HIV-1 Tat-induced diarrhea evokes an enteric glia-dependent neuroinflammatory response in the central nervous system.

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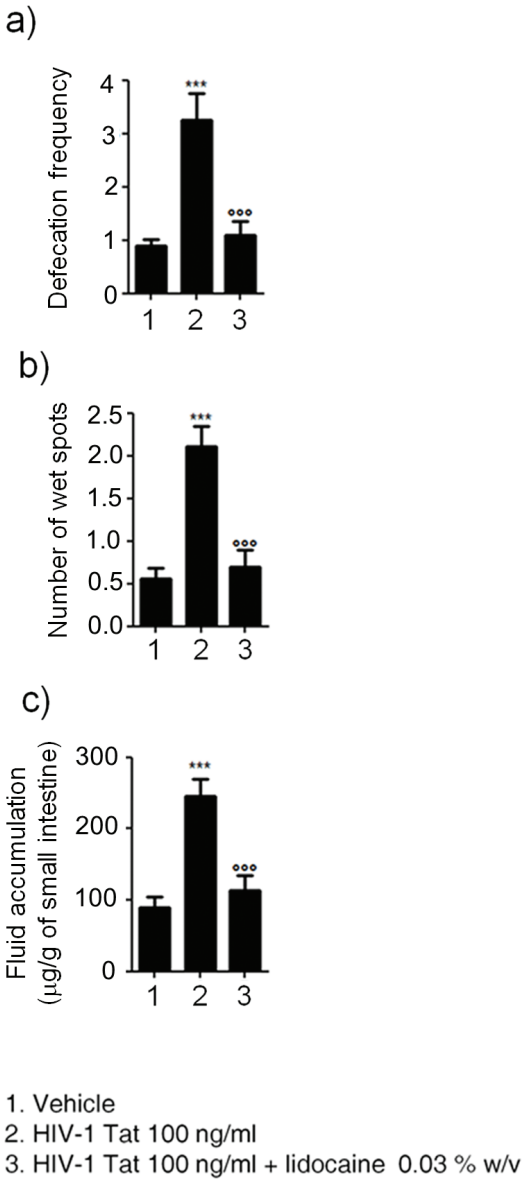
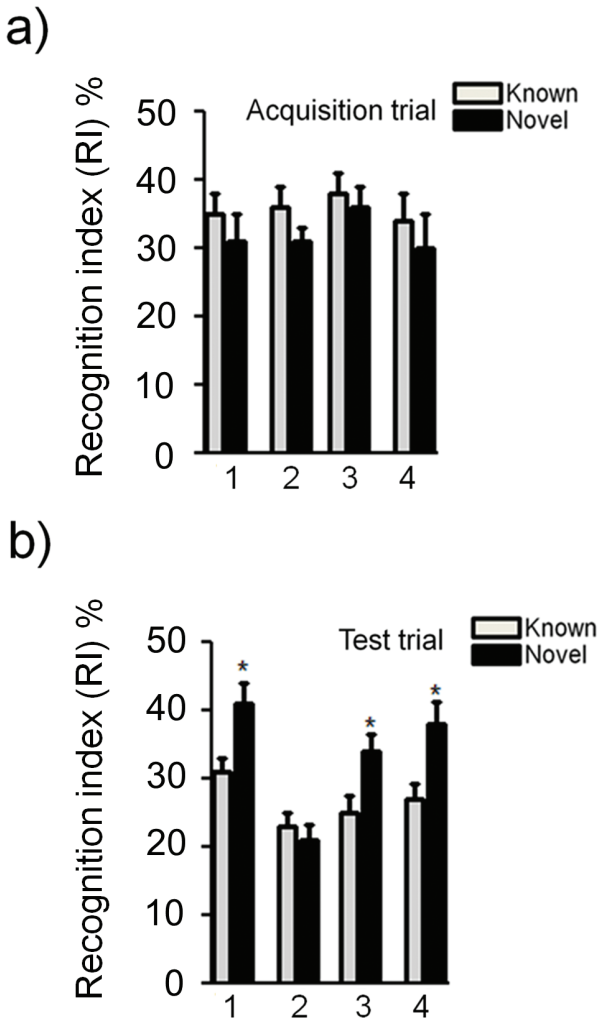
Supplementary Figure 1

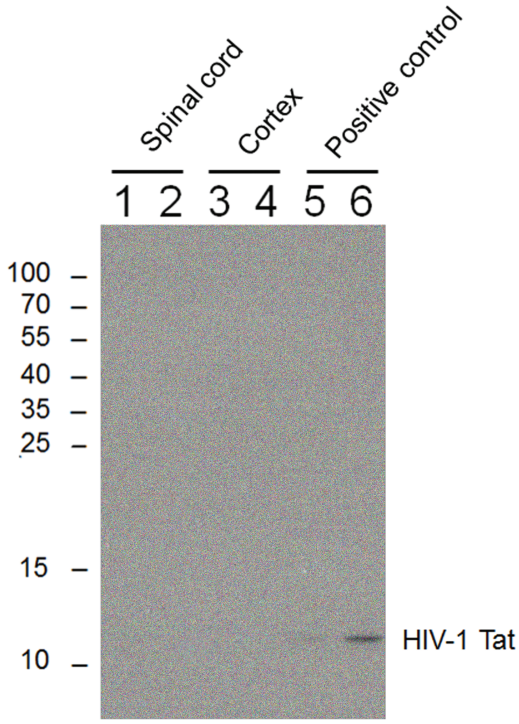


Supplementary Figure 2



1. Vehicle
2. HIV-1 Tat 100 ng/ml
3. HIV-1 Tat 100 ng/ml + lidocaine 0.03 % w/v
4. Bisacodyl 20 mg/kg

Supplementary Figure 3



1. Vehicle
2. HIV-1 Tat 100ng/ml
3. Vehicle
4. HIV-1 Tat 100ng/ml
5. HIV-1 Tat 30ng protein
6. HIV-1 Tat 100ng protein

Legends to supplementary figures

Supplementary Figure 1. Intracolonic administration of HIV-1 Tat protein (100 ng/ml) caused a an acute onset diarrhea, with a significant increase of (a) defecation frequency, (b) number of wet spots and (c) fluid accumulation, that were all significantly inhibited by lidocaine (***$p<0.001$ vs vehicle and lidocaine groups; °°°$p<0.001$ vs HIV-1 Tat group).

Supplementary Figure 2 To assess recognition memory, object recognition task was performed on different experimental groups at 21 after the administration of HIV-1 Tat. Recognition index (RI) defines the ratio of time spent exploring the novel object over the total time spent exploring both familiar and novel objects. In rats treated with HIV-1 Tat a significant reduction of the RI was observed as compared to vehicle group. Administration of lidocaine restored the novel object RI, while bisacodyl did not significantly affect the novel object RI (*$p<0.05$ vs vehicle-treated rats; n=8 for each group).

Supplementary Figure 3. Immunoblot analysis showing that following intracolonic administration, HIV-1 Tat protein was virtually absent in the spinal cord (lines 1-2) and the cortex (lines 3-4) homogenates of treated animals; lines 5 and 6, indicate positive controls of HIV-1 Tat protein at 30 and 100 ng, respectively.