

MEMBRANE RUPTURE AND REPAIR DURING VACCINIA VIRUS MEMBRANE BIOGENESIS

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The Poxviridae constitutes a family of viruses that includes small pox and several zoonotic viruses, such as monkeypox or cowpox. Members of this family are DNA viruses that replicate their double-stranded DNA directly into the cytoplasm of the host cells. Vaccinia Virus (VACV) is the best characterized poxvirus and is used in most studies as a model-poxvirus infection. VACV membrane acquisition follow an unconventional pathway that involve formation of open membrane intermediates derived from the ER of the host cell [1]. Membrane intermediates fuse together and build an open membrane sphere in the cytoplasm. Upon DNA uptake they form the closed immature particle (IV) that then mature to the fully infectious mature virion (MV) [2]. Molecular details of this unconventional membrane acquisition mechanism remain unknown. However, two classes of viral proteins have been identified as essential for VACV membrane assembly: the 3 structural proteins A14, A13 and A17, and 5 conserved viral membrane associated proteins (VMAPs) A11, A6, H7, L2 and A30.5. In order to characterize the role of the protein H7, we use EM immuno-labelling of thawed cryo-sections (Tokuyasu technique) of cells infected by a mutant virus in which H7 expression can be controlled. This method allow us to analyze the impact of the absence or mutation of H7 on the formation of new viral particles.

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