Supporting Information

Bajo *et al*. 10.1073/pnas.0802302105

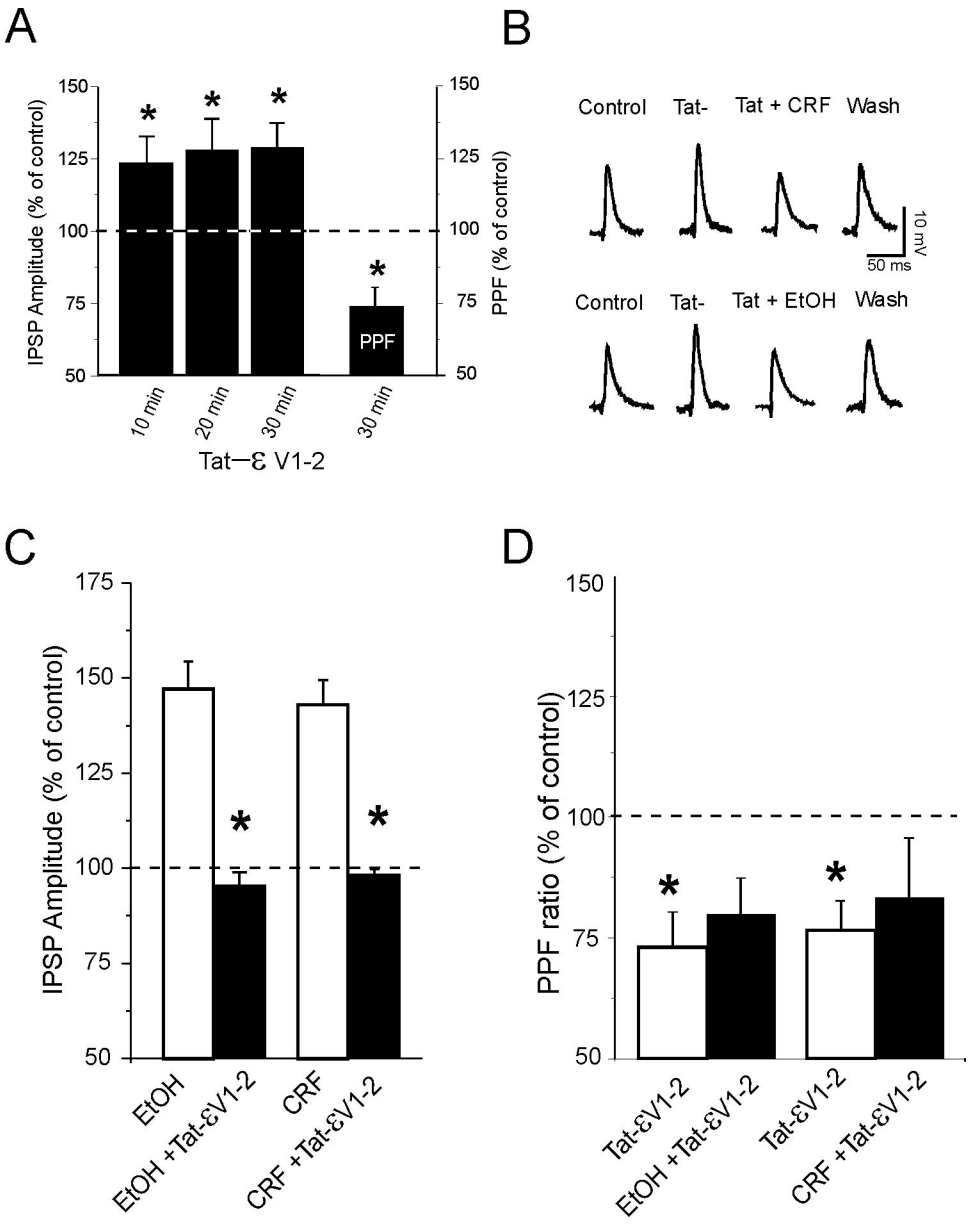

Fig. S1. Pharmacological antagonism of PKCε with a PKCε inhibitor, Tat-εV1–2 peptide (Tat-εV1–2; 500 nM), increases GABAergic transmission in CeA but blocks CRF (200 nM) and ethanol (44 mM) effects. (*A*) Superfusion of the inhibitor alone increased the evoked IPSP amplitudes and significantly decreased the paired pulse ratio of IPSPs in 5 CeA neurons from PKCε$^{+/+}$ mice. *, $P < 0.05$ by one group *t*-tests compared to control IPSP amplitude of 100% (dashed line). (*B*) Representative evoked IPSPs recorded under various treatment conditions as noted. The PKCε inhibitor (Tat-εV1–2) enlarged the baseline evoked IPSPs and reversed the usual increase of IPSP amplitudes by both CRF (*Upper*) and ethanol (*Lower*). (*C*) Pretreatment of CeA neurons with Tat-εV1–2 completely blocked both the CRF- and ethanol-induced increase in mean IPSP amplitudes (*, $P < 0.05$, unpaired, two-tailed, *t* tests). (*D*) Tat-εV1–2 also prevented CRF- and ethanol-induced decreases in PPF of IPSPs, confirming that the presynaptic action of CRF and ethanol at GABAergic synapses in the CeA is mediated by PKCε.