

Research milestone in CCHF virus could help identify new treatments

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New research into the Crimean-Congo hemorrhagic fever virus (CCHFV), a tick-borne virus which causes a severe hemorrhagic disease in humans similar to that caused by Ebolavirus, has identified new cellular factors essential for CCHFV infection. This discovery has the potential to lead to novel targets for therapeutic interventions against the pathogen.

The research, reported in a paper published today in the journal *PLoS Pathogens* and conducted by scientists at the Texas Biomedical Research Institute and their <u>colleagues</u>, represents a <u>milestone</u> in efforts to develop a treatment for CCHFV, which has a <u>fatality rate</u> approaching 30%.

"This new research is the first to indicate where the virus penetrates into the cell to infect it, revealing the site at which a drug therapy would need to act," said Robert Davey, Ph.D., of the Texas Biomed Department of Virology and Immunology, who led the research.

The virus is endemic to much of Eastern Europe, the Middle East, Asia and Africa, and recent studies have detected CCHFV in ticks collected in Spain, indicating that the virus continues to spread. CCHFV killed a US Army serviceman stationed in Afghanistan in 2009, and was initially mistaken for Ebolavirus.

CCHFV is primarily transmitted to people from ticks and from infected livestock during the slaughtering process, although human-to-<u>human</u> transmission can occur from close contact with blood or other fluids



from infected persons. There are no widely accepted therapies available to prevent or treat the disease.

Virus entry into the cell is the first and critical step in the virus replication cycle. To better understand the pathway for infection, researchers sought to identify cell proteins controlling CCHFV transport through the cell. Dr. Olena Shtanko, a postdoctoral scientist in the lab, demonstrated that after passing through early endosomes, membranebound vesicles within cells, the virus is delivered to multivesicular bodies which are made from large collections of these vesicles. Findings suggested that these multivesicular bodies are critical for infection by CCHFV, being the sites where the virus first penetrates into the cytoplasm to start replicating and taking over the cell.

"The next step in the process is to now identify drugs that can prevent interaction of the virus with the multivesicular bodies" Davey said.

Several new drug candidates are presently being tested by Shtanko with promising results.

Several other important viruses, like influenza virus (cause of the flu) and Lassa fever <u>virus</u> also use multivesicular bodies to infect cells. The identified drugs have the potential to be developed into broad spectrum antiviral treatments.

Provided by Texas Biomedical Research Institute

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