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The Role of the Quorum Sensing Signaling Pathway in Clinical Drug Resistance of *Escherichia coli*: A Review and Outlook

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Abstract: Quorum sensing (QS) represents a mechanism through which bacteria engage in communication via chemical signals, a phenomenon prevalent across diverse bacterial species. Recent investigations have elucidated that QS signaling pathways are pivotal in governing bacterial physiological processes, collective behaviors, and the emergence of drug resistance. *Escherichia coli* (*E. coli*), a prominent pathogenic bacterium, is increasingly exhibiting severe drug resistance issues, posing substantial hurdles for clinical interventions. Presently, a burgeoning body of research is exploring the connection between QS signaling pathways and the drug resistance mechanisms in E. coli, unveiling the coordinating function of QS within bacterial communities and its influence on antibiotic resistance. Despite some research advancements, the precise mechanisms underlying the QS signaling pathway remain ambiguous, and its potential applications are somewhat constrained. This article endeavors to systematically review the research progress concerning the QS signaling pathway in the context of clinical drug resistance mechanisms in *E. coli*, delving into its potential clinical applications and future research avenues, with the aim of offering novel insights and strategies to counteract drug resistance.

Keywords: Antibiotic resistance; Biofilm; *Escherichia coli*; Quorum sensing; QS inhibitors

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1. Introduction

Escherichia coli (*E. coli*) is a common bacterium found in the intestines of humans and animals. However, certain specific strains are significant pathogens widely associated with foodborne illnesses and hospital-acquired infections. In recent years, with the widespread use of antibiotics, the problem of antibiotic resistance in *E. coli* has become increasingly severe, particularly resistance to β-lactam antibiotics, posing a major challenge to global public health [1,2]. Antibiotic resistance not only leads to treatment failures but also increases patient mortality rates

and healthcare costs. Therefore, exploring new treatment strategies to address antibiotic resistance is a crucial direction in current medical research.

Recent studies have indicated that the quorum sensing (QS) signaling pathway in bacteria may play a significant role in the development of antibiotic resistance. QS is a mechanism by which bacteria regulate group behaviors through the secretion and perception of small molecule signals (such as autoinducers), affecting bacterial growth, pathogenicity, and biofilm formation, among other things [3,4]. In *E. coli*, the activation of the QS signaling pathway is associated with antibiotic resistance, particularly by regulating the expression of genes related to biofilm formation and drug resistance, thereby enhancing the bacteria's resistance to antibiotics [5,6].

Research has found that the *LuxS* gene in *E. coli* plays a crucial role in QS, as the enzyme it encodes is involved in the synthesis of the autoinducer AI-2, which is closely related to bacterial biofilm formation and drug resistance [4, 7]. Furthermore, certain QS inhibitors (such as plant extracts and synthetic small molecules) have been shown to interfere with the QS signaling pathways in *E. coli*, thereby inhibiting biofilm formation and reducing drug resistance [8,9].

Although there is a preliminary understanding of the mechanisms by which QS contributes to drug resistance in *E. coli*, further in-depth research is needed to elucidate the specific molecular mechanisms and signaling pathways involved. This will provide new insights and targets for clinical anti-infective strategies. Future research should focus on developing interventions targeting QS signaling pathways to reduce drug resistance in *E. coli* and enhance the efficacy of existing antibiotics [5,10]. Through this approach, new solutions to the problem of antibiotic resistance may be found, thereby improving patient treatment outcomes and prognosis.

2. Basic mechanisms of the QS signaling pathway

QS is a biological mechanism by which bacteria recognize their population density through the release and perception of signaling molecules (such as autoinducers). This allows bacteria to coordinate their behavior and exhibit collective characteristics, including biofilm formation, expression of virulence factors, and drug resistance. The core mechanisms of QS signaling pathways encompass multiple levels, including the types and functions of signaling molecules, interactions between bacteria, and signal transduction [11,12].

2.1. Types and functions of QS signaling molecules

QS signaling molecules mainly include N-acyl-L-homoserine lactones (AHLs), autoinducer-2 (AI-2), and other small molecules [13]. As the population density of bacteria changes, the concentrations of these signaling molecules also vary, playing a crucial role in physiological activities. For example, AHLs primarily function in Gram-negative bacteria, participating in the regulation of group behaviors and the synthesis of virulence factors. Against this backdrop, AI-2 is regarded as a universal signal among various bacteria, facilitating their mutual communication and the coordination of group behaviors [14].

Moreover, variations in the types and concentrations of signaling molecules can directly influence bacterial growth, metabolic activities, and biological functions such as biofilm formation [15]. These characteristics enable bacteria to adapt to complex environments.

2.2. Interactions and group behaviors

The interactions among bacteria constitute a crucial part of the QS signaling pathway. Relying on the QS

mechanism, bacteria can perceive the presence of other bacteria in their surroundings and adjust their behavior accordingly. For instance, when bacteria are in a high-density environment, they may choose to form biofilms to enhance their survival capabilities; conversely, in low-density conditions, they may remain in a motile state to explore new nutrient sources. The regulation of such group behaviors not only affects bacterial survival strategies but is also closely linked to host immune responses and drug resistance [16]. Research has shown that certain bacteria can inhibit the QS systems of their competitors through QS signaling molecules, thereby enhancing their own competitiveness and demonstrating mechanisms of social adaptation [17].

2.3. Signal transduction mechanisms

The QS signal transduction mechanism involves the interaction of multiple signaling pathways. Bacteria utilize specific receptors to recognize signaling molecules and activate relevant downstream signaling pathways. For example, in *E. coli*, the LuxS/AI-2 system influences bacterial growth and biofilm formation by regulating the expression of various genes [18]. Furthermore, the complexity of signal transduction is also reflected in the interactive behaviors among different bacterial species. Some bacteria can interfere with the QS signals of other bacteria by producing specific QS inhibitors, thereby gaining an advantage in the ecosystem. The diversity of such mechanisms enables bacteria to flexibly respond to complex environmental challenges and optimize their survival strategies [19].

In summary, the QS signaling pathway plays a pivotal role in bacterial physiological activities. The complexity and diversity of its mechanisms offer new perspectives for a deeper understanding of bacterial group behaviors and drug resistance. Future research will continue to unveil the detailed mechanisms of the QS signaling pathway, laying a theoretical foundation for the development of new-generation antimicrobial strategies.

3. Characteristics of QS in E. coli

3.1. Major QS signal molecules

In *E. coli*, the QS signaling pathway primarily relies on AHLs as key signaling molecules. The synthesis of AHLs is catalyzed by LuxI family proteins, and these molecules can transmit signals in a concentration-dependent manner within bacterial populations. Recent studies have revealed the presence of multiple AHLs in *E. coli*, such as C4-HSL and C6-HSL.

When the bacterial population reaches a certain density, these signaling molecules interact and effectively initiate the expression of specific genes. This process is crucial for biofilm formation and the secretion of various virulence factors [20]. Meanwhile, *E. coli* also possesses the SdiA receptor, which can recognize exogenous AHLs, enhancing its adaptability in complex environments and providing advantages in different competitive scenarios. This mechanism demonstrates the potential role of bacteria in developing drug resistance through the QS system, particularly in the evolution of multidrug-resistant strains [21].

3.2. Impact of QS on biofilm formation

During the biofilm formation process in *E. coli*, the QS signaling pathway plays a crucial role. Research has shown that QS-related signaling molecules significantly influence biofilm generation and maturation. For instance, C4-HSL is considered an important component that enhances the biofilm-forming potential of *E. coli*. In high-cell-density environments, bacteria can regulate biofilm-related genes, such as curli and cellulose synthesis genes,

through QS mechanisms, thereby promoting their attachment and aggregation on solid substrates.

Biofilm formation can be divided into multiple stages:

(1) Initial attachment;

(2) Microbial community formation;

(3) Maturation;

(4) Release.

The initial stage of this process involves bacteria binding to solid surfaces through their surface structures, and QS signals enhance the stability of this binding. Henly et al. pointed out that C4-HSL regulates relevant genes by activating specific transcription factors, promoting the early development of biofilms [22]. As the growth density of *E. coli* increases, the activity of QS signals intensifies, further enhancing the thickness and complexity of biofilms and forming a more effective protective barrier, thereby improving its resistance to drugs. Bai et al. also emphasized that QS not only affects bacterial adhesion capacity but also influences biofilm stability by regulating motility. Bacteria enhance the integrity of biofilms by synthesizing exogenous polymers, such as polysaccharides and adhesive proteins. These mechanisms not only facilitate biofilm formation but also endow bacteria with stronger survival competitiveness when facing antibiotic treatment [23].

In recent years, multiple researchers have conducted in-depth explorations of the role of QS signaling pathways in biofilm formation, providing new perspectives for understanding the drug resistance mechanisms of *E. coli*. For instance, Dobretsov et al. confirmed the function of QS signals in regulating intercellular interactions within biofilms [24], Ząbek et al. discovered the crucial role of specific QS molecules in the maturation process of biofilms [25]. These findings not only offer new insights into drug resistance mechanisms but also point the way for future research and development of therapeutic strategies.

4. Relationship between QS and drug resistance

During the survival and adaptation processes of bacteria, the QS mechanism plays a pivotal role. Particularly in *E. coli*, QS not only influences bacterial growth and reproduction but is also closely related to its drug resistance. With the escalating issue of antibiotic resistance, exploring the association between QS and drug resistance has become increasingly important.

4.1. The role of QS in the expression of drug resistance genes

Research has found that the QS system can directly affect bacterial sensitivity to antibiotics by regulating the expression of drug resistance genes [26]. The SdiA receptor in *E. coli* can perceive exogenous signaling molecules and regulates the expression of corresponding drug-resistance genes in the presence of antibiotics.

This mechanism is particularly evident when responding to antibiotics at sub-inhibitory concentrations. The transcriptional response of SdiA exhibits variations under different induction conditions, highlighting the significant role of QS in the regulatory mechanisms of drug resistance [27]. Meanwhile, QS enhances bacterial tolerance to antibiotics by regulating biofilm formation capacity, as bacteria within biofilms are more difficult to eradicate [28].

4.2. Regulation of drug targets by QS

In addition to influencing the expression of drug-resistance genes, the QS system also alters bacterial drug

sensitivity by regulating the expression of drug targets. In *Pseudomonas aeruginosa*, QS signaling molecules can affect the expression of genes related to drug efflux pumps, thereby influencing changes in antibiotic resistance [29].

Intervention strategies targeting the QS system can effectively reduce bacterial resistance to antibiotics. Certain natural compounds have been found to inhibit the production of QS signals, thereby enhancing bacterial sensitivity to antibiotics [30]. This regulatory mechanism provides a novel approach for the development of new antibiotics, namely by targeting the QS system to reduce bacterial resistance.

4.3. Impact of QS on bacterial survival strategies

QS not only affects drug resistance but also has a profound impact on bacterial survival strategies. In environments with high cell density, bacteria regulate group behaviors through QS mechanisms, such as biofilm formation and toxin release, which enhance their survival capabilities. In *E. coli*, QS signaling molecules promote biofilm formation, enabling bacteria to survive and resist antibiotic attacks in unfavorable environments [31].

Additionally, QS is closely related to bacterial metabolic activities and influences their efficiency in utilizing nutrients and their ability to withstand environmental stress [32]. Therefore, interfering with QS signal transmission can effectively alter bacterial survival strategies, reduce their pathogenicity and resistance trends, and provide new approaches for infection treatment.

5. Recent research advances

5.1. Latest achievements in research on QS signaling pathways

In recent years, significant progress has been made in the study of QS signaling pathways in *E. coli* and its related pathogens. Current research indicates that these signaling pathways play a pivotal role in various aspects of bacterial behavior, including group dynamics, pathogenicity, and antibiotic resistance. The AHL-type QS system found in *Acinetobacter baumannii* effectively regulates its biological functions and pathogenicity, offering a new perspective for understanding its role in clinical infections [33]. Additionally, QS signals have a profound impact on bacterial growth, metabolism, and biofilm formation, contributing to increased antibiotic resistance during treatment [34].

In the study of *E. coli*, researchers have identified new QS signaling molecules and their associated regulatory mechanisms. These signaling molecules play a crucial role in regulating biofilm formation and the production of virulence factors in bacteria. The discovery of the LuxS enzyme has highlighted its importance in QS and bacterial growth regulation, with the presence of autoinducer AI-2, it catalyzes being considered a key mediator for effective bacterial communication [35].

Through comparative studies of various strains, scholars have revealed the diversity of QS signaling pathways and their adaptability in different environments, laying a theoretical foundation for developing new antimicrobial strategies [21].

5.2. Clinical case analysis of antibiotic resistance

The issue of clinical antibiotic resistance in *E. coli* has become increasingly prominent. Research shows that QS signaling pathways play a critical role in the development of bacterial resistance. Studies on *Lactobacillus rhamnosus* GG with a deleted *LuxS* gene have demonstrated a significant reduction in its resistance to intestinal pathogens such as Enterotoxigenic *E. coli* (ETEC). This finding underscores the importance of QS signals in host

immune regulation [35].

Further analysis of the drug resistance of different clinical isolates revealed a positive correlation between the expression levels of QS genes and bacterial drug resistance. In particular, among multidrug-resistant *Pseudomonas aeruginosa*, a clear association was observed between the high expression of QS genes and biofilm formation as well as drug resistance [36].

Researchers have found that specific QS signaling molecules, such as AI-2, play a facilitating role in the process of bacterial tolerance to β-lactam antibiotics. This suggests that by regulating QS signaling pathways, it may be possible to reduce bacterial resistance to antibiotics, thereby enhancing their efficacy [37].

These research findings not only provide a new perspective for exploring bacterial resistance mechanisms but also lay an important scientific foundation for formulating clinical treatment plans.

5.3. Development and application prospects of QS inhibitors

In response to the challenges posed by bacterial resistance and biofilm-related infections, the development of QS inhibitors has become a research hotspot. In recent years, researchers have explored the potential of various natural and synthetic compounds as QS inhibitors. For instance, studies have shown that certain plant extracts (such as eucalyptus oil and cinnamon oil) exhibit significant QS inhibitory activity against multidrug-resistant *Pseudomonas aeruginosa*. These natural products reduce bacterial biofilm formation and the production of virulence factors by interfering with QS signaling pathways [38,39].

Additionally, synthetic QS inhibitors (such as certain small-molecule compounds) also show promising clinical application prospects. Research indicates that these compounds can effectively inhibit bacterial QS signaling, thereby enhancing the efficacy of antibiotics. For example, some novel compounds have demonstrated strong anti-biofilm capabilities in vitro and have shown potential to reduce infection severity in animal models [40].

In the future, with a deeper understanding of QS mechanisms and the integration of modern biotechnology with drug development approaches, QS inhibitors are expected to become a new strategy for combating bacterial infections.

6. Future research directions and clinical prospects

6.1. Improved experimental models and techniques

When exploring the role of the QS signaling pathway in clinical drug resistance of *E. coli*, improved experimental models and techniques are of paramount importance. Traditional experimental models often fail to fully simulate the complex microbial environment within the human body, leading to limitations in research findings.

Therefore, future research should focus on developing more precise *in vivo* and *in vitro* models. For instance, leveraging three-dimensional cell culture techniques and microfluidic chip technology can better mimic the growth and interactions of bacteria under physiological conditions [41].

These technologies provide a more authentic microenvironment, enabling researchers to observe bacterial behavior under different environmental conditions and their responses to antibiotics. Additionally, gene editing techniques (such as CRISPR-Cas9) can be employed to precisely regulate key genes within the QS signaling pathway, thereby aiding in the investigation of their specific roles in drug resistance mechanisms [42].

The introduction of these new technologies will significantly advance our understanding of the QS signaling pathway and offer novel insights for the development of anti-drug resistance strategies [30,31].

6.2. Controversies and challenges surrounding the QS signaling pathway

Although the QS signaling pathway plays a crucial role in regulating bacterial group behaviors, its specific role in clinical drug resistance remains controversial. Different research findings suggest that the QS signaling pathway may promote bacterial resistance in some cases, while in others, it may inhibit the development of resistance. These contradictory results may stem from variations in bacterial species, environmental factors, and their interactions under different experimental conditions. Furthermore, the complexity and diversity of the QS signaling pathway pose challenges for researchers in determining its specific mechanisms.

Future research needs to systematically analyze the QS signaling pathways of different bacterial species, particularly in clinically relevant environments, to better understand their roles in the development of drug resistance. Additionally, researchers should also pay attention to the diversity of QS signaling molecules and their variations under different physiological states, which will provide a basis for formulating targeted treatment strategies [43,44].

6.3. Possibilities for multidisciplinary interdisciplinary research

Interdisciplinary research has demonstrated tremendous potential in uncovering the relationship between QS signaling pathways and antibiotic resistance in *E. coli*. By integrating knowledge from multiple fields, including microbiology, molecular biology, pharmacology, and clinical medicine, researchers can explore the functions of QS signaling pathways and their impact on antibiotic resistance in a more systematic and comprehensive manner. For instance, advancements in microbiomics have enabled researchers to gain a deeper understanding of the interactions between host microbial communities and QS signaling pathways, providing new perspectives for elucidating bacterial resistance mechanisms.

Meanwhile, innovations in drug development, such as QS signaling pathway inhibitors based on small-molecule compounds, have opened up new prospects for anti-resistance strategies. Through close interdisciplinary collaboration, research teams can design more practical experimental protocols and explore innovative therapeutic approaches, thereby providing more effective support and guidance for clinical disease intervention [45,46].

7. Conclusion

The resolution of the antibiotic resistance issue necessitates multidisciplinary cooperation and efforts, encompassing coordination in basic research, clinical applications, and public health policies. By synthesizing perspectives and findings from diverse studies, we can more effectively tackle this global public health challenge. It is hoped that future research will further unravel the complexity of QS signaling pathways, providing us with more effective anti-infective strategies and ultimately improving patient prognosis and health outcomes.

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References

[1] Brink K, Hunt M, Mu A, et al., 2023, An E. coli Display Method for Characterization of Peptide-Sensor Kinase Interactions. Nat Chem Biol, 19(4): 451–459.

[2] Bourigault Y, Rodrigues S, Crépin A, et al., 2021, Biocontrol of Biofilm Formation: Jamming of Sessile-Associated Rhizobial Communication by Rhodococcal Quorum-Quenching. International Journal of Molecular Sciences, 22(15): 8241.

[3] Cui B, Chen X, Guo Q, et al., 2022, The Cell-Cell Communication Signal Indole Controls the Physiology and Interspecies Communication of Acinetobacter baumannii. Microbiol Spectr, 10(4): e102722.

[4] Fernandex-Ciruelos B, Potmis T, Solomin V, et al. 2023, Cross-Talk Between QseBC and PmrAB Two-Component Systems is Crucial for Regulation of Motility and Colistin Resistance in Enteropathogenic Escherichia coli. PLoS Pathog, 19(12): e1011345.

[5] Othman A, Shamekh I, Abdalla M, et al., 2023, Molecular Modeling Study of Micro and Nanocurcumin with In Vitro and In Vivo Antibacterial Validation. Sci Rep, 13(1): 12224.

[6] Sadeeshkumar H, Balaki A, Sutherland A, et al. 2023, Screening for Small Molecule Inhibitors of SAH nucleosidase using an SAH riboswitch. Anal Biochem, 2023(666): 115047.

[7] Mukheef S, Eleroui M, Al-Dahmoshi H, et al., 2022, Prevalence of Some Group A Beta-Lactamase Genes among Uropathogenic Escherichia coli isolated from Women with Cystitis. Cell Mol Biol (Noisy-le-grand), 68(10): 84–89.

[8] Hu J, Che C, Jiang W, et al., 2022, Avian Pathogenic Escherichia coli through Pfs Affects the Transcription of Membrane Proteins to Resist β-Lactam Antibiotics. Veterinary sciences, 9(3): 98.

[9] Xu F, Jiang M, Li D, et al., 2024, Protective Effects of Antibiotic Resistant Bacteria on Susceptibles in Biofilm: Influential Factors, Mechanism, and Modeling. Sci Total Environ, 2024(930): 172668.

[10] Soliman A, Shaaban M, Dawood A, et al., 2024, Microbiological Studies on Escherichia coli and Klebsiella pneumoniae Causing Vaginal and Urinary Tract Inflammation; Prevelance, Antibiotics-Resistance and Natural Products Susceptibility. Microb Pathog, 2024(196): 107000.

[11] Haque S, Yadav D, Bisht S, et al., 2019, Quorum Sensing Pathways in Gram-Positive and -Negative Bacteria: Potential of Their Interruption in Abating Drug Resistance. J. Chemother, 31(4): 161–187.

[12] Shaaban M, Elgaml A, Habib E, 2019, Biotechnological Applications of Quorum Sensing Inhibition as Novel Therapeutic Strategies for Multidrug Resistant Pathogens. Microb Pathog, 2019(127): 138–143.

[13] Truchado P, Gil-Izquierdo A, Tomas-Barberan F, et al., 2009, Inhibition by Chestnut Honey of N-Acyl-L-Homoserine Lactones and Biofilm Formation in Erwinia carotovora, Yersinia enterocolitica, and Aeromonas hydrophila. J Agric Food Chem, 57(23): 11186-11193.

[14] Daniels R, Vanderleyden J, Michels J, 2004. Quorum Sensing and Swarming Migration in Bacteria. FEMS Microbiol Rev, 28(3): 261–289.

[15] Nassif N, Bouvet O, Noelle R, et al., 2002, Living Bacteria in Silica Gels. Nat Mater, 1(1): 42–44.

[16] Miller M, Bassler B, 2001, Quorum Sensing in Bacteria. Annu Rev Microbiol, 2001(55): 165–199.

[17] Huang S, Liu X, Yang W, et al., 2022, Insights into Adaptive Mechanisms of Extreme Acidophiles Based on Quorum Sensing/Quenching-Related Proteins. mSystems, 7(2): e149121.

[18] Kaur A, Capalash N, Sharma P, 2020, Expression of Meiothermus ruber luxS in E. coli alters the antibiotic susceptibility and biofilm formation. Appl Microbiol Biotechnol, 104(10): 4457–4469.

[19] Nepomuceno V, Tylor K, Carlson S, et al., 2023, A Streptomyces tendae Specialized Metabolite Inhibits Quorum Sensing in Group A Streptococcus. Microbiol Spectr, 11(4): e527922.

[20] Deshamukhya C, Begom F, Das B, et al., 2024, Imipenem exposure influences the expression of quorum-sensing receptor sdiA in Escherichia coli. FEMS Microbiol Lett, 2024: 371.

[21] Grabski H, Ginosyan S, Tiratsuyan S, 2021, Molecular Simulations and Markov State Modeling Reveal Inactive Form of Quorum Sensing Regulator SdiA of Escherichia Coli. IEEE/ACM Trans Comput Biol Bioinform, 18(6): 2835–2840.

[22] Henly E, Norris K, Rawson K, et al., 2021, Impact of Long-Term Quorum Sensing Inhibition on Uropathogenic Escherichia coli. Antimicrob Chemother, 76(4): 909–919.

[23] Bai Y, Shi M, Wang W, et al., 2022, Novel Quorum Sensing Inhibitor Echinatin as an Antibacterial Synergist Against Escherichia coli. Front Microbiol, 2022(13): 1003692.

[24] Dobretsov S, Teplitski M, Paul V, 2009. Mini-Review: Quorum Sensing in the Marine Environment and Its Relationship to Biofouling. Biofouling, 25(5): 413–427.

[25] Zabek A, Junka A, Szymczyk P, et al., 2017, Metabolomics Analysis of Fungal Biofilm Development and of Arachidonic Acid-Based Quorum Sensing Mechanism. J Basic Microbiol, 57(5): 428–439.

[26] Pumirat P, Santajit S, Tunyong W, et al., 2024, Impact of AbaI Mutation on Virulence, Biofilm Development, and Antibiotic Susceptibility in Acinetobacter baumannii. Sci Rep, 14(1): 21521.

[27] Hai H, Yang M, Cheng Z, et al., 2024, Potential Role of SdiA in Biofilm Formation and Drug Resistance in Avian Pathogenic Escherichia coli. Animals, 14(15): 2199.

[28] Reen F, Gutierrez-Barranquero J, Parages M, et al., 2018, Coumarin: A Novel Player in Microbial Quorum Sensing and Biofilm Formation Inhibition. Appl Microbiol Biotechnol, 102(5): 2063–2073.

[29] Ahmed F, Aly U, Abd R, et al., 2021, Effect of Titanium Dioxide Nanoparticles on the Expression of Efflux Pump and Quorum-Sensing Genes in MDR Pseudomonas aeruginosa Isolates. Antibiotics, 10(6): 625.

[30] Cui S, Kim E, 2024, Quorum Sensing and Antibiotic Resistance in Polymicrobial Infections. Commun Integr Biol, 17(1): 2415598.

[31] Tonkin M, Khan S, Wani M, et al., 2021, Quorum Sensing - A Stratagem for Conquering Multi-Drug Resistant Pathogens. Curr Pharm Des, 27(25): 2835–2847.

[32] Goo E, An J, Kang Y, et al., 2015, Control of Bacterial Metabolism by Quorum Sensing. Trends Microbiol, 23(9): 567–576.

[33] Mayer C, Borges A, Flament-Simon S, et al., 2023, Quorum Sensing Architecture Network in Escherichia coli Virulence and Pathogenesis. FEMS microbiology reviews, 47(4): 31.

[34] Sikdar R, Elias M, 2022, Evidence for Complex Interplay between Quorum Sensing and Antibiotic Resistance in Pseudomonas aeruginosa. Microbiol Spectr, 10(6): e126922.

[35] Deng Z, Hou K, Valencak T, et al., 2022, AI-2/LuxS Quorum Sensing System Promotes Biofilm Formation of Lactobacillus rhamnosus GG and Enhances the Resistance to Enterotoxigenic Escherichia coli in Germ-Free Zebrafish. Microbiol Spectr, 10(4): e61022.

[36] Dong L, Sun L, Hu X, et al., 2021, Ostarine Attenuates Pyocyanin in Pseudomonas aeruginosa by Interfering with Quorum Sensing Systems. Antibiot, 74(12): 863–873.

[37] Asihwarya S, Gunasekaran K, Kumar P, et al., Structural, Functional, Resistome and Pathogenicity Profiling of the Cooum River. Microb Pathog, 2021(158): 105048.

[38] Sagar P, Sharma P, Singh R, 2022, Inhibition of Quorum Sensing Regulated Virulence Factors and Biofilm Formation by Eucalyptus globulus against Multidrug-Resistant Pseudomonas aeruginosa. J Pharmacopuncture, 25(1): 37–45.

[39] Rajkhowa S, Hussain S, Agarwal M, et al., 2024, Advancing Antibiotic-Resistant Microbe Combat: Nanocarrier-Based Systems in Combination Therapy Targeting Quorum Sensing. Pharmaceutics, 16(9): 1160.

[40] Khoshbakht R, Panahi S, Neshani A, et al., 2023, Novel Approaches to Overcome Colistin Resistance in Acinetobacter baumannii: Exploring Quorum Quenching as a Potential Solution. Microb Pathog, 2023(182): 106264.

[41] Zhou X, Zhu L, Li W, et al., 2022, An Integrated Microfluidic Chip for Alginate Microsphere Generation and 3D Cell Culture. Anal Methods, 14(12): 1181–1186.

[42] Fan Y, Tan F, He X, et al., 2025, CitACOS5, A Fatty Acyl-CoA Synthetase, is Crucial for Male Fertility in Citrus by Influencing Pollen Exine Formation. J Exp Bot, 76(14): 4027–4042.

[43] Schuster M, Li C, Smith P, et al., 2023, Parameters, Architecture and Emergent Properties of the Pseudomonas aeruginosa LasI/LasR Quorum-Sensing Circuit. J R Soc Interface, 20(200): 20220825.

[44] Xiao Y, Zou H, Li J, et al., 2022, Impact of Quorum Sensing Signaling Molecules in Gram-Negative Bacteria on Host Cells: Current Understanding and Future Perspectives. Gut Microbes, 14(1): 2039048.

[45] Winters M, Arneborh N, Appels R, et al., 2019, Can Community-Based Signalling Behaviour in Saccharomyces cerevisiae be Called Quorum Sensing? A Critical Review of the Literature. FEMS yeast research, 19(5): 46.

[46] Saha S, Aggarwal S, Singh D, 2023, Attenuation of Quorum Sensing System and Virulence in Vibrio cholerae by Phytomolecules. Front Microbiol, 2023(14): 1133569.