Supplementary material to:

Parvalbumin-containing fast-spiking basket cells generate the field potential oscillations induced by cholinergic receptor activation in the hippocampus.

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Supplementary figure 1. Further examples of electron microscopic validation of basket (A-F, n=3) and axoaxonic cells (G-L, n=3) identified with double immunofluorescent staining for biocytin and Ankyrin G.
Supplementary figure 2. The transition of in vitro CCh-induced oscillation from room temperature to 32-34 °C is continuous recorded in a submerged slice chamber. A) The colored spectrograms of 10-s-long epochs juxtaposed in time show that by increasing the bath temperature in the submerged recording chamber, the frequency of the CCh-induced oscillation increases from ~20 Hz to ~40 Hz. Warmer colors indicate peaks on the power spectra. Red arrow indicates a harmonic of ~20Hz oscillation at ~ 40 Hz. Power spectra of the oscillation are shown at different time-points (and temperatures) marked by yellow boxes before (left), during (right) and after (far right) the temperature induced acceleration of the oscillation. B) As shown on the unfiltered traces, by increasing the temperature, the frequency but not the amplitude nor the shape of the oscillation changes. The transition is continuous, without a break in the properties of the oscillation, suggesting that the generation mechanisms at different temperatures are identical. C) Summary plot showing the significant increase in the frequency of CCh-induced oscillations after changing from the room temperature (18.5 ± 0.9 Hz) to 32-34 °C (37.8 ± 1.6 Hz, n=4, p<0.0001).
Supplementary figure 3. The transition of in vitro CCh-induced oscillation from 32-34 °C to room temperature is continuous recorded in an interface slice chamber. A) The colored spectrograms of 10-s-long epochs juxtaposed in time show that by decreasing the bath temperature from 34 °C to room temperature in the interface recording chamber, the frequency of the CCh-induced oscillation decreases, a change that is reversible by restoring the bath temperature. Warmer colors indicate peaks on the power spectra. Power spectra of the oscillation are shown at different time-points (and temperatures) marked by yellow boxes before (left), during (right) and after (far right) the temperature induced modulation of the oscillation. B) As shown on the unfiltered traces, by decreasing the temperature, the frequency decreases, which can be reversed by increasing the temperature. The transition is continuous, without a break in the properties of the oscillation, suggesting that the generation mechanisms at different temperatures are identical. C) Summary plot showing the significant decrease in the frequency of CCh-induced oscillations after changing from 32-34 °C (33.1 ± 0.7 Hz) to the room temperature (20.1 ± 0.9 Hz, n=5, p<0.0001) without a change in the peak power (32-34 °C: 368.8 ± 119.7 µV^2/Hz; room tempr.: 690.1 ± 243.1 µV^2/Hz, n=5, p=0.19).
Supplementary figure 4. Firing phase and phase coupling of all recorded cells, the effect of filtering on spike phase detection.

A) Firing phase of individual cells within each population scattered considerably, though their phase coupling was higher than found in in vivo experiments. Preferred firing phase for each perisomatic inhibitory cell group was calculated in two different ways: either the average phase and coupling of all individual cells were circularly averaged (large closed symbols), or all spikes from the same group of cells were pooled and the circular average and coupling were calculated (large open symbols). As visible on the figure the result of the two different
ways of averaging gave similar phase and coupling strength for the populations and that the
average phases of the populations are close to each other. B) Filtering oscillation in different
ways results in different spike phase assignment. In in vivo studies the field signal is band-
pass filtered in a narrow range (BP, 5-30 Hz) to detect gamma oscillation. This results in a
loss of high-frequency components and the signal is sinusoid rather than saw-tooth shaped.
The negative peak of the sinusoid wave is shifted earlier compared to the negative peak of the
wide BP filtered trace (5-500 Hz, red bars indicate detected negative peaks). Therefore the
phase assigned to the detected spike (green bars, top trace) is systematically larger for the
narrow BP filtered trace (middle trace) than for the wide BP filtered trace (bottom trace), as
shown by the longer arrows between the red and green lines on the middle trace compared to
the bottom trace. While in our study there is a shift in the firing phase of the perisomatic
inhibitory cells, if detected on the basis of filtering used in in vivo studies versus how we
filtered the oscillation, the shift is near systematic and the preferred firing phase of the cells
overlaps. Regardless how firing phase was calculated, there was no significant difference in
the populations’ firing phase, due to the scatter of the firing preference of individual cells
shown on A.
Supplementary figure 5. Cholinergically induced oscillations at 32-34 °C can be also reversibly blocked by DAMGO application. The colored spectrograms of 10-s-long epochs versus time show that bath application of DAMGO effectively diminished the oscillation, which could be fully reversed after washing out the drug. Warmer colors indicate peaks on the power spectra. Power spectra of the oscillation are shown at different time-points marked by yellow boxes before (left) and after (middle) the DAMGO treatment. As shown on the unfiltered traces taken from the same periods, DAMGO application substantially reduced the peak amplitude, i.e. the power of the oscillation at this, physiologically more relevant temperature. C) Summary plot showing the significant decrease in the peak power of CCh-induced oscillations after DAMGO treatment (4.4 ± 0.5 µV²/Hz) compared to control (23.5 ± 5.4 µV²/Hz, n=3, p=0.05).
Supplementary figure 6. Effect of DAMGO could be reversed (left, n=3, baseline: 54.07 ± 18.69 µV²/Hz, DAMGO: 14.47 ± 3.71 µV²/Hz, +CTAP 55.60 ± 21.87 µV²/Hz, baseline and DAMGO+CTAP is significantly different from DAMGO p=0.035) or occluded (right, n=3, baseline: 47.57 ± 7.25 µV²/Hz, CTAP: 44.20 ± 6.51 µV²/Hz, +DAMGO 43.53 ± 3.72 µV²/Hz, no significant difference) by bath application of a MOR antagonist, CTAP.
Supplementary figure 7. DAMGO application suppresses network oscillations in parallel to the decrease of IPSC amplitudes, that results in de-synchronization of EPSCs. The left column shows in black parallel recordings of field oscillations (top trace) as well as EPSCs (middle) and IPSCs (bottom) recorded from two pyramidal cells during CCh-induced oscillation. As shown on the autocorrelograms (right column), postsynaptic currents were highly synchronous and phase locked to the field oscillation. Application of DAMGO (grey traces in the middle) significantly reduced synaptic inhibition (averaged charge changed from 37.02±3.38 pC to 17.02±0.90 pC, n=5, p=0.04), and resulted predominantly in the desynchronization of EPSCs without a change in their charge (from 8.72±0.67 pC to 10.09±0.79 pC, n=5, p=0.15). As shown by the autocorrelogram, the field oscillations were also inhibited (peak power, CCh, 83.2±7.5 µV²/Hz; in CCh+DAMGO, 28.4±2.9 µV²/Hz, n=5; p=0.04).
Supplementary figure 8. DAMGO decreases the frequency of miniature inhibitory, but not excitatory synaptic currents without affecting their amplitudes. (A) Raw data obtained in the presence of 500 nM TTX in CA3 pyramidal cells are shown. (B) DAMGO application in the presence of 5 µM CCh did not alter the amplitude of mIPSCs (n=7, p>0.1) or mEPSCs (n=7, p>0.1) as shown on the cumulative distributions. (C) In contrast, the frequency of mIPSCs were decreased (n=7, p<0.001) indicated by the rightward shift on the cumulative distributions, an effect that suggests the presynaptic locus of DAMGO action. The frequency of mEPSCs was not altered (n=7, p>0.1).
Supplementary figure 9. Carbachol-induced oscillations in hippocampal slices prepared from CB1 cannabinoid receptor knockout mice are also suppressed by bath application of the µ-opioid receptor agonist DAMGO. Field oscillation was monitored in the pyramidal cell layer of CA3 at 32-34 °C. A) The color figure shows the changes in the power spectra as a function of time, which were calculated from 10-s-long epochs. Warmer colors indicate the magnitude of the peak on the power spectra. Example traces from before and after DAMGO application (in white) are overlaid on the spectrogram. Left and right graphs indicate the power spectra under control conditions and after the drug treatment. The red arrow shows the rapid drop in the oscillation power. B) Summary plot of results obtained in four different experiments. In the control, oscillations had a mean frequency of $28.8 \pm 1.3$ Hz and their max. gamma power was $44.1 \pm 28.9 \mu V^2/Hz$ ($n=4$), which significantly differed from those values calculated in the presence of DAMGO $5.8 \pm 2.6 \mu V^2/Hz$ ($p=0.03$).