Hierarchical Selective Recruitment in Linear-Threshold Brain Networks
Part I: Intra-Layer Dynamics and Selective Inhibition

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Abstract—Goal-driven selective attention (GDSA) refers to the brain’s function of prioritizing, according to one’s internal goals and desires, the activity of a task-relevant subset of its overall network to efficiently process relevant information while inhibiting the effects of distractions. Despite decades of research in neuroscience, a comprehensive understanding of GDSA is still lacking. We propose a novel framework for GDSA using concepts and tools from control theory as well as insights and structures from neuroscience. Central to this framework is an information-processing hierarchy with two main components: selective inhibition of task-irrelevant activity and top-down recruitment of task-relevant activity. We analyze the internal dynamics of each layer of the hierarchy described as a network with linear-threshold dynamics and derive conditions on its structure to guarantee existence and uniqueness of equilibria, asymptotic stability, and boundedness of trajectories. We also provide mechanisms that enforce selective inhibition using the biologically-inspired schemes of feedforward and feedback inhibition. Despite their differences, both schemes lead to the same conclusion: the intrinsic dynamical properties of the (not-inhibited) task-relevant subnetworks are the sole determinant of the dynamical properties that are achievable under selective inhibition.

I. INTRODUCTION

The human brain is constantly under the influx of sensory inputs and is responsible for integrating and interpreting them to generate appropriate decisions and actions. This influx contains not only the pieces of information relevant to the present task(s), but also a myriad of distractions. Goal-driven selective attention (GDSA) refers to the active selective processing of a subset of information influx while suppressing the effects of others, and is vital for the proper function of the brain. Examples of GDSA range from selective audition in a crowded place to selective vision in cluttered environments to selective taste/smell in food. As a result, a long standing question in neuroscience involves understanding the brain’s complex mechanisms underlying selective attention [2]–[7].

A central element in addressing this question is the role played by the hierarchical organization of the brain [8]. Broadly, this organization places primary sensory and motor areas at the bottom and integrative association areas (prefrontal cortex in particular) at the top. Accordingly, sensory information is processed while flowing up the hierarchy, where decisions are eventually made and transmitted back down the hierarchy to generate motor actions. The top-down direction is also responsible for GDSA, where the higher-order areas differentially “modulate” the activity of the lower-level areas such that only relevant information is further processed. This phenomenon, hereafter called hierarchical selective recruitment (HSR), constitutes the basis for GDSA and has been the subject of extensive experimental research in neuroscience, see e.g., [4], [9]–[18]. However, a complete understanding of how, when (how quick), or where (within the hierarchy) it occurs is still lacking. In particular, the relationship between HSR and the dynamics of the involved neuronal networks is poorly understood. Our goal is to address this gap from a model-based perspective, resorting to control-theoretic tools to explain various aspects of HSR in terms of the synaptic network structure and the dynamics that emerge from it.

The starting point in the development of HSR is the observation that different stimuli, in particular the task-relevant and task-irrelevant ones, are processed by different populations of neurons. With each neuronal population represented by a node in the overall neuronal network of networks, HSR primarily relies on the selective inhibition of the task-irrelevant nodes and the top-down recruitment of the task-relevant nodes of each layer by the layer immediately above. This scheme is motivated by the observations that GDSA is not confined to a single layer, but is rather distributed in a graded manner along the thalamocortical hierarchy [8]. Here, we analyze the dynamics of individual layers as well as the mechanisms for selective inhibition in a bilayer network. The results here set the basis for the study of the mechanisms for top-down recruitment in multilayer networks in our accompanying work [19].

Literature review

In this work we use dynamical networks with linear-threshold nonlinearities (also called rectified linear units, ReLU, in machine learning) to model the activity of neuronal populations. Linear-threshold models allow for a unique combination between the tractability of linear systems and the dynamical versatility of nonlinear systems, and thus have been widely used in computational neuroscience. They were first proposed as a model for the lateral eye of the horseshoe

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1Note the distinction of this with stimulus-driven selective attention (the reactive shift of focus based on saliency of stimuli) which is not the focus of this work.

2Note that the role of memory (being distributed across the brain) is implicit in this simplified stimulus-response description. Indeed, many sensory inputs only form memories (without motor response) or many motor actions result chiefly from memory (without sensory stimulation). The hierarchical aspect is, nevertheless, always present.

3We focus primarily on GDSA because of its long history in neuroscience. Nevertheless, HSR is a generic framework for top-down discrimination between two or more groups of competing populations that may represent thoughts, memories, emotions, or motor actions in addition to sensory information.
crab in [20] and their dynamical behavior has been studied at least as early as [21]. A detailed stability analysis of symmetric (undirected) linear-threshold networks has been carried out in continuous [22] and discrete [23] time: however, this has limited relevance for biological neuronal networks, which are fundamentally asymmetric (due to the presence of excitatory and inhibitory neurons). An initial summary of the properties of general (possibly asymmetric) networks, including the existence and uniqueness of equilibria and asymptotic stability was given in [24], with limited rigorous justification provided later in [25]. Lyapunov-based methods were used in a number of later studies for discrete-time linear-threshold networks [26]–[28], but the extension of these results to continuous-time dynamics, which has more relevance to biological neuronal networks, is not clear. In fact, the use of Lyapunov-based techniques in continuous-time networks has remained limited to planar dynamics [29] and restrictive conditions for boundedness of trajectories [29], [30]. Recently, [31] presents interesting properties of competitive (i.e., fully inhibitory) linear-threshold networks, particularly regarding the emergence of limit cycles. However, the majority of neurons in biological neuronal networks are excitatory, making the implications of these results limited. Moreover, all the preceding works are limited to networks with constant exogenous inputs whereas time-varying inputs are essential for modeling inter-layer connections in HSR.

A critical property of linear-threshold networks is that their nonlinearity, while enriching their behavior beyond that of linear systems, is piecewise linear. Accordingly, almost all the theoretical analysis of these networks builds upon the formulation of them as switched affine systems. There exists a vast literature on the analysis of general switched linear/affine systems, see, e.g., [32]–[34]. Nevertheless, we have found that the conditions obtained by applying these results to linear-threshold dynamics are more conservative than the ones we obtain using direct analysis of the system dynamics. This is mainly due to the fact that such results, by the essence of their generality, are oblivious to the particular structure of linear-threshold dynamics that can be leveraged in direct analysis.

Selective inhibition has been the subject of extensive research in neuroscience. A number of early studies [4], [11], [12] provided evidence for a mechanism of selective visual attention based on a biased competition between the subnetwork of task-relevant nodes and the subnetwork of task-irrelevant ones. In this model, nodes belonging to these subnetworks compete at each layer by mutually suppressing the activity of each other, and this competition is biased towards task-relevant nodes by the layer immediately above. Later studies [13], [14] further supported this theory using functional magnetic resonance imaging (fMRI) and showed [35], in particular, the suppression of activity of task-irrelevant nodes as a result of GDSA. This suppression of activity is further shown to occur in multiple layers along the hierarchy [36], grow with increasing attention [37], [38], and be inversely related to the power of the task-irrelevant nodes’ state trajectories in the alpha frequency band ($\sim 8-14$ Hz) [16]. Here, we use insights from this body of work in developing a theoretical framework for selective inhibition.

**Statement of contributions**

The contributions of the paper are twofold. First, we analyze the internal dynamics of a single-layer linear-threshold network as a basis for our study of hierarchical structures. Our results are a combination of previously known results (for which we give simpler proofs) and novel ones, providing a comprehensive characterization of the dynamical properties of linear-threshold networks. Specifically, we show that existence and uniqueness of equilibria, asymptotic stability, and boundedness of trajectories can be characterized using simple algebraic conditions on the network structure in terms of the class of P-matrices (matrices with positive principal minors), totally-Hurwitz matrices (those with Hurwitz principal submatrices, shown to be a sub-class of P-matrices), and Schur-stable matrices, respectively. In addition to forming the basis of HSR, these results solve some of the long-standing open problems in the characterization of linear-threshold networks and are of independent interest. Our second contribution pertains the problem of selective inhibition in a bilayer network composed of two subnetworks. Motivated by the mechanisms of inhibition in the brain, we study feedforward and feedback inhibition mechanisms. We provide necessary and sufficient conditions on the network structure that guarantee selective inhibition of task-irrelevant nodes at the lower-level while simultaneously guaranteeing various dynamical properties of the resulting (partly inhibited, partly active) subnetwork, including existence and uniqueness of equilibria and asymptotic stability. Interestingly, under both mechanisms, these conditions require that (i) there exist at least as many independent inhibitory control inputs as the number of nodes to be inhibited, and (ii) the (not-inhibited) task-relevant part of the lower-level subnetwork intrinsically satisfies the desired dynamical properties. Therefore, when sufficiently many inhibitory control inputs exist, the intrinsic dynamical properties of the task-relevant part are the sole determiner of the dynamical properties achievable under feedforward and feedback inhibitory control. This is particularly important for selective inhibition as asymptotic stability underlies it. These results unveil the important role of task-relevant nodes in constraining the dynamical properties achievable under selective inhibition and have further implications for the number and centrality of nodes that need to be inhibited in order for an unstable-in-isolation subnetwork to gain stability through selective inhibition. For subnetworks that are not stable as a whole, these results provide conditions on the task-relevant/irrelevant partitioning of the nodes that allow for stabilization using inhibitory control.

**II. Preliminaries**

Here, we introduce notational conventions and review basic concepts on matrix analysis and modeling of biological neuronal networks.

**Notation**

We use $\mathbb{R}$, $\mathbb{R}_{\geq 0}$, and $\mathbb{R}_{\leq 0}$ to denote the set of reals, nonnegative reals, and nonpositive reals, respectively. We use bold-faced letters for vectors and matrices. $\mathbf{1}_n$, $\mathbf{0}_n$, $\mathbf{0}_{m \times n}$, and $\mathbf{I}_n$ stand for the $n$-vector of all ones, the $n$-vector of all
zeros, the \(m\)-by-\(n\) zero matrix, and the identity \(n\)-by-\(n\) matrix (we omit the subscripts when clear from the context). Given a vector \(x \in \mathbb{R}^n\), \(x_i\) and \((x)_i\) refer to its \(i\)th component. Given \(A \in \mathbb{R}^{m \times n}\), \(a_{ij}\) refers to the \((i,j)\)th entry. For block-partitioned \(x\) and \(A\), \(x_i\) and \(A_{ij}\) refer to the \(i\)th block of \(x\), \(i\)th block (e.g., row) of \(A\), and \((i,j)\)th block of \(A\), respectively. In block representation of matrices, \(\ast\) denotes arbitrary blocks whose value is immaterial to the discussion. For \(A \in \mathbb{R}^{n \times n}\), range(\(A\)) denotes the subspace of \(\mathbb{R}^n\) spanned by the columns of \(A\). If \(x\) and \(y\) are vectors, \(x \leq y\) denotes \(x_i \leq y_i\) for all \(i\). For symmetric \(P \in \mathbb{R}^{n \times n}\), \(P > 0\) (\(P < 0\)) denotes that \(P\) is positive (negative) definite. Given \(A \in \mathbb{R}^{n \times n}\), its element-wise absolute value, determinant, spectral radius, and induced norm-2 are denoted by \(|A|\), \(\det(A)\), \(\rho(A)\), and \(\|A\|\), respectively. Similarly, for \(x \in \mathbb{R}^n\), \(\|x\|\) is its 2-norm. For \(\sigma \in \{0, 1\}^n\), we make the convention that \(\Sigma = \text{diag}(\sigma) \in \mathbb{R}^{n \times n}\) denotes the diagonal matrix with the elements of \(\sigma\) on its diagonal. Likewise, for two matrices \(A\) and \(B\), \(\text{diag}(A, B)\) denotes the block-diagonal matrix with \(A\) and \(B\) on its diagonal. Given a subspace \(W\) of a vector space \(V\), \(W^\perp\) denotes the orthogonal complement of \(W\) in \(V\). For a set \(S\), \(|S|\) denotes its cardinality. For \(x \in \mathbb{R}\), \([x]^+ = \max\{0, x\}\), which is extended entry-wise to vectors and matrices.

In \(\mathbb{R}^n\), a hyper-plane with normal vector \(n \in \mathbb{R}^n\) passing through \(x \in \mathbb{R}^n\) is the \((n-1)\)-dimensional subspace \([y \in \mathbb{R}^n \mid n^T(x - y) = 0]\). A set of \(n\) hyperplanes is degenerate [39] if their intersection is a point or, equivalently, the matrix formed by their normal vectors is nonsingular. A set \(S \subseteq \mathbb{R}^n\) is called

- a polytope if it has the form \(S = \{x \in \mathbb{R}^n \mid Ax \leq b\}\) for some \(A \in \mathbb{R}^{m \times n}, b \in \mathbb{R}^m, m \in \mathbb{N}\),
- a cone if \(ex \in S\) for any \(e \in S\) and \(c \in \mathbb{R}_{\geq 0}\),
- a translated cone apexed at \(y\) if \(\{x + y \in S \mid x \in \mathbb{R}^n\}\) is a cone,
- convex if \((1 - \theta)x + \theta y \in S\) for any \(x, y \in S, \theta \in [0, 1]\),
- solid if it has a non-empty interior.

Throughout the paper, measure-theoretic statements are meant in the Lebesgue sense.

**Matrix Analysis**

Here, we define and characterize several matrix classes of interest and their inclusion relationships. These matrices play a key role in the forthcoming discussion.

**Definition II.1. (Matrix classes).** A matrix \(A \in \mathbb{R}^{n \times n}\) (not necessarily symmetric) is

(i) absolutely Schur stable if \(\rho(|A|) < 1\);
(ii) totally \(\mathcal{L}\)-stable, denoted \(A \in \mathcal{L}\), if there exists \(P = P^T > 0\) such that \((-I + A^T\Sigma P + P(-I + \Sigma A) < 0\) for all \(\Sigma = \text{diag}(\sigma)\) and \(\sigma \in \{0, 1\}^n\);
(iii) totally Hurwitz, denoted \(A \in \mathcal{H}\), if all the principal submatrices of \(A\) are Hurwitz;
(iv) a P-matrix, denoted \(A \in \mathcal{P}\), if all the principal minors of \(A\) are positive.

In working with P-matrices, the principal pivot transform of a matrix plays an important role. Given

\[
A = \begin{bmatrix} A_{11} & A_{12} \\ A_{21} & A_{22} \end{bmatrix},
\]

with nonsingular \(A_{22}\), its principal pivot transform is the matrix

\[
\pi(A) \triangleq \begin{bmatrix} A_{11} - A_{12}A_{22}^{-1}A_{21} & A_{12}A_{22}^{-1} \\ -A_{22}^{-1}A_{21} & A_{22}^{-1} \end{bmatrix}.
\]

Note that \(\pi(\pi(A)) = A\). The next result formalizes several equivalent characterizations of P-matrices.

**Lemma II.2. (Properties of P-matrices [40], [41]).** \(A \in \mathbb{R}^{n \times n}\) is a P-matrix if and only if any of the following holds:

(i) \(A^{-1}\) is a P-matrix;
(ii) all real eigenvalues of all the principal submatrices of \(A\) are positive;
(iii) for any \(x \in \mathbb{R}^n\) there is \(k\) such that \(x_k(A)x_k > 0\);
(iv) the principal pivot transform of \(A\) is a P-matrix.

The next result states inclusion relationships among the matrix classes in Definition II.1 that will be used in our ensuing discussion.

**Lemma II.3. (Inclusions among matrix classes).** For \(A, W \in \mathbb{R}^{n \times n}\), we have

(i) \(\rho(|W|) < 1 \Rightarrow -I + W \in \mathcal{H}\);
(ii) \(\|W\| < 1 \Rightarrow W \in \mathcal{L}\);
(iii) \(W \in \mathcal{L} \Rightarrow -I + W \in \mathcal{H}\);
(iv) \(A \in \mathcal{H} \Rightarrow -A \in \mathcal{P}\).

**Proof.** (i). From [42, Fact 4.11.19], we have that \(\rho(|W|) < 1\) for any principal submatrix \(W_\sigma\) of \(W\), which in turn implies \(\rho(W_\sigma) < 1\) by [42, Fact 4.11.17], implying the result.

(ii) It is straightforward to check that \(P = I_n\) satisfies \((-I + W^T\Sigma P + P(-I + \Sigma W) < 0\) for all \(\Sigma = \text{diag}(\sigma)\), \(\sigma \in \{0, 1\}^n\).

(iii) Pick an arbitrary \(\sigma \in \{0, 1\}^n\) and let the permutation \(\Pi \in \mathbb{R}^{n \times n}\) be such that

\[
\Pi \Sigma W \Pi^T = \begin{bmatrix} 0 & 0 \\ \hat{W}_{21} & W_{22} \end{bmatrix},
\]

where \(\hat{W}_{22}\) is the principal submatrix of \(W\) corresponding to \(\sigma\). Then

\[
P(-I + \Sigma W) = \Pi P^T \begin{bmatrix} -I & 0 \\ [\hat{W}_{21}] & -I + \hat{W}_{22} \end{bmatrix} \Pi
\]

\[
= \Pi \begin{bmatrix} \Pi P^T \end{bmatrix} \begin{bmatrix} -I & 0 \\ [\hat{W}_{21}] & -I + \hat{W}_{22} \end{bmatrix} \Pi.
\]

where \(\hat{P} = \begin{bmatrix} \hat{P}_{11} & \hat{P}_{12} \\ \hat{P}_{21} & \hat{P}_{22} \end{bmatrix} = \hat{P}^T > 0\). Thus, by assumption,

\[
\Pi^T \begin{bmatrix} * & * \\ \ast & \ast \end{bmatrix} \begin{bmatrix} * & * \\ \ast & \ast \end{bmatrix} \Pi < 0
\]

\[
\Rightarrow \begin{bmatrix} * & * \\ \ast & \ast \end{bmatrix} \begin{bmatrix} * & * \\ \ast & \ast \end{bmatrix} \Pi < 0
\]

\[
\Rightarrow \begin{bmatrix} -I + \hat{W}_{22} \end{bmatrix} \hat{P}_{22} + \hat{P}_{22}(-I + \hat{W}_{22}) < 0,
\]

proving that \(-I + W_{22}\) is Hurwitz. Since \(\sigma\) is arbitrary, \(-I + W\) is totally Hurwitz.
(iv) The result follows from Lemma II.2(ii).

For a general matrix $W$, neither of $\rho(|W|)$ and $\|W\|$ is bounded by the other. However, if $W$ satisfies the Dale’s law (as biological neuronal networks do), i.e., each column is either nonnegative or nonpositive, then $W = |W|D$ where $D$ is a diagonal matrix such that $|D| = I$. Then,

$$\|W\| = \sqrt{\rho(WW^T)} = \sqrt{\rho(|W|DD|W|^T)}$$

$$= \sqrt{\rho(|W||W|^T)} = \|W\| \geq \rho(|W|),$$

showing that, in this case, $\rho(|W|) < 1$ is a less restrictive condition. Figure 1 depicts a Venn diagram of the various matrix classes of interest to help visualize their relationships.

**Dynamical Rate Models of Brain Networks**

Here we briefly review, following [43, §7], the fundamental concepts and assumptions that underlie the linear-threshold network model used throughout the paper. In a lumped model, neural circuits are composed of neurons, each receiving an electrical signal at its dendrites from other neurons and generating an electrical response to other neurons at its axon. The transmission of activity from one neuron to another takes place at a synapse, thus the terms pre-synaptic and post-synaptic for the two neurons, respectively. Both the input and output signals mainly consist of a sequence of spikes (action-potentials), as shown in Figure 2 (top panel), which are modeled as impulse trains of the form

$$\rho(t) = \sum_k \delta(t - t_k),$$

where $\delta(\cdot)$ denotes the Dirac delta function. In many brain areas the exact timing $\{t_k\}$ of $\rho(t)$ seems essentially random, with the information mainly encoded in its firing rate (number of spikes per second). Thus, $\rho(t)$ is modeled as the sample path of an inhomogeneous Poisson point process with rate, say, $x(t)$ (cf. Figure 2, bottom panel).

Now, consider a pair of pre- and post-synaptic neurons with rates $x_{pre}(t)$ and $x_{post}(t)$, respectively. As a result of $x_{pre}(t)$, an electrical current $I_{post}(t)$ forms in the post-synaptic neuron’s dendrites and soma (body). Assuming fast synaptic dynamics, $I_{post}(t) \propto x_{pre}(t)$. Let $w_{post,pre}$ be the proportionality constant, so $I_{post}(t) = w_{post,pre}x_{pre}(t)$. The pre-synaptic neuron is called excitatory if $w_{post,pre} > 0$ and inhibitory if $w_{post,pre} < 0$. In other words, excitatory neurons increase the activity of their out-neighbors while inhibitory neurons decrease it. Notice that this is a property of neurons, not synapses, so a neuron either excites all its out-neighbors or inhibits them.

If the post-synaptic neuron receives input from multiple neurons, $I_{post}(t)$ follows a superposition law,

$$I_{post}(t) = \sum_j w_{post,j}x_j(t),$$

where the sum is taken over its in-neighbors. If $I_{post}$ is constant, the post-synaptic rate follows $x_{post} = F(I_{post})$, where $F$ is a nonlinear “response function”. Among the two widely used response functions, namely, sigmoidal and linear-threshold, we use the latter: $F(\cdot) = [\cdot]^+$. Finally, if $I_{post}(t)$ is time-varying, $x_{post}(t)$ “lags” $F(I_{post}(t))$ with a time constant $\tau$, i.e.,

$$\tau \dot{x}_{post}(t) = -x_{post}(t) + [F(I_{post}(t))]^+.$$  \(2\)

Equations (1)-(2) are the basis for our network model described next.

**III. Problem Formulation**

Consider a network of neurons evolving according to (1)-(2). Since the number of neurons in a brain region is very large, it is common to consider a population of neurons with similar activation patterns as a single node. The “firing rate” of such a node is then defined as the average of the individual firing rates. This convention also has the advantage of getting more consistent rates, as the firing pattern of individual neurons may be at times sparse. Accordingly, we use “node” and “population” interchangeably.\(^4\) Combining the nodal rates in a network model used for neuronal communication (top panel, measured intracellularly [44], [45]) and the corresponding (estimate of) firing rate (bottom panel).

\(^4\)Our discussion is nevertheless valid irrespective of whether network nodes represent individual neurons or groups of them.
works composed of hierarchically-connected linear-threshold subnetworks and the conditions under which their collective dynamics can give rise to HSR. Specifically, we tackle the following problems:

(i) the analysis of the relationship between structure \((\mathbf{W})\) and dynamical behavior (basic properties such as existence and uniqueness of equilibria (EUE), asymptotic stability, and boundedness of trajectories) for each subnetwork when operating in isolation from the rest of the network \((\mathbf{d}(t) \equiv \mathbf{d})\);

(ii) the analysis of the conditions on the joint structure of each two successive layers \(N_i^1\) and \(N_{i+1}^0\) that allows for selective inhibition of \(N_{i+1}^1\) by its input from \(N_i^1\), being equivalent to the stabilization of \(N_{i+1}^1\) to 0 (inactivity);

(iii) the analysis of the conditions on the joint structure of each two successive layers \(N_i^1\) and \(N_{i+1}^0\) that allows for top-down recruitment of \(N_{i+1}^1\) by its input from \(N_i^1\), being equivalent to the stabilization of \(N_{i+1}^1\) toward a desired trajectory set by \(N_i^1\) (activity);

(iv) the combination of (ii) and (iii) in a unified framework and its extension to the complete \(N\)-layer network of networks.

Problems (i) and (ii) are the focus of this paper, whereas we address problems (iii) and (iv) in the accompanying work [19].

IV. INTERNAL DYNAMICS OF SINGLE-LAYER NETWORKS

In this section, we provide an in-depth study of the basic dynamical properties of the network dynamics (3) in isolation. In such case, the external input \(\mathbf{d}(t)\) boils down to background activity and possibly nonzero thresholds, which are constant relative to the timescale \(\tau\). The dynamics (3) thus simplify to

\[
\tau \dot{x}(t) = -x(t) + [\mathbf{W}x(t) + d]^+, \quad t \geq 0. \tag{4}
\]

In the following, we derive conditions in terms of the network structure for EUE, local/global asymptotic stability, and boundedness of trajectories.

A. Dynamics as Switched Affine System

The nonlinear dynamics (4) is a switched affine system with \(2^n\) modes. Each mode of this system corresponds to a switching index \(\sigma \in \{0, 1\}^n\), where for each \(i \in \{1, \ldots, n\}\), \(\sigma_i = 1\) if the node is active (i.e., \((\mathbf{W}x(t) + d)_i > 0\)) and \(\sigma_i = 0\) if the node is inactive (i.e., \((\mathbf{W}x(t) + d)_i \leq 0\)). Clearly, the mode of the system varies with time and within the one corresponding to \(\sigma \in \{0, 1\}^n\), we have

\[
[\mathbf{W}x(t) + d]^+ = \Sigma (\mathbf{W}x(t) + d),
\]

where \(\Sigma = \text{diag}(\sigma)\). This switched representation of the dynamics motivates the following assumptions on the weight matrix \(\mathbf{W}\).

**Assumption 1.** Assume

(i) \(\det(\mathbf{W}) \neq 0\);

(ii) \(\det(\mathbf{I} - \Sigma \mathbf{W}) \neq 0\) for all the \(2^n\) matrices \(\Sigma = \text{diag}(\sigma), \sigma \in \{0, 1\}^n\).

Assumption 1 is not a restriction in practice since the set of matrices for which it is not satisfied can be expressed as a finite union of measure-zero sets, and hence has measure zero. By

![Fig. 3: The hierarchical network structure considered in this work. Each layer is only directly connected to the layers below and above it. Longer-range connections between non-successive layers do exist in thalamocortical hierarchies but are weaker than those between successive layers and are not considered in this work for simplicity.](image-url)
Assumption 1(i), the system of equations $Wx + d = 0$ defines a non-degenerate set of $n$ hyperplanes partitioning $\mathbb{R}^n$ into $2^n$ solid convex polytopic translated cones apexed at $-W^{-1}d$.
For each $\sigma \in \{0,1\}^n$, let $\Omega_\sigma$ be the associated switching region,
\[
\Omega_\sigma = \{ x \in \mathbb{R}^n_{\geq 0} \mid (2\Sigma - I)(Wx + d) \geq 0 \}.
\]
The piecewise-affine dynamics (4) can be written in the equivalent form
\[
\tau x = (-I + \Sigma W)x + \Sigma d, \quad \forall x \in \Omega_\sigma, \; \sigma \in \{0,1\}^n.
\]
Unlike linear systems, the existence of equilibria is not guaranteed for this system. In fact, for each $\sigma \in \{0,1\}^n$, according to (5), the point
\[
x^\ast_\sigma = x^\ast_\sigma(d) = (I - \Sigma W)^{-1}\Sigma d,
\]
is the corresponding equilibrium candidate. This equilibrium candidate is indeed an equilibrium if it belongs to the switching region $\Omega_\sigma$ where the description (5) is valid. We next identify conditions for this to be true.

B. Existence and Uniqueness of Equilibria

Here we characterize the EUE for the dynamics (4). Given $W \in \mathbb{R}^{n \times n}$, define the equilibria set-valued map $h : \mathbb{R}^n \triangleright \mathbb{R}^n_{\geq 0}$ by
\[
h(d) \triangleq \{ x \in \mathbb{R}^n_{\geq 0} \mid x = [Wx + d]^+ \}.
\]
The map $h$ can, in particular, take empty values. EUE then precisely corresponds to $h$ being single-valued on $\mathbb{R}^n$. If so, with a slight abuse of notation, we take $h : \mathbb{R}^n \to \mathbb{R}^n_{\geq 0}$ to be an ordinary function.

From the definition (6) of equilibrium candidate, note that $x^\ast_\sigma \in h(d)$ if and only if $x^\ast_\sigma \in \Omega_\sigma$. Then, using Assumption 1, and after some manipulations, we have
\[
Wx^\ast_\sigma + d = W(I - \Sigma W)^{-1}\Sigma d + d = (W^{-1} - \Sigma)^{-1}\Sigma d + d = (I - W\Sigma)^{-1}W\Sigma d + d = [(I - W\Sigma)^{-1}W\Sigma + I]d = (I - W\Sigma)^{-1}d.
\]
Therefore,
\[
x^\ast_\sigma \in h(d) \Leftrightarrow x^\ast_\sigma \in \Omega_\sigma \Leftrightarrow ((2\Sigma - I)(W - \Sigma)^{-1})d \geq 0.
\]
Accordingly, for $\sigma \in \{0,1\}^n$, let
\[
\Delta_\sigma \triangleq \{ d \in \mathbb{R}^n \mid M_\sigma d \geq 0 \},
\]
be the set of external inputs $d$ such that (4) has an equilibrium in $\Omega_\sigma$, which is a closed convex polytopic cone.

Note that if $M_\sigma d \geq 0$ for exactly one $\sigma \in \{0,1\}^n$, then a unique equilibrium exists according to (9). However, when $M_\sigma d \geq 0$ for multiple $\sigma \in \{0,1\}^n$, $\ell \in \{1,\ldots,\ell\}$, the network may have either multiple equilibria or a unique one $x^\ast_\sigma_1 = \cdots = x^\ast_\sigma_\ell$ lying on the boundary between $\{\Omega_{\sigma_i}\}_{i=1}^\ell$.

The next result shows that the quantities $M_\sigma d$ can be used to distinguish between these two latter cases.

**Lemma IV.1.** *(Existence of multiple equilibria).* Assume $W$ satisfies Assumption 1, $d \in \mathbb{R}^n$ is arbitrary, and $M_\sigma$ is defined as in (9) for $\sigma \in \{0,1\}^n$. If there exist $\sigma_1 \neq \sigma_2$ such that $d \in \Delta_{\sigma_1} \cap \Delta_{\sigma_2}$, then $x^\ast_{\sigma_1} = x^\ast_{\sigma_2}$ if and only if $M_{\sigma_1}d = M_{\sigma_2}d$.

*Proof.*

\[
x^\ast_{\sigma_1} = x^\ast_{\sigma_2} \iff Wx^\ast_{\sigma_1} + d = Wx^\ast_{\sigma_2} + d \iff (I - W\Sigma_1)^{-1}d = (I - W\Sigma_2)^{-1}d,
\]
where we have used (8). Since both $M_{\sigma_1}d$ and $M_{\sigma_2}d$ are nonnegative, (10) holds if and only if $(I - W\Sigma_i)^{-1}d_i = (I - W\Sigma_2)^{-1}d_i = 0$ for any $i$ such that $\sigma_1,i \neq \sigma_2,i$. This is equivalent to $M_{\sigma_1}d = M_{\sigma_2}d$. □

Our next result provides an optimization-based condition for EUE that is both necessary and sufficient.

**Proposition IV.2.** *(Optimization-based condition for EUE).* Let $W$ satisfy Assumption 1 and $M_\sigma$ be as defined in (9) for $\sigma \in \{0,1\}^n$. For $d \in \mathbb{R}^n$, define $\mu_1(d)$ and $\mu_2(d)$ to be the largest and second largest elements of the set
\[
\{ \min_{i=1,\ldots,n} (M_\sigma d)_i \mid \sigma \in \{0,1\}^n \},
\]
respectively. Then, (4) has a unique equilibrium for each $d \in \mathbb{R}^n$ if and only if
\[
\max_{||d||=1} \mu_1(d)\mu_2(d) < 0.
\]

*Proof.* First, note that $d = 0$ is a degenerate case where the origin is the unique equilibrium belonging to all $\Omega_\sigma$. For any $d \neq 0$ and $\sigma \in \{0,1\}^n$, $M_\sigma d \geq 0$ if and only if $M_\sigma d / ||d|| \geq 0$. Thus, EUE for all $d \in \mathbb{R}^n$ and all $||d|| = 1$ are equivalent. The result then follows from Lemma IV.1 and the fact that, for any $d$,
\[
\mu_1(d)\mu_2(d) < 0 \iff \mu_1(d) > 0 \text{ and } \mu_2(d) < 0
\]
\[
\iff \exists \text{ unique } \sigma \in \{0,1\}^n \text{ with } M_\sigma d \geq 0. \tag{\ref{eq:uniqueness-equilibrium}}
\]

The optimization involved in (11) is usually highly non-convex. However, since the search space $||d|| = 1$ is compact, global search methods can be used to verify (11) numerically. Next, we give our main result regarding the EUE that not only is verifiable analytically but also provides insight into the class of matrices $W$ that satisfy EUE.

**Theorem IV.3.** *(Existence and uniqueness of equilibria).* Consider the network dynamics (4) and assume the weight matrix $W$ satisfies Assumption 1. Then, (4) has a unique equilibrium for each $d \in \mathbb{R}^n$ if $I - W \in \mathcal{P}$.

*Proof.*

The uniqueness can be shown as a corollary to both [46, Thm 5.3] and [47, Thm 2.2]. However, we provide in the following a novel proof technique based on (9) that establishes both existence and uniqueness. From Lemma IV.1, we know that, for any $d$, (4) has a unique equilibrium if
and only if exactly one element of \( \{ M_\sigma d \mid \sigma \in \{0, 1\}^n \} \) is nonnegative. To check this, we need to check whether
\[
\exists \sigma_1, \sigma_2 \in \{0, 1\}^n \text{ s.t. } M_{\sigma_1} d \geq 0 \text{ and } M_{\sigma_2} d \geq 0.
\]
Note that this is equivalent to saying that there exist \( x, y \in \mathbb{R}^n \) such that \( M_{\sigma_1}^{-1} x = d = M_{\sigma_2}^{-1} y \). A more general question, which is relevant to our discussion, is whether
\[
M_{\sigma_1}^{-1} x = M_{\sigma_2}^{-1} y, \quad x, y \in \mathcal{O}_n \setminus \{0\},
\]
for any orthant \( \mathcal{O}_n \) of \( \mathbb{R}^n \) (including \( \mathcal{O}_n = \mathbb{R}^n_0 \) as a special case). Note that \( x = y = 0 \) is a trivial case and thus excluded. This is the question we analyze in the following. Notice that for any \( \sigma \in \{0, 1\}^n \),
\[
M_\sigma^{-1} = (I - W \Sigma) (2 \Sigma - I) - W \Sigma.
\]
Since nodes can be relabeled arbitrarily, we can assume without loss of generality that \( \sigma_1 = [1^n_1, 0^{n_2}, 0^{n_3}, 0^{n_4}]^T \) and \( \sigma_2 = [1^n_2, 0^{n_2}, 1^{n_3}_T, 0^{n_4}]^T \) where \( n_1, \ldots, n_4 \geq 0, \sum_{i=1}^4 n_i = n \). Then, it follows from (13) that
\[
M_{\sigma_1}^{-1} = \begin{bmatrix}
-I_{n_1} - W_{11} & -W_{12} & 0 & 0 \\
-W_{21} & -I_{n_2} - W_{22} & 0 & 0 \\
-W_{41} & -W_{42} & -I_{n_3} - W_{33} & 0 \\
-W_{31} & 0 & 0 & -I_{n_4}
\end{bmatrix},
\]
where \( W_{ij} \)'s are submatrices of \( W \) with appropriate dimensions. Taking the inverse of \( M_{\sigma_1}^{-1} \) as a 2-by-2 block-triangular matrix [42, Prop 2.8.7] (with the indicated blocks), we get
\[
M_{\sigma_1} = \begin{bmatrix}
-I_{n_1} - W_{11} & -W_{12} & 0 & 0 \\
-W_{21} & -I_{n_2} - W_{22} & 0 & 0 \\
-W_{41} & -W_{42} & -I_{n_3} - W_{33} & 0 \\
-W_{31} & 0 & 0 & -I_{n_4}
\end{bmatrix}^{-1} = \begin{bmatrix}
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0
\end{bmatrix}.
\]
so direct multiplication gives
\[
M_{\sigma_1} M_{\sigma_2}^{-1} = \begin{bmatrix}
B_1 & B_2 & B_3 & B_4
\end{bmatrix},
\]
with
\[
B_1 = \begin{bmatrix}
-I_{n_1} - W_{11} & -W_{12} \\
-W_{21} & -I_{n_2} - W_{22}
\end{bmatrix}^{-1} = \begin{bmatrix}
I_{n_1} - W_{11} & 0 \\
-W_{21} & I_{n_2} - W_{22}
\end{bmatrix},
\]
\[
B_2 = -\begin{bmatrix}
-I_{n_1} - W_{11} & -W_{12} \\
-W_{21} & -I_{n_2} - W_{22}
\end{bmatrix}^{-1} = \begin{bmatrix}
-I_{n_1} - W_{11} & 0 \\
-W_{21} & I_{n_2} - W_{22}
\end{bmatrix},
\]
\[
B_3 = -\begin{bmatrix}
W_{31} & W_{32} \\
W_{41} & W_{42}
\end{bmatrix} B_4 + \begin{bmatrix}
W_{31} & 0 \\
W_{41} & 0
\end{bmatrix},
\]
\[
B_4 = -\begin{bmatrix}
W_{31} & W_{32} \\
W_{41} & W_{42}
\end{bmatrix} B_2 - \begin{bmatrix}
-I_{n_3} - W_{33} & 0 \\
-W_{43} & -I_{n_4}
\end{bmatrix}.
\]
With this, after some computations one can show that
\[
M_{\sigma_1}^{-1} M_{\sigma_2}^{-1} = \begin{bmatrix}
I_{n_1} & ** & 0 \\
0 & ** & 0 \\
0 & ** & 0 \\
0 & ** & I_{n_4}
\end{bmatrix}.
\]
If \( S_{O_n} = \{ \sigma_0 \} \), we assign \( \sigma_0 \) to \( O_n \). If \( |S_{O_n}| > 1 \), it follows from (16) that

(i) \( M_\sigma d \) is the same for all \( \sigma \in S_{O_n} \); let \( x_{O_n}^* \) be this shared value,

(ii) \( |S_{O_n}| = 2^k \) for some \( k \geq 1 \),

(iii) there exists \( I_{O_n} \subseteq \{1, \ldots, n\} \) with \( |I_{O_n}| = k \) such that for any \( i \notin I_{O_n} \), \( \sigma_i \) is the same for all \( \sigma \in S_{O_n} \),

(iv) \( (x_{O_n}^*)_i = 0 \) for all \( i \in I_{O_n} \).

Due to (iv), \( x_{O_n}^* \) belongs to the intersection of \( 2^k \) orthants \( (O_n \) and \( 2^k - 1 \) other orthants that differ from \( O_n \) along the dimensions in \( I_{O_n} \)). Therefore, we can assign each of the \( 2^k \) elements of \( S_{O_n} \), to its corresponding orthant from the \( 2^k \) orthants that contain \( x_{O_n}^* \). By repeating this procedure for all unassigned \( \sigma_0 \in \{0, 1\}^n \), we can uniquely assign every \( \sigma_0 \in \{0, 1\}^n \) to an orthant in \( R^n \) such that no two are assigned to the same orthant. Therefore, one \( \sigma \in \{0, 1\}^n \) must be assigned to the positive orthant \( R^n_+ \), showing the existence of an equilibrium and completing the proof.

The fact that the condition in Lemma II.2(iv) is an equivalent characterization of P-matrices suggests that the sufficient condition of Theorem IV.3 is tight. Indeed, extensive simulations with random matrices did not reveal any instance of \( W \) that is not a P-matrix but for which (4) has a unique equilibrium for all \( d \in R^n \) (where we checked the latter using Proposition IV.2). This leads us to the following conjecture, which was also made in [24] as a claim without proof.

Conjecture IV.4. (Necessity of \( I - W \in P \) for EUE). Assume the weight matrix \( W \) satisfies Assumption 1. Then, (4) has a unique equilibrium for all \( d \in R^n \) if and only if \( I - W \in P \).

Remark IV.5. (Computational complexity of verifying \( I - W \in P \)). Although the problem of determining whether a matrix is in \( P \) is straightforward for small \( n \), it is known to be co-NP-complete [48], and thus expensive for large networks. Indeed, [49] shows that all the \( 2^n \) principal minors of \( A \) have to be checked to prove \( A \in P \) (though disproving \( A \in P \) is usually much easier). In these cases, one may need to rely on more conservative sufficient conditions such as \( \rho(|W|) < 1 \) or \( \|W\| < 1 \) (cf. Lemma II.3) to establish \( I - W \in P \). These conditions, moreover, have the added benefit of providing intuitive connections between the distribution of synaptic weights, network size, and stability. We elaborate more on this point in Section V-C.

Example IV.6. (Uniform excitatory-inhibitory networks). Consider a network of \( n \) nodes in which \( \alpha n, \alpha \in (0, 1) \) are excitatory, \((1 - \alpha) n \) are inhibitory, and the synaptic weight between any pair of nodes only depends on their type (the synaptic weight of any inhibitory-to-excitatory connection is \( w_{ei} < 0 \), and similarly for \( w_{ee} > 0, w_{ie} > 0, w_{ii} < 0 \)). Also, assume common external inputs \( d_e, d_i \in R \) for all excitatory and inhibitory nodes, respectively. Let \( x_e(t) \) and \( x_i(t) \) be the average firing rates of excitatory and inhibitory nodes, respectively. Then,

\[
\tau \begin{bmatrix} \dot{x}_e \\ \dot{x}_i \end{bmatrix} = - \begin{bmatrix} x_e \\ x_i \end{bmatrix} + \begin{bmatrix} (\alpha w_{ee} (1 - \alpha) w_{ei}) x_e \\ (\alpha w_{ie} (1 - \alpha) w_{ii}) x_i + d_e d_i \end{bmatrix}.
\]

This simplification of \( n \)-dimensional networks to planar dynamics is commonly known as the Wilson-Cowan model [50] and is a widely used model in computational neuroscience, see e.g., [51], [52]. Let \( W_{EI} \in R^{2 \times 2} \) be the corresponding weight matrix above. One can check that

\[
I - W_{EI} \in P \iff \alpha w_{ee} < 1,
\]

and

\[
\rho(|W_{EI}|) < 1 \iff \alpha w_{ee} < 1, \quad (1 - \alpha) n |w_{ii}| < 1, \quad \alpha (1 - \alpha) n^2 w_{ie} |w_{ei}| < (1 - \alpha) w_{ee} (1 - (1 - \alpha) n |w_{ii}|).
\]

Thus, according to Theorem IV.3, EUE only requires the excitatory dynamics to be stable (note that \( w_{ee} \) has to be smaller as \( n \) grows), while the more conservative \( \rho(|W_{EI}|) < 1 \) requires two extra conditions: the stability of inhibitory dynamics and a weak interconnection between excitatory and inhibitory subnetworks.

C. Asymptotic Stability

The EUE, as discussed above, is an opportunity to shape the network state, provided the equilibrium corresponds to a desired state (e.g., a memory, the encoding of a spatial location, or eye position) and it attracts network trajectories [53]–[57]. Here we investigate when the latter holds, i.e., the network equilibrium is asymptotically stable. Our main result on asymptotic stability is the following.

Theorem IV.7. (Asymptotic Stability). Consider the network dynamics (4) and assume \( W \) satisfies Assumption 1.

(i) [Sufficient condition] If \( W \in L \), then for all \( d \in R^n \), the network is globally exponentially stable (GES) relative to a unique equilibrium \( x^* \);

(ii) [Necessary condition] If for all \( d \in R^n \) the network is locally asymptotically stable relative to a unique equilibrium \( x^* \), then \( I - W \in H \).

Proof. (i) The EUE follows from Lemma II.3(iii)&(iv) and Theorem IV.3. GES follows from [58, Thm 1], but we give a simpler proof here for completeness. Consider an arbitrary trajectory \( x(t) \) of (4) and define

\[
\xi(t) = x(t) - x^*.
\]

After some manipulations, one can show that

\[
\tau \xi(t) = (-I + M(t)W) \xi(t),
\]

where \( M(t) \) is a diagonal matrix with diagonal entries

\[
m_{ii}(t) = \begin{cases} \frac{|W_{i}x(t) + d_i^+|}{W_{ii}(t)} & \text{if } W_{ii}(t) \neq 0, \\ 0 & \text{otherwise}. \end{cases}
\]

Since the function \([\cdot]^+\) is monotonically increasing and Lipschitz with constant 1, \( m_{ii}(t) \in [0, 1] \) for all \( i \in \{1, \ldots, n\} \). Thus, \( M(t) \) belongs to the convex hull of \( \{\Sigma_\sigma\}_{\sigma \in \{0, 1\}^n} \) for all \( t \geq 0 \). Let \( (\alpha_\sigma(t))_{\sigma \in \{0, 1\}^n} \) be a convex combination such that

\[
M(t) = \sum_{\sigma \in \{0, 1\}^n} \alpha_\sigma(t) \Sigma_\sigma, \quad t \geq 0.
\]
By assumption, there exists $P = P^T > 0$ and $\lambda > 0$ such that
\[-(I + W^T \Sigma)P + P(-I + \Sigma W) \leq -\lambda I, \quad \sigma \in \{0, 1\}^n.\]

Therefore, the evolution of the Lyapunov function $V(\xi) = \xi^TP\xi$ along (17) satisfies
\[
\frac{dV(\xi(t))}{dt} = \xi^T\left[-(I + W^T M(t))P + P(-I + M(t) W)\right]\xi
\]
\[
= \xi^T \left[ \sum_{\sigma \in \{0, 1\}^n} (-I + \alpha_\sigma(t) W^T \Sigma) P + P(-I + \alpha_\sigma(t) \Sigma W) \right] \xi
\]
\[
\leq -\lambda \|\xi\|^2 \leq -\lambda \rho(P) V(\xi(t)),
\]
proving GES.

(ii) Assume, by contradiction, that $-I + W \notin \mathcal{H}$, which means that there exists $\sigma \in \{0, 1\}^n$ such that $-I + \Sigma W$ is not Hurwitz. Then, consider the choice
\[
d = (2I - W)\sigma - 1_n.
\]
It is straightforward to show that $x^* = \sigma$ is an equilibrium point for (4) lying in the interior of $\Omega_\sigma$. By assumption, $x^*$ is (unique and) locally asymptotically stable, which contradicts $-I + \Sigma W$ not being Hurwitz. This completes the proof.

Similar to the problem of verifying whether a matrix is a P-matrix, cf. Remark IV.5, verifying total-Hurwitzness becomes computationally expensive for large $n$. The next result gives a usually more conservative but computationally inexpensive alternative.

**Proposition IV.8. (Computationally feasible sufficient conditions for GES).** Consider the network dynamics (4) and assume the weight matrix $W$ satisfies Assumption 1. If $\rho(|W|) < 1$ or $||W|| < 1$, then for all $d \in \mathbb{R}^n$, the network has a unique equilibrium $x^*$ and it is GES relative to $x^*$.

**Proof.** If $||W|| < 1$, the result follows from Lemma II.3(ii) and Theorem IV.7. For the case $\rho(|W|) < 1$, however, the claim follows from the argument in [25, Prop. 3], which we bring here for completeness. For simplicity, assume that $W$ is irreducible, i.e., the network topology contains a path from any node to any other (this assumption is without loss of generality since, if $W$ is not irreducible, the continuity of the spectral radius guarantees the existence of $\epsilon > 0$ such that $\rho(|W| + \epsilon 1_n 1_n^T) < 1$. The same argument can then be employed for $|W| + \epsilon 1_n 1_n^T$). By the Perron-Frobenius Theorem [42, Fact 4.11.4], there exists $\alpha \in \mathbb{R}^n_0$ such that $\alpha^T |W| = \rho(|W|) \alpha^T$. The map $|| \cdot ||_\alpha : v \rightarrow ||v||_\alpha \triangleq \alpha^T v$ is indeed a norm on $\mathbb{R}^n$. To show EUE, note that for any $y, z \in \mathbb{R}^n$,
\[
||W y + d||^+ - ||W z + d||^+ ||_\alpha
\]
\[
= \alpha^T |W y + d|| - \alpha^T ||W z + d||^+ \leq \alpha^T |W(y - z)| \leq \alpha^T ||W|| ||y - z|| = \rho(|W|) \alpha^T ||y - z|| = \rho(|W|) ||y - z||_\alpha,
\]
so $x \mapsto ||W x + d||^+$ is a contraction on $\mathbb{R}^n_0$ and has a unique fixed point by the Banach Fixed-Point Theorem, denoted $x^*$.

To show GES, let $t \mapsto x(t)$ be an arbitrary trajectory and consider $\xi(t) \triangleq (x(t) - x^*)e^t$. We have
\[
\tau \xi(t) = M(t)W \xi(t),
\]
where $M(t)$ is the same as in (17). Then, by using [24, Lemma] (which is essentially a careful application of Gronwall-Bellman’s Inequality [59, Lemma A.1] to (18)), we get
\[
\|\xi(t)\|_\alpha \leq \|\xi(0)\|_\alpha e^{\rho(W)t}
\Rightarrow \|x(t) - x^*\|_\alpha \leq \|x(0) - x^*\|_\alpha e^{-1-\rho(W)t},
\]
which gives GES by the equivalence of norms on $\mathbb{R}^n$.

From Lemma II.3(iii), the conditions of Theorem IV.7 and Proposition IV.8 are not conclusive when $W$ satisfies $-I + W \in \mathcal{H}$ but neither $W \in \mathcal{L}$ nor $\rho(|W|) < 1$. However,

(i) If a unique equilibrium $x^*$ lies in the interior of a switching region $\Omega_\sigma$ (a condition that can be shown to hold for Lebesgue-almost all $d$), then $x^*$ is at least locally exponentially stable.

(ii) In our extensive simulations with random $(W, d)$, any system satisfying $-I + W \in \mathcal{H}$ was GES for all $d$. These observations lead us to the following conjecture, whose analytic characterization remains an open problem.

**Conjecture IV.9. (Sufficiency of total-Hurwitzness for GES).** Consider the dynamics (4) and assume $W$ satisfies Assumption 1. The network has a unique GES equilibrium for all $d \in \mathbb{R}^n$ if and only if $-I + W \in \mathcal{H}$.

We next study the GES of the uniform excitatory-inhibitory networks of Example IV.6.

**Example IV.10. (Uniform excitatory-inhibitory networks, cont’d).** Consider again the excitatory-inhibitory network of Example IV.6. One can verify that
\[
-I + W_{EI} \in \mathcal{H} \Leftrightarrow \alpha n w_{ee} < 1. \quad (19)
\]
Thus, the (sufficient) condition for EUE and (necessary) condition for GES coincide, and they interestingly only restrict $w_{ee}$ while $w_{ei}, w_{ie},$ and $w_{ii}$ are completely free. Figure 4 shows sample phase portraits for the cases $\alpha n w_{ee} < 1$ and $\alpha n w_{ee} > 1$.

**D. Boundedness of Solutions**

Here we study the boundedness of solutions under the network dynamics (3). While our discussion so far has been about the dynamics (4) (with constant $d$), we switch for the remainder of this section to (3) for the sake of generality, as the same results are applicable without major modifications. Note that in reality, the firing rate of any neuron is bounded by a maximum rate dictated by its refractory period (the minimum inter-spike duration). Unboundedness of solutions in the model corresponds in practice to the so-called “run-away” excitations where the firing of neurons grow beyond sustainable rates for prolonged periods of time, which is neither desirable nor safe [60]. Since GES implies boundedness of solutions, any condition that is sufficient for GES is also sufficient
where $\delta$

**Proof.**

Network trajectories for the excitatory-inhibitory network of Fig. 4:

nondecreasing field shows that inhibition, overall, preserves boundedness. The next result for boundedness. However, boundedness of solutions can be guaranteed under less restrictive conditions. The next result shows that inhibition, overall, preserves boundedness.

**Lemma IV.11. (Inhibition preserves boundedness).** Let $t \mapsto x(t)$ be the solution of (3) starting from initial state $x(0) = x_0$. Consider the system

$$
\tau \dot{x}(t) = -x(t) + [[W]^+x(t) + d(t)]^+, \quad x(0) = x_0. \tag{20}
$$

Then, $x(t) \leq \bar{x}(t)$ for all $t \geq 0$.

**Proof.** Since $x(t) \geq 0$ for all $t$, we can write (3) as

$$
\tau \dot{x}(t) = -x(t) + [[W]^+x(t) + d(t)]^+, \tag{21}
$$

where $\delta(t) \triangleq (W - [W]^+)x(t) \leq 0$. Since the vector field $(x, t) \mapsto -x + [[W]^+x + d(t)]^+$ is quasi-monotone nondecreasing, the result follows by using the monotonicity of the function $[\cdot]^+$ and applying the vector-valued extension of the Comparison Principle given in [61, Lemma 3.4] to (20) and (21).

While the result about preservation of boundedness under inhibition in Lemma IV.11 is intuitive, one must interpret it carefully: it is *not* in general true that adding inhibition to any dynamics (3) can only decrease $x(t)$. This is only true if the network vector field is quasi-monotone nondecreasing, as is the case with the excitatory-only dynamics (20). Intuitively, this is because, if the network has inhibitory nodes, adding inhibition to their input can in turn “disinhibit” and increase the activity of the rest of the network.

The next result identifies a condition on the excitatory part of the dynamics to determine if trajectories are bounded.

**Theorem IV.12. (Boundedness).** Consider the network dynamics (3). If the corresponding excitatory-only dynamics (20) has bounded trajectories, the trajectories of (3) are also bounded by the same bound as those of (20).

The proof of this result follows from Lemma IV.11 and is therefore omitted. The following result, similar to Proposition IV.8, provides a more conservative but computationally feasible test for boundedness.

**Corollary IV.13. (Boundedness).** Consider the network dynamics (3) and assume that $d(t)$ is bounded, i.e., there exists $d \in \mathbb{R}_{\geq 0}^n$ such that $d(t) \leq d, t \geq 0$. If $\rho([W]^+) < 1$, then the network trajectories remain bounded for all $t \geq 0$.

**Proof.** If $d(t)$ is constant, the result follows from Theorem IV.12 and Proposition IV.8. If $d(t)$ is not constant, the same argument proves boundedness of trajectories for the dynamics

$$
\tau \dot{x}(t) = -\bar{x}(t) + [[W]^+\bar{x}(t) + d]^+, \quad \bar{x}(0) = x_0. \tag{22}
$$

The result then follows from the quasi-monotonicity of $(x, t) \mapsto -x + [W^+x + d]^+$, similar to Lemma IV.11.

**Example IV.14. (Uniform excitatory-inhibitory networks, cont’d).** Consider again the excitatory-inhibitory network of Example IV.6. Clearly, the corresponding excitatory-only dynamics have bounded trajectories if and only if

$$
\rho([W_{EI}]^+) < 1 \iff \alpha w_{ee} < 1, \tag{23}
$$

which is the same condition as (19). However, an exhaustive inspection of the switching regions $\Omega_\sigma$ and the eigenvalues of $\{-I + \Sigma W\}_\sigma$ reveals that (23) can be relaxed to

$$
-I + W \text{ be Hurwitz } \iff \begin{cases} (1 - \alpha w_{ee}) + (1 - (1 - \alpha)n w_{ii}) > 0, \text{ and} \\ (1 - \alpha w_{ee})(1 - (1 - \alpha)n w_{ii}) > \alpha(1 - \alpha)n^2 w_{ee} w_{ei}, \end{cases}
$$

showing that there is room for sharpening Theorem IV.12.

**Remark IV.15. (Comparison with the literature).** In this section, we have provided a comprehensive list of conditions that both extend and simplify the state of the art on stability of dynamical networks. Regarding network equilibria, we have extended [46, Thm 5.3] to

---

3A vector field $f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n$ is quasi-monotone nondecreasing [61] if for any $x, y \in \mathbb{R}^n$ and any $i \in \{1, \ldots, n\}$,

$$(x_i = y_i \text{ and } x_j \leq y_j \text{ for all } j \neq i) \Rightarrow f(x, t) \leq f(y, t).$$
guarantee both existence and uniqueness when $I - W \in \mathcal{P}$ (Theorem IV.3) and provided a novel optimization-based if and only if condition for EUE (Proposition IV.2). On exponential stability, Theorem IV.7 gives a simpler proof than [58, Thm 1] for the sufficiency of $W \in \mathcal{L}$ and a novel proof for the necessity of $-1 + W \in \mathcal{H}$. Finally, our result on boundedness of trajectories (Theorem IV.12) extends Corollary IV.13 (also available in [30, Thm 1]) to a much wider class of networks by exploiting the quasi-monotonicity of excitatory-only dynamics.

\[\square\]

V. SELECTIVE INHIBITION IN BILAYER NETWORKS

Here, we study selective inhibition in bilayer networks as a building block towards the understanding of hierarchical selective recruitment in multilayer networks. With respect to the model described in Section III, we consider two layers ($N = 2$), where the dynamics of the lower layer $N_2$ is described by (3) and the dynamics of the upper layer $N_1$ is arbitrary (this is for generality, we consider linear-threshold dynamics for $N_1$ too in a multilayer framework in our accompanying work [19]). Our goal is to study the selective inhibition of $N_2^o$ via the input that it receives from $N_1$.

As pointed out in Section III, when a group of neurons are inhibited, their activity is substantially decreased, ideally such that their net input (their respective component of $Wx(t) + d(t)$) becomes negative and their firing rate decays exponentially to zero. Therefore, the problem of selective inhibition is equivalent to the exponential stabilization of the nodes $N_2^o$ to the origin. To this end, we decompose $d(t)$ as

\[d(t) = Bu(t) + \tilde{d}.\] (24)

The role of $u(t) \in \mathbb{R}_{\geq 0}^n$ is to stabilize $N_2^o$ to the origin while the role of $\tilde{d} \in \mathbb{R}^n$ is to shape the activity of $N_2^i$ by determining its equilibrium. For this purpose of this section, we assume $\tilde{d}$ is given and constant.

Let $r \leq n$ be the size of $N_2^o$. We partition $x$, $W$, $B$, and $d$ accordingly.

\[x = \begin{bmatrix} x^o \\ x^i \end{bmatrix}, \quad W = \begin{bmatrix} W^{oo} & W^{oi} \\ W^{io} & W^{ii} \end{bmatrix}, \quad B = \begin{bmatrix} B^o \\ 0 \end{bmatrix}, \quad d = \begin{bmatrix} 0 \\ \tilde{d} \end{bmatrix},\] (25)

where $W^{oo} \in \mathbb{R}^{r \times r}$, $B^o \in \mathbb{R}^{r \times m}$ is nonpositive to deliver inhibition, and $\tilde{d} \in \mathbb{R}^{n-r}$. The first $r$ rows of $B$ are nonzero to allow for the inhibition of $N_2^o$, while the remaining $n - r$ rows are zero to make this inhibition selective to $N_2^i$. The sparsity of the entries of $\tilde{d}$ is opposite to therows of $B$ due to the complementary roles of $Bu(t)$ and $\tilde{d}$.

The mechanisms of inhibition in the brain are broadly divided [62] into two categories, feedforward and feedback, based on how the signal $u(t)$ is determined. In the following, we separately study each scenario, analyzing the interplay between the corresponding mechanism and network structure. We will later combine both mechanisms when we discuss the complete HSR framework in [19], as natural selective inhibition is not purely feedback or feedforward.

A. Feedforward Selective Inhibition

Feedforward inhibition [62] refers to the scenario where $N_1$ provides an inhibition based on its own “desired” activity/inactivity pattern for $N_2$ and irrespective of the current state of $N_2$. This is indeed possible if the inhibition is sufficiently strong, as excessive inhibition has no effect on nodal dynamics due to the thresholding in $]^{-}$. However, this independence from the activity level of $N_2$ requires some form of guaranteed boundedness, as defined next.

Definition V.1. (Monotone boundedness). The dynamics (3) is monotonically bounded if for any $d \in \mathbb{R}^n$ there exists $\nu(d) \in \mathbb{R}_{\geq 0}^n$ such that $x(t) \leq \nu(d), t \geq 0$ for any $d(t) \leq d, t \geq 0$.

From Lemma IV.11 and Proposition IV.8, (3) is monotonically bounded if $\rho([W]^{-}) < 1$ and the initial condition $x_0$ is restricted to a bounded domain. Also in reality, the state of any biological neuronal network is uniformly bounded due to the refractory period of its neurons, implying monotone boundedness. The next result shows that the GES of $N_2^i$ is both necessary and sufficient for feedforward selective inhibition.

Theorem V.2. (Feedforward selective inhibition). Consider the dynamics (3), where the external input is given by (24)-(25) with a constant feedforward control

\[u(t) \equiv u \geq 0.\]

Assume that (3) is monotonically bounded and

\[\text{range}([W^{oo} W^{oi}], \mathbb{R}^n) \subseteq \text{range}(B^{o}).\] (26)

Then, for any $\tilde{d}^i \in \mathbb{R}^{n-r}$, there exists $\bar{u} \in \mathbb{R}_{\leq 0}^n$ such that for all $u \geq \bar{u}$, $N_2$ is GES relative to a unique equilibrium of the form $x_e = [0_T \ (x_e^o)^T]^T$ if and only if $W^{1i}$ is such that the internal $N_2^i$ dynamics

\[\tau \dot{x}^i = -x^i + [W^{1o} x^o + W^{11} x^i + \tilde{d}^{i}]^+,\] (27)

is GES relative to a unique equilibrium.

Proof. ($\Rightarrow$) Define $u_s$ to be a solution of

\[B^{o} u_s = -[W^{oo} W^{oi}] + \nu(\tilde{d}).\] (28)

This solution exists by assumption (26). Let $\bar{u} = [u_s]^{-}$ and note that $B^{o} u \leq B^{o} \bar{u} \leq B^{o} u_s$. By construction, (3), (24), (25), (28) simplify to

\[\tau \dot{x}^o = -x^o,\]

\[\tau \dot{x}^i = -x^i + [W^{1o} x^o + W^{11} x^i + \tilde{d}^i]^+,\] (29)

whose GES follows from Lemma A.2.

($\Rightarrow$) By monotone boundedness and nonpositivity of $B^{o}$, $x(t) \leq \nu(d)$ for all $t \geq 0$ and any $u \geq \bar{u}$. Let $u = \bar{u} + [u_s]^{-}$ where $u_s$ is a solution to (28). Similar to above, this simplifies (3), (24), (25), (28) to (29), which is GES by assumption. However, for any initial condition of the form $x(0) = [0_T x^o(0) x^i(0)]^T$, the trajectories of (29) are the same as (27), and the result follows.

\[\square\]

As we show next, the condition (26) on the ability to influence the dynamics of the task-irrelevant nodes through control
also plays a key role in feedback selective inhibition. We defer the discussion about the interpretation of this condition to Section V-C below.

B. Feedback Selective Inhibition

The core idea of feedback inhibition [62], as found throughout the brain, is the dependence of the amount of inhibition on the activity level of the nodes that are to be inhibited. This dependence is in particular relevant to GDSA, as the stronger and more salient a source of distraction, the harder one must try to suppress its effects on perception. The next result provides a novel characterization of several equivalences between the dynamical properties of $\mathcal{N}_2$ under linear full-state feedback inhibition and those of $\mathcal{N}_2^e$.

**Theorem V.3. (Feedback selective inhibition).** Consider the dynamics (3), where the external input is given by (24)-(25) with a linear state feedback $u$

$$u(t) = Kx(t),$$

and $K \in \mathbb{R}^{m \times n}$ is a constant control gain. Assume that (26) holds. Then, there exists $K \in \mathbb{R}^{m \times n}$ such that

(i) $I - (W + BK) \in P$ if and only if $I - W^{11} \in P$;
(ii) $I - (W + BK) \in H$ if and only if $I - W^{11} \in H$;
(iii) $W + BK \in L$ if and only if $W^{11} \in L$;
(iv) $\rho([W + BK]) < 1$ if and only if $\rho([W^{11}]) < 1$;
(v) $\|W + BK\| < 1$ if and only if $\|W^{11}\| < 1$.

**Proof.** (i) $\Rightarrow$ For any $K = [K_e \; K_f] \in \mathbb{R}^{m \times n}$,

$$W + BK = \begin{bmatrix} W^{00} + B^T K_e & W^{01} + B^T K_f \\ W^{10} & W^{11} \end{bmatrix}. \quad (31)$$

Thus, since any principal submatrix of a P-matrix is a P-matrix, $I - W^{11} \in P$.

$\Leftarrow$ Since $m \geq R$, there Lebesgue-almost always exists $K \in \mathbb{R}^{m \times n}$ such that

$$- [W^{00} \; W^{01}] = B^T K. \quad (32)$$

Using the fact that the determinant of any block-triangular matrix is the product of the determinants of the blocks on its diagonal [42, Prop 2.8.1], it follows that $I - (W + BK) \in P$.

(ii) $\Rightarrow$ This follows from (31) and the fact that a principal submatrix of a totally-Hurwitz matrix is totally-Hurwitz.

$\Leftarrow$ Using the matrix $K$ in (32), the result follows from the fact that the eigenvalues of a block-triangular matrix are the eigenvalues of its diagonal blocks.

(iii) $\Rightarrow$ Let $P = P^T > 0$ be such that

$$- (I + (W + BK)^T \Sigma) P + P (I + \Sigma (W + BK)) < 0 \quad (33)$$

for all $\sigma \in \{0, 1\}^n$. Consider, in particular, $\sigma = \begin{bmatrix} 0^T \; \sigma^e \end{bmatrix}^T$ where $\sigma^e \in \{0, 1\}^{n-r}$ is arbitrary. Let $\Sigma^e = \text{diag}(\sigma^e)$ and partition $P$ in 2-by-2 block form similarly to $W$. Since

$$- (I + (W + BK)^T \Sigma) P + P (I + \Sigma (W + BK)) = \begin{bmatrix} * \\ * & - (I + \Sigma^1 W^{11})^T P^{11} + P^{11} (I + \Sigma^1 W^{11}) \end{bmatrix},$$

and any principal submatrix of a negative definite matrix is negative definite, we deduce $W^{11} \in L$.

$\Leftarrow$ Let $P^{11} \in \mathbb{R}^{(n-r) \times (n-r)}$ be such that

$$(-I + (W^{11})^T \Sigma^e) P^{11} + P^{11} (-I + \Sigma^1 W^{11}) < 0,$$

for all $\sigma^e \in \{0, 1\}^{n-r}$ and $K$ be as in (32). For any $\sigma = \begin{bmatrix} \sigma^e \end{bmatrix}^T$, (32) gives

$$-I + \Sigma (W + BK) = \begin{bmatrix} -I & 0 \\ * & -I + \Sigma^1 W^{11} \end{bmatrix}.$$

Thus, the dynamics

$$\tau \dot{x} = (I + \Sigma W + BK) x,$$

is a cascade of $\tau \dot{x}^e = -x^e$ and

$$\tau \dot{x}^s = - (I + \Sigma^1 W^{11}) x^s + * \cdot x^e,$$

where the latter has the ISS Lyapunov function $V^1(x) = (x^s)^T P^{11} x^s$. Using [63, Thm 3], (33) holds for $K = K$, $P = \text{diag}(I, P^{11})$, and any $\sigma \in \{0, 1\}^n$, giving $W + BK \in L$.

(iv) $\Rightarrow$ This follows from (31) and [42, Fact 4.11.19].

$\Leftarrow$ Consider the matrix $K$ in (32). The result then follows from the fact that the eigenvalues of a block-triangular matrix are the eigenvalues of its diagonal blocks.

(v) $\Rightarrow$ Note that for any $K \in \mathbb{R}^{m \times n}$,

$$\|W + BK\| = \rho\left(\begin{bmatrix} * & W^{00} W^{01} \end{bmatrix}^T \right) \geq \rho(W^{10} W^{11}) = \|W^{10} W^{11}\|_2^2,$$

where the inequality follows from the well-known interlacing property of eigenvalues of principal submatrices (c.f. [64]).

$\Leftarrow$ Consider the matrix $K$ in (32) and note that

$$\|W + BK\|^2 = \rho\left(\begin{bmatrix} 0 & 0 \\ W^{10} W^{11} \end{bmatrix}^T \right) = \rho(W^{10} W^{11}) = \|W^{10} W^{11}\|_2^2 < 1,$$

completing the proof. $\square$

**Remark V.4. (Feedback inhibition with nonnegative $u(t)$).** Even though Theorem V.3 is motivated by feedback inhibition in the brain, the result illustrates some fundamental properties of linear-threshold dynamics and the corresponding matrix classes that is of independent interest, which motivates the generality of its formulation. The particular application to brain networks requires nonnegative inputs, which we discuss next. The core principle of Theorem V.3 is the cancellation of local input $[W^{00} \; W^{01}] x$ to $\mathcal{N}_2^e$ with the top-down feedback input $B^T K x$, simplifying the dynamics of $\mathcal{N}_2^e$ to $\tau \dot{x}^e = -x^e$ that guarantee its inhibition. However, the resulting input signal $u = K x$ (being the firing rate of some neuronal population) may not remain nonnegative at all times. This can be easily addressed as follows. Similar to the proof of Theorem V.2, we let

$$u(t) = [K x(t)]^+.$$

This makes $u(t)$ nonnegative without affecting the selective inhibition of $\mathcal{N}_2^e$ in (3) due to the nonpositivity of $B^e$ and the thresholding in $[\cdot]^+$. $\square$
C. Network Size, Weight Distribution, and Stabilization

Underlying the discussion above is the requirement that $\mathcal{N}_2$ can be asymptotically stabilized towards an equilibrium which has some components equal to zero and the remaining components determined by $d$. Here, it is important to distinguish between the stability of $\mathcal{N}_2$ in the absence and presence of selective inhibition. In reality, the large size of biological neuronal networks often leads to highly unstable dynamics if all the nodes in a layer, say $\mathcal{N}_2$, are active. Therefore, the selective inhibition of $\mathcal{N}_2$ is not only responsible for the suppression of the task-irrelevant activity of $\mathcal{N}_2$, but also for the overall stabilization of $\mathcal{N}_2$ that allows for top-down recruitment of $\mathcal{N}_2$. This poses limitations on the size and structure of the subnetworks $\mathcal{N}_2$ and $\mathcal{N}_2$. It is in this context that one can analyze the condition (26) assumed in both Theorems V.2 and V.3. This condition requires, essentially, that there are sufficiently many “independent” external controls $u$ to enforce inhibition on $\mathcal{N}_2$. The following result formalizes this statement.

Lemma V.5. (Equivalent characterization of (26).) Let the matrices $W^o$ and $B^o$ have dimensions $r \times n$ and $r \times m$, respectively. Then, $\text{range}(W^o) \subseteq \text{range}(B^o)$ for Lebesgue-almost all $(W^o, B^o) \in \mathbb{R}^{r \times n} \times \mathbb{R}^{r \times m}$ if and only if $m \geq r$.

Proof. \(\Rightarrow\) Assume, by contradiction, that $m < r$, so $\text{range}(B^o) \subseteq \mathbb{R}^r$ for any $B^o$. Let $Q = Q(B^o)$ be a matrix whose columns form a basis for $\text{range}(B^o)$. Then, $\text{range}(W^o) \subseteq \text{range}(B^o)$ if and only if $Q(B^o)^T W^o = 0$. By Fubini’s theorem, we have

$$
\int_{\mathbb{R}^r \times \mathbb{R}^m} d(B^o) \int_{\mathbb{R}^r \times \mathbb{R}^m} \mathbb{I}(Q(B^o)^T W^o = 0) (W^o, B^o) d(W^o) B^o = 0
$$

where $\mathbb{I}$ denotes the indicator function. This contradiction proves $m \geq r$.

\(\Leftarrow\) Let $B = [B_1^o, B_2^o]$ where $B_1^o \in \mathbb{R}^{r \times r}$. It is straightforward to show that

$$\{(W^o, B^o) \mid \text{range}(W^o) \not\subseteq \text{range}(B^o)\} \subseteq \mathbb{R}^r \times A,$$

where $A = \{B^o \mid \text{det}(B_1^o) = 0\}$. Since $A$ has measure zero, the result follows from a similar argument as above invoking Fubini’s theorem.

Based on intuitions from linear systems theory, it may be tempting to seek a relaxation of (26) for the case where $m < r$. This is due to the fact that for a linear system $\tau x = Wx + Bu$, it is known [66, eq (4.5) and Thm 3.5] that the set of all reachable states from the origin is given by

$$\text{range} \left( \begin{bmatrix} B^o & W^{o0} B^o & \cdots & (W^{n-1})^{o0} B^o \\ 0 & W^{10} B^o & \cdots & (W^{n-1})^{10} B^o \end{bmatrix} \right),$$

which is usually much larger than $\text{range}(B)$. Therefore, it is reasonable to expect that (26) could be relaxed to

$$\text{range}([W^{o0}, W^{o1}]) \subseteq \text{range}([B^o, W^{o0} B^o, \cdots, (W^{n-1})^{o0} B^o]).$$

However, it turns out that this relaxation is not possible, the reason being the (apparently simple, yet intricate) nonlinearity in (3). We show this by means of an example.

Example V.6. (Tightness of (26).) Consider the feedback dynamics (3), (24), (30), where $n = 3$, $m = 1$, $r = 2$, and

$$W = \begin{bmatrix} 2\alpha & 0 & 0 \\ 0 & 3\alpha & 0 \\ 0 & 0 & \alpha \end{bmatrix}, \quad B = \begin{bmatrix} 1 \\ 1 \\ 0 \end{bmatrix}, \quad \alpha \in (0.5,1).$$

Clearly, (26) does not hold (so Theorem V.3(iv) does not apply), but $\text{range}([W^{o0}, W^{o1}]) \subseteq \text{range}([B^o, W^{o0} B^o])$. One can show that for all $K \in \mathbb{R}^{1 \times 3}$,

$$\rho(\|W + BK\|) \geq 2\alpha > 1,$$

while $\rho(\|W\|) = \alpha < 1$, verifying that (26) is necessary and cannot be relaxed to (34).

Therefore, Theorems V.2 and V.3 use completely different mechanisms for inhibition of $\mathcal{N}_2$, yet they are strikingly similar in one conclusion, namely, that the dynamical properties achievable under selective inhibition are precisely those satisfied by the task-relevant part $\mathcal{N}_2$. This has important implications for the size and structure of the part $\mathcal{N}_2$ that can remain active at any instance of time without resulting in instability. The next remark elaborates on this implication.

Remark V.7. (Implications for the size of $\mathcal{N}_2$.) Existing experimental evidence suggest that the synaptic weights $W$ in cortical networks are sparse, approximately follow a log-normal distribution, and have a pairwise connection probability that is independent of physical distance between neurons within short distances [67]. Figure 5(a) shows the value of $\rho(\|W\|)$ for random matrices with such statistics, which grows linearly with $n$. Notably, the network (representing $\mathcal{N}_2$ here) rapidly loses stability as its size grows. On the other hand, recent advances in machine learning suggest that the expressivity of a neuronal network (often loosely defined as its capacity to reproduce complex trajectories) is maximized when it operates at the boundary between stability and instability, commonly referred to as the edge of chaos [68]–[70]. While determining the optimal size of a network that leads to maximal expressivity is beyond the scope of this work, our results suggest a critical role for selective inhibition in keeping only a limited number of nodes in $\mathcal{N}_2$ active at any given time while inhibiting others. In other words, while the overall size of subnetworks in a brain network (corresponding to, e.g., the number of neuronal populations with distinct preferred stimuli in a brain region) is inevitably large, selective inhibition offers a plausible explanation for the mechanism by which the brain keeps the number of active populations at any given time bounded ($O(1)$), thus maintaining its local dynamics close to the “edge of chaos”.

Similarly, Figure 5(b) shows the probability of the three stability related conditions $I - W \in \mathcal{P}$, $-I + W \in \mathcal{H}$, and $\rho(\|W\|) < 1$ (cf. Section IV) as a function of average absolute weight in the network, showing a rapid drop from 1 to less than 0.1 around unit average absolute weight. Interestingly, several works in the neuroscience literature have
The effects of network size and weight distribution on its stability. Each circle represents a random matrix with 10% sparsity and synaptic weights log-normally distributed with parameters $\mu = -0.7$ and $\sigma = 0.9$ as given in [67]. As in cortical networks, 80% of nodes are excitatory and 20% inhibitory. The line illustrates a fit of the form $\log \rho(|W|) = \alpha \log n + \beta$ with $\alpha = 1$ and $\beta = -1.2$, showing a linear growth of $\rho(|W|)$ with $n$. The probability of $\rho(|W|) < 1$ as a function of the average absolute weight of the networks with the same statistics as in (a) but fixed size $n = 10$ and varying $\mu = -3.5, -3, \ldots, 2.5$. The probabilities are estimated empirically with $10^3$ sample networks for each value of $\mu$.

shown that neuronal networks maintain stability by re-scaling their synaptic weights that change during learning, a process commonly referred to as homeostatic synaptic plasticity [71]. Our results therefore open the way to provide rigorous and quantifiable measures of the optimal size and weight distribution of neuronal subnetworks that may be active at any given time in order to maintain any desired level of network stability.

VI. CONCLUSIONS

We have adopted a control-theoretic perspective to study hierarchical selective recruitment as a mechanism to explain goal-driven selective attention. Motivated by the organization of the brain, HSR consists of an arbitrary number of neuronal subnetworks that operate at different layers of a hierarchy. While HSR is not confined to any specific family of models, we here use the well-studied family of linear-threshold rate models to describe the dynamics of subnetworks at each layer of the hierarchy. We have provided a thorough analysis of the internal dynamics of each layer. Leveraging the switched-affine nature of linear-threshold dynamics, we have derived several necessary and sufficient conditions for the existence and uniqueness of equilibria (corresponding to P-matrices), local and global asymptotic stability (corresponding to totally-Hurwitz matrices), and boundedness of trajectories (corresponding to stability of excitatory-only dynamics). These results set the basis for analyzing the problem of selective inhibition. We have shown that using either feedforward or feedback inhibition, the dynamical properties of each subnetwork after inhibition are precisely determined by the dynamical properties of the task-relevant part that remains active. We have also provided constructive control designs that guarantee selective inhibition in both feedforward and feedback schemes. Among the important directions for future research, we highlight the study of output feedback control for selective inhibition and the analysis of the conditions on (single-layer) linear-threshold networks that lead to the emergence of limit cycles and their stability.

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**APPENDIX A**

Here we provide auxiliary results that are used in the proofs of main results of the paper. The following result is used in the proof of Theorem IV.3 on the EUE for the dynamics (4).

**Lemma A.1.** Consider a matrix $\mathbf{M} \in \mathbb{R}^{n \times n}$ with the block form

$$
\mathbf{M} = \begin{bmatrix}
\mathbf{I}_n & \mathbf{0} \\
\mathbf{0} & \mathbf{I}_{n_1}
\end{bmatrix}
$$

(35)
where $\ast$ means an arbitrary block and $\Gamma \in \mathbb{R}^{n_{23} \times n_{23}}$. Then, there exists nonzero $y \in \mathbb{R}^n$ such that $x \triangleq My$ and $y$ belong to the same orthant(s) if and only if there exists nonzero $y_{23} \in \mathbb{R}^{n_{23}}$ such that $x_{23} \triangleq \Gamma y_{23}$ and $y_{23}$ belong to the same orthant(s) or $y_{23} = 0$, where $x_{23}$ and $y_{23}$ denote the middle $n_{23}$-dimensional sub-vectors of $x$ and $y$, respectively.

**Proof.** It follows from (35) that

$$
M \begin{bmatrix}
y_{1,23} \\
y_{4,23}
\end{bmatrix} = \begin{bmatrix}
y_1 + \ast \cdot y_{23} \\
\Gamma y_{23} \\
y_4 + \ast \cdot y_{23}
\end{bmatrix}.
$$

Therefore, the ($\Rightarrow$) implication is immediate. For the ($\Leftarrow$) implication, note that if $y_{23}, \Gamma y_{23} \neq 0$ belong to the same orthant(s), then choosing $y_1 \in \{\pm c\}^{n_1}, y_4 \in \{\pm c\}^{n_4}$ with sufficiently large $c > 0$ puts $My$ in the same orthant(s) as $y$. If $y_{23} = 0$, then $My = y$ and the result is trivial.

Finally, the following result is used in the proof of Theorem V.2.

**Lemma A.2. (GES of cascaded interconnections).** Consider the cascaded dynamics

$$
\tau \dot{x}^o = -x^o,
$$

$$
\tau \dot{x}^1 = -x^1 + [W^{10} x^o + W^{11} x^1 + d^1]^+,
$$

where $x^o \in \mathbb{R}^r$ and $x^1 \in \mathbb{R}^{n-r}$. If $W^{11}$ is such that

$$
\tau \dot{x}^1 = -x^1 + [W^{11} x^1 + d^1]^+,
$$

is GES for any constant $d^1 \in \mathbb{R}^{n-r}$, then the whole dynamics (36) is also GES for any constant $d^1$.

**Proof.** We only prove the result for $d^1 = 0$. This is without loss of generality, since for $d^1 \neq 0$, we can apply the change of variables $x \rightarrow x - h([0^T (d^1)T]^T)$ and shift the equilibrium to the origin. Since (37) is GES, [19, Thm A.1] guarantees that there exists $x^1 \mapsto V^1(x^1)$ such that

$$(38a) \quad c_1 \|x^1\|^2 \leq V^1(x^1) \leq c_2 \|x^1\|^2,$$

$$
(38b) \quad \left\| \frac{\partial V^1(x^1)}{\partial x^1} \right\| \leq c_3 \|x^1\|,
$$

for some $c_1, c_2, c_3 > 0$, and, if $x^1(t)$ is the solution of (37),

$$(38c) \quad \frac{d}{dt} V^1(x^1(t)) \leq -c_4 \|x^1\|^2,$$

for some $c_4 > 0$. Since $[\cdot]^+$ is Lipschitz continuous, it follows from (38b) and (38c) that if $x^1(t)$ is the solution of (36),

$$(38d) \quad \frac{d}{dt} V^1(x^1(t)) \leq -c_4 \|x^1\|^2 + c_5 \|x^1\| \|W^{10} x^o\| \leq -c_4 \|x^1\|^2 + \frac{c_5}{2} \|W^{10}\|^2 \|x^o\|^2,$$

where the second inequality follows from Young’s inequality [72]. Now, let

$$
V(x) = \frac{c_5}{2c_4} \|W^{10}\|^2 \|x^o\|^2 + V^1(x^1).
$$

It is straightforward to verify that $V$ satisfies all the assumptions of [59, Thm 4.10] with $a = 2$, completing the proof.