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Original Article

Description of Antimicrobial Resistance Patterns at the National Institute of Hygiene in Lome, Togo

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ABSTRACT: The monitoring of antimicrobial resistance has become a priority, mainly in developing countries, to control the emergence of multidrug-resistant bacterial strains. This study evaluated the antibiotic resistance profiles of bacteria isolated at the National Institute of Hygiene in Togo. The laboratory records from January 2013 to December 2015 were examined, which showed that a total of 33,147 samples had been analyzed. Among them, vaginal swabs and urine samples were predominant, amounting to 38.17% and 33.24%, respectively. In total, 3,497 Gram-positive and 1,970 Gram-negative bacterial strains were cultured. *Enterobacteriaceae* (57.6%), *Staphylococcus* spp. (21.1%), and *Streptococcus* spp. (10.5%) were primarily isolated. The records showed that over 3 years, *Escherichia coli* was resistant to piperacillin (79.66%, 75.1%, and 83%), trimethoprim/sulfamethoxazole (79.3%, 82%, and 82.8%), ticarcillin (89%, 89.7%, and 93%), and amoxicillin (94.1%, 94%, and 96.09%), whereas *Staphylococcus aureus* was resistant to penicillin G. *Streptococcaceae* isolates were resistant to trimethoprim/sulfamethoxazole (78.11–87.1%), tetracycline (82.2–91.16%), and norfloxacin (86.16–94.3%). *Escherichia coli* and *S. aureus* isolated from urine were more resistant to antibiotics than those isolated from vaginal swabs. There is a need to develop new strategies to fight the emergence of multi-resistant bacteria in Togo.

INTRODUCTION

Bacteria-induced infectious diseases are widespread in environments lacking in hygiene practices and balanced nutrition (1). They are a major public health concern in developing countries, particularly in Africa, and the leading cause of morbidity and mortality (2). The diagnosis and monitoring of emerging infectious diseases are therefore of great importance (3). The emergence of multidrug-resistant strains represents a real public health problem on a global scale. In Burkina Faso, studies have shown that the uncontrolled use of antibiotics against pathogenic bacteria has increased their resistance (4). In Togo, objective studies on the sensitivity of bacteria to antibiotics have revealed the occurrence of resistance to molecules that are in prolonged use, which reinforces the fear of inappropriate use of antibiotics (5). But a national system for recording and tracking data on antimicrobial resistance (AMR) is lacking. This retrospective study was undertaken to evaluate common bacterial infections and their antibiotic susceptibility and resistance profiles at the National Institute of Hygiene (INH) in Lome from 2013 to 2015.

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MATERIALS AND METHODS

Sample preparation and bacterial classification: A total of 33,147 samples from sick individuals, were obtained from swabs (vaginal, urethral, and throat) and body fluids (pus, spit, urine, ejaculates, stools, articular, ascitic, and bronchial fluid) at the public health reference bacteriology laboratory of INH in Lome, from January 2013 to December 2015. INH is the national public health reference laboratory in Togo that collects specimens from outpatient clinics, which generally

do not take blood and cerebrospinal fluid samples in routine. The samples were processed according to the national standards of medical bacteriological procedures (6). In brief, Gram-negative bacteria were grown on eosin methylene blue (OXOID Ltd., Basingtok, UK), cystine lactose electrolyte deficient (OXOID Ltd.), and chromogenic urinary tract infection agar (OXOID Ltd). Samples were seeded on Chapman agar (OXOID Ltd.) to cultivate *Staphylococcus* spp., whereas *Streptococcus* spp. were grown on blood agar plates (OXOID Ltd.) containing 5% sheep blood and nalidixic acid. Gram staining and catalase test allowed the classification of *Staphylococcus* spp. and *Streptococcus* spp. Bacteria belonging to *Neisseria* genus were cultivated on Thayer-Martin agar medium. Bacterial identification was based on their culture characteristics and certain biochemical tests. In addition, Gram-negative bacteria (*Enterobacteriaceae*) were identified using the Analytical Profile Index 20E system (Biomerieux, Marcy-l'Étoile, France) after obtaining a negative oxidase test.

Antibiotic susceptibility: The antibiotic susceptibility test (AST) was performed using the Kirby-Bauer diffusion method with the standard operating procedure, according to the recommendations of the Antibiogram Committee of the French Society of Microbiology (CA-SFM 2013) (7). Resistance was defined according to the diameters specified in CA-SFM 2013 for a given antibiotic (7). Bacterial strains belonging to *Staphylococcaceae*, *Streptococcaceae*, and *Enterococcaceae* families, non-susceptible to a given antibiotic whose resistance category is not defined in the CA-SFM 2013, were considered as resistant in this study; otherwise, resistance was judged using the "R" category defined by CA-SFM 2013. The density of the bacterial preparations was evaluated using the MacFarland standards. The following antibiotics (Mast Group, Derby, UK) were tested: (i) aminoglycosides: amikacin 30 µg (AK), gentamicin 15 µg (GM15), gentamicin 500 µg (GM500), kanamycin 1000 µg (K1000), kanamycin 30 µg (K30), netilmicin 30 µg (NET), streptomycin 500 µg (S), and tobramycin 10 µg (TM); (ii) β-lactams: amoxicillin 20 µg (AMX), ampicillin 10 µg (AMP), augmentin or amoxicillin 20 µg/clavulanic acid 10 µg (AUG), aztreonam 30 µg (ATM), imipenem 10 µg (IMP), oxacillin 5 µg (OXA), penicillin G 10 U (PG), piperacillin 75 µg (PIP), piperacillin 30 µg/tazobactam 6 µg (PTZ), ticarcillin 75 µg (TIC), ticarcillin 75 µg/clavulanic acid 10 µg (TCC); (iii) cephalosporins: cefalotin 30 µg (CF), cefepime 30 µg (CPM), cefotaxime 30 µg (CTX), cefoxitin 30 µg (FOX), cefsulodin 30 µg (CFS), ceftazidime 30 µg (CAZ), and ceftriaxone 30 µg (CRO); (iv) quinolones/fluoroquinolones: ciprofloxacin 5 µg (CIP), nalidixic acid 5 µg (NA), norfloxacin 5 µg (NOR), ofloxacin 5 µg (OFX), and pefloxacin 5 µg (PFX); (v) protein synthesis inhibitors: fusidic acid 10 µg (FA) and chloramphenicol 30 µg (C); (vi) tetracyclines: minocycline 30 µg (MN) and tetracycline 30 µg (TE); (vii) polymyxins: colistin 25 µg (CL); (viii) macrolides: erythromycin 15 µg (E), lincomycin 15 µg (L), and pristinamycin 15 µg (PT), (ix) rifamycins: rifampicin 5 µg (RF); (x) glycopeptides: vancomycin 30 µg (VA); (xi) others: fosfomycin 50 µg (FOS) and trimethoprim 1.25 µg/sulfamethoxazole 23.75 µg (TMP/SMX). The production of extended spectrum beta-lactamases (ESBL) was revealed by the double disc synergy technique, as described by Olonitola et al. (8). The amoxicillin/clavulanic acid disc was placed on the inoculated Mueller-Hinton plate agar between the ceftazidime and ceftriaxone discs at a distance of 2–3 cm from each center. After 18 to 24 h, the appearance of a champagne cork image between the antibiotic discs demonstrated the production of ESBL enzymes.

Quality control: American Type Culture Collection strains were routinely used as internal controls. In addition, the laboratory participated in external quality control organized by the National Institute for Communicable Diseases (Johannesburg, South Africa).

Data processing and statistics: The AST and AMR bacterial profiles were recorded using the GB2 application platform (INH data management application). Statistical analysis was performed using the Epi Info software version 3.5.4 (Centers for Disease Control and Prevention, Atlanta, GA, USA). The chi-square test with a P value (P) ≤ 0.05 was considered statistically significant.

RESULTS

Frequencies of analyzed samples and bacterial strains isolated in samples: The analyzed samples comprised 38.17% vaginal swabs (12,653/33,147), 33.24% urine samples (11,018/33,147), 11.61% stool samples (3,848/33,147), and 11.21% ejaculates (3,716/33,147). The numerator represents the number of a specific sample type and the denominator represents the total number of samples analyzed. From 33,147 samples, 3,497 Gram-positive and 1,970 Gram-negative (5,467 in total) strains were cultured. Among these 5,467 bacterial strains, 47.81 % were isolated from urine samples, 21.65 % from vaginal swabs, 6.12 % from ejaculates, 6.10 % from urethral swabs, 15.34 % from pus, and 7.07% from other sources (articular fluid, ascitic liquid, bronchial liquid, stools, sputum, and throat swabs).

The most commonly occurring species were *Enterobacteriaceae*, 57.6% (3,149/5,467); *Staphylococcaceae*, 21.1% (1,154/5,467); and *Streptococcaceae*, 10.5% (576/5,467). The numerator represents the number of specific species and the denominator represents the number of positive cultures. Bacteria isolated from urine samples were represented by *E. coli*, 61.4% (1,215/1,976); *Klebsiella ozaenae*, 56.2% (200/356); *Klebsiella pneumoniae*, 49.7% (186/374); group B *Streptococcus*, 31.5% (130/413); and *S. aureus*, 18.7% (92/492). Further, *Staphylococcus saprophyticus*, 54.7% (82/150); other coagulase negative staphylococci (CNS), 45.36% (230/507); *Enterococcus faecalis*, 95% (152/160); and *Acinetobacter* spp., 62.9% (56/89), were found in urine samples. The bacterial strains isolated from pus were *Pseudomonas aeruginosa*, 83.5% (147/176); *Pseudomonas* spp., 73.5% (36/49); and *S. aureus*, 47.6% (234/492). Group B *Streptococcus* were isolated from 27.1% (112/413) of vaginal swabs and 22.3% of urethral samples (92/413). The *Streptococcus pneumoniae* strains were isolated from stools, effusion liquids, and throat swabs (106/111,

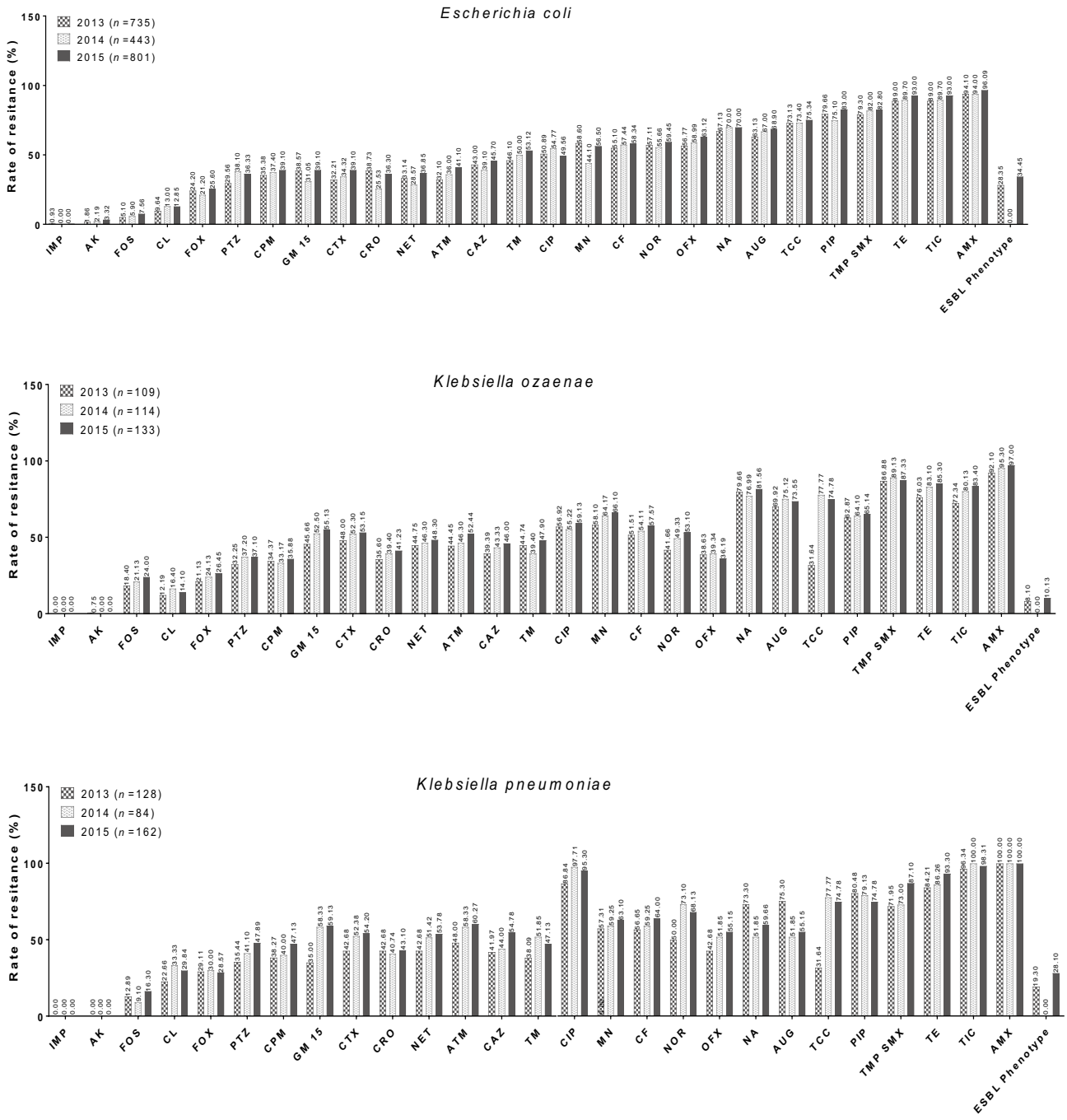

Fig. 1. Antibiotic resistance profiles of *Enterobacteriaceae*. Bars indicate the percentages (%) of bacteria strains resistance to each antibiotic. IMP, imipenem; AK, amikacin; FOS, fosfomycin; FOX, cefoxitin; PTZ, piperacillin/tazobactam; CPM, cefepime; GM, gentamicin; CTX, cefotaxime; CRO, ceftriaxone; NET, netilmicin; ATM, aztreonam; CAZ, ceftazidime; TM, tobramycin; CIP, ciprofloxacin; MN, minocycline; CF, cefalotin; NOR, norfloxacin; OFX, ofloxacin; NA, nalidixic Acid; AUG, augmentin; TCC, ticarcillin/clavulanic acid; PIP, piperacillin; TMP/SMX, trimethoprim/sulfamethoxazole; TE, tetracycline; TIC, ticarcillin; AMX, amoxicillin.

95.5%), whereas *Neisseria gonorrhoeae* was only found in urethral samples. The numerator represents the number of specific species per sample, and the denominator represents the number of isolates of the specific species in all samples.

Antibiotic resistance profile of *Enterobacteriaceae*: *Escherichia coli* was resistant over the 3 years to piperacillin (79.66%, 75.1%, and 83%), trimethoprim/sulfamethoxazole (79.3%, 82%, and 82.8%), ticarcillin (89%, 89.7%, and 93%), and amoxicillin (94.1%, 94%, and 96.09%) (Fig. 1). However, they were susceptible to imipenem, amikacin, and fosfomycin (> 90% of the isolates). Similar resistance was observed with *K. pneumoniae* and *K. ozaenae* strains excepted an important proportion of strains, resistant to minocycline (86.86–95.30%) and increase proportion of *K. pneumoniae* isolates resistant to ticarcillin/clavulanic acid (Fig. 1). *Escherichia coli* and *K. pneumoniae* ESBL-positive strains were more resistant to the majority of antibiotics than ESBL-negative strains.

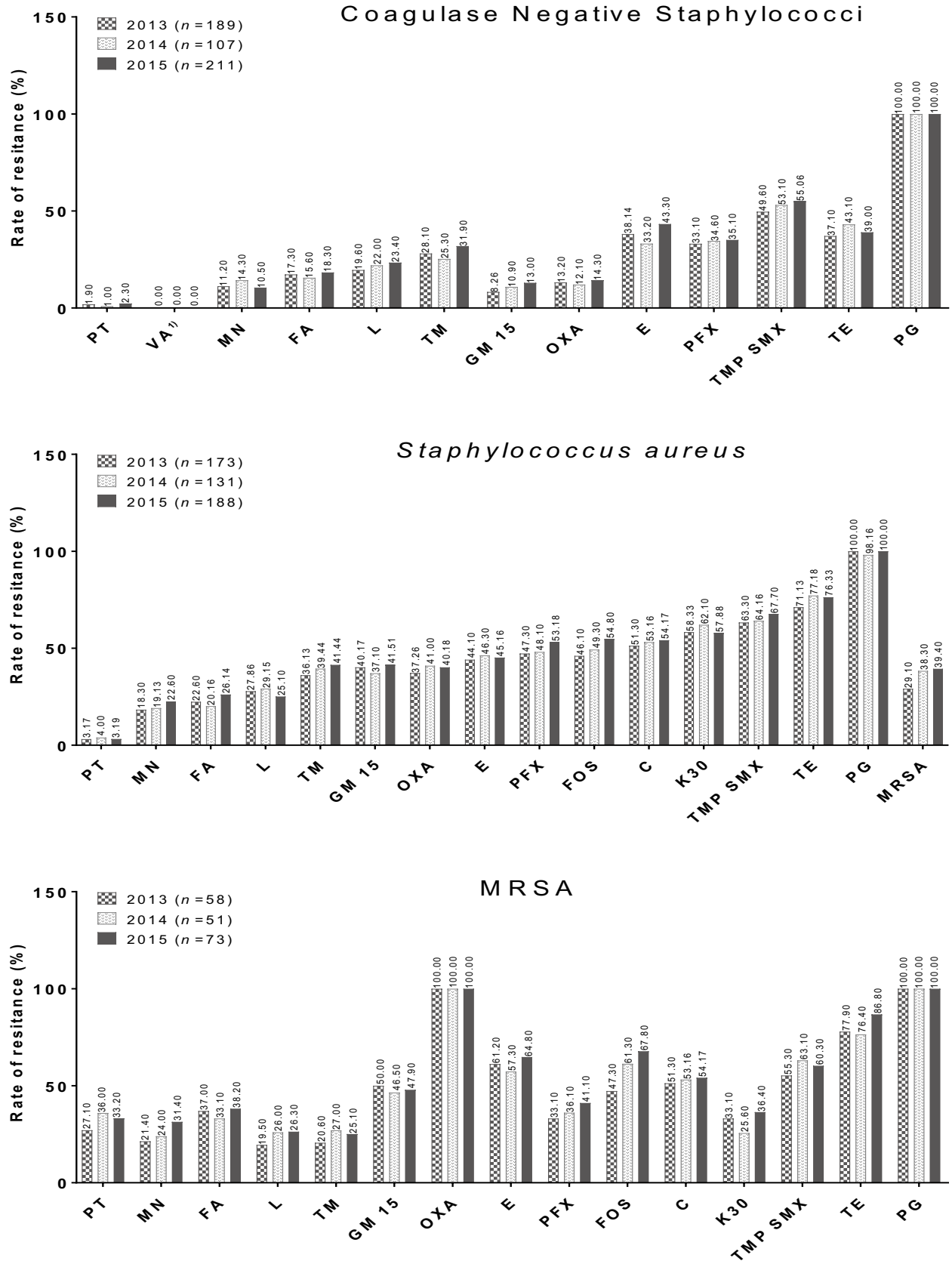

Fig. 2. Antibiotic resistance profile of *Staphylococcus* species. Bars indicate the percentages (%) of bacteria strains resistance to each antibiotic. PT, pristinamycin; VA[1)], vancomycin; MN, minocyclin; FA, fusidic acid; L, lincomycin; TM, tobramycin; GM15, gentamicin 15 µg; OXA, oxacillin; E, erythromycin; PFX, pefloxacin; FOX, cefoxitin; C, chloramphenicol; K30, Kanamycin 30 µg; TMP/SMX, trimethoprim/sulfamethoxazole; TE, tetracycline; PG, penicillin G; MRSA, Methicillin-resistant *Staphylococcus aureus*. [1)]: Bacterial strains belonging to *Staphylococcaceae*, *Streptococcaceae*, and *Enterococcaceae* families, non-susceptible to a given antibiotic whose resistance category is not defined in CA-SFM 2013, were considered resistant in this study.

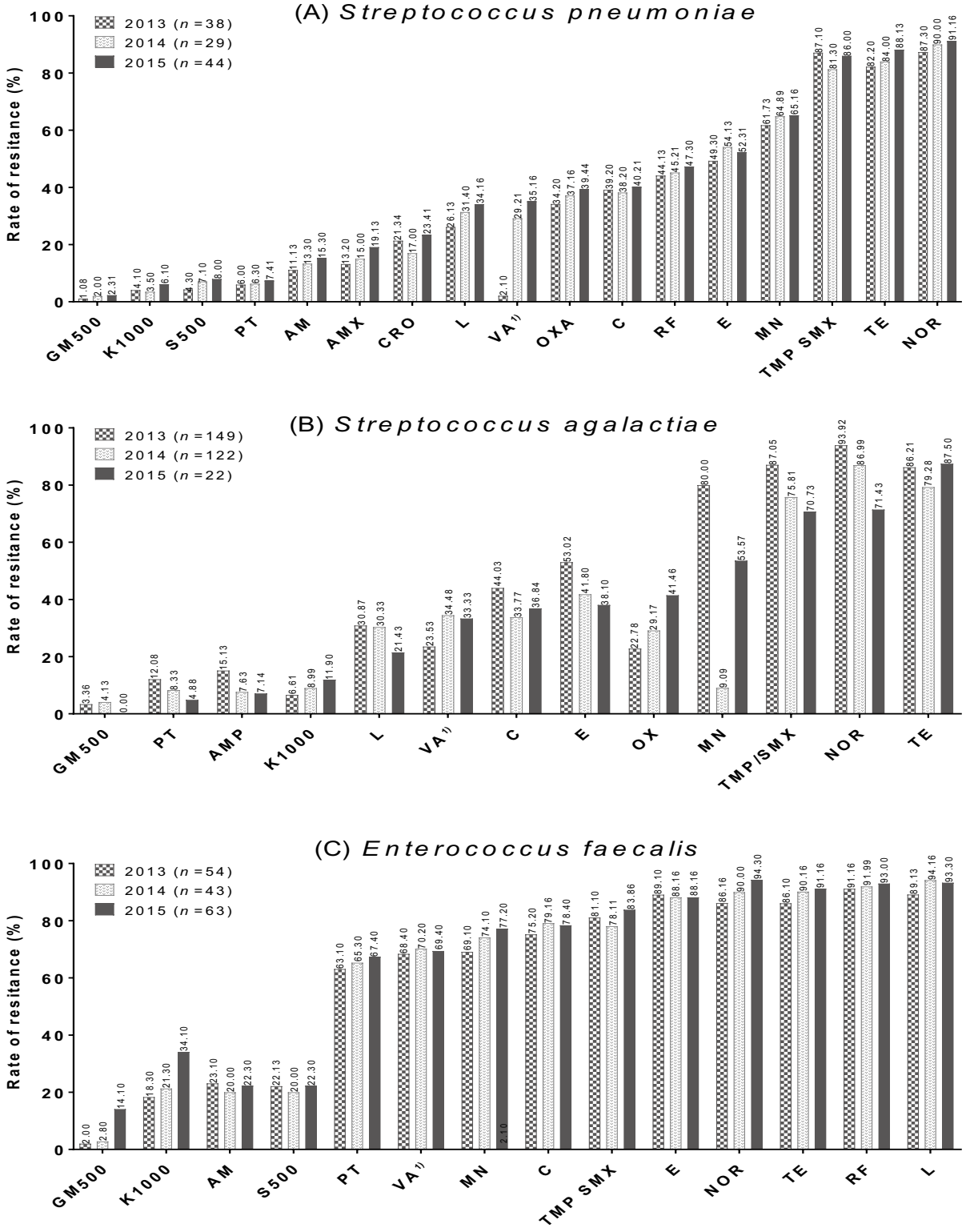

Fig. 3. Antibiotic resistance profile of *Streptococcus pneumoniae*, *Streptococcus agalactiae*, and *Enterococcus faecalis*. Bars indicate the percentages (%) of bacterial strains resistant to each antibiotic. (A) *Streptococcus pneumoniae*; GM500, gentamicin 500 µg; K1000, kanamycin 1000 µg; S500, streptomycin 500 µg; PT, pristinamycin; AM, ampicillin; AMX, amoxicillin; CRO, ceftriazone; L, lincomycin; VA[1)], vancomycin; OXA, oxacillin; C, chloramphenicol; RF, rifampicin; E, erythromycin; MN, minocyclin; TMP/SMX, trimethoprim/sulfamethoxazole; TE, tetracycline; NOR, norfloxacin. (B) *Streptococcus agalactiae*; AMP, ampicillin; OX, oxacillin; GM500, gentamicin 500 µg; K1000, kanamycin 1000 µg; C, chloramphenicol; E, erythromycin; L, lincomycin; PT, pristinamycin; TE, tetracycline; TMP/SMX, trimethoprim/sulfamethoxazole; NOR, norfloxacin; VA[1)], vancomycin; MN, minocyclin. (C) *Enterococcus faecalis*; GM500, gentamicin 500 µg; K1000, kanamycin 1000 µg; AM, ampicillin; S500, streptomycin 500 µg; PT, pristinamycin; VA[1)], vancomycin; MN, minocycline; C, chloramphenicol; TMP/SMX, trimethoprim/sulfamethoxazole; E, erythromycin; NOR, norfloxacin; TE, tetracycline; RF, rifampicin; L, lincomycin. [1)]: Bacterial strains belonging to *Staphylococcaceae*, *Streptococcaceae*, and *Enterococcaceae* families, non-susceptible to a given antibiotic whose resistance category is not defined in the CA-SFM 2013, were considered as resistant in this study.

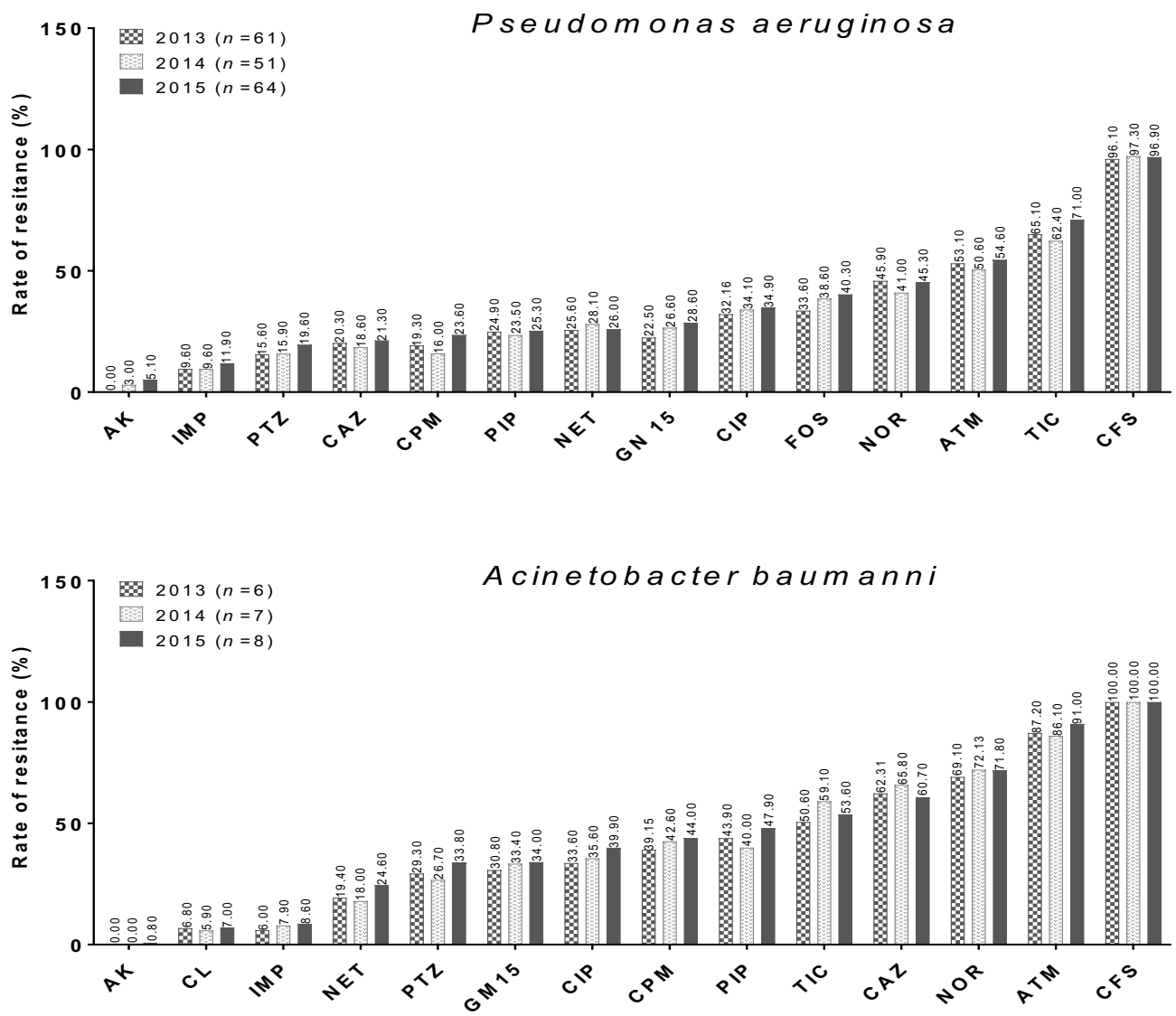

Fig. 4. Antibiotic resistance profiles of *Pseudomonas aeruginosa* and *Acinetobacter baumannii* isolates. Bars indicate the percentages (%) of bacteria strains resistance to each antibiotic. AK, amikacin; CL, colistin; IMP, imipenem; PTZ, piperacillin/tazobactam; CAZ, ceftazidime; NET, netilmicin; FOS, fosfomycin; CIP, ciprofloxacin; CPM, cefepime; GM15, gentamicin 15 µg; PIP, piperacillin; NOR, norfloxacin; ATM, aztreonam; TIC, ticarcillin; CFS, cefsulodin.

Antibiotic resistance profile of *Staphylococcus* species: We found that 37% of *S. aureus* were methicillin resistant (MRSA). The CNS were more resistant to penicillin G (100%) than trimethoprim/sulfamethoxazole (49–55%) and erythromycin (38–43%). CNS isolates (> 98%) were highly susceptible to antibiotics such as vancomycin and pristinamycin. However, *S. aureus* strains were highly resistant to penicillin G (98.16–100% resistant) but susceptible to pristinamycin (96.5–98.3% sensitive). Over 70% of the tested strains were susceptible to lincomycin, fusidic acid, and minocycline, and more than 50% of the strains were resistant to cefoxitin, chloramphenicol, kanamycin, trimethoprim/sulfamethoxazole, and tetracycline. Moreover, important resistance profiles were observed for the MRSA phenotypes. Indeed, MRSA phenotypes were resistant to tetracycline (79.9–86.8%), erythromycin (61.2–64.8%), and trimethoprim/sulfamethoxazole (55.3–60.3%) (Fig. 2).

Antibiotic resistance profile of *Streptococcaceae*: *Streptococcaceae* isolates were resistant to trimethoprim/sulfamethoxazole (78.11–87.1%), tetracycline (82.2–91.16%), and norfloxacin (86.16–94.3%). *Streptococcus pneumoniae* strains were susceptible to gentamycin, kanamycin, streptomycin, pristinamycin, ampicillin, amoxicillin, and ceftriaxone. *Streptococcus agalactiae* was susceptible to gentamicin, pristinamycin, ampicillin, and kanamycin 1,000 µg (> 84%) (Fig. 3).

The *E. faecalis* strains were resistant to 10 out of 14 antibiotics tested. Of the tested strains, 70% were susceptible to gentamicin, kanamycin, ampicillin, and streptomycin. But >87% resistance was observed for erythromycin, norfloxacin, tetracycline, rifampicin, and lincomycin (Fig. 3).

Antibiotic resistance profile of *P. aeruginosa* and *Acinetobacter baumannii*: *Pseudomonas aeruginosa* strains were highly susceptible to amikacin (3–5.18%

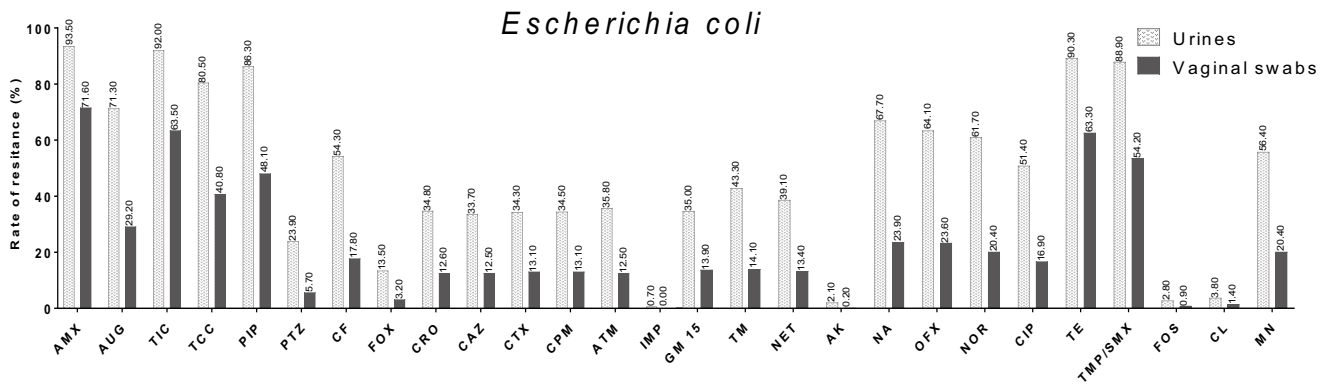

Fig. 5. Antibiotic resistance profiles of *Escherichia coli* strains isolated from vaginal swabs and urine samples. Bars indicate the percentages (%) of bacteria strains resistance to each antibiotic. AMX, amoxicillin; AUG, augmentin; TIC, ticarcillin; TCC, ticarcillin/clavulanic acid; PIP, piperacillin; PTZ, piperacillin/tazobactam; CF, cefalotin; FOX, cefoxitin; CRO, ceftriaxone; CAZ, ceftazidime; CTX, cefotaxime; CPM, cefepime; ATM, aztreonam; IMP, imipenem; GM15, gentamicin 15 µg; TM, tobramycin; NET, netilmicin; AK, amikacin; NA, nalidixic acid; OFX, ofloxacin; NOR, norfloxacin; CIP, ciprofloxacin; TE, tetracycline; TMP/SMX, trimethoprim/sulfamethoxazole; FOS, fosfomycin; CL, colistin; MN, minocycline.

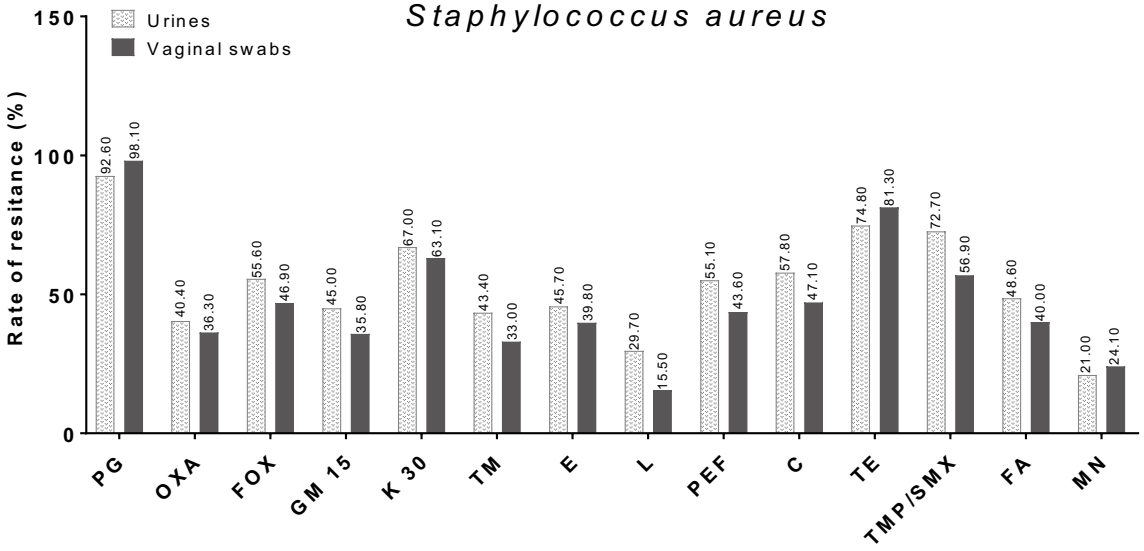

Fig. 6. Antibiotic resistance profiles of *Staphylococcus aureus* isolated from vaginal swabs and urine samples. Bars indicate the percentages (%) of bacteria strains resistance to each antibiotic. PG, penicillin G; OXA, oxacillin; FOX, cefoxitin; GM15, gentamicin 15 µg; K30, kanamycin 30 µg; TM, tobramycin; E, erythromycin; L, lincomycin; PEF, pefloxacin; C, chloramphenicol; TE, tetracycline; TMP/SMX, trimethoprim/sulfamethoxazole; FA, fusidic acid; MN, minocycline.

were resistant) as well as piperacillin, piperacillin/tazobactam, ceftazidim, cefepime, netilmicin, gentamicin, and imipenem (11–27% were resistant). However, they were resistant to ciprofloxacin, fosfomycin, norfloxacin, aztreonam, and ticarcillin (37–67%). Interestingly, *P. aeruginosa* was resistant to cefsulodin (> 96%).

Acinetobacter baumannii strains were susceptible to antibiotics such as amikacin, chloramphenicol, and imipenem (resistance < 8%). Moreover, the lowest resistance rate was observed for amikacin (0.9%). In contrast, the resistance rates were >70% for norfloxacin, aztreonam, and cefsulodine (Fig. 4).

Antibiotic resistance profiles of *N. gonorrhoeae*: All *N. gonorrhoeae* strains were tested with ceftriaxone, colistin, amoxicillin, tetracycline, and penicillin antibiotics. Approximately 10% of them were resistant to ceftriaxone, and 33.3% were resistant to colistin. In addition, high resistance rates to amoxicillin (90%), tetracycline (91.7%), and penicillin G (100%) were observed.

Comparison of *E. coli* and *S. aureus* antibiotic resistance profiles in urine samples and vaginal swabs: To determine the relationship between the origin of isolates and AMR, we compared the resistance profiles of the most common pathogens, *E. coli* and *S. aureus*, isolated from 2 main samples (urine and vaginal swabs). We found that *E. coli* isolated from urine had higher resistance rates to the 27 antibiotics tested than those isolated from vaginal swabs (Fig. 5). The rates of resistance to antibiotics observed among *S. aureus* strains isolated from urine samples were also higher than those isolated from vaginal swabs (Fig. 6).

DISCUSSION

In this study, we investigated common bacteria isolated from INH and their antibiotic resistance patterns. We could not estimate the vancomycin-intermediate *S. aureus* and vancomycin-resistant *S. aureus* phenotypes because we used the Kirby-Bauer diffusion method. The most frequent samples analyzed in this study were vaginal swabs (38.17%), followed by urine (33.24%). In Burkina Faso, Simporé et al. observed that urine samples were predominant (40.12% vs. 25.21% for vaginal swabs) (4). Another study in Morocco showed that urine samples were more frequently received (55.76%) compared to vaginal swabs (2.44%) (9). This difference may vary from a hospital bacteriology laboratory to a laboratory outside medical care facility. Indeed, studies have shown that urine cytobacteriological examinations are more urgent than vaginal swabs based on the high frequency of prescription by physicians, as urinary tract infection is the main reason for consultation in primary care medicine (10). In addition, INH is a national public health reference laboratory that collects specimens from outpatient clinics.

Enterobacteriaceae family (57.6%) and *S. aureus* (21.1%) were the most common pathogens isolated from samples. In contrast, a previous study targeting vaginal infections at Sokode in Togo found 4.40% *Enterobacteriaceae* and 5.49% *S. aureus* (11). However, our study results are comparable to those of Karou et al. 2012 (12) who found *E. coli* in 16.67% and *S. aureus* in 5.57% of the samples, in Ouagadougou. The slight differences in prevalence could be explained by the inclusion criteria and the focus of their study, which led to a distinct selection of samples. In urine, *Enterobacteriaceae* were the most isolated bacteria in this study. Our results are consistent with those obtained in studies in Morocco (9) and Senegal (13), with a predominance of *E. coli* and *Klebsiella* spp. *Escherichia coli* is believed to be the first germ frequently isolated in urinary tract infections (14) and is quantitatively the most important aerobic species present in the intestine, ranging from 10^7 to 10^9 bacteria/gram of stool contaminating urine samples (15). CNS was predominately found in urine (45.36%) compared to the rest of the samples. Similar results were observed for *S. saprophyticus*, *Enterococcus* spp., group B *Streptococcus*, and *Acinetobacter* spp. Indeed, it has been shown that these strains have a great ability to adhere to the urinary epithelium (16), which explains their high presence in urine. In contrast to fewer *N. gonorrhoeae* strains found in this study, another study performed in Togo in 2000 revealed high rates of *N. gonorrhoeae* with a prevalence of 28.78% in men and 7.69% in women (17), indicating that sexually transmitted infections due to *N. gonorrhoeae* declined in 2015 compared to 2000.

Enterobacteriaceae strains were highly resistant to most antibiotics except for imipenem, amikacin, and fosfomycin. Similar results were obtained for imipenem and amikacin in a study in Algeria (18). These drugs remain effective for any kind of resistant *Enterobacteriaceae*, even those producing beta-lactamase enzymes (19). Moreover, high resistance was noted to tetracycline, trimethoprim/sulfamethoxazole, and minocycline, when compared to a previous study where 28% and 30% of bacterial isolates had low sensitivity for tetracycline and minocycline, respectively (20). These results are consistent with those obtained in a study in Ouagadougou, where the authors found high resistance of Gram-negative bacilli to β-lactams and to trimethoprim/sulfamethoxazole (12). Indeed, *E. coli* is known to be naturally sensitive to all β-lactams. Nevertheless, in this study, high resistance rates were observed to all these molecules, especially amoxicillin (93.7%). This result is consistent with those obtained in a study in Nigeria (51.1–94.3%) (21) and in Marrakech (65%) (22). Indeed, the essential mechanism of acquired resistance to β-lactams is enzymatic, through the production of β-lactamases (23).

Escherichia coli strains were not susceptible to all the quinolones tested in this study, and a similar result was previously observed in a study in Morocco (22). The global epidemiological situation of resistance of *E. coli* strains to fluoroquinolones remains variable, with resistance ranging from 10% in the USA (24) to 50% in China (25). However, the rate of *E. coli* resistance to quinolones has increased in recent years (14). Resistance of the strains to third-generation cephalosporins could be explained by their production of beta-lactam inactivating enzymes, especially extended spectrum beta-lactamase, as shown by Toudji et al. (19). Their low sensitivity to cefoxitin may be due to the loss of membrane porins or the production of a cephalosporinase (26). In general, the sensitivity rates of *E. coli* strains in this study were higher than those observed in developed countries (27), not only to tigecycline but also to fluoroquinolones, cyclins, and trimethoprim/sulfamethoxazole. Indeed, antibiotic prescription and use policies differ from country to country and are not well implemented in low-income and middle-income countries (28). In contrast to inactive antibiotics, the isolated strains were highly susceptible to imipenem, amikacin, fosfomycin, and colistin, which could be explained by the fact that these antibiotics are not available without a prescription. Therefore, they are treatment options for severe *Enterobacteriaceae* infections (29).

The most effective antibiotics against *Staphylococcus* spp. are pristinamycin, vancomycin, and minocycline. The highest resistance was noted to penicillin G (100%). These results are similar to those obtained in a study in Mauritania (96–100%) (30), and interestingly a previous study reported that resistance to this antibiotic appeared in 1941 by the acquisition of a plasmid penicillinase that degrades penicillin (31). More than half of the staphylococcal strains were resistant to cefoxitin. The resistance to this antibiotic indicated that these strains developed cross-resistance between methicillin, oxacillin, and other beta-lactams by the production of a penicillin binding protein (PBP) with low affinity for beta-lactams like PBP2a (32), which explains the slight increase in MRSA prevalence in this study (37%) compared to another study carried out in a teaching hospital (35.7%) in 2011 (11). The gene *mec*A, encoding PBP2a is carried by a chromosome that also carries resistance genes to other antibiotics, which explains their multi-resistant profile (31).

All the tested molecules belonging to the β-lactams and aminoglycosides family showed an average effectiveness on *Streptococcus* spp., confirming the results of a previous study on common antibiotics used in *Streptococcaceae* (18). Our results are also comparable to those found in a study in France, where 40% resistance to erythromycin was observed (33); in Burkina Faso, bacteria isolated from samples showed resistance to tetracycline, trimethoprim/sulfamethoxazole, and minocycline (12). For *Enterococcaceae*, high resistance patterns to a majority of antibiotics have been shown, and this multi-resistance to antibiotics, particularly vancomycin, was previously reported in the USA (34). In contrast, the *Pseudomonas* spp. strains were susceptible to amikacin, imipenem, piperacillin/tazobactam, ceftazidime, and cefepime. This was also confirmed by studies showing only 10% resistance to ceftazidime and a high sensitivity to imipenem (35).

Significant differences were noted between the resistance percentages of *Staphylococcus* spp. isolated from urine samples and vaginal swabs to lincomycin, pefloxacin, and trimethoprim/sulfamethoxazole. Differences were also observed between the percentages of *E. coli* resistant to all antibiotic molecules tested, depending on the sample type (urine or vaginal swabs). These results suggest that there is a relationship between the bacterial resistance profile to antibiotics and its origin, confirming the results of a previous study by Karou et al. (12) in Ouagadougou, Burkina Faso, where bacteria isolated from urine showed higher resistance to antibiotics than those from vaginal swabs. Indeed, bacteria develop resistance to a majority of antibiotics that are eliminated in urine compared to vaginal discharge because physicians often prescribe more antibiotics to treat urinary infections (36).

In conclusion, this study highlighted bacterial resistance to antibiotics in Togo. Public health stakeholders must develop policies to better monitor the use of antibiotics in the country. Further studies to screen the mechanisms of AMR and characterize the main genes involved are necessary to establish effective strategies for the control of AMRs.

Conflict of interest None to declare.

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