Salmonella utilize multiple modes of infection

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Scientists from the Helmholtz Centre for Infection Research (HZI) in Braunschweig, Germany have discovered a new, hitherto unknown mechanism of *Salmonella* invasion into gut cells: In this entry mode, the bacteria exploit the muscle power of cells to be pulled into the host cell cytoplasm. Thus, the strategies *Salmonella* use to infect cells are more complex than previously thought. According to the World Health Organization, the number of *Salmonella* infections is continuously rising, and the severity of infections is increasing. One of the reasons for this may be the sophisticated infection strategies the bacteria have evolved. The striking diversity of invasion strategies may allow *Salmonella* to infect multiple cell types and different hosts.

"*Salmonella* do not infect their hosts according to textbook model," says Theresia Stradal, group leader at the Helmholtz Centre in Braunschweig, who has recently accepted a call to the University of Münster. "Only a single infection mechanism has seriously been discussed in the field up till now –without understanding all the details," adds Klemens Rottner, now Professor at the University of Bonn.

All entry mechanisms employed by *Salmonella* target the so-called actin cytoskeleton of the host cell. Actin can polymerise into fine and dynamic fibrils, also called filaments, which associate into networks or fibres. These structures stabilise the cell and enable it to move, as they are constantly built up and taken down. One of the most important core elements is the Arp2/3 complex that nucleates the assembly of actin monomers into filaments.

Extensions of the cell membrane are filled with actin filaments. In the commonly accepted infection mechanism, *Salmonella* abuses the Arp2/3 complex to enter the host cell: the bacteria activate the complex and thus initiate the formation actin filaments and development of prominent membrane extensions, so-called ruffles. These ruffles surround and enclose the bacteria so that they end up in the cell interior. Last year, the research groups headed by Theresia Stradal and Klemens Rottner discovered that *Salmonella* can also reach the cell interior without initiating membrane ruffles. With this, the researchers disproved a long-standing dogma.

In their recent study, the experts from Braunschweig now describe a completely unknown infection mechanism. The results have just appeared in the latest issue of the leading journal *Cell Host & Microbe*. In this new infection mechanism, *Salmonella* also manipulate the actin cytoskeleton of the host cell. This time, however, they do not induce the generation of new filaments, but activate the motor protein myosin II. The interplay of actin and myosin II in muscle cells is well known: in a contracting muscle, myosin and actin filaments slide along each other and this way shorten the muscle; it contracts.

In epithelial cells, the contractile structures are less organised but work similarly. Here, actin and myosin II form so-called stress fibres that tightly connect to the membrane. During an infection, stress fibres at the entry site can contract and pull the bacteria into the cell. "This way of infection operates independently from the Arp2/3 complex, the central component of the 'classic' infection mechanism," says Jan Hänisch, who worked on this project as postdoctoral researcher.

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