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AN OVERVIEW ON COLOR OVERLAYS

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Abstract:
The overlays used by the Irlen method and the Intuitive system are A4 plastic sheets, with one 'shiny' and one 'matte' side. Wilkins, on the basis of his group's research, concurs with Irlen's opinion that filters should be individually-prescribed, as there exists an optimal color for each individual which will reduce their visual stress symptoms and facilitate their reading to the greatest extent (Wilkins, 2003; Wilkins et al. 2005; though see Simmers et al., 2001). The basic set of colors provided by the Irlen method and the Intuitive system are broadly similar (the chromaticity of the Intuitive Overlays, along with other details, can be found in Wilkins, 1994). In both systems, a patient's optimal overlay is chosen using a process of elimination, often combined with questions relating to any visual stress symptoms the patient may be suffering from. This study describes the history of color overlays, the use of them as a treatment and various theoretical mechanisms which potentially explain the effects of colored filters on reading such as magnocellular theory.

Keywords: color overlays, Meares-Irlen syndrome, magnocellular theory, dyslexia

1. The history of color as a treatment

The use of light and color for their apparent healing properties has a history stretching back to at least the ancient Greeks and Egyptians (Gottlieb & Wallace, 2001). Many authors have gone beyond the effects color is popularly considered to have on mood (for which surprisingly little scientific evidence can be found - see e.g. Elliot et al., 2007 for an example), and made further claims. Several physicians in the latter part of the 19th Century, for instance, proposed many color-based therapeutic interventions. Collins (2002) recounts the story of A. J. Pleasonton, who, in the mid-19th Century, became famous for his attempts to cure many illnesses (and aid plant growth) using

light shone through blue glass. Babbitt (1878) proposed that colored light, dyes and lenses could be used not only to heal various disorders of the eye, but also a wide range of other medical conditions. Babbitt's pseudoscientific volume 'Principles of Light and Color' advocated the use of color as a panacea, a tradition that has been continued in various forms of 'complementary/alternative' and 'New Age' healing techniques; see Wauter (1999) for one example, involving the creation of homeopathic remedies using colored light shone through water. Needless to say, more recent color-based interventions are prima facie, far more plausible than these historical treatments.

More focused interventions for learning disabilities began with Henning's 'chrome-orthoptics', (Henning, 1936) and involved the use of light therapy, along with lenses and prisms, to affect the accommodative reflex of the eye. This was hypothesized to affect the autonomic nervous system, resulting in better concentration and improved learning. Although no scientific evidence was provided by Henning for the efficacy of this intervention or its mechanisms, the basic treatment methodology forms the basis for contemporary 'syntonics' (also referred to as 'light therapy' or 'photoretinology' – Howell & Stanley, 1988; College of Syntonic Optometry, 2009), a type of vision therapy named by Spitler (1941) which purports to 'balance' the visual system (Barrett, 2009).

Several studies have been undertaken to investigate the effect of colored light shone into the eye on visual functioning, specifically the size of the visual field and how it relates to reading. For instance, Kaplan (1983) and Liberman (1986) hypothesized that reading disorder is caused by a reduced visual field, causing deficits in peripheral vision and thereby inefficient reading. Both studies reported visual field size increases and improved reading ability after syntonic treatment. The interventions in these studies involved participants looking through various colored filters either prescribed or individually selected by preference at an incandescent light source. Unfortunately, neither study reported the details of reading tests administered to the participants, nor did they control for differing pre-treatment baselines in visual field size. These methodological issues, among others (Howell & Stanley, 1988) render their contribution to the evidence base for syntonic exceedingly weak.

2. The discovery of visual stress

While previous color-based interventions focus on remediating various symptoms that are causing learning inefficiency in standalone treatment sessions, the majority of the recent literature focuses on interventions in situ, those which are used to treat symptoms at the same time as learning or reading is taking place. In a volume on dyslexia, Critchley (1964) noted that some children who had difficulty reading on white paper had their reading facilitated by colored paper, but nothing further along these

lines was reported until Meares (1980) described several children in a reading clinic who were reporting symptoms of discomfort when reading. Meares hypothetically linked these symptoms to contrast sensitivity.

Subsequently, Irlen (1983) posited that some individuals with reading difficulty suffer from a distinct syndrome, which was dubbed 'Scotopic Sensitivity Syndrome'. This choice of name is curious; some have questioned the appropriateness of the term 'scotopic', since it relates to conditions of low light which would involve the function of rod cells in the eye (Helveston, 1990). There are no rod cells in the fovea, where printed words are projected, so the extent to which the term relates to reading is unclear (Menacker et al., 1993). Nevertheless, several other names for the proposed disorder have been used, including 'Meares-Irlen Syndrome' (occasionally 'Irlen-Meares' or 'Irlen Syndrome'), 'visual discomfort' and 'visual stress'.

The symptoms of the disorder (hereafter referred to only as visual stresses – the most neutral of the terms used) are described as various distortions and illusions of the text experienced to varying degrees by each individual. Irlen (1991) describes the five components of visual stress: light sensitivity (including difficulties reading under fluorescent lights), inadequate background accommodation (trouble reading in conditions of high contrast), poor print resolution (including illusory movements of text on the page), restricted span of recognition (also known as 'tunnel reading', a restriction on reading groups of words) and lack of sustained attention (p. 31). No indication is given as to the prevalence of each symptom within the population of individuals with visual stress.

The basis on which Irlen classified the symptoms as part of the visual stress disorder is not known. Information as to which of Irlen's patients were screened for other, possibly co-occurring, conditions such as attention deficit disorder or optometric, orthoptic, or ophthalmic abnormalities would be of interest in assessing the construct validity of visual stress (it has been noted by Scheiman, 2004, that many of the symptoms discussed by Irlen are remarkably similar to those reported by individuals with vision problems which are better understood by eye care professionals, such as accommodative anomalies or for a discussion of visual problems and their possible relation to visual stress). It should be noted that Irlen is of the opinion that dyslexia and visual stress are separate, but sometimes co-occurring, disorders (Irlen, 1991; 2010), both of which can contribute to reading disability.

Reading disability is by no means the only problem discussed by Irlen. Visual stress is also claimed to affect writing, depth perception, coordination, and motivation. Further, in a recent book, Irlen (2010) claims that conditions including, but not limited to ADHD, autism, chronic fatigue syndrome, epilepsy, Tourette syndrome, head injuries, agoraphobia, anxiety attacks, depression, and conduct disorder are related to

visual stress. These disorders are, apparently, either worsened by the presence of visual stress, are in part caused by the self-esteem issues associated with being unable to read or pay attention in a school classroom, or are in actual fact simply visual stress which has been misdiagnosed as another disorder. No references to studies which confirm any link between visual stress and these other diagnoses are provided.

As we will see below, researchers such as Wilkins (e.g. 2003) have hypothetically linked various other disorders to visual stress, but unlike Irlen they have tested these hypotheses and published their findings. Already noted above is the evidence linking reading disorders to adverse long-term outcomes such as offending behavior, but the connections between these findings and visual stress per se have never been investigated.

Irlen (1983, 1991, 2010) does not discuss the theoretical basis for visual stress in detail, nor does she make specific, testable claims about its aetiology. Visual stress is said to 'possibly [involve] a structural brain deficit involving the central nervous system' (Irlen, 1991, p. 57), resulting in full-spectrum light inhibiting perception. Irlen (2010) provides pictures from a SPECT scan of a visual stress sufferer which appear to show reduced activation in several brain areas when the individual is wearing Irlen colored lenses (p. 101). However, no scientific details are provided about the nature of these scans, and it does not appear that they have been published in a scientific journal.

3. Current methods of treatment

Irlen (1991) recounts her discovery of the effectiveness of color on reading, noticing that, when a colored, transparent plastic overlay was placed over the text, disabled readers tended to report fewer visual stress symptoms and had improved reading performance. Further to this, Irlen makes several intriguing claims, indicating that not only does each person have an optimal filter color which is unique to them, but that only the particular set of colors provided by the Irlen Institute are effective in treating the disorder. This latter claim is due to the fact that 'the Irlen Method has been developed, refined, and researched over many years' (Irlen, 1991, p. 194). No specifics are given, and indeed, it could conversely be argued that other types of color-based intervention (e.g. Wilkins' Intuitive System - see below) have been researched far more extensively and scientifically than the Irlen Institute's method. Nevertheless, it is claimed that 'inaccurate colour selection [i.e. that of non-Irlen Institute practitioners] can result in headaches, eye strain, and fragmented brain processing resulting in more distortions and reading problems' (Irlen Institute, 2010).

It is important to note that Irlen did not perform or publish any controlled clinical trials or provide any evidence in the initial descriptions of the syndrome and its

treatment. Indeed, Irlen's book (Irlen, 1991) is almost entirely based on anecdotes from the author's experience as an educational psychologist (Helveston, 2001), and makes a wide range of claims about clinical symptoms, syndrome prevalence and treatment efficacy with little empirical support. For instance, Irlen claims that visual stress is present in 12% of the normal population, and 65% of individuals diagnosed with dyslexia (Irlen, 1991). It is remarkable that in her most recent volume, Irlen (2010) provides almost no more supporting evidence for these claims (or indeed any other claim) than in the earlier works (Irlen, 1983, 1991). As we will see, a substantial number of studies have been published in the intervening time. It is striking that Irlen (2010) hardly makes mention of them.

The Irlen Method (along with other, similar treatments) has received a great deal of mass media publicity (Helveston, 2001), and the Irlen Institute now has clinics in many US states, several locations in the UK, and many other parts of the world (Irlen Institute, 2009). Clearly this high level of publicity warrants further scientific investigation into the efficacy of the treatment.

In spite of its unreliable nature, Irlen's work and the publicity surrounding it can be seen as an inspiration for many subsequent studies. For example, Wilkins (e.g. 2002, 2003) and his research team have been major contributors to the literature, with many published studies and reviews. Wilkins (1994) has, on the basis of this research, developed for purchase by disabled readers the 'Intuitive Overlays', an alternative set of colored overlays. In addition, Wilkins is the inventor of the 'Intuitive Colorimeter' (Wilkins, 2003), an instrument used in both research and clinical settings to choose a more precise optimal color for tinting lenses. The Intuitive Overlays, the Intuitive Colorimeter, and a reading measure designed by Wilkins, the Wilkins Rate of Reading Test, are important parts of Wilkins' research methodology (see e.g. Wilkins & Evans, 2009).

4. Colored Overlays

The overlays used by the Irlen method and the Intuitive system are A4 plastic sheets, with one 'shiny' and one 'matte' side. Wilkins, on the basis of his group's research, concurs with Irlen's opinion that filters should be individually-prescribed, as there exists an optimal color for each individual which will reduce their visual stress symptoms and facilitate their reading to the greatest extent (Wilkins, 2003; Wilkins et al. 2005; though see Simmers et al., 2001). The basic set of colors provided by the Irlen method and the Intuitive system are broadly similar (the chromaticity of the Intuitive Overlays, along with other details, can be found in Wilkins, 1994). In both systems, a

patient's optimal overlay is chosen using a process of elimination, often combined with questions relating to any visual stress symptoms the patient may be suffering from.

5. Colored Spectacle Lenses

Colored lenses usually involve more in-depth examination. In the Intuitive system, the aforementioned Intuitive Colorimeter allows a wider variety of colors to be chosen for lenses, with over 25,000 different colors having been prescribed, according to Wilkins et al. (2007). Using the colorimeter, the patient can themselves manipulate color, hue and saturation independent of one another, and thereby find their exact preferred color (Wilkins et al., 1992; Wilkins, 2003). A set of test lenses with a limited range of colors exists to use during diagnosis with the Colorimeter, and after diagnosis the practitioner can send away for the precise color lenses required (University of Essex, 2010). There exists a 'Society for Colored Lens Providers', which provides a code of conduct which practitioners must sign as well as instructional lessons and courses (Society for Colored Lens Providers, 2010). On the other hand, diagnosticians and screeners from the Irlen Institute – who do not subscribe to the Society for Colored Lens Providers – only require further diagnostic appointments before tinted lenses are prescribed, but no instruments similar to the Intuitive Colorimeter are used by their method.

Wilkins (2003) has criticized the Irlen method due to the fact two or more lenses are often used simultaneously; this may lead to complementary colors being used, which 'may counteract each other', at which point there is 'no guarantee that the colour formed by combining the trial lenses will be better than either of the lenses on their own' (p. 90). The Irlen Institute's method of tinting lenses is confidential, and no technical data have been made available for scientific scrutiny.

Both Irlen (1991) and Wilkins (2003) note that the chosen 'optimal' color of tinted spectacle lenses is often different from that of overlays. Wilkins (2003) offers a possible explanation in terms of 'the state of adaptation of the eyes differing for overlays and lenses. Lenses have an effect similar to that of a colored light: everything within the visual field is colored, and the eyes adapt to that color overlays, on the other hand are viewed while the eyes are adapted to colored light' (p. 92). The Irlen Institute gives a similar explanation (Irlen Institute, 2010).

Finally, in publicity on BBC 1 Television (The One Show, 19 January 2010) and BBC Radio 4 (All in the Mind, 26 May 2010), Stein has claimed to be able to treat up to 50% of individuals with dyslexia with yellow and blue lenses. The theory behind these claims is discussed in section below but no details of, firstly, how many children are being treated using this method or, secondly, the precise chromaticity or nature of the lenses being used were provided.

6. Theories of Color and Reading

Researchers have put forward various theoretical mechanisms which potentially explain the effects of colored filters on reading. This section discusses two such explanations: magnocellular deficit theory and cortical hyper excitability theory.

6.1 Magnocellular Deficit Theory

Many authors have proposed magnocellular deficits as an explanation of reading disorder (Stein, 2001). This review will focus on the visual aspects of this magnocellular impairment, though the theory has been posited to explain the auditory and motor as well as visual deficits which have been associated with dyslexia by some authors (Ramus, 2003).

Visual magnocellular theories often relate to the three types of cell found in the lateral geniculate nucleus (LGN), part of the brain's visual pathway leading to the primary visual cortex. The upper four layers of cells, known as the 'parvocellular' neurons owing to their small cell bodies, are involved in color vision and processing fine spatial detail, along with smaller, 'koniocellular' neurons which are found throughout the LGN (e.g. Chatterjee & Callaway, 2003). Theories are focused, however, on the lower two layers of larger cells with a higher conduction velocity, the 'magnocellular' neurons, which respond to low spatial resolutions and high temporal frequencies - for example those caused by movement (Palmer, 1999). Cells can be found in the retina which selectively project to parvocellular or magnocellular neurons (Shapley & Perry, 1986), underlining the importance of these two distinct types of cell for visual processing.

Post-mortem studies which showed evidence of abnormalities and ectopias in the brains of dyslexic, as compared to non-dyslexic, individuals (Galaburda et al., 1985; Humphreys et al., 1990; Livingstone et al., 1991; see Eckert, 2004, for a review) are routinely used as evidence for the magnocellular view. The theory proposes that these abnormalities, which can be caused by interrupted neuronal migration, disrupt the magnocellular system.

This disruption, claim magnocellular theorists, causes the magnocellular system to fail to suppress the action of the parvocellular system. In non-disordered individuals, this suppression 'causes the activity in the parvocellular system to terminate so as to prevent activity elicited during one fixation from lingering into that from the next fixation' (Skottun, 2000a, p. 111). However, without this suppression, input from one visual fixation would 'bleed' into the next, creating confused images and, by extension, disordered reading (Livingstone et al., 1991; Lovegrove et al., 1986). Subsequent research has, however, shown that it is the magnocellular system, not the parvocellular

system, which is suppressed at each saccade (Skottun & Parke, 1998), and as a result this hypothesis became untenable.

Newer formulations of the magnocellular deficit theory have been proposed (e.g. Stein & Walsh, 1997; Stein, 2001), positing that defective motion sensitivity in the magnocellular system leads to binocular instability – unsteady fixations when using both eyes simultaneously - and, therefore, to 'the letters [dyslexics] are trying to read to appear to move around and cross over each other' (Stein, 2001, p.12). This phenomenon is appreciably similar to one of the proposed symptoms of visual stress, described above.

It is worth noting at this point that the proponents of a magnocellular theory-related mechanism for the benefits (if any) of colored filters make no reference to visual stress, or to any other terms used to refer to the disorder. Nor, puzzlingly, do they make significant reference to any of the other studies into the effects of colour on reading, such as those by Wilkins and colleagues (e.g. Bouldoukian et al., 2002; Wilkins et al., 1994). Presumably these authors are of the opinion that the symptoms of 'visual stress' are in fact the result of a faulty magnocellular system, and that visual stress either does not exist or is not a separate condition from magnocellular disorder. As we will see, some experiments into colored filters from a perspective outside these theoretical 'camps' do make reference to the magnocellular theory (e.g. Christenson et al., 2001; Noble et al., 2004).

Skottun (2000a), in a review of the literature, found that the number of studies providing evidence in support of the magnocellular deficit theory are outnumbered by the number of studies finding evidence incompatible with the theory. Other reviewers note that in several demographic studies, sensory deficits are found only in a small proportion of disordered readers, whereas the vast proportion of these readers have a cognitive phonological deficit (e.g. Ramus, 2003; White et al., 2006; see Section 1.1). Magnocellular deficits are also sometimes found in individuals with no reading problems (Vellutino et al., 2004). This evidence reduces the likelihood that the magnocellular theory is an explanation of a high proportion of reading disorders. In addition, Ramus (2004) has proposed an interpretation of the evidence which explains the neuroanatomical abnormalities discussed above (e.g. Galaburda et al., 1985) in terms of the phonological deficit theory.

Skottun (2000a, 2004) and Skottun and Skoyles (2007b) have repeatedly pointed out that it is extremely difficult to isolate magnocellular function in any experimental setting. Many experiments risk stimulating both magnocellular and parvocellular systems (for further discussion, see Stein et al. (2000) and Skottun (2000b). Further, it is unclear whether some experimental techniques, such as those which use motion sensitivity (see e.g. Talcott et al, 2000), are involving an entirely separate (extrastriate)

brain pathway instead of either the magnocellular or parvocellular systems (Skottun, 2000a; Skottun & Skoyles, 2007b). In addition, some experiments have failed to find any evidence of the expected abnormalities on magnocellular-based tasks in dyslexic individuals (e.g. Johannes et al., 1996).

Related to this, many of the studies which purport to provide evidence for the magnocellular theory are based on spatial and temporal frequency measures – the most frequently-utilized way of differentiating between the magnocellular and parvocellular pathways. One problem with such tests has been demonstrated by Heath et al. (2006). These authors found that while individuals do perform reliably on visual and auditory versions of these tests, (i.e. test-retest reliability tends to be high), there do not seem to be reliable correlations within individuals on different tasks (i.e. construct validity is low). Studies which attempt to provide evidence for the magnocellular view, then, do not necessarily assess magnocellular function in its entirety.

Even so, as mentioned above, the hypothesized deficit in the magnocellular system has been proposed by some researchers (e.g. Christenson et al., 2001; Noble et al. 2004; Whiteley & Smith, 2001) as a potential explanation of the effects of colored filters on reading, perhaps by facilitating the performance of the poorly-functioning magnocellular pathway, thus aiding reading[i]. The magnocellular system's involvement with color may at first seem counter-intuitive, for it is the parvocellular system which is involved in color vision, as previously stated. Nevertheless, three studies have investigated the effect of color on reading function in this context – the first indirectly, and the second and third directly.

First, Stein et al. (2000b) used yellow filters as controls in an experiment on monocular occlusion as a treatment for dyslexia. The hypothesis in this experiment, which was based on previous work (Cornelissen et al., 1992; Stein & Fowler, 1985), was that, if a substantial proportion of dyslexics suffer from binocular instability, a method of treating this may be to occlude one eye for some length of time in order for the dyslexic to gain a more fixed frame of reference.

To test this, Stein et al. (2000b) sampled 143 dyslexic children's reading age along with orthoptic measures, including stability of fixation. 71 of these children were then given spectacles tinted with a yellow color (the control group), while a further 72 children were given spectacles with a yellow right lens and the left lens occluded with opaque tape (the treatment group). Curiously, a control group with colorless, non-

[i] It has been suggested by Stein that this is the mechanism for the effects of yellow colored filters. However, blue colored filters, which Stein also claims facilitate reading, are hypothesized to 'trick' the brain's internal clock functions into behaving as if it were early morning – the light of which is blue - leading to increased attention and thereby improved reading. There is, as yet, no published evidence for this hypothesis, though it is forthcoming (Stein, personal communication, June 2010).

occluded lenses in their spectacles was not included in the experiment. This may be due to the fact that this study was an attempted replication of previous studies which used colorless lenses, but it would have been nonetheless instructive to have such participants in this study.

Retesting of fixation stability and reading age occurred at 3, 6 and 9 months after the spectacles were issued. The treatment groups were found to develop stable fixations more quickly across this time than the control group, and reading age progressed much faster in the treatment group. The control group's progression was attributed to the yellow lenses 'boost[ing]' (p. 168) the magnocellular system.

Previous work on the monocular occlusion hypothesis was criticized for basing conclusions on flawed methodology (Bishop, 1989; though see a response from Stein, 1989). The newer study has drawn criticism also; Fawcett (2000) notes that, while advances in binocular stability did occur faster in the treatment group, by the end of the 9 months, only 6 more children in the treatment group had achieved stable fixation than the controls, which was not a significant difference. This draws the efficacy of monocular occlusion for achieving stable fixations into question; it is unclear whether the fixations could be aided by the yellow lenses, or simply appears as a matter of course during the maturation process, which is known to be the case in normal development (Bishop, 1989).

In addition, Fawcett (2000) suggests that more specific details about the reading performance of the children are required – if it were found that the children who achieved stable fixations are making less reading errors of a visual nature (e.g. skipping lines or words) as opposed to a linguistic nature (e.g. incorrect sounds), it would be more convincing evidence for Stein et al. (2000b)'s hypothesis.

Stein et al. (2000b) originally took their sample of 143 from a group of 700 dyslexics, after they found that 80% of that larger group did not have unstable fixations. This, combined with the fact that a proportion of the remaining 20% will probably gain stable binocular fixations spontaneously (p. 168), makes it clear that the monocular occlusion method will not be applicable to the vast majority of those with reading disorders. Perhaps for this reason, Stein and colleagues have performed no more research into monocular occlusion, preferring to further investigate the effects of the colored filters they previously used as a control.

The second study to be considered, Chase et al. (2003), used a variety of measures involving red and blue light to study the magnocellular pathway's contribution to reading. Previous research using filters had shown that the color blue may facilitate reading (e.g. Iovino et al., 1998, though see Christenson et al., 2001) and suggested that the magnocellular pathway's function is inhibited by red light (e.g. Hughes et al., 1996) - though see Pammer & Lovegrove, 2001. These latter authors, on

the basis of four separate experiments investigating red and blue light on magnocellular function, are skeptical that separate colours can differentially activate the magnocellular system. They also suggest that differences in stimulus contrast, as opposed to color, may be an explanation of the various inconsistent results from studies in this area.

Chase et al. (2003) attempted to differentiate between two possible mechanisms for blue light aiding reading: that the facilitation is due to either the presence of short wavelengths (blue light) or the absence of long wavelengths (red light). Their research involved the use of Irlen filters (Irlen, 1991) and isoluminant stimuli (those which are created specifically to stimulate only the color-sensitive parvocellular pathway – see Lu et al., 1999). They concluded that reading is facilitated by the removal of long wavelengths, and that a grey filter would do just as well as a blue filter to remove these. Additionally, they showed that the presence of long wavelengths appears to interfere with reading, and, using the isoluminant stimuli, that the parvocellular pathway is less inhibited by the presence of red light than the magnocellular pathway.

This research has been shown to have several flaws, however. Skottun (2004) has demonstrated that not only is it incorrect to say that red light has an effect on the magnocellular system, also it is not necessarily possible, as Chase et al (2003) assumed, to isolate magnocellular function by using red light – indeed, 'red light may have a profound effect on parvocellular neurons' (Skottun, 2004, p. 67). In addition, Jordan et al. (2007) have shown that creating true isoluminant stimuli is no simple matter, and that the stimuli created by Chase et al. (2003) – where the letters used were the same luminance as the background - may not have been processed in the manner the authors had assumed, confounding their results. A similar set of experiments would have to be performed bearing these caveats in mind before stronger conclusions could be drawn.

The next study to be discussed is by Stein's research group. Ray et al. (2005) hypothesized that, since blue light had been shown to inhibit magnocellular function through a mechanism involving delayed retinal S-cone cell stimulation (Stockman et al., 1991), the magnocellular pathway may be more sensitive to yellow light, even though it does not mediate color vision. To investigate the hypothesis, the researchers had a group of children with reading disorder read through a yellow filter for 3 months, to release the magnocellular pathway 'from the negative influence of the S-cones' (p. 2), and compared them with a similar group of children receiving a placebo treatment. After this time, the researchers found that motion sensitivity, convergence, accommodation, and reading ability (the latter measured by the child's British Ability Scales (BAS) reading score (Elliott et al., 1978)) had improved significantly more in the group using the yellow filters. See the next section for a more detailed discussion of these findings.

The theoretical explanations for this finding (involving reduced S-cone input), Ray et al. (2005) admit, are controversial. Indeed, Skottun & Skoyles (2007a) provide evidence that the inputs of S,M, and L-cone cells are summed before communication to the magnocellular system, making it unlikely that the influence of one type of cone in particular can be 'filtered out'. They also note that, when discussing mechanisms, Ray et al. (2005) seem to be proposing 'two contradictory views with regard to the effect of yellow stimuli on the magnocellular system' (Skottun & Skoyles, 2007a, p. 291n) – at one point suggesting that magnocells are inhibited by too much input from the S-cone cells in the eye, and at another suggesting the problem is due to an incorrect L/M-cone balance. This renders the theory somewhat confused. Due to these criticisms, and especially since no further experimental work has been done from this theoretical perspective, it is difficult to conclude that the magnocellular deficit theory has a great deal to say about the effects of color on any visual process.

More generally, as noted by Skottun (2005), the issues of, firstly, the magnocellular pathway's potential involvement in the reading process, and secondly, the magnocellular deficits potentially being a cause of reading disorder, are separate and should not be confused. It would be wholly unsurprising if the magnocellular pathway was involved in reading, along with the many other visual systems. More precise evidence would be required to show that a deficit in the magnocellular system alone was responsible for some reading problems.

It has been suggested that sensory and sensorimotor deficits may be a correlate, but not necessarily a cause, of dyslexia (Ramus, 2003; Vellutino et al., 2004; White et al., 2006), but it is unclear that these are due to magnocellular impairments in particular (Amitay et al., 2002). Indeed, Skoyles & Skottun (2004) note that 'not only are there cases of dyslexia that are not attributable to magnocellular deficits, but also there are a substantial number of instances of magnocellular deficits that do not lead to dyslexia' (p. 81). The hypothesis of magnocellular causality is therefore not assured. Finally, Castelo-Branco et al. (2007) found that magnocellular impairments are present in the developmental disorder Williams Syndrome, but they do not reliably predict any behavioral or perceptual outcome. This clearly challenges the magnocellular theory, which predicts a causal pathway between low-level visual impairments and high-level deficits.

From an entirely different perspective, Wilkins (2003) marshals four main lines of evidence against the magnocellular theory of colored filters' facilitation of reading. Firstly, experiments have found that individuals with no reading disability can also benefit from colored overlays and lenses (e.g. Jeanes et al., 1997). However, we have already seen above that magnocellular deficits are sometimes found without reading disorder (Skoyles & Skottun, 2004; Vellutino et al., 2004), blunting the effectiveness of

this line of criticism. Secondly, it is noted that the magnocellular theory is a theory about dyslexia specifically, and since dyslexia is not necessarily associated with visual stress (Wilkins, 2003; though see Singleton & Trotter, 2005), we cannot necessarily expect the theory to explain visual stress. Thirdly, Wilkins (2003) argues that the magnocellular theory cannot explain the color specificity found in several studies of color and reading (e.g. Jeanes et al., 1997; Wilkins et al., 1994; though see Simmers et al., 2001).

Finally, Wilkins (2003) notes that Simmers et al. (2001) examined a group of children who regularly used (and, apparently, benefitted from) colored lenses, and failed to find the magnocellular deficits which would be expected on the magnocellular theory (see also White et al., 2006). The authors suggest that this evidence shows that visual stress is separate from other disorders, such as dyslexia and attention deficits, which often contribute to poor reading. This is in line with the opinion of Irlen (1991).

6.2 Cortical Hyper excitability Theory

Since Wilkins (e.g. 2003) views the magnocellular theory as insufficient to account for the effects of color on reading, he and his colleagues have tentatively proposed an alternative theory based on cortical hyper excitability – certain areas of the cortex over-responding to particular visual stimuli. This theory draws comparisons between individuals with visual stress and those with several other potentially related disorders: migraine, epilepsy, multiple sclerosis and autism. This review will first look into how these connections have been made, and will then describe and discuss the neurophysiological basis of the theory.

7. Connections to other disorders

Noting that one of the symptoms often described by visual stress sufferers is headache, Maclachlan et al. (1993) surveyed a sample of 74 children with reading disorders. Their results indicated that individuals who benefit from colored overlays – i.e. those who, by the Wilkins Intuitive method, have visual stress - were almost twice as likely (60% to 31%) to have a family history of migraine than those who do not report any benefits from color. Wilkins' theory of visual stress (e.g. Wilkins, 1995; Wilkins et al. 2004; Wilkins et al., 2007) uses this finding as a basis for making other comparisons between visual stress and migraine.

Wilkins' theory of visual stress (Wilkins, 1995) hangs on the assumption that migraine (and therefore, hypothetically, visual stress) is caused by cortical hyper excitability. However, there is considerable controversy amongst researchers investigating migraine as to whether this is indeed the case. Studies which find cortical

hyper excitability in migraine (e.g. Mulleners, 2001; see Aurora & Wilkinson, 2007, for a review) are to be contrasted with those which come to the opposite conclusion – findings of cortical hypo excitability (reviewed by Ambrosini et al., 2003).

Coppola et al. (2007) suggest the controversy is due to a semantic confusion, and propose a new term – 'cortical hyper responsiveness' - which they suggest accounts for all the supposedly conflicting results. According to Coppola et al., (2007), the evidence suggests that different kinds of stimulation cause the migranous cortex to respond in different ways –repetitive stimulation will cause hyper excitability, but for low numbers of stimuli, cortical excitability will be reduced (i.e. hypo excitability). For this reason, the authors view 'hyper excitability' as too limiting a term to describe the true nature of migraine. Stankewitz and May (2009), on the other hand, suggest that the contradictory results arise from methodological issues. For instance, very few studies control for the circadian nature of migraine – the authors suggest several endocrinal influences may confound the estimates of cortical excitability. For these reasons, the assumption that migraine is simply caused by cortical hyper excitability is not an entirely firm basis for building a theory.

Nevertheless, the use of colored filters for individuals with migraine has been studied, and positive results have been found. Evans et al. (2002), in a sample of adults with migraine, found that pattern glare (defined as a hypersensitivity to certain visual patterns - see Chronicle & Wilkins, 1996; Wilkins and Nimmo-Smith, 1987) was the only reliable visual correlate of migraine from candidates of contrast reduction sensitivity, pattern glare, and a range of orthoptic and optometric disorders. In a follow-up randomized controlled trial using the same sample, Wilkins et al. (2002) found that colored lenses of an optimal color, as selected on the basis of assessment with the Wilkins Intuitive Colorimeter (Wilkins et al., 1992), caused a marginally significant reduction in headache. However, these studies are subject to sampling bias, since the criterion for selection of participants was that the individual reported some benefit from colored overlays (none of the sample had previously used colored lenses). For this reason, the results of the two studies should be regarded as provisional. Indeed, Evans et al. (2002) admit that 'there is still a need for a rigorous controlled trial to investigate the optometric correlates of migraine in a large, unselected, sample' (p. 140). Other disorders have been similarly linked to visual stress. Wilkins et al. (1999) performed a longitudinal study into the use of colored lenses in a population with photosensitive epilepsy. Having selected an optimal color of lens, 17 patients were followed up an average of 2.4 years later. The authors found that 13 of the patients still used their colored lenses, and they had noticed important improvements when wearing them, such as reduced dizziness. Three of the participants reported a reduction in number of seizures. Wilkins et al. (1999) argue that these effects cannot be attributed to the placebo

effect, since the duration of continued lens use is very long. This is a common argument in this area (see below), but it is not clear that it is valid. Placebo effects are not simply tied to novelty; there is no firm reason for thinking a placebo effect should necessarily be short-lived. Wilkins & Evans (2009) suggest that, since epilepsy is associated with seizures, in this case the lenses can be shown to help another condition involving cortical hyper excitability.

Another possible connection to visual stress comes from a study of lens use in multiple sclerosis (MS) by Newman Wright et al. (2007). Since estimates of the prevalence of visual deficits in MS range from 38-85% (Arnold, 2005), and a small proportion of MS sufferers experience seizures (Koch et al. (2008) give an average of around 3% from numerous studies), Newman Wright et al (2007) hypothesized that colored overlays may elicit similar positive responses to those Wilkins et al. (1999) found with lenses in epilepsy.

The authors did indeed show that performance on a the Wilkins Rate of Reading test (WRRT; Wilkins et al., 1996) and a new visual search task, the 'Circles Search Task' was superior when participants used overlays of their chosen, 'optimal' color. All but one of the 26 participants also reported a reduction in subjective visual stress symptoms. Newman Wright et al. (2007) note with surprise that, after several weeks the participants' performance on reading and visual search tasks without an overlay had also increased. They rule out practice as a sufficient mechanism for this result since performance with an overlay was invariably superior, but unfortunately do not consider a possible explanation based on a combination of practice and placebo effects.

The final disorder linked to visual stress by Wilkins and colleagues is autism. It has been observed that a third of individuals with autism develop seizures in early life (Gillberg, 1991), and various disorders of visual perception have been found in children with autism (Dakin & Frith (2005) for a review of the literature). With this in mind, Ludlow et al. (2006) used the WRRT to assess the effects of colored overlays on the reading ability of children with autism (presumably high-functioning autism; no further definition is given) compared with that of a sample of neuro typical children and those with moderate learning disability. It is to be noted that the autistic children participating in this experiment were not poor readers. The researchers found that reading rate with overlays increased only for the autistic children (indeed, the overlays appeared to inhibit the reading of the control group).

The conclusions that can be drawn from this study are limited, however. Since the autistic children in the sample were not those who suffered from seizures, the connection to cortical hyper excitability was not necessarily present in the sample. Since the phenotype of autism is extremely heterogeneous (see, e.g. Folstein & Rosen-Sheidley, 2001; Frith, 2006), there is some uncertainty as to whether this study assessed

the correct subtype of autism for any concrete conclusions about links to visual stress to be drawn. In addition, Ludlow et al. (2006) acknowledge that their study did not sufficiently control for novelty effects, which may have influenced the results.
It can be seen, then, that the connections drawn between visual stress and migraine, epilepsy, MS and autism are at present speculative and based on a small number of studies. More evidence will need to be gathered to make more solid associations between the disorders.

8. Proposed mechanism

Having provided these tentative lines of evidence which may show that visual stress involves cortical hyper excitability, the theory posits that visual stimuli involving high spatial frequency and high contrast will cause an abnormal reaction in individuals with a particular kind of hyper excitability. Lines of text have been compared to high-contrast visual gratings (e.g. Wilkins & Evans, 2009). Since this kind of visual stimulus has been shown to cause hyper neuronal activity leading to seizures in those with photosensitive epilepsy (Wilkins et al., 1980) and, more pertinently, perceptual distortions in a study of individuals with migraine (Huang et al., 2003), it is suggested that a similar phenomenon can be found in individuals suffering from visual stress.

It is therefore predicted that the main marker of visual stress will be found to be pattern glare, and this has been found to be the case (e.g. Hollis & Allen, 2006). Pattern glare is defined by Evans & Stevenson (2008) as being caused by stimuli 'with spatial frequency of about 3 cycles per degree (cpd), even width and spacing (duty cycle 50%), high contrast and be viewed binocularly' (p. 296 – see also Wilkins et al., 1984). Evans & Stevenson (2008) went on to define clinical thresholds for the Evans and Wilkins Pattern Glare Test (see Wilkins & Evans, 2001), and suggested that anyone scoring above a certain threshold on the test may be suffering from visual stress in everyday life.

Some researchers have attempted to tease apart two of the aspects of pattern glare mentioned above. Williams & LeCluyse (1990) had shown that blurring text (reducing both the spatial frequency and the contrast level) facilitated reading in disabled readers, and as a result Williams et al. (1995) used a search task in an effort to differentiate between the effects of spatial frequency and contrast reduction. Groups of individuals with reading disorder, those with reading disorder and attention deficit disorder, and a control group of normal readers were asked to search for a particular letter in arrays which had been altered visually in various ways. Intriguingly, the group with both ADD and reading disorder had their reading facilitated by both spatial frequency and contrast reduction, but in the reading disorder-only group, only contrast reduction appeared to aid reading – the poor readers had their search time reduced to

almost the level of the controls with reduced contrast, but no significant effect was found for changes in spatial frequency. The reasons for the differences between the two groups of poor readers are uncertain, but Williams et al. (1995) suggested that further work should investigate why contrast reduction aided reading rather than spatial frequency.

If, as in the Williams et al. (1995) study, contrast reduction is the only mechanism for reading improvement, it could potentially explain any benefits found by users of colored filters. However, this would not explain the findings of several studies which appear to show color specificity, that is, an 'optimal' color for each individual with which they report benefits that they do not report for any other color. On the basis of seminal work by Zeki (1983) on neurons in the visual system of monkeys which respond to particular colors and the finding by Xiao et al. (2003) that the cortex of monkeys is spatially arranged into areas for separate colors, Wilkins & Evans (2009) hypothesize that looking through color can 'redistribute excitation' in the cortex(p. 5). This leads to the reduction of visual stress symptoms as described in studies such as Wilkins et al. (1994), and potentially improved reading results.

While this line of reasoning could explain the benefits of color found in some experiments, the authors never explicitly state the reasons behind individual differences in color specificity. Indeed, a study examining the low-level visual aspects of visual stress – that is, aspects which are objective - found no evidence for this specificity (Simmers et al., 2001). In this small-scale study, the experimenters compared the accommodation reflex of the eye of 5 participants who used colored lens. They were unable to find a difference in the size of the accommodative reflex (which involves the eye's lens changing size to maintain a clear image) when the participants used a lens of the participant's favored color compared to a lens of a complementary color.

Finally, future research could examine one potential prediction of the cortical hyper excitability account. A recent study, operating on the assumption that migraine is caused by cortical hyper excitability, suggests that trans cranial magnetic stimulation (TMS) is potentially a method to restore normal cortical excitability (Brighina et al., 2009). If this is the case, an interesting question could be asked: could a similar methodology alter symptoms in individuals with visual stress? If Wilkins' theory of abnormal cortical hyper excitability in visual stress is correct, a potential prediction would be that the visual distortions associated with visual stress would be reduced with TMS.

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