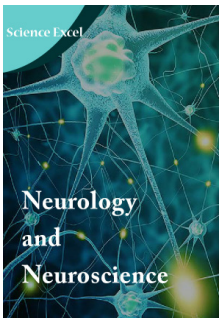
Neurology & Neuroscience

Original Article



Connectomics: Mapping the Brain's Complex Networks

Julian Ungar-Sargon

Borra College of Health Sciences, Dominica University River Forest, IL, USA

*Correspondence
Julian Ungar-Sargon MD PhD
Borra College of Health Sciences, Dominica University River Forest, IL, USA

Abstract

Traditional neuroscience has long focused on localization of function, mapping specific brain regions to discrete abilities or deficits. However, this approach risks oversimplifying the brain's inherently interconnected nature. Connectomics has emerged as a transformative field that maps neural connections as complex networks, shifting focus from isolated brain regions to integrated systems where function emerges from connectivity patterns.

This review examines how connectomics is revolutionizing our understanding of brain organization, disease mechanisms, and therapeutic relationships, with particular emphasis on clinical applications and the neurobiological basis of healing encounters.

We synthesized research from multiple scales of connectomic investigation—microscale synaptic mapping, mesoscale circuit analysis, and macroscale neuroimaging—alongside clinical studies demonstraeting network-based approaches to neurological and psychiatric disorders.

Connectomics reveals fundamental organizing principles including small-world architecture, hub connectivity, and modular organization that govern both healthy brain function and pathological states. Brain disorders increasingly appear as "connectome disorders" involving disrupted network patterns rather than focal lesions. Maladaptive responses such as diaschisis, transneuronal degeneration, and dedifferentiation can be understood through network topology, while adaptive responses including compensation and degeneracy depend on the brain's inherent redundancy and flexibility. Clinical applications range from precision neurosurgery guided by white matter tractography to network-informed brain stimulation therapies.

Connectomics provides a neurobiological framework for understanding how therapeutic relationships literally reshape brain networks. Social brain networks, including mirror neuron systems and default mode networks, create neural coupling between patients and clinicians. Therapeutic presence can regulate stress networks, facilitate neural synchronization, and promote narrative integration through mechanisms of co-regulation and neuroplasticity.

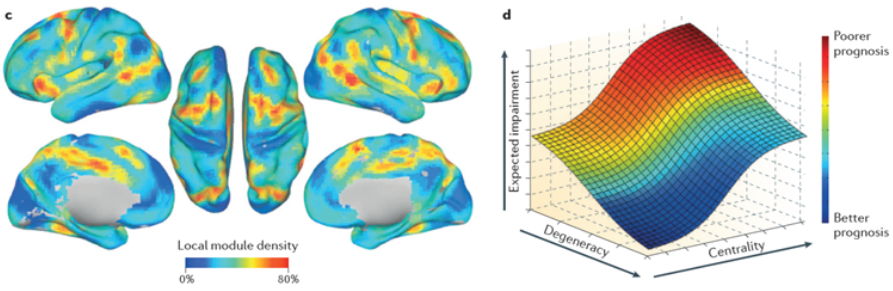
Dynamic connectomics, precision medicine based on individual connectivity profiles, and integration with artificial intelligence promise to further transform clinical practice. However, important limitations include the risk of network reductionism, cultural bias in universal models, and the potential for technological mediation to diminish human connection.

Connectomics represents both a technical revolution and conceptual reorientation that validates the brain as fundamentally social and interconnected. While providing powerful tools for understanding and treating brain disorders, it simultaneously affirms the irreducible importance of human relationships in healing. The shift from localization to connection transforms not only neuroscience but our understanding of what it means to be human.

- Received Date: 15 Sep 2025
- Accepted Date: 22 Sep 2025
- Publication Date: 26 Sep 2025

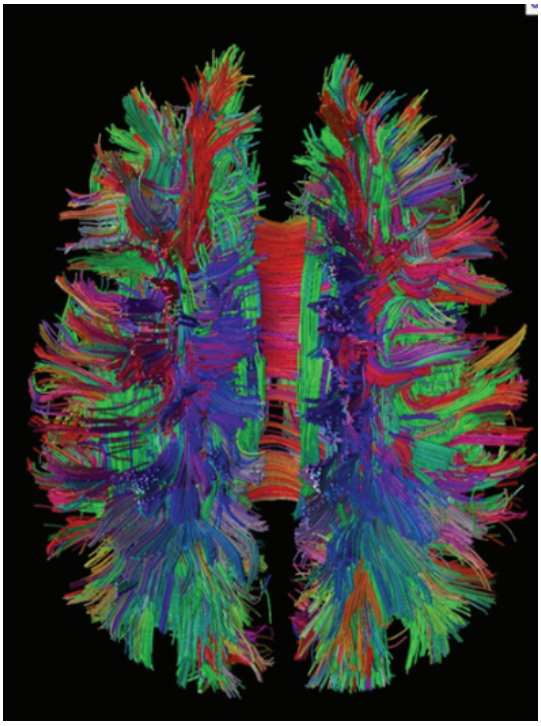
Keywords

connectomics, brain networks, therapeutic relationships, neuroplasticity, network medicine, social brain, diaschisis, hub vulnerability, precision medicine, neurosurgery



Modules, hubs and the topological characteristics of vulnerability and resilience (Fornito 15)

Citation: Ungar-Sargon J. Connectomics: Mapping the Brain's Complex Networks. Neurol Neurosci. 2025;6(8):040.



White matter fibre pathways of the brain as depicted with MR tractography
(Provided by Patric Hagmann, CHUV-UNIL, Lausanne, Switzerland)

Introduction

For much of modern neuroscience, the brain was understood primarily through localization of function: specific areas were thought to correspond to specific abilities or deficits. Broca's area meant language, the hippocampus meant memory, the occipital lobe meant vision [1]. While such localization has undeniable value, it has always risked oversimplifying a dynamic, interconnected system [2]. In recent decades, the field of connectomics has emerged to challenge and expand this view [3]. Connectomics seeks to map the brain not as a collection of isolated modules but as a complex network, where meaning arises from the patterns and strengths of interconnections [4]. This shift—from localization to connection—has changed how scientists, clinicians, and philosophers alike think about the human mind [5].

Theoretical Foundations of Connectomics

The term "connectome" was coined by Olaf Sporns in 2005, inspired by the Human Genome Project [6]. Just as the genome maps genetic information, the connectome seeks to map all neural connections in the brain, from synaptic micro-circuits to large-scale networks [7]. Theoretically, connectomics is grounded in network science: nodes (neurons or brain regions) are connected by edges (synapses, fiber tracts) [8]. Tools from graph theory allow neuroscientists to analyze properties like hubs, modules, and efficiency of information transfer [9].

This theoretical lens challenges the reductionist model of a brain as separate centers of activity. Instead, it emphasizes emergence: cognition, memory, and consciousness arise not from one site but from the orchestration of multiple interacting systems [10]. The "default mode network" (DMN), for example, is not a single anatomical site but a dynamic system of connections involved in self-referential thought and imagination [11].

The field operates across multiple spatial scales, each presenting unique technical challenges and insights [12]. At the microscale, researchers aim to map individual synaptic connections between neurons—the most detailed level of brain wiring. This requires imaging techniques with nanometer resolution capable of tracing neural processes across large tissue volumes [13]. The mesoscale focuses on connections between local neural populations and brain areas, examining how different cortical columns, nuclei, or functional regions communicate [14]. The macroscale level maps large-scale networks visible through neuroimaging techniques like diffusion tensor imaging (DTI) and functional magnetic resonance imaging (fMRI) [15].

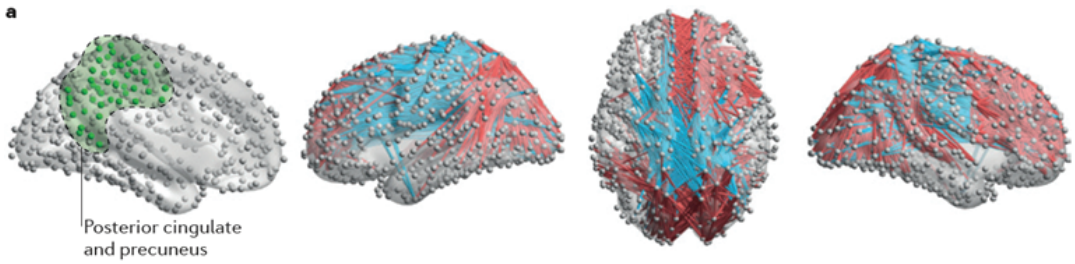
Network Principles and Brain Organization

Connectomics research has revealed fundamental organizing principles of brain networks that were not apparent from traditional anatomical studies [16]. Brain networks exhibit "small world" properties, combining local clustering of connections with long-range shortcuts that enable efficient information transfer across the entire brain [17]. This architecture balances the competing demands of functional specialization and global integration [18].

The discovery of highly connected "hub" regions that serve as critical nodes in brain networks has important implications for understanding both normal brain function and disease vulnerability [19]. These hubs often correspond to brain regions that are particularly susceptible to age-related changes and neurodegenerative diseases, suggesting that network topology influences disease progression patterns [20]. Brain networks also show hierarchical organization, with smaller modules embedded within larger systems [21]. This modular structure appears to support both the segregation of specialized functions and the integration necessary for complex cognitive tasks [22].

Connectomics and Brain Disorders: A Network Perspective

The ancient Roman physician Galen was one of the first to propose that pathology in one part of the nervous system



Network topology constrains the distributed effects of focal lesions on brain dynamics (Fornito 15)

could affect other regions when he posited that animal spirits could flow through interconnecting neural pathways [23]. This hypothesis was revisited nearly two millennia later by Brown-Séquard, who suggested that the effects of focal brain damage on remote regions resulted from actions at a distance [24]. von Monakow extended the concept, coining the term diaschisis (derived from Greek and meaning 'shocked throughout') to describe the depression of function that can arise in undamaged brain regions connected to a lesioned site [25].

At a similar time, Wernicke proposed an associative theory of brain function, in which higher-order cognitive processes arose from the integration of multiple, spatially distributed neural systems and in which disorders as diverse as aphasia and schizophrenia resulted from the disruption of specific associative pathways [26]. This work paved the way for Geschwind's introduction of the 'disconnexion syndrome' and the concomitant expansion of the range of clinical symptoms that may now be attributed to disordered brain connectivity [27].

Maladaptive Responses and Pathological Spread

Connectomics offers a powerful analytic framework for localizing pathology, tracking patterns of disease spread, and predicting which areas will be affected next [28]. However, simply tracking the spread of a disease will not necessarily elucidate the mechanisms through which this spread occurs. Such mechanisms may be construed as maladaptive, as they compound the degree of functional compromise that results from the insult [29].

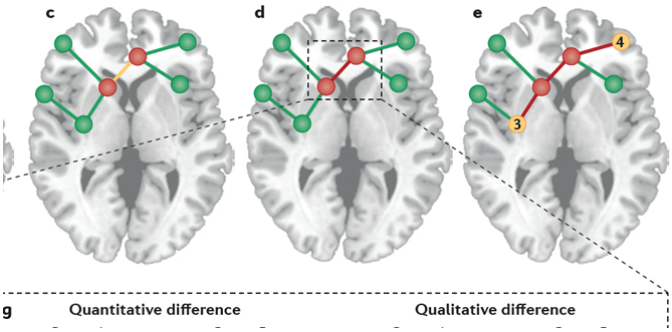
Diaschisis represents a temporary interruption of function in regions remote from an injured site [30]. Originally attributed to deafferentation of excitatory input to the remote area, diaschisis is now well-established, particularly following stroke [31]. It has been observed in the forebrain after damage to the brainstem or cerebellum, in cortical regions following subcortical infarction, and in contralesional cortex following focal cortical insult [32]. These distributed changes seem to be circuit-selective—lesions to either the fronto-parietal or the cingulo-opercular network affect connected areas within the same system but not the functions of the other network [33].

Studies of patients who have suffered stroke suggest that the severity of behavioral impairment following focal neural damage often correlates with the extent of activation and connectivity changes in regions remote from the injured site [34]. These associations between behavior and altered network functional connectivity occur even if anatomical connectivity between damaged and undamaged regions is intact [35], suggesting that a 'functional deafferentation' of remote sites may be sufficient to impair behavior [36].

Transneuronal degeneration represents a structural deterioration of areas remote from the initial insult, evolving over time and therefore requiring longitudinal characterization [37]. This can be either anterograde (damage of one neuron causes degeneration of its postsynaptic target) or retrograde (a presynaptic neuron deteriorates due to reduced trophic support from an injured postsynaptic target) [38]. The form of degeneration encompasses changes such as neuronal shrinkage, reductions in dendrite and synapse number, alterations of axonal myelin content and fiber number, and neuronal death [39].

Fast axonal transport contributes significantly to transneuronal degeneration [40]. Molecular motors continually shuttle organelles, lipids, mitochondria, neurotrophins and other molecules via microtubules and neurofilaments linking the soma and distal segments of the axon [41]. Pathology at the soma can disrupt anterograde transport of cargo necessary for axonal maintenance, while primary white matter pathology can inhibit retrograde transport of trophic factors essential for neuronal survival [42]. Transport mechanisms may also aid the suggested prion-like spread of tau and other pathologies in certain neurodegenerative diseases [43].

Dedifferentiation involves the diffuse, non-specific recruitment of brain regions to perform a task, thought to result from a breakdown of usually specialized and segregated neural activity [44]. This may be caused by aberrant neural plasticity or by focal cortical pathology that disrupts the balance between excitation and inhibition within discrete neural systems [45]. Another possible cause is the disruption of ascending neuromodulatory systems that tune the signal-to-noise ratio of neural information processing [46].



Network Topology and Disease Vulnerability

The brain's varied responses to insult are fundamentally constrained by connectome topology [47]. Not all brain regions are equal; rather, the functional impact of damage to any single network element strongly depends on the connection topology of that region [48]. Structural and functional brain networks are characterized by a heavy-tailed degree distribution—they have many low-degree nodes and a small number of putative hub nodes with very high degree [49]. Such networks are robust to random node failures but highly vulnerable to targeted hub attacks [50].

High-degree and topologically central hub regions are highly interconnected, forming a 'rich club'—a central core of hubs that facilitates efficient communication between disparate network elements [51]. These central hub nodes are concentrated in heteromodal association cortices, while primary sensory cortices tend to have low topological centrality [52]. Computational studies have shown that damage to highly central regions have a more diffuse effect on brain network structure and function than damage to topologically peripheral nodes [53].

Distinct hub types can be defined according to modular organization [54]. 'Provincial' hubs link primarily to other nodes in the same module and have an important role in functional specialization, whereas 'connector' hubs have links distributed across multiple different modules, thereby having a central role in functional integration [55]. Computational studies suggest that damage to connector hubs has a more widespread effect on network dynamics, whereas lesions to provincial hubs exert a more profound effect on local subsystems [56].

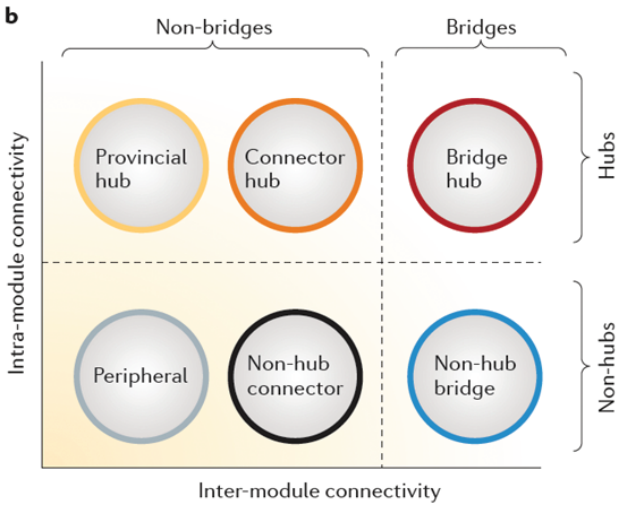
Adaptive Responses and Recovery Mechanisms

The brain can also respond to pathological perturbation in adaptive ways to maintain homeostasis and performance [57].

Compensation involves increases in activity or functional connectivity following pathological insult that preserve behavioral output [58]. In patients with stroke, focal ischemic insult often results in extensive recruitment of unaffected, remote brain areas [59]. The extent of focal neural damage and severity of behavioral impairment correlate with greater compensatory recruitment, functional reorganization, and altered functional connectivity of remote areas [60].

Degeneracy represents the capacity of structurally distinct elements of a system to carry out the same function—the ability of distinct neuronal systems to make overlapping contributions to the same output, offering both functional adaptability and robustness to damage [61]. Degeneracy provides the necessary foundation for compensation, as compensatory activity is simply not possible if other neural systems cannot assume the functions of a compromised network [62].

Neural reserve refers to the amount of remaining intact brain tissue that can still carry out a given task [63]. Generally, a brain with high neural reserve will be able to withstand greater damage before cognitive or behavioral deficits manifest [64]. Although degeneracy, compensation and reserve are closely related, degeneracy does not necessarily imply that compensatory activity will occur following an insult [65].



Clinical Applications and Medical Relevance

Connectomics holds tremendous promise for advancing medical understanding and treatment of brain disorders [66]. Many neurological and psychiatric conditions are increasingly understood as "connectome disorders"—diseases arising from disrupted patterns of brain connectivity rather than damage to specific brain regions [67].

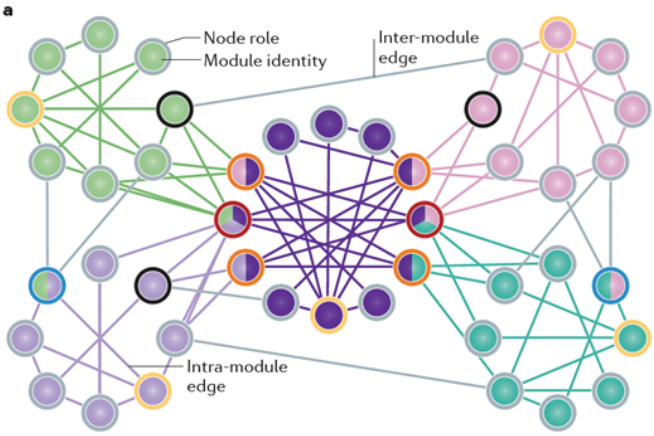
In epilepsy, connectomic approaches are revolutionizing surgical planning by mapping the networks involved in seizure generation and propagation [68]. Advanced tractography can identify critical white matter pathways that must be preserved during surgery, while network analysis helps predict how removing brain tissue might affect overall brain function [69]. Rather than targeting tissue based on anatomical landmarks alone, surgeons now consider how removing a tumor or hematoma might disrupt the functional network, leading to more precise surgical interventions with better outcomes and fewer complications [70].

The development of minimally invasive surgical techniques, such as those tested in the ENRICH trial for hemorrhagic stroke, relies heavily on detailed knowledge of white matter pathways to minimize damage to surrounding brain tissue [71]. These approaches use advanced imaging, connectomics, and trajectory planning to target pathology with reduced collateral damage [72].

Psychiatric disorders like depression, schizophrenia, and autism spectrum disorders show characteristic patterns of altered connectivity that connectomics is beginning to decipher [73]. These insights are leading to new therapeutic approaches, including targeted brain stimulation techniques that aim to restore normal connectivity patterns [74].

Stroke research has been particularly transformed by connectomic approaches [75]. Understanding how stroke damage affects not just the immediate injury site, but entire brain networks has led to better predictions of recovery and more targeted rehabilitation strategies [76]. Damage to one site can cause cascading effects throughout connected networks, and behavioral impairments often arise from how the insult affects distributed neural dynamics rather than its impact on the lesioned site alone [77].

Neurodegenerative diseases provide compelling examples of how pathology spreads through connected neural systems [78]. In Alzheimer disease, the accumulation of amyloid-β in specific brain regions reduces their functional connectivity with other areas and may cause hypometabolism in distal sites, rendering them targets for disease propagation [79]. The spatial distribution of grey-matter atrophy in patients with various neurodegenerative diseases corresponds closely with functionally and structurally connected networks, suggesting that degeneration occurs within connected neural systems [80].



Multi-Scale Mapping Approaches

Mapping the connectome requires sophisticated multi-scale approaches [81]. Microscale connectomics uses electron microscopy to reconstruct every synapse in small organisms, achieving the complete wiring of C. elegans with its 302 neurons and approximately 7,000 connections [82]. Mesoscale connectomics employs viral tracers and advanced imaging to trace circuits in animal models [83], while macroscale connectomics uses diffusion tensor imaging (DTI) and functional MRI (fMRI) to infer networks of connectivity in the living human brain [84].

Recent advances have yielded detailed maps through projects like the Human Connectome Project, which combines MRI

techniques with sophisticated computational modeling [85]. These tools allow researchers to identify hubs of connectivity that act as critical nodes linking different regions [86]. Machine learning and artificial intelligence are becoming increasingly central to connectomics research, with deep learning algorithms now automatically identifying and tracing neural processes in electron microscopy images [87].

Computational Challenges

The scale of data generated by connectomics research presents unprecedented computational challenges [88]. A complete human brain connectome at the microscale would contain trillions of connections, requiring entirely new approaches to data storage, analysis, and visualization [89]. Current efforts focus on developing standardized data formats, efficient algorithms for network analysis, and collaborative platforms for sharing massive datasets across research groups [90].

Graph theory methods provide essential tools for analyzing brain networks, offering mathematical frameworks for quantifying network properties such as clustering, modularity, and centrality. Complex network measures of brain connectivity require careful interpretation and understanding of their computational foundations. Fundamentals of brain network analysis emphasize the importance of methodological rigor in network construction, analysis, and interpretation.

Integration across different scales and data types remains a major challenge [91]. Combining microscale circuit details with macroscale network properties requires sophisticated computational models that can bridge these levels of organization [92]. The development of multi-scale brain models that incorporate detailed connectivity information represents one of the most important frontiers in computational neuroscience [93].

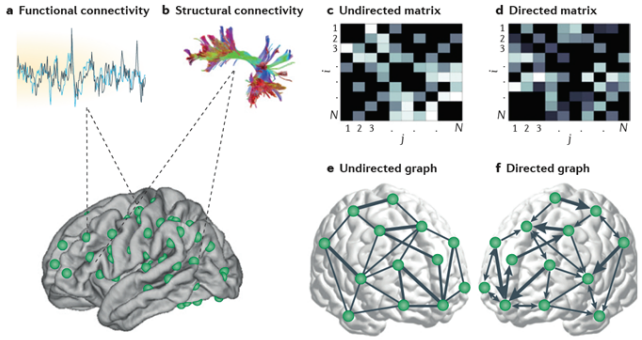
Timing and Development

The age at which an insult to the brain occurs critically influences the outcome of injury [94]. The enhanced plasticity of the developing brain sometimes affords greater capacity for recovery from injuries sustained earlier in life, as exemplified by children with congenital left hemisphere damage who may show age-appropriate language development [95]. However, certain types of insult occurring prenatally or in the first few years of life can result in more severe functional impairment than later injuries [96].

This variability may be partly explained by the timing of insult relative to developmentally critical periods—highly regulated, circuit-specific maturational periods characterized by exquisite sensitivity to environmental inputs [97]. These periods coincide with activity-dependent elimination of excess synapses and consolidation of long-range axonal projections [98]. The developmental trajectories of topological modifications are system-specific: sensorimotor and limbic systems develop adult-like topological properties by late childhood, whereas associative areas continue to mature throughout adolescence [99].

Large-scale longitudinal studies tracking the progression of brain network changes over time are crucial for elucidating how pathology dynamically evolves in the brain [100]. The progression of neurodegenerative disorders follows predictable patterns, with the largest declines in function occurring when disease impinges on hub regions [101]. This insight has enabled the development of computational models that can predict disease spread based on network topology—specifically, the spatial distribution of grey-matter atrophy in Alzheimer disease and frontotemporal dementia can be reproduced by simple computational models of disease diffusion simulated on empirically derived connectomic maps [102].

Large-scale longitudinal studies tracking the progression of brain network changes over time are crucial for elucidating how pathology dynamically evolves in the brain [100]. The progression of neurodegenerative disorders follows predictable patterns, with the largest declines in function occurring when disease impinges on hub regions [101]. This insight has enabled the development of computational models that can predict disease spread based on network topology—specifically, the spatial distribution of grey-matter atrophy in Alzheimer disease and frontotemporal dementia can be reproduced by simple computational models of disease diffusion simulated on empirically derived connectomic maps [102].



Diffusion tractography
An MRI technique for reconstructing large-scale white-matter fibres based on the preferential diffusion of water along the axes of these fibres (Fornito 15)

From Description to Prediction

Understanding connectome topology enables the development of formal, computational models that allow testable predictions about the specific profile of neural or behavioral changes expected following an insult [103]. Knowledge of network topology allows not only description of pathological processes but also generation of predictive models of disease spread and functional consequences [104].

The centrality of a node fundamentally influences the impact of damage to that network element [105]. High-degree and topologically central hub regions, when damaged, affect a disproportionate number of connections and can result in rapid network fragmentation [106]. These central hubs can act as conduits for rapid spread and progression of transneuronal degeneration, with greater functional compromise expected once disease processes encroach on hub nodes [107].

The differential involvement of topologically central versus peripheral regions at varying stages of illness may explain the punctuated and nonlinear pattern of functional decline often associated with neurodegenerative disorders [108]. Several converging lines of evidence indicate that central hub regions have increased susceptibility to brain disease effects, with many disorders disproportionately affecting hub regions [109].

Prognostic Applications

The topological dimensions of centrality and degeneracy define a parameter space enabling testable predictions regarding the extent of functional compromise and prognosis

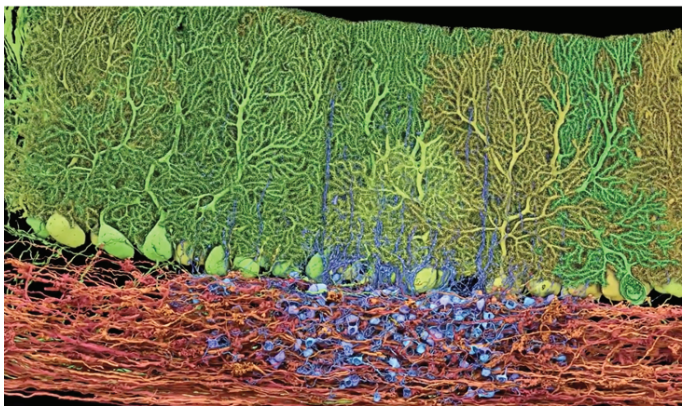
for recovery following insult [110]. The capacity for resilience, compensation, and functional restitution is closely tied to network degeneracy [111]. Areas embedded within specific modules display higher topological degeneracy as they form part of tightly interconnected 'cliques' of nodes [112], while bridge nodes—regions involved in multiple modules—act as 'convergence zones' allowing integration of specialized processes between distinct neural systems [113].

Recovery of function may be more probable following damage to regions with high clustering coefficients or high degrees of topological overlap with other nodes, and/or to regions deeply embedded within modules [114]. Conversely, recovery is less likely following damage to topologically central areas or to regions that support degeneracy, such as bridge nodes [115].

The Social Brain as Network Architecture

Connectomics reveals that humans are fundamentally social beings at the neural level [116]. The brain's architecture includes specialized networks for social cognition, empathy, theory of mind, and interpersonal communication [117]. The default mode network, heavily implicated in self-referential thinking, is also central to understanding others and maintaining social relationships [118]. Mirror neuron systems create literal neural connections between individuals during social interaction, suggesting that the boundaries between self and other are more porous than traditionally conceived [119].

This network perspective transforms how we understand the patient-doctor relationship from a simple information exchange to a complex neurobiological encounter where two brains literally influence each other's connectivity patterns [120]. The therapeutic relationship becomes a space where neural networks interact, synchronize, and potentially reorganize [121].



https://hms.harvard.edu/news/new-field-neuroscience-aims-map-connections-brain

The Neurobiology of Therapeutic Presence

Connectomic research demonstrates that sustained, empathetic attention from another person can activate neuroplasticity mechanisms in the patient's brain [122]. The clinician's presence and emotional regulation can help stabilize dysregulated networks in the patient through multiple mechanisms [123]:

Neural Synchronization: During meaningful therapeutic encounters, brain rhythms between patient and clinician can synchronize, creating states of shared neural coherence that facilitate healing and insight [124].

Stress Network Regulation: A calm, attuned clinician can help down-regulate overactive stress networks in the patient's brain through co-regulation mechanisms that operate below conscious awareness [125].

Narrative Network Integration: The process of constructing coherent narratives about illness and recovery literally reshapes connectivity between memory, emotion [126]

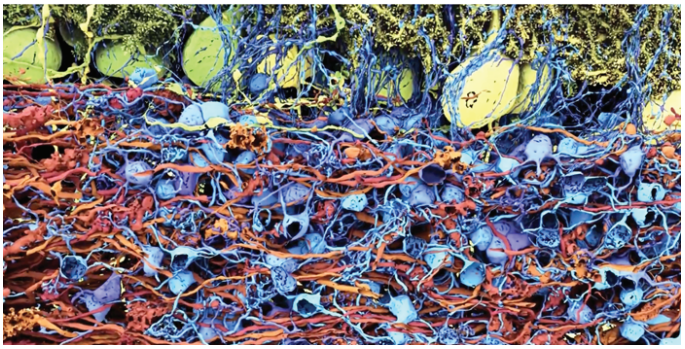
Trauma, Connection, and Network Disruption

Connectomics provides a neurobiological framework for understanding how relational trauma disrupts brain networks and how healing relationships can restore connectivity [127]:

Attachment and Network Development: Early attachment relationships literally wire the developing brain's social networks [128]. Secure attachment patterns create resilient network architectures, while disrupted attachment can lead to dysregulated connectivity patterns that manifest as mental health challenges [129].

Interpersonal Trauma and Network Fragmentation: Trauma often involves disconnection—both from others and from internal states [130]. Neurobiologically, trauma can fragment networks that normally integrate memory, emotion, and self-awareness, leading to symptoms like dissociation, emotional dysregulation, and interpersonal difficulties [131].

Therapeutic Relationship as Network Repair: The patient-clinician relationship provides a context for rebuilding damaged connectivity patterns [132]. Through sustained, attuned connection, new network patterns can emerge that support integration and resilience [133].



The Limits and Risks of Network Reductionism

While connectomics offers valuable insights into the neurobiology of relationships, several important limitations and risks must be acknowledged [134]:

The Measurement Problem: Current neuroimaging technologies can only capture crude approximations of the rich, dynamic processes occurring during human connection [135]. The most profound aspects of therapeutic relationships—meaning, hope, spiritual connection—may not be reducible to network properties [136].

Cultural and Individual Variation: Brain connectivity patterns vary significantly across cultures and individuals [137]. Imposing universal network models on diverse populations risks perpetuating cultural biases and missing important sources of resilience and healing wisdom [138].

The Risk of Technological Mediation: Over-reliance on brain imaging and network analysis could potentially distance clinicians from the direct, embodied experience of being present with patients [139]. The healing power of human connection may be diminished if it becomes overly medicalized or technologized [140].

Connectomics challenges traditional diagnostic categories

by revealing mental health as fundamentally about network connectivity rather than discrete disorders [141]:

Dimensional Rather Than Categorical: Instead of asking "Does this patient have depression?" clinicians might ask "How are this person's networks for emotional regulation, self-reflection, and social connection functioning?"[142]

Contextual and Relational: Mental health symptoms emerge from the interaction between individual network vulnerabilities and environmental stressors, including relationship patterns [143]. Treatment focuses on strengthening networks within supportive relational contexts [144].

Resilience and Network Flexibility: Mental health becomes less about the absence of problems and more about network flexibility—the ability to adaptively reorganize connectivity patterns in response to challenges [145].

Implications for Medical Education and Training

Connectomic insights suggest significant changes needed in how healthcare providers are trained [146]:

Relationship Skills as Clinical Competencies: If therapeutic relationships literally rewire brain networks, then relationship skills become core clinical competencies requiring systematic training and assessment [147].

Self-Awareness and Clinician Networks: Healthcare providers need awareness of their own network patterns and how these influence patient interactions [148]. Practices like mindfulness, self-reflection, and personal therapy become professional development rather than optional self-care [149].

Interdisciplinary Integration: Understanding patients as embodied beings with complex social networks requires integration across medical specialties, mental health disciplines, and social services [150].

Connectomics points toward several emerging directions for therapeutic relationships [151]:

Precision Relationship Medicine: Understanding individual connectivity patterns might inform how to tailor therapeutic approaches to specific patients [152]. Some individuals might benefit from highly structured, cognitive approaches while others need more embodied, emotionally-focused interventions [153].

Network-Informed Communities: Treatment approaches might increasingly focus on strengthening social networks and community connections as neurobiological interventions rather than simply social support [154].

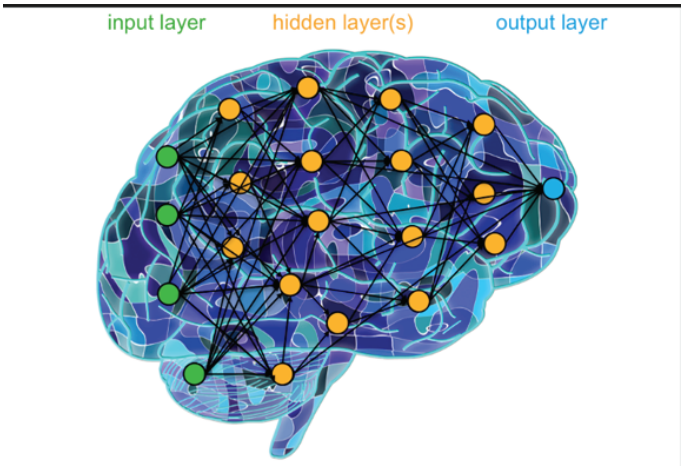
Technology-Augmented Connection: While preserving the primacy of human connection, technology might enhance therapeutic relationships through real-time feedback about neural synchronization, stress states, or network activation patterns [155].

Several ethical challenges emerge as connectomics influences therapeutic relationships [156]:

Privacy and Neural Intimacy: If brain connectivity patterns reveal deep aspects of personality, relationships, and vulnerability, how do we protect patient privacy while using this information therapeutically? [157]

Power Dynamics and Neural Influence: If clinicians can influence patient brain networks through their presence and attention, this raises questions about consent, manipulation, and the appropriate use of this influence[158].

Cultural Sensitivity and Network Diversity: How do we



honor diverse cultural approaches to healing and relationship while integrating neuroscientific insights? [159]

Technological Innovations

Complete microscale connectomes of mammalian brain regions are becoming feasible, promising unprecedented insights into computational principles underlying brain function [160]. The integration of connectomic data with functional measurements of neural activity will enable researchers to understand how anatomical connectivity gives rise to patterns of neural activity underlying behavior and cognition [161].

Dynamic connectomics—studying how neural connections change over time—represents an emerging frontier [162]. Understanding how neural circuits are modified by experience, development, and disease progression will be crucial for developing effective interventions [163]. Advanced imaging techniques are beginning to make it possible to track connectivity changes in living brains over extended periods [164].

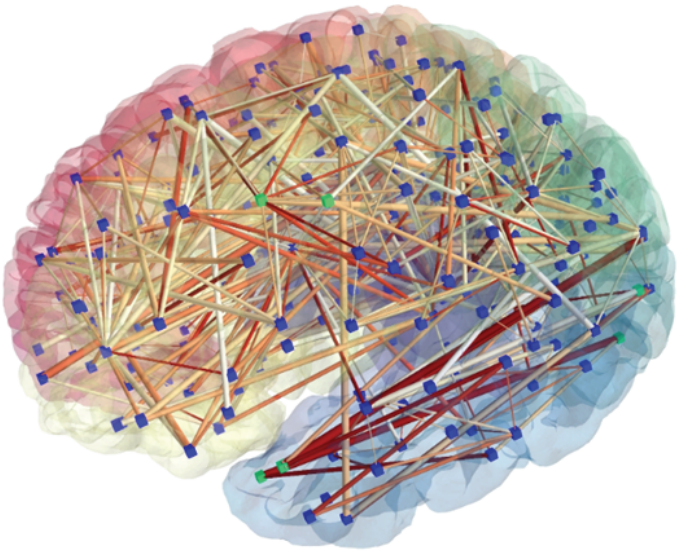
The therapeutic implications of connectomics continue to expand [165]. Precision medicine approaches based on individual connectivity profiles may enable personalized treatments for brain disorders [166]. Brain stimulation techniques guided by connectomic principles are showing promise for treating depression, chronic pain, and other conditions [167]. The development of brain-computer interfaces also relies heavily on understanding connectivity patterns that link neural activity to intended movements or thoughts [168].

Improvements in imaging technologies and network-mapping techniques will enhance precision in tracking pathological processes [169]. For structural connectivity, this involves developing more accurate fiber-reconstruction algorithms, measures with clear physiological interpretation, and capacity to resolve projection sources and targets [170]. For functional analysis, scaling effective connectivity models to deal with large-scale brain networks and diverse experimental paradigms will be particularly important [171].

Multi-modal validation of imaging measures, combined with detailed biophysical models, may help mitigate limitations of existing approaches [172]. Open-source platforms for integrating computational modeling with empirical data offer considerable promise for researchers and clinicians alike [173].

Consciousness and Identity

Connectomics reframes fundamental questions about consciousness, cognition, and human nature [174]. Some theorists propose that consciousness itself may be an emergent



property of large-scale network integration [175]. In this view, it is the pattern and coherence of connections—not a single cortical area—that gives rise to awareness [176]. This perspective challenges traditional notions of localized mental functions and suggests that understanding the mind requires comprehending the complex interplay of distributed neural networks [177].

If individuality is partly written in the connectome, then brain mapping raises significant questions about privacy, prediction of behavior, and even the possibility of "connectomic identity"[178]. While gross anatomy is fairly consistent across individuals, connectivity patterns vary significantly, providing a neural basis for differences in cognition, personality, and vulnerability to disease [179]. This raises important considerations about the potential misuse of connectomic information and the need for appropriate ethical frameworks governing brain mapping research [180].

Despite its promise, connectomics faces major challenges [181]. Complete mapping of a human connectome at synaptic resolution would generate exabytes of data, far beyond current analytic capacity [182]. Moreover, correlation is not causation: just because two regions are connected does not mean we fully understand their functional role [183]. Critics warn against a new reductionism—believing that once we map every connection, we will "explain" the mind [184]. The human experience involves embodiment, environment, and culture, which exceed neural maps [185].

Additional challenges include the development of standardized approaches for network analysis, the integration of data across multiple scales and modalities, and the translation of research findings into clinically useful tools [186]. The field must also address questions about the stability and reliability of connectivity measures, the optimal approaches for network parcellation and analysis, and the relationship between structural and functional connectivity patterns [187].

Conclusion

Connectomics represents both a technical revolution and a conceptual reorientation in neuroscience that extends far beyond the laboratory into the heart of human relationships and healing [188]. By shifting focus from isolated regions to integrated networks, it provides a richer and more realistic view of the brain as a complex, adaptive, and fundamentally social system [189]. For clinicians, it opens possibilities for new diagnostics, surgical precision, and therapeutic interventions, while simultaneously validating ancient wisdom about the healing power of human connection [190].

The field has already demonstrated remarkable success, from the complete connectome of C. elegans to detailed circuit maps in mammalian brains and large-scale human brain mapping projects [191]. These achievements have revealed fundamental principles of network organization that apply not only to individual brains but to the connections between brains during social interaction [192].

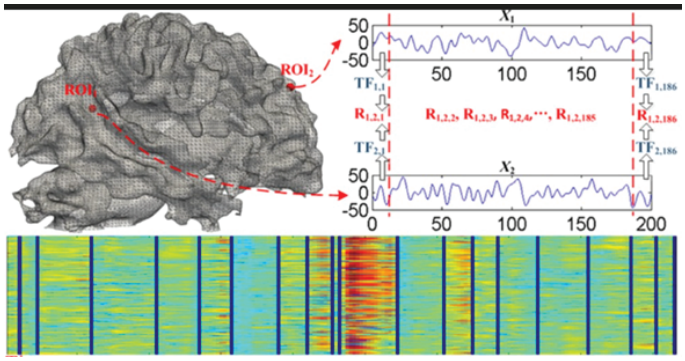
For medicine, connectomics has transformed understanding of neurological and psychiatric disorders as network phenomena rather than localized pathologies [193]. This perspective has led to new therapeutic approaches that recognize the patient-clinician relationship as a neurobiological intervention capable of reshaping brain connectivity patterns [194]. The development of minimally invasive procedures, network-guided brain stimulation, and personalized treatment approaches exemplifies the clinical translation of connectomic principles while preserving the centrality of human connection [195].

Perhaps most profoundly, connectomics is beginning to inform our understanding of consciousness, relationships, and human nature [196]. By revealing the detailed wiring patterns that distinguish human brains from other species and that vary among individuals, connectomics validates both human uniqueness and the fundamental interdependence of all human beings [197]. The discovery that our brains are literally wired for connection challenges individualistic models of health and illness, pointing toward more relational and community-based approaches to healing [198].

However, this scientific revolution also requires wisdom and humility [199]. While brain networks provide insights into the mechanisms of healing relationships, they cannot capture the full mystery and complexity of human connection [200]. The most profound aspects of therapeutic encounters—hope, meaning, spiritual connection, and love—may always transcend our ability to measure them [201]. The challenge is to integrate connectomic insights in ways that enhance rather than diminish the humanity of both patients and healers [202].

The journey from simple network maps to understanding the full complexity of brain connectivity and human relationships represents one of the most ambitious scientific endeavors ever undertaken [203]. As imaging technologies continue to improve and computational tools become more sophisticated, connectomics will likely play an increasingly central role in neuroscience research and clinical practice [204]. Yet the ultimate goal is not merely technical mastery but a deeper understanding of how brain connectivity gives rise to mind, relationship, and the capacity for healing [205].

The integration of connectomics with other fields—from genetics to artificial intelligence, from contemplative traditions to social justice movements—promises even greater advances in understanding brain organization and human flourishing [206]. As we continue to map the brain's wiring diagram, we move closer to answering some of humanity's most profound questions about consciousness, connection, and what makes us who we are [207]. The shift from localization to connection has not only changed neuroscience but has begun to transform our understanding of what it means to be human in relationship with others [208].



Addendum: Connectomics Applications in PTSD

Post-traumatic stress disorder (PTSD) exemplifies how psychological trauma manifests as disrupted brain network connectivity rather than focal brain damage [209]. Connectomic studies reveal that PTSD involves dysregulation across multiple neural networks, particularly those governing emotional regulation, memory processing, and self-referential cognition [210]. The disorder demonstrates how severe stress can fragment normally integrated brain networks, leading to the characteristic symptoms of hypervigilance, intrusive memories, emotional numbing, and dissociation [211]..

Key Network Alterations in PTSD

Default Mode Network Disruption: The default mode network, crucial for self-referential processing and autobiographical memory, shows altered connectivity patterns in PTSD patients [212]. This disruption correlates with difficulties in narrative coherence and self-integration that characterize the disorder [213]. Hyperconnectivity within posterior regions and reduced connectivity with prefrontal areas may underlie the intrusive re-experiencing of traumatic memories [214].

Salience Network Hyperactivation: The salience network, responsible for detecting and orienting to relevant stimuli, becomes hyperactive in PTSD [215]. This leads to enhanced threat detection but impaired ability to distinguish between actual threats and benign stimuli, contributing to hypervigilance and exaggerated startle responses [216].

Executive Control Network Impairment: Reduced connectivity within the executive control network, particularly involving the prefrontal cortex, compromises top-down regulation of emotional responses [217]. This network dysfunction underlies difficulties with emotional regulation, impulse control, and cognitive flexibility observed in PTSD [218].

Fear Network Dysregulation: Altered connectivity between the amygdala, hippocampus, and prefrontal cortex disrupts fear learning and extinction processes [219]. Hyperconnectivity between amygdala and sensory regions may contribute to heightened emotional reactivity, while reduced prefrontal-amygdala connectivity impairs fear extinction and emotional regulation [220].

Developmental and Attachment Perspectives

Connectomics provides insights into how early trauma affects developing neural networks. Childhood trauma can disrupt the formation of secure attachment networks, leading to lifelong vulnerabilities in emotional regulation and interpersonal relationships [221]. The developing brain's heightened plasticity means that early trauma can have particularly profound effects on network architecture, establishing patterns of dysregulation that persist into adulthood [222].

Studies of complex PTSD, often resulting from chronic childhood trauma, reveal more extensive network disruptions compared to adult-onset PTSD [223]. These include alterations in networks governing self-concept, emotional regulation, and interpersonal functioning, reflecting the broader developmental impact of early traumatic experiences [224].

Neurobiological Mechanisms of Network Disruption

Stress Response System Dysregulation: Chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis in trauma exposure leads to sustained elevation of stress hormones, which can damage neural connections and disrupt network integrity [225]. This particularly affects memory-related networks, contributing to the fragmented and intrusive nature of traumatic memories [226].

Inflammatory Processes: Trauma exposure activates neuroinflammatory processes that can alter synaptic function and network connectivity [227]. Elevated inflammatory markers in PTSD patients correlate with network disruptions, particularly in circuits governing mood regulation and cognitive function [228].

Epigenetic Modifications: Trauma can induce epigenetic changes that alter gene expression patterns affecting synaptic plasticity and network development [229]. These modifications may contribute to the intergenerational transmission of trauma-related network vulnerabilities [230].

Therapeutic Implications

Network-Informed Psychotherapy: Understanding PTSD as a network disorder informs therapeutic approaches that target specific connectivity patterns. Therapies that promote integration between fragmented networks—such as EMDR, somatic therapies, and narrative therapy—may be particularly effective [231].

Neurofeedback and Brain Stimulation: Real-time feedback about network activity through neurofeedback can help patients learn to regulate dysregulated networks [232]. Targeted brain stimulation techniques, guided by connectomic mapping, show promise for normalizing specific network dysfunctions in PTSD [233].

Mindfulness and Meditation: Contemplative practices that enhance interoceptive awareness and present-moment attention can strengthen networks involved in self-regulation and reduce hyperactivation of threat-detection systems [234]. Neuroimaging studies demonstrate that mindfulness training can normalize connectivity patterns in PTSD patients [235].

Therapeutic Relationships and Network Repair

The therapeutic relationship becomes particularly crucial in PTSD treatment when viewed through a connectomics lens. Trauma fundamentally involves disconnection—from others, from one's body, and from one's authentic self [236]. The patient-therapist relationship provides a context for rebuilding damaged connectivity patterns through several mechanisms:

Co-regulation and Neural Synchronization: A regulated, attuned therapist can help stabilize the patient's dysregulated nervous system through co-regulation processes [237]. This may involve neural synchronization between patient and therapist, creating states of safety that allow network reorganization [238].

Attachment Repair: For patients with early trauma, the therapeutic relationship can provide a corrective attachment experience that helps rewire attachment-related networks [239]. Secure therapeutic attachment can gradually normalize patterns of interpersonal neurobiology [240].

Narrative Integration: The process of constructing coherent narratives about traumatic experiences within a safe therapeutic relationship promotes integration between fragmented memory networks [241]. This narrative work literally rewires connections between memory, emotion, and self-referential brain regions [242].

Challenges and Limitations

Individual Variation: PTSD presents with considerable heterogeneity in symptoms and network patterns, making it challenging to develop universal connectomic models [243]. Individual differences in trauma type, developmental timing, genetic vulnerability, and resilience factors all influence network dysfunction patterns [244].

Measurement Limitations: Current neuroimaging techniques may not capture the full complexity of trauma-related network changes, particularly subtle alterations in network dynamics that occur during triggered states [245]. The episodic nature of PTSD symptoms also makes it difficult to capture representative network states [246].

Treatment Complexity: While connectomics provides valuable insights, PTSD treatment remains complex and multifaceted. Network-based approaches must be integrated with understanding of psychological, social, and cultural factors that influence trauma and recovery [247].

Future Directions

Precision Medicine Approaches: Individual connectomic profiles may eventually guide personalized treatment selection, determining which patients are most likely to benefit from specific therapeutic approaches [248]. This could improve treatment outcomes and reduce the trial-and-error approach often required in PTSD treatment [249].

Network-Based Biomarkers: Connectivity patterns may serve as objective biomarkers for PTSD diagnosis, severity assessment, and treatment monitoring [250]. This could complement clinical assessment and provide more precise measurement of treatment progress [251].

Prevention and Early Intervention: Understanding how trauma disrupts developing networks could inform prevention strategies and early interventions that protect network integrity or promote resilience in at-risk populations [252].

The application of connectomics to PTSD demonstrates how network neuroscience can illuminate the neurobiological basis of psychological trauma while validating the central importance of therapeutic relationships in healing. This approach bridges the technical precision of neuroscience with the relational wisdom of trauma therapy, suggesting that effective treatment must address both network dysfunction and the fundamental human need for safety, connection, and meaning [253].

References

1. Broca P. Perte de la parole: ramollissement chronique et destruction partielle du lobe anterieur gauche du cerveau. Bulletins Soc Anthropolgie. 1861; 2:235-238.
2. Mesulam MM. Large-scale neurocognitive networks and distributed processing for attention, language, and memory. Ann Neurol. 1990;28(5):597-613.
3. Sporns O. The human connectome: a structural description of the human brain. PLoS Comput Biol. 2005;1(4): e42.
4. Catani M, ffytche DH. The rises and falls of disconnection syndromes. Brain. 2005;128(10):2224-2239.
5. Fornito A, Zalesky A, Breakspear M. The connectomics of brain disorders. Nat Rev Neurosci. 2015;16(3):159-172.
6. Sporns O. The human connectome: a structural description of the human brain. PLoS Comput Biol. 2005;1(4): e42.
7. Van Essen DC, Ugurbil K. The future of the human connectome. NeuroImage. 2012;62(2):1299-1310.
8. Bullmore E, Sporns O. Complex brain networks: graph theoretical analysis of structural and functional systems. Nat Rev Neurosci. 2009;10(3):186-198.
9. Rubinov M, Sporns O. Complex network measures of brain connectivity uses and interpretations. NeuroImage. 2010;52(3):1059-1069.
10. Tononi G, Sporns O, Edelman GM. A measure for brain complexity: relating functional segregation and integration in the nervous system. Proc Natl Acad Sci USA. 1994;91(11):5033-5037.
11. Buckner RL, Andrews-Hanna JR, Schacter DL. The brain's default network: anatomy, function, and relevance to disease. Ann N Y Acad Sci. 2008; 1124:1-38.
12. Bohland JW, Wu C, Barbas H, et al. A proposal for a coordinated effort for the determination of brainwide neuroanatomical connectivity in model organisms at a mesoscopic scale. PLoS Comput Biol. 2009;5(3): e1000334.
13. White JG, Southgate E, Thomson JN, Brenner S. The structure of the nervous system of the nematode Caenorhabditis elegans. Phil Trans R Soc Lond B. 1986;314(1165):1-340.
14. Oh SW, Harris JA, Ng L, et al. A mesoscale connectome of the mouse brain. Nature. 2014;508(7495):207-214.
15. Fornito A, Zalesky A, Breakspear M. Graph analysis of the human connectome: promise, progress, and pitfalls. NeuroImage. 2013; 80:426-444.
16. Bassett DS, Bullmore E. Small-world brain networks. Neuroscientist. 2006;12(6):512-523.
17. Watts DJ, Strogatz SH. Collective dynamics of 'small world' networks. Nature. 1998;393(6684):440-442.
18. Achard S, Bullmore E. Efficiency and cost of economical brain functional networks. PLoS Comput Biol. 2007;3(2): e17.
19. van den Heuvel MP, Sporns O. Rich-club organization of the human connectome. J Neurosci. 2011;31(44):15775-15786.
20. Buckner RL, Sepulcre J, Talukdar T, et al. Cortical hubs revealed by intrinsic functional connectivity: mapping, assessment of stability, and relation to Alzheimer's disease. J Neurosci. 2009;29(6):1860-1873.
21. Meunier D, Lambiotte R, Fornito A, Ersche KD, Bullmore ET. Hierarchical modularity in human brain functional networks. Front Neuroinform. 2009; 3:37.
22. Sporns O, Betzel RF. Modular brain networks. Annu Rev Psychol. 2016; 67:613-640.
23. Siegel RE. Galen on the Affected Parts. Basel: Karger; 1976.
24. Koehler PJ. Brown-Séquard and cerebral localization as illustrated by his ideas on aphasia. J Hist Neurosci. 1996;5(1):26-33.
25. Finger S, Koehler PJ, Jagella C. The von Monakow concept of diaschisis: origins and perspectives. Arch Neurol. 2004;61(2):283-288.
26. Wernicke C. Some new studies on aphasia. Fortschr Med. 1885; 3:824-830.
27. Geschwind N. Disconnexion syndromes in animals and man.

Brain. 1965;88(2):237-294.
28. Alstott J, Breakspear M, Hagmann P, Cammoun L, Sporns O. Modeling the impact of lesions in the human brain. PLoS Comput Biol. 2009;5(6): e1000408.
29. Honey CJ, Sporns O. Dynamical consequences of lesions in cortical networks. Hum Brain Mapp. 2008;29(7):802-809.
30. Feeney DM, Baron JC. Diaschisis. Stroke. 1986;17(5):817-830.
31. Carrera E, Tononi G. Diaschisis: past, present, future. Brain. 2014;137(Pt 9):2408-2422.
32. Rehme AK, Grefkes C. Cerebral network disorders after stroke: evidence from imaging-based connectivity analyses of active and resting brain states in humans. J Physiol. 2013;591(1):17-31.
33. Nomura EM, Gratton C, Visser RM, et al. Double dissociation of two cognitive control networks in patients with focal brain lesions. Proc Natl Acad Sci USA. 2010;107(26):12017-12022.
34. He BJ, Snyder AZ, Vincent JL, et al. Breakdown of functional connectivity in frontoparietal networks underlies behavioral deficits in spatial neglect. Neuron. 2007;53(6):905-918.
35. van Meer MPA, van der Marel K, Wang K, et al. Recovery of sensorimotor function after experimental stroke correlates with restoration of resting-state interhemispheric functional connectivity. J Neurosci. 2010;30(11):3964-3972.
36. Carter AR, Astafiev SV, Lang CE, et al. Resting interhemispheric functional magnetic resonance imaging connectivity predicts performance after stroke. Ann Neurol. 2010;67(3):365-375.
37. Cowan WM. Contemporary Research Methods in Neuroanatomy. New York: Springer; 1970.
38. DeGiorgio LA, Dibinis C, Milner TA, Saji M, Volpe BT. Histological and temporal characteristics of nigral transneuronal degeneration after striatal injury. Brain Res. 1998;795(1-2):1-9.
39. Deller T, Del Turco D, Rappert A, Bechmann I. The dentate gyrus and neuronal plasticity in the adult brain. In: Scharfman HE, editor. The Dentate Gyrus: A Comprehensive Guide to Structure, Function, and Clinical Implications. Amsterdam: Elsevier; 2007. p. 501-528.
40. Hirokawa N, Niwa S, Tanaka Y. Molecular motors in neurons: transport mechanisms and roles in brain function, development, and disease. Neuron. 2010;68(4):610-638.
41. Perlson E, Maday S, Fu MM, Moughamian AJ, Holzbaur ELF. Retrograde axonal transport: pathways to cell death? Trends Neurosci. 2010;33(7):335-344.
42. Nave KA. Myelination and the trophic support of long axons. Nat Rev Neurosci. 2010;11(4):275-283.
43. Frost B, Diamond MI. Prion-like mechanisms in neurodegenerative diseases. Nat Rev Neurosci. 2010;11(3):155-159.
44. Li SC, Lindenberger U, Sikström S. Aging cognition: from neuromodulation to representation. Trends Cogn Sci. 2001;5(11):479-486.
45. Rehme AK, Eickhoff SB, Wang LE, Fink GR, Grefkes C. Dynamic causal modeling of cortical activity from the acute to the chronic stage after stroke. NeuroImage. 2011;55(3):1147-1158.
46. Winterer G, Weinberger DR. Genes, dopamine and cortical signal-to-noise ratio in schizophrenia. Trends Neurosci. 2004;27(11):683-690.
47. Albert R, Jeong H, Barabási AL. Error and attack tolerance of complex networks. Nature. 2000;406(6794):378-382.
48. Crossley NA, Mechelli A, Scott J, et al. The hubs of the human connectome are generally implicated in the anatomy of brain disorders. Brain. 2014;137(Pt 8):2382-2395.
49. Barabási AL, Albert R. Emergence of scaling in random networks. Science. 1999;286(5439):509-512.
50. Albert R, Jeong H, Barabási AL. Error and attack tolerance of complex networks. Nature. 2000;406(6794):378-382.
51. van den Heuvel MP, Sporns O. Rich-club organization of the human connectome. J Neurosci. 2011;31(44):15775-15786.
52. Harriger L, van den Heuvel MP, Sporns O. Rich club organization of macaque cerebral cortex and its role in network communication. PLoS One. 2012;7(9): e46497.
53. Honey CJ, Sporns O. Dynamical consequences of lesions in cortical networks. Hum Brain Mapp. 2008;29(7):802-809.
54. Guimerà R, Nunes Amaral LA. Functional cartography of complex metabolic networks. Nature. 2005;433(7028):895-900.
55. Power JD, Schlaggar BL, Lessov-Schlaggar CN, Petersen SE. Evidence for hubs in human functional brain networks. Neuron. 2013;79(4):798-813.
56. Alstott J, Breakspear M, Hagmann P, Cammoun L, Sporns O. Modeling the impact of lesions in the human brain. PLoS Comput Biol. 2009;5(6): e1000408.
57. Marder E, Goaillard JM. Variability, compensation and homeostasis in neuron and network function. Nat Rev Neurosci. 2006;7(7):563-574.
58. Ward NS. Neural correlates of motor recovery after stroke: a longitudinal fMRI study. Brain. 2003;126(Pt 11):2476-2496.
59. Saur D, Lange R, Baumgaertner A, et al. Dynamics of language reorganization after stroke. Brain. 2006;129(Pt 6):1371-1384.
60. Bestmann S, Swayne O, Blankenburg F, et al. The role of contralesional dorsal premotor cortex after stroke as studied with concurrent TMS-fMRI. J Neurosci. 2010;30(36):11926-11937.
61. Tononi G, Sporns O, Edelman GM. Measures of degeneracy and redundancy in biological networks. Proc Natl Acad Sci USA. 1999;96(6):3257-3262.
62. Noppeney U, Friston KJ, Price CJ. Degenerate neuronal systems sustaining cognitive functions. J Anat. 2004;205(6):433-442.
63. Barulli D, Stern Y. Efficiency, capacity, compensation, maintenance, plasticity: emerging concepts in cognitive reserve. Trends Cogn Sci. 2013;17(10):502-509.
64. Stern Y. Cognitive reserve in ageing and Alzheimer's disease. Lancet Neurol. 2012;11(11):1006-1012.
65. Friston KJ, Price CJ. Modules and brain mapping. Cogn Neuropsychol. 2011;28(3-4):241-250.
66. Sporns O. Contributions and challenges for network models in cognitive neuroscience. Nat Neurosci. 2014;17(5):652-660.
67. Menon V. Large-scale brain networks and psychopathology: a unifying triple network model. Trends Cogn Sci. 2011;15(10):483-506.
68. Terry JR, Benjamin O, Richardson MP. Seizure generation: the role of nodes and networks. Epilepsia. 2012;53(9): e166-9.
69. Bartolomei F, Lagarde S, Wendling F, et al. Defining epileptogenic networks: contribution of SEEG and signal analysis. Epilepsia. 2017;58(7):1131-1147.
70. Duffau H. The usefulness of the asleep-awake-asleep glioma surgery. Acta Neurochir (Wien). 2014;156(8):1493-1494.
71. Pradilla G, Ratcliff JJ, Hall AJ, et al. Trial of Early Minimally Invasive Removal of Intracerebral Hemorrhage. N Engl J Med. 2024;390(14):1277-1289.
72. Ratcliff JJ, Hall AJ, Porto E, et al. Early Minimally Invasive Removal of Intracerebral Hemorrhage (ENRICH): Study protocol for a multi-centered two-arm randomized adaptive trial. Front Neurol. 2023; 14:1126958.
73. Menon V. Large-scale brain networks and psychopathology: a unifying triple network model. Trends Cogn Sci. 2011;15(10):483-506.
74. Fox MD, Buckner RL, White MP, Greicius MD, Pascual-Leone A. Efficacy of transcranial magnetic stimulation targets for depression is related to intrinsic functional connectivity with the subgenual cingulate. Biol Psychiatry. 2012;72(7):595-603.

75. Rehme AK, Grefkes C. Cerebral network disorders after stroke: evidence from imaging-based connectivity analyses of active and resting brain states in humans. J Physiol. 2013;591(1):17-31.
76. Carter AR, Shulman GL, Corbetta M. Why use a connectivity-based approach to study stroke and recovery of function? NeuroImage. 2012;62(4):2271-2280.
77. He BJ, Snyder AZ, Vincent JL, et al. Breakdown of functional connectivity in frontoparietal networks underlies behavioral deficits in spatial neglect. Neuron. 2007;53(6):905-918.
78. Seeley WW, Crawford RK, Zhou J, Miller BL, Greicius MD. Neurodegenerative diseases target large-scale human brain networks. Neuron. 2009;62(1):42-52.
79. Myers N, Pasquini L, Göttler J, et al. Within-patient correspondence of amyloid-β and intrinsic network connectivity in Alzheimer's disease. Brain. 2014;137(Pt 7):2052-2064.
80. Zhou J, Gennatas ED, Kramer JH, Miller BL, Seeley WW. Predicting regional neurodegeneration from the healthy brain functional connectome. Neuron. 2012;73(6):1216-1227.
81. Sporns O, Tononi G, Kötter R. The human connectome: a structural description of the human brain. PLoS Comput Biol. 2005;1(4): e42.
82. White JG, Southgate E, Thomson JN, Brenner S. The structure of the nervous system of the nematode Caenorhabditis elegans. Phil Trans R Soc Lond B. 1986;314(1165):1-340.
83. Zingg B, Hintiryan H, Gou L, et al. Neural networks of the mouse neocortex. Cell. 2014;156(5):1096-1111.
84. Van Essen DC, Smith SM, Barch DM, et al. The WU-Minn Human Connectome Project: an overview. NeuroImage. 2013; 80:62-79.
85. Van Essen DC, Smith SM, Barch DM, et al. The WU-Minn Human Connectome Project: an overview. NeuroImage. 2013; 80:62-79.
86. Hagmann P, Cammoun L, Gigandet X, et al. Mapping the structural core of human cerebral cortex. PLoS Biol. 2008;6(7): e159.
87. Turaga SC, Murray JF, Jain V, et al. Convolutional networks can learn to generate affinity graphs for image segmentation. Neural Comput. 2010;22(2):511-538.
88. Kasthuri N, Lichtman JW. Neurocartography. Neuropsychopharmacology. 2010;35(1):342-343.
89. Kandel ER, Markram H, Matthews PM, Yuste R, Koch C. Neuroscience thinks big (and collaboratively). Nat Rev Neurosci. 2013;14(9):659-664.
90. Poline JB, Breeze JL, Ghosh S, et al. Data sharing in neuroimaging research. Front Neuroinform. 2012; 6:9.
91. Breakspear M. Dynamic models of large-scale brain activity. Nat Neurosci. 2017;20(3):340-352.
92. Deco G, Jirsa VK, McIntosh AR. Emerging concepts for the dynamical organization of resting-state activity in the brain. Nat Rev Neurosci. 2011;12(1):43-56.
93. Jirsa VK, Sporns O, Breakspear M, Deco G, McIntosh AR. Towards the virtual brain: network modeling of the intact and the damaged brain. Arch Ital Biol. 2010;148(3):189-205.
94. Anderson V, Spencer-Smith M, Wood A. Do children really recover better? Neurobehavioural plasticity after early brain insult. Brain. 2011;134(Pt 8):2197-2221.
95. Bates E, Reilly J, Wulfeck B, et al. Differential effects of unilateral lesions on language production in children and adults. Brain Lang. 2001;79(2):223-265.
96. Kennard MA. Cortical reorganization of motor function. Arch Neurol Psychiatry. 1942;48(2):227-240.
97. Hensch TK. Critical period regulation. Annu Rev Neurosci. 2004; 27:549-579.
98. Huttenlocher PR, Dabholkar AS. Regional differences in synaptogenesis in human cerebral cortex. J Comp Neurol. 1997;387(2):167-178.
99. Fair DA, Cohen AL, Power JD, et al. Functional brain networks develop from a "local to distributed" organization. PLoS Comput Biol. 2009;5(5): e1000381.
100. Thompson PM, Jahanshad N, Ching CRK, et al. ENIGMA and global neuroscience: A decade of large-scale studies of the brain in health and disease across more than 40 countries. Transl Psychiatry. 2020;10(1):100.
101. Palop JJ, Chin J, Mucke L. A network dysfunction perspective on neurodegenerative diseases. Nature. 2006;443(7113):768-773.
102. Raj A, Kuceyeski A, Weiner M. A network diffusion model of disease progression in dementia. Neuron. 2012;73(6):1204-1215.
103. Sporns O. Network attributes for segregation and integration in the human brain. Curr Opin Neurobiol. 2013;23(2):162-171.
104. Fornito A, Zalesky A, Breakspear M. The connectomics of brain disorders. Nat Rev Neurosci. 2015;16(3):159-172.
105. Freeman LC. Centrality in social networks conceptual clarification. Soc Networks. 1978;1(3):215-239.
106. Albert R, Jeong H, Barabási AL. Error and attack tolerance of complex networks. Nature. 2000;406(6794):378-382.
107. Kitsak M, Gallos LK, Havlin S, et al. Identification of influential spreaders in complex networks. Nat Phys. 2010;6(11):888-893.
108. Warren DE, Power JD, Bruss J, et al. Network measures predict neuropsychological outcome after brain injury. Proc Natl Acad Sci USA. 2014;111(39):14247-14252.
109. Crossley NA, Mechelli A, Scott J, et al. The hubs of the human connectome are generally implicated in the anatomy of brain disorders. Brain. 2014;137(Pt 8):2382-2395.
110. Sporns O. Network attributes for segregation and integration in the human brain. Curr Opin Neurobiol. 2013;23(2):162-171.
111. Tononi G, Sporns O, Edelman GM. Measures of degeneracy and redundancy in biological networks. Proc Natl Acad Sci USA. 1999;96(6):3257-3262.
112. Variano EA, McCoy JH, Lipson H. Networks, dynamics, and modularity. Phys Rev Lett. 2004;92(18):188701.
113. Nepusz T, Négyessy L, Bazsó F, Tusnády GE. Fuzzy communities and the concept of bridgeness in complex networks. Phys Rev E. 2008;77(1):016107.
114. Noppeney U, Friston KJ, Price CJ. Degenerate neuronal systems sustaining cognitive functions. J Anat. 2004;205(6):433-442.
115. Warren DE, Power JD, Bruss J, et al. Network measures predict neuropsychological outcome after brain injury. Proc Natl Acad Sci USA. 2014;111(39):14247-14252.
116. Blakemore SJ. The social brain in adolescence. Nat Rev Neurosci. 2008;9(4):267-277.
117. Adolphs R. The social brain: neural basis of social knowledge. Annu Rev Psychol. 2009; 60:693-716.
118. Mars RB, Sallet J, Schüffelgen U, et al. Connectivity-based subdivisions of the human temporoparietal junction area: evidence for different areas participating in different cortical networks. Cereb Cortex. 2012;22(8):1894-1903.
119. Rizzolatti G, Craighero L. The mirror-neuron system. Annu Rev Neurosci. 2004; 27:169-192.
120. Schilbach L, Eickhoff SB, Rotarska-Jagiela A, Fink GR, Vogeley K. Minds at rest? Social cognition as the default mode of cognizing and its putative relationship to the "default" system of the brain. Conscious Cogn. 2008;17(2):457-467.
121. Hasson U, Ghazanfar AA, Galantucci B, Garrod S, Keysers C. Brain-to-brain coupling: a mechanism for creating and sharing a social world. Trends Cogn Sci. 2012;16(2):114-121.
122. Cozolino L. The Neuroscience of Psychotherapy: Healing the Social Brain. 2nd ed. New York: Norton; 2010.
123. Schore AN. Affect Dysregulation and Disorders of the Self. New York: Norton; 2003.

124. Stephens GJ, Silbert LJ, Hasson U. Speaker-listener neural coupling underlies successful communication. Proc Natl Acad Sci USA. 2010;107(32):14425-14430.
125. Porges SW. The polyvagal theory: phylogenetic substrates of a social nervous system. Int J Psychophysiol. 2001;42(2):123-146.
126. McAdams DP. The psychology of life stories. Rev Gen Psychol. 2001;5(2):100-122.
127. van der Kolk BA. The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma. New York: Viking; 2014.
128. Schore AN. Attachment and the regulation of the right brain. Attach Hum Dev. 2000;2(1):23-47.
129. Teicher MH, Samson JA, Anderson CM, Ohashi K. The effects of childhood maltreatment on brain structure, function and connectivity. Nat Rev Neurosci. 2016;17(10):652-666.
130. Herman JL. Trauma and Recovery: The Aftermath of Violence--From Domestic Abuse to Political Terror. New York: Basic Books; 2015.
131. Lanius RA, Vermetten E, Loewenstein RJ, et al. Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. Am J Psychiatry. 2010;167(6):640-647.
132. Siegel DJ. The Developing Mind: How Relationships and the Brain Interact to Shape Who We Are. 2nd ed. New York: Guilford Press; 2012.
133. Lewis T, Amini F, Lannon R. A General Theory of Love. New York: Random House; 2000.
134. Kandel ER. In Search of Memory: The Emergence of a New Science of Mind. New York: Norton; 2006.
135. Ibanez-Marcelo E, Campioni L, Phinyomark A, Petri G, Santarcangelo EL. Topology highlights mesoscopic functional equivalence between imagery and perception: The case of hypnotizability. NeuroImage. 2019; 200:437-449.
136. Koenig T, Prichep L, Lehmann D, et al. Millisecond by millisecond, year by year: normative EEG microstates and developmental stages. NeuroImage. 2002;16(1):41-48.
137. Chiao JY, Cheon BK. The weirdest brains in the world. Behav Brain Sci. 2010;33(2-3):88-90.
138. Henrich J, Heine SJ, Norenzayan A. The weirdest people in the world? Behav Brain Sci. 2010;33(2-3):61-83.
139. Metzinger T. Being No One: The Self-Model Theory of Subjectivity. Cambridge, MA: MIT Press; 2003.
140. Turkle S. Alone Together: Why We Expect More from Technology and Less from Each Other. New York: Basic Books; 2011.
141. Insel T, Cuthbert B, Garvey M, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. Am J Psychiatry. 2010;167(7):748-751.
142. Cuthbert BN, Insel TR. Toward the future of psychiatric diagnosis: the seven pillars of RDoC. BMC Med. 2013; 11:126.
143. Rutter M, Moffitt TE, Caspi A. Gene-environment interplay and psychopathology: multiple varieties but real effects. J Child Psychol Psychiatry. 2006;47(3-4):226-261.
144. Silk JS, Davis S, McMakin DL, Dahl RE, Forbes EE. Why do anxious children become depressed teenagers? The role of social evaluative threat and reward processing. Psychol Med. 2012;42(10):2095-2107.
145. Kashdan TB, Rottenberg J. Psychological flexibility as a fundamental aspect of health. Clin Psychol Rev. 2010;30(7):865-878.
146. Institute of Medicine. Crossing the Quality Chasm: A New Health System for the 21st Century. Washington, DC: National Academy Press; 2001.
147. Levinson W, Roter DL, Mullooly JP, Dull VT, Frankel RM. Physician-patient communication. The relationship with malpractice claims among primary care physicians and surgeons. JAMA. 1997;277(7):553-559.
148. Kataoka HU, Koide N, Ochi K, Hojat M, Gonnella JS. Measurement of empathy among Japanese medical students: psychometrics and score differences by gender and level of medical education. Acad Med. 2009;84(9):1192-1197.
149. Epstein RM. Mindful practice. JAMA. 1999;282(9):833-839.
150. Institute of Medicine. Health Professions Education: A Bridge to Quality. Washington, DC: National Academies Press; 2003.
151. Hood L, Friend SH. Predictive, personalized, preventive, participatory (P4) cancer medicine. Nat Rev Clin Oncol. 2011;8(3):184-187.
152. Cuthbert BN. The RDoC framework: facilitating transition from ICD/DSM to dimensional approaches that integrate neuroscience and psychopathology. World Psychiatry. 2014;13(1):28-35.
153. Norcross JC, Wampold BE. Evidence-based therapy relationships: research conclusions and clinical practices. Psychotherapy (Chic). 2011;48(1):98-102.
154. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. PLoS Med. 2010;7(7): e1000316.
155. Dede C, Richards J, editors. Digital Teaching Platforms. New York: Teachers College Press; 2012.
156. Beauchamp TL, Childress JF. Principles of Biomedical Ethics. 7th ed. New York: Oxford University Press; 2012.
157. Ienca M, Andorno R. Towards new human rights in the age of neuroscience and neurotechnology. Life Sci Soc Policy. 2017;13(1):5.
158. Reardon S. 'Brain doping' may improve athletes' performance. Nature. 2016;531(7594):283-284.
159. Kleinman A. What Really Matters: Living a Moral Life amid Uncertainty and Danger. New York: Oxford University Press; 2006.
160. Markram H, Muller E, Ramaswamy S, et al. Reconstruction and simulation of neocortical microcircuitry. Cell. 2015;163(2):456-492.
161. Sporns O. Discovering the Human Connectome. Cambridge, MA: MIT Press; 2012.
162. Calhoun VD, Miller R, Pearlson G, Adalı T. The chronnectome: time-varying connectivity networks as the next frontier in fMRI data discovery. Neuron. 2014;84(2):262-274.
163. Bassett DS, Sporns O. Network neuroscience. Nat Neurosci. 2017;20(3):353-364.
164. Zalesky A, Fornito A, Cocchi L, Gollo LL, Breakspear M. Time-resolved resting-state brain networks. Proc Natl Acad Sci USA. 2014;111(28):10341-10346.
165. Reardon S. 'Brain organoids' grow in dishes. Nature. 2016;539(7628):180-181.
166. Schork NJ. Personalized medicine: Time for one-person trials. Nature. 2015;520(7549):609-611.
167. Fox MD, Buckner RL, White MP, Greicius MD, Pascual-Leone A. Efficacy of transcranial magnetic stimulation targets for depression is related to intrinsic functional connectivity with the subgenual cingulate. Biol Psychiatry. 2012;72(7):595-603.
168. Lebedev MA, Nicolelis MAL. Brain-machine interfaces: past, present and future. Trends Neurosci. 2006;29(9):536-546.
169. Jones DK, Knösche TR, Turner R. White matter integrity, fiber count, and other fallacies: the do's and don'ts of diffusion MRI. NeuroImage. 2013; 73:239-254.
170. Zalesky A, Fornito A. A DTI-derived measure of cortico-cortical connectivity. IEEE Trans Med Imaging. 2009;28(7):1023-1036.
171. Friston KJ, Kahan J, Biswal B, Razi A. A DCM for resting state fMRI. NeuroImage. 2014; 94:396-407.
172. David O, Guillemain I, Saillet S, et al. Identifying neural drivers

with functional MRI: an electrophysiological validation. PLoS Biol. 2008;6(12):2683-2697.
173. Ritter P, Schirner M, McIntosh AR, Jirsa VK. The virtual brain integrates computational modeling and multimodal neuroimaging. Brain Connect. 2013;3(2):121-145.
174. Chalmers DJ. The Conscious Mind: In Search of a Fundamental Theory. New York: Oxford University Press; 1996.
175. Tononi G. Integrated information theory: a provisional manifesto. Biol Bull. 2008;215(3):216-242.
176. Dehaene S, Changeux JP. Experimental and theoretical approaches to conscious processing. Neuron. 2011;70(2):200-227.
177. Varela FJ, Thompson E, Rosch E. The Embodied Mind: Cognitive Science and Human Experience. Cambridge, MA: MIT Press; 1991.
178. Gabrieli JD, Ghosh SS, Whitfield-Gabrieli S. Prediction as a humanitarian and pragmatic contribution from human cognitive neuroscience. Neuron. 2015;85(1):11-26.
179. Mueller S, Wang D, Fox MD, et al. Individual variability in functional connectivity architecture of the human brain. Neuron. 2013;77(3):586-595.
180. Farah MJ. Emerging ethical issues in neuroscience. Nature. 2002;421(6922):517-522.
181. Van Horn JD, Toga AW. Human neuroimaging as a "Big Data" science. Brain Imaging Behav. 2014;8(2):323-331.
182. Kasthuri N, Lichtman JW. Neurocartography. Neuropsychopharmacology. 2010;35(1):342-343.
183. Friston KJ. Functional and effective connectivity: a review. Brain Connect. 2011;1(1):13-36.
184. Rose N, Abi-Rached JM. Neuro: The New Brain Sciences and the Management of the Mind. Princeton, NJ: Princeton University Press; 2013.
185. Kleinman A. The Illness Narratives: Suffering, Healing, and the Human Condition. New York: Basic Books; 1988.
186. Poldrack RA, Gorgolewski KJ. Making big data open: data sharing in neuroimaging. Nat Neurosci. 2014;17(11):1510-1517.
187. Milham MP, Craddock RC, Son JJ, et al. Assessment of the impact of shared brain imaging data on the scientific literature. Nat Commun. 2018;9(1):2818.
188. Sporns O. Networks of the Brain. Cambridge, MA: MIT Press; 2010.
189. Bassett DS, Gazzaniga MS. Understanding complexity in the human brain. Trends Cogn Sci. 2011;15(5):200-209.
190. Siegel DJ. Mindsight: The New Science of Personal Transformation. New York: Bantam; 2010.
191. Lichtman JW, Denk W. The big and the small: challenges of imaging the brain's circuits. Science. 2011;334(6056):618-623.
192. Schilbach L, Timmermans B, Reddy V, et al. Toward a second-person neuroscience. Behav Brain Sci. 2013;36(4):393-414.
193. Insel TR. The NIMH Research Domain Criteria (RDoC) Project: precision medicine for psychiatry. Am J Psychiatry. 2014;171(4):395-397.
194. Lutz A, Slagter HA, Dunne JD, Davidson RJ. Attention regulation and monitoring in meditation. Trends Cogn Sci. 2008;12(4):163-169.
195. Grefkes C, Fink GR. Connectivity-based approaches in stroke and recovery of function. Lancet Neurol. 2014;13(2):206-216.
196. Damasio A. Descartes' Error: Emotion, Reason, and the Human Brain. New York: Putnam; 1994.
197. Adolphs R. Social cognition and the human brain. Trends Cogn Sci. 1999;3(12):469-479.
198. Cacioppo JT, Cacioppo S. Social relationships and health: the toxic effects of perceived social isolation. Soc Personal Psychol Compass. 2014;8(2):58-72.
199. Nagel T. What Is It Like to Be a Bat? Philos Rev. 1974;83(4):435-450.
200. Buber M. I and Thou. Trans. Walter Kaufmann. New York: Charles Scribner's Sons; 1970.
201. Marcel G. The Mystery of Being. Trans. G.S. Fraser. Chicago: Regnery; 1951.
202. Levinas E. Totality and Infinity: An Essay on Exteriority. Trans. Alphonso Lingis. Pittsburgh: Duquesne University Press; 1969.
203. Kandel ER. The Age of Insight: The Quest to Understand the Unconscious in Art, Mind, and Brain. New York: Random House; 2012.
204. Marcus G, Freeman J, editors. The Future of the Brain: Essays by the World's Leading Neuroscientists. Princeton, NJ: Princeton University Press; 2015.
205. Thompson E. Mind in Life: Biology, Phenomenology, and the Sciences of Mind. Cambridge, MA: Harvard University Press; 2007.
206. Sober E, Wilson DS. Unto Others: The Evolution and Psychology of Unselfish Behavior. Cambridge, MA: Harvard University Press; 1998.
207. Tomasello M. A Natural History of Human Thinking. Cambridge, MA: Harvard University Press; 2014.
208. Merleau-Ponty M. Phenomenology of Perception. Trans. Colin Smith. London: Routledge; 1962.
209. Lanius RA, Vermetten E, Loewenstein RJ, et al. Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. Am J Psychiatry. 2010;167(6):640-647.
210. Sripada RK, King AP, Welsh RC, et al. Neural dysregulation in posttraumatic stress disorder: evidence for disrupted equilibrium between salience and default mode brain networks. Psychosom Med. 2012;74(9):904-911.
211. van der Kolk BA. The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma. New York: Viking; 2014.
212. Bluhm RL, Williamson PC, Osuch EA, et al. Alterations in default network connectivity in posttraumatic stress disorder related to early-life trauma. J Psychiatry Neurosci. 2009;34(3):187-194.
213. Brewin CR, Gregory JD, Lipton M, Burgess N. Intrusive images in psychological disorders: characteristics, neural mechanisms, and treatment implications. Psychol Rev. 2010;117(1):210-232.
214. Miller DR, Hayes SM, Hayes JP, et al. Default mode network subsystems are differentially disrupted in posttraumatic stress disorder. Biol Psychiatry Cogn Neurosci Neuroimaging. 2017;2(4):363-371.
215. Menon V, Uddin LQ. Saliency, switching, attention and control: a network model of insula function. Brain Struct Funct. 2010;214(5-6):655-667.
216. Hayes JP, Hayes SM, Mikedis AM. Quantitative meta-analysis of neural activity in posttraumatic stress disorder. Biol Mood Anxiety Disord. 2012; 2:9.
217. Aupperle RL, Melrose AJ, Stein MB, Paulus MP. Executive function and PTSD: disengaging from trauma. Neuropharmacology. 2012;62(2):686-694.
218. Sheynin J, Liberzon I. The circuit of fear: implications for posttraumatic stress disorder. Biol Psychiatry. 2017;78(5):312-321.
219. Milad MR, Pitman RK, Ellis CB, et al. Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. Biol Psychiatry. 2009;66(12):1075-1082.
220. Rauch SL, Shin LM, Phelps EA. Neurocircuitry models of posttraumatic stress disorder and extinction: human neuroimaging research--past, present, and future. Biol Psychiatry. 2006;60(4):376-382.
221. Teicher MH, Samson JA, Anderson CM, Ohashi K. The effects

of childhood maltreatment on brain structure, function and connectivity. Nat Rev Neurosci. 2016;17(10):652-666.
222. Perry BD. Childhood experience and the expression of genetic potential: what childhood neglect tells us about nature and nurture. Brain Mind. 2002;3(1):79-100.
223. Cloitre M, Stolbach BC, Herman JL, et al. A developmental approach to complex PTSD: childhood and adult cumulative trauma as predictors of symptom complexity. J Trauma Stress. 2009;22(5):399-408.
224. Ford JD, Courtois CA. Complex PTSD, affect dysregulation, and borderline personality disorder. Borderline Personal Disord Emot Dysregul. 2014; 1:9.
225. McEwen BS. Stress, adaptation, and disease. Allostasis and allostatic load. Ann N Y Acad Sci. 1998; 840:33-44.
226. Bremner JD. Traumatic stress: effects on the brain. Dialogues Clin Neurosci. 2006;8(4):445-461.
227. Bam M, Yang X, Zhou J, et al. Evidence for local and systemic immune activation in post-traumatic stress disorder. Mol Psychiatry. 2016;21(9):1228-1240.
228. Michopoulos V, Powers A, Gillespie CF, Ressler KJ, Jovanovic T. Inflammation in fear- and anxiety-based disorders: PTSD, GAD, and beyond. Neuropsychopharmacology. 2017;42(1):254-270.
229. Gapp K, Jawaid A, Sarkies P, et al. Implication of sperm RNAs in transgenerational inheritance of the effects of early trauma in mice. Nat Neurosci. 2014;17(5):667-669.
230. Yehuda R, Daskalakis NP, Bierer LM, et al. Holocaust exposure induced intergenerational effects on FKBP5 methylation. Biol Psychiatry. 2016;80(5):372-380.
231. van der Kolk BA, Spinazzola J, Blaustein ME, et al. A randomized clinical trial of eye movement desensitization and reprocessing (EMDR), fluoxetine, and pill placebo in the treatment of posttraumatic stress disorder: Treatment effects and long-term maintenance. J Clin Psychiatry. 2007;68(1):37-46.
232. Kluetsch RC, Ros T, Théberge J, et al. Plastic modulation of PTSD resting-state networks and subjective wellbeing by EEG neurofeedback. Acta Psychiatr Scand. 2014;130(2):123-136.
233. Boggio PS, Rocha M, Oliveira MO, et al. Noninvasive brain stimulation with high-frequency and low-intensity repetitive transcranial magnetic stimulation treatment for posttraumatic stress disorder. J Clin Psychiatry. 2010;71(8):992-999.
234. Kearney DJ, McDermott K, Malte C, Martinez M, Simpson TL. Association of participation in a mindfulness program with measures of PTSD, depression and quality of life in a veteran sample. J Clin Psychol. 2012;68(1):101-116.
235. Holzel BK, Carmody J, Evans KC, et al. Stress reduction correlates with structural changes in the amygdala. Soc Cogn Affect Neurosci. 2010;5(1):11-17.
236. Herman JL. Trauma and Recovery: The Aftermath of Violence--From Domestic Abuse to Political Terror. New York: Basic Books; 2015.
237. Porges SW. The polyvagal theory: phylogenetic substrates of a social nervous system. Int J Psychophysiol. 2001;42(2):123-146.
238. Hasson U, Ghazanfar AA, Galantucci B, Garrod S, Keysers C. Brain-to-brain coupling: a mechanism for creating and sharing a social world. Trends Cogn Sci. 2012;16(2):114-121.
239. Schore AN. Attachment and the regulation of the right brain. Attach Hum Dev. 2000;2(1):23-47.
240. Siegel DJ. The Developing Mind: How Relationships and the Brain Interact to Shape Who We Are. 2nd ed. New York: Guilford Press; 2012.
241. McAdams DP. The psychology of life stories. Rev Gen Psychol. 2001;5(2):100-122.
242. Cozolino L. The Neuroscience of Psychotherapy: Healing the Social Brain. 2nd ed. New York: Norton; 2010.
243. Galatzer-Levy IR, Bryant RA. 636,120 Ways to Have Posttraumatic Stress Disorder. Perspect Psychol Sci. 2013;8(6):651-662.
244. Bonanno GA, Mancini AD. The human capacity to thrive in the face of potential trauma. Pediatrics. 2008;121(2):369-375.
245. Admon R, Milad MR, Hendler T. A causal model of post-traumatic stress disorder: disentangling predisposed from acquired neural abnormalities. Trends Cogn Sci. 2013;17(7):337-347.
246. Buckley TC, Blanchard EB, Neill WT. Information processing and PTSD: a review of the empirical literature. Clin Psychol Rev. 2000;20(8):1041-1065.
247. Kleinman A. What Really Matters: Living a Moral Life amid Uncertainty and Danger. New York: Oxford University Press; 2006.
248. Insel T, Cuthbert B, Garvey M, et al. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. Am J Psychiatry. 2010;167(7):748-751.
249. Stein DJ, Koenen KC, Friedman MJ, et al. Dissociation in posttraumatic stress disorder: evidence from the world mental health surveys. Biol Psychiatry. 2013;73(4):302-312.
250. Etkin A, Wager TD. Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. Am J Psychiatry. 2007;164(10):1476-1488.
251. Schnurr PP, Lunney CA. Symptom benchmarks of improved quality of life in PTSD. Depress Anxiety. 2016;33(3):247-255.
252. Masten AS, Motti-Stefanidi F. Multisystem resilience for children and youth in disaster: reflections in the context of COVID-19. Advers Resil Sci. 2020;1(2):95-106.
253. Levine PA. Waking the Tiger: Healing Trauma. Berkeley, CA: North Atlantic Books; 1997.
254. Sporns O. Graph theory methods: applications in brain networks. Dialogues Clin Neurosci. 2018;20(2):111-121.
255. Fornito A, Zalesky A, Bullmore E. Fundamentals of Brain Network Analysis. Amsterdam: Academic Press; 2016.
256. Rubinov M, Sporns O. Complex network measures of brain connectivity uses and interpretations. NeuroImage. 2010;52(3):1059-1069.