REVIEW

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Molecular and circuit mechanisms underlying interhemispheric communication in the mammalian brain

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Interhemispheric communication is a fundamental feature of the mammalian brain, supporting the bilateral integration of sensory, motor, cognitive, and emotional processes. While the corpus callosum has long been recognized as the principal commissural pathway, recent advances have illuminated a far more complex molecular and circuit-level architecture. This review synthesizes evidence from neuroanatomy, electrophysiology, molecular neuroscience, and neuroimaging to outline how interhemispheric signaling is organized and dynamically regulated. Fast excitatory and inhibitory neurotransmission provides the scaffold for callosal transfer, while neuromodulatory systems, including dopaminergic, cholinergic, serotonergic, and noradrenergic pathways, introduce a chemical layer of regulation that tunes excitability, synchrony, and hemispheric dominance. Developmental processes involving axon guidance molecules and neurotrophins shape the establishment of commissural networks, whereas activity-dependent plasticity refines functional architecture of these networks across the lifespan. Importantly, interhemispheric interactions are not static but fluctuate dynamically according to behavioral demands, as demonstrated by recent models of dynamic laterality. Disruption of these lateralized processes is implicated in a broad spectrum of conditions, including stroke, dyslexia, autism spectrum disorder, schizophrenia, and mood disorders. By bridging cellular, molecular, and systems-level insights, this review highlights interhemispheric communication as a key organizing principle of brain function and a promising target for therapeutic interventions aimed at restoring interhemispheric balance.

Key words: interhemispheric interactions, lateralization, cortical plasticity, brain rhythms, barrel field, stroke

INTRODUCTION

Interhemispheric interactions are fundamental to the coordinated functioning of the mammalian brain. Bilateral integration of sensory inputs, motor planning, cognitive processing, and emotional regulation relies on dynamic communication between the two cerebral hemispheres. While the anatomical substrates of this communication, primarily the corpus callosum, have been extensively characterized, the molecular and circuit-level mechanisms that enable interhemispheric synchronization remain unclear (Kuo & Nitsche, 2021). Already at this conceptual level, it is important to visualize how callosal fibers are spatially organized in the human and rodent brain, since their laminar and topographical arrangement constrains both excitatory transfer and feedforward inhibition. Fig. 2 illustrates the organizational principles of callosal topology in the human and Fig. 1 in rat brain and provides a comparative framework for subsequent sections of this review.

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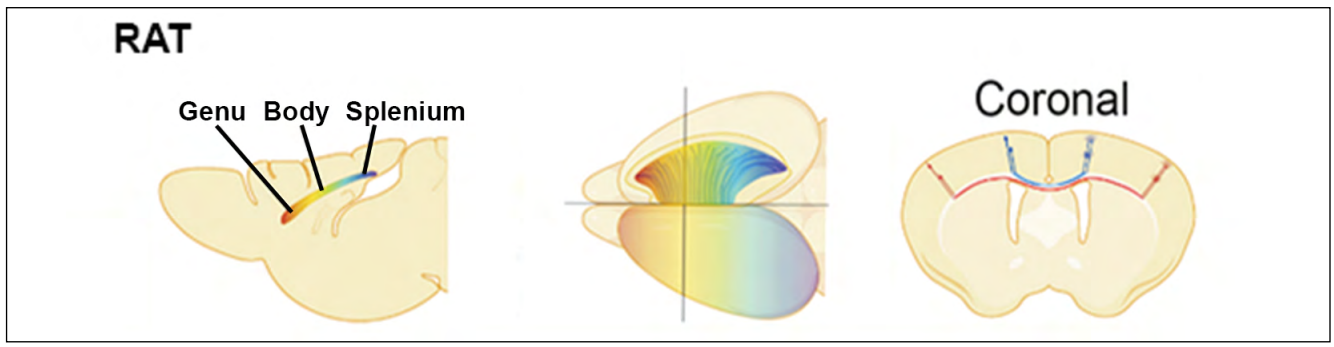


Fig. 1. Midsagittal, dorsal, and coronal views of the rat corpus callosum (CC). Callosal axons originating from each cortical hemisphere cross the midline and predominantly connect with homotopic cortical regions in a topographically organized manner. Projections from anterior and posterior cortical areas form the anterior and posterior CC, respectively (illustrated in rainbow colors), while projections from medial and lateral cortical regions give rise to the dorsal and ventral portions of the CC, respectively. *Adapted from Ku & Torii, 2020, with permission, under CC_BY.*

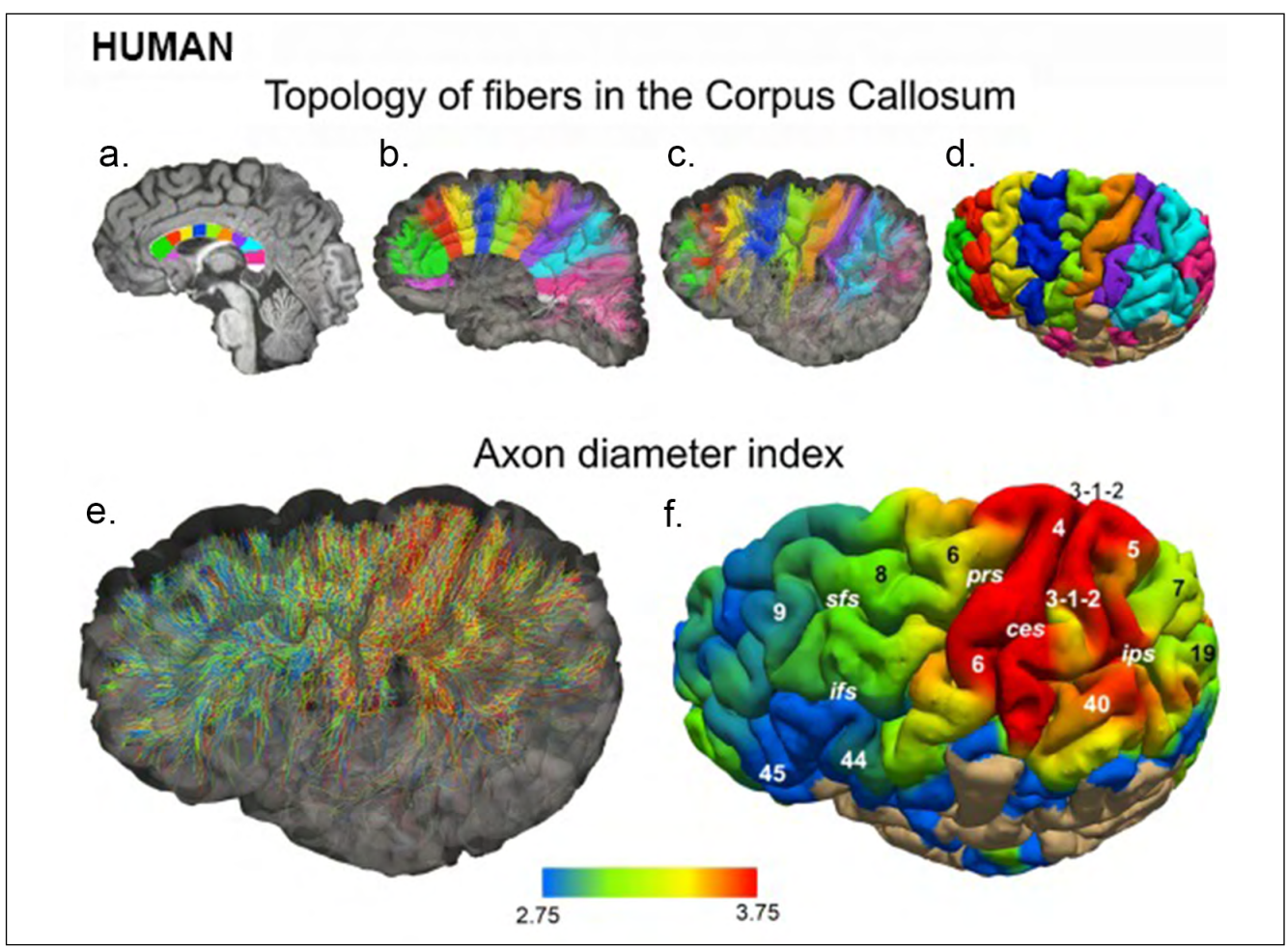


Fig. 2. a-d, Topology of fibers in the Corpus Callosum (CC) reconstructed by diffusion-weighted magnetic resonance imaging (DW-MRI). a. Subdivision of the mid-sagittal section of the CC in 11 sectors [corresponding to the regions of interest (ROIs)]. b, c. Streamlines colored according to ROIs projected on pial surface from medial (b) and lateral (c) views of the hemisphere. e, f. Axon diameter indexes of streamlines passing through the CC (e), colored according to their axon diameter index, and (f) projected onto the cortical surface. Colors correspond to the axon diameter index averaged across streamlines. Notice larger diameter indexes in the precentral and postcentral gyri, corresponding to motor (4) and somatosensory areas (3-1-2), the smaller indexes elsewhere as expected from histological work (Aboitiz et al., 1992) although skewed to larger estimates. Abbreviations: *ces*–central sulcus, *ifs*–inferior frontal sulcus, *ips*–intraparietal sulcus, *prs*–precentral sulcus, *sfs*–superior frontal sulcus. Numbers correspond to Brodmann areas. *Original figure reproduced from Innocenti et al., 2025, under CC BY-NC-ND 4.0 (no changes made).*

At the cellular and molecular scales, interhemispheric communication relies on neurotransmitter signaling, ion channel localization, synaptic plasticity, axonal pathfinding, and myelination processes. The excitatory–inhibitory balance between the hemispheres is primarily governed by glutamatergic and GABAergic systems, whose function is shaped by receptor subtype composition and activity-dependent synaptic remodeling (Flower et al., 2022) set by complex networks of neuromodulators, such as adrenaline, dopamine, serotonin, and acetylcholine.

Recent advances in transcriptomics, proteomics, and functional neuroimaging have revealed fine-tuned regulatory networks of neuromodulators that are involved in the functional lateralization of the two hemispheres, thereby highlighting the importance of which makes interhemispheric signaling more profound. These findings are particularly relevant to neurological and psychiatric conditions, such as stroke, dyslexia, autism spectrum disorder (ASD), and schizophrenia, in which interhemispheric balance disruption contributes to clinical symptoms (Fitzgerald, 2020).

Together, molecular mechanisms that integrate neuronal activity generate rhythmic circuit oscillations emerging from the electrophysiological states of individual neurons. The relationship between receptor distributions and functional laterality is exemplified in Fig. 3, which maps the density gradients of major neurotransmitter receptors across the human cingulate cortex. Because these gradients shape local excitability and cross-hemispheric gating, they provide molecular context for the oscillatory asymmetries illustrated in Fig. 8. These oscillatory patterns reflect interhemispheric competition, which plays a critical role in the selective activation and dominance of one hemisphere over its contralateral counterpart, thereby facilitating hemisphere-specific cognitive processing and behavioral problem-solving in the field (e.g., face recognition *vs.* reading; Thomas & Arslan, 2025; Liu et al., 2024; Haxby et al., 2000).

In addition, neurotrophic factors, such as brain-derived neurotrophic factor (BDNF; Cohen-Cory & Fraser, 1995), and axon guidance molecules, including netrins, semaphorins, and ephrins, contribute to the establishment and refinement of interhemispheric circuits (Szebenyi et al., 2001). This gradient of diverse factors is related to the splenial topography shown in Fig. 5e–g, where heterotopic *vs.* homotopic connectivity clusters define the emerging architecture of adult callosal pathways (Putnam et al., 2010).

The hippocampus, amygdala, and neocortex form an interconnected network that underpins core cognitive functions. As these regions differ markedly in their interhemispheric connectivity patterns, functional differences are related to the commissural organization illustrated in Fig. 2 and the limbic asymmetry mechanisms depicted in Fig. 4. The hippocampus is indispensable for encoding, consolidation, and retrieval of declarative and spatial memories, as well as for contextual representation and flexible navigation of cognitive maps (Eichenbaum, 2017; Hawkins et al., 2019). The amygdala is specialized in detecting biologically salient stimuli and assigning them affective value, thereby modulating mnemonic processes through its reciprocal connections with the hippocampus and prefrontal cortex (Phelps & LeDoux, 2005). The neocortex serves as the principal site for the long-term storage and integration of information, receiving processed outputs from the hippocampus for the systems-level consolidation, and contributing to the top-down modulation of both hippocampal and amygdalar activity during perception, decision-making, and emotional regulation (Frankland & Bontempi, 2005). Through dynamic interactions, the neocortex, the hippocampus, and the amygdala integrate contextual, emotional, and semantic information, enabling adaptive learning, flexible behavior, and goal-directed cognition. Nevertheless, interhemispheric interactions are becoming increasingly sophisticated as the functional specialization of one hemisphere becomes more pronounced.

Subcortical regions, including the basal ganglia, nucleus accumbens, and thalamus, contribute to reward processing, motivational salience, and gating of sensory information to cortical and hippocampal circuits (Haber & Knutson, 2010; Sherman, 2016). These structures are intricately connected to the amygdala and hippocampus, facilitating the integration of motivational and contextual cues into adaptive behavioral strategies.

Critically, many of these regions, most prominently the neocortex, hippocampus, and amygdala, possess robust interhemispheric connectivity, primarily *via* the corpus callosum and anterior commissure. Such bilateral integration supports the coordination of mnemonic, emotional, and executive functions across hemispheres, enabling coherent perception, unified memory representation, and adaptive decision-making in dynamic environments (Gazzaniga, 2000).

Beyond these structural substrates, neuromodulatory systems provide a crucial regulatory layer for interhemispheric communication. Dopaminergic, cholinergic, serotonergic, and noradrenergic pathways tune neuronal excitability, shape oscillatory synchronization, and bias hemispheric dominance according to task demands (Robbins, 2000; Sara, 2009; Tomer et

al., 2013). By dynamically tuning the balance between excitation and inhibition as a function of emotionally determined salience, neuromodulatory systems provide a flexible mechanism for interhemispheric coordination, thereby bridging molecular asymmetries with large-scale cognitive and affective specialization.

This review synthesizes the current knowledge on the cellular and molecular foundations of interhemispheric communication, emphasizing both developmental and experience-dependent plasticity, and highlighting the implications for clinical neuroscience.

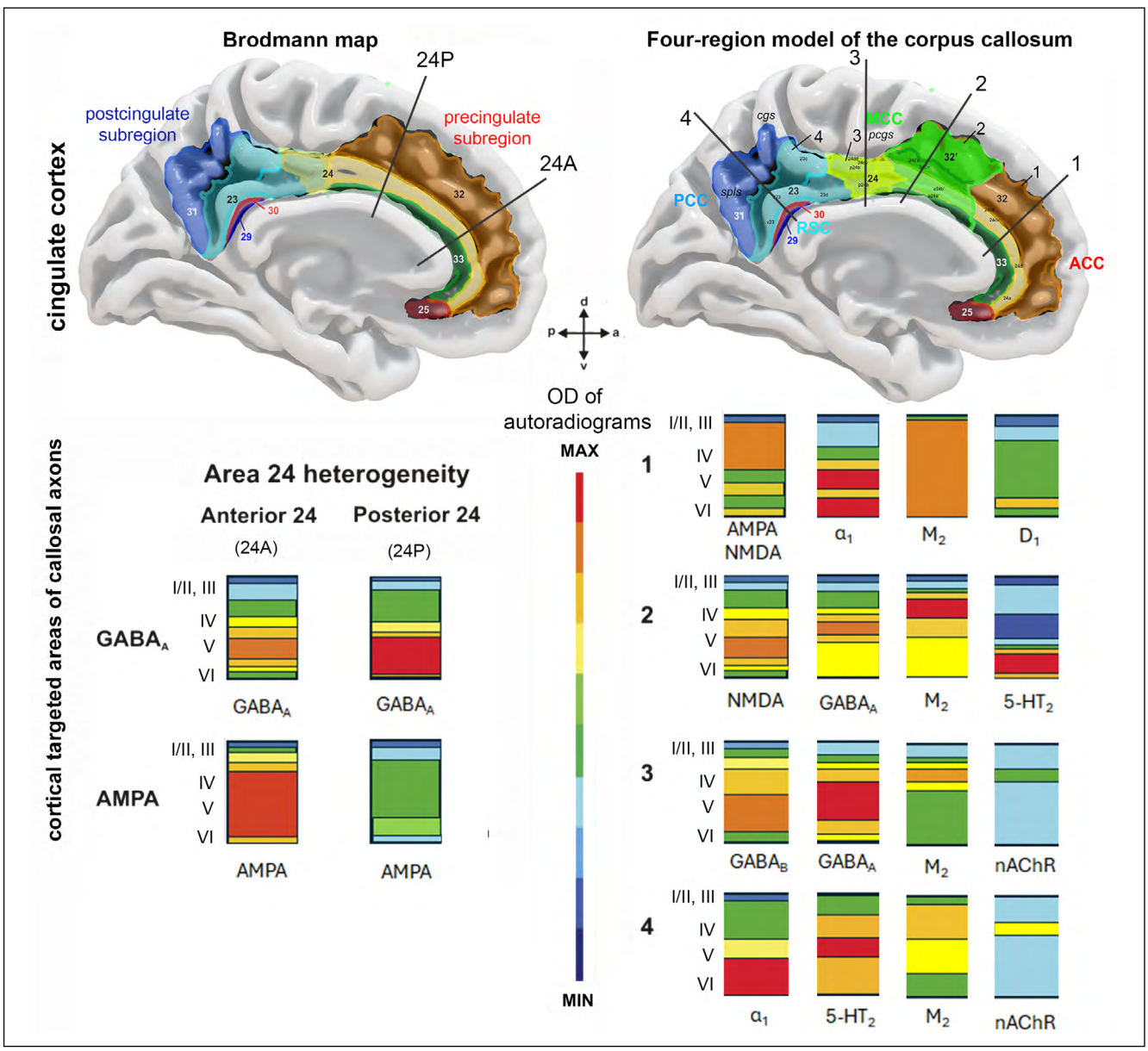


Fig. 3. Distribution of neurotransmitter receptors in the layers of human cingulate cortex. Schematic representation of cingulate cortex subregions defined according to Brodmann (1909) and the four-region model of Vogt et al. (2004). Bottom panels show modeled optical density (OD) distributions reconstructed from autoradiographic measurements, representing successive rostrocaudal levels of the human cingulate gyrus and revealing receptor-specific laminar and subregional organization as variations in OD across cortical layers and cytoarchitectonic subdivisions. Identified regions include the precingulate cortex (areas 33, 25, 24, and 32), the postcingulate cortex (areas 23 and 31), and the retrosplenial cortex (areas 29 and 30; see Fig. 5f). For clarity, the callosal (cas), cingulate (cgs), paracingulate (pcgs), and splenial (spls) sulci were digitally opened. Crosslines 1–4 indicate the rostrocaudal levels corresponding to the regions from which the autoradiographic sections used for laminar protein distribution visualization in the graphs were obtained. *Created by the authors based on experimental data from Palomero-Gallagher et al. (2009).*

Interhemispheric connections in the bilateral symmetry of the brain

The bilateral symmetry of the vertebrate brain relies on commissural systems that integrate activity across the hemispheres while preserving functional specialization. The corpus callosum is the largest of these pathways, coordinating homotopic and heterotopic cortical regions through predominantly excitatory projections, complemented by a smaller population of long-range GABAergic neurons that contribute to direct interhemispheric inhibition (Buhl &

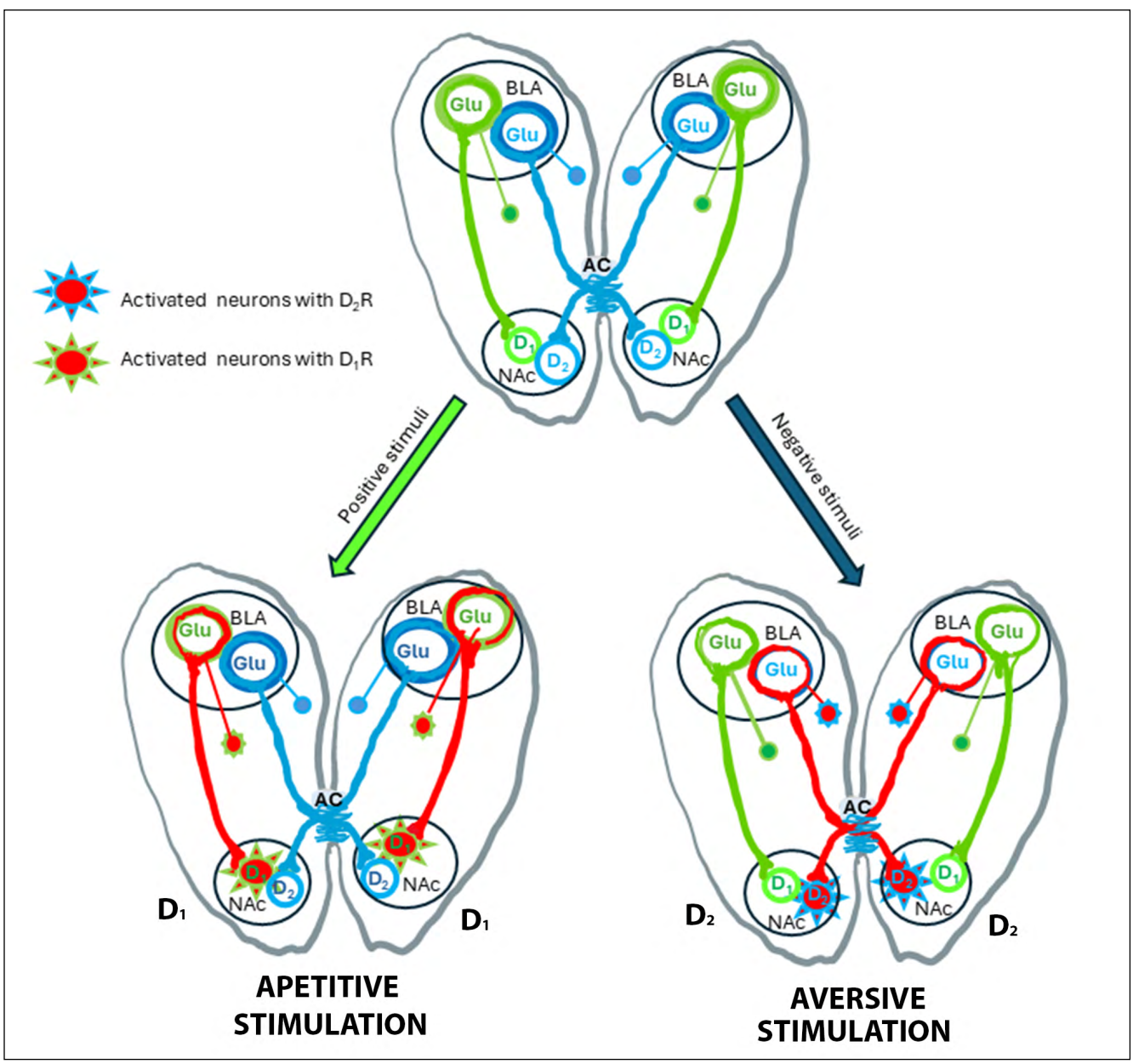


Fig. 4. Contralateral BLA–NAc circuit in encoding valence. The basolateral amygdala (BLA) sends glutamatergic projections to the ipsilateral nucleus accumbens (NAc) *via* the stria terminalis and ventral amygdalofugal pathways, and to the contralateral NAc *via* the anterior commissure (AC). Positive stimuli preferentially activate ipsilateral BLA–NAc projections, recruit D1-type medium spiny neurons (D1-MSNs; GABAergic neurons activated mainly *via* D1 dopamine receptors), enhance dopamine release in the NAc, and drive reward- and approach-related behavior. In contrast, negative stimuli predominantly activate contralateral BLA–NAc projections, engage D2-type medium spiny neurons (D2-MSNs), suppress dopamine release, and elicit aversive or avoidance responses. Glu+ –glutamatergic neurons; D1–D1-MSNs; D2–D2-MSNs. *Conceptual scheme created by the authors based on results of Tian et al. (2024), with permission. d. Reproduced from Bruzzone et al. (2022), with permission, Scientific Reports, under CC_BY.*

Singer, 1989; Rock et al., 2018). Other commissures, such as the anterior and hippocampal commissures, provide additional routes for the bilateral coordination of emotional, sensory, and mnemonic processes in mammals (Manzoni et al., 1989).

Development and disruption of interhemispheric connections

Callosal projections begin to form early in embryonic development, when axons emerging from pyramidal neurons extend towards the midline to establish both homotopic and heterotopic cortical connections. Differences in axonal diameter and length introduce conduction delays, which are essential for achieving precise temporal coordination between the hemispheres (Fig. 6; Caleo et al., 2013).

The topography of these projections is shaped by the interplay between molecular guidance cues and activity-dependent mechanisms. Specialized midline structures, such as the glial wedge and subcallosal sling, provide instructive signals that direct callosal axons towards their appropriate cortical targets, whereas neighboring axons contribute to pathfinding through contact-mediated interactions (Tagawa et al., 2008; Caleo et al., 2013). The refinement of these connections during critical developmental windows ensures the establishment of a balanced and efficient interhemispheric network.

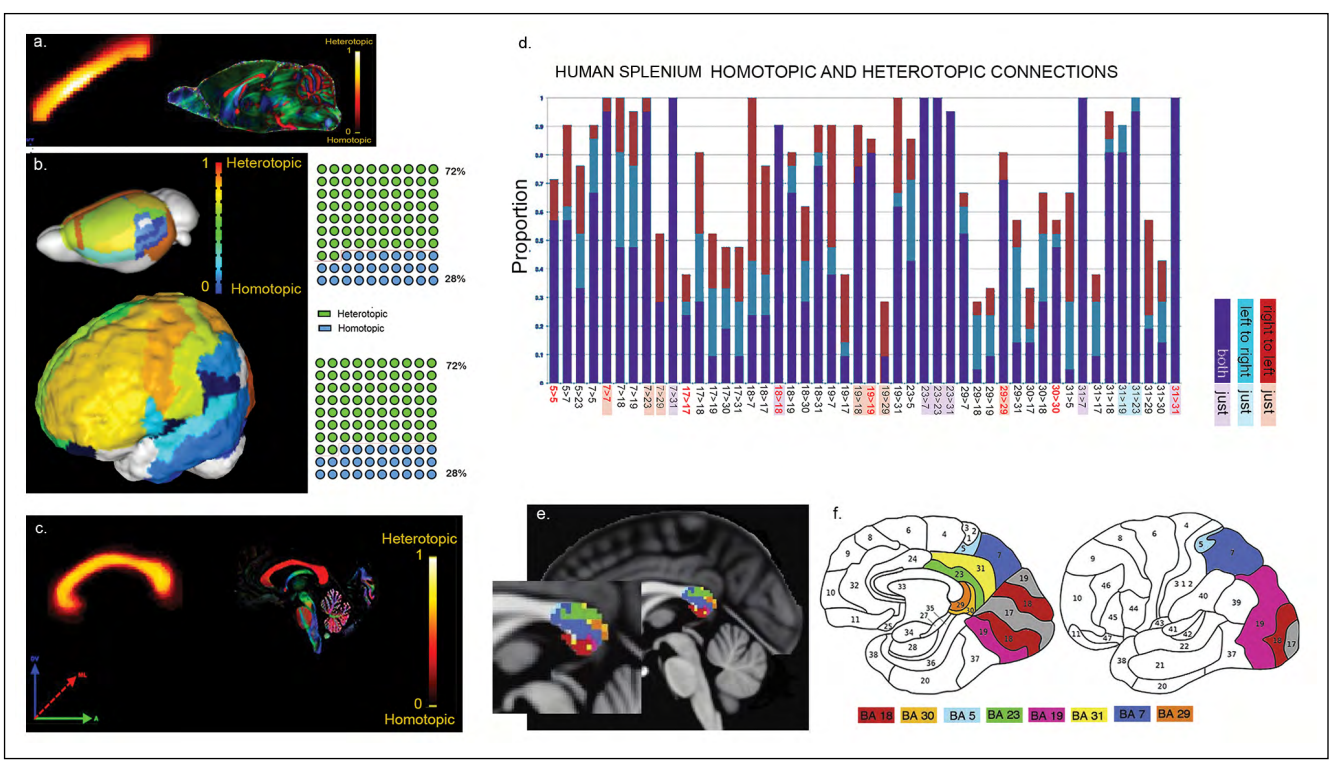


Fig. 5. Homotopic and heterotopic callosal connections. a. rat and c. human callosal heterotopicity maps. Voxel-based maps of the corpus callosum (CC) heterotopicity and the corresponding color-coded fiber orientation distribution (FOD) map (population-average subject) shows a homogeneous core of high heterotopicity surrounded by homotopic connections along the CC periphery. Diffusion axes: red=mediolateral (ML), green=anteroposterior (AP), blue=dorsoventral (DV). b. Heterotopicity maps of human and rat cortex. Schematics illustrate diffusion-weighted imaging (DWI) callosal tractography-based heterotopicity population averages. The color bar represents the heterotopicity index, with cool colors indicating homotopic areas and warm colors indicating heterotopic areas. Quantification shows that in rats ~78% of CC axons are heterotopic, while ~22% homotopic, in human ~72% of CC axons are heterotopic, while ~28% homotopic. *Adapted from Szczupak et al., (2023).* d. Homotopic and heterotopic splenial connectivity. For each cortical region connected through the splenium, homotopic versus heterotopic projections were quantified across participants. Cortical regions of origin/termination are shown on the x-axis (bold red=homotopic connections); the proportion of participants is shown on the y-axis. Tractography directionality is indicated: red=right-to-left, blue=left-to-right, violet=bidirectional. Unidirectional trajectories are marked in light red/blue on the y-axis, while fully bilateral trajectories are shown in light violet. e. Topographical organization of splenial projections. In 11 participants, for each voxel in the splenium, the cortical region of termination (represented by Brodmann areas) was identified, and voxels were color-coded according to their most likely cortical targets. The corresponding cortical regions are shown in the graph on the right. f. Schematic brain with Brodmann regions color-coded according to their splenial connectivity (panel A) and to the cingulate cortex subdivisions presented earlier in Fig. 3. *Composite figure: a-c, adapted from Szczupak et al. (2023), with permission, under CC_BY; e, f, adapted from Putnam et al. (2010), with permission.*

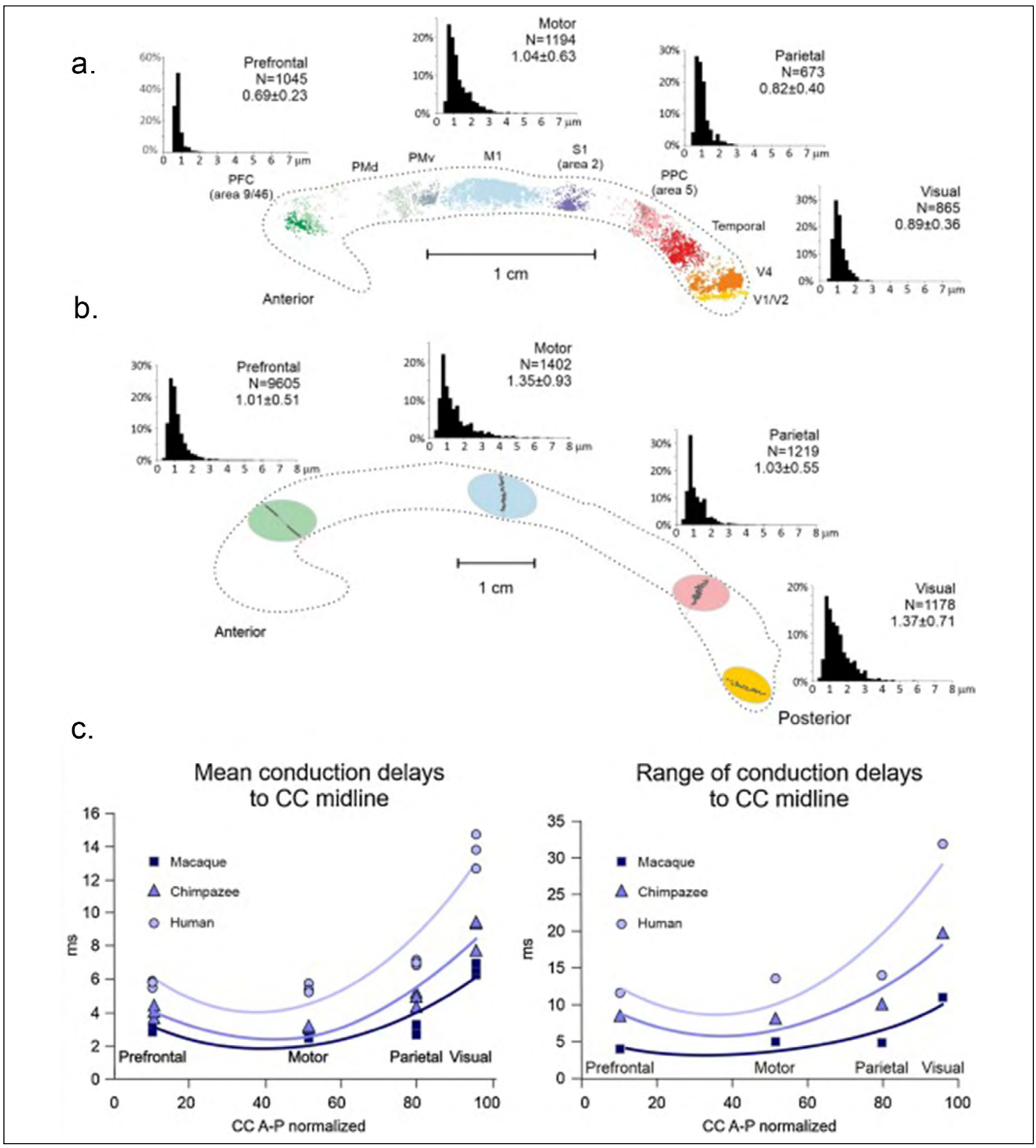


Fig. 6. Topographic organization of the corpus callosum (CC) of the macaque monkey. a. After injections of anterograde tracer (BDA) in different cortical areas, obtained by superposition of the outlines of the clusters of axon labeling from six different animals. Color gradients indicate axon labeling from prefrontal (9, 46), premotor (dorsal, PMd F2/F7; ventral, PMv, F4), motor (M1), somatosensory (S1, area 2), posterior parietal (area 5, PEc, PEip), temporal, extrastriate (V4), primary visual (V1/V2) cortex. The histograms indicate the distribution of axon diameters (n. of counts, mean ± SD) in selected prefrontal, motor, parietal, and visual sectors of the CC. Redrawn from Caminiti et al., 2009. b. Distribution of axon diameters sampled from discrete dorsoventrally oriented probes in different anteroposterior sectors of the CC, in humans, where fibers from prefrontal, motor, posterior parietal, and visual cortex cross the midline. Convention and symbols as in A. c. Mean conduction delays (left panel) and range of conduction delays (right panel) to the CC midline in monkeys, chimpanzees, and humans plotted against normalized antero-posterior CC dimension and fitted with a polynomial function. Adapted from Caminiti et al. (2009). *Original figure reproduced from Innocenti et al., 2025, under CC BY-NC-ND 4.0 (no changes made).*

Disruption of this process can result in the misrouting or absence of callosal fibers, as observed in corpus callosum agenesis, a condition frequently associated with cognitive and behavioral abnormalities (Paul et al., 2007). The structural and functional integrity of homotopic projections appears particularly important, as deficits in these circuits have been linked to neurodevelopmental and psychiatric disorders, including autism spectrum disorder, attention deficit hyperactivity disorder (ADHD), Down syndrome, and schizophrenia (Anderson et al., 2011; Hull et al., 2017). In cases of agenesis or hypoplasia, alternative commissural pathways and intrahemispheric reorganization may provide partial compensation; however, electrophysiological studies indicate that such adaptations are often insufficient, biasing processing towards intrahemispheric strategies and atypical patterns of lateralization (Bathurst & Kee, 1994).

Together, these findings underscore the importance of precise temporal and spatial coordination in the development of interhemispheric communication and highlight the vulnerability of this system to genetic, epigenetic, and environmental influences.

Evolutionary context of callosal and anterior commissure

Mammals rely on two major commissural systems to mediate interhemispheric transfer: the anterior commissure and corpus callosum. In eutherian mammals, both structures contribute to isocortical integration; however, the corpus callosum represents a unique evolutionary innovation absent in monotremes and marsupials (Aboitiz et al., 2003; Suarez et al., 2014). Fig. 1 and 2 illustrate this evolutionary shift by contrasting the broad, high-bandwidth callosal projections that link widespread isocortical regions with the more restricted anterior commissure, emphasizing how the emergence of the corpus callosum enabled increasingly complex bilateral integration in larger primate brains (Fig. 2). In primates, callosal pathways support nearly all isocortical integration (Fig. 5), whereas the anterior commissure is largely restricted to olfactory and temporal lobe connections. This macroscopic division of labor is mirrored at the microstructural level, as illustrated in Fig. 5f: regional variations in callosal fiber diameter and packing density determine conduction velocity and, in turn, the temporal fidelity of interhemispheric coupling.

Diffusion-weighted MRI studies have confirmed that human callosal fibers are the largest in the precentral and postcentral gyri, corresponding to the motor and somatosensory cortices, while smaller diameters in the associative areas predict slower conduction and reduced interhemispheric synchrony (Fig. 5f; Aboitiz et al., 1992; Innocenti et al., 2022). Comparative neuroanatomical analyses indicate that reliance on the anterior commissure imposes bandwidth and efficiency constraints when brain volume exceeds ~50 ml (Olivares et al., 2001). The evolutionary emergence of the corpus callosum in eutherians is thus regarded as a critical adaptation that enabled rapid and high-capacity integration in larger brains (Ashwell, 2016).

Interestingly, in certain callosotomized patients, alterations in interhemispheric connectivity have been detected specifically in the ventral and dorsal pontine decussations of cerebellar commissures (Hamdi et al., 2023, Chan et al., 2019). These findings suggest that brainstem–cerebellar pathways may contribute to the behavioral compensation observed in classic split-brain studies (Clarke & Zaidel, 1989, 1994; Eviatar & Zaidel, 1994). This perspective resonates with theoretical models proposing a movement-based origin of cognition, in which cortical columns predict the sensory consequences of actions and mental processes, and verification pathways through cerebellar circuits may serve as a secondary substrate for interhemispheric coordination (Box. 1; Hawkins et al., 2019). Box 1 elaborates this framework by schematically integrating sensorimotor prediction, cerebellar verification loops, and callosal transfer, thereby outlining a concrete circuit-level mechanism through which movement-based models of cognition may generate flexible interhemispheric coordination.

Cellular Architecture of Interhemispheric Connectivity

Corpus callosum and pyramidal cell dynamics

The human corpus callosum is the principal conduit for interhemispheric communication, integrating sensory, motor, and associative information across the cerebral hemispheres. It is predominantly composed of myelinated axons originating from cortical pyramidal neurons, which form the backbone of excitatory long-range projections in the neocortex (Innocenti, 1986; Rockland & Pandya, 1979). Although these axons are excitatory by phenotype, their terminations frequently recruit local inhibitory interneurons within the contralateral cortex, thereby establishing a finely tuned balance between excitation and inhibition across hemispheres (Fig. 8; Demir & Rosas, 2024; Szczupak et al., 2023). Fig. 9 illustrates the engagement of defined interneuron populations by callosal pyramidal bou-

Box 1. Predictive Coding as a Framework for Interhemispheric Integration.

Jeff Hawkins and colleagues (Hawkins et al., 2017; Lewis et al., 2022; George & Hawkins, 2009) have proposed that the neocortex is fundamentally organized around a movement-based predictive coding framework. According to this view, each cortical column continuously generates predictions about the next sensory input based on an internal model of the world, refined by both actual movements and simulated (mental) movements. Cognition, in this perspective, emerges as a hierarchical extension of motor prediction: the brain's capacity to forecast the sensory consequences of action forms the substrate for perception, memory, and abstract reasoning (Hole & Ahmad, 2021; Hawkins, 2019).

In the context of interhemispheric communication, such a framework raises intriguing possibilities. If cortical columns are fundamentally predictive units, then cross-hemispheric pathways may serve as verification channels, ensuring that predictions generated in one hemisphere can be checked, complemented, or corrected by contralateral networks. The cerebellum, long known for its role in motor prediction and error correction, could act as an additional hub in this system, relaying predictive signals across hemispheres *via* its bilateral connections to the cortex and brainstem (Clarke & Zaidel, 1989, 1994; Sun et al., 2022).

Although this account remains speculative, it highlights a provocative possibility: that the evolutionary expansion of callosal and cortico–cerebellar loops in primates may not only have enabled higher-order cognition but also embedded predictive coding into the very fabric of interhemispheric integration. If so, interhemispheric communication might be understood less as a simple exchange of information and more as a bihemispheric negotiation of predictions, aligning perception, action, and cognition across the two halves of the brain.

tons, whereas Fig. 8 depicts the resulting feed-forward inhibitory architecture.

Functionally, callosal projections ensure temporal and spatial coherence of cortical processing, supporting unified perception, motor coordination, and higher-order cognitive integration. They also provide the anatomical substrate for dynamic hemispheric specialization, enabling transient dominance shifts depending on task demands. Variations in callosal fiber density, axonal diameter, and regional distribution across cortical fields have been linked to species-specific degrees of lateralization and integrative flexibility (Aboitiz et al., 1992; Olivares et al., 2001).

This general organizational framework developed from more specialized commissural systems in the older cortex of the limbic forebrain, which first evolved distinct architectures and memory (hippocampus) and emotional (amygdala) lateralization. These fundamental functions give basis not only for executive functions but also for cognitive processing. Interspecies differences highlight the need for caution when extrapolating rodent data to human neuropsychology and underscore the importance of considering evolutionary divergence in the cellular basis of interhemispheric connectivity. Therefore, the subsequent sections will examine further the hippocampal and amygdalar commissures which exemplify how interhemispheric coordination has diversified from direct monosynaptic coupling in rodents to more distributed, polysynaptic mechanisms in primates.

Layer-specific routing and dendritic targeting

Callosal axons traverse the cortex in a highly layer-specific manner (Fig. 8). In rodents, the majority of contralateral projections arise from pyramidal neurons in supragranular layers II/III, which contribute approximately 80% of callosal fibers, whereas approximately 20% originate from layer V neurons; only a small minority arise from layers IV and VI (Wise, 1975; Fame et al., 2011; Chovsepian et al., 2017; Ku & Torii, 2021).

Axons from layer II/III neurons primarily target homotopic regions in the contralateral cortex, terminating within the same supragranular layers II/III, although some extend into layer V. In contrast, layer V pyramidal neurons project more broadly, sending collaterals to both deep (layer V/VI) and superficial (layer II/III) layers of the opposite hemisphere. These layer V neurons are subject to inhibitory control mediated by contralateral inputs that recruit local interneurons, resulting in $GABA_B$-dependent suppression of apical dendritic activity in layer I (Fig. 3).

Evidence suggests that neuronal-derived neurotrophic factor (NDNF)-positive interneurons in layer I preferentially receive excitatory transcallosal input and relay inhibition onto layer V dendrites in rodent models (Palmer et al., 2013; Larkum, 2013; Hermans et al., 2018). Interneurons containing somatostatin (SOM-INs) provide an additional regulatory layer for shaping interhemispheric and intercolumnar interactions. As schematized in Fig. 8d, SOM-INs occupy a strategic position at distal dendrites, where they can selectively filter callosal inputs and determine whether interhemispheric signals promote potentiation, depression, or remain functionally silent, thereby coupling local plasticity rules to long-range communication. These neurons also selectively target the distal dendrites of pyramidal cells achieving callosal input and dynamically adjust the excitatory–inhibitory balance in a layer-specific manner, thereby influencing both local and long-range plasticity (Liguz-Lecznar et al., 2016;

Urban-Ciecko & Barth, 2016). Learning-induced plasticity has been shown to alter SOM-IN activity, demonstrating that they are not static inhibitory elements but highly adaptable modulators of cortical circuits (Cybulska-Kłosowicz et al., 2013). Importantly, manipulations of SOM-INs in the somatosensory barrel cortex reveal that inhibition within one column can regulate plastic changes in neighboring columns (Dobrzanski et al., 2022), suggesting a mechanism through which SOM-INs may indirectly influence interhemispheric processing as well. Consistent with this, bilateral representational changes in secondary somatosensory areas following classical conditioning highlight the capacity for inhibitory circuits to support experience-dependent plasticity across hemispheres (Debowska, et al., 2011). Together, these findings place SOM-INs at the interface of local inhibitory control and large-scale interhemispheric reorganization, making them a critical substrate for adaptive communication between the hemispheres. Interneurons in the marginal zone do not project through the corpus callosum (Chovsepian et al., 2017) but act as a critical relay, shaping how interhemispheric signals modulate the excitability of deep pyramidal neurons. In Fig. 7 and 8, these marginal-zone and layer I interneurons are integrated into a broader framework in which non-callosal inhibitory relays transform descending interhemispheric drive into depth-specific modulation of pyramidal output, reinforcing that "indirect" inhibitory pathways are a core component of interhemispheric gating.

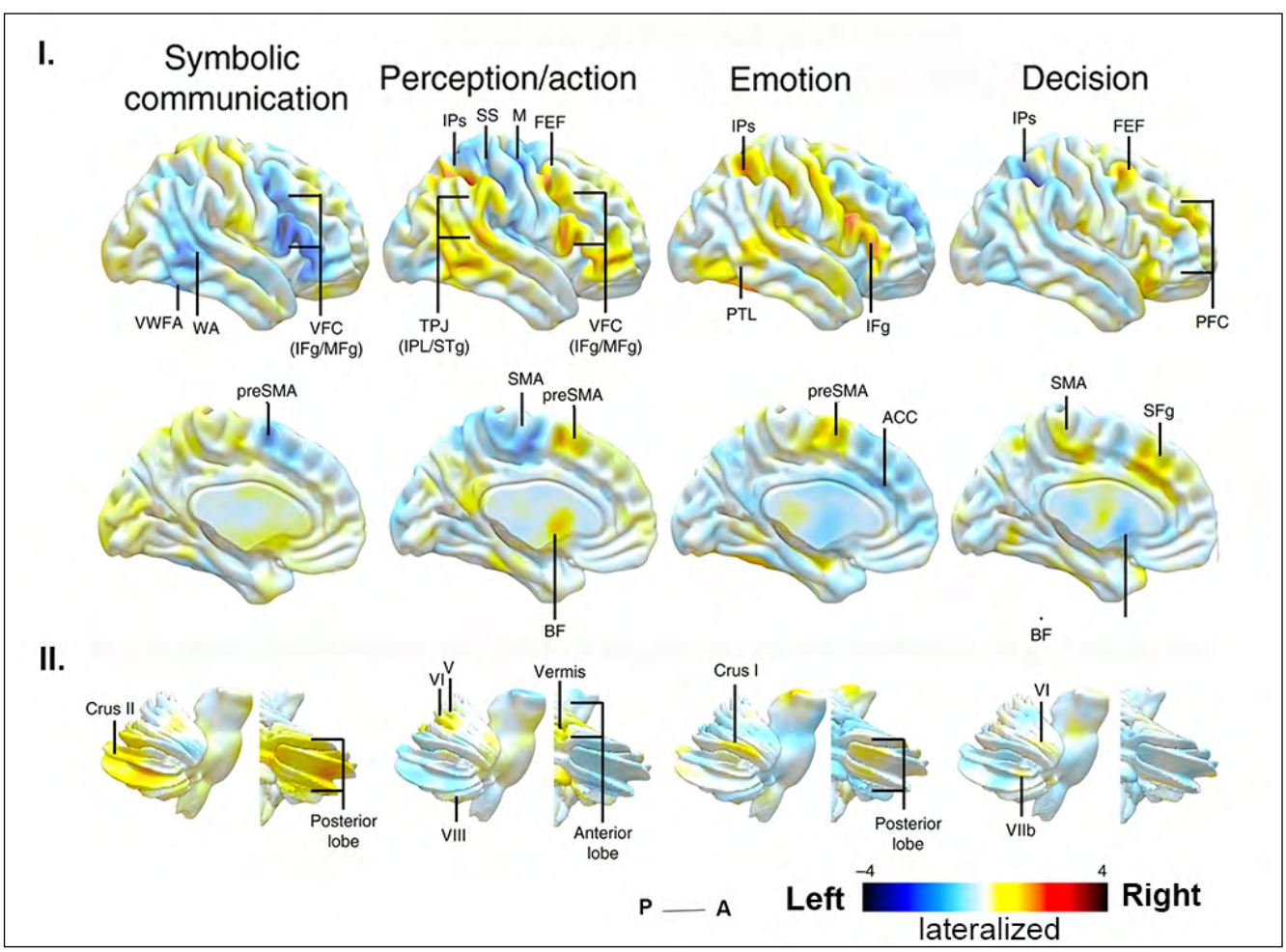


Fig. 7. Archetypes of functional lateralization. Archetype maps of symbolic communication, perception/action, emotion, and decision-making axes are shown for lateral and medial cortical views (I) and for the cerebellum (II). These maps were reconstructed from large-scale meta-analytic functional magnetic resonance (fMRI) data (Neurosynth) by computing laterality indices, applying dimensionality reduction, and extracting archetypal patterns of functional asymmetry. VWFA–visual word form area; WA–Wernicke's area; VFC–ventral frontal cortex; IFg–inferior frontal gyrus; MFg–middle frontal gyrus; TPJ–temporo-parietal junction; IPL–inferior parietal lobule; STg–superior temporal gyrus; IPs–intraparietal sulcus; SS–somatosensory cortex; M–motor cortex; FEF–frontal eye field; PTL–posterior temporal lobe; PFC–prefrontal cortex; SMA–supplementary motor area; preSMA–presupplementary motor area; ACC–anterior cingulate cortex; BF–basal forebrain. *Original figure reproduced from Karolis et al.(2019), with permission, under CC_BY.*

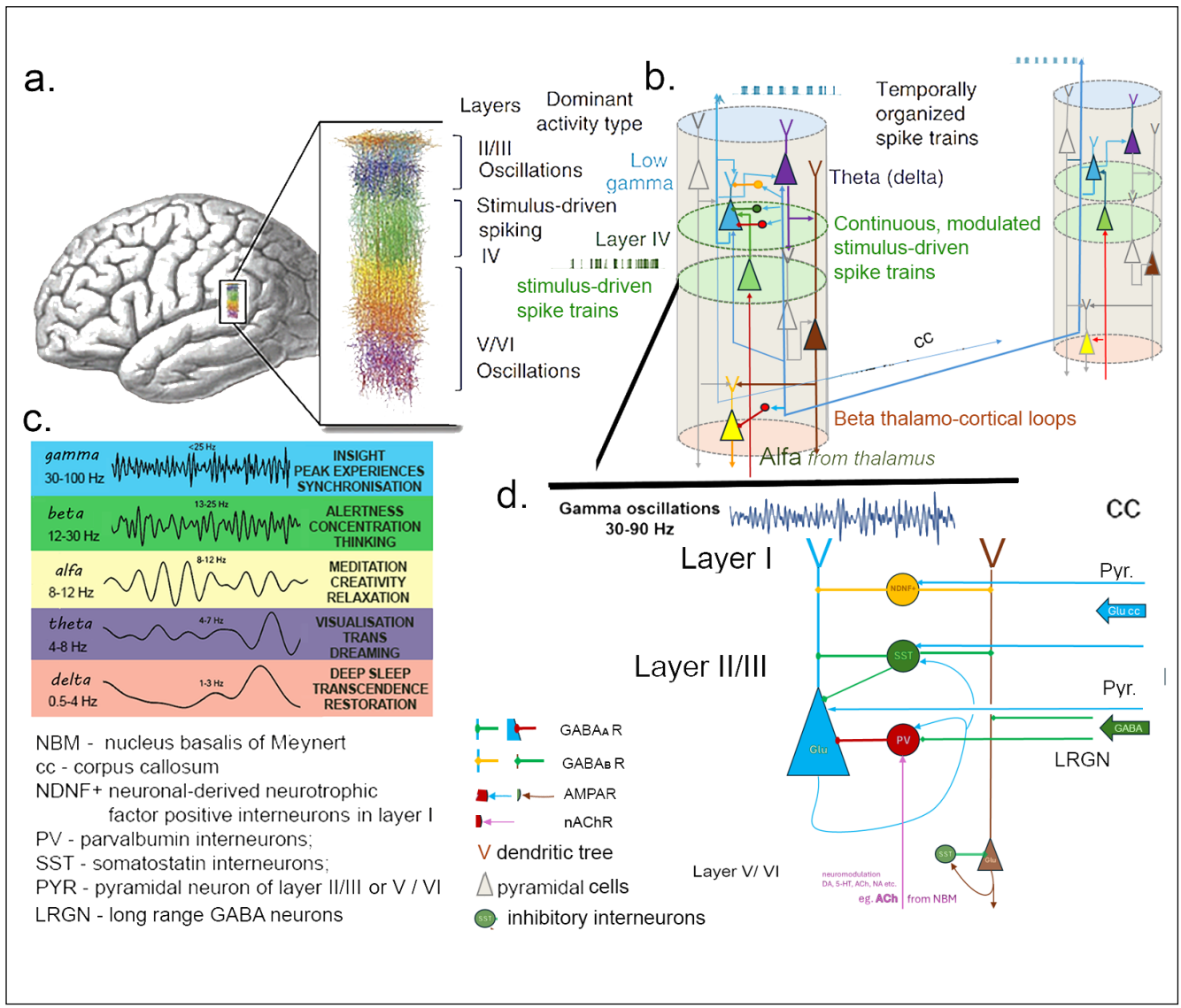


Fig. 8. Schematically integrates laminar cortical organization, oscillatory dynamics, and long-range interhemispheric and neuromodulatory influences into a unified circuit-level framework. a. The panel illustrates the fundamental laminar segregation of cortical processing. Oscillatory activity emerges predominantly from vertically aligned apical dendrites of pyramidal neurons in superficial (layers II/III) and deep layers (V/VI), where dendro-dendritic and recurrent interactions favor rhythmic synchronization. In contrast, layer IV acts as the principal recipient of sensory thalamic input and is dominated by stimulus-driven spiking, reflecting feedforward encoding of external information. *Modified by the authors from Oberlaender et al., (2011) under CC_BY_NC 4.0.* b. Cortical oscillations arise from recurrent interactions between excitatory pyramidal neurons and inhibitory interneurons, forming frequency-specific microcircuits. Fast gamma rhythms are generated locally through PING/ING mechanisms involving parvalbumin-positive (PV+) and somatostatin-positive (SOM+) interneurons. Slower rhythms (theta and delta) preferentially engage NDNF+ interneurons in layer I, which modulate apical dendritic integration. Beta oscillations emerge from deeper corticothalamic and cortico–basal ganglia loops involving layer V pyramidal neurons, whereas alpha rhythms are reinforced by corticothalamic feedback *via* the thalamic reticular nucleus, emphasizing the role of thalamocortical resonance. *Created by authors inspired by Guan et al. (2022).* c. These oscillatory regimes map onto distinct cognitive and behavioral states, reflecting different modes of information processing. Gamma oscillations support perceptual binding and moments of insight; beta rhythms stabilize task engagement and attentional control; alpha rhythms accompany relaxed, internally oriented states; theta oscillations are linked to memory, imagery, and navigation; and delta rhythms dominate deep sleep and restorative processes. Importantly, these bands do not operate in isolation but coexist and interact dynamically. *Created by the authors.* d. Superimposed on this laminar and oscillatory scaffold are interhemispheric and neuromodulatory controls. Excitatory callosal inputs from contralateral layer II/III pyramidal neurons preferentially target superficial layers, synchronizing gamma activity across hemispheres and enabling precise temporal alignment of bilateral cortical processing. In parallel, long-range GABAergic neurons (LRGNs), primarily SOM+ interneurons projecting interhemispherically, inhibit apical dendrites of infragranular pyramidal neurons, implementing a powerful mechanism of interhemispheric inhibition. Neuromodulatory systems—including acetylcholine, dopamine, serotonin, and noradrenaline — globally tune excitability, gain, and oscillatory balance, biasing the network toward integration or segregation depending on behavioral context. *Created by the authors.*

Laminar specialization of callosal inputs means that they do not simply provide direct excitation but instead engage distinct inhibitory and excitatory circuits, depending on their cortical targets. Electrophysiological mapping in the rodent somatosensory cortex further shows that spontaneous interhemispheric activity localizes predominantly to infragranular layers, with distinct current sources and sinks consistent with layer-specific callosal input (Baek et al., 2016). Importantly, such laminar specificity is further modulated by neuromodulatory systems: cholinergic input, for example, exerts layer-dependent control of pyramidal neurons and interneurons, with supragranular layers showing enhanced excitation and infragranular layers receiving stronger inhibitory modulation (Obermayer et al., 2017). This indicates that the effectiveness and polarity of callosal inputs are not fixed but can be reweighted dynamically depending on neuromodulatory tone, adding an additional regulatory layer to interhemispheric communication.

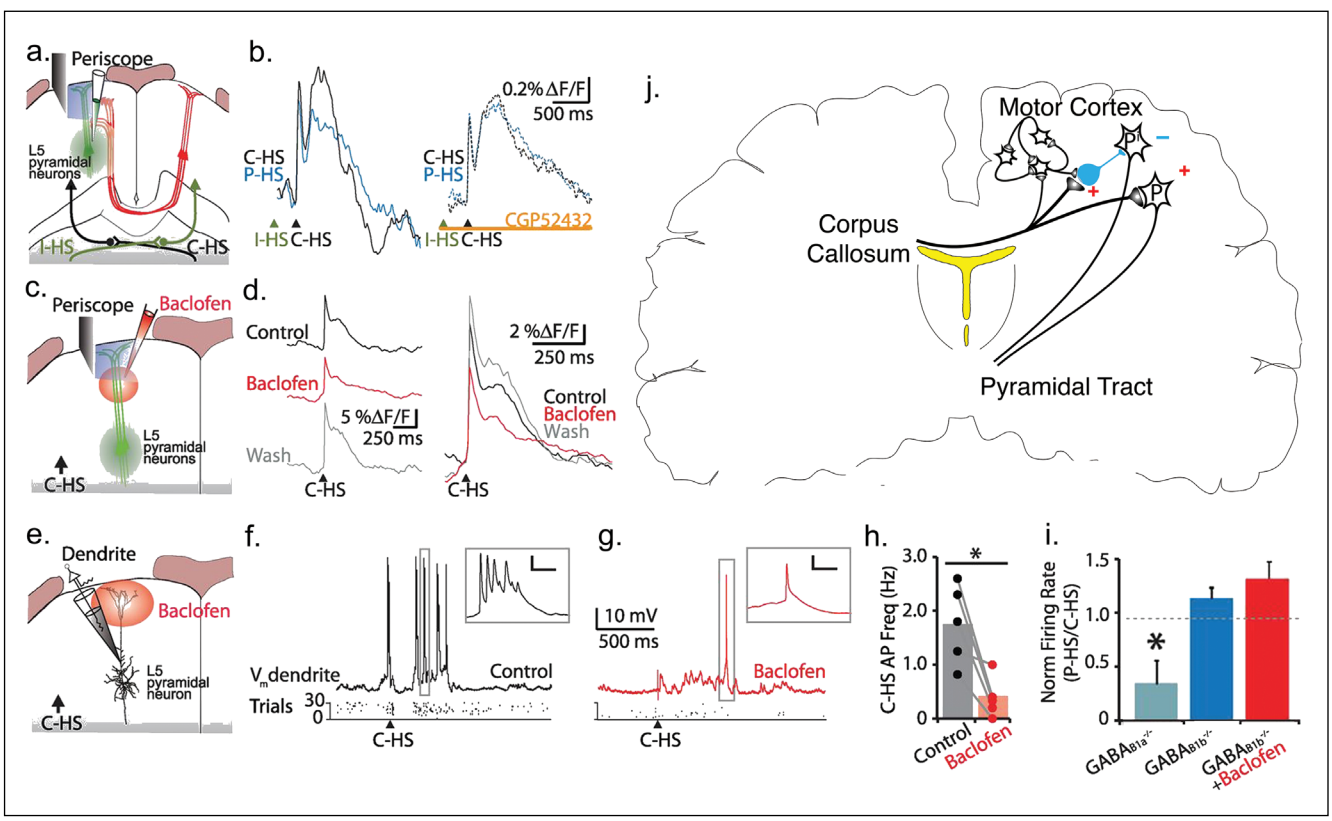


Fig. 9. Long-lasting interhemispheric inhibition is mediated by dendritic $GABA_B$ receptors which provides direct cellular evidence that interhemispheric inhibition is not mediated by fast, transient synaptic mechanisms, but instead relies on long-lasting dendritic $GABA_B$-dependent modulation of pyramidal neuron excitability. By acting on apical dendrites of layer V pyramidal neurons, callosally driven $GABA_B$ signaling imposes a sustained suppression of contralateral cortical output, effectively decoupling hemispheres over extended time windows. This mechanism establishes a biophysical substrate for stable hemispheric dominance, slow oscillatory coordination, and state-dependent lateralization, linking interhemispheric communication to dendritic integration rather than somatic spike control. a. *In vivo* two-photon calcium imaging of neural population activity in the contralateral to the stimulated hind limb (HS) sensorimotor cortex a calcium indicator dye is introduced into a population of layer V pyramidal neurons (OGB-1-AM); dendritic Ca^{2+} responses were recorded with periscope during contralateral and bilateral hindlimb stimulations (C-HS and P-HS respectively). b. (left) Average dendritic Ca^{2+} population response (fluorescence change, ΔF/F) during C-HS (black) and P-HS (blue). (Right) Ca^{2+} response to C-HS (black) and P-HS (blue) during application of selective antagonist of $GABA_B$ receptors (CGP52432) to cortical surface. c. (Left) Integral of the Ca^{2+} response to C-HS (black) and P-HS (blue) in control conditions and (right) during CGP52432 application. d. Average dendritic Ca^{2+} population response with C-HS before (black), during (red), and after (gray) baclofen application. (Left) Integral and (right) amplitude of Ca^{2+} response to C-HS. e. Experimental design: layer V dendritic patch during baclofen application. f. Dendritic patch-clamp responses to C-HS before (black) and g. during (red) baclofen. (Inset) Complex waveform from boxed region. Scale bar, 10 mV, 10 ms. h. Normalized firing rate in the dendrite to C-HS before (black) and (g) during (red) baclofen application. (i) Normalized somatic firing rate during C-HS and P-HS in $GABA_{B1a}^{-/-}$ and $GABA_{B1b}^{-/-}$ mice and during focal baclofen (50 μM) application (red) in mice lacking postsynaptic $GABA_B$ receptors ($GABA_{B1b}^{-/-}$). * $P<0.05$. j. interhemispheric feedforward inhibition bound to callosal stimulation. Baclofen–$GABA_B$ receptors agonist; C/I-HS: contra-/ipsilateral hindlimb stimulation (HS); P-HS: paired HS; *a–i –adapted with modification from Palmer et al. (2012), licence for modification from Science (New York, N.Y.),* DOI *10.1126/science.1217276, 2012, AAAS. j –adapted from Asanuma & Okuda 1962, CC_BY_SA 4.0, with permission.*

Long-Range GABAergic Neurons (LRGNs): Identity, Targets, and Functions

Although most GABAergic interneurons operate locally to sculpt excitatory ensembles within cortical microcircuits, a distinct subset of GABAergic neurons extends axons over long distances, including projections that traverse the corpus callosum. Such inhibitory commissural projections have been documented in the visual cortex of cats (Buhl & Singer, 1989; Peters et al., 1994), the somatosensory cortex of primates (Fabri & Manzoni, 2004), the auditory cortex of mice (Zurita et al., 2018), and the olfactory cortex of mice (Rock et al., 2018). Collectively, these studies establish the existence of transcallosal GABAergic connectivity across species, although their abundance, laminar origin, and functional roles appear to vary across cortical areas and taxa.

Available anatomical reconstructions indicate that commissural GABAergic axons in mice can reach lengths exceeding 4–6 mm, consistent with genuine long-range projections (Gonchar et al., 1995; Tamamaki & Tomioka, 2010). These neurons arise predominantly from infragranular layers, particularly layer VI and the deep portion of layer V (Gonchar et al., 1995; Tomioka et al., 2005; Fabri & Manzoni, 2004; Zurita et al., 2018), although contributions from supragranular layers have also been reported depending on cortical area and species (Gonchar et al., 1995; Tomioka et al., 2005). Rather than constituting a uniform population, callosal LRGNs thus represent a heterogeneous class of projection neurons whose laminar origins partially overlap with those of excitatory callosal pyramidal neurons but remain quantitatively and qualitatively distinct.

Origin, trajectories, and termination patterns

Across cortical regions, callosal LRGNs have been reported to originate from layers II/III, V, and VI, with relative proportions varying by area and species. Axons typically exit the parent column through infragranular layers, (Gonchar et al., 1995; Tomioka et al., 2005), enter the underlying white matter, traverse the corpus callosum, and terminate preferentially in homotopic regions of the contralateral hemisphere (Fig. 3, 10; Fabri & Manzoni, 2004; Zurita et al., 2018). Terminal arbors are most consistently observed in superficial layers (L1–L3), with additional, sparser boutons present in deeper layers, including layer V (Gonchar et al., 1995; Tomioka et al., 2005).

At the synaptic level, available evidence indicates that long-range GABAergic projections preferentially avoid perisomatic targets and instead innervate dendritic compartments of pyramidal neurons, (Gonchar et al., 1995; Tomioka et al., 2005; Tamamaki & Tomioka, 2010), as well as local inhibitory interneurons (Tomioka et al., 2005; Caputi et al., 2013). While precise postsynaptic specificity remains incompletely resolved for callosal LRGNs *per se* (Tamamaki & Tomioka, 2010; Zurita et al., 2018), converging data from long-range GABAergic systems suggest interactions with dendrite-targeting interneuron classes, including somatostatin-positive and NPY-positive populations, (Caputi et al., 2013; Melzer et al., 2017), and possibly neurogliaform-like cells associated with slow and volume-mediated inhibition (Tamas et al., 2003; Oláh et al., 2009). Importantly, these associations should be interpreted as probabilistic tendencies rather than exclusive wiring rules. Functionally this laminar and subcellular bias is consistent with a role for callosal LRGNs in modulating dendritic integration, synaptic gain, and temporal coordination of contralateral inputs, rather than mediating fast perisomatic inhibition.

Molecular profile and GABAergic subclasses

Long-range GABAergic neurons exhibit substantial molecular heterogeneity, reflecting diversity in both projection targets and developmental origin (Tomioka et al., 2005; Tamamaki & Tomioka, 2010). Corticocortical LRGNs linking distributed cortical territories, including contralateral homologues, are frequently associated with markers such as somatostatin (SOM), neuronal nitric oxide synthase (nNOS), and neuropeptide Y (NPY), and in some regions calretinin (CR) (Tomioka et al., 2005; Gonchar et al., 1995; Zurita et al., 2018, Yan et al 1996). In the primate cortex, long-distance inhibitory neurons expressing calbindin (CB) and CR have been described in detail (Tomioka & Rockland, 2007; Fabri & Manzoni, 2004). These molecular profiles overlap with Martinotti-like, neurogliaform-like, and ivy-like interneuron phenotypes (Tomioka et al., 2005; Caputi et al., 2013), although direct one-to-one correspondence between molecular class and projection pattern cannot be assumed.

By contrast, corticofugal GABAergic projection neurons targeting subcortical structures more frequently express parvalbumin (PV), often lack nNOS immunoreactivity, and may include minor VIP-positive subpopulations (Tomioka et al., 2005). This neurochemical divergence underscores that long-range inhibition encompasses multiple functionally distinct subclasses rather than a single canonical circuit motif.

Evidence from primate studies

Long-range GABAergic projections are not restricted to rodents. In the macaque cortex, Tomioka

and Rockland (2007) identified inhibitory neurons whose axons extend 8–12 mm through both gray and white matter, forming corticocortical inhibitory pathways that substantially exceed the typical projection lengths observed in rodents. These neurons were distributed across multiple cortical areas and exhibited laminar termination patterns biased toward supragranular layers, consistent with dendrite-targeting inhibitory motifs.

Although explicit transcallosal trajectories were not reconstructed in this study, the combination of axonal extent, laminar origin, and terminal distribution provides strong evidence for the existence of long-range corticocortical GABAergic pathways in primates. Complementary immunohistochemical analyses in the monkey somatosensory cortex further demonstrated that a subset of callosally projecting neurons expresses glutamic acid decarboxylase (GAD), confirming the presence of inhibitory commissural projections in primate neocortex (Fabri & Manzoni, 2004).

Together, these findings indicate that long-range inhibitory corticocortical neurons are conserved across species and are likely to play an increasingly important role in larger, more spatially extended cortices. While direct functional characterization of callosal LRGNs in primates remains limited, the available anatomical evidence supports the notion that dendrite-targeting long-range inhibition constitutes an evolutionarily conserved circuit motif, potentially contributing to in-

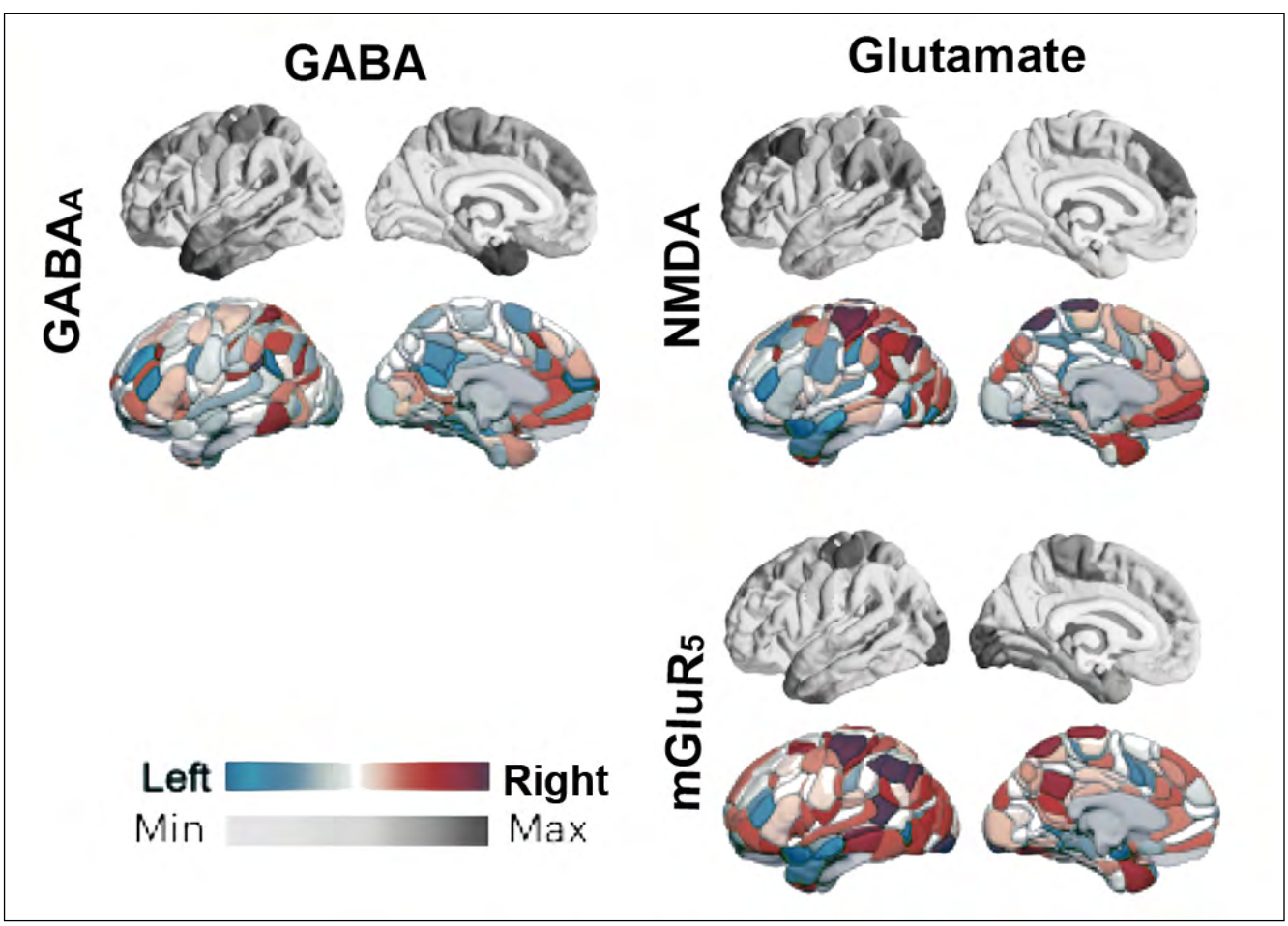


Fig. 10. Asymmetry of normalized glutamate and GABA receptor densities. Positron emission tomography (PET) density maps illustrate the distribution of major excitatory (glutamate) and inhibitory (GABA) receptor types ($GABA_A$, NMDA, mGluR5) in the human cortex. Data were obtained from 1,238 healthy participants in the BIL&GIN database (Mazoyer et al., 2016), all identified as left-hemisphere dominant for language (Labache et al., 2023). For each receptor, the top row (grayscale) shows mean density in the left hemisphere, while the bottom row depicts asymmetry maps, calculated by subtracting left from right hemisphere values across 163 homotopic cortical regions of the AICHA atlas (Joliot et al., 2015; Hansen et al., 2022). Asymmetries are projected onto the left hemisphere surface using a color scale (blue=leftward, red=rightward). Surface renderings in MNI space were generated with Surf Ice software (https://www.nitrc.org/projects/surfice/). *Hansen et al., 2022 joined with Labache et al., 2025; Springer Nature, with permissions, under CC_BY.*

terareal coordination and hemispheric integration in large-brained mammals.

Developmental considerations

Developmental analyses further suggest that distinct subclasses of GABAergic projection neurons follow different temporal trajectories. In the mouse, contralateral GABAergic projections increase during early postnatal development, whereas corticofugal inhibitory projections are relatively more prominent perinatally and decline thereafter (Tamamaki & Tomioka, 2010). These dynamics imply shifting roles for long-range inhibition during circuit assembly, refinement, and the emergence of mature interhemispheric coordination.

Functional implications of callosal LRGNs

Functionally, commissural GABAergic projections described in classical and contemporary studies (Buhl & Singer, 1989; Peters et al., 1994; Fabri & Manzoni, 2004; Rock et al., 2018) are predominantly homotopic and may be viewed as inhibitory counterparts to excitatory callosal pyramidal pathways. By acting on dendritic compartments and local inhibitory networks, callosal LRGNs are well positioned to modulate gain, suppress noise, and shape the temporal structure of interhemispheric interactions.

Speculatively, such circuits may contribute to oscillatory phase coordination, bilateral sensory integration, and the stabilization of functional lateralization, particularly in conditions requiring fine-grained balance between hemispheric cooperation and competition. While considerably sparser than excitatory callosal projections, long-range inhibitory pathways likely constitute a precision modulatory channel whose influence is disproportionate to their numerical representation.

Hippocampal Commissures

One striking evolutionary divergence in interhemispheric architecture concerns the hippocampus, which phylogenetically represents one of the earliest cortical-like structures, characterized by its trilaminar cytoarchitecture. A specialized subpopulation of CA1 pyramidal neurons extends neurites from basal dendrites to receive robust excitatory input from contralateral CA3 neurons, thereby coordinating mnemonic processing across hemispheres (Stevens et al., 2024). In rodents, the hippocampal formation assumes a characteristic C-shaped configuration within the medial temporal lobe, arching dorsally around the diencephalon and thalamus and positioned medially relative to the basal ganglia (Amaral & Lavenex, 2007). The dorsal hippocampus receives input from medial entorhinal cortex (MEC) regions containing grid cells with the smallest spatial scales, thereby providing the highest spatial resolution (Brun et al., 2008). In contrast, the ventral hippocampus exhibits strong connectivity with the prefrontal cortex and amygdala, supporting its role in affective processing and emotional regulation (Fanselow & Dong, 2010).

In primates, the hippocampus is positioned ventrally within the medial temporal lobe and is organized primarily along the anterior–posterior axis. Functionally, the anterior hippocampus in primates corresponds to the ventral hippocampus in rodents, whereas the posterior hippocampus corresponds to the rodent dorsal hippocampus (Strange et al., 2014).

Rodents possess robust bilateral hippocampal commissures, including dorsal and ventral pathways, which mediate monosynaptic projections from the CA3 to the contralateral CA1 and CA3 regions (van Groen & Wyss, 1990; Amaral & Lavenex, 2007). In primates, interhemispheric homologous hippocampal regions course through the posterior body of the corpus callosum and adjacent white matter, reflecting a reliance greater dependence on callosal and paralimbic pathways (Amaral et al., 1984; Demeter et al., 1985). The diffusion-based tractography and multimodal anatomical studies demonstrate that the forniceal commissure is strongly vestigial, consisting only of a sparse set of midline fibers with limited continuity between homologous hippocampal fields (Akeret et al., 2022).

Instead, communication between the left and right hippocampi is routed through polysynaptic, transcallosal and paralimbic circuits — most prominently *via* the retrosplenial cortex (Ziontz et al., 2021), cingulum bundle (Bubb et al., 2018), and splenial callosal fibers (Huang et al., 2021). This reorganization likely reflects the increasing lateralization of hippocampal operations in primates, particularly the segregation of verbal and spatial mnemonic processes to the left and right hemispheres, respectively (Burgess et al., 2002). To contextualize these multimodal differences, Fig. 7 synthesizes the functional interhemispheric connectivity gradients across the human hemispheres.

This rerouting is consistent with broader principles of primate white-matter evolution, in which interactions within the medial temporal lobe increasingly rely on distributed association pathways rather than direct commissural tracts. Despite the minimal structural continuity of the forniceal commissures (Akeret et al., 2022), human hippocampal networks maintain robust bilateral functional coupling, reflecting a shift toward large-scale integrative dynamics. Importantly, the ap-

parent paucity of commissural fibers may be amplified by the inherent limitations of diffusion MRI tractography in resolving small or lightly myelinated pathways (Thomas et al., 2014), further underscoring the evolutionary move away from monosynaptic interhippocampal communication.

Amygdala Interhemispheric Routes: Rodent *vs.* Primate

The amygdala (Am), a central limbic hub integrating emotional processing, motivation, and associative learning, exhibits species-specific patterns of interhemispheric connectivity.

In rodents, commissural fibers between the amygdalae are mediated primarily *via* the anterior commissure, with the basolateral amygdala (BLA) serving as the principal source of long-range projections. These axons innervate both homotopic and heterotopic regions of the contralateral amygdala, as well as associated structures, such as the nucleus accumbens and bed nucleus of the stria terminalis, enabling rapid bilateral coordination of affective and reward-related responses (Hetzel & Rosenkranz, 2014; Huang et al., 2019). Fig. 4 visualizes the differential interhemispheric inhibitory and excitatory pathways supporting lateralized affective processing in the amygdala and its cortical targets. It provides a circuit-level overview of these rodent amygdala commissural pathways, highlighting how BLA projections *via* the anterior commissure converge onto contralateral amygdala and ventral striatal targets to support rapid bilateral alignment of affective and reward-related processing.

Electrophysiological evidence demonstrates that these commissural pathways synchronize oscillatory activity between hemispheres during emotionally salient events, enhancing the salience and consolidation of affective memories (Popa et al., 2010). Pharmacological and lesion-based studies have further shown that disruption of contralateral BLA connectivity impairs social interaction, conditioned fear, and emotional memory formation in rodents (Huang et al., 2019). As in cortical and hippocampal systems, local interneurons in the target hemisphere provide inhibitory feedback, ensuring interhemispheric balance.

In primates, including humans, direct amygdala-to-amygdala projections are weak or inconsistent. Diffusion tensor imaging and tract-tracing studies indicate that interhemispheric communication involving the amygdala occurs predominantly *via* indirect polysynaptic routes through cortical hubs, particularly the orbitofrontal cortex, anterior temporal cortex, and cingulate gyrus, conveyed through the corpus callosum (Bach et al., 2011; Catani et al., 2013). Functional imaging further revealed that amygdala interhemispheric coupling is highly context-dependent, increasing during tasks involving emotional face recognition and threat detection, and in psychiatric disorders such as major depressive disorder (Irwin et al., 2004; Ocklenburg et al., 2018).

This evolutionary transition from direct subcortical commissural coupling in rodents to cortically mediated polysynaptic coordination of limbic functions in primates illustrates the broader shift toward distributed control of emotional integration. It highlights the need for translational caution when extrapolating rodent data to human affective neuroscience.

Novelty of the thalamic pathway

For decades the corpus callosum and other commissures were considered the sole interhemispheric pathways, with corticothalamic loops thought strictly ipsilateral. Szczupak et al. (2021) overturned this long-held assumption by identifying axons from the primary motor cortex that cross through the anterior commissure and previously undescribed intrathalamic bundles connecting rostral cortical regions (e.g., orbitofrontal cortex) to the contralateral thalamus. Despite Szczupak et al. (2021) claim that these thalamic commissures represent the first description of the direct corticothalamic commissural routes, some suggestions have been already previously made by researchers working on the rat barrel cortex model (Allowey et al., 1986).

Subsequent anatomical mapping revealed that commissural inputs to thalamus in rats arise mainly from contralateral prefrontal, medial motor, somatosensory and insular cortices, whereas rarely occipital and temporal areas. Data collected in the Allen Brain Institute mouse connectivity atlas (2011) reveals that the cortical inputs to thalamic nuclei, i.e., corticothalamic projections, are abundant and exhibit a marked rostral bias in their cortical origins. As outlined in Fig. 14f, these rostrally concentrated cortical sources position thalamic commissures as a potential parallel route for shaping contralateral thalamic gating, enabling one hemisphere to modulate thalamic excitability and thereby influence cross-hemispheric synchrony independently of the corpus callosum. The projections are less dense than ipsilateral corticothalamic tracts but exhibit bilateral symmetry, and no ascending fibers were found from the thalamus, indicating strictly top down communication. Four dis-

crete patches along the rostrocaudal axis carry these fibers, and the insular cortex, despite being a weak ipsilateral thalamic afferent, provides the strongest contralateral thalamic input (Meneses Iack et al., 2025).

To test evolutionary conservation, Szczupak et al. (2024), used diffusion MRI, viral tracing and resting state functional magnetic resonance imaging (fMRI) in New-World and Old-World primates; thalamic commissures were found in marmosets, capuchins and macaques and originated from frontal and orbital areas. The thalamic commissures develop during human embryogenesis, suggesting a conserved developmental program, yet none were detected in healthy adult humans. Thalamic commissures did appear in individuals with callosal dysgenesis, implying that they may serve as an alternative pathway when canonical commissures are absent or compromised (Szczupak et al., 2024). Box 2 expands on this compensatory hypothesis by depicting how thalamic commissures re-emerge in conditions of callosal dysgenesis, detailing the rostrocaudal distribution of these fibers and contrasting their role with that of canonical cortico-cortical commissures discussed in earlier sections.

Comprehensive analyses indicate that contralateral projections follow a rostro-caudal gradient and preferentially target higher order thalamic nuclei involved in cognitive control and arousal (Giguere & Goldman-Rakic, 1988; Mukherjee et al., 2021). Because thalamic commissures transmit signals solely from cortex to the opposite thalamus (Szczupak et al., 2021), they may enable one hemisphere to modulate thalamic gating in the other, thereby shaping cross hemispheric synchrony (Bardon et al., 2025) and supporting functional compensation when callosal pathways are disrupted (Box 2).

Macro-Evolutionary Trends: From Direct Subcortical to Cortically Mediated Integration

Above-mentioned alternative connectivity pathways exemplify a broader evolutionary trend: a shift from predominantly direct subcortical commissural connectivity in evolutionarily older mammalian lineages to primarily indirect cortically mediated integration in species with larger and more modular brains. In primates, the marked reduction of hippocampal commissures and the weakened anterior commissure contribution to amygdala connectivity appear to have promoted greater hemispheric autonomy and functional specialization in complex cognitive domains. However, this reorganization likely comes at the cost of slower and less synchronized cross-hemispheric processing, which in turn fosters hemispheric dominance in specific aspects of perception and cognition. Through lateralized attentional mechanisms, such dominance further consolidates the asymmetry of associated cognitive processes (Olivares et al., 2001; Gazzaniga, 2000).

Homotopic and Heterotopic Pathways

Most interhemispheric projections are homotopic, linking anatomically corresponding regions in the left and right hemispheres. fMRI studies have shown that synchronous activity supports unified perception and consistently demonstrated strong synchrony between homotopic regions, both at rest and during task performance (Gee et al., 2011; Hinkley et al., 2012). Fig. 7a–b exemplify this canonical scaffold by depicting tightly phase-locked responses between homotopic columns, visually emphasizing the symmetry-preserving nature

Box 2. Thalamic Commissures: Organization, Function, and Compensatory Role.

Recent anatomical and diffusion tractography studies have revealed previously underappreciated contralateral corticothalamic projections that traverse midline bridges within or near the anterior and habenular commissures (Giguère & Goldman-Rakic, 1988; Groenewegen, 1988). These fibers follow a rostrocaudal gradient, with prefrontal and cingulate origins projecting to higher-order thalamic nuclei such as the mediodorsal and intralaminar complexes, which participate in cognitive control, arousal, and attentional regulation (Mitchell, 2015; Saalmann & Kastner, 2011; Mukherjee et al., 2021).

Unlike the corpus callosum, which mediates direct corticocortical transfer, these thalamic commissures convey activity exclusively from cortex to the contralateral thalamus (Szczupak et al., 2021), enabling one hemisphere to modulate thalamic gating and excitability in the other. Such modulation may synchronize oscillatory activity across hemispheres (Bardon et al., 2025) and sustain bilateral coherence during periods of reduced callosal throughput, such as sleep, anesthesia, or callosal dysgenesis (Bonetti et al., 2023; Tyszka et al., 2011).

Functionally, these commissural routes may constitute an evolutionarily conserved compensatory substrate, capable of preserving interhemispheric communication when cortical commissures are compromised. Their existence highlights that bilateral coordination in the mammalian brain is supported not only by the corpus callosum but also by deep subcortical channels that dynamically maintain integration and specialization of hemispheres.

of these pathways and their role as the baseline mode against which heterotopic interactions diverge.

Homotopy: Synchrony and Suppressive Interactions

Homotopic callosal projections, composed largely of myelinated axons, connect the corresponding cortical columns—the functional subunits of the neocortex—across hemispheres (Innocenti, 1986; Salvador et al., 2005). These projections exhibit a high degree of topographic precision and symmetry, particularly in the primary and secondary sensory cortices. Electrophysiological evidence indicates that homotopic inputs can be both excitatory and inhibitory, depending on cortical layer and behavioral context (Innocenti & Frost, 1979). This functional duality is rendered explicitly in Fig. 8, where the contrast between reinforcing excitatory drive and transient inhibitory contralateral suppression is mapped onto specific microcircuit motifs, illustrating how identical callosal pathways may differentially modulate contralateral processing depending on layer and the circuit state. Fig. 8 provides a circuit-level illustration of these dual effects: Fig. 7a–c summarize how callosal inputs can either reinforce activity in matched contralateral columns or recruit local inhibitory interneuron networks that transiently dampen pyramidal cell firing, thereby implementing context-dependent gating of bilateral sensory representations. In a rodent model, Palmer et al. (2012) demonstrated that contralateral sensory input could transiently suppress the firing of layer V pyramidal neurons in the somatosensory cortex when paired with ipsilateral stimulation (Fig. 8). This suppression, mediated by layer I interneurons targeting apical dendrites, was contingent on active neuronal firing and dependent on metabotropic inhibitory mechanisms (Fig. 9). Fig. 7d specifically highlights this apical dendritic inhibition as a mechanistic substrate of interhemispheric competition, visually linking the microcircuit motif (layer I interneurons and their targets) to the macroscopic phenomenon of transient suppression in the engaged hemisphere.

From an evolutionary perspective Hawkins et al. (2019) have argued that the predominance of homotopic connectivity may reflect a conserved sensorimotor integration scaffold, essential for bilateral coordination in primitive nervous systems. This organizational bias likely underpins the synchronization of neural activity across the hemispheres and supports the bilateral integration of perceptual and cognitive functions (Box 1). Box 1 expands on this idea by contrasting a minimal, homotopy-dominated ancestral architecture with more elaborated mammalian configurations, emphasizing how a simple "mirror-linking" design can already support robust bilateral coordination while also providing a substrate on which later specializations (e.g., heterotopic projections) can be layered.

Heterotopy: Associative Integration as an Under-Recognized but Crucial Mechanism

In contrast, heterotopic connections link non-corresponding cortical regions across hemispheres. Historically underemphasized, these pathways are increasingly recognized as crucial for higher order associative processing. Rather than duplicating homotopic information, heterotopic projections enable cross-modal, non-redundant communication, integrating distinct cortical functions into global networks (Fei et al., 2024; Szczupak et al., 2023). In Fig. 5a we visualize this shift from mirror-symmetric to cross-associative wiring by depicting heterotopic callosal fibers that bridge non-identical cortical fields (e.g., prefrontal–parietal, temporal–parietal), thereby forming an anatomical substrate for distributed association networks. Complementary panels (Fig. 5b–c) outline how such heterotopic routes can bypass damaged homotopic channels or reroute information to preserved networks, providing a structural basis for compensatory interhemispheric reorganization.

Recent tractography and electrophysiological evidence suggest that heterotopic callosal projections are particularly enriched in the association cortices, where functional asymmetries and hemispheric specialization are most pronounced. Fig. 5a highlights this enrichment by mapping the densest heterotopic projections onto multimodal hubs, reinforcing the interpretation that associative integration, rather than symmetry preservation, is a primary function of these cross-field pathways.

These projections are thus thought to play a disproportionate role in supporting lateralized cognition by linking complementary rather than mirrored processes across hemispheres. Far from being secondary, heterotopic pathways may represent a key substrate for the flexibility and resilience of interhemispheric networks (Fei et al., 2024; Szczupak et al., 2023).

Structural and Functional Asymmetries

Structural Asymmetry of the Human Brain

Structural hemispheric asymmetries represent a fundamental organizational principle of the human brain, providing an anatomical foundation for lateral-

ized cognitive functions. One of the most consistently reported examples is the planum temporale on the superior temporal gyrus, typically exhibiting leftward enlargement, a feature thought to underlie the left hemisphere's dominance in phonological processing and speech perception (Geschwind & Levitsky, 1968; Tzourio-Mazoyer et al., 2010). Deviations from this asymmetry have been linked to schizophrenia and language-related disorders, including dyslexia (Shapleske et al., 1999; Oertel-Knöchel & Linden, 2011).

A second striking example is the fusiform face area (FFA) in the fusiform gyrus, which often shows rightward asymmetry in both structure and function, reflecting the right hemisphere's specialization for holistic face perception (Kanwisher et al., 1997; Pitcher et al., 2011). Similar rightward biases have been observed in regions subserving visuospatial attention, such as the posterior parietal cortex (Corbetta & Shulman, 2011). These rightward biases are captured in Fig. 7, where lateralization indices for FFA and posterior parietal regions are plotted to emphasize the co-occurrence of structural and functional asymmetry across domains (face processing, spatial attention), reinforcing the view that hemispheric specialization emerges from partially shared anatomical constraints.

These structural asymmetries emerge during development and are shaped by the interplay of genetic, epigenetic, and experiential influences (Sun & Walsh, 2006). Disruption of typical developmental trajectory of functional asymmetries may contribute to atypical lateralization patterns and even lead to neurodevelopmental and psychiatric conditions. Thus, structural asymmetry is not merely an anatomical curiosity but it is a clinically relevant dimension of brain organization.

Evolutionary Bases of Hemispheric Specialization

Hemispheric asymmetries are not unique to humans but have deep evolutionary roots across vertebrates. These asymmetries support the division of labor between hemispheres, thereby enhancing neural efficiency and avoiding redundancy (Vallortigara & Rogers, 2005). Remarkably, lateralization is not confined to vertebrates, but cephalopods, crustaceans, and insects also exhibit functional asymmetries in predator–prey responses, visual discrimination, and motor control (Frasnelli, Vallortigara, & Rogers, 2012; Byrne et al., 2006). This broad phylogenetic distribution suggests an adaptive imperative to differentiate the two mirrored processors, avoiding duplication and allowing parallel specialization.

Philosophically, McGilchrist (2010) has argued that the ultimate driver of this division is the need for two complementary modes of attention—one narrow and targeted, the other broad and vigilant—operating in parallel to optimize survival in complex environments. In Fig. 7, panel I, we transpose this conceptual distinction into a comparative neuroanatomical framework, schematically contrasting left-lateralized networks supporting focal, detail-oriented processing with right-lateralized networks subserving global, context-sensitive integration in humans. Fig. 15 links molecular differences in thalamic nuclei with large-scale patterns of hemispheric asymmetry providing a mechanistic bridge between abstract theoretical proposals on hemispheric attentional styles and empirically grounded structural and functional asymmetries in thalamocortical circuits.

In primates, leftward enlargement of the temporal regions associated with auditory–vocal processing, including the planum temporale, parallels left-hemisphere dominance for species-specific vocalizations in chimpanzees (Hopkins et al., 1998; Cantalupo & Hopkins, 2001). This continuity suggests that language-related asymmetry in humans likely evolved from pre-existing auditory–vocal specializations in a common ancestor. Similarly, rightward asymmetry in regions such as the fusiform gyrus for face recognition may have precursors in non-human primates, where right hemisphere biases in social visual processing have been documented (Parr et al., 2008). Beyond primates, birds and fish exhibit similar hemispheric biases for conspecific recognition and spatial vigilance, suggesting the convergent evolution of asymmetry as a general solution for efficient parallel processing (Vallortigara, 2000).

Overall, hemispheric asymmetry emerges as an ancient organizational principle that has been selectively refined in humans to support complex language, social cognition, and tool use. The combination of conserved and uniquely derived features underscores its dual role as both a universal and species-specific adaptation, with direct implications for understanding the neural basis of cognition, social cognition, and cognitive disorders.

Neurotransmitters and Electrophysiological Mechanisms

The efficacy and timing of interhemispheric communication depend critically on the interplay between excitatory and inhibitory neurotransmissions. Glutamate and GABA are the principal transmitters shaping callosal signaling, while neuromodulators such as dopamine (DA), acetylcholine (ACh), serotonin (5-HT),

and norepinephrine (NE) dynamically adjust gain and synchronization. Ultimately, the functional impact of neurotransmitters is determined not only by their presence but also by the receptor subtypes expressed, their localization within layers, and their developmental maturation (switch in receptor subunit composition). These receptor-level specializations introduce lateralized rules that strongly influence the interhemispheric integration (Fig. 9).

These neurotransmitters receptor-level biases are further illustrated in Fig. 9, which presents large-scale asymmetry maps for major excitatory (NMDA, mGluR5) and inhibitory ($GABA_A$) receptor classes across more than 160 homotopic cortical regions. These PET-derived gradients demonstrate that the architecture of excitation–inhibition balance is lateralized, providing a molecular substrate for the hemispheric differences in inhibitory tone, oscillatory dynamics, and plasticity discussed throughout next sections.

Glutamatergic Transmission (GluN2A/B Maturation; Lateralized Synaptic Rules)

Callosal axons primarily release glutamate, activating AMPA and NMDA receptors to mediate excitatory transmissions. AMPA receptors enable rapid synaptic signaling, while NMDA receptors support activity-dependent plasticity. Importantly, the subunit composition of NMDA receptors (GluN2A *vs.* GluN2B) varies across developmental stages and brain regions, shaping synaptic strength and plasticity, which was shown both on animal and human models (Mc Kay et al., 2018; Tumdam et al., 2024).

A developmental GluN2B-to-GluN2A switch marks synaptic maturation: GluN2B-rich receptors prolong depolarization and promote long-term potentiation (LTP) in the immature brain (McKay et al., 2012; Mareš et al., 2021), but later, GluN2A-containing receptors preferentially support LTP, while GluN2B receptors become more involved in long-term depression (LTD) (Gardoni et al., 2009). Thus, the timing of this subunit switch sets plasticity rules for interhemispheric circuits (McKay et al., 2018; Sun et al., 2017, 2022; Matta et al., 2011).

Evidence for hemispheric asymmetry in glutamatergic signaling is increasing. Capper-Loup et al. (2009; Fig. 9) found higher GluN2A mRNA expression in the left medial striatum than in the right striatum in rats, suggesting lateralization in circuits. Left-biased enrichment of GluN2A and its scaffolding protein PSD-95 in the lateral amygdala implicates glutamatergic asymmetry in affective dysregulation in human and rodent models (Karolewicz et al., 2008; Oh et al., 2012; Giza et al., 2006). Animal studies further show that imbalanced excitatory inputs can drive cardiac and behavioral asymmetries, highlighting the influence of glutamate on both autonomic and motor lateralization (Xavier et al., 2014; Sutton & Chandler, 2002). These excitatory inputs primarily reflect glutamatergic projections targeting autonomic and limbic nuclei, whose asymmetrical activation modulates both cardiac output and behavioral bias.

In summary, the differential expression of NMDA subunits across hemispheres provides a molecular substrate for lateralized plasticity, with implications ranging from developmental sensitive periods to mood disorders.

Continuum of Glutamatergic Laterality

The glutamatergic system, the brain's primary excitatory network, exhibits hemispheric lateralization along a graded continuum that is region- and task-dependent rather than uniform across the whole brain. Microdialysis and magnetic resonance spectroscopy (MRS) studies have revealed that glutamate release shifts dynamically with cognitive demands: in humans, greater task-induced glutamate increases occur in the left dorsolateral prefrontal cortex during verbal working memory, whereas the right hemisphere shows stronger increases during visuospatial tasks (Mohammadi et al., 2024).

This continuum extends into the sensory association cortices. Postmortem and Positron Emission Tomography (PET)/Magnetic Resonance Spectroscopy (MRS) studies in humans and animals have demonstrated subtle yet consistent receptor concentration asymmetries, particularly for NMDA receptor subunit composition. Emerging receptor mapping data suggest a possible left-hemisphere enrichment of NMDA receptor density within the planum temporale and superior temporal gyrus, potentially aligning with the left-hemispheric dominance in phonological and verbal processing (Palomero-Gallagher et al., 2019; Zilles & Amunts, 2018; Carlén et al., 2012; Marié et al., 2018) for related translational evidence). In parallel, fMRI-MRS studies have revealed greater right-hemisphere glutamate responsiveness in V1/V2 during visuospatial attention, pointing to an excitatory drive that favors right-lateralized spatial processing (DiNuzzo et al., 2022).

A similar gradient has been observed in the hippocampus. Studies on rodents have shown higher expression of GluN2B-containing NMDA receptors in the right hippocampus, correlating with spatial memory performance (Shinohara et al., 2008). At the synaptic level, optogenetic evidence indicates that inputs originating from the left CA3 produce more robust LTP in CA1 syn-

apses compared to those from the right CA3, consistent with lateralized differences in glutamate receptor composition (Kohl et al., 2011). Moreover, left CA1 synapses display higher GluA1: GluA2 AMPA ratios, biasing plasticity thresholds towards potentiation, thereby supporting the encoding of long-term information (Purkey & Dell'Acqua, 2020).

Finally, the callosal glutamatergic projections add another layer to this continuum. Excitatory inputs frequently terminate on pyramidal cells in layers II/III, but their downstream impact depends on the balance between direct excitatory drive and recruitment of local inhibitory interneurons. This interplay highlights that the effect of interhemispheric glutamatergic transmission is dynamic and context-dependent, varying with the excitatory–inhibitory balance across layers and circuits.

Neuropsychological Correlates of Glutamatergic Asymmetry

The molecular and synaptic asymmetries of hippocampal glutamate receptors are mirrored at the behavioral level. Some evidence in mice suggests that the right hippocampus favors the rapid integration of spatial information and ongoing computations, whereas the left hippocampus supports more stable long-term memory traces (Shinohara et al., 2008; Kohl et al., 2011).

Neuropsychological studies have provided clear evidence for functional dissociation. The left hippocampus is preferentially involved in verbal learning and episodic memory. Patients with left hippocampal lesions show impaired story recall and reduced verbal memory, while spatial memory often remains intact (Frisk & Milner, 1990). Similarly, in temporal lobe epilepsy, left hippocampal sclerosis is strongly associated with deficits in verbal recall (Glosser & Donofrio, 2001).

In contrast, the right hippocampus plays a dominant role in spatial and navigational memory. Damage to this region impairs topographic learning, route finding, and spatial recall in humans (Spiers et al., 2001). London taxi drivers, renowned for their exceptional navigational expertise, have been shown to possess enlarged posterior right hippocampi, reflecting experience-dependent specialization (Maguire et al., 1998). Consistent with this, Burgess et al. (2002) emphasized that the right hippocampus is essential for representing spatial layouts and navigation, whereas the left hippocampus is more critical for contextual and episodic details.

Taken together, these neuropsychological patterns resonate with receptor-level asymmetries: the leftward bias for GluA1: GluA2 ratios and stronger LTP induction align with their role in encoding durable verbal and episodic memories, while the rightward bias for GluN2B expression and distinct CA1 plasticity rules support a specialization in flexible spatial computation and rapid updating of memory representations during spatial navigation or even simple motor tasks.

GABAergic Modulation and Interhemispheric Inhibition

Inhibitory GABAergic signaling is equally critical for the interhemispheric balance. $GABA_A$ receptors mediate fast phasic inhibition, whereas $GABA_B$ receptors contribute to slower modulatory suppression.

Interhemispheric inhibition (IHI), studied using paired-pulse TMS, reflects the capacity of one hemisphere to suppress contralateral activity (Fitzgerald, 2020). An anatomical comparison confirmed that excitatory callosal terminals synapse onto GABAergic interneurons, engaging feedforward inhibition (Carr & Sesack, 1998).

A particularly clear mechanistic account of dendritic $GABA_B$-dependent interhemispheric inhibition emerges from the physiological experiments summarized in Fig. 9. Two-photon Ca^{2+} imaging (Fig. 9a) reveals that paired bilateral hindlimb stimulation (P-HS) produces a pronounced reduction in dendritic activity relative to contralateral stimulation alone, an effect abolished by pharmacological blockade of $GABA_B$ receptors (Fig. 9b–c). Complementary baclofen experiments (Fig. 9d–h) further show that activating these receptors suppresses dendritic excitability and decreases both dendritic and somatic firing rates. Crucially, Fig. 9j demonstrates that this dendritic suppression is embedded within a broader feedforward interhemispheric inhibitory circuit driven by callosal input. Callosal stimulation reliably recruits GABAergic interneurons that impose slow inhibitory gating onto layer V pyramidal neurons, thereby linking the dendritic $GABA_B$-mediated suppression observed in sensory cortex to a canonical interhemispheric feedforward inhibitory motif. This figure provides a conceptual bridge between the cellular-level dynamics recorded in Fig. 9a–i and the systems-level phenomenon of interhemispheric inhibition measured with paired-pulse TMS.

Together, the results presented in Fig. 9j establish that lasting interhemispheric inhibition arises through dendritic $GABA_B$ receptors acting within callosally driven feedforward circuits, providing a cellular substrate for the suppressive influence one hemisphere exerts on the other during bilateral sensorimotor processing.

Layer V pyramidal neurons, in particular, receive $GABA_B$-mediated inhibition *via* apical dendrites from callosal input during bilateral sensory stimulation. This inhibitory effect was somatotopically specific in the hind limb representation in mice (Palmer et al., 2012).

Electrophysiology in the auditory cortex shows an early excitatory response followed by a delayed inhibitory contralateral response (Mitani & Shimokouchi, 1985), which is a hallmark of interhemispheric inhibition.

Perisomatic interneurons (PV+ basket and chandelier cells) provide precisely timed inhibition of pyramidal neurons at their soma as presented on Fig. 8d, proximal dendrites, or axon initial segments. This inhibition does not merely silence excitatory cells but resets their firing phase, promoting synchronous rebound activity and supporting oscillatory coordination across the hemispheres.

While most GABAergic neurons are local, a subset of long-range GABAergic projection neurons crosses the corpus callosum, directly mediating interhemispheric inhibition (Buhl & Singer, 1989; Peters et al., 1994; Fabri & Manzoni, 2004; Rock et al., 2018).

Is There Lateralization in the Inhibitory Networks?

Unlike glutamate, GABA shows less consistent hemispheric asymmetry. MRS studies in healthy adults have reported no significant left–right GABA concentration differences in the frontal, parietal, or occipital cortices (Gao et al., 2013). However, during early development, GABA is initially excitatory due to high intracellular Cl^- levels, and the timing of its switch to inhibition differs slightly between hemispheres in hippocampal circuits (Khazipov et al., 2004; Khoshdel-Sarkarizi & Hami, 2019).

Receptor mapping showed subtle asymmetries in their density. Autoradiography in rodents indicates left > right $GABA_A$ receptor density in the CA1 and dentate gyrus (Poulter et al., 1992), whereas PET/SPECT in humans often reports rightward asymmetry of benzodiazepine-sensitive $GABA_A$ binding in cortical regions (Hipp et al., 2021). $GABA_B$ asymmetries have been noted in the postnatal hippocampus (right > left binding in CA2/dentate; Khoshdel-Sarkarizi, 2019), although findings in adults are inconsistent.

Overall, evidence suggests that GABAergic asymmetries are more transient and context-dependent than glutamatergic asymmetries, with developmental timing and receptor subtype distribution playing the largest roles (Fig. 10).

Neuropsychological Consequences of GABAergic Laterality

Although structural and receptor mapping studies point to only subtle and context-dependent asymmetries, neuropsychological and clinical data suggest that imbalances in GABAergic inhibition critically shape hemispheric specialization and vulnerability.

On the cognitive side, frontal alpha asymmetry, a widely studied electroencephalography (EEG) marker of affective style, has been linked to hemispheric differences in the inhibitory tone. Greater left frontal activity (relative to the right) is associated with approach-related affect and positive mood, whereas greater right frontal activity correlates with withdrawal and negative affect. Pharmacological studies have implicated GABAergic modulation of frontal networks as a key mediator of these asymmetries (Davidson, 2004; Reznik & Allen, 2018).

In the domain of motor control, interhemispheric inhibition (IHI) is essential for preventing mirror movements and ensuring unilateral dexterity. Disruption of GABAergic IHI mechanisms is a hallmark of post-stroke motor deficits in humans, wherein the reduced inhibitory tone from the damaged hemisphere allows maladaptive over-activation of the intact hemisphere (Murase et al., 2004). These imbalance can delay recovery, but therapeutic modulation of GABAergic tone, whether through non-invasive stimulation such as transcranial direct current stimulation tDCS (Bachtiar et al., 2015; Stagg et al., 2011) or pharmacological reduction of excessive tonic inhibition (Clarkson et al., 2010), has shown promise in rebalancing hemispheric interactions after stroke in people (Murase et al., 2004).

Developmentally, the timing of the GABA switch from excitatory to inhibitory (Khazipov et al., 2004) influences hippocampal circuit maturation. Subtle hemispheric differences in this switch may bias memory systems towards left-dominant verbal/episodic encoding or right-dominant spatial navigation. Disturbances in this process have been implicated in neurodevelopmental disorders, such as autism and schizophrenia, in which altered GABAergic inhibition contributes to atypical lateralization and impaired interhemispheric integration (Uhlhaas & Singer, 2010; Nelson & Valakh, 2015).

Taken together, while glutamatergic lateralization provides a stable scaffold for hemispheric specialization, GABAergic asymmetries appear to be more transient and state-dependent, acting as a fine-tuning mechanism that dynamically regulates excitatory dominance across hemispheres. When disrupted, these inhibitory imbalances manifest as both cognitive asymmetries (e.g., affective style, attentional biases) and clinical symptoms (e.g., mirror movements post-stroke, altered lateralization in psychiatric disorders).

Phylogenesis of Interhemispheric Inhibition

Evolutionary comparative studies indicate that phylogenetically older cortical systems (archi- and paleocortex) maintain denser, predominantly homotopic interhemispheric projections with strong inhibitory control (Aboitiz & Montiel, 2003; Suárez et

al., 2014), whereas newer associative regions exhibit sparser and more heterotopic callosal connectivity with complex excitatory–inhibitory interactions (Innocenti, 1986; Rockland & Ojima, 2003). This architectural gradient helps explain why sensorimotor cortices favor rapid, symmetrical, and inhibitory interhemispheric exchanges that support coherent bilateral action, whereas associative cortices depend on more selective and asymmetrical communication channels that allow the emergence of flexible, lateralized functions such as language, face processing, and high-level reasoning (Gazzaniga, 2000; Thiebaut de Schotten et al., 2011).

GABAergic inhibition is known to be crucial in shaping cortical responses to stimuli across sensory modalities, including visual (Sillito, 1975; Tsumoto et al., 1979; Sillito et al., 1980; Wolf et al., 1986), auditory (Müller & Scheich, 1988; Fuzessery & Hall, 1996), and somatosensory systems (Dykes et al., 1984; Alloway & Burton, 1986; Juliano et al., 1989). Importantly, Magnetic Resonance Spectroscopy (MRS) studies show that GABA concentrations vary substantially across cortical regions, and these local differences predict individual variation in tactile perceptual performance. In particular, Puts et al. (2011) demonstrated that GABA levels measured in the somatosensory cortex corresponding to each hand correlate with roughness-discrimination thresholds in a region-specific manner.

Long-Range GABAergic Neurons (LRGNs) – revisit in inhibitory context

Given the evidence discussed in the preceding subsection, GABAergic inhibition emerges as a crucial regulator of cortical responses to sensory inputs. Across modalities, inhibitory interneurons shape receptive fields and sharpen stimulus selectivity in vision (Sillito, 1975; Tsumoto et al., 1979; Sillito et al., 1980; Wolf et al., 1986), audition (Müller & Scheich, 1988; Fuzessery & Hall, 1996), and somatosensation (Dykes et al., 1984; Alloway & Burton, 1986; Juliano et al., 1989). Importantly, the GABAergic tone varies locally. MRS studies have demonstrated that individual differences in GABA concentration predict perceptual performance in tasks such as tactile roughness discrimination, with regionally specific effects (e.g., left *vs.* right hand areas; Puts et al., 2011).

The multiscale organization of these inhibitory and excitatory influences becomes clearer when considered within the oscillatory framework of the cortical column. As illustrated in Fig. 8, vertically aligned apical dendrites in superficial and deep layers form the anatomical substrate for prominent oscillatory activity (Fig. 8a), while layer IV remains the principal locus of stimulus-driven spiking. The resulting columnar dynamics emerge through recursive interactions between pyramidal neurons and major interneuron classes, with PV+ and SST+ interneurons coordinating gamma rhythms *via* PING/ING mechanisms and NDNF+ interneurons in layer I preferentially synchronizing slow oscillations (Fig. 8b). These rhythms map onto distinct cognitive states, from perceptual integration in gamma to memory-related theta and restorative delta (Fig. 8c).

Within this scaffold, interhemispheric inputs exert highly specific modulatory effects. Callosal projections from contralateral layer II/III pyramidal neurons selectively terminate in superficial layers, aligning local gamma activity across hemispheres, whereas SST+ long-range GABAergic projections inhibit the apical dendrites of deep pyramidal neurons, imposing interhemispheric inhibitory control. Importantly, anatomical and tracing studies indicate that callosal LRGNs frequently form synapses onto PV+ interneurons, positioning them to modulate fast perisomatic inhibition and thereby influence gamma-phase precision and cross-hemispheric synchrony more strongly than would be expected from dendrite-targeting SST+ pathways alone. Neuromodulatory systems, including cholinergic, dopaminergic, serotonergic and noradrenergic pathways, further tune these oscillatory regimes according to behavioral context, thereby integrating interhemispheric signaling with global network state (Fig. 8d). Together, these interactions situate interhemispheric communication within the broader oscillatory architecture of cortical processing.

Network Rhythms and Callosal Coupling

Electrophysiological Synchronization as Fundamental Basis for Coordination

Oscillatory coherence in the beta (13–30 Hz) and gamma (> 30 Hz) bands provides a powerful mechanism for interhemispheric integration. These rhythms coordinate the timing and flow of information between hemispheres and are shaped by axonal conduction delays (Fig. 6), local interneuron networks and neuromodulatory tone (Martínez et al., 2018; Belluscio et al., 2021; Krupnik et al., 2021). As outlined in Fig. 8a–b, the laminar architecture and interneuron dynamics of the cortical column create the scaffold on which these frequency-specific interactions emerge.

In humans the beta and gamma band coherence increases when iso oriented stimuli are presented bilaterally and decreases after callosal transection (Knyaze-

va et al., 1999). Cross-hemispheric phase locking and cross-frequency coupling have been consistently observed in sensory and motor cortices during tasks that require coordinated bilateral processing (Engel et al., 2013). A more specific demonstration comes from visual studies showing that orientation-selective responses to oblique stimuli spanning both hemifields elicit robust interhemispheric synchrony (Altavini et al., 2017). Early electrophysiological studies in humans reported strong phase locking and cross frequency coupling between homotopic visual and motor cortices during tasks requiring bilateral integration (Knyazeva et al., 1999; Mima et al., 2001), while pharmacological or thermal inactivation of callosal inputs abolished orientation biases in ongoing maps, underscoring the compensatory role of callosal pathways when primary visual areas are deactivated (Jiang et al., 2002; Altavini et al., 2017).

Interhemispheric synchrony is highly state dependent. In non-human primates, anesthesia induced by ketamine or dexmedetomidine increases low frequency phase locking between hemispheres while reducing local coherence within each hemisphere, reversing the awake pattern and indicating that cross hemispheric synchrony may serve as a marker of anesthetic induced unconsciousness (Bardon et al., 2025). This shift in global network state is consistent with neuromodulatory influences illustrated in Fig. 8d. Layer specific analyses reveal that infragranular layers of the rat somatosensory cortex exhibit stronger interhemispheric correlations and low frequency coherence than supragranular layers, suggesting distinct laminar channels for bilateral communication (Baek et al., 2016). The role of the thalamus in this integration remains to be clarified and is the subject of recent studies (Szczupak et al., 2021; Szczupak et al., 2024; Bardon et al., 2025). In the motor system, paired pulse transcranial magnetic stimulation shows that the ipsilateral silent period lengthens during movement of the contralateral hand, reflecting dynamic modulation of interhemispheric inhibition during bimanual coordination (Giovanelli et al., 2009).

EEG Oscillations and Interhemispheric Interactions

EEG rhythms provide a non-invasive window into the temporal coordination of neuronal populations across the hemispheres. Distinct frequency bands – alpha (8–13 Hz), beta (13–30 Hz), and gamma (>30 Hz) – emerge from the dynamic interplay between excitation and inhibition, and in turn contribute to regulating interhemispheric communication by enabling both large-scale integration (Slater et al., 2020) and the specialization of hemispheric functions. Their laminar and circuit-level generators are summarized in Fig. 8a–c, where gamma dominates in superficial layers, beta arises from deep corticothalamic loops, and slower rhythms reflect interactions with layer I interneurons. Alpha asymmetries have been consistently associated with individual differences in affective style and motivational orientation (Davidson, 1998; Coan & Allen, 2004), beta rhythms with motor control and inhibitory coupling (Serrien, Ivry, & Swinnen, 2006; Grefkes & Fink, 2014), and gamma oscillations with rapid perceptual binding and cross-hemispheric synchrony (Engel et al., 1991; Fries, 2015). Together, these oscillatory dynamics demonstrate that interhemispheric communication depends not only on the anatomical integrity of commissural pathways but also on the temporal coordination provided by frequency-specific synchronization. By aligning the excitability cycles of neuronal populations across the two hemispheres, oscillations regulate when arriving inputs can be effectively transmitted, integrated, or suppressed, thereby enabling flexible coordination of sensory, cognitive, motor, and affective processes. Fig. 8 illustrates how these rhythms are nested within a multiscale cortical architecture that governs cross-hemispheric information flow.

Frontal Alpha Asymmetry

Frontal alpha asymmetry (FAA) reflects stable individual differences in the relative activation of the left and right frontal cortices, typically measured during resting-state or task-related EEG. Rather than representing a universal left–right pattern, FAA captures person-specific asymmetries that correlate with affective and motivational tendencies. It is typically defined as the relative difference in alpha power recorded over homologous frontal electrode sites, most often F3/F4, and occasionally F7/F8. Because alpha oscillations are inversely related to cortical excitability, lower alpha power in one hemisphere is interpreted as greater neural activity in that region.

The FAA is considered an index of the differential engagement of the prefrontal cortices in approach–withdrawal motivational processes. Davidson (1992, 1998) proposed that relatively greater left frontal activation (reduced alpha) is associated with approach-related behaviors, positive affect, and reward sensitivity, whereas greater right frontal activation (reduced alpha) is linked to withdrawal motivation, negative affect, and heightened threat sensitivity.

Importantly, the FAA shows both trait-like stability and state-like variability. Twin and longitudinal studies suggest moderate heritability, implicating the FAA as a stable biomarker of individual affective style (Coan

& Allen, 2004). At the same time, experimental manipulations of mood and stress can acutely alter the FAA, underscoring its sensitivity to situational factors (Alyan et al., 2021; Kuusinen et al., 2021), consistent with neuromodulatory influences on alpha-band regulation depicted in Fig. 8d.

Interhemispheric Coherence of Gamma

In contrast to lower-frequency rhythms such as alpha, which are thought to index relative inhibition, top–down control, and motivational bias, gamma oscillations (30–80 Hz) are thought to support local cortical synchrony and long-range integration, emerging from the interplay of excitatory and inhibitory populations (Buzsáki & Schomburg, 2015). The microcircuit basis of these rhythms, including PV+-driven PING dynamics, is schematized in Fig. 8b, while Fig. 8d highlights how callosal inputs phase-align gamma cycles across hemispheres. Gamma rhythms have been implicated in feature binding and perceptual awareness (Tallon-Baudry, 2009; Nyhus & Curran, 2010), working memory, and top–down attentional modulation (Fig. 10; Yamamoto et al., 2014; Giraud & Poeppel, 2012).

Gamma coherence across the hemispheres is primarily supported by the corpus callosum. In animals, callosal transection reduces interhemispheric gamma synchrony, particularly in the visual cortices, impairing cross-hemifield feature binding (Engel et al., 1991). In humans, EEG shows increased gamma coherence between the parieto-occipital regions during cross-hemifield integration tasks, with performance depending on the strength of this synchronization (Fig. 14; Bland et al., 2020).

Asymmetries also emerge in gamma coherence. Evidence suggests the right-hemisphere dominance for gamma synchronization during global perceptual tasks, reflecting a holistic processing bias (Doesburg et al., 2008; Knyazev, 2007). Conversely, the left hemisphere gamma is recruited for fine-grained, linguistic, or analytic processing (Pulvermüller et al., 2003; Weiss & Mueller, 2012). Speech perception studies have revealed left-dominant gamma oscillations during phonemic decoding, while right-dominant theta/low-frequency rhythms track prosody, which is consistent with the asymmetric sampling in time (AST) model (Poeppel, 2003; Giraud & Poeppel, 2012; Hyafil et al., 2015). Beyond language, right hippocampal and parietal gamma coherence support spatial navigation and visuospatial attention (Tamura et al., 2017; Guan et al., 2022), whereas left-hemispheric gamma coupling is associated with verbal working memory and linguistic processing (Kambara et al., 2018; Inguscio et al., 2022).

Asymmetries in Sensory and Cognitive Domains by Beta Rhythms

Beta-band oscillations (~13–30 Hz) occupy an intermediate role between alpha and gamma oscillations, mediating long-range coordination and inhibitory control. As suggested by Fig. 8b, beta rhythms arise primarily from deep-layer pyramidal neurons and their corticothalamic loops, making them well suited for top-down regulation. Beta rhythms are prominent in sensorimotor networks, basal ganglia circuits, and frontoparietal systems, where they stabilize ongoing states and support proactive inhibition (Engel & Fries, 2010; Schmidt et al., 2013; Barone & Rossiter, 2021). Unlike gamma, which facilitates rapid feedforward processing, beta is more closely associated with top–down regulation and set maintenance (Buschman & Miller, 2007; Kilavik et al., 2013; Spitzer & Haegens, 2017).

Studies on motor functions have highlighted interhemispheric specialization. Beta desynchronization over the contralateral motor cortex precedes and accompanies voluntary motor execution, balancing the excitability between hemispheres to prevent mirror movements and enable fine unilateral control (Hinder et al., 2012; Stegemöller et al., 2009). In right-handed individuals, left motor beta desynchronization dominates during dominant-hand movement, whereas the right hemisphere contributes more strongly to bimanual coordination and posture (Serrien et al., 2003; Serrien & Brown, 2002). The laminar segregation of beta generators in Fig. 8a–b provides a structural explanation for why these asymmetries are more pronounced in frontal–motor circuits. Beta rhythms are generated predominantly in deep corticothalamic and cortico-basal-ganglia loops (Fig. 8a–b). Circuits known to act as major beta generators (Brittain & Brown, 2014; West et al., 2018) exhibit a strong lateralization within frontal–motor systems (Neumann et al., 2023). Additionally any modulation of these deep-layer generators disproportionately amplifies left–right asymmetries in these circuits (Brittain & Brown, 2014).

Although hemispheric asymmetries in motor beta rhythms are most prominently described in humans, animal studies offer a more nuanced and limited view. In rodents, neural recordings have shown a contralateral bias in motor cortical neurons: for example, in rat M1 (but not always in M2) there is a stronger representation of the contralateral forelimb in pyramidal tract (PT) neurons, indicating some lateralized motor encoding (Hira et al., 2018). However, direct evidence linking this laterality to beta-band (~13–30 Hz) desynchronization during movement in rodents remains scarce.

In non-human primates, local field potential (LFP) studies have documented beta-band (~15–30 Hz) sup-

pression in M1 and premotor areas during contralateral limb movements. For example, during reach-to-grasp tasks in macaque monkeys, LFP power in the beta band decreases more strongly in the hemisphere contralateral to the moving hand (Menzer et al., 2007; Falaki 2024). Moreover, in supplementary motor areas (SMA/pre-SMA), beta suppression has been associated with updating of action sequences (Hosaka et al., 2016), suggesting a role for beta in flexible planning rather than purely execution.

At the same time, the study concerning rodents basal ganglia has shown that transient beta oscillations emerge in task-performing animals within cortico-basal ganglia loops, but these are not straightforwardly lateralized in the same way as in humans; rather, they reflect complex interactions of sensory, motor, and network dynamics (Mirzaei et al., 2017). Thus, while animal data do support some contralateral organization of motor representations and beta dynamics, the pattern is weaker, more variable across individuals and species, and does not strongly mirror the robust hemispheric beta asymmetries seen in humans.

Beta interhemispheric coherence depends critically on the corpus callosum. TMS and EEG/magnetoencephalography (MEG) studies have demonstrated that callosal integrity shapes interhemispheric beta coupling, with coherence reduced following callosal lesions and in conditions such as multiple sclerosis, where white matter damage disrupts cross-hemispheric synchrony (Meyer et al., 1995; Leocani et al., 2000).

Thus, while gamma oscillations can be regarded as a special case of interhemispheric integration, particularly critical for perceptual binding and cross-hemifield coherence (see Engel et al., 1991; Doesburg et al., 2008), they should not be seen as the sole mechanism of hemispheric coordination. Slower rhythms, especially beta and partly alpha, support complementary functions including movement preparation, attentional gating, and the stabilization of ongoing sensorimotor states (see Engel & Fries, 2010; Serrien & Brown, 2002; Stegemöller et al., 2009; Corbetta & Shulman, 2011). However, the amplitude, frequency, and lateralization of these rhythms are not determined by local synaptic dynamics alone but are continuously shaped by ascending neuromodulatory systems originating from subcortical nuclei and brainstem structures providing the next regulatory level of interhemispheric balance.

Experimental evidence for thalamic receptor asymmetry

Functional and molecular studies indicate substantial heterogeneity in inhibitory tone and receptor composition within the thalamic reticular complex, where the interplay between fast $GABA_A$ and slow $GABA_B$-mediated inhibition, together with GluN2B-dependent thalamic excitation, shapes distinct oscillatory resonance modes (Crabtree, 2018; Halassa & Kastner, 2017; Zhang et al., 2009). Although hemispheric asymmetries have not been demonstrated directly, these mechanisms provide a plausible substrate for differential temporal dynamics between the hemispheres.

These lateralized thalamic dynamics underpin the differential temporal resolution and coupling strength of cortical networks across hemispheres. Although direct evidence for left–right molecular asymmetry within the TRN is currently lacking, recent *in vivo* electrophysiological, optogenetic, and receptor-mapping studies have reveal pronounced microcircuit heterogeneity and state-dependent functional diversity within TRN-thalamo-cortical loops (Crabtree et al., 2013; Hou, Smith, & Zhang, 2016). These findings provide a mechanistic foundation for hypothesizing hemispheric specialization in thalamic gating.

Whole-cell patch-clamp recordings in anesthetized and awake mice have demonstrated that TRN neurons in the left hemisphere receive stronger and more temporally precise $GABA_A$-mediated inhibition, generating sharply timed inhibitory postsynaptic currents (10–30 ms) and supporting beta-gamma frequency resonance (Ulrich & Huguenard, 1997). Optogenetic stimulation of corticothalamic axons from layer VI pyramidal neurons produced larger, fast-spiking inhibitory responses in the left TRN compared to the right, indicating higher perisomatic $GABA_A$ receptor density and efficiency in local feedback gating.

Conversely, pharmacogenetic silencing or selective antagonism of $GABA_B$ receptors in the right TRN abolished rhythmic alpha-theta bursting and disrupted slow-wave synchrony across thalamic relay nuclei (Huguenard & Prince, 1994; Crunelli & Leresche, 2002). These effects were absent after similar manipulations on the left side of the brain, confirming a right-biased contribution of $GABA_B$-dependent tonic inhibition to thalamocortical oscillations. Autoradiographic receptor mapping (Hansen et al., 2022) corroborated these physiological findings by revealing higher $GABA_B$ binding density in the right intralaminar and reticular nuclei, paralleled by leftward enrichment of NMDA GluN2B subunits in mediodorsal and pulvinar regions. Two-photon calcium imaging and immunohistochemistry in mice (Labache et al., 2024) further have demonstrated that GluN2B-rich synapses in the left thalamus and corticothalamic neurons exhibit prolonged decay kinetics and enhanced plasticity, extending excitatory drive and temporal precision.

Recent findings indicate that left thalamic beta-gamma activity is phase-locked to task-related cortical oscillations, suggesting a coherent interplay between thalamic and cortical regions critical for sensory integration and attention. In contrast, right alpha-theta coherence has been observed to correlate with vigilance, multisensory integration, and the overall arousal state of the mouse, thereby indicating hemispheric specialization in attentional mechanisms. High-field fMRI improves image resolution and enhances sensitivity, which is crucial for tracking oscillatory activities in rodent models (Zhou et al., 2011; Barth & Poser, 2011). The ability to discern subtle differences in oscillatory patterns correlates closely with perceptual tasks, highlighting the importance of the thalamus in synchronizing cortical activity during cognitive demands. For example, wide-field Ca^{2+} imaging in mice has shown that visually demanding attention tasks evoke distinct, region-specific oscillatory activity patterns that track the shifting allocation of attention across cortical networks (McDermott et al., 2017). This reinforces the notion that coordination between thalamic beta–gamma activity and cortical oscillations is essential for effective sensory processing and discrimination tasks.

Moreover, the differential roles of alpha and theta oscillations emphasize the complexity of neural mechanisms associated with arousal and attentional resources. Alpha oscillations, particularly in the context of the right hemisphere, have been implicated in the modulation of sensory input and attentional control (Knakker et al., 2015). Elevated theta oscillations have been linked to heightened states of vigilance and multisensory integration, further suggesting their role in maintaining focus and readiness for perceptual tasks (McDermott et al., 2018). The functional dissociation of cortical theta oscillations within different regions during attention suggests that these oscillations do not merely serve a monolithic function but adapt dynamically to task demands (Han et al., 2019).

Convergent *in vivo* evidence demonstrates that left TRN microcircuits dominated by $GABA_A$–GluN2B activity sustain fast, phasic beta–gamma coupling, while right TRN networks enriched in $GABA_B$ receptors maintain slow, tonic alpha–theta synchronization. These complementary regimes establish the physiological basis for hemispheric differences in temporal resolution, attention, and cognitive specialization.

Coherence among oscillatory activity across brain regions is vital for cognitive efficiency. The synchronization of different frequency bands, such as the alpha-theta coherence observed in the context of arousal, indicates how the brain flexibly shifts between states of rest and activity, thereby influencing behavioral outcomes (Eschenko et al., 2011; Nácher et al., 2013). In summary, the left thalamic beta-gamma activity's phase-locking alongside the right alpha-theta coherence elucidates the intricate neural network that governs cognitive functions such as attentional focus and sensory integration (Fig. 15).

Neuromodulatory Systems and Lateralized Interhemispheric Regulation

Electrophysiological signatures, such as alpha, beta, and gamma oscillations, provide mechanistic insights into how interhemispheric interactions are dynamically coordinated across large-scale cortical and subcortical networks. These rhythms emerge from the interplay of excitation and inhibition at the circuit level and constitute the immediate substrate for hemispheric specialization in the sensory, motor, and cognitive domains. However, these rhythmic stability and context-dependent modulation cannot be fully explained by fast synaptic transmission alone. A more detailed picture of these asymmetries emerges from the nonsymmetric distribution of the neuromodulatory activity (Fig. 11). PET-derived quantification across 1,238 individuals reveals that each neuromodulatory system (dopaminergic, serotonergic, cholinergic, and noradrenergic) exhibits distinct left–right cortical gradients (Fig. 11a), with several receptor families showing robust lateralization across homotopic regions of the AICHA atlas (Labache et al., 2025). White-matter projections and cortical distributions further indicate that these systems are not uniformly bilateral but instead form spatially segregated pathways influencing hemispheric excitability (Fig. 11b–c, Hansen et al., 2022). Together, these structural asymmetries provide the molecular substrate through which neuromodulatory nuclei modulate interhemispheric coordination, enabling the right and left hemispheres to adopt specific functional roles depending on behavioral context.

Neuromodulatory systems, including dopaminergic, serotonergic, cholinergic, and noradrenergic systems, provide a critical regulatory layer that adjusts neuronal excitability, gates synaptic plasticity, and shapes oscillatory synchronization. By modulating the gain and flexibility of interhemispheric coupling, these systems bias hemispheric dominance in a task-dependent manner. Importantly, converging evidence indicates that influences of neuromodulatory systems are lateralized, rather than symmetrical. For example, dopaminergic signaling is more strongly left-biased for approach-related behaviors and verbal working memory, whereas right-hemisphere dopamine supports spa-

tial orientation and avoidance learning (Tomer et al., 2008; Arsalidou et al., 2018). Serotonin and acetylcholine also exert asymmetric effects on hippocampal and cortical circuits, whereas noradrenaline shows robust right-hemisphere dominance in attentional control (Sara, 2009; Corbetta & Shulman, 2011).

Crucially, these neuromodulators arise from highly conserved brainstem and basal forebrain nuclei, including the locus coeruleus (noradrenaline), raphe nuclei (serotonin), ventral tegmental area, substantia nigra (dopamine), and nucleus basalis of Meynert (acetylcholine). Through diffuse ascending projections, these nuclei provide continuous regulatory influence on cortical excitability and interhemispheric balance. Their activity links arousal, vigilance, and motivational states to large-scale oscillatory dynamics, ensuring that hemispheric specialization is not static but flexibly tuned to environmental demands.

In this way, neuromodulators act as a bridge between molecular asymmetries and large-scale oscilla-

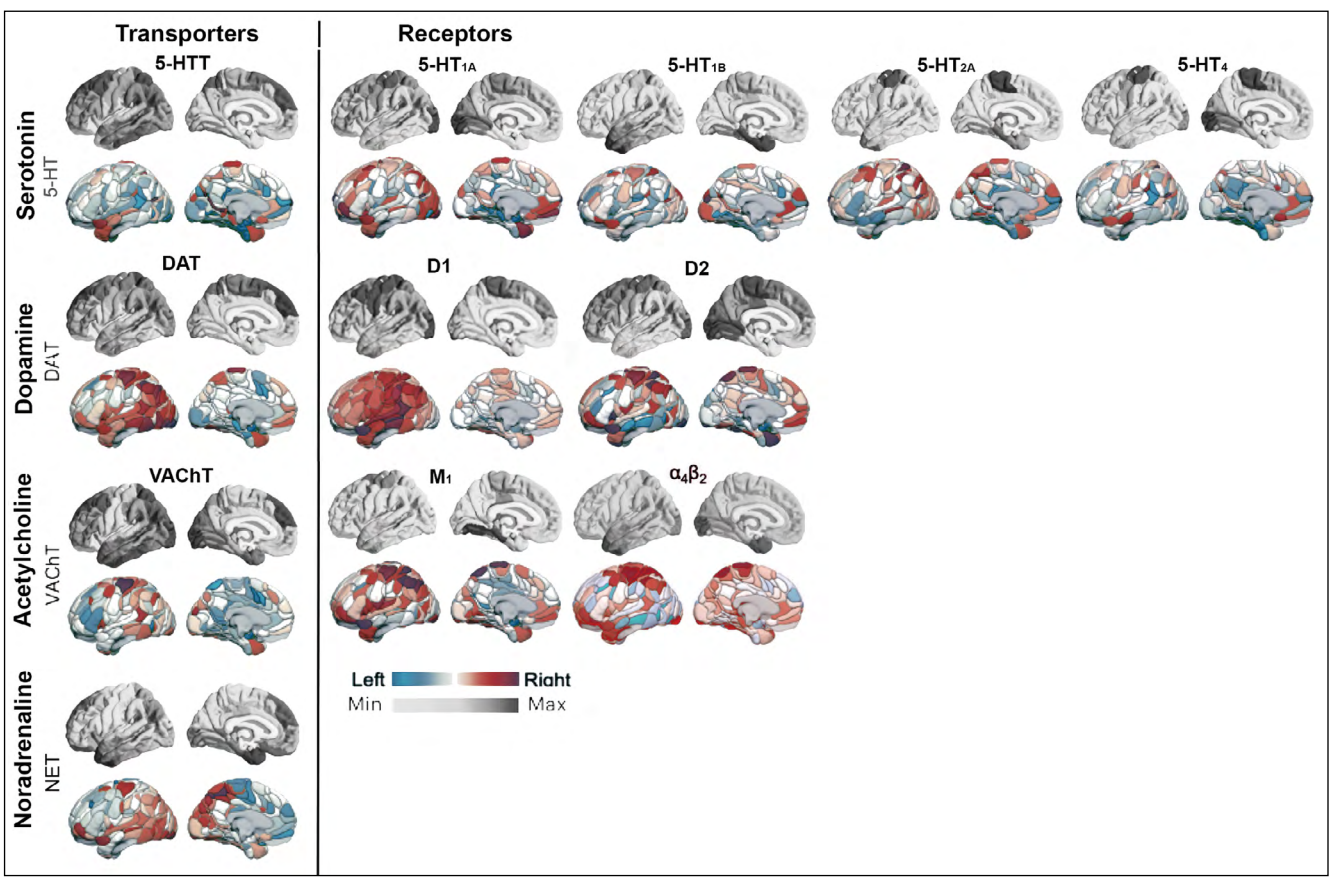


Fig. 11. Asymmetry of densities of neuromodulator transporters and receptors. These results establish the molecular substrate of hemispheric specialization by demonstrating systematic asymmetries in the cortical distribution of neuromodulatory receptors and transporters. Across acetylcholine, dopamine, serotonin, and noradrenaline systems, positron emission tomography (PET)-derived density maps reveal region-specific left–right biases in neuromodulator availability, indicating that hemispheric lateralization is embedded at the receptor and transporter level. These asymmetric molecular landscapes provide the biochemical framework upon which long-range neuromodulatory projections (Fig. 12) and regionally dominant transmitter systems (Fig. 13) are organized, suggesting that neuromodulation contributes directly to stable patterns of hemispheric specialization. a. PET normalized density maps illustrate the distribution of four neuromodulatory systems (ACh, DA, 5-HT, NA) in the human cortex. Data were obtained from 1,238 healthy participants in the BIL&GIN database (Mazoyer et al., 2016), all identified as left-hemisphere dominant for language (Labache et al., 2023). For each receptor/transporter, the top row (grayscale) shows mean density in the left hemisphere, whereas the bottom row depicts asymmetry maps, calculated by subtracting left from right hemisphere values across 163 homotopic cortical regions of the AICHA atlas (Joliot et al., 2015; Hansen et al., 2022). Asymmetries are projected onto the left hemisphere surface using a color scale (blue=leftward, red=rightward). Surface renderings in MNI space were generated with Surf Ice software (https://www.nitrc.org/projects/surfice/). b. Representative map of neurotransmitter system white matter projections, color-coded according to the highest value at the voxel level. c. Map of neuromodulatory system locations (receptor or transporter densities), also color-coded according to the highest value at the voxel level. The cortical (b1) and basal ganglia (b2) surfaces are represented. I. left , II. right sided views; III. superior, and IV. inferior views. Cross-sections: V. horizontal, VI. striatal, VII. coronal; Projection maps for each receptor and transporter are available at: https://identifiers.org/neurovault.collection: 15237. *Hansen et al., 2022 joined with Labache et al., 2025; Springer Nature, with permissions, under CC_BY.*

tory coordination; they not only tune the amplitude and frequency of rhythmic activity but also enforce hemispheric specialization across domains. This neuromodulatory layer critically shapes both vulnerability and recovery in clinical conditions such as stroke, depression, and attention deficit disorders (Cramer, 2015; Grimm et al., 2009; Siegel et al., 2016).

Dopamine and beta oscillations

Dopamine provides one of the clearest examples of neuromodulatory asymmetry that shapes hemispheric specialization. In rodents, dopaminergic signaling in the left prelimbic cortex is essential for memory consolidation in novel object recognition tasks (Papp et al., 2019; Winters et al., 2008). Translational evidence suggests that this leftward bias extends to humans, where the left prefrontal cortex is preferentially engaged in declarative and verbal memory processes (Nyberg et al., 1996; Fletcher & Henson, 2001). Dopaminergic modulation of novelty detection and memory consolidation has also been demonstrated in humans, where dopaminergic activation enhances hippocampal–prefrontal coupling during episodic learning (Bunzeck et al., 2007). Moreover, experimental evidence from rats has shown that dopamine increases the flexibility of prefrontal–hippocampal interactions during memory updating and consolidation (Cybulska-Kłosowicz et al., 2017).

Cybulska-Kłosowicz et al. (2017) demonstrated that dopaminergic tone supports the flexible integration of new information into existing memory networks, ensuring that learning remains dynamic rather than rigidly consolidated. This dual role, stabilization and flexibility, provides a mechanistic basis for the asymmetric dopaminergic contribution to hemispheric specialization in memory.

In contrast, right-hemisphere dopamine is more strongly recruited during spatial orienting, avoidance learning, and vigilance-related tasks (Tomer et al., 2008; Arsalidou et al., 2018). This dual pattern illustrates how dopamine supports not only left-lateralized verbal/episodic memory, but also right-lateralized spatial and attentional control, providing a dynamic modulatory mechanism for balancing the hemispheric contributions.

Disruption of dopaminergic asymmetry has been implicated in several disorders with lateralized clinical presentations. In Parkinson's disease, asymmetric degeneration of striatal dopamine pathways correlates with lateralized motor and non-motor symptoms (Hansen et al., 2025), whereas in hemispatial neglect, the relative depletion of dopamine in the nigrostriatal pathway may contribute to directional hypokinesia and attentional deficits (Heilman et al., 2003).

Dopamine plays a key role in modulating beta-band coherence across the hemispheres, particularly in cortico–basal ganglia–thalamic loops. PET studies have revealed hemispheric asymmetries in D2/D3 receptor availability within the striatum and prefrontal cortex, which predict individual differences in lateralized attention and approach–avoidance biases (Tomer et al., 2008; Zhao et al., 2025).

Beta desynchronization over the contralateral motor cortex precedes and accompanies movement execution, whereas excessive beta synchrony is observed in patients with Parkinson's disease, reflecting impaired dopaminergic transmission (Doherty et al., 2025).

Importantly, asymmetrical dopamine receptor distribution and binding across hemispheres predict orientational bias and lateralized attention in healthy individuals (Tomer et al., 2008). This lateralization links dopaminergic tone with hemispheric specialization of motor and cognitive functions.

Serotonin: Theta–Gamma Gating and Mood Lateralization

Serotonergic signaling also shows hemispheric asymmetry. Postmortem studies in humans (Arató et al., 1991) and *in vivo* PET investigations in healthy adult humans (Kranz et al., 2014) have reported greater serotonin metabolite levels and higher serotonin-transporter binding in the right frontal and cingulate cortices. Experiments on rodents suggest sex- and stimulus-dependent lateralization of serotonin release in the hippocampus, where serotonin modulates $GABA_B$-mediated inhibition in CA1 interneurons, thereby influencing working memory and mnemonic gating (Fraser & MacVicar, 1992; Freund, 1992).

Ascending serotonergic projections from the raphe nuclei regulate hippocampal and prefrontal oscillatory dynamics. By modulating local GABAergic interneurons, serotonin enhances theta and gamma activity (Puig & Gulledge, 2011) and contributes to the interhemispheric coordination of affective and memory processes *via* theta–gamma coupling. Lateralized serotonergic innervation and receptor binding have been reported in both cortical and limbic regions, shaping hemispheric dominance in mood regulation and stress reactivity (Puig & Gulledge, 2011).

Human PET studies have further revealed an asymmetric cortical distribution of 5-HT1A receptors, with higher binding in the right frontal cortex, consistent with lateralized serotonergic contributions to mood and cognition (Kranz et al., 2014). Converging event-re-

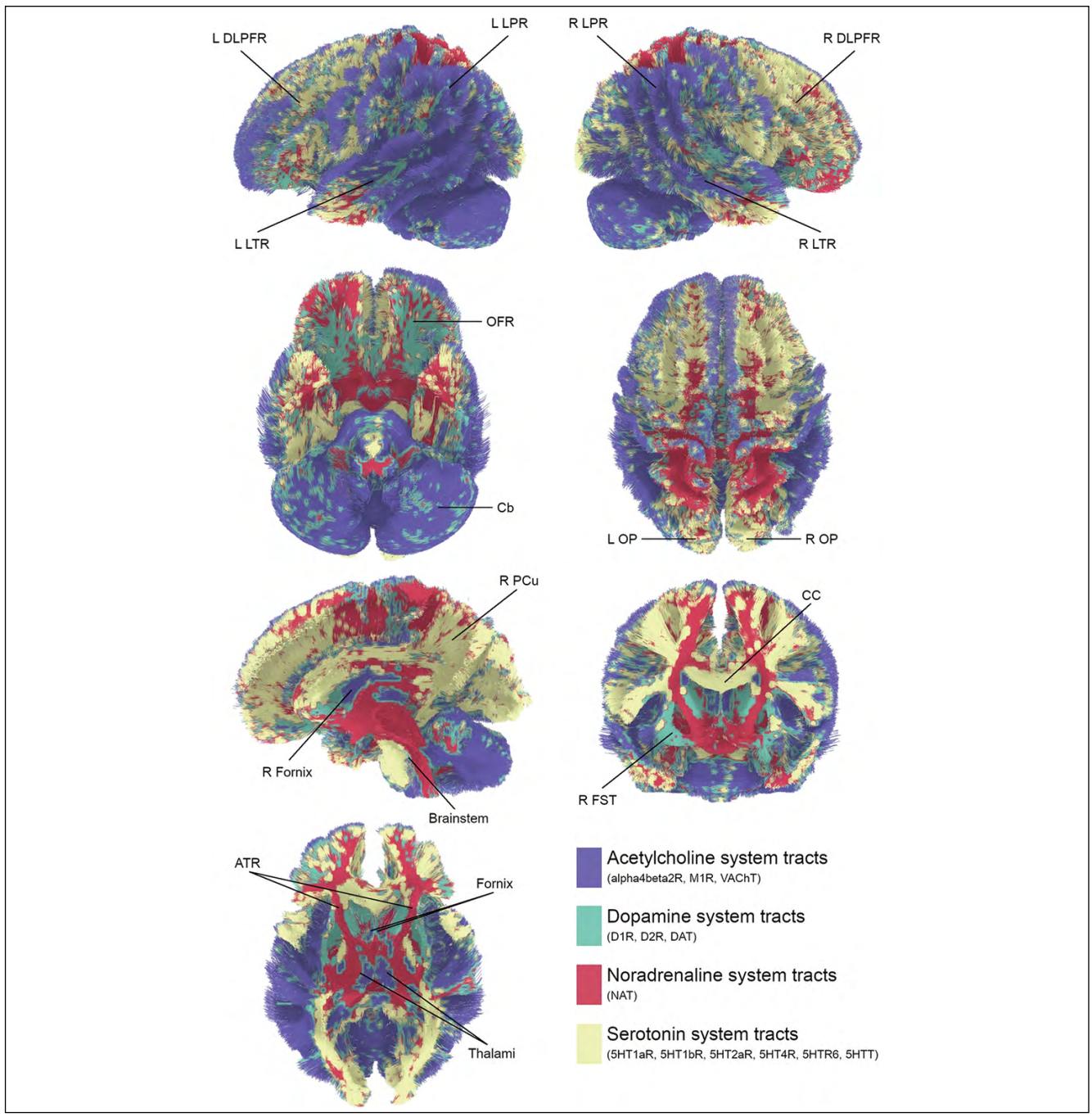


Fig. 12. Neurotransmitters' white matter mapping in cerebrum and cerebellum. The map was colored according to the neurotransmitter system of the map with the highest value at a voxel level. Building on the asymmetric receptor landscapes shown in Fig. 11. Fig. 12 maps the large-scale white matter trajectories through which neuromodulatory systems exert their influence across the cerebrum and cerebellum. The projection patterns reveal that neuromodulatory asymmetries are not confined to cortical surfaces but are propagated *via* distinct long-range fiber systems linking cortex, basal ganglia, thalamus, brainstem, and cerebellum. These pathways provide the structural routes through which hemispherically biased neuromodulatory signals can coordinate distributed networks, setting the stage for region-specific transmitter dominance illustrated in Fig. 13. Therefore the neuromodulatory asymmetry is a network-level property, supported by asymmetric long-range connectivity rather than isolated cortical effects. Abbreviations of regions/structures: ATR–anterior thalamic radiations, Cb–cerebellum, CC–corpus callosum, DLPFR–dorsolateral prefrontal region, FST–frontostriatal tracts, L–left, LPR–lateral parietal region, LTR–lateral temporal region, OFR–orbitofrontal region, OP–occipital pole, PCu–precuneus, R–right. Abbreviations of receptors/transporters: 5HT1aR–serotonin receptor 1a, 5HT1bR–serotonin receptor 1b, 5HT2aR–serotonin receptor 2a, 5HT4R–serotonin receptor 4, 5HT6R–serotonin receptor 6, 5HTT–serotonin transporter; alpha4beta2R–acetylcholine receptor alpha4beta2, D1R–dopamine receptor 1, D2R–dopamine receptor 2, DAT–dopamine transporter, M1R–muscarinic 1 receptor, NAT–noradrenaline transporter, VAChT–acetylcholine vesicular transporter. The projection maps for each receptor and transporter are available at https://identifiers.org/neurovault.collection: 15237. *Reproduced from Alves et al. (2025), Nat Commun, 2025, under CC BY-NC-ND 4.0 (no changes made).*

lated potential (ERP) evidence shows that pharmacological modulation of the serotonin system alters lateralized evoked potentials, such as the P300* component. For instance, Selective Serotonin Reuptake Inhibitor (SSRI) treatment increases the P300 amplitude more strongly over the left frontal cortex, reflecting enhanced recruitment of attentional and evaluative processes (Bruder et al., 2001). In contrast, 5-HT1A agonists, such as buspirone, tend to reduce P300 amplitude preferentially in the right hemisphere, consistent with serotonergic involvement in withdrawal- and avoidance-related processes mediated by the right prefrontal cortex (Hansenne, 1999). Pharmacological activation of 5-HT2A receptors with psychedelics, such as psilocybin, alters visual evoked potentials and gamma oscillations, asymmetrically enhancing right-hemisphere responses to negative emotional stimuli (Kometer et al., 2013). Mechanistically, these effects likely reflect serotonergic modulation of prefrontal and hippocampal GABAergic interneurons, altering the excitation–inhibition balance and theta–gamma coupling that synchronizes the generation of P300 (P300 is one of the most thoroughly characterized event-related potentials in EEG, a positive deflection occurring approximately 250–500 ms after a task-relevant stimulus. It is most commonly elicited in the oddball paradigm, in which an infrequent, deviant stimulus is embedded within a stream of frequent standard stimuli).

Together, these findings provide receptor-specific evidence that serotonergic systems shape hemispheric asymmetry not only at the molecular and circuit levels but also in neurophysiological signatures and clinical responsiveness, linking serotonin to both cognitive specialization and vulnerability to affective disorders.

Acetylcholine: Alpha Suppression, Theta Timing, PKC-Dependent Plasticity

Cholinergic projections from the basal forebrain and brainstem nuclei innervate the widespread cortical and hippocampal targets. While these projections are broadly bilateral, functional studies suggest lateralized cholinergic effects, particularly in attention, learning, and memory.

In rodents, muscarinic acetylcholine receptors (mAChRs), especially the M1 subtype, are more densely expressed in the right cerebral cortex (Pediconi et al., 1993). In humans, acetylcholine modulates callosal signaling by enhancing apical dendritic excitability in layers II/III and V, particularly *via* the mAChR–PKC pathway. Hemispheric differences in plasticity-related gene expression (e.g., pCREB, Arc) during emotionally valenced learning have been confirmed (Young & Williams, 2013). Bias left-hemisphere cholinergic efficacy during verbal memory encoding has been observed, whereas right-hemisphere cholinergic activity is preferentially engaged during spatial navigation (Robbins, 2000; Newman et al., 2012). The medial temporal lobe, particularly the hippocampus and surrounding structures, are crucial for the encoding and consolidation of verbal information (Graham et al., 2010). The cholinergic system enhances synaptic plasticity, which is fundamental for these processes. Studies have shown that cholinergic manipulation, *via* the administration of acetylcholinesterase inhibitors, improves performance in verbal memory tasks, indicating a robust role of left-hemisphere cholinergic activity during verbal memory encoding (Sarter et al., 2006). Conversely, spatial navigation relies significantly on right-hemisphere activity, particularly involving the hippocampus and parietal lobes. The cholinergic system here is engaged differently than in verbal tasks, facilitating the encoding of spatial memories and navigation strategies. The right hemisphere's spatial cognition mechanisms have been linked to the enhancement of memory retrieval processes, wherein cholinergic activity promotes attention to spatial cues and navigation-related behaviors (Deiana et al., 2011).

Acetylcholine is also a key regulator of attentional control and interhemispheric balance of cortical excitability. Phasic cholinergic release suppresses alpha oscillations, thereby facilitating communication between the hemispheres during tasks that require bilateral attentional resources (Makeig et al., 2004). Experimental work in both animals and humans has demonstrated that cortical cholinergic activity enhances cue detection and attentional resource allocation, particularly in prefrontal networks (Demeter et al., 1985).

Lesion and pharmacological studies further suggest that cholinergic asymmetries may bias attentional orienting, with evidence pointing towards a stronger contribution of the right hemisphere to spatial vigilance and reorienting. This lateralization positions acetylcholine as a key neuromodulator linking alpha suppression, interhemispheric coordination, and hemispheric specialization of attention

Noradrenaline: Gain Control and Right-Biased Vigilance Networks

Noradrenergic modulation exhibits a robust hemispheric bias, especially within attentional and emotional circuits. In the amygdala, norepinephrine release supports lateralized affective processing: the right basolateral amygdala (BLA) responds more strongly to negatively arousing stimuli, whereas the left BLA is

more responsive to positive affect (LaLumiere & McGaugh, 2005). Functional imaging further revealed right-lateralized catecholamine release in striatal and frontoparietal attention networks, consistent with the dominance of right-hemisphere vigilance systems (Martin-Soelch et al., 2001; Sara, 2009).

Projections from the locus coeruleus (LC) provide a global gain control mechanism, enhancing the signal-to-noise ratio and promoting long-range network synchrony. Noradrenaline facilitates beta and gamma band coherence during attentional engagement and learning, thereby coordinating large-scale cortical dynamics (Aston-Jones & Cohen, 2005). Importantly, this influence is asymmetric: lesion and imaging studies have demonstrated the preferential recruitment of right-hemisphere noradrenergic pathways during spatial vigilance and reorientation (Corbetta & Shulman, 2002).

Together, these findings position noradrenaline as a key neuromodulator of hemispheric specialization, supporting a right-biased attentional system that integrates cross-hemifield information and ensures adaptive behavioral responses under uncertain conditions.

Neuromodulatory Modulation of Oscillatory Asymmetries

Taken together, neuromodulatory systems introduce a chemical layer of regulation onto the electrophysiological scaffold of interhemispheric interactions. Dopamine is most often linked to beta-band synchronization in cortico–basal ganglia–thalamic loops, serotonin to theta–gamma coupling in hippocampal and prefrontal circuits, acetylcholine to alpha suppression and theta timing during attentional control, and noradrenaline to gain control mechanisms that enhance long-range beta/gamma coherence (Tomer et al., 2008; Puig & Gulledge, 2011; Makeig et al., 2004; Aston-Jones & Cohen, 2005).

However, these associations should not be regarded as exclusive. Each neuromodulator can modulate multiple oscillatory regimes, and their "preferred" frequency associations may reflect the focus of existing research rather than strict biological boundaries of the oscillatory regime. For instance, dopamine also influences gamma in prefrontal working memory tasks (Winterer & Weinberger, 2004), acetylcholine contributes to gamma synchronization in sensory cortices (Rodriguez et al., 2004), and serotonin modulates alpha activity in frontal asymmetry paradigms (Bruder et al., 2001). The apparent specificity may therefore be, at least partly, an artifact of how neuromodulatory effects have been investigated and synthesized in the literature (Avery & Krichmar, 2017; van den Brink et al., 2019). Yet, despite these methodological constraints, converging electrophysiological and imaging evidence indicates that neuromodulators bias, but do not rigidly determine the oscillatory channels through which interhemispheric communication unfolds.

At rest, hemispheres rely on similar frequency bands, but their synchronization patterns diverge: the left thalamus shows tighter phase-locking to cortical gamma bursts, whereas the right preferentially engages in low-frequency coherence with associative cortices (Fiebelkorn et al., 2019; Browne et al., 2025). Functional connectivity analyses further suggest that this microtemporal asymmetry arises from unequal GABAergic input to thalamic reticular neurons and asymmetric expression of NMDA-GluN2B and $GABA_B$ receptors, endowing each hemisphere with distinct oscillatory resonance profiles (Labache et al., 2025; Hansen et al., 2022).

Towards integration

While corpus callosum fibers and local excitatory–inhibitory dynamics provide the structural and electrophysiological foundation for interhemispheric communication, they mediate functional coherence primarily via oscillatory synchronization through callosal cortical tracts (Engel et al., 2013). These pathways balance facilitation with feed-forward inhibition, ensuring dynamic stability and flexible information exchange between hemispheres (Aboitiz & Montiel, 2003).

Neuromodulatory systems further determine the efficiency, context dependence, directionality, and lateralization of these interactions, selectively promoting attentional or computational modes within a given hemisphere (Gedankien et al., 2023a; Innocenti et al., 2022). Neuromodulators influence the transfer and integration of information across hemispheres by regulating oscillatory synchronization, synaptic plasticity, and attentional gating.

Specifically, acetylcholine modulates neuronal excitability, synaptic plasticity, and coordinated firing (Hasselmo & McGaughy 2004, Hasselmo & Sarter, 2011; Picciotto et al., 2012). Acetylcholine induces local gamma oscillations by recruiting GABAergic interneurons to generate rhythmic inhibition of pyramidal cells (Buhl et al., 1998; Fisahn et al., 1998). These gamma rhythms stabilized by the cholinergic system propagate through callosal pyramidal axons, thereby reinforcing interhemispheric coherence during tasks that require bilateral integration. Acetylcholine also supports the temporal coordination of theta oscillations during memory encoding (Gedankien et al., 2023b), drives re-

gion-specific beta and gamma rhythms to impose directional coupling (Buhl et al., 1998; Fisahn et al., 1998), and gates resting-state connectivity by suppressing Default Mode Network (DMN) activity to facilitate attention-driven states, and attention seems to diversify the hemispheres cognitive states. These interactions are exemplified in Fig. 14, where cholinergic modulation has been shown to reorganize large-scale network dynamics by suppressing default-mode connectivity, shaping theta–gamma coupling, and producing hemispheric differences driven by lateralized receptor distributions. Together, these effects demonstrate how neuromodulators impose flexible, state-dependent asymmetries on interhemispheric communication, bridging molecular gradients with network-level coordination (Sanda et al., 2024).

Bartolomeo (2019) reviewed how attentional systems are strongly lateralized, right hemisphere dom-

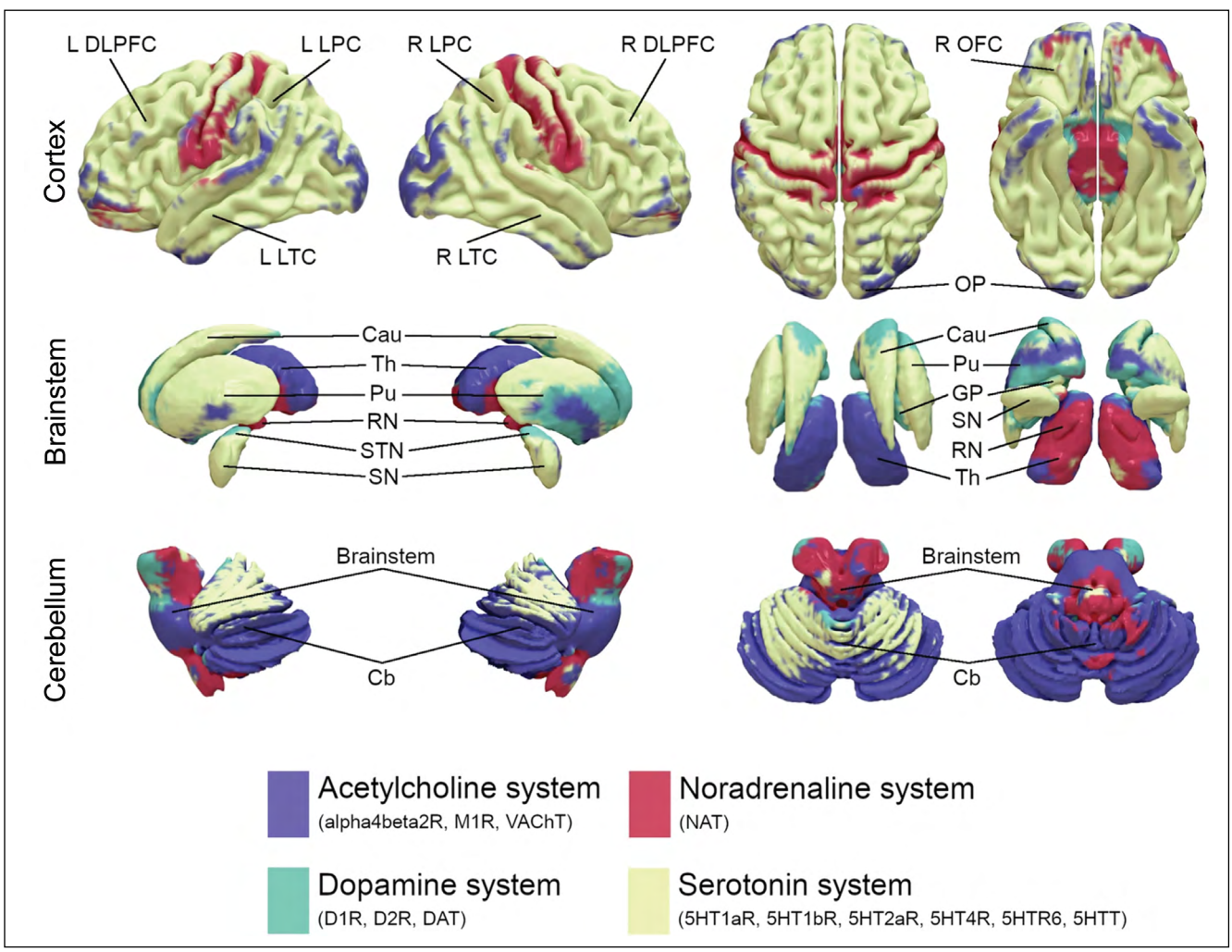
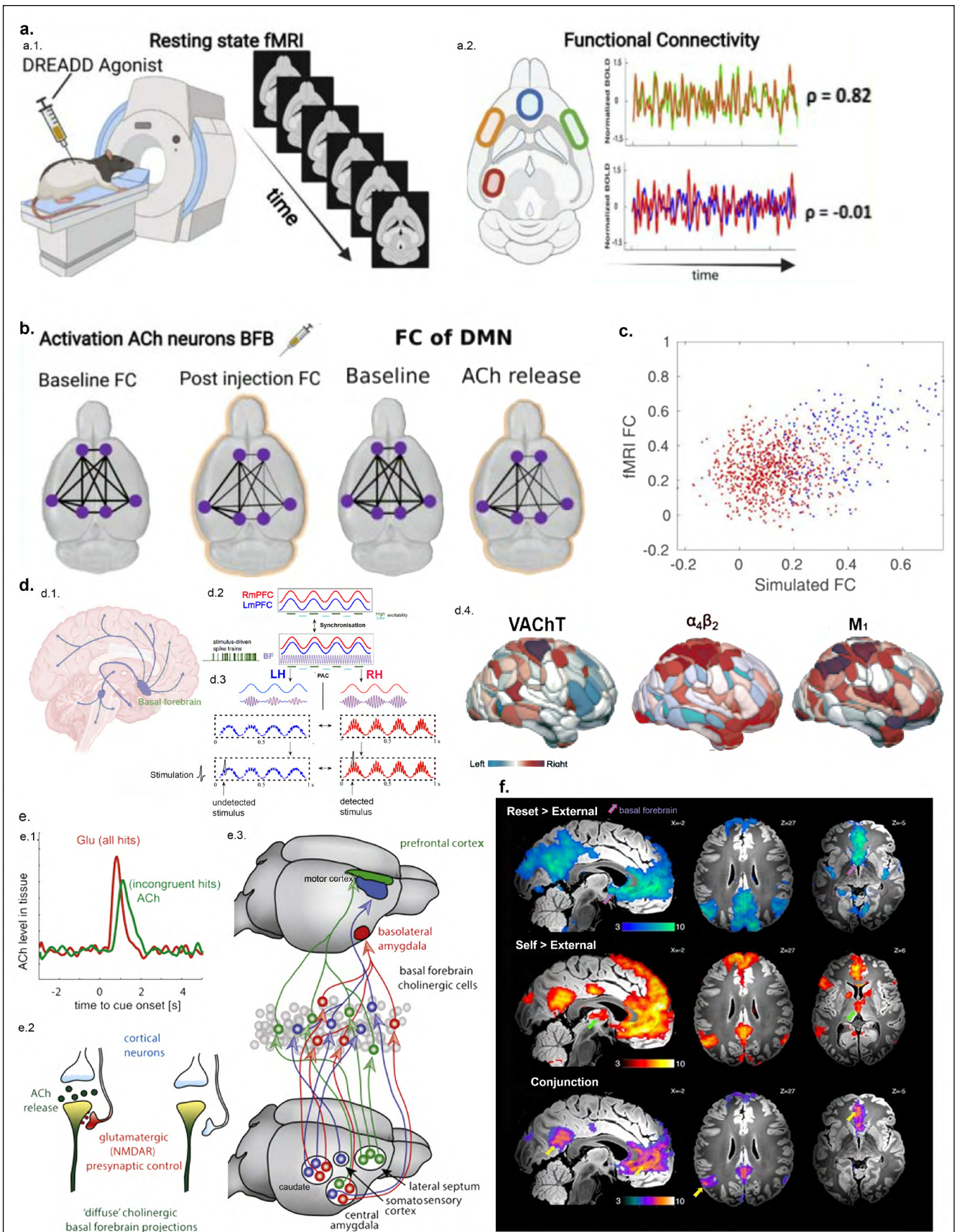


Fig. 13. The dominating neurotransmitter in the region. Integrating receptor asymmetries (Fig. 11) with projection architecture (Fig. 12), this figure identifies the neuromodulatory system that dominates each cortical, subcortical, and cerebellar region. The resulting spatial mosaic shows that different brain territories are governed by distinct neuromodulatory regimes, reflecting both local receptor density and the strength of incoming neuromodulatory projections. This regional dominance highlights how hemispheric lateralization and functional specialization emerge from the interaction between asymmetric molecular substrates and large-scale neuromodulatory wiring. The cortical (top row), basal ganglia (middle row), and brainstem and cerebellar (bottom row) surfaces are represented on the left (first column), right (second column), superior (third column), and inferior (fourth column) views. The map was colored according to the neurotransmitter system of the map (either receptor or transporter) with the highest value at a voxel level. Abbreviations of regions/structures: Cau–caudate nucleus, Cb–cerebellum, DLPFC–dorsolateral prefrontal cortex, L–left, LPC–lateral parietal cortex, LTC–lateral temporal cortex, OFC–orbitofrontal cortex, OP–occipital pole, Pu– pulvinar, R–right, RN–red nucleus, SN–substantia nigra, STN–subthalamic nucleus, Th–thalamus. Abbreviations of receptors/transporters: 5HT1aR–serotonin receptor 1a, 5HT1bR–serotonin receptor 1b, 5HT2aR–serotonin receptor 2a, 5HT4R–serotonin receptor 4, 5HT6R–serotonin receptor 6, 5HTT–serotonin transporter, alpha4beta2R acetylcholine receptor alpha4beta2, D1R–dopamine receptor 1, D2R–dopamine receptor 2, DAT– dopamine transporter, M1R–muscarinic 1 receptor, NAT–noradrenaline transporter, VAChT–acetylcholine vesicular transporter. *Experimental data, reproduced from Alves et al. (2025), Nat Commun, 2025, under CC BY-NC-ND 4.0 (no changes made).*


a.
a.1.
Resting state fMRI
DREADD Agonist
time
a.2.
Functional Connectivity
ρ = 0.82
ρ = -0.01
Normalized BOLD
time
b.
Activation ACh neurons BFB
FC of DMN
Baseline FC
Post injection FC
Baseline
ACh release
c.
fMRI FC
Simulated FC
d.
d.1.
Basal forebrain
d.2
RmPFC
LmPFC
Synchronisation
stimulus-driven spike trains
BF
d.3
LH
RH
PAC
Stimulation
undetected stimulus
detected stimulus
d.4.
VAChT
α4β2
M1
Left
Right
e.
e.1
Glu (all hits)
(incongruent hits) ACh
ACh level in tissue
time to cue onset [s]
e.2
cortical neurons
ACh release
glutamatergic (NMDAR) presynaptic control
'diffuse' cholinergic basal forebrain projections
e.3.
prefrontal cortex
motor cortex
basolateral amygdala
basal forebrain cholinergic cells
caudate
lateral septum
somatosensory cortex
central amygdala
f.
Reset > External
basal forebrain
Self > External
Conjunction

inance for sustained and spatial attention versus left hemisphere specialization for task-directed attention, demonstrating how attention fundamentally shapes hemispheric functions. Furthermore, Liu et al. (2022) notes that the hemispheres are asymmetrically engaged in attention, emotion, and cognition, reinforcing that attention plays a central role in establishing the distinct processing profiles of each hemisphere.

Hemispheric asymmetries in bottom-up processing and thalamic dynamics

Although sensory inputs reach both hemispheres through largely symmetrical thalamocortical relays, their cortical impact is profoundly asymmetric. Electrophysiological and fMRI studies demonstrate that identical bottom-up stimulation evokes faster, higher-gain responses in the left hemisphere for stimuli with temporal structure (e.g., speech, sequential tone trains), and stronger, longer-lasting synchronization in the right hemisphere for stimuli with spectral richness or emotional salience (Zatorre & Belin, 2001; Abrams et al., 2008). This lateralized reactivity arises not from asymmetry of the peripheral input, but from hemispheric differences in thalamocortical filtering, inhibitory tone, and neuromodulatory weighting. These hemispheric differences in bottom-up reactivity are further shaped by neuromodulatory gating of large-scale networks: as shown in Fig. 14f, basal forebrain and mediodorsal thalamus exert opposing influ-

Fig. 14. Functional connectivity of the default mode-like network (DMLN) under baseline and enhanced cholinergic conditions. This figure illustrates how cholinergic neuromodulation dynamically reshapes large-scale network organization by suppressing default-mode connectivity while preserving sensory processing, thereby promoting flexible reallocation of cortical resources. Using a combination of chemogenetic manipulation, resting-state functional magnetic resonance imaging (fMRI), computational modeling, and receptor mapping, the panels progress from causal experimental intervention to circuit-level and neurochemical mechanisms. Selective activation of basal forebrain cholinergic neurons reduces functional connectivity within the default mode-like network in rodents without disrupting sensory networks (a–c), demonstrating a state-dependent shift from internally oriented to externally engaged network configurations. At the electrophysiological and cellular level, cholinergic signaling modulates cortical theta–gamma coupling and introduces hemispheric asymmetries shaped by receptor distribution and transporter density (d), while tonic and phasic acetylcholine release provide temporal precision and spatial specificity through interactions with local glutamatergic circuits (e). Finally, ultra-high-field human fMRI reveals complementary roles of the basal forebrain and mediodorsal thalamus in suppressing or activating default mode network (DMN) regions depending on cognitive context, highlighting neuromodulation as a key mechanism linking network flexibility, attentional state, and interhemispheric coordination (f). a. Experimental framework. Chemogenetic activation (DREADDs) was used to selectively increase cholinergic activity in the rat basal forebrain. Resting-state fMRI was acquired following saline injection (baseline) and clozapine-N-oxide (CNO) administration, the latter upregulating cholinergic release. Functional connectivity (FC) of the DMLN (the rodent homologue of the human DMN) was assessed under both conditions. b. Simulation framework. Selective cholinergic activation of the DMN suppressed DMN activity and functional connectivity without altering sensory networks. The structural connectome of the DMLN provided the basis for simulations: synaptic inputs across nodes were converted into simulated blood-oxygenation-level–dependent (BOLD) imaging signals, and inter-regional correlations were used to estimate FC under baseline versus enhanced cholinergic states. c. Extension to human modeling. Using a computational model derived from diffusion-weighted MRI (DW-MRI) of the human connectome, selective cholinergic activation similarly suppressed DMN activity and connectivity without affecting sensory networks. *a-c, adapted from Sanda et al. (2024), with permission.* d. Cholinergic-dependent theta–gamma phase–amplitude coupling (PAC). (d.1) Anatomical projections of the basal forebrain cholinergic system. (d.2) Example oscillations in medial prefrontal cortex: gamma-band activity modulated by theta-rhythmic ACh release pulses from basal forebrain (compare with panel e.1). (d.3) Hypothetical model of interhemispheric differences: gamma amplitude synchronized by basal forebrain theta phase in left (LH) and right (RH) hemispheres, with asymmetries driven by differences in receptor concentrations. Lower panel illustrates functional consequences — stimuli detected versus undetected depending on attentional state. *d1-d3, created by the authors.* (d.4) Lateralization of acetylcholine transporter (VAChT) and receptor densities (α4β2, M1) in the human brain. *d4, adapted from Labache et al. (2025), with permission.* e. Tonic and phasic cholinergic signaling. (e.1) In rodent prefrontal cortex, tonic ACh levels are interspersed with phasic increases after behaviorally relevant cues, but only when these follow "non-cue" trials. These phasic bursts are preceded by glutamate increases occurring on all "cue-detect" trials. *After Thiele & Bellegrove, 2018, CC_BY*. (e.2) Local glutamatergic control of cholinergic release provides spatial and temporal specificity of cortical ACh signals. Release in rodent PFC partially depends on glutamate-driven presynaptic NMDA receptor activation from mediodorsal thalamic inputs. (e.3) Input–output segregation of basal forebrain cholinergic subcircuits: neurons projecting to prefrontal cortex receive input from lateral septum and central amygdala, whereas distinct subpopulations target motor cortex, somatosensory cortex, caudate, or basolateral amygdala *e1-e3, after Thiele & Bellgrove, 2018, CC_BY; (see also Gielow & Zaborszky, 2017.* f. Basal forebrain and mediodorsal thalamus (MD) modulation of default mode network (DMN) cortical regions. Ultra-high field fMRI in humans shows: top—DMN deactivation in Rest > External conditions (basal forebrain influence, violet arrows); middle—DMN activation in Self > External conditions (MD influence, green arrows); bottom—conjunction analysis of DMN activation versus deactivation, with cortical DMN regions highlighted (yellow arrows). Contrast maps are thresholded statistical parametric mapping (SPM) t-statistic images (PFDR<0.05) presented on the 'Synthesized_FLASH25' 500 μm MNI *ex vivo* template (Edlow et al., 2019). *Adapted from Harrison et al. (2022),with permission, CC-BY-NC.* Abbreviations: *fMRI–functional magnetic resonance, DW-MRI–diffusion-weighted magnetic resonance. a-c, adapted with permission from Sanda et al. (2024) with permission; d1-d3, created by authors; d4, adapted from Labache et al. (2025), with permission, CC-BY; e1-e3, after Thiele & Bellgrove, 2018, CC_BY; f, adapted with permission from Harrison et al. (2022), with permission, CC-BY-NC.*

ences on DMN cortical regions, biasing the brain toward externally oriented (BF-driven) or internally oriented (MD-driven) states, thereby introducing a lateralized context sensitivity into thalamocortical processing.

Thalamic symmetry and cortical asymmetry

At the thalamic level, nuclei such as the medial geniculate, pulvinar, and ventral posterior complex display bilaterally similar baseline firing and noradrenergic projections dominate in right thalamo-limbic loops, favoring contextual binding and affective weighting (Hansen et al., 2022). Consequently, the same stimulus can elicit analytic, temporally precise encoding in the left hemisphere and holistic, emotionally enriched representation in the right.

Bottom-up drive and neuromodulatory asymmetry

Bottom-up sensory drive itself is nearly symmetrical in the ascending pathways, optic radiations, lemniscal, and auditory tracts, but its effectiveness depends on the local neuromodulatory milieu. Cholinergic and dopaminergic terminals are denser in the left thalamus and fronto-temporal cortex, enhancing stimulus-driven amplification and predictive updating; in contrast, serotonergic interhemispheric communication, and that their cross-frequency interactions are likely essential for flexible hemispheric specialization in cognition, affect, and behavior.

Functional implications of thalamic functional asymmetry

During symmetrical stimulation (e.g., binaural tones, bilateral tactile input), EEG and MEG recordings show small but systematic interhemispheric phase lags — typically left-leading in beta/gamma, right-leading in theta/alpha bands (Luo & Poeppel, 2012). These delays reflect the intrinsic asymmetry of cortical microcircuits rather than differences in thalamic conduction. Thus, bottom-up stimulation is anatomically symmetrical but functionally lateralized, with hemispheric differences emerging from thalamocortical resonance, receptor topology, and neuromodulator balance.

In summary, thalamic activity remains largely bilateral in amplitude yet asymmetric in phase dynamics and receptor weighting, producing distinct modes of cortical entrainment. The hemispheres therefore transform the same afferent signal into complementary predictive templates: left-hemispheric high-frequency encoding optimizes sequential analysis, while right-hemispheric low-frequency coherence supports integrative perception and affective meaning.

Thalamic receptor asymmetry and temporal modes of cognition

Asymmetric receptor expression within the thalamic reticular nucleus (TRN) may underlie distinct temporal regimes of cortical processing. The left TRN exhibits denser perisomatic $GABA_A$-mediated inhibition and higher expression of NMDA GluN2B subunits, enabling rapid phase resetting, temporally precise inhibitory postsynaptic currents, and efficient beta–gamma synchronization (Ulrich & Huguenard, 1997; Huguenard & McCormick, 2007; Zilles & Palomero-Gallagher, 2017; Hansen et al., 2022; Zhang et al., 2023). These properties favor fast excitatory–inhibitory cycles and fine-grained temporal segmentation, optimizing rule-based computation, syntactic analysis, and verbal reasoning. Conversely, the right TRN displays a greater dendritic $GABA_B$ component and slower inhibitory kinetics, promoting alpha–theta resonance, extended temporal integration windows, and a bias toward associative processing (Huguenard & Prince, 1994; Crunelli & Leresche, 2002; Labache et al., 2024). This molecular and electrophysiological asymmetry establishes a dual thalamocortical architecture: a left-dominant, phasic mode specialized for precision and sequence, and a right-dominant, tonic mode favoring holistic synthesis and contextual integration.

Microtemporal asymmetry, cognitive style, and neurodivergence

The dynamic interplay between hemispheric modes may determine an individual's cognitive profile along the analytic–creative continuum. This framework is illustrated in Fig. 15, which schematically depicts how lateralized receptor composition and oscillatory kinetics within the thalamic reticular nucleus (TRN) give rise to distinct temporal regimes of cortical processing. Differences in inhibitory kinetics within the thalamic reticular nucleus (TRN), governed by the balance between fast $GABA_A$ (Anwar et al., 2017) and slower $GABA_B$-mediated inhibition as well as NMDA receptor–dependent excitation, are well positioned to bias thalamocortical circuits toward distinct oscillatory regimes. Faster, phasic inhibition favors beta–gamma synchronization associated with temporally precise, sequential processing, whereas slower inhibitory dynamics promote alpha–theta resonance linked to integrative and associative modes of information processing (Buzsáki & Schomburg, 2015; Halassa & Kastner, 2017; Poeppel, 2003). Whether such inhibitory and oscillatory biases exhibit systematic hemispheric asymmetries within the TRN remains an open empirical question. Flexible

transitions between these hemispheric regimes may provide the microtemporal substrate for individual variability in cognitive style, from systematic analytic reasoning to imaginative abstraction (Li et al., 2017). Predominance of the left, $GABA_A$–GluN2B-driven regime supports linear logic, symbolic manipulation, and rule-based inference — traits that typify mathematically oriented or systemizing cognition. Dominance of the right, $GABA_B$-mediated mode promotes divergent association, metaphorical abstraction, and multimodal imagination, characteristic of artistic and inventive thinking. Flexible alternation between these thalamocortical states, reflected in a high dynamic laterality index (Wu et al., 2022), may constitute the physiological substrate of integrative creativity observed in polymathic minds such as Einstein, where analytic precision and imaginative abstraction co-exist. In contrast, autism spectrum conditions frequently exhibit a left-lateralized and dynamically stable pattern within thalamocortical and frontotemporal networks (Floris et al., 2016; Sato et al., 2024), consistent with reduced interhemispheric reversibility and persistent beta–gamma coupling. Such stabilization enhances local precision and syntactic control but constrains cross-hemispheric integration and conceptual flexibility. At the opposite end, ADHD and related divergent-attention profiles are associated with excessive laterality fluctuation and variable thalamic gating, amplifying associative processing and spontaneous ideation at the cost of sustained focus. Within this framework, thalamic microtemporal asymmetry acts as a connecting mechanism between receptor-level dynamics, hemispheric specialization, and the diversity of human cognitive styles.

A concrete operationalization of these microtemporal dynamics is presented in Fig. 17, which introduces the dynamic laterality index (DLI) as a time-resolved measure of hemispheric dominance. Using sliding-window estimation of the correlation between each region of interest and hemisphere-specific global signals, DLI captures moment-to-moment fluctuations in lateralization (Fig. 17a). This approach distinguishes stable lateralization from its temporal variability, quantified by mean laterality (MLI), laterality fluctuations (LF), and laterality reversals (LR), as illustrated for a left-lateralized region in Fig. 13b. Temporal clustering of whole-brain laterality trajectories (Fig. 13c) reveals recurring spatial–temporal patterns, while state-transition analyses (Fig. 13d) show how individu-

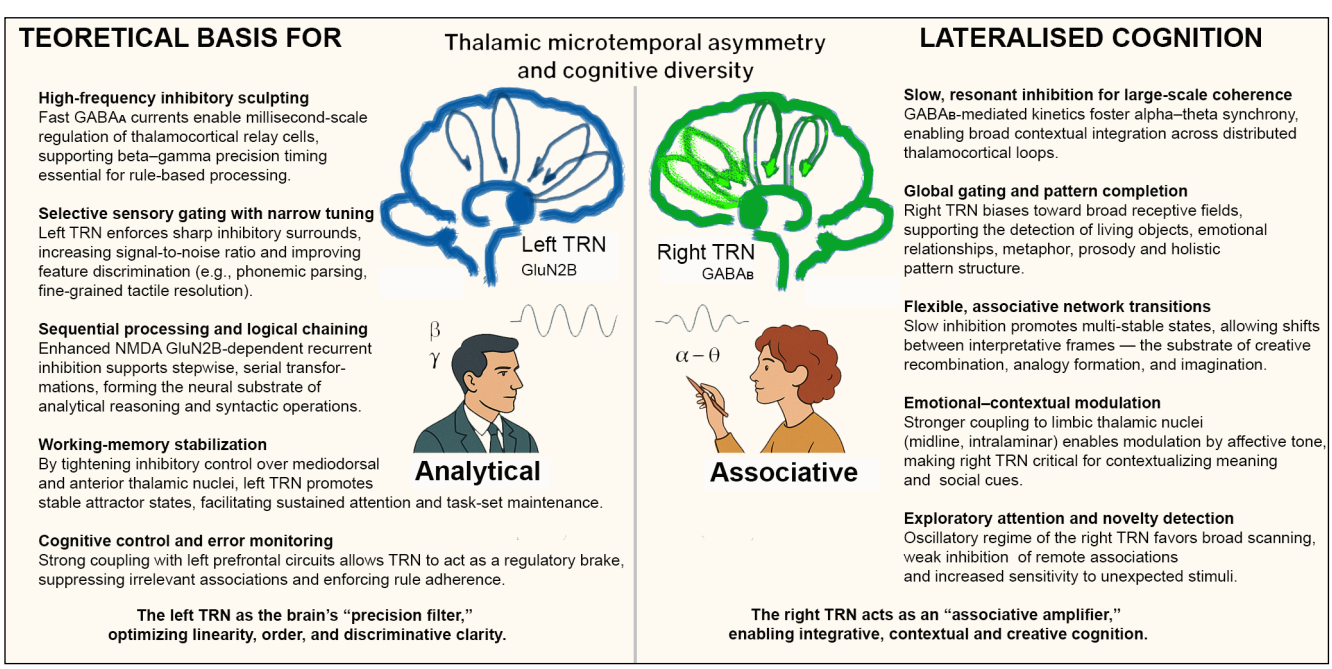


Fig. 15. Proposed microtemporal asymmetry of the thalamic reticular nucleus (TRN) and its implications for cognitive diversity. Molecular and kinetic asymmetries in TRN-mediated inhibition may contribute to circuit-level mechanisms underlying hemispheric differences in thalamocortical timing and, in turn, to variability in human cognitive styles. The left TRN is characterized by an enrichment of GluN2B-dependent excitatory input, supporting fast, phasic β–γ resonance and promoting an analytical processing style. In contrast, the right TRN shows a functional predominance of $GABA_B$-mediated slow inhibition, favoring α–θ oscillatory dynamics and facilitating associative, integrative modes of cognition. Together, these hemispheric differences in receptor composition and oscillatory thalamo-cortical timing provide a mechanistic basis for variability in human cognitive styles (Halassa & Kastner, 2017). *Created by the authors.*

als differ in the proportion of time spent in left-, right-, or bilaterally integrated states and in the probability of switching between them. Together, these findings indicate that hemispheric specialization is not static but dynamically reconfigures across timescales, linking microtemporal flexibility to individual cognitive style and neurodivergent profiles.

Attention as the Algorithmic Readout of Neuromodulatory Asymmetry

Attentional asymmetries represent one of the most robust dimensions differentiating hemispheric cognitive states. The right hemisphere plays a dominant role in sustaining vigilance, orienting towards salient stimuli, and distributing attention across both hemispaces, supporting global and exploratory processing (Marzi, 2025; Corbetta & Shulman, 2011). In contrast, the left hemisphere preferentially contributes to selective, goal-directed attention, particularly in tasks involving sequential analysis, linguistic processing, and fine-grained details (Mesulam, 1999; Zhu & Cai, 2025). Recent imaging and behavioral studies have confirmed that typical complementary lateralization of language and spatial attention confers measurable advantages: participants with typical lateralization show reduced interference and superior dual-task performance whereas atypical or bilateral patterns may impair efficiency (Cai et al., 2013; Zhu & Cai, 2025 Villar-Rodríguez et al., 2024, Bathurst & Kee, 1994). Updated neuroimaging atlases further delineate lateralized visuospatial attention networks that anchor these functional asymmetries (Labache et al., 2024). Collectively, these findings highlight that attentional mechanisms are not only shared across hemispheres but are asymmetrically weighted (Box 3), providing the clinical and neurocognitive foundation for divergent processing styles and explaining why attentional deficits, such as hemispatial neglect, are most severe following right-hemisphere damage (Heilman et al., 2003).

Downstream Signal Separate Filtration

Attention can be understood as a downstream filtering mechanism, selectively amplifying relevant neural signals while suppressing distractors. This process not only separates competing inputs but also sustains the continuity of cognitive activity — keeping the stream of consciousness alive. In electrophysiological terms, attention modulates the gain of sensory processing through biased competition, where attended stimuli elicit stronger neuronal firing, enhanced gamma synchronization, and improved signal-to-noise ratios across the cortical networks (Reynolds & Heeger, 2009; Fries, 2015). At the neurotransmitter level, neuromodulatory systems, particularly acetylcholine and norepinephrine, play a critical role in this filtering, dynamically adjusting network excitability to favor salient information (Aston-Jones & Cohen, 2005).

Thus, attention provides a dynamic gating function, ensuring that relevant signals are maintained in active processing ("kept alive") while irrelevant or redundant information is downregulated. This gating is a central factor differentiating hemispheric modes of cognition: the right hemisphere preferentially maintains a broad, sustained attentional field, whereas the left filters narrowly for goal-directed, sequential information (Bartolomeo, 2019).

Neuromodulatory Regulation of Interhemispheric Interactions and laterality dynamics

Neuromodulatory systems provide a higher-order regulatory level that shapes the oscillatory architecture of interhemispheric communication. By tuning the excitatory–inhibitory balance, adjusting the gain, and

Box 3. Attention as the Functional Readout of Interhemispheric Regulation.

Attention can be conceptualized as the emergent expression of interhemispheric coordination — a computational readout of neuromodulatory balance that converts molecular and oscillatory asymmetries into behavioral specialization. Operationally, attention functions as a neural gain-control and priority-weighting mechanism, dynamically modulating the signal-to-noise ratio within distributed cortical networks. In computational terms, it aligns with the principles of biased competition and divisive normalization, whereby competing neural representations are selectively amplified according to task relevance and contextual salience (Desimone & Duncan, 1995; Reynolds & Heeger, 2009). Within this framework, each neuromodulatory system adjusts a distinct gain parameter that biases attentional mode and hemispheric dominance: dopamine refines precision weighting and cognitive selection in left-lateralized goal-directed processes; acetylcholine enhances sensory gain and alpha suppression during bilateral cue detection; noradrenaline regulates global arousal and right-biased vigilance; and serotonin tunes temporal integration and affective gating. Thus, attention does not merely reflect cortical engagement but serves as the algorithmic outcome of neuromodulatory control — transforming molecular asymmetry into dynamic, lateralized patterns of cognition and behavior.

biasing hemispheric specialization, they selectively facilitate or constrain the crosstalk between homologous and heterologous networks. As recently has been finally shown the functional laterality is not a stabilized state of the brain. It fluctuates and has many shapes according to the situation which it has to deal with (Wu et al., 2022; Fig. 17) although some suggestions have been already posted (Serrien et al., 2006; Findlay et al., 2012). Wu and colleagues formalized this by introducing the dynamic laterality index (DLI), which quantifies time-varying shifts in hemispheric bias. From this, they distinguished laterality fluctuation (LF) — moderate variability around a dominant bias, from laterality reversal (LR) — full shifts from one hemisphere to the other. LF correlates positively with language and cognitive flexibility, whereas excessive LR predicts poorer outcomes.

Dopamine modulates interhemispheric signaling primarily through its impact on beta-band synchronization in cortico–basal ganglia–thalamic loops. This mechanism supports coordinated motor output and motivational biases; however, its asymmetry makes it particularly vulnerable, and disruption of dopaminergic tone leads to lateralized deficits in attention and motor control. Dopamine receptors are strongly lateralized, with left-hemisphere D1-dominated networks favoring goal-directed and approach behaviors, and right-hemisphere D2-rich circuits supporting vigilance, inhibition, and context monitoring. This asymmetry dynamically stabilizes hemispheric dominance during focused states while enabling flexible rebalancing through dopaminergic fluctuations (Box 4).

Serotonin acts on hippocampal and prefrontal interneurons to regulate theta–gamma coupling, thereby influencing the cross-hemispheric coordination of affective and mnemonic processes. Its lateralized receptor distribution contributes to the asymmetric vulnerability to mood disorders, linking serotonergic tone to both cognitive and emotional hemispheric biases.

Acetylcholine shapes alpha suppression and theta timing, providing flexible control of interhemispheric excitability during attention and learning. Through layer-specific modulation of pyramidal and interneuron populations, it facilitates cross-hemifield integration when bilateral attentional resources are required for the task.

Noradrenaline, ascending from the locus coeruleus, functions as a global gain-control system, enhancing

Box 4. Dopamine: beta-band synchrony, hemispheric stabilization, and motivational asymmetry.

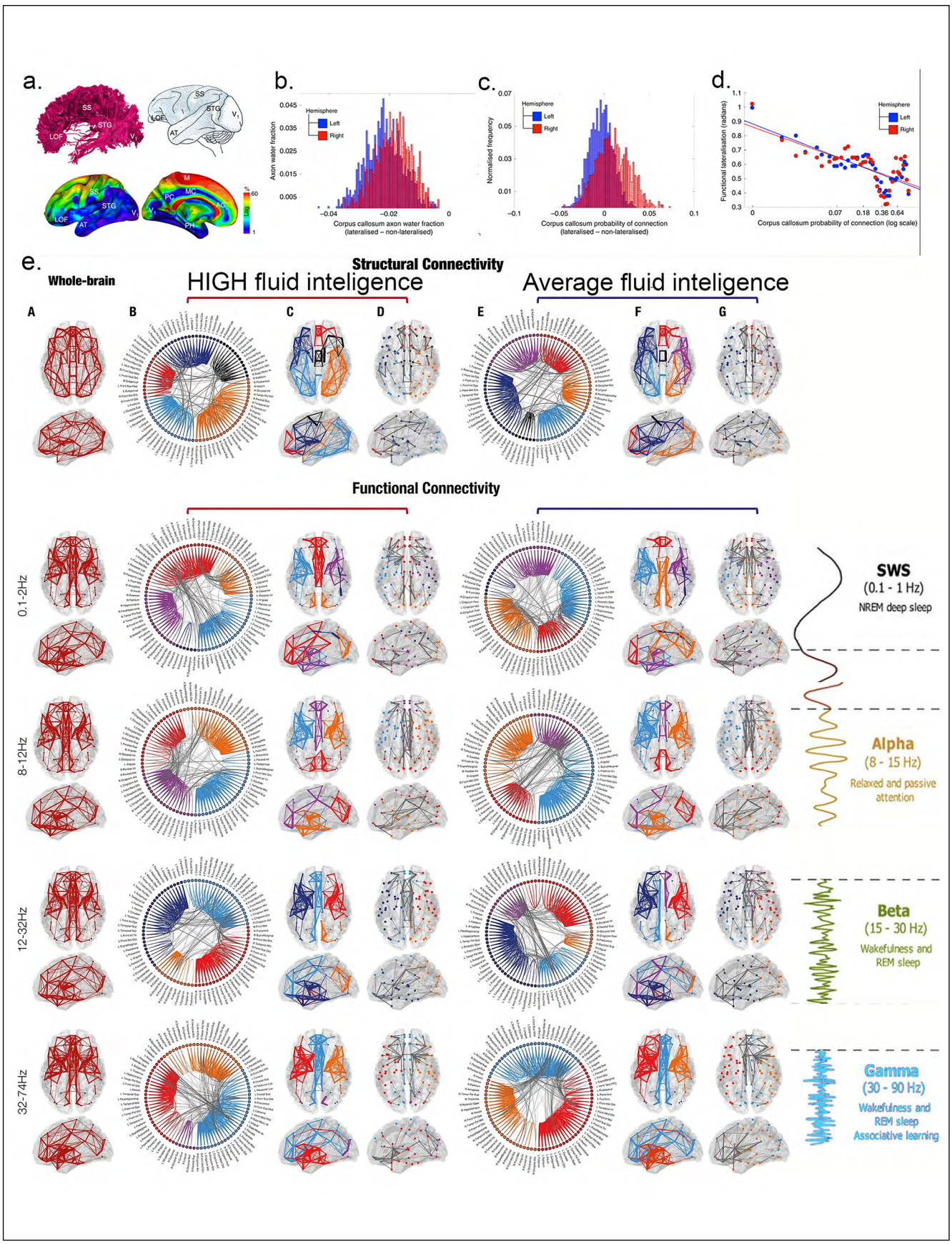
Dopamine represents one of the most powerful modulators of interhemispheric balance, influencing both motor and cognitive lateralization through its regulation of beta-band synchronization in cortico–basal ganglia–thalamic loops. This system, integrating prefrontal, striatal, and thalamic nodes, establishes the oscillatory backbone of goal-directed action and motivation. Within this architecture, dopaminergic tone adjusts the gain and timing of neuronal ensembles, synchronizing cortical hemispheres during the initiation and maintenance of voluntary behavior.

Anatomical and receptor mapping studies reveal clear dopaminergic asymmetry: D1-like receptors (D1, D5) are enriched in the left prefrontal and striatal regions, supporting approach-related, language, and sequential processing, while D2-like receptors (D2, D3, D4) show greater density in the right-hemisphere circuits associated with vigilance, avoidance learning, and spatial orientation. This receptor distribution shapes hemispheric specialization at multiple levels: molecular, electrophysiological, and behavioral. Through its modulation of β-band synchrony, dopamine stabilizes hemispheric dominance during focused task engagement, promoting segregation of functional networks and enhancing precision in cognitive control. Conversely, transient reductions or imbalances in dopaminergic tone can promote network re-integration, increasing cognitive flexibility and facilitating transitions between task sets or emotional states.

Functional imaging and pharmacological studies indicate that dopaminergic modulation of laterality is inherently dynamic. Fluctuations in dopamine release correlate with moment-to-moment shifts in hemispheric bias, as captured by the dynamic laterality index (DLI; Wu et al., 2022). In this framework, dopamine operates as a neuromodulatory stabilizer of hemispheric communication: high dopaminergic tone enforces lateralized control and cognitive focus, whereas reduced or desynchronized tone permits cross-hemispheric rebalancing and flexible reallocation of attention. Dysregulation of this system, through asymmetric receptor expression, lesion, or pharmacological depletion, results in the breakdown of interhemispheric coordination, manifesting as lateralized deficits in motor or cognitive function.

Clinically, such dysregulation is exemplified by akinetic mutism and post-encephalitic parkinsonism, conditions marked by dopaminergic depletion and functional silence across hemispheres (Sacks, 1973; Angeli et al., 2013). The restoration of movement and speech following L-DOPA administration — famously depicted in the movie Awakenings (1990), directed by Penny Marshall — illustrates that dopamine acts not only as a motor initiator but as a biochemical synchronizer that re-establishes reciprocal activity between hemispheres. Modern imaging supports this interpretation: right-hemisphere hypodopaminergia and disrupted β-coherence are consistently observed in states of mutism, catatonia, and hypoarousal, whereas recovery correlates with the re-emergence of interhemispheric β-band coupling and reinstatement of motivational drive.

In this context, dopamine can be viewed as a temporal bridge between hemispheres — linking left-lateralized executive control and right-lateralized arousal into a unified, dynamically balanced system. Its asymmetrical receptor architecture and oscillatory influence make it the principal neuromodulator of hemispheric stabilization and the molecular foundation of volition and intent.


a.
b.
c.
d.
SS
STG
LOF
AT
M
MC
PC
PH
Hemisphere
Left
Right
Axon water fraction
Corpus callosum axon water fraction (lateralised – non-lateralised)
Normalised frequency
Corpus callosum probability of connection (lateralised – non-lateralised)
Functional lateralisation (radians)
Corpus callosum probability of connection (log scale)
e.
Structural Connectivity
Whole-brain
HIGH fluid inteligence
Average fluid inteligence
A
B
C
D
E
F
G
Functional Connectivity
0.1-2Hz
8-12Hz
12-32Hz
32-74Hz
SWS
(0.1 - 1 Hz)
NREM deep sleep
Alpha
(8 - 15 Hz)
Relaxed and passive attention
Beta
(15 - 30 Hz)
Wakefulness and REM sleep
Gamma
(30 - 90 Hz)
Wakefulness and REM sleep
Associative learning

the signal-to-noise ratio and long-range coherence in the beta and gamma bands. Its preferential recruitment of right-hemisphere attention networks underpins vigilance, orientation, and cross-field monitoring, linking neuromodulatory asymmetry to adaptive behavioral responses.

Together, these neuromodulatory influences embed a chemical layer of regulation into the electrophysiological scaffold of the interhemispheric interactions. They do not merely amplify neuronal rhythms but also bias the directionality, flexibility, and resilience of cross-hemispheric communication. This integrative framework helps explain why neuromodulatory dysregulation contributes to asymmetric symptoms in conditions such as Parkinson's disease, depression, schizophrenia, and post-stroke syndromes, in which restoring interhemispheric balance remains a therapeutic challenge. Additionally, the systems introduce a chemical dimension to interhemispheric connectivity by selectively amplifying or suppressing communication channels, biasing hemispheric specialization, and enabling flexible, context-dependent regulation of cross-hemispheric information transfer (Avery & Krichmar, 2017; van den Brink et al., 2019). Increasing evidence suggests that these mechanisms are fundamental for adaptive cognition and for the pathophysiology of conditions marked by disrupted interhemispheric balance, such as post-stroke motor and mood disturbances (Casula et al., 2021), schizophrenia (Chang et al., 2019), and major depressive disorder (Wang et al., 2019).

Neurochemical Gradients, Conscious States, and Hemispheric Asymmetry

Recent multimodal neuroimaging has revealed that the large-scale dynamics underlying both cognition and consciousness are embedded within molecular gradients that mirror the structural–functional axes of lateralization (Hansen et al., 2022). As shown in Fig. 9, cortical territories exhibiting strong interhemispheric coupling (association cortices, precuneus, posterior cingulate, and superior temporal regions) align with high densities of serotonergic and cholinergic receptors, whereas lateralized regions — prefrontal,

Fig. 16. Lateralization and inter-hemispheric connectivity. This figure synthesizes how corpus callosum architecture constrains hemispheric lateralization while enabling flexible interhemispheric integration across cognitive states. Structural substrates of callosal connectivity are illustrated by tractography and axonal tracing in humans and non-human primates (a), followed by quantitative indices demonstrating reduced axonal water fraction and connection probability in lateralized cortical regions relative to non-lateralized areas (b, c). These anatomical features scale with functional organization, revealing a graded relationship between the degree of hemispheric specialization and interhemispheric coupling (d). At the network level, large-scale connectivity patterns show a dynamic trade-off between integration and segregation: individuals with higher fluid intelligence exhibit stronger interhemispheric integration during low-frequency oscillatory states, but increased hemispheric lateralization during task-related beta–gamma activity, highlighting flexibility as a core principle linking structural constraints to adaptive cognitive performance (e–g). a. Tractography of the corpus callosum in a representative subject *from the study by Karolis et al. (2019; top left*); cortical projection of the corpus callosum derived from axonal tracing in monkeys (*Myers 1965; top right*); cortical projections of the corpus callosum derived from tractography in the participants of the study (*bottom*). b. Histogram of the difference between lateralized and non-lateralized regions in the corpus callosum axonal water fraction, averaged across participants. c. Histogram of the difference between lateralized and non-lateralized regions in the corpus callosum probability of connection (c). The measure was calculated as the proportion of participants in which a connection exists between brain's voxels and corpus callosum to the overall Human Connectome Project (HCP) sample size. d. Dimensional relationship between the degree of functional lateralization and the corpus callosum probability of connectivity. *Adapted from Karolis et al. (2019), with permission*. e. inter- and intra-modular connectivity in high versus average fluid intelligence (Gf). At low-frequency oscillations (e.g., relaxing alpha or deep-sleep delta/theta states), individuals with higher Gf show greater interhemispheric connectivity compared to the average Gf group. In contrast, during task-related activity dominated by faster beta and gamma rhythms, the high Gf group exhibits stronger hemispheric lateralization, suggesting that enhanced intelligence is associated with a flexible shift between global integration at rest and functional specialization under cognitive load. (A) Whole-brain structural and functional connectivity in all participants computed from diffusion tensor imaging (DTI) data. (B) Circular connectogram representing inter- (in gray) and intra-module (colors) connections in high Gf participants between the 90 automated anatomical labeling (AAL) nodes. (C) Brain modules and intra-module connections overlaid on a standard brain template, in individuals with high Gf. Different modules are represented by edges with different colors. (D) Inter-module connections in individuals with high Gf. Different modules are represented by dots in different colors, while inter-module connections are represented by grey edges. (E) Circular connectogram representing inter- (in gray) and intra-module (different colors) connections in average Gf participants. (F) Brain modules and intra-module connections in individuals with average Gf. Different modules are represented by edges with different colors. (G) Inter-module connections in individuals with average Gf. Different modules are represented by dots in different colors, while inter-module connections are represented by gray edges. The whole-brain figures depict the whole-brain connections, with stronger connections being thicker. Color bars indicate the normalized average number of streamlines connecting the brain areas within connectivity modules. *Reproduced from Bruzzone, S. E. P., Lumaca, M., Brattico, E., Vuust, P., Kringelbach, M. L., & Bonetti, L. (2022). Dissociated brain functional connectivity of fast versus slow frequencies underlying individual differences in fluid intelligence: a DTI and MEG study. Scientific reports, 12(1), 4746. https://doi.org/10.1038/s41598-022-08521-5; used with permission from Leonardo Bonetti.* Abbreviations: LOF–lateral orbitofrontal cortex, SS–somatosensory cortex, STG–superior temporal gyrus, AT–anterior temporal, V1–primary visual area, M–primary motor area, PC–posterior cingulate gyrus, MC– middle cingulate gyrus, AC–anterior cingulate gyrus, PH–parahippocampal gyrus, AAL–nodes (Automated Anatomical Labeling) –nodes in fMRI as predefined brain regions by the AAL atlas. *a-c, adapted from Karolis et al. 2019, Nature Communication, with permission, under CC_BY;*

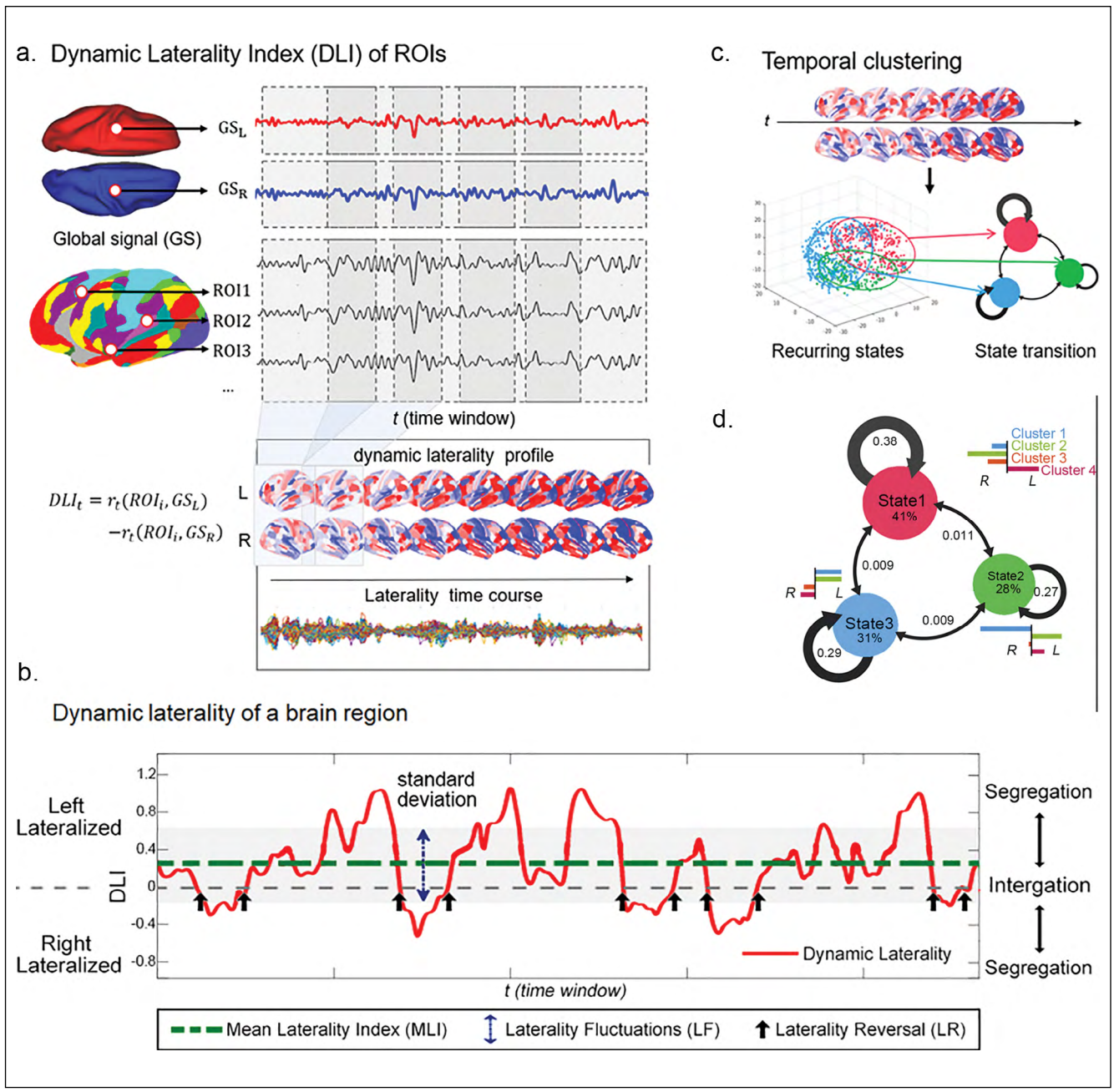


Fig. 17. Dynamic laterality. This figure highlights hemispheric lateralization as a dynamic, time-varying property that reflects continuous shifts between interhemispheric segregation and integration. Using a sliding-window approach, the dynamic laterality index (DLI) quantifies moment-to-moment differences in coupling of brain regions with left versus right hemispheric global signals (a, b). Temporal clustering of whole-brain laterality patterns reveals recurrent laterality states and their transitions, demonstrating that hemispheric dominance emerges from flexible network dynamics rather than fixed anatomical specialization (c, d). a. Definition of the dynamic laterality index (DLI). The DLI of the regions of interest (ROI) within time window t is defined as the correlation coefficient (z-transformed) between the left global signal (GS_L) and the ROI minus the correlation coefficient between right (GS_R) and the ROI. Using a sliding window approach, we obtained a time series of DLI for each ROI. b. Illustration of DLI and relevant dynamic laterality measures using an ROI that is left lateralized. The red curve represents the time series of DLI. The green dotted line is the MLI (0.26), and the blue double arrow denotes the standard deviation of the laterality time series, which measures the level of LFs. The black arrow represents the LR (the change of the sign of lateralization across 2 consecutive time windows). Large magnitude of laterality index indicates segregation at the hemispheric level, while small magnitude of laterality index (near 0) indicates integration across 2 hemispheres. c. Temporal clustering of whole-brain laterality patterns, which identifies potential recurring laterality patterns. d. The mean fraction of 3 states and the mean probability of switching between them. The bar plot next to each state represents the averaged laterality of the 4 spatial clusters in each state. L, left-lateralized; R, right-lateralized. *Adapted from experimental work of Wu et al.(2022),with permission, PLOS (2022), CC_BY.* Abbreviations: DLI–dynamic laterality index, GS–global signal, GSL–global signal of the left hemisphere; GSR–global signal of the right hemisphere, LF–laterality fluctuation, LR–laterality reversal, MLI–mean laterality index, ROI– region of interest.

perisylvian, and parietal executive areas — show comparatively elevated dopaminergic and glutamatergic receptor expression (Fig. 11). This molecular topology provides the neurochemical scaffold for the frequency-dependent integration and segregation described in Fig. 16a–g.

Fig. 16 illustrates how these molecular asymmetries translate into measurable differences in the architecture and dynamics of interhemispheric connectivity. Fig. 16 and 12 shows that the structural projections of the corpus callosum, mapped via human tractography and primate axonal tracing are not symmetrically distributed but follow the same lateralized gradients observed at the receptor level (Fig. 11; Labache et al., 2025; Alves et al., 2025). Regions with strong serotonergic–cholinergic signatures, typically multimodal associative areas, exhibit dense, bilateral callosal projections, whereas domains enriched in dopaminergic or glutamatergic receptors project more sparsely or focally across the midline. To quantify this principle, Fig. 2b–d demonstrate the lateralized territories which display reduced axonal water fraction and lower probability of callosal connection compared with non-lateralized regions, a pattern consistent with stronger local inhibition–excitation motifs and more selective gating of cross-hemispheric traffic (Karolis et al., 2019). Crucially, the dimensional relationship in Fig. 16d demonstrates that the degree of functional lateralization scales inversely with callosal connectivity probability, reinforcing the idea that neurochemical gradients impose structural constraints on the flow of interhemispheric information (Karolis et al., 2019). Finally, Fig. 16e–g extends these mechanisms to individual differences in cognition. High-Gf individuals, characterized by more efficient serotonergic–cholinergic integration at rest, show stronger low-frequency interhemispheric coupling (alpha, delta/theta). However, under cognitive load the same individuals express sharper lateralization in beta–gamma regimes, oscillatory bands linked to dopaminergic–glutamatergic signaling, demonstrating a flexible shift between bilateral integration and hemispheric specialization (Bruzzone et al., 2022). This frequency-specific reconfiguration mirrors the underlying receptor gradients: globally integrative states emerge where modulatory receptor density is high, while task-driven segregation prevails in dopaminergic–glutamatergic territories that favor precision, competition, and focal control. Together, these structural, oscillatory, and cognitive signatures encapsulated in Fig. 16 provide a mechanistic bridge between molecular asymmetries and the dynamic balance of hemispheric cooperation and competition that underlies human cognition.

Interhemispheric Dynamics and the Architecture of Cognition

Cognition arises from the brain's capacity to flexibly balance integration and segregation across distributed neural systems. Structural and functional data (Fig. 13 and Fig. 16) demonstrate that this balance is mediated by frequency-dependent interhemispheric dynamics, which in turn determine the efficiency and adaptability of large-scale cognitive operations.

As shown in Fig. 16, at rest, when cortical activity is dominated by slow delta–theta–alpha oscillations, individuals with higher fluid intelligence exhibit stronger interhemispheric coupling, particularly between homotopic associative areas (Bruzzone et al., 2022). This low-frequency synchrony supports global information sharing and the maintenance of a broad, context-sensitive representational space — an electrophysiological substrate for integrative cognition. The dense commissural architecture of the corpus callosum supports interhemispheric phase alignment among distributed cortical ensembles (Karolis et al., 2019), enabling large-scale coordination of neural activity associated with working memory maintenance and sustained situational awareness, even in the absence of explicit task engagement.

In sum, cognition can be understood as an emergent property of frequency-dependent, structurally constrained coordination between the hemispheres. The callosal–thalamic system acts as a regulator of this coordination, mediating both hemispheric integration during low-frequency, exploratory states and hemispheric segregation during high-frequency, task-focused states. This dual-mode architecture underlies the flexibility of human thought, permitting the brain to oscillate between unified and specialized processing while maintaining coherence across its two hemispheres.

Mechanistically, this dual-mode architecture operates through the dynamic regulation of interhemispheric coupling by frequency-specific oscillations and neuromodulatory influence. During low-frequency (delta–theta–alpha) synchronization, large-scale cortical networks (particularly the default mode, salience, and thalamocortical systems) achieve phase alignment across hemispheres, allowing distributed representations to integrate into a coherent global workspace (Dehaene & Changeux, 2011). This state supports associative and creative cognition, where semantic, episodic, and affective information converge into unified conceptual frames.

Neuromodulators such as acetylcholine and dopamine enhance cortical excitability and reduce long-range or interhemispheric coherence, promoting

local gamma- and beta-band synchronization within domain-specific modules. Acetylcholine enhances short-range synchrony by increasing intracortical gain and stabilizing gamma oscillations (Munk et al., 1996; Bauer et al., 2015), whereas dopamine promotes gamma- and beta-dominant attractor states within prefrontal microcircuits and facilitates local gamma↔beta transitions (Roopun et al., 2008; Durstewitz & Seamans, 2008).

At the same time when the cognitive demand increases and cortical activity shifts toward faster beta- and gamma-band rhythms (Kujala et al., 2024; Hashimoto et al., 2017), the network architecture reorganizes toward lateralized specialization (Doron et al., 2012) and enhanced intra-modular connectivity within hemispheres (Khan et al., 2018) mediated by frequency-dependent segregation mechanisms. This regime supports efficient, goal-directed computation—allowing one hemisphere to dominate specific cognitive domains such as linguistic processing (Geschwind & Levitsky, 1968; Hickok & Poeppel, 2007), executive control (Aron et al., 2004), or spatial reasoning (Corbetta & Shulman, 2002), with the right hemisphere showing well-established specialization for visuospatial analysis and spatial attention (Corbetta & Shulman, 2002; Jäger & Postma, 2003; Karnath & Rorden, 2012). Empirical evidence shows that high-frequency engagement during language tasks is strongly left-lateralized (Hashimoto et al., 2017) and that cognitive operations with greater semantic and syntactic load evoke widespread increases in beta- and gamma-band cortico-cortical coherence (Kujala et al., 2024). Developmental and systems-level analyses further demonstrate that gamma-mediated networks become increasingly integrative, whereas beta-mediated networks support growing local segregation (Khan et al., 2018), matching the proposed shift toward intra-modular efficiency under high cognitive demand. These dynamic shifts are consistent with network-level models of interhemispheric coordination and functional lateralization (Doron et al., 2012) as well as broader accounts of structural and functional hemispheric asymmetry (Wang, 2023). The alternation between low-frequency integration and high-frequency specialization, therefore, embodies a dynamic cognitive equilibrium: a capacity to couple and decouple hemispheric resources according to task requirements.

Therefore cognition is not merely a static state but a dynamic interplay that incorporates both slow oscillatory rhythms, which contribute to broad integration of information, and faster frequencies that facilitate specialized processing. This cross-frequency interaction enhances the brain's ability to alternate between heuristic exploration and analytical reasoning, essential for adaptive responses in complex environments (Yizhar et al., 2011; Sohal & Rubenstein, 2019). Evidence from empirical studies supports the notion that interhemispheric synchrony is reminiscent of low-frequency oscillations, whereas the content of consciousness aligns with brief, high-frequency activities manifested during focused cognitive tasks (Higley & Contreras, 2006; Yizhar et al., 2011).

Within this framework, the "left-brain interpreter", a concept described by Gazzaniga and LeDoux (1978) can be viewed as a higher-order cognitive manifestation of hemispheric asymmetry in these oscillatory regimes. The left hemisphere's bias toward linguistic sequencing and causal inference enables it to retrospectively integrate discrete, lateralized perceptual events, originating across both hemispheres, into a coherent, narrative representation. Functionally, this interpreter may operate as a metacognitive integrator, binding fast, content-specific activity (gamma/beta) into the slower, globally synchronized rhythms (alpha/theta) that support awareness and continuity of self.

In this sense, the interpreter is not an independent module but an emergent property of the left hemisphere's predictive and verbal networks acting upon interhemispheric synchrony. When callosal integration is intact, its narrative constructions are continuously constrained by contralateral feedback, ensuring consistency between hemispheric representations. When communication is disrupted — as in split-brain conditions — the left hemisphere continues to generate coherent explanations despite missing information, revealing the interpreter's role as a predictive narrator that imposes causal order on partial data.

Thus, the left-brain interpreter may represent the cognitive endpoint of cross-hemispheric negotiation: a linguistic and conceptual synthesis of distributed neural activity, shaped by oscillatory coupling, structural asymmetry, and the drive for coherence in conscious experience.

At the neurochemical level, these oscillatory regimes are stabilized by the interplay between glutamatergic excitation and GABAergic inhibition, modulated by long-range neuromodulatory systems. The balance between GABA and glutamate defines the excitatory–inhibitory (E/I) ratio that determines whether cortical ensembles engage in coherent low-frequency synchrony or fragment into fast, specialized subnetworks. In the context of hemispheric specialization, local differences in $GABA_A$ and $GABA_B$ receptor density, as well as asymmetric expression of glutamatergic receptor subtypes such as NMDA-GluN2A/B, can bias one hemisphere toward sustained inhibition and stability, and the other toward transient excitation and representational updating (Harms et al., 2020; Ocklenburg et al., 2017).

Superimposed on this microcircuit balance, neuromodulators provide a dynamic, context-dependent gain control that shapes interhemispheric coordination. Acetylcholine enhances signal-to-noise ratio and temporal precision of gamma oscillations (Buzsáki & Wang, 2012; Goard & Dan, 2009), thereby facilitating localized, content-specific processing; noradrenaline and dopamine adjust network flexibility, promoting transitions between global and local coupling (Aston-Jones & Cohen, 2005; Shine, 2019; Seamans & Yang, 2004); serotonin biases the system toward stability and introspective modes (Carhart-Harris & Friston, 2019; Cools et al., 2008). Through these mechanisms, neuromodulators act as meta-controllers of hemispheric dialogue—regulating when the brain enters a globally integrated (low-frequency) regime supporting conscious continuity (Engel & Fries, 2010; Palva & Palva, 2012), and when it shifts toward high-frequency, lateralized computations enabling focused cognition (Sohal et al., 2009).

From this perspective, the left-brain interpreter may be conceived as a neuromodulator-stabilized attractor state within left-hemispheric predictive networks (Gazzaniga, 2000; Friston, 2018): a GABA-constrained yet glutamate-driven assembly (Isaacson & Scanziani, 2011) whose coherence is gated by cholinergic and dopaminergic tone (Williams & Goldman-Rakic, 1995). Its causal narratives thus emerge not merely from linguistic circuitry but from the dynamic tuning of excitatory–inhibitory balance and neuromodulatory gain that governs cross-hemispheric information flow (Deco et al., 2011).

Interhemispheric Dynamics and the Architecture of Consciousness

Conscious experience is widely considered to depend on the brain's ability to integrate distributed information while maintaining functional differentiation, a principle formalized in models such as Integrated Information Theory (Tononi, 2004) and the Global Neuronal Workspace (Dehaene & Changeux, 2011). The data summarized in Fig. 16 illustrate that this integration–segregation balance is not only dynamic but also frequency- and hemisphere-dependent (Bruzzone et al., 2022). Such broadband synchronization may constitute the neurophysiological substrate of the background unity of consciousness — the pre-reflective coherence that binds perceptual and mnemonic content into a single experiential field. The corpus callosum, by enabling coherent low-frequency communication between hemispheres, serves as a physical conduit for this large-scale integration (Karolis et al., 2019).

In contrast, during task engagement dominated by faster beta- and gamma-band rhythms, the same individuals display enhanced hemispheric specialization and intra-modular coupling (Bruzzone et al., 2022). This reorganization suggests that conscious access and cognitive control rely on a context-dependent decoupling: while global synchrony provides the substrate for awareness, selective desynchronization between hemispheres allows for differentiated processing and attentional focus. This flexible alternation between global and local coordination parallels the biased competition model of attention (Desimone & Duncan, 1995), in which attention dynamically amplifies relevant neuronal ensembles while suppressing competing signals.

The brain's ability to switch between these activity modes depends on the integrity of callosal fibers (Casali et al., 2013; Roland et al., 2017), fronto-thalamic loops (Halassa & Kastner, 2017; Schmitt et al., 2017), and inhibitory interneuron networks that modulate excitation–inhibition balance (Buzsáki & Wang, 2012; Sohal et al., 2009). By dynamically reconfiguring cross-hemispheric phase relationships, these systems allow cognition to alternate between global integration (exploratory, associative) and local specialization (focused, executive) without loss of overall coherence (Engel & Fries, 2010, Palva & Palva, 2012). In essence, the flexibility of human thought emerges from rhythmic, neuromodulator-driven control of interhemispheric connectivity (Fig. 13 and Fig. 16).

The integrated image of neuromodulation together with rhythmic oscillations between the brain regions suggest that the level of consciousness may depend on interhemispheric synchrony within slow oscillations, whereas the content of consciousness reflects transient lateralized assemblies operating in faster frequencies. Consciousness, therefore, can be conceptualized not as a static state but as a temporally evolving pattern of cross-frequency, cross-hemispheric coordination — a dynamic equilibrium sculpted by both structural connectivity and moment-to-moment demands on information processing (Nir & Tononi, 2010).

Within this framework, consciousness emerges as a rhythmically mediated negotiation between hemispheric integration and lateralized specialization. Low-frequency coherence provides the binding necessary for a unified phenomenal field, whereas high-frequency segregation supports the precision of perceptual and cognitive contents. The dynamic adaptability of callosal and thalamic pathways thus enables transitions between these regimes, permitting consciousness to fluctuate smoothly between diffuse awareness and focused cognition.

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Neuromodulators and States of Consciousness

Pharmacological and neuroimaging studies demonstrate that distinct neuromodulatory regimes map onto discrete states of consciousness. Cholinergic activation, originating from the basal forebrain, promotes gamma-band synchronization and cortical desynchronization, facilitating externally oriented awareness and high-precision perception. In contrast, serotonergic and GABAergic predominance favors global low-frequency coupling and internally directed modes such as daydreaming, meditative absorption, or sleep onset (Hansen et al., 2022). Noradrenergic tone from the locus coeruleus modulates transitions between these states by shifting the cortex along the integration–segregation axis, influencing arousal, vigilance, and attentional breadth.

After a right-hemispheric ischemic stroke, these chemical gradients of balanced neuromodulation are abruptly disrupted. Diffusion-weighted neurometabolic mapping (Alves et al., 2025) shows focal depletion of monoaminergic and cholinergic markers within the damaged hemisphere accompanied by compensatory hyper-density in contralateral homologues. Clinically, such imbalance manifests as transient mutism or emotional flattening, even when left perisylvian language areas remain intact. The phenomenon can be interpreted as a failure of interhemispheric dopaminergic gating (Carr & Sesack 2000): without the modulatory drive from the right fronto-insular and anterior cingulate regions (Patel et al., 2018; Frühholz et al., 2015), left-hemisphere language generators lack the motivational and affective tone necessary for expressive speech (Fuertinger et al., 2018). Recovery of prosody and initiative often parallels the restoration of cross-callosal dopaminergic balance (Raymer, 2003; Jin et al., 2017).

Receptor mapping reveal local glutamatergic disinhibition coupled with serotonergic amplification (Hansen et al., 2022, Dawson, 2001), giving rise to slow, high-amplitude theta synchrony across temporal–parietal cortices (Liechti et al., 2016). Patients frequently report intense emotional or "transcendent" experiences, heightened meaningfulness, unity, and timelessness (Liechti et al., 2016), corresponding to a transient shift toward globally coherent low-frequency coupling within serotonergic-rich networks (Castañé et al., 2008). Such episodes illustrate that abnormal modulation of the excitation–inhibition ratio can transiently push the system into a hyper-integrated state resembling the upper extreme of conscious unification, as demonstrated in pathological hypersynchrony during epileptic seizures (Engel & da Silva, 2012), abnormal gamma hyper-synchrony arising from disrupted PV-interneuron–mediated inhibition (Uhlhaas & Singer, 2010), and globally increased network integration induced by psychedelic perturbations of cortical E/I balance (Carhart-Harris et al., 2014; Deco et al., 2018).

Integrative Model

Together, these findings suggest that neuromodulatory asymmetries orchestrate the dynamic transitions between hemispheric integration and specialization that define both cognitive flexibility and conscious experience. The left-lateralized glutamatergic/dopaminergic networks sustain high-frequency, goal-directed computation and causal reasoning, the substrate of the left-brain interpreter, whereas right-lateralized serotonergic/cholinergic systems support contextual integration, affective meaning, and self-referential processing. The corpus callosum and thalamic commissures (see Box 2) mediate continuous negotiation between these chemico-oscillatory regimes, ensuring that cognition and consciousness remain coupled despite molecular and functional asymmetries.

Disruption of this equilibrium, whether by focal lesions, pharmacological perturbation, or pathological hyper-synchrony, leads to predictable phenomenological consequences—from aphasic mutism to mystical insight—depending on the direction of the neuromodulatory imbalance. The interplay of receptor gradients (Fig. 9 and Fig. 8) with frequency-specific interhemispheric dynamics (Fig. 16) thus delineates a unified chemo-oscillatory architecture of the human mind.

Cortical Plasticity and Clinical Relevance of Interhemispheric Interactions

Interhemispheric communication is not static but exhibits substantial plasticity during development, learning, and in response to injury. Such plasticity can involve synaptic strengthening or weakening, remodeling of callosal projections, and changes in neurotransmitter sensitivity (Engel & da Silva, 2012; Uhl-

haas & Singer, 2010; Carhart-Harris et al., 2014; Deco et al., 2018).

Experience-Dependent Remodeling of Callosal Circuits

Activity-dependent plasticity is essential for the refinement of callosal connectivity. Studies in rodents have shown that monocular deprivation (Pham et al., 2004), whisker trimming (Chung et al., 2017), and unilateral sensory stimulation can induce structural and functional changes in contralateral cortical areas (Herzberg et al., 2024). Importantly, the contralateral "intact" cortex, homotopic to the area of induced plasticity, can actively participate in cortical map rearrangement (Jablonka et al., 2021; Fig. 18).

In our experiment, one month of partial whisker deprivation induced a marked remodeling of contralateral cortical activity in response to stimulation of the spared whiskers. The expansion of the spared whisker representation in the somatosensory cortex, visualized through metabolic parameters, depended strongly on the pattern of stimulation. When whiskers were stimulated bilaterally, the visualised cortical representation of the spared whiskers expanded approximately twofold, whereas unilateral stimulation confined to the deprived hemisphere produced only about half of that enlargement. These results indicate that a substantial component of cortical map reorganization during sensory deprivation arises from interhemispheric inputs, rather than from plasticity processes restricted to the deprived hemisphere alone.

Our results further indicate functional symptoms of interhemispheric cooperation in plasticity, where the experience-dependent widening of cortical whisker representations was surrounded by activity from the contralateral hemisphere. We hypothesize that this effect reflects a strengthening of inhibitory input from homotopic contralateral areas surrounding the spared whiskers representation (Fig. 18b, e; Jablonka et al., 2021). These mechanisms and their spatial organization are summarized in Fig. 18, which provides a multi-layered reconstruction of experience-dependent plasticity in the barrel field (BF) cortex. Autoradiographic 2DG mapping demonstrates how bilateral versus unilateral stimulation shapes cortical representation of spared whisker rows in intact and deprived animals (Fig. 18a–d), revealing that contralateral inhibitory influences emerge during the deprivation period and modulate the metabolic widening of spared row representations. The schematic panels (Fig. 18e.1–e.3) further illustrate the hypothesized circuitry underlying this plasticity: ipsilateral BF activity spreads across layers V–VI, while homotopic contralateral inputs exert inhibitory control around the spared representation, thereby shaping the spatial specificity of map expansion. Finally, laminar 2DG profiles (Fig. 18f) show that unilateral spared-whisker stimulation evokes measurable metabolic responses predominantly in layer V, consistent with strengthened callosal drive and reduced contralateral inhibition when bilateral homotopic stimulation is absent. Together, these findings highlight a cooperative interhemispheric architecture in which experience-dependent remodeling of BF maps depends critically on the balance between direct ipsilateral drive and contralateral homotopic inhibition.

Callosal axons can undergo pruning or arborization depending on synaptic activity patterns, and these changes are mediated by calcium-dependent cascades (Nakagawa-Tamagawa et al., 2021), neurotrophic factors such as BDNF (Cohen-Cory & Fraser, 1995) and NGF (Glebova & Ginty, 2004), and epigenetic mechanisms (Lim et al., 2015). Long-range GABAergic neurons (LRGNs), particularly parvalbumin-positive (PV) interneurons (Zurita et al., 2018), may contribute to interhemispheric plasticity by regulating the synchrony of oscillatory activity. On the other hand, layer V entire barrel field response to ipsilateral one row whisker stimulation may reflect the lack of previously described contralateral inhibition present only when paired with bilateral homotopic whiskers stimulation (Fig. 18f; Palmer et al., 2012). Our results also present the spread of the pathway probably directly from the callosal input from the stimulated row B whiskers area in subcortical slices, layer VI and low layer Vb (Fig. 18f Vb) to all the areas of all the whiskers representation in layer V (Fig. 18f Va).

Recent evidence highlights the importance of callosally projecting PV interneurons (cPV cells, Zurita et al., 2012). Inhibition of cPV connectivity to contralateral prefrontal–mediodorsal (PFC–MD) cortex produces deficits that parallel earlier findings of this group showing that cPV inhibition persistently disrupts increases in interhemispheric PV gamma synchrony that normally occur during rule-shift learning (Cho et al., 2023). This suggests that cPV synaptic plasticity is required for the generation of interhemispheric PV gamma synchrony. Conversely, gamma synchrony itself may facilitate cPV synaptic potentiation during learning (Fig. 12), linking microcircuit-level enhancements in PV-driven gamma-entrainment with the macroscale shift toward frequency-dependent lateralisation illustrated in Fig. 16. As shown in Fig. 16e–g, high-frequency beta–gamma regimes are preferentially recruited during cognitively demanding states in individuals with higher Gf, paralleling the cellular logic whereby gamma-phase alignment strengthens

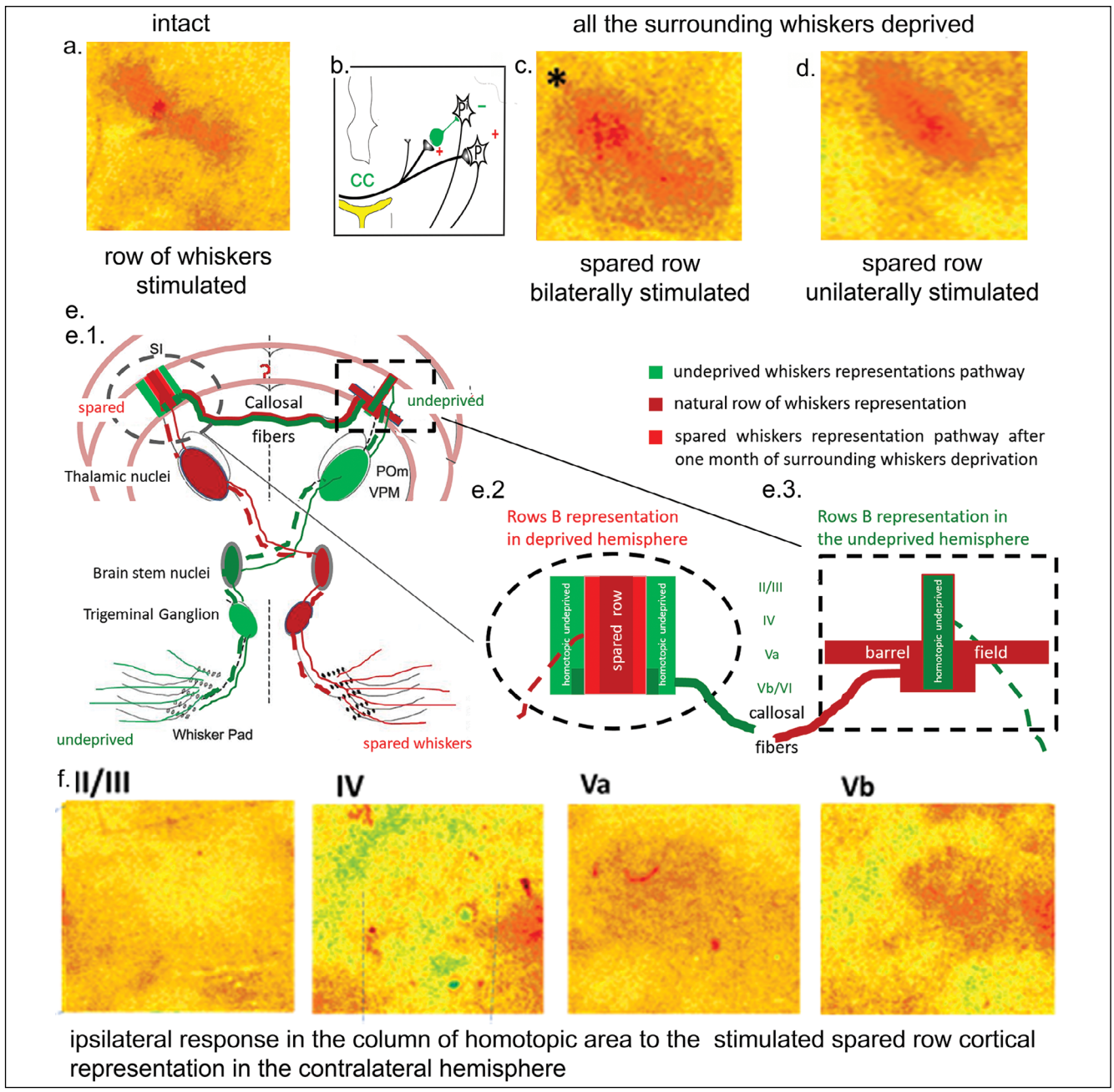

Fig. 18. Hypothetical reconstruction of experience-dependent plasticity (ExDP) in the barrel field (BF) cortex. Cortical representations of whisker rows were visualized with ^{14}C-2-deoxyglucose (2DG) functional brain mapping following whisker stimulation. a. Intact rats. b. Hypothetical contralateral inhibition developing in the area surrounding the spared row of whiskers representation a the less dense 2DG incorporation on Fig. 18c. c. Rats subjected to one-month unilateral deprivation of all whiskers except a single spared row, stimulated bilaterally during 2DG mapping. d. Same deprivation paradigm as in (b), but stimulated unilaterally during 2DG mapping. e. hypothetical connectivity and circuits interhemispheric integration during experience dependent plasticity. (e.1) Whisker–BF pathway. Cortical representations of whisker rows show activation profiles perpendicular to the row during bilateral stimulation of spared whiskers and their contralateral homotopic counterparts. (e.2) Cortical representation of a spared whisker row. (e.3) BF with contralateral homotopic row B representation (green) and spread of activation in response to ipsilateral spared whisker stimulation (red). Red: response to unilateral stimulation of spared whiskers from the deprived whisker pad. Dark red: response typical of undeprived whisker rows in intact animals. Green: activation evoked in deprived animals during bilateral stimulation of spared and contralateral homotopic whiskers. The green activation pattern in (e.2) is hypothesized to reflect transcallosal inhibition from contralateral homotopic row B, recruited during the deprivation period. Dashed line: contralateral whisker–BF pathway. Solid line: ipsilateral pathway. POm, posteromedial thalamic nucleus; VPM, ventroposteromedial thalamic nucleus. *Adapted from Shuler et al. (2001).* f. Cortical 2DG incorporation following the ipsilateral row B whiskers stimulation shown in four layers of the barrel field cortex. No response is seen in layers II/III and IV (slightly higher incorporation might be observed in deep layer IV). Layer Va shows evident 2DG incorporation in the entire surface of the BF with stronger activation of row B representation in layer Vb. *Adapted from Jablonka et al. (2021).*

cPV synapses and promotes hemisphere-specific processing. This suggests that gamma-locked PV plasticity may constitute a mechanistic substrate enabling the transition from globally integrated, low-frequency interhemispheric coupling (Fig. 16a–d) to lateralized, high-frequency specialization for example by aligning presynaptic spikes in callosally projecting PV neurons with postsynaptic spikes in PFC–MD neurons and/or by promoting burst firing at frequencies associated with PV synaptic plasticity (Lourenço et al., 2014).

These principles of gamma-dependent cPV potentiation resonate with the macroscale patterns shown in Fig. 16, where regions or individuals that rely more heavily on high-frequency (beta–gamma) modes during demanding cognitive operations exhibit stronger functional lateralization and tighter intra-modular coupling. In this view, gamma-entrained enhancement of cPV synaptic efficacy provides a plausible microcircuit mechanism for the shift toward specialized, hemisphere-specific processing observed in the high-Gf group (Fig. 16e–g), whereas low-frequency, broadly distributed synchrony supports the opposite regime — enhanced interhemispheric integration at rest (Fig. 16a–d). Thus, gamma-driven PV plasticity may be one of the cellular substrates enabling the flexible transition between bihemispheric integration and lateralized specialization captured in Fig. 16.

Hebbian mechanisms play a key role, however. In the absence of postsynaptic fragile X mental retardation protein (FMRP), callosal synaptic connections weaken specifically with experience-driven activity (Zhang et al., 2021). This suggests that uncorrelated or insufficient activity leads to processes of synaptic weakening, which inherently affects interhemispheric connectivity and integration (Zhang et al., 2021; Vitureira & Goda, 2013). The correlated activity between hemispheres strengthens interhemispheric connections (Tagawa et al., 2008), whereas uncorrelated input can weaken them (Schulte & Müller-Oehring, 2010, Zhang et al., 2021). The role of interhemispheric cooperation in mature brains is nuanced, often requiring a balance between Hebbian plasticity, which contributes to the enhancement of connections via coincident activity (Tagawa & Hirano, 2012), and homeostatic mechanisms, which help stabilize connections and counterbalance indiscriminate strengthening (Vitureira & Goda, 2013; Park et al., 2014; Oliveira et al., 2014). This plasticity is most prominent during critical periods of development (Uesaka et al., 2006) but persists into adulthood, albeit at a lower magnitude (Tagawa et al., 2008; Vitureira & Goda, 2013; Zhang et al., 2021; Park et al., 2014). However, in the early development it is strongly predetermined by ontogenetic potential (for more details, see the review by Krägeloh-Mann et al., 2017).

Experience-dependent changes are not limited to gray matter. White matter tracts also show structural remodeling: diffusion tensor imaging (DTI) in humans revealed that short-term training (six weeks of juggling practice) can induce measurable increases in fractional anisotropy (FA) within interparietal white matter tracts, particularly in regions underlying visuomotor control (Scholz et al., 2009). This reflects microstructural changes within white-matter pathways, such as enhanced myelination, increased axonal packing, or improved fiber coherence, that facilitate more efficient signal transmission between parietal regions engaged in visuomotor integration.

These principles of structural malleability and functional reorganization are further illustrated in Fig. 16, which integrates tractographic, metabolic, and electrophysiological evidence to describe how interhemispheric connectivity constrains and enables lateralized cortical plasticity. Fig. 12a demonstrates the large-scale anatomical layout of callosal projections, combining human tractography with classic axonal tracing in non-human primates to reveal the spatial specificity of interhemispheric pathways. Quantitative analyses (Fig. 12b–c) show that brain regions with strong functional lateralization possess reduced axonal water fraction and lower callosal probability of connection, whereas bilaterally engaged regions show denser, more reliable commissural coupling. This structural relationship scales with function: as shown in Fig. 12d, the degree of functional lateralization covaries inversely with callosal connectivity, indicating that plastic changes in hemispheric specialization depend on both within-hemisphere reinforcement and between-hemisphere decoupling.

Complementing these structural results, Fig. 12e–g reveal how individual differences in fluid intelligence (Gf) reflect shifts between interhemispheric integration and functional segregation across oscillatory regimes. At slow frequencies (delta–theta–alpha), individuals with higher Gf exhibit stronger global interhemispheric coupling, whereas during task-related beta–gamma activity they show enhanced lateralization within specialized modules. This frequency-dependent shift, visible in whole-brain structural/functional maps, circular connectograms, and module-level connectivity patterns, highlights that efficient cognition relies on the dynamic capacity to alternate between bilateral integration at rest and hemispheric specialization under cognitive load. Together, the structural and electrophysiological dimensions on Fig. 16 underscore that interhemispheric plasticity is constrained by callosal architecture yet flexibly modulated by oscillatory state and cognitive demands.

Post-Stroke Rebalancing of Interhemispheric Inhibition and Network Rhythms

Following unilateral ischemic injury, the balance of interhemispheric inhibition and excitation becomes profoundly disturbed. The intact hemisphere often exerts excessive transcallosal inhibition on the damaged side, suppressing cortical excitability and impeding recovery (Murase et al., 2004; Grefkes & Fink, 2014). This imbalance, reflected in asymmetric beta–gamma coupling and altered GABAergic tone, constitutes one of the key pathophysiological mechanisms limiting spontaneous recovery.

It has been demonstrated on animal models that in the subacute phase following focal ablation or stroke, callosal axons originating from the contralesional hemisphere exhibit directed sprouting toward the peri-infarct cortex (Jablonka et al., 2010; Dancause et al., 2005). This lesion-oriented transcallosal axonal growth forms novel heterotopic connections that partially restore interhemispheric balance and compensate for lost ipsilateral projections, consistent with animal studies demonstrating post-stroke callosal sprouting and remodeling (Lee et al., 2013). Axonal tracing and two-photon imaging reveal that such sprouting is activity-dependent and strongly modulated by excitatory and neuromodulatory inputs (Wang et al., 2023), while intensive sensory–motor rehabilitation further amplifies these structural changes (Jones et al., 2015). These anatomical adaptations parallel the recovery of transcallosal inhibitory function measured with paired-pulse TMS and coincide with improvements in motor coordination and speech-related initiation processes in human stroke survivors (Jones et al., 2015).

A clinically observable correlate of this reorganization is the phenomenon of mirror movements, in which voluntary movement of one limb evokes involuntary activation in the homologous contralateral limb. Mirror movements represent a failure of interhemispheric inhibitory gating through the corpus callosum and the transcallosal motor pathways (Nelles et al., 1998; Wittenberg et al., 2000). Mirror movements are most frequent during the early post-stroke phase, particularly in patients with extensive right-hemispheric lesions affecting prefrontal–premotor circuits. Functional MRI and electromyography (EMG) coherence studies show that mirror movements coincide with bilateral activation of M1 and premotor cortices and excessive interhemispheric beta synchrony. Therapeutically, motor rehabilitation protocols leverage this transient hyperconnectivity to promote adaptive rebalancing. Techniques such as constraint-induced movement therapy (CIMT), bilateral arm training, and mirror visual feedback (Michielsen et al., 2011) engage both hemispheres and enhance task-specific neuroplasticity. Non-invasive stimulation, using inhibitory repetitive transcranial magnetic stimulation (rTMS) over the intact hemisphere or excitatory transcranial direct current stimulation (tDCS) over the affected one, can further facilitate the restoration of balanced interhemispheric inhibition (Nowak et al., 2009; Grefkes & Ward, 2014). At the molecular level, effective rehabilitation after stroke appears to depend on the restoration of neuromodulatory balance — particularly within dopaminergic and cholinergic systems — that supports interhemispheric interactions and cortical plasticity (Boddington & Reynolds, 2017; Gower, 2018; Johnstone et al., 2017). Restoration of cross-callosal dopaminergic balance parallels the recovery of prosody, initiative, and spontaneous movement, underscoring dopamine's role as a biochemical synchronizer of hemispheric activity.

Together, these findings indicate that post-stroke rehabilitation capitalizes on the brain's intrinsic capacity for lesion-directed transcallosal rewiring, transforming maladaptive hyperconnectivity into compensatory network integration. Understanding and modulating these bihemispheric dynamics — through behavioral, pharmacological, and neuromodulatory means — remains central to modern neurorehabilitation.

Psychiatric and Neurodevelopmental Disorders (Dyslexia, Autism, ADHD, and Schizophrenia)

Disruptions in interhemispheric communication have been implicated in several neurological and psychiatric disorders. In dyslexia, atypical callosal morphology and reduced interhemispheric coherence are associated with deficits in phonological processing (Finn et al., 2014). In schizophrenia, aberrant interhemispheric transfer time and altered white-matter integrity of the corpus callosum are linked to disorganized thoughts and impaired cognitive integration (Whitford et al., 2010).

Autism spectrum disorder (ASD) is another condition in which interhemispheric connectivity is frequently altered. Diffusion tensor imaging (DTI) studies have consistently reported reduced fractional anisotropy in the corpus callosum, particularly in the splenium and genu, correlating with social-communication deficits (Just et al., 2007). Functional MRI studies reveal a leftward shift of cortical dominance, especially within temporal and prefrontal regions supporting language and social cognition (Nielsen et al., 2013; Sato et al., 2023). Importantly, recent dynamic laterality analyses indicate that individuals with ASD not only show atypical lateralization but also reduced flexibility of hemispheric switching, reflected in a lower variance of

laterality indices across time (Liu et al., 2024; Zhao et al., 2025). This rigidity of lateralization dynamics may hinder adaptive engagement of right-hemispheric networks responsible for social perception and contextual integration, thereby promoting an overreliance on left-hemisphere analytical processing.

In contrast, attention-deficit/hyperactivity disorder (ADHD) often exhibits the opposite pattern — a right-hemisphere shift in functional dominance. Structural and electrophysiological studies have revealed reduced callosal coherence and thickness between prefrontal and parietal cortices (Cao et al., 2009; Qiu et al., 2011), accompanied by hyperactivation of right frontoparietal and inferior frontal networks during attentional control tasks (Cortese et al., 2012). These findings suggest a compensatory right-hemispheric bias arising from reduced dopaminergic tone in left prefrontal circuits, resulting in inefficient interhemispheric regulation of attention and impulse control. Pharmacological normalization of catecholaminergic balance restores left-hemispheric recruitment and cross-hemispheric synchrony (Rubia et al., 2014).

Moreover, disorders of lateralization, such as alien hand syndrome and agenesis of the corpus callosum, provide striking examples of the necessity for intact interhemispheric communication (Geschwind et al., 1995; Sarva et al., 2014). These cases highlight the importance of callosal transmission for coherent voluntary action and a unified perceptual experience. Understanding the mechanisms of plasticity and pathology in interhemispheric communication thus has profound implications for both neuroscience and clinical practice, pointing toward targeted interventions that can enhance recovery and mitigate dysfunctions.

Recent evidence further shows that functional laterality is not a fixed trait but a dynamic state that adapts to situational demands (Fig. 14 and Fig. 17) (Wu et al., 2022). Temporal clustering of brain-wide laterality patterns revealed recurring "meta-states" whose occupancy and transitions covary with individual cognitive profiles (Wu et al., 2022). Neuromodulatory systems provide the mechanistic substrate for such flexibility. Dopaminergic, cholinergic, noradrenergic, and serotonergic pathways each bias interhemispheric processing in distinct yet overlapping ways (Durstewitz & Seamans, 2008; Aston-Jones & Cohen, 2005; Robbins, 2000). By dynamically recalibrating excitability, gain, and hemispheric specialization, they ensure that callosal and subcortical interactions remain adaptive rather than static. Disruption of this balance has clear clinical consequences. After stroke, excessive interhemispheric inhibition from the intact hemisphere hampers recovery (Murase et al., 2004; Grefkes & Fink, 2014). In hemispatial neglect or depression, asymmetries of attentional networks and neuromodulatory tone drive characteristic symptoms (Heilman et al., 2003; Grimm et al., 2009). These findings highlight that restoring neuromodulatory balance and recalibrating attentional asymmetry represent promising therapeutic strategies (Cramer, 2015).

Finally, we see the interhemispheric communication not as a static product of anatomy but as a dynamic interplay between synaptic transmission, oscillatory coordination, and neuromodulatory tuning. This multilayered system allows the hemispheres to operate in complementary yet adaptive ways, ensuring both stability and flexibility in cognition and behavior.

CONCLUSIONS

Interhemispheric communication operates along a hierarchy: anatomical pathways provide the structural scaffold, fast excitatory–inhibitory transmission defines synaptic rules, and neuromodulatory systems dynamically tune excitability and timing. The functional outcome of this architecture manifests most clearly through attention, which serves as the algorithmic readout of hemispheric lateralization (Corbetta & Shulman, 2011; Bartolomeo, 2019). The hemispheres work in complementary ways: the right hemisphere supports vigilance, spatial orienting, and broad monitoring, whereas the left hemisphere specializes in selective, sequential, and language-related processing. This division of labor is flexibly tuned by neuromodulators: dopamine promotes beta-band coupling in cortico–basal ganglia–thalamic loops; serotonin gates theta–gamma dynamics in hippocampal and prefrontal circuits; acetylcholine suppresses alpha rhythms during focused attention; and noradrenaline from the locus coeruleus provides right-biased "gain control" for vigilance networks (Robbins, 2000; Sara, 2009; Aston-Jones & Cohen, 2005; Makeig et al., 2004). Importantly, these influences are not mutually exclusive — neuromodulators act across multiple frequency bands, and their "preferred" associations may reflect research emphasis rather than strict biological segregation (Fries, 2015; van den Brink et al., 2019). In this sense, attention functions as the executive interface through which molecular and oscillatory asymmetries are translated into lateralized cognition and behavior.

Converging anatomical, electrophysiological, and neurochemical evidence demonstrates that cognition and consciousness emerge from the dynamic negotiation between hemispheric integration and specialization. Structural asymmetries of the corpus callosum and thalamocortical pathways, coupled with lateralized receptor distributions and oscillatory resonance fre-

quencies, constitute the substrate for this dual-mode organization. As shown by Bruzzone et al. (2022), individuals with higher fluid intelligence exhibit a capacity to flexibly shift between global, low-frequency interhemispheric coupling and local, high-frequency modular specialization. This frequency-dependent reconfiguration embodies the brain's ability to coordinate distributed processes without sacrificing efficiency or coherence — an ability that likely underpins abstract reasoning, creativity, and adaptive behavior. Neuromodulatory systems provide the dynamic control parameters of this architecture. Dopaminergic and cholinergic signaling promote focused, gamma-mediated specialization, whereas serotonergic and GABAergic tone stabilize large-scale alpha–theta synchrony supporting integrative and reflective cognition. Subcortical hubs such as the basal ganglia and thalamic reticular nucleus mediate transitions between these regimes, functioning as temporal gates that align hemispheric rhythms with contextual demands.

Disruptions of this balance have clear clinical consequences. After stroke, excessive interhemispheric inhibition from the intact hemisphere hampers recovery (Murase et al., 2004; Grefkes & Fink, 2014), while in hemispatial neglect and depression, asymmetries of attentional networks and neuromodulatory tone critically shape symptom profiles (Heilman et al., 2003; Grimm et al., 2009). Right-hemispheric stroke can induce mutism, apathy, or mirror movements, reflecting disrupted dopaminergic–GABAergic reciprocity and impaired callosal inhibition. Conversely, right-temporal hyperexcitability may elicit states of heightened unitive or transcendent awareness, arising from transient over-integration of serotonergic–limbic networks. Restoring neuromodulatory balance and recalibrating attentional asymmetry thus represent promising therapeutic strategies (Cramer, 2015).

Together, these findings suggest that the human mind operates as a bihemispheric, neuromodulator-tuned system, continuously oscillating between global coherence and local precision. This oscillatory–chemical complementarity, embedded in the structure of interhemispheric connections, enables thought to remain both stable and adaptive,unified yet diversified, across time and context.

Recent evidence, however, has further refined our understanding of the structural–functional relationship underlying interhemispheric communication. Patients with partial callosotomy, in whom only a small portion of callosal fibers remained intact, sometimes as little as one centimeter of the splenium, retained widespread patterns of interhemispheric functional connectivity and exhibited no behavioral signs of disconnection (Roland et al., 2017). In contrast, only complete callosotomy patients demonstrated extensive disruptions of interhemispheric network architecture, consistent with classical disconnection syndromes characterized by diminished information propagation across hemispheres.

Despite decades of assumptions linking callosal integrity directly to interhemispheric communication, these recent findings demonstrate that even minimal callosal remnants may sustain large-scale bilateral coordination. This suggests that alternative subcortical or extracallosal pathways, such as cerebellar, thalamic, and brainstem commissures, might substantially contribute to residual functional integration between hemispheres, reshaping the traditional view of structural dependence in interhemispheric communication (Tovar-Moll et al., 2014; Owen et al., 2013). It seems that communication in the brain is more like a stream which always finds the easiest path to flow in order to solve the external and internal problems of the body or the soul.

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