Legends to supplemental figures:

Supplemental Fig.1. Expression of TLR3 by tumor cell lines in vitro.
Immunostaining with anti-TLR3 mAb (T3.7C3, rat IgG1, 10 ug/ml, eBioSciences, France) or an isotype control antibody after cytospin of ex vivo expanded B16F10 and GL26 cells in culture medium in the presence or absence of type I IFN. A representative micrograph picture is shown at a ×20 power magnitude for each antibody.

Supplemental Fig. 2. Cytostatic effects of various compounds on B16-OVA in vitro.
A. Schedule of the in vitro treatment of cells. B. B16-OVA were seeded at 0.5×10^5/ml in triplicates in 24 well plates and were treated as indicated in A. The experiments were performed twice with similar results. Proliferation index was calculated as the ratio between absolute numbers of harvested and seeded cells. The means±SEM of proliferation index are shown for two experiments (non parametric test, Mann-Whitney). *p<0.05, **p<0.01, ***p<0.001.

Supplemental Fig. 3. The OVA-CpG vaccine elicited OVA-specific T cell responses.
3×10^5 B16-OVA cells were injected s.c. in the left thigh at day 0. OVA plus CpG ODN 1668 was injected in the right footpad at day 4. At day 9, the tumor and vaccine DLNs (inguinal plus popliteal) were harvested and the cells were restimulated in vitro at 1.2 millions/ml with OVA 1 mg/ml or PBS for 48 hrs in triplicate wells. The levels of IFN-γ were measured in the supernatants by ELISA. The graph depicts the results for four mice per group. N=4/group.*p<0.05.
Supplemental Fig. 4. Synergistic effects of type I IFN and poly(A:U) on CCL5 release by human tumor cells in vitro.

Four human primary breast cancer cell cultures were seeded at 2×10^5/ml in duplicate 24 well plates, treated with rhuIFNα2b at 1000 IU/ml for 18 hrs and then exposed to medium or poly(A:U). The supernatants were harvested 48 hours later to dose CCL5 production by commercial ELISA.

Supplemental Fig. 5. TRIF knockdown efficiently abrogate chemkine production by GL26 in vitro.

GL26 were transfected with lentiviruses to knockdown TRIF or Lamin expression. After poly(A:U) treatment, CCL5 and CXCL10 production by transfected cells and their parental cell line were monitored by ELISA.

Supplemental Fig. 6. ShCCL5 efficiently abrogated the production of CCL5 by B16-OVA.

B16OVA were transfected with lentiviruses encoding shRNA targeting either CCL5 or Lamin. CCL5 (left panel) (and as a control, CXCL10 (right panel)) production at before and after stimulation with poly (A:U) was monitored.