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Comparative virology: Risk Group-4 viruses elicit divergent immune responses in the human endothelium

Inhalt

Andes virus (ANDV), Ebola virus (EBOV), and Nipah virus (NiV) are Risk Group-4 viruses that cause fatal zoonotic spillover in South America, Africa, and Asia, respectively. All three viruses cause severe vascular diseases, although they belong to distinct virus families. A major question in comparative virology is if different viruses have similar effects on the same cell-types they collectively target.
To address this, we innovated new approaches to generate nearly pure populations of artery or vein endothelial cells (ECs) from human pluripotent stem cells (hPSCs), a model that offers multiple advantages: they are genetically normal and more physiologically relevant than cancer cell lines prevalently used in research. Infecting hPSC-derived artery and vein ECs with ANDV, EBOV, and NiV revealed that these viruses elicit starkly different host effects. ANDV strongly induced interferon secretion, whereas EBOV and NiV did not. Therefore, while Risk Group-4 viruses are often assumed to block interferon production, ANDV remains an exception. Curiously, while EBOV did not induce interferon production, it strongly upregulated inflammatory cytokine IL-6. Therefore, EBOV appears to induce "immunological misfiring", activating IL-6 while blocking interferon, thus decoupling these two arms of innate immunity.
This reveals starkly different effects of Risk Group-4 viruses on the human endothelium and highlight the utility of hPSC-derived vascular models to study high-risk pathogens.

Keywords

comparative virology, Andes virus, Ebola virus, Nipah virus, endothelial cells, human pluripotent stem cells

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