SI text

Neuronal Correlates of Perceptual Detection

In the experiments of de Lafuente and Romo (2005), the stimuli were sinusoidal with a fixed frequency of 20 Hz, and their amplitude varied from trial to trial. Stimulus-present trials where intervaled with an equal number of stimulus-absent trials. Neural recordings were obtained with an array of seven independent movable electrodes inserted into S1 and MPC.

The trials were grouped depending on the animals' perceptual report: hits and misses in the stimulus-present condition and correct rejections and false alarms in the stimulus-absent conditions. Using this classification, they found experimentally that the proportion of "yes" responses increases as a function of stimulus amplitude (fig. 3f from de Lafuente and Romo (2005)). But the main findings were observed in the neural recordings: the activity of MPC neurons was only weakly modulated by the stimulus amplitude, and it covaried with the monkeys' trial-by-trial reports. On the contrary, S1 neurons did not covary with the animals' perceptual reports, but their firing rate did show a monotonically increasing graded dependence with the stimulus amplitude. This is reflected in the Figure 3c of de Lafuente and Romo (2005), where the averaged firing rates of 59 S1 neurons and 50 MPC neurons over hit trials are plotted. The results suggest that S1 is primarily engaged with the encoding of the sensory information, whereas the MPC is directly involved with the generation of a percept. In particular, MPC neurons seem to reflect the core of the processing that links the encoding of the sensory information with the generation of a percept and therefore, the perceptual decision-making process itself. The fact that MPC neurons correlate with the behavioral

performance, but show an all-or-none firing rate response, suggests an underlying bistable dynamic in an attractor framework. The detailed investigation of these mechanisms are fundamental for understanding the computational principles involved in perceptual detection, hidden under the very specific type of responses of their neural correlates.

Network

The network contains $N_{\mathrm{E}} = 800$ (excitatory) pyramidal cells and $N_{\mathrm{I}} = 200$ inhibitory inter-neurons, consistent with the neurophysiologically observed proportion of 80% pyramidal cells versus 20% inter-neurons (Abeles (1991),Rolls and Deco (2002)). Each specific population of excitatory cells contains rN_{E} neurons (in our simulations $r = 0.1$). Neurons in the networks are connected via three types of receptors that mediate the synaptic currents flowing into them: AMPA, NMDA glutamate, and GABA receptors. The excitatory recurrent post-synaptic currents (EPSCs) are considered to be mediated by AMPA (fast) and NMDA (slow) receptors. The external background activity imposed onto the network from outside is assumed to be driven only by AMPA receptors. Inhibitory post-synaptic currents (IPSCs) to both excitatory and inhibitory neurons are mediated by GABA receptors.

We assume that the connections are already formed, e.g. by earlier Hebbian learning; the coupling will be strong if the pair of neurons have correlated activity and weak if they are activated in an uncorrelated way. It is reflected in the network in the connections between cells. Neurons within a specific excitatory population are mutually coupled with a strong weight ω_+, whereas neurons between two different selective populations have anti-correlated activity that results in weaker than average connections ω_- between them, always smaller than the strong connections weigths. The neurons in the inhibitory population are mutually connected with an intermediate weight $\omega = 1$. They are also connected with all excitatory neurons with the same intermediate weight, which for excitatory-to-inhibitory connections

is $\omega = 1$, and for inhibitory-to-excitatory connections is denoted by a weight ω_{I}. Neurons in a specific excitatory population are connected to neurons in the nonselective population with a feed-forward synaptic weight $\omega = 1$ and a feedback synaptic connection of weight ω_{NS}. All the weights have to be computed in a way that the overall recurrent excitatory synaptic drive in the spontaneous state remains constant (Brunel and Wang (2001)). In the NCYNN, we accomplish this restriction by varying the ω_- in relation to the ω_+ according to this formula $\omega_- = 1 - r(w_+ - 1)/(1 - r)$. In the CNYN, we fix the values of the ω_+ (that connects the "no" population with the "yes" population), the $\omega_{+'}$ (that connects the "yes" population with the "no" population), and the ω_-. In this case, we carry out the restriction by varying the connections between the non-selective and the selective pools. We, then, distinguish between the weigth that connects the non-selective population with the "yes" population ($\omega_{nsYes} = (r(\omega_+ - \omega_-))/0.8$) and the one that connects the non-selective with the "no" population ($\omega_{nsNo} = (r(\omega'_+ - \omega_-))/0.8$), where 0.8 corresponds to the proportion of pyramidal cells in the complete model.

In this model network, all neurons receive spontaneous background activity from outside the module through $N_{ext} = 800$ external excitatory connections carrying each a Poisson spike train at a spontaneous rate of 3 Hz, which is a typical value observed in the cerebral cortex. In addition, neurons in selective populations receive external inputs encoding stimulus specific information. They are assumed to originate from the somatosensory area, encoding the frequency and amplitude strength of the external applied vibrotactile stimulus. As shown in de Lafuente and Romo (2005), neurons in the somatosensory cortex are selective to a specific vibrotactile frequency and their firing rates increase in a covariate manner with the amplitude of the stimulation. Thus, when the stimulus is presented, the rate of the Poisson train to the MPC neurons of the selective population sensitive to the specific applied vibrotactile

frequency is increased by an extra value of $\lambda = A$, being A the amplitude of the vibrotactile stimulation.

Spiking Dynamics

We used the mathematical formulation of integrate-and-fire (IF) neurons and synaptic currents described in Brunel and Wang (2001). Here we provide a brief summary of this framework, which we have extended to multiple interacting networks. The dynamics of the sub-threshold membrane potential V of a neuron are given by the equation:

$$C_{\mathrm{m}}\frac{dV(t)}{dt} = -g_{\mathrm{m}}(V(t) - V_{\mathrm{L}}) - I_{\mathrm{syn}}(t),$$

where C_{m} is the membrane capacitance taken to be 0.5 nF for excitatory neurons and 0.2 nF for inhibitory neurons; g_{m} is the membrane leak conductance taken to be 25 nS for excitatory neurons and 20 nS for inhibitory neurons; V_{L} is the resting potential of -70 mV, and I_{syn} is the synaptic current. The firing threshold is taken to be $V_{\mathrm{thr}} = -50$ mV, and the reset potential $V_{\mathrm{reset}} = -55$ mV (ref. D. McCormick and Prince (1985)).

The synaptic current is given by a sum of glutamatergic, AMPA ($I_{\mathrm{AMPA,rec}}$), and NMDA ($I_{\mathrm{NMDA,rec}}$) mediated, recurrent excitatory currents, one AMPA ($I_{\mathrm{AMPA,ext}}$) mediated external excitatory current, and one inhibitory GABAergic current (I_{GABA}):

$$I_{\mathrm{syn}}(t) = I_{\mathrm{AMPA,ext}}(t) + I_{\mathrm{AMPA,rec}}(t) + I_{\mathrm{NMDA,rec}}(t) + I_{\mathrm{GABA}}(t).$$

The currents are defined by:

$$I_{\mathrm{AMPA,ext}}(t) = g_{\mathrm{AMPA,ext}}(V(t) - V_{\mathrm{E}}) \sum_{j=1}^{N_{\mathrm{ext}}} s_j^{\mathrm{AMPA,ext}}(t)$$

$$I_{\mathrm{AMPA,rec}}(t) = g_{\mathrm{AMPA,rec}}(V(t) - V_{\mathrm{E}}) \sum_{j=1}^{N_{\mathrm{E}}} w_j s_j^{\mathrm{AMPA,rec}}(t)$$

$$I_{\mathrm{NMDA,rec}}(t) = \frac{g_{\mathrm{NMDA}}(V(t) - V_{\mathrm{E}})}{1 + [\mathrm{Mg}^{++}] exp(-0.062V(t))/3.57} \times \sum_{j=1}^{N_{\mathrm{E}}} w_j s_j^{\mathrm{NMDA}}(t)$$

$$I_{\mathrm{GABA}}(t) = g_{\mathrm{GABA}}(V(t) - V_{\mathrm{I}}) \sum_{j=1}^{N_{\mathrm{I}}} s_j^{\mathrm{GABA}}(t)$$

where $V_{\mathrm{E}} = 0$ mV, $V_{\mathrm{I}} = -70$ mV, ω_j are the synaptic weights, each receptor has its own fraction s_j of open channels and its own synaptic conductance g. The NMDA synaptic current depends on the potential and controlled by the extracellular concentration of magnesium ($[\mathrm{Mg}^{++}] = 1$ mM) (Jahr and Stevens (1990)). The values for the synaptic conductances for excitatory neurons are $g_{\mathrm{AMPA,ext}} = 2.08$ nS, $g_{\mathrm{AMPA,rec}} = 0.104$ nS, $g_{\mathrm{NMDA}} = 0.327$ nS and $g_{\mathrm{GABA}} = 1.287$ nS; and for inhibitory neurons $g_{\mathrm{AMPA,ext}} = 1.62$ nS, $g_{\mathrm{AMPA,rec}} = 0.081$ nS, $g_{\mathrm{NMDA}} = 0.258$ nS and $g_{\mathrm{GABA}} = 1.002$ nS. These values are obtained from the ones used in Brunel and Wang (2001) by multiplication by a factor which corrects for the difference in the number of neurons used in our model and Brunel and Wang's model. In their work, the conductances were calculated so that in an unstructured network the excitatory neurons have a spontaneous spiking rate of 3 Hz and the inhibitory neurons a spontaneous rate of 9 Hz.

The fractions of open channels are described by:

$$\frac{ds_j^{\text{AMPA,ext}}(t)}{dt} = -\frac{s_j^{\text{AMPA,ext}}(t)}{\tau_{\text{AMPA}}} + \sum_k \delta(t - t_j^k)$$
$$\frac{ds_j^{\text{AMPA,rec}}(t)}{dt} = -\frac{s_j^{\text{AMPA,rec}}(t)}{\tau_{\text{AMPA}}} + \sum_k \delta(t - t_j^k)$$
$$\frac{ds_j^{\text{NMDA}}(t)}{dt} = -\frac{s_j^{\text{NMDA}}(t)}{\tau_{\text{NMDA,decay}}} + \alpha x_j(t)(1 - s_j^{\text{NMDA}}(t))$$
$$\frac{dx_j(t)}{dt} = -\frac{x_j(t)}{\tau_{\text{NMDA,rise}}} + \sum_k \delta(t - t_j^k)$$
$$\frac{ds_j^{\text{GABA}}(t)}{dt} = -\frac{s_j^{\text{GABA}}(t)}{\tau_{\text{GABA}}} + \sum_k \delta(t - t_j^k),$$

where the rise time constant for NMDA synapses is $\tau_{\text{NMDA,rise}} = 2$ ms (G. Spruston and Sakmann (1995)S. Hestrin and Nicoll (1990)), the rise time constants for AMPA and GABA are neglected because they are smaller than 1 ms, and $\alpha = 0.5$ ms^{-1}. All synapses have a delay of 0.5 ms. The decay time constant for the AMPA synapses is $\tau_{\text{AMPA}} = 2$ ms (G. Spruston and Sakmann (1995),S. Hestrin and Nicoll (1990)), for NMDA synapses is $\tau_{\text{NMDA,decay}} = 100$ ms (G. Spruston and Sakmann (1995)S. Hestrin and Nicoll (1990)), and for GABA synapses $\tau_{\text{GABA}} = 10$ ms (Salin and Prince (1996),Z. Xiang and Prince (1998)). The sums over k represent a sum over spikes formulated as δ-Peaks ($\delta(t)$) emitted by pre-synaptic neuron j at time t_j^k.

In our simulations, we performed 200 trials for values of λ between 0 Hz and 100 Hz in steps of 10 Hz. The constant input to the "no" population is of 50 Hz. Each trial was structured in three different periods: a pre-stimulus period of 200 ms during which there was no stimulus applied, a stimulus period of 500 ms during in which we applied the stimulus, and a post-stimulus period of 1,000 ms during which we took off the stimulus. For the analysis of the data, we adopted the same criteria used in Lafuente and Romo. For each trial we used a 500-ms window begining at the highest firing rate. According to the experimental data, this

criterion was chosen to maximize the number of correct responses. In all simulations, we used the following connectivity parameters (resulting from the mean field analysis): $\omega_+ = 2.15$ for the NCYN model and $\omega_+ = 1.99$, $\omega'_+ = 1.9$ for the CYNN model. The probabilistic character of the system results from the stochastic nature of the networks. The source of this stochasticity is due to finite-size effects. Fluctuations due to the finite-size effects are due to the fact that the populations are described by a finite number N of neurons. Therefore, the level of fluctuations, i. e., the number of neurons N, is a free parameter that regulates the level of noise and, consequently, the probabilistic behavior of the system. To keep the analysis as simple as possible, we fixed this parameter.

Mean Field

Due to the nature of the activity shown by the recorded MPC neurons, we are especially interested in the probabilistic type of activation of these all-or-none neurons observed across trials that covary with the animals' reported response. The simulation of this phenomenon with an integrate-and-fire neuron network allows the study of the dynamical probabilistic behavior of the neuronal spiking rates. However, these simulations are computationally expensive, which makes them rather unsuitable for systematic parameter explorations. In order to solve this problem, we simplify the dynamics via the *mean field* approach (Brunel and Wang (2001), Amit and Brunel (1997), B.Renart and Wang (2004)). With this approach, we study the characteristics of the network in the stationary conditions, i.e. for periods after the dynamical transients, and analyze the phase diagrams of the dynamics. In our particular case, we are interested in determining the parameters such that the network operates in a regime of bistability. This bistability corresponds to the two possible behavioral responses: percept detected or not detected. This means that at least for the stationary conditions, two different possible attractors are stable.

The essence of the mean-field approximation is to simplify the integrate-and-fire dynamics by replacing after the diffusion approximation (Tuckwell (1988)), the sums of the synaptic components by the average DC component and a fluctuation term. The stationary dynamics of each population can be described by the *population transfer function*, which provides the average population rate as a function of the average input current. The set of stationary, self-reproducing rates for the different populations in the network can be found by solving a set of coupled self-consistency equations. This enables a posteriori selection of the parameter region which shows in the bifurcation diagram the emergent behavior that we are looking for. After that, with this set of parameters, we perform the full non-stationary simulations using the *true dynamics* only described by the full integrate-and-fire scheme.

The mean-field approximation used in the present work was derived in Brunel and Wang (2001), assuming that the network of integrate-and-fire neurons is in a stationary state. In this formulation the potential of a neuron is calculated as:

$$\tau_x \frac{dV(t)}{dt} = -V(t) + \mu_x + \sigma_x \sqrt{\tau_x}\eta(t),$$

where $V(t)$ is the membrane potential, x labels the populations, τ_x is the effective membrane time constant, μ_x is the mean value the membrane potential would have in the absence of spiking and fluctuations, σ_x measures the magnitude of the fluctuations and η is a Gaussian process with absolute exponentially decaying correlation function with time constant τ_{AMPA}. The quantities μ_x and σ_x^2 are given by:

$$\mu_x = \frac{(T_{ext}\nu_{ext} + T_{AMPA}n_x^{AMPA} + \rho_1 n_x^{NMDA})V_E + \rho_2 n_x^{NMDA}\langle V\rangle + T_I n_x^{GABA} V_I + V_L}{S_x} \quad (1)$$

$$\sigma_x^2 = \frac{g_{AMPA,ext}^2(\langle V\rangle - V_E)^2 N_{ext}\nu_{ext}\tau_{AMPA}^2\tau_x}{g_m^2\tau_m^2} \quad (2)$$

where ν_{ext} Hz is the external incoming spiking rate, ν_I is the spiking rate of the inhibitory population, $\tau_m = C_m/g_m$ with the values for the excitatory or inhibitory neurons depending

of the population considered and the other quantities are given by:

$$
\begin{aligned}
S_x &= 1 + T_{ext}\nu_{ext} + T_{AMPA} n_x^{AMPA} + (\rho_1 + \rho_2) n_x^{NMDA} + T_I n_x^{GABA} && (3)\\
\tau_x &= \frac{C_m}{g_m S_x} && (4)\\
n_x^{AMPA} &= \sum_{j=1}^{p} r_j w_{jx}^{AMPA} \nu_j && (5)\\
n_x^{NMDA} &= \sum_{j=1}^{p} r_j w_{jx}^{NMDA} \psi(\nu_j) && (6)\\
n_x^{GABA} &= \sum_{j=1}^{p} r_j w_{jx}^{GABA} \nu_j && (7)\\
\psi(\nu) &= \frac{\nu\tau_{NMDA}}{1+\nu\tau_{NMDA}} \left(1 + \frac{1}{1+\nu\tau_{NMDA}} \sum_{n=1}^{\infty} \frac{(-\alpha\tau_{NMDA,rise})^n T_n(\nu)}{(n+1)!} \right) && (8)\\
T_n(\nu) &= \sum_{k=0}^{n} (-1)^k \binom{n}{k} \frac{\tau_{NMDA,rise}(1+\nu\tau_{NMDA})}{\tau_{NMDA,rise}(1+\nu\tau_{NMDA}) + k\tau_{NMDA,decay}} && (9)\\
\tau_{NMDA} &= \alpha\tau_{NMDA,rise}\tau_{NMDA,decay} && (10)\\
T_{ext} &= \frac{g_{AMPA,ext}\tau_{AMPA}}{g_m} && (11)\\
T_{AMPA} &= \frac{g_{AMPA,rec} N_E \tau_{AMPA}}{g_m} && (12)\\
\rho_1 &= \frac{g_{NMDA} N_E}{g_m J} && (13)\\
\rho_2 &= \beta \frac{g_{NMDA} N_E (\langle V_x \rangle - V_E)(J-1)}{g_m J^2} && (14)\\
J &= 1 + \gamma \exp(-\beta \langle V_x \rangle) && (15)\\
T_I &= \frac{g_{GABA} N_I \tau_{GABA}}{g_m} && (16)
\end{aligned}
$$

$$
\langle V_x \rangle = \mu_x - (V_{thr} - V_{reset})\nu_x\tau_x, \qquad (17)
$$

where p is the number of excitatory populations, r_x is the fraction of neurons in the excitatory x population, $\omega_{j,x}$ the weight of the connections from population x to population j, ν_x is the spiking rate of the x excitatory population, $\gamma = [Mg^{++}]/3.57$, $\beta = 0.062$, and the average membrane potential $\langle V_x \rangle$ has a value between -55 mV and -50 mV.

The spiking rate of a population as a function of the defined quantities is then given by:

$$\nu_x = \phi(\mu_x, \sigma_x), \tag{18}$$

where

$$\phi(\mu_x, \sigma_x) = \left(\tau_{rp} + \tau_x \int_{\beta(\mu_x,\sigma_x)}^{\alpha(\mu_x,\sigma_x)} du\sqrt{\pi}\exp(u^2)[1+\mathrm{erf}(u)] \right)^{-1} \tag{19}$$

$$\alpha(\mu_x, \sigma_x) = \frac{(V_{thr} - \mu_x)}{\sigma_x}\left(1 + 0.5\frac{\tau_{AMPA}}{\tau_x}\right) + 1.03\sqrt{\frac{\tau_{AMPA}}{\tau_x}} - 0.5\frac{\tau_{AMPA}}{\tau_x} \tag{20}$$

$$\beta(\mu_x, \sigma_x) = \frac{(V_{reset} - \mu_x)}{\sigma_x} \tag{21}$$

with $\mathrm{erf}(u)$ the error function and τ_{rp} the refractory period which is considered to be 2 ms for excitatory neurons and 1 ms for inhibitory neurons. To solve the equations defined by **E**q. 18 for all x's we integrate numerically **E**q. 17 and the differential equation below, which has fixed point solutions corresponding to **E**q. 18:

$$\tau_x \frac{d\nu_x}{dt} = -\nu_x + \phi(\mu_x, \sigma_x). \tag{22}$$

Parameters In this Appendix, we bring together the fixed parameters of the model in Table 2 and then provide information about the values of further parameters used in the simulations and how well the simulations fit the experimental data.

Table 1: Parameters used in the integrate-and-fire simulations for the NCYN-model

N_{E}	800
N_{I}	200
r	0.1
ω_+	2.15
ω_{I}	1.015
N_{ext}	800
ν_{ext}	2.4 kHz
C_{m} (excitatory)	0.5 nF
C_{m} (inhibitory)	0.2 nF
g_{m} (excitatory)	25 nS
g_{m} (inhibitory)	20 nS
V_{L}	-70 mV
V_{thr}	-50 mV
V_{reset}	-55 mV
V_{E}	0 mV
V_{I}	-70 mV
$g_{\mathrm{AMPA,ext}}$ (excitatory)	2.08 nS
$g_{\mathrm{AMPA,rec}}$ (excitatory)	0.104 nS
g_{NMDA} (excitatory)	0.327 nS
g_{GABA} (excitatory)	1.25 nS
$g_{\mathrm{AMPA,ext}}$ (inhibitory)	1.62 nS
$g_{\mathrm{AMPA,rec}}$ (inhibitory)	0.081 nS
g_{NMDA} (inhibitory)	0.258 nS
g_{GABA} (inhibitory)	0.973 nS
$\tau_{\mathrm{NMDA,decay}}$	100 ms
$\tau_{\mathrm{NMDA,rise}}$	2 ms
τ_{AMPA}	2 ms
τ_{GABA}	10 ms
α	0.5 ms^{-1}

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Table 2: Parameters used in the integrate-and-fire simulations for the CYNN-model

N_{E}	800
N_{I}	200
r	0.1
ω_+	1.99
ω'_+	1.9
ω_-	0.85
ω_{I}	1.015
N_{ext}	800
ν_{ext}	2.4 kHz
C_{m} (excitatory)	0.5 nF
C_{m} (inhibitory)	0.2 nF
g_{m} (excitatory)	25 nS
g_{m} (inhibitory)	20 nS
V_{L}	-70 mV
V_{thr}	-50 mV
V_{reset}	-55 mV
V_{E}	0 mV
V_{I}	-70 mV
$g_{\mathrm{AMPA,ext}}$ (excitatory)	2.08 nS
$g_{\mathrm{AMPA,rec}}$ (excitatory)	0.104 nS
g_{NMDA} (excitatory)	0.327 nS
g_{GABA} (excitatory)	1.25 nS
$g_{\mathrm{AMPA,ext}}$ (inhibitory)	1.62 nS
$g_{\mathrm{AMPA,rec}}$ (inhibitory)	0.081 nS
g_{NMDA} (inhibitory)	0.258 nS
g_{GABA} (inhibitory)	0.973 nS
$\tau_{\mathrm{NMDA,decay}}$	100 ms
$\tau_{\mathrm{NMDA,rise}}$	2 ms
τ_{AMPA}	2 ms
τ_{GABA}	10 ms
α	0.5 ms^{-1}