

NIH Public Access

Author Manuscript

Neural Comput. Author manuscript; available in PMC 2014 May 19.

Published in final edited form as:

Neural Comput. 2013 August ; 25(8): 1994–2037. doi:10.1162/NECO_a_00472.

Analysis of the stabilized supralinear network[‡]

Yashar Ahmadian^{1,4,*}, Daniel B. Rubin^{1,2,*}, and Kenneth D. Miller^{1,2,3,4,†}

¹Center for Theoretical Neuroscience, College of Physicians and Surgeons, Columbia University, NY, NY 10032

²Dept. of Neuroscience, College of Physicians and Surgeons, Columbia University, NY, NY 10032

³Doctoral Program in Neurobiology and Behavior, College of Physicians and Surgeons, Columbia University, NY, NY 10032

⁴Kavli Institute for Brain Science, College of Physicians and Surgeons, Columbia University, NY, NY 10032

Abstract

We study a rate-model neural network composed of excitatory and inhibitory neurons in which neuronal input-output functions are power laws with a power greater than 1, as observed in primary visual cortex. This supralinear input-output function leads to supralinear summation of network responses to multiple inputs for weak inputs. We show that for stronger inputs, which would drive the excitatory subnetwork to instability, the network will dynamically stabilize provided feedback inhibition is sufficiently strong. For a wide range of network and stimulus parameters, this dynamic stabilization yields a transition from supralinear to sublinear summation of network responses to multiple inputs. We compare this to the dynamic stabilization in the "balanced network", which yields only linear behavior. We more exhaustively analyze the 2-dimensional case of 1 excitatory and 1 inhibitory population. We show that in this case dynamic stabilization will occur whenever the determinant of the weight matrix is positive and the inhibitory time constant is sufficiently small, and analyze the conditions for "supersaturation", or decrease of firing rates with increasing stimulus contrast (which represents increasing input firing rates). In work to be presented elsewhere, we have found that this transition from supralinear to sublinear to sublinear summation can explain a wide variety of nonlinearities in cerebral cortical processing.

1 Introduction

In work to be presented elsewhere (presented as abstracts in Miller and Rubin (2010); Rubin and Miller (2010); Miller and Rubin (2011); Rubin and Miller (2011)), we have found that a large set of response properties of cells in primary visual cortex (V1) and other sensory cortical areas can be understood from a very simple circuit motif. The response properties have in common a change in integration with increasing input strength, so that responses to

[‡]This preprint has been published as a peer-reviewed article:

[†]To whom correspondence should be addressed: ken@neurotheory.columbia.edu.

^{*}These authors contributed equally to this work.

One set of properties the model can address involves contextual modulation or "surround suppression". A visual sensory neuron has a classical receptive field (CRF), corresponding to the region in which appropriate visual stimuli will drive the neuron's responses. The size of the CRF does not change with input strength (Song & Li, 2008). Stimuli outside the CRF can modulate responses to CRF stimuli, although they cannot drive responses, and typically are suppressive. However, the nature of the surround influence can vary with input strength (Sengpiel, Blakemore, & Sen, 1997; Polat, Mizobe, Pettet, Kasamatsu, & Norcia, 1998). A size tuning curve is obtained by centering an effective stimulus on the CRF center and studying response vs. stimulus radius. The summation field size is the stimulus size evoking peak response. This summation field size shrinks with input strength, as represented by stimulus contrast (Sceniak, Ringach, Hawken, & Shapley, 1999; Cavanaugh, Bair, & Movshon, 2002; Anderson, Lampl, Gillespie, & Ferster, 2001; Shushruth, Ichida, Levitt, & Angelucci, 2009; Tsui & Pack, 2011). This means that regions of the surround are changing from facilitating to suppressing with increasing input strength.

Another set of properties involves sublinear summation of the responses to multiple stimuli: the response to two simultaneously presented stimuli can be closer to the average than the sum of the responses to the stimuli presented individually. We refer to this property as "normalization", because it is the most prominent of a set of nonlinear response properties that have been given that name (reviewed in Carandini and Heeger (2012)). In at least some cases, this summation becomes supralinear when inputs are weak (Heuer & Britten, 2002; Ohshiro, Angelaki, & DeAngelis, 2011). If one thinks of surround suppression as representing the response to simultaneous presentation of a center stimulus that normally by itself evokes a certain response and a surround stimulus that normally by itself evokes zero response, then surround suppression can be thought of as an example of sublinear summation. Similarly, facilitation by the near surround for weak inputs then represents supralinear summation.

As we will show elsewhere, these and other response properties can be understood in some detail from a simple model. We consider a network of excitatory (*E*) and inhibitory (*I*) neurons, extended across a 1-D or 2-D space. The strengths of each type of connection $-E \Rightarrow E, E \Rightarrow I, I \Rightarrow E, I \Rightarrow I - fall off as functions of cortical distance. We are guided by previous results that showed that the inhibition received by cells is decreased when they are being suppressed by a surround stimulus, relative to their response to a CRF stimulus alone (Ozeki, Finn, Schaffer, Miller, & Ferster, 2009), and correspondingly that the firing of inhibitory cells, like that of excitatory cells, is suppressed by surround stimuli (Song & Li, 2008). These results led to the conclusion that the <math>E \Rightarrow E$ connections must be sufficiently strong that, when the network is being driven by a CRF stimulus, they would render the network unstable in the absence of feedback inhibition (Tsodyks, Skaggs, & Sejnowski, 1997; Ozeki et al., 2009), a conclusion also supported by other work (London, Roth, Beeren, Hausser, & Latham, 2010). We termed such a network an inhibition-stabilized network or ISN.

We then add to this the fact that individual neurons have a supralinear, power-law inputoutput function. This is based on intracellular recordings in anesthetized cat primary visual cortex (V1) that showed that a neuron's instantaneous firing rate is well described as a power law function of its instantaneous mean voltage relative to rest (rates and voltages measured in 30 ms bins) with powers ranging from 2 to 5, and that this holds true over the entire dynamic range of neuronal response to visual stimuli (N. Priebe, Mechler, Carandini, & Ferster, 2004; N. J. Priebe & Ferster, 2005, 2006; Finn, Priebe, & Ferster, 2007).¹ This power law relationship is predicted on theoretical grounds when mean input is subthreshold and spiking is driven by input fluctuations (Miller & Troyer, 2002; Hansel & van Vreeswijk, 2002), as appears to be the case in V1 (Anderson, Lampl, Gillespie, & Ferster, 2000).

Here we mathematically analyze the model. We focus particularly on exposing the origins of the generic transition in model behavior from supralinear to sublinear summation as input strength increases. This typically corresponds to a transition from a regime in which the excitatory network is stable by itself to one in which the excitatory subnetwork by itself is unstable, but is stabilized by feedback inhibition. Hence we refer to the network as the stabilized supralinear network or SSN. We also conduct a more detailed analysis of the 2-dimensional case consisting of a single excitatory and a single inhibitory population.

2 Setup: Equations for the Supralinear Network

 $\mathbf{r} = \begin{pmatrix} \mathbf{r}_E \\ \mathbf{r}_I \end{pmatrix}$ to be the *N*-dimensional vector of neuronal firing rates, ordered so that the top N_E neurons, represented by \mathbf{r}_E , are all excitatory neurons, and the remaining N_I neurons, represented by \mathbf{r}_I are inhibitory neurons, $N_E + N_I = N$. (We refer to the units in our model as "neurons", but, as discussed below, the equations represent average firing rates and so excitatory or inhibitory units may be better understood as local interconnected groups of excitatory or inhibitory neurons, over which the average is taken.) The matrix of connections

between the neurons is $\mathbf{W} = \begin{pmatrix} \mathbf{W}_{EE} & -\mathbf{W}_{EI} \\ \mathbf{W}_{IE} & -\mathbf{W}_{II} \end{pmatrix}$ where \mathbf{W}_{XY} is the matrix of connections from neurons of type *Y* (E or I) to neurons of type *X* and has non-negative entries. The

feedforward input to the neurons in the network is $\mathbf{h} = \begin{pmatrix} \mathbf{h}_E \\ \mathbf{h}_I \end{pmatrix}$.

We study the simplest standard firing-rate-model equations (reviewed in Ermentrout and Terman (2010), Chapter 11; Gerstner and Kistler (2002), Chapter 6; Dayan and Abbott (2001) Chapter 7), in which a neuron's firing rate approaches a nonlinear function of its input with first-order dynamics:

¹We are assuming that mean voltage is linear in the input. Nonlinearities such as spike-rate-adaptation currents could complicate this picture. We also are ignoring the fact that the power increases with contrast, because the noise level decreases with contrast (Finn et al., 2007), which yields increasing powers (Miller & Troyer, 2002; Hansel & van Vreeswijk, 2002). However the picture we describe in this paper primarily concerns stabilization against the otherwise explosive nonlinearity of a supralinear input-output function. Thus, the picture should hold so long as the input-output function is supralinear over the cell's dynamic range, as expected for fluctuation-driven spiking – the closer the cell is to threshold, the greater the increase in spiking driven by a given increment of input.

$$au \mathbf{T} \frac{d\mathbf{r}}{dt} = -\mathbf{r} + \mathbf{f} (\mathbf{W}\mathbf{r} + \mathbf{h})$$
 (1)

Here **T** is a diagonal matrix of relative time constants, *i.e.* the time constant of the *i*th neuron is τT_{ii} . **f** is a vector function of a vector argument that acts elementwise on its argument, $(\mathbf{f}(\mathbf{v}))_i = f_i(\upsilon_i)$, for some scalar functions of a scalar variable, f_i , where υ_i is the *i*th element of **v**. These rate model equations do not capture fast time scales that arise in spiking networks, and cannot capture synchronization of spikes across neurons, but tend to be reliable in describing steady states or slower aspects of dynamics when neurons spike asynchronously. We will focus on the steady state and its stability.

We will study the case in which the f_i are identical for all elements, $f_i \equiv f$, and f is a rectified power law with power n > 1:

$$f(x) = k([x]_{+})^{n}$$
 (2)

where $[x]_+ = x$, x > 0; = 0, otherwise. We will summarize this by saying

$$\tau \mathbf{T} \frac{d\mathbf{r}}{dt} = -\mathbf{r} + k(\mathbf{W}\mathbf{r} + \mathbf{h})^{\cdot n} \quad (3)$$

where \mathbf{v}^{n} is the vector with i^{th} element $([\upsilon_{i}]_{+})^{n}$ (the period in the exponent *.n*, based on Matlab notation, is to indicate that the operation is done element-by-element rather than to the vector as a whole). A power-law relation between the mean input and mean response arises in the case that spiking is driven by input fluctuations (Miller & Troyer, 2002; Hansel & van Vreeswijk, 2002), and similarly it is observed in V1 as the relation between the trial-averaged mean voltage and mean response. Note that the fact, noted in the Introduction, that the power law holds over the entire dynamic range of visual responses means that V1 neurons never reach firing rates at which intrinsic saturation of the input-output function plays a role. Since V1 firing rates in response to optimal visual stimuli are typically among the highest seen in cerebral cortex, the same conclusion is likely to apply to cortex more generally. For this reason, we model the input/output function. Model results will only apply when the model produces firing rates that remain within the non-saturating regime.

We now change variables to dimensionless ones. This allows us to determine the dimensionless combinations of parameters on which model behavior depends and in which expansions for small or large values may be undertaken. We let $\psi = ||\mathbf{W}||$ where $||\mathbf{W}||$ is some matrix norm or other measure of the size of \mathbf{W} , and write $\mathbf{W} = \psi \mathbf{J}$ with \mathbf{J} dimensionless and $||\mathbf{J}|| = 1$. Similarly we let $c = |\mathbf{h}|$ and write $\mathbf{h} = c\mathbf{g}$ with \mathbf{g} dimensionless and $||\mathbf{g}|| = 1$ (again, $|\mathbf{g}||$ indicates some measure of the size of a vector, *e.g.* a vector norm). Note that *c* and $\psi \mathbf{r}$ have the same units, so that $\psi \mathbf{r}/c$ is dimensionless, and that kc^n and \mathbf{r} have the same units, so that $kc^{n/2}(c/\psi) = kc^{n-1}\psi$ is dimensionless. We thus define the dimensionless variable and parameter:

$$\mathbf{y} = \mathbf{r}\psi/c$$
 (4)
 $\alpha = kc^{n-1}\psi$ (5)

Then equation 3 becomes

$$\tau \mathbf{T} \frac{d\mathbf{y}}{dt} = -\mathbf{y} + \alpha (\mathbf{J}\mathbf{y} + \mathbf{g})^{.n} \quad (6)$$

Thus, given **J**, **g**, **T**, and *n*, the dynamics depends only on the single parameter **a**.

The fact that Eq. 6 has a single α for all neurons is quite general: if neuron *i* had parameter α_i , this could be replaced with α by multiplying all weights J_{ij} and inputs g_i to neuron *i* by

 $\left(\frac{\alpha_i}{\alpha}\right)^{1/n}$, leaving the form of the equation unchanged. However the fact that the equation has a single *n* for all neurons is a real restriction. Consideration of *n*'s that vary between neurons or between neuron types remains a question for future study.

Note that we can rewrite Eq. 3 as $\tau \mathbf{T} \frac{d\mathbf{r}}{dt} = -\mathbf{r} + f(I)$ where $f(x) = x^n$ and the input $I = k^{\frac{1}{n}} \mathbf{W} \mathbf{r} + k^{\frac{1}{n}} c \mathbf{g}$. By incorporating the factors of *k* into the input *I*, the steady-state rate f(I) becomes of unit magnitude when the input *I* is of unit magnitude. This is a natural scaling for defining the effective recurrent weights, $k^{\frac{1}{n}} \mathbf{W}$, and the effective input strength, $k^{\frac{1}{n}} c$.

Then $\alpha = kc^{n-1}\psi = \left(k^{\frac{1}{n}} \|\mathbf{W}\|\right) \left(k^{\frac{1}{n}}c\right)^{n-1}$, that is, $\alpha = (\text{recurrent weight})(\text{feedforward strength})^{n-1}$. Note also that whether the input is dominated by feedforward input $c\mathbf{g}$ or recurrent input \mathbf{Wr} is determined by the size and structure of \mathbf{y} for a given α (because $\mathbf{Wr} + c\mathbf{g} = c(\mathbf{J}\mathbf{y} + \mathbf{g})$, so that the balance depends only on the relative sizes of $\mathbf{J}\mathbf{y}$ vs. \mathbf{g}), and is not impacted at all by the ratio c/ψ , which naively might be thought to determine the feedforward/recurrent balance. For a given α , this ratio simply scales $\mathbf{r} (\mathbf{r} = (c/\psi)\mathbf{y})$.

We will focus on the equation for the steady-state:

$$\mathbf{y} = \alpha (\mathbf{J}\mathbf{y} + \mathbf{g})^{\cdot n}$$
 (7)

However, in considering stability of the steady state we will need to use the dynamical equation 6.

3 Scaling Argument

In this section, we show that the supralinear network generically makes a transition between responses that scale in two different ways with α . For weak inputs ($\alpha \ll 1$), the net input (feedforward plus recurrent) to a neuron grows linearly with the feedforward strength. Because of the supralinear neuronal input/output function, this yields supralinear summation of responses to multiple sets of feedforward inputs. For stronger inputs ($\alpha \gg 1$), the recurrent input largely cancels the feedforward input, leaving only a net input component

that grows sublinearly with the feedforward input strength. This yields sublinear response summation for a broad range of parameters. The transition between these two scaling regimes, which we refer to as the supralinear and sublinear scaling regimes respectively, occurs for α of order of magnitude 1, for which we use the standard notation $\alpha \sim O(1)$. We then compare this dynamic input cancellation to that in the balanced network model of Van Vreeswijk and Sompolinsky ((1998)).

The transition from supralinear to sublinear scaling is generally marked by recurrent excitation becoming strong enough to yield instability and explosive growth of activity on its own, along with dynamical stabilization of network activity by feedback inhibition. The effective connection strength between two neurons tells how much the steady-state postsynaptic rate changes for a given change in steady-state presynaptic rate. This is given by the weight between the neurons times the postsynaptic gain. The gain is the slope of the input-output function (Eq. 2), which is monotonically increasing with the postsynaptic cell's

firing rate. That is, given the steady-state equation $r_i = k \left(\sum_j W_{ij} r_j + cg_i \right)^{n}$, the effective

weight from neuron *j* to neuron *i* is $\frac{dr_i}{dr_j} = nk \left(\sum_j W_{ij}r_j + cg_i\right)^{n-1} W_{ij} = nk^{\frac{1}{n}} r_i^{\frac{n-1}{n}} W_{ij}$, which monotonically increases with r_i . With increasing input, the network responses typically increase and hence effective connections grow until the recurrent excitatory-to-excitatory connections become strong enough to yield instability absent stabilization by feedback inhibition. The transition from supralinear to sublinear scaling is generally closely associated with this transition to potential excitatory instability, as we will see (Figs. 2–4 and Section 5.4).

3.1 Scaling for small a

For $a \ll 1$ we expect the steady state to satisfy $y \approx a g^{n}$, since then the **Jy** term is small relative to the **g** term and so adds only a small correction to this solution. More generally, we can write a formal expression for the steady state by starting with Eq. 7 and iteratively substituting $a(g + Jy)^{n}$ for each instance of **y**, yielding:

$$\mathbf{y} = \alpha (\mathbf{g} + \alpha \mathbf{J} (\mathbf{g} + \alpha \mathbf{J} (\mathbf{g} + \alpha \mathbf{J} (\dots)^{\cdot n})^{\cdot n})^{\cdot n}$$
(8)

or, in terms of **r**,

$$\mathbf{r} = k(c\mathbf{g} + k\mathbf{W}(c\mathbf{g} + k\mathbf{W}(c\mathbf{g} + k\mathbf{W}(\dots)^{\cdot n})^{\cdot n})^{\cdot n} \quad (9)$$

where the ellipses indicate infinite repetition of the pattern. Assuming quantities in the parentheses are positive (which they will be for sufficiently small α as we assume all components of **g** are positive) so that we can ignore rectification, Eq. (8) can be converted into an infinite series in increasing integer powers of α with dominating (lowest-order) term $\alpha \mathbf{g}^{n,2}$. The terms multiplying α^p will involve factors of **g** interspersed with **J**'s, with the sum of the powers on the **g**'s equal to p(n-1) + 1. Similarly for **r**, one obtains a series involving an infinite set of powers of *c*, with lowest-order term $k(c\mathbf{g})^{\cdot n}$ and higher-order terms proportional to $k^p c^{p(n-1)+1}$ and involving a set of **g**'s with summed power also equal to p(n - 1).

1) + 1. If this series converges, which it will for sufficiently small α , it will give a steady state solution.

Thus, for small a, feedforward inputs sum supralinearly to produce responses (i.e., the response **r** depends supralinearly on the feedforward input $c\mathbf{g}$). Intuitively: as previously

noted, the effective connection from neuron *j* to neuron *i* is $nk^{\frac{1}{n}}r_i^{\frac{n-1}{n}}W_{ij}$. For small α , $r_i \approx$

 $k(cg_i)^n$ so $k^{\frac{1}{n}}r_i^{\frac{n-1}{n}}W_{ij} \approx \alpha g_i^{n-1}J_{ij}$ is small, that is, effective connections are small. In this regime the network is essentially feedforward driven, with small modifications by the weak effective recurrent connections. Since individual cells respond supralinearly to their inputs, the network sums responses supralinearly.

3.2 Scaling for large a

The scaling $\mathbf{y} \sim \mathbf{a}$ (or equivalently $\mathbf{r} \sim c^n$) cannot hold once \mathbf{a} is sufficiently large. In particular, for sufficiently large α , the series in Eq. 8 will explode rather than converge. Physically, this occurs because inputs are raised to the power n > 1 to produce responses which in turn feed back in as inputs; once inputs are sufficiently large, this process is explosive, like a nuclear reaction going critical. We expect this to occur for $\alpha = O(1)$. Moreover, for $a \gtrsim 1$, the effective recurrent weights typically become strong enough that the excitatory subnetwork by itself becomes unstable in the absence of dynamic feedback inhibition.

For Eq. 7 for the steady state to be self-consistent, the dependence of $\alpha (\mathbf{J}\mathbf{y} + \mathbf{g})^n$ on the leading a must be cancelled, because otherwise $\mathbf{y} \sim \mathbf{a}$, which enters into $\mathbf{J}\mathbf{y}$ and (dominating over the feedforward input, g, for large enough a) is raised to the n^{th} power to give³ y = $\alpha(\mathbf{J}\mathbf{v}+\mathbf{g})^n \sim \alpha^{n+1}$, which in turn enters into $\mathbf{J}\mathbf{y}$, and so on – the infinite series in powers of α results, which will blow up for sufficiently large α . Thus to cancel the leading α , it must be

the case that, to leading order in α , $\mathbf{J}\mathbf{y}+\mathbf{g}\sim\alpha^{-\frac{1}{n}}$. This in turn requires that, to leading order, \mathbf{y} has the same α -dependence as $\mathbf{g}, \mathbf{y} \sim \alpha^{0}$, so that the leading order of \mathbf{y} can cancel the \mathbf{g} term leaving only terms of order $\alpha^{-\frac{1}{n}}$.

Thus, if the steady-state Eq. (7) is stable, the recurrent input Jy must have dynamically adjusted itself to approximately cancel the feedforward input g, leaving a remainder that becomes smaller with growing α . We refer to this as dynamic stabilization, for two reasons. First, mathematically, this dynamic cancellation is necessary for the existence of a stable steady state for large α . Second, physically, the cancellation typically arises as the excitatory subnetwork becomes unstable by itself and the network is dynamically stabilized by feedback inhibition, although as we will discuss later (Section 5.4) there are cases in which the cancellation arises without excitatory subnetwork instability.

²This can be done by expanding each power in Eq. 8 as

 $[\]alpha(\mathbf{g}+\alpha\mathbf{J}(\ldots)^{.n})^{.n} = \alpha(\mathbf{g}^{.n} + n\mathbf{g}^{.(n-1)} \cdot \ast \alpha \mathbf{J}(\ldots)^{.n} + \frac{n(n-1)}{2}\mathbf{g}^{.(n-2)} \cdot \ast (\alpha \mathbf{J}(\ldots)^{.n}) \cdot ^{2} + \cdots, \text{ where } \cdot \ast \text{ indicates element-by-element multiplication of two vectors to create another vector, and then collecting together the terms of each given order$ in a. ³This could be avoided if $\mathbf{J}\mathbf{y} = 0$ to leading order in a, but that requires fine tuning, *i.e.* it requires Det $\mathbf{J} = 0$.

Let us define

$$\beta = \alpha^{-\frac{1}{n}}$$
. (10)

We write $\mathbf{y} = \mathbf{y}_0 + \beta \mathbf{y}_1$, with both \mathbf{y}_0 and \mathbf{y}_1 approximately $O(\beta^0)$ so that $\beta \mathbf{y}_1$ scales approximately as β . It follows from the requirement $\mathbf{J}\mathbf{y} + \mathbf{g} \sim \beta$ that

$$\mathbf{y}_0 = -\mathbf{J}^{-1}\mathbf{g}.$$
 (11)

Substituting $\mathbf{y} = -\mathbf{J}^{-1}\mathbf{g} + \beta \mathbf{y}_1$ in Eq. (7) then yields

$$-\mathbf{J}^{-1}\mathbf{g}+\beta\mathbf{y}_1=(\mathbf{J}\mathbf{y}_1)^{\cdot n}.$$
 (12)

The latter equation shows that y_1 itself has some further dependence on β . We will further discuss below whether the assumption holds that βy_1 scales approximately as β .

These arguments can be translated in terms of **r**. Once *c* is sufficiently large, selfconsistency requires cancellation of the linear dependence of $\mathbf{Wr} + c\mathbf{g}$ on *c*, because otherwise $\mathbf{r} \sim c^n$, which enters back into \mathbf{Wr} and is raised to the *n* to yield c^{n^2} dependence, and so on. Cancellation requires that, to leading order, $\mathbf{r} \sim c$, which in turn requires that to leading order $\mathbf{Wr} + c\mathbf{g} \sim c^{\frac{1}{n}}$, that is, the net input to a cell grows sublinearly with the feedforward input strength. Writing $\mathbf{r} = c\mathbf{r}_0 + c^{\frac{1}{n}}\mathbf{r}_1$, with \mathbf{r}_0 and \mathbf{r}_1 both O(1), we find that

$$\mathbf{r}_{0} = \frac{1}{\psi} \mathbf{y}_{0} = -\mathbf{W}^{-1} \mathbf{g}_{\text{and}} \mathbf{r}_{1} = \frac{c}{\psi} \frac{\beta}{c^{\frac{1}{n}}} \mathbf{y}_{1} = \frac{1}{\psi(k\psi)^{\frac{1}{n}}} \mathbf{y}_{1}, \text{ with } \mathbf{r}_{1} \text{ satisfying}$$
$$-\mathbf{W}^{-1} \mathbf{g} + c^{-\frac{n-1}{n}} \mathbf{r}_{1} = (\mathbf{W} \mathbf{r}_{1})^{n}.$$

These solutions show that, if the network dynamically stabilizes, the net input to cells grows sublinearly with the feedforward input strength and responses are given by a sum of terms that are linear and sublinear in the feedforward inputs. In studies of 2-dimensional systems (one excitatory and one inhibitory population), we will find that, when the excitatory-neuron element of $-\mathbf{J}^{-1}\mathbf{g}$ (equivalently of \mathbf{r}_0) is negative, the sublinear term becomes dominant (as it must: $(\mathbf{y}_0)_E < 0$, so one must have $\beta(\mathbf{y}_1)_E > |(\mathbf{y}_0)_E|$ for $y_E > 0$) and network behavior becomes strongly sublinear. In this case, excitatory firing rates eventually peak and then are ultimately pushed to zero with increasing *c*, *i.e.* with decreasing β , but there is typically a large dynamic range of *c* beyond the supralinear-to-sublinear transition before this peak occurs (see Figure 2). The behavior from c = 0 until somewhat beyond the peak yields behavior much like that seen in biology, and so we guess that this dynamic range represents the dynamic range of the feedforward input to cortex. This will be discussed in Sections 5.2–5.3. In simulations, the dependence of \mathbf{y}_1 on β remains weak, so that $\beta \mathbf{y}_1$ decreases nearly as fast as β , as was assumed, over the range from the supralinear-to-sublinear transition until r_E has been pushed close to zero. By contrast, when the elements of $-\mathbf{J}^{-1}\mathbf{g}$ are positive, the

subleading correction, $c^{\frac{1}{n}}\mathbf{r}_1$, can have either sign, and the total response $\mathbf{r}=c\mathbf{r}_0+c^{\frac{1}{n}}\mathbf{r}_1$ can be a supralinear or sublinear function of c,⁴ according to whether the sign of \mathbf{r}_1 is negative or positive respectively. However, even in the case where the response as a function of c remains supralinear for large c (small β), the response to the sum of two non-aligned inputs,

 \mathbf{g}_1 and \mathbf{g}_2 , will typically sum sublinearly for large *c*, as we will see in Section 4.2. Given positive elements of $-\mathbf{J}^{-1}\mathbf{g}$, Eq. 12 shows that \mathbf{y}_1 goes to an O(1) constant as β decreases to

zero $(\mathbf{y}_1 \to \mathbf{J}^{-1}(-\mathbf{J}^{-1}\mathbf{g})^{\cdot \frac{1}{n}})$, and thus the term $\beta \mathbf{y}_1$ is guaranteed to decay approximately as β for sufficiently small β .

A more systematic account of the large α (small β) case can be obtained by formulating a solution like that of Eq. 8 for small α . When the elements of **y** are > 0 (and thus the elements of **Jy** + **g** are > 0), we can rearrange Eq. 7 for the steady state as

$$\mathbf{y} = -\mathbf{J}^{-1}\mathbf{g} + \beta \mathbf{J}^{-1}\mathbf{y}^{\cdot \frac{1}{n}} \quad (13)$$

Then we can formally write a steady-state solution by starting with equation 13 and iteratively substituting $-\mathbf{J}^{-1}\mathbf{g}+\beta\mathbf{J}^{-1}\mathbf{y}\cdot\frac{1}{n}$ for **y** to obtain

$$\mathbf{y} = -\mathbf{J}^{-1}\mathbf{g} + \beta \mathbf{J}^{-1} (-\mathbf{J}^{-1}\mathbf{g} + \beta \mathbf{J}^{-1} (\dots)^{\cdot \frac{1}{n}})^{\cdot \frac{1}{n}} \quad (14)$$

or

$$\mathbf{r} = -c\mathbf{W}^{-1}\mathbf{g} + \frac{1}{k^{1/n}}\mathbf{W}^{-1}(-c\mathbf{W}^{-1}\mathbf{g} + \frac{1}{k^{1/n}}\mathbf{W}^{-1}(\dots)^{\cdot\frac{1}{n}})^{\cdot\frac{1}{n}}$$
(15)

If quantities in parentheses are positive, a series solution in powers of β can be obtained from Eq. 14 in the same manner as outlined for Eq. 8. When this series converges, which it will for small enough β , it gives a steady-state solution. However, if elements of $-\mathbf{J}^{-1}\mathbf{g}$ are negative, then for small enough β the elements in parentheses will no longer be positive (and correspondingly, as mentioned above, in the 2-D case y_E is pushed to zero with decreasing β at finite β , so that Eq. 13 fails at that point). We can instead regard Eq. 13 as an iterative

scheme, $\mathbf{y}[p+1] = -\mathbf{J}^{-1}\mathbf{g} + \beta \mathbf{J}^{-1}\mathbf{y}[p]^{\frac{1}{n}}$, beginning from some initial condition $\mathbf{y}[0]$ (Eq. 8 can also be regarded in this way), which generates Eq. 14 as $p \to \infty$. Writing this as $\mathbf{y}[p + 1] = f(\mathbf{y}[p])$, if all of the eigenvalues of the Jacobian of *f* at the fixed point have absolute values less than 1, then the iteration will converge to the fixed point within some basin of attraction about the fixed point. Hence with suitable initial conditions, one can find solutions through this iterative scheme, although not for β 's less than that at which some elements of \mathbf{y} are pushed to zero.

These scaling arguments provide key insights into the supralinear network (Eq. 3) that is confirmed by other analysis and simulations: for small α , recurrence is weak and the network supralinearly adds responses to different feedforward inputs; with increasing α , there is a transition, for $\alpha = O(1)$, to a dynamic stabilization that causes the net input neurons receive, and in many cases their responses, to add sublinearly. Note that, when responses add sublinearly, individual neurons still supralinearly sum the net (feedforward plus recurrent) inputs they receive, but the network "conspires" to deliver net input that is so

⁴By saying that $\mathbf{r}(c)$ is a supralinear or sublinear function of c, we mean that $\frac{d^2\mathbf{r}}{dc^2}$ is > 0 or < 0 respectively.

Neural Comput. Author manuscript; available in PMC 2014 May 19.

strongly sublinear that, even after the neuron raises its net input to the power n, its responses add sublinearly. We have found in both high-dimensional and 2-dimensional simulations, and we will show below for the 2-dimensional case, that stabilization will occur provided feedback inhibition is sufficiently strong and the inhibitory time constant is not too slow relative to the excitatory time constant. This transition from supralinear to sublinear behavior in turn appears to underly a wide variety of nonlinearities in neocortical behavior.

3.3 Comparison to the balanced network

Van Vreeswijk and Sompolinsky (1996, 1998) introduced the "balanced network" model (see also Renart et al. (2010)). They considered a circuit of randomly connected stochastic excitatory and inhibitory units that could have activity states 0 or 1, in the limit in which both feedforward and recurrent inputs were very large. (The same model could also be studied in other regimes, but we use "balanced network" to refer to the model in the largeinput regime that they studied and "balanced state" to refer to the solution they characterized in that regime.) They studied the conditions in which the network would dynamically find its way to a balanced state in which the mean input is subthreshold, yet firing rates are nonzero (where firing rate is defined as the average activity), meaning firing is driven by

fluctuations. They assumed each unit received K inputs of strength $\frac{1}{\sqrt{K}}$, or a net input of strength \sqrt{K} , for K large (e.g., thousands of inputs). The mean field equations for the average E and I firing rates are the 2-dimensional version of the rate equation, Eq. 1, for one E and one I population, where both W and cg are of order \sqrt{K} ,⁵ and the function f is a sigmoidal function rising from 0 to 1 as the input moves from approximately -3 to 3, and saturating at 0 or 1 for smaller or larger values respectively. To be in the balanced state, the mean firing rate must be neither 0 nor saturated at 1, so the net input must be O(1) (*i.e.* between -3 and 3).

Thus, the condition for the balanced state is that $Wr + cg \sim O(1)$ where both W and cg are

 $O(\sqrt{K})$. The solution, much as in our scaling argument, is to write $\mathbf{r}=\mathbf{r}_0+\frac{1}{\sqrt{K}}\mathbf{r}_1+\cdots$,

where \mathbf{r}_0 and \mathbf{r}_1 are O(1) and the dots represent higher-order terms in $\frac{1}{\sqrt{K}}$. The balance condition is that the $O(\sqrt{K})$ term in the input vanishes, that is, $\mathbf{Wr}_0 = -c\mathbf{g}$, leaving as input

only the O(1) term $\frac{\mathbf{W}}{\sqrt{K}}\mathbf{r}_1$ and terms that are $O(\frac{1}{\sqrt{K}})$.

The dynamic cancellation condition for the balanced state, $\mathbf{r}_0 = -c\mathbf{W}^{-1}\mathbf{g}$, is of course identical to the condition we have found for approximate dynamic cancellation in the SSN.⁶ Although the condition is formally identical, the meaning is different in crucial ways:

1. In the balanced network, the cancellation is required because inputs are large and must cancel to leave something small, in order to avoid zero or saturated output. In

⁵The input is expressed in units of a variance term which itself is dynamically determined, but this term is O(1) and does not impact the points made here. We wrote the leading term as $c\mathbf{r}_0$ rather than \mathbf{r}_0 , but the leading terms are identical.

the SSN, partial cancellation can already arise when none of the inputs are large and is required so that the supralinear input-output functions do not give rise to an inconsistent, explosive scaling. The difference in the size of the inputs when cancellation occurs can be seen by recalling that, in the SSN, $\alpha = (\text{recurrent} \text{ weight})(\text{feedforward strength})^{n-1}$ (see paragraph above Eq. 7).⁷ In the balanced network, the recurrent weights and the feedforward weight are both $O(\sqrt{K})$, so the SSN's α would be $O(K^{\frac{n}{2}})$. In contrast, dynamic cancellation arises in the SSN when α is O(1).

2.

In the balanced network, the second-order term $\frac{1}{\sqrt{K}}\mathbf{r}_1$ is negligibly small relative to the first-order term \mathbf{r}_0 (because the stabilization is to cancel large things, leaving something small). The first-order term is linear in the input, $\mathbf{r}_0 = -c\mathbf{W}^{-1}\mathbf{g}$, and so in the balanced network responses are always linear in the input. In the SSN, the firstorder term in \mathbf{y} , $y_0 = J^{-1}\mathbf{g}$, is O(1), and while the second-order term βy_1 scales approximately as β , it can have a large pre-factor such that it can be comparable to or larger than the first-order term over a wide dynamic range, enabling a variety of sublinear behavior (and \mathbf{r}_0 and \mathbf{r}_1 are just c/ψ times the corresponding \mathbf{y} 's).

In particular, in the SSN, elements of \mathbf{r}_0 can be negative, meaning that for such an element $r_1 > |r_0|$ over the relevant dynamic range of behavior (discussed in more detail for the 2-D model in Section 5.2). In the balanced network, since all terms except \mathbf{r}_0 are negligibly small, the elements of \mathbf{r}_0 must be positive for activity to be nonzero.

In sum, in the balanced network, inputs are huge relative to the distance from rest to threshold, and must dynamically cancel for the network to neither saturate nor have 0 activity but instead be in the fluctuation-driven regime. The dynamic cancellation or stabilization yields network responses that are always linear in the input. In the SSN, the supralinear input-output function renders the network explosive – input is raised to a power greater than 1 to produce responses, which feed back as input. Stabilization against this explosive nonlinearity arises when inputs are relatively small, yielding a range of sublinear behavior.

Finally, for clarity we note that the balanced network and SSN are not necessarily different models, but rather very different solutions with different behaviors in different regimes, which may be found in the same model. For example, in the power-law model studied here, the balanced-state solution can be found if α is large, the elements of $\mathbf{y}_0 = -\mathbf{J}^{-1}\mathbf{g}$ are positive, and the feedforward input $k^{\frac{1}{n}}c$ and recurrent input $k^{\frac{1}{n}}\psi$ both have the same scaling, *e.g.* both are $O(\sqrt{K})$, so that (c/ψ) is O(1). In this case, as $\alpha \to \infty$, $\mathbf{y} \to \mathbf{y}_0$ and $\mathbf{r} \to \frac{c}{\psi}\mathbf{y}_0$, so \mathbf{r} is O(1); and the first-order correction is $c^{\frac{1}{n}}\mathbf{r}_1 = \frac{c}{\psi}\beta\mathbf{y}_1 = \left(\frac{c}{k\psi}\right)^{1/n}\frac{1}{\psi}\mathbf{y}_1$, which scales as

⁷Note that this expression for α holds when the input is scaled appropriately so that the steady state firing rates, *f*(input), are *O*(1) when the input is *O*(1) (see paragraph above Eq. 7). This relationship between input and steady state rates also holds for the balanced network, so we have a common scale for the input on which to compare the size of the recurrent weights and of the feedforward strengths in the two models.

 $\frac{1}{k^{\frac{1}{n}}\psi}$, e.g. as $\frac{1}{\sqrt{K}}$ (recall \mathbf{y}_1 is O(1) for large α). This is the balanced-state solution. Similarly, in the model studied in the balanced network, the input/output function is initially supralinear before saturating, so we expect that SSN-like solutions could be found for appropriate regimes of input and recurrent strengths with $\alpha = O(1)$.⁸

4 Reduction to a 2-dimensional system

Most of our analysis hereafter will focus on a 2-dimensional system of one excitatory and one inhibitory population, as it is difficult to say much in general in higher dimensions. A 2-D system of one E and one I population can be derived as a mean field equation from higher-dimensional models in which E and I neurons have random connectivity (*e.g.* van Vreeswijk and Sompolinsky (1998), Renart et al. (2010)). In particular, if the high-dimensional model involves integrate-and-fire neurons, their input-output functions in the fluctuation-driven regime can be reasonably approximated by power-law functions (Hansel & van Vreeswijk, 2002).

Here we consider a higher-dimensional system with structured connectivity. We show a heuristic derivation of a 2-D system that preserves a surprising amount of the behavior of the higher-dimensional system. We then show how the conditions for "normalization" – sublinear addition of responses to multiple stimuli – in the high-D system can be expressed as simple conditions in the 2-D system on the growth of \mathbf{r} with increasing c, or the growth of \mathbf{y} with increasing α . In particular, the transition to the high- α regime represents a transition to sublinear addition in the corresponding high-D system, even when the dependence of \mathbf{r} on c remains supralinear in the 2-D system.

4.1 Reduction

We consider a topographic network, with pairs of excitatory (*E*) and inhibitory (*I*) units arranged on a 1-D or 2-D grid with periodic boundary conditions. The grid dimensions mirror stimulus parameters such as orientation or position on the retina, such that units at a certain location in the grid prefer stimuli with the corresponding parameter(s) (other stimulus preferences may also be incorporated, *e.g.*, a 2-D retinotopic grid with a superimposed map of preferred orientations). We will use as an illustration a 1-D ring of cells of similar retinotopic position but varying preferred orientation, with preferred orientation represented by position on the ring, but the reduction framework is more general. Stimuli are localized on the grid (*e.g.* in the 1-D ring a single oriented grating stimulus evokes a localized Gaussian-shaped bump of input, centered at the neurons that prefer the stimulus orientation), though there may be more than one localized stimulus present (*e.g.* two superimposed oriented gratings of different orientations). For simplicity, we assume a single time constant for all *E* cells and one for all *I* cells. We let θ represent the position on the grid, and $r_E(\theta)$ and $r_I(\theta)$ the excitatory and inhibitory firing rates at position θ . Thus, we can write Eq. 3 as

⁸The value of *n* in the definition of α could be derived from an approximate power-law fit to the supralinear portion of the input/output function in that model.

$$\begin{aligned} \tau_E \frac{dr_E(\theta)}{dt} &= -r_E(\theta) + k(W_{EE} * r_E(\theta) - W_{EI} * r_I(\theta) + cg_E(\theta))^{\cdot n} \quad (16) \\ \tau_I \frac{dr_I(\theta)}{dt} &= -r_I(\theta) + k(W_{IE} * r_E(\theta) - W_{II} * r_I(\theta) + cg_I(\theta))^{\cdot n} \quad (17) \end{aligned}$$

Here, $W_{XY} * r_Y(\theta) = \theta' W_{XY}(\theta, \theta') r_Y(\theta') \theta$, where θ is the stimulus parameter volume per grid point.

We will consider "normalization", the sublinear addition of the responses to two stimuli (Carandini & Heeger, 2012). We let one stimulus be centered at $\theta = 0$. We let $\tilde{W}_{XY} = W_{XY}$

(0, 0), and we define $\vec{w}_{XY} \equiv \frac{W_{XY}(0,\theta)}{W_{XY}(0,0)} \Delta \theta$ to be the vector of weights to position 0, normalized by \tilde{W}_{XY} . Similarly, we let $\hat{\mathbf{r}}_E$, $\hat{\mathbf{r}}_I$ be the vectors of excitatory and inhibitory firing rates, respectively, normalized to equal 1 at position 0, with elements $\hat{r}_E(\theta)$, $\hat{r}_I(\theta)$. Then the equations for the units at position 0 are

$$\tau_E \frac{dr_E(0)}{dt} = -r_E(0) + k \left(\tilde{W}_{EE} r_E(0) (\vec{w}_{EE} \cdot \hat{\mathbf{r}}_E) - \tilde{W}_{EI} r_I(0) (\vec{w}_{EI} \cdot \hat{\mathbf{r}}_I) + cg_E(0) \right)^{.n}$$
(18)

$$\tau_{I} \frac{dr_{I}(0)}{dt} = -r_{I}(0) + k \left(\tilde{W}_{IE} r_{E}(0) (\overrightarrow{w}_{IE} \cdot \mathbf{\hat{r}}_{E}) - \tilde{W}_{II} r_{I}(0) (\overrightarrow{w}_{II} \cdot \mathbf{\hat{r}}_{I}) + cg_{I}(0) \right)^{.n}$$
(19)

Although we had previously incorporated changes in $|\mathbf{g}|$ into *c*, we now take addition of a second stimulus to simply alter \mathbf{g} with no change in *c*, so that in particular addition of a second stimulus that gives no input to position 0 does not alter $g_E(0)$ or $g_I(0)$. We now define

$$J_{XY} \equiv \tilde{W}_{XY} \frac{\left(\vec{w}_{XY} \cdot \hat{\mathbf{r}}_{Y}\right)}{\left(\vec{w}_{EE} \cdot \hat{\mathbf{r}}_{E}\right)} \quad (20)$$
$$\Psi \equiv \vec{w}_{EE} \cdot \hat{\mathbf{r}}_{E} \quad (21)$$

Note that in general the J_{XY} depend on the normalized shapes of the responses, \hat{r}_E and in some cases \hat{r}_I , which in turn may depend on *c* and/or **g**. Letting $r_E \equiv r_E(0)$, $g_E \equiv g_E(0)$, etc., our equations become

$$\tau_{E} \frac{dr_{E}}{dt} = -r_{E} + k (\Psi J_{EE} r_{E} - \Psi J_{EI} r_{I} + cg_{E})^{\cdot n} \quad (22)$$
$$\tau_{I} \frac{dr_{I}}{dt} = -r_{I} + k (\Psi J_{IE} r_{E} - \Psi J_{II} r_{I} + cg_{I})^{\cdot n} \quad (23)$$

As written, Eqs. (22)–(23), together with the definitions (20) and (21), are simply a rewriting of Eqs. (18)–(19) and hence exact. In particular, the seemingly closed system of equations

for the two variables $r_E = r_E(0)$ and $r_I = r_I(0)$ are in general parametrically dependent on the values of $\hat{r}_X(\theta)$ at other θ 's through the dependence of J_{XY} and Ψ on the normalized shapes of the response curves.

We now adopt the ansatz that, as the stimulus changes, the four dot products $\vec{w}_{XY} \cdot \hat{\mathbf{r}}_{Y}$ are all scaled by a common factor, whether the stimulus changes in strength (changing c) or in shape (changing g, e.g. by adding a second stimulus). This means that we treat the J_{XY} as constants independent of c and g. On the other hand, Ψ is scaled by this common scaling factor. We make the further ansatz that Ψ depends only on g and not on c. Since Ψ depends only on the shape of the response, $\hat{\mathbf{r}}_{E}$, and not its magnitude, this amounts to the ansatz that stimulus strength c alters response magnitude without altering response shape. With these two ansatze, Eq. 23 is simply the 2-dimensional version of Eq. 3, and is equivalent to Eq. 6 for a 2-dimensional y if Ψ replaces ψ in the definitions of y and α (Eq. 5). (We are no longer following our convention of $\|\mathbf{J}\| = 1$, as the matrix \mathbf{J} composed of these J's need not satisfy $\|\mathbf{J}\| = 1$ for any standard matrix norm; one could, however, return to this convention by dividing the J_{XY} 's by $\|\mathbf{J}\|$ and multiplying Ψ by the same factor.) If, furthermore, we also assume that the shapes of the population responses roughly follow the shape of the input, then, since the weights \vec{w}_{XY} are non-negative, the effect of adding a second non-negative stimulus is to increase Ψ . Hence "normalization" of the E or I population corresponds to a decrease in firing rates $r_E(0)$ or $r_I(0)$, respectively, with increasing Ψ , which for a second stimulus of infinitesimal strength corresponds to

$$\frac{dr_{\scriptscriptstyle E}(0)}{d\Psi} {<} 0, \frac{dr_{\scriptscriptstyle I}(0)}{d\Psi} {<} 0. \quad (24)$$

The ansatze, of course, are not in general true, but they can be close enough to true to give a good qualitative account of the higher-dimensional system (however, see the discussion at the end of Section 5.3 for a discussion of cases where the assumptions in our ansatze fail considerably and therefore Eq. (24) no longer expresses correctly the condition for normalization). To illustrate this, we simulate the model on a one-dimensional ring, which we think of as representing the preferred orientations of neurons representing a common position in visual space.⁹ We consider 180 E/I pairs at grid positions separated by 1° in preferred orientation, with 0° = 180°. All four connection types have the same width, following evidence that excitatory and inhibitory inputs received by cells in upper layers have similar orientation tuning (Ferster, 1986; Anderson, Carandini, & Ferster, 2000; Martinez, Alonso, Reid, & Hirsch, 2002; Marino et al., 2005). The connectivity takes the form

$$W_{XY}(\theta,\theta') = J_{XY} e^{-\frac{d(\theta,\theta')^2}{2\sigma_{\text{ori}}^2}}$$
(25)

⁹The paradigm we study here – suppression of response to one orientation by presentation of an orthogonal orientation – is known as "cross-orientation suppression". In V1, this appears to be primarily mediated by sublinear addition of the feedforward inputs to V1 evoked by the two stimuli (Lauritzen, Krukowski, & Miller, 2001; N. J. Priebe & Ferster, 2006; B. Li, Thompson, Duong, Peterson, & Freeman, 2006). However we use this paradigm to study how the model cortex sums responses to multiple stimuli, assuming the feedforward inputs sum linearly.

where $d(\theta, \theta')$ is the shortest distance around the circle between θ and θ' . We consider stimulation by either one oriented luminance grating or two orthogonal gratings of equal contrast. Each grating is represented by a Gaussian-shaped curve of feedforward input with width (standard deviation of the Gaussian) σ_{stim} and height *c*; a single grating is centered at $\theta = 0^\circ$, a second added grating is centered at $\theta = 90^\circ$. For any given stimulus (1 or 2 stimuli, stimulus height *c*, given stimulus width σ_{stim}) the equivalent 2-D model is found as follows: we use the same $\tau_{E,I}$, *k*, *n*, and J_{XY} 's as in the high-dimensional model and take Ψ to be the value of the convolution, at $\theta = 0$, of the connectivity Gaussian (Eq. 25 with $J_{XY} = 1$, *i.e.* the vector of weights to $\theta = 0$ normalized to equal 1 at $\theta = 0$) with \mathbf{g}^n (for our stimuli, \mathbf{g} is 1 at position 0). We use \mathbf{g}^n as a surrogate for the shape of the response, with the knowledge that at least at low contrast it gives a good approximation to this shape (Section 3.1).

The result is that the reduced 2-D model accurately reproduces the behavior of the full model as shown in Fig. 1. (See also Figs. 2–4 of Section 5.3 for a more detailed comparison in various parameter regimes; as explained at the end of Section 5.3, the bottom three rows of Figs. 4 show directly the quality of the approximations involved in the ansatze introduced after Eq. (23).) The firing rates of the cells at $\theta = 0$ vs. stimulus strength closely match the firing rates in the 2-D model (Fig. 1A). Both models show a similar transition from supralinear summation of responses to the two gratings for weak stimuli to sublinear summation or "normalization" for stronger stimuli (Fig. 1B). The network also shows a form of surround suppression, in which the "summation field size" – the stimulus width that yields maximal response for a given stimulus strength – shrinks monotonically with increasing stimulus strength, as is well known in real space (rather than orientation space) for V1 cells (Sceniak et al., 1999; Cavanaugh et al., 2002), and this behavior is extremely similar in the full and reduced models (Fig. 1C).¹⁰ Thus, the 2D model can provide a good basis for understanding more general models.

4.2 Conditions for Normalization in the 2-Dimensional System

Here we show that, when our ansatze hold, the high-dimensional network exhibits normalization precisely when the 2-dimensional network shows sublinear scaling. We

consider steady-state **r** or **y** and use expressions like $\frac{\partial r_x}{\partial \psi}$ to refer to the dependence of the

¹⁰Note that this "summation field size" for orientation selectivity should not be confused with the orientation tuning width, which is the width of the orientation tuning curve obtained by studying response vs. single orientations (more precisely: studying response vs. center orientation, using stimuli that evoke a fixed curve of feedforward input vs. orientation that is symmetric about the center orientation). The orientation tuning curve, representing the set of single orientations that can drive the cell, is analogous to the "minimal response field" in real space, which represents the sum of the set of small regions in visual space in which appropriate light stimuli can evoke spiking responses. The minimal response field in real space is invariant with stimulus contrast (Song & Li, 2008), and so too is the shape of the orientation tuning curve (Skottun, Bradley, Sclar, Ohzawa, & Freeman, 1987; Anderson, Lampl, et al., 2000; Ferster & Miller, 2000) (contrast is monotonically related to the firing rate of the inputs to cortex (e.g. Ohzawa, Sclar, and Freeman (1985)). The fact that the summation field size in real space is larger than the minimal response field indicates that stimuli in regions where light cannot directly drive spikes can facilitate responses to stimuli in the minimal response field. Recall that the size of this facilitating area shrinks with contrast. The model suggests that the same may be true in the orientation domain, in terms of cortical processing of feedforward input to cells of different preferred orientations. However, attempts to test this idea will likely be compromised by two facts: (1) simultaneous presentation of multiple orientations does not yield linear summation of the input to cortex evoked by the individual orientations (Lauritzen et al., 2001; N. J. Priebe & Ferster, 2006; B. Li et al., 2006) and (2) varying the feedforward orientation tuning by changing stimulus attributes -e.g. a sinusoidal luminance grating of a given size provides drive to cortical cells with an orientation tuning that narrows with increasing spatial frequency, and similarly a longer bar drives narrower orientation tuning than a shorter bar - also changes other attributes to which the neurons are independently sensitive, such as spatial frequency or bar length.

steady state on parameters. We have seen that $r_X (X \in \{E, I\})$ exhibits normalization in response to addition of an infinitesimal second stimulus if $\frac{\partial r_X}{\partial \Psi} < 0$ in the 2-D model (more generally, for a finite-strength second stimulus, if $\int_{\Psi_{\text{init}}}^{\Psi_{\text{final}}} \frac{\partial r_X}{\partial \Psi} d\Psi < 0$). Since Eqs. 22–23 are equivalent to Eqs. 5–6 with Ψ replacing ψ , we revert to the notation of Eqs. 3–6 and use ψ .

We work with the 2-D model and express the conditions $\frac{\partial r_x}{\partial \psi} < 0$ as a single vector

condition. We note first that $\frac{\partial \mathbf{r}}{\partial \psi} = c \left(\frac{1}{\psi} \frac{d\mathbf{y}}{d\psi} - \frac{\mathbf{y}}{\psi^2} \right)$ and $\frac{d\mathbf{y}}{d\psi} = \frac{d\mathbf{y}}{d\alpha} \frac{d\alpha}{d\psi} = kc^{n-1} \frac{d\mathbf{y}}{d\alpha}$.

Putting these together we find $\frac{\partial \mathbf{r}}{\partial \psi} = \frac{kc^n}{\psi} \left(\frac{d\mathbf{y}}{d\alpha} - \frac{\mathbf{y}}{\alpha} \right)$. Thus, the condition for normalization is that \mathbf{y} grow more slowly than linearly with increasing $\mathbf{a}: \frac{d\mathbf{y}}{d\alpha} < \frac{\mathbf{y}}{\alpha}$ or $\frac{d\ln \mathbf{y}}{d\ln \alpha} < 1$ or, roughly, that $\mathbf{y} \sim \alpha^p$ for p < 1. As we have seen, p becomes less than 1 precisely when the transition from the supralinear to the sublinear scaling regime occurs.

We can reexpress this in terms of **r**. Using algebra similar to the above, we find

 $\frac{\partial \mathbf{r}}{\partial c} = \frac{(n-1)\alpha}{\psi} \left(\frac{d\mathbf{y}}{d\alpha} + \frac{\mathbf{y}}{(n-1)\alpha} \right), \text{ from which we find that } \frac{d\mathbf{y}}{d\alpha} < \frac{\mathbf{y}}{\alpha} \text{ is equivalent to } \frac{\partial \mathbf{r}}{\partial c} < n\frac{\mathbf{r}}{c}.$ Thus, the condition for normalization is that **r** grow more slowly than c^n with increasing c:

 $\frac{d \ln \mathbf{r}}{d \ln c} < n$ or, roughly, that $\mathbf{r} \sim c^p$ for p < n. Again, p becomes less than n precisely at the transition from supralinear to sublinear scaling.

Finally, noting that the steady state condition is $\mathbf{r} = k(\mathbf{Wr} + c\mathbf{g})^{\cdot n}$, without loss of generality we write $\mathbf{Wr} = c\mathbf{f}(c)$ for some vector function \mathbf{f} of c, so that the steady state condition $\mathbf{r} = k(\mathbf{Wr} + c\mathbf{g})^{\cdot n}$ becomes $\mathbf{r} = kc^n(\mathbf{f}(c) + \mathbf{g})^{\cdot n}$. Thus we see that a component of \mathbf{r} grows more slowly than c^n precisely when the corresponding component of $\mathbf{f}(c)$ is a decreasing function

of c (that is, for corresponding components r and f, $\frac{\partial r}{\partial c} < n \frac{r}{c}$ precisely when f'(c) < 0). Thus, the condition for normalization can alternatively be expressed as the requirement that the

recurrent input **Wr** grow more slowly than linearly with *c*, *i.e.* $\frac{\partial (\mathbf{Wr}/c)}{\partial c} < 0$ yields

 $\frac{\partial \mathbf{W} \mathbf{r}}{\partial c} \! < \! \frac{\mathbf{W} \mathbf{r}}{c} \operatorname{or} \frac{\partial \ln(\mathbf{W} \mathbf{r})}{\partial \ln c} \! < \! 1 \! .$

5 Analyses of the 2-Dimensional Network

We will assume throughout this analysis that $g_E = 0$, $g_I = 0$. We will use the following definitions:

$$\Omega_E \equiv \text{Det} \mathbf{J} (-\mathbf{J}^{-1} \mathbf{g})_E = J_{II} g_E - J_{EI} g_I \quad (26)$$

$$\Omega_I \equiv \text{Det} \mathbf{J} (-\mathbf{J}^{-1} \mathbf{g})_I = J_{IE} g_E - J_{EE} g_I \quad (27)$$

We also note that there are three possible conditions: (1) $(-\mathbf{J}^{-1}\mathbf{g})_E > 0$ and $(-\mathbf{J}^{-1}\mathbf{g})_I > 0$; (2) $(-\mathbf{J}^{-1}\mathbf{g})_E < 0$ and $(-\mathbf{J}^{-1}\mathbf{g})_I > 0$; and (3) $(-\mathbf{J}^{-1}\mathbf{g})_E < 0$ and $(-\mathbf{J}^{-1}\mathbf{g})_I < 0$. The 4th condition, $(-\mathbf{J}^{-1}\mathbf{g})_E > 0$ and $(-\mathbf{J}^{-1}\mathbf{g})_I < 0$, is not mathematically possible for $g_E = 0$ and $g_I = 0$: $\Omega_E > 0$ and $\Omega_I < 0$ together imply Det $\mathbf{J} < 0$, and similarly $\Omega_E < 0$ and $\Omega_I > 0$ together imply Det $\mathbf{J} > 0$.

5.1 When Does the Network Dynamically Stabilize?

5.1.1 The case of infinitely fast inhibition—We first analyze the case of infinitely fast inhibition, $\tau_I/\tau_E = 0$, with constant feedforward inputs. We show that in this case, if Det $\mathbf{J} > 0$, the network is always driven to a stable fixed point from arbitrary starting conditions. The condition Det $\mathbf{J} > 0$ means that feedback inhibition is sufficiently strong: $J_{EI}J_{IE} > J_{EE}J_{II}$. In addition, we show that if Det $\mathbf{J} < 0$, sufficiently large initial firing rates will cause the system to "blow up", *i.e.* firing rates will grow arbitrarily large.

With $\tau_I = 0$, the value of y_I is "slaved" to, or instantaneously set by the value of, y_E

according to the $\frac{dy_I}{dt}$ part of Eq. 6 for **y**. Because of the nonlinearity, we cannot solve this for y_I as a function of y_E , but we can instead solve for y_E as a function of y_I :

$$y_E = \frac{1}{J_{IE}} \left(\left(\frac{y_I}{\alpha} \right)^{\frac{1}{n}} + J_{II} y_I - g_I \right) \quad (28)$$

Substituting this in the $\frac{dy_E}{dt}$ part of Eq. 6 yields, after a bit of algebra, an equation for $\frac{dy_I}{dt}$ induced by the slaving of y_I to the y_E dynamics:

$$\tau_E \frac{dy_I}{dt} = \frac{n\alpha^{\frac{1}{n}} y_I^{\frac{n-1}{n}}}{1 + J_{II} n\alpha^{\frac{1}{n}} y_I^{\frac{n-1}{n}}} \left(-J_{II} y_I - \left(\frac{y_I}{\alpha}\right)^{\frac{1}{n}} + g_I + \frac{\alpha}{J_{IE}^{n-1}} \left(-\text{Det} \mathbf{J} y_I + J_{EE} \left(\frac{y_I}{\alpha}\right)^{\frac{1}{n}} + \Omega_I \right)^{.n} \right)$$
(29)

For sufficiently large y_I , if Det $\mathbf{J} > 0$, the term inside the parentheses in the $\frac{\alpha}{J_{IE}^{n-1}}(\dots)^{\cdot n}$ term will be negative, and so will be set to zero after the thresholding involved in the $()^{\cdot n}$ operation. The dominant term will then be the $-J_{II}y_I$ term, which is negative. So for sufficiently large y_I , $\frac{dy_I}{dt} < 0$. On the other hand, if Det $\mathbf{J} < 0$, then for sufficiently large y_I , the $(\dots)^{\cdot n}$ will be positive and larger than the sum of the other terms, so that $\frac{dy_I}{dt} > 0.0$ and, since increasing y_I will increase $\frac{dy_I}{dt} > 0$, this derivative is ever-increasing.

For sufficiently small y_I , $\frac{dy_I}{dt} > 0$ if either g_I or g_E is nonzero, which can be seen as follows. For sufficiently small y_I , the source terms g_I and Ω_I , if nonzero, dominate the terms

involving y_I . Both g_I and the $(...)^n$ term containing Ω_I are non-negative, so if either is

positive $\frac{dy_I}{dt}$ will be positive; if $\Omega_I > 0$, the $(\dots)^n$ term is positive; if $\Omega_I = 0$, this implies $g_I > 0$ (given that at least one of g_I and g_E is nonzero, and that both are non-negative).

Thus, for Det $\mathbf{J} > 0$, y_I is driven to a stable fixed point, and y_E is then determined from Eq. 28, so the system will arrive at a stable fixed point. Note that the system could have multiple fixed points with varying levels of y_I . The topology of flow along the y_I axis tells us that there must be an odd number of fixed points, alternating from stable to unstable to stable with increasing y_I , with the outermost fixed points (those with lowest and highest y_I) being stable. In the simplest case, there is a single stable fixed point. In addition, for Det $\mathbf{J} < 0$, the system will blow up for sufficiently large initial firing rates.

5.1.2 More general requirements for stability—Changes in the time constants can alter the stability of the fixed points, but do not alter the number or positions of the fixed points. The results of the previous section tells us that, for Det $\mathbf{J} > 0$, the system always has a fixed point that is stable for $\tau_I = 0$. We consider such a fixed point, and ask when it retains or loses stability for finite τ_I .

We let the fixed point be $\begin{pmatrix} y_E \\ y_I \end{pmatrix}$, and assess stability by linearizing the dynamics about this fixed point. We let $q = \tau_I / \tau_E > 0$. Setting $\tau = \tau_E$ in Eq. 6, the matrix **T** is given by

 $\mathbf{T} = \begin{pmatrix} 1 & 0 \\ 0 & q \end{pmatrix}. \text{ Define the matrix } \mathbf{\Phi} = n\alpha^{\frac{1}{n}} \begin{pmatrix} y_E^{\frac{n-1}{n}} & 0 \\ 0 & y_I^{\frac{n-1}{n}} \end{pmatrix}. \text{ Writing the identity matrix as } \mathbf{I}, \text{ the Jacobian matrix of the 2-D system is:}$

$$\mathcal{J} \equiv \begin{pmatrix} \mathcal{J}_{EE} & -\mathcal{J}_{EI} \\ \mathcal{J}_{IE} & -\mathcal{J}_{II} \end{pmatrix} = \mathbf{T}^{-1} (\mathbf{\Phi} \mathbf{J} - \mathbf{1}) \quad (30)$$

A fixed-point of the dynamics will be stable if \mathcal{J} has a negative trace and a positive determinant.

The negative trace condition is $\mathcal{J}_{EE} < \mathcal{J}_{II}$, which becomes

$$\mathscr{J}_{EE} = n\alpha^{\frac{1}{n}} y_{E}^{\frac{n-1}{n}} J_{EE} - 1 \le 0 \text{OR} \left(\mathscr{J}_{EE} > 0 \text{AND}q < \frac{n\alpha^{\frac{1}{n}} y_{I}^{\frac{n-1}{n}} J_{II} + 1}{n\alpha^{\frac{1}{n}} y_{E}^{\frac{n-1}{n}} J_{EE} - 1} \right)$$
(31)

The condition $\mathcal{J}_{EE} = 0$ means that the excitatory subnetwork by itself is stable (or marginally stable), which guarantees that the network will always be stable, since only unchecked recurrent self-excitation can destabilize the network. When the excitatory subnetwork is unstable, we can further reduce the condition on q for n = 2:¹¹

$$q < \frac{\sqrt{1 + 4\alpha J_{II}(g_I + J_{IE}y_E)}}{2J_{EE}\sqrt{\alpha y_E} - 1} (n = 2, 2\sqrt{\alpha y_E}J_{EE} > 1) \quad (32)$$

The determinant condition, Det $\mathcal{J} > 0$, is always true for any fixed point that is stable at q = 0. To see this, note that the sign of the determinant does not depend on q for q > 0 (because

Det **AB** = Det **A** Det **B** for any matrices **A**, **B**, and Det $\mathbf{T}^{-1} = \frac{1}{q}$). So if we prove that Det $\mathscr{J} > 0$ for arbitrarily small q > 0, we will have shown that it holds for all q > 0. For q = 0, the determinant, which is the product of the two eigenvalues, was infinite: because the fixed point was stable, both eigenvalues had negative real part: one real part was infinite, corresponding to the instantaneous flow onto the inhibitory nullcline (the line in the y_E/y_I

plane on which $\frac{dy_I}{dt}$ =0); the other was finite, corresponding to the flow along the nullcline converging onto the fixed point. (Since the two real parts were unequal, both eigenvalues were real.) As *q* is moved infinitesimally from 0, the infinite eigenvalue becomes a large but finite negative eigenvalue, while the finite eigenvalue is perturbed by arbitrarily small amounts as *q* is made arbitrarily small. This means that there is a range of *q* > 0 for which the eigenvalues continue to have negative real parts, and therefore for which the determinant condition holds. Therefore, the determinant condition holds for all *q*. Thus, for a fixed point that is stable for *q* = 0, the fixed point remains stable so long as condition 31, or condition 32 for *n* = 2, is satisfied.

We also note that, for the case n = 2 and for q = 1, a sufficient condition to conclude that there is only a single fixed point, which is stable, is Det $\mathbf{J} > 0$ and $J_{EE}^2 < J_{IE} J_{II}$, which can be seen as follows. The determinant condition is Det $(\mathbf{\Phi}\mathbf{J} - \mathbf{1}) > 0$. We note that, for an arbitrary 2-dimensional matrix \mathbf{M} , Det $(\mathbf{M} - \mathbf{1}) = \text{Det }\mathbf{M} - \text{Tr }\mathbf{M} + 1$. Thus, the determinant condition is Det $\mathbf{\Phi}\mathbf{J} > \text{Tr }\mathbf{\Phi}\mathbf{J} - 1$. Since Det $\mathbf{\Phi} > 0$ (because firing rates and α are > 0), this condition will be satisfied if Det $\mathbf{J} > 0$ and Tr $\mathbf{\Phi}\mathbf{J} < 1$. The trace condition for stability is Tr $\mathbf{T}^{-1}\mathbf{\Phi}\mathbf{J} < 1 + q$. But, for q = 1 and given the structures of $\mathbf{\Phi}$ and \mathbf{J} , Tr $\mathbf{T}^{-1}\mathbf{\Phi}\mathbf{J}$ Tr $\mathbf{\Phi}\mathbf{J}$, so the condition Tr $\mathbf{\Phi}\mathbf{J} < 1$ ensures that the trace condition is also satisfied. This condition is

$$J_{EE} y_{E}^{\frac{n-1}{n}} - J_{II} y_{I}^{\frac{n-1}{n}} < \frac{1}{n\alpha^{\frac{1}{n}}}$$
(33)

For n = 2, we substitute the solution for $\sqrt{y_1}$ as a function of y_E (footnote 11) into Eq. 33 for

n = 2 to find $J_{EE}^2 - J_{IE}J_{II} < \frac{1+4\alpha g_I J_{II}}{4\alpha y_E}$. Since the right side is positive, a sufficient condition for this to be true is $J_{EE}^2 < J_{IE}J_{II}$. Recall that, if there is more than one fixed point, some will be unstable at q = 0, and they must remain unstable for some region of small but finite q. Since this condition guarantees that any fixed point is stable, we conclude that there can only be one fixed point, which is stable, when this condition holds.

In summary, for $q = \tau_I / \tau_E = 0$, the network always flows to a stable fixed point if Det **J** > 0. For q > 0, a fixed point that is stable at q = 0 remains stable when Eq. 31 or, for n = 2, Eq.

¹¹This condition is found by solving $\sqrt{y_I} = \sqrt{\alpha}(J_{IE}y_E - J_{II}y_I + g_I)$ as a quadratic equation for $\sqrt{y_I}$. Discarding the negative solution, this yields $\sqrt{y_I} = \frac{-1 + \sqrt{1 + 4\alpha}J_{II}(g_I + J_{IE}y_E)}{2J_{II}\sqrt{\alpha}}$. Substituting this into Eq. 31 for n = 2 yields Eq. 32.

32 is satisfied. Note that this condition does not ensure that the network always flows to a stable fixed point; for nonzero q there may be initial conditions outside the basin of attraction of the stable fixed point or points. A condition that ensures that any fixed point is stable for q = 1, and therefore that there is only one fixed point, is Det $\mathbf{J} > 0$ and

 $J_{EE}^2 < J_{IE} J_{II}$. If there are no limit cycles (stable or unstable), this ensures that the network will flow to the stable fixed point. Excepting Eqs. 31–32, these conditions involve feedback

inhibition being sufficiently strong: $J_{IE}J_{EI} > J_{EE}J_{II}$, and $J_{IE} > \frac{J_{EE}^2}{J_{II}}$.

In Fig. 2, bottom row, we will illustrate the range of q's yielding stability for various parameter choices with n = 2.

5.2 The case $(-J^{-1}g)_E < 0$ and supersaturation

We consider Eq. 3 for **r**, but substituting ψ **J** for **W**. We restrict to the case Det **J** > 0, which

ensures a stable fixed point for at least some range of $\frac{\tau_I}{\tau_E} > 0$. We note that for Det $\mathbf{J} > 0$, $(-\mathbf{J}^{-1}\mathbf{g})_E < 0$ and $(-\mathbf{J}^{-1}\mathbf{g})_I < 0$ are equivalent to $\Omega_E < 0$ and $\Omega_I < 0$, respectively.

We shall equate increasing or decreasing c with increasing or decreasing stimulus contrast. This is based on the fact that the contrast of a visual stimulus is monotonically (but nonlinearly) related to the firing rate of the inputs to V1 from the lateral geniculate nucleus (LGN) (*e.g.* Ohzawa et al. (1985)).

In simulations, we find that if $\Omega_E < \Omega_I < 0$ for $g_E = g_I$, then r_E grows with c for a range of c considerably beyond the transition from supralinear to sublinear behavior, but ultimately peaks and is pushed back to 0 with increasing c (see Fig. 2A). The inputs to cortex have limited dynamic range (*e.g.* stimulus contrast cannot increase beyond 100%), and so we imagine that this circuit may model cortex but that the maximal input strength seen biologically cannot drive excitatory responses too far beyond their peak. The decrease in response with increasing contrast after a peak response is referred to as "supersaturation", and is seen in virtually all V1 cells for contrasts larger than about 75% (Ledgeway, Zhan, Johnson, Song, & Baker, 2005; C. Y. Li & Creutzfeldt, 1984; Tyler & Apkarian, 1985; Peirce, 2007). This model behavior provides one possible explanation for supersaturation, although supersaturation might also in part reflect a supersaturation with increasing contrast.

Here we analyze this behavior. We shall find that (1) if **r** is a stable fixed point, then $\frac{\partial r_E}{\partial c}$ $\partial r = \frac{g_E}{\partial c}$ $Q < -\frac{g_I}{\partial c}$

and $\frac{\partial r_I}{\partial c}$ are negative precisely when $\Omega_E < -\frac{g_E}{n\psi k^{\frac{1}{n}}r_I^{\frac{n-1}{n}}}$ and $\Omega_I < -\frac{g_I}{n\psi k^{\frac{1}{n}}r_E^{\frac{n-1}{n}}}$, respectively (and so in particular can only be negative if $\Omega_E < 0$ or $\Omega_I < 0$, respectively); (2) if $\Omega_E < 0$, then there is a stable fixed point with $r_E = 0$ at a finite positive value of c, which

we calculate; and, (3) for n = 2, if in addition $\Omega_E < \frac{g_E^2}{g_I^2} \Omega_I$, then the set of fixed point

excitatory rates, $r_E(c)$, has a maximum (where $\frac{\partial r_E}{\partial c} = 0$ and $\frac{\partial^2 r_E}{\partial c^2} < 0$), which is typically the peak of r_E before it is pushed to zero, and we calculate the corresponding *c* and peak value of r_E .

The condition $\Omega_E < 0$ states that the linear term in *c* in the high-*c* expansion for r_E (Eq. 15) is negative, driving r_E to zero. A related criterion for supersaturation was noted by Persi, Hansel, Nowak, Barone, and van Vreeswijk (2011), who studied a high-dimensional ring network with power-law input/output functions much like the ring model studied here, though with varying connectivity widths for the four connection types and different power laws for E vs. I cells. They assumed a stimulus of orientation θ gave feedforward input

 $I_X e^{-\frac{d(\theta,\theta')}{2\sigma_{X,LGN}^2}}$ to cells of population *X* (E or I) and preferred orientation θ' . For $I_E = I_I$, their criterion for supersaturation was $J_{EI}\sigma_{I,LGN} - J_{II}\sigma_{E,LGN} > 0$, which appears closely related to

our criterion $\Omega_E < 0$, *i.e.* $J_{EI}g_I - J_{II}g_E > 0$. Our other condition, $\Omega_E < \frac{g_E^2}{g_I^2} \Omega_I$, states that the linear term in the expansion for r_I is either positive, or not so negative as to disrupt the ability of inhibition to drive r_E to zero.

These results suggest, but do not prove, that r_E will be driven to zero for arbitrary n whenever $\Omega_E < 0$ (although there is a stable fixed point with $r_E = 0$ at finite c, we have not proven that it is the *only* stable fixed point). We note that r_I can never be zero for finite c if $g_I > 0$ (since $r_E = 0$), so given $g_I > 0$, r_I can never be driven to zero with increasing c even for $\Omega_I < 0$.

For $\Omega_E < \Omega_I < 0$, $g_E = g_I$, we find in simulations that r_I only increases with increasing *c* (see

Fig. 2A). We speculate that, for $\Omega_E < 0$ and $\Omega_E < \frac{g_E^{f(n)}}{g_I^{f(n)}} \Omega_I$, where f(n) may equal n or may equal 2, r_E can never become large enough to set $\frac{\partial r_I}{\partial c} < 0$, while r_I always becomes large enough to set $\frac{\partial r_E}{\partial c} < 0$ and so ultimately to drive r_E to zero.

When $\Omega_I < \Omega_E < 0$ for $g_E = g_I$, we find unbiological behavior in simulations in which both r_E and r_I jump to very high levels at very low c, after which r_E monotonically decreases and is ultimately pushed to 0 (see Fig. 2E). Numerical calculations suggest a discontinuity at the

jump, which may explain why our calculations do not find a zero of $\frac{\partial r_E}{\partial c}$ for real positive c in this case. We have not tried to analyze this behavior.

5.2.1 When can r_E or r_l decrease with contrast?—We define the matrix

 $\Phi_{\mathbf{r}} = nk^{\frac{1}{n}} \begin{pmatrix} r_{E}^{\frac{n-1}{n}} & 0\\ 0 & r_{I}^{\frac{n-1}{n}} \end{pmatrix} = \frac{1}{\psi} \Phi_{\mathbf{r}}.$ Then a simple calculation shows that $\frac{\partial \mathbf{r}}{\partial c} = \Phi_{\mathbf{r}}(\psi \mathbf{J} \frac{\partial \mathbf{r}}{\partial c} + \mathbf{g})$ or $\frac{\partial \mathbf{r}}{\partial c} = (1 - \psi \Phi_{\mathbf{r}} \mathbf{J})^{-1} \Phi_{\mathbf{r}} \mathbf{g},$ which gives

$$\frac{\partial \mathbf{r}}{\partial c} = \frac{nk^{\frac{1}{n}} \left(\begin{array}{c} r_E^{\frac{n-1}{n}} \left(\Omega_E n\psi k^{\frac{1}{n}} r_I^{\frac{n-1}{n}} + g_E \right) \\ r_I^{\frac{n-1}{n}} \left(\Omega_I n\psi k^{\frac{1}{n}} r_E^{\frac{n-1}{n}} + g_I \right) \end{array} \right)}{\operatorname{Det}(\mathbf{1} - \psi \mathbf{\Phi}_{\mathbf{r}} \mathbf{J})} = \frac{\frac{1}{\psi} n\alpha^{\frac{1}{n}} \left(\begin{array}{c} y_E^{\frac{n-1}{n}} \left(\Omega_E n\alpha^{\frac{1}{n}} y_I^{\frac{n-1}{n}} + g_E \right) \\ y_I^{\frac{n-1}{n}} \left(\Omega_I n\alpha^{\frac{1}{n}} y_E^{\frac{n-1}{n}} + g_I \right) \end{array} \right)}$$
(34)

Stability requires that Det $(1 - \Phi_r W) > 0$. Thus, this expression shows that, for a stable fixed point, r_E or r_I decrease with contrast precisely when

$$\Omega_{\scriptscriptstyle E} < - \frac{g_{\scriptscriptstyle E}}{n\psi k^{\frac{1}{n}} r_{\scriptscriptstyle I}^{\frac{n-1}{n}}} = - \frac{g_{\scriptscriptstyle E}}{n\alpha^{\frac{1}{n}} y_{\scriptscriptstyle I}^{\frac{n-1}{n}}} \left(\frac{\partial r_{\scriptscriptstyle E}}{\partial c} < 0\right) \quad (35)$$

$$\Omega_{I} < -\frac{g_{I}}{n\psi k^{\frac{1}{n}} r_{E}^{\frac{n-1}{n}}} = -\frac{g_{I}}{n\alpha^{\frac{1}{n}} y_{E}^{\frac{n-1}{n}}} \left(\frac{\partial r_{I}}{\partial c} < 0\right) \quad (36)$$

5.2.2 The *c* **at which** r_E **becomes 0**—The c > 0 at which r_E first becomes 0 with increasing *c* can be determined as follows. First, at this *c*, $r_I = cg_E/\psi J_{EI}$, because this is the value of r_I that sets the input to r_E to zero when $r_E = 0$. The equation for the r_I steady state

then yields $\frac{cg_E}{\psi J_{EI}} = k \left(cg_I - cg_E \frac{J_{II}}{J_{EI}} \right)^n = kc^n \left(\frac{-\Omega_E}{J_{EI}} \right)^n$. The right side gives zero unless $\Omega_E < 0$, so a solution for c = 0 exists only for $\Omega_E < 0$. In this case, one can solve to find

$$c = J_{EI} \left(\frac{g_E}{k\psi(-\Omega_E)^n} \right)^{\frac{1}{n-1}}.$$
 This corresponds to $\alpha = \frac{J_{EI}^{n-1}g_E}{(-\Omega_E)^n}$ or $\beta = \frac{-\Omega_E}{(J_{EI}^{n-1}g_E)^{\frac{1}{n}}}.$ 12

Note that any fixed point $y_E = 0$, $y_I > 0$ is stable for any q since the Jacobian matrix is

$$\mathscr{I} = n\alpha^{\frac{1}{n}} \begin{pmatrix} -1 & 0\\ \frac{1}{q} y_{I}^{\frac{n-1}{n}} \psi J_{IE} & -\frac{1}{q} \begin{pmatrix} y_{I}^{\frac{n-1}{n}} \psi J_{II} + 1 \end{pmatrix} \end{pmatrix}, \text{ which has two negative eigenvalues}$$

(equal to the two diagonal entries of \mathcal{J}).

This shows that $r_E = 0$, $r_I = cg_E/\psi J_{EI}$ is a stable fixed point for this value of *c*, but does not rule out the existence of other fixed points.

5.2.3 Peak excitatory firing rate and corresponding contrast—For the case n = 2 and $\Omega_E < 0$, we can calculate explicitly the contrast at which the steady-state excitatory

firing rate reaches a local maximum, $\frac{\partial r_E}{\partial c} = 0$, and the corresponding excitatory firing rate. We will refer to this as the peak excitatory firing rate. We are imagining that there is a continuous curve of stable fixed points vs. contrast stretching from zero firing rate for c = 0 to the fixed point with $r_E = 0$ at positive c found in the previous section and that the

$$r_{I} = \frac{\left(\sqrt{1 + 4cg_{I}\psi J_{II}k^{2}} - 1\right)^{2}}{4k\psi^{2}J_{II}^{2}}, \text{ and } r_{E} \text{ remains } 0.$$

¹²Once r_E has been pushed to zero, for increasing c, r_I continues to increase according to $r_I = k(cg_I - \psi J_{II}r_I)^n$, which for n = 2 has

dynamics converge to these fixed points, in which case we are finding the peak steady-state excitatory firing rate. This has been the case in all the simulations we have studied. However, for other parameters it is possible that other stable fixed points may appear, that the maximum occurs at negative c and thus is biologically irrelevant (further discussed below), and/or that the local maximum found here is an unstable fixed point.

To find the peak excitatory firing rate and the *c* at which it occurs for the case n = 2, we have to solve the steady-state equations $k(\psi \mathbf{Jr} + c\mathbf{g})^2 = \mathbf{r}$, together with the extremum

condition $\frac{\partial r_E}{\partial c} = 0$, for the three variables r_E , r_I and c. The extremum condition is given by Eq. (35) with equality, which for n = 2, yields $\sqrt{4k\psi^2 r_I} = -\frac{g_E}{\Omega_E}$ (as noted above, this can only happen when $\Omega_E < 0$, which we assume here). In order to simplify the equations, we

will change variables according to $\mathbf{r} = \frac{\mathbf{x}^{2}}{4k\psi^{2}}$, where without loss of generality we require \mathbf{x} to be real and positive, ensuring the positivity of \mathbf{r} . We also rewrite the steady-state equations by taking their square roots as

$$\mathbf{J}\mathbf{x}^{\cdot 2} - 2\mathbf{x} = -4k\psi c\mathbf{g}, \quad (37)$$

while the extremum condition now becomes $x_I = -\frac{g_E}{\Omega_E} = \frac{g_E}{|\Omega_E|}$. Multiplying Eq. (37) by \mathbf{J}^{-1} , we obtain $\mathbf{x}^{\cdot 2} - 2\mathbf{J}^{-1}\mathbf{x} = 4k\psi c \frac{\mathbf{\Omega}}{\text{Det }\mathbf{J}}$ where the vector $\mathbf{\Omega} = \begin{pmatrix} \Omega_E \\ \Omega_I \end{pmatrix}$. We then multiply the *E* and *I* components of the latter equation by Ω_I and Ω_E , respectively (we assume $\Omega_I = 0$), and subtract the results. After simplifying, this yields $\Omega_I x_E^2 - \Omega_E x_I^2 + 2g_I x_E - 2g_E x_I = 0$. Finally, substituting for x_I using the extremum condition, we obtain

$$\Omega_I x_E^2 + 2g_I x_E - \frac{g_E^2}{|\Omega_E|} = 0, \text{ which has one positive solution}$$
$$x_E = \frac{g_I}{\Omega_I} \left[\sqrt{1 + \frac{g_E^2}{g_I^2} \frac{\Omega_I}{|\Omega_E|}} - 1 \right], \quad (38)$$

yielding

$$r_E^{\max} = \frac{x_E^2}{4k\psi^2} \quad (39)$$

for the maximum firing rate. Note that for this to be positive, x_E must be real, which means an additional condition needs to be satisfied, namely $g_E^2 \Omega_I > -g_I^2 |\Omega_E|$.

The contrast, c^{max} , at which r_E peaks can then be solved for using either component of Eq. (37), yielding

$$c^{\max} = \frac{1}{4k\psi g_E} \left(J_{EI} \frac{g_E^2}{\Omega_E^2} + 2x_E - J_{EE} x_E^2 \right)$$
(40)
$$= \frac{1}{4k\psi g_I} \left(J_{EI} \frac{g_E g_I}{\Omega_E^2} + \frac{g_E}{|\Omega_E|} - J_{IE} x_E^2 \right)$$
(41)

with x_E given by Eq. (38). Explicit calculation shows that this is always a maximum: the 2nd

derivative $\frac{d^2 r_E}{dc^2} < 0. \ c^{\text{max}}$ is not guaranteed to be positive – this is governed by rather complicated conditions on the g's and J's – but in practice we have found it to be positive for the simulation parameters we have used.¹³

When c^{\max} is positive and r_E^{\max} is indeed the peak steady-state excitatory firing rate, then Eqs. 39 and 41 show that both the maximum excitatory firing rate that can be achieved by the network and the contrast at which this maximum is achieved decrease with increasing ψ . In this case, when the 2-D reduced model, Eqs. 22–23, accurately captures the high-dimensional model, Eqs. 18–19, then in the high-D model, if the stimulus is widened or a second stimulus is added, the maximum excitatory firing rate will decrease and will occur at a lower contrast.

In sum, for Det $\mathbf{J} > 0$ and n = 2, the steady state solution for r_E has a maximum value as a

function of c (*i.e.*, a point with $\frac{\partial r_E}{\partial c} = 0$ and $\frac{\partial^2 r_E}{\partial c^2} < 0$), given by Eq. 39, precisely when (1) Ω_E < 0 and (2) $\frac{g_I^2}{g_E^2} \Omega_E < \Omega_I$.

5.3 Steady-state solutions for different parameter regimes

In Figs. 2–3 we illustrate model behavior, as a function of stimulus strength *c*, for 5 parameter regimes, with Det $\mathbf{J} > 0$, n = 2, and $g_E = g_I$ in all cases. In Fig. 2 we illustrate behavior across a large range of *c*, sufficient to see overall model behavior. To better illustrate the region around the transition to normalizing behavior, in Fig. 3 we replot Fig. 2 but restricting to the range c = 0 to 40. The 5 illustrated parameter regimes are: $\Omega_E < 0$ and $\Omega_I < 0$, with either $\Omega_E < \Omega_I$ (Figs. 2–3A) or $\Omega_E > \Omega_I$ (Figs. 2–3E); $\Omega_E < 0$ and $\Omega_I > 0$ (Figs. 2–3B); and $\Omega_E > 0$ and $\Omega_I > 0$, with either $\Omega_E < \Omega_I$ (Figs. 2–3C) or $\Omega_E > \Omega_I$ (Figs. 2–3D). We chose parameters relatively arbitrarily, by starting with a set of parameters that had worked well in simulations of the ring model (Fig. 1 and Fig. 2–3A) and changing small sets of

negative slope. We saw in section 4.2 that $\frac{\partial \mathbf{r}}{\partial c} = \frac{(n-1)\alpha}{\psi} \left(\frac{d\mathbf{y}}{d\alpha} + \frac{\mathbf{y}}{(n-1)\alpha} \right)$, so $\frac{\partial \mathbf{r}}{\partial c} = 0$ implies $\frac{d\mathbf{y}}{d\alpha} = -\frac{\mathbf{y}}{(n-1)\alpha}$, *i.e.* \mathbf{y} is locally evolving as $\alpha^{-\frac{1}{n-1}}$.

parameters to change the regime. However in small amounts of studies of other parameters in the different regimes we have found behaviors to be similar to those illustrated, with one exception. For $\Omega_E < 0$, the transition to sublinear scaling can occur and the excitatory firing rate can peak and be driven to zero without the excitatory rate ever reaching a level at which the excitatory subnetwork is unstable. This would be manifested in the figures as stability for all possible values of q (fifth row, described below). In simulations we only encountered this for $\Omega_I > 0$ with relatively weak J_{EE} (note that $\Omega_I > 0$ includes the case $J_{EE} = 0$, and presumably sufficiently small J_{EE} behaves similarly to that case), but it can occur for $\Omega_I < 0$ as well. The conditions in which the excitatory subnetwork does not become unstable are discussed more generally in Section 5.4.

For each set of parameters, we first illustrate firing rates (top row), with red and blue indicating r_E and r_I respectively. As expected, parameters with $\Omega_E < 0$ (columns A,B,E) all show r_E eventually pushed to zero with increasing c, while those with $\Omega_E > 0$ (columns C,D) show r_E moving toward linear growth with increasing c. The combination $\Omega_I < \Omega_E < 0$ (column E) leads, as mentioned previously, to unbiological behavior in which both E and I rates abruptly jump (discontinuously, in numerical calculations with c discretized in 0.00001 steps) to high rates at low c, after which r_E monotonically falls with increasing c.

If biology is represented by a case with $\Omega_E < 0$ and $\Omega_E < \Omega_I$ (columns A,B), we imagine the dynamic range of cortex, corresponding to the dynamic range of the firing rates of the inputs to cortex, represents a smaller range extending up to and slightly beyond the point at which r_E peaks as a function of c, as discussed in Section 5.2. An example is the range through c = 100 in Fig. 1A, reduced model, 1 stimulus, which uses essentially the same parameters as Figs. 2–3A. Biologically, supersaturation begins at high contrasts, *e.g.* 75% (C. Y. Li & Creutzfeldt, 1984), well beyond the contrasts (10%–20%) at which the transition from sublinear to supralinear summation (Heuer & Britten, 2002; Ohshiro et al., 2011) or from surround facilitation to surround suppression (Sengpiel et al., 1997; Polat et al., 1998) occur. That is, while the dynamic range of cortex ends shortly after supersaturation is seen, much of this dynamic range exhibits normalizing behavior. Similarly, the model shows a broad dynamic range between the onset of normalization and of supersaturation for most parameter choices we have explored, the only exception again being the case $\Omega_E < 0$ and $\Omega_I > 0$ for small J_{EE} .

We next illustrate normalization weights (second row), computed just as in Fig. 1B, right column, so that weights > 1 (weights < 1) indicate supralinear (sublinear) summation of responses to two orthogonal gratings of equal strength in the corresponding ring model. All but the case $\Omega_E > \Omega_I > 0$ show a regime of supralinear summation for very low contrasts (behavior in all cases is sublinear for c > 10), although the supralinear behavior is weak for $\Omega_I > 0$.

The third and fourth rows of Fig. 3 illustrate the iterative solutions that stem from the scaling solutions in the low- and high-contrast regimes (the high-contrast iterative solutions are also illustrated in the third row of Fig. 2). The values of **J** used (listed in legend of Fig. 2) are not normalized to have $\|\mathbf{J}\| = 1$, so for these iterations we take $\hat{\mathbf{J}} = \mathbf{J}/\|\mathbf{J}\|$ where $\|\mathbf{J}\|$ is the 2-norm of **J** (the maximum singular value of **J**), and redefine α and **y** such that $\alpha = kc^{n-1}\psi\|\mathbf{J}\|$

and $\mathbf{r} = \frac{\mathbf{y}c}{\psi \|\mathbf{J}\|}$. We show the iteration results as r_E vs. *c*. The reason for this rescaling is that, as discussed in Section 3, with this definition of α the transitions to the sublinearly normalizing regime happen at $\alpha \sim 1$, irrespective of $\|\mathbf{J}\|$.

The small- α (low contrast) iterations are shown in the third row of Fig. 3. Here, we treat the equation $\mathbf{y} = \alpha (\mathbf{\hat{T}} \mathbf{y} + \mathbf{g})^{.n}$ as the recurrence relation

$$\mathbf{y}[t] = \alpha (\mathbf{\hat{J}}\mathbf{y}[t-1] + \mathbf{g})^{n} \quad (42)$$

Iteration of this recurrence relation generates higher- and higher-order approximations to Eq. 8 for the steady state. We use the starting condition y[0] = 0. Results are shown for numbers of iterations ranging from 1 to 19 (red through yellow colors). As few as 5 iterations gives a good approximation for small c, while increasing the number of iterations to 19 adds little. The low-contrast iterations all fail before or very slightly after c = 10, which corresponds to a in the range 1.4 to 2.4 across the parameters. That is, the failure occurs for $a \sim 1$, as expected.

For the high-a or small- β (high contrast) case, we treat the equation $\mathbf{y} = \mathbf{\hat{J}}^{-1} \left(-\mathbf{g} + \beta \mathbf{y}^{\cdot \frac{1}{n}} \right)$ as the recurrence relation:

$$\mathbf{y}[t] = \mathbf{\hat{J}}^{-1} \left(-\mathbf{g} + \beta \mathbf{y}[t-1]^{\cdot \frac{1}{n}} \right) \quad (43)$$

Iterations generate approximations to Eq. 14 for the steady state. We use as starting conditions $y_E[0] = 0$ with $y_I[0] = 0$ for $\Omega_E > 0$, $y_I[0] = g_I/\hat{J}_{EI}$ for $\Omega_E < 0$. For $\Omega_E < 0$, using $\mathbf{y}[0] = 0$ would give complex solutions. We instead use as a starting condition the value of \mathbf{y} when y_E reaches zero with increasing *c*. Recall that β increases with decreasing *c*, $\beta = 1/\sqrt{\alpha}$. For small β (large contrasts) we expect a basin of attraction about the fixed point, such that the iterations will converge to the fixed point for initial conditions in the basin of attraction. As β increases (contrast decreases) the basin of attraction should disappear for $\beta \sim 1$, so that convergence will fail for any initial condition.

The third row of Fig. 2 (fourth row of Fig. 3) illustrates these high-contrast solutions, with blue through cyan colors corresponding to 1 through 19 iterations. Again, 5 iterations do about as well as larger numbers of iterations. The iterations give good approximations for high *c* but, for $\Omega_E < 0$, fail for larger *c* as r_E approaches zero. β is very small for these large *c*'s, so this presumably represents the initial conditions no longer being in the basin of attraction of the fixed point. For low *c* failure of convergence is expected for $\beta \sim 1$, although problems with the basin of attraction could also arise. None of the iterations work for *c* below about 9 or 10, corresponding to β roughly above .65 to .85, with the exception of column E. In that column, the largest number of iterations works down to the jump in r_E and r_I , which occurs at about c = 5.435 for the given parameters, or β around 1.1. In column D the iterations do not work below *c* about 190, which corresponds to β above about 0.15, a somewhat lower value than expected for the β at which iterations fail.

In the fourth row of Fig. 2 (fifth row of Fig. 3), we show the value of $q = \frac{\tau_I}{\tau_E}$ that divides stability (values below curves) from instability (values above curves) of the fixed point, according to Eq. 32. In all cases except $\Omega_I < \Omega_E < 0$, the fixed point remains stable for q < 1 across the range of studied stimulus strengths, indicating that fine tuning or unreasonably small values of q are not required.

Finally, the fifth row of Fig. 2 (sixth row of Fig. 3) shows the extent of the sublinearly normalizing regime. Specifically, the solid and dashed blue horizontal lines indicate the

range of stimulus strengths for which $\frac{\partial r_E}{\partial \psi} < 0$ and $\frac{\partial r_I}{\partial \psi} < 0$, respectively. As discussed in Section 4.2, these conditions are roughly equivalent (so long as the approximate ansatz for 2-D reduction introduced in Section 4.1 is valid) to sublinear normalization of *E* and *I* subnetworks in the full high-dimensional ring network considered in Section 4.1.¹⁴ The other horizontal lines in the plots of this row show the extent of the sublinear regime according to other criteria introduced in the next subsection, and are explained there.

In Fig. 4 we examine the quality of the approximate 2-dimensional reduction of the highdimensional ring model (Section 4.1) in the five different parameter regimes of Fig. 2. In the top two rows, we have plotted the peak responses of the *E* and *I* subnetworks and their respective normalization weights for the original high-dimensional ring network of Eqs. (16)–(17) with the connectivity given by Eq. (25), which can be compared with those in the top two rows of Fig. 2. We see that the 2-D model captures the behavior of the original model very well. Recall that the approximation involved in the reduction to the 2-D model

involved taking the ratios $\frac{\vec{w}_{XY} \cdot \hat{\mathbf{r}}_{Y}}{\vec{w}_{EE} \cdot \hat{\mathbf{r}}_{E}}$ in Eq. (20) as constants, independent of parameters of the input stimulus (*e.g.* its shape and strength), and absorbing all such dependencies into $\Psi = \vec{w}_{EE} \cdot \hat{\mathbf{r}}_{E}$. More generally, we could have defined $\Psi_{XY} = \vec{w}_{XY} \cdot \hat{\mathbf{r}}_{Y}$ (with *X*, $Y \in \{E, I\}$). In the model of Eq. (25), the vectors \vec{w}_{XY} are independent of *X* and *Y* by construction: $\vec{w}_{XY} \equiv \vec{w}$

where \vec{w} has elements $\vec{w}(\theta) \equiv \exp\left(-\frac{d(0,\theta)^2}{2\sigma_{\text{ori}}^2}\right)\Delta\theta$. Therefore in this case we only have two independent Ψ_{XY} , which we relabel as $\Psi_E \equiv \vec{w} \cdot \hat{\mathbf{r}}_E$ and $\Psi_I \equiv \vec{w} \cdot \hat{\mathbf{r}}_I$. Finally, we also made the approximation that the response shape curves, $\hat{\mathbf{r}}_X$, can be well approximated by the shape of the input, \mathbf{g} , raised to the power n, and we used the latter to calculate the ψ used in the 2-D model, *i.e.* we took $\psi = \vec{w} \cdot \hat{\mathbf{g}}^n$. The third and fourth rows of Fig. 4 show plots of Ψ_E (red), Ψ_I (blue), and ψ (green) as a function of stimulus strength, for the case of one or two grating stimuli, respectively. The bottom row of Fig. 4 plots the ratio of the curves in the fourth row (two gratings) to the corresponding ones in the third row (one grating). In the discussion of Section 4.2 we assumed that Ψ_X should typically be larger for the case of two

 ${\rm condition \ for \ normalization \ becomes} \int_{\Psi_{\rm init}}^{\Psi_{\rm final}} \! d\Psi \frac{\partial r_{\scriptscriptstyle X}}{\partial \Psi} \! < \! 0 .$

¹⁴Note that the onset of the conditions $\frac{\partial r_x}{\partial \psi} < 0$ (vertical dashed blue lines in 2nd row, Fig. 3, corresponding to onset of blue lines in bottom row) occurs for slightly higher *c* than the onset of normalizing behavior (2nd row, Fig. 3). This is because in the 2nd row we are assaying normalization in response to a finite-strength (equal-contrast) 2nd stimulus, for which, as discussed in Section 4.2, the

gratings; we then concluded that sublinear normalization weights for the E(I) subnetwork are hence roughly equivalent to $r_E(r_I)$ being a decreasing function of ψ in the 2-D reduced model. We see from the bottom row of Fig. 4 that in some parameter regimes and for high enough c this assumption can weakly fail for Ψ_E . Surprisingly, sometimes this failure is

accompanied by the condition $\frac{\partial r_E}{\partial \psi} < 0$ in the 2-D model, and yet the normalization weights in the full model are sublinear (<1). This is due to the failure of the other assumption in the ansatz, *i.e.* it is due to the fact that $\Psi_E = \Psi_I - \Psi$.

5.4 Different criteria for crossover to the sublinearly normalizing regime

As we saw in Section 4.2, the condition that the E and I responses in the high-dimensional

network be normalizing is roughly equivalent to $\frac{\partial r_E}{\partial \psi} < 0$ and $\frac{\partial r_I}{\partial \psi} < 0$ in the 2-D model, respectively. Here, r_E and r_I refer to their values at a stable fixed point. More generally, we have seen that for sufficiently low stimulus strengths the network is supralinear (with normalization weights > 1), but switches to a sublinear regime (with normalization weights < 1) as stimulus strength becomes sufficiently large. One can, however, come up with different notions or criteria for the transition from the supralinear to the sublinear regime as stimulus contrast grows. Furthermore, since this is typically a smooth crossover, and not a sharp phase transition, such different criteria in general do not yield the exact same value of contrast at the transition, although as we will see, they all yield the same order of magnitude for the transition contrast. As this crossover is at the heart of the present study, in this section we set out to examine more closely the different criteria for the transition to the sublinear regime and their inter-relationships.

We note that the criteria we will examine all involve addition of stimuli without change in the values of g_E or g_I (or, for normalization in the high-dimensional ring model, addition of a second stimulus at a different location on the ring but with the same relative strengths of inputs to E vs. I cells as the first stimulus). One can imagine a different kind of sublinearity

due to rectification, in which a stimulus 1 has a large enough $\frac{g_E}{g_I}$ that by itself it produces r_E

> 0 and r_I > 0, while a smaller stimulus 2 that has small $\frac{g_E}{g_I}$ by itself produces $r_E = 0$ and r_I > 0. Then addition of stimulus 2 to stimulus 1 would reduce r_E and, if the network is in the ISN regime in response to stimulus 1, reduce r_I (Ozeki et al., 2009; Tsodyks et al., 1997), relative to the response to stimulus 1 alone. This sublinearity due to rectification would also occur in a linear threshold network and is a separate effect from effects due to the sublinear

regime of the SSN. More generally, the results of adding stimuli with different values of $\frac{g_E}{g_I}$ are beyond the scope of what we consider in this paper.

Here we introduce the following five different criteria for the transition to the sublinearly normalizing regime of the SSN, expressed in terms of the 2-D model, and study their interrelationships:

1

The direct definition of normalization in the high-dimensional ring model, as it follows from the approximate 2-D reduction:

$$\frac{\partial \boldsymbol{r}}{\partial \psi} < 0 \iff \frac{\partial \ln \boldsymbol{r}}{\partial \ln c} < n \iff \alpha \frac{d \mathbf{y}}{d \alpha} < \mathbf{y} \quad (44)$$

where the equivalences (demonstrated in Section 4.2) hold component by component. Note that each component in Eq. (44) expresses a separate condition, *i.e.* the normalizing property for the *E* and the *I* rates, respectively.

We can obtain an expression for $\frac{d\mathbf{y}}{d\alpha}$ by looking at the variation of the fixed point equation Eq. (7), which yields $(-\mathbf{1} + \mathbf{\Phi}\mathbf{\hat{J}})\delta\mathbf{y} + (\mathbf{\hat{J}}\mathbf{y} + \mathbf{g})^n\delta\alpha = 0$. Here, $\mathbf{\Phi}$ is the diagonal matrix $n\alpha^{\frac{1}{n}} \text{diag}(\mathbf{y}^{1-\frac{1}{n}}) = n\beta^{-1} \text{diag}(\mathbf{y}^{1-\frac{1}{n}})$. Using Eq. (7) again, we obtain

$$\alpha \frac{d\mathbf{y}}{d\alpha} = -\left(\beta^{-1}\mathbf{K} - \mathbf{1}\right)^{-1}\mathbf{y} \quad (45)$$

where we defined

$$\mathbf{K} = \begin{pmatrix} K_{EE} & -K_{EI} \\ K_{IE} & -K_{II} \end{pmatrix} \equiv n \operatorname{diag}(\mathbf{y}^{1-\frac{1}{n}}) \mathbf{\hat{J}} \quad (46)$$

(note that given the positivity of $\operatorname{diag}(\mathbf{y}^{1-\frac{1}{n}})$, all K_{XY} are positive as defined). Thus Eq. (44) is equivalent (component by component) to

$$\mathbf{y} > -\beta (\mathbf{K} - \beta \mathbf{1})^{-1} \mathbf{y}.$$
 (47)

2 The instability of the excitatory subnetwork by itself (*i.e.* with r_1 frozen at its fixed point value). As we saw in Section 5.1.2, this can be expressed as

$$\mathcal{J}_{EE} > 0$$
 (48)

where the matrix $\mathscr{J} = \mathbf{T}^{-1}(\boldsymbol{\beta}^{-1}\mathbf{K} - \mathbf{1})$, defined in Eq. (30), is the Jacobian of the 2-D flow at the fixed point. Given that **T** is positive and diagonal, Eq. (48) is equivalent to $\boldsymbol{\beta}^{-1}K_{EE} > 1$, or

$$K_{EE} = n \hat{J}_{EE} y_E^{1 - \frac{1}{n}} > \beta.$$
 (49)

This criterion can also be written $n\alpha^{\frac{1}{n}}\hat{J}_{EE}y_E^{1-\frac{1}{n}} > 1$ or $nk^{\frac{1}{n}}W_{EE}r_E^{1-\frac{1}{n}} > 1$. For $\Omega_E > 0$, r_E becomes arbitrarily large and this criterion will always be met for nonzero \hat{J}_{EE} . For $\Omega_E < 0$, the latter form makes clear that the excitatory subnetwork becomes unstable iff it is unstable at the maximum value of r_E . For $\Omega_E < 0$ for n = 2, if we assume a single curve of stable fixed points vs. contrast that peaks at the maximum value of r_E given in Eq. 39, we find that the condition for instability at the maximum of r_E , and thus for the excitatory

network to become unstable, is $J_{EE}x_E > 1$ (x_E given by Eq. (38)). This can be reduced to the condition $g_E^2 \hat{J}_{EE}^2 > |\Omega_E| \left(g_I \hat{J}_{EE} + g_E \hat{J}_{IE}\right)$ (along with the condition $g_I^2 g_E^2 \Omega_E < \Omega_I$ required for real x_E). This illustrates that the excitatory subnetwork will never become unstable for sufficiently small J_{EE} . However, when other conditions are also imposed (excitatory subnetwork never becomes unstable AND Det $\mathbf{J} > 0$, $c_{\max} > 0$ (Eqs. 40–41), fixed point at c_{\max} stable) the requirements on the J's and g's for all of these conditions to be satisfied become far more complex.

3 Local instability of the low-contrast iteration scheme, Eq. (42), at its fixed point (a sufficient condition for its divergence). By local instability, we mean the instability of the linearization of Eq. (42) around the fixed point solution. It is seen from Eq. (42) that the Jacobian of this linearization is exactly $\Phi \hat{J} = \beta^{-1} K$ where K is the matrix defined above. The condition for stability of a linear recurrence equation is that the modulus of all eigenvalues of the (Jacobian) matrix be smaller than 1. Thus the iteration is linearly unstable around the fixed point if and only if at least one eigenvalue of K has modulus larger than β :

$$|\lambda_1| > \beta OR |\lambda_2| > \beta$$
 (50)

where λ_1 and λ_2 are the eigenvalues of **K**.

4 Stability of the high-contrast iteration scheme Eq. (43) at its fixed point (a necessary condition for its convergence). Similarly to the previous criterion, by this condition we mean the stability of the linearization of Eq. (43) around the fixed point. The Jacobian of the right side of Eq. (43) is given by

 $\hat{\mathbf{J}}^{-1}$ diag $\left(\frac{\beta}{n}\mathbf{y}^{\frac{1}{n}-1}\right) = \hat{\mathbf{J}}^{-1}\mathbf{\Phi}^{-1} = \beta \mathbf{K}^{-1}$. Thus the linearization is stable if and only if both eigenvalues of $\beta \mathbf{K}^{-1}$ have modulus smaller than 1. Since the eigenvalues of \mathbf{K}^{-1} are the inverse of the eigenvalues of \mathbf{K} , this is equivalent to both eigenvalues of \mathbf{K} having modulus larger than β :

$$|\lambda_1| > \beta \text{AND} |\lambda_2| > \beta.$$
 (51)

In particular, this criterion clearly implies criterion 3.

Even though these four criteria do not define exactly the same transition point (*i.e.* the smallest value of α , or the corresponding value of β , for which a criterion first holds), as we will now argue, they typically occur for similar values of α (or β) that are O(1). First, let us consider the parameter regimes for which $\Omega_E \propto (-\hat{\mathbf{J}}^{-1}\mathbf{g})_E > 0.^{15}$ As we saw in Sec. 3.2, in this case, for small β , $\mathbf{y}(\beta)$ asymptotically approaches the value $-\hat{\mathbf{J}}^{-1}\mathbf{g}$. As we have normalized the magnitude of $\hat{\mathbf{J}}$ and \mathbf{g} , this β -independent asymptotic value will be typically O(1). Therefore, from Eq. (46), \mathbf{K} also asymptotically approaches a β -independent limit with entries, eigenvalues, and norm of modulus O(1). Hence, the left hand sides of the

¹⁵As we noted after Eq. (26), for positive inputs, $g_E = 0$ and $g_I = 0$, $(-\mathbf{\hat{J}}^{-1}\mathbf{g})_E > 0$ implies $(-\mathbf{\hat{J}}^{-1}\mathbf{g})_I > 0$

Neural Comput. Author manuscript; available in PMC 2014 May 19.

inequalities in Eq. (47) and Eqs. (49)–(51) approach an O(1) positive constant value as β decreases (equivalently, as a increases), while the right hand sides asymptotically decrease linearly in magnitude with β to zero.¹⁶ Thus these criteria will always be satisfied for small enough β , and we expect that the transition (largest β for which the criterion holds) happens for $\beta \sim 1$.

The argument for parameter regimes for which $\Omega_E \propto (-\mathbf{\hat{J}}^{-1}\mathbf{g})_E < 0$ is less straightforward, as in this case $-\mathbf{\hat{J}}^{-1}\mathbf{g} = O(1)$ does not provide a valid asymptotic value for y. Instead, as we saw in the discussion of supersaturation in Sec. 5.2.1, y_E (or r_E) typically reaches a maximum and then decreases with increasing α , vanishing at a finite value of α (or β). However, as long as parameters ($\hat{\mathbf{J}}$ and \mathbf{g}) are such that (1) the maximal value of y_E (or r_E) is O(1) or larger, so that the left-hand sides of the inequalities are positive and of magnitude O(1) or larger; and (2) the contrast at which y_F (or r_F) is maximized is large enough and thus the corresponding $\beta \equiv \beta^{max}$ small enough, $\beta^{max} \ll 1$, so that the right-hand sides of the inequalities are of magnitude $O(\beta^{max})$; then there will be a finite region of β 's around β^{max} for which the above criteria are satisfied. Thus, as long as supersaturation does not begin too early, we expect that transitions according to all the above criteria typically happen for $\beta \sim$ O(1) also in this case.¹⁷ As we noted in Sec. 5.3, biologically supersaturation begins at high contrasts, e.g. 75% (C. Y. Li & Creutzfeldt, 1984), relative to the contrasts (10%-20%) at which the transition from supralinear to sublinear behavior occur (Heuer & Britten, 2002; Ohshiro et al., 2011; Sengpiel et al., 1997; Polat et al., 1998). Thus we expect that biologically relevant parameters should yield a relatively large c^{max} , and a correspondingly small $\beta^{max} \ll 1$.

In addition to the above criteria, we also introduce a fifth criterion for sublinear response, which is directly based on the sublinearity of the E and I contrast-response curves, such as those plotted in the first rows of Figs. 2–3. However, as we will see this last criterion is not always as strongly associated with the crossover that happens for α , $\beta \sim O(1)$.

5 Sublinear growth of r_E and/or r_I with stimulus strength c:

$$\frac{\partial \ln r}{\partial \ln c} < 1 \iff \frac{d \mathbf{y}}{d \alpha} < 0.$$
 (52)

Here, the inequality on the left is the mathematical expression of sublinear growth. As for criterion 1, we have two separate conditions here, stating the sublinear growth of the E and I rates, respectively. To see the equivalence with the right side in Eq. (52), note that from the definitions (4)–(5) we have $\ln \mathbf{r} = \ln \mathbf{r}$ $c - \ln \psi + \ln \psi + \text{const.}$, and $\ln \alpha = (n - 1) \ln c + \ln \psi + \text{const.}$ Thus

 $\frac{\partial \ln \mathbf{r}}{\partial \ln c} = 1 + (n-1) \frac{d \ln \mathbf{y}}{d \ln \alpha}$, from which (given that n > 1) the equivalence of the left

¹⁶The linear decrease of the right hand sides in Eqs. (49)–(51) with β is obvious. The right hand side of Eq. (47) asymptotically (as $\beta \rightarrow 0$) behaves like $-\beta \mathbf{K}^{-1}\mathbf{y}$, and thus asymptotically also decreases in magnitude linearly with β to zero.

¹⁷ An exception to this rule was noted at the beginning of Sec. Eq. (5.3). In that example, J_{EE} is atypically small, while supersaturation starts at $\alpha \sim 1$ and y_E is pushed to zero too fast, for a value of α that is not large, such that criteria 2, *i.e.* the instability of the excitatory subnetwork, is never realized.

side with $\frac{d \ln \mathbf{y}}{d \ln \alpha} < 0$ follows. Given the positivity of \mathbf{y} and \mathbf{a} the latter is equivalent to $d\mathbf{y}/d\mathbf{a} < 0$. Finally, it follows from Eq. (45) that Eq. (52) is equivalent component-wise to

$$(\mathbf{K} - \beta \mathbf{1})^{-1} \mathbf{y} > 0.$$
 (53)

The reasoning used for criteria 1–4 to argue that they should typically occur for $\beta \gtrsim 1$, cannot be used for this criteria, as the left hand side of Eq. (53) has an asymptotic value $\mathbf{K}^{-1}\mathbf{y}$ for small β , and this need not have a definite sign for either component. However, as discussed in Sec. 5.2.1, when $\Omega_E < 0$, and assuming a single curve of fixed points vs. *c*, *r*_E will eventually decrease with *c*,

i.e. eventually $\frac{\partial \ln r}{\partial \ln c}$ becomes negative, which means that for some lower value of *c* (or α) it must have become less than unity. Thus the transition according to this criterion should always occur when $\Omega_E < 0$. On the other hand, for parameter regimes for which $\Omega_E > 0$, criteria 2 may never be realized (as in columns C and D of Fig. 2),¹⁸ so this criteria is the least suitable way of characterizing this transition.

The range of stimulus strengths, *c*, corresponding to the sublinear regime according to each of these criteria is demonstrated in the bottom row of Figs. 2 and 3, for the five choices of the connectivity matrix **J** as explained in Section 5.3 (see the figure captions for further explanation). Moreover, vertical lines in the plots of Fig. 3 indicate the transition points from the supralinear (low *c*) to the sublinear (high *c*) regime. The values at the transitions of $\alpha = kc^{n-1}\psi \|\mathbf{J}\|$ (= $k\psi \|\mathbf{J}\|c$ for the value n = 2 used in the figures) according to these criteria are also given in the caption of Fig. 3. As expected, the transitions occur for $\alpha = O(1)$.

In summary, even though the transitions according to different criteria happen at different numerical values of α , the transitions according to criteria 1–4 typically happen for $\alpha \sim 1$, as motivated on general grounds in Section 3 and discussed in more detail here (with the exception that criterion 2 may not occur for some parameter regimes with weak $E \Rightarrow E$ connections). That is, all occur as part of the overall transition from supralinear behavior in the weak-input regime ($\alpha \ll 1$) to sublinear behavior in the strong-input regime ($\alpha \gg 1$). However, the transition according to criteria 5 may never be realized if $\Omega_E > 0$, and is less suitable as a marker of the overall transition studied here.

6 Discussion

We have shown in studies of a 2-D system (and found in simulation studies of higherdimensional systems, to be presented elsewhere) that the supralinear network will dynamically stabilize with increasing input strength provided the $I \Rightarrow E$ and $E \Rightarrow I$ connections mediating feedback inhibition are sufficiently strong and the inhibitory time

¹⁸This is because in those parameter regimes the zeroth order solution Eq. (11) for the high contrast regime yields positive values for both the *E* and *I* components, allowing for the subleading correction βy_1 to be negative without making the total $\mathbf{y} = \mathbf{y}_0 + \beta \mathbf{y}_1$ negative, as long as contrast is large enough (*i.e.* β is small enough). Now according to Eq. (52) criteria 2 is equivalent to $\mathbf{y}/\alpha < 0$, or $\mathbf{y}/\beta > 0$, while to leading order $\mathbf{y}/\beta = \mathbf{y}_1$. Hence if the allowed possibility $\mathbf{y}_1 < 0$ is realized, criteria 2 will never be.

constant is not too slow. This dynamic stabilization results in a change from responses scaling supralinearly to responses scaling sublinearly with the addition of a second input. The system can also yield "supersaturation", in which excitatory firing rates reach a peak with increasing input strengths and then decrease (as observed biologically (Ledgeway et al., 2005; C. Y. Li & Creutzfeldt, 1984; Tyler & Apkarian, 1985; Peirce, 2007), and as also noted theoretically by Persi et al. (2011)), with rates ultimately decreasing to zero for large enough input strengths (which presumably are beyond the dynamical range of biological inputs). The conditions for this to occur were characterized in the 2-D system. The strongest sublinear behavior, and hence behavior most likely to underly biological observations in cerebral cortex, occurs for parameters that lead to supersaturation. As we will show in work to be presented elsewhere (presented as Abstracts in Miller and Rubin (2010); Miller and Rubin (2011); Rubin and Miller (2011)), this framework offers a unifying explanation for a number of processes involving multi-input integration in sensory cortex, including normalization and surround suppression.

Many questions remain outstanding. As some examples: within the range of models analyzed here, can more precise results, analogous to those obtained here for 2-dimensional models, be obtained for higher-dimensional models, for which we only discussed general scaling arguments? For any dimensionality, can useful results be obtained as to when the network is globally stable? How will diversity of network parameters, including in particular of the power n, alter behavior? Presumably an even slightly larger mean n for I vs. E cells will enormously enhance the range of parameters that will stabilize; experiments suggest that I cells have significantly higher powers (Haider et al. (2010), Supp. Fig. S3d). How will cell-to-cell variability of *n* affect behavior? How will behavior be affected by taking into account the decreased noise level, and thus increase in n (Miller & Troyer, 2002; Hansel & van Vreeswijk, 2002), that occurs with increasing stimulus contrast (Finn et al., 2007), i.e. with increasing input firing rate? How will network behavior be modified by addition of short-term synaptic facilitation and depression (e.g. Fioravante and Regehr (2011))? Can analysis be done of more biophysically realistic models, such as networks of integrate-andfire neurons, which have an input/output function well approximated by a power law so long as they are firing on input fluctuations rather than the mean input (Hansel & van Vreeswijk, 2002)? Note that in these models, the noise level, which as just mentioned controls the power n, can itself be determined dynamically and differ between E and I cells (e.g. van Vreeswijk and Sompolinsky (1998); Renart et al. (2010)). What can we learn as we move beyond the steady state to network dynamics, particularly using more realistic models that can better capture faster dynamics and that incorporate synaptic delays? How will the network behave when we incorporate multiple types of inhibitory neurons (e.g. Isaacson and Scanziani (2011)), or of excitatory neurons, each with their own (largely still unknown) connectivity patterns and biophysical properties?

Despite the many open questions, we believe the basic findings are likely to be quite robust and to underly a wide range of cerebral cortical behavior: networks of units with supralinear input/output functions can dynamically stabilize, resulting in a transition from supralinear to sublinear input summation.

Acknowledgments

D.B.R. is supported by NIH training grant T32-GM007367 to the M.D./Ph.D. training program at Columbia University. Y.A. is supported by a postdoctoral fellowship from the Kavli Institute for Brain Science at Columbia University. K.D.M. is supported by R01 EY11001 from the NEI of the NIH and by the Gatsby Charitable Foundation through the Gatsby Initiative in Brain Circuitry at Columbia University.

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Figure 1. Two neuron approximation of the full ring model

(A) The reduced version of the model (right) produces qualitatively similar curves of response vs. stimulus strength *c* as the full model (left; for the full model, this is the response of the cells at $\theta = 0^{\circ}$). The top plots show the response curves for a stimulus composed of a single grating with orientation $\theta = 0^{\circ}$ and the bottom plots show the response for a two-grating stimulus composed of the grating at $\theta = 0^{\circ}$ and a grating at $\theta = 90^{\circ}$. In the 2-D reduced model, these two cases are represented by using $\Psi = 0.774$ for one grating and $\Psi = 1.024$ for two gratings (see Eq. (21) for the definition of Ψ and the text after Eq. (25) for the method we used to calculate these values). (B) Full and reduced models show a similar stimulus-strength-dependent transition from supralinear summation (weight < 1) of the responses to two gratings, where the weight *w* is defined as follows. For the full model, for either E or I cells, we let $R_1(\theta)$, $R_2(\theta)$, and $R_{12}(\theta)$ be the response to one grating, the other grating, or the superposition of the two, and we define

 $w = \frac{R_{12}(0)}{R_1(0) + R_2(0)}$, where $\theta = 0$ is the orientation of the first grating. For the reduced model,

we define the weight as $w = \frac{R_{12}}{R_1 + R_2}$, where R_1 , and R_{12} are the responses to one or two gratings (modeled by the two values of Ψ given above) and we set $R_2 = 0$ (by the way we defined the reduction, R_1 , R_2 and R_{12} should approximate the responses of the full model at $\theta = 0$). (C) Full and reduced models have nearly identical stimulus-strength-dependent tuning for the width in orientation, σ_{stim} , of a feedforward stimulus (full model: width of Gaussian stimulus centered at $\theta = 0$ with given stimulus strength *c* that gives the strongest response in cells at $\theta = 0$; reduced model: Ψ is computed for each stimulus width as described in the text after Eq. (25), and plot shows width whose Ψ gives maximal response). In all curves, red shows E cells and blue shows I cells. All responses are steady-state responses. Full model solutions found by simulating until convergence to steady state. Parameters: $J_{EE} = 2.5$, $J_{IE} = 2.4$, $J_{EI} = 1.3$, $J_{II} = 1.0$, $\tau_E = 20$ ms, $\tau_I = 10$ ms, k = 0.04, n = 2.0, $\sigma_{ori} = 32^\circ$; $\sigma_{stim} = 30^\circ$ in A,B.

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Figure 2. Behavior of the 2D Model in Different Parameter Regimes

Each column corresponds to a different connectivity matrix J, corresponding to different conditions on Ω_E and Ω_I as indicated at top. In all cases, Det $\mathbf{J} > 0$, n = 2.0, k = 0.04, and g_E $= g_I = 1$. The first column uses the same parameters as the 2-D reduced model in Fig. 1. In all figures the horizontal axis is stimulus strength c; Fig. 3 shows all plots (and one additional set of plots) for the smaller range $c \in [0, 40]$. Top row: E (red, top) and I (blue, bottom) firing rates, r_E and r_I , at fixed point. For cases with $\Omega_E < 0$, dashed vertical lines indicate analytic calculations for c at which r_E goes to zero (Sec. 5.2.2) and, for $\Omega_E < \Omega_I$, at which r_E peaks (Eq. 41). Second Row: Weights reecting supralinear (weight > 1) or sublinear (weight < 1) summation in an equivalent ring model, computed as in Fig. 1B. Red and blue indicate E- and I-subnetworks, respectively. Inset in column E shows supralinear responses at low values of c. Third Row: Iterative solutions for r_E in the high-contrast regime (Eq. 43). We plot $r_E[t] = y_E[t]c/(\psi ||\mathbf{J}||)$ vs. c, for t = 1, 5, 10, 14, 19 iterations (blue to cyan curves); black curves are exact solutions. Iterative solutions are shown only over the range for which they are real. (Iterative solutions in the low-contrast regime are shown in Fig. 3.) Fourth Row: Values of $q = \tau_I / \tau_E$ separating regions in which fixed point is stable (below red line) vs. unstable (above red line). Fifth Row: Horizontal lines showing the

extent of the sublinear regime according to the different definitions introduced in Sec. 5.4. Blue and red lines (*E* component solid, *I* component dashed): definitions 1 (normalization in corresponding high-dimensional ring model) and 5 (*r* a sublinear function of *c*), respectively. Green line: definition 2 (excitatory subnetwork unstable). The cyan lines show the range where the modulus of each eigenvalue of the Jacobian is > 1; sublinear regime according to definition 3 (instability of low-contrast iterative solution) or 4 (stability of high-contrast iterative solution) is the region in which either (def. 3) or both (def. 4) lines are present. Parameters used: $\psi = 0.774$ or, for two-grating case in 2nd row, $\psi = 1.024$ (the values of Ψ in Fig. 1); $J_{EI} = 1.3$; $J_{EE} = 2.5$, except 0.8 in (D); $J_{II} = 1.0$, except 2.2 in (C) and 5.0 in (D); $J_{IE} = 2.4, 4.7, 4.7, 3.6, 2.2$ in (A) to (E), respectively.

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Figure 3. Crossover of the 2D Model to the High Contrast Sublinear Regime for Different Network Parameters

The plots in this figure are the same as the ones in Fig. 2, except that (1) only the range of stimulus strengths, c, from 0 to 40 is shown, to highlight the transition to the sublinear regime as c grows and (2) we also illustrate the low-contrast iterative solutions for r_E , which have been inserted as the third row (conventions as for high-contrast iterative solutions, except here red to yellow curves represent 1 to 19 iterations). See the caption of Fig. 2 for explanation of plots and parameters. The extra horizontal axes at the bottom translate the stimulus strengths into values of α as defined in Eq. (5). In addition, vertical dashed lines in the first to fifth rows indicate the transition points to the sublinear regime, according to the different definitions introduced in Sec. 5.4 and illustrated in the bottom row, with definitions

1 to 5 corresponding to colors blue, green, orange, cyan and red, respectively (for definitions 1 and 5 the line is drawn at the point where the condition holds for both *E* and *I* components). The values of the α 's at these transition lines, in the order mentioned, are (1.4, 0.7, 1.4, 1.4, 2.4), (1.0, 1.4, 1.4, 1.4, 16.3), (2.4, 1.1, 3.1, 3.1, -), (0.2, 4.7, 1.0, 28.5, -), and (0.8, 0.6, 0.8, 0.8, 0.8) in columns A to E, respectively. Notice that the transitions to the sublinear regime typically happen for $\alpha \sim 1$, as expected.

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Figure 4. Behavior of the Full Ring Model in Different Parameter Regimes

Behavior of the steady state of the ring network of Sec. 4.1, in the same parameter regimes as in Fig. 2. The ring network's connectivity matrix is given by Eq. (25), with different **J**'s in different columns equal to those in the corresponding column of Fig. 2. The rest of the parameters are the same as in the left column of Fig. 1 (in particular, all parameters of column A match those of Fig. 1, left column). The signs and orderings of Ω_E and Ω_I are indicated on the top of each column. In all figures the horizontal axis is stimulus strength *c*. **Top row**: *E* (red) and *I* (blue) firing rates, $r_E(\theta = 0)$ and $r_I(\theta = 0)$, at fixed point. For cases with $\Omega_E < 0$, dashed vertical lines indicate analytic calculations for *c* in the 2-D reduced model at which r_E goes to zero (Section 5.2.2) and, for $\Omega_E < \Omega_I$, at which r_E peaks (Eq. 41). **Second Row**: Weights reecting supralinear summation (weight > 1) or sublinear summation (weight < 1) computed as in Fig. 1B. Again, red and blue indicate *E*-and *I*-subnetworks, respectively. Inset in column E shows supralinear responses at low values of *c*. **Third Row**:

The red and blue curves show $\Psi_E \equiv \vec{w} \cdot \hat{\mathbf{r}}_E$ and $\Psi_I \equiv \vec{w} \cdot \hat{\mathbf{r}}_I$, which we approximated by $\psi = \vec{w} \cdot \hat{\mathbf{g}}^n$ (green lines) in the 2-D reduction for the case of a one-grating stimulus (see the discussion at the end of Sec. 5.3). Fourth Row: The same as the third row, but for two-grating stimuli. Fifth Row: The red, blue and green curves show the ratios of the red, blue and green curves in the fourth row (two gratings) to those in the third row (one grating), respectively.