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Antibiotic resistance genes in diarrheagenic *Escherichia coli* (DEC) isolated from livestock organic wastes in Ouagadougou, Burkina Faso

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Diarrheagenic *Escherichia coli* (DEC) are often disseminated through the fecal matter of livestock and waste products including slurry and manure. The study aimed to characterize archived DEC recovered from cattle fecal matter, manure and slurry for quinolone resistance and extended spectrum beta-lactamases (ESBLs) with focus on trends in antimicrobial susceptibility patterns. The susceptibility of the bacteria was tested using standard laboratory procedures. Polymerase chain reaction (PCR) was carried out to detect the presence of *qnrA, qnrB, qnrS* genes and β-lactamase producing genes (bla_{ESBL}) such us bla_{TEM} and bla_{SHV}. About 91% of DEC strains were multidrug resistant (MDR) with non-susceptibility to ≥1 agent in ≥3 antimicrobial classes. The most common resistance was to amoxicillin-clavulanic acid (96.36%), followed by tretacycline (89.09%), ceftazidime (76.36%), and cefotaxime (70.780%). *qnrS* (18.2%) was the most prevalent quinolone resistant genes, followed by *qnrB* (7.2%) and *qnrA* (2%). bla_{TEM} (5.45%) was most prevalent than bla_{SHV} genes (3.6%). bla_{TEM} and bla_{SHV} genes were identified in double or multiple-carrying with *qnrS* and *qnrB*, no Beta-lactamase (ESBLs) producing strains were observed. This result highlights the importance of livestock fecal matter, manure, and slurries as a significant public health concern and a repository of antibiotic resistant gene.

Key words: Diarrheagenic Escherichia coli (DEC), Livestock's fecal matter, manure, slurry, antibiotics resistance, quinolone resistance genes, bla_{TEM}, bla_{SHV}, Burkina Faso.

INTRODUCTION

Diarrheagenic Escherichia coli (DEC) constitute one of the most important causes of gastrointestinal in developping countries (Okeke, 2009; Bonkoungou et al., 2012; Dembélé et al., 2015; Konaté et al., 2017)). Some

common pathotypes of DEC include the Enteroaggregative *E. coli* (EAEC), Enteropathogenic *E. coli* (EPEC), (ETEC), and Enteroinvasive *E. coli* (EIEC) (Okeke, 2009; Sidhu et al., 2013). DEC may account for life-threatening infections and harbor virulence properties such as haemolysins, toxins, effacement factors, and cytotoxic necrotic factors (Kaper et al., 2004).

DEC are present in livestock's fecal matter,waste as slurry, manure and are largely contracted through environment (Manyi Loh et al., 2016; Bako et al., 2017). Furthermore, the environment is increasingly being recognized for the role it might play in the global spread of clinically relevant antibiotic resistance (Singer et al., 2016). The nature of this crisis and its health and economic burdens prompt us to identify new alternatives as well as to implement new policies to combat resistance. The emergence of antimicrobial resistance mechanisms, especially those associated with mobile genetic elements, may enhances the possibility that virulence factors genes and antibiotic resistance genes are spread simultaneously, inducing the emergence of new pathogens (Chen et al., 2011; Koczura et al., 2012).

The last report of World Health Organization on antibiotics resistance showed that *E. coli* is commonly resistant to third-generation cephalosporins, including resistance conferred by extended spectrum beta-lactamases (ESBLs), and to quinolones (WHO, 2014).

In *E. coli*, the resistance is primarily associated with the association of mutations in the quinolone-resistance determining regions (QRDRs) of *gyrA* and *parC*, which encode topoisomerase II (DNA gyrase) and topoisomerase IV respectively (Hopkins et al., 2005).

DEC that harbor bla_{ESBL} genes such as the bla_{TEM} and bla_{SHV} ESBL genes (Hoseini et al., 2014; Strau et al., 2015) render ineffective many widely used beta-lactam antibiotics including the third-generation cephalosporin such as cefepime through a secretion of beta-lactamase thereby, limiting available therapeutic options for the treatment of infections caused by these bacteria (Straus et al., 2015).

This study aimed to investiguate the quinolone resistant mutations and ESBL genes among DEC isolated in cattle fecal matter, slurries and manure in Ouagadougou, Burkina Faso and and how these mutations correlates with antibiotic susceptibility profiles.

MATERIALS AND METHODS

Diarrheagenic *Escherichia coli* (DEC) strain

The study involved a total of 55 DEC strains identified from previews study (Bako et al., 2017) (Table 1). *E.coli* strains have been isolated from cattle feces and organic waste (manure and slurry) from four livestock markets in the city of Ouagadougou, Burkina Faso between May 2015 and May 2016. A 16-plex Polymerase Chain Reaction (PCR), was used to screen simultaneously the virulence genes specific for Shiga-toxin producing *E. coli* (STEC), Enteropathogenic *E. coli* (EPEC), Enterotoxigenic *E. coli* (ETEC), Enteroinvasive *E. coli* (EIEC) and Enteroaggregative *E. coli* (EAEC) (Müller et al., 2007; Antikainen et al., 2009, Kagambèga et al., 2012). The 16-plex PCR is based on the detection of 15 different pathogroup-specific virulence genes (Table 2). In addition, one *E. coli* specific gene, *uidA*, was included. Strains comprised 52 strains of Enterotoxinogene *E. coli* (ETEC), two strains of Shiga Toxin *E. coli* (STEC) and one strain of Enteroaggregative *E. coli* (EAEC).

The antibiotic susceptibility tests

Antibiotic susceptibility test was done onto Mueller-Hinton media (Liofilchem, Italy) plate media following the standardized disk diffusion method as described (Bauer et al., 1966) using 16 antibiotic disks. The 16 antibiotics was: amoxicillin clavulanic-acid (AUG, 30 µg), chloramphenicol (C, 30 µg), norfloxacin (NOR, 10 µg), tetracycline (TET, 30 µg), nalidixic-acid (NA, 30 µg), imipenem (IPM, 10 µg), aztreonam (ATM, 30 µg), ceftriaxon (CRO, 30 µg), trimethoprim -sulfate (SXT, 25 µg), ceftazidime (CaZ, 30 µg), nutrofurantoin (F, 300 µg), cefotaxime (CTX, 30 µg), ciprofloxacin (CIP, 5 µg), cephalotin (KF, 30 µg), gentamicin (CN, 10 µg), cefoxitin (FOX, 30 µg).

Inhibition diameters of the antibiotics were interpreted according to the European Committee on Antimicrobial Susceptibility Instructions (EUCAST 2015, 2017). The Double Disk Synergy Test (DDST) was used to detect extended-spectrum β-lactamase (ESBL) producing strain according to the european committee on antimicrobial susceptibility testing description. This test is based on the detection of synergy between an amoxicillin clavulanic-acid disc and two discs of third generation cephalosporin's (ceftriaxone and cefotaxime) separated by 2 to 3 cm. The synergy between the discs, gave the appearance of "champagne cork" shape.

Detection of quinolone resistance genes and some ß-lactamase genes

DNA extraction

DNA was extracted by the thermal shock method. A loopful of bacteria previously cultured on MacConkey sorbitol agar and reisolated on Mueller-Hinton media was transferred to an Eppendorf tube with 250 µL water (nuclease free). The mixture was boiled for 10 min and centrifuged for 1 min at 13000 *g*. The supernatant was used for in the PCR reactions.

Primers and PCR assay

Quinolone resistance genes as *qnrA, qnrB* and *qnrS*, ß-lactamase gene as bla_{TEM} and bla_{SHV}, were detected by conventional PCR using primers as described by Cattoir et al. (2007). The following primers were used: bla_{TEM} (blaTEM-R: CCAATGCTTATTCAGTGAGG; blaTEM-F: ATGAGTATTCAACATTTCCG), bla_{SHV} (blaSHV-R:

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Table 1. DEC strains and their origins.

S/N	Strain code	Source of strain	Pathotype
1	OMBKilFB2.3		ETEC
2	OMBSNFB5.1		ETEC
3	OMBSNFB13.2		ETEC
4	OMBSNFB7.3		ETEC
5	OMBSNFB4.4		ETEC
6	OMBSNFB13.4		ETEC
7	OMBKFB3.3		ETEC
8	OMBSNFB4.1		ETEC
9	OMBSNFB7.2	Cows fecal matter	ETEC
10	OMBKFB12.2		ETEC
11	OMBSNFB13.1		ETEC
12	OMBSNFB8.4		ETEC
13	OMBSNFB1.2		ETEC
14	OMBSNFB1.4		ETEC
15	OMBSNFB3.3		ETEC
16	OMBSNFB6.1		ETEC
17	OMBSNFB9.3		ETEC
18	OMBSNFB12.3		ETEC
19	OMBOIFM10.1		ETEC
20	OMBSNFM9.1		ETEC
21	OMBSNFM10.1		ETEC
22	OMBSNFM1.4		ETEC
23	OMBSNFM2.1		ETEC
24	OMBSNFM6.1	Sheep fecal matter	ETEC
25	OMBSNFM13.4		ETEC
26	OMBSNFM5.2		ETEC
27	OMBSNFM3.2		ETEC
28	OMBSNFM3.1		ETEC
29	OMBSNFM4.3		STEC
30	OMBKFM4.1		ETEC
31	OMBSNFM2.2	Sheep fecal matter	ETEC
32	OMBSNFM1.2		ETEC
33	OMBOIFC2.3		ETEC
34	OMBSNFC7.3		ETEC
35	OMBKilFC1.3		ETEC
36	OMBOIFC9.4		ETEC
37	OMBSNFC4.1	Goat fecal matter	ETEC
38	OMBSNFC7.3		ETEC
39	OMBSNFC3.3		ETEC
40	OMBOIFC4.1		ETEC
41	OMBKilFC2.4		ETEC
42	OMBSNLB6.3		ETEC
43	OMBOILB11.1		ETEC
44	OMBOILB9.1		ETEC
45	OMBOILB12.1		ETEC
46	OMBOILB10.1	Slurry	ETEC
47	OMBSNLB3.3		ETEC
48	OMBSNLB6.2		ETEC
49	OMBSNLB7.2		ETEC
50	OMBSNLB2'.1		ETEC

Table 1. Contd

51	OMBSNF6.4		EAEC
52	OMBOIF2.2		ETEC
53	OMBOIF2.1	Manure	ETEC
54	OMBOIF4.2		STEC
55	OMBOIF6		ETEC

EAEC, Enteroaggregative *E. coli* ; ETEC, Enterotoxinogenic *E. coli*, STEC, Shiga-toxin producing *E. coli.*

Table 2. The virulence genes in the 16-plex PCR.

Pathogroup	Gene	Locus	Action	Reference
STEC	*stx1*	Phage	Shiga toxin 1	(Paton and Paton, 1998)
STEC	*stx2*	Phage	Shiga toxin 2	(Paton and Paton, 1998)
STEC, some EPEC	EHEC-*hlyA*	Virulence plasmid pO157	Enterohemolysin	(Paton and Paton, 1998)
STEC, EPEC	*eae*	LEE pathogenicity island in the chromosome	Intimin, a protein causing attaching/effacing lesions	(Nataro and Kaper, 1998)
STEC, EPEC	*escV*	LEE pathogenicity island in the chromosome	A conserved area in LEE pathogenicity island, type III secretion system structure protein	(Muller et al., 2007)
STEC, EPEC	*ent*	OI-122 pathogenicity island in the chromosome	Enterotoxin or enterohemolysin, a homolog to ShET2 enterotoxin of *Shigella flexnerii*	(Muller et al., 2007, Afset et al., 2008)
tEPEC	*bfpB*	EPEC adherence factor (EAF) plasmid	Subunit of Bundle forming pilus (BFP)	(Nataro and Kaper, 1998, Muller et al., 2007)
ETEC	*elt*	Plasmid	Heat-labile enterotoxin LT-I	(Nataro and Kaper, 1998, Kaper et al., 2004)
ETEC	*estIa*	Plasmid or transposon	Heat-stable enterotoxin STIa (STIp, porcine)	(Nataro and Kaper, 1998, Kaper et al., 2004)
ETEC	*estIb*	Plasmid or transposon	Heat-stable enterotoxin STIb (STIh, human)	(Nataro and Kaper, 1998, Kaper et al., 2004)
EIEC	*invE*	Virulence plasmid pINV	Transcription regulator, regulates the *ipa* genes	(Hale, 1991, Muller et al., 2007)
EIEC	*ipaH*	Virulence plasmid pINV and the chromosome	Invasion plasmid antigen	(Hale, 1991)
EAEC	*aggR*	Chromosomal island, plasmid pAA	AggR regulon, transcription activator, regulates the genes of fimbrial biogenesis	(Kaper et al., 2004, Harrington et al., 2006)
EAEC	*pic*	Chromosome	Serine protease	(Henderson et al., 1999, Muller et al., 2007)
STEC, EPEC, ETEC, EIEC, EAEC, E. coli	*astA*	Plasmid	EAEC heat-stable enterotoxin (EAST-1)	(Nataro and Kaper, 1998)
STEC, EPEC, ETEC, EIEC, EAEC, E. coli	*uidA*	Chromosome	β-glucuronidase	(Muller et al., 2007)

EAEC, Enteroaggregative *E. coli* ; ETEC, Enterotoxinogenic *E. coli*, STEC, Shiga-toxin producing *E. coli.*

GATTTGCTGATTTCGCTCGG; blaSHV-F: TTATCTCCCGTTAAGCCACC), qnrB (QnrB-F: GATCGTGAAAGCCAGAAAGG; QnrB-R: ACGATGCCTGGTAGTTGTCC), qnrS (QnrS-F: ACGACATTCGTCAACTGCAA; QnrS-R: TAAATTGGCACCCTGTAGGC), qnrA (QnrA-F: TCAGCACAAGAGGATTTCTC; QnrA-R: GGCAGCACTATTACTCCCA). The reaction mixture (20 µl) contained 4 µl of 5x FIREPol® Master Mix Ready to Load with 7.5 mM MgCl2 (Solis biodyne, Estonia), 1 µl of each primer, and 1 µl of DNA template 14 µl of water (nuclease free). The samples were gently vortexed and the PCR were performed using the thermal cycling condition including the annealing temperatures for each gene. Thermocycling conditions were 94°C for 5 min, following to35 cycles at 94°C for 30s and annealing temperatures were respectively 52°C, 54°C, 57°C, 55°C, 54°C, for bla_{TEM}, bla_{SHV}, *qnrB,*

Table 3. The prevalence of diarrheagenic *Escherichia coli* (DEC) resistance to antibiotics.

Antibiotics	Cattle fecal matter									Strain isolated from slurry (n=9)			Strain isolated from manure (n= 5)			Total
	Strain isolated from cow fecal matter (n= 18)			Strain isolated from sheep fecal matter (n= 14)			Strain isolated from goat fecal matter (n= 9)									
	STEC	ETEC	EAEC	STEC	ETEC	EAEC	STEC	ETEC	EAEC	STEC	ETEC	EAEC	STEC	ETEC	EAEC	
Aztreonam (ATM)	0	11 (61.11%)	0	1 (7.14%)	10 (71.42%)	0	0	5 (55.55%)	0	0	4 (44.44%)	0	0	3 (60%)	1 (20%)	63.63%
Amoxicillin Acid Clavulanic (AUG)	0	17 (94.44%)	0	1 (7.14%)	12 (85.71%)	0	0	9 (100%)	0	0	9 (100%)	0	1 (20%)	3 (60%)	1 (20%)	96.36%
Cefalotin (KF)	0	7 (38.88%)	0	1 (7.14%)	9 (64.28%)	0	0	5 (55.55%)	0	0	3 (33.33%)	0	0	3 (60%)	1 (20%)	52.72%
Cefoxitin (FOX)	0	0	0	0	2 (14.28%)	0	0	3 (33.33%)	0	0	0	0	0	3 (60%)	1 (20%)	16.36%
Cefotaxime (CTX)	0	13 (72.22%)	0	1 (7.14%)	9 (64.28%)	0	0	7 (77.77%)	0	0	6 (66.66%)	0	0	3 (60%)	0	70.90%
Ceftriaxon (CRO)	0	8 (44.44%)	0	1 (7.14%)	7 (50%)	0	0	3 (33.33%)	0	0	1 (11.11%)	0	0	3 (60%)	1 (20%)	43.63%
Ceftazidime (CaZ)	0	16 (88.88%)	0	1 (7.14%)	11 (78.57%)	0	0	5 (55.55%)	0	0	6 (66.66%)	0	0	2 (40%)	1 (20%)	76.36%
Imipenem (IPM)	0	1 (5.55%)	0	0	1 (7.14%)	0	0	1 (11.11%)	0	0	0	0	0	1 (20%)	0	7.27%
Chloramphenicol	0	8 (44.44%)	0	0	6 (42.85%)	0	0	3 (33.33%)	0	0	0	0	0	1 (20%)	0	32.72%
Gentamicin (CN)	0	5 (27.77%)	0	1 (7.14%)	3 (21.42%)	0	0	2 (22.22%)	0	0	1 (11.11%)	0	0	1 (20%)	1 (20%)	25.45%
Acide Nalidixique (NA)	0	4 (22.22%)	0	1 (7.14%)	3 (21.42%)	0	0	0	0	0	0	0	0	1 (20%)	0	16.36%
Norfloxacin (NOR)	0	0	0	0	2 (14.28%)	0	0	0	0	0	0	0	0	0	1 (20%)	5.45%
Ciprofloxacin (CIP)	0	2 (11.11%)	0	0	2 (14.28%)	0	0	1 (11.11%)	0	0	0	0	0	0	1 (20%)	10.90%
Tretacycline (TE)	0	16 (88.88%)	0	0	12 (85.71)	0	0	7 (77.77%)	0	0	9 (100%)	0	1 (20%)	3 (60%)	1 (20%)	89.09%
Trimethropim/Sulfamethoxazol (SXT)	0	8 (44.44%)	0	0	7 (50%)	0	0	3 (33.33%)	0	0	2 (22.2%)	0	1 (20%)	2 (40%)	1 (20%)	43.63%
Nitrofurantoin (F)	0	5 (27.77%)	0	1(7.14%)	9 (64.28%)	0	0	2 (22.22%)	0	0	2 (22.22%)	0	0	1 (20%)	1 (20%)	38.18%

EAEC, Enteroaggregative *E. coli* ; ETEC, Enterotoxinogenic *E. coli*; STEC, Shiga-toxin producing *E. coli.*

qnrA, qnrS, and elongation at 72°C for 60 s. The ultimate extension was 72°C for 10 min. The amplicons were visualized by electrophoresis on 1% (weight / volume) gel agarose after migration in the TAE (Tris Acetic acid EDTA) buffer.

Statistical analysis

SPSS statistics 20 and Microsoft Excel were used for statistical analysis. Bivariate Spearman's rank correlation test was used to determine the association between variables of this study.

RESULTS AND DISCUSSION

The antibiotic susceptibility tests

The profile of antibiotics resistance revealed that DEC were resistant to all antibiotics used in this study. The most common resistance (Table 3) was for amoxicillin-clavulanic acid (96.36%) followed by tetracycline (89.09%), ceftazidime (76.36 %) and cefotaxime (70.90%). The resistance rates for ciprofloxacin and norfloxacin (anibiotics belonging to the family of quinolone) were 10.90% and 5.45% respectively. No ESBLs phenotype was reported in this study.

The prevalence of resistance to amoxicilin clavulanic acid (84.2%) is comparable to that obtained by Iweriebor et al. (2015) in a similar study conducted on DEC in Cape Town, South Africa.

This type of resistance is acquired and could be expressed by a decrease of the activity of the β-lactamase inhibitor which is clavulanic-acid, resulting from a penicillinase hyperproduction, or the inactivation of the inhibitor itself (Kamga et al., 2014). This fact is considered to be a consequence of selection pressure related to the abuse of these antibiotics (Kamga et al., 2014).

The resistance to tetracycline observed in this study is comparable to those obtained in South Africa (96.84%) and Nigeria (64.3%) in diarrheagenic Escherichia coli isolated from effluents from cattle (Ajayi et al., 2011; Iweriebor et al., 2015).

The resistance to tetracycline is widely disseminated in *E. coli*, where it is generally mediated by tetracycline efflux pumps, such as *tetA* (Stavropoulos and Strathdee, 2000; Møller et al., 2016). This high prevalence can be explained by the fact that in Burkina, oxytetracycline one of

antibiotic belonging to the tetracycline family is the most antibiotic used in animal health (Samandoulougou et al., 2016).

Ceftazidime is third generation cephalosporin antibiotics belonging to the family of β-lactam.

The resistance of strains to ceftazidime in this study is comparable to those obtained in South Africa (32%) and Nigeria (50.6%) in Escherichia coli isolated from cattle fecal matter and manure (Iweriebor et al., 2015; Ajayi et al., 2011).

The resistance to nalidixic acid, and ciprofloxacin can be explained in general by the fact that fluoroquinolones such as ciprofloxacin and nalidixic acid are less used in dairy cattle than in other species such as poultry (Lanz et al., 2003).

91% of DEC strains comprising 47 ETEC, 2 STEC and 1 EAEC isolated from cow fecal matter, sheep fecal matter, goat fecal matter, manure and slurry were multi Drug resistant with non-susceptibility to ≥1 agent in ≥3 antimicrobial classes. Among this multi-drug resistant strain, 2 (ETEC) strains isolated originated from cow fecal matter and sheep fecal matter were resistant to 14 antibiotics of 16 used in this study. No statistic significant correlation was found between the multi drug resistant character of the strains as well as the parameters such as the origin of the strain, the type of DEC. The multi-resistance could be explained by the combination of several resistance mechanisms which in most cases are encoded by molecular supports.

No statistic significant correlation was noted with the resistance to de different antibiotic family and the type of DEC pathotype.

Carriage of *qnrA, qnrB, qnrS*, bla_{TEM} and bla_{SHV} genes by DEC

This study is the first to focus on the sharing of genes coding for quinolone (qnrA, qnrB, qnrS) and Beta-lactam resistance (blaTEM, blaSHV) by DEC isolated from livestock's fecal matter, manure and slurries in Burkina Faso.

The PCR revealed the presence (Table 4) of *qnrA, qnrB, qnrS*, and bla_{ESBL} genes such us bla_{TEM} and bla_{SHV} among DEC. For quinolone gene, *qnrS* (18.2%) was most prevalent followed by *qnrB* (7.2%) and *qnrA* (2%). All *qnrS* positive strains comprised 14 ETEC from cattle fecal matter, six from slurry and one EAEC from manure. *qnrA* positive strains comprising three ETEC were isolated from sheep and cow fecal matter. *qnrB* positive strains comprising 11 ETEC were isolated from cattle fecal matter. Among all *qnr* gene positives strains, only 9 strains were resistant to antibiotic belonging to the quinolone family such us nalidixic-acid (NA), ciprofloxacin (CIP) and norfloxacin (NOR). Correlation has been found between the susceptibility of DEC to ciprofloxacin and the presence of the *qnrA* gene ($p = 0.003$). Correlation was also found between the resistance of DEC to cefoxitin, antibiotic form of cephalosporin class and the presence of *qnrS* ($p = 0.009$).

The prevalence of *qnrS* and *qnrB* in this study are comparable to those obtained for *qnrS* (5.60%) and *qnrB* (0.43%) from *E. coli* isolated from farm animal in China (Yue et al., 2008). There are no data concerning the carrying of qnrA gene by DEC isolated from livstock's fecal matter, manure and slurries.

In general, the presence of these acquired genes does not confer high level of fluoroquinolones resistance (Rodríguez-Martínez et al., 2011). This could explain the fact that a statistically significant correlation between the susceptibility to the antibiotics belonging to quinolone family was not obtained.

Double-carrying *qnrB* + *qnrS* (3.6%) and *qnrA* + *qnrS* (1.8%) has been also identified among DEC.

bla_{TEM} gene (5.45%) was the most prevalent bla_{ESBL} genes followed by bla_{SHV} gene (3.6%). bla_{TEM} or bla_{SHV} only positive strain (9.05%) were constituted by five ETEC, three from cattle fecal matter, one from manure and the second one from slurry. All positive bla_{TEM} gene and or the bla_{SHV} gene DEC resisted at least one antibiotic of the ß-lactam class.

The prevalance of blaTEM gene is comparable to those obtained in South Africa (27%) and South Korea (17.5%) in DEC (STEC) isolated from dairy cattle farms (Iweriebor et al., 2015; Dong et al., 2017).

Multiple carrying (Table 4) of blaESBL genes and quinolo resitance gens were also noted (24.2%) in DEC. These strains were composed to nine ETEC isolated from cattle fecal matter, one ETEC from slurry and one ETEC from manure. These strains were resistant to at least one antibiotic belonging ß-lactam family.

No significant statistic correlation was found between susceptibility to Beta-lactamin antibiotics involved in this sudy and the carrying of the bla_{TEM} and bla_{SHV} genes. Double carrying between bla_{TEM}, bla_{SHV}, *qnrB* and or *qnrS* gene was observed in 24.2% of the strains in this study. Indeed, *qnr* genes have been frequently associated with bla_{ESBL} genes such as blaTEM and blaSHV genes (Boyd et al., 2004; Woodford and Carattoli, 2009).

Conclusion

The study showed that there are a lot of multi drug resistant diarrheagenic *E. coli* which can get to the environment through cattle fecal matter slurry and manure from livestock market located in Ouagadougou, Burkina Faso. This is in line with WHO's observations on the emergence of resistance to beta-lactams, third-generation cephalosporins and quinolones. In fact, these pathogens carry molecular support such us qnrA, qnrB, qnrS, blaTEM and blaSHV. These results show the risk incurred by the population to the exposure of livestock cattle fecal matter and organic waste products of animal origin such as manure and slurries.

Table 4. Quinolone and Beta-lactam resistance genes identified among diarrheagenic *Escherichia coli* (DEC).

Resistance genes	Cattle Fecal matter (n=41)		Slurry (n=9)		Manure (n=5)		Total (n=55) (%)
	Nbr (%)	Pathotype	Nbr (%)	Pathotype	Nbr (%)	Pathotype	
Quinolone resistance only							
qnrA	1(2.4)	ETEC	0		0		1.8
qnrB	4(9.75)	ETEC	0		0		7.27
qnrS	5(12.19)	ETEC	5(55.55)	ETEC	0		18.2
qnrB + *qnrS*	2(4.87)	ETEC	0		0		3.64
qnrA + *qnrS*	1(2.43)	ETEC	0		0		1.8
Beta-lactamin resistance							
bla_{TEM}	2(4.87)	ETEC	0		1(20)	ETEC	5.45
bla_{SHV}	1(2.43)	ETEC	1(11.11)	ETEC	0		3.64
Beta-lactam + quinolone resistance							
bla_{SHV} +*qnrS*	2(4.87)	ETEC	0		0		3.64
bla_{SHV} + *qnrB*	2(4.87)	ETEC	0		0		3.64
bla_{TEM} +*qnrS*	2(4.87)	ETEC	1(11.11)	ETEC	1(20)	EAEC	7.27
bla_{TEM} + bla_{SHV}+ *qnrB*	1(2.43)	ETEC	0		0		1.8
bla_{TEM} + bla_{SHV} + *qnrS*	1(2.43)	ETEC	0		0		1.8
bla_{TEM} + bla_{SHV} + *qnrB* +*qnrS*	1(2.43)	ETEC	0		0		1.8

EAEC, Enteroaggregative *E. coli* ; ETEC, Enterotoxinogenic *E. coli*, STEC, Shiga-toxin producing *E. coli.*

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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