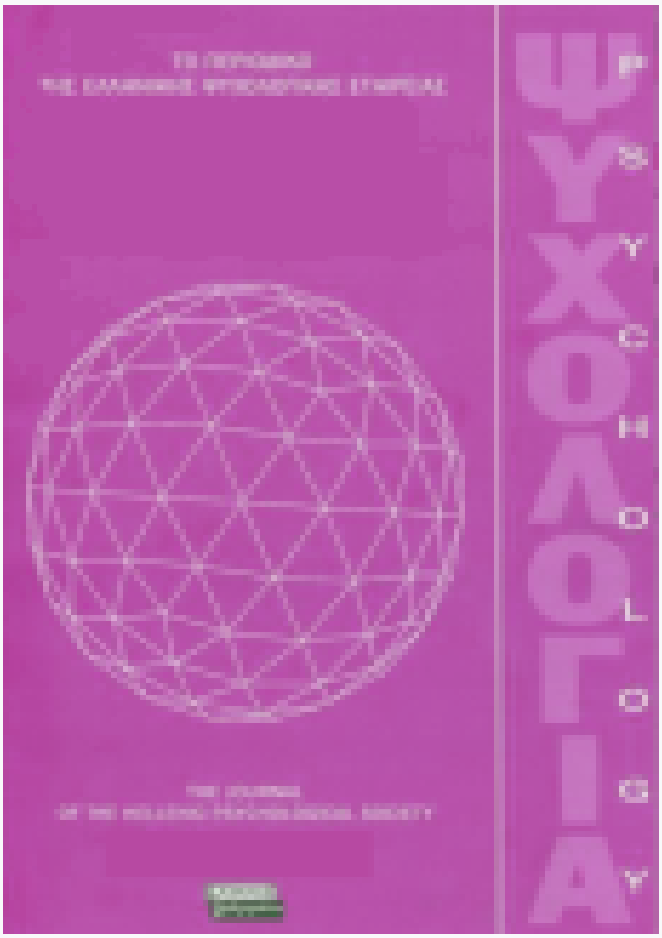
Psychology: the Journal of the Hellenic Psychological Society

Vol 8, No 3 (2001)



Are visual deficits a possible cause of dyslexia?

Francisco J. Martos

doi: 10.12681/psy_hps.24117

To cite this article:

J. Martos, F. (2020). Are visual deficits a possible cause of dyslexia?. *Psychology: The Journal of the Hellenic Psychological Society*, *8*(3), 368–383. https://doi.org/10.12681/psy_hps.24117

Are visual deficits a possible cause of dyslexia?

FRANCISCO J. MARTOS
University of Granada, Spain

ABSTRACT Although most authors claim that deficits in verbal and phonemic processes are the main cause of dyslexia, there also exists a wide number of investigations which support the idea that developmental dyslexia has an etiology linked to alteration in visual perception. The main aim of this paper was to review different hypotheses which have related dyslexia with a deficit in visual perception, namely: the lack of eye dominance; problems in parafoveal vision; deficit in oculomotor control; deficits in early stages of visual processing and magnocellular pathway dysfunctions. It is concluded that there is sufficient evidence to justify the conclusion about the existence of a perceptual dysfunction linked to the etiology of dyslexia. However, it would be necessary to explain how this deficit affects only reading and not other activities in the dyslexic's everyday life. Finally, we discuss the possible existence of different subtypes of developmental dyslexia and how this could explain the controversy in the results of the investigations about the dyslexia etiology.

Key words: Dyslexia, Reading disabilities, Visual perception dysfunctions.

Nowadays, most authors claim that developmental dyslexia is a disorder produced by a phonological or phonemic processing deficit or some other kind of language problem. Accordingly, verbal and linguistic processes would be the common denominator for all forms of dyslexia (Bryant & Goswami, 1988; Ellis, 1984; Vellutino, 1980, 1987). However, it was not always so. We must not forget that the first descriptions of dyslexia were made by ophthalmologists such as Broadbent (1872) or Hinshelwood (1917) who defined dyslexia as "word blindness" or "congenital verbal blindness" (Richardson, 1992). This means that the dyslexic syndrome seemed to be linked to visual dysfunctions. In fact, the visual perception deficit theory as an explanation of dyslexia etiology was very popular some years ago, and still has some support today.

The aim of this paper was just to review some of the more relevant studies in this field. Of course, we do not intend to carry out an exhaustive review of the literature about visual perception and dyslexia. We are only trying to show our research in this area and to present the state of the art about the most relevant

Acknowledgements: This research was supported by grant PB97-802 from the Higher Education Department of the Spanish Ministry of Education and Culture (*Dirección General de Enseñanza Superior e Investigación Científica, Ministerio de Educación y Cultura*).

Address: Francisco J. Martos, Facultad de Psicología, Universidad de Granada, 18071 Granada, Spain. Tel.: *34-958-246236, Fax: *34-958-243774, E-mail: fmartos@platon.ugr.es

hypotheses which have related dyslexia with a deficit in visual processing.

Dyslexia and eye dominance

One of the older theories on developmental dyslexia etiology connected this deficit to a problem in crossed laterality (Porac & Coren, 1976). However, more recently, dyslexia appears to be defined as a problem related with the lack of eye dominance. This approach posits that in near vision, such as reading, eyes converge. Accordingly, eyes are in different positions. Then, each eye sends to brain different signals from the oculomotor system. In order to avoid possible confusion, we learn to only pay attention to extraretinal signals from the dominant eye. If this dominance is not firmly established, the probability of confusing the association being produced between retinal and oculomotor signals increases. Therefore, dyslexia could be associated to these problems in the sense of lack of adequate development of consistent eye dominance. The series of studies carried out by Stein and Fowler (1981, 1982, 1985) investigated this hypothesis in depth.

Stein and Fowler used a variation of the "Dunlop Test" in order to determine the existence of stable eye dominance in dyslexic readers. They found that between 63 and 68 percent of dyslexic readers had unstable eye dominance. They also stated that these children used to make more visual than phonological errors when reading. However, just the opposite was true in the case of those dyslexics with stable eye dominance.

In another study (Stein & Fowler, 1985), children with unstable eye dominance were given glasses with one lens occluded for one year. The aim was children to establish a stable eye dominance. After this period of time, the dyslexic children who had had one eye occluded improved their reading age by 13.5 months, whilst normal readers only improved by 11.8 months. However, the most interesting results were those coming from dyslexics who did not have one eye occluded. They only improved by 3.9 months. The method to overcome dyslexic problems seemed to be easy and efficient.

Peripheral vision and dyslexia

The role played by parafoveal areas of the retina in vision and specifically in reading have also attracted a great deal of attention (Inhoff, Pollatsek, Posner, & Rayner, 1989). The investigations on dyslexia could not ignore these studies. Geyger and Lettvin (1986, 1987) claimed that differences exist between normal and dyslexic readers regarding their skill to extract information from their parafovea. They conducted several experiments with adult dyslexics to test their hypothesis. In a first study, they presented tachistoscopically two stimuli: one of them on the fixation point and the other one in the periphery. The experimenter manipulated the level of eccentricity in the second stimulus. The participants' task consisted in identifying the stimulus presented out of the fovea. The results showed that the number of correct responses decreased as eccentricity increased. However, the pattern was different between dyslexic and normal readers. When stimuli were projected in peripheral areas, 7.5, 10, or 12.5 degrees of eccentricity, the performance of dyslexics was significantly better than that of the normal ones. However, just the opposite happened when the stimuli was only 2.5 degrees of eccentricity.

In a second experiment, a series of three letters were presented in different locations of the periphery of the eye. The experimenter manipulated the level of eccentricity. Now, the participants' task consisted in identifying the three letters. The pattern of letter recognition in normal readers showed that as eccentricity increased the rate of recognition decreased. Usually, the last letter was recognised better than

the first one, and this one better than the letter which was in the central position. However, this pattern of results was different in dyslexic readers. In every case recognition of the central letter was better than that of normal readers and when the three letters were presented at 10 degrees of eccentricity, dyslexics recognised the central letter better than the other letters.

In another study, Geyger and Lettvin (1987) reported that the vision of an adult dyslexic was better at 7.5 degrees of eccentricity than closer locations to the fovea. This dyslexic was trained to read according to a "peculiar" strategy. He should cover the text with a sheet of white paper on which a fixation point was marked at 7.5 degrees, just the distance at which he had achieved his best performance identifying the string of letters. To read, he should look at the dot and make out the words in the window while moving the sheet over the text at a comfortable speed. According to the authors, four months later this person had improved his level of reading from the third grade level up to about the 10th grade level and he said that "I can see the forms of the words clearly".

According to the authors, the explanation of these results is in the different foveal and peripheral resolution of dyslexic readers which would produce an interaction between foveal and peripheral vision that degrades the ability to read in the foveal field. Dyslexics have masking where normal readers have the best resolution and vice versa. Therefore, the foveal reading practice would reinforce dyslexia.

Eye movements and dyslexia

In the search for the etiology of the dyslexic disorder within the domain of perceptual problems, a great deal of interest was created by the hypothesis relating dyslexia to a deficit in eye movement control. Nevertheless, the role of eye movements as an etiological factor of dyslexia only attained definitive importance after the publication of Pavlidis' studies (1981, 1983). In the Pavlidis' experiments, the participants' task usually consisted of following light sequences as accurately as possible with their eyes. The results showed that the dyslexic persons performed poorly on this task. The size and number of saccadics were significantly greater in dyslexics than in normal people although the greatest difference between the two groups appeared in the number of regressive movements. This difference was so great that there was no overlap in the data of the two groups. These results led Pavlidis (1981, 1985, 1990) to propose the recording of eye movements in an ocular tracking task as an objective criterion for the diagnosis of dyslexia. He has indicated that whatever the cause of dyslexia might be the presence of an alteration in the pattern of eye movements seems to be unequivocally linked to this disorder (Pavlidis, 1990).

Although some researchers have indeed reported data supporting this association (Jones & Stark, 1983) others have failed to replicate Pavlidis' results. The studies carried out by Browns, Haegerstron-Portnoy, Adams, Yingling, Galin, Herron, and Marcus (1983) or Olson, Kliegl, and Davidson (1983) did not find differences between normal and dyslexic readers in any of the eye movement parameters. Pavlidis (1983) argued against the studies that did not confirm his data by indicating that their negative results were probably caused by different demands in the selection of dyslexics. A not very strict selection criterion would lead to overlap among the samples of normal, dyslexic and retarded readers and, therefore, contradictory results would be expected to arise. We carried out one experiment in our labs at the University of Granada in which we wanted to examine the relationship between dyslexia and eye movement control in Spanish speaking children taking into account the above mentioned shortcomings (Martos & Vila, 1990).

The study compared the electrooculographic recordings of dyslexic children with those of

retarded and normal readers in three different tasks: one ocular tracking task and two reading tasks which differed in their degree of reading difficulty. The participants were 90 children – 30 dyslexics, 30 retarded readers and 30 normal readers – aged between 7 and 14. It is probably interesting to know the criteria followed to select the sample of dyslexic children because it has been stated that the different demands in their selection could explain the controversy in the results. Children were diagnosed as dyslexics in accordance with the following criteria: (a) Reading age two years below their chronological age; (b) at least 95 in the WISC-R, and (c) no hearing or visual disability, brain damage nor any kind of affective, educational, or family problems which might influence or explain the reading difficulty.

The results showed that in general there were no significant differences between dyslexic and retarded readers in their eye movements if we consider the reading tasks only (see Figure 1). Differences were apparent, however, in the reading tasks between each of the above mentioned groups and the group of normal readers. This lack of significant differences between dyslexic and retarded readers in the reading tasks could be interpreted as evidence that the presence of erratic eye movements in these two groups is a consequence and not a cause of their reading disability. On the contrary, with regard to the ocular tracking task, no significant differences were observed between retarded and normal readers but they were found to exist between each of these two groups and the group of dyslexics (see Figure 2). It is obvious, therefore, that the reason for erratic eye movements in retarded readers is their reading disability but this is not the case with dyslexic readers as we have already mentioned.

Nonetheless, the fact that eye movements and dyslexia might be strongly related does not tell us much about the cause of the disorder. We cannot conclude from such an association that the former is the cause of the latter. On the contrary, some of our results concerning the age factor suggest that this is not the case. The significant main effect of age in the two tasks indicated that eye movements generally improved as age increased. However, the opposite just

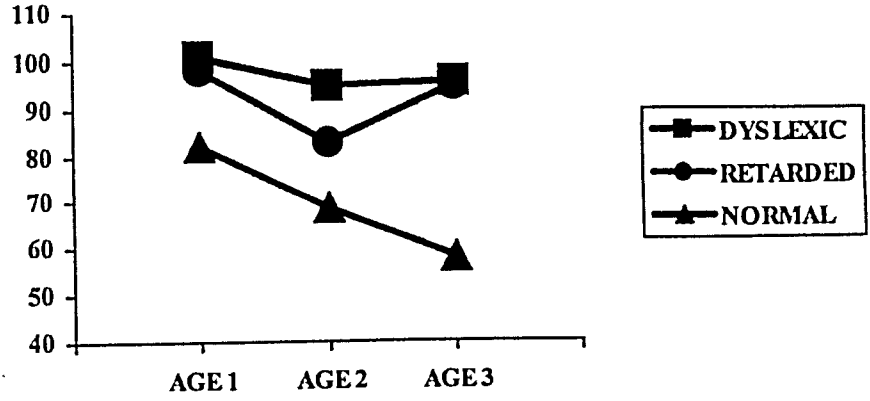


Figure 1

Mean number of saccadic movements during the second reading task in the dyslexic, retarded, and normal groups as a function of age (the graphic representation in the rest of parameters are similar to this).

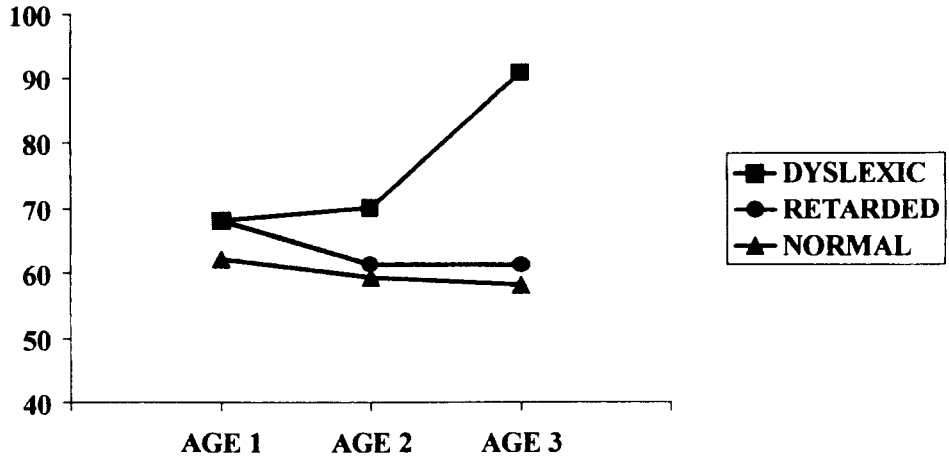


Figure 2
Mean number of regressive movements during the ocular tracking task in the dyslexic, retarded, and normal groups as a function of age (the graphic representation in the rest of parameters are similar to this).

happened with dyslexics. This effect is such that as dyslexics get older their eye movement pattern gets worse. It seems logical to think that the increase in the number of saccadics and regressives produced by dyslexics, as they get older, is due to some kind of learnt strategy that they apply in order to avoid or reduce some other kind of deficit.

Speed of visual information processing and dyslexia

Authors like Breitmeyer (1983), Breitmeyer and Ganz (1976), or Rayner (1987) have suggested that the presence of erratic eye movements in dyslexics should be considered not the cause of dyslexia but a symptom associated with the cause of dyslexia. From their perspective the control of saccadics would be affected as much by central cognitive processes as by peripheral motor or sensory processes. In this way, the existence of altered patterns in the oculomotor control of dyslexics could be caused by the existence of some kind of disorder in the initial stages of the visual information processing.

A lot of research has related the dyslexic etiology to a slower speed of information processing in the different stages of the reading activity (Arnett & Di Lollo, 1979; Badcock & Lovegrove, 1981; Di Lollo, Hanson, & McIntire, 1983; Lovegrove, Martin, & Slaghuis, 1986). Although different authors disagree with regard to the exact stage, which is the responsible for the deficit, nevertheless, all of them maintain that the lower rate in visual information processing produces some kind of perceptual or visual deficit which is the direct cause of dyslexia. In short, it is maintained that given the sequential nature of the reading processing, the lower rate in visual information processing at one stage would produce a stimulation overload in the visual system of the dyslexics. So a visual input may arrive at that stage before the current information has been completely processed. An information bottleneck would ensue leading to an

incomplete processing or integration of the stimuli. The mechanism underlying this pattern of effects would be the one known as visual masking. This explanation, as applied to dyslexia, suggests that perception of a visual stimulus is impaired because the degraded output of the faulty stage provides an inadequate input for all later stages. The perceptual impairment could take a variety of forms such as erasures, substitution, or mirror reversal. What type of perceptual degradation occurs would depend on the function performed by the faulty processing stage. Examples of these points of view can be found in Arnett and Di Lollo (1979), Badcock and Lovegrove (1981), Di Lollo, Hanson, and McIntire (1983), and Lovegrove, Martin, and Slaghuis (1986) among others.

Visual masking and dyslexia

In order to ascertain if there are differences between dyslexic and normal readers in the speed at which they process visual information, Bouma and Legein (1980), Arnett and Di Lollo (1979) and Di Lollo, Hanson, and McIntire (1983) carried out several experiments using backward masking tasks. The general pattern of results showed that dyslexic children need a longer ISI (Inter-stimulus Interval) to avoid the masking effect.

We carried out an experiment trying to measure the speed of visual information processing of three different groups: dyslexic, retarded, and normal readers. We wanted to determine whether the low speed of visual information processing is a differential characteristic of the dyslexic readers or whether, on the contrary, it is also present in other forms of reading disorders (Martos, 1987). The method used to measure the speed of visual information processing was a Backward Masking task. The researcher displayed, in a tachistoscope, two matrices of dots, on the left and on the right of the fixation point. One of these matrices had the central dot missing on a random base. After a brief interval of variable duration, one mask of dots, which filled the full visual range of the participants, was presented. The task was to decide which matrix had the central dot missing.

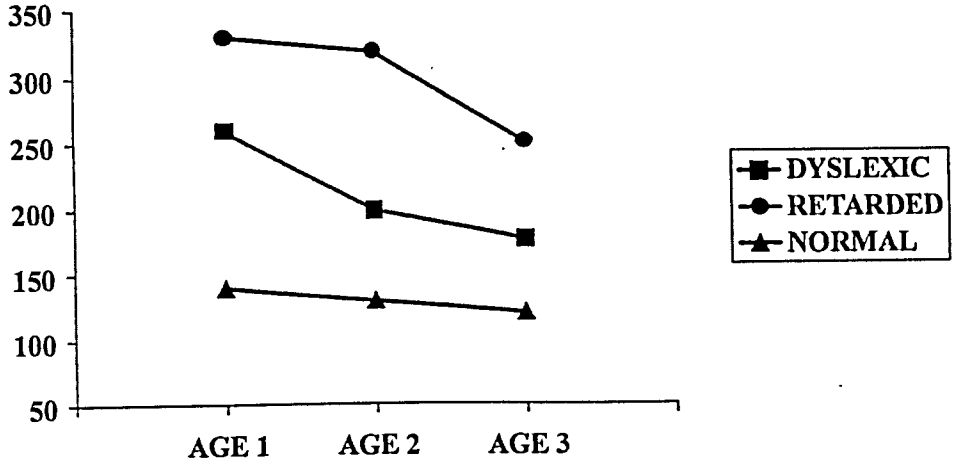


Figure 3
Duration of the interstimuli intervals (ISIs) in the dyslexic, retarded, and normal reader groups in a Backward Masking task.

The task finished when the researcher established the shortest interval between the stimulus and mask, in which the person achieved a rate of 75% of correct responses.

The results showed that dyslexic and retarded readers were slower than normal readers in the speed with which they process stimuli visually presented (see Figure 3). They needed longer ISIs to avoid the backward masking effect. But there were also significant differences between retarded and dyslexic readers. Retarded readers needed even longer ISIs than dyslexic readers. So, the slower speed of visual information processing is not a differential characteristic of developmental dyslexics.

Visible persistence and dyslexia

Another aspect of visual information processing which has been identified as a possible candidate for the explanation of these problems is visible persistence. The studies of Di Lollo, Hanson, and McIntire (1983) or Lovegrove, Martin, and Slaghuis (1986) are two good examples of this approach. Visible persistence refers to the period of time in which the image of the stimulus remains visible after the stimulus disappears. We also carried out an experiment trying to determine the existence of differences in the duration of visible persistence among dyslexic, retarded, and normal readers (Martos & Marmolejo, 1993). The existence of a retarded readers group allowed us to know, once again, if the longer period of visible persistence is a differential characteristic of dyslexia or, on the contrary, if it is also present in all children with reading problems.

We used two different methods of measuring visible persistence. In determining the Temporal Integration Threshold, we displayed, in a tachistoscope, two different stimuli (a vertical line followed by a horizontal line) in rapid succession, separated by an interstimuli interval (ISI) of variable duration. The display of both stimuli with a short ISI caused the observer to integrate them into a single image (a cross). The aim of the researcher was to establish the longest ISI at which the participants were able to maintain an integrated perception of both stimuli.

The second method was the determination of the Gap Detection Threshold. In this case, the same stimulus (i.e., a horizontal line) was displayed twice in rapid succession with a blank ISI of variable duration inserted between each one. The task was to find the shortest ISI at which the participants were able to distinguish the double flash from a single uninterrupted display. The main difference between the two methods is that in the Temporal Integration Threshold different retinal locations are stimulated, while in the Gap Detection Threshold, the stimuli impinge on the same retinal receptors.

The results showed that the visible persistence of normal readers was significantly shorter than that of dyslexic and retarded readers. However, there were no differences at all between dyslexic and retarded readers (see Figure 4).

The significant results also showed that the method of determining the Gap Detection Threshold produced a longer duration of visible persistence because the same retinal receptors are repeatedly stimulated. However, the different method used did not produce differences in the general pattern of results, considering the three groups of participants.

In conclusion, our results showed that dyslexic and retarded readers maintained longer visible persistence than normal readers but visible persistence did not distinguish between retarded and dyslexic children.

Speed of visual processing and lexical access

The various studies previously presented showed that dyslexic children could have difficulties in different stages of visual information processing. However, it would be very helpful if we

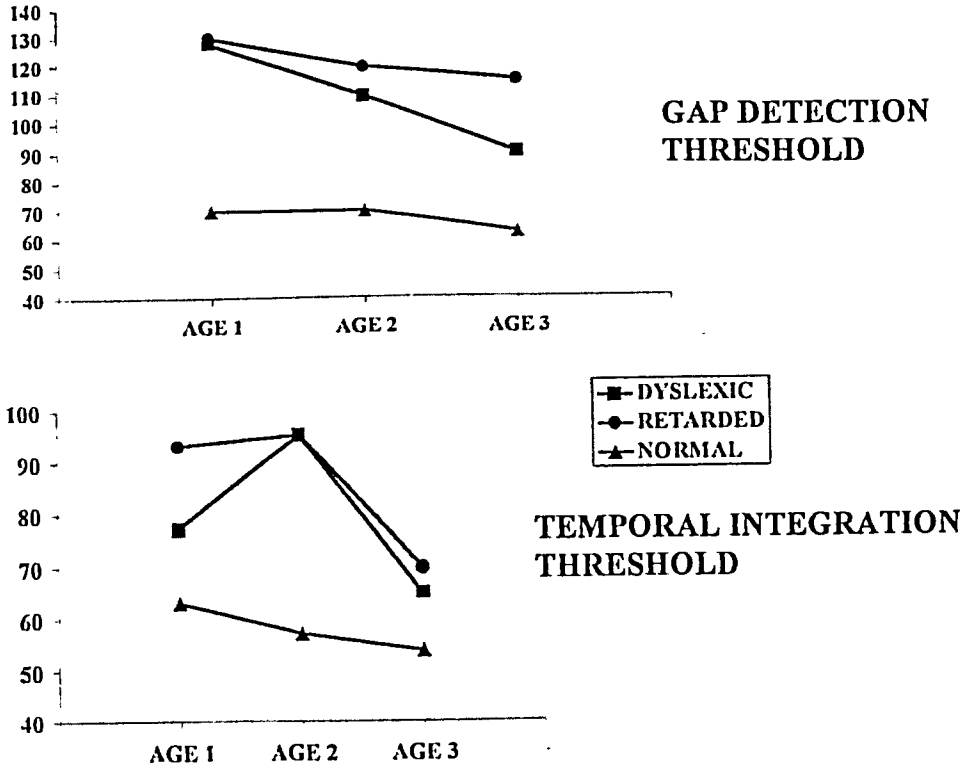


Figure 4

Mean values of the critical ISIs in the dyslexic, retarded, and normal reader groups as a function of age in the two methods used to determine the duration of visible persistence.

could find out if the slower speed of information processing in dyslexic readers is only evident in the initial stages of visual information processing, or whether, on the contrary, this "slowness" is a general characteristic of these person, which is also present in later stages of information processing such as lexical access or phonological processing. We tried to answer this question using an experimental task similar to the one originally used by Posner and Mitchell (1967) (Martos, 1995).

A pair of letters was visually presented and the participant had to respond, as soon as possible, specifying whether the two letters were equal or different. The stimuli were a pair of capital or small letters. This task allowed us to measure the latency of the participants' responses in three different conditions of stimuli presentation:

(1) *Physical Identity* (PI). Both letters were equal not only with regard to their lexical value but also in appearance (i.e., A-A).

(2) *Name Identity* (NI). The letters which formed the stimulus were identical in name and lexical value but different in their physical appearance (i.e., A-a).

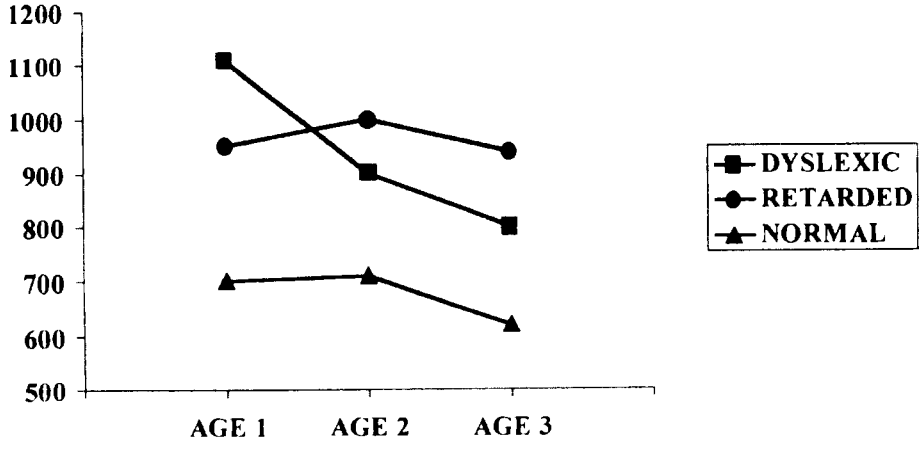


Figure 5
Mean values (msec) of the reaction time in the Physical Identity condition (PI) of stimuli presentation in the dyslexic, retarded and normal groups as a function of age.

(3) *No Identity* (NOI). The two letters were different in physical characteristics as well as in name and lexical value (i.e., A-E).

Physical Identify and Name Identify conditions are different in the number of levels of processing involved. In order to answer under the PI condition the participants only have to compare the perceptual patterns of the stimuli. However, under the NI condition there is an added process, this being access to the lexical code, whereby the person is able to recognise that although the stimuli are different in their physical characteristics, they represent the same letter. The NI condition includes the PI condition. Thus, by subtracting the PI condition reaction time from that of the NI condition (NI - PI) we were able to isolate the time required by the person to access the lexical code and to recognise the physically different letters. The NOI condition was only introduced to prevent the participants from answering quickly even though they had not had time to identify the stimulus. So in the PI condition just as with the NI condition, the participants had to answer as quickly as possible that both letters were the same. The answer had to be 'different' in the NOI condition.

The results showed that dyslexic readers were slower than normal readers in the speed at which they process visually presented stimuli. This lower rate of processing is already manifest in the PI condition, where the participants were required only to distinguish between two physically different stimuli (see Figure 5). These results are consistent with those obtained by Di Lollo et al. (1983) and Lovegrove et al. (1986), in the sense that the deterioration of dyslexic children is produced in the early stages of visual information processing. It cannot be argued against this interpretation of the results that the differences found between dyslexic and normal readers can be explained by the higher difficulty of the dyslexic readers in recognising letters. The number of errors made by dyslexic and normal readers was not significantly different. The number of errors was very low in all the groups, confirming that the task was very easy for all the

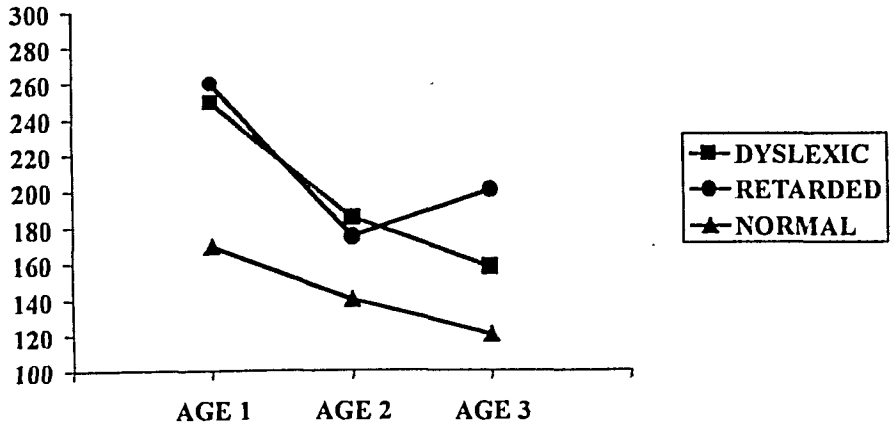


Figure 6
Mean values (msec) of the subtraction of time reactions of Physical Identity from time reactions of Name Identity conditions (NI-PI) in the dyslexic, retarded and normal groups as a function of age.

participants. There was another important finding regarding our results; it concerned the fact that the dyslexic readers were also slower than normal readers in the NI-PI condition (see Figure 6). Dyslexics were also slower in the lexical access time. Accordingly, the lower rate of information processing seems to reflect a general characteristic of dyslexic persons, which is present in all kinds of information processing, even in that not related to visual information processing. Besides this, there were no differences between dyslexic and retarded readers. Therefore, neither the lower rate of visual information processing nor lexical access speed can be a differential characteristic of developmental dyslexia.

The magnocellular theory of dyslexia

Recent research about dyslexia and visual processing has related the problem with a more concrete dysfunction not from damage to a single visual relay but from abnormalities of the magnocellular component of the visual system which would be specialized for processing fast temporal information. The evidence would be consistent with an increasingly sophisticated account of dyslexia that does not single out either phonological or visual or motor deficits. Rather, temporal processing in all three systems seems to be impaired. Dyslexics may be unable to process fast incoming sensory information adequately in any domain (Everett, Bradshaw, & Hibbard, 1999; Stein & Walsh, 1997).

Studies carried out in physiological psychology and neuropsychology have shown that the primate visual system consists of two parallel, largely independent systems: the *magnocellular system* and the *parvocellular system*, named after the two types of cell layers in the lateral geniculate nucleus (LGN). These different visual cortical pathways have their origins in two different types of retinal ganglion cells. The P type terminates in the parvocellular layers of the LGN and has general characteristic, which make

it more suitable for form and color vision, while the M type terminates in the magnocellular layers of the LGN and has characteristics, which make it more suitable for detecting dynamic form and motion. Accordingly, the magnocellular system is involved with the analysis of form, movement, depth and selective orientation whilst the parvocellular system is involved with color perception and fine details (Carlson, 1992; Zeki, 1993). The magnocellular theory about dyslexia maintains that dyslexic children suffer from a dysfunction in their magnocellular (M) pathway and this abnormality implies severe deficits in the processing of temporal visual information which affect the acquisition of reading skill. Most dyslexics would have lower sensitivity to rapidly changing visual and auditory stimuli as a result of slightly impaired development of magnocellular neurons and this may explain their visual instability and phonological reading problems (Stein & Walsh, 1997).

Demb, Boynton, and Heeger (1997, 1998) examined the relationship between brain activity and reading performance to test the hypothesis that dyslexia involves a deficit in the M pathway. Functional magnetic resonance imaging was used to measure brain activity in dyslexic and control participants in conditions designed to preferentially stimulate the M pathway. They found significant correlations between reading rate, speed discrimination thresholds and brain activity. Dyslexics showed reduced activity compared with controls both in the primary visual cortex (V1) and adjacent motion-sensitive (MT+) areas that are believed to receive a predominant M pathway input. On the contrary, participants with higher V1 and MT+ responses had lower perceptual thresholds (better performance) and were faster readers.

More recently, anatomic evidence has been found about the alteration of the M stream in the dyslexics brain. Jenner, Rosen, and Galaburda (1999) measured cross-sectional neuronal areas in primary visual cortex (area 17) in dyslexic and nondyslexic autopsy specimens. There was a significant interaction between hemispheres and diagnostic category; nondyslexic brains had larger neurons in the left hemisphere, whereas dyslexic brains had no asymmetry. On the other hand, cell layers associated with magnocellular input from the lateral geniculate nucleus did not show consistent changes in dyslexic brains. Thus, there is a neuronal size asymmetry in favor of the left primary visual cortex in nondyslexic that is absent in dyslexic brains. This is yet another example of anomalous expression of cerebral asymmetry in dyslexia to that, previously known, of the planum temporale. Both would reflect abnormality in circuits involved in reading.

It remains controversial the explanation about how abnormalities in the M pathway might affect children's reading. Talcott, Hansen, Willisowen, McKinnell, Richardson, and Stein (1998) have suggested that visual magnocellular impairment may be characteristic of up to 75 percent of developmental dyslexics. These children would be less sensitive to detection of coherent motion, of flicker fusion frequency, of moving visual stimuli, of backward masking, of temporal integration task, etc. (Boden & Brodeur, 1999; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998; Talcott et al., 1998). It suggested that when children read, impaired magnocellular function may degrade information about where letters are positioned with respect to each other, leading to reading errors which contain sounds not represented in the printed word. Steinman, Steinman, and Garzia (1998) also maintain that a deficient M stream produces specific abnormalities in the visual attention mechanisms of disabled readers. However, we think that the mechanism by which M pathway deficits affect reading has not still been addressed. The explanations given are very vague. It would be necessary to offer a theory about the mechanism underlying reading which can explain specifically how the M pathway dysfunction affects reading ability. Moreover, it must be explained why this alteration only affect reading and not other activities in the dyslexic children's everyday life.

There is no evidence that dyslexics show deficits in activities other than reading.

Against the magnocellular theory of dyslexia can also be argued the existence of many studies, which have not found visual problems in dyslexic children. On the other hand, many studies, some of them mentioned already, did not find differences in visual temporal processing between dyslexics and other children with reading deficits. Moreover, there seems to be accepted existence of severe deficit in verbal information processing or, more specifically, in phonemic awareness in people with dyslexia. However, it has not been explained how the visual perception dysfunction could produce such deficits.

The results of recent research could help us answer these questions. Cornelissen et al. (1998) and Talcott et al. (1998) have shown that impaired magnocellular visual functioning as well as phonemic and language deficits would affect how children read. From this approach, it could be supported the existence of subtypes of dyslexia. One subtype would be produced by visual temporal alteration caused by dysfunction of the M pathway and others would be more linked to verbal or phonemic deficits. Research carried out by Borsting, Ridder, Dudeck, Kelley, Matsui, and Motoyama (1996) and Slaghuis and Ryan (1999) have supported this notion. They distinguished between dyseidetic, dysphonetic, and dysphoneidetic (mixed) subgroups of dyslexia and their results support that a transient chanel disorder (linked to the M pathway) may only be present in the dysphoneidetic subgroup. The existence of different subtypes of dyslexia have also found anatomic support. Best and Demb (1999) examined the relationship between the abnormalities in the planum temporale and the deficit in the magnocellular visual pathway. They used sagital magnetic resonance images to measure the planum temporale to dyslexic persons with a documented magnocellular deficit and controls. Results showed that this type of dyslexic persons do not present the expected abnormal symmetry of the planum temporale. They suggest that the symmetry of the planum temporale would be related to a subtype of dyslexia and the abnormalities in the M pathway would be related to a different one. These results are in agreement with those previously reported from Jener, Rosen, and Galaburda (1999).

Conclusions

The evidence for a temporal processing deficit in the visual domain has been recently reviewed by Farmer and Klein (1995) and it was concluded that there exists enough evidence to link visual processing deficit to dyslexia. Besides this, studies such as those conducted by Eden, Stein, Wood, and Wood (1995) have also shown that a high percentage of children with reading disabilities suffer visual or oculomotor alterations. Furthermore, the experiments carried out by Geyger and Lettvin (1986, 1987) or more recently by Geyger, Lettvin, and Fahle (1994) or those experiments, above mentioned, which have related dyslexia to problems in the magnocellular pathway give enough support to relate dyslexia to problems in lateral masking; peripheral vision or other deficits linked to visual temporal processing. Accordingly, we think that there is evidence to accept the existence of a visual perceptual deficit linked to the etiology of dyslexia, although it is also true that other studies did not find this kind of relationship (Hayduk, Bruck, & Cavanagh, 1996; Spinelli, Angelelly, Deluca, Ripace, Judica, & Zoccoloti, 1997).

We agree with Rayner, Pollatsek, and Bilsky (1995) in the sense that there is enough evidence to accept that some dyslexic children have visual perception problems although, these kinds of visual deficit has never been clearly defined. Accordingly, it would be useful to explain adequately how these problems in visual perception can affect reading. In our judgement, the argument against the visual perception theory as etiology of dyslexia formulated by

Charles Hulme (1988) in his article "About the implausiblity of low-level visual deficits as a cause of childrens' reading disabilities" is still valid. If dyslexic children have problems in visual perception, why does this alteration not manifest itself in other activities of their daily life except reading?

Although the magnocellular theory has provided some arguments to explain how temporal visual alteration would affect reading decoding, we think that the mechanism by which M pathway deficits affect reading has not still been addressed. The explanations given are very vague. It would be necessary to offer a theory about the mechanism underlying reading which can explain specifically how the M pathway dysfunction affects reading skill. There is no evidence that dyslexics show deficits in activities other than reading. Accordingly, it must be explained why this deficit only affects reading and not other activities in the dyslexic children's everyday life.

On the other hand, our investigations have shown that dyslexic children are slower than normal readers in visual information processing. But this is not a differential characteristic of dyslexics because this characteristic is also present in other children with reading problems. If the lower rate of visual information processing is the cause of dyslexia, we would have to accept that dyslexic children are not different from retarded readers or other children with reading problems regardless of the cause.

We have also shown that the "slowness" of dyslexics appears in tasks not related to visual perception. They were also slower in lexical access tasks. This means that this slowness could be a general characteristic of dyslexia not only linked to visual processing.

Maybe, the controversy in the results could be explained because in the majority of cases researchers define dyslexia as if it were a single deficit. But if it is not a single deficit, it would be expected to find controversial results. The selection of children with phonological problems or children with visual problems, or both, when we select the sample on a random base could explain these differences. Hoghen (1996) has emphasized the importance of being strict in the selection of the samples. He has shown that results are different when children with different reading problems are included in the same experiment.

From our point of view, it is reasonable to assume that a so complex skill such as reading in which many stages with different processes are involved can be altered by problems in different levels of processing. Maybe, we should accept the existence of different subtypes of developmental dyslexia. In fact, recent investigations have found tasks which allow to distinguish between dyslexics who have problems in visual naming speed but not phonological problems and vice versa (Wolf, 1997). On the other hand, Ridder, Borsting, Cooper, McNeel, and Huang (1997) in their article, "Not all dyslexics are created equal" have shown that the pathophysiology of developmental dyslexia is more complex than originally thought and there is evidence to justify the existence of dyslexics with visuomotor or visuospatial disorders. Even more recently, the studies carried out by Best and Demb (1999) or Jener, Rosen, and Galaburda (1999) have pointed out the existence of anatomic abnormalities in the dyslexic brains which allow to distinguish between problems in the M cells of the LGN and problems with the anomalous asymmetry of the planum temporale. All these data suggest that dyslexia should not be defined as a unitary concept. On the contrary, it would be more convenient to accept different subtypes of dyslexia which could be distinguished in terms of their etiology.

In conclusion, we think that there is enough evidence to accept the existence of alterations in the early stages of visual information processing at least in some form of dyslexia. However, more research is needed to clarify the role played by this alteration in the acquisition of reading. In our opinion, investigations such as those carried out

by Boder (1973) some years ago distinguishing "dyseidetic" and "dysphonetic" dyslexia or those more recently conducted by Castles and Coltheart (1993), Fletcher, Morris, Reid, Stuebing, Shaywitz, Shankweiller, Katz, and Shaywitz (1997), Slaghuis and Ryan (1999), or Stanovich, Siegel, Gottardo, Chiappe, and Sidhu (1997) showing the existence of several types of developmental dyslexia, and trying to find out the different etiologies of them, point out which should be the way of future research.

References

Arnett, J. L., & Di Lollo, Y. (1979). Visual information processing in relation to age and to reading ability. *Journal of Experimental Child Psychology, 27*, 143-152.

Badcock, D., & Lovegrove, W. (1981). The effects of contrast, stimulus duration and spatial frequency of visible persistence in normal and specifically disabled readers. *Journal of Experimental Psychology: Human Perception and Performance, 7*, 495-505.

Best, M., & Demb, J. B. (1999). Normal planum temporale asymmetry in dyslexics with a magnocellular pathway deficit. *Neuroreport, 3*, 607-612.

Boder, E. (1973). Developmental dyslexia: A diagnostic approach based on three atypical reading spelling patterns. *Developmental Medicine and Child Neurology, 15*, 663-687.

Boden, C., & Brodeur, D. A. (1999). Visual processing of verbal and nonverbal stimuli in adolescents with reading disabilities. *Journal of Learning Disabilities, 32*(1), 58-71.

Borsting, E., Ridder, W. H., Dudeck, K., Kelley, C., Matsui, L., & Motoyama, J. (1996). The presence of a magnocellular defect depends on the type of dyslexia. *Journal of Vision Research, 36*, 1047-1053.

Bouma, H., & Legein, Ch. P. (1980). Dyslexia: A specific recoding deficit? An analysis of response latencies for letters and words in dyslexics and in average readers. *Neuropsychologia, 18*, 285-298.

Breitmeyer, B. G. (1983). Sensory masking, persistence, and enhancement in visual exploration and reading. In K. Rayner (Ed.), *Eye movements in reading* (pp. 3-30). New York: Academic.

Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression and information processing. *Psychological Review, 83*, 1-36.

Broadbent, W. H. (1872). On the cerebral mechanism of speech and thought. *Transactions of the Royal Medical and Chirurgical Society, 15*, 145-194.

Brown, B., Haegerstrom-Portnoy, G., Adams, A. J., Yingling, Ch. D., Galin, D., Herron, J., & Marcus, M. (1983). Predictive eye movements do not discriminase between dyslexic and control children. *Neuropsychologia, 21*, 121-128.

Bryant, P. E., & Goswami, U. (1987). Development of phonemic awareness. In J. Beech & A. Colley (Eds.), *Cognitive approaches to reading* (pp. 213-243). Chichester, UK: Wiley.

Carlson, N. R. (1992). *Foundations of physiological psychology.* Boston: Allin and Bacon.

Castles, A., & Coltheart, M. (1993). Varieties of developmental dyslexia. *Cognition, 47*, 149-180.

Cornelissen, P. L., Hansen, P. C., Hutton, J. L., Evangelinou, V., & Stein, J. F. (1998). Magnocellular visual function and children's single word reading. *Vision Research, 37*(3), 471-482.

Demb, J. B., Boynton, G. M., & Heeger, D. J. (1997). Brain activity in visual cortex predicts individual differences in reading performance. *Proceedings of the National Academy of Sciences of the United States of America, 94*(24), 13363-13366.

Demb, J. B., Boynton, G. M., & Heeger, D. J. (1998). Functional magnetic resonance imaging of early visual pathways in dyslexia.

Journal of Neuroscience, 18(17), 6939-6951.

Di Lollo, V., Hanson, D., & McIntire, J. S. (1983). Initial stages of visual information processing in dyslexia. *Journal of Experimental Psychology: Human Perception and Performance, 9*, 923-935.

Eden, G. F., Stein, J. F., Wood, M. H., & Wood, F. B. (1995). Verbal and visual problems in reading disability. *Journal of Learning Disabilities, 28*(5), 272-290.

Ellis, A. W. (1984). *Reading, writing and dyslexia: A cognitive analysis*. Hillsdale, NJ: Erlbaum.

Everett, J., Bradshaw, M. F., & Hibbard, P. B. (1999). Visual processing and dyslexia. *Perception, 2*, 243-254.

Farmer, M. E., & Klein, R. M. (1995). The evidence for a temporal processing deficit linked to dyslexia: A review. *Psychonomic Bulletin & Review, 2*(4), 460-493.

Fletcher, J. M., Morris, R., Reid, G., Stuebing, K., Shaywitz, S., Shankweiler, D. P., Katz, P., & Shaywitz, B. A. (1997). Subtypes of dyslexia: An old problem revisited. In B. A. Blachman (Ed.), *Foundations of reading acquisition and dyslexia* (pp. 95-114). Mahwah, NJ: Erlbaum.

Geyger, G., & Lettvin, J. Y. (1986). Enhancing the perception of form in peripheral vision. *Perception, 15*, 119-130.

Geyger, G., & Lettvin, J. Y. (1987). Peripheral vision in person with dyslexia. *The New England Journal of Medicine, 316*, 1238-1243.

Geyger, G., Lettvin, J. Y., & Fahle, M. (1994). Dyslexic children learn a new visual strategy for reading. A controlled experiment. *Vision Research, 34*, 1223-1233.

Hayduk, S., Bruck, M., & Cavanaugh, P. (1996). Low-level visual processing skills of adults and children with dyslexia. *Cognitive Neuropsychology, 13*, 975-1015.

Hinshelwood, J. (1917). *Congenital word-blindness*. London: Lewis.

Hoghen, J. H. (1996). A plea for purity. *Australian Journal of Psychology, 48*, 172-177.

Hulme, C. (1988). The implausibility of low-level visual deficits as a cause of children's reading difficulties. *Cognitive Neuropsychology, 5*(3), 369-374.

Inhoff, A. W., Pollatsek, A., Posner, M. I., & Rayner, K. (1990). Covert attention and eye movements during reading. *The Quartely Journal of Experimental Psychology, 41*, 63-89.

Jenner, A. R., Rosen, G. D., & Galaburda, A. M. (1999). Neuronal asymmetries in primary visual cortex of dyslexic and nondyslexic brains. *Annals of Neurology, 2*, 189-196.

Jones, A., & Stark, L. (1983). Abnormal patterns of normal eye movements in specific dyslexia. In K. Rayner (Ed.), *Eye movements in reading* (pp. 481-498). New York: Academic.

Lovegrove, W., Martin, F., & Slaghuis, W. (1986). A theoretical and experimental case for a visual deficit in specific reading disability. *Cognitive Neuropsychology, 2*, 225-267.

Martos, F. J. (1987). *Etapas iniciales del procesamiento de la información visual en lectores normales, retrasados y disléxicos* [Early stages of visual information processing in normal, retarded, and dyslexic readers]. Granada, Spain: Servicio de Publicaciones de la Universidad de Granada.

Martos, F. J. (1995). Speed of visual information processing in developmental dyslexia. In C. K. Leong & R. M. Joshi (Eds.), *Developmental and acquired dyslexia* (pp. 51-59). Dordrecht, The Netherlands: Kluwer.

Martos, F. J., & Marmolejo, A. (1993). Visible persistence and dyslexia. In P. Tallal, A. Galaburda, R. Llinas, & C. Von Euler (Eds.), *Temporal information processing in the nervous system* (pp. 383-386). *Annals of New York Academy of Sciences, 682*, 383-386.

Martos, F. J., & Vila, J. (1990). Difference in eye movements control among dyslexic, retarded readers and normal readers in the Spanish population. *Reading and Writing: An Interdisciplinary Journal, 2*, 175-188.

Olson, R. K., Kliegl, R., & Davidson, B. J. (1983). Dyslexic and normal readers' eye movements. *Journal of Experimental Psychology: Human Perception and Performance, 9*, 816-

825.

Pavlidis, G. Th. (1981). Do eye movements hold the key to dyslexia? *Neuropsychologia, 19*, 57-64.

Pavlidis, G. Th. (1983). The "dyslexia syndrome" and its objective diagnosis by erratic eye movements. In K. Rayner (Ed.), *Eye movements in reading* (pp. 441-466). New York: Academic.

Pavlidis, G. Th. (1985). Eye movements differences between dyslexics, normal and retarded readers while sequentially fixating digits. *American Journal of Optometry and Physiological Optics, 62*, 820-832.

Pavlidis, G. Th. (1990). *Perspectives on dyslexia: Vol I. Neurology, neuropsychology and genetics*. Chichester, UK: Wiley.

Porac, C., & Coren, S. (1976). The dominant eye. *Psychological Bulletin, 83*, 880-897.

Posner, M., & Mitchell, R. (1967). Chronometric analysis of classification. *Psychological Review, 74*, 392-409.

Rayner, K. (1987). Eye movements and the perceptual span: Evidence for dyslexic typology. In G. Th. Pavlidis & D. F. Fisher (Eds.), *Dyslexia: Its neuropsychology and treatment*. Chichester, UK: Wiley.

Rayner, K., Pollatsek, A., & Bilsky, A. B. (1995). Can a temporal processing deficit account for dyslexia? *Psychonomic Bulletin and Review, 2*(4), 501-507.

Richardson, S. (1992). Historical perspective on dyslexia. *Journal of Learning Disabilities, 25*, 40-47.

Ridder, W. H., Borsting, E., Cooper, M., Mcneel, B., & Huang, E. (1997). Not all dyslexics are created equal. *Optometry and Vision Science, 74*(2), 99-104.

Slaghuis, W. L., & Ryan, J. F. (1999). Spatiotemporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. *Vision Research, 3*, 651-668.

Spinelli, D., Angelelly, P., Deluca, M., Ripace, E., Judica, A., & Zoccoloti, P. (1997). Developmental surface dyslexia is not associated with deficits in the transient visual system. *Neuroreport, 8*, 1807-1812.

Stanovich, K. E., Siegel, L. S., Gottardo, A., Chiappe, P., & Sidhu, R. (1997). Subtypes of developmental dyslexia: Differences in phonological and orthographic coding. In B. A. Blachman (Ed.), *Foundations of reading acquisition and dyslexia* (pp. 115-141). Mahwah, NJ: Erlbaum.

Stein, J. F., & Fowler, S. (1981). Visual dyslexia. *Trends in Neuroscience, 4*, 77-80.

Stein, J. F., & Fowler, S. (1982). Diagnosis of dyslexia by means of a new indicator of eye dominance. *British Journal of Ophthalmology, 66*, 332-336.

Stein, J. F., & Fowler, S. (1985). Effect of monocular occlusion on visuomotor perception and reading in dyslexic children. *The Lancet, 13*, 69-73.

Stein, J. F., & Walsh, W. (1997). To see but not to read - The magnocellullar theory of dyslexia. *Trends in Neurosciences, 20*(4), 147-152.

Steinman, S. B., Steinman, B. A., & Garzia, R. P. (1998). Vision and atention II. Is visual attention a mechanism through which a deficient magnocellular pathway might cause reading-disability? *Optometry and Vision Science, 9*, 674-681.

Talcott, J. B., Hansen, P. C., Willisowen, C., McKinnell, I. W., Richardson, A. J., & Stein, J. F. (1998). Visual magnocellular impairment in adult developmental dyslexics. *Neuro-Ophthalmology, 4*, 187-201.

Vellutino, F. R. (1980). *Dyslexia: Theory and research*. Cambridge, MA: MIT Press.

Vellutino, F. R. (1987). Dislexia [Dyslexia]. *Investigación y Ciencia, 128*, 12-20.

Wolf, M. (1997). A provisional, integrative account of phonological and naming-speed deficits in dyslexia: Implications for diagnosis and intervention. In B. A. Blachman (Ed.), *Foundations of reading acquisition and dyslexia* (pp. 67-92). Mahwah, NJ: Erlbaum.

Zeki, S. (1993). *A vision of the brain*. Oxford, UK: Blackwell.