**Supplementary Methods.**

***Animal Studies.*** Female athymic nude mice (ages 4-5 weeks) were implanted subcutaneously with 1x106 A673 Ewing sarcoma cells in 1:1 dilution of 10%FBS DMEM/Growth Factor Reduced Matrigel [Corning Inc., Tewksbury, MA](47). Upon tumor growth to 150~200mm3, mice were recruited to the study and randomized based on tumor size. Mice were treated with a single intratumoral injection of rHSVQ1 or RAMBO at 5.5x106 PFU/mouse. Tumors were collected three days after treatment, and fixed in 10% neutral-buffered formalin prior to processing for immunohistochemistry.

***Immunohistochemistry***. For immuno-histochemical study, rabbit anti-CD68 antibody [Abcam, San Francisco, CA; ab125212] was used in dilution 1/400. The 4-µm sections cut from paraffin-embedded sarcoma tissue block were deparaffinized with xylene, rehydrated in a gradient series of alcohol, and rinsed by PBS. After heat-induced antigen retrieval in sodium citrate buffer at pH 6 for 15 minutes, each section was covered with 3% hydrogen peroxide for 20 min to block endogenous peroxidase activity. These sections were then incubated with primary antibody at 4 oC overnight, and subsequently treated with donkey anti-rabbit secondary antibody conjugated to biotin [Jackson Laboratories, Bar Harbor, ME]. Slides were developed utilizing avidin-conjugated horseradish peroxidase (HRP) with diaminiobenzidine (DAB) as substrate. Following development, the slides were counterstained with hematoxylin, mounted under coverslips with permount, and imaged.

***Co-culture Assays.*** To generate Vstat120-expressing U251 glioma cells, we transfected cells with Vstat120 expression plasmid(13) using Lipofectamine 2000 (ThermoFisher), per manufacturers protocol. Transfected cells were incubated 24 hours prior to overlay and expression of Vstat120 confirmed by western blot analysis.

**Supplementary Figure Legends.**

Supplementary Figure 1. Ewing sarcoma subcutaneous tumors stained for CD68+ macrophages, with hematoxylin counterstain. Nude mice were implanted with A673 Ewing sarcoma cells, and recruited to the study upon tumor development. Mice were randomized based on size, and treated with a single injection of oHSV. Three days after oHSV treatment, tumors were collected and sectioned for immunohistochemistry. Left panel, representative light microscopy images from treated with rHSVQ1, and stained for CD68+ macrophage cells (200x). Right panel, representative light microscopy images from tumors treated with RAMBO (200x).

Supplementary Figure 2. **(A)** Schematic of experimental design used: U251 glioma cells (brown) were infected with oHSV (rHSVQ1; green) for an hour before unbound virus was washed away. Microglia (BV2, blue; 2:1 ratio) and U251 glioma (control or Vstat120-transfected; 1:1 ratio) were overlaid onto infected glioma. 12h after overlay, co-cultures were imaged with phase contrast and fluorescence (GFP, indicated of oHSV infection). **(B)** Adding Vstat120-expressing glioma cells has no effect on rHSVQ1 replicative capacity, compared to adding control glioma. Upper images, phase contrast (10x). Lower images, GFP fluorescence, indicative of oHSV infection (10x). (C) Adding microglia to rHSVQ1-infected glioma in the presence of control glioma overlay results in a decrease in oHSV replication. The presence of Vstat120-expressing glioma rescues this effect, as indicated by an increase in GFP.

**Supplementary References.**

47. Eshun FK, Currier MA, Gillespie RA, Fitzpatrick JL, Baird WH, Cripe TP. VEGF blockade decreases the tumor uptake of systemic oncolytic herpes virus but enhances therapeutic efficacy when given after virotherapy. Gene Ther. 2010;17:922-9.