IOSR Journal of Computer Engineering (IOSR-JCE)
e-ISSN: 2278-0661,p-ISSN: 2278-8727, Volume 21, Issue 3, Ser. IV (May - June 2019), PP 01-12
www.iosrjournals.org

Visual Deficits and Reading Failure

Milena SlavchevaMihaylova[a], Margarita Zlatkova[a,b], NadejdaBocheva[a], Robert Longhurst[c]

[a –] *Institute of Neurobiology, BAS, Sofia, Bulgaria;*
[b –] *University of Ulster, Coleraine, UK;*
[c –] *Aston University, Birmingham, UK*
Corresponding author: Milena SlavchevaMihaylova.

Abstract: *This paper reviews advances in understanding the role of visual impairments in reading failure with a focus on perceptual distortions (visual stress), magnocellular deficits, abnormal eye movements and increased visual neural noise. The neurobiological evidence suggests that the increased neural noise might be caused by disbalance of excitation and inhibition in the cortical networks or by the inability to exclude the neural noise due to impaired attentional mechanisms. Deficits in attentional processes are further discussed in the context of the causal link between reading disabilities and eye movements together with other potential sources of abnormal eye movements.The review accentuates on the complexity of the reading process and its impairments and the necessity to consider the involvement of variety of factors, like sensory, attentional, phonological, oculomotor and their interaction.*

Keywords:*reading difficulties, neural noise hypothesis, magnocellular deficits, eye movements, attention impairment, visual stress, development disorders*

Date of Submission: 25-05-2019 Date of acceptance: 10-06-2019

I. Introduction

Reading is a complex activity involving multistage phonological, visual, cognitive and linguistic processes. Dysfunction in any of these processes can cause reading failure, but despite the extensive research, there is little understanding of the causal mechanisms. At present the dominant cognitive theory is poor processing of phonological information. Although there is convincing evidence supporting the phonological theory, it does not address visual processing or oculomotor dysfunctions related to reading difficulties. Some authors have been dismissive of the link between reading problems and visual function (Wilson, 2015; Creavin et al., 2015).

While the phonological theory neglects the visual contribution to reading difficulties, numerous studies have considered reading disabilities as closely associated with visual anomalies such as non-efficient visual processing related to magnocellular deficits, or visual stress, abnormal eye movements or anomalies in visual attention (e.g. Gori&Facoetti, 2015; Quercia et al, 2013).

Regardless of the underlying mechanisms, the problems in reading and spelling give cause for serious concern. Terms "dyslexia" and "reading disability" have often been used interchangeably, although there are other forms of learning disability and developmental disorders, which also impact reading. For example, children with autism spectrum disorder (ASD), attention deficit hyperactivity disorder (ADHD) and developmental dyslexia (DD) are estimated to represent approximately 10% of the population (Butterworth &Kovac, 2013). Although these three conditions are often co-morbid, each disorder refers to a specific pattern of behavioural and learning difficulties with different core defining features. Despite of this diversity, the abnormal visual perception in these disorders might be explained by a common hypothesis - the high neuronal noise in different brain networks (e.g. Dinstein et al., 2015; Saville et al., 2015).

There is increasing awareness that neither phonological problems nor other factors such as visual, oculomotor or attention deficits etc. taken separately can explain reading failure (Ramus et al., 2003). In the recent years, there is significant progress in this direction by considering factors acting at different levels and their interaction. The present review focuses on theoretical concepts and different lines of research related to visual contribution to reading deficits: the potential role of perceptual dysfunctions, magnocellular deficits, increased internal neural noise and abnormal eye movements and the origin of some controversies related to the role of visual processing in reading.

We appreciate that it is difficultto encompass, in a single review, reading failures occurring in different disorders and explained by different theories. Nevertheless, we consider that this effort is necessary since, in contemporary literature, an enormous number of divergent reports exist.

Since theoccurrence of reading failure may be due to different disorders and even a single disorder has variousmanifestations, any review of reading difficulties and their causes is intrinsically heterogenous and incomplete. It is essential to consider visual deficits in reading as related to a wide range of mechanisms,acting at different levels and different modalities, which is dictated by the multifactorial character of the reading process and its impairment.

1.1. Visual stress and colour filters

A presence of somatic (eye strain, headaches or nausea) and vision-related perceptual distortions such as blur and visual perception instabilities during reading have first been described by Meares in 1980 and then independently by Irlen (1983), currently known as Meares-Irlen syndrome (MIS) or visual stress (Wilkins, 1995). The manifestation of visual stress in certain individuals with reading difficulties has attracted considerable interest in the potential role of visual processing in reading. When viewing text, some people also experience visual illusions of motion, shape and colour. Some dyslexic individuals experience certain symptoms like 'jumping letters', 'dancing lines' etc. (Stein & Walsh, 1997). This effect is quite intensive with repetitive striped patterns presented at high contrast and subtending large visual angle (Wilkins &Nimmo-Smith, 1984) called patterned glare or pattern glare as it is now known (Evans &Drasdo, 1991; Evans & Stevenson, 2008). The patterns that cause pattern glare can trigger seizures or migraine in people susceptible to headaches or patients with photosensitive epilepsy (Wilkins & Evans, 2010).

The aetiology of these effects is still unclear. One explanation is that the periodic patterns of striped stimuli, that cause visual perception illusions and discomfort, evoke strong stimulation of the neurons in the visual cortex sensitive to gratings of specific spatial frequency (SF). This intense level of cortical stimulation can cause a failure of inhibitory interactions and widespread excitation, leading to perceptual distortions (e.g. Wilkins &Nimmo-Smith, 1984; Wilkins et al. 1984; Wilkins et al. 2004; Evans & Stevenson, 2008; Evans et al, 2017). Similar hyper excitability of the cortex has been reported in migraineurs. Some neurological evidence supporting this hypothesis comes from a study of gamma oscillations in the primary visual cortex, V1, in response to grating patterns. The authors have reported maximum cortical power at SF at the peak of visual illusions. Neuroimaging studies have shown stronger cortical excitation, provoked by visual stress stimuli in patients with migraine (Huang et al, 2003) or MIS (Chouinard et al, 2012) relative to controls. Alternative explanations include disturbances at the level of retinal image formation, caused by ocular motor abnormalities such as abnormal eye movements during fixation and binocular instability. Simmers et al. (2001) reported unstable accommodation and increased magnitude of accommodation micro fluctuations in individuals with visual stress. It is unclear however, whether the observed disturbances in the accommodation cause visual stress, or whether they are a consequence of the perceptual distortions that would reduce the signal for accommodation response. Similarly, there was no conclusive evidence for the causal role of binocular anomalies such as impaired convergence and/or accommodation (Evans et al, 1996). Monger et al (2016) reported that the number of illusions evoked by gratings depended on their SF and not on viewing distance, suggesting that they are not caused by accommodation disturbances. This has been interpreted as supporting the cortical origin of the illusions (Wilkins et al., 2016).

Since the lines of text form periodic pattern, resembling a grating of spatial frequency close to the maximum human contrast sensitivity, it has been claimed that the high contrast of the text creates pattern glare which can lie at the core of the reported reading difficulties (e.g. Wilkins &Nimmo-Smith, 1984; Conlon et al 1998; Wilkins et al 2004). Fernandez and Wilkins (2008) presented findings for rating a discomfort from complex images showing that the most discomfortable images had more power around 3 c/deg, near the maximum contrast sensitivity. Using reading-like visual search task, Conlon et al (1998) showed that the performance of people with high visual discomfort was significantly worse when the global pattern resembles square wave grating. It was suggested that, for some individuals, pattern glare contributes to the poor reading ability and creates further complications for dyslexics who experience it (e.g. Conlon et al 1998; Kriss& Evans, 2005; Evans & Stevenson, 2008; Longhurst, 2017).However,the diagnosis of visual stress is subject of controversy and estimates of its prevalence vary since there are no standard criteria to diagnose the condition (Kruk et al, 2008; Uccula et al, 2014; Henderson et al, 2014). Some criteria are based on subjective reports, while others are related to the increased reading rate (more than 5%) after a treatment with coloured lenses. Using the later criteria, Kriss&Evans (2005) reported a prevalence of 25% in a control group and 31% in a dyslexic group although the difference was not significant.

It should be mentioned that not all people with reading disability experience visual stress and some visual stress sufferers can be normal readers (Wilkins et al., 2016). Currently, visual stress is considered as independent from dyslexia, although it can be a comorbid condition (Kriss& Evans, 2005; Singleton & Trotter, 2005; Uccula et al, 2014; Wilkins et al., 2016).

There is a large number of studies investigating if visual stress can be alleviated and reading performance improved by using colour filters (for a review of the existing systems, see Grifiths et al. 2016). Irlen has introduced treatment by individually prescribed coloured lenses and overlays developed in Irlen clinics

(Irlen&Lass, 1989). Despite the authors' claims that this method gives immediate results to people affected by MIS, including dyslexia sufferers, there is considerable scepticism regarding the treatment with Irlen tinted lenses. In a comprehensive review of the literature published before 1991, Evans and Drasdo (1991) gave an outline of the controversies in this therapy and pointed out that the proper conclusion about its strength is hampered by the lack of rigor in the experimental design, by poor quality of research and by lack of sound theoretical ground in many of the studies. Studies which claim that significant percentage of their subjects (e.g. 70% - Irlen, 1983; 50-70% - Whiting, 1985) had visual problems reduced by the tints were either not published in refereed journals or had many methodological problems such as improper selection criteria, lack of control for the placebo effect, no control groups, small number of subjects etc. Some of the reviewed papers published in refereed journals and with better control of experimental parameters still reported differing results. In spite of several decades of intensive research, the same problems remain. In a recent systematic review of the different colour systems used to improve reading performance (Griffiths et al, 2016), it has been concluded that although some individuals might benefit from coloured lenses/overlays, the size effect is small and there is a risk that the reported improvements are placebo or practice effect. The method of prescribing individual colour filters, using Intuitive Colorimeter developed by Professor Wilkins (e.g. Wilkins et al., 2001) is more precise, but it was criticized for lack of repeatability of the colours chosen by the patients (Elliot & Wood, 2017).

The effect of coloured filters on reading has been viewed from a different perspective. Stein and his colleagues have claimed that the reading problems are related to magnocellular dysfunction. Ray et al (2005) found that wearing yellow filters improves performance in tasks that are believed to involve magnocellular activity, including reading. They assumed that S-cones have inhibitory input to the magnocellular system and by stopping the short wavelength light, the yellow filters improve the magnocellular function. These claims, however, were not confirmed in two later studies showing that S-cone inputs to both parvocellular and magnocellular system are insignificant (Sun et al., 2006; Lee et al., 2010). Later Stein and co-authors found that using a blue filter improves the reading performance even more in comparison to the yellow and introduced a two filter system (Hall et al., 2013; Harries et al., 2015; Stein, 2018). The inconsistency in the choice of colour filters used in different systems reduces the credibility in the effectiveness of the colour filter therapy as pointed out in Griffiths et al. (2016) recent review.

The discussion about the effect of colour on the reading process is not limited to alleviation of the visual stress symptoms. The effect of short-wavelength cut-off filters on visual performance has been debated for a long time and this debate continues. Although some authors report interesting effects, there is no agreement about the beneficial effects on visual skills, or the possible underlying mechanisms. The most frequently reported improvements with yellow filters include brightness enhancement, despite of the low light transmittance of the filters (e.g. Wolffsohn et al 2000; Wolffsohn et al., 2002), minor enhancement of contrast sensitivity especially in visually impaired patients (Frennesson& Nilsson, 1993; Wolffsohn et al., 2002) or faster reaction times (Kinney et al., 1983).

Most explanations consider the exclusion of the unwanted effects of the short wavelength light as underlying the observed improvements. The increase in the contrast sensitivity or spatial resolution may be related to the improved retinal image quality, due to the reduced chromatic aberration with blue-blocking filters (Sivak&Bobier, 1978). The reported increase in pupil diameter with yellow filters is due to the higher sensitivity of the pupil to short wavelength light, compared to the photopic luminosity curve (Chung & Pease, 1999). This can explain the subjective improvement in brightness despite the luminance reduction. According to Kelly (1990), the observed brightness enhancement is due to reduced rod intrusion to the chromatic pathway caused by the blue-blocking filters.

A different mechanism that might explain the role of coloured filters in dyslexia has been proposed by Northway et al (2010). The contrast threshold for letter discrimination of dyslexics and controls was similar in no-noise condition, while in a presence of visual noise, the dyslexics contrast sensitivity deteriorated more. It was restored to normal levels when wearing coloured lenses. The authors suggested that visually symptomatic dyslexics have impaired noise exclusion and that the coloured filters restore the exclusion of noise to normal level.

1.2. Discrepancies related to the Magnocellular/Dorsal deficit in dyslexia

The theory that magnocellular system deficits play a role in dyslexia is perhaps the most exploited and the most controversial among the theories that seek to explain the reading disability in dyslexia.The proposed aetiologyof magnocellular deficits is based on the separation of the visual system into distinct pathways with different roles and properties. Anatomically, the segregation between magno- (M),parvo- (P) and konio- (K) cellular pathways begins in the retina from where different types of ganglion cells send their signals to different layers in the lateral geniculate nucleus (LGN) and further to different sub-layers in striate cortex. At the cortical level, there are two main streams. The dorsal stream projects from the striate cortex to the middle temporal area (MT/V5) to the posterior parietal cortex and plays an important role in localization of visual stimuli and in visual information processing needed for understanding spatial relationships and controlling spatially directed actions.

The ventral stream projects to the inferotemporal area and supports the processing of object quality or identity (Ungerleider&Mishkin, 1982; Goodale& Milner, 1992; Milner &Goodale, 2008).

The investigators supporting the magnocellular theory postulate that a selective deficit in magnocellular pathway in dyslexic individuals results in deficiencies in visual processing and, via the posterior parietal cortex, can lead to abnormal binocular control and visuospatial attention (Stein & Walsh, 1997).Stein (2018) formulated DD as "a hereditary temporal processing defect, associated with impaired magnocellular neuronal development that impacts selectively on the ability to learn to read, leaving oral and non-verbal reasoning powers intact".

First anatomical and electrophysiological evidence for a magnocellular deficit in dyslexia was presented by Livingstone et al. (1991). Additional evidence for magnocellular dysfunction was obtained from anatomical, psychophysical and neuro-imaging studies (reviewed bySkottun, 2000; Ramus et al., 2003; Nandakumar&Leat, 2008; Vidyasagar&Pammer, 2010; Skottun, 2015; Stein, 2018). Some of the studies that describe deterioration of visual perception in dyslexia have used stimuli that do not selectively stimulate magnocellular pathway (reviewed by Skottun, 2000; Skottun, 2015; Stein, 2018). Other studies, failed to support the magnocellular deficit hypothesis. There are at least two potential sources of discrepancies between thedifferent studies.Firstly, using the term "magnocellular-dorsal deficit" introduces inconsistencies by combining subcortical magnocellular pathway and cortical dorsal stream. Secondly, the use of different stimulation conditions and parameters in different studies makes the comparison difficult.

Skottun (2015) criticised the tendency to consider the subcortical magnocellular and the cortical dorsal pathways as single entity. The author cited several studies exploring a "magnocellular-dorsal stream" deficit in dyslexia. The arguments against such approach are clear: magnocellular and parvocellular pathways are subcortical systems and their inputs into the visual cortex are substantially mixed. Although there is a segregation of the afferent fibres from magno- and parvo- systems, their signals can be mixed since the V1 cell's dendrites reach different sub-layers in layer 4. Further, the two processing streams onwards from V1, the dorsal and the ventral streams, receive substantial input from both magnocellular and parvocellular pathways (Goodale& Milner, 1992; Merigan&Maunsell, 1993). There is no one-to-one correspondence between the subcortical and cortical pathways. Although parietal stream depends largely on M pathway contributions, it also receives contributions from the P pathway (Nassi et al., 2006) while the temporal pathway receives major inputs from all subcortical pathways (Merigan&Maunsell, 1993; Ferrera et al., 1994). In addition, contrast sensitivity studies do not support magnocellular deficits while motion perception results are consistent with dorsal stream deficits in dyslexia (Skottun, 2015). The author concluded that to resolve this controversy, the magnocellular system and the dorsal stream should be regarded as separate entities. Despite Skottun's comprehensive review however, the tendency to mix subcortical and cortical paths continues.

Another issue is the use of the terms "magnocellular" and "transient" interchangeably in the literature on dyslexia. The terms 'transient' and 'sustained' are mainly used in human psychophysics (Skottun, 2015) but also extensively used to describe neurophysiology of the cat's visual system as well as primate ganglion cells which are part of M and P pathways. De Monasterio (1978) challenged such a dichotomy. He showed that the cells in the macaque retina which could be classified as X or Y cells tended to have more sustained or more transient response but these tendencies did not necessarily represent invariant properties. Vassilev (1996) suggested that the transient characteristics of the response strongly depend on light intensity and they are not inherent property of M or P cells.

Some of discrepancies about the contribution of visual magnocellular or dorsal deficits to dyslexia might be explained by differences in the testing methods or by the incorrect choice of visual stimulation parameters. Skottun (2000) reviewed many studies that intended to test magnocellular hypothesis by measuring grating contrast sensitivity. The author pointed out that it is not possible to segregate P- from M- system and to selectively stimulate only magno- system using gratings with SF higher than 1 c/deg. Skottun found that only four studies out of twenty two supported magnocellular theory in dyslexia. Eleven studies were described as providing positive evidence against this theory, and seven studies were considered as inconclusive.

1.3. Visual neural noise hypothesis and reading difficulties

Sperling et al. (2005) pointed out another problem with visual stimulation in studies showing magnocellular deficit in dyslexia, i.e. that these experiments were performed on noisy displays. Sperling et al. (2005; 2006) were probably the first to suggest that the reading impairments are not a result of magno-system defect but rather an inability to exclude the visual noise.The authors raised an alternative hypothesis based on theories of signal-noise discrimination, namely that dyslexia is associated with deficits in noise exclusion. The grating contrast thresholds measured by Sperling and co-authors in dyslexic and non-dyslexic children did not differ significantly for either M- or P-selective stimuli in the no-noise conditions. It should be noted that,in the high-noise conditions, dyslexic children's contrast thresholds were significantly higher than non-dyslexic's in both M- and P-selective tasks. However, the SF of their "M stimulus" (2 c/deg), was too high to selectively stimulate the magnocellular system, as we discussed above.

Sperling et al. (2006) presented additional support for their theory by examining noise effect in a motion-perception task. The results showed higher threshold under the high-noise condition for poor readers compared to good readers and similar performance between the two groups of observers in the low- and minimal-noise conditions. The authors interpreted the results as evidence for deficits in noise exclusion rather than in motion perception per se.

The neural noise theory is attractive because it could be applied to different sensory modalities and different hierarchical levels. The limited performance efficiency of poor readers in the presence of external noise in the sensory signals was also described in auditory modality as well. Hornickel and Kraus (2013) recorded brainstem responses to speech syllables and assessed the consistency of auditory brainstem responses in normal hearing children 6–13 years old with a wide range of reading abilities. The results obtained showed that poor readers have more variable auditory responses than good readers.

Recently, Hancock et al. (2017) formulated more general neural noise hypothesis of DD. The hypothesis is based on the evidence that low-level sensory-processing deficits (both visual and auditory) precede and underlie phonological problems. The authors suggested that neural noise stems from increased neural excitability in cortical networks. Based on the genetic risk data of dyslexia, the authors proposed two potential sources of increased neural noise that lead to hyperexcitability and disbalance of excitation and inhibition in the cortical networks: change in glutamatergicsignalling and disrupted neural migration. The first decreases the precision of spike timing, while the secondleads to abnormal structure of cortical microcircuits and synaptic connectivity. The disrupted balance of excitation and inhibition in the cortical networks affects the synchronization of neural activity and has detrimental effects on encoding speech and on multisensory integration involved in reading. Increased neural noise affects the robustness of stimulus representations and the spike timing-dependent plasticity needed to form the multisensory associations between text and speech. Another potential effect of increased noise is associated with synaptic depression needed for noise exclusion. The authors suggested that cortical hyperexcitability may disrupt the encoding of speech by affecting the synchronous neural processing and may therefore underline the phonological problems, which are well documented in reading difficulties. This hypothesis provides a biological mechanism that may explain the disrupted uni-sensory and multi-sensory processing in reading difficulties that ultimately manifests as characteristic impairments in phonological awareness and/or grapheme–phoneme mapping.

Other studies relate the disruption of multisensory integration due to impairments in noise rejection in dyslexic individuals to impaired visuospatial attention (Facoetti et al., 2010). The experimental data suggest deficiency in exploiting the relationship between a cue and visual or auditory target at short inter-stimulus intervals in dyslexic children with impaired nonwordreading accuracy i.e. for children with a phonological deficit. This result was interpreted as implying that the temporal and spatial windows of multisensory integration were broader in dyslexics than in normal readers inducing stronger interference of signal and noise in sensory processing. The impairments in noise rejection were related to the sluggish shift of spatial attention and consequently, to the reduced ability to modulate perceptual noise exclusion. The contribution of attention to reading difficulties in dyslexics was also suggested by Bosse et al. (2007) however, they supposed that it is related to the reduced visual span i.e. the amount of distinct visual elements that could be processed in a single eye movement and thus, in parallel. The authors showed that reading performance in dyslexics could be predicted by two independent measures: phonological awareness and visual attentional span.

1.4. Eye movements and reading disability

Another controversial topic is the casual link between the eye movements patterns and the reading difficulties i.e. whether abnormal eye movements are the cause (Pavlidis, 1985) or the consequence of reading difficulties (Morris & Rayner, 1991; Olson et al, 1991).

Reading involves a succession of saccades and fixations. When fixation disparity occurs due to a misalignment of the two foveas of the eyes, additional disparity-driven slow vergence movement would be involved in order to re-establish binocular eye alignment. Although saccades and fixation are the two main eye movements, involved in reading, in normal readers there is no association between the two and they utilize different aspects of the available information (Rayner &McConkie, 1976). However, correlations can be obtained locally between a given fixation duration and saccade length (Pollatsek, Rayner, &Balota, 1986), suggesting that text difficulty influences both eye movement types.

The typical amplitude of saccades depends mostly of the visual characteristics of the word – its length and the interval between the characters (Quercia et al, 2013). The saccades are predominately from left to right, but occasionally some regressive saccades from right to left occur (Olitsky& Nelson, 2003; Oliveira et al., 2013). Of the time spent in saccades, 5-20% may be leftward regressive saccades (Eden et al., 1994).

Typically, binocular coordination of saccades is not perfect. A transient divergent disconjugacy during the first part of the saccade occurs due to the larger and faster movements of abducting eye than of the adducting eye (Collewijn, Erkelens, & Steinman, 1988a, 1988b; Fioravanti, et al., 1995), while at the end of the saccade this

is reversed. A disconjugate, convergent post-saccadic drift further decreases the residual divergent misalignment between the eyes during the fixation.

The fixations are related to text comprehension (about 90-95% of the reading time) and the preparation of the next saccade (e.g. Eden et al, 1994). However, the acquired reading experience, the visual-motor, linguistic and attention factors, and the morphology of the text affect the strategy for saccades and fixations (Quercia et al, 2013).

There is considerable amount of evidence for differences in eye movements' behaviour during reading for normal and dyslexic individuals such as: an increase in saccade number, the number of fixations and their duration, the number of regressive saccades, as well as a smaller amplitude of saccades for dyslexic individuals (Rayner, 1998, review; De Luca et al, 1999; MacKeben et al., 2004; Hawelka et al, 2010; Bellocchi et al., 2013). Such differences in the pattern of eye movements during reading have been reported in different languages, irrespective of their degree of transparency (Hutzler&Wimmer, 2004).

Greater number of saccades or longer reading time for single word in dyslexics as compared to normal readers are observed predominantly for medium and long words (MacKeben et al, 2004). The word length has different effect on the number of regressive saccades (MacKeben et al, 2004) and on saccade length (De Luca et al., 2002). In dyslexic readers the number of saccades depends on the stimulus length for both words and pseudowords and the saccade amplitude is small and constant suggesting sequential sublexical reading (De Luca et al, 2002). The overall time spent on an item duringpseudoword reading is about 600 ms longer than that of controls (Hutzler et al., 2006).

Some researchers (Olson, Kliegl, & Davidson, 1983; Olson, Conners, & Rack, 1991; Hÿonä& Olson, 1995) however, demonstrated that when the task matched the reading level of the dyslexics, the eye-movement patterns were similar to that of normal readers.

To determine the link between eye movements and reading difficulties, numerous researchers used non-reading tasks that vary in the demand to oculomotor programming, procedural learning, and attentional involvement. The simplest non-reading tests are overlap and gap tasks. In the first, a fixation point is presented and while it is still on, a peripheral stimulus appears and the observer has to make a saccade towards it. In the gap paradigm (Saslow, 1967) there is a delay between the disappearance of the fixation point and the presentation of the peripheral target. The gap allows disengagement of active fixation before target appearance; this task is considered as a measure of oculomotor efficiency. The overlap task requires not only oculomotor response but also disengaging and shifting attention; it takes longer time and is related to visual orienting.

Differences between dyslexics and non-dyslexics like prolongation of the saccadic latency for peripheral target further away (Bucci et al, 2008), at intermediate viewing distance of 57 cm (Fischer & Weber, 1990) or far distance (150 cm) (Bucci et al, 2008), and reduced latency at closer distance (50 cm, Bednarek et al., 2006) were observed. However, other studies did not find such differences (e.g. Adler-Grinberg& Stark, 1978; Black et al., 1984, Lukasova et al., 2016).

The number of express saccades was also larger for the dyslexic children than for the controls in the gap condition and less - in the overlap condition (Fischer & Weber, 1990). The increased number of express saccades is a sign of weakness of fixation. Fischer and Hartnegg (2008) observed that in the overlap task at the end of the saccade in dyslexic group there were slow drifts of one or both eyes with different velocities.

Eden et al. (1994) found that dyslexics exhibit fixation instability at the end of saccades when making saccades back and forth between fixation targets. Bucci et al. (2008) observed worse binocular coordination during and after saccade in some dyslexic children. In addition, the saccadic latencies were longer for the dyslexic as compared to non-dyslexic children when the saccades were combined with vergence movements in the overlap paradigm (convergence and divergence), while in the gap paradigm this occurred only when the saccade was combined with convergence movement.

In similar vein, Raymond et al (1988) suggested that the problems in reading in dyslexics are related not to saccadic control, but to poor maintenance of fixation stability. Indeed, more unwanted saccades were observed for the dyslexics than for normal readers when fixating a white circle for 30s, especially in the last 15 s (Tiadi et al, 2016) and the fixation stability for small targets and left-eye fixation instability at 16 cm and infinity (Eden et al, 1994) was lower in dyslexics. However, deLuca et al. (1999) did not observe any difference between participants with surface dyslexia and normal readers when fixating a small white dot for 10 s.

Several studies found that the dyslexic children tend to have unstable binocular vergence control (Stein & Fowler, 1980, 1993; Bigelow & McKenzie, 1985; Simons & Grisham, 1987; Stein et al., 1988; Buzzelli, 1991; Evans et al., 1994; Kapoula et al, 2007).Stein, Riddell and Fowler (1988) observed that 64% of dyslexic children were unable to make proper convergence eye movements for small targets (of the size of the macula) but no deficiency was observed for large (7 deg) targets. However, Eden et al., (1994) found that poor vergence control occurs only for children with phonological deficit. Moreover, Ygge et al. (1993) found similar rates of accommodation, stereo-acuity, vergence problems in dyslexics and non-dyslexics.

The range of vergence was smaller in the dyslexic group compared to non-dyslexics (Evans et al, 1994; Eden et al, 1994). The amplitude of accommodation was also reduced in the dyslexic group (Evans et al., 1994).

A potential problem in reading might be due to deficient procedural motor learning. Using a task in which targets of fixed duration were presented to the left or to the right of the fixation point in a square-wave manner, Lukasova et al., (2016) showed that dyslexics made less predictive saccades and the latency reduction due to learning in the test progression is less evident.

Several studies explore tracking behaviour to differentiate the contribution of cognitive processing and motor mechanisms in the reading difficulties of dyslexic children. More saccadic intrusions during pursuit were reported for dyslexic than in non-dyslexic children (Adler-Grinberg& Stark, 1978; Black et al., 1984; Stark et al., 1990). Eden et al. (1994) observed larger number of jerky eye movements during smooth pursuit for dyslexic children for movements to the right.

The ability to program a sequence of saccades in dyslexics is studied in a task that imitates some of the characteristics of reading. A fixation stimulus presented to the left was followed by a sequence of targets with fixed delay and equal spacing to the right of it (Pavlidis, 1981; Biscaldi et al., 1998). The results of this task are contradictory: Biscaldi et al (1998) found that the dyslexic readers had a tendency to make saccades at irregular times, and an increased tendency to generate reflexive fast saccades, while De Luca et al., (1999) observed no significant differences between dyslexics of the surface type and normal readers.Similarly, Olson, Kliegland Davidson (1983) foundnodifferencesinthetotalnumberoffixations, percentageofregressions, orfixationstability.

Trauzettel-Klosinski et al. (2002) used a task of pictogram naming where the pictograms were arranged as if forming paragraphs of several lines.They observed no difference in the number of rightward saccades and a tendency for more leftward saccades in dyslexics. No significant differences in the pattern of eye movements were obtained in another reading-like task when the participants had to find two identical neighbouring letters in strings of consonants (Hutzler et al.,2006).

Deteriorated performance in some tasks like the overlap task implies impaired attention abilities in the dyslexic individuals. The relationship between saccades and visual attention was studied with the anti-saccade test (McDowell et al, 2008). In this task, at the appearance of a peripheral target, the observer has to move eyes in opposite direction. Biscaldi et al. (2000), Fischer and Hartnegg (2008) and Lukasova et al (2016) found higher number of errors, lower number of corrections and lower correction rate in anti-saccades in dyslexic readers. However, no differences in the latency of anti-saccades were observed between the two groups (Fischer & Weber, 1997; Biscaldi et al., 2000; Fischer &Hartnegg, 2008; Seassau&Bucci, 2013; Lukasova et al., 2016).These results were confirmed by Wilcockson et al. (2019), who suggested that the impaired performance in dyslexic individuals was caused by the sluggish attentional shift in this group due to a slower attentional disengagement from the target.

1.5. Comparison with other developmental disorders

As Nandakumar and Leat (2008) pointed out, not only children with a specific reading disorder (dyslexia) experience reading difficulties. Non-specific reading difficulties may also occur with other developmental disorders as ADHD or ASD (Nally et al., 2018). As already mentioned, these three conditions are often co-morbid. Indeed, both children with ADHD and ASD show abnormal eye movements that could cause reading difficulties. For example, children with ADHD show similar patterns of eye movements in reading as children with reading difficulties – greater number of saccades, shorter saccades and more frequent fixations (Deans et al, 2010). They also have more difficulty inhibiting automatic saccades, displayed longer reaction times, and greater variability (Munoz et al., 2003) in the anti-saccade task and have greater error rate (Feifel et al., 2004). As with the dyslexics, the ADHD subjects had greater difficulty to maintain fixation and made large saccades during a fixation task than controls (Gould et al, 2001). They also had longer reaction times, reduced peak velocities, increased durations of saccades in the overlap saccade task (Munoz et al,, 2003). Interestingly, individuals with ASD also showed reduced peak velocity and longer durations in a similar task (Schmitt et al, 2014). Both groups also show greater inter-subject variability.

In addition, these disorders share common hypotheses about atypical visual perception. Increased neuronal variability (neuronal noise) was not only described in dyslexia, but also in ASD (Simmons et al., 2009; Milne, 2011; Dinstein et al., 2015; Haigh et al., 2015; Weinger et al., 2014) and ADHD (Gilden& Hancock, 2007; Saville et al., 2015; Bubl et al., 2015).

The comparison of the atypical visual perception in ASD, DD and ADHD has received little attention, despite the high comorbidity of the disorders and some common deficits compared to the typical development. Mihaylova et al. (2016) compared reading speed and accuracy in visual noise in children with either DD or ASDto typically developing children.External visual noise was produced by random vertical displacement of letters in single words or pseudowords. Reading duration for each condition was calculated as the total duration of the test trials divided by the number of words correctly read. Time for reading words and pseudowords as well as error rates increased at high levels of text degradation with significant differences between observers with ASD, DD

and typically developing individuals. The positional noise effects were highest in children with ASD, lowest in control group with typical development and medium with observers with dyslexia. Moreover, different pattern of results for words and pseudowords for the separate groups of observers was found. Mihaylova et al. (2016) therefore suggested that there is a specific noise effect within each disorder. The authors hypothesized that increased neuronal variability affects specific processes and structures in each disorder that is consistent with the different reading difficulties. It is still an open question what is the specific neural process(es) in each disorder that affects the reported reading disability. A comparative study of reading performance of developmental disorders such as DD, ASD and ADHD would allow to assess the specificity of visual dysfunctions in each separate disorder and its contribution to degraded reading skills. Current research explores this hypothesis.

II. Discussion

The theories of reading disabilities, for the most part, consider separate mechanisms while ignoring their interaction. The visual contribution, which is still hotly debated, is often opposed to the phonological deficits instead of considering it as a component of more general multistage disorder. Some of the difficulties arise from the complexity of the reading process and from the heterogeneity of the population with reading disabilities, e.g. a presence of specific type of deficits in different individuals.

Evidence of the involvement of visual stress in reading is controversial. Currently, there is consensus that visual stress and dyslexia are independent but co-occurring conditions and that only a small subset of dyslexics (about 30%, depending on diagnostic criteria) report symptoms of visual stress.

Some authors have suggested that the deficient visual processing underlying visual stress, is not restricted to grating patterns and text and that its mechanisms can extend beyond the hyperexcitability at early cortical levels (Conlon & Humphreys, 2001; Kruk et al, 2008). Individuals with high visual discomfort showed slower performance in a visual search task, when it required higher level of attention (Conlon & Humphreys, 2001). The authors suggested that sensory sensitivity is increased when viewing multiple stimuli, not just gratings. They also suggested that poor ability to focus spatial attention caused the inefficient visual search of people with high visual discomfort.

Careful review of the published literature on the magnocellular theory of dyslexia shows that many studies report visual deficits. The question as to whether they are due to visual magnocellular pathway dysfunction alone is, as yet unresolved. These deficits correlate with phonological skills of observers, but cannot be explained solely by phonological theory.

It should be pointed out that verification of any visual hypothesis in reading difficulties has to be accompanied by very careful choice of stimulus parameters. The criticism of the magnocellular deficit theory is very often a response to inconsistency in the experimental design or incorrect interpretation. The terms transient system, magnocellular pathway and dorsal cortical stream cannot be used interchangeably because they refer to differing, although overlapping structures with different characteristics.

Visual magnocellular and phonological theories about dyslexia are sometimes artificially opposed. As Ramus et al. (2003) rightly pointed out, the visual theory does not exclude a phonological deficit, but rather accentuate on a visual contribution to reading problems, at least in some dyslexic individuals. The study of Cornelissen et al. (1998) mostly cited as a strong support of magnocellular hypothesis, is in fact consistent with the idea that impaired magnocellular visual function, together with phonological deficits, affect children's reading abilities. While the review article of Vidyasagar&Pammer (2010) localized the core deficit of dyslexia within the visual system, it does not reject the contribution of the phonological deficits, either. The authors rather suggested that phonological problems arise from poor visual coding. Similarly, Seidenberg's (2005) model considered the contribution of both phonological and visual pathways and their interaction. A complex multistage process such as reading cannot be explained by factors which involve a single stage. As Francisco et al. (2017) suggested, reading impairment may be a result of joined audiovisual processing deficit. The authors reported results which were consistent with an audiovisual temporal deficit in dyslexia and were not specific to speech-related events.

Neural noise plays an important role in both visual and auditory perception and its variability can affect reading abilities. More research is necessary in order to elucidate its role in reading. Comparison between different developmental disorders, which experience reading difficulties, may help to reveal fine mechanisms of reading failure in each of them,which will help in choosing the most appropriate individual treatment.

The intensive research on the relation of eye movements and reading difficulties still could not resolve the issue of the casual link between them. While differences in the eye movement characteristics of the dyslexic readers are observed even in tasks that do not require linguistic processing, the evidence is contradictory and depends on the reading level. Observed abnormalities in eye movements may be related to factors such as oculomotor programming, procedural learning, fixation instability, low binocular coordination, deficits in attentional processes or to some spatial disorder. Some of the contradictions may be related to methodological differences and to the different involvement of oculomotor, sensory and attentional factors. Nevertheless, a

potential explanation for the great diversity of findings could be the heterogeneity of the population with reading disability.

Surprisingly, some aspects of eye movement patterns, related to the mechanisms of visual stability and continuity of perception across saccades, are neglected. Phenomena like saccadic suppression (e.g. Mitrani, Mateeff&Yakimoff, 1970), peri-saccadic shift (e.g. Mateeff, 1978) and peri-saccadic compression (e.g. Ross, Morrone& Burr, 1997) occur during saccade preparation and the transfer of information across it. The first of these phenomena refers to the reduced sensitivity during the time of saccade. The other two are related to mislocalization of a brief target presented during the saccade; they differ in their temporal characteristics and sensitivity to contrast and luminance of the flashed target. Neither of these phenomena was studied experimentally with dyslexic individuals, even though reduced saccadic suppression was hypothesized in the magnocellular theory of dyslexia (e.g. Lovegrove, Martin &Slaghuis, 1986; Breitmeyer, 1993). Data in relation to the time course of these phenomena and the size of the effects may shed some light on the role of eye movements in reading disability.

Acknowledgments

This work was supported by the National Science Fund of Bulgaria, Grant DN 15/6 from 2017.

References

[1]. Adler-Grinberg, D., & Stark, L. (1978). Eye movement, scan paths, and dyslexia. American Journal of Optometry and Physiological Optics, 55, 557-570
[2]. Bednarek, D. B., Tarnowski, A., &Grabowska, A. (2006). Latencies of stimulus-driven eye movements are shorter in dyslexic subjects. Brain and Cognition, 60(1), 64–69.
[3]. Bellocchi,S., Muneaux, M., Bastien-Toniazzo, M., &Ducrot, S.(2013).I can read it in your eyes: what eye movements tell us about visuo-attentional processes in developmental dyslexia. Res.Dev.Disabil. 34,452–460.doi: 10.1016/j.ridd.2012.09.002
[4]. Bigelow ER, McKenzie BE. (1985). Unstable ocular dominance and reading ability. Perception, 14,329-335.
[5]. Biscaldi, M., Fischer, B., &Hartnegg, K. (2000). Voluntary saccadic control in dyslexia. Perception, 29(5), 509–521
[6]. Biscaldi, M., Gezeck, S., &Stuhr, V. (1998). Poor saccadic control correlates with dyslexia. Neuropsychologia, 36(11), 1189–1202.
[7]. Black, J. L., Collins, D. W., De Roach, J. N., &Zubrick, S. R. (1984). Smooth pursuit eye movements in normal and dyslexic children. Perceptual and Motor Skills, 59(1), 91–100.
[8]. Bosse, M.L.,Tainturier, M.J.,Valdois, S. (2007). Developmental dyslexia: The visual attention span deficit hypothesis. Cognition, 104,198-230.
[9]. Breitmeyer, B. G. (1993). Sustained (P) and transient (M) channels in vision: A review and implications for reading. In Visual Processes in Reading and Reading Disabilities Eds Willows, D M, Kruk, R S, Corcos, E(Hillsdale, NJ: Lawrence Erlbaum Associates) pp 95–110.
[10]. Bubl, E., Dörr, M., Riedel, A., Ebert, D., Philipsen, A., Bach, M., & van Elst, L. T. (2015). Elevated Background Noise in Adult Attention Deficit Hyperactivity Disorder Is Associated with Inattention. PLoSONE, 10(2),:0118271.doi:10.1371/journal.pone.0118271.
[11]. Bucci, M. P.,Brémond-Gignac, D., and Kapoula, Z. (2008). Latency of saccades and vergence eye movements in dyslexic children. Experimental Brain Research, 188, 1–12. doi:10.1007/s00221-008-1345-5.
[12]. Buzzelli, A. R. (1991). Stereopsis, accommodative and vergence facility: do they relate to dyslexia? Optometry and Vision Science, 68, 842 – 846.
[13]. Butterworth, B. &Kovas, Y. (2013) Understanding neurocognitive developmental disorders can improve education for all. Science, 340, 300 - 305.
[14]. Chouinard, B. D., Zhou, C. I., Hrybouski, S., Kim, E. S., &Cummine, J. (2012). A functional neuroimaging case study of Meares-Irlen syndrome/visual stress (MISViS). Brain Topography. 25, 293-307.
[15]. Chung, S. T. L., & Pease, P. L. (1999). Effect of yellow filters on pupil size. Optometry and Visual Science. 76, 59–62.
[16]. Collewijn, H., Erkelens, C.J., Steinman, R.M. (1988a). Binocular co-ordination of human horizontal saccadic eye movements. Journal of Physiology (London), 404, 157-182.
[17]. Collewijn, H., Erkelens, C.J., Steinman, R.M. (1988b). Binocular co-ordination of human vertical saccadic eye movements. Journal of Physiology (London), 404, 183-197.
[18]. Conlon, E., & Humphreys, L. (2001). Visual search in migraine and visual discomfort groups. Vision Research, 41, 3063–3068.
[19]. Conlon, E. G., Lovegrove, W. J., Hine, T., Chekaluk, E., Piatek, K, & Hayes-Williams, K. (1998). The effect of visual discomfort and pattern structure on visual search. Perception, 27, 21-33.
[20]. 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, 38, 471-482.
[21]. Creavin, A. L., Lingham, R., Steer, C., & Williams, C. (2015). Ophthalmic abnormalities and reading impairment. Pediatrics, 135, 1058-1065.
[22]. Deans, P., O'Laughlin, L., Brubaker, B., Gay, N., & Krug, D. (2010). Use of Eye Movement Tracking in the Differential Diagnosis of Attention Deficit Hyperactivity Disorder. (ADHD) and Reading Disability, Psychology, 1, 238–246.
[23]. De Luca, M., Borrelli, M., Judica, A., Spinelli, D., &Zoccolotti, P. (2002). Reading words and pseudowords: An eye movement study of developmental dyslexia. Brain and Language, 80(3), 617–626.
[24]. De Luca, M., Di Pace, E., Judica, A., Spinelli, D., &Zoccolotti, P. (1999). Eye movement patterns in linguistic and non-linguistic tasks in developmental surface dyslexia. Neuropsychologia, 37(12), 1407–1420.
[25]. Dinstein, I., Heeger D. J., Behrmann M. (2015). Neural variability: friend or foe? Trends in Cognitive Sciences, 19, 322-328.
[26]. Eden, G. F., Stein, J. F., Wood, H. M., & Wood, F. B. (1994). Differences in eye movements and reading problems in dyslexic and normal children. Vision Research, 34(10), 1345–1358.
[27]. Elliot, D. B., & Wood, J. M. (2017). Coloured filters show gender differences and poor repeatability. Ophthalmic and Physiological Optics.37, 635-639. https://doi.org/10.1111/opo.12417.
[28]. Evans, B. J., Allen, P. M., Wilkins, A. J. (2017). A Delphi study to develop practical diagnostic guidelines for visual stress (pattern-related visual stress). Journal of Optometry. 10, 161-168.

[29]. Evans, B. J., Drasdo, N. (1991). Tinted lenses and related therapies for learning disabilities--a review. Ophthalmic, & Physiological Optics, 11, 206-217.
[30]. Evans, B. J. W., Drasdo, N., & Richards, I. L. (1994). Investigation of accommodative and binocular function in dyslexia. Ophthalmic and Physiological Optics, 14, 5-19.
[31]. Evans, B. J., & Stevenson, S. J. (2008). The Pattern Glare Test: a review and determination of normative values. Ophthalmic and Physiological Optics, 28(4), 295-309.
[32]. Evans, B. J., Wilkins, A. J., Brown, J., Busby, A., Wingfield, D., Jeanes, R., & Bald, J. A. (1996). Preliminary investigation of the aetiology of Meares - Irlen Syndrome. Ophthalmic, & Physiological Optics, 16, 286-296.
[33]. Facoetti, A. Trussardi, A. N., Ruffino, M., Lorusso, M. L., Cattaneo, C., Galli, R., Molteni. M., Zorzi, M. (2010). Multisensory spatial attention deficits are predictive of phonological decoding skills in developmental dyslexia. Journal of Cognitive Neuroscience, 22, 1011–1025.
[34]. Feifel, D., Farber, R., Clementz, B. Perry, W., &Anllo-Vento, L. (2004). Inhibitory Deficits in Ocular Motor Behavior in Adults with Attention-Deficit Hyperactivity Disorder. Biological Psychiatry, 56 (5), 333-340.
[35]. Ferrera, V. P., Nealey, T. A., &Maunsell, J. H. R. (1994). Responses in macaque visual area V4 following inactivation of the parvocellular and magnocellular LGN pathways. Journal of Neuroscience, 14, 2080–2088.
[36]. Fernandez, D., & Wilkins, A. J. (2008). Uncomfortable images in art and nature. Perception, 37, 1098-1113.
[37]. Fischer B, Hartnegg K. (2008). Saccade control in dyslexia: Development, deficits, training and transfer to reading. Optometrists in Vision Development, 39(4), 181-190.
[38]. Fischer, B., & Weber, H. (1990). Saccadic reaction times of dyslexic and age-matched normal subjects. Perception, 1990, 19(6), 805-18.
[39]. Fischer, B., & Weber, H. (1997). Effects of stimulus conditions on the Performance of antisaccades in man. Experimental Brain Research, 116, 191–200.
[40]. Fioravanti, F., Inchingolo, P., Pensiero, S., &Spanio, M. (1995). Saccadic eye movement conjugation in children. Vision Research, 35, 3217–3228.
[41]. Francisco, A. A., Jesse A., Groen, M. A., & McQueen, J. M. (2017). A general audiovisual temporal processing deficit in adult readers with dyslexia. Journal of Speech, Language, and Hearing Research, 60, 144-158.
[42]. Frennesson, C., & Nilsson, U. L. (1993) Contrast sensitivity peripheral to an absolute central scotoma in age-related macular degeneration and the influence of a yellow or an orange filter. DocumentaOphthalmologica, 84, 135–144.
[43]. Gilden, D.L., & Hancock, H. (2007). Response variability in attention-deficit disorders. Psychological Science, 18,796-802.
[44]. Goodale, M. A., & Milner, A. D. (1992). Separate visual pathways for perception and action. Trends in Neurosciences, 15, 20-25.
[45]. Gori, S., &Facoetti, A. (2015). How the visual aspects can be crucial in reading acquisition? The intriguing case of crowding and developmental dyslexia. Journal of Vision, 15(1):15.1.8. doi: 10.1167/15.1.8.
[46]. Gould, T. D., Bastain, T. M., Israel, M. E., Hommer, D. W., & Castellanos, F. X. (2001). Altered Performance on an Ocular Fixation Task in Attention-Deficit/Hyperactivity Disorder. Biological Psychiatry, 50 (8), 633-635.
[47]. Griffiths, P. G., Taylor, R. H., Henderson, L. M., & Barrett, B. T. (2016). The effect of coloured overlays and lenses on reading: a systematic review of the literature. Ophthalmic and Physiological Optics, 36, 519-544.
[48]. Haigh, S.M., Heeger, D.J., Dinstein, I., Minshew, N., &Behrmann, M. (2015). Cortical variability in the sensory-evoked response in autism. Journal of Autism and Developmental Disorders, 45, 1176–1190.
[49]. Hall, R., Ray, N., Harries. P., & Stein, J. (2013). A comparison of two-coloured filter systems for treating visual reading difficulties. Disability and Rehabilitation. 35, 2221-2226.
[50]. Hancock, R., Pugh, K. R., &Hoeft, F. (2017). Neural Noise Hypothesis of Developmental Dyslexia. Trends in Cognitive Science, 21, 434-448.
[51]. Harries, P., Hall, R., Ray, N., & Stein, J. (2015). Using coloured filters to reduce the symptoms of visual stress in children with reading delay. Scandinavian Journal of Occupational Therapy, 22,153–160.
[52]. Hawelka S., Gagl B., Wimmer H. (2010). A dual-route perspective on eye movements of dyslexic readers. Cognition, 115(3):367–379.
[53]. Henderson, L. M., Taylor, R., Barrett, B., et al. (2014). Editorials: treating reading difficulties with colour. British Medical Journal, 349:g5160-g5260.3.
[54]. Hornickel, J., & Kraus, N. (2013). Unstable Representation of Sound: A Biological Marker of Dyslexia. The Journal of Neuroscience, 33, 3500–3504.
[55]. Huang, J., Cooper, T. G., Satana, B., Kaufman, D. I., &Cao,Y. (2003).Visual distortion provoked by a stimulus in migraine associated with hyperneuronal activity. Headache, 43, 664–671.
[56]. Hutzler, F., Kronbichler, M., Jacobs, A. M., &Wimmer, H. (2006). Perhaps correlational but not causal: No effect of dyslexic readers' magnocellular system on their eye movements during reading. Neuropsychologia, 44(4), 637–648.
[57]. Hutzler, F., &Wimmer, H. (2004). Eye movements of dyslexic children when reading in a regular orthography. Brain and Language, 89(1), 235–242.
[58]. Hÿonä, J., & Olson, R. K. (1995). Eye fixation patterns among dyslexic and normal readers: Effects of word length and word frequency. Journal of Experimental Psychology: Learning, Memory and Cognition, 21(6), 1430–1440.
[59]. Irlen, H. (1983). Successful treatment of learning difficulties. Paper presented at the Annual Convention of the American Psychological Association, Anaheim, California.
[60]. Irlen, H., & Lass, M. J. (1989). Improving reading problems due to symptoms of scotopic sensitivity syndrome using Irlen lenses and overlays. Education, 109, 413–417.
[61]. Kapoula, Z., Bucci, M.P., Jurion, F., Ayoun, J., Afkhami, F., & Bremond-Gignac, D. (2007). Evidence for frequent divergence impairment in French dyslexic children: deficit of convergence relaxation or of divergence per se? Graefes Arch ClinExpOphthalmol, 245 (7), 931-936.
[62]. Kelly, S. A. (1990) Effect of yellow-tinted lenses on brightness. Journal of the Optical Society of America. A, 7, 1905–1911.
[63]. Kinney, J. A., Schlichting, C. L., Neri, D. F., & Kindness, S. W. (1983). Reaction time to spatial frequencies using yellow and luminance-matched neutral goggles. American Journal of Optometry and Physiological Optics, 60,132–138.
[64]. Kriss, L., & Evans, B. J. W. (2005). The relationship between dyslexia and Meares-Irlen Syndrome. Journal of Research in Reading, 28, 350-364.
[65]. Kruk, R., Sumbler, K., &Willows, D. (2008). Visual processing characteristics of children with Meares-Irlen syndrome. Ophthalmic and Physiological Optics. 28, 35-46. doi: 10.1111/j.1475-1313.2007.00532.x.
[66]. Lee, B. B., Martin, P. R ., &Grünert, U. (2010). Retinal connectivity and primate vision. Progress in Retinal and Eye Research, 29, 622-639.

[67]. Livingstone, M. S., Rosen, G. D., Drislane, F. W., &Galaburda, A. M. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proceedings of the National Academy of Sciences of the United States, 88, 7943–7947.

[68]. Longhurst, R. (2017). Is There a Common Aetiology for Dyslexia, Visual Stress, and Pattern Glare? Optometry, & Visual Performance, 5, 57-66.

[69]. Lovegrove, W., Martin, F., &Slaghuis, W. (1986). A theoretical and experimental case for a visual deficit in specific reading disability. Cognitive Neuropsychology, 3,. 225-267

[70]. Lukasova, K., SilvaI, P. &Macedo, E. C. (2016). Impaired Oculomotor Behavior of Children with Developmental Dyslexia in Antisaccades and Predictive Saccades Tasks. Frontiers in Psychology, 7, 987. doi: 10.3389/fpsyg.2016.00987

[71]. MacKeben, M., Trauzettel-Klosinski, S., Reinhard, J., Durrwachter, U., Adler, M., &Klosinski, G. (2004). Eye movement control during single-word reading in dyslexics. Journal of Vision, 4(5), 388–402.

[72]. Mateeff S. (1978). Saccadic eye movements and localization of visual stimuli. Perception and Psychophysics, 24, 215–224.

[73]. McDowell, J. E., Dyckman, K. A., Austin, B. P., &Clementz, B.A. (2008). Neurophysiology and neuroanatomy of reflexive and volitional saccades: evidence from studies of humans. Brain and Cognition, 68, 255–270.doi: 10.1016/j.bandc.2008.08.016

[74]. Merigan, W. H., &Maunsell, J. H. R. (1993). How parallel are the primate visual pathways? Annual Review of Neuroscience, 16, 369-402.

[75]. Mihaylova, M., Shtereva, K., Hodzhev, Y., Manahilov, V. (2016). Reading in visual noise in developmental dyslexia and autism spectrum disorders. 39th European Conference on Visual Perception (ECVP) 2016 Barcelona, Perception 2016, Supplement, 15. sagepub.co.uk/journalsPermissions.nav, DOI: 10.1177/0301006616671273.

[76]. Milne, E. (2011). Increased intra-participant variability in children with autistic spectrum disorders: evidence from single-trial analysis of evoked EEG. Frontiers in Psychology, 2, 1–12.

[77]. Milner, A. D., &Goodale, M. A. (2008). Two visual systems re-viewed. Neuropsychologia, 46(3), 774-785.

[78]. Mitrani L, Mateeff S, Yakimoff N. (1970). Temporal and spatial characteristics of visual suppression during voluntary saccadic eye movement. Vision Research, 10(5), 417-422.

[79]. Monasterio, F. M. de (1978). Properties of concentrically organized X and Y ganglion cells of macaque retina. Journal of neurophysiology, 41, 1394-1417.

[80]. Monger, L. J, Shah, D., Wilkins, A. J. et al. (2016). The effect of viewing distance on response to the pattern glare test. Clinical and Experimental Optometry, 99, 47–50.

[81]. Morris, R. K., & Rayner, K. (1991). Eye movements in skilled reading: Implications for developmental dyslexia. In J. F. Stein (Ed.), Vision and visual dyslexia (pp. 233-242). Boston: CRC Press.

[82]. Munoz, D., Armstrong, I., Hampton, K., & Moore, K. (2003). Altered Control of Visual Fixation and Saccadic Eye Movements in Attention-Deficit Hyperactivity Disorder. Journal of Neurophysiology, 90 (1), 503-515.

[83]. Nally. A., Healy, O., Holloway, J., &Lydona, H. (2018). An analysis of reading abilities in children with autism spectrum disorders. Research in Autism Spectrum Disorders, 47, 14-25.

[84]. Nandakumar, K., &Leat, S. J. (2008). Dyslexia: a review of two theories. Clinical and Experimental Optometry, 91, 333–340.

[85]. Nassi, J. J., Lyon, D. C., & Callaway, E. M. (2006). The parvocellular LGN provides a robust disynaptic input to the visual motion area MT. Neuron, 50, 319–327.

[86]. Northway, N., Manahilov, V., & Simpson, W. (2010). Coloured filters improve exclusion of perceptual noise in visually symptomatic dyslexics. Journal of Research in Reading, 33, 223–230.

[87]. Olitsky, S.E., and Nelson, L. B. (2003). Reading disorders in children. Pediatric Clinics of North America, 50, 213–224. Available online at: www.pediatric.theclinics.com/article/S0031-3955(02)00104-9/abstract

[88]. Oliveira, D. G., Mecca, T. P., daSilva, P. B. ,Pinto, I. S., and Macedo, E. C. (2013). Text complexity and eye movements measures in adults readers. RevistaPsicologia: Teoria e Prática, 15 (3), 163-174, Available online at:http://pepsic.bvsalud.org/pdf/ptp/v15n3/12.pdf

[89]. Olson, R. K., Conners, F. A., & Rack, J. P. (1991). Eye movements in dyslexia and normal readers. In J. F. Stein (Ed.), Vision and visual dyslexia (pp. 243–250). London: Macmillan.

[90]. Olson, R. K., Kliegl, R., & Davidson, B. J. (1983). Eye movements in reading disability. In K. Rayner (Ed.), Eye movements in reading: Perceptual and language processes (pp. 467–480). New York: Academic Press.

[91]. Pavlidis, G. T. (1981). Do eye movements hold the key to dyslexia? Neuropsychologia, 19(1), 57–64.

[92]. Pavlidis, G. T. (1985). Eye movements in dyslexia: Their diagnostic signiffcance. Journal of Learning Disabilities, 18(1), 42-50.

[93]. Pollatsek, A., Rayner, K., &Balota, D.A. (1986). Inferences about eye movement control from the perceptual span in reading. Perception, & Psychophysics, 40, 123 - 130.

[94]. Quercia, P., Feiss, F., & Michel, C. (2013). Developmental dyslexia and vision. Clinical Ophthalmology, 7, 869–881.

[95]. Ramus, F., Rosen, S., Dakin, S. C., Day, B. L., Castellote, J. M., White, S., & Frith, U. (2003). Theories of developmental dyslexia: insights from a multiple case study of dyslexic adults, Brain, 126, 841-865.

[96]. Ray, N. J., Fowler, S., & Stein, J. F. (2005).Yellow Filters Can Improve Magnocellular Function: Motion Sensitivity, Convergence, Accommodation, and Reading. Annals of the New York Academy of Science, 1039, 283–293.

[97]. Raymond, J. E., Ogden, N. A., Pagan, J. E., and Kaplan, B. J. (1988). Fixational instability in dyslexic children. American Journal of Optometry and Physiological Optics, 65, 174-181.

[98]. Rayner, K. (1998). Eye movements in reading and information processing: 20 years of research. Psychological Bulletin, 124, 372–422.

[99]. Rayner, K., &McConkie, G. W. (1976). What guides a reader's eye movements.Vision Research, 16, 829-837.

[100]. Ross J., Morrone M. C., Burr, D. (1997). Compression of visual space before saccades. Nature 384, 598–601. doi:10.1038/386598a0

[101]. Saslow, M.G. (1967). Effects of components of displacement-step stimuli upon latency for saccadic eye movements. Journal of the Optical Society of America, 57,1024–1029.

[102]. Schmitt, L. M., Cook, E. H., Sweeney,J. A., Mosconi, M. W. (2014). Saccadic eye movement abnormalities in autism spectrum disorder indicate dysfunctions in cerebellum and brainstem. Molecular Autism, 5(1),47. https://doi.org/10.1186/2040-2392-5-47.

[103]. Seassau, M., &Bucci, M. P. (2013). Reading and visual search: a developmental study in normal children. PLoSONE 8:e70261.doi: 10.1371/journal.pone.0070261

[104]. Saville, C.W.N., Feige, B., Kluckert, C., Bender, S., Biscaldi, M., Berger, A., Fleischhaker, C., Henighausen, K., & Klein, C. (2015). Increased reaction time variability in attention-deficit hyperactivity disorder as a response-related phenomenon: evidence from single-trial event-related potentials. Journal of Child Psychology and Psychiatry, 56, 801-813.

[105]. Seidenberg, M. S. (2005). Connectionist Models of Word Reading. Current Directions in Psychological Science, 14 (5), 238-242.

[106]. Simmers, A. J., Gray, L. S., & Wilkins, A. J. (2001). The influence of tinted lenses upon ocular accommodation. Vision Research, 41, 1229–1238.
[107]. Simmons, D. R., Robertson, A. E., McKay, L. S., Toal E., McAleer, P., &Pollick, F. E. (2009) Vision in autism spectrum disorders. Vision Research, 49, 2705–2739.
[108]. Simons H, & Grisham J. (1987). Binocular anomalies and reading problems. Journal of the American Optometric Association, 58(7), 578-587.
[109]. Singleton, C.H.,&Trotter, S. (2005). Visualstressinadultswithandwithoutdyslexia. JournalofResearchinReading, 28, 365-378.
[110]. Sivak, J. G., &Bobier, W. R. (1978). Effect of a yellow ocular filter on chromatic aberration: the fish eye as an example. American Journal of Optometry and Physiological Optics, 55, 813–817.
[111]. Skottun, B. C. (2000). The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. Vision Research, 40, 111–127.
[112]. Skottun, B. C. (2015). The need to differentiate the magnocellular system from the dorsal stream in connection with dyslexia. Brain and Cognition, 95, 62-66.
[113]. Sperling, A.J., Lu, Z.-L., Manis, F.R., & Seidenberg, M.S. (2005). Deficits in perceptual noise exclusion in developmental dyslexia. Nature Neuroscience, 9, 862–863.
[114]. Sperling, A. J., Lu, Z. L., Manis, F. R., & Seidenberg, M. S. (2006). Motion-perception deficits and reading impairment: it's the noise, not the motion. Psychological Science, 12, 1047-1053.
[115]. Stark, L. W., Giveen, S. C., &Terdiman, J. F. (1990). Specific dyslexia and eye movements. In Stein, J. F. (Ed.), Vision and visual dyslexia. London: Macmillan Press.
[116]. Stein, J. (2018). What is Developmental Dyslexia? Brain Science, 8, 26. doi: 10.3390/brainsci8020026.
[117]. Stein, J.F., and Fowler, M. S.(1980). Visual dyslexia. British Orthoptic Journal, 37, 11-15.
[118]. Stein, J.F., and Fowler, M. S.(1993). Unstable binocular control in children with specific reading retardation. Journal of Research in Reading, 16, 30–45.
[119]. Stein, J.F., Riddell, P. M., Fowler, S. (1988). Disordered vergence control in dyslexic children. British Journal of Ophthalmology, 72(3), 162-166.
[120]. Stein, J., & Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. Trends in Neuroscience, 20, 147-152.
[121]. Sun, H, Smithson, H. E., Zaidi, Q., Lee, B. B. (2006). Specificity of cone inputs to macaque retinal ganglion cells. Journal of Neurophysiology, 95, 837–849
[122]. Tiadi, A., Gérard, C.L., Peyre, H., Bui-Quoc, E. and Bucci, M.P. (2016). Immaturity of Visual Fixations in Dyslexic Children. Frontiers in Human Neuroscience, 10, 58, doi: 10.3389/fnhum.2016.00058
[123]. Trauzettel-Klosinski, S., MacKeben, M., Reinhard, J., Feucht, A., Durrwachter, U., &Klosinski, G. (2002). Pictogram naming in dyslexic and normal children assessed by SLO. Vision Research, 42(6), 789–799.
[124]. Uccula, A., Enna, M., &Mulatti, C. (2014). Colors, colored overlays, and reading skills. Frontiers of Psychology, 5, 833.
[125]. Ungerleider, L.G., &Mishkin, M. (1982).Two Cortical Visual Systems. In: Ingle DJ, et al., editors. Analysis of Visual Behavior. The MIT Press, 549–586.
[126]. Vassilev A. (1996). Serial processing of visual signals and the parallel visual pathways. In: Perspectives on Cognitive Sciences (Ed. B. Kokinov). NBU Series in Cognitive Sciences vol. 2, 162-168.
[127]. Vidyasagar, T., &Pammer, K. (2010). Dyslexia: a deficit in visuo-spatial attention, not in phonological processing. Trends in Cognitive Sciences, 14, 57-63.
[128]. Weinger, P.M., Zemon, V., Soorya, L., Gordon, J. (2014). Low-contrast response deficits and increased neural noise in children with autism spectrum disorder. Neuropsychologia, 63, 10–18.
[129]. Whiting, P. R. (1985). How difficult can reading be? New insight into reading problems. Journal of the English Teachers Association, 49, 49-55.
[130]. Wilkins, A. J., &Nimmo-Smith, M. I. (1984). On the reduction of eye-strain when reading. Ophthalmic, & Physiological Optics, 4, 53–59.
[131]. Wilkins, A. J., Nimmo-Smith, I., Tait, A., McManus, C., Della Sala, S., Tilley, A., Arnold, K., Barrie, M., & Scott, S. (1984) A neurological basis for visual discomfort. Brain, 107, 989–1017.
[132]. Wilkins, A. J. (1995). Visual Stress. Oxford: Oxford University Press.
[133]. Wilkins, A. J., Lewis, E., Smith, F., Rowland, E., &Tweedie, W. (2001). Coloured overlays and their benefits for reading. Journal of Research in Reading, 24, 41-64.
[134]. Wilkins, A., Huang, J., & Cao, Y. (2004). Visual stress theory and its application to reading and reading tests. Journal of Research in Reading, 27, 152-162.
[135]. Wilkins, A., & Evans, B. J. W. (2010). Visual stress, its treatment with spectral filters, and its relationship to visually induced motion sickness. Applied Ergonomics, 41, 509-515.
[136]. Wilkins, A.J., Allen, P.M., Monger, L. and Gilchrist, J. (2016). Visual stress and dyslexia for the practising optometrist. Optometry in Practice, 17, 103-112.
[137]. Wilcockson, T.W.D., Mardanbegi, D., Sawyer, P., Gellersen, H., Xia, B. and Crawford T.G. (2019). Oculomotor and Inhibitory Control in Dyslexia. Frontiers in systems neuroscience. 08 January 2019 | https://doi.org/10.3389/fnsys.2018.00066
[138]. Wilson, C. (2015). Forget colour overlays – dyslexia is not a vision problem. New Scientist. [online] Available at: https://www.newscientist.com/article/dn27588-forget-colour-overlays-dyslexia-is-not-a-vision-problem/ [Accessed 30 May 2018].
[139]. Wolffsohn, J. S., Cochrane, A. L., Khoo, H., Yoshimitsu, Y., & Wu, S. (2000). Contrast is enhanced by yellow lenses because of selective reduction of short wavelength light. Optometry and Visual Science, 77, 73–81.
[140]. Wolffsohn, J. S., Dinardo, C. &Vingrys, A. J. (2002). Benefit of coloured lenses for age-related macular degeneration. Ophthalmic & Physiological Optics, 22, 300–311.
[141]. Ygge, J., Lennerstrand, G., Axelsson, I., & Rydberg, A. (1993). Visual functions in a Swedish population of dyslexic and normally reading children. ActaOphthalmologica(Copenh), 71(1), 1-9.

Milena SlavchevaMihaylova. "Visual Deficits and Reading Failure" IOSR Journal of Computer Engineering (IOSR-JCE) 21.3 (2019): 01-12.