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The need to differentiate the magnocellular system from the dorsal stream in connection with dyslexia

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ABSTRACT

A number of authors have postulated a "magnocellular-dorsal stream" deficit in dyslexia. Combining the magnocellular system and the dorsal stream into a single entity in this context faces the problem that contrast sensitivity data do not point to a magnocellular deficiency linked to dyslexia, while, on the other hand, motion perception data are largely consistent with a dorsal stream dysfunction. Thus, there are data both for and against a "magnocellular-dorsal stream" deficit in connection with dyslexia. It is here pointed out that this inconsistency is abolished once it is recognized that the magnocellular system and the dorsal stream are separate entities.

A number of authors have written of a "magnocellular-dorsal stream" deficit (or "magnocellular/dorsal stream" deficit) in connection with dyslexia (Boden & Giaschi, 2007; Facoetti, Corradi, Ruffino, Gori, & Zorzi, 2010; Gori, Cecchini, Bigoni, Molteni, & Facoetti, 2014; Gori & Facoeatti, 2014; Jednoróg, Gawron, Marchewka, Heim, & Grabowska, 2014; Jednoróg, Marchewka, Tacikowski, Heim, & Grabowska, 2011; Laycock, Crewther, Fitzgerals, & Crewther, 2009; Pammer, 2014; Ruffino, Gori, Boccardi, Massimo Molteni, & Facoetti, 2014; Zhao, Qian, Bi, & Coltheart, 2014) and Stein (2014) wrote of a magnocellular impairment throughout the dorsal visuomotor "where" pathway forward from the visual cortex. Also, Goswami (2015) wrote that "The dorsal pathway... encompasses the subcortical magnocellular system". In some papers the "magnocellular-dorsal stream" is contrasted with a "parvocellular-ventral stream" (Gori et al., 2014; Gori et al., in press; Zhao et al., 2014).

In primates the magnocellular system, along with the parvocellular and koniocellular systems, stretches from the retina to the input layers of the primary visual cortex, i.e. to Area V1 (Schiller & Logothetis, 1990; Shapley & Perry, 1986). Inside the visual cortex there is considerable mixing of the inputs from the different subcortical systems (Lachica, Beck, & Casagrande, 1992; Levitt, Yoshioka, & Lund, 1994; Martin, 1992; Merigan & Maunsell, 1993; Nealey & Maunsell, 1994; Sawatari & Callaway, 1996; Sincich & Horton, 2002; Vidyasagar, Kulikowski, Lipnicki, & Dreher, 2002). Onwards from the primary visual cortex it is possible to trace two processing steams each consisting of several cortical areas (Merigan & Maunsell, 1993). These are referred to as the dorsal and ventral streams. It has been proposed that these two streams are the continuations of, respectively, the subcortical magno- and parvocellular systems (Livingstone & Hubel, 1988).

One of the problems of linking the magnocellular system with the dorsal stream is that the dorsal stream (as determined in Area MT/V5) in addition to receiving substantial input from the magnocellular system (Maunsell, Nealey, & DePriest, 1990) also receives input from the parvocellular system (Nassi, Lyon, & Callaway, 2006) as well as from the koniocellular system (Sincich, Park, Wohlengemuth, & Horton, 2004). Also, lesions placed in the magnocellular system have quite different effects from those in the dorsal stream (Merigan & Maunsell, 1993; Rudolph & Pasternak, 1999) and response characteristics of cells in the dorsal stream are quite different from those in the magnocellular system. For instance, the contrast-response characteristics of cells in the magnocellular system are different from those of the dorsal stream (Sclar, Maunsell, & Lennie, 1990; Skottun, 2014). For these reasons it is difficult to portray the dorsal stream as the continuation of the magnocellular system or to portray the two as one system with one set of response characteristics.

In the case of merging the parvocellular system and the ventral stream, as Gori et al. (2014), Gori et al. (in press) and Zhao et al. (2014) have done, the main problem is that the ventral stream receives about equally potent inputs from the magno- and parvocellular systems (Ferrera, Nealey, & Maunsell, 1994).

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All studies that have examined contrast sensitivity following lesioning of the magno- and parvocellular layers of the monkey Lateral Geniculate Nucleus (LGN) have found that magnocellular lesions cause reduced sensitivity to low spatial frequencies and high temporal frequencies (Merigan, 1989; Merigan, Byrne, & Maunsell, 1991; Merigan, Katz, & Maunsell, 1991; Merigan & Maunsell, 1990; Merigan & Maunsell, 1993; Schiller, Logothetis, & Charles, 1990a; Schiller, Logothetis, & Charles, 1990b). Also, human psychophysics has indicated that low (below about 1.5 cycles/deg. See Skottun, 2000) and high spatial frequency stimuli are detected by mechanisms with different temporal characteristics (e.g., Legge, 1978; Tolhurst, 1975). Since there are no other known abnormalities which cause a contrast sensitivity deficit confined to low spatial frequencies such a deficiency is a clear and specific indicator of a magnocellular deficit.[1]

Evidence for low spatial frequency deficits in dyslexia is not compelling. When reviewed in 2000 (Skottun, 2000) it was found that in the case of dyslexia the number of studies that had found such deficits was lower than both the number of studies that found no contrast sensitivity deficits and the number of studies that found deficits of a nature incompatible with a magnocellular deficiency. This finding has been further strengthened by additional and more recent research (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Birch & Chase, 2004; Ramus et al., 2003; Roach & Hogben, 2004; Spinelli et al., 1997; Stuart, McAnally, & Castles, 2001; Williams, Stuart, Castles, & McAnally, 2003. For the case of Birch & Chase, 2004, see Skottun & Skoyles, 2005. In addition, Roach & Hogben, 2007, did not find a statistically significant difference between dyslexic subjects and controls). Also, Main et al., 2014, did not find a correlation between contrast thresholds for fast drifting (38 cycles/s), low spatial frequency (0.5 cycles/deg) stimuli and reading rate. These results should not be taken to mean that there may not be any dyslexic individuals who have a weakness in the magnocellular system. That is clearly possible.

For instance, it may be that magnocellular deficiencies are related to certain sub-types of dyslexia. Borsting et al. (1996), Ridder, Borsting, Cooper, McNeal, and Huang (1997), and Slaghuis and Ryan (2006) have proposed that magnocellular deficits are linked to dyslexia of dysphoneidetic sub-type. However, the data are not compelling. Borsting et al. (1996) found contrast sensitivity deficit which, although they were somewhat larger at the lowest spatial frequencies, afflicted all spatial frequencies tested (all the way up to 12.0 c/deg. See their Fig. 2b). Also, the high-spatial frequency deficits were larger when tested with 10 Hz modulation than with a modulation of 1 Hz (compare Fig. 2a and 2b of Borsting et al., 1996). Ridder et al. (1997) found the contrast sensitivity deficits for the subjects with dysphoneidetic dyslexia to be essentially independent of temporal frequency (see their Fig. 1). In the case of Slaghuis and Ryan (2006) who also proposed that magnocellular deficits were, in addition to the dysphoneidetic sub-type, linked to dyslexia of mixed sub-type, the claim faces the problem that the subjects of both of these subtypes showed large sensitivity reductions at combinations of moderate and high spatial frequencies and low temporal frequencies (see their Fig. 2. For a fuller discussion of Slaghuis & Ryan, 2006, see Skottun & Skoyles, 2007a). All these results are not what would be expected for a magnocellular deficit. Williams et al. (2003) divided the dyslexic subjects in their study into the sub-types: phonological dyslexia, surface dyslexia and dyslexia of mixed sub-type. They found no support of magnocellular deficits linked to any of these sub-types. In the present context it may be worth pointing out that Borsting et al. (1996) and Slaghuis and Ryan (2006) used moving stimuli. In the case of Slaghuis and Ryan (2006) subjects had to indicate the direction of motion. This may have confounded contrast sensitivity with motion perception. This may be significant here since the former may reflect magnocellular factors and the latter may reflect conditions in the dorsal stream. For all these reasons it is fundamentally unclear if, or to what extent, magnocellular deficits may be linked to specific sub-types of dyslexia. What seems clear, however, is that a magnocellular deficit can be ruled out as a general characteristic of dyslexia. (See also, Lueder et al., 2009, and Handler & Fierson, 2011.)

That the lack of support for a magnocellular deficit is not the result of insufficient test sensitivity is indicated by the fact that several studies have found positive evidence for deficiencies which are not compatible with a magnocellular deficit. For instance, Gross-Glenn et al. (1995) found clear deficiencies at high spatial frequencies (12 cycles/deg) but little evidence for a deficiency at low frequencies (0.6 cycles/deg). It seems quite clear that such a finding cannot be reconciled with a magnocellular deficiency by assuming a lack of test sensitivity.[2]

While there is little evidence for contrast sensitivity deficits indicative of a magnocellular deficiency there is evidence for deficits in motion perception in connection with dyslexia (Conlon, Lilleskaret, Wright, & Stuksrud, 2013; Conlon, Sanders, & Wright, 2009; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Demb, Boynton, Best, & Heeger, 1998; Everatt, Bradshaw, & Hibbard, 1999; Felmingham & Jakobson, 1995; Gori et al., 2014; Graves, Frerichs, & Cook, 1999; Hansen, Stein, Orde, Winter, & Talcott, 2001; Kubova, Kuba, Peregrin, & Novakova, 1996; Pellicano & Gibson, 2008; Qian & Bi, 2014; Ridder, Borsting, & Banton, 2001; Roach & Hogben, 2007; Schulte-Korne, Bartling, Deimel, & Remschmidt, 2004; Slaghuis & Ryan, 1999; Talcott, Hansen, Assoku, & Stein, 2000; Wilmer, Richardson, Chen, & Stein, 2004; Witton et al., 1998) or in connection with poor reading ability (Sperling, Lu, Manis, & Seidenberg, 2006; Talcott et al., 2003). Also Cornelissen, Hansen, Hutton, Evangelinou, and Stein (1998) found motion detection to be correlated with single word reading, and, most recently, Main et al. (2014) found speed discrimination to be correlated with reading ability. (It should also be pointed out that some studies have not found evidence for motion perception deficits linked to dyslexia, e.g., Bednarek, Saladane, & Garcia, 2009; Kiely, Crewther, & Crewther, 2001; Kronbichler, Hutzler, & Wimmer, 2002; Laycock, Crewther, Kiely, & Crewther, 2006; Roach & Hogben, 2004; Taroyan, Nicolson, & Buckley, 2011; Vanni, Uusitalo, Kiesila, & Hari, 1997. See also Sperling et al., 2006)

Physiological studies have linked motion perception to the dorsal cortical stream, particularly to Area MT (Britten, Shadlen, Newsome, & Movshon, 1992; Newsome, Britten, & Movshon, 1989). Studies that have involved placing lesions in the dorsal stream of monkeys (Areas MT and MST) have given deficient motion perception but relatively unaffected contrast sensitivity

[1] Even though there is compelling evidence that the magno- and parvocellular system determine contrast thresholds for low and high spatial frequency stimuli, respectively, it does not follow that they also mediate perception of low- and high spatial frequency stimuli at supra-threshold contrasts. One reason for caution in this regard is that Blakemore and Vital-Durand (1986) found no difference in spatial resolution between the systems when eccentricity was taken account of. Also, Spear, Moore, Kim, Xue, and Tumosa (1994) did not find a statistically significant difference in spatial frequency between the magno- and parvocellular systems. For a discussion see Skottun & Skoyles, 2008.

[2] It has been suggested that these results may be consistent with a magnocellular deficiency due to the brief duration of the stimuli which, it has been proposed, should have meant that these stimuli are associated with high temporal frequencies (Stein, 2014). Specifically, it was suggested (Stein, 2014) that stimuli of 17 ms and 34 ms duration respectively have amplitudes at 59 and 29 Hz and that these could drive the magnocellular system, The fact is that a stimulus of 17 ms duration has essentially zero amplitude at 59 Hz and a stimulus of 34 ms duration has essentially zero amplitude at 29 Hz. Most importantly, the stimuli in the study of Gross-Glenn et al. (1995) have their largest amplitudes at the lowest temporal frequencies including at 0 Hz (see Fig. 6 of Gross-Glenn et al., 1995).