SUPPLEMENTAL MATERIAL

Iguchi-Manaka et al., http://www.jem.org/cgi/content/full/jem.20081611/DC1

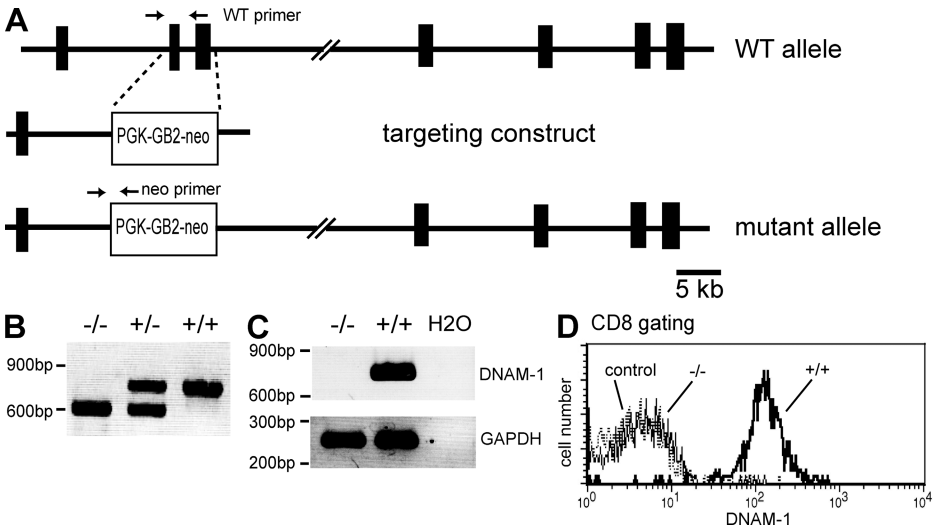

Figure S1. Generation of DNAM-1–deficient mice. (A) The DNAM-1 targeting vector is shown. (B) The genomic DNA from the tails of *Cd226*$^{+/+}$, *Cd226*$^{+/-}$, or *Cd226*$^{-/-}$ mice were analyzed for WT and mutant alleles by PCR. (C) Spleen cells from *Cd226*$^{+/+}$ or *Cd226*$^{-/-}$ mice were subjected to RT-PCR for *DNAM-1* and *GAPDH*. (D) Spleen cells from *Cd226*$^{+/+}$ or *Cd226*$^{-/-}$ mice were stained with FITC-conjugated anti-CD8 and biotin-conjugated anti–DNAM-1 (TX42), followed by PE-conjugated streptavidin, and analyzed by flow cytometry.

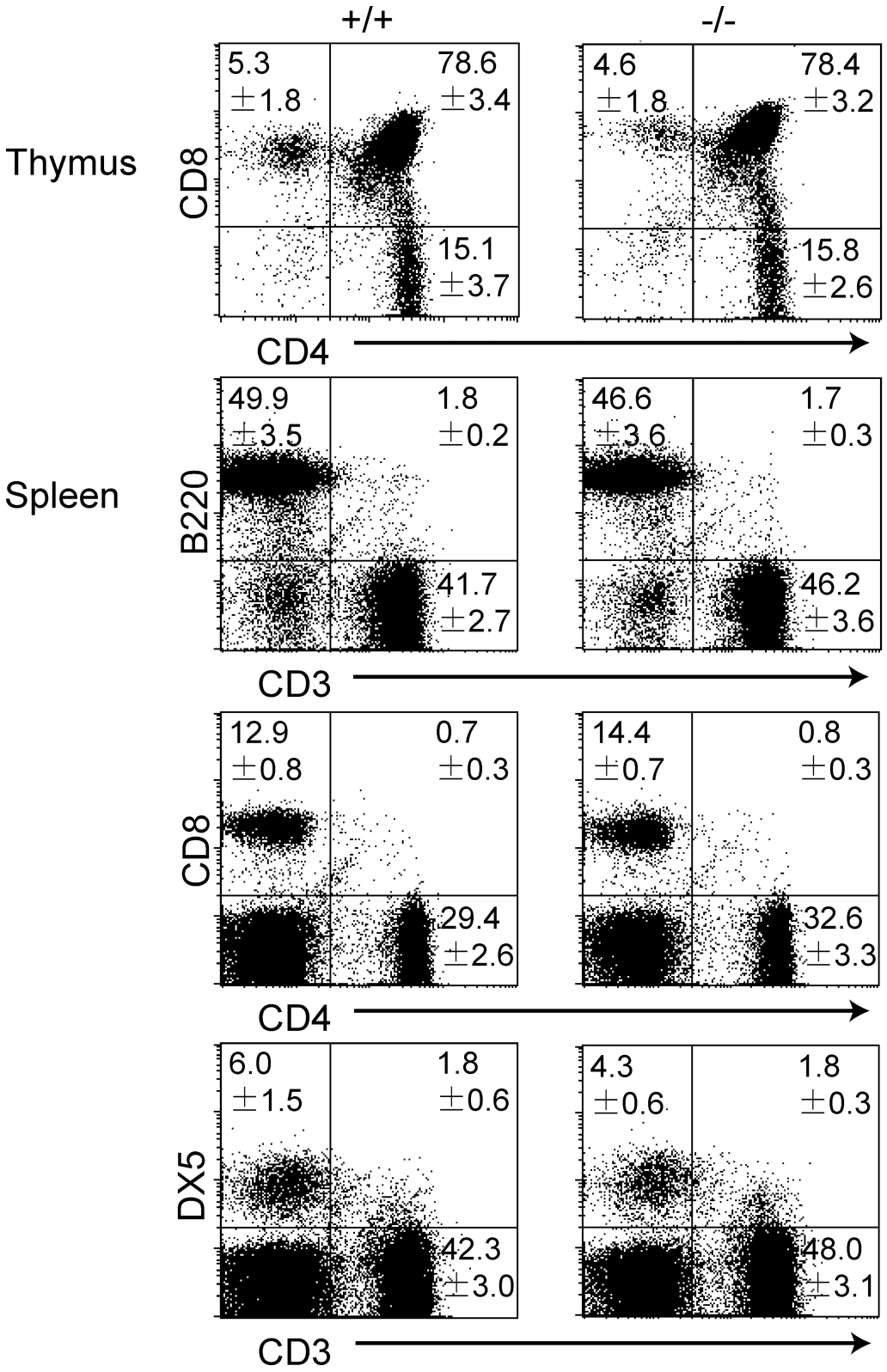

Figure S2. Lymphocytes population in DNAM-1–deficient mice. The thymus and spleen cells derived from WT (n = 5) and DNAM-1–deficient (n = 5) mice were stained with FITC- or PE-labeled mAbs indicated and analyzed by flow cytometry.

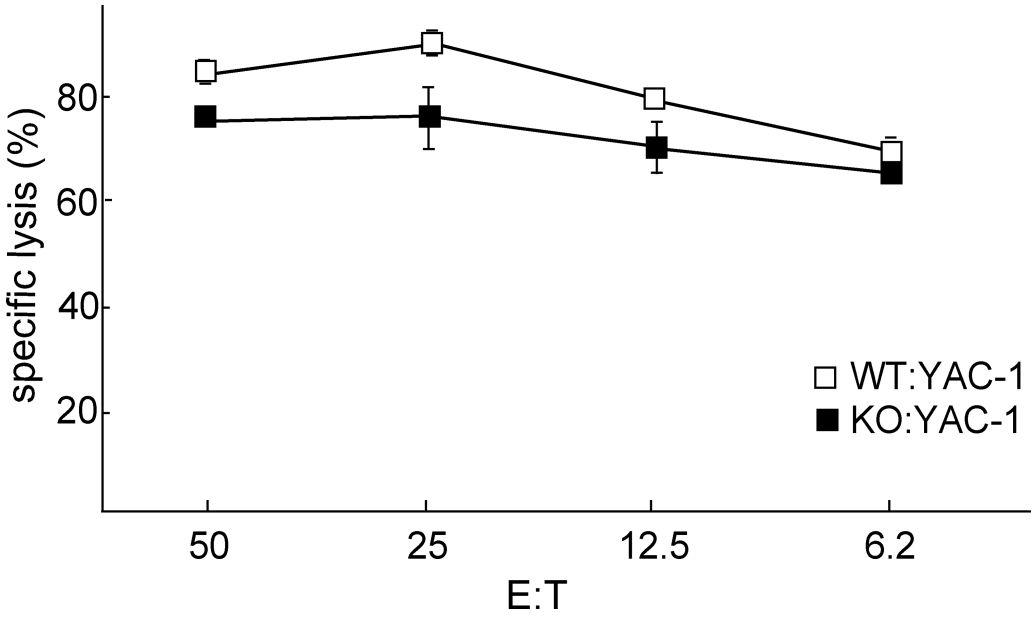

Figure S3. Cytotoxicities of Yac-1 by WT and DNM-1–deficient NK cells. NK cells derived from WT and DNAM-1–deficient mice were cocultured with ^{51}Cr-labeled Yac-1 cells for 3 h. Culture supernatants were harvested and ^{51}Cr releases were counted. Error bars show SD.