two-fold difference in SEAP level between 6a and 6b is probably due to variations in either cell density or transfection efficiency between these two experiments.

Figure 7. Endogenous *ISG56* and interferon expression in tumor cells with or without FusOn-H2 infection and the effect of externally added IFNs on ISRE activity. **a** and **b**. Endogenous ISG56 and interferon expression in tumor cells with or without FusOn-H2 infection. Tumor cells were seeded into six-well plates in duplicate and incubated overnight at 37^0C. One set of cells was harvested and the others were infected with 1 pfu/cell of FusOn-H2 for 24 h before harvesting. The total RNA was prepared for RT-PCR quantification of *ISG56* transcripts (a) or *IFN-β* transcripts (b) as described in Materials and Methods. ✦p<0.01 vs. MCF-7 or HuH-7, ✳p<0.05 vs. before infection. **c**. Effect of externally added IFNs on ISRE activity. Tumor cells were initially transfected with pJ-ISRE-SEAP. Then, different types of IFNs were added to the medium followed by 24 h of incubation before the collection of supernatants for SEAP assay. The fold of increase in SEAP activity was calculated by dividing the amount of SEAP released into the medium before addition of IFNs by that measured 24 h after IFN incubation. The Huh-7, PC3, HepG2, SW480 and A549 tumor cells are all permissive to FusOn-H2 replication.

S1. **Neutrophil infiltration in B16 tumors after virotherapy.** $2X10^5$ B16 cells were implanted subcutaneously to the right flank of C57BL/6 mice. Once tumors reached the approximate size of 5 mm in diameter, they were injected with $3X10^6$ pfu of either FusOn-H2 or Baco-1 (an HSV-1-based oncolytic virus) or PBS. Tumors were explanted two days later and tumor sections were prepared for H&E staining. The infiltrating neutrophils were quantitated by counting 10 fields (40X) under a microscope and the average numbers are plotted. ✦p<0.01 vs. Baco-1.