



Double-Membrane Vesicle Function in the Life Cycle of Viruses

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ABOUT THE STUDY

For viruses that induce single-membrane invaginated vesicles or spherules, it is generally assumed that the viral replicas complexes dwell on the invaginated membrane, with RNA replication taking place in the vesicle lumen. A neck-like link to the cytoplasm enables the import of all needed metabolites, including nucleotides, as well as the export of newly generated RNA intended for translation or packing into a nucleocapsid. However, the situation remains unclear for viruses inducing DMVs because both membranes of these DMVs are frequently sealed, with no connection to the cytoplasm. For polio and CVB3, it has been suggested that viral replication occurs primarily on the cytoplasmic side of the single-membrane tubular vesicles, subsequently ceasing after these vesicles are wrapped in membranes to form sealed DMVs. For SARS-CoV and EAV, viral RNA is usually discovered in the lumen of DMVs, and it is unclear whether viral replication occurs within the DMVs or on the cytosolic side of these vesicles.

The role of autophagy in DMV biogenesis is that DMVs are normally uncommon in cells, but autophagy induces the formation of massive double-membrane structures called autophagosomes, which engulf cytoplasmic components targeted for degradation. Thus, autophagosomes have been discovered as a putative source of DMVs related to viral replication. The lipidation of the cytosolic microtubule associated light chain 3 protein with phosphatidylethanolamine, which results in the formation of a membrane-associated species known as LC3-II, is a critical event in the induction of autophagy. This connection of LC3-II with both the inner and outer membranes of the expanding autophagosome is required for autophagosome development. LC3 lipidation has been reported after poliovirus and CVB3 infection. Furthermore, cutting down the expression level of Atg5, a critical factor in autophagosome formation, has

been demonstrated to reduce viral replication. However, this knock-down appears to primarily affect the creation of progeny viruses, indicating a role for autophagy in virus assembly and release. Functional studies in the coronavirus model using knock-down of Atg5 expression have shown contradictory results.

CONCLUSION

The synthesis and significance of virus-induced DMVs in viral infection have been debated since the initial pioneering ultrastructure findings of virus-induced DMVs. Components of the cellular autophagy system or related pathways appear to play a role in the life cycle of DMV-inducing viruses, but the relationship between autophagy and DMV synthesis is unknown. The synthesis of viral proteins, either alone or in combination, in diverse viral models has resulted in the identification of proteins sufficient to promote the creation of DMVs. A single poliovirus protein has also recently been shown in a cell-free test to cause the creation of a double membrane liposome *via* membrane curvature, resulting in the invagination of a single-membrane liposome. To produce virus-induced DMVs, one or more viral proteins may replace or collaborate with host-cell proteins involved in autophagosome formation in infected cells. This could explain why, depending on the cell type, suppression of autophagy has different effects on viral replication. Once produced, DMVs could serve as a scaffold for the formation of viral replication complexes by providing a structure and environment conducive to viral replication. These DMVs may later be entirely sealed, preventing the viral RNA they contain from actively participating in the RNA amplification process. This would allow the virus to evade the host cell's antiviral response triggered by dsRNA.

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