Structure and Assembly

108
Membrane anchorage of pUL31 is not sufficient for nuclear egress function

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Herpesviruses assemble capsids in the nucleus while further maturation takes place in the cytosol. Translocation of capsids through the nuclear envelope occurs by vesicle-mediated transport, i.e. budding of capsids at the inner nuclear membrane, acquiring a primary envelope which is lost after fusion with the outer nuclear membrane. This pathway is mediated by the nuclear egress complex consisting of two conserved viral proteins designated in the alphaherpesviruses as pUL34, a type II membrane protein which localizes in the nuclear envelope, and its nuclear complex partner pUL31. In the absence of either protein nuclear egress is greatly impaired while expression of both proteins is sufficient for vesicle formation at the inner nuclear membrane.
We could show that the N-terminal part of pUL34 comprising the pUL31 interaction domain is sufficient to mediate nuclear egress in the presence of a membrane anchor. In addition, data indicate that pUL31 is sufficient to induce membrane curvature when linked to membranes. We therefore hypothesized that the primary function of pUL34 might be the timely recruitment of pUL31 to the budding site to induce membrane bending requiring only membrane anchorage of pUL31 for vesicle formation. To test this we added different transmembrane regions of cellular inner nuclear membrane proteins, e.g. Emerin and lamina associated polypeptide (Lap)2ß to the C-terminus of pUL31 or constructed chimeric proteins consisting of full-length pUL31 fused to different parts of pUL34. However, while vesicular structures could be observed by indirect immunofluorescence indicating that membrane-anchored pUL31 alone is indeed sufficient for vesicle formation, none of the chimeric proteins supported nuclear egress.

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