Trends in Cognitive Sciences

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Feature Review

Processes and measurements: a framework for understanding neural oscillations in field potentials

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Various neuroscientific theories maintain that brain oscillations are important for neuronal computation, but opposing views claim that these macroscale dynamics are 'exhaust fumes' of more relevant processes. Here, we approach the question of whether oscillations are functional or epiphenomenal by distinguishing between measurements and processes, and by reviewing whether causal or inferentially useful links exist between field potentials, electric fields, and neurobiological events. We introduce a vocabulary for the role of brain signals and their underlying processes, demarcating oscillations as a distinct entity where both processes and measurements can exhibit periodicity. Leveraging this distinction, we suggest that electric fields, oscillating or not, are causally and computationally relevant, and that field potential signals can carry information even without causality.

Highlights

Brain oscillations are ubiquitous in neural recordings across species and brain areas.

There is substantial evidence that these rhythmic fluctuations correlate with cognitive functions, such as attention, memory, decision-making, and action.

However, controversy remains over their causal status: to what extent are oscillations responsible for changing cognitive and behavioral states?

A distinction between electrophysiology measurements and processes clarifies the dispute and moves inquiry into the empirical domain.

Field potentials as signals offer information about brain and cognitive states, while the electric fields that underpin such measurements have a multitude of causal effects.

Oscillations are hypothesized to facilitate the self-organized orchestration of neuronal computation, influencing how information is processed across ensembles.

Two perspectives on oscillations

Neuroscientists have long observed that aggregate neural signals, measured via electroencephalography (EEG), magnetoencephalography (MEG), and **local field potential (LFP)** (see Glossary) recordings, show regular fluctuations at characteristic frequencies [1,2]. The presence and characteristics of these **oscillations** correlate with various aspects of cognition and behavior [3–7], suggesting that they have a causal role in neural information processing. This hypothesis has given rise to the view that oscillations act as coordinators or orchestrators of brain activity [8,9].

A competing view is that oscillations, and electric field potentials more broadly, are merely byproducts of neural computation. Under this alternative, oscillations are not functional but are instead **epiphenomenal**: they passively reflect neuronal spiking without influencing neuronal computation itself. Resolving the tension between these two perspectives has important implications for the explanatory primacy of spikes versus aggregate macroscale brain activity in theories of cognition [10], as well as the merit of frameworks that grant brain oscillations an active role in neural computation [11].

Here, we outline how the question of whether oscillations and LFPs are epiphenomenal is ill-defined. Specifically, each concept in this question (oscillations, LFPs, and epiphenomenal) has multiple meanings. As a result, they should be disentangled for testable hypotheses to emerge. To resolve this, we outline a conceptual framework that reformulates the question into a set of more precise alternatives that we hope make disputes on the importance of neural signals more empirically tractable. Furthermore, we offer preliminary answers to the reformulated questions using experimental and computational evidence.

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This contribution unfolds as follows. First, we introduce a distinction between **processes** and **measurements**, which we use to classify key concepts in the electrophysiology literature. Second, we introduce two criteria that can be used to evaluate the relationships between concepts: (i) the **inference criterion**: which entity is informative toward which other entity? and (ii) the **causality criterion**: which entity is causal toward which other entity?

Leveraging this framework, we then consider the links between LFPs, neurobiological processes, cognition, and behavior. Oscillations are then considered separately in light of their unique causal and inferential role.

Measurements and processes

In scientific practice, it is important to distinguish between measurements and processes. Process refers to physical objects and events in the world, which are the targets of our investigations; measurements refer to the signals or recordings we use to capture information about such objects and events.

Examples of processes in neuroscience are neurobiological events across spatiotemporal scales, including action potentials, molecular cascades, and other dynamic interactions between neural structures occurring from the molecular scale to the scale of anatomical regions [12] and distributed networks [13]. Furthermore, behaviors, such as motor activity, are also physical processes with possible causal effects.

Measurements qualitatively or quantitatively capture processes with the goal of generating scientific inferences about those processes. Here, we use measurement in its noun form (i.e., roughly synonymous with terms such as signal or recording) rather than as a verb, which refers to the act of measurement[I]. While one might argue that measurements are themselves processes, they are privileged from the perspective of scientific investigation in that they are processes designed and used for inference about other processes, rather than being objects of study themselves.

The primary term of interest pertaining to the ill-defined question raised in the Introduction is the electric field potential, which we categorize as a measurement. Specifically, the field potential refers to a signal that quantifies the voltage difference between an electrode at a location of interest and a spatially separate reference electrode [14]. Such potentials can be local or global. LFPs are recordings in extracellular space using electrodes inside the brain. **Global field potentials** are recordings using non-invasive techniques, such as EEG, or magnetically using MEG, which capture the interaction of many sources in the brain simultaneously. We might also add a mesoscale for intermediate levels, such as subdural recordings (electrocorticography; ECoG).

For LFP recordings, it is common to separate the raw signal into two components. The first component comprises frequencies <1000 Hz, which usually retains the same name of LFP (which might cause terminological ambiguity in its own right). The second component comprises frequencies between ~300 and 3000 Hz, which are then processed for spike detection and inferences about single-neuron action potentials.

Thus, extracellular **spikes** and LFPs are signals that originate from the same voltage recordings, filtered to isolate the respective process that stereotypically (but not necessarily) underpins each. Specifically, spikes tap into action potentials, and local and global field potentials reflect aggregate **electric fields**, which are charged particles moving through space (such as across neuronal membranes). Both action potentials and electric fields (henceforth 'e-fields') are processes that can, in principle, exert causal effects on subsequent neural events, but measurements thereof

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cannot. One intuitive way to parse the distinction between measurement and process is to consider the role of reference electrodes: the location and physical properties of reference electrodes, and the chosen referencing scheme, change measurements [15–17]; however, e-fields, action potentials, and other physical processes are present and the same regardless of whether there are electrodes placed to measure them. For the remainder of our analysis, we focus on LFPs, with most of the conclusions carrying over to global field potentials unless stated otherwise.

Disentangling inference and causality

We next introduce two criteria: to what extent are LFPs, oscillations, and e-fields causally relevant, and to what extent are they inferentially informative?

The inference criterion is epistemic. Here, the question is whether information about one entity reduces uncertainty about the state of another. For example, if we see oscillations in the LFP at a specific frequency band, does this lower our uncertainty about which cognitive processes are currently active or which neurobiological processes are ongoing? Even if no direct causal link underpins any two processes, a measurement of one may still confer information about the other.

The causality criterion considers whether any one entity influences another. For the present purpose, we define a causal relationship as one where interventions on one entity can, in principle, produce changes in another (see [18–21] for relevant discussions).

Together with measurements and processes, the distinction between inference and causality offers a disambiguation tool that we can use to guide our analysis, setting the stage for further inquiry. Given this, we first evaluate the causal and inference criteria of the e-field and the LFP in relation to neurobiological processes and behavior, regardless of the specific temporal dynamics at play. After that, we turn to oscillations specifically, which can occur in both measurement and process, and which are poised to serve a general computational role in the brain.

Theoretical status of local field potentials and electric fields

Here, we introduce a roadmap to understanding the relationship between various key terms in the electrophysiology literature, evaluating their links via the criteria of causality and inference. Figure 1 provides an overview of our conceptual framework. Below, we unpack this roadmap and evaluate the questions it yields, starting with uncontentious ones and moving to increasingly complex relations.

Are LFPs causal toward any entity?

No: the LFP is a measurement, which does not causally influence its underlying (or any other) process.

Are electric fields causal to the LFP? (Figure 1; arrow 1)

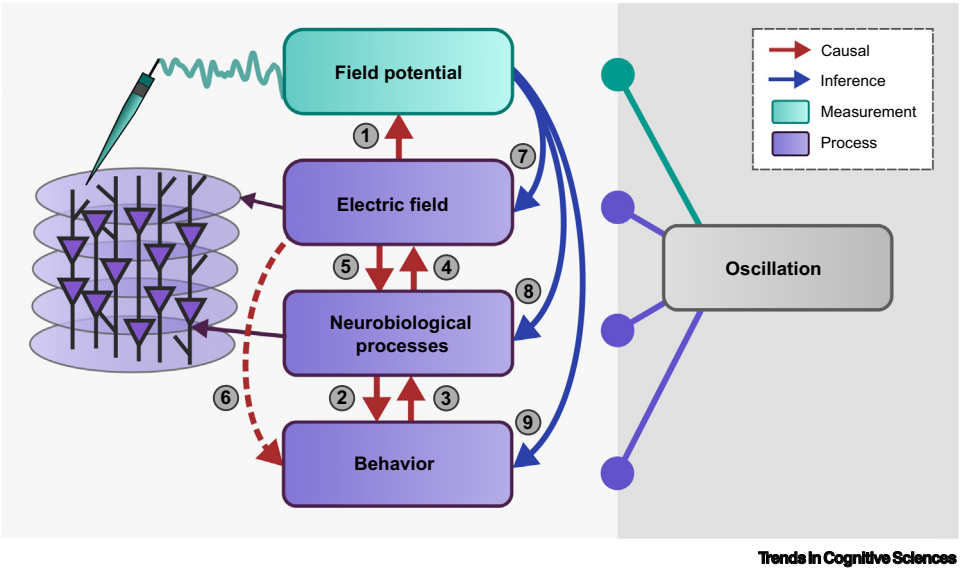
Yes: as discussed above, changes in extracellular e-fields sum up locally or globally to produce changes in microwires, EEG, or ECoG electrodes, and produce magnetic fields that are detected using MEG. This explains how the field potential signals come about. More technically, the negative gradient of the electric potential in any direction comprises the e-field component in that direction.

Are neurobiological processes causal to behavior and vice versa? (Figure 1; arrows 2 and 3)

Yes: axiomatic to the neurosciences, neurobiological processes influence behavior, either directly through efferent projections to the peripheral nervous system, or indirectly through long-lasting structural or functional changes that encode previous experience and future expectations. Conversely, behavior shapes sensory input available to the organism through, for example, active sensing (e.g., whisker movement) and bodily changes, thereby causally affecting neural processes.

Glossary

Causality criterion: process X is causal toward another process, Y, if there is a possible intervention on X that produces a change in Y, such as when an experimental lesion of a brain region alters downstream neuronal activity.
Electric field: physical field of charged particles, the movement of which (a process) is captured by field potential recordings (a measurement).
Ephaptic coupling: process in which extracellular e-fields influence neurons, in particular, their membrane voltage potential, via non-synaptic and non-gap junction means.
Epiphenomenon: events caused by processes in a system but that do not exert causal effects on the system itself, such as the steam whistle of a locomotive relative to its movement.
Field potential (local or global): voltage recordings of electric fields measured locally in extracellular space (LFP) or globally from masses of activity (EEG, or detection of their magnetic fields using MEG). All field potentials are measurements.
Inference criterion: measurement is inferential or informative of a process if it constrains our knowledge of what state that process is in.
Measurement: recording or signal that captures information about a physical process, offering a qualitative or quantitative indication of changes to this process (e.g., a time series of voltage values).
Oscillation: as a measurement: periodic structure in the LFP, commonly detected by bandpass filtering or observing peaks in the power spectrum. As a process: periodic structure in the electric fields, neurobiological processes (e.g., synaptic events), behavior, or any other process in the brain resulting from an oscillator mechanism (for details, see 'Inferring oscillations-in-process from oscillations-in-measurement' in the main text).
Process: physical events in the world that can be observed through a measurement (e.g., the movement of ions and proteins, but we also include static entities such as an axon).
Spike: as a measurement: sharp deflections in the high-pass filtered LFP, lasting ~1 ms, often with a stereotypical waveform across neighboring recording channels. As a process: an action potential, or the rapid change in electrical potential associated with the passage of



an impulse along the membrane of a neuron, caused by the opening and closing of sodium and potassium channels.

Figure 1. A conceptual roadmap for electrophysiology terms. In this framework, we separate four primary entities of interest. At the top, local and global field potentials measure e-fields without themselves exerting any causal effects on the brain (cyan). One step down, the e-fields captured by this measurement are produced by neuronal activity and other electrochemical processes, and constitute a physical process (purple). In turn, neurobiological processes produce behavior and might be influenced by e-fields. Furthermore, each of the entities can display the dynamical property of periodicity, instantiating a type of oscillation (right-hand side), a point returned to in 'Theoretical role of oscillations' and Figure 2 in the main text. Arrows denote directions of causality (red) and informativeness (blue), and the numbers next to arrows are referred to below. We make particular note of the **indirect or mediating** causal relationship (dashed arrow 6) between e-fields and behavior due to its prominence in related discussions (see 'Do neural e-fields causally influence behavior?' in the main text).

Are neurobiological processes causal to e-fields? (Figure 1; arrow 4)

Yes: e-fields in the brain are both directly generated and indirectly shaped by neurobiological processes [22]. First, as action potentials are initiated and propagated, a cascade of ion flow changes electric gradients across neuronal segments. Second, neurotransmitter binding at postsynaptic sites opens membrane channels (e.g., chloride), with the associated charged ion movement altering the e-field. Third, neuronal burst firing is associated with slow calcium fluctuations [23,24], which can change e-fields on a longer timescale [25]. It appears that the largest driver of extracellular e-fields are events near the synapse, as well as synchronous action potentials [15,16,26,27]. However, the exact distribution of causes is not fully mapped out [15].

Besides the generation of e-fields, many neural events indirectly influence e-fields. For example, the morphology of nearby neurons, glial cells, and the electrical conductance of surrounding tissue affect the spatiotemporal structure of nearby e-fields [28,29]. Together, these amount to biological filters that shape the e-field and the LFP measurements of it. While the empirical relations discussed up until this point should be subject to minimal controversy, the extent and mechanisms by which e-fields influence neurobiological processes and cognition are more contentious.

Do e-fields causally influence neurobiological processes? (Figure 1; arrow 5)

While further work is needed to understand the nature and extent of these effects, our answer is a preliminary and tentative 'yes'. Specifically, the available evidence points towards **ephaptic coupling**, a process by which extracellular e-fields, emanating from individual neurons, cell ensembles, or larger populations, exert causal influences on other neurobiological processes by

routes other than direct synaptic transmission [30]. In our view, this is a pertinent reformulation of the question of whether LFPs are epiphenomenal because it turns the problem into an empirically tangible one about causal relations between processes, instead of measurements.

Given that neurons comprise polarizable cell membranes, it is a biophysical triviality that surrounding e-fields must polarize their membranes and thereby alter neuronal excitability. Indeed, as a theoretical lower bound, neurons can be modulated by fields as weak as 0.01 V/m despite thermal and molecular shot noise [31,32]. However, the central question is whether effects exist that are sufficiently strong to produce meaningful effects on neurobiological processes. Here, we review experimental work suggesting that externally applied e-fields produce changes in spiking activity at the field strengths produced by normal brain activity. In parallel, computational work suggests that these principles also shape endogenous brain activity. We discuss both lines in turn.

First, experimentally induced e-fields polarize neurons both *in vitro* [14,33–37] and *in vivo* [38]. The direct effect of external fields on individual neurons is relatively small. Specifically, supplied fields generally polarize neuronal membranes by <0.5 V/m of the applied e-field, although these effects may be somewhat amplified by resonance within a neural circuit [39]. Additionally, relatively strong e-fields (20–100 V/m) are required to evoke an action potential in quiescent neurons. A misconception is that, because of these factors, weak fields less than ~1 V/m are causally inefficacious. However, in evaluating whether exogenous e-fields produce neurobiological effects is the recognition that action potential initiation is not the only criterion. Namely, weak subthreshold input can alter the temporal structure of spike trains, inducing rhythmic regimes that cause neurons to fire sooner or later than they normally would [14,36,40–45]. As reviewed in [46], external fields as weak as 0.5 V/m can alter the timing of neuronal spiking, with one study observing spike timing modulations at strengths as low as 0.2 V/m [47].

The relevance of such findings depends on whether endogenous fields are similarly strong. As reviewed in [48], a large body of work shows that they are. Starting with a high bound, pathological discharges in the epileptic hippocampus can reach nearly 70 V/m [34]. However, even healthy sharp-wave and ripple activity produces fields between 2 and 20 V/m [49]. Furthermore, the structure of the hippocampus is well suited to generate strong e-fields, and slow waves in the cortex produce e-fields of ~1–2 V/m [48].

A limitation of the external field approach is that it perturbs the natural dynamics of neural systems. Computational models, in which properties and effects of neurally generated e-fields are simulated, offer additional support for ephaptic coupling. In one study, biophysical simulation of neuronal ensembles predicted LFP signals recorded during a spatial task better when the extracellular fields were modeled to weakly feed back into local neuron clusters (although not for all task conditions; [50]). In another study, neural activity was found to propagate along an unfolded slice of hippocampus even when synaptic and gap junction transmission were blocked; simulations tailored to the data suggest that these effects emerge with biologically plausible field strengths [51]. Other efforts using *in vitro* and modeling-only approaches also support ephaptic coupling [52–54].

Furthermore, we highlight two promising future avenues. First, we can exploit propagation asymmetries between ephaptic and synaptic coupling mechanisms to estimate their relative causal contributions. While axons transmit signals at ~0.3 m/s, synchronous e-fields propagate at ~0.1 m/s from their source [53]. As a result, during periodic regimes of neuronal activity, the two effects should interact and amplify at regular moments and distances, and their interference effects on neurobiological processes should be empirically discernible [55]. Second, we can

apply pharmacological and genetic interventions that target bioelectric signaling pathways to acquire some direct control over endogenous fields and their dynamics [56,57].

In conclusion, these lines of evidence suggest that endogenous e-fields are sufficiently strong to alter neural activity (based on the effects of exogenous e-fields), offering more parsimonious accounts of some neural data. Thus, we believe that a common critique of ephaptic coupling (i.e., that it is simply too weak to produce meaningful effects) is unlikely to be true. Nevertheless, ephaptic effects may not be a major contributor to every brain circuit. E-fields most effectively polarize a neuron when they are aligned with the morphology of the cell, generally along its somatodendritic axis [58]. One intriguing possibility is that some circuits may be organized in ways that minimize ephaptic input, akin to designs that mitigate crosstalk in engineered circuits. For example, cells near a large fiber bundle could be oriented orthogonally to the e-field it emits or have otherwise difficult-to-polarize morphology. Given that ephaptic and synaptic inputs travel at different speeds, circuits could be specifically organized such that these signals either do or do not overlap, depending on what is most adaptive. These are testable hypotheses that, to our knowledge, have not been systematically examined.

Do neural e-fields causally influence behavior? (Figure 1: arrow 6)

A further question is whether ephaptic coupling, if it is occurring endogenously in the brain, can influence cognition. The causal and affirmative stance is that ephaptic effects are widespread and powerful enough to change information processing, cognition, and behavior [59]. Critically, underscoring the assumption that any potential causal effects of the e-field on behavior must be driven by its effects on neurobiological processes (as outlined in the previous section), we explicitly highlight such an indirect causal relationship by a dashed arrow in Figure 1. Alternatively, a noncausal stance is that any association we find between cognition and e-fields results only from the effects of those same processes that generated the e-field. As such, the noncausal view maintains that, if any change were to occur to endogenous e-fields, no higher-level phenomena would be affected since those causal neurobiological processes and their effects would be preserved in this hypothetical scenario.

The answer to this dispute is actively being pursued, with indirect lines of evidence supporting the causal position. First, weak external fields of 0.5 V/m increase measures of neuronal synchronization by similar amounts as observed during cognitive events, such as working memory maintenance [60], learning on a discrimination task [61], reward expectancy [62], and during other tasks (reviewed in [43]). Second, externally induced e-fields can influence human behavior across cognitive domains [63–66]. To highlight an illustrative example, the application of an alternating current to frontal and parietal areas changes reaction times on a working memory task when the induced fields oscillate at specific frequencies (6 Hz but not 35 Hz), with such changes depending on the phase difference between the exogenous currents [67]. While causal results of this kind are encouraging, their implications toward the hypothesis that endogenous fields influence behavior and cognition relies on a complex and perhaps underexposed train of inference (Box 1).

Are LFPs informative of e-fields, neurobiological processes, and behavior?

Regardless of whether e-fields causally affect neurobiological processes or behavior, it is a further question whether their corresponding measurement, the LFP, offers information toward processes that can be used to gain scientific insights. Indeed, phrases such as ‘causally relevant’, ‘epiphenomenon’, ‘exhaust fume’, and ‘byproduct’ leave the extent to which LFPs are scientifically meaningful open to interpretation. Here, we try to mitigate such underspecification by evaluating whether information about the state of any one framework entity reduces our uncertainty about the state of another.

Box 1. Interpretative logic of brain stimulation studies

As a general rule, brain stimulation studies adopt the following interpretive logic. First, a perturbational method is used to modulate an independent variable. For example, transcranial magnetic stimulation or electric stimulation might be harnessed to control the presence or strength of a 10 Hz oscillatory field induced artificially in the parietal cortex. Then, how this manipulation alters a dependent variable is measured, such as behavioral patterns in an attentional task, mediated by known or unknown neurobiological processes. Finally, statistically reliable effects of the manipulation are taken to offer evidence that changes in endogenous analogs of the independent variable (e.g., internally generated parietal alpha oscillations) also exert such causal effects on behavior via intermediary neurobiological processes. Critically, this inference contains a multitude of assumptions that warrant scrutiny. For example, to what extent do the supplied fields have similar spatiotemporal dynamics to neurally generated e-fields [200]? Are field-standard stimulation intensities large enough to induce causally effective fields inside the brain [37]? What are the mechanisms by which the exogenous fields are hypothesized to exert their effect (standard interneural communication or ephaptic coupling) and is this how the naturally behaving brain realizes behavior (or are the mechanisms different [201])? What theoretical role do entities of interest have in accounting for the observed findings? For example, what causal and explanatory function is granted to e-fields themselves, spikes, and population dynamics when accounting for the behavioral change? By centering such questions, we can clarify what brain stimulation results teach us about endogenous neural e-fields and their consequences for behavior, cognition, and neuronal computation.

LFP to e-field (Figure 1: arrow 7)

The LFP is informative of e-fields because the charged ions that comprise the e-field define what is measured by electrodes. However, the measurement is imperfect and non-unique, given that it depends on the placement of the reference electrode. One technique to make LFP measurements invariant to such details is to compute the second spatial derivative of the signal. This recovers the current source density, affording almost one-to-one inferences about the e-field [68,69]. Even then, LFP-to-field inferences are hampered by our ability to tell the sink and source apart, as well as other noise factors, such as electrode defects.

Two further inter-related issues are the inverse problem and source contamination. The inverse problem refers to the impossibility of finding a unique set of neuronal generators for any signal given insufficient observations; the source contamination problem refers to the fact that e-fields other than the neural source of interest, such as skin conductance, facial and eye muscles, and the electrocardiogram, also influence the signal. These issues are most striking for global field potentials derived from EEG and MEG, where many more neuronal sources explain variability in the signal, but they are also a problem for local measurements. With these limitations in mind, we emphasize that field potentials are distinctly informative of e-fields given their direct relation. By contrast, methods such as functional magnetic resonance imaging infer neural processes from signals produced by numerous interlocking processes and factors, including cerebral blood flow, blood volume, and metabolic oxygenation rates, some of which have nonlinear relations with each other [70–73].

LFP to neurobiology (Figure 1: arrow 8)

The LFP can be used to draw inferences about synaptic, cellular, microcircuit, neural populations, and more, because, as mentioned above, these neural structures influence the e-field and, therefore, LFPs. However, because there are many ways for a circuit to create the same e-field, it depends situationally on whether specific predictions can be made about neurobiological processes [22]. To offer a few examples where they can, at the single-cell level, the LFPs index the fluctuations of intracellular membrane potential due to excitatory and inhibitory synaptic contributions, both from oscillatory [74–76] and non-oscillatory regimes of activity [26,77–80]. At the circuit level, properties such as power in oscillatory frequency bands informs us of circuit motifs [81] (but see [82]), because different motifs robustly produce different rhythmic responses [83]. Furthermore, the frequency spectra of LFPs can be informative of nonactive properties of the surrounding neural tissue [29], and the exponent of the $^{1}/_{\text{frequency}}$ component scales with the depth

of cortical layers [84]. At the population level, high-frequency activity is strongly indicative of population spiking [85]. Similarly, $^{1}/_{\text{frequency}}$ power law exponents may approximate the balance of excitatory and inhibitory synaptic drives [78,86]. This nonexhaustive list continues to grow. For example, recent approaches combine computational model simulations [16,26,87], multimodal human brain data [88–90], and machine learning methods [91–94] to establish correlations and inverse models between neural dynamics and circuit parameters.

LFP to behavior and cognition (Figure 1: arrow 9)

Techniques that measure local (LFP from microwire) and global potentials (EEG and MEG) have been workhorses across paradigms in cognitive neuroscience. Here, we cover a subset of approaches and highlight more comprehensive reviews available elsewhere.

As a first example, event-related potentials (ERPs) reflect cross-trial, time-domain average neural responses to internal and external events of interest. There are different kinds of ERP, occurring at various temporal offsets and emerging from different sources in the brain [95–97]. A massive body of work has shown that these patterns offer reliable signatures of behavior and internal cognitive processes, including sensory processing [98], prediction error (P300 [99]; N400 [100]), action planning (readiness potential [101]), attention [102], and memory [103].

Second, the previously mentioned $^{1}/_{\text{frequency}}$ component across the LFP is predictive of arousal levels [104], and sleep stages [105], and explains variability in cognitive functions, including attention [106], memory performance [107], and language learning [108].

Third, research has capitalized on the full range of LFP dynamics to predict cognitive processing by searching for systematic relations between brain activity and behavior of interest in a data-driven fashion. Relevant approaches include statistical techniques, such as multivariate pattern analysis (MVPA [109–112]) and neural network approaches [113,114]. Besides predicting positional [115,116] and bodily variables [117], these techniques confer information about internal processes that can be difficult or impossible to assess behaviorally. For example, LFPs are informative toward attentional focus [118–120], the contents of working memory [121], the evolution of long-term memory processes [122], mental event replay [123,124], and internal decision-making variables [125]. In this way, the LFP adds to our repertoire of behavioral and cognitive measurements by shining a light on opaque internal neurocognitive processes, affording mechanistic insights that are hard to obtain via psychological experimentation.

The role of oscillations

Here, we home in on the phenomenon of oscillations (Figure 2). As with our previous discussion of LFPs and e-fields, our aim is to clarify entangled uses of the term ‘oscillation’ and to foster a common ground for scientific discussion and investigation. While it is generally accepted that ‘oscillation’ refers to a specific kind of temporal structure, the term has a multiplicity of uses across the cognitive and systems neuroscience literature. In contrast to its straightforward mathematical definition [a function $f(x)$ is oscillatory when $f(x) = f(x + T)$ for some non-zero period, T], defining oscillations is less straightforward in the biological sciences, in which nothing repeats perfectly or indefinitely. This results in two distinct ambiguities. First, the term ‘oscillation’ is used indiscriminately to refer to a specific kind of temporal structure in measurements or in processes. Second, there is a lack of consensus on how to identify and distinguish oscillations from other dynamical features, regardless of whether that concerns measurements or processes [126]. These ambiguities feed into two further sources of disagreement: (i) the degree to which oscillations in the e-field exert causal effects; and (ii) the role of oscillations in neural computation.

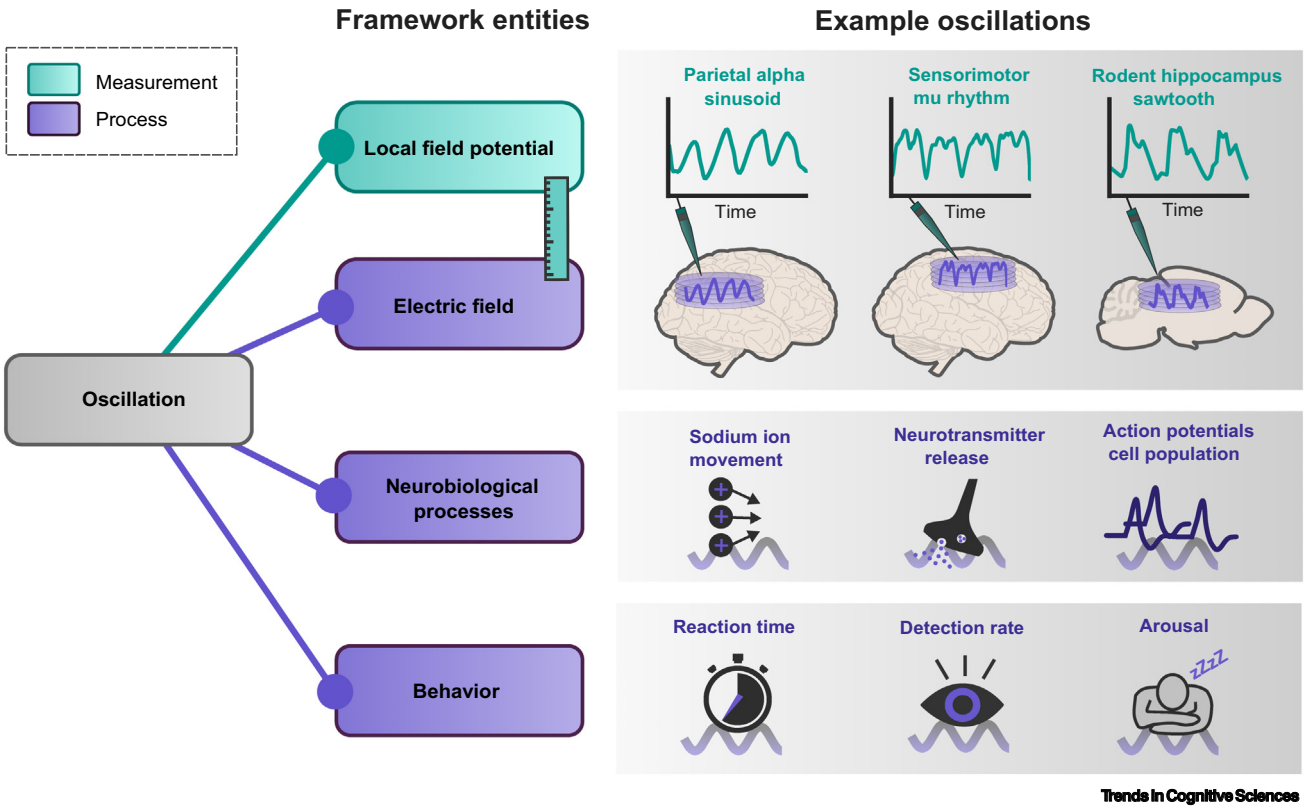


Figure 2. Oscillations-in-measurement and process. Oscillations are characterized by rhythmic temporal structure in any of the entities in our framework, manifesting variously in both measurements and physical processes. Oscillations-in-measurements are rhythmic fluctuations in the signal of electrophysiology recordings. At the level of processes, oscillations can instantiate as fluctuations in the electric field, or in other neurobiological events, such the release of neurotransmitters and action potentials across neuronal masses. At a behavioral level, oscillations can manifest, for example, as a waxing and waning of reaction time, stimulus detection rates, and arousal levels. Each of these characterizes one type of oscillation.

Oscillation in measurement and process

As with the distinction between e-fields and LFP, it is crucial to distinguish oscillations in the measurements we use to study neurobiological systems from oscillations in the processes themselves:

Oscillations-in-measurements are periodic-like structures in temporally evolving signals, such as the LFP (Figure 2, top row). This includes persistent rhythms, such as the 6–10 Hz fluctuations observed in data from the rodent hippocampus [3], as well as short-lived transients [127], including the several hundred millisecond-long bursts of 20 Hz oscillations observed in data from human primary somatosensory cortex [128]. These dynamical features may or may not be visible to the naked eye, and are more commonly quantified with techniques such as Fourier and wavelet transforms [129], temporal filters (e.g., bandpass), and approaches that extract oscillatory components by applying linear projections to the data [130–132].

By contrast, oscillations-in-process are typified by rhythmically repeating sequences of physical events (see below for demarcation criteria). In addition to e-fields, oscillations also occur in neurobiological processes, such as the spiking of individual neurons [133,134] and populations [11], the opening and closing of membrane ion channels [135], gene expression [136], and cerebral hemodynamics and metabolism [137], as well as in behavior [138], including memory [139] and attentional processes ([140,141]; but see [142,143] for analytical considerations; Figure 2).

Oscillations typically emerge from the interaction of multiple connected processes, such as through the presence of negative feedback or nested loops [144]. For example, networks of mutually inhibiting neurons [145] and more complex configurations of excitatory and inhibitory neurons naturally instantiate a push–pull dynamic [83,146–148], which produces interdependent rhythmic fluctuations in population spike rate, synaptic currents, and, ultimately, the surrounding e-field. At the cellular level, the interplay between depolarizing and hyperpolarizing conductances produces rhythmic fluctuations in the membrane potential and spiking dynamics of individual neurons [149,150].

Inferring oscillations-in-process from oscillations-in-measurement

In most cases, we are not interested in the measurements themselves, but rather the oscillations-in-process they may reflect, and it is worth noting explicitly and exploring how one might occur without the other. Furthermore, inferring oscillations-in-process from measurements carries with it two challenges of demarcation: (i) how are oscillations-in-measurement identified among other patterns in our signals; and (ii) what differentiates any rhythmically fluctuating process from a genuine oscillation-in-process?

The first challenge concerns the demarcation of oscillations-in-measurement from colored noise and other aperiodic fluctuations seen widely across neural signals [151]. Moreover, oscillations may take non-sinusoidal shapes, such as a sawtooth [152] or μ-like shape [153]. All of these produce narrowband spectral power at nonfundamental frequencies, such as around harmonic frequencies [107,154]. The field is actively developing techniques and guidelines to improve our signal analysis capabilities [155], including algorithms that factor in colored noise [156,157], methods that account for the waveform shape of oscillations [152,158], quantification techniques that rely on phase consistency [159], phase autocorrelations [160], and spatial filters that amplify the detection of weaker rhythms [132]. Importantly, these issues not only concern oscillations in brain activity, but also apply to the detection of any oscillation from time series measurements, including oscillations in cognitive and behavioral data [142,143].

Second, even when reliable rhythmic patterns are detected in neural recordings, they may not always be produced by oscillations-in-process. First, the pattern may be an illusory effect of data processing [161,162]. Second, when rhythmic trains of stimuli are presented (such as a sequence of sounds), recordings of brain activity will contain a rhythmic component simply because each stimulus produces an ERP, with the concatenation of such responses giving the illusion of an oscillation [163,164]. However, oscillations-in-process arise from oscillators and their unique properties: oscillators have a rate at which they preferentially fluctuate (i.e., they have a natural and resonance frequency), and they operate in a self-reinforcing fashion, both of which leave their mark on electrophysiological measurements ([165], but see [166] for a critical perspective). For example, the self-reinforcing nature of oscillators means that rhythmic activity is expected to persist for one or more cycles after external inputs have ceased [66,167–169], and the existence of preferred oscillator frequencies entails that, as the distance in frequency between external rhythm and oscillator increases, more stimulus intensity is required to produce synchronization [170,171]. Thus, repetitions of processes are necessary, but not sufficient, for the presence of an oscillation-in-process. What is also necessary is the involvement of an oscillator, and our ability to evaluate if this is the case (rather than assume as much) depends on the experimental context.

The implicit conflation of oscillations-in-process and oscillations-in-measurement could contribute to disagreements about whether oscillations are epiphenomenal. Indeed, if two discussants implicitly associate the word ‘oscillation’ in varying degrees with periodicity in the LFP versus

periodicity in physical processes, scientific exchanges between them will be misdirected from the outset. Box 2 provides an overview of related ambiguities.

The potential role of oscillatory ephaptic coupling

Previously, we reviewed evidence for the causal role of e-fields in neurobiological processing. Here, we evaluate a variation of the ephaptic coupling hypothesis that specifically considers oscillating e-fields. Put differently, what causal powers might be exerted by e-fields that fluctuate periodically, at characteristic frequencies?

A fundamental insight from the physics of oscillators is that weak perturbations can produce strong and diverse effects on systems of coupled oscillators [170]. In the brain, cortical neurons operate at a noise-driven, high-conductance state near threshold [172]. From this basis, weak e-fields have the potential to exert strong effects even if they cannot in isolation cause neurons to discharge action potentials. Specifically, a key idea is that oscillatory fluctuations in the e-field riding on top of background noise-like dynamics can translate into changes to spiking activity across masses of neurons. The prediction is that oscillations in the e-field, which closely match the intrinsic or synaptically driven oscillations in neurons, can amount to an additional force on top of classical mechanisms, where the coordinated temporal summation (which is intrinsically present in waxing and waning oscillations) influences the amount of rhythmic spiking across cell populations [173]. Moreover, as discussed previously, even if the e-field fluctuations are not aligned (in terms of shape, frequency, or phase) with the spectral content of endogenous or other input dynamics of neurons, the fields might compete for control over spike timing.

Similarly, even if e-fields are not oscillating, the degree of rhythmic spiking of neurons could be assisted through stochastic resonance, a phenomenon where weak and otherwise undetectable

Box 2. Ambiguity of the term ‘oscillation’

Having posed the distinction between oscillation-in-measurement and oscillation-in-process, we explore two terminological ambiguities that occur in the literature. First, for an oscillation-in-process, it is not always apparent whether authors are referring to fluctuating e-fields only or to the entire generating mechanism associated with them [202]. For example, the claim that alpha oscillations underpin attention might mean that the e-fields are doing the primary causal work (e.g., via ephaptic coupling); alternatively, it could mean that the neurobiological events that produced the e-fields are doing the heavy lifting.

Second, sometimes, the same terminology is used when referring to both a given oscillation-in-process as well as its corresponding oscillations-in-measurement (this differs from the previous point, which concerns processes only). For example, ‘theta oscillation’ might refer to fluctuations in physical events in the brain and to fluctuations in a measured signal between 4 and 8 Hz, with the implicit (although potentially incorrect) understanding that the two entities are coupled. This distinction is nontrivial, because theta-in-measurement can be explained by a plethora of other neural processes. For example, 4–8 Hz peaks in the signal might reflect e-fields from local circuit operations at a source of interest, such as the hippocampus, or might reflect volume conducted e-fields of surrounding structures, such as the medial septum and olfactory bulb [187,203]. Second, signal peaks might reflect different types of process even near the source of interest, such as atropine-sensitive and insensitive theta [204].

Putting all this together, ‘theta’ might simultaneously refer to: (1) peaks in the frequency spectrum between 4 and 8 Hz of a signal; (ii) fluctuating e-fields originating from dipole sources in the hippocampus; (iii) e-fields generated by axonal projection of volume conduction far away from the source; (iv) the underlying circuitry necessary to produce e-fields over and above the e-fields themselves; or (v) a mix of all of the above, including signal, generating mechanisms, and task variables inducing the oscillation. Such ambiguity naturally leads to confusion, as well as to empirical and conceptual disagreement regarding the epiphenomenal nature of oscillations. Although this example isolates ‘theta’ as a canonical example, it is equally applicable to the various connotations of alpha (thalamus, visual cortex, and attention), beta (motor, motor cortex, and basal ganglia), gamma (perception, sensory cortex, and pyramidal–interneuron–gamma circuits), and other characteristic frequency bands.

processes are boosted by the addition of noise [174,175]. Together, these findings preliminarily suggest that oscillatory e-fields are a physical process with sculpting functions, and that even non-oscillating e-fields can bolster rhythmic firing modes. With that said, the methodological considerations and caveats covered earlier (see 'Theoretical status of local field potentials and electric fields') apply here too.

Oscillatory computation

Regardless of the causal role of e-fields in oscillatory processes, a key question in neuroscience is what role, if any, oscillations-in-process across levels of organization have in neural computation. Under the causal stance with regard to neuronal computation, oscillations considered as an intertwined set of neurobiological processes serve an active coordinating function to information exchange across populations of neurons. Under the noncausal stance, even if oscillations reflect a mode of brain activity that correlates with cognition, oscillations are not granted processing capacities required to influence cognition. These stances subtly differ from the range of positions one might have on the causal status of e-fields. Specifically, one can coherently hold the selective view that e-fields are causally important for some homeostatic or other biological functions, while rejecting that oscillations-in-process, e-field or otherwise, have a causal role in the mechanisms of neural computations underlying cognition.

Here, we outline a view that oscillations-in-process implement a kind of neural syntax [176] (Figure 3): an arrangement of neural activity such that it is optimally interpretable to downstream readers [8]. We review how, as per our hypothesis, this syntax relies on the ability of oscillations to orchestrate neural activity in time, space, and frequency (Figure 3), endowing neural circuits capable of information processing with the ability to flexibly implement multiple functions. Before we start, we offer a brief note on terminology. Causal words, such as 'group', 'organize', and 'orchestrate', may come with a connotation of agency. However, in our usage throughout this section (and the whole paper), the idea is not that oscillations are isolated agential processes that can arrange brain activity in a self-contained manner, like a conductor or homunculus. Rather, the phrase describes the role of oscillatory processes in larger mechanistic architectures

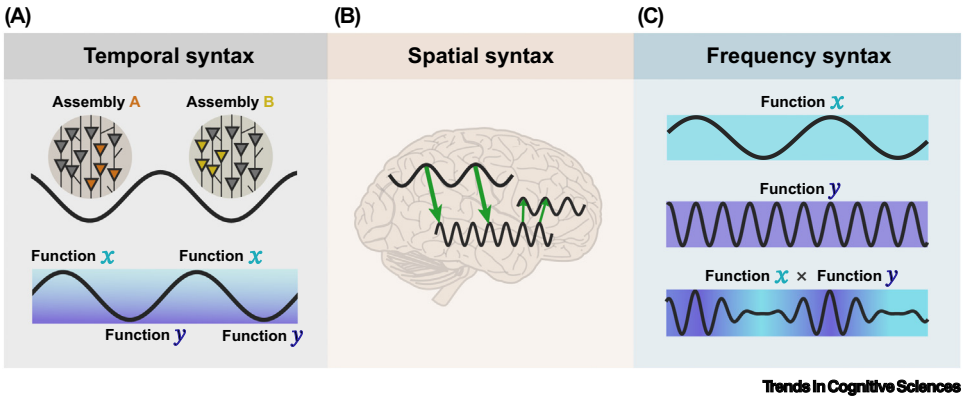


Figure 3. Oscillations as orchestrators of neural computation. Oscillations are hypothesized to support the organization of neuronal activity in time, space, and frequency, allowing neural circuits to engage in more than one computation. Under a temporal syntax, different computational functions alternate in a waxing and waning fashion, changing dynamically how circuits and their components activate. Under a spatial syntax, ensembles across regions of the brain synchronize excitable phases, establishing functionally distinct inter-regional communication channels. Under a frequency syntax, circuits and intracellular processes operating at different frequencies implement different functions. Each displayed waveform illustrates hypothesized oscillations-in-process, which may not produce clear oscillations-in-measurement, and which may add a $^{1}/_{\text{frequency}}$ component to the signal.

(see also Outstanding questions). Here, we discuss each form of orchestration in turn, starting with the organization of computation through a temporal syntax.

Like the clock of a digital computer, which discretizes electrical activity into functionally related packets, oscillations are theorized to help group functionally related spikes into distinct oscillatory cycles and/or to distinct phases within a cycle (Figure 3A). Prominent examples include the widely observed grouping of spikes into cell assemblies by 30–100 Hz (gamma) oscillations [146,177,178], and the sampling of possible behavioral trajectories by hippocampal theta oscillations in rodents [179–181] and humans (1–4 Hz; [182,183]), whereby spikes at early phases represent past, and those at later phases represent possible future, positions of the animal, respectively. By inducing temporal synchrony, grouping-by-oscillations allows assemblies of cells to discharge downstream (e.g., postsynaptic) readers, which require multiple active presynaptic partners to induce an action potential [184]. While grouping-by-oscillations is one way to introduce temporal synchrony, non-oscillatory autocorrelated processes can also achieve a similar effect over arbitrarily long timescales.

Oscillations not only group synchronous outputs, but also provide windows of opportunity in which neurons are sensitive to inputs via fluctuations between hyperpolarized and depolarized phases of neuronal membrane potentials [185]. Then, through the coordination of synchronous oscillatory phases between brain regions, oscillations provide a spatial syntax to neural computation, enabling information-coding ensembles to effectively communicate with downstream readers during systematic timeframes ([9,186]; but see [187] for critical considerations; Figure 3B). For example, theta oscillations enable communication between hippocampal region CA1 and entorhinal regions via the perforant path, from CA3 via the Schaffer collaterals at alternating phases of the theta oscillation [188], and through the coordination of distinct gamma rhythms [189]. Furthermore, some oscillations slowly propagate across stretches of cortex, modulating spiking along the way [190]. Such traveling waves change direction when the brain moves between encoding and recall states [191], streamlining with the notion that oscillations aid inter-regional information exchange.

Finally, under this syntax framework, oscillations coordinate neural processing via a frequency syntax, by which oscillations at different frequency bands carry functionally distinct information or perform separate computations (Figure 3C). For example, in the rodent basal forebrain, oscillations at 20–35 Hz (beta), 45–65 Hz (gamma), and 80 150 Hz (high gamma) are each seen at distinct cognitive processing stages in a spatial orientation task [192]. Top-down versus bottom-up signals in the neocortex are processed locally at different frequency ranges [193], and high-frequency regular ‘burst’ spiking is associated with synaptic plasticity [194,195]. Oscillations at varying frequencies can use cross-frequency coupling to implement a nested frequency syntax, such as when the amplitude of fast oscillations varies along the phase of slow oscillations to encode perceptual [196], memory [197,198], or linguistic content [199].

Overall, we propose that oscillations are best thought of as enabling functional multiplexing: the ability of the same neural tissue to perform multiple, state-dependent functions as required depending on the context. Each kind of syntax allows neural populations to process functionally distinct signals. In short, we hypothesize that oscillations-in-process generally are central to the organization of information processing within and across networks in the brain. Finally, a nascent question is how these ideas cohere with the perspective of neural population geometry (or neural manifold) (Box 3).

Box 3. Limit cycles and oscillatory computation

Recent theories of population coding posit that task-relevant activity emerging from large groups of neurons undergoes stereotypic patterns that are captured by movement along a manifold in lower-dimension projections of the data [10,205]. In these subspaces, we can detect motifs of aggregate brain activity that reliably underpin computation, which might take the shape of nonlinear surfaces or point or line attractors [206]. Importantly, limit cycles are a type of periodic attractor that emerges, or can be reconstructed, from periodic and sequentially activated neuronal populations, such as during repeating phase-locked firing patterns clocked by theta oscillations associated with sequential memory activation in humans [207] and computational models [208].

Importantly, however, while frequently observed rotational dynamics, such as those detected in the motor cortex [209,210] and spinal cord [211], are limit cycles in state space, they do not necessarily imply the existence of an oscillation-in-process. Rotational dynamics can be explained by the sequential activation of populations alone [212], and periodically repeating motifs can be induced by repetitive, stereotyped muscle contraction [211]. Such repeating activity is not a genuine oscillation, but more akin to our previous example of stimulus trains in auditory entrainment studies producing a concatenation of ERPs. In other words, oscillations can imply the existence of rotational dynamics along limit cycles, but rotational dynamics alone do not prove oscillations-in-process. Future experimental, modeling, and conceptual efforts are needed to establish how the oscillatory computation and manifold perspective tie together (for a key example, see [213]).

Concluding remarks

Oscillations, LFPs, and other key terms in the electrophysiologist's dictionary are used in various senses across the literature. Some variability might be healthy to foster pluralism, but too much divergence risks a conceptual quagmire where scientists, research groups, and subfields talk past each other. For example, if one party conceives of LFPs as electric fields generated locally in confined circuits, while another views them as an underdetermined signal with unboundedly many processes underpinning it, then disputes over the relevance of LFPs or what research avenues are worth pursuing become misdirected from the outset. In a similar vein, the claim that oscillations are epiphenomenal leaves open: (i) whether this concerns oscillations as processes, signals, or both; (ii) what oscillations are epiphenomenal toward; (iii) whether oscillations are informative despite their (apparent) causal inefficaciousness; and, if so, (iv) which other processes they are informative toward or not.

Neuroscience may benefit from distinguishing measurements and processes on the one hand, and informativeness and causality on the other. We propose that these axes of analysis unclutter terminological vagueness and orient debates toward experimental validation. Adopting this lens, we offer empirically driven answers on the causal and inferential role of processes and measurements in electrophysiology. Our answers are detailed below.

First, LFPs are measurements that reveal direct information about e-fields, as well as indirect information about neurobiological processes, cognition, and behavior. Many of these processes have causal relations between one another, although the evidence varies on a case-by-case basis. Perhaps most centrally, e-fields likely influence neuronal processing over and above being produced by it through ephaptic coupling. Furthermore, we posit that oscillations are best cast as a separate entity that can arise in processes and measurements. In this context, oscillations in processes across levels of scale appear to introduce a neural syntax by which multiplexed computation is enabled. However, not all measured neural activity is oscillatory, and neither does the presence of an oscillation in one entity necessitate oscillations in the other, meaning it is instructive to clarify what is oscillating when referring to neural processes or signals.

Overall, the answers obtained from our reframed questions energize research programs that tentatively consider electric fields and their oscillations as active causal phenomena, while the framework can be applied to other neural signals and processes (see Outstanding questions). Furthermore, even if these processes are not causally relevant, their measurements yield

Outstanding questions

For any specific cognitive function and its underpinning neuronal circuitry and biological processes, what is the relative contribution of ephaptic and non-ephaptic processes?

What experimental protocols and signal analysis techniques reliably distinguish oscillatory and non-oscillatory processes?

By what principles and mechanisms are oscillatory regimes modulated and reconfigured to support cognition on the fly?

How can we attain experimental control over the electric fields in the brain without introducing confounding causal influences?

Can the distinction between measurements, processes, and inferential and causal relevance clarify other domains? For example, the term 'spike' could carry related ambiguities, contextually referring either to sharp transients in the LFP or causally efficacious action potentials. In addition, as discussed in the main text, functional magnetic resonance imaging involves myriad interlocking measurements and processes.

More broadly, what meta-scientific innovations can mitigate conceptual ambiguity in the vocabulary and terminology used in neuroimaging research?

information about behavior, cognition, as well as other dimensions relevant across systems and cognitive neuroscience.

Acknowledgments

We thank Earl Miller for his comments on this manuscript, and Luiz Pessoa and participants of the Neuroscience Philosophy Salon for debate and discussion. We also thank Roby Duncan, Erik Peterson, and members of the Voytek lab for discussions that inspired an earlier version of this work while R.G. was at the University of California, San Diego, as well as Aleksejs Timcenko for discussions at the University of Tübingen. In addition, we thank Maria Wimber, Simon Hanslmayr, and members of their respective labs for relevant discussions while S.v.B. was at the University of Glasgow, and we thank Christoph Daube for his comments on this manuscript. D.L. is supported by the FRQNT Strategic Clusters Program 2020-RS4-265502, Centre UNIQUE, Union Neurosciences & Artificial Intelligence, Quebec, and the Richard and Edith Strauss Postdoctoral Fellowship in Medicine. R.G. is supported by the European Union's Horizon 2020 research and innovation program under Marie Skłodowska-Curie grant agreement No. 101030918 (AutoMIND).

Declaration of interests

The authors declare no competing interests.

Resources

[i]https://virati.medium.com/measurement-vs-measurement-346ace68be50

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