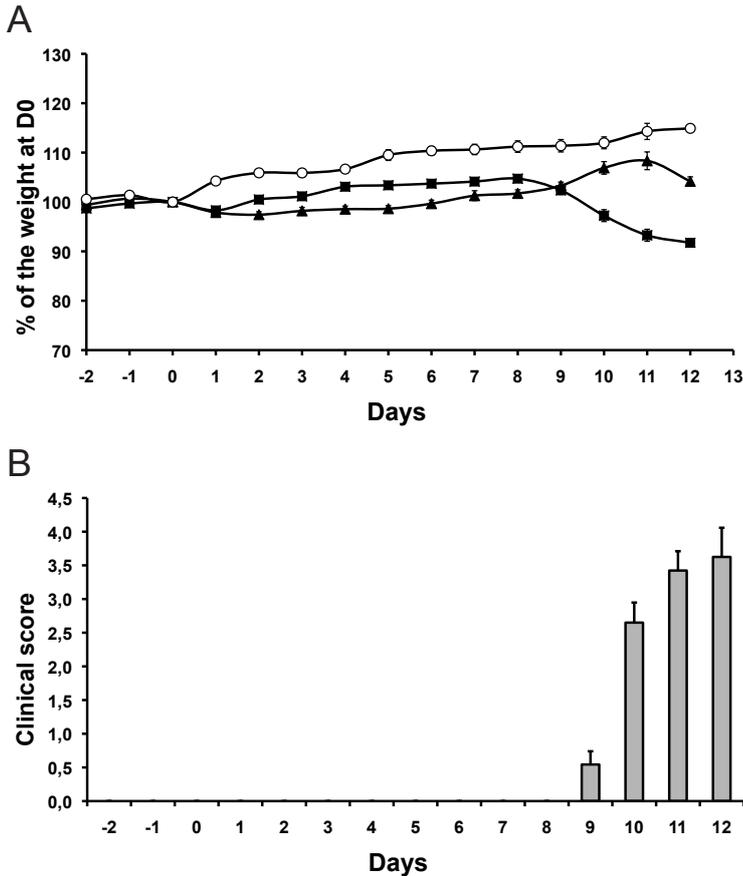


Additional File 2

Schmitt C, et al: Brain leukocyte infiltration initiated by peripheral inflammation or EAE occurs through pathways connected to the CSF-filled compartments of the forebrain and midbrain.



Time course of the daily weight and clinical score in control, PI and EAE-diseased animals. A. Weight of control (open circle), PI (triangle) and EAE-diseased (square) rats. Data are expressed as the mean \pm SEM of the percentage of the initial weight of the animal at the time of the induction (D0). Data are combined from 4 different experiments for a total of 93 rats, with a minimum of 5 animals at each time point up to D11. One control, 1 PI and 7 EAE diseased animals were kept up to D12. The weight of both PI and EAE diseased animals is significantly lower than the weight of control animals, from D1 post-treatment, up to D9 (PI), or D11 (EAE). The weight of EAE diseased animals becomes significantly lower than the weight of PI animal from D10 to D12 (all levels of significance: $p < 0,05$; Anova followed by post-hoc Tukey-Kramer test). B. Clinical course of EAE in rat. The first neurological signs (onset) appeared on D9 and the peak of the disease occurred on D11-12, matching the period of prominent weight loss reported in A. In mice, a similar weight profile and clinical course was also observed for PI and EAE animals, but the smaller number of animals did not allow a statistical analysis. Neurological signs in EAE-diseased mice were first observed on D8. The peak of the disease lasted for a longer period than in rat, i.e. between D11 and D16, and was also accompanied by a marked weight loss (not shown).