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# Effect of Quorum Quenching on Antimicrobial Resistance in Bacteria

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**A B S T R A C T**

Antimicrobial resistance (AMR) occurs when bacteria evolve to survive drugs designed to kill them. It is an emerging global health crisis driven by the overuse and misuse of antibiotics. This review examines how bacteria develop resistance, form biofilms, and coordinate virulence through quorum sensing (QS), and evaluates quorum quenching (QQ) as a strategy to combat resistant infections. Biofilms protect bacteria by surrounding them with an extracellular matrix that limits antibiotic penetration and helps them evade host immune defense. Through QS, bacteria regulate biofilm formation, virulence, and other collective behaviors by producing and responding to signaling molecules. QQ disrupts these signaling pathways by enzymatically degrading signaling molecules, inhibiting signal synthesis, and antagonizing receptors. By interfering with bacterial communication, QQ weakens biofilms, enhances antibiotic efficacy, and may reduce the development of resistance to antibiotics. Further research is needed to translate QQ-based approaches into effective and sustainable therapies for resistant bacterial infections in vivo.

## Introduction

AMR is a growing global health challenge that poses a significant threat to the treatment of infectious diseases worldwide.

Bacteria develop resistance through mechanisms that reduce the effectiveness of antimicrobial agents, such as antibiotics, often acquiring these traits through horizontal gene transfer or spontaneous mutations (1).

The widespread overuse and misuse of antibiotics has greatly accelerated the emergence of drug-resistant pathogens, compromising standard treatment protocols and increasing morbidity and healthcare costs (2).

QS drives bacterial pathogenicity by enabling bacteria to communicate through quorum-sensing molecules (QSM).

As these signaling molecules accumulate, they regulate the expression of virulence factors and promote biofilm formation (3). By coordinating these activities, QS helps bacterial populations adapt to unfavorable conditions, including antibiotic pressures.

QQ has emerged as a promising strategy to combat AMR by disrupting QS pathways (4). By interfering with bacterial communication, QQ suppresses virulence factor expression and biofilm-associated resistance mechanisms without directly killing bacteria, thereby reducing the selective pressure that drives resistance development (5).

## Antibiotics & Resistance

Antibiotics are antimicrobial substances that target bacteria and are the most effective agents for treating bacterial infections (6). Scientists derive antibiotics from fungi, molds, and bacteria or produce them synthetically and semisynthetically. Antibiotics either inhibit bacterial growth or kill bacteria and therefore fall into two broad categories: bacteriostatic and bactericidal.

- **Bacteriostatic drugs**, which inhibit the growth of the organism (7).
- **Bactericidal drugs**, which actually kill the organism (7).

However, this distinction is not absolute. Drug concentration, bacterial species, and growth stage influence antibiotic activity (8).

Bartolomeo Gosio first reported mycophenolic acid in 1893 after isolating the compound from Penicillium glaucum while studying pellagra (9). However, the scientific community largely overlooked this discovery, likely because Gosio published his findings in Italian. Researchers rediscovered the compound in the United States in 1913; however, its therapeutic potential was not recognized. Consequently, mycophenolic acid has not been used in clinical practice. Consequently, scientists often regard penicillin, discovered by Sir Alexander Fleming in 1928 (10), as the first antibiotic because it was the first to undergo widespread clinical development and use.

Antibiotic resistance occurs when bacteria survive exposure to antibiotics that would normally inhibit or kill them (11).

In 2019, the World Health Organization (WHO) identified AMR as one of the world's most serious health threats, placing it alongside Ebola and HIV. According to a Lancet study, AMR contributed to more than 1 million deaths annually between 1990 and 2021 (12). In 2021 alone, AMR contributed to approximately 4.71 million deaths, including 1.14 million deaths directly caused by resistant bacterial infections (12).

Global antibiotic consumption increased by 20.9% between 2016 and 2023 (13), although this increase was lower than the 35.5% increase recorded between 2008 and 2015 (14). The COVID-19 pandemic likely slowed down antibiotic consumption during this period. By 2023, global antibiotic use had reached 49.3 billion Defined Daily Doses (DDDs), up from 40.8 billion DDDs in 2016 (13). Current projections estimate that antibiotic consumption could rise by another 52.3% and reach 75.1 billion DDDs by 2030 (13). This growing reliance on antibiotics drives AMR. Physicians sometimes prescribe antibiotics before confirming a bacterial infection, unnecessarily exposing patients to these drugs (2). Many patients also fail to complete the prescribed treatment courses and later use leftover antibiotics for unrelated illnesses. In India, particularly in the northern regions, many individuals seek treatment directly from local chemists instead of consulting physicians. Chemists often dispense antibiotics without confirming bacterial infections, further accelerating the spread of antimicrobial resistance (15).

## Mechanisms of Resistance

With constant exposure to antibiotics, bacterial pathogens evolve diverse defense mechanisms that help them survive antimicrobial treatment (16). Understanding these mechanisms is essential for developing novel strategies to combat drug resistance. The mechanisms include the following:

- **Enzymatic degradation of antibiotics**: Bacteria produce enzymes that chemically modify or degrade antibiotics, preventing the drugs from interacting with their target sites (17). These enzymes alter the structures of antibiotics through processes such as phosphorylation, adenylation, and acetylation, rendering the drugs ineffective.
- **Modifications to the target site of a drug**: Drug-resistant pathogens can modify antibiotic target sites, preventing effective drug-target interactions. Bacteria achieve this through mechanisms such as methylation of target molecules, structural alterations of cellular targets, and point mutations in the genes encoding these targets (18).
- **Modification of cell permeability**: To exert their effects, antibiotics must enter bacterial cells and interact with specific target molecules (19). Many bacteria reduce antibiotic uptake by altering their cell permeability. They often achieve this by reducing or variably regulating porin expression, thereby limiting drug entry (19).
- **Efflux pumps**: Bacteria use membrane-spanning efflux pumps to actively expel antibiotics and other toxic compounds before they reach their target sites. Importantly, efflux pumps interact with QS systems.

They influence biofilm formation by mediating adherence, modulating QS pathways, and regulating the expression of biofilm-associated genes (20, 21).

- **Biofilm formation**: Bacteria form structured communities encased within a self-produced extracellular polymeric substance (EPS) matrix (22). This matrix anchors biofilms to biotic and abiotic surfaces while protecting resident cells from environmental stressors, antibiotics, and host immune defense (23). Consequently, biofilms reduce antibiotic efficacy by limiting drug penetration into the bacterial community.

Horizontal gene transfer (HGT) contributes to the spread of antibiotic resistance among bacterial populations (1). During HGT, bacteria transfer genetic material without reproduction through three primary mechanisms:

- **Conjugation**, transfer of genetic material via cell-to-cell contact via the pilus
- **Transformation**, uptake of genetic material from the environment
- **Transduction**, transfer of genetic material via bacteriophages.

Although HGT does not directly generate resistance mechanisms, it enables bacterial populations to acquire and disseminate resistance determinants.

## Biofilms

As previously noted, microorganisms form biofilms by embedding themselves within an extracellular polymeric substance (EPS) matrix that protects them from harsh environmental conditions, including temperature and pH fluctuations, high osmotic pressure, nutrient scarcity, and antibiotics, by limiting antibiotic penetration (24, 25). Additionally, biofilm-forming bacteria often reduce their metabolic activity, which decreases the effectiveness of antibiotics (26). Some cells also enter a dormant state, making them less susceptible to treatment because most antibiotics primarily target actively growing organisms (8).

Biofilms can contain a single microbial species or a mixture of bacteria, fungi, archaea, protozoa, and yeast (25). Microorganisms form these structures through a complex and tightly regulated process involving cell adhesion, EPS production, and eventual detachment from the biofilm. They also regulate the expression of specific genes during this process (27).

The EPS matrix forms a highly hydrated three-dimensional structure, in which water accounts for approximately 97% of the total composition. The remaining 3% consists of nucleic acids, proteins, polysaccharides and lipids. Biofilms also contain insoluble structures, such as amyloids, fimbriae, and flagella (18). Figure 2 shows the basic structure.

Although biofilm formation often characterizes bacterial communities, researchers should not regard biofilms as structures that require eradication. Biofilms contribute significantly to ecological and industrial processes, such as wastewater treatment and bioremediation, demonstrating that they do not inherently harm the environment (24).

## Quorum Sensing

Complex microbial communities rely on effective communication to coordinate their collective behaviors. Through quorum sensing (QS), bacteria monitor their population density and coordinate gene expression (3). Bacteria achieve this by releasing quorum-sensing molecules (QSM) into the extracellular environment. As cell density increases, these signaling molecules accumulate and, upon reaching a threshold concentration, bind to specific receptors that trigger signaling cascades and alter gene expression (28).

Through QS, bacteria regulate biofilm formation, secrete extracellular enzymes, produce antimicrobial compounds and express virulence factors (29, 30, 31). Gram-negative and Gram-positive bacteria use different signaling molecules because of the differences in their cellular structures.

In Gram-negative bacteria, such as *E. coli*, acyl-homoserine lactones (AHLs) mediate QS signaling (28), whereas autoinducer peptides (AIPs) mediate communication in gram-positive bacteria (33).

In addition, autoinducer-2 (AI-2) facilitates both inter- and intraspecies communication. Unlike AHLs and AIPs, AI-2 functions as a non-species-specific signaling molecule that enables communication between diverse bacterial species regardless of their gram classification (34). Because these signaling molecules regulate biofilm formation and virulence, targeting them offers a promising strategy for disrupting bacterial communication and treating difficult-to-treat infections (35).

## Quorum Quenching

By disrupting QS pathways, QQ weakens biofilm integrity and increases bacterial susceptibility to antibiotics and host immune defense (36). QQ has emerged as a promising strategy for controlling QS-mediated pathogens (37).

It disrupts or inhibits bacterial communication through several mechanisms, including the enzymatic degradation of signaling molecules, inhibition of signal synthesis, and receptor antagonism (4).

- **Enzymatic degradation of signalling molecules**: Certain bacteria produce enzymes that degrade QSM and disrupt bacterial communication. A recent study identified a strain isolated from activated sludge, W-7-EU, as *Pseudomonas nitroreducens*, which degrades N-(3-oxododecanoyl)-L-homoserine lactone (32). Lactonase treatment alone reduced biofilm formation by 69–77% in MDR *P. aeruginosa* isolates. When combined with ciprofloxacin and gentamicin, lactonase increased biofilm susceptibility by more than 3–12 fold and significantly reduced virulence factor production (38).
- **Inhibition of signal synthesis**: AI-2 functions as a universal signaling molecule that facilitates both interspecies and intraspecies communication in bacteria. In addition to coordinating communication and biofilm formation, AI-2 regulates virulence factor production (39). Consequently, inhibiting AI-2 synthesis can attenuate bacterial virulence (37). Quercetin, a natural flavonoid with QS-inhibitory properties, reduced biofilm formation and cell viability by more than 80% in MDR *P. aeruginosa* strains when combined with antibiotics such as levofloxacin and gentamicin. It also significantly reduced infection-associated cell killing in vitro (40).
- **Receptor antagonism**: This strategy blocks QS receptors and prevents bacteria from detecting and responding to their signaling molecules. Researchers achieve this through competitive or allosteric inhibition (5).
    - **Competitive inhibition**: Molecules that structurally resemble QSM compete for binding at receptor active sites, preventing QSM from activating the receptor. For example, aryl β-keto esters compete with AHL for receptor binding in *Vibrio harveyi*, disrupting QS-regulated behaviors (41). Similarly, a marine-derived QS inhibitor from *Pseudoalteromonas* sp. reduced biofilm biomass by 63% during formation and by 33% in mature biofilms. When combined with tobramycin, it increased antibiotic efficacy tenfold and reduced the MIC against *P. aeruginosa* from 0.75 to 0.075 µg/mL (42).
    - **Allosteric inhibition**: In this mechanism, antagonists bind to sites distinct from the active site. This binding induces conformational changes that reduce receptor affinity for its natural ligand and impair QS signaling (36).

Through these mechanisms, QQ offers a promising approach for treating resistant and difficult-to-treat bacterial infections by disrupting bacterial communication rather than directly killing bacteria.

Consequently, QQ reduces the selective pressure that drives the acquisition and spread of resistance determinants within bacterial populations (35).

One frequently cited advantage of QQ over conventional antibiotics is its lower potential to drive resistance development. Because QQ targets virulence rather than essential growth processes, it exerts considerably less selective pressure on bacterial populations.

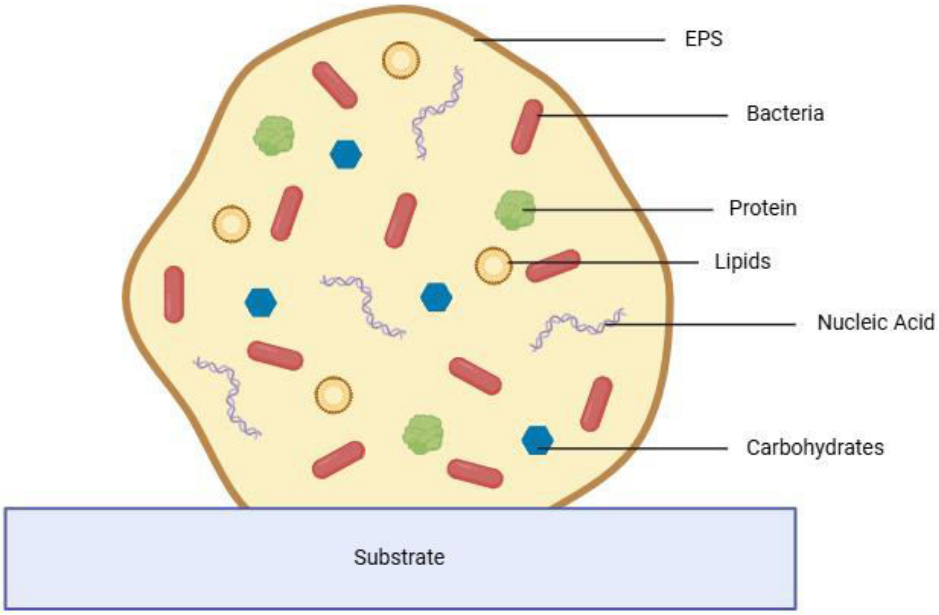
In contrast, conventional antibiotics strongly favor the survival and proliferation of resistant variants, allowing a single resistant bacterium to spread rapidly throughout a population. By avoiding direct bacterial killing, antivirulence approaches substantially reduce this selective pressure (36).

Nevertheless, resistance to QQ cannot be completely excluded. Many mechanisms that enable resistance to conventional antibiotics could also affect QS disruption, particularly genetic variation in QS components responsible for signal production, detection, and transduction (43). Computational models suggest that bacterial populations can adapt to QQ by reaching quorum at lower cell densities (44).

However, recent experimental studies demonstrate that resistance to QS inhibition spreads more slowly during host infection than antibiotic resistance (44).

These findings suggest that although bacteria can develop resistance to QQ, QQ retains a practical advantage in clinical settings. Consequently, researchers should deploy QQ agents alongside antibiotics rather than as standalone therapies.

**Fig.1** A basic structure of a Biofilm along with the various constituents




## Future Prospects

Future research should develop QQ agents that target QS signaling molecules with greater specificity and efficacy. Although current QQ enzymes, such as AHL-lactonases and AHL-acylases, efficiently degrade signaling molecules, they often lack stability in vivo and may affect both harmful and beneficial microbes. Researchers can improve these enzymes through techniques such as directed evolution and site-directed mutagenesis to selectively target harmful signaling molecules with greater efficiency (45).

Combining QQ agents with antibiotics represents another promising strategy for treating drug-resistant infections. By disrupting QS, QQ agents enhance antibiotic penetration and reduce the concentrations required to eliminate biofilm-associated bacteria (40). For example, combining lactonase-based QQ enzymes with ceftiofur significantly increases antibiotic efficacy against resistant *Pseudomonas aeruginosa* biofilms (46).

Lactonase-based QQ enzymes also exhibit low toxicity toward human cells and synergize strongly with aminoglycoside antibiotics in animal infection models. YtnP lactonase from *Bacillus paralicheniformis*, when combined with tobramycin or gentamicin, achieved 100% survival in zebrafish models of *P. aeruginosa* infection, while toxicity studies detected no harmful effects on human cells (47). Similarly, inhaled lactonase SsoPox-I reduced mortality in a rat model of *P. aeruginosa* pneumonia from 75% to 20% (48), supporting its potential for future clinical application.

However, researchers must still develop non-invasive delivery methods that more closely mimic human clinical administration. To maximize clinical efficacy, researchers must also develop effective delivery systems for QQ agents. Liposomes, niosomes, and polymer-based nanoparticles can protect QQ enzymes from degradation and facilitate sustained release at infection sites (49).

Despite encouraging findings, several important questions remain unanswered. Researchers have not yet determined whether long-term exposure to QQ agents will alter microbial community composition or affect beneficial microbiota. Additionally, the pharmacokinetics, optimal dosing strategies, and long-term safety profiles of many QQ agents remain poorly

understood. Addressing these knowledge gaps will be essential before QQ-based therapies can achieve widespread clinical application.

Nevertheless, current evidence suggests that QQ-based approaches could improve treatment outcomes, enhance antibiotic efficacy, and reduce the development and spread of antimicrobial resistance. Continued research remains essential for translating QQ-based therapies into sustainable antimicrobial solutions.

In conclusion, AMR poses a growing global health challenge. In India, the widespread availability of over-the-counter antibiotics continues to drive resistance through the overuse and misuse of these drugs (15).

At the same time, many pharmaceutical companies have reduced or abandoned antibiotic research and development (R&D) because of high development costs and limited financial returns, redirecting resources toward more profitable therapeutic areas (2). As a result, the decline in antibiotic R&D has narrowed the pipeline of new antimicrobial agents and limited our ability to treat resistant bacterial strains (11).

Consequently, researchers must explore alternative strategies to combat AMR. QQ offers one such approach by disrupting bacterial communication through mechanisms such as enzymatic degradation of signaling molecules, inhibition of signal synthesis, and receptor antagonism (4).

By targeting virulence and biofilm-associated behaviors rather than directly killing bacteria, QQ may help overcome resistant infections while reducing the selective pressure that drives resistance development.

Although QQ alone is unlikely to solve the global AMR crisis, it represents a promising adjunctive strategy that targets bacterial behavior rather than bacterial survival. By weakening biofilms and reducing virulence, QQ may extend the effectiveness of existing antibiotics and contribute to more sustainable antimicrobial therapies.

## Author Contributions

K.T.: Conceptualization, literature search, original draft, review & editing. R.K.: Supervision, review & editing
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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.

**Conflict of Interest** The authors declare no competing interests.

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