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Decoding Vascular Responses to Disparate Hemorrhagic Viruses: Andes and Ebola Viruses

Inhalt

Andes virus (ANDV) and Ebola virus (EBOV) are zoonotic pathogens that cause related severe vascular disease despite belonging to distinct virus families. So far, it is poorly understood whether unrelated viral hemorrhagic fevers elicit common or divergent host responses in the human vasculature.
To address this, we generated highly pure artery and vein endothelial cells (ECs) from human pluripotent stem cells, providing a physiologically relevant model of the human vasculature. Comparative infection studies revealed strikingly different host responses to ANDV versus EBOV. In artery ECs, ANDV induced robust antiviral and inflammatory programs, e.g. interferon-α/β, interferon-stimulated gene pathways, ISGylation, and cytokine storm signaling. Contrastingly, many of these pathways were suppressed during EBOV infection, indicating fundamentally different strategies of host immune modulation. A comparison of artery versus vein ECs showed mainly similar responses, with a few vessel-specific responses: COPII-mediated vesicle transport was increased in artery but reduced in vein following both infections, while EBOV selectively induced artery-specific activation of interleukin-1 family signaling and SUMOylation pathways.
These findings demonstrate that endothelial responses are both virus-specific and, to a lesser extent, vessel-type dependent. This highlights the utility of human stem cell-derived vascular models for understanding the mechanisms of viral vascular disease.

Keywords

Hemorrhagic Viruses, Andes virus, Ebola virus, Vascular Responses

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Yes, I am a Junior Scientist.

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