**Supplementary Tables and Figure Legends**

**Supplementary Table 1: Nomenclature of oncolytic HSV-1 and their genetics**

**Supplementary Figure 1: RUX does not impair 67C-4 MPNST tumor cell growth**

67C-4 cells were treated with RUX at increasing concentrations (125-1000 nM) or vehicle control and monitored by the Incucyte Zoom® for cell proliferation over time.

**Supplementary Figure 2: Viral replication is integral to RUX/C134 anti-tumor effect.** Studies were repeated as previously described in Figure 4 and included as RUX+UV-inactivated C134 and a RUX+C134+Acyclovir cohort (1mg/ml x 5d). The Rux+C134 treatment reduced tumor growth but the UV-C134 and ACV-treated cohorts derived no anti-tumor benefit from combination therapy.

**Supplementary Figure 3: Combined RUX and C134 treatment effect on the infiltrating lymphocytes**

As shown in Figure 3A, C57BL/6 mice were implanted with (2X106) 67C-4 cells subcutaneously in the flank and tumor-bearing mice were randomized and divided into 4 treatment cohorts based on treatment regimen; Mock, RUX alone, C134 alone or RUX+C134. On Day 6 (Day 3 post-C134 treatment), tumors were harvested, processed into single-cell suspensions and the TILs analyzed by flow cytometry for: **A)** T cells **B)** CD8 T cell **C)** CD4 T cell and the **D)** flow charts represent the expression of CD44 on CD8T cells. **D)** CD4, CD25, and **E)** CD4, CD44 expression differences based upon treatment cohort.

**Supplementary Figure 4:** **Virus treatment increases CD8 activation in the periphery (D3) and combination therapy maintains the activation (D5)**

**A)** To identify differences in the T cell response in the periphery, mice from the different treatment cohorts (Mock, RUX, C134, and RUX+C134) were sacrificed on D6 (D3 post-oHSV) and D8 (D5 post-oHSV treatment). The results show that mice treated with C134 significantly increased CD8 activation in the periphery 3d following oHSV administration in the tumor. **B)** By D5 post-oHSV treatment, the activated CD8 population begins to decline and is no longer significantly elevated in the mice treated with C134 alone. The activated CD8 population in the spleen remains significantly elevated in the RUX+C134 treatment cohort. **C)** On day 10 (Day 7 post-viral treatment), the expression of FoxP3 on CD8T cells form tumors were analyzed by flow cytometry.

**Supplementary Figure 5: 67C-4 tumor cells express EphA2 and control peptide doesn’t induce CD8 T cell activation**

A) 67C-4 cells were harvested and stained with EphA2-PE antibody and analyzed by flow cytometry. B) C57BL/6 mice were implanted with (2X106) 67C-4 cells subcutaneously in the flank. Tumor-bearing mice were randomized and divided into 4 treatment cohorts (Mock, RUX alone, C134 alone or RUX+C134). Tumor-bearing mice were administered Saline or RUX (60 mg/Kg/day, IP) for three consecutive days then treated with vehicle or 3.5X107 C134 intratumoraly (IT) on Day 3. Three days post viral treatment; spleens were harvested, processed into single-cell suspensions and CFSE-labeled. A) splenocytes were stimulated with/without mitomycin C-treated 67C-4 for 6 hours in presence of protein transport inhibitor containing Brefeldin A (Golgi-plug™) and CD8 T lymphocytes were analyzed by flow cytometry for IFN-γ production by intracellular staining. B) splenocytes were incubated with 10 µM control peptide for 3 days. On day 3, CD8 T lymphocytes were analyzed by flow cytometry for proliferation (CFSE) and activation (CD25).