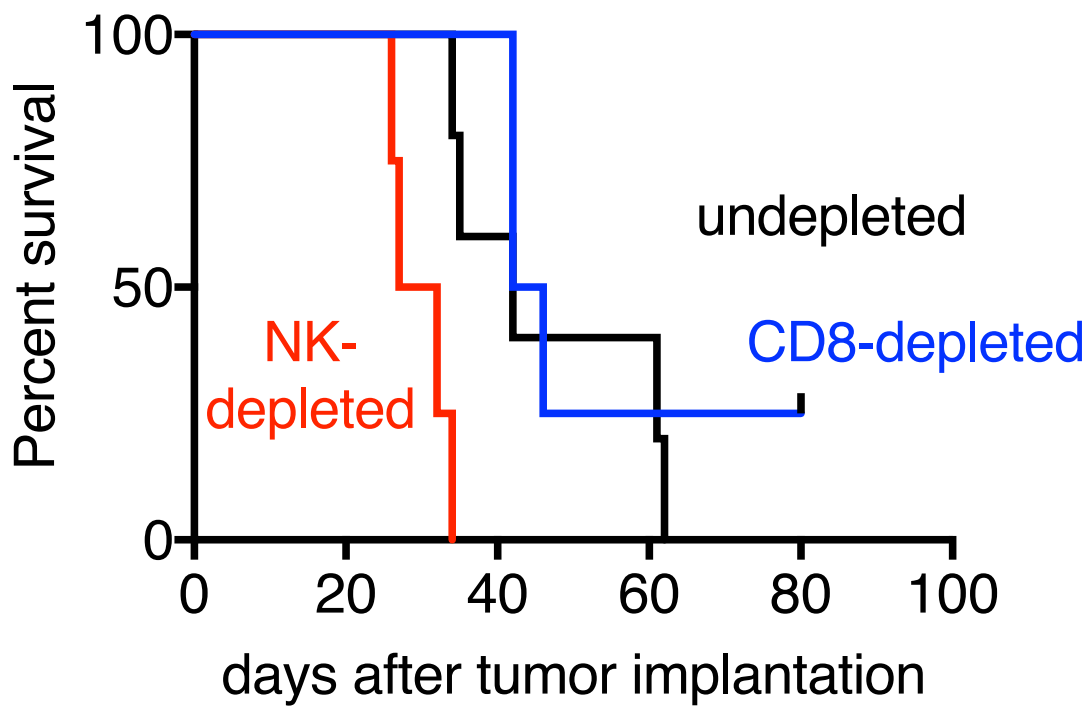
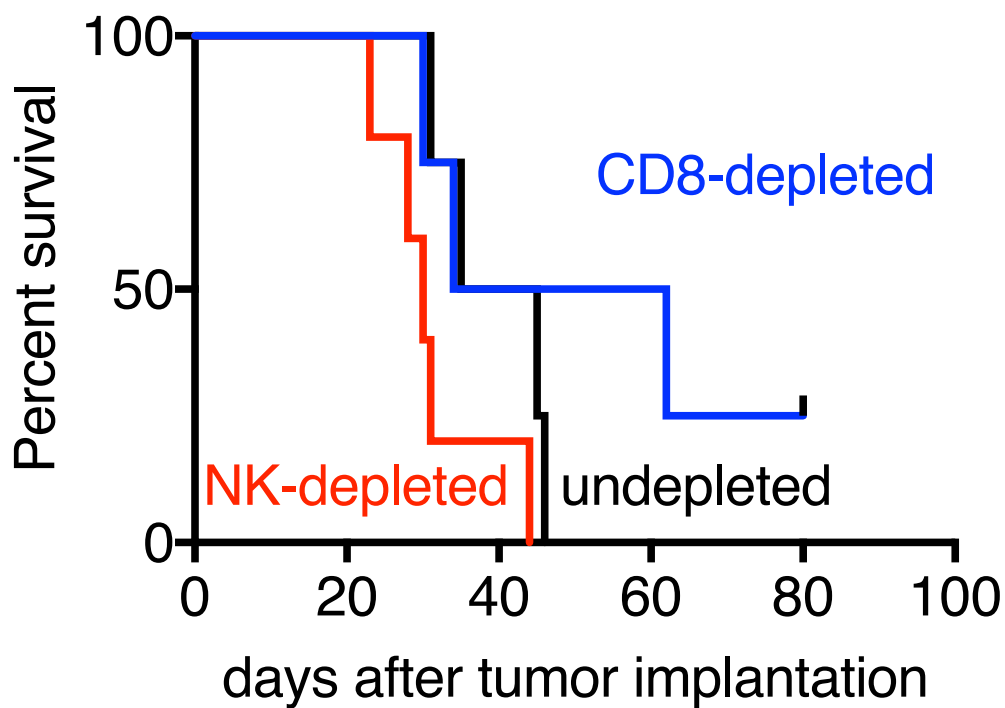


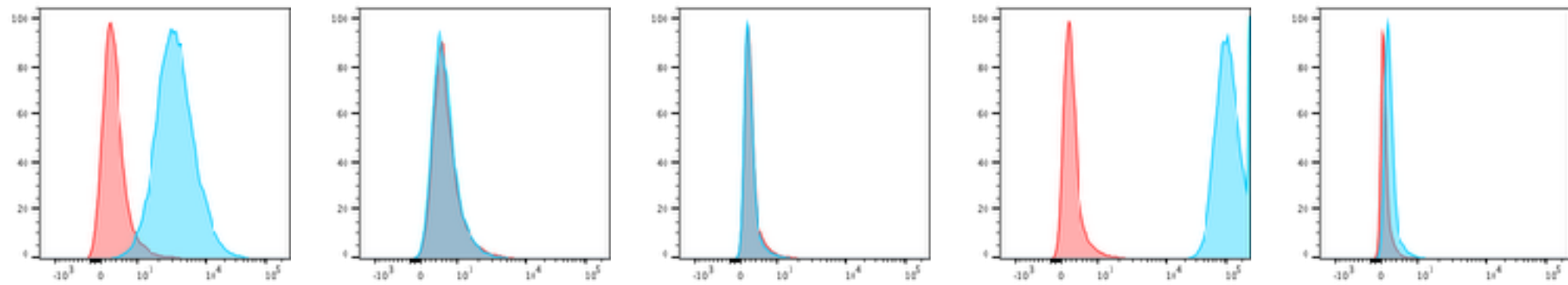
B16



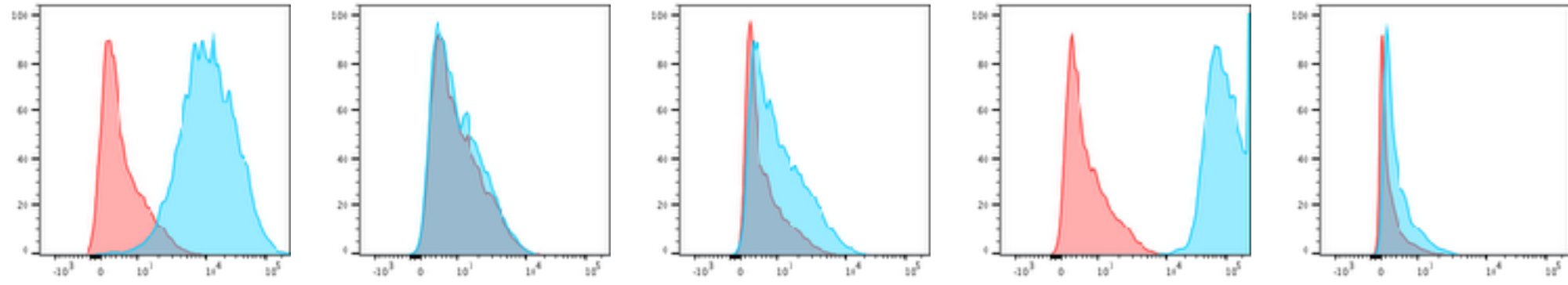
B16-*Pdl1*



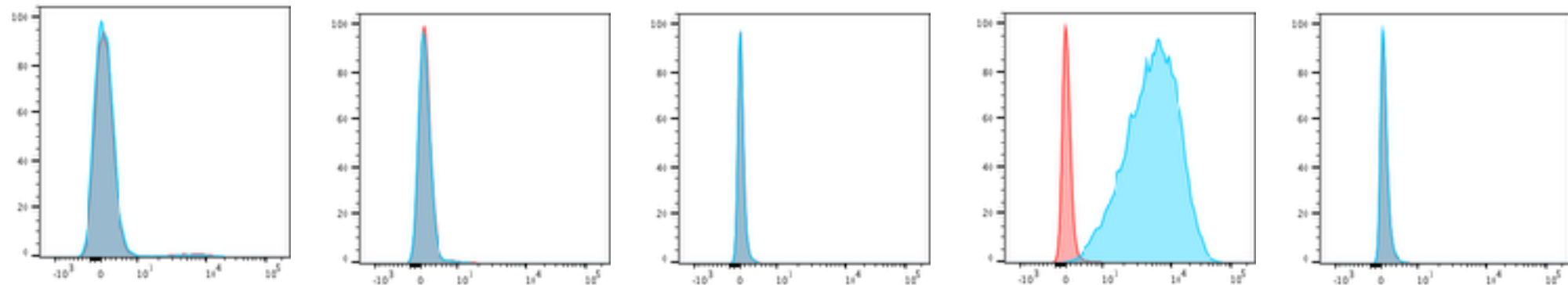
CT26



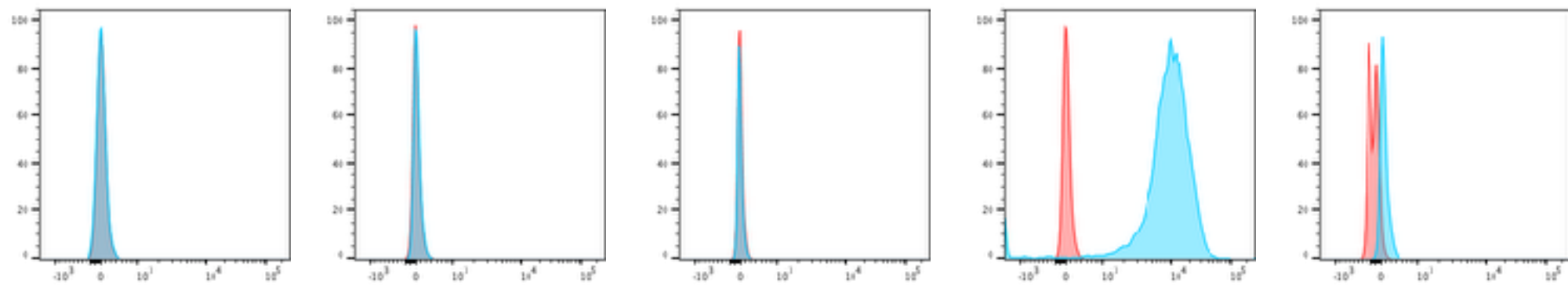
4T1



RMA-S



B16



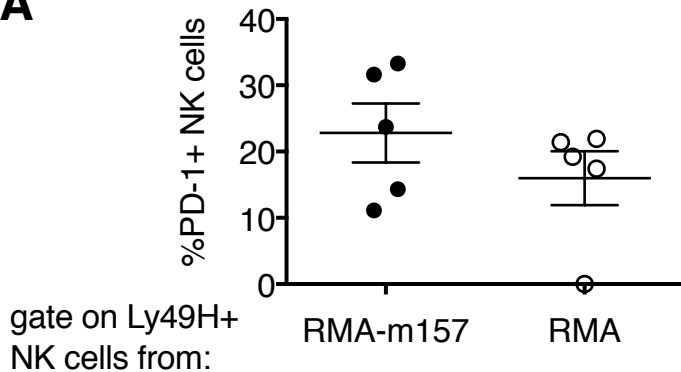
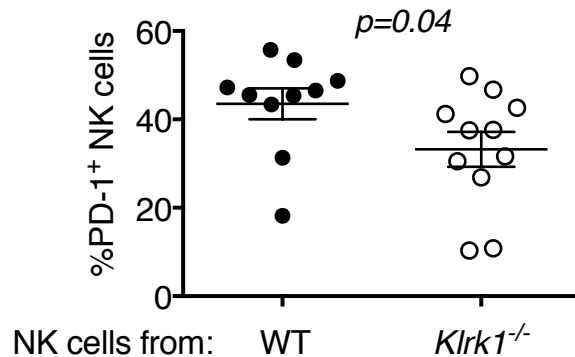
H60

Mult1

Rae1

PVR

Nectin-2

A**B**

Supplementary Figure 3

Supplementary Figure 1: In vivo establishment of B16 tumors is delayed by NK cells but not CD8 T cells.

Kaplan-Meier analyses of C57BL/6 mice injected i.v. with 20,000 B16 or B16-*Pdl1* cells. Some mice were depleted of CD8 and NK cells. n=4-5/group.

Supplementary Figure 2: Expression of NK-activating ligands on tumor cell lines.

CT26, 4T1, RMA-S and B16 cells were stained with control isotypes (red histograms) or antibodies specific for H60, MULT1, pan-RAE-1, PVR or Nectin-2 (blue lines). Data are representative of two experiments performed with similar outcomes.

Supplementary Figure 3: Engagement of activating receptors by tumor cells does not induce higher PD-1 expression on tumor-infiltrating NK cells.

(A) NK cells from RMA or RMA-*m157* s.c. tumors were stained for Ly49H and PD-1, and PD-1 expression was analyzed on Ly49H⁺ NK cells. **(B)** NK cells from RMA-RAE-1 s.c. tumors in wildtype mice or *Klrk1*^{-/-} mice (lacking expression of NKG2D) were stained with PD-1 antibody. A is representative of two experiments performed. In B, two experiments were combined. In A, n=5/group; in B, n=11/group. In A, statistical analyses with two-tailed unpaired Student's t-tests; in B with Mann-Whitney test.

Supplemental Data.

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