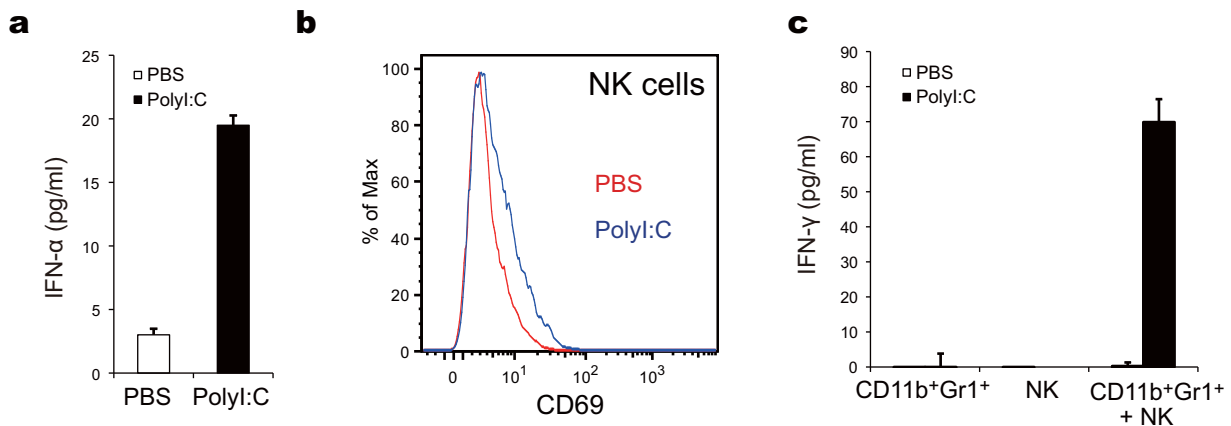
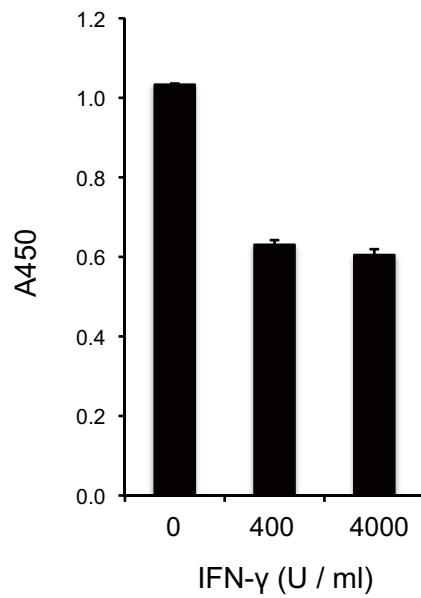


Supplementary Fig. 1. CD11b⁺Gr1⁺ cells of B16 tumor-bearing mice do not inhibit IFN- γ production by NK cells. NK cells (2×10^5) from naïve WT mice were co-cultured with or without CD11b⁺Gr1⁺ cells (2×10^5) from spleen of B16 tumor-bearing mice. Cells were incubated with 50 ng/ml PMA and 2 μ g/ml ionomycin for 24 hours. IFN- γ concentration in conditioned medium was determined.

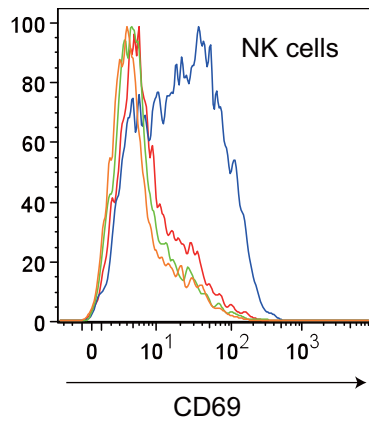


Supplementary Fig. 2. CD11b⁺Gr1⁺ cells of EL4 tumor-bearing mice activate NK cells after polyI:C. EL4 thymoma cells (1×10^6) were implanted sc into B6 WT mice. After tumor formation, 200 μ g polyI:C or PBS was injected i.p., CD11b⁺Gr1⁺ cells were isolated from spleen and co-cultured with naïve NK cells. IFN- α production from CD11b⁺Gr1⁺ cells, and NK cell CD69 expression and IFN- γ production was determined.

B16 cells



Supplementary Fig. 3. Inhibition of B16 cell proliferation by IFN-γ. B16D8 cells were seeded at 2×10^4 cells / 100 μ l in 96-well microplate and cultured for 24 hours in the presence of varying amounts of recombinant mouse IFN-γ (Biolegend). Cell proliferation was determined by standard XTT assay.

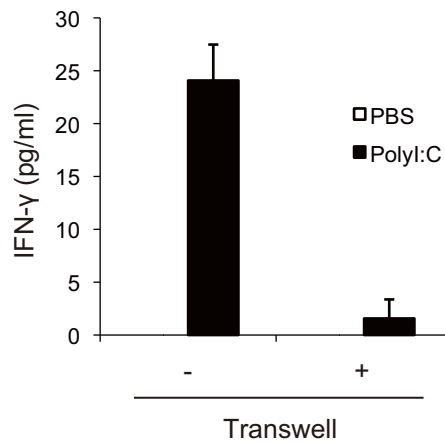
a

PBS, transwell -

PBS, transwell +

PolyI:C, transwell -

PolyI:C, transwell +

b

+

Supplementary Fig. 4. Abrogation of NK cell activation by CD11b⁺Gr1⁺ cells in a transwell system.

CD11b⁺Gr1⁺ cells were isolated from B16 tumor-bearing mice treated with polyI:C or PBS. A 0.4 μm transwell was inserted between CD11b⁺Gr1⁺ cells (2x10⁵) and NK cells (2x10⁵). CD69 expression on NK1.1⁺CD3ε⁻ (**a**) and IFN-γ concentration in conditioned medium (**b**) was determined after the culturing for 24 hours.