Synchronization in Cortical Networks

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abstract *Nature abounds with networks of coupled oscillators which through seemingly complicated interactions give rise to surprisingly comprehensible network-level properties. The tendency of a wide range of oscillator networks to robustly converge to synchrony is one such network-level property. The past few decades has seen a growth of empirical evidence and theoretical inquiry into the relevance of synchrony to various mysteries of brain function. The paper addresses some aspects of the following question: to what extent is synchronized cortical activity (for which evidence will be presented) relevant to the computational processes of the brain? In the introductory section a summary of the hypothesized functional roles of cortical synchronization is provided. Next, idealized mathematical models of synchronization in neuronal networks are discussed and their qualitative characteristics are highlighted. Finally, empirical evidence suggesting functional roles for cortical synchronization are reviewed. Specific attention will be given to the role of γ-band synchronization in visual attention and visual memory. In the concluding section, some questions which appear to the author as tractable open problems are suggested.*

Contents

1 Possible roles for cortical synchronization

> *"You imagine that I look back on my life's work with calm satisfaction, but from nearby it looks quite different. There is not a single concept of which I am convinced it will stand firm, and I feel uncertain whether I am in general on the right track." Albert Einstein*

This section is concerned with the central arguments for and against the assignment of various roles to synchronization in brain function. The central issue, and the crux of many such arguments, is the claim that the firing-rate model of the cortical neuron cannot be the only mode of cortical computation and representation. However, given the poverty of our understanding of the brain's computational mechanisms,

this statement is *a priori* likely to be true but of no direct consequence. The real question then ought to be more specific: what are the computational problems that remain unexplained with a firing-rate model and can be reasonably explained with an assignment of a functional role to synchronous activity? The underlying assumption, of course, is that although the computational requirements and mechanisms of various brain functions may very well be rather diverse, some general principles are most likely valid across subsystems and in the coordination among them. If synchronization can serve as one such general unifying principle, two refining questions must also be addressed. These will be the topic of the following sections: **i)** Is synchronized activity a stable mode of oscillation in realistic cortical networks? **ii)** How can such synchronized activity be measured with reasonable statistical confidence and what is its relationship to subthreshold oscillating field potentials? We will here simply accept the following conclusions from the evidence provided in subsequent sections. **1)** Synchronization is a robust phenomenon in networks of interacting neurons. **2)** There is ample evidence of synchronized activity in the brain.

1.1 Conventional ideas

The conventional framework of analyzing nervous function can be summarized in terms of Barlow's formulation of the *neuron doctrine*:

> *"1. To understand nervous function one has to look at interactions at a cellular level […] 2. The sensory system is organized to achieve as complete a representation of sensory stimuli as possible with the minimum number of active neurons. 3. Trigger features of sensory neurons are matched to redundant patterns of stimulation by experience as well as by developmental processes. 4. Perception corresponds to the activity of a small selection from the very numerous high-level neurons […]. 5. High impulse frequency in such neurons corresponds to high certainty that the trigger feature is present."*[1]

Hebb's notion of *cell assembly* has also been widely accepted as a general computational principle of the brain:

> *"The general idea is an old one, that any two cells or systems of cells that are repeatedly active at the same time will tend to become 'associated,' so that activity in one facilitates activity in the other. The details of speculation that follow are intended to show how this old idea might be put to work again, with the equally old idea of a lowered synaptic 'resistance'."[2]*

Two remarks that relate to the upcoming discussion are in order. First, Hebb's notion of a cell assembly suggests (although not usually acknowledged) that association networks can be identified, by an outside agent, through the synchronous activity of a collection of neurons. In fact, he proposes that "the duration of an idea is the duration of reverberatory activity in a closed system" of the cortex [2] which is closely in line with the current understanding of visual attention discussed later. Second, the suggestions of functional roles for synchrony aim to amend Barlow's 5th principle with an additional mode of neural coding. In fact, roughly speaking, it appears to be the case that firing-rate coding is the primary mode of operation in the periphery of the cortex (e.g. primary visual cortex) and that the temporal fine structure of ensembles become more relevant with higher dimensional representations in the downstream and in intracortical coordination (e.g. the hippocampus in memory or the thalamus in attention).

1.2 The decoding problem

As per Barlow's first 4 principles, it is generally assumed that all information transmitted by a neuron to its postsynaptic downstream is contained in the spike train times $\{t_i\}$. Consider the following maximum-likelihood idealization of the decoding problem in terms of reconstructing the stimulus waveform: given a spike train $\{t_i\}$ our goal is to reconstruct the stimulus waveform $s(\tau)$ such that $\Pr\big(s(\tau)|\{t_i\}\big)$ is maximized to some approximation. Suppose we somehow had access to probabilities $\Pr\big(t_i|s(\tau)\big)$ and the joint distribution of spikes $\Pr\big(\{t_i\}\big)$. Then the stimulus could be, at least in principle, recovered by an application of the Bayes rule.

Firing-rate coding can be viewed as an *independent-spike* assumption which says $\Pr\big(\{t_i\}\big) = \prod_i \Pr(t_i)$ and thus at any given moment the only source of information is the number of spikes fired and the temporal coherence of the incoming signals or lack thereof contains no information. On the contrary, the role of synchrony in neural coding can be formulated in terms of a *correlation code* (often also called a *temporal code*) which meaningfully represents and transmits the temporal fine-structure of the incoming signals. Notice that the firing-rate code is a special case of correlation code in the sense that if a neuron is correlation-coding it is not ignoring the rate information from its synaptic inputs.

A correlation-coding neuron essentially acts as a *coincidence detector* by exhibiting pronounced sensitivity to synchronous arrival of synaptic inputs. That neurons in general can exhibit such fine temporal resolution is established going back to the Jeffress model of sound localization in the brainstem. Whether *cortical* neurons operate under such conditions is thus our question of interest. As we will see later, pyramidal neurons are capable of operating in both modes of operation depending on synaptic depression rates, membrane conductance, and background synaptic activity.

1.3 The binding problem

Much of the promise of invoking synchronization to explain brain function is that it imposes temporal structure over a large spatial range in cortical circuits. By this we mean that, as opposed to the temporal structure of firing-rate encoder/decoder neurons which remains limited to their dendritic tree, a network of synchronized neurons is able to keep a global time (corresponding loosely to a clock in electronic circuits). Much of the early work is due to Von der Malsburg [3]–[5], whose "correlation theory" provided a possible answer to the so-called binding problem. There are more than one versions of "the binding problem" (see below); the version of the problem that Von der Malsburg addresses can be described in the context of visual recognition [6], [7]: suppose you are presented with a red square and a blue circle. For successful recognition, the piece of neural information corresponding to the perception of "red" at some position in the visual field should somehow be "bound" to the piece of information corresponding to the perception of "square" in the same place in the visual field. Otherwise, we cannot explain why the input would not appear as the wrong conjunction (or a "catastrophic superposition" [5]), e.g. a red circle. Von der Malsburg argued, as others have had, that the resolution of this problem in the conventional framework

would inevitably lead to unattainable "cardinal" cells [1]. The inability of the conventional framework to solve the binding problem is representative of its lack of a mechanism to represent relations among neural symbols. The correlation theory claims that this relation is carried in the synchronized firing of different symbols; in this case, the synchronized firing of the corresponding neural representations of "red" and "square" binds the two together. Notice that no new neurons are required to represent the conjunction of the symbols but the more complex symbol requires more complex spatiotemporal representation. Efficient processing requires that such "dynamic links" be created and discarded in real time, on a timescale much faster than Hebbian plasticity. In summary, the consequences of the correlation theory on mechanisms of neural coding are:

1. Synaptic plasticity must occur at multiple timescales with at least one component corresponding to Hebbian plasticity and a much faster subsecond component modulating the synaptic gain while dynamic links are being created and destroyed.
2. Rapid modulation of synaptic gain increases or decreases PSP magnitudes considerably.
3. Meaningful coincidences are of high order (more than a handful simultaneous inputs) and must be detected with fine temporal resolution of roughly 5-10ms.

All these predictions have since been more or less confirmed experimentally [9], [10], [11], [12]; we will return to the biophysical mechanisms in a later section.

1.3.0.1 Other binding problems

Beyond simple conjunction search tasks (e.g. "find the red square"), we can distinguish multiple problems that could potentially be considered as "the binding problem". One problem is *segmentation* that is the decomposition of high dimensional input stimuli to localized perceptions bound to regions of the visual field (e.g. identifying corners). This problem has two related components. One is to find the salient segments, and the other to represent and transmit their neural code. It is conceivable that the necessary computation for segmentation between saccades can be entirely performed in the visual system alone [2], However the conventional framework cannot explain the temporal stability of the neural representation of a segment given the instability of the response of classic receptive field neurons. Von der Malsburg's correlation theory explains the necessary stability of the neural representation of salient segments by invoking a combination of synchronization within segments and desynchronization between segments [4].

The other problem is that of *object recognition* which involves combining the salient features discovered in segmentation into boundaries of continuous objects. This translation is by no means trivial and the required computations are almost certain to involve multiple brain regions as the resolution of ambiguities involves incorporating additional acquired knowledge about what is and what is not likely to be a contour

[1] Some have disputed this claiming that there is no combinatorial problem [8] but their argument seems flawed to the author in that their resolution of the combinatorial explosion assumes the computed binding is somehow readily available.

[2] This is where yet another binding problem, known as the "combination" binding problem, has been raised. The problem presumes that the perception of the analyses done over various local regions of the visual field (e.g. over multiple saccades) should somehow be combined into a global unified perception. Although, it is not clear whether such a combination in fact happens or in any way is needed for any of the known computational processes of the brain. Some have argued that the apparent "richness" of perception (demanded by the combination problem to arise from the combination of all localized perceptions) is an illusion [13].

of a single object in the real world [14]. The correlation theory provides no resolution to this problem (although limited efforts have been made [15]).

1.3.0.2 High variability and coincidence detection

As a criticism to the correlation theory, it can been argued that cortical neurons are inherently unreliable (or that the cortical environment is too noisy) and thus incapable of high frequency processing [14]–[17]. First, noncortical excitable cells do not seem unreliable in any way and it is, therefore, plausible that cortical neurons are not intrinsically more erratic. For instance, much is known about the fine precision of brainstem neurons in auditory and visual processing. Second, the argument that highly variable neurons are not capable of high frequency information processing is merely a reflection of the *assumption* that individual neurons are computationally "simple" (which is a fair simplifying assumption given the complexities). If cortical neurons do not have an innate tendency to fire erratically, which seems to be the case, then their high variability *in vivo* probably reflects the fact that they *are*, in fact, involved in high frequency processing [17], [18]. Third, the observed variability of individual neurons *in vivo* does not, by itself, mean they are unreliable since much of the variability is the due to our idealization of complexity as randomness (that is, neurons are random processes much in the same way that molecules in a fluid move "randomly"). Specifically, it has been shown that under controlled recording settings where the stimulus is controlled and retinal, thalamic, and cortical neurons are simultaneously monitored, the apparent variability is much less than the common Poisson process idealization and that variation increases as neural signals propagate to downstream areas [19].

1.4 Detecting cortical synchronization

There are several issues concerning any experimental effort to verify synchronization. First, it is likely that an arbitrary collection of single unit readings are merely time-locked to a common input (possibly the external stimulus) and are not synchronized due to any interactions among themselves. Second, one needs a way of assigning some numerical measure of synchrony to an epoch of multiunit readings. For any such choice, an appropriate statistical measure is needed to reject the possibility of apparent regularities by chance. Third, much of the considerations developed for the first two questions are inapplicable to field potential recordings (LFP, EEG, MEG, ECoG) where individual units are not identifiable from the field reading. In this section we address these difficulties and review the available techniques.

1.4.0.1 Unit potentials

We provide an idealized stochastic model of the neuron, as per [20], which provides useful statistical approximations. The fluctuations in the membrane potential of a cortical neuron can be captured as follows: synaptic inputs arrive at the dendritic tree at times distributed according to a Poisson process. Each such PSP travels down the soma in a passive way. Whenever an action potential is fired, there may be leftover PSP in the dendritic branches which have not yet arrived at the soma. The delayed conduction of these potentials to the soma leads to membrane potential fluctuations at the soma. If one idealizes away the action

potentials, the central limit theorem implies that the membrane potential is a stochastic process which is at any point in time normally distributed (Fig. 6). One can then read off the probability of an action potential at any moment of time, i.e. the instantaneous firing rate (up to normalization), from the corresponding Gaussian tail (Fig. 6 B). If the membrane threshold is T and at some time the membrane potential has standard deviation σ then the instantaneous firing rate is:

$$K\frac{1}{\sqrt{2\pi}}\int_{T/\sigma}^{\infty} e^{-v^2}dv$$

where K is a scaling constant dependent on the variability of the fluctuations and on the refractory period of the cell. As the formula indicates, T/σ is a measure of the *excitability* of the cell. Although this method provides useful approximations, its applicability decreases at high firing rates since the ignored effect of the refractory period gets pronounced.

1.4.0.2 Establishing synchronization

Given two spike trains $\{t_i\}$ and $\{t'_i\}$, we can find the *cross-renewal density* (also called cross-intensity function) for $t \to t'$ which is a function $f(\tau)$ which gives the the conditional probabilty of t' firing at a time τ after t fires (Fig. 7) based on the spike train data. This function is closely related to the cross-correlation function (i.e they can be viewed as approximations of each other) and can be used to investigate synchronization relationships between the two spike trains. A cross-renewal density (or cross-correlation) peak at time lag 0 could imply synchronization. The standard technique to ensure that the two units are in fact in the same circuit and not merely independently time-locked to the stimulus is to *shuffle* the spike trains such that stimulus-locked variations are averaged out (Fig. 7 C,D). Then the difference between the original and shuffled cross-renewal densities is comparable to the no-stimulus cross-renewal density and any difference between the two must be caused by the stimulus but not independently time-locked to it.

There are a few difficulties with these statistical methods:

1. The assumption that a cross-correlation peak at 0 implies synchrony is not statistically justified but in common use [21]. The difficulty lies in finding other sources of statistical dependence between the two spike trains. Trial-to-trial variability has been studied and identified as a source of additional (undesired) correlation [22]. Quantitative methods have been developed to reduce such effects [23], [24].
2. When analyzing large populations of neurons, we need to attach a significance value to the observed correlation peak. This requires either formulating a null hypothesis and developing a probabilistic model for arbitrary unsynchronized channels to have a strong peak at 0 at a given time [24]. Alternatively, a Bayesian analysis can be performed to calculate a "synchronization likelihood" for a channel to be synchronized to all other channels at a given time [25].
3. All this is inapplicable when dealing macroscopic field potentials. Furthermore, in field potential recordings there is typically an interest in localizing the frequency range of any synchronous activity. What is usually done is to take the signal to the frequency domain (depending on the context, via Fourier or wavelet transforms) where the cross-correlation density of two signals x and y becomes

the *cross-spectral density* $G_{x,y}(f)$. Then the *coherence* at frequency f is defined as:

$$C_{x,y}(f) = \frac{|G_{x,y}(f)|^2}{G_{x,x}(f)G_{x,x}(f)}$$

At each frequency band, the coherence spectrum is taken as a measure of synchronized activity at that band. When some single unit spike data is additionally available we can find the degree to which a unit is entrained by a field oscillation can be identified by calculating the *spike-field coherence* which is the $C_{x,y}$ for x being the spike train (translated to the superposition of Dirac delta functions) and y being the field potential.

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2 Synchronization as a universal phenomenon

The recognition of synchronization goes back at least to Huygen's synchronizing pendulums. In 1665, he wrote to his father: *"I have noticed an admirable effect which no one could have ever thought of. It is that these two clocks standing next to one another separated by one or two feet keep an agreement so exact that the pendulums always oscillate together without variation [...] through a kind of sympathy."* [26]. By now, synchronization is understood as a universal phenomenon occurring in a large range of scales from elementary particles to populations of organisms. Some examples from physiology alone include circadian and cardiac pacemakers, β-cells in the pancreas, and, as we shall see, the cortex. Furthermore, synchronization as the spontaneous emergence of *temporal* order in a heterogeneous network of coupled oscillators is but one example of *entrainment* and a special case of *phase-locking*. In the latter case, oscillators converge to a arbitrary fixed relation between their phases whereas in synchrony this fixed relation is required to be identity.

2.1 Phase Resetting Curves

For any oscillator with period T, at any given time t one can define a *phase* $\phi(t) \in [0, 1]$ as the ratio of the time elapsed since the last occurance of an arbitrarily chosen landmark to the time length of the period (that is $\dot{\phi} = 1/T$ at all times). For an oscillating neuron, for instance, this arbitrary point can be the action potential upstroke. By periodicity, one also has to identify 1 with 0 to capture the fact that $\phi = 0$ and $\phi = 1$ represent precisely the same states of the oscillator. This leads to the canonical geometric idealization of an oscillator as a function ϕ mapping the time axis to the unit circle S.

Consider an oscillator identified by $\phi : \mathbb{R} \to S$ and suppose that when it passes some phase τ it is perturbed such that it eventually returns to the same periodic trajectory; i.e. the perturbation is not so large to send the system to another steady or periodic state. Naturally, after the perturbation the oscillator will have a shift in $\Delta\phi$ such that its next cycle may begin earlier or later than it would have without the perturbation. The *phase resetting curve* (PRC) is the graph of $\Delta\phi$ against the phase τ at which the perturbation is applied. For an oscillating neuron, for instance, the PRC measures the phase advance or delay of the upcoming spike (Fig. 1). A *type I* PRC is one which is always positive and a *type II* PRC is one which has positive and

negative values [3]. Both PRC types are physiologically relevant and have consequences on the existence and stability of the synchronous state.

The PRC is the natural mathematical tool in the study of interacting oscillators and has been successfully applied to a wide range of biological systems [27]. Much of the results about synchronization, too, can be best understand in terms of the PRC [28]. For instance, we will see that networks of neurons with type II resetting are more conductive to synchronization.

2.2 Synchronization in neuronal network models

Any model of neuronal networks must necessarily capture two classes of information: **i)** the behavior of individual neurons and their response to interactions, and **ii)** the structure of the network, namely its *connectivity*, degree of *coupling*, and *heterogeneity* amongst constituent neurons. Candidates for individual neuron models include high dimensional models (any of the variants of the Hodgkin-Huxley model), two dimensional models (specifically, the two dominant parameter regimes of the Morris-Lecar model), and the one dimensional integrate-and-fire model (or its leaky variant). Two categories of network connectivity, all-to-all and sparse, have been heavily studied. Coupling between two neurons corresponds to the excitatory or inhibitory postsynaptic potential delivered at synaptic input and is represented by a single number. Heterogeneity is naturally modeled by a distribution over the intrinsic frequencies. Among single neuron models, the high dimensional group (HH and variants) are practically analytically intractable in a network setting and numerical simulations are needed. The two dimensional models have the advantage of capturing the relaxation-oscillation quality and allow for variability in action potential shape to be studied while remaining amenable to geometric reasoning and simplified calculations. The integrate-and-fire model reduces the dynamics to a discharging capacitor which fires a "formal" spike and resets immediately; that is, it has no refractory period and has discontinuous trajectories.

2.2.0.1 A synchronization theorem

A population of identical, all-to-all connected, identically coupled, excitatory-only, integrate-and-fire neurons converges to a state of synchronous oscillation regardless of initial conditions.

The original context of this theorem [29] was to model the synchronous activity of cardiac pacemaker cells. The mathematical ideas involved [30], [31] are elegant yet simple (for a pictorial proof for 2 neurons cf. Fig. 5). From a biological viewpoint, however, its requirements are not even remotely satisfied in the cortex.

The goal of this section is to give a qualitative overview of some of what is known about how synchronization is affected by lifting some of these requirements. The common theme is that instead of a globally stable synchronous regime, a rich repertoire of oscillatory behavior including global synchrony, global asynchrony, clusters of synchronized subpopulations, or global oscillating activity with irregular individual spiking. Furthermore, the existence, stability, and spatial range of a stable synchronous state becomes

[3] Not to be confused with type I and type II excitability of a neuron which corresponds to whether or not oscillations have a nonzero frequency at birth. In this paper we are solely interested in phase resetting types and not excitability types.

dependent, often in surprising ways, on degree of coupling, existence of inhibitory connections, degree of connectivity, time constants, axonal delays, and even noise levels. In summary, we will see that:

1. Although lifting each simplification complicates the picture in some way, synchronization typically remains a possible stable state of the network under proper parameter regimes.
2. The mechanism of synchronization is highly dependent on network-level and neuron-level properties. Changing either component (e.g. connectivity, ionic currents in single-neuron model, etc.) typically affects the route to synchrony and the intrinsic synchronous frequency.
3. The stability of the synchronous state depends delicately on various factors which when allowed to vary can lead to counterintuitive effects. Therefore, arbitrary network configurations typically require ad hoc analysis of synchronization stability.
4. Excitatory-only networks and networks with neurons with only type I resetting curves are generally less conductive to synchrony. Typically, one needs either inhibitory connections or type II resetting curves for a stable synchronous state. An individual neuron's phase resetting curve type can be changed by manipulating the width of its afterhyperpolarization window.
5. Noise does not always have a signal exhaustion effect. In many cases, moderate amounts of noise stabilizes synchrony, improves downstream efficacy, and can possibly initiate synchronization.

2.2.0.2 Continuous neural response

One of the strongest simplifications of the integrate-and-fire model is the discontinuity in membrane potential after a spike. In all the following results the underlying single cell model is continuous. This can be done by either using a higher dimensional model or adding an action potential waveform to the integrate-and-fire model.

2.2.0.3 Phase resetting type

An integrate-and-fire neuron necessarily has type I phase resetting in an excitatory network since all synaptic inputs advance the time of successive spikes. To get a type II phase resetting curve the individual neuron model must have a refractory period with a afterhyperpolarization period during which synaptic input delays the subsequent spike (Fig. 3). The standard HH model exhibits such behavior while typical two dimensional relaxation oscillation models do not [32]. The "size" of the negative lobe in the phase resetting curve corresponds to the width of the afterhyperpolarization period. A neuronal RPC, unlike an HH model RPC, does not typically have a large afterhyperpolarization window. This implies that neuronal RPC's can effectively be of type I depending on the dynamics of Na^+ channel reactivation and the decay of the K^+ rectifying current.

If constituent neurons of an all-to-all connected excitatory-only network have type I phase resetting, the stability of the synchronous state depends on weak coupling; that is, strong coupling destabilizes the synchronous state [32], [33]. Note that this escapes the analysis of [30], [31] which assumes discontinuous integrate-and-fire neurons. On the contrary, if the neurons in the network have type II phase resetting

the synchronous state is stable [28], [32]. When the shape of a type II phase resetting is simple (e.g. with one negative and one positive region as in Fig. 1, 2) this is intuitively obvious since, roughly speaking, neurons that are "ahead" will slow down at the arrival of each synaptic input and those that are "behind" will speed up.

2.2.0.4 Inhibitory connections

Introduction of inhibitory connections [4] alone opens a new range of possibilities even while keeping the integrate-and-fire model for individual neurons. Aside from stable synchrony a sparsely-connected inhibitory and excitatory network can show stable synchrony or oscillating global activity while individual neurons fire irregularly [34]. Furthermore, inhibitory connections generally have a stabilizing effect on synchrony in networks of type I phase resetting neurons and a destabilizing effect on synchrony in networks of type II phase resetting neurons. In this context, excitatory connections have the opposite effects. Finally, inhibitory connections *alone* are capable of synchronization and as we shall see, are highly relevant to the cortex as underly the mechanisms of rhythmogenesis, for instance in the hippocampus and the thalamic reticular nucleus (cf. section on γ- synchronization).

2.2.0.5 Time scales

Earlier we said that all-to-all connected excitatory networks of neurons with type II phase resetting always synchronize. This is no longer necessarily true when the time constant is allowed to increase (within physiological bounds): for time constants larger than a threshold the stable synchronous state ceases to exist [32]. In *sparse* excitatory and inhibitory networks, however, time constants are only relevant in slow oscillation regimes whereas in fast oscillation regimes only synaptic timescales determine the stability of synchronization [34].

Another counterintuitive result is obtained from incorporating axonal time delays in spike transmission. It has been shown that the existence of axonal delays can lead to zero-lag long-range synchrony which would have been impossible otherwise. This is, again, highly relevant to the cortex as time delays in model networks are crucial in establishing long range gamma band oscillations, for instance both in the hippocampus and the thalamic reticular nucleus (cf. section on mechanisms of cortical synchronization).

2.2.0.6 Coupling strength

As mentioned above, coupling strength is the determining factor in the stability of the synchronous state when neurons in an all-to-all excitatory network have type I phase resetting while similar networks with type II phase resetting neurons have a stable synchronous state regardless. However, rather counter-intuitively, the discharge rate (oscillation frequency) of neurons in the latter case *decreases* with increased excitatory coupling [32]. More surprisingly, in an excitatory-inhibitory network strong coupling can lead to *oscillation death* [33]. In other words, not only does strong coupling destabilize the synchronous state it

[4] These inhibitory connections are mixed with excitatory connections. An entirely inhibitory network is more or less the symmetric reflection of an all excitatory network except for that it needs sustained applied current to maintain oscillation.

also destabilizes the oscillatory state for individual neurons. In this context, (chemical) synaptic connections in the cortex are all weak but gap junction connections are rather strong [35]. Additionally, gap junctions induce fundamentally different synchronization properties since their phase resetting behavior depends on the membrane potentials of both pre-synaptic and post-synaptic neurons [28].

2.2.0.7 Heterogeneity

All the above results concerns oscillators with identical intrinsic frequencies. If the neurons are drawn from a distribution of frequencies the stability criterion for synchrony takes an entirely new form: a subpopulation of a certain size must be synchronized before the entire network can have a stable synchronous state. The size of this threshold subpopulation is, to a first approximation, proportional to the intrinsic periods of neurons; that is, under fast spiking, a smaller synchronized subpopulation suffices for global synchrony [27].

2.2.0.8 Effects of noise

Background fluctuations and synaptic noise make neurons stochastic oscillators. Naturally, local coherences can be overwhelmed over long distances by large enough amplitudes of noise. However, within physiological values for the cortical neuron, noise has been shown to have a beneficial effect. This effect is an example of *stochastic resonance* (in the loose sense of the word) which is characterized by a threshold value of noise which has beneficial effects on some system-level property of interest. Stochastic resonance is typically understood in the context of signal-to-noise ratio of signal processing systems where the optimal value of SNR is achieved with a nonzero magnitude of noise (Fig. 4). In the context of synchronization, however, the "benefit" of noise is in acting as a stabilizing force on the synchronous state [36], the asynchronous state [37], or the spontaneous induction of synchronous firing [38].

2.2.0.9 Effects of adaption

In all the above results the firing rate of each neuron is considered to be constant. A cortical neuron, however, is exposed to various adaptation processes and similarly, the degree of coupling is exposed to variations due to plasticity. It would, therefore, be expected that as the firing rate and coupling of neurons evolve, their PRC's and correspondingly the stability of a synchronous state may change. Furthermore, Ca^{2+} dynamics, through its side effects on the afterhyperpolarization period can potentially change the phase resetting type of a cortical neuron.

2.2.0.10 Stability of the asynchronous state

An important dual case is that of a stable asynchronous state. The often neglected importance of this state is that the firing rate model requires a stable asynchronous state to have useful information content [37]. This is intuitively obvious since in a synchronous or partially synchronous state (e.g. when the network clusters into synchronous subpopulations) the information gain of a firing rate neuron drastically decreases

as it is bound to fire in unison with its synchronized neighbors rather than in response to stimuli. Yet again, the presence of noise mostly mitigates this problem [37].

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3 Empirical evidence

3.1 Correlation coding in cortical neurons

An early indirect indication of correlation coding was provided by Bialek and colleagues [10] who considered the problem of designing a filter which, from the point of view of the experimenter, reconstructs the stimulus waveform from the spike train of an blowfly H1 neuron. Assuming invariance under time translation, such a filter in its most general form can be written as [10], [39]:

$$s_{est}(t) = \sum_i F_1(t - t_i) + \sum_{i,j} F_2(t_t - i, t - t_j) + \dots$$

The optimization problem, when the stimulus $s(\tau)$ is known and controlled, is to find the filters $F_1, F_2, \dots$ such that the error $\int |s_{est}(\tau) - s(\tau)|^2 d\tau$ is minimized. Now suppose the neuron recieving this spike train has only first order dependence on the timing of spikes, that is all terms but F_1 vanish in the expression for s_{est}. Then fourier analysis leads to a precise characterization of the optimal kernel F_1 in terms of the autocorrelation function of the spike train and the crosscorrelation of the spike train with the stimulus [39]. The suprising result was that the reconstructed waveform using only spike train of individual H1 neurons approximated the correct stimulus to a reasonable degree (Fig. 8). Furthermore, incorporation of higher order terms (i.e $F_2, F_3, \dots$) led to marginal improvements in reconstruction quality. This implies that the exact spike time information in a single neuron can, at least in principle, reconstruct the entire stimulus waveform to a reasonable degree. In information-theoretic terms, neurons sensitive to exact spike timing can encode up to 3 bits of sensory information per spike amounting to rates as high as 300 bits per second. The temporal resolution necessary for such bandwidth is roughly 5-10ms [40]. All this does not imply that a downstream neuron actually uses this information, but it is clear evidence against the assumption that single neurons are inherently unreliable.

Later, Tsodyks and Markram [11] were able to show, by *in vitro* and simulation studies, that pyramidal neurons can reliably decode either for firing rate or for temporal correlation depending on the rate of synaptic depression. First, they demonstrated that with increased presynaptic firing rate, the stationary EPSP response of the pyramidal neuron follows a $1/f$ law (Fig. 9 left panel) where beyond a limiting frequency increased presynaptic firing rate has no effect on the *stationary* EPSP response. However, prior to the stationary EPSP phase a window of transient spikes is always present (Fig. 9 left panel A). With all its incoming connections *in vivo*, a pyramidal neuron thus has a *summation window* even at frequencies beyond the limiting frequency such that synchronized arrival of presynaptic spikes leads to a postsynaptic spike. The width of this summation window is in the promised 5-10 ms range (proportional to typical

membrane time constant). The modulation of synaptic depression rate was demonstrated by numerically simulating a model pyramidal neuron with Poisson spike trains arriving at its 500 dentritic synapses (Fig. 9 right panel). At high depression rates the neuron essentially acts as a coincidence detector for coherent firing rate transitions in the presynaptic population. At low rates of synaptic depression the neuron responds as a classical average firing-rate decoder. Similar results have reported for the control of operational mode of pyramidal neurons by membrane conductance [12] and background synaptic activity [41]. Aside from the necessity of a coincidence-detector mode for the correlation theory to hold, the existence of alternative operational modes for cortical neurons possibly has deep consequences on brain function, e.g. it has been implicated in "ignition" theories of consciousness [42].

3.2 Propagation of synchronous activity

Little is known about the spatial organization of synchronous activity. A natural model that emerges is that of *synfire chains* [20] in which the synchronous firing of a group of neurons which symbolically represent some piece of information leads to the synchronous firing of a subsequent group of neurons (Fig. 10). The propagation path of this chain of synchronous firing highly depends on the short-term and long-term plasticity of the connections between each cortical group to the subsequent group and the readiness for each activated group to maintain synchronization. Although obtaining experimental evidence of such an organization is extremely hard, some of its properties have been demonsrated *in vitro* [43] (Fig. 11): **i)** synchronization develops spontaneously even with uncorrelated input and is maintained over a large range of physiological conditions, **ii)** signal propagation through the network is greatly facilitated by synchrony, and **iii)** synchronization develops gradually from the early layers towards the deeper layers with the deeper networks firing synchronously and the early layers firing as integrators with essentially asynchronous output.

However, as we shall see, a more dynamic model of coordination and communication between synchronizing circuits probably underlies much of the brain's cognitive capacities.

3.3 Mechanisms of cortical synchronization

First, a few remarks along the lines of those in section 2 in direct regard to cortical synchronization are in order. First, the typical cortical neuron has type II resetting curves (i.e. they have a nonzero afterhyperpolarization period) and therefore inhibitory connections have a strong stabilizing effect on synchrony. Specifically, inhibitory interneurons have been implicated in almost all forms of EEG oscillations. Second, note that local cortical networks are quite sparse. For instance a pair of pyramidal neurons are connected in either direction with a probability of 0.1-0.2 [44]. We mentioned earlier that sparsely-connected excitatory-inhibitory network can show stable *global* oscillating behavior (e.g. to a first approximation this corresponds to measured field potentials) while individual neurons fire more or less irregularly [34]. Another result about sparsely-connected networks is this: in randomly connected sparse networks with inhibitory and excitatory connections approximate synchrony is stable granted that the average number of incoming connections per cell is large enough. The destabilizing effect of low connectivity can to some

extent be compensated by strengthening excitatory-to-inhibitory connections, and surprisingly, not vice versa [45].

3.3.1 EEG rhtyhms

EEG oscillations have have been known for more than a century and are classified by their frequency range: δ (1-4 Hz), θ (4-8 Hz), α (8-13 Hz), β (13-30 Hz), and γ (25-80 Hz). Although, the lower frequency oscillations were the first to be observed, β and γ have been more heavily studied possibly owing to their demonstrated implication in cognition and psychiatric disease [46]. From the perspective of the previous section, β and γ both represent possible synchronous frequencies for sparsely-connected excitatory-inhibitory networks. For instance, heterogeneous sparse networks of model HH neurons can generate β and γ-synchronization. Among *in vitro* models, one of the best studied examples is hippocampal pyramidal-basket networks. Inspired by hippocampus function, both β and γ have been studied for their ability to sustain long range synchronization (which recall, relies on axonal delays for its stability). In fact, the route to synchronization in both cases share a common qualitative feature: interneuron *doublet spiking* is always observed prior to synchronization [47] (Fig. 12). However, the two have differences in their ionic dynamics. Specifically, β-synchronization, due to its slower time course has access to afterhyperpolarization currents which lead to its stability in the long-range while γ is less robust in the long range [48]. This is consistent with other experimental evidence suggesting that γ-synchronization is relevant in local computations and β-synchronization in those involving distant structures.

3.4 Categories of cortical synchronization

There are three main sources for synchronization in the cortex. First, groups of neurons may fire in unison simply because they are independently time-locked to the same external stimulus. This does not preclude interactions between said neurons but no computational function is assigned to this kind of synchronization. Second, a properly connected group of neurons, typically in the sensory cortex, may self-organize into short-lived synchronous firing under some form of the conditions discussed in section 2. As per the correlation theory, sensory neurons enter and leave such transient cell assemblies every fraction of a second. The stability of these assemblies is modulated in real time by short-term plasticity and in the long run by Hebbian plasticity. Additionally, as a communication mechanism synchronization has consequences on the efficiency of delivered synapses by a synchronized population to their downstream populations. Third, as indicated in section 2, connectivity patterns, coupling strengths, and the balance between inhibitory and excitatory connections determine the degree to which a neuronal network is conductive to synchronization. The relevance of this "synchronizability" in cortical communication seems to be in the ability of a circuit to be entrained by sensory information (bottom-up) or to entrain sensory information (top-down). In other words, synchronization seems to serve as a mechanism for communication between distant cortical circuits [49]. In the following sections we present evidence for the implication of synchronization, specifically γ and β, in neural representation and communication both with consequences on computation.

3.5 Functional roles of gamma-band synchronization

3.5.1 Representation by synchrony

We have discussed in detail the possible role of synchronous activity in neural representation and its consequences on computation. In this section we briefly present the experimental evidence. Most of the early evidence is to Singer, Gray, and colleagues [15], [52], [53] who demonstrated, first in anesthetized and then in awake animals, that neurons encoding attributes of visual stimuli synchronize when these attributes are bound together as one perceptual object. Fig. 13 shows the experimental results from macaque MT neurons which synchronize in response to a moving bar in the visual field but fall out of synchrony where at the same position two smaller bars are presented. The relevance of this effect has been further elucidated by showing the involvement of such synchrony-based representations in object recognition and memory. Fig. 14 demonstrates the differential γ power of EEG activity in human subjects who are presented with objects with long term memory representations compared to objects without [54].

3.5.2 Communication by synchrony

Putting aside long-term plasticity for a moment, we note that under the conventional framework cortical coordination and communication are assumed to be predominantly controlled by anatomical connections. If synchronization is a valid form of neural representation it must also have functional consequences on how groups of neurons can communicate. Specifically, the principles dictating the dynamics of synchronization in a network (cf. section 2) are essential identical to that of entrainment of one population by another. It follows that if synchronization serves as a representation code, it could also be involved in communication. This role for synchronization is known as "communication through coherence" [50], [51]. Here we present evidence for the role of γ-synchronization in cortical communications involving attention (also see Fig. 15 for evidence of γ and β communication in working memory).

The idea that γ-band activity is in some way related to attention has long been suspected. The implicated circuitry is the thalamic reticular nucleus which has been known to have inhibitory projections to the thalamic relay neurons, and to contain bursting neurons leading its self-synchronizing properties. Crick's "searchlight hypothesis" aimed to use Von der Malsburg's short-term synapses to explain what is known about feature integration from visual conjunction search experiments (cf. section 1, [6]). The hypothesis says that the thalamic reticular nucleus probes the cortex, at the γ frequency, and decides "where the action is" [55]. Although thalamic rhythmogenesis and the role of synchronization in attention has been confirmed [56] (Fig. 17) the communication by coherence explains the dynamics in reverse order: synchronized ensembles of sensory neurons, representing some stimulus, *compete* to entrain higher areas where top-down influences are mediated by $\alpha - \beta$ and bottom-up influences are mediated by γ (and Crick's searchlight mechanism is delegated to θ-rhythmic γ modulation). The relaxation-oscillation properties of the target area neurons have consequences on the dynamics of this competition for entrainment. Most significantly, numerical results seem to validate the idea that a stimulus (i.e the neural representation of a stimulus) with more coherence has a stronger chance of entraining a target population and that a less coherent stimulus is left vulnerable to "distracting" stimuli entraining the target population to their

own synchronous rhythm. This competition of stimuli to entrain a higher-level circuit is hypothesized to underly the mechanism of selective attention [49], [57] (Fig. 16 shows numerical reseults for such competition). More specifically, once put in terms of oscillator entrainment, it becomes apparent that the factors affecting the outcome of this competition are numerous (e.g. phase difference, amplitude ratio, whether entrainment is done by projections to excitatory or inhibitory components of the target network) [57].

4 Future Directions

1. The communication by coherence hypothesis claims that in a race for attention, the winning signal is the more coherent one. Furthermore, it has been demonstrated that rhythmic stimulation of neurons in the visual cortex can lead to saccades to their corresponding receptive fields [58]. In order to confirm the prediction of the communication by coherence hypothesis, a similar experiment can be performed where signals with varying degrees of coherence are applied at two visual cortical areas. The prediction is that the more coherent signal should dictate the direction of saccade.
2. It is known that visual memory recall involves the activation of the corresponding sensory neurons in the visual cortex [59]. In order to investigate the role of γ-synchronization in memory recall, destabilizing stimulations can be delivered optogenetically to awake animals [60]. Mice can be trained to perform behavioral tasks requiring visual memory recall and a negative impact of γ destabilization can be observed behaviorally. However, it will be challenging to design a behavioral paradigm and a γ-destabilization strategy which is timed such that it does not disrupt the circuits involved in the behavioral task, e.g. the motor system.
3. Little is known about the entrainment properties of various kinds of neurons in the cortex. An exploratory set of *in vitro* experiments is to measure phase resetting curves of important types of cortical neurons. Such studies can pave the way for more accurate *in vitro* reconstructions of certain cortical networks. Furthermore, *in vitro* reconstructions of idealized models can help confirm the connection between theoretical and experimental results. (we have mentioned results from multiple *in vitro* studies which have provided great insight: [11], [43], [47]).

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5 Figures

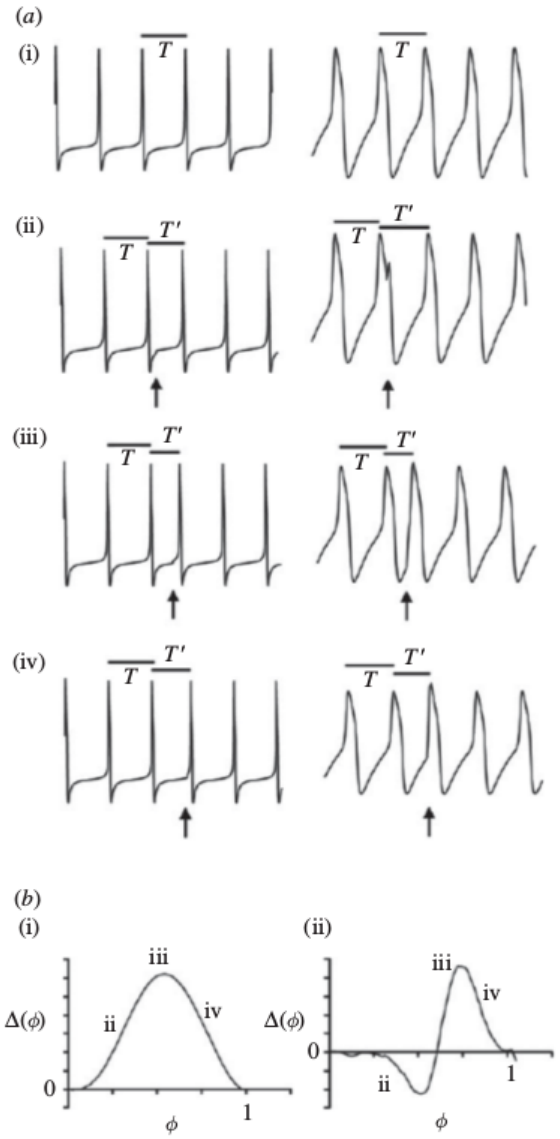

Figure 1: Example phase resetting curves of type I (*left*) and type II (*right*) for two typical oscillators modeled by two parameter regimes of the Morris-Lecar model. The arrows indicate the time of synaptic input. The times T and T' represent the time to next spike with and without perturbation. Synaptic inputs delivered at phases where the PRC is positive lead to earlier next spikes (i.e $T' < T$) and vice versa for phases where the PRC is negative. (taken from [28]).

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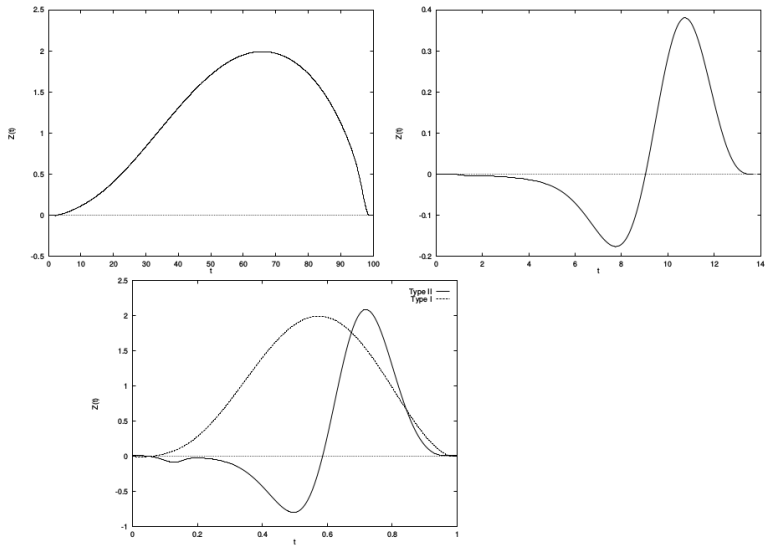

Figure 2: Phase resetting curves for the Connor neuron which has type I reseting neurons (*top left*), Hodgkin-Huxley neuron which has type a II phase resetting curve (*top left*), and a Morris-Lecar neuron under two main parameter regimes (*bottom*). In all-to-all connected networks of all-excitatory synapses only type II neurons lead to a stable synchronous state [32] (taken from [35]).

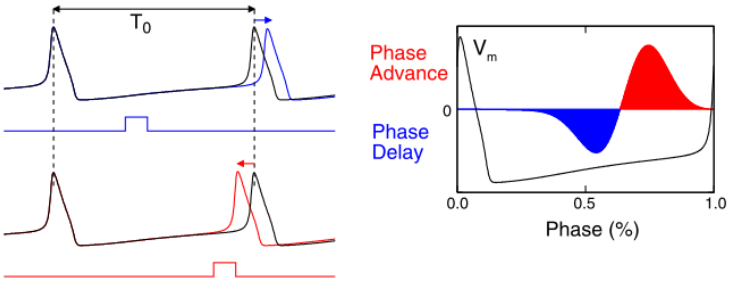

Figure 3: Type II phase resetting curve of a neuron with afterhyperpolarization effect in the refractory period. During the afterhyperpolarization window, any depolarization delays the successive spike by delaying the decay of the inward rectifying K^+ current as well as the recovery of inactivated Na^+ channels. The standard HH neuron exhibits this effect (taken from [44]).

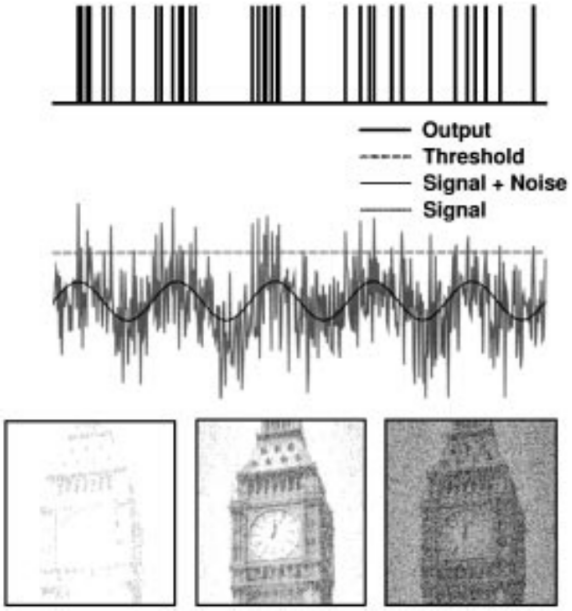

Figure 4: *Top*: An example of stochastic resonance in a threshold regime: a subthreshold signal leads to spikes in the presence of noise. The observation that SNR is zero for very large and very small amounts of noise makes the intuition clear that moderate amounts of noise (as shown) can have a positive effect on SNR. *Bottom*: Visual perception of stochastic resonance. An image of Big Ben is filtered to 0/1 pixels without added noise (*left*), with moderate added noise (*middle*), and large amounts of noise (*right*). The middle picture has the largest SNR of all three (taken from [61]).

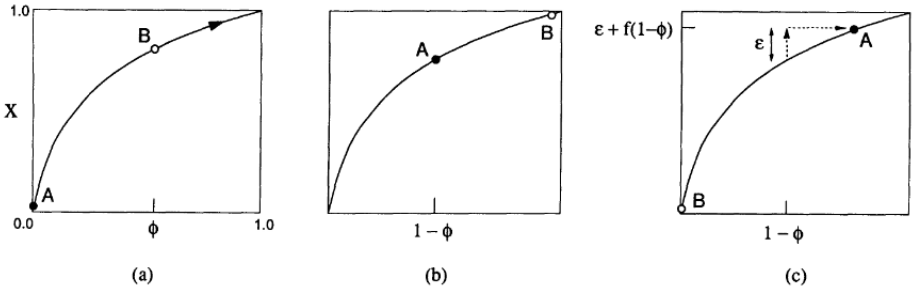

Figure 5: Pictorial proof of the synchronization theorem for two neurons A and B (taken from [30]). Let the integrate-and-fire model be $x = f(\phi)$ with threshold 1 where $\phi \in [0, 1]$ is the phase with respect to firing and f is any concave down increasing function (e.g. a charging capacitor's potential). We want to show that in the limit of arbitrarily long time the phase of A and B will converge. This can be discretized by seeking proof that in the limit of large number of firings of A, the phase of B just after A fires tends to zero. In (a) A has just fired and B is at some phase ϕ, in (b) at phase $1 - \phi$ later, B is about to fire and in (c) B has just fired and A, having just recieved a postsynaptic potential of ϵ is at $h(\phi) = \epsilon + f(1 - \phi)$. That is, A went with one firing of B from 0 to $h(\phi)$. Now B is at zero and we wish to know where it will be the next time A fires. That would be $h(h(\phi))$. So we wish to show $h(h(\phi)) < \phi$ where h has a known form in terms of f. The rest is some calculus to show this inequality.

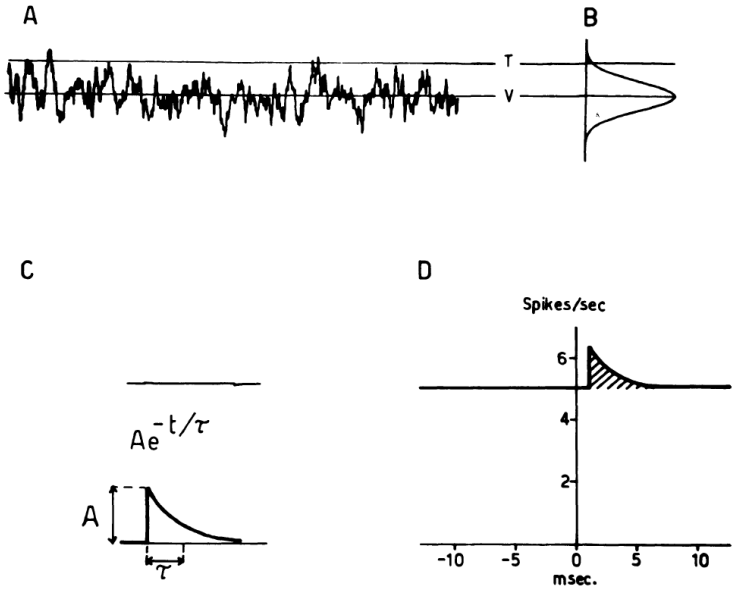

Figure 6: The idealized membrane potential of a cortical neuron. Action potentials are ignored and the probability of a spike at a given time corresponds to the Guassian tail in B. All EPSP's are assumed to have the same shape as in C (top trace an EPSP, IPSP pair drawn to scale of A). It can be shown that the effect of each EPSP on the instantaneous firing rate is as shown in D (taken from [20]).

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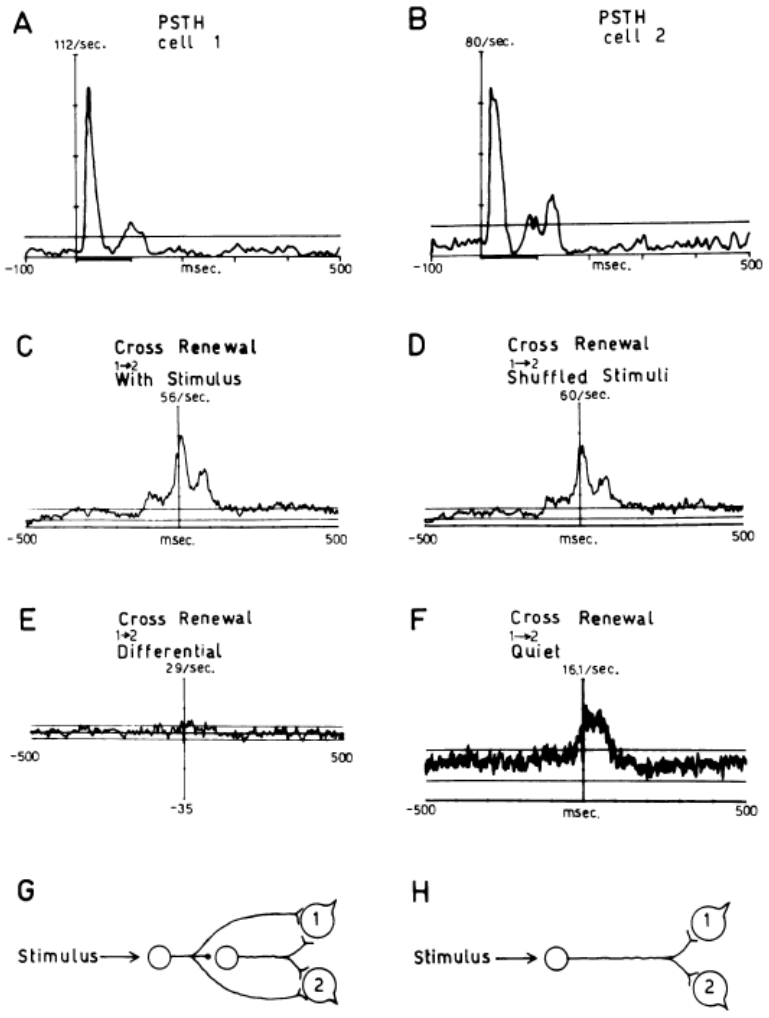

Figure 7: PSTH for two single units (A and B). The cross-renewal density (C) is shuffled (D) and the difference is plotted (E). When no stimulus is present the cross-renewal density is as in F. Two possible connectivity patterns (G and H) that can explain the difference between E and F. (taken from [20]).

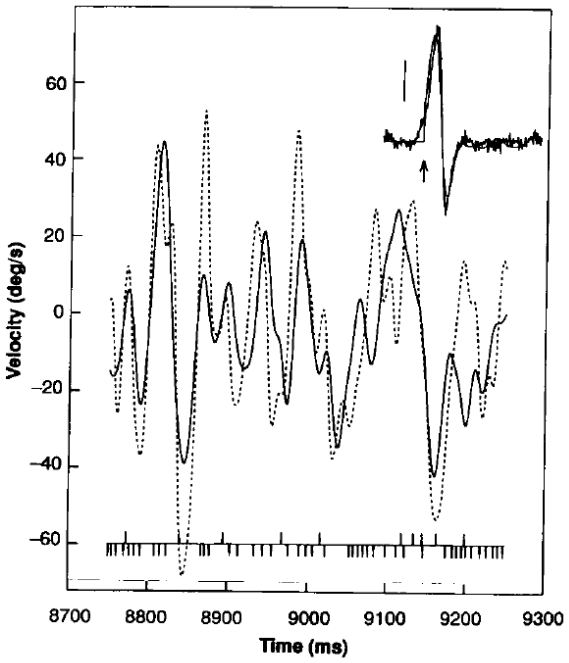

Figure 8: Bialek's reconstruction of a controlled stimulus from single unit recording of blowfly H1 neurons (taken from [10]). The stimulus $s(\tau)$ is the angular velocity of a rigidly moving random pattern. The solid line is the stimulus $s(\tau)$ and the dotted line the reconstructed stimulus $s_{est}(\tau)$. *Inset*: The optimal decoding kernel F_1. The motivation to study $H1$ neurons is the that the fly's response time (50-100 ms) only allows capturing 5-10 spikes which means the downstream neuron cannot be relying on average firing rates. This analysis shows that the exact spike timing does contain, at least to an observer who can decode it, a lot more information than generally expected under the "unreliable neuron" assumption.

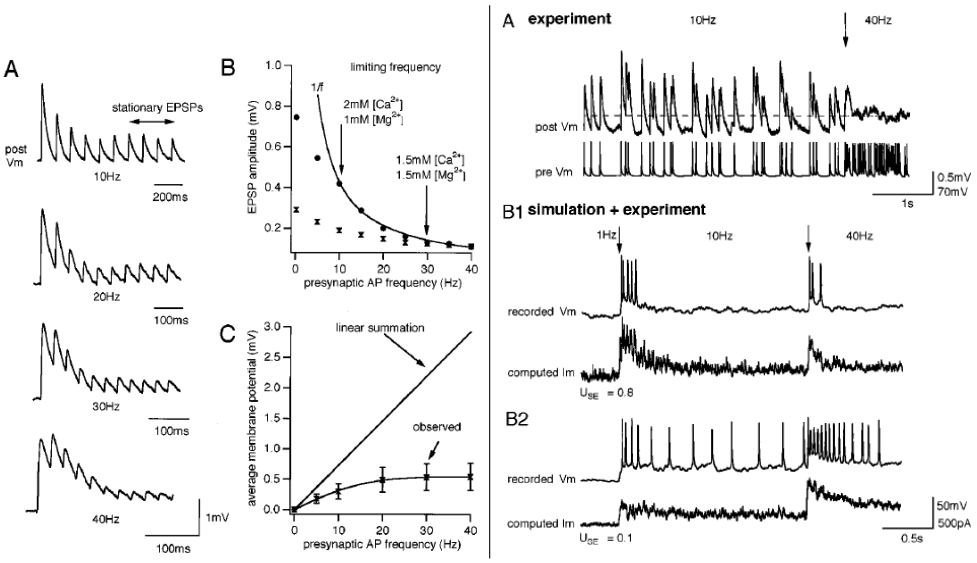

Figure 9: Experiments of Tsodyks and Markram showing two modes of information decoding in layer 5 pyramidal neurons of the rat somatosensory cortex (taken from [11]). *Left*: The $1/f$ law relating the amplitude of stationary EPSP to the input firing rate. In A the intracellular EPSP in response to stimulation with frequencies of 10-40 Hz or shown. The EPSP magnitude decreases with increased input frequency (B) for two values of extracellular Ca^{2+} and Mg^{2+} concentrations. Note the limiting frequency beyond which stationary EPSP essentially vanishes and the time-average membrane potential plateaus (C). *Right*: A: Intracellular recording of the postsynaptic potential of the same pyramidal neuron with an input spike train that transitions from 10 to 40 Hz firing rate at the indicated time. Notice that consistent with the results on the left panel, the postsynaptic average potential remains identical before and after the transition. B: Simulation of the model neuron with Poisson spike trains from a population of presynaptic neurons at two rates of synaptic depression U_{SE}. The presynaptic population transitions from 1 Hz firing rate to 10 Hz and then to 40 Hz at indicated times. In B1, the neuron is acting as a coincidence detector responding selectively to coherent increases in the incoming firing rates of the presynaptic population. In B2, the neuron is acting as a firing-rate encoder/decoder which monotonically increases its own firing rate with increases in the firing rate of the presynaptic population.

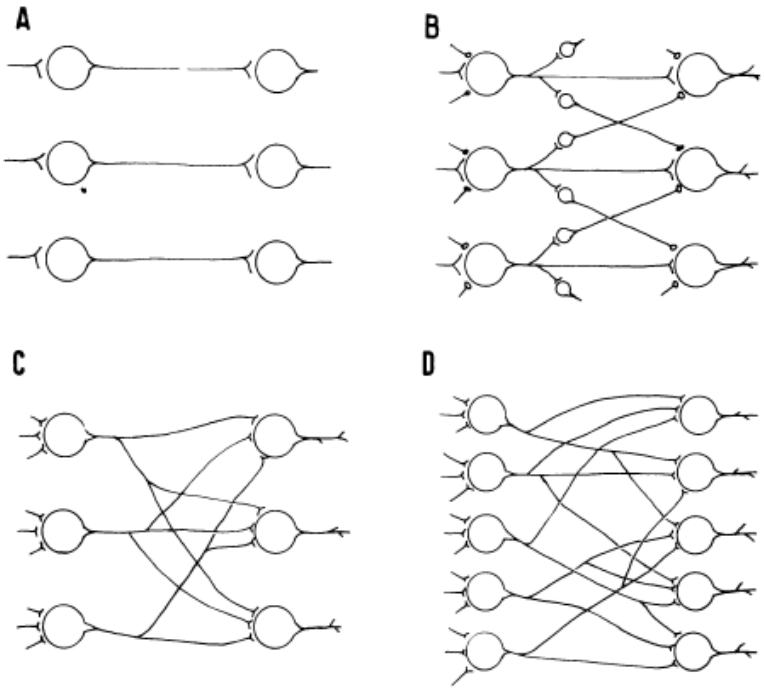

Figure 10: A one-to-one chain (A) with lateral inhibitions (B). A synfire chain (C) modified such that each layer has 5 neurons and connects to 3 neurons in the subsequent layer (taken from [20]).

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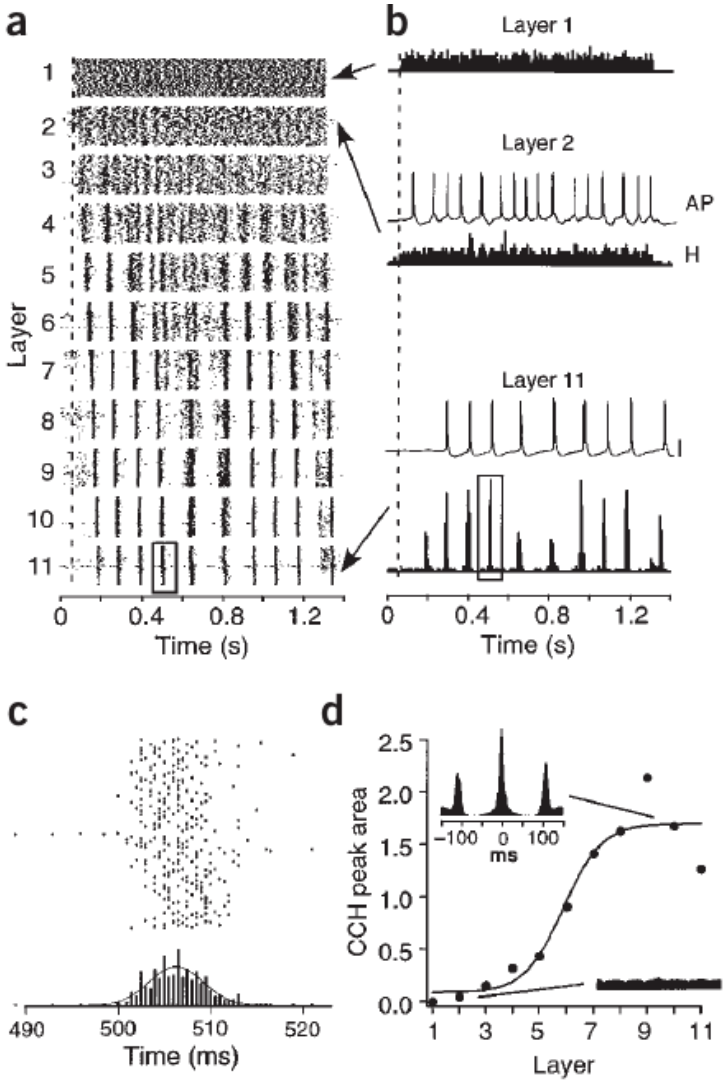

Figure 11: Reproduction of a synfire chain in vitro (taken from [43]). Rat somatosensory pyramidal neurons were iteratively connected into a multilayer feed-forward network with 200 neurons in each layer. Synchrony develops even with uncorrelated input. a) Raster plot of spikes in the entire network, b) representative membrane potentials (AP) and spike time histograms (H) in layers 1,2,11. c) magnified raster plot and spike time histogram for the identified box in a. d) Cross-renewal density (CCH) peak area is plotted against the layer depth. Notice that coherence develops gradually through the layers consistent with a.

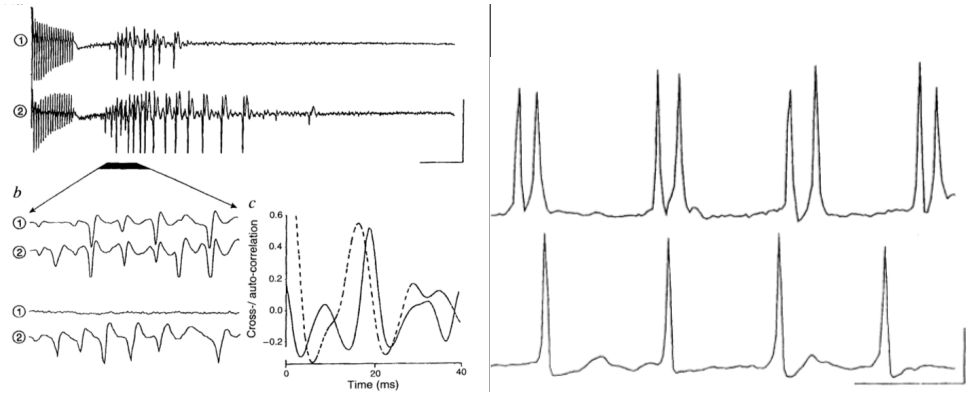

Figure 12: Spike doublets en route to long-range synchronization in vitro. *Left*: a) Two CA1 sites 4mm apart in a rat hippocampal slice are tetanically stimulated by a 100 Hz spike train of 20 stimuli which leads to temporary synchronization. b) Alternatively, only site 2 is stimulated which leads to a shorter oscillatory response. Scale bar for a,b: 200ms, 40mV. c) solid line is cross-correlation of the response at 1 and 2 when they are both stimulated (frequency = 52 Hz), dashed line is auto-correlation for response at 2 when 1 is not stimulated (frequency = 62 Hz). *Right* Expanded membrane potentials in either case, top trace: both sights stimulated (note the doublets), bottom trace: only 2 stimulated. Scale bar: 20ms, 40mV

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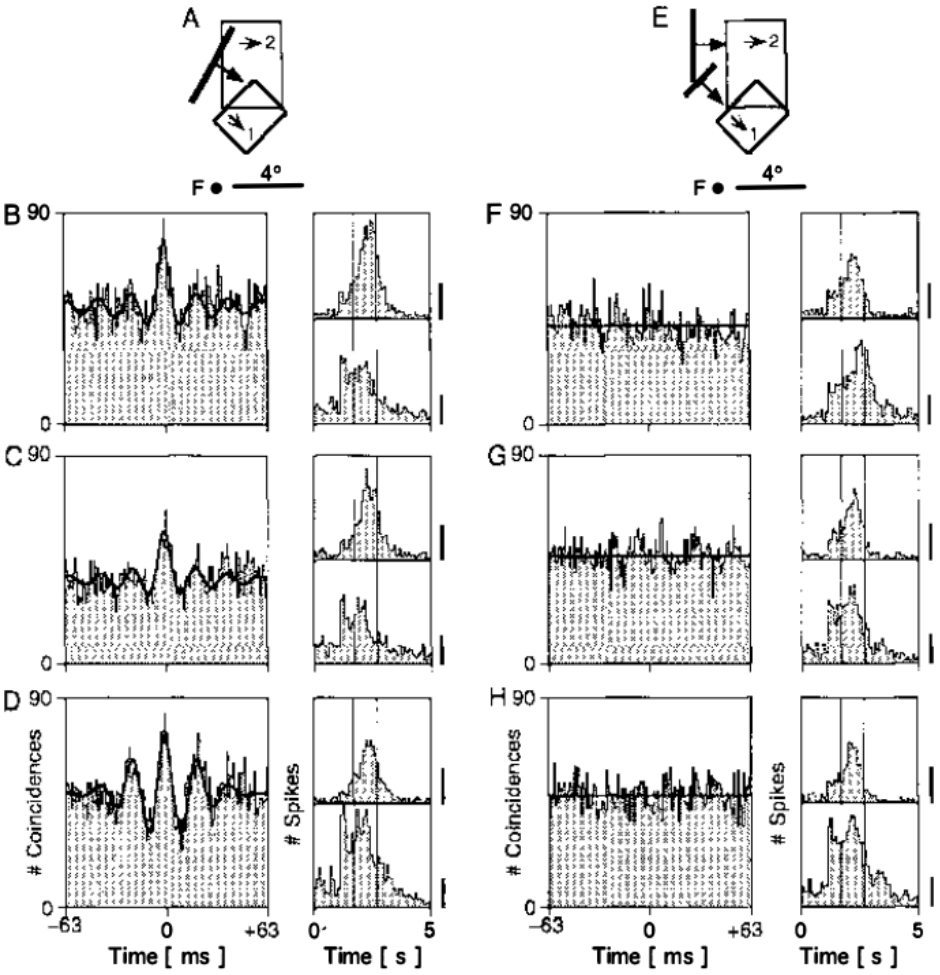

Figure 13: Evidence for the temporal correlation theory (taken from [53]). Single unit recordings from the MT area of awake macaque while two conditions of visual stimuli are presented. *Left*: A single moving bar is presented in 6 blocks of 10 trials at the overlap of the corresponding receptive fields of two MT neurons. PSTH plotted for each of the 6 blocks in on the right with vertical lines indicating the range of crosscorrelation diagrams on the left. Notice the consistent synchronization of MT neurons. *Right*: Two separate bars (indicated by different direction of movement) are presneted at the same positions such that one lies in the receptive field of one neuron and one in that of the other. Notice the complete absence of synchronization.

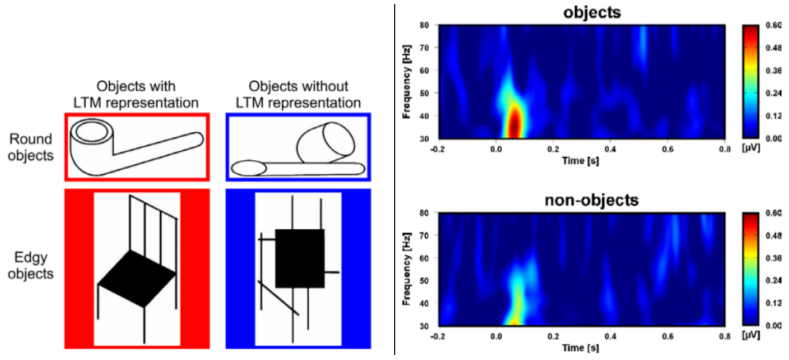

Figure 14: Differential γ-range activity when an object with long term memory representations is recognized (taken from [54]). *Left*: The stimuli presented to subjects. Right: Frequency-representation of single electrode EEG readings averaged over trials. Increased γ activity shortly before 100 ms marks object recognition. Note the highly elevated γ power for objects with long term memory representations.

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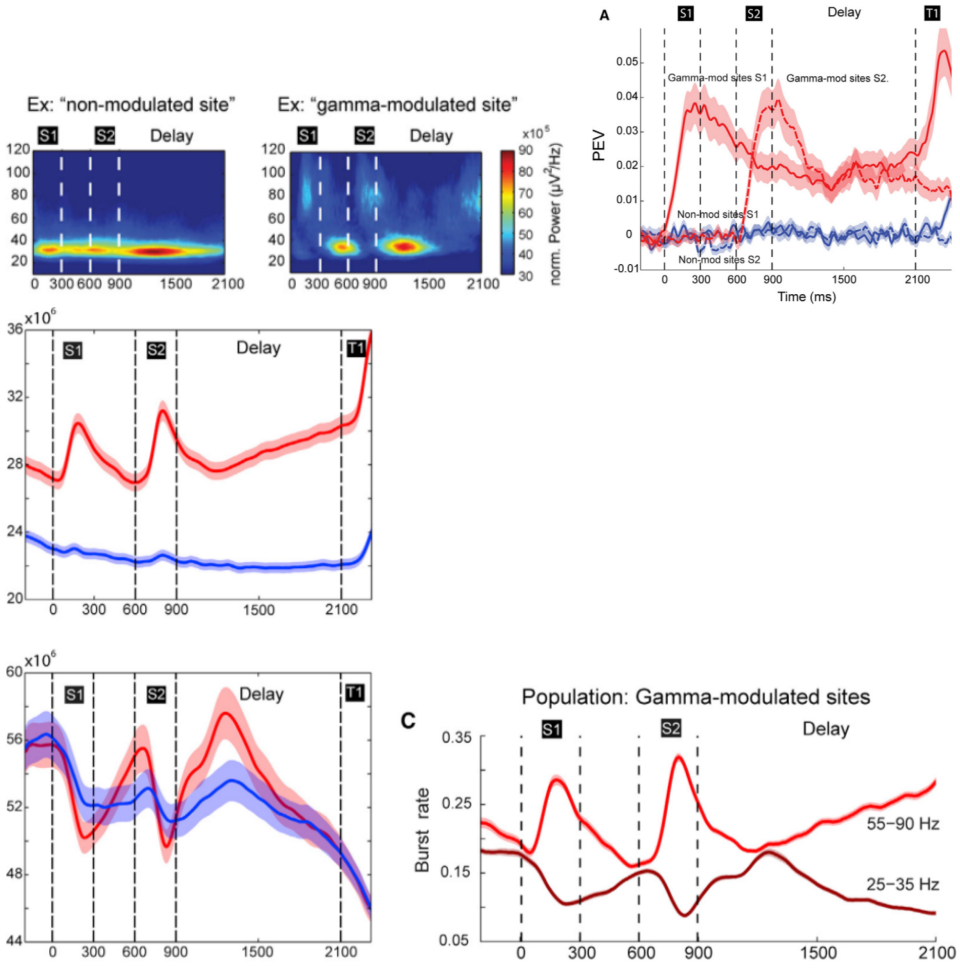

Figure 15: Coordinated temporal interaction between γ and β synchronization in working memory task (taken from [62]) in macaque prefrontal cortex. In each trial, following fixation on the center of the screen two colored squares are presented. Following a delay period, the stimulus is repeated with the color of one square changed. The monkeys are rewarded for a saccade to the changed square. *Top*: power spectrum of EEG recordings during an example trial is shown on the left. On the right, γ-modulated sites are identified by percentage explained variance (PEV) of the spike train by stimulus. *Bottom*: On the left, γ and β power (units of vertical axis are $\mu V^2/Hz$) throughout the trial. On the right, the trends in γ and β power in γ-modulated sites correlates with increased bursting rate at corresponding frequencies (dark red: γ-range burst rate, light red: β-range burst rate. Note that γ-dynamics precede β-dynamics.

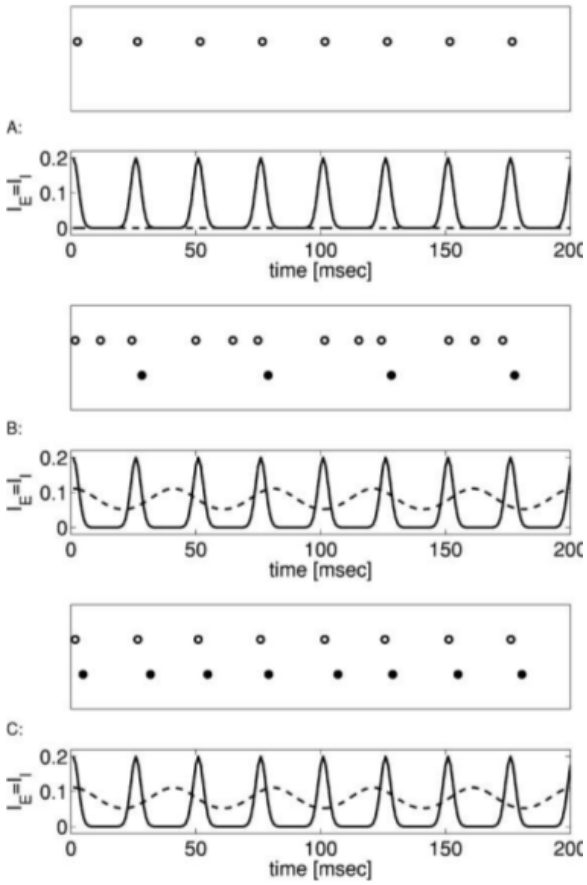

Figure 16: An incoherent distractor can promote its own suppression (taken from [57]). Numerical simulation results for two signals competing to entrain an excitatory-inhibitory network of theta neurons. Odd boxes show the response of the oscillating population with open circles indicating excitatory neuron spikes and dark circles indicating inhibitory spikes. Even boxes show two competing stimuli competing to entrain the population. A: only one stimulus and the inhibitory cell inactivated; population is entrained by stimulus. B: Since the inhibitory cell does not spike, the excitatory cell is left vulnerable to distracting stimuli (dashed line). C: If the distracting stimulus also drives the inhibitory cell it may lead to entrainment of the population to the original stimulus.

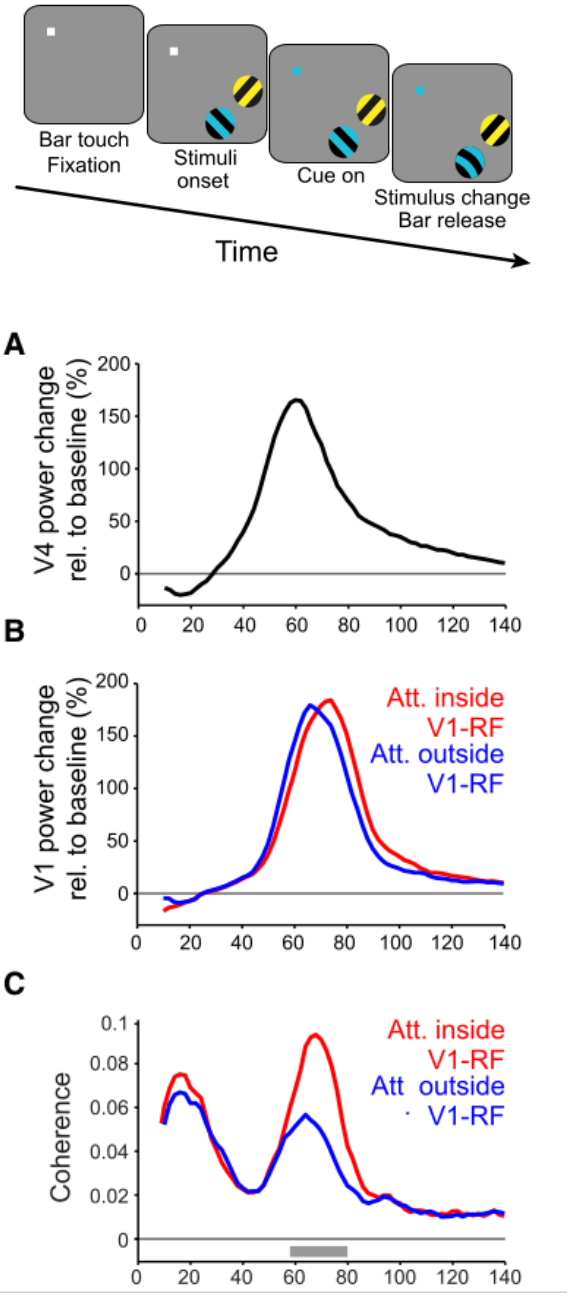

Figure 17: Attention is associated with increased γ-power (taken from [56]). *Top*: Behavioral paradigm. After fixation two stimuli are presented with different colors. Then, the fixation point changes color. The monkey is trained to make saccade to the stimulus with the color cued by the fixation point. Units from V1 and V4 are recorded. *Bottom*: A) Peristimulus V4 spectral power. B) power change relative to baseline in V1 when the stimulus activating the recording site was relevant (same color as cue, red) or irrelevant (blue). C) Spectral coherence between V1 and V4. Notice the elevated γ-power when the stimulus is relevant, i.e attended to.

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