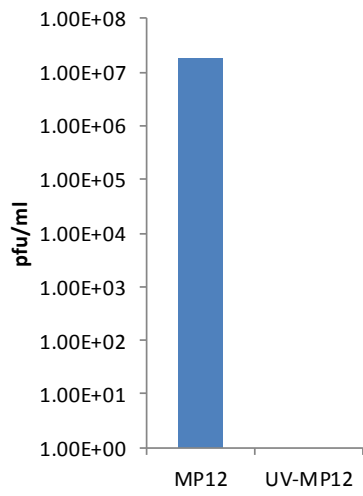
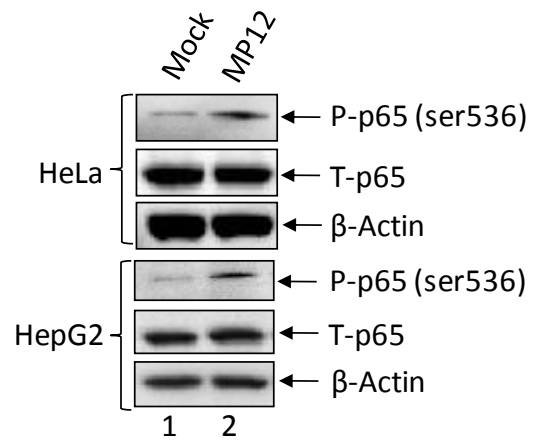


Supplemental Figure 1

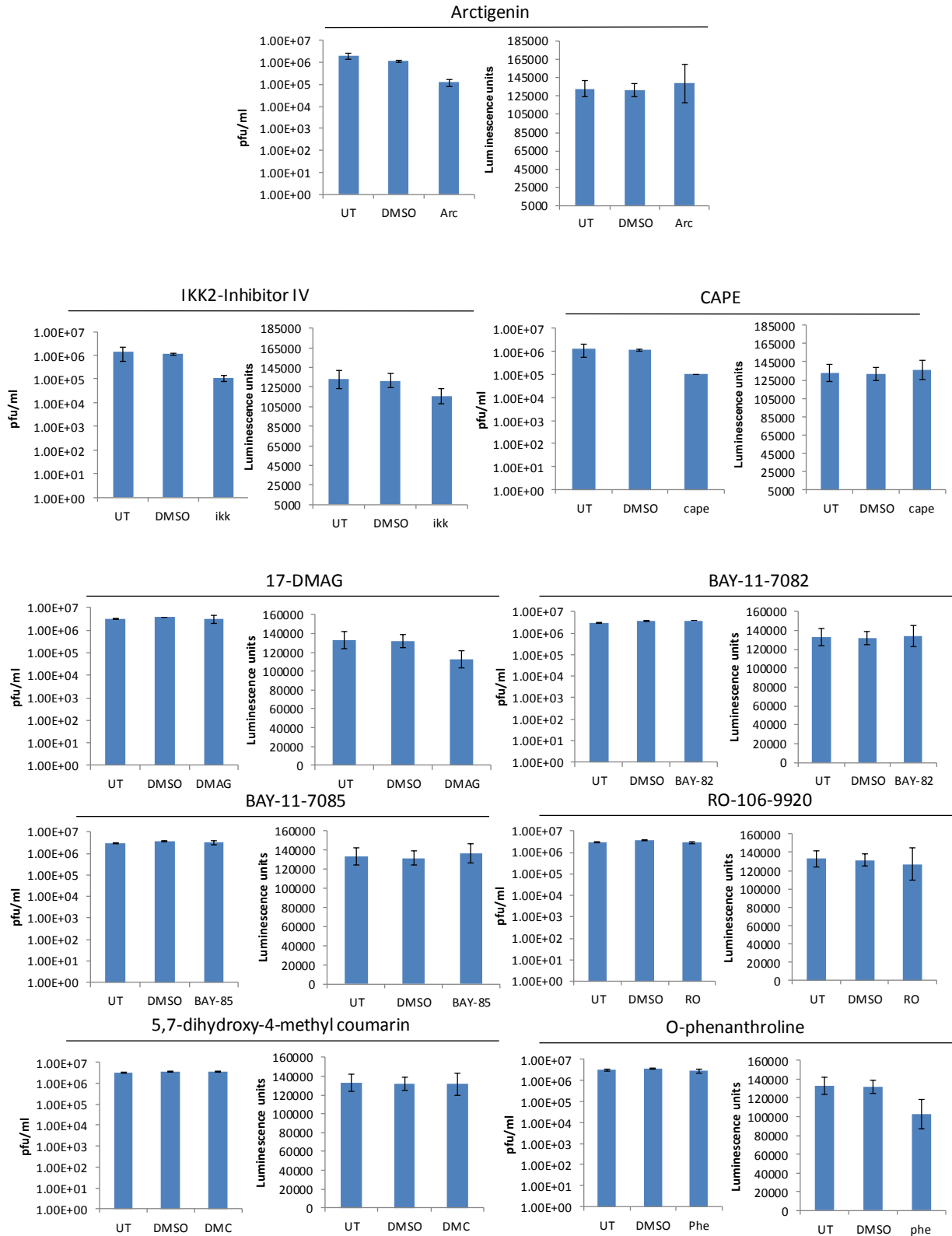
A)



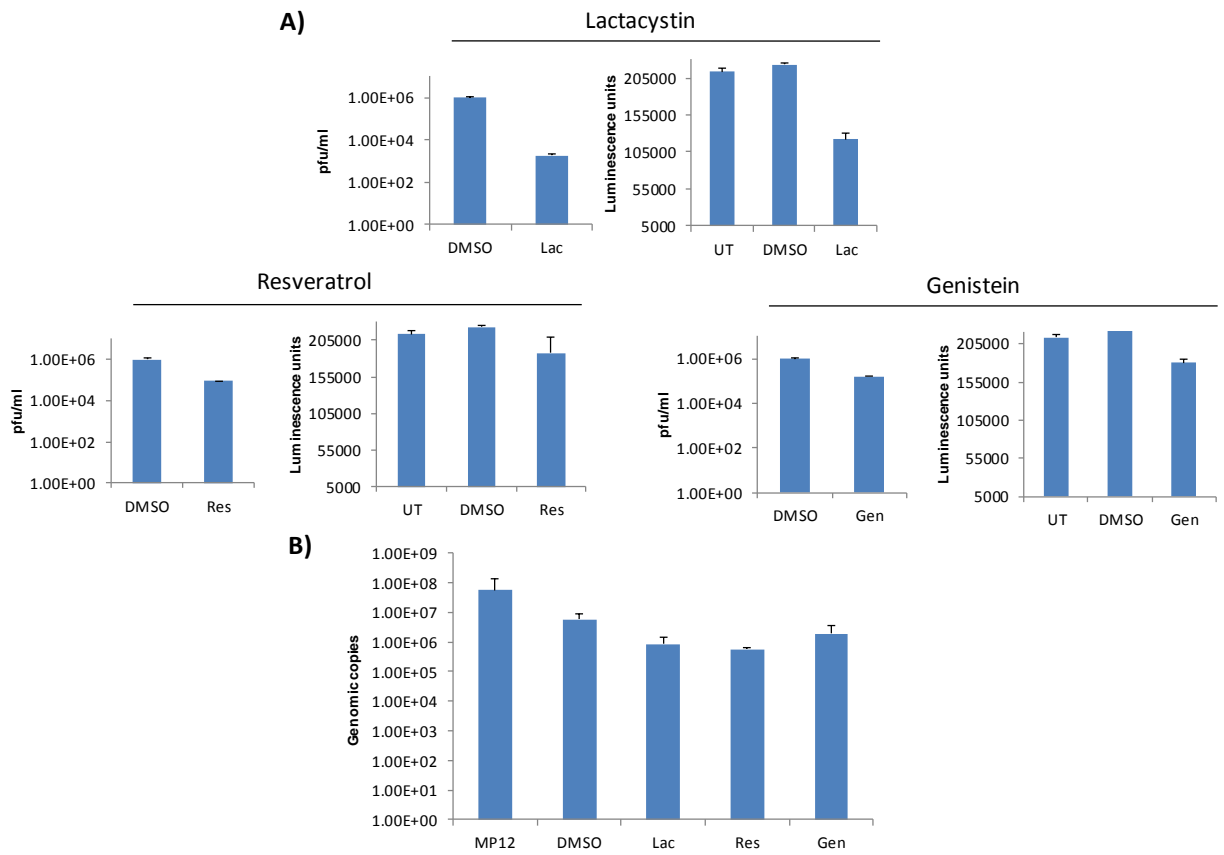
B)



Supplemental Figure 2



Supplemental Figure 3



Supplemental Figure 1. **A)** Inactivation of MP-12 after UV exposure was confirmed by carrying out plaque assays on vero cells. Mock infected cells were maintained as controls. Total number of colonies were counted and graphed. **B)** HeLa and HepG2 cells were infected with MP-12 virus. Phosphorylation status of p65 at 5 and 3 hours after infection respectively are indicated. Total p65 and β -actin levels were determined as controls.

Supplemental Figure 2. HSAECs were pretreated with multiple inhibitors of the NF κ B cascade for 2 hours, infected with MP-12 and post treated for up to 24 hours. All supernatants were quantified for infectious progeny virus by plaque assays and toxicity by CellTiter Glo assay.

Supplemental Figure 3. **A)** HSAECs were pretreated with the proteasome inhibitors, lactacystin, resveratrol and genistein for 2 hours. Pretreated cells were infected with MP-12 and continued to be post treated with the inhibitors. Twenty four hours later, supernatants were collected and quantified for infectious progeny virus by plaque assays. Inhibitor toxicity was also evaluated by measuring survival of inhibitor treated cells in comparison with DMSO treated and untreated cells. **B)** Viral genomic copies in culture supernatants obtained from MP-12-infected, proteasome inhibitor-treated HSAECs were evaluated using qRT-PCR.