Title:	Association of Salmonella Typhimurium effector protein SteA with host cellular proteins and its role in cell death mechanisms
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Abstract:	Salmonella enterica serovar Typhimurium is a major food-borne pathogen which causes self-limiting gastroenteritis in humans and typhoid-like diseases in mice. It gets transmitted through contaminated food, poultry, meat etc. and leads to 155,000 deaths each year. Salmonella Typhimurium has large gene cassettes in its chromosome called the Salmonella pathogenicity islands (SPIs) which encode/regulate various virulence factors required for successful pathogenesis. SteA is an effector protein which is regulated by both SPI-1 and SPI-2. Under SPI-1 condition SteA suppresses the proinflammatory responses of the host cell. To find other functions associated with SteA, following GST- pull down mass spectrometry data analysis was done using bioinformatics' tools which suggested that SteA might be involved cell death mechanisms and could modulate the DNA damage repair pathways. It was also observed that SteA is involved in Salmonella Typhimurium-induced cell death in RAW264.7 murine macrophages and hinders the poly-ubiquitination of PCNA. Co-immunoprecipitation studies suggested the interaction of SteA with Rab-7 and Rac-2. Further, experiments can help reveal the complex interplay of the DNA repair pathways involved in response to infection of Salmonella Typhimurium.
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