

Cell type specific contributions of inhibitory neuron to sensory representations

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Inhibitory neurons, which comprise 15~20% of cortical neuron population, have diverse morphological, biochemical and physiological characteristics and thus can be divided into several subgroups. Vasoactive Intestinal Peptide (VIP) positive interneurons, for example, is one of the most common forms of inhibitory neurons and comprise ~30% of the interneuron population in the neocortex. They receive depressing input from the excitatory pyramidal cells and form, in return, inhibitory synapses which do not undergo short-term adaptation in synaptic efficacy upon prolonged activation. On the contrary, α -actinin-2 positive (i.e. Neuroglia form or NGF) cells comprise ~8% of the neocortical interneuron population. They receive depressing excitatory input from pyramidal neurons and their inhibitory projections show significant short-term adaptation. Despite that the pattern of axonal and dendritic projections and properties of synapses made by and made onto the diverse subtypes of inhibitory neurons are well studied, contribution of the variety of inhibitory neurons to sensory representations is unclear. Here we start to address this question using a computational model of the stereotypical barrel cortex column whose network topology is based on the statistics of the cell type, density, localization and cell type specific dendritic and axonal projections observed in the rodent somatosensory cortex.

Using the model cortical column, we show that iterative stochastic removal of the subset of VIP+ cells results in significant disinhibition of excitation in the L2/3 pyramidal cells of the cortical column (0.58 \pm 0.02 firing probability 100% VIP+ neurons (Control) vs 0.66 \pm 0.02 firing probability no VIP+; $P < 0.001$). Interestingly disinhibition of excitation does not reduce the onset latency of the population Peri-Stimulus Time Histogram (pPSTH onset; 13.3 \pm 0.4 vs 13.3 \pm 0.4; $P > 0.9$). Neither does it change the latency of the pPSTH peak (14.6 \pm 0.2 vs 14.6 \pm 0.2; $P > 0.9$) arguing that VIP+ neuron mediated inhibition does not contribute to the early phase of sensory representations during which stimulus evoked responses propagate within the cortical column. Targeted removal of the L2/3 VIP+ neurons, nevertheless, facilitates stimulus evoked excitation. Comparisons of the late phase pPSTH (>18 ms after stimulus onset) showed not only a 1.4 fold (\pm 0.1 vs Control; $P < 0.001$) increased excitability but also prolongation of the spiking window (7.5 \pm 0.4 vs 10.2 \pm 0.6; $P < 0.001$) in the absence of VIP+ mediated inhibition. Results presented were specific to VIP+ neurons as removal of the NGF+ cells did not alter the stimulus evoked excitability in L2/3. These results support the conclusion that VIP, but not NGF, mediated inhibition regulate the maintenance, rather than propagation, of the sensory evoked responses in a cortical column.