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M-Stream Deficits and Reading-Related Visual Processes in Developmental Dyslexia

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Some visual processing deficits in developmental dyslexia have been attributed to abnormalities in the subcortical M stream and/or the cortical dorsal stream of the visual pathways. The nature of the relationship between these visual deficits and reading is unknown. The purpose of the present article was to characterize reading-related perceptual processes that may link the visual deficits to reading problems. We identified contrast sensitivity, position encoding, oculomotor control, visual attention, parafoveal/foveal interactions, and saccadic suppression as potential reading-related dorsal stream processes. We then evaluated the role of each process in reading and the status of each process in dyslexia. In theory, a number of dorsal stream processes (e.g., oculomotor control and visual attention) might contribute to reading problems in developmental dyslexia. More work is needed to demonstrate the connection empirically.

Keywords: dyslexia, reading, visual deficits, M stream, dorsal stream

Developmental dyslexia is a learning disability in which children have difficulty learning to read. Historically, dyslexia was seen as a basic deficit in visual processing, and early theories focused on phenomena such as letter reversals (i.e., reading "b" as "d") and mirror writing ("gnitirw rorrim") as the root of the problem (Orton, 1925). Subsequent research showed that children with dyslexia are not unusually prone to letter reversals and mirror writing, and it became widely accepted that visual processing is normal in developmental dyslexia (e.g., Hulme, 1988; Shaywitz, 1996; Vellutino, 1987). It is now a common belief that the main problem is one of phonological encoding and decoding (Hurford, Schauf, Bunce, Blaich, & Moore, 1994; Korkman & Pesonen, 1994; Pennington, Groisser, & Welsh, 1993; Shaywitz, 1996; Snowling & Rack, 1991). Many people with dyslexia have difficulty blending sounds together and segmenting words into their component sounds (reviewed in Shaywitz & Shaywitz, 2005). There is also increasing evidence for additional core deficits in naming speed in dyslexia (reviewed in Bowers & Newby-Clark, 2002; Wolf et al., 2002).

A large body of more recent neuroscience literature shows that many people with developmental dyslexia also show low-level problems in visual processing (see Farmer & Klein, 1995; Klein, 2002, for reviews); however, in contrast to the letter-reversal processing deficit suggested by Orton (1925), this research points to low-level problems in visual–temporal information processing. Temporal information processing is a broad term that includes temporal resolution of individual stimuli, temporal order judgments, temporal sequence matching, and perception of flicker and motion. These visual problems appear to arise from abnormalities in a part of the visual pathway known as the M stream (reviewed in Stein, 2001). It remains to be shown empirically, however, that the M stream is involved in normal reading and that M-stream visual deficits contribute to reading problems. The purpose of this article is to examine several hypotheses about the role of visual processes in normal reading, such as contrast sensitivity, position encoding, eye movements, and visual attention, based on the visual deficits seen in developmental dyslexia.

Developmental Dyslexia

Children who have difficulty learning to read, but are progressing at a rate commensurate with their general progress, have been referred to as "backward" or "poor" readers. In contrast, a reading disability is diagnosed if the individual is reading at a level that is significantly below the expected level (often based on general intellect) in the absence of other factors such as severe emotional, sensory, or neurological problems, and/or inadequate educational opportunities. This type of learning disability is known as *specific reading disability* or *developmental dyslexia* (Rayner & Pollatsek, 1989). Discrepancy definitions of dyslexia (i.e., discrepancy between reading achievement and performance expected on the basis of IQ, age, or educational level) are not universally accepted (Siegel, 1992), but they are the definitions most often used in the literature. As the visual attributes of dyslexia are discussed in this article, one should keep in mind that children have most likely been selected by discrepancy definition criteria and do not include poor readers. A discrepancy definition implicitly assumes that the

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causal mechanism is specific to people categorized as dyslexic. Alternatively, similar deficits may underlie the performance of dyslexic and poor readers (Siegel, 1992). For example, there is some evidence that poor readers and dyslexic readers perform similarly on visual tasks (e.g. Eden, Stein, Wood, & Wood, 1994). This article does not address this issue further.

Developmental reading disabilities are probably not a unitary disorder but may encompass a variety of deficits whose outcome is reading difficulties (Rayner & Pollatsek, 1989). For example, reading disabilities have been subtyped according to the ability to read pronounceable nonwords and irregular words such as *yacht* (Castles & Coltheart, 1993). This concept is based on a dual-route model of reading in which skilled readers use a lexical and a sublexical route. The lexical route is used to retrieve sound–meaning associations (similar to looking up a word in a dictionary). The sublexical route is used to assemble components of the word based on grapheme-to-phoneme correspondences (similar to sounding out the word). Nonwords are identified mainly by the sublexical route, whereas irregular words are identified mainly by the lexical route. In *phonological,* or *dysphonetic,* dyslexia, nonwords are the most difficult to read, possibly reflecting selective impairment of the sublexical route. In *surface,* or *dyseidetic,* dyslexia, irregular words are the most difficult to read, possibly reflecting selective impairment of the lexical route. In *dysphoneidetic* dyslexia, both nonwords and irregular words are difficult to read.

In another classification scheme, children with developmental dyslexia have been subtyped according to their reading speed and accuracy (Van Strien, Bouma, & Bakker, 1993). *Linguistic,* or *L-type,* dyslexia is characterized by relatively quick, but inaccurate, sentence reading and reading latencies that do not vary with word length. People with L-type dyslexia are thought to rely heavily on parallel processing of letter information in which all letters are processed at once and a word is directly mapped onto a stored visual representation. *Perceptual,* or *P-type,* dyslexia is characterized by slow, accurate reading and reading latencies that vary with word length. People with P-type dyslexia rely heavily on sequential, rather than parallel, processing of letter information. *Mixed,* or *M-type,* dyslexia shares characteristics of both P- and L-type dyslexia. Masutto, Bravar, and Fabbro (1994) found that children with L-type dyslexia made a larger number of reading errors, had low verbal short-term memory scores, and showed a right-ear advantage in dichotic listening. Children with P-type dyslexia made fewer reading errors, had a shorter attention span, obtained low scores on a measure of visuomotor coordination, and showed an absence of the right-ear advantage in dichotic listening.

These are just two examples of sub-typing schemes developed to clarify the etiologies of developmental dyslexia. Manis, Seidenberg, Doi, McBride-Chang, and Petersen (1996) argued that subtyping according to reading errors, as in Castles and Coltheart (1993), fails to fully capture the potential for multiple etiologies leading to the same pattern of reading errors. Comprehensive examination of the perceptual processes underlying reading errors is necessary. We return to this point in a later section.

Deficits in phonological processing are a frequently examined factor in dyslexia. Very early language deficits in tasks such as rhyming have been demonstrated in children who later become dyslexic (Scarborough, 1990). There is, however, less agreement about other factors that may also play a role. Verbal memory problems, repetition deficits, slower naming speed (see Snowling, 1991, for a review), and rapid auditory processing deficits (Tallal, 1980) have been found, just to list a few. In addition, visual temporal processing problems have been demonstrated in a number of studies (see Farmer & Klein, 1995, for a review), although some argue that such cases are rare (Rayner, Pollatsek, & Bilsky, 1995). These visual problems appear to arise from abnormalities in part of the visual pathway known as the M stream. We now review the anatomy and physiology of the M stream.

The M Stream

The subcortical human visual system is thought to consist of at least two parallel pathways (Figure 1). These parallel pathways, known as the parvocellular or P stream and the magnocellular or M stream, begin at the retina with the small P and the larger M retinal ganglion cells (reviewed in Shapley, 1990). Axons from the M and P ganglion cells project to the magnocellular and parvocellular layers, respectively, of the dorsal lateral geniculate nucleus (LGN) of the thalamus. Neurons in these pathways have distinct physiological properties as well. P cells have small receptive fields; respond in a slow, sustained fashion; are sensitive to differences in wavelength; and prefer stimuli of high contrast and high spatial frequency. M cells have larger receptive fields; respond in a fast, transient fashion; have broadband wavelength sensitivity; prefer low spatial frequencies; and are quite sensitive to low-contrast stimuli (Shapley, 1990). Magnocellular and parvocellular neurons project to separate layers in the primary visual cortex, V1 (Baizer, Ungerleider, & Desimone, 1991; Livingstone & Hubel, 1988; Maunsell, Nealey, & De Priest, 1990). From here, there is a mingling of the M and P streams, but the anatomical segregation is at least partially maintained in the next visual area, V2 (DeYoe & Van Essen, 1988; Ferrera, Nealey, & Maunsell, 1992; Lennie, Trevarthen, Van Essen, & Wassle, 1990; Shapley, 1990).

The functional role of these M and P streams in visual perception has been extensively debated (see Lennie, 1993, for a review). Magnocellular (but not parvocellular) lesions of the macaque LGN impair flicker sensitivity and contrast sensitivity for grating patterns of low spatial frequency and high temporal frequency (Merigan, Byrne, & Maunsell, 1991; Merigan & Maunsell, 1990). Parvocellular (but not magnocellular) lesions impair color and texture discrimination (Shapley, 1990). There appears to be general agreement that the M stream is involved in the processing of temporal change and low-contrast information and is tuned to low spatial frequencies. The P stream is involved in the processing of chromatic information and is tuned to low temporal and high spatial frequencies. There is, evidence, however, that information contributing to direction and speed discrimination may be carried by both the P and M streams (Merigan, Byrne, & Maunsell, 1991).

Additional processing streams have been identified in the visual cortex (Mishkin & Ungerleider, 1982). The dorsal stream, which connects V1 to the posterior parietal lobe, has been implicated in object localization, motion perception, and goal-directed movements. In addition, the dorsal stream appears to mediate selective visual attention (Posner, 1995). The ventral stream, which connects V1 to the inferotemporal region, has been implicated in object recognition (reviewed in Goodale & Milner, 1992). Area V4 in the ventral stream receives input from both the M and P streams (Maunsell et al., 1990). This area is specialized for color and form processing.

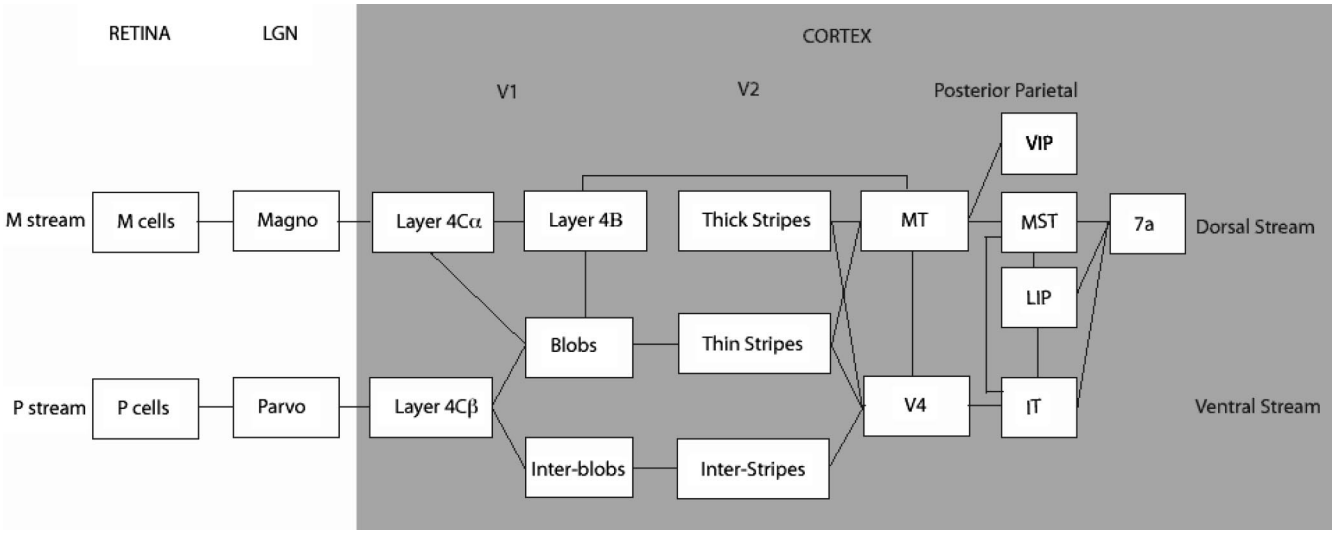

Figure 1. Anatomy of parallel pathways in the monkey visual system. LGN = lateral geniculate nucleus of the thalamus; V1 = cortical visual area 1; V2 = cortical visual area 2; V4 = cortical visual area 4; M = magnocellular; P = parvocellular; MT = medial temporal area; VIP = ventral intraparietal lobe; MST = medial superior temporal area; LIP = lateral intraparietal cortex; IT = inferior temporal cortex; 7a = area 7a of the parietal cortex.

The predominant anatomical projection from V1 to area V5 (also known as the medial temporal area [MT]) in the dorsal stream arises from the subcortical M stream (DeYoe & Van Essen, 1988; Maunsell & Newsome, 1987). Responses in MT are reduced or eliminated when responses from the magnocellular (but not the parvocellular) layers of the LGN are temporarily blocked (Maunsell et al., 1990). Neurons in MT are specialized for stimulus motion. Direct lesions to MT in monkeys produce elevated motion coherence thresholds (Newsome & Pare, 1988), and deficits in velocity discrimination and detection of motion-defined form (Schiller, 1993). MT also plays a role in maintaining pursuit eye movements, probably by providing information about target velocity (Keller & Heinen, 1991). Lesions to MT produce retinotopic deficits in the initiation of pursuit eye movements. These deficits disappear after several days, which suggests that MT is not essential for the production of pursuit eye movements.

Human lesion studies concur, for the most part, with the findings of monkey lesion studies. Patients with lesions to parietotemporal regions are impaired on global motion tasks, substitute saccades for smooth pursuit when tracking moving objects (Vaina, Lemay, Bienfang, Choi, & Nakayama, 1990), and have elevated speed thresholds for motion-defined letters (Regan, Giaschi, Sharpe, & Hong, 1992). Unilateral lesions to posterior parietal cortex affect the ability to disengage attentional focus to move to a target in the visual field contralateral to the lesion (Posner, Walker, Friedrich, & Rafal, 1984).

With neuroimaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI), it is possible to see which areas of the brain are most active while a human observer is performing a task. PET studies indicate that the temporo-parieto-occipital junction (human homologue of area MT) is activated when the observer is presented with moving dot stimuli (Cheng, Fujita, Kanno, Miura, & Tanaka, 1995; Zeki et al., 1991), but only when the dots are moving coherently (Cheng et al., 1995). Studies conducted with fMRI have found that MT is activated by translational or rotational motion (Buchel et al., 1998; Chawla, Phillips, Buechel, Edwards, & Friston, 1998; Haug, Baudewig, & Paulus, 1998; Howard et al., 1996; Tootell, Reppas, Kwong, et al., 1995) or illusory motion (Tootell, Reppas, Dale, et al., 1995). The degree of activation is modulated by attention (Buchel et al., 1998; Haug et al., 1998; O'Craven, Rosen, Kwong, Treisman, & Savoy, 1997), stimulus speed (Tootell, Reppas, Kwong, et al., 1995), and luminance contrast (Ffytche, Skidmore, & Zeki, 1995; Tootell, Reppas, Kwong, et al., 1995). Howard et al. (1996) noted fMRI activation to coherent motion, optic flow, and biological motion each in different but adjacent areas that overlap with MT. On the basis of the contrast gain response in MT relative to V1, Tootell, Reppas, Kwong, et al. (1995) argued that the properties of MT in humans are consistent with its receiving input from the M stream.

Taking anatomical, physiological, and behavioral properties into account, investigators have noted certain tasks that are particularly sensitive to M-stream/dorsal pathway integrity. These psychophysical tasks are flicker resolution; contrast sensitivity for low spatial frequency, high-temporal-frequency gratings; and direction and velocity discrimination for global motion stimuli. A deficit on any of these tasks cannot indicate the parts of the visual pathway involved. For this reason, the term *M-stream deficit* is used to refer to deficits arising from the functioning of the visual pathway at any level between the retina and the posterior parietal cortex.

M-Stream Deficits in Dyslexia

Anatomical evidence indicates that the cells in the magnocellular layers of the LGN are smaller and more disorganized in dyslexic brains relative to controls (Galaburda, 1993a, 1993b;

Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985). Visual evoked potentials (VEPs) to pattern-reversing stimuli measured over the occipital cortex of dyslexic subjects, are reduced in amplitude for low-contrast, high–temporal frequency stimuli (Galaburda & Livingstone, 1993; Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993). Motion-onset VEPs measured over occipital and parietotemporal cortex show an increased latency and a reduced amplitude in dyslexic individuals (Kubova, Kuba, Peregrin, & Novakova, 1995). Schulte-Korne, Bartling, Deimel, and Remschmidt (2004) found similar results. fMRI studies have found either a lack of activation (Eden, VanMeter, Rumsey, & Zeffiro, 1996) or decreased activation relative to controls in area MT (Demb, Boynton, & Heeger, 1997, 1998). A magnetoencephalography (MEG) study found that moving gratings did activate MT in individuals with dyslexia (Vanni, Uusitalo, Kiesila, & Hari, 1997). The response latency, however, was slower in the dyslexic subjects.

Behavioral studies also generally support an M-stream deficit. The deficits include abnormal temporal gap detection for low-contrast and low–spatial frequency stimuli (Badcock & Lovegrove, 1981); reduced critical flicker frequency (Chase & Jenner, 1993); decreased contrast sensitivity for flickering and moving stimuli, particularly at low spatial frequencies (Edwards et al., 2004; Felmingham & Jakobson, 1995; Martin & Lovegrove, 1984, 1987; A. Mason, Cornelissen, Fowler, & Stein, 1993); reduced velocity discrimination (Demb et al., 1998; Eden et al., 1996); elevated coherence thresholds for global motion stimuli (Casco, 1993; Edwards et al., 2004; Everatt, Bradshaw & Hibbard, 1999; Raymond & Sorensen, 1998; Richardson, 1995); and elevated speed thresholds for motion-defined form (Felmingham & Jakobson, 1995).

Others have failed to find evidence of an M-stream deficit in developmental dyslexia (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Ben-Yehudah, Sackett, Malchi-Ginzberg, & Ahissar, 2001; Gross-Glenn et al., 1995; Johannes, Klussmaul, Munte, & Mangun, 1996; Ramus et al., 2003; Spinelli et al., 1997; Victor, Conte, Burton, & Nass, 1993; Walther-Muller, 1995; Williams, Stuart, Castles, & McAnally, 2003). It has also been argued that apparent M-stream deficits are due to deficits in perceptual noise exclusion (Sperling, Lu, Manis, & Seidenberg, 2005) or task-related factors (Ben-Yehudah & Ahissar, 2004; Ben-Yehudah et al., 2001) rather than an M-stream deficit per se. It is also possible that a developmental anomaly in the posterior parietal cortex could cause a primary attention deficit, leading to a top-down reduction in performance on M-stream tasks. Furthermore, Ramus (2004) reinterpreted neurobiological data to argue that a sensorimotor syndrome, of which an M-stream deficit is an example, may be associated with but is not essential to the development of dyslexia. Levels of prenatal hormones influence the likelihood that an ancillary sensorimotor deficit occurs with dyslexia. According to Ramus's view, M-stream deficits are present in only a subgroup of people and are ancillary to the basic underlying cause of the reading problem, namely, phonological deficits.

Implicit in dyslexia subtyping is the assumption that dyslexia actually encompasses a group of conditions with a similar outcome—reading disability—but different underlying causes. Study results, therefore, would be influenced by the composition of the particular dyslexic sample under investigation. For example, it has been reported that up to 75% of dyslexic observers show visual temporal processing deficits (Lovegrove, Martin & Slaghuis, 1986). (The participants in this study were severely disabled; thus, 75% may be an overestimation of the prevalence of temporal processing deficits according to the inclusion criteria for most studies; Klein, 2002.) It is not clear, however, whether the affected individuals fall into a specific reading subtype. Visual temporal processing deficits were originally reported to be more prevalent in dyslexic observers with phonological reading difficulties (Borsting et al., 1996; Cestnick & Coltheart, 1999; Lovegrove, McNicol, Martin, Mackenzie, & Pepper, 1989; Ridder, Borsting, Cooper, McNeel, & Huang, 1997; Slaghuis & Ryan, 1999). More recently, however, visual temporal processing deficits have been associated with orthographic reading deficits (Sperling, Lu, Manis, & Seidenberg, 2003) or with mixed reading deficits (Ridder, Borsting, & Banton, 2001). In addition, some of these differences that appeared to depend on reading subtype have been shown to be due to behavioral methodologies instead (Ben-Yehudah et al., 2001).

Skottun (2001) has questioned the M-stream hypothesis on two counts: (a) it is unclear whether the M stream is important to reading and (b) M-stream deficits, if they exist, may not cause the reading problems. These are valid questions that do not yet have complete answers. We do not yet know how the various pathways within the visual system contribute to a task as complex as reading. We believe that there is sufficient evidence of visual deficits associated with reading problems to warrant a comprehensive examination of the implications of such a deficit on reading. The purpose of the present article is to identify and investigate possible role(s) for the M stream in reading. Once the M-stream's contribution to reading is identified, it will be easier to evaluate a causative role for the M stream in reading problems such as dyslexia.

Reading and the M Stream

Reading achievement has been found to correlate with performance on visual psychophysical tasks, such as coherent motion detection (Cornelissen, Bradley, Fowler, & Stein, 1994; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998) and velocity discrimination (Demb et al., 1997, 1998). Reading achievement also correlates with fMRI activation produced by moving stimuli in MT (Demb et al., 1997; Eden et al., 1996) and V1 (Demb et al., 1997). Orthographic decoding, but not phonological processing, has been correlated with VEPs believed to estimate dorsal stream brain function (Kinsey, Hansen, & Chase, 2006). Preschool children who were genetically at risk for dyslexia showed coherent motion thresholds similar to those of age-matched children who were not at risk for dyslexia (Boets, Wouters, van Wieringen, & Ghesquiere, 2006). When the data for the two groups were combined, however, there was a significant correlation between coherent motion and orthographic processing, on the one hand, and between auditory frequency modulation and phonological processing on the other. Reading comprehension correlates with both perceptual speed and coherent motion performance in prelingually deaf children (Samar & Parasnis, 2005). Unfortunately, correlational methods cannot reveal the precise nature of the relationship between reading and the M stream.

Techniques that directly perturb the function of a specific brain region are a more direct method of evaluating the function of that region. Disruption of right posterior parietal cortex by transmag-

netic stimulation (TMS) was found to have a detrimental effect on reading (Braet & Humphreys, 2006). When adult proficient readers were required to name single and mixed case words as fast and accurately as they could, reading of both lowercase and mixed case (e.g. MiXeD) words was perturbed by TMS. The effect was greater on mixed case words. The authors proposed that the right parietal lobe, part of the dorsal stream, mediates the recognition of words in unfamiliar formats.

Thus, there is initial evidence supporting a relationship between M-stream integrity and reading proficiency, most notably for orthographic aspects of word recognition. The evidence regarding the relationship between M-stream performance and dyslexia is more tenuous. This apparent contradiction might occur if only a subset of people with dyslexia have a visual processing deficit and if this subset is too small to sway the correlation within any dyslexic sample. Alternatively, M-stream function may be closely tied to some aspects of reading, either directly or indirectly, but people with reading difficulties may not exhibit an M-stream deficit. Finally, the apparent relationship between M-stream function and reading may be spurious. As we discuss later in this article, there are several aspects of reading that can be linked to processes associated with M-stream regions of the brain, making this last possibility less likely as an explanation.

M-stream deficits are typically revealed on tasks involving flickering or moving stimuli of low contrast and low spatial frequency. The relationship between motion perception and everyday reading is not immediately obvious. This has led to skepticism about M-stream deficits and dyslexia (e.g., Hulme, 1988). Let us turn for the moment to the developing child. Not all aspects of visual function are fully mature in children when they begin school (e.g., eye movements, Hainline, 1988; form perception, Giaschi & Regan, 1997). Although the neural groundwork is present from birth, visual input is necessary for synaptic connections to be made and lost (Huttenlocher, 1994). It has been proposed that, during development, slight impairments at the level of the lateral geniculate nucleus (LGN) of the thalamus lead to larger impairments in brain areas that receive thalamic input (Stein, 1991; Stein & Walsh, 1997). Neural fibers from the magnocellular layers of the LGN connect to the posterior parietal cortex indirectly via primary visual cortex and then to MT (Livingstone & Hubel, 1987). Deficits in the subcortical M stream are proposed to have their impact, therefore, by impairing cortical function in the dorsal pathway (but see Chase, Ashourzadeh, Kelly, Monfette, & Kinsey, 2003). Whereas an M-stream deficit may manifest itself as a deficit in motion perception, other characteristics of the M stream, such as good contrast sensitivity and fast neural transmission speeds, are potentially more relevant to reading. In addition, deficient subcortical M-stream function may lead to altered dorsal stream processes, such as visual selective attention and eye movements. It is these latter functions that are an integral part of reading.

We suggest that subcortical M-stream input and cortical dorsal stream processing influence some cognitive and perceptual operations that make everyday reading possible (see Figure 2). A model of normal sentence reading is summarized below. Hypotheses about the influence of specific reading-related M-stream deficits at each stage in the reading model are presented. Finally, the evidence for each type of deficit in dyslexia is discussed. This analysis provides an index of the likelihood that dyslexia is associated with degradation of each reading-related M/dorsal stream function. There are then several logical and empirical connections

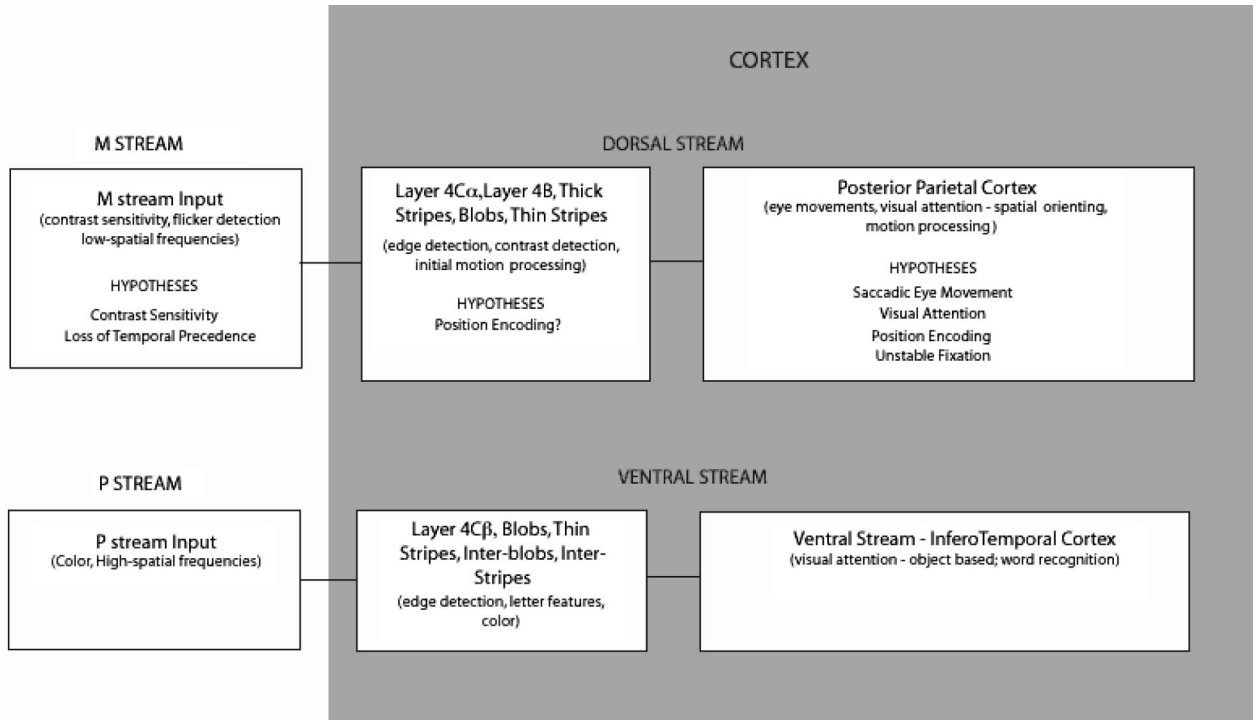

Figure 2. A schematic of the visual pathways and the hypothesized etiology of the reading deficit associated with damage to each region. M = magnocellular; P = parvocellular.

that must be made before one can say that M-stream deficits can contribute to reading problems. At present, these connections have not been made, but the means by which they might be evaluated are discussed below.

Seven Proposed Roles for the M Stream in Reading

Seven hypotheses about the role of the M stream in reading are presented in the context of one model of sentence reading. There are many models of word recognition (e.g., Coltheart, Curtis, Atkins, & Haller, 1993; Paap & Noel, 1991; Seidenberg, Plaut, Petersen, McClelland, & McRae, 1994), but most models begin after the features within words have already been identified (but see Whitney, 2001). The intent of such models is to elucidate higher level aspects of reading. They do not address the interplay between eye movements and the uptake of the featural-level information. Morrison's (1984) model, in which information processing and eye movements during reading are discussed, provides a framework for understanding hypotheses of M-stream function in reading.

According to Morrison's model (1984), when a word is first fixated, the attentional focus and the eyes are at the same spatial location. Detailed processing of the word takes place. Once processing has reached a certain criterion level, the attentional focus moves forward to the next word, although the eyes are still fixated at the same location. During the interval when the eyes are fixated on the first word and the attentional focus has moved to the next word $n + 1$, parafoveal processing of word $n + 1$ begins and saccadic programming takes place. Some information is available about word $n + 1$ before the eyes foveate it on account of preprocessing of the parafoveal information. This "parafoveal preview" presumably speeds reading time for the word and is called the parafoveal preview benefit. Some time later the eyes follow attention to the new location with a ballistic saccadic eye movement. The eyes and the attentional focus are once again at the same spatial location. If the word $n + 1$ is identified by parafoveal processing, this word might be skipped and the eyes would then saccade to word $n + 2$.

How might an M-stream deficit be manifested in reading? Even subtle M-stream deficits may disrupt reading at a number of points in this process (see Figure 3). Reduced contrast sensitivity at low spatial frequencies, due to a compromised M stream, could interfere with the visual analysis of the features that make up the word (Hypothesis 1; for a review of contrast sensitivity in reading, see Legge, 1993). Poor spatial localization due to an M-stream deficit, may lead to problems with position encoding of the letters within the word (Hypothesis 2; Cornelissen, Hansen, Gilchrist, et al., 1998; Cornelissen, Hansen, Hutton, et al., 1998). Unstable binocular fixation on the plane of the words, resulting from an unstable

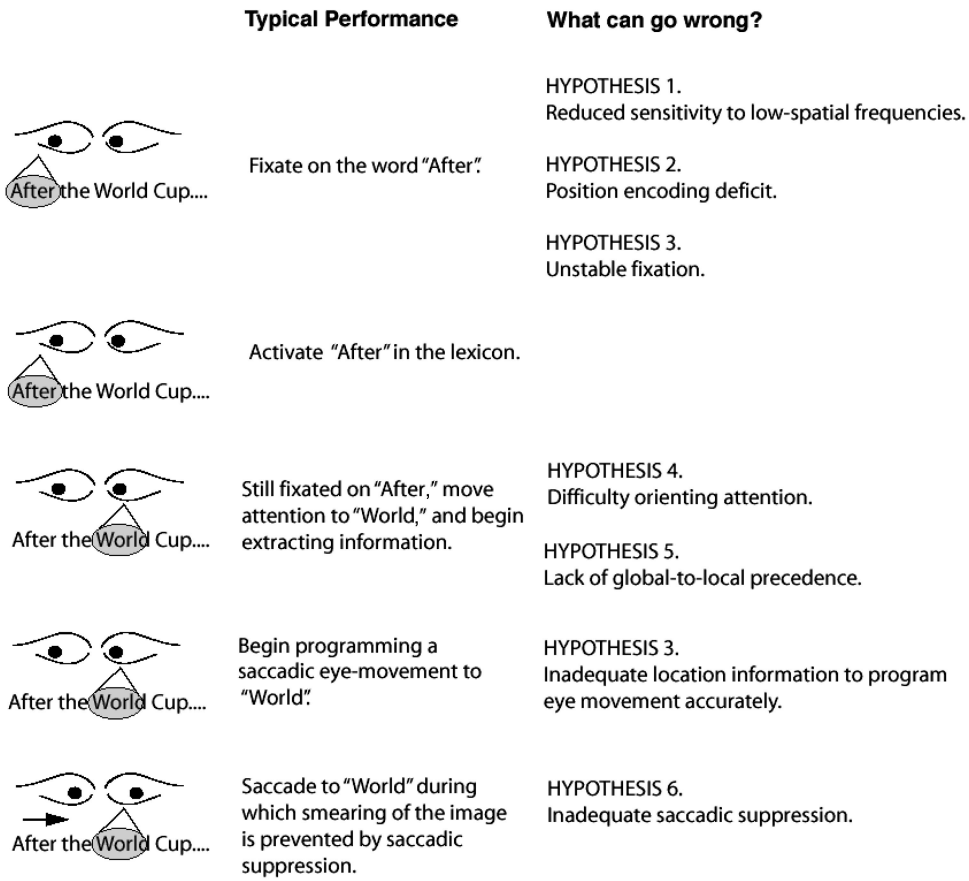

Figure 3. An overview of Morrison's (1984) model of reading and the hypotheses associated with each stage.

reference (dominant) eye or poor vergence control, may prevent or slow the uptake of features within the word (Hypothesis 3; Stein, 1991, 1994; Stein & Fowler, 1993; Stein, Riddell, & Fowler, 1988, 1989; Stein & Walsh, 1997). Imperfect posterior parietal lobe function, because of inadequate M-stream input, may affect attentional focusing on the fixated word and/or attentional orienting to the next word (Hypothesis 4; Steinman, Steinman, & Garzia, 1996, 1998; Stein & Walsh, 1997; Vidyasagar, 1999). A sluggish M stream may eliminate the temporal precedence of global information about parafoveal words (Hypothesis 5; Lovegrove et al., 1986; Steinman et al., 1996). Unreliable posterior parietal lobe processing of location information may lead to problems programming saccadic eye movements (Hypothesis 6; Pavlidis, 1981). Inadequate suppression of visual information during a saccade (saccadic suppression) could create smearing or superimposition of pre- and postsaccade images (Hypothesis 7; Breitmeyer, 1983, 1993). According to Breitmeyer, an M-stream deficit would compromise this process by removing the inhibition from transient neurons. Burr, Morrone, and Ross (1994), however, found that during saccades, the M stream was selectively suppressed, but the P stream was spared or even enhanced. This finding is problematic for Breitmeyer's theory. In addition, empirically testable consequences of an M-stream deficit are harder to pin down for this hypothesis. Moreover, it is difficult to find ways of supporting or refuting this theory from the available reading literature. Unfortunately that means that it cannot be evaluated as comprehensively as the other hypotheses in this article. Therefore, we do not discuss this theory further.

Contrast Sensitivity Deficits (Hypothesis 1)

Before the meaning or the sound of the word can be identified, visual analysis of the features that make up the word must take place. Contrast sensitivity is a laboratory measure of visual integrity based on the minimum amount of contrast necessary to detect patterns of different spatial and temporal frequencies (see Figure 4a). The M stream is preferentially sensitive to moving or flickering stimuli of low contrast and low spatial frequency (e.g., Merigan et al., 1991). The initial evidence for an M-stream deficit in dyslexia came from studies showing decreased contrast sensitivity for flickering and moving stimuli, particularly at low spatial frequencies (Edwards et al., 2004; Felmingham & Jakobson, 1995; Martin & Lovegrove, 1984, 1987; A. Mason et al., 1993). Contrast sensitivity has been shown to affect the perception of suprathreshold stimuli (e.g., De Valois & De Valois, 1993). Therefore, a contrast sensitivity deficit could produce a deficit at the high contrast levels of most reading material. Figure 4b shows how a word might look to someone with a contrast sensitivity deficit at low or high spatial frequencies.

Several studies, however, failed to find contrast sensitivity deficits in dyslexia (Ben-Yehudah et al., 2001; Williams et al., 2003); found deficits at high and low spatial frequencies (Olson & Datta, 2002); or found deficits at high but not low spatial frequencies (Gross-Glenn et al., 1995; Spinelli et al., 1997; reviewed in Skottun, 2000). These inconsistencies across studies have been attributed to luminance differences (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995), subject differences (Olson & Datta, 2002), or the temporal structure of the task (Ben-Yehudah & Ahissar, 2004).

During reading, transients are produced at a rate of approximately 4 Hz (Legge, 1993) as the eyes move across the text in a series of saccades and fixations. A rate this slow would stimulate the P stream as well as the M stream (Legge, 1993); therefore, it is the contrast and spatial frequency characteristics and not the temporal characteristics of the M stream that are relevant to the current hypothesis. An M-stream deficit would lead to poor contrast sensitivity and lower perceived contrast at low spatial frequencies. This, in turn, could interfere with the visual analysis of words. Several groups have examined interactions between contrast sensitivity and reading (Akutsu, Legge, Ross, & Schuebel, 1991; De Luca, Spinelli, & Zoccolotti, 1996; Legge, Ahn, Klitz, & Luebker, 1997).

Patients with spatial frequency–specific contrast sensitivity loss can provide insight into the importance of different spatial frequencies to reading. For example, patients with age-related contrast-sensitivity deficits at low spatial frequencies were slower at reading text written in large letters that contain low spatial frequencies but not text written in normal size letters that contain high spatial frequencies (Akutsu et al., 1991). Patients with central field loss producing contrast sensitivity deficits at high spatial frequencies had slower than average peak reading rates (Legge, Pelli, Rubin, & Schleske, 1984). Decreased contrast sensitivity, especially at medium spatial frequencies, led to longer fixations during reading (De Luca et al., 1996). These results suggest that loss of contrast sensitivity over a range of spatial frequencies, not just the low ones, affects reading speed and eye movements.

Medium spatial frequencies allow for the most efficient letter identification in observers with normal vision (around 3 cycles/image; Solomon & Pelli, 1994). Legge (1993) determined that the contrast sensitivity function for words (using the fundamental frequency of each character size as the equivalent of spatial frequency) was similar in shape to the contrast sensitivity function for gratings with a peak at medium spatial frequencies. Similarly, reading rates slowed considerably when the medium spatial frequencies were removed by blurring lenses but decreased to a lesser extent with the removal of lower spatial frequencies (Legge et al., 1984). We found that filtered words containing only low spatial frequencies (i.e., peak sensitivity of the M stream) were not easily identified as words and did not produce priming in a naming task (Boden & Giaschi, 2000). Filtered words with the low spatial frequencies removed were easily identified as words and produced priming in a naming task. These results suggest that medium and high spatial frequencies are important for reading.

The peak spatial frequency of the M stream may not match the optimal spatial frequencies for letter identification and word recognition, but Chase et al. (2003) argued that the magnocellular pathway is the dominant visual pathway for text perception based on a color manipulation paradigm with an oral reading task. They argued that the M stream is depressed by red light, and that red light was found to impair reading. Several control experiments were conducted to assure that the reading impairment was due to suppression of the M stream. Their conclusions rest on the assumption that color manipulations can adequately isolate P and M streams.

In summary, loss of sensitivity in the low spatial frequencies has the potential to reduce reading rates, but only for unusually large letters. Children's books are written in very large print, so perhaps this plays an important role while children are learning to read. The

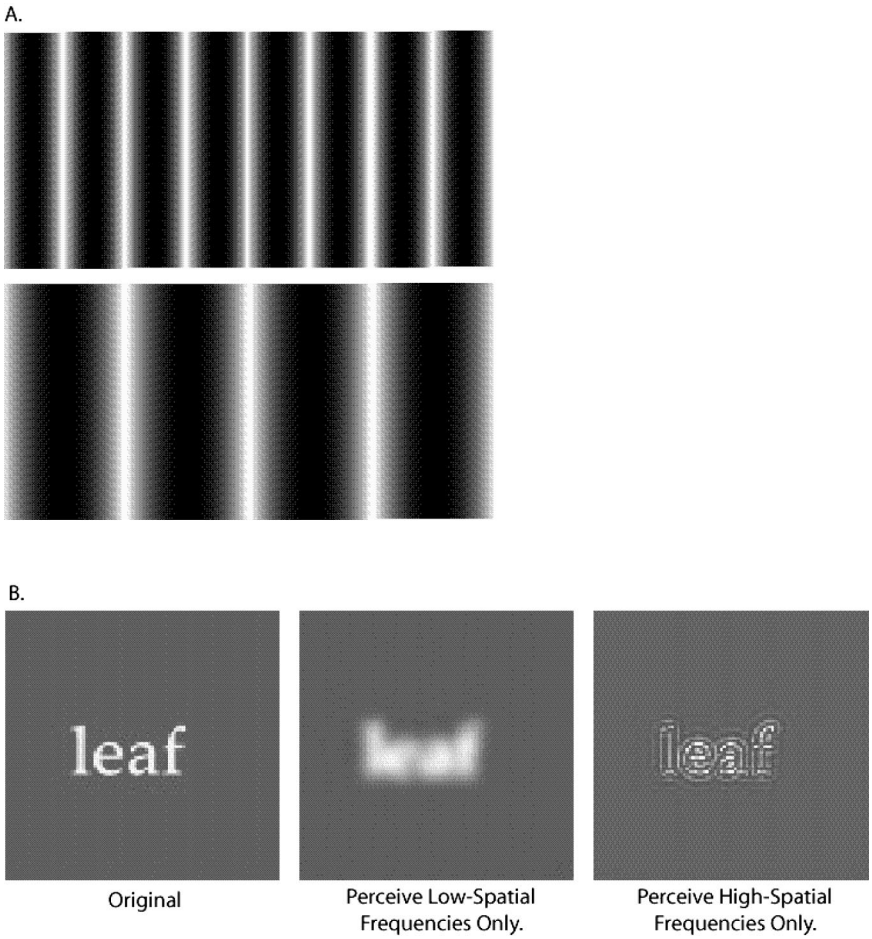

Figure 4. (A) Example of sinusoidal gratings used to determine contrast sensitivity: (upper) high spatial frequency; (lower) low spatial frequency. Contrast threshold is the smallest luminance difference between the light and dark bars for which the bars can be perceived. Contrast sensitivity is the inverse of contrast threshold. (B) An illustration of how the word "leaf" (Item 1) might be perceived by an individual with complete loss in the high spatial frequency range (Item 2) or low spatial frequency range (Item 3). Typically, people with dyslexia show only a mild reduction of contrast sensitivity, so these figures are an exaggeration.

evidence does not support the hypothesis that a contrast sensitivity deficit at low spatial frequencies interferes with the visual analysis of words. Moreover, most text is close to 100% contrast when we read, and contrast sensitivity losses reported in dyslexia are very small, so it is unlikely that such a mild loss of contrast sensitivity plays a direct role in explaining reading problems. Paradigms designed to isolate the M stream during reading tasks, such as those initiated by Chase et al. (2003), may provide a valuable tool for exploring a role for M-stream input in optimal word identification or paragraph reading.

Position Encoding Deficits (Hypothesis 2)

The position of letters within a word as well as the identity of individual letters may be important for word recognition (Driver & Baylis, 1995; Koriat & Norman, 1989; M. Mason, 1980; Whitney & Cornelissen, 2005). Cornelissen, Hanson, Hutton, et al. (1998) postulated that an M-stream deficit may create confusion about where the letters are positioned within a word. This hypothesis is consistent with the suggestion that the posterior parietal cortex, which receives M-stream input via MT, is important for encoding spatial position (Husain, 1991). Although we describe the position encoding deficits as a hypothesis separate from the visual attention hypothesis, these two hypotheses are in fact intertwined. That is, dysfunctional visual attention could be the root cause of poor spatial localization of letters within the word (Cornelissen, Hansen, Gilchrist, et al., 1998; Vidyasagar, 2001, 2004). Position encoding deficits might be thought of as a micro-level effect of an attentional deficit, whereas difficulties in assigning, moving, or distributing attention at the word level is a relatively macro-level effect, described in Hypothesis 4. We return to this point in a later section. In this section, we first describe the research from which this hypothesis emerged, then introduce a model of letter position encoding in reading as a context for understanding the effects of visual deficits on acquisition of word recognition skills.

M. Mason (1980) assessed letter and location identification in university students grouped as highly skilled or less skilled read-

ers. Both groups of readers were equivalent in identifying single letters. Highly skilled readers performed more accurately than did less skilled readers when they had to identify the serial position of a letter. These results suggest that location perception is an important factor in the reading process.

According to Cornelissen, Hansen, Hutton, et al. (1998), confusion about letter position would be manifested as "letter errors," in which children utter sounds that are not present in the printed word (orthographically inconsistent reading errors). These authors measured letter errors on a single-word reading task and phonological awareness in a group of schoolchildren ages 10 to 12 years that was not preselected in any way. The authors also measured coherent motion detection as an indicator of M-stream function. A significant positive correlation between motion detection and letter errors was found. Similarly, a positive correlation between phonological awareness and letter errors was obtained, although phonological awareness and motion detection were not correlated. The authors suggested that the letter errors were due to degraded encoding of letter position and that either an M-stream deficit or a phonological deficit could produce such errors.

A subsequent study (Cornelissen, Hansen, Gilchrist, et al., 1998) compared two groups of university students matched for IQ and reading ability. One group had good motion detection and the other group had poor motion detection. The poor motion group performed worse than the good motion group on two tasks in which optimal performance depended on accurate letter position encoding (lexical decision for words and anagrams; primed reaction time for letter targets and nonalphabetic foils). The authors concluded that information carried by the M stream may be required for encoding letter position. Alternatively, the authors suggested that attentional processing might affect performance on both motion detection and letter position tasks.

Perturbation of the function of human area MT by transcranial magnetic stimulation in adult proficient readers decreased reading performance on one (nonword naming) of two phonological tasks, but not on an orthographic reading task (Liederman et al., 2003). The patterns of errors on the nonword reading task and the absence of deficits on the other two reading tasks led the authors to conjecture that area MT contributes to image stabilization and/or letter localization, but probably not phonological decoding.

The sequential encoding regulated by inputs to oscillations within letter units (SERIOL) model is a model of orthographic processing describing how letter position is encoded and abstracted from the retinotopic representation to activate the lexical level (Whitney, 2001). This is a five-layer model (edge, feature, letter, bigram, and word) in which each layer is composed of nodes representing neural assemblies. Spatial representation is abstracted from the retinotopic representation by recoding spatial position as the relative time of firing of nodes in each layer, determined by relative activation levels. The representation of the word is built up from the edge detectors to the bigrams (letter pairs) and finally the word. Formation of the "temporal gradients" that help code for spatial location is influenced by visual attention in a manner that must be learned through experience with reading.

According to the first stage of reading acquisition proposed in the SERIOL model (Whitney, 2001; Whitney & Cornelissen, 2005), the beginning reader must activate a single letter in each fixation while simultaneously hearing the corresponding phonetic features. Temporal correspondence of the visual and phonetic features of the word allows the reader to form "graphonemic" representations. If the reader is unable to localize visual attention to a single letter in a word to enable the corresponding phonetic and graphemic representations to overlap temporally, noncorresponding phonetic and graphemic representations may be associated. Establishment of a reliable and stable phonetic–graphemic correspondence would be disrupted. This bottom-up influence of attention on reading acquisition differs from the "top-down" effects described in Hypothesis 4.

In summary, there is some support for the hypothesis that an M-stream deficit leads to reading problems by disrupting the encoding of letter position. A viable and testable model of position encoding in normal reading, the SERIOL model, has been proposed (Whitney, 2001), and a theory of reading acquisition generated from this model has been used to explain a potential route to reading problems in dyslexia (Whitney & Cornelissen, 2005). This is a promising line of research that is closely tied to the visual attention hypothesis (Hypothesis 4).

Oculomotor Deficits (Hypotheses 3 and 6)

Eye movements are an integral part of reading a sentence. Saccades of 2 to 4 degrees occur between fixations lasting 250 to 400 ms (Black, Collins, de Roach, & Zubrick, 1984c). During fixations, saccades are suppressed, but tiny microsaccades, tremors, and drifts keep the words from fading from view (Leigh & Zee, 1999), and small vergence adjustments keep the eyes aligned in the plane of the words to maintain single vision (Hendriks, 1996). It has been suggested that problems with the control of saccadic eye movements (Pavlidis, 1981) or the maintenance of stable binocular fixation (reviewed in Stein, 1991; Stein & Fowler, 1993) contribute to reading difficulties. A weak M-stream input to eye movement centers in the dorsal stream is the suggested source of such problems (Fischer & Hartnegg, 2000; Stein, 1994; Stein & Walsh, 1997).

Before considering the evidence for eye movement control problems in dyslexia, the role of the M and the dorsal streams in the control of saccadic, vergence, and smooth pursuit eye movements in humans will be reviewed. Smooth pursuit does not have a role in Morrison's reading model, but may be an indicator of M-stream integrity. The information summarized below is taken from Leigh and Zee (1999), and the reader is referred to this source for a more comprehensive review of the neural circuits controlling different types of eye movements.

V1 is of fundamental importance in the control of visually guided eye movements. Lesions to this region impair the accuracy of saccades and prevent the generation of smooth pursuit to visual stimuli in the affected part of the visual field only. Further visual processing necessary for the programming of saccadic and pursuit eye movements occurs in MT. This region encodes the direction and speed of moving stimuli, and lesions to MT disrupt saccades and smooth pursuit to moving targets. MT projects to the medial superior temporal area (MST), which is important for the generation of smooth pursuit eye movements. Lesions to this region produce directional defects in smooth pursuit. Based on lesion studies in cats, MT and MST have also been implicated in the control of vergence eye movements. MT and MST project to two regions of the parietal cortex: the posterior parietal cortex (area 7a

in monkeys) and the parietal eye field (lateral intraparietal area [LIP] in monkeys). The posterior parietal cortex is important for directing visual attention in extrapersonal space, and lesions to this region produce inattention, increased latency for visually guided saccades, and impaired smooth pursuit. This region has also been implicated in the control of vergence eye movements (Stein, 1991). The parietal eye field is important for triggering visually guided saccades. Lesions to this region increase saccadic latency, particularly when the fixation light is left on, which suggests that the parietal eye field is important for disengagement of fixation prior to a saccade.

Dorsal stream areas MT, MST, posterior parietal cortex, and parietal eye field are involved in the control of visually guided eye movements. Theoretically, therefore, an M-stream deficit would result in a paucity of visual information to these areas, which should detrimentally influence oculomotor behavior. Saccadic, vergence, and smooth pursuit eye movements should all be affected in dyslexic observers during reading and nonreading tasks if the deficit is truly oculomotor in nature.

Erratic patterns of saccadic eye movements during reading have been found in children with dyslexia (Lennerstrand, Ygge & Jacobsson, 1993; Poblano, de Caballero, Castillo, & Cortes, 1996; Rubino & Minden, 1973). The abnormal patterns include increased numbers of fixations, increased numbers of regressions (saccades to the left when reading proceeds from left to right), and longer fixation durations. These eye movement patterns are also seen in younger children who are not yet fluent readers and in proficient adult readers when the text is unfamiliar. In addition, dyslexic readers show normal eye movement patterns when the text is appropriate for their reading level. Therefore, it has been argued that abnormal saccadic eye movements do not cause dyslexia but rather are a consequence of reading problems (reviewed in Pirozzolo & Rayner, 1988).

If abnormal saccades do cause reading problems, they should be observed in nonreading tasks. Two studies found erratic patterns of eye movements in at least some children with dyslexia during nonreading tasks involving sequential saccades to symbols or spots of light arranged in rows (Elterman, Abel, Daroff, Dell'Osso, & Bornstein, 1980; Pavlidis, 1981). The abnormal patterns included an increased number of forward and regressive saccades and the inability to maintain fixation for more than 1 second. Bednarek, Tarnowski, and Grabowski (2006) found shorter latencies of visually guided saccades in children with dyslexia than in the control group. Shorter latencies correlated with spelling errors. This finding is unlikely to be explained by a visual attention deficit, as no group differences were found in either a central cueing or a peripheral cueing task. Several other studies have failed to replicate these findings (Black, Collins, de Roach, & Zubrick, 1984a, 1984b; Brown et al., 1983; Eden et al., 1994; Olson, Kliegl, & Davidson, 1983; Raymond, Ogden, Fagan, & Kaplan, 1988). These conflicting results have been attributed to differences in the sample of participants used in each study. Pirozzolo and Rayner (1988) suggested that abnormal eye movements only occur in a small subtype of dyslexia characterized by visuospatial deficits. Fischer, Biscaldi, and Otto (1993) reported more saccades of smaller amplitude and fixations of shorter duration on a sequential nonreading task in one particular subgroup of their dyslexic sample. Adults in this subgroup were more severely impaired and possibly constituted a different subtype of dyslexia.

A recent study found deficits in the voluntary control of saccades in a large group of children with dyslexia ranging in age from 7 to 17 years (Biscaldi, Fischer, & Hartnegg, 2000). A prosaccade task required saccades to a stimulus. An antisaccade task required the child to generate a saccade in the direction opposite to the stimulus side. Relative to age-matched controls with at least average reading ability, children with dyslexia demonstrated normal prosaccades to the stimulus but suppressed saccades with difficulty on the antisaccade task. This deficit was observed in up to 50% of the children with dyslexia and was attributed to either voluntary control systems of the frontal lobes or fixation and attention processes of the parietal and prefrontal cortex.

Other eye movement deficiencies that occur during fixations in reading have been implicated as potential causes of dyslexia. Poor fixation and vergence eye movements would reduce the stability and clarity of the image during a fixation, making reading of even single words more difficult. The tendency to switch from one referent eye to the other in central binocular viewing is referred to as unstable ocular motor dominance. Several groups have found that some children with dyslexia show unstable ocular motor dominance on the Dunlop test (Bigelow & McKenzie, 1985; Masters, 1988; Stein & Fowler, 1982), but these findings have been heavily criticized as a result of the subjective nature of the test and the failure to control for IQ (Bishop, 1989). There are also failures to replicate (e.g., Lennerstrand et al., 1993). Moreover, analysis of the percentage of dyslexic readers with instability of ocular dominance after taking into account the prevalence rates of dyslexia relative to controls suggests that such deficits are not more prevalent in dyslexia (Skoyles & Skottun, 2004). In spite of these criticisms, children with unstable ocular motor dominance were found to make more reading errors than age-matched controls (Stein, Riddell, & Fowler, 1986), and their reading errors were reduced when one eye was occluded (Cornelissen, Bradley, Fowler, & Stein, 1992). Some children (7 to 11 years old) who received treatment in the form of monocular occlusion for unstable ocular motor dominance showed improvement in binocular fixation and also in reading (Masters, 1988; Stein, Richardson, & Fowler, 2000). Interpretation of these results has been disputed (Bishop, 1989), and it seems likely that the reading problems experienced by these children would not fit the traditional definition of developmental dyslexia, which includes a lack of sensory problems.

Unstable ocular motor dominance has been linked to the control of vergence eye movements (Stein et al., 1988). The normal vergence adjustments required following saccades during reading are less than 0.5 degree, but vergence errors of up to 5 degrees have been recorded from participants with normal reading ability (Stein et al., 1989). Increased vergence errors during reading in subjects with dyslexia have not been reported, but some children with dyslexia show vergence control problems on nonreading tasks. The reported deficiencies in vergence eye movements include slower vergence velocities (Buzzelli, 1991; Hung, 1989), reduced vergence amplitudes (Eden et al., 1994; Evans, Drasdo, & Richards, 1996; Latvala, Korhonen, Penttinen, & Laippala, 1994), and a diminished ability to follow small targets moving in depth

(Hung, 1989; Masters, 1988; Stein et al., 1988). Other studies found vergence control on nonreading tasks to be normal in children (Lennerstrand et al., 1993) and adults (Moores, Frisby, Buckley, Reynolds, & Fawcett, 1998) with dyslexia.

Other evidence for poor fixational control in dyslexia comes from the direct recording of eye movements during fixation on a stationary target. On nonreading tasks, children with dyslexia showed more variability in eye position (Eden et al., 1994; Raymond et al., 1988) and more intrusive saccades (Fischer & Hartnegg, 2000) during fixation than did age-matched controls. These results suggest that poor maintenance of gaze stability rather than inadequate control over saccadic eye movements characterizes the oculomotor problems in dyslexia. Alternatively, a deficit independent of core deficits commonly used to explain dyslexia (i.e., phonological problems and M-stream deficits) may underlie both reading problems and eye movement abnormalities in some individuals with dyslexia. Impaired visual processing of multi-element arrays was found to increase the number of eye movements during reading, independently of coherent motion detection (a measure of M-stream integrity), phonological measures, and rapid naming (Hawelka & Wimmer, 2005).

There are also clear deficits in smooth pursuit in children with reading problems. These deficits include an increased number of saccadic intrusions, both in the direction of target movement and in the reverse direction during voluntary pursuit of a moving target (Black et al., 1984c; Eden et al., 1994; Lennerstrand et al., 1993; Masters, 1988; Sucher & Stewart, 1993). These deficits could be due to a fundamental problem in the pursuit system or to a more general problem in the maintenance of fixation on a target. Smooth pursuit eye movements may be associated with phonological awareness in preschool children (Callu et al., 2005), though it is not clear why.

In summary, there is good evidence for smooth pursuit and fixation deficits, conflicting evidence for vergence deficits and ocular motor dominance instability, and weak evidence for saccadic deficits in developmental dyslexia. The conflicting results may be due to sampling bias if eye movement control is a problem only in certain subtypes of dyslexia. Hypotheses about eye movement deficits in dyslexia due to abnormal M-stream function have been justified on anatomical grounds in the present article. Although the evidence supports some such deficits in dyslexia (Bednarek et al., 2006; Biscaldi et al., 2000; Black et al., 1984c; Buzzelli, 1991; Eden et al., 1994; Elterman et al., 1980; Evans et al., 1996; Fischer et al., 1993; Fischer & Hartnegg, 2000; Hung, 1989; Latvala et al., 1994; Lennerstrand et al., 1993; Masters, 1988; Moores et al., 1998; Pavlidis, 1981; Poblano et al., 1996; Raymond et al., 1988; Rubino & Minden, 1973; Stein et al., 1989; Sucher & Stewart, 1993), few studies have examined the effect of subtle disruption of eye movements on reading performance in normal readers, and at least one study dissociated motion coherence thresholds from eye movement deficits (Hawelka & Wimmer, 2005). Without further study of these issues, we can only conclude that normality of gross eye movements is likely to be necessary to reading on the grounds of common sense, that subtle eye movement deficits may or may not disrupt normal readers, and that anatomical and physiological evidence validates the dorsal stream control of some aspects of eye movements.

Visual Selective Attention Deficits (Hypothesis 4)

Once sufficient visual detail has been gained from the fovea during a fixation, the attentional focus moves forward to the next word in the sentence before the eyes move. This is referred to as covert spatial orienting of attention. This aspect of visual attention has been examined with a variety of paradigms.

There is fairly good evidence that the posterior parietal cortex (PPC), which receives input predominantly from the M stream, is integral to some aspects of visual spatial attention. Damage to the PPC affects the ability to disengage attention (Posner et al., 1984) and can result in neglect dyslexia (particularly in right hemisphere injuries). Neglect dyslexia is an acquired reading disorder in which the beginning words of a line of text or the beginning letters of single words are ignored. This disorder is thought to arise from a disruption in the allocation of spatial attention (Behrmann, Moscovitch, & Mozer, 1991; Brunn & Farah, 1991; Riddoch, Humphreys, Cleton, & Fery, 1990).

Neuroimaging studies have implicated the PPC in covert spatial orienting (Corbetta et al., 1998; Rosen et al., 1999) and other aspects of visual attention, including visual search (Corbetta, Shulman, Miezin & Petersen, 1995; Hopf et al., 2000; Leonards, Sunaert, Van Hecke, & Orban, 2000), nonspatial shifts of attention between target features (Le, Pardo & Hu, 1998), temporal orienting (Coull & Nobre, 1998), and attentive tracking of moving objects (Culham et al., 1998).

Psychophysically, there is some evidence that serial deployment of visual attention in visual search may be most efficient with M-stream input (Cheng, Eysel, & Vidyasagar, 2004). In addition, the M stream appears to be important to flanker tasks used to examine the ability to filter useful from extraneous visual information (Omtzigt & Hendriks, 2004).

It has been postulated that slight impairments in the subcortical pathway in dyslexia are magnified in the PPC and expressed as deficits in visual selective attention (Stein & Walsh, 1997; Steinman et al., 1996, 1998; Vidyasagar, 1999). More specifically, Vidyasagar proposed that M-stream–mediated visual spatial attention acts as a gatekeeper for information entering the ventral stream by spotlighting one or more letters for decoding by the ventral stream.

This concept is consistent with Pollatsek, Rayner, Fischer, and Reichle (1999), who suggested that the movement of the eyes probably does most of the work in selecting what information is to be processed at any given moment in reading. Visual acuity limits the perceptual span (region around fixation from which useful information can be extracted) to about 15 characters, but covert attention prevents information outside the current line of text and to the left of fixation from being attended. Covert spatial attention could serve to filter out letters that do not belong to the word being read (low level). Or it may allow special processing routines to be directed at the attended word to aid in the parallel integration of the component letters (higher level). It should be noted that some researchers would argue that reading is an automatic task (i.e., not under attention control; e.g., Brunn & Farah, 1991). Reading proficiency may dictate the level of automaticity in reading.

The relationship between attention mediated by the M stream and reading was examined with a covert orienting paradigm in children with at least average reading skills (Kinsey, Rose, Hanson, Richardson, & Stein, 2004). Four patches of moving dots

were presented, and 1 patch had a percentage of dots that moved coherently (the target). Motion coherence thresholds were obtained under valid (cue correctly indicated location of the target) and invalid (cue presented at a nontarget location) conditions. Performance was better on valid relative to invalid trials in a group of readers ranked in the top 25% of the sample. There was no difference as a function of cue type in lower ranked readers. It was suggested that less skilled readers have poorer attentional processing capacities. In addition, regression analysis showed a relationship between visual attention and reading that was stronger for nonwords than for irregular words.

The literature on visual attention in dyslexia is very mixed in both the methodology and outcome. We discuss first the studies assessing aspects of visual spatial attention that physiological, psychophysical, and neurological studies suggest are associated with the PPC and, therefore, presumably susceptible to M-stream deficits. These functions include covert spatial orienting and visual search. Next, we discuss the literature on other aspects of attention that have been examined in dyslexia.

Visual Attention Associated With the M Stream: Covert Spatial Orienting and Visual Search

Past studies have shown covert orienting deficits in poor readers (Brannan & Williams, 1987) and individuals with dyslexia (Buchholz & Davies, 2005; Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000) as well as selective attention deficits in individuals with dyslexia (Sobotka & May, 1977). Difficulties with both space- and object-based covert shifts of attention have been demonstrated in adult dyslexics who were selected to have phonological difficulties (Buchholz & Davies, 2005). Specifically, dyslexic participants showed slower responses to between-object shifts of attention in the left visual field and difficulty engaging stimuli in the periphery, but not the fovea. On the other hand, Harter, Anllo-Vento, and Wood (1989) interpreted an increased VEP response in individuals with dyslexia as an indication that a reading disability is actually associated with an enhancement of spatial orienting. A more recent study of covert orienting more carefully controlled stimulus and sensory confounds that cloud the interpretation of some of the original work on spatial orienting of attention. Using a spatial-cueing paradigm, Roach and Hogben (2004) demonstrated a marked spatial-cueing deficit in 5 adults with dyslexia. These adults, however, had global motion and flicker contrast sensitivity thresholds within the range of control participants. This finding was interpreted by the authors as evidence that M-stream function was not responsible for the visual spatial orienting deficit in the dyslexic group. Although we agree that the motion-processing deficits were weaker than the spatial-cueing deficits, the global motion thresholds were at the high end of normal.

Visual search involves looking for a particular item of interest, perhaps your friend, among other items in the visual scene (the other people in the crowd). In early studies, visual search efficiency was reportedly normal in dyslexia (Casco & Prunetti, 1996; Ruddock, 1991). Closer examination of the data revealed that observers with dyslexia were more likely to miss the target when it was present (Ruddock, 1991) and searched less efficiently when the aspect distinguishing the multifeatured target from the distractors was the spatial relationship of the features within the shape (Casco & Prunetti, 1996). More recently, visual search abnormalities have been found in individuals with dyslexia for tasks involving search for a single feature (Facoetti, Paganoni, & Lorusso, 2000) or for a conjunction of features (Buchholz & McKone, 2004). This was partially supported in another study by weak differences between individuals with dyslexia and controls (Vidyasagar & Pammer, 1999). If visual search performance is truly related to the integrity of the M stream, only individuals with dyslexia who have M-stream deficits should show visual search problems. Iles, Walsh, and Richardson (2000) indeed found that individuals with dyslexia who had elevated motion coherence thresholds showed impaired serial visual search, whereas those with normal thresholds were not impaired. Inconsistencies in the literature could, therefore, be due to heterogeneous proportions of dyslexics with M-stream deficits across studies.

Other Perspectives on Visual Attention

Whereas there is some psychophysical evidence that M-stream input is important to performance on flanker tasks (used as a measure of focusing; Omtzigt & Hendriks, 2004), focusing and filtering are not generally considered to be dorsal stream functions. Extrastriate regions of the ventral stream have been implicated in the focusing of attention and filtering out of distractor information (Heinze et al., 1994; Hopf et al., 2000; Leonards et al., 2000). Nevertheless filtering and focusing have been examined in dyslexia.

Using the line motion illusion as an index of visual attention, Steinman and colleagues found a narrower attentional focus and a stronger band of attentional inhibition surrounding the fovea in a small group of adults with dyslexia (Steinman et al., 1996, 1998). Interpretation of these results, however, is clouded by more recent work suggesting that attention is not the source of the line motion illusion (Christie & Klein, 2005).

A series of studies by Facoetti and her colleagues also suggest that individuals with dyslexia distribute their attention differently than normally reading controls, either in a more distributed fashion (Facoetti, Paganoni, & Lorusso, 2000) or asymmetrically to the right visual field (Facoetti & Molteni, 2001; Facoetti & Turatto, 2000). Asymmetric allocation of attention in dyslexia was also reported by Hari, Renvall, and Tanskanen (2001) and Sireteanu, Goertz, Bachert, and Wandert (2005). This asymmetry was termed a "left-sided mini-neglect" and was attributed to a right posterior parietal defect (Facoetti & Turatto, 2000; Hari et al., 2001; Sireteanu et al., 2005). The type of stimulus used may affect the symmetry of attentional distribution. For example, Geiger and Lettvin (1999) found that dyslexic children have a wider "form-resolving field" on both right and left sides when the stimuli are letters and words. Developmental dyslexia (or at least certain subtypes) may be characterized by a problem with the allocation of the "attentional spotlight," or the use of a strategy for deploying attention to cope with reading problems that has generalized to the attention tasks.

If poor readers and individuals with dyslexia have trouble filtering, they should be more affected by the presence of distractors than are good readers. Klein and D'Entremont (1999) measured performance on a flanker compatibility effect in poor and good readers. In this paradigm, stimuli flanking a target that are compatible with the target yield a faster and more accurate response than flankers that are incompatible. In good readers, proximity to

the target mattered, with flankers farther from the target having a smaller influence on responses. The effect of distractors was similar at all distances in poor readers, which might indicate greater difficulty filtering out extraneous information. In line with these findings, selective attention deficits, especially under high short-term memory load, have been noted in several studies (Enns, Bryson, & Roes, 1995; Rayner, Murphy, Henderson, & Pollatsek, 1989; Steinman et al., 1996, 1998; Tarnowski, Prinz, & Nay, 1986).

Assorted other attention deficits have been demonstrated in dyslexia (Davis, Castles, McAnally, & Gray, 2001; Duncan et al., 1994; Klimesch et al., 2001; Moores & Andrade, 2000; Solan, Larson, Shelley-Tremblay, Ficarra, & Silverman, 2001; Tarnowski et al., 1986), further weakening the specificity of attention deficits to M-stream–mediated functions. Thus, a variety of attention deficits are found in dyslexia. Some of these deficits may be due to an M-stream deficit, some are less likely to be reliant on an intact M stream and some could be related to strategies developed to cope with reading difficulty.

Valdois and her colleagues offer an alternative viewpoint (Bosse & Valdois, 2003; Valdois, Bosse, & Tainturier, 2004). These authors argue that a selective visual attention disorder is a second core deficit in dyslexia, independent of a phonological deficit. Unlike the hypotheses above, they do not link M-stream deficits with selective attention deficits. Citing the extensive literature supporting visual attention deficits in dyslexia we reported above, they argue that visual attention deficits interfere with the acquisition of lexical and possibly phonological information necessary for successful reading. Their connectionist multiple-trace memory model for polysyllabic word reading proposes a global and analytic mode of reading (Ans, Carbonnel, & Valdois, 1998). The global mode dominates, but the analytic mode is implemented when a word cannot be identified in global mode. Visual attention is necessary to focus the visual attentional window on a subcomponent of the word (e.g., the first syllable) for processing. An impoverished capacity to manipulate the visual attentional window within a word would impair word identification and also the formation of memory traces that form the basis for learning to read new words (regular or irregular). In this model, irregular words are more likely to require analytic mode, and thus a visual attentional deficit should primarily affect irregular word identification.

This model differs from the SERIOL model (Whitney, 2001; Whitney & Cornelissen, 2005), described in the previous section on the position encoding hypothesis, in that the SERIOL model only deals with orthographic processing and requires visual attention for the formation of gradients that encode spatial position of letters and bigrams to generate an orthographic representation of the word. Unlike Valdois and her colleagues (Bosse & Valdois, 2003; Valdois, Bosse, & Tainturier, 2004), Whitney and Cornelissen (2005) do contend that visual attention might be altered by a compromised M stream in dyslexia and that this might disrupt the formation of graphonemic representations during reading acquisition.

In summary, there are plausible roles for visual attention in reading and reading acquisition. The M-stream input to areas of the cortex involved in visuospatial attention and the presence of some form of visual attention deficit in some dyslexics are also credible. A specific visuospatial deficit resulting from a faulty M stream in the majority of individuals with dyslexia, however, is not well supported by the literature. Two theories have been put forward that place visual attention deficits in a central role, rather than as an ancillary deficit found alongside phonological problems. A recent theory by Valdois and her colleagues (Bosse & Valdois, 2003; Valdois, Bosse, & Tainturier, 2004) suggests that visual attention deficits may be at the core of dyslexia in a subgroup of individuals, independent of an M-stream deficit. In contrast, Whitney and Cornelissen (2005) argued that difficulty encoding spatial position during reading acquisition, possibly due to an impairment of visual attention related to an M-stream deficit, explains some problems learning to read. These two theories offer an avenue for understanding a level of word recognition that is not commonly examined in the reading literature: the stage from visual processing of the letters to an orthographic code. These theories also offer the possibility of re-interpreting our understanding of dyslexia from a condition that is driven by phonological deficits to one that has visual impairments at the core in some individuals.

Deficits in Parafoveal/Foveal Interactions (Hypothesis 5)

After the attentional gaze has moved to the parafovea, information uptake from the parafoveal text begins. The M stream may be valuable in rapid access to visual features of the parafoveal word. It is assumed that the low-spatial-frequency, global information from the parafoveal word carries sufficient information to enhance processing of local information (Lehmkuhle, 1993). A sluggish M stream would interrupt the temporal precedence of the global information and reduce the normal facilitation provided by parafoveal processing.

Visual information about the beginning and ending letters of words, word length, and word shape is obtainable from parafoveal vision (Rayner, Inhoff, Morrison, Slowiaczek, & Bertera, 1981). Not only do readers use this information to program saccades, but they also use partial letter information from words in the parafovea to facilitate processing of the parafoveal word when it is eventually fixated (Rayner, Well, Pollatsek, & Bertera, 1982). Notably, the initial three letters of the parafoveal word are almost as helpful as the whole word in parafoveal preview. This parafoveal information, however, is insufficient to access the meaning of the word (Inhoff & Rayner, 1980; Rayner et al., 1981). Non-foveal orthography influences fixation position independently of foveal processing load in adult normal readers (White & Liversedge, 2005). Along a similar vein, access to the meaning of sentences presented peripherally is inferior to that in the fovea even though the words can be recognized (Latham & Whitaker, 1996). Semantic access to words presented in the periphery appears to be decoupled from the ability to recognize those same words. M-stream deficits may reduce the usefulness of parafoveal information because the temporal precedence of the faster M stream is interrupted (Lehmkuhle, 1993; Lovegrove et al., 1986; Steinman et al., 1996).

Efficient reading, therefore, relies on constant interaction between information processed in the fovea and in the parafovea. Foveal and parafoveal processing in dyslexic observers will be examined first; then the temporal aspects of the temporal precedence hypothesis will be discussed. Initial reports suggested that those with reading disabilities show poorer acuity in a letter identification task in the fovea, but better performance in the periphery, relative to the performance of controls (Geiger & Lettvin, 1987). The finding of Geiger and Lettvin (1987) might have

been due to visual attention, as children with reading disabilities are less likely to be affected by a foveal distractor during a parafoveal letter detection task than are controls (Goolkasian & King, 1990). In answer to methodological concerns, several studies were conducted, which, for the most part, failed to support this finding (Goolkasian & King, 1990; Klein, Berry, Briand, D'Entremont, & Farmer, 1990). Farmer and Klein (1995) proposed that observers with dyslexia have visual persistence from a prior fixation interfering with processing of the current fixation. The perceptual span may be smaller in dyslexic readers not because they process parafoveal information less effectively but because they have difficulty processing the fixated word.

Now that we know something about initial processing in the fovea and parafovea in dyslexia, the temporal aspects of the temporal precedence hypothesis can be addressed. This hypothesis predicts that coarse detail information carried by the M stream must precede the fine detail information of the P stream for the benefits to be incurred (Lehmkuhle, 1993). A phenomenon known as metacontrast masking (extensively studied in its own right; see Breitmeyer, 1978a, 1978b) has been used to evaluate the relative speed of the M and P streams in dyslexia. A target is presented briefly, followed after a certain stimulus onset asynchrony (SOA), by a spatially adjacent masking stimulus. Visibility of the target is poor at short SOAs but improves with longer SOAs. Loss of visibility of the target is called masking and is thought to be due to M-stream inhibition of the P stream. Williams, Molinet, and LeCluyse (1989) argued that since the point of greatest masking provides an index of the relative speed of the M and P streams, masking was a useful tool to examine the hypothesis of a sluggish M stream. The processing rate for the M stream was slower for children with dyslexia, as compared with controls, and suggests a lack of M-stream activity in the periphery. Similar results were found with dyslexic adolescents (Edwards, Hogben, Clark & Pratt, 1996).

Williams, Brannan, and Lartigue (1987) found that visual search times for poor readers decreased when the visual arrays were slightly out of focus while blurring had no effect on good readers or adults. These authors suggested that blurring reduced the high spatial frequency content of the stimulus and restored the temporal precedence of the M stream by slowing the response of the P stream. An attempt to replicate this finding failed (Hogben, Pratt, Dedman, & Clark, 1996). Blurring was found to cause a general slowing of search times regardless of group membership. Methodological differences producing differences in spatial frequency content could be responsible for the discrepancy; therefore, it is impossible to draw firm conclusions from these two studies.

If the temporal precedence hypothesis is correct, individuals with dyslexia should show abnormal parafoveal–foveal interactions accompanied by a sluggish M stream. To summarize, several studies have noted abnormal foveal/parafoveal interactions (Geiger & Lettvin, 1987; Goolkasian & King, 1990; Rayner et al., 1981, 1989). The effect of eccentricity on letter identification does not vary between dyslexic and control groups (Goolkasian & King, 1990; Klein et al., 1990), but the effect of eccentricity on spatial localization does (Solman & May, 1990). The source of the abnormality in parafoveal/foveal interactions might be driven by visual attention, however, which is not consistent with the temporal precedence hypothesis. Yet, there is evidence of a sluggish M stream in individuals with dyslexia (Edwards et al., 1996; Williams et al., 1989).

Conclusions

The intent of this article was to characterize the reading-related perceptual processes hypothesized as the links between M-stream deficits and reading problems. Several steps were taken toward this goal. The typical procedures for the diagnosis of dyslexia, subtypes of dyslexia, and M-stream deficits were reviewed. The next step was to clarify, to the extent possible, the relationship among these three diagnostic schemes. The final step was to evaluate the strength of the connection of each reading-related dorsal stream process to reading and to dyslexia separately. The findings are summarized below.

There exists physiological and anatomical evidence implicating the M stream in functions critical to reading. Neural machinery controlling saccadic and pursuit eye movements acquires information via MT and MST. The posterior parietal cortex contributes to visual attention and receives predominant input from the M stream. Focusing and shifting of attention may be crucial to reading. Characteristics of proficient reading are consistent with roles ascribed to the M stream by reading disability researchers. Mixed findings exist in the reading disability literature. Although no one hypothesis is strongly implicated, there is some support for each hypothesis. It is conceivable, therefore, that faulty development of the sensory input stages of the M stream could affect the ability to learn to read.

Reading-related perceptual processes are abnormal in portions of the dyslexic population. Unstable fixation and oculomotor problems (Hypotheses 3 and 6, put forward by Stein, 1991, 1994; Stein et al., 1989; Stein & Walsh, 1997) are partially supported by findings of unsteady fixation (Eden et al., 1994; Sucher & Stewart, 1993) and abnormal vergence eye movements (Buzzelli, 1991; Latvala et al., 1994). Common sense dictates that if one cannot fuse the image or focus steadily on a word, that uptake of visual information will be impaired. There is little evidence of an oculomotor-based saccadic eye movement deficit. There is evidence of an attentional deficit in dyslexia (Hypothesis 4: proposed by Breitmeyer, 1993; Stein & Walsh, 1997; Steinman et al., 1996, 1998) and reading theories to explain how a visual attention deficit would act on reading and reading acquisition. There are fairly strong indications of a selective attention deficit (Rayner et al., 1989; Sobotka & May, 1977; Steinman et al., 1996, 1998) and some signs of a problem in disengaging attention (Brannan & Williams, 1987). Not all visual attention deficits found in individuals with dyslexia are likely to be mediated by the M stream, thus weakening the case for an M-stream–specific explanation of visual attention problems in dyslexia. Visual attention deficits independent of M-stream function may contribute to reading difficulties nevertheless (Bosse & Valdois, 2003; Whitney & Cornelissen, 2005). The results are mildly supportive of the temporal precedence hypothesis (Hypothesis 5: Lovegrove et al., 1986; Steinman et al., 1996, 1998). Foveal–parafoveal interactions are unusual in the dyslexic groups tested but can be attributed to attention problems, and there is some evidence of a sluggish M stream interacting abnormally with the P stream. Again, a lack of studies directly addressing the issue precludes strong conclusions. Moreover, it is

difficult to directly assess whether temporal precedence of M-stream information facilitates reading.

An additional hypothesis was raised by the literature on contrast sensitivity and reading. Low-spatial-frequency information, carried by the M stream, may be necessary for the most efficient reading or, more specifically, while learning to read. This is not a strong hypothesis, as neither the physiological properties of magnocellular cells nor the literature on contrast sensitivity in reading point specifically to the M stream.

The notion that M-stream deficits contribute to reading problems through reading-related dorsal stream functions holds promise. There are several avenues of research that show potential for elucidating the role of vision, and specifically the M stream, in reading. First, researchers need to establish whether individuals with dyslexia who demonstrate an M-stream deficit belong to one of the already identified subtypes, whether they belong to their own subtype, or whether the M-stream deficit is unrelated to the reading problem. Developmental dyslexia is generally diagnosed by a discrepancy definition in combination with exclusionary criteria. Not everyone agrees with this diagnostic approach (e.g., Siegel, 1992), and it may affect our ability to identify children who have visual problems at the core of their reading disability.

Studies have begun examining whether defects in higher order functions (e.g., attention, eye movements, and foveal/parafoveal interaction) in individuals with dyslexia are concurrent with an M-stream deficit (Hawelka & Wimmer, 2005; Iles et al., 2000; Roach & Hogben, 2004). The results are mixed. If it really is damage to the M stream that is leading to abnormalities in attention and so forth, then only the dyslexics with an M-stream deficit should experience these problems. Further work needs to be conducted with a greater emphasis on determining the characteristics of dyslexics who have an M-stream deficit. Is there a selection bias away from those who have an M-stream deficit or is such a deficit highly uncommon in this population?

Third, deficits in reading-related processes should cause characteristic reading errors. The present article has elucidated six hypotheses that are available for testing how the M stream may contribute to reading problems through reading-related dorsal stream processes (e.g., visual attention). Two of these hypotheses, position encoding (Hypothesis 2) and visual attention (Hypothesis 4), fit within the framework of models of word recognition. The challenge will be separating language-based reading errors from potentially visually based reading errors. Care must be taken in interpreting how an M-stream deficit will manifest itself in higher level perceptual and cognitive functions in any given child. A developmental reading disability, as opposed to acquired dyslexia, is by definition present from the time the child is born and through the earliest stages of development. Plasticity of the brain during development reduces the likelihood of a single highly specific deficit. Rather, the anatomical structure of areas nearby the defective site and those to which that site connects may also be affected. The impact on these secondary structures could, theoretically, vary from child to child as their experience and genetic makeup also differs. One would expect, therefore, more subtle and variable deficits in this population.

Future studies must begin by characterizing the dyslexic population with respect to two attributes: subtype of dyslexia (phonological/surface distinction looks promising) and the presence versus absence of an M-stream deficit. How would one go about identifying an M-stream deficit? Current vision literature indicates that the best tests with which to assess M-stream integrity are flicker sensitivity, contrast sensitivity for low spatial frequency and high–temporal frequency stimuli and global motion perception (reviewed in Edwards et al., 2004). Neither in practice nor in theory do these tests cleanly differentiate subcortical from cortical sites of damage or abnormality.

In conclusion, it is time to take seriously the role of the M stream in reading. There are a number of reading-related processes that might bridge the gap between the visual temporal processing deficit and reading. A great deal more work must be done, however. Two connections must be made more directly: the connection between the M stream and what we have been calling the reading-related processes (e.g., visual attention), and, perhaps more urgently, the connection between the reading-related processes and reading. On a theoretical basis these connections make sense, but they must be demonstrated empirically.

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