

# Researchers determine how Legionnaires' bacteria proliferate, cause disease

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A University of Louisville scientist has determined for the first time how the bacterium that causes Legionnaires' disease manipulates our cells to generate the amino acids it needs to grow and cause infection and inflammation in the lungs. The results are published online today (Nov. 17) in *Science*.

Yousef Abu Kwaik, Ph.D., the Bumgardner Endowed Professor in [Molecular Pathogenesis](#) of [Microbial Infections](#) at UofL, and his team believe their work could help lead to development of [new antibiotics](#) and vaccines.

"It is possible that the process we have identified presents a great target for new research in antibiotic and [vaccine candidates](#), not only for Legionnaires' disease but in other bacteria that cause illness," he said.

According to the [Centers for Disease Control and Prevention](#), Legionnaires' disease is a [lung infection](#) caused by the bacterium called Legionella. The bacterium got its name in 1976, when many people who went to a Philadelphia convention of the American Legion suffered from an outbreak of pneumonia of unknown causes that was later determined to be caused by the bacterium. Each year, between 8,000 and 18,000 people are hospitalized with Legionnaires' disease in the U.S. There is no vaccine currently available for it.

For two years, the researchers examined Legionella which is an intercellular bacterium that exists in [amoebae](#) in the water systems; it is

transmitted to humans through inhalation of [water droplets](#). Cooling towers and whirlpools are the major sources of transmission. The bacterium uses the amoeba's cellular process to "tag" proteins, causing them to degrade into their basic elements of [amino acids](#). These amino acids are used by the bacteria as the main source of energy to grow and cause disease.

"The bacteria live on an 'Atkins diet' of low carbs and [high protein](#), and they trick the host cell to provide that specialized diet," Abu Kwaik said.

The same process occurs in a host – animal or human – who inhales the bacterium and is diagnosed with Legionnaires' disease. However, the bacteria do not tag the proteins, but rather trick the host into tagging the proteins for degradation to generate the amino acids.

In the laboratory, Abu Kwaik and his team saw that by inactivating the bacterial virulence factor responsible for tricking the cell into tagging proteins for degradation in mice models, the pulmonary disease was totally prevented. This was totally due to disabling the bacteria from generating amino acids, he said.

The process was then reversed, and the disease became evident when the mice, infected by the disabled bacteria, were injected with amino acids to compensate for the inability of the altered bacteria.

"Bacteria need to live on high protein and amino acids as sources of nutrition and energy in order to replicate in a host. This is what causes pulmonary disease," Abu Kwaik said. "No one has known how they generate sufficient sources of nutrients from the host to proliferate. Our work is the first to identify this process for any bacteria that cause disease."

He added that the type of host infected does not appear to affect the

process. "Whether in a single-cell amoeba or a multi-cellular mammal, Legionella seems to know what to do; the process is the same, and is highly conserved through evolution. By interfering with the bacterium's sources of nutrients, we can stop it from thriving and causing disease."

Examining nutrient sources for organisms with the goal of stopping them from acquiring nutrients is a relatively new arena of basic research that deserves further study, he said. "We went after the basics – the food and energy source – which are prerequisite for the bacteria to grow and cause disease. It is not a process that is well understood yet, but by first discovering how an organism gets nutrients by tricking the host into degrading proteins, and then interfering with that process, we can, in effect, starve it to death and prevent or treat the disease."

Provided by University of Louisville

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