Synchronous oscillatory activity in sensory systems: new vistas on mechanisms

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The origin and nature, as well as the functional role, of synchronous oscillatory activity in the cortex are among the major unresolved issues in systems neurobiology. Recent advances in understanding the mechanisms underlying oscillations include the description of intrinsically bursting pyramidal cells in striate cortex *in vivo* and the discovery of inhibitory interneurons that fire spike doublets to induce synchrony. The behavioral consequences of coordinated activity in cortical neurons remain poorly understood.

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Current Opinion in Neurobiology 1997, 7:536–546

http://biomednet.com/elecref/0959438800700536

Abbreviations

GABA	γ-aminobutyric acid
$GABA_A$	type A GABA receptor
$GABA_B$	type B GABA receptor
NMDA	*N*-methyl-D-aspartate
RE	thalamic reticular (cell)
TC	thalamocortical (cell)

Introduction

Synchronous oscillatory activity has been found at multiple levels of all sensory systems and involves a broad range of frequencies and different behavioral states, but two major issues have attracted the most interest over the past few years. First, it has been suggested that cortical oscillations in the gamma frequency band (20–70 Hz) might be involved in object representation, using the temporal structure to perform binding [1,2]. This hypotheses is still controversial (for different points of view, the reader is referred to [3–6] and references therein). Second, oscillations at lower frequencies are common during the various stages of sleep, and it has been shown that thalamocortical interactions play a prominent role in regulating them [7–9].

In this review, we summarize advances made over the past year in both these fields, focusing first on the mechanisms that generate synchronous oscillations. We will not focus on older contributions as a number of good reviews are available [10–14]. Some relevant results obtained from studies in physics and mathematics are also included.

Mechanisms for generating oscillatory activity

The simplest way to generate collective oscillations is to assume that there are intrinsically oscillating cells that act as pacemakers and entrain an entire network. Alternatively, it is possible that oscillations arise as a network property through a suitable interaction of cells or groups of cells (Figure 1). Both these mechanisms have been investigated recently by experimental and theoretical studies on a variety of systems.

Intrinsically oscillating cells

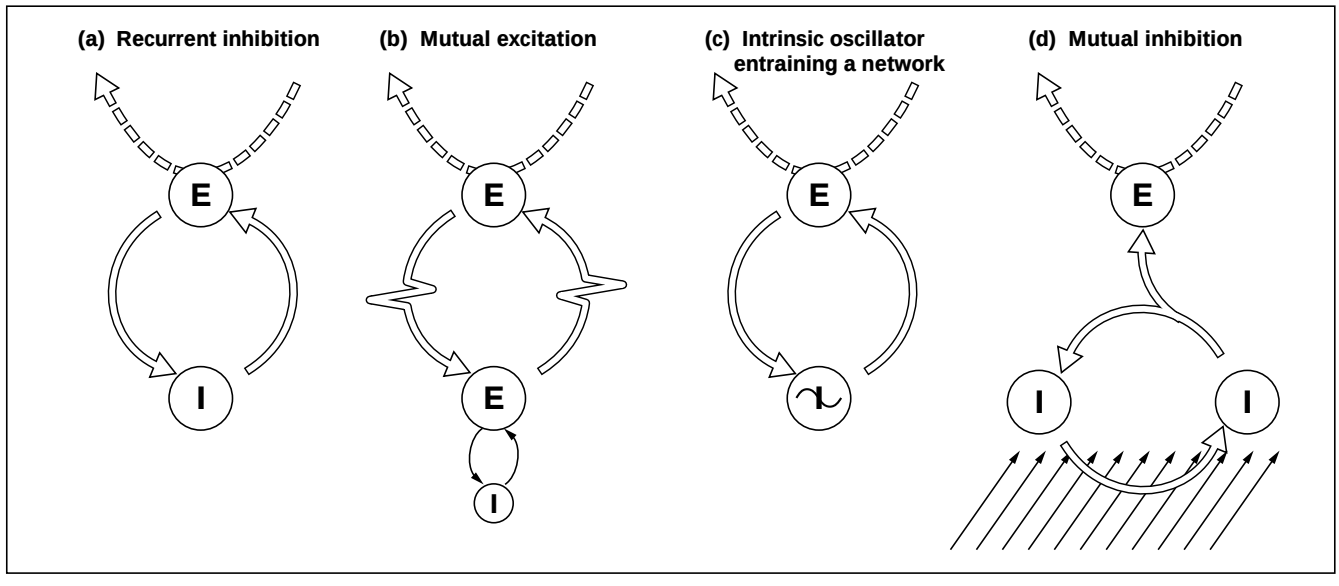
Although many cortical layer 5 pyramidal cells exhibit intrinsic oscillations in the 5–12 Hz range [15], cells that oscillate in the gamma frequency band (20–70 Hz) are less common. Gray and McCormick [16••] have recently reported such cells in intracellular *in vivo* recordings from cat striate cortex, which they called 'chattering cells'. These layer 2/3 pyramidal cells intrinsically generate 20 Hz to 70 Hz repetitive bursts in response to suprathreshold depolarizing current injection. These cells also exhibit pronounced oscillations in the membrane potential during visual stimulation that are largely absent during periods of spontaneous activity, suggesting that chattering cells are involved in the generation of stimulus-driven, collective oscillations.

Recording from slices of rat somatosensory cortex *in vitro*, Flint and Connors [17] found that neocortical neurons in layer 2/3 and layer 5 can independently generate two distinct forms of rhythmic population activity, each dependent upon activation of a different subtype of glutamate receptor. Whereas layer 2/3 cells show kainate-receptor-dependent oscillations from 1 Hz to 5 Hz, layer 5 cells exhibit NMDA-receptor-dependent activity around 8 Hz to 12 Hz.

Using computer modeling, Mainen and Sejnowski [18•] explored the influence of dendritic structure on the intrinsic firing patterns of neocortical cells. They reproduced the entire spectrum of firing patterns observed in the cortex in compartmental models of reconstructed neurons that shared a common distribution of ion channels and differed only in their dendritic geometry. A partial electrical coupling of fast active conductances localized to the soma and axon and of slow active currents located throughout the dendrites were sufficient to cause distinct firing properties, even in a two-compartment model. It is still possible, though, that variability in the types and densities of ion channels throughout the cell could further shape a neuron's intrinsic response properties [19].

Thalamic reticular neurons are involved in the genesis of synchronized thalamocortical oscillations, which depend,

Figure 1



Several mechanisms proposed to be involved in the generation of gamma oscillations. In each case, E (excitatory) and I (inhibitory) represent networks of neurons that are mutually connected, the continuous lines indicate the key connections for their respective mechanisms, and the dot-dashed arrows indicate the flow of specific information through the network. **(a)** Recurrent inhibition [29,34,42,44•]. **(b)** Delayed, mutual excitation [48,49]. **(c)** Intrinsically oscillating cells entraining a network [16••,20]. **(d)** Mutual inhibition model based on tonically excited (thin arrows) interneurons [37,39••,41]. Note that in the last model, the interneurons involved in generating the oscillation are separate from the circuits processing the specific information. Adapted from Figure 2 in [41].

in part, on their complex bursting properties. Destexhe *et al.* [20] showed that a high density of low-threshold calcium current (T current) has to be present in the dendrites of these cells compared to the soma to reproduce the firing pattern characteristics found experimentally *in vitro* and *in vivo*.

Network effects

Using a computer model of ferret thalamic slices, Destexhe *et al.* [21•] and Golomb *et al.* [22] studied oscillations in thalamocortical (TC) and thalamic reticular (RE) cells because of their reciprocal connectivity and bursting properties. TC cells excite RE cells, which, in turn, inhibit the TC cells with a fast ($GABA_A$-mediated) and a slow ($GABA_B$-mediated) component. Because of the presence of a T current, sufficiently strong excitatory input from the TC to the RE cells causes these to elicit a burst of action potentials, and TC cells, in turn, can generate a rebound burst following GABAergic inhibition. Under these conditions, interaction between the TC and RE cells produced sustained spindle oscillations. Only a few cells need to be excited (either TC or RE cells) to trigger a 9–11 Hz oscillation that propagates as a saltatory wave (Figure 2). TC cells *in vitro* display a reduced ability for burst firing after a sequence of bursts. In the model [21•], this was reproduced by assuming an activity-dependent upregulation of the hyperpolarization-activated current via a calcium-binding protein in TC cells. This mechanism was recently confirmed experimentally (D McCormick, personal communication). Suppression of $GABA_A$ inhibition led to slower waxing-and-waning oscillations (3–4 Hz) driven by $GABA_B$ inhibition, which resemble absence (petit mal) epilepsy.

The overall activity pattern changes as soon as one takes the cortex into account as well. Contreras *et al.* [23••,24] have shown that corticothalamic projections cause a global coherence of thalamic oscillations by comparing the spatiotemporal properties of synchronized thalamic spindle oscillations before and after removal of the cortex. In the cortex, synchrony was insensitive to cutting of horizontal intracortical connections, suggesting that corticothalamic projections may serve to synchronize cortical oscillations in distant parts of the cortex through thalamic mechanisms (Figure 3).

Considering the transition from spindling to delta sleep rhythms (1–4 Hz), Terman *et al.* [25] propose that a change in the conductance of one kind of cell effectively rewires the thalamocortical circuit. In their minimal model, they use an increase in the potassium leak conductance of the RE cells to induce the transition; however, in principle, there are many different possible changes in currents that might have the same network effect. Changes in the intrinsic properties of cells can, therefore, cause a functional reorganization of the network dynamics.

In a series of papers, Steriade and co-workers [26–28] presented results on fast (30–40 Hz) spontaneous oscillations in intracortical, corticothalamic, and intrathalamic networks showing, first, that fast oscillations are in phase

Figure 2 legend Spatial patterns of burst discharge during spindle oscillations. **(a)** The spatial activity of a linear network (a locally connected chain of RE and TC cell pairs) is represented as a sequence of snapshots of activity (320 frames with 10 ms between frames) showing a spindle oscillation that starts at the bottom of the chain and travels toward the top. The activity consisted in a series of distinct clusters of activity propagating in the same direction. **(b)** Expanded view of the initiation of the spindle sequence. **(c)** For each snapshot, 50 TC cells and 50 RE cells were displayed vertically as indicated. **(d)** The value of the membrane potential for each neuron was coded using a color scale ranging in 10 steps from –90 mV (blue) to –40 mV (yellow). Reproduced with permission from Figure 12 in [21•].

throughout the cortical depth, and second, that these oscillations are also present during sleep-like activity patterns, challenging the conventional view that high-frequency waves are present only during brain-activated states. They also demonstrated that the intracortical coherency of fast oscillations is coupled with synchronized fast rhythms in corticothalamic circuits. Finally, they showed that the coherence of the fast rhythms is short range whereas low-frequency sleep rhythms exhibit synchronization on a larger spatial scale.

In a three-layer model of the cortex, Fuentes *et al.* [29] analyzed the effects of different axonal arborization patterns on the generation of oscillatory responses. The frequency of the observed oscillations varied and the collective oscillations could be restricted to single layers depending on the branching pattern.

Networks of nonlinear oscillators tend to show only partial synchronization for a wide range of parameters [30,31], and instantaneous excitatory couplings tend to desynchronize rather than synchronize neuronal activity for a broad class of models [32,33]. Thus, inhibitory mechanisms, as well as delays, are important mechanisms for synchronizing neural populations.

Inhibitory mechanisms

Inhibition can be much more effective at synchronizing pyramidal cells than excitatory inputs [34]. This prediction was verified in a hippocampal slice preparation [35] in which single GABAergic interneurons can induce synchronous activity in the theta frequency range (4–7 Hz). Note, however, that GABA at $GABA_A$ receptors in the neonatal rat acts as an excitatory transmitter, but is still involved in synchronizing activity within interneurons of the CA3 subfield of rat hippocampal slices [36].

Inhibitory neurons connected by synapses that activate fast $GABA_A$ receptors are also able to induce synchronized 40 Hz oscillations in rat hippocampal and neocortical slices [37]. The frequency was determined both by the net excitation of interneurons and by the kinetics of the inhibitory postsynaptic potentials between them. This has also been confirmed in a modeling study [38].

On the basis of these findings, Traub and co-workers [39••] simulated a locally connected chain of networks and predicted that when the excitation of interneurons reaches a level sufficient to induce pairs of spikes in rapid succession (spike doublets), the network will generate gamma oscillations that are synchronized on a millisecond time scale from one end of the chain to the other. The model was confirmed in rat hippocampal slices [39••], in which interneurons fire spike doublets under conditions in which gamma oscillations are synchronized over several millimeters, whereas they fire single spikes under other conditions. The implications of this model for some hippocampal network phenomena in the rat have been explored [40] (for a review, see [41]).

Bush and Sejnowski [42] studied synchronization within and between cortical columns using a detailed computer model of sparsely connected excitatory and inhibitory cells. Synchrony was found to be sensitive to the strength of reciprocal inhibition between the inhibitory neurons in one column. Too weak or too strong reciprocal inhibition degraded intracolumnar synchrony. The only parameter that affected the oscillation frequency of the network was the strength of the external driving input. Synchrony between columns with 3% connectivity could only be established when connections from the pyramidal cells in one column to the inhibitory cells in the other column were included.

Extending leaky integrate-and-fire networks to include the effects of hyperpolarization-activated inward currents as well as intrinsic synaptic and threshold noise, Coombes and Doole [43] found that rebound currents are important for central pattern generation in neuronal circuits with inhibition. Moreover, rebound currents were shown to suppress chaotic network responses to external input, in favor of low-order periodic responses.

In a combined modeling and experimental study, Tsodyks *et al.* [44•] considered a population of excitatory principal cells with recurrent connections and strong interactions with inhibitory interneurons. They found that increasing an external inhibitory drive onto the inhibitory interneurons — without directly affecting any other part of the network — can, in some circumstances, cause the interneurons to increase their firing rates. To achieve this effect, recurrent connections among the excitatory cells have to be strong enough to make the excitatory network unstable when feedback inhibition is removed. These considerations can be extended to periodically varying input, where systematic relationships between the phase shift and depth of modulation for each interneuron can be predicted. These predictions were tested and confirmed

Figure 2

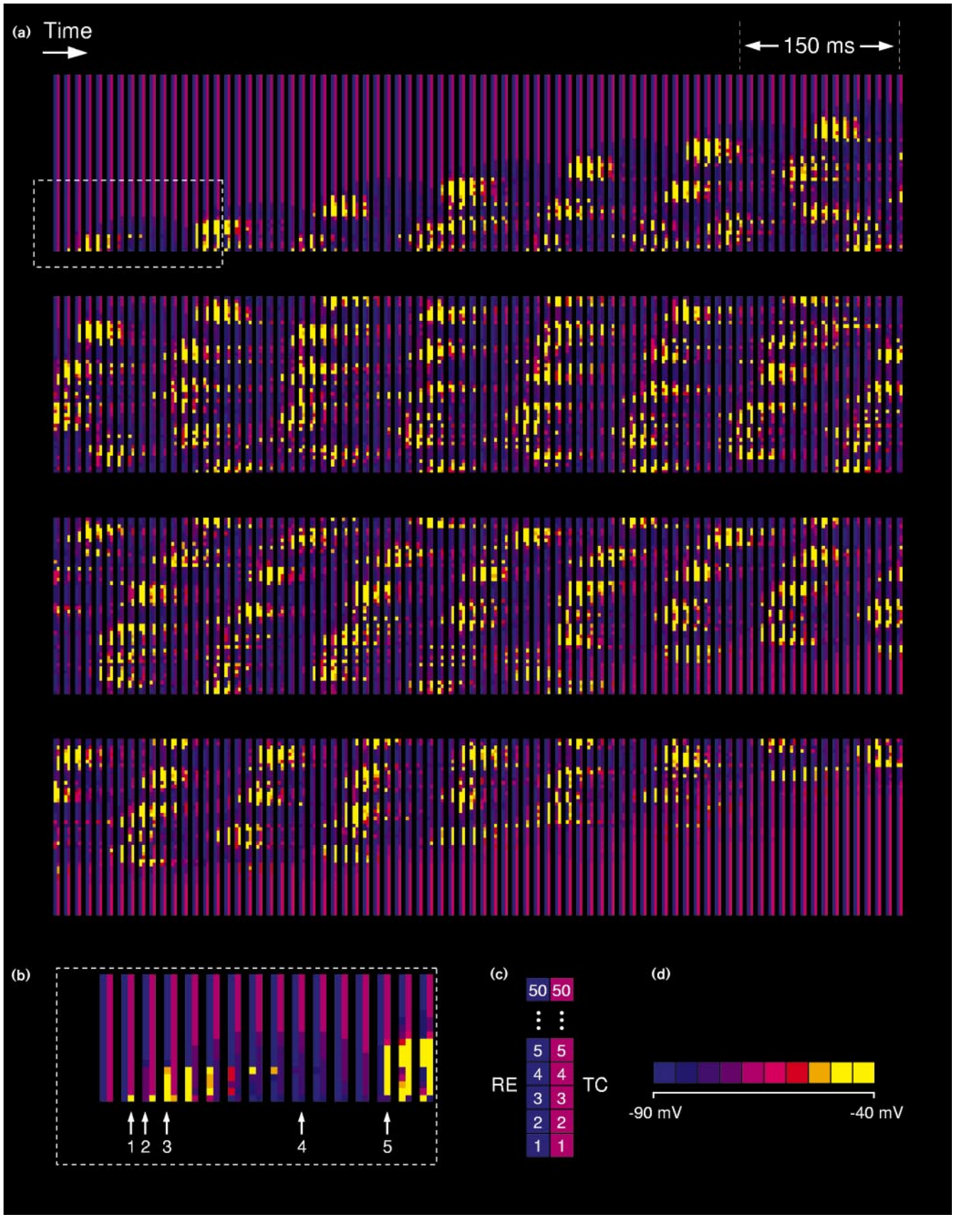


Figure 3 legend Cortical coherence of spindling under barbiturate anesthesia does not depend on intracortical connections. **(a)** Spontaneous spindle oscillations were recorded from the depth (~1 mm) of the suprasylvian cortex by means of eight tungsten electrodes – dots numbered 1–8 in **(b)** – with interelectrode distances of 1 mm. (a) Spindling sequences in the raw data (cortical leads *1–8*) showed frequencies at 7–9 Hz, lasted 2–5 s, and recurred every 2–7 s, almost simultaneously in all electrodes. **(d)** Cross-correlations between electrode *1* and each of the others, for consecutive individual spindles, were averaged (CROSS, Before cut; traces were displaced horizontally and vertically for clarity) and showed central peak values decreasing from 0.9 (*1–2*) to 0.5 (*1–8*). **(e)** After cut shows averaged cross-correlations calculated after a deep cut between electrodes *4* and *5*, as indicated by the black line in (b), that crossed from the marginal gyrus to the ectosylvian gyrus. **(c)** The histology of the cut is shown in a parasagittal section along the suprasylvian gyrus (anterior at left); some tracks of recording electrodes are also seen. After the transection, cross-correlations showed a similar decrease in central peak from 0.9 (*1–2*) to 0.5 (*1–8*). Correlations *1–4* and *1–5* were flat because of the local lesion produced by the cut. The most likely mechanism leading to synchrony was corticothalamic interactions. Reproduced with permission from Figure 2 in [24].

by recordings from interneurons in the CA1 region of the rat hippocampus *in vivo* [44•].

Delay-induced synchronization

Even a system undergoing a random walk can show oscillatory correlation if there is a delay [45]. Delays are difficult to manipulate in neuronal systems, so most studies, so far, have been theoretical ones.

One of the simplest network models with stable attractors is the Hopfield model, which has symmetric connectivity [46,47]. Delay-induced oscillations are possible in a Hopfield network when the network is frustrated, such as when there are many ground states [48]. In a three-neuron model, the oscillations occur through a supercritical Hopf bifurcation, unfolding a codimension-two bifurcation [48].

Pulse-coupled oscillators with delayed excitatory couplings of almost arbitrary structure have been shown to converge to a periodic solution where all neurons are phase-locked but not necessarily firing in unison [49]. In the case of discrete and uniform delays, a periodic solution is reached after a finite time. For a continuous delay distribution, a periodic attractor is, in general, only reached asymptotically.

A general framework for analysis

What neuronal characteristics are essential to ensure that coherent oscillations are asymptotically stable? Gerstner *et al.* [50••] addressed this question for spatially homogeneous networks of spiking neurons. They proved a theorem stating that a necessary and (in the limit of a large number of interacting neighbors) sufficient condition is that the postsynaptic potential increases in time as the neuron fires. This depends on a subtle interplay of axonal delays, postsynaptic potentials (excitatory as well as inhibitory), and refractory behavior. This locking theorem provides a simple geometric method to verify the existence and local stability of a coherent oscillation for a variety of models.

Modulation by subcortical projections

Whereas most experimental evidence suggests that gamma frequency oscillations in cortical areas are generated there, it has also been shown that subcortical areas such as the pulvinar [51] or the mesencephalic reticular formation [52] can act to facilitate stimulus-specific synchronization in the visual cortex of cats. Similar results have been obtained in the auditory system of rats [53,54]. Nuñez has proposed that 0.5–2 Hz rhythmic activity of cortical neurons during slow wave activity is enhanced as a result of bursting basal forebrain neurons. He suggests that they are rhythmically releasing acetylcholine in the cortex [55]. However, these cells could be GABAergic projection neurons [56] and could have a role similar to the GABAergic neurons in the medial septal nucleus that project to the hippocampus [44•].

Information processing based on synchronous oscillations

New evidence has been reported for stimulus-induced oscillations and synchronous firing in cortical neurons. Singer and colleagues (see [57•,58,59•]) have found stimulus-dependent synchronization of neuronal responses in the visual cortex of the awake macaque monkey [57•] and long-range synchronization of oscillatory responses in the cat retina and lateral geniculate nucleus [58], which depend on global stimulus properties such as size and continuity. This suggests that temporal correlations among responses of spatially segregated ganglion cells can already be exploited to convey information relevant for perceptual grouping [58]. Furthermore, Roelfsema *et al.* [59•] have shown that synchronization occurs both between areas of the visual and parietal cortex and between areas of the parietal and motor cortex in the awake cat. When cats responded to a sudden change of a visual pattern, the neuronal activity in these cortical areas exhibited synchrony without time lags. This synchrony was found to be particularly strong between areas subserving related functions (see Figure 4).

Kruse and Eckhorn [60] studied the suppression of sustained gamma oscillations by fast transient responses in cat visual cortex. They found that fast stimulus-locked components were primarily evoked by sudden stimulus accelerations, whereas oscillatory components were induced during slow, smooth movements. Oscillations were gradually reduced in amplitude and finally fully suppressed with increasing amplitudes of fast stimulus-

Figure 3

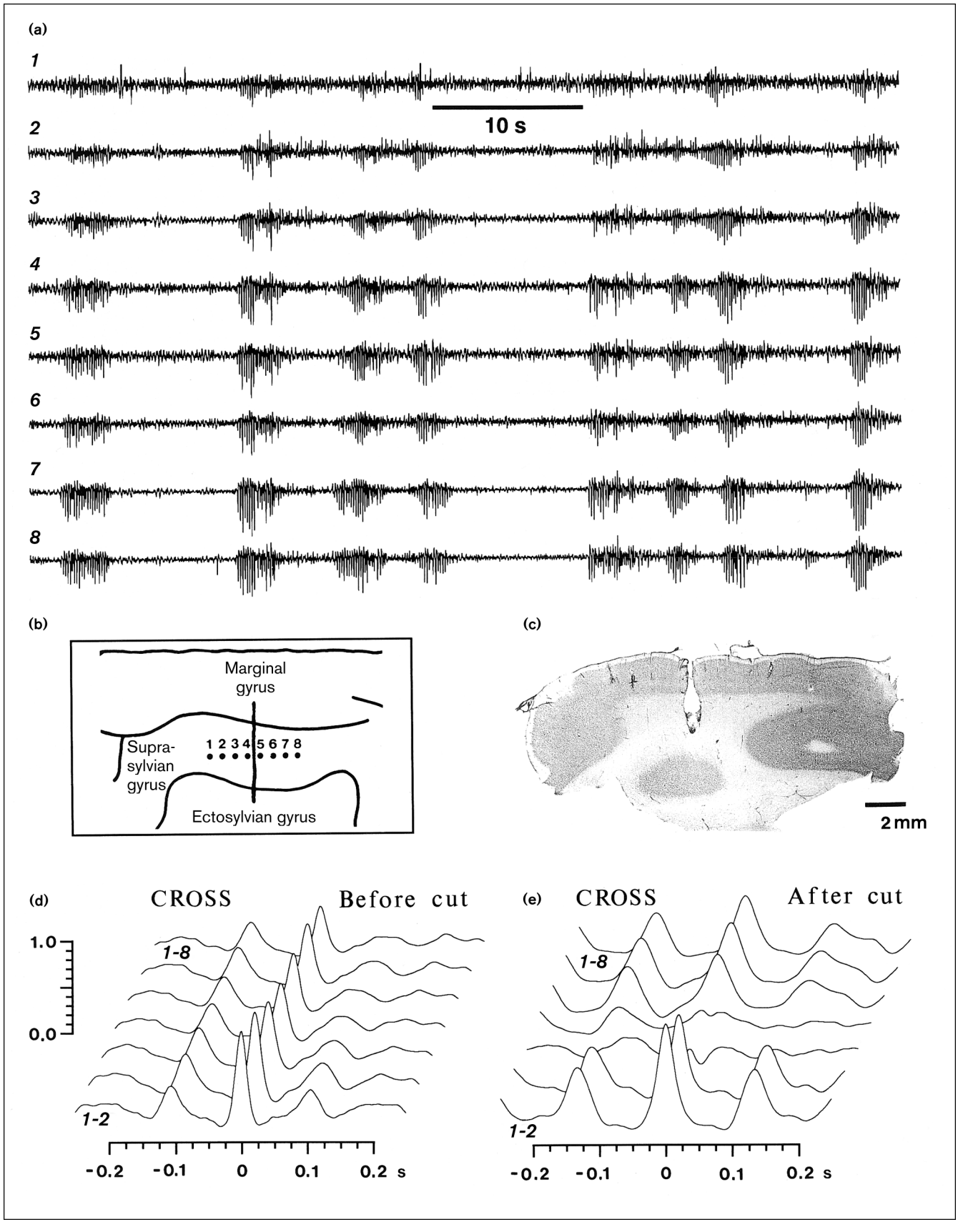

(a)
1
2
3
4
5
6
7
8
10 s
(b)
Marginal gyrus
Supra-sylvian gyrus
1 2 3 4 5 6 7 8
Ectosylvian gyrus
(c)
2 mm
(d)
CROSS
Before cut
1.0
0.0
1-8
1-2
-0.2
-0.1
0
0.1
0.2 s
(e)
CROSS
After cut
1-8
1-2
-0.2
-0.1
0
0.1
0.2 s

Figure 4 legend Patterns of interactions among areas of the visual, parietal and motor cortex of cats during a task in which a rapid response was required to receive a liquid reward. **(a)** Correlation functions among areas of the left hemisphere of one of the cats while it was waiting for the rotation of the grating, averaged over trials. In all cats, recordings from somatosensory and motor areas were made in the hemisphere contralateral (c) to the paw that the animal used for pressing a lever. l, lateral; m, medial. **(b)** Dependence of the correlation functions on the type of visual stimulation. (i) Correlation functions obtained during visual stimulation with a stationary grating patch. Top row, correlation between field potentials in visual areas 18 and 21, ipsilateral (i) to the paw used by the cat. Bottom row, correlations between field potentials in the lateral (5l) and medial (5m) subdivision of area 5 contralateral (c) to the paw used by the cat. (ii) Correlation functions obtained during stimulation with a moving grating within a larger aperture. Synchronization between areas 18 and 21 was stronger during stimulation with the moving grating than during stimulation with stationary grating. The strength of synchrony between the two subdivisions of area 5 was not affected by this change of the visual stimulus. (iii) Location of all recording sites ipsilateral and contralateral to the paw used by the cat. **(c)** Synchronization strength among the areas from which recordings were obtained during the task period, averaged across cats. Lines between the areas indicate the strength of synchrony. Areas that are not connected did not exhibit significant synchronization in any of the cats. Areas in the hemisphere contralateral and ipsilateral to the paw used by the cats are shown in the top and bottom row, respectively. Note that the interhemispheric asymmetry in the diagram reflects an asymmetry in the electrode placement, not in the pattern of synchrony. **(d)** (i) Distribution of time lags in the correlation functions. Grey bars show time lags when the cats waited for the rotation of the grating, black bars show time lags during the reward epoch. (ii) Distribution of integrated power in the alpha frequency range (7–13 Hz) when the cats waited for the rotation of the grating (grey bars) and in the reward epoch (black bars). Reproduced with permission from Figure 2 in [59•].

locked components. The authors argue that suppression of oscillations is necessary to prevent confusion during sequential processing of rapidly changing retinal images. In another study, Brosch *et al.* [61] tested the classic Gray *et al.* [62] result on different correlation patterns for a long bar versus two distinct bars moving either in the same or in different directions. They confirmed previous results [62] using bars and gratings for stimuli while recording from cells with non-overlapping receptive fields in areas 17 and 18 of cat visual cortex. Coherences of oscillations were found to be significantly higher for a whole-field grating or a long bar moving across both receptive fields of the recorded neurons compared to other stimuli.

Investigators in other laboratories have confirmed and extended these findings. Livingstone [63] has reported stimulus-enhanced correlations and oscillations in squirrel monkey striate cortex: layer-dependent delays were revealed by correlation peaks, providing evidence for cross-talk between magnocellular and parvocellular streams.

Oscillations have also been found outside the visual cortex. Murthy and Fetz [64•,65•] made an extensive study of oscillatory activity in the sensorimotor cortex of awake monkeys. They examined synchronization of local field potentials as well as single neurons in relation to various behavioral paradigms. Their results suggest that neurons across wide regions can become transiently synchronized specifically during local field potential oscillations, even if their spikes are uncorrelated during non-oscillatory periods. Episodes of 20 Hz to 40 Hz oscillations occur often and become synchronized over a large cortical area during exploratory forelimb movements. However, they found no reliable relation between the presence of oscillations and particular components of the movement. Synchronization also did not occur preferentially for co-activated, task-related neurons, suggesting that synchronous oscillations may not play an obvious role in sensorimotor association or binding. The authors proposed that synchrony might instead reflect an attention- or arousal-related mechanism that facilitates associations between widely distributed populations.

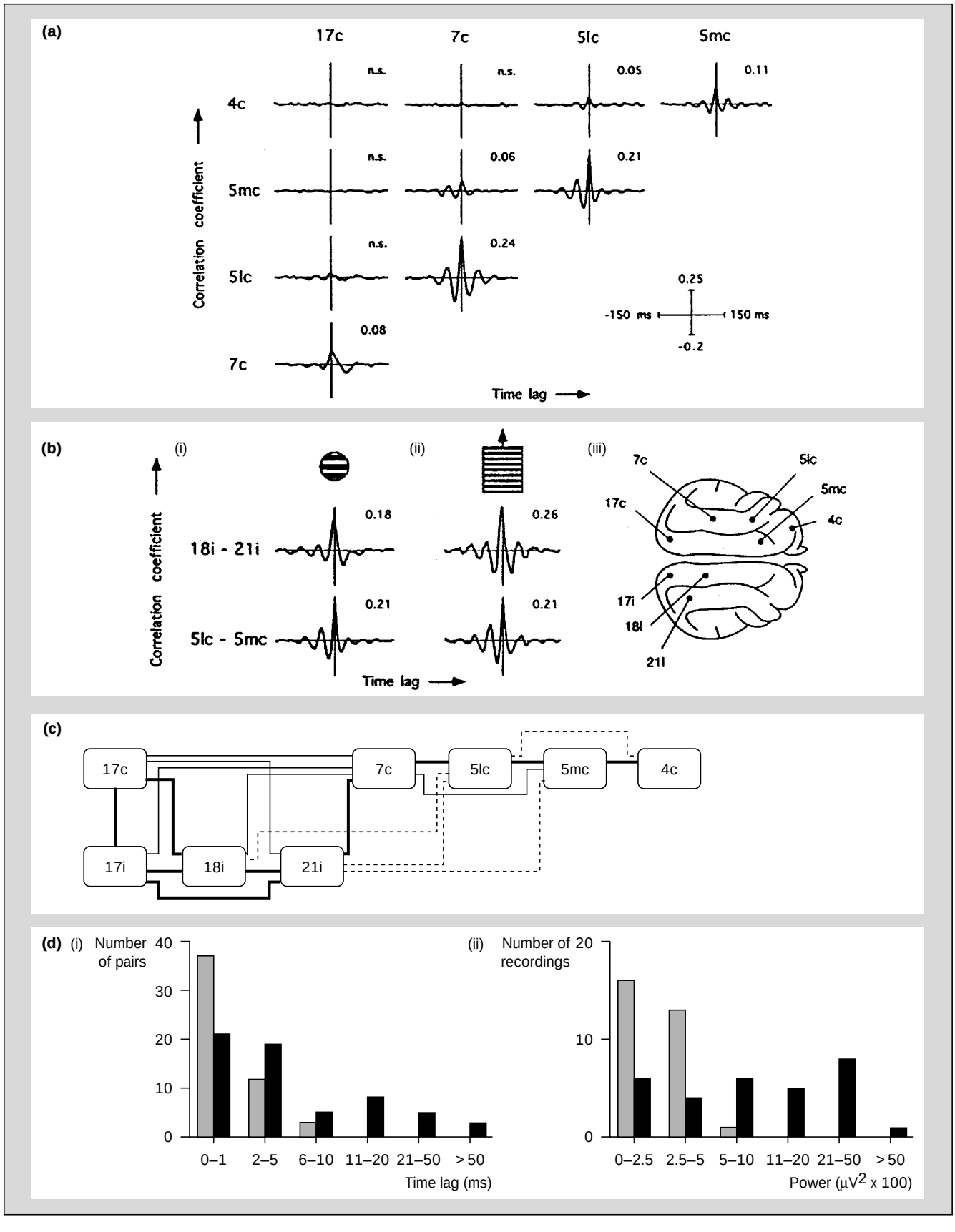
An attentional mechanism is consistent with recent results on oscillations during express saccades. Kirschfeld *et al.* [66] have proposed that the control of eye movements may involve cortical oscillations. Several other studies have questioned the importance of temporal structure for solving the binding problem [67,68]. (For a review on the generation and functional role of oscillations in the olfactory system, see Laurent, in this issue, pp 547–553.) Carandini *et al.* [69] have suggested that one function of gamma frequency oscillations might be to broaden and linearize the spiking responses of neurons to stimulus-related frequencies.

Functions for synchronous firing

If synchronized activity among different cells were to carry information in addition to that in their mean firing rates, then there should be neurons that detect synchronous firing — that is, neurons that act as coincidence detectors. On the basis of established biophysical properties of cortical neurons and on particular features of cortical dynamics, König *et al.* [70•] have argued that coincidence detection rather than temporal integration might be a common mode of operation for cortical neurons. This would allow temporal codes to be converted into firing rates.

Another issue that arises for the binding hypothesis is the problem of spatial scale. If an image is segmented into objects, these objects can typically be segmented further into their constituent parts. Roelfsema *et al.* [71] have proposed that the synchronization behavior of neurons that represent the various image components may accurately reflect this hierarchical clustering. They further suggest that the range of synchronizing interactions is a dynamical parameter of the cortical network, such that the grain (scale) of the resultant grouping process may be adapted to the behavioral requirements.

Figure 4


(a)
17c
7c
5lc
5mc
4c
5mc
5lc
7c
Correlation coefficient
n.s.
0.05
0.11
0.06
0.21
0.24
0.08
0.25
-150 ms
150 ms
-0.2
Time lag
(b)
(i)
(ii)
(iii)
18i - 21i
5lc - 5mc
0.18
0.26
0.21
0.21
Correlation coefficient
Time lag
7c
5lc
5mc
17c
4c
17i
18i
21i
(c)
17c
7c
5lc
5mc
4c
17i
18i
21i
(d)
(i) Number of pairs
40
30
20
10
0
0–1
2–5
6–10
11–20
21–50
>50
Time lag (ms)
(ii) Number of recordings
20
10
0
0–2.5
2.5–5
5–10
11–20
21–50
>50
Power (μV2 x 100)

Locally excitatory, globally inhibitory oscillator networks could perform image segmentation and figure/ground segregation where the phases of the oscillators encode the binding of pixels. This has been demonstrated on gray-level images of aerial views and magnetic resonance images of a human head [72].

Different parts of an object may be processed by the brain at different rates and may thus become desynchronized. Grossberg and Grunewald [73] have proposed a process called perceptual framing that resynchronizes cortical activities corresponding to the same external object.

An alternative approach to oscillatory function is proposed by Parodi *et al.* [74]. If spike arrival times were to be used for coding stimulus features, then moving targets would tend to confuse any reasonable decoding scheme. A resetting process is therefore needed, and this could result in synchronized, quasi-periodic spike firing. Visual motion induces synchronous oscillations, even in the visual area of the primitive three-layer cortex of reptiles [75], and as all the experiments on cats and monkeys have used moving targets, it may be that oscillations are somehow related to motion processing.

Synchronous and oscillatory brain activity, as reviewed here, are only two examples of temporal coding; others include relative time delay codes [76–78] and correlated activity not involving oscillations ([79–82,83•,84]; R Ritz, TJ Sejnowski, *Soc Neurosci Abstr* 1996, 22:1622). An issue of major importance for implementing temporal codes, not dealt with here, is the sensitivity of synaptic plasticity to precisely timed neuronal signals. As shown recently [85–87], the sign of synaptic change is sensitive to the relative timing of pre- and postsynaptic activity at the millisecond level. In addition, Amzica *et al.* [88] have shown that instrumental conditioning can locally increase fast oscillations in corticothalamic networks. Thus, temporal codes may be used by the brain to store and to process information.

Stimulus specificity of 40 Hz signals has also been found in human EEG studies [89], and an interesting suggestion for how we perceive time has been made by Andrews *et al.* [90]. On the basis of their results from flicker-fusion experiments, they speculate that we parse visual input into discrete episodes. This may be related to how the human visual system is able to recognize objects so rapidly [91].

Multiple photon microscopy, a promising new technique developed over the past few years, allows *in vivo* imaging with simultaneous high spatial and high temporal resolution [92,93]. This approach opens a new avenue to the study of the temporal mechanisms underlying neuronal function *in vivo*.

Conclusions

Synchronous oscillatory activity in the gamma band occurs in both awake, alert states of the brain, as well as during sleep states. Networks of inhibitory neurons may control the spatial organization of synchronous firing in cortical and hippocampal neurons, although evidence for neurons that intrinsically burst at these frequencies has also been found. Major progress has also been made in understanding the origin of oscillations at lower frequencies during sleep states, whose spatiotemporal patterns are organized by reciprocal interactions between the cortex and the thalamus. These advances provide a foundation for exploring the functions of these brain oscillations in coordinating coherent patterns of activity widely distributed within motor as well as sensory systems.

Acknowledgements

R Ritz is supported by the Deutsche Forschungsgemeinschaft and TJ Sejnowski by the Howard Hughes Medical Institute. We thank Mircea Steriade for a critical reading of the manuscript.

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- •• of outstanding interest

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