



GENOTYPIC CHARACTERIZATION OF *PSEUDOMONAS AERUGINOSA* FROM POINT-OF-SALES DEVICES IN LOKOJA, NIGERIA

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ABSTRACT

The presence of *Pseudomonas aeruginosa* on shared frequently used surfaces, such as Point-of-Sales (POS) devices present serious public health problem. This study assessed *P. aeruginosa* on POS devices frequently used within Lokoja metropolis, with the aim of determining the presence of antibiotic resistance and virulence genes in the *P. aeruginosa*. Following standard procedures, Seventy-two POS devices were randomly sampled using sterile swabs soaked in peptone water and cultured on sterile agar plates. Biochemical characterization of isolates was carried out while antibiotics susceptibility testing was carried out using the Kirby-Bauer disc diffusion method. Some resistance and virulence genes of *P. aeruginosa* were detected using polymerase chain reaction (PCR) assay. Bacterial isolates were characterized as *Staphylococcus aureus* (32.4%), *Klebsiella pneumoniae* (21.9%), *Pseudomonas aeruginosa* (17.2%), *Escherichia coli* (14.2%), *Bacillus subtilis* (10.1%), *Enterobacter* spp. (2.4 %) and *Enterococcus* sp. (1.8%). *Pseudomonas aeruginosa* isolates displayed antibiotic resistance against tetracyclines (58.6%), followed by ceftazidime (51.7%), gentamicin (48.3%), cefepime (48.3%), ceftriaxone (48.3%), cefoxitin (44.8%), levofloxacin (41.2%), ciprofloxacin (37.9%), amoxicillin-clavulanic acid (37.9%) and imipenem (24.1%). For the virulence genes, 72.4%, 38%, 28% of the *Pseudomonas aeruginosa* strains were positive for *OprL*, *toxA* and *lasB*, respectively while for the antibiotic resistance genes, 14% were positive for *blaVIM* and 10.3% for *blaIMP*. This study reveals the presence of pathogenic *P. aeruginosa* on POS devices with their virulence genes and antibiotics resistance genes. This emphasizes the health threat associated with shared surfaces such as the POS devices. Thus, users must maintain sanitary and hygienic practices while using these devices.

Keywords: Radiation, Radio-frequency, Smartphones, Spectrum Analyzer, Human health

INTRODUCTION

Over the last decade, there has been an increasing use of “cashless devices” to ease financial transactions that reduces the flow of cash (Aduku *et al.*, 2025). These cashless devices have been widely adopted in Nigeria since the approval of the cashless policy by the Central Bank of Nigeria in 2012 (Mamudu and Gayovwi, 2019). Since then, the point-of-sales (POS) devices have been used to ease daily transactions among retailers and consumers, as well as creating job opportunities, amongst other benefits (Rizvi *et al.*, 2020; Uriawan *et al.*, 2024).

Notably, the increased use of shared electronic devices such as keyboards, mobile phones, automated teller machines, POS etc. has resulted in a widespread spread of pathogenic bacteria, particularly, Gram-negative bacteria (Dawodu and Akanbi, 2021; Innocent-Adiele *et al.*, 2023; Mostafa *et al.*, 2024). Over the years, Gram-negative bacteria have been of interest because they are associated with hospital-borne infections as well as antibiotics resistance (Li *et al.*, 2015). Moreover, most Gram negative bacteria have evolved mechanisms that make them successful in causing diseases in humans (Kaye and Pogue, 2015; Lepe and Martínez-Martínez, 2022).

Pseudomonas aeruginosa is a Gram-negative bacteria that has been more frequently associated with nosocomial infections (Reynolds and Kollef, 2021) and is able to survive on surfaces due to innate, adaptive and acquired mechanisms that they possess (Yang *et al.*, 2024). Specifically, these mechanisms have enabled *Pseudomonas aeruginosa* to exhibit multidrug resistance through efflux pumping out of antibiotics (Rocha *et al.*, 2019), the production of hydrolytic enzymes encoded by genes often borne on plasmids (Horcajada *et al.*, 2019),

and their ability to form biofilms (Fernández-Billón *et al.*, 2023).

These mechanisms exhibited by *Pseudomonas aeruginosa* have made them successful in colonizing surfaces, causing diseases, and spreading antibiotics resistance genes within the environment (Qin *et al.*, 2022); thus, making them a serious public health threat. For example, studies have reported the presence of multidrug resistant *Pseudomonas aeruginosa* in various environments such as tissues, mucosal surfaces, etc. (Manner *et al.*, 2023) and inanimate surfaces such as contact lens' surfaces as well as other surfaces (Wu *et al.*, 2015; Porter *et al.*, 2024). Hence, the presence of *Pseudomonas aeruginosa* on shared contact surfaces could potentially spread antibiotic-resistance genes and limit the efficacy of antibiotics, and facilitate horizontal transfer of antibiotic-resistance genes (Urban-Chmiel *et al.*, 2022; Gauba and Rahman, 2023). Therefore, POS devices could act as reservoirs for the spread of *Pseudomonas aeruginosa*, since it is shared by many users with differing health status and hygienic conditions. Hence, this study aims to isolate and genotypically identify *Pseudomonas aeruginosa* from POS devices. This study will also provide useful insights to the hygienic condition of POS devices as well as the users of POS devices.

MATERIALS AND METHODS

Study Site and Sampling Area

Samples were collected from Lokoja local government area of Kogi State, Nigeria. The POS devices were randomly sampled from the major areas of Felele, Adankolo, Crusher, Phase two, and Army barracks axes.

Collection of Samples

Seventy-two samples were collected from the keypads and screens of the POS devices with sterile cotton swabs soaked in peptone water from 18 locations, 4 samples from each location within Lokoja metropolis, samples were stored inside an ice bag and transported to the Biological Sciences Lab within 1 hour of sample collection. Sample locations were grouped into three. Zone 1 (Lokongoma, Ganaja junction, Dunamis, Old Poly Quarters, 200 housing unit, 500 housing unit), Zone 2 (FUL, FTH, Old market, International Market, Nataco, KSP), Zone 3 (Phase 2, Army signal, Zone 8, Barracks, Shetima, Zango).

Isolation and Biochemical Characterization of Bacteria

The peptone-soaked swab sticks from the sampling points were streaked on the surface of sterile culture plates of Nutrient agar, Mac Conkey agar and Cetrimide agar. The plates were inverted and incubated for 24 hours at 37°C. Distinct colonies were observed and further sub-cultured by streaking on freshly prepared nutrient agar to obtain pure culture. The pure strains were subjected to Gram's reaction and biochemical tests such as, catalase, oxidase, methyl red, indole, citrate, lactose fermentation test, hydrogen sulfide production test, Voges-Proskauer test, nitrate, urease, motility test and Starch hydrolysis.

Antimicrobial Susceptibility Testing

The antibiotic susceptibility testing was performed by the Kirby—Bauer Disc Diffusion method as modified by the Clinical and Laboratory Standards Institute (CLSI, 2018). The following antibiotic disks (Oxoid Ltd., Basingstoke, Hampshire, UK) were used: imipenem (10 µg), gentamicin(10 µg), levofloxacin(5 µg), tetracycline (30 µg), amoxicillin-clavulanic acid (30 µg), ciprofloxacin (5 µg), ceftazidime (30 µg), ceftaxitin (30 µg), ceftriaxone (30 µg),

and cefepime (30 µg), were tested on each isolates (Chand et al., 2021). All plates were incubated at 37°C for 24 h. The diameters of inhibition zones were measured to the nearest millimeter using a ruler.

Detection of Antimicrobial Resistance and Virulence Determinants

DNA molecules were extracted by boiling method (Mentasti et al., 2019) and used to prepare the PCR reaction mixture, each containing 20 µl reaction volume: 2 µl of 10X buffer, 1 µl MgCl₂, 0.8 µl dNTPs, 0.5 µl of forward primer, 0.5 µl of reverse primer, 0.2 µl Taq polymerase, 13 µl of nuclease free water and 2 µl of DNA lysate. The PCR vial was placed in the PCR machine (Prime Thermal Cycler, UK) and it was subjected to the conditions of temperature for denaturation, annealing and elongation at different time intervals according to the type of primer sequence being used for a particular gene amplification.

Amplification was carried under the following conditions; pre-amplification at 94°C for 3 min, 35 cycles of amplification with each consisting denaturation at 94°C for 30 s, primer annealing temperature at 57°C for 60 s (*oprL*, *toxA*, and *lasB*), 54°C for *bla_{IMP}*, and *bla_{VIM}* and primer extension at 72°C for 60 s and a single step of final extension at 72°C for 7 min.

All isolates were analyzed for the presence of *bla_{IMP}*, *bla_{VIM}*, *oprL*, *toxA*, and *lasB* genes (Table 1). At the completion of the amplification, PCR products were resolved in 1.2% agarose gel pre-stained with ethidium bromide (50 µg/ml) in 1x concentrated TAE buffer (40 mM Tris-HCl, 2 mM acetate, 1 mM EDTA, pH 8.3). The DNA bands were visualized and photographed using a gel bio-imaging system (UVP Imaging System, Upland, CA, USA). The type-specific PCR products were recognized clearly by their distinct band sizes.

Table 1: Primers used for the Amplification of Genes

Genes	Primers	Sequences	Sizes	References
<i>oprL</i>	<i>oprL</i> -F	5-ATGGAAATGCTGAAATTCGGC-3	500 bp	(Adenipekun et al., 2023)
	<i>oprL</i> -R	5'-CTTCTTCAGCTCGACGCGACG-3		
<i>toxA</i>	<i>toxA</i> -F	5'-GGTAACCAGCTCAGCCACAT-3'	352 bp	(Adenipekun et al., 2023)
	<i>toxA</i> -R	5'-TGATGTCCAGGTCATGCTTC-3'		
<i>lasB</i>	<i>lasB</i> -F	5'-GGAATGAACGAAGCGTTCTC-3	300 bp	(Adenipekun et al., 2023)
	<i>lasB</i> -R	5'-GGTCCAGTAGTAGCGGTTGG-3'		
VIM	VIM2004A	GTTTGGTCGCATATCGCAAC	390	(Mendes et al., 2007)
	VIM2004B	AATGCGCAGCACCAGGATAG		
IMP	IMP F	CATGGTTTGGTGGTTCTTGT	488	(Queenan and Bush, 2007)
	IMP R	ATAATTGGCGGACTTTGGC		

Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Sciences software (SPSS version 24), and statistical significance was set at p < 0.05. Data were presented as frequencies and percentages.

RESULTS AND DISCUSSION

Isolation of Bacteria from POS Devices

The frequency of bacteria isolation is shown in Table 2. The bacterial isolated includes *Staphylococcus aureus* (32.4%), *Klebsiella pneumoniae* (21.9%), *Pseudomonas aeruginosa* (17.2%), *Escherichia coli* (14.2%), *Bacillus subtilis* (10.1%), *Enterobacter* spp. (2.4 %) and *Enterococcus* sp. (1.8 %).

Distribution of *Pseudomonas Aeruginosa* Isolates

P. aeruginosa isolates were commonly isolated from Zone 2 with the prevalence of 20 (69.0%), while Zone 1 had the least

prevalence 2 (6.9%). The distribution of the isolates is detailed in (Table 3).

Antibiotic Resistance Patterns of *P. Aeruginosa* Isolates

The antibiotic resistance patterns of *P. aeruginosa* isolates to different antibiotics are shown in Table 4. The highest antibiotic resistance pattern as shown by the zone of inhibition was displayed against tetracyclines (58.6%), while the lowest antibiotic resistance pattern was displayed against imipenem (24.1%).

Carbapenemase and Virulence Genes of *Pseudomonas Aeruginosa* Isolates

Twenty-one (72.4%) of the *Pseudomonas aeruginosa* were positive for *OprL*, 11 (38.0%) for *toxA*, 8 (28.0%) for *lasB*, 4 (14.0%) for *bla_{VIM}* and 3 (10.3%) isolates were positive for *bla_{IMP}* (Table 5 and Fig 1-5).

Table 2: The Prevalence of Bacteria Isolates from POS Devices

Name	Number	%
<i>Klebsiella pneumoniae</i>	37	21.9
<i>Pseudomonas aeruginosa</i>	29	17.2
<i>Enterobacter</i> spp.	4	2.4
<i>Escherichia coli</i>	24	14.2
<i>Staphylococcus aureus</i>	55	32.5
<i>Bacillus subtilis</i>	17	10.1
<i>Enterococcus</i> sp.	3	1.8
Total	169	100

Table 3: Distribution of *P. Aeruginosa* Isolates in the Locations

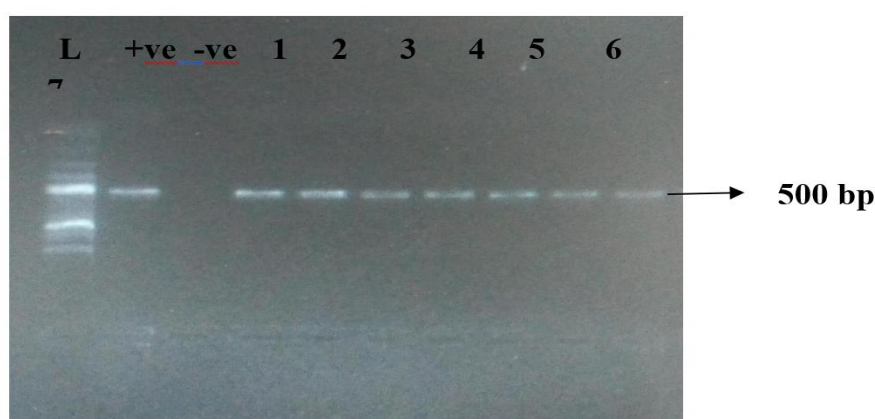
Location	No./ (%) of <i>P Aeruginosa</i>
Zone 1	2 (6.9)
Zone 2	20 (69.0)
Zone 3	7 (24.1)

Table 4: Antibiotic Resistance Patterns of *P. Aeruginosa* Isolates

S/N	Antibiotics	Number/Percentage Resistant
1	Ciprofloxacin (CIP)	11 (37.9)
2	Levofloxacin (LEV)	12 (41.2)
3	Cefoxitin (FOX)	13 (44.8)
4	Gentamicin (CN)	14 (48.3)
5	Ceftazidime (CAZ)	15 (51.7)
6	Cefepime (FEP)	14 (48.3)
7	Ceftriaxone (CRO)	14 (48.3)
8	Tetracycline (TE)	17 (58.6)
9	amoxicillin-clavulanic acid (AMC)	11 (37.9)
10	Imipenem (IPM)	7 (24.1)

Table 5: Distribution of Carbapenemase and Virulence Genes among *P. Aeruginosa* Isolates

Gene	Frequency	Percent (%)
<i>Bla_{VIM}</i>	4	14.0
<i>Bla_{IMP}</i>	3	10.3
<i>OPRL</i>	21	72.4
<i>tox_A</i>	11	38.0
<i>las_B</i>	8	28.0

**Figure 1: Electrophoresis Gel Picture of the *oprL* Gene**

Lane L = 100 bp ladder, Lane +ve = *oprL* positive control, Lane -ve = *oprL* negative control, Lane 1 - 7 = Sample representatives of *oprL* positive isolates.

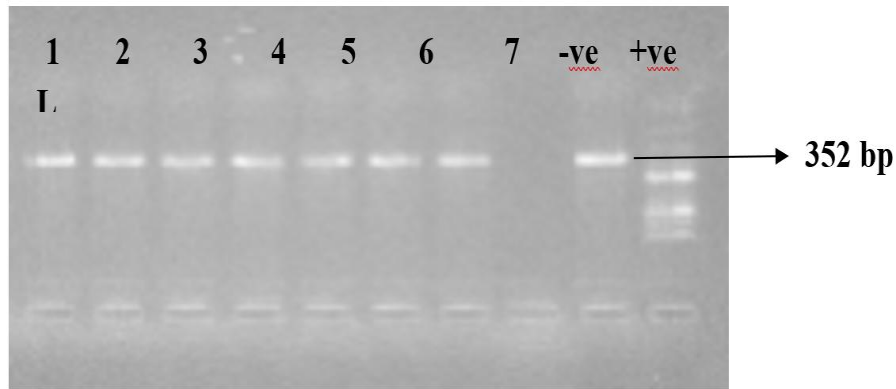


Figure 2: Electrophoresis Gel Picture of the *toxA* Gene

Lane = 100 bp ladder, Lane +ve = *toxA* positive control, Lane -ve = *toxA* negative control, Lane 1 - 7 = Sample representatives of *toxA* positive isolates.

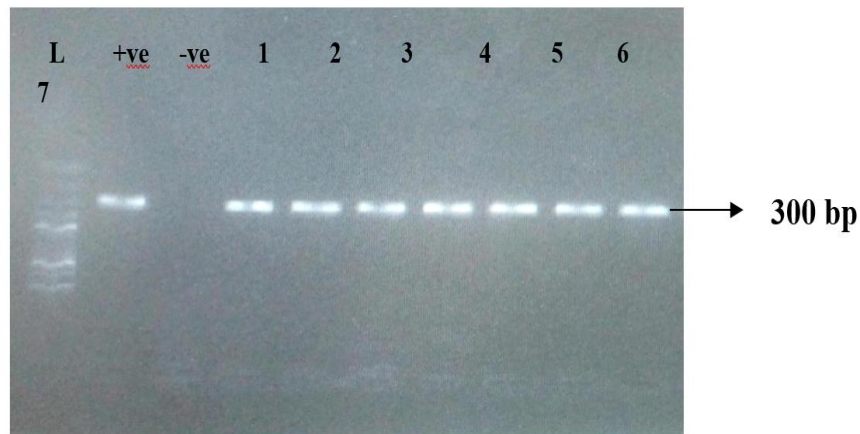


Figure 3: Electrophoresis gel Picture of the *lasB* Gene

Lane = 100 bp ladder, Lane +ve = *lasB* positive control, Lane -ve = *lasB* negative control, Lane 1 - 7 = Sample representatives of *lasB* positive isolates.

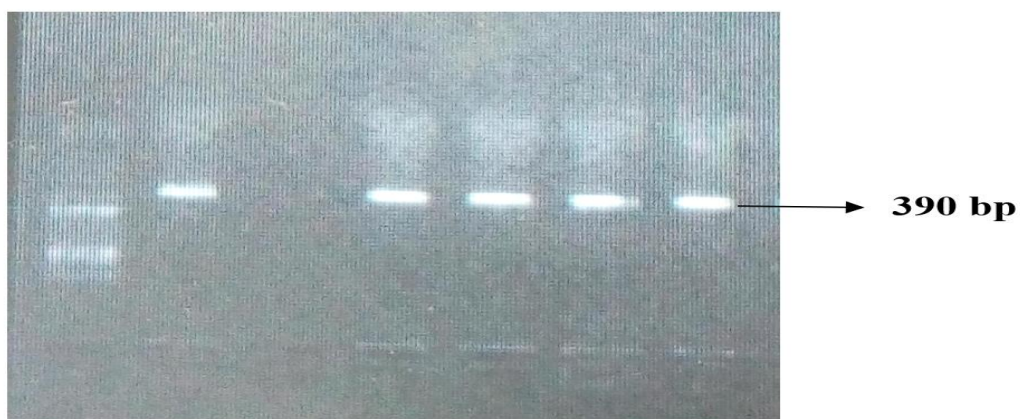


Figure 4: Electrophoresis Gel Picture of the *bla_{VIM}* Gene

Lane = 100 bp ladder, Lane +ve = *VIM* positive control, Lane -ve = *VIM* negative control, Lane 1 - 4 = *bla_{VIM}* positive isolates.

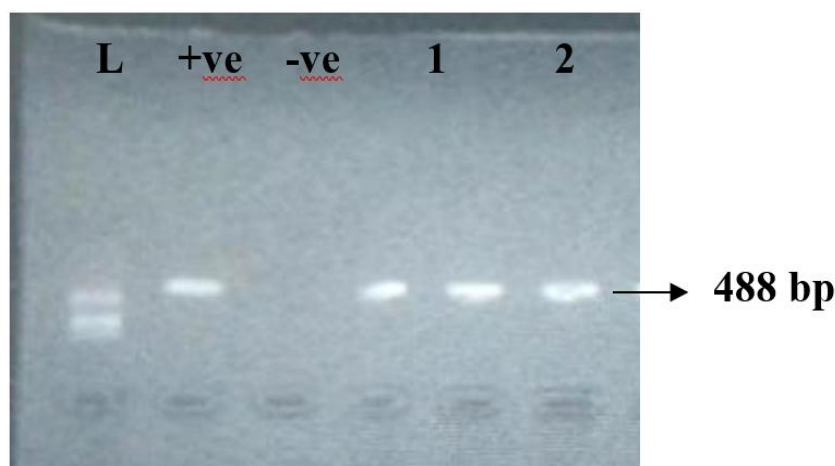


Figure 5: Electrophoresis Gel Picture of the *bla*_{IMP} Gene

Lane = 100 bp ladder, Lane +ve = *IMP* positive control, Lane -ve = *IMP* negative control, Lane 1 - 3 = *bla*_{IMP} positive isolates.

Discussion

Pseudomonas aeruginosa possesses different virulent factors and antibiotic resistance, and has been linked to hospital-associated infections (Khan *et al.*, 2018; Ntanda, 2024). Thus, its presence on frequently shared contact surfaces is of serious public health importance. This study reveals that the surfaces of POS devices were frequently colonized by *P. aeruginosa*. This is particularly indicative of the role POS devices could play in spreading *P. aeruginosa* within the environment. Moreover, *P. aeruginosa* has been reported to be able to bind and thrive on inanimate surfaces due to many survival mechanisms they possess (Moradali *et al.*, 2017; Zafer *et al.*, 2024).

This study detected a relatively higher frequency of *P. aeruginosa* isolates (17.2%) from POS devices in comparison with previous studies on the occurrence of bacteria on shared surfaces. For example, a study conducted by Yusha'u *et al.* (2024), detected 3.13% of bacterial isolates were *P. aeruginosa*. This could be because of reasons such as: One, the varying locations where these devices were sampled, as Lokoja is located at the North-central part of Nigeria and has commuters from the different parts of the country entering and exiting. Another reason could be as a result of the proximity of the sampling zones to the hospitals. For example, Zone 2 had the highest frequency of *P. aeruginosa* and covers areas of Lokoja where travelers enter and exit Lokoja, in addition to the Federal Teaching Hospital, Lokoja located within that zone. Meanwhile, *P. aeruginosa* has been reported to be highly prevalent in hospitals and on hospital devices (Brzozowski *et al.*, 2020; Assefa and Amare, 2022).

The presence of the *OPRL* gene detected in the *P. aeruginosa* isolates (72.2%), confirm the identity of *P. aeruginosa* from the POS devices, as *OPRL* gene is highly conserved in *P. aeruginosa* (Douraghi *et al.*, 2014). This gene encodes for the outer membrane porins (Harb, 2017), which are possibly responsible for pumping out of antibiotics, thus, conferring intrinsic resistance to antibiotics on the *P. aeruginosa*. Specifically, the presence of the *OPRL* gene in the *P. aeruginosa* isolates may be related to the antibiotic resistance against β -lactams, fluoroquinolones, aminoglycosides and tetracyclines, since these antibiotics require porins to penetrate the cell to reach their target.

The resistance pattern of the *P. aeruginosa* isolates showed that the isolates differed in their resistance patterns to the

antibiotics; with highest resistance to tetracyclines (58.6%). This could be related to the efflux pumping mechanism in *P. aeruginosa* which makes tetracycline antibiotic unable to reach its target. This is consistent with previous studies on resistance patterns of *P. aeruginosa* (Bernal-Rosas *et al.*, 2015; Rubin *et al.*, 2008).

In addition, *P. aeruginosa* displayed a high resistance to beta-lactam antibiotics and carbapenems. This could be associated with the production of beta-lactamase and carbapenemase enzymes which degrade the antibiotics, rendering them ineffective. This is also consistent with the *bla*_{IMP} and *bla*_{VIM} genes detected in 10.3% and 14% of isolates respectively. Similarly, previous studies have also reported the presence of extended-spectrum beta-lactamase genes as well as metallo-beta lactamase genes in *P. aeruginosa* strains, which are responsible for the production of beta-lactamase enzymes that can degrade a wide range of antibiotics (Aghamiri *et al.*, 2014; Jafari-Sales and Khaneshpour, 2020; Odewale *et al.*, 2024). Virulence genes *lasB* and *toxA* were detected in isolates, with 28% and 38% occurrence, respectively. The presence of these genes in the *P. aeruginosa* isolated from the POS devices suggest that these isolates are pathogenic. These virulent genes are important for invasion of host tissues, biofilm formation and inhibition of host protein synthesis (Farhan *et al.*, 2023). Thus, the POS devices could harbor and spread virulent strains if present on mobile genetic elements such as plasmids, integrons etc. of *Pseudomonas aeruginosa*, within the environment.

CONCLUSION

Pseudomonas aeruginosa was isolated from POS devices within Lokoja metropolis, Nigeria. The detection of virulence genes and antibiotics resistance genes in the *P. aeruginosa* underscores the role POS devices could play in the spread of antibiotics resistance and pathogenic *P. aeruginosa* within the environment. This also emphasizes the need to prioritize sanitary measures before and after using shared contact surfaces, to combat the spread of antibiotics resistance in the environment.

REFERENCES

Adenipekun, E. O., Akinleye, E. F., Tewogbade, O. A., and Iwalokun, B. A. (2023). Detection of virulence genes and multidrug resistance in *Pseudomonas aeruginosa* clinical

isolates from a public hospital in Lagos, Nigeria. *Scientific African*, 22, e01950.

Aduku, A. E., Bakare, A. A., Oyabambi, A., Akanni, E. O., and Isah, F. I. (2025). IMPACT OF POINT OF SALE (POS) TERMINAL ADOPTION ON THE RETURN ON PROFIT AFTER TAX (PAT) OF TIER-ONE BANKS IN NIGERIA. *Prestieesci Research Review*, 2(1), 710–735.

Aghamiri, S., Amirmozafari, N., Fallah Mehrabadi, J., Fouladtan, B., and Samadi Kafil, H. (2014). Antibiotic Resistance Pattern and Evaluation of Metallo-Beta Lactamase Genes Including bla-IMP and bla-VIM Types in *Pseudomonas aeruginosa* Isolated from Patients in Tehran Hospitals. *International Scholarly Research Notices*, 2014(1), 941507.

Assefa, M., and Amare, A. (2022). Biofilm-associated multidrug resistance in hospital-acquired infections: A review. *Infection and Drug Resistance*, 5061–5068.

Bernal-Rosas, Y., Osorio-Muñoz, K., and Torres-García, O. (2015). *Pseudomonas aeruginosa*: An emerging nosocomial trouble in veterinary. *Revista MVZ Córdoba*, 20, 4937–4946.

Brzozowski, M., Krukowska, Ż., Galant, K., Jursa-Kulesza, J., and Kosik-Bogacka, D. (2020). Genotypic characterisation and antimicrobial resistance of *Pseudomonas aeruginosa* strains isolated from patients of different hospitals and medical centres in Poland. *BMC Infectious Diseases*, 20, 1–9.

Chand, Y., Khadka, S., Sapkota, S., Sharma, S., Khanal, S., Thapa, A., Rayamajhee, B., Khadka, D. K., Panta, O. P., and Shrestha, D. (2021). Clinical Specimens are the Pool of oprL and toxA Virulence Genes Harboring Multidrug-Resistant *Pseudomonas aeruginosa*: Findings from a Tertiary Hospital of Nepal. *Emergency Medicine International*.

Clinical and Laboratory Standards Institute (CLSI) (2018) Performance Standards for Antimicrobial Susceptibility Testing. CLSI Approved Standard M100-S15. Clinical and Laboratory Standards Institute, Wayne.

Dawodu, O., and Akanbi, R. (2021). Isolation and identification of microorganisms associated with automated teller machines on Federal Polytechnic Ede campus. *PLoS One*, 16(8), e0254658.

Douraghi, M., Ghasemi, F., Dallal, M. S., Rahbar, M., and Rahimiforushani, A. (2014). Molecular identification of *Pseudomonas aeruginosa* recovered from cystic fibrosis patients. *Journal of Preventive Medicine and Hygiene*, 55(2), 50.

Farhan, R. E., Solymán, S. M., Hanora, A. M., and Azab, M. M. (2023). Molecular detection of different virulence factors genes harbor pslA, pelA, exoS, toxA and algD among biofilm-forming clinical isolates of *Pseudomonas aeruginosa*. *Cellular and Molecular Biology*, 69(5), 32–39.

Fernández-Billón, M., Llambías-Cabot, A. E., Jordana-Lluch, E., Oliver, A., and Macià, M. D. (2023). Mechanisms of antibiotic resistance in *Pseudomonas aeruginosa* biofilms. *Biofilm*, 5, 100129.

Gaubá, A., and Rahman, K. M. (2023). Evaluation of antibiotic resistance mechanisms in gram-negative bacteria. *Antibiotics*, 12(11), 1590.

Harb, C. P. (2017). *Genome-wide antibiotic resistance and virulence profiling of Pseudomonas Aeruginosa isolated from clinical samples in Lebanon. (c2017)*.

Horcajada, J. P., Montero, M., Oliver, A., Sorlí, L., Luque, S., Gómez-Zorrilla, S., Benito, N., and Grau, S. (2019). Epidemiology and treatment of multidrug-resistant and extensively drug-resistant *Pseudomonas aeruginosa* infections. *Clinical Microbiology Reviews*, 32(4), 10–1128.

Innocent-Adiele, H., Nkara, N., Osuala, O., Daokoru-Olukole, C., Okafor, O., Onu, E., Cooke, T., Nwankwo, G., Mbah, E., and Adim, C. (2023). Isolation and AntibioGram Pattern of Bacteria Isolated from Automated Teller Machine (ATM) at Abuja Campus, University of Port Harcourt, Nigeria. *Int. J. Pathog. Res*, 12(6), 127–135.

Jafari-Sales, A., and Khaneshpour, H. (2020). Molecular Study of BlaIMP and BlaVIM Genes in *Pseudomonas Aeruginosa* Strains, Producer of Metallo Beta Lactamases Isolated from Clinical Samples in Hospitals and Medical Centers of Tabriz. *Paramedical Sciences and Military Health*, 14(4), 18–25.

Kaye, K. S., and Pogue, J. M. (2015). Infections caused by resistant gram-negative bacteria: Epidemiology and management. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, 35(10), 949–962.

Khan, A., Miller, W. R., and Arias, C. A. (2018). Mechanisms of antimicrobial resistance among hospital-associated pathogens. *Expert Review of Anti-Infective Therapy*, 16(4), 269–287.

Lepe, J. A., and Martínez-Martínez, L. (2022). Resistance mechanisms in Gram-negative bacteria. *Medicina Intensiva (English Edition)*, 46(7), 392–402.

Li, X.-Z., Plésiat, P., and Nikaido, H. (2015). The challenge of efflux-mediated antibiotic resistance in Gram-negative bacteria. *Clinical Microbiology Reviews*, 28(2), 337–418.

Mamudu, Z. U., and Gayovwi, G. O. (2019). Cashless policy and its impact on the Nigerian economy. *International Journal of Education and Research*, 7(3), 111–132.

Manner, C., Dias Teixeira, R., Saha, D., Kaczmarczyk, A., Zemp, R., Wyss, F., Jaeger, T., Laventie, B.-J., Boyer, S., and Malone, J. G. (2023). A genetic switch controls *Pseudomonas aeruginosa* surface colonization. *Nature Microbiology*, 8(8), 1520–1533.

Mendes, R. E., Kiyota, K. A., Monteiro, J., Castanheira, M., Andrade, S. S., Gales, A. C., Pignatari, A. C., and Tufik, S. (2007). Rapid detection and identification of metallo-β-lactamase-encoding genes by multiplex real-time PCR assay and melt curve analysis. *Journal of Clinical Microbiology*, 45(2), 544–547.

Mentasti, M., Prime, K., Sands, K., Khan, S., and Wootton, M. (2019). Rapid detection of IMP, NDM, VIM, KPC and OXA-48-like carbapenemases from Enterobacterales and Gram-negative non-fermenter bacteria by real-time PCR and

melt-curve analysis. *European Journal of Clinical Microbiology and Infectious Diseases*, 38, 2029–2036.

Moradali, M. F., Ghods, S., and Rehm, B. H. (2017). *Pseudomonas aeruginosa* lifestyle: A paradigm for adaptation, survival, and persistence. *Frontiers in Cellular and Infection Microbiology*, 7, 39.

Mostafa, A. E., Hassan, M. G., El-Metwally, M. M., Abo Neima, S. E., and Zewail, M. (2024). Bacteria Associated with Automated Teller Machines: Isolation and Identification. *Egyptian Academic Journal of Biological Sciences, G. Microbiology*, 16(2), 139–149.

Ntanda, P. M. (2024). *Clinical and molecular evaluation of pseudomonas aeruginosa nosocomial infections among adult patients at the university teaching hospital in Lusaka province and Ndola teaching hospital in Copperbelt province of Zambia*.

Odewale, G., Jibola-Shittu, M.Y., Bala, H.E.M., Akogwu, R., Raimi, L.O. (2024). Antibiotic susceptibility and detection of extended-spectrum β -lactamase genes in gram-negative bacteria isolated from automated teller machines. *FUDMA Journal of Sciences*, 8(1):118-124. DOI: <https://doi.org/10.33003/fjs-2024-0801-2207>.

Porter, L., Sultan, O., Mitchell, B. G., Jenney, A., Kiernan, M., Brewster, D. J., and Russo, P. L. (2024). How long do nosocomial pathogens persist on inanimate surfaces? A scoping review. *Journal of Hospital Infection*, 147, 25–31.

Qin, S., Xiao, W., Zhou, C., Pu, Q., Deng, X., Lan, L., Liang, H., Song, X., and Wu, M. (2022). *Pseudomonas aeruginosa*: Pathogenesis, virulence factors, antibiotic resistance, interaction with host, technology advances and emerging therapeutics. *Signal Transduction and Targeted Therapy*, 7(1), 199.

Queenan, A. M., and Bush, K. (2007). Carbapenemases: The versatile β -lactamases. *Clinical Microbiology Reviews*, 20(3), 440–458.

Reynolds, D., and Kollef, M. (2021). The epidemiology and pathogenesis and treatment of *Pseudomonas aeruginosa* infections: An update. *Drugs*, 81(18), 2117–2131.

Rizvi, S., Kurtz, A., Williams, I., Gualdoni, J., Myzyri, I., and Wheeler, M. (2020). Protecting financial transactions through networks and point of sales. *Journal of Cyber Security Technology*, 4(4), 211–239.

Rocha, A. J., Barsottini, M. R. de O., Rocha, R. R., Laurindo, M. V., Moraes, F. L. L. de, and Rocha, S. L. da. (2019). *Pseudomonas aeruginosa*: Virulence factors and antibiotic resistance genes. *Brazilian Archives of Biology and Technology*, 62, e19180503.

Rubin, J., Walker, R., Blickenstaff, K., Bodeis-Jones, S., and Zhao, S. (2008). Antimicrobial resistance and genetic characterization of fluoroquinolone resistance of *Pseudomonas aeruginosa* isolated from canine infections. *Veterinary Microbiology*, 131(1–2), 164–172.

Urban-Chmiel, R., Marek, A., Stępień-Pyśniak, D., Wiczeorek, K., Dec, M., Nowaczek, A., and Osek, J. (2022). Antibiotic resistance in bacteria—A review. *Antibiotics*, 11(8), 1079.

Uriawan, W., Faroj, R. Z., Hadid, R. A., Khoirunnisa, S., Julianto, S., and Sopian, A. R. (2024). *Innovative Data Management Strategies in Point of Sale Application Development: Increasing Business Productivity*.

Wu, Y. T., Zhu, L. S., Tam, K. C., Evans, D. J., and Fleiszig, S. M. (2015). *Pseudomonas aeruginosa* survival at posterior contact lens surfaces after daily wear. *Optometry and Vision Science*, 92(6), 659–664.

Yang, J., Xu, J.-F., and Liang, S. (2024). Antibiotic resistance in *Pseudomonas aeruginosa*: Mechanisms and emerging treatment. *Critical Reviews in Microbiology*, 1–19.

Yusha'u, M., Ahmad, J., Adam, R., and Hamza, M. (2024). Investigating the contamination levels, bacterial diversity and susceptibility pattern of bacteria isolated from point of sale (pos) devices in Kano metropolis. *Bayero Journal of Pure and Applied Sciences*, 17(1), 43–50.

Zafer, M. M., Mohamed, G. A., Ibrahim, S. R., Ghosh, S., Bornman, C., and Elfaky, M. A. (2024). Biofilm-mediated infections by multidrug-resistant microbes: A comprehensive exploration and forward perspectives. *Archives of Microbiology*, 206(3), 101.

