Study finds new way deadly food-borne bacteria spread

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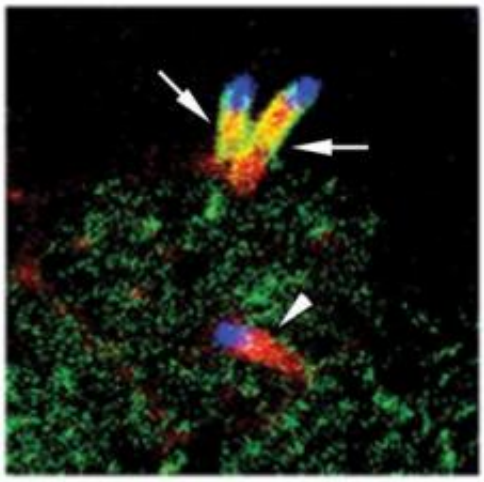

Listeria moves through the cytoplasm of human cells by using part of the cell "cytoskeleton" called actin. In this image, *Listeria* is labeled in blue and actin is labeled in red. Note that actin forms a tail-like structure behind bacteria. These actin tails push *Listeria* through the cytoplasm of the human cell. The arrowhead indicates a bacteria moving in the cytoplasm. *Listeria* that have contacted the plasma membrane of the cell can deform that membrane into protrusions. In this image, protrusions (indicated with arrows) are visible as yellow finger-like projections with bacteria at their tips. These protrusions push into adjacent healthy human cells, thereby allowing spreading of *Listeria*. In their work published in *Nature Cell Biology*, Ireton and co-workers have identified a Listeria protein that stimulates the formation of protrusions and spreading. Their findings suggest that this protein, called InlC, acts by relieving tension at the plasma membrane. Credit: Keith Ireton

University of Central Florida Microbiology Professor Keith Ireton has uncovered a previously unknown mechanism that plays an important role in the spread of a deadly food-borne bacterium.

Listeria monocytogenes is a bacterium that can cause pregnant women to lose their fetuses and trigger fatal cases of meningitis in the elderly or people with compromised immune systems. The bacterium has been linked to outbreaks traced to food processing plants in the U.S. and Canada.

In 2002, a multi-state outbreak of listeriosis - the serious disease caused by *Listeria* -- resulted in 46 confirmed cases, seven deaths and three stillbirths or miscarriages. Those cases in eight states were linked to people eating contaminated sliced turkey deli meat. From January to August 1985, there was another outbreak with 142 cases of listeriosis.

Scientists have long known that *Listeria* spreads from one human cell to another. Bacteria growing in one cell move fast enough to create a finger-like structure that protrudes from the cell and pushes into an adjacent cell. The bacteria then infect the adjacent cell.

Ireton and his team have discovered a previously unknown second process that aids in the spread of bacteria to healthy cells. The process, which gradually overwhelms the second cell's ability to defend itself from infection, is featured in this week's edition of the science journal Nature Cell Biology.

The plasma membrane, or outer layer, of healthy human cells normally exhibits tension. Such tension might be expected to prevent *Listeria* from spreading to adjacent uninfected cells. However, Ireton's lab found that a *Listeria* protein called InlC appears to relieve tension at the plasma membrane in infected cells, making it easier for moving bacteria to deform the membrane and then spread into adjacent, healthy cells.

Ireton's laboratory also reports that the way InlC relieves tension is by blocking the function of a human protein called Tuba. The normal role of Tuba in uninfected human cells appears to be to help generate tension at the plasma membrane. The *Listeria* protein InlC inactivates Tuba, reducing that tension and enabling bacteria to spread to nearby cells.

"The idea that a pathogenic bacterium can spread by controlling membrane tension in the human cell has not been previously described in the scientific literature," Ireton said. "Our discovery could have relevance for bacterial pathogens that cause Shigellosis or Rocky Mountain spotted fever, as these bacteria resemble Listeria in their ability to move inside the host cell and spread."

More research is needed, but Ireton says that discovering this mechanism could aid in future therapies and perhaps open a window into understanding how certain bacterial pathogens cause disease.

Source: University of Central Florida (news : web)