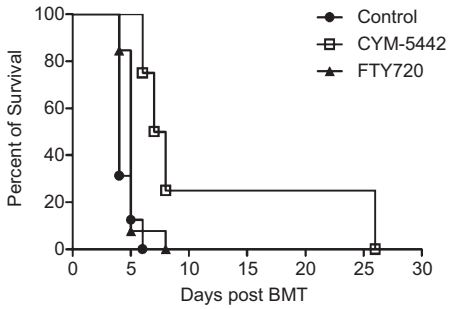
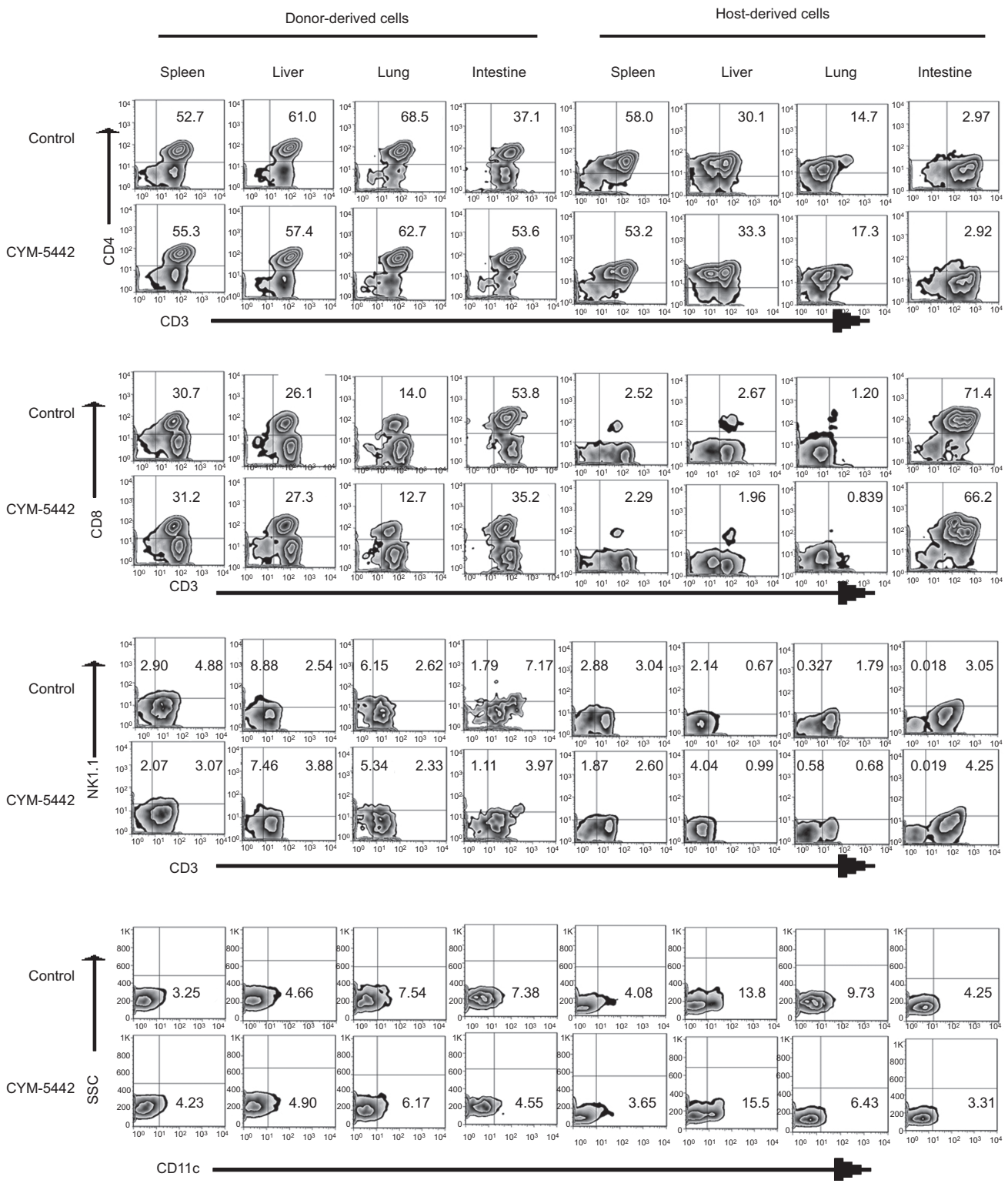
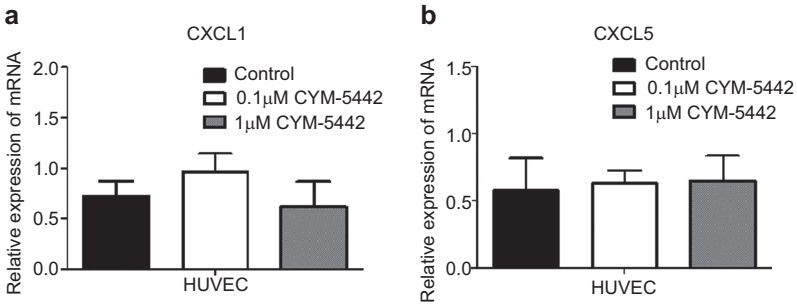


Supplemental Figure 1 The comparison of CYM and FTY720 treatment in inhibiting aGVHD. FTY720 treatment significantly prolonged the survival of GVHD mice compared to control-treated mice (n=8 per group; *P=0.0339). CYM-5442 treatment showed better therapeutic effect than FTY720 at the dose of 3 mg/kg (n=8 per group; ***P=0.0007). The data are representative of two independent experiments. Data are shown as mean±s.e.m.



Supplemental Figure 2 Representative flow chart of donor-derived and host-derived $CD4^+$ T cells, $CD8^+$ T cells, NK, NKT and DC from CYM-treated and control mice. Donor-derived cells were identified as $H2K^{b+}$ cells and host-derived cells were identified as $H2K^{d+}$ cells, and the cell subsets were analyzed by $CD3^+CD4^+$ for $CD4^+$ T cells, $CD3^+CD8^+$ for $CD8^+$ T cells, $CD3^-NK1.1^+$ for NK cells, $CD3^+NK1.1^+$ for NKT cells, $CD11c^+$ for DCs. Percent of donor-derived $CD4^+$ T cells, $CD8^+$ T cells, NK, NKT, DC and host-derived $CD4^+$ T cells, $CD8^+$ T cells, NK, NKT, DC in the spleen, liver, lung and small intestine have been shown.



Supplemental Figure 3 CYM did not affect the expression of CXCL1 and CXCL5 in endothelial cells. HUVECs were treated with 0.1 μM or 1 μM CYM or equal volume of DMSO for 24 h, and mRNA expressions of CXCL1 (**a**) and CXCL5 (**b**) were analyzed by real-time PCR.