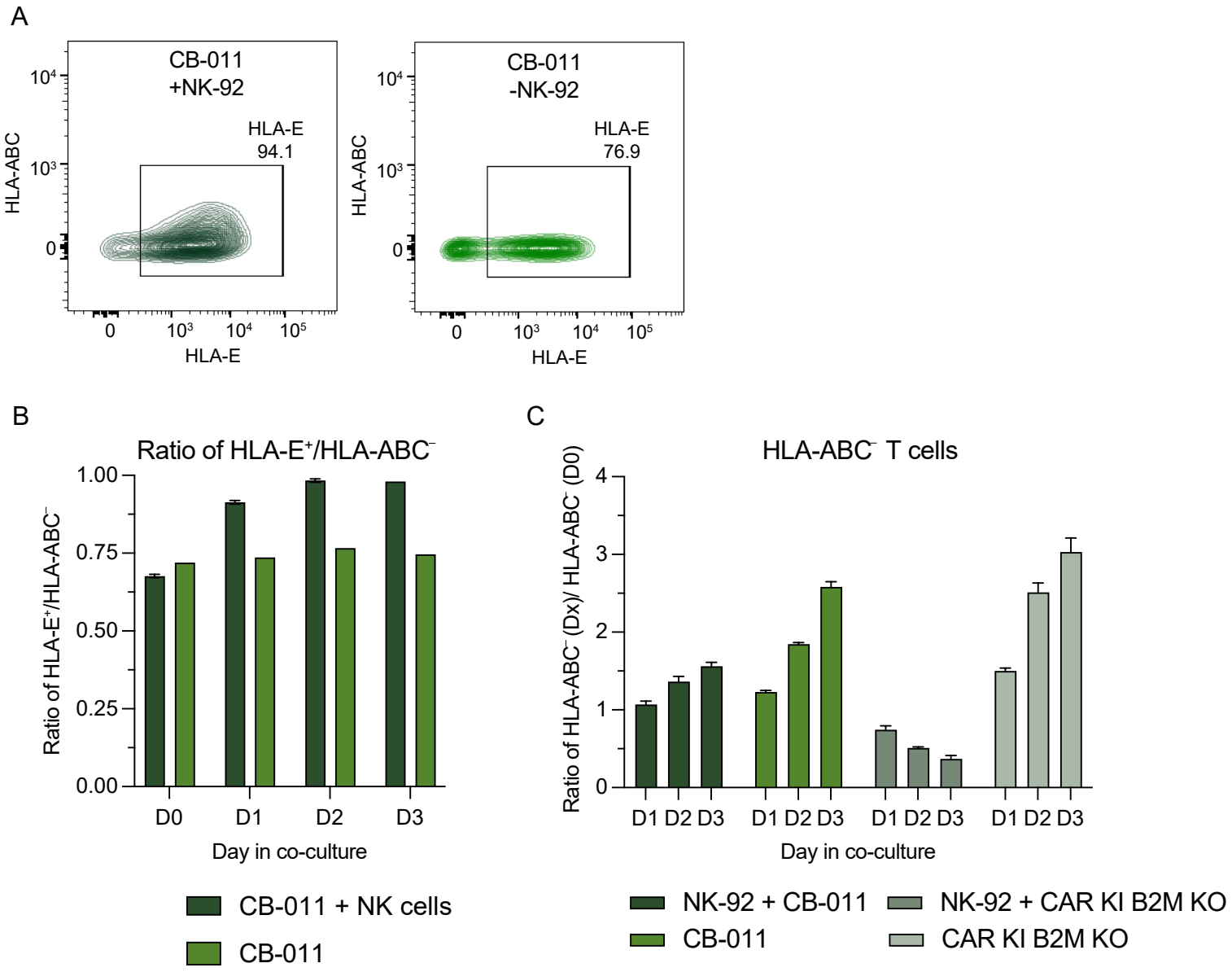
Figure S7. CB-011 immune-cloaking armoring strategy suppresses cytotoxic T and NK cell–mediated allograft rejection.



CB-011 cells were co-cultured with NK-92 cells at a 1:1 ratio in presence of IL-2. The percentage of HLA-E$^+$ and HLA-E$^-$ CB-011 cells were monitored over time. (A) Dot plots representing the percentage of the CB-011 HLA-E$^+$ population in presence (left panel) or absence (right panel) of NK-92 cells. The HLA-ABC$^-$ population represents a surrogate marker to identify the B2M KO population. (B) The ratio of HLA-E$^+$ CB-011 CAR-T cells within this population over 3 days of culture in the presence or absence of NK-92 cells is shown. In the absence of NK cells, the HLA-E$^+$ population was stable indicating no intrinsic survival advantage. In the presence of NK cells, the HLA-E$^+$ABC$^-$ population increased demonstrating protection of the HLA-E fusion expressing population from NK cell–mediated cytotoxicity. (C) CB-011 or CAR-T cells with a B2M KO without HLA-E fusion engineering (CAR KI B2M KO) were cultured in media supplemented with IL-2 in the presence or absence of NK cells. In the absence of NK cells, IL-2 supplemented T cells increased. In the presence of NK cells, T cells engineered without the HLA-E fusion were susceptible to NK cell–mediated lysis and numbers decline while CB-011 CAR-T cells were resistant.
B2M, beta-2 microglobulin; CAR, chimeric antigen receptor; D, day; HLA, human leukocyte antigen; IL-2, interleukin 2; KI, knock-in; KO, knockout; NK, natural killer.