
■ Impaired Neuronal Timing in Developmental Dyslexia—The Magnocellular Hypothesis

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Developmental dyslexia is not just a literacy problem. Dyslexics' reading and spelling difficulties are but two of a much larger number of differences between dyslexic and normal readers. The condition is a wide-ranging, genetically based, neurodevelopmental syndrome. Reading requires fast and accurate processing of transient visual and auditory stimuli, functions for which large neurones, known as magnocellular, are specialized. We review the evidence that many dyslexics have impaired function of the visual magnocellular system, which correlates with their reading impairment, whereas good readers have high magnocellular sensitivity. We discuss possible mechanisms for this relationship. Although there is no such clearly defined magnocellular pathway in the auditory system as there is for vision, there is an analogous set of large auditory neurones which are specialized for following changes in the frequency or amplitude of sounds. We review evidence that the sensitivity of this auditory transient system is reduced in many dyslexics and that this reduction correlates with, hence may cause, their impaired phonological ability. As for the visual magnocellular system we show that auditory transient sensitivity predicts phonological and reading ability not only in dyslexics but also in good readers. Thus the magnocellular hypothesis postulates that dyslexics have lower sensitivity to dynamic visual and auditory stimuli as a result of slightly impaired development of large neurones and that this may explain not only their visual problems when attempting to read, but also their phonological deficit. Copyright © 1999 John Wiley & Sons, Ltd.

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NEURODEVELOPMENT

We first review the evidence that developmental dyslexia (hereafter referred to simply as dyslexia) is a broad neurodevelopmental syndrome. Developmental 'word blindness' was first described by Pringle Morgan in 1896; he presumed that it was inherited, and it has since become clear that reading ability is indeed highly heritable (Morgan, 1896). Recent estimates put the amount of variance in reading-related skills that can be attributed to genetic control, its 'heritability', at about 60% (Pennington, 1991). Mode of inheritance is probably autosomal dominant with reduced penetrance; in other words, 50% of a dyslexic's children (or 100% if both husband and wife are dyslexic) inherit vulnerability to reading problems, although there is still a significant contribution made by early intrauterine and childhood environment. Linkage of reading ability to a site near the genes on the short arm of chromosome 6 which control the major histocompatibility (MHC) antigens is now firmly established (Cardon *et al.*, 1994; Grigorenko *et al.*, 1997; Fisher *et al.*, 1999), and in some families linkage to other immunologically related sites on chromosomes 1 and 15 has been reported.

These possible associations with genes affecting antibody function are of great interest, because there is some evidence that dyslexics and their families have a greater-than-normal incidence of asthma, eczema, hay fever and autoimmune diseases (Geschwind and Behan, 1982; Geschwind and Galaburda, 1987; Hugdahl, Synnevag and Satz, 1990), although this idea has been disputed (Bryden, McManus and B-Fleming, 1994). Nevertheless, if it is confirmed, one could speculate that dyslexics' neurodevelopmental abnormalities might be signs of a mild autoimmune attack during early intrauterine development. No linkage to the sex chromosomes has ever been reported, which is surprising given the probability that dyslexia is commoner in boys. This sex difference may have been exaggerated by under-referral of girls, who tend to be less troublesome (Shaywitz *et al.*, 1990), but it still lacks an explanation. In summary, it is clear that developmental dyslexia is usually hereditary and under the control of probably only a small number of genes.

Genes control the synthesis of proteins; in the case of dyslexia, currently it seems most likely that the proteins concerned are involved in brain development. Galaburda *et al.* (1985) studied a number of brains of known dyslexics *post mortem* and found that they had obvious neuropathological abnormalities, which probably occur towards the end of the second trimester of foetal development, at around 24 weeks gestation when the cerebral cortex is developing and folding most rapidly. The most common of these abnormalities were cortical ectopias occurring mainly in the temporo-parietal and frontal association areas in both hemispheres, but particularly on the left. These brain 'warts' are about 1 mm in size and they form because disobedient developing neurones migrate past the outer limiting membrane to form an abnormal outgrowth on the surface of the cortex. The cause of the breaching of the outer limiting membrane is unknown. The outgrowths are associated with both anatomic and biochemical disruption of all the normally neat six layers of cortex beneath them, for several millimetres on either side, and also in equivalent, 'homotopic', areas in the opposite hemisphere to which they are connected by fibres travelling in the corpus callosum. Other abnormalities of development were also seen in the dyslexic brains, such as microgyrias, which are tiny aberrant infoldings of the cortex. Experimentally these can be mimicked in newborn rodents by briefly applying a freezing probe to the scalp. In the female dyslexic brains particularly, glial

scars were also found; these suggest that in females the injury may take place a little later in cortical development.

These microscopical cortical abnormalities are associated with macroscopical differences. In most normal subjects the left planum temporale, an area behind the primary auditory cortex in the superior temporal gyrus that is known to be important for language function, is up to 10 times larger on the left than on the right. However, in dyslexic brains often the right planum is as large as or larger than the left; in other words, the brains of dyslexics are more symmetrical than normal (Hynd *et al.*, 1990). In addition, when Galaburda and colleagues examined the visual and auditory relay nuclei in the thalamus, they found selective disruption of the large, magnocellular, neurones there (Livingstone *et al.*, 1991; Galaburda, Menard and Rosen, 1994).

Clearly there is no reason why these developmental anomalies should only affect reading. Their predominance in the left hemisphere should affect other aspects of language and praxis; those in the right hemisphere should affect visuospatial skills also (Stein, 1994). Accordingly dyslexics demonstrate a wide variety of other symptoms. Children who subsequently develop reading problems are often slow to learn to crawl, walk and speak. They also tend to be somewhat clumsy, and many never learn to ride a bicycle. Fawcett, Nicholson and Dean (1996) have recently documented their tendency to display mild cerebellar signs such as muscle hypotonia (slackness), poor balance and slow tapping (a test for adiadochokinesis—inability to make rapidly repeating movements). In addition, we have recently shown by magnetic resonance spectroscopy that cerebellar metabolism in dyslexics is significantly different from that of normal readers (Rae *et al.*, 1998).

Another problem that dyslexics often demonstrate is difficulty with distinguishing left and right; they tend not to know one from the other, and it is claimed that they tend to use either hand indiscriminately, though again this is controversial (Miles, 1993). Such ambidexterity might result from failure to establish clear hemispheric dominance as a result of the lack of hemispheric asymmetry discussed earlier. Another characteristic of developmental dyslexics is that they have problems with timing and sequencing. Their unpunctuality is legendary and they tend to mix up the order of the days in the week and the months of the year (Miles, 1993).

To summarize so far therefore, developmental dyslexia is not just a problem of reading and writing; these are signs of a much more widespread, genetically based, neurodevelopmental difference between dyslexics and normal readers. However, it is unlikely that the genes which underlie dyslexia problems evolved to impede reading. Probably they have survived in the human genome because they carried compensating advantages which may have been more important thousands of years ago. After all, reading has only been important to the average person for the last 100 years or so.

NORMAL READING

The vast majority of children learn to speak without any particular teaching and with little difficulty. Yet a few years later when they come to learn to read, they need to be taught how to do it; they do not pick up reading by themselves. Why is reading so much more difficult than speaking?

By the time a child is 7 years old, he/she has a vocabulary of about 3500 words, mostly of one or two syllables. Isabelle and Alvin Liberman have argued persuasively

that it is because we speak in syllables that we find reading so difficult (Lieberman *et al.*, 1974). Each syllable is generated by a separate articulatory gesture, a continuous sequence of contractions of the laryngeal, voice canal and tongue muscles. There are over a thousand million possible syllables, but fortunately only a small proportion of these has to be learnt in order to speak any language fluently.

The letters we have to learn to read represent not syllables, but the smallest acoustic units that are distinguishable, phonemes. The great step forward for reading that was made a few thousand years ago in the Fertile Crescent was the discovery that syllables could be represented on paper by a far smaller number of symbols if they were broken down to their smallest acoustically distinguishable components, phonemes. Identifying which ones each syllable contains, phonological analysis, requires the segmentation of each into its constituent phonemes. There are only about 40 phonemes in English, and in our alphabetic system these are represented by a still smaller number of only 26 single letters. However, Chinese script still employs over 5000 syllabic or logographic symbols, imposing a huge memory burden on children.

In western writing we break each syllable down into individual phonemic segments which are represented by alphabetic symbols. Thus phonemes are a human invention, and unlike syllables they are not generated by neurologically distinct programs; physiologically they are 'arbitrary'. In order to identify them, a child needs to learn to make acoustic discriminations that are far more subtle than for speaking. Thus they are difficult to learn, and this is why children have to be explicitly taught how to break down syllables into phonemes and how these are represented by letters, whereas they usually have little trouble teaching themselves to speak.

Accurate Visual Processing Is Required for Reading

Phonological acoustic analysis is not the only difficulty that reading presents. Subtle visual details of letter shape and order must be identified as well. The problems this presents are exemplified by the so-called 'minim problem' (Venezky, 1993). When the invention of printing changed reading from being a monopoly of a small, highly educated elite to a much more common accomplishment, it became necessary to make words more visually distinct so that ordinary people could discriminate them easily. For example, when the word 'mother' was written by scribes in the middle ages, it would have been phonetically spelt 'muva'. When handwritten, this succession of vertical strokes was made distinguishable by the individual scribe. However, when standardized for printing, these up and down lines could be easily confused; 'muva' (mother) could be mistaken for 'umun' (human), 'wun' (one) or 'wimin' (women). Therefore English spelling was altered by introducing visual irregularities to make such confusions less likely. Thus the large number of homophones in the English language, words that sound similar but have different meanings, is the main reason why we have so many odd and irregular spellings.

This multiplicity of irregular spellings means that in English precise visual as well as auditory sequencing is important for reading. Obviously blind people cannot read normal text; but, once learnt, reading is not compromised by severely reduced vision. However, there are many neurological disorders, such as multiple sclerosis, posterior parietal lobe strokes and ophthalmoplegias, that affect visual sequencing more than visual acuity. Such visual sequencing problems are much more disabling than might be expected, because they prevent the patient from being able to read properly.

Nevertheless, the visual contribution to learning to read, hence to dyslexia, is constantly underestimated. One reason is that simple reduction in visual acuity does not impede reading greatly in already proficient readers. It is remarkable how poor vision has to become before it has any marked effect in skilled readers. Likewise, normal subjects can read in lighting conditions that are very far from ideal. Thus more subtle impairments of visual processing than simply a loss of visual acuity are responsible for reading problems.

Another reason why the importance of the visual input to the reading system is so often underrated is that language is such a unique feature of humans that it is natural to assume that all its defects are specifically linguistic. Our ability to speak and the manual dexterity which enables us to write are the most important evolutionary advances which have raised us above the apes. The neurological system for language which is mainly situated in the left hemisphere is what makes us human, and so there is a very natural resistance to the idea that it might depend on the same basic visual or auditory processes that we share with lower animals.

Two Routes for Reading?

In some ways it is even more difficult to understand why hearing has any important part to play in learning to read. After all, when we are reading to ourselves, we are not using our ears. When reading familiar words, it is not essential for skilled readers to use phonological analysis at all. We can recognize the visual patterns of whole words because they have been laid down in our 'sight vocabulary'. Thus we can in theory retrieve their meanings (semantics) using a direct visual route from word to meaning without needing to translate the letters into sounds or syllables. Paradoxically this is precisely the strategy that very young children use when they first begin to learn to read. Before they have learnt the alphabet principle that syllables can be broken down into phonemes and matched with letters, they learn to recognize only the whole pattern of the first simple words that they learn; then they associate these directly with their meanings. This is the strategy exploited by the 'flash card' and 'look and say' techniques for teaching young children to read.

However, a purely visual strategy cannot help with reading unfamiliar words, and most words are unfamiliar to a beginning reader. The letters of the new word have to be translated into their corresponding phonemes; from these the child can reconstruct its sounds if it is regularly spelt, hence its meaning. This is the auditory/phonological route for reading (Ellis, 1992). Recent evidence shows clearly, however, that these two routes tend to be activated together. The phonological system is primed even when the direct semantic route is the main one being used (Pennington *et al.*, 1990), and neural network models of reading suggest that the converse is true (Seidenberg, 1993). Nevertheless, for a child to become a good reader, both these strategies, the visual semantic and the auditory/phonological, need to be proficiently learnt. Thus it is natural to wonder whether children with developmental dyslexia may lack the ability to acquire one or both of these skills.

ACQUIRED DYSLEXIA

In patients with brain damage, either of the two routes can be selectively affected, suggesting that they are, at least to some extent, neuroanatomically distinct. If the

lesion disrupts the visual semantic route specifically, then the patient has to rely entirely on the phonological route for reading—surface dyslexia (Coltheart *et al.*, 1983). Such patients can read regular words relatively easily. They can also successfully read artificial nonsense words that are constructed using standard phonetic rules—‘non-words’ such as ‘midbod’, a word that means nothing but can easily be read by anyone who knows the phonetic rules of English pronunciation. On the other hand, surface dyslexics find words which are irregularly spelt, such as ‘yacht’, very difficult to read, because they do not obey the phonological rules and so their meaning cannot be deduced simply by applying the rules. They can only be recognized by visual analysis. Also, because surface dyslexics have to rely on phonetic analysis entirely, they tend to make spelling errors that are phonetically plausible but visually incorrect; they make ‘phonetic regularization errors’.

In contrast, in phonological or ‘deep’ dyslexics the phonological route has been damaged (Coltheart, 1980), so they have to rely on the visual semantic one alone. Therefore they tend to make visual errors in which the word they produce is visually similar to the correct one but with letters confused or in the wrong order, e.g. ‘bind’ might be misread as ‘blind’ or ‘bond’. In addition, deep dyslexics tend to make semantic errors in which the word produced is similar in meaning but unrelated in any phonetic or even visual way to the correct one, e.g. reading ‘ship’ as ‘boat’. This kind of error may occur because the patients recognize the visual form of the word, hence its meaning; but because they cannot check it against its phonetic form, they have no means of ensuring that they do not retrieve a different word with a similar meaning (Coltheart, 1980). Obviously phonological dyslexics cannot read non-words because they have never seen them before; they cannot pronounce them because they have not been entered into their sight vocabulary.

PHONOLOGICAL AND VISUAL DEVELOPMENTAL DYSLEXIA?

Not many acquired dyslexics fall neatly into either of these two categories, however. Most display a combination of both phonological and visual problems, perhaps with one kind of error predominating. In the same way, most developmental dyslexics make both visual and phonological reading errors, although one or the other kind may predominate (Castles and Coltheart, 1993; Seidenberg, 1993). However, the probable existence of these two different routes for reading emphasizes that successful acquisition of both visual and phonological processing skills is necessary to learn to read properly. Bradley and Bryant (1983) found that visual training in letter forms as well as phonic training in the sounds that the letters represent is necessary to achieve the fastest reading improvement in 4–8-year-olds. Thus acquisition of accurate visual and auditory processing skills is vital for successful reading. This conclusion also emphasizes that the difficulties that developmental dyslexics face probably have a neurological basis, since the reading errors made by acquired and developmental dyslexics can be so similar.

VISUAL MAGNOCELLULAR SYSTEM

The largest cells in the brain, which range in size from 50 to 150 μm in diameter, are known as magnocellular neurones. Many of them probably derive from common

progenitors, because they express the same surface antigen. This means that these nerve cells can be identified by specific antibodies, but it also makes them vulnerable to attack by these same influences. In general, owing to the fast kinetics of their membrane channels and their heavy myelination, magnocells are specialized for following changes, transients, in sensory and motor signals throughout the nervous system. Most is known about the visual magnocellular system, so we will start by discussing the function of visual magnocells, first in normal vision, then in the visual dysfunction of developmental dyslexics.

In the eye and optic pathways, large (magno) and small (parvo) retinal ganglion cells can easily be distinguished; about 10% are magnocells (Shapley and Perry, 1986). As befits their larger size, they have large receptive fields; this means that they tend to respond over a fairly large area of visual space, rather than to the fine details of an object which betray its identity. Another important characteristic is that they can follow rapid changes in illumination (visual transients—Enroth Kugel and Robson, 1966). Thus they signal when new events occur in the visual world, such as the flickering or movement of interesting targets. They also respond well at low light levels and to low contrasts, so that the difference between the light reflected from an object and from its background does not have to be very large. However, they do not signal colour or fine detail (Merrigan and Maunsell, 1993); hence they are not responsible for visual acuity as measured by standard eye tests.

The axons of retinal magno ganglion cells are large and thickly myelinated, so they conduct signals rapidly to the first visual relay in the brain, the lateral geniculate nucleus (LGN) of the thalamus. The deep layers of this nucleus are specifically magnocellular and relay timing signals rapidly to layer 4C α of the primary visual cortex. After this, magnocellular signals intermingle with parvocellular signals and the results are distributed to all the visual processing regions of the cerebral cortex. Nevertheless, there are two main output streams from the primary visual cortex (Ungerleider and Mishkin, 1982; Milner and Goodale, 1995). One pathway receives both parvo- and magnocellular input and projects forwards and downwards to the inferotemporal cortex (IFT) in the lower part of the temporal lobe. It is mainly responsible for analysis and recognition of forms and patterns, so it is often known as the 'what' stream.

The other, 'where', stream is dominated by magnocellular input and passes forwards and upwards dorsally to the cortex in front of the occipital lobe, the posterior parietal cortex (PPC). The main function of this dorsal 'where' system is to help control movements guided by visual input. It is because the timing and motion of visual targets are crucial for guiding eye and limb movements that this visual guidance system is dominated by the visual magnocellular input from the retina.

In addition to this pathway via the cerebral cortex, the visual magnocellular system also provides the main visual input to brain stem structures controlling reflex eye and other movements. The most important of these is the superior colliculus. In birds and lower vertebrates this region is the main area for the visual guidance of all eye and limb movements. However, in higher mammals the superior colliculus is mainly concerned with automatic reflex movements, and the posterior parietal cortex, which receives both from the dorsal 'where' system and from the superior colliculus, has taken over the visual guidance of voluntary movements.

VISUAL MAGNOCELLULAR SYSTEM IN DYSLEXICS

The sensitivity of the magnocellular component of visual processing can be assessed psychophysically using stimuli which selectively stimulate it. Flickering lights, low intensity, low contrast, coarse (low-spatial-frequency) gratings and moving targets have all been shown to stimulate magnocellular neurones selectively. On the other hand, colour and fine detail at high contrast are signalled only by the parvocellular system (Merrigan and Maunsell, 1993). In many laboratories, therefore, the sensitivity of the two systems has been compared in normal readers and dyslexics. These tests have shown that the visual responses mediated by the magnocellular system are slightly, but consistently and significantly, impaired in dyslexics when compared with normal readers. This was shown originally by Lovegrove *et al.* (1980), and it is now clear that the sensitivity of most, but not all, dyslexics to flickering stimuli (Talcott *et al.*, 1998), to flickering low-contrast coarse gratings (Mason *et al.*, 1993) and to motion stimuli (Cornelissen *et al.*, 1995, 1998) is lower than in normal readers, whereas their responses to coloured and finely detailed stimuli have turned out to be no different from, sometimes even better than, normal.

Motion sensitivity is best assessed by finding the proportion of a field of dots moving around randomly (a 'random dot kinematogram') which have to move together 'coherently' for the subject to see them moving as a cloud rather than independently in random directions. Both adult and child dyslexics have been shown to be significantly less sensitive than normal readers to such visual motion (Cornelissen *et al.*, 1995, 1998). Indeed, subjects' visual motion sensitivity predicts their reading ability, and this is true of good as well as poor readers (Witton *et al.*, 1998; Talcott *et al.*, 1998). Thus skilled readers tend to have high visual motion sensitivity, whilst poor readers have low visual motion sensitivity. This correlation applies particularly for irregularly spelt words, because reading these successfully requires accurate processing of their visual form. These relationships suggest, but by no means prove, that motion sensitivity, hence magnocellular sensitivity, controls in some way the visual processing required for reading. These results have recently been confirmed by Demb *et al.* (1998) using slightly different techniques. Many researchers have also recorded dyslexics' visual evoked potential (VEP) responses to flickering low-contrast stimuli which selectively excite magnocells, and they have confirmed that dyslexics' responses tend to be delayed compared with normal readers' (Livingstone *et al.*, 1991; Maddock, Richardson and Stein, 1992; but see Johannes, Kussmaul and Mangun, 1996). Likewise, Eden *et al.* (1996b) and Demb, Boynton and Heeger (1997) have found, using functional magnetic resonance imaging, that dyslexics show reduced activation of visual area V5/MT, the visual motion area that is dominated by magnocellular input, in response to moving stimuli.

As mentioned earlier, Galaburda and colleagues compared the magnocells in the deep layers of the visual thalamic nucleus (LGN) in dyslexic brains with those in normals and showed that the dyslexics' magnocells were significantly smaller and more disordered (Livingstone *et al.*, 1991). Thus the evidence that many dyslexics do indeed show abnormal development of the visual magnocellular system is now very strong.

However, it is not immediately obvious why impaired visual magnocellular function should have any effect on reading (Hulme, 1988). Print does not normally flicker or move around and its contrast is usually high. Hence the impaired contrast sensitivity of the dyslexic's magnocellular system is unlikely to be the direct cause of his/her reading difficulties. However, when reading, the eyes have to move across the text.

Only the optical centre of the eye has high enough visual acuity to discriminate the fine detail that defines each letter in small print. Therefore, when the eye is centred on a letter, only about five letters in advance and three letters behind can be seen clearly. When reading, therefore, a series of rapid (30 ms) rightwards eye movements (saccades) have to be made along the line of print, between relatively lengthy (250 ms) fixations during which individual words are identified. At the end of each line the eyes return leftwards to the beginning of the next line by making a larger return saccade.

It is probable that the magnocellular system plays a major role in controlling these eye movements. The magnocellular system dominates the visual projections to the posterior parietal cortex, which is the part of the cerebral cortex responsible for visually guided voluntary eye movements, and to the superior colliculus, which is responsible for automatic reflex eye movements. Thus the magnocellular system guides reading saccades on to their target and helps them to maintain stable fixation on each word being read before the next saccade is made. In fact, the eyes spend 90% of the time fixated on a word rather than moving from one to the next, so achieving stable fixation between saccades is an especially important function of the magnocellular system for accurate reading (Stein, 1991).

Stability of the Visual World

The eyes move all the time when we look at things, which means that we view the world in a series of snapshots taken from the many different angles traced out by our saccadic eye movements. It is not easy to explain how, despite these movements, our perception of the visual world remains so stable and stationary (Helmholtz, 1962). If a camera is panned in the same way as our eyes do, everything becomes blurred during the movement. Furthermore, each new camera angle puts targets in different positions on the film. Yet these problems do not trouble us.

The reason why our vision is not thoroughly blurred during saccades is because visual processing is strongly inhibited at this time, so that we actually see very little during them. This mechanism is known as saccadic suppression (Cambell and Wurtz, 1978; Burr, Morrone and Ross, 1994). However, the reason why the position of objects does not seem to move between one saccadic snapshot and the next has proved much more difficult to understand. It seems that there may be no special mechanism. Instead, our mind's eye assumes that everything remains in the same place during an eye movement unless there is strong evidence to the contrary. We tend not to notice changes in the visual scene if they occur during a saccade (saccadic blink). Even if one target is replaced by an entirely different one or moved over a large distance whilst a saccade is in flight, we often do not notice it at all (Rayner and Pollatsek, 1992). Our visual world remains stable, simply because it is much rarer for things to move in the outside world than for our eyes to move. Thus we need very strong cues to convince us that anything has moved. Normally we know when we move our eyes, and so the resulting movement of images across the retina can be safely ignored, because we know that we moved our eyes; and it is usually safe to assume that nothing in the world has moved. However, if our eyes are moved by an external agent without our ocular motor system intending it, the whole world does appear to move. For example, if one presses on the side of the eye to move it slightly, then the world does appear to move around.

The problem of knowing when the eyes rather than objects in the outside world have moved is considerably complicated by the existence of vergence eye movements.

When close targets are inspected, as when reading, the eyes are not parallel; instead, they need to converge on the letter fixated. These convergence eye movements are the ones that have evolved most recently and are the ones that are the most vulnerable to drugs and disease (Carpenter, 1988). This is why large quantities of alcohol cause double vision; the convergence system breaks down and the eyes diverge uncontrollably; but perception is not informed about what has happened, so fusion of the two images provided by the eyes cannot be maintained, and fluctuating double vision ensues. Again it is the magnocellular component of visual processing which seems to play the main part in stabilizing binocular vergence eye movements, hence allowing us to maintain stable fixation (Mowforth, Mayhew and Frisby, 1981).

To summarize so far the role of the magnocellular system in normal visual processing: it is responsible for timing events in the visual world; hence it plays a particularly important role in the perception of flicker and movement and in the control of eye movements. During reading saccades, magnocellular activation suppresses the blur of images streaming across the retina that would otherwise be seen. Moreover, the magnocells play an important part in helping to keep the two eyes fixed steadily in convergence on each word.

Dyslexics' Binocular Stability

Since dyslexics have impaired magnocellular sensitivity, this might lead to unstable eye control and hence they might experience unstable visual perceptions which would cause visual reading problems. Thus it is not surprising that many studies have shown that dyslexics often do show unstable visual fixation (Stein and Fowler, 1980, 1993; Eden *et al.*, 1994; Evans, Drasdo and Richards, 1994). Fortunately, as we have seen, their magnocellular impairment is only slight, so it does not cause major visual defects such as motion blindness, visual agnosia, squint or nystagmus. If their vision were very severely affected, they would of course be easily diagnosed using the standard clinical tests that are employed by ophthalmologists, whereas actually most dyslexics' eye control is clinically normal (Goulandris *et al.*, 1998). Nevertheless, if we use sufficiently sensitive tests, we can show that the eye movement control of many dyslexics is mildly impaired. One simple way to show this is to record their eye movement when they are asked to hold fixation on a small target, such as a letter, for a few seconds. Under these conditions the eyes of many dyslexics 'hunt' around the target much more than do normals', rather like a badly adjusted feedback control system (Stein, Riddell and Fowler, 1987; Eden *et al.*, 1994). Likewise, their convergence system is more unstable and limited in range than that of normal readers (Riddell, Fowler and Stein, 1987, 1988; Stein, Riddell and Fowler, 1988).

As might be expected, other kinds of eye movement which depend on the magnocellular system are also impaired in many dyslexics compared with normals. The pursuit eye movements that are used to track a moving target are in many ways similar to fixation eye movements. Both are designed to keep a target on the high-acuity optical axis of the eye; hence fixation can to some extent be considered a special case of smooth pursuit, and the same cortical centres have been shown to be engaged in both types of eye movement. Thus we and others have found that many dyslexics cannot pursue moving targets as smoothly as normals (Adler-Grinberg and Stark, 1978; Griffin *et al.*, 1998). They lag behind the target and then have to make many more catch-up saccades; these are known as saccadic intrusions. Likewise, the

binocular vergence control of dyslexics is significantly less stable than that of normal children (Stein and Fowler, 1980, 1993; Griffen *et al.*, 1998).

Since stable binocular control is essential for stable visual perception during reading fixations, many dyslexics with unstable binocular control have symptoms which seem to be a consequence of this unstable perception; they complain that words and letters seem to move around on the page, particularly in and out of the plane of the paper, and that letters appear to merge and cross over each other (Cornelissen *et al.*, 1991). These are exactly the symptoms one would expect if their two eyes tended to move around independently and unintentionally, causing an unstable visual world and fluctuating double vision. Clearly such visual confusion would make reading difficult. It is particularly damaging when a child reaches the phonological stage of learning to read, because he/she then has to inspect each letter separately in order to match it with the sounds that he/she is also just learning. Thus, if the letters are perceptually unstable, merging and changing places, it is highly confusing. It is not surprising, therefore, that among both normal and dyslexic children those with unstable binocular control are significantly worse readers than their age-matched peers with good control (Stein and Fowler, 1980; Stein, Riddell and Fowler, 1986).

Therefore children with binocular instability tend to make visual reading errors. When they attempt to read unfamiliar words, they sound out the confused and overlapping letters and words that their unstable 'mind's eye' presents them with (Cornelissen *et al.*, 1991, 1998). Naturally this tends to produce bizarre nonsense words. In many their skill at phonological segmentation is somewhat better than their visual analysis. Although this may help them with regularly spelt words, phonological skill is less useful for reading irregularly spelt ones, because the pronunciation of these cannot be obtained solely by applying grapheme to phoneme correspondence rules, but depends mostly on remembering their visual form (Olson *et al.*, 1989). Accordingly, when attempting to spell irregularly spelt words, visual dyslexics tend to misspell them by trying to spell them phonetically; the odd results, such as spelling 'experience' as 'egspiriens', are typical phonological regularization errors (Cornelissen *et al.*, 1994). These errors probably arise because these dyslexics' visual confusions prevent them successfully memorizing the proper visual forms of the words.

Of course, the visual instability which many dyslexics experience does not affect their reading alone; it impedes their ability to accurately localize and sequence all small targets. Thus it has been found that dyslexics with unstable binocular control are much less accurate at judging the exact location of small dots within a frame (Riddell, Fowler and Stein, 1990) or deciding whether two lines are oriented at the same angle (Eden *et al.*, 1996a), in determining the sequence of small drawings of objects such as fruits, or in counting the number of dots presented in temporal sequence (Eden *et al.*, 1995b). All these skills draw on the signals provided by the magnocellular system.

Clearly also, if we could alleviate these children's unstable vision, we might be able to help them to learn to read better. One very simple way of doing this is to increase the size of print and the separation of letters. This reduces the chances of their uncontrolled eye movements merging or transposing letters, and Cornelissen *et al.* (1991) showed that it assists the reading of those whose main problems are visual.

Reading with One Eye

Another way of helping to stabilize visual perception is to use only one eye for reading. This often helps children with unstable binocular fixation to reduce their

visual confusions, because it eliminates fluctuating double vision, which is one potent cause of their tendency to merge and transpose letters (Cornelissen *et al.*, 1992). If 7–9-year-old children with unstable binocular control are given occlusion of the left eye for reading and writing for a few months, their binocular control often improves permanently, and this helps them to learn to read. Rate of reading more than doubled in children whose binocular stability was improved by this simple procedure (Stein and Fowler, 1981, 1982, 1985).

This treatment is highly controversial. Nevertheless, four placebo-controlled trials (Stein and Fowler, 1981, 1985; Stein *et al.* 1997; Masters, 1988) have confirmed that monocular occlusion can help children with unstable binocular control to improve their fixation, and thereby help them to learn to read. However, it is only effective at the critical early age of 6–9 years old. Temporarily confining visual input to one eye seems to enable that eye's retina to guide its extraocular muscles to maintain it accurately pointing at its target without interference from the other eye's competing signals; this is known as utricular control (Ogle, 1962). Once one eye has 'learnt' to home in on the target and fixate it steadily, then it seems to be easier for the other eye to follow suit. Paradoxically, therefore, monocular occlusion can improve binocular fixation stability, after which the patch is no longer necessary. If it does so, the children achieve rapid reading progress.

AUDITORY TRANSIENT FUNCTION

A separate system of magnocellular neurones, such as that found in the visual pathways, has not been described in the auditory system. Nevertheless, anatomically magnocellular divisions of each of the auditory subcortical relay nuclei are well known. Galaburda, Menard and Rosen (1994) have shown that the magnocellular division of the auditory relay in the thalamus, the medial geniculate nucleus, is disorganized, particularly on the left side, in dyslexic brains *post mortem*. Moreover, it has recently become clear that auditory magnocellular neurones are specialized for tracking rapid frequency and amplitude changes in acoustic signals (Trussell, 1998). Thus it appears that auditory 'magnocells' may play a role in temporal analysis of sounds that is analogous to that in the visual system. Accurate tracking of acoustic amplitude and frequency transients is essential for identifying the phonological cues that characterize speech. Thus it is not implausible to suggest that dyslexics' problems with phonological analysis may be fundamentally caused by a defect in their auditory 'magnocellular system'.

As we have seen, dyslexics' phonological impairment takes the form of not being able to split the sounds of words into their constituent phonemes in order to match them with their alphabetic representation. This skill can be assessed by means of phonological tests such as the ability to produce spoonerisms, to detect rhymes, or in the game known as 'Pig Latin'. In this game the initial sound of a word is taken off the beginning and put on the end of a word with -ay after it, so that the word 'pig' becomes 'igpay'. Children in the know hugely enjoy playing this game because they can communicate with each other in a language that is completely mystifying to those who have not learnt it. Dyslexic children can seldom take part because they are so bad at phonemic segmentation. This has been shown formally by Olson *et al.* (1989) and also by Eden *et al.* (1995a). Dyslexics may be bad at this game because they have slightly impaired auditory sequencing skills as a consequence of impaired development of magnocellular neurones in their auditory as well as their visual systems.

Paula Tallal found that children with impaired acquisition of speech, developmental dysphasia, may have specific difficulty with rapidly judging the order in which complex sounds are presented, and she suggested that this might explain their problems with learning to speak properly (Tallal and Piercy, 1973). She has also speculated that the same might be true of developmental dyslexics (Tallal, 1980). However, her work has proved highly controversial, with failures to replicate and disagreement over whether her test really assesses auditory transient processing (of rapid changes within an acoustic signal) or whether it tests the ability to judge the order of rapidly presented sounds (temporal order judgements—Studdert Kennedy and Mody, 1995). The former might correlate with the rapid temporal processing required for phonological identification of phonemic features, but the latter might not.

However, the sensitivity of dyslexics to auditory transients can be tested using simpler stimuli that unequivocally require transient processing, i.e. stimuli that selectively stimulate the large auditory processing neurones that are analogous to visual magnocells. Many dyslexics show reduced sensitivity to changes in amplitude (AM) or frequency (FM) of simple auditory tones (Stein and McAnally, 1996; McAnally and Stein, 1996). What is even more important is that sensitivity to these auditory transient stimuli correlates very highly with subjects' phonological ability, and this is true of both normal readers and dyslexics (Witton *et al.*, 1998). As with visual motion sensitivity, this relationship suggests that phonological processing ability depends on auditory temporal processing ability in some way. Since phonological analysis depends on accurately detecting rapid changes in amplitude and frequency, the simplest hypothesis is that children's basic sensitivity to dynamic auditory stimuli determines their phonological segmentation skills.

Unfortunately, however, most dyslexics have both visual and auditory/phonological problems. Perhaps 20% of children have predominantly visual and 20% have mainly phonological difficulties, but the remaining 60% have both visual and phonological processing abnormalities. When subjects' sensitivity to auditory transient signals is compared with their performance in visual motion tests, the correlation between the two is greater than 0.5. This is true for both dyslexics and normal readers; that is, subjects are likely to be good or bad at both, and their sensitivity to both correlates with their literacy skills (Witton *et al.*, 1998). This suggests that a common influence underlies both, supporting the simple hypothesis that children's reading ability, whether high or low, may be dependent on the degree of development of their auditory and visual magnocellular systems. Taken together with the evidence that many dyslexics also have slightly impaired motor skills, this suggests that they can have mild generalized damage of all their magnocellular neurones. The variability in the involvement of visual, auditory and motor systems indicates that this damage is patchy and individually idiosyncratic.

Although it is probable that this neuronal damage occurs *in utero* and therefore precedes any reading problems, the direction of causality may not be exclusively from neurone to reading. Morais and Bertelson (1979) have produced convincing evidence in adult illiterates that the very process of learning to read improves subjects' phonological analysis abilities, and Castro-Caldes *et al.* (1998) have shown that this changes the way in which phonology is represented in their brains. These changes are likely to be even more pronounced in young children, perhaps even influencing neuronal cell size. These facts offer hope that targeting sensory training to children's particular weaknesses can help them to compensate for their impaired magnocellular development. Nevertheless, even when dyslexics' reduced exposure to print is

controlled for by comparing them with younger normal readers with the same reading age, in reading age match designs, dyslexics usually do worse. This suggests that they have a persistent deficit so that their reduced performance persists into adulthood.

CONCLUSIONS

Thus it is probable that developmental dyslexia results from patchily abnormal development of magnocellular neurones throughout the whole brain. The visual magnocellular system together with its auditory and motor equivalents can all be abnormal in dyslexics (Galaburda and Livingstone, 1993; Stein and Walsh, 1997; Rae *et al.*, 1998). These impairments could cause dyslexics' visual instability, reduced phonological skills and their plethora of motor problems, including unco-ordinated handwriting.

Taken together with the evidence for dyslexia's genetic basis, one attractive speculation is that impaired development of magnocellular neurones in the brain, whether sensory, association or motor, is due to a congenital attack by antibodies sensitive to their distinctive surface markers, and that this deficiency leads to failure to develop normal neural temporal processing. This hypothesis is circumstantially supported by the evidence for linkage to genes at the heart of the major histocompatibility complex (MHC) on chromosome 6 (Cardon *et al.*, 1994; Grigorenko *et al.*, 1997; Fisher *et al.*, 1999) and other possibly immunologically related sites on chromosomes 1 and 15. These associations support the general idea that fundamentally dyslexia results from some kind of adverse immunological influence on the development of magnocells in the nervous system. This hypothesis is highly speculative of course, but it potentially links together all the main findings.

The 'Gift' of Dyslexia

However, in closing, we must emphasize that all the neurological problems that dyslexics face are mild. Dyslexia would not be so common if it caused serious perceptual impairments without compensating advantages to having such unusual nervous systems. Dyslexics are by no means damned to eternal incompetence. Indeed, there are many, many examples of the talents that dyslexics develop in other areas of life. A hundred years ago their difficulties with reading would have been hardly noticed. Even now, if their problems are recognized early enough and their education is directed towards their strengths rather than their weaknesses, the dyslexic brain need not be too disabling.

A very good example of this is the history of the sculptor Auguste Rodin, brought to our attention by that great Swedish philanthropist and supporter of interdisciplinary research in dyslexia, Per Udden. Rodin's father had the great good sense to recognize early in his son's dismal educational progress that he would never be an academic, but that he was clearly very talented artistically. Therefore he scrapped his plans for his son's academic education; instead, he encouraged Auguste to develop his artistic talent, which he did with phenomenal success, even though to his dying day the sculptor needed an amanuensis to help him write his letters.

There are a large number of talented people in history whom we now believe were probably dyslexic, e.g. Albert Einstein, Winston Churchill, Alexander the Great,

Thomas Edison, to name but a few. Their success emphasizes that difficulty with learning to read is not a wholly tragic life sentence but is often accompanied by great talents. Furthermore, there is now much evidence from animal studies that the brain is far more malleable than once used to be thought, particularly in youth (Merzenich *et al.*, 1996). Therefore, if we can discover precisely how dyslexics' mild problems arise, and learn to recognize them early enough, there is every hope that we will be able to obviate them, especially by encouraging development of the talents whose benefits have preserved this interesting neuronal magnocellular abnormality in the human genome.

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