Title:	Characterization of cell death responses induced by salmonella enetrica typhimurium effector protein stea
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Abstract:	Salmonella enterica serovar Typhimurium is a pathogenic gram-negative bacterium responsible for causing self-limiting gastroenteritis through contaminated food and water. Salmonella uses its secretion system to invade in epithelial cells of humans and disseminate them into the body by living inside a vacuole known as Salmonella-containing vacuole. Type III secretion system translocates the effector protein, encoded within Salmonella pathogenicity island-1 (SPI-1) and SPI-2 where SPI-1 induced effector protein are responsible for causing invasion and SPI-2 effector proteins are involved in the intracellular replication and dissemination of Salmonella typhimurium inside the host cell. Similarly, SteA is an effector protein induced by T3SS encoded by both SPI-1 and SPI-2. It is an essential molecule that helps in the upregulation of the expression of genes that regulate ECM organization and reported as it interacts with Cullin-1, a component of SCF E3 ubiquitin ligases and responsible for the virulence of Salmonella Typhimurium. This project aimed to observe the effect of SteA effector protein on cell cytotoxicity in SPI-2 induced conditions and characterizing the type of cell death SteA promoting in macrophages. The experiments involved checking the cell death by LDH, which indicated that SteA could directly affect macrophages cell health. Further, various experiments were performed to check apoptosis markers to characterize the type of cell death occurring in macrophages due to SteA. The data revealed that SteA could affect the cell cycle progression.
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