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Developmental Dyslexia—a useful concept?

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Abstract

Until recently Developmental Dyslexia (DD) was diagnosed if a person's reading and spelling was far behind what would be expected on the basis of their oral and non-verbal skills and they had a family history of similar reading problems; it was thought to be due to a hereditary failure of the reading circuits to develop properly in the brain. However, now it is widely believed that DD is due to failure to grasp the fact that the letters in a word represent the sounds in that word- the so-called phonological theory. Since the basis of all reading is the phonological principle, DD cannot now be distinguished from the many other causes of reading failure, and its very existence is doubted by some. Nevertheless, there is growing evidence in favour of the idea that DD can be specifically identified as due to disordered development of magnocellular neurons in the brain. These cells are specialised for temporal processing. Therefore, magnocellular weakness causes poor timing of visual events, hence inaccurate sequencing of the letters and sounds in words, which impedes learning to read. This means that people with developmental dyslexia do not have a diseased brain, but simply a different brain. Indeed, in many ways this may actually be a superior brain. Impaired growth of magnocellular neurones during development probably leaves room for parvocellular neurones to grow more connections. These may impart a holistic, rather than a linear, sequential, cognitive style to people with DD, and this can confer on them a more fertile imagination, innovation, originality and creativity. These talents may explain why the gene variants promoting dyslexia have been retained in the human genome. Therefore, our educational systems need to nurture, not condemn, dyslexics. Society urgently needs their strengths to help us cope with our increasingly complex world.

Keywords: Developmental Dyslexia, reading problems, magnocellular weakness and parvocellular strengths

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INTRODUCTION

This article describes a view of dyslexia that derives from the author's experience as a neurophysiologist and neurologist interested in the visual guidance of attention and movement. It was a great honour to be asked to give a talk to the Dyslexia Association of Singapore. Singapore's educational system is widely regarded as one of the best in the World. Decades of generous State funding (<20% of the National Budget) for teachers, schools and the educational infrastructure has meant that for the last twenty years or so, the average 15 year old from Singapore has scored the highest marks of all the countries in the world in the triennial Programme for International Student Assessments (PISA, 2019).

The Singapore system has achieved this high standard by imposing a fairly rigid diet of rote learning facts derived from likely exam questions & regurgitating them accurately. For reading, this involves teaching systematic phonics from infancy onwards to every child. But paradoxically, just as many educationalists in the US & UK were beginning to wonder how they could emulate Singapore's success by teaching yet more systematic phonics to children (Guardian, 2022(2), at the same time in Singapore itself, some people were beginning to wonder whether bludgeoning every child with phonics in their 'one size fits all' programme was really the best way, because it seemed that this might be suppressing children's natural creativity. Many were beginning to argue that a more flexible system, capable of adapting to each child's individual learning style might be better, in particular to suit the needs of developmental dyslexics (DD).

DYSLEXIA DEFINITION

However, at the moment there is a great deal of confusion about what DD is or even whether it can truly be said to exist at all. Here therefore, we will outline how there is probably quite a simple neurological explanation of why some children have difficulty with learning to read despite normal or high oral and non-verbal abilities. We will also discuss why this condition is so common – why the gene variants that cause the condition may have survived in our genome by carrying compensating advantages. Forward thinking people in Singapore want to nurture these advantages by developing a more flexible Educational System, because Singapore, above almost all other nations, depends upon the ingenuity and creativity of her Human Capital for successful survival.

The word 'dyslexia' (in Greek literally, 'disordered words') was coined by Rudolf Berlin in 1884 to apply to the rare patients he studied in whom brain damage had caused selective loss of their ability to read, yet had left them with their previous speech and oral comprehension skills intact (Berlin, 1884). We would now call this 'acquired dyslexia'. 9 years later J. Pringle Morgan borrowed Berlin's idea to help explain why his 14 yr old patient, Percy, after 8 yrs of schooling, still could not even read his own name. His teacher claimed that he would be judged the brightest boy in the class, if the assessment

could be entirely oral. So Morgan suggested that he might have a hereditary version of Berlin's dyslexia, which he called 'congenital word blindness' Morgan, 1896). We now call this 'developmental dyslexia' (DD) because it is now clear that it does not always have a visual basis. Morgan thought that the normal reading circuits in the left hemisphere had failed to develop properly in Percy - probably under genetic control.

For the next 75 years it was generally agreed that the key characteristic of developmental dyslexia was unexpectedly slow reading progress, despite normal speech and oral comprehension; ie it was thought to be caused by a specific neurological deficit that could be diagnosed by demonstrating a discrepancy between a dyslexic child's normal or high oral abilities, and her exceptionally poor reading.

THE PHONOLOGICAL THEORY

Around 1970 however the 'phonological theory' of dyslexia was introduced. This suggested that developmental dyslexia was mainly due to failure to learn and absorb the 'phonological principle' (Liberman et al., 1974). This is the principle that underlies all reading, namely that spoken words can be split into a series of elementary sounds, phonemes, which are represented by the letters in the printed word. It is clear that any child who fails to learn to read, has found it difficult to grasp the rules of how letters translate into sounds because this phonological principle is the basis of all reading.

However, this 'theory' of dyslexia is not really a theory at all in the sense of providing explanations; it is more of a tautology, merely repeating, using different words, that the children fail to learn the basic principle of reading. A proper theory should explain *why* things happen- why some children fail to learn this basic phonological principle.

Worse however, the theory has given rise to the mistaken belief that failure to absorb the phonological principle is the key feature of dyslexia. But since every child who fails to learn to read has failed to understand how letters translate into word sounds, such failure cannot specifically identify developmental dyslexia, since all the other children who find reading difficult for other reasons are also finding it hard to grasp the phonological principle. Hence this approach makes it impossible to distinguish developmental dyslexia from the numerous other possible causes of poor reading, such as low intelligence, poor teaching, truancy, social deprivation, or lack of family support.

DYSLEXIA CONFUSION

So today, the concept of dyslexia has become very confused and actually impossible to define clearly. Since it cannot now be clearly distinguished from any of the other causes of poor reading, some people believe that it cannot be said to exist at all, and that the whole concept should be abandoned (Elliott & Grigorenko, 2014). On the contrary however, others earn the scorn of the first group by arguing that dyslexia can be

identified as a positive brain difference, often leading to an unusually advantaged cognitive style that permeates the whole of life, involving the gamut of mental and physical health, education, intellect, culture, sport, social interactions and career success (West, 2009, Eide & Eide, 2011).

This article will basically support the second group, by briefly covering the consistent, strong and persuasive evidence that developmental dyslexia does indeed exist; that it can be easily identified; and that the brain differences it involves do indeed permeate the whole of life, and can carry with them significant positive advantages. Otherwise DD would not be so common.

Visual timing

The starting point for this account is the role of visual timing signals in the visual guidance of attention and movement. It is obvious that reading commences with the visual system - moving your eyes to see the letters and words in the right order. It does not start with their sounds or phonology. Learning that words consist of visual sequences of letters is the very first step in learning to read.

When children are first confronted with a printed word, they see it as a whole object, like a bug, or a mouse. You don't see a mouse as a whisker, then nose, then ears, then body, then tail, but as a whole mouse. Hence the first thing children have to do when beginning to learn to read is to grasp that written words actually consist of a sequence of letters which they have to learn to identify in the right order. This visual sequencing involves timing when the eyes or visual attention alight on the first letter of the word, identifying it, then timing when the eyes move on to the next letter, identifying that and so on.

Visual primes Auditory sequencing

It has been clearly demonstrated that it is only when children have learnt to properly sequence the letters in a word visually, do they begin to grasp that the spoken version of the word can also be split down into a corresponding sequence of separate sounds, phonemes (Morais et al., 1979). Thus learning to see a word as a sequence of letters primes children to learn to disassemble its spoken form into its sequence of phonemes. Only then can they grasp the phonological principle. Seeing the letters properly in the right sequence comes first. Thus visual sequencing is the initial step in learning to read, and it primes phonological analysis, and together these processes build up a background understanding of how each spoken word is formed of a sequence of separate sounds (the phonological principle).

Visual Magnocellular Neurons

This timing and sequencing is known to be carried out by a specialised set of timing neurons in the visual processing system, known as the visual magnocellular system (Merigan & Mounsell, 1993). This starts with large ganglion cells in the retina at the back of the eye, the magnocellular cells (magnus = large in Latin). These rapidly translate light detected by the rod and cone light receptors into nervous impulses that are sent up large fibres in the optic nerve, back to the brain. These large neurons form only 10% of all the ganglion cells in the eye. But they are specialized for timing when things happen. They are up to 50 times larger in area than the parvo (P- small) cells. So they cannot detect small details, like the difference between a small 'a' and an 'o'. But they do time events very rapidly and precisely. For instance, in the word dog, they time when the eyes look at the O, after the D. So they enable you to remember that the O came after the D. Thus, they play a crucial role in enabling you to sequence the letters in a word in the right order.

On the other hand, the majority of ganglion cells in the retina (90%) are small parvocells (L. parvus=small) which detect the detail you need to distinguish and identify the letters. The M- cells guide your eye movements to the right location, and they instruct the P- cells to identify what the letter is at that location.

Impaired development of M- cells

But because M cells are large, they need more upkeep, and so they are very vulnerable to drugs and damage. It has been shown convincingly that their development is impaired in many neurodevelopmental conditions such as dyslexia, dyspraxia, ADHD, and autism. In dyslexia, the evidence is now overwhelming, that the M- cells are abnormal (Meillleur et al., 2020, Benito-Kwiecinski et al., 2021). Here there is only space to give a few examples. But the amount of evidence in favour of the M- hypothesis grows literally every day. So it is baffling that most of it is completely ignored whilst the phonological theory of dyslexia still dominates completely.

Some of the most convincing evidence for impaired development of visual magnocells in dyslexia was first uncovered by Livingstone & Galaburda over 30 yrs ago (Livingstone et al., 1991). They studied the brains of dyslexic people who had been patients of the celebrated dyslexia neurologist, Samuel Orton, in the 1930s. He had persuaded them to bequeath their brains to the Orton brain bank, now at Harvard University. Galaburda's detailed histological study of the magnocellular layers of the thalamic visual lateral geniculate nucleus (LGN) in these brains showed that their M-cells were, on average, 30% smaller than in an ordinary control brain. Also, these cells spilled over into the Parvo layers so that there was no longer a clear space between the M- and P- layers, unlike in ordinary brains.

Nowadays, we do not have to wait for people to die before we can look at their brains with sufficient resolution. Using powerful magnetic resonance imaging (MRI) with a 7 Tesla magnet, Giraldo-Chika and colleagues were able to measure the thickness of the visual M cell layers in the LGN in living people with dyslexia (Giraldo-Chica, Hegarty & Schneider, 2015). And this has confirmed, so far in nearly 20 dyslexics, that they have significantly thinner LGN m- cell layers, particularly on the left side.

Visual reading problems

Earlier it was mentioned how reading starts with seeing letters properly in the right order. Many dyslexic children complain of visual problems when they try to read. These odd perceptions of letters moving around, changing places, blurring, splitting into two etc. seem to be the result of their impaired M- cell function. This is because the M cells control the movements of the eyes and attention when reading. If they are not working properly, the eyes wander out of control, like if you were drunk and things look blurry and in motion; this is called oscillopsia. (Maybe that's never a problem in Singapore!)

Interventions to improve M- function

The best way of demonstrating cause and effect is by Intervention. If you can show that a treatment that improves M- cell function in dyslexics helps them to improve their reading, this is strong evidence that M- activity plays a causal role in reading development. That these interventions are so successful attest to the crucial role of M- cell timing for learning to read and they provide strong evidence in favour of the visual magnocellular contribution to dyslexia. Here there is room for only a few examples, but there are many more that could be mentioned.

First, the most 'physiological' intervention would be to directly train M cells to respond better to moving stimuli and see whether this helps children with their reading. Terry Lawton has carried out numerous studies confirming this. She trains dyslexic children to detect low contrast moving stripes on a stationary striped background, and uses a computer programme that iteratively reduces the contrast as they improve, thus training the M- cells to get even better at it. As well as greatly improving M- cell sensitivity, this significantly accelerates their reading progress (Lawton & Stein, 2022).

Action video games

Another popular way of improving visual timing is to encourage children to play action video games – ie. games in which the child has to actively track targets. In general these have been clearly shown to improve the speed and accuracy of the direction of visual attention (Bevalier & Davidson, 2013). In a recent metanalysis of their effects in dyslexic children it was found that a commonly available action video game, not involving any reading at all, increased their reading rate and fluency and that these gains lasted for at

least 2 months and also accumulated if the games were continued (Peters et al., 2019). Visuo-attentional interventions are therefore highly effective in helping dyslexic children's reading, even though they do not involve any reading or phonics at all. They can help the children to improve their reading usually as much or more than other strategies most of which need much more teacher time and expense, and they are probably effective for all orthographies, since they don't involve reading a script.

Yellow filters

Although M- cells do not contribute to seeing colour, nevertheless, because they mainly receive from both red and green retinal cone receptors, they are most strongly stimulated by a combination of red and green light, which is yellow. Therefore, another completely different approach to improving M- cell timing function is to ask dyslexics to view text through 'Unique Yellow' filters. (They are called unique because almost everybody (even those with anomalous colour vision – slightly colour blind) agrees that the yellow is pure, neither greenish or reddish. Also they match the mix of wavelengths supplied by maximal sunlight at midday in the summer). Thus our yellow filters pass precisely the right mix of red and green light to stimulate M- cells maximally.

After just three months wearing these unique yellow filters, the dyslexics' magnocellular function improved very significantly: their visual motion sensitivity, contrast sensitivity and vergence control improved, hence their reading progress almost doubled, whereas control dyslexics, receiving just dark glasses limiting wavelengths across the whole spectrum, progressed by only one month in the three months, ie they went backwards, which is often the case with dyslexics who receive no special help (Ray, Fowler & Stein, 2005).

Blue filters

Another group of dyslexics benefit from viewing text through the opposite colour, 'unique blue' filters. These pass most light at 475 nanometres, - Oxford blue! These children advanced their reading by 13 months in the three months, whereas those receiving the placebo dark glasses went backwards again (Clisby et al., 2000).

Unique blue filters probably work via the brain's internal clock in the suprachiasmatic nucleus in the hypothalamus. This clock needs to be synchronized with changing day length between winter and summer. At sunrise the first rays of sun light in the morning are blue, and they are detected by special retinal ganglion cells which contain the blue sensitive pigment, melanopsin (Spitschan, 2019). These project to the hypothalamus and activate the M- timing cells via the 'blue nucleus', the locus coeruleus. So blue increases arousal and the children's ability to concentrate, and this is probably how it helps them to improve their reading so greatly.

Thus these treatment interventions that target the magnocellular timing systems are all successful in helping dyslexics to learn to read. Hence these examples add to the mounting evidence that impaired M- cell development is an important contributor to dyslexic people's reading problems.

Omega 3 DHA

Moving to an example of a slightly more speculative intervention, this concerns the brain's nutrition. As mentioned earlier, M- cells are highly vulnerable to drugs, disease and poor nutrition. The nutrient to which they are most sensitive to lacking in the diet, is the omega 3 long chain polyunsaturated fatty acid, (DHA), docosahexaenoic acid. This is because DHA is an essential component of the membranes of all nerve cells, as it provides just the correct physico-chemical environment for nerve cells' electrical activity (Crawford et al., 2013). Because M- cells are large, they contain more DHA than do smaller cells. So lack of DHA in the diet is a particular danger for them. Our main source of DHA is oily fish, and worldwide consumption of oily fish is decreasing, though less so in Singapore, maybe, than in the UK.

Many children with dyslexia in the UK come from highly disadvantaged backgrounds and we found that they had very low levels of DHA in their red cells, suggesting that they had not consumed enough throughout their lives. We therefore decided to see whether improving the nutrient content of their diets by giving them supplements of fish oil omega 3s might help them to improve their M- cell function, hence their reading. Indeed it did! In a randomised control trial (RCT), compared with similar children who received placebo supplement capsules, those who received the omega 3s improved their M- cell sensitivity, improved their concentration and greatly accelerated their reading progress (Richardson & Montgomery, 2005).

DEVELOPMENTAL DYSLEXIA DOES EXIST

What has been demonstrated so far in this article is that developmental dyslexia does indeed exist, contrary to the nay-sayers. There is now a great deal of evidence that backward reading in otherwise intelligent children is at least partly caused by impaired development of the brain's magnocellular timing systems, which are essential for sequencing letters and sounds properly. Part of this evidence is the often dramatic effectiveness of such apparently crazy treatments as video games, yellow, or blue filters, or omega threes in helping people with dyslexia to improve their reading skills.

The advantages of dyslexia

However, you might well ask, do we really want to fix dyslexics' timing weaknesses when those weaknesses can often be accompanied by such great talents. Everybody has their favourite list of famously successful dyslexics, such as Leonardo da Vinci, Rodin, Picasso,

Churchill, Einstein, and Edison. Might improving their reading have eliminated their exceptional talents?

There is no doubt that some dyslexics are prodigiously talented in areas other than reading. Dyslexia is 4x commoner in London art students than in the general population (Everatt, Steffert & Smythe, 1999). Likewise Lundberg & Wolf found that it is 10x commoner in Swedish art students than in other University students (Wolff & Lundberg, 2002). But these talents are not just confined to the arts. Dyslexia is also much commoner amongst entrepreneurs; being 35x commoner among entrepreneurial millionaires than among their managers! (Logan, 2008). And they are twice as likely to own two or more businesses.

It is often argued that dyslexics develop such talents only because they had to work so hard to find ways of avoiding reading. So it is claimed that they tend to choose art or architecture, practical engineering, computing or politics because none of these require much reading. But this argument is not very convincing. Actually nowadays all those professions do demand a great deal of reading. Furthermore, simply avoiding reading doesn't explain why so many dyslexics are so exceptionally successful at these professions.

Balanced Polymorphism

When a gene variant, ostensibly disadvantaging, is nevertheless common in the human genome, it is likely that it has confers some hidden compensating advantage, in a so called 'balanced polymorphism'. The classic example of this is Sickle Cell Anaemia (Weaver & Hedrick, 1992). If you inherit 2 copies of the sickling gene ('a'), all your red blood cells become thin and sickle shaped and so they clump together, sludge, damage and block your small blood vessels and cause severe anaemia and death quite quickly. However, if you inherit just one copy, only half your red cells sickle and these are much more resistant than normal, to the malaria parasite. The parasite cannot get into the sickled cells, hence you are more likely to survive into adulthood and pass on that gene to your children. Hence the 'a' recessive gene sickling variant survives in our genome as a defence against malaria.

Analogously, the same genes that contribute to dyslexia, may make people inherently more likely to become good artists etc. because the genes endow upon them unusual talents that enable them to flourish in these professions. Furthermore, learning to read was only invented c. 5000 years ago, and it has only become crucial in the last hundred years or so for most people; this is much too short a time for these genes to have been removed by natural selection for reading.

Dyslexia Talents

Because most of the emphasis in dyslexia research has been upon on the deficits that lead to reading failure, there have been comparatively few studies looking for possible compensating advantages that might have been conferred by a genetic balanced polymorphism. Usually the advantages have been discovered only as an unexpected by product of studies searching for deficits. One of the very first was in 1982, that of Lovegrove and colleagues (Lovegrove et al., 1982) who showed that, although dyslexics had lower contrast sensitivity to coarse gratings (spatial frequency (SF) = 2 Hz), to which mainly the M-cells are sensitive, they were actually a great deal more sensitive (400x so) to fine gratings (SF = 16 Hz). Only parvo cells respond at these high spatial frequencies, so this suggests that parvo cells are more richly connected in people with dyslexia.

Parvocellular advantage

This parvo advantage may be a result of their M- cell deficit. 90% of all the neurons that are born in the last three months of pregnancy are eliminated in the first few months of infancy, because they fail to make useful connections – 'use it or lose it' runs the adage. Magnocellular neurones in dyslexics start with the disadvantage of being smaller than normal, so that in this ruthless competition, their main competitors, parvo cells, can proliferate even more than is usual in ordinary brains. Anyway, in all brains parvo cells make much more branching and extensive connections with each other than do magno cells (Kravitz et al., 2012). So, in dyslexics the parvo cell networks probably end up, after the first few months of infancy, being considerably more interconnected than in ordinary brains.

This parvo superiority in people with dyslexia has been found to manifest itself by their having much higher sensitivity than in ordinary people both to the fine details that distinguish one letter from another (Geiger & Lettvin, 1987) and also for discriminating colours (Dautrich, 1993). But this difference is found, not at the centre of gaze (the fovea), but in peripheral vision where ordinary brains have very poor sensitivity to fine detail and virtually no colour vision at all, because normally there are very few parvocells this far out from the fovea. We confirmed these findings by demonstrating that the EEG potentials evoked in dyslexics by fine gratings (SF = 16 Hz) filling the whole visual field, are larger in dyslexics than in ordinary readers (Stein, 2021). Taken together all these results support the conclusion that the impaired development of the visual magnocellular system in people with dyslexia is accompanied by superior connectivity of their parvocellular visual networks.

This means that they can detect small details (high spatial frequencies) and small colour differences, particularly away from the centre of gaze, better than ordinary readers can. This difference probably explains why they like to think in pictures visually rather than with words (West, 1991). Due to their superior peripheral detail vision they can pick up

features that ordinary readers take longer to find and may miss altogether. But this does not imply that they have overall visuospatial superiority; it is only found when peripheral detail is required quickly.

This wider field of vision for detail manifests itself in their performance at detecting 'impossible figures' - whether drawings represent real objects, or ones that could not exist in reality, such as Penrose's triangle, Schuster's trident or M.C. Escher's impossible waterfalls. Most people have to shift fixation from one end of the picture to the other in order to detect their impossibility, but many dyslexic people can see the drawing all at once, hence to spot their inconsistencies more rapidly. So they are much faster at deciding whether they are impossible (von Karolyi & Winner, 2004).

Holistic thinking

This visual superiority endowed by their increased parvocellular connectivity, means that the way people with dyslexia tend to think is 'holistic' – seeing the big picture all at once. So they can see problems as if from a helicopter**,** flying above the forest of problems, and spotting solutions, rather than working sequentially from the roots. Likewise, this enables them to create a complete mental picture from separate pieces, by quickly seeing how they all fit together. Thus they can often see more easily than ordinary brains how the flat 2 dimensional plans of a building would look in 3 dimensions when built; this is why the architect, Richard Rodgers, himself a dyslexic, preferred to employ only dyslexics in his drawing office.

These exceptional characteristics are not confined to vision, but extend to mentation and abstraction. Thus, people with dyslexia often seem to have more vivid imaginations with a stronger tendency to daydream, all suggesting a superior ability to harness the brain's ability to alter and create new perceptions. They are said to be more curious about how things work, more insightful and intuitive, more highly aware of their environment with a heightened awareness of everything. They seem to have this uncanny ability to see patterns, both literally and also in data or ideas, to fill in their missing parts and to see what doesn't fit in. All these characteristics clearly contribute to their heightened artistic talents, innovation and general creativity.

None of these talents and traits can be easily studied scientifically however, because they depend so much on very subjective judgements which are not readily evaluated objectively. Here it is argued that the superior parvocellular connectivity of people with dyslexia is what explains how they develop this different way of thinking from ordinary brains. This holistic style contrasts with the more common linear, step by step, sequential thought processes of ordinary brains. So if a dyslexic survives school s/he is often very successful in life. But most schooling enforces a 'one size fits all' linear analytic programme, which is totally inappropriate for the dyslexic learning style and may harm

them for life. This appears to be what Singapore seems to be realising now and is taking steps to correct.

Exploration v. exploitation

The gene variants underlying the development of this holistic cognitive style have probably survived in our genome because in the search for optimum approaches to problems, they promote 'exploration' of new possibilities rather than sticking with 'tried and trusted' behaviour that works in predictable situations but may not be appropriate when circumstances change. As Helen Taylor points out, successful cooperation in groups depends on most people being 'exploitative', exploiting strategies that have worked well in the past, but a minority of about 10% should be more explorative - suggesting new approaches to new problems (Taylor, Fernandes & Wraight, 2022). Without these creative thinkers the chances of finding innovative solutions to new problems would be greatly diminished. Note that this has nothing to do with reading, and probably the selective pressure towards these proportions predates reading by several million years.

Note also that since humans are unique in being able to think using abstract symbols (Deacon, 2007), the dyslexic cognitive style advantages us not only in the search for solutions to practical problems, such as finding food, but also for thinking - searching in 'abstract space' for solutions to abstract cognitive problems that are often represented using symbols such as in language or mathematics. This generalisation of holistic thinking into such abstract meta-analysis is what underlies the general creativity and innovation for which the dyslexic mind is so famous.

It allows people with dyslexia to fully utilise all aspects of their individuality (perception, intellect, emotions, imagination, experience, actions and skills,) to achieve a more comprehensive and effective understanding of most situations and problems, than can ordinary brains. They are often able to understand complex systems faster and more completely than most people. Their holistic cognitive style enables them to grasp the many different types of relationships between the many elements in a system, and to discern any pattern within it, together with any missing pieces; and thus to spot crucial errors quickly. In particular, this explains their often remarkable 'people skills' - rapidly integrating all they know, all they've experienced and heard about a person to form an unusually accurate judgement - 'reading' a person's character. Even if these descriptions are only true of a minority of dyslexics, we should ensure that our educational systems do not stifle the development of such talents, as Society needs them urgently.

CONCLUSIONS

Due to the current dominance of the phonological theory as to its cause, there is currently no agreement about what Developmental Dyslexia is, or even whether it really

exists. Nevertheless, there is growing evidence in favour of the idea that it is due to disordered development of temporal processing by the brain, because the magnocellular neuronal systems which are responsible for timing, fail to develop properly. This magnocellular weakness leads to poor linear sequencing of the letters and sounds in words which impedes learning to read. This means that people with developmental dyslexia do not have diseased, but simply different, brains. Indeed, in many ways they may actually be superior brains. This is because impaired growth of magnocellular neurones during development may allow parvocellular neurones to flourish more, and grow more connections, which imparts a holistic, rather than a linear, sequential, cognitive style to DD. This confers greater imagination, innovation, originality and creativity and explains why the gene variants promoting dyslexia have been retained in the human genome. Therefore, we need to nurture, not disparage, dyslexics. Society needs them urgently to help us cope with our increasingly complex world.

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