Salmonella Survival Inside the Macrophage

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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 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 addition, 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


