

Researchers uncover secrets of salmonella's stealth attack

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A single crafty protein allows the deadly bacterium *Salmonella enterica* to both invade cells lining the intestine and hijack cellular functions to avoid destruction, Yale researchers report in the April 17 issue of the journal *Cell*.

This evolutionary slight-of-hand sheds new insights into the lethal tricks of *Salmonella*, which kills more than 2 million people a year.

"In evolutionary terms, this hijacking of <u>cellular machinery</u> to diversify the function of a bacterial protein is mind boggling," said Jorge Galan, senior author of the paper and the Lucille P. Markey Professor of Microbial Pathogenesis and Cell Biology and chair of microbial pathogenesis at Yale.

Salmonella causes disease when it takes control of <u>cells</u> lining the intestinal track using its own specialized "nano-syringe" called a type III secretion system. Using this structure, *Salmonella* injects bacterial proteins that mimic proteins of the host cell and help the pathogen avoid destruction.

The Yale study describes the crucial role a bacterial protein called SopB plays in both *Salmonella*'s forced entry into the cell and its subsequent internal camouflage act. First, SopB works within the external membrane of the cell, called the plasma membrane, to coax the cell into taking in the pathogen, which is then encapsulated within a tiny bubble-like compartment called a vesicle.



SopB's second trick helps prevent the vesicle from being sucked into the lyosome, the organelle within the cell that degrades proteins. In order to accomplish this, SopB must move from the plasma membrane of the cell to the membrane of the internal vesicle containing the pathogen. The Yale group found that *Salmonella* coaxes the cell to "mark" the SopB protein with a tag called ubiquitin. Addition of this tag makes the bacterial protein recognizable to the cellular machinery that normally moves proteins from the plasma membranes to internal vesicles.

"These studies provide a unique insight into the mechanisms by which this important pathogen causes disease," Galan said. "In addition, this finding may point to a novel paradigm that may be applicable to other important pathogens."

Source: Yale University (<u>news</u>: <u>web</u>)

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