Differential transcriptional responses to Ebola and Marburg virus infection in bat and human cells

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Abstract

The unprecedented outbreak of Ebola in West Africa resulted in over 28,000 cases and 11,000 deaths, underlining the need for a better understanding of the biology of this highly pathogenic virus to develop specific counter strategies. Two filoviruses, the Ebola and Marburg viruses, result in a severe and often fatal infection in humans. However, bats are natural hosts and survive filovirus infections without obvious symptoms. The molecular basis of this striking difference in the response to filovirus infections is not well understood. We report a systematic overview of differentially expressed genes, activity motifs and pathways in human and bat cells infected with the Ebola and Marburg viruses, and we demonstrate that the replication of filoviruses is more rapid

in human cells than in bat cells. We also found that the most strongly regulated genes upon filovirus infection are chemokine ligands and transcription factors. We observed a strong induction of the JAK/STAT pathway, of several genes encoding inhibitors of MAP kinases (DUSP genes) and of PPP1R15A, which is involved in ER stress-induced cell death. We used comparative transcriptomics to provide a data resource that can be used to identify cellular responses that might allow bats to survive filovirus infections.

Involved units

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Leibniz-HKI-Authors

Jörg Linde

Details

Topics

Management of heterogeneous experimental data

Identifier

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