

Viral production is not essential for deaths caused by food-borne pathogen

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Escherichia coli. Credit: Rocky Mountain Laboratories, NIAID, NIH

The replication of a bacterial virus is not necessary to cause lethal disease in a mouse model of a food-borne pathogen called Enterohemorrhagic *Escherichia coli* (EHEC), according to a study

published January 10 in the open-access journal *PLOS Pathogens* by Sowmya Balasubramanian, John Leong and Marcia Osburne of Tufts University School of Medicine, and colleagues. The surprising findings could lead to the development of novel strategies for the treatment of EHEC and life-threatening kidney-related complications in children.

EHEC is a Shiga toxin-producing pathogen associated with serious disease outbreaks worldwide, including more than 390 food-poisoning outbreaks in the U.S. in the last two decades. Humans acquire EHEC by ingesting contaminated food or water, or through contact with animals or their environment. Infection may progress to life-threatening [hemolytic uremic syndrome](#) (HUS), the leading cause of kidney failure in children. Treatment for EHEC or HUS remains elusive, as antibiotics have been shown to exacerbate disease. The bacteria begin to produce Shiga toxin when a virus present in the EHEC genome is induced to leave its dormant state and begin to replicate, a process promoted by many antibiotics. Until now, it was generally believed that extensive virus replication was necessary for the bacteria to produce sufficient toxin to cause disease.

Using an EHEC disease mouse model, the authors show that an inducing signal needed to begin viral replication is essential for [lethal disease](#). But surprisingly, sufficient Shiga toxin was produced to cause lethal mouse disease, even without [viral replication](#). According to John Leong, one of the authors, "An important next step will be to learn what parts of the viral life cycle occur in human patients, and whether there are ways to prevent those aspects that lead to disease".

More information: *PLOS Pathogens* (2019).
[journals.plos.org/plospathogen ... journal.ppat.1007474](https://journals.plos.org/plospathogen/article/doi/10.1371/journal.ppat.1007474)

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