New Delhi, Nov 20: Multiple membrane trafficking networks operate in the eukaryotic cell and are hijacked by viruses to establish infection. Recent studies have highlighted that viruses can exploit distinct pathways depending on the cell type. Japanese encephalitis virus (JEV), a neurotropic flavivirus, can infect neuronal cells through a clathrin-independent endocytic mechanism.

A team of researchers at Regional Centre for Biotechnology (DBT-RCB) performed an RNA interference-based study targeting 136 proteins in the human cell line IMR-32 to characterize the membrane trafficking requirements for JEV infection of neuronal cells. Through quantitative RT-PCR and plaque assays it was validated that the JEV infection in neuronal cells was independent of clathrin, and host-factors that were crucial for establishment of infection were further identified. Several of these proteins were involved in regulation of actin filament organization such as RHOA, RAC1, proteins of the ARP2/3 complex and N-WASP family, LIMK1, PAK1 and ROCK2.

The small molecule inhibitors of ARP2/3 complex, CK-548 and of the N-WASP, Wiskostatin inhibited virus replication highlighting the important roles of these proteins in the virus lifecycle. The authors also identified ATG12, BECN1, VAPA, VAPB and VCP proteins as crucial host-factors for JEV replication across epithelial and neuronal cell lineages.
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