SUPPLEMENTAL METHODS

Whole cell current-clamp: action potential, input resistance, membrane time constant analyses

Whole cell current-clamp experiments were performed with a K-gluconate based solution of the following composition: K-gluconate 130; NaCl 10; Mg-ATP 2; Na-GTP 0.3, HEPES 10; EGTA 0.6, 0.3% biocytin, adjusted to pH 7.2 and 275 mOsm. Sub-threshold depolarizing and hyperpolarizing current pulses (-40pA to +40pA; 20pA steps) were delivered. The steady-state voltage responses were plotted against the amplitude of current injection and the slope of the linear fit of this relationship was determined to calculate input resistance. To elicit action potential discharge, depolarizing current pulses of +60 to +300pA (60pA steps; 1s duration) were delivered from which the firing frequency (in Hz) was calculated. To determine the extent of spike frequency accommodation the spike frequency in the first and last 200ms epochs of the 1s depolarizing pulse of 300pA were compared.

Whole-cell voltage clamp: evoked EPSCs and miniIPSC analyses

Whole cell voltage-clamp experiments were performed with an intracellular solution of the following composition (in mM); Cesium methanesulfonate 135, NaCl 3, HEPES 10, EGTA 0.6, MgATP 4, NaGTP 0.3, QX-314Cl^- 5, 0.3% biocytin adjusted to pH 7.2 and 280 mOsm.

Evoked EPSCs were elicited by stimulation using a concentric bipolar electrode (FHC, Bowdoin, ME) placed in the subiculum (see Fig. 2A) or the CA3 pyramidal layer region. Cells were voltage-clamped at -60mV (close to the calculated E_{Cl^-} of -58mV) in order to isolate AMPA-receptor mediated currents without the need of $GABA_A$-receptor

antagonists. In all cases an incision was made between the CA1 and CA3 (see Fig. 2A). Evoked NMDA-receptor mediated responses were recorded at a holding potential of +40mV in the presence of 10μM CNQX and picrotoxin or gabazine.

Miniature IPSCs (mIPSCs) were recorded using the Cs-based intracellular solution at a holding potential of 0mV (to isolate outward mIPSCs). The extracelullar solution was supplemented with 1μM TTX. The application of 10μM gabazine selectively inhibited all currents confirming their identity as $GABA_A$-receptor mediated (data not shown). Gap-free recordings lasted between 5-10 minutes and data was discarded if the series resistance value deviated by more than 15% by the end of the acquisition period. Data was analyzed using MiniAnalysis Software (Synaptosoft Inc, Decatur, GA).

All holding potential values stated are after correction for the calculated junction potential offset of 10mV.

Cell-attached recordings: Resting membrane potential measurements and effects of $GABA_A$-receptor activation on membrane potential.

Resting membrane potentials were attained using cell-attached patch recordings as previously described (Verheugen et al., 1999;Chittajallu et al., 2004;Banke and McBain, 2006). This approach is based on ascertaining the reversal potential of K^+-currents through cell-attached patch to estimate Vm. The intracellular patch solution contains the estimated intracellular K^+-concentration and is of the following composition (in mM); KCl 150, $CaCl_2$ 2, $MgCl_2$ 1, Hepes 10; 0.3 % biocytin; adjusted to pH 7.4 with KOH (final K^+ concentration 155mM) and osmolarity was 270-290 mOsm. Extracellular solution was as previously described above. After the cell-attached patch was attained, a voltage ramp protocol (-120 to +180 mV) was applied to activate voltage-gated K^+-channels. Under the conditions of the experiment, the equilibrium potential for K^+ (E_K) across the patch is approximately 0 mV. Thus K^+ currents will reverse when the pipette

potential (Vpip) cancels out Vm, giving a direct quantitative measure for the cells membrane potential (i.e. at K^+ reversal, Vm +Vh = Ek ~ 0 mV). Note: in cell attached patches Vh = - Vpip. The liquid junction potential was minimal (calculated at 2 mV) and therefore was not corrected for in these experiments.

REFERENCES

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