Table S1. **Both BM-derived and CNS resident cells require IFN-γ receptor expression to prevent onset of atypical disease**

Mouse genotype (percentage of donor cells in blood ± SD)	Incidence of limb dysfunction	Mean peak limb dysfunction clinical score	Incidence of flaccid paralysis	Incidence of dystonia	Incidence of nonclassical EAE
WT	5/5	4.1 ± 1	5/5	0/5	none
IFN-γR KO	8/8	3.4 ± 2	0/8	8/8	8/8
IFN-γR KO BM into WT (92.7 ± 0.6)	6/6	3.6 ± 0.8	6/6	0/6	6/6
WT BM into IFN-γR KO (85.7 ± 3.1)	6/6	4.7 ± 0.7	6/6	0/6	4/6

Chimeric mice were generated by injection of BM into lethally irradiated C57BL6/J (WT) or congenic IFN-γR–deficient (IFN-γR KO) mice. Chimerism was determined by examination of CD45 allelic markers. MOG-specific Th1 cells generated from C57BL6/J mice were injected into chimeras and nonirradiated WT or IFN-γR KO. Mice were examined over time for clinical signs of movement dysfunction, vertigo/disequilibrium, and ataxia. Results are shown ± SD. Results are representative of three separate experiments.