Abstract

Salmonella enterica are Gram-negative bacteria that cause systemic disease in their specific hosts. One of the recently appreciated features of Salmonella pathogenicity is the capacity of the bacteria to impair host adaptive immunity by interfering with DC function and T cell activation. It is likely that this feature of virulent Salmonella is needed to promote systemic dissemination in the host. Recent studies have suggested explanations for some of the molecular mechanisms developed by virulent Salmonella to impair DC and T cell function. Several of these mechanisms require the expression of virulence genes encoded within Salmonella pathogenicity islands. Targeted deletion of these genes diminishes Salmonella pathogenicity and leads to efficient activation of T cells by Salmonella-infected DCs. In this review, recent data that support the subversion of DC function by Salmonella as a means to evade host adaptive immunity and cause systemic infection are discussed. These new findings suggest a new pathogenesis model with DCs as key targets for Salmonella virulence factors.