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Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity

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Visuo-perceptual processing in autism is characterized by intact or enhanced performance on static spatial tasks and inferior performance on dynamic tasks, suggesting a deficit of dorsal visual stream processing in autism. However, previous findings by Bertone *et al*. indicate that neuro-integrative mechanisms used to detect complex motion, rather than motion perception *per se*, may be impaired in autism. We present here the first demonstration of concurrent enhanced and decreased performance in autism on the same visuo-spatial static task, wherein the only factor dichotomizing performance was the neural complexity required to discriminate grating orientation. The ability of persons with autism was found to be superior for identifying the orientation of simple, luminance-defined (or first-order) gratings but inferior for complex, texture-defined (or second-order) gratings. Using a flicker contrast sensitivity task, we demonstrated that this finding is probably not due to abnormal information processing at a sub-cortical level (magnocellular and parvocellular functioning). Together, these findings are interpreted as a clear indication of altered low-level perceptual information processing in autism, and confirm that the deficits and assets observed in autistic visual perception are contingent on the complexity of the neural network required to process a given type of visual stimulus. We suggest that atypical neural connectivity, resulting in enhanced lateral inhibition, may account for both enhanced and decreased low-level information processing in autism.

Keywords: autism, enhanced perceptual functioning; first and second order information processing; lateral inhibition; neural networks; perception; visuo-spatial information processing

Abbreviations: ADI = autism diagnostic interview; ADOS-G = autistic diagnostic observation schedule general; FXS = fragile X syndrome; HFA = high-functioning persons with autism; PEST = parameter estimation by sequential testing; TD = typically developing

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Introduction

Autism is a variant phenotype with a neurogenetic basis, defined by negative symptoms affecting social interaction, communication and imagination, as well as by positive symptoms, namely repetitive patterns of behaviours and interests and cognitive strengths (APA, 1994). Given the diagnostic and adaptive importance of social manifestations, it is not surprising that the investigation of underlying neural dysfunction in autism has been for the most part symptom-driven. Accordingly, most neurofunctional research in autism is primarily concerned with assessing higher-level cognitive and/or social capacities. Recent imaging studies indicating atypical brain activation during face processing (Critchley *et al.*, 2000; Schultz *et al.*, 2000; Pierce *et al.*, 2001; Hubl *et al.*, 2003), or during voice processing (Gervais *et al.* 2004), as well as impaired mentalizing ability (Castelli *et al.*, 2002) and modified language integration (Just *et al.*, 2004) exemplify this research direction. Although different with regard to the nature of the dysfunction they describe (cortical

rededication of brain regions devoted to face processing, Schultz *et al.*, 2000; reduced feedback modulation between higher and lower cortical areas, Castelli *et al.*, 2002, Frith, 2003; decreased connectivity between cortical regions, Just *et al.*, 2004, McAlonan *et al.* 2004), all these models converge towards an atypical large-scale neural connectivity in autism, i.e. impoverished integration of information between cortical regions involved in their respective tasks (Frith, 2003; Just *et al.*, 2004). This would be either restricted to the subcomponents of the 'social brain', or generalized to the entire brain.

However, and as alluded to by Belmonte *et al.* (2004), the emphasis on higher-level, symptomology-related functioning has resulted in the over-looking of atypical perceptual processing in autism. Top-down models for autistic patterns of cognitive performance are based on the assumption that lower-level perceptual information processing in autism is intact, or that enhanced performance is due to the hyper-functioning of an otherwise typical low-level perceptual processing system. However, atypical processing of low-level perceptual information is also a characteristic feature of autism (Happé, 1999). The performance of persons with autism on tasks necessitating the detection of a static visual target embedded in a larger field has been found to be either enhanced (Plaisted *et al.*, 1999; O'Riordan *et al.*, 2001; Caron *et al.*, 2004; Pellicano *et al.*, 2005) or more locally oriented (Shah and Frith, 1983, 1993; Jolliffe *et al.*, 1997; Mottron and Belleville, 1999; Lahaie *et al.*, 2005) when compared to typically developing observers. Hypotheses explaining such perceptual assets in autism include superior processing of low-level static information (Plaisted *et al.*, 1998; Mottron and Burack, 2001) or a by-product of limited integration of low-level information in higher-order operations (Frith, 2003).

Recent research has shown that visual information processing in autism presents a dichotomous picture, with intact or enhanced performance on tasks necessitating static spatial information processing, and inferior performance regarding dynamic information analysis. Persons with autism are consistently less sensitive to a variety of complex motion stimuli which include full-field radiating flow fields (Gepner *et al.*, 1995), adapted global motion stimuli (Spencer *et al.*, 2000), random dot kinematograms (Milne *et al.*, 2002), biological motion stimuli (Blake *et al.*, 2003) and texture-defined motion patterns (Bertone *et al.*, 2003). All the aforementioned complex motion stimuli are processed in motion-sensitive, extra-striate areas located within the dorsal visual pathway (i.e. Goodale and Milner, 1992) and necessitate feed-forward integrative processing to be perceived (Watamaniuk and Sekular, 1992; Wilson *et al.*, 1992; Neri *et al.*, 1998; Bertone *et al.*, 2003).

Such findings of decreased complex motion have been attributed, for the most part, to either a motion-processing deficit *per se* (Gepner *et al.*, 1995), or to a dorsal stream dysfunction (Spencer *et al.*, 2000; Milne *et al.*, 2002; Blake *et al.*, 2003) since motion-sensitive areas operate within this pathway (Ungerleider and Mishkin, 1982; Merigan *et al.*, 1991; Goodale and Milner, 1992; Merigan and Maunsell, 1993; Milner and Goodale, 1995). We refer to this interpretation as the pathway-specific hypothesis. This interpretation has been supported by concurrent demonstrations of intact global form analysis and typical perception of the global aspect of hierarchical stimuli (Ozonoff *et al.*, 1994; Plaisted *et al.*, 1999; Spencer *et al.*, 2000; Blake *et al.*, 2003; Mottron *et al.*, 2003), believed to be mediated by mechanisms operating within the ventral pathway (Gallant *et al.*, 1993, 1996; Wilson *et al.*, 1997, 1998; Wilkinson *et al.*, 2000).

Bertone *et al.* (2003) proposed alternatively that decreased complex motion sensitivity in autism might be explained by the reduced efficiency of neuro-integrative mechanisms operating at a perceptual level in autism. We refer to this interpretation as the complexity-specific hypothesis. They measured sensitivity to two types of motion stimuli, differing in the amount of neuro-integrative analysis required to perceive the motion. The pathway-specific hypothesis would predict a decrease in sensitivity to any motion stimuli, regardless of the amount of neuro-integrative processing involved in its processing. Simple, first-order motion (luminance-defined) perception was found unaffected for persons with autism, but there was a selective decrease for complex, second-order (or texture-defined) motion perception (Chubb and Sperling, 1988; Cavanagh and Mather, 1989). The first versus second-order dissociation was impossible to account for in the framework guiding previous investigations (Gepner *et al.*, 1995; Spencer *et al.*, 2000; Milne *et al.*, 2002; Blake *et al.*, 2003) since motion sensitivity was measured for only complex motion types (i.e. assessing functioning of motion-sensitive mechanisms at only one level along the dorsal pathway). Since the processing of simple motion is carried out by standard motion analysis at early levels within the dorsal pathway, Bertone *et al.* (2003) suggested that their results (and others demonstrating inferior autistic sensitivity to complex motion) might be better explained by a complexity-specific hypothesis. This hypothesis predicts that even low-level visual information necessitating complex neuro-integrative resources should be affected in autism. Therefore, a similar explanation, for decreased perceptual integration, may account for local bias in static stimuli, and for defective perception for dynamic, second-order information.

In order to further differentiate between these two hypotheses and to more precisely characterize visuo-perceptual profile of abilities in autism, two experiments were performed. Static information processing in autism was assessed by using stimuli that varied in the complexity of the presented information. This was accomplished by measuring the ability of high-functioning persons with autism (HFA) and typically developing (TD) observers to identify the orientation of both simple (first-order) and complex (second-order) static gratings. Importantly, and in relation to the anatomical substrate of the pathway-specific hypothesis, these two types of stimuli are processed at two different levels of the ventral stream. The pathway-specific hypothesis would predict that the complexity of the presented stimulus would not affect performance for the HFA group since it contends that only dynamic

information processing (mediated by dorsal stream functioning) is affected in autism. Conversely, the complexity-specific hypothesis would predict a decrease in HFA performance for the second-order condition only, since it contends that inefficient neuro-integrative functioning preferentially affects complex information analysis in autism, regardless of whether the presented visual information is static or dynamic. In addition, we also evaluated the functional integrity of the sub-cortical visual processing using a flicker contrast sensitivity task with stimuli that preferentially evaluated the magnocellular and parvocellular systems. Accordingly, previous findings of decreased global motion stimuli sensitivity in autism have been interpreted as evidence of deficient pre-cortical (i.e. magnocellular) processing of dynamic information (Milne *et al.*, 2002).

Methods

Participants

Thirteen HFA individuals with normal intelligence (average Wechsler FSIQ = 100.4, SD = 13.6) were recruited from a specialized clinic for persons with autism. A diagnosis of autism was obtained using the algorithm of the Autism Diagnostic Interview (ADI; Lord *et al.*, 1994) combined with the Autistic Diagnostic Observation Schedule General (ADOS-G; Lord *et al.*, 2000). Current and retrospective standardized assessments were conducted by a trained researcher (LM) who obtained reliability on these instruments. All HFA had a score above the ADI/ADOS cut-off in the four areas relevant for diagnosis (social, communication, restricted interest and repetitive behaviour, and age of symptom onset). Thirteen TD participants were recruited from the community as a comparison group. These were screened for a past or current history of psychiatric, neurological or other medical disorders and all had a typical academic background and development (mean IQ = 108.2, SD = 13.1). The groups were matched as closely as possible in terms of laterality, gender and chronological age and full-scale IQ. The mean chronological age of the control and autism groups was 22.3 (SD = 6.1) and 20.5 years (SD = 4.3), respectively. All participants had normal or corrected-to-normal vision (Table 1). Informed written consent was obtained from all participants.

Apparatus

For all testing, stimulus presentation and data collection were controlled by a Power Macintosh G4 microcomputer and presented on a 14-inch AppleVision color monitor refreshed at a rate of 75 cycles per second (Hz). The screen resolution was 1152 × 870 pixels. The VPixx©(www.vpixx.com) graphics program controlled stimulus generation and animation. The luminance of the monitor was gamma-corrected (implemented with a colour calibration within the VPixx© program) to minimize the non-linearities in the display. Calibration and luminance readings were verified using a Minolta CS-100 Chroma Meter colorimeter on a regular basis.

Table 1 Participant characteristics for HFA and TD participants

Participant characteristic	HFA participants	TD participants
Number	13	13
Age (y : m)		
Mean	22 : 3	20 : 5
SD	6.1	4.3
Range	11.0–31.0	14.0–27.0
FSIQ		
Mean	100.4	108.2
SD	13.6	13.1
Range	82–120	90–137

Psychophysical tasks

Orientation-identification task

In order to differentiate between the pathway-specific and complexity-specific hypotheses, all participants completed two psychophysical tasks. The orientation-identification task assessed static information processing, using stimuli that varied in complexity and are processed by mechanisms operating at different levels along the ventral stream. For this task, the pathway-specific hypothesis would predict no differences in sensitivity between TD and HFA groups since only dorsal visual stream-mediated dynamic information processing is argued to be dysfunctional in autism (Spencer *et al.*, 2000; Milne *et al.*, 2002; Blake *et al.*, 2003). On the other hand, the complexity-specific hypothesis would predict a selective decrease in sensitivity for the complex (second-order), but not the simple (first-order) gratings for the HFA group, since it contends that complex information processing, whether static or dynamic, is affected in autism.

The static stimuli used in the orientation-identification task are a static version of the motion stimuli used in the translational condition of the Bertone *et al.* (2003) study. As described below, these static stimuli were constructed in the same manner as their dynamic counterparts (i.e. Ledgeway and Smith, 1994; Bertone and Faubert, 2003), by either adding or multiplying greyscale noise to a modulating sinewave (velocity = 0). Static and dynamic forms of first- and second-order information are initially processed in parallel by separate feed-forward mechanisms, using similar principles of detection (Chubb and Sperling, 1988; Wilson *et al.*, 1992; Baker, 1999). This processing is exemplified by filter-rectify-filter analysis where the first stage filters, operating within V1, extract first-order orientation or motion direction, whereas second-order orientation or motion information is detected at a second stage of filtering at a coarser spatial scale (in areas V2/V3), but only after rectification of the second-order signals (e.g. Chubb and Sperling, 1988; Wilson *et al.*, 1992; Sperling *et al.*, 1994; Sutter *et al.*, 1995; Smith *et al.*, 1998; Wilson, 1998; Nishida *et al.*, 1997; Baker, 1999; Bertone and Faubert, 2003). For this reason, first-order static information can also be considered 'simple' whereas second-order static information is considered more 'complex' since it recruits more extensive neural circuitry as well as additional processing prior to orientation identification.

Stimuli

Static stimuli were presented to the participants within a circular region at the centre of the display that had a diameter

of 10° when viewed from a distance of 57 cm. The mean luminance of the remainder of the display during testing was 15.00 cd/m^2 (u′ = 0.1912, v′ = 0.4456 in CIE (Commission Internationale de l'Eclairage) u′ v′ colour space) where L_{min} and L_{max} were 0.02 and 30.02 cd/m^2, respectively. The static stimuli consisted of first- and second-order gratings presented either vertically or horizontally. The first-order motion stimuli (Fig. 1) were luminance-defined noise stimuli produced by adding static greyscale noise to a modulating sinewave. The noise consisted of dots (1 pixel × 1 pixel, measuring ~2.235 arc min) with individual luminances randomly assigned as a function of sin(x), where (x) ranged from 0 to 2π. The luminance-contrast of the first-order stimuli was varied to determine orientation-discrimination thresholds by varying the amplitude of the modulating sinewave. The amplitude of the luminance modulation for the first-order patterns could be varied from 0.0 to 0.5 defined as:

$$\text{Luminance modulation depth} = (L_{max} - L_{min})/(L_{max} + L_{min}),$$

where L_{min} and L_{max} refer to the average highest and lowest local luminances in the pattern. The first-order luminance modulation levels used in the constant stimuli presentations (0.10, 0.05, 0.035, 0.02, 0.0125 and 0.00625) were chosen based on pilot studies. The second-order stimuli (Fig. 1b) were texture-modulated noise stimuli produced by multiplying rather than summing the same modulating sinewaves to the greyscale noise. The texture-contrast (contrast modulation depth) was also varied to find orientation-discrimination thresholds by varying the amplitude of the modulating sinewave. The amplitude of the texture-modulation that defined the contrast of the second-order stimuli could be varied within a range of 0.0 and 1.0 defined as:

$$\text{Contrast modulation depth} = (C_{max} - C_{min})/(C_{max} + C_{min}),$$

where C_{max} and C_{min} are the maximum and minimum local contrasts in the pattern. Second-order contrast modulation levels used during the constant stimuli procedures were 1.0, 0.429, 0.250, 0.143, 0.067 and 0.032. All first- and second-order static stimuli had a spatial frequency of 0.75 cycles per degree (c.p.d.) and a drift frequency of 0 cycles per second (Hz).

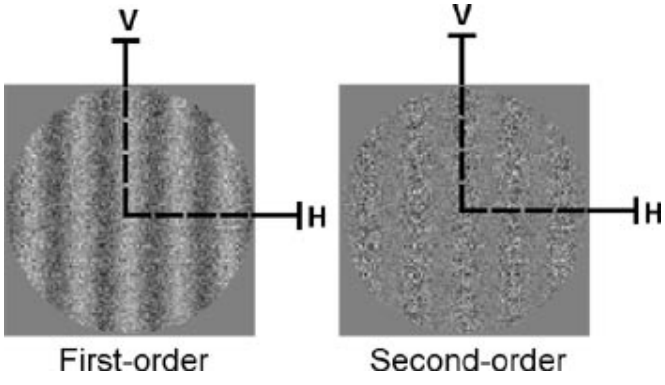

Fig. 1 Schematic representation of static stimuli used for experiment 1. First- and second-order stimuli are presented in their vertical (V) orientation. Both static and dynamic forms of first- and second-order information are initially processed in parallel by separate passive mechanisms using similar principles of detection. Specifically, first stage filters, operating within V1, extract first-order orientation or motion direction whereas second-order information is detected at a second stage of filtering at a coarser spatial scale (in areas V2/V3), but only after full-wave rectification of the second-order signals (Wilson *et al.*, 1992; Chubb and Sperling, 1988, Cavanagh and Mather, 1989; Sperling *et al.*, 1994; Baker, 1999). For this reason, first-order information can be considered to be simple and second-order information complex because the latter type recruits more extensive neural circuitry as well as additional processing prior to detection.

Procedure

Participants were tested individually in a dimly lit room. Procedural instructions were given verbally to each participant prior to each experimental block. Before the actual testing, practice trials were completed so that the participants could familiarize themselves with fixation, stimuli presentation and responding. Each participant was then presented with static first- and second-order stimuli oriented either vertically or horizontally for 750 ms. They were then required to identify the orientation of each stimulus by pressing one of two buttons on a keypad (2 alternative forced-choice). For each testing session, first- and second-order stimuli were presented in random order ten times in either orientation at each level of modulation (for a total of twenty trials at each level of modulation). Psychometric functions were then fitted to the responses for each condition in order to obtain orientation-identification thresholds at a 75% correct level of performance. Throughout testing, the participants were reminded to fixate at the centre of each pattern. The experimenter remained present throughout testing and initiated successive trials.

Flicker contrast sensitivity task

Efficient ventral and dorsal visual stream functioning is dependent on afferent parvocellular and magnocellular input, respectively, originating from retinal and thalamic levels of information processing (i.e. Shapley, 1990; Merigan and Maunsell, 1990). Magnocellular neurons respond preferentially to low-spatial/high-temporal frequency defined stimuli (best suited for dynamic information processing), whereas parvocellular neurons respond preferentially to high-spatial/low-temporal frequency defined stimuli (best suited for static information processing) (Tohlurst, 1975; Derrington and Lennie, 1984; Merigan and Maunsell, 1990, 1993; Shiller *et al.*, 1990; Merigan *et al.*, 1991; Lynch *et al.*, 1992; Chapman *et al.*, 2004). The purpose of this task was to assess the integrity of pre-cortical (magno- and parvocelluar) visual functioning in autism. Pre-cortical magnocellular functioning in autism has been assessed by Pellicano *et al.* (2005) using a flicker task

(minimum contrast needed to detect a stimulus flickering at 10 Hz). They demonstrated no significant difference in sensitivity between autism and control groups; parvocellular functioning was also evaluated in the present study.

Stimuli

As was the case for the orientation-discrimination task, flicker stimuli were presented to the participants within a circular region at the centre of the display that had a diameter of 10° when viewed from a distance of 57 cm. The mean luminance of the remainder of the display during testing was 17.70 cd/m^2 [u′ = 0.1912, v′ = 0.4456 in CIE (Commission Internationale de l'Eclairage) u′ v′ colour space] where L_{min} and L_{max} were 0.01 and 35.40 cd/m^2, respectively. A two-alternative temporal forced choice (2ATFC) paradigm was used to measure the minimum contrast needed to detect a 0.5 c.p.d. sinusoidal luminance grating counterphasing at a rate of 6 Hz (magnocellular condition) and a 6 c.p.d. sinusoidal luminance grating counterphasing at a rate of 1 Hz (parvocellular condition).

Procedure

For both magno- and parvocellular conditions, participants were presented with trials consisting of flickering stimuli of a certain luminance contrast for 750 ms, followed (or preceded) by a stimulus containing no flicker information (i.e. static grey region). Participants were required to identify the trial that contained the flickering stimuli (i.e. first or second presentation). Luminance contrast was the physical variable being manipulated for each condition using an adaptive PEST (parameter estimation by sequential testing) routine (Pentland, 1980). This method was chosen over the more conventional staircase procedures because the PEST has been shown to significantly reduce the number of trials necessary to determine a threshold compared to staircase methods at a given level of accuracy (Taylor *et al.*, 1983). A session ended when the PEST routine converged on the 81% level on a psychometric Weibull function (Weibull, 1951), representing the flicker contrast thresholds for each condition, which were then transformed into flicker contrast sensitivity measures. For the PEST routine to end for each condition, a preset level of accuracy (95% confidence interval at threshold was within 0.1 log units of the PESTed threshold) had to be met. The maximum number of trials allowed, fixed at one hundred for each condition, was never met. The total time taken for each participant to complete both orientation-discrimination and flicker sensitivity tasks took, on average, ~60 min.

Results

Orientation-identification task: enhanced and diminished autistic performance depends on stimulus complexity

Orientation-identification thresholds for HFA and TD participants were measured using static gratings differing only in the attribute defining their orientation; luminance for the first-order condition and texture for the second-order condition (Fig. 1). Results revealed two very different patterns of HFA performance, contingent on the complexity of the stimuli used in each condition.

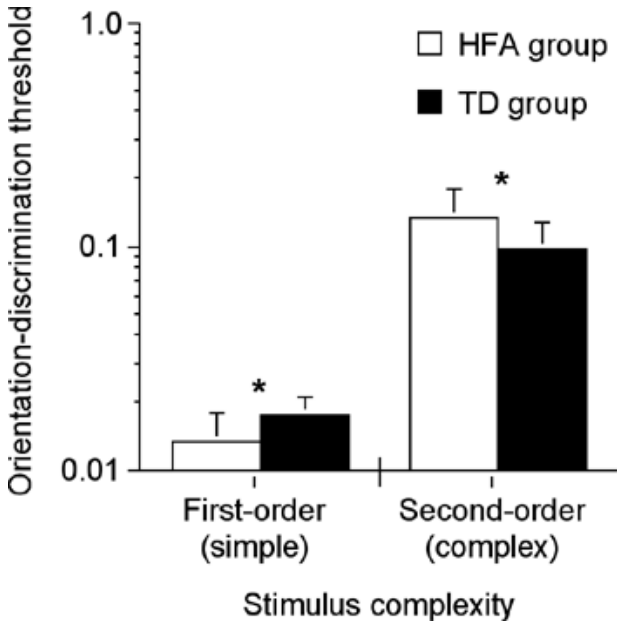

Fig. 2 Orientation-discrimination thresholds as a function of stimulus complexity for HFA and TD participants. Since first- and second-order stimuli are constructed using different image attributes, the absolute difference between first- and second-order thresholds is uninformative. Error bars represent 1 standard deviation.

As shown in Fig. 2, HFA orientation-identification thresholds were significantly lower for the first-order condition when compared to the TD participants [F (1,24) = 7.872, P = 0.0098]. These findings represent another demonstration of superior performance in tasks necessitating elementary visuo-spatial information processing (i.e. position discrimination, visual search, etc.), albeit at a lower level of processing. In contrast, HFA thresholds were significantly higher for the same task using complex second-order stimuli [F (1,24) = 5.042, P = 0.0342], representing the first demonstration of a perceptual visual deficit for a static task in autism. Taken as a whole, these findings suggest that enhanced autistic performance on visuo-spatial tasks is complexity dependent, and that persons with autism are selectively less sensitive to complex visual information, whether it is static or dynamic in nature. These results will be discussed in the context of a complexity-specific account of visuo-perceptual processing in autism in later sections.

Flicker sensitivity task: unaffected pre-cortical visual functioning in autism

Flicker contrast sensitivity did not differ significantly between HFA and TD participants for either magnocellular

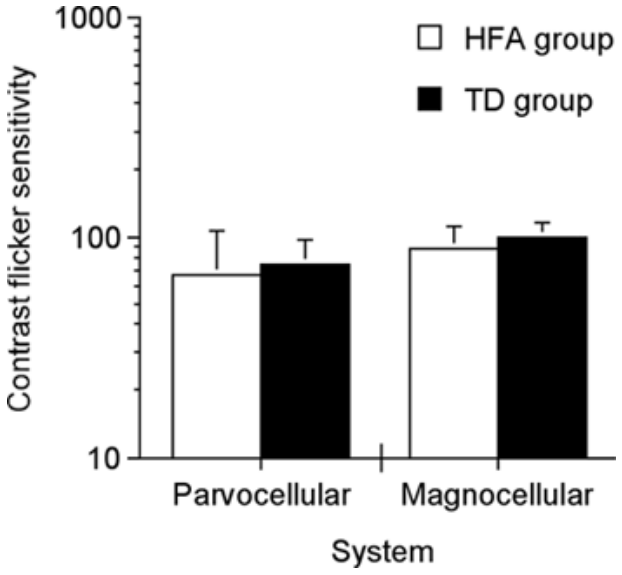

Fig. 3 Contrast flicker sensitivity measures for parvocellular and magnocellular functioning for HFA and TD groups. Error bars represent 1 standard deviation.

[$F(1,24) = 1.729$, $P = 0.2009$] or parvocellular [$F(1,24) = 0.451$, $P = 0.5810$] conditions (see Fig. 3). These findings are consistent with those of Pellicano *et al.* (2005). In addition to demonstrating intact simple motion perception in autism (Bertone *et al.*, 2003), the current findings do not support a pathway-specific account of perceptual abnormalities in autism. Accordingly, a dorsal pathway dysfunction resulting in decreased complex motion perception would entail a disruption of afferent magnocellular visual inputs (Chapman *et al.*, 2004).

The finding of unaffected parvocellular (pre-cortical) functioning suggests that enhanced and diminished performance on the orientation-discrimination task result from atypical processing at early cortical levels in autism, and is not the result of abnormal pre-cortical input.

Discussion

Pathway—versus complexity—specific information processing hypotheses in autism

The present study represents the first evaluation of ventral stream processing in autism at two levels of neural complexity, assessed by measuring orientation-discrimination thresholds for simple luminance- and complex texture-defined stimuli for both HFA and TD observers. By demonstrating that complex static information processing is selectively impaired in autism, we propose that atypical visual information analysis in autism is best described by a complexity-specific account. Regardless of whether the visual information is dynamic (Bertone *et al.*, 2003) or static (current findings), diminished neuro-integrative functioning at a perceptual level preferentially affects complex information analysis. We are able to forward this suggestion because both ventral (current study) and dorsal visual stream (Bertone *et al.*, 2003) functioning have now been evaluated at two levels of complexity, using static and dynamic stimuli differing solely and comparably in level of complexity. Our proposition contradicts two underpinnings of the pathway-specific hypothesis: impairment of dorsal stream and integrity of ventral stream. These two aspects will be discussed separately.

Impairment of dorsal stream

We contend that previous demonstrations of decreased motion sensitivity, attributed either to motion impairments or to dorsal stream dysfunction (Gepner *et al.*, 1995; Spencer *et al.*, 2000; Milne *et al.*, 2002; Blake *et al.*, 2003), may be re-interpreted according to the complexity-specific hypothesis. Measuring complex motion sensitivity in isolation does not allow for differentiation between pathway and complexity-specific hypotheses. Accordingly, the integrity of first-order movement perception evident in Bertone *et al.* (2003) indicates that it is not movement *per se* which is impaired. This argument has recently been supported by the results of Pellicano *et al.* (2005) who demonstrated that only complex motion processing (global motion) is affected in autism, while no dysfunction of early visual processing was demonstrated using a flicker contrast sensitivity task. These findings were interpreted as intact lower-level dorsal stream functioning in autism (Bertone *et al.*, 2003), and evidence against a generalized 'dorsal stream deficit'. Pellicano *et al.*'s (2005) results are congruent with the complexity-specific hypothesis, since deficits in motion processing were only found at higher (i.e. processed by extra-striate mechanisms) but not lower (pre-cortical or magnocellular) levels of analysis along the dorsal visual stream, possibly reflecting neuro-integrative dysfunction in autism (Bertone *et al.*, 2003). In addition, there has yet to be a direct demonstration of either a physiological or anatomical abnormality of thalamic magnocellular structures in autism, as has been demonstrated in FXS (Kogan *et al.*, 2004*b*).

Integrity of ventral stream

The pathway-specific hypothesis is supported by a differentiation between impaired movement perception and preserved static perception in autism. Previous studies proposed that ventral stream processing is unremarkable in autism by demonstrating a typical detection of circular forms composed of locally-oriented line segments (Spencer *et al.*, 2000; Blake *et al.*, 2003). We propose that the argued static/dynamic dissociation reported in these studies may be stimulus dependent, since static circular stimuli were not equivalent to their complex dynamic counterparts (which were not circular in nature) in terms of processing requirements [see Bertone (2004) for complete discussion] and, therefore, differ in terms of their sensitivity to neuro-integrative dysfunction.

Integrating complex visual information, whether static or dynamic, is more efficient when local information is organized in a circular manner due to specialized analysis (Freeman and Harris, 1992; Kovács and Julesz, 1993; Wilson *et al.*, 1997; Wilkinson *et al.*, 1998; Kovács *et al.*, 1999; Burr and Santoro, 2001; Achtman *et al.*, 2003). Within the context of experimental approaches used by Spencer *et al.* (2000) and Blake *et al.* (2003), one can argue that the decrease in sensitivity for the complex motion condition (and not the complex form condition) may have been, at least in part, due to the fact that only one condition used circular stimuli (complex form condition). As mentioned by Pellicano *et al.* (2005), the detection of complex form contours from individual oriented line elements may be achieved at earlier levels than previously believed i.e. orientation selective mechanisms operating in V1 [see Hess *et al.* (2003) for a review]. Although such complex motion and form tasks selectively assess dorsal and ventral stream processing, respectively, they do not in effect assess functioning in either stream at the same level of neural complexity. We believe that the static stimuli used in the present study (i.e. stationary first- and second-order gratings) are better matched to their dynamic counterparts in terms of how 'efficiently' they are processed (see Kogan *et al.*, 2004*a*), allowing for a more accurate assessment of dorsal and ventral stream processing in autism. In conclusion, neuro-integrative processing can be demonstrated to be dysfunctional in both ventral and dorsal visual streams in autism, as long as the stimuli used to assess the functional integrity of both streams are not circular in nature.

The neural origin of enhanced static information processing in autism

Our results demonstrate that the performance of HFA participants is inferior at discriminating the orientation of second-order gratings but superior for first-order gratings. This dichotomous performance contrasts two types of information differing only in the physical attribute orientation, luminance versus texture. In typical participants, the first- versus second-order distinction is associated with two distinct levels of integration among visual areas (V1 only versus V1 + V2/V3). Two alternative and partially overlapping interpretations of this pattern of findings, involving neural versus regional level of organization, will be discussed.

A first interpretation of this pattern of performance would be that functional regions involved in the visual system operate better in isolation than in synchrony. It can be summarized as 'superior when autonomous, inferior when synchronized'. According to this interpretation, first-order patterns would be processed at a higher level of performance because they may be analysed within a single brain region (or a brain region more limited in surface), whereas second-order information requires communication among regions to be automatic and constant. This trend of interpretation is inspired by the model initially proposed by Castelli *et al.* (2002) for higher-order performances in autism. Castelli *et al.* found diminished functional synchrony between extrastriate areas and the superior temporal sulcus during a theory of mind task, while the early visual processing areas (extrastriate cortex) were activated normally in individuals with autism. A so-called 'under connectivity' between regions involved in syntactic processing was also recently established using fMRI (Just *et al.*, 2004). During the processing of complex syntactic sentences, HFA individuals presented with a reduced functional synchrony, measured by the correlation between the average time course of all the activated voxels in each member of pairs of anatomically defined ROIs implicated in language processing. Finally, a recent study using diffusion tensor imaging (Barnea-Goraly *et al.*, 2004) revealed a reduction of white matter tracts adjacent to the grey matter of associative cortex (temporo-parietal junctions, superior temporal sulcus) and between prefrontal and temporal cortices. Some neural network models of visual processing implicate feedback or top-down connectivity as being an important part of information coding in V1 (Rao and Ballard, 1999; Angelucci *et al.*, 2002). These models are defined by the existence of feedback connections between higher (i.e. V2, V3, V4, MT) and lower (i.e. V1) visual areas that result in the amplification and focused activity of neurons in lower-order areas, implicated in such perceptual processing as orientation selectivity and figure ground differentiation (Hupé *et al.*, 1998; Bullier *et al.*, 2001). This 'superior when autonomous, inferior when synchronized' hypothesis receives, therefore, increasing support from studies on large-scale communication/synchrony among brain regions. Apart from the current findings, it has, however, not yet found support from subregions implicated in low level processing.

A second trend of interpretation is that the same atypical neural system mediating orientation information processing in autism may have two opposite perceptual consequences, depending on the complexity of the information provided. We propose that this dichotomous performance is best explained by an atypical neural connectivity mediating the extraction of low-level orientation information within the visual processing hierarchy in autism (Cohen, 1994; Gustaffson, 1997*a*, *b*; McLelland, 2000; Grice *et al.*, 2001; Brock *et al.*, 2002; Bertone *et al.*, 2003, 2004). The efficient orientation selectivity and detection of neurons in the primary visual cortex (V1) is largely dependent on cortical lateral interactions between orientation-selective neurons (Andrews, 1965). Therefore, the type of abnormal connectivity the most congruent with enhanced sensitivity to simple luminance-defined gratings is that of strong or excessive lateral inhibition [This model is also consistent with the hypothesis of increased *latent* inhibition, proposed by K. Plaisted (in prep.)], as first suggested by Gustafsson (1997*a*, *b*). Gustafsson's model is based on a 'feature map' model of cortical functioning where neurons selective to a certain orientation are arranged in columns. These columns are optimally activated (increased neuronal activity within each column) when a specific orientation (i.e. vertical) is presented (Kohonen, 1995). Lateral inhibition allows proximal columns to be activated by similar

orientations, resulting in a bell-shaped tuning curve function (or 'Mexican hat' function) in the orientation domain, for each orientation-selective column. For typically developing individuals, a possible functional role of lateral inhibition is to sharpen the orientation tuning of each of these columns (Sillito *et al.*, 1995; Gilbert *et al.*, 1996; Gilbert, 1998; Somers *et al.*, 2002). We propose that increasing lateral inhibition would narrow the range of a particular orientation activating each column, resulting in an improved ability for both the detection of oriented stimuli and the discrimination between different orientations. As suggested by Gustafsson (1997*a*, *b*), narrowing the tuning of each column would facilitate 'discrimination' between orientations (resulting in enhanced edge detection), whereas widening their tuning curves would facilitate 'generalization'. Increased lateral inhibition would, therefore, produce an increased performance on the orientation-discrimination task stimuli for the HFA group, since luminance-defined stimuli can be analysed by a single orientation-selective neuron with a narrowly tuned receptive field in V1. Physiological support for this suggestion has come form recent neuropathological studies demonstrating numerous and more narrow minicolumns (i.e. columns of orientation selective neurons) in the autistic brain (Casanova *et al.*, 2002*a*, *b*).

Although the narrowing of microcolumns may be responsible for enhanced static information processing in autism, why did not persons with autism demonstrate enhanced sensitivity to the simple motion stimuli (i.e. translational condition) used in the Bertone *et al.* (2003) study? A possible explanation would be based on the differential filtering properties of early mechanisms mediating static and dynamic information. Human motion-detectors operating in the primary visual cortex have a very broad orientation tuning (broadband detectors) (i.e. Anderson and Burr, 1991; Georgeson and Scott-Samuel, 2000). In contrast, a subset of neurons in V1 are extremely selective for orientation, in part the result of both lateral and feed-back connections. Therefore, the processing of simple dynamic information may be less affected by atypical neural connectivity such as strong or excessive lateral inhibition. To our knowledge, there has yet to be a clear demonstration of an 'autistic advantage' or enhanced autistic performance on a task necessitating dynamic information analysis.

Enhanced edge detection caused by increased lateral inhibition may also be implicated in other findings of improved autistic performance on visuo-spatial tasks, inasmuch as these tasks involve the discrimination of luminance-defined stimuli, mediated by low-level perceptual processing (Plaisted, 1999; O'Riordan and Plaisted, 2000; O'Riordan *et al.*, 2001; Caron *et al.*, 2004). As suggested by Casanova *et al.* (2003), if the enhanced discrimination on visuo-spatial tasks in autism is indeed the result of altered low-level information processing, its neural origin is most likely at the level of the microcolumn, where visual information is initially filtered/processed before being fed forward to higher-level visual mechanisms. Interestingly, enhanced low-level perceptual functioning has also been demonstrated in the auditory modality (Mottron and Burack, 2000). Although speculative, a similar explanation may be provided for enhanced low-level auditory perception, since neural organization within the primary auditory cortex has a columnar arrangement similar to that of the primary visual cortex (Abeles and Goldstein, 1970). Increased lateral inhibition between frequency-specific columns may, therefore, result in an increased temporal resolution, with the benefit of enhanced pitch sensitivity in autism (Bonnel *et al.*, 2003) and diminished local-to-local interference (Foxton *et al.*, 2003).

Conversely, the same neural atypicality may have a detrimental effect on other types of low-level information in autism, such as complex texture-defined static information. Neurons comprising feature-specific columns in V1 selectively respond to oriented edges defined by changes in luminance, such as the simple luminance-defined, first-order stimuli used in our task (Fig. 1). In contrast, enhanced edge detection mediated by lateral inhibition for complex texture-defined information has been demonstrated, but only after additional information processing (i.e. full-wave rectification, see legend of Fig. 1) (Lu *et al.*, 1996). After such processing, the resulting texture-defined spatial information is much coarser in nature, as defined by the filter-rectify-filter hypothesis (Chubb and Sperling, 1988; Cavanagh and Mather, 1989; Wilson *et al.*, 1992; Sperling *et al.*, 1994; Baker, 1999). It is, therefore, less likely that the 'narrowing' of the orientation-selective, luminance-driven columns in V1 improve orientation-discrimination of texture-defined stimuli for the HFA group. On the contrary, the same altered local neural networks in autism may hinder the processing for more complex types of static information necessitating integration via lateral connections between orientation-selective V1 neurons analysing nearby spatial locations (Field *et al.*, 1993) since a larger neural circuitry is involved.

In conclusion, the primary function of lateral and feedback connectivity within low-level visual areas is to identify, modulate and optimize neural signals belonging to elementary visual features (i.e. orientation). These signals are subsequently fed forward and integrated by specialized mechanisms operating in higher visual areas. It is possible that both lateral and feedback connectivity are atypical in autism, since each type of connectivity is implicated in orientation selectivity. Lateral and feedback connectivity are differentially involved in integrating signals within (lateral connections) and between (feedback) visual areas (Angelucci *et al.*, 2002). However, our data show that atypical connectivity may be implicated initially within low-level visual areas rather than (or in addition to) between higher and lower visual areas (i.e. between V1 and specialized visual areas such as the superior temporal cortex or visual association areas). In this sense, atypical autistic visual information processing, and probably, visually related abnormal behaviour manifested by persons with autism, may be related to low-level perceptual differences to a greater extent than previously believed (Belmonte *et al.*, 2004).

Enhanced simple versus impaired complex perceptual performance: specific to autism?

We have measured first and second order information processing along each visual pathway in order to successfully characterize the perceptual functioning in other neurodevelopmental conditions characterized by visually related symptoms (Habak and Faubert, 2000; Bertone *et al.*, 2003; Kogan *et al.*, 2004*a*). As shown in Table 2, such investigations using the same stimuli and experimental paradigm have resulted in different patterns of performance, specific to each condition and consistent with their respective phenotype. Therefore, a hypothesis regarding abnormal neural connectivity as differentiating autism from other conditions manifesting decreased complex motion sensitivity can be forwarded.

Additional support for this argument is evidenced in a recent study assessing both dynamic and static information processing in another type of developmental disorder, fragile X syndrome (FXS). In a study employing the orientation-discrimination task used in the present study, Kogan *et al.* (2004*a*) found a selective decrease in sensitivity for the second-order static condition for the FXS group (see Table 2). Based on this finding, Kogan *et al.* (2004*a*) suggest that in addition to pervasive deficits regarding motion processing in FXS (consistent with abnormal magnocellular neuropathology in FXS, see Kogan *et al.*, 2004*b*), integrative cortical dysfunction is also present in FXS, affecting both dynamic and static complex information processing in this condition. However, the finding of enhanced sensitivity to simple static information remains specific to autism. Conversely, decreased sensitivity to complex motion stimuli (i.e. global motion) has been demonstrated in at least a dozen other neurological disorders (Regan, 1991; Trick and Silverman, 1991; Gilmore *et al.*, 1994; Trick *et al.*, 1994, Cornelissen *et al.*, 1995, 1998; Atkinson *et al.*, 1997; Gunn *et al.*, 2002; Chen *et al.*, 2003; Mapstone *et al.*, 2003; Kogan *et al.*, 2004*b*; McKendrick and Badcock, 2004).

Table 2 Schematic representation of the sensitivity compared to control participants for normally-ageing persons (Habak and Faubert, 2000), persons with fragile X syndrome (FXS; Kogan *et al.*, 2004*a*) and HFA [dorsal (Bertone *et al.*, 2003) and present results] using the same task

	Normal ageing		FXS		HFA	
	Simple	Complex	Simple	Complex	Simple	Complex
Ventral	=	↓↓	=	↓↓	↑↑	↓↓
Dorsal	↓↓	↓↓	↓↓	↓↓	=	↓↓

Equal signs (=) and double arrows (↓↓, ↑↑) represent no difference and significant difference (respectively) in sensitivity between control and clinical groups at the $P = 0.05$ level.

Conclusion: integration within and between regions in autism

The present results are interpreted as behavioural evidence of altered 'local' neural networks in autism, possibly affecting the low-level processing of elementary stimulus features such as spatial frequency, orientation and contrast. Given the fact that these abnormal networks are the initial components of standard larger-scale networks responsible for higher-order information analysis, subsequent larger-scale networks integrating across specific stimulus features would also be modified in autism (McClelland, 2000; Grice *et al.*, 2001; Brock *et al.*, 2002; Bertone *et al.*, 2003; Just *et al.*, 2004). At least in the context of the present experimental paradigm, excessive lateral inhibition seems to be a promising candidate to account for the perceptual consequences of abnormal neural connectivity. This hypothesis is congruent both with superior visuo-static information processing and with neuro-integrative dysfunction. Other systems-based explanations have been forwarded to account for dichotomous abilities in autism for both perceptual (enhanced perceptual functioning, Mottron and Burack, 2001; temporal binding deficit hypothesis, Brock *et al.*, 2002) language (underconnectivity hypothesis; Just *et al.*, 2004) or relation between high and low level cognitive processes (weak coherence hypothesis, Frith 1989; reduced feed back flow of information, Frith 2003). Although different in respect of the purported synaptic dysfunction, these hypotheses predict impaired information processing if it is contingent on integrating information between specialized networks located in different brain regions and enhanced processing when limited within local networks. The current demonstration of both enhanced and diminished information processing in autism using the same task indicate that atypical connectivity can affect different levels of processing within the same 'local' network and is not necessarily contingent on reduced inter-network interactions.

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