

Fig. S3 VSV Δ G-G and VSV Δ G-PIV5 differ in their dependence on dynamin and clathrin for entry. C10 (A and C) and CHO-HVEM (B and D) cells were pretreated with dynamin inhibitors Dynasore (80 µM), Dyngo-4a (25 µM), MiTMAB (5 µM), or the CME inhibitor Pitstop-2 (30 µM) and infected with VSV Δ G-G or VSV Δ G-PIV5 at a MOI of 1. Infectivity was quantitated by flow cytometry at 6 hours post infection. CHO-HVEM cells treated with Dyngo-4a or MiTMAB used the same DMSO control as indicated by the same bar graph appearing twice each in panels C and D. Significance was calculated using a two-tailed Student's T-test with Welch's correction (p < 0.05 = *; p < 0.01 = **; p < 0.001 = ***). E and F) C10 and CHO-HVEM cells were pretreated with dynamin inhibitors Dynasore, Dyngo-4a, MiTMAB, or Pitstop-2 at the same concentrations as in panels A-D and then incubated with 50 µg/ml of AF488-labeled transferrin. Cells were fixed, counterstained with DAPI, and imaged by confocal microscopy. Scale bar = 25 µm.