal impairment, amikacin deserves formal evaluation in local empirical protocols, whether as a component of combination therapy or as a carbapenem-sparing option. The oral alternatives explored—fosfomycin, tetracyclines, nitrofurantoin—offer no usable fallback in bloodstream infections, for reasons of both resistance and pharmacokinetics. In *Serratia* spp., broadly preserved susceptibility to β -lactams contrasts with the presence of two carbapenem non-susceptible isolates out of nine; this genus indeed combines a baseline of intrinsic resistance with a genuine capacity to acquire additional mechanisms under selective pressure [26], which precludes treating it as a genus without stakes despite the small number observed here.

This study has several limitations. Its retrospective, single-centre design limits the generalisability of the findings. Resistance mechanisms were inferred from phenotypic profiles alone, without systematic synergy testing or molecular confirmation, so the phenotypes reported must be considered probable; in *Enterobacter* spp. and *Serratia* spp., which carry an inducible chromosomal cephalosporinase, the ceftazidime-based algorithm cannot separate a derepressed AmpC from an associated ESBL at all. Ceftazidime itself was absent from the panel in roughly 40% of the third-generation-cephalosporin non-susceptible isolates, so a substantial fraction of isolates remained unclassifiable; we have reported these as a distinct category rather than redistributing them, which makes the ESBL proportions reported here conservative. Most *Enterobacter* isolates were identified only at genus level, which precludes any species-specific interpretation of chromosomal AmpC expression within that genus. The number of *Serratia* spp. isolates was small, making the corresponding percentages unstable, and several agents were tested on fewer than five isolates. Because not all agents were tested on all isolates, denominators vary from one agent to another, which may introduce a selection bias favouring the most resistant isolates for agents tested as second-line options; the same caveat applies with particular force to the carbapenem figure, which rested on a single agent in two thirds of cases. The laboratory information system carries no unique patient identifier, so episodes could only be de-duplicated at the level of the blood culture request, and a small number of episodes may represent repeat sampling of the same patient. Finally, the absence of clinical data on severity, management and patient outcome precluded any assessment of the impact of resistance on mortality, and any formal distinction between community-onset bacteraemia and healthcare-associated infection. These limitations notwithstanding, the fact that all analysed isolates were recovered from blood cultures gives the results direct clinical value, since they reflect the resistance of pathogens responsible for proven systemic infection ra-

ther than mere colonisation.

5. Conclusions

KES-group Enterobacterales represented close to one in five significant isolates among documented bloodstream infections at Dalal Jamm National Teaching Hospital in 2024, with a marked concentration in paediatric units, intensive care and clinical haematology. The profiles observed—non-susceptibility to third-generation cephalosporins in almost all *K. pneumoniae* isolates, a predominance of ESBL-type phenotypes among the isolates that could be classified, AmpC-type phenotypes in *Enterobacter* spp. and carbapenem non-susceptibility in roughly one isolate in five—reflect a level of multidrug resistance that compromises usual empirical regimens. Against that background, the retained activity of amikacin is the one encouraging signal of this series.

These findings justify coordinated action combining continuous microbiological surveillance of blood cultures, revision of local empirical therapy protocols, strengthened infection prevention and control in high-risk units, and continuing education of care teams. Two laboratory measures are immediately actionable and inexpensive: the systematic inclusion of ceftazidime and of at least two carbapenems in the panels applied to Enterobacterales from blood cultures, without which resistance phenotypes cannot be interpreted, and the routine reporting of amikacin alongside gentamicin. These findings also call for the introduction of molecular surveillance able to identify resistance genes and circulating clones, the only means of documenting possible cross-transmission and of guiding containment measures within a One Health perspective.

Acknowledgements

The authors thank the entire technical staff of the Bacteriology-Virology laboratory of Dalal Jamm National Teaching Hospital.

Ethics Approval

The study was conducted in accordance with the Declaration of Helsinki and was reviewed and authorised by the management of Dalal Jamm National Teaching Hospital, Guédiawaye, Senegal, which is the competent authority for the secondary use of anonymised hospital laboratory data at this institution. The study was not submitted to a research ethics committee: it involved no intervention, no contact with patients and no identifiable data, and national regulations do not require committee review in these circumstances. Given the retrospective and anonymised nature of the study, individual informed consent was waived. Data were anonymised before analysis.

Data Availability

The anonymised dataset underlying this analysis is available from the corresponding author upon request.

Author Contributions

Conceptualization, ABD, and HD, methodology, EBD, OG; formal analysis: ABD, HD, investigation, FD, HD, AT, data curation, HD, OG; writing original draft preparation, ABD; writing-review and editing, RK, DD, FS, MD, AT, FD, HD, DB, ASB; supervision, LF, RK, NCTK MC. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Evans, L., Rhodes, A., Alhazzani, W., *et al.* (2021) Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2021. *Critical Care Medicine*, **49**, e1063-e1143.
- [2] Pilmis, B. and Dortet, L. (2025) Facteurs de risque et impact des traitements sur la mortalité des patients bactériémiques à entérobactéries productrices de métallobactamase: Étude multicentrique. *Médecine et Maladies Infectieuses Formation*, **4**, S34-S35. <https://doi.org/10.1016/j.mmifmc.2025.04.069>
- [3] Ruiz-Giardin, J.M., Martin-Díaz, R.M., Jaqueti-Aroca, J., Garcia-Arata, I., San Martín-López, J.V. and Sáiz-Sánchez Buitrago, M. (2015) Diagnosis of Bacteraemia and Growth Times. *International Journal of Infectious Diseases*, **41**, 6-10. <https://doi.org/10.1016/j.ijid.2015.10.008>
- [4] Livrelli, V., De Champs, C., Di Martino, P., Darfeuille-Michaud, A., Forestier, C. and Joly, B. (1996) Adhesive Properties and Antibiotic Resistance of Klebsiella, Enterobacter, and Serratia Clinical Isolates Involved in Nosocomial Infections. *Journal of Clinical Microbiology*, **34**, 1963-1969. <https://doi.org/10.1128/jcm.34.8.1963-1969.1996>
- [5] Barbosa, T.A., Bentlin, M.R., Rugolo, L.M.S.d.S., Lyra, J.C., Ferreira, A.M., Santos, A.C.M.L.d., *et al.* (2025) Molecular Characterization of Gram-Negative Bacilli Isolated from a Neonatal Intensive Care Unit and Phenotypic and Molecular Detection of ESBL and Carbapenemase. *Antibiotics*, **14**, Article No. 342. <https://doi.org/10.3390/antibiotics14040342>
- [6] Navon-Venezia, S., Kondratyeva, K. and Carattoli, A. (2017) *Klebsiella pneumoniae*: A Major Worldwide Source and Shuttle for Antibiotic Resistance. *FEMS Microbiology Reviews*, **41**, 252-275. <https://doi.org/10.1093/femsre/fux013>
- [7] Guérin, F. (2015) Infections à Enterobacter cloacae complex: résistance aux antibiotiques et traitement. *Journal des Anti-infectieux*, **17**, 79-89. <https://doi.org/10.1016/j.antinf.2015.03.002>
- [8] Tebano, G., Zaghi, I., Cricca, M. and Cristini, F. (2024) Antibiotic Treatment of Infections Caused by AmpC-Producing Enterobacterales. *Pharmacy*, **12**, Article No. 142. <https://doi.org/10.3390/pharmacy12050142>
- [9] Monardo, R., Park, L.P., Ruffin, F., Cox, P., Dinh, A.Q., Korn, R.E., *et al.* (2025) Clinical and Genomic Characterization of Serratia Bloodstream Infections. *CMI Communications*, **2**, Article ID: 105128. <https://doi.org/10.1016/j.cmicom.2025.105128>
- [10] Michel-Briand, Y. (2007) La résistance aux β -lactamines les plus récentes: Les mécanismes d'apparition et de diffusion de la résistance chez les entérobactéries. *Bulletin de l'Académie Nationale de Médecine*, **191**, 35-51.

- [https://doi.org/10.1016/s0001-4079\(19\)33114-0](https://doi.org/10.1016/s0001-4079(19)33114-0)
- [11] Cattoir, V. (2014) Traitement des infections dues à entérobactéries productrices de carbapénèmases. *Journal des Anti-Infectieux*, **16**, 99-105.
<https://doi.org/10.1016/j.antinf.2014.07.002>
 - [12] World Health Organization (2025) Global Antibiotic Resistance Surveillance Report 2025. WHO.
 - [13] Berthod, D., Troillet, N., San Millán, D. and Pouget, R. (2012) [b]Entérobactéries[/b] résistantes: Explosion des β -lactamases à spectre élargi. *Revue Médicale Suisse*, **8**, 1925-1929. <https://doi.org/10.53738/revmed.2012.8.357.1925>
 - [14] Ndir, A. (2015) Épidémiologie et Impact Médico-Économique des Infections Hospitalières Causées par les Entérobactéries Productrices de Bêta-Lactamases à Spectre Étendu au Sénégal. Ph.D. Thesis, Université Pierre et Marie Curie-Paris VI.
 - [15] Comité de l'Antibiogramme de la Société Française de Microbiologie (2024) CA-SFM/EUCAST: Recommandations 2024. SFM.
 - [16] Magiorakos, A.P., Srinivasan, A., Carey, R.B., Carmeli, Y., Falagas, M.E., Giske, C.G., *et al.* (2012) Multidrug-Resistant, Extensively Drug-Resistant and Pandrug-Resistant Bacteria: An International Expert Proposal for Interim Standard Definitions for Acquired Resistance. *Clinical Microbiology and Infection*, **18**, 268-281.
<https://doi.org/10.1111/j.1469-0691.2011.03570.x>
 - [17] Philippon, A. and Arlet, G. (2012) Entérobactéries et bêta-lactamines: Phénotypes de résistance naturelle. *Pathologie Biologie*, **60**, 112-126.
<https://doi.org/10.1016/j.patbio.2011.12.002>
 - [18] Shinga Wembulua, B., Lakhe, A., Diallo Mbaye, K., *et al.* (2021) Profils de Sensibilité aux Antibactériens des Isolats Sanguins chez 74 Patients Infectés par le VIH Hospitalisés à la Clinique des Maladies Infectieuses et Tropicales du CHU de Fann, Dakar de 2013 à 2016. *Médecine Tropicale et Santé Internationale*, **1**, Article No. 88.
 - [19] Njall, C., Adiogo, D., Bitá, A., Ateba, N., Sume, G., Kollo, B., *et al.* (2013) Écologie bactérienne de l'infection nosocomiale au service de réanimation de l'hôpital Laquintinie de Douala, Cameroun. *Pan African Medical Journal*, **14**, Article No. 140.
<https://doi.org/10.11604/pamj.2013.14.140.1818>
 - [20] Saraswati, P.A., Saputro, I.D., Widodo, A.D.W., Budi, A.S. and Permana, P.B.D. (2025) Factors Associated with Clinical Outcomes of Bloodstream Infections at a Burn Unit in Indonesia: A Three-Year Analysis. *Annals of Burns and Fire Disasters*, **38**, 124-135.
 - [21] Ndir, A., Diop, A., Faye, P.M., Cissé, M.F., Ndoye, B. and Astagneau, P. (2016) Epidemiology and Burden of Bloodstream Infections Caused by Extended-Spectrum Beta-Lactamase Producing Enterobacteriaceae in a Pediatric Hospital in Senegal. *PLOS ONE*, **11**, e0143729. <https://doi.org/10.1371/journal.pone.0143729>
 - [22] Rahden, P., Barrow, E., Bah, H., Bittaye, S.O., Nygren, D. and Badjan, A. (2025) Bloodstream Infections at a Tertiary Hospital in the Gambia—A One-Year Retrospective Study. *BMC Infectious Diseases*, **25**, Article No. 170.
<https://doi.org/10.1186/s12879-025-10533-1>
 - [23] Sands, K., Carvalho, M.J., Portal, E., Thomson, K., Dyer, C., Akpulu, C., *et al.* (2021) Characterization of Antimicrobial-Resistant Gram-Negative Bacteria That Cause Neonatal Sepsis in Seven Low- and Middle-Income Countries. *Nature Microbiology*, **6**, 512-523. <https://doi.org/10.1038/s41564-021-00870-7>
 - [24] Robin, F., Gibold, L. and Bonnet, R. (2012) Résistances naturelles et acquises aux β -lactamines chez les entérobactéries: Comment les identifier en pratique quotidienne?

Revue Francophone des Laboratoires, **2012**, 47-58.

[https://doi.org/10.1016/s1773-035x\(12\)71676-3](https://doi.org/10.1016/s1773-035x(12)71676-3)

- [25] European Centre for Disease Prevention and Control (2025) Carbapenem-Resistant Enterobacterales-Third Update. ECDC.
- [26] Sandner-Miranda, L., Vinuesa, P., Cravioto, A. and Morales-Espinosa, R. (2018) The Genomic Basis of Intrinsic and Acquired Antibiotic Resistance in the Genus *Serratia*. *Frontiers in Microbiology*, **9**, Article No. 828.
<https://doi.org/10.3389/fmicb.2018.00828>