Scientists identify early impact of Ebola virus on immune system

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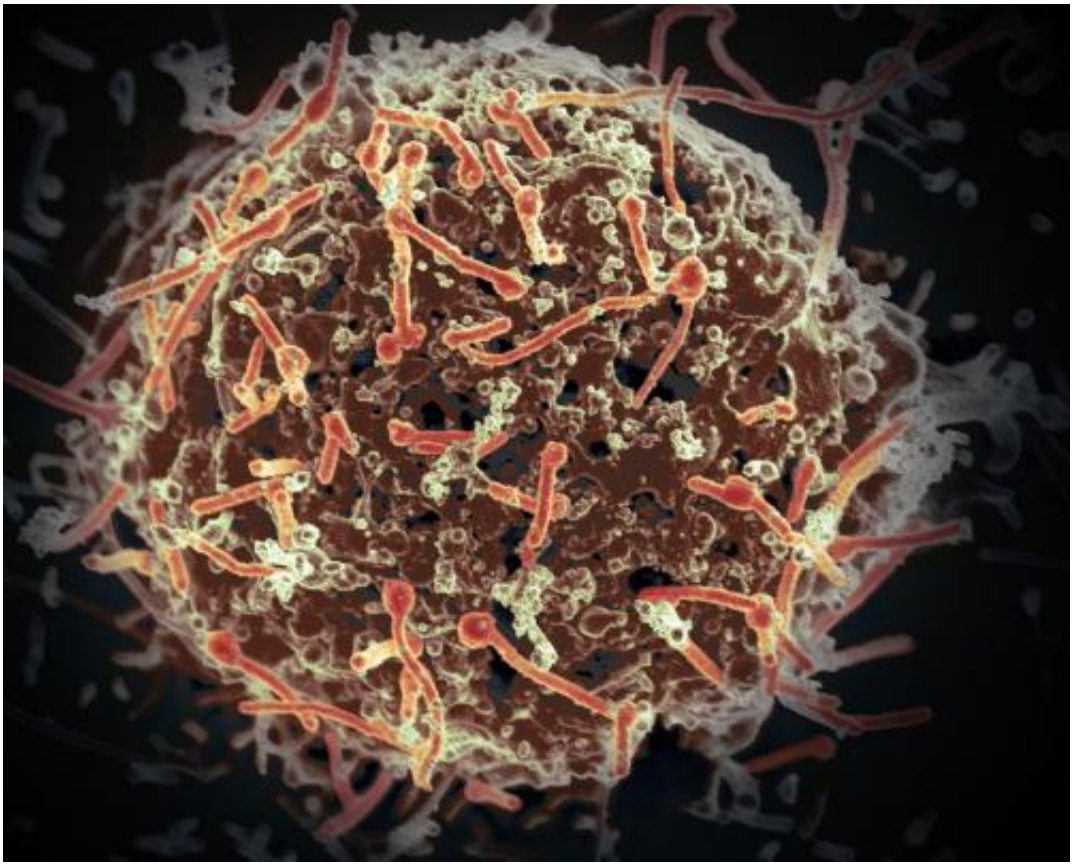

The Ebola virus, isolated in November 2014 from patient blood samples obtained in Mali. The virus was isolated on Vero cells in a BSL-4 suite at Rocky Mountain Laboratories. Credit: NIAID

A new mouse model of early Ebola virus (EBOV) infection has shown National Institutes of Health (NIH) scientists and colleagues how early

responses of the immune system can affect development of EBOV disease. The model could help identify protective immune responses as targets for developing human EBOV therapeutics.

Scientists from NIH's National Institute of Allergy and Infectious Diseases led the study with colleagues from the University of Washington and Columbia University.

The scientists analyzed signals that host cells use to alert the immune system to EBOV infection, and the immune system's responses. They focused on signaling events that begin within hours of a virus infection and involve the cellular mitochondrial antiviral signaling protein or MAVS.

The scientists already knew MAVS had a key anti-EBOV role, and in the current study examined it in an animal model for the first time. Many cell types produce MAVS, but MAVS produced by macrophages were found to be critical in controlling EBOV infection and to limiting the organ and tissue damage caused by EBOV.

In their experiments, macrophages coordinated the development of more advanced immune responses and the production of type I interferon, a compound with potent antiviral activity.

They also learned that EBOV could cause disease in mice by suppressing MAVS signaling and manipulating the interferon response.

With a goal of eventual drug development, the researchers are continuing their work to pinpoint the precise immune responses controlled through MAVS and to learn more about how EBOV sometimes delays immune signaling.

More information: M Dutta et al. A systems approach reveals MAVS

signaling in myeloid cells as critical for resistance to Ebola virus in murine models of infection. *Cell Reports*. DOI: 10.1016/j.celrep.2016.12.069