Powerful bacterial immune response defined by new study

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T-cells, the elite guard of the immune system in humans and other mammals, ignore normal biologic protocol and swing into high gear when attacked by certain fast-moving bacteria, reports a team of researchers led by a UC Davis immunologist.

The description of this previously undefined immune pathway provides information vital for designing vaccines and medicines to prevent or treat deadly infectious diseases caused by bacteria such as *Salmonella* and *Chlamydia*. The results from this recent mouse-based study will be reported online Feb. 6 in the journal *Immunity*.

"Our study shows that the body's immune system functions very differently when it faces a rapidly growing pathogen like *Salmonella* or *Chlamydia*," said immunologist Stephen McSorley, an associate professor at UC Davis' Center for Comparative Medicine, which investigates diseases that afflict both humans and animals.

"The strict rules that normally govern T-cell activation are relaxed so that the host animal has the best possible chance of a maximal response and ultimately staying alive," he said.

T-cells, which belong to a group of white blood cells called lymphocytes, normally respond defensively to the presence of substances known as antigens, which are produced by invading bacteria and viruses. It's widely known that T-cells launch an immune defense when they recognize specific antigens.

However in this study, the researchers demonstrated in the mouse that certain T-cells don't require the presence of specific antigens to launch an effective immune response. During fast-moving *Salmonella* and *Chlamydia* infections, a cascade of other antimicrobial interactions occur that trigger these T-cells to respond defensively to the bacterial attacks, even without the presence of specific antigens.

The researchers also showed that when this defensive pathway was disrupted during *Salmonella* infection, the mice had greater difficulty getting rid of the bacterial disease.

The researchers note that further study is needed to determine if this newly defined antimicrobial pathway also can provide protection against co-infections by multiple disease-causing microbes.

Provided by UC Davis