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Comment on letter to the editor: magnocellular reading and dyslexia

In his letter to the editor, Skottun (2003) emphasized two different questions about magnocellular reading and dyslexia: (1) does the magnocellular (M) pathway have a role in reading; and (2) does a M-deficit causes developmental dyslexia? We examined the first question—can the magnocellular pathway read?

This question takes on a special importance in the study of developmental dyslexia. Some researchers have expressed doubts about whether dyslexics have M-deficits (Ramus, 2003; Skottun, 1997, 2000a; Snowling, 2001). A few have gone further to argue that even if dyslexics do have M-deficits, such impairments could not be the cause of their reading problems because the M-pathway does not read, or as Skottun (2000b) wrote "The notion that a magnocellular deficit should cause reading problems is highly counterintuitive."

We believe it is important to resolve this issue. If the M-pathway has no role in text processing, then a dyslexic M-deficit cannot cause a reading problem. However, if the M-pathway contributes to reading, then the possibility of dyslexic M-deficits should be studied more intensively. Until we have a better understanding of the role of the M-pathway in text perception, it will be difficult to determine how a dyslexic M-deficit might interfere with reading development.

Our paper (Chase, Ashourzadeh, Kelly, Monfette, & Kinsey, 2003) provides evidence that the M-pathway contributes to text perception. In his response to our work, Skottun (2003) appears to have modified his earlier position when he writes "deficits in the magnocellular system could have the potential to cause reading problems…", so perhaps we are not too far apart on this issue.

If we can agree that the M-pathway does have a role in reading, then we could focus on questions of causation: (1) what information about text does the M-pathway provide; and (2) are some developmental dyslexics trying to learn to read with this information missing or distorted in some way? Too many studies look at dyslexic M-pathway function without any consideration for how the tasks and stimuli used relate to the process of text perception.

Some studies do not find a significant number of dyslexics with M-deficits (e.g., Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; but see also Chase & Stein, 2003; Ramus et al., 2003). Certainly, dyslexics without M-deficits have other conditions, such as phonological weaknesses (Snowling, 2000), that impair reading development. However, many other dyslexic studies have found M-deficits (Demb, Boynton, Best, & Heeger, 1998; Demb, Boynton, & Heeger, 1998; Eden et al., 1996; Talcott et al., 2002), including some that show strong correlations with reading performance (Chase, Dougherty, Ray, Fowler, & Stein, submitted for publication; Sperling, Lu, Manis, & Seidenberg, 2003; Talcott et al., 2000). Several M-deficit models of these findings have been proposed (Chase, 1996; Cornelissen et al., 1998; Stein & Walsh, 1997; Stein, 2003), hypothesizing a role for the M-pathway in the perception of global letter patterns, letter location, and general visual temporal processing. These ideas need to be explored further, particularly with respect to dyslexic perceptual performance and how such skills may affect reading development (e.g., Habib, 2003).

Conflicting results often occur in studies of developmental neuropathology because of the variety of neurological malformations that are produced during neurogenesis (Courchesne, Townsend, & Chase, 1995). Dyslexia is caused by factors that disrupt neural migration in fetal development (Galaburda, 1994). These migration errors produce multiple cortical and subcortical abnormalities that are unique to each dyslexic individual (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985), can disrupt development in many different locations, and affect the different cognitive and perceptual functions necessary for reading in diverse ways. Some dyslexics have subcortical abnormalities that affect M-pathway functions (Livingstone, Rosen, Drislane, & Galaburda, 1991). From a neuropsychological perspective, development dyslexia has multiple causes. One may be a M-deficit subtype.

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Christopher Chase *
Arash Ashourzadeh
Charlotte Kelly
Department of Psychology
Claremont McKenna College
850 Columbia Avenue
Claremont, CA 91711, USA

Sharon Monfette
Department of Psychology
Mt. Holyoke College
50 College St., South Hadley
MA 01075, USA

Kristofer Kinsey
University Laboratory of Physiology
Parks Rd., Oxford
OX13PT, UK

* Corresponding author.
E-mail address: chris.chase@mckenna.edu (C. Chase).