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Title:	Role of Salmonella enterica Typhimurium secreted effector Protein, SteA in Host Cell Infection
Authors:	<a href="#">Shivansh</a>
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Abstract:	Salmonella enterica Typhimurium is a gram-negative bacterium that enters the body through contaminated food and water and causes salmonellosis to more than 550 million people each year. S. Typhimurium enters the gut where it interacts with epithelial cells and enters inside it in an endocytic fashion but it prevents its digestion by lysosomes and lives inside the vacuole. In the Salmonella containing vacuole (SCV), it multiplies feeding on the host nutrients and eventually invades into other epithelial cells as well as macrophages in the peyer's patches which are just beneath the epithelial cells. S. Typhimurium is able to do so with the help of effectors regulated by Salmonella pathogenicity island I (SPI-1) and SPI-2. Type three Secretion System- 1 (T3SS-1) and Type three Secretion System-2 (T3SS-2) translocate the effector proteins essential for its virulence, directly into host cell and has acquired such machinery through horizontal gene transfer through its course of evolution. Throughout its life inside the cell, S. Typhimurium uses the endocytic vacuole SCV that essentially help it to survive and replicate within the host cell while avoiding exposure and to live undetected. SteA is an effector molecule secreted by both T3SS- 1 and T3SS-2 and is shown to be crucial for suppression of host's innate immune responses in SPI-1 condition. Correlating with the work already done on the role of this effector molecule in SPI-1 conditions, we aim to see how it modulates host immune responses in SPI-2 conditions and affects cytotoxicity upon infection in healthy cells. In this study towards exploring whether SteA plays any role on host cell death we observed that SteA promotes cytotoxicity in macrophages.
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