Journal of Developmental and Physical Disabilities, Vol, 12, No. 2, 2000

Visual and Auditory Processing Impairments in Subtypes of Developmental Dyslexia: A Discussion

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There has been a large body of research exploring sensory processing deficits in dyslexia, in both the visual and the auditory domains. Recently, there has also been evidence to suggest that dyslexia may be a heterogeneous disorder, with different patterns of dyslexia being identifiable. In this paper, we examine the relationship between these two bodies of research. First, we briefly review the evidence for sensory processing impairments in dyslexia, in both the visual and the auditory domains. Second, we consider how such deficits might affect the development of different component processes in reading and, therefore, be associated with different subtypes of dyslexia. Finally, we present some illustrative data, which points to the importance of considering different component processes of reading when investigating sensory processing deficits in dyslexia.

KEY WORDS: visual processing; auditory processing; temporal processing; dyslexia subtypes.

Dyslexia, or specific reading disability, is generally defined as the failure to acquire reading proficiency despite an adequate level of intelligence, normal hearing sensitivity and visual acuity, a supportive learning environment, and an absence of behavioral problems (Critchley, 1970). Most estimates of the prevalence of this disorder suggest that it affects approximately 4–10% of the population, although some figures reach as high as 20–25% (Rutter, 1978; Shaywitz *et al.,* 1990). Given the primacy of literacy skills for successful functioning in a modern, technological society, there is no

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1056-263X/00/0600-0145$18.00/0 © 2000 Plenum Publishing Corporation

question that people with this disorder are at a considerable disadvantage. For this reason, a large amount of research attention has focused on exploring the nature and causes of specific reading problems.

Two important developments in our understanding of this disability have been (a) that dyslexia may be associated with sensory processing deficits other than those which may be detected through routine tests of visual acuity or auditory sensitivity and (b) that dyslexia may not be a unitary disorder—there are several component processes involved in reading and varieties of dyslexia may be identified based on selective deficits in these component processes. While research in these two domains has proceeded rapidly in parallel, few attempts have been made to explore the relationship between the two. Are sensory processing deficits associated with all forms of dyslexia or do they occur only in specific varieties of reading disorder? More specifically, what is the mechanism by which sensory processing deficits might impair development of component processes in reading?

The aims of this paper are threefold. First, we briefly review the evidence for sensory processing impairments in dyslexia, in both the visual and the auditory domains. Second, we consider how such deficits might affect the development of different component processes in reading, as represented by different subtypes of dyslexia. Finally, we present some illustrative data, which points to the importance of systematically exploring the relationship between these important psychophysical and psycholinguistic variables.

VISUAL PROCESSING IN DYSLEXIA

There has been a large amount of research exploring visual deficits in dyslexia. Early work focused primarily on "higher" visual processes, such as visual form perception, since such processes were thought most likely to be implicated in a complex cognitive skill such as reading. However, this research did not reveal clear deficits in dyslexics as compared to normal readers (Vellutino, 1979).

More recently, attention has turned to early sensory processes in vision. Here, evidence for a visual processing deficit in dyslexia has been much more robust. A brief description of the dominant psychophysical model of the early visual system is required to explain the latter results (e.g., Breitmeyer and Ganz, 1976). This model postulates two parallel subsystems or channels, which are anatomically segregated into the magnocellular and parvocellular layers of the lateral geniculate nucleus (Kaplan *et al.,* 1991). The two systems are tuned to respond to different spatial and temporal frequencies. The parvocellular system is most sensitive at high spatial fre-

quencies (i.e., fine patterns) and has a low temporal resolution and a long response persistence, while the magnocellular system is most sensitive at low spatial frequencies (i.e., coarse patterns), has a high temporal resolution, and responds transiently to sudden changes in the visual input.

Within this framework, Lovegrove and colleagues have proposed that dyslexics have a deficit in the magnocellular channel of the visual system, which is responsible for rapid temporal visual processing (for reviews, see Lovegrove, 1996; Stuart and Lovegrove, 1992). Using measurements of threshold luminance contrast, they reported several studies indicating that dyslexics had more difficulty than normal readers in detecting stimuli which flicker at rapid rates (Lovegrove *et al.,* 1980, 1982, Martin and Lovegrove, 1984, 1987). Conversely, dyslexics had normal or near-normal performance in detecting static patterns which were gradually turned on and off to avoid sharp transients. This is the pattern that would be expected from an impairment to the magnocellular system: monkeys with selective lesions of the magnocellular layers of the lateral geniculate nucleus have been shown to be completely blind to rapidly flickering stimuli but to have normal detection of static patterns (Merigan and Maunsell, 1993). Also consistent with the magnocellular deficit hypothesis is preliminary evidence from a small number of human postmortem studies that dyslexics do in fact have abnormalities in the magnocellular layers of the lateral geniculate nucleus (Livingstone *et al.,* 1991).

Several possible mechanisms by which visual sensory processing deficits might influence reading have been forwarded. An early theory proposed that magnocellular deficits impair saccadic suppression and make reading of connected text difficult (Breitmeyer and Ganz, 1976). Another hypothesis postulates that visual magnocellular deficits lead to unstable binocular fixation, which, in turn, makes reading uncomfortable (Stein, 1996); this discomfort might lead to reading avoidance and therefore to impaired development of word recognition skills.

AUDITORY PROCESSING IN DYSLEXIA

Some evidence has also been reported for auditory sensory processing deficits in dyslexia. Using a gap detection task, McCroskey and Kidder (1980) found that reading-disabled children needed longer inter-stimulus intervals to separate two sounds than did normal readers. Dyslexics have also been found to be less sensitive than normal readers to changes in amplitude (McAnally and Stein, 1997; Menell *et al.,* 1999) and frequency (McAnally and Stein, 1996; Witton *et al.,* 1998) of acoustic stimuli. In addition, a physiological response to frequency change (the mismatch nega-

tivity) is delayed in dyslexics (Watkins *et al.,* 1995). Based on the results of a more complex temporal order judgment task, Tallal (1980) has reported that dyslexics are impaired relative to younger normal readers in determining whether two tones presented in rapid succession are the same or different. At an anatomical level, Galaburda *et al.* (1994) have also reported that, in dyslexics, the left medial geniculate nucleus (the auditory thalamus) contains more small cells than in normal controls.

The dominant theory of the mechanism by which auditory sensory processing deficits might cause dyslexia is via their effect on the perception of speech signals (see, e.g., Tallal *et al.,* 1996a). Auditory sensory deficits are proposed to cause impaired speech perception: there is evidence that dyslexics categorize speech stimuli less well than normal readers (Godfrey *et al.,* 1981; Steffens *et al.,* 1992; Werker and Teas, 1987) and that their physiological responses to speech stimuli are different from those of control listeners (Schulte-Körne *et al.,* 1998). Such speech perception deficits may, in turn, lead to deficits in the ability to process and manipulate speech sounds or *phonemic awareness* deficits. Phonemic awareness problems are proposed subsequently to result in difficulties in learning letter–sound correspondences during the process of reading development (Bradley and Bryant, 1983; Liberman and Shankweiler, 1985).

A GENERAL SENSORY PROCESSING DEFICIT?

As a result of the findings in these separate modalities, there have been several recent suggestions that dyslexia may be attributable to a *general* sensory processing deficit, which occurs across auditory and visual modalities and possibly other modalities as well (Farmer and Klein, 1995; Stein, 1996; Stein and Walsh, 1997; Tallal *et al.,* 1996a). The most frequently forwarded theory of the mechanism by which this proposed general deficit produces reading difficulties is as follows: dyslexics have a defect (either congenital, developmental, or acquired) in magnocellular or homologous large neurons within the central nervous system, which results in impairments to auditory and visual sensory processing. Although deficits in both of these modalities occur, it is the auditory deficit that carries the weight, as it produces the speech perception and subsequent language and reading impairments described above. Visual temporal processing deficits do not, in themselves, play a causal role in dyslexia but are simply another manifestation of the general sensory impairment. Support for the theory that a general deficit operates via the auditory pathway, and that visual deficits serve as a marker for this deficit, has come from findings that both auditory

(Tallal *et al.,* 1996b) and visual (Lovegrove *et al.,* 1988) sensory impairments correlate highly with phonemic awareness problems.

A general theory is appealing as it presents, for the first time, an opportunity to reconcile the diverse range of findings regarding visual, auditory, and phonemic processing in dyslexia. However, it has received little in the way of direct examination as, to date, sensory processing has rarely been examined across the visual and auditory modalities within the same people. The one study that has been conducted to date was supportive of the hypothesis of a general deficit: Witton *et al.* (1998) examined thresholds to visual coherent motion and frequency modulation (i.e., motion across the sensory epithelium of the cochlea) in the same people and found a high correlation between thresholds in each modality. As well as the need for further within-subject studies, the question of the relationship between sensory processing deficits and component processes in reading, as reflected by different varieties of dyslexia, remains to be comprehensively addressed.

VARIETIES OF DYSLEXIA

Not all studies have reported evidence for sensory processing deficits in dyslexia. In the visual domain, several researchers have failed to replicate the findings of Lovegrove and colleagues (Gross-Glenn *et al.,* 1995; Walther-Müller, 1995; Cornelissen *et al.,* 1995). As both Hogben (1996) and Slaghuis and Pinkus (1993) have pointed out, this inconsistency in results may be attributable to the enormous variability in the methods used for selecting dyslexic participants for visual processing studies. In some cases, the criteria used have simply not been specified (e.g., Gross-Glenn *et al.,* 1995). The study by Walther-Müller (1995) applied rigorous criteria, but the language used was German, which has a more regular orthographic structure than English. Therefore, it is possible that participants included in studies which have failed to report magnocellular visual system deficits in dyslexia were quite different from those used in studies which have reported a positive finding.

In the auditory domain, there have also been failures to report group differences on several measures, including temporal resolution (McAnally and Stein, 1996), frequency discrimination (Watson, 1992), and physiological responses to frequency change (Schulte-Körne *et al.,* 1998). With regard to temporal order judgment tasks, there has been some controversy about the validity of the tasks used for the assessment of low-level auditory function (see Studdert-Kennedy and Mody, 1995). In addition, once again there are issues of subject selection: most research employing temporal order judgments has investigated language-impaired (e.g., Tallal and Piercy,

1973, 1974, 1975; Tallal and Stark, 1981; Merzenich *et al.,* 1996; Tallal *et al.,* 1996b; Wright *et al.,* 1997) or, less selectively, ''learning-impaired'' participants (Kraus *et al.,* 1996). These children are likely to have poor phonemic awareness skills and also to be dyslexic, but, contrary to the commentary by Barinaga (1996), there are many dyslexic people who do not exhibit spoken language problems and who may not share these auditory deficits. In summary, across both the visual and the auditory domains, it would seem that more precise selection of participants is needed if the inconsistencies in the data on sensory functioning in dyslexia are to be resolved.

The need for careful selection of participants becomes even more apparent in the light of research suggesting that dyslexia may not be a unitary disorder. Many reading researchers have questioned the notion that developmental reading disorders occur in only one form. A complex and multifaceted process such as reading, it has been argued, will surely be likely to fail in an equally complex and multifaceted range of ways. Consequently, there has been a relatively long history of attempts to classify reading disorders into different categories or ''subtypes'' (e.g., Boder, 1971; Fletcher and Morris, 1986). As Stanovich *et al.* (1997) have recently noted, much of the earlier work in subtyping was somewhat disappointing, since it was not based on explicit models of the reading system and therefore tended to be descriptive rather than explanatory. However, more recently, researchers have made predictions about patterns of developmental reading disorder based on precise and, in some cases, fully computationally implemented models of the skilled reading system (Castles and Coltheart, 1993; Coltheart *et al.,* 1993; Manis *et al.,* 1996; Plaut *et al.,* 1996). This has led to renewed interest in different patterns of dyslexia and in the possible differential etiology of the processing deficits which underlie them.

A recent example of theory-driven research into subtypes of dyslexia is the large-scale study by Castles and Coltheart (1993). This work was based on a dual-route model of normal reading which proposes that skilled readers have at their disposal two, at least partially independent, processes for reading aloud: a *lexical* or word-specific process, which involves gaining access to mental representations of whole words with which a reader is familiar, and a *nonlexical* process, which involves using a system of rules to convert letters into sounds (Coltheart *et al.,* 1993). Castles and Coltheart reasoned that, if these two processes indeed function independently in the skilled reading system, they should be capable of being separately developed in children learning to read. Therefore, two patterns of reading disorder should be able to be identified: developmental *phonological* dyslexia, where there has been a selective deficit in developing the nonlexical procedure, and developmental *surface* dyslexia, where there has been a selective

deficit in acquiring the lexical procedure. The former would be able to be identified through specifically poor nonword reading (e.g., *giph*), since such items are presumed to require the use of conversion rules, while the latter would be identified through specifically poor irregular word reading (e.g., *yacht*), since these items are thought to require access to word-specific information.

Castles and Coltheart (1993) tested a sample of 53 poor readers on their ability to read aloud sets of irregular words and nonwords. Based on their scores on these tasks, eight participants were identified as pure developmental phonological dyslexics: their nonword reading was poor, compared with that of chronological age-matched controls, but their irregular word reading was within normal range. A further 10 participants were classified as pure developmental surface dyslexics: their exception word reading was poor but their nonword reading fell within normal range. A further 27 participants were poor on both tasks, and were therefore not classified as ''pure'' cases, but nevertheless showed a significant discrepancy between their scores on the exception word and nonword tasks. Castles and Coltheart concluded that these results were best interpreted in terms of a dual-route model, with the subtype profiles representing different levels of development of the lexical and nonlexical procedures. Other, similar, subgroups have been identified using slightly different measures (e.g., Boder, 1971).

There are reasons to suspect that dyslexics who differ in their mastery of the lexical and nonlexical component processes of reading may also differ in the likelihood that they will show a general sensory processing deficit. Poor readers with impairments in phonological skills, who have difficulty converting letters into sounds, have also been shown to display phonemic awareness impairments (Campbell and Butterworth, 1985; Rack *et al.,* 1992). As outlined above, it is these phonemic awareness deficits which have been proposed to provide the link between general sensory processing and reading impairments. Thus, on this view, people with poor nonlexical reading skills (phonological dyslexics) might be expected also to show sensory processing deficits in both the visual and the auditory modalities, as much as these deficits are multimodal. People with poor lexical processing skills (surface dyslexics), on the other hand, have been found *not* to show deficits in phonemic awareness (Castles and Coltheart, 1996; Hanley *et al.,* 1992). Thus, there may be less reason to suspect the presence of a general sensory deficit in these people.

Although these issues have been raised in the literature (see, e.g., Farmer and Klein, 1995; Martin, 1995), there has been little research directly examining the relationship between patterns of dyslexia and sensory processing deficits. The work that has been done, however, has been tentatively

supportive of the hypothesis that sensory deficits may be more prevalent in phonological dyslexia than in surface dyslexia. Looking only within the visual modality, and using Boder's (1971) classification scheme, Borsting and colleagues (Borsting *et al.,* 1996; Ridder *et al.,* 1997) have reported that magnocellular pathway deficits do not occur in dyseidetic dyslexics (who have lexical-type reading impairments) but do occur in severe dysphonetics (who have nonlexical-type impariments) and in dysphoneidetics (who have a mixed pattern of deficits). In addition, Spinelli *et al.* (1997) have found no evidence for visual magnocellular impairments in Italian surface dyslexic participants. In the auditory domain, Joanisse *et al.* (1998) found a difference in the categorical perception of speech stimuli in phonological dyslexics who also demonstrated deficits in grammar and vocabulary, while phonological and surface dyslexics without these language impairments performed normally on categorical perception tasks.

In the following section, we present some further data from the auditory domain that point to the importance of examining sensory processing deficits in the context of different component processes in reading.

COMPONENT PROCESSES IN READING AND SENSORY PROCESSING: SOME ILLUSTRATIVE DATA

Some support for a differential association between sensory processing and the component processes in reading may be illustrated with reference to the data of McAnally and Stein (1996). They studied the smallest detectable change in frequency in adult dyslexics and matched controls. The dyslexics were not preselected on the basis of differential performance in reading irregular or nonwords, so the sample was probably heterogeneous with regard to subtypes. However, word and nonword reading error data were collected, so it is possible to look post hoc at correlations between performances on the auditory task and those on the two reading measures.

While performance in frequency discrimination correlated significantly with both word and nonword reading, the numbers of word and nonword reading errors were themselves highly correlated [$r(24) = .77$, $p < .001$]. Differential associations with word and nonword reading errors were therefore determined by partialing out the performance on each of these reading tasks. When the number of errors made while reading words was partialed from the correlation of auditory frequency discrimination and nonword errors, the correlation remained significant [$r(21) = .49$, $p = .017$]; listeners who were poorer at detecting changes in frequency made more errors in reading nonwords. On the other hand, when the number of errors made reading nonwords was partialed from the correlation of frequency discrimi-

nation and word errors, the correlation was not significant [$r(21) = .25$, $p = .26$]. This gives support to the hypothesis that nonlexical skill, as measured by the ability to read nonwords, is related to the ability to discriminate fine changes in the frequency of nonverbal acoustic stimuli. While irregular word reading was not specifically tested, the lack of partial correlation of frequency discrimination with word reading is consistent with a lower reliance on this auditory task for word reading, perhaps because alternate strategies exist.

CONCLUSIONS

In summary, much more work is needed to test adequately the general sensory deficit theory of dyslexia and to uncover its relationship with different patterns of reading impairment. Specifically, both visual and auditory processing need to be examined in the *same* participants, and these participants need to be carefully selected to represent different levels of functioning on component processes in reading. The recent study by Witton *et al.* (1998) showed a high correlation between the threshold for visual coherent motion and that for auditory frequency modulation and also found a significant correlation between both thresholds and nonword reading ability. However, in this study, no attempt was made to classify dyslexics into subtypes based on differential reading ability for irregular or nonwords, and no indication was given of any association between these sensory thresholds and word reading performance.

Were visual and auditory deficits to be explored systematically across dyslexia subtypes, the potential would exist for examining a range of hypotheses about the relationship between auditory and visual processing and reading development. Based on an account of general sensory deficits as affecting reading via speech perception and phonemic awareness impairments, it would be expected that visual and auditory deficits should tend to cooccur in people who display phonological and mixed dyslexic profiles but not in those who show a surface dyslexic pattern. Such a finding would provide the first clear link between general sensory deficits and phonological processes in reading. If this pattern were not found, other possible relationships between sensory processing and reading could be explored. For example, it may be that (a) visual and auditory deficits rarely cooccur, in which case the general sensory processing deficit theory of dyslexia would need to be brought into question; (b) they cooccur to the same extent in all subgroups of dyslexics, suggesting that sensory processing deficits have a more global impact on component processes of reading; and (c) visual or auditory deficits each occur in isolation in particular subgroups of dyslexics,

suggesting that sensory processing impairments have differential effects across modalities on component processes in reading. Regardless of the outcome, much is to be gained from the careful exploration of links between these important psychophysical and psycholinguistic variables.

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