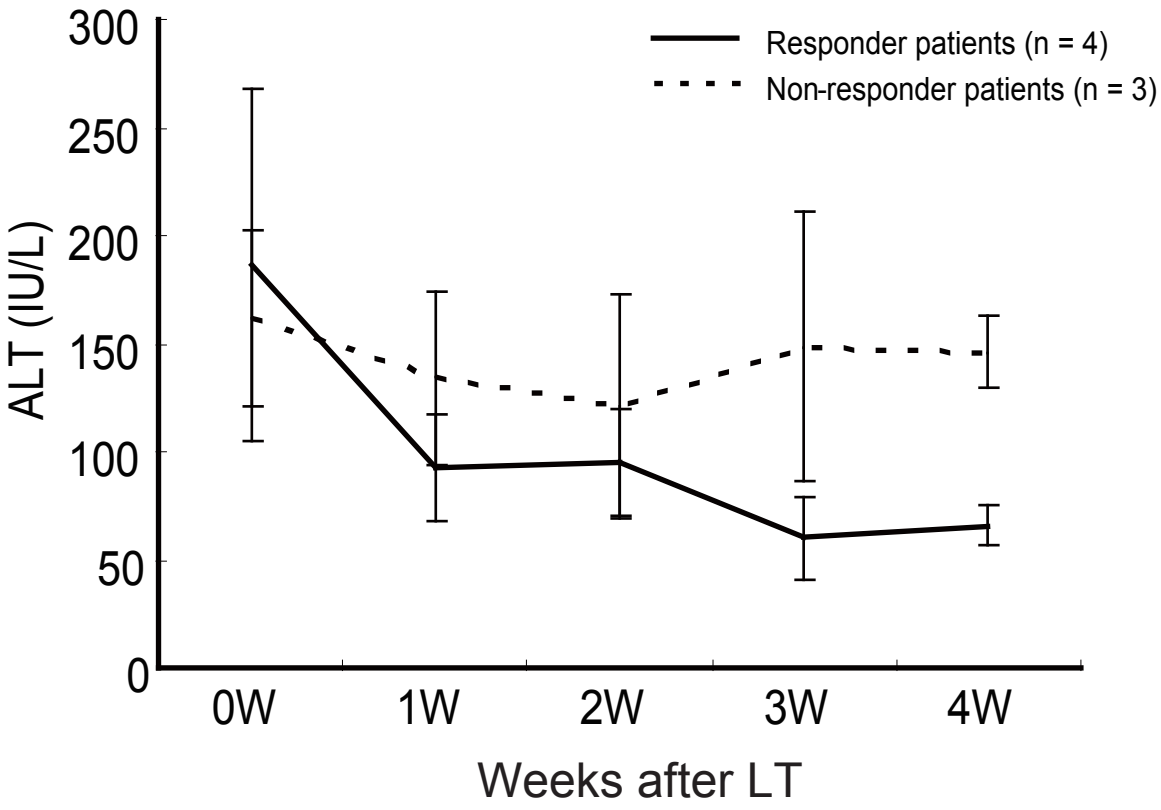
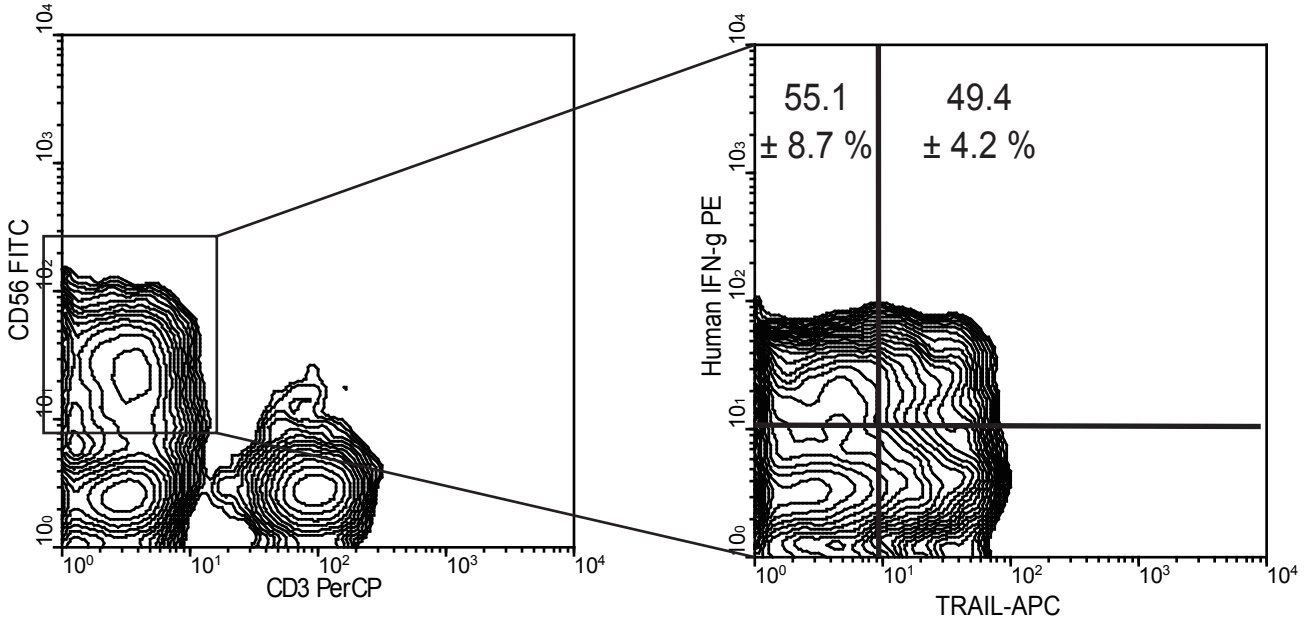


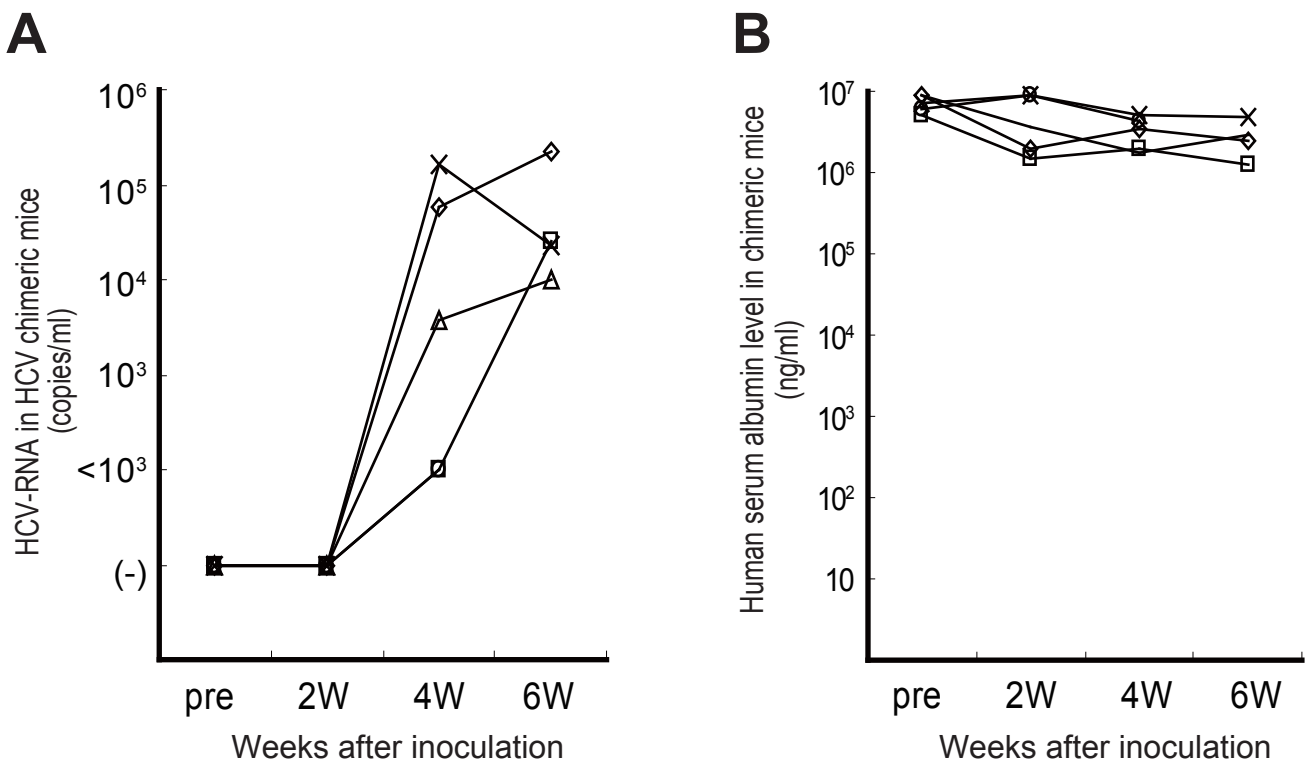

**Supplemental Figure 1**

Serial changes in serum alanine transaminase (ALT) in the HCV-infected recipients who received immunotherapy before and after LT. At 2 weeks after LT, HCV RNA remained undetectable in 4 patients (responder patients), whereas it exceeded 10 KIU/ml in 3 patients (non-responder patients). The ALT level in the responder patients was slightly lower than that in the non-responder patients until postoperative week 4, although this difference was not significant.



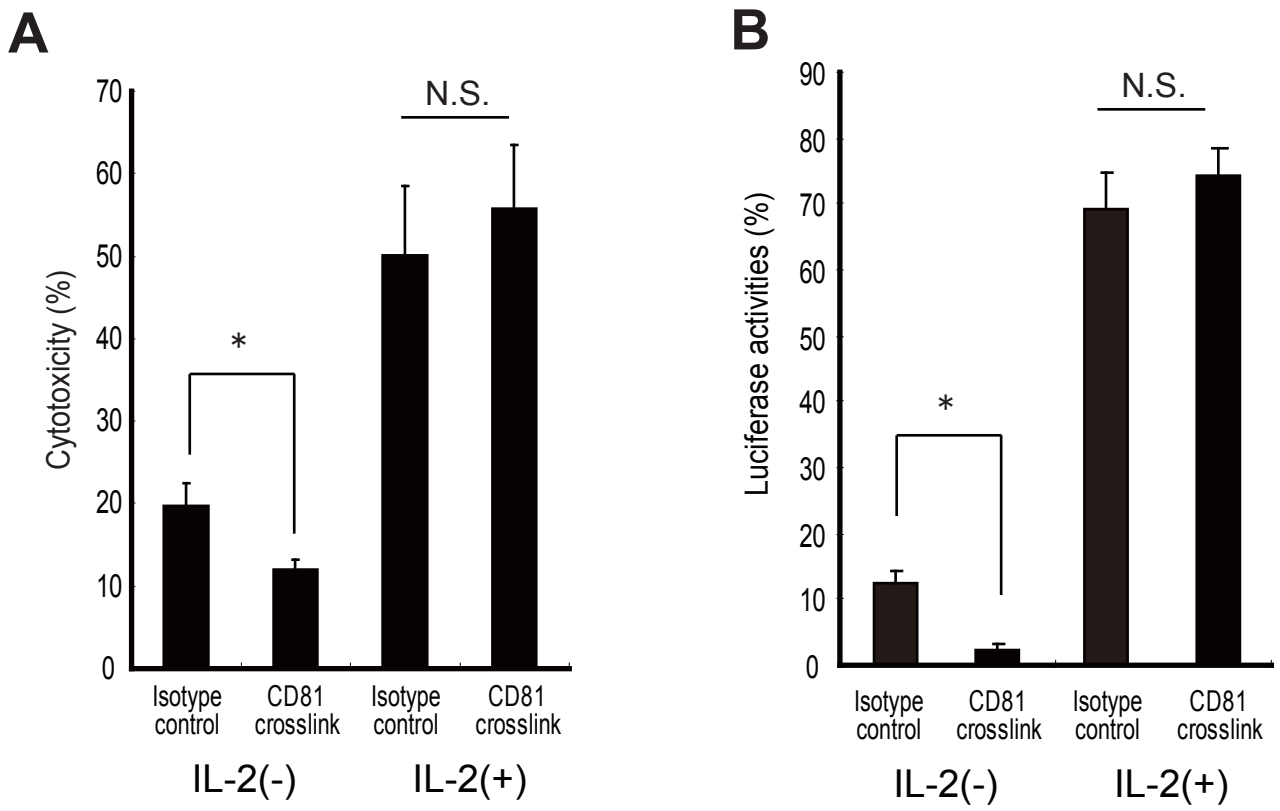

## Supplemental Figure 2

The proportion of TRAIL-positive and TRAIL-negative NK cells producing IFN-γ was not statistically different (49.4% ± 4.2% and 55.1% ± 8.7%, respectively; n = 4). Cells were stained for surface CD3/CD56/TRAIL and intracellular IFN-γ. Results show the expression of TRAIL and IFN-γ in gated $CD3^{-}CD56^{+}$ cells, and are representative of 4 independent experiments.




## Supplemental Figure 3

High serum HCV RNA titer in human hepatocyte-chimeric mice after inoculation of serum samples obtained from HCV-infected patients. Serial changes in HCV RNA titers (A) and human serum albumin levels (B) in the sera of mice inoculated with human serum samples positive for HCV genotype 1b. Each mouse was intravenously injected with 50 μL of the serum samples. Mice serum samples were obtained every 2 weeks after the inoculation, and HCV RNA titers and human serum albumin levels were analyzed.




## Supplemental Figure 4

IL-2 stimulation abrogated the inhibition of NK cells induced by the cross-linking of HCV-E2 and CD81. The effects of the cross-linking of HCV-E2 and CD81 on the activation of NK cells were investigated using a microtiter culture system. Microtiter plates (Microtest 96; BD Pharmingen) were coated with an isotype-matched control antibody or anti-CD81 mAb (clone JS-81, 5 μg/mL) in sodium bicarbonate buffer (pH 8.2). The standard 4-h $^{51}$Cr-release assay (A) or luciferase assay using genomic HCV replicon-containing hepatic cells (B) was used to assess the effect of HCV-E2 and CD81 cross-linking on the cytolytic or anti-HCV activities of NK cells. Human K-562 tumor cells (A) or HCV replicon cells (B) were used as target cells for both freshly isolated and IL-2-activated NK cells. The data represent NK activities of freshly isolated and IL-2-stimulated NK cells obtained from healthy volunteers (n = 6; A) or donor LMNCs (n = 4; B), with and without the cross-linking of HCV-E2 and CD81, against target cells (E:T ratio, A; 10:1, B; 2:1). Statistical analyses were performed using the paired t test. *$P < 0.05$ for control wells coated with isotype-matched irrelevant mAb vs. cross-linked CD81 wells coated with anti-CD81 mAb.