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Salmonella Pathogenicity Island 2 (SPI2) effectors facilitate enterocyte transmigration in neonatal non-typhoidal Salmonella infections

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Non-typhoidal *Salmonella* (NTS) belong to the most prevalent causes of infectious diarrheal disease in humans and pigs worldwide, but also contribute to invasive infections in infants. The pathogenicity of NTS is conferred by horizontally acquired chromosomal regions, called *Salmonella* pathogenicity islands (SPIs), encoding sets of effector proteins delivered into the host cell via specific type-three secretion systems. Several *in vitro* studies identified SPI2 as a requirement for the establishment of an intracellular compartment allowing bacterial survival and replication inside the host cell, the *Salmonella* containing vacuole (SCV).
We used our previously established neonatal mouse model to clarify the role of SPI2 in establishment and progression of systemic NTS infections in the neonate host.
Oral infection with wildtype and SPI2-deficient NTS resulted in similar bacterial loads of the gastrointestinal tract, whereas re-isolation rates of mutants from systemic organs were significantly decreased. In contrast to the general understanding of SPI2 as prerequisite for SCV formation *in vitro*, mutants established and maintained SCVs and even grew to high numbers without harming the host cell. By evaluating isogenic SPI2 effector protein deficient *Salmonella* strains, we demonstrate that the effector SifA significantly contributes to the SPI2-dependent phenotype *in vivo*. Its absence prevents transmigration of enterocytes and subsequent systemic dissemination.

Keywords

Salmonella , effector proteins, neonatal infection, enterocyte transmigration

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Professional Status of the Speaker

Postdoc

Junior Scientist Status

No, I am not a Junior Scientist.

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