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Contrast Sensitivity for Motion Detection and Direction Discrimination in Adolescents with Autism Spectrum Disorders and their Siblings

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Abstract

The magnocellular (M) pathway hypothesis proposes that impaired visual motion perception observed in individuals with Autism Spectrum Disorders (ASD) might be mediated by atypical functioning of the subcortical M pathway, as this pathway provides the bulk of visual input to cortical motion detectors. To test this hypothesis, we measured luminance and chromatic contrast sensitivity, thought to tap M and Parvocellular (P) pathway processing respectively. We also tested the hypothesis that motion processing is impaired in ASD using a novel paradigm that measures motion processing while controlling for detectabilty. Specifically, this paradigm compares contrast sensitivity for detection of a moving grating with contrast sensitivity for direction-of-motion discrimination of that same moving grating. Contrast sensitivities from adolescents with ASD were compared to typically-developing adolescents, and also unaffected siblings of individuals with ASD (SIBS). The results revealed significant group differences on P, but not M, pathway processing, with SIBS showing higher chromatic contrast sensitivity than both participants with ASD and TD participants. This atypicality, unique to SIBS, suggests the possible existence of a protective factor in these individuals against developing ASD. The results also revealed impairments in motion perception in both participants with ASD and SIBS, which may be an endophenotype of ASD. This impairment may be driven by impairments in motion detectors and/or by reduced input from neural areas that project to motion detectors, the latter possibility being consistent with the notion of reduced connectivity between neural areas in ASD.

Keywords

Autism endophenotype; Motion perception; Magnocellular; Parvocellular; Visual psychophysics

Autism spectrum disorders (ASD) are pervasive developmental disorders characterized by deficits in a variety of social, communicative, and emotional behaviors (APA, 2004; Carter, Davis, Klin, & Volkmar, 2005; Hobson, 2005; Tager-Flusberg, Paul, & Lord, 2005; WHO, 1992). In addition to these primary symptoms, there is substantial evidence for atypicalities in visual (see Simmons, et al., 2009) and auditory perception (see Kellerman, Fan, &

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Gorman, 2005; Mottron, Dawson, Soulières, Hubert, & Burack, 2006). Most relevant to the current study, individuals with ASD have been shown to exhibit impairments in motion perception (see Milne, Swettenham, & Campbell, 2005). To date, there are two main hypotheses regarding the origins of this motion perception deficit. The first hypothesis is based on some evidence that the impairment may be restricted to stimuli that require motion integration, e.g., random dot kinematograms (Pellicano, Gibson, Mayberry, Durkin, & Badcock, 2005, but see Vandenbroucke, Scholte, van Engeland, Lamme, & Kemner, 2008, for opposing evidence from moving plaid stimuli), and/or higher-level computation e.g., second-order motion (Bertone, Mottron, Jelenic, & Faubert, 2003). Such findings are consistent with an "integration hypothesis", which postulates that impaired motion perception in ASD lies in impaired ability to integrate perceptual information, specifically complex perceptual information, rather than representing a deficit in motion perception per se. In support of this hypothesis, several studies have reported that integration of certain types of static elements is compromised in individuals with ASD, although this effect may be restricted to individuals with Autistic Disorder, and not Asperger's syndrome (Spencer & O'Brien, 2006; Tsermentseli, O'Brien, & Spencer, 2008, and see Del Viva, Igliozzi, Tancredi, & Brizzolara, 2006; Kemner, Lamme, Kovacs, & van Engeland, 2007, for negative findings).

An alternative hypothesis is that the deficit in motion perception reflects a true impairment in cortical motion detectors, for example, within the middle temporal area, MT (see Albright, 1993 for a review of MT), or within subcortical pathways that feed into cortical motion detectors. There are two main subcortical pathways from the retina to the cortex, the magnocellular (M) pathway and the parvocellular (P) pathway. In brief, these two pathways, which originate in the retinal ganglion cells of the retina and remain segregated through the lateral geniculate nucleus up into primary visual cortex, differ markedly in their response properties (see Dobkins & Albright, 2004; Merigan & Maunsell, 1993, for reviews). [There is also a third subcortical pathway, referred to as the koniocellular (K) pathway. A lot less is known about this pathway (see Dobkins, 2000; Hendry & Reid, 2000) and thus will not be discussed further here.] Because cortical motion detectors receive the bulk of their input from the M pathway (Maunsell, Nealey, & DePriest, 1990), it has been proposed that the motion perception impairment in ASD may originate in atypicalities within the subcortical M pathway (McCleery, Allman, Carver, & Dobkins, 2007; Milne, et al., 2002; Plaisted, Swettenham, & Rees, 1999). We refer to this hypothesis as the "M pathway hypothesis".

A number of perceptual studies have investigated the integrity of the M and P pathways by measuring performance on visual stimuli/tasks that are thought to differentially activate the M and/or P pathways. One approach has been to measure luminance contrast sensitivity of sinusoidal gratings presented at specific combinations of spatial and temporal frequencies as M neurons are more sensitive than P neurons to low spatial and high temporal frequencies, whereas P neurons are more sensitive than M neurons to high spatial and low temporal frequencies (Merigan & Maunsell, 1990, 1993; Schiller, Logothetis, & Charles, 1990). An alternative approach is to measure luminance and chromatic (red/green) contrast sensitivity, as the response properties of the M and P pathways mean that they are preferentially tuned for luminance and chromatic stimuli respectively (see Dobkins, 2009). Specifically, M neurons are more sensitive than P neurons to luminance contrast, and conversely, P neurons are more sensitive than M neurons to red/green chromatic contrast (Lee, Pokorny, Smith, Martin, & Valberg, 1990; Shapley, 1990; Smith, Pokorny, Davis, & Yeh, 1995).

It should be noted, however, that whether these approaches truly (and perfectly) isolate the M and P pathways has been called into question (see Lennie & D'Zmura, 1988; Skottun, 2000, for reviews), for two reasons. First, lesion studies have shown that, at some spatio-temporal frequencies, both M and P pathway lesions impair luminance contrast sensitivity

(Merigan & Eskin, 1986; Merigan, Katz, & Maunsell, 1991; Merigan & Maunsell, 1990; Schiller, et al., 1990). Second, there are about eight times as many P than M neurons, and thus while each individual P neuron may have lower luminance contrast sensitivity than each M neuron, probability summation across neurons may give the P pathway the upper hand on luminance contrast sensitivity. While we acknowledge that the dichotomy is not complete, it is nonetheless reasonable to assert that there exists a *bias* for the P and M pathways to contribute to Luminance and Chromatic CS, respectively. As such, atypical chromatic contrast sensitivity can be interpreted as atypical P pathway processing, whereas atypical luminance contrast sensitivity may be due to atypicalities in either the M or the P pathway.

With these caveats in mind, there have been many studies that have attempted to investigate the integrity of the M and P pathways in individuals with ASD by measuring luminance contrast sensitivity across a range of spatial and temporal frequencies. For example, one study that adopted this approach (M pathway stimulus = 0.5 cpd, 6 Hz, P pathway stimulus = 6 cpd, 1 Hz), reported no differences in contrast sensitivity between individuals with ASD and typically developing (TD) controls, on either the M or the P pathway stimulus (Bertone, Mottron, Jelenic, & Faubert, 2005, and see Pellicano, et al., 2005 for similar findings using only an M pathway stimulus). Bertone et al. (2005) also presented participants with an orientation-identification task using grating stimuli that were masked with luminance-defined noise. They reported enhanced luminance contrast sensitivity in individuals with ASD on this task. Unfortunately, the spatiotemporal frequency of these stimuli (0.75 cycles/degree, 0 Hz), and the addition of the luminance noise, make it difficult to know whether this effect was M- or P-pathway related. Another study (M pathway stimulus = 0.5 cpd, 12.5 Hz, P pathway stimulus = 13.4 cpd, 2 Hz), likewise, found no difference in contrast sensitivity between individuals with ASD and TD controls on the M pathway stimulus, however, they did find reduced contrast sensitivity for the P pathway stimulus in children and adolescents with ASD (Davis, Bockbrader, Murphy, Hetrick, & O'Donnell, 2006). In sum, contrary to the "M pathway hypothesis", the results from these studies suggest that M pathway processing is intact in ASD (but see Plaisted & Davis, 2005 for a discussion of why the size of the stimuli in these previous studies may not have been optimal for assessing M pathway processing), and that, if anything, P pathway processing may be impaired.

Luminance and chromatic contrast stimuli have yet to be employed for investigating M and P pathway processing in individuals with ASD, but we have recently used these stimuli in a forced-choice preferential looking study of 6-month-old infants who are at risk for developing ASD (McCleery, et al., 2007). These infants are referred to as "high-risk" for ASD because they are thought to carry some of the genes for ASD since they have an older sibling diagnosed with the disorder (see Zwaigenbaum, et al., 2009, for the logic behind the high-risk infant approach). In the study, we found that high-risk infants exhibited significantly higher luminance contrast sensitivity compared to "low-risk" control infants (from families without ASD history), yet typical chromatic contrast sensitivity. The luminance and chromatic stimuli were presented at a temporal frequency of 4.2 Hz, as infants were previously found to have optimal contrast sensitivity at this frequency (Dobkins, Anderson, & Lia, 1999). The luminance stimuli employed in the study were presented at a very low spatial frequency of 0.27 cpd, making luminance contrast sensitivity measured in the study to be less likely to be mediated by the P pathway than the M pathway. Additionally, the P pathway is unlikely to be implicated in the higher luminance contrast sensitivity in the high-risk infants compared to the low-risk infants, as there was no difference in chromatic contrast sensitivity between the two infant groups, suggesting typical P pathway functioning in the high-risk infants. These results therefore suggest atypical (and enhanced) M pathway functioning associated with ASD, which we suggest could have negative repercussions on the developmental of those areas of the brain that are

innervated by the M pathway (see Discussion). A difference score between luminance and chromatic contrast sensitivity was also calculated in the study, and compared between groups. The high-risk infants showed higher luminance vs. chromatic contrast sensitivity than the low-risk infants, who showed lower luminance vs. chromatic contrast sensitivity. This result suggests that there is also a significant difference in the relative functioning of the M and P pathways in the high-risk infants as compared to the low-risk infants.

We more recently reported that the severity of this atypicality is the same for the vast majority of our high-risk infants who did not go on to develop ASD as it is for the smaller percentage that did go on to develop ASD (Dobkins, Carver, Price, & Akshoomoff, 2010). Together, these findings suggest that atypical M pathway processing may be an endophenotype of ASD, i.e., it may represent a genetically-mediated risk variable that is carried in both individuals with ASD and their first degree relatives (see Gottesman & Gould, 2003; Gottesman & Sheilds, 1972; Szatmari, et al., 2007).

In sum, the results of previous contrast sensitivity studies investigating the integrity of M and P pathway processing in ASD have been somewhat mixed; studies of children/adults with ASD, which employed differing spatial/temporal frequencies, suggest no M pathway deficit (yet a possible P pathway deficit), while our previous study of high-risk infants, which employed luminance and chromatic stimuli, suggests possibly enhanced M pathway processing associated with ASD. There are at least three possible methodological reasons for the discrepancies across studies. First, the differences could be due to the different methodologies employed for distinguishing M and P pathway function (spatiotemporal frequency vs. luminance/chromatic). Second, the differences could be due to the different age groups tested (children/adults vs. infants). Third, the differences could be due to the different diagnostic status of the participants (diagnosed with ASD vs. having a sibling diagnosed with ASD).

Thus, the first goal of the current study was to control these differences across studies by obtaining luminance and chromatic contrast sensitivities in adolescents with ASD, as well as their “unaffected” adolescent siblings (SIBS) using the stimuli we employed in our previous high-risk infant study. Our decision to test SIBS was based both on wanting to compare them to our previously tested high-risk infants as well as wanting to investigate endophenotypes of ASD in adolescents, as this is becoming a strong and recognized approach to studying ASD (for example, Belmonte, Gomot, & Baron-Cohen, 2009; Bolte & Poustka, 2003; Dalton, Nacewicz, Alexander, & Davidson, 2007; Dorris, Espie, Knott, & Salt, 2004; Pellicano, 2008). Specifically, if the effects we observed in high-risk infants persist into adolescence, we hypothesized that individuals with ASD and their SIBS would exhibit higher luminance contrast sensitivity than TD controls, indicative of atypical M pathway processing. Although, at first glance, this prediction would seem to go against the bulk of data showing no differences in luminance contrast sensitivity between ASD and TD individuals, as reviewed above, one paper has shown enhanced luminance contrast sensitivity in adults with ASD (Bertone, et al., 2005), therefore we consider it important to measure luminance contrast sensitivity in a group of adolescents with ASD and also their unaffected SIBS.

The second goal of the current study was to examine decreased efficiency of motion processing in adolescents with ASD, as well as their SIBS, using a novel approach that controls for detectability. In the field of vision science, the “detection/motion” DET – MOT paradigm compares contrast sensitivity for detection of a moving grating stimulus (DET) to contrast sensitivity for direction-of-motion discrimination (MOT) of the same moving grating stimulus (Dobkins & Teller, 1996; Graham, 1989; Green, 1983; Lindsey & Teller, 1990; Palmer, Mobley, & Teller, 1993; Watson, Thompson, Murphy, & Nachmias, 1980).

Here, a DET – MOT difference score of 0 indicates that contrast levels sufficient for detection of motion are sufficient for discriminating direction of motion, which suggests efficient motion processing. By contrast, a DET – MOT difference score greater than 0 indicates that contrast levels sufficient for detection of motion are not sufficient for discriminating direction of motion. This suggests inefficient motion processing, either because motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors (see Dobkins, 2005; Dobkins & Albright, 2004, for review). This DET – MOT paradigm is especially valuable for studies of clinical populations, as one need to ensure that an impairment observed on a motion task is not simply a result of a lesser ability to detect the presence of the motion stimulus per se. The DET – MOT paradigm has the equally important benefit of determining whether an apparent lack of impairment on a motion task results from a combination of inefficient motion processing that is counteracted by an overall enhanced ability to simply detect the motion stimulus.

The DET – MOT paradigm measures direction discrimination abilities while controlling for stimulus detectability. An elevated DET – MOT difference score indicates inefficient motion processing, either because the motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors. Given that there are numerous examples within the literature of impaired motion perception in ASD, we predicted that participants with ASD would exhibit higher DET – MOT difference scores than the TD controls. If inefficient motion processing is an endophenotype of ASD then we also expected to see higher DET – MOT difference scores in the SIBS group compared to controls. As described in the Discussion, by looking to see whether group differences on this metric were greater for luminance or chromatic stimuli, we hoped to differentiate between impairments in motion detectors vs. reduced input to motion detectors.

Methods

Participants

A total of 23 adolescents with ASD, 42 typically-developing (TD) adolescents, and 13 adolescents with siblings diagnosed with ASD (SIBS) participated in the study. They were recruited from community resources in San Diego and the San Diego Unified School District. The participants with ASD were diagnosed by a licensed clinical psychologist or medical doctor not associated with this research, based on DSM-IV-TR criteria (APA, 2004), and confirmed in our laboratory using the Autism Diagnostic Observational Schedule (ADOS, see *Psychometric Assessments*, below). These participants had no known specific neurological or genetic conditions (e.g., Fragile X, Rett Syndrome) that could account for their diagnosis of ASD. Written informed consent was obtained from all participants, as well as from their parents. The study took 2–3 hours to complete, and participants were paid USD10 per hour. The procedures followed were in accordance with the ethical standards of the UC San Diego Human Research Protection Program, and the 1964 Declaration of Helsinki.

Data from three participants with ASD were excluded because they were unable to complete the visual tests (n = 2), or because their ADOS and SCQ scores fell below the cut-off for ASD (n = 1, see below). Data from a further three participants with ASD, and three TD participants were excluded because their performance IQ scores were under 85 (see below). Data from one SIBS participant were also excluded because, despite not having a formal diagnosis of ASD, he met the ADOS cut-off for ASD (see below). This resulted in a final sample of 17 participants with ASD, 39 TD participants and 12 SIBS participants (eight of whom were siblings of eight participants with ASD, and four of whom had siblings with ASD who were not participants in our study). The external diagnoses of the 17 participants

with ASD were: three with Autistic Disorder, nine with Asperger's Syndrome and five with Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). The mean ages of groups were: ASD = 15 years 0 months (s.d. = 1 year 10 months), TD = 15 years 3 months (s.d. = 1 year 2 months), SIBS = 15 years 1 month (s.d. = 1 year 9 months), and there were no significant age difference between the three groups ($p = 0.810$, one-factor ANOVA, see Table 1). All participants had normal or corrected to normal vision, and reported no color vision deficiencies. Because we employed red/green stimuli, we ensured that no participants were color deficient by testing all with the Ishihara colour deficiencies test (Ishihara, 1992). The proportion of participants who had corrected-to-normal vision in the three groups was: ASD = 35.2%; TD = 35.8%; SIBS = 50%. There were proportionally more girls in the TD group (43.5%) and the SIBS group (58.3%) than the ASD group (6%). Analyses are presented to ensure that any group differences found were not related to gender. Further participant information (including ages, gender, and assessment scores) is presented in Table 1.

Note that our justification for having more participants in the control group was to maximize the accuracy of our measures in typical individuals. Because the number of participants in each of the three groups was different, we conducted additional between-group posthoc analyses that equated the number of participants (17 in each group, for comparisons between the TD participants and participants with ASD, and 12 in each group for comparisons between the TD and SIBS participants). The results for ASD-TD comparisons with N=17 were the same as those reported below. The results for SIBS-TD comparisons with N=12 showed trends in the same direction ($p < 0.1$) as those reported below (the weaker effects presumably due to lower power). We thus present the results with data from all participants included[1].

Psychometric Assessments

For each participant, two psychometric assessments were conducted. i) The Lifetime version of the Social Communication Questionnaire (SCQ), which consists of 40 'Yes/No' questions asking parents if their child currently displays specific autism-related behaviors or whether those behaviors were present between the ages of four to five years (Rutter, Bailey, Lord, & Berument, 2003). The SCQ cut-off score for ASD is 15. ii) The Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999), which is an experimenter administered (by author HCK or by a trained research associate) test that measures cognitive abilities. It is comprised of four standardized sub-tests that assess expressive language, perceptual organization, abstract verbal reasoning and nonverbal fluid reasoning abilities. The two verbal sub-test scores can be converted into a "verbal IQ" score, and the two non-verbal sub-test scores can be converted into a "performance IQ" score. The four sub-tests when considered together yield a "full scale IQ" that provides a composite measure of the participant's intelligence.

In addition, the Autism Diagnostic Observation Schedule (ADOS) (Modules Three or Four) was administered with each ASD and SIBS participant by author HCK. The ADOS is a play-based experimenter-administrated assessment designed to elicit behaviors (or lack of behaviors) associated with a diagnosis of ASD (Lord, et al., 2000). The assessment was conducted to verify the ASD diagnoses for the participants with ASD and to confirm that the SIBS participants did not meet the criteria for ASD. The ADOS cut-off score for "ASD" is seven, and for "Autistic Disorder" is ten. (Note that the ADOS does not distinguish Asperger's Syndrome, and that the specific diagnosis determined from the ADOS does not always conform to that of the external diagnosis, see Risi, et al., 2006). By this criterion, in

[1]In these additional equal sample size analyses we matched the groups on gender and number of participants with corrected-to-normal vision. Gender and visual issues were therefore unlikely to contribute to the between-group differences found. This issue is also considered in the Results section.

the “ASD group” (the term we use throughout the paper to refer to participants who had an outside diagnosis of Autistic Disorder, Asperger’s Syndrome or PDD-NOS), nine participants were classified as having ASD and seven as having Autistic Disorder. One participant with an external diagnosis of Asperger’s Syndrome fell below the ASD cut-off on the ADOS. However he scored above the SCQ cut-off for ASD, therefore his data were included in the ASD group for our analyses. Additional analyses confirmed that results were identical with this ASD participant removed. All SIBS participants who were included in the sample scored below the ASD cut-offs on the ADOS and the SCQ.

As shown in Table 1, the results of one-way ANOVAs revealed significant group differences on the SCQ ($p < 0.001$). As expected, this is driven by group differences between participants with ASD and TD participants (2-tailed t-test, $p < 0.001$) and between participants with ASD and SIBS participants (2-tailed t-test, $p < 0.001$). There were no group differences in verbal IQ, performance IQ, or full-scale IQ (see Table 1), however the participants with ASD had lower (although not significantly so) verbal IQs than the other two groups. This pattern of results is consistent with previous literature reporting that individuals with ASD are known to have lower verbal IQ compared to their performance IQ (Siegal, Minshew & Goldstein, 1996; Rumsey, 1992; Yirmiya & Sigman, 1991). Given that the requirements of the experiments were to press a button in response to a visual stimulus, we prioritized matching performance IQ over and above verbal IQ. In order to establish whether the, albeit non-significantly, lower verbal IQ in the ASD group may confound any reported results we assessed the relationship between the dependent variables in our study i.e., contrast sensitivity values for the DET and MOT conditions with luminance and chromatic stimuli (see below), and both verbal and performance IQ. None of the variables correlated with verbal IQ: DET_{lum}, $r_s = 0.13$; $p>0.1$; DET_{chr}, $r_s = -0.01$, $p> .1$; MOT_{lum}, $r_s = 0.19$, $p >0.1$ and MOT_{chr}, $r_s = 0.12$, $p>0.1$. By contrast, all of the variables correlated with performance IQ: DET_{lum}, $r_s = 0.38$, $p<.01$; DET_{chr}, $r_s = 0.31$, $p<0.01$; MOT_{lum}, $r_s = 0.25$, $p<0.05$ and MOT_{chr}, $r_s = 0.24$, $p<0.05$.

Visual Apparatus

Visual stimuli were generated using the Cambridge Research System (CRS) toolbox for MATLAB. They were presented on a on a high resolution RGB monitor (19.8” SONY GDM-F520 monitor, 100Hz frame rate, 1024×768 pixels at dot pitch of 0.22mm), driven by a CRS VSG VSG2/3F digital video board in a Microsoft Windows XP computer with Intel Pentium 4 processor. The 14-bit video board allowed for 16,384 discrete luminance and chromatic levels. Gamma correction was performed to linearize the voltage/luminance relationship for the monitor display, using a PR-650 SpectraColorimeter (Photoresearch). At a viewing distance of 50 cm, the viewable portion of the monitor subtended 40.5 × 30.9 degrees of visual angle.

Stimuli

The stimuli in these experiments were luminance (light/dark) and chromatic (isoluminant, red/green) horizontally-oriented, moving sinusoidal gratings presented on a background with the same luminance (23 cd/m2) chromaticity (CIE = 0.489, 0.453). All gratings subtended 2.0 × 2.0° of visual angle, and were presented at the center of gaze at a spatial frequency of 1.0 cycles/degree and a temporal frequency of 5.5 Hz. These stimulus parameters were selected to: 1) minimize chromatic aberration and possible spatial inhomogeneity of the display (Lindsey & Teller, 1990), 2) optimize stimulus size for assessing M pathway processing (Plaisted & Davis, 2005) and 3) replicate parameters from previous studies in adults that yielded the expected large difference between chromatic MOT and chromatic DET sensitivity (Palmer, et al., 1993). Contrast of stimuli is described in terms of cone contrast, i.e., the amount of response modulation produced in the long-wavelength-selective

(L) and medium-wavelength-selective (M) cones in the eye (see Dobkins, Anderson, & Lia, 1999; Gunther & Dobkins, 2002, for methodological details). Zero percent contrast refers to a uniform field, which is indistinguishable from the background.

Visual Paradigm

Participants were tested in a dark room and viewed the video monitor binocularly from a chin rest situated 50 cm away. Participants were instructed to maintain fixation on a small cross (length and width = 0.2 degrees) in the center of the monitor. Fixation was not monitored because the stimuli were centrally located, thus there was no reason for participants to move their eyes i.e. break fixation to detect/discriminate the stimulus. Before beginning the main experiment, red/green isoluminance was determined for each participant using standard motion photometry (Dobkins & Teller, 1996; Lindsey & Teller, 1990). The stimulus conditions for the motion photometry procedure were identical to those employed in the main experiments (i.e., same size, orientation, spatiotemporal frequency, location). On each motion photometry trial, participants maintained fixation on the small cross in the centre of the monitor and began each trial with a key press, after which a moving red/green grating was presented at the center of gaze. Participants adjusted the luminance contrast in the grating until the percept of motion was least salient. Each participant's isoluminance point was determined from the mean of 10 trials. Note that there were 10 cases (ASD = 3; TD = 3; SIB = 4) where a participant's settings were deemed unreliable because their settings were too variable. Here, we used the mean isoluminance point obtained from college students at UC San Diego (mean age = 21 years), which we considered to be a close enough approximation since isoluminance points do not vary largely across individuals at the spatiotemporal frequency employed in the current study (Dobkins, Gunther, & Peterzell, 2000). This was confirmed after the data had been collected as there was no relationship between isoluminance point and age in our participants ($r (N = 58) = -0.2$, $p>0.1$).

The isoluminance points ranged between −8.0% to 4.7% luminance contrast and there was not a significant difference between the isoluminance points of the three groups ($F(2,57) = 1.6$, $p=0.2$).

There were two main experiments in this study, the "detection (DET) task", and the "motion (MOT) task". These tasks employed the same moving stimuli, but required different responses from the participants: in the DET task, participants were required to detect the presence of the stimulus; in the MOT task, participants were required to indicate the direction of the stimulus. In both the DET and MOT tasks, participants maintained fixation of the small cross in the center of the monitor and began each trial with a key press. In the DET task, a moving grating (either luminance or chromatic, moving either upward or downward) appeared at the centre of gaze in one of two 250 ms intervals, separated by a 500 ms gap, with the beginning of each of the two time intervals accompanied by a beep. After each trial, participants reported via key presses whether the visual stimulus appeared during the first or second interval, i.e., in a standard two-alternative forced choice manner. It is important to note that while the grating moved either upward or downward (randomly across trials), direction was irrelevant to the participant's task—they were only instructed to detect its presence. In the MOT task, a moving grating (either luminance or chromatic, moving either upward or downward) appeared at the centre of gaze for 250 ms, with the beginning of this single interval accompanied by a beep. After each trial, participants reported via key presses whether the stimulus moved upward or downward, i.e., again in a standard two-alternative forced choice manner. For both DET and MOT, feedback was provided in the form of a beep (different pitch from the beeps that sounded during stimulus presentation) indicating a correct response.

The task order i.e. whether the DET or the MOT task was performed first, was counterbalanced across participants. Within each task, luminance (LUM) and chromatic (CHR) gratings were randomized across trials (120 trials of each stimulus type). Motion direction was also randomized across trials within a block (120 trials of each motion direction). The total number of trials obtained from each participant was 480, 120 for each of the 4 conditions: 2 task types (DET and MOT) × 2 grating types (LUM and CHR).

Adaptive Staircase Procedure

Within each block of trials, the contrast of luminance and chromatic gratings varied across trials in an adaptive staircase procedure. Specifically, on the first trial a luminance or chromatic stimulus was presented, its contrast was 85%. The contrast for subsequent trials of each of grating type varied in a 1 down/2 up procedure, based on the PEST method (see Taylor & Creelman, 1967). Contrast was decreased by one step size after a correct response, and was increased by two step sizes after an incorrect response. The maximum step size was 0.14 log units (1.38-fold change in contrast). The value of the step size was determined by an acceleration factor of 1.2 and a reversal factor of power of 1.1. Following either two correct or two incorrect responses, the step size was multiplied by the acceleration factor. Following a reversal in correctness, the step size was multiplied by (1/acceleration factor)^reversal power.

Obtaining Contrast Sensitivity

For each participant, a contrast threshold was determined for each of four conditions: two stimulus types (LUM and CHR) and two task types (DET and MOT), based on 120 trials each. Threshold was calculated by fitting a psychometric Gumbel function (Gumbel, 1958) to “percent correct versus contrast” data, using maximum likelihood method (Johnson, Kotz, & Balakrisnan, 1995; Watson, 1979). Contrast sensitivity was then calculated as the inverse of cone contrast threshold. Contrast sensitivity was logged since logarithmic, but not linear, contrast sensitivity data typically conform to normal distributions (see Gunther & Dobkins, 2002), and because when plotted in log, a fixed vertical distance between two sensitivity values represents a fixed relative sensitivity between the two.

Composite Indices and Data Analyses

In the current data set, there were no outliers, defined as participants whose values were +/− 3 standard deviations from the group mean. However, the logged contrast sensitivity values violated assumptions of normality (DET_{lum} Shapiro-Wilk test : $p<0.05$; $Z_{skew} = -0.40$, $Z_{kurtosis} = 1.053$; DET_{chrom}, Shapiro-Wilk test: $p<0.01$; $Z_{skew} = -2.05$, $Z_{kurtosis} = 1.1$; MOT_{lum} Shapiro-Wilk test: $p<0.05$; $Z_{skew} = -1.37$, $Z_{kurtosis} = 1.33$; MOT_{chrom} Shapiro-Wilk test: $p<0.01$; $Z_{skew} = -0.65$, $Z_{kurtosis} = 1.6$) and assumptions of homogeneity of variance (Levene’s test: $F(2,65)<3.42$, $p>0.039$). This was also true for linear values. Under these conditions, it is still traditional to use logged values, however, non-parametric statistics had to be employed to compare differences in contrast sensitivity across participant groups. And, Figures illustrating the average group sensitivity data are presented using median values.

There are no non-parametric statistics that test for interactions, thus to investigate our questions of interest, we instead computed “composite indices” from the logged contrast sensitivity values, which enabled us to test the questions outlined below with non-parametric statistics: The first index was a “LUM – CHR DET difference score”, i.e., difference in logged contrast sensitivity between LUM and CHR stimuli for the DET task (i.e., $DET_{lum} - DET_{chr}$), which was computed to determine if participants with ASD (or SIBS) show atypical relative sensitivity to luminance vs. chromatic contrast, which we use as a proxy for atypical sensitivity of M vs. P pathways. Note that we used just the DET, and not the MOT,

data for this analysis, because DET sensitivity is a more pure metric of "detectability", as it requires only detection, not directional discrimination, of a moving grating. Also note that the actual value of this LUM – CHR DET score has no particular meaning (because it varies with spatial and temporal frequency, see Dobkins, et al., 2000). Thus, the most fundamental outcome measurement is the comparison of LUM – CHR DET difference scores across participant groups, and not the absolute value of LUM – CHR DET difference scores per se.

The second index was a "DET – MOT difference score", i.e., difference in logged contrast sensitivity between DET and MOT tasks (i.e., DET – MOT), which was computed to determine if participants with ASD (or SIBS) show inefficient motion processing, either because motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors. This was computed separately for luminance and chromatic stimuli. Values above 0 indicate higher sensitivity for DET than MOT. And, the higher the value, the less efficient is motion processing. In theory, the DET – MOT difference score should not assume a negative value (i.e., higher sensitivity for MOT than DET), as this would indicate that a participant was able to discriminate the direction of a stimulus at a contrast lower than that required to simply detect its presence! However, in practice, it is possible to obtain a negative difference score for one of several reasons, including: i) inherent differences in task difficulty, specifically, the DET task (which has two intervals) might be harder than the MOT task (which has a single interval), ii) Although the order of block type (DET vs. MOT) was randomized and counterbalanced across participants, within a participant, order effects could contribute to differences in MOT vs. DET sensitivity, and/or iii) noise in the sensitivity estimate(s). Any or all of these factors could bias the DET – MOT difference score. It is for this reason that the most fundamental outcome measurement is the comparison of DET – MOT difference scores across participant groups, and not the absolute value of DET – MOT difference scores per se. For example, if one participant group exhibits relatively higher DET – MOT difference scores than the other groups, this is consistent with that group possessing relatively less efficient motion processing, either because motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors.

All indices were checked for assumptions of normality and homogeneity of variance, and assumptions of normality were not met for two of the indices, the luminance and the chromatic DET – MOT difference score (Kolmogorov-Smirnov test: $p>0.005$). However, as non-parametric statistics make no assumptions of the distribution of the data and because they are more conservative than parametric statistics (Field, 2005), our use of non-parametric statistics for all analyses was justified.

Results

Figure 1 plots group log contrast sensitivity data for the three different participant groups (ASD: white, TD: grey and SIBS: patterned), separately for detection of luminance gratings (DET_{lum}), detection of chromatic gratings (DET_{chr}), direction-of-motion discrimination of luminance gratings (MOT_{lum}) and direction-of-motion discrimination of chromatic gratings (MOT_{chr}). These contrast sensitivities were used to compute composite indices that addressed two questions, which are addressed in turn, below.

Question 1. Is ASD associated with atypicalities in the relative integrity of M vs. P pathway processing?

As described in Methods, to address this question, we computed a "LUM – CHR DET difference score" i.e., difference in logged contrast sensitivity between LUM and CHR stimuli for the DET task (i.e., $DET_{lum} – DET_{chr}$). Figure 2 plots group LUM – CHR DET difference score for the three different participant groups (ASD, TD and SIBS). The results

of a Kruskal-Wallis test revealed a significant group difference ($H(2)=8.58$, p=0.014). Further posthoc investigation, utilizing Mann-Whitney *U* tests, revealed that the group difference was driven by a smaller LUM - CHR DET difference score in the SIBS participants as compared to both the participants with ASD ($U=49.0$, p=0.018, r=0.44) and the TD participants ($U=105.0$, p=0.003, r=0.40), yet there were no differences between participants with ASD and TD participants ($U=327.0$, p=0.944, r=0.01).

To determine whether the group difference in LUM – CHR DET difference scores was driven by differences in DET_{lum} or DET_{chr} sensitivities (or both), we conducted additional posthoc analyses. Mann-Whitney *U* tests revealed no group difference in DET_{lum} sensitivity (all p values > 0.296), yet higher DET_{chr} sensitivity in SIBS participants as compared to both the participants with ASD ($U=54.0$, p=0.033, r=0.39) and the TD participants ($U=113$, p=0.006, r=0.38). This group difference in DET_{chr}, but not DET_{lum}, sensitivity can be observed in Figure 1. These findings suggest relatively enhanced chromatic contrast sensitivity, and, by proxy, enhanced P pathway processing in SIBS, but not participants with ASD. This finding in SIBS is opposite to that observed in our study of 6-month-old "high-risk" infants (who are also SIBS); high-risk infants exhibit enhanced luminance, but not chromatic, sensitivity. In the Discussion, we address the discrepancy between our previous infant and current adolescent results, and propose that the atypicality in adolescent SIBS might be a marker of a protective factor in individuals who have a genetic predisposition for ASD, but who do not develop behaviors associated with ASD sufficient for a diagnosis.

The smaller LUM – CHR DET difference score in SIBS participants compared to the TD participants is unlikely to be driven by gender effects as the number of males and females in the TD and SIBS groups were very similar. Furthermore, the male and female LUM – CHR DET difference scores were not different in either the TD or the SIBS groups (TD: $U=149$, p=0.292, r=0.17, SIBS: $U=16.0$, p=0.876, r=0.07). Similarly, to ensure that this was not a confound of the fact that a slightly higher percentage of the SIBS group were wearing glasses compared with the other two groups, we compared the LUM – CHR DET difference score of those participants who were wearing glasses and those who were not, and found no significant differences either at a total group level ($U=427$, p=0.176, r=0.17), or within the three experimental groups (ASD: $U=29.0$, p=0.733, r=0.10; TD: $U=124$, p=0.141, r=24, SIBS: $U= 15.0$, $p<1.000$, $r<0.001$).

Question 2. Is ASD associated with less efficient motion processing, either because motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors?

To address this question, we used the "DET – MOT difference score", i.e., difference in logged contrast sensitivity between DET and MOT tasks (i.e., DET – MOT). As described in Methods, difference scores above 0 indicate lower sensitivity for MOT than DET, and the higher the value, the less efficient motion processing, either because motion detectors are themselves impaired and/or because of reduced input from neural areas that project to motion detectors. We present luminance and chromatic DET – MOT difference scores separately because a related samples difference test, i.e., Wilcoxon signed rank test, revealed a significant difference between luminance and chromatic stimuli ($T=619.0$, $p<0.001$, r=0.29). Specifically, CHR DET – MOT difference scores were higher than LUM DET – MOT difference scores, which is in line with results from previous studies and is thought to reflect the fact that neural areas underlying chromatic sensitivity, i.e., the P pathway, provide weaker input to motion detectors than neural areas underlying luminance sensitivity, i.e., the M pathway, (see Dobkins, 2005; Dobkins & Albright, 2004 for review). For the LUM condition, the results of a Kruskal-Wallis test revealed a significant group difference in the DET – MOT difference score ($H(2)=8.46$, p=0.015). Further posthoc investigation, utilizing Mann-Whitney *U* tests, revealed that the group difference was driven by higher

LUM DET – MOT difference scores for ASD and SIBS participants, as compared to the TD participants (ASD: U=193.0, p=0.013, r=0.33, SIBS: U=138.0, p=0.033, r=0.30), which indicates relatively weaker luminance input to motion processing in the ASD and SIBS groups. There were no significant differences between ASD and SIBS participants (U=99.0, p=0.913, r=0.01). Note that a similar trend was observed for the CHR condition (H(2)=2.22, p=0.330), i.e., the CHR DET – MOT difference scores for ASD and SIBS participants were higher than those of TD participants, as can be seen in Figure 3. We believe that this effect did not reach significance because of the large variability in the CHR condition, the explanation for which we return to in the Discussion.

To determine whether the group difference in LUM DET – MOT difference scores was driven by differences in DET_{lum} or MOT_{lum} sensitivity (or both), we conducted additional posthoc analyses. We already know from the analyses above (question 1) that there were no group differences in DET_{lum} sensitivity (all p values > 0.296). By contrast, for MOT_{lum} sensitivity, Mann-Whitney U posthoc tests did reveal significant group differences. Specifically, participants with ASD exhibited significantly lower MOT_{lum} sensitivity than TD participants (U=134.0, p<0.001, r=0.47), although SIBS did not differ significantly from either of the other two groups (p>0.118 in both comparisons). These group differences can be observed in Figure 1. As we mention in the Methods, and return to in the Discussion, the more important metric is the DET – MOT difference score. Thus, the fact that both participants with ASD and SIBS participants exhibited elevated luminance DET – MOT difference scores indicates that decreased efficiency of motion processing may be an endophenotype of ASD. We return to what drives the decreased efficiency in the Discussion.

Discussion

In this study we measured luminance and chromatic contrast sensitivity both for detecting moving gratings and discriminating the direction of moving gratings in order to investigate: 1) the relative integrity of the M and P pathways, and 2) efficiency of motion processing, in three different groups: participants with ASD, unaffected siblings of individuals with ASD (SIBS) and a TD matched control group. With regard to (1), we found group differences on relative sensitivity to the P pathway (chromatic) compared to the M pathway (luminance) stimulus, with SIBS showing relatively higher chromatic vs. luminance contrast sensitivity than both participants with ASD and TD participants. This atypicality, unique to SIBS, suggests the possible existence of a protective factor in these individuals against developing ASD. With regard to (2), we found evidence for inefficient motion processing for luminance stimuli in both participants with ASD and SIBS participants, which suggests that the effect may be an endophenotype in ASD (i.e., a genetically-mediated risk factor seen in individuals with ASD and their first-degree relatives). We rule out reduced motivation or inattention as an explanation of these findings, as our study was designed such that the luminance and chromatic stimuli were randomly presented within tasks. If one group of participants were less, or in the case of the SIBS participants, more, motivated than the other, then we could expect to see decreased, or increased, performance across all four visual measures in that group, however this was not the case in our data: where group differences occurred, they were associated with specific visual measures. Furthermore, our use of composite indices, where contrast sensitivity in one condition was compared across groups relative to contrast sensitivity in another condition, negates any influence of inter-participant variability. Below, we discuss further, each of our main findings, and end with a discussion of endophenotypes in ASD.

Magnocellular (M) and Parvocellular (P) pathway Processing in ASD and First-Degree Relatives

As discussed in the Introduction, it has been suggested that impairment in motion perception in ASD could result from compromised integrity of the subcortical M pathway, which provides the bulk of visual input to cortical motion detectors, referred to as the "M pathway hypothesis". Commensurate with most previous studies (see Introduction), we found no evidence for this hypothesis; there were no group differences in luminance contrast sensitivity (DET_{lum}). Although using luminance contrast sensitivity as a direct measure of the M-pathway may be controversial (as noted in the Introduction), the fact that we did not find any deficit in either luminance or chromatic contrast sensitivity in the ASD group suggests that the basic integrity of both the M and P pathway is intact. Another recent study from our laboratory showed no differences in luminance contrast sensitivity for static stimuli (across a range of spatial frequencies) between adolescents with ASD and typically developing controls (Koh, Milne, & Dobkins, 2010). Together, these null results suggest that impaired motion perception observed in children/adults with ASD is unlikely to be a simple result of atypical luminance contrast sensitivity (mediated by either the M or the P pathway).

Despite finding no group differences in DET_{lum}, we did find group differences in relative luminance vs. chromatic contrast sensitivity (i.e. the LUM – CHR DET difference score), which was driven by significantly higher DET_{chr} sensitivity in SIBS participants as compared to both the participants with ASD and the TD participants. Given that the P pathway likely mediates chromatic contrast sensitivity, this result suggests enhanced P pathway processing in SIBS.

Interestingly, this pattern of results differs from what we found in our previous studies of High-Risk infants at 6 months. These High-Risk infants, who are by definition younger SIBS of individuals with ASD, are thought to carry some of the genes for ASD and thus atypicalities that are seen in this High-Risk cohort are considered a potential endophenotype for the disorder. We found that High-Risk infants exhibited atypically high luminance contrast sensitivity, yet typical chromatic contrast sensitivity (McCleery, et al., 2007). These results were found both in High-Risk infants who did vs. did not go on to develop ASD (Dobkins, et al., 2010). Thus, the current results differ in two ways from the previous results. First, whereas infant SIBS appear to have elevated luminance contrast sensitivity at 6 months regardless of whether they develop ASD (Dobkins, et al., 2010; McCleery, et al., 2007), this effect is no longer apparent in participants with ASD or SIBS participants by adolescence (current study). Second, whereas infant SIBS appear to have typical chromatic contrast sensitivity at 6 months (Dobkins, et al., 2010; McCleery, et al., 2007), by adolescence, only SIBS, and not participants with ASD, appear to have elevated chromatic contrast sensitivity (current study).

With regard to luminance contrast sensitivity, these age-related changes could be explained by proposing that the early atypicality in luminance contrast sensitivity in High-Risk infants is a risk factor (i.e., endophenotype) for developing ASD. The fact that the endophenotype is no longer apparent by adolescence suggests that only the early part of the developmental trajectory for luminance contrast (M pathway) sensitivity is atypical (and presumably, atypically fast, since luminance contrast sensitivity is enhanced at 6 months). This early atypicality could have negative repercussions for other aspects of brain development that interact with the M pathway. A remarkably similar phenomenon is seen for development of brain size in ASD, which exhibits an atypically fast early trajectory and then appears typical by adolescence (see meta-analysis of Redcay & Courchesne, 2005; Zwaigenbaum, Stone, & Dobkins, 2008, for similar results in High-Risk infants regardless of their outcome). We return to a discussion of endophenotypes in ASD at the end of the Discussion.

With regard to relative luminance vs. chromatic contrast sensitivity, the age-related changes could be explained by proposing that the atypically enhanced chromatic vs. luminance contrast sensitivity in adolescent SIBS is a marker of a protective factor in individuals who have a genetic predisposition for ASD. This protective factor presumably comes on line later in development and lessens the likelihood of developing behaviors associated with ASD sufficient for a diagnosis. More specifically, in the current study, the atypically high chromatic vs. luminance contrast sensitivity suggests that there may be enhanced development of the P pathway later in life as a neural attempt to compete with and/or override the enhanced development of the M pathway earlier in life. Interestingly, recent results from our developmental studies in typical infants suggest that M pathway development is more tied to preprogrammed mechanisms, whereas P pathway development is more influenced by experience (Bosworth & Dobkins, 2009; Dobkins, Bosworth, & McCleery, 2009). If true, it may be that effects of experience on P pathway development may be acting as the protective factor in SIBS who carry some of the genes of ASD but do not go on to develop ASD.

In a similar vein, a recent paper has reported another potential protective factor in SIBS, related to visual attention. Specifically, this study showed that both participants with ASD and SIBS show impaired performance, compared to TD controls, on a task that requires spatially-divided attention. Concurrently measured BOLD activity indicated that although the time-course of fronto-cerebellar activation was delayed (relative to TD participants) in both participants with ASD and SIBS, the magnitude of the delayed activation was greater in SIBS participants than in TD controls and participants with ASD (Belmonte, et al., 2009). The authors proposed that stronger activation in brain regions associated with attention may indicate more efficient compensatory processes that are activated in SIBS participants than participants with ASD, providing evidence for the existence of protective factors in SIBS against developing ASD. In sum, both studies (Belmonte, et al., 2009 and the current study) have found evidence of some factor being greater / stronger in SIBS than in either ASD or TD participants. Future studies tracking the developmental time course of these factors (with relation to the onset, or lack of onset of, ASD) will be needed to provide further insight into their potential protective effects.

Decreased Efficiency of Motion Processing in ASD and First-Degree Relatives

As discussed in the Introduction, there have been several studies reporting impaired motion perception in ASD, with some evidence to suggest that the impairment may be specific to complex motion or motion that requires integration of local motion signals. With regard to whether impairments are seen for simple (local motion) motion tasks, results have been mixed. Using the same MOT_{lum} metric of the current study, i.e., contrast sensitivity for discerning the motion direction of luminance gratings, one previous study reported reduced sensitivity in adolescents with ASD, although this was found only for those who also had a history of language delay (Takarae, Luna, Minshew, & Sweeney, 2008). By contrast, another study using the MOT_{lum} metric reported typical performance in adolescents with ASD (Bertone, et al., 2003, no information concerning language development was obtained for this sample). In line with Takarae et al., the current study revealed significantly reduced MOT_{lum} sensitivity in ASD participants, suggesting that local motion perception is impaired in ASD. As discussed in the Methods, there is a potential problem with only measuring MOT_{lum} (that could account for differences observed across studies), which is that performance on this task will be affected by how detectable the stimuli are outside the domain of motion processing. As such, an apparent impairment observed on the MOT_{lum} task could be a simple result of a lesser ability to detect the presence of the motion stimulus per se. And, in fact, such a result would be consistent with the “M pathway hypothesis”. That is, if luminance (M pathway) contrast sensitivity is reduced, so will be sensitivity on

the MOT_{lum} task. Conversely, an apparent lack of impairment on the MOT_{lum} task could result from a combination of less efficient motion processing counteracted by an overall enhanced ability to simply detect the motion stimulus. It is for this reason that the current study employed the DET – MOT paradigm, which measures direction discrimination abilities while controlling for stimulus detectability. Using this paradigm, we observed significantly higher DET – MOT difference scores in participants with ASD and SIBS participants, compared to TD controls, indicating that there is a true inefficiency in motion processing in individuals with ASD and SIBS. Notably, we observed this impairment despite the fact that our motion task was a "local" task, not requiring integration.

Note that DET – MOT difference scores in the ASD and SIBS group were statistically higher only in the luminance condition, suggesting that cortical motion detectors are not impaired in ASD, and implying that the root of the effect must be at the level of the input. However, there was a trend for elevated DET – MOT difference scores in the ASD and SIBS groups in the chromatic condition also. It is possible that the data were too noisy to pull out a significant effect in this condition. Given the high degree of variability in the chromatic DET – MOT difference scores (see Figure 3) we urge caution in our conclusion that this effect is restricted to the luminance condition, and highlight that we have not conclusively ruled out the possibility that cortical motion detectors are impaired in ASD. Because we were concerned about whether using a mean isoluminance point rather than an individual's measured isoluminance point may have contribute to the variability in the chromatic DET – MOT differences scores, we plotted the data so that the individual chromatic DET – MOT differences scores in those participants for whom a mean isoluminance point was used could be compared with those participants whose parameters were set to their own, measured, isolumiannce point. There was no indication that using a mean isoluminance point contributed to the variability in the data.

In any event, because the elevated DET – MOT difference scores were seen in both participants with ASD and SIBS, they suggest that reduced efficiency of motion processing is an endophenotype in ASD. Further studies will be required to determine the level of the impairment. At the very least, the fact that luminance detection sensitivity (DET_{lum}) appears typical in the ASD/SIBS participants of the current studies demonstrates that the reduced efficiency is not at the level of the integrity of M pathway processing itself. This leaves open the possibility that the reduced efficiency reflects impairment of motion detectors or reduced input to motion detectors. The latter could occur at any point in the motion hierarchy, for example, within subcortical areas and directional mechanisms in primary visual cortex, or between primary visual cortex and area MT. A recent study has reported no difference in functional connectivity between primary visual cortex and area MT in individuals with ASD and TD controls (Brieber, et al., 2010). This suggests that if our data do represent reduced input to motion detectors, then this is more likely to be from subcortical areas to directional mechanisms in primary visual cortex, than from primary visual cortex to area MT. Interestingly, the possibility of reduced input is in line with several lines of research suggesting decreased functional connectivity between neural areas in ASD (e.g., Belmonte, et al., 2004; Just, Cherkassky, Keller, Kana, & Minshew, 2007; Müller, 2007; Rippon, Brock, Brown, & Boucher, 2007). If the current data do reflect perceptual evidence for decreased connectivity in ASD, this would predict decreased connectivity in SIBS revealed with neural methods. This prediction receives some support from existing data, as a study have reported that the phase consistency of 40Hz (gamma-band) activity elicited by auditory stimuli, in both those with ASD and parents of someone with ASD is lower when compared with control participants (Rojas, Maharajh, Teale, & Rogers, 2008). This may suggest deficits in synchronizing neural oscillations either within or between neural assemblies, in those with ASD and their first degree relatives.

Endophenotypes in ASD

In both the current study of adolescents and our previous studies of High-Risk infants (Dobkins, et al., 2010; McCleery, et al., 2007), we have found atypicalities (compared to typically developing controls) in both individuals with ASD and their siblings. These atypicalities may reflect a genetic predisposition that runs in families of ASD, i.e., an endophenotype. Note that while an endophenotype is considered a genetically-mediated risk factor for a disorder, by definition, its presence alone is not thought to correlate with the presence of the disorder. With this in mind, there are several ways that an endophenotype could be associated with development of ASD. First, it may be that an endophenotype is more severe in individuals with ASD than in family members without ASD. This notion is consistent with the report of milder versions of the hallmarks of ASD in family members, referred to as the "broader autism phenotype" (see Bailey, et al., 1995; Bailey, Palferman, Heavey, & Le Couteur, 1998; Pickles, et al., 2000; Piven, et al., 1997). However, in the current study of adolescents, and in our previous studies of infants, this was not the case, i.e., the severity of the atypicality was the same for family members with vs. without ASD. Second, the severity of an endophenotype may be similar between individuals with ASD and their family members without ASD (as reported in the current study), but what leads to the development of ASD is an inability to compensate for the endophenotype (often referred to as "lack of resilience" in the developmental disorder literature, see Curtis & Cicchetti, 2003; Kim-Cohen, 2007 for reviews). This lack of compensation could be in the form of being deficient in some critical biological protection factor (e.g., hormones or a particular gene) or an inability to compensate at the behavioral level (e.g., because of a personality trait or temperament). Or, in the case of the current study, we suggest that the protective factor may be related to chromatic (P pathway) development. Further alternative explanations are that developing ASD may result from being exposed to an environmental trigger, or that developing ASD may result from possessing a critical number of different endophenotypes, even if each on its own is mild.

Conclusion

The current study set out to investigate 1) the M pathway hypothesis in ASD, by measuring contrast sensitivity to M and P pathway stimuli, and 2) the efficiency of motion processing in ASD, by obtaining relative contrast sensitivity for discrimination of motion direction vs. detecting the same moving stimulus, in adolescents with ASD, and siblings of individuals with ASD, compared to TD controls. Interpretation of the results in light of previous literature, suggest that relatively enhanced chromatic vs. luminance contrast sensitivity in adolescence may be a marker of a protective factor in siblings of individuals with ASD, who carry some of the genes of ASD, but do not go on to develop ASD. Similarly, it is postulated that impaired local motion processing may be an endophenotype in ASD that has a basis in reduced connectivity between neural areas involved in motion perception, in ASD.

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References

Albright, TD. Cortical processing of visual motion. In: Wallman, J.; Miles, FA., editors. Visual Motion and its Use in the Stabilization of Gaze. Amsterdam: Elsevier; 1993. p. 177-201.

APA. Diagnostic and Statistical Manual of Mental Disorders. 4th ed.. Washington, DC: American Psychiatric Association; 2004.

Bailey A, Le Couteur A, Gottesman I, Bolton P, Simonoff E, Yuzda E, et al. Autism as a strongly genetic disorder: evidence from a British twin study. Psychological Medicine 1995;25(1):63–78. [PubMed: 7792363]

Bailey A, Palferman S, Heavey L, Le Couteur A. Autism: The Phenotype in Relatives. Journal of Autism and Developmental Disorders 1998;28(5):369–392. [PubMed: 9813774]

Belmonte MK, Allen G, Beckel-Mitchener A, Boulanger LM, Carper RA, Webb SJ. Autism and Abnormal Development of Brain Connectivity. The Journal of Neuroscience 2004;24(42):9228–9231. [PubMed: 15496656]

Belmonte MK, Gomot M, Baron-Cohen S. Visual attention in autism families: 'unaffected' sibs share atypical frontal activation. Journal of Child Psychology and Psychiatry 2009;51(3):259–276. [PubMed: 19912448]

Bertone A, Mottron L, Jelenic P, Faubert J. Motion Perception in Autism: A "Complex" Issue. Journal of Cognitive neuroscience 2003;15(2):218–225. [PubMed: 12676059]

Bertone A, Mottron L, Jelenic P, Faubert J. Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. Brain 2005;128(10):2430–2441. [PubMed: 15958508]

Bolte S, Poustka F. The recognition of facial affect in autistic and schizophrenic subjects and their first-degree relatives. Psychological Medicine 2003;33(5):907–915. [PubMed: 12877405]

Bosworth RG, Dobkins K. Chromatic and Luminance Contrast Sensitivity in Fullterm and Preterm Infants. Journal of Vision 2009;9(13):1–16.

Brieber S, Herpertz-Dahlmann B, Fink GR, Kamp-Becker I, Remschmidt H, Konrad K. Coherent motion processing in autism spectrum disorder (ASD): An fMRI study. Neuropsychologia. 2010

Carter, A.; Davis, N.; Klin, A.; Volkmar, F. Social Development in Autism. In: Volkmar, F.; Paul, R.; Klin, A.; Cohen, D., editors. Handbook of Autism and Pervasive Developmental Disorders. Vol. Vol. One. Hoboken, NJ: John Wiley and Sons, Inc.; 2005. p. 312-334.

Curtis WJ, Cicchetti D. Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. Development and Psychopathology 2003;15(3):773–810. [PubMed: 14582940]

Dalton KM, Nacewicz BM, Alexander AL, Davidson RJ. Gaze-fixation, brain activation, and amygdala volume in unaffected siblings of individuals with autism. Biological Psychiatry 2007;61(4):512–520. [PubMed: 17069771]

Davis R, Bockbrader M, Murphy R, Hetrick W, O'Donnell B. Subjective Perceptual Distortions and Visual Dysfunction in Children with Autism. Journal of Autism and Developmental Disorders 2006;36(2):199–210. [PubMed: 16453070]

Del Viva MM, Igliozzi R, Tancredi R, Brizzolara D. Spatial and motion integration in children with autism. Vision Research 2006;46(8–9):1242–1252. [PubMed: 16384591]

Dobkins KR. Moving colors in the lime light. Neuron 2000;25(1):15–18. [PubMed: 10707968]

Dobkins, KR. Enhanced Red/Green Color Input to Motion Processing in Infancy: Evidence for Increasing Dissociation of Color and Motion Information during Development. In: Munakata, Y.; Johnson, MH., editors. Attention and Performance. Vol. Vol. XXI. Oxford University Press; 2005.

Dobkins KR. Does Visual Modularity Increase Over the Course of Development? Optometry & Vision Science 2009;86(6):583–588.

Dobkins, KR.; Albright, TD. Merging Processing Streams: Color Cues for Motion Detection and Interpretation. In: Chalupa, L.; Werner, JS., editors. The Visual Neurosciences. Cambridge: MIT Press; 2004. p. 1217-1228.

Dobkins KR, Anderson CM, Lia B. Infant temporal contrast sensitivity functions (tCSFs) mature earlier for luminance than for chromatic stimuli: evidence for precocious magnocellular development? Vision Research 1999;39(19):3223–3239. [PubMed: 10615492]

Dobkins KR, Bosworth RG, McCleery JP. Effects of gestational length, gender, postnatal age, and birth order on visual contrast sensitivity in infants. Journal of Vision 2009;9(10):11–21.

Dobkins, KR.; Carver, LJ.; Price, E.; Akshoomoff, NA. Enhanced Visual Contrast Sensitivity in Infant Siblings of Children with Autism Spectrum Disorders. Paper presented at the International Meeting for Autism Research (IMFAR); 2010; Philadelphia. 2010.

Dobkins KR, Gunther K, Peterzell DH. What mechanisms underlie red/green isoluminance, luminance contrast sensitivity and chromatic contrast sensitivity at various spatial and temporal frequencies? Vision Research 2000;40(6):613–628. [PubMed: 10824265]

Dobkins KR, Teller DY. Infant Motion: Detection (M:D) Ratios for Chromatically Defined and Luminance-defined Moving Stimuli. Vision Research 1996;36(20):3293–3310. [PubMed: 8944288]

Dorris L, Espie CAE, Knott F, Salt J. Mind-reading difficulties in the siblings of people with Asperger's syndrome: evidence for a genetic influence in the abnormal development of a specific cogniitve domain. Journal of Child Psychology and Psychiatry 2004;45(2):412–418. [PubMed: 14982254]

Field, A. Discovering Statistics using SPSS. 2nd ed.. London: SAGE Publications Ltd.; 2005. Non-parametric Tests; p. 333-334.

Gottesman I, Gould TD. The endophenotype concept in psychiatry: etymology and strategic intentions. American Journal of Psychiatry 2003;160(4):636–645. [PubMed: 12668349]

Gottesman, I.; Sheilds, T. Schizophrenia and Genetics: A twin study vantage point. New York: Academic Press; 1972.

Graham, NVS. Visual pattern analyzers. New York: Oxford University Press; 1989.

Green M. Contrast detection and direction discrimination of drifting gratings. Vision Research 1983;23(3):281–289. [PubMed: 6868403]

Gumbel, EJ. Statistics of Extremes. New York: Columbia University Press; 1958.

Gunther KL, Dobkins KR. Individual differences in chromatic (red/green) contrast sensitivity are constrained by the relative number of L-versus M-cones in the eye. Vision Research 2002;42(11): 1367–1378. [PubMed: 12044743]

Hendry SHC, Reid RC. The Koniocellular Pathway in Primate Vision. Annual Review of Neuroscience 2000;23(1):127–153.

Hobson, P. Autism and Emotion. In: Volkmar, F.; Paul, R.; Klin, A.; Cohen, D., editors. Handbook of Autism and Pervasive Developmental Disorders. Vol. Vol. One. Hoboken, NJ: John Wiley and Sons, Inc.; 2005. p. 406-422.

Ishihara, S. Ishihara's tests for colour-blindness. Tokyo: Kanehara & Co. Ltd.; 1992.

Johnson, NL.; Kotz, S.; Balakrisnan, N. Continuous univariate distributions. Vol. Vol. 2. New York Chicester: Wiley; 1995.

Just MA, Cherkassky VL, Keller TA, Kana RK, Minshew NJ. Functional and Anatomical Cortical Underconnectivity in Autism: Evidence from an fMRI Study of an Executive Function Task and Corpus Callosum Morphometry. Cerebral Cortex 2007;17(4):951–961. [PubMed: 16772313]

Kellerman GR, Fan J, Gorman JM. Auditory abnormalities in autism: toward functional distinctions among findings. CNS Spectrums 2005;10(9):748–756. [PubMed: 16142214]

Kemner C, Lamme VAF, Kovacs I, van Engeland H. Integrity of lateral and feedbackward connections in visual processing in children with pervasive developmental disorder. Neuropsychologia 2007;45(6):1293–1298. [PubMed: 17101159]

Kim-Cohen J. Resilience and Developmental Psychopathology. Child and Adolescent Psychiatric Clinics of North America 2007;16(2):271–283. [PubMed: 17349508]

Koh HC, Milne E, Dobkins KR. Spatial Contrast Sensitivity in Adolescents with Autism Spectrum Disorders. Journal of Autism and Developmental Disorders. 2010 in press.

Lee BB, Pokorny J, Smith VC, Martin PR, Valberg A. Luminance and chromatic modulation sensitivity of macaque ganglion cells and human observers. Journal of Optical Society America 1990;7(12):2223–2236.

Lennie P, D'Zmura M. Mechanisms of color vision. Critical Reviews in Neurobiology 1988;3(4):333–400. [PubMed: 3048707]

Lindsey DT, Teller DY. Motion at isoluminance: Discrimination/detection ratios for moving isoluminant gratings. Vision Research 1990;30(11):1751–1761. [PubMed: 2288088]

Lord C, Risi S, Lambrecht L, Cook EH, Leventhal BL, DiLavore PC, et al. The Autism Diagnostic Observation Schedule—Generic: A Standard Measure of Social and Communication Deficits Associated with the Spectrum of Autism. Journal of Autism and Developmental Disorders 2000;30(3):205–223. [PubMed: 11055457]

Maunsell JH, Nealey TA, DePriest DD. Magnocellular and parvocellular contributions to responses in the middle temporal visual area (MT) of the macaque monkey. Journal of Neuroscience 1990;10(10):3323–3334. [PubMed: 2213142]

McCleery JP, Allman E, Carver LJ, Dobkins KR. Abnormal Magnocellular Pathway Visual Processing in Infants at Risk for Autism. Biological Psychiatry 2007;62(9):1007–1014. [PubMed: 17531206]

Merigan WH, Maunsell JHR. How Parallel are the Primate Visual Pathways? Annual Review of Neuroscience 1993;16(1):369–402.

Merigan WH, Katz LM, Maunsell JH. The effects of parvocellular lateral geniculate lesions on the acuity and contrast sensitivity of macaque monkeys. Journal of Neuroscience 1991;11(4):994–1001. [PubMed: 2010820]

Merigan WH, Maunsell JH. Macaque vision after magnocellular lateral geniculate lesions. Visual Neuroscience 1990;5:347–352. [PubMed: 2265149]

Merigan WH, Eskin TA. Spatio-temporal vision of macaques with severe loss of Pb retinal ganglion cells. Vision Research 1986;26:1751–1761. [PubMed: 3617516]

Milne E, Swettenham J, Campbell R. Motion perception and autistic spectrum disorder: A review. Current Psychology of Cognition 2005;23(1–2):3–36.

Milne E, Swettenham J, Hansen P, Campbell R, Jeffries H, Plaisted K. High motion coherence thresholds in children with autism. Journal of Child Psychology and Psychiatry 2002;43(2):255–263. [PubMed: 11902604]

Mottron L, Dawson M, Soulières I, Hubert B, Burack JA. Enhanced Perceptual Functioning in Autism: An Update, and Eight Principles of Autistic Perception. Journal of Autism and Developmental Disorders 2006;36(1):27–43. [PubMed: 16453071]

Müller RA. The study of autism as a distributed disorder. Mental Retardation and Developmental Disabilities Research Reviews 2007;13(1):85–95. [PubMed: 17326118]

Palmer J, Mobley LA, Teller DY. Motion at isoluminance: discrimination/detection ratios and the summation of luminance and chromatic signals. Journal Optical Society of America. 1993

Pellicano E. Autism: face-processing clues to inheritance. Current Biology 2008;18(17):R748–R750. [PubMed: 18786377]

Pellicano E, Gibson L, Mayberry M, Durkin K, Badcock DR. Abnormal global processing along the dorsal visual pathway in autism: a possible mechanism for weak visuospatial coherence? Neuropsychologia 2005;43(7):1044–1053. [PubMed: 15769490]

Pickles A, Starr E, Kazak S, Bolton P, Papanikolaou K, Bailey A, et al. Variable expression of the autism broader phenotype: findings from extended pedigrees. Journal of Child Psychology and Psychiatry 2000;41(4):491–502. [PubMed: 10836679]

Piven J, Palmer P, Landa R, Santangelo S, Jacobi D, Childress D. Personality and language characteristics in parents from multiple-incidence autism families. American Journal of Psychiatry 1997;74(4):398–411.

Plaisted K, Davis G, Milne E, Swettenham J, Campbell R. Commentary on "Motion Perception in Autism" (Milne, E., Swettenham, J., and Campbell, R.): Examining magnocellular processing in autism. Current Psychology of Cognition 2005;23(1–2):172–179.

Plaisted K, Swettenham J, Rees L. Children with Autism Show Local Precedence in a Divided Attention Task and Global Precedence in a Selective Attention Task. Journal of Child Psychology and Psychiatry 1999;40(5):733–742. [PubMed: 10433407]

Redcay E, Courchesne E. When Is the Brain Enlarged in Autism? A Meta-Analysis of All Brain Size Reports. Biological Psychiatry 2005;58(1):1–9. [PubMed: 15935993]

Rippon G, Brock J, Brown C, Boucher J. Disordered connectivity in the autistic brain: Challenges for the new psychophysiology. International Journal of Psychophysiology 2007;63(2):164–172. [PubMed: 16820239]

Risi S, Lord C, Gotham K, Corsello C, Chrysler C, Szatmari P, et al. Combining Information From Multiple Sources in the Diagnosis of Autism Spectrum Disorders. Journal of Amer Academy of Child & Adolescent Psychiatry 2006;45(9):1094–1103. 1010.1097/1001.chi.0000227880.0000242780.0000227880e.

Rojas D, Maharajh K, Teale P, Rogers S. Reduced neural synchronization of gamma-band MEG oscillations in first-degree relatives of children with autism. BioMed Central Psychiatry 2008;8(1):66. [PubMed: 18673566]

Rutter, M.; Bailey, A.; Lord, C.; Berument, SK. Social Communication Questionnaire. Los Angeles, CA: Western Psychological Services; 2003.

Schiller PH, Logothetis NK, Charles ER. Functions of the colour-opponent and broad-band channels of the visual system. Nature 1990;343(6253):68–70. [PubMed: 2296292]

Shapley R. Visual Sensitivity and Parallel Retinocortical Channels. Annual Review of Psychology 1990;41(1):635–658.

Simmons D, Robertson AE, McKay LS, Toal E, McAleer P, Pollick F. Vision in autism spectrum disorders. Vision Research 2009;49(22):2705–2739. [PubMed: 19682485]

Skottun BC. The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. Vision Research 2000;40(1):111–127. [PubMed: 10768046]

Smith VC, Pokorny J, Davis M, Yeh T. Mechanisms subserving temporal modulation sensitivity in silent-cone substitution. Journal of Optical Society America 1995;12(2):241–249.

Spencer J, O'Brien JMD. Visual form-processing deficits in autism. Perception 2006;35(8):1047–1055. [PubMed: 17076065]

Szatmari P, Maziade M, Zwaigenbaum L, Merette C, Roy MA, Joober R, et al. Informative phenotypes for genetic studies of psychiatric disorders. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics 2007;144(5):581–588.

Tager-Flusberg, H.; Paul, R.; Lord, C. Language and Communication in Autism. In: Volkmar, F.; Paul, R.; Klin, A.; Cohen, D., editors. Handbook of Autism and Pervasive Developmental Disorders. Vol. Vol. One. Hoboken, NJ: John Wiley & Sons, Inc.; 2005. p. 335-364.

Takarae Y, Luna B, Minshew N, Sweeney JA. Patterns of visual sensory and sensorimotor abnormalities in autism vary in relation to history of early language delay. Journal of the International Neuropsychological Society 2008;14:980–989. [PubMed: 18954478]

Taylor MM, Creelman CD. PEST: Efficient estimates on probability functions. Journal of the Acoustical Society of America 1967;41(4A):782–787.

Tsermentseli S, O'Brien J, Spencer J. Comparison of Form and Motion Coherence Processing in Autistic Spectrum Disorders and Dyslexia. Journal of Autism and Developmental Disorders 2008;38(7):1201–1210. [PubMed: 18034294]

Vandenbroucke M, Scholte HS, van Engeland H, Lamme V, Kemner C. Coherent versus Component Motion Perception in Autism Spectrum Disorder. Journal of Autism and Developmental Disorders 2008;38(5):941–949. [PubMed: 17952582]

Watson AB. Probability summation over time. Vision Research 1979;19(5):515–522. [PubMed: 483579]

Watson AB, Thompson PG, Murphy BJ, Nachmias J. ** Summation and discrimination of gratings moving in opposite directions. Vision Research 1980;20(4):341–347. [PubMed: 7414966]

Wechsler, D. Wechsler Abbreviated Scale of Intelligence (WASI). San Antonio, TX: Harcourt Assessment; 1999.

WHO. International Classification of Diseases and Related Health Problems (ICD-10). 1992.

Zwaigenbaum L, Bryson S, Lord C, Rogers S, Carter A, Carver L, et al. Clinical assessment and management of toddlers with suspected autism spectrum disorder: insights from studies of high-risk infants. Pediatrics 2009;5(123):1383–1391. [PubMed: 19403506]

Zwaigenbaum, L.; Stone, MK.; Dobkins, KR. Prospective Evaluation of Head Growth in Infants at Increased Risk of Autism. Paper presented at the International Meeting for Autism Research (IMFAR); 2008; London, England. 2008.

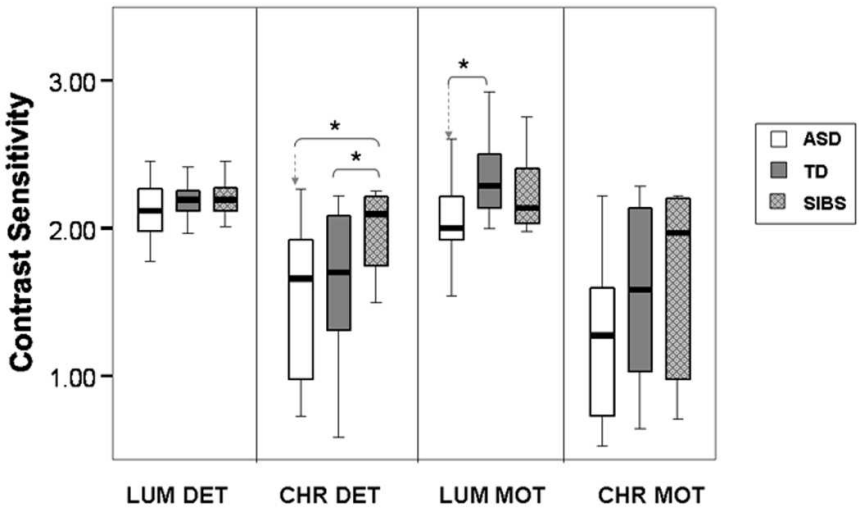

Figure 1.

Box-plots of group log contrast sensitivity data for the three different participant groups (ASD: white, TD: grey, SIBS: patterned). Data are plotted separately for the four different combinations of DET or MOT task, with LUM or CHR stimuli. Horizontal bars within the boxes indicate the median values, the upper and lower edges of the box represent the 75th and 25th quartiles, respectively, and the error bars depict the maximum and minimum of the distribution. * indicates $p<0.05$.

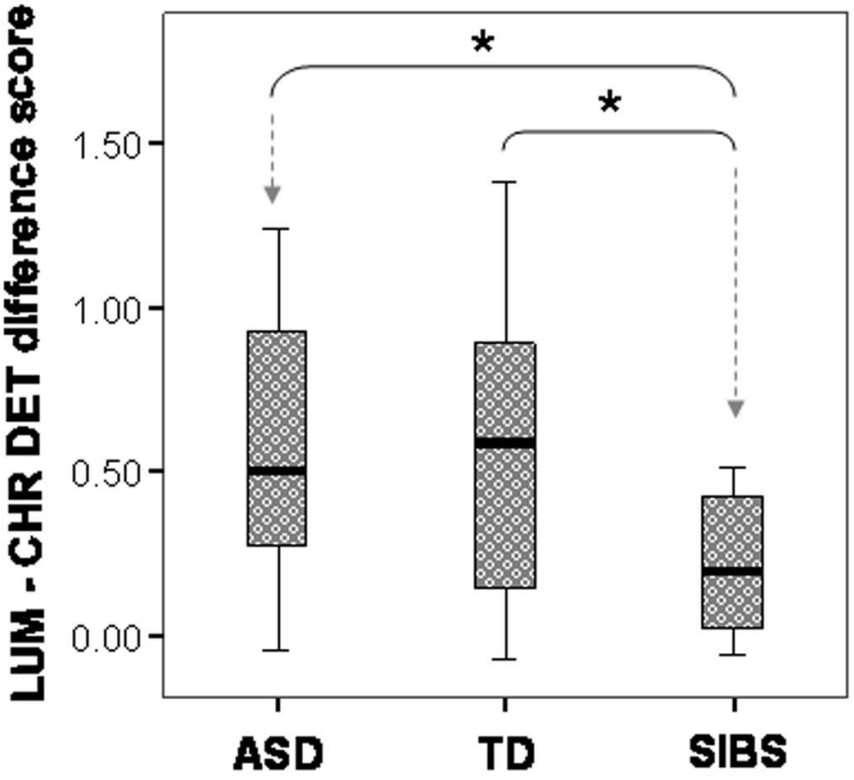

Figure 2.

Box-plots of group LUM - CHR DET difference scores, with data plotted separately for the three different participant groups: Autism Spectrum Disorder (ASD), typically developing (TD) and siblings of individuals with ASD (SIBS). Horizontal bars within the boxes indicate the median values, the upper and lower edges of the box represent the 75th and 25th quartiles, respectively, and the error bars depict the maximum and minimum of the distribution. * indicates $p<0.05$. Note that the most fundamental outcome measurement is the comparison of LUM - CHR scores across participant groups, and not the absolute value of LUM - CHR difference scores per se (see Methods for further explanation). The lower LUM - CHR DET difference score in SIBS is driven by their CHR DET sensitivity being significantly higher than that of ASD or TD participants (rather than their LUM DET sensitivity being lower), see text and Figure 1.

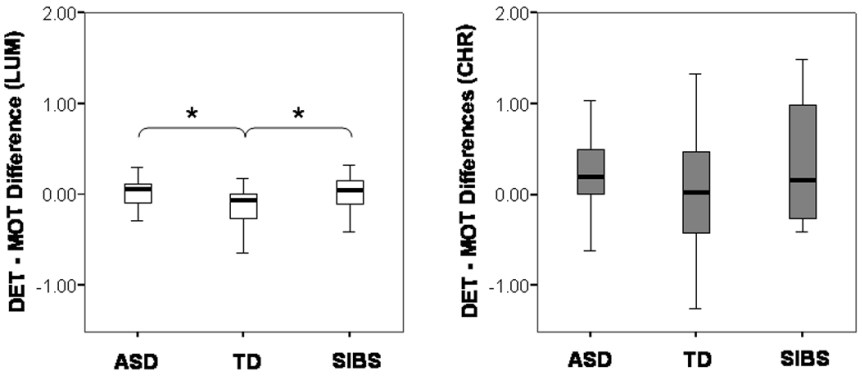

Figure 3.

Box-plots of group DET – MOT difference scores for the two different grating types (LUM: white, CHR: grey). Data are plotted separately for the three different participant groups: ASD, TD and SIBS. Horizontal bars within the boxes indicate the median values, the upper and lower edges of the box represent the 75th and 25th quartiles, respectively, and the error bars depict the maximum and minimum of the distribution. * indicates $p<0.05$. Note that the most fundamental outcome measurement is the comparison of DET – MOT scores across participant groups, and not the absolute value of DET – MOT difference scores per se (see Methods for further explanation).

Table 1

Participant Information: Gender, Chronological Age, IQ Scores, SCQ Scores. ADOS Scores were obtained only for participants with ASD and SIBS.

	ASD (N=17)	**TD (N=39)**	**SIBS (N=12)**	**F & p values**
Gender	16 boys	22 boys	5 boys	
	1 girl	17 girls	7 girls	
Visual issues	11 Normal vision	25 Normal vision	6 Normal vision	
	6 Corrected-to-normal vision	14 Corrected-to-normal vision	6 Corrected-to-normal vision	
Chronological Age (years: months)				
M	14: 10	15: 3	15: 1	F(2,65)=0.445, p=0.643.
SD	1: 10	1: 2	1: 9	
Range	12: 4 – 17: 11	12: 0 – 17: 8	13: 0 – 17: 11	
Verbal IQ				
M	98	109	106	F(2,65)=2.27, p=0.112
SD	24	15	10	
Range	55 – 133	77 – 133	86 – 123	
Performance IQ				
M	105	108	109	F(2,65)=0.67, p=0.517
SD	12	11	10	
Range	86 – 127	86 – 129	86 – 124	
Full Scale IQ				
M	102	109	108	F(2,65)=1.98, p=0.147
SD	17	12	10	
Range	70 – 125	79 – 133	93 – 124	
SCQ Score [a]				
M	23	3	2	F(2,62)=130.7, p<0.001
SD	7	3	2	
Range	6 – 32	0 – 10	0 – 4	
ADOS Total [b]				
M	9		0	t(df=17.4)=11. 8, p<0.001
SD	3		1	
Range	4 – 16		0 – 1	

Note.

[a] Parents of three TD participants did not return the SCQ.

[b] One participant with ASD who had an external diagnosis of Asperger's Syndrome scored below the ADOS cut-off for ASD (total score = 4). He was nonetheless considered ASD, and his data were included in our analyses, because he scored above the cut-off for ASD on the SCQ (score = 22). One SIBS participant felt uncomfortable completing the ADOS assessment, therefore we do not have his ADOS score. However, by the SCQ he was not considered to be on the ASD spectrum (SCQ score = 2), and we therefore retained his data for analysis.