Symposium

Cross-Species Approaches to Understanding Human Cognition

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Recent methodological advances including high-density intracranial recordings and population-level analytical frameworks enable deeper and more direct integration between animal models and human neuroscience. As a result, there is an increasing need for standardized behavioral paradigms and circuit-level analyses that can be used in both animals and humans. Cross-species approaches provide crucial foundations for treating neurological and psychiatric disorders. Parallel experiments validate therapeutic targets and enable development of mechanistically grounded behavioral biomarkers for early disease detection. Here, we provide a brief review of recent findings that have direct implications for attention deficits, memory impairments, and decision-making dysfunctions. By linking mechanism to behavior, these approaches guide targeted interventions and effective clinical translation. While challenges remain, these integrated approaches are advancing our understanding of the mechanistic foundation of human cognition.

Introduction

Over the past two decades, systems neuroscience has experienced a profound technological renaissance. In model organisms, the refinement of optogenetics and chemogenetics has provided unprecedented causal control over defined neural circuits (Deisseroth, 2015). This allows researchers to manipulate activity with millisecond precision and cell-type specificity. Concurrently, the development of high-density extracellular electrophysiology, such as Neuropixels probes, has enabled the simultaneous recording of thousands of individual neurons. Researchers can now map multisite population dynamics in rodents and nonhuman primates (NHPs) during complex behaviors, yielding high-resolution insights into the underlying microcircuitry and population coding (Ruff and Cohen, 2019; Urai et al., 2022).

Parallel to fundamental animal research, human neuroscience has been revolutionized by intracranial recordings in neurosurgical patients. Critically, modern clinical research, facilitated in part by programs such as the NIH Brain Research through Advancing Innovative Neurotechnologies (BRAIN) Research Opportunities in Humans (ROH), now capitalizes on highly detailed macro- and microscale recordings to uncover dynamical network processes that underlie human cognition (Courellis et al., 2024; Khanna et al., 2024; Leonard et al., 2024; Woolnough and Tandon, 2025; Bhaya-Grossman et al., 2026; Zubair et al., 2026). Techniques such as stereoelectroencephalography (SEEG) and high-density electrocorticography (ECoG) provide a rare, direct window into large-scale network dynamics, enabling the precise mapping of distributed cortical networks underlying complex sensory and cognitive processing with spatial resolutions that vastly exceed other noninvasive tools (Hamilton et al., 2021). Simultaneously, the integration of microelectrode arrays and specialized depth electrodes, some with stimulation capabilities (Topalovic et al., 2023), are beginning to allow neuroscientists to observe individual single-unit firing and population coding in humans (Paulk et al., 2022). Together, this dual micro-macro approach enables the detailed tracking of dynamic higher-order cognitive tasks directly at both the single-neuron and network-wide levels.

Despite these parallel revolutions, a challenging translational gap persists. Integrating the highly controlled findings from animal models with the nuanced cognitive data from human subjects remains a central challenge (Krakauer et al., 2017). Historically, animal and human research have operated in distinct silos, constrained by differing methodologies and analytical scales. While rodent models offer unparalleled access to circuit-level interventions, their behaviors often require significant extrapolation to relate to human cognition. Conversely, human intracranial studies are inherently opportunistic. They are restricted to clinically determined brain regions in neurosurgical patients, where the tissue itself may be affected by the underlying disease. NHPs offer a particularly promising solution to this divide. Their highly developed prefrontal cortex and complex social behaviors make them a vital translational bridge (Roelfsema and Treue, 2014; Constantinidis et al., 2018).

Received May 27, 2026; revised Aug. 8, 2026; accepted Aug. 11, 2026.

Author contributions: E.A.B., M.R.C., C.C., S.K., P.H.R., and M.S. wrote the paper.

The contributions of the National Institutes of Health (NIH) author(s) are considered works of the United States Government. The findings and conclusions presented in this paper are those of the author(s) and do not necessarily reflect the views of the NIH or the US Department of Health and Human Services. This work was supported by the NIH ORIP (P510D010425, E.A.B.; P50MH132642, S.K.; R01EY017699, S.K.; R01MH137624, S.K.; R21 MH139624, C.C.; RF1MH132789, P.H.R.).

The authors declare no competing financial interests.

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https://doi.org/10.1523/JNEUROSCI.1201-26.2026

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To maximize this potential, researchers are leveraging highly precise genomic and physiological mapping in rhesus macaques to model human cognitive architectures (DeCasien et al., 2024). Without a unified framework that fully exploits this bridge, the field risks accumulating vast but disconnected datasets. This fragmentation prevents a cohesive, cross-species understanding of brain function and slows therapeutic development.

To overcome these barriers, researchers are shifting toward methodologies explicitly designed for cross-species integration. A critical component is the development of standardized behavioral paradigms, particularly touchscreen-based systems and naturalistic visual tasks deployed across species (Jutras and Buffalo, 2010; Goudar et al., 2024). By aligning behavioral output and sensory stimuli, researchers can more accurately isolate the underlying neural computations. NHPs and humans are especially well suited to this approach because they share a remarkably similar oculomotor system, as both species rely on saccadic eye movements and active visual exploration to sample the environment, allowing eye tracking and gaze-contingent paradigms to serve as a direct behavioral bridge between species (Meister and Buffalo, 2016; Buffalo, 2025). This shared sensorimotor repertoire provides a uniquely precise, standardized foundation for advanced data integration and comparative analysis.

Equally important is the adoption of shared analytical frameworks. The sheer volume of high-dimensional neural data necessitates advanced computational approaches like dimensionality reduction and latent state space modeling (Cunningham and Yu, 2014). Projecting neural population activity into a shared, low-dimensional manifold helps identify conserved neural dynamics across species, a technique particularly effective in analyzing visual and attention networks (Ni et al., 2018; Goudar et al., 2023; Rueckemann et al., 2025; Zhang et al., 2026). These shared analytical spaces allow for direct mathematical comparisons of how different species encode cognitive variables. This approach transforms qualitative behavioral comparisons into quantitative, rigorous neural alignments.

Translating findings across species also requires a deep understanding of anatomical homology. Despite differences in cortical organization across mammals, fundamental cortical-subcortical circuits, particularly those linking the frontal cortex with the amygdala and reward-related structures, remain highly conserved (Rudebeck and Izquierdo, 2022). This conserved circuit architecture enables mechanistic insights derived from animal models to inform the interpretation of human intracranial recordings. Modern comparative frameworks use abstract, multidimensional "connectivity fingerprints" to identify homologous brain regions across the primate lineage by comparing their patterns of structural and functional connectivity (Mars et al., 2018, 2021).

Furthermore, identifying conserved neural signatures serves as a universal language for cross-species comparison. For instance, specific oscillatory dynamics, such as hippocampal theta- and gamma-band synchrony during spatial and visual navigation, provide a direct template for interpreting human temporal lobe recordings (Jutras et al., 2009, 2013; Goyal et al., 2020; Kastner et al., 2020). When combined with targeted neuromodulation, researchers can manipulate these shared rhythms to test if driving specific network frequencies yields predictable cognitive changes across species (Lozano-Soldevilla and VanRullen, 2019; Alekseichuk et al., 2020).

This minireview highlights the innovative approaches successfully bridging the translational gap between model organisms and human cognition. Coinciding with themes from a dedicated symposium at the 2026 Society for Neuroscience meeting, this review expands on the points highlighted above and synthesizes the latest advances in cross-species integration driven by leading researchers in the field.

Behavior Serving as Glue for Cross-Species Comparisons

Behavior is not merely an output of neural activity; it is the common currency that allows observations made at different levels and in different species to be translated into one another. Because it is challenging to obtain identical neural measurements across species, shared behavioral signatures provide a foundation for linking basic and translational neuroscience. Similar behavioral patterns permit meaningful comparisons even when the underlying experimental approaches differ.

NHPs are an ideal animal model for linking behavior to neural substrates and functional frameworks that relate to human behavior and brain function. In contrast to rodents and other mammals, both humans and NHPs explore their environment primarily through vision and have thus highly developed visual systems that occupy ~40–50% of their brains (Krubitzer and Kaas, 1990; Felleman and Van Essen, 1991). A primate-specific characteristic is the ability to make saccadic eye movements about every 200–250 ms to bring the visual information of interest into central or foveal vision. Foveal vision is in many regards the foundation of primate behavior enabling a variety of behaviors such as face perception as a basis for social communication (Haxby et al., 2002), eye–hand coordination for fine motor control (Goodale and Milner, 1992), as well as reading and literacy as human-specific cognitive inventions (Dehaene and Cohen, 2007).

NHPs express complex behaviors in many domains related to perception, cognition, and action that are relevant to human behavior. In one approach to use the behavior-as-glue principle as a link for research in NHPs to human behavior and brain function, classical tasks from human cognitive psychology served to establish similar behavioral patterns in NHPs. One of the most established tasks in the attention literature is the Eriksen Flanker task (Eriksen and Eriksen, 1974). In this task, a visual cue guides attention to a specific field location, where a target (e.g., a specific shape) needs to be discriminated. The target is flanked by distractor stimuli that are either identical or different in shape as compared with the target. Human participants typically show a reaction time cost when the target is flanked by dissimilar distractors, which is also true for NHPs trained on an Eriksen Flanker task (Saalmann et al., 2012; Martin et al., 2019). Importantly, NHPs also exhibit temporal dynamics in their behaviors similar to those observed in humans during sustained attention, suggesting that they sample their environment in theta-rhythmic cycles that are characterized by alternating periods of relatively enhanced or diminished perceptual sensitivity at the attended location (Fig. 1*A*; Fiebelkorn et al., 2019). Comparative intracranial electrophysiology in NHPs and human epilepsy patients indicate that these behavioral fluctuations are related to intrinsic brain rhythms in the theta range (4–6 Hz), measured in the local field potential (LFP; Fiebelkorn et al., 2018; Helfrich et al., 2018). These rhythms organize neural activity into two alternating attentional states—one state promoting sampling visual information at the presently attended location and the other state promoting shifting attention to another location in the visual field. These examples illustrate that common behavioral paradigms enable substantive comparisons that allow detailed circuit-level measurements in NHP to be

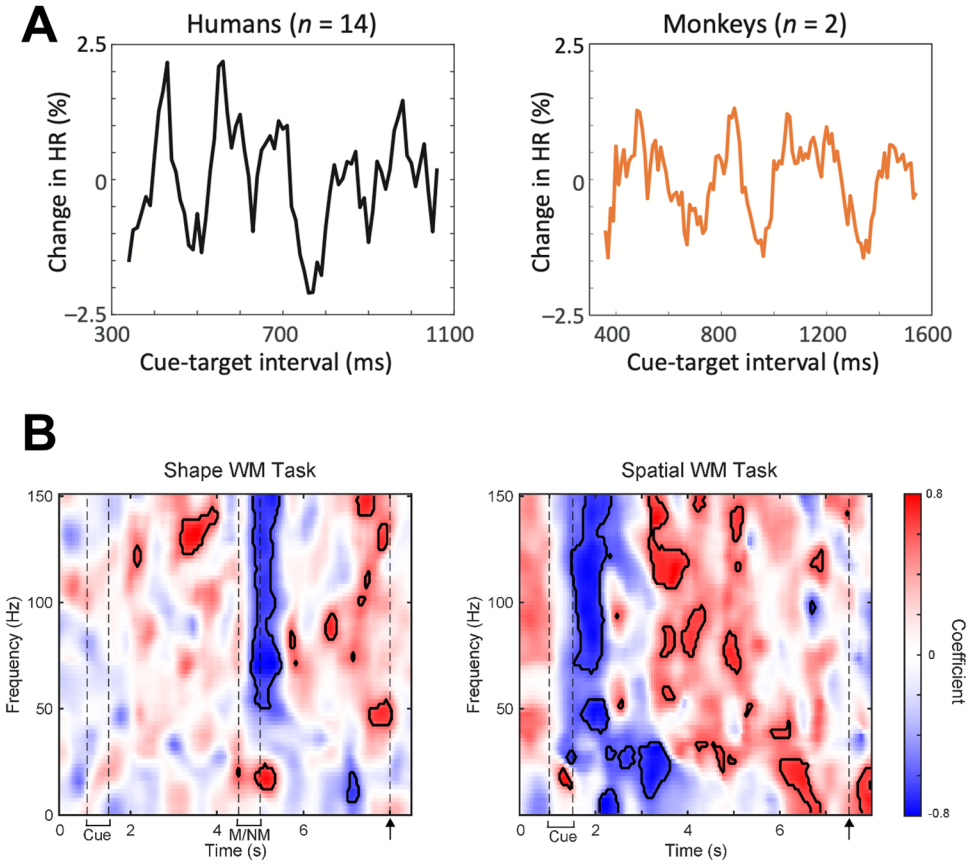


Figure 1. Behavior-as-glue. ***A***, Behavioral performance in a spatial attention task as a function of time from the cue for human (adapted from Fiebelkorn et al., 2013) and monkey participants (adapted from Fiebelkorn et al., 2018). For both species, behavioral performance is characterized by theta-rhythmic cycles. ***B***, Time-resolved, frequency-power correlation between activity in the human prefrontal cortex recorded during performance of a shape (left) and spatial working memory task (right) and performance of the Wisconsin Card Sorting Task (WCST). Vertical lines indicate the times of appearance of task stimuli and subject response (arrow). The spatial and shape WM tasks were designed in monkey neurophysiological studies. This analysis reveals what aspects of neural activity recorded in laboratory tasks predicts the performance in WCST, an instrument used in clinical studies to evaluate cognitive flexibility. From Jurva et al. (2025).

interpreted in the context of human cognition and cognitive dysfunction in disease.

In some cases, behavior itself provides the bridge between basic and translational neuroscience. Because it is difficult to obtain identical neural measurements in humans and animal models, shared behavioral signatures can anchor comparisons across species. One example comes from longitudinal studies of visually guided behavior in a macaque model of Alzheimer's disease-related pathology. This work builds on decades of basic research in healthy humans and NHPs and on studies showing that eye movements are sensitive indicators of early dysfunction in patients with Alzheimer's disease and mild cognitive impairment. Human studies have identified abnormalities in inhibitory control during delayed saccade tasks (Abel et al., 2002), altered visual search strategies (Moser et al., 1995; Rösler et al., 2000), and changes in novelty preference measured using visual paired-comparison tasks (Crutcher et al., 2009; Lagun et al., 2011; Zola et al., 2013; Bott et al., 2017). Related paradigms have long been used in healthy NHPs, including visual paired-comparison tasks that revealed recognition deficits following selective hippocampal damage (Zola et al., 2000). Building on this foundation, closely related saccade, visual foraging, and preferential-looking tasks were used to study disease progression in a longitudinal macaque model of Alzheimer's disease (Ruff et al., 2026; model established in Beckman et al., 2021; Beckman et al., 2024). This study revealed progressive disorganization of visually guided behavior despite preserved performance on simple tasks and intact encoding of many visual features. Parallel measurements of neuronal populations and disease biomarkers demonstrated that behavioral changes were accompanied by declining coordination within and between cortical areas. Thus, behavior served as a common framework linking pathology, neuronal population organization, and cognitive dysfunction across species, while providing candidate behavioral assays for detecting early disease-related changes.

Neuropsychological testing using standard batteries are important instruments to assess cognitive function in various patient populations. For example, tests such as the Wisconsin Card Sorting Test, Wechsler Adult Intelligence Scale, along with mental arithmetic and digit span tasks have a long tradition in the assessment of working memory and executive function in clinical neuropsychology. These domains are particularly affected in many psychiatric disorders and neurological conditions (Menon, 2011; Snyder et al., 2015; Sha et al., 2019; Friedman and Robbins, 2022). Constantinidis and colleagues used a three-step approach to link neuropsychological performance in humans to underlying neural signature markers. They first trained NHPs to perform laboratory tasks that assess memory such as delayed response and delayed match–nonmatch tasks (Tang et al., 2022; Mozumder et al., 2026). They then adopted these tasks for human subjects having undergone implantation of intracranial electrodes prior to epilepsy surgery,

who also are routinely tested in various neuropsychological test batteries (Singh et al., 2024). This combinatorial process as a final step allowed bridging the divide between neural activity recorded in these laboratory tasks and clinical, behavioral measures of cognitive function from clinical neuropsychology by determining what aspects of neural activity in the laboratory test best predicted behavior in the neuropsychological instruments (Jurva et al., 2025). They found that the amplitude of high-gamma (70–150 Hz) power in the LFP recorded from the prefrontal and parietal cortex during the delay period of working memory tasks was positively correlated with neuropsychological performance in human patients (Fig. 1*B*). This LFP measure in turn was present in sites where persistent spiking activity was generated by prefrontal neurons in NHPs. These results provide a case study of how neural activity in animal models can be revealing of cognitive functions and clinical conditions in humans.

Together, these approaches provide strong evidence that behavioral measures provide a framework for generating and testing hypotheses across levels of analysis, from neural circuits to cognition and disease. In this way, behavior serves as the connecting glue to cellular and circuit mechanisms in animal models to cognitive phenomena and clinical measures in humans, while also providing opportunities to develop new behavioral assays that are sensitive to early disease-related changes.

Neural Circuits/Anatomy and Neurophysiology

In addition to intersecting at the level of behavior, there are key anatomical and physiological similarities between humans and other primates. Here, we will concentrate on similarities between macaques and humans as the brain anatomy and physiology of this species of NHP has been extensively characterized and compared with humans. This work has revealed that human and macaque brains are both highly differentiated and expanded compared with nonprimate species (Carmichael and Price, 1994; Petrides and Pandya, 2002). For example, within the frontal lobes of both, there are many clearly identifiable areas that can be parcellated based on variation in cytoarchitecture. Further, the anterior frontal cortex in both humans and macaques is composed of homotypic cortex with densely packed "granule cells" in layer IV. This feature of frontal cortex anatomy is unique to the euarchonta grand order of mammals that includes all primates, as well as prosimians, flying lemurs, and tree shrews (Preuss and Wise, 2022), and is the defining feature of prefrontal cortex (Brodmann, 1909). Of the available animal models in this grand order, however, macaques have highly similar frontal cortex to humans, emphasizing their importance for understanding human PFC function in health and disease (Fig. 2*A*). Other cortical specializations that are unique to primates are also apparent in other parts of the brain including visual and motor cortex (Wise, 2023). These relate to primate-specific adaptations such as the existence of a fovea, whose representation is expanded in the visual cortex, and specialized binocular disparity columns and color blobs, for stereoscopic color vision. Similar primate motor specializations are present for advanced reaching and grasping capabilities (Kaas, 2012). Recent molecular and cellular analyses of the brains of primates have also emphasized the close correspondence between humans and macaques (Sousa et al., 2017; Ma et al., 2022; Totty et al., 2025). These primate-specific adaptations emphasize the importance of macaques for understanding human brain function in health and disease.

In addition to similarities in the cytoarchitecture of the brain between humans and macaques, the pattern of connections between areas is also highly conserved across these two species of primate. Based on combining tract-tracing experiments with diffusion tractography estimates of anatomical connectivity in humans and macaques, we know that many of the major fiber pathways are comparably organized in these two species (Jbabdi et al., 2013; Folloni et al., 2026; Fig. 2*B*). Functional neuroimaging assessments of connectivity in macaques and humans also point to a shared pattern of brain organization with all the main intrinsic human resting-state functional connectivity networks also present in macaques (Vincent et al., 2007; Fig. 2*C*). Using functional resting-state connectivity as a proxy for anatomical connectivity (Honey et al., 2009), Rushworth and

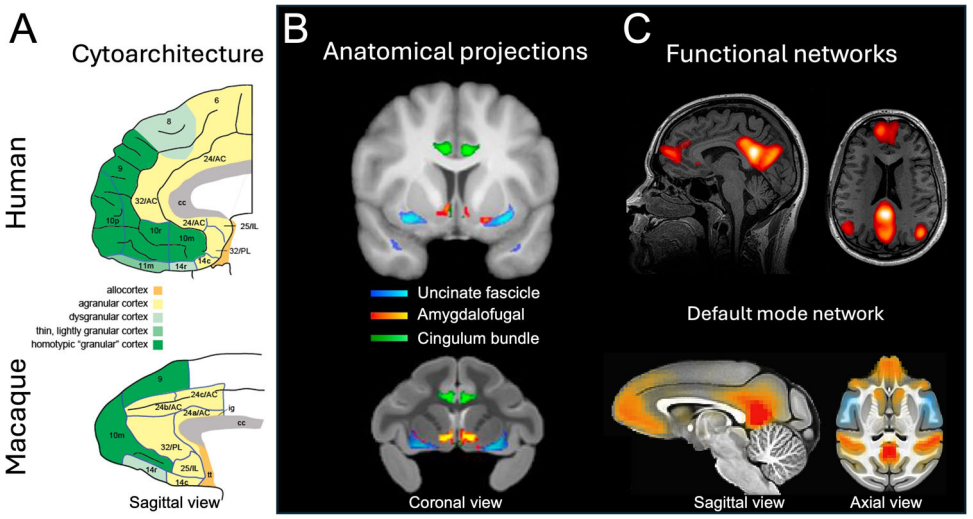


Figure 2. Comparative cytoarchitecture, anatomical projections, and functional networks of human and macaque frontal lobe. ***A***, Schematic of cytoarchitecture of the human (top) and macaque (bottom) frontal lobes from a sagittal view. Different cortical areas are outlined and the type of cortex present in each area is denoted by colored shading. Area labels use the convention detailed in Carmichael and Price (1994). ***B***, Coronal section through the frontal lobe of human (top) and macaque (bottom) showing the location of three major white matter pathways that carry projections to and from the frontal cortex; uncinate fascicle (blue), amygdalofugal pathway (red), and cingulum bundle (green). ***C***, Sagittal (left) and axial views of the default mode network in humans (top) and macaques (bottom).

colleagues have compared the connectivity "fingerprints" of the macaque and human brain (Mars et al., 2016). These analyses emphasize that there is a high degree of correspondence in the functional and likely anatomical connectivity of the cortex across these species (Mars et al., 2013; Neubert et al., 2015). Importantly, these analyses also found that there are key differences between the connectivity of humans and macaques. For instance, parts of the ventrolateral PFC in humans have a higher degree of connectivity with posterior auditory association areas compared with the homologous areas in macaques (Neubert et al., 2014). This is likely related to parts of ventrolateral PFC being specialized for language production in humans as this is where Broca's area is located (Broca, 1861). Parts of the human anterior prefrontal cortex also do not have a clear homolog in macaques (Neubert et al., 2015). Taken together, these analyses show that the macaque brain shares many key similarities with those of humans, but it is important to emphasize that it is not simply a miniature version of a human brain.

As highlighted earlier, macaques exhibit advanced cognitive abilities and can perform tasks to the same level as humans. With this "behavioral glue" and the knowledge that the macaque and human brains are similarly organized, studying the patterns of neural activity engaged in macaques provides an important route for determining the basis of human cognition. Here parallel functional neuroimaging investigators in humans and macaques have been a critical translational bridge. These studies have confirmed that homologous brain areas are engaged in these two species of primates across perceptual, sensory, and cognitive domains. For instance, studies using comparable task designs have found that when macaques and humans view images (Orban et al., 2004; Kravitz et al., 2011), faces (Tsao et al., 2008; Pinsk et al., 2009), flexibly learn and reverse their choices between different stimuli (O'Doherty et al., 2001; Ghahremani et al., 2010; Chau et al., 2015; Fujimoto et al., 2026a), or learn abstract sequences (Desrochers et al., 2015; Yusif Rodriguez et al., 2024) shared brain circuits are engaged. Due to limitations in temporal and spatial resolution, functional neuroimaging cannot, however, reveal the specific patterns of single-neuron or LFP activity engaged during cognition. This level of resolution can only be obtained with direct brain recordings, and thus neurophysiology studies in macaques are foundational for determining the neural basis of human cognition

One of the clearest examples of where research in macaques has revealed the neurophysiological basis for human cognition is in the study of visuospatial attention or selective visual processing (Fiebelkorn and Kastner 2019). For many years it was thought that attention was a single spotlight that enhanced processing of information of specific features such as a spatial location (Moran and Desimone, 1985). Increasing evidence, however, indicates that primate visuospatial attention is characterized by alternating periods of heightened and diminished sensitivity at a frequency in the theta range (Fig. 1*A*; Lakatos et al., 2008; Busch and VanRullen, 2010; Fiebelkorn et al., 2013). The mechanisms supporting these rhythmic fluctuations in primate attention were unknown. Using comparative intracranial recordings from the frontoparietal cortex in macaques and humans performing an identical attention task, Kastner, Knight, and colleagues found that neural oscillations in the theta range (4–8 Hz) in these areas were directly related to the rhythmic changes in attention such that the phase of the neural theta rhythm was predictive of behavioral target detectability (Fiebelkorn et al., 2018; Helfrich et al., 2018). Single-unit and LFP recordings in macaques provided a deeper mechanistic understanding by revealing that theta rhythms shape our perception of the world on a rapid timescale by organizing neural activity into two alternating attentional states. One state promotes sampling visual information at the presently attended location and emphasizes neural mechanisms related to sensory processing; the other state promotes shifting attention (or gaze) to another location in the visual field and emphasizes neural mechanisms related to motor processing (Fiebelkorn et al., 2018; Fiebelkorn and Kastner 2019). Sampling and shifting cannot occur at the same time and require temporal coordination of the associated neural populations and the wider circuits in which they are embedded. Thus, studies involving macaques helped to establish the neural basis of rhythmic fluctuations in human attention.

While a powerful tool to uncover the neural mechanisms of cognition, direct recordings of brain activity in humans are confined to parts of the brain that are being clinically investigated for pathological epileptiform activity. This underlines a key limitation of direct neural recordings from the human brain: they are being acquired from a brain that is exhibiting pathological activity. It is also important to note that the primary point of comparison between macaque and human neural recordings involves detailed analyses of LFP activity. Further developments of single-neuron recordings in the human brain are evolving (Coughlin et al., 2023) and these tied with high-density and longitudinal recordings in macaques have the potential to further reveal the signatures of human cognition.

One key opportunity afforded by macaques is the ability to record in parts of the healthy brain that are implicated in different brain disorders but are challenging or almost never targeted in humans undergoing intracranial monitoring for epilepsy. For instance, direct recordings of neural activity from the serotonin-producing neurons in the dorsal raphe during behavior are not possible due to its location in the midbrain. However, there is a critical need to understand the physiology of this area as it has been directly implicated in a host of psychiatric disorders and frontline treatments for depression target the serotonin system (Robbins and Arnsten, 2009). Single-neuron recordings in macaques performing affective tasks have begun to establish the patterns of activity elicited by these cells (Feng et al., 2024), but further studies are required to fully characterize their activity. Another example is the basal forebrain where direct recordings, inactivation, and electrical modulation of neural activity have established a key role of these cholinergic neurons in cognitive function (Qi et al., 2021; Pennington et al., 2024). Consequently, studies in macaques remain essential for establishing the patterns of activity involved in human cognition.

Neuromodulation

A particularly clear example of the importance of NHP research comes from the development of Deep Brain Stimulation (DBS) for Parkinson's disease. During the 1980s, Benabid and colleagues had identified high-frequency DBS as a therapy for tremor (Benabid et al., 1987), but a treatment for Parkinson's disease remained elusive. Working with 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP)-treated NHPs, Delong and colleagues identified abnormal activity within the subthalamic nucleus (STN), and subsequent lesion of STN led to a normalization of motor behavior (Bergman et al., 1990; Wichmann and Delong, 2006). These experiments in macaques thus provided a mechanistic framework that subsequently guided the development of STN DBS for Parkinson's (Volkmann and Sturm, 1998; Obeso et al., 2001), a therapy used in many thousands of individuals worldwide.

A case study of the iterative and bidirectional relationship between human and animal research has been the use of DBS for the treatment of treatment-resistant depression (Mayberg et al., 2005; Alagapan et al., 2023). Similar to Parkinson's, neuroimaging experiments identified hyperactivity within the subcallosal anterior cingulate cortex (ACC) as contributing to typical sadness as well as major depression (Mayberg et al., 1999). In the initial clinical trial, DBS targeted to this location reduced pathological subcallosal ACC activity in people who responded to the therapy (Mayberg et al., 2005). Subsequent development of this approach now targets the white matter adjacent to the subcallosal ACC and is effective in as many as 70% of patients treated (Riva-Posse et al., 2018). Despite this the mechanisms underlying long-term therapeutic effects of subcallosal ACC DBS remain poorly understood. Seeking to clarify the circuits and mechanisms involved, Fujimoto and colleagues recently modeled the effect of subcallosal ACC DBS in macaques. Mirroring findings in humans (Elias et al., 2022), they confirmed that subcallosal ACC DBS altered patterns of fMRI; however, they also found that DBS caused a selective remodeling of white matter distant to the site of stimulation (Fujimoto et al., 2026b). This finding was unexpected and now motivates a more thorough examination of the role of white matter plasticity in the recovery from treatment-resistant depression and potentially other psychiatric disorders.

Experiments targeting the cholinergic basal forebrain in NHPs provide another illustrative example. In NHPs, stimulation of cholinergic forebrain structures enabled detailed investigation of stimulation-induced changes in neural activity and cognitive function (Qi et al., 2021; Pennington et al., 2025). Because it was possible to directly measure neural responses while varying stimulation parameters, these studies identified conditions that maximized engagement of targeted circuits while minimizing undesirable effects. In turn, this work provided crucial preclinical support for human trials investigating cholinergic DBS as a treatment for dementia currently under way.

NHP models can provide functional models for brain regions that are difficult or rarely studied in humans but which could be therapeutically targeted with DBS. Higher-order thalamic nuclei such as the pulvinar and mediodorsal nuclei are still "terra incognita" in the human brain. Studies in NHPs have revealed that higher-order thalamic nuclei, and specifically the pulvinar, contribute to attention, working memory, cognitive control, and network coordination (Saalmann et al., 2012; Zhou et al., 2016). These discoveries have important implications for neuromodulation. In epilepsy, stimulation of higher-order thalamic nuclei may function as a circuit-level intervention capable of disrupting pathological network synchronization and preventing seizure propagation (Wu et al., 2023; Damiani et al., 2025). In schizophrenia, modulation of thalamic circuits may potentially restore dysfunctional interactions between thalamic and cortical networks that contribute to cognitive and psychiatric symptoms (Meyer et al., 2025).

The importance of NHP research extends beyond DBS. Noninvasive stimulation techniques, such as TMS and focused ultrasound (FUS), influence distributed brain networks, but their circuit-level effects remain incompletely characterized. In this space, studies in NHPs have been essential for isolating the peripheral and central effects of TMS (Perera et al., 2023), the potential for TMS protocols to help the recovery from stroke (Kadono et al., 2021), and the brain-wide effects and safety of FUS (Folloni et al., 2019; Verhagen et al., 2019). Animal models can also provide "ground truth" validation for techniques such as source localization in magnetoencephalography (MEG; Alberto et al., 2021). Opto- and chemogenetic methods in primates provide unprecedented opportunities to dissect the cellular and circuit mechanisms underlying neuromodulation. Although the clinical application of opto- or chemogenetics remains in its infancy, experiments using these methods can establish causal links between specific cell populations, network dynamics, and behavior (Tamura et al., 2017; Chen et al., 2023; Oyama et al., 2024). Such knowledge may inform the design of future protocols that more selectively engage specific circuits in the human brain to treat psychiatric or neurological disorders.

Despite their importance, nonhuman primate models are not a universal solution. Unlike Parkinson's disease, many psychiatric disorders lack faithful primate models, limiting the ability to reproduce human disease mechanisms. Nevertheless, NHP research offers unique advantages even when disease models are imperfect. Monkeys can often serve as their own controls in longitudinal studies, enabling highly sensitive assessments of neuromodulation effects. Furthermore, aging itself can be studied in primates, which age naturally at much faster rate than humans, providing opportunities to investigate age-related changes in neural circuits relevant to dementia and cognitive decline.

Conclusions

The examples discussed throughout this review illustrate a common theme: progress in understanding the neural basis of complex cognitive behavior is accelerated when studies in humans and NHPs inform one another. Human studies reveal cognition and disease in their natural context and identify the questions that matter most, but they are constrained by the practical realities of studying individuals with rich and heterogeneous lives. Measurements often consist of relatively brief and infrequent encounters, and many variables cannot be controlled or manipulated. In contrast, studies in NHPs permit repeated measurements in the same individuals over months or years, careful control of sensory and behavioral conditions, and causal manipulations of specific circuits and/or cell types that are impossible in people. These complementary strengths create opportunities to isolate mechanisms and establish principles that would be difficult to uncover using either approach alone. Together, studies in humans and NHPs create a complementary and iterative cycle where observations in one domain refine hypotheses in the other, transforming cross-species comparisons from descriptive parallels into mechanistic tests of shared neural principles.

Recent technological advances are making this dialogue increasingly powerful. Large-scale recordings in NHPs (Jun et al., 2017; Trautmann et al., 2025), chronic and ambulatory recordings in neurosurgical patients (Topalovic et al., 2023), high-density probes in humans (Paulk et al., 2022; Khanna et al., 2024; Leonard et al., 2024), circuit-neuroscience informed TMS (Cole et al., 2022), focused ultrasound (Folloni et al., 2019; Verhagen et al., 2019), and increasingly sophisticated analytical frameworks for studying the relationship between neural populations and behavior (Saxena and Cunningham, 2019; Rust and Cohen, 2022; Urai et al., 2022) are creating unprecedented opportunities to compare neural computations across species. At the same time, quantitative behavioral paradigms provide a common language for relating neural activity to cognition and behavior. Together, these advances allow common behavioral, physiological, and analytical frameworks to be applied across species and levels of analysis, bringing studies in humans and NHPs into increasingly direct dialogue. As a

result, systems neuroscience approaches that once seemed confined to laboratory studies are increasingly being brought to bear on fundamental questions about human cognition in health and disease.

Throughout this review, we have highlighted complementary frameworks for integrating observations across species and levels of analysis. Among these, behavior serves as a particularly powerful bridge because it provides a common experimental framework even when identical neural measurements cannot be obtained in humans and NHPs. Comparative anatomy, neurophysiology, and neuromodulation provide additional links, allowing mechanisms identified in one species to inform interpretation and causal testing in another. Human and NHP studies provide different but complementary windows into the same problems, and their greatest value lies not in isolation but in combination. By integrating these approaches, we can uncover principles that would remain hidden in either system alone. As new technologies continue to bring studies in humans and NHPs closer together, this dialogue will become increasingly important. Recognizing this, the BRAIN Initiative emphasized the importance of pursuing parallel studies in human and nonhuman models (BRAIN Working Group, 2014; Jorgenson et al., 2015; Ngai, 2026). To that end, the BRAIN Initiative Cell Atlas Network (BICAN) and Armamentarium projects have developed next-generation cellular and molecular tools using human, NHP, and mouse data to define cell-type taxonomies. These tools have already been deployed in mouse and NHP models, offering significant clinical promise for cell-type-specific molecular, optogenetic, and chemogenetic interventions in humans (Ben-Simon et al., 2025). Future progress will depend on exploiting the unique strengths of each approach while maintaining close connections between species, measurement standards, and analytical frameworks.

Ultimately, the central challenge of systems neuroscience is to understand how neural circuits give rise to cognitively complex behavior. Meeting that challenge will require tighter integration between studies in humans and NHPs. By treating studies in humans and NHPs as complementary components of a unified experimental framework rather than parallel lines of investigation, we will be better positioned to discover fundamental principles of brain function and translate those discoveries into improved diagnosis and treatment of neurological and psychiatric disease.

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