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Viral activation of the Raf/MEK/ERK kinase cascade promotes nuclear export of viral ribonucleoproteins (RNPs) by regulating matrix protein binding to the RNPs

Content

It was already shown that viral infection leads to the activation of a variety of signaling processes in the infected cells. Some of these activities are necessary for an efficient viral replication. The dependence of the Influenza A virus (IAV) on cellular signaling pathways leads to the opportunity of a novel antiviral strategy by targeting host factors that are essential for viral replication. It was already shown that viral infection induces the Raf/MEK/ERK kinase cascade for an efficient nuclear export of newly synthesized viral ribonucleoproteins (vRNP) and that this mechanism can be blocked with specific MEK-inhibitors. Such antiviral strategies are reducing the possibility of inducing viral resistance and enable a larger timeframe for a further antiviral treatment. However, the detailed mechanism how cellular kinases contribute to the nuclear export of the viral genome is still enigmatic. Here we shed first light on the mode of action by investigating the role of the Raf/MEK/ERK/RSK/MSK signaling pathway for the interaction of vRNPs with the viral M1 protein at the chromatin by using specific inhibitors against MEK (CI-1040), RSK (BI-D1870) and MSK (SB747651A) (Haasbach et al., (2017) Antiviral. Res. 2017 142:178-184).

Choose primary session

Virus host cell interaction

Choose secondary Session

Pathogenesis

Contribution Type : Oral presentation