

# Controversy about the visual magnocellular deficit in developmental dyslexics

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In a recent review, Bernt Skottun<sup>1</sup> strongly attacked the hypothesis that some dyslexics show impaired development of the magnocellular pathway of their visual systems. Significantly, however, he aimed his criticisms at only a very small component of this magnocellular theory. Lest anyone should believe that his critique has dealt the theory a mortal blow it is worth retracing some of the steps in the argument.

Morgan, Hinshelwood and Orton, who first described what we now call developmental dyslexia, termed it 'word blindness' because they believed that dyslexics' difficulty with reading was perceptual and mainly caused by visual confusion. However, following the Chomskian revolution in linguistics in the 1960s, the most widely accepted theory of dyslexia now is that it is primarily a linguistic problem and due to children's failure to acquire adequate phonological skills. To read a new word we must be able to translate the letters into the sounds that they stand for; most developmental dyslexics can only do this with great difficulty. This is seen as an impairment specific to language processing, perhaps the result of inheriting a linguistic processor that is dysfunctional. This might be not so severe as to impair the acquisition of speech, but sufficient to impair the fine grained phonological representations that are necessary for reading. This would reduce the accuracy of those representations and delay their recovery for reading. Thus, there is now a great deal of evidence that the majority of dyslexics have reduced phonological skills that correlate strongly with their reading difficulties and at least partially explain them<sup>2</sup>.

In the past 10 years, however, the idea that impaired visual processing contributes to dyslexics' difficulties has been making something of a comeback. Most authorities now accept that at least some, if not most, dyslexics have some degree of visual impairment. Much of the credit for this should go to Bill Lovegrove who, following a period working with Fergus Campbell in Cambridge, UK, went back to Australia and applied his grating contrast-sensi-

tivity technique to dyslexics. He showed that their contrast sensitivity was slightly reduced at low spatial frequencies, low luminances and short stimulus durations, whereas it was slightly elevated at high spatial frequencies<sup>3</sup>. On the basis of these apparently selective visual deficits he suggested that the development of the visual 'transient' system in dyslexics was mildly impaired, and that this might also explain their more sensitive performance at high spatial frequencies.

Therefore, with Bruno Breitmeyer, Lovegrove put forward the hypothesis that during reading, a slowed visual transient system might fail to suppress the sustained system's eidetic image of the previous fixation during each saccadic eye movement, so that the subsequent fixation would be superimposed upon it. Thus, the letters would jumble up, thoroughly confusing the reader<sup>4</sup>.

It is important to note, however, that the particular mechanism that Breitmeyer and Lovegrove suggested, whereby an impaired visual transient (magnocellular) system might impair reading, is not essential to deciding whether dyslexics have reduced transient system sensitivity in the first place. Indeed the work of Burr *et al.*, among others, has shown that in saccadic suppression the transient system inhibits the previous activity of the *transient* rather than the sustained system<sup>5</sup>; in other words, this aspect of the Breitmeyer/Lovegrove hypothesis is almost certainly incorrect. However, several alternative hypotheses have been put forward<sup>6</sup>. For example, in 1981, Stein and Fowler suggested that impaired transient sensitivity could result in unsteady binocular control, which might cause letters to appear to move around and over each other<sup>7</sup> – this in turn might contribute to the visual perceptual instability of which many dyslexics complain. Since then, more evidence has been adduced in favour of this hypothesis<sup>7-9</sup>.

Uncertainty about the precise mechanism by which a visual transient weakness might impair dyslexics' reading does not, however, alter Lovegrove's fundamental observation that many dyslexics do have reduced transient system sensitivity. Since that pioneering

paper, much more evidence has been obtained in support of it, both by himself and by other researchers (e.g. Refs 10,11). Thus, a number of independent groups have confirmed that dyslexics' grating contrast sensitivity is reduced, particularly at low spatial frequencies, low luminances, and short durations or high temporal frequencies. Dyslexics have also been shown to have shorter duration of visual persistence at high spatial frequencies<sup>12</sup>, which is consistent with a transient system deficit. Likewise their sensitivity to flicker, particularly of low spatial frequency gratings, is reduced<sup>13</sup>, as is the critical flicker fusion frequency, the highest temporal frequency at which they perceive flicker<sup>14</sup>. These are well-accepted measures of visual transient system function.

Over the same period of time, the idea has become widely accepted that visual transient processing is mainly mediated by large cells, known as magnocellular M-cells, that have rapid temporal dynamics in both the peripheral and central visual systems<sup>15</sup>. From experiments in monkey visual cortical areas it is now clear that moving stimuli are the most selective for this magnocellular/transient system<sup>16</sup>. Therefore, we and others measured the visual motion sensitivity of dyslexics, and it has been demonstrated that their motion sensitivity is indeed reduced<sup>17</sup>. Furthermore, the degree of individuals' reduction in motion sensitivity correlates with their reading impairment<sup>18</sup>. We have further demonstrated that the motion sensitivity of normal readers, whether child or adult, good or bad reader, predicts the visual orthographic component of their reading skill<sup>19</sup>. This is probably because it determines the precision with which they can locate and order letters in a word<sup>20</sup>.

Thus the idea that many, if not most, developmental dyslexics have mildly impaired development of the visual magnocellular (M-cell) system has taken firm root. More objective measures of visual M-cell function, such as recording visual evoked potentials<sup>21,22</sup> in response to moving stimuli, and fMRI activation of MT/V5<sup>23,24</sup> (which is dominated by magnocellular input), have confirmed

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this view. But perhaps the most convincing evidence of all was the demonstration by Livingstone and Galaburda in post-mortem dyslexic brains that neurones in the magnocellular layers of the lateral geniculate nucleus (LGN) were smaller and more disorganized than in control brains<sup>21</sup>. At present, we can only speculate what precise effect these pathological changes would have on M-cell performance psychophysically. But it is clear that such impaired development throughout the whole period of growth of the nervous system would have far more subtle effects, perhaps including consequential changes to the parvocellular or P-system, than the gross effects of wholly ablating the M-cell layers of the LGN in adult monkeys, which is the experimental model that dyslexics are often compared with.

Therefore, the visual magnocellular impairment in most dyslexics is mild, and all researchers would agree that it is not found in every dyslexic. Hence some studies have failed to confirm every detail of the hypothesis. But as we said in a recent review<sup>25</sup>, it is illogical to conclude that absence of evidence for some aspects of a magnocellular deficit in some dyslexics is evidence of its absence in all. The tests used in those studies might not have been sensitive enough, and the subjects chosen might not have been dyslexics with abnormal visual magnocellular function.

Bernt Skottun, however, argues prolifically against the magnocellular theory; he has written at least four papers challenging it<sup>1,26–28</sup>. In all of them he puts forward two main arguments. First, he maintains that the magnocellular hypothesis depends crucially on the M-system normally inhibiting the P-system during saccades. As explained earlier, and as we first pointed out in our 1997 review<sup>25</sup>, and as others also have reiterated<sup>29</sup>, this is not so. It is now clear that the visual confusion of dyslexics is not due to a failure of the M-cell/transient system on P-cell/sustained inhibition. As mentioned earlier, there are a number of potential mechanisms for dyslexics' poor reading that can plausibly be linked to poor M-cell/transient function. In any event, arguments about these are irrelevant to whether dyslexics have a magnocellular deficit in the first place.

Skottun's second argument depends upon focussing exclusively on grating contrast-sensitivity measurements as indices of M- and P-cell functioning. Such measurements are dominated by the peripheral retinogeniculate pathway. This focus is unjustifiably narrow, as it entirely ignores the strong evidence in dyslexics of impaired magnocellular involvement more centrally, in functions such as motion sensitivity<sup>17–19,23,24</sup>, ocular motor control<sup>7–9</sup>, visual search and visual attention<sup>30</sup>. These more central functions of the cerebral cortex are clearly closer to the cognitive skills required for reading that we are all interested in.

Nevertheless, even within the narrow confines of focussing on dyslexics' grating contrast performance, Skottun's arguments are tendentious. He points out, quite correctly, that many studies do not show greater reductions in contrast sensitivity at low rather than high spatial frequencies. Such a pattern would be expected if the peripheral magnocellular pathway were selectively and severely damaged, comparable, for instance, with the effects of ablation of the M-cell layers of the LGN in adult monkeys. Nobody, however, suggests that dyslexics' magnocellular deficit is as severe as this. Furthermore, many of the studies Skottun mentions were conducted at short stimulus durations or high temporal frequencies. Under these conditions, contrary to his assertion<sup>27</sup>, both low and high spatial frequencies would be affected by a magnocellular impairment<sup>31</sup>.

Moreover, few studies have separated dyslexics into those with and those without visual symptoms; in other words, most studies did not look separately at those who were likely to have a visual magnocellular deficit. When we did this however<sup>32</sup> (not mentioned by Skottun), we found, with static gratings, that the contrast sensitivity of those with visual symptoms was reduced at low spatial frequencies compared with poor readers without visual symptoms; but it was increased at high spatial frequencies. These results are precisely what would be expected if the magnocellular system is impaired in the dyslexics who complain of visual problems when reading. Using gratings flickering at 20 Hz, these dyslexics' sensitivity was reduced at all spatial frequencies, which is also consistent with a magnocellular deficit<sup>31</sup>.

Skottun does not seem to be denying that many dyslexics do have a mild visual deficit. Rather he seems to be suggesting that they have a parvocellular impairment as well. It may well be that impaired development of the M-system has some secondary consequences on the P-system. Nevertheless, if Skottun believes that this is the case, then he should also review the studies that have measured dyslexics' P-system performance, for example, in colour contrast and fine form discriminations. None of these has shown comparable deficits<sup>33,34</sup>.

In addition, Skottun's exclusive focus on the peripheral magnocellular system misses a crucial point about the wider implications of the magnocellular theory. It is not only a visual theory. The visual magnocellular system is the most researched and the best understood, but anatomically there are magnocellular-like divisions of the auditory, somesthetic and motor systems also. Linking all these systems in a generalized magnocellular theory can provide an explanation of how many seemingly irreconcilable features of dyslexia might be brought together<sup>6</sup>. Visual, somesthetic, phonological and motor deficits are all found in dyslexics, most probably because the development of magno-

cellular neurones in all these systems is impaired<sup>35</sup>, presumably under genetic control<sup>36</sup>.

One of the problems that dogs the study of dyslexia, and is really quite unnecessary, is the common tendency of researchers defending their particular turf to assume that physiological and psychological explanations must engage in head on competition and be mutually exclusive. In fact, of course they are not; they offer two different levels of explanation that are completely complementary; but do not have to match each other in every detail. It would be a pity to add to this problem another unnecessary dispute that depends so much on selection of just one kind of evidence.

In summary, Skottun is quite correct that not all studies of developmental dyslexics have confirmed that their visual deficit is entirely consistent with an exclusively magnocellular (transient-system) lesion; this is particularly true of those in which static grating contrast sensitivity was measured. However, because the impairment in dyslexics cannot be considered as equivalent to complete ablation of the magnocellular system (because their deficit is mild), and, in addition, not all dyslexics have it anyway, this is only to be expected. Nevertheless, the majority of studies using the most sensitive tests of overall M-cell/transient function have found that many dyslexics do demonstrate a mild magnocellular impairment. Furthermore, the M-pathway sensitivity of both good and poor readers can predict their visual orthographic skill for reading.

Skottun's critique thus serves as a useful antidote to any complacent view that the matter is settled; but one should not throw out the baby with the bathwater. We cannot conclude from his careful selection of just one aspect of the evidence relating only to the peripheral visual system that some dyslexics do not have some degree of impairment of the development of the visual magnocellular system, when there is so much other evidence to suggest that they do.

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## On the conflicting support for the magno-cellular-deficit theory of dyslexia

Response to Stein, Talcott and Walsh (2000)

Bernt C. Skottun

I have recently reviewed a substantial portion of the data bearing on magno-cellular deficits in dyslexia<sup>1</sup>. The main conclusion of this review is that although there are clearly studies that are consistent with a magno-cellular deficit, these studies are outnumbered both by the studies that have found no deficits and, by studies that have found deficits that are incompatible with magno-cellular impairment. These observations contrast with earlier claims of consistent support for a magno-cellular deficit among dyslexic readers<sup>2,3</sup>.

Stein et al. [Stein, J., Talcott, J. and Walsh, V. (2000) Controversy about the visual magno-cellular deficit in developmental dyslexics. *Trends Cognit. Sci.* 4, 209–211]<sup>4</sup> have written a comment on this review in which they claim that my arguments are 'tendentious'. This characterization is puzzling given that some of their summary statements – for example, that '[magno-cellular impairment] is not found in every dyslexic' – are not very different from my conclusions. Also, Stein et al. do not dispute

the finding that a large number of studies have found impairments that are incompatible with such deficits. Stein et al. break no new ground and bring forth nothing to alter the conclusions of my review.

The comment by Stein et al. does contain a number of errors and inaccuracies. It attributes to me the view that dyslexia should be the result of a parvo-cellular deficit. I have never held that view. It also makes the claim that magno-cellular deficits might manifest themselves at high spatial frequencies. In support of this contention are cited electrophysiological recording data obtained with high (75%) contrast gratings<sup>5</sup>. It seems that Stein et al. are confusing suprathreshold responses and contrast detection. Whilst it is quite clear that magno-cellular neurons can be activated by relatively high-frequency stimuli given sufficient contrast, these neurons do not mediate contrast sensitivity at frequencies above approximately 1.5 cycle/deg (as shown by lesion studies and human psychophysics<sup>1</sup>).

My review was confined (mainly) to contrast sensitivity. Stein et al. characterize this as a 'very small component' of the theory (p. 209) and as an 'unjustifiably narrow' focus (p. 210). Although there are other avenues for exploring magno-cellular deficits, contrast sensitivity remains the main psychophysical test as it is the main basis for distinguishing between magno-cellular (or transient) and parvo-cellular (or sustained) systems psychophysically in humans. Therefore, if other lines of research were to uncover consistent evidence for magno-cellular deficits one would still need to explain why the contrast sensitivity data provide such limited evidence for these deficits. For this reason, the lack of consistent support for magno-cellular deficits from contrast sensitivity studies is significant.

In this connection, one should note that visual deficits found using other tests typically lend themselves to alternative explanations. For instance, deficits in movement perception seem to be better conceptualized in terms of

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