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The significance of the Endoplasmic Reticulum to Golgi trafficking in Hepatitis C virus Lifecycle

HCV relies on the host lipids and lipid pathways for its propagation. It is known to exists as hybrid of lipoprotein and virus particle termed lipoviral particle (LVP). Many viruses utilize the conventional ER to Golgi secretory route for their egress, however with respect to HCV there is disagreement with respect to the route embarked by HCV for its egress. In this study we attempted to characterize the role of the individual components of the ER-Golgi secretory compartment to decipher the role of distinct components of the secretory machinery in HCV lifecycle.

METHODS: Chemical and genetic inhibition of proteins that play critical role in the ER-Golgi secretory pathway.

RESULTS: Our observations suggest that HCV inhibits global protein secretion which may facilitate HCV secretion leads to accumulation of COPII vesicle components. Silencing of COPII inner and outer coat proteins inhibited HCV assembly and secretion whereas cargo adaptor SEC24A knockdown increases virus entry by increasing the LDLR levels by degrading its negative regulator PCSK9 and Inhibition of other SEC24 isoforms shows defect in HCV secretion. HCV remodels ERES and ERGIC compartments and also utilizes SEC16A through autophagy pathway for its replication and release whereas ERGIC-53 knockdown inhibited HCV virus entry and subsequent lifecycle.

CONCLUSIONS: HCV inhibits global protein secretion by utilizing the host secretory pathway and remodels ERES for its replication and egress whereas ERGIC-53 is playing a key role in the virus entry process.FLI-06 a chemical inhibitor that is known to disrupt ERES and Golgi apparatus inhibited virus replication and secretion.

http://doi.org/10.51219/URForum.2022.Kiran-Avula

ISBN: 978-1-8382915-5-6