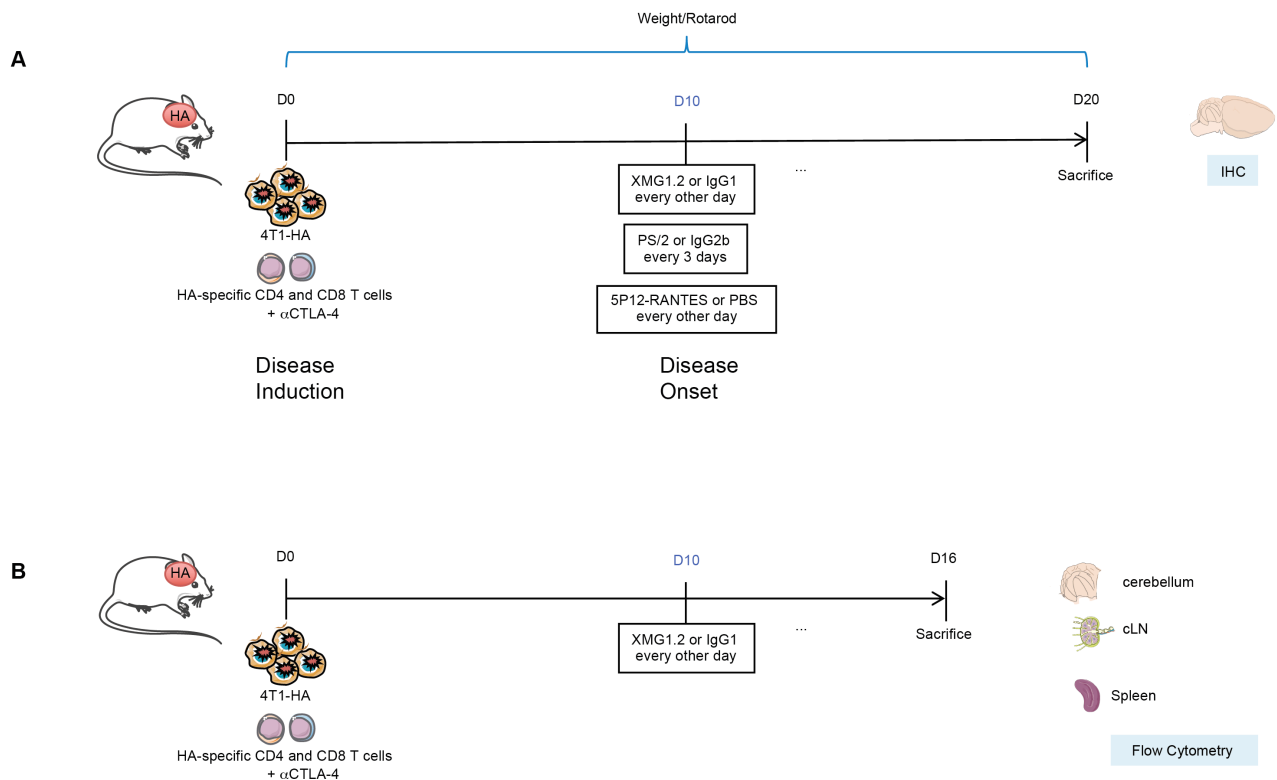
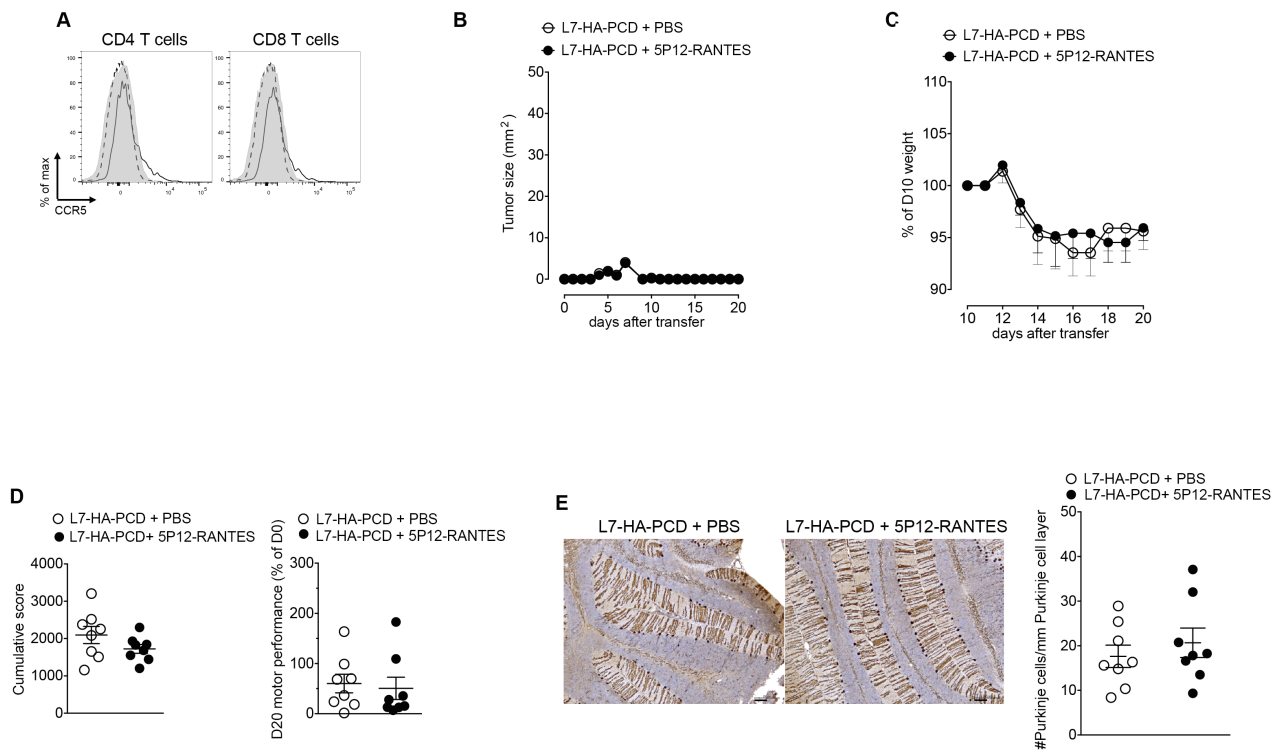


Supplementary figures

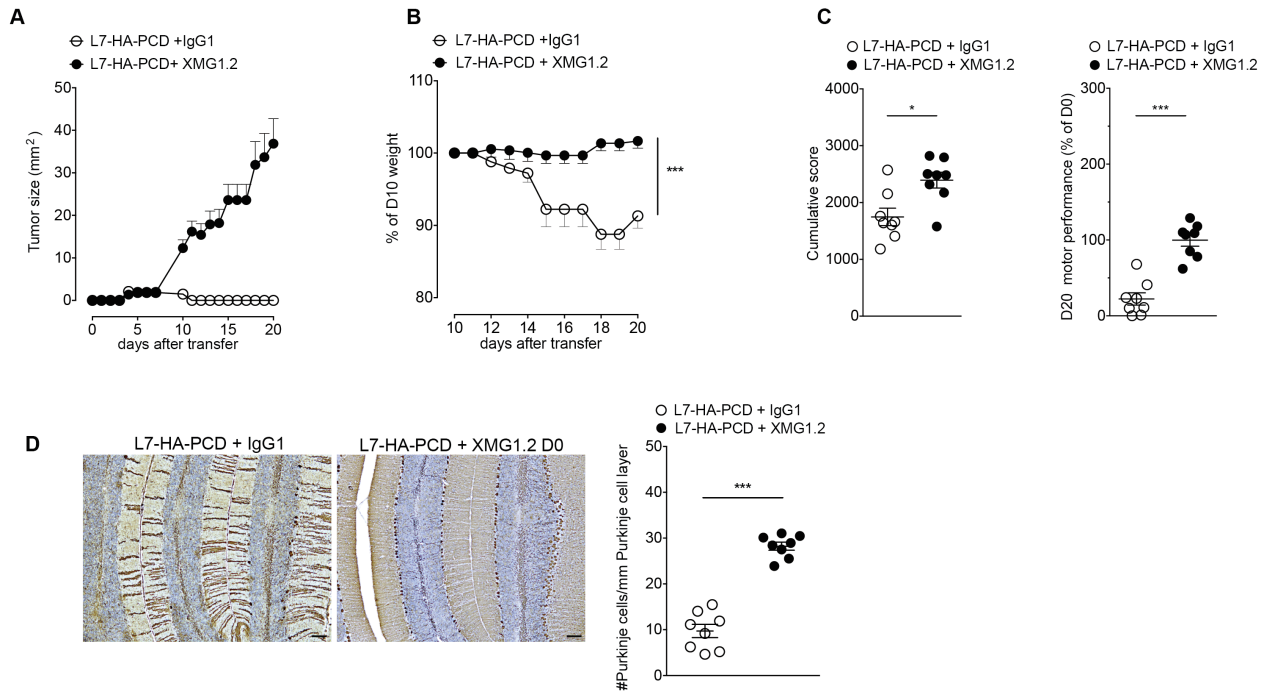


Suppl. Fig 1. PCD induction and treatment scheme.

Naïve HA-specific CD4 and CD8 T cells were isolated from TCR-transgenic mice, and transferred i.v. into L7-HA or WT littermates on day 0 (disease induction), together with the simultaneous s.c. implantation of 4T1-HA tumor cells and anti-CTLA-4 mAb injection. On day 10 (clinical onset of neurological disease), mice were treated with mAbs blocking α 4 integrin (PS/2) or neutralizing IFN γ (XMG1.2) or with a CCR5 antagonist (5P12-RANTES), or their respective controls. Mice were followed clinically daily and sacrificed for histological analysis at day 20 (a) or at the peak of disease (day 16) for characterization of cerebellum-infiltrating cells by flow cytometry (b).



Suppl. Fig 2. Blockade of the CCR5 chemokine receptor is ineffective to treat ongoing PCD.
a. Evaluation by flow cytometry of CCR5 expression on CD4 and CD8 T cells from different organs of L7-HA-PCD mice at day 16. Dotted line: spleen, grey: cLN, and solid line: cerebellum. **b.** Tumor size, **c.** mouse weight, **d.** rotarod performance, and **e.** Purkinje cell density from L7-HA-PCD mice treated with 5P12-RANTES or PBS from day 10, n = 8 per group. Bar = 100 μ m.



Suppl. Fig 3. Absence of tumor control and lack of PCD development upon IFN γ neutralization starting at day 0.

a. Tumor size, **b.** mouse weight, **c.** rotarod assessment, and **d.** Purkinje cell density from L7-HA-PCD mice treated with XMG1.2 or IgG1 from day 0 onwards. Bar = 100 μ m. n=8 mice per group, Mann-Whitney U test, *P<0.05; **P<0.01; ***P<0.001.