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Notes and Comment

On the use of metacontrast to assess magnocellular function in dyslexic readers

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It has been proposed that dyslexia is the result of a deficit in the magnocellular system. Reduced metacontrast masking in dyslexic readers has been taken as support for this view. In metacontrast, a masking stimulus reduces the visibility of a spatially adjacent target stimulus when the target stimulus precedes the masking stimulus by about 30–100 msec. Recent evidence indicates that the latency difference between the magnocellular and parvocellular subcortical pathways is at most 20 msec and may be as small as only 5 msec, or even less. This makes it difficult to attribute the latency in metacontrast to the latency differences between the magnocellular and parvocellular systems. It is therefore problematic to attribute reduced metacontrast masking to a deficit in the magnocellular system.

It has been claimed that dyslexia is the result of a deficit in the magnocellular system. It was initially thought that this caused reading difficulties by reducing magnocellular inhibition of the parvocellular system at the time of saccadic eye movements (Breitmeyer, 1993; Livingstone, Rosen, Drislane, & Galaburda, 1991; Lovegrove, 1991; Lovegrove, Garzia, & Nicholson, 1990; Lovegrove, Martin, & Slaghuis, 1986). Without such inhibition (or with reduced inhibition of this kind), it was believed that parvocellular responses generated during one fixation would linger into the next so as to create confusion. Essential to this theory was the postulate that the parvocellular system is suppressed by the magnocellular system at the time of each saccade. Contrary to this postulate, it has become clear that it is the magnocellular system and not the parvocellular system that is the target of this suppression (see Skottun & Parke, 1999, for a brief review). This makes it difficult to maintain the magnocellular deficit theory in its original form (Hogben, 1997; Skottun & Parke, 1999). However, some researchers are proposing alternative hypotheses for how reading problems could result from a magnocellular deficit (Stein & Walsh, 1997). It seems that these efforts would be warranted only if there were strong and convincing evidence that dyslexic readers show reduced sensitivity in their magnocellular system.

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The main part of the evidence bearing on the question of a magnocellular deficit comes from contrast sensitivity studies. A recent survey of that evidence (Skottun, 2000) showed that, although some studies have found contrast sensitivity reductions consistent with a magnocellular deficit, a larger number of studies have found sensitivity reductions that are inconsistent with such a deficit. In addition to contrast sensitivity, there are other kinds of data that have been interpreted as being in support of the *magnocellular deficit theory*. Among these, is evidence from *metacontrast masking*. The purpose of the present report is to examine this evidence in light of recent data regarding the latency difference between the magnocellular and parvocellular systems.

Metacontrast

In metacontrast masking, one stimulus, the masking stimulus, affects the visibility (or visual appearance) of a spatially adjacent target stimulus (see Lefton, 1973, for review). One important feature of this effect is that it is largest when the target stimulus precedes the masking stimulus by 30–50 msec (Weisstein & Haber, 1965), 100 and 200 msec (Werner, 1935), or by over 100 msec (Alpern, 1953). Lefton, in a 1973 review of the literature, concluded that most studies found maximum masking for stimulus onset asynchronies (SOAs) of between 30 and 100 msec.

The fact that the masking effect is largest when the target precedes the mask has been interpreted to mean that the masking stimulus is mediated by a fast pathway and the response to the target stimulus is mediated by a slower pathway. The fast and the slow pathways have been thought to correspond to the magnocellular and parvocellular systems, respectively (Breitmeyer & Ganz, 1976; Breitmeyer & Williams, 1990; Edwards, Hogben, Clark, & Pratt, 1996; Williams, Molinet, & LeCluyse, 1989).

The Distinction Between Magno- and Parvocellular Systems

In primates, the magnocellular and parvocellular systems originate in the retina and are fully segregated in the lateral geniculate nucleus (LGN), where magnocellular and parvocellular neurons occupy separate layers (see Merigan & Maunsell, 1993; Schiller & Logothetis, 1990; Shapley, 1990; Shapley & Perry, 1986, for reviews). From the LGN, neurons in the magnocellular and parvocellular layers send axons to separate sublamina of layer 4 of the primary visual cortex (V1). After the cortical input layers, a considerable amount of mixing of the magnocellular and parvocellular inputs occurs (Malpelli, Schiller, & Colby, 1981; Merigan & Maunsell, 1993;

Nealey & Maunsell, 1994; Sawatari & Callaway, 1996). It seems therefore that, in order for a latency difference to be attributed unequivocally to a difference between the magnocellular and parvocellular systems, it will have to occur prior to the input layers of cortical area V1 (see also the Discussion and Conclusions section).

Latency Difference Between the Magnocellular and Parvocellular Pathways

The latency differences between the magnocellular and parvocellular systems have been measured and estimated by several investigators. On the basis of conduction velocities alone, one would expect the latency from the retina to the input layers of the visual cortex to be about 8 msec shorter for magnocellular neurons than for parvocellular cells (Maunsell et al., 1999). Actual measurements are in general agreement with these estimates and have typically been less than 10 msec (Marrocco, 1976; Maunsell et al., 1999; Maunsell & Gibson, 1992; Schroeder, Tenke, Arezzo, & Vaughan, 1989;). However, one report (Nowak, Munk, Girard, & Bullier, 1995) has indicated that the latency difference may be as large as 20 msec (this was measured in V1 and would include any potential differences in temporal integration). Mitzdorf and Singer (1979) using electrical stimulation found latency differences between magno- and parvocellular neurons to be 6 msec. In their review article, Nowak and Bullier (1997) estimated the average latency difference between individual magnocellular and parvocellular neurons to be 5.5 msec.

The above estimates are for single cells and do not take into account the fact that there are about 10 times as many parvocellular cells as there are magnocellular cells (Ahmad & Spear, 1993; Peters, Payne, & Budd, 1994). This has led some researchers (Maunsell et al., 1999) to assume that there is more convergence of parvocellular inputs than of magnocellular inputs. Maunsell et al. (1999) estimated that convergence may substantially reduce the difference in latency between the two systems. In fact, under some conditions, parvocellular latency may be even shorter than for the magnocellular system! This suggests, therefore, that there may be only a negligible difference in latency between the magnocellular and parvocellular systems.

Implications for Metacontrast

Lefton (1973) reported that most studies have found maximum metacontrast masking when SOAs are between 30 and 100 msec. Taking the middle of this range as an estimate of average SOA for maximum masking, we obtain an SOA value of 65 msec. If we adopt Nowak and Bullier's (1997) estimate of 5.5-msec average latency difference for single magnocellular and parvocellular neurons (at the entry to the visual cortex), this would make less than one tenth of the SOA of metacontrast masking accounted for by the difference between magnocellular and parvocellular neurons (between retina and cortex). This result is based on single neurons. If we were to take convergence into account (Maunsell et al., 1999), the discrepancy between the magnocellular and parvocellular systems on the one hand and metacontrast masking on the other would be even larger.

These values make it difficult to account for the latency in metacontrast, either in terms of individual magnocellular and parvocellular neurons or in terms of the overall systems. It seems quite clear that the entire SOA for maximum masking cannot be accounted for on the basis of the latency difference between the magnocellular and parvocellular systems at the point of entry into the visual cortex.[1]

Studies of Metacontrast in Dyslexia

Two studies have investigated metacontrast in connection with dyslexia (Edwards et al., 1996; Williams et al., 1989). Both studies found some reduction in the masking effect in dyslexic readers as compared with controls. This has been interpreted as evidence for a deficit in the magnocellular system.

The two studies differ with regard to which SOA gives the maximum masking. In the study of Williams et al. (1989), the maximum effect was found for SOA values of around 10–20 msec, whereas, in the report of Edwards et al. (1996), the maximum effect occurred at around 60 msec. The first value (i.e., 10–20 msec) is rather low. The second value (i.e., 60 msec) is very close to the center of the typical range (i.e., 30–100 msec), as identified by Lefton (1973).

Discussion and Conclusions

The purpose of the present report is to evaluate the appropriateness of metacontrast as a test for magnocellular deficits. As was pointed out above, the SOA at which maximum metacontrast masking occurs is typically much larger than the latency difference between the magnocellular and parvocellular systems at the entry to the visual cortex. No doubt there are cases that differ from the average. Nowak et al. (1995) measured neuronal latency differences as large as 20 msec in V1. Also, the SOA for maximum metacontrast masking may be as small as 20 msec, as in the study of Williams et al. (1989). However, even if maximum metacontrast masking can be obtained with an SOA of 20 msec under some conditions, under most conditions, the SOA for maximum effect is much larger. If one wishes to be able to account for all instances of metacontrast on the basis of a single principle, this principle will have to be able to account for maximum masking effects at SOAs of at least 30–100 msec and probably even larger. It appears very difficult to do this on the basis of the difference in latency between the magnocellular and parvocellular systems.[2]

It may be argued that the latency in metacontrast does not reflect the latency difference between the subcortical magnocellular and parvocellular systems themselves, but rather between two subsequent cortical streams,

which respectively receive their inputs from these two systems. In this case, the bulk of the latency difference would be generated cortically but in such a manner as to be correlated with the subcortical magnocellular and parvocellular systems. Though this is a possibility, it is not unproblematic. One problem stems from the large degree of mixing of magnocellular and parvocellular input that takes place in V1 (Ferrera, Nealey, & Maunsell, 1994; Malpelli et al., 1981; Merigan & Maunsell, 1993; Nealey & Maunsell, 1994; Sawatari & Callaway, 1996).[3] Beyond V1, there are two main cortical streams: the parietal and temporal streams (Ungerleider & Mishkin, 1982). This might suggest that it would be possible for the relevant latency difference to be between these two streams. However, studies of neuronal responses following magnocellular or parvocellular blockage have revealed some degree of mixing in both of these streams. It appears that the input to the parietal stream (as determined in area MT) is largely from magnocellular neurons (Maunsell, Nealey, & DePriest, 1990; Merigan & Maunsell, 1993).[4] On the other hand, the temporal stream appears to receive approximately equally strong magnocellular and parvocellular inputs (Ferrera et al., 1994). It is therefore difficult to map directly the subcortical magnocellular and parvocellular pathways onto the cortical parietal and temporal streams. However, it may be argued that only some degree of correlation between the subcortical pathways and the cortical streams, rather than a precise one-to-one mapping, is all that is required, in which case, extrastriate latencies would be relevant. However, it would seem that the latency differences are still too small. The latency difference between V1 and MT (i.e., V5) is about 10 msec (Maunsell, 1987; Raiguel, Lagae, Gulyàs, & Orban, 1989), and the one between V1 and V4 is about 22 msec (Maunsell, 1987).[5] That is to say, only a 12-msec difference, which is still too short to account for SOAs in metacontrast of 65 msec (even if one added a 20-msec subcortical difference to the 12 msec). It seems therefore doubtful that one will be able to account for sufficiently large latency differences on the basis of the magno- and parvocellular systems, even if extrastriate streams were included.

In conclusion, the present overview shows that, owing to the latencies involved, it is difficult to account for metacontrast masking in terms of the magnocellular and parvocellular systems. It seems, therefore, that reduced metacontrast masking should not be taken as evidence for reduced magnocellular sensitivity and that, in the case of dyslexic readers, it should not be taken as evidence for a magnocellular deficit.

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NOTES

1. This does not mean that there are no large time differences in the visual system but only that these differences cannot be linked unequivocally to the difference between magnocellular and parvocellular systems.

2. The latency difference between magno- and parvocellular systems depends on luminance level. It should therefore be pointed out that the luminance levels used by Williams et al. (1989; 2.5–3.5 cd/m^2) and by Edwards et al. (1996) (background luminance of 3.0 cd/m^2) are comparable to a luminance level (3.4 cd/m^2) at which Maunsell et al. (1999) found only a very slight difference in latency.

Purushothaman, Ögmen, and Bedell (2000) recently proposed a model for metacontrast based on networks of cells rather than on single neurons. In this model, responses to the target stimulus initiate rapid and ongoing oscillations in networks of sustained neurons. The masking stimulus, on the other hand, activates groups of transient cells, which in turn inhibit the sustained neurons. This creates multimodal metacontrast masking because the masking effect varies (with SOA) as the transient inhibition comes into and goes out of phase with the oscillations in the sustained neurons. Although this model may account for masking of long duration, the SOA to the first local maximum would still have to reflect the latency difference between transient and sustained neurons (see Figure 3A of Purushothaman et al., 2000). The latency of this first maximum was found to be (see their Figures 5, 6, 8, 11, and 12) between 40 and 75 msec, which is too long to be accounted for on the basis of differences between magnocellular and parvocellular neurons. (A puzzling feature of this model is that it hypothesizes that oscillations are a characteristic of late responses in sustained neurons. This appears to conflict with the observations of Maunsell and Gibson, 1992, who found that rapid oscillations, in V1 at least, are associated mainly with early responses in neurons with short latencies.)

3. Although one clearly can find V1 neurons that receive pure magnocellular or pure parvocellular inputs (Malpelli et al., 1981), it is not clear to what extent they are associated with the input to V1 or the output from V1. Therefore these findings should not be taken as evidence for pure magnocellular or parvocellular streams beyond V1.

4. This is somewhat puzzling given that the input to MT from V1 is from complex cells (Movshon & Newsome, 1996), of which only 12% receive pure magnocellular input according to Malpelli et al. (1981).

5. As has been pointed out by Nowak and Bullier (1997), this difference may be accounted for by the fact that the connection between V1 and MT is monosynaptic, whereas the one between V1 and V4 is bisynaptic.

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