OPEN ACCESS Freely available online

ISSN: 2168-9431
Single Cell Biology

Short Communication

Signaling for Survival: Genetic Regulation Mechanisms in *Salmonella* and *E. coli*

Marcello Falasca*

Department of Molecular Biology, University of Piemonte Orientale, Novara, Italy

DESCRIPTION

Salmonella and *Escherichia coli* (*E. coli*) are two well-known bacterial pathogens that employ cell-to-cell signaling processes to regulate genes potential for their survival, virulence, and adaptation to varying environments.

Quorum sensing in *Salmonella* and *E. coli*

Quorum Sensing (QS) is a mechanism used by bacteria to monitor population density through the production, secretion, and detection of signaling molecules called autoinducers. As the bacterial population grows, the concentration of autoinducers increases, triggering changes in gene expression once a threshold concentration is reached. Both *Salmonella* and *E. coli* utilize QS regulate various physiological processes and virulence factors [1].

Autoinducer molecules

Salmonella and *E. coli* produce different types of autoinducer molecules depending on the species and strain. For instance, *E. coli* commonly uses Acyl-Homoserine Lactones (AHLs) as autoinducers, while *Salmonella* may use AI-2 (autoinducer-2), which is synthesized *via* the enzyme LuxS. These molecules diffuse freely across bacterial cell membranes and accumulate extracellularly as bacterial populations increase [2].

Regulatory network

In *Salmonella*, QS influences the expression of genes involved in biofilm formation, motility, and the secretion of virulence factors such as Salmonella Pathogenicity Islands (SPIs). *SPI-1* and *SPI-2* are critical for invasion of host cells and survival within macrophages, respectively, and their expression is tightly controlled by QS mechanisms [3].

Similarly, *E. coli* employs QS to regulate genes associated with biofilm formation, motility, and toxin production. For example, Enterohemorrhagic *E. coli* (EHEC) strains use QS to coordinate the expression of shiga toxins, which are potential virulence factors in causing severe foodborne illnesses [4].

Mechanisms of gene regulation

Once the autoinducer reaches a critical threshold concentration, it binds to specific transcriptional regulators known as LuxR-type proteins or other regulatory proteins, initiating changes in gene expression. These proteins typically act as transcription factors that either activate or repress target genes involved in QS-regulated pathways [5].

Feedback loops within QS systems allow bacteria to fine-tune gene expression in response to environmental cues and fluctuations in population density. Negative feedback mechanisms may involve the downregulation of autoinducer synthesis or degradation enzymes once the target gene products reach sufficient levels.

Salmonella and *E. coli* have evolved sophisticated QS systems that enable them to adapt to diverse host environments, evade immune responses, and establish infections. QS-regulated genes contribute to bacterial colonization of host tissues, evasion of host immune defenses, and modulation of inflammatory responses, thereby enhancing pathogenicity [6].

Host-pathogen interactions

In *Salmonella*, QS-mediated regulation of *SPI* genes facilitates the secretion of effector proteins that manipulate host cell signaling pathways, facilitating invasion and intracellular survival. QS also coordinates the expression of factors involved in resistance to host antimicrobial peptides and oxidative stress encountered within host cells [7].

E. coli, particularly pathogenic strains like EHEC, utilizes QS to modulate adherence to intestinal epithelial cells, promote biofilm formation on host tissues, and regulate the production of toxins that disrupt host cell functions. These adaptations enhance the bacterium's ability to establish infections and cause disease in susceptible hosts [8].

Correspondence to: Marcello Falasca, Department of Molecular Biology, University of Piemonte Orientale, Novara, Italy, E-mail: Mfalasca@edu.it

Received: 01-Mar-2024, Manuscript No. SCPM-24-26292; **Editor assigned:** 04-Mar-2024, SCPM-24-26292 (PQ); **Reviewed:** 18-Mar-2024, QC No. SCPM-24-26292; **Revised:** 25-Mar-2024, Manuscript No. SCPM-24-26292 (R); **Published:** 01-Apr-2024, DOI: 10.35248/2167-0897.24.13.079

Citation: Falasca M (2024) Signaling for Survival: Gene Regulation Mechanisms in *Salmonella* and *E. coli*. Single Cell Biol. 13:079.

Challenges and future directions

Despite significant progress in understanding QS mechanisms in *Salmonella* and *E. coli*, challenges remain in interpreting the full complexity of QS-regulated networks and their interactions with host environments. Future research efforts aim to elucidate additional QS systems, identify novel autoinducers, and develop strategies to disrupt QS signaling as a therapeutic approach against bacterial infections [9].

Cell-to-cell signaling processes, particularly quorum sensing, play pivotal roles in regulating gene expression and coordinating adaptive responses in *Salmonella* and *E. coli*. These mechanisms enable bacterial pathogens to sense and respond to changes in their surroundings, enhance virulence, and adapt to diverse host environments. Understanding how QS systems influence bacterial pathogenesis provides valuable insights into developing targeted therapies and interventions to fight *Salmonella* and *E. coli* infections effectively. Continued research into QS mechanisms promises to reveal new strategies for mitigating bacterial virulence and improving public health outcomes [10].

REFERENCES

1. Molero C, Rodríguez-Escudero I, Aleman A, Rotger R, Molina M, Cid VJ. Addressing the effects of *Salmonella* internalization in host cell signaling on a reverse-phase protein array. Proteomics. 2009;9(14):3652-3665.
2. Gupta T, Kaur H, Kapila S, Kapila R. *Lactobacillus fermentum* (MTCC-5898) alleviates *Escherichia coli*-induced inflammatory responses in intestinal epithelial cells by modulating immune genes and NF-κB signalling. J Appl Microbiol. 2021;131(6): 3008-3017.
3. Joon A, Chandel S, Ghosh S. Role of TLRs in EGFR-mediated IL-8 secretion by enteroaggregative *Escherichia coli*-infected cultured human intestinal epithelial cells. Cell Commun Signal. 2023;17(4):1355-1370.
4. Murthy AM, Sullivan MJ, Nhu NT, Lo AW, Phan MD, Peters KM, et al. Variation in hemolysin A expression between uropathogenic *Escherichia coli* isolates determines NLRP3-dependent *vs* -independent macrophage cell death and host colonization. FASEB J. 2019;33(6):7437-7450.
5. Zhang YG, Singhal M, Lin Z, Manzella C, Kumar A, Alrefai WA, et al. Infection with enteric pathogens *Salmonella typhimurium* and *Citrobacter rodentium* modulate TGF-beta/Smad signaling pathways in the intestine. Gut Microbes. 2018;9(4):326-337.
6. Li J, Zakariah M, Malik A, Ola MS, Syed R, Chaudhary AA, et al. Analysis of *Salmonella typhimurium* protein-targeting in the nucleus of host cells and the implications in colon cancer: An in-silico approach. Infect Drug Resist. 2020:2433-2442.
7. Lisowski C, Dias J, Costa S, Silva RJ, Mano M, Eulalio A. Dysregulated endolysosomal trafficking in cells arrested in the G1 phase of the host cell cycle impairs *Salmonella* vacuolar replication. Autophagy. 2022;18(8):1785-1800.
8. Chaussé AM, Roche SM, Moroldo M, Hennequet-Antier C, Holbert S, Kempf F, et al. Epithelial cell invasion by *Salmonella typhimurium* induces modulation of genes controlled by aryl hydrocarbon receptor signaling and involved in extracellular matrix biogenesis. Virulence. 2023;14(1):2158663.
9. Yeo GH, Lin L, Qi CY, Cha M, Gifford DK, Sherwood RI. A multiplexed barcodelet single-cell RNA-seq approach elucidates combinatorial signaling pathways that drive ESC differentiation. Cell stem cell. 2020;26(6):938-950.
10. Sobue R, Kurono N, Etchuya R, Maeda S. Identification of a novel DNA element that promotes cell-to-cell transformation in *Escherichia coli*. FEBS Lett. 2011;585(14):2223-2228.