



Image Source: CDC

Shigellosis, also known as “bacillary dysentery” is an acute infection of the intestine caused by *Shigella* bacteria. Complications from this infection lead to more than 200,000 deaths every year, primarily among infants. UC Berkeley co-authors, Patrick Mitchell and Justin Roncaoli, develop the first oral infection mouse model for *Shigella* infection that recapitulates human disease. They demonstrate a specific host-protective function for inflammasomes in intestinal epithelial cells. These findings, published in [Elife](#), open up new prospects for the development of vaccines to combat this group of bacteria, which is on the [WHO list of 12 priority pathogens](#).

Shigella is a group of pathogenic enterobacteria (bacteria found in the digestive tract) that cause bacillary dysentery, which is also known as shigellosis. They are transmitted via the fecal-oral route, for example through food or water contaminated with fecal matter. After ingestion, Shigella bacteria invade the cells of the intestinal wall and then the colonic mucosa, causing major inflammation combined with severe tissue damage. This causes symptoms such as abdominal pain, vomiting, diarrhea containing blood or mucus, and fever. With no commercialized vaccine (the infection is currently treated with antibiotics), shigellosis remains a major public health problem, and without a mouse model, progress has been very slow.

“It has been a longstanding mystery why Shigella is a human-specific pathogen and is unable to infect mice” lead author and UC Berkeley professor Russell Vance, said. “Our results suggest that there is a mouse-specific resistance pathway that involves a special component of the immune system called inflammasomes. By genetic elimination of a particular inflammasome from mice we can render mice susceptible to Shigella infection”.

There is currently no licensed Shigella vaccine, and very limited knowledge of what vaccine-induced immune responses would be desirable to elicit to mediate protection. This new mouse model will finally allow to address fundamental questions about the immune response to Shigella, and the development of new vaccine candidates and therapeutics.



The Vance Lab (May 2019) - <https://vancelab.berkeley.edu/>

Special shout-out to Justin Roncaioli, a [CEND 2019 fellow](#)!