International Scholars Journals

Advanced Journal of Microbiology Research ISSN 2736-1756 Vol. 19 (4), pp. 001-008, April, 2025. www.internationalscholarsjournals.org/ © International Scholars Journals

Full Length Research Paper

Epidemiology of bacterial resistance and detection of metallo-β-lactamase (*blaNDM*) and imipenemase (*blaIMP)* resistance genes at CERBA

Géraud Joël GUIGMA*[1,2], Prosper BADO[1], Valérie Bapio BAZIE[1,2], Tampoubila Edwige YELEMKOURE[1], Serge Théophile SOUBEIGA[1,2], Delwende Leslie KABORE, Désiré ILBOUDO[1], Amana METUOR DABIRE[1,3], Albert Théophane YONLI[1] and Jacques SIMPORE[1].

[1]Center de Recherche Biomoléculaire Pietro Annigoni (CERBA), 01 BP 364/Ouagadougou/ Burkina Faso
[2]Center National de la Recherche Scientifique et Technologique (CNRST)/Institut de Recherche en Sciences de la Santé (IRSS)/Ouagadougou/ Burkina Faso
[3]University of Daniel Ouezzin Coulibaly/Burkina Faso

Abstract

Received 6 January, 2025; Revised 28 February, 2025; Accepted 3 March, 2025; Published 7 April, 2025

Introduction: Antimicrobial resistance is a major public health concern. Producing new β-lactamases and carbapenemases is one form of resistance that preoccupies many scientists. The risk of the spread of carbapenemase-producing Enterobacteriaceae (EPCs) is a major public health issue, as these enzymes restrict therapeutic options and are often associated with other mechanisms, conferring multi-resistance on strains. Our study aimed to characterize the *blaIMP* and *blaNDM* resistance genes in Enterobacteriaceae isolates from urine cultures and genital swabs at CERBA from 2020 to 2023. **Methodology**: Pathogens were isolated on agar media, then identified using the API 20 E gallery; Imipenem-resistant strains were subjected to the traditional Hodge test to verify carbapenemase production. Detection of the *IMP* and *NDM* resistance genes coding for carbapenemases was carried out by multiplex real-time PCR at CERBA. **Results**: 1119 samples have been received for bacteriological analysis since January 2020. We noted 14.2% positivity to a clinically pathogenic strain. Bacterial species diversity was dominated by *Escherichia coli* in **54.71%** of cases, followed by *Klebsiella pneumoniae* (**15.72%)**. We observed a predominance of the *NDM* gene **(97.9%)** over *IMP* **(2.1%).** In some cases, we noted the coexistence of the *IMP* and *NDM* genes in *Escherichia coli.* **Conclusion**: This study enabled us to characterize the *IMP* and *NDM* resistance genes in isolation (*IMP/NDM*) or coexisting together (*IMP+NDM*) in Enterobacteriaceae isolates at CERBA. This study also enabled us to determine the frequency of bacterial species in bacterial culture samples at CERBA.

Key words: IMP, NDM, Enterobacteriaceae, β-lactamase, Carbapenemase.

Introduction

The advent of antibiotic therapy has revolutionized the medical world since the 1940s, thanks to the discoveries of British epidemiologist Flemming. Unfortunately, in recent years, antibiotic resistance (AMR) has become a major global public health problem. An estimated 4.95 million deaths were associated with bacterial resistance in 2019, including 1.27 million deaths directly caused by bacterial resistance to antibiotics [1]. Among antibiotic-resistant bacteria, those resistant to beta-lactams and carbapenems were classified by the World Health Organization in 2017 as one of the three main classes of antibiotic-resistant bacteria worldwide [2]. In fact, there are

Correspondent*: joelguigma1@gmail.com

several resistance mechanisms that simply reflect the evolution and adaptation of the microbial world towards the aggressors that are antibiotics. Beta-lactams act by blocking peptidoglycan synthesis, and thus cell membrane synthesis [3]. Bacterial resistance is the result of a permanent war of survival in which micro-organisms, constantly under attack from antibiotics, try to find a countermeasure [4].

In northern countries, antimicrobial resistance is well known and the subject of strategic surveillance and response plans [5]. On the other hand, in Africa, and especially in Sub-Saharan Africa, few data are describing the phenomenon of antibiotic resistance, its surveillance and the response to it. [6].

This phenomenon is not a recent development in Burkina Faso, where research has demonstrated the presence of multiple strains of enterobacteria exhibiting high levels of resistance to antibiotics, particularly beta-lactam antibiotics [27]. During his doctoral research, Abdoul Salam Ouedrago also found that multiresistant strains were widespread in Burkina Faso[28]. In Burkina Faso, although data are collected in health facilities, studies are still needed to strengthen the AMR surveillance system at national level.

The aim of this study was to screen the epidemiology of strains circulating and isolated in pathological products, and to determine the prevalence of resistance according to bacterial species and antibiotic families.

Methodology

Study framework

The study was carried out at the Pietro Annigoni Biomolecular Research Center/ National Reference Laboratory for Human Papilloma Virus (CERBA/LNR-HPV); within the clinical microbiology analysis unit. Clinical samples from all over Patients from all walks of life, irrespective of age or sex, who present themselves for a diagnostic evaluation, accompanied by a documented report that details the analysis of urine and/or genital secretions using microbiological methods were included in the study.

The present study complies with the sampling criteria, i.e. the majority of urine samples were collected using the mid-jet method.

All samples were collected in a sterile environment in close proximity to the Bunsen burner. The subsequent biological analyses were conducted in accordance with the Clinical and Laboratory Standards Institute (CLSI) guidelines for the isolation, identification and antibiotic susceptibility testing of microorganisms.

Identification of pathogens

Pathogens were identified using standard bacteriological methods. Culture in agar medium, isolation of the pathogens and finally identification by biochemical methods using the API 20E gallery.

Detecting antibiotic resistance

Resistance was determined immediately on reading the antibiogram. In fact, the antibiogram was performed using the Kirby Bauer method [7]. We prepared a 5mL bacterial suspension in sterile water, then inoculated it onto 4mm Muller Hinton agar. Antibiotic discs were placed at a distance of 25mm between each disc. Petri dishes were then incubated at 35 ± 2 degrees Celsius for 24 hours.

Detection of resistance genes

All carbapenem-resistant strains were verified by the traditional Hodge test, which involved identifying carbapenemase-producing strains [8], [9]. They were then characterized by molecular biology. We used the Sacace™ DNA-Sorb-B nucleic acid extraction kit version 2020 (K-1-1/B/100) for bacterial nucleic acid extraction, following the manufacturer's protocol. Real-time PCR was performed by targeting two specific genes in the genome of these bacteria. The *blaNDM* and *blaIMP* genes, which specifically code for resistance to metallo-betalactamases and imipenemases (Table I).

Statistical analysis

All data were entered in Excel and analyzed using R 4.3.1 software.

Results

Out of 1119 samples, 159 were positive for at least one clinically pathogenic germ, representing a positivity rate of 14.2%.

There was a slight predominance of infected females (50.9%) versus males (49.2%), i.e. a sex ratio of 0.96.

The 18 to 59 age group was the most exposed to bacterial infections, with a percentage of 57.86% (Table II).

In all positive cultures, enterobacteria predominated, with *Escherichia coli* followed by *Klebsiella pneumoniae*. Our results show a diversity of germs divided into fifteen (15) species, with *Escherichia coli* the most incriminated species in 54.71% of infections (Table III).

Among the antibiotics tested, we observed total resistance to certain antibiotics such as fusidic acid, oxacillin, tobramycin and norfloxacin. We found strong resistance to amoxicillin, amoxicillin + clavulanic acid, ampicillin, tetracyclines, erythromycin and cotrimoxazole. On the other hand, sensitivities are good to imipenem, ciprofloxacin and chloramphenicol. Aztreonam and ticarcillin combined with clavulanic acid showed Intermediate sensitivity in half the cases.

Our results show a general distribution of susceptibility according to antibiotic families. Many of the species

Table I. Primers for real-time PCR.

Genes	Primers	Sequences	Size (bp)	References
blaIMP	For	5' CATGGTTTGGTGGTTCTTGT 3'	488	[10]
	Rev	5' ATAATTTGGCGGACTTTGGC 3'		
blaNDM	For	5' CAGCACACT TCCTATCTC 3'	292	[10]
	Rev	5' CCGCAACCATCCCCTCTT 3'		

Table II. Age distribution

Age ranges	Workforce
(years)	**n (%)**
0 -14	18 (11,32)
15 -17	9 (5,66)
18 - 59	92 (57,86)
over 60	40 (25,16)
Total	**159 (100)**

Table III Frequency of bacterial species in the total population.

Bacterial species	Number (%)
Escherichia coli	**87 (54,71)**
Klebsiella pneumoniae	**25 (15,72)**
Staphylococcus aureus	**22 (13,83)**
Staphylococcus epidermidis	**9 (5,66)**
Pseudomonas aeroginosa	**4 (2,57)**
Klebsiella ornithylotica	**3 (1,88)**

isolated were resistant to aminopenicillins, cephalosporins, quinolones and sulfonamides. On the other hand, very few are resistant to aminoglycosides, carbapenems, macrolides and phenicoles (Figure 1).

Figure 1: Distribution of species according to their sensitivity to antibiotic families

Five species, comprising 47 strains, were found to be resistant to antibiotics. We then looked for the presence of two resistance genes and their distribution throughout the sample. We found a predominance of the NDM gene (97.9%) over IMP (2.1%). With a high number of *Escherichia coli* species, one of which possessed both genes (Figure 2).

Table III. Continued

Streptococcus Group B	**3 (1,88)**
Salmonella paratyphi	**2 (1,26)**
Streptococcus Group D	**2 (1,26)**
Citrobacter freundii	**1 (0,63)**
Enterobacter cloacae	**1 (0,63)**
Proteus mirabilis	**1 (0,63)**
***Salmonella* spp**	**1 (0,63)**
***Serratia* spp**	**1 (0,63)**
Staphylococcus saprophyticus	**1 (0,63)**

Table IV Sensitivity frequencies by antibiotic.

Antibiotic	Resistant (%)	Intermediate (%)	Sensitive (%)
Amoxicillin	**88.9**	11.1	0
Amoxicillin+Ac clav.	**97.6**	2.4	0
Imipenem	12	12	**76**
Piperacillin	**68**	16	16
Ceftriaxone	40,48	14,3	45,2
Fusidic acid	**100**	0	0
Cefotaxine	41,4	9,7	**53,4**
Ticarcillin + Ac clav.	21,7	56,5	21,7
Ceftazidime	15	25	60
Aztreonam	12,5	37,5	50
Oxacillin	**100**	0	0
Ampicillin	**80**	20	0
Tobramycin	**100**	0	0

Gentamycin	44,1	41,2	14,7
Netilmicin	0	0	**100**
Doxycycline	23.1	15,4	**71,5**
Tetracycline	**83,3**	8,3	8,4
Norfloxacin	**100**	0	0
Ciprofloxacin	32,6	8,2	**59,2**
Crotrimoxazole	**87,5**	0	12,5
Erythromycin	**82.6**	4.4	13
Chloramphenicol	19.1	4.8	**76.2**

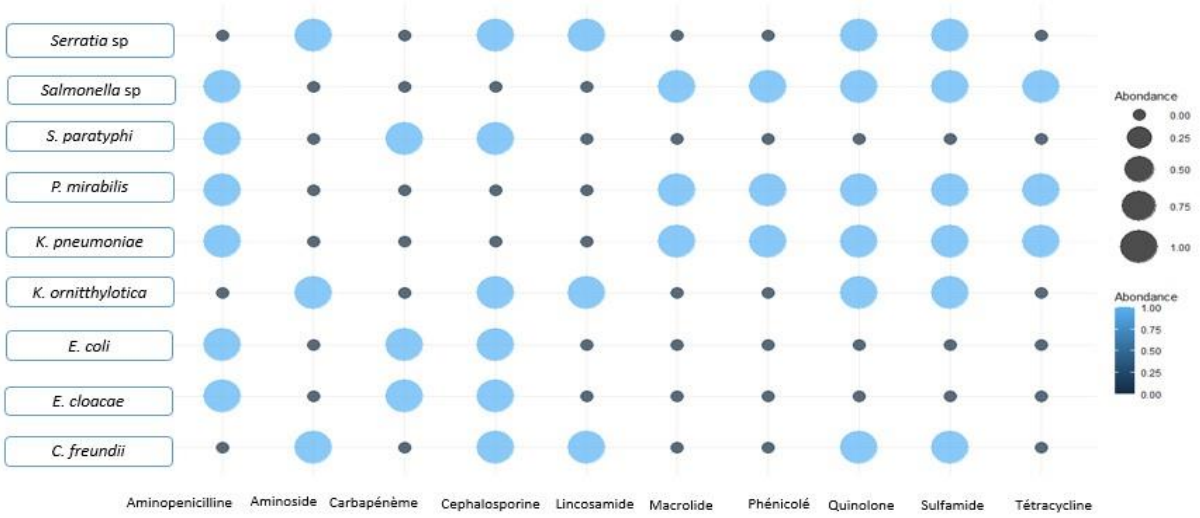

Figure1: Distribution of species according to their sensitivity to antibiotic families.

Discussion

In a cohort of 1,119 people, we had a bacterial culture positivity rate of 14.2%.

We found a moderate predominance of female urinary tract infections (50.85%) versus males (49.15%), for a sex ratio of 0.96. The age group most represented in our study was 18 to 59. The work of several scientists [11], [12], [13] show similar results to our own. Indeed, these results show that young people and women are the most exposed to urinary tract and genital tract infections. There are several possible explanations for these results: firstly, the lack of hygiene, especially in low-income countries. This factor carries with it a certain stigma, leading people to be less concerned about individual and community hygiene. [14] This phenomenon condemns people to remain exposed to microbial infections. Secondly, the proximity of the female urinary tract to the anus could be a major factor in explaining the predominance of women in the study. Also, the high level of sexual activity in the [18-59] age bracket could explain the high level of ITUs in this study.

In this study, we isolated 15 different bacterial species. *Escherichia coli was the* most isolated bacterium with a frequency of (**54.7%),** followed by *Klebsiella pneumoniae* (**15.7%)** and *Staphylococcus aureus* (**13.8%).** Other minorities included *Citrobacter freundii, Enterobacter*

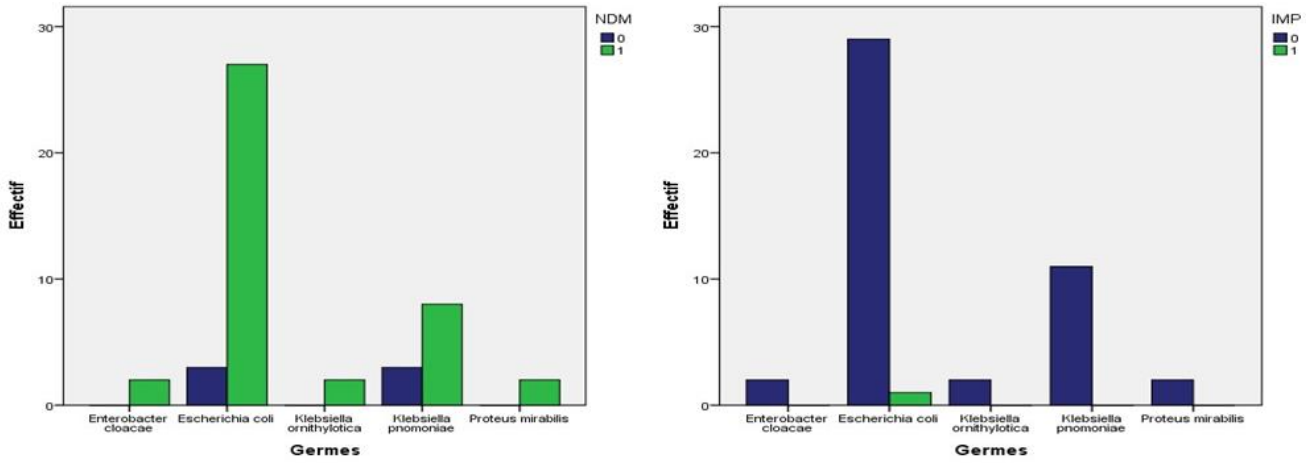

Figure 2: Distribution of bacterial species according to the presence of the blaNDM and blaIMP genes.

cloacae, Proteus mirabilis, Salmonella spp, *Serratia* spp and *Staphylococcus saprophyticus*, each accounting for 0.63%. These results are similar to those of Gebretensai et al, 2023 [15]. Indeed, from a cohort of 441 samples analyzed, 16 species were identified with a predominance of *Escherichia coli* followed by *Klebsiella pneumoniae*; similarly Merga et al., 2018 [16] had similar results to ours. This similarity of results could be explained by the widespread ecology of enterobacteria in our environments, especially the ubiquitous nature of *Escherichia coli. In* addition to the ecology of these strains, the hygienic behavior of our populations and their lifestyles also play a major role in exposure to these germs.

However, among the antibiotics we tested on these bacterial strains, we found total resistance (of 100%) to certain antibiotics such as fusidic acid, Oxacillin, Tobramycin and Norfloxacin. We found strong resistance to amoxicillin, amoxicillin + clavulanic acid, ampicillin, tetracyclines, erythromycin and cotrimoxazole. Fusidic acid, oxacillin and norfloxacin were tested on grampositive cocci. We were therefore confronted with 100% methicillin-resistant *Staphylococcus aureus* **(MRSA).** These results differ slightly from those of Balamou et al, (2023)[17] who found an **MRSA** rate of 85.7% in their study at the Kindia regional hospital in Guinea. Nevertheless, the MRSA rate remains high. This discrepancy in results could be explained by the non-hospital nature of the Pietro Annigoni Biomolecular Research Center (CERBA); on the other hand, the high MRSA rate could be explained by the excessive and uncontrolled use of antibiotics. In fact, the availability and accessibility of certain antibiotics is lacking due to the shortage of pharmacies and pharmacists, which could be a factor encouraging people to self-medicate. According to the work of Sana et al. 2023 [18]Burkina Faso has 0.2 pharmacists per 10,000 inhabitants. *S. aureus*, use target mutation as a process to escape the effect of meticillin by modifying its binding protein PLP to PLP2a [19]the understanding of this phenomenon has shed light on the deleterious affinity of meticillin and, on the justification for the production of cephalosporin antibiotics. The work of Balamou et al. has shown that MRSA are highly sensitive to cephalosporins. [17].

As for Enterobacteriaceae, we have noted a diversification of resistance depending on the family. Aminopenicillins (amoxicillin, amoxicillin + clavulanic acid), third-generation cephalosporins (Ceftriaxone, cefotaxime...), which make up the beta-lactam subfamilies, were the most prone to resistance (figure 2). This hetero-resistance to antibiotics could be explained by several factors, including the ecology of each strain of Enterobacteriaceae. Subjected differently to anthropogenic pressures, in addition to their natural genetic resistances, they will consequently behave heterogeneously in relation to particular antibiotics or families of antibiotics. This heterogeneity, coupled with microbial ecology, results in acquired resistance to antibiotics. This fact is well described by Andersson et al, (2019) [20] regarding the sharing of resistance genes within Enterobacteriaceae, which is responsible for the phenotypic expression of antibiotic resistance. These genetic exchanges in microenvironments are not without effect; they are becoming one of the major concerns of modern medicine and constituting a major challenge for clinicians, biologists and sociologists [1].

In our study, a set of enterobacterial strains showed a multi-resistant phenotype on at least three antibiotic families (total resistant). These strains included *E. coli* (the majority), *Klebsiella pneumoniae* and *Klebsiella ornithylotica*, *Enterobacter cloacea* and *Proteus mirabilis.* Their phenotypic expressions in relation to antibiotics led us to verify the presence or absence of two resistance genes responsible for the production of beta-lactamase enzymes: NDM (New Delhi

Metallo-beta-lactamase) and IMP (imipenimase). Our results show a high presence of the NDM gene (97.9%) in the total resistant strains; and predominantly dominated by *E coli*; in contrast to the *IMP* gene (2.1%). These results

differ from those of Lionel et al, (2023) [21] in Burkina Faso, who found a predominance of strains carrying the *IMP* gene (32%) versus 16% for the *NDM* gene. This difference in results could be explained by the variability of bacterial strains and also by the positive or negative evolution of bacterial strains.

Nordemann, on the other hand, describes the *NDM gene* as the most recent and most widespread on the Asian continent. [22]. The *NDM* gene is an antibiotic resistance gene that codes for the enzyme New Delhi Metallo beta-lactamase; this enzyme can hydrolyze carbapenems, the class of antibiotics of last resort. [23]. The gene encoding IMP is not negligible, although it is not sufficiently present in the population; this enzyme can inactivate beta-lactam antibiotics such as penicillins, cephalosporins (C1G, C2G, C3G and C4G) and carbapenems. [24].

We have also observed the coexistence of the two genes in one strain of *Escherichia coli,* the simultaneous presence of the two genes being proof of gene transfer between different bacterial strains or species. Indeed, these genes were first detected in a strain of *Acinetobacter baumannii* in 1992 for IMP [25] while the *NDM* gene was first identified in a *Klebsiella pneumoniae* species in 2008 in India [26]. Today, these genes are present in a clinical strain of *Escherichia coli.*

References

[1] C. J. L. Murray *et al*, "Global burden of bacterial antimicrobial resistance in 2019: a systematic analysis," *The Lancet*, vol. 399, no. 10325, pp. 629-655, Feb. 2022, doi: 10.1016/S0140-6736(21)02724-0.

[2] E. Tacconelli *et al*, "Surveillance for control of antimicrobial resistance," *Lancet Infect. Dis.* vol. 18, no. 3, pp. e99-e106, Mar. 2018, doi: 10.1016/S1473-3099(17)30485-1.

[3] J.-D. Cavallo, R. Fabre, F. Jehl, C. Rapp, and E. Garrabé, "Betalactamines," *EMC - Mal. Infect.* vol. 1, no. 3, pp. 129-202, Aug. 2004, doi: 10.1016/j.emcmi.2004.03.003.

[4] V. I. Band and D. S. Weiss, "Heteroresistance to beta-lactam antibiotics may often be a stage in the progression to antibiotic resistance," *PLOS Biol.* vol. 19, no. 7, p. e3001346, Jul. 2021, doi: 10.1371/journal.pbio.3001346.

[5] G. M. Rossolini and E. Mantengoli, "Antimicrobial resistance in Europe and its potential impact on empirical therapy," *Clin. Microbiol. Infect.* vol. 14, pp. 2-8, Dec. 2008, doi: 10.1111/j.1469-0691.2008.02126.x.

[6] A. S. Ouedraogo, H. Jean Pierre, A. L. Bañuls, R. Ouédraogo, and S. Godreuil, "Emergence and spread of antibiotic resistance in West Africa: contributing factors and threat assessment," *Médecine Santé Trop.* vol. 27, no. 2, pp. 147-154, May 2017, doi: 10.1684/mst.2017.0678.

[7] F. Jehl, A. Chabaud, and A. Grillon, "L'antibiogramme : diamètres ou CMI ?," *J. Anti-Infect.* vol. 17, no. 4, pp. 125-139, Dec. 2015, doi: 10.1016/j.antinf.2015.08.003.

[8] W. Song *et al*, "Combined Use of the Modified Hodge Test and Carbapenemase Inhibition Test for Detection of Carbapenemase-Producing *Enterobacteriaceae* and Metallo-βLactamase-Producing *Pseudomonas* spp.", *Ann. Lab. Med.* vol. 35, no. 2, pp. 212-219, Mar. 2015, doi: 10.3343/alm.2015.35.2.212.

[9] N. Patidar, N. Vyas, S. Sharma, and B. Sharma, "Phenotypic Detection of Carbapenemase Production in Carbapenem-Resistant Enterobacteriaceae by Modified Hodge Test and Modified Strip Carba NP Test," *J. Lab. Physicians*, vol. 13, no. 01, pp. 014-021, Mar. 2021, doi: 10.1055/s-0041-1723859.

[10] S. Huang, W. Dai, S. Sun, X. Zhang, and L. Zhang, "Prevalence of Plasmid-Mediated Quinolone Resistance and Aminoglycoside Resistance Determinants among Carbapeneme Non-Susceptible Enterobacter cloacae," *PLoS ONE*, vol. 7, no. 10, p. e47636, Oct. 2012, doi: 10.1371/journal.pone.0047636.

[11] L. E. Nicolle, "Uncomplicated Urinary Tract Infection in Adults Including Uncomplicated Pyelonephritis," *Urol. Clin. North Am.* vol. 35, no. 1, pp. 1-12, Feb. 2008, doi: 10.1016/j.ucl.2007.09.004.

[12] I. Monica, "Urinary Tract Infections and Associated Factors among Youth: A Case Study at KIUTH, Bushenyi," 2023, [Online]. Available: http://www.inosr.net/inosr-scientificresearch/

[13] D. A. Mengistu, A. Alemu, A. A. Abdukadir, A. Mohammed Husen, F. Ahmed, and B. Mohammed, "Incidence of Urinary Tract Infection Among Patients: Systematic Review and Meta-Analysis," *Inq. J. Health Care Organ. Provis. Financ.* vol. 60, p. 004695802311687, Jan. 2023, doi: 10.1177/00469580231168746.

[14] SFMTSI, "Maladies tropicales et pauvreté: impact sur les droits de la femme et de l'enfant. Journée scientifique de la SFMTSI (ex SPE). May 25, 2022," *MTSI*, p. Vol. 2 No 2 (2022): MTSIRevue, Jun. 2022, doi: 10.48327/MTSI.V2I2.2022.245.

[15] Y. Gebretensaie, A. Atnafu, S. Girma, Y. Alemu, and K. Desta, "Prevalence of Bacterial Urinary Tract Infection, Associated Risk Factors, and Antimicrobial Resistance Pattern in Addis Ababa, Ethiopia: A Cross-Sectional Study," *Infect. Drug Resist.* vol. Volume 16, pp. 3041-3050, May 2023, doi: 10.2147/IDR.S402279.

[16] Y. Merga Duffa, K. Terfa Kitila, D. Mamuye Gebretsadik, and A. Bitew, "Prevalence and Antimicrobial Susceptibility of Bacterial Uropathogens Isolated from Pediatric Patients at Yekatit 12 Hospital Medical College, Addis Ababa, Ethiopia," *Int. J. Microbiol.* vol. 2018, pp. 1-8, Oct. 2018, doi: 10.1155/2018/8492309.

[17] Balamou, O., Boiro, M. Y., Barry, A. A., and Kalivogui, S., "Sensibilité aux antibiotiques de

staphylococcus aureus communautaire résistant à la meticilline (SARM) à l'hôpital régional de Kindia," *Journal de la Recherche Scientifique de l'Université de Lomé*, pp. 207-213, 2023.

[18] B. Sana, A.-S. Ouedraogo, and R. Semdé, "Circuit des antibiotiques en Afrique francophone:
état des lieux, enjeux et perspectives," *Médecine Mal. Infect. Form*, vol. 2, no. 1, pp. 13-18, Mar. 2023, doi: 10.1016/j.mmifmc.2022.12.002.

[19] B. Mlynarczyk-Bonikowska, C. Kowalewski, A. Krolak-Ulinska, and W. Marusza, "Molecular Mechanisms of Drug Resistance in Staphylococcus aureus," *Int. J. Mol. Sci.* vol. 23, no. 15, p. 8088, Jul. 2022, doi: 10.3390/ijms23158088.

[20] D. I. Andersson, H. Nicoloff, and K. Hjort, "Mechanisms and clinical relevance of bacterial heteroresistance," *Nat. Rev. Microbiol.* vol. 17, no. 8, pp. 479-496, Aug. 2019, doi:
10.1038/s41579-019-0218-1.

[21] L. E. B. Bambara, A. M. Dabire, O. Traore, Y. Tiemtore, T. Zohoncon, and J. Simpore, "Genetic and Kinetic Profiles of Bacterial Resistance Enzymes to IMP and NDM Type Antibiotics in Gram-negative Pathogenic Bacilli," *Int. J. Biochem. Res. Rev.* vol. 32, no. 6, pp. 7-21, Aug. 2023, doi: 10.9734/ijbcrr/2023/v32i6819.

[22] P. Nordmann, "Carbapenemase-producing Enterobacteriaceae: Overview of a major public health challenge," *Médecine Mal. Infect.* vol. 44, no. 2, pp. 51-56, Feb. 2014, doi:
10.1016/j.medmal.2013.11.007.

[23] P. Nordmann, L. Poirel, T. R. Walsh, and D. M. Livermore, "The emerging NDM carbapenemases," *Trends Microbiol*, vol. 19, no. 12, pp. 588-595, Dec. 2011, doi: 10.1016/j.tim.2011.09.005.

[24] S. Abbas *et al*, "Molecular Detection of blaIMP Genes in Metallo-Beta-Lactamase Producing Clinical Gram-Negative Isolates," *Clin. Lab.* vol. 65, no. 08/2019, 2019, doi: 10.7754/Clin.Lab.2019.190202.

[25] H. Goudarzi, E. S. Mirsamadi, Z. Ghalavand, M. Hakemi Vala, H. Mirjalali, and A. Hashemi,
"Rapid detection and molecular survey of blaVIM, blaIMP and blaNDM genes among clinical isolates of Acinetobacter baumannii using new multiplex real-time PCR and melting curve analysis," *BMC Microbiol.* vol. 19, no. 1, p. 122, Dec. 2019, doi: 10.1186/s12866-0191510-y.

[26] A. U. Khan, L. Maryam, and R. Zarrilli, "Structure, Genetics and Worldwide Spread of New Delhi Metallo-β-lactamase (NDM): a threat to public health," *BMC Microbiol.* vol. 17, no. 1, p. 101, Dec. 2017, doi: 10.1186/s12866-017-1012-8.

[27] Absatou Ky Ba *et al.*, 'Résistance aux antibiotiques des souches de staphylococcus aureus et des enterobactéries isolés au LNSP de Ouagadougou (Burkina Faso)', *African Journal Online*, Burkina Faso, Ouagadougou, 2017. [Online]. Available: https://www.ajol.info/index.php/stss/article/view/190628

[28] A.-S. Ouédraogo, 'Prévalence, circulation et caractérisation des bactéries multirésistantes au Burkina Faso', 2016.