

Autophagy: Chapter 8. Non-Lipidated LC3 is Essential for Mouse Hepatitis Virus Infection

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Coronaviruses (CoVs) are enveloped viruses responsible for severe respiratory diseases in birds and mammals. In infected cells they induce double-membrane vesicles (DMVs) and convoluted membranes (CMs), which are thought to be the site of virus replication. Until recently, both the origin of the CoVinduced vesicles and the exact localization of CoV replication remained unknown. It was assumed that the vesicles derive from the endoplasmic reticulum (ER). Nevertheless no conventional protein markers of the ER, ER-to-Golgi intermediate compartment (ERGIC), Golgi, or coatomer proteins could be detected in these structures. Recent data from our laboratory and others shed light on this mystery. It appears that the Mouse Hepatitis Virus (MHV), a prototype CoV, co-opts ERAD tuning vesicles as replication platforms. These vesicles are released from the ER, but do not contain conventional ER markers or coatomer proteins. Rather, they contain ERAD factors such as SEL1L, EDEM1, and OS-9 that are constitutively cleared from the folding compartment by so called ERAD tuning programs, and display non-lipidated LC3 (LC3-I) periferically associated at their limiting membrane. In MHV-infected cells, the ERAD tuning vesicle markers co-localize with viral non-structural proteins and double-stranded RNA, which are DMV markers. The unconventional role of LC3-I in the MHV infection cycle is further supported by the fact that Atg5 and Atg7, both essential proteins for LC3-I to LC3-II conversion and macroautophagy, are dispensable for CoV replication and DMV formation. These new insights into CoV replication might lead to new therapies to treat CoV infections. They also reveal a novel role for LC3, in its non-lipidated form, in both maintenance of cellular proteostasis and viral infection, the latter function supported by recent findings showing involvement of LC3-I in equine arteritis virus replication.

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