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Visual factors in reading

Chris Singleton* and Lisa-Marie Henderson
University of Hull, UK

This article reviews current knowledge about how the visual system recognizes letters and words, and the impact on reading when parts of the visual system malfunction. The physiology of eye and brain places important constraints on how we process text, and the efficient organization of the neurocognitive systems involved is not inherent but depends on the experience of learning to read. Although studies have reported that many children with severe reading difficulties have problems with visual fixation of words, in this context the concept of 'visual dyslexia' remains controversial. Evidence that a significant proportion of children with dyslexia have impairment of the magnocellular component of the visual system (which responds to contrast and movement) has led to alternative theories challenging the phonological deficit model of dyslexia. Deficits in the magno system have also been proposed to explain symptoms of visual stress that many people experience when reading, and to account for the alleviation of these symptoms by the use of coloured overlays or tinted lenses. However, a competing theory that posits cortical hypersensitivity to pattern glare as the cause of visual stress is generally more favoured at the present time. Our scientific understanding of reading would be much improved if visual factors were better integrated into theories that currently focus almost exclusively on phonological factors, but some progress towards this is being made.

Visual factors in reading

Reading involves the complex integration of visual and phonological information, beginning with visual encoding of letters. Sensitivity to the position of local characters in a word-like string of letters predicts reading ability in children and word recognition in adults (Pammer *et al.*, 2004). This suggests that readers are sensitive to the internal visual characteristics of words (Pammer & Vidyasager, 2005), a view supported by evidence from neuro-imaging studies (see Tarkiainen *et al.*, 1999), and findings that visual word recognition is impaired in dyslexic readers (Samelin *et al.*, 1996). There are several theoretical models of word recognition (see Lupker, 2005, for a review) but the most widely respected are variants of an interactive activation model first proposed by McClelland and Rumelhart (1981). The basic principle

*Corresponding author. Department of Psychology, University of Hull, Hull HU6 7RX, UK. Email: c.singleton@hull.ac.uk

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behind these models is that features of letters are first detected by the visual system, with activation 'cascading' to a letter detection stage, and finally to a word detection stage. However, when sufficient features of a letter become activated for that letter to be recognized, feature recognition is inhibited, and when sufficient letters of a word become activated for that word to be recognized, letter recognition is then inhibited. This feedback restrains the system from further (wasteful) identification of features or letters when the next stage is satisfied with what has already been identified. Interactive activation models (see Seidenberg & McClelland, 1989; Grainger & Jacobs, 1996; Coltheart *et al.,* 2001; Whitney & Cornelissen, 2005) also provide explanations for many of the psychological characteristics of word recognition, such as word frequency effects (more common words are recognized faster) and semantic effects (e.g., when words within a category, such as 'animals', are expected, these words are recognized faster than other words).

Eye movements in reading

When reading, the eyes do not move smoothly but in a series of very quick jumps (saccades) in order to fixate successive stable images. This process is activated so that (usually) each individual word is rapidly fixated in turn, each fixation (ideally) giving us a clean, clear image that is stored in working memory. About 10–15% of fixations made by skilled readers are regressions: i.e., movements of the eyes backwards to previous parts of the text. Eye movements are influenced by the reader's proficiency: skilled readers tend to make shorter fixations, longer saccades and fewer regressions (Rayner, 1998). In beginning readers, average fixation duration is 50% longer than in adult readers and twice as many fixations and regressions are made. Because children with reading difficulties also show shorter saccades, longer and more fixations, and more regressions than normal readers, it has sometimes been suggested that faulty eye movements are a cause of poor reading. However, the weight of evidence does not support this view; rather it implies that efficient eye movements are disciplined during normal reading development and consequently if reading development is abnormal then eye movements will tend to lack efficiency (Olson *et al.*, 1983; Hyönä & Olson, 1995; Kulp & Schmidt, 1996; Rayner, 1998).

Eye movements are also a function of the difficulty of the task. Proficient readers often skip function words (prepositions, conjunctions, articles and pronouns) and spend more time fixating content words (nouns, verbs, adjectives and adverbs), especially if these are unusual. In skilled readers the decision *when* to move the eyes during reading appears to be related to high-level factors such as the lexical and syntactic complexity of the text, and regressions are usually a response to comprehension failure (Kennedy *et al.*, 2003). The decision *where* to move the eyes seems to be more a function of low-level factors such as word length. However, explaining exactly how the brain controls eye movements in reading is no easy task, given the speed at which several complex processes are being executed (for review see Rayner *et al.*, 2005). For example, how do we know (without looking) that a function word is coming up and hence we may be free to skip it? Part of the explanation seems to lie in the fact that when reading the perceptual span extends some characters ahead of the fixated word, and a limited amount of information is available about the text that falls within that span. Beginning readers have smaller perceptual spans than more experienced readers, thus the experience of reading itself enables children to develop greater fluency and increased speed, not only by practising word recognition skills but also by learning to exert top-down control over eye movements so that patterns of fixation can become more efficient.

Vision and reading difficulties

If the lenses of the eye do not focus properly various types of refractive error arise, such as short-sightedness (myopia), long-sightedness (hypermetropia or hyperopia) and astigmatism (a condition in which the eyeball is not spherical, causing lack of clarity in some aspects of vision). About 10% of schoolchildren are short-sighted, 5% long-sighted and 5% have some degree of astigmatism; without correction by spectacles these problems can impact on reading acquisition (Evans, 2001). The automatic focusing of the eye to retain a clear image (accommodation) can also be subject to a number of anomalies that may respond to eye exercises. Refractive and orthoptic problems are quite common and therefore it is essential that children's vision is checked when starting school and followed up by further checks in later primary stages. Children who display anomalies or symptoms at any time should be referred for full eye examination (see Lightstone & Evans, 1995; Thomson, 2002).

Several studies have reported findings of poor control of fixation in children with dyslexia (see Fischer & Weber, 1990; Eden *et al.*, 1994). More than half of a large sample of children with reading difficulties studied by Stein and Fowler (1982) were reported not to have a stable or fixed 'reference eye' when given tests that required either convergence or divergence of the eyes. They claimed this demonstrated these children had failed to establish 'ocular motor dominance' and called the condition 'visual dyslexia'. Stein and Fowler (1985) further reported that monocular occlusion of one eye frequently led to both a development of a fixed reference eye and improvement in reading. Subsequent studies did not always replicate these findings (see Bigelow & McKenzie, 1985; Newman *et al.,* 1985) and discrepancies could well have been due to the unreliability of the methods employed to assess vergence control (see Bishop, 1989; Stein & Fowler, 1993). Subsequently, Cornelissen and colleagues reported that the type of errors children made in reading and spelling were a function of whether or not they had a stable reference eye (Cornelissen *et al.*, 1991, 1994). However, Goulandris *et al.* (1998) did not find that orthoptic tests discriminated between dyslexic and reading-age matched controls, and Everatt *et al.* (1999) found that poor vergence control was not common in adult dyslexics. In summary, it seems unlikely that poor vergence control is a significant cause of reading difficulties and hence the concept of 'visual dyslexia' remains highly controversial (see Beaton, 2004, and Evans, 2001, for reviews).

Magnocellular deficit theory of reading difficulties

There are two types of cells in the neural pathways that subserve vision, differentiated by size and function. Magnocells are large and code information about contrast and movement, while parvocells are smaller and code information about detail and colour. These two systems work together, the magno system inhibiting impulses in the parvo system while the eyes are moving, enabling us to perceive a stationary image even though the eyes are in almost constant movement. Research to date is consistent with the position that a proportion of individuals with dyslexia have a low-level visual impairment affecting the magno visual system (Cornelissen *et al.*, 1995; Evans, 1997; Stein & Walsh, 1997; Talcott *et al.*, 2000) that persists beyond childhood (Conlon *et al.*, 2000). The magno system is important for directing visual attention, control of eye movements, and visual search—three skills having key roles in reading (Edwards *et al.*, 1995). Thus if the magno system is dysfunctional it is not unreasonable to anticipate problems in smooth and efficient processing of text. However, mechanisms by which this might occur remain highly speculative at present (see Merigan & Maunsell, 1993; Eden *et al.*, 1996; Stein & Walsh, 1997; Vidyasagar, 1999, 2001; Hari *et al.*, 2001; Stein, 2001; Facoetti *et al.*, 2003; Pammer & Vidyasagar, 2005).

Evidence for the magno deficit comes from four main types of psychophysical study.

- Dyslexics have a reduced ability to detect flicker (see Evans *et al.*, 1994).
- Dyslexics often have reduced ability to detect coarse detail, but a normal ability to detect fine detail (Livingstone *et al.*, 1990).
- There tends to be a prolonged persistence of the visual image causing masking of vision on successive fixations (see Slaghuis & Ryan, 1999).
- There is a decreased ability to detect fine motion (see Cornelissen *et al.*, 1995).

Anatomical support for the magno deficit was provided by Livingstone, Galaburda and colleagues, who found that the magno cells in the brains of deceased dyslexics postmortem were 30% smaller and more disorganized than in normal brains. Weaker evidence for the magno deficit is also provided by electrophysiological studies (see Demb *et al.*, 1997).

Although the balance of evidence indicates that the main visual difference between good and poor readers is in their magno function (Stein *et al.*, 2000), the magno impairment in dyslexia is subtle and has been disputed (see Skotton, 2000). Hulme (1988) argued that if there is a direct relationship between visual impairments and reading difficulties, then dyslexic children should have more problems in reading prose rather than single words; this is seldom the case. It is unclear how impaired pronunciation and manipulation of isolated words and non-words, hallmarks of dyslexia, could be caused by magno deficits (Hayduk *et al.*, 1996). It has also been suggested that the magno deficit may reflect a more generalized deficit in attention (Stuart *et al.*, 2001) or that only a subgroup of dyslexics have a magno deficit (Borsting *et al.*, 1996).

Visual stress

Meares (1980) and Irlen (1983) observed that some people experience perceptual distortions when reading printed text and that the problem can often be alleviated by using coloured overlays (sheets of transparent plastic that are placed upon the page).

This condition, usually now called 'visual stress', has also been known by various other labels, including 'Meares-Irlen syndrome', 'visual discomfort' and 'scotopic sensitivity syndrome'. The main symptoms include asthenopia (sore, tired eyes; headaches; photophobia) and visual perceptual distortions (illusions of shape, motion and colour; transient instability; diplopia [double vision]). Prevalence rates range from 5–34% for the general population depending on the criteria used (Wilkins *et al.*, 2001; Evans & Joseph, 2002; Kriss & Evans, 2005). Visual stress can interfere with the ability to read for long periods and so is likely to hinder reading development, since the rapid and accurate decoding of text promotes reading fluency (Tyrell *et al.*, 1995). Although visual stress can occur in good readers it is more frequently observed in poor readers (Kriss & Evans, 2005) and often reported by dyslexics (Cornelissen *et al.*, 1994; Stein & Walsh, 1997). Thus, if visual stress is not identified early in childhood there may be serious negative consequences for educational attainment. Wilkins *et al.* (2004) also maintain that reading materials and reading tests for young readers are often printed in text that is too small and have visually stressful characteristics, hence these materials may not only cause discomfort which discourages interest in reading but may also result in underestimation of children's reading competence.

Visual stress is most commonly and successfully treated by the use of coloured overlays or precision tinted lenses (Wilkins *et al.*, 1992, 1994; Evans *et al.*, 1995, 1996; Robinson & Foreman, 1999; Wilkins, 2003). Indeed, visual stress has frequently been defined and diagnosed as a condition that is 'alleviated by the use of individually prescribed coloured filters' (Kriss & Evans, 2005, p. 350). The typical protocol starts with a screening test using coloured overlays, which is usually administered by a teacher or optometrist. If children express a preference for a coloured overlay then this is evaluated by measuring improvement in reading speed with the overlay and/or the voluntary sustained use of the overlay for at least half of a school term. The Wilkins rate of reading test (WRRT) was devised to test for immediate benefit in rate of reading: this assesses visual factors in reading and is relatively unaffected by cognitive factors (Wilkins *et al.*, 2001). Symptom questionnaires (see Irlen, 1991; Conlon & Hine, 2000) are frequently used to give an initial clue as to whether a child suffers from visual stress and may benefit from an overlay. However, although questionnaires may indicate susceptibility to visual stress in adults (see Evans & Joseph, 2002; Singleton & Trotter, 2005), data obtained by questioning children about suspected visual perceptual symptoms can be unreliable. Diagnosis by evaluation of the child's response to using an overlay is usually argued to be the next best thing. Unfortunately, at present there is no completely objective diagnostic test for visual stress, but a new computerized reading-like visual search task has recently shown promise as a screening system (Singleton & Henderson, in press).

There are two competing theories of visual stress. The first is provided by Wilkins and colleagues (see Evans, 2001; Wilkins, 1995, 2003), who maintain that visual stress is caused by pattern glare, i.e., a general over-excitation of the visual cortex due to hypersensitivity to contrast. The second is the magnocellular deficit theory (Livingstone *et al.*, 1991; Stein & Walsh, 1997; Stein, 2001). Stein (2001) argues that boosting magno performance using yellow filters can improve reading performance by increasing contrast and motion sensitivity. Yellow filters cut out the short wavelength blue light, implying that blue light may inhibit magno function in visual stress. However, Wilkins (2003) dismisses the magno deficit theory of visual stress, arguing that it is unable to account for the idiosyncrasy and specificity in optimal colour for reading. Wilkins also regards visual stress as independent of dyslexia, even though in some individuals the two conditions may be seen to have symptoms in common. Nevertheless, susceptibility to visual stress in a dyslexic child is likely be a critical factor mediating the efficacy of any conventional educational intervention.

Conclusions

Cornelissen (2005) observed that the field of reading research is split into 'language researchers' and 'vision researchers', the former being the more numerous and more conspicuous. The lack of communication between these camps has obstructed development of a full understanding of reading. Recent research, supported by evidence from brain-scanning studies, seems to be generating wider respect for the role of vision in reading, but we are still some way from a satisfactory theory that properly integrates visual factors with the other cognitive processes involved in reading. The phonological deficit theory of dyslexia has attained the status of orthodoxy (for reviews see Snowling, 2000; Ramus, 2001; Vellutino *et al.*, 2004) and attempts to integrate visual factors (such as magnocellular deficits) into that perspective (see Stein, 2001) have not been particularly successful. The mantle has recently fallen on a small vanguard of vision researchers (see Pammer & Vidyasager, 2005; Whitney & Cornelissen, 2005) whose models offer interesting prospects for the integration of visual factors into our understanding of the processes of reading and are evidence of progress towards more of a balanced theoretical perspective in the field as a whole.

Notes on contributors

Chris Singleton is a chartered psychologist and Senior Lecturer in Educational Psychology at the University of Hull. His main research interests are in the identification of dyslexia and other learning problems, for which he has developed innovative computer-based assessment systems that are widely used in schools, colleges and universities.

Lisa-Marie Henderson graduated with first class honours in psychology from the University of Hull, receiving the British Psychological Society's prize for top graduate of 2005. After a spell as Research Assistant for Chris Singleton,

investigating visual stress, she is currently a postgraduate research student at the University of York.

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