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Phenotypic and Genotypic Antibiotic Resistant Diarrheagenic *Escherichia coli* Isolated from Patients with Diarrhea in Ouagadougou, Burkina Faso

Haoua Cissé[1*], Assèta Kagambèga[1,2], Soutongnooma Caroline Bouda[1], Abdallah Sawadogo[1], Nicolas Barro[1]

[1]Laboratoire de Biologie Moléculaire d'Epidémiologie et de Surveillance des agents Transmissibles par les Aliments (LaBESTA), Université Joseph KI-Zerbo, Ouagadougou, Burkina Faso
[2]Ecole Normale Supérieure (ENS), Ouagadougou, Burkina Faso
Email: *haoucis6@gmail.com

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Abstract

Background: The emergence and spread of multidrug-resistant bacteria have become a major public health problem worldwide, particularly in developing countries such as Burkina Faso. This study aims to determine phenotypic and genotypic antibiotic resistant *diarrheagenic Escherichia coli* (DEC) from patients with diarrhea in Ouagadougou, Burkina Faso. **Methodology:** Microbiological and biochemical analysis were done to detect two hundred and ninety-two (292) strains. The susceptibility of the strains to antibiotics was determined by the agar disc diffusion method. 16-plex-PCR assays were carried out to detect both virulence and resistance genes encoding betalactams, quinolones, phenicols, tetracyclines and virulence gene of DEC. **Results:** Diarrheagenic *Escherichia coli* was detected in 8% (23/292) of patients with diarrhea using the 16-plex-PCR and 39.1% (9/23) of the DEC detected carry at least one resistance gene. Resistance rate in disc diffusion test was 86.96% to tetracycline, 65.23% to cotrimoxazole, 17.4% to nalidixic acid, 17.4% to norfloxacin, 17.4% to ciprofloxacin, 13.04% to ceftriaxone, 13.04% to cefotaxime, 8.7% to gentamicin, 8.7% to Chloramphenicol, 0% to netilmicin. The prevalence of different resistance genes in the studied strains varied from 44.4% to 5.5%. The gene Tet coding for resistance to tetracycline was found in 8 strains (44.4%). The CatA gene coding for resistance to Chloramphenicol was detected in 38.9% of isolates. The *qnrS*, bla_{SHV} and bla_{OXA} genes were each detected in 5.5% of isolates. No strain hosts the *qnrA*, *qnrB* and bla_{TEM} genes. **Conclusion:** This study identified β-lactams, quinolones, phenicols and te-

tracyclines resistance genes in DEC isolates from patients with diarrhea in Ouagadougou, Burkina Faso. These results indicate the need for a surveillance program to reduce the prevalence of resistance to Enterobacteriaceae strains in hospitals.

Keywords

Diarrheagenic *Escherichia coli*, Phenotypic, Genotypic, Resistance, Ouagadougou

1. Introduction

Escherichia coli is one of the pathogens for which resistance to antimicrobials poses a critical challenge in human health [1]. It includes commensal bacteria of the digestive tract, pathogenic bacteria and bacteria adapted to the environment [2] and causes a wide range of conditions. Strains of *E. coli* responsible for diarrhea (DEC) can be classified into five pathogenic groups (Enteroinvasive *E. coli* (EIEC), Enteropathogenic *E. coli* (EPEC), Enterohemorrhagic *E. coli* (EHEC), Enterotoxigenic *E. coli* (ETEC) and Entero-aggregative *E. coli* (ECEC) according to their virulence traits, invasion characteristics and induced pathologies [3] [4] [5] [6]. Several diarrheagenic pathotypes have been recognized based and virulence properties and the mechanisms of pathogenicity [7]. They are one of the most important causes of gastroenteritis morbidity worldwide and in Burkina Faso in particular [4] [8] [9] [10].

Antibiotics from the β-lactam family, quinolones, phenicols and tetracyclines are among the antibiotics commonly used for the treatment of a wide variety of bacterial infections in humans. This antibiotic treatment of common bacterial infections plays a crucial role in reducing morbidity and mortality of diseases; however, the increased and uncontrolled use of these antibiotics has led to an increase in antimicrobial resistance and therefore poses an increasingly serious threat to global public health [11].

Genetic mutation and the role of genetic mobile elements such as plasmids, integrons and transposons are the most common ways for antibiotic resistance distribution [12] [13]. According to [14], 700,000 deaths were attributable to bacterial resistance and this figure could rise to 10 million per year with about 4,150,000 deaths per year for the African region by 2050 if no action is taken.

Despite being a public health problem and although several antibiotic resistance gene studies have been carried out in Burkina Faso, this study examines both phenotypic and genotypic resistance of ECD across age groups in Burkina Faso.

2. Material and Methods

2.1. Site and Period of the Study

This study was conducted between August and November 2014 in four different hospitals in Ouagadougou, the capital city of Burkina Faso (**Figure 1**).

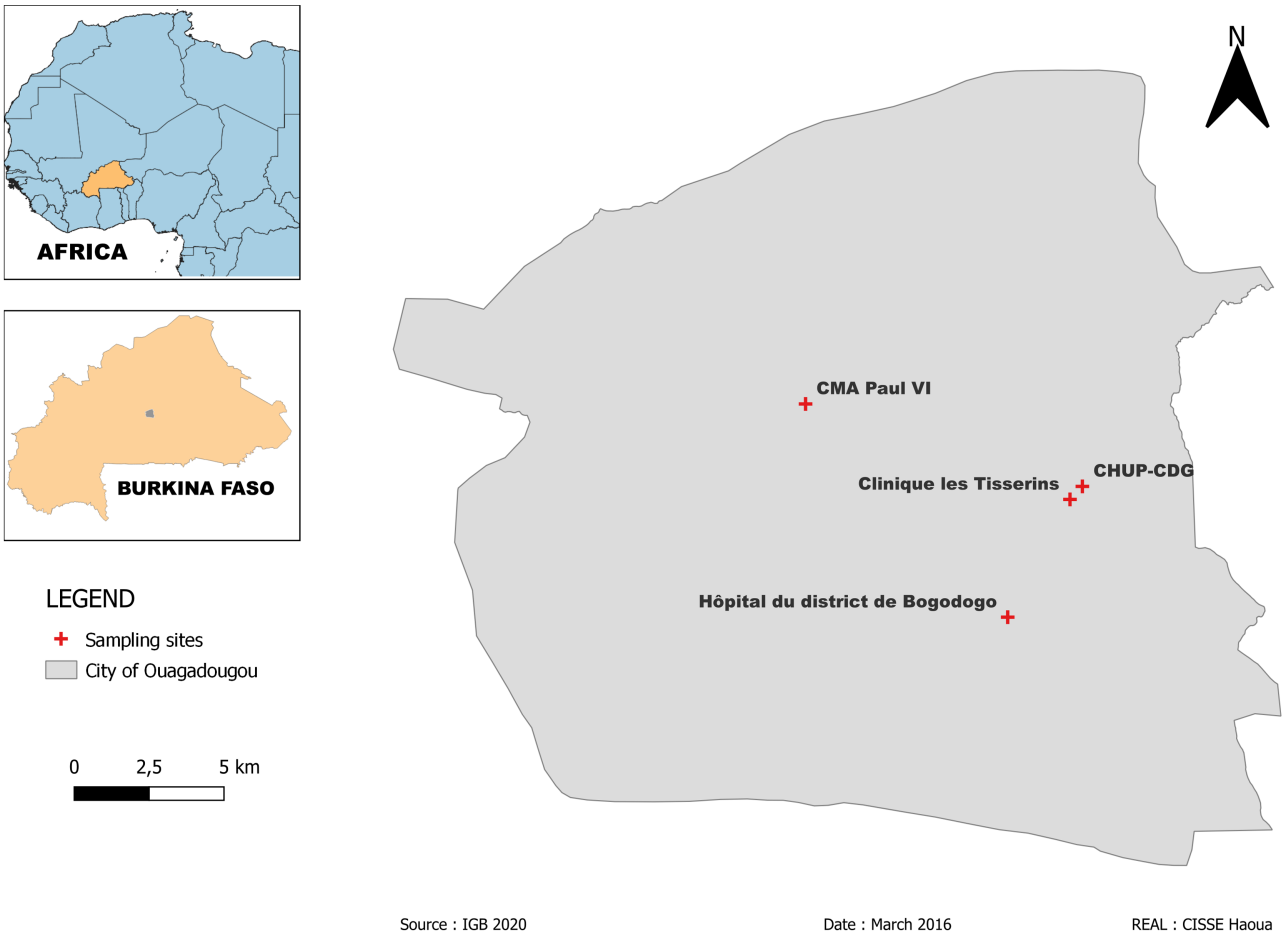

Figure 1. Map of Ouagadougou with the sites where sampling was done (CMA Paul VI, CHUP-CDG, Clinique les Tisserins and Hôpital du district de Bogodogo).

2.2. Sampling

The samples were collected between August, September (rainy season), October and November (dry season) in 4 hospitals (CMA Paul VI, Centre Hospitalier Universitaire Pédiatrique Charles De Gaulle, Clinique les Tisserins and Hôpital du district de Bogodogo) in Ouagadougou characterized by the alternation of two seasons, a dry season and a rainy or winter season. The study population consisted of people suffering from diarrhea during the study period and who were hospitalized or seen in consultation in one of the participating health centers. The samples were collected in sterile containers from diarrheal patients and transported to the Laboratory of Molecular Biology, Epidemiology and Surveillance of Bacteria and Viruses Transmissible through Food (LaBESTA), University of Ouagadougou, within 24 h in a cool box at 4˚C for immediate analysis. Sampling was done after obtaining the informed consent of patients. Sampling size was determined by the binomial formula described by Dagnelie (1998) by applying the formula No = $t^2 p(1-p)/m^2$ with a confidence interval of 95% and a margin of error "m" of 5%, we have t = 1.96. p being the prevalence of *E. coli*. According to [15], the prevalence of DEC was 24% among children with

diarrhea in Ouagadougou, Burkina Faso. Applying the formula, the estimated sample size was 280. Furthermore, as our study was periodic, we considered all the samples collected, reducing the size from 280 to 415. Moreover, as our study was periodic, we considered all samples collected, thus increasing the sample size from 280 to 415.

2.3. Bacterial Isolation

Isolation of suspected *E. coli* was carried out on to Eosin Methylene Blue (Liofilchem, Italy) and plates were incubated at 37˚C for 18 - 24 h. The colonies with green metallic color (suspect colonies) were selected and used for biochemical tests such as lactose, beta-glucuronidase, indole, citrate, and mannitol. *E. coli* strains isolated were confirmed by using API 20E tests (BioMérieux, France).

2.4. Antimicrobial Susceptibility Test

Antibiotic susceptibility of the of the strains of DEC was determined on Mueller Hinton agar using the following antibiotics Nalidixic acid 30 µg, Norfloxacin 5 µg, Ciprofloxacin 5 µg, gentamicin 10 µg, Netilmicin 30 µg, Ceftriaxone 30 µg; Cefotaxime 30 µg, Chloramphenicol 30 µg, sulfamethoxazole-trimethoprim 1.25 - 23.75 µg, Tetracycline 30 µg as described by the European Committee of Antimicrobial Susceptibility Testing [16].

2.5. Multiplex Polymerase Chain Reaction (16-Plex PCR)

16-plex PCR was used to identify resistance genes and to detect simultaneously 16 genes from the five main pathogroups of *E. coli* (enterohemoragic *E. coli*: EHEC, enteropathogenic *E. coli*: EPEC, enteroaggregative *E. coli*: EAEC, enteroinvasive *E. coli*: EIEC and enterotoxigenic *E. coli*: ETEC) [17].

To do this, extraction of genomic DNA was carried out by the thermal shock method. A fresh bacterial colony mass on Muller Hinton agar (Liofilchem, Italy) was taken emulsified in an Eppendorf tube with 1 ml of sterile water. The mixture was heated to 100˚C for 10 min and then centrifuged for 10 min at 12,000 rpm at 4˚C. Supernatant was then collected and used in the PCR reactions as DNA matrices. Molecular research of resistance markers involved the search for beta-lactam (bla_{OXA}, bla_{SHV}, bla_{TEM}), quinolone (qnrA, qnrB and qnrS), phenicol (CatA) and tetracycline (Tet) resistance genes in DEC strains.

The PCR assays were carried out in a 25 ml reaction mixture, which consisted of 2.5 µl of the supernatant added to 22.5 µl reaction mixture. This mixture contained 5U of Taq DNA polymerase (Accu Power, South Korea), deoxyribonucleic triphosphate (10 mM), buffer GC (10×), $MgCl_2$ (25 mM) and PCR primers (10 µM).

Amplification is carried out in a thermocycler (AB Applied Biosystems) according to the following PCR program: 94˚C for 5 minutes, 30 cycles of 94˚C for 30 s, 63˚C for 60 s and 72˚C for 60 s and the final elongation to 72˚C for 7 min.

Following PCR, the reaction products were separated using electrophoresis in

1.5% agarose gel (weight/volume) stained with Redsafe solution (Prolabo, France) and visualized under UV light (Gel Logic 200). Primers (Gene Cust, France) used for these amplifications are described in Table 1.

Table 1. Sequences of primers used.

Genes	Genetic resistance factors	Primers sequence (5' to3')	Weight (bp)
BlaOXA	β-Lactam genes (bla)	F: ATG AAA AAC ACA ATA CAT ATC R: AAT TTA GTG TGT TTA GAA TGG	813
BlaSHV		F: TTA TCT CCC TGT TAG CCA CC R: GAT TTG CTG ATT TCG CTC GG	768
BlaTEM		F: ATG AGT ATT CAA CAT TTC CG R: CCA ATG CTT ATT CAG TGA GG	1080
QnrA	Quinolones genes (Qnr)	F: TCAGCAAGAGGATTTCTA R: GGCAGCACTATTACTCCC	657
QnrB		F: GATCGTGAAAGCCAGAAAGG R: ACGATGCCTGGTAGTTGTCC	469
QnrS		F: ACGACATTCGTCAACTGCAA R: TAAATTGGCAACCTGTAGGC	417
CatA	Chloramphenicol genes (Cat)	F: AGTTGCTCAATGTACCTATAACC R: TTGTAATTCATTAAGCATTCTGCC	547
Tet	Tetracycline genes (Tet)	F: CCT CAG CTT CTC AAC GCG TG R: GCA CCT TGC TGA TGA CTC TT	634

2.6. Statistical Analysis

The chi-square (χ^2) test or Fisher's exact test of MedCalc was used to determine the statistical significance of the data. A value of $p < 0.05$ indicated statistical significance.

3. Results

Microbiological and biochemical analysis of the 415 samples identified and isolated 292 strains of *E. coli*. The 16-plex-PCR revealed 23 strains of *diarrheagenic Escherichia coli* (DEC) and five (05) types of resistance genes.

3.1. Phenotypic Resistances

Antibiogram of DEC strains showed resistance to several antibiotics. The highest resistances were observed with Cyclins (tetracycline) and Sulfonamides (cotrimoxazole) in proportions of 86.96% and 65.23% respectively (Figure 2). Low resistances are found for quinolones (nalidixic acid (17.4%), norfloxacin (17.4%) and ciprofloxacin (17.4%)), third-generation cephalosporins (ceftriaxone (13.04%),

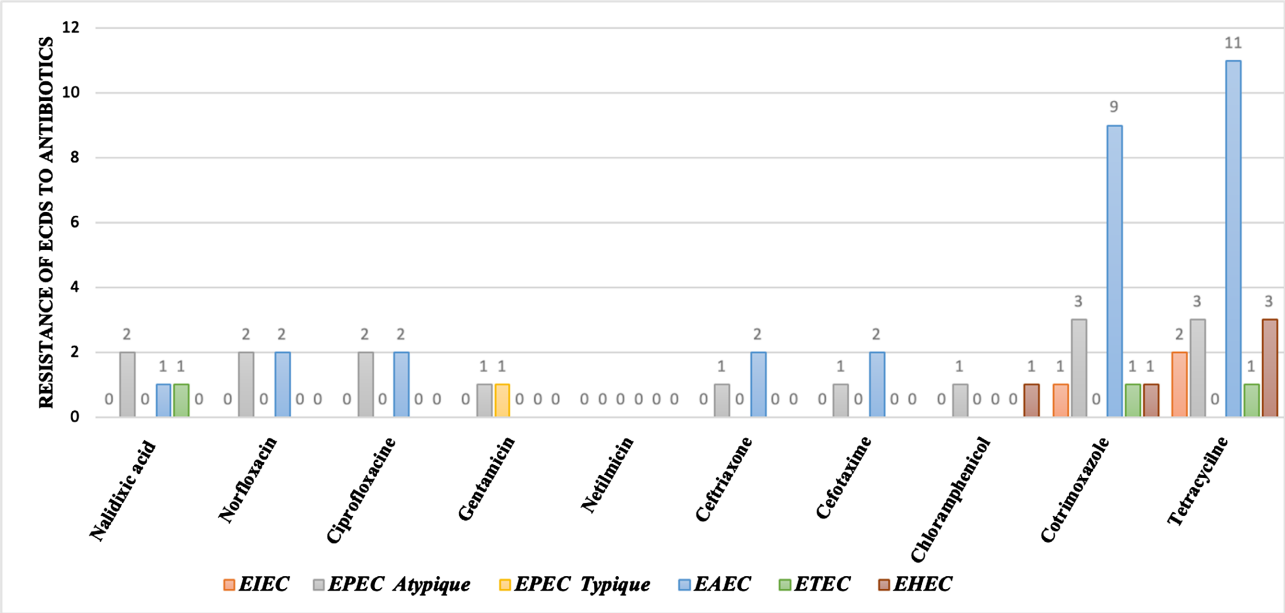

Figure 2. Resistance profile of ECDs strains to antibiotics.

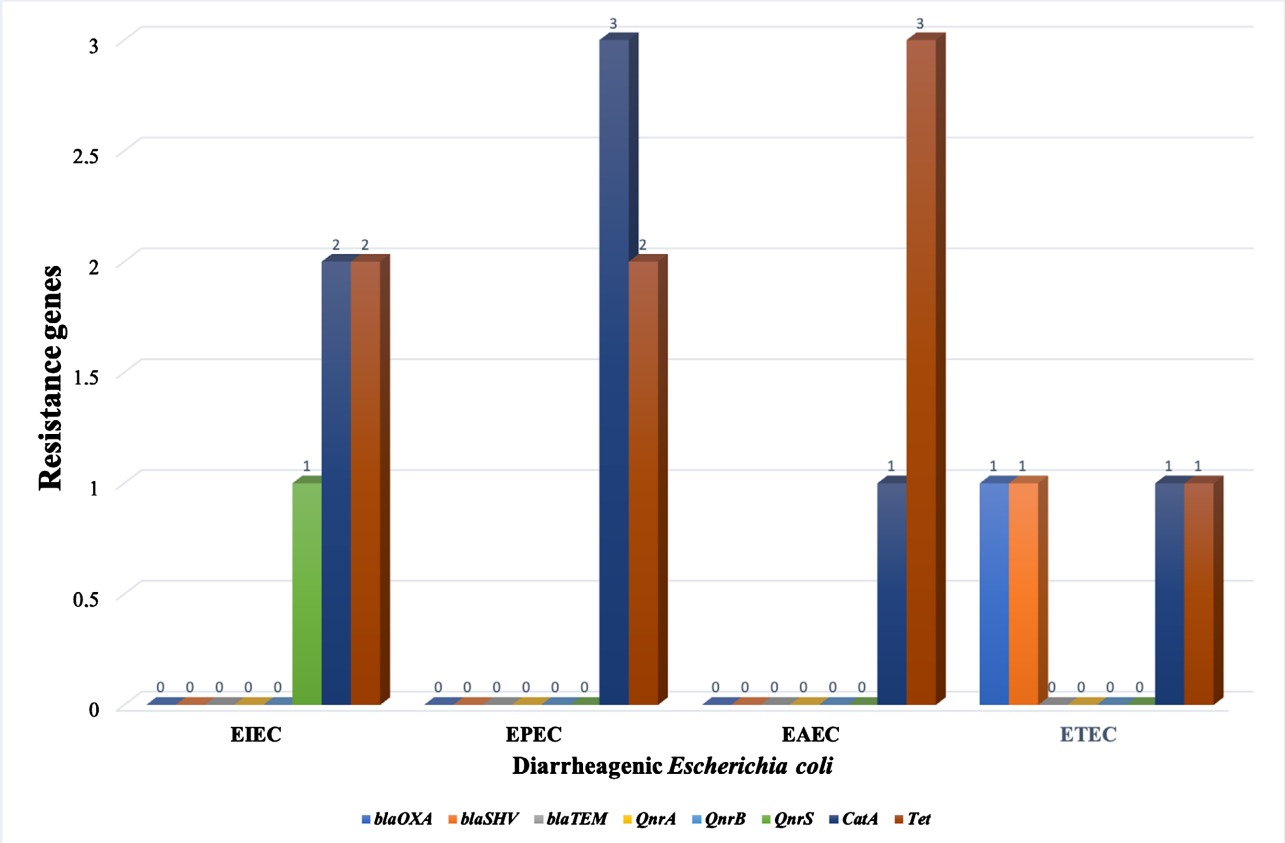

Figure 3. Carrying resistance genes.

cefotaxime (13.04%)), aminoglycosides (gentamicin (8.7%)) and phenicols (Chloramphenicol (8.7%)). However, no strain showed resistance to netilmicin, an antibiotic from the aminoglycoside family.

Table 2. Relationship between genotypic and phenotypic profile of DEC's microbial resistance.

Resistance genes/profile DEC	*β*-Lactam genes (bla)	Quinolones genes (Qnr)	Phenicol gene (Cat)	Cyclines gene
EIEC		QnrS	CatA	Tet/TE
EIEC			CatA	Tet/TE
EPEC			CatA	Tet/TE
EAEC				Tet/TE
EAEC			CatA	Tet
EAEC	CTR			Tet/TE
EPEC	CTR/CTX	NA/NX/CIP	CatA	TE
ETEC	bla_{OXA}/Bla_{SHV}	NA	CatA	Tet/TE
EPEC		NA/NX/CIP	CatA/C	Tet/TE

Legend: CTR: Ceftriaxone; CTX: Cefotaxime; C: Chloramphenicol; NA: Nalidixic Acid; TE: Tetracycline; NX: Norfloxacin; CIP: Ciprofloxacin.

In addition, the analysis of the antibiotic resistance profile also showed that 30.43% or 7 of the 23 ECD strains studied have resistance to at least 3 families f antibiotics.

3.2. Characterization of Betalactams, Quinolones, Phenicols and Tetracyclines Genes

Molecular research of resistance genes isolated in DEC revealed that of the twenty-three strains studied, eight (8) strains showed the presence of the *Tet* gene responsible for tetracycline resistance, a frequency of 34.78% (8/23). The beta-lactam resistance genes found were bla_{OXA}, bla_{SHV} each carried by an isolate (4.35%). The *CatA* gene was detected in seven (7) isolates or 30.43% while the quinolone resistance gene found was *qnrS* detected in one strain (4.35%). No bla_{TEM}, *qnrA* and *qnrB* genes were detected.

All of the nine isolates (100%) that carry resistance genes showed irregular relation between genotypic and phenotypic resistance. However, one of our isolates carried both *β*-Lactam (bla_{OXA}/bla), chloramphenicol (CatA) and tetracycline (Tet) resistance genes and had quinolone (NA) and tetracycline resistance phenotypes. Furthermore, of the DECs studied, all phenotypically resistant strains (89%) carry at least one resistance gene while strains (11%) that are phenotypically susceptible carry resistance genes but do not express.

4. Discussion

Diarrheagenic *Escherichia coli* is a major etiology of bacterial diarrhea, especially in developing countries [18] [19]. In the present study, of the 292 samples examined, DEC strains were isolated from 8% of them. Analysis of phenotypic

resistances of DEC strains shows that they are mainly resistant to tetracycline (86.96%). These results corroborate with those obtained by [20]-[26] who achieved high levels of tetracycline resistance in ECD strains isolated from patients with diarrhea. This resistance to tetracycline be explained by the fact that tetracyclines are one of the most widely used classes of antimicrobial agents in human and veterinary medicine because they have several advantages, including a wide spectrum of activity, low cost, oral administration and few side effects [27]. Tetracycline resistance is usually caused by the acquisition of tetracycline resistance genes and is attributed to one or more elements such as the acquisition of mobile genetic elements carrying tetracycline-specific resistance genes, mutations within the ribosomal binding site and/or chromosomal mutations resulting in increased expression of intrinsic resistance mechanisms [28].

Presences of antimicrobial resistance genes have been reported previously for clinical *E. coli* isolates from humans and animals [29]. Sulfonamide resistance was also observed in this work with a rate of 65.23% for sulfamethoxazole-trimethoprim. These results are lower than those obtained by [22] [26] [30] and [31] and higher than those obtained by [32].

This prevalence of resistance to sulfamethoxazole-trimethoprim is thought to be due to the fact that it is one of the most commonly prescribed antibiotics for suspected bacterial diarrhea and the most financially accessible. The high level of resistance to the most widely used antibiotics is also explained by their uncontrolled use and selection pressure.

In addition, our DEC strains showed relatively low resistance rates of 17.4% for quinolones (Nalidixic acid, Norfloxacin, Ciprofloxacin) and 13.04% for cephalosporins (Ceftriaxone, Cefotaxime). This relatively low resistance of quinolones could be explained by the fact that Qnr genes are known to confer a low-level resistance to fluoroquinolone in Enterobacteriaceae [33]. Resistance rates higher than those observed in our study were reported by [24].

For aminoglycosides and phenicols, DEC strains showed low levels of resistance. No resistance was observed with netilmicin (aminoglycoside) in this work. The total sensitivity of DEC strains to netilmicin could be explained by its efficacy and the fact that it is a limited-use molecule. It could therefore be an antibiotic of choice for the treatment of infections due to resistant bacteria.

In this study, a high prevalence of *E. coli* multidrug resistance was observed (30.43%). This multidrug resistance would be due to an abusive and uncontrolled use of antibiotics commonly used in clinical settings and would lengthen the duration and costs of treatment of these bacterial infections [34].

The prevalence of the Tet gene is 34.78% (8/23) followed by the *CatA* gene (30.43%), the bla_{OXA}, bla_{SHV}, *qnrS* genes (4.35%) and an absence of the genetic determinants bla_{TEM}, *qnrA* and *qnrB* (0%). The prevalence of qnr genes is 4.35%, with a predominance of the qnrS gene (4.35%) and an absence of the genetic determinant *qnrA* and *qnrB* (0%). These results are lower than those obtained in Morocco by [35] and in Niger by [36] where the qnrS gene was the most common followed by the *qnrB* and *qnrA* gene with respective proportions of 2.64.

and 64.3% for *qnrS*, 1.32% and 26.2% for *qnrB* and 0% and 9.5% for *qnrA*. This difference could be explained by host, environmental and pathogen factors [37]. Indeed, Qnr genes are generally mediated by plasmids and can be easily propagated through gene transfer mechanisms [38] [39].

In the present study, four (44%) of our isolates are resistant to antibiotics studied without carrying corresponding resistance genes while (66.67%) isolates carry resistance genes but are not phenotypically resistant to antibiotics belonging to the same family. Indeed, the irregular relationship between genotypic and phenotypic resistances observed is in concordance with the results obtained by [40] [41] and can be explained by the fact that the prevalence of resistance phenotypes is most often a loyal reflection of antibiotic prescription habits and also of presence of other mechanisms of causing antimicrobial resistance such as efflux pumps [41] [42]. However, the presence of co-resistance to β-Lactam (bla_{OXA}/bla_{SHV}), chloramphenicol (CatA) and tetracycline (Tet) resistance genes has observed. This co-resistance is explained by the presence of resistance genes to different families of antibiotics on the same plasmid. This represents a model of efficiency dissemination of several simultaneous mechanisms of resistance [25]. In addition, high prevalence rates of DEC strains in BF animal feces were reported in the study conducted by [43] [44]. These results could indicate contamination through food, fruits or vegetables, or water or the environment contaminated by animal feces.

Concerning the limits, this study was conducted for four months out of the twelve months of the year.

5. Conclusion

The study we conducted showed that *E. coli* remains a major causative agent of diarrhea in hospitals. The DEC strains studied are both pathogenic and have multiple resistance to several antibiotics. This study revealed high prevalence of resistance of DEC to tetracycline and sulfonamides. Genotypic profiles revealed the presence of bla_{OXA}, bla_{SHV}, *CatA*, *qnrS* and Tet with high prevalence for Tet genes. These resistance genes are likely to be transferred to commensal or pathogenic bacteria.

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Authors' Contributions

CH performed the collection of the samples, and carried out the isolation, iden-

tification and characterization of the strains as well as writing the manuscript. BSC supervised the work in the laboratory and the writing of the manuscript. KA read and corrected the manuscript. BN read and approved the final version of the manuscript.

Conflicts of Interest

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

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