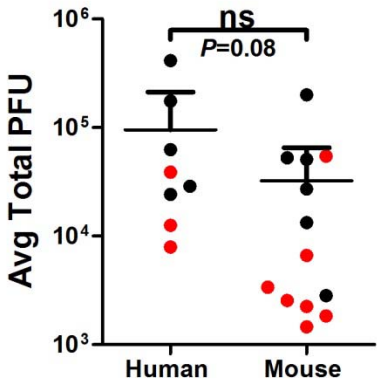
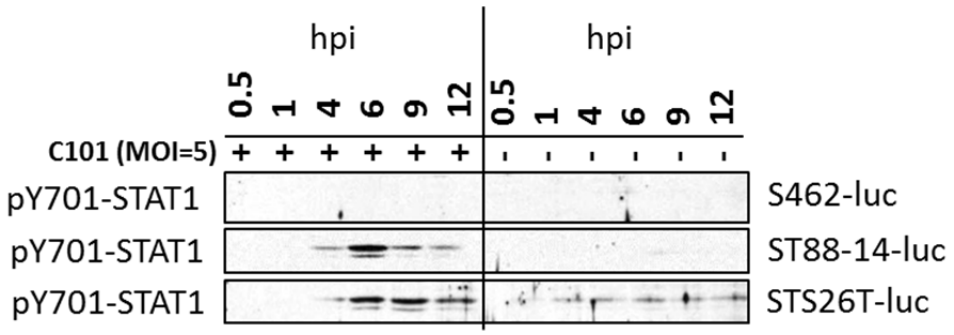
Supplemental Table 1: Description of Viruses

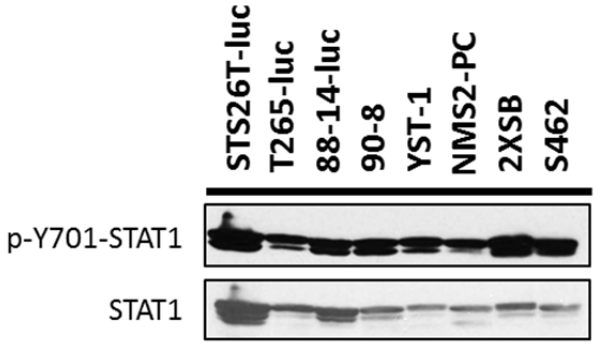
Virus	$\gamma_1$34.5	eGFP	Other Transgenes	Transgene Function
R3616	dual deletion			
C101	dual deletion	eGFP		
C134	dual deletion		HCMV IRS1	PKR evasion
C154	dual deletion	eGFP	HCMV IRS1	PKR evasion
M002	dual deletion		IL-12	T-cell activation
M201	dual deletion	eGFP	IL-12	T-cell activation
HSV-1 (F)	wild-type			
M2001	wild-type	eGFP		



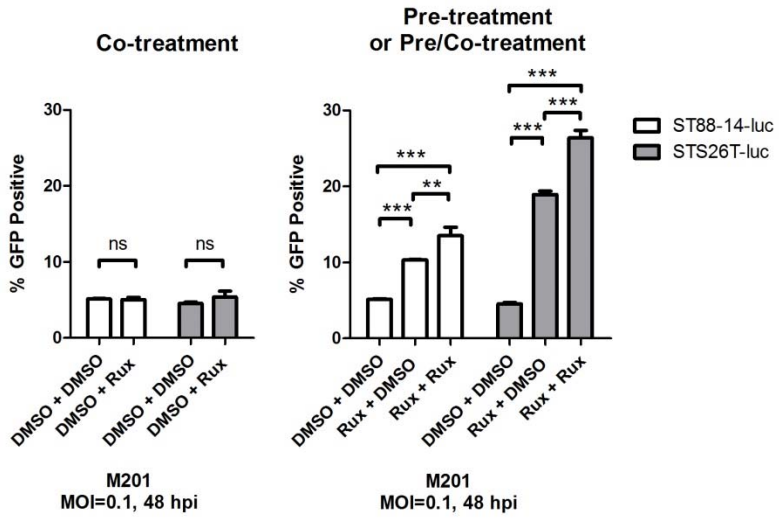
Supplemental Figure 1. Statistical comparison of titers between human and mouse MPNST cell lines. Human and mouse MPNST cell lines were infected with R3616 (MOI=1, 24 hpi) and titers of virus compared between human and mouse lines by Mann-Whitney U test. Black and red circles indicate STAT1 unresponsive and responsive cell lines respectively.



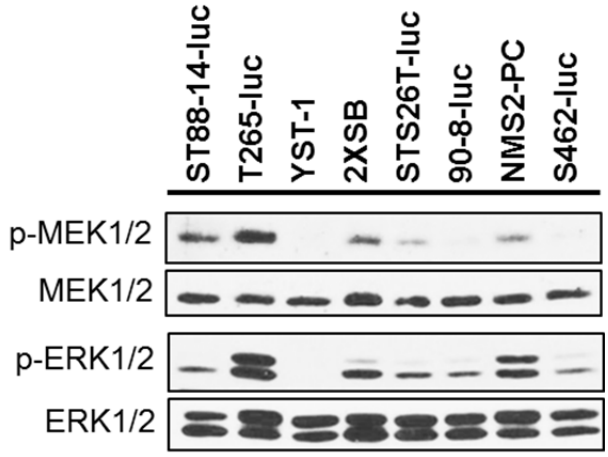
Supplemental Figure 2. Time course of STAT1 phosphorylation in response to oHSV infection. The optimal time point by which to detect STAT1 phosphorylation by western blot was determined by infecting cell lines with C101 at MOI=5 and lysates collected at various times after infection.



Supplemental Figure 3. STAT1 phosphorylation in response to IFNβ in human MPNST lines. The ability of human MPNST cell lines to respond to IFNβ and induce STAT1 phosphorylation was determined by incubation with 200 IU/ml human IFNβ for 30 min. STAT1 phosphorylation was subsequently detected by western blot.



Supplemental Figure 4. **Effect of JAK inhibitor ruxolitinib on M201 spread.** Cell lines were pretreated with either DMSO or 250 nM ruxolitinib (Rux) for 48 hrs prior to infection. One hour after oHSV infection, each group was further co-treated with DMSO or ruxolitinib (250 nM). The GFP expressing variant of M002 (M201) was used to assess effects on oHSV spread by multistep infection (MOI=0.1, 48 hpi).



Supplemental Figure 5. Endogenous MEK/ERK activation in human MPNST lines. Basal activation of Ras signaling in human MPNST cell lines was determined by probing MEK and ERK for phosphorylation by western blot.