



# Visual Dyslexia

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## Abstract

**Purpose** To revive the idea which was previously generally accepted, that learning to sequence written text visually is the crucial first step in learning to read. But in the last few years the phonological theory of dyslexia (PT) has become dominant. It asserts that DD is due to a psycho-linguistic deficit causing difficulty with grasping the concept of phonemes. This ignores a century of research showing that the development of a network of visual magnocellular neurons (M- cells) that signal the timing of visual events precedes and is essential for the development of phonological awareness.

**Recent Findings** We review the cerebral structures with which the visual M- system is associated in reading alphabetic and other scripts and show in each case how their development is impaired in DD. The simplest way to improve M- cell function involves viewing text through yellow or blue filters; we discuss how these may work. Even simpler is improving children's nutrition; and we discuss some aspects of this. The genes which are associated with the M- system also play an important role in regulating the immune system; hence we discuss how DD is associated with autoimmune conditions such as asthma and eczema and disordered control of inflammation.

**Summary** There is now a very great weight of evidence demonstrating that visual sequencing of text mediated by the visual magnocellular timing system is the first essential for learning to read, and that impaired development of this system is a major cause of Developmental Dyslexia.

**Keywords** Reading · Visual magnocellular · Visual timing · Sequencing · Major histocompatibility complex · Colour filters · Omega 3s

## Introduction

In recent years the concept of Developmental Dyslexia (DD) has become highly controversial and contentious [1]. From being a respected neuroscience based neurological diagnosis in the 20th C., it has been so criticised in the 21st C that many educationalists now believe that it is not a useful concept at all, and that it should be abandoned altogether. This is because they now regard it as a purely psycho-linguistic deficit. They argue that the crucial disorder is failure to grasp the phonological principle, namely that in order to learn to read, sequences of letters in a written word must be translated into the sequence of sounds, phonemes, that they represent [2]. Of course, that translation is an important component of learning to read. But it is by no means the only pathway; and

it is far less relevant when reading and writing logographic scripts. Here we will argue that visual processing comes first for all scripts.

The phonological theory proposes that phonological awareness is crucial for reading, but it pays no attention to how this usually develops; nor what physiological mechanisms must underlie it. And abandoning the neurological medical model ignores a century of valuable research that shows unequivocally that learning to sequence written characters visually is, in fact, primary. In addition, it ignores how visual analysis underlies the acquisition of phonological awareness, among other skills [3]. It has also rendered the term Developmental Dyslexia almost useless clinically, because it makes no distinction between dyslexia and other causes of reading failure, to such an extent that many educationalists now believe the whole concept should be discarded [4].

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## Social Causes

Almost everybody who fails to learn to read has failed to grasp the phonological principle, not just people with dyslexia. Thus, it could be argued that all those who fail have dyslexia, or none, have it [1]. In reality, most children who have great difficulty with learning to read are not dyslexic, but suffer social disadvantage: poverty, a chaotic home life and lack of parental support, poor teaching, truanting etc. These impediments prevent their learning to read, but they are not classical dyslexia. They can be counteracted by better support, in particular in phonics [2]; and the failure of successive governments to provide enough funding for such support is a sad sign of the current lack of compassion in our Society.

More phonics support will not help most people with true dyslexia however, because they need help upstream of phonics, with the visual sequencing that underlies the ability to grasp that principle. As things stand in the UK today, these needs are not usually detected because dyslexia is neither screened for nor diagnosed, except by families with the financial means required for a private diagnosis. Now therefore, privately diagnosing DD has become further accused of damaging the already unfavourable prospects of vulnerable communities by gaining richer families the additional help that the diagnosis attracts, thereby depriving poorer children of these limited resources. So there is still considerable stigma attached to those who succeed in obtaining the diagnosis ([www.succeedwithdyslexia.org/blog/dyslexia-me-what-is-social-stigma/](http://www.succeedwithdyslexia.org/blog/dyslexia-me-what-is-social-stigma/)). Because the phonological theory bundles all the causes of reading failure together, it makes the medical model seem to unfairly favour those who are dyslexic, i.e. that dyslexia is somehow more admirable than other causes of reading backwardness. Ideally every child who has problems learning to read should receive all the help they need irrespective of the cause - with no judgement at all about their merit.

In this narrative review we will first present the classical concept of developmental dyslexia that is adopted here. Then we will discuss in more detail the weaknesses and misunderstandings of the phonological theory, and then the basic physiology of the visual sequencing that is a crucial prerequisite for learning to read and acquiring the phonological principle. This will lead us on to suggestions as to how dyslexia can be reinstated as a useful diagnosis and how it can best be identified and treated.

## What is Developmental Dyslexia?

In 1968 the World Federation of Neurology advised that the term developmental dyslexia should be applied to

children who “fail to learn to read despite adequate intelligence, conventional instruction and sociocultural opportunity”. However, this definition has now been abandoned because these conditions are too vague. In practice, the crucial test was to demonstrate a large difference between a child’s reading and his oral vocabulary, comprehension and reasoning. The most commonly observed cognitive impairment found in dyslexia is a difficulty in phonological processing (i.e. in phonological awareness, phonological processing speed and phonological memory). However, phonological difficulties do not fully explain the variability that is observed as noted in a recent Delphi consensus study [5]. Most dyslexia practitioners still use the difference between a child’s oral language skills and reading as a marker of dyslexia. Hence most studies still look for a significant discrepancy between a child’s good oral comprehension and reasoning, yet poor reading, as the main criterion for a dyslexia diagnosis.

## The Phonological Theory (PT) of Dyslexia

The phonological theory was introduced as a result of the revolution in linguistic theory introduced by Noam Chomsky and elaborated in ‘The Sound Pattern of English’ [6]. This showed how innate recurrent networks in the brain could generate an almost infinite number of different words from just 42 basic phonemes which are represented by 26 letters or groups of letters. Importantly, it also explained how these phonemes are actually only theoretical; they are never explicitly heard in real speech because words are spoken as continuous sounds without interruptions. Furthermore, the pronunciation of phonemes varies greatly according to context; e.g. the end /z/ sound of s in cats and busses compared with /ss/ in bus. The concept of phonemes is thus a convenient mental abstraction, rather than referring to any consistent sounds [7].

An important component of learning to read in alphabetic scripts is indeed for children to absorb this abstract idea implicitly, in order to direct their translation of letters into actual word sounds. But it is by no means the only pathway to reading. Indeed, even in English, it can only directly help with about half of common words. The remainder do not have regular letter/sound correspondences and need visual sequence memory to remember them. And logographic scripts are morphosyllabic with the sound of the word a relatively minor feature of them.

In fact, the phonological ‘theory’ is not really a theory at all. Theories are meant to point to an explanation of the phenomena to which they refer, but the PT provides none. Instead it is more of a tautology. Phonological awareness is indeed an important path to alphabetic reading, but the PT doesn’t explain why it is deficient; it merely repeats, using other words, that it is missing.

## Logographic Scripts

Probably a majority of writing systems are not alphabetic, but either logographic or syllabary, without direct letter to phoneme correspondences. Obviously, particularly given the agreed genetic basis of dyslexia, whether it exists in countries using these scripts is a fascinating issue. There was a celebrated early report about an English/Japanese bilingual who was dyslexic in English but not in Japanese [8], which supported the then Japanese official view that dyslexia did not exist in Japanese. This report did not suggest this and subsequently it has become clear that dyslexia does in fact exist in both Japan and China [9]. This review's author suggested that actually it might be even commoner in logographic scripts because of their visual complexity.

Recently it has become clear that the demands of reading and writing in both alphabetic and logographic scripts have a big effect on the development of the left hemisphere connectome for reading [10]. Chinese dyslexic children exhibit increased activation in the right inferior occipital/middle temporal regions coupled with increased activation of left frontal areas. These differences probably help to compensate for their weaker visual-spatial orthographic analysis that is probably caused by impaired development of their visual magnocellular function [9].

## Word Blindness

It is obvious to most people that, rather than phonology, learning to *see* letters or character strokes properly in the right order is actually the real start of learning to read. The pioneers who developed the concept of developmental dyslexia in the 19th and 20th Centuries, such as Kussmaul, Berlin, Morgan, Hinshelwood, Orton and Critchley [11], all thought it was obvious that the first step in learning to read was to learn to see the text properly. So they often called it 'word blindness'. This referred, not to eye trouble, but to higher level lack of proper visuomotor coordination between what the two eyes are seeing and how they move over letters and words with each new fixation. That process requires the brain to signal accurately the moment in time at which the two eyes focus together on the first letter in a word, then move to the next and so on. Similar timing and sequencing is also required for logographic character reading and writing.

Modern research has demonstrated unequivocally that visual timing is carried out by 'magnocellular' nerve cells and that these work more slowly and less precisely in dyslexia [12]. This is why so many people with dyslexia complain of visual difficulties such as letters and words appearing to blur, to move around, change their order, go double and give them eye or headaches. This is sometimes called visual stress [13]; but more important than the discomfort is probably the accompanying failure of visuomotor integration

that results in difficulties in accurately sequencing the letters and strokes in a word.

## Visual Sequencing

When children are first confronted with a written word, they see it, not as a sequence of letters, but as a single object, like a cat or mouse [14]. They wouldn't normally have to deconstruct a cat into an orderly sequence of whiskers, nose, ear, body, tail; but in the case of written words they have to learn how to do so – how to disassemble them into the sequence of their constituent letters or strokes. This was first shown explicitly by Morais and colleagues in illiterate Portuguese who were first learning to read as adults [15]. They found that these adults could not split word sounds down into their constituent phonemes until they had first learned to perceive the same written word as a sequence of letters. This turned out to be also true of young children learning to read. Visual sequencing is primary and the development of phonological awareness depends crucially on learning how to do it. In logographic scripts accurate visual sequencing is just as important, but even more difficult, because the eyes and visual attention not only have to follow horizontally, but also vertically.

## Visuomotor Integration

Visual sequencing of letters and character strokes is much more complex than it may at first appear. The child has to learn how to inspect each element in the right order, initially by moving the two eyes to fixate on the first element, then on the next and so on. Later in the learning process she will move her attention from letter to letter similarly. But each time she moves her eyes the position of the letter or character feature shifts on her retina. Therefore, in order to keep their apparent position stable on her personal cognitive map of space, the coordinates of her eye movements must be used to compensate for this shift.

This computation is probably performed by the cerebellum which forms an internal model that is able to predict the quantitative features of the shift accurately during the actual eye movement, and thus compensate for it. The main signal provided for this model is provided by the visual 'magnocellular' timing system which is sensitive to the motion of the images across the retina during eye movements and projects heavily on to the cerebellum [16].

## The Visual Magnocellular System

Around 10% of the ganglion cells in the retina are 'magnocellular' which means that they are much larger (magnus in Latin) than the more numerous, smaller, 'parvo' cells [17]. The magnocellular neurons (M-cells) signal the timing of

visual events. They are sometimes also called ‘blob detectors’ because they have low spatial but high temporal resolution. This means they do not contribute to visual acuity, but they are very sensitive to visual motion, even at low contrast and luminance.

The M- cells form the basis of the brain’s magnocellular system for signalling the timing of visual events. This extends via the Superior Colliculus in the brainstem and the Lateral Geniculate Nucleus (LGN) in the thalamus, to the primary and secondary visual cortices and to the motion sensitive visual cortex (MT/V5). Beyond the posterior cerebral cortex the system extends as the visuomotor ‘dorsal stream’ [18] projecting to the parietal, premotor and prefrontal cortices, and subcortically to the cerebellum, brainstem and basal ganglia. This is the system that is primarily responsible for visuomotor control and for the direction of visual attention. Hence it is crucial for visually sequencing the elements in a written word. It forms a vital component of the ‘reading connectome’, the network of connections that has to develop successfully for fluent reading.

Throughout the whole M- system the M-neurones form a molecularly related network, distinct from other neurones, and they express unique proteoglycan signature molecules on their surface [19]. This means that the lineage of these cells can be identified throughout the nervous system using specific antibodies such as CAT 301. This network is specialised for using motion and timing signals to visually control movements, particularly of the eyes for reading, which demands compensating for the resulting shifts of images across the retina, as we have seen.

In order to understand how the visual magnocellular system contributes to reading, hence how it impedes reading when it fails to develop properly, we now need to consider in greater detail how it has been studied and how it functions. First, we will discuss recent results tracing it stage by stage from the eye to the visual cortex and beyond, and also via the various subcortical pathways.

Most of the evidence has depended on measuring subjects’ perceptual sensitivity to visual stimuli whose physical properties are varied systematically, using so called ‘psycho-physical’ techniques. The most commonly used are gratings whose contrast, distance between bars (spatial frequency) and presentation timing (temporal frequency) are varied. The responses to these stimuli can then be compared between typically developing children and those with dyslexia.

## Retina

The high contrast and temporal sensitivity, but low spatial acuity of magnocellular retinal ganglion cells were first recorded from Y parasol cells in the cat and thereafter in primates including man [17]. They all express the Cat 301 sensitive proteoglycan which identifies them as members of

the magnocellular lineage. It is now possible to record their electrical contribution to the electroretinogram and thus to demonstrate their impaired development as found in dyslexics [20]. Probably, using modern adaptive optics, it will soon be possible to measure their actual size in the living human eye to compare those in typically developing with dyslexic children.

## Lateral Geniculate Nucleus (LGN)

Recordings from the LGN in a patient with epilepsy not involving the LGN, showed a 60 Hz response with a latency of 40 msec that was almost certainly generated by M- cells. It was driven by the refresh rate of the presentation screen, but not perceived by the patient at all [21]. This shows that the M- cells’ high temporal sensitivity does not necessarily reach consciousness. These cells are also the most vulnerable to the effects of raised intraocular pressure (glaucoma). The first clear demonstration of M- cell atrophy was therefore reduced thickness of the M- cell layers in humans with early glaucoma [22]. Such changes had already been shown in dyslexic brains post mortem [23]. Now high strength magnetic resonance imaging (MRI) has confirmed that the LGN M- cell layers are significantly thinner in the left LGN of live dyslexics, particularly in males, although a similar pattern was seen in females [24].

## Visual Cortical Areas

Having a bigger representation of central visual field in the primary visual cortex (V1) appears to be associated with superior visuospatial skills. Therefore, children with dyslexia and impaired visuospatial function might have an altered central visual field representation. Vidyasagar et al. [25] found that teenage dyslexics showed reduced visuospatial efficiency in a conjunctive visual search test. This was actually accompanied by a larger central visual field overall, but with a much reduced asymmetry between left and right. Normally, the asymmetry slightly favours the left, but in the dyslexics it greatly favours the right. This confirms a much earlier literature suggesting that early in development, the right hemisphere becomes more developed in dyslexia [26].

The visual cortical area that receives the most magnocellular motion signals is area MT, also known as V5; this lies in the prestriate cortex, ventrolaterally, just above the ear, posterior to the meeting point of the ascending limb of the inferior temporal and the lateral occipital sulci. The best stimulus for exciting the M- system selectively, hence activating MT, is a display of dots moving around randomly in which a variable proportion of the dots can be moved in a swarm in one direction (a random dot kinematogram -RDK).

Numerous studies have confirmed that dyslexic people need a greater proportion of the dots to swarm in order to

see the ‘coherent motion’ [27]. Eden and colleagues were the first to show by functional MRI imaging that teenagers with dyslexia needed a much denser swarm to activate V5/MT than did typically developing children [28]. Since then a multitude of functional imaging studies have demonstrated that the development of the network of connections necessary for reading (the reading ‘connectome’) is impaired in dyslexia and that the degree of this impairment predicts the degree of impairment of their reading progress [29]. Since a major input to this connectome is the magnocellular timing system these observations suggest that its abnormalities in dyslexia are in part due to aberrant development of the magnocellular system.

### Superior Colliculus

The superior colliculus is a brainstem nucleus that receives in its superficial layers branches of mainly visual magnocellular axons from the optic tract and also from visual cortical areas. Its deepest layers also receive projections from cutaneous and proprioceptors and from somesthetic & auditory cortex. The superior colliculus is responsible for subcortical control of whole body orientation, including eye movement control. It is usually subordinate to the cerebral cortex, but it can respond very rapidly in emergencies. Its magnocellular input has been shown to be decreased in dyslexia [30].

### Cerebellum

The cerebellum (little brain) resides under the back of the occipital lobe of the cerebral cortex. It contains more neurones than the whole of the cerebral cortex put together. Its major input is from all parts of the cerebral cortex. In addition it receives inputs from the whole brainstem and spinal cord. Its visual input is mainly magnocellular from the optic nerve, superior colliculus and visual cortices [16]. Although it is best known for its role in controlling movements, it is now clear that its huge expansion in humans has occurred because it is crucially involved in thinking, feeling and cognition – in the words of Sir Charles Sherrington: ‘Thoughts are but movements confined to the brain’.

In relation to dyslexia, the cerebellum first became of particular interest because of its crucial role in the control of eye movements for sequencing letters and strokes and integrating their effects on visual feedback. But now its role in cognition has become clear, its importance in other aspects of reading development is generally acknowledged [31].

### Eye Movement Control

As we have seen on earlier pages, in order to sequence the letters in a word properly, the movements of the two eyes have to be correctly integrated with what each eye is seeing.

This important computation is carried out by the cerebellum which, in addition to its large visual magnocellular timing input, also receives signals about the contractions of all eight eye muscles together with current eye position. These signals enable it to develop an internal model to predict the visual consequences of each eye movement and thus compensate for them, and thereby to maintain the stability of the perception of space [16]. Accordingly, there is a large literature showing that eye movement control is defective in dyslexia [32].

### The Reading Connectome

This short exposition of some of the recent discoveries about visual structures involved in the sequencing of letters and strokes has given us more insight into the reading connectome, the complex network of connections that have to develop properly for learning to read. This mediates the 3 basic contributions that the visual system makes to learning to read: (1) sequencing letters, (2) matching letters with the phonemes they represent, and (3) learning to memorise the visual form of words.

So far we have discussed mainly (1), the visual sequencing of letters and (2) the letter/phoneme associations which are mediated by developing connections between visual, left posterior parietal, left middle temporal and prefrontal cortices. Xu et al. [33] used MEG recordings to track the evolution of brain activity when young adults were learning novel symbol phoneme associations. This showed how the right superior temporal cortex began activating to the visual input far earlier than the left parietal cortex during successful learning.

(3) The memorising the visual appearance of words has been demonstrated to depend mainly on the development of a cortical area under the left occipital lobe, now known as the visual word form area [34]. All of the areas and connections discussed above have been shown to be less developed in people with dyslexia [35].

### Auditory Sequencing

This review is focussing mainly on the crucial role of visuo-motor mechanisms in reading. But the left superior temporal cortex also receives auditory information about the sequence of phonemes in a spoken word; and so it appears to be the area where all these influences are usually integrated. i.e. It is the true seat of grapheme/phoneme associations, for grasping the phonological principal. But we should remember that visual sequencing also primes auditory sequencing of the sounds in the spoken word [15], and that the auditory system contains CAT 301 staining magnocellular neurones analogous to the visual ones [19]. This system is organised very similarly to the visual magnocellular one and provides

timing signals for accurate auditory sequencing [36]. One should therefore consider the auditory magnocellular timing systems in tandem with the visual, and note that in some people with dyslexia, perhaps 25% [37], the auditory timing deficit may be more obvious than the visual.

## Genetics

One of the few things that everybody agrees on about dyslexia is that it is highly heritable. So far however, the most important contribution of modern molecular genetics to dyslexia has not been to greatly increase our understanding of its biochemical basis. But by clearly demonstrating its neurodevelopmental origin, it has made it very difficult to argue that dyslexia is purely cultural and psycho-linguistic. So in principle it should be easily distinguishable from socially caused reading disability.

One advantage of dyslexia to genetic studies is that reading can easily be measured, hence genetic variant associations with dyslexia can be more easily detected in smaller samples than for conditions such as schizophrenia which is very much a subjective, clinical, rather than psychometric, diagnosis. Consequently, there are only 8 common gene variants that have so far been strongly linked with dyslexia [38], compared with over 100 for schizophrenia [39]. But 2 of the common variants are on the same gene, *DCDC2*. So far therefore, only 7 candidate genes have been identified for dyslexia. Almost certainly, however, many more will be discovered as sample sizes increase in genome wide association studies (GWAS).

It is also important to remember that dyslexia is not a monogenic condition, unlike, for example, cystic fibrosis, where one gene is the sole cause of the disease. No dyslexia gene variant is the single cause of the condition; each only confers some vulnerability; and they all interact with other genes, epigenetic effects and the environment to determine their eventual developmental outcome.

Importantly, 4 of the so far recognised dyslexia gene variants, *KIAA0319*, *DCDC2*, *DYX1C1* & *ROBO1*, are involved in controlling the migration of neurons to their final locations in the developing brain, which explains why magnocellular cells end up in anomalous sites in dyslexia [40]. In addition, *DCDC2* [41] and *KIAA0319* & *ROBO1* [42] have all been found to be associated with specific M-cell deficiencies. These genes have also turned out to have roles in ADHD and autism, which helps to explain why dyslexia and these two conditions are so often found together; they are 'comorbid'. Indeed, these three are so interrelated that which label a person receives often seems to depend mainly on which of ADHD, autism or dyslexia the psychiatrist they see first is most interested. Often successful treatment of their reading problems improves their ADHD or autistic symptoms as well.

## Autoimmunity

The development of both the visual and auditory magnocellular systems is under the control of the Major Histocompatibility Complex (MHC) which consists of 224 genes situated on the short arm of Chromosome 6 in humans [43]. Many of the genes associated with dyslexia, such as *DCDC2* & *KIAA0319* are among them. The function of the MHC is to present fragments of invading microorganisms to the immune system so that antibodies or T cells can be mobilised to neutralise them, unless they are 'self' in which case they are protected.

In the case of the gene, *KIAA0319*, the protein probably facilitates migration of magnocells to their correct locations in the brain, and if it is absent, mismigration occurs [44]. In dyslexia, M- cells are particularly vulnerable to antibody attack. This is most clearly demonstrated in autoimmune conditions, such as systemic lupus erythematosus (SLE). Mothers with SLE are over twice as likely than normal to have male children who are dyslexic [45]. Likewise, children with dyslexia demonstrate their immunological vulnerability by having a much higher incidence of autoimmune conditions such as asthma, eczema and allergies than do their typically developing peers [46].

In the light of recent discoveries about how pregnant mothers can produce antibodies that attack the developing brain of their foetus [47] and how dyslexia is associated with immunological problems, we decided to test whether mothers of dyslexic or autistic children may make these antibodies. We took serum from mothers who had had two or more dyslexic or autistic children and injected it into the uteri of pregnant mice. We then tested the offspring of those mice for behavioural abnormalities and for antibody binding to the cerebellum and looked for anomalies in cerebellar metabolism by magnetic resonance spectroscopy (MRS). These young mice were indeed affected. In a standard rotating rod test of motor coordination, they fell off the rod much more quickly [48]. These abnormalities were found to be associated with the human mother's antibodies binding to the mouse pups' cerebellar Purkinje cells, and their severity correlated with MRS indices of impaired cerebellar metabolism [48]. These results confirmed that dyslexia is indeed associated with abnormal immunological reactions.

## Neurodiversity

If developmental dyslexia is simply regarded as a 'disease' due to impaired development of magnocellular timing systems in the brain, apart from the misleading stigma that ensues, it is difficult to explain why the gene variants helping to cause it remain so common. Lack of visual motion sensitivity would make you very vulnerable to rapidly moving ferocious tigers and end your chances of passing

on your genes. Their survival in our genome implies they must have some compensating advantages in a ‘balanced polymorphism’. The term ‘neurodivergence’ was introduced because there is a wide range of ways that people perceive and respond to the world. Unusual ones are not necessarily diseased, and it seems that often they ought to be embraced and encouraged [49].

The concept of neurodivergence thus picks up on the possible strengths of the neurodevelopmentally modified brain, and also on the way in which dyslexia genes affect to some degree the whole spectrum of neurodevelopmental comorbidities from developmental language impairment (dysphasia), developmental dyspraxia, dyslexia through to more psychiatric conditions such as obsessional compulsive disorder (OCD), ADHD and autism. Far less stigma attaches to using the title Neurodiversity, than it does to the psychiatric terms. Furthermore, it embraces both the medical disease model and the psychological misfit idea, and it has therefore become very popular. The outstanding talents of many neurodivergent people are now well known and discussed; they have helped some of them to achieve great fame in all walks of life.

### Counter-Arguments

All the research so far discussed has been correlational however. Theoretically therefore, the deficits demonstrated could be the result rather than the cause of reading failure. Lack of reading experience could lead to lack of maturation of the necessary pathways. So, unlikely as this may seem, reading failure could be caused by too little exposure to written words, rather than genetic inheritance interacting with adverse environments. There are 3 kinds of evidence that counter that line of argument:

### Control Tasks

First is the use of control tasks. If we show that people with dyslexia are impaired at M- cell tasks, and that this impairment predicts the degree of their reading problems; yet they are as good or even better at psychophysical tests that do not depend on M- cell performance, this strongly suggests that their reading failure is associated with their M- defect. For example, Lovegrove et al. [50] used a fine grating stimulus as a control and showed that people with dyslexia actually have a higher sensitivity than typically developing controls (TDCs) to high spatial frequency, low temporal frequency gratings that best stimulate P, rather than M- cells. This implies that their M- cell deficit may be balanced by P-cell abundance, emphasising again how their M- deficit specifically underlies their reading problems.

Another example is the use, as a control group, of younger typically developing children matched with dyslexics on

their current reading level (RL), a ‘reading level match’ design. Then if the DD visual magnocellular function is worse than that of the RL matched younger children, this implies that this deficit is a cause of their worse reading. This kind of design has repeatedly confirmed that inferior visual magnocellular function contributes to DD reading problems [12].

### Cohort Studies

Second, we must pay attention to the time when a neurological deficit first appears. Cohort studies follow groups of children with or without family histories of dyslexia. Since dyslexia is so highly hereditary, around 50% of the Family History group will develop dyslexia, whereas only c. 8% of those without a genetic vulnerability will do so. Many such cohort studies have been carried out and they have shown that neurological deficits manifest long before reading starts in many of those with genetic vulnerability [51], indeed sometimes in utero [52]. Its appearance can predict reading problems up to 8 years later. These results demonstrate that genetically based dyslexia impairs neural pathway development, right from the earliest stages of brain growth, and this impacts on learning to read many years later.

### Training M-Cells

Third, perhaps the most convincing way of demonstrating that an M-cell deficit causes later reading problems, rather than vice versa, is to show that improving M system performance can remediate the reading failure. The most direct way to improve M- cell performance is to train children with visual grating stimuli optimised to selectively activate magnocellular retinal ganglion cells whilst the stimulus is adaptively made more and more difficult to see. This can have a dramatic effect on their reading [53]. There is now a plethora of other studies that have shown that there are many other effective ways of training children’s visual M-cells to perform better. Visual perceptual training benefits reading fluency and comprehension; visually based reading acceleration programs improved reading accuracy and rate; and action video games can increase rate and fluency [54].

### Coloured Filters

There have been frequent reports that a much simpler approach - viewing text through coloured filters - can greatly help children with visual reading difficulties to avoid the odd visual illusions that impede their reading, and that when they do so, this can help them to learn to read [55–57]. But these claims have been vigorously contested, particularly by those who adhere to the phonological theory and see no role for aberrant visual processing at all [58]. Listed as ‘one of the

most persistent neuromyths in education', nevertheless the reports continue and there are definitely some people who have personally experienced notable improvements in their visual symptoms from viewing text through coloured filters [59]. This subject certainly remains highly controversial, but the mounting evidence for the importance of the M- system's contribution to the control of eye movements and their integration with visual input in reading means that it has become sensible to consider the colour sensitivity of M- cells.

### Yellow Filters

When we began thinking about what colours might optimally activate M- cells, yellow was the obvious choice. M- cells do not contribute to perceptual colour sensitivity, but because they receive mainly from the cones that are sensitive to long (red) and medium (green) wavelengths they are most activated by a mixture of the two, warm yellow. This also happens to be the colour of light at noon on a summer's day.

In addition, in 1985 we had shown that temporarily wearing a patch occluding the left eye in children with visual reading problems could promote stable right eye dominance, and in those in which it did so, their symptoms diminished and their reading improved greatly [60]. One of the placebos we used was a pair of pale yellow spectacles transmitting most light at a wavelength of 550 nm. Unexpectedly the reading of the children who received these did much better than those who received ordinary plano glasses. We had also found that we could improve the acuity of children with reduced vision in both eyes, so called 'binocular amblyopia', simply by giving them yellow filters [61]. Accordingly, we decided to test whether yellow filters might facilitate magnocellular function in dyslexia. We found that they did indeed help; they particularly helped children who suffered from blur due to inadequate accommodation, and double vision due to poor binocular fusion [62].

The commercial systems that sell patented filter lenses of many colours need an expensive prescribing process to choose the right colour. However, they all tend to cluster around the yellow or blue part of the spectrum. We therefore carried out a randomised control trial measuring the improvements in visual symptoms and reading and spelling of dyslexic children comparing a multicoloured contact lense system, Chromagen, with the Dyslexia Research Trust (DRT) yellow or blue filters [63]. If the children's visual problems were mainly defocus blur, due to defective accommodation, and double vision, due to poor binocular fusion, we gave them yellow filters. However they were given our blue filters if they complained more of letters or words seeming to fizz or move around, due to unstable binocular fixation, and they suffered from sleep problems and headaches,. The children's symptoms and reading improved with our blue or yellow filters as much as when using the very large

number of different tint colours in the Chromagen range of contact lenses. For spelling however, which is usually much more difficult to remediate, the simple DRT blue or yellow filters were much more effective.

### Blue Filters

We were surprised that a remarkably large number of children benefitted from viewing text through our blue filters. This has only recently become explicable with the discovery of a wholly new class of intrinsically photosensitive retinal ganglion cells (IPRGCs) which contain the blue sensitive photopigment, melanopsin [64]. Its function is to entrain our body clock to prevailing seasonal day length, and it is situated in the suprachiasmatic nucleus (SCN) of the hypothalamus. Without information about the timing of dawn and dusk the SCN clock simply oscillates 12 h on and 12 h off. The function of the IPRGCs is therefore to signal the increasing light levels at dawn in order to switch the SCN onto its daylight phase, then at dusk on to its night time phase, at different times according to the season.

Deficient entrainment of the SCN clock to seasonal changes in day length has also turned out to be a potent cause not only of sleep disorders, but also of cognitive dysfunction, including the reading problems [65]. Wearing blue glasses with maximum transmission at 450 nm in the morning helps the clock to entrain to current day length. This arouses the child; and the first system facilitated by this increased arousal is the visual magnocellular system via the SCN's projection to a noradrenergic nucleus, the locus coeruleus (blue in Latin) [66], situated in the upper brainstem. Hence blue filters can achieve a multitude of benefits: helping children wake up, focus their attention, improve their mood, assist their reading, improve their sleeping and reduce their headaches.

### Nutrition

An altogether different approach to improving magnocellular neuronal performance is to improve the nutrition of children's brains. A considerable body of research has shown that deficient consumption of key nutrients has significantly deleterious effects on learning to read [67]. Correcting these deficiencies should boost these systems' timing functions, thereby providing further evidence that properly functioning magnocellular systems are fundamental for successful brain development. However these studies are extremely difficult to carry out, and since the results do not increase the sales of any particular food and, indeed, often point the finger at dangerous commercial practices, they are also very difficult to fund. Hence such studies are rare and extremely controversial.

Our interest in this approach derived from our genetic studies that suggested that a gene variant on chromosome 18, Melanocortin Receptor5 (MCR5), is associated with dyslexia [68]. This gene, unlike the other 4 MCRs involved in the central melanocortin system, is actually not highly expressed in the brain, but in exocrine glands such as the salivary glands, breasts, gut and pancreas. It helps to control appetite, energy balance and body weight by altering the weighting of ‘eat now’ versus ‘stop now’ signals according to prevailing conditions. These signals are derived not only from the brain’s peripheral sensory inputs but also from the gut itself. It therefore impacts not only on the control of body weight but also on gut/brain integration. Hence it is thought also to participate in regulation of the gut microbiome – the trillions of viruses and bacteria that inhabit our intestines [69].

### **Omega 3s**

A very important class of nutrient for the brain consists of the long chain polyunsaturated Omega 3 fatty acids: Docosahexaenoic acid (DHA) & Eicosapentaenoic acid (EPA). DHA is found in highest quantity in brain cells where its location spanning the inner and outer leaflets of the cell membrane provides the optimum physicochemical structural environment for successful neuronal electrical and synaptic development [70]. In addition, EPA is the basis for the synthesis of a very large number of important cytokines. But the modern Western diet contains fewer and fewer of these crucial molecules that make the brain work properly.

### **Antisocial Behaviour**

Prisoners in jail live in a closely regulated environment. Hence they ought to be ideal sites to carry out properly controlled trials of altering prisoners’ nutrition. It has often been suggested that simply improving prisoners’ diets should improve their social behaviour because our large brains evolved to enable our social interactions and cooperation. Gesch et al. tested this idea by comparing giving young offenders in jail daily capsules either containing placebo cornflower, or the omega 3s DHA & EPA together with essential vitamins and minerals [71]. Indeed, simply increasing their intake of omega 3s etc. without any special psychological help, did help them to reduce their rate of violent offences by a large amount, more than one third, compared with those given placebo. These effects have been replicated many times [72]. Since it has recently become clear that visual magnocellular timing plays a crucial role in mediating amicable social interactions [73], these results reflect again how important magnocellular timing functions are to successful brain development.

DHA also plays an important role in providing the cellular framework for biochemical reactions. Cognitive decline and dementia in the elderly is often associated with raised homocysteine in the blood. This can be ameliorated by treatment with a combination of Vitamins B6, B9 (folate) and B12. But this does not occur if the patient’s omega 3 DHA level is too low [74]. Therefore, this treatment should always be accompanied by DHA supplements. This confirms how DHA provides the framework for the reactions to take place and is thus an essential component for their success.

### **Inflammation**

Chronic inflammation has recently become acknowledged as another important contributor to maldevelopment of the magnocellular signalling of visual timing; and the important roles of DHA & EPA in alleviating this became particularly relevant during the COVID-19 pandemic [75]. Because they help to balance pro- and anti- inflammatory systems according to current requirements, an adequate intake of them promotes well-being and robust defence against pathology. These considerations emphasise how these long chain omega 3s play such important homeostatic roles, and again demonstrate how successful progress of visual magnocellular timing systems is crucial for normal brain development.

### **Conclusions**

The aim of this narrative review has been to present recent discoveries about the signalling of visual timing by the magnocellular system, and to argue how important this system is for learning to read, hence that its impaired development underlies developmental dyslexia. Our review of the function and dysfunction of many of the structures in the visual magnocellular system has demonstrated that reading both alphabetic and logographic scripts is controlled by the genetic and environmental factors that lead to developmental dyslexia. These impair the development of the normal visual magnocellular connectome for reading. The ‘bottom line’ is that impaired visual magnocellular development can be demonstrated beginning in the foetus in the 3rd trimester, at all sites and stages up to adulthood. The good news is that many of these deficits can be ameliorated by relatively simple and cheap interventions such as coloured filters and improved nutrition, and that if the problems are identified accurately at the appropriate times, more complicated treatments, such as specific, adaptive, visual magnocellular training, can be even more effective at overcoming them.

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Review of the hodology (network of connections) in typically developing compared with dyslexic children. Altered structural connectivity predicts poor reading skills.

- T. Lawton and J. F. Stein, “Visual Neural Timing Problems May Interfere with Reading, Attention, and Memory : Looking Beyond 20/20 Acuity,” *Optom Vis Perform*, vol. 10, no. 1, pp. 9–21, 2022.

Summarizes how impaired operation of the timing functions mediated by the visual magnocellular system can be remediated rapidly by a short period of exercises aimed at improving sensitivity to moving gratings.

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**Data Availability** No datasets were generated or analysed during the current study.

## Declarations

**Competing Interests** The authors declare no competing interests.

**Human and Animal Rights and Informed Consent** All reported studies/experiments involving human or animal subjects performed by the author were in accordance with the ethical standards of institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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