Supplementary data

Supplementary Figure 1. Passive membrane properties of the neurons recorded and minimal stimulation.

(a-e) Bar graphs of (a), resting membrane potential (b), membrane potential for spike threshold (c), the number of spikes in response to a 200 pA current pulse of 500 ms, membrane time constant (d) and input membrane resistance (e) for CA3 pyramidal cells from WT, GluK2$^{-/-}$, GluK3$^{-/-}$ and GluK5$^{-/-}$ mice. Rise time of unitary Mf-EPSCs (f) and Mf-EPSPs (g) in WT, GluK2$^{-/-}$, GluK3$^{-/-}$ and GluK5$^{-/-}$ mice.

Supplementary Figure 2. Presynaptic KARs increase frequency facilitation.

(a) Examples of frequency facilitation of Mf-EPSCs recorded when shifting stimulation from 0.1 to 1 and 3 Hz for WT, GluK2$^{-/-}$, GluK3$^{-/-}$ and GluK5$^{-/-}$ mice. Traces are the average of 15 recordings for 0.1 Hz and 40 for 1 and 3 Hz. (b) Summary graphs of frequency facilitation from 0.1 to 1 Hz (top) and 3 Hz (bottom) (WT: n = 8; GluK2$^{-/-}$: n = 7; GluK3$^{-/-}$: n = 6; GluK5$^{-/-}$: n = 6). Scale bars represent 10 ms and 100 pA.

Supplementary Figure 3. Lack of effect of GABAergic inhibition on Mf spike transmission in acute hippocampal slices.

A lack of difference in (a) discharge probability at 20 Hz (GABA blocked, n=6; GABA intact, n = 5, p = 0.60), 50 Hz (GABA blocked, n = 4; GABA intact, n = 8, p = 0.35) and 100 Hz (GABA blocked, n = 6; GABA intact, n = 8, p = 0.76) (b) spike delay, in the presence and absence of GABAergic inhibition by picrotoxin and CGP 55845, in WT mice (GABA blocked, n=6; GABA intact, n = 5, p = 0.98), 50 Hz (GABA blocked, n = 4; GABA intact, n = 8, p = 0.74) and 100 Hz (GABA blocked, n = 6; GABA intact, n = 8, p = 0.20). Experiments in (b) are similar to Fig. 4 and discharge probability (a) was calculated from the 3rd pulse onwards.

Supplementary Figure 4. KARs control CA3 pyramidal cell output in response to another physiological granule cell firing pattern of stimulation.

(a, b) Raster plots of spike discharge from 10 consecutive recordings (top) in response to the physiological pattern illustrated at the top of the panel, with representative recordings (bottom). This pattern of stimulation was extracted from an *in vivo* recording as the one shown in figure 7 but with an average frequency of 1.4 Hz. Data are from a single CA3 pyramidal cell of a WT (a) and a GluK2$^{-/-}$ mouse (b). (c-e) Plots of the number of spikes during 10 consecutive recordings as a function of

the instantaneous frequency of each stimulation for WT (n = 6) and GluK2$^{-/-}$ (n = 9) (c), GluK3$^{-/-}$ (n = 9) (d) and GluK5$^{-/-}$ (n = 9) (e) mice.

Supplementary Figure 5. Spike transmission at Mf synapse is not impaired in GluK1$^{-/-}$ mice.

Similar plot as in Fig. 7d with the same pattern of stimulation, in cells recorded from GluK1$^{-/-}$ mice, showing a lack of impact of the GluK1 subunit in synaptic integration and spike transmission at the MF-CA3 synapse (average discharge probability, WT: 0.48± 0.06 Hz, n=10; GluK1$^{-/-}$: 0.41± 0.04, n=9, p=0.41).