Development and Psychopathology (2025), **37**, 1095–1107
doi:10.1017/S0954579424000944

Regular Article

The typical and atypical developing mind: a common model

Andreas Demetriou[1], George Spanoudis[2] and Timothy C. Papadopoulos[2]

[1]Cyprus Academy of Sciences, Letters, and Arts, University of Cyprus, and University of Nicosia, Nicosia, Cyprus and [2]Department of Psychology & Center for Applied Neuroscience, University of Cyprus, Nicosia, Cyprus

Abstract

We present a theory of atypical development based on a developmental theory of the typical mind integrating developmental, cognitive, and psychometric theory and research. The paper comprises three parts. First, it outlines the theory of typical development. The theory postulates central cognitive mechanisms, such as relational integration, executive and inferential processes, and domain-specific processes underlying different environmental relations, such as visuospatial or quantitative relations. Cognitive development advances in cycles satisfying developmental priorities in mastering these systems, such as executive control from 2–6 years, inferential control from 7–11 years, and truth control from 12–18 years. Second, we discuss atypical development, showing how each neurodevelopmental disorder emerges from deficiencies in one or more of the processes comprising the architecture of the mind. Deficiencies in relational integration mechanisms, together with deficiencies in social understanding, yield autism spectrum disorder. Deficiencies in executive processes yield attention-deficit and hyperactivity disorder. Deficiencies in symbolic representation yield specialized learning difficulties, such as dyslexia and dyscalculia. Finally, we discuss clinical and educational implications, suggesting the importance of early diagnosis of malfunctioning in each of these dimensions and specific programs for their remediation.

Keywords: architecture of mind; atypical mind; cognitive development; neurodevelopmental disorders; typical mind

(Received 2 April 2024; accepted 5 April 2024; First Published online 10 May 2024)

Introduction

In search of a common model for typical and atypical development

Atypical development involves several neurodevelopmental disorders, such as autism spectrum disorder (ASD), attention-deficit and hyperactivity disorder (ADHD) and specific learning disabilities, which impede understanding, learning, and social relations. A common theory integrating typical and atypical development would improve our understanding of developmental issues (Cicchetti & Cohen, 1995; Cichetti & Tucker, 1994; Karmiloff-Smith, 1998, 2015; Thomas & Karmiloff-Smith, 2002). On the one hand, drawing on a theory of normal mental organization and development may direct researchers of psychopathology to map the underlying causes and developmental course of different psychopathological conditions more precisely. On the other hand, precisely mapping the psychopathology of different mental conditions may highlight the boundaries of different cognitive processes (Cicchetti & Cohen, 1995). So far, however, theories of atypical development are fragmented, drawing on theories of typical development in a specific domain. For instance, a lack of a Theory of Mind was considered to cause autism (Baron-Cohen, 2000). These theories lack a comprehensive frame that would place abnormalities under the perspective of the architecture and development of the mind and specify the strengths and weaknesses of each condition relative to the others. This paper presents a theory that comprehensively integrates developmental, cognitive, and psychometric research on the human mind (2018a; Demetriou & Spanoudis, 2018, Demetriou et al., 2017, 2018b, 2023) and extends it to atypical development, aiming to provide a common model for understanding the variability in development.

This theory represents the human mind as a network of modular and transmodular systems carrying different tasks of understanding and problem-solving. Modular systems serve specific purposes in interaction with the environment. For instance, perceptual systems specialize in processing specific physical information in the environment, abstracting specific patterns of relations in physical stimuli, such as color, size, and position. Central systems integrate over modular systems to capture stability amidst variation, fill in lags in information, plan action, and evaluate information and decisions. Overall functioning may be deficient if the contribution of any modular or transmodular system relative to a goal is deficient. Thus, each system in the architecture of the mind may be a dimension of typical or atypical development depending on its attainment level (Demetriou & Spanoudis, 2018; Demetriou et al., 2018a; 2018b, 2024).

The paper comprises three parts. First, we outline the theory for typical development. Second, we discuss atypical development, showing how neurodevelopmental disorders emerge from deficiencies in specific dimensions of typical development. Finally, we discuss clinical and educational implications.

Corresponding author: Andreas Demetriou; Email: ademetriou@ucy.ac.cy

Cite this article: Demetriou, A., Spanoudis, G., & Papadopoulos, T. C. (2025). The typical and atypical developing mind: a common model. *Development and Psychopathology* **37**: 1095–1107, https://doi.org/10.1017/S0954579424000944

Architecture of the typical mind

The mind is organized hierarchically. Specialized processes are grounded in perception, dealing with different types of relations in the environment. General processes integrate information within and across specialized systems. This architecture has been extensively tested in psychometric and developmental research (Demetriou et al., 1993, 2002, 2018a, 2023, 2024), and it aligns with current research on brain organization (Spanoudis & Demetriou, 2020). This architecture overlaps with the currently dominant psychometric model of the human mind, the Cattell–Horn–Carroll model, sharing the hierarchical conception of the mind. Both models assume the combined use of task-specific, domain-specific, and general processes in understanding (Carroll, 1993; Haier, 2017; Jensen, 1998).

Specialized problem-solving domains

Six specialized domains of thought are described: 1) *Categorical thought* forms categories about entities drawing on their similarities and differences, allowing mental economy in the organization and use of information. 2) *Spatial thought* processes the placement of objects in space relative to the thinker and one another, allowing efficient action. 3) *Causal thought* processes how changes in objects or events produce changes in other objects and events. 4) *Quantitative thought* processes numerosity and magnitudes, allowing precision in processing relations. 5) *Social thought* processes relationships with other individuals, dealing with actions, emotions, thoughts, intentions, and beliefs. 6) *Language* processes patterns of sound, allowing communication between individuals.

Specialized domains involve several types of processes: 1) Core processes rooted in perception abstract information from physical aspects of the environment, reflecting their current appearance: for example, visual similarity (e.g., color); spatial arrangement (e.g. further than, next to); magnitude or numerosity (e.g., subitization of sets up to 3–4 elements); transfer of force across objects (e.g., displacement because of physical contact); emotions or beliefs (e.g., facial expressions of mental states); words (e.g., sound patterns indicating objects), in the six domains, respectively. 2) Mental operations associated with each domain include sorting, mental rotation, arithmetic operations, experimentation, theory of mind, reading, and so on. Domain-specific operations emerge from interactions between core processes and general integration and inferential processes. For example, sorting organizes objects according to dimensions of perceptual similarity; arithmetic operations relate magnitudes; reading organizes visual signs according to word-relevant sound patterns. 3) Knowledge, beliefs, and skills crystallized over the years in each domain. Table 1 illustrates how processes in each domain relate to networks in the brain.

The emergence of mental operations from core operations in domains is symbolically biased toward symbol systems conducive to representing object properties and relations. Symbol systems may be personal and subjective, such as mental images, or collective and arbitrary, such as writing or mathematical symbolism. Symbol systems preserve object properties and relations to support thinking. Personal symbols are grounded in experience, which directly signifies meaning relative to object properties or relations, such as color similarity across objects, spatial arrangement in spatial reasoning, or magnitude relations in quantitative reasoning. Arbitrary symbol systems are learned by associating personal symbols that ground meaning in experience. Writing must be mapped onto oral language; number names must be mapped onto magnitude representations. These systems often facilitate the representation and processing of specific objects and relations more precisely than personal symbols. Mathematical symbolism is far more precise and flexible in representing quantitative relations than mental images (Demetriou et al., 1993). We show below that mastering arbitrary symbol systems may be a significant source of learning difficulties.

Central mechanisms

Relational integration

A cross-modal comparator/relational mechanism integrates properties across objects and events, searching for stability across variations in time and space, registering, representing, and tagging them for future use. In its simplest form, it is present in perception as a gain control mechanism involving variation, search, and integration processes (Buzsaky, 2019). Saccadic eye movements, head movements or multimodal perception produce multiple object representations which must be integrated in sake of object identification and recognition (Nanay, 2018). Gain control mechanisms coordinate representations across spaces abstracting object invariance (Ferguson & Cardin, 2020). Cognizance enables awareness of the objects of mental activity and mental processes (Dehaene, 2014; Demetriou et al., 2018b; Seth, 2021). It re-enacts and re-processes past experiences to compare and evaluate across experiences to optimize current or future understanding or action. Thus, alignment and abstraction are partly stimulus or association and partly cognizance-driven, operating as an integrated mechanism (AACog) to capture, interpret, and evaluate relations according to goals (Demetriou et al., 2018b). Metacognition (Efklides, 2008), Theory of Mind (Wellman, 2014), reflection (Dehaene, 2014), and self-concepts (Demetriou, 2000; Harter, 2012) are products of cognizance.

Executive functions

Executive functions (EFs) are rooted in the gain control mechanism above. Attention control is internalized gain control using relational and cognizance processes for goal-relevant selection of actions or mental processing (Demetriou et al., 2018a). Working memory (WM) preserves products of AACog operations in time to allow integration with current online or remembered information. EF are the strategic aspect of AACog because they enable focusing on stimuli or representations, inhibit attention to stimuli irrelevant to a goal, shift between them (Diamond, 2013; Zelazo, 2015), and maintain information for processing.

Inference

Forms of inference, inductive, analogical, and deductive reasoning, encode rules for the operation of AACog and handling its products. These rules constrain how relations may be searched, abstracted, interpreted, and evaluated. Integration across processes in understanding and problem-solving contributes to the development of reasoning, guiding how representations must be combined in chains of valid inference. The description of this development is beyond the present concerns. It suffices that inference gradually encodes statistical regularities in the environment into rule systems, optimizing alignment and abstraction. Cognizance is critical because it renders reasoning an object of reflection,

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

Table 1. Brain areas and networks associated with cognitive domains, processes, and networks in neurodevelopmental disorders

Cognitive System	Brain Areas and Networks	Networks affected in disorders
Categorical thought	"What pathway": Extends from PVC (V1) to (ITE)	
Visuospatial thought	"Where pathway": DVS extends from the PVC (V1) to the PP	*Aphantasia*
Depth and orientation perception	DM (visual area V3B) or KO	VISa cortex, particularly OT and OPa regions.
Distance or "further" relations	Place cells in the HP and the Er	Altered patterns of functional connectivity within the DMN
Egocentric spatial relations	PP	
Quantitative thought	ANG in the PaC	*Dyscalculia*
Larger than	Extends from the PVC (V1), processing visual features (e.g., lines and edges to higher-level visual areas)	Reduced activation/atypical functioning in IPS; atypical activation and connectivity between IPS and PFC; visual processing networks: OcC and FFG associated with written numerals and math symbols; VAN
	IT involved in object recognition	
	IPS processing size comparison	
Causal thought		
Causal perception	RH V5 visual cortex, registering causally interacting objects	
Causal reasoning	dlPFC	
Resolving cause & effect relations	ACC and pMFC	
Social thought		
Face recognition	Extends from IOG to FFA	
Social aspects of face (e.g., eye gaze, facial movements, indexing mental states)	TS AMYG, OFC, INS	
Evaluating the emotional significance of stimuli and modulating responses to them	ACC	
Detecting conflict and errors in social contexts	MNS	
Understanding others' intentions, emotions, and mental states		
Language		*Dyslexia*
Speech sound discrimination	AUDp in the STG Broca's area in lpFrL	The phonological system does not have the necessary resolution for recognizing letters; Deficient (reduced activation) reading network: lpSTG, phonological processing); lOT (including the VWFA for orthographic processing); lIFG for articulatory processing
Processing grammar, syntax, and speech production	Wernicke's area in lpSTG	
Understanding spoken and written language	AF, white matter tract connecting Broca's and Wernicke's areas	
Integrating language production and comprehension in processing complex syntactic structures		
Relational integration	Connectivity among the MTL, adjacent PaC, TPa and FL areas; DMN, SN, and CEN	
Executive function	dlPFC	*ADHD*
Central executive function	ACC	SN: AINS, dACC. VAN: TPJ, VFrC; CR; DMN, FSN, striatum
Attention control center	IPS	
Focus of attention	BG	
Attention filter	HP	
Cognizance		*Autism*
Self-referential processing, introspection, metacognition	mPFC, precuneus	DMN
Awareness and monitoring of ongoing cognitive processes related to goal-directed behavior	lPFC	Social brain network: FFA, STS, AMYG, OFC, and ACC; MNS, IFG, premotor, IPL

(*Continued*)

Table 1. (*Continued*)

Cognitive System	Brain Areas and Networks	Networks affected in disorders
Inference		
Representation of 1st order relations	PP	*Williams syndrome, Down syndrome, Familial Mental Retardation*
Integration of 1st order into 2nd order relations	PFCrl	
Interference control	PFCvl	

Note. The dorsolateral prefrontal cortex (dlPFC) is involved in executive decisions; the anterior cingulate cortex (ACC) is involved in attention control; the intraparietal sulcus (IPS) serves as a hub of activity or focus of attention; the basal ganglia (BG) is a subcortical region involved in channeling attention; and the hippocampus (HP) is a key structure among subcortical regions involved in consolidating new explicit memories; DVS = dorsal visual stream; PVC = primary visual cortex; ITE = inferior temporal cortex; PP = posterior parietal cortex; DM (visual area) = dorsomedial visual area V3B; KO = kinetic occipital region; HP = hippocampus; Er= entorhinal cortex; DMN = default mode network; OT = occipitotemporal; OPa = occipitoparietal; VISa = visual association cortex; ANG = angular gyrus; PaC = parietal cortex; IT = inferotemporal cortex; IPS = intraparietal sulcus; PFC = prefrontal cortex; OcC = occipital cortex; FFG = fusiform gyrus; VAN = ventral attention network; pMFC = posterior medial frontal cortex; FFA = fusiform face area; IOG = inferior occipital gyrus; TS = temporal sulcus; OFC = orbitofrontal cortex; AMYG = amygdala; INS = insula; MNS = mirror neuron system; AUDp = primary auditory cortex; STG = superior temporal gyrus; lpFrL = left posterior frontal lobe; VWFA = visual word form area; IFG = inferior frontal gyrus; AF = arcuate fasciculus; MTL = middle temporal lobe; CEN = central executive network; TPa = tempoparietal; SN = salience network; LPFC = lateral prefrontal cortex; TPJ = temporoparietal junction; VFrC = ventral frontal cortex; CR = cerebellum; FSN = frontostriatal network; IPL = inferior parietal lobe; PFCrl = rostrolateral prefrontal cortex; PFCvl = ventrolateral prefrontal cortex.

enabling explicit rule formation and selection as needed (Demetriou et al., 2018b). Mental models supporting inference (Johnson-Laird & Khemlani, 2013), rationality schemes carrying cultural standards for inference (Stanovich, 2011), and formalized reasoning rules (Moshman, 2011) are complementary frames for inference suffering in neurodevelopmental disorders for the reasons discussed below.

Learning and development

Developmental priorities and milestones

Learning enables organisms to capitalize on experience to deal with novelty. It is a cumulative process enhancing knowledge, skills, and problem-solving processes in each domain and the general systems. In domains, learning enhances and refines domain-relevant concepts and skills. In the general systems, learning improves AACog processes, rendering them increasingly goal-based, systematic, flexible, and exhaustive. Abstraction becomes inclusive and refined, building increasingly intertwined hierarchies of concepts and rules. Cognizance becomes increasingly differentiated and accurate, providing the knowledge base for selecting goal-relevant processes. Reasoning becomes increasingly precise in generalizing knowledge and experience, improving the predictive power of inductive, analogical, or deduction rules.

In psychometric theories of intelligence, learning is associated with general cognitive ability, *g*, which defines the upper level of complexity and abstraction that can be reached across processes. IQ is an accurate index of *g* (Jensen, 1998). In cognitive developmental theory, learning is associated with developmental level, akin to psychometric theory. It is assumed that ascending the levels of cognitive development enhances the scope and complexity of concepts that can be learned. Learning and development occur at all fronts involved in the architecture above. As learning accumulates across domains, *g* is reformed to integrate higher levels of control of mental processing (Demetriou et al., 2024).

In infancy, *g* is episodic, reflecting behavioral interactions with persons and objects. The mental space of *g* prioritizes aligning perceptions and actions for the sake of episodic control. Thus, the accuracy of gross and refined motor movements is a good index of *g* in infancy. From 2 to 6 years in early childhood, *g* is marked by attention control and representational and linguistic awareness. This is reflected in the fast learning of symbolic systems, such as language, number representations, drawing, and other relevant systems. In later childhood, from 7 to 11 years, controlling inference, a primary connector of representations, is the dominant priority. Thus, indicators of representational interlinking, such as inductive reasoning, awareness of inferential processes, and WM, mark *g* in this period. In adolescence, from 12 to 17 years, a significant priority is mastering processes that allow one to evaluate knowledge and decisions for cohesion, validity, and truth. This is often expressed in mastering the complex aspects of specific domains, such as mathematics (Demetriou et al., 2018a, 2023).

Interfacing typical and atypical development

Learning in specific domains is a function of the domains involved, the state of the central systems, and developmental readiness relative to the demands of the tasks to be learned. At the entry level, learning novel concepts and skills depends on the representational readiness of the domain involved to learn necessary symbols to allow AACog and inferential processes to construct necessary relations. This is a two-faced process.

On the one hand, it requires a minimum level of precision in the fundamental information delivered by domain-specific core processes, such as visual and/or acoustic object descriptors, perception of small numbers, cause–effect pairings, etc. Precision in these recordings is necessary for grounding arbitrary symbols, such as letters or number names, into meaningful mental units that would be further interrelated. Learning to read requires representing visual and sound symbols for letters and words (Papadopoulos et al., 2016). Learning arithmetic operations requires representing magnitudes and numbers, which can be mentally operated on (Koponen et al., 2013). Learning social skills and conventions requires representing one's and others' mental states, indexing attitudes, beliefs, and emotions driving social interactions. On the other hand, minimum efficiency is required in inter-relating fundamental core representations with symbol systems to build the network of relations and related rules at the task.

If any of these requirements fail, learning and development stall. In the first case, new symbols would not be appropriately learned *in the domain concerned*, impeding learning in this domain. Noticeably, domain-specific representational deficiencies together with relatively intact general relational processes would cause modular defects but not necessarily general defects because general processes may still be practiced in intact domains. Deficiencies in sound perception or script representation would impair learning to read (Franceschini et al., 2012). Deficiencies in

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

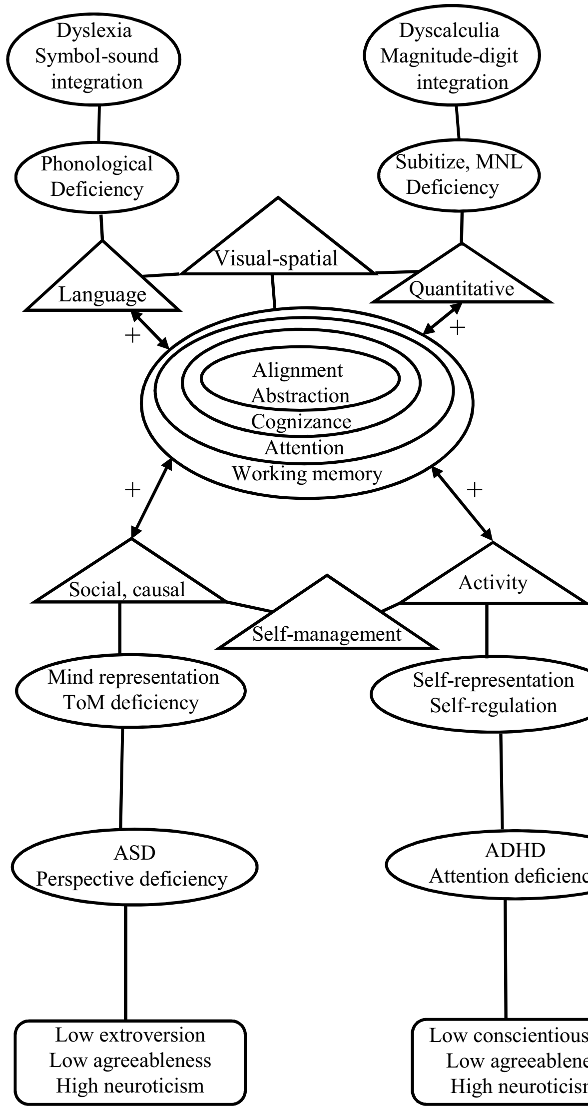

Figure 1. A schematic representation of the causal pathways underlying the neurodevelopmental disability explanatory model. *Note*. Plus sign (+) depicts additive effects. Bidirectional arrows depict the dynamic interplay among triangles (domains) and ovals (latent abilities).

magnitude representation would impair learning arithmetic (Butterworth, 2010). Self-directed attention deficits would impair goal focus (Nigg, 2001). Deficiencies in awareness would handicap attention-guided rule induction (Demetriou et al., 2021; Spanoudis & Demetriou, 2020). Deficiencies in understanding others' representations would impair interpersonal interactions (Baron-Cohen, 2020). In the second case, defects in central processes, such as relational integration, may cause generalized learning problems because hierarchies of relations would not be constructed even if entry-level representations are intact. However, there may be cases, such as the idiot savants, where the coexistence of an impeccable domain with severe central deficiencies may result in impressive performance in this specific domain.

The model presented here highlights initial background risk factors for neurodevelopmental disorders both functionally and developmentally. Each domain-specific or general dimension is a developmental pathway where development may turn atypical when deficiencies exceed a certain level, even before actual pathological conditions manifest themselves (Cicchetti & Cohen, 1995). Atypical development may be manifested at any developmental turning point when development falls short of the priorities of the phase concerned and the demands imposed by the environment. For instance, dyslexia or dyscalculia are specialized conditions emerging when deficiencies in core language-related or magnitude-related representations do not meet the demands of symbol learning imposed by school for learning to read or arithmetic. Autism emerges from deficiencies in repressing social cues and awareness of representations (Ball & Karmiloff-Smith, 2014; Pennington et al., 2019; Westermann & Mareschal, 2002). ADHD emerges from deficiencies in EFs. Deficiencies in general relational and integration processes may impede rule-based thought, even if representational capacities are intact across the board. These deficiencies may be more pervasive and long-lasting, as in the case of delayed thought development in Down syndrome. Figure 1 illustrates how different neuro-developmental disorders relate to general and domain-specific mechanisms.

The atypical mind

Some genetic conditions, such as Down or Williams syndrome (WS), are connected to specific genes that affect central mechanisms associated with brain organization and functioning, causing atypical physical, cognitive, and socio-emotional development (e.g., Cicchetti & Beeghly, 1990; Donnai & Karmiloff-Smith, 2000; Korbel et al., 2009; Paterson, 2009). Other genetic conditions, such as dyslexia, exert specialized effects, affecting domain-specific functions, such as language (Schumacher et al., 2007; van Bergen et al., 2023). Also, specific learning deficits may be associated with anomalies in particular brain networks associated with each deficit, although the direction of causality is often unclear. These networks are indicated in Table 1. The environment is crucial because it contextualizes genetic plans, channeling how they may be implemented and expressed in the brain (Ball et al., 2019).

We discuss four neurodevelopmental described in DSM-V (Straussner, 2013): two related to specific domains, that is, specific reading difficulties or dyslexia and dyscalculia and three involving more general or pervasive processes, such as ADHD, ASD, and Down syndrome. Two other disorders, namely, WS and aphantasia, are also discussed to complete the argument that there are conditions related to all aspects of mental architecture. We show that each disorder is associated with a specific pattern of deficiencies in brain networks and functions, which are known to be associated with networks and operations serving the various systems of the mental architecture described above (Table 1).

Developmental difficulties in specific domains

Around 20% of children in early primary school struggle with reading and writing, and approximately 5%–10% of them have dyslexia. Also, ∼5% of children face challenges in learning arithmetic and may develop dyscalculia (Reigosa-Crespo et al., 2012). These two conditions are distinct and relate to deficiencies in the perception and representation of information in each domain, although commonalities may exist in some individuals, ranging from ∼ 17%–26%. These may often relate to common underlying genetic and environmental factors rather than direct causal interactions (van Bergen, 2023).

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

Language difficulties and dyslexia

Developmental language disorder (DLD) and dyslexia are distinct conditions that involve difficulties with language processing (Spanoudis et al., 2019). DLD mainly affects oral language skills such as speaking and understanding, while dyslexia predominantly affects reading and written language abilities. Both conditions share similarities, such as phonological awareness and language comprehension deficits. However, dyslexia involves difficulties decoding written words and spelling, while DLD may cause broader communication difficulties beyond reading and writing. Both conditions can significantly impact academic performance and social interactions (Carroll & Snowling, 2004). The following section focuses on these primary conditions and discusses them in the abovementioned theory.

DLD affects core language and communication skills, such as syntax, semantics, or phonology. DLD arises from an impairment in learning language that affects both expressive and receptive systems (Leonard, 2014). Children with DLD have difficulty processing phonological information (Munson et al., 2005) and struggle with acquiring a mental lexicon (Moyle et al., 2007), using morpho-syntactic information (Rice et al., 2000), understanding pragmatics (Bishop & Norbury, 2002), retrieving text (Norbury & Bishop, 2003), and understanding sentence structure and semantics (Bishop & Norbury, 2002; Montgomery, 2004). Language impairments have high variability, which justifies classifying DLD children into separate subtypes related to specific causes (Marinis, 2015).

Dyslexia is more specific, impairing reading and related skills. The primary causes of dyslexia are phonological, naming speed (Papadopoulos et al., 2009), and WM deficits (Vellutino et al., 2004), which affect reading ability (O'Brien et al., 2012), spelling, or both (Moll & Landerl, 2009; Papadopoulos et al., 2021). Some individuals with dyslexia may exhibit deficits in processing speed, which can impact their ability to complete tasks efficiently (Georgiou et al., 2008; Swanson, 2015).

Reading and understanding grammar and syntax requires children to recognize recurring sound patterns, keep them in memory, and match them to corresponding representations already available. They must also identify similarities and differences between word forms, such as the everyday use of "-ed" to denote past tense (Dehaene et al., 2010). In reading difficulties, the phonological system does not have the necessary resolution for recognizing letters, composing words, or naming them quickly. This is due to a deficiency in the reading network, which includes the left posterior superior temporal gyrus (pSTG), responsible for phonological processing, the left occipitotemporal cortex (including the visual word form area, VWFA) for orthographic processing, and the left inferior frontal gyrus (IFG) for articulatory processing and WM. Brain activation in individuals with dyslexia appears more strongly manifested over the left frontocentral and center-parietal regions (Christoforou et al., 2022, 2023). Individuals with dyslexia exhibit reduced activation in these regions, contributing to difficulties in phonological processing, word recognition, and fluency (Dębska et al., 2021; Turker et al., 2023) (Table 1).

Dyscalculia

It was noted that circa ~ 5% of children having trouble in learning arithmetic may develop dyscalculia (Reigosa-Crespo et al., 2012). Dyscalculia makes it challenging to manipulate mathematical symbols, recall number-related facts and rules, and retrieve arithmetic data (Cardenas et al., 2021; Dehaene, 2011; Reeve & gray, 2014). These difficulties reflect deficits in magnitude comparison, counting strategies, identification of numbers, and arithmetic procedures. Butterworth (2005) proposed the "defective number module hypothesis", implying that dyscalculia is caused by difficulty in coding numerosity, that is, the process of linking symbols to representations of quantities: represent number "one" as a quantity of one, "two" as a quantity of two, and so on. A deficit in coding numerosity makes it hard to learn counting because counting words would not be linked to their corresponding representations. As a result, children with dyscalculia cannot construct holistic number schemes enabling the processing of quantitative relations, such as the Approximate Number System, which allows intuitive estimations of quantities and the Mental Number Line, which facilitates comparisons between numbers and magnitudes (Mussolin et al., 2010).

Challenges and manifestations vary among individuals with dyscalculia; the weak representation of numerical magnitude limits some; others have trouble accessing and manipulating numerical quantities (Price et al., 2007). Additionally, children with dyscalculia have difficulty learning Arabic numerals, number words, and their meanings (Chu et al., 2016). This is manifested in difficulty with simple tasks such as counting small sets up to 9 elements, comparing small magnitudes, such as 5 to 7, or performing simple mental arithmetic by adding or subtracting numbers from 1 to 9. These difficulties must be discriminated from deficiencies in mathematical learning caused by impairments in general processes, such as attention control and WM (Gersten et al., 2005; Peng & Fuchs, 2016; Peng et al., 2018; Swanson & Jerman, 2006).

Several brain networks related to number representation and processing are deficient in individuals with dyscalculia (Table 1). They exhibit reduced activation and atypical functioning in the IPS and atypical activation and connectivity between the IPS and prefrontal cortex (PFC), both implicated in number processing (Rosenberg-Lee et al., 2015). Dyscalculics also display alterations in visual processing networks, including the occipital cortex and the fusiform gyrus, which are involved in processing written numerals and mathematical symbols. The ventral attention network, another central network for mathematics, is also altered in individuals with dyscalculia (Kucian, 2016).

Noticeably, dyscalculia does not affect linguistic coding, which is affected by dyslexia. Dyscalculics have difficulty in associating Arabic numerals with their magnitudes, but they do not in associating letters with phonemes; individuals with dyslexia find it hard to recognize and name letters and digits, but they have no problem with magnitude processing, symbolic or non-symbolic (Rubinsten & Henik, 2006). However, some children may face difficulties in both domains (Landerl et al., 2009; Wang et al., 2012).

Aphantasia. Mental images are subjective perceptual experiences of stimuli not currently perceived or voluntarily generated modality-specific representations of stimuli with previously experienced features or novel combinations of features (Kosslyn et al., 2006). These mental images produce the subjective perceptual experience of the imagined stimuli (Pearson et al., 2015). Individuals with aphantasia cannot create mental images voluntarily, even though their perception and memory are normal. Some individuals report that they cannot generate mental images, while they can describe their perceptual experiences verbally. Aphantasia is not a neurodevelopmental disorder like the disorders discussed before, and it is broader than the specific difficulties faced by individuals with dyslexia or dyscalculia in visualizing written letters or magnitudes.

Individuals with aphantasia may display reduced activation or atypical functioning in the visual association cortex, particularly in the occipitotemporal and occipitoparietal regions involved in higher-level visual processing, including object recognition and scene construction. Moreover, some studies reported altered patterns of functional connectivity within the default mode network (DMN in individuals with aphantasia, perhaps associated with difficulties in generating mental images in tasks requiring the DMN (Keogh et al., 2021; Milton et al., 2021).

Developmental disabilities in central processes

ADHD primarily affects EFs rather than AACog processes. ASD involves the relational integration and awareness processes of the AACog mechanism together with domain-specific processes, such as social understanding. Cognitive, social, and behavioral weaknesses associated with these disabilities are more widespread than domain-specific difficulties because of the role of general mechanisms in the functioning of domain-specific mechanisms. We discuss ADHD and ASD in detail below.

Attention-deficit/hyperactivity disorder

ADHD is characterized by persistent inattention, hyperactivity, and impulsivity or disinhibitory control (Barkley, 1997). Children with ADHD are easily distracted, have difficulty sustaining focus, and struggle with planning and organization (Papadopoulos et al., 2005). They forget critical details, find it difficult to follow instructions and fail to complete tasks. They may engage in excessive talk, have difficulty being quiet, and struggle to engage in leisure activities. These difficulties may be socially disruptive.

There is only a limited overlap between the brain networks affected by ADHD and ASD (Wang et al., 2021). In ADHD, some networks connected to attention are affected, such as the salience network, including the anterior insula and dorsal anterior cingulate cortex (dACC) (Sutcubasi et al., 2020). This network is involved in detecting and orienting attention to salient stimuli. The Ventral Attention Network (VAN), including the temporoparietal junction (TPJ) and the ventral frontal cortex, is also affected (Table 1). This network is related to shifting attention because it reorients attention to unexpected stimuli. The cerebellum, associated with motor control, is also affected. Although relational integration processes may be spared in ADHD, they may not function properly because they are deprived of required information not attended to, compromising integration. The DMN, which is associated with self-awareness, and the frontostriatal network, which includes the PFC and the striatum (caudate nucleus and putamen) and is related to planning, cognitive control, and WM is also affected in ADHD. However, individuals with ADHD have only slightly lower IQ scores (about 3 points) than typically developing individuals; this difference is not clinically significant (Bridgett & Walker, 2006). The severity of symptoms may vary across ADHD cases and age, perhaps because of delays in cortical maturation (Shaw et al., 2007).

Autism spectrum disorder

ASD relates to difficulties in all core processes in AACog. Perceptual difficulties impair search, alignment, and abstraction processes. Perception in ASD individuals is strongly attracted by local details or featural information, failing to systematically scan and align features in search of an underlying theme. Happe (1999) proposed the Weak Central Coherence theory to account for the dominant symptoms of ASD. This is "*a processing bias for featural and local information, and relative failure to extract gist or "see the big picture" in everyday life.*" (Happe & Frith, 2006, p. 6). The inability to experience wholes without full attention to the constituent parts and a persistent preoccupation with parts of objects stand as diagnostic criteria for ASD (DSM-IV, Lewis, 1996). Relatedly, autistic children are inept in attending to, recognizing, and remembering human faces, compromising social interactions (Behrmann et al., 2006).

Difficulties in alignment and abstraction coexist with severe problems in cognizance, hindering Theory of Mind (ToM) and mental awareness (Baron-Cohen, 2000) and hampering social communication and social interaction in ASD. Deficiencies in awareness of the representational nature of mind and human experience disable persons with ASD from considering others' perspectives and negotiating differing points of view, beliefs, and intentions.

The DMN is impaired in ASD, too (Nair et al., 2020). The DMN includes the medial prefrontal cortex (mPFC), posterior cingulate cortex, and angular gyrus, active during rest and self-referential thinking. Areas in the social brain network which are critical for processing social information, such as the fusiform face area (FFA), superior temporal sulcus, amygdala, orbitofrontal cortex (OFC), and anterior cingulate cortex (ACC), also show atypical activation patterns during social tasks in ASD, such as face processing or ToM tasks (Cheng et al., 2015). Altered activation and connectivity within the Mirror Neuron System (MNS) were also reported (Chan & Han, 2020; Perkins et al., 2010; Rizzolatti et al., 2002); the MNS is involved in understanding and imitating the actions and emotions of others (Iacoboni & Dapretto, 2006) (Table 1).

These deficiencies cause self-isolation and social friction when interacting with others. Children with autism have difficulty developing and maintaining social relationships, including making eye contact and engaging in back-and-forth conversations. It is also difficult for them to understand social cues, such as facial expressions and body language, as signs indicating the perspectives, beliefs, and motives of others. Playing imaginatively or demonstrating pretend skills is also complex for children with ASD (Kasari et al., 2013).

Deficiencies in the perceptual alignment and abstraction processes appear early in infancy when these processes are dominant developmental priorities (Shao & Gentner, 2022). Sensory sensitivities may also be displayed, including an aversion to loud noises or the texture of things. Deficiencies in cognizance involving ToM and perceptual awareness appear later, in 3-4 years, when they dominate as priorities. Unless diagnosed and treated, these deficiencies multiply with age (Dahiya et al., 2020; Hudry et al., 2021). Specifically, ASD individuals experience difficulties managing attention because it is captured by interests in specific activities. ASD children often have a limited range of interests, focusing on a particular topic or object and showing little interest in other activities. The overall profile of difficulties above is reflected in considerably lower-than-average IQ in most individuals with ASD (by 20–30 points) (Charman, Pickles, et al., 2011; Fombonne, 2003). However, many ASD individuals have average or superior IQ (Charman et al., 2011).

Disorders affecting relational integration and inference. Several disorders affect relational processing as such. One of these disorders, Williams Syndrome (WS), is a rare neurodevelopmental condition caused by a genetic deletion on chromosome 7. The condition leads to various cognitive, social, and physical characteristics. Individuals with WS typically have an IQ between 50 and 70, implying severe problems in relational processing. Notably, the frontoparietal network related to relational integration is impaired in WS. Also, individuals with WS have impaired visuospatial and mathematical skills, perhaps because the dorsal stream, known as the "where" pathway, is impaired. This pathway is responsible for visual processing, extending from the primary visual cortex to the parietal cortex, and involves spatial processing, motion perception, and visuomotor integration (Donnai & Karmiloff-Smith, 2000; Karmiloff-Smith et al., 2018). Notably, verbal and social skills are not affected. WS individuals show atypical activation within the ventral stream. This "what" pathway extends from the primary visual cortex to the temporal cortex and involves object recognition and face processing. This may relate to their interest in faces and social stimuli (Karmiloff-Smith, 1997).

Down syndrome is another genetic condition caused by an extra copy of chromosome 21 (trisomy 21). Most individuals with Down syndrome present a degree of intellectual disability, with an IQ ranging from 40 to 70 (Cicchetti & Beeghly, 1990). Interestingly, the IQ of these children declines after middle childhood, indicating failure to develop and consolidate rule- or principle-based thought requiring advanced analogical and deductive reasoning (Carr, 2005). Notably, it was found decades ago "that retarded and nonretarded persons traverse the same stages of development in the same order, differing only in the rate at which they progress and in the ultimate ceiling they attain, . . . regardless of etiology, with the possible exception of individuals suffering from pronounced EEG abnormalities" (Weiss & Zigler, 1979, p. 846).

Shared and distinct architectural and developmental aspects

Dimensions of atypical development

This paper presented a theory aiming to account for typical and atypical development in the same theoretical framework. The theory describes a cognitive architecture comprising local and central systems. Local systems provide initial interpretations of objects and their relations and build advanced domain-specific concepts and skills in interaction with central systems. Central systems compute relations within or across local systems, checking for consistency of experience across them to optimize understanding or action. The relative autonomy of local systems is meaningful because it allows efficiency in the initial processing of different types of information. Central controls are needed to deal with inconsistencies in incoming data, which require integration for better choices. In a modular system enjoying integration at multiple levels, things may go wrong for many reasons. Deficits in local modules would hamper performance in the domain affected and spread to other modules according to demands. Deficits in central systems may cause generalized deficiencies in local modules, even if they are intact, although often, modules may operate well. For instance, these deficits may compromise the homostatic self-regulatory structures of the mind, cascading in several directions (Cichetti & Tucker, 1994; Masten & Cicchetti, 2010).

Therefore, all modules may be dimensions of individual differences and atypical development. Modules depending on a specific symbol system are more prone to disorder. Language disability and dyslexia, dyscalculia, aphantasia, and ASD depend on linguistic, arithmetic, iconic, and mentalistic/social symbol systems, respectively. Therefore, deficiency in the operation of a symbol system is critical for a domain-specific disorder. Interestingly, two domains unrelated to a specific symbol system, categorical and causal thought, are unrelated to a particular neurodevelopmental condition. Relations in these domains may be represented by alternative symbolic means. When central systems operate sufficiently, relations in symbol-free systems may be constructed via alternative symbol systems, bypassing deficient ones. For instance, the same causal relation may be described verbally, may be visualized as an interaction of the factors involved, or defined as a mathematical relation. Also, if central systems are intact, overall learning may, in the long run, compensate for the effects of symbol-specific deficiencies. All disorders may coexist with average IQ, suggesting intact general cognitive mechanisms (Peng et al., 2020).

The effects of deficiencies in central systems are broader, but they still depend on the system involved and the possible involvement of domain-specific systems. For example, deficiency in relational integration in WS exerts a broad debilitating effect, generally causing low intelligence. Difficulties in information integration prevent learning across domains. Notably, symbolic aspects of language, such as grammar and syntax, are spared in WS, but relational language is compromised (Mervis & Velleman, 2011). In ADHD, executive rather than AACog processes are deficient. As a result, total IQ is generally normal in ADHD despite problems in various domains which need focused, effortful, and time-dedicated learning, such as mathematics. Awareness appears compromised in ADHD, but this is secondary, arising from a lack of stable representations that the mind's eye can turn to because of low attention span. Deficiencies in ASD are broader because they involve all aspects of the AACog mechanisms and the social domain. Thus, overall relational integration is compromised because information intake is fragmented, yielding low-quality information for integration.

Similarly, in actual life, both ADHD and ASD may fail to recognize mental states, empathize with others, and adjust one's mental state to others, but the reasons are different. In ADHD, the mental states of others may be understood, but they are not noted because they are not attended to. In ASD, mental states cannot be interpreted because they are incomplete. Thus, social impairments in the two conditions are associated with different pathways, channeled by inattention, impulsivity, and hyperactivity in ADHD to social ineptness and stereotyped behavior in ASD (Sokolova et al., 2017).

Developmental aspects of atypical development

Failing to attain the priorities of some developmental cycles is more critical for atypical development than others. Priorities of representational thought, representational awareness, and executive control are critical for progression to the following cycles. Regardless of the system involved, individuals with symbolic difficulties may suffer consequences in various other aspects of mental functioning. Problems at any early level of cognitive functioning can cause problems at follow-up levels. Also, the higher the source of the problem, the broader the problem. For example, inappropriate attention or arousal disrupts planning, WM or processing speed, and achievement areas. Even if attention or arousal is within tolerable limits, planning problems could disrupt lower levels of processing. Similarly, if one or many general cognitive skills are weak, they can produce a particular learning

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

problem across achievement areas, with significant impairments in processing speed. For example, poor phonological processing could affect word decoding, resulting in overemphasizing visual cues in spelling and an inability to follow a plan in problem-solving. Finally, learning problems can give rise to secondary affective issues, which can feed back upon the higher levels of processing. Likewise, individuals may also face obstacles in developing the metacognitive skills required for self-evaluation and self-regulation, hampering problem-solving, conceptual change, and skill acquisition in different domains (Susac et al., 2014).

Difficulties in handling symbolic systems may affect reasoning, limiting flexibility in inference even for individuals attaining rule-based or principle-based thought. Many ASD individuals have average or high IQs in adolescence and adulthood (Charman et al., 2011). These individuals solve principle-based analogical and deductive reasoning problems, including fallacies (Green et al., 2014). However, they do not contextualize logical arguments with background information, failing to examine arguments from alternative points of view (McKenzie et al., 2010) and thus appearing less insightful in reasoning (Lewton et al., 2019). Therefore, what seems to be a weak central coherence (Happé & Frith, 2006) or complexity management (Williams et al., 2006) may reflect a residual difficulty in symbolic flexibility dated since the consolidation of representational thought. These considerations must be considered for the design of diagnostic and treatment programs addressed to the various disorders.

Diagnosis and treatment

Deficiencies must be diagnosed and treated in time to enhance children's abilities to learn and develop normally. Modern technologies bring the knowledge reflected in this paper closer to clinicians and teachers of children with learning difficulties. Artificial Intelligence (AI) and related technologies, such as virtual reality, must be integrated into the evaluation and treatment practices addressed to children with developmental disorders, connecting online developmental norms with children's performance. Likewise, mathematical models that analyze how learning occurs at a micro-level can help us understand behavior better. These models can capture the detailed sequence of events during an intervention or learning session. Studying this process can help us focus on the process of developing new strategies and skills rather than just the product (Christoforou et al., 2023).

In infancy, children at risk for various reasons must be evaluated for precursors, such as deficiencies in sound or visual perception or relational understanding, which may indicate proneness for mental disorders (Wolff & Piven, 2021). In kindergarten, assessment must address attention control, integration of symbolic elements (e.g., stylized pictures standing for objects, scripts for letters, numbers, and magnitudes), and understanding others' perspectives. In primary school, assessment must address rule induction to create concepts, the use of rules to organize objects, flexibility in shifting between contexts, and self-regulation according to strengths and weaknesses. In high school, assessment must evaluate how reasoning is used to check the truth, reliability, and accuracy of information, understand how epistemic or ideological contexts may constrain knowledge and beliefs, and how a differentiated self-concept is formed.

Remedial programs must be developmentally structured to enhance performance in general processes, processes in the symbol systems affected, and in using general processes to process specific information of interest to meet developmental priorities. In domain-specific disorders, instruction must enable different means to symbolize concepts and actions by means, such as language, images, and drawings, and noting their pros and cons would be necessary for mastering mental representation. For example, training aspects of phonological processing, such as grapheme-phoneme correspondence, word decoding, and spelling accuracy, benefits reading learning in children with reading difficulties (Lyytinen et al., 2009; Tilanus et al., 2019).

Similarly, interventions for dyscalculia should target enhancing number sense, arithmetic, and mathematical reasoning, which involve symbolic integration of numerical information. For instance, identifying number order randomly arranged exerts a generally beneficial effect on understanding numbers (Iseman & Naglieri, 2011; Park & Brannon, 2013). A program directed to the spatial representation of numbers and the use of the mental number line, associating representations of numbers and space and grasp of ordinality of numbers improves understanding of the relationship between magnitudes, accurate number representation, and comprehension of ordinality (Kucian et al., 2011).

Central processes must be addressed in ADHD and ASD. Interventions for ADHD must focus on improving attention, self-regulation, coping strategies, and functioning in various settings. These may indirectly improve symbolic integration. The treatment of ASD must support stimulus search and exploration, their mapping on each other according to different criteria, and their symbolic integration and representation. Interventions must also improve the functioning of ASD children in social situations by helping them learn and improve coping strategies and skills. For instance, interventions must practice interpersonal interactions requiring joint attention and shifting between objects and representations, turn-taking, and awareness of each other's mental representations. Also, training in using relatively intact aspects of reasoning, such as analogical reasoning, would compensate for weakness in the contextual and interpersonal embedding of information to be interpreted. This would improve social and communicative abilities in children with autism.

Conclusions

In conclusion, the following general principles hold as they integrate typical and atypical development.

1. Development in any specific domain depends on the state of processes specific to this domain, such as linguistic representation in reading or magnitude representation in mathematics. Therefore, development in this domain may stall if critical processes are deficient, even if general or other specific processes are intact.
2. Overall development is a function of the state of central processes. When central relational mechanisms are intact, development, even if delayed, will reach rule-based or principle-based thought, which is important for social functioning. This explains why the IQ of individuals with deficiencies in central processes is low, and the IQ of individuals with deficiencies in a specific domain is generally normal (Brandenburg et al., 2021; Deb et al., 2022; McDonough et al., 2017).
3. Deviation from normality increases with increasing deficiency in central processes or the number of deficient specific processes. Quality and rate of development would be affected because the poverty of domains would impede or distort the development of central processes (see Figure 1).

4. The importance of attaining developmental priorities varies as a function of the developmental cycle. Attaining the priorities of representational thought is more critical than other cycles. Failing to meet the significant priorities of this cycle, representational awareness, and attention control, if concurrently present with deficiencies in a specific domain, would result in a developmental disorder related to this domain and may continue to exert adverse effects much later.
5. Based on the model presented here, a complete diagnostic system would map the precise profile of children at risk about all domain-specific and central dimensions of the architecture of mind. Also, such a model which focuses on understanding systems-level brain development at the level of the individual child can specify the likelihood of a disorder, explain comorbidity across neurodevelopmental disorders and help understand heterogeneity within neighboring conditions. Evidence-Based Intervention, in turn, can lead to experimentally implement new multimodal remedial methodologies for treating several domains of impairment.

Acknowledgements. Special thanks are due to Dimitris Anastasiou for his comments on an earlier version of this article.

Funding statement. No funding was obtained for this paper.

Competing interests. There are no competing interests.

References

Ball, G., & Karmiloff-Smith, A. (2014). Why development matters in neurodevelopmental disorders. In J. van Herwegen, & D. Riby (Eds.), *Neurodevelopmental disorders: Research, challenges and solutions. Research methods in developmental psychology-A handbook series* (pp. 19–33). Psychology Press.

Ball, G., Malpas, C. B., Genc, S., Efron, D., Sciberras, E., Anderson, V., Nicholson, J. M., & Silk, T. J. (2019). Multimodal structural neuroimaging markers of brain development and ADHD symptoms. *American Journal of Psychiatry*, *176*, 57–66.

Barkley, R. A. (1997). *ADHD and the nature of self-control*. Guilford Press.

Baron-Cohen, S. (2000). Theory of mind and autism: A review. *International Review of Research in Mental Retardation*, *23*, 169–184.

Baron-Cohen, S. (2020). *The pattern seekers: A new theory of human invention*. Penguin.

Behrmann, M., Avidan, G., Leonard, G. L., Kimchi, R., Luna, B., Humphreys, K., & Minshew, N. (2006). Configural processing in autism and its relationship to face processing. *Neuropsychologia*, *44*(1), 110–129.

Bishop, D. V., & Norbury, C. F. (2002). Exploring the borderlands of autistic disorder and specific language impairment: A study using standardised diagnostic instruments. *Journal of Child Psychology and Psychiatry*, *43*(7), 917–929.

Brandenburg, J., Huschka, S. S., Visser, L., & Hasselhorn, M. (2021). Are different types of learning disorder associated with distinct cognitive functioning profiles? *Frontiers in Psychology*, *12*, 725374.

Bridgett, D. J., & Walker, M. E. (2006). Intellectual functioning in adults with ADHD: A meta-analytic examination of full-scale IQ differences between adults with and without ADHD. *Psychological Assessment*, *18*(1), 1–14.

Butterworth, B. (2005). Developmental dyscalculia. In J. I. D. Campbell (Eds.), *The handbook of mathematical cognition* (pp. 455–467). Psychology Press.

Butterworth, B. (2010). Foundational numerical capacities and the origins of dyscalculia. *Trends in Cognitive Sciences*, *14*(12), 534–541.

Buzsaki, G. (2019). *The brain from inside out*. Oxford University Press.

Cárdenas, S. Y., Silva-Pereyra, J., Prieto-Corona, B., Castro-Chavira, S. A., & Fernández, T. (2021). Arithmetic processing in children with dyscalculia: An event-related potential study. *PeerJ*, *9*, e10489.

Carr, J. (2005). Stability and change in cognitive ability over the life span: A comparison of populations with and without Down's syndrome. *Journal of Intellectual Disability Research*, *49*(12), 915–928.

Carroll, J. B. (1993). *Human cognitive abilities: A survey of factor-analytic studies*. Cambridge University Press.

Carroll, J. M., & Snowling, M. J. (2004). Language and phonological skills in children at high risk of reading difficulties. *Journal of Child Psychology and Psychiatry*, *45*(3), 631–640.

Chan, M. M. Y., & Han, Y. M. Y. (2020). Differential mirror neuron system (MNS) activation during action observation with and without social-emotional components in autism: A meta-analysis of neuroimaging studies. *Molecular Autism*, *11*(1), 1–18.

Charman, T., Jones, C. R. G., Pickles, A., Simonoff, E., Baird, G., & Happé, F. (2011). Defining the cognitive phenotype of autism. *Brain Research*, *1380*, 10–21.

Charman, T., Pickles, A., Simonoff, E., Chandler, S., Loucas, T., & Baird, G. (2011). IQ in children with autism spectrum disorders: Data from the special needs and autism project (SNAP). *Psychological Medicine*, *41*(3), 619–627.

Cheng, W., Rolls, E. T., Gu, H., Zhang, J., & Feng, J. (2015). Autism: Reduced connectivity between cortical areas involved in face expression, theory of mind, and the sense of self. *Brain*, *138*(5), 1382–1393.

Christoforou, C., Ktisti, C., Richardson, U., & Papadopoulos, T. C. (2023). Microgenetic analysis of reading remediation: A novel computational framework. *Advances in Cognitive Psychology*, *19*, 297–315.

Christoforou, C., Papadopoulos, T. C., & Theodorou, M. (2022). Toward the study of the neural-underpinnings of dyslexia during final-phoneme elision: A machine learning approach. *Brain Informatics, LNAI*, *13406*, 74–85.

Christoforou, C., Theodorou, M., Fella, A., & Papadopoulos, T. C. (2023). Phonological ability and neural congruency: Phonological loop or more? *Clinical Neurophysiology*, *156*, 228–241.

Chu, F. W., VanMarle, K., & Geary, D. C. (2016). Predicting children's reading and mathematics achievement from early quantitative knowledge and domain-general cognitive abilities. *Frontiers in Psychology*, *7*, 775.

Cicchetti, D., & Beedghly, M. (1990).*Children with Down syndrome: A developmental perspective*.Cambridge University Press.

Cicchetti, D., & Cohen, D. J. (1995). Perspectives on developmental psychopathology. In D. Cicchetti, & D. J. Cohen (Eds.), *Developmental psychopathology: Theory and method*. vol. *1*, (pp. 3–20). Wiley.

Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology*, *6*(4), 533–549.

Dahiya, A. V., McDonnell, C., DeLucia, E., & Scarpa, A. (2020). A systematic review of remote telehealth assessments for early signs of autism spectrum disorder: Video and mobile applications. *Practice Innovations*, *5*(2), 150–164.

Deb, S. S., Roy, M., Bachmann, C., & Bertelli, M. O. (2022). Specific learning disorders, motor disorders, and communication disorders. In M. O. Bertelli, S. Deb, K. Munir, A. Hassiotis, & L. Salvador-Carulla (Eds.), *Textbook of psychiatry for intellectual disability and autism spectrum disorder*. Springer.

Dębska, A., Banfi, C., Chyl, K., Dzięgiel-Fivet, G., Kacprzak, A., Łuniewska, M., Plewko, J., Grabowska, A., Landerl, K., & Jednoróg, K. (2021). Neural patterns of word processing differ in children with dyslexia and isolated spelling deficit. *Brain Structure and Function*, *226*(5), 1467–1478.

Dehaene, S. (2011). *The number sense: How the mind creates mathematics*. Oxford University Press.

Dehaene, S. (2014). *Consciousness and the brain: Deciphering how the brain codes our thoughts*. Penguin Books.

Dehaene, S., Nakamura, K., Jobert, A., Kuroki, C., Ogawa, S., & Cohen, L. (2010). Why do children make mirror errors in reading? Neural correlates of mirror invariance in the visual word form area. *Neuroimage*, *49*(2), 1837–1848.

Demetriou, A. (2000). Organization and development of self-understanding and self-regulation: Toward a general theory. In M. Boekaerts, P. R. Pintrich, & M. Zeidner (Eds.), *Handbook of self-regulation* (pp. 209–251). Academic Press.

Demetriou, A., Christou, C., Spanoudis, G., & Platsidou, M. (2002). The development of mental processing: Efficiency, working memory, and

thinking. *Monographs of the Society for Research in Child Development*, *67*(1), vii–154.
Demetriou, A., Efklides, A., & Platsidou, M. (1993). The architecture and dynamics of developing mind: Experiential structuralism as a frame for unifying cognitive developmental theories. *Monographs of the Society for Research in Child Development*, *58*(5/6), v–167.
Demetriou, A., Golino, H., Spanoudis, G., Maris, N., & Greiff, S. (2021). The future of intelligence: The central meaning-making unit of intelligence in the mind, the brain, and artificial intelligence. *Intelligence*, *87*, 101562.
Demetriou, A., Makris, N., Kazi, S., Spanoudis, G., & Shayer, M. (2018a). The developmental trinity of mind: Cognizance, executive control, and reasoning. *WIREs Cognitive Science*, *9*(4), e1461.
Demetriou, A., Makris, N., Kazi, S., Spanoudis, G., Shayer, M., & Kazali, E. (2018b). Mapping the dimensions of general intelligence: An integrated differential-developmental theory. *Human Development*, *61*(1), 4–42.
Demetriou, A., & Spanoudis, G. (2018). *Growing minds: A general theory of intelligence and learning*. Routledge.
Demetriou, A., Spanoudis, G., Christou, C., Greiff, S., Makris, N., Vainikainen, M.-P., Golino, H., & Gonida, E. (2023). Cognitive and personality predictors of school performance from preschool to secondary school: An overarching model. *Psychological Review*, *130*(2), 480–512.
Demetriou, A., Spanoudis, G., Greiff, S., Panaoura, R., Vaijnikainen, M. P., Kazi, S., & Makris, N. (2024). *Educating the developing mind: A developmental theory of instruction*. Routledge.
Demetriou, A., Spanoudis, G., Kazi, S., Mouyi, A., Žebec, M. S., Kazali, E., Golino, H., Bakracevic, K., & Shayer, M. (2017). Developmental differentiation and bindingof mental processes with re-morphing g through the lifespan. *Journal of Intelligence*, *5*(2), 23.
Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*(1), 135–168.
Donnai, D., & Karmiloff-Smith, A. (2000). Williams syndrome: From genotype through to the cognitive phenotype. *American Journal of Medical Genetics*, *97*, 164–171.
Efklides, A. (2008). Metacognition: Defining its facets and levels of functioning in relation to self-regulation and co-regulation. *European Psychologist*, *13*(4), 277–287.
Ferguson, K. A., & Cardin, J. A. (2020). Mechanisms underlying gain modulation in the cortex. *Nature Reviews Neuroscience*, *21*(2), 80–92.
Fombonne, E. (2003). Epidemiological surveys of autism and other pervasive developmental disorders: an update. *Journal of Autism and Developmental Disorders*, *33*, 365–382.
Franceschini, S., Gori, S., Ruffino, M., Pedrolli, K., & Facoetti, A. (2012). A causal link between visual spatial attention and reading acquisition. *Current Biology*, *22*(9), 814–819.
Georgiou, G. K., Parrila, R. K., & Papadopoulos, T. C. (2008). Predictors of word decoding and reading fluency in English and Greek: A cross-linguistic comparison. *Journal of Educational Psychology*, *100*(3), 566–580.
Gersten, R., Jordan, N. C., & Flojo, J. R. (2005). Early identification and interventions for students with mathematics difficulties. *Journal of Learning Disabilities*, *38*(4), 293–304.
Green, A. E., Kenworthy, L., Mosner, M. G., Gallagher, N. M., Fearon, E. W., Balhana, C. D., & Yerys, B. E. (2014). Abstract analogical reasoning in high-functioning children with autism spectrum disorders. *Autism Research*, *7*(6), 677–686.
Haier, R. J. (2017). *The neuroscience of intelligence*. Cambridge University Press.
Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences*, *3*(6), 216–222.
Happé, F., & Frith, U. (2006). The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *36*(1), 5–25.
Harter, S. (2012). *The construction of the self: Developmental and sociocultural foundations*. The Guilford Press.
Hudry, K., Chetcuti, L., Boutrus, M., Pillar, S., Baker, E. K., Dimov, S., Barbaro, J., Green, J., Whitehouse, A. J. O., Varcin, K. J., & AICES Team (2021). Performance of the autism observation scale for infants with community-ascertained infants showing early signs of autism. *Autism*, *25*(2), 490–501.
Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews Neuroscience*, *7*(12), 942–951.
Iseman, J. S., & Naglieri, J. A. (2011). A cognitive strategy instruction to improve math calculation for children with ADHD and LD: A randomized controlled study. *Journal of Learning Disabilities*, *44*(2), 184–195.
Jensen, A. R. (1998). *The g factor: The science of mental ability*. Praeger.
Johnson-Laird, P. N., & Khemlani, S. S. (2013). Toward a unified theory of reasoning. In B. H. Ross (Ed.), *The psychology of learning and motivation* (pp. 1–42). Academic Press.
Karmiloff-Smith, A. (1997). Crucial differences between developmental cognitive neuroscience and adult neuropsychology. *Developmental Neuropsychology*, *13*(4), 513–524.
Karmiloff-Smith, A. (1998). Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences*, *2*(10), 389–398.
Karmiloff-Smith, A. (2015). An alternative to domain-general or domain-specific frameworks for theorizing about human evolution and ontogenesis. *AIMS Neuroscience*, *19*(2), 91–104.
Karmiloff-Smith, A., Brown, J. H., Grice, S., & Paterson, S. (2018). Dethroning the myth: Cognitive dissociations and innate modularity in Williams syndrome. *Williams Syndrome*, 227–242.
Kasari, C., Brady, N., Lord, C., & Tager-Flusberg, H. (2013). Assessing the minimally verbal school-aged child with autism spectrum disorder. *Autism Research*, *6*(6), 479–493.
Keogh, R., Pearson, J., & Zeman, A. (2021). Aphantasia: The science of visual imagery extremes. In J. J. S. Barton, & A. Leff (Eds.), *Handbook of clinical neurology* (pp. 277–296). Elsevier.
Koponen, T., Salmi, P., Eklund, K., & Aro, T. (2013). Counting and RAN: Predictors of arithmetic calculation and reading fluency. *Journal of Educational Psychology*, *105*(1), 162–175.
Korbel, J. O., Tirosh-Wagner, T., Urban, A. E., Chen, X.-N., Kasowski, M., Dai, L., Grubert, F., Erdman, C., Gao, M. C., Lange, K., Sobel, E. M., Barlow, G. M., Aylsworth, A. S., Carpenter, N. J., Clark, R. D., Cohen, M. Y., Doran, E., Falik-Zaccai, T., Lewin, S. O., & Korenberg, J. R. (2009). The genetic architecture of Down syndrome phenotypes revealed by high-resolution analysis of human segmental trisomies. *Proceedings of the National Academy of Sciences*, *106*(29), 12031–12036.
Kosslyn, S. M., Thompson, W. L., & Ganis, G. (2006). *The case for mental imagery*. Oxford University Press.
Kucian, K. (2016). Developmental dyscalculia and the brain. In D. B. Berch, D. C. Geary, & K. Mann Koepke (Eds.), *Development of mathematical cognition: Neural substrates and genetic influences* (pp. 165–193). Elsevier Academic Press. https://doi.org/10.1016/B978-0-12-801871-2.00007-1
Kucian, K., Grond, U., Rotzer, S., Henzi, B., Schönmann, C., Plangger, F., Gälli, M., Martin, E., & von Aster, M. (2011). Mental number line training in children with developmental dyscalculia. *NeuroImage*, *57*(3), 782–795.
Landerl, K., Fussenegger, B., Moll, K., & Willburger, E. (2009). Dyslexia and dyscalculia: Two learning disorders with different cognitive profiles. *Journal of Experimental Child Psychology*, *103*(3), 309–324.
Leonard, L. B. (2014). Children with specific language impairment and their contribution to the study of language development. *Journal of Child Language*, *41*(S1), 38–47.
Lewis, G. (1996). DSM-IV. Diagnostic and statistical manual of mental disorders, 4th edn. By the American psychiatric association. (Pp. 886; £ 34.95.) APA: Washington, DC. 1994. *Psychological Medicine*, *26*(3), 651–652.
Lewton, M., Ashwin, C., & Brosnan, M. (2019). Syllogistic reasoning reveals reduced bias in people with higher autistic-like traits from the general population. *Autism*, *23*, 1311–1321.
Lyytinen, H., Erskine, J., Kujala, J., Ojanen, E., & Richardson, U. (2009). In search of a science-based application: A learning tool for reading acquisition. *Scandinavian Journal of Psychology*, *50*(6), 668–675.
Marinis, T. (2015). Providing some more pieces to the puzzle: L2 adults, L2 children and children with specific language impairment. *Linguistic Approaches to Bilingualism*, *5*, 499–504.
Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491–495.

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

McDonough, E. M., Flanagan, D. P., Sy, M., & Alfonso, V. C. (2017). Specific learning disorder. In S. Goldstein, & M. DeVries (Eds.), *Handbook of DSM-5 disorders in children and adolescents*. Springer.

McKenzie, R., Evans, J. S. B., & Handley, S. J. (2010). Conditional reasoning in autism: Activation and integration of knowledge and belief. *Developmental Psychology*, *46*(2), 391–403.

Mervis, C. B., & Velleman, S. L. (2011). Children with Williams syndrome: Language, cognitive, and behavioral characteristics, and their implications for intervention. *Perspectives on Language Learning and Education*, *18*(3), 98–107.

Milton, F., Fulford, J., Dance, C., Gaddum, J., Heuerman-Williamson, B., Jones, K., & Zeman, A. (2021). Behavioral and neural signatures of visual imagery vividness extremes: Aphantasia versus hyperphantasia. *Cerebral Cortex Communications*, *2*(2), tgab035. https://doi.org/10.1093/texcom/tgab035

Moll, K., & Landerl, K. (2009). Double dissociation between reading and spelling deficits. *Scientific Studies of Reading*, *13*(5), 359–382.

Montgomery, J. W. (2004). Sentence comprehension in children with specific language impairment: Effects of input rate and phonological working memory. *International Journal of Language & Communication Disorders*, *39*, 115–133.

Moshman, D. (2011). *Adolescent rationality and development: Cognition, morality, and identity (3rd ed.)*. Psychology Press.

Moyle, M. J., Ellis Weismer, S., Evans, J. L., & Lindstrom, M. J. (2007). Longitudinal relationships between lexical and grammatical development in typical and late-talking children. *Journal of Speech, Language, and Hearing Research*, *50*(2), 508–528.

Munson, B., Kurtz, B. A., & Windsor, J. (2005). The influence of vocabulary size, phonotactic probability, and wordlikeness on nonword repetitions of children with and without specific language impairment. *Journal of Speech, Language, and Hearing Research*, *48*(5), 1033–1047.

Mussolin, C., Mejias, S., & Noël, M. P. (2010). Symbolic and nonsymbolic number comparison in children with and without dyscalculia. *Cognition*, *115*(1), 10–25.

Nair, A., Jolliffe, M., Lograsso, Y. S. S., & Bearden, C. E. (2020). A review of default mode network connectivity and its association with social cognition in adolescents with autism spectrum disorder and early-onset psychosis. *Frontiers in Psychiatry*, *11*, 614.

Nanay, B. (2018). The importance of amodal completion in everyday perception. *i-Perception*, *9*(4), 2041669518788887.

Nigg, J. T. (2001). Is ADHD a disinhibitorydisorder? *Psychological Bulletin*, *127*, 571.

Norbury, C. F., & Bishop, D. V. (2003). Narrative skills of children with communication impairments. *International Journal of Language & Communication Disorders*, *38*, 287–313.

O'Brien, B. A., Wolf, M., & Lovett, M. W. (2012). A taxometric investigation of developmental dyslexia subtypes. *Dyslexia*, *18*, 16–39.

Papadopoulos, T. C., Csépe, V., Aro, M., Caravolas, M., Diakidoy, I. A., & Olive, T. (2021). Methodological issues in literacy research across languages: Evidence from alphabetic orthographies. *Reading Research Quarterly*, *56*(S1), S351–S370.

Papadopoulos, T. C., Georgiou, G. K., & Kendeou, P. (2009). Investigating the double-deficit hypothesis in Greek: Findings from a longitudinal study. *Journal of Learning Disabilities*, *42*(6), 528–547.

Papadopoulos, T. C., Panayiotou, G., Spanoudis, G., & Natsopoulos, D. (2005). Evidence of poor planning skills in children with attention deficits. *Journal of Abnormal Child Psychology*, *33*(5), 611–623.

Papadopoulos, T. C., Spanoudis, G. C., & Georgiou, G. K. (2016). How is RAN related to reading fluency? A comprehensive examination of the prominent theoretical accounts. *Frontiers in Psychology*, *7*, 1217.

Park, J., & Brannon, E. M. (2013). Training the approximate number system improves math proficiency. *Psychological Science*, *24*(10), 2013–2019.

Patterson, D. (2009). Molecular genetic analysis of down syndrome. *Human Genetics*, *126*(1), 195–214.

Pearson, J., Naselaris, T., Holmes, E. A., & Kosslyn, S. M. (2015). Mental imagery: Functional mechanisms and clinical applications. *Trends in Cognitive Sciences*, *19*(10), 590–602.

Peng, P., & Fuchs, D. (2016). A meta-analysis of working memory deficits in children with learning difficulties: Is there a difference between verbal domain and numerical domain? *Journal of Learning Disabilities*, *49*(1), 3–20.

Peng, P., Lin, X., Unal, Z. E., Lee, K., Namkung, J., Chow, J., & Sales, A. (2020). Examining the mutual relations between language and mathematics: A meta-analysis. *Psychological Bulletin*, *146*(7), 595–634.

Peng, P., Wang, C., & Namkung, J. (2018). Understanding the cognition related to mathematics difficulties: A meta-analysis on the cognitive deficit profiles and the bottleneck theory. *Review of Educational Research*, *88*, 434–476.

Pennington, B. F., McGrath, L. M., & Peterson, R. L. (2019). *Diagnosing learning disorders: From science to practice* (3rd ed.). Guilford Publications.

Perkins, T., Stokes, M., McGillivray, J., & Bittar, R. (2010). Mirror neuron dysfunction in autism spectrum disorders. *Journal of Clinical Neuroscience*, *17*(10), 1239–1243.

Price, G. R., Holloway, I., Räsänen, P., Vesterinen, M., & Ansari, D. (2007). Impaired parietal magnitude processing in developmental dyscalculia. *Current Biology*, *17*(24), R1042–R1043.

Reeve, R. A., & Gray, S. (2014). Number difficulties in young children. In S. Chinn (Eds.), *The Routledge international handbook of dyscalculia and mathematical learning difficulties* (pp. 44–59). Routledge.

Reigosa-Crespo, V., Valdés-Sosa, M., Butterworth, B., Estévez, N., Rodríguez, M., Santos, E., Torres, P., Suárez, R., & Lage, A. (2012). Basic numerical capacities and prevalence of developmental dyscalculia: The havana survey. *Developmental Psychology*, *48*(1), 123–135.

Rice, M. L., Wexler, K., Marquis, J., & Hershberger, S. (2000). Acquisition of irregular past tense by children with specific language impairment. *Journal of Speech, Language, and Hearing Research*, *43*(5), 1126–1144.

Rizzolatti, G., Craighero, L., & Fadiga, L. (2002). The mirror system in humans. In M. I. Stamenov, & V. Gallese (Eds.), *Mirror neurons and the evolution of brain and language* (pp. 37–59). John Benjamins Publishing Company.

Rosenberg-Lee, M., Ashkenazi, S., Chen, T., Young, C. B., Geary, D. C., & Menon, V. (2015). Brain hyper-connectivity and operation-specific deficits during arithmetic problem solving in children with developmental dyscalculia. *Developmental Science*, *18*(3), 351–372.

Rubinsten, O., & Henik, A. (2006). Double dissociation of functions in developmental dyslexia and dyscalculia. *Journal of Educational Psychology*, *98*(4), 854–867.

Schumacher, J., Hoffmann, P., Schmäl, C., Schulte-Körne, G., & Nöthen, M. M. (2007). Genetics of dyslexia: The evolving landscape. *Journal of Medical Genetics*, *44*(5), 289–297.

Seth, A. (2021). Illuminating consciousness. *New Scientist*, *251*(3350), 44–48.

Shao, R., & Gentner, D. (2022). Perceptual alignment contributes to referential transparency in indirect learning. *Cognition*, *224*, 105061.

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., Clasen, L., Evans, A., Giedd, J., & Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences*, *104*(49), 19649–19654.

Sokolova, E., Oerlemans, A. M., Rommelse, N. N., Groot, P., Hartman, C. A., Glennon, J. C., Claassen, T., Heskes, T., & Buitelaar, J. K. (2017). A causal and mediation analysis of the comorbidity between attention deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD). *Journal of Autism and Developmental Disorders*, *47*(6), 1595–1604.

Spanoudis, G., & Demetriou, A. (2020). Mapping mind-brain development: Towards a comprehensive theory. *Journal of Intelligence*, *8*(2), 19.

Spanoudis, G. C., Papadopoulos, T. C., & Spyrou, S. (2019). Specific language impairment and reading disability: Categorical distinction or continuum? *Journal of Learning Disabilities*, *52*(1), 3–14.

Stanovich, K. (2011). *Rationality and the reflective mind*. Oxford University Press.

Straussner, S. L. A. (2013). The DSM-5 diagnostic criteria: What's new? *Journal of Social Work Practice in the Addictions*, *13*(4), 448–453.

Susac, A., Bubic, A., Vrbanc, A., & Planinic, M. (2014). Development of abstract mathematical reasoning: The case of algebra. *Frontiers in Human Neuroscience*, *8*, 679.

https://doi.org/10.1017/S0954579424000944 Published online by Cambridge University Press

Sutcubasi, B., Metin, B., Kurban, M. K., Metin, Z. E., Beser, B., Sonuga-Barke, E. (2020). Resting-state network dysconnectivity in ADHD: A system-neuroscience-based meta-analysis. *The World Journal of Biological Psychiatry*, *21*(9), 662–672.

Swanson, H. L. (2015). Intelligence, working memory, and learning disabilities. In T. C. Papadopoulos, R. K. Parrila, & J. R. Kirby (Eds.), *Cognition, intelligence, and achievement: A tribute to J. P. Das* (pp. 175–196). Academic Press.

Swanson, H. L., & Jerman, O. (2006). Math disabilities: A selective meta-analysis of the literature. *Review of Educational Research*, *76*(2), 249–274.

Thomas, M. S. C., & Karmiloff-Smith, A. (2002). Modeling typical and atypical cognitive development: Computational constraints on mechanisms of change. In U. Goswami (Eds.), *Blackwell handbook of childhood cognitive development* (pp. 575–599). Blackwell.

Tilanus, E. A. T., Segers, E. & Verhoeven, L. (2019). Responsiveness to Intervention after Second versus Third Grade Diagnosis of Dyslexia. *Reading and Writing Quarterly*, *25*, 1–21. https://doi.org/10.1080/10573569.2019.1667929.

Turker, S., Kuhnke, P., Jiang, Z., & Hartwigsen, G. (2023). Disrupted network interactions serve as a neural marker of dyslexia. *Communications Biology*, *6*, 1114. https://doi.org/10.1038/s42003-023-05499-2.

van Bergen, E., de Zeeuw, E., Hart, S., Boomsma, D., de Geus, E., & Kan, K. J. (2023). Comorbidity and causality among ADHD, dyslexia, and dyscalculia. https://doi.org/10.31234/osf.io/epzgy

Vellutino, F. R., Fletcher, J. M., Snowling, M. J., & Scanlon, D. M. (2004). Specific reading disability (dyslexia): What have we learned in the past four decades? *Journal of Child Psychology and Psychiatry*, *45*(1), 2–40.

Wang, K., Li, K., & Niu, X. (2021). Altered functional connectivity in a triple-network model in autism with co-occurring attention deficit hyperactivity disorder. *Frontiers in Psychiatry*, *12*, 736755.

Wang, L.-C., Tasi, H.-J., & Yang, H.-M. (2012). Cognitive inhibition in students with and without dyslexia and dyscalculia. *Research in Developmental Disabilities*, *33*(5), 1453–1461.

Weisz, J. R., & Zigler, E. (1979). Cognitive development in retarded and nonretarded persons: Piagetian tests of the similar sequence hypothesis. *Psychological Bulletin*, *86*(4), 831–851.

Wellman, H. M. (2014). *Making minds: How theory of mind develops*. Oxford University Press.

Westermann, G., & Mareschal, D. (2002). Models of atypical development must also be models of normal development. *Behavioral and Brain Sciences*, *25*(6), 771–772.

Williams, D. L., Goldstein, G., & Minshew, N. J. (2006). Neuropsychologic functioning in children with autism: Further evidence for disordered complex information-processing. *Child Neuropsychology*, *12*(4-5), 279–298.

Wolff, J. J., & Piven, J. (2021). Predicting autism in infancy. *Journal of the American Academy of Child and Adolescent Psychiatry*, *60*(8), 958–967.

Zelazo, P. D. (2015). Executive function: Reflection, iterative reprocessing, complexity, and the developing brain. *Developmental Review*, *38*, 55–68.