Supporting Information

Edwards *et al.* 10.1073/pnas.0803558105

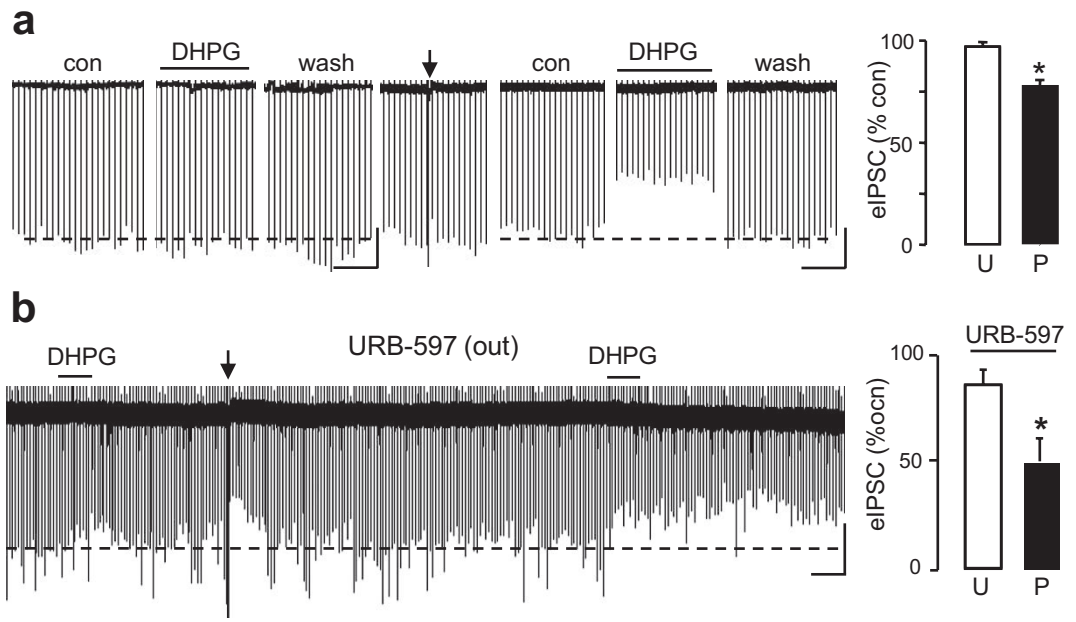

Fig. S1. DSI primes eCB_{mGluR} induced by lower DHPG concentration. (*a*) DSI primed eCB_{mGluR} in response to 10 μM DHPG. [Scale bars: 30 s/250 pA (first period), 30 s/300 pA (second).] Group data for *a* show that eIPSCs were not reduced (97.0 $\pm$ 2.6% of baseline) before DSI, but after the DSI step they were reduced to 77.0 $\pm$ 3.85% of baseline ($P < 0.001$, paired *t* test, $n = 10$). (*b*) URB-597, 1 μM (present throughout) did not prevent priming of 50 μM DHPG reduction of eIPSCs. (Scale bars: 1 min/300 pA.) Group data for *b* show eIPSC suppression to 85.8 $\pm$ 7.01% of baseline before a DSI trial and to 50.1 $\pm$ 10.7% afterward ($P < 0.05$, $n = 4$).

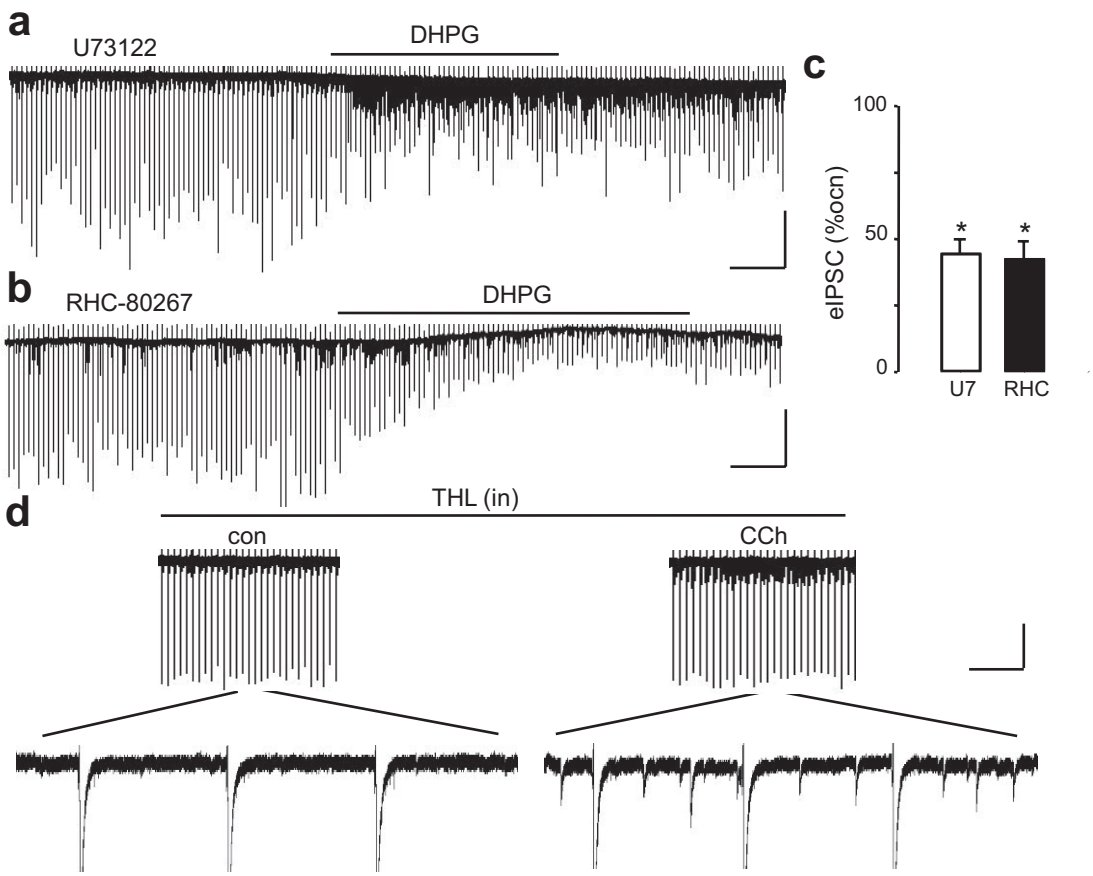

Fig. S2. Inhibitors of PLC and DGL do not inhibit priming. (*a*) Slices were pretreated with U73122 (PLC antagonist, 6 μM; Tocris) or (*b*) RHC-80267 (DGL antagonist, 70 μM; Sigma) for >1.5 h. The drugs were also included in the bath and in the pipette during the experiment. Cells received one DSI step followed >10 min later by 50 μM DHPG. Example traces show typical eCB_{mGluR} responses. [Scale bars: 1 min/400 pA (U73122) and 1 min/500 pA (RHC-80267).] (*c*) Group data for *a* and *b*. eCB_{mGluR} for slices treated with U73122 was 44.3 $\pm$ 5.60% ($n = 3$), and with RHC-80267 it was 42.4 $\pm$ 6.46% ($n = 3$). (*d*) THL prevents eCB_{mAChR} without affecting the ability of CCh to induce spontaneous IPSC firing, showing that THL does not act as a mAChR antagonist. Lower traces were expanded and amplified to reveal the smaller intervening CCh-induced sIPSCs; the large eIPSCs occurring every 4 s were truncated for display purposes. (Scale bars: 1.5 s/300 pA.)