BRAIN UPDATE

Common neural substrates of diverse neurodevelopmental disorders

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Neurodevelopmental disorders are categorized and studied according to their manifestations as distinct syndromes. For instance, congenital prosopagnosia and dyslexia have largely non-overlapping research literatures and clinical pathways for diagnosis and intervention. On the other hand, the high incidence of neurodevelopmental comorbidities or co-existing extreme strengths and weaknesses suggest that transdiagnostic commonalities may be greater than currently appreciated. The core-periphery model holds that brain regions within the stable core perceptual and motor regions are more densely connected to one another compared to regions in the flexible periphery comprising multimodal association regions. This model provides a framework for the interpretation of neural data in normal development and clinical disorders. Considering network-level commonalities reported in studies of neurodevelopmental disorders, variability in multimodal association cortex connectivity may reflect a shared origin of seemingly distinct neurodevelopmental disorders. This framework helps to explain both comorbidities in neurodevelopmental disorders and profiles of strengths and weaknesses attributable to competitive processing between cognitive systems within an individual.

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Neurodevelopmental disorders—or congenital disorders of brain system development causing cognitive and behavioral impairments—have wide-ranging consequences for academic, social, and mental health outcomes.[1,2] These disorders—affecting ~53 million children within the first 5 years of life (i.e. a prevalence of 12%)[3]—are the third-ranked form of child disability, after vision and hearing loss. The worldwide annual burden of childhood disability is estimated to be as high as $69 500 per child, annually. Although there is a heavier burden in developing countries,[4] financial disparities between families with and without children with neurodevelopmental disorders are evident across all levels of socioeconomic status.[5]

Neurodevelopmental disorders can have effects confined to specific domains, such as reading (dyslexia) or facial recognition (congenital prosopagnosia), or domain-general functions such as attention (attention deficient hyperactivity disorder, ADHD) or

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social communication (autism spectrum disorder, ASD). Much of the accumulated knowledge concerning neurodevelopmental disorders has emerged from within-disorder research, rather than comparison across disorders. Indeed, as with case-control research in general, the presence of comorbidities can be grounds for exclusion as it contaminates the 'purity' of the phenotype of interest. This practice has promoted depth of knowledge concerning individual disorders at the expense of recognizing shared features across disorders.

In this update, we synthesize neuroimaging research in samples of putatively distinct neurodevelopmental disorders affecting specific perceptual, mnemonic, and academic functions. Many of these disorders mimic classical focal lesion syndromes (e.g. acquired dyslexia or prosopagnosia) such that they implicate discrete neurofunctional circuits (e.g. left-lateralized language circuits in dyslexia or ventral temporal circuits in prosopagnosia). Yet network models of brain organization hold that function is also supported by inter-regional connectivity, including stable trait-like differences in brain organization that are reliable[6] and emerge during development,[7] thus providing a rich source of information about brain-behaviour relationships.[8,9] Moreover, these models have the advantage of accommodating interactions within and between large-scale systems in addition to functions localized to discrete circuits.[10]

Examining commonalities in network-level function across diagnostic groups can provide insights into which connectome alterations are shared among brain disorders, as has been done with neurodegenerative diseases (Box 1)[11] and psychiatric disorders.[17] The core-periphery model of brain organization distinguishes stable core perceptual and motor regions from multimodal association regions in the flexible periphery, with core regions more densely interconnected than those in the periphery.[24] The present review illustrates a common pattern of altered connectivity between core basic processing units and higher-level periphery association cortices across neurodevelopmental disorders affecting perceptual, academic, and mnemonic function, resonating with recent criticism of the core-deficit hypothesis in neurodevelopmental disorders.[25] This transdiagnostic approach helps to account for comorbidities and paradoxical extreme strengths and weaknesses within an individual.[26]

Neurodevelopmental disorders across cognitive domains

Perceptual functions: congenital prosopagnosia and amusia

Individuals with a lifelong incapacity to perceive faces are classified as having congenital prosopagnosia. Across individuals, face-processing is supported by a network spanning the ventral occipital temporal cortex that includes the fusiform face area and occipital face area, structurally connected by the inferior longitudinal fasciculus and inferior occipito-frontal fasciculus (Fig. 1B). This core face-processing network is situated within an extended face-processing network that includes frontal regions, typically associated with executive functioning and attention (i.e. regions in the frontal lobes including the inferior frontal gyrus and orbital frontal cortex, and the anterior temporal lobe).[31] Congenital prosopagnosia (i.e. the neurodevelopmental disorder in which people cannot process faces) is thought to arise from a failure to propagate neural signals between the functioning ventral occipito-temporal cortex and the extended nodes of the face-processing network,[31–37] in addition to dysfunction of activity and connectivity within the ventral occipito-temporal cortex.[38–41]

Individuals with a lifelong inability to perceive pitch are characterized as having congenital amusia. Pitch perception is supported by a frontal-temporal network that includes the primary auditory cortex, superior temporal gyrus, and inferior frontal gyrus,[42] structurally connected by the superior longitudinal fasciculus (including the arcuate fasciculus) (Fig. 1B). Individuals with congenital amusia exhibit reduced functional and structural connectivity between auditory temporal regions and frontal regions, but intact bottom-up representations of pitch corresponding with typical patterns of connectivity between primary auditory cortex and the superior temporal gyrus.[43–50] This suggests that the pitch perception deficit arises from poor feedback control between the inferior frontal

Box 1 Pathology in neurodegenerative diseases.

In the early stages of neurodegenerative diseases, pathology aggregates in specific, localized brain regions with selectively vulnerable neuronal populations (e.g. Seeley *et al.*[12]), then progresses through anatomically linked regions,[13] consistent with the network degeneration hypothesis (for review see Drzezga[14]). This hypothesis was systematically tested in a seminal paper in which network-sensitive neuroimaging methods were used to reveal that distinct neurodegenerative systems (Alzheimer's disease; behavioural variant frontotemporal dementia; semantic dementia; progressive nonfluent aphasia; corticobasal syndrome) influenced human intrinsic functional connectivity systems present in healthy human adults[11] (see also Zhou *et al.*[15]). Even in healthy adults, structural atrophy mirrors characteristic patterns in neurodegenerative diseases, coupled with the individual's predisposition to express that disease.[16] The pressure of connectome-wide communication—with long-range connections maintained by centralized hub regions that allow for rapid transmission of information across functional domains– elevates the risk of local brain changes (such as neurodegenerative diseases) spreading easily across the network.[17] Local network disruption (i.e. failure of a particular node in a network) initiates 'cascading network failure', in which nodes fail across the network over time,[18, 19] such that the spread of neuropathological effects is mediated by structural and functional connectivity within the human connectome (for a review see Iturria-Medina and Evans[20]). Neurodegenerative disorders provide information on connectivity through 'fault lines' that are vulnerable to spreading pathology. Intriguingly, network effects can be retrospectively traced, even to childhood,[21] to cognitive strengths and weakness predating disease onset,[22] suggesting a neurodevelopmental origin. For example, structural imaging and cognitive measures can be used to predict the onset of frontotemporal dementia 5–10 years before the onset of symptoms in adults at risk of developing the neurodegenerative disorder later in life.[23] Thus, insights into individual differences in normal function and neurodevelopmental syndromes are valuable in delineating the organizational principles of the human connectome.

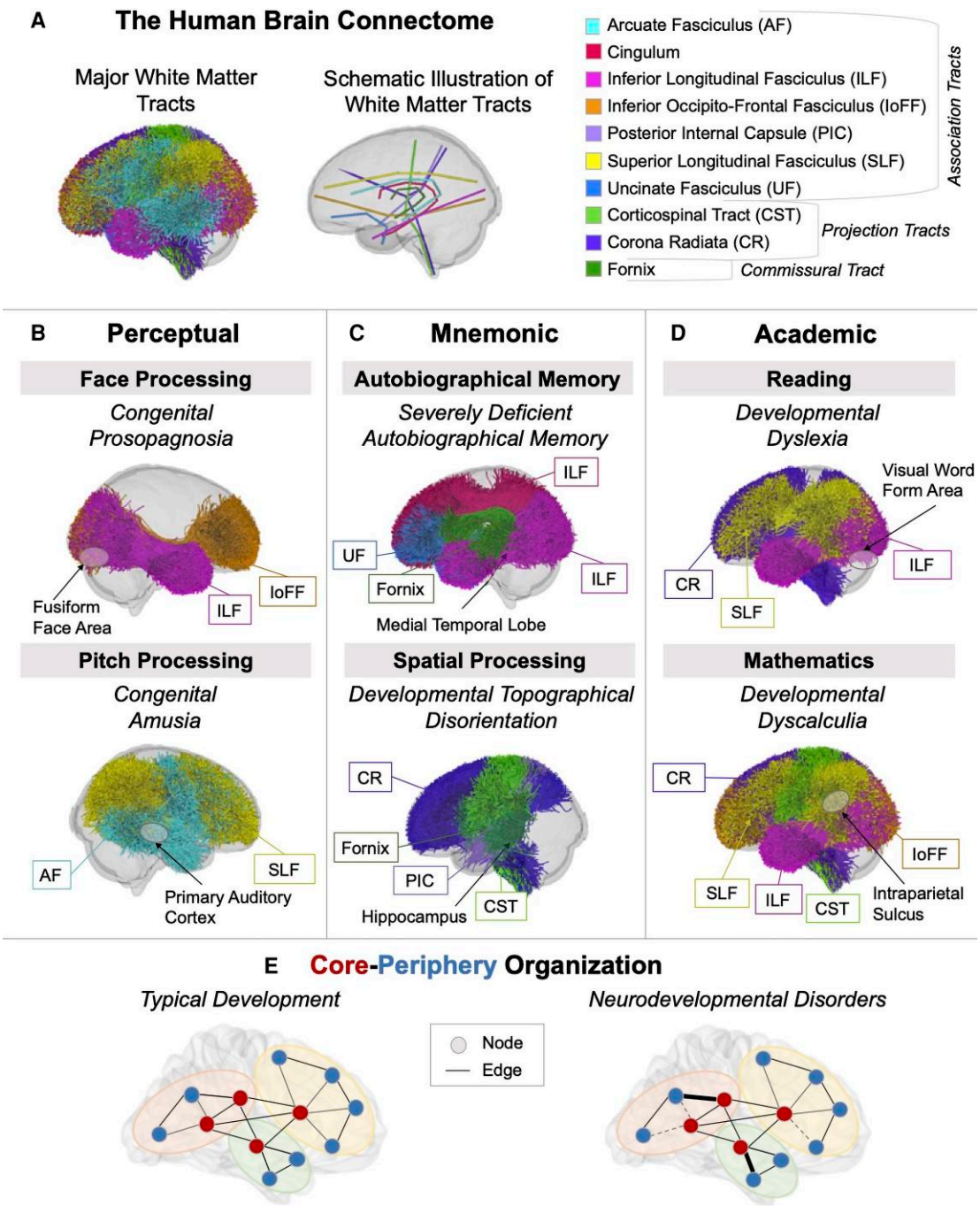

Figure 1 Long association fibres associated with perceptual, mnemonic, and academic functions. Individual differences, with neurodevelopmental disorders representing extreme ends of the distributions, have been studied in the context of specific processing domains (e.g. language, perception). Analysis of commonalities across these domains reveal general principles of human information processing and consequently, the architecture of the human brain connectome. (**A**) Visualization of anatomical connectivity within the human brain (*left*) and a schematic depiction of key white matter tracts (*right*). **B**–**D** illustrate white matter tracts forming distributed networks associated with perceptual (right hemisphere), mnemonic (left hemisphere) and academic abilities (left hemisphere). Each panel displays key white matter tracts associated with two exemplar cognitive domains (labelled in grey boxes) along with regions supporting relevant lower-level sensory processing. Individual differences that associate with variation in structural and functional network connectivity exhibit aberrant connectivity within these same networks in individuals with neurodevelopmental disorders (italicized text) (e.g. the inferior longitudinal fasciculus (ILF) and inferior occipito-frontal fasciculus (IoFF) relate to individual differences in face-processing and congenital prosopagnosia). (**E**) Schematic illustration of network topology applied to neurodevelopmental disorders. The small circles represent brain regions (i.e. nodes) and the lines represent connections between brain regions (i.e. edges). The connectome is comprised of modules (i.e. densely connected nodes depicted with green, orange, and yellow ovals). Networks with core-periphery organization also exhibit a set of tightly connected nodes (i.e. hub nodes) referred to as the core (four red inner circles) which are sparsely connected to a set of relatively isolated nodes referred to as the periphery (eight blue outer circles). Neurodevelopmental disorders share the common neural substrate of altered connectivity between the network core and periphery. These altered patterns may be characterized by weaker connections (dashed lines), but also instances of enhanced connections (bold lines) that may emerge through network reorganization or compensation. (Images in **A**–**D** were created using the anatomically curated white matter atlas generated by the O'Donnel Research Group (ORG), derived from 100 healthy human brain scans from the Human Connectome Project[27] and visualized using slicerDMR.[28,29] Panel **E** was inspired by Bassett *et al.*[30])

gyrus (i.e. a high-level processing region) and the superior temporal gyrus.[43] In direct contrast, individuals with absolute pitch show enhanced connectivity both within superior temporal lobe structures and across the frontal-temporal network.[51]

Mnemonic functions: autobiographical memory, imagery, and navigation

Encoding, storage, and retrieval of information on memory tasks are well-known to engage distributed cortical representations crucial for conscious apprehension of mnemonic content.[52] Hippocampal-neocortical connectivity, through the fornix (the main efferent pathway from the hippocampus) and through long association fibres to frontal and parietal lobes, relates to performance on laboratory and naturalistic autobiographical memory tasks.[53–55] In Severely Deficient Autobiographical Memory (SDAM) (Fig. 1C), there is a lifelong inability to vividly recollect past autobiographical events, while other functions are preserved. Individuals with SDAM show evidence of intact basic perceptuo-mnemonic processing (e.g. intact performance on neuropsychological memory tests) and grossly normal medial temporal lobe anatomy, yet they exhibit reduced large-scale neural synchrony in relation to conscious recollection or re-experiencing of events encountered in both the laboratory and real life.[56,57] By contrast, individuals on the opposite extreme end of the spectrum of autobiographical memory ability [i.e. individuals with highly superior autobiographical memory (HSAM)[58]] exhibit enhanced prefrontal-hippocampal functional connectivity.

The fundamental deficit in SDAM (subjective recollection of past events) is inaccessible to experimental verification. Visual imagery is a cognitive capacity that is closely related to autobiographical memory.[59,60] Individuals with aphantasia, a lifelong inability to voluntarily create mental images in the mind, report low visual (and other sensory) subjective imagery (including reduced autobiographical recollection) with a compelling objective behavioural correlate in binocular rivalry or perceptual competition between distinct, simultaneously- presented monocular stimuli. Priming a stimulus prior to the task normally induces a bias such that primed stimuli are perceived above chance. Aphantasics are immune to such perceptual priming effects.[61,62] That is, they fail to benefit from the primed image even though perceptual processing is intact. Similarly, they exhibit a physiological response in response to perceived, but not imagery-driven, fear-inducing stimuli.[63] The visual imagery deficit exhibited by aphantasics is not instantiated in perceptual processing units but rather in the feedback connections between the frontal and visual cortex.[64,65] As noted with other disorders, those on the other extreme end of the distribution of visual imagery ability (i.e. hyperphantasics) exhibit stronger functional connectivity between prefrontal regions and the visual network.[65]

Spatial cognition in humans is supported by a network that includes the hippocampus, parahippocampus, retrosplenial cortex, and prefrontal cortex.[66] Developmental Topographical Disorientation (DTD) is characterized as a lifelong inability to navigate new and familiar environments.[67–69] Individuals with DTD exhibit intact processing in lower-level perceptual regions (e.g. the parahippocampal place area), but reduced structural and functional connectivity between the hippocampus, parahippocampal place area, retrosplenial cortex, and prefrontal cortex[67,69,70] and within the default mode network[71] (Fig. 1C). Those with strong spatial orientation abilities exhibit increased levels of global efficiency within the spatial orientation network and increased node centrality in the hippocampus, supramarginal gyrus, and primary motor cortex.[72] Similarly, graph theoretical techniques on low-density EEG data revealed that individuals with strong, compared to weak, spatial navigation abilities showed more functional connectivity.[73]

Academic abilities: reading and mathematical competence

Reading is supported by a left-lateralized network, highly overlapping with the language network, which includes the superior temporal gyrus (Wernicke's area), inferior frontal gyrus (Broca's area), and fusiform gyrus, and is connected by the inferior longitudinal fasciculus, superior longitudinal fasciculus, inferior fronto-occipital fascicle, and corona radiata[74–76] (Fig. 1D). Individuals with developmental dyslexia, (i.e. a specific reading disability), exhibit intact phonetic representations in the auditory cortex, but a reduction in functional and structural connectivity between these temporal regions and the left inferior frontal gyrus.[74,77–79] Dyslexic individuals also present with reduced connectivity in the visual word-form area but increased connectivity within the right hemisphere.[80] Notably, individuals with dyslexia exhibit complex aberrant connectivity and even hyperconnectivity in brain systems beyond the reading network such as the limbic system and motor system, supporting the idea of a broad network-level origin of a behaviourally specific disorder.[80,81]

Mathematical thinking, including basic number processing and higher-level symbolic manipulations and calculations, is another key skill within the academic domain predictive of later success.[82] Developmental dyscalculia, a specific math learning disorder, is as equally prevalent as dyslexia but considerably less studied.[83] Mathematical thinking is subserved by a fronto-parietal network that includes the intraparietal sulcus, inferior parietal lobule, superior parietal lobule, and prefrontal cortex,[84] and is structurally connected by a range of white matter pathways including the inferior longitudinal fasciculus, superior longitudinal fasciculus, inferior fronto-occipital fascicle, corona radiata, corticospinal tract, and posterior segment of the corpus callosum[85,86] (Fig. 1D). Developmental dyscalculics present with reduced structural connectivity within these white matter tracts (for a review see Matejko and Ansari[86]). Individuals with dyscalculia also show abnormalities in numerical representations and in functional connectivity within the mathematical network.[87,88] Functional connectivity findings in this domain are complex and differ in adults and children.[89] Adults with dyscalculia exhibit functional hyperconnectivity in temporo-occipital regions and abnormal functional activation in fronto-parietal regions during number processing, whereas children exhibit hyperconnectivity in the fronto-parietal network, but also the default mode network.[90,91] These network-level differences between dyscalculics and control participants may reflect the strategies that the individual uses to compensate for the underlying weaknesses.

Interim summary

Consideration of neuroimaging findings across perceptual, mnemonic, and academic domains, as outlined in Fig. 1, converge to support the claim that neurodevelopmental disorders across distinct cognitive domains share the common neural substrate of reductions in long-range projections between local perceptual regions and higher-level prefrontal regions that govern conscious access of information. Other disorders (e.g. alexithymia, developmental coordination disorder, dysgraphia) are not reviewed in detail due to the smaller number of studies, although they show similar

patterns to the domains discussed above, namely aberrant activity and intrinsic connectivity within domain-specific localized regions and dysfunction in interconnectivity across large-scale brain networks.[92–94] Due to limitations in the inferences that can be drawn from case-control brain imaging data, the observed distributed effects cannot be conclusively separated from local (modular) effects. Our aim is not to rule out modular dysfunction, but to highlight the power of the network-level approach for explaining commonalities across neurodevelopmental disorders.

Applying network neuroscience to neurodevelopmental disorders

Diverse developmental disorders share network-level reductions in long-range cortical neuronal projections between local perceptual regions and higher-level prefrontal regions that govern conscious access of information. Deficits associated with neurodevelopmental disorders range from very specific limitations of learning to more widespread impairments of executive functions, social skills, or intelligence. The disorders reviewed above are defined by specific phenotypes in the context of relative preservation of other functions. Previous research interpreting domain-specific neurodevelopmental disorders from the perspective of encapsulated neurofunctional systems neglects the known dynamic interactivity across brain systems.[95,96] The non-random and unique organizational principles of brain networks as derived from neuroimaging data can be leveraged to formally characterize neural substrates of normal and disordered brain function.[97,98]

Core-periphery organization (see Box 2) was recently proposed as a framework to characterize linkages between functional brain modules. The core-periphery model posits that the human connectome is composed of a stable core (i.e. sensorimotor and visual regions) with limited temporal connectivity variability and a flexible periphery (i.e. multimodal association regions) with frequently changing patterns of connectivity (Fig. 1E).[24] Like modularity, core-periphery organization consolidates and strengthens across developmental time, leading to an optimized modular yet integrated topology that simultaneously supports intra-modular functional specialization and inter-modular coordination.[103] Critically, core-periphery organization predicts individual differences in cognition across development more accurately than module organization alone.[104] Thus, core-periphery organization is an optimal model that can be used to understand neuropsychiatric disorders (for a review see Bassett *et al.*[30]) and neurodevelopmental disorders.

Core-periphery abnormalities are observed in domain-general disorders (e.g. schizophrenia, ASD, depression), emerging early, prior to the onset of clinical symptomology.[17,30] The findings reviewed above reveal that domain-specific neurodevelopmental disorders are linked to reduced connectivity between lower-level, domain-specific perceptual processing regions and higher-level processing networks (in addition to variation in the lower-level processing units themselves). This suggests that domain-specific disorders are a consequence of a developmental abnormality in the connectivity 'between' the core and periphery, rather than dysfunction of the core itself (as proposed for severe neuropsychiatric disorders[30]). More specifically, network-level abnormalities associated with domain-specific neurodevelopmental disorders emerge from abnormal recurrent back-projections from the high-level processing networks (primarily within association cortex) that make up the periphery, into core perceptual regions (e.g. visual or auditory cortex). Connections in networks may consequentially reconfigure to optimize functioning within core-periphery organizational constraints, as is known to occur across distinct kinds of pathology[17] (see Fig. 1E).

Harnessing connectivity in the transdiagnostic approach

The core-deficit hypothesis[25] (see Box 3) attempts to account for multifaceted neurobiological phenomena with a single and specific mechanistic impairment, such as the phonological deficit model of dyslexia.[107] (The term 'core' as used here is distinct from the above-described core-periphery model.) Yet such attempts to account for specific neurodevelopmental disorders via a single mechanistic core deficit have been unsuccessful. Accordingly, findings that

Box 2 Network neuroscience and the core-periphery model.

Advances in neuroimaging methodologies have allowed for the modeling of structural and functional connections across brain regions that have been applied to the identification of brain networks supporting complex behaviors. Fig. 1E provides a schematic illustration of core-periphery network organization[30] applied to neurodevelopmental disorders. Network neuroscience models draw upon graph-theoretic frameworks whereby system elements or nodes supporting local processing are connected by edges. Nodes and edges combine to form network communities, linked by highly connected nodes in the brain network that occupy central positions in the overall organization of the network (i.e. hub nodes).[99] Networks tend to minimize the cost of wiring by forming locally dense clusters of nodes, referred to as 'modules' that are highly connected to each other, but sparsely connected to other clusters .[100] Modularity within the brain reflects the anatomical underpinnings of distinct functional systems that support information segregation. Across development, connectivity within modules strengthens while connectivity between modules weakens, with the exception of the strengthening of select hub edges linking modules.[101] Network modules thus become more distinct across developmental time. This integrated modular topology present in adulthood supports functional specialization of brain networks across distinct areas of cognition. While network segregation is beneficial from a physical and metabolic cost perspective, networks must also contain attributes that support integration for global communication within the network. Long-range connections, typically passing through multiple hub regions, enable efficient communication between brain modules that are spatially and functionally distinct. Hub regions tend to be densely connected to each other, forming a central 'core' (sometimes referred to as a 'rich club') within a network.[102] A network composed of a core made up of densely connected hubs, and a periphery of low-degree nodes that preferentially connect to the core is said to have core-periphery organization. Developmental disorders are characterized by weaker core-periphery connectivity, with instances of stronger core-periphery connections due to compensatory network reorganization, possibly accounting for the presence of extreme intra-individual profiles of strengths and weaknesses.

Box 3 The core-deficit hypothesis.

Children exhibit a complex combination of relative strengths and weaknesses across a wide range of cognitive domains. A developmental learning disability is diagnosed when a specific weakness is isolated (e.g. dyslexia in the case of low reading achievement). However, some children who reach the diagnostic criteria for multiple neurodevelopmental disorders are never formally diagnosed (e.g. Pearson,[62]Bathelt *et al.*,[105] Siugzdaite *et al.*[106]). This may be because the 'core-deficit' hypothesis is foundational to developmental psychology research, particularly research on neurodevelopmental disorders.[25] The core-deficit hypothesis posits that a single mechanistic impairment explains all observed cognitive and neural profiles within a particular diagnostic category. An example of the core-deficit model within the reading domain is phonological deficit theory, which argues that children with a reading impairment selectively struggle with phonemic awareness.[107] In the mathematical domain, the core-deficit model holds that developmental dyscalculia is a consequence of a deficiency within an evolutionarily ancient system specifically used to process quantities (i.e. the approximate number system).[108] This core-deficit account has been promoted across multiple cognitive domains in spite of serious limitations, such as its inability to explain many aspects of disorders (e.g. comorbidities[109]). While a core-deficit model held promise to enhance understanding of complex behavioural phenomena by identifying fundamental cognitive or neural underpinnings, research supporting such models suffered from a range of methodological issues, including highly selective, small samples and measurements chosen using circular logic (e.g. using a phonological awareness task as the assessment tool for selecting a group of children who have dyslexia).[25] Neuroimaging methods used in developmental research drew upon studies of human adults that originated from research on focal brain damage. Empirical studies avoiding these pitfalls (e.g. Siugzdaite *et al.*[106]) challenge the core-deficit model. Moreover, the core-deficit hypothesis cannot explain paradoxical advantages that are consistently reported in individuals with neurodevelopmental disorders.[110] As a result of these limitations, researchers are embracing larger, diverse samples, a broader array of assessment methods, and network models of brain function (e.g. Bulthé *et al.*[87]).

specific neurodevelopmental disorders are underpinned by encapsulated neural regions or circuits are subtle and difficult to reproduce. Instead, researchers have proposed that developmental disorders should be reconceptualized as 'few specific disorders and no specific brain regions'.[26]

Our review suggests that while there is dysfunction within specific regions associated with neurodevelopmental disorders, a 'common feature' of domain-specific neurodevelopmental disorders is disruption in long-range projections between regions in the core to regions in the periphery, such that intact lower-level processing fails to ignite conscious awareness.[105,111] To the extent that this impaired conscious access is common across diverse neurodevelopmental disorders, domain-specific effects may be a product of the research approach (i.e. restricting samples to a single diagnosis) and therefore illusory, differing only in location and not in fundamental mechanisms.

Viewing neurodevelopmental disorders through a network lens helps to explain comorbidities that occur as a consequence of network disorganization.[106] Cognitive profiles of children with a wide range of neurodevelopmental disorders (i.e. both domain-general and domain-specific) including ADHD and ASD, but also dyslexia and selective language impairments, are associated with the connectedness of neural hubs as opposed to a one-to-one mapping between cognition and brain activation.[106] Children with comorbid dyslexia and dyscalculia can be distinguished from typically developing children and children with only dyslexia 'or' dyscalculia by structural aberrations within medial temporal lobe, and reductions in functional connectivity in circuits linking the medial temporal lobe to domain-specific regions critical for reading and math, respectively.[112] Relatedly, artificial neural networks can identify data-driven neurocognitive dimensions underlying learning difficulties that are unrelated to diagnoses but reflect distinct patterns of brain organization.[113] Future research is needed to implement recently developed model-based approaches to uncover whether connectivity between core and particular periphery modules of brain networks account for differences between diagnostic groups of individuals with domain-specific neurodevelopmental disorders.

Reconceptualizing neurodevelopmental disorders through the lens of network neuroscience also provides insight into 'paradoxical' advantages exhibited by certain individuals with domain-specific developmental disorders (i.e. unexplained advantages in domains that are seemingly unrelated to the individual's disorder),[110,114] broadening the perspective beyond disability to the proposed profile-based interpretation of strengths and weaknesses.

Network competition, such as a computational trade-off between encoding specific details of an individual experience and extracting regularities across experiences,[115,116] suggests that a deficit in one network may be accompanied by additional resource allocation to a distinct compensatory network. There are several examples of extreme strengths observed in individuals with domain-specific neurodevelopmental disorders. Within the mnemonic domain, individuals with SDAM and aphantasia display strengths in non-episodic processes (e.g. extracting meaning or regularities, enabling rapid and efficient acquisition of concepts with reduced interference from specific episodes) and are over-represented in high-level scientific professions.[114] Within the academic domain, children with specific learning disorders sometimes exhibit paradoxical strengths in other areas of learning (e.g. individuals with dyslexia are gifted in other domains,[110] (see also Eberi *et al.*[117]). The presence of extreme high and low abilities within the same individual is a consequence of competition and parallel processing between distributed systems that undergo network-level reorganization with the goal of optimization, given a deficit, within the brain's core-periphery structure. Conceptualizing neurodevelopmental disorders under the guiding principles of network neuroscience and core-periphery organization enhances the potential for these ideas to support learners across the full spectrum of ability, rather than only those with neurodevelopmental disorders.

The identification of 'learning styles' (e.g. visual versus auditory learners[118]), an early profile-based approach in learning theory, has been dismissed as a 'neuromyth' due to a lack of scientific evidence.[119] However, the notion that people have different profiles of cognitive strengths and weaknesses is—at least intuitively—self-evident. Indeed, it may be the intuitive appeal of learning styles—and the fact that they were originally identified in relation to focal

lesion syndromes and not a neurodevelopmental framework—that led to an oversimplification of underlying neuro-anatomical mechanisms and overselling of related diagnostic and interventional products. A more nuanced anatomically-based approach to profiles would provide the basis for evidence-based diagnosis and intervention as well as contribution to theory across a wide range of abilities according to one's unique cognitive profile.[113,120]

Conclusion

Historically, neurodevelopmental disorders have been examined within, not across, cognitive domains. Synthesizing functional and structural connectivity findings across neurodevelopmental disorders suggests that distinct neurodevelopmental disorders are characterized by reductions in structural and functional connectivity between lower-level perceptual processing modules and higher-level control networks. The core-periphery network provides a useful framework for interpreting these findings. Specifically, seemingly distinct neurodevelopmental disorders share a dysfunction in long-range connections between the core and periphery of the human connectome. Implementing a network approach to identify a common origin of neurodevelopmental disorders across content domains may explain features such as comorbidity and paradoxical advantages within individuals in ways not evident from analyses focusing on surface-level differences in specific content domains. Such an approach would help to define a 'connectome landscape of brain dysconnectivity',[17] valuable for the development and implementation of individualized prevention and intervention methodologies to support atypical learners across development.

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Competing interests

The authors report no competing interests.

References

1. Beckman L, Janson S, von Kobyletzki L. Associations between neurodevelopmental disorders and factors related to school, health, and social interaction in schoolchildren: results from a Swedish population-based survey. *Disabil Health J*. 2016;9:663-672.
2. Augustine L, Lygnegård F, Granlund M. Trajectories of participation, mental health, and mental health problems in adolescents with self-reported neurodevelopmental disorders. *Disabil Rehabil*. 2021;5:1595-1608.
3. Olusanya BO, Davis AC, Wertlieb D, *et al*. Developmental disabilities among children younger than;5, years in 195 countries and territories, 1990–2016: a systematic analysis for the global burden of disease study 2016. *Lancet Glob Heal*. 2018;6:e1100-e1121.
4. Shahat ARS, Greco G. The economic costs of childhood disability: a literature review. *Int J Environ Res Public Health*. 2021;18:3531.
5. Raouafi S, Achiche S, Raison M. Socioeconomic disparities and difficulties to access to healthcare services among Canadian children with neurodevelopmental disorders and disabilities. *Epidemiol Health*. 2018;40:539-547.
6. Elliott ML, Knodt AR, Cooke M, *et al*. General functional connectivity: shared features of resting-state and task fMRI drive reliable and heritable individual differences in functional brain networks. *Neuroimage*. 2019;189:516-532.
7. Becht AI, Mills KL. Modeling individual differences in brain development. *Biol Psychiatry*. 2020;88:63-69.
8. Tavor I, Parker Jones O, Mars RB, Smith SM, Behrens TE, Jbabdi S. Task-free MRI predicts individual differences in brain activity during task performance. *Science* 2016;352:216-220.
9. Seitzman BA, Gratton C, Laumann TO, *et al*. Trait-like variants in human functional brain networks. *Proc Natl Acad Sci U S A*. 2019;116:22851-22861.
10. Bertolero MA, Thomas Yeo BT, D'Esposito M. The modular and integrative functional architecture of the human brain. *Proc Natl Acad Sci U S A*. 2015;112:E6798-E6807.
11. Seeley WW, Crawford RK, Zhou J, Miller BL, Greicius MD. Neurodegenerative diseases target large-scale human brain networks. *Neuron*. 2009;62:42-52.
12. Seeley WW, Carlin DA, Allman JM, *et al*. Early frontotemporal dementia targets neurons unique to apes and humans. *Ann Neurol*. 2006;60:660-667.
13. Buckner RL, Snyder AZ, Shannon BJ, *et al*. Molecular, structural, and functional characterization of Alzheimer's disease: Evidence for a relationship between default activity, amyloid, and memory. *J Neurosci*. 2005;25:7709-7717.
14. Drzezga A. The network degeneration hypothesis: Spread of neurodegenerative patterns along neuronal brain networks. *J Nucl Med*. 2018;59:1645-1648.
15. Zhou J, Gennatas ED, Kramer JH, Miller BL, Seeley WW. Predicting regional neurodegeneration from the healthy brain functional connectome. *Neuron*. 2012;73:1216-1227.
16. Raj A, Kuceyeski A, Weiner M. A network diffusion model of disease progression in dementia. *Neuron*. 2012;73:1204-1215.
17. van den Heuvel MP, Sporns O. A cross-disorder connectome landscape of brain dysconnectivity. *Nat Rev Neurosci*. 2019;20:435-446.
18. Jones DT, Knopman DS, Gunter JL, *et al*. Cascading network failure across the Alzheimer's disease spectrum. *Brain*. 2016;139:547-562.
19. Jones DT, Graff-Radford J, Lowe VJ, *et al*. Tau, amyloid, and cascading network failure across the Alzheimer's disease spectrum. *Cortex*. 2017;97:143-159.
20. Iturria-Medina Y, Evans AC. On the central role of brain connectivity in neurodegenerative disease progression. *Front Aging Neurosci*. 2015;7:90.
21. Mesulam MM, Weintraub S. Spectrum of primary progressive aphasia. *Baillieres Clin Neurol*. 1992;1:583-609.
22. Spreng RN, Rosen HJ, Strother S, *et al*. Occupation attributes relate to location of atrophy in frontotemporal lobar degeneration. *Neuropsychologia*. 2010;48:3634-3641.
23. Rohrer JD, Nicholas JM, Cash DM, *et al*. Presymptomatic cognitive and neuroanatomical changes in genetic frontotemporal dementia in the genetic frontotemporal dementia initiative

(GENFI) study: a cross-sectional analysis. *Lancet Neurol*. 2015; 14:253-262.
24. Bassett DS, Wymbs NF, Rombach MP, Porter MA, Mucha PJ, Grafton ST. Task-based core-periphery organization of human brain dynamics. *PLoS Comput Biol*. 2013;9:e1003171.
25. Astle DE, Fletcher-Watson S. Beyond the dore-ceficit hypothesis in developmental disorders. *Curr Dir Psychol Sci*. 2020;29: 431-437.
26. Thomas MSC. Developmental disorders: few specific disorders and no specific brain regions. *Curr Biol*. 2020;30:R304-R306.
27. Zhang F, Wu Y, Norton I, *et al*. An anatomically curated fiber clustering white matter atlas for consistent white matter tract parcellation across the lifespan. *Neuroimage*. 2018;179:429-447.
28. Norton I, Essayed WI, Zhang F, *et al*. SlicerDMRI: open source diffusion MRI software for brain cancer research. *Cancer Res*. 2017;77:e101-e103.
29. Zhang F, Noh T, Juvekar P, *et al*. SlicerDMRI: diffusion MRI and tractography research software for brain cancer surgery planning and visualization. *JCO Clin Cancer Informatics*. 2020;4: 299-309.
30. Bassett DS, Xia CH, Satterthwaite TD. Understanding the emergence of neuropsychiatric disorders with network neuroscience. *Biol Psychiatry Cogn Neurosci Neuroimaging*. 2018;3: 742-753.
31. Zhao Y, Zhen Z, Liu X, Song Y, Liu J. The neural network for face recognition: insights from an fMRI study on developmental prosopagnosia. *Neuroimage*. 2018;169:151-161.
32. Thomas C, Avidan G, Humphreys K, Jung KJ, Gao F, Behrmann M. Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia. *Nat Neurosci*. 2009;12:29-31.
33. Eimer M, Gosling A, Duchaine B. Electrophysiological markers of covert face recognition in developmental prosopagnosia. *Brain*. 2012;135:542-554.
34. Avidan G, Tanzer M, Hadj-Bouziane F, Liu N, Ungerleider LG, Behrmann M. Selective dissociation between core and extended regions of the face processing network in congenital prosopagnosia. *Cereb Cortex*. 2014;24:1565-1578.
35. Lohse M, Garrido L, Driver J, Dolan RJ, Duchaine BC, Furl N. Effective connectivity from early visual cortex to posterior occipitotemporal face areas supports face selectivity and predicts developmental prosopagnosia. *J Neurosci*. 2016;36: 3821-3828.
36. Grossi D, Soricelli A, Ponari M, *et al*. Structural connectivity in a single case of progressive prosopagnosia: The role of the right inferior longitudinal fasciculus. *Cortex*. 2014;56:111-120.
37. Fox CJ, Iaria G, Barton JJS. Disconnection in prosopagnosia and face processing. *Cortex*. 2008;44:996-1009.
38. Corrow SL, Dalrymple KA, Bartn JJ. Prosopagnosia: current perspectives. *Eye Brain*. 2016;8:165-175.
39. Song Y, Zhu Q, Li J, Wang X, Liu J. Typical and atypical development of functional connectivity in the face network. *J Neurosci*. 2015;35:14624-14635.
40. Song S, Garrido L, Nagy Z, *et al*. Local but not long-range microstructural differences of the ventral temporal cortex in developmental prosopagnosia. *Neuropsychologia*. 2015;78:195-206.
41. Liu J, Wang M, Shi X, *et al*. Neural correlates of covert face processing: FMRI evidence from a prosopagnosic patient. *Cereb Cortex*. 2014;24:2081-2092.
42. Yuskaitis CJ, Parviz M, Loui P, Wan CY, Pearl PL. Neural mechanisms underlying musical pitch perception and clinical applications including developmental dyslexia. *Curr Neurol Neurosci Rep*. 2015;15:51.
43. Peretz I. Neurobiology of congenital amusia. *Trends Cogn Sci*. 2016;20:857-867.
44. Leveque Y, Fauvel B, Groussard M, *et al*. Altered intrinsic connectivity of the auditory cortex in congenital amusia. *J Neurophysiol*. 2016;116:88-97.
45. Albouy P, Mattout J, Sanchez G, Tillmann B, Caclin A. Altered retrieval of melodic information in congenital amusia: Insights from dynamic causal modeling of MEG data. *Front Hum Neurosci*. 2015;9:20.
46. Albouy P, Peretz I, Bermudez P, Zatorre RJ, Tillmann B, Caclin A. Specialized neural dynamics for verbal and tonal memory: FMRI evidence in congenital amusia. *Hum Brain Mapp*. 2019; 40:855-867.
47. Wang J, Zhang C, Wan S, Peng G. Is congenital amusia a disconnection syndrome? A study combining tract-and network-based analysis. *Front Hum Neurosci*. 2017;11:473.
48. Chen JL, Kumar S, Williamson VJ, Scholz J, Griffiths TD, Stewart L. Detection of the arcuate fasciculus in congenital amusia depends on the tractography algorithm. *Front Psychol*. 2015;6:9.
49. Zhao Y, Chen X, Zhong S, *et al*. Abnormal topological organization of the white matter network in mandarin speakers with congenital amusia. *Sci Rep* 2016;6:1-11.
50. Hyde KL, Zatorre RJ, Peretz I. Functional MRI evidence of an abnormal neural network for pitch processing in congenital amusia. *Cereb Cortex*. 2011;21:292-299.
51. Loui P, Zamm A, Schlaug G. Enhanced functional networks in absolute pitch. *Neuroimage*. 2012;63:632-640.
52. Diamond N, Levine B. The prefrontal cortex and human memory. In: Miller BL, Cummings Jeffrey L, eds. *The human frontal lobes: Functions disorders*. 3rd ed. The Guildford Press; 2018:137-157.
53. Fuentemilla L, Càmara E, Münte TF, *et al*. Individual differences in true and false memory retrieval are related to white matter brain microstructure. *J Neurosci*. 2009;29:8698-8703.
54. Schott BH, Niklas C, Kaufmann J, *et al*. Fiber density between rhinal cortex and activated ventrolateral prefrontal regions predicts episodic memory performance in humans. *Proc Natl Acad Sci USA* 2011;108.
55. McDermott KB KSK, Christ SE. Laboratory-based and autobiographical retrieval tasks differ substantially in their neural substrates. *Neuropsychologia*. 2009;47:2290-2298.
56. Palombo DJ, Sheldon S, Levine B. Individual differences in autobiographical memory. *Trends Cogn Sci*. 2018;22:583-597.
57. Fuentemilla L, Palombo DJ, Levine B. Gamma phase-synchrony in autobiographical memory: evidence from magnetoencephalography and severely deficient autobiographical memory. *Neuropsychologia*. 2018;110:7-13.
58. Santangelo V, Cavallina C, Colucci P, *et al*. Enhanced brain activity associated with memory access in highly superior autobiographical memory. *Proc Natl Acad Sci USA*. 2018;115: 7795-7800.
59. Rubin DC, Deffler SA, Umanath S. Scenes enable a sense of reliving: implications for autobiographical memory. *Cognition*. 2019;183:44-56.
60. Armson MJ, Diamond NB, Levesque L, Ryan JD, Levine B. Vividness of recollection is supported by eye movements in individuals with high, but not low trait autobiographical memory. *Cognition*. 2021;206:104487.
61. Keogh R, Pearson J. The blind mind: no sensory visual imagery in aphantasia. *Cortex*. 2018;105:53-60.
62. Pearson J. The human imagination: The cognitive neuroscience of visual mental imagery. *Nat Rev Neurosci*. 2019;20: 624-634.
63. Wicken M, Keogh R, Pearson J. *Proc Biol Sci*. 2021;288:20210267.
64. Keogh R, Bergmann J, Pearson J. Cortical excitability controls the strength of mental imagery. *Elife*. 2020;9:e50232.

65. Milton F, Fulford J, Dance C, *et al*. Behavioral and neural signatures of visual imagery vividness extremes: aphantasia versus hyperphantasia. *Cereb Cortex Commun*. 2021;2:tgab035.
66. Epstein RA, Patai EZ, Julian JB, Spiers HJ. The cognitive map in humans: spatial navigation and beyond. *Nat Neurosci*. 2017;20: 1504-1513.
67. Iaria G, Burles F. Developmental topographical disorientation. *Trends Cogn Sci*. 2016;20:720-722.
68. Iaria G, Barton JJS. Developmental topographical disorientation: a newly discovered cognitive disorder. *Exp Brain Res*. 2010;206:189-196.
69. Iaria G, Arnold AEGF, Burles F, *et al*. Developmental topographical disorientation and decreased hippocampal functional connectivity. *Hippocampus*. 2014;24:1364-1374.
70. Kim JG, Aminoff EM, Kastner S, Behrmann M. A neural basis for developmental topographic disorientation. *J Neurosci*. 2015;35: 12954-12969.
71. Conson M, Bianchini F, Quarantelli M, *et al*. Selective map-following navigation deficit: a new case of developmental topographical disorientation. *J Clin Exp Neuropsychol*. 2018;40: 940-950.
72. Arnold AEGF, Protzner AB, Bray S, Levy RM, Iaria G. Neural network configuration and efficiency underlies individual differences in spatial orientation ability. *J Cogn Neurosci*. 2014;26:380-394.
73. Sharma G, Gramann K, Chandra S, Singh V, Mittal AP. Brain connectivity during encoding and retrieval of spatial information: individual differences in navigation skills. *Brain Inform*. 2017;4:207-217.
74. Norton ES, Beach SD, Gabrieli JDE. Neurobiology of dyslexia. *Curr Opin Neurobiol*. 2015;30:73-78.
75. Wandell BA, Le RK. Diagnosing the neural circuitry of reading. *Neuron*. 2017;96:298-311.
76. Vandermosten M, Boets B, Wouters J, Ghesquière P. A qualitative and quantitative review of diffusion tensor imaging studies in reading and dyslexia. *Neurosci Biobehav Rev*. 2012;36:1532-1552.
77. Boets B, Op De Beeck H, Vandermosten M, *et al*. Intact but less accessible phonetic representations in adults with dyslexia. *Science* 342;2013:1251-1255.
78. Lebel C, Benischek A, Geeraert B, *et al*. Developmental trajectories of white matter structure in children with and without reading impairments. *Dev Cogn Neurosci*. 2019;36:100633.
79. Schurz M, Wimmer H, Richlan F, Ludersdorfer P, Klackl J, Kronbichler M. Resting-state and task-based functional brain connectivity in developmental dyslexia. *Cereb Cortex*. 2015;25: 3502-3514.
80. Finn ES, Shen X, Holahan JM, *et al*. Disruption of functional networks in dyslexia: A whole-brain, data-driven analysis of connectivity. *Biol Psychiatry*. 2014;76:397-404.
81. Müller-Axt C, Anwander A, von Kriegstein K. Altered structural connectivity of the left visual thalamus in developmental dyslexia. *Curr Biol*. 2017;27:3692-3698.e4.
82. Duncan GJ, Dowsett CJ, Claessens A, *et al*. School readiness and later achievement. *Dev Psychol*. 2007;43:1428-1446.
83. Peters L, De Smedt B. Arithmetic in the developing brain: a review of brain imaging studies. *Dev Cogn Neurosci*. 2018;30: 265-279.
84. Sokolowski HM, Fias W, Mousa A, Ansari D. Common and distinct brain regions in both parietal and frontal cortex support symbolic and nonsymbolic number processing in humans: a functional neuroimaging meta-analysis. *Neuroimage*. 2017; 146:376-394.
85. Moeller K, Willmes K, Klein E. A review on functional and structural brain connectivity in numerical cognition. *Front Hum Neurosci*. 2015;9:1-14.
86. Matejko AA, Ansari D. Drawing connections between white matter and numerical and mathematical cognition: A literature review. *Neurosci Biobehav Rev*. 2015;48:35-52.
87. Bulthé J, Prinsen J, Vanderauwera J, *et al*. Multi-method brain imaging reveals impaired representations of number as well as altered connectivity in adults with dyscalculia. *Neuroimage*. 2019;190:289-302.
88. Price GR, Holloway ID, Räsänen P, Vesterinen M, Ansari D. Impaired parietal magnitude processing in developmental dyscalculia. *Curr Biol*. 2007;17:R1042-3.
89. Kaufmann L, von Aster M, Göbel S, Marksteiner J, Klein E. Developmental dyscalculia in adults: Current issues and open questions for future research. *Lernen und Lernstörungen*. 2020;9:126-137.
90. Rosenberg-Lee M, Ashkenazi S, Chen T, Young CB, Geary DC, Menon V. Brain hyper-connectivity and operation-specific deficits during arithmetic problem solving in children with developmental dyscalculia. *Dev Sci*. 2015;18:351-372.
91. Jolles D, Ashkenazi S, Kochalka J, *et al*. Parietal hyper-connectivity, aberrant brain organization, and circuit-based biomarkers in children with mathematical disabilities. *Dev Sci*. 2016;19:613-631.
92. Fang Y, Li M, Mei M, Sun X, Han D. Characteristics of brain functional and structural connectivity in alexithymic students. *Neuropsychiatr Dis Treat*. 2018;14:2609-2615.
93. McLeod KR, Langevin LM, Goodyear BG, Dewey D. Functional connectivity of neural motor networks is disrupted in children with developmental coordination disorder and attention-deficit/hyperactivity disorder. *NeuroImage Clin*. 2014;4:566-575.
94. Berthier ML, Dávila G, Torres-Prioris MJ, *et al*. Developmental dynamic dysphasia: Are bilateral brain abnormalities a signature of inefficient neural plasticity? *Front Hum Neurosci*. 2020;14:73.
95. McIntosh AR. Towards a network theory of cortical areas. *Neural Netw*. 2000;13:861-870.
96. Deco G, Jirsa VK, McIntosh AR. Emerging concepts for the dynamical organization of resting-state activity in the brain. *Nat Rev Neurosci*. 2011;12:43-56.
97. Bassett DS, Sporns O. Network neuroscience. *Nat Neurosci*. 2017;20:353-364.
98. Cole MW, Bassett DS, Power JD, Braver TS, Petersen SE. Intrinsic and task-evoked network architectures of the human brain. *Neuron*. 2014;83:238-251.
99. van den Heuvel MP, Sporns O. Network hubs in the human brain. *Trends Cogn Sci*. 2013;17:683-696.
100. Sporns O, Betzel RF. Modular brain networks. *Annu Rev Psychol*. 2016;67:613-640.
101. Baum GL, Ciric R, Roalf DR, *et al*. Modular segregation of structural brain networks supports the development of executive function in youth. *Curr Biol*. 2017;27:1561-1572.e8.
102. van den Heuvel MP, Sporns O. Rich-club organization of the human connectome. *J Neurosci*. 2011;31:15775-15786.
103. Sydnor VJ, Larsen B, Bassett DS, *et al*. Neurodevelopment of the association cortices : patterns, mechanisms, and implications for psychopathology. *Neuron*. 2021;109:2820-2846.
104. Gu S, Xia CH, Ciric R, *et al*. Unifying the notions of modularity and core-periphery structure in functional brain networks during youth. *Cereb Cortex*. 2020;30:1087-1102.
105. Bathelt J, Gathercole SE, Butterfield S, Astle DE. Children's academic attainment is linked to the global organization of the white matter connectome. *Dev Sci*. 2018;21:e12662.
106. Siugzdaite R, Bathelt J, Holmes J, Astle DE. Transdiagnostic brain mapping in developmental disorders. *Curr Biol*. 2020;30: 1245-1257.e4.

107. Wagner RK, Torgesen JK. The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychol Bull*. 1987;101:192-212.
108. Mazzocco MMM, Feigenson L, Halberda J. Impaired acuity of the approximate number system underlies mathematical learning disability (dyscalculia). *Child Dev*. 2011;82:1224-1237.
109. Happé F, Ronald A, Plomin R. Time to give up on a single explanation for autism. *Nat Neurosci*. 2006;9:1218-1220.
110. Toffalini E, Pezzuti L, Cornoldi C. Einstein and dyslexia: is giftedness more frequent in children with a specific learning disorder than in typically developing children? *Intelligence*. 2017;62:175-179.
111. Montemayor C, Haladjian HH. Recurrent processing theory versus global neuronal workspace theory: a comment on 'the relationship between attention and consciousness: an expanded taxonomy and implications for "no-report" paradigms' by Pitts et al. *Philos Trans R Soc B Biol Sci*. 2019;374:20180517.
112. Skeide MA, Evans TM, Mei EZ, Abrams DA, Menon V. Neural signatures of co-occurring Reading and mathematical difficulties. *Dev Sci*. 2018;21:e12680.
113. Astle DE, Bathelt J, Team TC, Holmes J. Remapping the cognitive and neural profiles of children who struggle at school. *Dev Sci*. 2019;22:e12747.
114. Zeman A, Milton F, Della Sala S, *et al*. Phantasia–the psychological significance of lifelong visual imagery vividness extremes. *Cortex*. 2020;130:426-440.
115. Schapiro AC, Turk-Browne NB, Botvinick MM, Norman KA. Complementary learning systems within the hippocampus: A neural network modelling approach to reconciling episodic memory with statistical learning. *Philos Trans R Soc B Biol Sci*. 2017;372(1711):20160049.
116. Freedberg MV, Reeves JA, Fioriti CM, Murillo J, Voss JL, Wassermann EM. A direct test of competitive versus cooperative episodic–procedural network dynamics in human memory. *Cereb Cortex*. 2022;00:1-18.
117. Erbeli F, Peng P, Rice M. No evidence of creative benefit accompanying dyslexia: a meta-analysis. *J Learn Disabil*. 2022;55(3):242-253.
118. Gardner H. *Frames of Mind: The Theory of Multiple Intelligences*. Basic Books; 1983.
119. Newton PM, Salvi A. How common is belief in the learning styles neuromyth, and does it matter? A pragmatic systematic review. *Front Educ*. 2020;5:602451.
120. Sokolowski HM, Ansari D. Understanding the effects of education through the lens of biology. *NPJ Sci Learn*. 2018;3:17.