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The role of MT+/V5 during biological motion perception in Asperger Syndrome: An fMRI study

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Abstract

Asperger Syndrome (AS), a condition on the autistic spectrum, is characterized by deficits in the ability to use social cues to infer mental state information. Few studies have examined whether these deficits might be understood in terms of differences in visual information processing. The present study employed functional magnetic resonance imaging to examine differences in brain activity among individuals with AS while performing a task that typically leads to the automatic interpretation of human movement. Despite similar behavioural performance, significantly less activity was found for the AS group (relative to a control group) in inferior, middle and superior temporal regions, including the human analogue of MT+/V5. These data suggest that AS is associated with unique patterns of brain activity during the perception of visually presented social cues.

Keywords: Asperger Syndrome; Autism; fMRI; Motion perception; MT+/V5; Temporal lobe

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1. Biological motion, brain function, and autism-spectrum disorders

One of the most salient characteristics of autism-spectrum conditions is the inability to successfully process socially relevant information (Bailey, Phillips, & Rutter, 1996; Baron-Cohen, 1995). A major focus among scientists studying autism and Asperger Syndrome (AS) has been to develop theories to explain this disability. One such theory holds that individuals with autism-spectrum conditions possess a specific deficit in the ability to understand mental states (referred to as a theory of mind [ToM] deficit; Baron-Cohen, Tager-Flusberg, & Cohen, 2000a). While deficits in ToM abilities have been reasonably well established in autism (Baron-Cohen & Ring, et al., 2000b), one possibility is that deficits in comprehending socially relevant information might stem from more fundamental *perceptual* deficits involved in decoding social cues.

1.1. Biological motion perception and the brain

Humans appear to be equipped to rapidly recognize animate or biological movement, independent of other visual information processes. A key test of this ability consists of the perception of a moving human entity in the absence of any visual cues that would, when stationary, indicate that the form is human (Johansson, 1973). Using a technique where the human form is represented by a configuration of 11 dots placed on the major joints of the body (called a point-light figure), Johansson showed that individuals could only identify the assembly of dots as human when it was set in motion. This finding supports the theory that certain stimulus categories can be best identified though the visual processing of motion.

Not surprisingly, there appears to be substantial overlap between brain regions involved in biological motion perception and other visual motion processes (such as global motion perception; Singh, Barnes, Hillebrand, Forde, & Williams, 2002). However, biological motion perception appears to rely on some neural circuits that are distinct from those implemented in more general motion processes (Singh, Barnes, & Hillebrand, 2001). Portions of the MT+/V5 region in particular appear to be involved in the processing of biological motion, independent from their role in general movement perception (Vaina, Lemay, Bienfang, Choi, & Nakayama, 2001; Zeki et al., 1991). Recent findings on biological motion perception suggest a level of heterogeneity in MT+/V5 that has not been fully accounted for by most neuropsychological models of motion perception.

Other temporal lobe regions also play a role in the processing of biological motion (Grezes, Costes, & Decety, 1998; Grezes & Decety, 2001; Grossman et al., 2000). Superior temporal regions in particular have an important role in processing the human form (Allison, Puce, & McCarthy, 2000; Bonda, Petrides, Frey, & Evans, 1995; Grezes et al., 1998). Studies have also highlighted the role of inferior temporal and extrastriate regions in biological motion processing. Fusiform regions appear to be implemented in some biological motion processing tasks (Bonda, Petrides, & Evans, 1996; Singh, Williams, & Smith, 2000; Singh et al., 2001). Together, these studies suggest that the analysis of biological motion involves a network of posterior brain regions, encompassing the human analogue of MT+/V5, superior and middle temporal gyri, and the fusiform gyrus.

Findings regarding the possible role of frontal regions in biological motion processing have been less consistent. In one study, increased activity was found in dorsolateral prefrontal cortex (Singh et al., 2000, 2001). Grezes and Decety (2001) reported increased activation in orbital and mesial frontal areas when contrasting a point-light figure condition to a condition in which the point-light figures were inverted. However, these reported activations were not consistent across all contrasts involving upright biological motion perception (Grezes & Decety, 2001). Studies by Howard et al. (1996), Grossman et al. (2000), and Bonda et al. (1996) did not report any significant activations in frontal regions when contrasting biological motion conditions to comparison conditions.

1.2. Biological motion perception in autism spectrum conditions

Individuals with autism spectrum conditions demonstrate significant differences in understanding socially meaningful information — differences related to specific patterns of neuropsychological function (Baron-Cohen & Ring, et al., 2000b). Functional imaging studies examining social intelligence in autism within the general framework of ToM (Baron-Cohen & Ring, et al., 2000b) have focused on locating areas of the brain that may demonstrate modularity in that domain of cognitive function (Baron-Cohen et al., 1994, 1999; Frith & Frith, 2000; Happé et al., 1996). Much of this research has focused on the possible role of frontal and subcortical (e.g., amygdala) regions in mentalising (Happé et al., 1996).

One prior study has examined biological motion perception among individuals with autism spectrum conditions (Blake, Turner, Smoski, Pozdol, & Stone, 2003). Blake et al. (2003) reported that a sample of individuals with autism performed significantly poorer on a task of biological motion perception than a non-autistic sample. Several studies using variants of the classic Heider and Simmel (1944) paradigm have shown that, normal volunteers spontaneously interpret the apparently animate motion of geometic shapes as having goals, desires, intentions, and thoughts, whereas adults with AS have been shown to mentalize to a lesser degree, or if anything, to systemize such motion (Bowler & Thommen 2000; Castelli, Frith, Happe, & Frith, 2002; Klin, Jones, Schultz, & Volkmar, 2003). Note that such low-level abnormalities in the perception of biological motion are not incompatible with a higher-level ToM deficit, since in one key model of the mindreading system, ToM is a relatively developmentally late outcome stemming from more fundamental perceptual processes such as the ‘intentionality detector’ (ID) or an eye direction detector (EDD) (Baron-Cohen, 1995; Baron-Cohen & Ring, 1994).

A number of studies have found differences in temporal lobe structure and function in people with autism spectrum conditions, occasionally including areas involved in biological motion perception. Bolton and Griffiths (1997), examining a sample of individuals with tuberous sclerosis, found an association between the number of tubers located in the temporal lobes and the diagnosis of autism. Schultz et al. (2000), examining a mixed group of individuals with autism and AS, found a significant activity decrease in the right fusiform gyrus during a face matching task relative to a control group. Critchley et al. (2000) also found deficits in fusiform activation among individuals with Asperger Syndrome in a task involving facial affect recognition.

In the present study, we hypothesized that, when performing a task involving biological motion perception, individuals with AS would show significantly less brain activity than non-AS individuals in a number of areas related to the processing of human movement — specifically, the fusiform, middle and superior temporal gyri, including the human analogue of MT+/V5. Furthermore, as a number of recent studies on both AS and non-AS populations have found differences in brain activity in inferior and left medial frontal regions during tasks requiring social intelligence, we predicted that we would also see activation differences in these areas (Frith & Frith, 1999; Klin, Schultz, & Cohen, 2000; Singh et al., 2001).

2. Method

2.1. Participants

Twenty male participants – 10 with a diagnosis of AS and 10 controls (C) – were recruited for participation in the present study. Individuals in the AS group all received a diagnosis using DSM-IV (APA, 1994) and ICD-10 (World Health Organization, 1994), criteria. The mean and standard deviations were 27.6 (7.1) and 25.6 (4.9) years of age for the AS and C groups, respectively. All participants were right-handed. None of the participants reported taking any medication currently or having any history of neurological insult. Participants' consent was obtained according to the Declaration of Helsinki (World Health Organization, 1991). This study was approved both by the Ethical Committee of the Institute of Psychiatry (University of London) and the Local Research Ethics Committee (LREC) at Addenbrooke's Hospital Clinical School (University of Cambridge).

2.2. Experimental design and procedure

Participants performed two separate experiments during the MRI procedure; only the data from the first paper are examined in the current paper. Seven separate animation sequences were developed for each of seven experimental conditions. Data from two of these sequences are examined in this paper. The presentation order of the two experiments was counterbalanced across the two groups. In order to confirm that the two groups were matched in their overall pattern of intellectual abilities, nine individuals in the AS group and seven individuals in the C group were administered the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). See Table 1 for IQ information for both groups.[1]

The experimental task followed an AB boxcar design in which blocks of experimental trials alternated with fixation periods. Each experimental condition block contained 12 trials, each lasting 2 s (1 s trial presentation time, 1 s inter-stimulus interval). The presentation order of all animation sequences was generated randomly for each participant. During the biological motion discrimination condition, 13 dots appeared on a back-projected visual display in the configuration of a human form, with each dot placed over a major joint. Using the Cutting human movement algorithm

Table 1
Descriptive information for Asperger Syndrome and control groups

Group	*N*	Age, mean (S.D.)	Full-scale IQ[a] (FSIQ)	Verbal IQ[a] (VIQ)	Performance IQ[a] (PIQ)
Asperger Syndrome	10	27.6 (7.1)	109	101	115
Controls	10	25.6 (4.8)	119	114	120

Note: FSIQ, VIQ and PIQ were taken from the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). Two-tailed independent-samples *t*-tests were performed on each descriptive measure. No significant differences were found between groups [$ts(14) < 1.73$; ns].

[a] Data were missing for three subjects in the normal control group and one subject in the Asperger Syndrome group.

(Cutting, 1978), each dot was set in motion to give the appearance of a figure walking ''on the spot'' — stationary in the center of the visual display. Participants indicated via response box whether the figure appeared to be walking either to the left or the right side of the screen.

The same stimuli were used for the randomized biological motion condition, except that each of the 13 dots was displaced by a maximum of 65 pixels in the vertical dimension. The amount of displacement was determined randomly by the experiment program for each individual trial. This displacement had the effect of perturbing the visual percept of the walking figure, such that the randomised stimuli were less easily identifiable as resembling a human form. However, the configuration of the walking human form was still sufficiently preserved to allow both groups of participants to guess the walking direction with accuracy levels above chance. This condition was chosen in order to provide a contrast that closely matched the non-randomized biological motion condition in terms of the translational motion and cognitive/visual information processing demands of the stimuli, but lacking in the presentation of fully coherent human movement.

Response data for all conditions were recorded via a two-button response box. In order to measure the relative ability of participants in the AS and C groups in completing each of the experimental tasks, accuracy scores were computed for each experimental run by dividing the number of correct responses by the number of trials administered.

2.3. fMRI data acquisition

Gradient-echo echoplanar imaging (EPI) data were acquired at 1.5 Tesla (T) using a GE LX-NV/CV system equipped with ultra-fast SR150 field gradients allowing a maximum gradient amplitude of 40 mT/m (General Electric, Milwaukee WI). For each activation experiment, 16 slices of functional MRI data were acquired parallel to the anterior commisure/posterior commisure plane using the following parameters: repetition time (TR) = 2000 ms; number of repetitions = 96; echo time (TE) = 40 ms; flip angle = 90°; slice thickness = 7 mm; interslice gap = .7 mm; matrix size = 64 × 64. To facilitate later registration of fMRI data into standard space, a higher resolution EPI dataset comprising 43 near-axial slices was also acquired using the following parameters: TR = 6000 ms;

TE = 40 ms; inversion time (TI) = 1500 ms; flip angle = 90°; slice thickness = 3 mm; interslice gap = .3 mm; matrix size = 128 × 128.

2.4. Data analysis

2.4.1. Response data

Experimental task performance was examined by calculating an average accuracy score across all responses within both conditions. In order to compare performance across the AS and C groups, two-tailed independent-samples *t*-tests were run, comparing the mean accuracy for each of condition.

2.4.2. Functional MRI data

Statistical analyses were implemented using the brain activation and morphological mapping software package (Bullmore et al., 1999). All functional images were corrected for participant motion by applying a rigid body transformation of each functional image onto a mean image.

Linear regression was used to estimate signal changes in response to our experimental manipulation (Bullmore et al., 1999, 2001). Regression analysis modeled the contrast between experimental conditions after each contrast was convolved with a pair of Poisson kernels ($l = 4$ and 8 s) to model local hemodynamic response functions. The resulting statistical maps were registered to the standard space of Talairach and Tornoux (1988) using an affine transformation to a template image.

For between-group analysis, an analysis of variance (ANOVA) model was fitted at each intracerebral voxel to data acquired from subjects in both the AS and C groups. Cluster level analysis involved the application of a preliminary probability threshold ($p < .05$) to the ANOVA-derived statistical maps and setting all subthreshold voxels to zero. Significant groups effects were identified by summing the values of the suprathreshold voxel statistics statistics (the ''mass'' of each cluster) and implementing a cluster-level permutation test. As brain activity in relation to our stimulus manipulation was measured individually for each participant, and then tested for between-group effects in a second-level analysis that explicitly incorporates inter-participant variability, between-groups analyses modeled random effects.

Our permutation testing procedure provides a stringent criterion for statistical significance. *p*-values were selected for statistical significance thresholds such that less than one false positive cluster would be expected over all clusters tested in each map. This corresponded to a threshold of $p < .004$ for the walker condition, and $p < .006$ for the randomised walker condition.

3. Results

3.1. Behavioural response data

See Table 2 for group-wise means and standard deviations of the response accuracy for each condition. Independent-sample *t*-tests revealed no significant differences between

Table 2
Experimental task accuracy

Group	Walker condition, mean (S.D.)	Randomised walker condition, mean (S.D.)
Asperger Syndrome	.99 (.02)	.66 (.16)
Controls	.99 (.01)	.77 (.07)

Note: Accuracy scores were calculated by dividing the number of correct responses for each condition and dividing it by the total number of responses given for all trials within that condition. Two-tailed independent-samples *t*-tests were performed for both conditions. No differences were found between groups [$ts(18) < 1.80$; ns].

groups in response accuracy for either the biological motion condition or the randomised biological motion condition; $ts(18) = .46$ and 1.84, respectively, ns.

3.2. Haemodynamic response data

3.2.1. Biological motion condition

When contrasting brain activity for the walker versus fixation comparison, a number of clusters emerged bilaterally within the posterior portion of the brain (see Fig. 1). Each

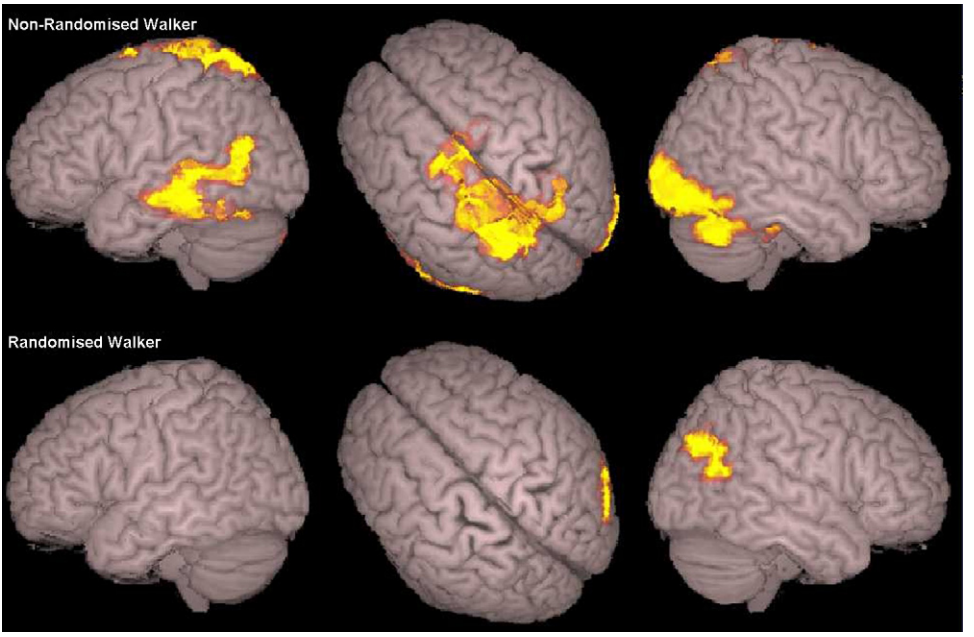

Fig. 1. Areas of reduced brain activity among individuals with Asperger Syndrome during the randomised and nonrandomized walker conditions. The statistical maps represent the comparison of brain activity across the two groups for the contrast of non-randomised walker versus fixation (top) and randomised walker vs. fixation (bottom), overlayed on a template brain that has been normalised into the MNI coordinate systems and rendered using mri3dX (http://www.jiscmail.ac.uk/lists/mri3dx.html). Clusters are significant at $p < .004$ for the non-randomized walker contrast, and .006 for the randomised walker contrast (each corresponding to a Type I error probability of less than one cluster). Activations in yellow represent clusters of activity that significantly lower for the AS group as compared to controls.

Table 3
Activation differences in walker vs. fixation comparison between the AS and control groups

Brain region	Talairach coordinates (*x*, *y*, *z*)	Cluster size (voxels)
Cerebellum	37.2, −42.2, −20.0	24
	51.0, −76.2, −20.0	56
	−30.9, −76.8, −16	68
Fusiform gyrus (BA37)	44.6, −64.2, −16.0	192
	37.9, −68.4, −12.0	76
	−30.9, −76.8, −12	68
Inferior temporal gyrus (BA37)	49.3, −51.4, −12.0	52
	39.6, −66.0, −1.0	212
Hippocampus	40.3, −32.2, −8.0	12
Middle temporal gyrus (BA21)	44.1, −47.8, −8.0	24
	−50, −43.9, −4	256
	−51.7, −39.8, −1	228
	−51.7, −39.8, 1.0	228
	−54.2, −36.3, 4.0	180
Middle occipital gyrus (BA19)	−38.2, −71, −8	64
	41.6, −68.4, −4.0	300
	28.9, −81.5, 4.0	152
	−39.2, −66.9, 4.0	20
	38.8, −80.4, 12.0	40
	27.0, −93.8, 16.0	88
	25.0, −92.9, 20.0,	108
	−39.2, −66.9, 8.0	20
Inferior occipital gyrus (BA18)	46.6, −79.3, −8.0	76
	33.9, −73.6, 1.0	308
Superior temporal gyrus (BA22)	−54.2, −36.3, 8.0	180
	−55.9, −36.8, 12.0	72
	−55.0, −50.0, 16.0	128
	−53.0, −50.8, 20.0	144
	−39.6, −59.3, 28.0	64
Cuneus (BA18)	25.5, −84.2, 8.0	168
	18.9, −98.8, 12.0	40
	−6.6, −109.7, 12.0	16
	21.6, −84.2, 24.0	20
Inferior parietal (BA39)	−44.5, −57.4, 24.0	72
Angular gyrus (BA39)	−36.3, −59.2, 32.0	84
	−47.5, −63.6, 35.0	28
Precuneus (BA7)	−20.6, −56.2, 35.0	16
	−21.6, −55.3, 40.0	16
	−21.6, −52.8, 45.0	24
Precentral gyrus (BA4)	−17.8, −41.1, 50.0	96
	−14.2, −41.9, 55.0	140
	−11.5, −41.2, 60.0	124

Note: Two-dimensional cluster peaks were calculated within each of 16 fMRI slices acquired. All of the clusters presented were significant at the $p < .05$ level (corrected). Cluster of less than 10 voxels were excluded.

Table 4
Activation differences in randomised walker vs. fixation comparison between the AS and control groups

Brain region	Talairach coordinates (x, y, z)	Cluster size (voxels)
Superior temporal gyrus (BA39)	49.5, −63.4, 28.0	80
Angular gyrus (BA39)	49.5, −63.4, 32.0	80
Angular gyrus (BA39)	52.8, −63.4, 35.0	12

Note: Two-dimensional cluster peaks were calculated for each of 16 fMRI slices acquired. All of the clusters presented were significant at the $p < .004$ level.

cluster reflected significantly decreased brain activity in the AS, when contrasted with the C group (see Table 3 for two-dimensional foci of activity and their respective stereotactic coordinates). In the left hemisphere, a large column of activation extended from cerebellum through the fusiform, inferior, middle and superior temporal gyri, including the middle occipital gyrus and cuneus region. A second column of activity in the right hemisphere extended from the cerebellum through the fusiform, middle temporal, superior temporal, middle occipital and superior occipital regions. This cluster of activity overlapped with the human analogue of MT+/V5, as measured by Dumoulin et al. (2000).

3.2.2. Randomised biological motion condition

When contrasting brain activity for the random walker versus fixation comparison, the AS group showed significantly less activity relative to the C group in a single three-dimensional cluster centered around the inferior parietal lobe, in Brodmann area 40 (see Fig. 1). This cluster of activity extended from the right superior temporal gyrus to the angular gyrus (see Table 4 for two-dimensional foci of activity and their respective stereotactic coordinates). No other significant differences were found when contrasting the two groups.

3.2.3. Two-way interaction between the two levels of group and condition

An ANOVA examining the interaction between group and brain activity measured during both levels of the walker condition yielded no significant clusters of activation.

4. Discussion

The current study provides evidence suggesting that individuals with AS show diminished activity in a number of brain regions related to the perception of human movement, including the human analogue of MT+/V5. Furthermore, most of these areas did not differ across groups for the control condition. As both conditions necessitated the perceptual integration of the same number of dots, moving at the same speed over the same visual distance, the primary difference between them is the extent to which each dot was positioned in a configuration that outlined a walking human form.

It is important to note that autism-spectrum conditions are frequently associated with abnormalities in attentional processes, and these abnormalities are likely related to some of the differences in social information processing found in AS (Plaisted, O'Riordan, & Baron-Cohen, 1998). Though the AS group was less accurate during the randomised

walker condition, this effect was non-significant, suggesting that differences in activation are not likely to be attributable to a lack of attentional resource allocation.

While these data support the hypothesis that individuals with AS show differences in posterior visual processing areas of the brain when compared to individuals without AS, the one statistical analysis that tested this hypothesis most directly – the group-by-condition ANOVA – did not yield any significant clusters when thresholding the data so that fewer than one false positive cluster would be expected. It is likely that our sample size was insufficient to achieve the statistical power necessary to observe significant differences in activity. Furthermore, both tasks necessitated the use of biological motion processing strategies, despite the fact that this process was made more challenging in the randomized biological motion condition by the perturbation of dot location. It could be that the similarity of the cognitive demands across the two tasks limited the effect size of the experimental manipulation.

Activation differences were observed in a number of specific areas related to the visual processing of biologically relevant information. A number of studies have suggested that superior temporal regions play an important role in the perception of biological motion, including complex human movements such as eye gaze, hand motion, and whole body movement (Grafton, Fagg, Woods, & Arbib, 1996; Grezes, Costes, & Decety, 1999; Grossman et al., 2000; Puce, Truett, Bentin, Gore & McCarthy, 1998; Vaina, Lemay, Bienfang, Choi & Nakayama, 1990). Furthermore, a neuroimaging study by Baron-Cohen et al. (1999) found significant deficits in superior temporal sulcus activity among a sample of individuals with AS while completing a task that involved mental state inferences based on eye stimuli.

In the present experiment, the right superior temporal gyrus was one of the only areas that differed significantly between the two groups in both the randomized and nonrandomized tasks. It is not surprising that there is some overlap between brain activity in the two conditions, as both necessitate the perception of a human form. As the behavioural results indicated that, overall, participants were able to identify the percept of a walking form substantially above chance levels during the randomized condition, the successful implementation of some biological motion processes must have occurred.

This study also provides further support for the hypothesis that autism-spectrum conditions involve abnormal patterns of activity in inferior temporal regions – particularly in the fusiform gyrus. Schultz et al. (2000) and Critchley et al. (2000), using experimental paradigms involving facial identity and affect recognition, respectively, showed that individuals with high-functioning autism and AS show diminished fusiform activity when compared to a normal control sample. Our data compliment that of Singh et al. (2001) in extending the possible role of the fusiform gyrus to include the processing of a broader category of biologically relevant visual information.

The hypothesis that individuals with AS would show significantly less activation in prefrontal regions was not supported by the data from this study. This appears contrary to recent finding by Singh et al. (2000) regarding significant activity in medial prefrontal, bilateral precentral and right dorsolateral prefrontal regions during biological motion perception. However, the randomized biological motion condition in the paradigm used in the Singh et al. (2000) used a much larger perturbation factor (K. D. Singh, personal

communication, November, 2000). It is possible that our lower perturbation factor eliminated some working memory or executive functioning component of the task that had previously resulted in frontal activation differences.

The regions of the brain showing divergent patterns of functional brain activity between the AS and C groups are likely part of a larger neural network related to autism spectrum conditions – particularly those autistic traits that are related to socio-emotional functioning. Temporal regions in particular are highly interconnected with other regions of the brain that appear to differ functionally among individuals with autism-spectrum conditions—specifically, the amygdala (via the stria terminalis) and prefrontal regions (Baron-Cohen & Tager-Flusberg, et al., 2000a; Tranel & Hyman, 1990).

Though Bonda et al. (1996) found amygdala activity in an fMRI study on a non-clinical group using a biological motion paradigm, the authors hypothesized that this activity may have resulted from the affective content of the stimuli (dancing point-light figures). This finding has not been adequately replicated to infer with confidence that the amygdala plays a significant role in the processing of human movement in isolation of other socially meaningful behaviour. Therefore, despite the literature suggesting that autism-spectrum conditions may relate to differences in amygdalar functioning, we did not hypothesize that the current paradigm would elicit differences in this region.

In this study we have demonstrated that specific brain regions related to social perception are abnormal in Asperger Syndrome. These findings appear to be consistent with the ''weak central coherence'' theory of autism (Shah & Frith, 1993). Weak central coherence theory posits that many of the differences in the neuropsychological profile of individuals with autism-spectrum conditions may be understood in terms of a decreased ability to implement information processes that bind together related features of a stimulus. The relatively less brain activity found in the AS group in fusiform gyrus regions is particularly consistent with weak central coherence theory, as this region has been implicated in the processing of visual configuration (sometimes referred to as ''holistic processing'') (Gauthier, Behrmann, & Tarr, 1999; Gauthier et al., 2000; Kanwisher, McDermott, & Chun, 1997; Schultz et al., 2000). However, one difficulty in interpreting our data in terms of central coherence theory is the ambiguity regarding whether the theory refers specifically to differences in perceptual binding, feature integration, or the meaning/relevance of the visual information that fail to cohere.

An important consideration for future research is whether these activity differences are present among autism-spectrum conditions or are unique to AS. Given that autism and AS share deficits in social intelligence, it is likely that they would show similar differences in visual cortex activity in response to social stimuli. Although in the present study all individuals in the AS group were diagnosed by mental health professionals using DSM-IV and ICD-10 criteria, one tool that is frequently used in research to distinguish autism from AS – the Autism Diagnostic Interview-Revised – was not administered (APA, 1994; Lord, Rutter, & LeCouteur, 1994; World Health Organization, 1994). Future studies including both autism and AS groups and multiple assessments tailored for parsing differential diagnoses will shed light on this question.

Observed differences in pattern of brain activity suggests that the difficulties in comprehending social information commonly reported among individuals with AS might stem in part from differences in perceptual information processing. In recent

years, an increasing amount of attention has been paid to areas of the brain that may be implemented in both the perception and implementation of human actions (Gallese & Goldman, 1998). The significantly decreased superior temporal activity found in the current study suggests that AS may involve fundamental differences in the functioning of this system. We conclude that reduced activation of key regions of the 'social brain', including the human analogue of MT+/V5, is characteristic of autism, and recommend that future work test if this reduction is restricted to the perception of social information or if this is part of a general difference in how all information is encoded by the autistic brain (Baron-Cohen, 2002).[1]

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[1] As of the time of this writing, we were unable to administer the WASI to three of the NC participants and one of the AS participants.

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