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Systematic Review

Antibacterial Resistance Genes Frequently Detected in Nigeria

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ABSTRACT

The emergence and reemergence of antibacterial resistance has made it more difficult for a choice of effective antibiotics that treat bacterial infections. Bacteria that are resistant to antimicrobial agents are usually spread from person to person, or from non-human sources in the environment. These non-human sources could aid in the spread of resistant genes as well as resistant pathogens. Systematic review was conducted in accordance to the guideline of PRISMA, 55 relevant literatures from the 152 searched were identified from PubMed, Google Scholar and AJOL using related specific search terms. Only studies carried out or related to Nigeria from January, 2000 to August, 2022. All data obtained from the reviewed articles were presented in percentages using MS Excel 2013. The most prevalent antibacterial genes were blaCTX-M-15 and blaTEM with the prevalence of 2.77%; sul1 and tetA with the prevalence rate of 2.31%; tetM with the prevalence rate of 2.08%; and blaCTX-M, blaSHV, mecA, sul2 and tetB having a prevalence of 1.85%. The sources from which these resistance genes were identified are humans (55.7%) and non-humans (44.3%). The most prevalent bacteria harboring these resistant genes were Escherichia coli (13.2%), Staphylococcus aureus (5.66%) and Klebsiella pneumoniae (5.03%). Antibiotics resistance has continued to threaten the success of modern medicine, with more resistance strains emerging constantly. The wide distribution of antibacterial resistance genes in non-human sources makes them reservoirs of these resistant genes which have the potential of being transferred from commensal microorganisms to human pathogens.

Keywords: *Resistance, Antibacterial, Genes and Nigeria.*

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INTRODUCTION

Diseases or infections caused by bacteria are treated by antibiotics. These antibiotics work by disrupting one of the processes important for the survival of invading bacteria, such as the formation or integrity of the cell wall (McGeer *et al*., 2001). Generally, the major risk factor for a rise in bacterial resistance is an increase in antibiotics usage. So, resistance development in bacteria to some antibiotics has led to policies on antimicrobial usage in veterinary medicine, nutrition and agriculture (Caprioli *et al*., 2000). Some do not use the antibiotics for prevention and treatment of infections caused by bacteria but also as promoters of growth (Omoya and Ajayi, 2016). Resistance is considered as the ability of microbes to withstand the effects of an antimicrobial agent. A major factor that promotes the spread of resistance is when antibiotics are used inappropriately and irrationally (Ndihokubwayo *et al*., 2013). A phenomenon where microbes acquire genes that enable them to withstand the effects of antimicrobial agent is referred to as antibiotic resistance. Antimicrobial resistant bacteria are usually spread from person to person, or from non-human sources in the

environment. These non-human sources could aid in the spread of resistant as well as pathogens within hospitals (Odetoyin *et al*., 2020). Antibiotic resistance creates a set of specific challenges for clinical, therapeutic and public health interventions with local, national and global dimensions, thereby threatens the success of medical interventions at all levels of health care (WHO, 2001). Many antimicrobial drugs are losing their ability to treat infectious diseases as a result of resistance. Antimicrobial agent resistance typically occurs as a result of four (4) main mechanisms namely alteration of target-sites, enzymatic inactivation of the drug, extrusion by efflux and reduced cellular uptake (Smith, 2004).

Bacteria resistance to antibiotics bacteria is a global problem (Okoli, 2004). Rate at which resistance among bacterial populations has been reported to be contingent on the external use of some antibiotics in particular and is high in a particular environment. Use of drugs, appropriately, judiciously and rationally in veterinary medicine generally requires that applicable to their clinical needs, patients receive medicines for an adequate period of time, in doses that meet their own individual requirements, and at the lowest cost to the client (WHO, 2007). However, twenty (20) to fifty (50) % of the total antibacterial medicines used in human beings and animals have been reported to be inappropriate (Cizman, 2003). This is one of the major risk factors of the rising prevalence resistance among bacteria to antimicrobial agents in both humans and animals.

World Health Organization (WHO) has identified bacterial resistance to antibacterial agents as a major threat to humans and animals, due to lack of new antibacterial agents in the development pipeline, and infections caused by multi-drug resistant pathogens are becoming untreatable (Goossens, 2011; Carlet *et al*., 2011). Therefore, this has led to the need for antimicrobial agents' stewardship, especially in veterinary medicine (Caprioli *et al*., 2000). Therefore, investigation of the resistance genes is important, especially in developing countries such as Nigeria, where there is high incidence of inappropriate use of antibiotics (Egwuenu *et al*., 2017).

The use of Antibiotics animals at a sub therapeutic dose, either for therapy, prophylactic or growth promotion purposes can lead to transfer of resistant genes from animals to humans and thereby establishing a reservoir of microbes that are resistant (Angulo *et al*., 2004; Maripandi and Al-Salamah, 2010). Contamination of aquaculture products can occur from potential biological agents such as bacteria, viruses, parasites and biotoxins (Huss *et al*., 2003; WHO, 1999). Harbouring of these resistant genes reflects antibiotic use in their areas of origin (Okoli *et al*., 2005).

Two (2) stages in the emergence of resistant bacterial strains to antibiotics are Gene acquisition or genetic mutation and Selective pressure. Mutation(s) in the DNA sequence of the relevant gene(s) in the bacterial chromosome can lead to resistance, or because of the transfer of the existing antibiotic resistance gene into the bacterium from another resistant bacterium (horizontal gene transfer or gene acquisition). Once mutation or gene responsible for resistance is present (expressed), the cells harboring it can grow in the presence of the antibiotics, and therefore increase in numbers at the expense of cells that are susceptible. Resistant organisms are naturally favoured.

Continuous exposure to an antibiotic provides the strongest selection pressure and the total amount of antibiotic used is a general indicator of the selection pressure and (Taiwo, 2011;
Wernli *et al*., 2011).

Transfer of resistant bacteria can occur from one environment to another (e.g. animal to human or from human to animal). Direct contact is the process through which such spread occur (for example, between human and animal), it can also occur indirectly (for instance in water or food). The spread of resistant microorganisms is well documented globally, and presumably this could be due to movement of hosts or contaminated products between locations (including between continents) (Wernli *et al*., 2011).
Mutations in the bacterial genome can lead to resistance and this is spread by transmission of the bacterium; whereas horizontal gene transfer allows for resistance to be spread between commensal and pathogenic bacteria and vice versa, and also between diverse bacterial species. The most frequent mechanism underpinning antibacterial resistance is horizontal transfer of gene between a bacterium that is resistant and the one that is susceptible, this happens in the absence of selection (Wernli *et al*., 2011).

There is scarcity of published information on antibacterial resistant genes in Nigeria. This review will provide an idea of the commonly detected resistant genes among bacteria from both human and non-human sources. This review aims to identify antibacterial resistance genes frequently detected in Nigeria from January, 2000 to August, 2022.

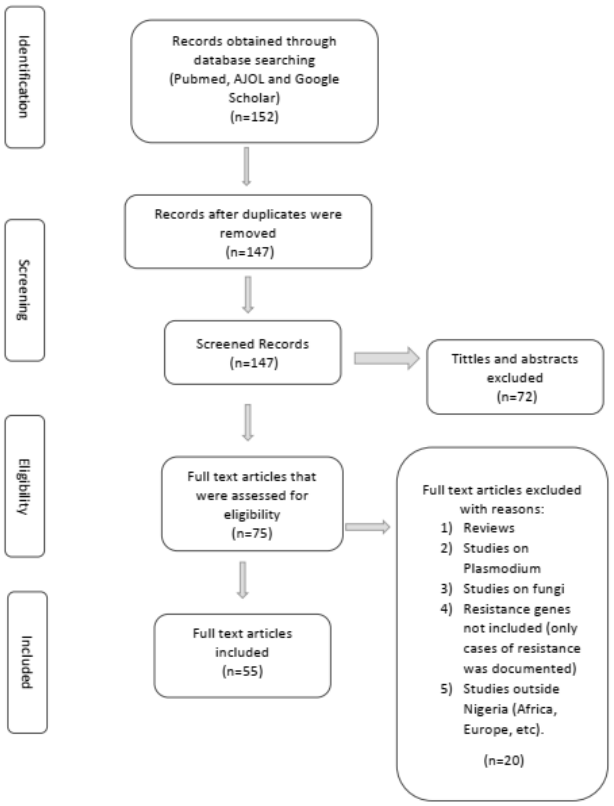

Figure. 1:
Selection Process of the Review (Adapted from Bernabé *et al*., 2017)

MATERIALS AND METHODS

The systematic review was conducted in accordance to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analysis) 2020 guidelines (Matthew *et al*., 2021). Relevant literatures will be identified from PubMed, Google Scholar, and AJOL databases using specific search terms of antibacterial resistance genes frequently detected in Nigeria.

Inclusion and exclusion criteria: Published literatures carried out in Nigeria from January, 2000 to August, 2022 that included antibacterial resistant genes in humans and non-human sources in Nigeria will be included in this study. Only publications in English language will be included in this review. Only free articles (or free access articles) will be included in these studies. Studies on resistant genes in fungi, viruses, parasites, etc. will be excluded from these studies. Also, studies reporting resistant genes outside the scope of the review will be excluded as well.

Data Analysis: All data obtained from the reviewed articles will be presented in percentages using MS Excel 2013.

RESULTS AND DISCUSSION

Antibiotics resistance has continued to threaten the success of modern medicine, with more resistance strains emerging constantly. This resistance has made it more difficult for a choice of effective antibiotics that can treat those infections that are caused by bacteria. The emergence and reemergence of antibiotic resistance has breached the last line of antibiotics, according to WHO we may be out of option in 2050 if alternative measures are not taken (WHO, 2017).

The most prevalent antibacterial genes were blaCTX-M-15 and blaTEM with the prevalence of 2.77%; sul1 and tetA with the prevalence rate of 2.31%; tetM with the prevalence rate of 2.08%; and blaCTX-M, blaSHV, mecA, sul2 and tetB having a prevalence of 1.85%. These genes encode resistance for Beta-lactam antibiotics, sulfonamide, methicillin and tetracycline. The beta lactam antibiotics resistance genes (RG) were the most isolated in Nigeria. In agreement to the prevalence of blaCTX-M-15, Awosile *et al*. (2022), in their review on beta-lactamase resistant genes in Enterobacteriaceae reported blaCTX-M-15 gene in Nigeria to be 46%. In agreement to the resistance genes identified in this study though with differences in the prevalence rate, Escher *et al*. (2021) studied antimicrobial resistant genes in Africa, and the common resistance genes identified include sul1 (36.2%), sul2 (32.0%), tetA (42.0%), strB (34.9%), blaTEM (28.8%); and sul1 (27.8%) were most prevalent in humans. However, in contrast with the findings in this study, Mengistu *et al*. (2022) reported tetM (20.6%), blaCMY-2 (13%), ermB (6.1%), blaCMY-1 (4.6%), blaCTX-M-15 (3.1%) and mcr-4 (0.8%). Also, Capkin *et al*. (2015) in Turkey reported ampC as the most frequent resistant genes to antibiotics isolated, followed by tetA, sul2, blaCTX-M1, and blaTEM in coliform bacteria. Odoi *et al*. (2021) reported that β-lactamase encoding genes (blaSHV,blaTEM, blaCTX-M, blaVIM, and blaIMP) were not detected; only AmpC inducible cephalosporinases (50%) was observed in the multidrug resistant strains in their study. The study by Abdolmaleki *et al*. (2019) on hospital cockroaches was not in agreement with this study, they reported that BlaZ, aacA D, tetK, msrA, dfrA, ermA, gyrA, grlA and rpoB were the most frequently detected resistance genes to antibiotics amongst the Methicillin resistant Staphylococcus aureus strains. Differences in those genes reported and their prevalence could be due to variation in study location and period under review.

Based on our findings, the sources from which these resistance genes were identified are humans (55.7%) and non-humans (44.3%). The presence of the resistant genes in the non-human sources could lead to transfer of these genes to humans. This is a great challenge as they may serve as reservoirs for these resistance genes.

Our findings showed that the most prevalent bacteria harboring these resistant genes were Escherichia coli (13.2%), Staphylococcus aureus (5.66%) and Klebsiella pneumoniae (5.03%). In a study carried out in Bangkok, Thailand, resistant bacteria to antibiotics bacteria were screened for β-lactamase relating genes, such as AmpC (MOX and ACC genes), blaCTX-M, and Int1 genes. Bacteria resistant to antibiotics were A. caviae, P. vulgaris, Enterobacter Aerogenes, and K. pneumoniae. A. caviae, P. penneri,K. Pneumoniae, and A. hydrophilla were positive for MOX gene; blaCTX-M, and Int1 genes; ACC and Int1 genes; and ACC gene, respectively (Thongkao and Sudjaroen, 2019). This is in contrast to the result obtained in this review.

The mechanisms of resistance as identified by some of the authors from our findings were efflux pump upregulation, mutation, ribosomal protection proteins, Possession of virulence genes, possession of inactivating enzymes, detection folate pathway antagonists, carbapenemase production and reduced production of outer membrane porins. Lamikanra *et al*. (2011) used quantitative real-time PCR (qPCR) to measure transcription of 13 genes representing the major known efflux systems in Escherichia coli. Compared to E. coli K-12 strain MG1655, sixteen isolates indicated a twofold or greater up-regulation in transcription of the outer-membrane extrusion factor gene tolC; Ugwu *et al*. (2015) observed Ser84Leu mutation within the QRDR of the gyrA protein, with 3 isolates showing 2 extra substitutions, Ser98Ile and Arg100Lys (one strain) and Glu88Asp and Asp96Thr. Their study points out the possibility of transfer of mecA and other AMR genes from MRCoNS to bacteria that are pathogenic, which is a serious public health and veterinary concern. Dinic *et al*. (2012), observed that mutations on codon 530–533(wt8) and mut1 D516V were not confirmed by sequencing; however, the lack of a wt4 band was confirmed (D516A), and an additional mutation L511P (wt2) was found. Adesoji *et al*. (2015) also reported efflux pump mechanism of resistance. Ogbolu *et al*. (2013), observed that DHA-like enzymes were present predominantly in the resistant strains. The strains were found to possess plasmids ranging from < 20 to > 200 kb as observed by plasmid extraction and electrophoresis. Transformation experiments revealed that the plasmids once transferred coded for beta-lactam resistance. Beta-lactamase transformants showed significantly increased resistance to amoxicillin and ceftazidime.

Table 1:
Commonly reported antibacterial resistance genes in human and non-human samples

S/N	Resistant Genes Type	Molecular mechanism of resistance	Bacteria	Specimen	Remarks	Reference
1	*tetM, gyrA, ParC, ermB, ermTR, lnu, mef, and aac6'-aph2*	Possession of MLSB genes, lincosamide nucleotidyltransferase, macrolide efflux, CAT genes, bifunctional aminoglycoside-inactivating enzyme with 6'-acetyltransferase and 2''-phosphotransferase activities.	*Streptococcus agalactiae,*	Vaginal and rectal swab	Human sample	(Bob-Manuel *et al*., 2021)
2	*blaTEM, blaCTX-M* and *blaSHV*	-	*Klebsiella pneumoniae, Escherichia coli, Enterobacter* spp, *Serratia* spp, *Pantoea* spp and *Citrobacter* spp	Blood culture from children <5years	Human Sample	(Duru *et al*., 2020)
3	*tet39*	-	*B. cereus, C. cellulans, Alc. faecalis, St. maltophilia, Ent. hormachei, Brev. diminuta, P. rettgeri* and *L. sphaericus.*	Poultry waste polluted river	Non-human sample	(Adelowo and Fagade, 2009)
4	*mcr-1*	-	*Escherichia coli* and *Klebsiella pneumoniae.*	Faecal and cloacal swab samples	Non-human sample	(Anyanwu *et al*., 2021)
5	*mecA, aacA-aphD, tetK, tetM, ermA* and *msrA.*	Possession of virulent genes	*S. aureus*	Urine, wound swab, semen and throat swab, etc.	Human sample	(Shittu *et al*., 2011)
6	*blaCTX-M, blaTEM, blaSHV, blaGES, blaVEB, blaNDM, blaGOB, blaLRA, blaB, blaCMY, blaLEN, blaOKP, blaPDC, blaADC, blaOXA; mcr-3, mcr-1, mcr-4, mcr-5, mcr-2, mcr-8, mcr-6, mcr-7; mcr-9, cat, cml, cmr, cpt, dha1; fexA, floR, pexA, cmlV, rph, rphD, rif, iri, arr2, arr4, arr5, arr7; fosA, fosB, fosC, fosD, fosE, fosF, fosG, fosH, fosX; fomA, fomB, fomC; otrA, tetM, tetO, tetS, tetT, tetW, tet(32), tet(36), tet(44), tetQ, tet(34), tet(37), tet(47), tet(48), tet(49), tet(50), tet(51), tet(52), tet(53), tet(54), tetX), otrB, otrC, tetA, tetB, tetAB, tetC, tetD, tetE, tetG, tetH, tetJ, tetL, tetR, tcr-3, tet(30), tet(31), tet(33), tet(35), tet(38), tet(39), tet(40), tet(41), tet(42), tet(43), tet(45), tet(57), tet(58), tet(59), tet(60), tetU, tetV, tetY, tetZ; qnrB, qnrC, qnrD, qnrS, qnrVC; vanR, vanS.*	Antibiotic efflux, Antibiotic targ*et al*teration, Antibiotic target replacement.	*Bifidobacterium adolescentis,* S*treptomyces rishiriensis, Escherichia coli* and *Corynebacterium striatum.*	chronically polluted soil	Non-human sample	(Salam, 2020)
7	*aac-6-Ib-cr, blaCTX-M-15* and *oqxAB.*	Plasmid carries replication-associated proteins linked with *IncFIA, IncFIB, IncFIC* and *IncFII.*	*E. coli* and *Escherichia fergusonnii*	Fecal sample	Human sample	(Monárrez *et al*., 2019)
8	*aac (6') – I* and *ant (2") – I.*	-	*Pseudomonas aeruginosa*	urine, wound swab, pus, ear swab, blood and vagina swab, etc.	Human sample	(Odumosu *et al*., 2015)

9	*spa*	-	*S. aureus*	Nares swab	Human sample	(Ayeni *et al*., 2015)
10	*dfrA14, tetD, qnrS, and oqxAB; blaCTX_M-15*.	Possession of virulence genes including yersiniabactin (*ybt* genes and *fyuA*), aerobactin (*iuc*, salmochelin (*iro*), capsule expression upregulators *rmpADC* and *rmpA2*); capsular loci (KL102, KL123, KL2, KL62, KL27, and O1v1, O2v2, O1v2); combination of mutation in the quinolone resistance determining region (QRDR) of *gyrA* and *parC*; efflux-mediating mutations in the *acrR* gene.	*K. pneumoniae*	Blood, urine, ocular, stool, throat, and rectal swabs	Human sample	(Afolayan *et al*., 2021)
11	*ant (3'')[c], aph (3")[c], ant (3")[b],* and *aph(6)-1d[d]*.	-	*Acinetobacter* species, *Aeromonas* species, *Alcaligenes* species, *Bacillus* species, *Bordetella* species, *Brevundimonas* species, *Chromobacterium* species, *Klebsiella* species, *Leucobacter* species, *Morganella* species, *Pantoae* species, *Proteus* species, *Psychrobacter* species and *Serratia* species.	Treated and untreated water	Non-human sample	(Adesoji *et al*., 2019)
12	*mecA and spa*.	Possession of virulence genes	*S. aureus*	Chicken and pig carcasses and their handlers	Human and non-human sample	(Okorie-Kanu *et al*., 2020)
13	*blaCTX-M-15, blaCTX-M-14, blaCTX-M-11, blaSHV-28, blaSHV-33, blaCTX-M-65,*	-	*Klebsiella pneumoniae, Escherichia coli, Enterobacter asburiae, Enterobacter cloacae, Enterobacter hormaechei, Atlantibacter hermannii, Citrobacter werkmanii* and *Proteus mirabilis.*	Clinical isolates	Human sample	(Jesumirhewe *et al*., 2020)
14	*sul1, sul2,* and *Intl1*.	-	*Bacillus methylotrophicus, Acinetobacter* spp, *Klebsiella pneumoniae, Enterobacter hormaechei, Serratia marcescens, Aeromonas aquariorum* and *Staphylococcus saprophyticus.*	Waste water	Non-human sample	(Obayiuwana *et al*., 2018)

15	*blaVIM-1*	-	*P. aeruginosa*	Clinical isolates from all samples except stool	Human sample	(Zubair and Iregbu, 2018)
16	*sul1, sul2,* sul3, dfrA1, dfrA7, *intl1* and *intl2.*	-	*Citrobacter* species, *Enterobacter* species, *Pseudomonas* species, *Achromobacter* species *and Escherichia* species.	Wetland sediments	Non-human sample	(Adelowo *et al*., 2018)b-Nov
17	*Tet(A),* tet(B), *Tet(E),* tet(M) and *Tet30.*	Efflux pumps and ribosomal protection proteins.	*Alcaligenes* spp, *Bacillus* spp, *Leucobacter* species, *Proteus* species, *Aeromonas* species, *Klebsiella* species and *Morganella* species.	Treated and untreated water	Non-human sample	(Adesoji *et al*., 2015)a
18	*blaTEM, blaSHV* and *blaCTX-M*	-	Extended spectrum beta lactamase producing *Escherichia coli*.	urine, pleural and peritoneal aspirate, blood, wound swabs and cerebro-spinal fluids.	Human sample	(Nwafia *et al*., 2019)
19	*Sul2, strB, catB3 and* Class 1 integrase.	-	*Vibrio parahaemolyticus, V. vulnifus, V. fluvialis, Vibrio alginolyticus, Vibrio cholarae, Vibrio mimicus, Vibrio harveyi, Vibrio* species.	Ready-to-eat shrimp	Non-human sample	(Beshiru *et al*., 2020)
20	*blaTEM, sul1, sul2, sul3, qnrS, aadA, strA, strB, catA1, cmlA1, tetA, tetB,* class 1 and 2 integrons.	-	*E. coli*	Waste, litter, soil and water samples from poultry farms.	Non-human sample	(Adelowo *et al*., 2014)
21	*qnrS1*	Multiple mutations in *gyrA* and/or *parC.*	*Salmonella* species	Faeces, samples from cattle, camel, poultry-related sources (rodents, litter, water and lizards), catfish, vegetables and humans.	Human and Non-human sample	(Raufu *et al*., 2013)
22	*Sul1, sul2* and *vanA.*	-	*Escherichia* and *Staphylococcus* species	Chicken litter	Non-human sample	(Olonitola *et al*., 2015)
23	*blaVIM, blaGES, blaNDM, blaPDC, blaACT-7, blaAmpC, blaCMY-31, blaSHV-11, blaCTX-M-15, blaSRT-2, blaTEM-1* and *blaOXA.*	Carbapenemase production and reduced production of outer membrane porins	*Klebsiella* species, *Escherichia coli, Pseudomonas* species and *Proteus* species.	Isolates from clinical samples	Human sample	(Ogbolu *et al*., 2020)

24	*Pvl* and *mecA*	-	*S. aureus*	Throat, nasal and wound swab	Human sample	(O'malley *et al*., 2015)
25	*floR*	-	*Proteus penneri*, *Pseudomonas* spp, *Proteus vulgaris*, *Serratia mercescens* and *Acinetobacter baumanni*.	Drinking water distribution system	Non-human sample	(Adesoji and Call, 2020)
26	*gyrA*, *parC* and *qnrS1*	Overactive efflux pump	*E. coli*	Fecal samples	Human sample	(Lamikanra *et al*., 2011)
27	*mecA*, *pvl* and *dfrA*	Possession of spa gene.	MRSA, MSSA, CoNS (*S. sciuri*, *S. haemolyticus*, *S. warneri* and *S. epidermidis*.	Staphylococcal isolates from clinical samples	Human sample	(Shittu *et al*., 2012)
28	*mecA*, *blaZ*, *tet*(K), *tet*(M), *tet*(L), *erm*(B), *erm*(C), *aacA-aphD*, *aphA3*, *str*, *dfrK*, *dfr*G, *cat*pC221, and *cat*pC223	Mutation	*S. sciuri*, *S. lentus*, *S. cohnii* and *S. haemolyticus.*	nasal and ear swabs of pigs	Non-human sample	(Ugwu *et al*., 2015)
29	*IncF*, *blaTEM-1*, *blaSHV-1*, *blaOXA-1*, *blaCTX-M-15*, *blaCTX-M-3* and *blaAmpC*.	Possession of DHA-like enzymes and IncF plasmids encoding beta-lactamases	*P. mirabilis*, *K. pneumoniae*, *B. cepacia*, *M. morganii* and *C. freundii*.	aspirates, ear swab, wound swab, throat swab, high vaginal swab, eye swab, sputum, urine, cerebrospinal fluid and blood culture	Human sample	(Ogbolu *et al*., 2013)
30	*bla*VIM-5, *bcr*2, *aph*(3")-lb, *aph*(6)-ld, *tetB*, *tetC*, *tetG*, *floR*, and *macAB.*	Integrons containing the aminoglycoside adenyltransferase gene and the carbenicillin-hydrolysing class A beta-lactamase gene; multidrug resistance efflux protein; mutation.	*Pseudomonas putida* group (*Pseudomonas plecoglossicida* and *Pseudomonas guariconensis*)	polluted urban wetlands	Non-human samples	(Adelowo *et al*., 2018)a
31	*tetA* and *sul1*	Multidrug efflux pump; dihydropteroate synthase gene; virulence genes, including a hemolysin.	*E. coli*	*E. coli* isolates	Human sample	(Hazen *et al*., 2017)
32	*aac(6')-Ib, qnrA, acc(6')-Ib-cr*, *bla*TEM-1, *bla*CTX-M-15 and class 1 integrons (*IntI1*, *IntI2)*.	-	*Proteus mirabilis*	Wound, ear, eye, vaginal swabs, urine, sputum, peritoneal effluents, finger abscesses, pus and stool.	Human sample	(Alabi *et al*., 2017)
33	*mecA*	-	*Staphylococcus* species	Wound swabs, endocervical swabs, high vaginal swabs, eye swabs, ear swabs, urethral swabs,	Human sample	(Ibadin *et al*., 2017)

				seminal fluid, blood, urine, aspirates and urinary catheter tips.		
34	*mecA, blaTEM, blaSHV, blaCTX-M, blaKPC, blaNDM* and *blaVIM*	-	MRSA, *Enterobacter* species, *Klebsiella* species, *Citrobacter* species, *E. coli*, *Providencia* spp, *Morganella morganii*, *H. alvei, A. baumannii.*	Tissue Biopsies/aspirates	Human sample	(Adeyemo *et al*., 2021)
35	*mecA, blaZ,* tet(K), *tet*(M), *tet*(L), *erm*(B), *lnu*(A), *aacA-aphD, aphA3, str*, *dfr*(G), *cat*pC221, and *cat*pC223	Chromosomal mutation	*S. sciuri* subspecies *rodentium, S. lentus, S. haemolyticus*, and *S. simulans.*	Groin area swabs of dogs	Non-human sample	(Chah *et al*., 2014)
36	*pvl* and *tetM*	-	*S. aureus*	Cloacae and nasal swabs from birds (broilers and layers)	Non-human sample	(Nworie *et al*., 2017)
37	*blaCTX-M-15, blaCTX-M-14, blaCTX-M-55, qnrS1,* and *mcr1*.	Detection Folate pathway antagonists; chromosomal mutation.	*E. coli*	Human stool, cecal content of slaughtered beef cattle, abattoir waste water and meat stall swabs.	Human & non-human sample	(Aworh *et al*., 2022)
38	*aac(3)-IIa*, *aac(6')-lb-cr*, *aph(3'')-Ib*, *aadA5*, *aadA2*, *aadA1*, *aph(6'')-Ib*, *bla*CMH-3, *bla*CTX-M-15, *bla*OXA-1, *catB4, dfrA17*, *bla*TEM-1B, *catA1*, *catB4*, *dfrA14*, *fosA*, *oqxA*, *oqxB*, *tetA, mphA, sul1, sul2, tet(B)*, *bla*CMY-89 and *dfrA18*.	Possession of IncF conjugative plasmids (replicon type II-IA-IB), *IncH*-like plasmids of replicon types *IncH12* and *H12A*, *IncR* plasmids; virulence genes (*ast*, *capU*, *gadAB*, *iss*, and genes for the biosynthesis and uptake of the siderophore aerobactin); genes coding for transposases, integrases, and other insertion elements.	MDR *Escherichia coli*, *Enterobacter cloacae* and *Citrobacter freundii.*	polluted wetlands	Non-human sample	(Adelowo *et al*., 2020)
39	*blaCTX-M* and *blaTEM.*	-	*E. coli*	Fecal samples from cattle and pigs	Non-human sample	(Olowe *et al*., 2015)
40	*blaSHV, blaTEM* and *blaCTX-M-15*.	-	*Enterobacter cloacae*	Urine	Human sample	(Bebe *et al*., 2020)
41	*blaTEM, blaCTX-M-15, blaSHV, blaOXA-1, blaCMY-2*; *qnrB, qnrD, qnrS*; *aac(6')-Ib*; *aadA1*, *aadA2*, *aacC2*; *gyrA* and *parC.*	-	*E. coli*	Urine	Human sample	(Onanuga *et al*., 2019)
42	*blaCTX-M-15, qnrA1, qnrB1* and *aac-(6¢)-lb-cr*.	-	*Escherichia coli*	clinical Escherichia coli isolates	Human sample	(Aibinu *et al*., 2011)
43	*dfrA7*	-	*Escherichia coli*	Clinical isolates	Human sample	(Labar *et al*., 2012)

44	*blaCTX-M, blaTEM* and *blaSHV*	-	*E. coli*	Rectal swabs from children with diarrhea	Human sample	(Saka *et al*., 2020)
45	*blaCTX-M-15*	Mutation (insertion sequence *blaISEcP1* upstream of the *blaCTX-M-15* genes).	*E. coli, K. pneumoniae, P. mirabilis, M. morganii* and *C. freundii*.	Clinical isolates	Human sample	(Raji *et al*., 2015)
46	*tetA, tet30, sul1, sul 2, blaTEM, bla*SHV, *aph*(*3″*)[c], *ant*(*3″*), *aph*(6)*1d*[d], class 1 integron (*int 1* and *v1*).	Efflux pump mechanism.	*P. putida, P. otitidis* and *P. fluorescens.*	treated and untreated water	Non-human sample	(Adesoji *et al*., 2015)b
47	*mcr-1.1; aadA1, aph(6)-Id, aph(3)-Ib, aac(3)-IId, aac(6)-Ib-cr; blaTEM-1, blaOXA-1, blaOXA-10, blaOXA-129, blaCTX-M-15, blaCTXM-65*; *qnrB1, qnrB19, qnrB52, qnrS1, qnrS2, qnrS3, qnrS7, qnrS11, qnrS13*; *dfrA1, dfrA8, dfrA12, dfrA14, dfrA15, dfrA17, dfrA21, dfrA27; mdfA, mphA, mefB, ermB, ereA, mphE, msrE; cmlA1, catA1, catA2, catB3, floR;* ARR-2, ARR-3; *sul1, sul2, sul3; tet*A, *tet*B, *tet*M.	Mutations that occur in the *gyr*A, *par*C, and *par*E genes; possession of *IncF* plasmid replicon, phage plasmid.	MDR *E. coli*	Stool of apparently healthy poultry workers, faecal samples obtained from chickens as well as from poultry litter and water obtained from farm and environments.	Human and non-human sample	(Aworh *et al*., 2021)
48	*blaNDM* and *blaVIM*	-	*E. cloacae, K. pneumoniae, P. mirabilis, P. rettgeri* and *P. aeruginosa.*	Bacterial isolates from intra-abdominal, urinary tract, skin and soft tissue, lower respiratory tract, and bloodstream infections	Human sample	(Kazmierczak *et al*., 2016)
49	*rpoB, katG, inhA* and *oxyR*	Mutation: (*rpoB* mutations occurring in codons 526 to 529, 530 to 533; mutations found in *katG* were point mutation, S315T1, and the *inhA* promoter was mutated at the C15T position).	*M. tuberculosis*	Sputum	Human sample	(Dinic *et al*., 2012)
50	*tetA, bla*$_{TEM}$, *qnrB* and *qnrS.*	-	*Salmonella enterica* serovars	Fecal sample	Human and non-human sample	(Ajayi *et al*., 2019)
51	*mec (SSCmec), msr*, and *mupB*	-	*Staphylococcus aureus*	Urine, blood, semen, endo-cervix vaginal swab, wound swab, aspirate.	Human sample	(Obasuyi *et al*., 2020)
52	*StrA, strB, aac (3)-II, aac (3)-1V, tetA, tetB, sul1, dfr/A, sul3 and dfr/G.*	-	*Salmonella species*	Chicken droppings	Non-human sample	(Nwiyi *et al*., 2018)

53	*blaTEM*	Possession of virulence genes (*invA*, *sopB*, *mgtC*, *spi4D*, *ssaQ* and *spvC*)	*Salmonella enterica* serovars	Droppings of layer chickens	Non-human sample	(Shittu *et al*., 2022)
54	*tetA*, *tetB*, *tetG* and *tetM*	-	*Escherichia coli*	Urine, stool, soil and poultry.	Human and non-human sample	(Perewari *et al*., 2022)
55	*catA1*; *sul1*; *tet(E)*; *aac(3)-IV*; *ermC*; bla_{TEM}, bla_{CTX-M} and $bla_{NDM-1.}$	-	*Acinetobacter* spp	Pharmaceutical wastewaters	Non-human sample	(Obayiuwana and Ibekwe, 2020)

Table 2:
Prevalence of antibacterial resistant genes in Nigeria

S/N	Resistant gene	Frequency	Prevalence (%)
1	*aac (3)-II*	1	0.23
2	*aac (6') – I*	1	0.23
3	*aac(3)-IIa*	1	0.23
4	*aac(3)-IId*	1	0.23
5	*aac(3)-IV*	2	0.46
6	*aac(6')-Ib*	2	0.46
7	*aac(6)-Ib-cr*	2	0.46
8	*aac(6')-lb-cr*	2	0.46
9	*aac-(6¢)-lb-cr*	1	0.23
10	*aac6'-aph2*	1	0.23
11	*aacA-aphD*	3	0.69
12	*aacC2*	1	0.23
13	*aadA*	1	0.23
14	*aadA1*	3	0.69
15	*aadA2*	2	0.46
16	*aadA5*	1	0.23
17	*ant (2'') – I*	1	0.23
18	*ant (3")*[b]	1	0.23
19	*ant (3'')*[c]	1	0.23
20	*ant(3")*	1	0.23
21	*aph(3)-Ib*	1	0.23
22	*aph(3'')-Ib*	2	0.46
23	*aph(3")*[c]	2	0.46
24	*aph(6)1d*[d]	2	0.46
25	*aph(6)-Id*	1	0.23
26	*aph(6)-ld*	1	0.23
27	*aph(6'')-Ib*	1	0.23
28	*aphA3*	2	0.46
29	*arr2*	1	0.23
30	*ARR-2*	1	0.23
31	*ARR-3*	1	0.23
32	*arr4*	1	0.23
33	*arr5*	1	0.23
34	*arr7*	1	0.23
35	*bcr2*	1	0.23
36	*blaACT-7*	1	0.23
37	*blaADC*	1	0.23
38	*blaAmpC*	2	0.46
39	*blab*	1	0.23
40	*bla*CMH-3	1	0.23
41	*blaCMY*	1	0.23
42	*blaCMY-2*	1	0.23
43	*blaCMY-31*	1	0.23
44	*bla*CMY-89	1	0.23
45	*blaCTX-M*	**8**	**1.85**
46	*bla*CTX-M-11	1	0.23
47	*blaCTX-M-14*	2	0.46
48	*bla*$_{\text{CTX-M-15}}$	**12**	**2.77**
49	*blaCTX-M-3*	1	0.23
50	*blaCTX-M-55*	1	0.23
51	*blaCTXM-65*	2	0.46
52	*bla*$_{\text{GES}}$	2	0.46
53	*blaGOB*	1	0.23
54	*blaKPC*	1	0.23
55	*blaLEN*	1	0.23
56	*blaLRA*	1	0.23
57	*blaNDM/1*	4	0.92
58	*blaOKP*	1	0.23
59	*blaOXA*	2	0.46
60	*blaOXA-1*	4	0.92
61	*blaOXA-10*	1	0.23
62	*blaOXA-129*	1	0.23
63	*blaPDC*	2	0.46
64	*blaSHV*	**8**	**1.85**
65	*blaSHV-1*	1	0.23
66	*blaSHV-11*	1	0.23
67	*bla*SHV-28	1	0.23
68	*bla*SHV-33	1	0.23
69	*blaSRT-2*	1	0.23
70	*blaTEM*	**12**	**2.77**
71	*blaTEM-1*	4	0.92
72	*bla*TEM-1B	1	0.23
73	*blaVEB*	1	0.23
74	*bla*$_{\text{VIM}}$	3	0.69
75	*blaVIM-1*	1	0.23
76	*blaVIM-5*	1	0.23
77	*blaZ*	2	0.46
78	*cat*	1	0.23
79	*catA1*	4	0.92
80	*catA2*	1	0.23
81	*catB3*	2	0.46
82	*catB4*	2	0.46
83	*cat*pC221	2	0.46
84	*cat*pC223	2	0.46
85	Class 2 integrons	1	0.23
86	*cml*	1	0.23
87	*cmlA*	1	0.23
88	*cmlA1*	1	0.23
89	*cmlV*	1	0.23
90	*Cmr*	1	0.23
91	*cpt*	1	0.23
92	*dfr/A*	2	0.46
93	*dfr/G*	3	0.69
94	*dfrA1*	2	0.46
95	*dfrA12*	1	0.23
96	*dfrA14*	3	0.69
97	*dfrA15*	1	0.23
98	*dfrA17*	2	0.46
99	*dfrA18*	1	0.23
100	*dfrA21*	1	0.23
101	*dfrA27*	1	0.23
102	*dfrA7*	2	0.46
103	*dfrA8*	1	0.23
104	*dfrK*	1	0.23
105	*dha1*	1	0.23
106	*ereA*	1	0.23
107	*erm*(B)	4	0.92
108	*erm*(C)	2	0.46
109	*ermA*	1	0.23
110	*ermTR*	1	0.23
111	*fexA*	1	0.23
112	*floR*	4	0.92
113	*fomA*	1	0.23
114	*fomB*	1	0.23
115	*fomC*	1	0.23
116	*fosA*	1	0.23
117	*fosA*	1	0.23
118	*fosB*	1	0.23
119	*fosC*	1	0.23
120	*fosD*	1	0.23
121	*fosE*	1	0.23
122	*fosF*	1	0.23
123	*fosG*	1	0.23
124	*fosH*	1	0.23
125	*fosX*	1	0.23
126	*gyrA*	3	0.69
127	*IncF*	1	0.23
128	*inhA*	1	0.23
129	*int1* (Class 1 integron)	6	1.39

130	*intI2* (Class 1 integron)	3	0.69
131	*Iri*	1	0.23
132	*katG*	1	0.23
133	*Lnu*	1	0.23
134	*lnu*(A)	1	0.23
135	*macAB*	1	0.23
136	*mcr1*	3	0.69
137	*mcr-1.1*	1	0.23
138	*mcr-2*	1	0.23
139	*mcr-3*	1	0.23
140	*mcr-4*	1	0.23
141	*mcr-5*	1	0.23
142	*mcr-6*	1	0.23
143	*mcr-7*	1	0.23
144	*mcr-8*	1	0.23
145	*mcr-9*	1	0.23
146	*mdfA*	1	0.23
147	*mec (SSCmec)*	1	0.23
148	*mecA*	**8**	**1.85**
149	*mef*	1	0.23
150	*mefB*	1	0.23
151	*mphA*	2	0.46
152	*mphE*	1	0.23
153	*Msr*	1	0.23
154	*msrA*	1	0.23
155	*msrE*	1	0.23
156	*mupB*	1	0.23
157	*oqxA*	1	0.23
158	*oqxAB*	2	0.46
159	*oqxB*	1	0.23
160	*otrA*	1	0.23
161	*otrB*	1	0.23
162	*otrC*	1	0.23
163	*oxyR*	1	0.23
164	*parC*	3	0.65
165	*pexA*	1	0.23
166	*Pvl*	3	0.69
167	*qnrA*	1	0.23
168	*qnrA1*	1	0.23
169	*qnrB*	3	0.69
170	*qnrB1*	2	0.46
171	*qnrB19*	1	0.23
172	*qnrB52*	1	0.23
173	*qnrC*	1	0.23
174	*qnrD*	2	0.46
175	*qnrS*	5	1.15
176	*qnrS1*	4	0.92
177	*qnrS11*	1	0.23
178	*qnrS13*	1	0.23
179	*qnrS2*	1	0.23
180	*qnrS3*	1	0.23
181	*qnrS7*	1	0.23
182	*qnrVC*	1	0.23
183	*Rif*	1	0.23
184	*Rph*	1	0.23
185	*rphD*	1	0.23
186	*rpoB*	1	0.23
187	*Spa*	2	0.46
188	*Str*	2	0.46
189	*strA*	2	0.46
190	*strB*	3	0.69
191	*sul1*	**10**	**2.31**
192	*sul2*	**8**	**1.85**
193	*sul3*	4	0.92
194	*tcr-3*	1	0.23
195	*tet(31)*	1	0.23
196	*tet(32)*	1	0.23
197	*tet(33)*	1	0.23
198	*tet(34)*	1	0.23
199	*tet(35)*	1	0.23
200	*tet(36)*	1	0.23
201	*tet(37)*	1	0.23
202	*tet(38)*	1	0.23
203	*tet(39)*	1	0.23
204	*tet(40)*	1	0.23
205	*tet(41)*	1	0.23
206	*tet(42)*	1	0.23
207	*tet(43)*	1	0.23
208	*tet(44)*	1	0.23
209	*tet(45)*	1	0.23
210	*tet(47)*	1	0.23
211	*tet(48)*	1	0.23
212	*tet(49)*	1	0.23
213	*tet(50)*	1	0.23
214	*tet(51)*	1	0.23
215	*tet(52)*	1	0.23
216	*tet(53)*	1	0.23
217	*tet(54)*	1	0.23
218	*tet(57)*	1	0.23
219	*tet(58)*	1	0.23
220	*tet(59)*	1	0.23
221	*tet(60)*	1	0.23
222	*tet*(K)	3	0.69
223	*tet*(L)	3	0.69
224	*tet30*	3	0.69
225	*tet39*	1	0.23
226	*tetA*	**10**	**2.31**
227	*tetAB*	1	0.23
228	*tetB*	**8**	**1.85**
229	*tetC*	2	0.46
230	*tetD*	2	0.46
231	*tetE*	3	0.69
232	*tetG*	3	0.69
233	*tetH*	1	0.23
234	*tetJ*	1	0.23
235	*tetM*	**9**	**2.08**
236	*tetO*	1	0.23
237	*tetQ*	1	0.23
238	*tetR*	1	0.23
239	*tetS*	1	0.23
240	*tetT*	1	0.23
241	*tetU*	1	0.23
242	*tetV*	1	0.23
243	*tetW*	1	0.23
244	*tetX*	1	0.23
245	*tetY*	1	0.23
246	*tetZ*	1	0.23
247	*v1* (Class 1 integron)	1	0.23
248	*vanA*	1	0.23
249	*vanR*	1	0.23
250	*vanS*	1	0.23
Total		**433**	**100**

Table 3:
Sources of the Antibacterial Resistant Genes

S/N	Sample Sources	Frequency	Prevalence (%)
1	Human	34	55.7
2	Non-human	27	44.3
Total		**61**	**100**

Table 4:
Prevalence of the Bacteria Carrying the Resistant Genes

S/N	Bacteria	Frequency	Prevalence (%)
1	*Achromobacter* spp	1	0.63
2	*Acinetobacter baumanni*	2	1.26
3	*Acinetobacter* spp	3	1.89
4	*Aeromonas aquariorum*	1	0.63
5	*Aeromonas* spp	2	1.26
6	*Alc. faecalis*	1	0.63
7	*Alcaligenes* spp	2	1.26
8	*Atlantibacter hermannii*	1	0.63
9	*B. cepacia*	1	0.63
10	*B. cereus*	1	0.63
11	*Bacillus methylotrophicus*	1	0.63
12	*Bacillus* spp	2	1.26
13	*Bifidobacterium adolescentis*	1	0.63
14	*Bordetella* spp	1	0.63
15	*Brev. diminuta*	1	0.63
16	*Brevundimonas* spp	1	0.63
17	*C. cellulans*	1	0.63
18	*C. freundii*	3	1.89
19	*Chromobacterium* spp	1	0.63
20	*Citrobacter* species	3	1.89
21	*Citrobacter werkmanii*	1	0.63
22	*Corynebacterium striatum*	1	0.63
23	*Enterobacter asburiae*	1	0.63
24	*Enterobacter cloacae*	4	2.52
25	*Enterobacter* species	3	1.89
26	*Enterobacter hormaechei*	3	1.89
27	*Escherichia coli*	**21**	**13.2**
28	*Escherichia fergusonnii*	1	0.63
29	*Escherichia* species	2	1.26
30	*H. alvei*	1	0.63
31	*Klebsiella pneumoniae*	**8**	**5.03**
32	*Klebsiella* spp	4	2.52
33	*L. sphaericus*	1	0.63
34	*Leucobacter* spp	2	1.26
35	*M. tuberculosis*	1	0.63
36	*Morganella morganii*	3	1.89
37	*Morganella* species	2	1.26
38	*P. fluorescens*	1	0.63
39	*P. guariconensis*	1	0.63
40	*P. otitidis*	1	0.63
41	*P. plecoglossicida*	1	0.63
42	*P. putida*	1	0.63
43	*P. rettgeri*	2	1.26
44	*Pantoea* species	2	1.26
45	*Proteus mirabilis*	5	3.14
46	*Proteus penneri*	1	0.63
47	*Proteus* spp	3	1.89
48	*Proteus vulgaris*	1	0.63
49	*Providencia* spp	1	0.63
50	*Pseudomonas aeruginosa*	3	1.89
51	*Pseudomonas* spp	3	1.89
52	*Psychrobacter* spp	1	0.63
53	*S. aureus*	**9**	**5.66**
54	*S. cohnii*	1	0.63
55	*S. epidermidis*	1	0.63
56	*S. haemolyticus*	3	1.89
57	*S. lentus*	2	1.26
58	*S. sciuri*	2	1.26
59	*S. sciuri* subspecies *rodentium*	1	0.63
60	*S. simulans*	1	0.63
61	*S. warneri*	1	0.63
62	*Salmonella enterica* serovars	2	1.26
63	*Salmonella species*	2	1.26
64	*Serratia marcescens*	2	1.26
65	*Serratia* species	2	1.26
66	*St. maltophilia*	1	0.63
67	*Staphylococcus saprophyticus*	1	0.63
68	*Staphylococcus* species	2	1.26
69	*Streptococcus agalactiae*	1	0.63
70	*Streptomyces rishiriensis*	1	0.63
71	*Vibrio alginolyticus*	1	0.63
72	*V. cholarae*	1	0.63
73	*V. fluvialis*	1	0.63
74	*V. harveyi*	1	0.63
75	*V. mimicus*	1	0.63
76	*V. parahaemolyticus*	1	0.63
77	*V.* species	1	0.63
78	*V. vulnifus*	1	0.63
Total		**159**	**100**

The transformant strains carried a common plasmid approximately of 108-kb. A positive result was seen for IncF, indicating this is the replicon type of the frequent plasmids encoding beta-lactamases (Ogbolu *et al*., 2013). Adelowo *et al*. (2018) observed A gyrA mutation (Thr83Ile) conferring fluoroquinolone resistance present in all the P. plecoglossicida strains but not in the P. guariconensis isolates, consistent with the sensitivity of the latter strains to ciprofloxacin. Aworh *et al*. (2021) reported mutations in the gyrA, parC, and parE genes; possession of incF plasmid replicon, phage plasmid. Other mechanisms of the resistance was due to the virulence-associated genes the organisms harbour, including the transcriptional regulator hha and the putative calcium sequestration inhibitor (csi) (Hazen *et al*., 2017). Adelowo *et al*. (2020) found MDR and potentially pathogenic E. coli belonging to the globally distributed ST10 complex harboring blaCTX-M-15 on a self-transmissible IncF plasmid in a polluted urban wetland (Awba) in Ibadan, southwestern Nigeria. The wetland also harbored MDR E. cloacae with blaCTX-M-15 on a self-transmissible IncH plasmid, and C. freundii with blaTEM-1B on an IncR plasmid. Shittu *et al*. (2022) reported the possession of virulence genes (invA, sopB, mgtC, spi4D, ssaQ and spvC). Ogbolu *et al*. (2020) reported carbapenemase production and reduced production of outer membrane porins.

CONCLUSION

Antibiotics resistance has continued to threaten the success of treatment of bacterial infections, with more resistance strains emerging constantly. The most prevalent antibacterial genes were blaCTX-M-15, blaTEM, sul1, tetA, tetM, blaCTX-M, blaSHV, mecA, sul2 and tetB. The resistance genes were identified from humans and non-humans sources. The most prevalent bacteria harboring the antibiotic resistant genes was Escherichia coli, followed by Staphylococcus aureus and Klebsiella pneumoniae. The wide distribution of antibacterial resistance genes in non-human sources makes them reservoirs of these genes that were resistant, which could potentially be spread from microorganisms that were commensals to human pathogenic ones. Harbouring these resistant genes reflects antibiotic use in those regions. This calls for immediate intervention to curb increased spread of the antibacterial resistance genes.

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