

Supporting Information for

“Lyssavirus P protein isoforms diverge significantly in subcellular interactions underlying mechanisms of interferon antagonism”

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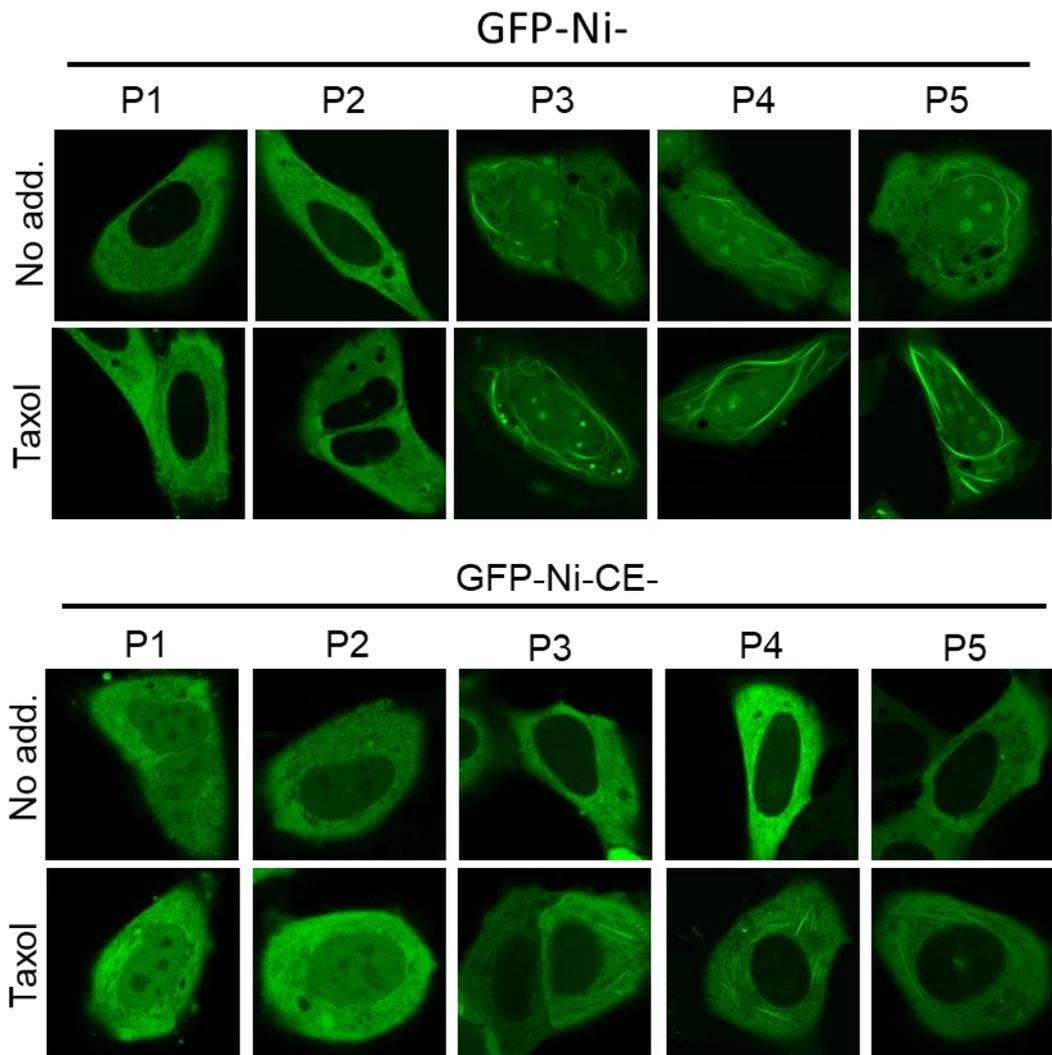


Figure S1. Live cell CLSM images of HeLa cells expressing the indicated GFP-fused P protein isoforms, of Nishigahara (upper panel) and Ni-CE (lower panel). Images are representative of cells from ≥ 5 fields of view.

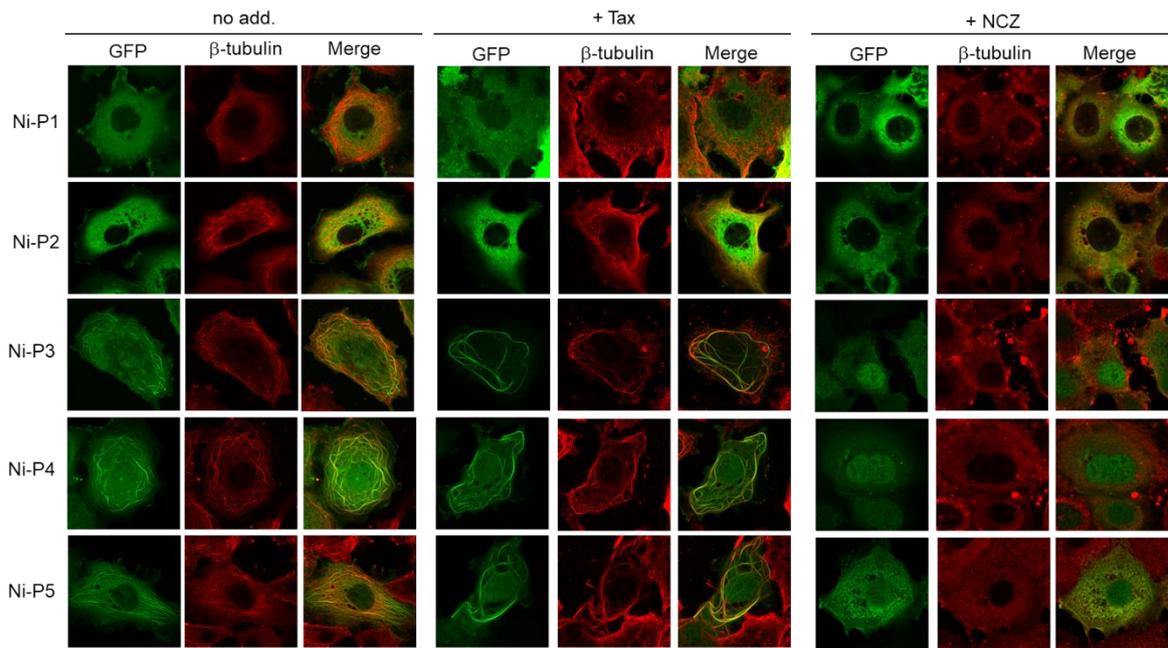


Figure S2. CLSM images of COS-7 cells expressing the indicated GFP-fused Ni-P isoforms. Cells were fixed and immunostained for β -tubulin following treatment without (left panels) or with Taxol (center panels) or Nocodazole (right panels). Images are representative of cells from ≥ 4 fields of view.

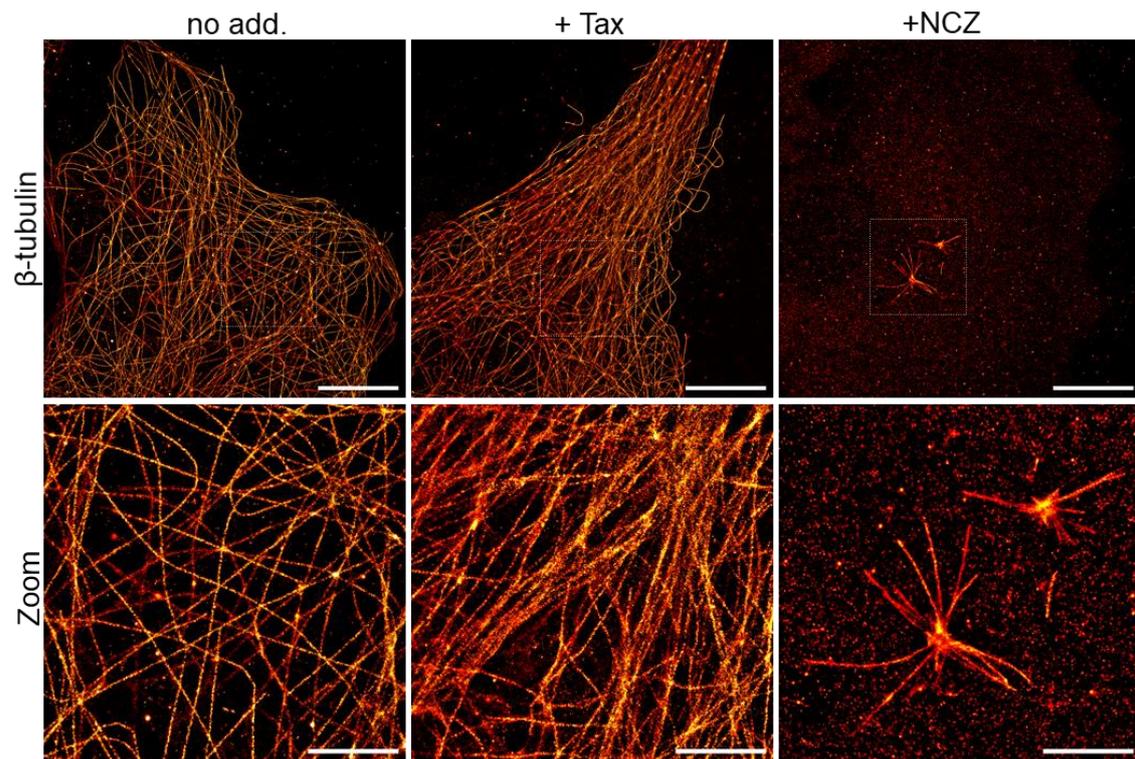


Figure S3. Super-resolution imaging of immunolabelled MTs in fixed COS-7 cells treated with Taxol or Nocodazole. dSTORM images indicate expected stimulation of microtubule filament assembly by Taxol and disassembly by Nocodazole (resulting in loss of networks except for MT-organizing centres). Scale bars = 10 μm (top) and 3 μm (bottom).

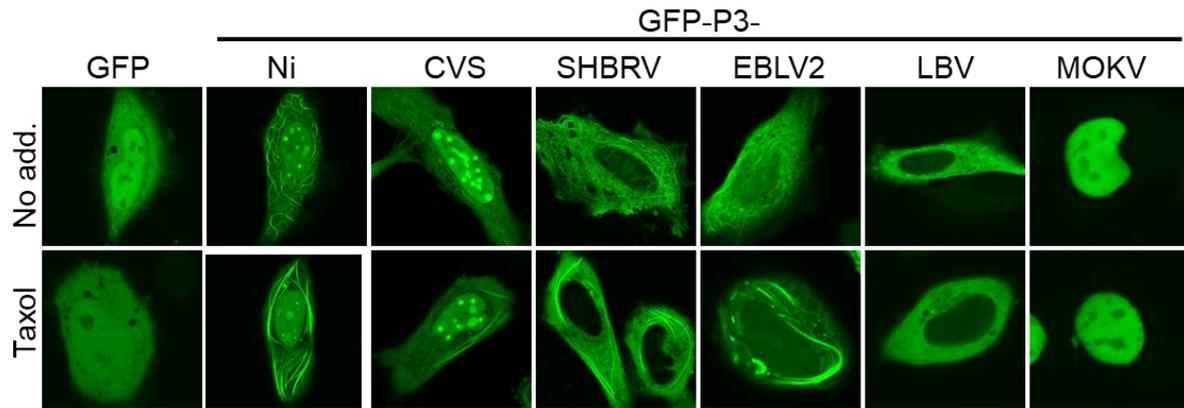


Figure S4. Live cell CLSM images of HeLa cells expressing GFP-fused P3 of the indicated lyssaviruses, following treatment with or without Taxol. Images are representative of cells from ≥ 5 fields of view (except GFP, 3 fields of view).

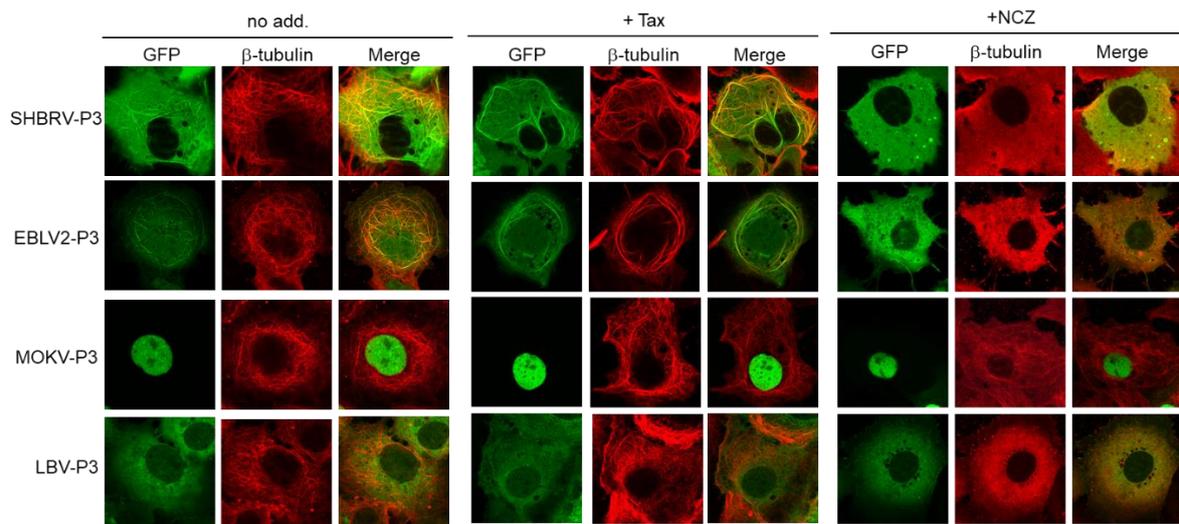


Figure S5. CLSM images of COS-7 cells expressing the indicated GFP-fused lyssavirus P3 proteins. Cells were fixed and immunostained for β -tubulin following treatment without (left panel) or with Taxol (centre panel) or Nocodazole (right panel). Images are representative of cells from ≥ 3 fields of view.

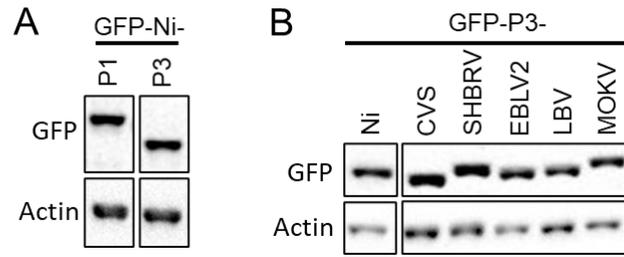


Figure S6. Western blot analysis of lysates of COS-7 cells using the indicated antibodies. Irrelevant intervening lanes have been removed.

