Attention

Top-down attention: Information in working memory selectively enhances neural representations of sensory stimuli [Desimone01].

Top-down
- Goal-directed
- Relevant stimuli

Bottom-up
- Stimulus-driven
- Salient stimuli

Neuronal signatures
- Enhanced sensitivity
- Enhanced selectivity

Network signatures
- Enhanced gamma rhythms
- Enhanced spike synchrony
Macaque brain

Visual areas in the macaque brain.
Visual Cortex (V4): Spatial attention

Recordings from a V4 cell showing enhanced responses in the attended mode (black) relative to the unattended mode (gray). Spike rates are affected multiplicatively [Maunsell1999].
Attention to the neuron's preferred direction of motion increased the neuron's response, but attention to the null direction of motion decreased its response. Thus, attention to a particular direction of motion does not increase responses across all neurons. Rather, it has a push–pull effect that increases responses only for neurons that prefer motion close to the attended direction [Treue06].

Visual cortex (V4): Gamma increases
Simultaneously recorded spikes and local field potential (LFP) with attention directed inside (red) or outside (blue) the cell's receptive field. The LFPs' spike-triggered averages (STA) and the STAs' power spectra were computed [Sejnowski01,Steinmetz00].

Somatosensory cortex (SII): Synchrony increases

Spike trains recorded from a pair of neurons (red and green) in secondary somatosensory cortex with (a) and without (b) attention. Synchronous spikes (within 2.4ms) are indicated (blue) and quantified in the cross-correlation histogram above,
with (red) and without (blue) attention (excess over Poisson) [Sejnowski01,Steinmetz00].

Cholinergic modulation

Acetylcholine enhances the effect of attention (green – black vs red - blue) in V1; bars of various lengths were presented [Thiele08]. It may act by enhancing both excitatory and inhibitory synaptic transmission, as has been shown in vitro.
Controlling synchrony

When inhibition's strength ($\Delta G$) surpasses a critical level, synchrony appears (left, demo; right, $G_{\text{max}}$, $G_{\text{min}}$, and $G$ over a cycle versus $\Delta G$). Rate-based models predict that synchrony appears when the loop-gain exceeds unity (i.e., $m > 1$, where $m$ and $1/s$ are the $A(G)$ and $G(A)$ curves' slopes, respectively. However, these models ignore heterogeneity, which makes synchrony more difficult to achieve.

Kuramoto model addresses heterogeneity

Phases of coupled oscillators with weak (left) and strong (right) coupling. Color and ball-size indicate the oscillators’ different intrinsic frequencies; dashed circle and marker indicate the order parameter’s magnitude and phase (i.e., vector strength).

Read tutorial: http://tutorials.siam.org/dsweb/cotutorial/index.php?s=4&p=0
Download Java applet: http://www.johnwordsworth.com/tutorials/Kuramoto/media/applet/Kuramoto.jnlp

Instead of pulse-coupling, this model uses phase-coupling:

$$\dot{\theta}_i = \omega_i + \frac{K}{N} \sum_{j=1}^{N} \sin[\theta_j - \theta_i], \quad i = 1 \ldots N$$
Instead of vector strength, an order parameter is defined:

\[
\text{re}^{i\psi} = \frac{1}{N} \sum_{j=1}^{N} \text{re}^{i\theta_j}, \quad i = 1 \ldots N
\]

Relating coupling strength \((K)\) to the PRC

The Kuramoto model's sinusoidal phase-coupling corresponds to a PRC that is a flipped sinusoid. To obtain the Kuramoto model's coupling strength, \(K\), we must multiply the PRC's maximum advance/delay, \(\Delta T_{\text{max}}\), by the network's total spike rate.

Consider only the \(j\)th oscillator's effect on the \(i\)th oscillator:

\[
\dot{\theta}_i = \ldots + \frac{K}{N} \sin[\theta_j - \theta_i] + \ldots
\]

If the \(i\)th oscillator's phase is \(\Theta\) when the \(j\)th oscillator's phase is 0 — which, by definition, is when it spikes — then we have \(\theta_i = \Theta + \theta_j\), or \(\theta_j - \theta_i = -\Theta\). This assumes that the phase-difference remains constant throughout that cycle. In which case, the total change in the \(i\)th oscillator's phase over the complete cycle — which, by definition, is the PRC — will be:

\[
\text{PRC}[\Theta] = \int_0^T \dot{\theta}_i \, dt = \int_0^T \frac{K}{N} \sin[\Theta - \theta] \, dt = -\frac{T}{N} \frac{K}{N} \sin[\Theta]
\]

Hence, this model assumes the PRC is a flipped sinusoid. The PRC's maximum delay/advance, \(\Delta T_{\text{max}}\), is related to the coupling strength, \(K\), by

\[
\Delta T_{\text{max}} = \frac{T}{N} \frac{K}{N}
\]

\[
\Longleftrightarrow K = N \frac{\Delta T_{\text{max}}}{T}
\]

Hence, we must multiply the PRC's amplitude — or inhibition's synaptic strength \((\Delta G)\) — by the network's total firing rate to convert it into the Kuramoto model's coupling strength \((K)\).