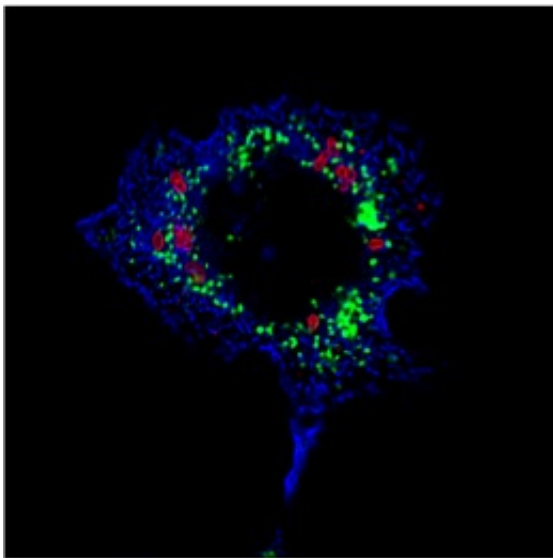


# Salmonella jams signals from bacteria-fighting mast cells

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In this *Salmonella*-infected mast cell, the *Salmonella* are shown in red. The green areas are packages of signaling molecules harbored in mast cells. Following infection by *Salmonella*, mast cells are no longer able to release their pre-stored chemical signals. Credit: Duke Medicine

A protein in *Salmonella* inactivates mast cells—critical players in the body's fight against bacteria and other pathogens—rendering them unable to protect against bacterial spread in the body, according to researchers at Duke Medicine and Duke-National University of Singapore (Duke-NUS).

The study, conducted in mice, was published Dec. 12, 2013, in the

journal *Immunity*.

"Ever since [mast cells](#) were discovered to be critical mobilizers of the body's powerful immune system, it has always been suspected that certain pathogens would have evolved mechanisms directed at undermining this cell," said senior study author Soman N. Abraham, Ph.D., professor of pathology, immunology, and molecular genetics and microbiology at Duke Medicine and professor of emerging infectious diseases at Duke-NUS.

*Salmonella* bacteria are a leading cause of foodborne illness. According to the CDC, approximately 42,000 cases of *Salmonella* infection are reported each year in the United States, but since many mild cases are not diagnosed, the number is likely much higher. Often transmitted through contaminated eggs, meat, raw fruits and vegetables, a 2012 *Salmonella* outbreak linked to cantaloupe sickened hundreds of people across 24 states.

While most individuals infected with *Salmonella* recover quickly, the infection can cause serious illness or even death, particularly among those with [weakened immune systems](#). *Salmonella* is also becoming increasingly resistant to antibiotic treatment, leading researchers to develop vaccines to try and prevent the bacterial infection.

Studies have shown that *Salmonella* can rapidly invade the body's cells and hinder the immune system from mounting responses against future infections by impeding the actions of specific immune cells or targeting the lymph nodes. The rapid spread of *Salmonella* after breaching the gut barrier, however, suggests a more immediate mechanism for subverting the immune system.

An important component of the immune system is the mast cell, a distinct type of immune cell that initiates an early response to combat

and clear invading pathogens. Mast cells are located in large numbers in the skin, gut, lung and bladder lining, which are common sites for pathogens to enter and attack the body.

Upon encountering invading bacteria or viruses, mast cells release large amounts of chemical signals, which recruit various pathogen-clearing immune cells from the blood to the site of infection. However, *Salmonella* has been shown to be an exception, as mast cells do not clear the bacteria.

To learn how *Salmonella* handicaps mast cells, Abraham and his colleagues studied *Salmonella* infection in mice. They found that when mice were exposed to *Salmonella* via injection or oral administration, a protein called *Salmonella* protein tyrosine phosphatase (SptP) shut down the mast cells' ability to release chemical signals without impacting other cellular functions.

The researchers observed that SptP inactivated at least two key cellular components involved in exporting [chemical signals](#) out of mast cells. As a result of the mast cells being unable to call for help, [immune cells](#) were not recruited to the infection site, allowing *Salmonella* to multiply and spread unchecked.

In another experiment, the researchers administered SptP to mice infected with *E. coli*, a relatively innocuous type of bacteria. With SptP suppressing the mast cells, *E. coli* was able to spread inside the mouse just as quickly as *Salmonella*, suggesting that inactivating mast cells is a key determinant in the spread of pathogens. The researchers also found that *Yersinia pestis*, the pathogen responsible for plague, expressed an SptP-like protein that also suppressed mast cells. They now think that the spread of plague bacteria in the body may also involve mast cell suppression.

By pinpointing SptP as the mechanism that inactivates mast cells in *Salmonella* infection allowing for bacterial spread in the body, Abraham said the researchers can apply their findings to seek out better preventive and treatment options for this significant public health concern.

"The current vaccines against *Salmonella* are largely ineffective and short lived," Abraham said. "Our discovery of the virulent properties of SptP raises the possibility of using this information to evoke effective and long-lived protection against *Salmonella* infection."

Provided by Duke University Medical Center

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