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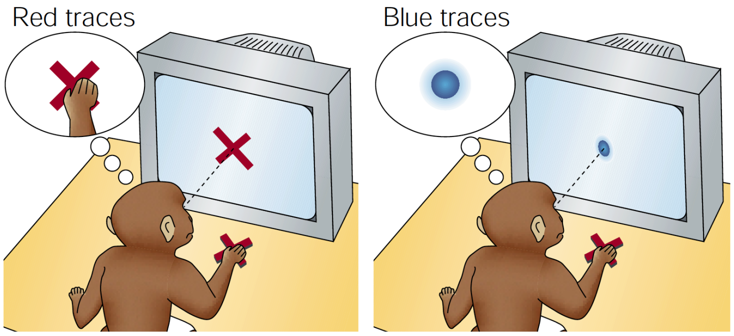
Attention



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 rhythyms
  - Enhanced spike synchrony

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Macaque brain

Visual areas in the macaque brain.

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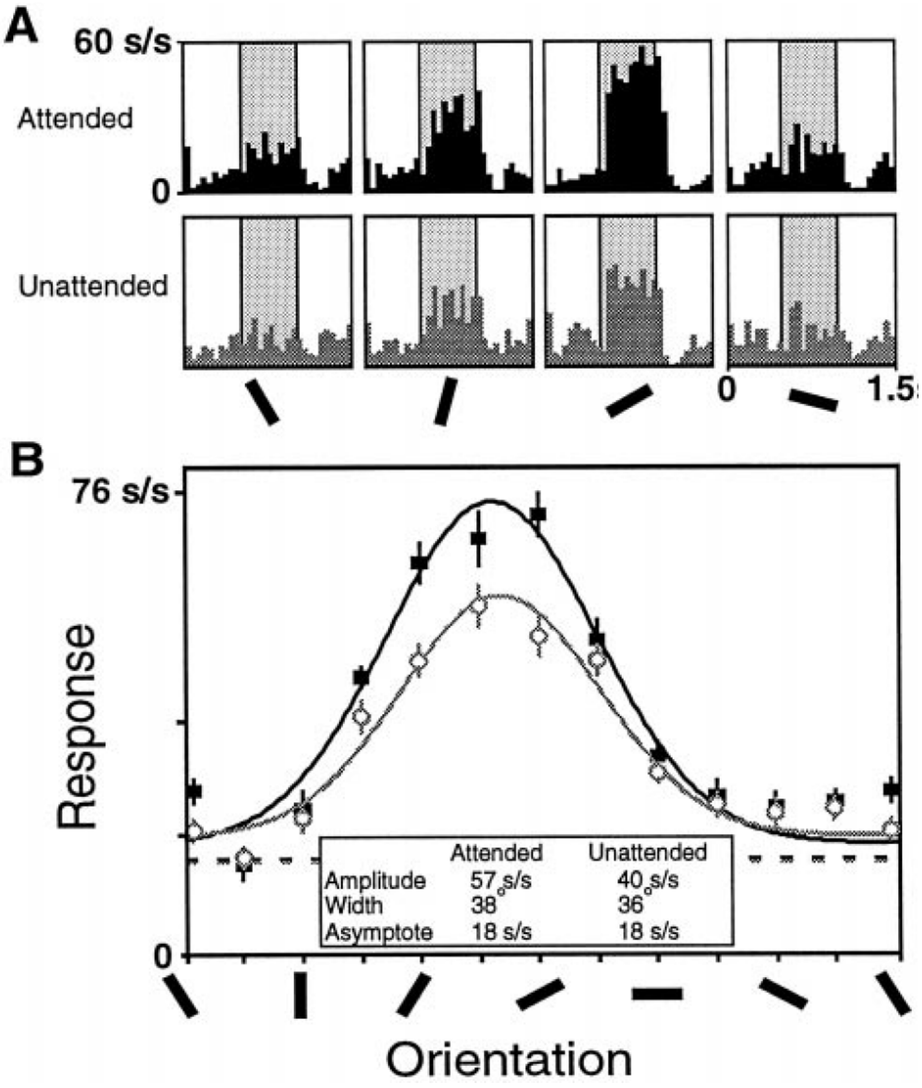
Visual Cortex (V4): Spatial attention



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].

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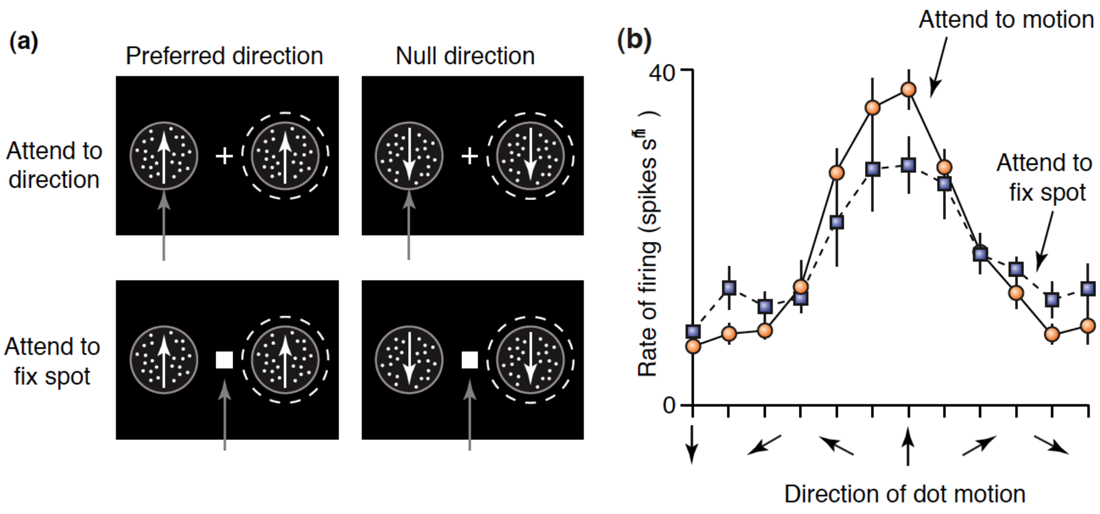
Visual Cortex (MT): Feature attention



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– pulleffect that increases responses only for neurons that prefer motion close to the attended direction [Treue06].

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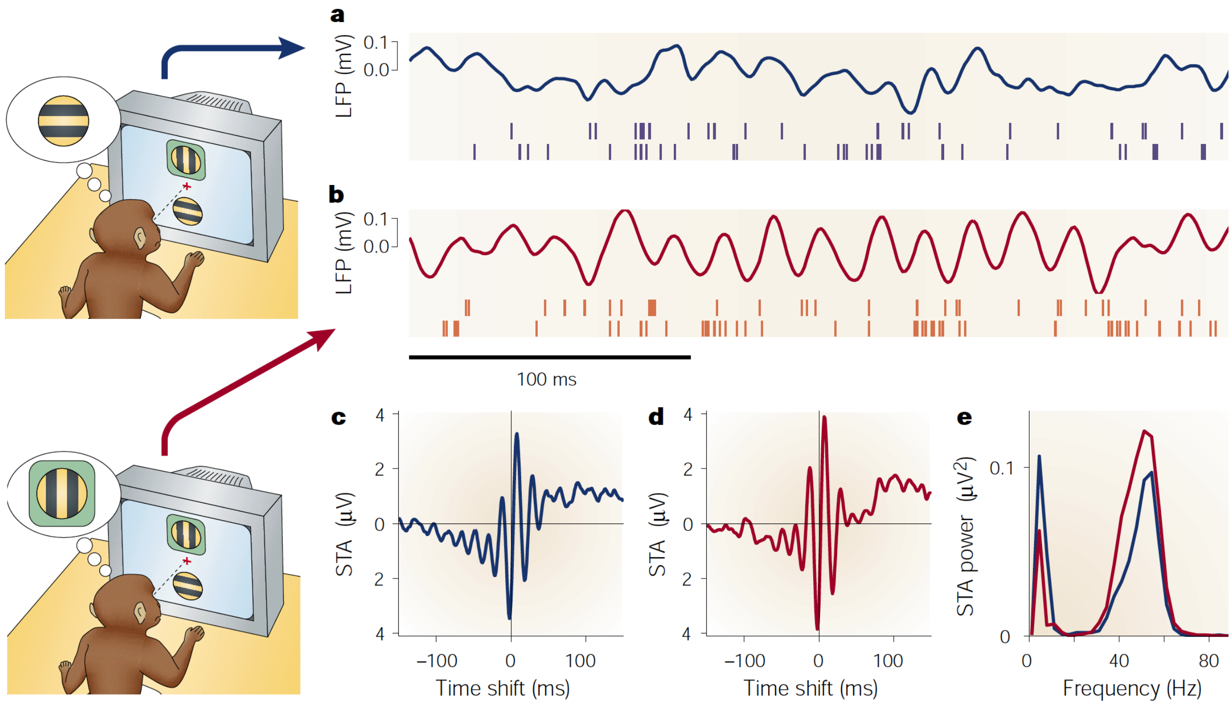
Visual cortex (V4): Gamma increases



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].

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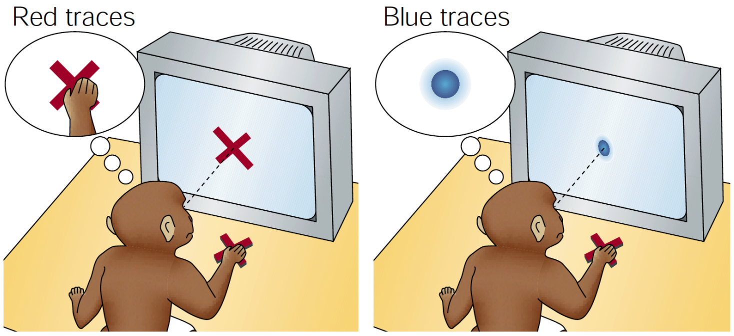
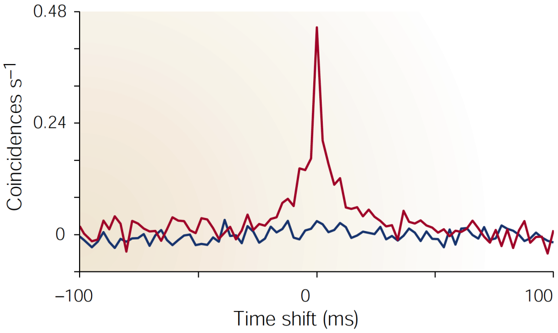
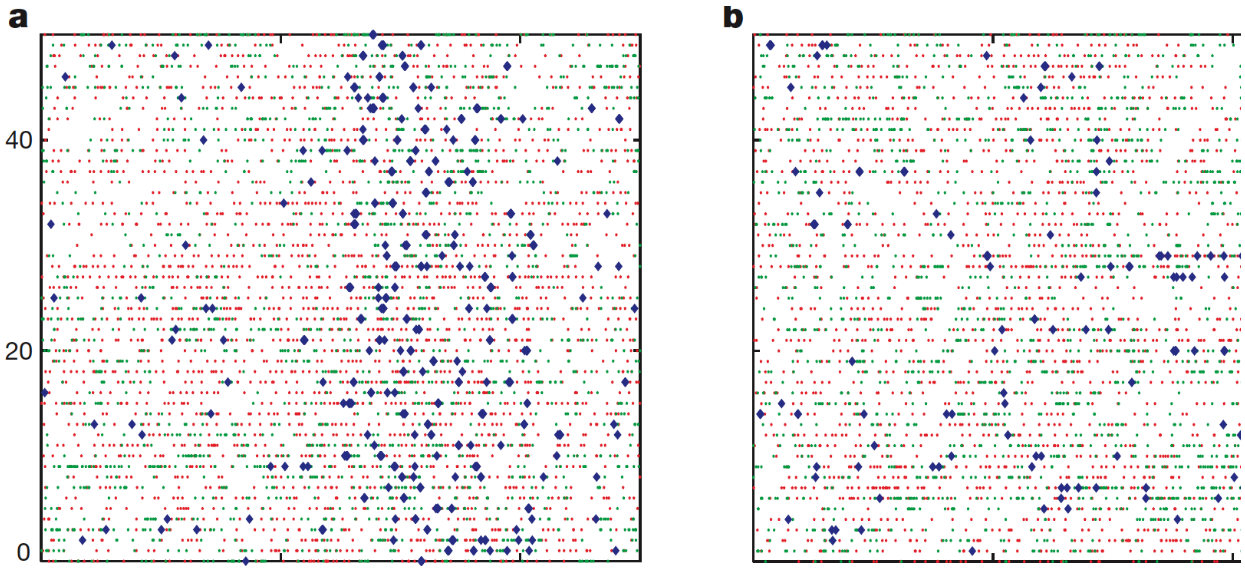
Somatosensory cortex (SII): Synchrony increases







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].

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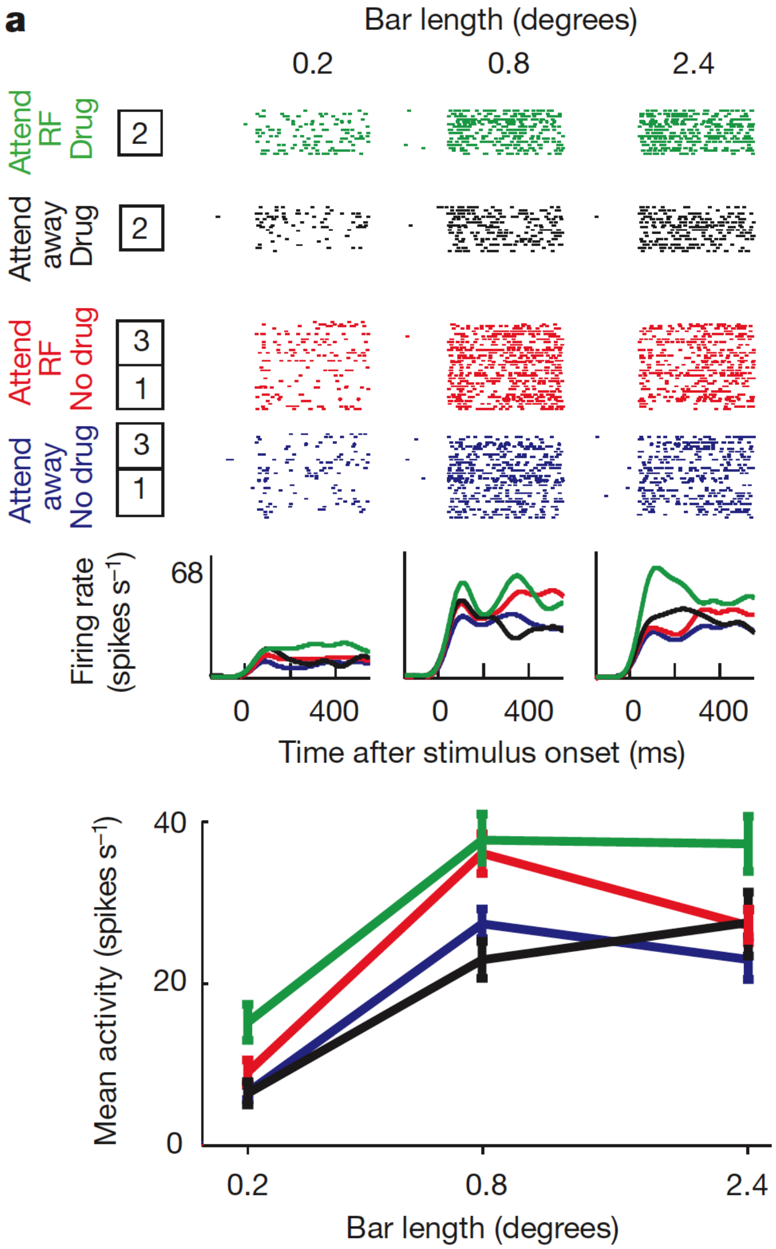
Cholinergic modulation



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 inhibitiory synaptic transmission, as has been shown *in vitro*.

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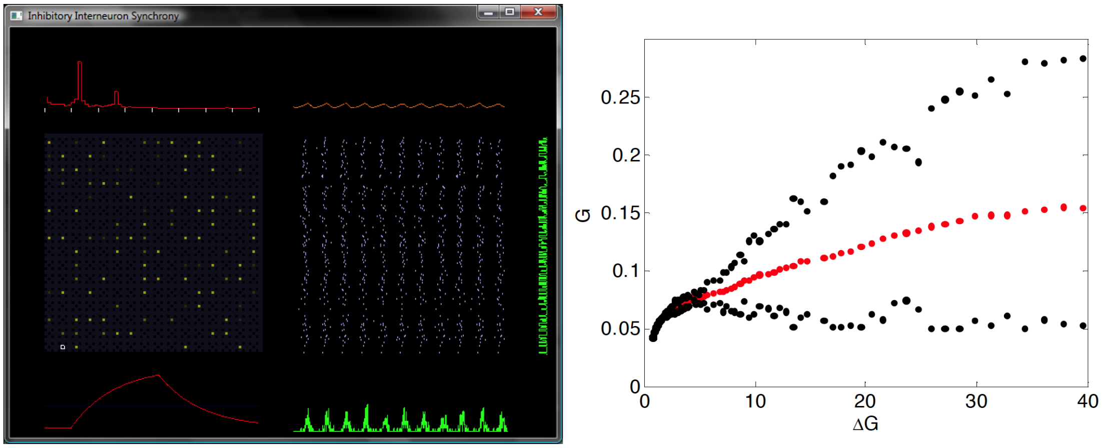
Controlling synchrony



When inhibition's strength (ΔG) surpasses a critical level, synchrony appears (*left,* demo; *right*, $G_{\max}$, $G_{\min}$, and $\langle G \rangle$ over a cycle versus ΔG). Rate-based models predict that synchrony appears when the loop-gain exceeds unity (i.e., $m\,s > 1$, 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.

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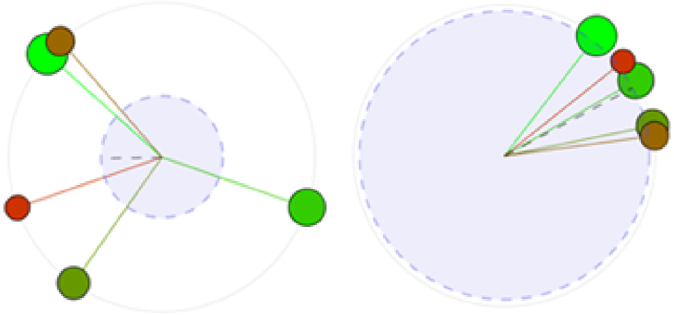
Kuramoto model addresses heterogeneity



Phases of coupled oscillators with weak (left) and strong (right) coupling. Color and ball-size indicate the oscillators' different intrinsic frequences; 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:

$$\dot{\theta}_i = \omega_i + \frac{K}{N}\sum_{j=1}^{N}\sin[\theta_j-\theta_i], \qquad i = 1\ldots N$$

Instead of vector strength, an order parameter is defined:

$$re^{i\psi} = \frac{1}{N}\sum_{j=1}^{N} e^{i\theta_j}, \qquad i = 1...N$$

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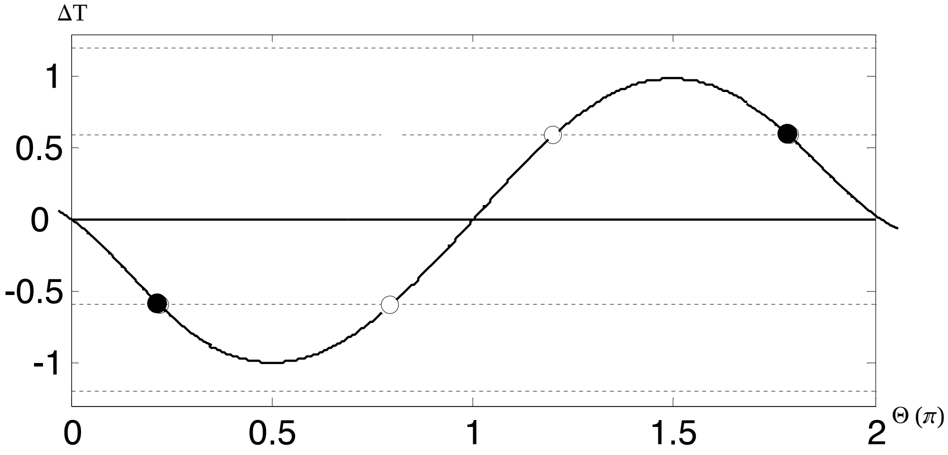
Relating coupling strength (*K*) to the PRC



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, ΔT_{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 Θ 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:

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

Hence, this model assumes the PRC is a flipped sinusoid. The PRC's maximum delay/advance, ΔT_{max}, is related to the coupling strength, *K*, by

$$\Delta T_{max} = T\,K / N$$
$$\Longleftrightarrow \quad K = N\,\Delta T_{max} / T$$

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