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## *Developmental Dyslexia*

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Most children learn to read and spell with ease while others have extraordinary difficulty. The possible causes and correlates of such difficulty have been the focus of a great deal of theorizing since before the turn of the twentieth century, when W. Pringle Morgan (1896) described a 14-year-old boy named Percy who suffered from pronounced difficulty learning to read and spell, despite normal achievement in other academic areas. Because he could find no evidence of definitive brain injury that might have caused the boy's reading and spelling problems, Morgan theorized that these problems were caused by a congenital defect that resulted in difficulty in storing visual impressions of words. James Hinshelwood (1900, 1917) held a similar view and provided the field with the first extensive description of reading impairment in otherwise normal children, which he characterized as "congenital word blindness."

The writings of Morgan and Hinshelwood called initial attention to the possibility that reading difficulties in some children may represent a neurodevelopmental disorder affecting cognitive abilities underlying the ability to learn to read, rather than frank brain injury or environmental causes such as a limited home background or inadequate instruction. Serious consideration was given to this possibility in Samuel T. Orton's seminal monograph in 1925 describing reading difficulties and correlated symptom patterns in poor readers judged to be afflicted by what he called strephosymbolia ("twisted symbols"). Orton believed strephosymbolia to be a perceptual disorder manifested in an aberrant tendency to perceive visual symbols as reversed images ("seeing" b as d or was as saw) and suggested that such difficulties are caused by a developmental delay in the establishment of hemispheric dominance. Delay in establishing hemispheric dominance was said to disrupt development of the child's ability to suppress mirror image counterparts of letters and words and this was presumed to cause optical reversibility in visual perception along with letter orientation and letter sequencing errors in oral reading and writing (Vellutino, 1979).

Orton's theory was widely accepted, especially among practitioners, and dominated the field for well over five decades. Moreover, it motivated the emergence of other visual deficit theories (e.g., Hermann's [1959] spatial confusion theory). More recently, cognitive scientists studying reading processes have turned their attention to severely impaired readers who have at least average intelligence, who do not have general learning problems, and whose reading difficulties are not associated with extraneous factors such as uncorrected sensory deficits, socioeconomic disadvantage, emotional problems, or frequent absences from school. "Dyslexia" and "specific reading disability" are contemporary terms commonly used by reading researchers to refer to this symptom pattern in such children. The disorder has been estimated to occur in 10% to 15% of the population of school children (Lyon, Fletcher, & Barnes, 2002; Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992).

In the present chapter, we provide a selective review of research conducted over the past two to three decades evaluating influential theories of the basic cause(s) of developmental dyslexia. Research in this area of inquiry has pursued causal explanations at the biological, cognitive, behavioral, and environmental levels of analysis (Vellutino, Fletcher, Snowling, & Scanlon, 2004). Our primary concern in this chapter is to distinguish between manifest causes of early reading difficulties and underlying causes of such difficulties. We define manifest causes in terms of observed deficiencies in the knowledge and component skills the child must acquire in order to become a proficient reader and underlying causes in terms of biologically based cognitive deficits or environmental deficits that might impair the acquisition of those skills. Thus, we first define dyslexia and provide documentation of its primary behavioral manifestations. We proceed to discuss theories of dyslexia, specifying cognitive deficits presumed to underlie this disorder, and then go on to address the question of whether there are subtypes of dyslexia. We close with a brief discussion of research documenting the importance of distinguishing between reading problems caused primarily by biologically based cognitive deficits and reading problems caused primarily by experiential and instructional deficits.

Due to space limitations, our review cannot be exhaustive (see Vellutino et al., 2004, for a more detailed review). For example, we do not discuss acquired dyslexia nor do we discuss important areas of inquiry in the study of dyslexia that are treated more extensively in this volume, such as the neurobiological and genetic foundations of dyslexia, and its cross-linguistic manifestations (see Price & McCrory, Pennington & Olson, and Caravolas, this volume).

### **Manifest Causes of Dyslexia: Deficiencies in Reading Subskills**

The study of specific reading disability has made clear that developmental reading difficulties in children with dyslexia are manifested in basic and pervasive deficiencies in word identification, phonological (letter-sound) decoding, and spelling. Deficiencies in these word-level skills may be accompanied by deficiencies in language comprehension and related skills such as vocabulary knowledge and syntactic competence, but this is not necessarily the case. Thus, dyslexia is generally defined at the behavioral level as a develop-

mental disorder characterized by significant difficulties in learning to decode print. The evidence for this generalization is straightforward.

First, there is a great deal of evidence that most children who have difficulties comprehending written text also have basic deficiencies and dysfluencies in word identification, relative to normally developing readers (e.g., Leach, Scarborough, & Rescorla, 2003; Shankweiler et al., 1999). Conversely, children who have basic deficiencies and dysfluencies in word identification are invariably found to have poor reading comprehension (Gough & Tunmer, 1986; Hoover & Gough, 1990; Snowling, 2000a; Vellutino, Scanlon, & Tanzman, 1994; Vellutino et al., 1996).

Second, studies have shown that there is a developmental asymmetry in the acquisition of skill in reading. Whereas word identification skills tend to be more important determinants of reading comprehension in beginning readers than they are in skilled readers, language comprehension skills are more important determinants of reading comprehension in skilled readers than they are in beginning readers. These studies document that adequate facility in word identification is a necessary (though not a sufficient) condition for adequate reading comprehension (Catts, Hogan, & Fey, 2003; Foorman, Francis, Shaywitz, Shaywitz, & Fletcher, 1997; Hoover & Gough, 1990; Vellutino et al., 1994).

Third, there is convergent evidence that most children with dyslexia have significant difficulty learning to map alphabetic symbols to sound and acquiring facility in phonological (letter-sound) decoding (Fletcher et al., 1994; Liberman & Shankweiler, 1979, 1991; Snowling, 1980, 2000a; Stanovich & Siegel, 1994; Torgesen, Rose, Lindamood, Conway, & Garvan, 1999; Torgesen et al., 2001a; Vellutino, 1979; Vellutino et al., 1994, 1996; Vellutino, Scanlon, & Spearing, 1995; Wagner & Torgesen, 1987; Wagner, Torgesen, & Rashotte, 1994). Such difficulties, in turn, appear to be related to limitations in their ability to acquire phonological awareness – that is, conceptual understanding of the idea that spoken words consist of individual speech sounds (phonemes) or combinations of speech sounds (syllables, onsets, and rimes).

The problems experienced by impaired readers in acquiring phonological awareness is confirmed by robust differences between these children and their normally developing peers on measures evaluating sensitivity to rhyme, phoneme segmentation, sound blending, and like measures of phonological awareness. Importantly, there is evidence for a causal relationship between deficiencies in phonological awareness and alphabetic mapping on the one hand and difficulties in acquiring facility in word identification and spelling on the other. Direct evidence for this causal relationship comes from studies finding that training designed to help children acquire phonological awareness and alphabetic mapping skills has a beneficial effect on word identification, spelling, and reading ability in general (Blachman, 2000; Bradley & Bryant, 1983; Foorman, Francis, Fletcher, Schatschneider, & Mehta, 1998; Hatcher, Hulme, & Ellis, 1994; Olson, Wise, & Ring, 1999; Scanlon & Vellutino, 1996; Scanlon, Vellutino, Small, & Fanuele, 2000; Snowling, 2000a; Torgesen et al., 1999, 2001a; Vellutino & Scanlon, 1987; Vellutino et al., 1996).

Deficiencies in phonological awareness and alphabetic mapping also tend to be accompanied by deficiencies in orthographic awareness – that is, sensitivity to the constraints on how the letters in printed words are organized (sud-legal; yxl-illegal). Phonological

and orthographic awareness work in concert to facilitate the acquisition of general orthographic knowledge. General orthographic knowledge is reflected in the child's growing sensitivity to the regularities and redundancies in the writing system (e.g., at in cat, fat, rat). This knowledge is critically important for acquiring reading strategies that help beginning readers reduce the load on visual memory imposed by an alphabetic system, and, thereby, promote automatic word identification (Ehri, 1999). Thus, it should not be surprising to find that dyslexic children are deficient in acquiring general orthographic knowledge (Bruck, 1990; Vellutino et al., 1994).

Finally, deficiencies in lexical skills such as word identification and spelling, along with deficiencies in related skills such as phonological awareness, that are observed in dyslexic children early in their reading development, continue to be evident in the same individuals well into adulthood (Bruck, 1990, 1992; Hatcher, Snowling, & Griffiths, 2002; Pennington, Van Orden, Smith, Green, & Haith, 1990; Satz, Buka, Lipsitt, & Seidman, 1998; Shaywitz et al., 1999). Such deficiencies are apparent in individuals with dyslexia across levels of intelligence outside the mentally deficient range and are indexed either by discrepancies with IQ or simply by low reading scores, independent of discrepancies with IQ (Steubing et al., 2002). Thus, in terms of manifest reading behaviors, dyslexia is most accurately defined as a basic and pervasive disorder affecting the child's ability to learn to decode print.

## Underlying Causes of Dyslexia: Cognitive Deficit Theories

### *Visual perceptual and visual memory deficits*

For many years, a dominant view was that developmental reading difficulties are caused by dysfunction in the visual system. During the 1970s and 1980s, a series of related studies were conducted that systematically evaluated these theories using a wide variety of visual processing paradigms that were designed to minimize the influence of linguistic coding processes (Fletcher, Foorman, Shaywitz, & Shaywitz, 1999; Snowling, 2000a; Vellutino, 1979). These studies were motivated by the observation that research supporting theories of dyslexia, which implicated dysfunction in visual processes as basic causes of the disorder, typically did not control for confounding by verbal mediation. Thus, in several studies comparing dyslexic and normal readers on tasks evaluating visual memory, spatial orientation, and visual sequencing in the processing of letters and words (e.g., b, d, was, saw, loin, lion), it was found that performance on such tasks was equivalent in these groups when the task required a written rather than a naming response (Vellutino, 1979). More impressive were findings from studies showing that performance in dyslexic and normal readers was equivalent on tasks evaluating the same processes when the letters and words were taken from a novel orthography (written Hebrew).

If visual abilities do not distinguish reliably between dyslexic and normal readers, then it might be expected that such abilities would not strongly predict performance on measures of reading ability. In fact measures of visual abilities have been found to be relatively poor predictors of performance on measures of word identification, spelling,

pseudoword decoding, and reading comprehension (Vellutino et al., 1994). Taken together, these findings suggest that difficulties in learning to read are not caused by impairments in visual processing of the types implicated in visual deficit theories of dyslexia that dominated the early literature.

### *Low-level visual deficits*

Difficulties in learning to read have also been attributed to low-level visual deficits, in particular, visual tracking problems caused by oculomotor deficiencies (Getman, 1985); visual masking effects caused by a hypothesized deficit in the "transient visual system" (Badcock & Lovegrove, 1981; Breitmeyer, 1989; Lovegrove, Martin, & Slaghuis, 1986; Stein, 2001); and abnormalities in visual motion perception (Eden et al., 1996). Moreover, transient system and motion perception deficits have both been linked to dysfunction in the magnocellular visual subsystem. The magnocellular subsystem is one of two parallel components of the visual system, the other being the parvocellular system. The magnocellular system consists of large neurons that are sensitive to movement and rapid changes in the visual field. It is often called the "transient system," insofar as it is presumed to be responsible for suppressing the visual trace that normally persists for a short duration (250 milliseconds) after a visual stimulus has disappeared. The parvocellular system consists of densely packed, small neurons that are sensitive to color and fine spatial details. In reading, the parvocellular system is believed to be operative during eye fixations and the magnocellular (transient) system is believed to be operative during saccadic movements of the eyes.

The visual tracking theory of dyslexia has been discredited by well-controlled eye movement studies finding no differences between dyslexic and normal readers on visual tracking of nonverbal stimuli (Olson, Kliegl, & Davidson, 1983; Stanley, Smith, & Howell, 1983). As regards magnocellular dysfunction, it has been suggested that dyslexics suffer from a deficit in the inhibitory function of the transient system. This deficit is said to produce a visual trace of abnormal duration that creates masking effects and consequent visual acuity problems when these children are reading connected text. Indirect evidence for this suggestion has been provided by studies demonstrating that poor and normal readers have different contrast sensitivity functions, such that poor readers require greater luminosity than normal readers when processing low spatial frequency grids (Badcock & Lovegrove, 1981; Lovegrove et al., 1986; Martin & Lovegrove, 1984). Observations of abnormal motion perception in individuals with dyslexia are offered as confirmatory evidence of magnocellular dysfunction in this population (Eden & Zeffiro, 1998). Additional support for this possibility is provided by anatomical and electrophysiological studies demonstrating structural and functional anomalies in the magnocellular pathways of a small number of dyslexic individuals (Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Livingstone, Rosen, Drislane, & Galaburda, 1991).

However, no causal relationship has been established between transient system dysfunction and early reading difficulties. Moreover, there is no evidence that dyslexic readers experience visual acuity and visual masking problems under normal reading conditions. Indeed, the performance patterns prompting inferences of transient system deficits in poor readers have also been observed in some normal readers. As pointed out by Hulme

(1988), the trace persistence theory of reading disability predicts that dyslexic children should be impaired only when they are reading connected text and not when they are reading printed words one at a time under foveal vision conditions. This, of course, is counter to the common observation that poor readers have difficulty in word identification under both conditions. Additionally, Eden, Stein, Wood, and Wood (1995) found that while low-level visual processes contributed unique variance in predicting reading skills in poor readers, the amount of variance was quite small compared to the variance contributed by phonological skills.

Finally, intervention studies based on visual deficit hypotheses do not appear to facilitate the word recognition difficulties that reflect the core difficulty in children with dyslexia (Iovino, Fletcher, Breitmeyer, & Foorman, 1999). Thus, we doubt that anomalies in low-level visual processes associated with magnocellular dysfunction are causally related to difficulties in learning to read, though such anomalies may well serve as biological markers signifying deficits in other systems that may be impaired in dyslexia (Eden & Zeffiro, 1998; Fletcher et al., 1999).

### *Language-based deficits*

*Phonological coding deficits.* There is now strong evidence that reading difficulties in dyslexia can be traced to language-based deficits. Indeed, there is especially strong evidence that such difficulties, in most cases, can be traced to weak phonological coding – a deficient ability to use speech codes to represent information in the form of words and word parts. Thus, children with dyslexia are believed to be encumbered by poorly specified phonological representations that make it difficult for them to acquire phonological skills such as phonological awareness, alphabetic mapping, and phonological (letter-sound) decoding, along with related skills such as orthographic awareness (Elbro, 1997; Liberman & Shankweiler, 1979, 1991; Snowling, 2000a; Stanovich, 1988; Vellutino, 1979). It has also been suggested that weak phonological coding may be the cause of other problems that contribute to difficulties in learning to read, especially difficulties in storing and retrieving words in spoken language (Elbro, 1997; Fletcher et al., 1999; Gathercole & Baddeley, 1990; Snowling, 2000a; Torgesen, Wagner, Rashotte, Burgess, & Hecht, 1997; Vellutino, 1979; Vellutino et al., 1994, 1996; Wagner et al., 1994). Difficulties in word storage and retrieval could impair the child's ability to establish strong connective bonds between the visual and verbal counterparts of printed words. This, in turn, could impair his or her ability to store quality representations of word spellings, thus impeding the acquisition of fluency in word identification. Finally, weak phonological coding could also impair reading comprehension by virtue of the deleterious effect it has on working memory (Daneman & Carpenter, 1980).

Support for weak phonological coding as a basic cause of reading disability comes from studies showing that poor readers tend to perform below the level of normal readers on tests evaluating phonological skills such as phonological awareness, letter-sound decoding, visual-verbal learning, and verbal memory. These studies also show that such measures predict reading performance quite reliably (Blachman, 2000; Fletcher et al., 1994;

Stanovich & Siegel, 1994; Vellutino et al., 1994, 1995, 1996; Wagner et al., 1994). Finally, several studies have documented that poor readers tend to perform below normal readers on both speech (categorical) perception and production tasks, thereby providing additional (though somewhat inconsistent) evidence that dyslexic readers are encumbered by weak phonology (Godfrey, Syral-Lasky, Millay, & Knox, 1981; Griffiths & Snowling, 2001; Manis et al., 1997; Mody, Studdert-Kennedy, & Brady, 1997).

*Semantic and syntactic deficits.* Although existing evidence indicates that reading problems in most children are caused by deficient phonological skills, deficiencies in semantic and syntactic skills may also play a role (Dickinson & Tabors, 2001; Snow & Tabors, 1993; Vellutino, 1979; Vellutino & Scanlon, 1982). Given the likelihood that children will have less difficulty in learning to read words that are in their speaking vocabularies than in learning to read words that are not, it seems reasonable to consider the possibility that deficient vocabulary knowledge is a cause of reading problems in at least some children. Support for this possibility comes from studies finding that impaired reading development was associated with vocabulary deficits in both disadvantaged children and children with limited proficiency in English (Dickinson & Tabors, 2001; Snow Barnes, Chandler, Goodman, & Hemphill, 1991; Tabors & Snow, 2001). Furthermore, vocabulary knowledge acquired before first grade has been found to be a good predictor of later word-level reading skills as well as reading comprehension (Catts, Fey, Zhang, & Tomblin, 1999; Dickinson & Tabors, 2001; Scarborough, 1990; Snow et al., 1991; Snowling, Gallagher, & Frith, 2003; but see Schatschneider, Fletcher, Francis, Carlson, & Foorman, 2004; Storch & Whitehurst, 2002).

Additional support comes from studies using experimental tasks simulating beginning reading (Vellutino & Scanlon, 1987; Vellutino, Scanlon, & Spearing, 1995). In such tasks, children find it easier to learn to “read” high meaning (concrete) words than to learn to read low meaning (abstract) words or nonsense words. Thus, we suggest that semantic deficiencies could be a factor contributing to reading difficulties in some children, especially second-language learners or those who come from impoverished backgrounds (see also Goswami, 2001, and Metsala & Walley, 1998, for interesting discussions on the role of vocabulary development in the acquisition of phonological skills). However, such deficiencies tend to be more closely linked to comprehension processes than to word recognition processes (Storch & Whitehurst, 2002).

Finally, given the demonstrated importance of linguistic context in facilitating and monitoring word identification, especially in poor readers (Perfetti & Roth, 1981; Stanovich, 1980; Tunmer, 1989; Tunmer & Chapman, 1998), it is possible that syntactic deficits that impede a child’s ability to use linguistic context to facilitate word identification and reading for meaning could contribute to difficulties in learning to read. We doubt, however, that such deficits would be a primary cause of such difficulties. Indeed, syntactic knowledge does not often distinguish between dyslexic and normally achieving readers, as these populations have typically been defined. A possible exception occurs in cases where children with long-standing reading disorders have been compared with controls (e.g., Stanovich, 1986; Vellutino, Scanlon, & Tanzman, 1988; Vellutino et al., 1995). In such cases, vocabulary and syntactic deficits may be a consequence of prolonged reading difficulties, rather than their cause.

*Low-level auditory deficits.* Another theory of dyslexia that has attracted widespread attention in recent years is Tallal's (1980) temporal order perception theory (see Farmer & Klein, 1995, and Tallal, 2003, for recent reviews). In a study motivated by previous research with language-impaired children (Tallal & Percy, 1973), Tallal (1980) found that poor readers (selected from a sample of children with significant oral language disorders) performed below normal readers in making temporal order judgments (TOJ). These temporal order judgments involved detecting the order of pairs of high and low tones presented either at short (e.g., 50 ms) or long (e.g., 400 ms) interstimulus intervals (ISIs); the poor readers performed as well as the normal readers on the TOJ task at long ISIs, but were impaired when the interstimulus intervals were short. Because of a high correlation between performance on the TOJ task and performance on a nonsense word decoding task ( $\rho = .81$ ), Tallal inferred that children with dyslexia suffer from a basic, nonlinguistic deficit in temporal resolution of rapidly changing auditory stimuli. In turn, this basic deficit was said to impair speech perception, and, thereby, the acquisition of skills such as phonological awareness and phonological decoding. A later study by Reed (1989) replicated Tallal's findings with TOJ tasks involving stop consonants and brief tones, but not when they involved steady-state vowels.

Although these results would appear to offer support for Tallal's theory of dyslexia, they are inconclusive, because it is not clear that the poor readers' difficulties on both the verbal and nonverbal TOJ tasks arise from the same underlying perceptual mechanism. Thus, in a series of experiments that varied discriminability of speech stimuli, Mody, Studdert-Kennedy, and Brady (1997) found that poor readers had more difficulty than normal readers making temporal order judgments at short ISIs only when the stimuli were acoustically similar consonant-vowel (CV) syllables (e.g., /ba/-da/), but not when they involved CV syllables that were acoustically very different (e.g., /ba/-sa/, Experiments 1a and 1b). Moreover, when these two groups were given TOJ tasks using non-speech stimuli that were acoustically matched to the onset transitions of the speech stimuli (Experiment 2), no statistically significant differences between dyslexic and normal readers emerged at any of the ISIs used in the experiment.

Support for Tallal's TOJ theory of dyslexia is also undermined by results from two recent studies evaluating the theory with well-defined samples of dyslexic readers that controlled for the presence of attention-deficit hyperactivity disorder (ADHD) (Breier, Fletcher, Foorman, & Gray, 2002; Waber et al., 2001). In both of these studies, the dyslexic readers performed below the level of normal readers on TOJ tasks involving speech stimuli, and only one of these studies reported differences on the nonverbal TOJ tasks (Waber et al., 2001). However, there were no differential effects attributable to variations in ISIs observed in either study, suggesting that previous studies may not have used adequately defined samples of poor readers free from other problems such as ADHD or pervasive oral language difficulties (Fletcher et al., 1999). Thus, although there is strong support for the possibility that children with dyslexia have difficulties with speech perception that produce deficits on temporal processing tasks, there is, at best, weak and equivocal support for the contention that they have a pervasive deficit in auditory temporal processing that is causally related to the reading problem (see also Best & Avery, 1999; Bishop, Carlyon, Deeks, & Bishop, 1999; Nitttrouer, 1999, for similar conclusions).



### *Subtypes of dyslexia*

In attempting to account for the diverse range of cognitive deficits associated with dyslexia, some researchers have suggested that the population of dyslexic children is heterogeneous (Ellis, 1984; Lyon et al., 2002; Rourke, 1975). Further, such heterogeneity may be at least partially explained by the existence of distinct subtypes. The literature on subtypes is voluminous, representing hundreds of published studies since 1978. This research is generated by approaches to subtyping based either on rational division of poor readers into subtypes in accord with clinical experience and visual inspection of patterns of performance, or the application of multivariate classification methods (e.g., cluster analysis) to batteries of cognitive, reading, or neuropsychological tests. Many of these studies have not yielded results that have been replicated or shown to be useful beyond the partitioning of individuals into groups (Hooper & Willis, 1989; Lyon et al., 2002). In the next section, we review the evidence for four subtyping hypotheses that have been more persistent and have maintained some focus of interest in the field, largely because they do appear to have a theoretical basis.

### *Double deficit subtypes*

*Accuracy versus rate subtypes.* Lovett (1984) (Lovett, Steinbach, & Frijters, 2000) proposed two subtypes of reading disability based on a distinction between accuracy of word identification and fluency in word identification in reading connected text. In a series of studies involving the two dyslexic subtypes (accuracy vs. rate disabled) and a normal reader sample, the accuracy-disabled readers performed poorly on a range of oral and written language measures. In contrast, the rate-disabled readers displayed deficiencies that were more apparent in difficulties in reading connected text and spelling. Reading comprehension was highly correlated with word recognition skill in the accuracy-disabled group and this group was therefore found to be deficient on all measures of reading achievement. The rate-disabled group, however, was impaired only on reading comprehension measures related to fluency. Moreover, in intervention studies (Lovett, Ransby, & Barron, 1988; Lovett et al., 2000), differences between the accuracy-disabled and rate-disabled groups, in the efficacy of different treatments, were apparent in that training in word recognition skills improved reading outcomes in both groups, whereas training in contextual reading improved reading outcomes only in the rate-disabled group. However, the evidence for subtype by treatment interactions was weak and reading gains on standardized measures observed in these studies did not move many children into the average range, in spite of statistically significant results (Lyon et al., 2002).

*Phonological awareness versus rapid naming subtypes.* Wolf, Bowers, and their colleagues (Bowers, Golden, Kennedy, & Young, 1994; Bowers & Wolf, 1993; Wolf, Bowers, & Biddle, 2000; Wolf, Pfeil, Lotz, & Biddle, 1994) have suggested that there are three subtypes of reading disability defined by (1) deficiencies in phonological awareness that disrupt word recognition; (2) slow naming speed that disrupts orthographic processing; and (3) "double deficits" in both phonological awareness and rapid naming. They also

suggest that naming speed deficits are caused by disruption in a “precise timing mechanism” that influences speed of processing and, thereby, temporal integration of the letters in printed words (Bowers & Wolf, 1993; Bowers et al., 1994; Wolf et al., 1994, 2000). Within this view it is assumed that if a word’s letters cannot be identified with sufficient ease and rapidity, they will not be processed close enough in time to detect orthographic patterns (e.g., at in cat, rat, fat). In turn, this problem will impair the child’s ability to store distinct and unitized representations of word specific spellings.

Three types of research provide support for Wolf and Bower’s version of the double deficit theory. First, studies finding that naming speed tasks (e.g., rapid naming of letters or digits) contribute variance to performance on tests evaluating reading achievement beyond that contributed by tests evaluating phonological skills (e.g., Manis, Doi, & Bhadha, 2000; Wolf et al., 2000). Second, studies finding that the double deficit subtype generally performs below the single deficit subtypes on tests evaluating reading achievement (Wolf et al., 2000). Third, studies finding that phonological skills are more highly correlated with accuracy in word identification than is rapid naming ability, whereas rapid naming ability is more highly correlated with fluency in word identification than are phonological skills (Manis et al., 2000; Wolf et al., 2000).

Although these findings are suggestive, the double deficit theory can be questioned on several grounds. First, the type of serial letter processing said to be impaired in children manifesting naming speed deficits has long since been discredited as a component process in word recognition (Gough, 1984). Second, recent research suggests that observed relationships between rapid naming and measures of reading ability may be an artifact of the failure to control for prior reading ability, and, thereby, for the variance phonological skills and rapid naming ability share with reading ability. Thus, Torgesen et al. (1997) found that phonological awareness but not rapid naming accounted for unique variance on reading and orthographic coding tasks administered at later points in time when initial reading performance was controlled. Finally, the larger differences observed between children in double and single deficit subgroups have been found to be due primarily to deficiencies in phonological awareness and related phonological skills, rather than to the combined effects of phonological and naming speed deficits (Compton, DeFries, & Olson, 2001; Schatschneider, Carlson, Francis, Foorman, & Fletcher, 2002). These latter findings compromise a basic assumption of the double deficit theory of reading disability, while favoring phonological deficit explanations of this disorder.

*Phonological versus orthographic subtypes.* An influential approach to subtyping has been cast within the dual-route framework of reading (Coltheart, this volume). According to this model, the reading system comprises two subsystems – a sublexical system (“route”) mediated by phonological rules that relate graphemes to phonemes, and a visual-orthographic lexical system that by-passes the phonologically mediated system. Some children, described as having “phonological dyslexia,” have problems with the operation of the phonological route, whereas others, described as having “surface dyslexia,” have difficulties with the visual-orthographic route (Castles & Coltheart, 1993). Thus, whereas “phonological dyslexics” show poorer reading of pseudowords than exception words, “surface dyslexics” show better pseudoword than exception word reading.

Although there is little doubt that phonological dyslexia is a valid subtype, whether surface dyslexia can be reliably defined is arguable (Stanovich, Siegel, & Gottardo, 1997).

Murphy and Pollatsek (1994) did not obtain evidence supporting the surface dyslexia subtype. In contrast, Manis, Seidenberg, Doi, McBride-Chang, & Peterson (1996) and Stanovich et al. (1997) did obtain evidence that supported this subtype, but the evidence was observed primarily in children younger than those used in Murphy and Pollatsek (1994) (see also Coltheart, this volume).

Stanovich et al. (1997) obtained additional evidence suggesting that most children with dyslexia have difficulties at both the phonological and orthographic level of the word recognition process. Children who were identified as showing surface dyslexia, based on comparisons with age-matched normal readers, did not show this reading profile when the comparison group was younger reading-age matched children. At the same time, Stanovich (2000) suggested that surface dyslexia appeared to represent a subtype that was not stable across definition or age, and may represent a transient delay in the development of word recognition skills. This finding was recently supported by Zabell and Everatt (2002), who found that adults with orthographic and phonological dyslexia did not differ on measures of phonological processing.

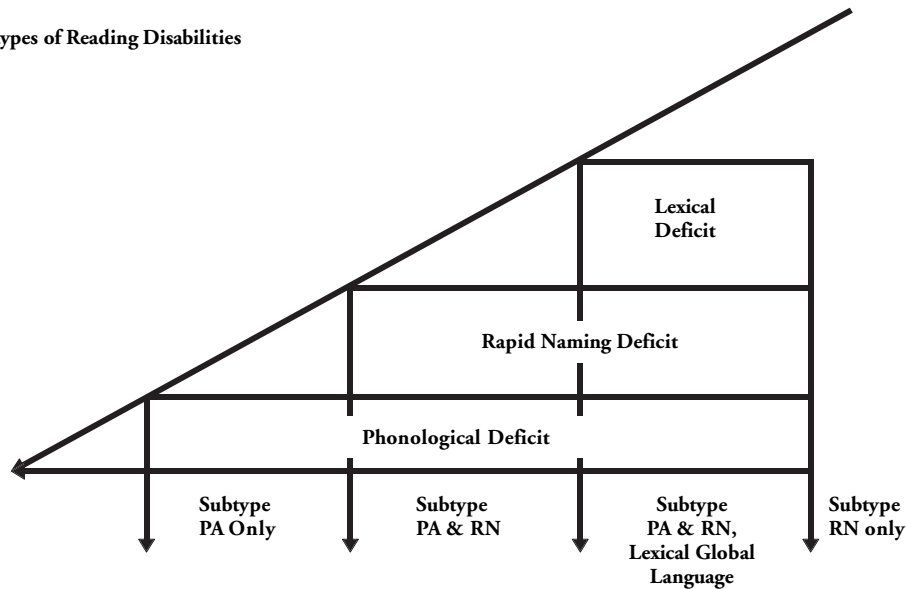
*Phonological core-variable differences classification.* In all the subtyping schemes discussed, the largest group remains one with a basic impairment in phonological processing. To account for the primacy of phonological deficits, and variation in other cognitive skills characteristic of dyslexia, Stanovich (1988) formulated the phonological core-variable differences model. This model suggests that phonological processing is at the core of all word recognition disabilities. However, children may have difficulties outside the phonological domain that do not directly contribute to the word recognition difficulties. For example, impairments in vocabulary could interfere with comprehension, leading to more pervasive disturbances of language that would result in a “garden variety” form of reading disability. Others could show fine motor and visual perceptual problems that are unrelated to word recognition or other domains of reading.

In a large-scale study of the performance of normally developing and reading disabled children on a range of cognitive measures, Morris et al. (1998) provided support for this model. The study relied on a number of theories to select potential variables to be used in subtyping, including measures of phonological skills, rapid naming, short-term memory, vocabulary, and visual perceptual skills.

Nine subtypes emerged from Morris's analyses, including five subtypes with specific reading disability, two subtypes with pervasive impairments in language and reading, and two representing normally achieving groups of children. Importantly, six of the seven reading disability subtypes shared impairment in phonological awareness skills; the largest specific subtype had impairments in phonological awareness, rapid naming, and verbal short-term memory. The other reading disability subtypes varied in rapid automatized naming and verbal short-term memory abilities. The two subtypes with pervasive impairments in language were clearly indexed by impairments in these areas and in vocabulary knowledge.

Figure 19.1 presents a schematic that summarizes the major finding of this study. It shows that the subtypes essentially varied in impairment in phonological processing, rapid naming, and lexical skills: one group of subtypes impaired in phonological awareness and/or verbal short-term memory, a subtype impaired in these two skills as well as rapid

## Subtypes of Reading Disabilities



**Figure 19.1** Subtypes of reading disabilities.

naming, and a subtype that adds lexical deficits representing children with more pervasive language impairments. Finally, one subtype in figure 19.1 was not impaired in phonological awareness, but had difficulties in rate of processing, as manifested in performance on rapid naming tests and other measures evaluating speed of processing. This rate-based subtype was not impaired in word recognition accuracy, but had difficulties on measures of reading fluency and comprehension, consistent with more recent formulations of the double deficit model and the accuracy-rate subtypes.

Altogether, these results highlight the prominent role of phonological processing as a causal deficit in dyslexia, as well as the need for more research to better understand the relationship between reading disability and other related cognitive deficits.

### *Experiential and instructional factors*

Although there is evidence that some poor readers have structurally and functionally different architectures for processing spoken and written language compared with normal readers (Grigorenko, 2001; Lyon et al., 2002; Vellutino et al., 2004), it is also apparent that early reading difficulties in some poor readers may be caused primarily by adverse environmental conditions for language and literacy development or by poor teaching. Indeed, the adverse effects of inadequate prereading experience and/or inadequate instruction may lead to reading skill deficiencies that mimic the effects of those seen in children with dyslexia who do not meet the traditional exclusionary criteria. Because the acquisition of important reading subskills, such as phonological awareness and letter-sound

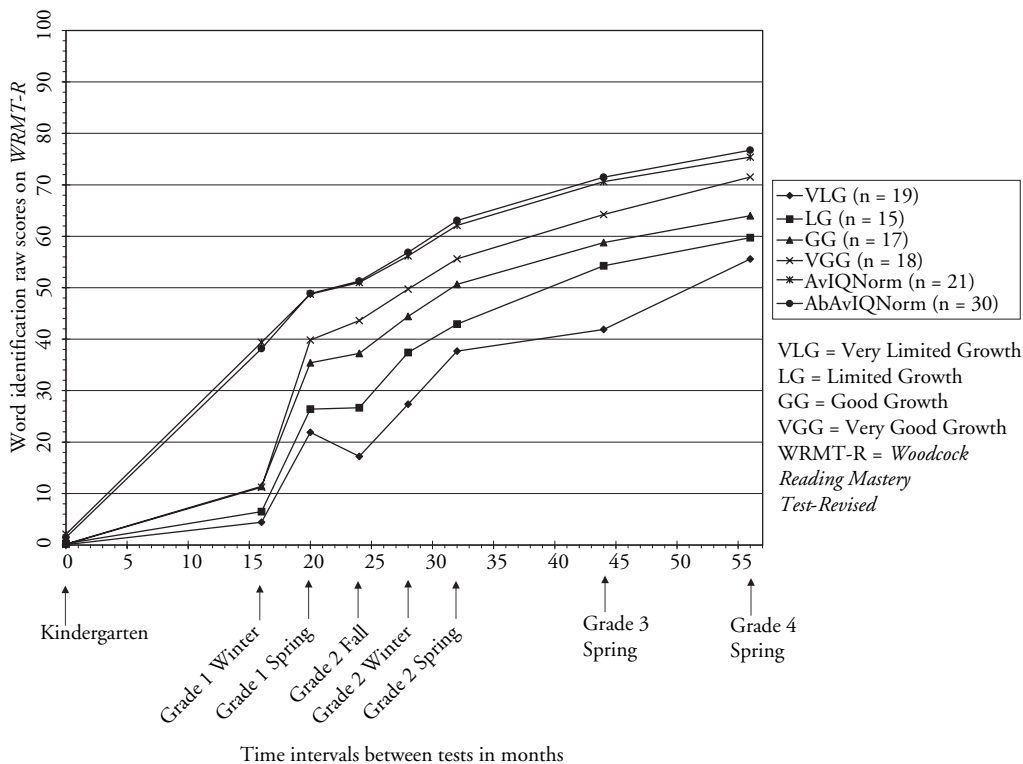
decoding, can be adversely influenced by the type of reading instruction to which a child has been exposed (Foorman et al., 1998), it is important to establish the presence of adequate instruction before assuming that the cause of early reading difficulties is biological in origin. Many children who are identified as dyslexic may not have received the instruction they needed (Lyon et al., 2001).

In a longitudinal-intervention study that was specifically designed to distinguish between children who have reading difficulties because of adverse environmental circumstances and those who have constitutionally based difficulties (Vellutino et al., 1996), the reading achievement of children identified in mid-first grade as poor or normally developing readers was periodically assessed from the time they entered kindergarten through the end of fourth grade, that is before and after their reader status was determined and before and after implementation of remedial intervention for the poor readers. The poor readers were given daily one-to-one tutoring for up to two semesters (depending on progress), and tests evaluating reading-related cognitive abilities were administered to children in all groups in kindergarten, first, and third grades. The findings of the study are consistent with the possibility that early reading difficulties in most impaired readers are related to limitations in early literacy experience and instruction.

First, it was found that emergent literacy skills such as letter naming and phonological awareness were deficient in virtually all of the kindergarten children who were subsequently identified as poor readers in first grade. Second, almost 70% of the tutored children were brought to within an average range of reading achievement after only one semester, and most maintained this level of functioning through the end of fourth grade (see figures 19.2 and 19.3). Because the intervention program was comprehensive, highly individualized, and reasonably well balanced, in terms of the emphasis placed on both word-level and text processing skills, it is fair to assume that it helped compensate for core reading instructional approaches that often did not differentiate instruction for children struggling to learn to read and who often received little explicit instruction in the alphabetic principle.

Third, the poor readers who were found to be the most difficult to remediate performed well below the normal readers, and quite often below the poor readers who were readily remediated, on kindergarten, first, and third grade tests evaluating phonological abilities such as phonological awareness, verbal memory, confrontational naming, and rapid serial naming. Furthermore, although there were no statistically significant differences among the groups on semantic, syntactic, and visual measures, the tutored groups tended to perform below the normal readers on these measures as well as on most of the phonological measures.

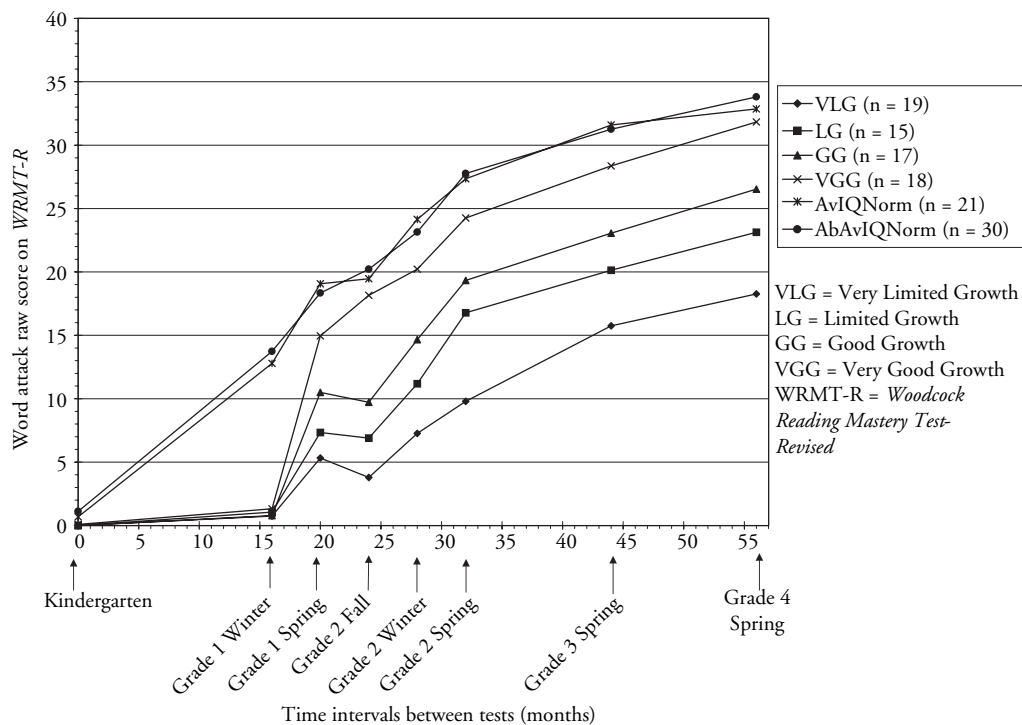
Vellutino et al. (1996) interpreted this pattern of results as evidence that experiential and instructional deficits are often the primary cause of early reading difficulties. And, given that the normal readers in this study generally scored above national norms on the measures of reading achievement, the poor scores of the tutored children on the semantic, syntactic, and visual measures were thought to imply that they were less well prepared to learn to read than the normal readers, rather than implying that the cognitive abilities evaluated by these measures were seriously deficient in these children. This interpretation is more in keeping with "gradation of risk," rather than categorical conceptualizations of dyslexia (Ellis, 1984; Olson & Gayan, 2001; Pennington & Lefty, 2001; Snowling et al., 2003; Scarborough, 1990; Stanovich, 1988).



**Figure 19.2** Growth curves for mean raw scores on the WRMT-R word identification subtest for normal and tutored poor readers.

It is interesting to note, in connection with this latter point, that in this study, the performance decrements of the children who were found to be difficult to remediate on the various measures of reading-related cognitive abilities, administered in kindergarten, first, and third grade, were generally greater than that among children who were more readily remediated. This finding is consistent with results from recent longitudinal studies of children at family-risk for dyslexia because they have a first-degree affected relative. In these studies, at-risk children tended to perform below preschool children from nondyslexic families, not only on measures of reading achievement administered at later points in their development, but also on measures of reading-related cognitive abilities, such as phonological awareness, speech perception, rapid naming, verbal memory, and oral language abilities. This was found to be true, even in high-risk children who went on to be normal readers (Pennington & Lefty, 2001; Scarborough, 1990). Note, however, that children not only inherit genes that may make them at risk for dyslexia, but also share environments that result in greater or lesser access to reading materials, parents who read to them, and schools with effective instructional programs (Olson & Gayan, 2001).

Additional support for the possibility that early reading difficulties in many impaired readers are caused primarily by experiential and instructional deficits comes from other intervention studies which have shown that most impaired readers can acquire at least average-level reading skills if they are identified early and are provided with comprehen-



**Figure 19.3** Growth curves for mean raw scores on the WRMT-R word attack subtest for normal and tutored poor readers

sive and well-integrated reading instruction tailored to their individual needs (Clay, 1985; Iversen & Tunmer, 1993; Pinnell, 1989; Scanlon et al., 2000; Torgesen et al., 1999, 2001a). Moreover, evidence from classroom-based studies suggests that comprehensive, well-balanced reading instruction can prevent long-term reading difficulties in children who would otherwise qualify for a diagnosis of dyslexia (Foorman et al., 1998; Scanlon & Vellutino, 1996).

One other finding from the Vellutino et al. (1996) study is worth noting. The widespread use of IQ scores to classify children as disabled readers or to predict reading achievement was questioned by the finding that the tutored groups did not differ on tests of intelligence, nor did they differ from an average IQ normal reader group on these tests. At the same time, the average IQ normal reader group did not differ from an above-average IQ normal reader group on tests of basic word level skills (e.g., word identification, phonological decoding) administered from kindergarten through the end of fourth grade. In addition, IQ-achievement discrepancy scores were not significantly correlated with initial growth in the reading performance of the tutored children following one semester of one-to-one daily tutoring. These findings are consistent with a large body of research showing that poor readers with IQ discrepant and IQ nondiscrepant reading scores cannot be adequately differentiated, vis-à-vis response to remediation or prognos-

sis (Fletcher et al., 2002; Lyon et al., 2001, 2002; Vellutino, Scanlon, & Lyon, 2000). Moreover, they are consistent with the results of two recent meta-analyses showing null to small differences between the cognitive skills of these two populations (Hoskyn & Swanson, 2000; Steubing et al., 2002). The combined results have led many to conclude that IQ is irrelevant to reading disability.

In summary, the research to date suggests that individual differences in reading ability result from complex interactions between naturally endowed cognitive abilities underlying the ability to learn to read on the one hand, and literacy experiences and instruction on the other. While some children will have little difficulty learning to read, despite less than optimal literacy experiences and instruction, others will have a great deal of difficulty learning to read, even when literacy experiences and instruction are optimal (Lyon et al., 2001, 2002). Future research on biological factors would do well to focus on children who demonstrate an inability to respond to instruction that appears effective for most of their peers.

## Conclusions

Much has been learned about the causes and correlates of early reading difficulties in children with dyslexia. There is strong evidence that problems in acquiring adequate word identification skills constitute the basic difficulty for most of these children. Word identification problems, in turn, appear to result from underlying deficiencies in phonological skills, such as phonological awareness, alphabetic mapping, and phonological decoding, that lead to difficulties in establishing associative bonds between a word's spoken and printed counterparts. Because of the unique structural properties of an alphabetic system, it is clear that these and other phonological skills carry greater weight as determinants of reading ability in novice readers than do semantic and syntactic skills, whereas semantic and syntactic skills carry greater weight as determinants of reading ability, especially comprehension, in more advanced readers.

As regards underlying causes in children who might qualify for a diagnosis of dyslexia, the relevant research suggests that reading difficulties in most such children are caused by basic deficits in phonological coding. Phonological coding deficits tend to be manifested in reliable and robust differences between dyslexic and normal readers, not only on measures evaluating phonologically based reading subskills such as alphabetic mapping and phonological decoding, but also on measures evaluating phonological skills such as phonological awareness, verbal memory, and name encoding and retrieval. Semantic and syntactic deficits do not appear to be a primary cause of reading difficulties in most dyslexic children. Where they occur, they are quite likely a consequence of long-standing reading difficulties or of a comorbid oral language disorder. Yet, semantic and syntactic deficits may be a primary cause of reading difficulties in some children, in particular those from disadvantaged or bilingual populations. They could certainly exacerbate and complicate reading difficulties caused primarily by other factors.

Reading disability research has also established that reading difficulties are not caused by visual deficits of the types proposed in seminal theories of dyslexia, such as Orton's



(1925) optical reversibility theory and other visual deficit theories that subsequently appeared in the reading disability literature. And, although more recent research provides suggestive evidence that some poor readers may suffer from low-level sensory deficits in both the visual and auditory spheres, the evidence is inconclusive, and, in some instances, equivocal and controversial.

Nevertheless, because of the heterogeneity in cognitive functioning often observed in impaired readers, there have been significant disagreements about the primacy of phonological deficits as the central cause of dyslexia, along with a concomitant increase in attempts to identify “subtypes” of dyslexia. We have reviewed four hypotheses regarding subtypes of dyslexia: two variants of the double deficit subtype hypothesis, Lovett’s accuracy and rate subtypes (Lovett, 1984), and Wolf and Bowers’s phonological and naming speed deficit subtypes (Wolf et al., 2000); Castles & Coltheart’s (1993) distinction between (1993) phonological dyslexia and surface dyslexia; and subtypes predicted by Stanovich’s (1988) phonological core-variable differences model of dyslexia. We concluded that the extant evidence is most compatible with Stanovich’s phonological core-variable differences model, although we underscored the need to better understand the relationship between specific reading disability and other cognitive deficits that may be reliably observed in children who qualify for a diagnosis of dyslexia.

Finally, there is now considerable evidence, from recent intervention studies, that reading difficulties in most beginning readers may not be directly caused by biologically based cognitive deficits intrinsic to the child, but may in fact be related to the opportunities provided for children to learn to read. As such, current estimates of the incidence of reading disabilities as an intrinsic biological disorder may be greatly inflated. However, these same studies, along with recent family-risk and life-span dyslexia studies, provide strong reason to believe that a small but significant percentage of impaired readers may well be afflicted by basic cognitive deficits of biological origin, especially phonological deficits, that make it difficult for them to acquire basic word level skills, despite instruction to which most children respond. Among children of this description, the most severely impaired are difficult to remediate, and we suggest that a diagnosis of dyslexia can be more confidently applied to such children than to impaired readers who are readily remediated. Future research targeting children with dyslexia should focus on those who are demonstrably nonresponders to instruction. Such studies may help establish not only the nature of the specific cognitive difficulties associated with dyslexia, but also may help establish its neurobiological basis.<sup>1</sup>

## Note

1. Much of the work discussed in this chapter was supported by grants from the National Institute of Child Health and Human Development to the co-authors. The data for the intervention study reported in Vellutino et al. (1996) were collected as part of a project conducted under the auspices of a special Center grant (P50HD25806) awarded to the Kennedy Krieger Institute by NICHD. Other grants involving Vellutino supported this review, specifically RO1 HD34598 and RO1 HD42350. Several grants involving Fletcher also supported this review, including P50 HD21888, R01 HD35938, and NSF 9979968.