Penicillin/streptomycin, 1% HEPES, 1% non-essential amino acids and 1% sodium pyruvate) overnight at 37°C, 5%CO_2.

The next day, PLBs were harvested via pipetting and exposed to 40Gy x-ray irradiation to arrest cell proliferation potential. These cells represent the "stimulator" cell fraction in the MLR assays.

Isolation of blood T cells and setup of MLR plates

PBMCs from either buffy coat sources or from refrozen leukopak material were taken from frozen storage and thawed. PBMCs were then washed, labelled with pan T cell isolation beads and blood T cells isolated via MACS LS columns. Resultant blood T cell fractions were then washed and stained with Cell Tracker Violet (CTV). CTV+ blood T cells were then co-cultured with irradiated stimulator PBLs. In parallel, frozen vials of GDX12 were defrosted, washed and immediately stained with CTV. CTV+ GDX012 cells were then cocultured with irradiated stimulator PBLs. In all cases, a ratio of 1:1 effector-stimulator cells were setup per well. Co-cultures were setup in complete RPMI media. Cultures were then incubated at 37°C, 5% CO_2 for 5 days. Cultures were not fed with extra media after this initial setup.

EXAMPLE 9: Expanded Vδ1+ T cell composition prevent tumour growth *in vivo*

A cell line derived xenograft model was used to assess the biodistribution and efficacy of GDX012 *in vivo*. Immunodeficient NOD SCID gamma (NSG) mice were challenged with an i.v. injection via the tail vein of either 0.5×10^6 or 1×10^6 cells of the human B-cell acute lymphoblastic leukaemia (ALL) cell line, NALM-6, which has been stably transduced to express a firefly luciferase (FLuc) and green fluorescent protein (GFP) gene. Mice were subsequently administered with or without a single i.v. injection via the tail vein of 20×10^6 GDX012 cells 24 hours or 6 days after tumour challenge. Control and treated mice all received i.p. injections of recombinant human IL-15 (1 µg/mouse every 2–3 days for the duration of the study) to support GDX012 survival. Tumour burden was assessed twice a week using s.c. administration of luciferin and in-life whole body bioluminescence imaging (BLI). After 4 weeks, mice were terminated and hind limb long bones removed. The bone marrow was flushed from the hind limb long bones using RPMI-1640 and collected for flow cytometric analysis. Briefly, cells were stained with eFluor780 fixable live/dead discrimination dye, then stained with FITC-conjugated anti-human CD45, PE-conjugated anti-human CD19 and APC-conjugated anti-human TCRγδ antibodies. Cells were finally fixed in 4% paraformaldehyde and run on a MACSQUANT10 flow cytometer.

A single administration of GDX012 in a systemic ALL *in vivo* model was able to reduce the growth of disseminated tumour compared with controls (**Figure 12**). In addition, GDX012 cells were able to home to and preferentially control tumour burden specifically within the bone marrow (**Figure 13**).