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Molecular Characterization of Quinolone Resistance Gene in *Pseudomonas Aeruginosa* Isolated from Stored House Hold Water in Karu Metropolis, Nasarawa State.

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ABSTRACT

Pseudomonas aeruginosa is a critical pathogen that harbors plasmids-mediated resistant genes that are easily transferable amongst its population and to other bacteria. It is important to study the patterns of resistance to antibiotics in the case of infection with *P. aeruginosa*, as this will determine the most effective course of treatment. This study aimed to characterise the plasmids-mediated quinolone resistance genes from *P. aeruginosa* isolated from household stored water in Karu metropolis, Nasarawa State, Nigeria. 180 water samples were collected from Karu environs, and *P. aeruginosa* was isolated using standard microbiological techniques. Antimicrobial susceptibility testing was done using the disc diffusion method, and the quinolones-resistant genes were amplified by polymerase chain reaction. The percentage occurrence of *P. aeruginosa* in household stored water around Karu Metropolis was 23.3%, with highest occurrence in New Karu at 26.6%. These isolates were highly resistant to common antibiotics used in the treatment of *P. aeruginosa* infection such as cefuroxime and cefexime (100%), cefutaxone and Amoxicillin Clavulanate (97.6%), ceftriaxine/Sulbactam (95.2%), nitrofurantoin (92.9%) but with low resistance to levofloxacin (9.5%) and Ofloxacin (4.8%). The plasmids quinolones resistant genes detected were *qnrS* (66.7%) and *qepA* (33.3%); however, *qnrA, qnrB* and oqxA were not detected. The presence of plasmid-mediated quinolones resistance genes (PMQR) in *P. aeruginosa* is of public health concern, as this will limit the number of antibiotics available for bacterial infection treatment. Further investigations of PMQR genes in other bacteria species should be done which will help in giving insight on proper antibiotics prescription.

Keywords: Water, *Pseudomonas aeruginosa*, Plasmids, Quinolone resistance genes.

1. Introduction

Pseudomonas aeruginosa is a gram-negative rod-shaped opportunistic pathogen bacterium, commonly found in the environment (Qin *et al.,* 2022). The existence of *P. aeruginosa* in drinking water is related to its ability to colonize surfaces; it can also survive in deionized water. This microbe is widely known as a serious pathogen in hospital environments causing infections such as endocarditis, osteomyelitis, pneumonia, urinary tract infections, gastrointestinal infections, meningitis, folliculitis and keratitis. It is also a leading cause of septicemia and a major pathogen in burns and patients with cystic fibrosis (CF) (Mena & Gerba, 2020).

Pseudomonas aeruginosa causes acute and chronic infections, with a high rate of mortality of up to 40% recorded (Bassetti, 2018). Most infections are caused by gram-negative rods, with *P. aeruginosa* playing the leading role in ill and immunocompromised patients (El Zowalaty *et al.,* 2015). The major challenges leading to high mortality lie in the appearance of drug-resistant strains of these bacteria. *P. aeruginosa* water pollution is related to water stored in tanks, pipeline waters, and household filters. Drinking such water can result in diarrhoea, vomiting with severe dehydration, which are some of the symptoms associated with *P. aeruginosa* (Alatraktchi, 2022). *P. aeruginosa* has shown resistance to disinfectants used in treating water such as chlorine, chloramines, ozone, or iodine. In hospitals, tap water is also considered a means of transmission (Mena & Gerba, 2020). *P. aeruginosa* can cause diarrhoea in patients with prolonged use of antibiotics, as they have developed resistance to quinolones (Alatraktchi, 2022).

Quinolones are broad-spectrum antimicrobial agents with bactericidal effects and good oral bioavailability; they are used to treat bacterial infections caused by *P. aeruginosa* (Pham *et al.,* 2019). Their mechanism of action is by inhibiting nucleic acid synthesis through the disruption of topoisomerase IV and DNA gyrase, causing tension during the unwinding of DNA (Deoxyribonucleic acid) and breakage of bacterial chromosomes (Pham *et al.,* 2019). Bacteria have acquired resistance to quinolones due to frequent mutation. The resistance mechanism is

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mediated by chromosomal mutations and plasmid gene uptake that can alter the target site, reduce drug intake, modify the quinolones, or even reduce drug accumulation by either decreased uptake or increased efflux (Pham *et al.,* 2019). The use of quinolones in the treatment of gram-negative bacilli has led to the development of resistance due to the non-adherence of patients to drug prescriptions and also self-medication (Bassetti, 2018). Plasmid-mediated quinolone resistance (PMQR) is a common mechanism of resistance (Adwan & Omar, 2021). Plasmid-mediated genes are easily transferable to other bacteria, which makes them confer resistance to these antibiotics. The PMQR genes responsible for resistance include *qnrA, qrnB, oqxAB, qnrS, and qepA* (Taha & Omar, 2019).

2. Materials and Methods

2.1. Study Area

This study was conducted in Karu Metropolis, Karu Local Government Area Council, Nasarawa State, Nigeria. It is located at Latitude and longitude coordinates 7.6005786, 9.03368 with an area of 2,640 km^2.

2.2. Study Design

A cross/sectional study design with a systematic random sampling technique was utilized. The sample size was determined as described by Jung (2014).

$$n = Z^2 P(1-P)/d^2$$

Where: n= Sample size

Z= Normal deviation at the desired confidence interval. In this case, it will be taken at 95%; Z value at 95% is 1.96.

P= Expected proportion from the previous studies.

d= degree of precision. Since the proportion of the population with the characteristic is not known, then 50% will be used, i.e. 0.05

Z= 1.96

P= 13.5% (0.14) from Darija *et al.,* 2021

d= 0.05

$$n = 1.96^2\ 0.014\ (1 - 0.014)/0.05^2$$

$$n = 170$$

2.3. Sample Collection

One hundred and eighty water samples were collected from household storage water tanks in sterile containers in Karu Metropolis.

2.4. Isolation of Pseudomonas aeruginosa

Isolation of *P. aeruginosa* was carried out as described by Kalu *et al.* (2022)**.** Exactly 1 ml of the water sample was dispensed into 10 ml of tryptic soy broth and incubated for 24h at 37°C. After 24h of incubation, the broth cultures were inoculated onto Cetrimide Agar plates aseptically; all plates were incubated for 24h at 37°C.

2.5. Summarized culture identification of Pseudomonas aeruginosa

The *Pseudomonas aeruginosa* culture showed greenish slimy growth with a characteristic grape like odour. Gram staining revealed Gram-negative rods. Biochemical tests confirmed it was catalase-positive, oxidase-positive, citrate-positive, and negative for indole, Methyl Red (MR), and Voges-Proskauer (VP) tests. *Pseudomonas aeruginosa* is a glucose non fermenter and is thus negative for MR and VP.

2.6. Antimicrobial Susceptibility Testing

The standard disc diffusion method was used. Two pure colonies of the isolates were inoculated into 5 ml of sterile 0.85% (w/v) NaCl. The turbidity of the bacteria suspension was adjusted to 0.5 McFarland Standard to get 1 x10^8 CFU/ml. A sterile swab stick was used to streak the standardized bacterial suspension on Muller Hinton agar plates and the antibiotics discs were aseptically placed. The plates were allowed to stand for 1 hour and then incubated at 37^{0}C for 24 h. The zone of inhibition was measured in millimetres and was interpreted as per the susceptibility break point of the Clinical and Laboratory Standard Institute (CLSI, 2020).

2.7. Determination of Multiple Antibiotic Resistance (MAR) Index

The MAR index was determined as described by Ngwai *et al.* (2014).

MAR Index = Number of antibiotics isolates resistance/Number of antibiotics tested.

2.8. Molecular Detection of Quinolone Resistance Gene

The isolates resistant to quinolone antibiotics were used in the molecular detection of plasmid-mediated quinolone-resistant genes.

2.9. Primers:

The primers used in this study are as shown in Table 1.

Table 1. Primer sequences of Plasmid-mediated quinolones resistance genes.

Target Gene	Primer	Oligonucleotide sequence (5′–3′)	Amplicon size in base pairs (bp)
qnrA	*QnrA –F*	5'- CAGCAAGAGGATTTCTCACG-3'	630 bp
	QnrA -R	3'- AATCCGGCAGCACTATTACTC-5'	
qnrB	*QnrB-F*	5'- GGCTGTCAGTTCTATGATCG -3'	488 bp
	QnrB-R	3'- GAGCAACGATGCCTGGTAG -5'	
qnrS	*qnrS –F*	5'- GCAAGTTCATTGAACAGGGT -3'	128 bp
	qnrS –R	3'- TCTAAAC CGTCGAGTTCGGCG -5'	
qepA	QepA -F	5'- GCAGGTCCAGCAGCGGGTAG -3'	218
	QepA -R	3'- CTTCCTGCCCGAGTATCGTG -5'	
oqxA	OqxA -F	5'- CTCGGCGCGATGATGC -3'	392
	OqxA -R	3'- CCACTCTTCACGGGAGACGA -5'	

F= Forward; R= Reverse; bp = Base pair; **Source:** Morteza *et al.* (2022).

2.10. DNA Extraction from Pseudomonas aeruginosa Isolates

All the quinolone-resistant isolates were cultured into Luria Betani broth for 18-24h at 37°C. The plasmid isolation was by the phenol-chloroform method (Shahcheraghi et al., 2009). The extracted DNA was quantified using the Nanodrop 1000 spectrophotometer.

2.11. DNA Amplification by multiplex Polymerase Chain Reaction (PCR) for Plasmid Mediated Quinolones Resistant Genes (QnrA, qnrB, qnrS, qepA, OqxA)

Multiplex polymerase chain reaction (mPCR) was performed using the ABI 9700 Applied Biosystems thermal cycler. A 25 µl reaction was set up containing 2.5 µl MgCl2 (50 mM), 2.5 µl 10 × buffer, 0.9 µl of each primer (2.7 µM), 1 µl of 8 mM dNTPs per reaction, 0.5 µl DNA polymerase and 5 µl of DNA template. The PCR amplification was carried out at the following conditions; pre-denaturation at 95°C for 15 min, denaturation at 95°C for 45 sec., annealing at 63°C for all genes except for oqxA and oqxB (both at 59°C) for 60 sec., elongation at 72°C for 90 sec (33 cycles), and final elongation at 72°C for 10 min. Primer oligonucleotide sequences and amplicon sizes utilised for the amplification of genes are as presented in Table 1. One percent (1%) agarose gel was used in resolving the DNA fragments stained with ethidium bromide and visualized under UV light in a gel-doc system (Hero-Lab with CCD camera attached to it).

3. Results

3.1. Occurrence of Pseudomonas aeruginosa

The occurrence of *P. aeruginosa* from stored water around Karu Metropolis, Nigeria is shown in Table 2. The overall occurrence of the isolates out of 180 water samples collected was 42 (23.3%), New Nyanya had the highest occurrence at 26.6% but lowest in Masaka at 20.0% as shown in Table 2.

Table 2: Occurrence of *Pseudomonas aeruginosa* in stored household water in Karu Metropolis, Nasarawa State, Nigeria.

Areas	No. of Samples	No. of Occurrence in Area (%)
Ado	60	14(23.3%)
Masaka	60	12(20.0%)
New Karu	60	16(26.6%)
Total	180	42(23.3%)

% = Percentage, No = Number

3.2. Resistance of Pseudomonas aeruginosa

The resistance of *P. aeruginosa* to the tested antibiotics from stored water in Karu Metropolis is represented in Table 3. The isolates were highly resistant to cefuroxime (100%), ceftriaxone (100%), cefotaxine (97.6), amoxicillin clavelanate (97.6%), ceftriaxone Sulbactam (95.2%), Nitrofurantoin (92.9%) but showed low resistance to Ofloxacin (4.8%) and Levofloxacin (9.5%).

Table 3: Antimicrobial Resistance of *Pseudomonas aeruginosa* isolated from stored household water in Karu Metropolis, Nasarawa State, Nigeria.

Antimicrobial	Disc Content	No. (%) Resistance (n=42)
Cefuroxime	30 µg	42(100%)
Cefotaxime	25 µg	41(97.6)
Imipenem/Cilastatin	10/ 10	31(73.8%)
Ofloxacin	5 µg	2(4.8%)
Gentamicin	10 µg	22(52%)
Levofloxacin	5 µg	4(9.5)
Ceftriaxone Sulbactam	45 µg	40(95.2)
Amoxicillin Clavulanate	30 µg	41(97.6%)
Cefexime	5 µg	42(100%)
Nitrofurantoin	30µg	39(92.9%)

% = Percentage, n= Number of sample, No. = Number

3.3. Antimicrobial Resistance Phenotypes

The antimicrobial resistance phenotypes of the resistant isolates from stored water are as shown in Table 3.4. Antimicrobial-resistant isolates were distributed into different categories as antimicrobial resistance phenotypes and the most common phenotypes were CXM-CTX-IMP-GN-CRO-AUG-ZEM-NF (Cefuroxime (CXM), Cefotaxime (CTX), Imipene/Cilastatin (IMP), Gentamycin (GN), Amoxicillin Clavulanate (AUG), Ceftriaxone Sulbactam (CRO), Cefexime (ZEM), Nitrofurantoin (NF) (38.1%) and CXM-CTX-IMP-CRO-AUG-ZEM-NF (Cefuroxime (CXM), Cefotaxime (CTX), Imipene/Cilastatin (IMP), Ceftriaxone Sulbactam (CRO), Amoxicillin Clavulanate (AUG), Cefexime (ZEM), Nitrofurantoin (NF) (19.0%).

Table 4. Antimicrobial Resistance Phenotypes of Resistant *P. aeruginosa* from stored household water in Karu Metropolis, Nasarawa State, Nigeria.

Antimicrobial Resistance Profile Phenotypes	Frequency (%) (n=42)
CXM, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, AUG, ZEM, NF	1(2.4)
CXM, CTX, CRO, ZEM, NF	1(2.4)
CXM, CTX, CRO, AUG, ZEM	1(2.4)
CXM, CTX, IMP, CRO, AUG, ZEM	1(2.4)
CXM, CTX, IMP, AUG, ZEM, NF	1(2.4)
CXM, CTX, CRO, AUG, ZEM, NF	4(9.5)
CXM, CTX, LBC, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, GN, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, IMP, CRO, AUG, ZEM, NF	8(19.0)
CXM, CTX, IMP, LBC, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, IMP, GN, CRO, AUG, ZEM, NF	16(38.1)
CXM, CTX, GN, LBC, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, IMP, GN, LBC, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, IMP, OFX, GN, CRO, AUG, ZEM, NF	1(2.4)
CXM, CTX, IMP, OFX, GN, LBC, CRO, AUG, ZEM, NF	2(4.8)

Nitrofurantoin (NF), Cefuroxime (CXM), Ceftriaxone Sulbactam (CRO), Cefexime (ZEM), Levofloxacin (LBC), Amoxicillin Clavulanate (AUG), Cefotaxime (CTX), Imipene/Cilastatin (IMP), Ofloxacin (OFX), Gentamycin (GN), % = Percentage, n= number of samples % = Percentage, n= number of sample

3.4. Multiple Antimicrobial Resistance (MAR) Index

The MAR index of antimicrobial resistance isolates from stored water is shown in Table 5. All the isolates were MAR isolates with MAR index of >0.5 and the most common MAR Index in the isolates was 0.8(42.9%) and the-lest common were 1.0(4.7%) and 0.9(4.7%).

Table 5. Categories of Antimicrobial Resistance in Antimicrobial Resistant Pseudomonas aeruginosa from stored household water in Karu Metropolis, Nasarawa State, Nigeria.

Categories of Antimicrobial Resistance	No. (%) *P. aeruginosa* isolates n= 42
MDR	40(95.3)
XDR	0(0.0)
PDR	2(4.7)

MDR= Multi-drug resistance, XDR= Extended drug resistance, PDR: Pan drug resistance, % = Percentage, No. = Number, n= Number of samples

3.5. Categories of Antimicrobial Resistance

The resistances of antimicrobial-resistant isolates from stored water were classified into different categories of antimicrobial resistance where 95.3% of isolates were Multiple Drug Resistance which was the highest, none was Extended Drug Resistance and 4.7% was Pan Drug Resistance. (Table 6)

Table 6: Multiple Antimicrobial Resistance (MAR) Index of antibiotic resistant *Pseudomonas aeruginosa* isolated from stored household water in Karu Metropolis, Nasarawa State, Nigeria.

No. of Antimicrobial Resistance (a)	No. Antimicrobial Tested (b)	MAR Index (a/b)	Frequency (%) (n=42)
10	10	1.0	2(4.7)
9	10	0.9	2(4.7)
8	10	0.8	18(42.9)
7	10	0.7	10(23.8)
6	10	0.6	6(14.3)
5	10	0.5	4(9.5)
4	10	0.4	0
3	10	0.3	0
2	10	0.2	0
1	10	0.1	0

3.6. Expression of Plasmid Mediated Quinolones Resistance (PMQR) Genes in the Isolates

There was amplification of *qnrS* and *qepA* PMQR genes (Figure 1) from *P. aeruginosa* in this study and the prevalence of was 66.7% for *qnrS* and 23.3% for *qepA* but *qnrA, qnrB, oqxA* were not amplified (Table 7). The agarose gel elecphoresis is shown in Figure 1 below which shows the presence of *qnrS* (128bp) gene at lane 1 and lane 2, qepA gene which is the lane 3 at 218bp and the molecular ladder at Lane M (1500bp). The results reveal the presence of plasmids mediated quinolone resistant genes in some *Pseudomonas aeruginosa* at Karu Metropolis.

Table 7. Occurrence of Plasmids Mediated Genes in Quinolone resistant *Pseudomonas aeruginosa* isolated from stored household water in Karu Metropolis, Nasarawa State, Nigeria.

Target Gene	Percentage of Occurrence
QnrA	0%
QnrB	0%
QnrS	66.7%
QepA	23.3%
OqxA	0%

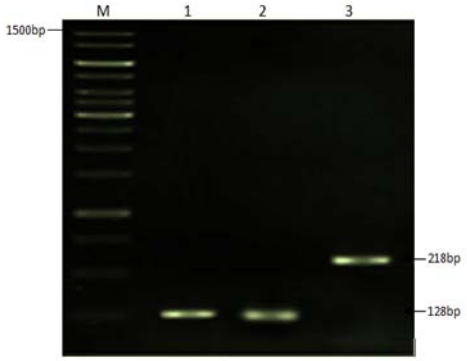

Figure 1. Amplified plasmid-mediated quinolones resistance genes with band sizes of 218bp (QnrS) and 128bp (*qepA*). Lane M: DNA ladder (1500bp), Lane 1: (*qepA*), Lane 2: (*qepA*), and Lane 3: (QnrS).

4. Discussion

Water is essential for man's daily activities and in developing countries where water has to be stored in tanks to mitigate the effect of water shortages. There are high chances of this water being contaminated with bacteria such as *P. aeruginosa*. The presence of *P. aeruginosa* can easily cause a wide range of infections, especially in immunocompromised individuals.

The isolation of *P. aeruginosa* from stored water in tanks at Karu Metropolis is consistent with the findings of Chegini (2020), Collins *et al.* (2022), Schiavano *et al.* (2017), Wei *et al.* (2020), Stanley (2017), Nrior *et al.* (2020) and Morteza *et al.* (2022) which were found to be reoccurring microbes, from different water sources. This is also a confirmation that *P. aeruginosa* can survive in a nutrient-deficient environment. The percentage of occurrence of *P. aeruginosa* in this study was 23.3%; this is similar as well to earlier research conducted by Lei *et al.* (2020) and Stanley *et al*. (2017), both of whom reported an occurrence of 22.5%. Aromolaran and Amodu (2021) also reported an occurrence of 21.43%, significantly lower than 85.11% reported by Collins *et al.* (2022).

The high resistance of the isolates to cefuroxime (100%), cefexime (100%), cefotaxime (97.6%), amoxicillin/Cluvalanate (97.6), ceftriaxone/Sulbactam (95.2%), imipenem/celastatin (73.8%), and nitrofurantoin (92.9%) in this study may be due to their inappropriate use for treatment of infection caused by *P. aeruginosa.*

The high resistance of isolates to imipenem/cilastatin in our study was slightly higher than 69.2% as reported by Schiavano *et al.* (2017). The resistance of the isolates to β-lactams antimicrobial agent such cefuroxime, ceftriaxone/Sulbactam and imipenem may be due to production of β –lactamase. The percentage of resistance to amoxicillin (97.6 %) and cefexime (95.2%) in our study was higher than 75% resistant to amoxicillin and cefexime in the study conducted by Nrior *et al.* (2020).

Our findings also showed that the occurrence of multidrug-resistant isolates was higher than Pan-drug resistance isolates and the percentage of occurrence of MDR (Multiple Drug Resistance) isolates was 95.3%; this is higher than 1.88% and 28.8% in the studies reported by Schiavano *et al.* (2017) and Nikola *et al.* (2022), respectively. The occurrence of MDR isolates in our study may have public health implications like prolonged hospital stay, high cost of treatment, less available drugs for treatment and also an increase in morbidity by diseases caused as a result of bacterial infection.

The low resistance and high susceptibility of isolates to levofloxacin and ofloxacin in this study is an indication that such antimicrobial agents may not have been misused or abused in the study area. The low resistance of the isolates to quinolones mentioned is also an indication that the agent may be effective for the treatment of *P. aeruginosa* infection. The low percentage of resistance of the isolates (4.8%) to ofloxacin in this study contradicts with the findings of Morteza *et al.* (2022), Nrior *et al.* (2020) who reported 45.9% and 50%, respectively.

The detection of *qnrS* and *qepA* genes in levofloxacin and ofloxacin resistant isolates in our study is an indication that they may be responsible for the resistance. The percentage of occurrence of *qnrS* in our studies was 66.6%; this is higher than 20.8% and 10.5% reported by Morteza *et al.* (2022) and Venkataramana *et al.* (2022). Similarly, the occurrence of *qepA* gene in this study was in agreement with the study conducted by Venkataramana *et al.* (2022). Qnr genes protect the target of quinolone antibiotics called DNA gyrase and thus make bacteria confer resistance, thus the expression of this gene in the isolates used in this study could be the reason why quinolones antibiotic is not effective in the treatment of bacterial infection.

The presence of *qepA* in the isolates is an indication that the mechanisms of resistance might be due to increase in efflux pumping of the quinolones antimicrobial agents. The following genes *qnrA, qnrB*, oqxA were not detected in the isolates. In a study by Wei *et al.* (2020), they observed no resistance to quinolones antibiotics in contrast to the observation from our study. The presence of these genes in the study area is of great concern, as these isolates can serve as a reservoir for resistance genes, and these genes can be passed on to other organisms which will result in multidrug resistance.

5. Conclusion

P. aeruginosa was isolated from stored water, and most of the isolates are resistant to the common antibiotics used in the treatment of bacterial infection in Nigeria. These isolates harbor plasmids mediated quinolones resistance genes (Qnr and qepA) which can be spread within the area. The finding from this study is of great concern to public health as these resistant genes can be transferred to other bacteria of same or closely related species. The high multidrug resistance pattern of the isolates might be due to overuse, self-medication and also non-adherence to prescription. Antimicrobial resistance has reduced the number of antimicrobial agents available in the market for treatment of bacterial infection.

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Conflict of Interests

All the authors declare that there is no conflict of interests.

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