

Supplemental Figure legends

Figure S1. Initiation of action potentials during somatic and dendritic current ramp injections in CA1 pyramidal neurons. *(A)*, Simultaneous somatic (black traces) and dendritic ($\sim 300 \mu\text{m}$ away from the soma; blue traces) voltage responses to somatic current ramp injections. Current ramps were injected either into the soma or into the apical dendrite from a background membrane potential of $\sim -73 \text{ mV}$. Overlay of the first somatic and dendritic action potential suggests that the first action potential was initiated at the perisomatic region. *(B)*, Somatic and dendritic voltage responses from the same neuron in *(A)* when a current ramp was injected into the distal dendrite. Again the first action potential was initiated from perisomatic region. Same result was obtained from 4 different neurons and with different current ramp speeds.

Figure S2. Ca^{2+} -spikes in CA1 pyramidal neuron apical dendrites under hyperexcitable conditions. Simultaneous somatic (black traces) and dendritic (blue) voltage response to somatic injection of depolarizing current pulses from a membrane potential of $\sim -68\text{mV}$, under control condition *(A)*, after bath-applying 3 mM 4-aminopyridine (4-AP) *(B)*, and after subsequently bath-applying 1 μM TTX *(C)*. *(D)* The dendritic voltage responses recorded before and after bath-applying 4-AP are shown superimposed. All the records *(A-D)* were obtained from the same cell.

Figure S3. Effects of retigabine on somatic action potential bursting in CA1 pyramidal neurons. *(A)*, Perisomatic application of retigabine (20 μM) enhanced the spike frequency adaptation, but had little effect on the burst of spikes at the beginning of the spike train. *(B)*, Summary diagram showing the effect of perisomatic application of retigabine (20 μM) on burst frequency. *(C)*, Dendritic local application of retigabine (20 μM) had no effect of spike frequency adaptation or spike bursting. The panels to the right in *(A)* and *(C)*, show bursts before and after retigabine application on an expanded time scale. *(D)*, Summary diagram showing the effect of dendritic

application of retigabine (20 μM) on burst frequency. In all of the experiments illustrated in this figure, the slices were incubated with 10 μM DNQX and 2-3 mM 4-AP prior to and during testing. Action potential trains were evoked by injecting 900ms-long depolarizing current pulses into the soma from a membrane potential of $\sim -68\text{mV}$.

Figure S4. Effects of focally applied XE991 on the somatic and the dendritic Ca^{2+} -spikes of CA1 pyramidal neurons. *(A)*, The Ca^{2+} -spikes at the very proximal apical dendrite (24 μm from the soma) of a neuron before (black) and after (red) focal application of XE991 (20 μM) near the recording site. *(B)*, The Ca^{2+} -spikes recorded from the distal apical dendrite (313 μm from the soma) of a neuron before (black) and after (red) focal application of XE991 (20 μM) near the recording site. *(C)*, The effect of XE991 on Ca^{2+} -spikes was plotted against the distance of the recording sites from the soma. The ***XE991 effect*** was calculated as % increase in the entire area of the depolarizing response during the current pulse (including the Ca^{2+} -spikes), after focal application of XE991, compared to the area before the application of XE991 (=100%). The data points were fitted with a mono-exponential curve (exponential decay constant = 56 μm).