Supplemental material

**Fig. S1.** The passive membrane properties of 6 excitatory spiny neurons and 4 smooth basket cells were estimated by direct fitting of the simulated potentials to the experimentally measured voltage responses during a series of somatic current injections. The fit was applied to the first 100 ms (in one neuron 50 ms) of the on- and off- set responses (start and end of pulse, marked by the dashed lines and the dashed-dotted lines, respectively), which included the transient RC response and the steady state depolarization. The simulations were made in NEURON based on the 3D reconstruction (including soma, dendrites and axons) and the data (I-V) obtained from each individual neuron. An evolutionary algorithm was applied to search the parameter space and was instructed by a least squares minimization error to find the best matching $C_m$, $R_m$ and $R_i$ for each neuron. The search ranges were as follows: $C_m$ 0.3-2 $\mu$F/cm$^2$, $R_m$ 2000-28,000 $\Omega$/cm$^2$, $R_i$ 60-220 $\Omega$. Spines were incorporated in the spiny neurons by dividing $C_m$ and $1/R_m$ in the dendrites by a factor of 1.8 (see Methods for explanation). **A-B.** Show an example of the experimental (black lines) and fitted traces (red lines) of a spiny stellate (A, shown also as the postsynaptic neuron in Fig. 2B) and smooth basket (B, cell shown also as the postsynaptic neuron in Fig. 2F) neurons.

**C-D.** The averaged membrane properties of the smooth basket cells and the spiny neurons. The individual experiments are represented by the open symbols and their averages by the corresponding filled symbol. Data are plotted ± s.d.

Fitted values were as follows: smooth cells: $C_m = 1.3\pm0.44$ $\mu$F/cm$^2$ (range: 0.76-1.84); $R_m = 3938\pm2134$ $\Omega$cm$^2$ (range: 2006-6987) and $R_i = 118\pm55$ $\Omega$cm (range: 60-173) (Fig. S1). Excitatory neurons (n=6) without spines: $C_m = 1.2\pm0.58$ $\mu$F/cm$^2$ (range: 0.4-1.82); $R_m = 11124\pm6755$ $\Omega$cm$^2$ (range: 5270-22000) and $R_i = 134\pm46$ $\Omega$cm (range: 72-181) (Fig. S1). Excitatory neurons with spine scaling factor of 1.8: $C_m = 0.85\pm0.34$ $\mu$F/cm$^2$ (range: 0.35-1.23); $R_m = 16107\pm6921$ $\Omega$cm$^2$ (range: 9723-27516) and $R_i = 128\pm57$ $\Omega$cm (range: 67-219) (Fig. S1).

Spiny neurons without spine scaling had higher $R_m$ values compared to the smooth basket neurons but similar $C_m$ and $R_i$ values. Incorporation of spines increased $R_m$ and decreased $C_m$ but did not affect the $R_i$. The time constant ($R_m*C_m$) of the spiny neurons was therefore not much affected by the spine scaling.

**Fig. S2.** Simulations of the EPSP propagation in the excitatory spiny neurons. Comparison of the kinetics with and without incorporating spines. Simulations were repeated in 6 excitatory
neurons with (A) and 4 without (B) incorporating spines. Spines were incorporated by scaling Rm and Cm in the dendrites by a factor 1.8 (see Methods for details). Attenuation (a) plots of the EPSP amplitudes, defined as the ratio between EPSP spine and EPSP soma. In both cases (A, with / B, without spine scaling) attenuation varied up to 40 folds. EPSP latencies (b) and rise time (c) were plotted against the dendritic distance from the soma. The incorporations of spines had only a very small effect on either of these distributions. The overlap compartments (red marks) were likewise nearly identical in both cases. This results from the fact that we directly fitted the experimental data of each neuron with its own morphology, such that in either case (with/without spines) the fitted Rm, Cm, Ri were different for each neuron, but provided similarly good fits (Holmes et al., 2006). The fit algorithms, thus, found the parameters that best reproduce these data. We have also tried a lower spine scaling factors 1.3 (based only on our own LM data) and found that the fitted parameters remained unchanged compared with the case of no spine scaling. A higher spine scaling factor of 2, produced in some of the neurons larger deviations (and worse fits) from the experimental data. We thus concluded that even if the scaling factor we used of 1.8 was not perfect for each individual neuron, the true factor must lie within that range.

Fig. S3. Plots of the EPSP attenuation (a), latencies (b) and rise time (c) distributions on the inhibitory basket neurons simulated with a slow (identical in kinetics to the E-E conductance shown in Fig 7 and in S2). Latencies were and rise times were significantly longer under with the slow synapse, however, overlap compartments that reproduce the experimentally observed data were very few and detected in one neuron. More than 99% of the compartments yielded latencies and rise times much longer than those observed for the E-I synaptic connections. These data suggest that in order to reproduce the experimental data, a faster conductance must underlie the E-I EPSPs.

Fig. S4. Simulations of spiking in the recurrent network in three cases: Control case (blue lines and symbols) where all recurrent synapses had equal slow kinetics and the same latency (1.4 ms). Second case (red lines and symbols) in which all recurrent synapses had the same latency (1.4 ms) but the E-I synapses had faster kinetics. Third case (green lines and symbols) in which all synapses had the same slow kinetics but the latency of E-I was shorter (0.5 ms). A-D Plots the firing of the excitatory and E-H of the inhibitory neurons. (A; E) Plots of the membrane potential of an exemplary excitatory neuron (A) and of an inhibitory neuron (E). Note that the first AP evoked by the thalamic input overlaps in all three cases (B; F), Spiking-times raster plots. (C; G) Peri stimulus histograms (PSTH) of the mean excitatory (C) and inhibitory (G) population rates. (D; H) Mean excitatory and inhibitory currents of all
excitatory (D) and inhibitory (H) cells. The simulations show that fast kinetics of E-I synapses (second case, red) halves the total number of spikes in the excitatory neurons and cuts the recurrent activity short after 20 ms compared to 40 ms in the control case (blue). The initial (at times 7-12 ms) mean population firing rate is also slightly reduced in the excitatory neurons before the mean inhibitory current at ~16 ms overweighs the excitatory current and stops the recurrent firing. In the inhibitory neurons the total number of spikes and the duration of spiking are likewise halved in the case of fast E-I synapses (second case, red), but the initial firing rate is increased. This results from an increase in the synchrony of the excitatory input current (sharper positive peaks in H).

In the third case (green), shorter latency of E-I synapses reduces the total number of spikes and the duration of recurrent firing in excitatory and inhibitory neurons by about 30% compared to control case. The major difference to the second case is that the firing rates of both neuronal populations are evenly reduced during the entire recurrent firing time.

Reference List