Colour vision in Attention-Deficit/ Hyperactivity Disorder: A pilot visual evoked potential study

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Abstract

Background: Individuals with Attention-Deficit/Hyperactivity Disorder (ADHD) are reported to manifest visual problems (including ophthalmological and color perception problems, particularly for blue-yellow stimuli), but findings are inconsistent. Accordingly, this study investigated visual function and color perception in adolescents with ADHD using VEP.

Method: Participants were 31 adolescents (aged 13-18); 16 with a confirmed diagnosis of ADHD, and 15 healthy peers, matched for age, gender, and IQ. All underwent ophthalmological exam, color vision testing (Mollon-Reffin Minimalist Colour Vision Test), as well as electrophysiological testing (color Visual Evoked Potentials; cVEP) which measured the latency and amplitude of the neural P1 response to chromatic stimuli (Blue-Yellow, Red-Green).

Result: No group differences were found in clinical measure of color perception or opthalmological exam. However, significantly larger P1 amplitude was found for blue and yellow stimuli, but not red/green stimuli, in the ADHD group compared to controls.

Discussion: Larger amplitude in the P1 component for blue-yellow in ADHD group compared to control group may account for no difference in colour perception task. Perhaps activating more resources in early sensory processing (P1) compensated for any underlying problems including compromised retinal input of s-cones due to hypo-dopaminergic tone.

Keywords: ADHD, adolescent, color vision deficit, Visual evoked potential

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Introduction

Attention-deficit/Hyperactivity disorder (ADHD) is one of the most frequently diagnosed childhood psychiatric disorders, with worldwide prevalence rates estimated at 5.3% (Polanczyk& Jensen, 2008). However, despite the long history of research since its first medical description in 1775 (Barkley & Peters, 2012), to date, it remains unclear what is the 'deficit' in ADHD. Current theories posit that executive function deficits account for ADHD symptoms. However, based on substantial number of studies, ADHD is also associated with visual perceptual problems that appear unrelated to any executive dysfunction (See appendix table 1). Especially, ADHD is a neuro-developmental disorder which is associated with delayed cortical maturation in many regions, including the occipital cortex (Shaw et al., 2007; Hoekzema et al., 2012). Specifically, color perception may be altered in ADHD population (see appendix table 2). For instance, in our previous study, young adults with ADHD reported significantly more self-perceived visual difficulties in everyday tasks as well as poorer hue discrimination specifically on blue (Kim, Chen, & Tannock, 2013). Furthermore, children with ADHD have been found to score poorly on clinical tests of blue-yellow color perception, but not red-green (Banaschewski et al., 2006, Roessner et al., 2008), and showed decreased game performance in a virtual environment when important on-screen information was displayed predominantly in blue-yellow colors compared to performance with information displayed in red-green colors (Silva & Frere, 2011). Finally, several studies report decreased speed in color processing in the ADHD population (Tannock et al., 2000; Lawrence et al., 2004). The possibility of color perception problems in ADHD is of clinical importance, given the extensive use of color in educational settings, as well as the frequent use of color stimuli in many of the standard neuropsychological tests used in the assessment for ADHD and related disorders (e.g. Colour-Word Stroop Test, Wisconsin Card Sorting Test, A Quick Test of Cognitive Speed, Rapid Automatized Naming).

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Color vision mechanisms, particularly short-wavelength pathway, is particularly vulnerable to insult from toxins, and are highly sensitive to CNS drugs and the neurotransmitter, dopamine. Hence, the "retinal dopaminergic" hypothesis of color vision (Tannock, Banaschewski, & Gold, 2006) proposes that a deficiency in central nervous system (CNS) dopamine in ADHD may induce a hypo-dopaminergic tone in the retina, which in turn would have deleterious effects on short-wavelength (S) cones, which are sensitive to blue-yellow light wavelengths. S-cones are very sensitive to dopamine (as well as other neurochemical agents) and relatively scarce in number, so that the purported low dopaminergic tone in ADHD may affect their blue color perception. To date, tests of this hypothesis in the ADHD population have relied solely on clinical tests of color perception, which do not inform about mechanisms underlying poor performance on B-Y stimuli. Also, most of the studies focused in testing children with ADHD (Baneschewski et al, 2006; Roessner et al., 2008; Kim et al., 2013).

Accordingly, this pilot study aimed to explore the B-Y color mechanism in an extended population (adolescents with ADHD) using electrophysiological technique (colour visual-evoked potential; VEP). VEP technique is suggested to be as a sensitive and objective measure of chromatic input in visual pathways (Crognale et al., 1993). In this study, we measured the neural response (P1) to chromatic and achromatic stimuli, thereby providing a more direct assay of color processing in this population. The P1 component of the VEP (peak latency 136-146 msec) is an early response to the visual stimuli and it is mainly generated from the dorsal extrastriate cortex where color processing is localized (Luck, 2005; Di Russo et al., 2001; Conway et al, 2007, 2010; Wade et al., 2002). In addition, we conducted an ophthalmological exam (e.g., visual acuity, refraction, fundus exam) to test general visual functions in ADHD. Finally, color perception was assessed with a test sensitive to blue-yellow perceptual problems (Mollon-Reffin Minimalist Color Vision Test), but which

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minimizes demands on attention (Shute & Westall, 2000). We hypothesized that the adolescents with ADHD would show normal visual function on ophthalmological exam, but altered B-Y color vision as indexed by both the clinical color vision test and by the latency or amplitude of P1. Specifically, we expected ADHD group to show more error in the clinical color vision test, and longer latency as well as decreased amplitude of P1 for B-Y compared to control group.

Methods

Participants:

A total of 31 adolescents, aged 13 to 18 years, participated; 16 (81% male, mean age: 16) with a confirmed DSM-IV (APA, 1994) diagnosis of ADHD (described below) and 15 (67% male, mean age: 15) healthy controls matched for age, sex, and IQ. No significant differences were found in age and sex between the groups. Adolescents with confirmed ADHD were recruited from a larger-scale study on working memory (Canadian Institutes of Health Research operating grant # 11398); those in the comparison group were recruited through notices posted in the research setting (a large pediatric hospital in an urban area). All adolescents participating in the study were native English speakers. Adolescents were excluded if mothers reported a history of major perinatal complications such as prematurity, low birth weight, any history or current presentation of psychosis, comorbid Tourette syndrome, phenylketonuria, autism, or other pervasive developmental disorders. Also adolescents were excluded if they had a history or current use of cocaine or other substances, or had below average intellectual functioning (defined as a standard score of at least 80 on either the Verbal or Performance Scale of the WISC-III).

The DSM-IV diagnosis of ADHD had been confirmed by a systematic and comprehensive clinical diagnostic assessment conducted within the past one to two years, as a part of the

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larger scale study. Assessment consisted of a semi-structured clinical diagnostic interview [Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version; K-SADS-PL; Kaufman et al., 1997], as well as the Conners' Rating Scales-Revised (Conners, 1997), completed by parents and teachers. The K-SADS had been conducted separately with the adolescent and parent, and the clinician summarized the information from both informants. Diagnosis of ADHD in adolescents had been based on the following algorithm: 1) met DSM-IV criteria according to the clinician summary based on the K-SADS-PL interviews; and 2) met the clinical cut-offs for inattentive or hyperactive/impulsive symptoms on the Conners' teacher questionnaires (t-score > 70) to confirm pervasiveness of symptoms across settings.

For the current study, parents of all participants were asked to complete the Strengths and Weaknesses of ADHD-symptoms and Normal Behavior Scale (SWAN; Swanson et al., 2005), using a 7-point likert scale for each item (score of '1' indicating the child's abilities were far below those of peers; score of '7' indicating abilities far above those of peers). Total scores for inattention and hyperactivity/impulsivity were computed, with lower scores indicating more problems. Also, parents as well as teachers completed the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001) to obtain standardized ratings of current behaviour. Adolescents in the comparison group who had any scores in the clinical range were excluded. Informed consent from the participating adolescents and their parents was obtained before the test.

Participants with ADHD who were being treated with stimulant medication (n= 7; 35% of the sample) were requested to stop any stimulant medication for at least 24 hours prior to the study. However, since we had no reliable method for confirming that participants had indeed ceased their treatment for more than 24 hours, we opted to classify participants with ADHD into two groups: those with and without current medication treatment.

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This study was approved by our institutional Research Ethics Board; all participants provided written informed consent prior to commencing the study.

Measures:

Opthalmological exam: a comprehensive vision exam, conducted by a trained ophthalmologist, included the following measures:

- *Contrast sensitivity* (Pelli, Robson & Wilkins 1988): Contrast sensitivity as measured by the Pelli-Robson Contrast Sensitivity Test which provides a quick, reliable and widely accepted method used in clinical setting. Higher scores indicate better contrast sensitivity (i.e. can discriminate fainter letters better on a white chart). The highest possible score is 2.25.
- *Visual acuity* (Vistech Consultants, Inc. Dayton, USA): It was measured with the logMAR crowded test. Lower scores indicate better visual acuity/resolution. Lowest score is -0.3.
- *Refraction* (Saunders et al., 1992): It was measured using a near retinoscopy technique. Spherical correction and cylindrical correction are reported for left, right, and both eyes. Since uncorrected refractive error might confound the results, adolescents with uncorrected refractive error greater than 3.00 diopters spherical correction, or 1.50 diopters cylindrical correction was excluded from the study.
- *Fundus exam*: A basic fundus examination was carried out with the ophthalmoscope to determine the ocular media, posterior pole and macular area of the retina.

Mollon-Reffin Minimalist Color Vision Test (M-RM): M-RM was chosen for its sensitivity and a good specificity for tritan (blue-yellow) errors, particularly with young participants (Shute et al., 2000). M-RM requires the individual to identify a single colored cap from 5 grey caps of varying lightness. Three sets of test caps were used that coincide with tritan (blue-yellow), protan (red), deutan (green) confusion axis. For each set, the number of the

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least saturated caps (1 to 3) that the participant correctly identifies is used as the participant's score.

Visual Evoked Potentials (VEP; NeuroScan Acquire 4.0 program): VEP is an objective, non-invasive technique that particularly reflects cone activity in the central 6-10 degrees in the retina (Regan, 1989). It permits recording of an occipital lobe brain wave in response to visual stimulation that begins in the retina and ends at the visual cortex (Young, Eggenberger, Kaufman, 2012). In the current study, three types of stimuli were used. The first, the achromatic grating was a white-gray luminance stimulus to verify that meaningful VEP signals could be collected. The second was an isoluminant grating for long and medium wavelength color mechanisms (red-green). The third type was an isoluminant S-grating specific for S-cone activation-deactivation (blue-yellow). Achromatic and chromatic stimuli were presented in a patterned onset-offset presentation. This means that the stimulus alternated between "on" (for 100 ms) and "off" (for 400 ms) at a repeated rate of 2Hz, until 60 sweeps were collected. The time of luminance presentation consistently occurred between chromatic stimuli so as not to saturate the colour vision system.

Stimulus parameters were selected to optimize the chromatic response and differentiate between the chromatic and achromatic VEP response (see Elia et al., 2005 for the details). Chromatic and achromatic stimuli were produced using Vision Research Graphics (VRG) software (Durhan, NH). Specifically, the red-green color grating consisted of vertical bars varying from red to green with respective chromaticity coordinates of x=0.3574, y=0.3099 and x=0.3064, y=0.3372. The violet to yellow-green grating consisted of alternating violet (x=0.2893, y=0.2496) and yellow-green (x=0.3409, y=0.3523) bars. Each of the color stimuli pairs: red and green or blue or yellow were isoluminant. This was to ensure that the cortical responses being recorded arose predominantly from color selective cortical cells and not from luminance-responsive cells (Suttle & Harding, 1999). These stimuli were presented on a 21-

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inch RGB color graphics monitor (FlexScan f930; Eizo, Cypress, CA) with 26º X 20º field dimensions.

We positioned 6-mm diameter gold disc electrodes (Genuine F-F5GH; Grass Instrument Division, Astro-Med, Inc., West Warwick, RI) with protected terminals (Safelead; Grass) on the scalp, as stated in the international 10-20 system of electrode placement, on the visual occipital cortex in positions Oz, O1, and O2 along with two additional electrodes on nonvisual areas of the cortex at Pz (ground) and Cz (reference), to obtain cortical responses to color stimuli. Color VEPs were recorded at a viewing distance of 75 cm. Each participant was tested binocularly.

For VEP data analysis**,** waveforms were recorded for achromatic, L-M and S patterns. Sixty presentations were acquired and averaged for each stimulus, which was presented twice. Thus, a total of 120 presentations per each condition were recorded. We measured both VEP latency as well as amplitude. Since latency of VEP waveform generated by chromatic stimuli (both red-green and blue-yellow) is typically negative wave, in adults (Porciatti & Sartucci, 1998), the latency of chromatic onset-offset VEP data was measured from pattern onset to the first negative component. Peak amplitudes were measured from the trough of the first negative wave to the peak of the preceding positive wave for wave generated by chromatic stimuli (Figure 1 shows an example for a male participant in this study).

Analysis:

Data points (behavioural and ERP) with SD's >3 were regarded as outliers and adjusted using a winsorizing technique (Tabachnick, B., & Fidell, L., 2001). This was applied to a total of seven data points: one data point from Left Acuity, Left contrast sensitivity, right spherical correction, Left cylindrical correction, Right cylindrical correction, and 2 data points from Red-Green Latency. Also, 3 control participants were excluded from VEP tests due to weak VEP signals and very low motivations (observed tiredness, boredom and lack of sleep). We

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used relative amplitude (difference in luminance to chromatic amplitude) to control for inter-individual variability. Planned orthogonal contrast analyses were used to test the hypothesized group differences in color perception and other visual functions. We first compared the ADHD and control groups, and then the medicated versus non-medicated ADHD groups. Effect sizes (ES) were calculated using Cohen's *d* (Cohen, 1989). Conventionally, Cohen's d ranging 0.2-.03 is considered to be a small effect size, 0.5 as medium and 0.8 as large, respectively.

Results

Sample characteristics and performance on vision measures are summarized in Tables 1 and 2, respectively. As expected, adolescents with ADHD showed significantly more inattentive [t (27) =-6.627, p=.000] and hyperactivity symptoms [t (27) =-2.990, p=.006] than control adolescents based on parent's report on SWAN, but the two ADHD subgroups did not differ. Also, ADHD group showed significantly more overall difficulties in school [t (27) = -4.233, p=.000] as well as in home settings [t (27) = 3.304, p=.003].

There were no group differences in general vision based on the ophthalmological tests including visual acuity, contrast sensitivity, and refraction. Clinical notes on the fundus exam suggested that the fundus was within normal limits for virtually all participants except 1 participant in each ADHD and Control group (see appendix 3for detail). Moreover, the ADHD and comparison groups did not differ in color perception, as measured with M-RM (see Table 2).

On VEP measures, no significant group differences were found for the P1 latency, but the ADHD group (both medicated and non-medicated participants) showed significantly larger P1 amplitude in response to blue-yellow stimuli than did the comparison group [t (25) = 2.35, p<.05; Cohen's d= .80, see figure 2], but the groups did not differ in either latency or amplitude in terms of the P1 response to red-green stimuli [t (24) = .183, p=.86; Cohen's d=

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0.11]. The group differences in P1 amplitude in response to blue-yellow stimuli appear to be driven primarily by the 'medicated' ADHD group, since their P1 amplitude was significantly larger compared to that of the non-medicated subgroup [t (25) = 2.18, p<.05; Cohen's d= .77].

Inattentive symptoms from parent rating of SWAN were significantly correlated with P1 amplitude in response to B-Y stimuli [r (27) = -.386, p=.046] but not for R-G stimuli [r (27) = -.195, p=.330], indicating that more severe inattention was related to greater P1 amplitude for B-Y (see scatter plot in figure 3). By contrast, there was no significant relationship between hyperactivity/impulsivity scores and the P1 amplitude for either B-Y [r (27) = -.286, p=.146 or R-G stimuli [r (27) = -.132, p=.495]

Discussion

This study represents the first attempt to use VEP as well as a clinical test to assay color perception in adolescents with ADHD. Moreover, we conducted ophthalmological testing to allow us to disaggregate color perception problems from problems in vision. The major findings in this pilot study were that: 1) the ADHD group showed a much larger P1 amplitude in response to blue-yellow stimuli than did the comparison group, but did not differ in terms of the P1 latency, and there were no group differences in the P1 amplitude or latency in response to red or green stimuli; 2) inattention significantly correlated with the P1 amplitude, but only for B-Y stimuli; and 3) there was no evidence of either ophthalmological or color perception problems in the ADHD group based on the clinical measures

The present study yielded several novel findings, including evidence of greater amplitude in the P1 component of the neural response to B-Y chromatic stimuli in the ADHD group, together with a significant positive relationship between severity of inattention symptoms and the P1 amplitude for B-Y stimuli. The magnitude of this group difference in P1 amplitude was notably larger for blue-yellow chromatic stimuli compared

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to that for red-green stimuli (e.g., Cohen's d for B-Y was .80; and for R-G was .11). Although the group difference in the P1 amplitude for B-Y stimuli appears to be driven primarily by the adolescents with ADHD who were being treated with stimulant medication, there are several reasons why we do not believe that the group difference can be attributed to the effects of stimulant medication per se. First, P1 amplitude correlated positively with the SWAN inattention scores (see Figure 3) and the two ADHD subgroups did not differ in SWAN scores (see Table 1). Second, participants in the 'medicated' group had been asked to stop their medication for at least 24 hours before the test session and indicated that they had done so, although we were unable to confirm this was the case. Thus, we believe that the finding does indicate that the ADHD group had greater P1 amplitude for B-Y stimuli than controls, but did not differ in P1 latency or amplitude for R-G stimuli. This interpretation is further supported by the specific and positive correlation between the severity of inattention and P1 amplitude for B-Y stimuli.

A differential neural response to B-Y and R-G stimuli can be explained by different visual pathways that blue-yellow (Short wavelength-cones) and red-green (Medium-Long wavelength cones) retinotopic information are connected to (Figure 4). Specifically, B-Y information is transmitted to koniocellular layer of the LGN, and from LGN, they are directly projected to Middle temporal area (MT) and the parietal bypassing V1 (Martin, White, Goodchild, Wilder, & Sefton, 1997; Roy et al., 2009; White, Wilder, Goodchild, Sefton, & Martin, 1998; Jayakumar, Dreher, & Vidyasagar, 2013). By contrast, the smaller parvocellular ganglion cells, which are linked to long and medium wavelength cones (L-M cones or "red" and "green" cones), project to area V1 of the primary visual cortex, through V2 and V4 to areas of the inferior temporal lobe (Lamme, Super, & Spekreijse, 1998). The dorsal visual stream, to which B-Y pathways project, is suggested to be closely linked to attention mechanism due to the anatomical proximity with areas that operate spatial

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attention (posterior parietal lobe; pulvinar nucleus of the thalamus and superior colliculus) such as directing attention with and without saccadic movement (Posner & Peterson, 1990; Williams et al., 1994; Posner, 1988). Furthermore, area MT has been found to modulate attention-dependent responses and direct attention in the early visual cortex (Bisley & Pasternak, 2000; Saalmann, Pigarev, & Vidyasagar, 2007) Interestingly, the pathway carrying B-Y signals, being presumptively an early evolutionary invention is thought to be co-opted to aid in focal-spatial attention (Jayakumar et al., 2013). Children with ADHD were found to have problem in directing attention (Swanson et al., 1991). In the presence of impaired attention and a hypo-dopaminergic state in retina in individuals with ADHD, it is possible that the greater P1 amplitude in ADHD reflects a compensatory over-activation of the extrastriate cortex, especially in response to B-Y chromatic stimuli. We can speculate that adolescents with ADHD were challenged in processing colour information, hence required greater activation in extrastriate area.

Notably, our null findings in colour vision perception in clinical tests are in contradistinction with findings from previous studies reporting more errors in blue-yellow color perception in the ADHD group compared to controls (Banaschewski et al., 2006; Roessner et al., 2008). A methodological difference may account for this discrepant outcome. The Farnsworth-Munsell 100 Hue Test: (FMT), used in previous studies, requires the participant to arrange the caps in the best colour order (i.e. from yellowish green to turquoise green). This process involves both accurate movement execution and sustained attention, which are known to be impaired in ADHD. Furthermore, color perception and motor impairments have been associated (although not causal) with FMT errors scores in patients with Parkinson's disease (Haug et al., 1995). By contrast, the M-RM, used in the current study, does not place large demands on attention ability (Shute et al., 2000). Thus, on the one hand, previous findings of B-Y color perception differences may be attributable

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in part to group differences in attention or motor control. On the other hand, the M-RM assesses blue-gray discrimination and not blue-yellow contrast, which we speculate is what may be impaired by a dopamine deficit in the ADHD population. Thus, future studies might want to give greater consideration to the choice of color perception tests and to incorporate other approaches to assaying color vision.

We acknowledge the limitations of this pilot study, which need to be taken into account when interpreting the findings. Sample sizes were small particularly for the comparison of the two ADHD groups, which limits the generalizability of the findings and necessitate their replication in larger samples**.** Also, although we were able to confirm which participants were being treated with medication, we were unable to confirm whether they had stopped medication at least 24 hours prior to the study as requested. Moreover, we acknowledge that this duration of washout may not be sufficient to eliminate any residual central (or retinal) effects of medication. We attempted to deal with the possible confound of medication by comparing those who were and were not being treated with medication. However, observed differences in the P1 response to B-Y stimuli in the two ADHD groups cannot be attributable to the effects of medication, because it is quite possible that those receiving medication differ in a systematic way from those not receiving mediation. Also, it is possible that the VEP latency and amplitude measured in occipital lobe may not capture the impairment at a receptor level caused by hypo-dopaminergic condition in ADHD group. Multifocal electroretinogram, a tool to detect and quantify central cone function, especially in disease stages with no or subtle visible retinal changes, might be an option. This way, we can directly observe the effect of low dopamine condition in retina particularly to blue and yellow cones.

Despite the limitations, we believe our preliminary findings provide foundations for future investigations on this topic. Future studies should include different age groups and

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more precise and effective tests to assess neural and behavioral components of colour perception, and to investigate the effects of covert attention on color perception of B-Y versus R-G chromatic stimuli.

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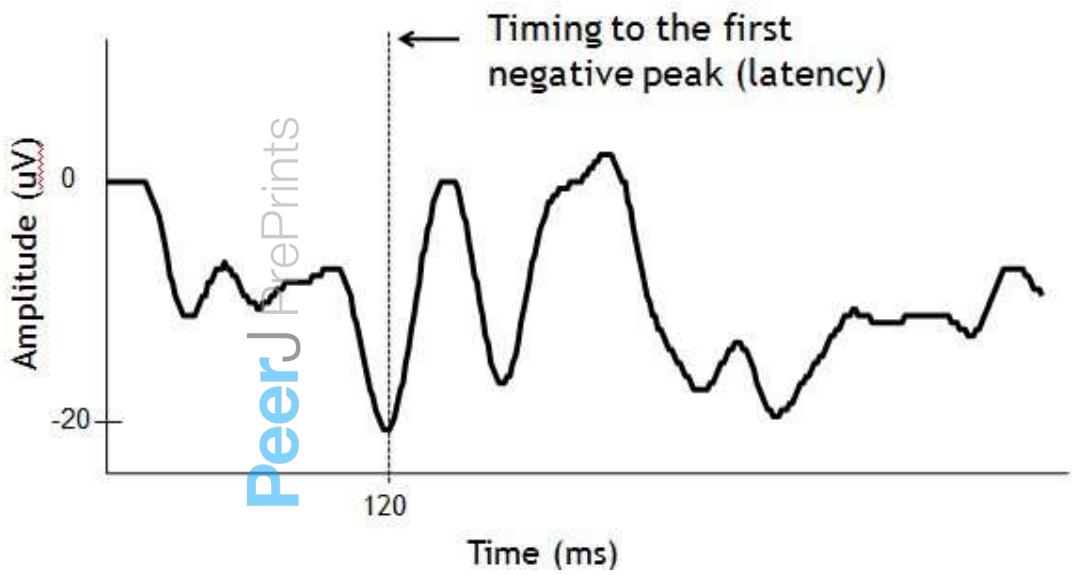
Figure 1. A color VEP data of a participant from blue-yellow (S-cone onset) stimuli.



Latency (time of response to stimulus) for S response onset is measured from pattern onset (time of stimulus presentation) to the trough of the first large negative wave. Amplitudes of the waveforms were measured from the trough of the first negative wave to the peak of the positive wave.

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Figure 2. Amplitude (uV) of the VEP response to chromatic onset stimuli (left=Blue-Yellow, right=Red-Green)

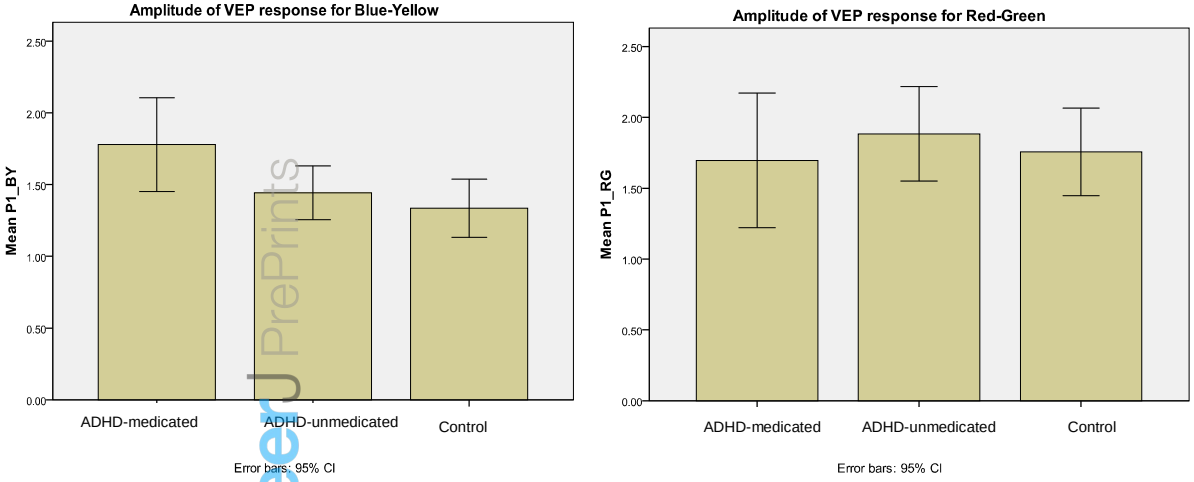

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Figure 3. Scatter plot between Inattentive symptom on SWAN and P1 amplitude (uV) on Blue-Yellow.

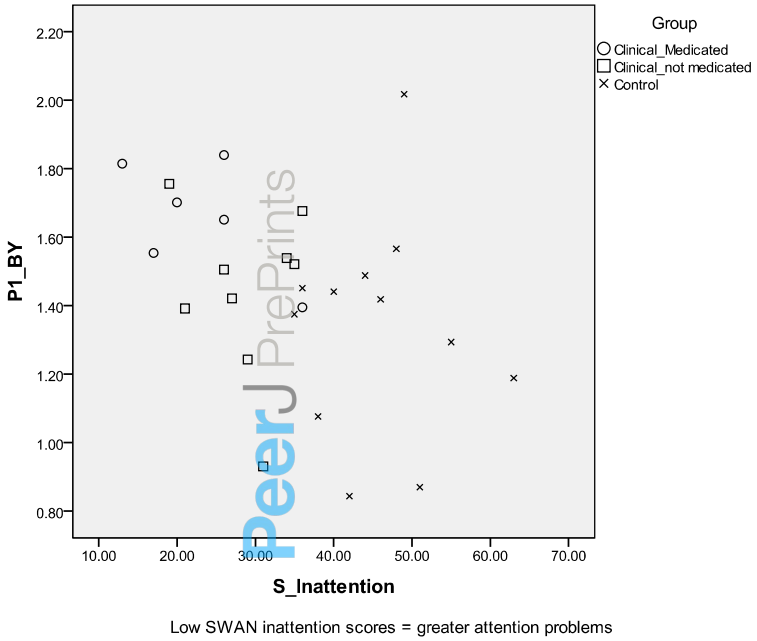

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Figure 4. Separate visual pathways for Red and Blue retinotopic information

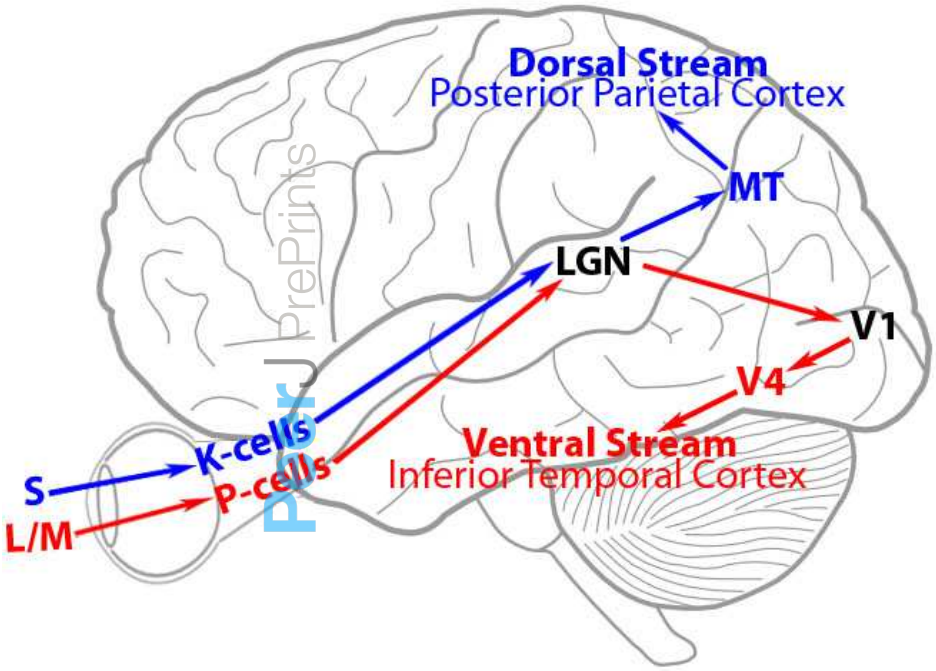

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Table 1. ADHD symptoms for ADHD and Control group

		Descriptives								Planned Orthogonal Contrast analysis	
		Medicated ADHD (N=7)		Non Medicated ADHD (N=9)		ADHD (N=16)		Controls (N=15)		Med. ADHD(N=7) vs. Non Med. ADHD(N=9	ADHD (N=16) vs. Control (N=15)
Measures		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean diff.(sig.)	Mean diff.(sig.)
SWAN-Parent	Inattention	23.00	8.15	28.67	6.02	26.40	7.26	44.27	8.07	-5.67	**-17.87*** **
	Hyperactivity	32.00	15.32	35.56	9.77	34.13	11.90	45.73	9.38	-3.56	**-11.60** **
SDQ-Teacher	Total Problem	15.14	6.04	15.22	6.08	15.19	5.86	7.33	4.02	0.08	**7.86*** **
SDQ-Parent	Total Problem	13.33	7.23	11.00	4.21	11.93	5.50	5.73	4.99	2.33	**6.2** **

***P<.001, ** P<.01

1) SWAN rating scale: The Strengths and Weaknesses of ADHD-symptoms and Normal-behavior (Swanson et al., 2005)

2) SDQ questionnaire: Strengths and Difficulties Questionnaire (Goodman,1997) 3) Med. ADHD: Medicated ADHD, 4) Non Med: Non medicated ADHD

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Table 2. Summary scores on Vision

	Descriptives								Planned Orthogonal Contrast analysis	
	Med. ADHD (N=7)		Non Med. ADHD (N=9)		ADHD (N=16)		Controls (N=12)		ADHD (N=16) Vs. Control (N=12)	Med. ADHD (N=7) Vs. Non Med. (N=8)
Measures	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean difference	Mean difference
Vision										
Contrast sensitivity(R)	1.69	.08	1.65	.09	1.67	.09	1.64	.06	.03	.04
Contrast sensitivity(L)	1.70	.10	1.66	.09	1.68	.09	1.66	.07	.03	.04
Contrast sensitivity(Bi)	1.85	.10	1.79	.11	1.82	.10	1.81	.09	.01	.06
Visual Acuity (R)	-.11	.10	-.08	.16	-.09	.13	-.04	.15	-.05	-.03
Visual Acuity (L)	-.09	.10	-.12	.09	-.11	.09	-.09	.08	-.02	-.03
Visual Acuity (Bi)	-.16	.05	-.18	.08	.07	.02	-.15	.08	-.02	.02
Spherical correction (R)	-.38	1.05	-.09	.94	-.21	.96	-.16	1.32	.05	-.29
Cylindrical correction (R)	.08	.20	.03	.28	.05	.24	-.07	1.04	-.02	.05
Spherical correction (L)	-.25	.88	-.19	1.67	-.21	1.34	-.01	1.60	-.21	-.06
Cylindrical correction (L)	.17	.26	.09	.19	.13	.21	.17	.41	-.04	.08
Colour vision										
Red Tritan (L)	1.00	.00	1.11	.33	1.06	.25	1.00	.00	.06	-.11
Green Tritan (L)	1.00	.00	1.00	.00	1.00	.00	1.07	.26	-.07	.00

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Blue Tritan (L)	1.00	.00	1.11	.33	1.06	.25	1.07	.26	-.01	-.11
Red Tritan (R)	1.14	.38	1.00	.00	1.06	.25	1.13	.35	-.05	.14
Green Tritan (R)	1.00	.00	1.00	.00	1.00	.00	1.00	.00	.00	.00
Blue Tritan (R)	1.29	.49	1.22	.44	1.25	.45	1.13	.35	.12	.07
VEP										
Blue-Yellow latency (ms)	145.54	4.66	149.56	10.20	147.80	8.27	152.42	9.22	-4.62	-4.02
Red-Green latency (ms)	140.55	9.08	143.13	6.17	142.00	7.42	141.36	6.30	.64	-2.58
Blue-Yellow Amplitude(μV)	1.78	.35	1.44	.24	1.59	.33	1.34	.32	**.25***	**.34***
Red-Green Amplitude (μV)	1.70	.45	1.88	.43	1.81	.43	1.76	.49	.05	-.18

*P<.05

1) R: Right eye only

2) L: Left eye only

3) Bi: Binocular vision

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