



EXPLORING NEURAL PATHWAYS IN LEARNING DISABILITIES: A SYSTEMATIC REVIEW BRIDGING STRUCTURE, FUNCTION, AND COGNITION

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ABSTRACT

Learning disabilities (LDs) are neurodevelopmental disorders affecting millions of children and adults worldwide. Despite average or above-average intelligence, individuals with LDs struggle with acquiring fundamental academic skills such as reading, writing, and arithmetic. This systematic review explores neural pathways implicated in learning disabilities, synthesizing structural, functional, and cognitive research findings. The review draws from studies published between 2005 and 2024, identified through databases such as PubMed, Scopus. Inclusion criteria focused on neuroimaging studies (MRI, fMRI, DTI), neuropsychological assessments, and combined methodologies targeting dyslexia, dyscalculia, and written expression disorder. Exclusion criteria eliminated studies focusing solely on environmental factors or non-neurodevelopmental causes. Findings indicate that structural abnormalities in the left temporoparietal junction, inferior frontal gyrus, and occipitotemporal regions play a critical role in dyslexia, while dyscalculia relates strongly to deficits in the intraparietal sulcus and inferior parietal lobule. Functional disconnections, particularly in fronto-parietal and fronto-temporal circuits, disrupt efficient cognitive processing across all LD types. Moreover, these structural and functional anomalies correlate directly with deficits in phonological processing, working memory, and visuospatial processing. This review bridges the gap between structural findings and cognitive dysfunction, arguing for an integrated neurocognitive model of learning disabilities. Such a model enhances diagnostic precision, intervention development, and educational accommodations. By mapping neural substrates to cognitive phenotypes, researchers and clinicians gain deeper insight into the biological roots of LDs, fostering more effective, individualized treatment approaches.

KEYWORDS

Neural Pathways, Learning Disabilities, Cognitive Functioning, Diffusion Tensor Imaging (DTI), Functional MRI (fMRI)

INTRODUCTION:

Revisiting Learning Disabilities through a Neurocognitive Lens Learning disabilities (LDs) represent a highly heterogeneous cluster of neurodevelopmental conditions that impair an individual's ability to acquire, process, and utilize basic academic skills such as reading, writing, and mathematical calculations. First recognized in the mid-20th century, LDs have traditionally been conceptualized through behavioral and educational frameworks. However, rapid advancements in cognitive neuroscience and neuroimaging have gradually repositioned these disorders as manifestations of underlying neural circuit dysfunctions, involving structural abnormalities, functional disconnections, and impaired cognitive processes. Redefining LDs: From Behavioral to Neural Paradigm In the last two decades, a paradigm shift has occurred in LD research, moving beyond superficial symptom descriptions into the realm of structural and functional brain correlates. Dyslexia, the most extensively studied LD subtype, has become a model disorder for understanding how atypical brain development disrupts language processing. Emerging work on dyscalculia and disorders of written expression further reinforces the importance of multimodal investigations that integrate anatomical imaging, functional connectivity analyses, and detailed neuropsychological profiling. The Need for a Systematic Review Despite substantial progress, existing literature remains fragmented. Structural neuroimaging studies often operate independently of functional or cognitive investigations, resulting in siloed interpretations that miss critical cross-domain insights. This systematic review addresses that gap by weaving together anatomical, functional, and cognitive findings, building an integrative understanding of the neural pathways implicated in LDs. By consolidating evidence across these domains, this review seeks to clarify the biological underpinnings of LDs and inform more effective diagnostic and intervention strategies. Key Disorders Under Review The review focuses on three primary types of LDs—dyslexia, dyscalculia, and written expression disorder. These disorders differ in their core deficits but share overlapping neural vulnerabilities, particularly in domains such as working memory, attention regulation, and visuospatial processing. By comparing and contrasting neural signatures across these disorders, the review highlights both shared and disorder-specific pathways, enabling a richer neurobiological taxonomy of learning disabilities. Advances in Neuroimaging Technologies Modern neuroimaging tools have played a transformative role in reshaping LD research. Structural MRI has delineated atypical gray matter volumes in language and visuospatial processing regions. Functional MRI (fMRI) has exposed hypoactivation in task-relevant networks during reading and calculation tasks, while diffusion tensor imaging (DTI) has mapped compromised white matter tracts essential for efficient inter-regional

communication. Together, these methods paint a comprehensive portrait of how structural, functional, and connective deficits collectively impair academic skill acquisition. Linking Neural Deficits to Cognitive Dysfunction Cognitive deficits in LDs are not random but closely tied to specific neural vulnerabilities. For instance, impaired phonological processing in dyslexia correlates directly with reduced gray matter volume in the left temporo-parietal cortex, while visuospatial difficulties in dyscalculia map onto right parietal underdevelopment. Similarly, working memory deficits, common across multiple LDs, link to reduced functional connectivity within the fronto-parietal network. These direct structure-function-cognition linkages underscore the importance of integrative research approaches. Implications for Diagnosis and Intervention An integrated neurocognitive framework for LDs offers substantial clinical benefits. Diagnosis can move beyond behavioral checklists into biologically-informed assessments that combine imaging biomarkers with neuropsychological profiles. Treatment strategies can also be tailored to target specific neural and cognitive deficits, fostering more precise, individualized interventions. Moreover, understanding the neural underpinnings of LDs enhances advocacy efforts, countering harmful stereotypes that conflate LDs with low intelligence or poor motivation. Objectives of This Systematic Review The overarching objective of this review is to synthesize current knowledge on the neural pathways implicated in learning disabilities, with a special focus on bridging structural, functional, and cognitive findings. Specifically, the review aims to: 1. Identify key structural abnormalities associated with dyslexia, dyscalculia, and written expression disorder. 2. Map functional disconnections that impair domain-specific and domain-general cognitive processes. 3. Link these structural and functional findings to cognitive deficits observed in individuals with LDs. 4. Propose an integrative neurocognitive model of learning disabilities that can guide future research, diagnosis, and intervention.

METHODOLOGY:

Research Design and Rationale This study adopts a systematic review design, aimed at synthesizing existing literature on the neural pathways implicated in learning disabilities (LDs) by bridging structural, functional, and cognitive research findings. Systematic reviews are particularly well-suited for topics like LDs, where the volume of literature is extensive, but the findings are often fragmented across different methodologies and theoretical frameworks. This review aims to consolidate high-quality evidence into a cohesive narrative that supports future integrative research and clinical applications by applying transparent and replicable inclusion and exclusion criteria.

Research Questions

The following core questions drove the review:

1. What structural brain abnormalities are consistently associated with dyslexia, dyscalculia, and written expression disorder?
2. What functional disconnections or atypical activations have been identified in individuals with these learning disabilities during cognitive tasks?
3. How do these structural and functional deficits translate into specific cognitive impairments (e.g., phonological processing, working memory, visuospatial abilities)?
4. What commonalities and disorder-specific patterns emerge when comparing across different types of LDs?
5. What theoretical and practical implications emerge when bridging neural and cognitive findings into an integrative framework for LDs?

Search Strategy and Databases A comprehensive literature search was conducted across three primary academic databases: PubMed (covering biomedical and neuroscience studies) PsycINFO (covering psychological and neuropsychological literature) Scopus (covering interdisciplinary studies, including educational neuroscience) The search was limited to peer-reviewed publications between 2005 and 2024, ensuring that only contemporary research was included to reflect advances in neuroimaging, cognitive neuroscience, and learning disability diagnosis. **Search Terms** The search strategy employed combinations of keywords and controlled vocabulary terms (MeSH in PubMed and Thesaurus terms in PsycINFO) including: “learning disabilities” OR “specific learning disorders”, “dyslexia” OR “reading disability” “dyscalculia” OR “mathematical learning disability” “disorder of written expression” OR “writing disability” Combined with: “neuroimaging” OR “structural MRI” OR “functional MRI” OR “DTI” “neural pathways” OR “brain connectivity” “cognition” OR “neuropsychological assessment” OR “working memory” OR “phonological processing”

Inclusion Criteria Studies Were Included If They Met The Following Criteria:

1. Focused on participants diagnosed with dyslexia, dyscalculia, or written expression disorder according to DSM-IV, DSM-5, or ICD-10/ICD-11 criteria.
2. Employed structural neuroimaging (e.g., MRI, DTI), functional neuroimaging (fMRI), or combined approaches.
3. Reported cognitive data from neuropsychological assessments relevant to academic skills and cognitive processes (e.g., phonological awareness, visuospatial abilities, working memory).
4. Published in peer-reviewed journals.
5. Full text available in English.

Exclusion Criteria The Following Types Of Studies Were Excluded:

1. Studies focusing exclusively on environmental or psychosocial factors (e.g., family dynamics, socioeconomic status) without neuroimaging or cognitive data.
2. Case reports with fewer than 5 participants.
3. Studies on learning difficulties arising from acquired brain injury (e.g., traumatic brain injury, stroke).
4. Non-peer-reviewed sources (e.g., conference abstracts, book chapters).
5. Studies on comorbid conditions where LD-specific data could not be isolated (e.g., global intellectual disability, autism spectrum disorder without independent LD diagnosis). **Study Selection Process** The selection process followed PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines, consisting of:

1. Initial database search yielding 2,134 records.
2. Removal of duplicates, reducing the pool to 1,762-records.
3. Title and abstract screening by two independent reviewers, excluding 1,264 irrelevant papers.
4. Full-text review of 498 articles, with final inclusion of 84 studies that met all criteria.

Data Extraction For each included study, the following data were extracted using a standardized data extraction sheet: Study citation and year of publication, Sample size and demographic details (age, gender, diagnosis confirmation), Imaging modality (structural MRI, fMRI, DTI), Cognitive assessments employed (e.g., phonological awareness tasks, numerical processing tasks), Key structural findings (e.g., reduced gray matter volume, altered white matter tracts), Key functional findings (e.g., hypoactivation during reading tasks, altered resting-state connectivity), Reported structure-function-cognition

linkages (e.g., correlations between brain volumes and test scores) **Quality Assessment** To ensure only high-quality evidence was synthesized, each study underwent quality appraisal using a modified version of the Newcastle-Ottawa Scale (NOS) for non-randomized studies.

Key Domains Included:

Clear diagnostic criteria for LDs, Adequate sample size and demographic matching, Validated neuroimaging protocols, Use of standardized cognitive measures, Transparent reporting of structure-function correlations Control for key confounds (age, gender, handedness, IQ) Studies scoring below 5/9 on the adapted NOS were excluded, leaving 64 high-quality studies for synthesis. **Data Synthesis and Integration** Data synthesis followed a narrative synthesis approach, given the heterogeneity of imaging modalities, cognitive tests, and analytical techniques used across studies. However, where possible, meta-analytical pooling was applied for structural volumetric findings (e.g., left temporo-parietal gray matter reduction in dyslexia), providing quantitative effect sizes. The integration framework was triangulated, organizing findings into Structural abnormalities: Gray matter volume, cortical thickness, white matter tract integrity. Functional abnormalities: Task-based hypoactivation, resting-state connectivity patterns. Cognitive impairments: Phonological processing, working memory, visuospatial processing. Wherever possible, direct structure-function-cognition linkages were emphasized — identifying studies that explicitly correlated brain measures with cognitive test performance. **Addressing Heterogeneity** Recognizing the substantial heterogeneity inherent in LD research, particularly in terms of diagnostic criteria, age ranges, and imaging parameters, the review adopted several strategies to enhance comparability: Stratified synthesis by disorder subtype (dyslexia, dyscalculia, written expression disorder). Sub-analyses based on age group (children vs. adolescents vs. adults). Comparison of imaging findings across monolingual vs. bilingual populations. Highlighting methodological trends over time (e.g., increasing use of multimodal imaging since 2015). **Ethical Considerations** As a secondary research project synthesizing published data, this systematic review did not involve direct participant contact or new data collection. Ethical approval was therefore not required. However, care was taken to only include studies that had undergone ethical review, ensuring participant protection in primary research.

Limitations Of Methodology:

Several limitations were acknowledged in advance:

1. Potential publication bias favoring positive findings.
2. Heterogeneity in diagnostic criteria across studies.
3. Overrepresentation of English-speaking samples, limiting generalizability to multilingual populations.
4. Limited longitudinal data tracking developmental changes in neural pathways.
5. Exclusion of non-English publications, possibly omitting valuable global perspectives.

RESULTS AND DISCUSSION:

Overview of Included Studies The systematic review synthesized data from 64 studies spanning 2005 to 2024, covering approximately 5,840 participants across diverse age groups (children, adolescents, and adults) and diagnostic subtypes (dyslexia: 34 studies; dyscalculia: 20 studies; written expression disorder: 10 studies). The studies employed structural MRI (n=42), functional MRI (n=29), diffusion tensor imaging (n=21), and neuropsychological assessments (n=57), with several studies employing multimodal approaches. **Structural Abnormalities in Learning Disabilities** A consistent pattern of reduced gray matter volume was observed across all three LD subtypes, albeit with regional specificity: **Disorder Key Structural Findings** **Example Studies** **Dyslexia** Reduced gray matter in left temporo-parietal junction, inferior frontal gyrus, occipito-temporal cortex Hoeft et al., 2007; Richlan et al., 2013 **Dyscalculia** Reduced volume in intraparietal sulcus, inferior parietal lobule, angular gyrus Kucian et al., 2011; Ashkenazi et al., 2013 **Written Expression Disorder** Abnormal cortical thickness in left precentral gyrus, superior parietal lobule Richards et al., 2015 Across studies, these structural deficits correlated strongly with domain-specific impairments, such as phonological decoding (dyslexia), numerical magnitude representation (dyscalculia), and grapho-motor integration (written expression disorder). Importantly, co-occurring reductions in frontal and parietal gray matter were noted across all LD types, indicating shared vulnerabilities in higher-order executive systems essential for learning. **White Matter Connectivity and Disrupted Neural Networks** Diffusion tensor imaging (DTI)

studies provided additional insights into white matter integrity, highlighting disrupted connectivity in critical learning-relevant tracts: Disorder Key Tracts Affected Implicated Processes Dyslexia Arcuate fasciculus, inferior longitudinal fasciculus Phonological processing, orthographic mapping Dyscalculia Superior longitudinal fasciculus, corpus callosum Numerical magnitude processing, visuospatial reasoning Written Expression Disorder Superior fronto-occipital fasciculus, corticospinal tract Motor planning, grapho-motor coordination These findings underscore that learning disabilities are not confined to isolated cortical deficits, but rather reflect disruptions in distributed networks, where inefficient information transfer between distant brain regions impairs real-time cognitive processing. Functional Hypoactivation and Task-Based Deficits Functional MRI studies further illuminated atypical neural activation patterns during academic tasks: Disorder Task Functional Finding Example Study Dyslexia Phonological decoding Hypoactivation in left inferior frontal gyrus and temporoparietal cortex Pugh et al., 2010 Dyscalculia Numerical comparison Reduced parietal activation, especially in intraparietal sulcus Kucian et al., 2013 Written Expression Disorder Writing fluency Frontal hypoactivation during grapho-motor tasks Berninger et al., 2009 These functional deficits align closely with structural vulnerabilities, reinforcing the concept that anatomical abnormalities manifest as functional inefficiency when these regions are engaged during learning tasks. For example, in dyslexia, reduced temporoparietal volume correlates with weaker phonological processing networks, resulting in slowed and inaccurate decoding during reading. Cognitive Impairments: Bridging Structure and Function The review found robust evidence linking neuropsychological deficits to underlying neural anomalies across all LD types. Specific cognitive profiles emerged: Disorder Core Cognitive Deficits Neural Correlates Dyslexia Phonological awareness, rapid naming, working memory Left temporoparietal and frontal networks Dyscalculia Numerical magnitude processing, spatial reasoning, working memory Intraparietal sulcus, parietal-frontal circuits Written Expression Disorder Orthographic coding, fine motor coordination, working memory Left precentral gyrus, parietal-premotor linkages A striking commonality across all LD subtypes was working memory impairment, consistently linked to frontal-parietal disconnection. This underscores that while each LD has domain-specific cognitive vulnerabilities, they share underlying executive function weaknesses, particularly in working memory — the cognitive workspace essential for holding and manipulating information during academic tasks. Multimodal Convergence: An Integrative Neurocognitive Model Perhaps the most compelling outcome of this review is the convergence across structural, functional, and cognitive domains, supporting an integrative neurocognitive model of LDs. This model conceptualizes LDs as resulting from: Focal structural vulnerabilities in task-specific regions (language areas in dyslexia, numerical regions in dyscalculia). Distributed network inefficiencies affecting domain-general processes like working memory and attention regulation. Cascading cognitive consequences that impair acquisition of foundational academic skills. This model moves beyond traditional “modular deficit” theories that view LDs as isolated language, math, or motor problems. Instead, LDs emerge from multi-level neurodevelopmental disruptions spanning microstructural anomalies, large-scale connectivity inefficiencies, and impaired cognitive functioning. Comparative Insights: Shared and Unique Pathways A comparative synthesis across LD subtypes highlights: Aspect Dyslexia Dyscalculia Written Expression Disorder Primary Deficit Phonological processing Numerical cognition Orthographic-motor integration Primary Neural Regions Temporo-parietal cortex Intraparietal sulcus Precentral gyrus Common Deficits Working memory, processing speed Working memory, spatial attention Working memory, motor planning Shared Vulnerabilities Frontal-parietal disconnection Frontal-parietal disconnection Frontal-parietal disconnection These patterns confirm that while each LD type involves unique academic challenges and focal neural deficits, they also share underlying executive and attentional inefficiencies, reflecting common neural vulnerabilities across learning disorders. Implications for Research and Practice This synthesis has far-reaching implications for research, diagnosis, and intervention. It highlights the need for: Multimodal diagnostic assessments combining cognitive testing with neuroimaging. Personalized interventions targeting both domain-specific deficits (e.g., phonics training in dyslexia) and domain-general functions (e.g., working memory training across all LDs). Early identification programs that screen for common executive vulnerabilities, even before academic difficulties manifest. It also calls for longitudinal research to track how these neural and cognitive

deficits evolve over development — clarifying whether early interventions can alter developmental trajectories and mitigate long-term academic and psychosocial consequences.

Table 1: Summary of Key Studies Examining Neural Pathways in Learning Disabilities

Author & Year	Sample Population	Methodology	Key Findings
Hoef et al., 2011	60 children (30 with dyslexia, 30 controls)	fMRI, DTI	Dyslexia associated with reduced white matter integrity in left temporo-parietal pathways.
Vandermosten et al., 2012	40 children (dyslexia & controls)	DTI Tractography	Deficits in arcuate fasciculus linked to phonological processing impairment.
Finn et al., 2014	50 adolescents (varied learning profiles)	Resting-state fMRI	Disrupted connectivity between prefrontal cortex and temporoparietal junction in reading disabilities.
Lebel & Beaulieu, 2009	100 children (ages 5-18)	DTI Longitudinal Study	Increased white matter coherence associated with improved reading over time.

Table 2: Neural Pathways And Cognitive Processes In Specific Learning Disabilities

Neural Pathway	Cognitive Function	Learning Disability Linked	Evidence from Literature
Arcuate Fasciculus	Phonological Processing	Dyslexia	Hoef et al., 2011; Vandermosten et al., 2012
Inferior Longitudinal Fasciculus	Visual Processing	Dyslexia & Dysgraphia	Chang et al., 2015
Superior Longitudinal Fasciculus	Working Memory & Reading	Dyslexia & Dyscalculia	Vestergaard et al., 2011
Corpus Callosum	Interhemispheric Transfer	Dyslexia & ADHD (co-occurring)	Hasan et al., 2012

Table 3: Imaging Techniques Used In Research On Neural Pathways And Learning Disabilities

Imaging Modality	Strengths	Limitations	Common Application in LD Research
DTI (Diffusion Tensor Imaging)	Tracks white matter integrity	Sensitive to motion artifacts	Mapping arcuate fasciculus in dyslexia
fMRI (Functional MRI)	Measures functional connectivity	Temporal resolution lower than EEG	Examining reading network activation
MEG (Magnetoencephalography)	High temporal resolution	Costly, limited availability	Phonological processing in real time
sMRI (Structural MRI)	Provides anatomical detail	Limited functional data	Structural anomalies in dyslexia, dysgraphia

Table 4: Statistical Analysis of Neural Pathway Abnormalities in Learning Disabilities

Study	Sample Size	Neural Pathway	Test Used	Effect Size (Cohen's d)	p-value	Key Findings
Hoef et al., 2011	60 (30 dyslexic, 30 controls)	Arcuate Fasciculus	Independent t-test	d = 1.02	p < 0.001	Significant reduction in white matter coherence in dyslexic group.
Vandermosten et al., 2012	40 (20 dyslexic, 20 controls)	Left Superior Longitudinal Fasciculus	ANOVA	$\eta^2 = 0.18$	p < 0.01	Dyslexic participants showed lower fractional anisotropy (FA) values.
Finn et al., 2014	50 adolescents	Temporo-parietal Connectivity	Pearson Correlation	r = 0.65	p < 0.001	Strong correlation between reduced connectivity and poor reading performance.
Lebel & Beaulieu, 2009	100 children (5-18 years)	Corpus Callosum	Linear Regression	$\beta = 0.42$	p = 0.002	Increased callosal integrity predicted better language skills.

CONCLUSION:

The present systematic review consolidates two decades of research on the neural underpinnings of learning disabilities (LDs), offering a comprehensive, integrative understanding of how structural anomalies, functional inefficiencies, and cognitive deficits converge to shape academic difficulties. By synthesizing data across dyslexia, dyscalculia, and written expression disorder, this review highlights both disorder-specific vulnerabilities and shared neural and cognitive dysfunctions, challenging simplistic, domain-isolated conceptualizations of LDs. Key Findings in Structure, Function, and Cognition A primary conclusion is that learning disabilities are rooted in neurodevelopmental alterations that span multiple levels of brain organization. Structural imaging studies consistently demonstrate reduced gray matter volume and altered cortical thickness in regions crucial for academic skill acquisition. In dyslexia, this manifests as abnormalities in the left temporo-parietal cortex, compromising phonological processing. In dyscalculia, reduced volume in the intraparietal sulcus impairs numerical magnitude representation. Written expression disorder shows atypical development in precentral and parietal cortices, undermining grapho-motor fluency. White matter findings further reveal that these localized deficits are embedded within disrupted large-scale networks, where inefficient connectivity between frontal, parietal, and temporal regions impairs information flow critical for real-time cognitive processing. This aligns with functional imaging studies, which consistently show hypoactivation in task-relevant networks across all LD types during phonological, numerical, or writing tasks. The cognitive consequences of these neural disruptions are both domain-specific and domain-general. Each LD presents a unique primary impairment — phonological decoding in dyslexia, numerical magnitude processing in dyscalculia, and grapho-motor planning in written expression disorder. However, all LDs share significant impairments in working memory, processing speed, and executive function, indicating that core executive networks serve as common neural bottlenecks across learning domains. This shared vulnerability underscores the need to view LDs not merely as isolated academic problems but as developmental disorders of brain organization affecting the ability to coordinate, hold, and manipulate information across cognitive domains. Toward an Integrative Neurodevelopmental Model The

review supports a triangulated neurodevelopmental model, wherein focal structural anomalies trigger functional inefficiencies, which, in turn, impair the development of foundational cognitive abilities necessary for academic learning. This cascading process highlights the importance of early identification — structural and functional abnormalities may precede academic symptoms, offering opportunities for preemptive intervention. Moreover, the overlap in executive and working memory deficits across LDs challenges current categorical diagnostic systems, which often artificially separate dyslexia, dyscalculia, and writing disorders into distinct entities. Instead, evidence favors a dimensional approach, recognizing that many children with learning difficulties show mixed or overlapping profiles, with shared neural vulnerabilities contributing to difficulties across multiple academic domains. Implications for Future Research and Clinical Practice Future research should embrace multimodal approaches, combining neuroimaging with comprehensive neuropsychological profiling, to capture the full spectrum of neural and cognitive vulnerabilities in LDs. Longitudinal studies tracking the developmental evolution of these vulnerabilities from preschool through adolescence can clarify whether early interventions targeting executive function and working memory might prevent cascading academic difficulties. For clinical practice, the findings advocate for integrated assessment protocols, where traditional cognitive testing is complemented by neuroimaging-based risk profiling in high-risk children (e.g., those with family histories of LDs). Intervention models should similarly adopt a dual focus, combining domain-specific skill building (e.g., phonics instruction in dyslexia) with domain-general executive function training, ensuring that foundational cognitive capacities are strengthened alongside academic remediation. In sum, this review reframes LDs not just as academic disorders, but as developmental disorders of brain organization, shaped by complex interactions between structure, function, and cognition. Embracing this integrated perspective is essential for advancing more accurate diagnosis, earlier identification, and more effective, holistic interventions — ultimately transforming outcomes for children with learning disabilities.

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