Journal of Child Psychology and Psychiatry 45:1 (2004), pp 2–40

Specific reading disability (dyslexia): what have we learned in the past four decades?

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We summarize some of the most important findings from research evaluating the hypothesized causes of specific reading disability ('dyslexia') over the past four decades. After outlining components of reading ability, we discuss manifest causes of reading difficulties, in terms of deficiencies in component reading skills that might lead to such difficulties. The evidence suggests that inadequate facility in word identification due, in most cases, to more basic deficits in alphabetic coding is the basic cause of difficulties in learning to read. We next discuss hypothesized deficiencies in reading-related cognitive abilities as underlying causes of deficiencies in component reading skills. The evidence in these areas suggests that, in most cases, phonological skills deficiencies associated with phonological coding deficits are the probable causes of the disorder rather than visual, semantic, or syntactic deficits, although reading difficulties in some children may be associated with general language deficits. Hypothesized deficits in general learning abilities (e.g., attention, association learning, cross-modal transfer etc.) and low-level sensory deficits have weak validity as causal factors in specific reading disability. These inferences are, by and large, supported by research evaluating the biological foundations of dyslexia. Finally, evidence is presented in support of the idea that many poor readers are impaired because of inadequate instruction or other experiential factors. This does not mean that biological factors are not relevant, because the brain and environment interact to produce the neural networks that support reading acquisition. We conclude with a discussion of the clinical implications of the research findings, focusing on the need for enhanced instruction.

The question of why some children have difficulty learning to read has been the focus of a great deal of research over the past four decades and much has been learned about the probable and improbable causes of such difficulty. Of special interest in this very rich and prolific area of inquiry have been children who have at least average intelligence, who do not have general learning difficulties, and whose reading problems are not due to extraneous factors such as sensory acuity deficits, socioeconomic disadvantage, and like factors. Reading problems in such children are manifested in extreme difficulties in acquiring basic reading subskills such as word identification and phonological (letter-sound) decoding. Such difficulties have been estimated to occur in approximately 10% to 15% of school age children (Benton & Pearl, 1978; Harris & Sipay, 1990; Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992) and tend to be accompanied by specific deficits in cognitive abilities related to reading and other literacy skills. This symptom pattern is often called 'dyslexia', or, alternatively, 'specific reading disability', and the terms are often used interchangeably. We will adopt this convention to refer to children whose reading difficulties occur at the level of basic reading subskills and are not caused by the exclusionary factors just mentioned (Lyon, 1995; Lyon, Fletcher, & Barnes, 2002; Shaywitz, 1996).

The primary purpose of the present paper is to provide a selective and relatively up-to-date review of research, conducted over the past four decades, emanating both from historically influential and contemporary conceptualizations of the basic cause(s) of developmental dyslexia. We focus on conceptualizations specifying cognitive and biological deficits that may underlie this disorder and special emphasis is placed on contemporary and somewhat controversial theories of dyslexia that have gained some degree of prominence in more recent years. We briefly describe and critically analyze each of the theories instantiated and summarize some of the more important findings amassed by researchers who have evaluated its correlates and extensions, in the interest of distinguishing between probable and improbable causes of early reading difficulties in children presumed to be dyslexic.

Our discussion, throughout, is based on the assumption that causal relationships between given skills and abilities hypothesized to underlie the ability to learn to read, on the one hand (e.g., visual perception, verbal memory), and reading ability on the other (e.g., word identification, reading comprehension), can only be confidently inferred in instances where results are generated by experimental or quasi-experimental research designs.

Published by Blackwell Publishing, 9600 Garsington Road, Oxford OX4 2DQ, UK and 350 Main Street, Malden, MA 02148, USA

Such research must document improved performance on measures of reading ability as a function of improved performance on measures of the skills or abilities hypothesized to underlie reading ability (Shadish, Cook, & Campbell, 2002). Absent such documentation, no causal relationships can be inferred, even in instances where poor and normal readers are shown to be reliably different on given measures, at least not until such correlated relationships are substantiated through controlled experimentation. It follows that research demonstrating that poor and normal readers do not differ reliably on measures of skills and abilities hypothesized to underlie reading ability, in most cases, can be taken as evidence that such variables are not causally related to reading ability.[1] It will become apparent that there are fewer causal relationships that have been documented in the reading disability literature that is reviewed herein than there are correlated relationships that are not fully understood.

A secondary purpose of our review is to present research evidence documenting the importance of distinguishing between early reading difficulties that may be caused primarily by cognitive and biological deficits and early reading difficulties that may be caused primarily by experiential and instructional deficits. Results from recent intervention studies suggest that explanations of reading difficulties in most children must incorporate experiential and instructional deficits as possible causes of such difficulties, rather than focus exclusively on the types of cognitive and biological deficits that have predominated theory and research in this area of inquiry throughout the previous century. Selected findings from some of this research are discussed, placing special emphasis on their implications for practitioners.

However, to set the stage for our review, we first discuss the components of literacy in terms of the knowledge, skills, and abilities presumed to underlie reading ability. The intent here is to embed our discussion of the different theories of dyslexia within the context of a process model that specifies possible sources of difficulties in learning to read. We then discuss the manifest causes of such difficulties, as reflected in deficiencies in basic reading subskills such as word identification, phonological awareness, and phonological decoding. However, because the causal relationships between deficits in these reading subskills and early reading difficulties have been reasonably well established in previous research, we do not discuss this research in great detail. Thus, the main body of the text is devoted to discussion of the various cognitive deficit theories of dyslexia that have generated a great deal of empirical research in the last four decades and we highlight converging evidence from cross-sectional, longitudinal, and cross-linguistic studies supporting the view that linguistic coding deficits are the most probable causes of reading difficulties in dyslexic children. This view is given additional support from results of neurobiological, genetic, family risk, and life-span development studies, which are discussed in subsequent sections of the review. We then summarize the most important findings from a first grade reading intervention study that was specifically designed to evaluate the utility of using response to remedial intervention as a primary vehicle for distinguishing between cognitive/biological and experiential/instructional deficits as basic causes of early reading difficulties. We close with a brief discussion of the implications of the results of this and other studies reviewed in this paper for practitioners working with reading impaired children. Note, however, that our review is not exhaustive and we do not presume to discuss all areas of research on dyslexia. For example, there is an extensive research literature on subtypes of dyslexia that we did not address, largely because this research has not been fruitful in enhancing our understanding of dyslexia subtypes at the cognitive level, with few studies finding evidence of relations between subtypes and biological or intervention findings (Lyon et al., 2002).

Components of reading ability

Normal reading ability assumes adequate language comprehension and fluent word identification. Written words are encoded (symbolized) representations of spoken words, and spoken words are encoded representations of environmental experiences and entities. Thus, the ability to learn to read depends on the acquisition of a variety of different types of knowledge and skills, which, themselves, depend on normal development of reading-related linguistic and non-linguistic cognitive abilities. Figure 1 presents a model depicting the cognitive processes and different types of knowledge involved in learning to read. The model depicts processes whereby world knowledge and domain specific knowledge stored in permanent memory are transformed into units of spoken and written language. These include linguistic processes and knowledge that allow one to acquire a spoken word vocabulary and language skills in general, as well as visual, linguistic, and metalinguistic processes and knowledge that allow

[1] Exceptions to this generalization would, of course, include instances where deficiencies in a basic skill or ability might be causally related to difficulties in learning to read at beginning stages of reading development, but not at later stages, because of experiences that corrected such deficiencies (e.g., reading instruction) and, thereby, masked the causal relationship. For example, deficiencies in letter-name knowledge has been shown to reliably distinguish between poor and normal readers early in their reading development, but not later (Vellutino et al., 1996), despite the distinct possibility that deficiencies in letter-name knowledge might well be a factor contributing to difficulties in learning to read.

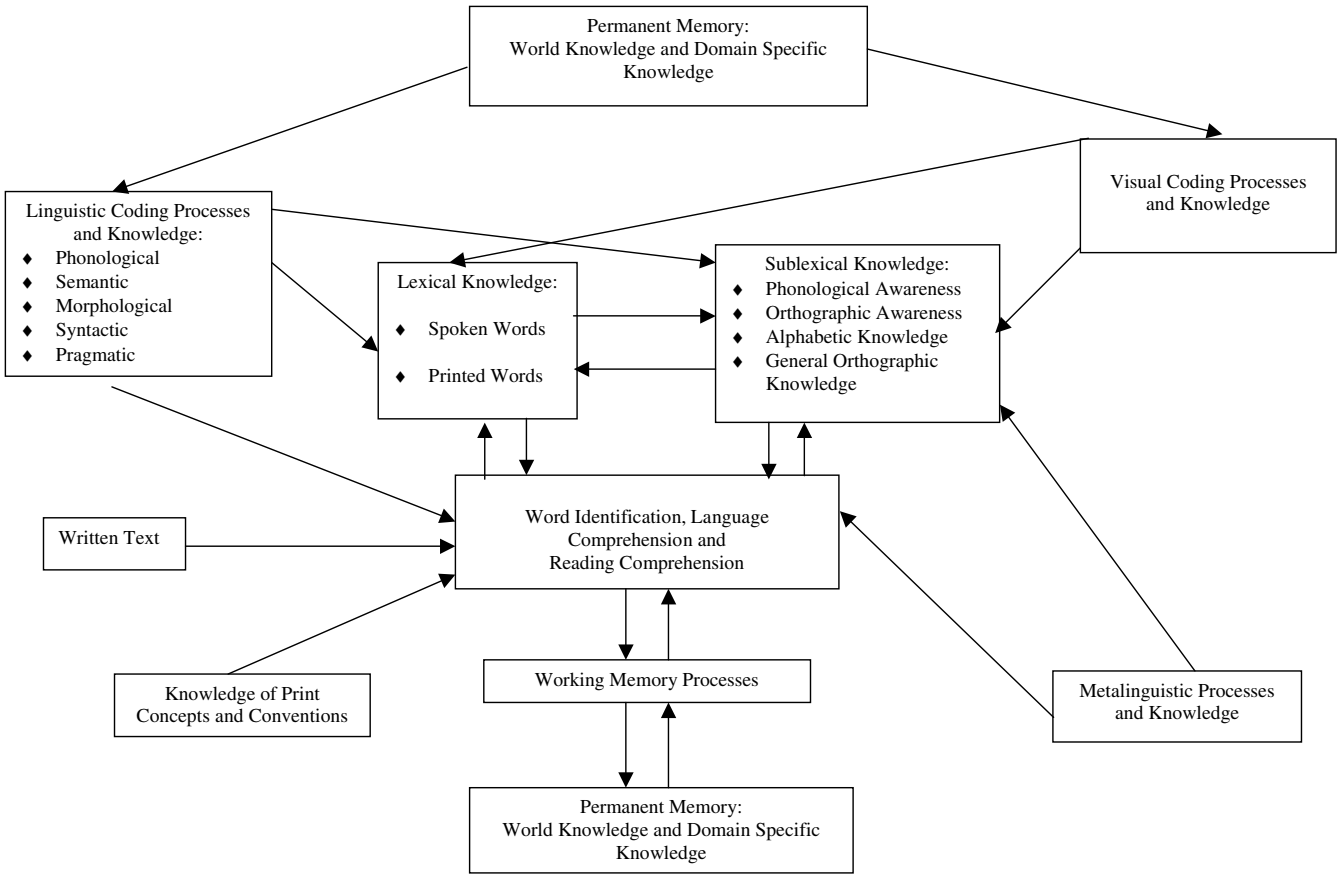

Figure 1 Cognitive processes and different types of knowledge entailed in learning to read

one to acquire knowledge and skills that are influential determinants of the ability to learn to read. Visual coding processes, broadly defined, refer to sensory and higher-level visualization processes that facilitate storage of representations defining the visual attributes of environmental stimuli, including the graphic symbols used to represent written words. Linguistic coding processes refer to processes that facilitate language acquisition and the use of language for coding, storing and retrieving information. They include phonological coding – the ability to use speech codes to represent information in the form of words and word parts; semantic and morphological coding – the ability to store information about the meanings of concepts represented by words and word parts (e.g., ing, ed); syntactic coding – the ability to store word order rules that set constraints on how words are organized in sentences; and pragmatic coding – the ability to store information about conventions governing the use of language as a medium of communication (e.g., modifications in meaning signaled by changes in volume, pitch, and intensity in spoken language or punctuation marks in written language, use of understandable language on the part of the speaker or writer, etc.).

Linguistic and visual coding processes together facilitate the establishment of firm associations between the spoken and written counterparts of printed words, in the interest of helping the child acquire a sight word vocabulary – that is, a corpus of printed words the child is able to identify (name) on sight as lexical units (unanalyzed meaning-bearing units). This associative learning process, itself, depends on the child's understanding of print concepts and conventions: that written words represent words in spoken language, that they are comprised of letters, that they are processed from left to right (in written English), that they are demarcated by spaces, and so forth. However, because of the heavy load on visual memory imposed by the high degree of similarity characteristic of words derived from an alphabet (pot/top; was/saw), sight word learning also depends on the child's ability to acquire understanding and functional use of the alphabetic principle. Understanding the alphabetic principle is important for acquiring proficiency in phonological (letter-sound) decoding, which is the primary vehicle beginning readers use for reducing the load on visual memory imposed by an alphabetic writing system.

Such proficiency, in turn, will require that the child actively engage in the type of metalinguistic analysis (analysis of language structures) that will facilitate acquisition of sublexical (letter-level) knowledge, in particular, phonological and orthographic awareness, alphabetic knowledge, and general orthographic knowledge. Phonological awareness refers to

conceptual understanding and explicit awareness that spoken words consist of individual speech sounds (phonemes) and combinations of speech sounds (syllables, onset-rime units). Such knowledge is believed to be important for learning that letters carry sound values and for learning to map alphabetic symbols to sounds. Orthographic awareness refers to the child's sensitivity to constraints on how the letters in written words are organized (vid is legal, xqr is illegal). Phonological and orthographic awareness are reciprocally related cognitions that ultimately work in concert to help the child acquire and make functional use of general orthographic knowledge, in the form of sensitivity to the regularities and redundancies characteristic of an alphabetic writing system (e.g., 'at' in 'cat', 'fat' and 'rat'; 'ing' in 'walking' and 'running'). These processes are complemented by another type of metalinguistic knowledge: syntactic awareness (not shown in Figure 1). Syntactic awareness refers to the child's sensitivity to grammatical form in terms of errors that violate conventional usage in spoken and written language ('Mom brung the cat to the vet'). It facilitates detection of reading errors, and, thereby, conjoint use of context-based strategies and phonological decoding strategies for word identification and comprehension during text processing. Together, these three types of knowledge help the child acquire and consolidate both alphabetic knowledge and the more general orthographic knowledge that ultimately leads to mastery of the alphabetic code and increasing accuracy and fluency in word identification and spelling.

Finally, the model in Figure 1 depicts both the permanent memory and the working memory systems and processes involved in learning to read. The double-directional arrows represent the reciprocal and interactive relationships between the different coding and memory systems involved in (a) establishing firm connective bonds between lexical and sublexical components of spoken and printed words, and (b) encoding, storing, and retrieving the different types of information entailed in learning to read.

It should be clear that, given adequate exposure to print, adequate literacy instruction, and adequate motivation, the child's ability to acquire the skills and subskills necessary to learn to read is dependent on normal development and functioning of these different coding and memory systems and processes. Thus, difficulties in learning to read could come about, either from specific deficiencies in reading-related cognitive abilities resulting from abnormal development and consequent dysfunction in one or more of these coding and memory systems and processes or from a less than adequate mix of reading-related cognitive abilities (the child's phenotype) resulting from the interaction of the child's particular genetic endowment (the child's genotype) and the particular environmental and instructional experiences to which he or she has been exposed. Most theories of dyslexia are of the former variety and can be classified as qualitative difference, 'all or none type' theories (you either have or do not have dyslexia) because they are based on the assumption that reading difficulties are ultimately caused by structural or functional anomalies in the brain. However, some scholars have given serious consideration to quantitative difference, continuous abilities type theories of dyslexia (e.g., Olson & Gayan, 2001; Pennington & Lefly, 2001; Shaywitz et al., 1992; Snowling, Gallagher, & Frith, 2003; Stanovich, 1988; Vellutino et al., 1996). Such theories suggest that reading ability is a multifactorial trait that is determined by the action of a particular assortment of genes (called quantitative trait loci) that are important for acquiring reading skills and subskills, interacting with environmental factors to produce quantitative variation in cognitive abilities underlying reading ability and reading achievement. Continuous abilities type theories of dyslexia are based on the assumption that reading disability occurs along a continuum defining levels of reading ability and that there is a gradation of risk for becoming dyslexic, depending on the particular assortment of reading-related cognitive abilities with which the child is endowed and the degree to which that child's home and school environments capitalize and build on his or her cognitive strengths and compensate for his or her cognitive weaknesses. Because such theories are not well developed, we only touch upon them in this review. In the next section, we briefly discuss manifest causes of specific reading disability, in terms of deficiencies in reading subskills that have been found to be causally related to early reading difficulties.

Manifest causes of specific reading disability: deficiencies in reading subskills

Reading may be defined as the process of extracting and constructing meaning from written text for some purpose. Skilled reading entails on-line comprehension of meaning from running text. It is a complex process that depends on adequate development of two component processes: word identification and language comprehension. Word identification is a lexical retrieval process (see Figure 1) that involves visual recognition of a uniquely ordered array of letters as a familiar word and implicit (or explicit) retrieval of the name and meaning of that word from memory. Language comprehension involves integration of the meanings of spoken or written words in ways that facilitate understanding and integration of sentences in spoken or written text in the interest of understanding the broader concepts and ideas represented by those sentences. Thus, in order to comprehend what one reads, one must be able to identify the words contained in running text with enough accuracy and fluency to allow computation of the meanings embodied in the text within the

limits of working memory. One must also have adequate language comprehension, and, ultimately, adequate world knowledge and adequate domain-specific knowledge. However, research in the study of reading disability has made it clear that early reading difficulties in the population of children defined herein, that is, children who might qualify for a diagnosis of dyslexia in accord with the criteria outlined earlier, are manifested primarily in inadequate facility in printed word identification as well as inadequate facility in related skills such as spelling and phonological (letter-sound) decoding. Such difficulties may or may not be accompanied by significant deficits in language comprehension, but they are not necessarily accompanied by such deficits. Thus, specific reading disability (dyslexia) in otherwise normal children has been and continues to be defined as a basic deficit in learning to decode print. There are several pieces of evidence to support this definition.

First, we know from studies evaluating the relationship between printed word identification and written and oral language comprehension processes that reading comprehension is impaired in an individual who has inadequate facility in word identification, in terms of both accuracy and fluency, even if that individual has adequate language comprehension skills (Gough & Tunmer, 1986; Perfetti, 1985; Snowling, 2000a; Stanovich, 1991; Vellutino, 1979, 1987; Vellutino, Scanlon, & Tanzman, 1994; Vellutino, Scanlon, & Chen, 1995a; Vellutino et al., 1996). These same studies demonstrate the converse, that is, that children who have inadequate facility in reading comprehension are typically found to have inadequate facility in word identification and related word-level skills such as spelling and phonological decoding. Thus, it would seem that the most basic and most ubiquitous cause of difficulties in learning to read is inadequate facility in word identification, which, itself, appears to be caused by basic difficulty in learning to decode print.

This possibility is given added credibility by results from a second line of research, that is, regression studies evaluating skills and abilities underlying reading ability (Catts, Hogan, & Fey, 2003; Curtis, 1980; Foorman, Francis, Shaywitz, Shaywitz, & Fletcher, 1997; Hoover & Gough, 1990; Vellutino, Scanlon, Small, & Tanzman, 1991; Vellutino et al., 1994). These studies have shown that there is a developmental asymmetry in the acquisition of skill in comprehending written text such that facility in word identification carries much greater weight as a determinant of reading comprehension in children at the early stages of reading development than in children at later stages, whereas language comprehension processes carry much greater weight as determinants of reading comprehension in children at later stages of reading development than in children at early stages of reading development. Such research has also shown that tests evaluating word identification skills were much better predictors of performance on reading comprehension tests than were tests evaluating language comprehension skills in beginning and less skilled readers, whereas the opposite pattern was evident in more skilled readers. Moreover, tests evaluating sublexical knowledge, such as phonological awareness, application of letter-sound correspondence rules (i.e., phonological decoding), and spelling ability, along with related phonological skills such as name retrieval, and verbal memory, were much better predictors of facility in word identification than were tests evaluating vocabulary knowledge, general knowledge, and syntactic processing, which were found to be better predictors of facility in language comprehension (Vellutino et al., 1991, 1994). This pattern of results suggests that adequate facility in word identification and other word-level skills is a necessary (though not sufficient) condition for reading for meaning and is quite in keeping with the idea that dyslexia is accurately defined, at the behavioral level, as a basic impairment in print decoding.

The regression studies evaluating the components of reading ability cited above also suggest that adequate facility in word identification, itself, depends heavily on the beginning reader's ability to acquire facility in alphabetic coding. Additional support for this possibility comes from a large number of studies providing independent and convergent evidence that children who have difficulty in mapping alphabetic symbols to sound also have difficulty learning to read and spell (e.g., Fletcher et al., 1994; Liberman & Shankweiler, 1979, 1991; Olson, Forsberg, Wise, & Rack, 1994; Share & Stanovich, 1995; Shankweiler et al., 1979; Siegel & Ryan, 1984; Snowling, 1980; Stanovich & Siegel, 1994; Tunmer, 1989; Vellutino, 1979, 1987, 1991; Vellutino & Scanlon, 1987a, b; Vellutino et al., l995a; Vellutino et al., 1991, 1994, 1996; Wagner & Torgesen, 1987). Moreover, such difficulties have been found to continue well into adulthood (Bruck, 1990, 1992, 1993; Satz, Buka, Lipsett, & Seidman, 1998; Spreen, 1989).

Yet, there is also a great deal of evidence that acquisition of facility in alphabetic mapping depends, in part, on the acquisition of phonological awareness – which we defined earlier as conceptual grasp and explicit awareness that spoken words are comprised of individual speech sounds (phonemes) and combinations of speech sounds (syllables, onset rimes). Compared with normally developing readers, poor readers commonly manifest difficulty acquiring phonological awareness and phonological analysis skills during their childhood years and continue to be deficient in phonological analysis during their adult years. Bruck, 1992; Liberman, Shankweiler, Fischer, & Carter, 1974; Liberman & Shankweiler, 1979, 1991; Snowling, 2000a; Wagner & Torgesen, 1987). More direct evidence for the possibility that deficiencies in phonological awareness and alphabetic

mapping may be causally related to reading difficulties comes from naturalistic studies, controlled laboratory studies, and intervention studies in which it was found that training that helped children acquire these skills had a beneficial effect on word identification, spelling, and reading ability in general (Adams, 1990; Blachman, 1994, 2000; Bradley & Bryant, 1983; Foorman, Francis, Novy, & Liberman, 1991; Foorman, Francis, Fletcher, Schatschneider, & Mehta, 1998; Hatcher, Hulme & Ellis, 1994; Lundberg, Frost, & Petersen, 1988; Olson, Wise, & Ring, 1999; Scanlon & Vellutino, 1996; Scanlon, Vellutino, Small, & Fanuele, 2000; Torgesen, Rose, Lindamood, Conway, & Garvan, 1999; Vellutino & Scanlon, 1987a; Vellutino et al., 1996; Williams, 1980). Thus, although there is abundant evidence that difficulty in learning to identify printed words is the manifest cause of reading difficulties in beginning readers, there is also abundant evidence that this problem, itself, is causally related to significant difficulties acquiring phonological analysis skills and mastering the alphabetic code, regardless of more distinct causes (intrinsic vs. environmental and instructional). A caveat to the latter generalization concerns the role of other language-based skills (e.g., semantic and syntactic skills) in learning to read. We discuss these possibilities in greater detail below.

In our analysis of the components of reading ability, consolidation of alphabetic knowledge was said to be dependent, not only on phonological awareness and the ability to map alphabetic symbols to sounds, but also on orthographic awareness, which we defined as the child's sensitivity to constraints on how the letters in written words are organized. Phonological awareness and orthographic awareness are reciprocally related cognitions that facilitate alphabetic mapping and help the child acquire the more general orthographic knowledge (e.g., 'at' in 'cat', 'fat' and 'rat'), that helps to make the writing system more manageable and ultimately leads to mastery of the alphabetic code (Ehri, 1999). It follows that children who have difficulty in acquiring phonological awareness and learning to map alphabetic symbols to sound will also have difficulty acquiring orthographic awareness and general orthographic knowledge. There is abundant evidence that the child who has limited phonological awareness and limited alphabetic mapping skills also has limited orthographic awareness and limited orthographic knowledge (cf. Siegel, Share, & Geva, 1995). These limitations have been observed in both dyslexic children and adults (e.g., Bruck, 1990, 1992; Manis, Custodio, & Szeszulski, 1993; Snowling, 2000a; Olson et al., 1994; Vellutino et al., 1994, 1995a).

Finally, given the importance of acquiring knowledge of print concepts and conventions in learning to read along with the importance of acquiring pragmatic knowledge as an important component of language and reading comprehension, it seems reasonable to inquire whether deficiencies in such knowledge have been found to be causally related to specific reading disability. Although there is considerable evidence that limited knowledge of print concepts and conventions and limited pragmatic knowledge have often been observed in children who also experience early reading difficulties and can certainly contribute to early reading and language difficulties (e.g., Adams, 1990; Snow, Burns, & Griffin, 1998), limitations in such knowledge are probably not basic causes of specific reading disability, in the biological sense, and are, in most cases, caused by experiential and instructional deficits rather than by biologically based cognitive deficits. Support for this inference comes from studies finding that many children have extreme difficulty learning to read, despite having entered school with age-appropriate pragmatic skills and a reasonably firm grasp of print concepts and conventions, having come, in most such cases, from enriched home backgrounds (e.g., Vellutino et al., 1996).

Underlying causes: cognitive deficit theories of dyslexia

Deficits in general learning abilities

The study of basic cognitive deficits as underlying causes of specific reading disability has a long history (Fletcher, Foorman, Shaywitz, & Shaywitz, 1999; Lyon et al., 2002; Snowling, 2000a; Vellutino, 1979, 1987; Vellutino & Scanlon, 1982). Etiological theories that have been proffered over the years are legion, and most have been falsified both empirically and logically. Dyslexia has most often been attributed to deficiencies in visual, linguistic, and low-level sensory functions, and we exemplify such theories below. However, dyslexia has also been attributed to deficiencies in general learning abilities that are involved in all learning enterprises and not just learning to read. For example, specific reading disability has been variously attributed to deficiencies in selective attention (Douglas, 1972), associative learning (Brewer, 1967; Gascon & Goodglass, 1970), cross-modal transfer (Birch, 1962), serial-order processing (Bakker, 1972), and both pattern analysis and rule learning (Morrison & Manis, 1982). Such theories can be questioned on logical grounds alone. As stated elsewhere, 'dysfunction in one or another of these rather basic and general learning abilities would seem to be ruled out as significant causes of the disorder in a child who has at least average intelligence and who does not have general learning difficulties, given that all of these cognitive abilities are entailed on virtually all tests of intelligence and are most certainly entailed in all academic learning' (Vellutino et al., 1996, p. 602). More important, however, is the fact that each of these hypotheses has also been discredited by empirical research. This research has been

summarized elsewhere and will not be reviewed here (Vellutino, 1979, 1987; Vellutino & Scanlon, 1982; see also Katz, Shankweiler, & Liberman, 1981; Katz, Healy, & Shankweiler, 1983). Most of the studies reporting differences between poor and normal readers on measures of these general learning abilities did not control for reader group differences in verbal coding ability and/or working memory processes that might be affected by verbal coding deficits. In subsequent studies that did implement such controls, group differences on measures of these abilities were generally eliminated.

To cite one example, Birch (1962) hypothesized that reading impaired children may be encumbered by a developmental lag in the establishment of cross-modal transfer. He intuited that this lag would impair their ability to represent the same information in two sensory-based systems, as exemplified in learning to read. Initial support for this theory was provided by Birch and Belmont (1964), who observed that poor readers performed below the level of normal readers in matching auditorily presented rhythmic patterns with visual representations of those patterns. Because this task confounds cross-modal transfer with working memory and verbal coding ability, Vellutino and his associates (Vellutino, 1979, 1987; Vellutino & Scanlon, 1982) conducted a series of studies that compared poor and normal readers on both intramodal (visual–visual; auditory–auditory) and intermodal (visual–auditory) non-verbal learning tasks that minimized the influence of verbal coding ability. They also compared these groups on visual–verbal learning tasks, and found that the poor readers performed below the level of the normal readers only on the visual–verbal learning tasks. These findings are at variance with Birch's cross-modal explanation of reading disability. They are also at variance with etiological theories which suggest that deficits in association learning or attention are root causes of specific reading disability, given that all of the tasks used in these studies involved paired associates learning and required the utmost in attention, concentration, and working memory. The data are more in keeping with verbal deficit explanations of the disorder.

Finally, Birch's cross-modal theory of reading disability was derived from the more basic assumption that cross-modal transfer is a developmental phenomenon that is not established until early childhood (Birch & Lefford, 1963). However, this assumption has since been obviated by infant research which shows that perception of equivalences across modalities is present either at birth or shortly after birth (e.g., Gibson, 1969; Bryant, 1974; Meltzoff & Kuhl, 1994). Thus, the theory is no longer viable.

Visual deficits

Visual perceptual and visual memory deficits. Theories implicating deficiencies in the visual system have been the most ubiquitous and most influential theories of dyslexia, from before the turn of the century (Morgan, 1896; Hinshelwood, 1917) up through the 1970s and 1980s, when linguistic deficit explanations of the disorder began to compete with visual deficit explanations (Lyon et al., 2002; Snowling, 2000a; Vellutino, 1979, 1987). However, despite their popularity, the most prominent visual deficit theories in the early reading disability literature had little empirical support, and confounded the visual and verbal components of reading and spelling.

The demise of these theories was initiated over two decades ago through a series of related studies that systematically evaluated traditional and widely accepted etiological conceptualizations such as Orton's (1925) optical reversibility theory of dyslexia (i.e., perceiving letters and words as reversed forms), Hermann's (1959) spatial confusion theory (i.e., inherent spatial disorientation), and a variety of other theories that implicated deficits in visual processes such as visualization, visual sequencing, and visual memory as basic causes of reading difficulties. The studies challenging these theories made use of a wide variety of visual processing paradigms that were carefully designed to control for or minimize the influence of verbal coding (e.g., visual discrimination, spatial orientation, visual memory, and visual learning paradigms), and they generally replicated given findings with independent samples of poor and normal readers.

To be brief, in studies conducted comparing poor and normal readers across a broad age range (most often grades 2 through 8), few significant differences between these groups were found on measures of visual processing ability when the influence of verbal coding was controlled. For example, in experimental studies evaluating such processes (Vellutino, 1979, 1987; Vellutino & Scanlon, 1982), it was found that memory for visually presented letters and words that were visually similar (such as 'b', 'd', 'was', and 'saw') was as good in poor readers as it was in normal readers when the task required a written response rather than a naming response, which did differentiate these two groups. At the same time, there were no statistically significant differences between poor and normal readers on measures evaluating visual recognition and visual recall of letters and words from an alphabetic orthography with which the two groups were unfamiliar, specifically, written Hebrew. Moreover, these groups did not differ on measures evaluating orientation and left to right processing of the letters in Hebrew words, compared with children who were learning to read and write Hebrew, whose accuracy was greater and whose (right to left) processing strategies were different from the children who were unfamiliar with Hebrew. Research conducted later provided additional confirmation that poor and normal readers have comparable visual abilities when task requirements for verbal mediation were minimized (Fletcher et al., 1999). Thus, it

seems reasonable to conclude that visual deficits of the types that had been touted in the early literature are no more prevalent in poor readers than they are in normal readers. It may also be concluded that certain hypothesized and highly popularized deficits such as Orton's optical reversibility and Hermann's spatial confusion are pseudo-problems that have no psychological reality.

The final source of evidence is derived from regression studies evaluating the components of reading ability (e.g., Vellutino et al., 1991, 1994) discussed earlier. It was found that visual abilities were relatively poor predictors of word identification, spelling, pseudoword decoding, and reading comprehension. This was true at all age and grade levels evaluated in these studies (grades 2 through 7). These results, together with the results just discussed, provide strong evidence that reading is primarily a linguistic skill (see Frost, 1998 for a recent review).

Low-level visual deficits. Specific reading disability has been attributed to visual tracking problems associated with oculomotor deficiencies (Getman, 1985), in addition to visual masking effects associated with a hypothesized deficit in the 'transient visual system' (Breitmeyer, 1989; Lovegrove, Martin, & Slaghuis, 1986; Stein, 2001). Reading disability has also been associated with abnormalities in perception of visual motion (Eden et al., 1996). At the same time, transient system and motion perception deficits in disabled readers have both been linked to functional anomalies in the magnocellular visual subsystem.

The visual tracking theory of reading disability has been discredited by well-controlled eye movement studies finding no differences between poor and normal readers on visual tracking of non-verbal stimuli (Olson, Kleigl, & Davidson, 1983; Stanley, Smith, & Howell, 1983). However, the transient system theory has some empirical support (Breitmeyer, 1989; Badcock & Lovegrove, 1981; Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Lovegrove et al., 1986; Lovegrove, Garzia, & Nicholson, 1990; Martin & Lovegrove, 1984), as does the motion perception theory (Eden et al., 1996). Thus, both warrant further comment.

First, note that the visual system is comprised of two parallel systems, the magnocellular system and the parvocellular system, both residing in the layers of the lateral geniculate nucleus of the visual cortex. The magnocellular system – often called the transient system – consists of large neurons that have high conduction velocity and demonstrate a high degree of sensitivity to movement and rapid changes in the visual field. In contrast, the parvocellullar system consists of 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 magnocellular system is (presumably) responsible for suppressing the activity of the parvocellular system when the eyes are in motion, thereby inhibiting the visual trace that normally persists for a short duration (approximately 250 milliseconds) after a visual stimulus has been terminated. It has been suggested that dyslexics suffer from a deficit in the inhibitory function of the transient system, producing a visual trace of abnormal longevity that creates masking effects along with visual acuity problems when such children are reading connected text. Thus, Lovegrove and his associates have shown that poor and normal readers process high and low spatial frequency grids differently. They also have different contrast sensitivity functions, such that the poor readers require greater luminosity than the normal readers for distinguishing low frequency grids (Badcock & Lovegrove, 1981; Lovegrove et al., 1986, 1990; Martin & Lovegrove, 1984). That dyslexics are subject to trace persistence has been inferred from such findings.

However, as pointed out by Hulme (1988), the trace persistence theory of reading disability predicts that dyslexics should be impaired only when they are reading connected text and not when they encounter printed words one at a time under foveal vision conditions. Yet, we know that poor readers find it as difficult to identify printed words one at a time under foveal vision conditions as to identify them when they are reading connected text. In addition, a significant number of normally achieving readers were also found to have transient system deficits in the studies conducted by Lovegrove and his associates. Moreover, there is no evidence that dyslexics experience visual acuity and visual masking problems under normal reading conditions. Furthermore, dyslexic children evaluated typically had phonological deficits of the types that have been shown to be causally related to reading difficulties (e.g., deficiencies in letter-sound decoding and phonological awareness). At the same time, Eden, Stein, Wood, and Wood (1995) found that while 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 measures of phonological skills.

Finally, given that some normal readers have been found to manifest abnormalities consistent with transient system deficits, and absent any clinical evidence that dyslexics typically experience the types of visual perceptual anomalies that are said to be a consequence of transient system deficits (e.g., visual masking caused by trace persistence), it would appear that transient system deficits have not been shown to be causally related to reading difficulties.

All things considered, we doubt that visual trace persistence associated with transient system deficits is a significant cause of specific reading disability. Yet, it might be a correlate of the disorder, and perhaps even a biological marker. This possibility is given some

support by anatomical and electrophysiological studies demonstrating structural and functional anomalies in the magnocellular pathways of a small number of those with dyslexia studied (Lehmkuhle et al., 1993; Livingstone, Rosen, Drislane, & Galaburda, 1991). However, a recent review of the literature by Skottun and Parke (1999) presented evidence from several studies of saccadic suppression that strongly suggests that it is the magnocellular system that is suppressed during saccadic movements of the eyes, not the parvocellular system. Such findings are contrary to the major premise of the transient system deficit theory of dyslexia, so there is considerable reason to question the viability of the theory.

As a correlate, dysfunction in the magnocellular systems in dyslexia has also been implicated by recent studies demonstrating that perception of visual motion is deficient in this population (Eden et al., 1996; Eden & Zeffiro, 1998). It has been reasonably well established through study of nonhuman primates and patients with vascular lesions that a component of the visual system located at the temporal–occipital–parietal junction (the MT/V5 complex) is sensitive to motion and this area of the brain is believed to be dominated by input from the magnocellular neurons. Thus, on the strength of the possibility that dysfunction in the magnocellular system might be the result of a partially compensated developmental lesion, Eden et al. (1996) used functional magnetic resonance imaging (fMRI) to evaluate visual motion processing in dyslexic and normal adults and found that for dyslexics, presentation of moving stimuli failed to produce the same task-related pattern of activation in the MT/V5 complex as that observed in the normal controls. Similar results have been obtained in contrasts of 9- and 10-year-old dyslexic and normal readers (Cornelissen, Hansen, Hutton, Evangeliou, & Stein, 1997; Raymond & Sorensen, 1998). The combined results lend additional credence to the idea that the magnocellular pathways may be deficient in some dyslexic individuals.

In their interesting review of this literature, Eden and Zeffiro (1998) stop short of inferring a causal relationship between magnocellular dysfunction and specific reading disability and put forward an intriguing hypothesis that potentially explains both linguistic and visual (magnocellular) deficits observed in dyslexics. This hypothesis could also account for the logical disconnect between the consistent observation of low-level visual processing deficits in these children and the absence of associated clinical symptoms that might be causally related to their reading difficulties. Specifically, Eden and Zeffiro (1998) underscore the complexity of the symptom pattern that has been observed in dyslexics and point out that low-level visual deficits that have been observed in some of these individuals have typically been accompanied by deficits in reading-related language and language-based skills, such as phonological awareness and verbal working memory, and sometimes, by low-level auditory deficits as well (see discussion below). And, although they acknowledge that these low-level sensory deficits have not been shown to be causally related to difficulties in learning to read, they make special note of the fact that sensory deficits and the reading-related language deficits that sometimes co-occur in dyslexic children and adults have not been shown to be causally related to each other. To explain this pattern of results, Eden and Zeffiro (1998) suggest that 'these perceptual and cognitive abnormalities arise from dysfunction of a neural system common to both' (p. 279).

In support of this conjecture, the investigators cite research conducted by Rumsey et al. (1997), comparing adult dyslexic and normal reading controls on rhyme detection/judgment and pseudoword reading tasks, in which it was found that the dyslexics showed 'significantly less task-related signal increase in temporoparietal areas bilaterally, consistent with a role for the angular gyrus (and nearby temporal and parietal areas) in reading' (p. 280). Also consistent with this possibility are results they cite from a study conducted by Horwitz, Rumsey, and Donahue (1998) demonstrating that measures of activity in temporal and parietal areas thought to be involved in reading were found to be correlated when normal readers were engaged in a pseudoword reading task and uncorrelated when dyslexic subjects performed the same task (see also discussion below). Thus, in combining experimental findings from behavioral, electrophysiological, and functional neuroimaging studies that provide documentation of phonological processing deficits in dyslexia with experimental findings from similar studies demonstrating magnocellular system deficits in this group, and coupling these findings with the demonstrated involvement of temporoparietal areas in reading, phonological processing, and magnocellular system functioning, Eden and Zeffiro suggest that 'the cortical regions surrounding the temporoparietal junction emerge as possible candidates for the principal loci of cerebral dysfunction in dyslexia' (p. 281). In other words, dyslexic persons may have structural and functional anomalies in adjacent regions of the brain (respectively) supporting linguistic and visual processes of the types found to be deficient in this group. But, whereas the linguistic deficits have been demonstrated to be causally related to reading disability, the visual deficits have not been demonstrated to be causally related to reading disability, though they may serve as biological markers that aid differential diagnosis. This is a highly plausible hypothesis that merits further consideration.

Language and language-based deficits

Semantic and syntactic deficits. If reading were primarily a linguistic skill, as many scholars as-

sume, then it would seem that reading disability could be caused by deficiencies in the semantic, syntactic, or phonological components of language. For example, some have theorized that vocabulary deficits may be a basic cause of difficulties in learning to read in some impaired readers (e.g., Dickinson & Tabors, 2001; Snow & Tabors, 1993; Vellutino, 1979, 1987; Vellutino & Scanlon, 1982). It seems reasonable to infer, on logical grounds, that a child will have less difficulty in learning to read words that are in his or her speaking vocabulary than in learning to read words that are not in his or her speaking vocabulary. There is some evidence to support this possibility. It has been demonstrated, in simulated reading tasks, that normal as well as poor readers had more difficulty establishing connective bonds between low meaning words and the characters representing those words than between high meaning words and their representative characters. In one such study (Vellutino, Scanlon, & Spearing, 1995b), low meaning words were words the children had heard before, but did not produce many semantic associates on a word association task where those words were used to stimulate as many associations as came to mind. High meaning words were not only familiar to the children, but produced a large number of semantic associations on the word association task and were more likely than the low meaning words to be in the children's speaking vocabularies. It was found that the high meaning words were easier to learn to 'read' (on the simulated reading task) than were the low meaning words. Similar results were obtained in experimental simulations of beginning reading that compared poor and normal readers' ability to learn to 'read' nonwords with their ability to learn to 'read' the same nonwords after pairing these stimuli with novel cartoon characters that imbued them with meaning (Vellutino & Scanlon, 1987a). These findings were apparent, regardless of whether the characters were ideographic or alphabetic in nature.

Additional support for the possibility that deficiencies in semantic knowledge may be causally related to difficulties in learning to read comes from studies finding that vocabulary knowledge in pre-first grade children is a good predictor of early and later reading achievement (Dickinson & Tabors, 2001; Scarborough, 1990, 1991; Snow, Barns, Chandler, Goodman, & Hemphill, 1991; Snowling et al., 2003). Moreover, deficient vocabulary knowledge has been found to be a significant cause of reading difficulties in second language learners having limited proficiency in spoken English (Tabors & Snow, 2001). Thus, it follows that a child who has a limited vocabulary could have difficulties in acquiring fluency in printed word identification, even if he or she has adequate phonological decoding skills.

Vocabulary knowledge has also been implicated in the acquisition of reading-related phonological skills. To be brief, Metsala and Walley (Metsala, 1999; Metsala & Walley, 1998; Walley, 1993; see also Goswami, 2001) have proposed that phonological representations become increasingly less global and more highly specified with developmental changes in vocabulary knowledge, especially in the case of words having similar phonological properties (e.g., phonological 'neighbors' such as 'sail', 'mail', 'fail', 'jail', 'bail' etc.). This is because encoding and retrieval of such words for functional use requires more in the way of (implicit) segmental analysis than words having fewer phonological neighbors. These investigators have provided some evidence to support this hypothesis. Thus, if it proves to be valid, then it would be expected that deficiencies in vocabulary knowledge would be accompanied by poorly specified phonological representations, which, by some accounts, would impair reading development (see discussion below).

Finally, given the demonstrated utility of linguistic context in facilitating and monitoring word identification, especially in poor readers (Perfetti & Roth, 1981; Stanovich, 1980; Tunmer, 1989; Tunmer & Chapman, 1998), it would seem that syntactic deficits that impede a child's ability to use linguistic context to aid word identification could contribute to beginning reading problems in such children. Yet, vocabulary and syntactic knowledge do not often distinguish between children from the population defined herein and normally achieving readers, except in contrasts involving older children characterized by long-standing reading disorder (e.g., Fletcher, Satz, & Scholes, 1981; Shankweiler et al., 1999; Snowling, 2000a; Stanovich, 1986; Vellutino, Scanlon, & Tanzman, 1988; Vellutino et al., 1995b, 1996). Such findings suggest that early reading difficulties in children from this population may not be caused primarily by vocabulary and syntactic deficits and may, more often, be a consequence of prolonged reading problems. These deficits may also be co-morbid, reflecting the co-occurrence of oral language and reading difficulties (e.g., Catts et al., 2003). However, existing data do not preclude the possibility that vocabulary and syntactic deficits could contribute to difficulties in learning to read in some children. They would inevitably be a significant cause of reading comprehension problems, even in children who have adequate facility in word identification (Snowling, 2000b).

It is also worth noting that most of the studies that have appeared in the reading disability literature purport to use sampling criteria that exclude disadvantaged children and children who have limited proficiency with spoken English from their research samples. This, of course, may account for the apparent discrepancy between studies evaluating the relationship between vocabulary and/or syntactic knowledge and early reading difficulties in children impaired by specific reading disability as compared with studies evaluating disadvantaged

children or children with limited English proficiency. Thus, we suggest that limited vocabulary knowledge and/or syntactic deficits might be significant sources of difficulties in learning to read in at least some beginning readers, but likely have little to do with the word recognition and phonological decoding problems that are the primary markers of such difficulties in most impaired readers.

Phonological coding deficits. We earlier defined phonological coding as the ability to use speech codes to represent information in the form of words and parts of words. In contrast to the weak support for semantic and syntactic deficit explanations of specific reading disability in the population of children typically studied, there is now strong and highly convergent evidence in support of weak phonological coding as an underlying cause of the disorder. As we indicated earlier, the most compelling evidence for a causal relationship between phonological skills deficiencies, as manifest causes of inadequate achievement in beginning readers, is provided by training and intervention studies which have documented that direct instruction designed to facilitate phonological awareness and letter-sound mapping has a positive effect on word identification, spelling, and reading ability in general. In addition, poor readers have been consistently found to perform below the level of normal readers on phonological awareness and letter-sound decoding tasks as we also indicated earlier (Blachman, 2000; Fletcher et al., 1994; Shankweiler et al., 1979; Share & Stanovich, 1995; Stanovich & Siegel, 1994; Snowling, 2000a; Torgesen, Wagner, & Rashotte, 1994; Wagner, Torgesen, & Rashotte, 1994; Vellutino, 1979, 1987; Vellutino & Scanlon, 1987a, b; Vellutino et al., 1994, 1995a, b, 1996; Wagner & Torgesen, 1987). Such findings have led to a growing consensus that the most influential cause of difficulties in learning to read is the failure to acquire phonological awareness and skill in alphabetic coding. Difficulties in acquiring phonological awareness and skill in alphabetic coding are believed to be due, in many cases, to weak phonological coding characterized by poorly specified phonological representations (Griffiths & Snowling, 2002).

Weak phonological coding is presumed to underlie other problems that may contribute to difficulties in learning to read. Some scholars suggest, for example, that weak phonological coding can lead to difficulties in storing and/or retrieving printed words as unitized and distinctive orthographic representations as well as to difficulties in processing information in working memory (Brady, Shankweiler, & Mann, 1983; Elbro, 1997; Gathercole & Baddeley, 1990; Katz, 1986; Shankweiler et al., 1979; Share & Stanovich, 1995; Stanovich & Siegel, 1994; Snowling, 2000a; Torgesen et al., 1994; Torgesen, Wagner, Rashotte, Burgess, & Hecht, 1997; Vellutino, 1979, 1987; Vellutino et al., 1994, 1995a, b, 1996; Wagner et al., 1994; Wagner & Torgesen, 1987). More specifically, it has been suggested that difficulties in name storage and retrieval could impair the beginning reader's ability to establish connective bonds between the spoken and graphic counterparts of printed words, which, in turn, could impair the reader's ability to store quality representations of word spellings, and, thereby, impede his or her ability to acquire fluency in word identification. Fluency in word identification is, of course, a critically important prerequisite for adequate reading comprehension (Perfetti, 1985). And, given the demonstrated importance of working memory in language processing (Baddeley, 1986; Daneman & Carpenter, 1980; Ericsson & Kintsch, 1995), working memory deficits associated with weak phonological coding could impair reading comprehension, independent of dysfluency in word identification.

Thus, in accord with these possibilities, poor readers have been consistently found to perform below the level of normally achieving readers, not only on tests evaluating word identification, phonological awareness, and letter-sound decoding, but also on tests evaluating confrontational naming, rapid naming, verbal learning, and verbal memory (Blachman, 1997; Bowers & Wolf, 1993; Katz, 1986; Snowling, 2000a; Torgesen et al., 1994; Vellutino, 1979, 1987; Vellutino & Scanlon, 1987a, b; Vellutino et al., 1994, 1995a, b, 1996; Wagner et al., 1994; Wolf, Bowers, & Biddle, 2000a). Along with phonological awareness and phonological decoding deficits, this collection of deficits has been commonly attributed to weak phonological coding. Thus, weak phonological coding is hypothesized to be the central cause of specific reading disability in many if not most impaired readers, as articulated, for example, in what has been called the 'phonological core variable differences' model of specific reading disability (Stanovich, 1988; Stanovich & Siegel, 1994).

Finally, a number of researchers have attempted to provide more direct evidence for the inference that weak phonological coding is the central cause of specific reading disability and that dyslexic children are impaired by poorly specified phonological representations. The studies conducted have compared dyslexic and normal readers on both speech perception and production tasks, using both word and non-word stimuli varying in degree of phonetic similarity, in addition to reading age and chronological age-matched designs to evaluate reader group differences in speech discrimination and encoding. Studies using speech perception tasks typically evaluated categorical perception of stop consonants in these two groups and obtained suggestive, though somewhat inconsistent, evidence that dyslexic readers perceive phonetic boundaries less sharply than do normal readers (Adlard & Hazan, 1997; Brandt & Rosen, 1980; Godfrey, Syral-Lasky, Millay, & Knox, 1981; Hurford & Sanders, 1990; Manis et al., 1997; McBride-Chang, 1995; Mody, Studdert-

Kennedy, & Brady, 1997).[2] However, a small number of studies that have used the 'gating' paradigm to assess spoken word identification rather than more basic speech perception processes have provided less consistent evidence of dyslexic difficulties in such perceptual processing (Griffiths & Snowling, 2001; Metsala, 1997).

Studies using speech production tasks (which entail both speech perception and speech production mechanisms) have typically evaluated verbal repetition of both high and low frequency words, in addition to nonsense words presented under both noise-masked and noise-free stimulus conditions. The researchers have generally found reader group differences on these tasks, especially under noise-masked conditions, and more reliably when the stimuli were nonsense words (Brady et al., 1983; Elbro, 1997; Elbro, Borstrom, & Petersen, 1998; Snowling, Goulandris, Bowlby, & Howell, 1986).

Noteworthy is the observation that reader group differences in these studies tended to be more reliable in contrasts involving more severely impaired readers, suggesting that basic deficits in speech perception and production may underlie phonological skills deficiencies in only some poor readers (Heath, Hogben, & Clark, 1999). This pattern of results suggests that deficiencies in phonological skills such as phonological awareness and letter-sound decoding may, in some children, be caused by factors other than basic deficits in speech perception and/or production, such as inadequate instruction and experience. If so, then it would be important to develop the means for distinguishing between these two groups of impaired readers (see discussion below). Nevertheless, the evidence garnered from these more direct tests of the weak phonological coding theory of reading disability, although inconclusive, is highly suggestive. Additional research of this type is certainly warranted.

The double deficit hypothesis. Not all scholars accept the view that specific reading disability and reading-related cognitive deficits such as those mentioned in the previous section are caused exclusively or primarily by limitations in phonological coding. And, not all scholars accept the view that the name retrieval deficits that have often been observed in poor readers are due to weak phonological coding and (by extension) phonological memory problems. In particular, Wolf, Bowers, and their colleagues (Bowers & Wolf, 1993; Wolf & Bowers, 1999; Wolf et al., 2000a) postulate the existence of three subtypes of reading disability: one caused by deficiencies in phonological skills such as phonological awareness and letter-sound decoding, in accord with the phonological deficit model; a second caused by slow naming speed that specifically disrupts orthographic processing and reading fluency; and a third caused by a combination of both types of deficit (the 'double deficit'). The latter is considered to be the most serious form of reading disability, owing to the combined effects of phonological and rapid naming skills deficiencies.

According to this view, naming speed deficits are caused by disruption of a 'precise timing mechanism' that normally influences temporal integration of the phonological and visual counterparts of printed words, thereby impairing the child's ability to detect and represent orthographic patterns. It is asserted 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 redundancies and regularities. This, in turn will impair the child's ability to store distinct and unitized representations of word specific spellings. Thus, Bowers and Wolf (1993) hypothesize 'that slow letter (or digit) naming may signal disruption of the automatic processes which support induction of orthographic patterns, which, in turn, result in quick word recognition' (p. 70). Disruption in this timing mechanism has also been assumed to be manifested in slow object and color naming, both of which have also been observed in many poor readers.

Four types of evidence have been offered to support the double deficit theory of reading disability (see Wolf et al., 2000a for a recent review). First, naming speed tasks, especially letter and digit naming tasks, have consistently been found to account for unique variance in reading performance beyond that explained by phonological skills such as phonological awareness (Manis, Doi, & Bhadha, 2000; Wolf et al., 2000a). Second, a number of studies have shown that children grouped into either single deficit or double deficit subgroups, on the basis of their performance on speed of naming and phonological awareness tasks (respectively), tend to perform below children manifesting neither deficit on independent measures of reading achievement. Similarly, the double deficit groups typically perform below the single deficit groups on such measures (Wolf et al., 2000a). Third, phonological awareness and rapid naming appear to be differentially related to reading subskills, insofar as the former has been shown to be more strongly correlated with accuracy

[2] Note that categorical perception in phonological processing refers to a well established phenomenon in speech perception whereby listeners perceive a continuum defining a range of changing acoustic signals produced by the vocal apparatus as a single phoneme and sharply distinguish that phoneme from a second phoneme occupying an adjacent position on the same continuum and differs from the first by a single phonetic feature. For example, the initial consonants in the syllables /ba/ and /da/ differ by only a small change in the Voice Onset Time (VOT) – that is, the amount of time between closure of the lips and vibration of the vocal cords. Thus, using synthetic speech to produce continuous changes in VOT, researchers have observed that listeners draw sharp categorical boundaries between these two syllables, despite the fact that the changes in the acoustic signals, which define these boundaries, are continuous.

in word identification and letter-sound decoding whereas the latter has been shown to be more strongly correlated with speed of word identification and speed of letter-sound decoding (Manis et al., 2000; Wolf et al., 2000a). Finally, Morris et al. (1998) used cluster analysis techniques with reading disabled populations and provided documentation for naturally occurring subgroups of disabled readers (as opposed to artificially constituted subgroups) of the types specified by double deficit theorists. Moreover, in accord with results obtained in other studies, the double deficit group was found to be more severely impaired in reading than the single deficit groups.

Although such findings are suggestive, the double deficit hypothesis can be challenged on theoretical, interpretive, and methodological grounds. As regards its theoretical underpinnings, we suggest that the 'precise timing mechanism' that presumably underlies the formation of orthographic codes lacks the type of specification that would lend it psychological reality and allow it to be evaluated experimentally as a valid hypothetical construct. Indeed, descriptions of its properties and its role in word recognition tend to be complex and abstruse (e.g., Wolf et al., 2000a) and do not readily generate testable hypotheses that would facilitate such experimentation. More important is the fact that characterizations of the central deficit that is said to result from disruption of this timing mechanism – that is, inadequate temporal integration of letter identities – are reminiscent of earlier conceptualizations of the word recognition process which were based on the assumption that skilled word recognition is the end result of serial processing of a word's component letters (Gough, 1972). If the double deficit theory of reading disability is also based on this assumption (as it appears to be), then it is based on a conceptualization of word recognition that has long since been discredited (see Gough, 1984 for a review of this work). There is, in fact, good evidence that serial processing of a word's component letters is an immature mode of processing that is gradually abandoned as the child acquires facility in word recognition (LaBerge & Samuels, 1974). It may be inferred from this evidence that rapid serial processing and temporal integration of letter identities may not be the primary means by which orthographic codes are formed.

Complicating the picture even further is the fact that more recent formulations of the double deficit theory have viewed the fluency component of reading as a non-phonological deficit that is related primarily to the fluency of reading connected text. This observation has led to interventions focused on speed of processing of whole words and text characteristics in attempts to address this putative component of reading disability (Wolf, Miller, & Donnelly, 2000b). Although this formulation is consistent with the finding of Morris et al. (1998), that the subtype impaired only in rate of processing was not impaired in word recognition skills, it appears to be a significant departure from initial formulations of the double deficit theory, which suggest that a timing deficit resulting in poor integration of orthographic patterns is one of the two major processing deficits contributing to word recognition problems in poor readers (see above discussion).

Another question that has been raised about the double deficit theory of reading disability is concerned with the consistent finding that measures of rapid naming skill tend to account for unique variance in reading performance when phonological skills such as phonological awareness have been controlled (phonological awareness is the phonological skill almost always evaluated in the double deficit literature). Specifically, Torgesen et al. (1997) pointed out that such studies have not typically controlled for autocorrelation effects created by the variance that rapid naming and phonological skills share with reading performance (e.g., Bowers, 1995). Thus, in a longitudinal study addressing this question, these investigators found that second grade measures of phonological awareness accounted for unique variance on fourth grade timed and untimed reading and orthographic coding tasks, when performance on second grade reading measures was controlled. However, measures of speeded letter and digit naming did not account for unique variance on the fourth grade reading and orthographic coding tasks, when second grade reading performance was controlled. Essentially the same pattern of results was obtained with third grade predictors and fifth grade reading outcome measures, thereby calling into question conclusions drawn from previous double deficit studies that did not control for autocorrelation.

We should also point out that the consistent finding, that phonological and rapid naming skills each account for unique variance on measures of reading performance, need not be interpreted as support for the double deficit hypothesis and does not necessarily undermine phonological access explanations of slow naming speed. Given the likelihood that acquisition of these skills may be influenced, not only by common underlying cognitive abilities such as phonological coding and phonological access, but also by appreciably different cognitive abilities, different educational histories, and/or motivational factors, it is not surprising to find that they contribute unique as well as shared variance to reading performance. For example, whereas speeded naming tasks have a strong speech-motor component that may significantly influence performance on these tasks, phonological awareness tasks do not have a strong speech-motor component and are less apt to be influenced by individual differences in speech-motor articulation. Conversely, unlike speeded naming tasks, phonological aware-

ness tasks have strong metacognitive and metalinguistic components that no doubt influence performance on such tasks. Moreover, both types of tasks may be differentially affected by instructional factors (e.g., instruction that focuses on letter recognition but not phonological awareness or vice versa) and by personality and motivational characteristics of the child (e.g., willingness to offer a response in the face of uncertainty and/or inclination to comply with the request to name things as quickly as possible). Such differences could also account, in part, for the consistent finding that phonological awareness and rapid naming tasks tend to be differentially related to different types of reading performance, such as accuracy versus speed of word identification and letter-sound decoding. Yet, such variability does not rule out the possibility that individual differences in phonological coding and phonological access may be the primary factor influencing performance on both types of tasks.

Perhaps the most serious challenge to the double deficit theory of reading disability inheres in certain methodological problems associated with constituting hypothesized subgroups of impaired readers on the basis of scores on phonological awareness and rapid naming tasks. To be brief, Schatschneider, Carlson, Francis, Foorman, and Fletcher (2002) have demonstrated that because these two types of measures tend to be significantly correlated, and because the relationship between phonological awareness and reading performance tends to be curvilinear, it is likely to be the case that double deficit reader groups will have phonological awareness scores that are substantially lower than those of single deficit reader groups found to be deficient only in phonological awareness. Compton, DeFries, and Olson (2001) have obtained similar results. Moreover, Compton et al. (2001) also found that when the double and single deficit groups were matched on the phonological awareness and rapid naming tasks used to define these (respective) groups, many of the differences on the reading measures disappeared. Furthermore, they were unable to find matches for the most severely impaired readers. It is also worth noting that, in many of these studies, the naming speed deficit groups were found to have at least average-level word recognition skills, unlike the phonological deficit groups (e.g., see Manis et al., 2000). Such findings suggest that the larger differences typically observed between single and double deficit groups on reading tasks are due primarily to deficiencies in phonological awareness and related phonological skills, rather than to the combined effects of phonological and rapid naming deficits, thereby compromising a basic assumption of the double deficit theory of reading disability. They also favor phonological deficit explanations of specific reading disability, and further question the role played by rapid naming skills in reading development, as propounded by double deficit theorists. We should also point out that relationships between rapid naming tests and reading tests tend to be stronger for letter/digit tasks than for color/object tasks. Such relationships are also stronger for fluency than for accuracy tasks. Thus, rapid letter/digit naming may be a simple, early measure of reading speed as opposed to a component process (Schatschneider et al., 2002).

Finally, Kail, Hall, and Caskey (1999) evaluated the unique contributions made by general processing speed (timed matching and target search tasks), naming speed (letter and digit tasks), and print exposure (author recognition) to reading performance (word recognition and reading comprehension tasks) in randomly selected groups of children across a broad age span (ages 7 to 13 years). Using step-wise regression techniques, these investigators found that, whereas print exposure predicted performance on the reading measures, it did not predict performance on the rapid naming measures, as would be anticipated by double deficit conceptualizations of reading disability. In contrast, general processing speed did predict speed of naming, but not reading performance when age was controlled. At the same time, none of these variables predicted performance on the reading comprehension measure when word recognition was controlled. The investigators concluded from these findings that the rapid naming–reading link may stem from individual differences in 'global processing speed', rather than from individual differences in reading-specific skills such as speed of letter naming, implying that speed of naming deficits may not be causally related to phonologically based reading disability.

However, because Kail et al. (1999) did not employ timed reading tasks, relationships with fluency are possible. Moreover, rapid naming deficits have been observed in populations of children with learning impairments that are not associated with reading disabilities, including attention deficit hyperactivity disorder (Tannock, Martinussen, & Friitjers, 2000). Such findings have implications for phonological deficit as well as for double deficit conceptualizations of the relationship between naming speed and reading development and certainly question the notion that rapid naming deficits are specifically related to reading disability (Waber, Wolff, Forbes, & Weiler, 2000).

Low-level auditory deficits

We should also mention another theory of reading disability that has attracted widespread attention in recent years – one that implicates deficiencies in low-level auditory processing as the basic cause of the phonological deficits typically observed in dyslexics. The reference here is to Tallal's (1980) temporal order perception theory of dyslexia (see Farmer & Klein, 1995 and Tallal, Miller, Jenkins, & Merzenich, 1997, for recent reviews). This causal hypothesis

was based on earlier research conducted by Tallal and her associates, which demonstrated that children suffering from specific language impairments had difficulty making temporal order judgments (TOJ) with high and low tones presented at long (400 ms) versus short (50 ms) interstimulus intervals (ISIs; Tallal & Percy, 1973, 1975). In extending this research, Tallal (1980) found that dyslexics performed below normal readers on essentially the same TOJ tasks at short ISIs, but as well as the normal readers at long ISIs. Despite the fact that only 9 of the 20 impaired readers in the sample had difficulty with the TOJ task (a task with which many of the normal controls also had difficulty) and largely because of a high correlation between performance on the TOJ task and performance on a nonsense word decoding task (rho = .81), Tallal inferred that dyslexic readers suffer from a basic, non-linguistic deficit in temporal resolution of rapidly changing auditory stimuli that impairs speech perception. She also inferred that this disorder underlies the phonological deficits typically observed. However, this inference was speculative because Tallal did not use speech stimuli to assess temporal order judgment at varying inter-stimulus intervals. In addition, many children had raw scores of 0 on the reading task, which would inflate nonparametric correlations. Yet, in a later study, Reed (1989) attempted to replicate Tallal's findings with dyslexic and normal reading children, using both stop consonants and steady-state vowels along with brief tones, and found those with dyslexia had more difficulty than the normals in making temporal order judgments with the stop consonants and tones, but performed as well as the normal readers with the steady state vowels. Those with dyslexia also performed below the normal readers on a phoneme discrimination task.

Although these findings would appear to offer support for Tallal's interpretation of reader group differences on TOJ tasks, they do not confirm that the poor readers' difficulties on both the verbal and non-verbal TOJ tasks arise from the same underlying perceptual mechanism. Thus, in a series of experiments that more carefully controlled the types of verbal and non-verbal stimuli presented to participants, Mody et al. (1997) provided documentation that the difficulties of poor readers on TOJ tasks using speech stimuli were due to speech discrimination deficits rather than temporal order judgment deficits (see also Studdert-Kennedy & Mody, 1995). In the first experiment in this study (Experiment 1a), the investigators verified that poor readers had more difficulty than normal readers with TOJ tasks involving phonetically and acoustically similar speech stimuli (e.g., /ba/–/da/) at short interstimulus intervals (ISI). However, when each of these stimuli was paired with a consonant–vowel stimulus that was acoustically and phonetically very different (e.g., /ba/–/sa/, Experiment 1b), the groups performed at comparable levels on the TOJ tasks. In a third experiment (Experiment 2), the poor and normal reader groups were given TOJ tasks using nonspeech stimuli that were acoustically matched to the onset transitions of the speech stimuli used in these experiments. No statistically significant group differences emerged at any of the ISIs used in the experiment. In fact, the normal readers were slightly worse on these tasks than the poor readers. The investigators concluded from these results that a general auditory deficit of the type posited by Tallal is an unlikely source of the phonological deficits typically observed in poor readers.

In virtually all of the studies providing support for Tallal's temporal order perception theory of dylexia, questions can be raised about the adequacy of the criteria used to identify children as dyslexic. As our review of research on the core deficits showed, it is essential to define children as dyslexic on the basis of difficulties identifying words in isolation. In addition, it has been shown that 30%–70% of children with dyslexia also have attention deficit–hyperactivity disorder (ADHD), depending on the setting and how ADHD is defined (Fletcher, Shaywitz, & Shaywitz, 1999). Two recent studies provided evaluations of the auditory temporal processing hypothesis in well-defined samples of children with dyslexia that also controlled for the presence of ADHD. Waber et al. (2001) selected children with dyslexia and no ADHD from a larger group of children originally referred for clinical evaluations of learning problems. Auditory processing tests involving speech and nonspeech stimuli were used that varied in the interstimulus interval, thus permitting evaluation of Tallal's (1980) hypothesis that rate of processing was impaired in children with dyslexia. The results revealed significant differences between good and poor readers in the discrimination of speech and nonspeech stimuli, but no effect of interstimulus interval. Thus, group differences in perceptual ability were apparent that did not appear to be related to rapid temporal processing.

In the second study, Breier, Fletcher, Foorman, and Gray (2002) administered temporal order judgment and discrimination tasks that also varied in demands for processing of acoustic stimuli across interstimulus intervals. The authors created 4 groups of children with sample sizes of about 40 per group: children with reading disability and no ADHD, children with reading disability and ADHD, children with ADHD and no reading disability, and typically achieving children with no ADHD. The results were not consistent with the auditory temporal processing hypothesis. As in Waber et al. (2001), children with dyslexia did not show a specific sensitivity to variations in interstimulus intervals, although they tended to perform more poorly than those children without reading difficulties. In addition, phonological processing measures were only correlated with the processing of speech stimuli. However, in contrast to Waber et al. (2001), the

difficulties were more apparent on speech than nonspeech stimuli. It may be that Waber et al. (2001) found differences on nonspeech stimuli because they included children with both reading and oral language difficulties in their sample. Breier et al. (2002) specifically excluded children with oral language difficulties. Moreover, Heath et al. (1999) also found nonspeech auditory processing deficits in only those poor readers with concomitant oral language difficulties and not those poor readers without oral language difficulties. Both Waber et al. (2001) and Breier et al. (2002) concluded that children with dyslexia have difficulties with speech perception that produce deficits on temporal processing tests, but do not have a pervasive deficit in auditory temporal processing. The speech perception difficulties are related to and possibly contribute to phonological processing difficulties that in turn, affect reading skills.

Results from several other studies testing various aspects of Tallal's theory have led to similar conclusions (Best & Avery, 1999; Bishop, Carlyon, Deeks, & Bishop, 1999; Bradlow et al., 1999; McNally, Hansen, Cornelissen, & Stein, 1997; Nittrouer, 1999), thus questioning the viability of the theory. The most significant problem is that sensory deficits at the auditory level do not explain the word recognition difficulties in a parsimonious manner. The link with phonological processing is tenuous, though there is evidence that speech perception difficulties are related to phonological processing capabilities, a finding in many studies of children with dyslexia (see Mody et al., 1997). Dyslexia implies more than reading difficulties and children with dyslexia differ from typically achieving children on a variety of dimensions, many of which relate to co-morbidities associated with dyslexia. However, these differences do not explain the reading problem and they tend to be small relative to those associated with phonological processing. Yet, they could be related to the underlying neurobiological problems that cause dyslexia in some children, as suggested by Eden and Zeffiro (1998) and other scholars (Fletcher et al., 1999).

Dyslexia in different languages

Our discussion so far has focused on the cognitive characteristics of dyslexia and its manifestations from the perspective of children learning to read in English. However, it needs to be borne in mind that English has an opaque (or deep) orthography in which the relationships between letters and sounds are inconsistent and many exceptions are permitted. As such, English presents a significantly greater challenge to the beginning reader than other more regular alphabetic systems that contain consistent mappings between letters and sounds and are described as transparent (or shallow) orthographies. Indeed, several studies now show that the developmental progress of children learning to read in transparent orthographies such as German or Italian is generally faster than that of children learning written English (Harris & Hatano, 1999). Such children also show correspondingly faster development of phoneme awareness (Cossu, 1999). It follows from these findings that the utility of phonological abilities as predictors of reading development varies across different languages. For instance, while rhyming skill predicts learning to read in English (Bradley & Bryant, 1983), it is a poor predictor of subsequent reading achievement in German (Wimmer, Landerl, & Schneider, 1994) and Dutch (de Jong & van der Leij, 1999), where rapid naming ability accounts for larger proportions of unique variance in reading ability.

Although there has been a burgeoning of research on dyslexia in different languages in recent years (Goulandris, 2003), cross-linguistic studies that directly compare dyslexia in English (about which we know a great deal) and dyslexia in a different language are still comparatively rare. Nonetheless, the prevailing view is that the core phonological deficits of dyslexia are harder to detect in children who have learned to read in transparent orthographies such as German or Italian. In such languages, impairments can be identified most clearly on tasks that require implicit phonological processing, such as those evaluating verbal short-term memory, rapid naming and visual–verbal paired associate learning (Wimmer, Mayringer, & Landerl, 1998), rather than on tests evaluating explicit phonological processing such as phonological awareness and phonological (letter-sound) decoding. The reading problems associated with dyslexia also differ in regular orthographies as compared to less regular orthographies such as written English. For example, Wimmer and his colleagues have conducted studies showing that German-speaking dyslexic children can read long unfamiliar words and also nonwords as well as their normal reading peers (Frith, Wimmer, & Landerl, 1998). However, the fluency of their reading is affected; they read single words more slowly than controls and sometimes, reading comprehension difficulties follow as the consequence of a 'bottleneck' in the reading process (Wimmer et al., 1998). This disparity notwithstanding, a common finding is that spelling presents a major stumbling block for children with dyslexia across alphabetic writing systems. Dyslexic spelling deficits are very well documented in English (Treiman, 1997) and work with French-speaking children, for example, shows that children with dyslexia lag behind in their spelling development as well as in their use of phonological spelling strategies (Caravolas, 2003).

A more stark comparison is afforded between learning to read in alphabetic languages and learning to read in non-alphabetic languages such as Chinese or Japanese. The Chinese writing system differs from an alphabetic system in that it contains a large

number of visual symbols or characters that represent units of meaning (morphemes) rather than phonemes as in an alphabet. The task of learning to read is therefore a considerable feat for Chinese children who must learn literally hundreds of visually complex characters that contain phonetic and radical components. It is not surprising, therefore, that visual skills predict read ability in Chinese better than they predict reading ability in alphabetic writing systems. More surprising, at first glance, is that phonological skills are also predictors of individual differences in Chinese children's reading skills (Ho & Bryant, 1997), even though readers of Chinese do not develop phoneme awareness to the level observed among readers of alphabetic languages (Read, Zhang, Nie, & Ding, 1986).

The most comprehensive study of dyslexia in Chinese to date was reported by Ho, Chan, Tsang, and Lee (2002), who classified data from 30 children with dyslexia on tests evaluating visual processing, phonological processing, and rapid naming, as well as on tests evaluating their knowledge of the orthographic regularities of characters and radicals. Results from this study indicate that a rapid naming deficit was the most dominant type of cognitive deficit in Chinese dyslexic children, affecting some 60% of the cases. However, over half of the dyslexic children exhibited deficits in three or more cognitive domains and there was a significant association between the number of cognitive deficits and the degree of reading and spelling impairment. This study was small in scale and needs to be replicated on a larger sample. However, its findings provide tentative support for the idea that dyslexia in children learning to read written Chinese is associated with multiple deficits, rather than with a core phonological deficit as has been proposed for dyslexia in children learning to read written English.

The cross-linguistic study of dyslexia is at a relatively early stage. Moreover, the extant data are difficult to interpret. A fundamental difficulty is in equating diagnostic criteria across languages. In English, the diagnosis of dyslexia proceeds on the basis of a discrepancy between reading accuracy and age (or in some cases IQ). The same criteria cannot be used in transparent writing systems where accuracy levels are typically high (Paulesu et al., 2001). Rather, criteria tend to center on speed and fluency rather than error-rate. Thus, it is entirely possible that different individuals will fulfill criteria for dyslexia in one language but not in another. Indeed, Wydell and Butterworth (1999) reported a case of a bilingual boy who was able to read normally in Japanese, but manifested many of the reading behaviors characteristic of dyslexicia when attempting to read English.

Taken together, the evidence underlines the fact that although dyslexia, quite likely, has a neurobiological basis, it is not just a product of nature but, rather, a complex condition that depends on the dynamic interaction between certain innate susceptibilities as well as the home and school environments on one hand, and the cultures in which children learn to read on the other. It is not an all or none condition. Just as some individuals reach the threshold for a diagnosis of dyslexia and others with a similar cognitive profile do not succumb to reading failure, certain transparent orthographies, like German and Italian, benefit learning to the point where dyslexia may be 'hidden' in the majority of cases, while other, less transparent orthographies, like English, may aggravate the problem.

Underlying causes: biological foundations of dyslexia

Neurobiological factors

The World Federation of Neurology definition of dyslexia provides a major role for neurobiological factors by indicating that dyslexia is 'dependent upon fundamental cognitive disabilities, which are frequently of constitutional origin' (Critchley, 1970, p. 11). However, this statement for many years was little more than an assumption based on several sources of indirect information. One source was the linguistic and behavioral characteristics of adults with documented brain injury, where fractionations of reading from other skills in otherwise normal individuals could be observed. The second involved associations of nonspecific indices of neurological dysfunction with dyslexia, including perceptual-motor problems, 'soft' neurological signs, and motor clumsiness, along with a potpourri of findings on electrophysiological measures (Dykman, Ackerman, Clements, & Peters, 1971; Taylor & Fletcher, 1983). Finally, it was commonly assumed that if dyslexia was not due to intellectual, sensory, socio-cultural, or instructional factors, it must be due to constitutional factors. Thus, the constitutional nature of dyslexia was inferred on the basis of what it was not, rather than on the basis of direct evidence of central nervous system involvement (Rutter, 1978). During the past 15 years, it has become possible to more directly evaluate the hypothesis that dyslexia is caused by constitutional factors that are intrinsic to the child. In the next section, we review studies of a) brain structure, b) brain function, and c) genetics.

Brain structure

Post mortem studies. The most significant research on brain structure utilizes post mortem studies or anatomical magnetic resonance imaging (aMRI). Computed tomography studies were also utilized, but will not be reviewed here as MRI superseded these modalities (see Hynd & Semrud-Clikman, 1989).

Post mortem evaluations of the brains of people with dyslexia are rare since it is not a cause of

death. Nonetheless, the findings, while representing a cumulative total of 10 cases, are instructive in that they indicate that the underlying neuroanatomical basis of dyslexia is complex and not reducible to a single finding or area of the brain. One set of findings focus on a structure on the plane of the temporal lobe known as the planum temporale. This structure has been reported to be often larger in the left hemispheres than the right hemispheres of neurologically normal adults (Geschwind & Levitsky, 1968). In post mortem studies of dyslexics, it has been reported that there are unexpected symmetries in the left versus the right hemispheres of those whose brains were studied (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Humphreys, Kaufmann, & Galaburda, 1990). This area of the left hemisphere supports language functions, so the symmetry is viewed as a partial cause of language deficiencies that, in turn, lead to reading problems.

A second set of findings involves small, focal anomalies that appear when microscopic evaluations of the brain are performed. These anomalies were found to be more common in the left hemispheres of people with a history of dyslexia, though they were also found to be more frequent in other parts of the brain. Microscopic examinations of subcortical structures have also shown differences in the thalamus that may be related to visual processing (Livingstone et al., 1991).

These studies, while implicating difficulties at the level of brain structure, have some limitations. Obviously, the educational histories, reading characteristics, and other factors that influence brain organization (e.g., handedness) are difficult to evaluate on a post hoc basis. There are no specific control groups, and all the historical information is retrospective, often derived from a period where the diagnosis of specific reading problems was not advanced. Given these difficulties and the limited number of available brains, investigators have turned to methods based on neuroimaging.

Anatomical magnetic resonance imaging. Noninvasive methods based on aMRI have revolutionized the search for neural correlates of dyslexia. The resolution is excellent and sufficient so that precise measurements of brain structure can be completed. A variety of structures have been evaluated, including the planum temporale, temporal lobes, and corpus callosum. Unfortunately, while the aMRI data are easily acquired, the measurement of brain structures is arduous and often requires considerable human effort, which restricts sample size. There are also variations in how structures are measured and in the definition of landmarks. Thus, it is not surprising that the results are mixed and tend to vary across laboratories.

For the planum temporale, different studies report symmetry (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; Larsen, Høien, Lundberg, & Odegarrd, 1990), reversals in the expected pattern of asymmetry (Hynd et al., 1990), and no relationships of size or symmetry of the planum temporale in dyslexia (Rumsey et al., 1997; Schultz et al., 1994). Leonard et al. (1996) reported that higher degrees of asymmetry of the temporal lobes, favoring the left hemisphere, were associated with better reading performance, regardless of whether the child was disabled in reading. This finding implies lack of specificity with regard to children with reading disability, but Leonard et al. (2001) failed to replicate the finding. Other studies report that the temporoparietal brain areas are smaller (Duara et al., 1991; Kushch et al., 1993), or not different in those with or without reading difficulties (Hynd et al., 1990; Jernigan, Hesselink, Sowell, & Tallal, 1991). Consistent with this theme, studies that measure the corpus callosum find differences in its size (Duara et al., 1991; Hynd et al., 1990) as well as no differences in its size (Larsen et al., 1990; Schultz et al., 1994) between groups with and without dyslexia.

The differences across studies clearly relate to many factors, including small samples and variation in imaging methods. Subject-level factors are clearly important (Filipek, 1996; Shaywitz et al., 2000). Schultz et al. (1994) found statistically significant differences on several measures between children with dyslexia and age matched controls, including the planum temporale and other left hemisphere structures. However, when age, gender, and handedness were covaried, the only reliable finding was a small reduction in the size of the left temporal lobes in the group with dyslexia. Similarly, Pennington et al. (1999) found reductions in the size of the insula and anterior superior cortex in both hemispheres of a group of twins with dyslexia. An area of the brain defined as posterior to the splenium of the corpus callosum, comprising the posterior temporal, parietal, and occipital areas, was larger in both hemispheres of the group with dyslexia. These differences persisted when age, gender, and IQ were controlled, but were relatively small.

Although the findings are consistent, there is evidence suggestive of subtle differences in several brain structures between groups with and without dyslexia. But these studies have diminished in frequency because of their difficulty and relatively low yield, especially relative to functional neuroimaging methods (see below). However, new modalities for structural neuroimaging may lead to resurgence. To illustrate, Klingberg et al. (2000) used diffusion tensor imaging to evaluate the integrity of the cerebral white matter in the left hemisphere language regions, showing less development of white matter in a group with dyslexia. These results suggested reduced myelination of these language-mediating areas. In the future, more application of these methods are likely, especially in conjunction with multi-modal studies that also employ functional brain imaging with the same person.

Brain function

Most current studies of neural factors in dyslexia use functional neuroimaging modalities that assess the response of the brain to cognitive challenges. Functional neuroimaging in dyslexia utilizes five different modalities, including variations in electrophysiological methods, positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetic source imaging (MSI), and magnetic resonance spectroscopy (MRS). A review of these methods is beyond the scope of this paper (see Papanicolaou, 1998). The methods all measure changes in the brain that occur during cognitive processing as a basis for mapping where and/or when in the brain these changes occurred. Thus, metabolic changes reflected by glucose utilization or shifts in blood flow from one part of the brain to another part of the brain occur during cognitive processing and can be captured by PET or fMRI. In contrast, when neurons discharge, changes in the brain electrical activity occur that can be captured at the scalp by electrophysiological methods. Changes also occur in the magnetic fields surrounding these electrical discharges at the neuronal level, which can be detected with MSI.

Finally, MRS detects changes in brain chemistry that occur in relation to changes in state (Hunter & Wang, 2001). Methods that are sensitive to metabolic activity capture changes that occur after the cognitive activity has occurred and are not sensitive to when the change occurred. Methods such as MSI (and EEG) take place in real time and provide information on the time course of neural events. Their spatial resolution is weak, so the maps are coregistered on an MRI scan.

Previous research has used all five modalities. However, since the resolution of maps from electrophysiological procedures does not have very good spatial resolution, these studies will not be further discussed. There are a few MRS studies, which will be discussed within the context of intervention. The findings from PET, fMRI, and MSI converge. They show that tasks requiring reading and phonological processing are associated with increased activation in the basal surface of the temporal lobe, the posterior portion of the superior and middle temporal gyri, extending into temporoparietal areas (supramarginal and angular gyri), and the inferior frontal lobe. These activations are often bilateral in PET and fMRI (Eden & Zeffiro, 1998; Rumsey et al., 1997; Shaywitz et al., 2000), but tend to be more lateralized in the MSI studies (Simos, Breier, Fletcher, Bergman, & Papanicolau, 2000a; Simos et al., 2000b).

In MSI studies, children who are typically achieving show a pattern in which the occipital areas of the brain that support primary visual processing are initially activated. Then regions in the basal temporal areas in both hemispheres are activated. Shortly thereafter, there is virtually simultaneous activation of three areas in the left temporal and parietal areas of the left hemisphere, roughly corresponding to the superior temporal gyrus, Wernicke's area, and the angular gyrus. In contrast, children with dyslexia activate the same areas of the right hemisphere, with a similar time course. Other studies using PET and fMRI also show evidence of right hemisphere activation in the posterior temporal parietal regions (Shaywitz et al., 2002). This could reflect compensatory processes or could indicate that other nonlinguistic factors are related to reading disability (Grigorenko, 2001; Joseph, Noble, & Eden, 2001; Wood & Grigorenko, 2001). These differences in activation patterns across modalities as well as other inconsistencies across studies are apparent (Poeppel, 1996). However, a network of areas is consistently implicated in studies of people with dyslexia. Particularly important is the angular gyrus, which studies of acquired reading disorders commonly implicate as a feature of the pattern of lesions that cause pure alexia. Horwitz et al. (1998) and Pugh et al. (2000) conducted statistical analyses of PET and fMRI results and showed that the angular gyrus in the left hemisphere was poorly connected with other areas involved in the mediation of reading in dyslexic compared with proficient readers.

Most recently, imaging studies have been conducted before and after attempts to remediate reading impairment (Richards et al., 2000; Simos et al., 2002a). Richards et al. (2000) used MRS to evaluate changes in brain chemistry as part of a comprehensive, phonologically oriented three-week (30-hour) intervention. Before intervention, MRS showed a higher metabolic rate of lactate in the left anterior quadrant of the left hemisphere when children with dyslexia completed a reading task. After intervention, measures of lactate metabolism taken during reading were not different in children with dyslexia compared with controls.

In a similar study using MSI, Simos et al. (2002b) evaluated neural responses to an intense phonological intervention in 8 children with severe dyslexia. The children were 7–17 years, with 6 of 8 reading below the fourth percentile and the other two reading below the 19th percentile. After about 80 hours of instruction over 8 weeks, their scores on measures of word reading accuracy were in the average range. In each child, there was a significant increase in the activation of neural circuits in the left hemisphere that tend to be activated in good readers, but were not activated in the dyslexics at baseline (see example in Figure 2). A comparison group of good readers assessed 8 weeks apart showed no significant changes in left hemisphere activation. Although the changes were statistically significant, the sample size was small and replication is needed. In addition, latency data showed delays in the evoked fields associated with the left hemisphere response. Such delays may relate to the failure of these types of interventions to produce dramatic gains in reading fluency with older children (Torgesen et al., 2001).

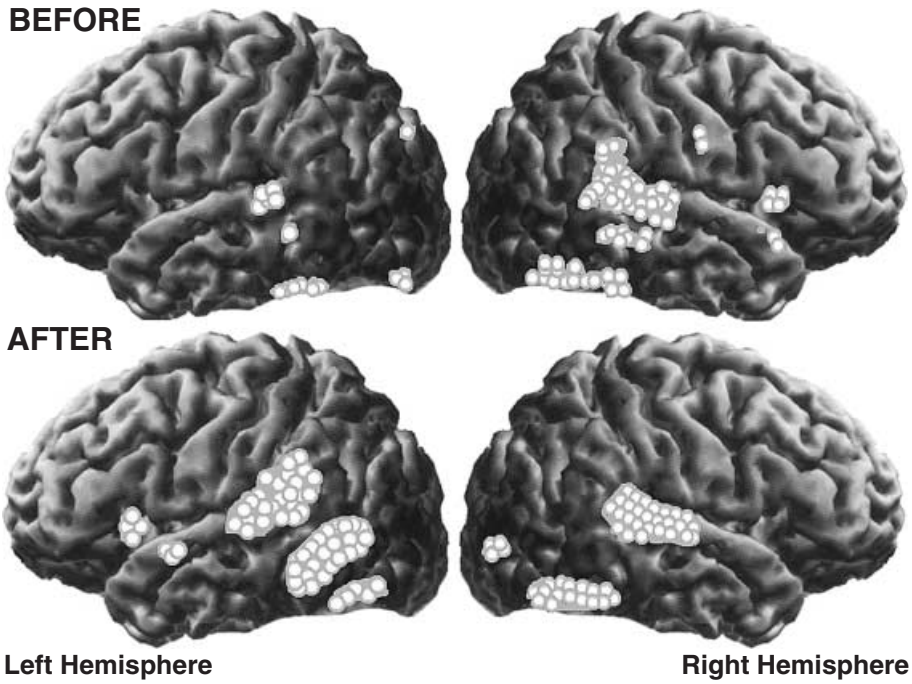

Figure 2 Brain activation maps from a child with severe dyslexia before and after an 8 week intense intervention in which word reading skills moved into the average range. The upper panel shows the typical brain activation map from magnetic source imaging studies of dyslexia, with predominant activity in temporal and parietal areas of the right hemisphere, but little activation in homologous areas of the left hemisphere. In the lower panel there is a significant increase in the activation of these left temporoparietal areas associated with the significant improvement in word reading accuracy that parallels the patterns observed in proficient readers (based on Simos et al., 2002a).

Results from these two studies suggest that instruction may be necessary to establish the neural networks that support word recognition abilities in dyslexia. The results may also extend beyond dyslexia and imply that environmental factors are essential in establishing these networks in all children. Simos et al. (2002b) identified children who showed risk characteristics for reading difficulties at the end of kindergarten due to poor development of phonological awareness skills. Application of MSI during a task assessing the child's ability to identify letter sounds showed brain activation profiles that were quite similar to those identified in older children and adults with dyslexia. Children who were not at risk showed the patterns of left hemisphere activation seen in older proficient readers. These children are being followed and the children at risk for reading difficulties receive intensive intervention in Grade 1. Follow-up imaging will be completed at the end of grade 1 to determine whether the patterns of the at-risk children shift towards those of the not-at-risk children. But the direction of the research suggests that the brain and environment, either through instruction or some other form of early literacy support, interact to produce the neural networks that must be in place to mediate the unique component of reading – word recognition.

Genetic studies

Studies of the heritability of dyslexia also show evidence for a prominent role of environmental factors as well as converging evidence that reading skills have a strongly genetic influence (see recent reviews by Grigorenko, 2001; Pennington, 1999; Olson, Forsberg, Gayan, & DeFries, 1999; Olson & Gayan, 2001). In family segregation studies, the risk of dyslexia is 8 times higher in children where there is a parental history of reading difficulties. Different studies report that 25%–60% of the parents of dyslexic children also display reading difficulties. In twin studies, concordance rates are almost always above 80% for monozygotic twins and usually below 50% for dizygotic twins. As monozygotic and dizygotic twin pairs share the same environment, differences in concordance rates presumably relate to the heritability. Statistical approaches to family and twin studies also produce evidence for the heritability of reading disability. From these studies, about 50–60% of the variance in reading achievement and reading-related abilities can be explained by genetic factors. By the same token, the studies also show that environmental factors account for significant variance in reading skills, but the contribution of genetic factors is consistently higher (Olson et al., 1999; Grigorenko, 2001). Finally, linkage studies of families with many individuals with dyslexia have been used to identify specific genes involved in reading disability. Five different laboratories have identified an area on chromosome 6 (Grigorenko, 2001). Replicated findings have also occurred for chromosome 15 (Grigorenko et al., 1997; Smith, Pennington, Kimberling, & Ing, 1990). Potential

markers on chromosome 1 and 2 have been reported but not replicated (Grigorenko, 2001).

Fisher and DeFries (2002) provide a detailed review of genetic studies of dyslexia. They place the study of the heritability of dyslexia squarely within the study of other complex human problems, and link it closely with studies of the heritability of reading skills. Their extensive review of genetic methodologies and their application to dyslexia and reading should be consulted for a more extensive evaluation of an area of research with a long history (see also Grigorenko, 2001).

Fisher and DeFries (2002) emphasize that the heritability of dyslexia has biological and environmental influences, with Olson and Gayan (2001) providing an extensive discussion of shared and non-shared environmental factors. Studies of twins are especially useful in teasing apart the contribution of shared environmental and genetic factors. Olson and Gayan (2001) observe, for example, that while twin pairs (identical and fraternal) may share similar environments, the differences in the genes of fraternal twins are more likely to lead them to select different environments. As groups, identical and fraternal twins are treated differently in schools so comparisons of identical and fraternal twins help establish not only the differential influence of genetic factors, but also the differential impact of environmental factors. Olson and Gayan (2001) also summarize behavioral-genetic studies involving a variety of reading skills and reading-related subskills, such as phonological and orthographic awareness. For example, word recognition shows a genetic influence of .45 and a shared environmental influence of .15. Related subskills, however, have higher genetic influences (e.g., phonological awareness −.56; orthographic awareness −.58), lower shared environmental influences (phonological awareness −.24; orthographic awareness −.20) and higher non-shared environmental influences (phonological awareness −.20; orthographic awareness −.22). Thus, individual differences in reading are partly due to shared genetic influences and partly due to shared and non-shared environmental influences. Olson and Gayan (2001) also note that these influences appear linked to early literacy development. It is also clear that pre-schoolers with early language difficulties are at greater risk for reading problems (Snowling, Bishop, & Stothard, 2000) and that children with oral language problems also show evidence that these difficulties have a genetic component (see discussion below). Studies that link the heritability of early language difficulties and the extensive research on the heritability of dyslexia are emerging and will be a significant contribution to our understanding of both (Olson & Gayan, 2001).

Like the initial studies of intervention and neuroimaging, genetic studies suggest that environmental factors are important determinants of individual reading profiles for many children with dyslexia. Both types of studies also show that intrinsic biological factors are important determinants of such profiles. Neither type of study fully explains why some children develop dyslexia and others do not do so, although the idea that people may vary in the intensity and quality of instruction necessary to establish word recognition skills is clearly apparent. Keep in mind that parents with reading problems read less frequently to their children than parents who do not have reading problems and are likely to have fewer books in the home (Wadsworth, Olsen, Pennington, & DeFries, 2000). The genetic studies also do not indicate that there are specific genes that produce dyslexia. As Gilger (2002) pointed out, the genetic influences appear to affect all levels of reading ability. Thus, many people are not born with dyslexia, but, rather, have a susceptibility that requires more intense instruction.

Dyslexia across the life-span

Additional support for the possibility that reading difficulties, in some children, are caused, in part, by genetically based cognitive deficits is provided by several family risk studies that have recently appeared in the reading disability literature. A strategy that has been increasingly used to identify the early precursors of dyslexia is to follow the developmental progress of children at high risk of dyslexia because of a genetic liability associated with the occurrence of dyslexia in at least one family member. This approach capitalizes on the fact that dyslexia runs in families and that there is approximately a 50% risk that a child will develop reading difficulties, given that they have a parent with dyslexia.

The first prospective study of children at family risk of dyslexia was reported by Scarborough (1990), who followed the progress of 32 two-year-olds from families with a history of reading disability and compared them with children from control families having similar socioeconomic backgrounds. At 8 years, 65% of the high-risk sample (20 children) was classified as reading-disabled. Examination of the data from earlier assessments of these children showed that, at 30 months, those who later became dyslexic used as wide a range of vocabulary in their conversation as controls and children from high-risk families who were normal readers. However, they used a more restricted range of syntactic devices and made more speech production errors. At 36 and 42 months, the high-risk children's vocabulary development was less well developed than that of controls and syntactic difficulties persisted (Scarborough, 1991). At 5 years of age, the children who later became dyslexic had poor letter knowledge, poorly developed phonological awareness, and expressive naming difficulties.

Thus, contrary to the findings of studies of school-age dyslexic samples that implicate a specific phonological deficit (Snowling, 2000a), Scarborough's

study found that dyslexia was characterized by oral language difficulties that transcended phonology in the pre-school years. Converging evidence has since been reported by several studies of children at high risk of dyslexia before formal schooling begins (Byrne, Fielding–Barnsley, Ashley, & Larsen, 1997; Gallagher, Frith, & Snowling, 2000; Locke et al., 1997; Lyytinen, Poikkeus, Laakso, Eklund, & Lyytinen, 2001; Lefly & Pennington 1996). However, it needs to be borne in mind that this method of recruitment is very different from that used in more conventional group studies that have selected children who fulfill 'discrepancy' and other more conventional criteria for reading disability. Furthermore, the finding of more general language delays in affected children applies to group data and, within groups of affected cases, some children may have more selective impairments or pre-school language delays that may resolve into more specific phonological deficits. An alternative interpretation of these findings, however, is that poor phonological processing carries the risk for reading disability in high-risk samples, but the impact of the risk may be modified or mitigated by variations in other cognitive and language skills.

In relation to this alternative version of the phonological deficit hypothesis, three studies provide support for the view that the risk of dyslexia is continuous rather than 'all-or-none', in the sense in which we discussed this dichotomy earlier. Pennington and Lefly (2001) followed the progress of 67 children at high risk of dyslexia and 57 controls considered to be at low risk, from before entry into kindergarten through second grade. At the end of the study, children who showed a discrepancy between IQ and composite reading and spelling scores were classified as reading-disabled. Using this criterion, 34% of the high-risk group were diagnosed as 'reading-disabled' in second grade, compared to only 6% of the low-risk (control) group. Consistent with the phonological deficit hypothesis, children who became reading-disabled showed deficits on tests of speech perception, verbal short-term memory, rapid serial naming, and phonological awareness at all testing points, relative to both the control children and the high-risk unimpaired children. Importantly, however, children at high risk of reading disability who were considered to be normal readers at the end of second grade scored significantly lower than children in the low-risk control group on most measures of reading and spelling. They also had more difficulty on tests of implicit phonological processing (particularly verbal short-term memory and rapid serial naming measured around second grade), although they were unimpaired on tests evaluating explicit phonological awareness. So it seems that the unimpaired children shared at least some of the cognitive characteristics of reading difficulties, despite the fact that their reading outcomes were within the normal range. More research is needed to determine how the high-risk unimpaired children managed to 'compensate' on tests of explicit phonological awareness in the face of weak implicit phonological processing skills.

In a similar vein, Snowling et al. (2003) followed the progress of 56 children from families in which at least one parent was dyslexic. These children were assessed periodically from the age of 3 years 9 months to age 8 years and their performance on measures of reading skills and reading-related cognitive abilities was compared to that of normal reading control children from similarly advantaged home backgrounds, but with no history of reading failure in family members. Sixty-six percent of the children had reading skills more than one standard deviation below the mean of the normal reading controls (the rate of discrepancy-defined dyslexia was lower at 32%). In line with previous studies, retrospective analyses revealed that the children who went on to develop reading disabilities had slower vocabulary development and poorer narrative skills in the pre-school years than high-risk children who went on to develop normal reading ability. They also had more difficulty on tests of verbal short-term memory and phonological processing (nonword repetition). At 6 years, the high-risk impaired group continued to have difficulty on tasks evaluating vocabulary development and language processing skills, and they also performed poorly on tests evaluating explicit phonological awareness. On all of these tests, the unimpaired group performed within the normal range. Nonetheless, they knew fewer letters than controls at age 3 years 9 months (although more than the reading impaired children), and at 6 years, their reading, spelling and reading comprehension skills were less advanced than those of the normal reading controls. Importantly, on two measures evaluating phonological reading and spelling strategies, one involving nonword decoding and the other involving the phonetic accuracy of early spelling, the high-risk unimpaired group performed as poorly as the high-risk impaired group and much less well than expected for their age. Since these children were not poor readers at 8 years, it appears that they were able to compensate for deficits in phonological reading and spelling strategies at 6 years, perhaps by using intact language skills to 'bootstrap' inefficient decoding mechanisms (cf. Nation & Snowling, 1998). Indeed, it seems that both affected and unaffected members of dyslexic families share the risk of reading failure that is characterized by poor 'phonic' skills (e.g., poor nonword reading). However, while some succumb to reading impairments, others do not.

Finally, further evidence for a 'broader phenotype' of dyslexia comes from a study of Danish children conducted by Elbro et al. (1998), who reported that non-dyslexic children in dyslexic families had deficits, relative to controls, on tests evaluating morphological awareness and articulatory accuracy.

Surprisingly, the difficulties these children were experiencing did not extend to letter knowledge, phoneme awareness, verbal STM or the distinctness of phonological representations. However, it is important to note that the high-risk children in the Elbro et al. study were being educated very differently than the high-risk children in the studies we have discussed thus far, most of whom were educated either in the United States or in the United Kingdom. For example, the children in the Elbro et al. study were in kindergarten classes for longer periods of time and they were learning to read in a different orthography, albeit an opaque one. It seems likely, therefore, that environmental factors play a role in the determination of the 'dyslexia phenotype'.

Taken together, the findings from the family risk studies suggest that dyslexia is a multifactorial trait in which basic constitutional (genetic) vulnerabilities (notably in phonological skills) interact with other cognitive skills and environmental factors to produce an increased risk of dyslexia in a continuous way. Arguably, when the level of risk reaches a certain threshold, the classic dyslexia profile emerges, but the evidence suggests that there are varying degrees of 'sub-clinical' impairment, particularly in dyslexic families.

Additional support for this conceptualization comes from studies evaluating the manifestations and correlates of dyslexia at the other end of the age spectrum, that is, studies of adults with a history of early reading difficulties. The hypothesis that the core phonological deficit in dyslexia is manifested in dysfunctional phonological processes receives support from the finding that reading difficulties and related phonological deficits persist across development, even in people with dyslexia who have compensated for their reading difficulties (Bruck, 1990, 1992, 1993; Hatcher, Snowling, & Griffiths, 2002; Pennington, Orden, Smith, Green, & Haith, 1990). Such findings apply equally to adults with dyslexia who read in transparent languages (Paulesu et al., 2001).

For example, Bruck (1990, 1993) compared college students who had childhood diagnoses of dyslexia with age-matched and reading-level matched (6th graders) control students on measures of word recognition, spelling, and reading comprehension skills, and found that, despite having reading comprehension skills that approximated those of the age-matched control students, the dyslexic students manifested inaccurate and dysfluent word recognition and spelling skills, relative to the age-matched controls. Moreover, the dyslexics used strategies and processes, for both word recognition and spelling, that were immature and more like those of the reading-level matched controls than like those of the age-matched controls. Like the reading-level (sixth grade) controls, the adult with dyslexia relied more than the age-matched controls on spelling-sound information, syllabic information, and use of context for word recognition, and they relied more heavily on spelling-sound information than on visual information, in both word recognition and spelling of irregular words such as *put* and strange words such as *yacht*. This was found to be the case, despite the fact that the dyslexics had deficient letter-sound knowledge (as manifested in poor nonword decoding and poor nonword spelling skills). Evidence that the persistent word recognition and spelling problems of the dyslexic college students were due, in part, to a core phonological deficit is reported in another paper by Bruck (1992), who found that these same students performed significantly below both the age-matched and the reading-level matched controls on a variety of phonological awareness measures.

Bruck's (1990) finding that adults with dyslexia had reasonably good reading comprehension skills, despite dysfluent word recognition skills, is somewhat at variance with conventional views of reading comprehension that are based on the assumption that fluent word recognition is a prerequisite for adequate reading comprehension. To further evaluate this disparity, Bruck divided the dyslexic students into poor and good comprehender groups and found that the good comprehenders had higher childhood IQs and better developed vocabulary knowledge than the poor comprehenders, but no better word recognition skills. Bruck inferred from these results that the superior cognitive abilities of the good comprehenders allowed them to compensate more effectively than the poor comprehenders for their deficient word recognition skills. This interpretation is quite in keeping with the implication from family risk studies that the ability to acquire functional reading skills is determined, in part, by the particular mix of cognitive abilities with which the individual is endowed interacting with unique environmental circumstances that serve to facilitate or impede the acquisition of such skills.

Studies of adult dyslexia conducted by Bruck (1990, 1992, 1993) and others (see above references) provide documentation that the major symptom pattern defining dyslexia, in terms of basic deficits in word recognition and related literacy and language-based skills, persist well into adulthood. To compare competing theories of the etiology of dyslexia and to further examine the core deficit in adults, Ramus et al. (2003) conducted a multiple-case study involving 16 university students identified as dyslexics and 16 controls. The students identified as dyslexics had reading and spelling skills within the normal range, even though their literacy skills were poorer than expected, given their general cognitive ability (IQ). Each student completed a number of different tests assessing auditory, phonological, visual, and motor domains of processing. Phonological deficits on tasks evaluating rapid automatized naming, spoonerisms, and

nonword repetition were observed in all 16 of the students with dyslexia. In addition, 10 of the dyslexic students showed auditory impairments, 4 had problems with motor skills, and 2 showed deficits on visuo-magnocellular tasks.

Auditory deficits were much more common than other forms of sensory impairment in this group, lending face valid credence to Tallal's (1980) hypothesis that language learning and reading difficulties can be traced to auditory processing problems. However, the nature of the auditory deficit varied across these students – some but not all showed problems on rapid auditory processing tasks, and some had problems with basic auditory processing. Others only had problems on speech perception tasks, and six manifested no deficits in any of these cognitive domains. To explain the heterogeneity in these data, the investigators suggested that phonological skills may be involved in 'top-down control' of auditory skills, but auditory processing skills are not universally impaired in dyslexia. We would add, in keeping with a suggestion made by Eden and Zeffiro (1998), that the low-level visual and auditory deficits that were observed in some of the dyslexics in this study were unlikely causes of their lower than expected reading levels, but could have been classified as biological markers signifying functional anomalies, both in areas of the brain that support these sensory processes and in adjacent areas that support reading and language processes.

In sum, results from the life-span dyslexia studies exemplified in the foregoing sections complement results from the genetic studies discussed earlier. Together, these studies provide documentation that reading difficulties in otherwise normal individuals, in many cases, are caused primarily by biologically based cognitive deficits that can be identified quite early in life and that persist well into adulthood. They also provide documentation that there is a gradation of risk for becoming dyslexic, depending on the particular assortment of (reading-related) cognitive abilities with which the individual is endowed and the degree to which the individual's environmental experiences allow him or her to capitalize on cognitive strengths in ways that will compensate for cognitive weaknesses. Thus, the major conclusions that can be reasonably drawn from the life-span dyslexia studies we have discussed accord quite well with the major conclusions that can be reasonably drawn from the genetic studies we have discussed.

Cognitive and biological versus experiential and instructional causes of early reading difficulties

Specific reading disability, as an etiological construct, incorporates the assumption that children who qualify for this diagnosis on the basis of psychometric and exclusionary criteria such as those outlined earlier suffer from basic cognitive deficits of biological origin. Empirical support for this possibility is seminal and suggestive, if not always conclusive. As we have discussed, such support comes from results obtained in genetic, neuroanatomical, and psycho-physiological studies, which, collectively, provide some reason to believe that poor and normal readers have structurally and functionally different architecture for processing spoken and written language (Grigorenko, 2001; Lyon et al., 2001). However, as pointed out by Clay (1987), virtually all reading disability research has been compromised by the failure to control for the child's educational history, given that the adverse effects of inadequate pre-reading experience and/or inadequate instruction can often lead to reading skills deficiencies that mimic the effects of basic cognitive deficits. Consider, for example, that the acquisition of skills such as phonological awareness and letter-sound decoding can be greatly influenced by the type of reading instruction to which a child has been exposed. Yet, it is commonly assumed that difficulties in acquiring one or both of these skills are a manifestation of basic deficits in phonological coding. Indeed, it is possible (and we think highly likely) that many of the children placed in single deficit (or even double deficit) sub-categories in recent studies evaluating the double deficit theory of reading disability (e.g., Wolf, Bower, & Biddle, 2000a) were children whose reading difficulties were caused primarily by inadequate instruction. Similarly, IQ scores are typically used to help diagnose specific reading disability. Most intelligence tests, however, contain tasks and items that depend heavily on the acquisition of knowledge and skills, like vocabulary knowledge and domain-general knowledge, that are acquired in part through reading. As a consequence, intelligence in children having long-standing reading difficulties may be underestimated, particularly as they grow older (Ackerman, Weir, Holloway, & Dykman, 1995; Shaywitz et al., 1995; Stanovich, 1986; Vellutino et al., 1988, 1995b). Thus, there is a pressing need for research that facilitates identification of markers that would aid in distinguishing between children whose reading problems are caused primarily by cognitive deficits of biological origin and those whose reading problems are caused primarily by limitations in pre-reading experience and/or inadequate instruction.

One such study was recently reported by Vellutino, Scanlon, and their associates (Vellutino et al., 1996). This was a longitudinal study that incorporated an intervention component (daily one-to-one tutoring) to distinguish between cognitively versus experientially impaired readers. Thus, children classified in first grade as impaired or normally developing readers were tracked from the time they entered kindergarten through the end of fourth grade, that is, before and after they were classified, and before and after intervention. Inter-

vention was initiated in mid-first grade, and was terminated either at the end of first grade or in the middle of second grade, depending on the child's progress. Given results obtained in previous intervention studies (Clay, 1985; Iversen & Tunmer, 1993; Pinnell, 1989; Wasik & Slavin, 1993), it was expected that most, but not all of the tutored children would be successfully remediated, and the investigators were especially interested in comparing the entry-level skills and cognitive profiles of children who were the most difficult to remediate, with those of children who were readily remediated, relative to normally achieving readers. It was also expected that the entry-level skills of the impaired readers would be uniformly deficient. And from the convergent evidence implicating phonological coding deficits as a basic cause of reading difficulties in some impaired readers, it was anticipated that the children who were the most difficult to remediate would perform below the children who were readily remediated on measures evaluating cognitive abilities such as phonological awareness, verbal memory, and rapid naming. These groups were not expected to differ on measures evaluating semantic, syntactic, and visual skills. These predictions generally were confirmed.

First, it was found that relative to normally achieving readers, entry-level literacy skills such as letter naming and phonological awareness were deficient in the group of kindergarten children identified as impaired readers in first grade. Second, 67.1% of the tutored children were brought to within the average range of reading ability in only one semester of remediation, and the majority maintained this level of functioning through the end of fourth grade (see Figures 3 and 4). Third, the children who were found to be the most difficult to remediate performed well below the normal readers, and quite often below the children who were readily remediated on kindergarten, first, and third grade tests evaluating phonological abilities such as those mentioned previously. In addition, the children who were readily remediated often performed as well as the normal readers on such tests. However, there were no significant differences among any of the groups on the semantic, syntactic, and visual measures, although mean effect sizes tended to be negative, thereby reflecting a general trend, on the

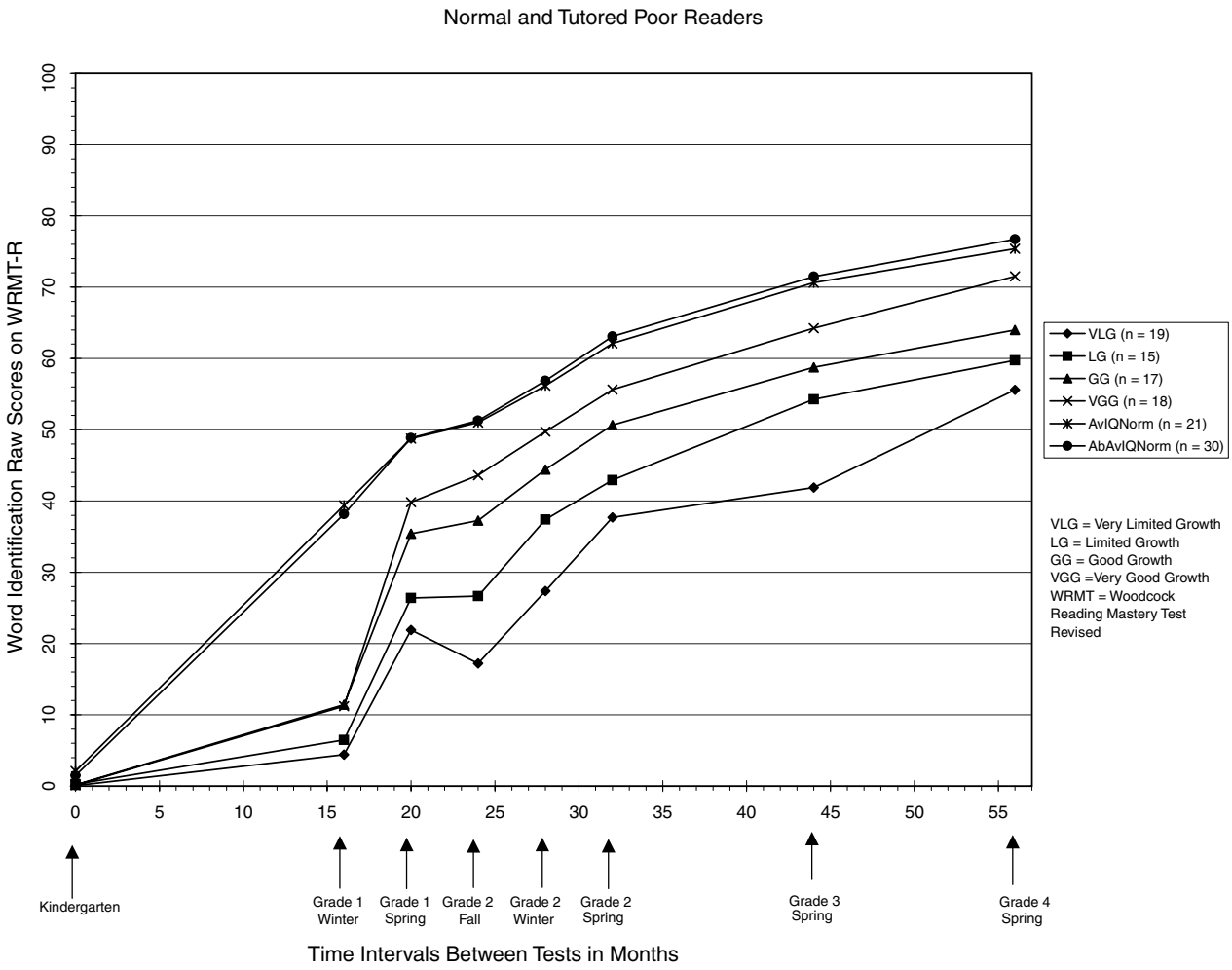

Figure 3 Growth curves for mean raw scores on the WRMT-R word identification subtest for normal and tutored poor readers

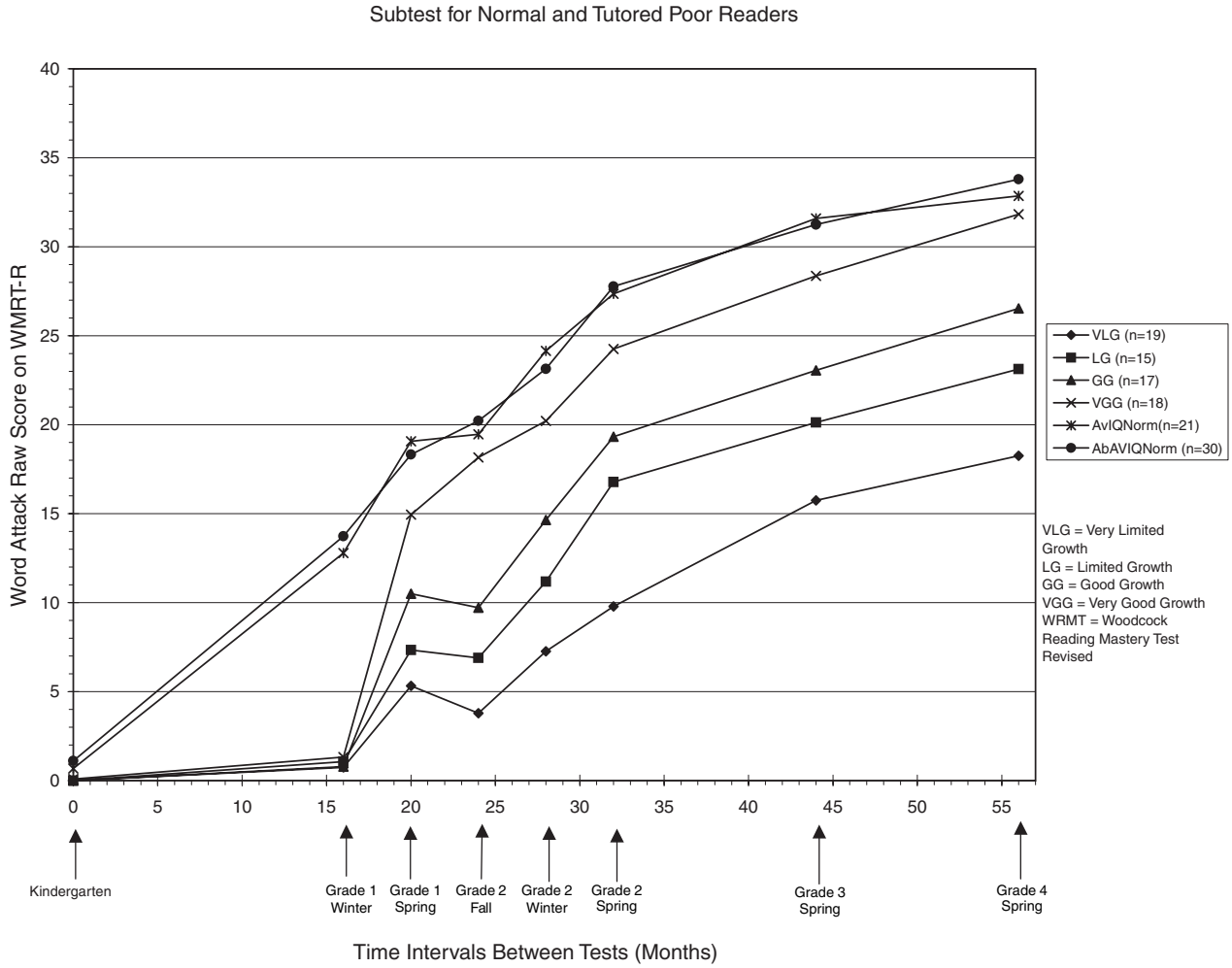

Figure 4 Growth curves for mean raw scores on the WRMT-R word attack subtest for normal and tutored poor readers

part of the impaired readers, to perform below the level of the normal readers on these measures as well as on the phonological measures. Because the normal readers in this study generally scored above national norms on the reading measures, the negative effect sizes on the semantic, syntactic, and visual measures were taken as an indication that they were characterized by a more 'optimal mix' of reading-related cognitive abilities than were the impaired readers, rather than an indication that the cognitive abilities evaluated by these measures were seriously deficient in the impaired readers (Vellutino et al., 1996). This analysis is more in keeping with what we have called 'gradation of risk', continuous ability type conceptualizations of dyslexia (Olson & Gayan, 2001; Pennington & Lefly, 2001; Snowling et al., 2003; Stanovich, 1988; Vellutino et al., 1996) than with what we have called 'all or none' type conceptualizations of dyslexia, as discussed in earlier sections of this paper.

Finally, contraindicating the use of IQ scores to identify disabled readers or to predict reading achievement in beginning readers, the investigators found that the tutored groups did not differ on any of the intelligence tests, nor did they differ from an average IQ normal reader group on these tests. Moreover, the average IQ normal reader group did not differ from an above average IQ normal reader group on any of the reading tests, except for tests of reading comprehension, on which the above average IQ group performed at significantly higher levels than the average IQ group (and, of course, at higher levels than the tutored groups). In addition, IQ-achievement discrepancy scores were not significantly correlated with initial growth in reading performance following one semester of one-to-one tutoring (Vellutino, Scanlon, & Lyon, 2000; see also Share, McGee, & Silva, 1989). Presently, there is a large body of research showing that children with IQ discrepant and IQ non-discrepant reading scores cannot be adequately differentiated, vis-à-vis response to instruction or prognosis (see Francis, Fowler, & Shaywitz, 1994; Fletcher et al., 1994, 2002; Lyon et al., 2001, 2002; Vellutino, Scanlon, & Lyon, 2000). There are also two recent meta-analyses showing null to small differences between the cognitive skills of these two populations (Hoskyn & Swanson, 2000; Steubing et al., 2002).

These results are quite in keeping with Clay's (1987) contention that reading difficulties in beginning readers are, in most cases, caused primarily by experiential and/or instructional deficits. Indeed, the impaired reader sample initially identified in first grade, using exclusionary criteria such as those typically used to identify 'disabled readers' in such research, represented approximately 9% of the (available) population from which these children were drawn. Yet, the impaired readers who continued to qualify for this diagnosis after only one semester of remediation represented only 1.5% of the population from which these children were drawn, which is a far cry from the 10% to 15% figures that have emerged as estimates of the incidence of reading disability in the relevant literature (e.g., Shaywitz et al., 1992; Harris & Sipay, 1990).

Additional support for the contention that reading difficulties in many children are caused primarily by experiential and/or instructional deficits comes from other intervention studies which show that most impaired readers can acquire at least grade-level reading skills if they are identified early and are provided with comprehensive and intensive reading instruction tailored to their individual needs (Clay, 1985; Iversen & Tunmer, 1993; Pinnell, 1989; Scanlon et al., 2000; Torgesen et al., 1999; Wasik & Slavin, 1993). Similarly, recent classroom observation and classroom intervention studies (Foorman et al., 1991; Foorman et al., 1998; Scanlon & Vellutino, 1996) have shown that comprehensive and well-balanced reading instruction that facilitates the acquisition of phonological awareness and letter-sound decoding skills along with other word-level skills, in addition to oral language and reading comprehension skills, can prevent long-term reading difficulties in children who would otherwise qualify for a diagnosis of reading disability. Nevertheless, the observation of reading-related cognitive deficits in the poor readers who were found to be difficult to remediate, relative to both the normal readers and the poor readers who were found to be readily remediated, suggests that reading difficulties in some impaired readers may be caused, in part, by basic cognitive deficits of biological origin. Thus, additional research designed to trace the ultimate origins of such deficits is certainly warranted.

Implications for practitioners

The knowledge acquired from the research reviewed in this paper has several important implications for practitioners involved in diagnosing specific reading disability (dyslexia), especially those who work in school systems and serve as consultants to teachers and other school officials responsible for educational and remedial planning. First, the research questions the utility of psychometric assessment as the sole or even primary vehicle for determining the origin of reading difficulties for purposes of educational and remedial planning. In many instances, this enterprise is motivated by what some have called 'a search for pathology' (Ysseldyke & Christensons, 1988) – that is, assessment to determine what we have called underlying (cognitive and biological) rather than manifest causes of a child's reading difficulties in terms of poorly developed reading subskills. This enterprise is typically implemented for purposes of classification associated with official mandates requiring such classification, such as categorical funding for 'learning disabled' children versus 'socioeconomically disadvantaged' children. Accordingly, the child is given an individually administered battery of tests that typically include an intelligence test, one or more measures of reading achievement, and a variety of measures to evaluate reading-related cognitive abilities. Other 'exclusionary' criteria are typically employed to rule out extraneous factors as causes of the child's reading difficulties, for example, uncorrected sensory deficits, emotional disorder, frequent absences from school, and/or socioeconomic disadvantage. The end result of this process is that the child's reading difficulties, in most cases, are attributed to a basic cognitive deficit of one description or another, which, in turn, tends to be attributed to some sort of neuro-developmental anomaly.

Yet, as pointed out by Clay (1987), psychometric/exclusionary approaches to assessment do not control for the child's educational history and early literacy experiences. And as we have seen from intervention studies such as those just discussed, there is strong evidence that most early reading difficulties are caused primarily by experiential and instructional deficits, rather than basic cognitive deficits associated with neuro-developmental anomaly. Moreover, despite the progress made in identifying what we have called manifest and underlying causes of difficulties in learning to read, it should also be clear from the research reviewed herein that there is no uniform consensus as to what might be called the 'ultimate' or neuro-biological cause(s) of specific reading disability and controversy abounds in this area of inquiry. Although some etiological theories enjoy greater currency than others, none of these theories yet provides a clear-cut, definitive, and unequivocal set of diagnostic criteria that would pinpoint the ultimate (neuro-biological) origin of the child's reading difficulties in the same way that a biopsy pinpoints the type and locus of a physical disease such as cancer. As a consequence, the approach to diagnosing the underlying cause(s) of a child's reading difficulties tends to vary with the training and/or conceptual biases of the clinician. The reports written by these clinicians often have little prescriptive value vis-à-vis educational or remedial planning. Thus, given the state of the art, it could be argued with some legitimacy that psycho-

metric assessment performed by practitioners for the sole purpose of 'diagnosing' reading disability is a questionable enterprise that might better be abandoned. This, of course, would mean that official mandates, which require traditional diagnostic assessments for purposes of official classification, should also be abandoned.

A related problem underscored by current research – one that also has important implications for practitioners – has to do with current psychometric-exclusionary approaches to diagnosing specific reading disability that adopt the IQ–achievement discrepancy as their central defining criterion. In most venues and locales, specific reading disability is defined on the basis of a significant discrepancy between a child's expected reading achievement, as predicted by that child's IQ, and his or her actual achievement, as defined by a score on a standardized test of word identification and/or other word-level skills (see Lyon et al., 2002 and Vellutino et al., 2000 for historical reviews of this practice). Aside from the obvious fact that exclusionary factors discussed above provide no clear-cut criteria for identifying either manifest or underlying causes of a child's reading difficulties, it should be apparent, from the results obtained in the intervention study conducted by Vellutino et al. (1996), that the IQ–achievement discrepancy criterion is not a sufficiently precise metric to warrant its continued use to define specific reading disability. In this study, intelligence test scores did not distinguish between poor and normally achieving readers or between poor readers who were difficult to remediate and those who were readily remediated. They also failed to predict word-reading performance in normally achieving readers. Moreover, IQ–achievement discrepancy scores did not predict initial growth in reading in poor readers who received intensive remediation.

Altogether, the evidence supporting the use of IQ–achievement discrepancy classifications models is quite limited (Fletcher et al., 2002; Hoskyn & Swanson, 2000; Lyon et al., 2001, 2002; Stuebing et al., 2002; Vellutino et al., 2000; see also Share et al., 1989; Siegel, 1988, 1989). Thus, it is apparent that a child may need little more than average or even low average intelligence to learn to decipher print. Some scholars even assert that it has no relevance at all (e.g., Siegel, 1988, 1989). Although this may be an extreme view, the fact remains that in any child learning to read in an alphabetically based orthography, regardless of level of intelligence, the ability to learn to decode print will be determined primarily by phonological skills such as phonological awareness, facility in alphabetic mapping, name encoding and retrieval, and verbal memory. Indeed, how else would we account for hyperlexia (i.e., atypically strong decoding ability), which occurs sometimes even in children with mental deficiency. Thus, if our analysis proves to be correct, not only would the IQ–achievement discrepancy to define reading disability be invalidated, there would be no role for IQ tests in this enterprise. Accordingly, practitioners would be well advised to abandon the use of such measures for children with word-level reading disabilities, which, of course, constitute the core deficit in dyslexic individuals. This suggestion does not discount the utility of using intelligence tests (among other relevant measures) to aid in diagnosing the origin of reading comprehension difficulties in children with adequate word-level skills, especially intelligence tests that evaluate verbal abilities and other cognitive abilities that are entailed in comprehending linguistic text, for example verbal reasoning, inferencing, and logical deduction. Such higher-level intellectual skills are not entailed, however, in learning to decode print, in keeping with our contention that intelligence tests have little utility for diagnosing specific reading disability.

Still another problem with currently employed psychometric approaches to assessing the origin of a child's reading difficulties is that they typically provide no direction for educational or remedial planning (Lyon & Moats, 1988, 1993). This problem occurs, not only because such approaches tend to focus on cognitive and biological rather than manifest causes of a child's reading difficulties, but also because the clinicians performing such assessments tend to have limited background and expertise for diagnosing and remediating deficiencies in foundational reading subskills such as phonological awareness, word identification, alphabetic mapping, and language comprehension (Fish & Margolis, 1988). Consequently, their role in assisting educators in educational and remedial planning is limited to the administration of norm-referenced tests, which typically provide little that is of practical value for purposes of remedial instruction. And because of the lack of expertise in educational and remedial planning characteristic of clinicians working in school settings (and elsewhere), such assessments tend to be 'static' rather than 'dynamic' insofar as they evaluate existing abilities in terms of a child's relative position in a normative population and provide little or no information about the type of instruction that might be most effectively tailored to the child's individual needs (Reschly, Tilly, & Grimes, 1999).

This state of affairs underscores perhaps the most general implication of the reading disability research reviewed herein: specifically, the need for a radical change in the perceived and implemented role of clinical assessment in diagnosing and remediating reading difficulties. Rather than select psychometric tests for purposes of evaluating cognitive abilities that underlie reading ability, in the interest of detecting underlying (cognitive and biological) causes of a child's reading difficulties for purposes of categorical labeling (e.g., 'specific reading disability', 'attention deficit disorder', etc.), the clinician would more profitably select psychometric tests that have

demonstrated validity for assessing strengths and weaknesses in reading subskills (what we have termed 'manifest causes' of reading difficulties) for purposes of developing an appropriate educational plan tailored to the child's individualized needs. Moreover, such tests should be based on criterion-referenced standards derived from a thorough understanding of the components of reading ability, rather than norm-reference standards based exclusively on placement in a normative distribution.

Results from current intervention studies suggest that the most informative and most effective approach to distinguishing between cognitive/biological and experiential/instructional causes of early reading difficulties would be to implement an initial period of remedial intervention, in lieu of assessment of reading-related cognitive abilities as a 'first cut' approach to diagnosis. Accordingly, psychometric assessment of strengths and weaknesses in a child's reading subskills would not be used to classify that child as 'reading-disabled' at this stage of the diagnostic process, but, rather to provide the educator with guidance for purposes of initiating remedial instruction. This approach would not only evaluate the child's existing skills and abilities to insure individualized and well-balanced instruction, but would also evaluate the child's initial response to remediation to aid in determining whether his or her reading difficulties are caused primarily by instructional and experiential deficits. Assessment of strengths and weaknesses in reading-related cognitive abilities could thereafter be implemented to cross-validate initial impressions derived from the child's initial response to remediation. Recall that in the Vellutino et al. (1996) intervention study discussed earlier, the cognitive profiles of children who were found to be readily remediated were closer to those of normally achieving readers than were the cognitive profiles of those who were found to be difficult to remediate. Thus, assessment of reading-related cognitive abilities for cross-validation purposes would appear to be useful.

However, the primary purpose of these complementary approaches to assessment would be to develop a long-range remedial plan that would facilitate acquisition of functional reading skills, rather than categorical labeling. But, if such labeling is necessary, then the implication of the approach to assessment we are suggesting is that the assignment of a categorical label be deferred until an attempt is made to remediate the child's reading difficulties (see Abbott, Reed, Abbott, & Berninger, 1997 and Reschly et al., 1999 for additional support for this approach to diagnostic assessment). The net effect of this approach would be to shift our conceptualization of dyslexia away from scores on a set of tests toward response to intervention as the primary means for defining the disorder. From a policy standpoint, identification of children who might be eligible for special educational services would focus on the child who has not demonstrated the accelerated growth in reading skills that characterizes many poor readers who receive intensive intervention. Without this component, it becomes impossible to distinguish those with a true disability from those who are instructional casualties.

Finally, it should be clear that clinical practitioners working with children who have reading difficulties should reset their priorities and shift the focus of their clinical activities so as to place much greater emphasis on instructional and remedial activities and much less emphasis on psychometric activities in their work with reading impaired children. We suspect that this shift would not only enhance their acumen in clinical diagnosis, but, more importantly, would facilitate the development of effective educational programs for correcting reading difficulties in these children. Such a shift in focus will require that the practitioner update and/or upgrade her or his knowledge base so as to learn more about the reading process and reading development, and focus on instructional factors that would facilitate or impair such development. In other words, the most effective practitioner would not simply be a psychometrician, as is true of many practitioners working with reading impaired individuals, but would also be a recognized expert in the psychology of reading and reading development, the psychology of reading disability, and the psychology of reading instruction.

Summary and conclusions

We have learned much about manifest and underlying causes of reading difficulties in otherwise normal children over the past four decades. It is clear from the relevant research that reading is primarily a linguistic skill, contrary to the once popular notion that it is primarily a visual skill. And, because of the structural properties of an alphabetic system, it is also clear that linguistic abilities are themselves differentially weighted in reading development such that phonological skills carry greater weight as determinants of beginning reading ability than do semantic and syntactic skills, whereas semantic and syntactic skills carry greater weight than do phonological skills in more advanced readers. It follows, as the evidence confirms, that inadequate facility in word identification constitutes the manifest and most ubiquitous cause of reading difficulties. Moreover, there is reliable and highly convergent evidence that word identification problems, themselves, are causally related to deficiencies in phonological awareness, alphabetic mapping, and phonological decoding that lead to difficulties in establishing connective bonds between a word's spoken and written counterparts. However, causal relationships between word identification problems and deficiencies in such phonological skills are more prominent

in dyslexics learning to read in opaque orthographies such as written English than in dyslexics learning to read in more transparent orthographies such as German or Italian. In transparent orthographies such as these, word identification problems are more often associated with deficiencies in implicit phonological processes (e.g., verbal memory, name retrieval, etc.) that impair fluency in word identification and text processing, and, thereby, reading comprehension.

As regards underlying causes, the research strongly suggests that reading difficulties in most children are caused by deficits in phonological coding. Such deficits are believed to account for the reliable and robust differences observed between poor and normal readers on measures of phonologically based skills such as phonological awareness, alphabetic mapping, phonological decoding, verbal memory, and name encoding and retrieval. However, some researchers suggest that dysfluency in name retrieval is caused by a hypothesized timing deficit that impairs temporal integration of a word's component letters, but the evidence to support this hypothesis is tenuous and the issue has become controversial. Semantic and syntactic deficits, in most cases, do not appear to be a primary cause of reading difficulties in otherwise normal children, but they are a likely consequence of long-standing reading disorder or a co-morbid oral language disorder. They may, however, be a primary cause of early reading difficulties in some children, especially those from disadvantaged or bilingual populations.

Reading disability research has also established that reading difficulties are not caused by visual deficits of the types most often proposed over the years. Contrary to popular belief, impaired readers do not see letters and words in reverse, nor do they suffer from inherent spatial confusion or other visual anomalies of the types proposed in the early literature. More recent research provides suggestive evidence that some poor readers may suffer from low-level sensory deficits in both the visual and auditory spheres, but the evidence is inconclusive, and in, some instances, equivocal and controversial. Moreover, no causal relationships have been established between such deficits and difficulties in learning to read.

Similarly, there is no reason to believe that deficiencies in general learning abilities such as attention, association learning, cross-modal transfer, serial memory, pattern analysis, and rule learning are basic causes of reading difficulties in impaired readers who do not have general learning difficulties. Etiological theories, which implicate deficits in such abilities as causally related to reading difficulties, can be ruled out on logical grounds alone and they have not fared well in empirical research.[3]

Finally, recent intervention studies have clearly demonstrated that reading difficulties in most beginning readers are not invariably caused by basic cognitive deficits of biological origin, from which it can be concluded that current estimates of the incidence of true reading disabilities are greatly inflated. However, recent studies of neurological and genetic correlates of dyslexia, along with recent life-span development and intervention studies, provide strong reason to believe that a very small percentage of impaired readers may well be afflicted by basic cognitive deficits of biological origin, especially phonological deficits that lie at the root of their difficulties in learning to read. These and other findings we have discussed have obvious implications for the diagnosis and remediation of reading disability, the most general implication being the need for practitioners to shift the focus of their clinical activities away from emphasis on psychometric assessment to detect cognitive and biological causes of a child's reading difficulties for purposes of categorical labeling in favor of assessment that would eventuate in educational and remedial activities tailored to the child's individual needs. It was suggested that a 'first cut' approach to such assessment should entail well-balanced and individualized remedial intervention that would build upon the child's existing knowledge base. The evidence suggests that a child's response to this type of intervention would provide guidance as to his or her long-term instructional needs, regardless of the origin of his or her reading difficulties.

Acknowledgments

Much of the work discussed in this paper 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. Martha Bridge Denckla was the principal investigator overseeing the various research projects initiated under this grant. Several grants involving Fletcher supported this review, including P50 HD21888, R01 HD35938, and NSF 9979968.

[3] These assertions in no way discount the possibility that reading difficulties could conceivably arise because of deficits in general learning abilities such as selective attention, associative learning, and cross-modal transfer. Indeed, several studies have demonstrated that some children who have difficulties learning to read suffer from both phonological and attention deficits (e.g., Fletcher, Shaywitz, & Shaywitz, 1999), and such co-morbidity would be expected in children who have multiple handicaps. These appear largely a matter of the child having two problems as opposed to the hypothesis that the reading difficulties are a probable consequence of such co-morbidity.

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Manuscript accepted 18 August 2003