Magnocellular visual evoked potential delay with high autism spectrum quotient yields a neural mechanism for altered perception

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Everyone has autistic characteristics to a greater or lesser degree, encapsulated in the Autism Spectrum Quotient, a scale that measures the degree to which an adult of normal intelligence displays traits associated with autism spectrum disorders. Recent psychophysical analyses of autism spectrum disorders point to superior local processing, and impaired or ignored global and contextual processing. The aim of this study was to test whether low- and high-scoring individuals on the Autism Spectrum Quotient differ on a measure of local and global processing, motion processing and visual pathway integrity. Fifteen low-scoring individuals and 14 high-scoring individuals derived from a normal population participated in the study. The results indicate that the initial cortical response to the magnocellular afferents is weaker at low contrast in the high autistic tendency group and that a second-order response, reflecting magnocellular activity, demonstrated a delay for high versus low scorers when the parvocellular pathway was also active in response to a high contrast stimulus. High-scoring individuals also demonstrated difficulty in identifying the global components of locally salient hierarchical Navon figures. Furthermore, cross-validated discriminant analysis, using four physiologically and three psychophysically derived parameters, correctly classified 83% of individuals who scored either high or low on the Autism Spectrum Quotient. These findings in the group scoring high on the Autism Spectrum Quotient indicate that a delay in primary visual/prestriate cortical processing of magnocellular input diminishes the advantage of its early arrival to primary visual cortex. This appears to be associated with impaired global visual perception, predicting with high accuracy behavioural tendencies associated with autism spectrum disorders. It has been proposed that perceptual impairment in autism may be attributed to a dysfunction of horizontal connections within early visual areas, presumably parvocellular in nature. However, the timing of such form processing aberrations is much later than the timing of abnormal magnocellular visual processing measured directly here. Thus it is proposed that a magnocellular processing delay decreases the ability of autistic individuals to benefit perceptually from feedback normally associated with the magnocellular advantage.

Keywords: autism; Asperger’s disorder; psychophysiology; VEPs; visual cortex
Abbreviations: AQ = Autism Spectrum Quotient; K1 = first-order kernel; K2.1 = first slice of second-order kernel; K2.2 = second slice of second-order kernel; V1 = primary visual cortex; VEP = visual evoked potentials

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Introduction

Autism spectrum disorders have provided challenges for parents, educators and scientists alike, in trying to understand the nature of this debilitating social and behavioural problem of human development. The manifestations of autism spectrum disorders include repetitive actions such as hand flapping or rocking, odd social communication, a narrow focus of interest and resistance to change (DSM-IV-TR, 2000). Autism spectrum disorders have a multifactorial inheritance structure, suggesting a complex interaction between genetic and environmental factors (Persico and Bourgeron, 2006).

While autism has tended to take on a certain popular appeal through association with rare savantism, as typified by the movie ‘Rain Man’, it is more normally associated with some level of intellectual dysfunction. However, certain perceptual skills exhibited by those with autism spectrum disorders exceed normal populations matched for mental ability. These include superior performance in recognition of hidden figures (Shah and Frith, 1983) and intellectual dysfunction. However, certain perceptual skills exhibited through association with rare savantism, as typified by the movie ‘Rain Man’, it is more normally associated with some level of intellectual dysfunction. However, certain perceptual skills exhibited by those with autism spectrum disorders exceed normal populations matched for mental ability. These include superior performance in recognition of hidden figures (Shah and Frith, 1983) and social communication, a narrow focus of interest and resistance to change (DSM-IV-TR, 2000). Autism spectrum disorders have a multifactorial inheritance structure, suggesting a complex interaction between genetic and environmental factors (Persico and Bourgeron, 2006).

The observation of deficits in global identification of stimuli and in processing motion has led to the suggestion that there may be a magnocellular visual pathway deficit in autism spectrum disorders (Plaisted et al., 1999; Spencer et al., 2000; Milne et al., 2002; Bolte et al., 2007). Magnocellular pathway neurons are relatively more sensitive to moving stimuli and processing lower spatial frequencies, and less sensitive to chromatic stimuli than are parvocellular neurons (Merigan and Maunsell, 1993). Furthermore, the magnocellular pathway is more sensitive to stimuli of low contrast than is the parvocellular pathway (Livingstone and Hubel, 1988) and has a faster conduction speed than the parvocellular pathway (Schroeder et al., 1989; Maunsell and Gibson, 1992; Maunsell et al., 1999). Thus, a magnocellular deficit hypothesis for autism spectrum disorders appears plausible. However, the finding of normal flicker contrast sensitivity in children or young adults with high functioning autism (Bertone et al., 2005; Pellicano et al., 2005) caused Dakin and Frith (2005), in reviewing the underlying basis of autism, to propose a neural locus beyond low-level cortical motion processing areas—perhaps within the motion sensitive system of the superior temporal sulcus. However, direct physiological measures of neural function in autism have been largely related to neuroimaging studies of social communication and intention (Pelphrey et al., 2005; Dapretto et al., 2006; Just et al., 2007), rather than towards early perceptual processing.

A second neuroscientific approach to the altered perceptual capability in autism spectrum disorders has come from theories developed from primate studies of the rapidity of processing of form information throughout the brain (reviewed in Lamme and Roelfsema, 2000). Stimuli used in such studies have been typically second-order, employing texture rather than luminance contrast to specify boundaries. These studies have evolved into an elegant theoretical basis for perceptual grouping (Roelfsema, 2006) that includes mechanisms of feed-forward, horizontal and feedback (recurrent) processing, with the idea that feed-forward processing is immediate with horizontal and recurrent processing occurring after a delay, and have concentrated typically on visual processing in the ventral cortical stream. When applied to the study of autism spectrum disorders, EEG data suggest a specific impairment in object boundary detection, present as early as 120 ms after stimulus delivery (Vandenbroucke et al., 2008).

Such theoretical ideas have ignored the more rapid processing of visual information by the magnocellular pathway. In humans, the latency of the magnocellular response in primary visual cortex is 25–45 ms earlier than the parvocellular response (Baseler et al., 1994; Klistorner et al., 1997; reviewed Laycock et al., 2008), conforming with the idea of a temporal ‘magnocellular advantage’ (Bullier, 2001; Laycock et al., 2007), with the notion that there is sufficient time for signals from the primary visual cortex (V1) to feed forward to visual area V5 (middle temporal) and the early dorsal stream rapidly enough that feedback to V1 or prefrontal cortex (V2) can occur prior to, or coincident with, parvocellular pathway information arrival. Indeed, cooling and lesion experiments, inactivating area V5/MT+ in monkeys, demonstrate a role for feedback in foreground background segregation and in boosting the gain of the centre mechanism of receptive fields (Bullier, 2001; Super and Lamme, 2007).

Our study was designed to evaluate whether magnocellular versus parvocellular, global versus local and dorsal versus ventral processing exhibit systematic differences between those with high and low scores on the AQ from a normal population. We used non-linear visual evoked potentials (VEP), employing a pseudo-random binary stimulus sequence to derive, simultaneously, components sensitive to magnocellular and parvocellular pathway function (Klistorner et al., 1997), on the basis of similarity of contrast response functions to those recorded in primate and on the basis of latencies. We also applied measures of motion coherence threshold (random dot kinematogram) under two conditions where individual dots were either easy or hard to track. Finally we addressed aspects of global versus local object recognition with interference to either the global or local aspect of hierarchical figures. We expected that individuals with high AQ scores would show a physiologically defined magnocellular deficit when compared with low AQ scorers, and would also perform relatively poorly on psychophysical tasks involving global attention and coherent motion threshold determination.
Material and methods

Subjects

Participants were recruited through an online version (Opinio, ObjectPlanet, Inc. Oslo, Norway) of the AQ scale. The AQ was completed by 129 respondents online and those who scored 11 or below (range 4–11) and 20 or above (range 20–34) were recruited for the low (n = 15) and high (n = 14) scoring groups, respectively. The online version was a true representation of that used by Baron-Cohen et al. (2001) and the scoring procedure is outlined in this article. The only difference to the paper version was that participants were required to click a button rather than circle their response. A 30 min speeded version of the Raven’s Advanced Progressive Matrices Test (Raven, 2003) was administered to participants to test non-verbal intelligence. No difference was found between the high and low AQ groups on this measure (P > 0.05). All participants had normal or corrected-to-normal vision and there were no differences between the two groups in the amount of individuals who wore glasses. Participants were excluded if they had been diagnosed with neurological conditions such as epilepsy, or an attentional deficit.

Psychophysical procedure

Participants completed a salience-biased local/global Navon figures task (Navon, 1977) and a motion processing task. In the Navon figures task, participants were instructed to attend to either the local or global level of a locally or globally salient Navon figure and identify the letter at that level. Participants would press the space bar once they had mentally identified the letter at the target level and a reaction time to this button press was recorded. Participants were then required to identify the letter at the target level on the keyboard in order to measure accuracy. Reaction time and accuracy to target letter identification was recorded. The Navon figures were generated using Mathematica (Wolfram Research, version 6), with the global letters subtending 3.0 × 2.0° and the local forms subtending 0.3 × 0.2°, when viewed at 57 cm distance. The figures were made of a combination of the following letters: K, D, N, P, C, S, V, H, E, T and F. The globally salient forms were created by a 2.0 pixel Gaussian blur filter (Photoshop, Adobe Software, v9.0), while locally salient letters were created by randomly colouring the local letters either red or white (Fig. 3A). Using VPixx software, an attention task level (global or local) was first presented, followed after a delay of 1 s by the Navon figure. This remained on-screen until the spacebar was pressed, the target letter then being identified by the participant, with reaction time to spacebar press and letter identification performance logged. This task involved 60 trials, randomized in presentation.

In the motion processing task, participants were presented with random dot kinematograms consisting of either limited lifetime dots or infinite lifetime dots that were moving either up, down, left or right. Participants were instructed to press the arrow key that corresponded to the direction of motion of the dots. Motion coherence thresholds were estimated using the Parametric Estimation by Sequential Testing (VPREST) adaptive staircase procedure. The limited lifetime dots only remained on the screen for 10 frames (100 ms) after which time another dot would appear at a different location. This technique was used to make it difficult for individuals to track individual dots, forcing participants to process the overall motion globally. The infinite lifetime dots remained on the screen for the entire time it took for the dot to travel from one side of the circle to the other, allowing for local tracking.

Psychophysics—data analysis

Reaction time divided by accuracy was used as the dependent variable in the Navon figures task to control for a trade-off between accuracy and reaction time (Mevorach et al., 2006). A mixed design ANOVA with score (high versus low) as the between-subjects factor and target level identity (global versus local), congruency (congruent versus incongruent) and saliency (globally salient versus locally salient) as within-subjects factors, was conducted (using SPSS v16.0).

As the motion task data were not normally distributed, we used the appropriate non-parametric statistical analyses (Mann-Whitney U-test) on the ratio between limited and infinite lifetime motion thresholds.

EEG—recording and data analysis

We recorded non-linear achromatic multifocal VEPs over occipital cortex (electrodes placed at International 10/20 standard positions Oz, Fz and with a left ear ground) from both high and low AQ scorers, using binary pseudorandom stimulus sequences (VERIS, version 3.01, EDI, San Mateo, USA).

The interpretation of the Wiener kernel expansion of the VEP is well explained in the article by Sutter (2000). Briefly, the response to each stimulus patch can be extracted because the stimulus sequences for each patch are mutually decorrelated. Thus, if the stimulus was a binary black/white exchange, then the first-order kernel K1 is simply the sum of all responses to white R_W minus the sum of all responses to black R_B throughout the pseudorandom stimulus sequence, i.e. $K_1 = R_W - R_B$.

Similarly, the second-order response takes account of the history of stimulation and the first slice of the second-order response (temporal non-linearity) is $K_2.1 = R_{WW} + R_{BB} - R_{WB} - R_{BW}$, where $R_{WW}$ is the sum of all responses where there are two white stimulus frames in a row, $R_{BB}$ is the sum of all responses where a white frame is followed by a black frame, and so on. Thus the first slice measures neural recovery within the duration of one frame, or 13 ms. The second slice includes an intervening frame of either polarity, and thus measures recovery within two frames, or 27 ms. The Wiener kernel expansion is an infinite series; however, for the VEP recorded under conditions such as this, most energy is found in $K_1$, $K_2.1$ and $K_2.2$, i.e. the first-order and first two slices of the second-order response (Klistorner et al., 1997).

The kernels of the multifocal non-linear VEP show a Wiener kernel structure that separates the inputs of the magnocellular and parvocellular pathways along the dimension of interaction time in slices of the second-order kernel (Klistorner et al., 1997), mimicking the contrast response functions of magnocellular and parvocellular neurons in primate lateral geniculate nucleus. The first slice in the second-order response (K2.1) with shorter response latency is dominated by magnocellular input, while the second slice (K2.2) is dominated by parvocellular activity with a small contribution from the magnocellular pathway. A 19 element cortically scaled hexagonal unstructured multifocal stimulus (without contour) within was presented using VERIS software (EDI, San Mateo, USA, version 3), using a 75 Hz frame rate CRT monitor. The signal was amplified 100 000 times and band-pass filtered between 3 Hz and 1 kHz. The data sampling rate was 1 kHz. Stimulus hexagons were achromatic and were either presented at 24 or 96% temporal contrast ($L_1 - L_2/L_1 + L_2$), where $L_1$ and $L_2$ are the luminances used in the binary stimulus sequences) around a mean luminance of 65 cd/m² in a pseudorandom binary pattern.
m-sequences lasting 4 min. While all 19 hexagons were stimulated, only the responses recorded from the central stimulus patch were analysed, owing to the high cortical magnification of the foveal response and to the variable efficacy of the generators in the folded cortical brain structure contributing to VEP (Klistorner et al., 1997).

Kernels of the VEPs were extracted using the VERIS program. IGOR Pro (Wavemetrics, Lake Oswego) was then used to extract amplitudes and latencies of peaks within the VEPs, with the window settings for each of these peaks established after visually analysing the data (e.g. for the K2.1 high contrast response, the window setting for the P1 was 75–125 ms, in order to include all participants’ major positivities). Grand mean average waveforms with standard error waves were calculated. A wave of t-values was calculated and plotted together with significance thresholds of t = ±1.96.

Results

Magnocellular and parvocellular pathway activity

For the high AQ group, a significant delay was found in the peak of the non-linear VEP component that reflects magnocellular activity (first slice of the second-order kernel, K2.1; Klistorner et al., 1997), recorded using high contrast (96%) stimuli (Fig. 2A). The onset of interference in the peak of the magnocellular response (Fig. 1D) coincides well with the arrival of parvocellular pathway response in visual cortex (around 75 ms, Fig. 1D, second arrow), but also with estimates of the arrival of feedback to V1, following magnocellular feed-forward projections to V5/MT+ (Laycock et al., 2007). At lower contrast (24%), where parvocellular generated activity is relatively weaker (Shapley, 1990) (cf. Fig. 1C and D), such a peak latency difference was not observed in the mean waves generated by the magnocellular pathway (Fig. 1A). Significant VEP differences between the high and low AQ groups were also found in the first-order response (K1).

While the first-order response (K1) contains both magnocellular- and parvocellular pathway contributions (Klistorner et al., 1997), the earliest part of this response is likely to be magnocellular in origin (Maunsell and Gibson, 1992), indicating that the magnocellular contribution to K1 (but not K2.1) at low contrast is weaker for the high AQ compared with the low AQ group. The mean ratio of the parvocellular derived peak-to-peak (K2.2: N2/P2) amplitude at high contrast to the magnocellular derived peak-to-peak (K2.1: N1/P1) amplitude at low contrast (Fig. 2F) was also higher for the high AQ group (t-test, P = 0.018).

There is a possibility that the abnormal high contrast K2.1 response observed with the high AQ population was due to an intrusion of the parvocellular waveform (normally dominating the second slice—K2.2 kernel) into the first slice (K2.1). This idea was modelled by adding to the K2.1 (low AQ) waveform sufficient of the K2.2 (low AQ) necessary to replicate the sudden deviation between low and high AQ K2.1 waveforms observed at around 80 ms. It is clear that this model is reasonably successful in characterizing the subsequent 10 ms (Fig. 2A, comparing the red and black waveforms). Thereafter, however, the model diverges from the waveform that it is supposed to replicate by as much as the high AQ data diverge from the low AQ data.

While the statistical monitoring of the mean waveforms is indicative of a manifest population difference between high and low AQ, individual waveforms also show such a deviant behaviour, with high AQ individuals showing this abnormal, delayed, high contrast K2.1 response, a feature not observed in low AQ individuals (Fig. 2B). These individual waveforms were selected to represent the variation in waves recorded across the two groups most accurately.

Performance on local and global salient Navon figures

The high AQ group had relative difficulty ignoring the incongruent Navon local level in local salient trials (Fig. 3B) when compared to the low AQ group. A mixed design ANOVA indicated a significant interaction between target level identity, saliency and score [F(1,27) = 9.20, P < 0.01]. Such a statistical interaction was not found for global salient identification [F(3, 1) = 0.20, P = 0.889], although repeated measures analysis showed that blurring the Navon figures was effective, with local identification more difficult, as evidenced by ~110 ms on average more than global identification [F(1,27) = 27.8, P < 0.001], while incongruent identification was more difficult than congruent by approximately 65 ms [F(1,27) = 8.6, P = 0.007].

Coherent motion thresholds

Motion coherence thresholds, with either infinite lifetime or limited lifetime dots, were measured in order to assess whether the high AQ group tended to track individual dot motions (made difficult by the limited life condition).

Consistent with findings from studies of autistic groups, mixed results were found in terms of motion coherence performance, with mean thresholds lower for the high AQ group than the low AQ group for infinite lifetime dots and with the reverse for limited lifetime dots (Fig. 4B). Due to non-normal distribution of the data, non-parametric analysis of the ratios of limited to infinite lifetime thresholds (Fig. 4B) was performed. This showed a trend towards impaired motion processing performance for the high AQ compared with the low AQ group (Mann–Whitney U-test; P = 0.067).

Discriminant analysis

The degree to which the physiological and psychophysical data could predict membership of the high and low AQ groups was ascertained using discriminant analysis (Jmp software, version 8).

All 29 participants could be correctly identified in terms of their AQ score using four VEP parameters [amplitude of the first-order N1 response negativity for 24 and 96% contrast, latency at high contrast for K2.1 response, amplitude of the second negativity (N2) of the 96% contrast K2.2 response] and three psychophysical parameters (reaction time divided by accuracy for local identification of congruent locally salient forms; reaction time divided by accuracy for local identification of incongruent globally salient forms from the Navon figure task; and coherent motion thresholds for limited lifetime dot displays). See Table 1 for details of the parameters and their canonical coefficients.
The canonical correlation for this selection of predictors was 0.853 [Wilks’ lambda = 0.273, F(7,21) = 7.99, P < 0.0001]. The robustness of this analysis was checked using the ‘leave-one-out’ cross-validation technique where the probability of prediction for each participant is determined from \((n-1)\) remaining sample members. Under these conditions, 83% of the high and low AQ sample were correctly predicted with individual probability values of >90% found in 21 of the 29 participants (Fig. 5).

**Discussion**

The results suggest that autistic tendency, as measured by the AQ, relates to parameters objectively derived from early visual processing. What is remarkable is that none of the 50 items of the AQ scale relate to vision *per se*. However, using a mixture of electrophysiological recording (VEP) and psychometric reaction time testing for speed of identification of global and local forms, particularly those in which the global content has been impaired,
it is possible to predict validly class membership for individuals high or low on the AQ scale with a high rate of reliability (83% correct). In addition to the development of a biomarker for autistic tendency, our understanding of the neuronal mechanisms underlying autistic perception, particularly with respect to the question of a magnocellular function deficit has been improved. The physiological data suggest that the initial cortical response to the magnocellular afferents is weaker at low contrast in the high AQ group. In addition, we found a delay in the completion of magnocellular processing in occipital cortex for stimuli of high contrast, providing a novel neural mechanism for the explanation of perceptual abnormalities. Both of these findings are characteristic of a system with a greater degree of non-linear response, possibly corresponding to a lower flicker fusion frequency for the magnocellular system for the high AQ group. Taken together with the lack of difference in $K_1$ at high contrast between low and high AQ groups, it is also possible that there is a greater threshold excitatory post-synaptic potential required to fire the magnocellular recipient neurons of striate cortex in the high AQ group.

Strong evidence now exists indicating that the dorsal pathway is capable of feeding back information into the primary visual cortex from higher cortical areas (reviewed in Bullier, 2001; Laycock et al., 2007). Processing in the magnocellular pathway is rapid enough for feedback to V1/V2 to coalesce with afferent parvocellular information. This early arrival of magnocellular pathway (compared with parvocellular) information to the striate cortex (10–20 ms in the primate literature: Maunsell and Gibson, 1992; Maunsell et al., 1999; and 25–45 ms in the human multifocal VEP: Baseler and Sutter, 1997; Klistorner et al., 1997) has been termed ‘the magnocellular advantage’ by Laycock et al. (2007). In the current study (see arrows in Fig. 1C and D), the initial parvocellular response starts 30 ms after the magnocellular response. The magnocellular advantage is thought to be important for integrating global information retroinjected from early dorsal stream cortical areas to the ‘active blackboard’ visual areas V1 and V2 (Bullier, 2001), allowing for integration of visual signals for ventral stream recognition processes. Thus, a magnocellular processing delay in autism could lead to the disruption of the magnocellular advantage seen in normal individuals (without strong autistic tendency).

Our recordings show that the arrival times of magnocellular and parvocellular afferent pathways to cortex are very similar for the high and low AQ, but the deviant waveforms develop in the P1 positivity of the K2.1 kernel response. Such recurrent, but fast, magnocellular pathway inputs have not been given a special place in other successful models of cortical form processing that emphasize the ‘feedforward sweep’ (Lamme and Roelfsema, 2000; Roelfsema, 2006) of the first spikes arriving at successive ventral stream areas. Recent findings of a specific impairment in object boundary detection in autism spectrum disorders (Vandenbroucke et al., 2008) have been attributed to a (red) with low contrast indicated by a thin stroke and high contrast by a thicker stroke. The delay that is seen in the grand mean average for high contrast stimulation is also apparent in the high AQ individual waveforms, but not to any great extent in those from low AQ individuals.

Figure 2 Modelling parvocellular intrusion and individual waveforms. (A) The possibility that the abnormal $K_{2.1}$ kernel response at high contrast was due to an intrusion of the parvocellular response $K_{2.2}$ waveform was modelled by fitting a linear combination of $K_{2.1}$ (low AQ) and $K_{2.2}$ (low AQ). While such a model reproduces the deviation between the high and low AQ participants in $K_{2.1}$ at around 80 ms, it is clear that the model (black curve) only fits well between prior to 90 ms before deviating from the high AQ (red) waveform. (B) Magnocellular VEPs recorded from individual participants, the upper four sets of traces with low AQ (green) and the lower four with high AQ.
Figure 3 Task and behavioural data. (A) Stimuli used in the global/local processing task that used hierarchical Navon figures involved three conditions including target level identity, congruency and saliency. Participants were required to identify the letter at the local or global level of each stimulus. Local salient Navon figures had impaired grouping characteristics rendering global identification more difficult, while the global salient letters were blurred, making local recognition more difficult (Mevorach et al., 2006). (B and C) Reaction time moderated by accuracy (RT/Acc) for each condition (mean ± SE) indicates that the high AQ group had difficulty in withdrawing attention from the local level of the local salient stimuli when having to identify the (incongruent) global level.

Figure 4 Motion performance. (A) Whilst both high and low scorers found the limited lifetime condition to be more difficult than the infinite lifetime condition, this difference was more pronounced in the high AQ individuals, although not significantly so. (B) Motion threshold ratio (limited lifetime threshold/ infinite lifetime threshold) indicated a relative difficulty in dealing with the limited lifetime dots for the high AQ group.

Table 1 Parameters used in discriminant analysis (in order of entry), together with the canonical coefficients derived from the classification

<table>
<thead>
<tr>
<th>Parameter</th>
<th>F-ratio</th>
<th>Canonical coefficient</th>
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<tr>
<td>$K_{1,024},N_{1,amp}$</td>
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<td>2.269 484</td>
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<tr>
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<td>−3.97600</td>
</tr>
<tr>
<td>LL_glob_RT/acc</td>
<td>5.21</td>
<td>4.016631</td>
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RT = reaction time.
The interpretation of our data could have implications for the major cognitive models of abnormal perception—the Weak Central Coherence model (Happe and Frith, 2006) and the Enhanced Perceptual Function model (Mottron et al., 2006), arguing, respectively, that the dorsal cortical stream pathway is either weaker, or that there is a superiority of low-level functioning. The parvocellular system, more dominant at the higher contrast has not been reported.

While our study contrasted high and low AQ groups, there is no manifest reason why the results should not be directly applicable to populations with even higher AQ scores—crossing the threshold for diagnosis as high functioning autism or Asperger’s syndrome (Bayliss and Tipper, 2005; Stewart et al., 2009; Grinter et al., 2009a, b). However, limitations on population size in this study would suggest generalization across the broad autistic spectrum prior to making claims relating to the clinical phenotype. Whether an early cortical problem such as a ‘magnocellular disadvantage’ could substantially contribute to the core deficits of autistic behaviour is debatable. However, we would argue that if the ventral cortical stream, deprived of global information such as figure/background segmentation, and relying largely on parvocellular input, is continually exerting priority in situations of high pattern complexity or contrast during development, then cognitive sequelae such as inappropriate visual perceptions within dynamic social interaction are a logical outcome.

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References


