Trends in Cognitive Sciences

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Feature Review

The emerging field of cognitive brain–computer interfaces

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Brain–computer interfaces (BCIs) have achieved transformative success in restoring movement and communication. However, extending these approaches to decoding or recovery of cognitive function, such as attention or memory, poses fundamentally new challenges. Cognitive BCIs will need to contend with distributed and dynamic neural processes that differ sharply from the more localized, stable representations underlying motor and language control, imposing new technical and conceptual demands. Conversely, neuromodulation, long used in neurological and psychiatric therapies, offers a complementary methodological path and initial translational applications through causal modulation of cognitive circuits. Integrating these approaches into adaptive, closed-loop systems could allow cognitive BCIs to restore mental function and bridge systems neuroscience and next-generation neurotherapeutics capable of monitoring and shaping human cognition in real time.

Highlights

Advances in neural recording and decoding are expanding brain–computer interfaces from motor and language restoration to cognitive functions.

Emerging cognitive brain–computer interfaces aim to monitor or alter internal states such as attention, memory, emotion, and decision-making.

There are significant differences in the neural basis of cognitive versus motor/language processes, which will require different approaches in cognitive brain–computer interface.

Clinical applications represent a natural starting point for cognitive brain–computer interface development, given the predominance of cognitive deficits in psychiatric disorders.

Multimodal data, layered behavioral labels, large datasets, and foundation models provide a potential pathway toward the methodological development of cognitive brain–computer interfaces.

Eventually, closed-loop devices, building on motor decoding and clinical neuromodulation foundations, will be essential for cognitive brain–computer interfaces.

Cognition: The next frontier in brain–computer interfaces

Brain–computer interfaces (BCIs) (see Glossary) have advanced rapidly over the past decade, enabling the partial restoration of lost motor and speech functions in people with paralysis. Through the convergence of high-resolution invasive neural recording technologies and improvements in machine learning and decoding algorithms, BCIs are becoming a mature technology with clinical utility in motor and language restoration.

These developments have brought a new possibility into view: using BCIs to decode not only overt actions but also the internal, partially unobservable cognitive processes that determine how we attend, remember, decide, and feel. This shift toward **cognitive BCIs (cBCIs)** is timely for several reasons. First, it has the potential to open novel therapeutic avenues: brain disorders such as depression, anxiety, post-traumatic stress disorder, and obsessive–compulsive disorder (OCD) are, in part, disorders of cognition [1–4], and current treatments often offer limited relief for their cognitive symptoms. Second, cognitive neuroscience is producing increasingly precise characterizations of the neural basis of cognitive processes, offering insights into the mechanisms and biomarkers necessary for translation. Finally, the rapid maturation of adaptive and **closed-loop neuromodulation** provides a methodological foundation for interventions that respond to brain states in real time, which are likely to be essential for tracking and modulating rapidly shifting cognitive states.

This review focuses primarily on translational cBCIs, grounded in human invasive electrophysiology and neuromodulation methods. These methods provide the temporal resolution, signal quality, and causal approaches needed to support effective cBCI implementation. Together, emerging evidence from cognitive and translational neuroscience and advances in decoding and neuromodulation technologies represent an inflection point for a new generation of cognition-focused neurotechnology. This review outlines the opportunities and challenges for

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cBCIs by first considering the foundational successes of motor and language BCIs and then contrasting the neural basis of these systems with that of cognitive processes. It then examines clinical neuromodulation as a complementary translational bridge toward adaptive, closed-loop cBCI architectures that integrate real-time decoding with targeted modulation of cognitive circuits.

Motor BCIs: The decoding foundation

BCIs are evolving from experimental tools used for neural decoding in research settings into clinically viable tools capable of partially restoring lost communication and motor functions. By combining invasive recordings of neural activity with decoding models, they translate patterns of brain activity into actionable outputs such as cursor control, robotic arm movement, direct motor restoration, or synthetic speech (Figure 1). In recent years, BCI development has been driven by significant and simultaneous advances in invasive recording hardware, large-scale computational modeling, and adaptive machine learning algorithms.

Intracortical motor BCIs now enable some individuals with paralysis to achieve real-time control of external effectors. Implants in the primary motor cortex record dense and temporally precise neural activity related to movement intention, which can be translated by decoding algorithms into two- or three-dimensional cursor or limb control [5–7]. Clinical studies have demonstrated reliable performance in individuals with spinal cord injury, amyotrophic lateral sclerosis, and stroke, typically using penetrating **microelectrode arrays** (Utah arrays) and, in some cases, less invasive interfaces such as surface **electrocorticography (ECoG)** systems [5,8]. These advances have yielded smooth, low-latency (<100 ms) control with relatively stable signal performance, bringing these systems closer to functional motor restoration.

Language BCIs emerged from motor BCI research and have developed in parallel, targeting the decoding of speech from cortical activity in motor, premotor, and peri-Sylvian regions. Unlike motor BCIs, which decode continuous trajectories, speech BCIs must interpret high-dimensional, temporally precise articulatory signals to produce speech with high accuracy, with the goal of achieving speech production at speeds close to natural language production. Recent breakthroughs combining spiking or high-frequency neural activity with deep learning and large language models have enabled near-natural speech reconstruction, including handwriting-based text BCIs that achieve communication rates of up to 90 words per minute with over 97% accuracy across vocabularies exceeding 100 000 words [9–12].

In parallel with academic progress, industry is developing increasingly biocompatible, high-channel-count, and less invasive neural recording and stimulation technologies with improved signal quality [13]. These platforms are now entering clinical trials, primarily for motor and speech restoration, but with expanding applications [14]. Together, recent academic and industrial advances mark the maturation of invasive neurotechnology from experimental proof of concept toward clinically meaningful, scalable translational platforms capable of partially restoring motor and communication functions.

Toward cBCIs: Expanding beyond decoding and stimulation

In contrast to motor and language BCIs, and despite the urgent need to develop new treatment options for patients with neurological or psychiatric conditions characterized by cognitive deficits, comparatively little progress has been made in developing BCI devices targeting nonmotor brain function. cBCI technology aims to achieve that goal, building on advances in motor BCIs to create systems capable of decoding and modulating internal cognitive processes such as attention, memory, decision-making, emotion, and social cognition, representing a set of new areas of development for BCI technology [15].

Glossary

Brain–computer interface (BCI): an electrophysiological recording system capable of decoding brain activity to enable communication, control, or other therapeutic interventions.
Closed-loop neuromodulation: a bidirectional system that senses neural activity, decodes a functional or pathological state, and provides feedback neuromodulation in real time.
Cognitive BCI (cBCI): a BCI designed to decode or influence internal cognitive states (e.g., attention, memory, emotion, and decision-making).
Deep brain stimulation (DBS): a neuromodulation therapy involving implanted electrodes that deliver electrical stimulation to deep brain nuclei, commonly used for the treatment of movement disorders.
Ecological momentary assessment (EMA): a data collection method that captures individuals' behaviors, experiences, or symptoms in real time, typically through repeated prompts, that is, as those delivered via mobile devices.
Electrocorticography (ECoG): the recording of LFP cortical activity using electrodes placed directly on the brain surface.
Foundation model: a large, general-purpose model trained on broad and diverse data at scale, which can be adapted or fine-tuned for many downstream tasks.
High-frequency activity (HFA): neural activity in the 70–200 Hz range, reflecting local activation and neuronal spiking, commonly used in decoding.
Local field potential (LFP): aggregate electrical activity reflecting the summation of synaptic and dendritic processing by neuronal populations.
Low-intensity focused ultrasound (LIFU): a noninvasive neuromodulation technique that allows the modulation of activity in deep brain areas through directed sound waves.
Microelectrode array: a high-density electrode array capable of recording high-density neurophysiological activity from a small patch of the brain, commonly using penetrating electrodes capable of recording single-unit or multi-unit activity.
Neural oscillations: oscillatory components of neural activity that capture the aggregate activation and deactivation of neuronal populations. Canonical frequency bands are associated with a variety of cognitive

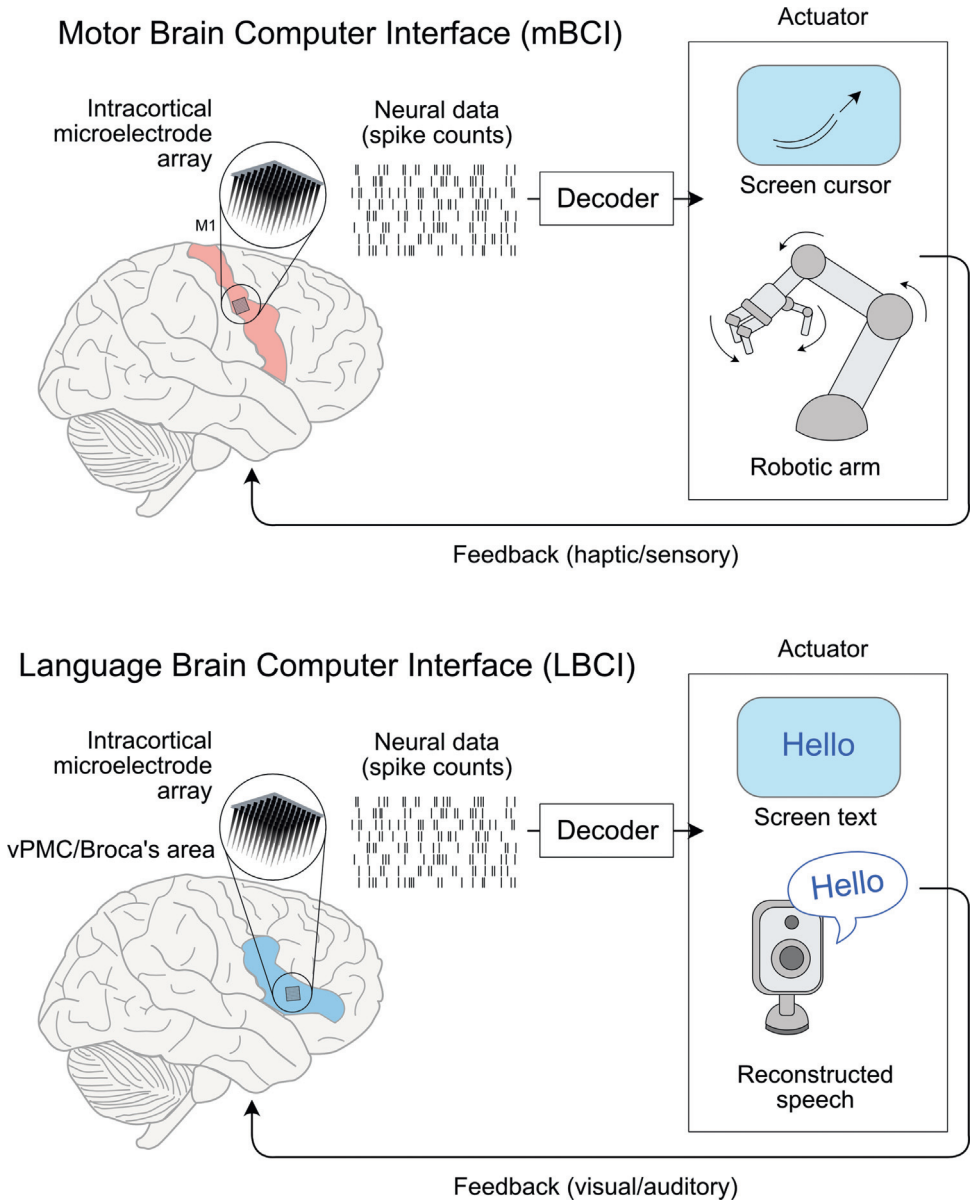

Figure 1. Motor and language BCI approaches. Current BCI methods focus on restoring motor (top) and language (bottom) functions in patients with paralysis. The architecture is unidirectional, with dense neural data (typically spike counts) collected from a single specialized brain region through high-density microelectrode arrays. The processed neural data are decoded to infer movement or speech intent and used to control an external actuator, which, for example, may operate a computer cursor or robotic limb or enable speech production, thereby providing significant therapeutic benefit to patients with paralysis. BCI: brain–computer interface; vPMC: ventral premotor cortex.

processes and include delta (1–3 Hz), theta (3–8 Hz), alpha (9–12 Hz), beta (13–30 Hz), and low gamma (30–60 Hz).
Stereoencephalography (sEEG): recording of LFP brain activity using depth electrodes inserted into deep and cortical structures, often used for epilepsy diagnosis.
Reactive neurostimulation (RNS): a closed-loop neuromodulation system that detects pathological neural activity (e.g., seizures) and delivers targeted stimulation to prevent seizures.
Transcranial magnetic stimulation (TMS): a noninvasive neuromodulation technique that allows for the modulation of activity in superficial brain areas through magnetic pulses.

However, fundamental differences between the neural bases of motor and cognitive processes mean that cBCI development will face a distinct set of challenges from those of motor BCIs (Figure 2). This section reviews some of the most relevant challenges, including the distributed nature of cognitive processes, the importance of interregional coordination and **neural**

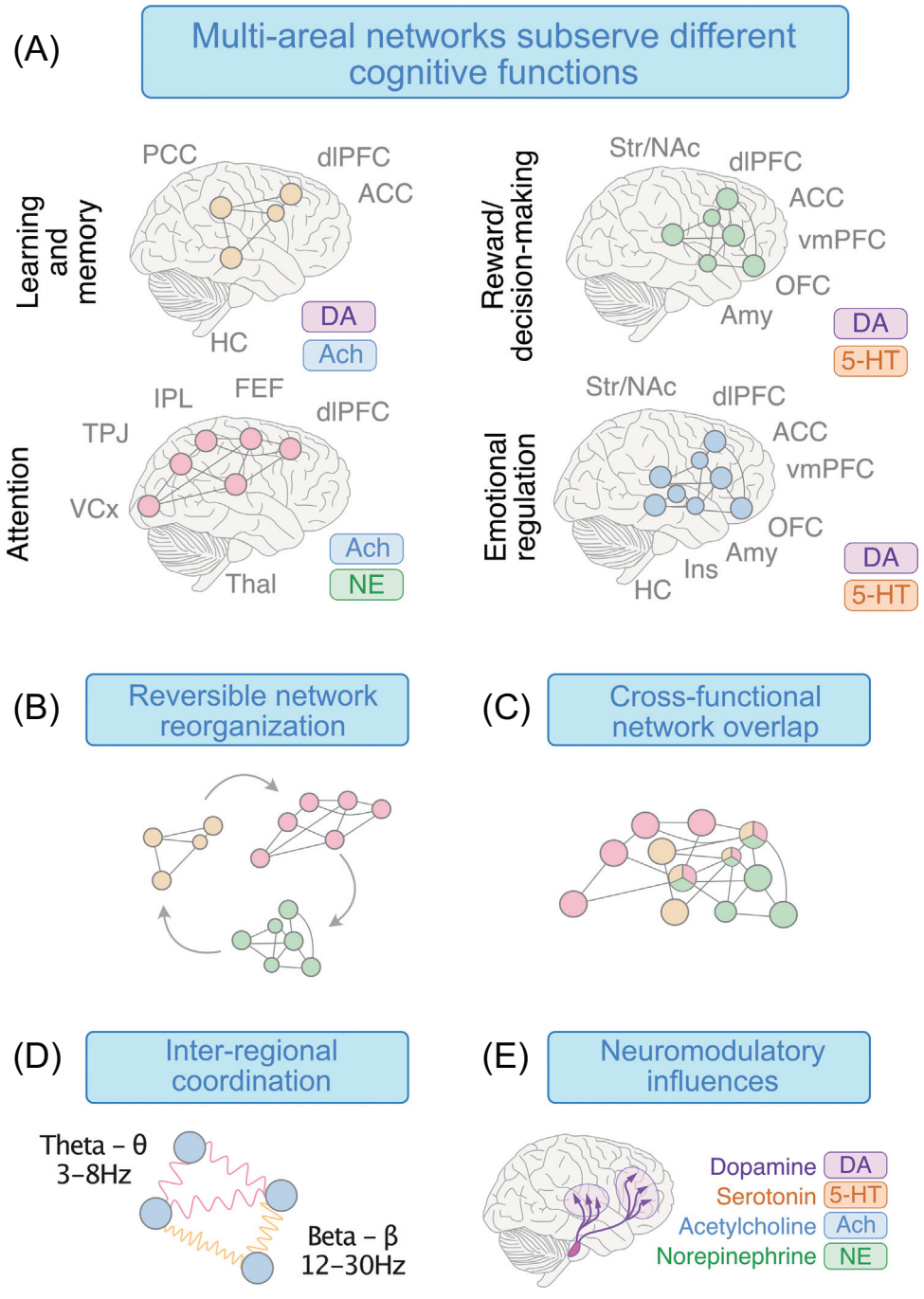

Figure 2. The neural basis of cognitive processes. The organization of the neurobiological basis for cognitive processes is fundamentally different from that of motor processes. Consequently, successful development of cognitive BCIs will require leveraging these differences. (A) Cognitive processes, such as memory and attention, depend on distributed networks of interconnected brain areas, unlike the more discretely organized regional representations of motor and language functions. (B) Cognitive networks are organized transiently and subject to fast, reversible establishment and dismantlement in service of current behavioral goals. (C) Brain networks subserving different cognitive functions overlap, with some key brain areas involved in multiple cognitive functions. (D) Distinct patterns of neural oscillations are associated with cognitive functions,

(Figure legend continued at the bottom of the next page.)

oscillations, the dynamic nature of cognitive states, the role of neuromodulatory systems, and the challenges of generalization and behavioral 'ground truth'.

From localized to distributed neural representation

Motor and language BCIs represent a natural starting point for BCI development because their neural substrates are well characterized and topographically organized. Firing rates in the motor cortex represent movement direction, velocity, and force [16]; speech articulation maps onto well-defined premotor regions [17]. This localization provides precise anatomical targets to record relevant information from ensemble neuronal activity, which can be passed on to decoding algorithms.

In contrast, there is now growing evidence that cognitive processes do not reside in a single cortical locus [18]. Instead, attention, decision-making, and emotion are supported by multiple distributed brain regions, including prefrontal, parietal, temporal, limbic, and subcortical areas (Figure 2). For example, decision-making engages the ventromedial prefrontal cortex, orbitofrontal cortex (OFC), anterior cingulate cortex, basal ganglia, and dopaminergic midbrain regions [19,20]; working memory engages multiple fronto-parietal regions [21]; memory engram ensembles are distributed across networks connected to the amygdala and hippocampus [22], with replay activity coinciding with widespread cortical activity [23]; attention involves top-down control of prefrontal regions onto parietal regions [24]; prefrontal networks underlie executive function [25]; task engagement is best decoded using multiareal brain activity [26]; and brain-wide activations underlie complex behavior [19].

This distributed architecture poses several intertwined challenges. First, it may complicate signal localization, since there is no prime candidate region that needs to be targeted for adequate decoding. Moreover, it is possible that decoding cognitive states with optimal accuracy may require multiareal sampling and integration of information across regions, rather than dense sampling of individual brain regions [27]. In addition, because there is significant overlap in networks underlying distinct cognitive processes, anatomical targeting alone may not be sufficient for decoding individual cognitive processes (Figure 2).

This distributed representation also has advantages: for example, precisely because these processes are represented in activity across multiple brain regions, cognitive state decoding from individual brain areas (e.g., by targeting 'key nodes' in the circuit) may be possible, although likely at lower accuracy than with multiregion recording methods. Similarly, because cognitive states are often expressed across large-scale networks, noninvasive modalities such as electroencephalography (EEG) may be able to capture low-dimensional signatures of arousal, alertness, or attention [28,29], and provide opportunities for scalable, low-resolution decoding that may complement higher-quality, anatomically precise intracranial methods while facilitating translation to ecologically valid settings beyond the laboratory.

Finally, signal integration for circuit organization is not a local process: cognitive states depend not only on local activity in individual brain areas but also on coordination and integration across

and act as a fundamental organization mechanism to create transient brain networks by facilitating cross-areal functional communication. (E) Neuromodulators such as dopamine and serotonin act through diffuse, multiareal modulation of synaptic transmission and are intimately implicated in cognitive function and dysfunction in neuropsychiatric disorders. ACC: anterior cingulate cortex; Amy: amygdala; BCI: brain–computer interface; dlPFC: dorsolateral prefrontal cortex; FEF: frontal eye fields; HC: hippocampus; Ins: insula; IPL: inferior parietal lobule; OFC: orbitofrontal cortex; PCC: posterior cingulate cortex; Str/NAc: striatum/nucleus accumbens; Thal: thalamus; TPJ: temporo-parietal junction; VCx: visual cortex; vmPFC: ventromedial prefrontal cortex.

regions, processes widely understood to depend on neurophysiological mechanisms that reflect these functional interactions, including neural oscillations.

Interregional coordination, oscillations, and mesoscale dynamics

The distributed organization of cognition has a direct consequence for cBCI feature design: decoders may need to estimate not only activity within a local population but also how activity is coordinated across regions. The majority of BCI efforts have focused on individual neuron spiking activity [5,10] or **high-frequency activity (HFA)** [12,30]. For cognitive states, these signals remain important, but they may need to be complemented by features that capture interregional activation and functional coupling, beyond local activity alone. Oscillatory dynamics provide one candidate mechanism for such coordination: the rhythmic synchronization of neuronal populations coordinates when and where information is transmitted, thereby potentially organizing information flow across distributed circuits [31]. The appearance of rhythmic activity reflects reversible organization of circuit-level neuronal ensembles, with different oscillatory patterns associated with different cognitive functions and circuits.

At the local scale, band-limited oscillatory power and aperiodic spectral structure, captured by **local field potentials (LFPs),** can describe the state of individual brain regions in a cognitive circuit. Importantly for decoding algorithms, there are multiple aspects of oscillatory activity at play: oscillations in different frequency bands (theta, 3–8 Hz; alpha, 8–12 Hz; beta, 13–30 Hz; and gamma, >30 Hz) appear predominantly in individual brain areas during different cognitive functions. For example, in attention, increased gamma activity in sensory cortices enhances the processing of attended stimuli [32], while alpha-band oscillations suppress irrelevant inputs [33]; prefrontal beta synchrony reflects top-down control [34]; and theta oscillations play roles in spatial navigation, memory, and cognitive control [35–38]. In addition to changes in oscillatory dynamics, changes in aperiodic spectral structure, reflecting shifts in excitatory–inhibitory balance, can track arousal and task engagement [28,39] and provide a complementary account of effects not directly attributable to band-limited power modulation. Overall, these mesoscale signals can capture local coordinated population activity and may therefore provide a more stable substrate for cBCI decoding than the activity of individual neurons alone, because they can remain behaviorally informative even as individual unit participation drifts [19,27,40–42].

In addition, given the distributed nature of cognitive processes, cBCIs may benefit from sensing cross-areal dynamics underlying functional connectivity rather than local activity alone. Oscillatory coherence is one candidate readout of such coordination because phase alignment between regions can create windows in which inputs arrive during more favorable phases of neuronal excitability [43], allowing distributed regions to exchange information effectively [43,44]. For example, gamma coherence plays a central role in visual perception [45], theta coherence between prefrontal and parietal regions coordinates distributed attention networks and cognitive control [36,46], beta coherence between the hippocampus and amygdala reflects emotional state [42], and theta synchrony-locked stimulation can induce hippocampal network connectivity changes [35]. These multiareal oscillatory patterns highlight both the need to sample neural activity from several brain areas and the importance of evaluating the oscillatory mechanisms underlying their functional coordination.

Local computations and large-scale network states are also coordinated, for example, through cross-frequency coupling mechanisms, which capture interactions between neural oscillations at different frequencies (i.e., slow-frequency phase to high-frequency amplitude). In this way, these mechanisms may integrate local population activity with the large-scale network dynamics critical for multiregional cognitive processes. Cross-frequency coupling is widespread in cortical

areas [47] and has behavioral correlates; for example, theta–gamma cross-frequency coupling organizes the sequential encoding of memory items [48,49], and prefrontal–amygdala theta coupling signals successful top-down control of affective responses [50]. For cBCIs, these features may improve recognition and decoding of cognitive states that depend on both local computation and global context.

Causal stimulation studies are critical because they can help dissociate the contribution of individual brain areas within circuits to behavior and cognition (see 'Clinical neuromodulation: The modulatory bridge' section). In the context of circuit organization, they can also test whether oscillatory features merely correlate with cognition or can be used to precisely manipulate cognitive circuits. While still limited to specific circuits and behaviors, recent closed-loop and phase-locked stimulation studies provide proof of principle that the oscillatory state can shape stimulation effects: theta phase-locked stimulation of the OFC demonstrates a causal role for hippocampus–orbitofrontal theta phase in decision-making [51], and closed-loop stimulation of the temporal cortex can improve memory [52].

Oscillatory phase-based communication schemes, however, should be viewed as one member of a broader family of temporal and population-level features. Functional communication can also depend on precise spike timing relationships and the temporal organization of activity bursts, which can similarly gate when information is transmitted or received, even in the absence of sustained coherence [48,53,54]. Moreover, interareal coordination may be expressed not only through timing or rhythmic phase relationships but also through the geometry of population activity—a population-based description of neuronal activity organization present in human invasive recordings [55]. In the context of cross-areal communication, low-dimensional communication subspaces may determine which patterns of neural activity are effectively exchanged across regions [16,56–58]. Under this view, understanding cross-areal communication dynamics will likely require models that integrate oscillatory metrics with denser population-level recordings, allowing researchers to capture how distributed circuits represent and transmit cognitive information.

Taken together, these mechanisms define a feature hierarchy for cBCIs. Local spiking and HFA can capture activity within sampled populations; oscillatory power in individual frequency bands and aperiodic activity can capture local mesoscale population activity and excitatory/inhibitory balance; coherence and phase relationships, as well as activity bursts and population geometry, can capture interregional coordination; and cross-frequency coupling can relate local computations to broader network states. cBCIs are, therefore, likely to benefit from incorporating oscillatory and phase-based representations rather than relying solely on firing rates or HFA power.

At the same time, this richer feature space creates an interpretability and generalization challenge. The same neural feature can participate in multiple circuits and underlie multiple cognitive processes, creating functional overlap that parallels the anatomical overlap discussed earlier. For instance, midfrontal theta can signal conflict monitoring in decision-making [59], sustained attention [60], top-down control during emotional regulation [61], working memory [36], flexible information encoding [37], and behavioral switching [62]. Similarly, beta oscillations have been associated with the maintenance of task rules in goal-oriented behavior [63] and, more generally, in cognitive control [46], status-quo maintenance [64], working memory maintenance [48], and mood fluctuations [42], in addition to a well-known role in motor preparation [65]. At the neuronal level, this challenge is partially driven by the mixed selectivity of individual neurons, particularly in the prefrontal cortex [66]: within a single individual, the same dorsolateral prefrontal cortex population can encode attention [24], working memory content [63], or reward variables [67,68].

The goal-dependent and time-varying expression of these features provides potential opportunities to disambiguate these anatomically and functionally overlapping states and highlights another critical characteristic of cognition, namely its dynamic nature.

The dynamic nature of cognition and the limits of static models

Cognition is inherently dynamic, both in neural implementation and behavioral manifestations. Mental states evolve across multiple timescales, from milliseconds (neural synchrony) to seconds (working memory maintenance) and minutes to hours (motivational or emotional fluctuations and circadian rhythms). This temporal complexity challenges traditional BCI approaches, which are based on relatively static decoders that map patterns of neural activity to behavioral outputs under the assumption of stable relationships between them. In motor BCIs, such static mappings are often sufficient because the representation of movement in the motor cortex is relatively stable across trials and time. Although issues with signal stability exist in motor BCIs, primarily related to recording longevity, neural representation drift, and biological processes such as gliosis around electrodes, these often reflect comparatively slow and largely unidirectional changes (signal decrease over time) and are therefore fundamentally different from rapidly shifting cognitive states.

Adaptive behavioral strategies in changing environments are characterized by rapid, context-dependent fluctuations in neural activity. Such flexibility requires the establishment of transient, reversible network states that can be configured to meet evolving task demands. For example, attentional states can be rapidly deployed and withdrawn through shifting frontoparietal connectivity, while working memory representations depend on the establishment of prefrontal ensembles that are reconfigured once they are no longer task-relevant [63], and transient beta oscillatory bursts support cognition and mental states [54,69]. This reversibility underscores the fluid nature of cognitive processes, in which neuronal ensembles are flexibly formed, maintained, and dismantled in response to internal goals and external contexts.

Other relevant factors operate over longer timescales. Cognitive and affective states fluctuate with circadian phases, hormonal states, stress levels, and other slowly varying physiological factors, and neural excitability may vary accordingly [70]. For example, circadian cortisol rhythms affect arousal and attention, while hormonal fluctuations (e.g., estrogen and progesterone) have been linked to changes in memory, emotion regulation, and network connectivity. cBCIs will therefore benefit from models that are sensitive to slow biological states and incorporate circadian and hormonal cycles [71].

This dynamism is also relevant for clinical applications. Neurological and psychiatric disorders, often accompanied by hallmark cognitive deficits, are increasingly understood not as fixed trait abnormalities but as evolving circuit disturbances in neural state transitions and network stability. Relevant cognitive and affective states, such as mood or anxiety levels, often fluctuate over minutes, days, or months [72]. In some cases, the ability to flexibly enter and exit adaptive states may be pathologically altered [73,74]. For example, excessive persistence of internally oriented default-mode activity may underlie ruminative states characteristic of depression [75]. In other cases, network instability manifests as rapid and maladaptive transitions between competing attractor states, such as quick shifts between manic and depressive episodes in bipolar disorder [76]. Under this view, psychiatric syndromes can be conceptualized as disorders of cognitive dynamics (i.e., failures of network flexibility) rather than only as static deficits. This notion has led to recent efforts to model latent structures in neural activity underlying cognitive and emotional states [41,77,78].

Finally, neural changes also continuously occur at the cellular level. For example, representational drift, whereby neurons alter their tuning or participation in functional ensembles over time [79], poses a challenge for the longevity of decoders built on spiking activity. At the synaptic level, synaptic weights evolve continuously through Hebbian plasticity mechanisms, even in the absence of overt learning, allowing neural representations to gradually adapt [80]. Because of these mechanisms, dynamic changes underlying cognitive states occur against a background of continual adaptation, which can be leveraged during calibration [78] or user adaptation to neuroprosthetics [79]. These processes can also be modeled explicitly to support continuous, unsupervised recalibration of the decoding models [81].

cBCIs will therefore need to account for the dynamic nature of cognition. One possibility is the development of new state-dependent, context-aware models capable of dynamically inferring which cognitive process is currently engaged before attempting to decode or modulate it. These context-sensitive models could be trained across tasks to derive candidate neural signatures for cognitive contexts (see 'The challenge of generalization and ground truth' section). In addition, multiareal recordings, in combination with latent models designed to capture network dynamics, may be better suited to reveal circuit-wide neural signatures of cognitive states and could support the development of robust cognitive state markers [82–84]. Hybrid, multimodal BCIs that incorporate nonneural data, such as movement, speech, or skin conductance measures, may help disambiguate anatomically or functionally overlapping brain states by providing time-resolved behavioral readouts [85]. Finally, decoding models will need to allow for continual learning and adaptation, such as Bayesian or reinforcement-learning frameworks capable of updating internal priors about brain–behavior relationships or behavioral switching between cognitive states [86].

This temporal adaptability will be essential not only for accurate decoding but also for safe and effective modulation of cognitive states—a central goal of cBCI technology, which will initially be focused on translational applications.

Beyond electrical activity: The role of neuromodulators and chemical signaling

While most current BCIs measure and manipulate electrical activity (spikes or LFPs), cognition is also profoundly shaped by neuromodulatory systems that operate through slower neurochemical signaling mechanisms [87]. Neurotransmitters such as dopamine, serotonin, acetylcholine, and norepinephrine act through a diversity of membrane receptors and intracellular signaling cascades, exerting complex influences on network excitability, plasticity, and signal-to-noise ratios rather than directly impacting cross-membrane ion flow and electrical signals. Through these mechanisms, neuromodulatory effects have broad, though incompletely understood, impacts on cognition. For example, dopaminergic prediction error signals from the midbrain drive reward learning and decision updating [88,89]; serotonergic tone modulates impulsivity and mood [90,91]; acetylcholine enhances sensory precision and attention [92]; and noradrenergic systems regulate arousal, attention, and vigilance [93].

Clinically, pharmacological manipulation of these neuromodulatory systems represents the cornerstone of neuropsychiatric treatments, reflecting the central role these systems play in regulating cognitive functions commonly affected in brain disorders such as mood, motivation, cognition, and behavioral flexibility [94]. For example, serotonergic agents, such as selective serotonin reuptake inhibitors, are widely used to treat depression, anxiety, and OCD [95,96]; dopaminergic antagonists are essential for managing psychosis in schizophrenia [97], while dopamine replacement therapies remain the gold standard for Parkinson's disease [98]; and noradrenergic and dopaminergic stimulants are highly effective for attention-deficit/hyperactivity disorder [99].

Despite their predominance, current pharmacological treatments for brain disorders have limited effectiveness, with substantial heterogeneity in treatment response and a significant proportion of patients failing to achieve remission [100,101]. Pharmacological interventions targeting neuromodulatory systems act broadly, influencing multiple brain regions rather than only those most directly implicated in a given symptom [102,103], which can lead to undesirable side effects and variable treatment responses across individuals [104,105]. Moreover, these approaches are primarily open-loop, providing ongoing modulation that is unable to adapt to moment-to-moment changes in brain state or behavior—a particularly salient limitation for disorders characterized by dynamic neural dysfunction [76,106]—and require trial-and-error prescribing that can take weeks or months to optimize. In contrast, adaptive neuromodulation approaches may address some limitations of current pharmacological interventions, including their lack of spatial, temporal, and mechanistic specificity [102,106,107].

Given the intimate interplay between neuromodulatory systems and cognitive function, cBCIs could benefit from the ability to sense and eventually modulate neurochemical states, potentially through multimodal sensors capable of detecting neurotransmitter concentrations. Electrochemical recording methods, such as fast-scan cyclic voltammetry, are capable of rapidly estimating neurotransmitter levels and have been deployed in human patients [108,109]. Incorporating these data streams could allow cBCIs to better infer broad cognitive states directly influenced by neuromodulatory states, such as motivational drive or emotional valence—dimensions of brain function whose electrical underpinnings are not yet well understood.

Such chemical sensing is particularly relevant for translational applications in psychiatry, where dysregulation of neuromodulatory systems underlies many disorders for which treatment options predominantly rely on the pharmacological manipulation of neuromodulatory systems. cBCIs capable of monitoring both electrical activity and neurotransmitter dynamics could, for example, detect and respond to shifts in dopaminergic tone to modulate dopaminergic release [110]. This integration of electrophysiological and neurochemical information represents a defining challenge and opportunity for cBCIs aimed at modulating cognition.

The challenge of generalization and ground truth

A central challenge for cBCIs is generalization across individuals, tasks, and contexts. Although motor BCIs already require patient-specific calibration, variability is likely to be even greater for cognition because the anatomical, physiological, and functional organization of cognitive circuits can differ substantially across individuals. Recent segmentation efforts in psychiatry further suggest that neurophysiological biotypes may map onto clinically meaningful subtypes [111,112], making one-size-fits-all decoders unlikely to perform well. Therefore, cBCI implementation is likely to require some degree of (if not full) patient-by-patient optimization, a common approach in BCI decoding and neuromodulation interventions [11,113–115]. An alternative, potentially complementary strategy would be to develop models that generalize more robustly across individuals, anatomical coverage, and recording sites (see below).

A second central challenge is the establishment of ground truth. Motor and speech BCIs benefit from overt behavioral outputs (i.e., movement and language output) that can be directly observed and used to train and validate decoders. By contrast, cBCIs seek to infer internal states that are only partially observable. Moreover, because different cognitive processes recruit overlapping networks, the correspondence between neural activity and behavior depends on state and context. Robust cBCIs will likely require explicit mappings between behavioral labels and neural activity, starting with training and validation datasets. Consequently, a key issue is the choice of behavioral labels to map neural activity onto.

Reaction times, error rates, self-reports, clinical scales, and **ecological momentary assessments (EMAs)** each capture part of the underlying cognitive process but do not provide a temporally precise or unambiguous index of latent cognitive states. This challenge is captured in broader frameworks in computational psychiatry and the Research Domain Criteria (RDoC) of the National Institutes of Mental Health (NIMH), which emphasize behaviorally relevant dimensions and computational processes (e.g., cognitive control, valuation, threat sensitivity, reward learning, and arousal) over coarse diagnostic categories or individual symptoms [1–4,116,117]. For cBCIs, the implication is that decoders should preferentially target these latent dimensions because they are more likely to map onto interpretable circuit mechanisms and clinically actionable biomarkers [117].

Despite their limitations, clinical scales, self-reports, and EMAs are widely used in clinical research and can be administered repeatedly to serve as intermittent anchors. In addition, task-based behavioral assays offer a pragmatic route to approximate ground truth. Carefully designed tasks, especially when combined with computational models of behavior, can reveal latent cognitive parameters at the individual level and, in some settings, capture clinically relevant dysfunction more precisely than symptom scales or self-reports alone [118,119]. These approaches can also link specific computations (e.g., learning rates, reward sensitivity, attentional biases, or control allocation) to physiological correlates, thereby formalizing interpretable model parameters as candidate biomarkers.

Together with standardized benchmarks that can maximize cross-site data collection compatibility, structured cognitive tasks provide the strongest and most interpretable labels. However, they are episodic and limited in scale; EMA, self-reports, and clinical scales suffer from similar limitations in terms of temporal resolution and collection sparsity. In contrast, continuous multimodal measures (audio/video, heart rate, wearables, sleep/wake, and other related physiological signals) provide weaker labels at any given moment but offer far greater temporal density, longitudinal coverage, and ecological validity. When interpreted jointly with neural recordings, these continuous streams can serve as informative proxies for internal state and context. Indeed, longitudinal ecological monitoring synchronized with neural data is already feasible in psychiatric and inpatient settings [71,120].

Given the relative strengths of these different approaches, behavioral labeling is best understood as a continuum of partial but complementary behavioral proxies for cognitive states rather than a single source of ground truth (Figure 3). A layered data collection strategy may, therefore, be most useful, with cognitive tasks and standardized benchmarks in some patients, intermittent anchors in many patients, and continuous multimodal context in most or all patients.

Real-world validity would further benefit from extending these approaches beyond the laboratory through longitudinal, ecologically valid assessments and common data modalities that can serve as anchors across datasets. This would allow models to capture how cognitive states unfold across naturalistic settings and clinically relevant timescales [121]. The key opportunity, therefore, is to link these data collection strategies: high-precision laboratory assays can anchor latent-variable models to mechanistically interpretable constructs, while large-scale naturalistic data can extend those constructs into real-world settings [85,113,122]. Task-based and ecological approaches are again complementary, the former providing construct validity and interpretability, and the latter providing scale, robustness, and relevance to everyday function [71,113,121].

Finally, recent **foundation model** approaches in neuroscience provide a potential methodological framework for addressing generalization across cognitive domains, individuals, and even biological levels of analysis [123]. Foundation models emerged from machine learning, particularly

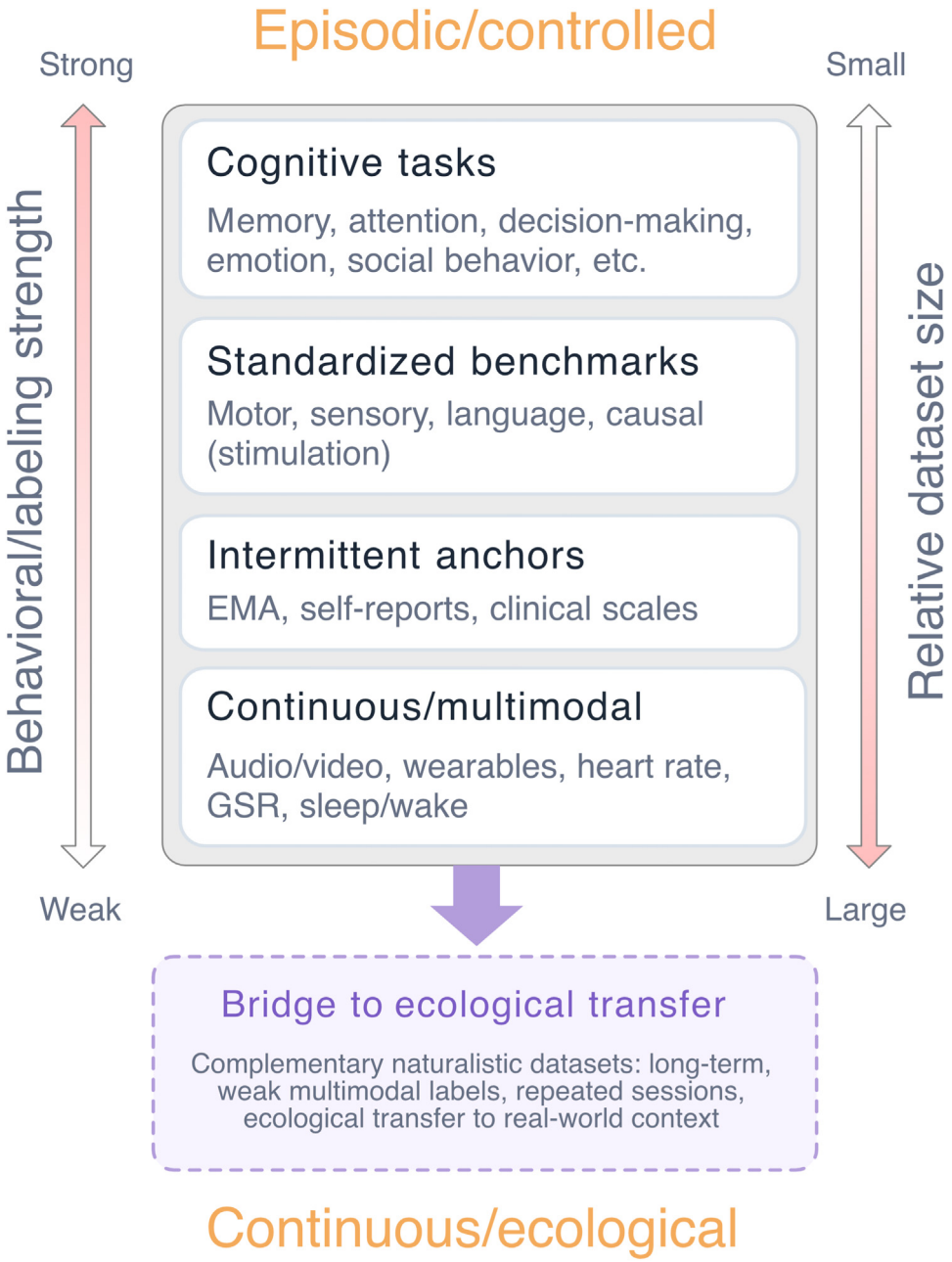

Figure 3. A layered behavioral labeling strategy and ecological bridge for cognitive BCI datasets. A central challenge in developing intracranial cognitive BCIs is the establishment of behavioral 'ground truth' for internal cognitive states. Behavioral data sources differ in label strength, temporal continuity, and scale. Standardized benchmarks provide the strongest and most controlled labels and are therefore valuable for cross-patient alignment, but they are episodic, limited in temporal resolution, and difficult to coordinate across sites. Cognitive tasks extend labeling to specific domains such as memory, attention, decision-making, and social cognition, and provide a foundation for computational modeling, but there is substantial variation in task design and modeling strategies. Intermittent anchors such as ecological momentary assessment (EMA), self-reports, and clinical scales provide sparse but behaviorally and clinically relevant observations outside tightly controlled task settings. Continuous multimodal recordings provide the weakest labels but offer the greatest temporal coverage, dataset scale, and repeated sampling, and are closest to ecological validity. A layered data collection strategy would combine these complementary approaches at different sampling densities

(Figure legend continued at the bottom of the next page.)

in natural language processing and computer vision, where large models are typically first pretrained on broad, heterogeneous datasets and then adapted or fine-tuned for more specific downstream tasks. In this sense, a foundation model represents a significant improvement over decoders built for one subject, task, or behavioral label by providing general-purpose representations designed to learn low-dimensional structures that can generalize and be reused across related problems. Intracranial foundation models may therefore represent a particularly relevant intermediate step for cBCI development. Rather than fitting separate subject- or task-specific decoders, these models would aim to learn low-dimensional, transferable representations from large, heterogeneous neural datasets. Recent work in animal and human neuroscience suggests that this approach can capture shared neural structures across subjects, recording contexts, and modalities, enabling stronger transfer to novel stimuli or downstream tasks [124–127]. Similar principles could prove valuable for cBCIs, where training data will be sparse within any single individual and heterogeneous across patients, sites, and tasks. Models trained under these conditions may be able to identify latent neural structures underlying specific aspects of cognition or disambiguate between them (Figure 4).

Building such models will require at least two advances. First, training these models will require larger, better-standardized, and better-annotated multisite intracranial datasets. In this context, variation across subjects, recording modalities, tasks, etc., is a strength rather than a weakness because it provides the necessary variability for models to learn representations that generalize. Coordinated data collection and behavioral label standardization across sites, including common scales, benchmarks, and data acquisition protocols, would facilitate the accumulation of large datasets that will likely be essential for model training and difficult to collect at individual sites (Figure 3). Emerging standards and open datasets can provide an important starting point, but current public datasets remain limited in cohort size, task breadth, and behavioral annotation labels compared to what true foundation-model training is likely to require—likely tens or hundreds of thousands of hours across hundreds of patients [128–130]. Second, model development will likely need to incorporate recent advances from both animal models [124] and dynamical systems modeling of neural population activity, including methods that explicitly dissociate behaviorally relevant dynamics from other neural or input-driven components [41,77,131,132]. Despite these challenges, these developments suggest a plausible path toward cBCIs that generalize more effectively across individuals and contexts while remaining grounded in interpretable neural mechanisms.

Clinical neuromodulation: The modulatory bridge

In parallel with decoding-based BCIs, clinical neuromodulation techniques represent a complementary translational pathway centered on the direct modulation of neural activity rather than its decoding. Established invasive methods, such as **deep brain stimulation (DBS)**, **reactive neurostimulation (RNS)**, and **stereoencephalography (sEEG),** provide safe acute or chronic modulation of brain activity in patients with diverse neurological and psychiatric conditions (Figure 5) [133]. These are complemented by established noninvasive methods, such as **transcranial magnetic stimulation (TMS),** which are undergoing refinement [133,134], as well as novel noninvasive approaches, such as **low-intensity focused ultrasound (LIFU)**, currently being developed [135]. These neuromodulation approaches are more commonly

(common standardized benchmarks in all patients, cognitive tasks and intermittent anchors in many patients, and multimodal contextual recordings in all patients), allowing strongly labeled, controlled data to be linked to longer-duration, more naturalistic observations. A complementary naturalistic dataset would serve as a bridge to ecological transfer, supporting the transition from laboratory-defined constructs to real-world cognitive and clinical state tracking over clinically relevant timescales (months to years). BCI: brain–computer interface; GSR: galvanic skin response.

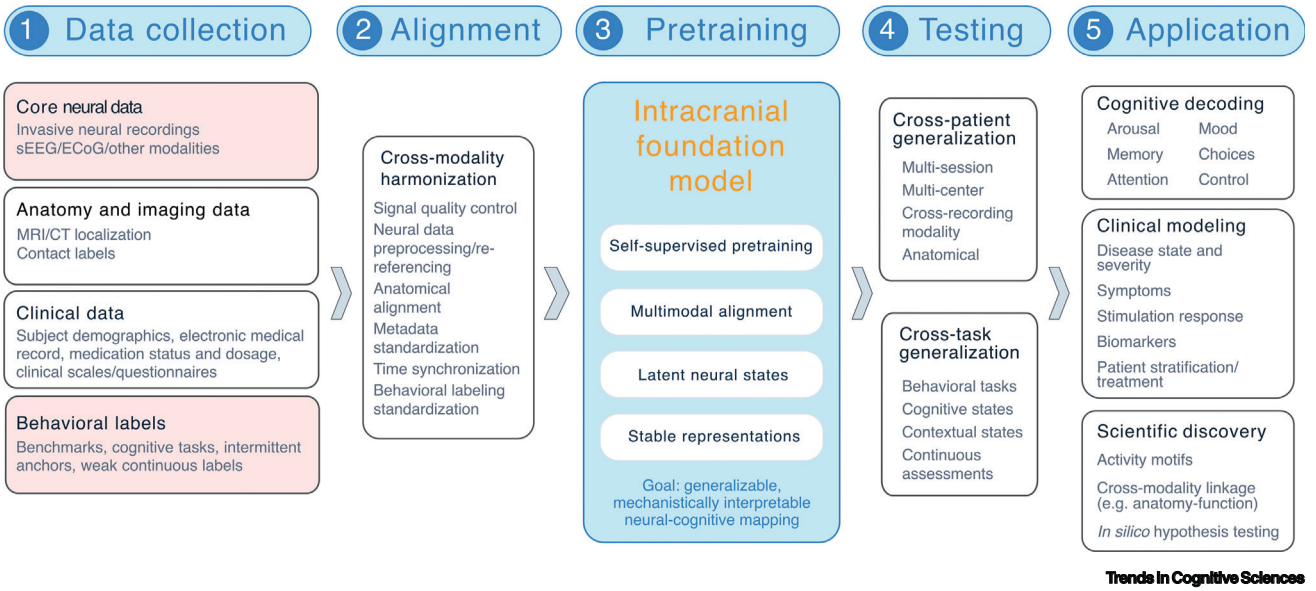

Figure 4. Intracranial foundation models for cognitive BCIs. Foundation models aim to learn low-dimensional, transferable representations from large, heterogeneous neural datasets to capture shared neural structure across subjects, recording contexts, and modalities, thereby enabling generalizable models. Foundation models for cBCIs will likely require broad, heterogeneous datasets that integrate neural activity with anatomical, clinical, and behavioral data. (1) Large datasets provide the diversity needed to distinguish general principles of brain function from idiosyncratic characteristics of recordings, patients, or experimental paradigms. (2) Alignment is the critical step that makes such diversity usable: signals, anatomy, metadata, timing, and labels must be harmonized, and the data must be preprocessed and standardized. (3) Pretraining then uses self-supervised learning to identify latent neural structure and learn representations that are stable across patients, sessions, and recording contexts. (4) Testing assesses whether the resulting representations support robust performance across patients and tasks. (5) Downstream applications use the same pretrained representations to support a variety of applications, including cognitive decoding for cBCIs, clinical modeling, and mechanistic *in silico* discovery. BCI: brain–computer interface; cBCIs: cognitive brain–computer interfaces; CT: computed tomography; ECoG: electrocorticography; sEEG: stereoencephalography.

used in neurological disorders (e.g., movement disorders and epilepsy), where clearly defined biomarkers can guide implantation and therapeutics [136], but are increasingly expanding to psychiatric conditions [137], including major depressive disorder [113,138] and OCD [139,140], in which targeted electrical stimulation can alter pathological network dynamics, restore function, and yield robust therapeutic outcomes.

Beyond their clinical use, neuromodulation tools are increasingly used in human cognitive neuroscience research, enabling causal interrogation of the neural substrates of attention, memory, emotion, and decision-making. This work provides a more comprehensive understanding of the neurobiological basis of human cognition and can help identify key targets for stimulation. This type of evidence is especially important for the implementation of cBCIs, given the distributed nature of cognitive processes [18–20,141]. Controlled experimental tasks, in combination with intracranial recordings, allow researchers to study the electrophysiological activity underlying human thought and behavior [19,37,49,55,142–145]. When combined with stimulation approaches, these tasks also provide valuable causal evidence for the role of individual brain areas [146–148]. In medial temporal and prefrontal–limbic circuits, for instance, invasive stimulation approaches have been demonstrated to induce short-term plasticity [149] and network connectivity changes [35], and can enhance or disrupt memory encoding [35,52] and modulate mood [40,150], providing rare causal evidence linking specific circuits to mental functions. In some cases, causal approaches can build on and refine insights from distributed encoding by testing whether brain areas that show similar activity patterns make distinct causal contributions (see, for instance, [151] followed by [152] for an example in social behavior).

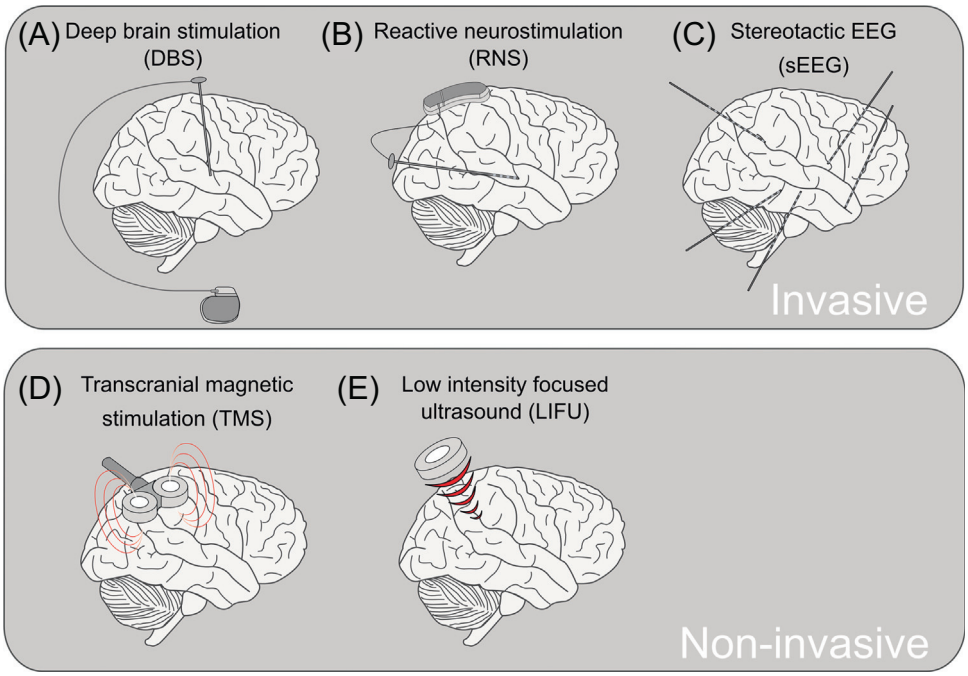

Figure 5. Clinical neuromodulation methods. Multiple invasive and noninvasive stimulation methods are available for diagnostic and therapeutic applications. (A) Deep brain stimulation is commonly used to treat the symptoms of neurological disorders, including movement disorders such as Parkinson's disease and dystonia, and increasingly for psychiatric conditions such as depression and OCD. (B) Reactive neurostimulation is used for chronic seizure prevention in patients with drug-resistant epilepsy. (C) Stereotactic EEG is used primarily for the acute recording and localization of epileptic seizures, although electrical stimulation can also be delivered and is routinely used in the clinic to evaluate cortical function. (D) Transcranial magnetic stimulation enables noninvasive modulation of superficial brain regions using magnetic pulses. (E) Low-intensity focused ultrasound can modulate deep brain activity using focused acoustic waves. EEG: electroencephalography; OCD: obsessive–compulsive disorder.

Together, these open-loop neuromodulation approaches serve as bidirectional research interfaces, bridging clinical neurosurgery and cognitive neuroscience.

Although neuromodulation is a natural candidate effector for translational cBCIs, the wide variety of stimulation settings (e.g., waveform, frequency, amplitude, pulse width, and timing) and the resulting combinatorial explosion in parameter combinations present a significant challenge: not only whether or where to stimulate, but also how. This problem is compounded by the multiareal nature of cognitive processes (see 'From localized to distributed neural representation' section) and by the relative lack of knowledge regarding the effects of stimulation on different neuronal populations and dynamics [153,154]. Fast exploration of stimulation parameters may provide a starting point to narrow the vast space of possibilities [155,156]. *In silico* testing may present a valuable opportunity to try and optimize stimulation strategies in a fast and safe way, complementary to *in vivo* approaches [157]. Alternatively, control models derived from engineering offer a principled framework for navigating the high-dimensional stimulation parameter space and linking stimulation patterns to their effects on underlying neural dynamics. Rather than exhaustively searching over parameter combinations, these models aim to assess how different stimulation patterns modulate low-dimensional neural state space trajectories and determine policies that steer activity toward desired cognitive states [158–160]. This approach naturally extends to network, rather than single- or multi-site modulation, making it attractive for the

modulation of distributed cognitive networks. In addition, these control-based frameworks can account for the state dependence of stimulation effects (see 'Closed-loop neuromodulation for cBCIs' section), enabling adaptive interventions that evolve alongside neural dynamics [40,161].

From an implementation standpoint, neuromodulation efforts also provide a regulatory framework for cBCI development. Because their clinical infrastructure provides explicit safety profiles and therapeutic endpoints, neuromodulatory methods could provide a regulatory foundation for cBCIs that build on the same principle of targeted, circuit-level modulation for clinical applications. This foundation could support the development of adaptive, closed-loop control of complex cognitive and emotional states.

Closed-loop neuromodulation for cBCIs

As discussed, most current neuromodulation systems operate in open-loop mode: they deliver stimulation according to preprogrammed parameters without real-time adaptation. In contrast, current motor BCI systems have traditionally focused on recording and decoding brain activity. Therefore, these two complementary fields have largely developed around opposite unidirectional pathways: brain-to-device in BCIs, and device-to-brain in neuromodulation.

In recent years, closed-loop approaches have begun to emerge in motor restoration, with noninvasive spinal cord stimulation used as the final effector to restore movement [162,163] and tactile feedback [164]. This provides initial evidence for closed-loop effectiveness in BCI. Arguably, the development of cBCIs will also require both decoding and stimulation, primarily by means of neuromodulation, in a single closed-loop architecture (Figure 6). More specifically, the different features of brain activity underlying cognition, including distributed network dynamics, temporal variability, oscillatory coordination, and representational flexibility, mean that cBCIs cannot rely on the static, unidirectional architectures that define current motor BCIs or open-loop neuromodulation systems (Figure 7). Instead, closed-loop control, in which neural and behavioral states are continuously recorded, decoded, and used to guide stimulation in real time, will be necessary.

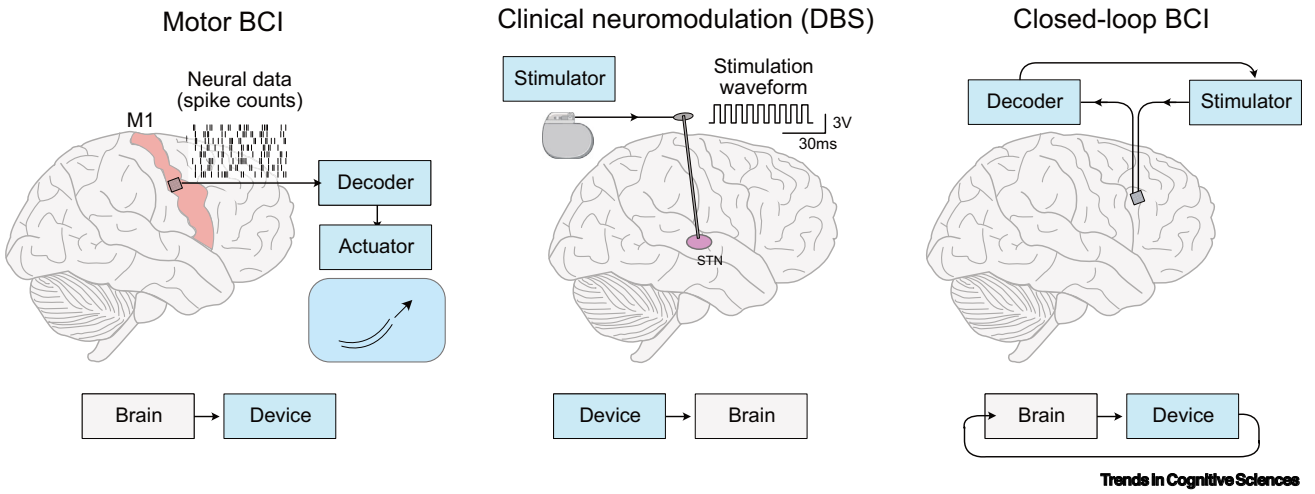

Figure 6. Closed-loop neuromodulation for cognitive BCIs. Cognitive BCIs will build on modeling and decoding approaches commonly used in motor BCIs (left) and on clinical stimulation methods used in an open-loop manner (center). The resulting closed-loop architectures (right) will be uniquely suited for cognitive state decoding and modulation. BCI: brain–computer interfaces; DBS: deep brain stimulation.

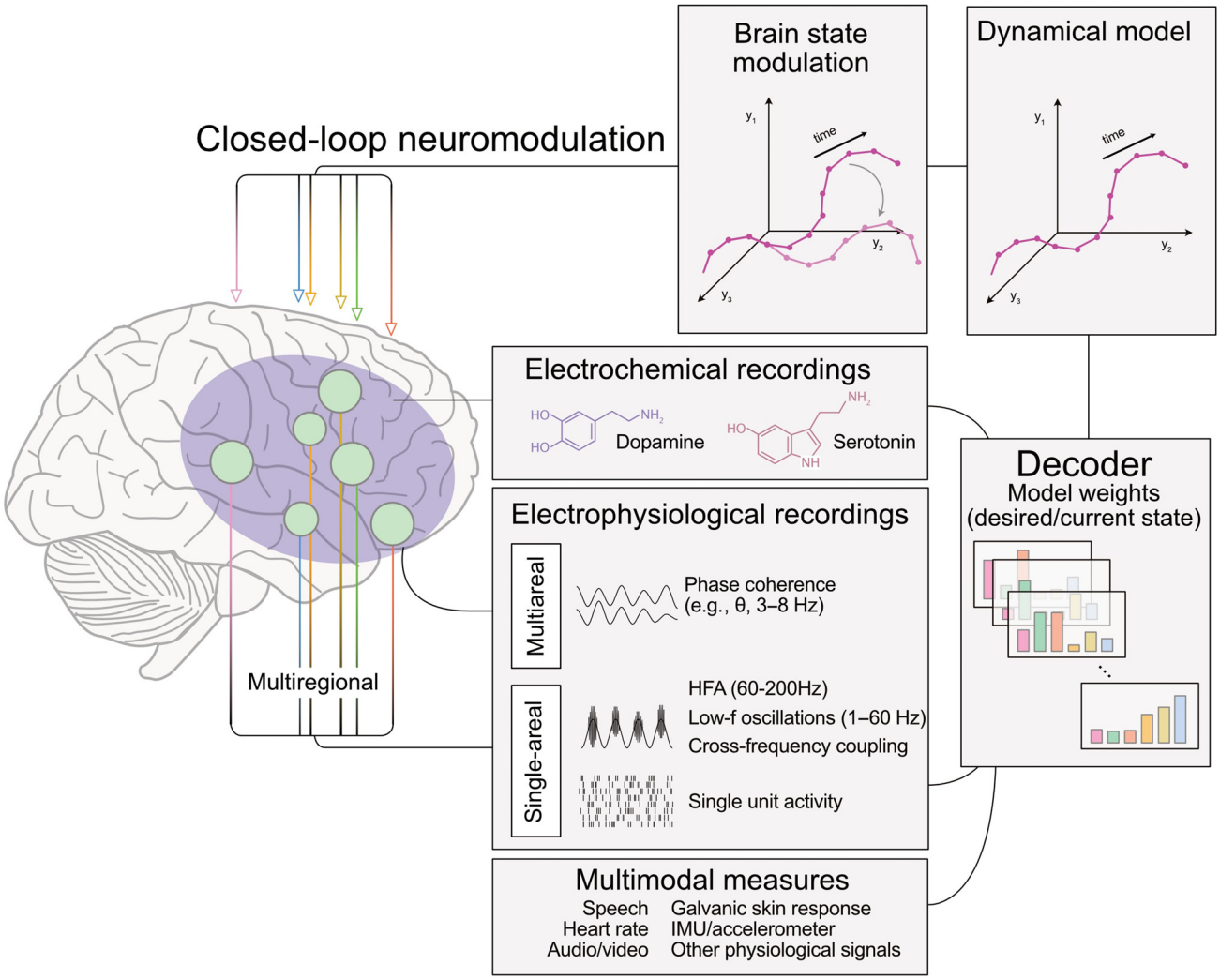

Figure 7. Closed-loop implementation for cognitive BCIs. Cognitive BCIs will need to incorporate features not currently used in motor BCIs to adequately capture the neural dynamics underlying cognitive states, given the differences in the neural bases of these processes. Multiareal, multimodal recordings could incorporate activity from several key brain regions, including single-neuron spiking activity, oscillatory measures (e.g., coherence and cross-frequency coupling) that capture both local and interregional population dynamics, and neurochemical estimates of neuromodulators (e.g., dopamine and serotonin) concentrations. In addition, multimodal and peripheral physiological signals (e.g., audio, IMU/accelerometer data, and other contextual measures) could provide complementary information and help disambiguate cognitive states. Novel decoders will need to be able to dynamically estimate the current cognitive state and guide it toward desired target states through targeted neuromodulation. BCI: brain–computer interfaces; HFA: high-frequency activity; IMU: inertial measurement unit.

From a practical standpoint, closed-loop BCI is likely to take decoding algorithms developed in unidirectional motor BCI as a starting point for development and progressively incorporate some of the defining features of cognitive processes, such as oscillatory dynamics and neuromodulatory states [41,51,52,77]. In addition, novel models for cBCI would benefit from incorporating sensitivity to behavioral context and goals to minimize interference between neurally overlapping cognitive processes. At the same time, because the initial goals of cBCI are therapeutic—namely, restoration of damaged or lost cognitive abilities in brain disorders—near-term development will likely build on neuromodulation approaches such as DBS, potentially by modeling cognitive symptoms not managed by current approaches, incorporating multiareal recording

methods, and adding dynamic modeling to account for disease progression and dynamics in service of closed-loop implementations.

Some initial efforts in closed-loop neuromodulation are already at various stages of development in neurology (e.g., RNS in epilepsy) [165], motor BCI (through spinal cord stimulation or closed-loop haptic feedback), and psychiatry [106]. Generally, combining sensing and stimulation within a single architecture is considered superior to open-loop implementation [106,166], further strengthening the notion that the convergence of neuromodulation and BCI design in closed-loop approaches will be necessary.

A further translational question concerns the training burden and durability of benefit. Some cBCI applications may require substantial calibration or user learning, although the development trend is toward relatively rapid deployment through adaptive models [81,114,167]. Likewise, early improvements after cBCI implantation could reflect transient control, longer-lasting plasticity, or a combination of both [52,149,167]. Distinguishing these possibilities and quantifying how performance changes across repeated use, training schedules, interruptions, and evolving clinical states will be essential for evaluating the burden of cBCIs and determining whether they can produce durable functional benefits outside tightly controlled settings [40,71,81,106,113,165].

A critically relevant observation is that oscillatory states not only reflect ongoing neural activity but also govern the brain's response to stimulation. The effects of electrical stimulation vary as a function of ongoing oscillatory phase, frequency content, and broader network activity. For example, phase-locked stimulation paradigms have shown that identical stimulation pulses can produce markedly different physiological and behavioral outcomes depending on the timing relative to intrinsic rhythms, particularly in the theta band [35,168–170]. Recent studies have provided proof of principle for the utility of oscillation-informed closed-loop decoding [26,161], further demonstrating that closed-loop BCIs will benefit from estimating the instantaneous dynamical state of neural circuits, such as oscillatory phase, to inform control and stimulation policies.

Finally, biomarker-driven neurofeedback and other feedback modalities may also complement direct stimulation, particularly when the goal is to promote self-regulation or reduce the invasiveness of neural recording methods [171,172]. In these approaches, decoded neural signals are mapped onto sensory feedback (e.g., visual presentation on a screen), enabling individuals to learn to modulate their own brain activity [173]. Prior work using EEG and fMRI neurofeedback suggests that such control can, in some contexts, improve attentional performance, emotional regulation, and clinical symptoms, although effects remain heterogeneous across paradigms and disorders [174–176]. More broadly, incorporating behavioral and physiological signals (e.g., pupil diameter, heart rate variability, or task performance) into multimodal feedback loops may further enhance scalability and real-world applicability, extending cBCIs beyond purely invasive stimulation-based interventions [85,173].

Concluding remarks

The development of cBCIs represents an imminent new direction for BCI. Building on well-validated BCI and neuromodulatory approaches, which together provide starting points for cognitive decoding algorithms and modulation strategies, respectively, the field has already begun to move toward clinical applications [13] that prelude the development of full cBCI technology.

cBCI implementation will require leveraging the differences between the neurobiological implementation of cognitive versus motor processes, the dominant space for BCI application.

Outstanding questions

How do distributed, overlapping, and dynamically reconfiguring neural circuits causally generate specific cognitive states?

Which multimodal biomarkers can robustly decode cognitive states across individuals, contexts, and timescales?

How should cBCIs incorporate state dependence arising from ongoing brain dynamics (e.g., oscillatory phase) and nonneural measurements?

What should count as ground truth for internal cognitive states?

How can we combine controlled tasks, ecological momentary assessments, clinical scales, and multimodal continuous signals to assess cognitive state?

Can cBCIs generalize across individuals, tasks, recording sites, contexts, and cognitive modalities?

Can generalization be achieved without sacrificing patient-specific precision?

How can stimulation policies be safely optimized for distributed cognitive circuits, given the very high-dimensional stimulation parameter spaces?

How will intracranial foundation models be useful for cBCI? How can we collect, label, and homogenize the large intracranial datasets that will be necessary to build them?

Will cBCIs produce durable, real-world functional benefits outside the laboratory?

Unlike motor commands, cognitive functions emerge from distributed, overlapping networks shaped by oscillatory coordination, neuromodulatory influences, and substantial sharing of biological substrates across domains. Defining actionable and robust biomarkers for cognitive processes will require multiareal, multimodal sensing, adaptive algorithms, and control frameworks capable of decoding rapidly fluctuating cognitive states in real time. It will also require an intrinsically dynamic and chemically modulated neurobiological view of brain function, as well as a deeper integration of existing BCI technologies with neuromodulatory methods already in clinical use.

From a technical standpoint, although challenges exist (see Outstanding questions), many of the necessary components for developing cBCIs already exist: high-density single-region and distributed multiareal invasive recordings, real-time spectral signal analysis and feedback tools, a growing array of safe and flexible neuromodulation approaches, subsecond neuromodulator measurements, and increasingly sophisticated modeling frameworks with natural extensions into closed-loop control and an increased ability for generalization. A central challenge will be to distill overlapping neurobiological features underlying cognition into domain-specific and disease-specific biomarkers that can be reliably decoded and safely modulated, and to develop integrated frameworks that incorporate the essential dimensions of the neurobiology underlying cognitive states: network dynamics, oscillations, neuromodulation, and dynamic context.

In summary, cBCIs will represent not simply an extension of motor and language BCIs, but a fundamental expansion of our ability to understand and modulate human brain function. By integrating and refining already existing approaches, cBCIs may ultimately become a new class of neurotechnology capable of identifying and correcting dysfunctional brain states as they dynamically unfold, opening a path toward mechanistically precise interventions for brain disorders and a deeper causal understanding of human cognition.

Acknowledgments

I would like to acknowledge members of the Icahn School of Medicine departments of Neuroscience, Neurosurgery, and Neurology; the Center for Advanced Circuit Therapeutics; the Friedman Brain Institute; and the Saez Lab for fruitful discussions.

Declaration of interests

The author declares no competing interests.

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