VACCINIA VIRUS MODULATES DISTINCT MAPK SIGNALING PATHWAYS ACCORDING TO ITS TEMPORAL NEEDS DURING THE ENTIRE INFECTIVE CYCLE

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Manipulation of transduction pathways associated with key cellular functions such as survival, proliferation inflammation and apoptosis may be a fertile way to facilitate virus replication. Here we showed that Vaccinia virus (VACV) infection led to the simultaneous activation of distinct mitogen-activated protein kinase (MAPK) pathways, i.e. MKK4/7/JNK1/2 and MEK/ERK1/2 and the signals conveyed by both pathways converge to the transcriptional regulator c-JUN. Thus, while a 6- to 10-fold activation of MEK/ERK was verified upon infection from 3-12 hour post-infection (hpi), coincident with the time required for viral exponential growth and its requirement to activate c-JUN, a 10- to 15-fold activation of MKK4/JNK1/2 in turn was observed at late times of the virus infective cycle, i.e. 24-36 hpi, a time-frame associated with the viral egress of infected cells. It has long been known that signal duration and strength determines the biological outcome. Our data provided evidence that by modulating diverse MAPK signaling pathways VACV accomplishes proper viral temporal needs to biological responses.
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