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Effect of environment on multidrug resistant (MDR) status of *Escherichia coli*

Okpokiri M.[1]*, Okerentugba P. O.[2], Agbagwa O. E.[2] and Otokunefor K.[2]

[1]Department of Microbiology, Rivers State University, Port Harcourt, Rivers State, Nigeria.
[2]Department of Microbiology, University of Port Harcourt, Port Harcourt, Rivers State, Nigeria.

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ABSTRACT

A suitable environment could be vital for the emergence and transfer of antibiotic-resistant *E. coli.* The effect of the environment on the multidrug-resistant status of *E. coli* was investigated in this study. Soil and faecal samples were collected from three environments: animal farms, hospitals and oil-contaminated soil. The *E. coli* isolates in the respective environments were isolated using standard microbiological methods. The antibiotic susceptibility pattern was determined using the Kirby-Bauer disc diffusion method. Results revealed a 50% prevalence of *E. coli* isolates from the animal farm environment samples, while the lowest prevalence of 22.7% was recorded in hospital environment samples. For the human faecal samples, a 34.8% prevalence was recorded in hospital and oil-contaminated environments, while the lowest prevalence of 30.4% was recorded in faecal samples collected from the animal farm environment. The *E. coli* isolates from animal farms were 100% resistant to ceftriaxone and Augmentin, while isolates from human faecal samples showed that isolates from hospital faecal samples were 100% resistant to imipenem and Ampiclox while 80% resistance was recorded for cefuroxime. The isolates displayed resistance to antibiotics belonging to the cephalosporin, aminoglycosides, quinolones and fluoroquinolones classes. The MAR index of the isolates in the hospital environment sample, oil-contaminated environment sample and oil-contaminated human sample were 0.4, in the hospital human sample the MAR was 0.5, in the animal farm environment sample the MAR index was 0.6, while a MAR index of 0.7 was recorded for the animal farm human samples. Although the isolates displayed very high antibiotic resistance, gentamycin and levofloxacin exhibited higher antibiotic potency and are recommended for the treatment of infections caused by *E. coli* from these environments. More so, the environment could be associated with the high resistance observed in the study.

Keywords: *E. coli*, multidrug resistant, environmental diversity.

*Corresponding author. Email: okpokiri.meka@ust.edu.ng.

INTRODUCTION

Antimicrobial resistance (AMR) is a multifaceted global health problem that increases hospitalisation time, treatment costs, morbidity and mortality (Iwu et al., 2022). Over the past half-century, the use of antimicrobials to treat infections in humans and animals has generated enormous antimicrobial pressure, not only on targeted pathogens but also on commensal bacteria (Szmolka and Nagy, 2013). In a previous study, inappropriate use of antibiotics by humans, factories, and farms, poor hygiene and sanitation, and inefficient prevention and control of infections in healthcare settings are considered important reasons for the emergence and distribution of antibiotic-resistant bacteria (Pormohammad et al., 2019).

Antimicrobial resistance genes (ARGs) are largely sourced from the environment and can be transferred to clinically significant pathogenic bacteria (Iwu et al., 2022). Furthermore, it has been increasingly acknowledged in recent years that the environment plays a significant role as a source and a channel for the dissemination of resistance (Bengtsson-Palme et al., 2018). The environment's diverse ecological niches offer a highly diversified gene pool that is unmatched by that of the

human and animal domains, making it an ideal environment for the uptake of novel resistance determinants (Schulz et al., 2017). The agricultural ecosystem is the most complex of all the environmental niches because of the different activities that affect the persistence and spread of AMR within the system (Mafiz et al., 2018). Multidrug-resistant (MDR) and extended-spectrum beta-lactamase (ESBL) producing *E. coli* is a significant example of an antibiotic resistance pathogen which can cause potentially fatal infections (Pormohammad et al., 2019).

According to Sahoo et al. (2012), the predominant facultative flora in the gastrointestinal tracts of both humans and animals is *E. coli*. Nonetheless, some strains of *E. coli* have acquired the capacity to infect the central nervous system, urinary tract, and gastrointestinal tract (Azimi et al., 2018; Gholizadeh et al., 2018). Antibiotic resistance develops when *E. coli* is exposed to antibiotics for an extended period (Reinthaler et al., 2013; Carattoli, 2008). Accordingly, *E. coli* and other antibiotic-resistant bacteria found in animals may be a reservoir for human colonisation and infection (Sahoo et al., 2012). A study conducted by Reinthaler et al. (2013) revealed that humans can potentially come into contact with drug-resistant *E. coli* through direct or indirect means, such as by consuming contaminated food or water. Although multidrug-resistant pathogenic strains of *E. coli* are important for spreading pathogens from the environment to food and water, there is a shortage of information about their presence in different environments within Port Harcourt. Therefore, the study aimed to assess the multidrug resistance of *E. coli* from different environmental samples.

MATERIALS AND METHOD

Sample size determination

The sample size for comparative study was determined using the following formula for comparison of two proportions (Charan and Biswas, 2013).

$$n = \frac{(Z^{*}+Z^{pwr})^2 \{P_1(100\%-P_1) + P_2(100\%-P_2)\}}{(P_1-P_2)^2}$$

n= Sample size

$$n= \frac{(1.96+0.84)^2\{23.3(100-23.3) +10.4(100-10.4)\}}{(23.3-10.4)^2}$$

n = 300 samples for human and environmental samples.

Sample collection

One hundred and fifty soil samples (50 each from the sample locations) were collected at a depth of 0-15 cm with the aid of an auger from the animal farm, hospital and oil-polluted soil environment. The composite soil sample was made up of three samples collected from three points for each environmental sample. A total of one hundred and fifty faecal specimens were collected from each environment (animal farm, hospital and oil-polluted soil). Each sample was collected into a labelled sterile container and transported to the Medical Microbiology Laboratory, University of Port Harcourt, Rivers State, in an iced-packed container.

Isolation of *E. coli*

This was done using the standard plate count (Prescott et al., 2011). In this method, tenfold serial dilution of Harrigan and McCanc as described by Wemedo et al. (2012), was carried out by transferring one gram of the soil sample into a test tube containing 10mL sterile normal saline. After this, a serial 10-fold dilution was made by transferring 1mL from the previous dilution into another test tube containing 9mL sterile saline. This was done until a dilution of 10^{-6} was reached. Similar preparation was done for all the soil samples. After dilution, 0.1mL of an appropriate dilution was transferred to the surface of the prepared McConkey agar (TM media, India) plates in duplicates. The aliquot was spread using a sterile bent glass rod and the plates were incubated at 44°C for 24-48 hours for the isolation *E. coli.* The faecal samples were prepared as described by Cheesbrough (2006) and inoculated on prepared McConkey agar plates in duplicates. Similar incubation temperatures and duration were used.

Confirmation of isolates

The plates were read after incubation and plates showing growth on the McConkey agar plates were isolated and subcultured on freshly prepared nutrient agar plates. The plates were incubated at 37°C for 24 hours. The pure cultures after incubation were gram-stained and viewed under microscopy to confirm if they were gram-negative bacterial isolates. After which, they were subjected to biochemical tests: indole, methyl red, Voges Proskauer, citrate utilization, sugar fermentation, gas production and motility tests were also carried out. All the tests were carried out as described in Prescott et al. (2011). More so, the pure isolates were stored frozen in bijou bottles containing 5mL sterile 10% glycerol (Amadi et al., 2014).

Antibiotic susceptibility testing

The antibiotic susceptibility testing was performed with 17 antibiotics discs using commercially prepared antibiotics

(Abtek) disc and it contained the following antibiotics; Gentamycin (10µg), Ciprofloxacin (5µg), Nitrofurantoin (30µg), Augmentin (30µg), Ofloxacin (5µg), Cefixime (5µg), Ceftazidime (30µg), Cefuroxime (10µg), Ceftriaxone (30µg), Cloxacillin (5µg) and Erythromycin (5µg). The antibiotic susceptibility of the *E. coli* isolates was performed as per the Kirby-Bauer disk diffusion method (Robinson et al., 2023). Briefly, a bacterial culture of 0.5 McFarland standard was prepared in normal saline. The bacterial lawn of standardized culture was prepared on the Muller Hinton agar plate. Plates were allowed to dry and antibiotic discs were placed on Mueller Hinton plates using sterile forceps. Plates were incubated at 37°C for 24 hours. The zone of inhibition around each antibiotic disc was measured and results were interpreted according to CLSI (2022).

Determination of multiple antibiotic resistance index

Multiple antibiotic resistances (MAR) index was ascertained for each isolate by using the formula:

MAR = a/b,

Where, a = the number of antibiotics to which the isolate depicted resistance and

b = the total number of antibiotics to which the test isolates has been evaluated for susceptibility (Sandhu et al., 2016).

Statistical analysis

Descriptive statistics was employed to determine the percentages of the antibiotic responses (susceptibility, intermediate and resistant) and the prevalence of *E. coli* in the environments. All analysis was carried out using the statistical package for social science (SPSS v27).

RESULTS

The prevalence of *E. coli* isolates from the different environment samples showed that the highest prevalence (50%) was recorded in animal farm environment samples, followed by oil-contaminated environment soil (27.3%), and the least prevalence of 22.7% was recorded in hospital environment samples (Figure 1).

The highest *E. coli* prevalence (34.8%) was recorded in faecal samples collected from hospital and oil-contaminated environments, and the least prevalence of 30.4% was recorded in faecal samples collected from animal farm environment (Figure 2).

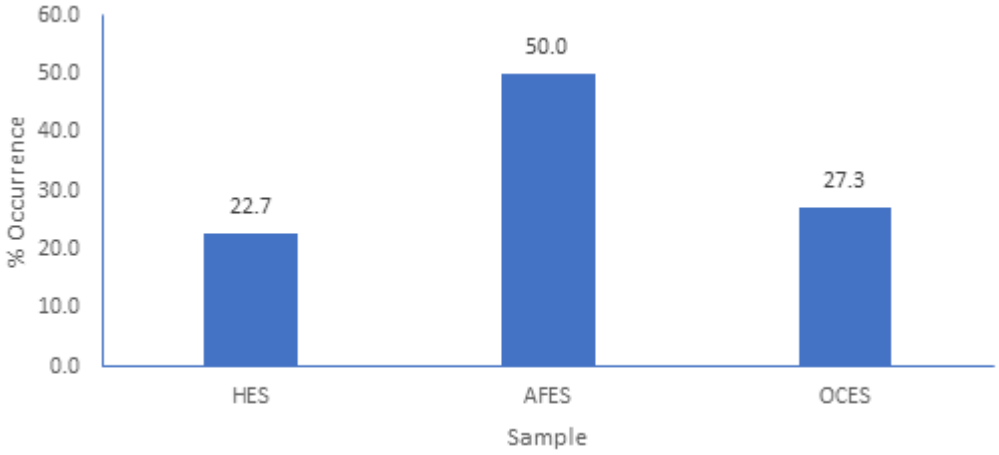

Figure 1. Prevalence of *E. coli* across the environmental samples.
Key: HES - Hospital Environmental Samples, AFES - Animal Farm Environmental Samples, OCES – Oil-Contaminated Environmental Samples.

The antibiotics susceptibility pattern of *E. coli* isolates from the soil of the different environmental samples showed that the isolates from animal farms were 100% resistant to ceftriaxone and augmentin, and resistance to cefixime, cefuroxime and nitrofurantoin was 80%. For gentamycin, ofloxacin and nalidixic acid, a resistance of 20% was recorded. All the isolates from the animal farm were 100% susceptible to Levofloxacin, and 80% susceptibility to ofloxacin and gentamycin were recorded (Table 1). The isolates from the hospital soil samples were 100% resistant to Ampliclox. The percentage resistance to ofloxacin, cefotaxime, and cefuroxime was

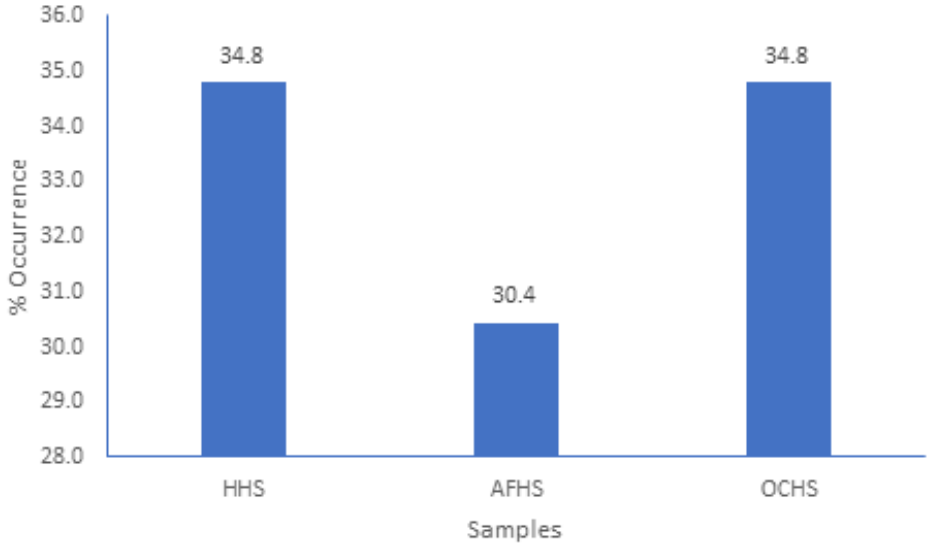

Figure 2. Prevalence of *E. coli* across the human samples.
Keys: HHS - Hospital Human Samples, AFHS - Animal Farm Human Samples, OCHS – Oil-Contaminated Human Samples.

Table 1. Antibiotic susceptibility of *E. coli* from different environmental samples.

Antibiotics	Animal farm			Hospital sample			Oil-contaminated sample		
	R [n (%)]	I [n (%)]	S [n (%)]	R [n (%)]	I [n (%)]	S [n (%)]	R [n (%)]	I [n (%)]	S [n (%)]
Cefixime (10µg)	4(80)	0(0.00)	1(20)	1(20)	0(0.00)	4(80)	3(60)	0(0.00)	2(40)
Ofloxacin (10µg)	1(20)	0(0.00)	4(80)	3(60)	0(0.00)	2(40)	1(20)	0(0.00)	4(80)
Imipenem (300µg)	2(40)	2(40)	1(20)	4(80)	0(0.00)	1(20)	4(80)	0(0.00)	1(20)
Cefotaxime (5µg)	3(60)	1(20)	1(20)	3(60)	0(0.00)	2(40)	1(20)	0(0.00)	4(80)
Ampiclox (5µg)	3(60)	1(20)	1(20)	5(100)	0(0.00)	0(0.00)	1(20)	0(0.00)	4(80)
Cefuroxime (30µg)	4(80)	0(0.00)	1(20)	3(60)	0(0.00)	2(40)	2(40)	0(0.00)	3(60)
Ceftriaxone (30µg)	5(100)	0(0.00)	0(0.00)	1(20)	0(0.00)	4(80)	2(40)	0(0.00)	3(60)
Nalixidic acid (30µg)	1(20)	3(60)	1(20)	2(40)	0(0.00)	3(60)	4(80)	0(0.00)	1(20)
Levofloxacin (30µg)	0(0.00)	0(0.00)	5(100)	2(40)	0(0.00)	3(60)	1(20)	0(0.00)	4(80)
Nitrofurantoin (30µg)	4(80)	0(0.00)	1(20)	2(40)	0(0.00)	3(60)	3(60)	0(0.00)	2(40)
Augmentin (300µg)	5(100)	0(0.00)	0(0.00)	2(40)	0(0.00)	3(60)	4(80)	0(0.00)	1(20)
Gentamycin (10µg)	1(20)	0(0.00)	4(80)	1(20)	0(0.00)	4(80)	1(20)	1(20)	3(60)

Keys: n = number of isolates; R = resistant; S = susceptible; I = intermediate.

60%, while resistance to imipenem was 80%. The susceptibility of the isolates to cefixime, ceftriaxone and gentamycin was 80%, and lower susceptibility of 40, 20 and 40% was recorded for ofloxacin, imipenem and cefotaxime, respectively. Susceptibility of 60% was recorded for nalidixic acid, levofloxacin, nitrofurantoin and augmentin. On the other hand, the resistance of the *E. coli* isolates from the oil-contaminated soil to augmentin and imipenem was 80% while 60% resistance was recorded for cefixime and nitrofurantoin. A low resistance of 20% was recorded for ofloxacin, cefotaxime, ampiclox, levofloxacin and gentamycin, respectively. Thus, the isolates from this environment showed a high susceptibility of 80% to ofloxacin, cefotaxime, ampiclox, levofloxacin and gentamycin, respectively (Table 1).

The antibiotic susceptibility pattern of *E. coli* isolates from human faecal samples in the respective environment showed that isolates from hospital faecal samples were 100% resistant to imipenem and ampiclox, and 80% resistance was recorded for cefuroxime. A percentage resistance of 40% was recorded for ofloxacin and cefotaxime. The isolates were seen to be 100% susceptible to gentamycin, while susceptibility of 80% was recorded for cefixime, nalixidic acid, levofloxacin and nitrofurantoin. The percentage resistance of the isolates from the oil-contaminated human samples showed that the isolates were 100% resistant to nalidixic acid, and 80% resistance was recorded for ofloxacin, imipenem, and augmentin, respectively. None of the isolates from this location was resistant to gentamycin but an intermediate response of 20% was recorded while 80% susceptibility was recorded for gentamycin, nitrofurantoin, levofloxacin, ceftriaxone, cefuroxime, ampiclox and cefotaxime, respectively. More so, the isolates of *E. coli* from animal farm human samples were 100% resistant to cefixime, cefuroxime, ceftriaxone, and augmentin, and 60% resistance was recorded for imipenem and cefotaxime. The resistance of the isolates to ampicillin and nitrofurantoin was 80%, respectively. The isolates were 100% susceptible to ofloxacin, levofloxacin and gentamycin, respectively (Table 2).

The multiple antibiotic-resistant indices (MARI) of the *E. coli* isolates from the respective samples showed that it was above 0.2 (Figure 3).

The multidrug resistance of *E. coli* from different locations to the classes of antibiotics showed that all the isolates from the respective samples exhibited multidrug resistance (MDR) (Table 3).

Table 2. Susceptibility pattern of *E. coli* from human samples.

Antibiotics	Hospital human samples			Oil-contaminated human samples			Animal farm human samples		
	R [n (%)]	I [n (%)]	S [n (%)]	R [n (%)]	I [n (%)]	S [n (%)]	R [n (%)]	I [n (%)]	S [n (%)]
Cefixime (10µg)	1(20)	0(0.00)	4(80)	3(60)	0(0.00)	2(40)	5(100)	0(0.00)	0(0.00)
Ofloxacin (10µg)	2(40)	0(0.00)	3(60)	4(80)	0(0.00)	1(20)	0(0.00)	0(0.00)	5(100)
Imipenem (30µg)	5(100)	0(0.00)	0(0.00)	4(80)	0(0.00)	1(20)	3(60)	2(40)	0(0.00)
Cefotaxime (5µg)	2(40)	0(0.00)	3(60)	1(20)	0(0.00)	4(80)	3(60)	2(40)	0(0.00)
Ampliclox (5µg)	5(100)	0(0.00)	0(0.00)	1(20)	0(0.00)	4(80)	4(80)	0(0.00)	1(20)
Cefuroxime (30µg)	4(80)	0(0.00)	1(20)	1(20)	0(0.00)	4(80)	5(100)	0(0.00)	0(0.00)
Ceftriaxone (30µg)	1(20)	1(20)	3(60)	1(20)	0(0.00)	4(80)	5(100)	0(0.00)	0(0.00)
Nalidixic Acid (30µg)	1(20)	0(0.00)	4(80)	5(100)	0(0.00)	0(0.00)	1(20)	2(80)	0(0.00)
Levofloxacin (30µg)	1(20)	0(0.00)	4(80)	1(20)	0(0.00)	4(80)	0(0.00)	0(0.00)	5(100)
Nitrofurantoin (30µg)	1(20)	0(0.00)	4(80)	1(20)	0(0.00)	4(80)	4(80)	0(0.00)	1(20)
Augmentin (30µg)	3(60)	0(0.00)	2(40)	4(80)	0(0.00)	1(20)	5(100)	0(0.00)	0(0.00)
Gentamicin (10µg)	0(0.00)	0(0.00)	5(100)	0(0.00)	1(20)	4(80)	0(0.00)	0(0.00)	5(100)

Keys: n = number of isolates; R = resistant; S = Susceptible; I = intermediate .

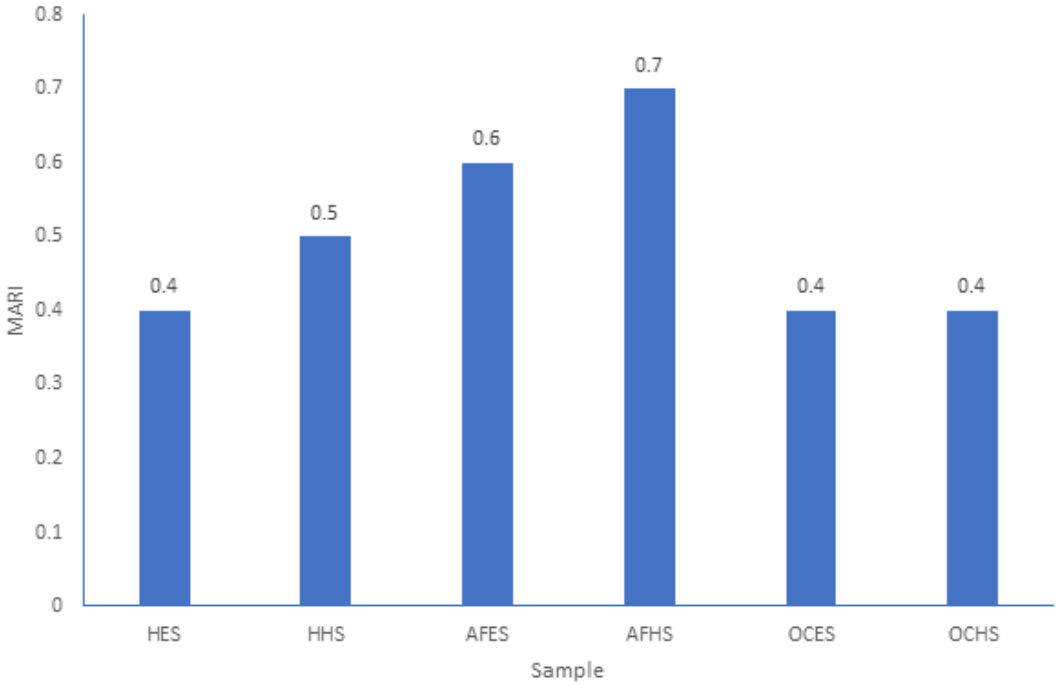

Figure 3. MAR indices of *E. coli* from the different locations.
Keys: HES - Hospital Environmental Samples, HHS - Hospital Human Samples, AFES - Animal Farm Environmental Samples, AFHS - Animal Farm Human Samples, OCES - Oil-Contaminated Environmental Samples, OCHS - Oil Contaminated Human Samples.

Table 3: Multidrug resistance of *E. coli* to different antibiotics.

S/N	Antibiogram	Isolate code	No. of isolates	No. of antibiotics classes	Class of antibiotics
1	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	HES	10	2	Fluoroquinolone and Carbepenem
2	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	HHS	20	3	Carbapenem, Penicillin and Aminoglycoside
3	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	AFES	16	5	Cephalosporin, Carbapenem, Penicillin, Quinolone and Nitrofuran
4	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	AFHS	7.5	5	Cephalosporin, Carbapenem, Penicillin, Quinolone and Nitrofuran
5	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	OCES	15	2	Carbapenem and Quinolone,
6	ZEM-OFX-CTX-ACX-CXM-CRO-NA-LB-AUG-GN	OCHS	16	2	Carbapenem and Quinolone

Keys: CEFEXIME (ZEM), OFLOXAXIN (OFX), IMIPENEM (IMP), CEFOTAXIM (CTX), AMPICLOX (ACX), CEFUROXIME (CXM), CEFTRIAZONE (CRO), NALIDIXIC ACID (NA), LEVOFLOXACIN (LBC), NITROFURATOIN (NF), AUGMENTIN (AUG), GENTAMYCIN (GN).

DISCUSSION

The effect of the environment on multidrug-resistant *E. coli* was investigated. The disparity in the distribution or prevalence of *E. coli* in the respective samples could be attributed to the nature of the sample, the number of *E. coli* isolates present in the sample, and the prevailing factors enhancing the availability of the isolates (Robinson et al., 2023). In the present study, the prevalence of *E. coli* was higher in the environmental samples characterised by animals (animal farm environment) as well as faecal samples obtained from hospitals, which is indicative of the characteristics of *E. coli. E. coli* is known as an indicator microorganism mostly found in the large intestines of humans and animals (Prescott et al., 2011). Thus, there is a high prevalence in animal farm environments and faecal samples collected from humans in the hospital. It is well-documented that anthropogenic activities and geographical differences could influence the prevalence of microorganisms (Iwu et al., 2022).

The present study revealed high antibiotic resistance in the *E. coli* isolates from the respective environments. High multidrug resistance was also observed. The environment largely plays a vital role in the distribution of antibiotic-resistant isolates. The selective pressure of antibiotics at low concentrations is amplified in certain circumstances, and this would enhance the competition of antibiotic-resistant strains and increase their occurrence and risk of antibiotic resistance spread in natural environments (Na et al., 2018). This agreed with Iwu et al. (2022), who opined that the advent of pathogenic bacteria that are resistant to antibiotics has made it necessary to continuously look into their presence in the environment and how they might infect people. In the present study, the MAR indices of *E. coli* isolates in the hospital environment sample, oil-contaminated environment sample and oil-contaminated human sample was 0.4, in the hospital human sample the MAR was 0.5, in the animal farm environmental sample MAR index was 0.6 while a MAR index of 0.7 was recorded for the animal farm human samples. Thus, high MAR indices were recorded in the environmental and animal farms compared to the MAR index from the hospital human sample. This agreed with Pormohammad et al. (2019), who reported a higher prevalence of multidrug-resistant *E. coli* strains from animal and environmental sources than humans. More so, the resistant patterns of the *E. coli* isolates in the different environments and samples were somewhat similar especially as they shared similar resistance to similar antibiotics. This similarity could be due to interactions or cross-contamination between the environment and humans. A fascinating case study involving MDR commensal *E. coli* from cohabiting humans, pets, and their homes revealed that all cohabiting species, including humans, had direct interactions and cross-contamination patterns (Szmolka and Nagy, 2013). The *E. coli* isolates in the present study were highly resistant to ampicillin, ofloxacin, nitrofurantoin and augmentin. Except for the isolates from hospital human samples which showed 100% susceptibility to gentamycin, gentamycin resistance was recorded in the isolates from the other samples. This agreed with a previous study which showed 71.67% resistance of *E. coli* isolates to ampicillin and 88.3% susceptibility to gentamicin (88.33%) (Adzitey et al., 2020). It has been documented that a high-risk source of contamination where multiple antibiotics or growth promoters are used is indicated by MAR index values > 0.2, whereas values < 0.2 indicate bacteria from the source that uses fewer antibiotics (Adzitey et al., 2020). Thus, the high MAR indices in the present study signified high-risk sources and overuse of antibiotics.

The increased antibiotic resistance exhibited by the *E. coli* isolates in the present study could be attributed to the transfer of antibiotic-resistant genes from the environment especially due to horizontal gene transfer (Prescott et al., 2011) to the isolates and when resistant isolates come in contact with food and water, it is imminent that they could be consumed by man. It has been suggested that the presence of other environmental and public health relevant antimicrobial resistant bacteria as well as clinically relevant antibiotic-resistant genes (ARGs), may be indicated by the occurrence of antimicrobial-resistant *E. coli* in the environment (Gekenidis et al., 2018). More so, the high resistance to quinolones and fluoroquinolones could be attributed to the extended use of antibiotics from this class in agriculture and to treat poultry infections (Szmolka and Nagy, 2013).

CONCLUSION

The study revealed the presence of high levels of antibiotic-resistant *E. coli* in different environments and samples indicating that environment could play a major role in antibiotic resistance. More so, the study revealed that the MAR index was higher in the *E. coli* isolates from animal farm human samples. Also, the high MAR indices in the different environments could be an indication of a possible danger of multidrug-resistant *E. coli* isolates especially if food crops grown in these environments are consumed. Gentamycin and levofloxacin are recommended for use in the treatment of infections caused by *E. coli* from these environments. The need for screening antibiotic-resistant genes in these environments is recommended for future studies.

REFERENCES

Adzitey F, Assoah-Peprah P, Teye GA, Somboro AM, Kumalo HM, Amoako DG, **2020**. Prevalence and antimicrobial resistance of

Escherichia coli isolated from various meat types in the tamale metropolis of Ghana. Int J Food Sci, 2–7.
Azimi T, Nasiri MJ, Chirani AS, Pouriran R, Dabiri H, **2018**. The role of bacteria in the inflammatory bowel disease development: a narrative review. APMIS, 126(4): 275–283.
Bengtsson-Palme J, Kristiansson E, Larsson DGJ, **2018**. Environmental factors influencing the development and spread of antibiotic resistance. FEMS Microbiol Rev, 42(1): 68–80.
Carattoli A, **2008**. Animal reservoirs for extended spectrum β-lactamase producers. Clin Microbiol Infection, 14: 117–123.
Gekenidis MT, Qi W, Hummerjohann J, Zbinden R, Walsh F, Drissner D, **2018**. Antibiotic-resistant indicator bacteria in irrigation water: High prevalence of extended-spectrum beta-lactamase (ESBL)-producing *Escherichia coli*. PLoS One, 13, e0207857.
Gholizadeh P, Mahallei M, Pormohammad A, **2018**. Microbial balance in the intestinal normal microbiome and its association with diabetes, obesity and allergic disease. Microb Pathog, 127: 48-55.
Iwu CD, Kayode AJ, Igere BE, Okoh AI, **2022**. High levels of multi drug resistant Escherichia coli pathovars in preharvest environmental samples: A ticking time bomb for fresh produce related disease outbreak. Front Environ Sci*,* 10(March), 1–15.
Mafiz AI, Perera LN, He Y, Zhang W, Xiao S, Hao W, **2018**. Case Study on the Soil Antibiotic Resistome in an Urban Community Garden. Int J Antimicrob Agents, 52: 241–250.
Na G, Lu Z, Gao H, Zhang L, Li Q, Li R, Yang F, Huo C, Yao Z, **2018**. The effect of environmental factors and migration dynamics on the prevalence of antibiotic-resistant *Escherichia coli* in estuary environments. Sci Rep*,* 8(1): 1–10.
Pormohammad A, Nasiri MJ, Azimi T, **2019**. Prevalence of antibiotic resistance in escherichia coli strains simultaneously isolated from humans, animals, food, and the environment: A systematic review and meta-analysis. Infect Drug Resist, 12, 1181–1197.
Pormohammad A, Pouriran R, Azimi H, Goudarzi M, **2019**. Prevalence of integron classes in gram-negative clinical isolated bacteria in Iran: a systematic review and meta-analysis. Iran J Basic Med Sci*,* 22(2): 118–127.
Reinthaler FF, Galler H, Feierl G, **2013**. Resistance patterns of Escherichia coli isolated from sewage sludge in comparison with those isolated from human patients in 2000 and 2009. J Water Health, 11(1): 13–20.
Robinson VK, Wemedo SA, Akani NP, Amadi LO, **2023**. Prevalence and Antibiotic Susceptibility of Methicillin-Resistant Staphylococcus aureus isolated from Clinical Specimens from a Teaching Hospital in Port Harcourt. Int J Microbiol Appl Sci, 2(2): 31–36.
Sahoo KC, Tamhankar AJ, Sahoo S, Sahu PS, Klintz SR, Lundborg CS, **2012**. Geographical variation in antibiotic-resistant *Escherichia coli* isolates from stool, cow-dung and drinking water. Int J Environ Res Public Health, 9(3): 746–759.
Sandhu R, Dahiya S, Sayal P, **2016**. Evaluation of Multiple Antibiotic Resistance Index and Doxycycline Susceptibility of *Acinetobacter* species among inpatients. Indian J Res, 3(3): 299 – 304.
Schulz F, Eloe-Fadrosh EA, Bowers RM, Jarett J, Nielsen T, Ivanova NN, **2017**. Towards a balanced view of the bacterial tree of life. Microbiome, 5: 140.
Szmolka A, **Nagy** B, **2013**. Multidrug-resistant commensal Escherichia coli in animals and its impact for public health. Front Microbiol, 4(SEP): 1–13.

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