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Understanding, Educating, and Supporting Children with Specific Learning Disabilities: 50 Years of Science and Practice

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Abstract

Specific learning disabilities (SLD) are highly relevant to the science and practice of psychology, both historically and currently, exemplifying the integration of interdisciplinary approaches to human conditions. They can be manifested as primary conditions—as difficulties in acquiring specific academic skills—or as secondary conditions, comorbid to other developmental disorders such as Attention Deficit Hyperactivity Disorder. In this synthesis of historical and contemporary trends in research and practice, we mark the 50th anniversary of the recognition of SLD as a disability in the US. Specifically, we address the manifestations, occurrence, identification, comorbidity, etiology, and treatment of SLD, emphasizing the integration of information from the interdisciplinary fields of psychology, education, psychiatry, genetics, and cognitive neuroscience. SLD, exemplified here by Specific Word Reading, Reading Comprehension, Mathematics, and Written Expression Disabilities, represent spectrum disorders each occurring in approximately 5–15% of the school-aged population. In addition to risk for academic deficiencies and related functional social, emotional, and behavioral difficulties, those with SLD often have poorer long-term social and vocational outcomes. Given the high rate of occurrence of SLD and their lifelong negative impact on functioning if not treated, it is important to establish and maintain effective prevention, surveillance, and treatment systems involving professionals from various disciplines trained to minimize the risk and maximize the protective factors for SLD.

Keywords

Specific Learning Disabilities; Individuals with Disabilities Education Act; Identification; Etiology; Response to Intervention

Fifty years ago, the US federal government, following an advisory committee recommendation (United States Office of Education, 1968), first recognized specific

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learning disabilities (SLD) as a potentially disabling condition that interferes with adaptation at school and in society. Over these 50 years, a significant research base has emerged on the identification and treatment of SLD, with greater understanding of the cognitive, neurobiological, and environmental causes of these disorders. The original 1968 definition of SLD remains statutory through different reauthorizations of the 1975 special education legislation that provided free and appropriate public education for all children with disabilities, now referred to as the Individuals with Disabilities Education Act (IDEA, 2004). SLD are recognized worldwide as a heterogeneous set of academic skill disorders represented in all major diagnostic nomenclatures, including the Diagnostic and Statistical Manual-5 (DSM-5, American Psychiatric Association, 2013) and the International Statistical Classification of Diseases and Related Health Problems (ICD-11, World Health Organization, 2018).

In the US, the SLD category is the largest for individuals who receive federally legislated support through special education. Children are identified as SLD through IDEA when a child does not meet state-approved age- or grade-level standards in one or more of the following areas: oral expression, listening comprehension, written expression, basic reading skills, reading fluency, reading comprehension, mathematics calculation, and mathematics problem solving. Although children with SLD historically represented about 50% of the children aged 3–21 served under IDEA, percentages have fluctuated across reauthorizations of the special education law, with some decline over the past 10 years (Figure 1).

This review is a consensus statement developed by researchers currently leading the National Institute of Child Health and Human Development (NICHD) supported Consortia of Learning Disabilities Research Centers and Innovation Hubs. This consensus is based on the primary studies we cite, as well as the meta-analytic reviews (*), systematic reviews (**), and first-authored books (***) that provide an overview of the science underlying research and practice in SLD (see references). The hope is that this succinct overview of the current state of knowledge on SLD will help guide an agenda of future research by identifying knowledge gaps, especially as the NICHD embarks on a new strategic plan. The research programs on SLD from which this review is derived represent the integration of diverse, interdisciplinary approaches to behavioral science and human conditions. We start with a brief description of the historical roots of the current view of SLD, then provide definitions as well as prevalence and incidence rates, discuss comorbidity between SLD themselves and SLD and other developmental disorders, comment on methods for SLD identification, present current knowledge on the etiology of SLD, and conclude with evidence-based principles for SLD intervention.

Three Historical Strands of Inquiry that Shaped the Current Field of SLD

Three strands of phenomenological inquiry culminated in the 1968 definition and have continued to shape current terminology and conventions in the field of SLD (Figure 2). The first, a medical strand, originated in 1676, when Johannes Schmidt described an adult who had lost his ability to read (but with preserved ability to write and spell) because of a stroke. Interest in this strand reemerged in the 1870s with the publication of a string of adult cases who had lived through a stroke or traumatic brain injury. Subsequent cases involved children

who were unable to learn to read despite success in mathematics and an absence of brain injury, which was termed “word blindness” (W. P. Morgan, 1896). These case studies laid the foundation for targeted investigations into the presentation of *specific unexpected* difficulties related to reading printed words despite typical intelligence, motivation, and opportunity to learn.

The second strand is directly related to the formalization of the American Psychiatric Association’s Diagnostic and Statistical Manual (DSM). Rooted in the work of biologically oriented physicians, the 1952 first edition (DSM-I) referenced a category of chronic brain syndromes of unknown cause that focused largely on behavioral presentations we now recognize as hyperkinesis and Attention Deficit Hyperactivity Disorder (ADHD). The 1968 DSM-II defined “mild brain damage” in children as a chronic brain syndrome manifested by hyperactive and impulsive behavior with reference to a new category, “hyperkinetic reaction of childhood” if the origin is not considered “organic.” As these categories evolved, they expanded to encompass the academic difficulties experienced by many of these children.

After almost 30 years of research into this general category of “minimal brain dysfunction,” representing “... children of near average, average, or above average general intelligence with certain learning or behavioral disabilities ... associated with deviations of function of the central nervous system.” (Clements, 1966, pp. 9–10), the field acknowledged the heterogeneity of these children and the failure of general “one size fits all” interventions. As a result, the 1980 DSM-III formally separated academic skill disorders from ADHD. The 1994 DSM-IV differentiated reading, mathematics, and written expression SLD. The DSM-5 reversed that, merging these categories into one overarching category of SLD (nosologically distinct from although comorbid with ADHD), keeping the notion of specificity by stating that SLD can manifest in three major academic domains (reading, mathematics, and writing).

The third strand originated from the development of effective interventions based on cognitive and linguistic models of observed academic difficulties. This strand, endorsed in the 1960s by Samuel Kirk and associates, viewed SLD as an overarching category of spoken and written language difficulties that manifested as disabilities in reading (dyslexia), mathematics (dyscalculia), and writing (dysgraphia). Advances have been made in understanding the psychological and cognitive texture of SLD, developing interventions aimed at overcoming or managing them, and differentiating these disorders from each other, from other developmental disorders, and from other forms of disadvantage. This work became the foundation of the 1968 advisory committee definition of SLD, which linked this definition with that of minimal brain dysfunction via the same “unexpected” exclusionary criteria (i.e., not attributable *primarily* to intellectual difficulties, sensory disorders, emotional disturbance, or economic/cultural diversity).

Although its exclusionary criteria were well specified, the definition of SLD did not provide clear inclusionary criteria. Thus, the US Department of Education’s 1977 *regulatory* definition of SLD included a cognitive discrepancy between higher IQ and lower achievement as an inclusionary criterion. This discrepancy was viewed as a marker for unexpected underachievement and penetrated the policy and practice of SLD in the US and

abroad. In many settings, the measurement of such a discrepancy is still considered key to identification. Yet, IDEA 2004 and the DSM-5 moved away from this requirement due to a lack of evidence that SLD varies with IQ and numerous philosophical and technical challenges to the notion of discrepancy (Fletcher, Lyon, Fuchs, & Barnes, 2019). IDEA 2004 also permitted an alternative inclusion criterion based on Response-to-Intervention (RTI), in which SLD reflects inadequate response to effective instruction, while the DSM-5 focuses on evidence of persistence of learning difficulties despite treatment efforts.

These three stands of inquiry into SLD use a variety of concepts (e.g., word blindness, strephosymbolia, dyslexia and alexia, dyscalculia and acalculia, dysgraphia and agraphia), which are sometimes differentiated and sometimes used synonymously, generating confusion in the literature. Given the heterogeneity of their manifestation and these diverse historical influences, it has been difficult to agree on the best way to identify SLD, although there is consensus that their core is unexpected underachievement. A source of active research and controversy is whether “unexpectedness” is best identified by applying solely exclusionary criteria (i.e., simple low achievement), inclusionary criteria based on uneven cognitive development (e.g., academic skills lower than IQ or another aptitude measure, such as listening comprehension), or evidence of persisting difficulties (DSM-5) despite effective instruction (IDEA 2004).

Manifestation, Definition, and Etiology

That the academic deficits in SLD relate to other cognitive skills has always been recognized, but the diagnostic and treatment relevance of this connection has remained unclear. A rich literature on cognitive models of SLD (Elliott & Grigorenko, 2014; Fletcher et al., 2019) provides the basis for five central ideas. First, SLD are componential (Melby-Lervåg, Lyster, & Hulme, 2012; Peng & Fuchs, 2016): Their academic manifestations arise on a landscape of peaks, valleys, and canyons in various cognitive processes, such that individuals with SLD have weaknesses in specific processes, rather than global intellectual disability (Morris et al., 1998). Second, the cognitive components associated with SLD, just like academic skills and instructional response, are dimensional and normally distributed in the general population (Ellis, 1984), such that understanding typical acquisition should provide insight into SLD and vice versa (Rayner, Foorman, Perfetti, Pesetsky, & Seidenberg, 2001). Third, each academic and cognitive component may have a distinct signature in the brain (Figure 3) and genome (Figure 4). These signatures and etiologies likely overlap because they are correlated, but are not interchangeable, as their unique features substantiate the distinctness of various SLD (Vandermosten, Hoeft, & Norton, 2016). Fourth, the overlap at least partially explains their rates of comorbidity (Berninger & Abbott, 2010; Szucs, 2016; Willcutt et al., 2013). Fifth, deficiencies in these cognitive and academic processes appear to last throughout the lifespan, especially in the absence of intervention (Klassen, Tze, & Hannok, 2013).

The DSM-5 and IDEA 2004 reflect agreement that SLD can occur in word reading and spelling (Specific Word Reading Disability; SWRD) and in specific reading comprehension disability (SRCD). SWRD represents difficulties with beginning reading skills due at least in part to phonological processing deficits, while other language indicators (e.g., vocabulary)

may be preserved (Pennington, 2009). In contrast, SRCD (Cutting et al., 2013), which is more apparent later in development, is associated with non-phonological language weaknesses (Scarborough, 2005). The magnitude of SRCD is greater than that of vocabulary or language comprehension difficulties, suggesting that other problems, such as weaknesses in executive function or background knowledge, also contribute to SRCD (Spencer, Wagner, & Petscher, 2018).

Math SLDs are differentiated as calculations (SMD) versus problem solving (word problems) SLD, which are associated with distinct cognitive deficits (L. S. Fuchs et al., 2010) and require different forms of intervention (L. S. Fuchs et al., 2014). Calculation is more linked to attention and phonological processing, while problem solving is more linked to language comprehension and reasoning; working memory has been associated with both. Specific written expression disability, SWED (Berninger, 2004; Graham, Collins, & Rigby-Wills, 2017) occurs in the mechanical act of writing (i.e., handwriting, keyboarding, spelling), associated with fine motor-perceptual skills, or in composing text (i.e., planning and revising, understanding genre), associated with oral language skills, executive functions, and the automaticity of transcription skills. Although each domain varies in its cognitive correlates, treatment, and neurobiology, there is overlap. By carefully specifying the domain of academic impairment, considerable progress has been made in the treatment and understanding of the factors that lead to SLD.

Identification methods have searched for other markers of unexpected underachievement beyond low achievement, but always include exclusionary factors. Diagnosis solely by exclusion has been criticized due to the heterogeneity of the resultant groups (Rutter, 1982); thus, the introduction of a discrepancy paradigm. One approach relies on the aptitude-achievement discrepancy, commonly operationalized as a discrepancy between measures of IQ and achievement in a specific academic domain. IQ-discrepancy was the central feature of federal regulations for identification from 1977 until 2004, although the approaches used to qualify and quantify the discrepancy varied in the 50 states. Lack of validity evidence (Stuebing et al., 2015; Stuebing et al., 2002) resulted in its de-emphasis in IDEA 2004 and elimination from DSM-5.

A second approach focuses on identifying uneven patterns of strengths and weaknesses (PSW) profiles of cognitive functioning to explain observed unevenness in achievement across academic domains (Flanagan, Alfonso, & Mascolo, 2011; Hale et al., 2008; Naglieri & Das, 1997). According to these methods, a student with SLD demonstrates a weakness in achievement (e.g., word reading), which correlates with an uneven profile of cognitive weaknesses and strengths (e.g., phonological processing deficits with advanced visual-spatial skills). Proponents suggest that understanding these patterns is informative for individualizing interventions that capitalize on student strengths (i.e., maintain and enhance academic motivation) and compensate for weaknesses (i.e., enhance the phonological processing needed for the acquisition and automatization of reading), but little supporting empirical evidence is available (Miciak, Fletcher, Stuebing, Vaughn, & Tolar, 2014; Taylor, Miciak, Fletcher, & Francis, 2017). Meta-analytic research suggests an absence of cognitive aptitude by treatment interactions (Burns et al., 2016), and limited improvement in academic

skills based on training cognitive deficits such as working memory (Melby-Lervåg, Redick, & Hulme, 2016).

Newer methods of SLD identification are linked to the development of the third historical strand, based on RTI. With RTI, schools screen for early indicators of academic and behavior problems and then progress monitor potentially at-risk children using brief, frequent probes of academic performance. When data indicate inadequate progress in response to adequate classroom instruction (Tier 1), the school delivers supplemental intervention (Tier 2), usually in the form of small-group instruction.

A child who continues to struggle requires more intensive, individualized intervention (Tier 3), which may include special education. An advantage of RTI is that intervention is provided prior to the determination of eligibility for special education placement. RTI juxtaposes the core concept of underachievement with the concept of inadequate response to instruction, that is, *intractability* to intervention. It prioritizes the presence of functional difficulty and only then considers SLD as a possible source of this difficulty (Grigorenko, 2009). Still, concerns about the RTI approach to identification remain. One concern is that RTI approaches may not identify “high-potential” children who struggle to develop appropriate academic skills (Reynolds & Shaywitz, 2009). Other concerns involve low agreement across different methods for defining inadequate RTI (D. Fuchs, Compton, Fuchs, Bryant, & Davis, 2008; L. S. Fuchs, 2003) and challenges schools face in adequately implementing RTI frameworks (Balu et al., 2015; D. Fuchs & Fuchs, 2017; Schatschneider, Wagner, Hart, & Tighe, 2016).

Prevalence and Incidence

Because the attributes of SLD are dimensional and depend on the thresholds used to subdivide normal distributions (Hulme & Snowling, 2013), estimates of prevalence and incidence vary. SWRD’s prevalence estimates range from 5 to 17% (Katusic, Colligan, Barbaresi, Schaid, & Jacobsen, 2001; Moll, Kunze, Neuhoff, Bruder, & Schulte-Körne, 2014). SRCD is less frequent (Etmanskie, Partanen, & Siegel, 2016), but still represents about 42% of all children ever identified with SLD in reading at any grade (Catts, Compton, Tomblin, & Bridges, 2012). Estimates of incidence and prevalence of SMD vary as well: from 4 to 8% (Moll et al., 2014). Cumulative incidence rates by the age of 19 years range from 5.9% to 13.8%. Similar to SWRD, SMD can be differentiated in terms of lower- and higher-order skills and by time of onset. Computation-based SMD manifests earlier; problem-solving SMD later, sometimes in the absence of computation-based SMD (L. S. Fuchs, D. Fuchs, C. L. Hamlett, et al., 2008). SWED is the least studied SLD. Its prevalence estimates range from 6% to 22% (P. L. Morgan, Farkas, Hillemeier, & Maczuga, 2016) and cumulative incidence ranges from 6.9% to 14.7% (Katusic, Colligan, Weaver, & Barbaresi, 2009).

Comorbidity and Co-Occurrence

One reason SLD can be difficult to define and identify is that different SLDs often co-occur in the same child. Comorbidity involving SWRD ranges from 30% (National Center for Learning Disabilities, 2014) to 60% (Willcutt et al., 2007). The most frequently observed co-

occurrences are between (1) SWRD and SMD (Moll et al., 2014; Willcutt et al., 2013), with 30–50% of children who experience a deficit in one academic domain demonstrating a deficit in the other (Moll et al., 2014); (2) SWRD and early language impairments (Dickinson, Golinkoff, & Hirsh-Pasek, 2010; Hulme & Snowling, 2013; Pennington, 2009) with 55% of individuals with SWRD exhibiting significant speech and language impairment (McArthur, Hogben, Edwards, Heath, & Mengler, 2000); and (3) SWRD and internalizing and externalizing behavior problems, with 25–50% of children with SWRD meeting criteria for ADHD (Pennington, 2009) and for generalized anxiety disorder and specific test anxiety, depression, and conduct problems (Cederlof, Maughan, Larsson, D'Onofrio, & Plomin, 2017), although comorbid conduct problems are largely restricted to the subset of individuals with both SWRD and ADHD (Willcutt et al., 2007).

The co-occurrence of SMD is less studied, but there are some consistently replicated observations: (1) individuals with SMD exhibit higher rates of ADHD, and math difficulties are observed in individuals with ADHD more frequently than in the general population (Willcutt et al., 2013); (2) math difficulties are associated with elevated anxiety and depression even after reading difficulties are controlled (Willcutt et al., 2013); and (3) SMD are associated with other developmental conditions such as epilepsy (Fastenau, Shen, Dunn, & Austin, 2008) and schizophrenia (Crow, Done, & Sacker, 1995).

SLD is clearly associated with difficulties in adaptation, in school and in larger spheres of life associated with work and overall adjustment. Longitudinal research reports poorer vocational outcomes, lower graduation rates, higher rates of psychiatric difficulties, and more involvement with the justice system for individuals with SWRD (Willcutt et al., 2007). Importantly, there is evidence of increased comorbidity across forms of SLD with age, with accumulated cognitive burden (Costa, Edwards, & Hooper, 2016). Individuals with comorbid SLDs have poorer emotional adjustment and school functioning than those identified with a single impairment (Martinez & Semrud-Clikeman, 2004).

Identification (Diagnosis)

Comorbidity indicates that approaches to assessment should be broad and comprehensive. For SLD, the choice of a classification model directly influences the selection of assessments for diagnostic purposes. Although all three models are used, the literature (Fletcher et al., 2019) demonstrates that a single indicator model, based either on cut-off scores, other formulae, or assessment of instructional response, does not lead to reliable identification regardless of the method employed. SLD can be identified reliably only in the context of multiple indicators. A step in this direction is a hybrid method that includes three sets of criteria, two inclusionary and one exclusionary, recommended by a consensus group of researchers (Bradley, Danielson, & Hallahan, 2002). The two inclusionary criteria are evidence of low achievement (captured by standardized tests of academic achievement) and evidence of inadequate RTI (captured by curriculum-based progress-monitoring measures or other education records). The exclusionary criterion should demonstrate that the documented low achievement is not *primarily* attributable to "other" (than SLD) putative causes such as (a) other disorders (e.g., intellectual disability, sensory or motor disorders) or (b) contextual factors (e.g., disadvantaged social, religious, economic, linguistic, or family environment).

In the future, it is likely that multi-indicator methods will be extended, with improved identification accuracy, by the addition of other indicators, neurobiological, genetic, or behavioral. It is also possible that assessment of specific cognitive processes beyond academic achievement will improve identification, but presently there is little evidence that such testing adds value to identification (Elliott & Grigorenko, 2014; Fletcher et al., 2019). All identification methods for SLD assume that children referred for assessment are in good health or are being treated and that their physical health, including hearing and vision, is monitored. Currently, there are no laboratory tests (i.e., DNA or brain structure/activity) for SLD. There are also no tests that can be administered by an optometrist, audiologist, or physical therapist to diagnose or treat SLD.

Etiological Factors

Neural structure and function—Since the earliest reports of reading difficulties, it has been assumed that the loss of function (i.e., acquired reading disability) or challenges in the acquisition of function (i.e., congenital reading disability) are associated with the brain. Functional patterns of activation in response to cognitive stimuli show reliable differences in degrees of activation between typically developing children and those identified with SWRD, and reveal different spatial distributions in relation to children identified with SMD and ADHD (Dehaene, 2009; Seidenberg, 2017). In SWRD, there are reduced gray matter volumes, reduced integrity of white matter pathways, and atypical sulcal patterns/curvatures in the left-hemispheric frontal, occipito-temporal, and temporo-parietal regions that overlap with areas of reduced brain activation during reading.

These findings together indicate the presence of atypicalities in the structures (i.e., grey matter) that form the neural system for reading and their connecting pathways (i.e., white matter). These structural atypicalities challenge the emergence of the cognitive—phonological, orthographic, and semantic—representations required for the assembly and automatization of the reading system. Although some have interpreted the atypicalities as a product of reading instruction (Krafnick, Flowers, Luetje, Napoliello, & Eden, 2014), there is also evidence that atypicalities can be observed in pre-reading children at risk for SWRD due to family history or speech and language difficulties (Raschle et al., 2015), sometimes as early as a few days after birth with electrophysiological measures (Molfese, 2000). What emerges in a beginning reader, if not properly instructed at developmentally important periods, is a suboptimal brain system that is inefficient in acquiring and practicing reading. This system is complex, representing multiple networks aligned with different reading-related processes (Figure 3). The system engages cooperative and competitive brain mechanisms at the sublexical (phonological) and lexical levels, in which the phonological, orthographic, and semantic representations are utilized to rapidly form representations of a written stimulus. Proficient readers process words on sight with immediate access to meaning (Dehaene, 2009). In addition to malleability in development, there is strong evidence of malleability through instruction in SWRD, such that the neural processes largely normalize if the intervention is successful (Barquero, Davis, & Cutting, 2014).

The functional neural networks for SMD also vary depending on the mathematical operation being performed, just as the neural correlates of SWRD and SRCD do (Cutting et al., 2013).

Neuroimaging studies on the a(typical) acquisition of numeracy posit SMD (Arsalidou, Pawliw-Levac, Sadeghi, & Pascual-Leone, 2017) as a brain disorder engaging multiple functional systems that together substantiate numeracy and its componential processes (Figure 3). First, the intraparietal sulcus, the posterior parietal cortex, and regions in the prefrontal cortex are important for representing and processing quantitative information. Second, mnemonic regions anchored in the medial temporal lobe and hippocampus are involved in the retrieval of math facts. Third, additional relevant regions include visual areas implicated in visual form judgement and symbolic processing. Fourth, prefrontal areas are involved in higher-level processes such as error monitoring, and maintaining and manipulating information. As mathematical processes become more automatic, reliance on the parietal network decreases and reliance on the frontal network increases. All these networks, assembled in a complex functional brain system, appear necessary for the acquisition and maintenance of numeracy, and various aberrations in the functional interactions between networks have been described. Thus, SMD can arise as a result of disturbances in one or multiple relevant networks, or interactions among them (Arsalidou et al., 2017; Ashkenazi, Black, Abrams, Hoeft, & Menon, 2013). There is also evidence of malleability and the normalization of neural networks with successful intervention in SMD (Iuculano et al., 2015).

Genetic and environmental factors—Early case studies of reading difficulties identified their familial nature, which has been confirmed in numerous studies utilizing genetically-sensitive designs with various combinations of relatives—identical and fraternal twins, non-twin siblings, parent-offspring pairs and trios, and nuclear and extended families. The relative risk of having SWRD if at least one family member has SWRD is higher for relatives of individuals with the condition, compared to the risk to unrelated individuals; higher for children in families where at least one relative has SWRD; even higher for families where a first-degree relative (i.e., a parent or a sibling) has SWRD; and higher still for children in families where both parents have SWRD (Snowling & Melby-Lervåg, 2016). Quantitative-genetic studies estimate that 30–80% of the variance in reading, math or spelling outcomes is explained by heritable factors (Willcutt et al., 2010).

Since the 1980s, there have been systematic efforts to identify the sources of structural variation in the genome, i.e., genetic susceptibility loci that can account for the strong heritability and familiality of SWRD (Figure 4). These efforts have yielded the identification of nine regions of the genome thought to harbor genes, or other genetic material, whose variation is associated with the presence of SWRD and individual differences in reading-related processes. Within these regions, a number of candidate genes have been tapped, but no single candidate has been unequivocally replicated as a causal gene for SWRD, and observed effects are small. In addition, multiple other genes located outside of the nine linked regions have been observed to be relevant to the manifestation of SWRD and related difficulties. Currently there are ongoing efforts to interrogate candidate genes for SWRD and connect their structural variation to individual differences in the brain system underlying the acquisition and practice of reading.

There are only a few molecular-genetic studies of SMD and its related processes (Figure 4). Unlike SWRD, no “regions of interest” have been identified. Only one study investigated the

associations between known single-nuclear polymorphisms (SNP) and a composite measure of mathematics performance derived from various assessments of SMD-related componential processes and teacher ratings. The study generated a set of SNPs that, when combined, accounted for 2.9% of the phenotypic variance (Figure 4 shows the genes in which the three most statistically significant SNPs from this set are located). Importantly, when this SNP set was used to study whether the association between the 10-SNP set and mathematical ability differs as a function of characteristics of the home and school, the association was stronger for indicators of mathematical performance in chaotic homes and in the context of negative parenting.

Finally, studies have investigated the pleiotropic (i.e., impacting multiple phenotypes) effects of SWRD candidate genes on SMD, ADHD, and related processes. These effects are seemingly in line with the “generalist genes” hypothesis, asserting the pleiotropic influences of some genes to multiple SLD (Plomin & Kovas, 2005).

Environmental factors are strong predictors of SLD. These factors penetrate all levels of a child’s ecosystem: culture, demonstrated in different literacy and numeracy rates around the world; social strata, captured by social-economic indicators across different cultures; characteristics of schooling, reflected by pedagogies and instructional practices; family literacy environments through the availability of printed materials and the importance ascribed to reading at home; and neighborhood and peer influences. Interactive effects suggest that reading difficulties are magnified when certain genetic and environmental factors co-occur, but there is evidence of neural malleability even in SWDE (Overvelde & Hulstijn, 2011). Neural and genetic factors are best understood as risk factors that variably manifest depending on the home and school environment and child attributes like motivation.

Intervention

Although the content of instruction varies depending on whether reading, math, and/or writing are impaired, *general principles of effective intervention* apply across SLD[i]. First, intervention for SLD is *explicit* (Seidenberg, 2017): Teachers formally present new knowledge and concepts with clear explanations, model skills and strategies, and teach to mastery with cumulative practice with ongoing guidance and feedback. Second, intervention is *individualized:* Instruction is formatively adjusted in response to systematic progress-monitoring data (Stecker, Fuchs, & Fuchs, 2005). Third, intervention is *comprehensive and differentiated,* addressing the multiple components underlying proficient skill as well as comorbidity. Comprehensive approaches address the multifaceted nature of SLD and provide more complex interventions that are generally more effective than isolated skills training in reading (Mathes et al., 2005) and math (L. S. Fuchs et al., 2014). For example, children with SLD and ADHD may need educational and pharmacological interventions (Tamm et al., 2017). Anxiety can develop early in children who struggle in school, and

[i]For examples of effective evidence-based interventions see www.evidenceforessa.org, intensiveintervention.org, What Works Clearinghouse, www.meadowscenter.org, www.FCRR.org/literacyroadmap, www.understood.org/en/about/our.../national-center-for-learning-disabilities, https://ies.ed.gov/ncee/edlabs/infographics/pdf/REL_SE_Implementing_evidencebased_literacy_practices_roadmap.pdf, among others.

internalizing problems must be treated (Grills, Fletcher, Vaughn, Denton, & Taylor, 2013). Differentiation through individualization in the context of a comprehensive intervention also permits adjustments of the focus of an intervention on specific weaknesses.

Fourth, intervention *adjusts intensity* as needed to ensure success, by increasing instructional time, decreasing group size, and increasing individualization (L. S. Fuchs, Fuchs, & Malone, 2017). Such specialized intervention is typically necessary for students with SLD (L. S. Fuchs et al., 2015). Yet, effective instruction for SLD begins with differentiated general education classroom instruction (Connor & Morrison, 2016), in which intervention is coordinated with rather than supplanting core instruction (L. S. Fuchs, D. Fuchs, C. Craddock, et al., 2008).

In addition, intervention is more effective when provided early in development. For example, intervention for SWRD was twice as effective if delivered in grades 1 or 2 than if started in grade 3 (Lovett et al., 2017). This is underscored by neuroimaging research (Barquero et al., 2014) showing that experience with words and numbers is needed to develop the neural systems that mediate reading and math proficiency. A child with or at risk for SWRD who cannot access print because of a phonological processing problem will not get the reading experience needed to develop the lexical system for whole word processing and immediate access to word meanings. This may be why remedial programs are less effective after second grade; with early intervention, the child at risk for SLD develops automaticity because they have gained the experience with print or numbers essential for fluency. Even with high quality intensive intervention, some children with SLD do not respond adequately, and students with persistent SLD may profit from assistive technology (e.g., computer programs that convert text-to-speech; Wood, Moxley, Tighe, & Wagner, 2018).

Finally, interventions for SLD must occur in the context of the academic skill itself. Cognitive interventions that do not involve print or numbers, such as isolated phonological awareness training or working memory training without application to mathematical operations do not improve reading or math skill (Melby-Lervåg et al., 2016). Physical exercises (e.g., cerebellar training), optometric training, special lenses or overlays, and other proposed interventions that do not involve teaching reading or math are ineffective (Pennington, 2009). Pharmacological interventions are effective largely due to their impact on comorbid symptoms, with little evidence of a direct effect on the academic skill (Tamm et al., 2017).

No evaluations of recovery rate from SLD have been performed. Intervention success has been evaluated as closing the age-grade discrepancy, placing children with SLD at an age-appropriate grade level, and maintaining their progress at a rate commensurate with typical development. Meta-analytic studies estimate effect sizes of academic interventions at 0.49 for reading (Scammacca, Roberts, Vaughn, & Stuebing, 2015), 0.53 for math (Dennis et al., 2016), and 0.74 for writing (Gillespie & Graham, 2014).

Implications for Practice and Research

Practitioners should recognize that the psychological and educational scientific evidence base supports specific approaches to the identification and treatment of SLD. In designing SLD evaluations, assessments must be timely to avoid delays in intervention; they must consider comorbidities as well as contextual factors, and data collected in the context of previous efforts to instruct the child. Practitioners should use the resulting assessment data to ensure that intervention programs are evidence-based and reflect explicitness, comprehensiveness, individualization, and intensity. There is little evidence that children with SLD benefit from discovery, exposure, or constructivist instructional approaches.

With respect to research, the most pressing issue is understanding individual differences in development and intervention from neurological, genetic, cognitive, and environmental perspectives. This research will ultimately lead to earlier and more precise identification of children with SLD, and to better interventions and long-term accommodations for the 2–6% of the general population who receive but do not respond to early prevention efforts. More generally, other human conditions may benefit from the examples of progress exemplified by the integrated, interdisciplinary approaches that underlie the progress of the past 50 years in the scientific understanding of SLD.

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Biography

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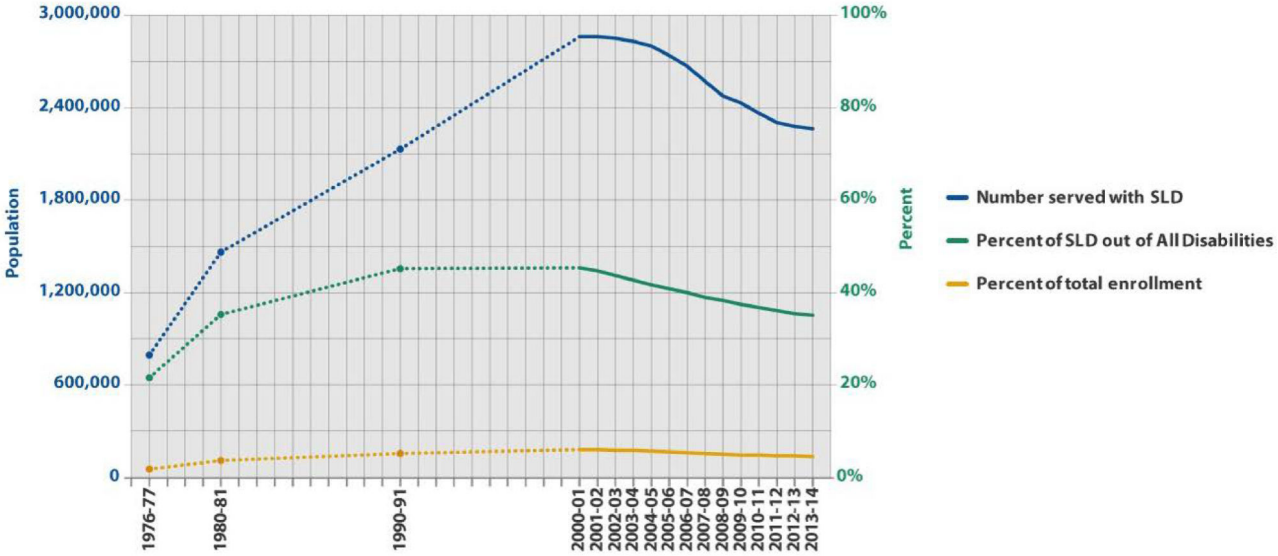

Figure 1.

The Individuals with Disabilities Education Act (IDEA), enacted in 1975 as Public Law 94–142, mandates that children and youth ages 3–21 with disabilities be provided a free and appropriate public school education in the least restricted environment. The percentage of children served by federally mandated special education programs, out of total public school enrollment, increased from 8.3 percent to 13.8 percent between 1976–77 and 2004–05. Much of this overall increase can be attributed to a rise in the percentage of students identified as having SLD from 1976–77 (1.8 percent) to 2004–05 (5.7 percent). The overall percentage of students being served in programs for those with disabilities decreased between 2004–05 (13.8 percent) and 2013–14 (12.9 percent). However, there were different patterns of change in the percentages served with some specific conditions between 2004–05 and 2013–14. The percentage of children identified with SLD declined from 5.7 percent to 4.5 percent of the total public school enrollment during this period. This number is highly variable by state: for example, in 2011 it ranged from 2.3% in Kentucky to 13.8% in Puerto Rico, as there is much variability in the procedures used to identify SLD, and disproportional demographic representation. Figure by Janet Croog.

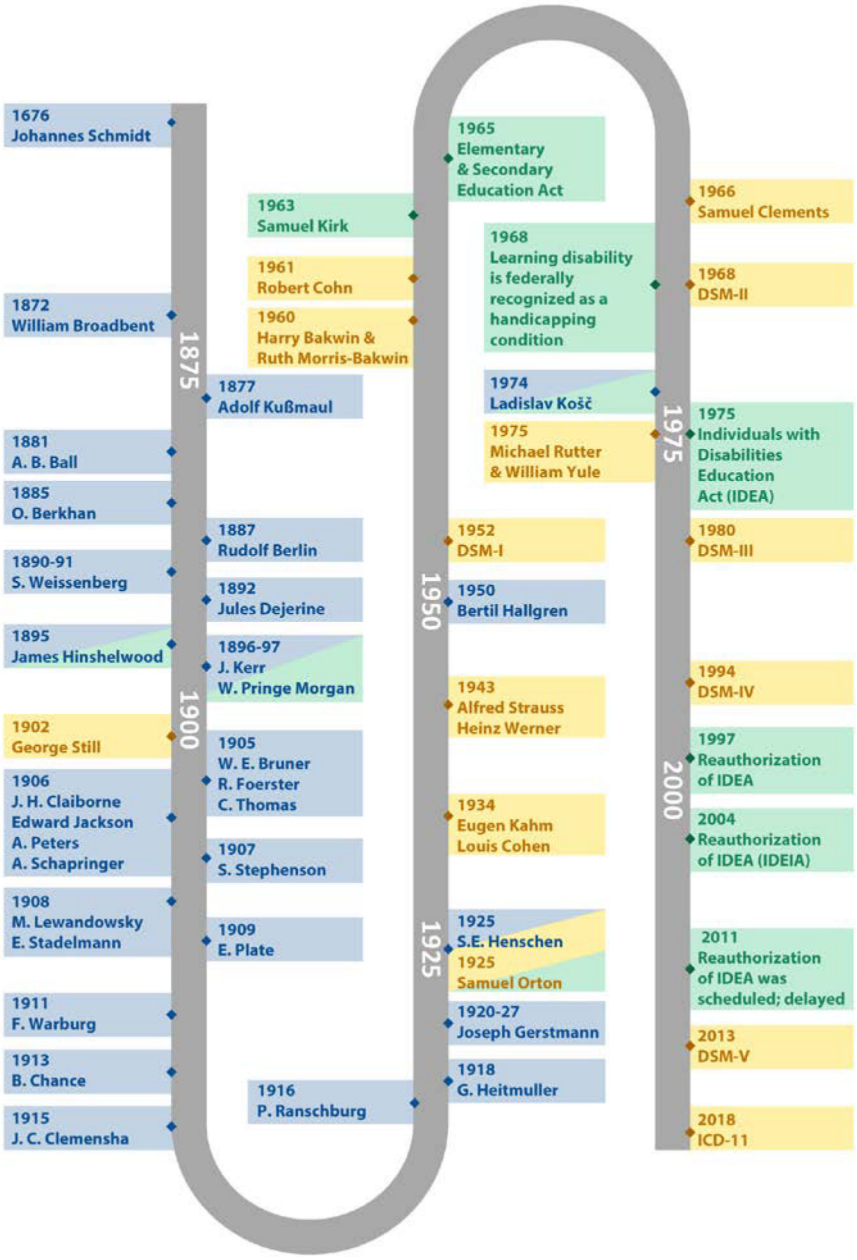

Figure 2.

A schematic timeline of the three stands of science and practice in the field of SLD. The colors represent the strands (blue—first, yellow—second, and green—third). Blue: provided phenomenological descriptions and generated hypotheses about the gene-brain bases of SLD (specifically, dyslexia or SRD); it also provided the first evidence that the most effective treatment approaches are skill-based and reflect cognitive models of the conditions. Yellow: differentiated SLD from other comorbid conditions. Green: stressed the importance of focusing on SLD in academic settings and developing both preventive and remediational evidence-based approaches to managing these conditions. Due to space constraints, the names of many highly influential scientists (e.g., Marilyn Adams, Joseph Torgesen, Isabelle

Liberman, Keith Stanovich, among others) who shaped the field of SLD have been omitted. Figure by Janet Croog.

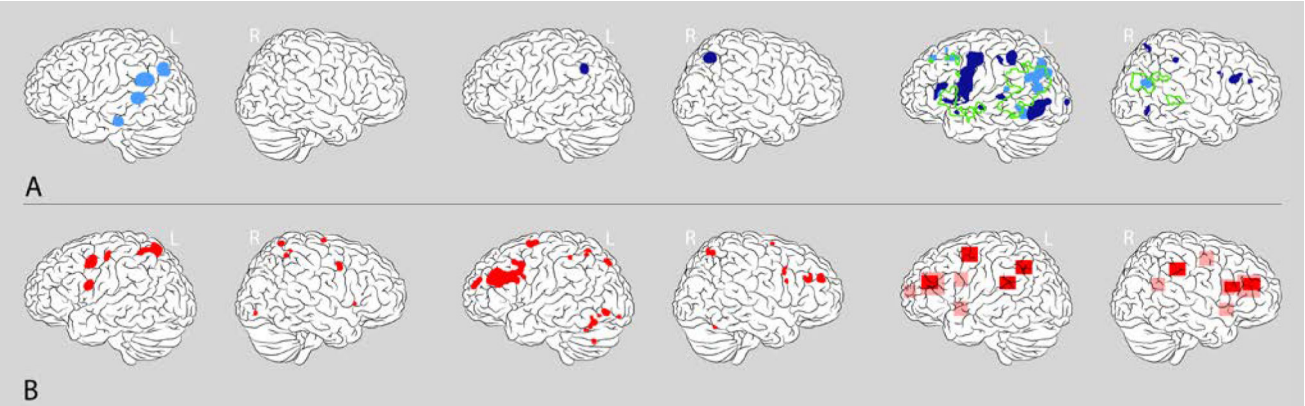

Figure 3.

Results of meta-analyses of functional neuroimaging studies that exemplify the distribution of activation patterns in different reading- (**A**) and mathematics- (**B**) related networks, corresponding to componential models of the skills. **A** (Left panel, light blue): A lexical network in the basal occipito-temporal regions and in the left inferior parietal cortex. **A** (Middle panel, dark blue): A sublexical network, primarily involving regions of the left temporo-parietal lobe extending from the left anterior fusiform region. **A** (Right panel): Activation likelihood estimation map of foci from the word>pseudowords (light blue) and pseudowords>words (dark blue) contrasts. The semantic processing cluster is shown in green. **B** (Left panel): A number-processing network, primarily involving a region of the parietal lobe. **B** (Middle panel): An arithmetic-processing network, primarily involving regions of the frontal and parietal lobes. **B** (Right panel): Children (red) and adult (pink) meta-analyses of brain areas associated with numbers and calculations. Figure by Janet Croog.

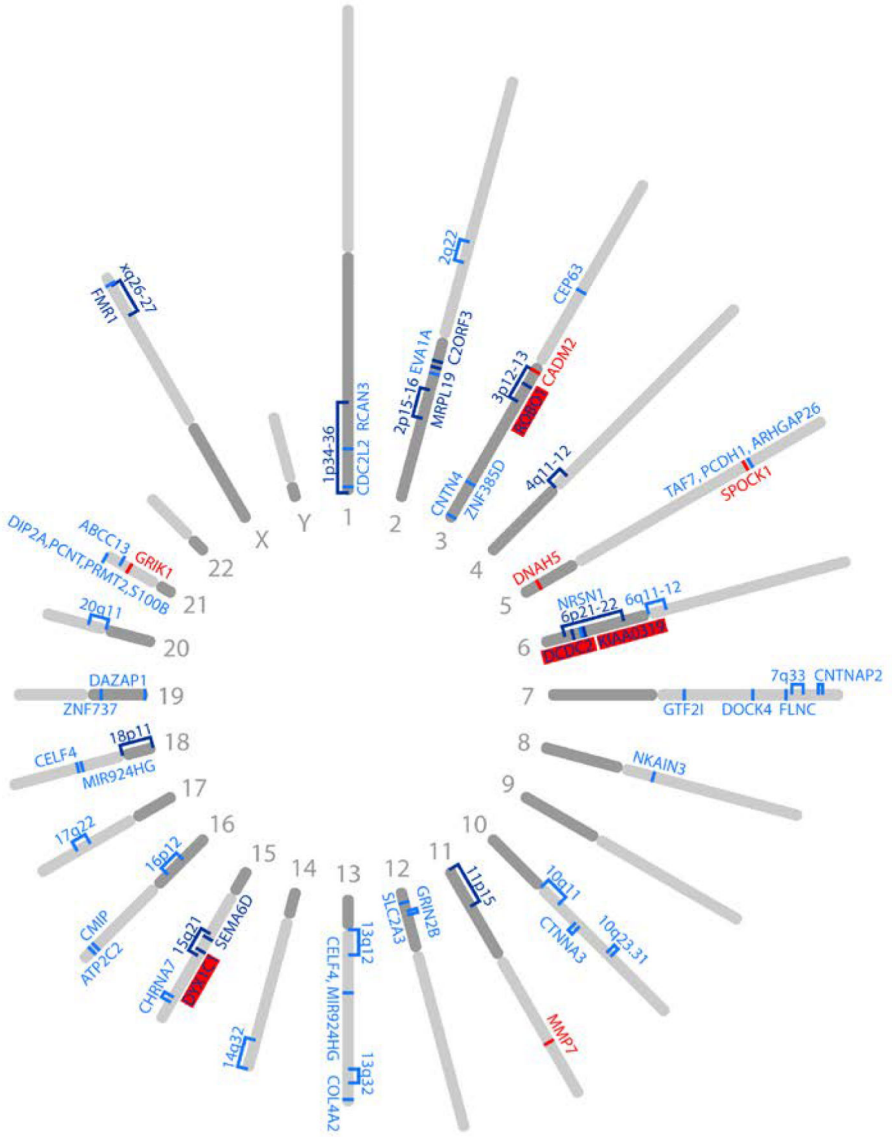

Figure 4.

A schematic representation of the genetic regions and gene-candidates linked to or associated with SRD and reading-related processes (shown in blue), and SMD and mathematics-related processes (shown in red). Dark blue signifies more studied loci and genes. Blue highlighted in red indicate the genes implicated in both SRD and SMD. Figure by Janet Croog.