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Study reveals how SARS virus hijacks host cells

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UC Irvine infectious disease researchers have uncovered components of the SARS coronavirus – which triggered a major outbreak of severe acute respiratory syndrome in 2002-03 – that allow it to take over host cells in order to replicate.

This insight is critical for a full understanding of any outbreaks caused by such viruses and may prove beneficial in the development of therapies not only for human coronavirus infections but for other pathogenic illnesses as well. Study results appear online in the July/August issue of *mBio*.

Megan Angelini, a graduate student in Professor Michael Buchmeier's laboratory in the Department of Molecular Biology & Biochemistry at UC Irvine, and colleagues found that three proteins in the SARS coronavirus – nsp3, nsp4 and nsp6 – have the ability to hijack a host cell's internal membranes and utilize them to make more virus.

"Understanding how the virus uses the host cell to reproduce itself could lead to potential therapies for these kinds of pathogens," said Buchmeier, who is also deputy director of the Pacific Southwest Regional Center of Excellence for Biodefense & Emerging Infectious Diseases at UC Irvine.

Additionally, he said, since membrane rearrangement is a tactic employed by all known positive-sense, single-stranded RNA viruses, including those responsible for polio and dengue fever, this work adds to that body of knowledge.

Although the majority of infections caused by coronaviruses in humans are relatively mild, the SARS outbreak of 2002 and 2003 and the emergence last fall of the Middle East respiratory syndrome coronavirus highlight their ability to potentially infect millions around the globe.

More information: mbio.asm.org/content/4/4/e00524-13

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