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Visual motion sensitivity and reading

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Abstract

Reading is more difficult than speaking because an arbitrary set of visual symbols must be rapidly identified, ordered and translated into the sounds they represent. Many poor readers have particular problems with the rapid visual processing required for these tasks because they have a mild impairment of the visual magnocellular system. This deficit has been demonstrated using neuropathological, evoked potential, functional magnetic resonance imaging and psychophysical techniques. The sensitivity of the M-system in both good and bad readers correlates with their orthographic abilities, suggesting that the M-system plays an important part in their development. This role is probably to mediate steady direction of visual attention and eye fixations on words. Thus many children with reading difficulties have unsteady eye control and this causes the letters they are trying to read to appear to move around, so that they cannot tell what order they are meant to be in. Therefore, boosting M-performance using yellow filters, or training eye fixation, can improve reading performance very significantly. Several genetic linkage studies have associated reading difficulties with the MHC control region on the short arm of chromosome 6. This system has recently been shown to help regulate the differentiation of M-cells. This association could also explain the high incidence of autoimmune conditions in poor readers. Other chromosomal sites are associated with the metabolism of polyunsaturated fatty acids (PUFAs) as found in fish oils, and this could explain why PUFA supplements can improve reading.

Keywords: Reading; Dyslexia; Vision; Magnocellular; Eye movements; Attention; Genes; MHC; Fish oils

1. Introduction

In his report on the state of the UK educational system Claus Moser stated that 20% of British adults cannot locate the page reference for ‘plumbers’, given the alphabetical index to the Yellow Pages. A similar proportion of Americans is as illiterate. This is of course an indictment of the educational systems in these countries; but it also emphasise how difficult reading is for many people. The late Alvin Lieberman used to say that reading is difficult because speaking is so easy. But we speak in syllables, whereas reading demands the ability to translate visual symbols into much smaller units, phonemes, which are not naturally separated in speech (Liberman, Shankweiler, & Studdert-Kennedy, 1967).

The earliest Sumerian hieroglyphic scripts, dating from some 5000 years ago, represented numbers literally (e.g. depicting five sheaves of corn); and modern Mandarin or Japanese logographs still primarily represent the of meaning words, rather than the separate sounds of which they are composed. So the reader of Mandarin has to learn at least 4000 different logographs, making fluent reading of advanced texts still a rarity in China. The alphabetic idea that word sounds can be split down to a much smaller number of phonemes (ca. 50 in most languages) that could be represented by an even smaller number of visual symbols, letters, was introduced only ca. 3000 years ago, and it is credited to the Phoenicians who passed it on to us via the Greeks. Thus the alphabetic principle is a cultural invention and not, like speech and language itself, an ‘instinct’ enshrined in our genetic makeup. We have to be taught to read, but speech usually comes naturally by infants imitating their parents.

Reading is difficult because small, sparsely detailed, written symbols have to be identified visually, put in the right order and then translated into the sounds that they stand for. For most words in their sight vocabulary practised readers can do this very rapidly, translating quite long groups of letters into their sounds. However although children learning to read soon begin to recognise very common short words en bloc, for every word that they have not yet memorised in their sight vocabulary, they have to visually sequence the letters and sound them out. Thus visual analysis plays a very important part in learning to read.

However whether visual processing ever plays a limiting role in reading remains very controversial (Hulme, 1988). Clinically the vision of poor readers is usually normal and it has been difficult to show that they have any specific visual

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deficits. Instead, because reading requires the translation of visual symbols into the phonemes they represent, it has been easy to show that poor reading is associated with deficient phonological processing. Hence most students of reading believe that the crucial deficit in poor readers is impaired phonological skill (Snowling, 2000).

There is no doubt that this is true; indeed it is almost a truism because phonological processing is the essence of reading. But the question arises why do so many children fail to gain these phonological skills? Although many poor readers start with nonvisual phonological problems at the syllabic level (e.g. poor ability at spoonerisms, rhyming and non word repetition (Bradley & Bryant, 1983) there is now much evidence that the ability to split words right down to their constituent phonemes depends heavily upon learning how word sounds are represented visually by letters. Thus fine grained knowledge of individual phonemes only develops after the child has learnt that words can be disassembled. She learns this by experiencing how these phonemic components are represented by the letters used to write them down (Morais, Cary, Alegria, & Bertelson, 1979). Therefore, it turns out that visual analysis of words is essential to grasp their structure at the phonemic level, hence that being able to form accurate visual representations of the written, orthographic, form of words actually precedes the acquisition of understanding their phonological structure. Hence young poor readers are worse at orthographic than phonological tasks and the former predict overall reading ability in unselected 10 years old better than do purely phonological tests (Talcott et al., 2000).

In this paper therefore, I will show how important low level visual processing is to reading. In particular I will present evidence that individual differences in the processing of visual motion by the visual magnocellular system correlate strongly with how well people can acquire orthographic skill. We and many others have found that exceptional difficulties with learning to read (developmental dyslexia) are associated with impaired development of visual motion processing. On the whole however, I am not going to use the term 'dyslexia' here because I want to emphasise that poor reading is one extreme of normal variation in reading. Very poor reading (dyslexia) is not qualitatively different from normal reading, just worse.

Since text is stationary when people read it, it seems odd to many people to argue that the network responsible for processing visual motion could be the system that is most impaired in poor readers. But actually the retinal images of letters and words are not at all stationary most of the time. When the eyes scan this stationary text, their images move about wildly on the retina. Only during brief fixations on words that last only ca. 300 ms, can their visual forms be taken in. The reason why the visual magnocellular system is so important in reading therefore seems to be because it plays such a dominant role in stabilising these brief fixations, in addition to directing the eye movements between them.

So visual magnocellular sensitivity helps to determine orthographic ability because it mediates the precision with which visual attention and eye fixation can be directed on letters in order to identify their correct order. Therefore, I will next discuss how magnocellular performance, indexed by visual motion sensitivity, correlates with the direction of attention and of eye fixations and movements, and how this in turn affects how well they can locate and spatially sequence small targets, such as the order of letters in a word.

I will next outline how boosting the magnocellular function of poor readers and treating their eye movement deficits can greatly improve their reading. This beneficial effect of intervention suggests that this magnocellular hypothesis about reading is likely to be quite close to the truth. I will conclude by describing some genetic and environmental reasons why magnocellular neurones are particularly vulnerable.

2. The visual magnocellular system

Ten percent of retinal ganglion cells are noticeably larger than the rest-magnocellular—with thicker myelination and more rapidly conducting axons. Their greater dendritic area means that they draw information from a 10 times larger area of retina than the smaller parvocells, hence they respond best to lower (ca. 0.5 cycle/degree as opposed to 5 cycle/degree) spatial frequencies but they have $10\times$ higher luminance, temporal and contrast sensitivity. They are probably not involved in colour perception, but it is now clear that they receive from long, medium and short wavelength cones, probably in the same proportions that these receptors are found in the retina (Roorda & Williams, 1999). So their greatest sensitivity is to yellow light.

The magnocellular system is specialised for timing visual events, hence for detecting visual motion. It communicates this information very rapidly to the visual cortex via the magnocellular layers of the Lateral Geniculate Nucleus and to the Superior Colliculus for the reflex control of eye movements. This system dominates the dorsomedial 'where' projection from the primary visual cortex to the middle temporal motion area, and from there to attentional and eye movement control regions in the posterior parietal cortex and beyond these to the frontal eye fields and cerebellum.

3. Visual magnocellular function in poor readers

After spending a sabbatical with Fergus Campbell in Cambridge to learn how to measure subjects' contrast sensitivity by using sinusoidal gratings over a range of different spatial frequencies, Lovegrove returned to his University in Tasmania to become the first to show that many very poor readers (dyslexics) have lower contrast sensitivity than good readers at the low spatial and high temporal frequencies that are signalled by magnocellular retinal ganglion cells

(Lovegrove, Martin, Blackwood, & Badcock, 1980). He was able to demonstrate a double dissociation: his Tasmanian dyslexics actually also had slightly higher contrast sensitivity than good readers at the higher spatial but lower temporal frequencies served by the parvocellular ganglion cells. We found the same kind of double dissociation comparing English poor readers who had unstable vision when reading (visual dyslexics) with good readers (Mason, Cornelissen, Fowler, & Stein, 1993).

However the claim that poor readers may have a magnocellular deficit has caused much controversy (Skottun, 2000). The differences in contrast sensitivity from good readers are small; not all studies have found them to be confined to magnocellular processing; and not all poor readers show them, especially if small numbers are studied and they are classified as poor readers mainly on their phonological, rather than on their visual/orthographic, performance. These problems probably arise because the magnocellular system is responsible for directing attention to all stimuli, even those that would maximally activate the parvo system. Hence it is impossible to entirely dissociate magno from parvo processing. Nevertheless most researchers in this area now concede that the main visual difference between good and poor readers is in their magnocellular function because the balance of evidence from such a wide variety of techniques so strongly supports this view (Stein, Talcott, & Walsh, 2000). Neuropathological, evoked potential, functional imaging results and a range of psychophysical tasks targeting magnocellular function all point in the same direction.

Perhaps the strongest evidence was provided by Livingstone, Galaburda and colleagues who studied the magnocellular layers of the LGN in the brains of deceased dyslexics post mortem (Livingstone, Rosen, Drislane, & Galaburda, 1991). They found that the magnocells in these brains were 30% smaller and significantly more disorganised than in control brains. Consistent with this they recorded reduced and delayed averaged evoked potentials in response to a visual motion stimulus in alive dyslexics. This result has been confirmed by most authors (Lehmkuhle & Williams, 1993), and disputed very rarely (Victor, Conte, Burton, & Nass, 1993). Likewise areas, such as the middle temporal area (MT/V5), that were expected to be activated by a visual motion stimulus in functional magnetic resonance imaging studies have proved to be less activated in poor readers (Eden, VanMeter, Rumsey, Maisog, & Zeffiro, 1986), and it has also been shown that their degree of activation correlates with reading proficiency (Demb, Boynton, & Heeger, 1997).

4. Psychophysical techniques

However most of the work relating magnocellular function to reading has used less expensive psychophysical methods, taking advantage of the fact that this system can be selectively activated using quite simple techniques. Low contrast, coarse, low spatial frequency, moving or flickering stimuli will stimulate magnocellular far more than parvocellular retinal ganglion cells, whereas the latter respond best to high contrast, finely detailed, high spatial frequency, coloured stimuli. Therefore, measurements of static and dynamic contrast sensitivity (Lovegrove et al., 1980; Mason et al., 1993; Slaghuis & Ryan, 1999) and of flicker fusion thresholds (Brannan & Williams, 1988) have been widely used to test whether retinal magnocellular sensitivity is lower in poor than in good readers. The consensus is that poor reading is weakly associated with reduced magno-sensitivity whereas parvocellular ganglion cell sensitivity has usually been found to be as good or better in poor readers.

Because of the ‘rectifying nonlinearity’ of magnocellular ganglion cells they are as sensitive to a black to white transition as to a white to black one. Hence a flickering, coarse grating presented at very low contrast so that it is detected only by magnocells, appears to have twice as many stripes as it has in reality. This is the ‘spatial frequency doubling illusion’ and because it is mediated exclusively by magnocellular retinal ganglion cells its sensitivity is a very good index of their function. This can be assessed very rapidly, and, as expected, poor readers are significantly less sensitive to the illusion than good readers (Pammer & Wheatley, 2001).

Thus although few of the stimuli used are entirely selective or depend exclusively on retinal magnocellular ganglion cell function the balance of evidence leaves little doubt that, as a group poor readers, have worse retinal magnocellular performance, whereas their parvocellular sensitivity may even be slightly better than good readers’. Showing good parvocellular sensitivity at the same time serves as a control for the possibility that poor readers are simply worse at all psychophysical tasks, due to factors like lack of vigilance or low motivation. These factors can also be controlled for explicitly by including ‘catch’, very easy, trials in the psychophysical procedure. Measuring the proportion of these that are missed gives an index of general attention, and this can be used as a covariate to ensure that group differences are not simply due to lack of vigilance, etc. Although it has been suggested that many of the differences attributed to poor magnocellular function in poor readers may simply be due to low ability to attend to the stimuli (Stuart, McAnally, & Castles, 2001), in most studies where this has been controlled for by comparing magno to parvo function or by explicitly measuring vigilance by means of catch trials, the magno deficit has survived.

Nevertheless there is great overlap between groups; some poor readers have good magnocellular sensitivity and some good readers have poor magnocellular sensitivity. Although significant statistically, the overall correlation of magno indices with reading ability is seldom better than $r = 0.45$, i.e. magno sensitivity only accounts for some 20% of the variance in people’s reading. This has prompted some, particularly those supporting a purely phonological explanation for reading problems, to deny that poor readers have any significant visual deficit at all, and to assert that the small

impairments found may be simply epiphenomena with no relevance to reading.

However the balance of evidence is now so strong, and there are now so many convincing demonstrations that treatments designed to improve poor readers' magnocellular function improve their reading, that this negative view is a difficult one to sustain. It seems more likely that the weakness of the relationship between magnocellular function and reading is due to limitations in our ability to measure people's magnocellular function accurately and selectively.

5. Cortical visual magnocellular system

In order to try to improve measures of visual magnocellular function to correlate better with reading ability, attention has turned from the retina to its cortical projections. Although there are many interconnections, the output of the primary visual cortex is partially divided into two streams. That projecting ventrolaterally is often known as the 'what' stream because it is thought to mediate object recognition. It receives approximately equal inputs from magno- and parvo-sources, and it projects forward to the inferotemporal cortex. The alternative dorsomedial 'where' stream projects forward to MT/V5 and the posterior parietal cortex. Milner and Goodale have shown that this dorsal stream is better termed the 'how' stream because its main function is to provide the visual guidance for the visual direction of attention, eye and limb movements (Milner & Goodale, 1995). It receives predominately magnocellular input, and consists itself of large, heavily myelinated, magnocellular neurones which express the same surface recognition, major histocompatibility (MHC), molecules as the retinal magnocells (Hockfield & Sur, 1990). Hence tests targeting the functions of the dorsal stream have been used to assess more of the visual magnocellular system in dyslexics, with greater sensitivity.

Unlike the primary visual cortex (V1), the middle temporal visual motion area (MT/V5), which is a pivotal station in the dorsal stream, is best stimulated not by orientated gratings but by randomly spaced dots moving in the same direction (random dot kinematograms—RDKs). No single moving dot has to last for longer than 100 ms; but because MT neurones sum over both time and over a large receptive field, so long as there is a significant motion vector in the whole ensemble of dots, global coherent motion is seen. By reducing the proportion of dots moving in the same direction, Newsome et al. showed in monkeys that the threshold of individual MT neurones' sensitivity to the global motion is equal to that of the whole animal. Furthermore at threshold the direction signalled by an individual MT neurone is the direction perceived by the monkey (Newsome, Britten, & Movshon, 1989).

The sensitivity of the cortical motion system can be measured in exactly the same way in humans using RDKs. Hence, we have been able to show that, as expected, poor readers tend to have lower sensitivity (Cornelissen & Stein, 1995; Talcott, Hansen, & Stein, 1998; Witton et al., 1998). Furthermore good readers tend to have higher motion sensitivity, so that coherent motion thresholds can account for about 25% of the variance in people's reading ability (Talcott et al., 1998, 2000; Witton et al., 1998). Importantly we have been able to show that motion sensitivity measured in this way accounts for variance in indices of visual/orthographic reading skill independently of any correlation with phonological ability (Talcott et al., 2000). This was important to demonstrate because it provided further evidence that M-cell sensitivity plays a causal role in the development of orthographic skill, rather than being simply an irrelevant epiphenomenon.

However seemingly small differences in the motion stimuli employed and in the psychophysical techniques chosen can make quite big differences to the ability of a RDK test to discriminate good from poor readers. The simplest way to programme a RDK stimulus is replot the background (non coherently moving) dots randomly, in which case the background appears to twinkle. However, if instead the dots are moved a few millimetres in random directions from their original position (Brownian motion), compared to those moving coherently all in the same direction, the task is perceptually harder, but better at discriminating between good and bad readers. This difference probably arises because random replotting is less likely than Brownian motion to activate neurones responding to other movement directions that would compete with those detecting the coherent motion. Hence the motion is easier to see.

We have also found that measuring motion sensitivity using two RDK panels side by side, with to and fro coherent motion, gives better discrimination than a single panel in which the different directions of movement have to be identified. We attribute this difference to the requirement in the former task to shift attention or eye movements over a wider area. In fact whether eye movements are allowed or not seems to make little difference to thresholds. Thus the different attentional requirements of different tasks seem to affect discrimination strongly, a conclusion that is not unexpected given the important part that the M-system plays in the direction of attention (Vidyasagar, 1999).

Systematically altering the characteristics of the RDK stimulus can be used to study the nature of the M-cell weakness in poor readers in greater detail. Thresholds remain the same over a fairly wide range of stimulus velocities in both good and poor readers whereas they are strongly affected by the density of the random dots (Talcott, Hansen, Assoku, & Stein, 2000). This suggests that it is the reduction in M-cell size shown in Galaburda's neuropathological studies of dyslexic brains post-mortem (Livingstone et al., 1991) that has the most deleterious effect on their function. These smaller M-cells seem to spatially under-sample moving stimuli compared with those in good readers, and that may be the main reason why they are less sensitive to motion.

6. Posterior parietal cortex

The visual cortical dorsal stream projects onwards to the posterior parietal cortex (PPC), so a wide variety of attentional and visuomotor tasks thought to be mediated by the PPC have also been investigated in poor readers. In visual search tasks qualitatively distinct targets can be detected rapidly from a background of distractors because they 'pop out'. But if target and distractor share similarities each has to be scanned in series to locate the differences. Even if no overt eye movements are made this scanning is carried out by covert shifts of attention. Hence it takes longer to detect the target the larger the number of distractors. Numerous studies have now confirmed that detection time is longer in poor readers with low visual motion sensitivity, particularly if the items are closely crowded together (Atkinson, 1991; Facoetti, Paganoni, & Lorusso, 2000).

The time taken to mentally compare rotated objects with each other varies with how far they have been rotated. Functional imaging and lesion studies have suggested that the PPC plays a crucial part in such mental rotation. Accordingly many poor readers are much slower at this task than good readers, and in functional imaging studies we have shown that their activation of the right intraparietal sulcus is reduced. We and others have found that this reduction is associated with a mild tendency of many poor readers to neglect the left side of space (left 'minineglect') (Hari, Renvall, & Tanskanen, 2001; Stein, Riddell, & Fowler, 1989).

Two illusions, the Ternus grouping effect and the line motion illusion, both of which are attributed to higher level magnocellular characteristics, have been particularly studied in poor readers. If the first of a line of small lights is transposed to the other end, at long interstimulus intervals the light seems to move from one end to the other. But at short ISIs the whole set of lights appears to move as a coherent group. This is the Ternus effect. Since it is thought to depend on the temporal resolving power of the magno system it can be used as a measure of magnocellular sensitivity. Accordingly many dyslexics retain the grouping effect at longer interstimulus intervals, confirming that they have lower magnocellular sensitivity (Cestnick & Coltheart, 1999). The line motion illusion is a similar phenomenon. Here a light is flashed some distance from the end of a line. At short interstimulus intervals this makes the line appear to move towards it. Again this critical interval is longer in many poor readers (Steinman, Steinman, & Garzia, 1998).

7. Magnocellular system and visual attention

Our capacity for processing visual information is limited; only a small part of the visual world can be analysed in detail at any one time. The word 'attention' is often used very loosely in this context as a portmanteau to conceal our ignorance of the true mechanisms of how we select what features to analyse, but here I want to restrict discussion to the role of the magnocellular system in directing visual processing resources to particular locations in space and also in time. This is the familiar idea that visual attention acts as an 'internal search light' to locate salient features in the visual world. This means that I am going to restrict myself to some extent to 'bottom up' features that grab attention, rather than discussing 'top down' cognitive factors that may direct attention according to expectation or motivation.

There is now much evidence that the M-system plays a preeminent role in the bottom up direction of attention (Vidyasagar, 1999). As noted earlier its main projections are to areas that are thought to control the direction of attention, eye and limb movements, such as the posterior parietal cortex, frontal eye fields and cerebellum. Attention and eye movements are of course closely related, because normally attention is disengaged from one target and shifted to another as a prelude to the eyes shifting from one to the other. Hence it has been known for a long time from the results of lesions in both the posterior parietal cortex and the frontal lobes that that they play important roles in the direction of visual attention, and recent evidence has also implicated the cerebellum (Courchesne, Townsend, & Akshoomoff, 1994). The importance of these areas has now been abundantly confirmed by the results of functional imaging experiments (Kastner, De Weerd, Desimone, & Ungerleider, 1998). Furthermore the greater speed with which the magnocellular system operates turns out to be key to models of how the strength of visual inputs are assessed to generate a 'winner takes all' saliency map of the visual world in the posterior parietal cortex. This controls the bottom up direction of the attentional spotlight on features likely to be important (Taylor & Stein,1999).

It is therefore not at all surprising that there have been many demonstrations that because their M-system is weaker, the speed with which poor readers can disengage and reengage attention on new targets is reduced compared with good readers (Brannan & Williams, 1987). This difference is so great that it seems promising to form the basis of a test that could be used to identify visual M-cell weakness in children with reading problems. Therefore, we have been developing such a test by measuring the effect of cueing attention to a patch of RDK coherent motion in good and poor readers. Our preliminary results suggest that good readers gain a big advantage from such cueing whereas poor readers seem to benefit far less, so that we have found remarkably high correlations (ca. $r = 0.7$) between the advantage subjects gain from attentional cueing and their reading ability.

It seems hopeful that we may be able to develop this into a more reliable test of M-cell sensitivity for diagnostic use. We aim to make this so simple that it can be administered to 6-year-old children just starting to learn to read, in order to predict those that are likely to encounter visual problems. This should enable teachers to start remedial measures before children begin to fail, and thus help to avoid the misery and loss of self confidence that such failure engenders.

8. Eye movement control

Most of those who research reading mechanisms believe that it is rare for poor acquisition of the visual representation of letters and words to be able to explain reading difficulties, but rather they argue that weak phonological skill is the main cause. Further it is often argued that if there were a visual problem it would be more likely to involve the parvocellular system because this would be more responsible for parsing the fine detail by which we distinguish letters. Yet as we have seen there is very little evidence of a parvocellular deficit in poor readers. Thus poor readers actually have very little difficulty discriminating individual letters. It is the sequencing of groups of letters in the right order that many children find so difficult. Indeed, often they actually report that this is because the letters appear to blur and move around when they are trying to decide what order they should be in (Cornelissen, Bradley, & Stein, 1991).

This illusion is very similar to what patients report who suffer unstable eye fixation control after parietal, frontal or cerebellar lesions. Since the magno system is known to provide the main visual projection to these eye control centres, in 1980 (Stein & Fowler, 1980). I put forward the hypothesis that these kinds of reading problems might be due to unsteady eye control. Since then we have published numerous studies supporting this idea. We first showed that a large proportion of children with reading problems had unstable eye dominance associated with unsteady perception of letters (Stein & Fowler, 1982). These children tended to have unsteady fixation and poor vergence control (Stein, Riddell, & Fowler, 1988). This means that in addition they tend to have poor ability to accurately localise or sequence small targets, such as letters, so that they tend to make a large proportion of letter position errors when attempting to read (Cornelissen et al., 1991) (Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1997). Hence they fail to lay down crisp and reliable memory representations of the visual form of words in their reading network. Even in well taught, well compensated, adult dyslexics this continues to manifest itself in permanently bad spelling.

Their unstable eye dominance can be improved however. We showed first in open control trials (Stein & Fowler, 1981; Cornelissen & Stein, 1992), then in two randomised double blind control trials (Stein & Fowler, 1985; Stein, Fowler, & Richardson, 2000), that patching the left eye when reading, to encourage stable eye dominance, helped many unstable children to gain stable dominance; this had the desired effect of steadying their perceptions. Afterwards these children doubled their reading progress compared with placebo treated children. But patching was only successful in younger children up to the age of 10.

Nevertheless older children can often be trained to stabilise their fixation by exercises. We give them feedback about how steadily they are holding their eyes, by taking advantage of the phenomenon of physiological diplopia. If they gain good fixation control their reading improves greatly thereafter (Stein, Riddell, & Fowler, 1987). These beneficial effects of stabilising gaze on perception and reading provide further evidence that unsteady eye control is an important cause of children's inability to lay down robust and reliable orthographic memories for reading.

In a sense it is paradoxical that the M-system that mediates motion processing plays such a particularly crucial part in maintaining a static binocular gaze. But this is especially important during the brief 300 ms fixations during which words are recognised when reading. Whenever unwanted movements of the eyes take place, the fixated images slip over the retina and generate powerful motion signals. These are very rapidly fed back by the M-system to ocular motor centres, particularly the superior colliculus, to bring the eye back on target. Hence in recent studies we have found that there is a high correlation between individuals' magnocellular sensitivity indexed by RDK thresholds and their fixation steadiness measured as the standard deviation of any eye movements that occur when they are attempting to steadily fixate a small target.

Since smooth pursuit of a moving target seems to be controlled by the same system that mediates fixation we find that smooth pursuit gain and distortion (power generated by the eye movements at frequencies other than that of the target) also correlate with M-sensitivity. These results confirm that, as suggested, M-cell input does indeed play an important part in maintaining steady fixation and in controlling smooth pursuit. Taken together with our work showing that unsteady eye control leads to the unsteady appearance of letters, hence failure to acquire crisp orthographic representations, this explains why M-cell function plays such an important part in the development of orthographic skills for reading.

9. Coloured filters

Another controversial treatment for children's visual reading problems is to give them coloured filters. First in 1980 Meares in Australia (Meares, 1980), then Irlen in the USA in 1991 (Irlen, 1991) and Wilkins (Wilkins & Neary, 1991) and ourselves in the UK in 1992 (Fowler, Mason, Richardson, Welham, & Stein, 1992) found that coloured filters can alleviate the uncomfortable visual symptoms that impair many children's ability to read. These include not only blur, movement, transpositions and reversals of the letters but also shimmer, glare and eye and head aches when reading. There have been very few properly controlled studies of these treatments, and those that have been published are not very convincing. Nevertheless there is strong anecdotal evidence to suggest that some children and adults are helped very greatly by coloured filters. Until recently however there has been no theory that has been able to link these effects plausibly with what is known about the visual impairments found in poor readers. Hence it has not been possible to predict which subjects will benefit or why, and

the gap has been filled with wild unsubstantiated claims, often put forward by practitioners who stand to benefit financially from the sale of their particular patented filter.

However it is now known that although the magnocellular system does not contribute to the perception of colour, nevertheless in daylight M-ganglion cells probably receive summed input from all three classes of cone roughly in proportion to their density in the retina, i.e. 45% from L- (red), 45% from M- (green) and 10% from S- (blue) cones (Roorda & Williams, 1999). This means that the peak sensitivity of the system is at yellow wavelengths. But there are individual differences in L-/M-ratios; so there may be slight differences in the precise position of different subjects' peak sensitivity.

Nevertheless we have found that about one-third of the children with visual reading problems that we see are markedly improved by a standard deep yellow light. Their contrast and motion sensitivity together with their eye control improve greatly and after 3 months wearing deep yellow filters or using yellow overlays when reading, their reading improves greatly. We have just completed a double blind randomised control trial comparing the effect of yellow filters with a placebo treatment that has confirmed this result. The placebo was a card with a window cut in it that the children ran over the text, so that they only saw one word at a time, since this technique has been claimed to help dyslexics. We did not know which treatment had been given to which child when we retested their visual performance and reading, and the children did not know which treatment was supposed to be the most effective. Nevertheless the reading age of those given the yellow filters improved by 8 months over the 3 months they were wearing them, whereas those given the placebo improved by less than 2 months; a six month advantage to those given the yellow filters.

However deep yellow filters do not boost midrange wavelengths in daylight; their main effect is 'negative blue', cutting out the short wavelength blue light. Their great effectiveness in some subjects implies that blue light may unduly inhibit visual function in these subjects. Recently it has been shown that S-cone input normally does indeed inhibit magno function (Stockman, MacLeod, & DePriest, 1991). Thus, negative blue, yellow, filters may help subjects with weak magno function by reducing this S-cone inhibitory input. This probably boosts their M-system to increase contrast and motion sensitivity, and this in turn helps them stabilise their eyes when reading.

About 10% of children with visual reading problems, especially those particularly complaining of glare, are not helped by yellow however, but they are greatly improved by deep blue ('negative yellow') filters. We have recently measured the sensitivity of these children's motion (M-) system to cone isolating coloured stimuli and found that this is ca. 10 times less sensitive to S-cone modulation than in controls. Thus this small group of children seems to have weaker S-cone input to M-cells than normal, so that blue filters probably help them by cutting down stimulation of their L- and M-cones. This may help the system to return to an appropriate balance of L-,M- and S-input.

10. Genetic and environmental influences on M-cell development

Thus there is now compelling evidence that reading proficiency depends on the contribution of the visual magnocellular system to the direction of attention and of eye movements to enable the acquisition of reliable orthographic representations. Poor readers seem to have weak magnocellular function. This leads to unfocussed visual attention and unstable eye control and these problems cause letters to appear to move around so that they find it difficult to determine their order. This in turn impairs their ability to acquire good orthographic skills.

After making use of this knowledge to help children circumvent these problems, naturally the most interesting question thereafter is why this magnocellular impairment is so common. Twenty percent of the population cannot read properly. Why is this?

Reading ability is strongly hereditary; twin studies suggest a heritability of 60% (Pennington & Smith, 1988). But since writing was only invented 5000 years ago it is most unlikely that any special genes have evolved for reading. It is much more likely that they evolved to control the development of magnocells for visuomotor guidance, and reading has piggy backed on that system. However, serious magnocellular deficiencies would be disastrous. Not noticing a sabre toothed tiger moving swiftly towards you would certainly prevent you passing on that gene to the next generation! So alleles that bequeathed severely deficient magnocellular function would be quickly selected out. But even alleles causing weak decreases in selective fitness would die out within a few generations unless they carried some advantages also. So it is likely that even the mild M-deficits that we see carry compensating characteristics to their holder that balance the disadvantage, to set up 'a balanced polymorphism'. And indeed poor readers are often found to be greatly talented in other areas.

Genetic linkage studies have shown, as expected, that actually many gene sites are involved in controlling reading ability (Marlow et al., 2002), probably as many as 10. However, one site is particularly interesting to us, we have confirmed that reading problems are linked to a site on the short arm of chromosome 6. This site has been located by at least four other groups, and it has attracted a great deal of attention because it is situated within the MHC immune regulatory region on C6. Genetically abnormal MHC control might explain why many poor readers and their families seem to have a much higher incidence of autoimmune problems, such as asthma, eczema and hay fever, and also more serious autoimmune diseases, such as disseminated lupus erythematosis (DLE) (Tonnessen, Lokken, Hoien, & Lundberg, 1993). They may also have a greater propensity

for inflammatory disorders, but interestingly it appears that this same propensity may protect them from high blood pressure and its deleterious effects on the heart, cerebral blood vessels and kidneys (Taylor & Stein, 2002). Possibly it also protects them from cancer.

Even more excitingly, Carla Shatz and co-workers have shown that this same MHC system plays a very important part in controlling the development of magnocells, including the visual ones, throughout the brain (Corriveau, Huh, & Shatz, 1998). Thus the allele inherited by poor readers may impair the development of visual magnocells via the MHC system, and this may partly explain their reading problems.

One possible mechanism for this damage may be transfer of antimagno-antibodies across the placenta that may damage magnocells during the rapid development of the foetal brain during the 3rd to 6th month in utero. We have injected into pregnant mice serum from mothers who have had two or more dyslexic children. We found that antibodies from this serum can pass across the placenta and the immature foetal blood brain barrier, and bind to the magnocellular cells of the developing cerebellum in the pups, so that after birth their coordination is impaired and their cerebellar metablism is abnormal in magnetic resonance spectroscopy studies (Vincent et al., 2002).

Yet other alleles connected with poor reading seem to be involved in polyunsaturated (essential) fatty acid (PUFAs) metabolism. This is of great interest also because polyunsaturated fatty acids constitute 20% of the dry weight of the brain; magnocells are known to be particularly vulnerable to PUFA deficiency (Ahmad, Moriguchi, & Salem, 2002); modern diets are deficient in oily fish which is the main source of PUFAs; and many poor readers show signs of PUFA deficiency (Taylor et al., 2000). Our brains contain so much PUFA because we evolved with the benefit of an abundant supply of oily fish. PUFA flexibility endows nerve membranes with high flexibility so that their channels can open and close very rapidly. Also omega 3 PUFAs, in particular, serve as substrates for synthesis of anti inflammatory prostaglandin and thromboxane signalling molecules. We have recently completed a number of randomised control trials that have confirmed that supplementing the diet of children with reading problems with extra PUFAs can actually improve their attention, behaviour and reading (Richardson et al., 1999). This adds to the evidence that an adequate supply of PUFAs in the diet is indeed important to the well being of our magnocells.

11. Conclusion

Thus, it seems that the genes that we inherit from our parents control the development of visual magnocells in utero and influence their health later by a combination of MHC controlled immunological effects and influences on PUFA metabolism, in concert with the availability of PUFAs in the diet. The performance of these magnocellular neurones, in turn, determines how well we can learn to fixate, attend to and perceive the important visual features of print. This therefore, is how the sensitivity of our visual magnocellular neurones helps to determine how well we can gain and use orthographic information for reading.

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