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Supplemental Information

Combination of IAP Antagonists and

TNF-*α***-Armed Oncolytic Viruses Induce Tumor**

Vascular Shutdown and Tumor Regression

Shawn T. Beug, Stephanie J. Pichette, Martine St-Jean, Janelle Holbrook, Danielle E. Walker, Eric C. LaCasse, and Robert G. Korneluk







Figure S2. SMC treated cancer cells are killed at a faster rate with infection by TNF α -armed oncolytic VSV. Enumeration of DEVD-FITC signals from EMT6 cells treated with vehicle or 5 μ M of the SMC LCL161 and PBS or 1 MOI of the indicated virus. Images were collected over 16 h using a time-lapse microscope and quantitated using the IncucyteZoom software. Mean, SEM.



Figure S3. Vero cells are resistant to SMC-mediated and TNFα-induced cell death.

Alamar Blue assays of Vero cells treated with vehicle or 5 μM of the SMC LCL161 and 0.01% BSA or 10 μg/ml TNFα for 48 h. Mean, SD.



Figure S4. SMC treatment does not alter the kinetics of TNF α -armed oncolytic VSV. EMT6 and SNB75 cells were treated with vehicle or 5 μ M of the SMC LCL161 and infected with 1 or 0.01 MOI and the supernatant was collected at the indicated times for quantitation of the virus titer using BHK-21 or Vero cells for 48 h, respectively. Dead cells were scored to determine the TCID₅₀/mL.



Figure S5. Responsiveness of a SMC sensitive and resistant cell lines to the combinaiton of SMC and TNF α -armed oncolytic VSV.

Alamar blue viability assays of mouse (CT-26) and human (786-0, SF539, H460, H661) cancer cells treated with vehicle or 5 μ M of the SMC LCL161 and increasing MOI of the indicated virus for 48 h. Mean, SD.



Figure S6. Efficacy of siRNA-mediated knockdown.

SNB75 cells were transfected with combinations of non-targeting (NT), RIP1, CASP8, TNF-R1 or DR5 siRNA for 48 h. Equal amounts of soluble protein were separated on polyacrylamide gels followed by transfer to nitrocellulose membranes and probed with the indicated antibodies.