Research & Reviews: Journal of Veterinary Sciences

Salmonella Survival Inside the Macrophage

Rahul M Nandre[1]*, Preeti Mahajan[2]

[1]College of Veterinary Medicine, Kansas State University, Manhattan, Kansas 66506, USA.

[2]College of Veterinary Medicine, Chonbuk National University, Jeonju, 561756 South Korea.

Review Article

Received date: 01/09/2015
Accepted date: 29/09/2015
Published date: 01/10/2015

***For Correspondence**

Rahul M Nandre, College of Veterinary Medicine, Kansas State University, Manhattan, Kansas 66506, USA. Tel: +785 770 5701.

E-mail: rahulbiotech@gmail.com

REVIEW

Salmonella survive and replicate in the macrophages [1]. The interaction of bacterial species such as Salmonellae with macrophages is a complex process. It involves the coordinated orchestration of signals and responses including bacterial and mammalian gene products. After the spread of Salmonella to the spleen and liver, the initial foci of infection consist of spatially separated phagocytes with a single bacterium carriage in each [2]. After bacterial phagocytosis, the expressed SPI-2 remodels the vacuole into an intracellular replication niche by altering endocytic trafficking, the vacuolar membrane, and modifying the vacuolar-associated actin polymerization and formation of tubular lysosomes, extending from the vacuole. SPI-2 translocates at effector proteins through the phagosomal membrane into the eukaryotic cell cytoplasm to promote intracellular replication and systemic spread [3]. These effector proteins have diverse subcellular localizations after translocation including the SCV, nucleus, and actin cytoskeleton. In macrophages, intracellular Salmonella induces a variety of regulatory systems, which promote bacterial surface remodeling of the protein, carbohydrate, and membrane components of the bacterial envelope. These are bacterial receptors, which induce bacterial defense responses to protect the bacteria from macrophage-killing mechanisms. The bacterial defense responses are essential to avoid killing by cyclic AMPs and nitrogen and oxygen radicals. Salmonella uses the nitrite transporter NirC to quench NO production by NO synthase (iNOS). Since NO has a repressing effect on SPI-2 expression, this enhances the chances of bacterial cells to survive inside macrophages [4]. Among different regulators, the two important regulator systems are OmpR and PhoPQ [5,6]. OmpR is important for the expression of genes, which encode a second TTSS located within SPI2 that is essential for intracellular survival [6]. In adition, the PhoPQ system regulates genes to increase resistance to anti-microbial defenses of macrophage. Also, it decreased sensing of Salmonella through alteration of bacterial molecules recognized by macrophage innate immune receptors [7]. PhoP-repressed genes composed of the SPI-1 TTSS and flagellar genes may decrease the host immunostimulatory activity [8,9]. PhoP-activated genes modulate resistance to antimicrobial peptides and reactive oxygen species [10]. After activation of PhoQ within the macrophage phagosome, resistance to anti-microbial peptides is acquired by modifications of the bacterial cell surface including modifications of LPS and membrane protein composition [11]. PhoQ-activated genes are also important in altering host processing and presentation of Salmonella antigens [12]. In addition, differential resistance/susceptibility of chicken lines to systemic salmonellosis has encouraged the identification of host loci associated with disease outcome. The SAL1 locus has been mapped to avian chromosome 5 and is thought to be involved in survival of Salmonellae within macrophages [13]. Additional candidate loci involved in the resistance to systemic salmonellosis include regions containing the Nramp1 and TLR4 [14].

REFERENCES

1. Nandre RM, et al. Immunogenicity of a Salmonella Enteritidis mutant as vaccine candidate and its protective efficacy against salmonellosis in chickens. Vet Immunol Immunopathol. (2011); 144: 299-311.
2. Sheppard M, et al. Dynamics of bacterial growth and distribution within the liver during Salmonella infection. Cell Microbiol. (2003);5: 593-600.

3. Ohlson M, et al. SseJ deacylase activity by Salmonella enterica serovar Typhimurium promotes virulence in mice. Infect Immun. (2005);73: 6249-6259.

4. Das P, et al. Novel role of the nitrite transporter NirC in Salmonella pathogenesis: SPI2-dependent suppression of inducible nitric oxide synthase in activated macrophages. Microbiology. (2009);155: 2476–2489.

5. Gunn JS, et al. Genetic and functional analysis of a PmrA-PmrB-regulated locus necessary for lipopolysaccharide modification, antimicrobial peptide resistance, and oral virulence of Samonella enterica serovar Typhimurium. Infect Immun. (2000);68: 6139-6146.

6. Lee AK, et al. OmpR regulates the two-component system ssrA-ssrB in Salmonella pathogenicity island J Bacteriol. (2000);182: 771-781.

7. Alpuche Aranda CM, et al. Salmonella Typhimurium activates virulence gene transcription within acidified macrophage phagosomes. Proc Natl Acad Aci USA. (1992);89: 10079-10083.

8. Adams P, et al. Proteomic detection of PhoPQ- and acid-mediated repression of Salmonella motility. Proteomics. (2001);1: 597-607.

9. Miao EA, et al. Cytoplasmic flagellin activates caspase-1 and secretion of interleukin 1-β via Ipaf. Nature Immunol. (2006);7: 569-575.

10. Fields PI, et al. A Salmonella locus that controls resistance to microbicidal proteins from phagocytic cells. Science. (1989);243: 1059–1062.

11. Gibbons HS, et al. Role of Mg2+ and pH in the modification of Salmonella lipid A after endocytosis by macrophage tumour cells. Mol Microbiol. (2005);55: 425-440.

12. Wick MJ, et al. The phoP locus influences processing and presentation of Salmonella Typhimurium antigens by activated macrophages. Mol Microbiol. (1995);16: 465-476.

13. Wigley P, et al. Salmonella enterica serovar Pullorum persists in splenic macrophages and in the reproductive tract during persistent, disease-free carriage in chickens. Infect Immun. (2001);69: 7873-7879.

14. Leveque G, et al. Allelic variation in TLR4 is linked to susceptibility to Salmonella enterica serovar Typhimurium infection in chickens. Infect Immun. (2003);71: 1116-1124.