Competitive Dynamics in Cortical Responses to Visual Stimuli


Neurons in the inferotemporal cortex (IT) of the macaque monkey sometimes respond to presentation of a visual stimulus by emitting a series of bursts at a frequency of 5 Hz (Nakamura et al. 1991, 1992). Rollenhagen and Olson (2005) recently observed that 5-Hz oscillations are enhanced by the presence of another stimulus in the visual field. In the presence of an eccentric flanking image, displaying a central preferred image tends to elicit a strong initially positive oscillatory response (see Fig. 1A). Conversely, in the presence of a central preferred image, an eccentric flanking image, although ineffective in isolation, tends to elicit a strong initially negative oscillatory response (see Fig. 1B). They demonstrated that similar phenomena arise in an extremely simple network in which fatiguing neurons responsive to the central image and the eccentric flanker inhibit each other. They did not, however, systematically explore the dependency of oscillatory activity on the properties of the network nor did they consider the possible relation of oscillatory activity to general principles that govern visual processing in IT. These issues are taken up in the present paper.

In this study, we have used a simulation-based approach to characterize oscillatory activity and related phenomena in networks consisting of populations of neurons that are responsive to different visual stimuli and that mutually inhibit each other. We show that by varying a single parameter, the strength of cross-inhibition, it is possible to transform a network from operating in normalization mode (whereupon simultaneous presentation of the two stimuli, both neuronal populations are moderately active) to operating in a winner-take-all mode (whereupon simultaneous presentation of the two stimuli, one population is maximally active and the other is suppressed). At strengths of cross-inhibition that is transitional between those that give rise to normalization and winner-take-all behavior, networks exhibit oscillatory behavior (whereupon simultaneous presentation of the two stimuli, the two populations are active in alternation) with a frequency that decreases with stronger inhibition. We speculate that the occurrence of oscillatory activity in IT is the result of network properties transitional between those that give rise to normalization (thus favoring the simultaneous representation of multiple stimuli) and those that give rise to winner-take-all behavior (thus favoring the representation of only one stimulus out of several). The inhibition strengths between various pools of neurons may take different values during development or as a result of experience, with the consequence that pools representing antagonistic stimuli have stronger mutual inhibition than that of pools representing compatible stimuli.

Methods

Network architecture

We studied a model network of excitatory and inhibitory Hodgkin–Huxley-type conductance-based neurons. The network was an abstracted canonical cortical circuit (Douglas and Martin 2004; Raizada and Grossberg 2003) consisting of two excitatory and two inhibitory pools of 40 neurons each (Fig. 2A). Increases beyond this number had no appreciable effect on the results. Varying the numbers of inhibitory versus excitatory neurons also did not alter the qualitative results. Excitatory cells were coupled by α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)–like excitatory synapses to other cells within their pool. There were excitatory projections from excitatory pool 1 (E1) to inhibitory pool 2 (I2) and from excitatory pool 2 (E2) to inhibitory pool 1 (I1). Neurons from the inhibitory pools formed γ-aminobutyric acid-A (GABA)–like inhibitory connections on neurons in the excitatory pools, with I1 neurons projecting to E1 neurons and I2 projecting to E2. External inputs (active in response to visual stimulation) terminated on neurons of the excitatory pools. Neurons in E1 and E2 show much higher responses for presentations of preferred visual stimuli compared to nonpreferred stimuli (in Fig. 2A, stimulus 1 “Object” is the preferred stimulus for E1 and nonpreferred for E2, and stimulus 2 “Flanker” is the preferred stimulus for E2 and nonpreferred for E1).

Two slow processes gave rise to neuronal fatigue. The first was spike-frequency adaptation in the excitatory neurons attributed to a calcium-dependent potassium current (Huguenard and McCormick 1992; McCormick and Huguenard 1992; Wang et al. 2003). The second was synaptic depression with a time constant that was long.
in time when the object (a foveal image that in itself elicited a strong excitatory and inhibitory neurons, respectively. Flanker, presented against the backdrop of an already present object, in time when the flanker (an eccentric image that in itself elicited a weak)

\[\text{FIG. 1. Data from a neuron in inferotemporal cortex that responded to } V, H_2O849\]

\[\text{FIG. 2. A: network of 2 pools of excitatory neurons (E1 and E2) and 2 pools of inhibitory neurons (I1 and I2). Arrows represent } J \text{ external inputs coming to the excitatory neurons from preferred (thick arrows) and nonpreferred stimuli (thin arrows); 2) self-excitatory projections within the excitatory pools; and 3) projections from the excitatory pools to the opposite inhibitory pools, which in turn project to their ipsilateral excitatory pools. This results in mutual inhibition between the excitatory pools. B: reduced version of the network in A consisting of 2 mutually inhibitory units. As in A, the external inputs are from preferred and nonpreferred stimuli.}\]
\[
\frac{dx_l[k]}{dt} = (A \cdot \sigma (V_l) \cdot (1 - s_l[k]) - s_l[k]) / \tau_r
\]
\[
\frac{dx_i[k]}{dt} = (A \cdot \sigma (V_i) \cdot (1 - s_i[k]) - s_i[k]) / \tau_i
\]
\[
\frac{dp_e[k]}{dt} = (-f_e \cdot \sigma (V_e) \cdot \varphi_i[k] + (1 - \varphi_i[k])) / \tau_e
\]
\[
\frac{dp_i[k]}{dt} = (-f_i \cdot \sigma (V_i) \cdot \varphi_e[k] + (1 - \varphi_e[k])) / \tau_i
\]
\[
\sigma (V) = 1/(1 + \exp[-(V + 20)/4])
\]

where \(N\) is the number of excitatory neurons (equal to the number of inhibitory neurons), \(V_{e1} = -80, V_{i1} = 0, A = 20, \tau_r = 8, \tau_i = 10, \tau_{pr} = 1000, \tau_{pi} = 800, J_{ei} \) and \(J_{ie} \) varied from 0 to 1, and \(f_e \) and \(f_i \) varied from 0 to 0.05.

Similarly, the synaptic current to the \(j\)th inhibitory neuron with voltage \(V[j]\) (inhibitory neurons receive inputs only from excitatory neurons) was
\[
I_{exi} = J_e \cdot g_e \cdot (V[j] - V_i)N
\]
where \(g_e = \Sigma s_i[k] \cdot \varphi_i[k], V_{i1} = 0, \) and \(J_e \) varied from 0 to 1. The sum in \(g_e \) ran over the contralateral excitatory pool with \(k = 41 \) to 80 for \(I1 \) and \(k = 1 \) to 40 for \(I2 \). As described above \(s_i[k] \) was the gating variable of the synapses from the \(k\)th excitatory neuron with depression factor \(\varphi_i[k] \).

**External currents**

The external current \(I_{ext} \) to the excitatory neurons consisted of a constant input \(I\), which ranged from 0 to 2.5. The relative value of the external inputs to \(E1 \) and \(E2 \) neurons depended on the stimulus presented. If we presented stimulus 1 (Object), which was preferred by \(E1 \) and not preferred by \(E2 \), then the external current to \(E1 \) was higher than the external current to \(E2 \), and for a presentation of stimulus 2 (Flanker), which was preferred by \(E2 \) and not preferred by \(E1 \), the external current to \(E1 \) was higher than the external current to \(E2 \). For simultaneous presentation of both stimuli external currents to both populations were equal.

To reproduce experimental data observed by Rollenhagen and Olson (2005) we also added additional white noise to the external inputs, although all features of the model discussed in the paper such as different modes and transitions between them can be obtained in the absence of noise. For the external input to excitatory neurons we used \(I_{ext} = 2 + \text{noise}(0.5)\), where function \(\text{noise}(0.5)\) generated a random number uniformly distributed between \(-0.5 \) and \(0.5 \). The external input \(I_{ext} \) to the inhibitory neurons was \(\text{noise}(0.25)\).

**RESULTS**

**Conductance-based cortical circuit model**

The neuronal pools in our model were intended to represent two pools embedded in a network of many interconnected pools in various brain regions. Any given visual stimulus activated neurons in some pools and not in others. We considered specifically the case of two excitatory pools maximally activated by different visual stimuli and competing with each other by projections mediated by inhibitory interneurons. We presumed that different pools would inhibit each other with varying strengths of inhibition determined during development or by experience-dependent learning. By varying only the strength of the mutual inhibition, with all other parameters fixed to biophysically plausible values, we demonstrated that it was possible to elicit a broad range of distinct behaviors.

**Three functional modes dependent on strength of cross-inhibition.** To simulate the simultaneous onset of two visual stimuli, one optimal for each excitatory pool, we applied currents \(I_1 = 2.5\) to \(E1\) and \(I_2 = 2.5\) to \(E2\) and applied white-noise inputs to the inhibitory pools. We then examined the impact on network behavior of varying the strength of the inhibitory parameter \(J_{ei}\) which represented the strength of inhibition exerted by inhibitory neurons on their excitatory targets. As inhibition increased, the behavior of the network passed through three modes. At low inhibitory strength, both pools of excitatory neurons were continuously active (Fig. 3A) because inhibition was too weak to allow one of the pools to suppress the other one. Although both pools of excitatory neurons were active, neither was as active as it would have been in the sole presence of its preferred stimulus, and thus the network operated in what we termed normalization mode. If the strength of the inhibition was increased, the network entered an oscillatory mode in which the two pools were alternately active (Fig. 3B). At high inhibitory strength, the network operated in a winner-take-all mode in which only one pool remained active and the other pool was suppressed (Fig. 3C). There was a transient time (different for different modes) that was necessary for the network to settle down into a mode. It was around 100 ms for the oscillation mode and 400 ms for the winner-take-all mode. Once the network reached a given mode it was stable in it for the remaining simulation time.

**Normalization mode.** At low levels of inhibition the network operated in normalization mode, by which we mean that neurons in each excitatory pool responded to the simultaneous onset of both stimuli by firing at a rate less than that elicited by the preferred stimulus alone and more than that elicited by the nonpreferred stimulus alone. As may be seen in Fig. 4, neurons of pool 2 fired vigorously in response to stimulus 2 (Fig. 4A), fired weakly in response to stimulus 1 (Fig. 4B) and fired at a moderately reduced rate in response to the simultaneous presentation of stimuli 1 and 2 (Fig. 4C). That the rate of firing elicited by the simultaneous presentation of stimuli 1 and 2 was indeed reduced relative to the rate elicited by stimulus 1 alone.
is demonstrated in Fig. 4D. As indicated above, a larger value of input corresponded to a preferred stimulus and a smaller value corresponded to a nonpreferred stimulus. When both stimuli were present, the total input was the sum of the two inputs. For presentation of stimulus 1 “Object,” which was preferred by E1 neurons and nonpreferred by E2 neurons, we used as external currents $I_1 = 2.4$ and $I_2 = 0.1$. For presentation of stimulus 2 “Flanker,” which was preferred by E2 neurons and nonpreferred by E1 neurons, we used $I_1 = 0.1$ and $I_2 = 2.4$. For simultaneous presentation of both stimuli together, we used $I_1 = 2.5$ and $I_2 = 2.5$.

The degree to which the nonpreferred stimulus weakened the response to the preferred stimulus depended on the strength of the inhibition. In Fig. 4E, we plot the mean firing rates of one excitatory neuron from pool E2 when the preferred stimulus (stimulus 2, green line), the nonpreferred stimulus (red line), or both stimuli (black line) were presented across a range of values of inhibition strength $J_e$. As the strength of inhibition increased, the normalization effect became more pronounced.

**Oscillatory mode.** At higher strengths of inhibition, when simultaneous equal inputs to both pools were applied, the network responded with a form of oscillatory behavior in which pools of excitatory neurons preferring one stimulus or the other were alternately active. To demonstrate this effect, we maintained the input currents $I_1 = 2.5$ and $I_2 = 2.5$ while varying the strength of the inhibitory synapses $J_{ei}$. We observed that the frequency of the oscillations depended on the strength of the inhibition, with stronger inhibition giving rise to oscillations at a lower frequency (Fig. 5, A–D).

The oscillations arose from the combined influence of two fatigue mechanisms: spike-frequency adaptation with a timescale of 100 ms and synaptic depression with a timescale of 1,000 ms. Spike-frequency adaptation, mediated by a hyperpolarizing current, developed in the active pool of excitatory neurons and decreased in the suppressed pool of excitatory neurons, thus favoring a recovery of activity in the latter pool. Synaptic depression both in the active excitatory neurons and the active inhibitory neurons steadily weakened the net inhibition to the suppressed pool, also favoring recovery. Eventually the neurons in the suppressed pool overcame the inhibition of the active pool and began to fire, suppressing in turn the neurons of the initially active pool. The upper limit on the oscillatory frequency was determined by the timescale of the more rapid phenomenon, spike-frequency adaptation. As the strength of the inhibitory synapses was increased, the ability of spike-frequency adaptation to overcome suppression decreased. In this situation, the synaptic depression became relevant and thus the frequency of the oscillatory activity was lower. Thus there was a smooth transition from a fast oscillation of approximately 5 Hz to oscillations at increasingly lower frequencies as inhibition was increased (Fig. 5D). The slowest oscillations were at the timescales of perceptual oscillations such as binocular rivalry (Grossberg 1994; Laing and Chow 2002).

We found that as the oscillations became slower, they also became more stochastic. This stochasticity occurred even in the absence of any noise inputs. The origin of this stochasticity was discussed in Laing and Chow (2002). Also, similar to what was shown in Laing and Chow (2002), the model was able to reproduce all the phenomena of binocular rivalry including: a lack of correlation in dominance times from epoch to epoch, a dominance time that obeyed a Gamma-like distribution, and Levelt’s Second Proposition.

**SIMULATION OF 5-HZ OSCILLATIONS IN IT.** The above analyses of network behavior considered responses to the simultaneous onset of two visual stimuli. However, the model also reproduced firing patterns observed by Rollenhagen and Olson (2005) in response to staggered onset of two visual stimuli. While recording from a given neuron, they assessed how the response to onset of stimulus 2 was affected by the ongoing presence of stimulus 1, where stimulus 2 was the central image.
preferred by the neuron and stimulus 1 was an eccentric nonpreferred stimulus (Fig. 1A) or vice versa (Fig. 1B). As a basis for direct comparison to results obtained in the experiment, we assessed the responses of the spike-based network to successive onset of the two stimuli. When the recorded pool’s preferred stimulus appeared against the backdrop of its nonpreferred stimulus, firing took the form of an initially positive oscillatory response. When the recorded population’s nonpreferred stimulus appeared against the backdrop of its preferred stimulus, the response was an initially negative oscillation (Fig. 6, bottom), again just as in the physiological experiment (Fig. 1B).

**Winner-take-all mode.** With very strong inhibition, neither of the fatigue mechanisms was sufficiently strong to permit the initially suppressed neuron pool to escape and fire. When both visual stimuli were present, one pool of excitatory neurons became active and the other was suppressed, with the selection of the active pool dependent on stochastic factors early in the trial (Fig. 3C). This winner-take-all mode was the culmination of the trend whereby, with increasing inhibitory strength, the oscillatory frequency became progressively slower. The winner-take-all state can be thought of as a limiting case of the oscillation with zero frequency. We note that if the inhibition is not strong enough in a neural circuit then winner-take-all behavior may never be observed experimentally.

**Rate-based model**

To gain further insight into the behavior of the networks described above (two pools of excitatory conductance-based neurons reciprocally suppressing each other through inhibitory interneurons) we considered a classic rate-based model, consisting of two units inhibiting each other in the presence of fatigue (see Fig. 2B) (Carpenter and Grossberg 1983; Laing and Chow 2002). The units represented pools of excitatory neurons that inhibited each other through inhibitory neurons. For simplicity we did not include dynamics of the inhibitory units. As in the conductance-based model, we implemented two different fatigue mechanisms: spike-rate adaptation and synaptic depression. The equations describing the dynamics were

\[
\frac{d u_i}{d t} = -u_i + F(I_i - b \cdot u_i \cdot s_i - a_i)
\]

\[
\frac{d a_i}{d t} = (s_i + k_{adap} \cdot a_i)\tau_{adap}
\]

\[
\frac{d s_i}{d t} = (1 - s_i - k_{sd} \cdot s_i \cdot u_i)\tau_{sd}
\]

where \(u_i\) is the firing rate of unit \(i\), \(s_i\) gives the amount of depression of the synapse from unit \(i\), \(a_i\) is the adaptation current of unit \(i\), \(F\) is a gain function, \(b\) is the strength of inhibition, and \(I_i\) is an external current to unit \(i\) (resulting from the presence of that neuron’s preferred visual stimulus). For the simulations we used for the gain function \(F(x) = \sqrt{2 \ln [1 + \exp(x/2)]}\), and parameters had values \(\tau_{adap} = 100\), \(\tau_{sd} = 500\), \(k_{adap} = 0.5\), \(k_{sd} = 0.5\), and \(I_1 = I_2 = 5\).

The results of the simulations were not critically dependent on the choice of the gain function. We observed the same three modes with any reasonable gain function including step functions, sigmoidal functions, and piecewise linear functions. The choice of the gain function with the general form \(F(x) = \sqrt{slope \ln [1 + \exp((x - thr)/slope)]}\), where parameters can control the slope and threshold was out of convenience.

**Bifurcation diagram.** The advantage of the reduced model was that the dependency of network behavior on parametric variables could be analyzed mathematically. A standard method is to construct a bifurcation diagram that presents the dependency of the system’s behavior, including points of transition between qualitatively different behaviors, on a system parameter. As we observed in the conductance-based network, a critical parameter governing the transition between different kinds of dynamics was the strength of inhibition. We examined the behavior of the rate-based model with varying inhibition strength \(b\) and with varying input strengths to both units. The results, summarized in the bifurcation diagram of Fig. 7, were similar to those obtained with the conductance-based model. At low values of \(b\), the network exhibited normalization behavior (curve between point 1 and point 2 in Fig. 7): in the presence of both stimuli, the excitatory units maintained a steady state of activation less than would have occurred if only the preferred stimulus were on. At intermediate values of inhibition, oscillatory responses occurred (open circles forming a closed curve joining points 2, 4, and 5); the activities of the units were alternating in time, when one unit was active the other was suppressed, and after a period of time they exchanged states. At high values, winner-take-all responses occurred (top curve between points 4 and 6 or bottom curve between points 5 and 8): one unit won the competition and the other unit could not recover from suppression. Points 2, 4, and 5 are transition points (bifurcation points) between different kinds of dynamics.

**Normalization mode.** As in the spiking model for low levels of inhibition, the rate-based network operated in normalization mode. The unit activities in response to both stimuli were less...
and represents an unstable "ON–ON" state. Parameters used are the adaptive mechanisms of spike-rate adaptation or synaptic cation point. This is in concordance with the spiking model of the period of oscillation on the bifurcation parameter. The of the known features of Hopf bifurcations is the dependency used tion strength (black line) were presented across a range of values of inhibi-
line), the nonpreferred stimulus (red line), or both stimuli OSCILLATORY MODE. As we increased the strength of inhibi-
that elicited by the nonpreferred stimulus alone (see Fig. 8 A). For presentation of a stimulus that was preferred by unit 1 and not preferred by unit 2, we used as external currents \( I_1 = 4.9 \) and \( I_2 = 0.1 \). For presentation of a stimulus that was not preferred by unit 1 and preferred by unit 2, we used \( I_1 = 0.1 \) and \( I_2 = 4.9 \). For simultaneous presentation of both stimuli, we used \( I_1 = I_2 = 5 \). The degree to which the nonpreferred stimulus weakened the response to the preferred stimulus depended on the strength of inhibition. In Fig. 8 B, we plotted the mean activity of unit 1 when the preferred stimulus (green line), the nonpreferred stimulus (red line), or both stimuli (black line) were presented across a range of values of inhibition strength \( b \). As the strength of inhibition increased, the normalization effect became more pronounced.

OSCILLATORY MODE. As we increased the strength of inhibition, the network transitioned from normalization mode to oscillations through a Hopf bifurcation (point 2 in Fig. 7). One of the known features of Hopf bifurcations is the dependency of the period of oscillation on the bifurcation parameter. The period increases as the parameter moves away from the bifurcation point. This is in concordance with the spiking model where the period of oscillation increased with inhibition.

The existence of an oscillatory region depended entirely on the adaptive mechanisms of spike-rate adaptation or synaptic depression. In a classic winner-take-all network consisting of units that inhibit each other in the absence of any fatigue mechanisms, the bifurcation diagram would take the form of a stable line, corresponding to normalization mode, at low values of inhibition (curve between points 1 and 3) and two branches of winner-take-all mode, at high values of inhibition (curves between points 3 and 6 and points 3 and 8), and there would be no oscillatory region.

ROBUSTNESS OF THE MODES. The results of simulations in the rate model were not critically dependent on the other parameters. For example, they persisted across a wide range of input strength as shown by the two-parameter diagram (Fig. 9) for the strength of the inhibition \( b \) and equal external currents to both units \( I_1 = I_2 = I \). At any fixed value of external currents \( I_1 = I_2 = I \) as the inhibition strength \( b \) increased, the network passed through normalization, oscillation, and winner-take-all modes. Changes of the other parameters deformed the regions but did not alter their topology.

MULTIPLE STIMULI. Thus far we have considered specifically the case of two excitatory pools maximally activated by different visual stimuli and competing with each other by projections mediated by inhibitory interneurons. We have demonstrated that by changing only the strength of the mutual inhibition, with all other parameters fixed to biophysically plausible values, it was possible to elicit a broad range of distinct behaviors. However, presenting even two stimuli may activate more than two pools of neurons.

Thus we next probed whether similar phenomena would arise from the competition of multiple neuronal pools evoked by the presentation of multiple stimuli or by natural scenes. We ran simulations of a rate model of three units reciprocally inhibiting each other. We again found the same three distinct modes of behavior—normalization, oscillations, and winner-take-all depending on the strength of the inhibition. For weak inhibition, all three units were active simultaneously as in normalization mode. If inhibition was strong enough, one unit was active and suppressed the other two, with the choice of the dominating unit being random.

The oscillatory regime showed richer behavior than that in the two-unit network. With an increase in inhibition, the network switched from normalization mode to oscillation mode. Immediately after the transition, the oscillations took the form of alternations between one very active unit and two simultaneously less active units (each half as active as the very active unit). As the inhibition was increased, one unit became permanently suppressed while the other two activated alternately. For appropriately chosen parameters, we also observed a pattern in which the units fired in a sequential manner with

![FIG. 7. Bifurcation diagram, for the rate-based model, showing dependency of neuronal activity in the presence of both visual stimuli on the strength of inhibition \( b \). \( u \) represents the activity of a representative unit. Because input current was symmetric, the 2 units responded equally. Curve 1–2 represents stable steady states with both units active (normalization mode). Point 2 is a bifurcation point where oscillations begin (oscillatory mode). Oscillations cease at points 4 and 5, and are followed by curves 4–6 and 5–8 that represent stable "ON" and "OFF" states respectively (winner-take-all mode). Curve 3–7 represents an unstable "ON–ON" state. Parameters used are \( F(u) = \sqrt{2 \ln (1 + \exp (u/2))} \), \( \tau_{\text{adap}} = 100 \), \( \tau_d = 500 \), \( k_{\text{adap}} = 0.5 \), \( k_d = 0.5 \), \( I_1 = I_2 = 5 \).

![FIG. 8. Normalization effect in the rate model. A: rate model analog of Fig. 4 D. Activity of unit 1 as a function of time during presentation of preferred stimulus 2 (green), nonpreferred stimulus 1 (red), and both stimuli together (black). For a preferred stimulus, we applied a current, \( I_1 = 4.9 \), for nonpreferred stimulus, \( I_2 = 0.1 \), and for presentation of both stimuli, \( I_1 = I_2 = 5 \). Strength of inhibition was fixed at \( b = 7 \). Other parameters had values \( \tau_{\text{adap}} = 100 \), \( \tau_d = 500 \), \( k_{\text{adap}} = 0.5 \), \( k_d = 0.5 \). B: rate model analog of Fig. 4 E. Mean firing rate elicited by the preferred stimulus (green), the nonpreferred stimulus (red), and both stimuli together (black) as a function of the strength of inhibition \( b \).]
It is widely thought that visual stimuli actively compete for neuronal representation in inferotemporal cortex and other areas of the visual system (Desimone and Duncan 1995; Steinberg and Logothetis 1990; and Rollenhagen and Olson, 2005). However, competition has been envisioned as having three different functional consequences. 1) In the biased competition framework (Desimone and Duncan 1995), competition takes the form of normalization: two simultaneously presented images elicit levels of activation intermediate between those elicited by the images in isolation (Carandini and Heeger 1994; Reynolds et al. 1999). 2) It has been suggested that competition could help to resolve ambiguous displays through a winner-take-all mechanism (Douglas and Martin 2004; Usher and McClelland 2001). In this scenario, when complicated images are presented, the representations of the various components compete with the outcome that the unit representing one component remains active and suppresses the others. 3) Oscillatory neural activity could arise from the combination of a classic winner-take-all mechanism with an adaptive process. This has been proposed as a mechanism for the alternating binocular rivalry that occurs when incompatible images are presented to the two eyes (Blake 1989; Blake and Logothetis 2002; Grossberg 1994; Laing and Chow 2002). Oscillatory activity has been observed over a wide range of frequencies for binocular rivalry and ambiguous stimuli (Carter and Pettigrew 2003; Logothetis et al. 1996) but generally on the order of a few hertz or less. However, oscillations with frequencies of approximately 5 Hz have also been observed in the visual responses of inferotemporal neurons (Nakamura et al. 1991; Steinberg and Logothetis 1990; and Rollenhagen and Olson, 2005).

Here, we argue that the varied responses observed when disparate images are presented in various configurations could arise in a simple cortical circuit combining recurrent excitation and opponent inhibition with spike-frequency adaptation and synaptic depression (Carpenter and Grossberg 1983; Douglas and Martin 2004; Laing and Chow 2002; Raizada and Grossberg 2003; Wang et al. 2003). In such a circuit, neurons selective for a specific image mutually excite each other and inhibit pools of neurons selective for other images. We have shown through simulations and analysis that, as the effective strength of inhibition between pools is steadily increased, there is a transition from normalization to oscillatory behavior and, finally, to winner-take-all behavior. Which phenomenon is observed in a given area and under a given stimulus regime will depend critically on the intensity of inhibitory interactions as determined both by the strength of inhibitory connections and the degree to which the display activates inhibitory interneurons. These conclusions are in good agreement with the observation that two comparable kinds of rivalry can be observed in a network consisting of two layers of reciprocally inhibitory units, with the particular form dependent on the effective strength of inhibition (Wilson 2003). A bifurcation diagram of this model reveals three regimes: with weak inhibition, simultaneous activity of units at both levels; with moderate inhibition, oscillatory behavior; and, with strong inhibition, winner-take-all behavior.

**Comparison between recurrent and feedforward models of normalization**

It has been noted by Reynolds et al. (1999) that normalizing behavior (the tendency for a neuron to fire at an intermediate rate when presented with a preferred and a nonpreferred image) is intrinsic to a model based on feedforward shunting inhibition first proposed by Grossberg and colleagues (Grossberg 1973; Grossberg and Levine 1975) as a means for maximizing the dynamic range of a neuron. Our model differs in that normalization arises from mutual inhibition between two populations of neurons. It can account for phenomena that the model based on feedforward shunting inhibition cannot easily account for, notably oscillatory and winner-take-all behavior. However, with respect to normalization, there are many commonalities between the two models.

To exhibit normalization, we find that a network should obey a set of general conditions. Consider a given neuron (or a pool of neurons) that receives input from two stimuli 1 and 2 and is highly responsive to stimulus 1 but weakly responsive to stimulus 2. We separate the inputs into excitatory and inhibitory components. Thus for stimulus 1 alone, the neuron receives inputs $E_1$ and $I_1$ and fires at rate $R_1$, for stimulus 2 alone the neuron receives inputs $E_2$ and $I_2$ and fires with rate $R_2$, and for the stimuli presented together the inputs are $E_3 > E_1, E_2$ and $I_3 > I_2, I_1$ and the rate is $R_3$. The inputs represent the total input arising from both feedforward and feedback sources (in the case of no excitatory feedback, $E_3 = E_1 + E_2$). We define normalization as the case where $R_3 < R_1 < R_1$. [We do not require perfect normalization or averaging, which would have $R_3 = (R_1 + R_3)/2$].

Normalization will occur in any network provided the response of the neuron to excitatory and inhibitory inputs satisfies the following conditions (Moldakarimov and Chow, unpublished observations): 1) the firing rate increases with excitation and decreases with inhibition, 2) the firing rate is
minimal for zero excitatory input (larger or the same for other input combinations), and 3) if $E_2 > E_1$ then $I_2 > I_1$. Although these conditions are sufficient to ensure normalization they are definitely not necessary. However, they can be satisfied by biophysically plausible networks.

The first condition is satisfied by almost all neuron models. The second condition is not as straightforward. In our simulations, when no excitation is present, the neurons are essentially shut off and fire only randomly because of noise. Inhibition does not decrease the firing rate any further. Here, the firing rate of the pool presented with the nonpreferred image is similar to that if no image is presented. The third condition can be satisfied by adjusting the synaptic weights of the inputs to the neuron. It implies that when a neuron receives strong excitation it is accompanied by weak inhibition and vice versa.

The three conditions are satisfied by the feedforward networks of Grossberg (1973), Grossberg and Levine (1975), and Reynolds et al. (1999), which have a firing rate of the form $R \propto E/(E + I + C)$, where $C$ is a constant. As we can see, the firing rate increases with excitation and decreases with inhibition. Second, the firing rate without excitation is a unique minimum. The third condition in the feedforward network can be relaxed to $E_2 I_2 > E_1 I_1$.

In our recurrent model, the first two conditions are satisfied automatically by our conductance-based model and by the choice of gain function in our rate model. The third condition cannot be externally imposed as in a feedforward model because the net excitatory input and net inhibitory input are not independent. However, it can be satisfied for weak reciprocal inhibition, which agrees with our previous conclusions about normalization. The recurrent network could be considered to be a generalization of the feedforward network. The basic principles of normalization are the same.

Future directions

Although the model that we have described accounts in a broad way for oscillatory phenomena observed in IT by Rollenhagen and Olson (unpublished observations), it is worthwhile to point out that there are subtleties of oscillatory activity in IT for which it does not provide a ready explanation. One example is that the slight but consistent difference in frequency of contrast. Another potential discrepancy between the behavior of the model and the behavior of neurons in IT concerns the results of turning on the two stimuli simultaneously. In our model, simultaneous onset elicits oscillatory activity. However, in several physiological studies, the simultaneous onset of two stimuli has not elicited obvious oscillatory activity, if the absence of any mention of oscillations is to be taken as evidence (Miller et al. 1993; Missal et al. 1999). In the model, there was generally a delay (around 100 ms for the oscillatory mode and 400 ms for the winner-take-all mode) before the network settled into its final state. It is possible that the failure to observe oscillatory activity in the physiological experiments was a result of not leaving the stimuli on long enough. It is also possible, however, that the discrepancy signals the presence in IT of features not captured in the model.

Electrophysiological experiments conducted under independent conditions in different laboratories have demonstrated that interstimulus competition can give rise to both normalizing responses (Heeger et al. 1990; Reynolds et al. 1999) and oscillatory responses (Rollenhagen and Olson, unpublished observations). We have argued that both phenomena could arise from a single neural mechanism, with the phenomenon observed in a given area under a given set of circumstances dependent on the intensity of inhibitory interactions. However, to demonstrate that this is the case will require further experiments involving systematic manipulation of the strength of inhibition between pools of neurons responsive to different stimuli. A direct approach might be the use of GABA agonists or antagonists. An indirect approach would be to control stimulus strength through systematic distortion or manipulation of contrast.

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