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Letter to the Editor

Some Remarks on the Magnocellular Deficit Theory of Dyslexia

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The data bearing on the magnocellular or transient system deficit theory of dyslexia (Lovegrove *et al.*, 1986, 1990; Livingstone *et al.*, 1991) are highly conflicting. While some studies have produced results compatible with this theory (e.g. Lovegrove *et al.*, 1982; Livingstone *et al.*, 1991) other studies have yielded seemingly incompatible results (e.g. Smith *et al.*, 1986; Victor *et al.*, 1993; Gross-Glenn *et al.*, 1995). It is therefore commendable that researchers have started to investigate the possible causes for these conflicting results. Borsting *et al.* (1996) have produced evidence that magnocellular deficits may be confined to only one subgroup of dyslexic individuals. Cornelissen *et al.* (1995) have presented evidence to indicate that the contrast sensitivity loss predicted by the magnocellular/transient system deficit theory can be observed in dyslexic individuals at low but not at high luminance levels. Although the empirical findings of Cornelissen *et al.* (1995) and Borsting *et al.* (1996) could account for some of the discrepancies in the data, the magnocellular/transient system deficit theory faces substantial problems which have yet to be resolved. There are two specific issues which seem particularly important for future research to address.

The first issue pertains to the many empirical reports of contrast sensitivity loss in dyslexia at high spatial-frequencies. The magnocellular/transient system deficit theory of dyslexia predicts contrast sensitivity deficits associated with dyslexia at low spatial frequencies. The findings of Borsting *et al.* (1996) that magnocellular deficits may be confined to only one subgroup of dyslexic readers may account for why some studies have found low frequency contrast sensitivity deficits while others have not found such deficits. However, the observations of Borsting *et al.* (1996) do not seem to be able to account for the many instances of dyslexic readers having reduced sensitivity only to high spatial-frequencies (Hill & Lovegrove, 1993; Gross-Glenn *et al.*, 1995), for general reductions in sensitivity which are most pronounced at high spatial-frequencies (Martin & Lovegrove, 1987, 1988), or for deficits which are strongest at medium frequencies (i.e. at 4 c/deg) and less severe at both higher and lower spatial-frequencies (Lovegrove *et al.*, 1980). Most of these observations suggest, if anything, a parvocellular deficit rather than either a magnocellular deficit or the absence of a magnocellular deficit. Like the observations of Borsting *et al.* (1996), the findings of Cornelissen *et al.* (1995) that low frequency deficits are absent at high luminance levels do not by themselves account for the many reports of contrast sensitivity deficits at high and medium spatial frequencies.

The second issue is with regard to the magnocellular/transient system deficit theory itself. In the course of reading one makes a series of brief fixations interspersed with small saccades. According to the magnocellular/transient system deficit theory (Lovegrove *et al.*, 1986, 1990; Livingstone *et al.*, 1991; Breitmeyer, 1993; Lovegrove, 1993; Borsting *et al.*, 1996) the actual detection and identification of the printed characters and words is carried out by the parvocellular/sustained system during the fixations. The postulated role of the magnocellular/transient system is to inhibit the parvocellular/sustained system during each saccade. The function of this inhibition is thought to be to prevent parvocellular activity generated during one fixation from lingering into the next. However, recent results (Burr *et al.*, 1994) indicate that suppression associated with saccades in normal persons is confined within the magnocellular system and does not target the parvocellular system as required by the magnocellular/transient system deficit theory. It would therefore seem that an essential premise of this theory is lacking.

The work of Borsting *et al.* (1996) and Cornelissen *et al.* (1995) is welcome in that it provides recognition of some of the problems facing the magnocellular/transient system deficit theory of dyslexia. However, some important issues have yet to be resolved. I have tried to draw attention to two issues which seem particularly significant.

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