Neuropsychologia 47 (2009) 3225–3235

Contents lists available at ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

# No evidence for impaired perception of biological motion in adults with autistic spectrum disorders

Patrick Murphy[a], Nuala Brady[a,*], Michael Fitzgerald[b], Nikolaus F. Troje[c]

[a] School of Psychology, University College Dublin, Belfield, Dublin 4, Ireland
[b] Department of Psychiatry, Trinity College Dublin, Dublin 2, Ireland
[c] Department of Psychology & School of Computing, Queen's University, Kingston, Ontario K7 M 3N6, Canada

ARTICLE INFO

*Article history:*
Received 8 January 2009
Received in revised form 28 July 2009
Accepted 31 July 2009
Available online 8 August 2009

*Keywords:*
ASDs
Autism
Asperger's syndrome
Point light displays
Coherent motion
Biological motion
Motion perception

ABSTRACT

A central feature of autistic spectrum disorders (ASDs) is a difficulty in identifying and reading human expressions, including those present in the moving human form. One previous study, by Blake et al. (2003), reports decreased sensitivity for perceiving biological motion in children with autism, suggesting that perceptual anomalies underlie problems in social cognition. We revisited this issue using a novel psychophysical task. 16 adults with ASDs and 16 controls were asked to detect the direction of movement of human point-light walkers which were presented in both normal and spatially scrambled forms in a background of noise. Unlike convention direction discrimination tasks, in which walkers walk 'on the spot' while facing left or right, we added translatory motion to the stimulus so that the walkers physically moved across the screen. Therefore, while a cue of coherent, translatory motion was available in *both* the normal and scrambled walker forms, the normal walker *alone* contained information about the configuration and kinematics of the human body. There was a significant effect of walker type, with reduced response times and error when the normal walker was present. Most importantly, these improvements were the same for both participant groups, suggesting that people with ASDs do not have difficulty integrating local visual information into a global percept of the moving human form. The discrepancy between these and previous findings of impaired biological motion perception in ASDs are discussed with reference to differences in the age and diagnosis of the participants, and the nature of the task.


## 1. Introduction

Autistic disorder and Asperger's syndrome, the two conditions that collectively constitute the autistic spectrum disorders (ASDs), are pervasive neuro-developmental disorders characterised by severe impairments throughout the lifespan in social interaction and repetitive patterns of interest and behaviour (APA, DSM-IV, 1994). Abnormal language development and impairments in verbal and non-verbal communication are also features of autistic disorder (APA, DSM-IV, 1994). Perhaps the most influential cognitive theory forwarded to explain ASDs is that they result from problems forming a "theory of mind" (Baron-Cohen, Leslie, & Frith, 1985). According to this account the social cognition deficits seen in ASDs result from an inability to form an understanding of the intentions, emotions and knowledge of other people. However, while this theory has been quite successful in explaining many of the deficits associated with ASDs, it cannot easily explain other cognitive abnormalities associated with these conditions. People with ASDs are thought to show "weak central coherence" (WCC); an apparent processing bias for featural and local information, with a relative failure to extract global information (Happé, 1999). Behavioural studies show superior performance in ASDs relative to controls when local information is most salient to a task and poorer performance when extracting global information is required (see Happé & Frith, 2006, for review). In the visual domain, there are reports of a lack of susceptibility of people with autistic disorder to illusions that depend on global processing (Happé, 1996), reduced ability to form gestalts (Brosnan, Scott, Fox, & Pye, 2004), and superior ability on search tasks where global patterns disrupt the performance of normal observers (O'Riordan, Plaisted, Driver, & Baron-Cohen, 2001). However, despite this evidence, a relationship between WCC and social cognition deficits in ASDs has yet to be established (Beaumont & Newcombe, 2006).

Perceiving biological motion is one situation where the visual system integrates local information into a global form. Johansson (1973) showed that when human motion is represented by point-light displays (PLDs), which consist of 10–13 points of light on the major joints of the body, human observers could easily identify the moving human form despite the paucity of information in the displacing local dots. Perception of biological motion in PLDs is robust

* Corresponding author. Tel.: +353 1 716 8247; fax: +353 1 716 1181.
*E-mail address:* nuala.brady@ucd.ie (N. Brady).

0028-3932/$ – see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neuropsychologia.2009.07.026

when the local motions are masked by noise (Cutting, Moore, & Morrison, 1988; Bertenthal & Pinto, 1994), and even in the absence of local image motion (Beintema & Lappe, 2002), further indicating the importance of global form. Also, perception of biological motion in PLDs is compromised by inversion (Pavlova & Sokolov, 2000), which suggests that human bodies, like faces, may be processed as a global configuration by the visual system. Biological motion in PLD form can also convey a range of socially relevant information, including gender (Mather & Murdoch, 1994), affect (Pollick, Paterson, Bruderlin, & Sanford, 2001), personality traits such as trustworthiness (Heberlein, Adolphs, Tranel, & Damasio, 2004), identity (Jokisch, Daum, & Troje, 2006; Troje, Westhoff, & Lavrov, 2005) and whether the actor is a friend or a stranger (Loula, Prasad, Harber, & Shiffrar, 2005). The global processing involved in perceiving biological motion and the socially relevant information contained therein raises an obvious question for researchers: might a deficit in perceiving biological motion form a basis for some of the social cognition difficulties in ASDs?

Five studies have investigated this hypothesis, with contradictory findings emerging. In the first such study, Moore, Hobson, and Lee (1997) showed that children with autistic disorder and children with mental retardation were equally able to recognise moving people and other non-biological moving objects presented in PLDs. The dependent variable used was the exposure required for correct identification of the stimuli. Ability to identify a moving person did deteriorate at shorter exposure times for the autistic children, although this difference was not significant. The ability of both groups to recognise emotional states in human PLDs was also assessed and the autistic group, but not the mentally retarded group, performed significantly poorer than typically developing controls.

Blake, Turner, Smoski, Pozdol, and Stone (2003) revisited the issue with a different psychophysical task. Here, a group of children with autistic disorder were matched for chronological age with a group of typically developing children. The children were asked to identify whether a person was present in a series of normal and temporally scrambled human PLDs. Participants also completed a global form task, which required grouping lines into a circular target in a background of distracters. The autistic group was found to be selectively impaired on the biological motion task, and their performance correlated with severity of autistic symptoms. One important aspect of this methodology absent in Moore and colleagues' study is that temporal scrambling in the biological motion task rendered local motion much less informative in identifying the figure. As such, the impaired ability of the autistic group to discriminate between normal and scrambled walkers is interpreted as evidence for compromised mechanisms for integrating local motion into global biological motion.

Dakin and Frith (2005) have questioned the conclusion that the impairment is specific to motion. They argue that cells tuned to low spatial frequencies in the primary visual cortex are sufficient to group the target contours in the control task used by Blake and colleagues. This would mean that the form task did not require integration of local information into a global form. Therefore, the problem in ASDs could still be one of weak central coherence that extends to both form and motion rather than one that is specific to biological motion perception. A subsequent study by Del Viva, Igliozzi, Tancredi, and Brizzolara (2006), which uses the path integration task of Field, Hayes, and Hess (1993) in which global from is only available via the integration of local perceptual elements, lends support to the conclusions of Blake et al. (2003), showing no differences in contour integration between children with autistic disorder and controls.

Two further studies using very similar methodologies found no impairment in biological motion perception in adolescents and adults (Hubert et al., 2007) and children (Parron et al., 2008) with Asperger's syndrome and autistic disorder. In these two studies, the participants with ASDs were impaired in identifying emotional states exhibited by human PLDs, but not in identifying subjective states or actions in human PLDs or moving everyday objects in PLD form. These studies suggest that impaired emotional recognition in ASDs results from a purely "top-down" processing deficit. However, the stimuli used (~5 second clips of unmasked PLD motion) do not provide information on sensitivity to biological motion in ASDs or on local versus global processing strategies. Actions in the videos such as kicking and jumping, for example, are likely to contain far more overt and rigorous action than actions in the emotional states. A perceptual contribution to deficits in perceiving the emotional states cannot therefore be ruled out.

Finally, as part of a functional MRI study discussed below, Freitag et al. (2008) found increased response time, but not increased error, in identifying both normal and scrambled biological motion PLDs in a group of adolescents and adults with ASDs. This group were age and IQ matched with typically developing controls. The authors suggested that the result indicated that a greater cognitive effort was required on the part of the experimental group to differentiate the two types of stimuli. Performance was compared with imitative and adaptive gross motor abilities that are known to be poorer among people with ASDs. Dynamic balance ability was found to correlate with reaction time in identifying normal but not scrambled walkers, suggesting that perception of biological motion may be related to these abilities in ASDs.

There is quite a degree of overlap between cortical areas involved in biological motion perception and neurological abnormalities observed in ASDs. Several studies have implicated the superior temporal sulcus (STS), particularly the posterior region, in the perception of human biological motion (Beauchamp, Lee, Haxby, & Martin, 2002; Grossman & Blake, 2001a, 2001b; Grossman, Batelli, & Pascaul-Leone, 2005; Grossman, Donnelly, Price, Pickens, & Morgan, 2000; Howard et al., 1996; Peuskens, Vanrie, Verfaillie, & Orban, 2005; Puce, Allison, Bentin, Gore, & McCarthy, 1998; Thompson, Clarke, Stewart, & Puce, 2005). Given its location, it has been proposed that the STS integrates form and motion information from the ventral and dorsal streams, respectively, into a percept of the moving human body (Giese & Poggio, 2003). Also, acquired difficulties in perceiving biological motion have correlated with lesions suffered to the STS (Vaina & Gross, 2004). The STS is also activated when lip-reading, by viewing mouth movement, by gaze monitoring and by viewing static images where biological motion is implied (see Allison, Puce, & McCarthy, 2000, for review) and by non-visual social stimuli, such as people's footsteps (Bidet-Caulet, Voisin, Bertrand, & Fonlupt, 2005). STS functionality, therefore, is not restricted to perceptual processes, but has a wider function in the social brain. Also, STS cells can be selectively responsive to movement of the whole body or parts thereof (see Puce & Perrett, 2003, for review), indicating a complex role for this area in biological motion processing.

Several studies provide evidence for anatomical and functional abnormalities in the STS in people with ASDs. A voxel-based morphometry study found bilaterally significant decreases in grey matter concentration in the STS in children with autistic disorder (Boddaert et al., 2004). Anatomical shifting of the STS was reported in an MRI study of 22 children with pervasive developmental disorders, 21 of whom were diagnosed with autistic disorder (Levitt, Blanton, Smalley, Thompson, & Guthrie, 2003). Functional MRI studies show reduced or abnormal STS activity in people with ASDs when attributing mental states to moving shapes (Castelli, Frith, Happé, & Frith, 2002), when perceiving intent in gaze shifts (Pelphrey, Morris, & McCarthy, 2005) and when viewing dynamic emotional expressions in faces (Pelphrey, Morris, McCarthy, & LaBar, 2007). Thus far only Freitag et al. (2008) have found evidence for atypical STS activation in ASDs while viewing full-body

movement lacking overt emotional or intentional content. In this study, hypoactivation was observed in the right STS while observing PLD walkers, although this reduced activation did not reach significance.

Difficulties perceiving biological motion might also result from difficulties involved in the processing of general coherent motion. Various studies have reported such difficulties in ASDs and, through differing methodologies, have attributed their results to magnocellular pathway deficits (McCleery, Allman, Carver, & Dobkins, 2007; Milne et al., 2002) or dorsal stream deficits (Pellicano & Gibson, 2008; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Spencer et al., 2000). One study that recruited children with autistic disorder indicated that motion-processing problems might be restricted to second-order, texture-defined motion (Bertone, Mottron, Jelenic, & Faubert, 2003). The same authors later provided evidence that a difficulty with moving and stationary second-order stimuli exists in adults with high-functioning autism, which they attributed to increased lateral inhibition between orientation-selective neurons in the primary visual cortex (Bertone, Mottron, Jelenic, & Faubert, 2005). Importantly, some studies have found no difficulties with coherent motion processing in ASDs (de Jonge et al., 2007; Del Viva et al., 2006; Vandenbroucke, Scholte, van Engeland, Lamme, & Kemner, 2008), which suggests that motion-processing difficulties may be restricted to a sub-group of people with ASDs. For the current study, a potential dorsal stream deficit is perhaps most salient to our considerations, as abnormal activity in area V5 on the dorsal stream has been reported in Asperger's syndrome while viewing biological motion (Herrington et al., 2007). Also, global motion processing in the dorsal stream is known to develop in early childhood, and this development is thought to be vulnerable in a range of developmental disorders including ASDs (Braddick, Atkinson, & Wattam-Bell, 2003).

The current study is designed to test the idea that anomalies in perceptual processing underlie deficits in social cognition. To this end, we introduce a novel variant of a direction discrimination task involving PLD walkers that manipulates cues of coherent, translatory motion and biological motion.

In a typical direction discrimination task, PLD walkers walk 'on the spot' without translation while facing either to the left or the right (e.g., Thompson, Hansen, Hess, & Troje, 2007). Information about the global form or configuration is assumed to be of key importance in detecting biological motion, especially as stimulus inversion disrupts performance. Research by Troje and Westhoff (2006) highlights the importance of local motion carried by individual points, particularly those representing the feet which are salient in conveying information about the direction of motion. They showed that participants could discriminate the direction of motion of spatially scrambled PLD walkers even though this scrambling effectively destroys the global form of the walker.

In the current study too, participants with ASDs and neurologically typical controls were asked to discriminate the direction of motion of both normal and spatially scrambled PLD walkers. However, we added translatory motion to the PLD walkers so that they physically moved across the screen to the right or left rather than walked 'on the spot'. In addition, we presented the PLD walkers in a background of spatially scrambled walker noise with half of the noise points moving leftward and half moving rightward. This noise effectively masks the trajectory of individual stimulus dots so that, especially at high noise densities, it is unlikely that the intrinsic motion of the feet was used to discriminate the direction of motion as in Troje and Westhoff (2006). Instead the task could be solved by detecting a single dot or a cloud of dots that move coherently by translating together to the left or to the right. This cue of coherent, translatory motion was available in *both* the normal and the scrambled walker condition, and is likely to be the crucial cue in the scrambled condition. The normal walker condition *alone* contained information regarding the human form, available via 'structure from motion' processes. If the participants on the autistic spectrum were specifically impaired in perceiving biological motion, through a failure to integrate local signals into a coherent global percept, then this final source of information would not be available to them. If this occurred, the pattern of performance across the normal and scrambled walker trials would be expected to differ between the ASDs group and the controls.

## 2. Participants and methods

### 2.1. Participants

Adult participants for the experimental group were recruited from the Irish Health Service via a vocational training centre for adults with ASDs, from a university social group for students with Asperger's syndrome or were referred for the study by the third author, a psychiatrist whose research speciality is ASDs. Of twenty-two initially recruited, four performed at chance on the psychophysical task and their data were excluded. One participant was still awaiting a formal clinical diagnosis and was therefore excluded from the study. One participant with self-reported difficulties with depth perception and with differentiating left from right was excluded. The remaining 16 individuals (13 male), of mean age 25.56 years (SD = 7.67 years) were included in the study. Of these, 2 were in full time employment, 5 were in third level or adult education, 6 were attending a Health Services vocational training centre for adults with ASDs, and 3 were residents of a group home for adults with ASDs. All had received a diagnosis of Asperger's syndrome ($n = 15$) or autistic disorder ($n = 1$) from clinicians experienced in diagnosing ASDs. Information regarding the diagnostic procedure used by these clinicians was provided by the third author. All diagnosed Asperger's syndrome and autistic disorder using a structured assessment according to DSM-IV criteria (APA, DSM-IV, 1994). An absence of a clinically significant language delay was necessary to differentiate Asperger's syndrome from autistic disorder. Normal cognitive development and functioning was also used to separate Asperger's syndrome from autistic disorder in some cases. Participants did not partake in a research diagnosis using a diagnostic instrument (e.g., ADOS, Lord, Rutter & LeCouteur, 1994). However, the discriminative ability of such instruments and clinical diagnoses are very similar, with most discrepancies being false positive diagnoses using the diagnostic instrument (Mazefsky & Oswald, 2006).

The control group was recruited from the student population in University College Dublin and from the community. Of eighteen initially recruited, two participants, including the first named author, were experienced with psychophysical tasks relating to PLD biological motion and were excluded from the study to control for practice effects. The remaining 16 participants were gender-matched with the ASDs group and their mean age was 26.40 years (SD = 2.85 years). All participants in both groups had normal or corrected-to-normal vision.

Participants were assessed for non-verbal ability using Raven's Standard Progressive Matrices (Raven, Raven, & Court, 1998). Full instructions were given and the test was completed at the participant's leisure. 15 members of the ASDs group and 14 controls completed tests. The control group were found to have significantly higher non-verbal ability, $t(15) = 4.81$, $p < 0.0001$ (Table 1). The box plot in Fig. 1 shows the distribution of scores on this test for both groups. This illustrates the significant difference between both groups in terms of mental functioning, and also the greater heterogeneity in this regard in the ASDs group. Using norms for the British population (Raven et al., 1998) six of the ASDs participants, ($n = 5$ with Asperger's syndrome) were found to lie at or

**Table 1**
Age and non-verbal ability.

| Group | Age/years (SD)[a] | Raven's matrices score (SD)[b] |
|---|---|---|
| ASD group | 25.56 (7.67) | 43.73 (9.64)* |
| Controls | 26.40 (2.85) | 56.21 |

* Significant difference between groups ($p < 0.0001$).
[a] $n = 16$ for each group.
[b] $n = 15$ for ASD group, $n = 14$ for Controls.

below the 5th percentile for their age group, indicating intellectual impairment (which translates to IQ < 70).

### 2.2. Ethics

Participation in the study was voluntary and written consent was received from all participants. Ethics approval for the study was received from the Human Research Ethics Committee at UCD and from the Irish Health Service Executive. Where appropriate, participants were explicitly informed that participation in the study was unrelated to any training or rehabilitative courses they were undertaking within the health service. All participants were informed of the duration and difficulty of the experimental task before consenting to participate and were advised of their right to withdraw from the study at any time without prejudice.

### 2.3. Stimuli & display

The point-light walker stimulus was created in the Physiotherapy & Performance Science Laboratory at UCD using a CODA 3-D Motion Analysis System (Charnwood Dynamics, Leicestershire, U.K.). It consisted of thirteen active markers, attached to the major joints of a male subject who walked in place on a treadmill at a comfortable pace, which were recorded for approximately 30 s at 60 Hz. A 0.92 second sample of the recording, corresponding to 1 complete step cycle, was processed in Matlab® to create an 11-point saggitally viewed walker, where 1 step cycle ran over 56 frames.

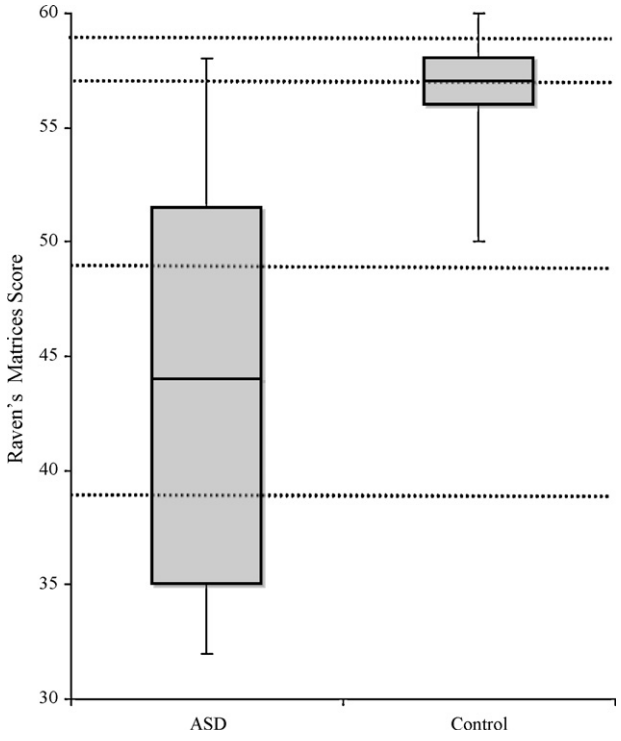


**Fig. 1.** Box plot showing scores of both groups for Raven's Standard Progressive Matrices. The large rectangle for each group shows the distribution of the middle 50% of scores, the lines bisecting these rectangles represent the medians of the distributions and the extension of the tails attached to the rectangles includes 100% of the scores. The broken lines crossing the graph represent (from the bottom up) the 5th, 25th, 50th, 75th and 95th percentiles for the general population, as per the norms for the British population (mean age = 25 years) when the test is self-administered and completed at leisure (Raven et al., 1998).

The experiment was run on a Dell Dimension 9100 PC using software written in Matlab® (R2007a, The Mathworks, Natick, Massachusetts) with extensions from the Psychophysics Toolbox (Brainard, 1997). The display was run at 60 Hz with a spatial resolution of 1024 by 768 pixels. The walker, 11 white dots presented on a dark background, subtended ~11.9 degrees in height at a viewing distance of 50 cm and was presented within a background strip of noise ~34.77 degrees wide by 16.58 degrees high. The noise was created by scrambling a variable number of walkers (for a noise density of 50, 100, 150, 200 or 250 points) so that the starting location of each point was randomly chosen within the mask area. The starting phase (i.e., position in walk cycle) of each noise point was also randomized, and for each of the five noise densities half of the points were sampled from a leftward facing and half from a rightward facing walker.

On each trial, the walker appeared in the centre of the display (jittered between 0 and 0.7 degrees vertically and 0 and 1.13 degrees horizontally) and was presented with variable onset time (0, 125, 250, 375 or 500 ms after the onset of the noise mask). The spatial and temporal uncertainty associated with stimulus onset was introduced to minimise the usefulness of a fixation strategy across trials. We added translatory motion to the stimulus; each of the 11 stimulus dots translated either rightward or leftward by a fixed distance on each movie frame so that the walker physically moved across the screen, rather than walking 'on the spot'. Each trial lasted 392 frames (~6.5 s) and the translation was such that the walker completed 7 full step cycles in walking from the centre of the screen to the edge. The trial duration was chosen to allow enough time for participants to find the stimulus and respond, and as reported below, this upper time limit proved adequate for even the slowest participants from the ASDs group. The number of step cycles used to translate the walker across the screen was chosen to ensure a naturalistic walk, i.e., 56 frames for 1 step cycle ~0.93 s at 60 Hz, so 7 step cycles ~6.5 s, the trial duration. The original walker was recorded at a step cycle of ~0.92 s on a treadmill.

The walker was presented in either normal or scrambled form. To control for stimulus density, the starting positions of the 11 scrambled walker points were tightly constrained to the region occupied by the normal walker. We emphasise here that *both* the normal and scrambled walkers contained translatory motion that could be used as a cue to direction. The normal walker *alone* contained information about the form and kinematics of the original human walker. An example of a normal and scrambled walker in noise is shown in Fig. 2. A chin rest was used to maintain viewing distance but participants were free to move their eyes and were encouraged to track the stimulus in deciding its direction of motion. The computer screen provided the only illumination in an otherwise darkened room.

### 2.4. Procedure

Each participant first completed a series of 15 practice trials to gain familiarity with the task and the concept of the PLDs. To this end, the practice trials consisted of low noise densities (0, 30, 40, 50, 80 points), although the duration of the trials and the velocity of the stimulus were equal to that in the experiment. Participants were advised that the experiment itself would contain higher noise densities and thus be more difficult. Participants were asked to respond as quickly as possible on each trial by pressing one of two keys on the keyboard to indicate whether the stimulus was moving to the left or the right (key 'm' for right and 'c' for left). All participants

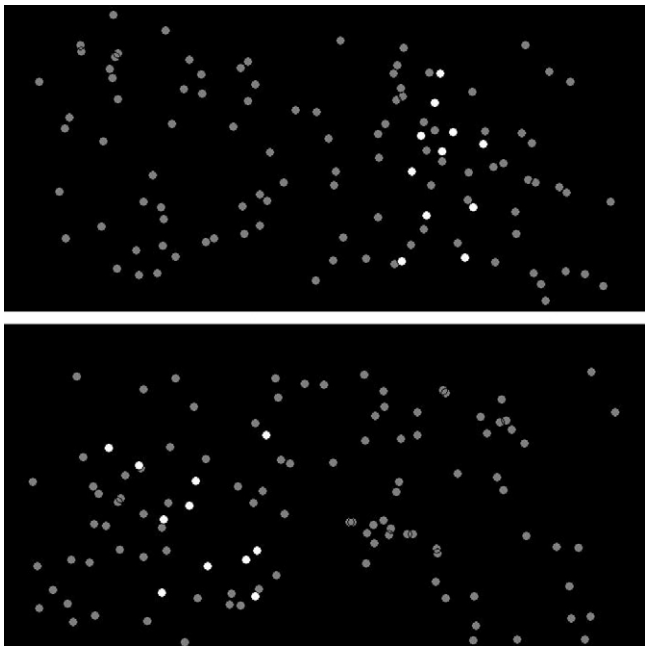

**Fig. 2.** Single frames from two trials in which a normal walker is moving rightward (top panel) and a scrambled walker is moving leftward (bottom panel) in noise of 100 point density. Both walker and noise dots were white on black background in the experiment, so the walker was distinguished only by its motion.

were advised to use their left hand to indicate leftward motion and their right hand for rightward motion. Following the practice trials, each participant then completed 400 trials comprising 4 trials each of 5 noise densities (50, 100, 150, 200, 250 points) by 5 onsets (0, 125, 250, 375, 500 ms) by 2 directions of motion (right, left) by 2 stimulus types (normal, scrambled walker). The 400 trials were presented within a single block in randomized order. Each trial was initiated by the participant pressing the spacebar and ended as soon as the participant responded; in the case of the participant not responding the trial ended after ~6.5 s.

Response times were measured with respect to stimulus onset. Response times of 250 ms or less were treated as anticipation errors and excluded from the data analyses and trials on which participants did not respond were treated as errors. Participants were made aware that not responding during the trial would be treated as an error.

## 3. Results

Both reaction time (RT) on correct trials and error data were analysed. Reaction times less than 250 ms were omitted as anticipatory errors; these accounted for less than 0.07% of correct trials. As the RT distribution was approximately log normal, RT was log transformed prior to analyses. Errors were calculated prior to RT filtering and included trials on which a participant responded incorrectly or failed to respond within the trial time. Errors accounted for 16.02% of all trials and timeout errors counted for 43.17% of all error trials. The data were modelled using the multilevel, mixed effects model in R (http://lme4.r-forge.r-project.org/) with factors of *Group* (ASDs/Controls), *Walker Type* (normal/scrambled), noise *Density*, and walker *Onset* time.

### 3.1. Reaction time

Fig. 3 plots reaction time against noise density on the left and against onset on the right. The effect of walker type is clearly evident in both plots with considerably higher reaction times in the scrambled walker condition. Looking to the density plot on the left, background noise slows performance in both the normal and scrambled walker condition with log RT increasing linearly with noise density. Although the ASDs participants are a little slower than the controls they show comparable performance. Of particular interest, they are faster in the normal than in the scrambled walker condition, and this increase in reaction time is similar to that shown by the control group. The improved performance in the normal walker condition indicates that the participants with ASDs, like the controls, used the extra source of visual information available in these trials: that of the form of the human body. This suggests normal perception of biological motion. The log RT for the ASDs group increases with increasing noise density in both the normal and scrambled condition in a manner comparable to that of the controls, suggesting similar sensitivity to noise. The onset plot on the right shows that participants are faster to detect and correctly identify the direction of motion of a walker when it is delayed relative to the background noise. This RT advantage for delayed targets is seen for both the normal and scrambled walkers and occurs for both the ASDs and control groups.

Statistical analyses confirm these observations. Table 2 shows both fixed effects, which are averaged across participants, and random effects (the standard deviation of coefficients across par-

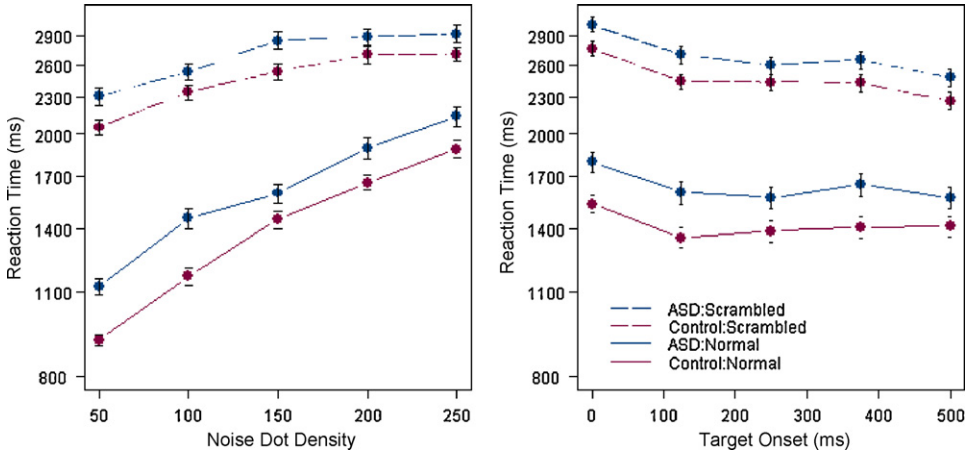


**Fig. 3.** Mean reaction time is plotted on log *y*-axis as a function of noise density (left plot) and walker onset (right plot). Error bars show ±1S.E. of the mean. Separate traces are shown for the normal walker (solid lines) and scrambled walker (broken lines) conditions and for the ASD (blue) and the control (red) participants. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

**Table 2**
Effects of experimental variables on response time and percentage error.

| | Response time | | | Random effects | Error | | | Random effects |
|---|---|---|---|---|---|---|---|---|
| | Fixed effects | | | Std. dev. | Fixed effects | | | Std. dev. |
| | Estimate | Standard error | *T*-value | | Estimate | Standard error | *Z*-value | |
| Group | 0.147 | 0.078 | 1.87 | | 0.517 | 0.381 | 1.36 | |
| Density | 0.241 | 0.019 | 12.81** | 0.066 | 1.043 | 0.073 | 14.33** | 0.144 |
| Onset | −0.029 | 0.008 | −3.55* | 0.030 | 0.026 | 0.026 | 0.998 | 0.049 |
| Walker Type | 0.584 | 0.049 | 11.83** | 0.191 | 1.508 | 0.150 | 10.06** | 0.462 |
| Group × Density | −0.007 | 0.027 | −0.27 | | −0.029 | 0.098 | −0.297 | |
| Group × Onset | −0.002 | 0.012 | −0.13 | | −0.0001 | 0.035 | −0.005 | |
| Group × Walker Type | −0.012 | 0.070 | −0.18 | | 0.021 | 0.208 | 0.100 | |

* Significant at $p < 0.001$.
** Significant at $p < 0.0001$.

ticipants). As log transformed RT is regressed on linear variables, the estimated co-efficients are approximately percentages, e.g., on average, increasing noise density by 100 dots leads to an increase in RT of ∼24% (+/1 ∼1.9%). The main effects of *Density*, *Onset* and *Walker Type* are highly significant while the main effect of *Group* approaches significance at $p = 0.0562$. Most importantly for our research question, the two-way interaction between *Group* and *Walker Type* is not significant, which shows that reaction time for both groups is similarly affected by the presence of the normal or scrambled walker. The two-way interactions between *Group* and noise *Density* and *Group* and walker *Onset* are not significant, showing that for both the control participants and those with ASDs, reaction time is similarly affected by noise density and walker onset.

### 3.2. Error

The pattern of the data in Fig. 4, which plots percentage error against noise density on the left and against walker onset on the right, shows similar trends to the RT data. First, participants make more errors when the walkers are scrambled than when they are normal. Secondly, there is a clear effect of background noise as error increases linearly with noise density in all conditions. Thirdly, the effect of target onset is subtle; error appears to increase a little as targets are delayed for the normal walkers but not for the scrambled walkers. Finally, and mirroring the RT data, although the participants with ASDs are a little more error prone than controls, they show a similar increase in error with noise density and target onset. Most significantly, they show a similar increase in error with stimulus type. This indicates that the ASDs group, like the control group, shows a marked improvement in accuracy when the form of the human body is present in the stimulus.

Table 2 shows the output of a multilevel, mixed effects model using the binomial family. The main effects of *Density* and *Walker Type* are highly significant while neither the main effect of *Onset* ($p = 0.318$) nor *Group* ($p = 0.175$) reach significance. Most importantly, the two-way interaction between *Group* and *Walker Type* is not significant, which shows that error for both the ASDs and the control group is similarly affected by the presence of the normal or scrambled walker. The two-way interactions between *Group* and noise *Density* and *Group* and walker *Onset* are not significant, showing that for both the ASDs and control participants, error is similarly affected by noise density and walker onset.

### 3.3. Sensitivity, $d'$

Fig. 5 plots $d'$, a measure of sensitivity that is independent of response bias calculated from the proportion of 'hits' and 'false alarms' (Macmillian & Creelman, 1991), against noise density on the left and against walker onset on the right. The current task may be defined as a two alternate forced choice of saying whether the signal is moving to the right; here 'hits' correspond to trials in which the walker is moving rightward and the response is right, and 'false alarms' correspond to trials in which the walker is moving right-

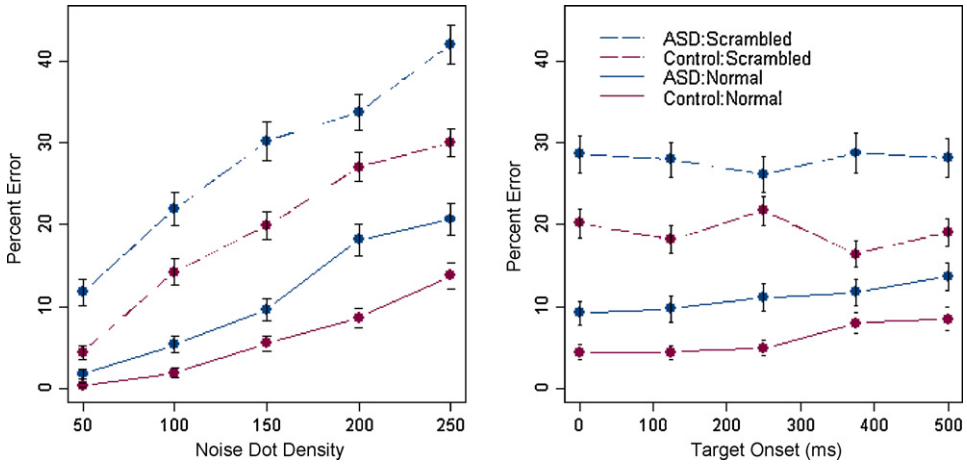


**Fig. 4.** Mean percentage error is plotted as a function of noise density (left plot) and walker onset (right plot). Error bars show ±1S.E. of the mean. Separate traces are shown for the normal walker (solid lines) and scrambled walker (broken lines) conditions and for the ASD (blue) and the control (red) participants. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

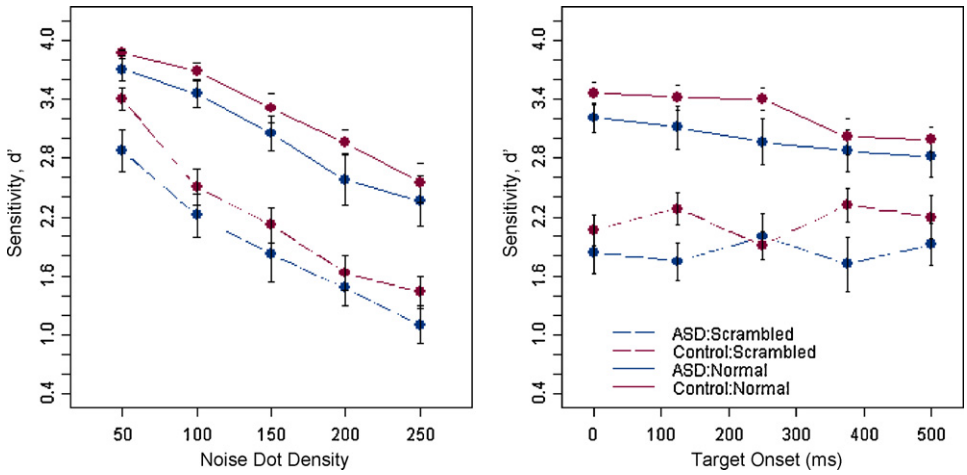


**Fig. 5.** Sensitivity, $d'$, is plotted as a function of noise density (left plot) and walker onset (right plot). Error bars show ±1S.E. of the mean. Separate traces are shown for the normal walker (solid lines) and scrambled walker (broken lines) conditions and for the ASD (blue) and the control (red) participants. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of the article.)

ward and the response is left.[1] Fig. 5 shows that sensitivity is higher for normal than for scrambled walkers, and that sensitivity declines markedly as background noise increases.

A mixed effects ANOVA with dependent variable of $d'$, a between subjects factor of *Group* (ASDs/control) and within subjects factors of *Density* (5 levels) and *Walker Type* (normal/scrambled) showed significant main effects of *Density*, $F(4,120) = 106.3$, $p < 0.0001$, and of *Walker Type*, $F(1,30) = 357.9$, $p < 0.0001$. The main effect of *Group* was not significant, $F(1,30) = 1.92$, $p = 0.18$. A significant *Density* × *Walker Type* interaction, $F(4,120) = 4.90$, $p = 0.001$, is readily interpreted from Fig. 5 where sensitivity decreases more rapidly in the scrambled than in the normal walker condition. Most importantly for our findings, no interactions involving *Group* were significant, showing that the ASDs and controls groups were similarly affected by noise density and walker type, i.e., the increase in sensitivity seen for the normal over the scrambled walker was comparable for the ASDs and control participants.

Analysis of the onset data showed a significant main effect of *Walker Type*, $F(1,30) = 386.1$, $p < 0.0001$. Neither the main effect of *Group*, $F(4,120) = 0.83$, $p = 0.51$, nor the main effect of *Onset*, $F(4,120) = 1.83$, $p = 0.13$, were significant. A significant *Onset* × *Walker* interaction, $F(4,120) = 3.56$, $p = 0.009$, reflects the differential effect of delaying target onset on sensitivity in the normal and scrambled walker conditions. While sensitivity is roughly constant across onset for scrambled walkers, sensitivity shows a small but steady decline with increased target delay for normal walkers. No interactions involving Group were significant, showing that the ASDs and control groups were similarly affected by target onset and walker type.

Finally, an overall measure of sensitivity was calculated for the 16 ASDs participants who completed the Raven's Standard Progressive Matrices by taking the mean of their $d'$ scores (as calculated across 5 densities, 5 onsets and 2 walker types). As it is calculated across both walker types, this metric serves as a general measure of overall performance and does not disambiguate between performance on trials with and without biological motion. This overall measure of sensitivity was found to be positively correlated with scores on the Raven's test (Pearson's $R = 0.48$, $t = 1.88$, df = 13, $p = 0.041$).

## 4. Discussion

Individuals on the autistic spectrum and neurologically typical controls completed a novel psychophysical task that required detecting the direction of movement of a point-light walker that translated in noise across a screen. Walkers were presented in both normal and scrambled form. There were three different cues to the direction of motion available in the displays. First, both the normal and scrambled walker displays contained a cue of coherent, translatory motion in that all points in the stimulus translated together from the centre of the screen rightward or leftward at a normal walking pace during the course of each trial. This was arguably a very potent cue in both walker conditions. Secondly, the normal and scrambled walker displays also shared local motion cues to direction and research by Troje and Westhoff (2006) has shown that points representing the feet are particularly salient in conveying direction, operating for both normal and scrambled walkers viewed in isolation. However, as our walkers were presented in scrambled walker noise, we expect that this cue only played a minor role and is likely to have been completely masked at high noise densities. Thirdly, the shape or configuration of the human form, which was only available in the normal walker display, provides another powerful cue, with the facing direction of a PLD walker being immediately evident once it is articulated. However, in order to avail of this cue, participants must be able to integrate local information into a global form and to then discriminate this global form from the background noise.

The results are unequivocal. There is a strong and highly significant effect of walker type on performance; both response time and error decrease and sensitivity increases when the normal, intact walker is present. Most importantly, this improvement is the same for the ASDs and the control group, showing that participants with ASDs were able to use information about the configuration of the human form available via structure from motion processes. The manipulation of noise density and stimulus onset time allowed us study performance across different levels of task difficulty. Both groups of participants were equally affected by these parameters suggesting comparable attention to the task. While the ASDs participants were somewhat slower overall (this effect approached significance at the 5% level), there was no significant difference between the ability of the two groups to detect the direction of

[1] While missed trials (i.e., trials on which the participant failed to respond during the trial duration) were treated as errors in the RT and Error analyses, missed trials were randomly assigned a 'right' or 'left' response to model the process of guessing common to 2AFC tasks. Missed trials accounted for ~6.9% of all trials and were more common among the ASDs group.

the normal and scrambled walkers when accuracy was used as a measure of performance and there was no difference in sensitivity between the two groups. A similar finding of slower reaction times but comparable accuracy in ASDs was reported by Freitag et al. (2008) for a biological motion task that involved discriminating normal from scrambled walkers.

Our findings contradict any strong form of the 'weak central coherence' theory, which suggests a reliance on local cues in autism, but is consistent with the revised theory which allows for the use of global cues when these are important to the task (Happé & Frith, 2006). The participants with ASDs had no apparent difficulty integrating the minimal local information in the PLDs into coherent human motion, suggesting that the neurological mechanisms for perceiving biological motion are normal. Our findings are also discrepant with those of Blake et al. (2003), who interpreted their data as evidence for a specific deficit in perceiving global biological motion in ASDs. As such they question the idea that a bottom-up perceptual deficit may provide a basis for some of the social cognition difficulties in ASDs. Before discussing these issues, we first consider our task in more detail.

It might be argued that the ASDs group's superior performance on the normal walker trials resulted from local, rather than global processing mechanisms. Maybe, for example, a fixation strategy was used by the ASDs group to track local dot trajectory. Using such a strategy, the predictable pattern of the normal walker might lead to quicker responses and reduced error. We believe this is highly unlikely for two reasons. Firstly, both spatial jitter and a variable stimulus onset delay were included to disrupt such a strategy. Secondly, the pattern of performance for scrambled and normal walker trials across the various noise densities and stimulus onset delays was strikingly similar for both groups. The most reasonable conclusion therefore, is that the strategies used by both groups were similar and that the participants with ASDs perceived the global form of the moving human body in the normal PLD walker.

A weakness of the current study is that the groups were not matched for intellectual ability. This does not affect our interpretation of the main result of interest, i.e., there was a similar improvement for both groups when the normal walker was present, showing that the participants with ASDs were unaffected in this regard by both their autistic symptoms and intellectual level relative to controls. However, the data did indicate that the ASDs group were generally more error prone and somewhat slower in identifying direction for scrambled and normal walkers, although these differences were slight. There are two potential sources for this difference. Both types of trials involved perception of coherent motion, so perhaps a deficit in this regard explains the slightly greater error and increased reaction time for both normal and scrambled walkers. Alternately, the lower intellectual level of the participants with ASDs might account for the general increased error and reaction time. Our measure of overall sensitivity correlated positively with the Raven's test scores for the ASDs group, suggesting that the latter is the case. However, replication of this study with IQ matched groups will be necessary before this can be concluded.

How might one explain the discrepancy between our results and those of Blake et al. (2003)? An obvious difference between these studies is the age of participants. One possibility is that the autistic children in Blake et al. (2003) study were simply developmentally delayed in processing biological motion. Perhaps by adulthood this ability "catches up" with that of neurologically typical adults, as per our results.

A second difference concerns the nature of tasks used. The children in the Blake et al. (2003) study were asked to discriminate normal from temporally scrambled walkers engaged in different activities such as running, jumping, kicking and throwing, and these actions were performed 'on the spot' for a period of 1 second. In contrast, we asked participants to say which direction a PLW was 'walking', so that both normal and scrambled walkers translated across the screen. While our results show markedly improved performance in the normal walker condition, arguing for normal perception of biological motion, it is possible that the perception of biological motion is naturally enhanced by the addition of the coherent motion across the screen by means of cue combination.

Thirdly, and related to the last point, the two tasks likely vary in the attention demands they place on participants. Symptoms of attention deficit-hyperactivity disorder and autistic symptoms are commonly co-morbid (Goldstein & Schwebach, 2004) and difficulties with attention relative to IQ score have been found in ASDs (Mayes & Calhoun, 2003). Given this, any increase in attentional demands in a psychophysical task may contribute to impaired performance in children with ASDs relative to controls, independent of the children's IQ and any variation in perceptual demands of the task.

Finally, a comparison of our results with those of Blake et al. (2003) must consider the diagnosis received by participants and their cognitive profiles. The participants in Blake et al. (2003) were children diagnosed with autistic disorder, while all except one of the adult experimental group in our study had a diagnosis of Asperger's syndrome. It could be concluded that the discrepancy in results reflects a genuine difference between autistic disorder and Asperger's syndrome. This would be supported by reports of perceptual differences between these two conditions. For example, two studies have found evidence for a deficit in perceiving coherent motion in high-functioning autism, but not in Asperger's syndrome (Spencer & Ó Brien, 2006; Tsermentseli, Ó Brien, & Spencer, 2008). Also, there is evidence for a slightly stronger bias towards local visual detail in high-functioning autism relative to Asperger's syndrome (Rinehart, Bradshaw, Moss, Brereton, & Tonge, 2000, Rinehart, Bradshaw, Moss, Brereton, & Tonge, 2001).

Two important points must be noted before making such a conclusion. Firstly, although some initial studies did suggest that Asperger's Syndrome and autistic disorder coincided with quite separate neuropsychological assets and deficits (Klin, Volkmar, Sparrow, Cicchetti, & Rourke, 1995), there is now a range of evidence to show that these conditions converge in terms of behavioural, cognitive and neurological profiles (Frith, 2004; Howlin, 2003; Macintosh & Dissanayake, 2004). According to the DSM-IV (APA, 1994) classification, an absence of a significant delay in language and cognitive development distinguishes Asperger's syndrome from autistic disorder, while the remaining diagnostic criteria largely overlap. When intellectual disability is absent in autistic disorder, it is usually described as high-functioning autism and the boundaries between this disorder and Asperger's syndrome is a matter of some controversy. Macintosh and Dissanayake (2004) reviewed studies that compared these two disorders, and found "very few qualitative distinctions" between them, based on cognitive and neuropsychological profiles and symptomatology. Howlin (2003) found that, although parents' reports suggested differences between Asperger's syndrome and high-functioning autism in children, the differences in functioning by adulthood were slight. Differences between these subgroups appear to mainly reflect differences in the severity of symptoms rather than 'alternate symptom profiles' (Macintosh & Dissanayake, 2004), leading many to conclude that Asperger's syndrome is equivalent to high-functioning autism (Mayes, Calhoun, & Crites, 2001).

The second important point concerns our ASDs sample. Although 15/16 of these participants had received a clinical diagnosis of Asperger's Syndrome, non-verbal IQ testing showed them to differ greatly in terms of ability (Fig. 1). It seems more likely that they represent the autistic spectrum, rather than a sub-group therein. Indeed, the low scores of some of the ASDs group here are generally more compatible with a diagnosis of autistic disor-

der rather than Asperger's syndrome. This reflects the fact that the cognitive profiles of people with the clinical features of Asperger's Syndrome can be "remarkably uneven" (Attwood, 2007, pp. 44–45) and that clinical judgement might place people with a borderline intellectual impairment in this clinical group if some cognitive skills are within the normal range (Attwood, 2007, p. 45). More generally, Mayes et al. (2001) note that the specific requirement of DSM-IV that Asperger's syndrome is distinguished from autistic disorder by a lack of cognitive delay is not adhered to by all clinicians. Interestingly, those scoring the lowest on the Ravens Progressive Matrices test here were either living in a group home for people with autistic disorder and/or attending vocational training services for people with autistic disorder. Normal language development was given as the main criterion to differentiate the two conditions for these participants, and lower functioning participants may have been diagnosed with Asperger's syndrome for this reason. Overall, our ASDs group would therefore appear to represent a heterogeneous range on the autistic spectrum and does not support the existence of a sub-group therein with a deficit in perceiving biological motion.

Our results do not suggest a perceptual contribution to difficulties interpreting the underlying meaning of human actions in ASDs. Perhaps STS abnormalities previously observed in ASDs do not preclude integration of local body motion into a percept of the moving human form. Also, since normal performance on this higher level visual task argues for preserved lower level visual function, previous hypotheses regarding magno-cellular or dorsal stream dysfunction in ASDs are not supported by our data. As such, our results support the hypothesis that the origin of social cognition deficits in ASDs is top-down rather than bottom-up. Accepting this premise, there are two other likely explanations for the difficulties that people with ASDs have identifying the emotional states present in human motion (Hubert et al., 2007; Parron et al., 2008). The first is other neurological abnormalities observed in ASDs that correlate with areas of the social brain involved in such tasks. This could include areas of the STS not crucial to biological motion perception, but also the amygdala (see Baron-Cohen et al., 2000) and the orbitofrontal cortex (Baron-Cohen et al., 1994). Frontal "mirror-neuron" areas involved in motoric representations of observed actions are also activated by biological motion (Saygin, Wilson, Hagler, Bates, & Sereno, 2004). Abnormalities in these areas, might underlie the inability to decipher intentions from an intact percept of human motion (Boria, Fabbri-Destro, Cattaneo, Sparaci, & Sinigaglia, 2009). The second possibility is that children with ASDs lack the predisposition of other children to attend to human motion and fail to thus develop the ability to decipher its underlying psychological correlate. This is supported by one case study that showed reduced attention to upright PLD walkers in a 15-month old infant with autism compared to a typically developing 9-month old (Klin & Jones, 2008). Both of these explanations may not be mutually exclusive and could indeed be alternate descriptions of the same underlying cause.

Autistic disorder and Asperger's syndrome are heterogeneous, behaviourally defined disorders. This predicts a heterogeneous neuropathology in ASDs, with different areas of the social brain underpinning varying difficulties in the affected individuals (see Amaral, Schumann, & Nordahl, 2008, for review of this evidence). If a difficulty processing biological motion existed in ASDs, it would be surprising if it were common to all, or even the majority of individuals diagnosed with these conditions. This seems even more unlikely when we consider the range of brain areas and cognitive mechanisms implicated in perceiving biological motion. Along with the STS, imaging studies of biological motion have shown significant activity in the extrastriate body area (Downing, Jiang, Shuman, & Kanwisher, 2001), the fusiform face area (Grossman & Blake, 2002) and motion sensitive area V5 (Peuskens et al., 2005). In addition, findings from studies of impaired biological motion processing following brain injury are inconsistent. These studies have variously implicated the extrastriate cortex, medial temporal and posterior parietal lobes (Cowey & Vaina, 2000), the parietal lobes (Battelli, Cavanagh, & Thornton, 2003), the anterior temporal and parietal lobes (Vaina & Gross, 2004), and superior temporal and premotor cortical areas (Saygin, 2007). This highlights that neurological impairments in a complex developmental disorder will not overlap in a neat fashion with neural substrates for perception of a complex social stimulus. A greater understanding of clinical sub-groups within ASDs is required before we can understand the interaction of perceptual anomalies and social cognition deficits in this disorder. This could reveal and quantify bottom-up and top-down contributions to the varied symptoms of ASDs.

A related issue is the difficulty separating the cortical substrates that underpin higher level perceptual processing and social-cognition tasks. In neurocognitive research, imaging data are often tacitly interpreted as evidence for the localisation of function within the brain. However, for perception of higher order stimuli, such as the moving human body, there is ample evidence for modulation of activity in areas associated with perception by the social demands of the task. A good example of this is in the fusiform face area of the temporal lobe, an area known to show abnormal activation in ASDs (Pierce, Muller, Ambrose, Allen, & Courchesne, 2001). Although associated with configural face perception, it is now known that fusiform face area activity is modulated by emotional demands while viewing faces (see Vuilleumiera & Pourtois, 2007 for review). More relevant to the current study, activity in brain areas associated with biological motion perception, such as the extrastriate body area, is also modulated by the underlying psychological meaning of the actions (Takahashi et al., 2008). All of this indicates that the lines between perception and social cognition can become blurred when dealing with higher order stimuli. Acquiring psychological information from human expressions activates distributed circuits across the brain, and activity in those areas regarded as perceptual may in fact underpin the processing of social information. Research into ASDs provides evidence for this; differences in fusiform face area activity between autistic and control subjects are greater when information must be acquired from the face as opposed to when the face is observed passively (see DiCicco-Bloom et al., 2006 for review).

Consideration of this issue is important given that some researchers have suggested that underconnectivity in large-scale neural networks may form a basis for autistic symptoms (Belmonte et al., 2004). Imaging studies have shown abnormal functional connectivity between language and visual imagery centres (Kana, Keller, Cherkassky, Minshew, & Just, 2006), between parietal and frontal areas during working memory tasks (Koshino et al., 2005) and between the fusiform face area and amygdala when viewing faces (Kleinhans, Richards, Sterling, Stegbauer, & Mahurin, 2008). Local activity was not necessarily abnormal in these studies, which indicates that the problem in ASDs could in fact be the integration of information processed in different brain regions. Abnormal functional connectivity has also been found in the resting, or "default" brain network in ASDs (Cherkassky, Kana, Keller, & Just, 2006; Murias, Webb, Greenson, & Dawson, 2007), which has been found to show abnormal activity in ASDs (Kennedy & Courchesne, 2008). This network is thought to play an important role in self-other distinction and social cognition (Schilbach, Eickhoff, Rotarska-Jagiela, Fink, & Vogeley, 2008), abilities that are severely compromised in ASDs. It is quite plausible that the difficulties people with ASDs have in deciphering internal states exhibited by biological motion might result from a similar neural dysfunction. Perhaps local neural circuitry that underpins biological motion perception may be normal, but its functional connectivity with the wider social brain or frontal mirror neuron areas may be compromised.

With this in mind, future neurobehavioural studies could compare how people on the autistic spectrum assess the emotional content in different classes of stimuli. For example, does a difficulty assessing the emotional content of faces correlate with a similar difficulty for body movements or gestures? Do such deficits correspond across the visual and auditory domains for all individuals on the autistic spectrum? Such investigations could reveal much about the connectivity between different cortical areas in ASDs and provide evidence for sub-groups of individuals with discrete social cognition difficulties.

## Acknowledgments

We thank Stuart Jackson for his recording of the point-light walker used in this study, and we thank all participants for their time and effort. We thank two anonymous reviewers for comments on an earlier draft. Any errors are ours.

## References

Allison, T., Puce, A., & McCarthy, G. (2000). Social perception from visual cues: Role of the STS region. *Trends in Cognitive Science*, *3*, 267–278.

Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, *31*(3), 137–145.

Attwood, T. (2007). *The complete guide to Asperger's syndrome*. London: Jessica Kingsley Publishers.

Baron-Cohen, S., Leslie, A. M., & Frith, U. (1985). Does the autistic child have a "theory of mind"? *Cognition*, *21*, 37–46.

Baron-Cohen, S., Ring, H. A., Bullmore, E. T., Wheelwright, S., Ashwin, C., & Williams, S. C. (2000). The amygdala theory of autism. *Neuroscience & Biobehavioral Reviews*, *24*, 355–364.

Baron-Cohen, S., Ring, H., Moriarty, J., Schmitz, B., Costa, D., & Ell, P. (1994). Recognition of mental state terms: Clinical findings in children with autism and a functional neuroimaging study of normal adults. *British Journal of Psychiatry*, *165*, 640–649.

Battelli, L., Cavanagh, P., & Thornton, I. M. (2003). Perception of biological motion in parietal patients. *Neuropsychologia*, *41*(13), 1808–1816.

Beauchamp, M. S., Lee, K. E., Haxby, J. V., & Martin, A. (2002). Parallel visual motion processing streams for manipulable objects and human movements. *Neuron*, *34*, 149–159.

Beaumont, R., & Newcombe, P. (2006). Theory of mind and central coherence in adults with high-functioning autism or Asperger's syndrome. *Autism*, *10*(4), 365–438.

Beintema, J. A., & Lappe, M. (2002). Perception of biological motion without local image motion. *Proceedings of the National Academy of Sciences of the United States of America*, *99*, 5661–5663.

Belmonte, M. K., Allen, G., Beckel-Mitchener, A., Boulanger, L. M., Carper, R. A., & Webb, S. J. (2004). Autism and abnormal development of brain connectivity. *The Journal of Neuroscience*, *24*(42), 9228–9231.

Bertenthal, B. I., & Pinto, J. (1994). Global processing of biological motions. *Psychological Science*, *5*, 221–225.

Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2003). Motion perception in autism: A "complex" issue. *Journal of Cognitive Neuroscience*, *15*, 218–225.

Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, *128*, 2430–2441.

Bidet-Caulet, A., Voisin, J., Bertrand, O., & Fonlupt, P. (2005). Listening to a walking human activates the temporal biological motion area. *Neuroimage*, *28*, 132–139.

Blake, R., Turner, L. M., Smoski, M. J., Pozdol, S. L., & Stone, W. L. (2003). Visual recognition of biological motion is impaired in children with autism. *Psychological Science*, *14*(2), 151–157.

Braddick, O., Atkinson, J., & Wattam-Bell, J. (2003). Normal and anomalous development of visual motion processing: Motion coherence and 'dorsal-stream vulnerabliity'. *Neuropsychologia*, *41*, 1769–1784.

Brainard, D. H. (1997). The psychophysics toolbox. *Spatial Vision*, *10*, 433–436.

Brosnan, M. J., Scott, F. J., Fox, S., & Pye, J. (2004). Gestalt processing in autism: Failure to process perceptual relationships and the implications for contextual understanding. *Journal of Child Psychology and Psychiatry*, *45*, 459–469.

Boddaert, N., Chabane, N., Gervais, H., Good, C. D., Bourgeois, M., Plumet, M.-H., et al. (2004). Superior temporal sulcus anatomical abnormalities in childhood autism: A voxel-based morphometry MRI study. *Neuroimage*, *23*, 364–369.

Boria, S., Fabbri-Destro, M., Cattaneo, L., Sparaci, L., Sinigaglia, C., et al. (2009). Intention understanding in autism. *PLoS ONE*, *4*(5), e5596.

Castelli, F., Frith, C., Happé, F., & Frith, U. (2002). Autism, Asperger's syndrome and brain mechanisms for the attribution of mental states to animated shapes. *Brain*, *125*, 1839–1849.

Cherkassky, V. L., Kana, R. K., Keller, T. A., & Just, M. A. (2006). Functional connectivity in a baseline resting-state network in autism. *Neuroreport*, *17*(16), 1687–1690.

Cowey, A., & Vaina, L. M. (2000). Blindness to form from motion despite normal static form perception and motion detection. *Neuropsychologia*, *38*(5), 566–578.

Cutting, J. E., Moore, C., & Morrison, R. (1988). Masking the motions of human gait. *Perception & Psychophysics*, *44*(4), 339–347.

Dakin, S., & Frith, U. (2005). Vagaries of visual perception in autism. *Neuron*, *48*, 497–507.

de Jonge, M. V., Kemner, C., de Haan, E. H., Coppens, J. E., van den Berg, T. J. T. P., & van Engeland, H. (2007). Visual information processing in high-functioning individuals with autism spectrum disorders and their parents. *Neuropsychology*, *21*(1), 65–73.

Del Viva, M. M., Igliozzi, R., Tancredi, R., & Brizzolara, D. (2006). Spatial and motion integration in children with autism. *Vision Research*, *46*, 1242–1252.

DiCicco-Bloom, E., Lord, C., Zwaigenbaum, L., Courchesne, E., Dager, S. R., Schmitz, C., et al. (2006). The developmental neurobiology of autism spectrum disorder. *The Journal of Neuroscience*, *26*(26), 6897–6906.

Downing, P. E., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science*, *293*(5539), 2470–2473.

Field, D. J., Hayes, A., & Hess, R. (1993). Contour integration by the human visual system: Evidence for a local 'Association Field'. *Vision Research*, *33-2*, 173–193.

Freitag, C. M., Konrad, C., Haberlen, M., Kleser, C., von Gontard, A., Reith, W., et al. (2008). Perception of biological motion in autism spectrum disorders. *Neuropsychologia*, *46*, 1480–1494.

Frith, U. (2004). Emanual Miller lecture: Confusions and controversies about Asperger's syndrome. *Journal of Child Psychology and Psychiatry*, *45*(4), 672–686.

Giese, M. A., & Poggio, T. (2003). Neural mechanisms for the recognition of biological movements. *Nature Reviews Neuroscience*, *4*, 179–192.

Goldstein, S., & Schwebach, A. (2004). The comorbidity of pervasive developmental disorder and attention deficit hyperactivity disorder: Results of a retrospective chart review. *Journal of Autism and Developmental Disorders*, *34*(3), 329–339.

Grossman, E., Batelli, L., & Pascaul-Leone, A. (2005). Repetitive TMS over posterior STS disrupts perception of biological motion. *Vision Research*, *45*, 2847–2853.

Grossman, E. D., & Blake, R. (2001a). Brain activity evoked by inverted and imagined biological motion. *Vision Research*, *41*, 1475–1482.

Grossman, E. D., & Blake, R. (2001b). Brain areas active during visual perception of biological motion. *Neuron*, *35*, 1167–1175.

Grossman, E., Donnelly, M., Price, R., Pickens, D., Morgan, V., et al. (2000). Brain areas involved in perception of biological motion. *Journal of Cognitive Neuroscience*, *12*, 711–720.

Happé, F. (1996). Studying weak central coherence at low levels: Children with autism do not succumb to visual illusions. A research note. *Journal of Child Psychology and Psychiatry*, *37*(7), 873–877.

Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences*, *3*, 216–222.

Happé, F., & Frith, U. (2006). The weak central coherence account: Detail-focused cognitive style in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *36*(1), 1–25.

Heberlein, A. S., Adolphs, R., Tranel, D., & Damasio, H. (2004). Cortical regions for judgements of emotions and personality traits from point-light walkers. *Journal of Cognitive Neuroscience*, *16*(7), 1143–1158.

Herrington, J. D., Baron-Cohen, S., Wheelwright, S. J., Singh, K. D., Bullmore, E. D., Brammer, M., et al. (2007). The role of MT+/V5 during biological motion perception in Asperger's syndrome: An fMRI study. *Research in Autism Spectrum Disorders*, *1*, 14–27.

Howard, R. J., Brammer, M., Wright, I., Woodruff, P. W., Bullmore, E. T., & Zeki, S. (1996). A direct demonstration of functional specialization within motion-related visual and auditory cortex of the human brain. *Current Biology*, *6*, 1015–1101.

Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger's syndrome. *Journal of Autism and Developmental Disorders*, *33*(1), 3–13.

Hubert, B., Wicker, B., Moore, D. G., Monfardini, E., Duverger, H., Da Fonseca, D., et al. (2007). Brief report: Recognition of emotional and non-emotional biological motion in individuals with autistic spectrum disorders. *Journal of Autism and Developmental Disorders*, *37*(7), 1386–1392.

Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. *Perception and Psychophysics*, *14*, 195–204.

Jokisch, D., Daum, I., & Troje, N. F. (2006). Self recognition versus recognition of others by biological motion: Viewpoint dependent effects. *Perception*, *35*, 911–920.

Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2006). Sentence comprehension in autism: Thinking in pictures with decreased functional connectivity. *Brain*, *129*, 2484–2493.

Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self- and other-reflection in autism. *Social Cognitive and Affective Neuroscience*, *3*, 177–190.

Kleinhans, N. M., Richards, T., Sterling, L., Stegbauer, K. C., Mahurin, R., et al. (2008). Abnormal functional connectivity in autism spectrum disorders during face processing. *Brain*, *131*(4), 1000–1012.

Klin, A., & Jones, W. (2008). Altered face scanning and impaired recognition of biological motion in a 15-month-old infant with autism. *Developmental Science*, *11*(1), 40–46.

Klin, A., Volkmar, F. R., Sparrow, S. S., Cicchetti, D. V., & Rourke, B. P. (1995). Validity and neuropsychological characterization of Asperger Syndrome: Convergence with nonverbal learning disabilities syndrome. *Journal of Child Psychology and Psychiatry*, *36*(7), 1127–1140.

Koshino, H., Carpenter, P. A., Minshew, N. J., Cherkassky, V. L., Keller, T. A., & Just, M. A. (2005). Functional connecitivity in an fMRI working memory task in high-functioning autism. *Neuroimage*, *24*, 810–821.

Levitt, J. G., Blanton, R. E., Smalley, S., Thompson, P. M., Guthrie, D., McCracken, J. T., et al. (2003). Cortical sulcal maps in autism. *Cerebral Cortex*, *13*, 728–735.

Lord, C., Rutter, M., & LeCouteur, A. (1994). The Autism Diagnostic Interview-Revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, *24*, 659–685.

Loula, F., Prasad, S., Harber, K., & Shiffrar, M. (2005). Recognizing people from their movement. *Journal of Experimental Psychology: Human Perception and Performance*, *31*(1), 210–220.

Macintosh, K. E., & Dissanayake, C. (2004). The similarities and differences between autistic disorder and Asperger's disorder: A review of the empirical evidence. *Journal of Child Psychology and Psychiatry*, *45*(3), 421–434.

Macmillian, N. A., & Creelman, C. D. (1991). *Detection theory: A user's guide*. Cambridge: Cambridge University Press.

Mather, G., & Murdoch, L. (1994). Gender discrimation in biological motion displays based on dynamic cues. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, *258*(1353), 273–279.

Mayes, S. D., & Calhoun, S. L. (2003). Ability profiles in children with autism. *Autism*, *7*(1), 65–80.

Mayes, S. D., Calhoun, S. L., & Crites, D. L. (2001). Does DSM-IV asperger's disorder exist? *Journal of Abnormal Child Psychology*, *3*, 263–271.

Mazefsky, C. A., & Oswald, D. P. (2006). The discriminative ability and diagnostic utility of the ADOS-G, ADI-R, and GARS for children in a clinical setting. *Autism*, *10*(6), 533–549.

McCleery, J. P., Allman, E., Carver, L. J., & Dobkins, K. R. (2007). Abnormal magnocellular pathway visual processing in infants at risk for autism. *Biological Psychiatry*, *62*, 1007–1014.

Milne, E., Swettenham, J., Hansen, P., Campbell, R., Jeffries, H., & Plaisted, K. (2002). High motion coherence thresholds in children with autism. *Journal of Child Psychology and Psychiatry*, *43*, 255–263.

Moore, D. G., Hobson, R. P., & Lee, A. (1997). Components of person perception: An investigation with autistic, non-autistic retarded and typically developing children and adolescents. *British Journal of Developmental Psychology*, *15*(4), 401–423.

Murias, M., Webb, S. J., Greenson, J., & Dawson, G. (2007). Resting state cortical connectivity reflected in EEG coherence in individuals with autism. *Biological Psychiatry*, *62*, 270–273.

O'Riordan, M. A., Plaisted, K. C., Driver, J., & Baron-Cohen, S. (2001). Superior visual search in autism. *Journal of Experimental Psychology: Human Perception & Performance*, *27*, 719–730.

Parron, C., Da Fonseca, D., Santos, A., Moore, D. G., Monfardini, E., & Deruelle, C. (2008). Recognition of biological motion in children with autistic spectrum disorders. *Autism*, *12*(3), 261–274.

Pavlova, M., & Sokolov, A. (2000). Orientation specificity in biological motion perception. *Perception and Psychophysics*, *62*(5), 889–899.

Pellicano, E., & Gibson, L. Y. (2008). Investigating the functional integrity of the dorsal visual pathway in autism and dyslexia. *Neuropsychologia*, *46*, 2593–2596.

Pellicano, E., Gibson, L., Maybery, M., Durkin, K., & Badcock, D. R. (2005). Abnormal global processing along the dorsal visual pathway in autism: A possible mechanism for weak visuospatial coherence? *Neuropsychologia*, *43*, 1044–1053.

Pelphrey, K. A., Morris, J. P., & McCarthy, G. (2005). Neural Basis of eye gaze processing deficits in autism. *Brain*, *128*, 1038–1048.

Pelphrey, K. A., Morris, J. P., McCarthy, G., & LaBar, K. S. (2007). Perception of dynamic changes in facial affect and identity in autism. *Social Cognitive and Affective Neuroscience*, *2*, 140–149.

Peuskens, H., Vanrie, J., Verfaillie, K., & Orban, G. A. (2005). Specificity of regions processing biological motion. *European Journal of Neuroscience*, *21*, 2864–2875.

Pierce, K., Muller, R., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform 'face area' in autism: Evidence from functional MRI. *Brain*, *124*(10), 2059–2073.

Pollick, F. E., Paterson, H. M., Bruderlin, A., & Sanford, A. J. (2001). Perceiving affect from arm movement. *Cognition*, *82*, B51–B61.

Puce, A., Allison, T., Bentin, S., Gore, J. C., & McCarthy, G. (1998). Temporal cortex activation in humans viewing eye and mouth movements. *Journal of Neuroscience*, *18*, 2188–2199.

Puce, A., & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Philosphical Transactions of the Royal Society of London Series B-Biological Sciences*, *358*, 435–445.

Raven, J., Raven, J. C., & Court, J. H. (1998). *Manual for Raven's progressive matrices and vocabulary scales*. Texas, San Antonio: Harcourt Assessment.

Rinehart, N., Bradshaw, J. L., Moss, S. A., Brereton, A. V., & Tonge, B. J. (2000). Atypical interference of local detail on global processing in high-functioning autism and Asperger's disorder. *Journal of Child Psychology and Psychiatry*, *41*, 769–778.

Rinehart, N., Bradshaw, J. L., Moss, S. A., Brereton, A. V., & Tonge, B. J. (2001). A deficit in shifting attention in high-functioning autism but not Asperger's disorder. *Autism*, *5*, 67–80.

Saygin, A. P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain*, *130*, 2452–2461.

Saygin, A. P., Wilson, S. M., Hagler, D. J., Bates, E., & Sereno, M. I. (2004). Point-light biological motion perception activates human premotor cortex. *The Journal of Neuroscience*, *24*(27), 6181–6188.

Schilbach, L., Eickhoff, S. B., Rotarska-Jagiela, A., Fink, G. R., & Vogeley, K. (2008). Minds at rest? Social cognition as the default mode of cognizing and its putative relationship to the "default system" of the brain. *Consciousness and Cognition*, *17*, 457–467.

Spencer, J., & Ó Brien, J. M. D. (2006). Visual form processing deficits in autism. *Perception*, *35*, 1047–1055.

Spencer, J., O'Brien, J., Riggs, K., Braddick, O., Atkinson, J., & Wattam-Bell, J. (2000). Motion processing in autism: Evidence for a dorsal stream deficiency. *Neuroreport*, *11*(12), 2765–2767.

Takahashi, H., Shibuya, T., Motoichiro, K., Sassa, T., Koeda, M., & Yahata, N. (2008). Enhanced activation in the extrastriate body area by goal-directed actions. *Psychiatry and Clinical Neurosciences*, *62*(2), 214–219.

Thompson, J. C., Clarke, M., Stewart, T., & Puce, A. (2005). Configural processing of biological motion in human superior temporal sulcus. *The Journal of Neuroscience*, *25*(39), 9059–9066.

Thompson, B., Hansen, B. C., Hess, R. F., & Troje, N. F. (2007). Peripheral vision: Good for biological motion, bad for signal noise segregation? *Journal of Vision*, *7*(10):12, 1–7, http://journalofvision.org/7/10/12/. doi:10.1167/7.10.12.

Troje, N. F., & Westhoff, C. (2006). The inversion effect in biological motion perception: Evidence for a "life detector"? *Current Biology*, *16*, 821–824.

Troje, N. F., Westhoff, C., & Lavrov, M. (2005). Person identification from biological motion: Effects of structural and kinematic cues. *Perception & Psychophysics*, *63*, 1293–1313.

Tsermentseli, S., Ó Brien, J. M., & Spencer, J. V. (2008). Comparison of form and motion coherence processing in Autistic Spectrum Disorders and Dyslexia. *Journal of Autism and Developmental Disorders*, *38*, 1201–1210.

Vaina, L. M., & Gross, C. G. (2004). Perceptual deficits in patients with impaired recognition of biological motion after temporal lobe lesions. *Proceedings of the National Academy of Sciences of the United States of America*, *101*, 16947–16951.

Vandenbroucke, M. W. G., Scholte, H. S., van Engeland, H., Lamme, V. A. F., & Kemner, C. (2008). Coherent versus component motion perception in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *38*, 941–949.

Vuilleumiera, P., & Pourtois, G. (2007). Distributed and interactive brain mechanisms during emotion face perception: Evidence from functional neuroimaging. *Neuropsychologia*, *45*(1), 174–194.