Supplemental Results

To further test the robustness of our main result we constructed a set of active cortex models with different physiological properties: all synapse types display strong short-term depression (STD) with U= 0.25 (model 1); none of the synapses display STD (model 2); none of the synapses display STD and PY cells lack I_{KCa} (model 3); PY cells lack I_{KCa} (model 4); PY-PY synapses lack an NMDA component (model 5); the NMDA:AMPA ratio at PY-PY synapses is 0.3 instead of 0.15 (model 6); each IN cell is electrically coupled to on average 10 neighboring IN cells randomly chosen within the synaptic footprint (the average summated coupling conductance that each cell receives equals the leak conductance of individual IN cells) (model 7); IN cells fire at 5 Hz instead of 10 Hz (model 8); PY cells fire at 10 Hz (homeostasis target firing rate T= 10 Hz) and IN cells at 20 Hz (model 9). Two other models assumed different contributions of synaptic activity to input conductances: $\bar{g}_{\text{IN-PY}}$ and $\bar{g}_{\text{IN-IN}}$ were either doubled (model 10) or halfed (model 11). A final set of models considered different values of $r_{PY}= G_{PY-PY}/(G_{PY-PY}+G_{ex-PY})$ and $r_{IN}= G_{PY-IN}/(G_{PY-IN}+G_{ex-IN})$ in the default model: $r_{PY}= r_{IN}= 0.1$ (model 12), $r_{PY}= r_{IN}= 0.4$ (model 13) and $r_{PY}= 0.25$ and $r_{IN}= 0.125$ (model 14).

The amount of HSP that was required to reach the homeostasis target firing rate after deafferentation varied between the models (Fig. S1, top panel). In particular, models 3, 10, 13 and 14 required smaller amounts (~40% HSP) than the default model (~60% HSP, model 0) whereas model 12 required twice the amount. Nevertheless, homeostatic synaptic plasticity restored bursting states in all models except model 3, although with differences in frequency, intensity and duration of burst discharges (Fig. S1, LNP traces). The results of model 3 indicate that either STD or I_{KCa} was required in PY cells to develop bursting. Two other interesting observations were made: (1) In model 2, burst discharges propagated for short distances only and the average firing rate F varied sharply with small changes in the amount of HSP in the steady state. (2) In model 12, even very large HSP values (up to 200%) failed to restore firing rates in fully deafferented cortex because of the small $\bar{g}_{\text{PY-PY}}$ values (results are shown in partially (90%) deafferented cortex).

The sparseness of synaptic connections and the size of unitary EPSCs and IPSCs did not affect burst discharge properties (as long as total conductance values remained unchanged). The amount of HSP required to evoke bursts, their duration, number of spikes per cell and speed of propagation were similar in models with 2-fold changes in sparseness and/or number of synaptic connections per cell. A gaussian synaptic footprint shape (σ= 0.4 radius of connectivity) instead of the default square footprint did not affect burst properties except that propagation was slower (1.72 cm/s versus 2.83 cm/s at 70% HSP). In models with dense local synaptic connections (1,000 PY and 250 IN cells) relatively large HSP values (≥53%) were required to evoke propagating burst discharges compared to the sparse default model (≥47%), but all other burst measures were similar. Also, burst discharges sometimes failed to propagate at HSP values of 53-61%, which was never observed in the sparse model (at HSP values ≥47%). Surprisingly, the amount

of HSP to reach the homeostasis target firing rate was lower in the dense model than in the sparse model (63% versus 69%).

Besides glutamatergic inputs, cholinergic and other neuromodulatory inputs may contribute to the spontaneous firing of cells in intact cortex. Activation of cholinergic synapses increases firing rates of pyramidal neurons by decreasing an M-type K^+ current and a Ca^{2+}-activated K^+ current (McCormick, 1992). In large burst-firing layer 5 pyramidal cells, ACh causes an additional decrease in resting K^+ conductance. The excitability of cortical interneurons is differentially regulated by ACh, causing depolarization in some GABAergic subtypes while hyperpolarization in others (Kawaguchi, 1997; Xiang et al., 1998). Cholinergic effects may be incorporated in our active cortex model by reducing $\bar{g}_{M}$, $\bar{g}_{KCa}$ and $\bar{g}_{K,leak}$ in PY (and IN) cells and reducing extrinsic glutamatergic excitation to keep firing rates fixed. The resulting deafferented model, deprived of both extrinsic glutamatergic and cholinergic inputs, is identical to the default deafferented model. Thus, our results do not depend on the exact implementation of cholinergic effects on intrinsic neuronal excitability.

References

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