International Journal of Research Studies on Environment, Earth, and Allied Sciences (IJRSEAS)
| *Volume-3 Issue-2* | *April-2026* | *ISSN: 3066-0637* | *Website:* https://eesrd.us

Manuscript ID:
IJRSEAS-2026-030258

OPEN ACCESS

Quick Response Code:

Website: https://eesrd.us

DOI: 10.5281/zenodo.21472753

DOI Link: https://doi.org/10.5281/zenodo.21472753

Volume: 3

Issue: 2

Pp. 335-340

Month: April

Year: 2026

E-ISSN: 3066-0637

Submitted: 16 Mar. 2026

Revised: 27 Mar. 2026

Accepted: 17 Apr. 2026

Published: 30 Apr. 2026

Address for correspondence:
Dr. K. Vidya Prabhakar
Assistant Professor, Dept. of Biotechnology, Vikrama Simhapuri University Nellore, AP, India
Email: prathimagolla@gmail.com

How to cite this article:
G. Prathima & K. Vidya Prabhakar. (2026). Assistant Professor, Dept. of Biotechnology, Vikrama Simhapuri University Nellore, AP, India. International Journal of Research Studies on Environment, Earth, and Allied Sciences, 3(2), 335–340. https://doi.org/10.5281/zenodo.21472753

The Quorum-Sensing System and Its Signalling Role in Bacteria

Dr. G. Prathima[1], Dr. K. Vidya Prabhakar[2]
[1]Department of Biotechnology, Vikrama Simhapuri University, Nellore, AP, India
[2]Assistant Professor, Dept. of Biotechnology, Vikrama Simhapuri University Nellore, AP, India

Abstract

Robert Hooke and Anton van Leeuwenhoek discovered microscopic organisms between 1665 and 1683. It took more than 150 years after this discovery to develop microscopy, which became the foundation for understanding microbes. During the period 1950-2000, extraordinary changes in microbiology occurred, including the discovery of the DNA structure by Watson and Crick in 1953. Thus, with advances in microscopy and related fields such as microbial metabolism, scientists were able to study microorganisms and apply them to industrial purposes. The importance of microbes in human life is vast. They can be friends and foes at times. Many microbial products contribute to human nutrition and to some diseases. Others help improve the standard of living. As a result, microbes and microbiology play an important role in advancing human health and welfare. Among them, bacteria are more closely related to human life than the broader microbial community. We are supported by natural flora from birth, and it grows and helps build immunity during growth. Similarly, many bacteria are beneficial to humans' daily lives by serving as probiotics and assisting in the fermentation of many food products.

Keywords: *Quorum sensing; Bacterial communication; Autoinducers; Biofilm formation; Gram-positive bacteria; Gram-negative bacteria; LuxI/LuxR system; Cell-density signalling; Bacterial virulence; Gene regulation.*

Introduction

Bacteria were once regarded as solitary microorganisms that responded independently to environmental stimuli. However, advances in microbiology and molecular biology have demonstrated that bacterial cells possess sophisticated communication systems that enable coordinated group behaviour. This phenomenon, known as **quorum sensing (QS)**, allows bacteria to detect their population density through the production, release, and detection of small signalling molecules called **autoinducers**. When these signalling molecules accumulate beyond a threshold concentration, they activate specific regulatory pathways that alter gene expression and synchronize the behaviour of the bacterial community.

Quorum sensing plays a pivotal role in regulating numerous physiological processes, including biofilm formation, bioluminescence, virulence factor production, sporulation, antibiotic production, motility, competence, and symbiotic interactions. By coordinating these activities, bacterial populations can adapt efficiently to changing environmental conditions, enhance survival, and optimize resource utilization. The discovery of quorum sensing in *Vibrio fischeri* provided the first evidence that bacteria communicate through chemical signals to regulate collective behaviour, fundamentally changing our understanding of microbial biology.

Different bacterial groups employ distinct quorum-sensing mechanisms. Gram-negative bacteria primarily utilize **N-acyl homoserine lactones (AHLs)** mediated by the LuxI/LuxR regulatory system, whereas Gram-positive bacteria predominantly rely on secreted oligopeptides and two-component signal transduction systems. In addition, universal signalling molecules such as autoinducer-2 (AI-2) facilitate interspecies communication, highlighting the complexity of microbial interactions within diverse communities.

The growing understanding of quorum sensing has significant implications in medicine, agriculture, biotechnology, and environmental science. Since QS regulates pathogenicity and biofilm formation in many clinically important bacteria, disrupting these signalling pathways—known as quorum quenching—has emerged as a promising strategy to combat antimicrobial resistance without exerting the selective pressure associated with conventional antibiotics.

This review summarizes the evolution, molecular mechanisms, signalling pathways, and biological significance of quorum sensing in Gram-positive and Gram-negative bacteria. It also highlights the role of quorum sensing in bacterial physiology, host-pathogen interactions, and its potential applications in developing innovative therapeutic and biotechnological approaches.

Quorum Sensing

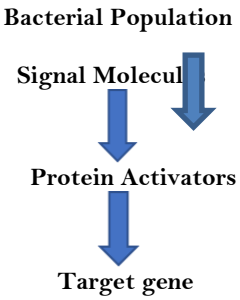
This technique allows bacteria to coordinate their actions and communicate with each other. These processes usually entail the control of particular genes in response to population density. Certain signal molecules are produced and released in order to coordinate gene expression. Signal molecules known as autoinducers are emitted and picked up by particular receptors. The quantity of autoinducers generated in the cell diffuses into the surrounding environment when population density is low. The production of autoinducers increases with the size of the bacterial population. The protein that functions as a transcriptional regulator is greatly activated by the generated autoinducers. The transcription of QS-regulated genes is either increased or decreased when the regulators bind to the target DNA sequence altering the transcription of genes that are controlled by QS. altering the transcription of genes that are controlled by QS. The bacterial population consequently displays phenotypic expression. (Podbielski et al., 2004; Henke et al., 2004; Gonzalez et al., 2006).

Quorum Sensing Evolution

It was originally believed that bacteria were simple, unicellular organisms whose only function was to proliferate. Most people believed that they solely reacted to outside stimuli. This idea was altered by a quick study in later years, which showed that these organisms may behave like multicellular organisms (Witzany et al., 2008). In the 1960s and 1970s, several biochemical and genetic investigations revealed indications of "organized social behaviour" in bacteria. In order to coordinate particular bacterial behaviours within the population, this kind of organized social behaviour makes use of complex communication systems. At first, it was believed to be an uncommon occurrence. Numerous bacteria can sense and react to light, according to recent studies. The phenomenon of bacterial individuality depends on cell density. Quorum sensing is the term for the phenomenon, which suggests that. The term "quorum sensing" suggests that it will sense. Quorum sensing in the bright marine bacterium *Vibrio fischeri* was initially reported by Nelson and Hastings in 1970.

They found that until these bacteria attain a high population density, they are unable to glow. This finding led to the theory that *Vibrio fischeri's* bioluminescence was regulated by cell-to-cell communications known as autoinducers (Fuqua et al., 2001; Losick et al., 1997; Nealson et al., 1970).

Quorum-Sensing Pathway



More signal molecules are released into the environment as the number of bacteria increases. As the number of bacteria increases, so does the amount of signalling molecules. They are recognized by the chemicals released by protein activators. Consequently, the target genes become active, controlling several bacterial behaviours, including motility, pathogenicity, biofilm formation, and more (Moghaddam et al., 2014).

Quorum Sensing - The Architectural System

As previously mentioned, quorum-sensing systems employ and release autoinducers, which are tiny signalling molecules. AHLs (Gram-negative bacteria), peptides (Gram-positive bacteria), and AI-2 (Gram-negative bacteria) are the three groups of quorum-sensing signal molecules that have been investigated the most. Other quorum-sensing systems include autoinducer-3 (AI-3), the diffusible signal factor (DSF), and Pseudomonas quinolone signal (PQS). The most prevalent signal molecules in Gram-negative bacteria are called AHLs, and they are made up of a variable acyl chain that can have four to eight carbon atoms connected to an invariant homoserine lactone ring (HSL).

Autoinducers

There are two primary parts to the bacterial quorum-sensing mechanism.

1. Receptor proteins (LuxI/LuxR)
2. Autoinducers

Small compounds known as autoinducers have the ability to pass through cell membranes and into the surrounding environment (Aendekerk et al., 2002; Kaplan, 1985; Pearson et al., 2000). These autoinducers (AI) are categorized into two types: AI Type I and AI Type II, which are species-specific and nonspecific, respectively A wide range of chemically distinct substances is recognised as signalling molecules generated in response to environmental changes and controlling a wide range of genes. Acyl homoserine lactones are naturally occurring signal molecules used by most Gram-negative bacteria. Furthermore, the structures of the 2-alkyl-4-quinolones, -butyrolactones, furanone, long-chain fatty acid derivatives, fatty acid methyl esters, peptides, and 4,5-dihydroxy2,3-petadione (DPD) derivatives produced by Gram-negative bacteria are unknown (Winzer et al., 2002; Winzer et al., 2003; Sperandio et al., 2003; Vendeville et al., 2005; Williams, 2007). Vibrio Sp. is a well-known example of bioluminescence based on quorum sensing. The pathogenicity and other characteristics of many other Gram-

negative bacteria are similarly controlled by quorum sensing (Charu Gera et al., 2006). Oligopeptide autoinducers (AIP) are used by gram-positive bacteria.

Quorum Sensing in Gram-negative bacteria

Research indicates that LuxI and LuxR homologs create autoinducer homoserine lactones that are utilized by Gram-negative bacterial systems. A transcriptional regulator protein that identifies the cognate HSL and initiates the proper phenotype is encoded by the LuxR homolog. Such cell-to-cell activity has been seen in more than thirty Gram-negative bacterial species. There is a layer of intricacy in LuxI/LuxR systems, despite the fact that many bacteria use them for quorum sensing. Homoserine lactone (HSL) rings with acyl chains varying in length from C4 to C18 make up AHLs (Geisinger et al., 2009). These side chains occasionally exhibit unsaturated double bond modifications, particularly at the C3 position.

Vibrio fischeri, a bioluminescent marine bacterium, was the first to discover the AHL-cognate regulatory circuit and its autoinducer. Euprymna scolopes is a bacterium that inhabits the light organ of the Hawaiian Bobtail Squid. The bacteria and the squid grow in tandem within the squid's light organ's nourishing interior, allowing the bacteria to reach high cell density and, via quorum sensing, activate the luciferase-encoding operon. To avoid predation, the squid host uses bacterial-induced light to illuminate itself (Ruby et al., 1996). In order to control bioluminescence by quorum sensing, LuxI and LuxR are required. N-3-(oxohexanoyl)-homoserine lactone synthase is autoinduced by LuxI, a quorum-sensing enzyme (Engebrecht et al., 1984; Schaefer et al., 1996). Quorum-sensing regulation of bioluminescence in *V. fischeri* requires LuxI and LuxR. N-3-(oxohexanoyl)-homoserine lactone synthase is the quorum-sensing autoinducer LuxI (Engebrecht et al., 1984; Schaefer et al., 1996). The acylation and lactonization processes between the S-adenosyl methionine (SAM) substrates and hexanoyl-ACP are catalysed by LuxI synthase (More et al., 1996). 3-oxoC6HSL readily enters and exits the cell after synthesis, and as cell population density rises, so does its concentration (Kaplan et al.,1985). LuxR is a transcriptional activator of the luciferase luxICDABE operon and a cytoplasmic receptor for 3-oxoC6HSL (Engebrecht et al., 1984).

To control bioluminescence by quorum sensing, LuxI and LuxR are required. LuxI is an autoinducer of N-3-(oxohexanoyl)-homoserine lactone synthase that senses quorum (Engebrecht et al., 1984; Schaefer et al., 1996). The quorum-sensing regulation of bioluminescence in *V. fischeri* requires LuxI and LuxR.

The quorum-sensing autoinducer N-3-(oxohexanoyl)-homoserine lactone synthase enzyme is known as LuxI (Engebrecht et al.,1984; Schaefer et al.,1996). The acylation and lactonization processes between hexanoyl-ACP and S-adenosyl methionine (SAM) substrates are catalysed by LuxI synthase (More et al., 1996). After synthesis, 3-oxoC6HSL readily enters and exits cells, and as cell population density rises, so does its concentration (Kaplan et al., 1985).

LuxR is a transcriptional activator of the luciferase luxICDABE operon and a cytoplasmic receptor for 3-oxoC6HSL (Engebrecht et al., 1984). Only when the 3-oxoC6HSL ligand is present can the LuxR protein remain stable; otherwise, it degrades quickly. LuxR binds the accumulated 3-oxoC6HSL, and the LuxR-AHL complex stimulates the production of the luxICDABE operon by identifying a concordant binding region upstream of it (Stevens et al., 1994). The fatty acid reductase complex, which starts and retrieves the luciferase aldehyde substrate, is encoded by the luxCDE genes, whereas the luciferase subunits are encoded by the luxAB genes.

Positive feedback from autoinduction occurs when the quorum-sensing circuit activates, inducing the creation of the autoinducer and flooding the surrounding regions with the signal molecule. As the cell population transitions from low to high cell-density quorum-sensing mode, this "autoinduction" positive feedback loop is meant to sustain synchrony. For quorum-sensing-controlled gene expression in Gram-negative bacteria, the *V. fischeri* LuxI/LuxR regulatory system is considered the model. Numerous bacterial genomes have been found to have homologs of luxI and luxR. These additional LuxIR-type quorum-sensing systems regulate global, cell-density-dependent gene expression (Case et al., 2008). These AHL quorum-sensing systems are frequently linked to positive feedback loops involving LuxR-type proteins that trigger luxI-type gene expression (Fuqua et al., 1994). Homologs of luxI and luxR have been found in many bacterial genomes, and other LuxIR-type quorum-sensing systems regulate global, cell-density-dependent gene expression (Case et al., 2008).

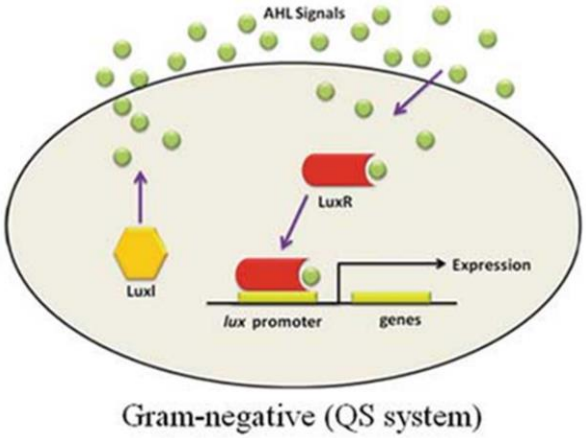


Fig. 1. Quorum-sensing circuit in Gram-negative bacteria

According to biochemical analyses of LuxR-type proteins, there are three types of these receptors. Class 1 receptors (LasR) are beautifully constructed and require AHL for folding. They have a strong bond with their ligands. AHLs are likewise necessary for the folding of SSClass 2 receptors (such as *V. fischeri* LuxR), but the mature proteins attach to their ligands reversibly. Class 3 receptors, like EsaR, do not need AHLs to fold and have reversible ligand binding. Since protein translation is a somewhat lengthy process, the necessity for AHLs for protein folding in Class 1 and 2 LuxR-type receptors implies that these receptors are resistant to sudden changes in the ambient concentration of AHLs. Additionally, it is hypothesized that the primary factor influencing these receptors' resilience to disruption is the variation in ligand binding affinity (Schuster et al., 2006).

Quorum Sensing in Gram-positive bacteria

Secreted peptides are used as quorum-sensing autoinducers by gram-positive bacteria. Oligopeptide exporters mediate the release of peptide signals because they do not diffuse over the membrane. In contrast, "two-component"-type membrane-bound sensor histidine kinases are used as receptors by Gram-positive bacteria, while LuxR-type proteins are frequently used by Gram-negative bacteria as autoinducer sensors. The activity of a response regulator, a regulatory protein that modulates signalling, is influenced by a phosphorylation cascade. For instance, octapeptides and a "two-component" AgrC/AgrA system are used by the invasive pathogen Staphylococcus aureus to control its virulence system. Quorum sensing is used by different bacterial species to control different phenotypes. Bacterial competence is developed by *Bacillus subtilis* and *Streptococcus pneumoniae* through quorum sensing.

Enterococcus faecalis controls conjugation through quorum sensing, while Staphylococcus aureus controls pathogenicity. The ATP-binding cassette (ABC) transporter in Gram-positive bacteria uses two adaptor response proteins to produce the peptide autoinducer that serves as a quorum-sensing signal. Peptide autoinducers are recognized by gram-positive bacteria, and this two-component signalling is synchronized by the phosphorylation/dephosphorylation cascade. When cell density rises, peptide autoinducers build up. The genetic circuit's two-component sensor kinases pick up the secreted peptide signal. Consequently, a cognate response regulator protein is phosphorylated after the peptide ligand complex starts a phosphorylation cascade. The activation of the response regulator facilitates DNA binding, which leads to the transcription of target genes that sense quorum.

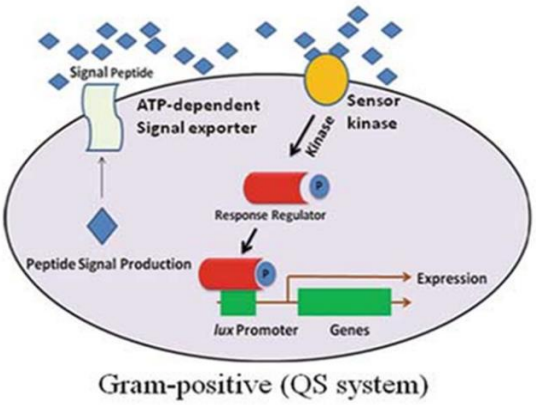


Fig. 2. Quorum-sensing circuit in Gram-positive bacteria

Conclusions

QS is a crucial regulatory mechanism used by many bacteria to control collective characteristics that allow them to take advantage of particular conditions. For example, QS grants symbionts access to nutrient-rich environments in host bacterial communities. These communities use QS to control the formation of biofilms, which improves population members' access to resources and enables them to outcompete biofilm-producing neighbours. Three themes emerge when considering the cases described above. To develop QS gene regulation, regulatory networks have first been added to the basic autoinducer-generating and detection apparatuses. Given the worldwide genomic programs controlled by QS, these extra regulatory systems probably guarantee that changed gene expression occurs only under extremely precise circumstances.

Alternatively, other regulatory systems may have appropriated QS to control additional products in response to certain environmental situations because QS regulates global gene expression patterns. Second, QS is associated with the formation of biofilms in these and other situations. QS has an impact on when this process takes place in each of these species, despite the fact that the mechanism and timing of production vary. Communication is clearly beneficial to members of biofilm communities, and bacterial communication is probably stronger when cells are in the close relationships that biofilms provide. Third, it seems that sRNA-mediated regulation is significant.

Acknowledgments

The authors are grateful to the Head, Department of Biotechnology, Vikrama Simhapuri University, Nellore, AP, India, for providing laboratory space and instruments.

Financial support and sponsorship
Nil.

Conflicts of interest
The authors declare that there are no conflicts of interest regarding the publication of this paper.

References
1. Abee T, Kovács ÁT, Kuipers OP et al., 2011, Van Der Veen S. Biofilm formation and dispersal in Gram-positive bacteria. Curr. Opin. Biotechnol, 22:172–179.
2. Aendekerk, Séverine, Bart Ghysels, Pierre Cornelis, Christine Baysse et al., 2002, Characterization of a New Efflux Pump, MexGHI-OpmD, from *Pseudomonas aeruginosa* That Confers Resistance to Vanadium. Microbiology, 148, 2371–81.
3. Ansaldi M, Dubnau, et al., 2004, DJ, Bacteriol. 186, 15-21.
4. Bacon, Schneidee K, Palmer TM, Grossman, 2001 A.D.J. Bacteriol.184, 410-419.
5. Barroso KCM, Previato-Mello M, Batista BB, Batista JH, da Silva Neto JF et al.,2018, EmrR-dependent upregulation of the efflux pump EmrCAB contributes to antibiotic resistance in Chromobacterium violaceum. Front. Microbiol, 9, 2756,
6. Batista JH, da Silva Neto JF et al.,2017, Chromobacterium violaceum pathogenicity: updates and insights from genome sequencing of novel *Chromobacterium* Species. Front. Microbiol, 8, 2213,
7. Bloom-Ackermann Z, Ganin H, Kolodkin-Gal I et al., 2015, Quorum-sensing Cascades Governing Bacterial Multicellular Communities. Isr. J. Chem, 56:302–309.
8. Brumbach KC, Eason BD, Anderson LK et al., 2007, The Serratia -type hemolysin of *Chromobacterium violaceum*. FEMS Microbiol. Lett, 267, 243– 250,
9. Cairns L, Hobley L, Stanley-Wall NR et al., 2014, Biofilm formation by *Bacillus subtilis*: new insights into regulatory strategies and assembly mechanisms. Mol. Microbiol,93:587–598.
10. Casalta JP, Peloux Y, Raoult D, Brunet P, Gallais H. 1989, Pneumonia and meningitis caused by a new non fermentative unknown gram-negative bacterium. J. Clin. Microbiol 27, 1446–1448.
11. Case, Rebecca J, Maurizio Labbate, Staffan Kjelleberg et al., 2008, AHLDriven Quorum-Sensing Circuits: Their Frequency and Function among the Proteobacteria. ISME Journal,345–49.
12. Ciprandi A, da Silva WM, Santos AV, de Castro Pimenta, AM, Carepo MSP, Schneider, MPC, AzevedoV, Silva A et al., 2013, *Chromobacterium violaceum*: Important insights for virulence and biotechnological potential by exoproteomics studies. Curr. Microbiol, 67, 100– 106.
13. de Brito CFA, Carvalho CMB, Santos FR, Gazzinelli RT, Oliveira SC, Azevedo V, Teixeira SMR et al., 2004, *Chromobacterium violaceum* genome: molecular mechanisms associated with pathogenicity. Genet. Mol. Res, 3, 148– 152.
14. Dragos A., Kiesewalter H, Martin M, Hsu CY, Hartmann R, Wechsler T, Eriksen C, Brix S, Drescher K, Stanley-Wall N, et al., 2018, Division of Labor during Biofilm Matrix Production. Curr. Boil, 28:1903–1913.
15. Engebrecht, J, Silverman M et al.,1984, Identification of Genes and Gene Products Necessary for Bacterial Bioluminescence," Proceedings of the National Academy of Sciences of the United States of America, 81, 4154–58.
16. Flemming HC, Wingender J et al., 2010, The biofilm matrix. Nat. Rev. Genet, 8:623–633.
17. Fuqua C, MR Parsek, EP Greenberg, 2001, Regulation of Gene Expression by Cell-to-Cell Communication: Acyl-Homoserine Lactone Quorum Sensing. Annual Review of Genetics, 35, 48-58.
18. Fuqua WC, Winans SC et al., 1994, A LuxR-LuxI Type Regulatory System Activates Agrobacterium Ti Plasmid Conjugal Transfer in the Presence of a Plant Tumor Metabolite. Journal of Bacteriology, 176, 2796–2806.
19. Geisinger ET, Muir W, Novick RP et al.,2009, Agr Receptor Mutants Reveal Distinct Modes of Inhibition by Staphylococcal Autoinducing Peptides," Proceedings of the National Academy of Sciences, 106.
20. Gera, Charu, Srivastava S et al., 2006, Quorum-Sensing: The Phenomenon of Microbial Communication," Current Science, 666–76.
21. Gest H, 2004, The Discovery of Microorganisms by Robert Hooke and Antoni van Leeuwenhoek, Fellows of The Royal Society. Notes and Records of the Royal Society, 58, 187–201.
22. González, Juan E, Neela D, Keshavan, et al., 2006, Messing with Bacterial Quorum Sensing. Microbiology and Molecular Biology Reviews, MMBR, 70, 859–75.
23. Greenberg EP et al., 1997, Quorum sensing in gram negative bacteria. ASM News, 63, 371– 377.
24. Hagiya H, Murase T, Suzuki M, Shibayama K, Kokumai Y, Watanabe N, Maki M, Otsuka F. 2014, *Chromobacterium violaceum* nosocomial pneumonia in two Japanese patients at an intensive care unit. J. Infect. Chemother.
25. Hall CW, Mah TF et al.,2017, Molecular mechanisms of biofilm-based antibiotic resistance and tolerance in pathogenic bacteria. FEMS Microbiol. Rev. 41, 276– 301,
26. Hall-Stoodley L, Costerton JW, Stoodley P et al., 2004, Bacterial biofilms: from the Natural environment to infectious diseases. Nat. Rev. Genet. 2:95–108.
27. Henke, Jennifer M, Bonnie L. Bassler, 2004, Three Parallel Quorum Sensing Systems Regulate Gene Expression in Vibrio harveyi. Journal of Reference 139 Bacteriology, 186, 6902–14.
28. Howard AJ, Ison CA et al., 1996, *Haemophilus, Gardnerella and other bacilli*; Collee J G, Fraser AG, Marmion BP, Simmons A. Eds.14th ed, Mackie and McCartney Practical Medical Microbiology, pp. 329–41.

29. Jefferson KK et al., 2004, What drives bacteria to produce a biofilm? FEMS Microbiol. Lett,236:163–173.
30. Kalamara M, Spacapan M, Mandic-Mulec I, Stanley-Wall NR et al., 2018, Social behaviours by *Bacillus subtilis*: quorum sensing, kin discrimination and beyond. Mol. Microbiol, 110:863–872.
31. Kaplan HB, EP, Greenberg, 1985, Diffusion of Autoinducer Is Involved in Regulation of the *Vibrio fischeri* Luminescence System. Journal of Bacteriology, 163, 1210–14.
32. Kaplan, HB, Greenberg EP et al., 1985, "Diffusion of Autoinducer Is Involved in Regulation of the Vibrio fischeri Luminescence System," Journal of Bacteriology, 163, 1210–14.
33. Kobayashi K et al., 2007, Gradual activation of the response regulator DegU controls serial expression of genes for flagellum formation and biofilm formation in *Bacillus subtilis*. Mol. Microbiol.
34. Kumar MR. 2012, *Chromobacterium violaceum:* A rare bacterium isolated from a wound over the scalp. Int. J. Appl. Basic Med. Res. 2, 70–72.
35. López D, Kolter R et al.,2010, Extracellular signals that define distinct and coexisting cell fates in *Bacillus subtilis*. FEMS Microbiol Rev,34:134–149.
36. Lopez D, Vlamakis H, Kolter R et al., 2010, Biofilms. Cold Spring Harb. Perspect. Boil.
37. Losick R, Kaiser D, 1997, Why and How Bacteria Communicate. Scientific American, 276, 68–73.
38. Magnuson R, Solomon J, Grossman, 1994, A.D. Cell 77, 207-216.
39. Mandic-Mulec I, Stefanic P, Van Elsas JD. et al., 2015, Ecology of *Bacillaceae*. Microbiol. Spectr, 3:59–85.
40. McClean KH, Winson MK, Fish L, Taylor A, Chhabra, SR, Camara M, Daykin M, Lamb, JH, Swift S, Bycroft BW, Stewart GS, Williams P et al., 1997, Quorum sensing and *Chromobacterium violaceum*: expression of violacein production and inhibition for the detection of N-acyl homoserine lactones. Microbiology, 143, 3703–3711,