

CRISPR/Cas9-Mediated Deletion of *Foxn1* in NOD/SCID/IL2rg^{-/-} Mice Results in Severe

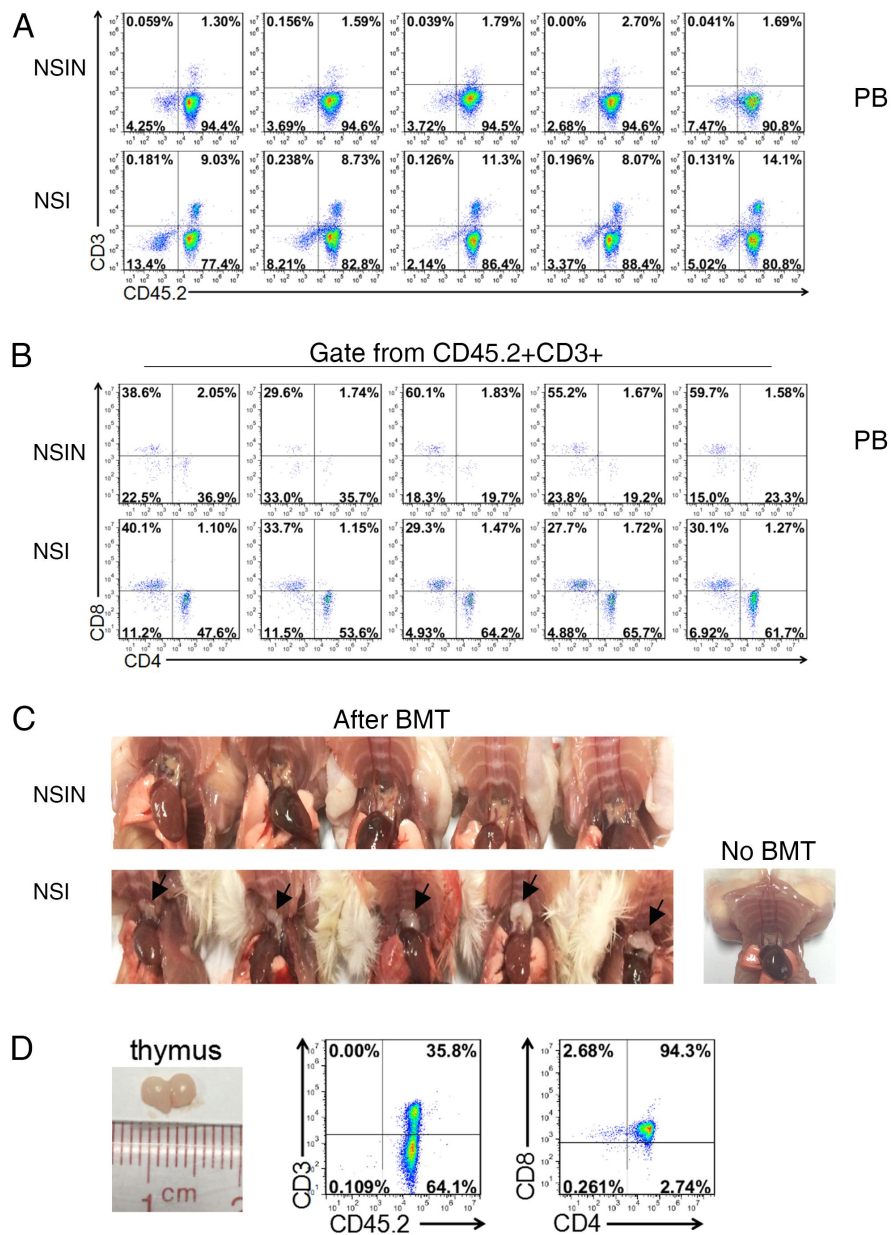
Immunodeficiency

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Supplementary Figure 1. NSIN mice are more resistant to allogeneic T cell development than NSI mice. (A) Donor BMNC reconstitution in the T (CD45.2⁺ CD3⁺) cell lineage in the PB of NSIN (upper) and NSI (lower) mice (n=5 for each group) 4 weeks after bone marrow transplantation (BMT). (B) Donor T cells were analyzed for CD4 and CD8 expression by flow cytometry. (C) Loss of *Foxn1* inhibited the regeneration of thymi after BMT in NSIN mice. (D) Phenotype of cells from the regenerated thymi in NSI mice.

Supplementary Figure 2. Representative FACS analysis of Nalm6-GL cells in NOG, NSI, and NSIN mice. (A–C) Percentages of Nalm6-GL cells in the PB, SP, and BM of mice intravenously injected with (A) low (1×10^4), (B) medium (1×10^5), and (C) high (1×10^6) numbers of Nalm6-GL cells. GFP⁺ cells represent Nalm6-GL cells.

Supplementary Figure 1



Supplementary Figure 2

