Title:	Salmonella Effector SteA Suppresses Proinflammatory Responses of the Host by Interfering With IκB Degradation
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Abstract:	Salmonella enterica serovar Typhimurium is known to cause its virulence by secreting various effector proteins directly into the host cytoplasm via two distinct type III secretion systems (T3SS-1 and T3SS-2). Generally, T3SS-1-delivered effectors help Salmonella Typhimurium in the early phases of infection including invasion and immune modulation of the host cells, whereas T3SS-2 effectors mainly help in the survival of Salmonella Typhimurium within the host cells including maintenance of Salmonella-containing vacuole, replication of the bacteria, and dissemination. Some of the effectors are secreted via both T3SS-1 and T3SS-2, suggesting their role in distinct phases of infection of host cells. SteA is such an effector that is secreted by both T3SS-1 and T3SS-2. It has been shown to control the membrane dynamics of the Salmonella-containing vacuole within the host cells in the late phases of infection. In this manuscript, toward characterizing the T3SS-1 function of SteA, we found that SteA suppresses inflammatory responses of the host by interfering with the nuclear factor kappa B pathway. Our initial observation showed that the mice infected with steA-deleted Salmonella Typhimurium (ΔsteA) died earlier compared to the wild-type bacteria due to heightened immune responses, which indicated that SteA might suppress immune responses. Furthermore, our study revealed that SteA suppresses immune responses in macrophages by interfering with the degradation of IκB, the inhibitor of nuclear factor kappa B. SteA suppresses the ubiquitination and hence degradation of IκB by acting on Cullin-1 of the Skp-1, Cullin-1, F-box (SCF)-E3 ligase complex. Our study revealed that SteA suppresses a key step necessary for E3 ligase activation, i.e., neddylation of Cullin-1 by interfering with dissociation of its inhibitor Cand-1.
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