Revista Virtual de Química

Review

http://dx.doi.org/10.21577/1984-6835.20250051

Do Bacteria Gossip? *Quorum Sensing*: The Chemical 'Gossip' between Species

As Bactérias Fofocam? Quorum Sensing: A 'Fofoca' Química entre Espécies

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Submissão: 12 de Maio de 2025

Aceite: 15 de Setembro de 2025

Publicado online: 24 de Setembro de 2025

Bacterial communication through *Quorum sensing* coordinates group behaviours based on population density using chemical signals called autoinducers. This review explores the diversity of these signals, including acyl-homoserine lactones and autoinducing peptides specific to Gram-negative and Gram-positive bacteria, respectively, as well as inter-species signals like autoinducer-2, autoinducer-3, and indole. It details the molecular mechanisms underlying signal synthesis, detection (often via two-component systems or LuxR-type regulators), and downstream gene regulation controlling processes such as virulence, biofilm formation, bioluminescence, and competence. Furthermore, the manuscript discusses how environmental factors like nutrient availability, carbon sources, and stress conditions, integrated through mechanisms like the stringent response and catabolite repression, modulate *Quorum sensing* networks. The profound ecological significance of *Quorum sensing* is highlighted through examples of inter-species cross-talk, host-microbe interactions in pathogenesis and symbiosis, plant-microbe associations, and communication within complex marine ecosystems, demonstrating its crucial role in shaping microbial communities and their interactions with multicellular organisms.

Keywords: *Quorum sensing*; bacterial communication; autoinducers; inter-species signalling; host-microbe interactions; environmental factors.

1. Introduction

What would life be without communication? Without communication, life would be empty and profoundly anarchic. Communication between living organisms is a fundamental phenomenon, and it is not an exclusive privilege of humans or the most evolved animals, but permeates all living beings in all kingdoms of nature. Before continuing, it is necessary to elucidate the word communication, which has its origins in the Latin *communicare*, which means "to make common", "to share", "to associate". Thus, communication is not limited to the senses of hearing and vision; it is broader, more diverse, and extraordinary in living beings.

Communication between living organisms can occur in many ways, encompassing a variety of sensory modalities and communicative signals. The most common forms of communication include visual, chemical, auditory, tactile, and electrical signals. These signals can be emitted and received by different structures and specialised organs, allowing the exchange of information and the coordination of behaviours between organisms. Therefore, communication favours interaction and integration in a community, which is essential for survival, adaptation, reproduction, and social relations. It also plays a crucial role in the structuring and functioning of ecosystems.[1]

In the animal kingdom, communication plays a crucial role in several dimensions. Animals can communicate through visual signals, such as body displays and vibrant colours, which serve to attract mates, establish social hierarchies, and warn of dangers.[2] In addition, vocalisations and songs are widely used to communicate at a distance, mark territory, attract mates, and coordinate group behaviours.[3] There is also chemical communication, with the emission of pheromones playing important roles in reproduction, identification of conspecifics, and food search. Besides facilitating reproduction, communication in the animal kingdom is essential for survival, defence, and cooperation. It also allows for the efficient coordination of behaviours and adaptive strategies.[4]

In the plant kingdom, communication occurs subtly and silently, almost imperceptible to human ears. Plants communicate through chemical, electrical, and even auditory signals. They can emit chemical signals, such as the release of volatile organic compounds, in response to damage caused by herbivores, pathogens, or environmental changes.[5–7] In addition, root

communication allows the exchange of chemical and electrical signals, enabling communication between different parts of the plant or between neighbouring plants. This communication between plants is essential for coordinating defence responses, competition for resources, and establishing mutualistic interactions.[8]

Microorganisms also communicate with each other, and according to studies over the past decades, they "talk" a lot. Communication between microorganisms plays an essential role in their natural microbiomes. Despite their small size, microorganisms are masters at exchanging information and coordinating collective activities. Communication between them occurs through various mechanisms, including chemical signals, exchange of molecules, electrical communication, and even transfer of genetic material. These communication processes allow microorganisms to interact and coordinate their activities in response to environmental stimuli, such as changes in nutrient conditions, the presence of predators, pathogens, or any other environmental challenge.[2] One of the most extensively studied mechanisms of communication between microorganisms is *Quorum sensing*, which involves the production and detection of signalling molecules known as autoinducers. These chemical signals coordinate the behaviour of microorganisms, regulating gene expression and triggering collective behaviours, such as biofilm formation, enzyme production, expression of virulence factors, and even cooperation between different microbial species.[3]

In this review, we will address the role of *Quorum sensing* in bacterial communication and its role in marine ecosystems.

2. *Quorum Sensing*

Quorum sensing (QS) is defined as the process of cell-to-cell chemical communication that occurs most often between bacteria. It is based on the production, detection, and response to external signalling molecules. Bacterial units are capable of detecting the number of other cells present in the environment, allowing a group of bacteria to act synchronously, producing a coordinated behavioural response, which will depend on changes in population density and the species composition present in the community they inhabit.[9–11] Perception of the environment can direct cellular differentiation in bacteria, influencing the expression of genes related to diverse biological pathways.[12,13]

Communication between bacteria occurs through the production of molecules called autoinducers (AIs), which possess this name due to their capacity to be recognised by their receptors, leading to autoregulation. When the population density of bacterial cells is high, the high concentration of produced autoinducers leads to the initiation of a signalling cascade that will permit the activation of specific genes. These genes control the regulation of diverse processes that vary between species, such as bioluminescence, competence, swimming and swarming motility, symbiosis, antibiotic production, sporulation, biofilm formation, protein secretion, plasmid exchange, secondary metabolite production, and the secretion of virulence factors in bacteria. QS communication also occurs between kingdoms, mediated by certain types of autoinducers.[9,10,13–15]

For a molecule to be considered a *Quorum sensing* signal, it needs to meet several characteristics:[9]

I. Signal production occurs only during specific growth stages and depends on changes in physiological and environmental conditions.
II. The signal must diffuse into the extracellular space and be recognised by specific receptors.
III. The accumulation of a threshold concentration of the signal permits the initiation of a coordinated response.
IV. The cellular response must encompass more than the physiological changes required to metabolise or eliminate the signalling molecule.

The nature of the autoinducer varies between species and between systems within the same species, with each pathway responding to its own autoinducer signal. Likewise, the amount of signal required for this activation varies for each system.[9,12,16–18]

The term *Quorum sensing* was first introduced in 1994, decades after the initial studies describing this communication process. The first publications describing the communication capability among a group of bacteria were made in the late 1960s and early 1970s. In one of these studies, it was observed that a molecule produced by the species *Vibrio fischeri* was capable of inducing bioluminescence when the population density reached a certain concentration. Subsequently, this autoinducer molecule was identified as N-(3-oxohexanoyl)-L-homoserine lactone (**2**, Figure 1).[9,19]

N-(3-oxohexanoyl)-L-homoserine lactone is part of a group of molecules called AI-1, belonging to the acyl-homoserine lactone (AHL) family (**1-8**, Figure 1). Besides the AHL system, other *Quorum sensing* signalling molecules can be found in different bacterial species, such as Autoinducer-2 (AI-2) (**9**), Autoinducer-3 (AI-3) (**10**), diffusible signal factor (DSF) (**11**), the *Pseudomonas* quinolone signal (PQS) (**12**), cholera autoinducer-1 (CAI-1) (**13**), palmitic acid methyl ester (PAME) (**14**), indole (**15**), and autoinducing peptides (AIPs) (**16**).[11,19,20]

For true communication to occur between bacteria, two requirements must be met: first, one or more individuals must produce a signal to be perceived by other individuals present; second, the recipients of this signal must alter their behaviour in response to the signalling. The demonstration of one bacterium responding to a signal produced by another does not necessarily mean that communication has occurred between them, as true communication requires that both individuals receive benefits and co-evolve from this

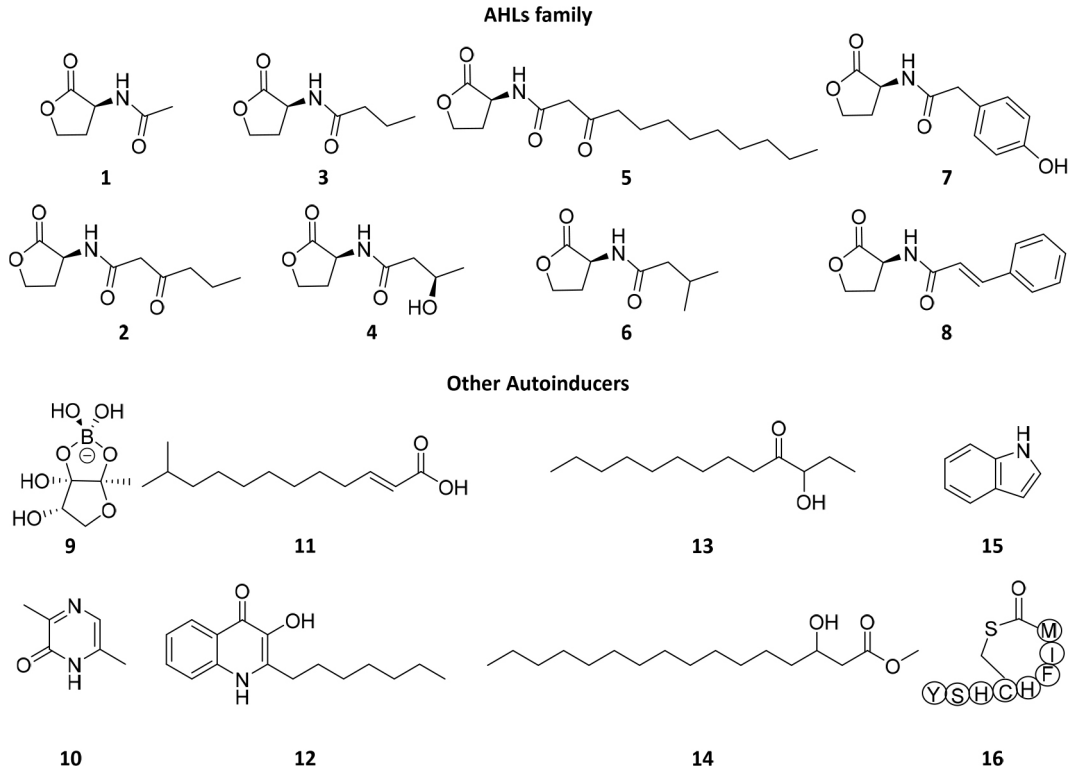


Figure 1. Structures of autoinducers molecules produced by bacteria

communication, allowing it to persist in the evolutionary lineage of the species.[16,19,21,22]

Quorum sensing is important for the survival of bacteria in nature and influences the actions of bacterial groups.[23] Depending on the bacterial strain, various bacterial processes can be under the control of *Quorum sensing* regulation, especially the systems involved in the production of secondary metabolites, virulence, and symbiosis.[24]

3. An Evolutionary Perspective on *Quorum Sensing*

The widespread existence of *Quorum sensing* (QS) presents an intriguing evolutionary puzzle. From an evolutionary standpoint, explaining the stability of both cooperation and communication is a significant challenge. Communication is susceptible to cheaters who may not signal honestly, and cooperation is vulnerable to individuals who benefit without paying the cost; it is not always a win-win game. Quorum sensing appears to combine both of these challenges, making its evolution and maintenance a subject of considerable scientific interest. To understand how QS systems may have emerged and been naturally selected, it is crucial to distinguish between different types of chemical interactions and to consider the selective pressures that favor collective action.

It is hypothesized that QS evolved from metabolic cues that were co-opted for communication— a process known as evolutionary exaptation. Many QS signals are, in fact, derivatives or byproducts of fundamental cellular processes (primary metabolism). Molecules like AHLs are synthesized from two fundamental metabolic building blocks: SAM and acyl-carrier proteins (acyl-ACPs), which are essential for fatty acid synthesis. AI-2 is a direct byproduct of the activated methionine cycle, a process fundamental to the recycling of S-adenosyl-L-methionine (SAM) in many bacteria. Its origin as a metabolic by product leads many to consider it primarily a metabolic cue, rather than a universally evolved communication signal. The indole nucleus derives directly from the degradation of the amino acid tryptophan. All these QS are deeply tied to primary metabolism. In an ancestral state, the passive leakage of these molecules could have served as an inadvertent but reliable cue for metabolic activity and cell density. Natural selection would then favor the evolution of receptors capable of detecting these cues, allowing cells to gain information about their environment.[22,25,26]

The critical question is what selective advantage drove the transition from simple cue detection to sophisticated, regulated signalling. Two major, non-exclusive hypotheses provide an answer:

I. Coordinating the Production of Public Goods: The classic explanation is that QS evolved to coordinate cooperative behaviors that are only effective when undertaken by a large group. Many QS-regulated traits involve the secretion of "public goods" - metabolically costly molecules like exoenzymes or siderophores that benefit the entire local community. Producing

these goods at low cell density would be wasteful and inefficient. Kin selection theory provides a robust framework for this cooperation, as limited dispersal in microbes often means that interacting individuals are close relatives (high genetic relatedness), which makes altruistic cooperation evolutionarily stable.[25,26]

II. Collective Sensing and the "Wisdom of the Crowds": A more recent and complementary hypothesis proposes that QS evolved as a mechanism for collective environmental sensing. Individual bacteria may have noisy or imperfect estimates of environmental conditions (e.g., nutrient availability, stress levels, pH). By producing autoinducers at a rate that reflects their individual perception of the environment and then sensing the pooled concentration, cells can average out the noise and arrive at a more accurate collective assessment. This "wisdom of the crowds" functionality explains why QS is beneficial even for regulating "private goods" like competence or sporulation, which are not shared but still depend on making the right decision at the right time. This model elegantly reconciles observations that autoinducer levels depend not only on cell density but also on a host of environmental factors. For example, the production of *p*-coumaroyl-HSL by bacteria depends on the precursor *p*-coumaric acid, which is released by senescing algae, directly linking the QS signal to an environmental cue from a host. This ability to integrate multiple environmental and social inputs is fundamental to the function of quorum sensing.[25,26]

4. Autoinducers and Mechanisms

There are many specific autoinducer compounds; however, their behaviour can vary according to the species. Autoinducers can be classified into two major categories: those produced by Gram-negative bacteria and those produced by Gram-positive bacteria. In Gram-negative bacteria, the autoinducer is a molecule produced within the cell and exported across the cell membrane. The primary and most common intraspecies autoinducer in Gram-negative bacteria is acyl-homoserine lactone (AHL) molecules. Some bacteria are also capable of producing other autoinducer molecules different from AHLs, such as PQS, PAME, DSF, and CAI-1. In Gram-positive bacteria, autoinducers are typically peptides, often referred to as pheromones. These peptides, which exhibit a wide structural variety, are produced through the proteolysis of precursor peptides and undergo post-translational processes, such as cyclization and the addition of groups like geranyl, isoprenyl, or methyl to the peptide structure, resulting in changes in the physicochemical characteristics of the peptide relevant to its required activity. However, in addition to these two categories, some molecules serve as inter-species signals, which can be produced and detected by both Gram-negative and Gram-positive bacteria. Among these molecules are AI-2, AI-3, and indole.[14,27–31]

4.1. *Quorum sensing* in Gram-Negative bacteria

4.1.1. AHL Quorum sensing *system*

The AHL *Quorum sensing* system was first described between the 1960s and 1970s in the marine bacterium *V. fischeri* through the identification of "autoinduction" activity in high-density cultures. This activity is responsible for controlling bioluminescence as part of the symbiotic association that occurs between the bacterium and the species *Monocentris japonica* (fish) and *Euprymna scolopes* (squid).[32]

The system is based on the N-acyl-homoserine lactone (AHL) molecule, which requires an AHL synthase protein, and a transcription factor called an "R protein", such as LuxR (*V. fischeri*) and LasR (*Pseudomonas aeruginosa*), whose activity varies according to the concentration of AHL present. The basic chemical structure of AHL in these systems is similar, consisting of a homoserine lactone (HSL) ring and an acyl chain. This acyl chain can vary both in length and in the number of saturations present; it can also have variations in the substituent groups attached to the carbon at position 3 of the chain, such as hydrogen atoms, carbonyl, and hydroxyl groups. These variations help bacteria to distinguish their own AHL molecules from the products of other species, allowing for the specification of different systems (Table 1).[14,28,33–35]

There are three known families of AHL synthetases: the LuxI-type synthase family, described in a large number of Gram-negative bacteria, which utilize S-adenosylmethionine (SAM) and an acyl-acyl carrier protein (acyl-ACP) as substrates to produce AHL molecules (Figure 2).[35] The LuxM-type family, primarily identified in species of the *Vibrio* genus, includes proteins such as LuxM (*Vibrio harveyi*), AinS (*V. fischeri*), and VanM (*Vibrio anguillarum*). This family can utilise SAM and either acyl-ACP or acyl-CoA as substrates; however, it exhibits different structures compared to those found in the first family. The lysophosphatidic acid acyltransferase protein family, such as HdtS (*Pseudomonas fluorescens*) and Act (*Acidithiobacillus ferrooxidans*), which are responsible for acylating lysophosphatidic acid (LPA) (Figure 3) and synthesising AHL. The synthesis mechanism of this last family has not yet been fully elucidated.[35,63,64]

AHL molecules are synthesised and interact specifically with "R protein" transcription factors when the bacterial population density is high. This interaction causes conformational changes in the "R protein", activating it and leading to the binding of this protein to a specific DNA sequence within the target promoter region (Figure 4). This process often induces the transcription of the cognate AHL synthase gene, resulting in a positive feedback loop that further increases the AHL concentration.[14,28,65]

Table 1. AHL signalling molecules and related processes

Species	Regulatory Proteins	Molecule Signaling	Processes	References
Vibrio fischeri	LuxI/LuxR	3-oxo-C6-HSL	Luminescence	36
Vibrio fluvialis	VfqI/VfqR	3-oxo-C10-HSL	Virulence	37
Vibrio harveyi	LuxM/LuxN	3-hydroxy-C4-HSL	Luminescence	38–42
Vibrio anguillarum	VanI/VanR	3-oxo-C10-HSL	Virulence	43,44
Pseudomonas aeruginosa	LasI/LasR	3-oxo-C12-HSL	Secondary metabolites, enzymes, RhlR- biofilm, virulence	38,45–49
	RhlI/RhlR	C4-HSL	Exoproducts, rhamnolipid biosurfactant, secondary metabolites, RpoS, maturation, biofilm	
Pseudomonas aureofaciens	PhzI/PhzR	3-oxo-C6-HSL	Phenazine production	50–52
Pseudomonas fluorescens	PhzI/PhzR	3-oxo-C6-HSL	Phenazine production	53,54
Burkholderia cepacia	CepI/CepR	3-oxo-C8-HSL	Proteases regulation, siderophore expression	55
Erwinia carotovora	ExpI/ExpR	3-oxo-C6-HSL	Exoenzymes	46,56
	CarI/CarR		Antibiotics production	
Erwinia chrysanthemi	ExpI/ExpR	3-oxo-C6-HSL 3-oxo-C8-HSL 3-oxo-C10-HSL	Virulence, secondary metabolites, exoenzymes, carbapenem antibiotic production	57,58
Pantoea stewartii	EsaI/EsaR	3-oxo-C6-HSL	Capsule and cell envelope biosynthesis, surface motility and adhesion, stress response	59,60
Serratia liquefaciens	SwrI/SwrR	3-oxo-C4-HSL 3-oxo-C6-HSL	Virulence, swarming	46,61
Ralstonia solanacearum	RasI/RasR	3-OH-C12-HSL	Cellulase production, motility, biofilm, oxidative response, virulence	62

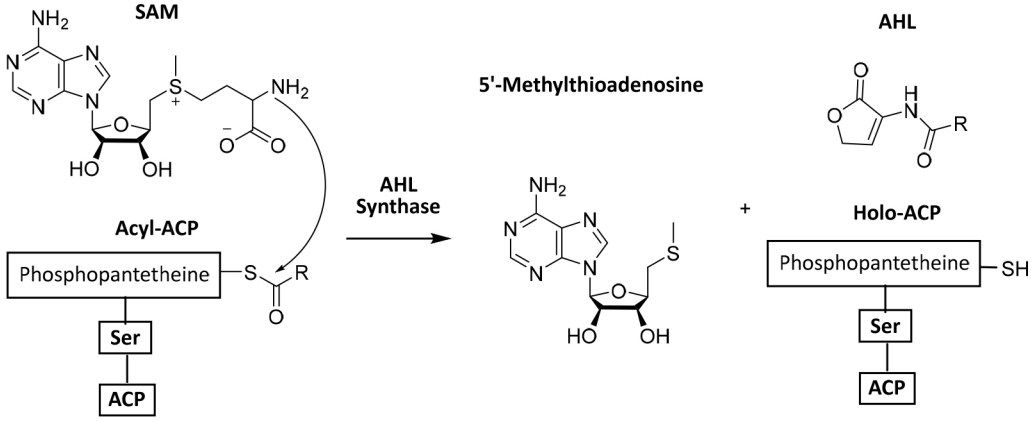


Figure 2. AHL synthesis via S-adenosylmethionine (SAM) and acyl-ACP

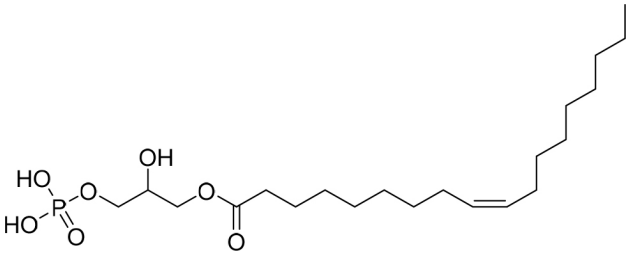


Figure 3. Chemical structure of lysophosphatidic acid (LPA)

4.1.2. Other signalling molecules in Gram-Negative bacteria

Gram-negative bacteria can produce and perceive other signalling molecules that regulate the expression of QS genes. One example is 2-heptyl-3-hydroxy-4-quinolone (**12,** Figure 1), produced by *Pseudomonas aeruginosa*. It belongs to the alkylquinolone family, members of which are collectively known as PQS (*Pseudomonas* quinolone signal). PQS binds to the regulator PqsR, which is

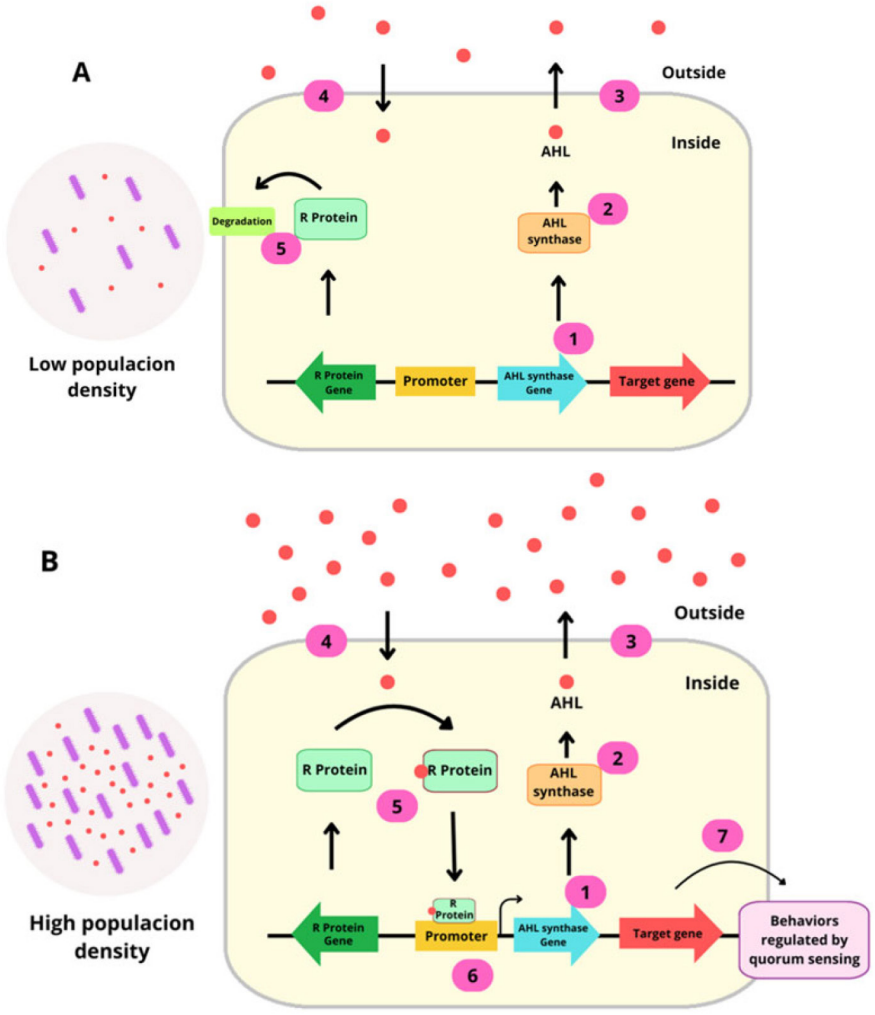


Figure 4. AHL *Quorum sensing* system at low population density (**A**) and at high population density (**B**)

responsible for activating the expression of various virulence factors and components associated with biofilm formation. Its production is positively controlled by the LasI/LasR system, which regulates the 3-oxo-C12-HSL molecule. PQS also acts in the activation of rhlI gene expression, which is responsible for C4-HSL synthesis. AHLs and PQS play an important role in coordinating virulence, antibiotic resistance, biofilm formation, and fitness in *P. aeruginosa*. Furthermore, PQS possesses iron-chelating properties, contributing to the regulation of genes involved in siderophore biosynthesis.[63,66–73]

The molecule 3-hydroxypalmitic acid methyl ester (3-OH-PAME) (**14**), produced by the phytopathogenic bacterium *Ralstonia solanacearum*, is synthesised by the PhcB protein during bacterial growth. At high cell density, 3-OH-PAME is detected by the kinase sensor PhcS, which phosphorylates the response regulator PhcRQ. This relays the information to the transcriptional regulator PhcA, which is responsible for expressing virulence factors and secondary metabolites necessary for the late-stage plant infection process and biofilm formation (Figure 5).[65,74–77]

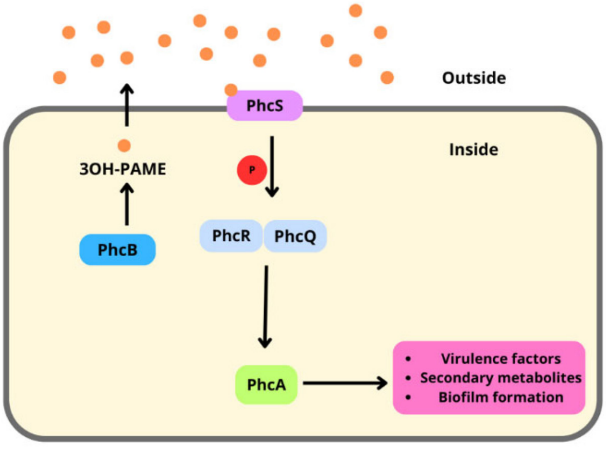


Figure 5. Regulation of QS by 3OH-PAME

Other produced autoinducers belong to the diffusible signal factor family, which are cis-2-unsaturated fatty acids. The plant pathogen *Xanthomonas campestris* produces cis-11-methyl-2-dodecenoic acid (**11**), which is known by the acronym DSF and gives its name to this autoinducer

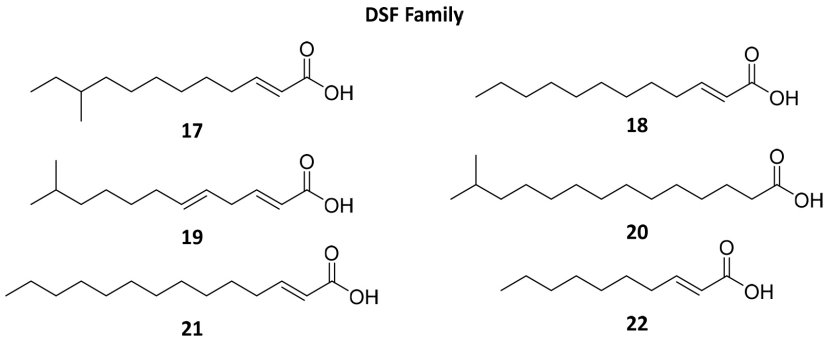


Figure 6. Chemical structures of the Diffusible Signal Factor (DSF) signalling molecules. (Z)-10-methyldodec-2-enoic acid (**17**), (Z)-dodec-2-enoic acid (**18**), (2-Z)-11-methyldodeca-2,5-dienoic acid (**19**), 13-methyltetradecanoic acid (**20**), (Z)-tetradec-2-enoic acid (**21**), (E)-dec-2-enoic acid (**22**)

family (**17-22**, Figure 6). DSF is produced by RpfF enzyme and detected by the two-component sensor RpfC, which transmits the signal to the RpfG protein. RpfG regulates the secondary messenger cyclic di-guanosine monophosphate (c-di-GMP), which functions as an inhibitory ligand for the global transcription factor Clp. Clp is responsible for the expression of hundreds of genes, including those encoding virulence factors (Figure 7). The DSF system can also be found in other species such as *Burkholderia cepacia*, *Stenotrophomonas maltophilia*, and *Xanthomonas campestris*.[78–82]

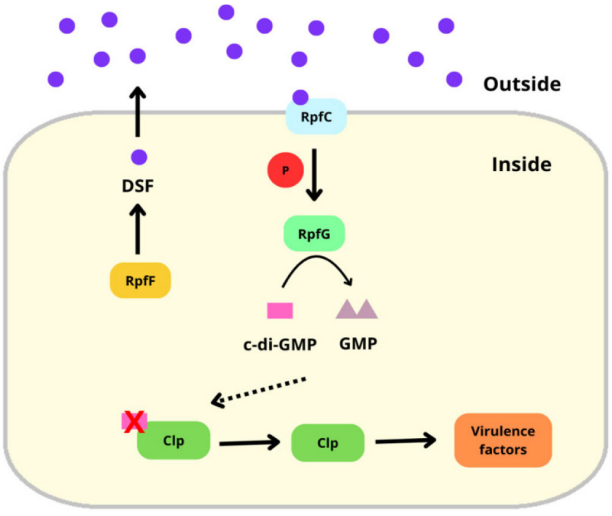


Figure 7. Regulation of QS by DSF

The species *Vibrio cholerae* produces an α-hydroxyketone known as CAI-1 (**13**) (cholera autoinducer 1). This molecule is synthesised by the acyl-CoA transferase CqsA and recognised by the transmembrane kinase sensor CqsS. At high concentrations, CAI-1 binds to CqsS, leading to the dephosphorylation and inactivation of the response regulator LuxO. LuxO is responsible for repressing the master QS transcription factor HapR. The activation of HapR represses virulence and biofilm genes, whilst also activating the expression of proteases that allow *V. cholerae* to escape from the host back into the environment (Figure 8). CqsA enzymes can be found in all species of the *Vibrio* genus (Figure 9), and they can produce different CAI-1 molecules, which possess varying acyl chain lengths and modifications. *Vibrio* species can respond to CAI-1 molecules produced by other species with differing affinities, suggesting that CAI-1 is used for intra-*Vibrio* communication.[63,65,68,83,84]

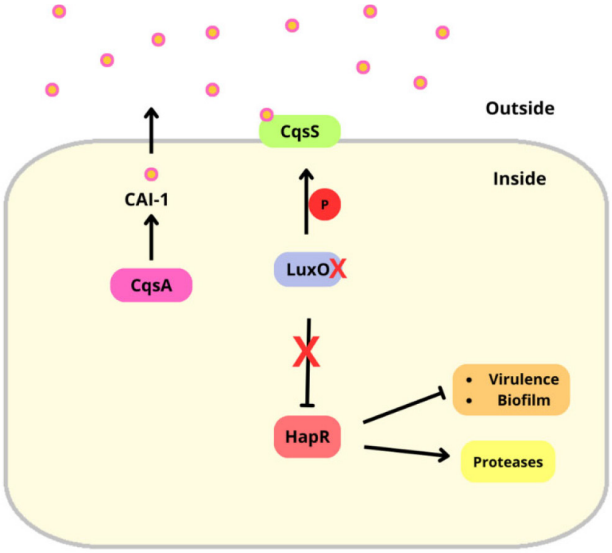


Figure 8. CAI-1-mediated *Quorum sensing* in *Vibrio cholerae*

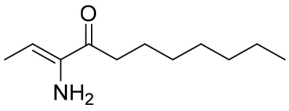


Figure 9. CAI-1 molecule produced by the species *Vibrio harveyi*

4.2. *Quorum sensing* in Gram-Positive bacteria

The regulation of gene expression by peptides in Gram-positive bacteria shares many similarities with *Quorum sensing* expression in Gram-negative bacteria that utilise AHL molecules. The difference is that the response depends on an autoinducing peptide (AIP) (**16**) via a two-component signal transduction system. An unmodified or post-translationally modified peptide is secreted via ATP-binding

cassette (ABC) transporters and functions as the signalling molecule. Sensor kinases within the two-component system detect the secreted peptide molecules, which leads to a series of events culminating in the phosphorylation of the response regulator protein. These regulator proteins become active upon phosphorylation and alter transcription rates by binding to promoter sites, thereby activating the expression of *Quorum sensing*-related genes (Figure 10).[14,28,85,86]

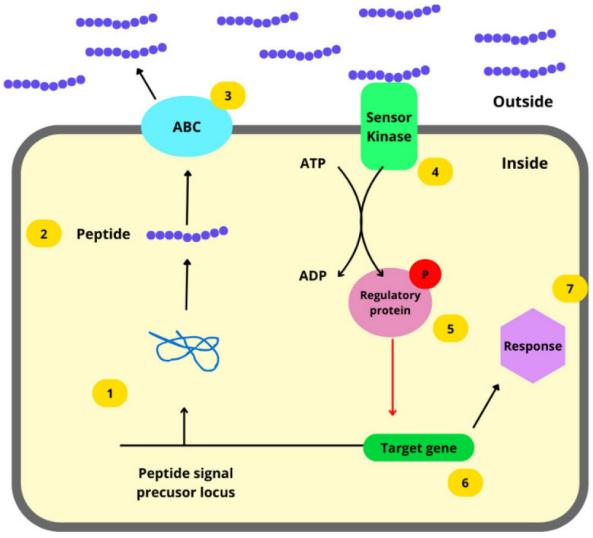


Figure 10. Mechanism of peptide-mediated *Quorum sensing* in Gram-positive bacteria

One example is the process utilised by the species *Streptococcus pneumoniae*: three separate operons are required for the process to be initiated: comCDE, comAB, and comX. The comC gene encodes the precursor peptide signal (ComC), which is cleaved and exported by the products of the comAB genes, becoming the competence-stimulating peptide (CSP) (Figure 11). CSP is detected by and binds to the ComDE two-component system. Binding leads to the autophosphorylation of the membrane-bound sensor kinase ComD. In response, ComD phosphorylates the response regulator ComE. This leads to the binding of ComE to a regulatory sequence upstream of the gene encoding the competence-specific sigma factor (comX), activating the expression of various competence genes. The competence state contributes to virulence and biofilm formation and allows the cell to transform its own genome through the lysis of neighbouring cells, leading to the incorporation of exogenous DNA. Identical regulatory sequences for ComE binding are also found upstream of the comCDE and comAB operons, resulting in a positive feedback loop similar to that observed in the Lux system.[14,28,86–89]

4.2.1. Interspecies signalling

4.2.1.1. AI-2 Quorum sensing *system*

The furanosyl borate diester (**9**), also known as autoinducer-2 (AI-2), represents a group of signalling molecules found in both Gram-positive and Gram-negative

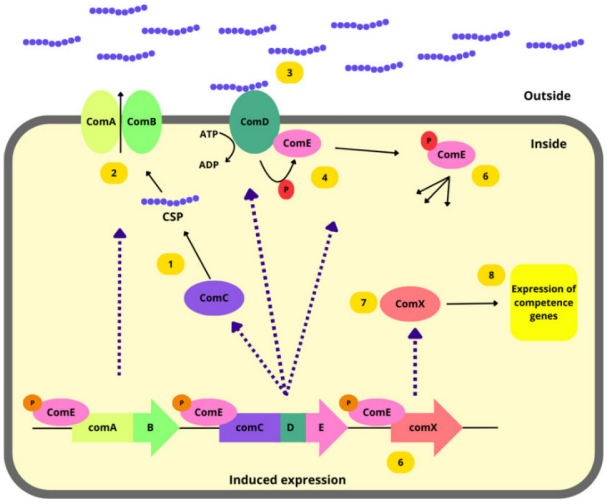


Figure 11. *Quorum sensing* regulation of competence development in *Streptococcus pneumoniae*

bacteria, functioning as an inter-species signal. AI-2 is synthesised by diverse bacteria and permits communication both between individuals of the same species and between different species, leading to its recognition as a universal *Quorum sensing* signal. AI-2 is a byproduct of the conversion of S-ribosyl-homocysteine to homocysteine, a reaction catalysed by LuxS within the activated methyl cycle. The LuxS enzyme, or its homologues, produces the AI-2 precursor molecule, 4,5-dihydroxy-2,3-pentanedione (DPD) (**24**), as it catalyses the cleavage of the thioester bond in S-ribosyl-homocysteine. DPD spontaneously cyclises in aqueous solution, resulting in a mixture of interconverting isoforms collectively known as AI-2 (Figure 12).[15,90–93]

AI-2 molecules exist in two identified forms: S-THMF-borate (**9**), which is recognised by LuxP and found only in *Vibrio* species, and R-THMF (**25**), which is recognised by LsrB and found in enteric bacteria and some members of other families. Bacteria can chemically recognise distinct AI-2 molecules and generate specific responses. The LuxS/AI-2 *Quorum sensing* system modulates various cellular processes, involved primarily in the regulation of virulence factors, bacterial luminescence, sporulation, motility, toxin production, biofilm formation, and drug resistance.[15,90–94]

In Gram-negative bacteria, two AI-2 pathways have been completely elucidated. The first pathway, found in the species *V. harveyi*, detects extracellular AI-2 via the membrane sensor LuxPQ, leading to the dephosphorylation of the cascade proteins LuxU and LuxO. LuxO regulates the expression of small RNAs that bind to the mRNA of the regulator LuxR, thereby repressing its translation. The subsequent production of LuxR inhibits the expression of virulence and biofilm genes whilst activating the expression of genes related to bioluminescence (Figure 13A). In the second pathway, found in *Escherichia coli* and *Salmonella enterica serovar Typhimurium*, extracellular AI-2 is recognised and internalised by the ABC-type transporter LsrACDB. Inside the cell, the AI-2 molecule is phosphorylated by the kinase protein LsrK, thereby

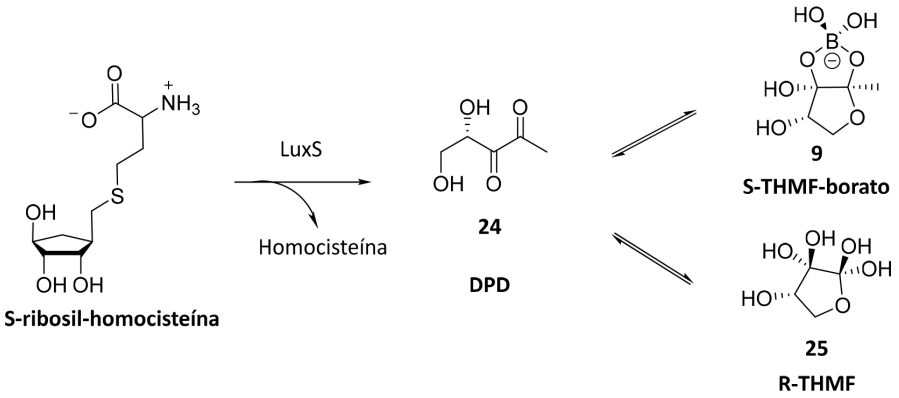


Figure 12. Synthesis of AI-2

activating it. Activated AI-2 (phospho-AI-2) binds to the repressor LsrR, inactivating it and permitting gene transcription (Figure 13B). Phosphorylated AI-2 activates the expression of various genes, including those that increase AI-2 uptake via the expression of the lsr genes. Furthermore, this pathway increases antibiotic resistance, promotes surface colonisation, and enhances adhesion, aggregation, and biofilm formation.[68,93,95–97]

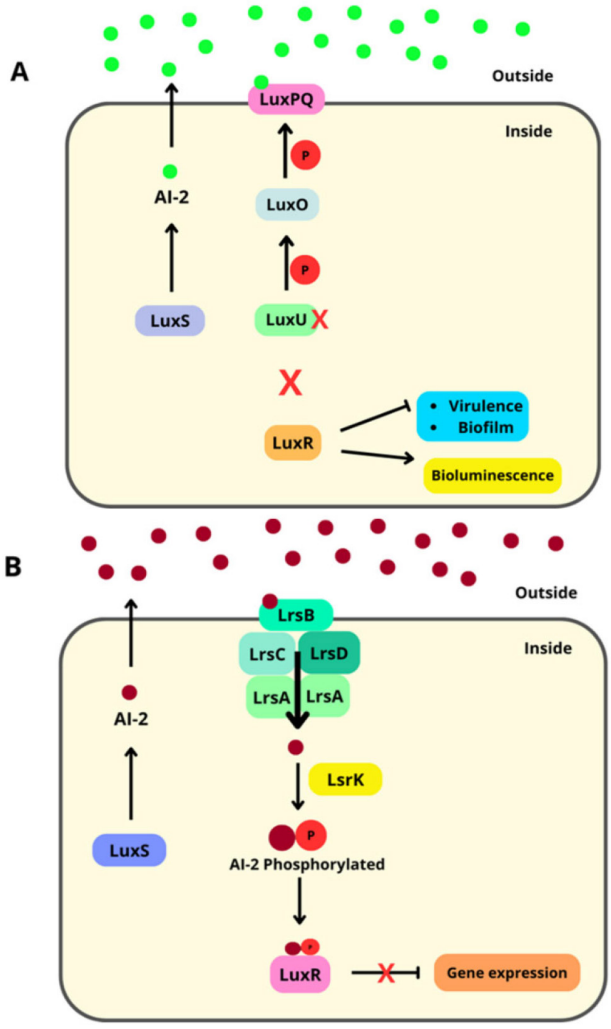


Figure 13. Schematics of AI-2 pathways

In Gram-positive bacteria, homologues of the systems present in Gram-negative bacteria are absent. However, the RbsB protein (ribose-binding protein) may regulate AI-2 uptake due to the likely structural similarity between AI-2 and the ribose molecule, although the complete mechanism has not yet been elucidated. A fructose-specific phosphoenolpyruvate: sugar phosphotransferase system (PTS), termed FruA, is highly conserved in Gram-positive bacteria. AI-2 signalling via FruA stimulates the Leloir pathway, leading to an increase in capsular polysaccharide production and virulence.[92,94]

4.2.1.2. AI-3 signaller

Another group of molecules responsible for inter-species communication is the family known as autoinducer-3 (AI-3) (**10**). It is related to communication between prokaryotic and eukaryotic cells. The products of this group belong to the pyrazinone family (**26**, Figure 14). Among the reactions responsible for product formation are threonine dehydrogenase, mediating AI-3 production, and aminoacyl-tRNA synthetases, potentially related to spontaneous cyclisation. AI-3 analogues (**27-30**, Figure 14) can be found in both Gram-positive and Gram-negative bacteria, and the sensor that recognises these molecules and regulates gene expression is the histidine kinase sensor QseC, found in species such as enterohemorrhagic *E. coli* (EHEC) and *V. cholerae*. QseC is also capable of recognising the host hormones epinephrine and norepinephrine; however, AI-3 and its analogues do not affect adrenergic receptor signalling. [15,96,98] Upon activation, QseC promotes its autophosphorylation and the phosphorylation of QseB, initiating a phosphorylation cascade that activates the expression of virulence genes. This includes the activation of the QseEF two-component system, further increasing the expression of virulence genes (Figure 15).[99–101]

Indole (**15**, Figure 1) is another molecule utilised in intercellular, inter-species, and inter-kingdom signalling, produced by various bacteria from the degradation of tryptophan by tryptophanase (TnaA). A wide variety of species can produce indole, and consequently, it is widespread in the natural environment, possessing an important role in bacterial pathogenesis, even in species

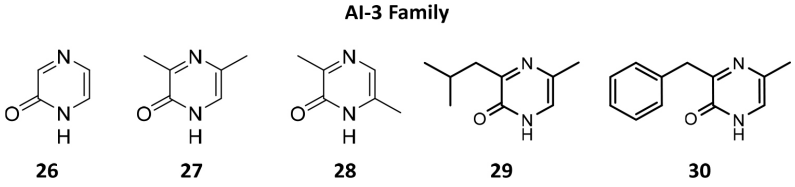


Figure 14. Pyrazinone molecule and AI-3 analogues. 3,5-dimethyl-1H-pyrazin-2-one (**27**), 3,6-dimethyl-1H-pyrazin-2-one (**28**), 5-methyl-3-(2-methylpropyl)-1H-pyrazin-2-one (**29**), 3-benzyl-5-methyl-1H-pyrazin-2-one (**30**)

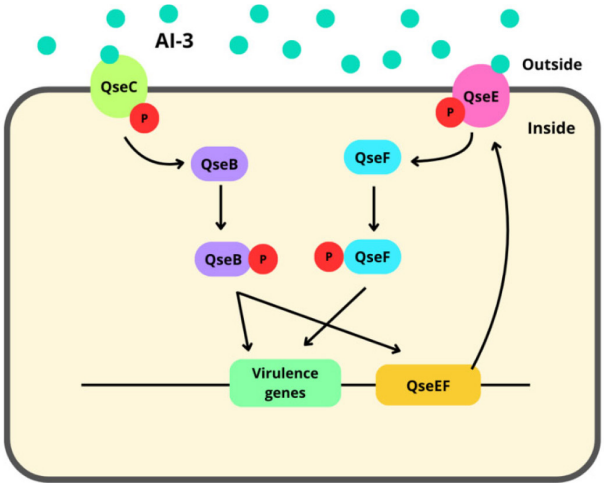


Figure 15. Quorum sensing mediated by AI-3

incapable of producing it. Indole and its analogues (**31-34**, Figure 16) are related to diverse biological functions such as spore formation, plasmid stability, antibiotic resistance, acid resistance, biofilm formation, and virulence. Indole can act to either increase or decrease biological functions, depending on the species, also affecting the production of other *Quorum sensing* signals in bacteria, such as AHLs. Indole acts on different receptors present in various bacterial species, such as the CpxAR two-component system found in EHEC, which is responsible for antimicrobial peptide resistance. The receptor IsrR is specific for the indole molecule and is responsible for decreasing the expression of virulence genes. The gene encoding this receptor can be found in EHEC, *Klebsiella*, and *Shigella*.[15,96,102–105]

5. Factors Influencing *Quorum Sensing* (QS)

Various factors can influence the production or inhibition of *Quorum sensing*, including environmental factors and other organisms present in the environment. Environmental factors such as the deprivation of various nutrients (e.g., nitrogen, magnesium, and phosphorus) and oxidative stress can influence the detection and expression of both regulatory and QS-regulated genes. Low nutrient availability in the medium leads to the activation of a stress survival strategy known as the stringent response. This stringent response is mediated by guanosine tetraphosphate and guanosine pentaphosphate [(p)ppGpp]. At high concentrations, (p)ppGpp binds to RNA polymerase, altering its selectivity and inhibiting various cellular processes, including the expression of QS-related genes.[63,106–109]

The carbon sources available to bacteria also represent another factor influencing *Quorum sensing*. Known as Carbon Catabolite Repression (CCR), this process involves the regulation of genes encoding catabolic enzymes and carbohydrate transport systems, depending on the type of sugar present. It allows bacteria to adapt to the presence of multiple carbon sources, as they can selectively utilise the preferred source whilst inhibiting the expression of enzymes that catabolise non-preferred carbon sources. In the presence of less preferred substrates, an increased level of cyclic AMP (cAMP) induces higher production of AHL and AI-2 in various bacteria.[109,110]

In *P. aeruginosa*, the alteration of different factors leads to a variety of responses related to *Quorum sensing*. Low phosphate concentration leads to increased expression of proteins from the RhlR and PQS systems, thereby increasing the production of C4-HSL and PQS molecules. This occurs independently of the Las system, which is the principal *Quorum sensing* response regulator in this species.[111–113] Low iron concentrations increase the expression of the lasR and pqsR genes, which can induce the expression of many iron-responsive genes. Furthermore, the Fur protein (ferric uptake regulator) increases the expression of two small regulatory RNAs (sRNAs), PrrF1 and PrrF2. These sRNAs are responsible for inhibiting the expression of a gene whose product degrades the PQS precursor substance. [63,114–117]

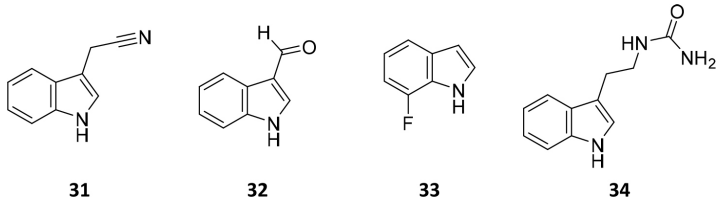


Figure 16. Indole analogues. 2-(1H-indol-3-yl)acetonitrile (**31**), 1H-indole-3-carbaldehyde (**32**), 7-fluoro-1H-indole (**33**), 2-(1H-indol-3-yl)ethylurea (**34**)

Alterations in magnesium availability also lead to changes in *Quorum sensing* systems. At low magnesium levels, there is an increase in the induction of the LasI protein and proteins of the Pqs system, leading to increased levels of 3-oxo-C12-HSL and PQS. [118–121] Oxygen limitation leads to a reduction in PQS production, given that the PqsH protein, responsible for PQS synthesis, requires oxygen to function.[122]

5.1. Interactions between other microorganisms and hosts

Quorum sensing holds great biological importance for bacteria, as it often represents a competitive advantage over other organisms in the ecosystem, whether these are terrestrial or marine organisms, including humans. In nature, bacteria normally coexist with other species, leading to the development of communication methods between them; that is, the *Quorum sensing* of one species can influence, and be influenced by, the *Quorum sensing* or other activities carried out by neighbouring species. One example is the production of LuxR-type transcriptional regulators in AHL non-producing species, allowing them to "eavesdrop" on the conversation of other bacteria. The species *E. coli* and *Salmonella enterica serovar Typhimurium* possess the LuxR-type regulator SdiA, which can be activated by AHLs produced by other species. This may serve as an indication that the current environment is suitable for inducing their virulence genes, or for producing autoinducer-degrading enzymes.[63,123]

In *Chromobacterium violaceum*, the presence of C6-HSL or other short-chain AHLs (C4-C8) leads to the induction of violacein pigment, exoprotease, and chitinase production. However, the *Quorum sensing* signal from *Pseudomonas aeruginosa* (3-oxo-C12-HSL) and other long-chain AHLs (C10-C14) are capable of inhibiting the production of these structures.[124,125] *Bacillus subtilis* produces lipopeptides known as fengycins, which function as antagonists of the AgrC *Quorum sensing* receptor present in *S. aureus*. This leads to the repression of Agr-controlled virulence factor production, thereby suppressing the ability of *S. aureus* to colonise rats.[10,126]

The regulation of *Quorum sensing* genes influences the interaction of bacteria with their hosts, varying according to the species and organisms involved. Both symbiotic and pathogenic microorganisms utilise *Quorum sensing* signals for colonisation and infection, whilst eukaryotic hosts simultaneously develop strategies to detect, respond to, and even manipulate these signals. The green alga *Chlamydomonas reinhardtii* also produces molecules capable of mimicking AHL molecules, leading to the stimulation of LasR or CepR. This can have stimulatory, inhibitory, or additive effects on the *Quorum sensing* of the wild-type bacterium *Sinorhizobium meliloti*.[10,63,127–130]

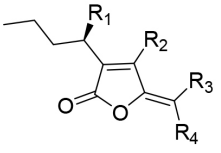
Pantoea agglomerans can induce virulence genes via the QS genes pagI and pagR. Following tumour formation in the plant, the bacterium produces the hormone indoleacetic acid (IAA), as well as cytokines that modulate the expression of pagI and pagR.[131,132]

Quorum sensing influences interactions with plants, leading to the activation of hundreds of bacterial genes that play an important role in plant-microorganism interactions. These include genes responsible for biofilm formation, nitrogen fixation, hydrolytic enzyme synthesis, motility, among others. Examples include interactions of plants with their nitrogen-fixing rhizobial symbionts and pathogens such as *Ralstonia solanacearum*, *Erwinia carotovora*, *Agrobacterium tumefaciens*, *Xanthomonas campestris*, and *Serratia marcescens*.[63,127,133,134]

The plant *Medicago truncatula* produces substances capable of modulating AHL molecule synthesis, either stimulating or inhibiting bacterial signalling. Simultaneously, AHLs stimulate the expression of plant defence and stress management genes, phytohormone production, and metabolic regulation.[134–136] Extract from the plant *Mangifera indica L.* was shown to inhibit violacein production in *Chromobacterium violaceum*, as well as decreasing the production of proteases, pyocyanin, chitinases, exopolysaccharides, and swarming motility in *Pseudomonas aeruginosa* PAO1.[137]

In the marine environment, the bioluminescence produced by *V. fischeri* and *V. harveyi* in the light organ of squid is important for the host animal's competitiveness. Another interaction occurs between the species *V. anguillarum* and the green alga Ulva. The AHL signal produced by the microorganism can attract the macroalga's zoospores, as well as releasing morphogenetic factors that stimulate the macroalga's cell division. Simultaneously, the alga provides substrates for the formation and establishment of the *V. anguillarum* biofilm.[63,127,138]

The red marine alga *Delisea pulchra* produces halogenated furanones (**35-39**, Figure 17) capable of interacting with bacterial AHL receptors, regulating the expression of *Quorum sensing* genes, potentially through receptor degradation.[127,139–141]



	R_1	R_2	R_3	R_4
35	H	Br	Br	Br
36	H	Br	H	Br
37	OAc	Br	H	Br
38	OH	Br	H	Br
39	OAc	Br	H	I

Figure 17. Furanones produced by marine microalga *Delisea pulchra*

6. Applications for *Quorum Sensing*

Understanding the function of quorum sensing in activating various behavioral processes in bacteria, such as virulence and biofilm formation, has enabled research into the possible applications of this communication system. The relationship between quorum sensing and the

control of bacterial populations has gained prominence for its potential in developing clinical therapies for treating diseases and combating antibiotic resistance. Research focused on finding and synthesizing novel molecules that can be used as quorum sensing inhibitors has been explored for the potential development of new antibacterial drugs. By binding to QS receptors, these molecules would weaken bacteria by reducing the effective concentration of autoinducers and preventing QS signaling. Therefore, enhances the sensitivity of these bacteria to antibacterial treatments.[142–145]

Other studies involve combining autoinducers, particularly from the AHL system, with the engineering of genetic circuits such as genetic oscillators, toggle switches, and logic gates for use in therapeutic applications. In Gram-negative bacteria, the development of a genetic circuit with the luxI gene promoter regulating the expression of a specific lysis gene allows the bacterium also to produce a cytotoxic agent continuously once the AHL molecule reaches a threshold concentration and activates gene expression. Consequently, there is a decrease in the number of bacteria due to the expression of the lysis gene. The surviving bacteria restart the cycle once they again produce AHL molecules, thereby enabling population control. In another engineered system, the luxI and aiiA genes are regulated by the luxI promoter; when activated by an increase in AHL, this leads to the production of homoserine lactonase (AiiA), which is responsible for degrading AHL molecules.[142,146–148]

In Gram-positive bacteria, the existence of QS-related mechanisms that are not yet fully elucidated complicates research on the application of quorum sensing using genetic circuits. Another factor is the difference in the signaling molecules used by Gram-negative versus Gram-positive bacteria. AHL molecules are small and diffuse easily across membranes, whereas AIPs exhibit low diffusion, especially in solid media, and require a transporter for passage out of the cell, which has delayed studies in Gram-positive bacteria.[142,149]

7. Conclusion

Quorum sensing represents a sophisticated and fundamental communication system enabling bacteria to coordinate complex behaviours and adapt to diverse environments. This review has highlighted the remarkable chemical diversity of autoinducer signals (e.g., acyl-homoserine lactones, autoinducing peptides, autoinducer-2, autoinducer-3, indole) and the intricate regulatory networks governing their production and perception across Gram-negative and Gram-positive bacteria, including widely used signals facilitating inter-species and inter-kingdom communication. The integration of *Quorum sensing* pathways with cellular metabolic status and environmental cues, such as nutrient availability and stress responses, underscores its role as a key adaptive strategy. Furthermore, the critical importance of *Quorum sensing* extends beyond intra-species coordination, profoundly influencing microbial community structure, host-microbe interactions (both pathogenic and symbiotic), plant health, and the functioning of complex ecosystems like the marine environment. Understanding this chemical 'gossip' not only reveals fundamental principles of microbial life but also offers potential avenues for controlling bacterial behaviour in medical, agricultural, and environmental contexts.

Despite notable advances, a central limitation of current Quorum Sensing (QS) studies is that much of our fundamental knowledge has been generated under simplified laboratory conditions, such as pure cultures. This model often fails to replicate the complexity of natural habitats, where factors like fluid flow, surface topography, and coexistence in dynamic polymicrobial communities exert profound influences. Recent studies reveal that, instead of synchronous activation, the QS response can be heterogeneous within the same population, and communication between spatially distinct bacterial aggregates, the so-called "calling distance," appears to be limited to very short distances, suggesting that many interactions may be primarily local rather than population-wide. Additionally, translating QS knowledge into real-world applications, such as in industrial formulations or anti-QS therapies, still faces the challenge of the stability and longevity of active molecules, like quorum-quenching enzymes, in complex matrices. Therefore, overcoming these limitations requires the integration of principles from ecology, engineering, sociobiology, and chemistry to understand how bacterial communication actually operates and evolves in its native ecosystems. Future research should continue to unravel the complexity of these signalling networks, particularly in polymicrobial communities and their natural habitats.

Acknowledgements

The authors would like to thank the scholarships granted by the Rio de Janeiro State Research Support Foundation (FAPERJ) and the Coordination for the Improvement of Higher Education Personnel (CAPES, Scholarship Financing Code 001).

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