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Visual Magnocellular Deficits In Dyslexia : Are These Deficits Due To Co-morbidity With ADHD?

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VISUAL MAGNOCELLULAR DEFICITS IN DYSLEXIA:

ARE THESE DEFICITS DUE TO CO-MORBIDITY WITH ADHD?

by

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Visual Magnocellular Deficits in Dyslexia:

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Master of Arts 2011

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Abstract

Some cases of dyslexia may be accounted for by a visual problem involving the magnocellular pathway. Research on dyslexia and problems in the magnocellular pathway has been controversial. Some studies indicate that individuals with dyslexia have problems in this pathway whereas other studies have not. It may be that only the individuals with both dyslexia and ADHD have problems in this pathway while individuals with dyslexia only are spared. In support of this, research has shown that individuals with schizophrenia have attention deficits (similar to those seen in individuals with ADHD) and problems in the magnocellular pathway. In the present study, controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only completed central and peripheral backward masking experiments. It was predicted that the two groups of participants with ADHD would have problems in the magnocellular pathway. Some evidence was found in support of this.

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Dyslexia: Recognized as a Phonological Problem, but may also be a Visual Problem

The focal point of this paper is on a population of adults with dyslexia. **Dyslexia**[1], or reading disorder as defined in the Diagnostic and Statistical Manual of Mental Disorders-4th Edition-Text Revision (DSM-IV-TR; American Psychiatric Association, 2000), is diagnosed when an individual's reading level is unexpectedly low given the individual's age, intelligence, and education. Further, reading level must interfere with academic achievement and reading difficulties need to exceed those associated with a sensory deficit (if a sensory deficit exists). Today, the common view of dyslexia is that it is a verbal deficit in phonological speech awareness. The **phonological model of dyslexia** assumes that speech is inherent and natural whereas reading must be learned (S. E. Shaywitz & Shaywitz, 2005; Snowling, 1996). Accordingly, new readers need to understand that spoken words can be broken down into phonemes, the smallest units of sound that convey meaning in a language, and that phonemes are represented by the letters in a written word. Regarding individuals with dyslexia, the conventional perspective is that they struggle with learning how letters and their sounds are related (S. E. Shaywitz & Shaywitz, 2005; Snowling, 1996).

Contrary to popular belief, this phonological model of dyslexia may not represent the main deficit in all individuals with the disorder. In particular, it does not take into account the visual deficits that have been found in some individuals with dyslexia. For example, Martin and Lovegrove (1987) found evidence for transient subsystem deficits in the visual systems of children with specific reading disabilities (SRD). Importantly, the **transient subsystem** involves

[1] Definitions of bolded terms, in addition to being provided in the body of the paper, are provided in a glossary at the end of this paper.

what is called the visual **magnocellular pathway (m-pathway)** which through its key functions can influence reading ability (Schechter, Butler, Silipo, Zemon, & Javitt, 2003; Stein & Walsh, 1997). A transient subsystem deficit in children with SRD can thus suggest an m-pathway deficit, which in turn can explain the reading problems that occur. To fully understand this, it is necessary to review (1) what the m-pathway is and (2) how it can disturb reading.

1. The Visual Magnocellular Pathway

The primate visual system consists of the magnocellular, parvocellular, and koniocellular pathways. Deficits in the magnocellular pathway have been observed in individuals with dyslexia and have been postulated to be the cause of reading problems in this group. This pathway begins in the retina where the axons of some ganglion cells project to the magnocellular layers of the lateral geniculate nucleus (LGN; Merigan & Maunsell, 1993). From there neural information is sent to the primary visual cortex (V1) and then to the medial temporal area (area MT) and to the middle superior temporal area (area MST), both of which are part of extra-striate cortex. The m-pathway is most stimulated by objects that move and flicker (quick onset, quick offset) and processes these objects with short bursts of neural firing (i.e., transient neural firing; Merigan & Maunsell, 1993). As mentioned earlier, the transient subsystem involves the visual magnocellular pathway. In research, the transient subsystem and the visual magnocellular pathway are often cited as being the same (e.g., Schechter et al., 2003).

Knowledge of the type of stimuli that maximally excite the m-pathway has been obtained from lesion studies involving monkeys. Researchers would create lesions in certain parts of the magnocellular pathway and then infer from the deficits what those parts did functionally. Schiller, Logothetis, and Charles (1990), for instance, found that motion detection and motion discrimination were impaired in rhesus monkeys with damaged magnocellular layers of the

LGN. Schiller et al. (1990) injected a neurotoxin into these layers and then presented the monkeys with several tasks. One task was designed to test motion detection. In this task, stimuli moved randomly on the screen while other stimuli moved together in a particular direction (i.e., motion coherence task). Another task was designed to test motion discrimination. In this task, a stimulus differed from other stimuli in velocity or direction. Schiller et al. (1990) concluded that the magnocellular layers of the LGN mediate the temporal aspects of vision, which are integral aspects of motion.

Rudolph and Pasternak (1999) conducted a study with similar results involving the extra-striate parts of the m-pathway (i.e., areas MT and MST). They first injected a neurotoxin into areas MT and MST of two macaque monkeys. They next presented these monkeys with moving gratings and random dot stimuli, which varied in the type and amount of visual noise. The monkeys had to discriminate the direction of motion. Rudolph and Pasternak (1999) found that the monkeys, with lesions in areas MT and MST, were impaired in this discrimination, particularly in the presence of visual noise. Based on this finding, these researchers inferred that areas MT and MST of the m-pathway are responsible for the perception of motion direction (i.e., in conditions of visual noise). This inference is supported by other research indicating that neurons in area MT are selective to motion direction and speed (Maunsell & Van Essen, 1983).

In addition to finding that the m-pathway processes motion information, researchers have also found that it is selective in another way: it responds to stimuli with low luminance contrasts (e.g., 2%; Merigan & Maunsell, 1993). Luminance contrasts represent the difference between the luminance of a brighter area and the luminance of an adjacent darker area. Low luminance contrasts in particular indicate that the difference in luminance of these two areas is small.

Perceptually then, the two areas would not appear to be very different in this regard (e.g., white and a very light grey vs. white and black).

The m-pathway is also best stimulated by objects that have low spatial frequencies and high temporal frequencies (Merigan & Maunsell, 1993). **Spatial frequency** is a measure of how fine a re-occurring visual pattern is in terms of the number of lines or points per unit distance (Leibovic, 1990). It is often given in cycles per degree on the retina (c/deg; see Figure 1 for example). **Temporal frequency**, on the other hand, is the speed of a drifting stimulus (e.g., drifting grating) in hertz multiplied by the spatial frequency (Leventhal, 1991). Objects that have low spatial frequencies appear blurrier than those with high spatial frequencies and objects that have high temporal frequencies appear to move quicker than those with low temporal frequencies.

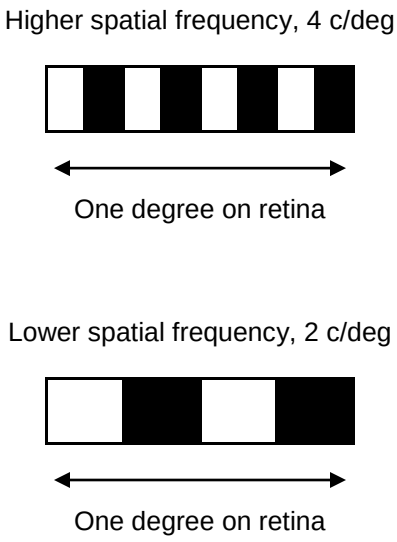

Figure 1. Pictorial example of spatial frequencies.

Merigan, Byrne, and Maunsell (1991) observed that motion detection contrast sensitivity and opposite direction discrimination contrast sensitivity were reduced in macaque monkeys with impaired magnocellular layers of the LGN. They injected a neurotoxin into these layers and

then presented the monkeys with detection and discrimination tasks. These tasks involved gratings that drifted and that varied in spatial and temporal frequencies. Merigan et al. (1991) concluded that the LGN segment of the m-pathway mediates the visibility of stimuli that have low spatial frequencies and high temporal frequencies, conditions that support motion perception.

Taken altogether, it is important to know about the m-pathway because it is believed to be impaired in individuals with dyslexia (Stein, 2001). An m-pathway that is impaired in individuals with dyslexia can disturb reading through: (1) the lack of parvocellular suppression or (2) binocular instability (Skottun & Parke, 1999; Stein, 2001; Stein & Walsh, 1997). Each of these is discussed in the next section. It is also important to know about the m-pathway as the features of it (e.g., that it responds to low luminance contrasts and flicker) formed the basis for the experiments used in the present study.

2. How a Visual Magnocellular Deficit can lead to Reading Problems in Dyslexia

Researchers have proposed two theories for how a deficit in the m-pathway can lead to problems in reading. One theory involves the **sustained subsystem** or more specifically, the **parvocellular pathway (p-pathway)**. The p-pathway is one of three pathways in the visual system. Before this theory is discussed, the p-pathway is described. The p-pathway begins in the retina and extends to the extra-striate cortex (Merigan & Maunsell, 1993). It is stimulated by object colour, texture, and pattern and processes such details with sustained neural firing (Schiller et al., 1990). This is contrary to the m-pathway, which processes flicker and motion with short bursts of neural firing. The parvocellular pathway does not respond well to luminance contrasts below 10% and is maximally affected by stimuli that have high spatial frequencies and

low temporal frequencies (Merigan & Maunsell, 1993). Again, this is contrary to the m-pathway[2].

In the p-pathway theory of how an impaired m-pathway can hinder reading (Skottun & Parke, 1999), it is postulated that as individuals read, they make a series of fixations and saccades (i.e., rapid eye movements). The p-pathway is stimulated by the fixations while the m-pathway is stimulated by the saccades. In individuals without dyslexia, the p-pathway is believed to be suppressed by the m-pathway during each saccade. Neural activity during one fixation is thus prohibited from continuing into the next and visual confusion is prevented. Visual confusion is the phenomenon in which images of two different objects fall onto the same spot on the retinas of the eyes and become integrated. When this confusion is prevented, letters on a page do not appear to overlap or shift.

In individuals with dyslexia, however, the p-pathway is not suppressed by the impaired m-pathway at each saccade. Consequently, these individuals experience confusion while reading (Skottun & Parke, 1999) and tend to complain that letters overlap or move around. For various reasons this theory has fallen out of favour (see Stein & Walsh, 1997). For example, it has been found that the m-pathway, versus the p-pathway, is suppressed during each saccade (Burr, Morrone, & Ross, 1994).

An alternative theory for how an impaired m-pathway can disrupt reading has been proposed. In this theory, the m-pathway plays a role in binocular (two eyes) control and binocular stability (Stein, 2001; Stein & Walsh, 1997). According to this theory, individuals without dyslexia have m-pathways that keep the left and right eyes stabilized during fixation. As a result, these individuals perceive only one image of text. In contrast, individuals with dyslexia have m-pathway impairments that do not keep the two eyes stabilized during fixation. As a

[2] It should be noted that the differences between the p-pathway and the m-pathway are not clear-cut.

result, these individuals perceive overlapping images of text that make it difficult to read (Stein, 2001; Stein & Walsh, 1997). This binocular instability theory is supported by research indicating that monocular occlusion, in which one eye is covered, helps individuals with dyslexia to read (e.g., Stein & Fowler, 1985; Stein, Richardson, & Fowler, 2000). By covering one eye, a competing image of what is seen on a page is eliminated. This then prevents visual confusion.

Evidence for a Visual Magnocellular Deficit in Dyslexia

As mentioned earlier, Martin and Lovegrove (1987) discovered transient subsystem deficits, implying magnocellular deficits, in children with SRD. Following this, Livingstone, Rosen, Drislane, and Galaburda (1991) examined the LGN in autopsy specimens. The brains came from: (1) individuals who had been diagnosed with dyslexia during life and (2) controls who had been tested enough during life to preclude a diagnosis of dyslexia. These researchers found group differences in the magnocellular layers of the LGN. The layers were more disorganized and the neurons had smaller cell bodies in the individuals with dyslexia than in those without the disorder. This was evidence for a magnocellular deficit in the individuals with dyslexia: smaller cell bodies suggest thinner axons, which suggest slower conduction velocities. Livingstone et al. (1991) did not find any group differences in the parvocellular layers of the LGN.

Eden et al. (1996) conducted a functional magnetic resonance imaging (fMRI) study to further investigate whether there are m-pathway problems in dyslexia. Here, individuals with dyslexia and controls viewed two types of stimuli: a coherently moving, low contrast, random dot stimulus designed to target the m-pathway and a stationary, high contrast, pattern stimulus designed to target the p-pathway. Eden et al. (1996) found no activation in area MT and a decreased response in other motion sensitive areas for the random dot stimulus in individuals

with dyslexia. This was in comparison to the activation seen in controls. However, similar activation in the p-pathway occurred for the pattern stimulus in both groups. These results point to a specific deficit in the m-pathway in individuals with dyslexia.

Talcott et al. (1998) reached a similar conclusion. They presented their participants, adults with and without dyslexia, with a motion coherence task and a critical flicker fusion task. Critical flicker fusion is the highest frequency at which flicker can be detected, when the luminance contrast is 100%. Talcott et al. (1998) found that the individuals with dyslexia were less sensitive than controls for detecting coherent motion in the motion coherence task and for detecting high frequency flicker in the critical flicker fusion task. In addition, more than 72% of the individuals with dyslexia could be characterized as having a visual problem on the basis of their performance on the two tests. Only 17% of controls could be characterized as such.

Fast forward a few years later and it can be seen that research on magnocellular deficits in dyslexia is still being done. This suggests that the proposal of an m-pathway deficit in the disorder is tenable, despite the conventional phonological theory. In 2010, Wang, Bi, Gao, and Wydell examined the m-pathways of Chinese-speaking children with dyslexia. They recruited three groups of children: (1) children with dyslexia, (2) children who were average readers and of the same chronological age as the children with dyslexia, and (3) children who were average readers and of the same reading level as the children with dyslexia. Children in the third group were younger than the children in the other two groups (i.e., the children with dyslexia and the children of the same chronological age as the children with dyslexia). In one part of the study, all participants viewed gratings that moved and had to indicate whether the stimuli moved upwards or downwards. Wang et al. (2010) found that the children with dyslexia responded significantly slower than the age-matched group but similar to the reading matched group. This result

indicated that the children with dyslexia were performing at a level appropriate for a younger group. The children with dyslexia were also less accurate in their responses than the other two groups. The pattern of results suggested a magnocellular deficit in the group with dyslexia.

Evidence against a Visual Magnocellular Deficit in Dyslexia

Given the decades of research that has been done on visual deficits in dyslexia, one may wonder why the **visual magnocellular proposal** is not as popular as the phonological model of dyslexia. One explanation for this may be the subset of findings suggesting that individuals with the disorder lack any visual m-pathway impairments. Johannes, Kussmaul, Munte, and Mangun (1996), for example, did not find any differences in visual processing between young adults with dyslexia and controls to stimuli that were designed to target the magnocellular pathway (i.e., low contrast, rapidly reversing). If individuals with dyslexia have an m-pathway problem, then they should process the visual information differently than controls. Interestingly, however, Johannes et al. (1996) conceded that their electrophysiological techniques may not have been powerful enough or of the proper design to detect low-level visual abnormalities. As a result, they state: "…our results do not support, but at the same time do not exclude, early visual deficits in dyslexia" (p. 1126).

Amitay, Ben-Yehudah, Banai, and Ahissar (2002) similarly found that the majority of the reading disabled (RD) individuals in their study did not show impairments on tasks targeting the m-pathway. According to the visual magnocellular proposal, they should have been impaired. This result may seem contrary to what is being presented in this paper, until the following is noted. Amitay et al. (2002) divided the heterogeneous group of reading disabled participants into those who were RD-poor and RD-good based on their performance on the tasks. They found that the reading disabled participants who were RD-poor were impaired on all of the magnocellular

tasks. This result demonstrates that a reading disability cannot be fully accounted for by a phonological model and that there is room for a visual deficit explanation.

Further critics of a visual deficit in dyslexia include Skoyles and Skottun (2004). They point out that there are individuals with dyslexia who do not have any magnocellular deficits and individuals without dyslexia who do. However, they also mention that m-pathway deficits could account for a limited number of cases with dyslexia. This again implies that a phonological explanation for dyslexia may not accurately capture all cases of dyslexia and that a visual deficit account may be necessary. As will be discussed next, the results of Amitay et al. (2002) and Skoyles and Skottun (2004), while seemingly contradictory to this paper, can be explained in this paper. It is suggested here that dyslexia alone does not involve a magnocellular problem, but that dyslexia plus a co-morbid disorder do.

The Role of Co-Morbidity between Dyslexia and Attention Deficit Hyperactivity Disorder

In sum, there are two lines of opposing research: (1) research that suggests an m-pathway problem in dyslexia and that this problem can lead to reading difficulties and (2) research that does not suggest an m-pathway disturbance. Even though many studies support the visual magnocellular proposal (Eden et al., 1996; Livingstone et al., 1991; Martin & Lovegrove, 1987; Talcott et al., 1998; Wang et al., 2010), the relationship between reading difficulties and m-pathway deficits remains tentative and controversial. One reason for the differences between findings may be the diversity of dyslexia cases that are examined in the same sample. In most studies, individuals with dyslexia are treated as a homogenous group to be compared against a control group (Johannes et al., 1996). Such individuals, however, are quite diverse in that some of them have dyslexia and a co-morbid disorder and some of them have only dyslexia.

Multiple studies (August & Garfinkel, 1990; Germano, Gagliano, & Curatolo, 2010; Gilger, Pennington, DeFries, 1992) have in fact demonstrated that dyslexia is often co-morbid with **attention-deficit/hyperactivity disorder (ADHD)**. ADHD is a neurodevelopmental disorder characterized by persistent inattention and/or hyperactivity-impulsivity. These features are maladaptive and inconsistent with developmental level (Criterion A of ADHD in DSM-IV-TR; American Psychiatric Association, 2000). According to Germano et al. (2010), the rate of reading disorder in samples of individuals with ADHD ranges from 18% to 45%. In samples of those with reading disorder, 18% to 42% meet the criteria for ADHD too.

In the present study, emphasis was placed on the co-morbidity between dyslexia and ADHD. The presence of an additional diagnosis of ADHD may explain why some vision research has found magnocellular deficits in dyslexia and why some has not. It is possible that only the individuals with both dyslexia and ADHD have visual m-pathway problems while individuals with just dyslexia are spared. Previous research has not placed much focus on this. To understand the importance of ADHD to dyslexia, it is necessary to consider a third disorder, schizophrenia. **Schizophrenia** is a disorder in which there are characteristic symptoms such as delusions, hallucinations, disorganized speech, catatonic behavior, affective flattening, alogia, and/or avolition (Criterion A of schizophrenia in DSM-IV-TR; American Psychiatric Association, 2000). Research has shown that individuals with schizophrenia have attention deficits, similar to those seen in individuals with ADHD, and that they also have visual m-pathway problems (Kairalla et al., 2008; Marchetta, Hurks, De Sonneville, Krabbendam, & Jolles, 2008; Rund, Oie, & Sundet, 1996; Schechter et al., 2005; Schwartz, Maron, Evans, & Winstead, 1999).

The Role of Co-Morbidity between ADHD and Schizophrenia

Research indicates that there is a magnocellular deficit in individuals with schizophrenia. Butler et al. (2001) for example, used steady-state visual-evoked potentials and found deficits in lower-level visual processing in schizophrenia, which were reflective of deficits in the m-pathway. Schwartz et al. (1999) also demonstrated deficits in this pathway in schizophrenia through a task in which the central feature was motion-related. Other researchers (Revheim et al., 2006) have not only come to the same conclusion of a magnocellular deficit, but have also found that this deficit is related to reading impairments in schizophrenia. These schizophrenia studies are critical for the support of a magnocellular deficit in dyslexia.

Specifically, some studies have found magnocellular deficits in dyslexia and some have not. In those that have, the researchers may have used a sample containing individuals with dyslexia who also had ADHD. Why ADHD would be an important co-morbid disorder to consider is based on schizophrenia research: This research has identified magnocellular deficits in individuals with schizophrenia. Such individuals, as it turns out, tend to have attention deficits or co-morbidity with ADHD (see Figure 2).

Similar to individuals with ADHD, individuals with schizophrenia have attention deficits. For instance, Breton et al. (2011) found that there is a deficit in the executive control of attention in schizophrenia. Kairalla et al. (2008) also found that there are problems with sustained attention and selective attention in the disorder. Parallel to this finding, there are problems with sustained attention in adults with ADHD (Marchetta et al., 2008). Furthermore, in a sample of 83 children with schizophrenia/schizoaffective disorder, ADHD was the most frequent co-morbid condition: it occurred in 84% of the children (Ross, Heinlein, & Tregellas, 2006).

Overall, then, there are magnocellular deficits in schizophrenia. Because this disorder shares a critical feature with ADHD (i.e., attention deficits), this suggests that there may be magnocellular deficits in ADHD too. As mentioned before, ADHD is co-morbid with dyslexia. This can thus explain why some researchers have found an m-pathway deficit in dyslexia while others have not: It is only the individuals with both dyslexia and ADHD who may have magnocellular problems while those with just dyslexia are spared (see Figure 2). Previous studies on this topic have not emphasized this nor have they focused on examining dyslexia as a disorder with and without co-morbidity (even though this is acknowledged at times). Instead, individuals with dyslexia are often treated as a homogeneous group to be compared against a control group.

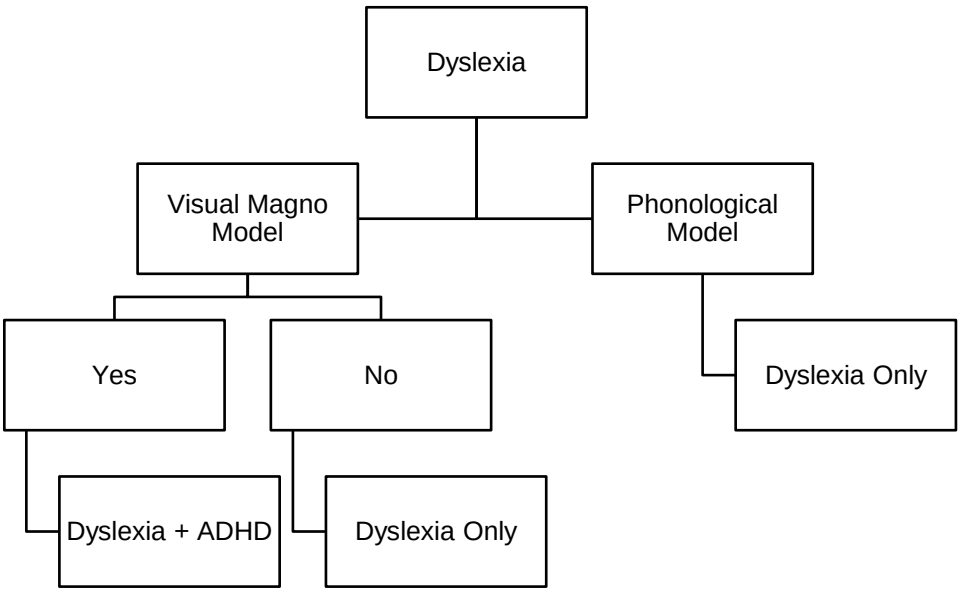

Figure 2. Theoretical account of dyslexia advocated in this paper.

Clarifications and Research Purposes

It is important to clarify the stance of the present paper: It is not argued here that the visual magnocellular proposal is more "correct" than the phonological model of dyslexia or that the proposal should completely replace the model. Rather, it is argued that both visual magnocellular and phonological problems explain the reading level observed in dyslexia, depending on the presence of co-morbidity with ADHD. The phonological model, in its purest form, does not take into account the visual problems that have been found in some individuals with dyslexia. Indeed, much research has identified m-pathway deficits in this disorder. The visual magnocellular proposal may serve as the better explanation for the reading impairment seen in a subset of individuals with dyslexia (i.e., those with both dyslexia and ADHD).

There were two purposes of this study. The first purpose was to determine whether magnocellular deficits occur in dyslexia that is co-morbid with ADHD or in dyslexia only. If magnocellular deficits occur only in dyslexia that is co-morbid with ADHD, the second purpose was to determine if similar deficits occur in ADHD only. To meet these purposes, four groups of adult participants were recruited: (1) controls, (2) individuals with dyslexia only, (3) individuals with both dyslexia and ADHD, and (4) individuals with ADHD only. Participants completed a series of m-pathway related experiments and two predictions were made. First, controls and participants with dyslexia only should perform similarly to each other while participants with both dyslexia and ADHD and participants with ADHD only should perform alike. Second, controls and participants with dyslexia only should perform differently from the participants with both dyslexia and ADHD and the participants with ADHD only.

Presumably, controls do not have any magnocellular problems. Thus, if controls and participants with dyslexia only perform similarly to each other, this would suggest that the

participants with dyslexia only do not have any magnocellular problems. If participants with both dyslexia and ADHD and participants with ADHD only perform alike (and both of these groups perform differently from the controls and participants with dyslexia only), this would suggest that they have magnocellular problems. Previous research has indicated that individuals with schizophrenia, who have attention deficits, have magnocellular impairments too.

Theory Underlying Visual Backward Masking Experiments

The experiments used to evaluate m-pathway functioning were based on **visual backward masking**, a psychophysical tool in vision research (Hermens, Luksys, Gerstner, Herzog, & Ernst, 2008). When backward masking is performed on a computer, a target stimulus is presented on a screen and is quickly followed by a masking stimulus. The aim of the masking stimulus is to interrupt the processing of the target stimulus and to reduce the visibility of it. Individuals participating in such experiments are then asked to indicate the orientation or the location of the target. Physiologically, the magnocellular and parvocellular pathways are believed to be at play. Visual backward masking has been found to involve both the transient and sustained subsystems of the primate visual system (Breitmeyer & Ganz, 1976).

As mentioned earlier, transient mechanisms are quick to respond to stimuli but demonstrate short response durations. Because of these properties, such mechanisms tend to respond to the onset, offset, and location of stimuli (Merigan & Maunsell, 1993). In backward masking, transient responses are presumed to be operating when the target stimulus first appears on the screen (Merritt & Balogh, 1989). Sustained mechanisms, on the other hand, are slow to respond to stimuli and demonstrate long response durations. Functionally, these mechanisms process the fine details of stimuli, allowing for identification (Merigan & Maunsell, 1993). In backward masking, sustained responses are believed to operate while the target remains on the

screen (Merritt & Balogh, 1989). In the last stretch of the backward masking sequence, transient mechanisms are stimulated once again by the masking stimulus that appears on the screen. Such mechanisms interrupt the sustained processing of the target, leading to a reduction in visibility for it (see Figure 3; Merritt & Balogh, 1989). When masking occurs in this way, individuals should struggle with answering questions about the orientation and location of the target. As a consequence, the response accuracy should be low.

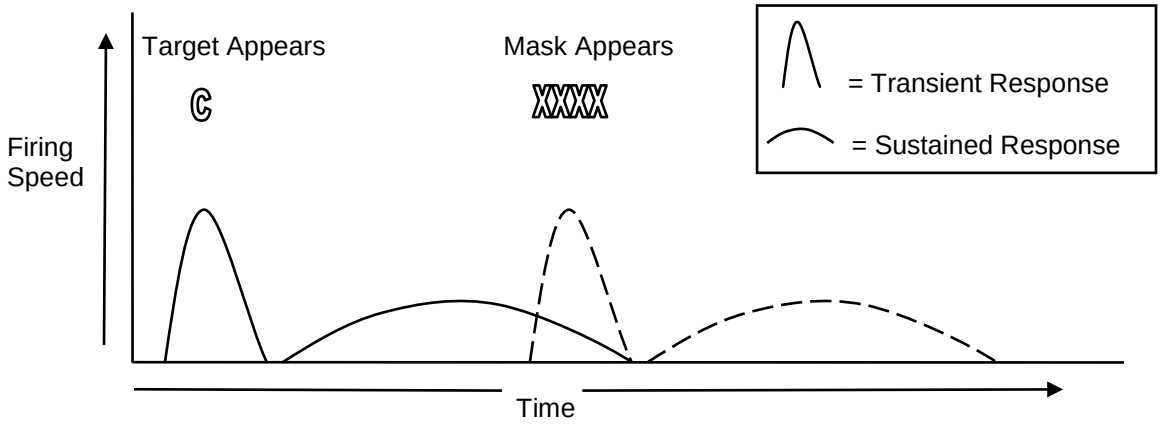

Figure 3. Schematic of backward masking on a physiological level. The transient response to the mask interrupts the sustained processing of the target.

Theoretically, if the transient subsystem or the magnocellular pathway it involves, is hypo- or hyper-vigilant in some disorders (e.g., dyslexia and ADHD, ADHD alone), then masking of the target stimulus would not happen effectively. Under this scenario, the mask would appear, but the transient response that underlies it would be too slow or too fast and as a result, would "miss" the sustained response to the target. The transient response would fail to interrupt the sustained response and visibility for the target would be high (see Figure 4 for example). When masking occurs in this way, individuals should find it easy to answer questions

about the orientation and location of the target. As a consequence, the response accuracy should be high.

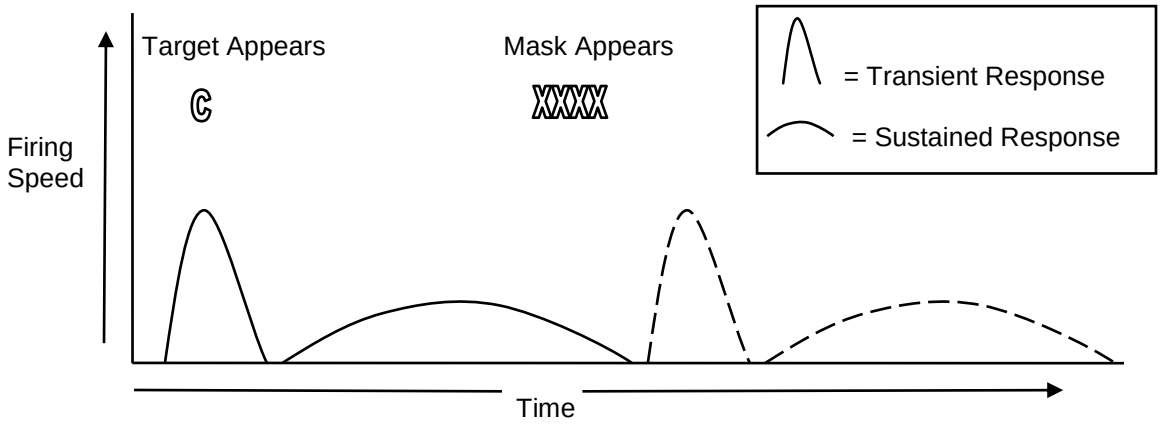

Figure 4. Schematic of backward masking on a physiological level. The hypo-vigilant transient response to the mask does not interrupt the sustained processing of the target.

Overall, by varying the time interval between the target and the mask (i.e., the **inter-stimulus interval; ISI**), it is possible to determine whether transient/magnocellular responses differ across the four groups of participants. If the four groups do not differ in magnocellular functioning, then all of them should be masked of the target (i.e., they should not see the target) at the same ISI. However, if the four groups do differ, then they should all be masked of the target at different ISIs. In particular, if the participants with attention deficits (i.e., those with both dyslexia and ADHD, those with ADHD only) are masked at a specific ISI and participants without attention deficits (controls, those with dyslexia only) are masked at a different ISI, this would suggest that the participants with attention deficits have magnocellular impairments. This conclusion would be supported by research indicating that patients with schizophrenia, who have attention deficits, have magnocellular impairments (Kairalla et al., 2008; Marchetta et al., 2008; Rund et al., 1996; Schechter et al., 2005).

In the present study, five ISIs were tested in three visual backward masking experiments. The target and mask were designed so that they maximally stimulated the magnocellular pathway over the other pathways of the visual system. This was done by ensuring, for example, that the stimuli were of low luminance contrast. The ISIs were also kept short (i.e., the target was presented and the mask followed soon after instead of long after). The backward masking experiments were modeled after those in the literature examining magnocellular deficits in schizophrenia (e.g., Bedwell, Orem, Rassovsky, Allen & Sutterby, 2009; Butler et al., 2002; Green, Nuechterlein, Breitmeyer, & Mintz, 2006).

To date, it seems that only Rund et al. (1996) has used backward masking on individuals with ADHD. These researchers, however, only tested two ISIs between the target and the mask. The present study extends this line of research by further investigating the ADHD population and by testing five ISIs. Five ISIs should be a more sensitive indicator of any m-pathway problems in individuals with attention deficits.

Method

Participants

There were four groups of adult participants over the age of 18 years in this study: controls (n = 13), individuals with dyslexia only (n = 4), individuals with both dyslexia and ADHD (n = 6), and individuals with ADHD only (n = 12). Individuals in the last three groups were formally diagnosed, by a professional, with ADHD/attention problems, dyslexia/reading problems, or both. They were recruited from the Access Centre at Ryerson University and were also personal contacts of the researcher. Individuals in the control group were personal contacts of the researcher. All participants gave informed consent and each participant was reimbursed

with $10 for costs such as travel. The age range of the sample was 19 to 36 (*M* = 25.56, *SD* = 4.48). There were 14 males and 21 females.

Measures

Screening tests.

The following tests were administered to the four groups of participants to examine their visual and cognitive abilities.

96% Regan contrast letter chart.

This chart is a measure of static visual acuity for far vision (Paragon Services, Inc.). On this chart, there are letters in black printed on a white background. The letters are printed on several lines. Letter lines are large at the top of the chart and become smaller towards the bottom of it (20/100 to 20/10 at a viewing distance of 10 feet). Participants were instructed to read each line and to stop when they made two or more errors on the same line (i.e., they failed the line). The last line they passed was recorded and the corresponding Snellen fraction was assigned. For example, if the last line a participant passed was line 8, the corresponding Snellen fraction of 20/20 was assigned (what the participant sees at 20 feet is what should normally be seen at that distance).

4% Regan contrast letter chart.

This chart is a measure of contrast sensitivity (Paragon Services, Inc.). On this chart, there are letters in light grey printed on a white background. These letters are printed on several lines. Letter lines are large at the top of the chart and become smaller towards the bottom of it (20/200 to 20/16 at a viewing distance of 10 feet). Participants were instructed to read each line and to stop when they made two or more errors on the same line (i.e., they failed the line). The last line they passed was recorded and the corresponding Snellen fraction was assigned.

Stereotest house fly.

This is a test of binocular depth perception (Stereo Optical Co., Inc.), or the ability to see in three dimensions (3D) using the left and right eyes. A picture of a fly appears in 3D when three dimensional glasses are worn. Participants were asked to pinch the wings of this fly. If their fingers remained above the plane of the picture while doing so, they passed the test (indicating they saw the fly in 3D). They were given a score of 1200 seconds of visual arc. If their fingers touched the plane of the picture while doing so, they failed the test (indicating they did not see the fly in 3D). They were given a score of 2500 seconds of visual arc.

Stereotest circles.

This test of binocular depth perception provides a finer measure of depth discrimination than the House Fly (Stereo Optical Co., Inc.). In this test, there are nine squares with each square containing four circles. Participants were asked to indicate which of the four circles appeared in 3D while wearing three dimensional glasses. The total number of correct responses out of nine was recorded and this number was converted into seconds of visual arc. If participants achieved one correct response out of nine, they were given a score of 800 seconds. If participants achieved nine correct responses out of nine, they were given a score of 40 seconds.

College ADHD response evaluation (CARE; student response inventory).

This is a 59-item questionnaire used to evaluate behaviours common to ADHD (Glutting, Sheslow, & Adams, 2002). It is designed for students at the university level. Participants were given the CARE and asked to select one response for each item (agree, undecided, or disagree). The CARE was scored so that raw scores were obtained on the inattention scale, hyperactivity scale, and impulsivity scale. Higher raw scores indicated more inattention, hyperactivity, and impulsivity compared to lower raw scores.

Nelson-Denny reading test (NDRT; form g).

The NDRT can be used to assess reading rate in a population of students in four year college/university (Brown, Fishco, & Hanna, 1993). Participants were given a passage (i.e., passage one) and were asked to read it for 1 minute. At the end of this minute, they were instructed to circle the number beside the last line they just read. This number represents the number of words on that line and on the lines before it. Accordingly, it is a measure of reading rate in number of words per minute. Although participants were told that there would be a follow-up test of comprehension, comprehension was not formally evaluated. Participants were told this to ensure they read at their regular pace and did not speed through the passage.

Symbol digit modalities test (SDMT; written administration).

The SDMT[3] can be used to measure the cognitive speed of processing in adults (Smith, 1973). The test objective is to substitute randomized presentations of geometric symbols with specific numbers. Participants were shown a legend in which each symbol was paired with a number from one to nine. They were then given 8 rows and 15 columns of symbols and 90 seconds to make as many symbol-digit substitutions as possible. The instruction was to complete these accurately and quickly. The total number of correct responses was recorded.

Useful field of view test (UFOV; processing speed subtest).

This computerized subtest of the UFOV gives a measure of the speed of visual processing (Visual Awareness Research Group, Inc.). A picture of a car or a truck appears in the center of the computer screen and is followed by a picture of visual noise. Once the visual noise ceases, there is an option to indicate whether a car or a truck was seen. Participants were asked to do this for 4 practice trials and 35 test trials. After the completion of 35 test trials, a score was given in milliseconds and this score was later evaluated against cutoff points (e.g., a score of 17

[3] The SDMT is similar to, but different from, the Digit Symbol Substitution Test (Wechsler, 1955; 1981).

milliseconds falls between the cutoff point of > 0 to ≤ 30 and represents normal central vision and processing speed). The UFOV was administered on a desktop computer with a cathode ray tube (CRT) monitor (MAG Innovision model number: 986N). Monitor viewable size was 45.47 cm.

Experiments: Stimuli features.

The following three experiments were based on the principles of backward masking, as described in the introduction section of this paper (i.e., fixation cross, target, mask, and a question or two about the target). All experiments were administered on a desktop computer with a liquid crystal display (LCD) monitor (Samsung model number: 940BW). Monitor viewable size was 48.26 cm. All stimuli sizes are reported in degrees (^{o}) of visual arc, which were calculated by dividing the size of an object by the distance at which it was seen, and then taking the inverse tangent of that. Reporting sizes in degrees indicate how large or small an object is on the retina. Luminance scores were obtained using a photometer and luminance contrasts were calculated using Michelson Contrast: $C_M = (L_{max} - L_{min}) / (L_{max} + L_{min})$. The contrasts are presented as a percentage and indicate the relationship between the luminance of a brighter area and that of an adjacent darker area. All experiments were piloted on several individuals who were not the participants.

Peripheral backward masking experiment.

The peripheral experiment was designed to assess the performance of participants on a task when the stimuli were presented peripherally (see Figure 5). Magnocellular deficits, if they exist in individuals with attention deficits, may be restricted to one part of the visual field (e.g., peripheral vs. central). Participants first viewed a fixation cross in the middle of the screen. The fixation cross was 0.14^{o} in size and the stimulus field was 19.78^{o} in width and 11.99^{o} in height. A

trial was initiated with the push of a button, and when this button was pushed, a target appeared on the screen. The target was a Landolt C, a standardized symbol in which the stroke width and gap width are one fifth the size of the diameter. This target was 0.27° in size (Bedwell, Brown, & Miller, 2003) and had a luminance contrast of 3.4% (Schechter et al., 2003). This luminance contrast was chosen to stimulate the m-pathway over the other pathways of the visual system. Research has indicated that the m-pathway responds best to low luminance contrasts (e.g., 2%) and that the p-pathway responds poorly to luminance contrasts under 10% (Merigan & Maunsell, 1993).

The target appeared in one of four orientations (the gap in the Landolt C could face the top, bottom, left, or right of the screen) and in one of four locations (the Landolt C could appear in the upper right, upper left, bottom right, or bottom left quadrant of the screen). In addition, the target appeared 2.75° away from the center of the screen (Bedwell et al., 2003) and remained on the screen for only 13 ms (Bedwell et al., 2009). After 13, 27, 33, 40, or 47 ms had elapsed (the ISIs; Bedwell et al., 2003; Bedwell et al., 2009), the mask appeared and completely covered the stimulus field with rows and columns of Xs. These ISIs were chosen because they should stimulate the magnocellular pathway (these ISIs should give rise to the perception of flicker). Each X was 0.40° in size and had a luminance contrast of 8.2% (Schechter et al., 2003). The mask was presented for only 13 ms. Next, two questions were displayed on the screen and the participants needed to report the orientation of the Landolt C and the location of it on the screen. The two questions appeared in random order. The peripheral experiment consisted of 60 trials in total, 12 trials at each of five ISIs (Bedwell et al., 2009).

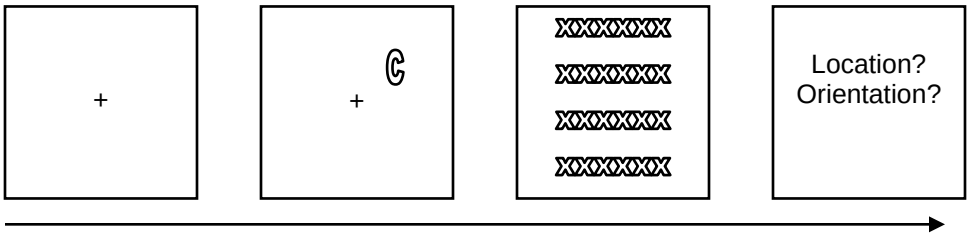

Figure 5. From left to right, example of a trial in the peripheral experiment.

Peripheral confirmatory backward masking experiment.

It can be argued that the peripheral experiment above required participants to dual task as they needed to attend to the orientation and location of the target. There may be differences, however, in the four groups of participants and their abilities to dual task effectively. The participants with attention deficits may find it more difficult to dual task than the participants without attention deficits (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Pasini, Paloscia, Alessandrelli, Porfirio, & Curatolo, 2007). Thus, if group differences are found in the peripheral experiment (e.g., controls and participants with dyslexia only perform alike, participants with both dyslexia and ADHD and participants with ADHD only perform alike, controls and participants with dyslexia only perform differently from the participants with both dyslexia and ADHD and ADHD only), these differences may be explained by two accounts: (1) the presence of magnocellular impairment in the participants with attention deficits and the absence of these impairments in the participants without attention deficits and (2) the presence of dual tasking difficulties in the participants with attention deficits and the absence of these difficulties in the participants without attention deficits.

To eliminate the dual tasking alternative explanation, a confirmatory experiment was designed in which the target appeared in one of four orientations on the screen but in one quadrant only (i.e., lower left quadrant; see Figure 6). At the end of every trial, participants

needed to indicate just the target orientation. Overall then, the confirmatory experiment did not require participants to dual task as in the peripheral experiment. Thus, if similar results are obtained across the two experiments, this would suggest that group differences (if found) could be attributed to a magnocellular account. However, if different results are obtained, this would suggest that group differences (if found) could be attributed to a dual tasking account.

The confirmatory experiment had only two ISIs (13 and 47 ms) and 24 trials (12 trials at each of two ISIs). All other stimulus features were the same as in the peripheral experiment.

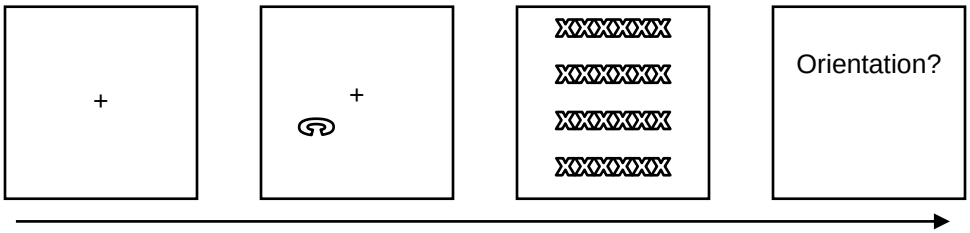

Figure 6. From left to right, example of a trial in the confirmatory experiment.

Central backward masking experiment.

The central experiment was designed to assess the performance of participants on a task when the stimuli were presented centrally (see Figure 7). Similar to the peripheral experiment, magnocellular deficits, if they exist in individuals with attention deficits, may be restricted to one part of the visual field (e.g., central vs. peripheral). Contrary to the experiments above, the target in the central experiment appeared in the center of the screen and was smaller in size at 0.13^{o}. It changed in orientation so that the gap in the Landolt C faced the top, bottom, left, or right of the screen. Accordingly, participants needed to answer only one question at the end of every trial. All other stimulus features were the same as in the peripheral experiment. The central experiment consisted of 60 trials, 12 trials at each of five ISIs.

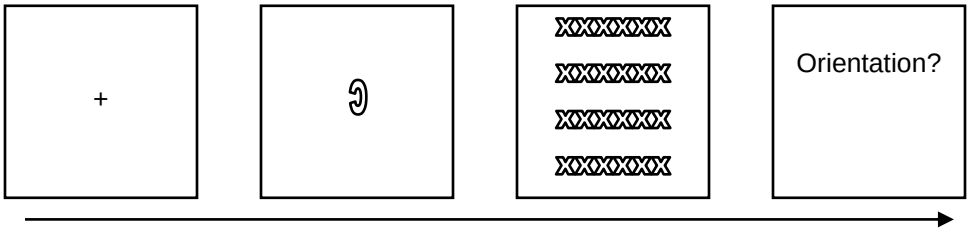

Figure 7. From left to right, example of a trial in the central experiment.

Procedure

Participants were tested one at a time by the same experimenter in the Vision Science Laboratory at Ryerson University. Before testing began, participants were reimbursed with ten dollars for their travel costs and given an overview of the consent form as well as time to read it. They were then asked a series of questions regarding the nature of their disorders. Controls were also asked these questions as a quick check to ensure they were placed in the correct experimental group. Additional questions were asked of all participants and these questions involved vision, gender, and age.

Participants next completed four of eight screening tests. The first test measured their static visual acuity for far vision using the 96% Regan Contrast Letter Chart. They were instructed to begin reading the letters on the third line, to read the letters from left to right, and to continue onto the next lines until they could no longer see the letters. The second test measured their contrast sensitivity using the 4% Regan Contrast Letter Chart. Participants received the same instructions as for the 96% chart, except that they were to begin reading the letters on the first line. For both of these tests, participants were seated ten feet away from the charts. As for the third and fourth tests that measured depth perception, participants were required to wear 3D glasses. They first completed the stereo fly, in which they had to pinch the 3D wings of a house

fly picture. They then completed the stereo circle, where they had to indicate which of four circles in each of nine panels "popped out" or appeared in three dimensions.

After these four screening tests, participants moved on to complete the peripheral experiment. Participants were fitted with a chin rest and seated 114 cm away from the computer monitor. They were informed that they would see several images on the computer screen. The first image would be a little cross (i.e., fixation cross) and whenever they saw the cross, they were to stare at the center of it. Then, when they were ready, the experimenter would press the space bar on the computer keyboard to initiate a trial. They were told that the letter C (i.e., target) would appear in one of four orientations. The experimenter indicated on a piece of paper the four different orientations the C could be in. The experimenter next told participants that the letter C would be in one of four quadrants on the computer screen (i.e., different locations). The experimenter drew imaginary quadrants on the screen and pointed specifically to the area in each quadrant the C would appear in. Participants were told that the C would not be anywhere in the far corners. The experimenter summarized this information by clarifying that there would only be one C in one orientation in one quadrant.

Participants were then told that the C was a certain size (the experimenter drew the size on a piece of paper), a certain shade of grey (the experimenter pointed to the 4% Regan Contrast Letter Chart and said it would be about the same shade), and on the screen for a short amount of time. They were then told that after the C went off the screen, a series of X's would appear and cover the entire screen (i.e., mask). They were then instructed that the X's would only be on the screen for a short amount of time. The experimenter summarized the sequence of events thus far by clarifying that once the X's were seen, the C would no longer be there.

After that, participants were told that two response boxes would appear on the screen and that they would need to indicate where the C was on the screen and in which orientation it was in. They were shown a printed screenshot of the response boxes and further told that the boxes would change order so that they would sometimes need to respond to orientation before location and to location before orientation. Participants responded orally and the experimenter entered their responses into the computer. If they did not perceive the orientation or location of the letter C, they were encouraged to make a guess. They completed at least four practice trials to become familiar with the peripheral experiment. If there were no questions or concerns, they then completed 60 trials. They could request a break at any point before the experimenter initiated another trial.

After the peripheral experiment ended, participants completed the confirmatory experiment. The experimenter provided similar instructions as in the peripheral experiment, except that the letter C would appear in only one quadrant in all 24 trials. The experimenter demonstrated this by displaying a printed screenshot of the response box for orientation and putting away the screenshot of the response box for location. The experimenter emphasized the importance of focusing on the little cross even after discovering which quadrant the C would appear in. Participants received no practice trials for the confirmatory experiment.

Participants then received a formal break, after which they completed the remaining four tests of the screening battery. The first of these tests was the CARE survey. The experimenter read to participants the instructions at the top of the survey and indicated what the three response options were. Once participants were done with this, they moved on to the Nelson Denny test. The experimenter instructed them to read the passage at their regular reading pace and to read the passage so that they understood it, as they would be quizzed on the content of it afterwards (even

though they were not). Following this, participants moved on to the Symbol Digits test. The experimenter explained to them that they were to substitute symbols on the page with the corresponding digits. They were given time to practice using the substitution key. Participants next completed the final screening test, the UFOV, on a computer. They were seated 24 inches away from the computer screen. The experimenter explained that they would see either a car or a truck on the screen and that their task would be to indicate which they saw. Participants practiced the task a few times before beginning the actual test. They responded orally and the experimenter entered their responses into the computer for them.

Following these screening tests, the focus of participants was brought back to the backward masking experiments. The experimenter started them on the central experiment, which was the final activity of the test session. Participants received the same instructions as in the peripheral experiment except for a few changes. They were told that as before, they would see a cross in the middle of the screen and to fixate on it. However, when a trial was initiated, the cross would disappear and be replaced by the target letter C. The experimenter emphasized the central location of the C by pointing at the center of the screen. The experimenter mentioned that the C would again appear in one of four orientations but be smaller in size than the C they saw in the peripheral and confirmatory experiments. The C would continue to be the same shade of grey and on the screen for only a short amount of time. The experimenter further explained that after the C went off the screen, a series of X's would appear, and then the response box for orientation only. Participants received at least 4 practice trials before completing the central experiment with 60 trials. They could request a break at any point before the next trial was initiated. Once participants completed the central experiment, the test session ended. They were given a debriefing form with contact information.

Results

Review of Research Purposes and Predictions

There were two purposes in this study. The first purpose was to determine whether magnocellular deficits occur in dyslexia that is co-morbid with ADHD or in dyslexia only. If magnocellular deficits occur in dyslexia that is co-morbid with ADHD, the second purpose was to determine if similar deficits occur in ADHD only. To achieve these purposes, four groups of participants were recruited and two general predictions were made. First, controls and participants with dyslexia only should perform similarly to each other while participants with both dyslexia and ADHD and participants with ADHD only should perform alike. Second, controls and participants with dyslexia only should perform differently from the participants with both dyslexia and ADHD and participants with ADHD only.

Presumably, controls do not have any magnocellular problems. Thus, if controls and participants with dyslexia only perform similarly to each other, this would suggest that the participants with dyslexia only do not have any m-pathway problems. If participants with both dyslexia and ADHD and participants with ADHD only perform alike, this would suggest that they have magnocellular problems as previous research has indicated that individuals with schizophrenia have attention deficits (similar to those seen in individuals with ADHD) and magnocellular impairments (Kairalla et al., 2008; Marchetta et al., 2008; Rund et al., 1996; Schechter et al., 2005). Before the results for the backward masking experiments are provided, results for the screening tests are presented.

1. Screening Tests

Screening tests were administered to all participants to obtain some descriptive information about them. The means and standard deviations of each of the screening tests for each of the four groups are presented in Table 1. Overall, there were no significant differences between the groups on tests of acuity, contrast, depth perception, reading rate, and visual processing speed ($p > 0.05$). In contrast, there were significant differences between the groups on tests of inattention, hyperactivity, impulsivity, and cognitive processing speed ($p < 0.05$).

Table 1

Group means and standard deviations on each of the screening tests. All comparisons, marked by corresponding letters, were significant at the 0.05 level. For each comparison, a confidence interval was computed. A comparison was significant if the lower bound and upper bound of the confidence interval did not cross zero

Screening Tests *M* (*SD*)	Control (n = 13)	Dyslexia (n = 4)	Dyslexia + ADHD (n = 6)	ADHD (n = 12)
96% Acuity	20/14.15 (3.47)	20/16.25 (4.33)	20/15.75 (4.00)	20/15.04 (3.49)
4% Contrast	20/45.92 (13.23)	20/55.75 (19.47)	20/55.67 (12.71)	20/49.67 (12.92)
Stereo Fly	1200 (0.00)	1200 (0.00)	1200 (0.00)	1200 (0.00)
Stereo Circle	40.00 (0.00)	40.00 (0.00)	40.00 (0.00)	49.17 (28.75)
CARE: Inattention	10.62 (6.01)[a,b]	16.25 (7.23)	20.67 (4.76)[b]	22.67 (4.14)[a]
CARE: Hyperactivity	5.85 (6.20)[c,d]	11.75 (9.78)	20.00 (1.79)[d,e]	13.25 (5.40)[c,e]
CARE: Impulsivity	5.92 (5.38)[f]	7.25 (6.02)	14.83 (7.73)	12.75 (6.18)[f]
NDRT	206.39 (46.77)	137.00 (28.00)	156.83 (49.15)	219.83 (105.16)
SDMT	68.54 (9.48)[g]	54.50 (3.70)[g]	57.20 (7.83)	60.33 (7.44)
UFOV	17.00 (0.00)	17.00 (0.00)	17.00 (0.00)	17.00 (0.00)

a. Significant difference between controls and individuals with ADHD only in inattention scores

b. Significant difference between controls and individuals with both dyslexia and ADHD in inattention scores

c. Significant difference between controls and individuals with ADHD only in hyperactivity scores

d. Significant difference between controls and individuals with both dyslexia and ADHD in hyperactivity scores

e. Significant difference between individuals with both dyslexia and ADHD and individuals with ADHD only in hyperactivity scores

f. Significant difference between controls and individuals with ADHD only in impulsivity scores

g. Significant difference between controls and individuals with dyslexia only in SDMT scores

2. Individual Differences in Best Masking across Experiments

To illustrate the predictions of this study, each of the following line graphs was created to depict the performance of a single participant from one of the four groups on one of the experiments. The graphs were not created to represent the overall performance of each group. Performance was defined in terms of the percentage of correct answers at each ISI to either: (1) the location of the target when it appeared peripherally, (2) the orientation of the target when it appeared peripherally, or (3) the orientation of the target when it appeared centrally.

It should be noted that the line graphs were designed to have proportional y-axes (i.e., different y-axes) to better reflect the individual data. As a consequence, focus should not be paid to the height of the line in each graph, but rather, to when the dips and peaks of the line occur (i.e., at what ISIs). The lowest dip, found at a particular ISI, was the point at which a participant scored the least number of correct answers. This indicated that the participant did not see the target often and thus experienced **best masking**. The highest peak, found at a particular ISI, was the point at which a participant scored the most number of correct answers. This indicated that the participant saw the target often and thus experienced **worst masking**. The line graphs below were divided into subsections, with each subsection representing a specific experiment.

Peripheral backward masking experiment (target appeared in one of four quadrants on the screen and in one of four orientations).

Peripheral target location.

In Figure 8, performance was the percentage of correct answers at each of five ISIs to the location of the target when it appeared peripherally. The control participant (A.C.) was best masked at a high ISI (40 ms) and was worst masked at a low ISI (27 ms). Similarly, the participant with dyslexia only (K.K.W.) was best masked at a high ISI, 40 ms, and worst masked at a lower ISI, 33 ms. The participant with both dyslexia and ADHD (C.B.) and the participant with ADHD only (W.L.) showed the opposite results: C.B. was best masked at a low ISI, 27 ms, and worst masked at the highest ISI, 47 ms. W.L. was best masked at the lowest ISI, 13 ms, and worst masked at higher ISIs, 33 and 47 ms. This pattern, from the target location aspect of the peripheral experiment, was in line with the general predictions made.

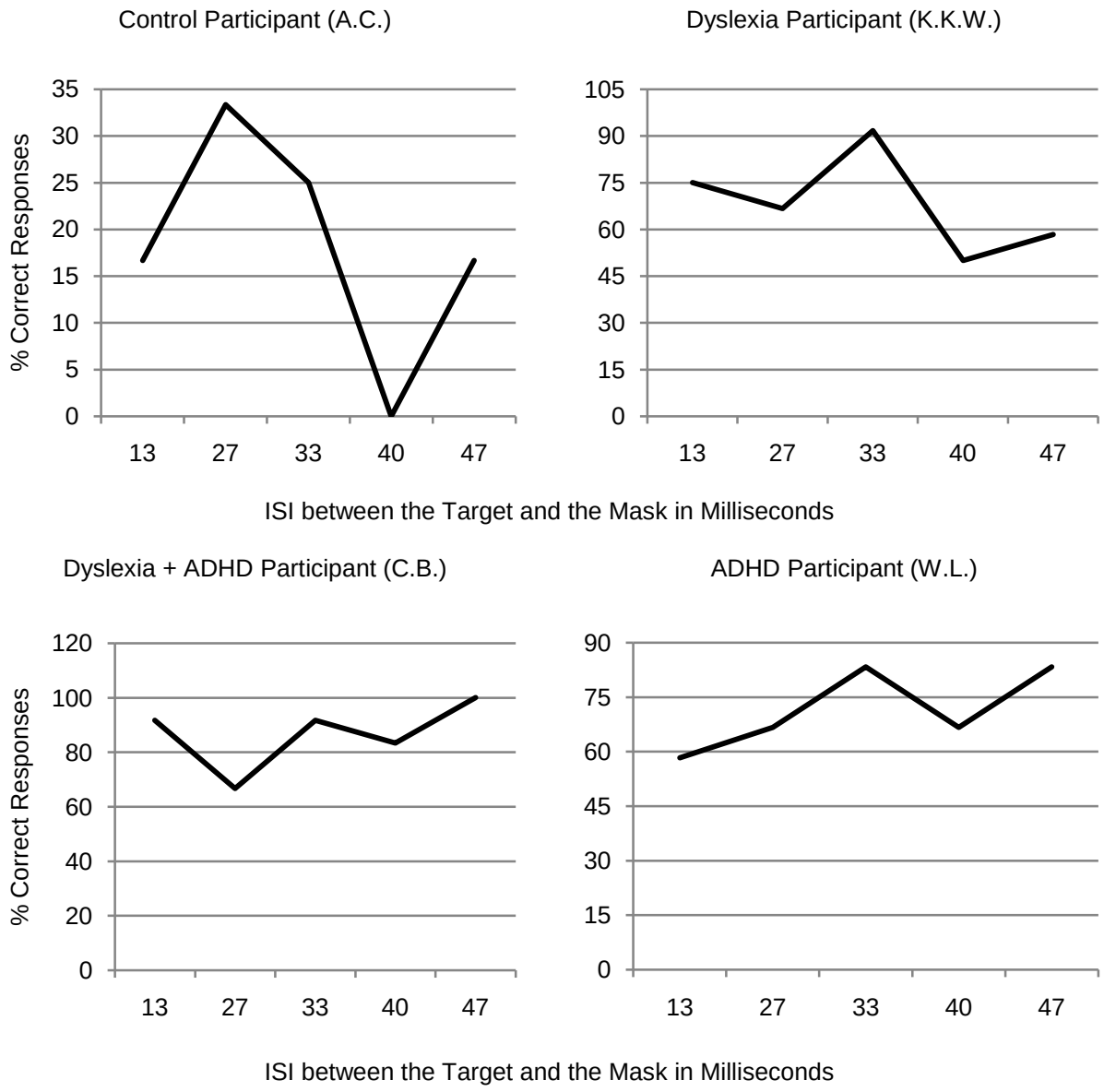

Figure 8. Performance of a control, a participant with dyslexia only, a participant with both dyslexia and ADHD, and a participant with ADHD only on the target location aspect of the peripheral experiment.

Peripheral target orientation.

In Figure 9, performance was the percentage of correct answers at each of five ISIs to the orientation of the target when it appeared peripherally. As before, the control (J.W.) and the participant with dyslexia only (K.K.W.) were best masked at high ISIs (40 to 47 ms). They were worst masked at lower ISIs (13 to 33 ms). The participant with both dyslexia and ADHD, K.N., and the participant with ADHD only, I.S., demonstrated the opposite finding: they were best masked at the lowest ISI (13 ms) and worst masked at a higher ISI (33 ms). This pattern, from the target orientation aspect of the peripheral experiment, was in line with the general predictions made.

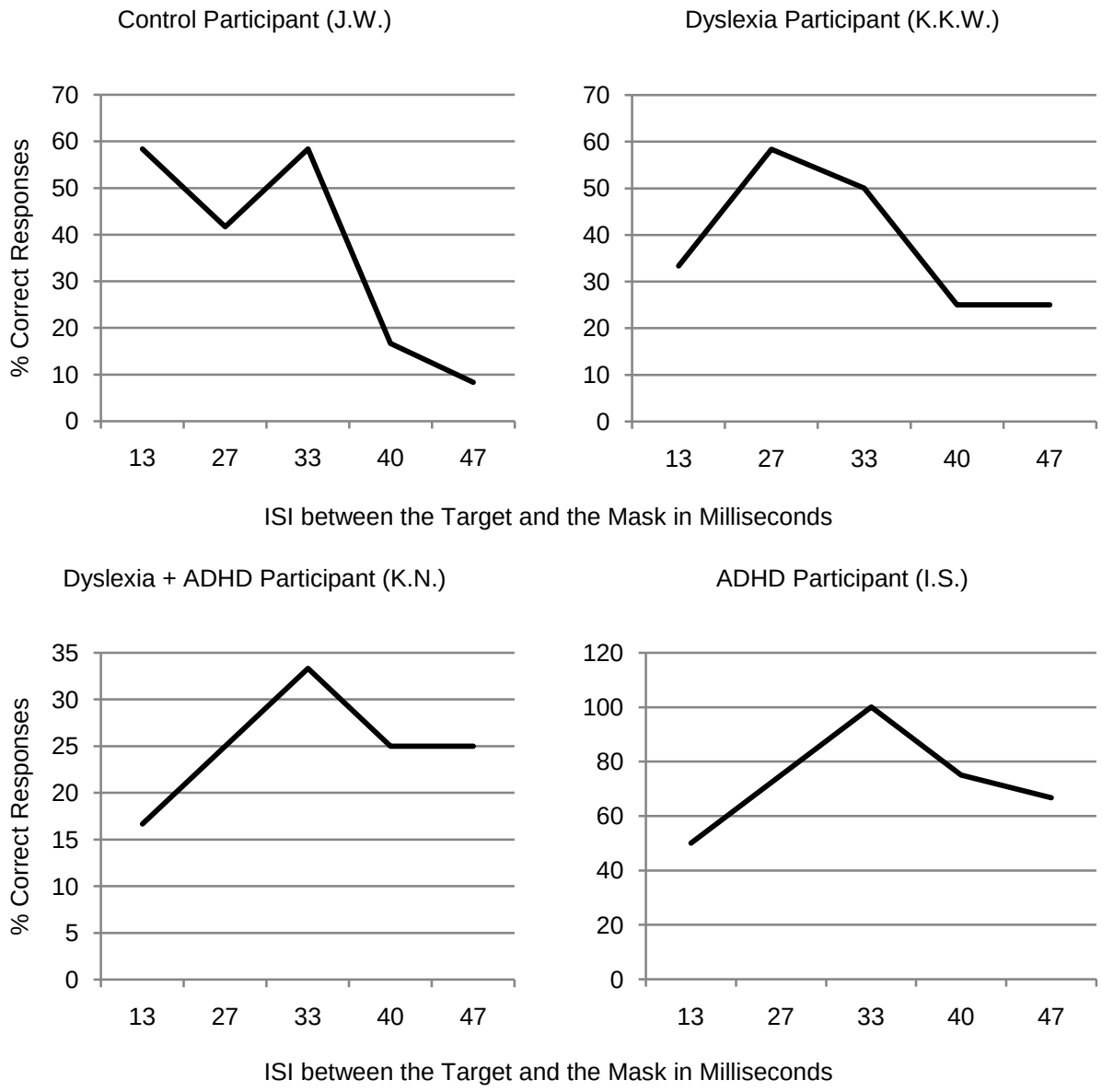

Figure 9. Performance of a control, a participant with dyslexia only, a participant with both dyslexia and ADHD, and a participant with ADHD only on the target orientation aspect of the peripheral experiment.

Peripheral confirmatory experiment (target appeared in one quadrant on the screen and in one of four orientations).

Peripheral target orientation.

In Figure 10, performance was the percentage of correct answers at each of two ISIs to the orientation of the target when it appeared peripherally. The control (S.M.) and the participant with dyslexia only (A.L.G.) were best masked at the highest ISI (47 ms) and were worst masked at the lowest ISI (13 ms). In contrast, the participant with both dyslexia and ADHD (A.F.) and the participant with ADHD only (M.N.) were best masked at the lowest ISI and were worst masked at the highest ISI. This pattern, from the confirmatory experiment, was in line with the general predictions made.

In addition, the pattern of masking in the confirmatory experiment was similar to the pattern of masking in the target orientation aspect of the peripheral experiment. Because the results of the two experiments matched, individual differences may be accounted for by the presence of magnocellular impairments in the participants with attention deficits and the absence of these impairments in the participants without attention deficits (rather than the presence of dual tasking difficulties in the participants with attention deficits and the absence of these difficulties in the participants without attention deficits).

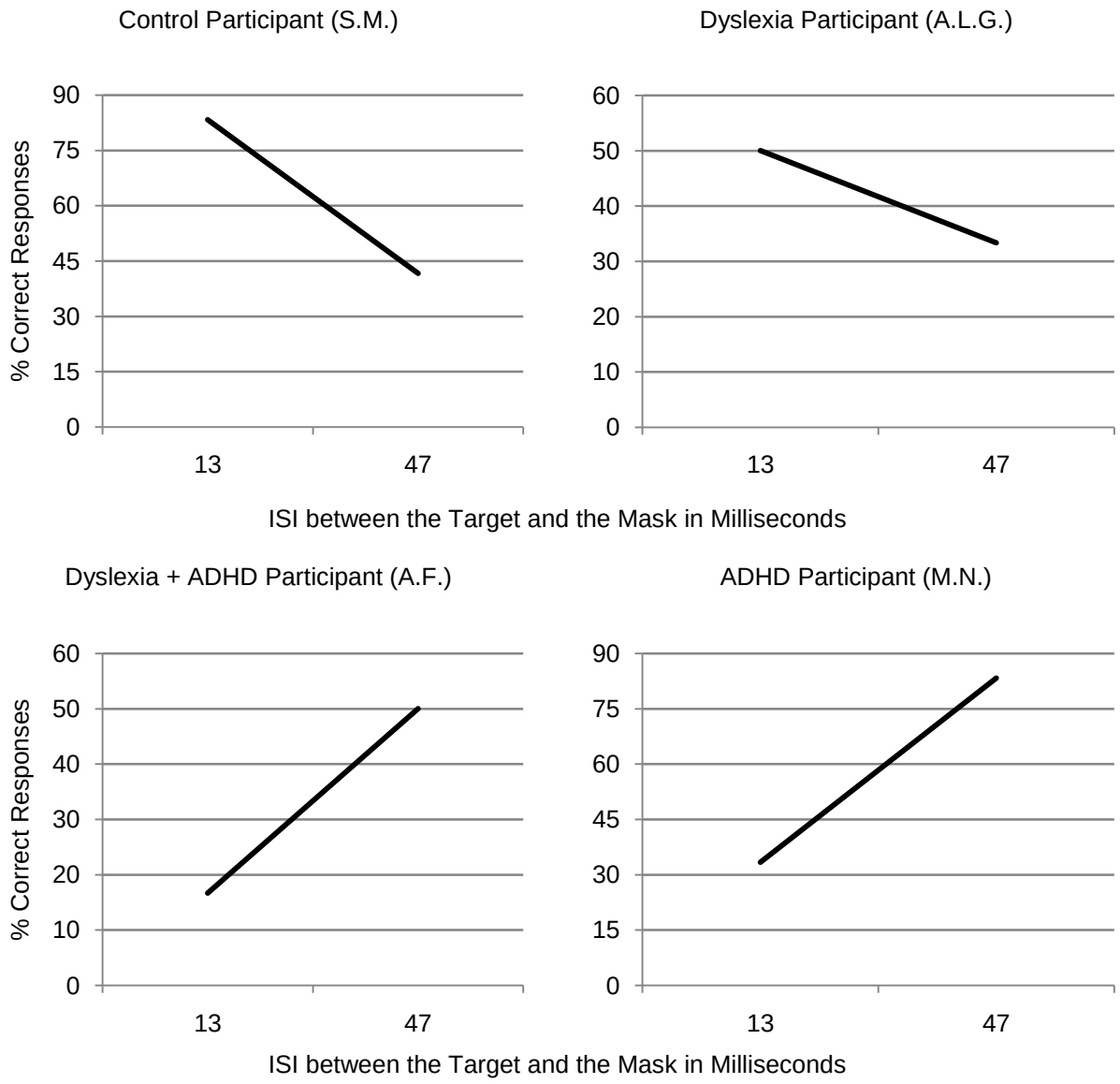

Figure 10. Performance of a control, a participant with dyslexia only, a participant with both dyslexia and ADHD, and a participant with ADHD only on the confirmatory experiment.

Central backward masking experiment (target appeared in the center of the screen and in one of four orientations).

Central target orientation.

In Figure 11, performance was the percentage of correct answers at each of five ISIs to the orientation of the target when it appeared centrally. The control (S.O.) and the participant with dyslexia only (K.K.W.) were best masked at the highest ISI (47 ms) and were worst masked at lower ISIs (33 to 40 ms). In contrast, the participant with both dyslexia and ADHD (C.B.) and the participant with ADHD only (S.B.) were best masked at the lowest ISI (13 ms) and were worst masked at higher ISIs (33 to 40 ms). This pattern, from the central experiment, was in line with the general predictions made.

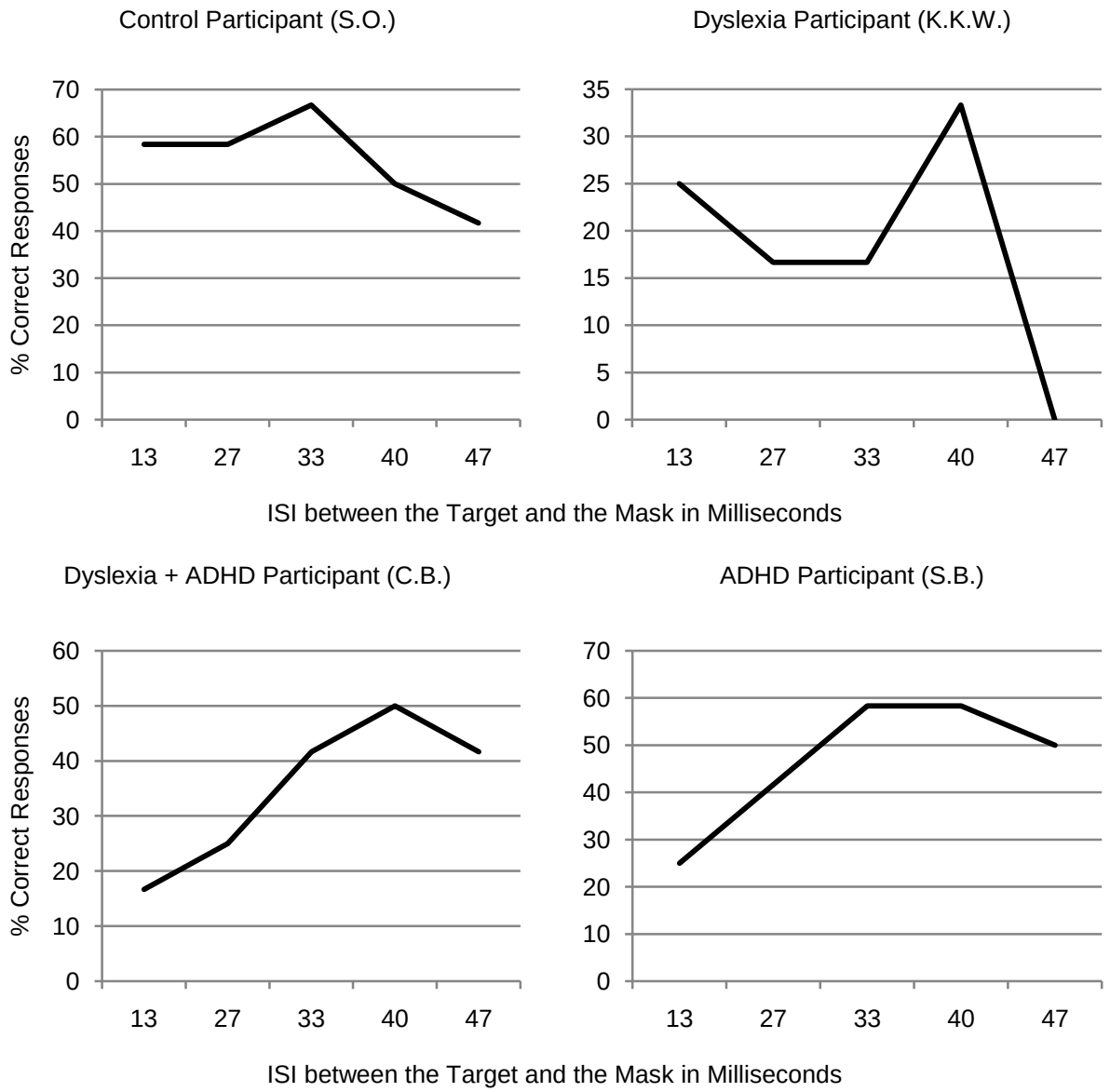

Figure 11. Performance of a control participant, a participant with dyslexia only, a participant with both dyslexia and ADHD, and a participant with ADHD only on the central experiment.

Summary.

Altogether, the line graphs demonstrated that individual differences in masking could be achieved and that these differences were in line with predictions. Analyses were next conducted to see if group differences could be found.

3. Group Differences in Masking across Experiments

The objective of the following analyses was to see if there were any differences between the four groups in the number of correct answers achieved at each ISI. The number of correct answers referred to either: (1) the location of the target when it appeared peripherally, (2) the orientation of the target when it appeared peripherally, or (3) the orientation of the target when it appeared centrally. Differences between the four groups in the number of correct answers achieved at each ISI could indicate differences between the four groups in masking at each ISI. For instance, it was specifically predicted that controls and participants with dyslexia only would score smaller correct answers at a certain ISI and that participants with both dyslexia and ADHD and participants with ADHD only would score smaller correct answers at a different ISI. This would suggest that controls and participants with dyslexia only were more likely to be masked at a different time interval than those with both dyslexia and ADHD and those with ADHD only. This, in turn, would suggest that the transient systems, or the m-pathways, of participants with both dyslexia and ADHD and participants with ADHD only were problematic in some way (e.g., that the pathways were hypo- or hyper-vigilant at processing relevant stimuli). Such conclusions would be supported by research showing that individuals with schizophrenia, who have attention deficits, have m-pathway deficits as well.

Framed in terms of the two research purposes, finding this pattern of results would suggest: (1) m-pathway impairments were not present in the individuals with dyslexia only but rather, were present in the individuals with both dyslexia and ADHD and (2) similar to the m-pathway impairments in the individuals with both dyslexia and ADHD, there were impairments in the individuals with ADHD only. On a broader scale, this would then show that the visual

magnocellular proposal could account for some cases of dyslexia and that the phonological model could not account for all cases of dyslexia.

The following analyses involved parametric two-way mixed analyses of variances (ANOVAs; see Figure 12). In all ANOVAs, the between-subjects factor was group (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only) and the within-subjects factor was ISI (13, 27, 33, 40, and 47 ms). Focus should be placed on the interactions between group and ISI, as these interactions addressed the objective above. Results were divided into subsections, with each subsection representing a particular experiment.

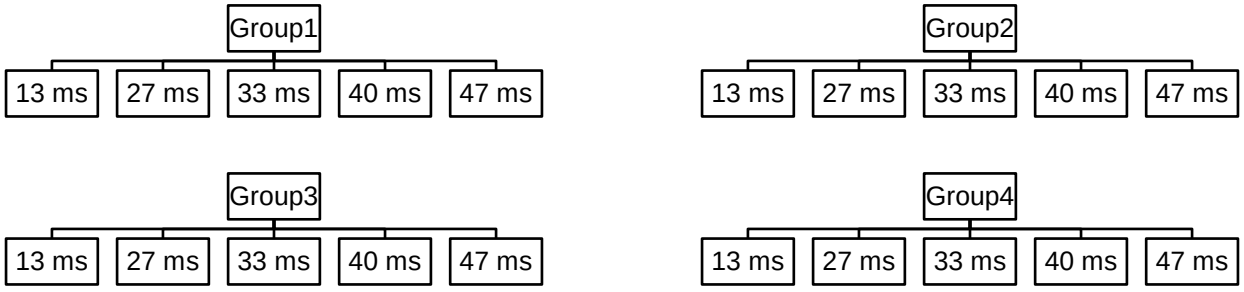

Figure 12. Conceptual layout of a single two-way mixed ANOVA. The between-subjects factor was group and the within-subjects factor was ISI. The layout for the confirmatory experiment was different as there were only two ISIs (13 and 47 ms). The dependent variable was the number of correct answers.

Peripheral backward masking experiment (target appeared in one of four quadrants on the screen and in one of four orientations).

Peripheral target location.

There was a non-significant effect of group (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only), of ISI (13, 27, 33, 40, or 47 ms), and of the group x ISI interaction on the mean number of correct answers to the location of the target when it appeared peripherally ($p > 0.05$).

For the between factor of group, Levene's tests were non-significant ($p > 0.05$), indicating that the variances were homogeneous across the five levels (13, 27, 33, 40, and 47 ms) of the within factor. For the within factor of ISI, Mauchly's test was non-significant ($p > 0.05$), indicating sphericity could be assumed. When the number of correct answers at each ISI was split by group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

Peripheral target orientation.

There was a non-significant effect of group (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only), of ISI (13, 27, 33, 40, or 47 ms), and of the group x ISI interaction on the mean number of correct answers to the orientation of the target when it appeared peripherally ($p > 0.05$).

For the between factor of group, Levene's tests were non-significant ($p > 0.05$), indicating that the variances were homogeneous across the five levels (13, 27, 33, 40, and 47 ms) of the within factor. For the within factor of ISI, Mauchly's test was non-significant ($p > 0.05$), indicating sphericity could be assumed. When the number of correct answers at each ISI was

split by group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

Peripheral confirmatory experiment (target appeared in one quadrant on the screen and in one of four orientations).

Peripheral target orientation.

There was a non-significant effect of group (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only), of ISI (13 or 47 ms), and of the group x ISI interaction on the mean number of correct answers to the orientation of the target when it appeared peripherally ($p > 0.05$).

For the between factor of group, Levene's tests were non-significant ($p > 0.05$), indicating that the variances were homogeneous across the two levels (13 and 47 ms) of the within factor. For the within factor of ISI, Mauchly's test for sphericity was not computed. In the confirmatory experiment, the within factor had only two levels. Two levels created only one variance and as a result, the homogeneity/heterogeneity of variance of the differences between the levels of the within factor was not a concern. When the number of correct answers at each ISI was split by group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

The results from the confirmatory experiment matched the results from the target orientation aspect of the peripheral experiment. Because both sets of results were non-significant, issues concerning dual tasking were not relevant.

Central backward masking experiment (target appeared in the center of the screen and in one of four orientations).

Central target orientation.

There was a non-significant effect of group (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only), of ISI (13, 27, 33, 40, or 47 ms), and of the group x ISI interaction on the mean number of correct answers to the orientation of the target when it appeared centrally ($p > 0.05$).

For the between factor of group, Levene's tests were non-significant ($p > 0.05$), indicating that the variances were homogeneous across the five levels (13, 27, 33, 40, and 47 ms) of the within factor. For the within factor of ISI, Mauchly's test was non-significant ($p > 0.05$), indicating sphericity could be assumed. When the number of correct answers at each ISI was split by group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

Summary.

Altogether, the results from the parametric two-way mixed ANOVAs could not show that the controls and the participants with dyslexia only were more likely to be masked at a different ISI than those with both dyslexia and ADHD and those with ADHD only. The non-significant interactions between group and ISI specifically indicated this. Thus, inferences could not be drawn about the existence of m-pathway impairments in the participants with both dyslexia and ADHD and the participants with ADHD only.

Such results, however, may not have been accurate given that a specific assumption of the parametric two-way mixed ANOVAs was violated. Each of the Shapiro-Wilk tests above indicated that there were some significant deviations from normality. While ANOVAs may be

robust to deviations from normality when sample sizes are equal across groups, they are not robust to these deviations when the sample sizes are unequal. In the research presented here, there were 13 controls, 4 participants with dyslexia, 6 participants with both dyslexia and ADHD, and 12 participants with ADHD only. Overall then, a non-parametric test should have been conducted rather than a parametric one. Unfortunately, a non-parametric version of the two-way mixed ANOVA does not exist (which was why in this section, the parametric-two way mixed ANOVA was applied). An alternative approach was taken and is described next.

4. Specific Analysis at each ISI: Group Differences in Masking across Experiments

The objective of the following analyses was, once again, to see if there were any differences between the four groups in the number of correct answers they achieved at each ISI. The number of correct answers referred to either: (1) the location of the target when it appeared peripherally, (2) the orientation of the target when it appeared peripherally, or (3) the orientation of the target when it appeared centrally. The non-parametric version of the one-way between ANOVA, the Kruskal-Wallis test, was used rather than the parametric two-way mixed ANOVA. Several Kruskal-Wallis tests were performed and each test was based on the number of correct answers of each group at each ISI in each experiment (this was akin to finding the interaction between group and ISI in the parametric two-way mixed ANOVA; see Figure 13). The Kruskal-Wallis test, however, was not based on the raw number of correct answers but was based on the ranks of these data.

In this test, the number of correct answers from each participant in each group was combined and ranked in a single series. The smallest number of correct answers was given a rank of 1, the next smallest number of correct answers was given a rank of 2, and this continued until all the numbers of correct answers were given ranks. Next, the sum of ranks in each group was

calculated and then compared. The Kruskal-Wallis test spoke to the question of whether differences in the sums of ranks were due to chance or due to the between factor of group (i.e., controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only; see Siegel, 1956). If these differences were due to the between factor of group, Mann-Whitney post-hoc tests were then conducted to see where the differences lay. The Mann-Whitney post-hoc tests were based on ranks of data as well.

In the following analyses, it was predicted that controls and participants with dyslexia only would score smaller correct answers at a particular ISI and that participants with both dyslexia and ADHD and participants with ADHD only would score smaller correct answers at a different ISI. This would indicate that the controls and participants with dyslexia only were more likely to be masked at a different time interval than those with both dyslexia and ADHD and those with ADHD only. In turn, this could suggest that there were m-pathway deficits in the participants with dyslexia and ADHD and the participants with ADHD only. The outcomes of the Kruskal-Wallis analyses were divided into subsections, with each subsection representing a specific experiment.

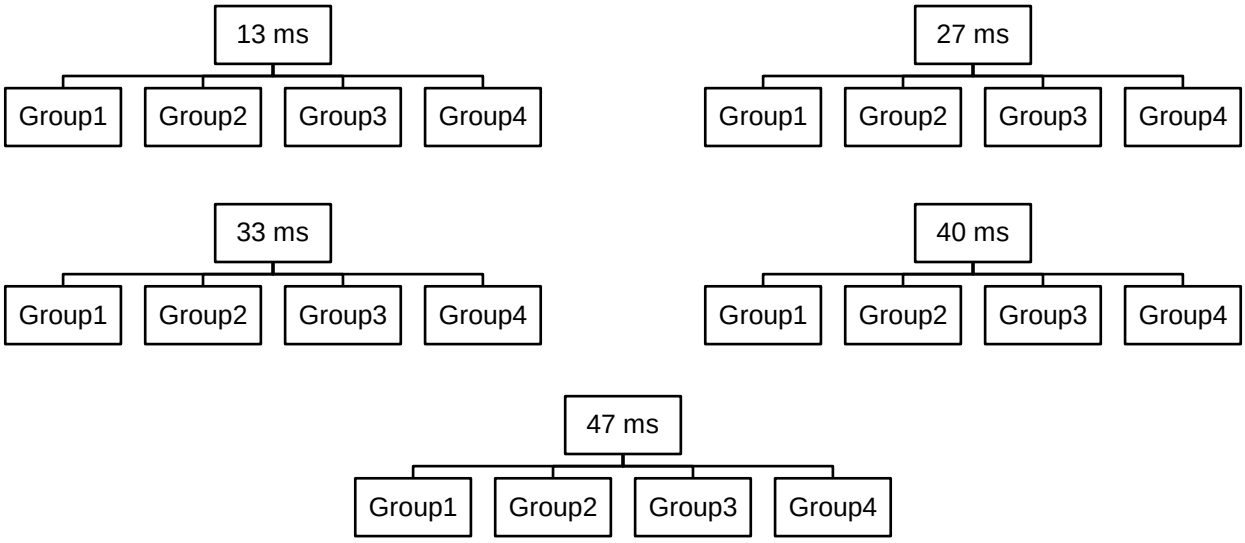

Figure 13. Conceptual layout of the Kruskal-Wallis tests. Each test was based on the number of correct answers of each group at each ISI in each experiment. Sums of ranks were calculated.

Peripheral backward masking experiment (target appeared in one of four quadrants on the screen and in one of four orientations).

Peripheral target location.

ISI of 13 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the location of the target when it appeared peripherally and the mask appeared 13 ms later, $H(3) = 0.97$, $p = 0.81$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

ISI of 27 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the location of the target when it appeared peripherally and the mask appeared 27 ms later, $H(3) = 4.64$, $p = 0.20$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 33 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the location of the target when it appeared peripherally and the mask appeared 33 ms later, $H(3) = 0.93$, $p = 0.82$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 40 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the location of the target when it appeared peripherally and the mask appeared 40 ms later, $H(3) = 3.12$, $p = 0.37$. Levene's test was non-significant ($p > 0.05$), indicating the variances

of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

ISI of 47 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the location of the target when it appeared peripherally and the mask appeared 47 ms later, $H(3) = 5.22$, $p = 0.16$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

Peripheral target orientation.

ISI of 13 ms.

There was a significant effect of group on the sums of ranks: There was a significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 13 ms later, $H(3) = 9.52$, $p = 0.02$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

As a follow-up to the Kruskal-Wallis test, Mann-Whitney post-hoc tests were performed to see where the differences lay. Because a Bonferroni correction was applied, the number of comparisons was kept to two. This led to a new critical value of 0.025 (0.05 regular critical value / 2 comparisons). It was decided that the most important comparisons to make would be: (1)

dyslexia only versus dyslexia co-morbid with ADHD and (2) dyslexia only versus ADHD only. These comparisons could show whether the participants with dyslexia only were different from those with attention deficits (i.e., those with both dyslexia and ADHD and those with ADHD only). As proposed in this paper, only the participants with attention deficits should have magnocellular problems. After all, individuals with schizophrenia have attention deficits and magnocellular deficits as well.

There was a non-significant difference between participants with dyslexia only and participants with both dyslexia and ADHD on the sums of ranks: There was no significant difference between these two groups in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 13 ms later, $U = 3.50$, $p = 0.07$. This result, however, did approach significance (i.e., $p = 0.07$ was not too different from $p = 0.025$). The participants with dyslexia only had the larger sum of ranks (30.50) compared to the participants with both dyslexia and ADHD, who had the smaller sum of ranks (24.50; see Figure 14). Accordingly, the participants with dyslexia only had the larger number of correct answers to the orientation of the target when it appeared peripherally and the mask appeared 13 ms later. Participants with both dyslexia and ADHD had the smaller number of correct answers. Going one step further, this suggested that the participants with dyslexia only were less likely to be masked at 13 ms than the participants with both dyslexia and ADHD, who were more likely to be masked at this ISI. The difference between the two groups was almost significant in this regard.

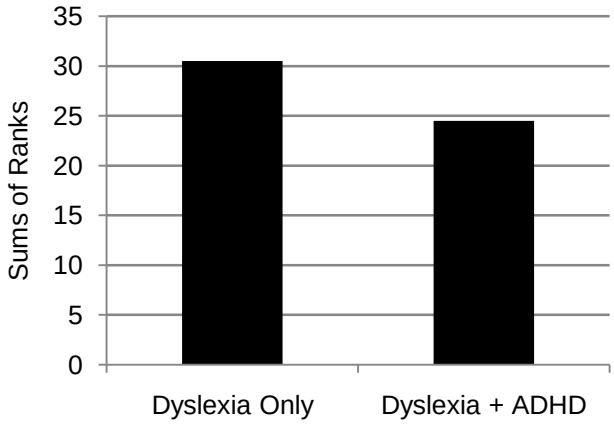

Figure 14. Target orientation aspect of the peripheral experiment at an ISI of 13 ms. The difference in the sums of ranks between dyslexia only and dyslexia co-morbid with ADHD was almost significant.

There was also a non-significant difference between participants with dyslexia only and participants with ADHD only on the sums of ranks: There was no significant difference between these two groups in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 13 ms later, $U = 23.50$, $p = 0.95$.

ISI of 27 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 27 ms later, $H(3) = 5.86$, $p = 0.12$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 33 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 33 ms later, $H(3) = 2.10$, $p = 0.55$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p \leq 0.05$).

ISI of 40 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 40 ms later, $H(3) = 1.25$, $p = 0.74$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 47 ms.

There was a significant effect of group on the sums of ranks: There was a significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 47 ms later, $H(3) = 9.39$, $p = 0.03$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the

groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

As a follow-up to the Kruskal-Wallis test, Mann-Whitney post-hoc tests were performed to see where the differences lay. The number of comparisons was kept to two and the new critical value was 0.025. The comparisons were: (1) dyslexia only versus dyslexia co-morbid with ADHD and (2) dyslexia only versus ADHD only. There was a non-significant difference between participants with dyslexia only and participants with both dyslexia and ADHD on the sums of ranks: There was no significant difference between these two groups in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 47 ms later, $U = 10.50$, $p = 0.76$.

There was also a non-significant difference between participants with dyslexia only and participants with ADHD only on the sums of ranks: There was no significant difference between these two groups in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 47 ms later, $U = 6.00$, $p = 0.03$. This result, however, did approach significance (i.e., $p = 0.03$ was not too different from $p = 0.025$)[4]. The participants with dyslexia only had the smaller sum of ranks (16.00) compared to the participants with ADHD only, who had the larger sum of ranks (120.00; see Figure 15). Accordingly, the participants with dyslexia only had the smaller number of correct answers to the orientation of the target when it appeared peripherally and the mask appeared 47 ms later. This was relative to participants with ADHD only. Going one step further, this suggested that the participants with dyslexia only were more likely to be masked at 47 ms than the participants with ADHD only, who were less likely to be masked at this ISI. The difference between the two groups was almost significant in this regard.

[4] Note, however, that $U = 6.00$, $p = 0.03$ would be significant at the alpha level of 0.05.

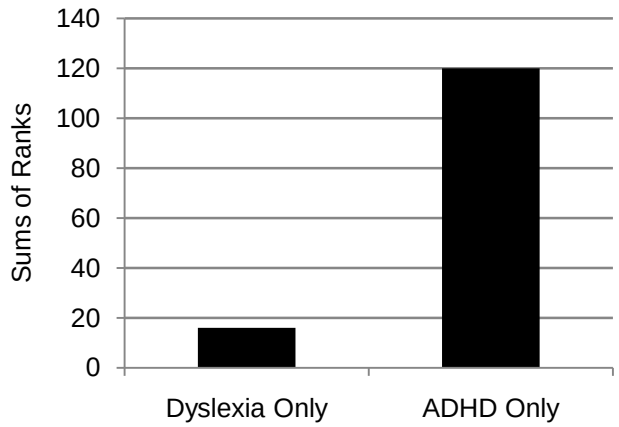

Figure 15. Target orientation aspect of the peripheral experiment at an ISI of 47 ms. The difference in the sums of ranks between dyslexia only and ADHD only was almost significant.

Peripheral confirmatory experiment (target appeared in one quadrant on the screen and in one of four orientations).

Peripheral target orientation.

ISI of 13 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 13 ms later, $H(3) = 2.54$, $p = 0.47$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

The Kruskal-Wallis result from this confirmatory experiment did not match the Kruskal-Wallis result from the target orientation aspect of the peripheral experiment at 13 ms (see above). The Kruskal-Wallis result from the confirmatory experiment revealed a non-significant effect of

group on the sums of ranks at 13 ms while the Kruskal-Wallis result from the target orientation aspect of the peripheral experiment revealed a significant effect. This discrepancy implied that two accounts could be used to explain outcomes (i.e., dual tasking difficulties vs. the presence or absence of magnocellular deficits). This is elaborated on in the discussion section.

ISI of 47 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and the mask appeared 47 ms later, $H(3) = 0.92$, $p = 0.82$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

The Kruskal-Wallis result from this confirmatory experiment did not match the Kruskal-Wallis result from the target orientation aspect of the peripheral experiment at 47 ms (see above). The Kruskal-Wallis result from the confirmatory experiment revealed a non-significant effect of group on the sums of ranks at 47 ms while the Kruskal Wallis result from the target orientation aspect of the peripheral experiment revealed a significant effect. This discrepancy implied that two accounts could be used to explain outcomes (i.e., dual tasking difficulties vs. the presence or absence of magnocellular deficits). This is elaborated on in the discussion section.

Central backward masking experiment (target appeared in the center of the screen and in one of four orientations).

Central target orientation.

ISI of 13 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared centrally and the mask appeared 13 ms later, $H(3) = 3.28$, $p = 0.35$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

ISI of 27 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared centrally and the mask appeared 27 ms later, $H(3) = 3.69$, $p = 0.30$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 33 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they

made to the orientation of the target when it appeared centrally and the mask appeared 33 ms later, $H(3) = 2.18$, $p = 0.54$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

ISI of 40 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared centrally and the mask appeared 40 ms later, $H(3) = 0.43$, $p = 0.93$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

ISI of 47 ms.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared centrally and the mask appeared 47 ms later, $H(3) = 1.26$, $p = 0.74$. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

Summary.

Altogether, the results from the Kruskal-Wallis tests for the target location aspect of the peripheral experiment, the confirmatory experiment, and the central experiment were non-significant. In contrast, some of the results from the Kruskal-Wallis tests for the target orientation aspect of the peripheral experiment were significant. This suggested differences somewhere between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the number of correct answers they made to the orientation of the target when it appeared peripherally and at ISIs of 13 and 47 ms. Some evidence was provided for particular differences between: (1) dyslexia only and dyslexia co-morbid with ADHD at 13 ms and (2) dyslexia only and ADHD only at 47 ms. These results from the Mann-Whitney tests were almost significant.

The participants with dyslexia only were more likely to be masked at 47 ms and less likely to be masked at 13 ms. On the other hand, the participants with ADHD only were less likely to be masked at 47 ms and the participants with both dyslexia and ADHD were more likely to be masked at 13 ms. Here, an opposite pattern of masking was demonstrated: a group without attention deficits (dyslexia only) was more likely to be masked at a high ISI while the groups with attention deficits (dyslexia and ADHD, ADHD only) were more likely to be masked at a low ISI. This pattern was in line with the predictions made in the present study. It was predicted that controls and participants with dyslexia only would more likely be masked at a different time interval than those with both dyslexia and ADHD and those with ADHD only.

To examine this further, the next set of analyses involved comparing the ISIs at which the participants were best masked. Best masking entailed the one specific ISI out of the five ISIs (e.g., 13, 27, 33, 40, or 47 ms) that participants scored the least number of correct answers on.

5. Group Differences in Best Masking across Experiments

The objective of the following analyses was to see if there were any differences between the four groups of participants in the ISI at which they were best masked or, in other terms, the ISI at which they scored the least number of correct answers. The least number of correct answers referred to either: (1) the location of the target when it appeared peripherally, (2) the orientation of the target when it appeared peripherally, or (3) the orientation of the target when it appeared centrally. Based on the last set of analyses, it was predicted that controls and participants with dyslexia only would be best masked at a high ISI and that participants with both dyslexia and ADHD and participants with ADHD only would be best masked at a low ISI. If this pattern were found, it would indicate that the transient systems, or the m-pathways, of the participants with both dyslexia and ADHD and the participants with ADHD only were problematic in some way. The m-pathway problems would be confined to these two groups because of the presence of attention deficits.

Framed in terms of the two research purposes, finding this pattern of results would suggest: (1) m-pathway impairments were not present in the individuals with dyslexia only but rather, were present in the individuals with both dyslexia and ADHD and (2) similar to the m-pathway impairments in the individuals with both dyslexia and ADHD, there were impairments in the individuals with ADHD only. On a broader scale, this would then show that the visual magnocellular model could account for some cases of dyslexia and that the phonological model could not account for all cases of dyslexia.

The following analyses involved Kruskal-Wallis tests. The between-subjects factor was group (i.e., controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only) and the dependent factor was the ISI at which best masking

occurred (i.e., **best mask ISI**). This was done for each experiment. Kruskal-Wallis tests were performed over parametric one-way between ANOVAs because of the large standard deviations that surrounded the mean ISI at which the least number of correct answers was scored[5].

In each Kruskal-Wallis test, the best mask ISI of each participant in each group was combined and ranked in a single series. The smallest best mask ISI was given a rank of 1, the next smallest best mask ISI was given a rank of 2, and this continued until all best mask ISIs were given ranks. Next, the sum of ranks in each group was calculated and then compared. The Kruskal-Wallis test spoke to the question of whether differences in best mask ISI were due to chance or due to the between factor of group. If these differences were due to group, Mann-Whitney post-hoc tests were conducted to see where the differences lay. The Mann-Whitney post-hoc tests were based on ranks of data as well. The outcomes of the Kruskal-Wallis analyses were divided into subsections with each subsection representing a specific experiment (see below).

Peripheral backward masking experiment (target appeared in one of four quadrants on the screen and in one of four orientations).

Peripheral target location.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they scored the least number of correct answers to the location of the target when it appeared in the periphery, $H(3) = 6.54$, $p = 0.09$. In other words, there was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD

[5] There was homogeneity of variance though. However, this may be a misleading result due to the low and unequal sample sizes across the four groups of participants.

only in the ISI at which they were best masked in this target location aspect of the peripheral experiment. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

Peripheral target orientation.

There was a significant effect of group on the sums of ranks: There was a significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they scored the least number of correct answers to the orientation of the target when it appeared in the periphery, $H(3) = 8.35$, $p = 0.04$. In other words, there was a significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they were best masked in this target orientation aspect of the peripheral experiment. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p < 0.05$).

As a follow-up to the Kruskal-Wallis test, Mann-Whitney post-hoc tests were performed to see where the differences lay. Because a Bonferroni correction was applied, the number of comparisons was kept to two. This led to a new critical value of 0.025 (0.05 regular critical value / 2 comparisons). As in previous analyses, the comparisons were between: (1) dyslexia only and dyslexia co-morbid with ADHD and (2) dyslexia only and ADHD only. These comparisons were chosen because they could show whether the participants with dyslexia only were different from those with attention deficits (i.e., those with both dyslexia and ADHD and those with ADHD

only). As proposed in this paper, only the participants with attention deficits should have m-pathway impairments.

There was a significant difference between participants with dyslexia only and participants with both dyslexia and ADHD on the sums of ranks: There was a significant difference between these two groups in the ISI at which they scored the least number of correct answers to the orientation of the target when it appeared in the periphery, $U = 2.50$, $p = 0.02$. In other words, there was a significant difference between the participants with dyslexia only and the participants with both dyslexia and ADHD in the ISI at which they were best masked in this target orientation aspect of the peripheral experiment. The participants with dyslexia only had the larger sum of ranks (31.50) compared to the participants with both dyslexia and ADHD, who had the smaller sum of ranks (23.50; see Figure 16). Accordingly, the participants with dyslexia only were more likely to score the least number of correct answers (i.e., were best masked) at higher ISIs. The participants with both dyslexia and ADHD were more likely to score the least number of correct answers (i.e., were best masked) at lower ISIs.

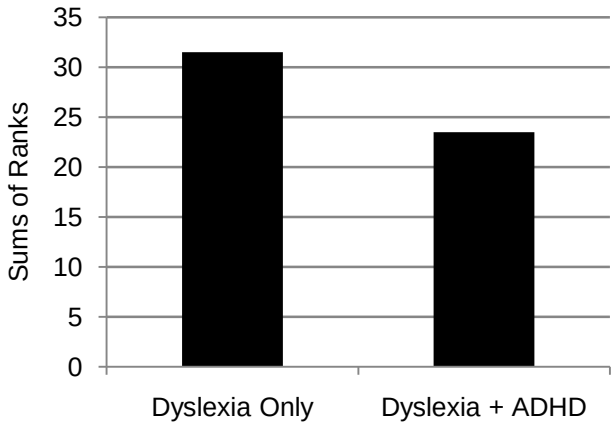

Figure 16. Target orientation aspect of the peripheral experiment. The difference in the sums of ranks between dyslexia only and dyslexia co-morbid with ADHD was significant.

There was a significant difference between participants with dyslexia only and participants with ADHD only on the sums of ranks: There was a significant difference between these two groups in the ISI at which they scored the least number of correct answers to the orientation of the target when it appeared in the periphery, $U = 2.50$, $p = 0.00$. In other words, there was a significant difference between the participants with dyslexia only and the participants with ADHD only in the ISI at which they were best masked in this target orientation aspect of the peripheral experiment. The participants with dyslexia only had the smaller sum of ranks (55.50)[6] compared to the participants with ADHD only, who had the larger sum of ranks (80.50; see Figure 17). Accordingly, the participants with dyslexia only were more likely to score the least number of correct answers (i.e., were best masked) at lower ISIs. The participants with ADHD only were more likely to score the least number of correct answers (i.e., were best masked) at higher ISIs.

[6] Readers may notice that the sum of ranks for dyslexia only was 31.50 when compared to dyslexia co-morbid with ADHD and that the sum of ranks for dyslexia only was later 55.50 when compared to ADHD only. In the Mann-Whitney tests used here, two groups were compared at a time and only the raw scores of these two groups were combined in a single series and then ranked from smallest to largest. Thus, the combined raw scores in the first comparison (dyslexia only vs. dyslexia co-morbid with ADHD) were different from the combined raw scores in the second comparison (dyslexia only vs. ADHD only). The different combinations of raw scores led to different rankings. This is why the same group, dyslexia only, had two different sums of ranks: the sums of ranks changed based on the comparison made.

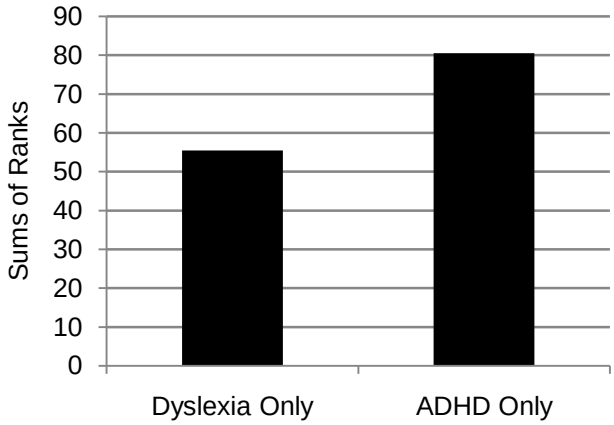

Figure 17. Target orientation aspect of the peripheral experiment. The difference in the sums of ranks between dyslexia only and ADHD only was significant.

Peripheral confirmatory experiment (target appeared in one quadrant on the screen and in one of four orientations).

Peripheral target orientation.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they scored the least number of correct answers to the orientation of the target when it appeared in the periphery, $H(3) = 3.29$, $p = 0.35$. In other words, there was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they were best masked in this confirmatory experiment. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were some significant deviations from normality ($p > 0.05$).

The Kruskal-Wallis result from this confirmatory experiment did not match the Kruskal-Wallis result from the target orientation aspect of the peripheral experiment. The Kruskal-Wallis result from the confirmatory experiment revealed a non-significant effect of group on the sums of ranks while the Kruskal-Wallis result from the target orientation aspect of the peripheral experiment revealed a significant effect. This discrepancy implied that two accounts could be used to explain outcomes (i.e., dual tasking difficulties vs. the presence or absence of magnocellular deficits). This is elaborated on in the discussion section.

Central backward masking experiment (target appeared in the center of the screen and in one of four orientations).

Central target orientation.

There was a non-significant effect of group on the sums of ranks: There was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they scored the least number of correct answers to the orientation of the target when it appeared in the center, $H(3) = 5.00$, $p = 0.17$. In other words, there was no significant difference between controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only in the ISI at which they were best masked in this central experiment. Levene's test was non-significant ($p > 0.05$), indicating the variances of the groups were homogeneous. When looking at each group, Shapiro-Wilk tests indicated there were no significant deviations from normality ($p > 0.05$).

Summary.

Altogether, the results from the Kruskal-Wallis tests and Mann-Whitney post-hoc tests could not definitively show that the controls and participants with dyslexia only were best masked at a high ISI while the participants with both dyslexia and ADHD and participants with ADHD only were best masked at a low ISI. The results from the Krukal-Wallis tests for the target location aspect of the peripheral experiment, the confirmatory experiment, and the central experiment were non-significant. However, the result from the Kruskal-Wallis test for the target orientation aspect of the peripheral experiment was significant. When dyslexia was compared to dyslexia co-morbid with ADHD, it was found that the participants with dyslexia only were best masked at a high ISI while the participants with both dyslexia and ADHD were best masked at a low ISI. In contrast, when dyslexia only was compared against ADHD only, the participants with dyslexia only were best masked at a low ISI while the participants with ADHD only were best masked at a high ISI. Overall then, no consistent pattern was found in which dyslexia only was best masked at a high ISI and the groups with attention deficits were best masked at a low ISI.

The Kruskal-Wallis tests conducted on best mask ISI may not have been explicit enough to determine whether controls and participants with dyslexia only were best masked at a high ISI while participants with both dyslexia and ADHD and participants with ADHD only were best masked at a low ISI. For example, it was not clear what constituted a high or low ISI. A more explicit approach was taken and is described next.

6. Group Differences in Best Masking at Specific High ISIs across Experiments

The objective of the following analyses was to see how many participants in each of the four groups were best masked at the high ISIs of 40 and 47 ms, or in other terms, scored the least number of correct answers at 40 and 47 ms. The least number of correct answers referred to either: (1) the location of the target when it appeared peripherally, (2) the orientation of the target when it appeared peripherally, or (3) the orientation of the target when it appeared centrally. The decision to examine best masking at these specific ISIs was based on what had been found so far. In section 4 of the results, it was found that: (1) participants with dyslexia only were more likely to be masked at 47 ms and less likely to be masked at 13 ms, (2) participants with ADHD only were less likely to be masked at 47 ms, and (3) participants with both dyslexia and ADHD were more likely to be masked at 13 ms. In section 5, it was found that some of the groups differed in masking at high versus low ISIs. For example, the participants with dyslexia only were best masked at high ISIs while the participants with both dyslexia and ADHD were best masked at low ISIs (although, this pattern was inconsistent in light of the other comparisons that were also made).

Altogether, counting the number of participants in each group who were best masked at the explicit ISIs of 40 and 47 ms could reveal whether controls and participants with dyslexia only were best masked at an ISI that was different from when participants with both dyslexia and ADHD and participants with ADHD only were best masked. It was predicted that more controls and participants with dyslexia only would be best masked at 40 and 47 ms and that less participants with both dyslexia and ADHD and participants with ADHD only would be best masked at these ISIs (suggesting that more participants with both dyslexia and ADHD and participants with ADHD only would be best masked at other, lower ISIs). If this pattern were

found, there would be evidence for m-pathway deficits in the participants with attention deficits. After all, individuals with schizophrenia have attention deficits and magnocellular deficits as well.

The following graphs were created to depict the percentages of controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only who were best masked at 40 to 47 ms. Here, the y-axes represented a range of ISIs (e.g., 40 to 47 ms) rather than discrete categories of ISIs (40 and 47 ms). This was done as some participants scored the least number of correct answers at both 40 and 47 ms, resulting in a best mask ISI of 43.50 ms (40 + 47 / 2).

The following analyses involved contingency chi-square tests. The chi-square tests were performed on the percentages rather than on the raw number of participants in each group who were best masked at high ISIs. This was done because some of the groups had low sample sizes, which led to expected frequencies that were less than 5, creating problems for the chi-square test (e.g., loss of statistical power). Below, results were divided into subsections, with each subsection representing a particular experiment.

Peripheral backward masking experiment (target appeared in one of four quadrants on the screen and in one of four orientations).

Peripheral target location.

As illustrated in Figure 18, when the target appeared in one of four locations in the periphery of the screen, a larger percentage of participants with both dyslexia and ADHD were best masked at high ISIs, which ranged from 40 to 47 ms. In contrast, smaller percentages of controls, participants with dyslexia only, and participants with ADHD only were best masked at

these high ISIs. Thus, more controls, participants with dyslexia only, and participants with ADHD only were best masked at other, lower ISIs, which ranged from 13 to 39 ms[7].

A contingency chi-square revealed a significant association between group and the percentage of participants in each group who were best masked at ISIs of 40 to 47 ms, $X^2(3) = 82.26$, $p = 0.00$. By comparing observed and expected frequencies, it was further revealed that: (1) 16.67% of participants with ADHD only were best masked at high ISIs, implying that most of them were best masked at low ISIs, (2) 66.67% of participants with both dyslexia and ADHD were best masked at high ISIs, so the majority of them were best masked at these ISIs, (3) 15.38% of controls were best masked at high ISIs, implying that most of them were best masked at low ISIs, and (4) 25.00% of participants with dyslexia only were best masked at high ISIs, implying that most of them were best masked at low ISIs.

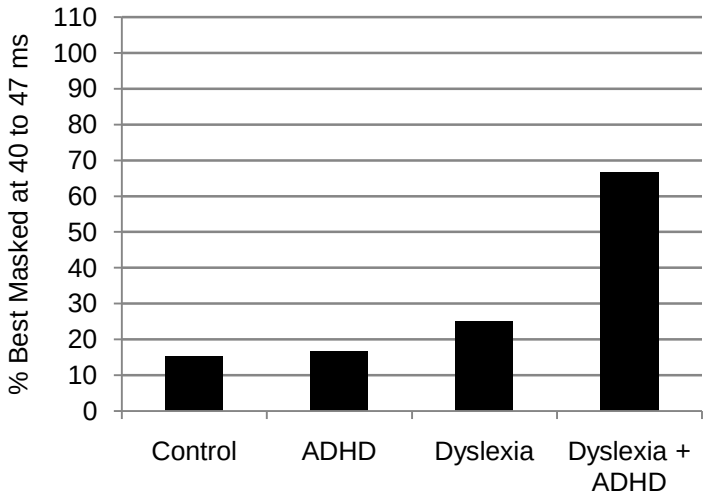

Figure 18. Target location aspect of the peripheral experiment. Percentages of participants from each group who were best masked at the ISIs of 40 to 47 ms.

[7] Some participants were best masked at two or more ISIs. The mean of the ISIs was taken and often resulted in a number in-between the discrete categories of the ISIs tested. For example, if a participant scored the least number of correct answers at both 13 and 27 ms, he or she was best masked at the ISI of 20 ms (13 + 27 / 2). Hence, the range of 13 to 39 ms.

Peripheral target orientation.

As illustrated in Figure 19, when the target appeared in one of four orientations in the periphery of the screen, larger percentages of controls and participants with dyslexia only were best masked at high ISIs, which ranged from 40 to 47 ms. In contrast, smaller percentages of participants with both dyslexia and ADHD and participants with ADHD only were best masked at these high ISIs. Thus, more participants with both dyslexia and ADHD and participants with ADHD only were best masked at other, lower ISIs, which ranged from 13 to 39 ms.

A contingency chi-square revealed a significant association between group and the percentage of participants in each group who were best masked at ISIs of 40 to 47 ms, $X^2(3) = 147.69$, $p = 0.00$. By comparing observed and expected frequencies, it was further revealed that: (1) 16.67% of participants with both dyslexia and ADHD were best masked at high ISIs, implying that most of them were best masked at low ISIs, and (2) 100.00% of participants with dyslexia only were best masked at high ISIs. In terms of participants with ADHD only and controls, notable differences between observed and expected frequencies were not found.

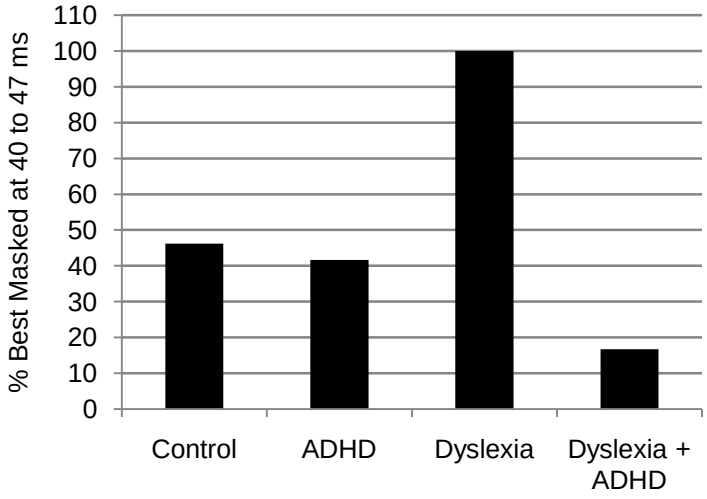

Figure 19. Target orientation aspect of the peripheral experiment. Percentages of participants from each group who were best masked at the ISIs of 40 to 47 ms.

Peripheral confirmatory experiment (target appeared in one quadrant on the screen and in one of four orientations).

Peripheral target orientation.

As mentioned earlier, the confirmatory experiment had only two ISIs, 13 and 47 ms. Thus, the results presented in this section entail the percentages of participants who were best masked at the exact ISI of 47 ms and not at the ISIs of 40 to 47 ms. As illustrated in Figure 20, when the target appeared in one location and in one of four orientations in the periphery of the screen, a larger percentage of participants with ADHD only were best masked at 47 ms. In contrast, smaller percentages of controls, participants with dyslexia only, and participants with both dyslexia and ADHD were best masked at this highest ISI. Thus, more controls, participants with dyslexia only, and participants with both dyslexia and ADHD were best masked at other, lower ISIs, which ranged from 13 to 30 ms (30 is the mean of 13 and 47, for those participants who were best masked at both ISIs).

A contingency chi-square revealed a significant association between group and the percentage of participants in each group who were best masked at the highest ISI of 47 ms, $X^2(3) = 41.34$, $p = 0.00$. By comparing observed and expected frequencies, it was further revealed that: (1) 75.00% of participants with ADHD only were best masked at the highest ISI, so the majority of them were best masked at this ISI, (2) 33.33% of participants with both dyslexia and ADHD were best masked at the highest ISI, implying that most of them were best masked at lower ISIs, and (3) 38.46% of controls were best masked at the highest ISI, implying that most of them were best masked at lower ISIs. In terms of participants with dyslexia only, notable differences between observed and expected frequencies were not found.

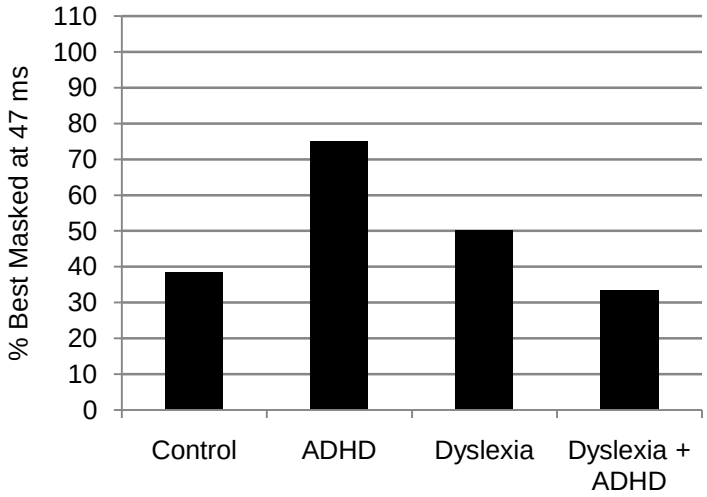

Figure 20. Confirmatory experiment. Percentages of participants from each group who were best masked at the highest ISI of 47 ms.

To determine whether the results of the confirmatory experiment matched those of the target orientation aspect of the peripheral experiment, the two needed to be equated so that they could be compared. Thus, in the target orientation aspect of the peripheral experiment, the percentage of participants in each group who were best masked at the exact ISI of 47 ms (instead of 40 to 47 ms) was calculated. As illustrated in Figure 21, it can be seen that larger percentages of controls and participants with dyslexia only were best masked at 47 ms compared to those with both dyslexia and ADHD and those with ADHD only.

A contingency chi-square revealed a significant association between group and the percentage of participants in each group who were best masked at the highest ISI of 47 ms, $X^2(3) = 127.59$, $p = 0.00$. Overall, the results from the confirmatory experiment did not match those of the target orientation aspect of the peripheral experiment. The pattern of masking across groups at 47 ms was different. This discrepancy implied that two accounts could be used to explain

outcomes (i.e., dual tasking difficulties vs. the presence or absence of magnocellular deficits). This is elaborated on in the discussion section.

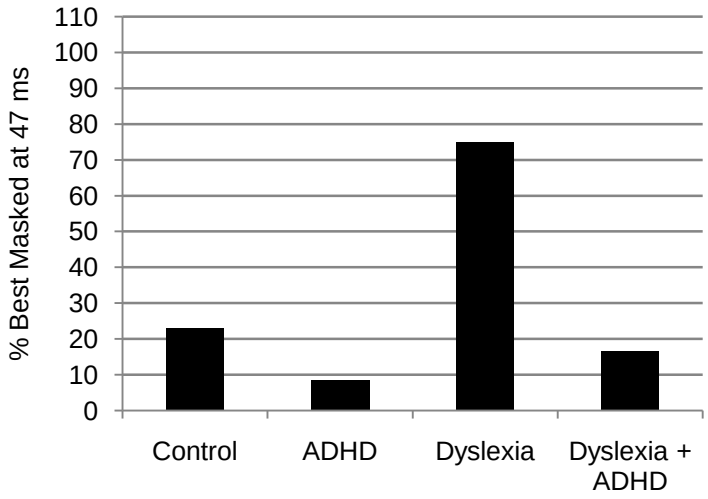

Figure 21. Target orientation aspect of the peripheral experiment. Percentages of participants from each group who were best masked at the highest ISI of 47 ms.

Central backward masking experiment (target appeared in the center of the screen and in one of four orientations).

Central target orientation.

As illustrated in Figure 22, when the target appeared in one of four orientations and in the center of the screen, larger percentages of controls and participants with dyslexia only were best masked at high ISIs, which ranged from 40 to 47 ms. In contrast, smaller percentages of participants with both dyslexia and ADHD and participants with ADHD only were best masked at these high ISIs. Thus, more participants with both dyslexia and ADHD and participants with ADHD only were best masked at other, lower ISIs, which ranged from 13 to 39 ms.

A contingency chi-square revealed a significant association between group and the percentage of participants in each group who were best masked at ISIs of 40 to 47 ms, $X^2(3) =$

45.40, $p = 0.00$. By comparing observed and expected frequencies, it was further revealed that: (1) 16.67% of participants with ADHD only were best masked at high ISIs, implying that most of them were best masked at lower ISIs, and (2) 16.67% of participants with both dyslexia and ADHD were best masked at high ISIs, again implying that most of them were best masked at lower ISIs. In terms of controls and participants with dyslexia only, notable differences between observed and expected frequencies were not found.

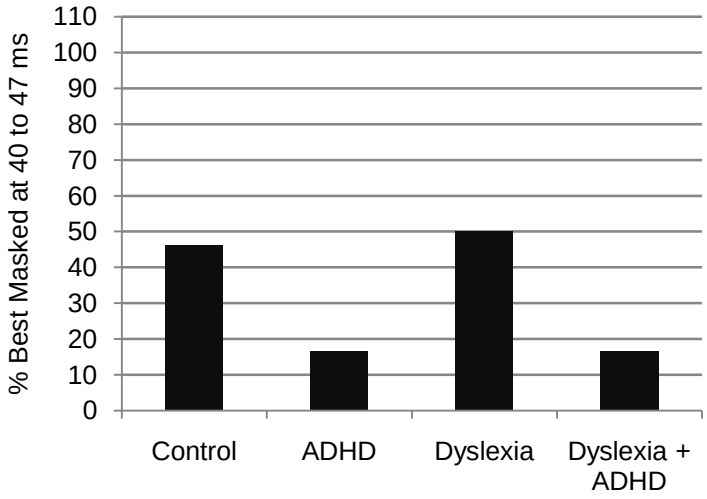

Figure 22. Central experiment. Percentages of participants from each group who were best masked at the ISIs of 40 to 47 ms.

Summary.

Altogether, there was some evidence that the participants without attention deficits (e.g., participants with dyslexia only) were best masked at high ISIs of 40 to 47 ms while participants with attention deficits (e.g., participants with both dyslexia and ADHD, participants with ADHD only) were best masked at other, lower ISIs (13 to 39 ms). This evidence stemmed from the target orientation aspect of the peripheral experiment and the central experiment. In the target orientation aspect of the peripheral experiment, a larger percentage of participants with both

dyslexia and ADHD were best masked at low ISIs of 13 to 39 ms whereas a larger percentage of participants with dyslexia only were best masked at high ISIs of 40 to 47 ms. In the central experiment, larger percentages of the participants with both dyslexia and ADHD and the participants with ADHD only were best masked at low ISIs of 13 to 39 ms. All of these results were in line with the predictions made. However, results from the target location aspect of the peripheral experiment and from the confirmatory experiment showed a different pattern of best masking across the four groups.

Discussion

Evidence for a Visual Magnocellular Deficit in the Participants with Attention Deficits

There were two purposes in this study: (1) to see if magnocellular deficits occur in dyslexia that is co-morbid with ADHD or in dyslexia only and (2) if magnocellular deficits occur in dyslexia that is co-morbid with ADHD, to see if similar deficits occur in ADHD only. Overall, there was some evidence for m-pathway deficits occurring in the participants with both dyslexia and ADHD and not in the participants with dyslexia only. There was also some evidence for similar deficits occurring in the participants with ADHD only. The results are in line with the theoretical account advocated in this paper and these specific results are summarized below. It is helpful to have this summary as many analyses were conducted.

Based on the research purposes, two predictions were made. First, controls and participants with dyslexia only should perform similarly to each other whereas participants with both dyslexia and ADHD and participants with ADHD only should perform alike. Second, controls and participants with dyslexia only should perform differently from the participants with both dyslexia and ADHD and the participants with ADHD only. Presumably, controls do not have any magnocellular problems. Thus, if controls and participants with dyslexia only perform

similarly to each other, this would suggest that the participants with dyslexia only do not have any magnocellular problems. If participants with both dyslexia and ADHD and participants with ADHD only perform alike, this would suggest that they have magnocellular problems. Research has indicated that individuals with schizophrenia have attention deficits and magnocellular impairments as well.

Specific results support these predictions. In terms of the target orientation aspect of the peripheral experiment, it was modestly found that participants with dyslexia only were more likely to be masked at the highest ISI of 47 ms and less likely to be masked at the lowest ISI of 13 ms. The two groups with attention deficits demonstrated the opposite pattern, but performed in line with one another. The participants with ADHD only were less likely to be masked at 47 ms and the participants with both dyslexia and ADHD were more likely to be masked at 13 ms.

Within the same aspect of the peripheral experiment, all the participants with dyslexia only were best masked at the high ISIs of 40 to 47 ms. Few participants with ADHD only were best masked at these high ISIs, implying that the majority of them were best masked at lower ISIs. Similarly, in the central experiment, few participants with attention deficits (e.g., dyslexia co-morbid with ADHD, ADHD only) were best masked at these high ISIs, again implying that they were best masked at lower ISIs. Altogether, it can be seen that the participants with dyslexia only performed differently from the participants with attention deficits while the two groups of participants with attention deficits performed alike.

Another result demonstrating support for the general predictions made in this study comes from the target orientation aspect of the peripheral experiment. The participants with dyslexia only were best masked at a higher ISI when compared to participants with both dyslexia and ADHD, who were best masked at a lower ISI. Here, best masking specifically referred to the

least number of correct answers made to the orientation of the target when it appeared in the periphery of the screen. It can be seen here that once again, the participants with dyslexia only performed differently from the participants with both dyslexia and ADHD. Admittedly, however, this finding was attenuated by the comparison showing that participants with dyslexia only were best masked at a lower ISI and participants with ADHD only were best masked at a higher ISI.

Importance of this Evidence for Conceptualizations of Dyslexia

Collectively, the findings above show that the participants with attention deficits (dyslexia co-morbid with ADHD, ADHD only) were masked of the target at a different time interval than the participants without attention deficits (i.e., dyslexia only). This suggests that the participants with both dyslexia and ADHD and the participants with ADHD only have magnocellular deficits while those with dyslexia only do not. Inferences about magnocellular deficits are possible given that the backward masking experiments were designed to stimulate the m-pathway over the other pathways of the visual system (e.g., low contrast, flickering stimuli). Overall, it may be the ADHD component that is specifically related to m-pathway impairments. This connection is supported by studies indicating that individuals with schizophrenia have: (1) attention deficits and (2) magnocellular impairments.

Researchers such as Kairalla et al. (2008) have found that individuals with schizophrenia have specific sustained and selective attention deficits. They assessed sustained attention using a computer task in which the participants had to view a black screen for a period of time and then press a button on a joystick when a red circle appeared on the screen. To assess selective attention, another computer task was used where participants had to press a button when a particular geometric shape appeared on the screen. Interestingly, these researchers showed that individuals with schizophrenia have an attention deficit that other researchers have found in

individuals with ADHD. For example, Marchetta et al. (2008) found that individuals with ADHD have sustained attention problems too. In this case, sustained attention was evaluated using a computer task where three, four, or five dots continuously appeared on the screen. Participants had to press a button when four dots appeared. This similarity between schizophrenia and ADHD in attention deficits is important for inferences of magnocellular impairments in individuals with both dyslexia and ADHD and individuals with ADHD only.

Several studies have indicated that individuals with schizophrenia have transient subsystem or magnocellular impairments. For instance, Rund et al. (1996) compared adolescents with schizophrenia, adolescents with ADHD, and controls on a backward masking task. They found that the participants with schizophrenia and the participants with ADHD had backward masking deficits that were similar to each other and different from the controls. The backward masking deficits indicated the presence of magnocellular impairment. Schechter et al. (2005) also found this impairment using transient visual evoked potentials (tVEP). When compared to controls, they found that the participants with schizophrenia/schizoaffective disorder had decreased P1 responses to stimuli that were biased towards the m-pathway. Because P1 reflects early activation of the visual cortex through the m-pathway, decreased P1 responses indicated a specific magnocellular deficit.

In another study, Schwartz et al. (1999) made a similar conclusion about individuals with schizophrenia having a magnocellular deficit, except that they used a motion defined letter task. In this task, dots that comprise a letter move to the right while dots that comprise the background move to the left. Participants needed to identify the letter. As it turned out, participants with schizophrenia reported fewer correct letters when compared to controls. Because the letters were

motion-related and the m-pathway is stimulated by objects that move, it was inferred that the individuals with schizophrenia, by reporting fewer correct letters, had m-pathway deficits.

Altogether, these studies show that individuals with schizophrenia have attention deficits that are similar to those found in individuals with ADHD and that they also have magnocellular impairments. More importantly, these studies on schizophrenia support the notion that in the present study, the participants with both dyslexia and ADHD and the participants with ADHD only were the ones with magnocellular deficits and not the participants with dyslexia only. Interestingly, research suggests an anatomical link between visual attention and the m-pathway: to attend to a specific stimulus, attention needs to be allocated (Omtzigt & Hendriks, 2004; Steinman, Stein, & Lehmkuhle, 1997). The parietal cortex plays a role in attention allocation. The parietal cortex is also part of the dorsal stream, which receives much of its input from the m-pathway. It seems then, that attention allocation is to some degree mediated by input from the m-pathway (Omtzigt & Hendriks, 2004). Accordingly, attention deficits may affect the m-pathway and vice versa.

Overall, the findings of this study can explain why some research has found magnocellular impairments in individuals with dyslexia (Eden et al., 1996; Livingstone et al., 1991; Martin & Lovegrove, 1987; Talcott et al., 1998; Wang et al., 2010) and why some research has not (Amitay et al., 2002; Johannes et al., 1996; Skoyles & Skottun, 2004). It depends on whether or not the dyslexia that is examined is co-morbid with ADHD. The present study is one of the first to make this distinction explicit. Previous studies have predominantly mentioned the effects of co-morbidity, but have not examined it in-depth.

This study provides some evidence that the visual magnocellular model may be used to explain cases of dyslexia that are co-morbid with ADHD while other models (e.g., the

phonological model) may be used to explain cases of dyslexia that are not co-morbid with ADHD. In addition, this study provides some evidence that the magnocellular model may be used to explain any reading deficits in individuals with ADHD only. One theory on how a magnocellular deficit can disrupt reading is through binocular instability (Stein, 2001; Stein & Walsh, 1997)[8]. When the m-pathway is unimpaired, it plays a role in the control and stabilization of the left and right eyes so that only a single image of text is perceived. However, if individuals with attention deficits are masked at a different time interval than those without attention deficits (as has been found here), then they may have an m-pathway that is impaired and ineffective at stabilizing the two eyes. These individuals may then experience visual confusion and perceive letters on a page as overlapping or moving around. It should be noted that while the connection between backward masking deficits, binocular instability, and reading problems is plausible, it was not directly tested in this study.

Evidence from Confirmatory Experiment may Limit this Conceptualization of Dyslexia

Because the peripheral experiment required participants to name both the orientation and location of the target on the screen, it can be argued that this experiment required participants to dual task. There may be differences, however, in the four groups of participants and their abilities to dual task effectively. For instance, the participants with attention deficits, such as those with both dyslexia and ADHD and those with ADHD only, may find it more difficult to dual task than the participants without attention deficits (i.e., controls, participants with dyslexia only).

Pasini et al. (2007) found that boys with ADHD were slower at completing a divided attention test than boys without ADHD. Divided attention is the ability to attend to more than

[8] The binocular instability theory is discussed here rather than the p-pathway suppression theory as the latter theory has been debunked by research showing that the m-pathway is suppressed at each saccade (e.g., Burr et al., 1994).

one stimulus at the same time. Because divided attention is another way of viewing dual tasking, Pasini et al. (2007) essentially found that boys with ADHD were slower at dual tasking than boys without ADHD. This suggests that the boys with ADHD had problems with dual tasking when compared to the boys without the disorder. Pasini et al. (2007) used the Trail Making Test B (TMT B), in which there are numbers from 1 to 13 and letters from A to L scattered within circles on a piece of paper. Participants were instructed to connect the numbers and letters in order, alternating between numbers and letters. Performance was measured in seconds to complete the sequence. In terms of the adult literature, Boonstra et al. (2005) found via a meta-analysis that adults with ADHD performed worst on the TMT B than controls. As before, this suggests that adults with ADHD have problems with dual tasking when compared to controls.

Overall, and within the context of the present study, group differences in masking can be attributed to two accounts. First, differences may be due to some participants having magnocellular deficits while the other participants do not have these deficits. This would be in-line with the theoretical account advocated in this paper. Second, differences may be due to some participants having dual tasking difficulties while the other participants do not have these difficulties. This would be an undesirable alternative explanation. Because of the possibility of these two accounts, the peripheral confirmatory experiment was designed. In this experiment, participants needed to name just the orientation of the target as it appeared in only one location on the screen. Thus, participants did not need to dual task.

For the magnocellular account to hold true over the dual tasking one, the target orientation results from the confirmatory experiment should match the target orientation results from the peripheral experiment. For the dual tasking account to hold true, the results from the two experiments should not match. Here, it was found that in the peripheral experiment, there

were group differences in: (1) the number of correct answers made to the orientation of the target at the ISIs of 13 and 47 ms, (2) the least number of correct answers made to the orientation of the target, and (3) the percentage of participants who scored the least number of correct answers to the orientation of the target at the ISI of 47 ms. In all cases, the confirmatory experiment results were different. Because the results from the two experiments did not match, this suggests that the significant (or almost significant) group differences in the target orientation aspect of the peripheral experiment were due to dual tasking difficulties rather than to the presence or absence of magnocellular deficits. This is an issue for the conceptualization of dyslexia co-morbid with ADHD as a visual rather than a phonological problem. The following, however, needs to be considered.

Evidence from Confirmatory Experiment may not Limit this Conceptualization of Dyslexia

There may be another reason why the results from the target orientation aspect of the peripheral experiment and the results from the confirmatory experiment did not match (see Figure 23). Each participant from each group completed the peripheral experiment before completing the confirmatory experiment. The two backward masking tasks were not counter-balanced. It is possible that participants, after completing the peripheral experiment, experienced fatigue or boredom when completing the confirmatory one (i.e., a carry-over effect). This may have especially been the case given that the two experiments were similar in appearance (same size target, target in periphery). Thus, when compared to the peripheral experiment, participants may have been uninterested or less attentive to the confirmatory one. This could lead to participants not seeing a target when they normally would, affecting masking at each ISI. This, in turn, can explain why group differences were not found.

Altogether, when the results from both the target orientation aspect of the peripheral experiment and the confirmatory experiment do not match, there are two interpretations. The dual tasking interpretation implies that the results of the peripheral experiment do not reflect the absence or presence of m-pathway deficits in the groups. Rather, based on the confirmatory experiment, the results reflect dual tasking difficulties. According to the counter-balancing interpretation, however, the results from the peripheral experiment do reflect the absence or presence of magnocellular deficits in the groups. The confirmatory results are different because participants were uninterested in the task. Anecdotally, participants did tend to complain at the end of the confirmatory experiment that it was "boring". The counter-balancing interpretation provides some support for the theoretical account advocated in this paper (that there is a magnocellular deficit in the participants with attention deficits) and limits the possibility that dual tasking difficulties can explain group differences in the target orientation aspect of the peripheral experiment.

Before moving on, it should be mentioned that the carry-over effects of not counter-balancing the experiments in the study, while directly affecting the confirmatory experiment, may not have drastically affected the central experiment. This is because after the confirmatory experiment was administered, participants were given a formal break. After this break, they then switched to completing some paper and pencil tests in a different area of the vision science laboratory. All of this should have helped to reduce fatigue or boredom before completing the central experiment. This may explain why a similar pattern was found in the central experiment as in the target orientation aspect of the peripheral experiment. For instance, a similar pattern in the percentage of participants from each group who were best masked at the highest ISIs of 40 to

47 ms were found in both the central experiment and in the target orientation aspect of the peripheral experiment.

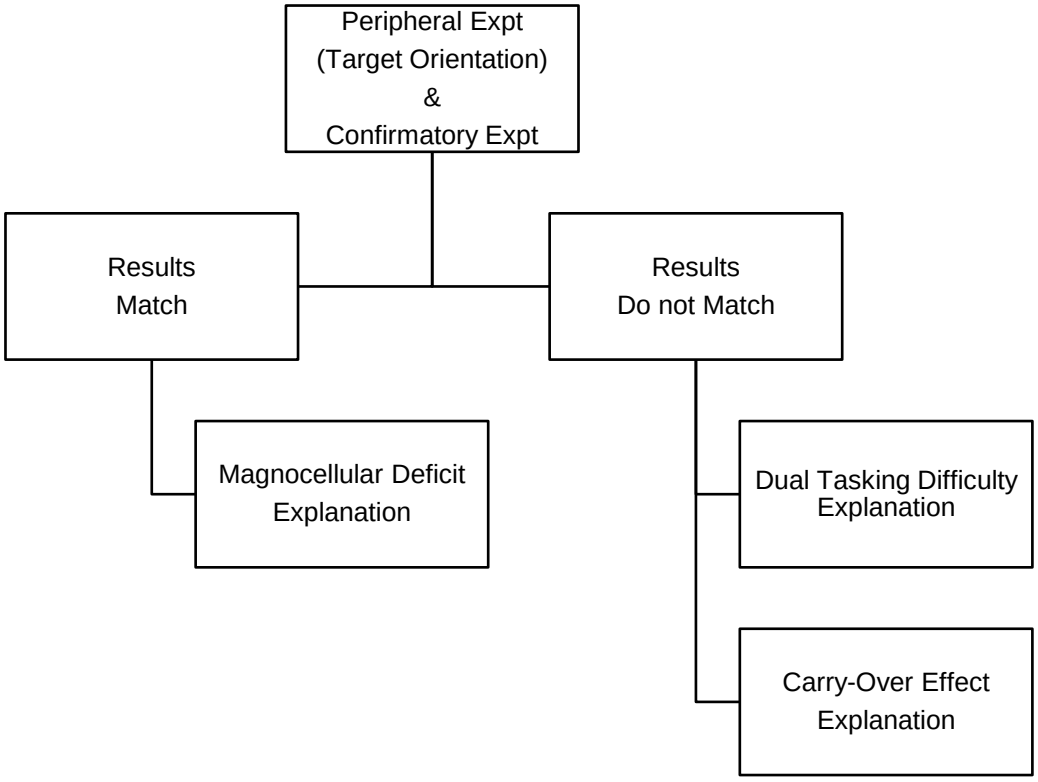

Figure 23. Target orientation aspect of the peripheral experiment and confirmatory experiment

Other Research Findings

Central masking and the target orientation aspect of peripheral masking.

In the present study, there was a tendency for the participants with attention deficits to be masked at a different time interval than the participants without attention deficits whether the target appeared in one of four orientations in the periphery of the screen or in the center of it. This suggests that individuals with attention deficits have magnocellular impairments that affect the orientation perception of stimuli (e.g., low contrast, flickering) appearing in both the periphery of the visual field and in the center of it. Given that the magnocellular pathway is

responsible for motion processing (predominantly, not exclusively; Merigan & Maunsell, 1993), this suggestion is consistent with previous research indicating that motion processing is not different based on peripheral or central visual fields.

McKee and Nakayama (1984) measured the thresholds of some participants for differential motion and velocity discrimination in the central and peripheral visual fields. To find the threshold for differential motion, McKee and Nakayama (1984) presented two adjacent targets that moved in opposite directions. Participants had to identify the direction of one of the two targets. To find the threshold for velocity discrimination, they presented a single target that moved across a distance at one of five velocities (chosen from a narrow range of velocities). Here, participants had to judge whether the target moved faster or slower than the mean of the range. McKee and Nakayama (1984) found that peripheral thresholds for differential motion and velocity discrimination were not better than central thresholds.

Smith, Hess, and Baker (1994) presented their participants with an orientation-direction task at a range of eccentricities (i.e., distances from the point of fixation). This was intended to tap both central and peripheral vision. In the orientation-direction task, a grating stimulus changed in orientation (i.e., vertical or horizontal). The grating also changed in the direction of motion. In the vertical orientation, it could move either left or right and in the horizontal orientation, it could move either up or down. Participants had to judge the grating orientation and the direction of motion. Smith et al. (1994) found that direction sensitivity, when compared to orientation sensitivity, was as good in the periphery as in the center. Research has shown that direction sensitivity is supported by area MT of the magnocellular pathway (Maunsell & Van Essen, 1983).

van de Grind, van Doorn, and Koenderink (1983) presented participants with a motion coherence task in both the periphery and center of the visual field. The motion coherence task involved stimuli that moved randomly (i.e., noise) with a subset of these stimuli moving together in a particular direction (i.e., signal). Participants had to indicate the direction. van de Grind et al. (1983) concluded that the peripheral retina (which gives rise to peripheral vision, which is used to process stimuli in the periphery of the visual field) is not specialized for motion perception. Altogether, these studies show that motion is not processed differently depending on peripheral or central visual fields. It is not surprising, then, that magnocellular deficits in the participants with attention deficits would affect the orientation perception of stimuli appearing in both the peripheral and central visual fields.

Before moving on, it is interesting to note that more group differences were actually found in the target orientation aspect of the peripheral experiment than in the central experiment. This may mean that magnocellular impairments in individuals with attention deficits affect the orientation perception of stimuli appearing in the periphery more than those appearing in the center of the visual field (even though group differences were found in both experiments). It may be that individuals with attention deficits, who have these magnocellular impairments, have "weaker" peripheral processing of stimulus orientation (stimuli that have low contrast and that flicker) when compared to central processing. This can only be speculated at the moment.

Magnocellular impairments that affect the orientation perception of stimuli appearing in the periphery more than those appearing in the center may have an effect on reading. To understand this, a model of reading needs to be introduced. According to Hochberg (1970), when individuals fixate on a word during reading, peripheral cues regarding nearby words are obtained. Peripheral cues include the length of words, the first and last letters of words, spacing

between the words, and shapes of words. These cues help determine which word the eyes should fixate on next, as individuals do not fixate on every single word in a sentence, and facilitate rapid reading.

In theory, m-pathway deficits that affect the orientation perception of stimuli in the periphery could affect the orientation perception of peripheral cues. By affecting these cues, individuals with attention deficits would be deprived of a source of information regarding where to fixate next (e.g., on which word) when reading. As a result, reading would be slow. It is not clear how this theory fits in with the magnocellular theory involving binocular instability (i.e., magnocellular impairments disrupt reading through ineffective binocular control of the two eyes). However, the two theories do share a common feature involving fixation problems.

Peripheral confirmatory masking and central masking.

In the confirmatory experiment, participants needed to use their peripheral vision to detect the target in one of four orientations in a single quadrant. In contrast, in the central experiment, participants needed to use their central vision to detect the target in one of four orientations in the center of the screen. It can be argued, however, that the two experiments were the same if participants did not follow the instructions given to them by the experimenter. For instance, in the confirmatory experiment, participants may have learned which quadrant the target continuously appeared in. Accordingly, they may have directed their gaze and attention to the quadrant rather than to the fixation cross. In doing so, detecting the target would be easier and accuracy would be higher. By doing this, however, participants would have switched from using their peripheral vision to detect the target to using their central vision. Central vision is used whenever a participant looks directly at an object.

It is interesting to note then that because: (1) few group differences in masking were found in the confirmatory experiment, which was a central task if participants did not follow instructions and (2) few group differences in masking were found in the central experiment, it can be argued that magnocellular impairments in individuals with attention deficits do not have much of an effect on the orientation perception of stimuli appearing in the center of the visual field. Because group differences were predominantly found in the target orientation aspect of the peripheral experiment, it can be argued further that magnocellular impairments have more of an effect on the orientation perception of stimuli appearing in the periphery of the visual field (similar to what was discussed at the end of the previous section regarding "weaker" peripheral processing than central processing).

This is plausible but may not be the case. When group differences in best masking at high ISIs were found in the confirmatory experiment, the group differences were not in line with the predictions of this study (i.e., that controls and participants with dyslexia only should be best masked at a similar time interval while participants with both dyslexia and ADHD and participants with ADHD only should be best masked at the same time interval). In contrast, when group differences in best masking at high ISIs were found in the central experiment, the group differences were in line with the predictions[9]. The group pattern in the central experiment may be a more accurate indicator of best masking in the central field than the group pattern in the confirmatory experiment[10], as the central experiment was specifically designed to target central vision whereas the confirmatory experiment was not (it cannot be confirmed whether the

[9] It should be noted that in the confirmatory experiment, the percentages of participants in each group who were best masked at the exact ISI of 47 ms were calculated. In the central experiment, the percentages of participants in each group who were best masked at the ISIs of 40 to 47 ms were calculated.

[10] As mentioned earlier though, the results from the confirmatory experiment may be limited by the contamination of carry-over effects (e.g., boredom).

confirmatory experiment was indeed turned into a central task). Because group differences in masking (in line with predictions) were found in both the central experiment and in the target orientation aspect of the peripheral experiment, it seems that magnocellular impairments in individuals with attention deficits affect the orientation perception of stimuli appearing in the center and periphery of the visual field.

Peripheral masking: Target location and orientation.

In the target orientation aspect of the peripheral experiment, some group differences were found and these were in line with the predictions made in this study. However, in the target location aspect of this experiment, group differences were either: (1) not found or (2) not in line with the predictions made. This would mean that any magnocellular impairment in individuals with both dyslexia and ADHD and in individuals with ADHD only affects the orientation of stimuli more than the location of stimuli, when these stimuli appear in the periphery.
One implication of this involves thinking of the orientation of the Landolt C (gap in the letter facing the left, right, top or bottom of the screen) as the location of the gap within the C.

The location of the gap within the Landolt C spanned a smaller area on the computer screen than the location of the C in one of four quadrants, which spanned a larger area (see Figure 24). Because group differences were found on the location of the gap within the C (i.e., orientation) and not on the location of the C on the screen, this suggests that magnocellular impairments in individuals with attention deficits affect the location of stimuli in the periphery that span smaller areas than larger areas. It is possible, that because input from the magnocellular pathway mediates visual attention (Omtzigt & Hendriks, 2004; Steinman et al., 1997), that impairments in the pathway lead to deficits in visual attention.

The deficits in visual attention, however, may be mild and may only be observed when attention needs to be paid to objects that span small areas rather than large ones. More attention may need to be exerted when viewing objects that span small areas because more distracters, in surrounding areas, are visible. Altogether, magnocellular impairments may lead to visual attention deficits, and these attention deficits may be mild, affecting only the perception of stimuli that span small areas in the periphery. This can thus explain why group differences were found in the location of the gap within the C (i.e., orientation) and not in the location of the C on the screen, when both were presented in the periphery of the visual field.

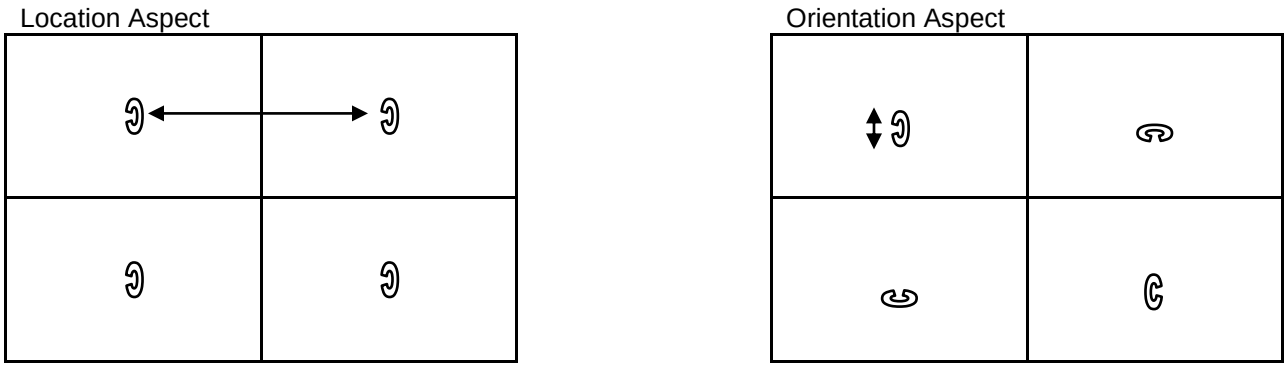

Figure 24. In the peripheral experiment, only one target appeared at a time in one of four locations on the screen and in one of four orientations (i.e., locations of the gap in the C). The location of the C on the screen spanned a larger area than the location of the gap within the C, which spanned a smaller area.

There may be another reason why group differences were not found in the target location aspect of the peripheral experiment: the target location task was too easy. Looking at the data, it seems that participants from each of the four groups performed relatively well on this task across the five ISIs (13, 27, 33, 40, and 47 ms; see Table 2).

Table 2

Each percentage is the average of correct answers of a particular group at an ISI. It can be seen that the percentages are quite high

	13 ms	27 ms	33 ms	40 ms	47 ms
Control	72.44%	65.38%	77.33%	75.00%	73.08%
ADHD	79.17%	79.86%	77.08%	79.17%	82.64%
Dyslexia	66.67%	54.17%	75.00%	58.33%	62.50%
Dyslexia + ADHD	76.39%	72.22%	70.83%	69.44%	69.44%

The target location task may not have been sensitive enough to detect group differences and there may have been a ceiling effect. In the future, this task can be made more difficult by decreasing the size of the target or having it appear further in the periphery of the screen. It is expected that by making the task more difficult, participants with attention deficits would be masked of the target location at low ISIs and participants without attention deficits would be masked at higher ISIs. If this is found, this would suggest that the participants with both dyslexia and ADHD and participants with ADHD only have magnocellular impairments. This would also suggest that the impairments affect the location perception of m-pathway stimuli appearing in the periphery of the visual field. As mentioned before, however, it is not clear what this effect would have on reading given the theory of binocular instability.

Controls and participants with dyslexia.

Throughout this paper, it is mentioned generally that the participants with attention deficits differed from the participants without attention deficits in masking. However, controls and participants with dyslexia only were not directly compared in this study to see if they were

indeed masked at the same range of ISIs. Throughout several of the analyses presented in the results section, a Bonferroni correction was applied and so the number of comparisons was kept to two. This was done so that the new critical value would not be too small (a critical value that is too small can lead to a type two error in which no significant findings are found when there are significant differences between groups). The comparisons were reserved for: (1) dyslexia only versus dyslexia co-morbid with ADHD and (2) dyslexia only versus ADHD only. In the study here, it was more important to determine if dyslexia only was different from dyslexia that is co-morbid with ADHD. This could settle whether there are magnocellular deficits in dyslexia only or in dyslexia co-morbid with ADHD. In turn, this could settle the debate on whether dyslexia is a visual or language problem.

Research Strengths and Weaknesses

Researchers who have examined whether or not there are magnocellular deficits in dyslexia have made calls for future studies to address dyslexia and its co-morbidities. This study was one of the first to specifically look at dyslexia that is co-morbid with ADHD. The study design also allowed for a thorough examination of magnocellular deficits in individuals with attention deficits by comparing four groups of participants (controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only). On a different note, this study adds to the small body of research on adults with ADHD. For instance, within this small body of research, most of the studies focus on cognitive function and almost no studies focus on vision or visual processing in the brain. The present study may be one of the first to examine visual processing deficits in adults with ADHD.

This study had a number of weaknesses. The first weakness concerns the low and uneven sample sizes across the four groups of participants. These weaknesses, during statistical analysis,

led to the violation of parametric ANOVA assumptions (e.g., normally distributed data). Because of this, non-parametric ANOVAs (e.g., Kruskal-Wallis tests) were used and these have less power to detect genuine differences between groups when such differences really exist. Non-parametric ANOVAs have less power as they are based on ranked data. Ranked data contain less information about how large or small the difference between raw scores is. This can result in misleading findings of non-significance (see Table 3 for example of how this works). It is interesting to note that even with non-parametric tests, group differences were still found in the target orientation aspect of the peripheral experiment and in the central experiment. With larger sample sizes, it is expected that group differences in masking would be more pronounced and that these differences would reflect the presence of magnocellular deficits in some participants and the absence of these deficits in other participants.

Table 3

Example of how ranked data, which contain less information about how large or small the difference between raw scores is, can lead to a loss of power to detect genuine differences. The difference between the raw scores of 100 and 5 is 95 and the difference between the raw scores of 10 and 5 is 5. However, when these raw scores are ranked, they are given the same values (100 is greater than 5 and 10 is greater than 5, so both receive a rank of 2). This attenuates the differences between scores.

Raw Score	Rank
100	2
5	1

Raw Score	Rank
10	2
5	1

The second weakness concerns the use of schizophrenia research to support the notion that individuals with ADHD (i.e., individuals with both dyslexia and ADHD, individuals with ADHD only) have magnocellular impairments. It was postulated that because individuals with

schizophrenia have attention deficits and magnocellular impairments, that individuals with ADHD, because of the attention deficits, would have magnocellular impairments as well. While research suggests that the nature of the attention deficits in schizophrenia and ADHD are similar (e.g., Kairalla et al., 2008; Marchetta et al., 2008), building a strong case for the possibility of magnocellular impairments in ADHD, the two disorders are inevitably quite different from one another. Thus, it may be that in schizophrenia, it is the combination of attention deficits and some other symptom that is related to the magnocellular problem. In ADHD, this other symptom would be missing (as it would be unique to schizophrenia) and this may explain why in the present study, it was only modestly found that the individuals with attention deficits had magnocellular impairments. Nevertheless, the one study by Rund et al. (1996) did find that adolescents with ADHD performed similarly to the adolescents with schizophrenia and that both groups performed differently from controls on a backward masking task. This provides some direct evidence that the theoretical account of the present study was tenable (see Figure 2).

A third weakness of the present research concerns the peripheral experiment. The peripheral experiment required the identification of both the location and orientation of the target. This introduced a dual tasking alternative explanation (which then introduced the counter-balancing explanation). In the future, it may be better to test peripheral target location and peripheral target orientation in two separate experiments. To test peripheral target location, a backward masking experiment could be designed so that the target appears in only one orientation but in one of four quadrants. To test peripheral target orientation, an experiment could be designed so that the target appears in one of four orientations but in only one quadrant. This would be similar to the confirmatory experiment in the present study, except that five ISIs would be tested rather than just two. In addition, it seems that the target location aspect was too

easy for the participants of each group and led to the ceiling effect observed. As mentioned before, this aspect of the peripheral experiment should be made more difficult by decreasing the size of the target or having it appear further in the periphery.

Research Applications

Some of the findings of this study suggest there is: (1) a magnocellular deficit in the participants with both dyslexia and ADHD and in the participants with ADHD only and (2) no deficit in the participants with dyslexia only. These findings can be applied to the work of clinical and educational psychologists: when diagnosing an individual with dyslexia, it is imperative to also check for attention deficits (i.e., ADHD). Whether or not the dyslexia is co-morbid with ADHD has implications for the type of remediation that is given to address the reading problems observed. In particular, individuals who have both dyslexia and ADHD may benefit more from vision-based remediation than from a phonological-based one.

Vision-based remediation can include monocular occlusion. Monocular occlusion, or covering one of two eyes, is a procedure that is often done on young children to improve their binocular stability. By covering one of the two eyes, it is believed that the seeing eye learns to control its own direction without interference from the occluded eye. The occluded eye follows suit and learns to control its own direction as well. Researchers such as Stein et al. (2000) have found that monocular occlusion can lead to better binocular stability and reading in children with dyslexia, ages 7 to 11 years old.

For maximal success, vision-based remediation would need to be applied at a young age when the visual system of children is still plastic (i.e., during the critical window of visual development). This raises an interesting question of whether the remediation of magnocellular impairment/binocular instability during the critical window would lead to the alleviation of

reading problems later on in life. This seems possible given the connections between the m-pathway, binocular control, and reading. One difficulty, however, may be in pinpointing which child is in need of remediation during the critical window, as he or she may not yet have a diagnosis of dyslexia co-morbid with ADHD or ADHD only (conditions, as has been found in this paper, to be linked to magnocellular deficits). For instance, reading difficulties may be attributed to the child being too young to be a proficient reader and not to a developing dyslexia problem. Attention deficits may be attributed to the child being too curious about multiple events in the environment and not to a developing ADHD problem. One way around this may be for specialists, such as optometrists, to specifically check for binocular control problems in children during eye exams. If problems are found, they should be addressed as soon as possible. Delays could have detrimental, long-lasting effects on reading.

On a different note, but still within the realm of research applications, it is worthwhile to check for reading deficits in ADHD. Individuals with ADHD have been found to attain less education than they are capable of (Biederman et al., 2008) and this may be due to reading problems that are overlooked, due to the primary problem of inattention and/or hyperactivity-impulsivity in ADHD. To help such individuals succeed in the classroom-setting, it is recommended that remediation address both reading at a visual level and inattention and/or hyperactivity-impulsivity problems.

Future Studies

There are a number of avenues for future research. This study was one of the first to provide some evidence that: (1) there are magnocellular deficits in individuals with both dyslexia and ADHD and in individuals with ADHD only and (2) there are no such deficits in individuals with dyslexia only. Future research may want to confirm these findings using different

techniques. For instance, to check whether the m-pathways of the participants with attention deficits do indeed process relevant stimuli (e.g., low contrast, moving) at a different time than participants without attention deficits, electroencephalography (EEG) could be used and visual evoked potentials could be recorded. EEG has high temporal resolution and should be able to reveal more precisely any m-pathway hyper- or hypo-activity.

Another study could be done in which controls, individuals with dyslexia only, individuals with both dyslexia and ADHD, and individuals with ADHD only complete a motion coherence experiment. All participants would, for example, view low contrast dots moving randomly on a screen with a subset of these dots moving together in a particular direction. If the participants with attention deficits (dyslexia co-morbid with ADHD, ADHD only) are less able to state the direction of the dots than the participants without attention deficits (controls, dyslexia only), this would suggest that they have magnocellular problems. Overall, the point is that more research should be done so that a stronger case can be made of an m-pathway deficit in individuals with attention deficits.

Future research may also want to investigate whether adults with attention deficits have (1) magnocellular deficits that lead to binocular problems and (2) binocular problems that lead to reading difficulties. Controls, participants with dyslexia only, participants with both dyslexia and ADHD, and participants with ADHD only could complete tests of magnocellular function, binocular instability, and reading. Participants with attention deficits should have magnocellular impairments and binocular problems in comparison to those without attention deficits. Furthermore, participants with dyslexia only and participants with both dyslexia and ADHD should have reading difficulties (as dyslexia is a reading disorder). Interestingly, however,

participants with ADHD should have reading difficulties as well. Controls would be spared. Analyses could be done to investigate the relationships between these tests.

In terms of research applications, it was noted that individuals with ADHD, because of a magnocellular deficit, may have reading problems too. Not all individuals with ADHD may have visual reading difficulties though. This may have to do with the subtypes of ADHD, which include: (1) inattentiveness, (2) hyperactivity-impulsivity, and (3) combined, in which there are inattentive and hyperactive-impulsive components. It is possible that only the individuals with the subtypes involving inattention would have magnocellular deficits that would then affect binocular control and reading. Individuals with the subtype of hyperactivity-impulsivity would be spared. This expected outcome is based on research indicating that individuals with schizophrenia have attention deficits and magnocellular impairments (Kairalla et al., 2008; Marchetta et al., 2008; Rund et al., 1996; Schechter et al., 2005).

To test this expectation, future research could recruit individuals with the three subtypes of ADHD and have them complete the experiments of this study. The features of the central and peripheral backward masking experiments were selected so that they maximally stimulated the m-pathway over the other pathways of the visual system. These experiments could thus reveal whether there are m-pathway impairments in the individuals with the subtype of ADHD involving inattention.

Conclusion

There was some, but modest, evidence for the participants with attention deficits (dyslexia co-morbid with ADHD, ADHD only) being masked of the target at a different time interval than the participants without attention deficits (i.e., dyslexia only) whether the target appeared in one of four orientations in the center of the screen or in the periphery of it. The

implications of this are that: (1) dyslexia co-morbid with ADHD can be accounted for by a visual magnocellular model while dyslexia only can be accounted for by a different model, such as the phonological one and (2) ADHD only has magnocellular impairments, which suggests they have reading problems too. Both of these implications are based on schizophrenia research.

Results were discussed in-depth and the strengths and weakness of this study were presented. This study was one of the first to examine whether magnocellular impairments exist in dyslexia co-morbid with ADHD or in dyslexia only. It was also one of the first to examine magnocellular impairments in adults with ADHD only. The main weakness of this study was that there were low and uneven sample sizes across the four groups of participants. Overall, research applications were recommended and future studies were proposed.

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Glossary

Attention Deficit Hyperactivity Disorder (ADHD): A neurodevelopmental disorder characterized by persistent inattention and/or hyperactivity-impulsivity. These features are maladaptive and inconsistent with developmental level.

Best Masking: When an individual is best masked, he or she scored the least number of correct answers.

Best Mask ISI: The ISI at which an individual scores the least number of correct answers.

Dyslexia: A reading disorder that is diagnosed when an individual's reading level is unexpectedly low given the individual's age, intelligence, and education. Reading level must interfere with academic achievement and reading difficulties need to exceed those associated with a sensory deficit, if one exists.

Inter-stimulus interval (ISI): The time between the target and the mask in a backward masking experiment.

Magnocellular Pathway (M-Pathway): One of three pathways in the visual system of primates. This pathway is most stimulated by objects that move and flicker (quick onset, quick offset). It is also most stimulated by objects that have low luminance contrasts, low spatial frequency, and high temporal frequency. The m-pathway processes these objects with short bursts of neural firing (i.e., transient neural firing).

Parvocellular Pathway (P-Pathway): One of three pathways in the visual system of primates. This pathway is most stimulated by object colour, texture, and pattern. It is also most stimulated by objects that have high luminance contrasts, high spatial frequency, and low temporal frequency. The p-pathway processes these objects with sustained neural firing.

Phonological Model of Dyslexia: In this model, it is assumed that speech is inherent and natural whereas reading must be learned. New readers need to understand that spoken words can be broken down into phonemes (the smallest units of sound that convey meaning in a language), and that letters in a written word represent these phonemes. Regarding individuals with dyslexia, the conventional perspective is that they struggle with learning how letters and their sounds are related.

Schizophrenia: A disorder in which symptoms include delusions, hallucinations, disorganized speech, catatonic behavior, affective flattening, alogia, and/or avolition. Research has found that individuals with schizophrenia have attention deficits and magnocellular impairments.

Spatial Frequency: A measure of how fine a re-occurring visual pattern is in terms of the number of lines or points per unit distance. It is often given in cycles per degree on the retina (c/deg).

Sustained Subsystem: The sustained subsystem involves the parvocellular pathway. In research, the two are often cited as being the same.

Temporal Frequency: The speed of a drifting stimulus (e.g., drifting grating) in hertz multiplied by the spatial frequency.

Transient Subsystem: The transient subsystem involves the magnocellular pathway. In research, the two are often cited as being the same.

Visual Backward Masking: When backward masking is performed on a computer, a target stimulus is presented on a screen and is quickly followed by a masking stimulus. The aim of the masking stimulus is to interrupt the processing of the target stimulus and to reduce the visibility of it. Individuals participating in a backward masking experiment are then asked to indicate the orientation or location of the target. The features of backward masking stimulate the magnocellular and parvocellular pathways.

Visual Magnocellular Proposal (also referred to as a model): In this proposal, magnocellular deficits in dyslexia are what lead to the reading problems in the disorder. Magnocellular deficits can lead to reading problems through the lack of parvocellular suppression or through binocular instability.

Worst Masking: When an individual is worst masked, he or she scored the most number of correct answers.