Role of the Salmonella pathogenicity island 1 effector proteins SipA, SopB, SopE, and SopE2 in Salmonella enterica subspecies 1 serovar Typhimurium colitis in streptomycin-pretreated mice.

Abstract:
Salmonella enterica subspecies 1 serovar Typhimurium (serovar Typhimurium) induces enterocolitis in humans and cattle. The mechanisms of enteric salmonellosis have been studied most extensively in calf infection models. The previous studies established that effector protein translocation into host cells via the Salmonella pathogenicity island 1 (SPI-1) type III secretion system (TTSS) is of central importance in serovar Typhimurium enterocolitis. We recently found that orally streptomycin-pretreated mice provide an alternative model for serovar Typhimurium colitis. In this model the SPI-1 TTSS also plays a key role in the elicitation of intestinal inflammation. However, whether intestinal inflammation in calves and intestinal inflammation in streptomycin-pretreated mice are induced by the same SPI-1 effector proteins is still unclear. Therefore, we analyzed the role of the SPI-1 effector proteins SopB/SigD, SopE, SopE2, and SipA/SspA in elicitation of intestinal inflammation in the murine model. We found that sipA, sopE, and, to a lesser degree, sopE2 contribute to murine colitis, but we could not assign an inflammation phenotype to sopB. These findings are in line with previous studies performed with orally infected calves. Extending these observations, we demonstrated that in addition to SipA, SopE and SopE2 can
induce intestinal inflammation independent of each other and in the absence of SopB. In conclusion, our data corroborate the finding that streptomycin-pretreated mice provide a useful model for studying the molecular mechanisms of serovar Typhimurium colitis and are an important starting point for analysis of the molecular events triggered by SopE, SopE2, and SipA in vivo.