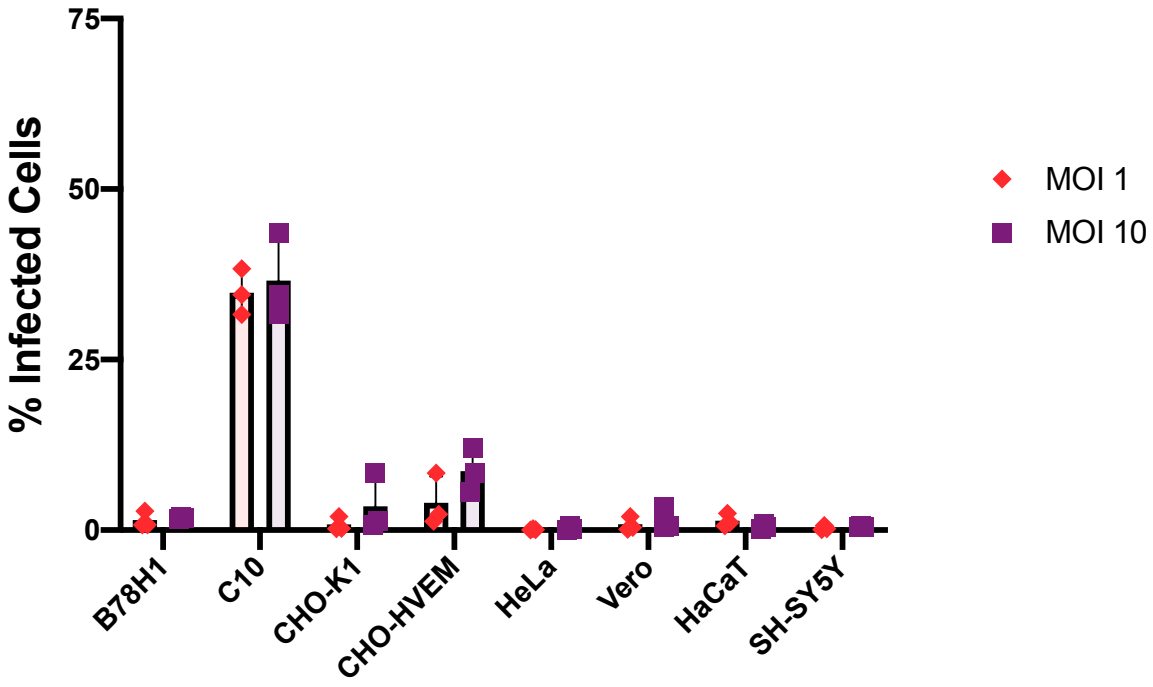
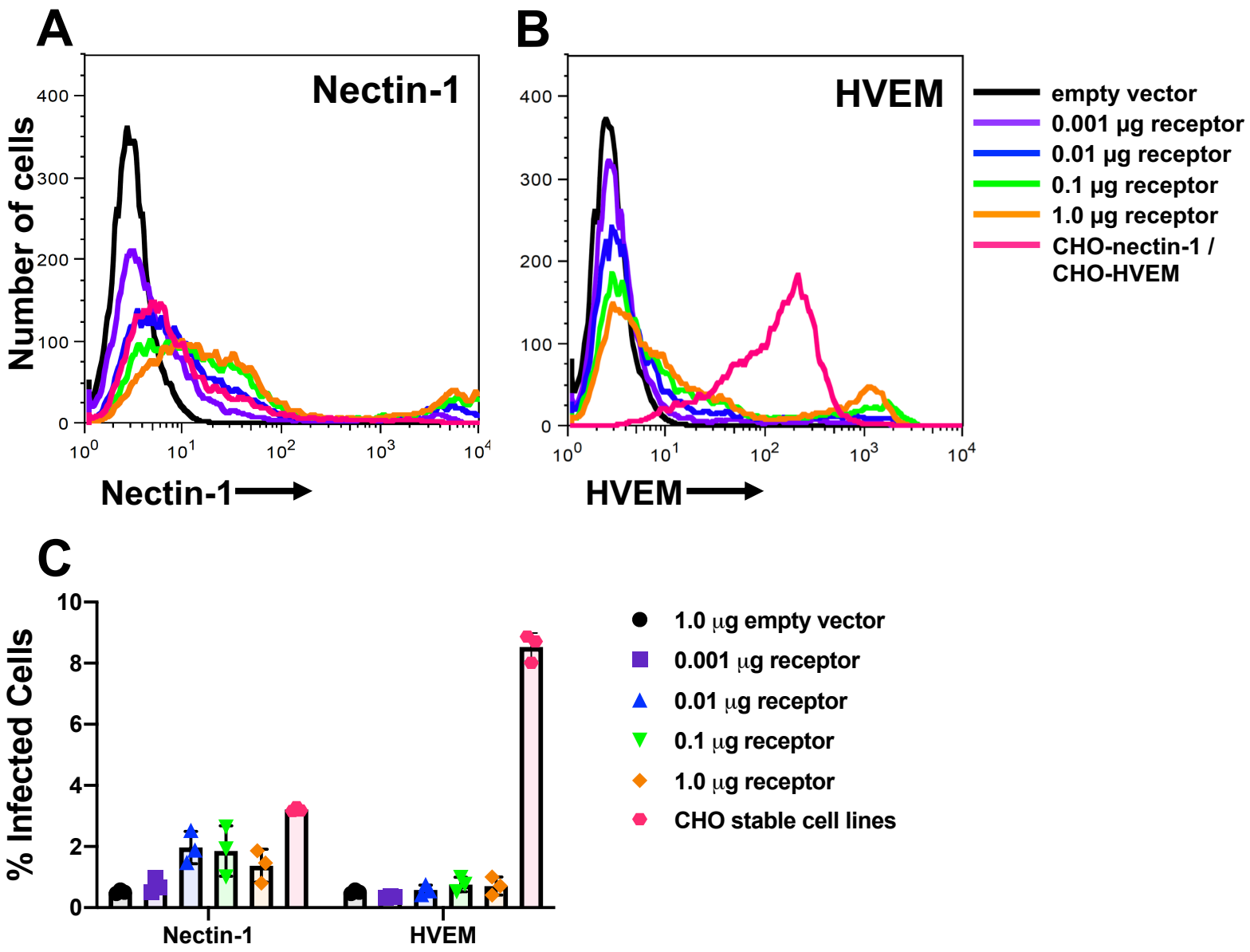
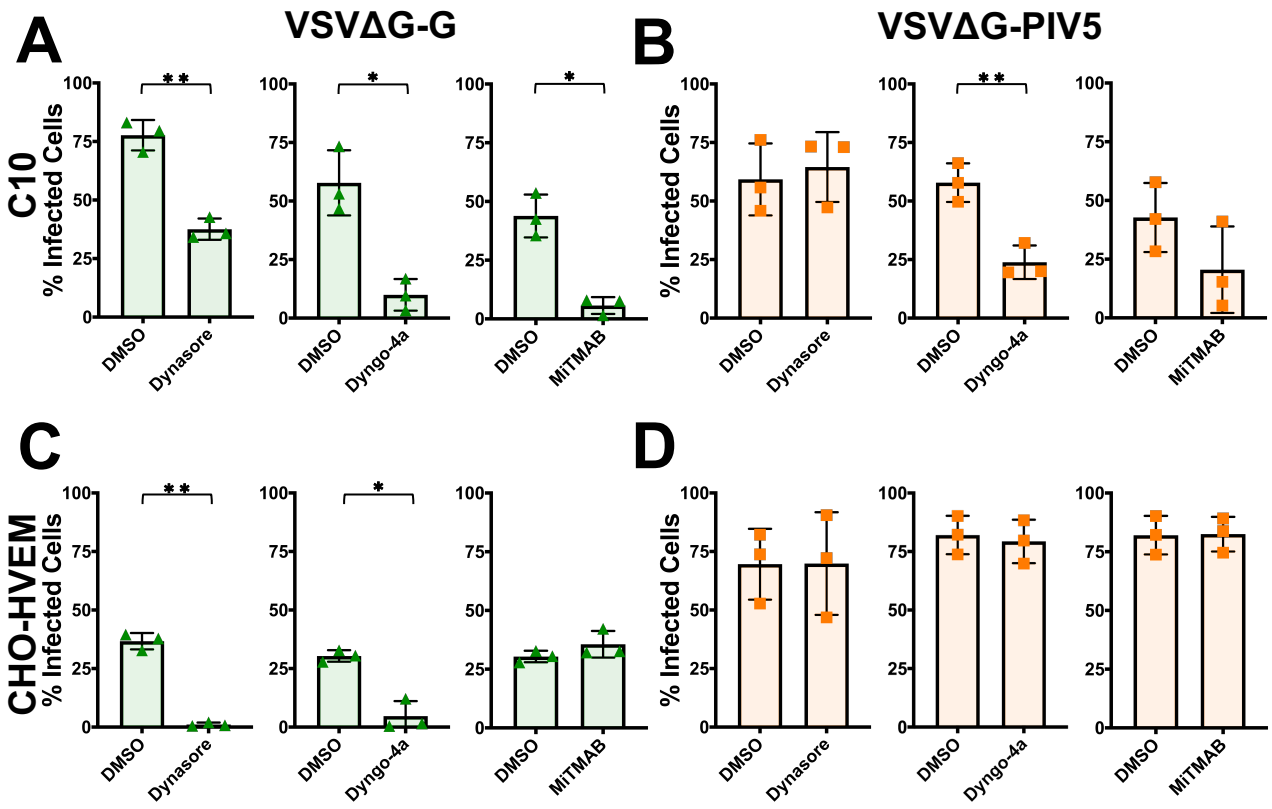


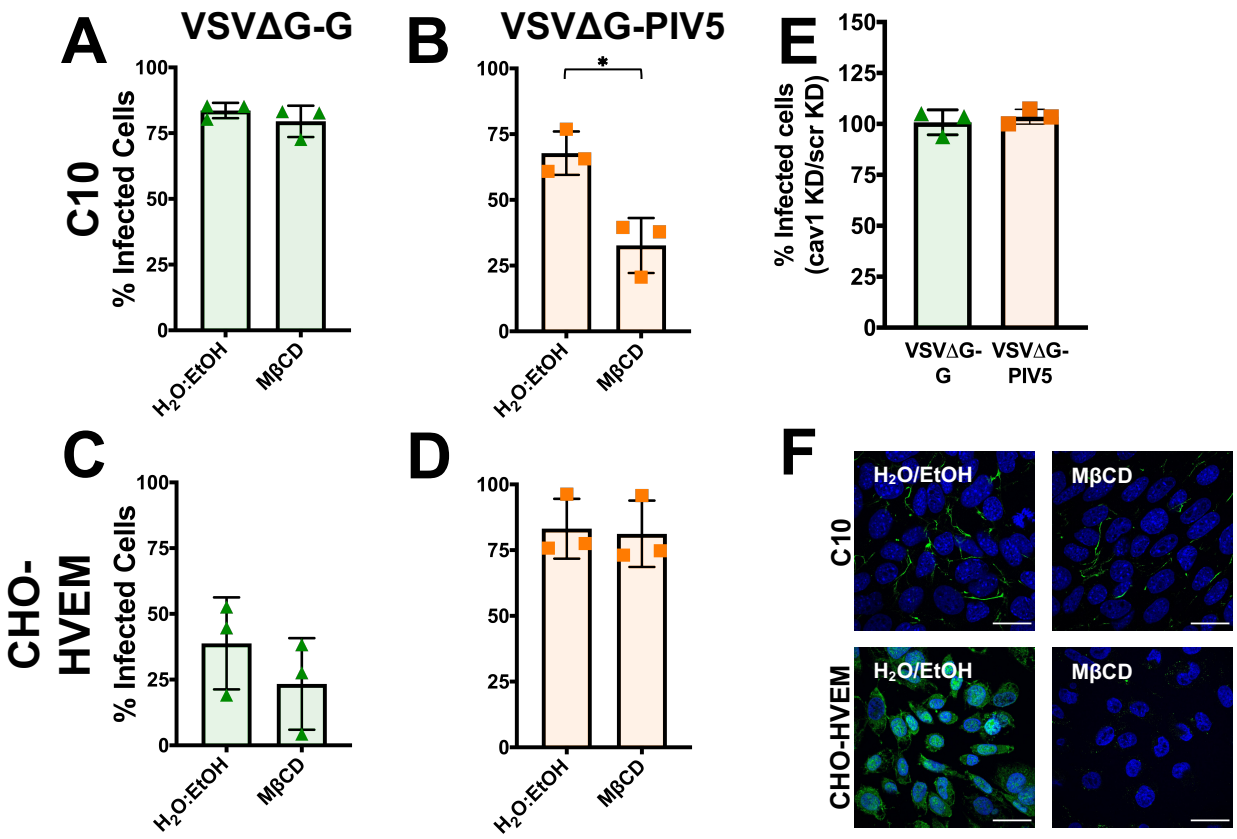
S1 Fig. Infecting cells at with VSVΔG-BHLD at a higher MOI does not increase entry to an appreciable extent. Receptor null (B78H1 and CHO-K1) and receptor bearing cells (C10, CHO-HVEM, HeLa, Vero, HaCaT, and SH-SY5Y) were infected at MOI =1 (red) or MOI = 10 (purple). Entry efficiency was assessed by flow cytometry at 6 hours post infection.



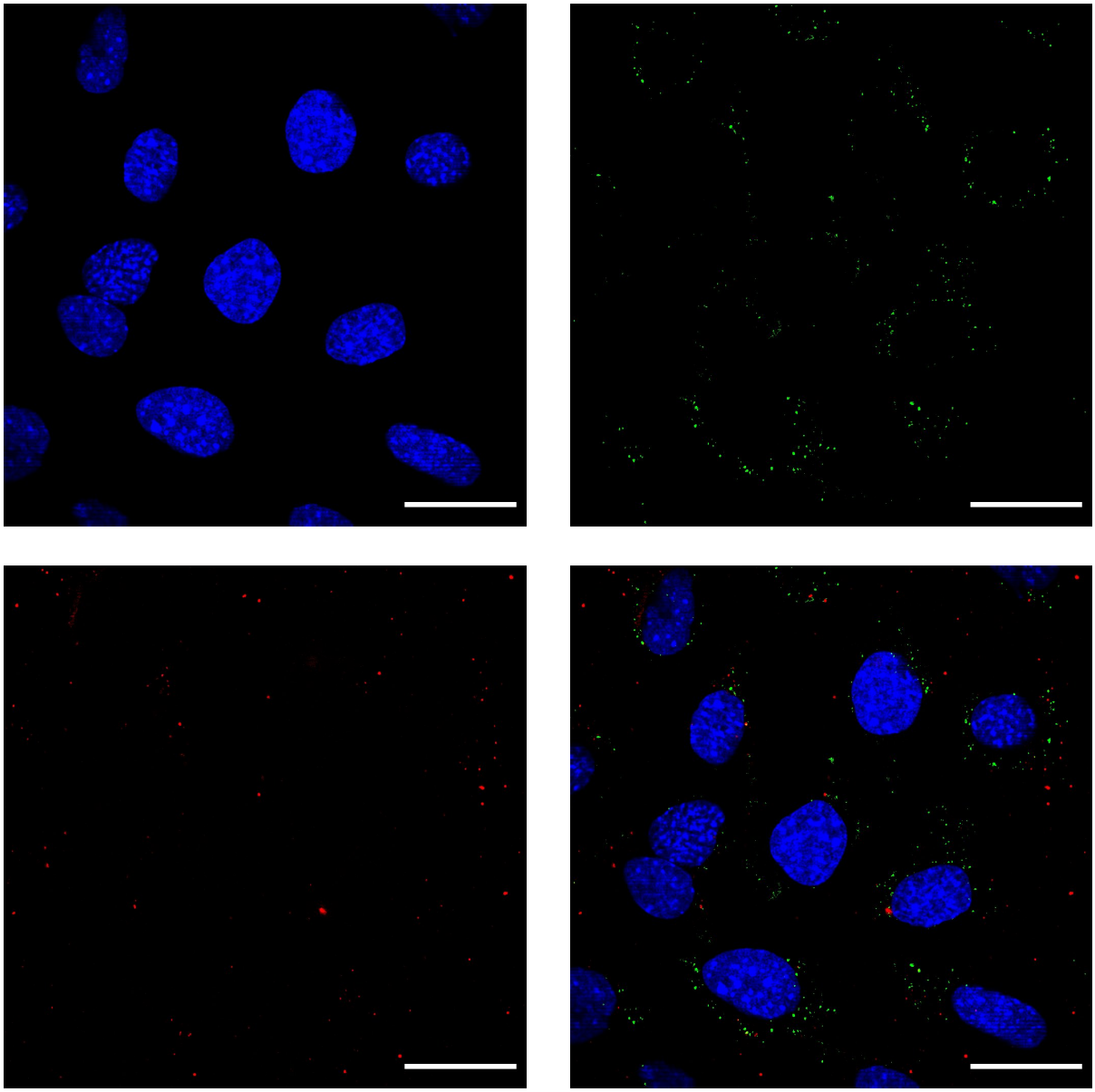
S2 Fig. Increased amounts of surface nectin-1 or HVEM do not increase VSVΔG-BHLD entry. CHO-K1 (receptor-null) cells were transfected with increasing amounts of plasmids encoding nectin-1 (pBG38) (A) or HVEM (pSC386) (B). Surface expression was analyzed by flow cytometry 24 hours post transfection. C) Cells transfected with nectin-1 or HVEM were infected with VSVΔG-BHLD at MOI = 1. Entry efficiency was assessed at 6 hours post infection by flow cytometry. In each panel, receptor-bearing stable cell line data (CHO-nectin-1 and CHO-HVEM) were inserted as points of comparison.



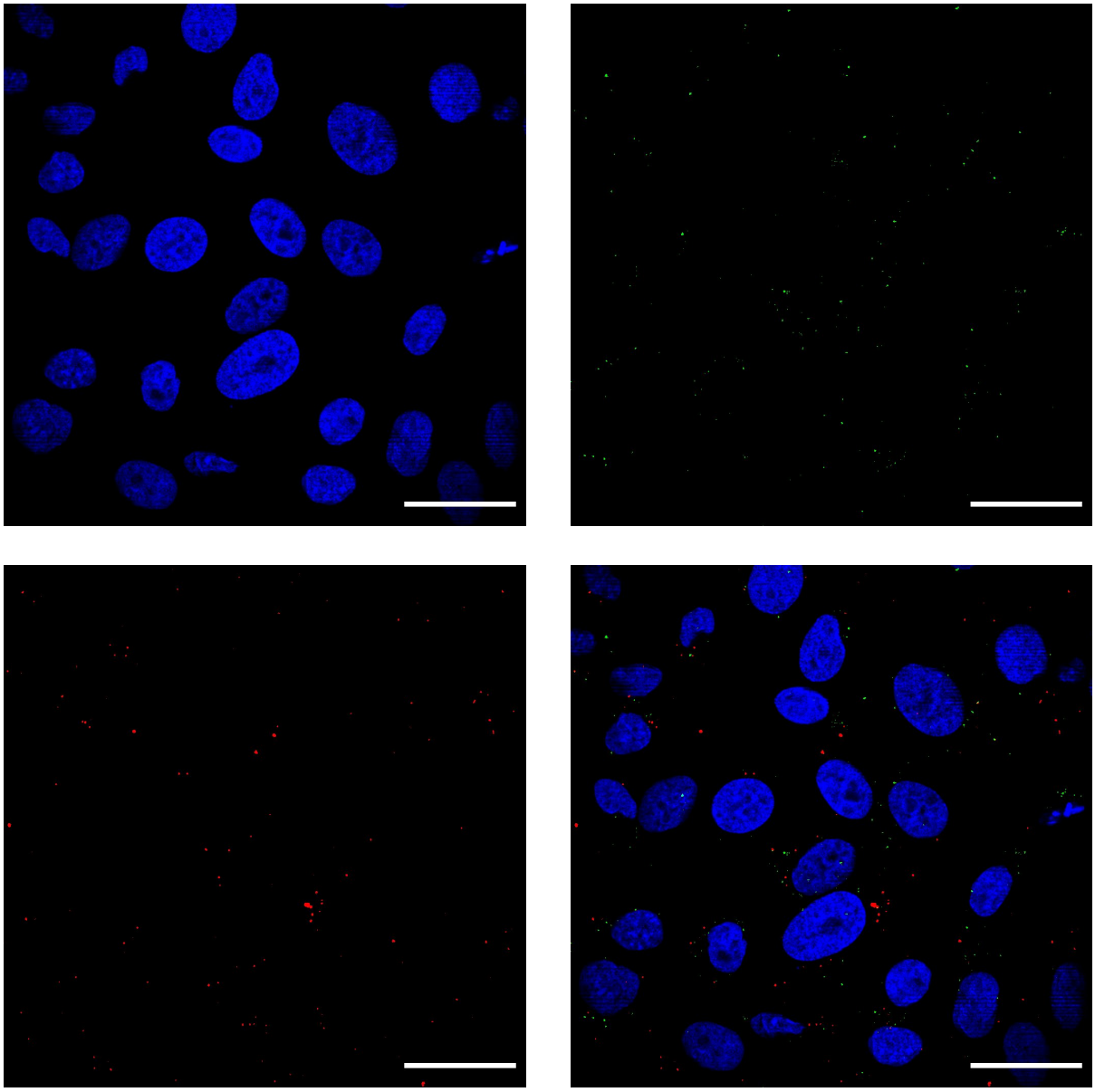
S3 Fig. VSVΔG-G and VSVΔG-PIV5 differ in their dependence on dynamin for entry. C10 (A and B) and CHO-HVEM (C and D) cells were pretreated with dynamin inhibitors Dynasore (80 μM), Dyngo-4a (25 μM), or MiTMAB (5 μM) and infected with VSVΔG-G or VSVΔG-PIV5 at a MOI of 1. Infectivity was quantitated by 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 ($p < 0.05$ = *; $p < 0.01$ = **; $p < 0.001$ = ***).



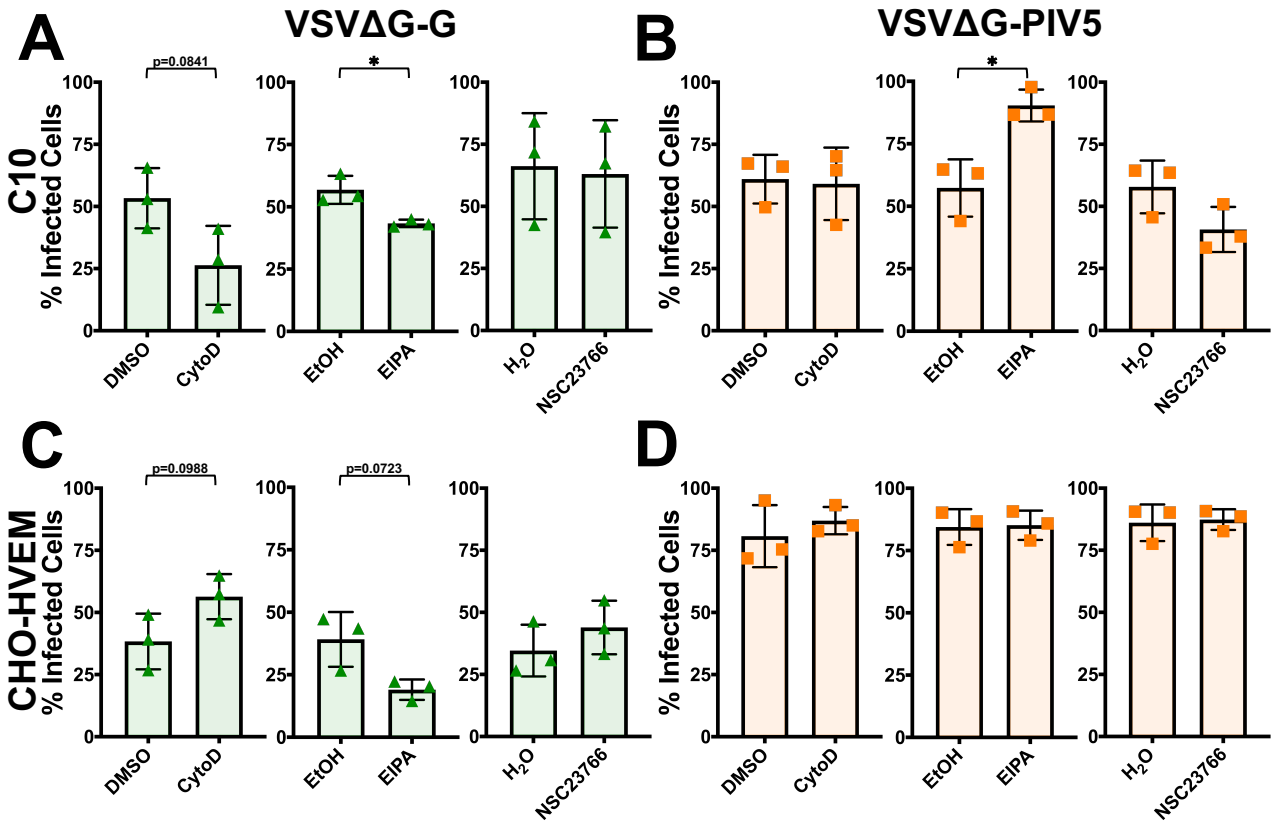
S4 Fig. VSVΔG-G entry does not require cholesterol whereas VSVΔG-PIV5 entry requires cholesterol in a cell-type-dependent manner. C10 (A and B) and CHO-HVEM (C and D) cells were pretreated with a cholesterol-removal drug methyl-β-cyclodextran, MβCD (5 mM) and infected with VSVΔG-G or VSVΔG-PIV5 at a MOI of 1. Infectivity was quantitated by flow cytometry at 6 hours post infection. (E) CHO-HVEM cells were transfected with a caveolin-1 siRNA (cav-1) or a scrambled control siRNA (scr) (both 50 pm) and infected with VSVΔG-G or VSVΔG-PIV5 at a MOI of 1. Infectivity was quantitated by flow cytometry at 6 hours post infection. Significance was calculated using a two-tailed Student's T-test with Welch's correction ($p < 0.05$ = *; $p < 0.01$ = **; $p < 0.001$ = ***). F) C10 and CHO-HVEM cells were treated with either a solvent control (H_2O/EtOH) or methyl-β-cyclodextrin (MβCD), then incubated with cholera toxin subunit B labelled with Alexa Fluor 488. Confocal microscopy was performed on the solvent control and methyl-β-cyclodextrin treated cells. Cells were fixed, counterstained with DAPI, and imaged by confocal microscopy. Scale bar = 25 μm.



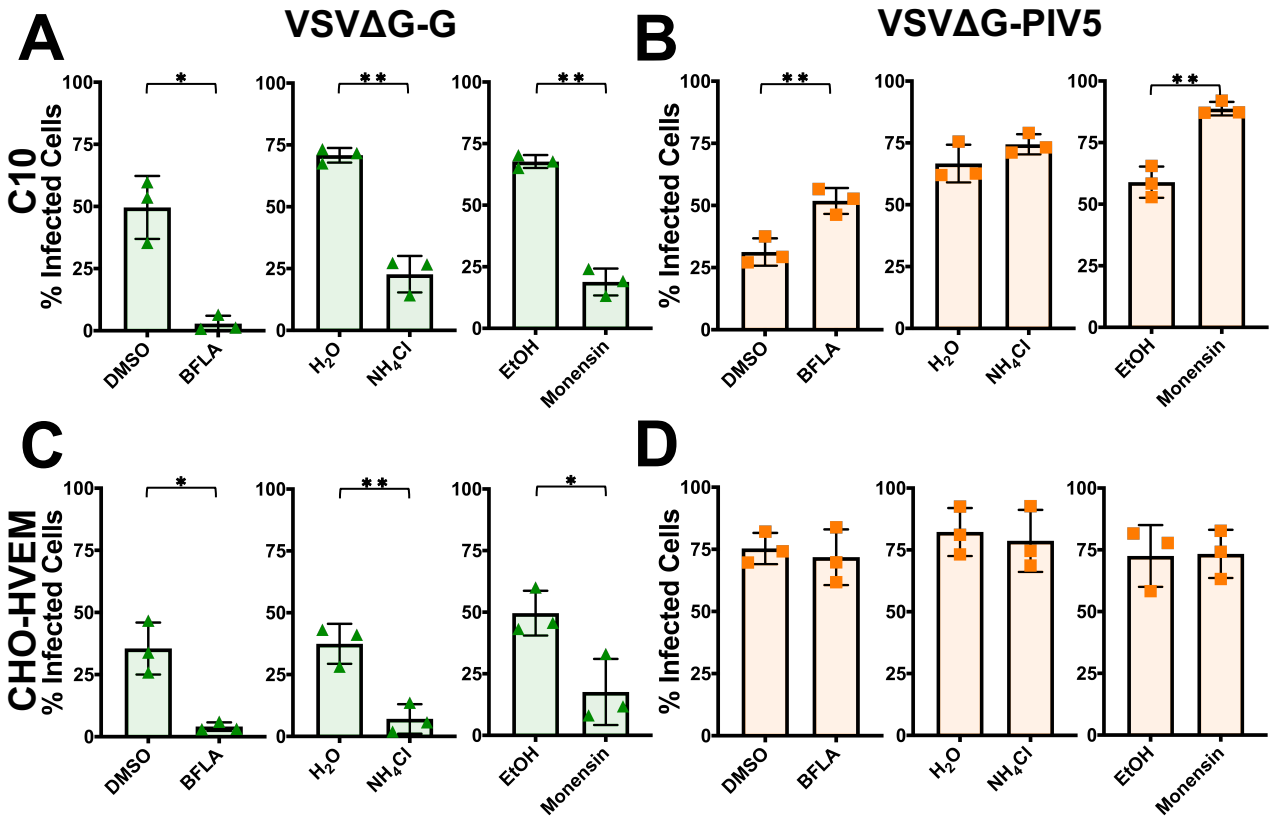
S5 Fig. VSVΔG-BHLD does not co-localize with the fluid-phase marker 70 kDa dextran in C10 cells. C10 cells were incubated with 1 mg/ml of rhodamine-B labelled 70 kDa dextran and VSVΔG-BHLD (MOI = 1) for one hour at 4˚C. Cells were then shifted to 37 ˚C for 20 minutes. Cells were fixed, counterstained with DAPI, and imaged by confocal microscopy. gB was detected by immunofluorescence using the rabbit pAb R68 and anti-rabbit IgG conjugated to FITC. Green = gB (marker for VSVΔG-BHLD particles); Red = 70 kDa dextran. Scale bar = 25 μm.



S6 Fig. VSVΔG-BHLD, in large, does not co-localized with the fluid-phase marker, 70 kDa dextran, in CHO-HVEM cells. CHO-HVEM cells were incubated with 1 mg/ml of rhodamine-B labelled 70 kDa dextran and VSVΔG-BHLD (MOI = 1) for one hour at 4˚C. Cells were then shifted to 37˚C for 20 minutes. Cells were fixed, counterstained with DAPI, and imaged by confocal microscopy. gB was detected by immunofluorescence using the rabbit pAb R68 and anti-rabbit IgG conjugated to FITC. Green = gB (marker for VSVΔG-BHLD particles); Red = 70 kDa dextran. Scale bar = 25 μm.



S7 Fig. VSVΔG-G and VSVΔG-PIV5 entry does not require macropinocytosis. C10 (A and B) and CHO-HVEM (C and D) cells were pretreated with macropinocytosis inhibitors cytochalasin D (2 μM), EIPA (25 μM), or NSC23766 (200 μM) and infected with VSVΔG-G or VSVΔG-PIV5 at a MOI of 1. Infectivity was quantitated by flow cytometry at 6 hours post infection. Significance was calculated using a two-tailed Student's T-test with Welch's correction ($p < 0.05$ = *; $p < 0.01$ = **; $p < 0.001$ = ***).



S8 Fig. VSVΔG-G but not VSVΔG-PIV5 entry requires endosomal acidification. C10 (A and B) and CHO-HVEM (C and D) cells were pretreated with inhibitors of endosomal acidification BFLA (100 nM), NH_4Cl (50 mM), or monensin (15 μM) and infected with VSVΔG-G or VSVΔG-PIV5 at MOI = 1. Infectivity was quantitated by flow cytometry at 6 hours post infection. Significance was calculated using a two-tailed Student's T-test with Welch's correction ($p < 0.05$ = *; $p < 0.01$ = **; $p < 0.001$ = ***).