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Detection of Genetic Supports Regulated the Quorum Sensing in Multidrug-Resistant *P. aeruginosa* and *E. coli*

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A B S T R A C T

Quorum sensing (QS) is a system of intercellular communication and regulation of the transcription of resistance, virulence, and pathogenicity genes. The aim of this study was to identify genetic markers controlling quorum sensing in multidrug-resistant *P. aeruginosa* and *E. coli*. A set of fifty (50) strains, composed of *P. aeruginosa* (30) and *E. coli* (20), were isolated from animal products. These strains underwent phenotypic and biochemical identification. Antibiotic resistance profiles were determined by the Muller-Hinton agar diffusion method. Quorum sensing markers (*LasI/LasR*) and (*RhlI/RhlR*) were detected by PCR. The prevalence of the *Las* gene (*LasI/LasR*) and the *Rhl* gene (*RhlI/RhlR*) was 80% and 60%, respectively, in *P. aeruginosa*. In *E. coli*, the prevalence of QS genes was 40% for *Las* (*LasI/LasR*) and 40% for *Rhl* (*RhlI/RhlR*). The total prevalence of the *Las* gene and the *Rhl* gene was 64% (*LasI/LasR*) and 68% (*RhlI/RhlR*), respectively, in the studied strains. *E. coli* strains exhibited penicillin resistance exceeding 25% for amoxicillin (67.5%), amoxicillin-clavulanic acid (46.6%), and piperacillin (28.5%). This resistance was less than 25% for ciprofloxacin (23.7%), ceftazidime (18.6%), cefoxitin (17.8%), cefepime (14.3%), and imipenem (14.6%). *P. aeruginosa* strains expressed multidrug resistance to ticarcillin (54%), aztreonam (47%), ticarcillin-clavulanic acid (32%), piperacillin (29%), levofloxacin (24%), and ciprofloxacin (29%). The detection and control of genetic factors influencing quorum sensing in multidrug-resistant bacteria can improve diagnosis and contribute to the fight against biofilm infections.

Introduction

Quorum sensing (QS) is an intercellular communication system used by many microorganisms, particularly bacteria (Rosignoli *et al.,* 2026; Wang *et al.,* 2025). This communication system allows bacteria to collectively synchronize their behavior with population density and their environment (Rosignoli *et al.,* 2026; Qin *et al.,* 2022).

Indeed, the QS mechanism involves the production and detection of small extracellular molecules, called autoinducers (AIs), which are released and whose concentration increases proportionally to cell density (Salsabila *et al.,* 2025; Rather *et al.,* 2021; Yang *et al.,* 2021). Many Gram-negative bacteria, including *Pseudomonas aeruginosa*, *E. coli*, and *Acinetobacter spp*., utilize this system. and *Burkholderia spp*., use a different class of auto-inducers (AI): acylhomoserine lactones (AHSLs) (Soto-Aceves *et al.,* 2021; Yang *et al.,* 2021). AHSLs are composed of a lactone ring and an aliphatic acyl chain varying in size and function.

In Gram-negative bacteria, and particularly in *P. aeruginosa*, four types of QS systems, including the las, rhl, pqs, and integrated QS (IQS) systems, have been studied to date (Ayman *et al.,* 2026; Rosignoli *et al.,* 2026; Chu and Yang, 2024). Among these communication systems, the las and rhl systems are the most extensively studied (Rather *et al.,* 2021; Yang *et al.,* 2021). The integrated QS (IQS) system, however, was recently added to the *P. aeruginosa* QS system, and its mechanism is therefore not well understood (Raya *et al.,* 2025; Touati *et al.,* 2025; Chu & Yang, 2024).

Thus, these two systems (las, rhl), the most exploited, together amplify and coordinate the expression of numerous virulence genes, biofilm formation, and the production of proteases and siderophores (Rosignoli *et al.,* 2026; Soto-Aceves *et al.,* 2021). Each system is defined by a pair composed of a regulatory protein and an auto-inducing enzyme: LasR/LasI for the las system and RhlR/RhlI for the rhl system (Ayman *et al.,* 2026; Chu and Yang, 2024; Yang *et al.,* 2021). Indeed, the las system, the first to be exploited, consists of a lasR regulatory gene encoding the LasR protein and a *LasI* gene encoding an autoinductive synthase LasI involved in the synthesis of the long-chain homoserine lactones (HSL) 3-oxo-C12-HSL (Salsabila *et al.,* 2025; Chu and Yang, 2024; Yang *et al.,* 2021). The LasR/3-oxo-C12-HSL complex is a transcriptional activator of virulence genes and an activator of the *LasI* gene (Rosignoli *et al.,* 2026; Rather *et al.,* 2021).

The virulence genes activated by this system include: lasB, lasA, and aprA, encoding two elastases and an alkaline protease, respectively, each contributing to the destruction of lung tissue (Touati *et al.,* 2025; Yang *et al.,* 2021).

The *toxA* gene, encoding an ADP-ribosyl exotoxin, and the *xcpR* and *xcpP* genes, encoding proteins of the type II secretion machinery, are also activated (Raya *et al.,* 2025; Soto-Aceves *et al.,* 2021). Furthermore, the rhl system, the second to be exploited, functions according to the same mechanism and comprises the *RhlR* gene, encoding the regulatory protein RhlR, and the *RhlI* gene, encoding an autoinducing synthase RhlI (Touati *et al.,* 2025; Soto-Aceves *et al.,* 2021). This synthase is necessary for the synthesis of a second type of short-chain AHSL: N-butyryl-L-homoserine lactone (C4-HSL) (Rosignoli *et al.,* 2026; Yang *et al.,* 2021). The RhlR-C4-HSL complex controls the expression of the rhlAB operon required for the production of rhamnolipids, and the expression of a series of genes including *lasB*, *lasA*, *aprA*, and *rhlI* (Ayman *et al.,* 2026; Touati *et al.,* 2025; Soto-Aceves *et al.,* 2021).

In addition to the regularly studied las and rhl systems and the less explored integrated QS (IQS) system, the latest pqs system synthesizes the signaling molecule 2heptyl3hydroxy4quinolone (*Pseudomonas aeruginosa* quinolone signal: PQS) (Rosignoli *et al.,* 2026; Wang *et al.,* 2025). This molecule recognizes its receptor PqsR, which regulates the release of environmental DNA during biofilm formation (Touati *et al.,* 2025; Yang *et al.,* 2021). These numerous genetic components involved in establishing the cell communication system amplify bacterial pathogenicity and virulence (Raya *et al.,* 2025; Qin *et al.,* 2022).

Indeed, these different systems demonstrate the involvement of quorum sensing (QS) in numerous bacterial mechanisms harmful to humans and human activity in the environment (Papaneophytou, 2026; Rosignoli *et al.,* 2026). These harmful processes include the biofilm formation cycle, the synthesis of virulence factors, and multi-resistance to antimicrobials, particularly antibiotics (Raya *et al.,* 2025; Soto-Aceves *et al.,* 2021). Several studies and quorum quenching strategies have been conducted to control quorum sensing and interfere with it in order to inhibit these

harmful processes that promote bacterial pathogenicity and virulence (Rosignoli *et al.,* 2026; Touati *et al.,* 2025).

In Côte d'Ivoire, some studies have demonstrated the activity of essential oils on quorum sensing induced by the production of N-acyl-homoserine lactone (AHL) (Benie *et al.,* 2021). Other researchers have studied the effect of these essential oils on the biofilm formation cycle in multidrug-resistant *P. aeruginosa* (Benie *et al.,* 2021). Despite this limited research conducted in Côte d'Ivoire, data are lacking regarding the control and manipulation of the molecular determinants involved in the quorum sensing process. The objective of this study is to identify genetic markers controlling quorum sensing in *P. aeruginosa* and *E. coli* isolated from animal products.

Materials and Methods

Bacterial Isolates

A total of fifty (50) bacterial strains, composed of *P. aeruginosa* (30) and *E. coli* (20), isolated from animal products (beef, fresh fish, and smoked fish), were included in this study. These *P. aeruginosa* and *E. coli* strains were isolated on Cetrimide and TBX agar, respectively. These bacterial strains isolated on selective media underwent biochemical and molecular identification by PCR.

Determination of the resistance profile of *P. aeruginosa* and *E. coli* strains

The phenotypic resistance profile of *P. aeruginosa* and *E. coli* strains was determined by the Müeller-Hinton (MH) agar diffusion method. Commonly used antibiotic discs for human therapy were selected. The tested antibiotic discs are listed in Tables 1 and 2. The reference strains *P. aeruginosa* ATCC 27853 and *E. coli* ATCC 25922 were used for quality control of the antibiogram. The entire surface of the Müeller-Hinton agar was aseptically swabbed with the bacterial suspension in saline solution at a turbidity of 0.5 McFarland. This inoculum of approximately 10^8 CFU/mL was prepared from 18-hour-old colonies taken from ordinary agar. Fifteen minutes after placing the antibiotic-impregnated discs on the surface of the inoculated agar, the plates were inverted and incubated, ideally at 37°C, for 24 hours. After 24 hours, the zones of inhibition were measured using calipers (ruler). The interpretation of the results was performed according to the guidelines of the French Society for Microbiology's Antibiogram Committee (CA-SFM/EUCAST, 2024).

Detection of genetic substrates for quorum sensing

Extraction and purification of genomic DNA

All isolated strains of *P. aeruginosa* and *E. coli* were revived in brain-heart broth (BHC) for 24 h. The genomic DNA of these strains was then extracted by the thermal lysis method and purified according to the technique described by Al-Kilabi *et al.,* (2020). After extraction, the DNA was diluted and stored at -20 °C to serve as a template for polymerase chain reaction (PCR).

Reaction mixture

The reaction mixture was carried out according to the method described by Pournajaf *et al.,* (2018). This reaction mixture, with a total volume of 25 µL, consisted of 16.5 µL of sterile Milli-Q water (Milli-Q™, Millipore Corporation, USA), 4 µL of loading buffer at a concentration of 5X, 1.5 µL of $MgCl_2$ at 2 mM (Promega Corporation, Madison, WI 53711-5399, USA), 0.2 µL of dNTP at 10 mM, 0.1 µL of each primer at 10 mM (Integral DNA Technology, California, USA), 0.1 µL of GoTaq® G2 Flexi DNA polymerase at a final concentration of 1.5 U (Promega Corporation, Madison, WI 53711-5399, USA) and 2.5 µL of DNA template. Reference strains and RNase-free DNA water were used respectively as a positive control and as a negative control for each PCR reaction.

Amplification of Quorum Sensing Genes (*LasI*, *LasR*, *RhlI*, *RhlI*)

The amplification of the quorum-sensing genes *LasI*, *LasR*, *RhlI*, and *RhlR* was performed according to the technique described by Al-Kilabi *et al.,* (2020). The amplification program consisted of an initial 2-minute denaturation at 95°C followed by a cyclic phase repeated thirty (30) times. The cyclic phase repeated thirty (30) times comprised a 30-second denaturation step at 95°C, a 60-second primer binding (hybridization) step at 65°C, and a 90-second elongation step at 72°C. The amplification of the quorum-sensing genes concluded with a final 4-minute elongation at 72°C. The amplification products were stored at +4°C until the

thermocycler was shut down. The amplification programs and nucleotide sequence of the primers used for quorum sensing gene detection are described in Table 3.

Electrophoresis of amplification products

Gene amplification products were detected on a 1.5% agarose gel after 120 V incubation for 30 min and visualized using a trans-illuminator with a UV plate (Molecular Imager Gel Doc™ EZ, Bio-Rad, USA). The DNA band and its size were identified relative to the positive control and the molecular weight marker.

Statistical analysis

Excel was used to plot the curves. The differences between groups were analyzed with SPSS 20.0 statistical software using the K^2 method. The significant level was determined at $p < 0.05$.

Results and Discussion

Differential characteristics in *P. aeruginosa* and *E. coli*

Fresh specimens indicate that *P. aeruginosa* and *E. coli* are motile rods. Gram staining shows that both of these bacteria are Gram-negative rods. *Pseudomonas aeruginosa* appears in clusters or as isolated cells, while *E. coli* appears as isolated cells or in small chains (Figure 1). The catalase test showed that both bacteria were catalase-positive, as evidenced by the release of bubbles. The appearance of a purple stain on the oxidase strip indicated that the *P. aeruginosa* strains are oxidase-positive.

Resistance of *P. aeruginosa* strains to antibiotics

The *E. coli* and *P. aeruginosa* strains involved in quorum sensing exhibited varying levels of multidrug resistance to the tested antibiotics (Figure 2). The *E. coli* strains involved in quorum sensing showed penicillin resistance greater than 25% for amoxicillin (67.5%), amoxicillin-clavulanic acid (46.6%), and piperacillin (28.5%). This resistance was less than 25% for ciprofloxacin (23.7%), cefepime (14.3%), cefoxitin (17.8%), ceftazidime (18.6%), and imipenem (14.6%) (Figure 3). In order of increasing importance, *P. aeruginosa* strains expressed multidrug resistance to ticarcillin (54%), aztreonam (47%), ticarcillin-clavulanic acid (32%), piperacillin (29%), and ciprofloxacin (29%). These strains were also resistant to levofloxacin (24%) and less resistant to cefepime (11%), cefoxitin (10%), ceftazidime (9%), and imipenem (7%) (Figure 4).

Genetic and molecular determinants of quorum sensing in *P. aeruginosa* and *E. coli*

Electrophoretic profiles show genetic determinants involved in quorum sensing in *P. aeruginosa* and *E. coli*. Some strains of *P. aeruginosa* and *E. coli* harbored the *Las* gene or the *Rhl* gene which codes respectively for the production of elastase and rhamnolipids (figures 5).

Prevalence of genetic determinants involved in quorum sensing

The results indicated a diversity in the prevalence of genetic determinants involved in quorum sensing in *P. aeruginosa* and *E. coli*. The *Las genes* (*LasI* (80%); *LasR* (80%)) and the *Rhl* gene (*RhlI* (60%); *RhlR* (60%)) were detected in *P. aeruginosa* with distinct prevalences. In *E. coli*, the Las and Rhl genes were detected with prevalences of 40% (*LasI/LasR*) and 40% (*RhlI/RhlR*), respectively. The total prevalence of the *Las* and *Rhl* genes was 64% (*LasI/LasR*) and 68% (*RhlI/RhlR*), respectively, in the bacterial strains studied (Table 4).

Major antibiotic resistance phenotypes associated with quorum sensing

Analysis of the results revealed that the major resistance phenotypes associated with quorum sensing were primarily linked to penicillin antibiotics and monobactams (Figure 6). The rate of major resistance phenotypes associated with quorum sensing ranged from 28.3% to 64.1% for resistance to ticarcillin-clavulanic acid and amoxicillin associated with the *Las* gene (Figure 6).

This rate of major resistance phenotypes associated with quorum sensing ranged from 32.7% to 54.3% for resistance to amoxicillin-clavulanic acid and ticarcillin associated with the *Rhl* gene. The rate of major resistance phenotypes associated with quorum sensing ranged from 40.4% to 41.6% for resistance to aztreonam associated with the *Las* and *Rhl* genes, respectively. Cephalosporins, carbapenems and quinolones showed less than 25% resistance associated with quorum sensing (Figure 6).

Quorum sensing (QS) is an intercellular communication and virulence gene transcription regulation system used by many microorganisms, particularly bacteria (Rosignoli *et al.,* 2026; Yang *et al.,* 2021). Generally, in *P. aeruginosa* and *E. coli*, pathogenicity and antimicrobial resistance are attributed to their ability to form a protective biofilm and to the production of numerous membrane and extracellular factors via quorum sensing (Yang *et al.,* 2021; Rather *et al.,* 2021; Maisuria *et al.,* 2016). This study demonstrated the involvement of multidrug-resistant *E. coli* and *P. aeruginosa* strains in the intracellular communication system known as quorum sensing (Qu *et al.,* 2024; Pumbwe *et al.,* 2008).

Indeed, this study indicated a diversity in the prevalence of genetic determinants involved in quorum sensing in *P. aeruginosa* and *E. coli*. The *Las* genes (*LasI* (80%); *LasR* (80%)) and the *Rhl* gene (*RhlI* (60%); *RhlR* (60%)) were detected in *P. aeruginosa* with distinct prevalences. In *E. coli*, the Las and *Rhl* genes were detected with respective prevalences of 40% (*LasI/LasR*) and 40% (*RhlI/RhlR*). The total prevalence of the Las and Rhl genes was 64% (*LasI/LasR*) and 68% (*RhlI/RhlR*), respectively, in the bacterial strains studied.

These different prevalences of autoinducers, relatively high in *P. aeruginosa* and *E. coli* strains, could justify the existence of a communication system between these bacteria isolated from animal products (Rosignoli *et al.,* 2026; Yang *et al.,* 2021). Consequently, quorum sensing in these *P. aeruginosa* and *E. coli* strains could be controlled by the LasI/R and RhlI/RhlR systems, involved in the synthesis of certain virulence genes (Ayman *et al.,* 2026; Rather *et al.,* 2021; Yang *et al.,* 2021).

Indeed, in Gram-negative bacteria, two main effector/receptor quorum sensing systems involving homoserine lactones coexist and play a role in regulating the expression of virulence genes (Yang *et al.,* 2021; Rasamiravaka *et al.,* 2015). The LasI/LasR system, which codes for the production of virulence factors such as LasA, LasB elastases and exotoxin A (ToxA), involves a 3-oxo-C12-homoserine lactone (3-oxo-C12-HSL) (Rather *et al.,* 2021; Soto-Aceves *et al.,* 2021).

The second RhlI/R system, responsible for the production and regulation of factors such as rhamnolipids, pyocyanin, and cyanide, involves a C4-homoserine lactone (C4-HSL) (Ayman *et al.,* 2026; Yang *et al.,* 2021; Maisuria *et al.,* 2016).

The biosynthesis of rhamnolipids by these studied strains could be explained by several environmental stimuli such as cell density, stress, and nutritional deficiency (Rather *et al.,* 2021; Déziel *et al.,* 2004; Dekimpe and Déziel, 2009).

The presence of these *Las* and *Rhl* genes in both *P. aeruginosa* and *E. coli* isolated from smoked fish suggests that these strains could be involved in the production of bacterial pheromones promoting multi-resistance and the establishment of quorum sensing (Chu and Yang, 2024; Yang *et al.,* 2021).

Furthermore, the profile of *E. coli* and *P. aeruginosa* strains involved in quorum sensing showed penicillin resistance exceeding 25% for amoxicillin, amoxicillin-clavulanic acid, ticarcillin, ticarcillin-clavulanic acid, and piperacillin. This resistance of *E. coli* and *P. aeruginosa* strains to penicillins could be explained by an intrinsic resistance mechanism linked to the modification of penicillin-binding proteins (PBPs) (Rosignoli *et al.,* 2026; Salsabila *et al.,* 2025; Yehia *et al.,* 2020). Also, beta-lactamases are enzymatic proteins produced by bacteria that can hydrolyze the beta-lactam cycle of penicillins, rendering them inactive (Qu *et al.,* 2024; Rather *et al.,* 2021; Yehia *et al.,* 2020).

Also, resistance of *E. coli* and *P. aeruginosa* strains was less than 25% to ciprofloxacin, cefepime, cefoxitin, ceftazidime, and imipenem. This resistance of certain *E. coli* and *P. aeruginosa* strains to cephalosporins, carbapenems, and fluoroquinolones could be induced by chromosomal mechanisms, in combination with resistance mechanisms to extended-spectrum beta-lactamases (ESBLs) (Rosignoli *et al.,* 2026; Wang *et al.,* 2025).

The high resistance of *P. aeruginosa* strains to aztreonam and penicillins could be explained by acquired resistance (plasmids, transposons) (Touati *et al.,* 2025; Qin *et al.,* 2022).

This acquired resistance could be due to increased impermeability of the outer membrane or to the production of inactivating enzymes that can expel antibiotics according to Yang *et al.,* (2021); Kumar and Schweizer, (2005).

Table.1 Antibiotic molecules and their concentration for the antibiogram of *P. aeruginosa*

Family	Antibiotics	Abbreviation	Concentration (µg)
Penicillin	*Ticarcillin*	TIC	75
	Piperacillin	PIP	100
	Ticarcillin-clavulanic acid	TCC	75-100
Cephalosporin	*Cefoxitin*	FOX	30
	Cefepime	FEP	30
	Ceftazidime	CAZ	10
Carbapenem	*Imipenem*	IMP	10
Monobactams	*Aztreonam*	ATM	30
Aminoglycosides	*Kanamycin*	*K*	30
Fluoroquinolone	*Ciprofloxacin*	CIP	5
	Levofloxacin	*LVX*	5

Aztreonam (ATM), Imipenem (IPM), Cefepime (FEP), Ceftazidime (CAZ), Cefoxitin (FOX), Ticarcillin (TIC), Ticarcillin-clavulanic acid (TCC), Piperacillin (PIP), Ciprofloxacin (CIP), Levofloxacin (LVX), Kanamycin (K)

Table.2 Antibiotic molecules and their concentration for the antibiogram of *E. coli*

Family	Antibiotics	Abbreviation	Concentration (µg)
Penicillin	*Amoxicillin*	AMO	20-30
	Piperacillin	PIR	100
	Amoxicillin-acide Clavulanique	AMC	20-10
Cephalosporin	*Cefoxitin*	FOX	30
	Cefepime	FEP	30
	Ceftazidime	CAZ	10
Carbapenem	*Imipenem*	IMP	10
Aminoside	*Gentamicin*	GEN	10
Fluoroquinolone	*Ciprofloxacin*	CIP	5
	Nalidixic acid	NA	30

Amoxicillin-Clavulanic acid (AMC), Amoxicillin (AMO), Piperacillin (PIR), Ciprofloxacin (CIP), Gentamicin (GEN), Cefepime (FEP), Cefoxitin (FOX), Ceftazidime (CAZ), Imipenem (IMP), Nalidixic acid (NA)

Table.3 Detection primers for the molecular supports of quorum sensing

Genes coding for quorum sensing		Primer sequence (5′→3′)	Amplification program	Size bp	References
las	***LasI***	F: 5'-CGTGCTCAAGTGTTCAAGG -3' R: 5'-TACAGTCGGAAAAGCCCAG -3'	95°C, 2 min 30 x [95°C, 30 s; 65°C, 60s; 72°C, 900s] 72°C, 4 min; 4°C.	**295**	**CostaLima *et al.*, 2018**
	LasR	F: 5'-AAGTGGAAAATTGGAGTGGAG-3' R: 5'-GTAGTTGCCGACGACGATGAAG- 3'		**130**	
rhl	***RhlI***	F: 5'-TTCATCCTCCTTTAGTCTTCCC-3' R: 5'-TTCCAGCGATTCAGAGAGC -3'		**155**	
	RhlR	F: 5'-TGCATTTTATCGATCAGGGC-3' R: 5'-CACTTCCTTTTCCAGGACG-3'		**133**	

The las gene encodes elastase production; the rhl gene encodes rhamnolipid production. The las gene consists of a lasR regulatory gene encoding the lasR protein and a lasI gene encoding an autoinductive synthase, lasI, involved in the synthesis of long-chain homoserine lactones (HSLs), specifically 3-oxo-C12-HSL. The rhl gene comprises the rhlR gene, encoding the rhlR regulatory protein, and the rhlI gene, encoding the self-inductive synthase, rhlI, necessary for the synthesis of short-chain homoserine lactones (HSLs): N-butyryl-L-homoserine lactone (C4-HSL).

Table.4 Prevalence of genetic determinants involved in quorum sensing

Genes involved in quorum sensing	*P. aeruginosa* (n=30)		*E. coli* (n=20)		Total (n=50)	
	Effective (N)	Prevalence (%)	Effective (N)	Prevalence (%)	Effective (N)	Prevalence (%)
LasI	24	80,0	8	40,0	32	64,0
LasR	24	80,0	8	40,0	32	64,0
RhlI	18	60,0	16	80,0	34	68,0
RhlR	18	60,0	16	80,0	34	68,0

No significant difference was observed between samples ($p > 0.05$).

Gene Las: Encoding for the production of elastase; Rhl: Encoding for the production of rhamnolipids

Figure.1 *P. aeruginosa* (A) and *E. coli* (B) at x100 magnification

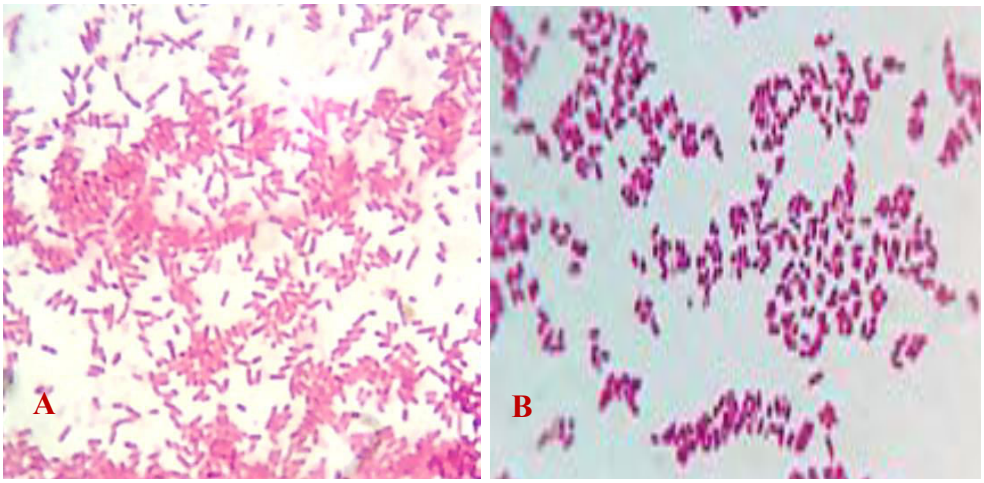


Figure.2 Multidrug resistance of *P. aeruginosa* (A) and *E. coli* (B) to antibiotics

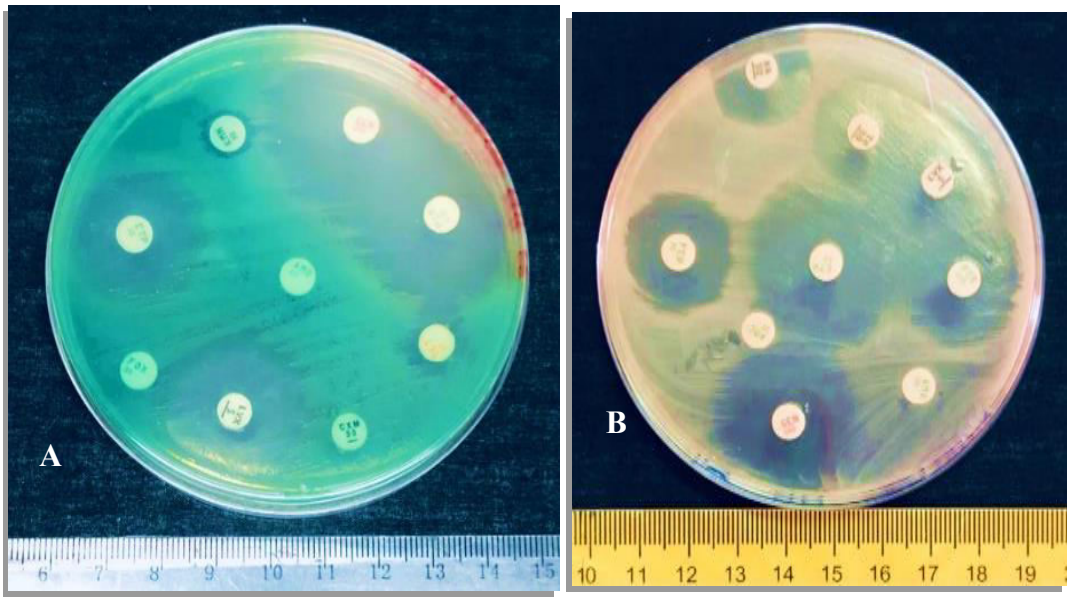
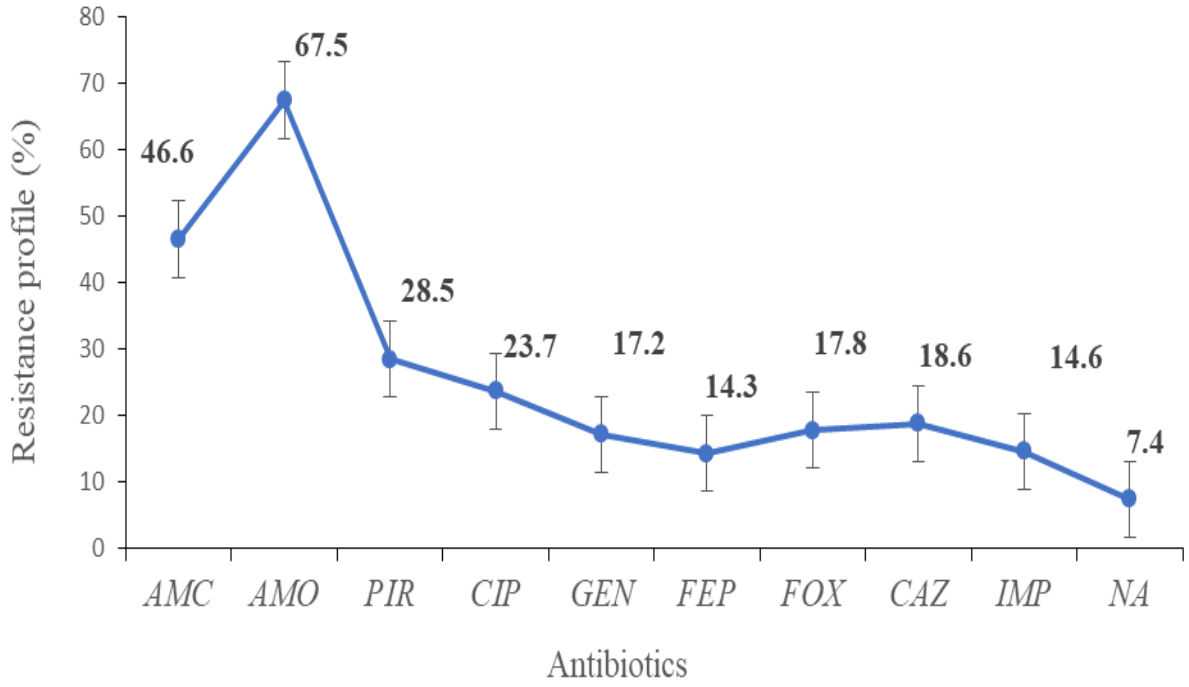
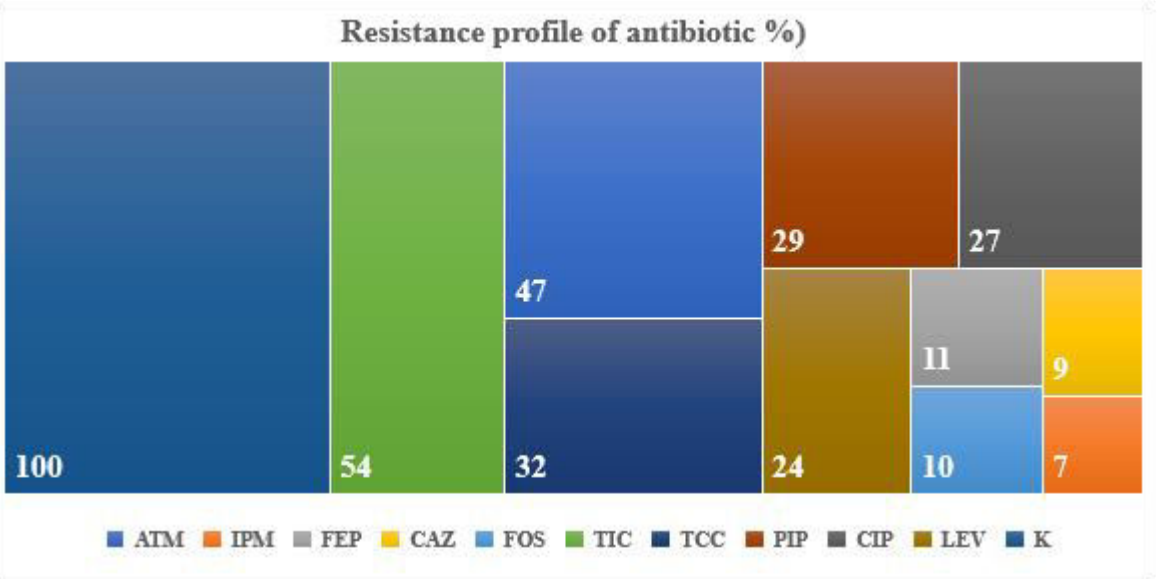


Figure.3 Resistance profile of *E. coli* strains involved in quorum sensing



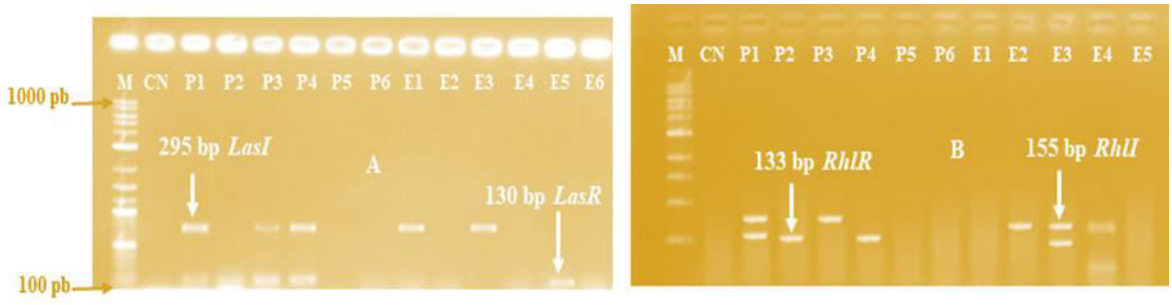
Amoxicillin-Clavulanic acid (AMC), Amoxicillin (AMO), Piperacillin (PIR), Ciprofloxacin (CIP), Gentamicin (GEN), Cefepime (FEP), Cefoxitin (FOX), Ceftazidime (CAZ), Imipenem (IMP), Nalidixic acid (NA)

Figure.4 Resistance profile of *P. aeruginosa* strains involved in quorum sensing



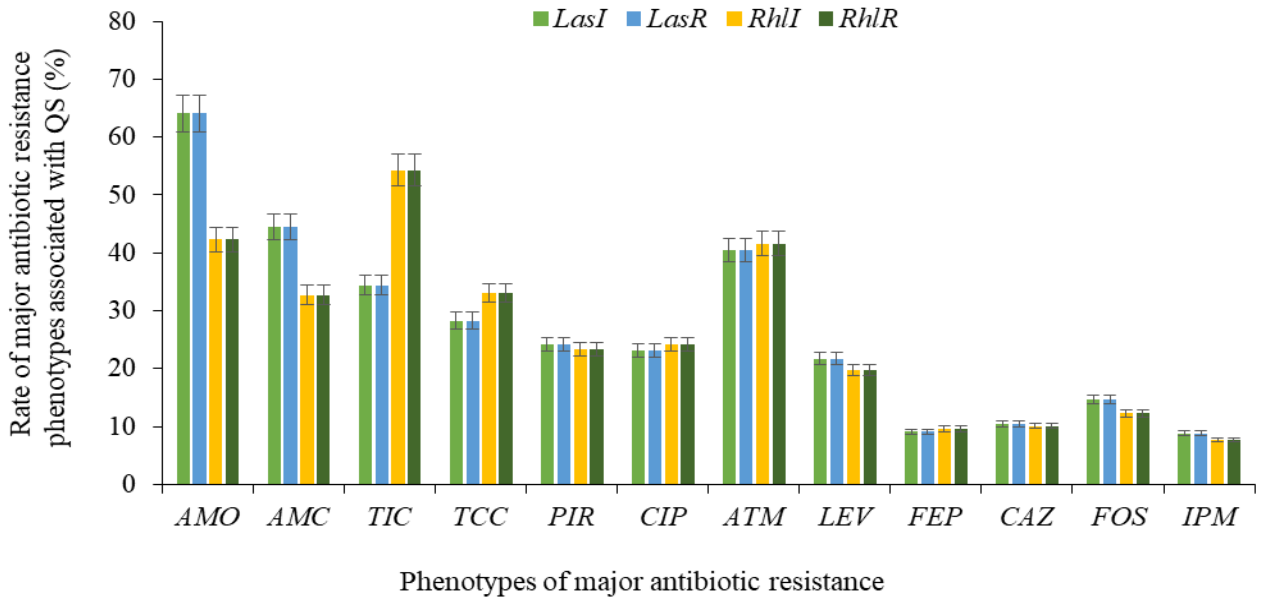
Aztreonam (ATM), Imipenem (IPM), Cefepime (FEP), Ceftazidime (CAZ), Cefoxitin (FOX), Ticarcillin (TIC), Ticarcillin-clavulanic acid (TCC), Piperacillin (PIP), Ciprofloxacin (CIP), Levofloxacin (LVX), Kanamycin (K)

Figure.5 Electrophoretic profile of genes involved in quorum sensing



A: Presence of the LasI gene in P. aeruginosa (P1, P3, P4 lines) and in E. coli (E1, E3 lines); Presence of the LasR gene in P. aeruginosa (P1, P2, P3, P4 lines) and in E. coli (E5 line). B: Presence of the LasI gene in P. aeruginosa (P1, P3 lines) and in E. coli (E2, E3, E4 lines); Presence of the LasR gene in P. aeruginosa (P1, P2, P4 lines) and in E. coli (E3 line). CN: negative control; M: molecular weight marker.

Figure.6 Phenotypes of major antibiotic resistance associated with quorum sensing



Amoxicillin-Clavulanic Acid (AMC), Amoxicillin (AMO), Aztreonam (ATM), Imipenem (IPM), Cefepime (FEP), Ceftazidime (CAZ), Cefoxitin (FOX), Ticarcillin (TIC), Ticarcillin-Clavulanic Acid (TCC), Piperacillin (PIP), Ciprofloxacin (CIP), Levofloxacin (LVX), Gene Las: Encoding for elastase production; Rhl: Encoding for rhamnolipid production

This study finally showed that major resistance phenotypes linked to the *Las* and *Rhl* genes of quorum sensing were primarily associated with penicillin antibiotics, quinolones, and monobactams (Papaneophytou, 2026; Chu and Yang, 2021).

The presence of numerous resistance phenotypes and genetic determinants involved in quorum sensing necessitates improvements in the food production and processing chain, particularly for products of animal origin.

In conclusion, the study indicated that strains of *P. aeruginosa* and *E. coli* isolated from smoked fish harbored genetic markers involved in quorum sensing, including the *Las* (*LasI/LasR*) and *Rhl* (*RhlI/RhlR*) genes, which code for the production of elastase and rhamnolipids, respectively. This study showed that major resistance phenotypes linked to the Las and Rhl genes of quorum sensing were primarily associated with penicillin antibiotics, quinolones, and monobactams.

The study also demonstrated that all these detected genetic determinants could be involved in the resistance, virulence, and pathogenicity of strains of animal origin. Improving the food production and processing chain, and particularly that of products of animal origin, remains essential.

Authors' Contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

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