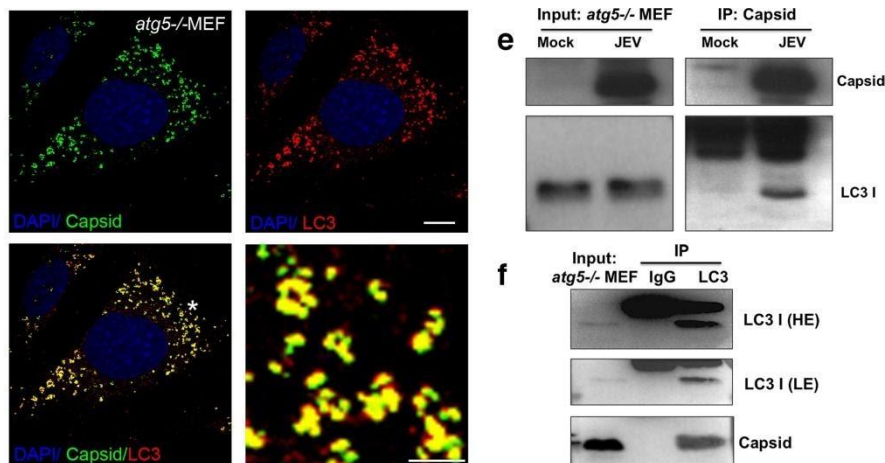


DBT-RCB study gains new insight on Japanese encephalitis virus

New Delhi, Dec 02: A team of researchers from the lab of Dr. Manjula Kalia of DBT-Regional Centre for Biotechnology (DBT-RCB) have found that Japanese encephalitis virus capsid protein interacts with non-lipidated MAP1LC3 on replication membranes and lipid droplets.



MAP1LC3 (Microtubule-associated protein 1 light chain 3) is a protein with a well-defined function in autophagy, but its role is still incompletely understood in several other autophagy-independent processes. Studies have shown that it is a host-dependency factor for the replication of several viruses.

Japanese encephalitis virus (JEV), a neurotropic flavivirus, replicates on ER-derived membranes that are marked by autophagosome-negative non-lipidated MAP1LC3 (LC3-I). Depletion of LC3 exerts a profound inhibition on virus replication and egress.

In the new study, the researchers further characterised the role of LC3 in JEV replication, and through immunofluorescence and immunoprecipitation showed that LC3-I interacts with the virus capsid protein in infected cells. This association was observed on capsid localized to both the replication complex and lipid droplets (LDs). JEV infection decreased the number of LDs per cell indicating a link between lipid metabolism and virus replication.

The capsid-LC3 interaction was independent of the autophagy adaptor protein p62/Sequestosome 1 (SQSTM1). Further, no association of capsid was seen with the Gamma-aminobutyric acid receptor-associated protein family, suggesting that this interaction was specific for LC3. High-resolution protein-protein docking studies identified a putative LC3-

interacting region in capsid, ⁵⁶FTAL₅₉, and other key residues that could mediate a direct interaction between the two proteins.

The link to the research paper: <https://pubmed.ncbi.nlm.nih.gov/33095129/>

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