**Supplementary Materials and Methods**

**Construction of SFV-derived vectors**

To generate the XCL1-sFlt3L construct, the coding sequence for soluble Flt3L was amplified by PCR from its expression plasmid (mFlex, Aldevron, Fargo, ND) and coding sequences for the autocatalytic peptide 2A from foot and mouth disease virus and a furin binding site were added upstream of the protein-coding region, together with Mlu I restriction sites for cloning onto a mouse XCL1 expression plasmid (MR200473, Origene, Rockville, MD). The amplified product was isolated, digested with Mlu I and cloned into the MR200473 vector, downstream of the XCL1-coding region and without altering the translation reading frame. The accuracy of the cloning process was verified by DNA band analysis following enzymatic digestion with Sac I and by sequencing of the region surrounding the insertion site. XCL1, sFlt3L and XCL1-Flt3L were amplified by PCR and had Xma I target sites added at both 5’ and 3’ regions. All three PCR products were digested with Xma I for insertion into the pSFV-b12a vector backbone (1), which includes genes for the viral replicase. Clones that were demonstrated to be correctly inserted as assessed by digestion [sFlt3L: Nhe I; XCL1: EcoR V.HF-Msc I; XF: Nhe I] and sequencing were selected and amplified. The plasmid vector for SFV-LacZ (pSFV-enhLacZ )has been previously reported (2). mRNAs were produced in vitro from the transgene-coding and two helper plasmids coding the viral structural proteins, as previously described (3) Viral particles were produced by co-electroporation of transgene-coding and helper mRNAs into BHK cells. Electroporated cells were incubated for 48 h at 33ºC in GMEM BHK-21 medium. Debris was cleared from the supernatant by centrifugation at 2,000 g. The cleared supernatants were ultracentrifuged at 160,000 g using a SW40Ti rotor (Beckman Coulter) and resuspended in Tris-NaCl buffer, aliquoted and immediately frozen in liquid N2. Aliquots were kept at -80ºC until used. The generated vectors were titrated by immunofluorescent detection of viral replicase on BHK cell monolayers infected by serially diluted SFV particles in MEM-0.2% BSA (infection medium), followed by an overnight culture in GMEM BHK-21 medium for protein expression. An in-house anti-replicase rabbit polyclonal antibody was used for staining to demonstrate viral infection.

**mRNA quantitative analysis**

BHK, MC38 or B16-OVA cells were cultured on 6-well culture plates to confluence. Infection was carried out using 3 x 107 SFV particles, and cells were allowed an overnight incubation to ensure transgene expression. RNA was extracted from cell suspensions using the RNAeasy kit (Qiagen, Hilden, Germany) and according to the manufacturer’s instructions and cDNA was generated. We designed primers to amplify the coding sequences for mouse sFlt3L (FW TGTGGCAGGGTCTAAGATGC; RV CTTCTAGGGCTATGGGACTCC), XCL1 (FW TAGCTGTGTGAACTTACAAACCC; RV ACAGTCTTGATCGCTGCTTTC), β-actin (FW AGCCTCGCCTTTGCCGA; RV CTGGTGCCTGGGGCG), and the viral replicase (FW GACGCGTCGTCAGCCAGGG; RV ccacgacccctgcacctgc). The generated cDNAs were amplified by real-time PCR (BioRad, Hercules, CA) and results were analyzed using CFX manager software.

For *in vivo* RNA extraction, MC38 tumors were established and 108 SFV particles were administered intratumorally when tumors reached an approximate size of 25 mm2. 24h later, tumor single cell suspensions were generated by 15-minute collagenase/DNAse digestion and mechanical disruption. mRNA was extracted from cell suspensions using the RNAeasy kit, cDNA was generated and Flt3L, XCL1, β-actin and the viral replicase were amplified and analyzed by real-time PCR (BioRad iQ5).

**Western Blotting**

Infection and incubation of BHK cells were performed as described above. After trypsinization, cells were lysed in RIPA buffer in the presence of a protease inhibitor (Complete, Roche, Basel, Switzerland) and the lysate protein concentration was quantified by BCA (Thermo Fisher Scientific, Waltham, MA). The lysate was boiled for 5 minutes in β-mercaptoethanol-containing loading buffer. Electrophoresis on polyacrylamide gel was carried out and proteins were transferred to PVDF membranes. Membranes were blocked with TBS-5% skimmed milk and stained with primary antibodies against mouse Flt3L (R&D AF427) or XCL1 (R&D AF486), followed by secondary staining with HRP-conjugated Goat Anti-Rat IgG (Pierce, Appleton, WI). SuperSignal™ Femto Substrate (Thermo Scientific) was used for detection. After detection, membranes were washed with azide-containing TBS buffer and re-stained with anti-mouse β-actin (Sigma, St. Louis, MO). Secondary staining was carried out with HRP-conjugated Goat Anti-Rabbit IgG (BioRad) and Pierce™ ECL Western Blotting Substrate (Thermo Scientific) was used for detection.

**Tissue Processing and Flow cytometry**

Excised tumors and tumor-draining lymph nodes were incubated in collagenase/DNAse for 30 minutes at 37ºC, followed by mechanical disaggregation and filtering through a 70-µm cell strainer (Thermo Fisher Scientific). Single-cell suspensions were then stained for flow cytometry. The fluorochrome-tagged mAbs used are listed in Supplementary Table 1. For identification of epitope-specific T cells, phycoerythrin-conjugated H-2Kb-OVA257-264 tetramer (MBL, Woburn, MA) or H-2Kb-KSPWFTTL pentamer (gp70, Proimmune) were used. For intranuclear staining, cells were fixed and permeabilized using the TrueNuclear transcription factor staining kit (Biolegend, San Diego, CA) and then stained according to manufacturer’s instructions.

Where indicated, OT-II CD4 T cells were purified using CD4 magnetic beads (Miltenyi), labeled with 5 µM Violet Proliferation Dye (BD) and 5 x 106 cells injected into the tail vein of tumor-bearing mice. For cytokine staining, 250 µg BrefeldinA (Sigma) were administered to tumor-bearing mice 90 minutes before TDLN extraction. TDLN cell suspensions were restimulated for 4 hours with PMA (100 ng/ml) plus ionomycin (1 µg/ml) in the presence of GolgiPlug (BD), followed by extracellular staining, incubation with Cytofix/Cytoperm buffer (BD) and intracellular staining. Flow cytometry was performed using FACS Canto II (BD Biosciences, Franklin Lakes, NJ) or CytoFLEX (Beckman Coulter) equipment.

The following antibodies were used:

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| **Reagent**  | **Source (mAb clone)** |
| Zombie NIR | Biolegend |
| SAV-APCAF750 | Invitrogen |
| Cell Proliferation Dye eFluor 450 | Thermo Fisher |
| FITC B220 | Biolegend (RA3-6B2) |
| APC CD11b  | Biolegend (M1/70) |
| FITC CD11b | Biolegend (M1/70) |
| BUV395 CD11b | BD Horizon (M1/70) |
| PE CD11c | Biolegend (N418) |
| APC CD11c | Biolegend (N418) |
| BV510 CD11c | Pharmingen (HL3) |
| APCR700 CD11c | BD Horizon (N418) |
| FITC CD25 | Pharmingen (7D4) |
| PE Foxp3 | eBioscience (FJK-16S) |
| PrCPCy5.5 Foxp3 | Invitrogen (FJK-16S) |
| APC CD3 | Biolegend (145-2C11) |
| PEC7 CD45 | Biolegend (30-F11) |
| BUV661 CD45 | BD Horizon (30-F11) |
| BV421 CD4 | Biolegend (RM4-5) |
| PrCPCy5.5 Vα2 TCR | BD Pharmingen (B20.1) |
| BV510 CD8 | Biolegend (53-6.7) |
| BUV395 CD8 | BD Horizon (53-6.7) |
| FITC IAb | Pharmingen (AF6-120.1) |
| Biotin IAb | Pharmingen (KH74) |
| PE Gr1 | Biolegend (RB6-8C5) |
| BV421 F4/80 | Biolegend (BM8) |
| PrCPCy5.5 CD103 | Biolegend (2E7) |
| PE CD103 | Biolegend (2E7) |
| APC XCR1 | Biolegend (ZET) |
| APC CD137 | Biolegend (17B5) |
| BV785 PD-1 | Biolegend (29F.1A12) |
| BV650 LAG-3 | Biolegend (C9B7W) |
| AF488 Ki67 | BD Pharmingen (B56) |
| APCR700 CTLA-4 | BD Horizon (UC10-4F10-11) |
| BV605 IFN-γ | Biolegend (XMG1.2) |
| PE IL-10 | Biolegend (JES5-16E3) |
| APC IL-17 | eBiosciences (eBio17B7) |

**Software and statistical analyses**

Flow cytometry data were analyzed using FlowJo (BD Biosciences) or Cytexpert (Beckman Coulter) software. Statistics on tumor growth data were analyzed with Prism software (GraphPad Software, La Jolla, CA). Mean diameters of tumors over time were fitted using the formula *y = A x e (t-t0)/(1 + e(t-t0)/B)*, where t represents time, A the maximum size reached by the tumor, and B its growth rate. Treatments were compared using the extra sum-of-squares F test (4). Tumor survival was compared with log-rank (Mantel-Cox) tests. Unless specified otherwise, all other analyses between groups were performed using unpaired Mann-Whitney tests. Unless specified otherwise, graphs depict mean ± SEM.

**REFERENCES**

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