Functional Dissection of *Salmonella* SPI2 injectisome

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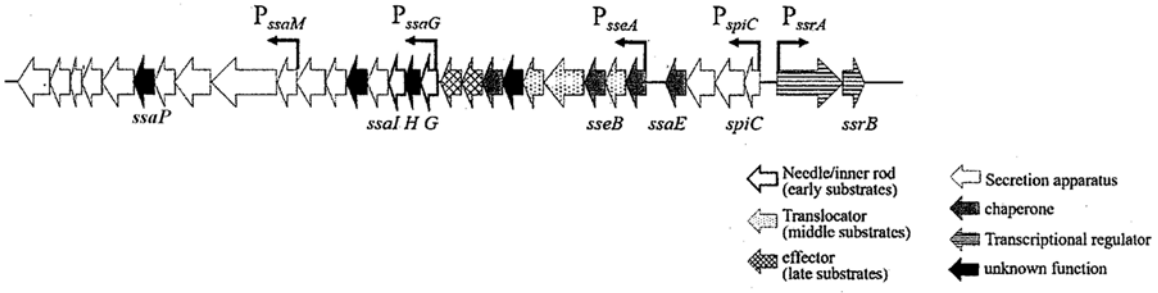
The type III secretion system (T3SS) is essential for the pathogenic potential of Gram-negative bacteria by delivering effector proteins directly into the eukaryotic cytoplasm. At its core lie the injectisome (a transmembrane secretion apparatus) and a network of specialized chaperones that target secretory proteins (secretory substrates; hereafter 'substrates') to the antechamber of the injectisome. Secretion of substrates through the injectisome occurs in consecutive steps and different switching mechanisms ensure the secretion hierarchy; secretion of early/middle/late substrates. Although several export apparatus components have been shown to be responsible for mediating different steps of secretion, the precise mechanism including substrate switching is not fully understood.

The *Salmonella* pathogenicity island 2 (SPI2) T3SS is assembled after acidification (pH ~5.0) of *Salmonella* containing vacuoles (SCV) in host cell. Bacteria grown *in vitro* at pH 5.0 secrete translocator (middle substrate) but negligible levels of effectors (late substrates). Exposure of bacteria to pH 7.2 after growth at low pH causes the secretion of effectors, implicating that the secretion of early and middle substrates of SPI2-T3SS can be specifically dissected at low pH. Here I will describe that SsaH is a novel chaperone responsible for secretion of early substrate (SsaI; inner rod) and another chaperone SsaE regulates secretion of early substrate (SsaI) as well as middle substrate (SseB;translocator). Furthermore, I will present the possible role of SsaP which is assumed to be a molecular ruler on the substrate switching.



Genetic organization of *Salmonella* pathogenicity island 2 (SPI2)