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Autor(en) des Beitrags:
Hapfelmeier, S; Stecher, B; Barthel, M; Kremer, M; Müller, AJ; Heikenwalder, M; Stallmach, T; Hensel, M; Pfeffer, K; Akira, S; Hardt, WD

Titel des Beitrags:
The Salmonella pathogenicity island (SPI)-2 and SPI-1 type III secretion systems allow Salmonella serovar typhimurium to trigger colitis via MyD88-dependent and MyD88-independent mechanisms.

Abstract:
Salmonella typhimurium can colonize the gut, invade intestinal tissues, and cause enterocolitis. In vitro studies suggest different mechanisms leading to mucosal inflammation, including 1) direct modulation of proinflammatory signaling by bacterial type III effector proteins and 2) disruption or penetration of the intestinal epithelium so that penetrating bacteria or bacterial products can trigger innate immunity (i.e., TLR signaling). We studied these mechanisms in vivo using streptomycin-pretreated wild-type and knockout mice including MyD88(-/-) animals lacking an adaptor molecule required for signaling via most TLRs. The Salmonella SPI-1 and the SPI-2 type III secretion systems (TTSS) contributed to inflammation. Mutants that retain only a functional SPI-1 (M556; sseD::aphT) or a SPI-2 TTSS (SB161; DeltainvG) caused attenuated colitis, which reflected distinct aspects of the colitis caused by wild-type S. typhimurium: M556 caused diffuse cecal inflammation that did not require MyD88 signaling. In contrast, SB161 induced focal mucosal inflammation requiring MyD88. M556 but not SB161 was found in intestinal epithelial cells. In the lamina propria, M556 and SB161 appeared to reside in different leukocyte cell populations as indicated
by differential CD11c staining. Only the SPI-2-dependent inflammatory pathway required aroA-dependent intracellular growth. Thus, S. typhimurium can use two independent mechanisms to elicit colitis in vivo: SPI-1-dependent and MyD88-independent signaling to epithelial cells and SPI-2-dependent intracellular proliferation in the lamina propria triggering MyD88-dependent innate immune responses.