3.2 Exploratory analysis of a theoretical model

Sean Bittner and John Cunningham, September 15, 2019

Dynamical models with two populations (excitatory (E) and inhibitory (I) neurons) of visual processing have been used to reproduce a host of experimentally documented phenomena in V1. When an inhibition stabilized network (ISN, the I population stabilizes an otherwise unstable E population), these models exhibit the paradoxical effect [1], selective amplification [2], surround suppression [3], and sensory integrative properties [4]. Since I neurons mostly fall into one of three classes (parvalbumin (P)-, somatostatin (S)-, and vasointestinal peptide (V)-expressing neurons) [5, 6], theorists look to extend these dynamical models to four populations [7]. A current challenge in theoretical neuroscience is understanding the distributed role of inhibition stabilization across these subtypes.

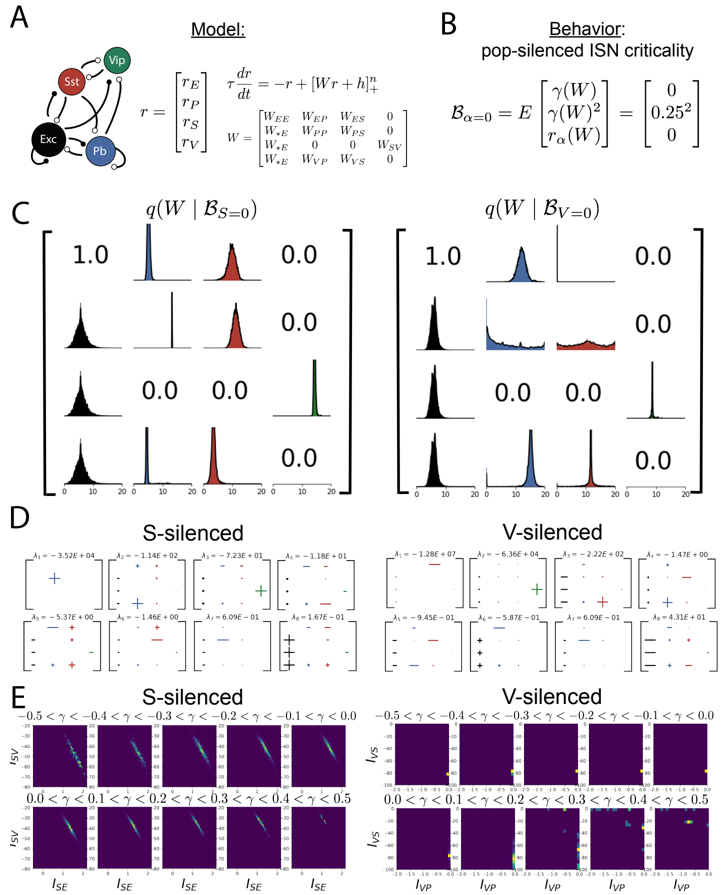

Figure 1: A.) Model of primary visual cortex (V1) Neurons: E (black), P (blue), S (red), and V (green). Parameters: weights of the dynamics matrix W. B.) The DSNs are conditioned on population-silenced ISN criticality. C.) DSN distribution of the parameters of the V1 model conditioned on population-silened ISN criticality. D.) Eigenmodes of the hessian of each DSN ordered by eigenvalue. E. Input to silenced population across ISN regimes of the DSN posterior.

These four populations exhibit neuron-type specific connectivity (Fig. 1A) [8], in which some populations do not project to others. Since S and V are the only populations that mutually inhibit each other, a popular conceptualization is that S and V have winner-take-all dynamics. In fact, evidence in mice suggests that V silences S when presented with large stimuli, and S silences V for small stimuli [9]. Here, we use DSNs to understand the possible sources of inhibition stabilization in this V1 model, when either S or V is inactive. The behavior of the DSN distributed models are constrained to produce two things: 1.) a mean-zero distribution of ISN coefficients $\gamma(W) = 1 - f^{'}(f^{-1}(r_E(W)))W_{EE}$ with some variance, and 2.) α-population silencing $r_\alpha(W) = 0$, for $\alpha \in \{S, V\}$ (Fig. 1B). When $\gamma < 0$ the network is ISN, and not ISN otherwise. Constraining the DSN behavior to a zero-mean distribution of ISN coefficients gives us samples of both ISN and non-ISN networks, optimizied to have greatest variety of stabilization motifs.

When optimized to produce a variety of stabilization motifs, there are informative differences between S-silenced and V-silenced DSN posteriors. The marginal posteriors for each weight matrix element (W_{EE} is fixed to 1.0, and W_{*E} is one parameter), are visualized by their location in the dynamics matrix (Fig. 1C). Low-variance marginals, like $q_\theta(W_{PP} \mid \mathcal{B}_{S=0})$, $q_\theta(W_{VP} \mid \mathcal{B}_{S=0})$, and $q_\theta(W_{SV} \mid \mathcal{B}_{S=0})$, indicate that either the $\gamma(W)$, S-silencing, or both are sensitive to changes in such parameters. Whereas, $q_\theta(W_{PP} \mid \mathcal{B}_{V=0})$ and $q_\theta(W_{PS} \mid \mathcal{B}_{V=0})$ have high variance indicating degeneracy with respect to $\gamma(W)$ and V-silencing.

As with the STG circuit, we evaluate the Hessian of the DSN posterior at $\gamma(W) = 0$, and visualize the eigendecompositions ordered by eigenvalues (Fig. 1D). In accordance with the marginals, W_{PP}, W_{VP}, and W_{SV} are pronounced in the Hessian eigenvectors with the greatest magnitude eigenvalues. The low magnitude eigenvalues indicate degenerate dimensions of the weight matrix w.r.t. $\mathcal{B}_{\alpha=0}$.

Having a distribution optimized to be as random as possible allows us to see the variety of way in which populations are silenced across ISN regimes. We show how E- and V-input to a silenced S population and P- and S- input to a silenced V population change with ISN regime (Fig. 1E).

Sean Bittner
srb2201@columbia.edu

References

[1] Misha V Tsodyks, William E Skaggs, Terrence J Sejnowski, and Bruce L McNaughton. Paradoxical effects of external modulation of inhibitory interneurons. *Journal of neuroscience*, 17(11):4382–4388, 1997.

[2] Brendan K Murphy and Kenneth D Miller. Balanced amplification: a new mechanism of selective amplification of neural activity patterns. *Neuron*, 61(4):635–648, 2009.

[3] Hirofumi Ozeki, Ian M Finn, Evan S Schaffer, Kenneth D Miller, and David Ferster. Inhibitory stabilization of the cortical network underlies visual surround suppression. *Neuron*, 62(4):578–592, 2009.

[4] Daniel B Rubin, Stephen D Van Hooser, and Kenneth D Miller. The stabilized supralinear network: a unifying circuit motif underlying multi-input integration in sensory cortex. *Neuron*, 85(2):402–417, 2015.

[5] Henry Markram, Maria Toledo-Rodriguez, Yun Wang, Anirudh Gupta, Gilad Silberberg, and Caizhi Wu. Interneurons of the neocortical inhibitory system. *Nature reviews neuroscience*, 5(10):793, 2004.

[6] Bernardo Rudy, Gordon Fishell, SooHyun Lee, and Jens Hjerling-Leffler. Three groups of interneurons account for nearly 100% of neocortical gabaergic neurons. *Developmental neurobiology*, 71(1):45–61, 2011.

[7] Ashok Litwin-Kumar, Robert Rosenbaum, and Brent Doiron. Inhibitory stabilization and visual coding in cortical circuits with multiple interneuron subtypes. *Journal of neurophysiology*, 115(3):1399–1409, 2016.

[8] Carsten K Pfeffer, Mingshan Xue, Miao He, Z Josh Huang, and Massimo Scanziani. Inhibition of inhibition in visual cortex: the logic of connections between molecularly distinct interneurons. *Nature neuroscience*, 16(8):1068, 2013.

[9] Mario Dipoppa, Adam Ranson, Michael Krumin, Marius Pachitariu, Matteo Carandini, and Kenneth D Harris. Vision and locomotion shape the interactions between neuron types in mouse visual cortex. *Neuron*, 98(3):602–615, 2018.