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Research Article

COMPARATIVE STUDY OF PREVALENCE, ANTIBIOGRAM PATTERN AND PLASMID PROFILING OF *Pseudomonas Aeruginosa* AND *Escherichia coli* FROM CLINICAL AND ENVIRONMENTAL SOURCES

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Abstract

Pseudomonas aeruginosa and *Escherichia coli* are both gram negative bacteria with an incriminating epidemiology in common and vast majority of human pathogenic infections; coupled with their relatively high resistance to several antibiotics used for their treatment management. This study aimed at determining the prevalence of *Pseudomonas aeruginosa* and *Escherichia coli* from both clinical and environmental sources, and their antibiotic susceptibility pattern in relation to their plasmid profile, using standard microbiological methods. A total of 100 samples of both clinical and environmental origin were collected; ranging from urine, wound swab, water and soil. Result obtained revealed variation in the percentage prevalence of *Pseudomonas aeruginosa* and *Escherichia coli* across clinical and environmental samples; with *Escherichia coli* recording a higher percentage prevalence of 55% and 57.10% in urine and wound swab respectively for clinical samples, while *Pseudomonas aeruginosa* had a higher percentage prevalence of 62.50% in water for environmental sample. Overall, while *Escherichia coli* had a higher percentage prevalence of 55.55% in clinical samples, *Pseudomonas aeruginosa* recorded a higher percentage prevalence of 56.25% in environmental samples. Clinical isolates showed higher overall sensitivity to antibiotics compared to environmental isolates before plasmid curing. Post-curing, much of the resistance in clinical *Escherichia coli* was plasmid-mediated, while *Pseudomonas aeruginosa* exhibited predominantly chromosomal-mediated resistance. Environmental isolates demonstrated a more alarming resistance profile, especially in *Escherichia coli*, which was resistant to all tested antibiotics before curing, but with an improved sensitivity post-curing. Plasmid-mediated resistance was found to be the primary driver of multidrug resistance in environmental isolates and clinical *Escherichia coli*, while chromosomal-mediated resistance was more prominent in clinical *Pseudomonas aeruginosa* isolates. This study underscores the need for continuous monitoring of resistance patterns and targeted interventions to safeguard public health. Further research on genetic mechanisms of resistance and exploring alternative treatment options to enhance infection management strategies is recommended.

Keywords: *Pseudomonas aeruginosa*, *Escherichia coli,* clinical, environmental, plasmid.

INTRODUCTION

Human opportunistic pathogens like *Pseudomonas aeruginosa* and *Escherichia coli* are ubiquitous environmental bacteria that causes numerous opportunistic human infections. The emerging presence of multi-drug-resistant isolates to many antimicrobials used for hospital patients has attracted the attention of many researchers in recent decades (Morris and Cerceo, 2020). A wide range of biochemical and physiological mechanisms may be responsible for resistance. *Pseudomonas aeruginosa* is a leading cause of hospital-acquired infections, giving rise to a wide range of opportunistic infections. Its high intrinsic resistance to antibiotics and ability to develop multidrug resistance pose serious therapeutic problems (Kunz-Coyne *et al.,* 2022). *Pseudomonas aeruginosa* is highly ubiquitous in water systems, and has intrinsic antimicrobial resistance due to low outer membrane permeability, as well as an extensive efflux pump system (Lorusso *et al.,* 2022).

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Pseudomonas aeruginosa belongs to a vast genus of obligate aerobic, non-fermenting, saprophytic, Gram-negative bacilli widespread in nature, particularly in moist environments (Ntanda, 2024). The organism has been incriminated in cases of meningitis, septicaemia, pneumonia, ocular and burn infections, hot tubs and whirlpool-associated folliculitis, osteomyelitis, cystic fibrosis-related lung infection, malignant external otitis and urinary tract infections with colonized patients being an important reservoir (Mena and Gerba, 2009). Cross-transmission from patient to patient may occur via the hands of the health care staff or through contaminated materials and reagents (Dwivedi *et al.,* 2009). *Escherichia coli*, a Gram-negative bacillus is the most prevalent facultative anaerobic species in the gastrointestinal tract of humans and animals, usually a harmless microbe, but it is also a medically important bacteria causing a number of significant illnesses (Basavaraju and Gunashree, 2022). Vegetables may be contaminated through insufficiently-treated water and fertilizers or may be compromised by the use of biocides during cultivation (EFSA, 2014). Similarly, animals can also become infected from water or food contaminated with wastes of human or animal origin or with human carrier workers.

Most *Escherichia coli* are harmless and actually are an important part of a healthy human intestinal tract. However, some *Escherichia coli* are pathogenic by either causing diarrhea or illness outside of the intestinal tract. The types of *Escherichia coli* that can cause diarrhea can be transmitted through contaminated water or food, or through contact with animals or persons (CDC, 2003). Antibiotic resistance in *Escherichia coli* has been globally identified in isolates from environmental, animal and human sources (Pormohammad *et al.,* 2019). This is a consequence of the use of antimicrobials in medicine and their application in animal husbandry, which have brought about phenotypic changes, often due to chromosomal mutations (Erb *et al*., 2007) . Several studies have revealed that *Escherichia coli* is resistant to a number of antibiotics (Von and Reinhard, 2000; Orrett and Shurl, 2001; Iqbal and Patel, 2002; Al-Tawfiq, 2006; Pormohammad *et al.,* 2019).

MATERIALS AND METHODS

Sample collection

A total of one hundred (100) samples which comprises urine (25), wound swab (25), soil (25) and water (25) were collected. Urine and water samples were aseptically collected into universal sterile containers. Wound specimens were collected with sterile swab sticks and soil samples were collected with sterile containers from the top 5-10cm layer.

Sample processing

Urine: The samples were mixed thoroughly by inverting the containers several times. Using a sterile wire loop, the samples were inoculated on MacConkey agar and Nutrient agar. The plates were incubated at 37ºC for 24h. Distinct colonies were sub-cultured on nutrient agar repeatedly to obtain pure cultures. The isolates were stored on nutrient agar slants for identification.

Wound: The wound swabs collected with sterile swab sticks were suspended in normal saline for 30 minutes after which they were inoculated on MacConkey agar and Nutrient agar plates. The plates were incubated at 37ºC for 24h. Distinct colonies were sub-cultured on nutrient agar repeatedly to obtain pure cultures. The isolates were stored on nutrient agar slants for identification.

Water: A three-fold serial dilution method was used with sterile distilled water in a test tube. Diluents were plated out on Nutrient Agar and MacConkey agar. The plates were incubated at 37ºC for 24 h. Discrete colonies were picked from the agar plates based on size and colour of colonies and sub-cultured on nutrient agar to obtain pure cultures. The isolates were stored on nutrient agar slants for identification.

Soil: Ten grams (10g) of soil sample was weighed into 90ml of sterile distilled water in a cornical flask and shaken for some minutes to dissolve completely. The resulting suspension was allowed to settle after which serial dilution was carried out. Diluents were plated out on Nutrient Agar and incubated at 37ºC for 24h. Discrete colonies were picked from the agar plates based on size and colour and sub-cultured on nutrient agar to obtain pure cultures. The isolates were stored on nutrient agar slants for identification.

Identification of the Isolates

All isolates were Gram stained and examined microscopically. Biochemical tests were carried out based on Gram reactions. (Cheesbrough, 2000).

Antibiotic Susceptibility Test

Antibiotic susceptibility testing of the isolates was carried out using Kirby-Bauer disc diffusion technique which was adopted from Amengialue *et. al.* (2013) in accordance with National Committee for Clinical Laboratory Standards (NCCLS) guideline. The following antibiotics were used: augmentin (30µg), gentamycin (10µg), pefloxacin (30µg), streptomycin (30µg), chloramphenicol (30µg), sparfloxacin (10µg), ciprofloxacin (10µg), amoxicillin (30µg) and septrin (30µg). Results were recorded by measuring the zone of inhibition and comparing with the CLSI susceptibility testing (CLSI, 2015).

Plasmid Curing

Isolates were subjected to standard plasmid curing method. An aliquot of overnight culture was inoculated into 9mL of freshly prepared nutrient broth. The nutrient broth was incubated for 4 hours for minimal growth of microorganisms. After 4 hours, 1ml of sodium deodecylsulphate (SDS) curing agent was added sufficiently to the mixture to bring the concentration to 1%. It was incubated for 24 hours at 37oC. One milliliter of the cured culture was added into 9ml of freshly prepared nutrient broth and was incubated for another 24 hours at 37oC. Post Plasmid Antimicrobial Susceptibility was carried out as described by (Ehiaghe *et al*.2022).

Statistical Analysis

Data obtained from this research was analyzed using the Statistical Package for Social Sciences (SPSS) version 22.0 (IBM Inc., USA). The results were presented in percentage bar chat and heatmap

RESULTS

Figure 1 shows the percentage distribution of *Escherichia coli* and *Pseudomonas aeruginosa* across all samples used in this study. From urine samples, *Escherichia coli* was higher, amounting to 55% of the total bacteria isolates, while *Pseudomonas aeruginosa* constituted the remaining 45%. In wound swab samples, *Escherichia coli* had a slightly higher occurrence of 57.1% compared to *Pseudomonas aeruginosa* which was 42.9%. In water samples, there was a shift in pattern of occurrence as *Pseudomonas aeruginosa was* predominant with 62.5% while *Escherichia coli* accounted for 37.5% of the bacteria isolates. Soil samples had equal percentage occurrence of 50% each for *Escherichia coli* and *Pseudomonas aeruginosa*. This result underscores how the relative abundance of these bacteria varies by sample type, potentially reflecting differences in environmental niches and bacterial adaptation. Figure 2 compares the frequency occurrence of *Escherichia coli* and *Pseudomonas aeruginosa* between clinical and environmental samples. *Escherichia coli* was more frequently isolated from clinical samples, constituting 55.55% of the total isolates, while *Pseudomonas aeruginosa* accounted for 44.44%. In contrast, *Pseudomonas aeruginosa* was more in environmental samples, making up 56.25% of the isolates while *Escherichia coli* had 43.75%.

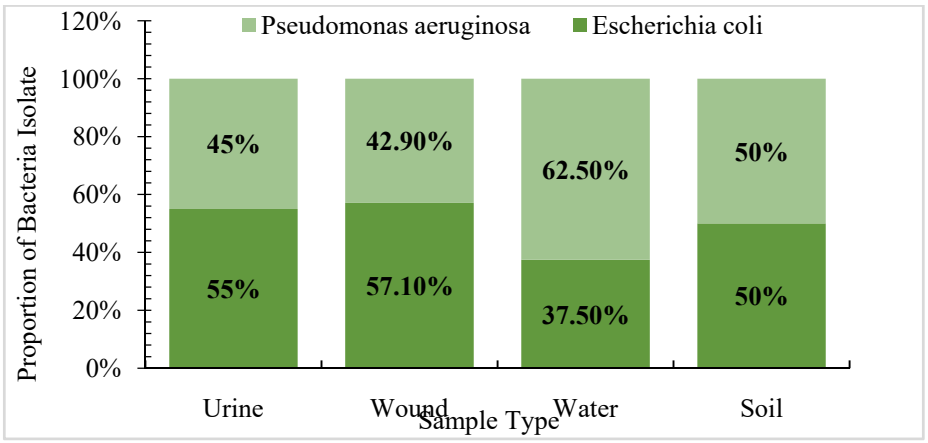

Figure 1. Percentage proportion of *Escherichia coli* and *Pseudomonas aeruginosa* in different sample types

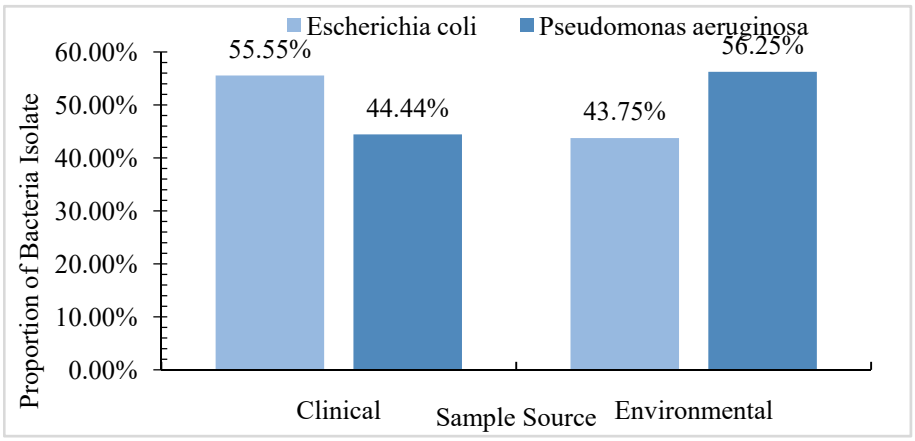

Figure 2. Percentage proportion of *Escherichia coli* and *Pseudomonas aeruginosa* in clinical and environmental sources

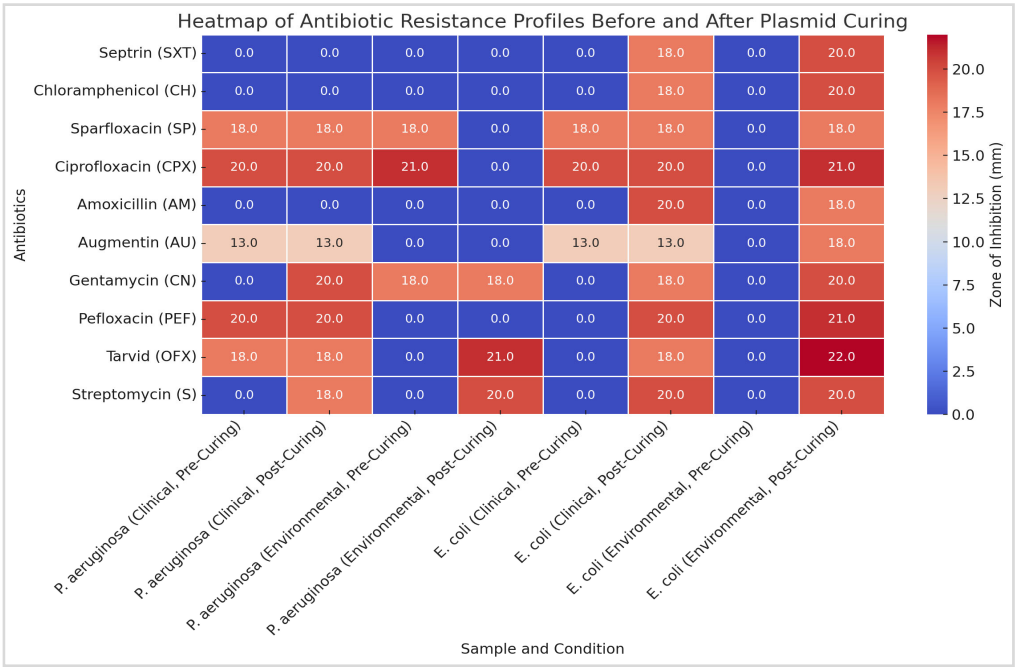

Figure 3. Comparison of antibiotic resistance profiles before and after Plasmid curing between clinical and environmental isolate

This data reveals a clear distinction in bacterial prevalence based on the sample source. *Escherichia coli* tends to be more prevalent in clinical settings, potentially indicating its role in human infections. Conversely, *Pseudomonas aeruginosa* shows a higher occurrence in environmental samples, likely due to its adaptability and survival in diverse ecological conditions.

Figure 3 provides a comprehensive result of antibiotic resistance profiles in *Pseudomonas aeruginosa* and *Escherichia coli* from both clinical and environmental samples, analyzed before and after plasmid curing. The size of the zone of inhibition (measured in mm) reflects the sensitivity of bacteria isolates to different antibiotics, where larger zones indicate increased sensitivity. This data is critical for

understanding the mechanisms of resistance and the role of plasmids in antimicrobial resistance.

DISCUSSION

The empirical findings of this research provide significant insight into the prevalence, antibiotics susceptibility pattern and plasmid profile of *Pseudomonas aeruginosa* and *Escherichia coli* in respect to their sources. Result findings in figure 1 shows variation in the percentage prevalence of *Pseudomonas aeruginosa* and *Escherichia coli* across clinical and environmental samples, with *Escherichia coli* recording a higher percentage prevalence of 55% and 57.10% in urine and wound swab respectively for clinical samples, while *Pseudomonas aeruginosa* had a higher percentage prevalence of 62.50% in environmental sample (water). In all, while *Escherichia coli* had a higher percentage prevalence of 55.55% in clinical samples, *Pseudomonas aeruginosa* recorded a higher percentage prevalence of 56.25% in environmental samples (see figure 2). The prevalence of *Escherichia coli* in urine sample, being the highest in this study, corroborated the findings of Tenaillon *et al*. (2010), Anyadoh-Nwadike *et al.* (2015) and Ehiaghe *et al.* (2020) who reported *Escherichia coli* as the most dominant pathogen in urine associated with urogenital infection. Meanwhile, Ehiaghe *et al.,* 2025 and Ehiaghe *et al.,* 2022 reported *Pseudomonas aeruginosa* to be more prevalent in wound sample; which is in contrast with the finding of this study that showed *Escherichia coli* to be more prevalent in wound sample. The higher prevalence of *Pseudomonas aeruginosa* in water sample aligns with the finding of Malamattathil *et al*., 2014 who reported *Pseudomonas aeruginosa* as the most prevalent environmental bacteria solated from surface and drinking water.

Prior to plasmid curing, clinical isolates of *Pseudomonas aeruginosa* showed resistance against several antibiotics, including septrin, chloramphenicol, amoxicillin, gentamycin and streptomycin, as indicated by a 0.0mm zone of inhibition. However, intermediate resistance was observed for augmentin with a zone of 13.0mm. Notably, these isolates remained sensitive to sparfloxacin, ciprofloxacin, pefloxacin and tarvid, with zones of inhibition ranging from 18.0 - 20.0mm. Meanwhile, the results were slightly different in clinical *Escherichia coli* isolates, showing sensitivity or intermediate resistance to fewer antibiotics such as sparfloxacin, ciprofloxacin and augmentin. Furthermore, the environmental isolates of *Pseudomonas aeruginosa* exhibited a resistance profile similar to their clinical counterparts for septrin, chloramphenicol, amoxicillin, pefloxacin, tarvid, and streptomycin. However, sensitivity was retained for ciprofloxacin, sparfloxacin, and gentamycin, demonstrating a slight difference in resistance patterns between clinical and environmental strains. In contrast, environmental *Escherichia coli* isolates were markedly resistant, showing no sensitivity to any tested antibiotics. This high resistance, even to sparfloxacin and ciprofloxacin, which were effective against clinical strains, underscores the enhanced multidrug-resistant nature of environmental *Escherichia coli* isolates. These findings are similar to the result of Manikandan *et al*. (2011) who reported multidrug resistance by bacteria isolated from urinary tract infection, as well as the work of Gales *et al*. (2001) who reported presence of multidrug resistance in *Pseudomonas aeruginosa*. Harbottle *et al*. (2006) reported that the overuse of antibiotics has become the major factor for the emergence and dissemination of multi-antibiotics resistance strain of several bacteria; with Gehan *et al*. (2011) reporting complete resistance to amoxacillin by *Pseudomonas aeruginosa*. However, result from this work is in contrast with the findings of Shahid and Malik (2005) who reported that clinical isolates were more resistant to antibiotics than environmental isolates. Moreover, it is known that many antibiotic resistance genes reside in plasmids which facilitate their transfer (Nikaido, 2009) and thus, these suggest that *Pseudomonas aeruginosa* and *Escherichia coli* strains from clinical and environmental sources in this study may have develop a resistant gene.

Plasmid curing of the isolates resulted in notable changes in resistance profiles, enabling differentiation between plasmid-mediated and chromosomal-mediated resistance mechanisms. In clinical *Pseudomonas aeruginosa* isolates, resistance to septrin, chloramphenicol, and amoxicillin remained unchanged (0.0 mm), confirming that this resistance was chromosomal-mediated. Sensitivity to sparfloxacin, ciprofloxacin, and pefloxacin was also retained, suggesting these responses were not plasmid-dependent. This observation is similar to the findings of Anyiam and Idegbe, 2024 who reported continued resistance in *Pseudomonas aeruginosa* post-curing. However, gentamycin and streptomycin showed increased zones of inhibition (up to 20.0mm and 18.0mm, respectively) post-curing, indicating that their resistance was plasmid-mediated. For clinical *Escherichia coli*, significant shifts in sensitivity were observed after curing, corroborating the findings of Ehiaghe *et al*. (2020). Antibiotics such as septrin (18.0mm), chloramphenicol (18.0mm), and amoxicillin (20.0mm), which previously demonstrated resistance, became effective. This suggests that much of the resistance in *Escherichia coli* was plasmid-mediated. In contrast, ciprofloxacin and augmentin retained their original zones of inhibition, confirming chromosomal-mediated resistance.

Environmental *Pseudomonas aeruginosa* lost sensitivity to sparfloxacin and ciprofloxacin after curing (zones reduced to 0.0 mm), indicating that sensitivity to these antibiotics was plasmid-mediated. In contrast, gentamycin resistance remained chromosomal, while streptomycin became sensitive post-curing, highlighting plasmid-mediated resistance. Notably, tarvid's sensitivity improved (zones increased to 21.0 mm), further demonstrating the impact of plasmid removal on antibiotic efficacy. Environmental *Escherichia coli* exhibited stark changes after plasmid curing. Sensitivity significantly increased across multiple antibiotics, including septrin (20.0 mm), chloramphenicol (20.0 mm), amoxicillin (18.0 mm), and tarvid (22.0 mm). This strongly indicates that multidrug resistance in environmental *Escherichia coli* is primarily plasmid-mediated. Overall, clinical isolates showed higher sensitivity to antibiotics compared to environmental isolates before plasmid curing. This suggests that clinical isolates might be subject to more stringent antibiotic stewardship practices, limiting their resistance profiles. Post-curing, it became evident that much of the resistance in clinical *Escherichia coli* was plasmid-mediated, while *Pseudomonas aeruginosa* exhibited predominantly chromosomal-mediated resistance. The latter's intrinsic resistance mechanisms pose significant challenges for treatment, requiring alternative therapeutic approaches. Environmental isolates demonstrated a more alarming resistance profile, especially in *Escherichia coli*, which was resistant to all tested antibiotics before curing. This high level of resistance can be attributed to the acquisition of multidrug-resistant plasmids in the environment. Post-

curing sensitivity improvements suggest that horizontal gene transfer in environmental settings plays a significant role in spreading resistance genes. In this study, plasmid-mediated resistance was found to be the primary driver of multidrug resistance in environmental isolates and clinical *Escherichia coli*. Plasmids often carry genes encoding resistance to multiple antibiotics, making infections caused by these strains particularly challenging to treat. The restoration of sensitivity after plasmid curing offers a potential strategy to combat resistance. Chromosomal-mediated resistance was more prominent in clinical *Pseudomonas aeruginosa*, aligning with the observation of Ehiaghe *et al*., 2022 who reported dominant chromosomal resistance mechanisms in *Pseudomonas aeruginosa*. This intrinsic resistance is encoded in the bacterial genome, making it less amenable to eradication and necessitating alternative treatment approaches, such as combination therapies or novel antibiotics. The resistance profiles of environmental *Escherichia coli* pose a significant public health threat, as these strains can act as reservoirs of multidrug-resistant genes. The potential for horizontal transfer of these plasmids to clinical strains underscores the need for monitoring environmental reservoirs and implementing measures to curb the spread of resistance genes.

Conclusion

The results collectively underscore the complex interplay between bacterial distribution patterns and antibiotic resistance mechanisms across clinical and environmental settings. The percentage dominance of *Escherichia coli* in clinical samples and *Pseudomonas aeruginosa* in environmental samples reflects the ecological and physiological niches that these species occupy, highlighting their adaptability to distinct environments. Antibiotic resistance profiling further reveals stark differences between clinical and environmental isolates, with environmental strains, particularly *Escherichia coli*, exhibiting heightened multidrug resistance. Plasmid curing confirm that plasmid-mediated resistance plays a pivotal role in this phenomenon, as demonstrated by the restoration of antibiotic sensitivity in both clinical and environmental isolates post-curing. Conversely, chromosomal-mediated resistance remains prominent in *Pseudomonas aeruginosa*, especially in clinical isolates, posing enduring challenges for treatment due to its intrinsic genetic basis. These findings highlight the dual threat posed by multidrug-resistant bacteria in clinical and environmental settings. On the one hand, clinical isolates demonstrate the critical need for stringent antibiotic stewardship and targeted therapeutic strategies. On the other, environmental isolates act as reservoirs of resistance genes, facilitating horizontal gene transfer and exacerbating the global antimicrobial resistance crisis. Addressing these challenges requires integrated efforts, including continuous monitoring of resistance patterns, interventions to limit plasmid-mediated resistance, and the development of novel treatment approaches tailored to the unique resistance mechanisms of these pathogens.

Conflict of interest: All authors have declared that they have no conflict of interest whatsoever

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