Elucidating the role of SteA of Salmonella typhimurium in modulating host responses

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Salmonella typhimurium is a gram negative, invasive bacteria which is known to cause salmonellosis. S. typhimurium is about 85% similar to Salmonella typhi which causes typhoid in humans. Before invading, Salmonella secrete various effectors proteins directly into the cells of the host via a needle like assembly called the type 3 secretion system (T3SS-1). Further, following the invasion, again Salmonella secrete some effector proteins into the invaded cells via T3SS (T3SS-2). These effectors have been shown to modulate various functions of the cell, thus helping Salmonella to thrive in the host. These effector proteins are mainly regulated by Salmonella pathogenecity islands (SPIs). The effector proteins secreted via T3SS-1 can affect the immune responses of host cells.
SteA is an effector protein secreted by both T3SS-1 and T3SS-2 during infection. However, the role of SteA in T3SS-1 has not been characterized. To understand the role of SteA, we infected mice with wild type (wt) and ΔsteA strains and observed lower survival of mice infected with ΔsteA compared to the wild type. Further, we observed that SteA is involved in the suppression of immune responses of the host cells during infection. We have further deduced the molecular mechanism involved in this immune suppression using techniques such as molecular biology, ELISA, western blotting, luciferase assay, confocal microscopy etc.

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