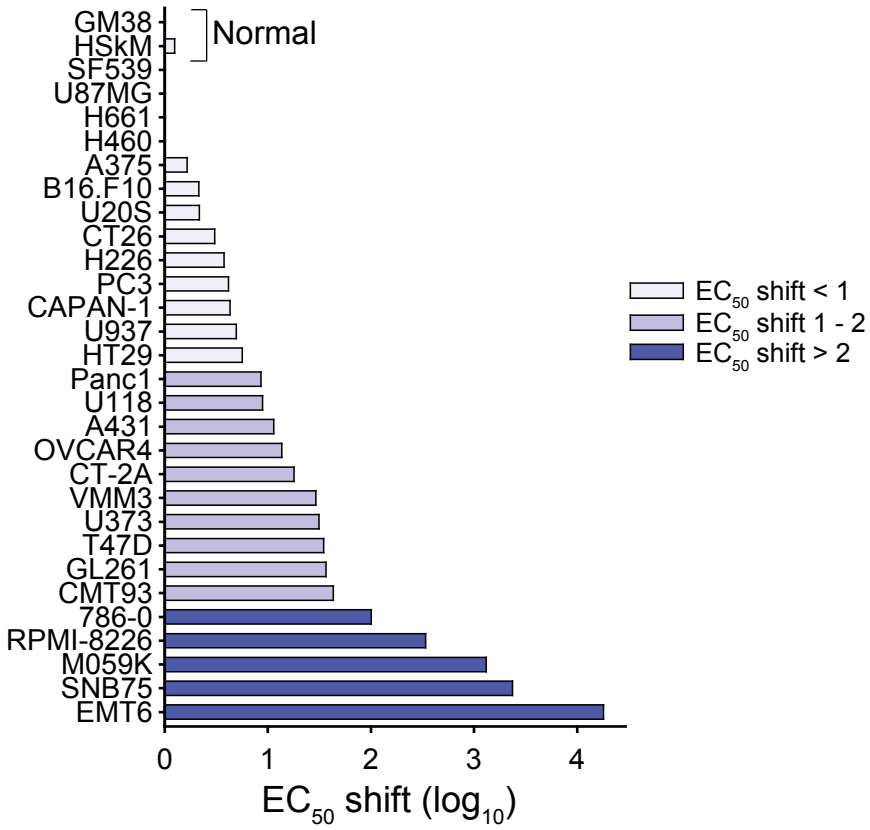
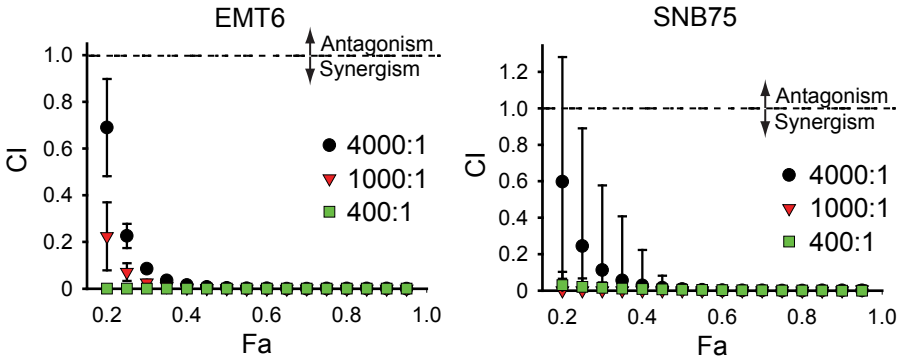
Supplementary Figure 1 Responsiveness of a panel of cancer and normal cells to the combinatorial treatment of SMC and oncolytic virus.



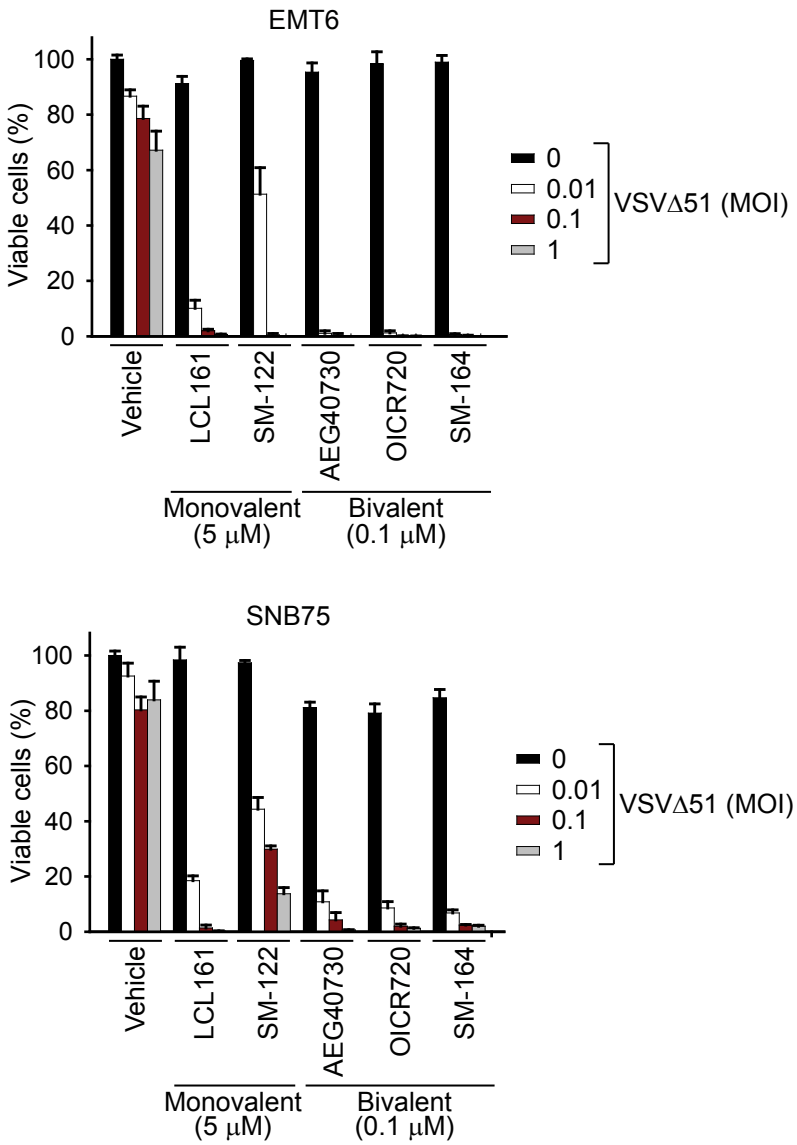
The indicated cancer cell lines (n = 28) and non-cancer human cells (primary human skeletal muscle (HSkM) and human fibroblasts (GM38)) were treated with 5 μM LCL161 and increasing VSVΔ51 for 48 hr. The dose required to yield 50% viable cells in the presence in SMC versus vehicle was determined using nonlinear regression and plotted as a $\log_{10}$ EC50 shift toward increasing sensitivity. Representative data from at least two independent experiments using biological replicates (n = 3).

Supplementary Figure 2 SMC and oncolytic virus co-treatment is highly synergistic in cancer cells.



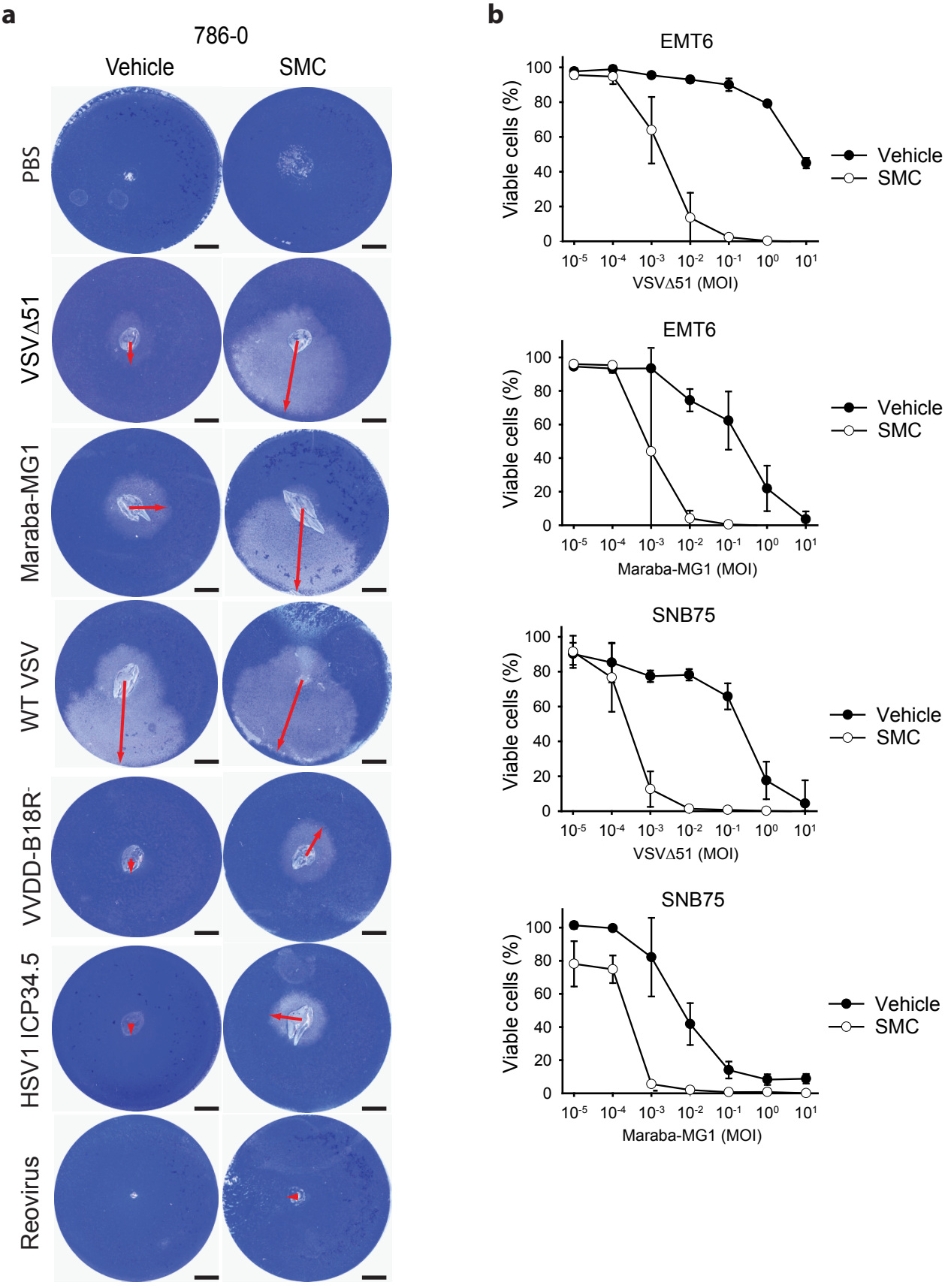
Alamar blue viability of cells treated with indicated dilutions of a fixed ratio combination mixture of VSVΔ51 and LCL161 (PFU : μM LCL161) at 48 hr post-treatment. Combination indexes (CI) were calculated according to Chou and Talalay[47] using Calcusyn. Plots represent the algebraic estimate of the CI in function of the fraction of cells affected (Fa). Error bars, mean ± s.e.m. Representative data from three independent experiments using biological replicates (n = 3).

Supplementary Figure 3 Monovalent and bivalent SMCs synergize with oncolytic viruses to cause cancer cell death.



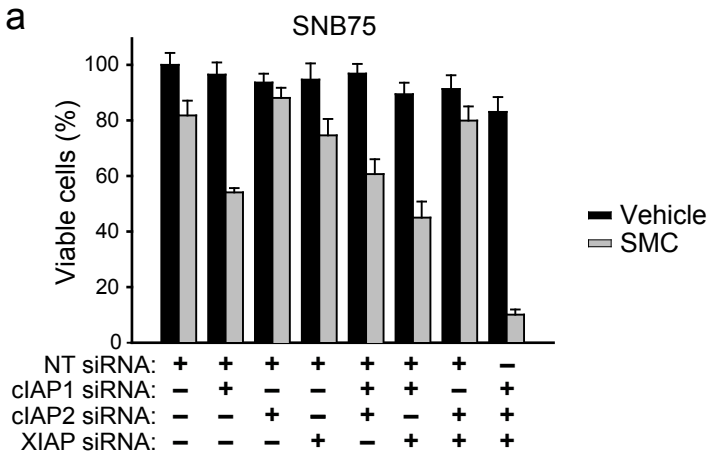
Alamar blue viability assay of cells treated with 5 μM monovalent SMCs (LCL161, SM-122) or 0.1 μM bivalent SMCs (AEG40730, OICR720, SM-164) and VSVΔ51 at differing MOIs. Cells were treated for 48 hr. Error bars, mean ± s.d. Representative data from three independent experiments using biological replicates (n = 3).

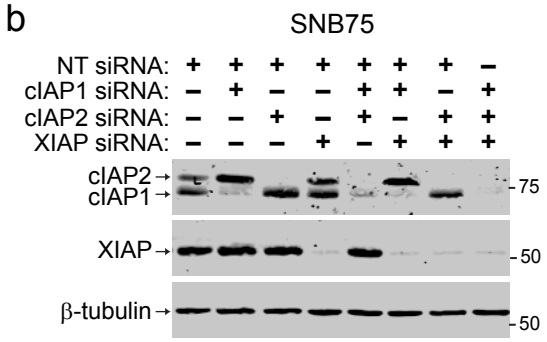
Supplementary Figure 4 SMC-mediated cancer cell death is potentiated by oncolytic rhabdoviruses.



a, Virus spreading assay of cells that were overlaid with 0.7% agarose in the presence of vehicle or 5 μM LCL161 and 500 PFU of the indicated viruses were dispensed in to the middle of the well. Cytotoxicity was assessed 96 hr post-treatment by crystal violet staining. Arrow denotes extension of the cell death zone from the origin of OV infection. Scale bar, 5 mm. **b,** Alamar blue viability of cells treated with 5 μM LCL161 and increasing MOIs of VSVΔ51 or Maraba-MG1 at 48 hr post-treatment. Error bars, mean ± s.d. Representative data from two independent experiments using biological replicates (n = 3).

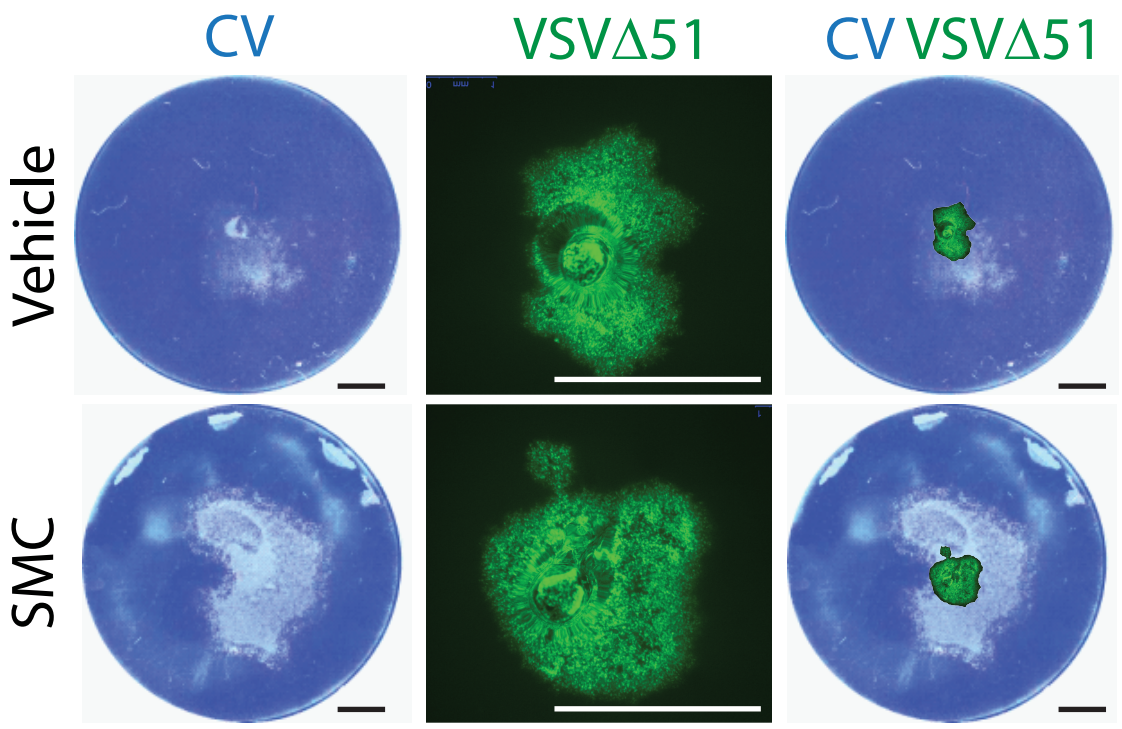
Supplementary Figure 5 cIAP1, cIAP2 and XIAP cooperatively protect cancer cells from oncolytic virus induced cell death.





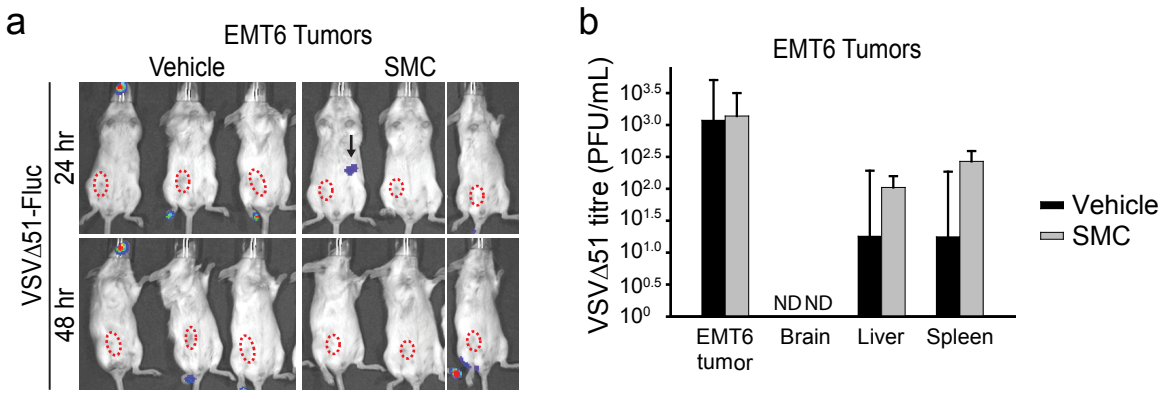
a, Alamar blue viability of cells transfected with nontargeting (NT) siRNA or siRNA targeting cIAP1, cIAP2 or XIAP, and subsequently treated with 5 μM LCL161 and 0.1 MOI VSVΔ51 for 48 hr. Error bars, mean ± s.d. Representative data from three independent experiments using biological replicates (n = 3). **b,** Representative siRNA efficacy Western blots for the experiment depicted in (a) are displayed.

Supplementary Figure 6 Images used for superimposed images depicted in Fig. 1g.



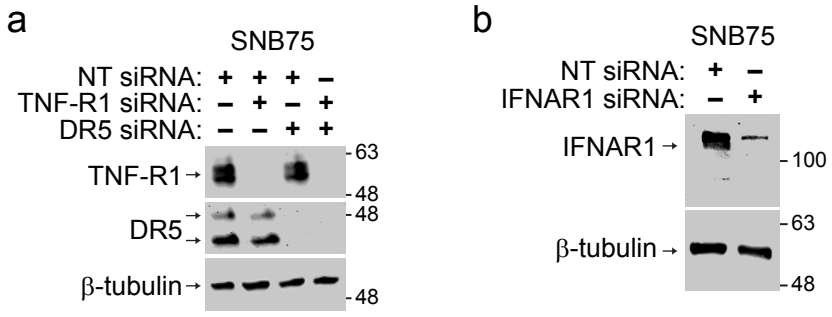
Cells were overlaid with agarose media containing 5 μM LCL161, inoculated with 500 PFU of VSVΔ51-GFP in the middle of the well, and infectivity measured by fluorescence and cytotoxicity was denoted by crystal violet (CV) staining at 96 hr post-treatment. Scale bar, 5 mm.

Supplementary Figure 7 SMC treatment does not affect oncolytic virus distribution or replication in vivo.



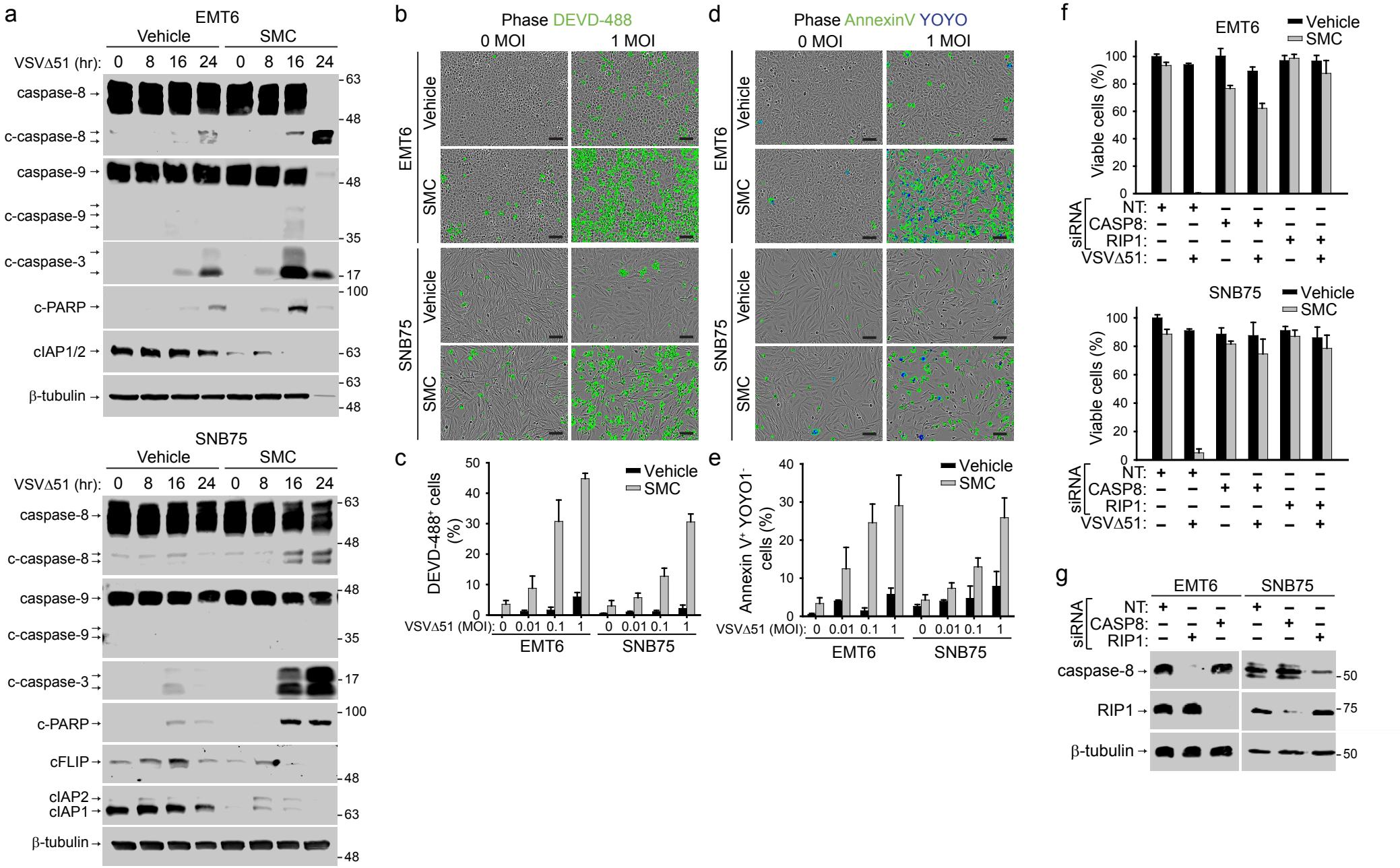
a, EMT6-bearing mice were treated with 50 mg/kg LCL161 (per os) and $5x10^8$ PFU firefly luciferase tagged VSVΔ51 (VSVΔ51-Fluc) via i.v. injection. Virus distribution and replication was imaged at 24 and 48 hrs using the IVIS. Red outline denotes region of tumours. Representative data from two independent experiments. Arrow indicates spleen infected with VSVΔ51-Fluc. **b,** Tumors and tissues at 48 hr post-infection were homogenized and virus titrations were performed for each group. Error bars, mean ± s.e.m.

Supplementary Figure 8 Verification of siRNA-mediated knockdown.



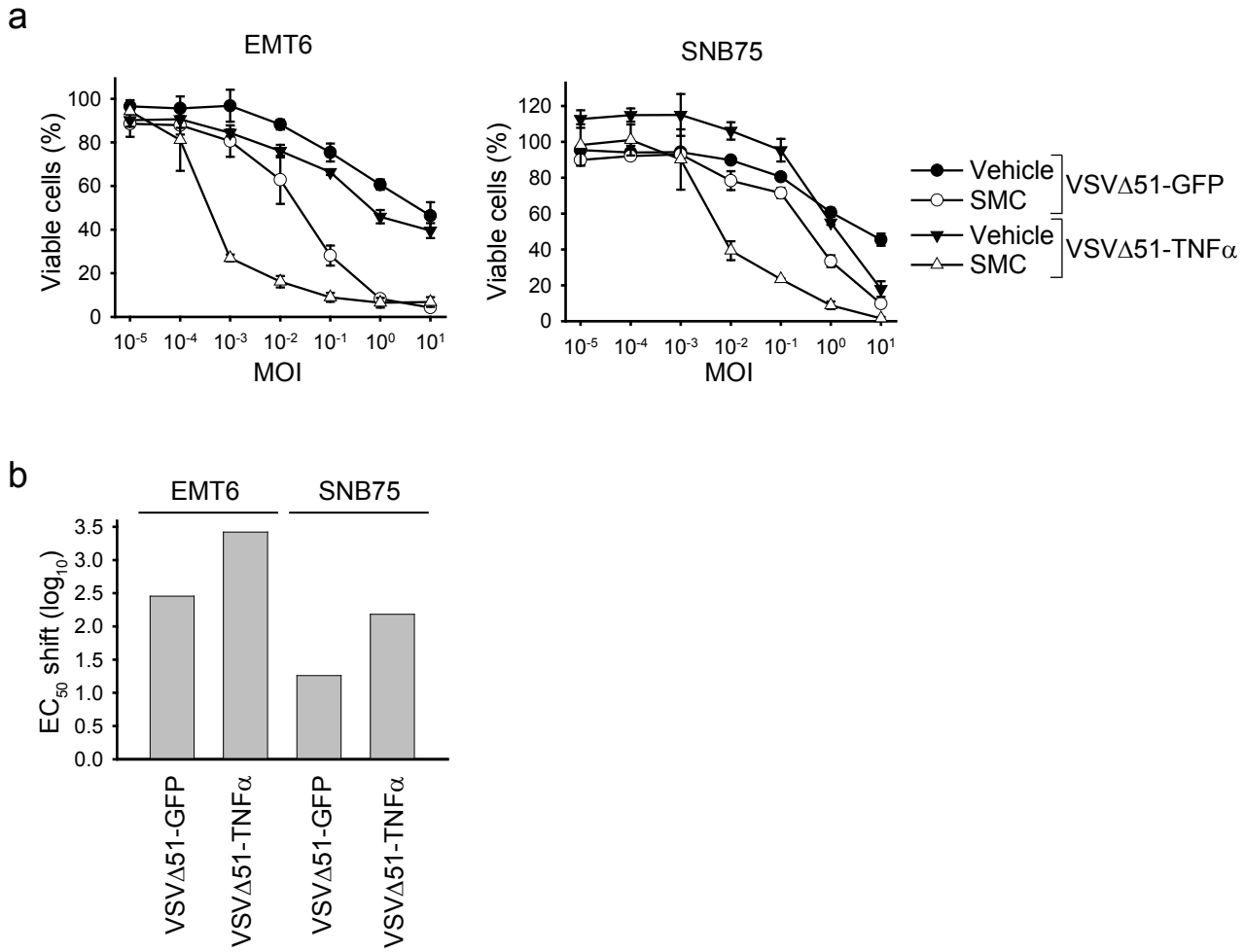
Verification of siRNA-mediated knockdown of non-targeting (NT), TNFR1, DR5 and IFNAR1 by Western blotting from Fig. 3a (a) and Fig. 3b (b).

Supplementary Figure 9 SMCs synergizes with oncolytic viruses to induce caspase-8- and RIP-1-dependent apoptosis in cancer cells.



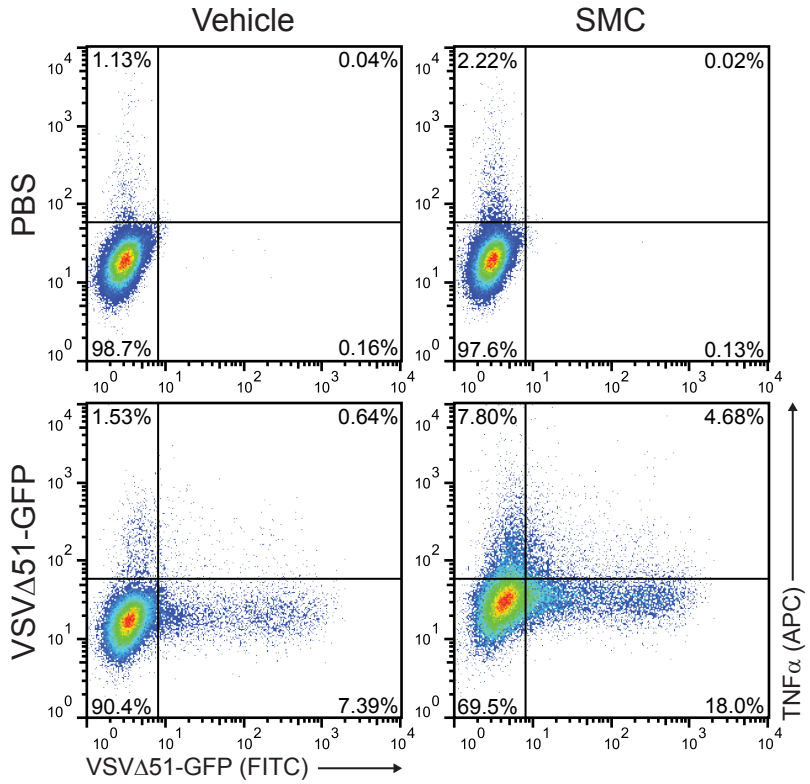
a, Western blotting for caspase and PARP activation was conducted on cells pretreated with 5 μM LCL161 (2 hr) and subsequently treated with 1 MOI of VSVΔ 51. **b,** Micrographs of caspase activation were acquired with cells that were cotreated with 5 μM LCL161 and VSVΔ51 in the presence of the caspase-3/7 substrate DEVD-488 for 24 hr. Scale bar, 100 μm. **c,** The proportion of DEVD-488-positive cells from (b) was plotted (n = 12). Error bars, mean ± s.d. **d,** Apoptosis was assessed by micrographs of translocated phosphatidyl serine (Annexin V-CF594, green) and loss of plasma membrane integrity (YOYO-1, blue) in cells treated with 5 μM LCL161 and VSVΔ51 for 24 hr. Scale bar, 100 μm. **e,** The proportion of Annexin V-CF594-positive and YOYO-1-negative apoptotic cells from (d) was plotted (n = 9). Error bars, mean ± s.d. **f,** Alamar blue viability of cells transfected for 48 hr with nontargeting (NT) siRNA or siRNA targeting caspase-8 or RIP1, and subsequently treated with 5 μM LCL161 and 0.1 MOI of VSVΔ51 (n = 3) for 48 hr. Error bars, mean ± s.d. n = 3. **g,** Representative siRNA efficacy western blots for (f) are displayed. All panels: representative data from three independent experiments using biological replicates.

Supplementary Figure 10 Expression of TNFα transgene from oncolytic viruses potentiates SMC-mediated cancer cell death further.



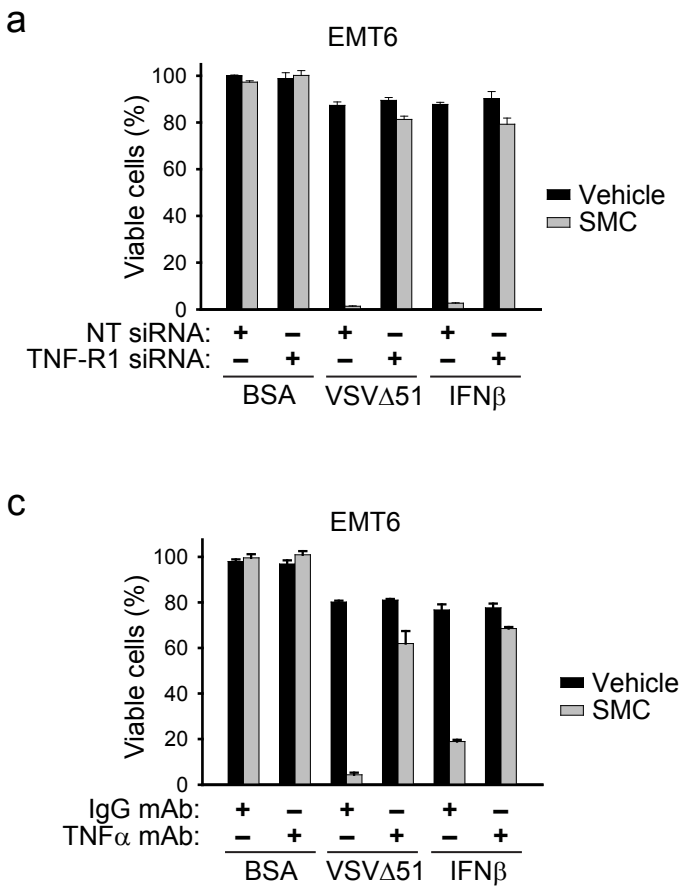
a, Alamar blue viability assay of cells cotreated with 5 μM SMC and increasing MOIs of VSVΔ51-GFP or VSVΔ 51-TNFα for 24 hr. Error bars, mean ± s.d. **b,** Representative EC_{50} shifts from (a). The dose required to yield 50% viable cells in the presence in SMC versus vehicle was determined using nonlinear regression and plotted as EC_{50} shift. Representative data from three independent experiments using biological replicates (n = 3).

Supplementary Figure 11 Oncolytic virus infection leads to enhanced TNFα expression upon SMC treatment.



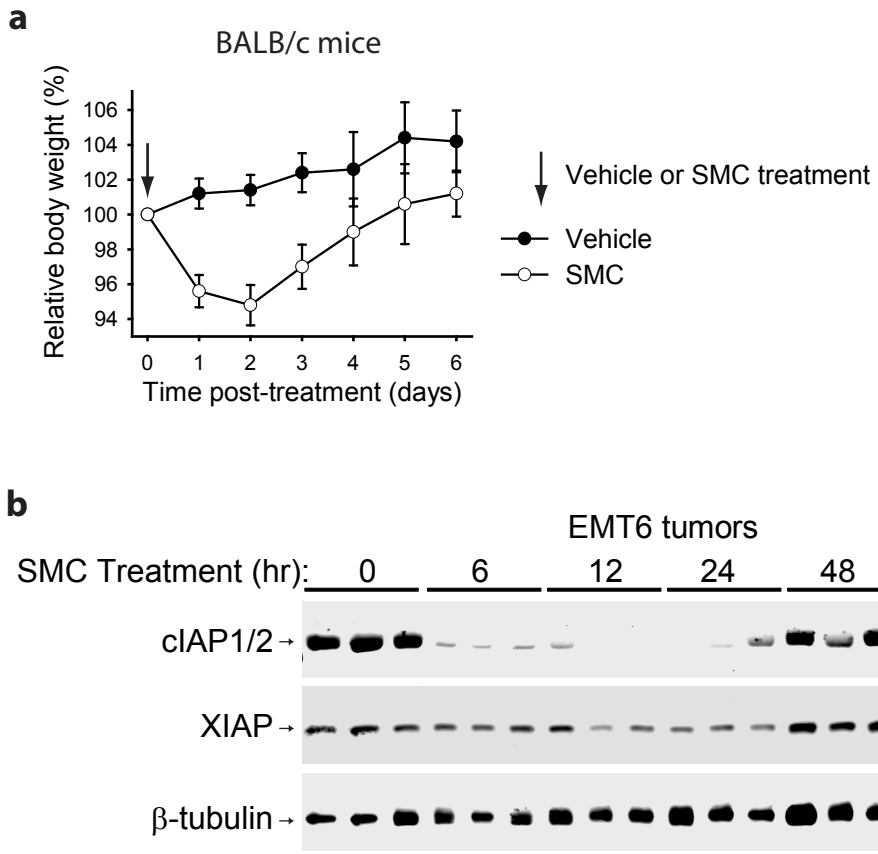
EMT6 cells were cotreated with 5 μM SMC and 0.1 MOI VSVΔ51-GFP for 20 hr, and cells were processed for the presence of intracellular TNFα via flow cytometry. Representative data from four independent experiments.

Supplementary Figure 12 TNFα signalling is required for type I IFN induced synergy with SMC treatment.



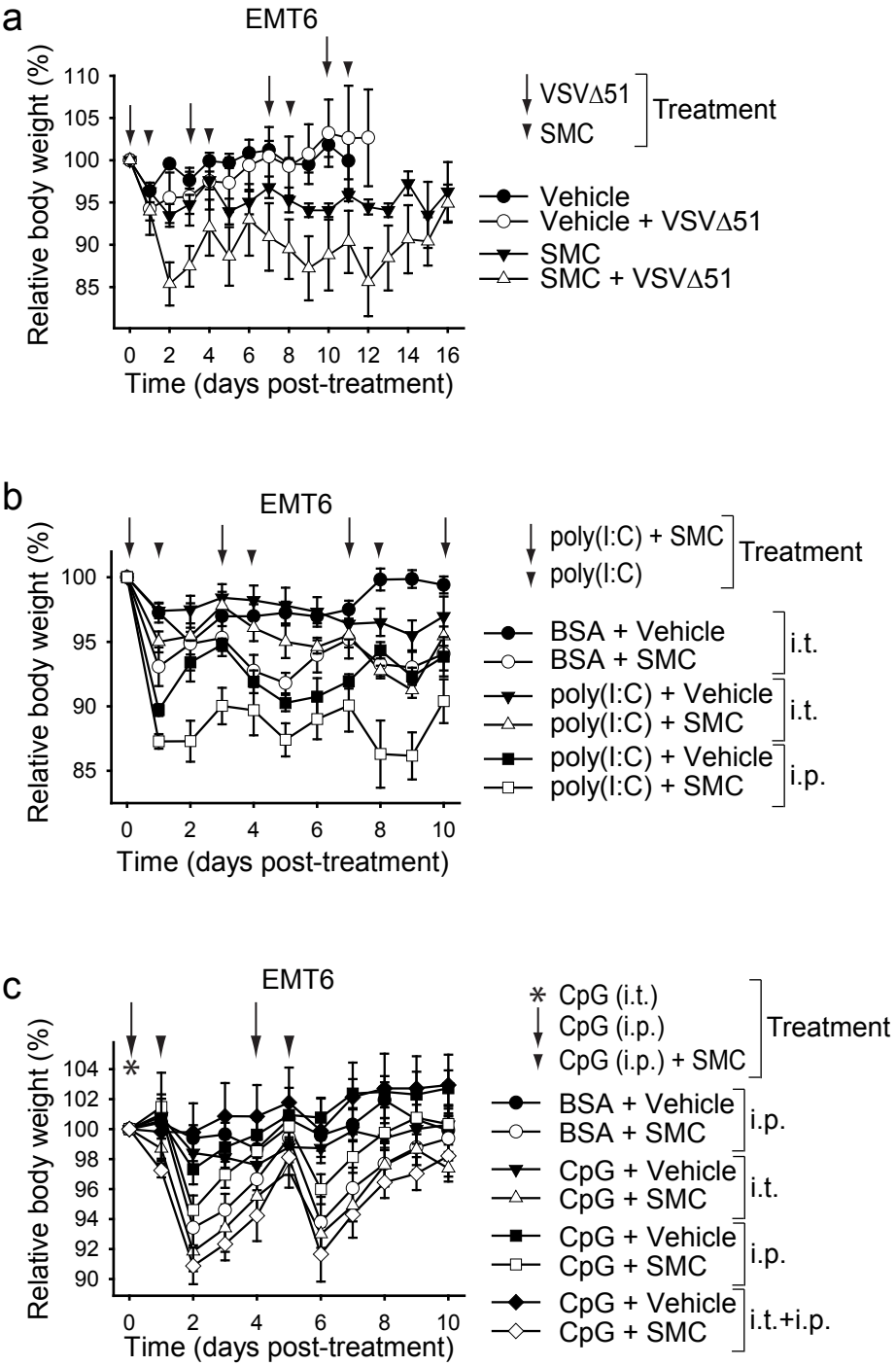
a, Alamar blue viability assay of EMT6 cells transfected with nontargeting (NT) or TNF-R1 siRNA for 48 hr and subsequently treated with 5 μM LCL161 and VSVΔ51 (0.1 MOI) or 250 U/mL IFNβ for 48 hr. Error bars, mean ± s.d. b, Representative siRNA efficacy blot from (a). c, Viability of EMT6 cells that were pretreated with TNFα neutralizing antibodies for 2 hr and subsequently treated with 5 μM LCL161 and 0.1 MOI of VSVΔ51 or 250 U/mL of IFNβ. All panels: representative data from at least three independent experiments using biological replicates (n = 3).

Supplementary Figure 13 SMC treatment causes minimal transient weight loss and leads to downregulation of cIAP1/2.



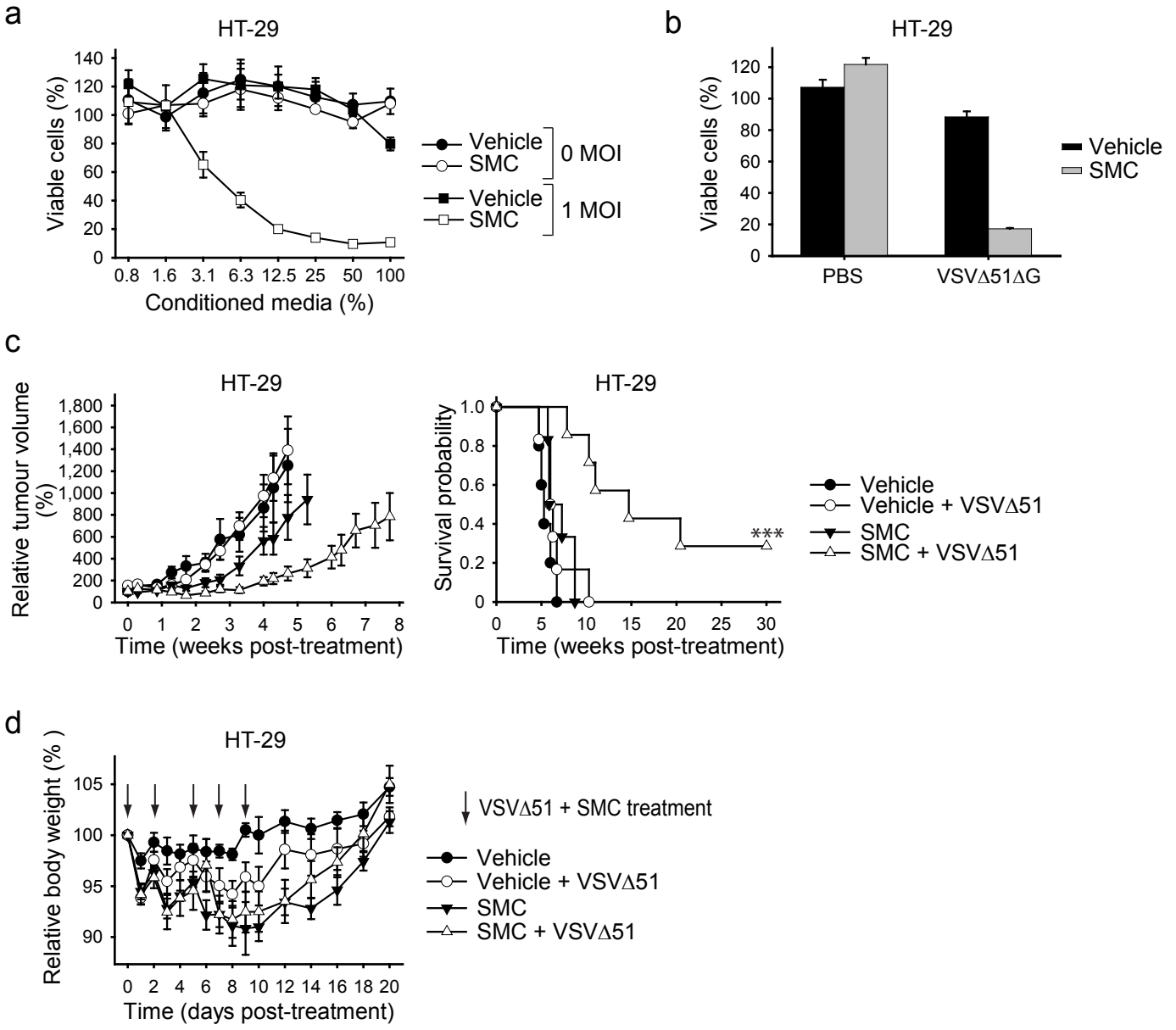
a, Weights from SMC treated mice female BALB/c mice (50 mg/kg LCL161, per os) were recorded after a single treatment (day 0). n = 5 per group. Error bars, mean ± s.e.m. **b,** EMT6-tumour bearing mice were treated with 50 mg/kg LCL161 (per os) and tumours harvested at the indicated times for Western blotting using the indicated antibodies.

Supplementary Figure 14 SMC treatment induces transient weight loss in a syngeneic mouse model of cancer.



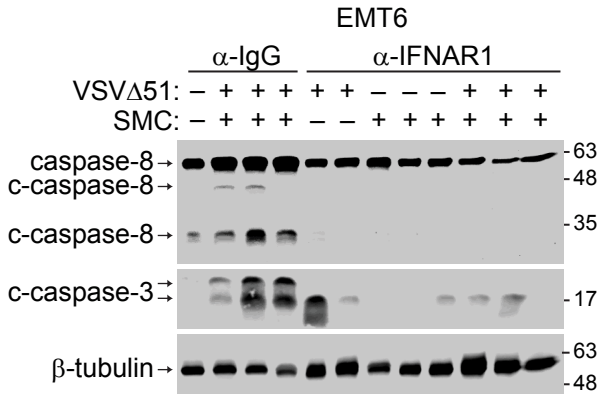
a-c, Measurement of mouse weights upon SMC and oncolytic VSV (a), poly(I:C) (b) or CpG (c) co-treatment in tumour-bearing animals from the experiments depicted in Fig. 4a, 5b, 5d, respectively. Error bars, mean ± s.e.m.

Supplementary Figure 15 VSVΔ51-induced cell death in HT-29 cells is potentiated by SMC treatment in vitro and in vivo.



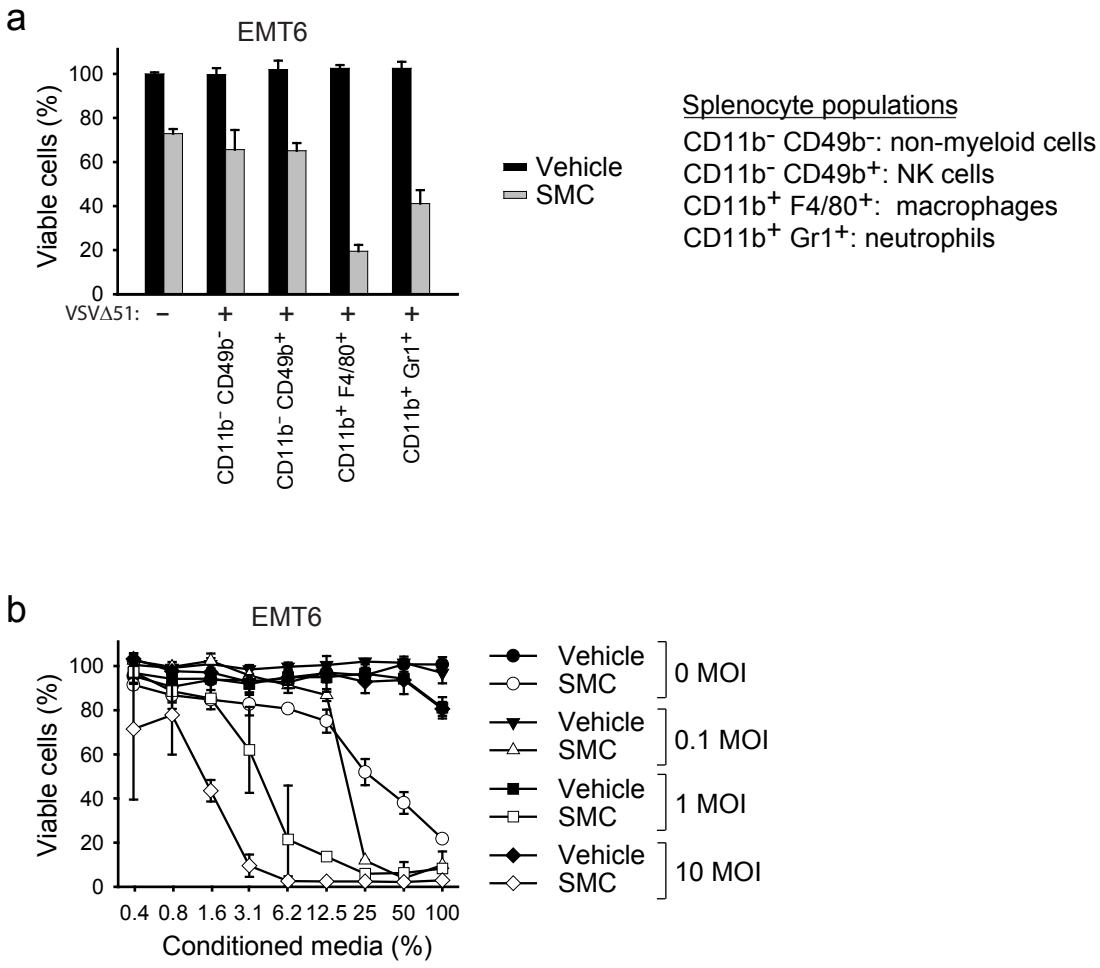
a, Cells were infected with VSVΔ51 for 24 hr and the cell culture supernatant was exposed to UV light for 1 hr. The UV-inactivated supernatant was applied to new HT-29 cells at the indicated dose in the presence of 5 μM LCL161 for 48 hr. Viability was ascertained by Alamar blue. Error bars, mean ± s.d. Representative data from three independent experiments using biological replicates (n = 3). **b,** Alamar blue viability of cells cotreated with 5 μM LCL161 and a non-spreading virus VSVΔ51ΔG (0.1 MOI). Error bars, mean ± s.d. Representative data from three independent experiments using biological replicates (n = 3). **c,** CD-1 nude mice with established HT-29 tumours were treated with 50 mg/kg LCL161 (per os) and $1x10^8$ PFU VSVΔ51 (intratumoral). Vehicle, n = 5; vehicle + VSVΔ51, n = 6; SMC, n = 6; SMC + VSVΔ51, n = 7. Left panel depicts tumour growth relative to day 0 post-treatment. Right panel represents the Kaplan-Meier curve depicting mouse survival. Error bars, mean ± s.e.m. Log-rank with Holm-Sidak multiple comparison: ***, $p < 0.001$ d, Measurement of mouse weights upon SMC and OV co-treatment in tumour-bearing animals from the experiment depicted in (c). Error bars, mean ± s.e.m.

Supplementary Figure 16 Type I IFN signalling is required for SMC and oncolytic virus synergy in vivo.



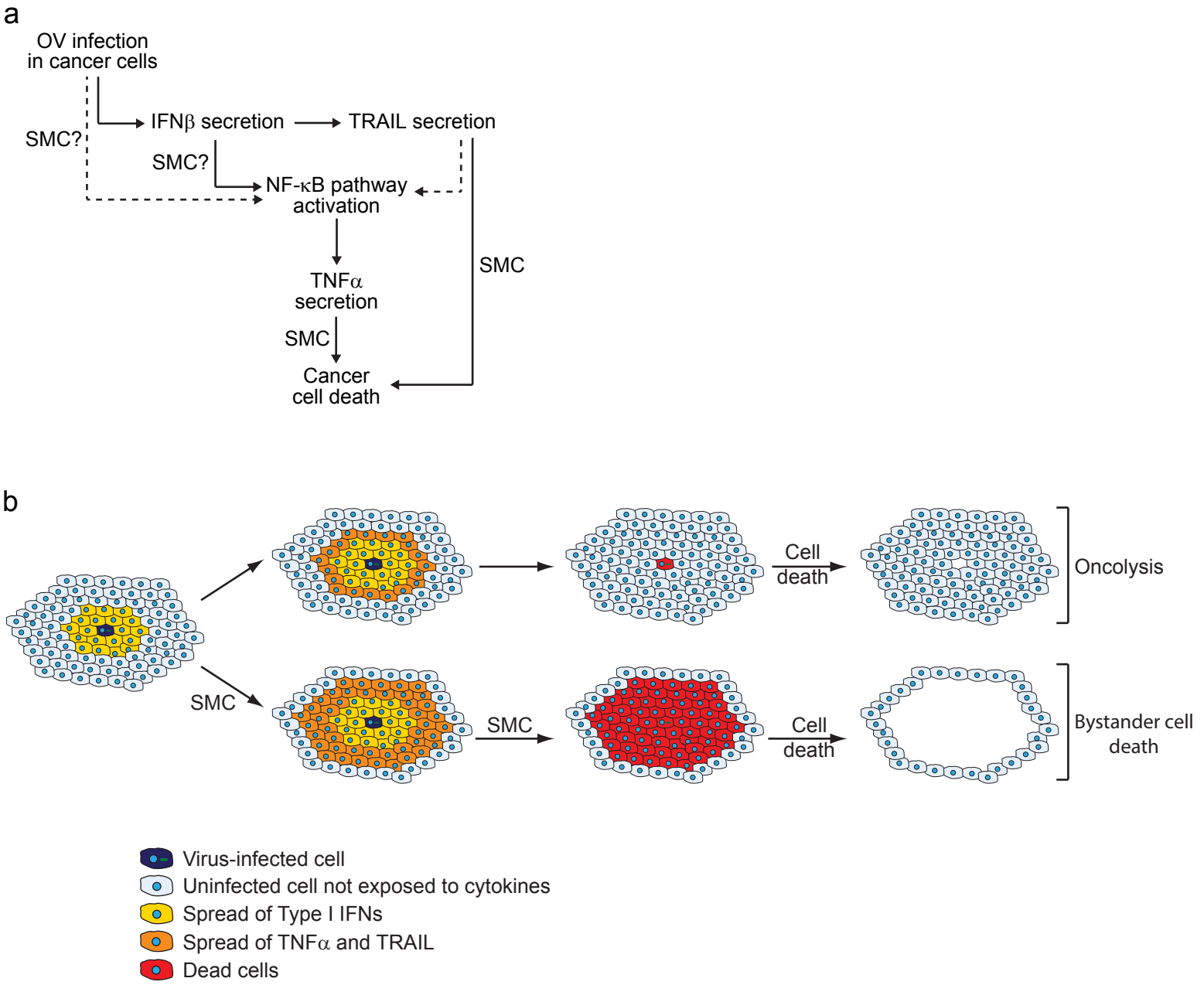
EMT6 tumour bearing mice were treated with vehicle or 50 mg/kg of the LCL161 for 4 hr, and subsequently treated with neutralizing IFNAR1 or isotype antibodies for 20 hr. Subsequently, animals were treated with PBS or VSVΔ51 (5x108 PFU) i.v. for 18 hr. Tumours were processed for Western blotting with the indicated antibodies.

Supplementary Figure 17 Oncolytic virus infection of innate immune cells leads to cancer cell death in the presence of SMCs.



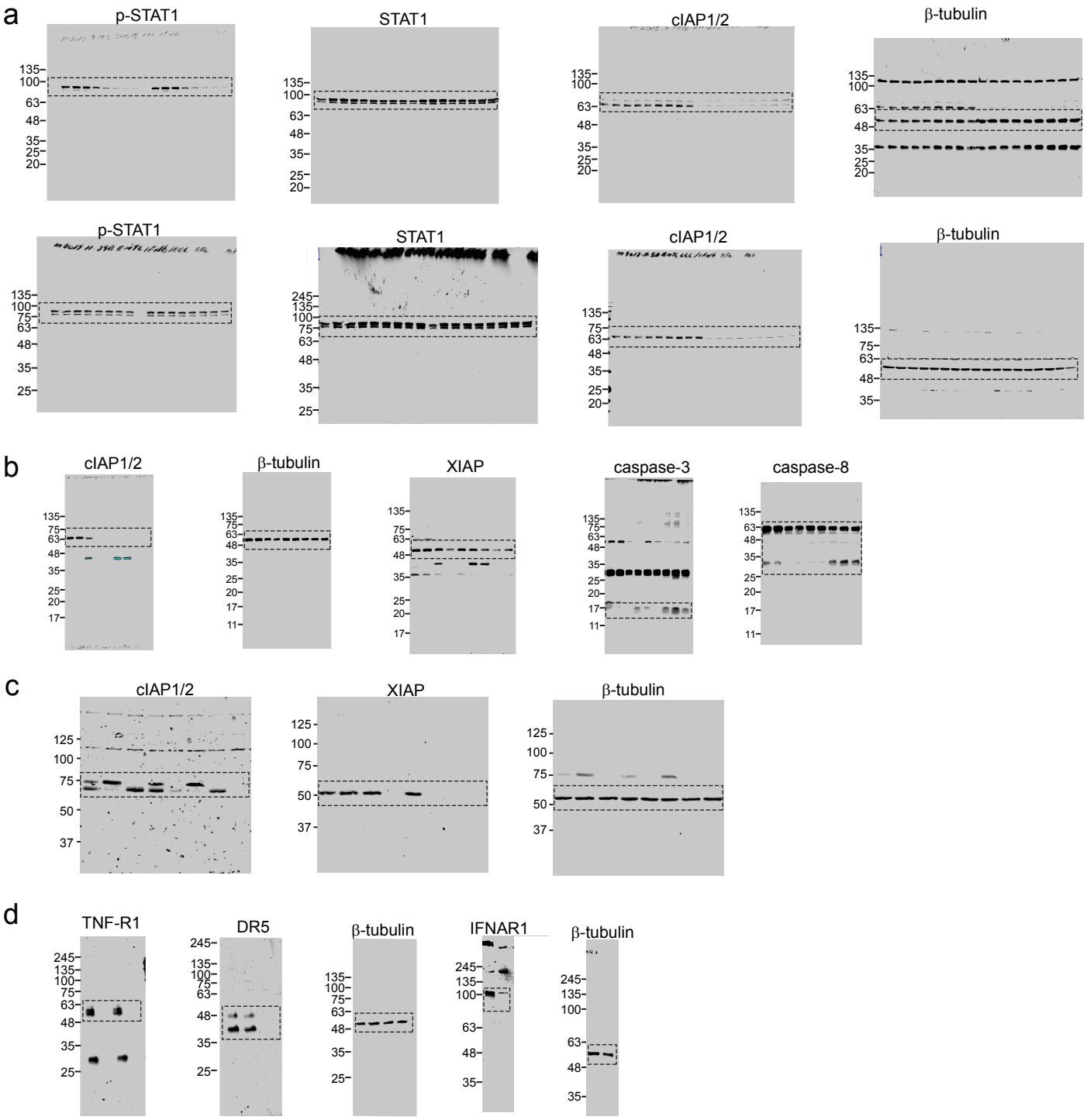
a, Immune subpopulations were sorted from splenocytes ($CD11b^+$ $F4/80^+$: macrophage; $CD11b^+$ $Gr1^+$: neutrophil; $CD11b^-$ $CD49b^+$: NK cell; $CD11b^-$ $CD49b^-$: non-myeloid cells) and were infected with 1 MOI of VSVΔ51 for 24 hr. Cell culture supernatants were applied to SMC-treated ETM6 cells for 24 hr and EMT6 viability was assessed by Alamar Blue. Error bars, mean ± s.d. **b,** Bone marrow derived macrophages were infected with VSVΔ51 and the supernatant was applied to EMT6 cells in the presence of 5 μM SMC, and viability was measured by Alamar blue. Error bars, mean ± s.d.

Supplementary Figure 18 Schematic of oncolytic virus-induced type I IFN and SMC synergy in bystander cancer cell death.



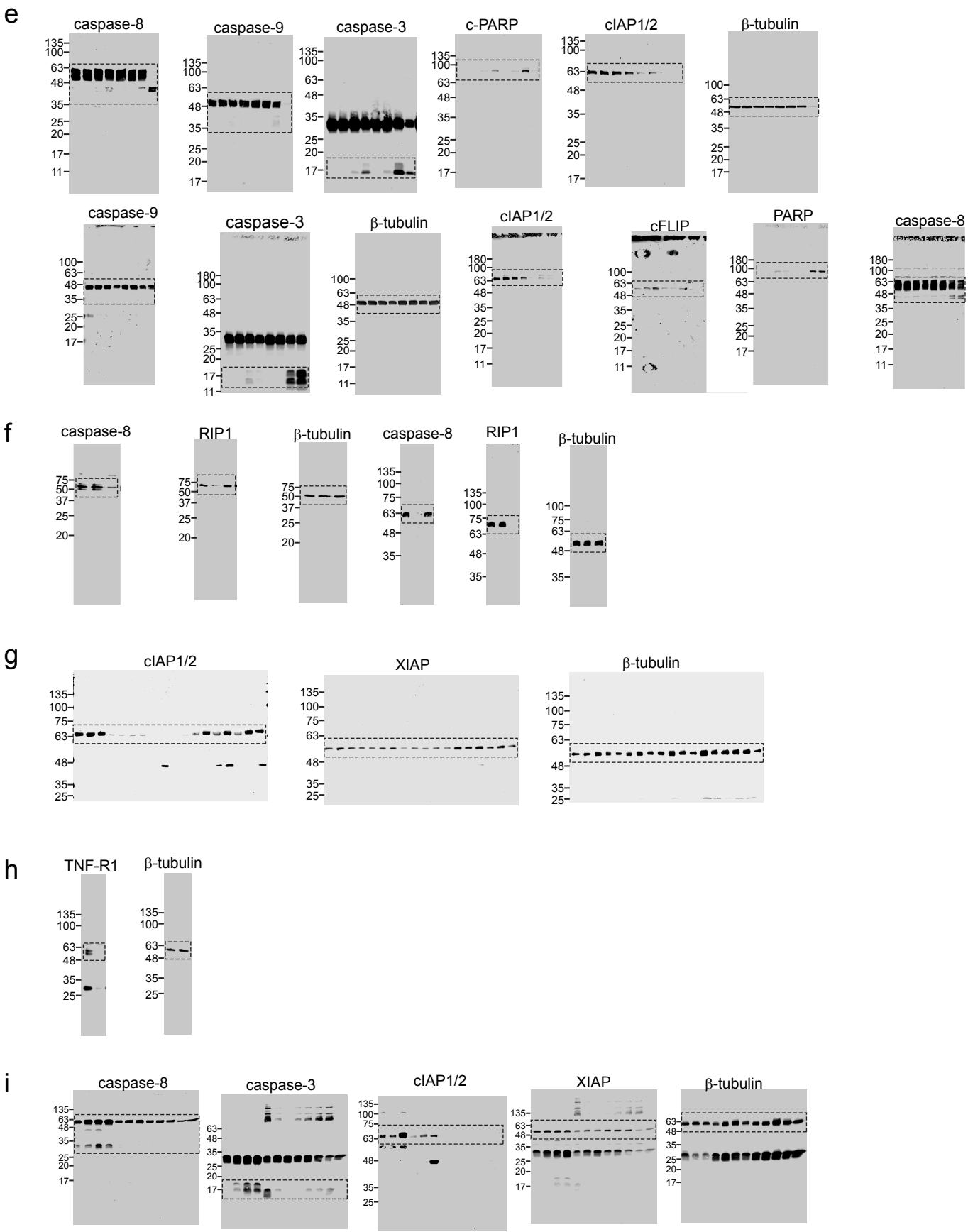
a, Virus infection in refractory cancer cells leads to the production of Type 1 IFN, which subsequently induces expression of IFN stimulated genes such as TRAIL. Type 1 IFN stimulation also leads to the NF-κB-dependent production of TNFα. IAP antagonism by SMC treatment leads to upregulation of TNFα and TRAIL expression and apoptosis of neighbouring tumour cells. **b,** Infection of a single tumour cell results in the activation of innate antiviral Type 1 IFN pathway, leading to the secretion of Type 1 IFNs onto neighbouring cells. The neighbouring cells also produce the profinflammatory cytokines TNFα and TRAIL. The singly infected cell undergoes oncolysis and the remainder of the tumour mass remains intact. On the other hand, neighbouring cells undergo bystander cell death due upon SMC treatment as a result of the SMC-mediated upregulation of TNFα/TRAIL and promotion of apoptosis upon proinflammatory cytokine activation.

Supplementary Figure 19 Full-length immunoblots



Full-length western blots pertaining to Fig. 2e **(a)**, Fig. 4e **(b)**, Supplementary Fig. 5b **(c)**, Supplementary Fig. 8 **(d)**, Supplementary Fig. 9a **(e)**, Supplementary Fig. 9g **(f)**, Supplementary Fig. 14 **(g)**, Supplementary Fig. 12 **(h)** and Supplementary Fig. 17 **(i)**.

Supplementary Figure 19 Full-length immunoblots



Full-length western blots pertaining to Fig. 2e **(a)**, Fig. 4e **(b)**, Supplementary Fig. 5b **(c)**, Supplementary Fig. 8 **(d)**, Supplementary Fig. 9a **(e)**, Supplementary Fig. 9g **(f)**, Supplementary Fig. 14 **(g)**, Supplementary Fig. 12 **(h)** and Supplementary Fig. 17 **(i)**.

Supplementary Table 1 List of interferon-stimulated genes affected by VSVΔ51 infection or IFNβ treatment in cancer cells. SNB75 cells were infected with 1 MOI of VSVΔ51 or treated with 250 U/mL IFNβ for 24 hr, and the cells were processed for RT-qPCR with primers targeting indicated genes (Cytokine Libraries I and II from realtimeprimers.com).

VSV	IFNb	Gene name	Gene ID
25465.4	**1017.8**	CCL8	Chemokine (C-C motif) ligand 8
13388.9	**44.9**	IL29	Interleukin 29 (interferon, lambda 1)
5629.3	**24.3**	IFNB1	Interferon, beta 1, fibroblast
1526.8	**16.2**	TNFSF15	Tumor necrosis factor (ligand) superfamily, member 15
847.0	**24.6**	CCL5	Chemokine (C-C motif) ligand 5
747.7	**17.2**	CCL3	Chemokine (C-C motif) ligand 3
650.9	**60.6**	TNFSF10	Tumor necrosis factor (ligand) superfamily, member 10
421.3	**296.1**	IL12A	Interleukin 12A
289.3	**10.7**	TNFSF18	Tumor necrosis factor (ligand) superfamily, member 18
255.3	**18.8**	CCL7	Chemokine (C-C motif) ligand 7
154.2	**19.2**	IL6	Interleukin 6 (interferon, beta 2)
150.8	**12.9**	IL1RN	Interleukin 1 receptor antagonist
108.1	**25.5**	CCL20	Chemokine (C-C motif) ligand 20
78.6	**6.2**	CXCL1	Chemokine (C-X-C motif) ligand 1
64.7	**14.8**	CCL2	Chemokine (C-C motif) ligand 2
62.5	**14.5**	CCL4	Chemokine (C-C motif) ligand 4
55.6	1.2	CXCL3	Chemokine (C-X-C motif) ligand 3
55.2	**4.3**	TNF	Tumor necrosis factor (TNF superfamily, member 2)
48.8	**4.3**	IGF1	Insulin-like growth factor 1 (somatomedin C)
48.4	2.8	CXCL2	Chemokine (C-X-C motif) ligand 2
38.5	**3.8**	CCL11	Chemokine (C-C motif) ligand 11
37.5	**3.8**	HGF	Hepatocyte growth factor
36.5	**75.1**	NGFB	Nerve growth factor, beta polypeptide
32.9	**4.0**	FGF14	Fibroblast growth factor 14
24.7	**25.6**	FGF20	Fibroblast growth factor 20
21.5	**16.4**	IL1B	Interleukin 1, beta
20.0	**36.3**	CSF2	Colony stimulating factor 2 (granulocyte-macrophage)
18.3	2.6	GDF3	Growth differentiation factor 3
17.2	2.0	CCL28	Chemokine (C-C motif) ligand 28
12.0	2.1	CCL22	Chemokine (C-C motif) ligand 22
11.3	2.5	CCL17	Chemokine (C-C motif) ligand 17
10.5	2.0	CCL13	Chemokine (C-C motif) ligand 13
10.5	**15.3**	IL20	Interleukin 20
9.7	**22.8**	FGF16	Fibroblast growth factor 16
8.8	**3.6**	TNFSF14	Tumor necrosis factor (ligand) superfamily, member 14
8.2	2.7	FGF2	Fibroblast growth factor 2 (basic)
7.1	**8.1**	BDNF	Brain-derived neurotrophic factor

7.1	**9.7**	IL1A	Interleukin 1, alpha
7.1	**10.9**	ANGPT4	Angiopoietin 4
7.0	1.5	TGFB3	Transforming growth factor, beta 3
7.0	**5.8**	IL22	Interleukin 22
6.9	**9.7**	IL1F5	Interleukin 1 family, member 5 (delta)
6.7	2.4	IFNW1	Interferon, omega 1
6.6	**12.6**	IL11	Interleukin 11
6.6	**25.1**	IL1F8	Interleukin 1 family, member 8 (eta)
6.3	-1.3	EDA	Ectodysplasin A
5.9	**8.0**	FGF5	Fibroblast growth factor 5
5.8	**5.0**	VEGFC	Vascular endothelial growth factor C
5.2	**4.9**	LIF	Leukemia inhibitory factor
5.0	1.3	CCL25	Chemokine (C-C motif) ligand 25
4.9	**8.3**	BMP3	Bone morphogenetic protein 3
4.9	1.6	IL17C	Interleukin 17C
4.8	-2.3	TNFSF7	CD70 molecule
4.3	2.5	TNFSF8	Tumor necrosis factor (ligand) superfamily, member 8
4.3	2.5	FASLG	Fas ligand (TNF superfamily, member 6)
4.2	2.7	BMP8B	Bone morphogenetic protein 8b
4.2	**6.0**	IL7	Interleukin 7
4.1	**5.2**	CCL24	Chemokine (C-C motif) ligand 24
4.0	-2.2	INHBE	Inhibin, beta E
4.0	**5.8**	IL23A	Interleukin 23, alpha subunit p19
3.8	-1.1	IL17F	Interleukin 17F
3.7	2.9	CCL21	Chemokine (C-C motif) ligand 21
3.5	**8.5**	CSF1	Colony stimulating factor 1 (macrophage)
3.5	3.0	IL15	Interleukin 15
3.4	**5.7**	NRG2	Neuregulin 2
3.3	N/A	INHBB	Inhibin, beta B
3.3	N/A	LTB	Lymphotoxin beta (TNF superfamily, member 3)
3.3	N/A	BMP7	Bone morphogenetic protein 7
3.0	-3.8	IL1F9	Interleukin 1 family, member 9
2.9	**6.1**	IL12B	Interleukin 12B
2.8	**6.2**	FLT3LG	Fms-related tyrosine kinase 3 ligand
2.7	**3.0**	FGF1	Fibroblast growth factor 1 (acidic)
2.5	-2.0	CXCL13	Chemokine (C-X-C motif) ligand 13
2.4	2.2	IL17B	Interleukin 17B
2.3	**7.8**	GDNF	Glial cell derived neurotrophic factor
2.3	-1.7	GDF7	Growth differentiation factor 7
2.3	-2.4	LTA	Lymphotoxin alpha (TNF superfamily, member 1)
2.2	1.7	LEFTY2	Left-right determination factor 2
2.1	**5.0**	FGF19	Fibroblast growth factor 19
2.1	**9.8**	FGF23	Fibroblast growth factor 23
2.1	**4.8**	CLC	Cardiotrophin-like cytokine factor 1

2.1	**3.0**	ANGPT1	Angiopoietin 1
2.0	**10.6**	TPO	Thyroid peroxidase
2.0	2.1	EFNA5	Ephrin-A5
1.9	**6.4**	IL1F10	Interleukin 1 family, member 10 (theta)
1.9	**7.6**	LEP	Leptin (obesity homolog, mouse)
1.8	**3.0**	IL5	Interleukin 5 (colony-stimulating factor, eosinophil)
1.8	**5.7**	IFNE1	Interferon epsilon 1
1.8	2.7	EGF	Epidermal growth factor (beta-urogastrone)
1.7	**3.4**	CTF1	Cardiotrophin 1
1.7	-1.9	BMP2	Bone morphogenetic protein 2
1.7	3.0	EFNB2	Ephrin-B2
1.6	1.0	FGF8	Fibroblast growth factor 8 (androgen-induced)
1.6	-2.0	TGFB2	Transforming growth factor, beta 2
1.5	-1.6	BMP8A	Bone morphogenetic protein 8a
1.5	**3.3**	NTF5	Neurotrophin 5 (neurotrophin 4/5)
1.5	1.0	GDF10	Growth differentiation factor 10
1.5	1.5	TNFSF13B	Tumor necrosis factor (ligand) superfamily, member 13b
1.5	2.5	IFNA1	Interferon, alpha 1
1.4	-1.3	INHBC	Inhibin, beta C
1.4	2.8	FGF7	Galactokinase 2
1.4	**3.3**	IL24	Interleukin 24
1.4	-1.1	CCL27	Chemokine (C-C motif) ligand 27
1.3	1.9	FGF13	Fibroblast growth factor 13
1.3	1.4	IFNK	Interferon, kappa
1.3	2.0	ANGPT2	Angiopoietin 2
1.3	**7.6**	IL18	Interleukin 18 (interferon-gamma-inducing factor)
1.3	**7.0**	NRG1	Neuregulin 1
1.3	**4.9**	NTF3	Neurotrophin 3
1.2	**15.0**	FGF10	Fibroblast growth factor 10
1.2	1.9	KITLG	KIT ligand
1.2	-1.3	IL17D	Interleukin 17D
1.2	1.1	TNFSF4	Tumor necrosis factor (ligand) superfamily, member 4
1.2	1.3	VEGFA	Vascular endothelial growth factor
1.1	2.4	FGF11	Fibroblast growth factor 11
1.1	-1.4	IL17E	Interleukin 17E
1.1	-2.1	TGFB1	Transforming growth factor, beta 1
1.0	**3.1**	GH1	Growth hormone 1
-1.0	**6.1**	IL9	Interleukin 9
-1.0	-2.5	EFNB3	Ephrin-B3
-1.0	1.8	VEGFB	Vascular endothelial growth factor B
-1.0	-1.2	IL1F7	Interleukin 1 family, member 7 (zeta)
-1.0	-2.1	GDF11	Growth differentiation factor 11
-1.1	1.3	ZFP91	Zinc finger protein 91 homolog (mouse)
-1.2	-1.1	BMP6	Bone morphogenetic protein 6

-1.2	-1.2	AMH	Anti-Mullerian hormone
-1.3	-1.0	LEFTY1	Left-right determination factor 1
-1.3	2.4	EFNA3	Ephrin-A3
-1.3	-1.3	LASS1	LAG1 longevity assurance homolog 1
-1.5	1.0	EFNA4	Ephrin-A4
-1.8	1.3	PDGFD	DNA-damage inducible protein 1
-1.8	1.8	IL10	Interleukin 10
-1.9	1.6	GDF5	Growth differentiation factor 5
-1.9	1.3	EFNA2	Ephrin-A2
-1.9	-1.5	EFNB1	Ephrin-B1
-1.9	-1.4	GDF8	Growth differentiation factor 8
-1.9	1.6	PDGFC	Platelet derived growth factor C
-2.2	2.4	TSLP	Thymic stromal lymphopoietin
-2.3	-1.5	BMP10	Bone morphogenetic protein 10
-2.4	-4.6	CXCL12	Chemokine (C-X-C motif) ligand 12
-2.5	**4.0**	IFNG	Interferon, gamma
-2.6	1.2	EPO	Erythropoietin
-2.7	-2.1	GAS6	Growth arrest-specific 6
-2.9	2.9	PRL	Prolactin
-2.9	-2.1	BMP4	Bone morphogenetic protein 4
-2.9	-5.7	INHA	Inhibin, alpha
-3.0	-1.3	GDF9	Growth differentiation factor 9
-3.1	-1.5	FGF18	Fibroblast growth factor 18
-3.2	N/A	IL17	Interleukin 17
-3.2	-1.1	IL26	Interleukin 26
-3.4	1.2	EFNA1	Ephrin-A1
-3.8	-1.1	FGF12	Fibroblast growth factor 12
-4.0	-2.3	FGF9	Fibroblast growth factor 9 (glia-activating factor)
-4.5	1.4	CCL26	Chemokine (C-C motif) ligand 26
-8.0	**9.7**	CCL19	Chemokine (C-C motif) ligand 19
N/A	N/A	BMP15	Bone morphogenetic protein 15
N/A	N/A	CCL15	Chemokine (C-C motif) ligand 14
N/A	N/A	CCL16	Chemokine (C-C motif) ligand 16
N/A	N/A	CCL18	Chemokine (C-C motif) ligand 18
N/A	N/A	CCL23	Chemokine (C-C motif) ligand 23
N/A	N/A	CD40LG	CD40 ligand (TNF superfamily)
N/A	N/A	CSF3	Colony stimulating factor 3 (granulocyte)
N/A	N/A	CXCL5	Chemokine (C-X-C motif) ligand 5
N/A	N/A	FGF4	Fibroblast growth factor 4
N/A	N/A	FGF6	Fibroblast growth factor 6
N/A	N/A	GH2	Growth hormone 2
N/A	N/A	IL2	Interleukin 2
N/A	N/A	IL21	Interleukin 21
N/A	N/A	IL28A	Interleukin 28A (interferon, lambda 2)

N/A	N/A	INHBA	Inhibin, beta A
N/A	N/A	NRG3	Neuregulin 3
N/A	N/A	TNFSF11	Tumor necrosis factor (ligand) superfamily, member 11
N/A	N/A	TNFSF13	Tumor necrosis factor (ligand) superfamily, member 13
N/A	**6.5**	NRG4	Neuregulin 4
N/A	**6.1**	IL3	Interleukin 3 (colony-stimulating factor, multiple)
N/A	1.8	TNFSF9	Tumor necrosis factor (ligand) superfamily, member 9

Total cytokines and chemokines from the cytokine library = 176

Number of genes with > 3-fold upregulation compared to control

VSVΔ51: 69

IFNβ: 70

Overlap: 44

Supplementary Table 2 Catalogue numbers of primers used for RT-qPCR. Primers were obtained from realtimeprimers.com.

Catalogue	Species	Gene
MHK-1	Mouse	Mouse Housekeeping Gene Primer Set
VMPS-3027	Mouse	IFNb
VMPS-3154	Mouse	IRF1
VMPS-3157	Mouse	IRF3
CMPS-1 (Mm.3233)	Mouse	IRF7
VMPS-4035	Mouse	MX1
VMPS-4036	Mouse	MX2
VMPS-3019	Mouse	IFIT1
CMPS-1 (Mm.233471)	Mouse	OAS1
CMPS-1 (Mm.228363)	Mouse	OASL
HHK-1	Human	Human Housekeeping Gene Primer Set
VHPS-4476	Human	IFNb
VHPS-4626	Human	IRF1
VHPS-4629	Human	IRF3
CHPS-1 (Hs.166120)	Human	IRF7
VHPS-5959	Human	MX1
VHPS-5960	Human	MX2
VHPS-4465	Human	IFIT1
VHPS-6421	Human	OAS1
VHPS-6424	Human	OASL
VHPS-9415	Human	TNFa
VHPS-9439	Human	TRAIL
VHPS-4531	Human	1L1b