Researchers identify vulnerabilities of the deadly Ebola virus

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Disabling a protein in Ebola virus cells can stop the virus from replicating and infecting the host, according to researchers from the Icahn School of Medicine at Mount Sinai. The data are published in July in the journal *Cell Host and Microbe*.

Ebola viruses cause severe disease in humans because they can deactivate the innate immune system. Christopher Basler, PhD, Associate Professor of Microbiology at Mount Sinai and his team have studied how Ebola viruses evade the immune system, and discovered that a viral protein called VP35 is critical to deactivating the immune system. They found that when VP35 interacts with an important cellular protein called PACT, it blocks PACT from activating the immune system, allowing the virus to spread.

"Ebola viruses are extremely lethal, and are a great threat to human health as a bioweapon," said Dr. Basler. "Currently, there is no approved vaccine or treatment. Our findings will hopefully pave the way for future antiviral treatments."

With the help of collaborators at the University of Texas with access to special high containment facilities, Dr. Basler and his team infected healthy cells with Ebola virus cells that had mutated versions of VP35. The mutations disabled VP35's ability to interact with PACT, therefore allowing it to activate the immune system and prevent the virus from replicating. Next, the researchers overexpressed PACT in healthy cells, and infected them with Ebola virus cells. They found that overexpressing

PACT also inhibited viral replication.

Armed with this discovery, Dr. Basler and his team hope to develop drugs that disrupt the interaction of VP35 with PACT, or drugs that overexpress PACT.

Provided by The Mount Sinai Hospital