Flagella and chemotaxis are required for efficient induction of Salmonella enterica serovar Typhimurium colitis in streptomycin-pretreated mice.

Abstract:
Salmonella enterica subspecies 1 serovar Typhimurium is a common cause of gastrointestinal infections. The host's innate immune system and a complex set of Salmonella virulence factors are thought to contribute to enteric disease. The serovar Typhimurium virulence factors have been studied extensively by using tissue culture assays, and bovine infection models have been used to verify the role of these factors in enterocolitis. Streptomycin-pretreated mice provide an alternative animal model to study enteric salmonellosis. In this model, the Salmonella pathogenicity island 1 type III secretion system has a key virulence function. Nothing is known about the role of other virulence factors. We investigated the role of flagella in murine serovar Typhimurium colitis. A nonflagellated serovar Typhimurium mutant (fliGHI) efficiently colonized the intestine but caused little colitis during the early phase of infection (10 and 24 h postinfection). In competition assays with differentially labeled strains, the fliGHI mutant had a reduced capacity to get near the intestinal epithelium, as determined by fluorescence microscopy. A flagellated but nonchemotactic cheY mutant had the same virulence defects as the fliGHI mutant for causing colitis. In competitive infections, both mutants colonized the intestine of streptomycin-pretreated mice by day 1 postinfection but were outcompeted by the wild-type strain by day 3.
postinfection. Together, these data demonstrate that flagella are required for efficient colonization and induction of colitis in streptomycin-pretreated mice. This effect is mostly attributable to chemotaxis. Recognition of flagellar subunits (i.e., flagellin) by innate immune receptors (i.e., Toll-like receptor 5) may be less important.

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