

The neurobiology of reading difficulties

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INTRODUCTION

Despite the high incidence of reading difficulties, which may trouble up to 10% of all children, there is no agreement about their causes. This controversy is encapsulated in arguments about whether extreme reading difficulties should be noncommittally called 'specific reading difficulty' or, using the neurological description 'developmental dyslexia'. Specific reading difficulty is preferred by those who believe reading problems are purely linguistic, whereas developmental dyslexia implies commitment to the idea that reading difficulties result from impaired neurological development.

In our laboratory we take the latter view. We study the neurophysiological processes which underlie reading; hence we espouse the neurological model. Therefore, in this chapter I will discuss how normal reading depends on the quality of its sensory input. It requires both a highly sensitive visual magnocellular system to acquire good orthographic skills, and a sensitive auditory transient system to parse the phonological structure of words. Then I will speculate about the genetic and immunological mechanisms that may be responsible for the wide variety of abnormalities that are seen in developmental dyslexics. My overall conclusion will be that reading difficulties are neither specific to reading nor exclusively linguistically based, but a consequence of mildly impaired development of a particular kind of neurone in the brain, magnocellular neurones, so that dyslexia has widespread manifestations which are not at all confined to reading.^{1,2} However they are best thought of as

individual differences between people rather than a consequence of neurological 'disease'.

NORMAL READING

Recent functional imaging studies have shown that reading involves both hemispheres of the brain, but the importance of the left is enhanced as the phonological or orthographic demands of the task are made harder.³ Silent reading engages mainly the posterior part of the left hemisphere, focusing on the left angular and supramarginal gyri. Reading out loud shifts activity forwards towards Broca's area in the frontal cortex, so that the whole of the temporo-frontal articulatory loop is engaged. Homologous areas in the right hemisphere are also activated to a lesser extent, but their role is enhanced only when the intonation or emotional content of speech is emphasized.

Activation of posterior more than anterior areas during silent reading emphasizes the importance of the visual input to reading. Vision is obviously important, and Morgan first described developmental dyslexia as 'word blindness'.⁴ However, its role in learning to read tends to be rather neglected nowadays because following the linguistic tradition most people believe that a poor reader's main problem is lack of phonological skill.^{5,6} But, in fact, familiar words are recognized entirely visually, without any requirement for phonological mediation. Likewise irregular words cannot be successfully sounded out: hence they must be read by the visual route.

It is true, on the other hand, that unfamiliar words (and all words are unfamiliar to beginning readers) have to be sounded out using the letter sound correspondences that have to be learned. This engages more anterior parts of the articulatory loop. Even if the letters are sounded out entirely mentally, in the 'mind's ear', the whole articulatory loop is engaged.

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Thus there are two reading mechanisms that are at least conceptually separable: the whole word semantic route which draws heavily on the visual system, and the phonological route which draws heavily on the auditory articulatory system.^{7,8} These two routes therefore depend upon the processing powers of the visual and auditory systems, and it is these that I shall now deal with.

THE VISUAL MAGNOCELLULAR SYSTEM

The ganglion cells whose axons carry visual signals from the retina to the rest of the brain can be divided into two main types: 90% are known as parvo cells from their small cell bodies and restricted dendritic spread.^{9,10} These are responsible for colour and fine detail. The remaining 10% are large magno cells. Their dendrites cover a retinal area up to 500 times that of parvo cells. They are not only larger, but they are more heavily myelinated which means that their conduction velocities and membrane dynamics are much higher. Hence although their spatial resolution is coarser and they do not support colour vision, they respond faster and their signals arrive at the brain 10–20 msec earlier than that of parvo cells. Thus they are important for timing events in the visual world and for detecting changes with time, such as those caused by visual motion, but they do not signal colour or fine detail.¹¹

On arrival at the main visual relay nucleus in the thalamus, the lateral geniculate nucleus (LGN), magno axons are separate from parvo and pass into the magnocellular layers of the LGN. The magnocellular LGN cells then project to layer IV C alpha of the primary visual cortex, whereas the parvocellular layers project to layer IV C beta. From there, parvo and magno streams intermingle, but the output of the visual system splits into two main streams, the 'what' and 'where' pathways.¹² The ventrolateral stream projects towards the inferotemporal cortex and receives roughly equal inputs from magno and parvo pathways. It is called the 'what' pathway because it is specialized for identifying the shape, pattern and colour of objects, and therefore for identifying what they are.

In contrast, the dorsomedial pathway passes towards the visual motion area (V5/MT) in the Superior Temporal sulcus, and thence to the posterior parietal cortex. It is termed the 'where' pathway because it is specialized for detecting the current position and motion of targets, and for directing attention and movements towards them.¹³ It is therefore dominated by input from the visual magnocellular system. As befits its function in helping the visual guidance of movements, this system projects onwards to all the areas involved in the guidance of eye movements:

the frontal eye fields, the superior colliculus and the cerebellum.

DYSLEXICS' VISUAL MAGNOCELLULAR DEFICITS

One advantage of the separation of the visual magno- and parvocellular systems is that their sensitivity can be assessed psychophysically in normal subjects using stimuli that selectively activate one or the other system. Spatial contrast and temporal flicker sensitivity are limited mainly by the performance of the peripheral visual system up to the level of the visual cortex. Lovegrove et al. therefore used sinusoidal gratings to show that the contrast sensitivity of dyslexics was impaired compared with controls, particularly at low spatial and high temporal frequencies.¹⁴ Thus it was he who first suggested that dyslexics may have a selective impairment of the magnocellular system. Under certain circumstances it was found that dyslexics' contrast sensitivity at the high spatial frequencies that are mediated by the parvocellular system was actually higher than in controls.¹⁵ Likewise Martin and Lovegrove showed that dyslexics' flicker sensitivity tends to be lower than controls, and we have confirmed this.^{16,17} Again, this result suggests that dyslexics may have a specific magnocellular impairment.

However, these results have been hotly disputed,¹⁸ but see Stein et al.¹⁹ The impairment is slight and is only found in some two thirds of dyslexics. Most studies have involved small numbers, so that there have been many failures to replicate Lovegrove's results. Much larger numbers are needed together with screening dyslexics for those who have visual symptoms and therefore are most likely to have a significant magnocellular deficit.

Testing sensitivity to visual motion has proved much more reliable, however, because motion engages not only the peripheral visual system but also central processing stages up to at least area V5/MT. In monkeys it has been found that detecting coherent motion in a display of dots moving about randomly (random dot kinematograms-RDK) is a sensitive test for probing the magnocellular system.^{20,21} We have therefore developed a RDK test of motion sensitivity for use with adults and children. We present two panels of randomly moving dots side by side. In one of the panels, selected at random, a proportion of the dots is moved together 'coherently' so that they look like a cloud of snowflakes blown in the wind. The subject is asked in which panel the cloud appears to be moving. The proportion of dots that is moved together is then reduced until the subject can no longer tell on which side the dots are moving together. His threshold is then defined as the proportion of dots that have to move together for him to see the coherent motion correctly on 75% of occasions. Using this test we have found that in

children and adults with dyslexia, defined as those whose reading is more than two Standard Deviations behind that expected from their age and IQ, about 75% have worse motion sensitivity than controls matched for age and IQ.^{17,22,23} Our conclusion from psychophysical studies that many dyslexics have poor motion sensitivity has now been confirmed by a succession of functional imaging studies.^{24,25,26}

LGN ABNORMALITY IN DYSLEXIA

Lest anyone is still in doubt that many dyslexics have impaired development of the visual magnocellular system, the most direct evidence would be provided by examining the magnocellular layers of the relay nucleus of the peripheral visual system, the lateral geniculate nucleus (LGN) in the thalamus. Samuel Orton, a pioneer in the study of developmental dyslexia in the 1930s, persuaded some of his patients to donate their brains to medical science. These donations have now begun to materialize, and Albert Galaburda and colleagues have now studied some of these histologically. They have shown unequivocally that the magnocellular layers of the LGN are indeed disordered and the neurones some 30% smaller in area than in control brains.^{27,28} Such differences are known to arise during the early development of the brain, during the phase of rapid neuronal growth and migration during the fourth or fifth month of foetal development. One could not adduce stronger evidence than this that the visual magnocellular system fails to develop quite normally in dyslexics.

MAGNOCELLULAR FUNCTION PREDICTS ORTHOGRAPHIC SKILL

Since our hypothesis is that impaired visual magnocellular function impedes reading, it is incumbent on us to show this. Therefore we have been correlating magnocellular sensitivity with visual reading ability. In both children and adults, whether dyslexic, good readers or unselected primary school children, we have demonstrated that visual motion sensitivity correlates strongly with their reading ability.^{29,30} In particular, motion sensitivity predicts visual orthographic skill. We measure this using the Olsen pseudo-homophone test.³¹ In this test we present, on a computer screen, two words side by side that sound the same but have different spellings, i.e. 'rain' beside 'rane'. The subject is asked which is the correct spelling. Since the words sound the same, this task cannot be solved phonologically by sounding out the letters. The visual form or orthography of the word must be recalled correctly. Hence Joel Talcott found that the correlation between visual motion sensitivity and performance in this pseudo-homophone test is very strong.³⁰

This was true not only in dyslexics but across the whole range of reading abilities. Good spellers in this test had high motion sensitivity, whereas poor performers had low motion sensitivity. Likewise, we found that the correlation between subjects' motion sensitivity and their performance in Castle and Coltheart's test of the spelling of irregular words was very high, because success in this test also depends on a good visual memory of their spelling.⁸

In contrast, the correlation between subjects' visual motion sensitivity and tests of phonological skill, such as the ability to read nonsense words or to form Spoonerisms, is much lower. In fact, when we controlled statistically for the correlation that exists between subjects' phonological and orthographic abilities we found that motion sensitivity continued to account for a high proportion of the variance in orthography, but now of course independently of phonology. In other words, motion sensitivity accounts for children's orthographic skill independently of its relationship with their phonological skill, as you would expect if this basic visual function helps to determine how well the visual component of reading develops.

DYSLEXICS' BINOCULAR CONTROL

One problem that constantly bedevils the hypothesis that dyslexics have impaired magnocellular function is that people find it very difficult to understand how a system devoted to detecting visual motion could possibly be relevant to reading.³² After all, we don't usually have to track moving targets when reading; the page is usually kept stationary. In fact, the retinal images of print are not stationary, and many dyslexic children complain that letters seem to move around when they are trying to read, i.e. their visual world is highly unstable.³³ This is because during reading visual images are actually very far from being stationary on the retina, and dyslexics fail to compensate for this. The eyes remain fixated on individual words for only about 300 msec before saccading to the next. Even during the fixations, however, they are not totally stable but move around by up to 1 degree of visual angle – equivalent to 4 or 5 letters' worth. In normal readers, the visual magnocellular system detects such unintended motion of the letters over the retina, 'retinal slip', and this signal is used to help stabilize the eyes. For movements of less than 1° the magnocellular system further sharpens the image by enabling us to discount the motion. But it seems that many dyslexics fail to be able to stabilize their vision when reading, so that they tend to transpose letters when attempting to read.³⁴ We believe that their unstable visual perceptions are the result of the insensitivity of their visual magnocellular systems.

DYSLEXICS' BINOCULAR INSTABILITY

To test this hypothesis we measured how accurately dyslexics and controls can fixate small targets the size of letters at the normal reading distance of about 30 cm. We found that the amplitude of the unintended eye movements made during attempts to fixate for 3 s was indeed much greater in the dyslexics than in the controls.³⁵ Furthermore, these movements were different in the two eyes so that the degree of binocular convergence varied randomly from moment to moment. This would explain why so many dyslexic children complain not only that the letters move from side to side, but also slide over each other and in and out of the plane of the page.^{36,37} We found that the unstable binocular control of these dyslexics was also reflected in slower and less smooth vergence pursuit tracking of a target moving in depth, with a much greater tendency for vergence to break down to conjugate gaze, hence.^{38,39,40} The high incidence of binocular instability in dyslexics has also been confirmed by Evans et al.⁴¹

Thus it seems that an important way in which dyslexics' impaired magnocellular function may interfere with reading may be by causing binocular instability. Since these eye movements are unintended and uncontrolled, they may be misinterpreted as movements of the letters. Since this instability often causes the two eyes' lines of sight to cross over each other, the letters appear to move around, slide over each other, and change places. We reasoned that if this is so then simply blanking the vision of one eye should simplify the visual confusion and help these children to see the letters properly. This is exactly what we have found in three different trials.^{42,43,44} In children with binocular instability, occluding the left eye for reading and close work relieved their binocular perceptual confusion⁴⁵ and helped them to learn to read. The results were often dramatic and in our most recent double blind controlled trial of monocular occlusion in dyslexic children with binocular instability we were able to help those who received the occlusion almost to catch up with the reading age of their peers. In contrast, those who did not receive occlusion and who did not gain binocular stability remained lagging 2 years behind their chronological age. This progress is far greater than most remediation techniques achieve with dyslexics.

After 3 months, occlusion not only had the children's reading improved to this great extent, but also they could now fixate stably with their two eyes, so that they no longer needed to wear the patch. We believe that this gain of binocular stability results from the magnocellular signals from the seeing eye now successfully routing themselves to control the muscles of that eye. This is called utricular control, and it is crucial for the final stages of precise vergence control because it enables each

eye to home in accurately on a target so that both fixate properly on it.

So now we think we can explain how magnocellular function impacts on reading, and in particular helps to develop orthographic skill. Poor readers have slightly impaired development of their magnocellular neurones. As a consequence the dense magnocellular input that visuomotor centres in the PPC, superior colliculus and cerebellum receive is both delayed and smeared in time. In consequence, utricular control over the muscles controlling the eye that supplied the magnocellular input is less sharply focused in time and therefore less able to stabilise the eyes during fixation or during vergence pursuit. Therefore the eyes' lines of sight may cross over each other, hence the letters can appear to do so also. This is why these dyslexics tend to reverse the order of neighbouring letters and to reverse the order to letter features, thus confusing ds with bs and ps with qs. These are precisely the kinds of error that we find in dyslexics with unstable binocular control.

AUDITORY TRANSIENT SYSTEM

Only about two thirds of dyslexics have significant visual problems. The remaining third, together with about half of those with visual symptoms in addition, have another major cause of reading difficulties: inability to learn letter sound correspondences and the phonological structure of words. As described earlier, unfamiliar regular words can be read by producing the sounds that each letter stands for, 'sounding out' the letters, and then blending them together to identify the word. Acquisition of this phonological skill is perhaps the most important and most difficult component of learning to read.^{5,47, 48}

SOUND FREQUENCY MODULATION

The cues that distinguish the different letter sounds are changes in the frequency and amplitude of speech sounds. Thus the difference between /d/ and /b/ is that in /d/ the second and third formants rise in frequency in the first 40 ms, but in b they go down. Everything else is identical in the two sounds. Thus phonological analysis draws very heavily on the ability of the auditory system to track frequency and amplitude changes, acoustic transients, accurately. Such processing can be assessed, as in the visual system, using much simpler transients than those found in speech, for example by measuring subjects' sensitivity to sinusoidal frequency or amplitude modulations of a single sinusoidal tone. Therefore we have been measuring people's sensitivity to frequency and amplitude modulations of simple tones to see whether this relates to their phonological abilities.

Ken McAnally and I,⁴⁹ and more recently Caroline Witton et al.,²⁹ measured subjects' sensitivity to frequency modulations by asking subjects to listen to frequency modulated (warbling) tones and then adjusting the amount of warble, the modulation depth, until they could no longer distinguish this from a pure tone. Using 2, 20 and 40 Hz frequency modulations of a 500 Hz or 1000 Hz carrier stimulus we were able to show that groups of adult and child dyslexics were significantly worse than matched controls at hearing these changes in frequency, i.e. they required significantly greater modulation depths. Again, however, there were some dyslexics who were just as good as controls at this task.

PHONOLOGICAL SKILL IS PREDICTED BY FM SENSITIVITY

Since we expected this stimulus to mimic to some extent the phonological cues that enable people to distinguish letter sounds, we needed to correlate their FM sensitivity with their phonological ability. This can best be tested by getting subjects to read nonsense words: nonwords, such as 'tegwop'. This word means nothing, yet it can be read by applying the phonological rules properly. However, you are not assisted by the word having any meaning, so that nonsense word reading has very little 'top down' input. Performing this task depends mainly on rapid and accurate application of letter/sound conversions.⁴⁸

We therefore measured the number of errors and the time our subjects' took to read a list of nonwords and correlated this with their FM sensitivity. This showed that the relationship was very strong in both children and adults, so that in a class of 30 normal 10 year olds 2 Hz FM sensitivity accounted for over 50% of their variance in nonword reading.^{30,50} When we controlled for the variance shared with orthographic ability to allow for the fact that the subjects had to visualize the nonwords in order to read them, FM sensitivity still accounted for a substantial proportion of nonword reading variance independently of orthography. Thus high auditory FM sensitivity seems to enable children to develop strong phonological skills, whereas poor FM sensitivity prevents this, in the same way as high visual motion sensitivity promoted orthographic skill and low sensitivity prevented it.

AMPLITUDE MODULATION (AM)

Peter Menell⁵¹ and Caroline Witton (submitted) have also demonstrated that sensitivity to amplitude modulations is important for developing phonological skill. Caroline measured sensitivity to 20 Hz amplitude modulations and found not only that dyslexics are significantly less sensitive to these than controls, but that in both dyslexics

and normal readers covering the whole range of reading ability AM sensitivity also helps to account for their nonword performance. What is particularly interesting is that the auditory system processes 2 Hz FM and 20 Hz AM stimuli rather differently. As a result, subjects perform differently in the two tests; their scores do not correlate with each other. Therefore it was exciting to find that 2 Hz FM and 20 Hz AM accounted for independent components of the subjects' variance in nonword reading. In other words, our FM and AM tests probably relate to the development of different aspects of phonological skill. It was found that 2Hz FM is quite slow and represents the frequency of syllable production and intonation rather than individual phonemes, whereas 20 Hz is clearly in the range of the transients that enable us to distinguish between phonemes.

AUDITORY MAGNOCELLS?

While the magnocells in the visual system form a clearly separate entity responsible for processing visual transients, there is no such anatomically defined magnocellular division in the auditory system. We cannot so simply attribute the FM and AM deficits that we have found in poor readers to impaired development of an auditory magnocellular system. Nevertheless, there are large celled magnocellular divisions of all the auditory relay nuclei, and good evidence that these are specially responsible for following changes in frequency or amplitude of acoustic signals with time.⁵² Thus there is probably an auditory magnocellular system whose function is to process auditory transients such as those important for phonological analysis, but one must bear in mind that it is not an anatomically separate system as is the case for the peripheral part of the visual magnocellular system.

This auditory analogy with the visual magnocellular system is given a further boost from further neuropathological studies of Galaburda and colleagues⁵³ in dyslexic brains post mortem. They found that, as in the LGN, the magnocellular division of the medial geniculate nucleus (MGN), which is the auditory relay nucleus of the thalamus, was disordered, particularly on the left, the side that projects to the language hemisphere.

AETIOLOGY OF DYSLEXICS' LOW SENSITIVITY TO SENSORY TRANSIENTS

In summary, dyslexics seem to have impaired processing of both visual and auditory transients, because they tend to have impaired development of magnocellular neurones in both the visual and auditory systems. These impairments help to explain their failure to develop adequate orthographic and phonological skills, respectively. Of course, the question arises why they fail to

develop good sensitivity to sensory transients. Those who argue that dyslexia is a specifically linguistic problem suggest that the low level auditory and visual deficits are purely epiphenomena, not on the main causal chain leading to reading problems.⁵⁴ But this argument seems highly implausible given the striking correlations between the sensory deficits and the cognitive skills required for reading. Also our finding that setting right the binocular instability associated with the visual magnocellular deficit very greatly improves dyslexics' reading strongly suggests a causal connection,¹⁹ as does Merzenich et al.'s⁵⁵ finding that auditory training using artificially slowed frequency transients can improve children's ability to hear them and thus improves their ability to make phonological discriminations.

Much more likely is the possibility that there is a difference in the development of magnocellular systems in dyslexics' brains that blunts their transient sensitivity and thus compromises their ability to develop orthographic and phonological skills. There is now a great deal of evidence for this view and a mechanism for the development of these differences can even be sketched out. The evidence is genetic, immunological and neurological.

GENETIC LINKAGE

It is well known that dyslexia tends to run in families, and careful studies have shown that it has a heritability of c. 60%, i.e. 60% of the variance in people's reading abilities can be attributed to the alleles they inherit.⁵⁶ This is a high value that is similar of the heritability of height and other cognitive abilities such as verbal ability or general intelligence, but much higher than the heritability of diabetes or heart attacks. We have taken advantage of the large number of dyslexics we have seen over the years to collect, so far, over 200 families in which two or more children have severe reading problems. We retest the children in standardized orthographic and phonological tests and take blood for DNA analysis from the children and their parents. In collaboration with Prof. Tony Monaco and Simon Fisher we then look for linkage of these scores treated as quantitative traits (QTLs) to chromosomal markers. We are currently in the process of carrying out a screen of the whole human genome to find which sites link to reading ability, but we have already confirmed⁵⁷ the findings of Cardon et al.⁵⁸ and Grigorenko et al.⁵⁹ from smaller studies carried out in Colorado and Connecticut respectively that both phonological and orthographic impairments link to a site on the short arm of chromosome 6 near the Tumour Necrosis Factor and Major Histocompatibility Complex (MHC) immunological sites. This linkage may turn out to be very significant, as there is accumulating evidence that an immunological

mechanism contributes to the impaired development of magnocells.

MAGNO SURFACE MARKERS

Visual magnocells express specific surface antigens, such as one known as Cat 301, and these same antigens are found on large cells all over the nervous system.⁶⁰ For example, they are also expressed in the magnocellular nuclei of the auditory system, on the large cells in the dorsal column somaesthetic nuclei, the hippocampus and on other magno cells throughout the brain. One site of special interest is the cerebellum. This structure is the brain's main timing device, the brain's autopilot, and it receives dense input from all the magno systems throughout the brain.⁶¹ Thus magnocells probably represent a distinct cerebral system with a separate developmental lineage, common surface antigens and the heavy myelination and rapid membrane dynamics that confer upon them their enhanced sensitivity to temporal transients. But their common surface antigens make them all vulnerable to antibody attack in auto-immune individuals.

GENERALISED MAGNOCELLULAR SYSTEM

Therefore we were not surprised to find that there is a strong tendency, especially in normal readers, for auditory and visual transient sensitivity to be similar in individuals.²⁹ This again suggests that the development of auditory and visual magnocells is indeed under some sort of common control. Recently Corriveau and Shatz⁶² have found that Class 1 MHC molecules play an important part in the development of visual magnocellular neurones in the cat LGN and also in the hippocampus. Since Hockfield and Sur's work⁶⁰ suggests that all magnocells throughout the brain derive from a common developmental lineage, it is reasonable to speculate that their development is regulated by the MHC system. Hence our finding that reading disability is genetically linked to a site close to the MHC complex on chromosome 6 takes on added significance, as does the evidence that dyslexics and their families very often demonstrate mildly abnormal immunological responses. They have a higher incidence of asthma, eczema, hayfever and other immune conditions than controls,⁶³ and families with serious autoimmune conditions such as systemic lupus erythmatosus (SLE) often have dyslexics among them.

UNSATURATED FATTY ACID SUPPLEMENTS

The reports that the symptoms of many dyslexics can be improved by food supplements of highly unsaturated

fatty acids (HUFAs), such as Efalex, can be fitted into this schema. Magnocellular function is dependent upon the rapid dynamics of their membrane ionic channels. These in turn depend greatly upon their high content of unsaturated fatty acids, because they are highly flexible and can accommodate the rapid conformational changes that magnocellular membrane channels need to make. The turnover of membrane fatty acids is under the control of phospholipases, in particular PLA2. But the activity of this enzyme is increased by IGG antibody, since immune reactions all need to mobilize HUFAs from cell membranes in order to provide precursors for the cytokines that are required for effective cellular responses to foreign material. Modern diets can contain dangerously low quantities of HUFAs. Therefore, under these conditions relative HUFA deficiency can arise, and magnocellular function may be particularly compromised. Hence supplementing these subjects' diets with HUFAs can often relieve their symptoms.

SUMMARY

As Miles⁶⁴ first emphasized, dyslexics' literacy problems are really just part of a much more generalized syndrome caused by impaired development of magnocells throughout the brain. We speculate that this results from genetically mediated, disordered immunological regulation of their development in the foetus, exacerbated by modern diets low in unsaturated fatty acids. This would explain not only the auditory and visual transient deficits that directly undermine the development of phonological and orthographic skills, but also the plethora of other problems that beset dyslexics. Their legendary clumsiness, impaired coordination and balance can now be attributed to abnormal cerebellar function, for which there is now a great deal of evidence.^{65,66,67} Their reduced sequencing and timing ability may be attributed partly to impaired cerebellar function and partly, perhaps, to reduced magnocellular input to the left hemisphere, which normally receives more than the right. This, in turn, would lead to impaired hemispheric specialization that would explain their relative failure to establish fixed hemispheric dominance, their tendency to problems with telling left from right, their mixed handedness, unfixed eye dominance and many other cognitive symptoms. In short, immunologically mediated mild magnocellular deficiency could explain all the wide variety of problems that dyslexics face.

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