An evolutionary perspective on reading and reading disorders

Mary Helen Immordino-Yang* and Terrence W. Deacon

Overview: An evolutionary perspective on reading can contribute to understanding dyslexia and other learning disorders. Human beings evolved speech over many thousands of years, but writing and reading are recent inventions, only a few thousand years old. People perform reading by a kludge of processes that evolved for other purposes, with wide variation in component processes across people and languages. Research on brain anatomy and function shows strong localization of spoken language functions, but an evolutionary approach suggests that localization will be much more variable for reading. Also, children process language across many more brain regions than do adults, suggesting that dyslexia in children may involve more brain systems as well. Processes involved in reading vary from lower-level, modality-specific processes such as vision and hearing, to mid-level linguistic processes, to higher-level processes of memory and attention. Spoken language involves a tighter integration across levels than does reading, and reading requires a greater contribution from higher-level processes because of its recent origin. One tool for investigating how these processes develop and function is analysis of brain volume in living humans by the use of modern brain-imaging tools, discussed by Vernne Cavinness in an essay for this chapter. The Editors

In a time when learning styles, individual differences, variation in development, and separate intelligences are coming to the fore in education (Fischer & Bidell, 1998; Fischer, Rose, & Rose, this volume; Gardner, 1983; D. Rose, this volume; Rose & Meyer, 2002), practitioners and researchers are looking to explain the cognitive and neuropsychological processes that underlie scholastic achievement. To this end, it is increasingly important not only to study the observable behaviors associated with scholastic skills, but to consider these skills as deriving from systems of neuropsychological capacities with evolutionary histories and constraints (Christiansen & Kirby, 2003). In particular, basic literacy competence is fundamental to scholastic success, yet a significant percentage of

children, despite sufficient general intelligence, do not attain this goal. How can thinking about reading as a task recruiting various evolutionarily derived neural systems help to shed light on this problem?

This essay attempts to outline some consequences and predictions of taking an evolutionary perspective on developmental dyslexia. Though perhaps far more theoretical and speculative than empirical or prescriptive, these remarks may help to focus attention on aspects of the problem that have been given less attention than warranted. In bringing an evolutionary perspective to bear on a localization tradition of research, we aim to bring some of the diversity of findings and explanations into a more unified view, as well as to suggest future areas of research.

Evolution and the history of reading

Reading is a complex skill, and unlike spoken language, it is a skill for which there is no evolutionary background. The earliest conventional written symbols date from about 3500 BC, long after the evolution of anatomically modern Homo sapiens, who appeared about 200,000 years ago, and of oral language, which probably gradually evolved over as much as 2.5 million years. The evolutionary “maturity” of our faculty for spoken language is reflected in our numerous human specializations for vocal-oral and syntactic processing, by the integration of diverse language adaptations (e.g., vocal tract changes, discourse predispositions), and by the marvelously predictable and robust nature of language acquisition (Deacon, 1997, 2003, 2004; Hauser, Chomsky, & Fitch, 2002).

In contrast, phonologically based reading is totally immature in an evolutionary sense. Though various logographic (i.e., partially or wholly iconic) systems of representation have been discovered independently by different peoples around the world, the only known discovery/invention of the alphabetic principle was in Greece, around 1000 BC (for a concise overview see Crystal, 1997; see also Wolf & Ashby, this volume). Thus, the alphabetic principle is a recently available tool and not a built-in organic function of the brain. As a result, reading in an alphabetic system is not an intuitive task, nor is it an automatic extension of spoken language. It does not develop spontaneously, and without explicit instruction it will not develop at all. Most importantly for this discussion, unlike oral language competence, literacy competence is exceedingly variable from individual to individual and full literacy may remain unachievable for a significant fraction of the otherwise normal population (worldwide estimates hover just below 10 percent), despite educational support.

It is not clear why written forms of communication only appeared at the very most recent end of human evolution, when it is likely that our oral language capacity is so old. Perhaps, as today, the recording of specific speech acts

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was never of sufficient reproductive consequence for most hunter-gatherer or primitive agricultural peoples. Whatever the reason, the non-organic status of written communication underscores the fact that searching for biological bases for reading competence or for dyslexia is unlike looking for the cause of organic disorders, such as congenital blindness or diabetes, in a number of important respects. For one, we know of no genes that code for reading or writing alone, and we should not expect to find any (see Galaburda & Sherman, this volume).

The neural systems recruited for these skills and the genes that produce these systems almost certainly have homologues, or corresponding genes, in most primates and probably in most mammals (Deacon, 2004). If we do happen to find genes that are correlated with developmental dyslexia or agaphnia (writing disorder), the chances are good that those genes will not even be unique to humans, much less unique to reading and writing. Secondly and more importantly, because reading is not a unitary biological function, shaped and organized by natural selection into a unified system, it is also not likely that there will be any unitary underlying biological cause for dyslexic syndromes. Instead, we should anticipate that although some congenial neural defects may interfere more seriously than others with the acquisition of this skill, there will be an array of potential contributors to reading disability that have cumulative or interactive effects of varying severity on susceptibility to dyslexia. This suggests that many possible inherited disturbances could interrupt literacy acquisition because such a post hoc collaboration of brain systems is likely to be more vulnerable to a wider range of disturbances: genetic, developmental, and even experimental.

So from an evolutionary perspective, one implication of this post-evolutionary status of literacy is that any reductionist effort to approach dyslexia as a simple genetic disorder will likely oversimplify the problem. In a biological context, developmental dyslexia is not a biological abnormality; instead, it is a class of special conditions within the normal variation—an unhappy coincidence of traits, only some of which would otherwise be deleterious. Taken together, these traits may affect the development of reading preferentially simply because of the biological fragility of this skill. Like the canary in the mine, reading may be more sensitive to minor insults than other cognitive and sensorimotor skills.

Another implication of the evolutionary perspective is that there will be no necessarily “natural” course of literacy development common to all children. In other words, even if it is the case that there is a modal age in which children can begin to acquire certain reading skills, this is not because of a developmental bioprogram with respect to reading, but rather because reading must piggyback on other capacities developing for other reasons according to their own schedules. Indeed, given the biologically ad hoc nature of this behavioral capacity, diverse developmental trajectories might be expected for the development of reading (Fischer & Immordino-Yang, 2002).

This leads to a further caution: dyslexia expressed in adults, or acquired due to insult in adulthood, may not provide a firm basis for retrospective deduction about the organization of reading skills in children’s brains. Lacking developmental “canalization,” reading skill may develop along diverse trajectories and still converge upon similar endpoints in adult brain organization (Fischer & Bidell, 1998; Fischer, Rose, & Rose, this volume). Children’s brains are less differentiated, less efficient at certain tasks, and in transition toward mature anatomical development. Neural mechanisms critical for a well-learned reading competence in adults may not be available in immature brains, and the process of initially acquiring this skill may itself transiently depend on systems not critical for the mature capacity. In addition, there may be shifts in the dependence of this skill on specific brain systems during development. Though localization data from adults with acquired and developmental dyslexia offer essential clues to brain areas important for reading in adults, these data are likely to underrepresent the essential systems developmentally. Alternatively, we suggest that in order to apply such data retrospectively to children, we need first to determine if there is any change of distribution of functions from young novice to adult expert reader, as well as to elucidate the nature of this proposed developmental reorganization.

The localization perspective

Modern neuropsychology has succeeded in giving us confidence that brain function is comprehensible in large part because it has provided extensive evidence that the brain can be analyzed into parts that contribute discrete sub-functions for perceptual, motor, and mnemonic processes of the whole. The assumption that the substrates for functions such as color discrimination or verbal short-term memory are “located” in a center or circuit in the brain, and that these centers or circuits are similarly located across most individuals’ brains, has been the basis for extraordinary advancements in the neurosciences and neurology. But does it make sense to search for the reading center or centers in the brain? This enterprise has been on the whole quite successful, even if—unlike color discrimination and other functions—reading cannot be treated as an evolved function for which the brain’s architecture was designed. Obviously, every function that is dependent on the working brain must be performed by a structure located in the brain, and the brain is not homogeneous nor are functions so deeply distributed that they do not have local correlates (Lieberman, 2002). Every activity of a brain has some focus in locations. The question is whether reading uses many structures diversely or just a few focal regions.

Evidence of brain problems or disconnection

In the late nineteenth century, aphasiologists suggested that reading abilities might be localized in the brain with respect to the connections between the component functional centers involved, e.g. vision and audition. They reasoned
that acquired reading problems might be the result of an interruption in the neurological pathways connecting occipital and left superior temporal cortical areas. In a classic case, Dejerine (1891) described a patient with what he termed "pure word blindness without agraphia." This patient presented with an inability to read despite apparently intact speech. On autopsy, the patient's brain demonstrated posterior corpus callosal and left parietal damage. Dejerine explained the patient's symptoms in terms of disconnection between visual areas and language areas, arguing that neither eye could send visual information to the left hemisphere Wernicke's area for language. This approach did not posit a reading center per se, but did predict that communications between centers for more basic functions would be essential for reading to be possible.

Modern neuroanatomical research efforts to understand dyslexia along similar lines, such as the work of Geschwind (Geschwind, 1982, 1983, 1984) and Galaburda (Galaburda & Livingstone, 1993; Galaburda & Sherman, this volume), built on the work of these nineteenth-century aphasiologists. They suggested that dyslexia in children, like acquired alexia in adults from brain damage, might also be the result of disconnection between, or discrete damage to, parts of the brain devoted to the component visual and auditory analysis centers of the cerebral cortex. In one of the pioneering studies of anatomical correlates to developmental dyslexia, Galaburda and Kemper (1979) found that at autopsy the brains of people with lifetime dyslexia had apparently congenital abnormalities of the cerebral cortex that were located in critical language-specialized areas as well as intermodal areas. This finding implied that inherited difficulties with reading might be traceable to congenital variants of organization of cortical cells in regions that correlate with adult acquired aphasia and alexia.

In a similar vein, following initial work by Galaburda and colleagues (see Galaburda, Rosen, & Sherman, 1990; Galaburda, 1995), a number of studies found associations between dyslexia and extensive asymmetries in the superior temporal cortex, suggesting that abnormal lateralization might also be a risk factor for dyslexia (e.g., Kuschel et al., 1993; Rumsey et al., 1997). What exactly such atypical asymmetries of surface areas and volumes of these brain areas on the two sides of dyslexic brains would contribute is less obvious than for the case of cytoarchitectural anomalies, and studies failing to replicate these results have left these correlations uncertain (see discussion by Schultz et al., 1994). More recently, studies of size of cortical components (morphometry) have also included analyses of global cortical organization and development. Case studies have widened the range of structures implicated in dyslexia, extending to differences in frontal versus posterior cortical growth, and particularly differences involving the growth of the insula (Benes & Paré-Bingeov, this volume; Pennington et al., 1999).

An evolutionary perspective on reading

One common feature shared by these localization findings is that most have been focused on cortical areas specialized for language. Yet dyslexia often occurs in the absence of expression or comprehension problems involving speech. Reading difficulties secondary to language difficulties are not considered primary reading deficits, and may be more easily correlated with anatomical abnormality and damage. In fact, left hemisphere damage in the early stages of reading acquisition (i.e., middle childhood) does tend to lead to difficulties in learning to read, but recent research suggests that such reading difficulties may be a consequence of secondary linguistic or cognitive deficits rather than of an isolated reading problem per se (Pitchford, 2000).

Differences between children and adults

Research makes it increasingly clear that the cortical areas we associate with adult language functions are not necessarily the areas responsible for oral language processing in children. Rather than being localized from the beginning, early oral language seems to be much more globally distributed in the brains of children than in adults (see review by Bates, 1992). Focal brain injury in infants in the early stages of language learning leads to patterns of disruption of language learning that are distinct from brain damage associated with language processing problems in adults. In particular, both left and right hemisphere damage can lead to significant language delays in infants, with no obvious effect of lesion size (Marchman, Miller, & Bates, 1991). What is more, in infants language comprehension deficits are associated with right hemisphere injury, not with injury to Wernicke's area in the left hemisphere (Wulfeck et al., 1991), while the severity of expressive deficits is linked to left posterior injury rather than injury to Broca's area in the left temporal lobe (Thal et al., 1991).

Children also tend to show more plasticity than adults in response to brain damage (Aram, 1999; Huttenlocher, 2002; Saiz et al., 1990). In extreme cases of early brain damage, either hemisphere can develop language function, with the extent of language being determined by the extent of compensation by the non-damaged hemisphere (Mills et al., 1994; Vicari et al., 2000). In one extreme case, an adolescent boy who underwent a complete left functional hemispherectomy at age eleven temporarily lost but later recovered close to normal language function (Immordino-Yang, 2004). In addition, children with early focal lesions often do not show long-term impairment of reading or oral language (Aram, Gillespie, & Yamashita, 1990), suggesting that the compensatory brain reorganization to support oral language resulted in a system that could later support reading as well. In all, such evidence suggests that early reading skill is not localized or fixed in children to nearly the extent that it appears to be in adults.

This evidence gives cause to question the direct applicability of adult localization findings to the study of reading disorders in children.
Localization logic also suggests that, aside from issues around primary language deficits and localization, disturbances of basic auditory and visual centers and their interconnections should be relevant. It goes without saying that blindness or inability to analyze word sounds would directly block reading, as would inability to associate visual information with phonological information. Therefore, secondary limitations in using these modalities can be expected to contribute severe impediments as well.

Although this relation clearly holds with adult brain damage, there is a question about how it applies to young brains and to a skill that seems to involve the contributions of many more systems in children than in adults. Analysis requires paying attention to developmental patterns of reorganization and specialization, such as the dynamic redistribution of function that may occur as part of the learning process in children. One possibility is greater plasticity in children. Another is that these basic systems are relatively impervious to neural work-arounds or individual developmental compensations via neural plasticity, because they involved specialized modalities and are relatively mature by the time children begin to encounter written material in school. Thus, modality-specific processing weaknesses may either constitute a limit to the level of automatization possible or else provide only a degraded signal for higher-order processors. These linked questions of developmental change and neural commitment are important to understanding the neurobiology of reading difficulties and may provide windows into intervention approaches not obvious from localization logic (Deacon, 2000). As mentioned above, we cannot ignore developmental patterns of reorganization and specialization, or the dynamic redistribution of function that likely occurs as part of the learning process. Assuming that the component functions involved in reading are equivalently organized in adults' and children's brains may lead us to miss important factors.

In addition, the reading task itself may be very different during learning and afterwards. Because beginning readers are struggling to pick out and process the relevant cues involved in reading, the task for them likely recruits a widely distributed set of diverse functional systems. They must rely heavily on attentional and mnemonic strategies to sort out ambiguities and manage alternative ways of processing what they read. As they mature and gain reading skill and experience, the burden can shift from effortful higher-order and global processes to automated processes carried out in more specialized areas. This presents yet another reason to expect global functioning to be more critical in early reading acquisition, with specialized processing becoming more prominent later.

In fact, such a suggestion parallels evidence from adult skill learning. For instance, there is some evidence from electrical stimulation work in adult neurosurgery patients that brain functions in language and reading become localized as literacy proficiency develops. Specifically, patients with low verbal IQ and relatively poor reading skills tend to show stimulation-induced reading disruption in a wider range of brain locations than do high verbal IQ patients (Ojemann, 1979, 1991). Such findings imply that with the development of reading fluency, the functions involved in reading might become progressively more localized, only reaching mature patterns late in the development of reading skill.

Also relevant is evidence from adults that the brain regions used to initially acquire new skills are often not the most active when the same skill becomes thoroughly mastered (for example, with a word-association task studied by Rakel et al., 1994). That is, tasks that are difficult because they are novel at first produce stronger, more global activation of cerebral cortex, and later produce weaker, more localized, and more subcortically prominent activations as the tasks become more familiar and less effortful.

But while evidence for a global to local processing shift is fairly well understood in skill automation learning in adults, this process is not yet well documented in children. It seems clear that for reading to become efficient, the component tasks involved must be sufficiently automated. Future research in this area could provide insight into the role of automation of processing in normal and dyslexic reading acquisition. For example, there is evidence that the cerebellum may be involved in the cross-neuron summing that enables automatization and hence localization with multiple encounters with a particular task, and dyslexic children have been demonstrated to show cerebellar weaknesses (Fawcett et al., 1996; Fawcett & Nicolson, 1999). However, the role of the cerebellum in automation of reading skill has not been well documented as yet.

In dyslexics, cerebellar dysfunction appears mainly to have been studied in the context of motor learning and sequencing problems.

Another source of disruption in the automation process could stem from abnormalities of the magnocellular layer subdivision of the visual system, as described by Galaburda and Livingstone (1993). Galaburda suggests that this primary neurological deficit is a main anatomical locus of difficulties in dyslexia, which is also consistent with a related correlate that has been systematically investigated by Tallal and colleagues (Tallal et al., 1998; also Wolf & Ashby, this volume). Such processing deficits could impede the ability to automate low-level visual processing, forcing the reader to maintain effortful visual attention in order to actively compensate for the processing weakness. Automation of low-level perceptual processing is important for fluent reading, so that high-level attentional processing can be devoted to semantic and strategic functions (Wolf & Katzir-Cohen, 2001).

In sum, while the localization argument has provided a fruitful initial approach to analyzing brain correlates of dyslexia, it can be misleading to use data from adults with acquired or developmental dyslexia to simply reverse engineer predictions about children's brains. Adults with acquired neurological conditions affecting reading have completed their primary learning of language.
and reading, yet it may be in large part the learning process that primarily organizes the brain into the functional modules found in adults. If the representation of function changes significantly during development, as we suggest it does, then applying adult data to children requires first understanding the change of distribution of function. Analyzing the problem of reading acquisition through a lens focusing on the evolutionary histories of the brain systems may be a useful way of interpreting the meaning and application of localization findings for children.

**Integrating evolutionary perspectives**

Because people have no evolutionary specializations that are reading specific, learning to read means recruiting and organizing diverse brain systems to function in specialized capacities uncharacteristic of their evolutionary design (Deacon, 1997). In this way, reading is a “kludge,” a clumsy work-around system consisting of components designed for other tasks. The systems we use to process and produce written language could be considered the neuropsychological equivalents to Rube Goldberg machines in that they combine various components designed for other tasks into one fragile, complex system. The combinatorial nature of this system means greater variance in the ways people process written than oral language as well as a tendency toward idiosyncrasies. This produces a continuum of reading abilities that reflects the variation in individuals’ cognitive profiles. Add to this existing variation the possibility of inherited disturbances in any component of the system, and the potential for reading problems grows exponentially. Certain combinations of genes may produce reading difficulties because, in a combinatorial system, even small, localized disturbances can result in interaction effects that impair the functioning of the system as a whole.

Due to its combinatorial nature, reading is a complex and changing activity in which the workload must be dynamically distributed between systems of different levels. This complexity means that certain reading difficulties can derive from high-level processes, such as a specific linguistic processing problem or high-level attentional or mnemonic dysfunction. Other reading difficulties could arise from sensory processing problems, including attentional problems in scanning, problems with short-term memory for visual information, or basic perceptual problems registering the shapes or relative positions of letters. However, the term “dyslexia” does not distinguish between these different levels of disability. To account for these diverse sources of disability and to describe their respective impact on the functioning of the reading system, we suggest thinking of reading competence and dyslexia in terms of a nested model that takes into account evolutionary and systemic considerations in addition to level-specific processing problems (Figure 2.1).

Figure 2.1 A nested model of dyslexia. In this model, low-level, modality-specific processing is nested within linguistic processing, which is in turn nested within high-level, attentional, and mnemonic processing. Processing problems originating in the center circles will result in the burden being passed up to the next intact level. High-level processing problems, on the other hand, will cause the entire system to appear disorganized and dysfunctional.

In a nested model of reading, the process of learning to read requires the person to successfully automate lower-order skills and integrate or “nest” them into higher-order ones as subroutines. Modality-specific processing, such as visual or auditory, is considered the lowest level and under normal circumstances the most automated. Alternatively, the most global processing systems, those associated with high-level mnemonic and attentional processes, are considered the highest level and least automated. These systems function to organize and direct the lower-level systems, as well as to evaluate and assign meaning and context to the text. Language-specific factors, such as phonological and syntactic analysis, fall at an intermediate level, which includes processing that is usually automated but can be brought to conscious attention in certain circumstances, such as learning new words or parsing the speech of a dysfluent foreigner.

This model leads us to ask, how do other components compensate when there is a neuropsychological disturbance somewhere in the kludge system that decodes and recodes written text? For the kludge to function with a weak link, the workload must be redistributed among the other nested constituents of the system, depleting their processing resources. We see this redistribution...
as occurring according to two organizing principles that follow from a developmental and evolutionary logic. First, in general, the more automated the processing the fewer attentional resources it should require (arrow pointing inward in Figure 2.1), and the more conscious the processing the more attentional resources it should require (arrow pointing outward). Because lower-level, modality-specific processes would tend to be the most automated, while higher-level processes would tend to be the most conscious and effortful, the more modality-specific the cause of reading difficulty, the easier it may be to naturally develop (or train) a workaround that allows at least some level of reading competence. On the other hand, the more global the underlying problem (e.g., working memory, sequence analysis, automatization, etc.), the more difficult it may be to surmount the initial stages of learning to read, thus blocking access to many possible alternative strategies.

Second, the model is hierarchical in that lower-level disturbances will result in the burden of processing being passed up to the next level, where the task becomes more effortful. Since there is a limit on higher-level processing resources, this shift from largely automatic to more conscious processing will bog down the higher-level systems by depleting attentional resources. The more significant the dysfunction in automated processing, the more high-level resources will be devoted to compensating for lower-level analysis. This characteristic hampers or, in rare cases, prevents higher-level processing, such as interpreting the meaning or context of the written passage. Conversely, high-level deficits will result in failure to regulate the relations between lower-order automated functions.

In this way, children with lower-level reading problems, such as problems of visual processing, may place more work on phonological analysis by having to read aloud. That is, these children will be forced to reconstruct through linguistic means what the visual system did not fully process. Likewise, problems with automated linguistic processing will cause the task to be passed up to the attentional system. As processes that should be automated become increasingly effortful and conscious, fewer attentional resources will be left for meaningful interpretation or expression of ideas, and the phonological and linguistic aspects of reading will become an effortful chore that is increasingly devoid of ideological content. Analogous problems would be apparent as well in such children’s writing, as the effort involved in retrieving orthographic representations of phonemes could overwhelm the executive functions to the point that the child could no longer hold in mind the content they wished to express. In addition, should there be disturbances at the attentional mnemonic level, the system will not be well organized, leading to repercussions ranging from problems with comprehending and expressing meaning to problems recruiting and directing even intact lower-level, automated processes. Thus, just as the components of the reading process are hierarchically nested, early successes and failures involving these components are nested in that later reading experiences build on earlier ones.

The above scenarios underscore the crucial role of high-order attentional processes in learning to read. Because reading is a multi-modal skill, involving multiple sensory modalities and visuo-motor skills, precise control of attention is critical to recruit and manage the diverse functions needed to become skilled. Coordinating the visual flow of incoming text with the auditory processing of the words it represents, while keeping the special demands of each from interfering with the other, requires considerable attentional resources. In addition, a fluent reader must maintain visual attention in a linear cycle to follow the stacked lines of text, while systematically attending to relevant and passing over irrelevant features in the orthographic flow. If attentional systems are bogged down with the burden of compensating for lower-level processing, meaningful reading will be exceedingly hard to accomplish. Likewise, if the attentional system itself is weak, it will be compromised in its ability to perform the unifying, organizing, and evaluative functions necessary to organize and regulate the components of the system.

In fact, one reason why normal children do not generally begin to read until the age of five or six may be that attentional and mnemonic capacities are not sufficiently developed until then. It is interesting to note that many dyslexic children overcome their reading fluency problems as adolescents (Fink, 1993, this volume), when a maturational spurt in executive functions may provide the additional attentional resources needed to compensate for such lower processing problems (Case, 1991; Fischer, 1980; Fischer, Rose, & Rose, this volume). This time lag between learning to speak and learning to read has another consequence that we hinted at earlier: literacy skill is generally parasitic on oral language abilities.

Despite attentional considerations, there are certain cases, notably hyperlexia, in which the developmental dependency between oral and written language is not so asymmetric. That is, some children may acquire phonological decoding skills of text at an unusually young age (Rispens & Van Berckelaer, 1991). However, these children’s reading shows an apparent disjunction between phonology and semantics, in which poor comprehension and abstract thought are coupled with exaggeratedly good and sometimes compulsive phonological decoding of text (Aram, 1997). Thus, while hyperlexic children may be able to read aloud proficiently, they are less adept at relating this skill to the oral language and narrative skills that they should be developing (Nation, 1999). This pattern suggests that without sufficient attentional resources, the only way to read may be to focus exclusively on one narrow aspect of the task, i.e. translating the written word into spoken sound. True reading, reading for comprehension, requires the coordination of several nested levels of processing, not just phonological decoding of text.
Conclusion: implications for practice

Thinking about dyslexia from an evolutionary and neural systems standpoint contributes two potentially useful insights, not always considered.

First, textual codes are recent technologies lacking in specific evolutionary support. This means that the brain systems that are recruited to analyze them are kludges and should be expected to exhibit considerable individual variation. The message for practice is that we should not expect there to be one central disorder underlying all forms of dyslexia, nor should we expect to find one successful therapeutic approach. Moreover, reading skills are parasitic on neural adaptations for spoken language, which are themselves comparatively recent and unprecedented modifications on a much older basic mammalian and primate brain plan. The historical recency of reading suggests that diagnostic and therapeutic approaches that carve the problem according to an evolutionary-anatomical logic are likely to be more effective than those based exclusively on either a linguistic or general learning theory paradigm.

Second, the kludginess of reading skill also means that its neural supports are more diversely distributed within the brain and involve many loosely linked systems. This diversity of components makes it more dependent than other more evolutionarily entrenched functions on attentional and working memory systems for coordinating and integrating the component sub-functions. Thus, reading may be particularly susceptible to disturbances at higher levels of general processing even while it is also subject to problems from lower-level processes (such as visual, auditory, or even language-specific processing).

The nested model of dyslexia has several implications for diagnosing and teaching dyslexic children. It suggests that rather than studying reading behavior linguistically, or even from the perspective of the requisite sensory integration, we need to assess the components of the system hierarchically from the top down, and from early to late in the learning process. Apparent problems with visual recognition or coding transfer between modalities may be strongly influenced by automation or working memory. Even an intact low-level process will appear disorganized and ineffective without the attentional system selectively regulating its operation and its integration with other component processes.

With regard to possible genetic factors in dyslexia, inherited global attentional problems will be likely to consistently cause reading difficulties, whereas lower-level disturbances may or may not have repercussions for reading in a particular generation or individual, because distributive neural works-are will be possible. In general, given the inevitably distributed multi-system nature of reading processes, serious, persistent deficits will typically involve multiple impairments at multiple levels, which complicates the analysis of inherited risk factors. Multi-level and multi-system disturbances undermine the possibility of redistributing functional load, which would allow more robust systems to compensate for more fragile ones. Again, this consideration suggests that multiple modalities of therapy, not a single technique, may be essential for successful intervention in cases of severe dyslexia.

In conclusion, from an evolutionary perspective, it is not dyslexia that stands out as deviant, but rather reading ability itself. Given the lack of evolutionary adaptations for reading combined with the serendipitous origins of this technology, it is in many ways a miracle that reading is possible at all, much less with the automatic ease that many readers develop. Perhaps part of the credit for the surprising success of most people at acquiring this skill lies in the social evolution of textual coding systems themselves, many of which have undergone a considerable change toward "user-friendliness." The gradual replacement of many early logographic and incompletely coded phonetic systems with systems that sacrifice content representation for mere speech representation has better enabled the already evolutionarily streamlined oral language adaptations to carry more of the analytic load involved in reading. Nonetheless, the study of dyslexia should be expected to show wide variation in the timing and patterns of acquisition of reading skill. We should look to this variation to teach us about the logic of the developmental organization of language functions more generally and of reading more specifically in the young child's brain and mind.