Abstract ID# 770

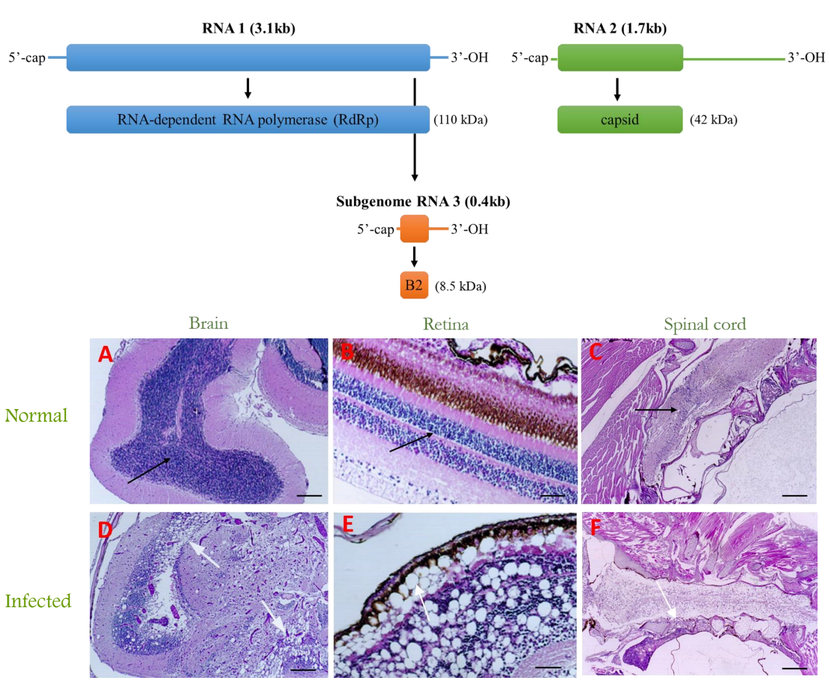
NNV recruits phospo-eIF4E and initiates cap-dependent translation in remodeled microtubule-organizing center

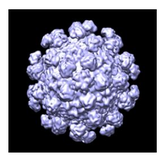
Vertika Bajpai[1,2]*, Chen-Hung Li[2] and Chi-Yao Chang[1,2]

[1]Institute of Fisheries Science, National Taiwan University, Taipei, Taiwan
[2]Institute of Cellular and Organismic Biology, Academia Sinica, Taipei, Taiwan

Introduction

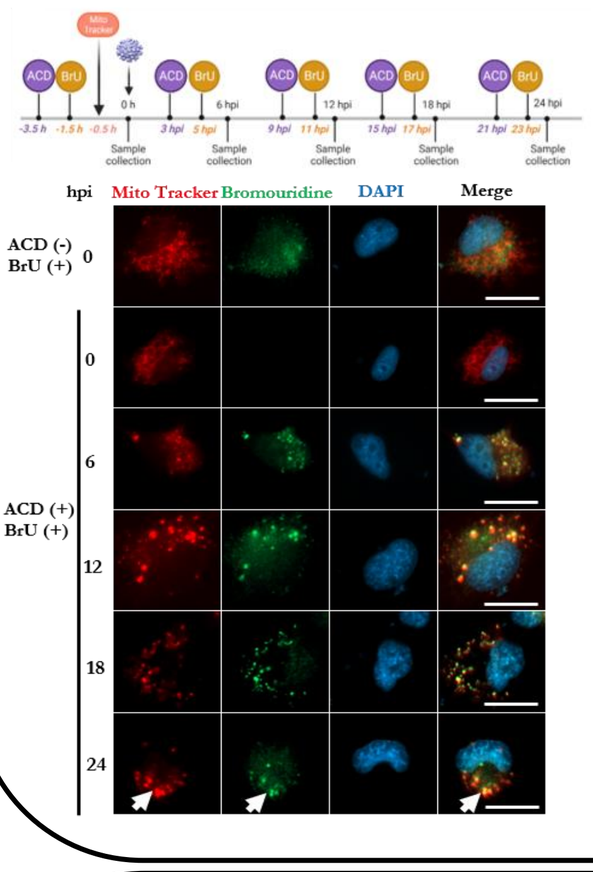
Nervous Necrosis Virus belongs to genus *Betanodavirus*, family *Nodaviridae.*

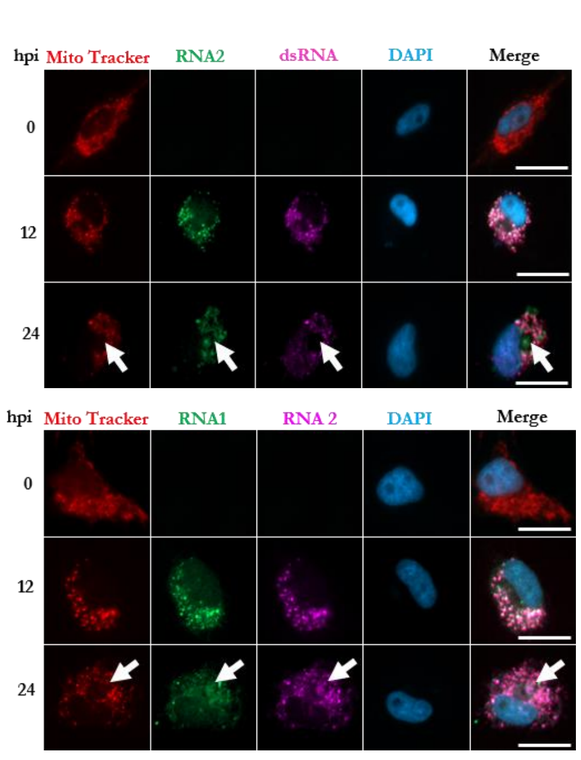




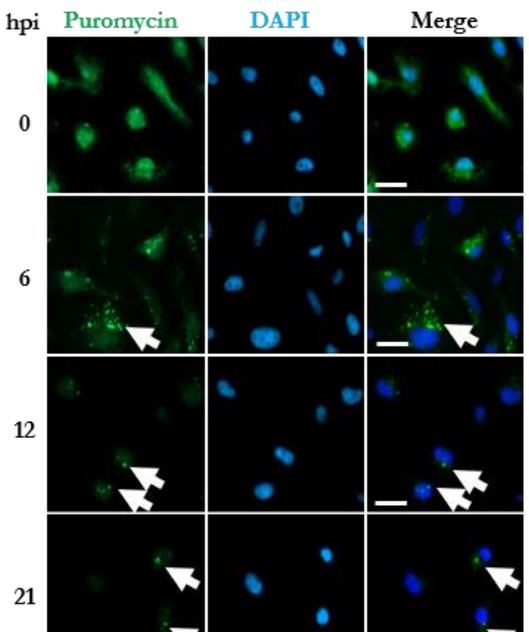
Results

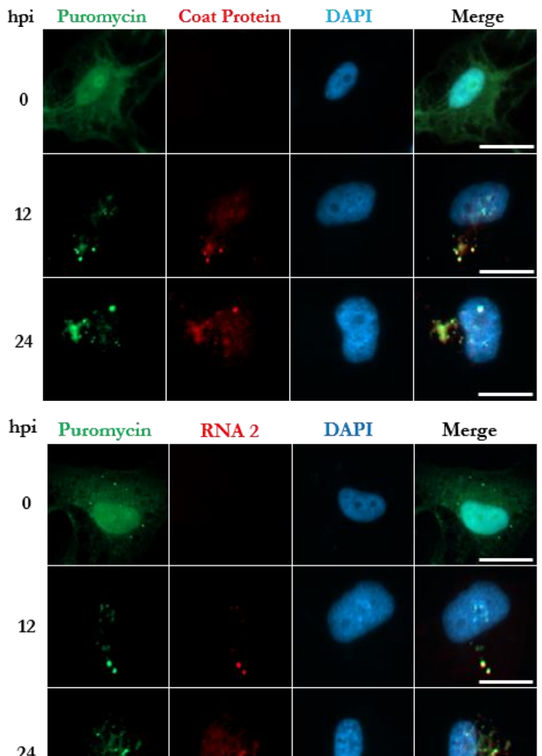
1. NNV replication and transcription occurs in mitochondria



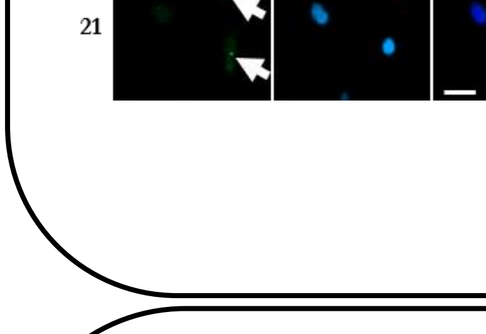


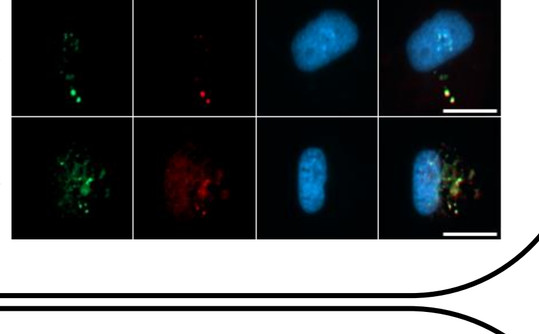
2. Neo-synthesized proteins correlate with NNV RNA2 and coat protein



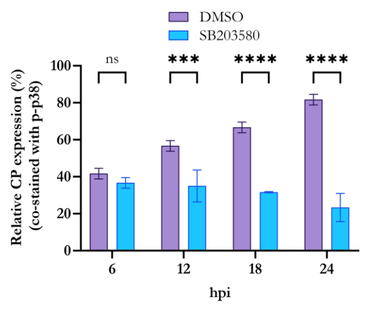


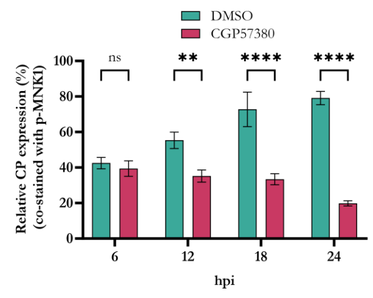
3. Viral translation is initiated by binding of p-eIF4E to NNV viral RNAs

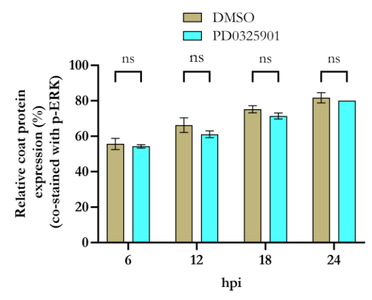




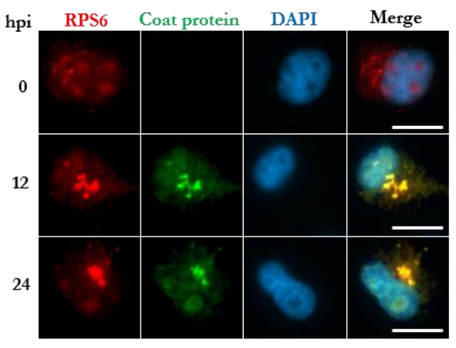
4. NNV activates upstream kinases p38 MAPK and MNK1, both of which are essential for viral propagation

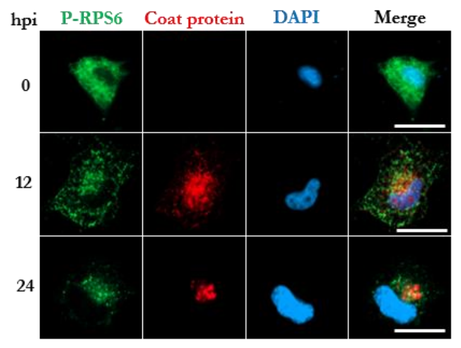


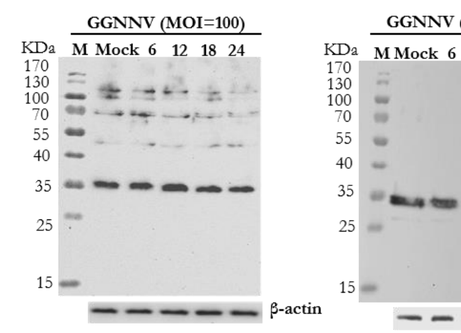


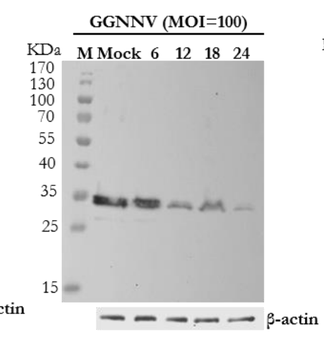


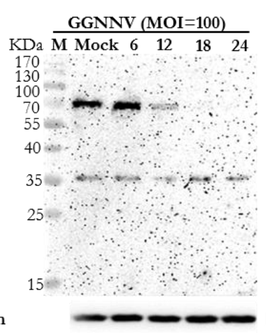
5. RPS6 but not p-RPS6 is important for NNV coat protein synthesis

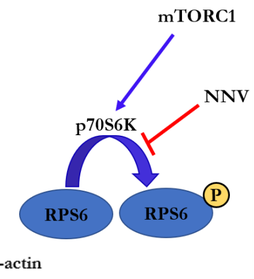


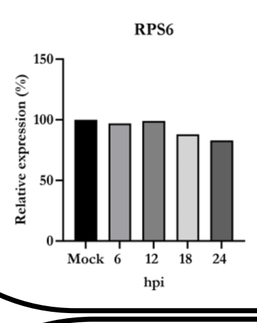


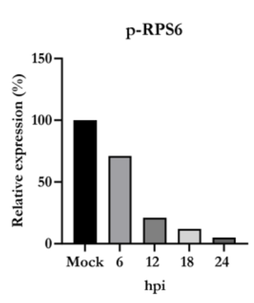


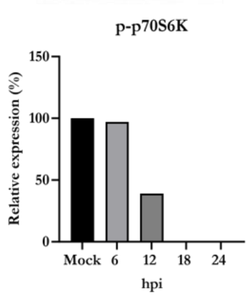




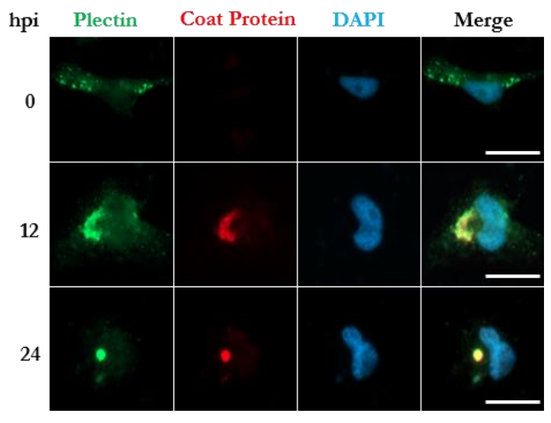


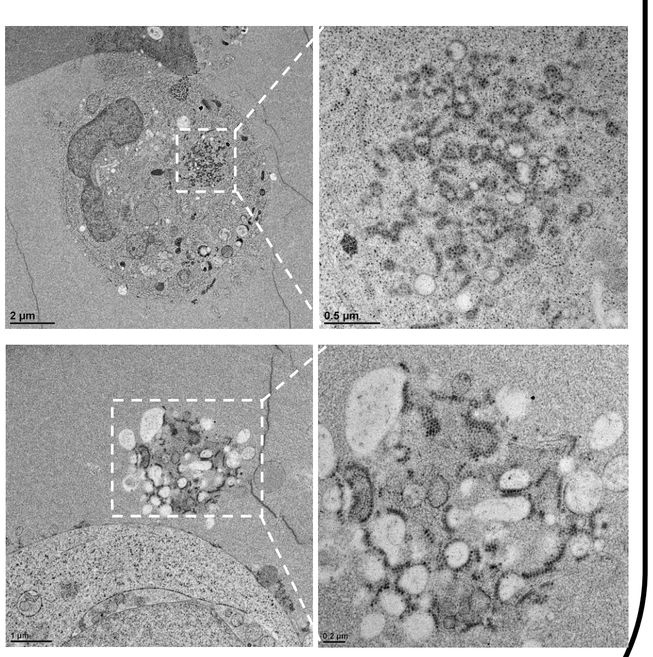






6. Cytoskeletal proteins and trans-Golgi support architecture of NNV factories





Proposed model

In conclusion, NNV hijacks cap-dependent translation for translation of its proteins and activates upstream kinase p38 MAPK, MNK1 for its propagation.

Future works include

- Immunogold labelling using trans-Golgi antibody to confirm remodeling of MTOC is Golgi derived.
- Understanding how NNV RNAs are transported from site of replication (mitochondria) to MTOC.