# Biological Motion Processing in Autism Spectrum Disorders: A Behavioural and fMRI Investigation

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# Abstract

There has been much controversy as to whether people with Autism Spectrum Disorders (ASDs) have a specific impairment in processing biological motion, with some studies suggesting there is an impairment (Blake, et. al. 2003; Klin et. al. 2003, Klin & Jones, 2008, Klin et. al. 2009) and others finding that people with ASDs show intact abilities to detect biological motion and categorise actions, but are impaired in emotion categorisation (Moore et. al. 1997; Hubert et. al. 2007, Parron et. al. 2008). Recent studies have found that although behavioural measures of biological motion processing show no differences, adults with ASDs show different patterns of brain activation to controls in response to intact point-light displays (PLDs), with the STS, MT+ and ITG regions showing reduced activity in this population (Herrington et. al. 2007; Parron et. al. 2009). The current thesis aimed to clarify the nature of these difficulties and to try to elucidate the brain regions used to process configural information from PLDs using novel techniques and stimuli.

The first set of experiments were designed to behaviourally test people with ASDs ability to detect biological motion in noise, to categorise actions and to categorise affect from PLDs. Despite finding differences in the two groups in detection of biological motion and affect categorisation in pilot experiments, there were no significant differences between the groups in the main experiments. However, the ASD group showed slightly poorer performance at detecting biological motion and significantly more variability in the action categorisation tasks, suggesting that there may have been an underlying difference between the two groups. Furthermore, an analysis of the pattern of errors tentatively suggested that the ASD group may be using different strategies to categorise affect than controls, particularly for negative affects.

We then devised a novel technique for manipulating the amount of configural information available in a PLD without the need to add different degrees of background noise and used this technique to assess the contribution of configural cues in a direction discrimination task behaviourally and neurally. The results confirmed that in typically developed individuals configural cues significantly improved the participants' ability to correctly determine the direction of locomotion of a point light walker. Furthermore, the fMRI task found that regions of the inferotemporal, parietal and frontal regions were sensitive to the amount of configural information present in the displays that corresponded to increases in

individual participants' behavioural performance. Lastly, we used the same technique, though with a more powerful fMRI design, to assess the behavioural and neural differences between people with ASDs and controls in response to displays containing different degrees of configural information. We found that both groups were comparable in their ability to discriminate the direction of locomotion from PLDs. However, the brain regions used to process this information were found to be substantially different. In displays in which the configural information enabled participants to accurately judge the direction of locomotion, the control group utilised a similar group of regions as found in the previous experiment. The ASD group showed a pattern of activation suggesting that they predominantly used regions in the temporal and occipital cortex, and more specifically a region in the fusiform gyrus. The results of Granger Causality Mapping analysis, which allows for the mapping of directional to and from seeded regions, confirmed that whereas the control group utilised a network of regions starting from the ITG and connecting to parietal and occipital regions, the ASD group seemed to utilise two separate networks, processing form information in the fusiform gyrus and motion information separately in middle-temporal regions.

The results are discussed in terms of a potential dysfunction of the ITG region in early childhood and two different models of biological motion processing that have been proposed in the recent literature. In TD individuals the model of Giese & Poggio (2003) may be more applicable, in that it proposes the integration of static form cues with motion signals in areas such as the STS. However, a dysfunctional ITG or dysfunctional connections from the ITG to more dorsal regions would disrupt the integration of form and motion processing and force the brain to place additional processing demands on form processing regions in the fusiform gyrus. This would be more in line with the model proposed by Lange and Lappe (2006) in which information can be derived from biological motion in noise without recourse to the actual motion information, through a process of temporal analysis of static postures. Both systems though, may be intact in TD individuals and may share processing requirements depending on the task. Furthermore, it is hypothesised that a dysfunctional ITG may force the brain to place additional demands on regions in the fusiform gyrus and this neural rewiring may be the cause of the developmental delay seen in processing biological motion in people with ASDs (Annaz et. al. 2009). Future studies should examine the roles of the ITG and fusiform area in more detail, both in TD people and in people with ASDs, and determine the specific nature of these neural differences and there behavioural implications for both groups.

## Declaration

I declare that this thesis, submitted to the University of Glasgow for the degree of Doctor of Philosophy, is the result of my own research, except where otherwise acknowledged, and that this thesis has not been submitted for a higher degree to any other university or institution.

Signed: …….
(Lawrie S McKay)

Date: ……..

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# Chapter 1: General Introduction

This thesis focuses on the nature of biological motion processing in people with Autism Spectrum Disorders (ASDs), and the neural symptoms that may underlie any differences in processing between this clinical population and typically developed individuals. In this section some of the current issues in ASD research and the diagnosis of ASD will be discussed, followed by a review of the literature on the visual symptoms experienced by people with ASD, paying particular attention to issues in motion and biological motion perception and the cognitive and neural theories that may account for these issues. Following this review will be an outline the aims of the subsequent chapters.

## 1.1 Issues in ASD research

Autism Spectrum Disorders are typically thought of as primarily affecting social behaviours. These have been characterized as the "triad of impairments", typified by difficulties in social communication, difficulties in social interaction and a limited range of behaviours or interests (Frith, 2003). Further to difficulties in one, or all, of these three categories, a diagnosis of Autism requires a delay in language acquisition or development by 3 years old, which is often one of the first indicators of the disorder. Unlike Autism, Aspergers syndrome does not have the accompanying language delay, though shows similar symptoms in respect to the triad of impairments. The term ASD is used to refer to both autism and Aspergers Syndrome, along with Pervasive Developmental Disorder – Not Otherwise Specified (PDD-NOS). The degree to which these are distinct disorders or part of a common condition is part of an ongoing controversy (Volkmar, State & Klin 2009).

Further controversy concerns whether the prevalence of ASDs is increasing or whether the increase in the numbers is due to improved diagnosis and awareness of the disorders (Charles, Carpenter, Jenner and Nicholas, 2008). On either side of the debate as to whether the prevalence is increasing are those who support a genetic component as a cause for ASDs and those who support an environmental/epi-genetic component as the cause. Those favouring the genetic argument suggest that the rise in diagnosis is due to improved diagnostic techniques, identifying people who in the past would perhaps have been misdiagnosed or not diagnosed at all. To support the view that it is primarily a genetic cause, proponents point to the fact that there is a higher prevalence amongst males in the order of a ratio of 4:1 and also that monozygotic twins are more likely to share the disorder

than dyzygotic twins (Bailey, Le Couteur, Gottesman, Bolton, Simonoff, Yuzda, Rutter, 1995; Baron-Cohen, 2003; Bourgeron, 2008). Those who support environmental causes of ASDs cite environmental stressors, such a pollution, diet and lifestyle (Altevogt, Hanson & Leshner; Rutter, 2009; Thornton, 2006) as causes of a genuine increase in the prevalence of the disorders. It is likely however, that both environment and genetics play a part in the development of ASDs, though whether that means there is a genuine increase in ASDs is still a matter of much debate.

## 1.2 Diagnosis and other issues in Autism Spectrum Disorders

The signs and symptoms of ASDs are almost entirely behavioural and as such, the majority of diagnostic tools are based on either direct or indirect observation of individual behaviour. There are a large number of diagnostic tools such as the Autism Diagnostic Interview (ADI; Lord, Rutter and Le Couteur, 1994), the Autism Diagnosis and Observation Schedule (ADOS; Lord, Rutter, DiLavore and Susan Risi, 2000), the Developmental, Dimensional and Diagnostic Interview (3Di; Skuse, Warrington, Bishop, Chowdhury, Lau, Mandy & Place, 2004) and the Diagnostic Interview for Social Communication Disorders (DISCO; Leekam, Libby, Wing, Gould & Taylor, 2002). Some of these diagnostic tools, such as the ADI, utilise history gathering procedures, usually from parents or primary care givers who have known the individual from infancy. Others, such as the ADOS, primarily use direct observation, as it is independent of third parties. Given the merits of both approaches, usually a combination of tools are used, most notable the aforementioned ADI together with the ADOS, which together form the basis of the "gold standard" in autism diagnosis (Spitzer & Williams, 1988). To meet this gold standard usually requires specialist training and/or collaboration with clinical partners and it can be difficult to obtain all the criteria for an adult population, as the ADI requires developmental history from parents or caregivers who may not be available by adulthood.

Quite often in research however, populations are characterised as "diagnosed by a clinical specialist" or generally classed as having being diagnosed using standard diagnostic criteria such as the DSM-IV. There are also a number of self-report and parental/caregiver report questionnaires that are used widely in research such as the Autism Spectrum Quotient(AQ; Baron-Cohen, Wheelwright, Skinner, Martin & Clubley, 2001b) or the Social Responsiveness Scale (SRS; Constantino, Davis, Todd, Schindler, Gross, Brophy, Metzger, Shoushtari, Splinter & Reich, 2003). As such, the participants in these studies

often have a confirmed diagnosis of autism, but without the aforementioned "gold standard".

Another issue related to diagnosis is what level of functioning within the ASD group should be studied. If a group with more severe autism is to be studied there can be a range of complications for the research project, such as conveying instructions, maintaining attention and recording responses. Furthermore, matching a suitable control group in terms of IQ, verbal IQ and mental age can be almost impossible if they are to be recruited from the general population. Quite often, control groups are taken from another clinical population such as those with Attention Deficit Hyperactivity Disorder (ADHD) or Down's Syndrome. However, the same issues in terms of attention, instructions and recording of responses often are as salient for the clinical control group as they are for the ASD group. If the ASD group to be studied are from a high-functioning autism (HFA) population, meaning one with average or above average IQ, or an aspergers (AS) population, then the need for a clinical control group is often negated as the mental ages (MA), IQs and verbal IQs (VIQs) can be matched from people taken from the general population. However, the limitation of studying HFA and AS groups is that the symptoms are less pronounced and therefore experimental differences between the groups will be smaller.

Even when one has an age and IQ matched population, very often there is a lack of homogeneity in responses within the ASD group (Jones & Klin, 2009). This can be due to the ASD group, although all diagnosed with ASD, experiencing different levels of severity, even within sub-groups such as HFA or AS. Given that primary symptoms of ASDs are social reticence and maintenance of a routine, it can be difficult to find volunteers for studies, and those that do volunteer can have a range of differing symptoms. Even with the most rigorous care in matching samples it can be difficult to find statistical differences between the groups and, if found, to determine whether these differences are due to the disorder, attention, the breaking of routine or a lack of task understanding. This picture is further complicated by the use of medication amongst people with ASDs, typically in adults and usually for a co-morbid clinical condition such as anxiety disorders or depression.

A final, though no less important, point in ASD research is the nature of its unusual "asymmetric" developmental trajectory. Symptoms can change dramatically over time

(Fecteau, Mottron, Berthiaume & Burack, 2003), so that what is true for 8-12 year olds is not necessarily true for adolescents. As such, together with all the aforementioned issues, this makes the study of people with ASDs a very difficult field. This is perhaps most evident in the rather understudied field of sensory symptoms in ASDs, which will be the topic of the next section.

## 1.3 Sensory Symptoms in ASDs

As mentioned, ASDs are typically defined and diagnosed according to social symptoms based on the triad of impairments. However, there is a large body of evidence that demonstrates that there are a wide range of sensory symptoms in ASDs. Sensory symptoms were in fact featured in the original descriptions of ASDs (Asperger, 1944; Kanner, 1943) and were further investigated by Wing(1969) and Hermelin and O'Conner (1970). Furthermore there are a number of self reports by people on the Autism Spectrum detailing sensory disturbances, for example Grandin (1992, 2009), Grandin and Scariano (1986), Jackson (2002) and Williams (1998) and reports by caregivers such as Jackson (2003). Typically, these reports describe both hyper- and hypo-sensitivities to visual, auditory, tactile and gustatory stimuli with the specific pattern differing from individual to individual (Jones, Quigney & Huws, 2003).

Although first hand accounts of these sensory symptoms offer genuine insights into how people with ASDs perceive the world, there are some limitations to the contribution they can make to scientific research. The first is that they typically come from high-functioning individuals and as such, the extent to which they can be applied to individuals with more severe symptoms is limited. Furthermore, the accounts are usually written in conjunction with someone who is not on the autism spectrum, such as is the case of the account by Grandin and Scariano (1986), which opens reports up to interpreter bias. These effects can be compounded by a confusion between "real" versus "echoed" memories and a lack of insight into typical perceptual experiences (Bogdashina, 2003). There are also issues with caregiver reports, which have been widely collected either independently (e.g. Baker, Lane, Angley & Young, 2009; Baranek, David, Poe, Stone & Watson, 2006; Robertson & Simmons, 2008) or together with reports from the individuals themselves (e.g. Leekam, Nieto, Libby, Wing & Gould, 2007). Although these reports suggest similar results, that sensory symptoms are more common in ASDs than in other clinical conditions, that the severity of symptoms declines with age and that these sensory symptoms are correlated

with the severity of the social symptoms, there are issues with observer bias, in terms of unintentionally over- or under-estimating the sensory difficulties (Nader, Oberland, Chambers & Craig, 2004).

Despite these shortcomings however, there seem to be complementary lab-based studies that support the self and caregiver reports of sensory disturbances in people with ASDs. By far, the most studied of these are visual disturbance and these will be the focus of the remaining review, beginning with a short summary of visual symptoms in ASDs.

## 1.4 Visual Symptoms in ASD

There are a range of reported visual symptoms in ASDs. Some come from the aforementioned self reports such as the following extract from Williams (1998)

"*my bed was surrounded and totally encased by tiny spots which I called stars, like some kind of mystical glass coffin. I have since learned that they are actually air particles yet my vision was so hypersensitive that they often become a hypnotic foreground with the rest of 'the world' fading away."* Donna Williams, pp.15, "Nobody Nowhere".

As mentioned in the previous section there are, however, limitations to these self reports. Others have instead listed typically encountered visual sensitivities in people with ASDs, such as Leekam et. al. (2007) and Bogdanisha (2003):

Hyper:

1. Focusing on tiny pieces of dust particles
2. Dislike of the dark and bright lights
3. Dislike of sharp flashes of light
4. Looking down most of the time
5. Covering/closing eyes at bright lights

Hypo

1. Attracted to light
2. Looking intensely at objects or people
3. Moving fingers of objects in front of the eyes
4. Fascination with reflections and/or brightly coloured objects
5. Running hands around the edges of objects

This list is not-exhaustive, but it does give a general idea of the type of visual symptoms that are experienced by people with ASDs. There are also a number of social symptoms that may have a visual component, such as unusual socially directed pointing, difficulties with the interpretation of gestures, unusual eye contact, difficulty with following the gaze of others and difficulties with joint attention. In the following sections studies of visual processing in ASDs will be evaluated.

## 1.5 Studies of Visual Processing in people with ASDs

This section will address a range of studies investigating possible differences in visual processing in people with ASDs and typically developed control populations. Firstly, low-level visual symptoms will be covered, such as optometric issues, contrast sensitivity and spatial vision, then studies of higher level visual processing will be reviewed such as motion and biological motion processing and the processing of faces.

### 1.5.1 Static Contrast Sensitivity

Bertone, Mottron, Jelenic and Faubert (2005) compared contrast thresholds for orientation identification between a group of 13 HFA participants to a matched control population. The stimuli were luminance-modulated (first-order) or contrast-modulated (second-order) greyscale noise. In both cases the stimuli had a duration of 750ms and had a sinusoidal frequency of 0.75 c/deg. To reduce noise in the data due to attentional lapses, the ASD group were carefully guided through the procedure with the experimenter remaining in the room to remind participants to fixate and to initiate successive trials. They found that the ASD group had significantly lower thresholds with the first-order stimuli than the matched controls and significantly higher thresholds for the second-order stimuli.

Sanchez-Marin and Padilla-Medina (2008) measured the detectability of a static bright bar embedded in Gaussian noise in a relatively small sample of 6 participants with autism and 6 controls aged between 7 and 17 years of age. They found that the ASD group performed significantly worse than the control group at a range of signal to noise ratios, and argued that this could reflect the influence of increased levels of internal noise in visual processing pathways in autism. Although interesting, the results of the study are limited by the small sample size and the lack of matching criteria for the control group, which is a particularly salient point as the ASD group had relatively severe symptoms.

In contrast to the aforementioned studies, de Jong, Kemner, de Haan, Coppens and van der Berg (2007) found no significant differences in contrast sensitivity between a group of 29 people with ASDs and an age matched control group. The participants in this study were carefully diagnosed using a range of diagnostic instruments, including the ADI-R and the ADOS. However, the technique used to measure contrast sensitivity utilised the Vistech® contrast sensitivity charts and as such, was not as sensitive as a computer based test would have been. This does not however detract from their findings and suggests that if there are differences between the two groups then these may be small or negligible. The latter is supported by studies by Behrmann, Avidan, Leonard, Kimchi, Luna, Humphreys and Minshew (2006a) and Milne, Scope, Pascalis, Buckley and Makeig (2009) who have also reported no differences in contrast sensitivity for gratings of different spatial frequencies between groups with ASDs and matched controls.

### 1.5.2 Dynamic Contrast Sensitivity

Bertone, et al 2005 also measured contrast thresholds for a flickering grating stimulus using a temporal 2AFC paradigm that utilised a conventional 0.5c/deg grating counterphasing at 6hz in order to stimulate the magnocellular pathway, and a 6c/deg grating counterphasing at 1hz in order to stimulate the parvocellular pathway. They found no significant difference between the two groups in contrast stimuli for either of the two stimuli, suggesting intact dynamic contrast sensitivity in the ASD group. Support for this finding comes from Pellicano, Gibson, Maybery, Durkin and Badcock (2005) who, using a similar design but with a Gaussian blob (3.15 deg sigma) that flickered sinusoidally at 10hz, found no difference between a well diagnosed group with ASDs and a matched control group.

However, Bertone, Mottron, Jelenic and Faubert (2003) found significantly higher thresholds for second-order motion detection, but not first order motion detection in a well defined group with ASDs compared with a typical control group. The stimuli consisted of drifting grating stimuli defined either by luminance for first order motion, or contrast for second order motion. These gratings were either vertical sinusoids, radially symmetrical sinusoids or angled sinusoids. The findings that the ASD group showed higher thresholds for second-order motion detection was independent of the motion type suggesting a pervasive disturbance in dynamic contrast sensitivity. Although this has yet to be

replicated a novel study by McCleery, Allman, Carver and Dobkins (2007) lends some support to the Bertone et al (2003) findings. They investigated contrast thresholds in a high-risk group consisting of 6-months old infants who had older siblings with ASDs. This group is 10-20 times more likely to be diagnosed with ASDs than children with typically developed older siblings (Dawson, Webb, Schellenberg, Dager, Friedman, Aylward & Richards, 2002b; Plomin & McGuffin, 2003) and the study was actually aimed at defining the characteristics of this population before diagnosis was possible. They found that the high risk group had significantly lower motion detection thresholds for grating of .27c/deg drifting upwards and downwards at 15.6deg/s than did the general population. They found no performance differences with similarly drifting isoluminant chromatic red-green gratings and interpreted these results as reflecting differential sensitivities of magnocellular and parvocellular pathways in the high-risk and control groups. Lastly, using a moving version of the bar embedded in Gaussian noise stimuli mentioned in the previous section, Sanchez-Marin and Padilla-Medina (2008) found that, as with the static version of the task, the children with ASDs were significantly worse at detecting the stimulus at a range of signal to noise ratios. However, the same methodological reservations mentioned in the static experiment also apply to the dynamic version of the task.

### 1.5.3 Summary of Contrast Sensitivity

When stimuli are defined by luminance contrast, no study with well matched controls has demonstrated significantly higher contrast detection thresholds in ASD. Lower contrast sensitivity thresholds in people with ASDs has been demonstrated in static contrast tasks (Bertone et. al., 2005) and in a group of high-risk infants to dynamic contrast sensitivity (McCleery, et. al., 2007). Where stimuli are defined by contrast modulation, that is to say second-order, the modulation thresholds of the ASD groups were significantly higher than those of controls (Bertone, et. al., 2003, 2005).

### 1.5.4 Spatial Grouping and Contour Integration

The weak central coherence theory (WCC) suggests that individuals with ASD have difficulty integrating information, including visual information, from different spatial and/or temporal sources. It was first put forward by Frith (1989) and has since been elaborated by Happé and Frith (2006). A number of studies have since investigated the

WCC theory using contour integration tasks similar to those used by Field, Hayes and Hess (1993). In a recent review however Dakin and Frith (2005) criticized a number of these contour integration studies for using oriented line segments, suggesting that these were not true tests of contour integration as they would be detectable by low-pass filtering and as such were effectively contrast detection tasks. These criticisms were aimed at studies by Spencer, O'Brien, Riggs, Braddick, Atkinson and Wattam-Bell (2000), Blake, Turner, Smoski, Pozdol and Stone (2003) and Milne, White, Campbell, Swettenham, Hansen and Ramus (2006), all of which failed to show a significant difference between ASD and control groups. However, two more recent studies using Gabor elements, which are not susceptible to the same criticism, have reported no differences between ASD and control groups at this task (Del Viva, Igliozzi, Tancredi & Brizzorola, 2006; Kemner, Lamme, Kovacs & van Engelund, 2007). A limitation of these studies however, are that only high functioning ASD populations have been used and all of the studies used closed, rather than open contours.

Despite this lack of evidence for poor contour integration in people with ASDs, recent studies have reported deficits in the group for visual form processing. Spencer and O'Brien (2006) used oriented "glass" patterns composed of correlated dot triplets. Structured elements made up of these dot triplets were displayed on either the left or right of the display centre and intermixed with randomly oriented elements that created a "noise" mask. Spencer and O'Brien tested 3 groups of children, a HFA group, an AS group and typical control group matched for Chronological Age (CA) and Verbal Mental Age (VMA). They reported higher thresholds in the HFA group for detecting the structured elements than both the AS and control group. Given the negative findings of the aforementioned studies, this result is surprising and, if replicable, suggests that ability in visual form integration may depend on a person's position on the autism spectrum. However, there are three main limitations of the study. Firstly, diagnosis was simply stated as "met the relevant diagnostic criteria in DSM-IV" and as such there is no way to test whether performance on the test was correlated to the severity of symptoms, which would have been desirable given the differences in performance between the HFA and AS group. Secondly, these Glass patterns, although not made up of line segments, are still susceptible to the criticism of Dakin and Frith (2005) given that there would be low spatial-frequency luminance artefacts. However, unless the HFA group were for some reason insensitive to these luminance cues, the criticism of Dakin and Frith would not explain the difference in performance between the HFA and AS group. Lastly, the

stimulus duration was unusually brief for this type of experiment, being around 250ms. As this is too brief to make stimulus-elicited eye-movements, the problem for the HFA group may have been one of divided attention between the two-halves of the display rather than one of form coherence. Tsermentseli, O'Brien and Spencer (2008) replicated the same pattern as O'Brien and Spencer (2006) in a sample of adults, lending some support to finding that there is a form processing deficit in HFA, but not in AS. It should be noted though, that the criticisms that apply to the Spencer and O'Brien study also apply to the Tsermentseli, O'Brien and Spencer (2008) study.

Support for the findings of O'Brien and Spencer (2005) and Tsermentseli, O'Brien and Spencer (2008) comes from a study by Brosnan, Scott, Fox and Pye, (2004) who looked at a range of tasks in a group of 25 children with autism and 25 CA and VMA-matched controls. They found that in tasks involving gestalt grouping principles of proximity, similarity and closure in simple line and dot figures, the children with autism performed at chance, unlike the control children. Furthermore, the autism group were found to have impairments relative to the control group in identifying impossible figures, though the difference was smaller when reproducing these figures as drawings. A similar result has also been found in adults with HFA (Bölte, Holtman, Poustka, Scheurich and Schmidt, 2007).

### 1.5.5 Susceptibility to visual illusions

It has been suggested that people with ASDs are less susceptible to visual illusions than typical individuals. The first to suggest this was Happé (1996) who claimed that people with ASDs were less susceptible to the Ponzo and Poggendorff illusions, the Ebbinghaus/Titchener circles, the Hering and Kanisza triangle, but not the Müller-Lyer lines and these findings were supported by Bölte et. al. (2007). However, Ropar and Mitchell (1999, 2001) disputed these findings and later commentators such as Dakin and Frith (2005) and Happé and Frith (2006) have proposed that the discrepancy could be due to methodological issues. In particular, in the original Happé (1996) study participants were asked to judge whether different elements in an illusion were "the same" or "different" in an illusion in which two lines appeared different but were actually the same length. As the participants in the ASD group repeatedly reported "the same", this was taken to mean that they were not susceptible to the illusion. However, in the Ropar and Mitchell (1999, 2001) studies, a computer based illusion was used in which participants

could adjust the sizes of the illusory sized elements to match the perceived size of the target element. They reported no significant differences in the susceptibility to the illusion between an ASD group and a control group. Similar findings have since been found by Hoy, Hatton and Hare (2004) and Milne and Scope (2008).

An interesting recent study by Walter, Dasonville and Bochsler (2009) may however shed some light on the discrepancy between the aforementioned studies. Walter et. al. (2009) tested a large sample of 146 undergraduate students on a battery of tasks including psychophysical tests of a number of standard illusions and the Autism Spectrum, Empathising and Systemising Quotient questionnaires (AQ: Baron-Cohen et. al. 2001b, EQ: Baron-Cohen & Wheelwright, 2004, SQ: Baron-Cohen, Richler, Bisarya, Gurunuthan & Wheelwright, 2003) which measure traits that are associated with autism in the general population. The key result was that susceptibility to the Zölner, Rod-and-frame, Roelofs, Ponzo and Poggendorff illusions was related to scores on the SQ in such a way that immunity to these illusions was associated with high scores on the SQ. The systemizing trait is associated with the autism spectrum, but is not a diagnostic feature in itself and as such, suggests that previous results may have been influenced by the prevalence of this trait in the tested populations.

### 1.5.6 Visual Completion

Visual illusions form a way of testing participants' sensitivity to context. Another way to test this is using shape completion in which a shape, usually a circle, is presented partially occluded by another shape, such as a rectangle. After a short break the participant is presented with two alternative shapes and asked to decide which shape matched the partially occluded shape presented previously. DeWit, Schlooz, Hulstijn and van Lier (2007) used a variant of the shape constancy task in which the occluded shape was presented as a prime after which the participant were presented with two different, or identical alternative shapes and asked to report if they were the same or different. Participants would be expected to perform more quickly when the prime matched the targets than when it did not and should therefore provide evidence of different types of shape sensitivity. Unlike the other studies covered so far, the De Wit et. al. (2007) study chose to focus on PDD generally, rather than autism or AS specifically (only 3 of the 16 participants in the clinical group had AS and none had autism). The results suggested that the PDD group, although having equivalent overall reaction times as the control group,

differed in the type of priming stimuli that were effective. De Wit et. al. (2007) claimed that the results demonstrated that the PDD group were able to integrate context so as to complete a partially occluded shape, but that they showed difficulty with unusual, unfamiliar or complex shapes. This may suggest that people with PDD-NOS have greater difficulty in learning novel shapes or a greater sensitivity to differences between them, although it is unknown how this result would translate to groups with autism or AS.

### 1.5.7 Summary of Scene Integration and Context Effects

A number of studies have reported no difference in contour integration between groups with ASD and typical control groups (Spencer, et. al., 2000; Blake, et. al., 2003; Milne, et. al., 2006). Though many of these studies have come under just criticism of their methods, their findings have been supported by studies that do not have their weaknesses (Del Viva, et. al, 2006; Kemner, et. al., 2007). There is some evidence that visual form integration is impaired in people with HFA, but not AS, compared to control populations (Spencer and O'Brien, 2006; Tsermentseli, et. al., 2008; Brosnan, et. al. 2004; Bölte, et. al., 2007), though these studies are also not without some methodological issues. As with the findings on contour and visual form integration, there is mixed evidence for decreased susceptibility of people with ASDs to perceive visual illusions (Happé 1996; Bölt, et. al. 2007; Ropar & Mitchell, 1999, 2001; Hoy, et. al. 2004; Milne & Scope, 2008). However, the discrepancies in the literature may have a specific root in the "systemizing" trait, which is not usually distinctly measured in research populations with ASDs (Walter, et. al., 2009). Lastly, there is some evidence for difficulties in processing novel shapes or an increased sensitivity to the differences between them, (De Wit, et. al, 2007), though these findings at the moment can only be generally applied to people with PDD and the extent to which they can be generalized to autism or aspergers syndrome is, as yet, unknown.

### 1.5.8 Embedded Figures Task

It is a recurrent theme in reports on ASD that both children and adults appear to be sensitive to minute changes in their environments that typical peers fail to detect (Wing, 1976). Although it is difficult to test this in natural environments, the Embedded Figure Test (EFT; Witkin, Oltman, Raskin & Karp, 1971) provides a way of measuring this ability by asking participants to find simple figures, such as a triangle, in a complicated pattern that makes up a real image, such as a pram/baby stroller. Shah and Frith (1983) used this

task to test visual search in a group of 20 children with autism, a learning disabled group matched for MA, and a control group with chronological ages approximately matching the MA of the clinical groups. They found that the autism groups' performance level in the task was not only significantly higher than those found in the control group, but that they were comparable with performance levels that would be expected of children of the same CA, and that detection of the embedded figure was almost immediate and without the need for visual search. These results have since been replicated in a number of studies (Ropar & Mitchell, 2001; Pellicano et. al. 2005; Jarrold, Gilchrist & Bender, 2005; De Jong, Kemner & van Engelund, 2006; Falter, Plaisted & Davis, 2008). A recent study by Edgin and Pennington (2005) tested a group with ASDs and matched controls from ages 7 to 17 using the EFT and found that the performance advantage, measured in reaction times, is largest amongst the younger children and gradually decreases with age until, at the highest age band, there is little or no performance advantage at all.

Joliffe and Baron-Cohen (1997) tested visual search in adults with ASDs and IQ-matched controls using the adult version of the EFT. They asked participants to draw around the embedded figure and used completion time as their measure of performance. In support of Shah and Frith (1986), they found that completion times in the ASD group were twice as fast as those in the control group, although there were no significant differences in overall accuracy. They claim that the key element of the procedure that is required to elicit this difference is to require participants to view and describe the overall design before attempting to locate the embedded figure. This encourages more global processing of the overall design and may impair detailed search in those with a tendency to process more globally anyway. As a follow up to this study, Ring, Baron-Cohen, Wheelwright, Williams, Brammer, Andrew and Bullmore (1999), using the same cohort as in the Joliffe and Baron-Cohen (1997), used fMRI to determine the areas in the brain used when performing the EFT. In the controls they found activation in pre-frontal cortical areas that were not found in the ASD group, whilst in the ASD group they found greater activation in occipito-temporal regions. They suggest that this indicated that while the control group were invoking working memory regions to perform the task, the ASD group were predominantly using visual areas.

Although a number of studies have refuted the findings of the superior performance of individuals with ASD in the EFT task (Brian & Bryson, 1996; Kaland, Mortenson & Smith, 2007; Ozonoff, Pennington & Rogers, 1991; Schooz, Hulstijn, van der Broek, van

der Pijll, Garbeëls, van der Gaag & Rotteveel, 2006), none have provided convincing disconfirmation of the results of either the Shah and Frith (1983) findings or the Jolliffe and Baron-Cohen (1997) findings. Given the results of Shah and Frith (1983) and Jolliffe and Baron-Cohen, together with the findings of Ring et. al. (1999) that the ASD group utilise different brain regions when performing the task, it seems likely that people with ASD do show superior performance in the EFT, though the advantage over controls may decrease with age.

### 1.5.9 Block Design

The block design sub-test (BDT) forms a part of many standardised IQ test batteries (e.g. Wechsler, 1974, 1981), and involves constructing a particular bichromatic design as quickly as possible from a fixed number of identical blocks with bichromatic patterns on each face. Shah and Frith (1993) used the BDT to test participants' ability to break down the global whole, the bichromatic design, into its constituent elements, the various bichromatic patterns on each side of the blocks. They tested 5 different groups on the task: a high IQ(>85) and low IQ(<85) group with a mean age of ~18 years old, a typical IQ group of children with a mean age of ~16 years old, a younger group of typical IQ children with a mean age of ~11 years old and a group of learning disabled individuals with IQs matched to the ASD group and a mean age of ~18 years old. Shah and Frith (1993) found that of all the sub-tasks on the Wechsler sub-tests, the ASD group's performance peaked for the BDT. They then divided the test patterns into 2 categories, those with horizontal and vertical lines only, and those with oblique lines only. These were then presented either at the normal orientation or at an oblique orientation and as either "whole" (undivided) or "segmented" in which the design is partially solved by splitting it into its constituent elements. Shah and Frith found that the ASD groups only performed better than controls when the block design was presented as "whole", suggesting that the ASD groups' superiority was indeed due to a superior ability to break the original pattern down into sub-patterns. A number of recent studies have also supported this finding (Happé, 1994; Ruhl, Werner & Poutska, 1995; Siegel, Minshew & Goldstein, 1996, Ehlers, Nyden, Gillberg, Sandberg, Dahlgren, Hjelmquist & Oden, 1997; Dennis, Lockyer, Lazenby, Donnelly, Wilkinson & Schoonheyt, 1999; Ropar & Mitchell, 2001; Spek, Scholte & van Berckelaer-Onnes, 2008).

Caron, Mottron, Berthiaume and Dawson (2006) used a novel approach to test ASD and control groups using the BDT. They divided the ASD and the control group into two further groups, those with an individual "Block Design Peak" and those without. Forty-seven percent of the ASD-diagnosed group showed such a peak, compared with only 2% of the control group. They also divided up the designs into those with low "perceptual coherence" (PC) in which the solution is easier as resulting from the design having a large number of intensity edges that align with block segmentation points, and high PC designs in which the contours from the blocks combine to make elongated patterns that cross segmentation points, making the solution more difficult to see. The results from Caron et. al. (2006) were similar to those of Shah and Frith (1993) and also highlighted the role of perceptual coherence in the BDT and also the potential role of block design "giftedness", which appears to be more prevalent amongst the ASD population.

Bölte, Hubl, Dierks, Holtman and Poustka (2008) tested an ASD group and a control group matched on NVIQ performing the BDT in an fMRI environment. Despite equivalent behavioural performance Bölte et. al. (2008) found that there was significantly lower activation in the V2v area in the ASD group than in the control group, which they attribute to a reduction in the formation of visual contours in the ASD group.

Overall, there appears to be general support for superior, or at least equivalent, performance in the BDT among people with ASDs and that it is solved in a different way and perhaps using different brain regions than in typical individuals. However, given the complexity of the task and that there are multiple stages involving a range of processing requirements, such as segmenting the image(visual), choosing the correct block design (visual and decision making) and constructing the design (visuo-motor), it is difficult to say from which domain the performance enhancement is derived.

### 1.5.10 Visual Search

The Embedded Figure task and the Block Design Task can both be regarded as visual search tasks. There are however, more direct methods of investigating visual search. Plaisted, O'Riordan and Baron-Cohen (1998b) used two such tasks to investigate visual search in a group of children with ASD and a VMA-matched typical control group. The two tasks they used involved a "feature" search task, in which the target shared colour with a set of distracters but was unique in shape, for instance a green 'T' in amongst a field of

red ‘X’s, and a “conjunctive” search task in which the target shared colour with one set of distracters and shape with another, for instance a green ‘T’ in amongst a field of red ‘T’s and green ‘X’s. Performance on these tasks is measured using the reaction time to find the target and in typical individuals times are longer for conjunctive search than for feature search. Plaisted et. al. (1998b) found that not only was there no significant slowing from the feature search task to the conjunctive search task in the ASD group, but that they were actually faster in the conjunctive search task than controls.

This result was confirmed by O’Riordan, Plaisted, Driver and Baron-Cohen (2001) who, using a control group matched for non-verbal ability, extended it to look at search asymmetries. The task was to find either a tilted line in a background of vertical lines or to find a vertical line in a background of tilted lines. Reaction times were equivalent between the two groups for the former task, but the ASD group showed a considerable advantage over the control group in the latter task. To confirm that the key factor in the superior performance was the ability of the ASD groups to discriminate between the targets and distracters, O’Riordan and Plaisted (2001) used a triple conjunction search task in which the target shared features with 3 classes of distractors, and found superior performance in the children with ASD compared with typical controls and also in adults with ASDs (O’Riordan, 2004).

Even in “difficult” feature search tasks in which the targets look very like the distracters (e.g. a red X-shaped clown amongst green T-shaped and red O-shaped clowns) and conjunction search tasks (e.g. a red X-shaped clown amongst green X-shaped clowns and red T-shaped clowns), there is still superior performance amongst people with ASDs (Jarrold et. al. 2005). Caron et. al. (2006) showed a similar pattern for visual search as they found with block designs, in that participants that showed block design peaks performed both feature and conjunction search faster irrespective of diagnosis.

Neural correlates of this superior performance in visual search were investigated by Keehn, Brenner, Palmer, Lincoln and Muller (2009). They tested a group of adolescents with ASD and a NVIQ matched control group performing visual search in an fMRI environment. They found that, despite only marginally superior performance in the ASD group, the BOLD activations were more extensive in the ASD group, recruiting regions from the frontal, parietal and occipital cortices. The activity found in the control group on the other hand was more confined to occipito-temporal regions.

In summary, there seems to be a large amount of evidence supporting enhanced visual search in people with ASD even in the most complex visual search tasks. This ability may also be a key factor in the seemingly higher levels of performance seen in the embedded figures and block design task, though the fMRI literature seems to suggest that the ASD groups employ rather different networks for each task than the typical groups. The combined literature seems to suggest that there may be some enhanced discrimination of micropattern elements that may, as we will see in the forthcoming sections, prove to be a hindrance in other tasks.

### 1.5.11 Spatial Scale

A number of studies have suggested that global grouping processes are deficient due to the unusual performance in the Navon task (Navon, 1977), in which a large figure such as a large letter 'S' is built up from smaller figures, such as the small letter 'c'. Typical observers find it easier to report the larger than the smaller letters, giving rise to the term "global superiority effect". A number of studies have reported that people with ASDs are more sensitive to the local elements of the display than to the global elements in Navon-type tasks (Rinehart, Bradshaw, Moss, Brereton & Tonge, 2000, 200l; Gross, 2005; Behrman et. al. 2006a), which might be predicted from the aforementioned findings on visual search. However, a number of other studies have failed to replicate this difference (Deruelle, Rondan, Gepner & Fagot, 2006; Edgin & Pennington, 2005; Mottron, Burack, Iarocci, Belleville & Anns, 2003; Mottron, Burack, Stauder & Robaey, 1999b; Ozonoff, Strayer, McMahon & Filloux, 1994, Rondan & Duruelle, 2007).

There is however support for enhanced attention to local stimuli that comes from studies such as that of Jarrold and Russel (1997), in which children with autism tended to count each individual dot rather than rapidly and automatically enumerating them (see also Gagnon, Mottron, Bherer & Joannette, 2004; Mottron, Belleville & Menard, 1999a). Plaisted, Swettenham & Rees, (1999) argued that the finding of local precedence in children with ASD depended on task instructions. In their version of the Navon task there were three different sets of instructions. The participants were told that the stimulus was either at the global level or local level (selective attention task) or gave them no instructions (divided attention task). Plaisted et. al. (1999) found no global advantage in the divided attention task in the ASD group, but that in the selective attention task both groups performed comparably, suggesting that the key problem for the children with ASDs

was in switching attention between the scales and that the default state for the ASD group may be at a finer scale than in typical children.

Rondan and Deruelle (2007) used another variation of the Navon task in which participants were given a choice between matching a pattern with the same spatial configuration but different constituent shapes (configural matching) or the same shape in a slightly different configuration (local matching). Despite showing similar performance on a more standard Navon task, the ASD group, unlike the control group, favoured the local shape match over the configuration match, leading Rondan and Deruelle to suggest that it is configural, rather than global, processing that is disrupted in people with ASD.

Wang, Mottron, Peng, Berthiaume and Dawson (2007) attempted to definitively explore spatial attention in ASD using an exhaustive array of different Navon-type tasks varying on factors such as response type, exposure time and stimulus type. The result of this ambitious study suggested that in ASD there is atypical local-to-global interference and local advantages in incongruent conditions in which local and global stimuli are unmatched. Wang et. al. (2007) argue that this local bias for visual processing gives people with ASD a flexibility that is unavailable to typical individuals and which may sometimes enhance and sometimes diminish performance in visual tasks (e.g. embedded figures or Navon tasks respectively).

## 1.6 Motion Perception

Much of the literature on visual symptoms in ASDs relates to motion perception. There have been a number of reviews on this topic, such as Dakin and Frith (2005), Milne, Swettenham and Campbell (2005) and associated commentaries, most recently Kaiser and Shiffrar (2009). The following sections will cover research into local motion, optic flow, motion coherence, and lastly biological motion.

### 1.6.1 Local Motion

The first to examine local motion processing in ASDs was the aforementioned Bertone et. al. (2003) study, which found that there were no differences between a group with ASD and a control group in first-order motion processing, but that the ASD group had higher modulation thresholds in the second-order direction discrimination task. Methodology and

stimuli similar to that used by Bertone et. al. (2003) were employed by Kogan, Bertone, Cornish, Boutet, Der Kaloustian, Andermann, Faubert and Chaudhuri (2004) to examine local motion processing in Fragile-X-Syndrome (FXS), which is a genetic disorder that shares some of its symptomatology with ASD. They found that, despite the similar symptoms of ASD and FXS, the FXS group in their study showed higher modulation thresholds for local motion direction discrimination for both first- and second-order stimuli, suggesting that the pattern of results found in the Bertone et. al. (2003) study are particular to ASD.

A recent study by Vandenbroucke, Scholte, van Engelund, Lamme and Kemner (2008) examined two grating plaid motion processing in a group of adults with HFA and typical controls. These stimuli consist of overlapping square-wave gratings and can be perceived either as two component gratings sliding over each other or as a single coherently translating plaid patter. They found that the two groups did not differ in the relative amount of time that the plaid was seen moving as a coherent whole, rather than as two transparent components, and also no difference in rivalry rate. Their conclusion was that the two groups did not differ in ability to integrate motion information at low spatial frequencies, though there may be difficulties with higher spatial frequencies such as those used by Bertone et. al. (2003).

### 1.6.2 Optic Flow

*Optic flow* refers to the pattern of dynamic information that is projected onto the retina whenever an individual moves through their environment (Gibson, 1950). The first study to suggest that people with ASDs processed optic flow differently from controls was by Gepner, Mestre, Masson and De Schonen (1996b). They found that when children with ASD were presented with a large expanding or contracting optic flow field whilst standing on a force plate, they showed less postural reaction to the stimuli than did typical controls. This finding was replicated by Gepner and Mestre (2002) in a small number of children with autism and developmental delay. Interestingly, the same study found that a sample of children with AS and typical IQ were *more* posturally reactive than were the controls. Given the small sample sizes only tentative conclusions can be drawn, but they do suggest that people with ASD have abnormal visual motion processing motoric responsiveness to environmental motion.

### 1.6.3 Motion Coherence

Motion coherence refers to the ability of observers to detect small numbers of signal dots moving coherently in a similar direction from a display of randomly moving dots referred to as 'noise' (Newsome & Paré, 1988). Thresholds are defined, typically using a 2AFC task, as the proportion of dots required to be moving coherently before the observer can reliably report the direction of motion, usually up vs. down or left vs. right. The stimuli were originally used to test motion coherence thresholds in macaque monkeys following microscopic lesions in Area V5/MT.

Spencer et. al. (2000) used slightly different stimuli, originally developed by Wattam-Bell (1994) to investigate preferential looking in infants, to investigate motion coherence thresholds in people with ASDs. The technique uses signal dots that oscillate backwards and forwards and noise dots that appear transiently for the same duration in random locations. On one half of the display the dots all move in the same direction and on the other side of the display the dots move in the exact opposite direction. This results in a rather segmented percept that has been described as looking at a "road in a snowstorm" (Atkinson and Braddick, 2005). Spencer et. al. (2000) found that the group of children with ASDs had significantly higher motion coherence thresholds than the CA-matched control group. They also found that motion coherence thresholds decreased between the ages of 7 and 11 in both groups and that the ratio of ASD thresholds to control thresholds remained the same across the ages. Lastly they found that whilst children in the control group reached adult levels by age 11, this was not true of the ASD group. Whether thresholds were consistently higher through to adulthood, or just developmentally delayed, could not be answered by the Spencer et. al. (2000) study as there were no data collected on teenagers.

A criticism levelled at Spencer et. al. (2000) by Milne et. al. (2002), was that they did not match their control population for IQ. To address this, Milne et al. (2002) used a well defined group of children aged 9.5 to 15.5 years of age and a group of controls who were matched for CA and NVIQ. They used stimuli more akin to those developed by Newsome and Paré (1988) with a single, centrally presented region and a motion coherence direction discrimination task. Milne et. al. (2002) found that the performance ratios in terms of mean motion coherence thresholds were similar to those found by Spencer et. al. (2000) and that, although the performance range was higher in the ASD group (6-64%) than in the

controls group (2-29%), the difference was still significant when two of the ASD group who had excessively high thresholds were removed.

An apparently coherent picture of motion coherence thresholds in ASDs seems to from the two aforementioned studies, that juveniles with ASDs have consistently higher motion coherence thresholds. This is consistent with neural theories of ASDs that suggest either Magnocellular pathway or dorsal stream vulnerability (Braddick, Atkinson & Wattam-Bell, 2003; Milne et. al., 2005). Despite this apparent consistency between these two earlier studies, recent studies have complicated their interpretation.

There are two extreme positions amongst the more recent literature. The first, held by Del Viva (2006), is that there are no differences in motion coherence thresholds between groups with ASD and control groups. This conclusion came after testing 3 groups, one carefully diagnosed group with ASD, one matched in CA, and another matched in VMA, using optic flow stimuli first used by Morrone, Burr and Vaina (1995). Motion coherence thresholds were determined for rotational, translational and radial motion and Del Viva et. al. (2006) found no significant differences between any of the groups for each type of motion.

The other extreme amongst the recent papers is represented by Pellicano et. al. (2005), who found highly significant differences between the global dot motion thresholds of their ASD and control groups. Even though the variance within the ASD groups was far larger than in the control group, there was still no overlap between the 95% confidence intervals based on the data. This position is supported by the aforementioned studies of Spencer and O'Brien (2006) who found significantly higher motion coherence thresholds in the children in the HFA group than in their control group, though not their AS group, Tsermentseli et. al. (2008) who found the same pattern in adults and Milne et. al. (2006) who found that a sub-population of their ASD population (around 20%) had motion coherence thresholds outside the typical range. Most recently, Atkinson (2009) found that not only were an ASD group less accurate in a motion coherence task than an CA/VIQ/PIQ-matched control group, but the ASD group also had higher motion coherence thresholds than the control group. So it appears that the data to date gives a rather confusing picture of motion coherence in ASD, with some claiming no difference, others claiming there are partial differences or that differences only pertain to a sub-population, and others claiming that there are large and highly significant differences across the spectrum.

The differences between these studies may have been the source of the conflicting and contradictory pattern of results. As such, some brief consideration will be paid to methodological issues such as the diagnosis of the populations and the matching of the control group, the stimuli used and the experimental design.

### *Diagnosis and Control Matching*

Both the Del Viva et. al. (2006) study and the Pellicano et. al. (2005) study took considerable care in diagnosing their ASD groups, both using the ADI, but only the Del Viva et. al. (2005) using the ADOS in conjunction. Although Del Viva et. al. (2006) matched 2 control groups, one on CA and one on VMA, Pellicano et al. had twice the number of participants matched for NVIQ. Here we find a difference however, in that whereas Pellicano et. al. (2005) matched on NVIQ, which in turn resulted in different VIQ for the two comparison groups, the Del Viva et. al. (2006) study matched on VIQ and as such, there was no difference in verbal abilities between the groups. It seems possible that, despite Pellicano et. al. (2005) checking that the receptive language of their ASD group was sufficient to understand the task, that in some way the VIQ of the groups may have been related to task performance.

### *Stimuli*

The stimuli used by these two studies were substantially different in that, despite both studies using the same number of dots in their display, those of Del Viva et. al. (2006) were black *and* white on a grey background, four times larger in diameter and moving 1.6 times faster than those used by Pellicano et. al. (2005). Furthermore, the total duration of the stimuli used by Del Viva et. al. (2006) was substantially shorter than that used by Pellicano et. al. (2005), being 160ms versus 600ms respectively. The individual dot lifetimes were actually shorter in the Pellicano et. al. (2005) study than in the Del Viva et. al. (2006) study, being only 30ms vs 66ms respectively. This arose from a criticism of the Milne et. al. (2002) study in that the dot lifetimes were too long, which allowed for tracking of the individual dots through consecutive frames. Given these details, it is possible that the shorter durations of the Del Viva et. al. (2006) study could have caused problems for the ASD group. However, another possibility is that the longer lifetime of the dots, the two different colours, the size of the dots and the brief overall presentation

meant that the ASD group were less likely to mis-combine the signal and noise dots. Barlow and Tripathy (1997) discuss and model correspondence noise, which results from unavoidable correspondence between signal and noise points, in these classes of stimuli in considerable detail and the implication for ASDs is that children with ASDs may be more susceptible to correspondence noise than their typical counterparts.

### *Experimental Design*

Both the Del Viva et. al. (2006) and the Pellicano et. al. (2005) studies utilised staircase routines in order to determine motion coherence thresholds. Staircase routines have a larger advantage over method of constant stimuli designs in that thresholds can be estimated much faster using the former than the latter. Their use in ASD research is widespread (Dakin and Frith, 2005), predominantly to minimise attentional demands on the clinical group. Staircase procedures do however have a limitation, in that they are prone to errors from concentration lapses, which can mis-direct the threshold search in an unusual direction. Pellicano et. al. (2005) used a PEST procedure with auditory feedback to converge on the 75% correct threshold and averaged all points following the fourth reversal of the staircase routine to determine the threshold. Del Viva et. al. (2006) used a QUEST procedure to alter stimulus levels with no feedback. However, they then fitted the proportion correct data with a Weibull function and derived the 75% correct thresholds from the curve. As such, the Del Viva et. al. (2006) studies methods can be considered the more robust and reliable of the two designs.

### *Summary of Motion Coherence*

Given the range of findings from previous studies, and the methodological differences between the Pellicano et. al. (2005) and the Del Viva et. al. (2006) studies, it is difficult to form a definitive conclusion as to whether there are motion coherence problems in ASDs. From a strict scientific standpoint, the Del Viva et. al. (2006) study may be the most reliable given their most carefully diagnosed ASD group, carefully matched controls and robust psychophysical methods and, as such, their findings of no differences between the groups should be seen as more correct. The other is to consider the much larger number of studies that have found significant differences, either between both groups or between sub-populations with the ASD group and controls, and to say that a position in between that held by Del Viva et. al. (2006) and Pellicano et. al. (2005), such as that held by Milne et.

al. (2006) and Spencer and O'Brien (2006), is more appropriate. Furthermore the 'purity' of the ASD group and the psychophysical methodology can go a long way to explaining discrepancies between the recent studies of motion coherence in ASD.

### 1.6.4 Biological Motion

Biological motion processing refers to the ability to detect and process information from an animate life form, be it human or animal, and is typically studied using point-light displays (PLDs), which are where the motion of an actor is reduced to moving points of light or dots in place of the major joints and head. There is an increasing literature debating whether people with ASDs have a specific difficulty when it comes to biological motion processing and the issue is almost as contentious as that of motion processing in general.

In typical individuals, the preference for biological motion is evident by 2 days old, with infants preferentially attending to canonical displays of human movements over inverted or random motion PLDs (Simion, Regolin & Bulf, 2008). This sensitivity to biological motion increases with age, such that by 3-months old infants demonstrate equivalent levels of visual sensitivity to phase differences in PLDs of human and animal motion, but by 5 months old infants only respond to phase differences in human stimuli (Pinto, 2006). This sensitivity to human motion continues to be refined with age so that by age 5, children perform at adult levels when detecting unmasked PLDs of human motion (Blake, Turner, Smoski, Pozdol & Stone, 2003; Pavlova, Krägeloh-Mann, Sokolov & Birbaumer, 2001), though the ability to detect PLDs in a noise mask continues to improve into adulthood (Freire, Lewis, Maurer & Blake, 2006).

The first study to investigate biological motion processing in people with ASDs was Moore et. al. (1997). They used PLDs consisting of between 5 and 10 points representing either a person walking or an inanimate household object (e.g. a pair of scissors opening and closing), which were presented in increasing durations from 40ms up to 5000ms. The groups in the Moore et. al. (1997) study consisted of 17 children and young adults with ASDs and a CA and VA matched control group with learning difficulties. The participants' task was simply to say what they thought the points of light were attached to. The number of participants that were able to correctly identify either the person or the household object was recorded for each time level of stimulus duration. These data were then fitted with a psychometric function. Moore et. al. (1997) reported that the ASD group

required slightly longer to correctly identify the PLD when they were only comprised of 5 points, but the difference was non-significant. As such, Moore et. al. (1997) concluded that there were no significant differences between participants with ASDs and the matched controls in their ability to detect biological motion. In a second experiment however, Moore et. al. (1997) found that participants with ASDs were poorer than the controls in spontaneously attributing and describing the affects of point-light actors, but not the mechanical components of actions. These findings have been replicated in children (Parron, Da Fonséca, Santos, Moore, Monfardini, & Deruelle, 2008) and in adults with HFA and AS (Hubert, Wicker, Moore, Monfardini, Duverger, Da Fonséca & Deruelle, (2007).

Moore and colleagues take the findings of their research to reflect intact processing of biological motion in people with ASD, but with an impairment in attributing affect to human gestures. There are a number of limitations of these studies however. The first, levelled at the original Moore et. al. (1997) study, was that the psychophysics used were rather unconventional and that the stimulus presentation method was unorthodox. As mentioned Moore et. al. (1997) repeated each and every stimulus in non-linear increments from 40ms to 5000ms. These were presented sequentially in such a way that presentation times were actually cumulative and the number of people who responded correctly at each level of stimulus duration was recorded. A more robust and methodologically sound technique would have been to present each stimulus until the participant responded correctly and record the time taken, resulting in a reaction time for each stimulus. The second criticism is the manner in which all three studies recorded responses, using free response which is affected by expectancy, attention and motivation, making them unreliable (Blake et. al. 2003).

In an attempt to address the shortcomings of the Moore et. al. (1997) study, Blake et. al. (2003) addressed the issue of biological motion processing using standard psychophysical techniques and a well known procedure of manipulating the phase relations between the points on a PLD in such a way that substantially reduces the perception of the human form (Bertenthal & Pinto, 1994). Blake et. al. (2003) showed 1s displays of intact and phase scrambled PLDs of various everyday actions to a group of children with ASDs and a group of typically developed children whose CA matched the mental age of the ASD group. The participants' task was simply to say whether each display, presented in a random order, was or was not a person. The control participants showed a clear advantage over the ASD

group in discriminating between the two stimuli, with d' scores in the control group being 2.5 times greater than in the ASD group. Furthermore, Blake et. al. (2003) found that the d' scores negatively correlated with symptom severity as measured by the ADOS, the childhood rating scale and, in the ASD group, only MA. More recently, Price, Shiffrar and Kerns (2009) have shown that a group with ASD aged between 8 and 23 were less sensitive to gait coherence than age matched controls.

It seems that biological motion processing may be affected from a very early age in people with ASD. A series of papers from the research group of Klin have shown that infants from 15-24 months old are less responsive to canonical displays of human motion (Klin, Jones, Schultz, & Volkmar 2003; Klin & Jones, 2008; Klin, Lin, Gorrindo, Ramsay & Jones, 2009). Furthermore, Annaz, Remington, Milne, Coleman, Campbell, Thomas and Swettenham (2009) recently showed that typically developed children improve at discriminating 1s displays of intact biological motion from scrambled biological motion between the ages of 5 & 12 and that by aged 12 they are significantly better at this task than children with ASD, who show a flat developmental trajectory.

The most robust finding in terms of biological motion processing in people with ASDs, is that reported initially by Moore et. al. (1997), that people with ASDs have difficulty attributing affect to point light displays of human actions (Hubert, et. al. 2007; Parron, et. al. 2008. A recent paper by Atkinson (2009) showed that even with a forced-choice design rather than free response, people with ASDs were poorer at classifying emotions than a well matched control sample and that this difference was unaffected by stimulus type, i.e. whether PLD or Full Light actions. However, Atkinson also measured motion coherence thresholds and accuracy in a direction discrimination task using Random Dot Kinematograms. Accuracy was lower and motion coherence thresholds were higher for the ASD group than the control group. Furthermore, motion coherence thresholds were inversely correlated with accuracy on the emotion categorisation task, suggesting that deficits in affect judgements are at least partly due to a motion processing deficit.

If this is the case, then from the results of Atkinson (2009), it should follow that if there are low-level motion processing deficits then there should be problems in processing biological motion. This seems to support the previous findings, but the most recent paper, a revisitation of the Blake et. al. (2003) paper by Murphy, Brady, Fitzgerald and Troje (2009), using extremely well controlled PLD stimuli and a direction discrimination task,

found that both a typical control group and an ASD group were comparable across conditions, (intact PLD vs. Scrambled PLD) in terms of both reaction time and accuracy. This led Murphy et. al. (2009) to conclude that the ASD group had no difficulty in integrating local motion signals into coherent human motion. They did find a slightly slower reaction time in the ASD group than the control group that approached significance, a finding that was also found by Freitag, Konrad, Haberlan, Kleser, von Gotard, Rieth, Troje and Krick (2008) and reminiscent of the small effect found by the original Moore et. al. (1997) study. One weakness of the Murphy et. al. (2009) study, that the authors themselves acknowledge, is that the two groups were not matched for intellectual ability. This they say, could account for the slight differences in accuracy and reaction time noticed between the ASD group and controls. Alternatively, the authors acknowledge that it may be a general motion processing issue, though the former account seems more likely even to the authors.

It seems that even in studies that fail to find significant differences between ASD and control groups in action categorisation or direction discrimination, the ASD group usually performs slightly worse than the controls. Two studies that have investigated biological motion processing in ASD and the brain regions involved are those of Herrington, Baron-Cohen, Wheelwright, Singh, Bullmore, Brammer & Williams (2007) and Freitag, et. al. (2008). Both studies found no significant differences between their ASD or control groups on a direction discrimination task (Herrington et. al. 2007) or an action categorisation task. However, both reported reduced neural activity in the ASD group whilst doing the tasks in Superior Temporal Areas, most notable in both studies the STS. The STS, particularly the posterior portion, is a region that has been widely reported to have functions relating to biological motion processing (Beauchamp, Lee, Haxby & Martin, 2002; Grossman, Batelli & Pascaul-Leone, 2005; Grossman & Blake, 2001a, 2001b; Grossman, Donnelli, Price, Pickens & Morgan, 2000; Howard, Brammer, Wright, Woodruff, Bullmore & Zeki, 1996; Peuskens, Vanrie, Verfaille & Orban, 2005; Puce, Allison, Bentin, Gore & McCarthy, 1998; Thompson, Clarke, Stewart & Puce, 2005) and has been implicated in dysfunctional human motion perception in ASD by a number of studies (e.g. Boddaert, Chabane, Gervais, Good, Bourgeois, Plumet et. al. 2004; Pelphrey, Morris, McCarthy & LaBar, 2007; Waiter, Williams, Murray, Gilchrist, Perret & Whitten, 2004).

***Summary of Vision in ASD***

From the reviewed literature on vision in ASDs, it can be seen that in a large number of domains there are certain patterns of impaired or enhanced abilities in processing. However, for each of these specific particularities of visual processing in ASD, such as scene integration or motion coherence, there is widespread evidence for and against the suggested dysfunctions. The literature is consistently inconsistent, varying based on the age of the groups tested, the severity of the symptoms within the ASD group, and the matching criteria for controls as well as factors more familiar to experimental psychologists such as methodology, stimuli and analyses. Overall however, the general picture seems to be that there are certain patterns of differences between people with ASDs and typically developed individuals, though these differences manifest themselves differently across the sub-disorders that make up the autism spectrum and seem to become less pronounced with age.

## 1.7 Theories of ASD

Here we will review some of the current theories of visual processing in ASDs. These include cognitive theories, such as Weak Central Coherence and neural theories such as dorsal stream vulnerability, neural noise theories and mirror neuron dysfunction .

### 1.7.1 Cognitive Theories

***Theory of Mind***

Probably the most famous and so far influential theories of ASD in the past 25 years has been the Theory of Mind (ToM) hypothesis, championed by Simon Baron-Cohen and colleagues at the University of Cambridge (Baron-Cohen, Leslie & Frith, 1985; Baron-Cohen, 1995). The theory initially focused on explaining why children with autism performed poorly on "false-belief" tasks, in which participants were asked to interpret a situation from another persons' point of view. The ToM argued that the primary difficulty in ASD was an inability to read others' minds in social situations, though Frith (2003) prefers to call this ability "mentalizing". The ToM did not have any specific predictions about visual processing, except in terms of social stimuli such as faces, in which it predicted difficulties in processing facial expressions.

Recently however, the ToM has metamorphosed into the "Extreme Male Brain" hypothesis (Baron-Cohen, 2002, 2003), which argues that ASD is characterized as poor "empathizing" and high "systemizing". Empathizing has its roots in the original ToM hypothesis in that it involves interpreting the emotions of others, but extends this to necessitate that these emotions must also be felt by the observer. Systemizing is the ability to analyse rule-based systems and is associated with mathematics, engineering and scientific ability. In visual processing terms, the implications are implicit in that, if someone is a low empathizer and a high systemiser, it would be expected that that person would have a difficulty in recognizing and interpreting complex facial expression, but that they would also perhaps display a certain skill in visual analysis of the components of complex images. The systematizing trait has, as aforementioned, been linked with the ability, or inability, to perceive certain visual illusions (Walter et. al., 2009), though there are other hypotheses that offer alternative explanations for these abilities.

***Weak (Central) Coherence***

The weak coherence theory, originally weak central coherence, first postulated by Uta Frith (1989), was built on the aforementioned local processing biases experienced in people with ASDs, along with their inability to extract the gist or meaning of events in everyday life. Support was derived from the findings of superior performance in the embedded figure task (Shah & Frith, 1983), superior performance on the block design task (Shah & Frith, 1993) and the reduced sensitivity to visual illusions (Happé, 1996). This theory has also recently undergone some revision in three important ways in response to recent empirical findings (Happé & Frith, 2006). Firstly, weak coherence is no longer classed as a core deficit in central processing and is now seen more as a secondary outcome characterized by a local or detail-focused processing style. Secondly, in some situations the local or detail focused processing style can be overcome. Lastly, weak coherence is now classed as only one aspect of a more detailed cognitive profile which includes problems with ToM.

Amongst the most recent cognitive theories of ASD has been that of Executive Dysfunction (Hill, 2004, 2008; Ozonoff, Pennington & Rogers, 1991; Russel, 1997), which focuses on the difficulties that individuals with ASD have with tasks involving mental flexibility, planning, inhibition, generativity and multi-tasking (Hill, 2008). The executive dysfunction theory of autism aimed at explaining all the non-social symptoms seen in people with ASD that were not accounted for by ToM or weak coherence. As with ToM,

there are no specific predictions that can be derived from the executive dysfunction account of ASD, but difficulties with visual attention, scene exploration and eye-movements might be expected.

### 1.7.2 Neural Theories of ASD

A number of researchers have attempted to understand the neural basis of ASDs by comparing its signs and symptoms to those of conditions of known neurological origin. However, this approach has led to a plethora of brain regions being implicated in ASD, including the cerebellum (Courchesne, 1997), the STS and the FFA (Zilbovicious, Meresse, Chabane, Brunelle, Samson & Boddaert, (2006), the Amygdala (Baron-Cohen et. al. 2000; Howard, Cowell, Boucher, Broks, Maves, Farrant & Robert, 2000) and the frontal lobes (Courchesne & Pierce, 2005). Attempts to map the triad of impairments, which is thought to characterize autism, onto specific brain regions has led to the idea of "social brain" dysfunction in ASD (Baron-Cohen & Belmonte, 2005; Frith, 2003). The social brain comprises an even more bewildering array of areas, including the Superior Temporal Sulcus, Superior Temporal Gyrus, Inferior Occipital Gyrus, Fusiform Gyrus, the medial Pre-frontal cortex, the Amygdala, Anterior Cingulate Cortex and the Orbitofrontal cortex and covers functions including facial expression, eye-gaze, the integration of sensory information and executive functioning.

Given these widely distributed regions, researchers have yet to answer the question as to what could cause the unspecified neuropathology in these brain areas or the connections between them. A recent attempt by Baron-Cohen and co-workers suggests that the difficulties results from abnormalities in the levels of hormonal testosterone in the mothers' womb, which result in neural developmental abnormalities. Other ideas include the absence of top-down control (Frith, 2003), malfunctioning social reward mechanisms resulting from dysfunction in the amygdala (Schultz, 2005), abnormal cortical minicolumns (Casanova, Buxhoeveden, Switala & Roy, 2002), neural connectivity abnormalities (Bemonte et. al. 2004; Minshew & Williams, 2007; Rippon, Brock, Brown & Boucher, 2007), imbalanced spectrally times adaptive resonance (Grossberg & Seidman, 2006) and abnormal neural synchronization and inhibition (Rubenstein & Merzenich, 2003).

Given this large number and diversity of theories into the neural basis of ASD it is difficult to derive a common thread amongst them all. Whilst some have been derived based on purely neuroanatomical results (e.g. Casanova et. al. 2002), others have been theory- or data-driven such as Frith (2003) implicating areas that are known to be responsible for top-down control. Following however, are brief summaries of the most recent theories speculating on the neural basis of sensory and other symptoms in ASDs.

***Dorsal Stream Vulnerability***

The "dorsal stream vulnerability" hypothesis (Atkinson & Braddick, 2005; Braddick et. al. 2003; Spencer et. al. 2000) is based on the aforementioned differences in form and motion coherence in people with ASDs and is inclusive of a range of neurodevelopmental disorders such as hemiplegia, FXS, dyslexia and Williams syndrome. The theory postulates that the neural mechanisms supporting global motion processing sensitivity are particularly susceptible to damage because of the more stringent neural timings of this pathway (Atkinson & Braddick, 2005). The theory, rather than specifically aiming to explain autism, is a more general description of potential consequences of developmental disorders which may be linked to underlying neuropathological causes.

If there are motion coherence problems in ASD, then this would be a strong case for a deficit in MT+, which receives a large amount of input from magnocellular pathways. However, there has been no study to date that has specifically explored motion coherence in MT+ in people with ASDs, for instance with Random Dot Kinematograms (Kaiser & Shiffrar, 2009). Furthermore, the support for form and motion processing when taken together, provides an unclear picture of the relationship between dorsal stream functioning and ASD. There has also been recent evidence of ventral stream dysfunction in ASD (Spencer & O'Brien, 2006; Tsermentseli, et. al. 2008) and implications of parvocellular disturbances from studies of colour vision in ASD (e.g. Franklin, Sowden, Burley, Notman & Alder, 2008; Heaton, Ludlow, & Robertson, 2008; Franklin, Sowden, Notman, Gonzalez-Dixon, West, Alexander, Loveday & White, 2009), suggesting that any dysfunction in the dorsal stream is likely accompanied by dysfunction in the ventral stream.

***Enhanced Perceptual Functioning***

An increasingly influential theory has been that of Enhanced Perceptual Functioning (Mottron & Burack, 2001; Mottron, Dawson, Soulitiéres, Hubert & Burack, 2006).

Although sharing much with the weak central coherence theory (Frith, 1989; Frith & Happé, 2006) and the reduced generalization theory (Plaisted, 2001), the origins of the theory are different, being based on work with autistic savants with a particular focus on those with enhanced graphical abilities. The theory states that the key aspect of ASD is a heightened response to sensory stimuli from early childhood (Mottron & Burack, 2001) and has been supported by empirical data from the Montreal-based labs of Mottron, Burack and co-workers (e.g. Bertone et. al., 2003, 2005). They suggest that this heightened response to sensory stimuli in childhood leads to an increased attentional focus towards low-level stimuli and sensory phenomena, which comes at the expense of social interactions leading to atypical rewiring of the brain during neuronal development. Mottron and Burack (2001) claim that this atypical neurodevelopment manifests itself in four main areas; rededication of cortical areas, suppression of inhibition, growth of neuronal connections and "functional persistence", which refers to the excessive refinement of low-level processes at the expense of high level processes.
In the most recent revision of the theory (Mottron, et. al. 2006), "eight principles of autistic perception" are listed along with supporting evidence. These are:

1. The default setting of perception in individuals with autism is more locally oriented than that of typical individuals
2. Increased gradient of neural complexity is inversely related to level of performance in low-level task
3. Early atypical behaviours have regulatory function towards perceptual development
4. Perceptual primary and associative brain regions are atypically activated during social and non-social tasks.
5. Higher-order processing is optional in autism and mandatory in typical
6. Perceptual expertise underlies savant syndrome
7. Savant syndrome provides a model for subtyping Pervasive Developmental Disorders
8. Enhanced functioning of primary perceptual brain regions may account for the perceptual atypicalities in autism

As the theory is relatively recent it is consistent with much of the current literature. However, this recency also means that it has so far escaped empirical testing. It has been carefully distinguished from its predecessors and competitors such as weak coherence and reduced generalization theory by claiming that, unlike weak coherence, the EPF theory

focuses on superior rather than inferior abilities and that it is more comprehensive than the reduced generalization theory. Drawing on Minshew, Goldstein and Siegel's (1997) informational complexity theory the predictions of EPF are enhanced performance on "simple" tasks, such as grating detection (Bertone, et. al. 2005) and diminished performance on "complex" tasks such as second-order motion (Bertone et. al. 2003). However, there are again some problems thrown up by work on colour discrimination in that this task is considered to be low-level, and yet there is no evidence of enhancement of this ability in people with ASDs (e.g. Franklin, et. al. 2009). This highlights a problem for EPF: what constitutes a "simple" or "complex" task? Perhaps a more serious question is, what are the neurological correlates of these eight "principles of autistic perception". These questions remain to be answered, but at the moment EPF is still a broad enough theory to account for many of the empirical findings on sensory processing in ASD.

***Neural Noise Theory***

This theory is a relatively new theory derived from the idea of a "noisy system" and other neural theories of autism that suggest a proliferation of connections within the sensory systems of people with ASDs (e.g. Belmonte et. al. 2004, Casanova et. al, 2002). As mentioned earlier, there is abundant data showing that individuals with ASDs often report hypo- and hyper-sensitivities within the same sensory modality. Furthermore, there are data suggesting that people with ASDs can show enhanced perceptual functioning for first-order contrast stimuli and impaired perceptual functioning for second-order contrast stimuli. Combined with the widely reported heterogeneity within ASD groups, these led Simmons, McKay, McAleer, Toal, Robertson & Pollick, (2007) to suggest that neural noise could account for the complex pattern of enhancements and impairment seen through the ASD population (see also Simmons, Toal, McKay, Robertson, McAleer & Pollick, 2008).

The theory points out that although adding noise to a fixed signal decreases the signal-to-noise ratio, making detectability worse, the phenomenon of stochastic resonance can amplify the signal-to-noise ratio under appropriate circumstances (i.e. a non-linear system with a fixed threshold; Wiesenfield & Moss, 1995). This controversial idea has recently been successfully used to model contrast discrimination data from the typical population (Goris, Wagemans & Wichman, 2008b, Goris, Zaenan & Wagemans, 2008a). If applied to first- and second-order contrast stimuli, the theory would predict that detection of first-order luminance defined gratings in noise would be enhanced in people with ASD through

stochastic resonance mechanisms. However, second order gratings would be harder to detect for people with ASD as they require combination of information from more visual filters, essentially increasing the noise level beyond the point where stochastic resonance can help (Schofield & Georgeson, 1999).

The applications of the theory are quite wide and Dakin and Frith (2005) have already postulated that increased levels of local motion noise could potentially explain the results of the data on motion coherence thresholds in ASD, and a number of others have suggested a role of neural noise in ASD (Rubenstein & Merzenich, 2003; Belmonte & Yugelun-Todd, 2003; Thornton, 2006; Sanchez-Marin & Padina-Marilla, 2008; Alcantara, 2008; Lugo, Doti & Faubert, 2008; Franklin et. al. 2009). Preliminary data suggest that levels of neural noise could be measured in participants with ASD using equivalent noise analysis similar to that used by Dakin, Mareschal & Bex (2005) (Simmons et. al., 2007). The theory also has applicability to the superior performance of people with ASDs in the block design task and embedded figures task, through an amplification of local differences whilst masking global differences, essentially aiding visual search.

The neural mechanisms behind this postulated increase in neural noise was built in to the original theory, coming from work by Belmonte et. al. (2004) and Casanova et. al. (2002) who found increased neural connection in the sensory cortex, raising the possibility of more, but less efficient, crosstalk within the cortex (See Baron-Cohen & Belmonte, 2005; Minshew et. al. 2007 for reviews. Recently there has been genetic work that has suggested that glutamergic and GABA-ergic synapses could be affected in ASD (Garber, 2007; Persico & Bourgeson, 2006), and mis-firing synapses could be another source of neural noise.

The biggest strength of the neural noise theory is its adaptability to explain a range of sensory disturbances in ASD and its neural plausibility. The largest impediment to the theory at the moment is that although it is readily testable using established vision science paradigms, it has, as yet, not been fully empirically tested.

***Mirror-Neuron System dysfunction***

Mirror neurons are a particular type of neuron, first found in area F5 of the monkey brain, that fire whenever the monkey performs an action or observes another monkey or human perform the same action (Di Pellegrino, Fadiga, Fogassi, Gallese & Rizzolatti, 1992;

Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti, Fadiga, Fogassi, & Gallese, 1996a). These mirror neurons respond only when the monkey sees or performs object oriented action (Rizzolatti & Luppino, 2001) and not when the monkey sees an imitated action, the object alone or a nonobject-directed (intransitive) action, though the monkey does not necessarily have to see the whole action if it knows the goal. Neurons with similar properties have also reportedly been found in the monkey STS (Perrett, Harries, Bevan, Thomas, Benson, Mistlin, Chitty, Hietanen & Ortega, 1989; Perret, Mistlin, Harries & Chitty, 1990; Jellema, Baker, Wicker & Perret, 2000) and in the IPL (Fogassi, Gallese, Fadiga & Rizzolatti, 1998; Gallese, Fogassi, Fadiga & Rizzolatti, 2002). These areas form part of what has become known as the Mirror-Neuron System (MNS) and have been implicated in the mediation of imitation (Jeannerod, 1994) and action understanding (Rizzolatti, Fogasssi & Gallese, 2001).

There are no studies that have examined single cell recordings of mirror neurons in humans so most of the evidence comes from non-invasive brain imaging techniques, such as EEG, MEG, Transcranial Magnetic Stimulation (TMS) and fMRI. The earliest evidence of mirror neurons in humans predates the discovery in monkeys. Cohen-Seat, Gestaut, Faure and Heuyer (1954) and Gastaut and Bert (1954) found desynchronisation of mu rhythms that occurred both during active movements of the participant and also during passive observation of movements performed by others. More recently, regions found to activate whilst observing the actions of others have been found in a wide range of visual areas in the temporal, parietal and occipital lobes, but also areas with predominantly motor functions in the inferior part of the pre-central sulcus, the rostral part of the inferior parietal lobule and the posterior part of the IPL (e.g. Rizzolatti, Fadiga, Matelli, Bettinardi, Paulesu, Perani & Fazio, 1996b; Grézes, Costes & Decety, 1998; Grézes & Decety, 2001; Grézes, Armony, Rowe & Passingham, 2003; Grafton, Arbib, Fadiga, & Rizzolatti, 1996; Iacaboni, Koski, Brass, Bekkering, Woods, Dubeau, Mazziotta & Rizzolatti, 2001; Casile, Dayan, Caggiano, Hendler, Flash & Giese, 2009), which together form the basis of the human mirror neuron system (Rizzolatti & Craighero, 2004).

The human mirror-neuron system has been implicated, as in monkeys, with action understanding and imitation (e.g. Buccino, Binkofski, Fink, Fadiga, Fogassi, Gallese Seitz, Zilles, Rizzolatti & Freund; Buccino, Ritzl, Fink, Zilles, Freund & Rizzolatti, 2004). It has been demonstrated that during observed actions, the aforementioned regions along with the STS respond more strongly when participants are asked to imitate the observed action than

when simply observing the same action (Iacaboni et. al. 1999; Buccino et. al. 2004), and that during imitation activation progresses from the occipital cortex to the IPL via the STS, then to the IFG and finally the primary motor cortex (Nishitani & Hari, 2002)

It is possible that this MNS system is in some way disrupted in people with ASDs as there have been a number of studies that have shown impaired imitation in this population (e.g. Avikainen, Wohlschlager, Liuhanen, Hanninen & Hari, 2003; Roger, 1999; Rogers, Bennetto, McEvoy & Pennington, 1996), though there are also several studies that find no such difference (for a review see Williams, Whiten & Singh, 2004). Imitation impairments are particularly salient when examining social functioning in people with ASD, as it is thought to underpin the ability to internally represent the actions of others to facilitate social cognition, empathy, theory of mind and the development of language (Rogers and Pennington, 1991). Recently, Oberman and Ramachandran (2008) have proposed that a dysfunctional MNS, in conjunction with faulty simulator networks covering a range of other areas such as the cerebellum, amygdala, insular cortex and cingulate cortex, results in faulty internal simulation of the actions of others, which in turn could be responsible for the wide variety of symptoms seen in ASDs. In support of a dysfunctional MNS system in autism are findings that mu rhythm suppression is reduced in people with ASD compared with controls when observing actions (Oberman, Hubbard, McCleery, Altschuler, Ramachandrtan & Pineda, 2005), that the temporal progression of activity in the MNS in people with ASDs is delayed relative to controls (Nishitani, Avikainen & Hari, 2004), and reduced activation of the MNS system during imitative finger movements (Williams, Waiter, Gilchrest, Perrett, Murray & Whiten, 2006). Furthermore, there is also evidence that the connections between visual areas and inferior frontal mirror neurons (Villalobos, Mizuno, Dahl, Kemmotsu & Müller, 2005) and between the frontal and parietal areas (Just, Cherkassky, Keller, Kana & Minshew, 2006) may be dysfunctional in people with ASDs. A recent study examined activity in the MNS in a group with ASDs and a matched control group in response to both observation and imitation of facial expressions (Dapretto, Davies, Pfeifer, Scott, Sigman, Bookheimer & Iacaboni, 2005). The results showed that there was a reduction in MNS activity in both tasks and that the activity in the ASD group correlated with symptom severity.

A recent study by Leighton, Bird, Charman and Heyes (2008) suggests that the reported findings of impaired imitation may not be due to impaired MNS functioning. They showed that although people with ASDs were impaired relative to a control group on an

imitative ‘cup and pen’ task, in which participants had to match the action of another, they were equally impaired when performing the same actions in non-imitative version of the same task. Leighton et. al. (2008) argue that this suggests that people with ASDs are poorer on these tasks not due to faulty “mirroring”, but as a result of general non-specific deficits in abilities required for successful imitation, though there is no real suggestion as to what these might be. Hamilton, Brindley and Frith (2007) showed that a group of children with ASDs showed the same tendencies as VA-matched controls to imitate the goals of adults, imitate in a mirror fashion the actions of adults and to imitate grasp in motor planning. As these tasks supposedly rely on the MNS, Hamilton et. al. (2007) claim that there is no general imitative impairment in people with ASDs and also that there is not a global MNS deficit. Recent EEG evidence of an intact MNS comes from Oberman, Ramachandran and Pineda (2008) who showed that mu suppression in a group with ASDs was comparable with control when viewing their own hand or a familiar persons’ hand (e.g. guardian or sibling) performing a grasping action, though not when observing a non-familiar persons hand performing the same action. This suggests that the MNS does respond to observed actions in people with ASD, provided the person being observed is in some way personally familiar to them.

Overall, there seems to be a plausible argument for the dysfunctional MNS hypothesis given the reports of imitation impairments in ASD and the fMRI and EEG evidence in imitation tasks. There also seems to be a potential route for this dysfunction in the link between early visual areas in the occipital cortex and the parietal regions through the STS, which has been implicated as a possible cause of biological motion processing problems in people with ASDs. However, there is also evidence that there may not be a global deficit in imitation and that there may be typical responses of the MNS system in ASDs provided the stimuli are personally familiar to the observer. Although a dysfunctional MNS may be at the root of some general or specific deficits in imitation, empathy or social awareness, given the large number of other brain regions associated with the wide range of impairments seen in ASDs, it seems unlikely that a dysfunctional MNS is the sole cause of deficits in ASDs, or even the majority of them.

## 1.8 The aims and outline of this Thesis

The overall aim of the present thesis is to examine biological motion processing in people with ASDs using novel behavioural, psychophysical and fMRI techniques. Given that the data on biological motion processing in ASDs suggest deficits in action categorisation, low-level processing of gait and the categorisation of emotion and that the presence of these deficits is manifest in the groups used in some studies but not in others, the current thesis will utilise a number of tasks that cover a range of processing requirements and use the same group of well defined people with ASDs and matched controls. It is hoped that the work presented in the coming chapters will shed light, not only on the behavioural differences between people with ASDs and typical controls in processing and using information from biological motion, but also the pattern of underlying neural networks that are responsible for these differences. Below is a brief outline of the aims of each of the subsequent chapters.

In Chapter 2 we use a battery of experiments to investigate the detection of biological motion, the categorisation of human actions and the categorisation of affect in people with ASDs and a control population. The aim was to use novel stimuli, combined with well controlled psychophysical and statistical methods, to determine whether people with ASDs showed impairments in the range of domains suggested by the previous literature. Prior to conducting experiments on a large sample of well defined people with ASDs, pilot versions of each experiment were run on a small sample of people with ASDs and a control group. Based on the pilot data and comments gathered from international meetings the methods for each experiment were adjusted for the main experiment. The data from both the pilot experiment and the main experiments are presented in Chapter 2 and the results are discussed in relation to the previous literature.

In Chapter 3 we devise and test a novel technique for quantifying the contribution of configural information from point-light displays, with a view to using it to examine biological motion processing in people with ASDs in an fMRI design. Typically masking the structural information that gives rise to the percept of a walker in PLDs requires large numbers of additional ‘noise’ points. This technique, although used in Chapter 2, posed two particular problems. Firstly, the addition of noise points greatly increases the stimulus complexity such that any group effects observed could potentially be confounded by the increase in task demands, especially in a group with attentional difficulties such as ASDs.

Secondly, the addition of noise masks is unsuitable for use in an fMRI task, as the increase of global motion in the stimuli resulting from the increases in the number of masking points excites motion sensitive areas such as MT+. This increase in MT+ activation could, in theory, cascade through other biological motion processing areas making it difficult to determine whether group differences were due to disrupted biological motion processing specifically or more general motion processing. The technique resolved these issues and is described in full in Chapter 2. The results of the experiment are described in relation to the literature on configural processing of biological motion in typically developed adults.

The aim of Chapter 4 was to test the suitability of the methods devised in Chapter 3 for differentiating the brain regions responsible for processing biological motion processing. As well as replicating the behavioural results from Chapter 3, individual motion coherence thresholds were derived for use in the fMRI experiment. These individual thresholds were used to determine the stimuli levels that participants saw in the fMRI task, ensuring that all participants were achieving equivalent performance inside the scanner. Furthermore, the experimental design for the fMRI task was optimised using novel approaches that are described more fully within the chapter, with the purpose of not only determining the regions that are important in biological motion processing, but also how these regions influence each other using Granger Causality Mapping (GCM). The results of the fMRI task are discussed, both in relation to the literature on the neural basis of biological motion processing in typically developed adults, and also in relation to the suitability of the method for use in examining the neural basis of biological motion processing in people with ASDs.

In Chapter 5 a revised version of the fMRI experiment was designed and implemented in a group of high functioning adults with ASDs and an age- and IQ-matched control group. The revised design addressed the limitations of the previous experiment outlined in the discussion of Chapter 4. The participants were tested both on the behavioural task from experiment 3 and the new fMRI task, with the purpose of assessing any behavioural differences between the two groups when processing biological motion and also the underlying neural differences between the groups. The results of these experiments are discussed in relation to the literature on biological motion processing in ASDs and in a framework of the potential neural theories of ASD that could account for these differences.

In Chapter 6 the results of all the experiments are summarised and discussed in relation to each other, with particular emphasis being placed on combining the results from Chapter 2 and 5 to understand the nature of biological motion processing in people with ASDs and the underlying neural differences between the two groups tested. Particular attention will be paid to the contribution that the current thesis makes to the literature in the field and the future directions that research should take when investigating biological motion processing in ASDs.

# Chapter 2: Detection of Biological Motion, Action Categorisation & Affect Categorisation in Autism Spectrum Disorders

## 2.1 Introduction

Autism Spectrum Disorders (ASDs) are typified by impairments in social interaction, repetitive patterns of interest and behaviour and, in autism but not necessarily aspergers syndrome (AS) or PDD-NOS, abnormal language development (DSM-IV, 1994). In addition to these symptoms, which are the principal criteria used in the diagnosis of ASDs, there are a number of sensory abnormalities reported by people with ASDs (Grandin, (1992, 2009), by parents or caregivers (e.g. Jackson, 2003) and which were reported in the original accounts of ASDs (Asperger, 1944; Kanner, 1943). Although these disturbances manifest as hyper- and hypo-sensitivities in a range of domains (Jones et. al. 2003), the visual domain has been the most extensively studied.

There have been a number of studies that demonstrate a local processing bias in people with ASDs in the visual domain (Happé, 1999) that result in superior performance in behavioural tasks requiring local processing and poorer performance when global processing is required (see Happé & Frith, 2006 for a review). These processing biases result in reduced susceptibility to a range of visual illusions such as the Ponzo and Poggendorff illusions, the Ebbinghaus/Titchener circles, and the Hering and Kanisza triangles (Happé, 1996; Bölte et. al., 2007), though there may be sub-groups within the ASD population that are particularly affected (Walter et. al., 2009). This inability to integrate local features into global percepts also seems to be present in motion processing in people with ASDs. A number of studies have reported that motion coherence thresholds are significantly higher in people with ASDs than in typically developed individuals (e.g. Spencer et. al. 2000; Milne et. al. 2002; Spencer & O'Brien, 2006; Pellicano, et. al. 2005; Tsermentseli et. al. 2008; Atkinson, 2009), though, as mentioned in the previous chapter, there is still considerably debate as to whether these effects are indicative of ASDs.

Another situation that requires local features to be integrated into a global percept is in perceiving biological motion. Typically biological motion is studied using point-light displays (PLDs). These were first created by Johansson (1973) who showed that when

human motion is represented as points of light attached to the major joints of the body and head, observers perceive these local motion signals as being a coherent human form. The robustness of this integration is evidenced by studies showing that the human form can still be perceived in large amounts of masking noise (Cutting, Moore & Morrison, 1988; Bertenthal & Pinto, 1994) and in the absence of local image motion (Bientemma & Lappe, 2002), though it is disrupted when the PLD is inverted (Pavlova & Sokolov, 2000). These studies all lend support to the idea that, like faces, human bodies may be processed as a global configuration. Furthermore, a range of socially salient information can be extracted from PLDs of humans, including affect (Pollick, Patterson, Bruderlin & Sanford, 2001), gender (Mather & Murdoch, 1994), personality traits (Heberlein, Adolphs, Tranel & Damasio, 2004) and identity (Jokisch, Daum & Troje, 2006; Troje, Westhoff & Lavrov, 2005).

Given the range of socially relevant cues that can be extracted from biological motion it seems plausible that a deficit in perceiving biological motion could form the basis of, or contribute to, some of the social difficulties experienced by people with ASDs. A number of studies have investigated biological motion processing in people with ASDs, in domains ranging from low-level feature processing to action and affect categorisation, all with mixed results. This has resulted in controversy as to whether biological motion processing is impaired in people with ASDs and contributes to the social difficulties seen in this group, or whether it is intact and the social deficits result from dysfunction in a more cognitive top-down processing system.

The first to tackle this issue was Moore et. al. (1997) who compared a group of children with ASD and a CA- and VA-matched group of children with learning difficulties in a task requiring the participants to identify PLDs of humans and non-biological moving objects. They found that the exposure time required to identify both the human PLDs and the non-biological PLDs was equivalent in both groups, though the required exposure times for the human stimuli were slightly longer in the ASD group than in the learning impaired group. Furthermore, despite being equivalent at identifying the human PLDs, the ASD group were significantly poorer than the learning disabled group at recognising emotional states, but not at describing the mechanical components of the emotional actions. As such, Moore et. al. (1997) concluded that biological motion processing was intact in children with ASDs and was not the root of the social processing deficits seen in the same group.

However, a number of criticisms have been levelled at the Moore et. al. (1997) study. Firstly, the stimulus presentation method was rather unconventional, in that the stimuli were presented in a blocked fashion, with the same stimuli being shown in blocks of increasing stimuli duration, resulting in cumulative exposure times rather than reaction times. Secondly, the psychophysical methods based on this cumulative exposure time were also rather unorthodox, with the number of participants correctly identifying the stimuli at each stimulus exposure time being modelled with a psychometric function. As such, the same participants would be present at multiple stimulus levels, with all being included at the longest duration. Thirdly, the Moore et. al. (1997) study used free response, which can be affected by expectancy, attention and motivation (Blake et. al. 2003). Lastly, as the stimuli were essentially intact PLDs the ability of the ASD group to integrate local biological motion signals into the percept of a human was not tested (Murphy et. al. 2009).

To address the shortcomings of the Moore et. al. (1997) study, Blake et. al (2003) combined standard psychophysical methods with a well known technique of manipulating the phase relations between the points of a PLD, which results in the substantial reduction of the global percept of a human form (Bertenthal & Pinto, 1994). Blake et. al. (2003) compared a group of children with ASDs and a group of typically developed CA-matched controls on a task involving the discrimination of intact and phase scrambled PLDs of human actions. The participants simply had to say whether the stimulus was or was not a person, and d' scores were calculated for each participant. Blake et. al. found that the d' scores were 2.5 times higher in the control group than in the ASD group and that for both groups these scores correlated highly with measures of symptom severity. This suggests that the children with ASDs were poorer at integrating the local motion signals into the global percept of the human form. A more recent study has shown that the ability of typical children to discriminate between intact and scrambled PLDs improves between the ages of 5 and 12, whereas children with ASDs show a flat developmental trajectory on the same task (Annaz et. al. 2009). Furthermore, the lack of sensitivity to biological motion has been demonstrated in autistic children as young as 15 months old, who are less responsive to canonical PLDs of human motion than are typically developed children (Klin et. al 2003, 2008, 2009), suggesting that the salience of the human form may be reduced from infancy in people with ASDs.

A number of studies however, have found that there are no biological motion processing deficits in groups with ASDs. Using very similar methodologies Hubert et. al. (2007) and Parron et. al. (2008) found no significant differences between groups with ASDs and controls in children and adults respectively. They found that their respective ASD group were comparable with controls in identifying subjective states or actions from PLDs of humans and PLDs of non-biological everyday objects. They did however, report that the ASD group were impaired at identifying the correct affective states from PLDs of human actions. These studies both suffer from some of the same criticisms that apply to the Moore et. al. (1997) study, most notably that they both used free response. Using a forced choice paradigm a recent study by Atkinson (2009) found that affect categorisation of PLDs was impaired in people with ASD for happy and angry affects, but not fear or sadness. Unlike the studies of Hubert et. al. (2007) and Parron et. al. (2008) however, Atkinson (2009) also found that the ASD group were significantly poorer at correctly labelling actions from PLDs, regardless of whether they were instrumental or non-instrumental actions. Atkinson (2009) also tested both groups using a psychophysical motion coherence paradigm, and found that the ASD group were poorer in accuracy and had higher overall motion coherence thresholds than the control group. It was also reported that those with the highest motion coherence thresholds performed the worst in categorising affects, but only within the ASD group. Atkinson argues that the processing of affect is more reliant on global motion and form processing than action identification, which can be accomplished using relatively local motion and form cues. As such, a low level motion integration deficit could account for the poor performance in the affect categorisation task, whilst allowing for a spared ability to categorise non-emotional actions.

The current study aimed to investigate biological motion processing in a group of people with ASDs and a control group using 3 experiments that each required different levels of processing. The first experiment utilised a simple biological motion detection task in which participants had to detect a coherent walker in a field of randomly positioned noise, which was generated from the same points that made up the walker. In this task the key processing requirement was to integrate the correct local motion signals of the individual joint locations, of which there were between 3 and 15, into the global percept of a human walker. In essence we aimed to compare noise tolerance thresholds for each group at each stimulus level to determine if the ASD group were impaired relative to controls in integrating these local motion signals. If the ASD group were impaired in global motion or

biological motion processing, as suggested by the aforementioned studies of Blake et. al. (2003), Annaz et. al. (2009), Klin et. al. (2003, 2008, 2009) and Atkinson (2009), then it would be expected that the ASD group would have lower noise-tolerance thresholds at each stimulus levels than the control group. The aim of the second experiment was to investigate action categorisation in people with ASDs using stimuli in which two point-light actions were blended together in varying amounts, creating stimuli that varied in the degree to which they appeared to be a knock or a lift. The task was simply to say whether the viewed action was a knock or a lift. From Atkinson (2009) we would predict that the ASD group and the control group should perform comparably and show the same subjective biases, as the task can be accomplished using relatively local motion and form cues. The final experiment aimed to examine the categorisation of affect from PLDs of throwing actions performed as either angry, happy, neutral or sad. According to Atkinson (2009) the processing of affect requires global motion and form processing. As such, it would be predicted that the ASD group should be less accurate at correctly identifying the affect of the throwing movements, particularly for the angry and happy actions. For all three tasks a pilot experiment was conducted on a small sample of people with ASDs and controls prior to running the main experiments. The data from these pilot experiments were presented at various international meetings, following which, the main experiments were redesigned based on the comments gathered. For completeness, the methods and results of both the pilot and the final versions of each experiment are included in this chapter.

## 2.2 Methods

### 2.2.1 Task 1: Detection of Biological Motion

#### 2.2.1.1 Participants:

*Experiment 1:* The participants in the ASD group consisted of 5 adult males aged between 18 and 25 who had been diagnosed independently as having autism or aspergers syndrome, and who had also met the required criteria to be classed as having ASD on the Social Responsiveness Scale (SRS) by Constantino, Davis, Todd, Schindler, Gross, Brophy, Metzger, Shoushtari, Splinter, Reich (2003) and the Autism Spectrum Quotient(AQ) by Baron-Cohen, Wheelwright, Skinner, Martin, and Clubley (2001). In addition, four of the

five participants had undergone clinical assessment specifically for the purpose of the study and had been confirmed using the Autism Diagnosis Interview-Revised, known as the ADI-R, (LeCouteur, Rutter, Lord, & Rios, 1989; Lord, Rutter, & Le Couteur, 1994) as having an ASD. All participants can be considered to be of average or above average intelligence, as four of the five have attended university and the fifth attended a school for the academically gifted. Control participants consisted of 5 age-matched males between the ages of 18 and 25 and all were university students.

*Experiment 2:* The participants in the ASD group consisted of 10 adult males aged between 18 and 36, who had been diagnosed independently as having autism or aspergers syndrome. Of these 10 participants, all had a confirmed autism spectrum diagnosis. Furthermore, 3 of the 10 participants had undergone further clinical assessment in the form of the ADI-R (Le Couteur et. al 1994) and the Social Responsiveness Scale (Constantino et. al. 2003) and had also met diagnostic criterion on these measures. The remaining 7 of the 10 were unable to complete these assessments due to a lack of available developmental history. Each participant also underwent the Wechsler Abbreviated Scale of Intelligence (WASI) test as an estimate of verbal, performance and full-scale IQ. This is an abbreviated version of the Wechsler Adult Scale of Intelligence (WAIS) and the scores correlate well between the two. There were originally 12 participants recruited for the study. However, 2 had to be excluded due to non-typical FSIQs.

Control participants were 10 age- and FSIQ-matched adults who were confirmed as not being on the Autism Spectrum as determined by assessment using the AQ with a cut-off of 18, which ensured all controls were either in the "average" or "below average" categories in terms of autistic traits measured by the AQ.

Table 2.1: Means and SDs of the age in years and IQ for each group.

| | AGE | | IQ | |
|---|---|---|---|---|
| **Group** | **MEAN** | **STDEV** | **MEAN** | **STDEV** |
| ASD | 27.70 | 6.09 | 126.10 | 5.43 |
| Control | 27.70 | 6.06 | 125.78 | 7.46 |

Paired samples t-tests confirmed that there were no significant difference between the two groups for either age ($t(9) = 0.00$, $p = 1.00$) or IQ ($t(9) = 1.11$, $p = .30$).

#### 2.2.1.2 Design

The design for Experiment 1 & 2 was identical and so shall be covered only once. A two-interval two-alternative forced choice task was used in which participants had to determine which side of a display containing randomly positioned points of biological motion contained a coherent human walker. We tested participants at 7 levels of signal strength (3, 5, 7, 9, 11, 13, 15), which were the number of walker points used to make up the coherent walker out of the possible maximum of 15.

For each level of signal strength, a QUEST paradigm (Watson & Pelli, 1983) was used to determine the number of noise points that were required for participants' performance to reach the 75% correct thresholds. This was implemented using Matlab and the psychophysics toolbox (Brainard, 1997; Pelli, 1997).

The QUEST procedure uses an adaptive Bayesian technique to estimate a psychometric function based on a set of priors. For this experiment the QUEST procedure estimated the required number of signal points required for performance to be at the 75% correct threshold from the estimated psychometric function and then showed stimuli with that number of signal points. The participants' response was then recorded as correct or incorrect and fed back into the algorithm. Using this information the QUEST algorithm updated its estimate of the psychometric function and its estimate of the signal strength required to produce performance at the desired threshold level. This procedure continued for, in our experiment, 40 trials and in each trial the psychometric function is more accurately predicted based on the accumulated data from the previous trials, eventually converging on the most probable signal strength for the desired performance threshold. The QUEST procedure was used, rather than a method of constant stimuli approach, as it was unknown how many noise points would be needed to mask the walker in either of the two experimental groups. In order to have fully tested this, it would have been necessary to test a large range of noise masks and given the attentional and cognitive demands of such a task for a TD participant, it was felt that this would have been too much for an ASD group. As an illustration, if a method of constant stimuli design had been used with 20 trials per level and a maximum number of 100 noise points, then as many as 14000 trials may have been needed. The QUEST procedure was able to determine the 75% correct thresholds over forty trials for each stimulus level, giving a far more acceptable total of 280 trials.

#### 2.2.1.3 Stimuli:

The stimuli were identical in Experiment 1 and 2 and as such will only be covered once. Stimuli were presented on a CRT monitor with a resolution of 1280 x 1024 pixels, on a display size subtending 22.62° of visual arc by 17.06° at a distance of 1m from the participant, with a refresh rate of 60 Hz. All point-light displays used were generated from an existing motion capture library (Ma, Patterson & Pollick, 2006). Using data from earlier experiments on the distinctiveness of human movements, a distinctive walker was chosen from within the motion capture library to ensure that the target stimuli were easily detectable. The walker was scaled to a height of 200 pixels, subtending a visual angle of approximately 1.47° x 3.36°. The number of points from the walker that were displayed during the experiment was systematically varied between 3 and 15 in increments of two. The points displayed at each stimulus level were randomly sampled from the 15 available in the full point-light display, and were displayed on either the left or the right side of a horizontal rectangular display window subtending approximately 8.93° x 4.19° (500 x 250pixels). On whichever side of the display the walker was not present, a scrambled walker comprised of the same points as the target walker was presented. The local motions of these points were spatially scrambled to new joint locations such that no point maintained its original starting position. This ensured that in the initial frame the target walker and the scrambled walker were indistinguishable, and that the dot densities in the region of the walkers were the same on the left and right side of the display. This prevented participants from detecting the target walker based on an increased number of points or dot densities on one side of the displays, and that the only way to distinguish the target walker was by using configural cues derived from the relationship between the local motion signals. The position of the target walker and the scrambled walker was horizontally and vertically jittered by up to 0.78° and 0.67° (40 pixels) respectively from the centre of the display. The amount of spatial jitter was determined randomly to ensure that participants were unable just to focus on the centre of each side of the display and had to engage in visual search.

On both sides of the display noise masks were added that were sampled randomly from the walker points. These noise points were sampled from the point-light walker and randomly positioned within the bounds of the display window (See Figure 2.1).

This noise was based upon the technique developed by Hiris, Humphrey, & Stout (2005). Using biological motion derived from the target walker as a noise mask ensured that participants could not just look for a local biological motion signal in random noise, and therefore were forced to detect the coherent walker signal.

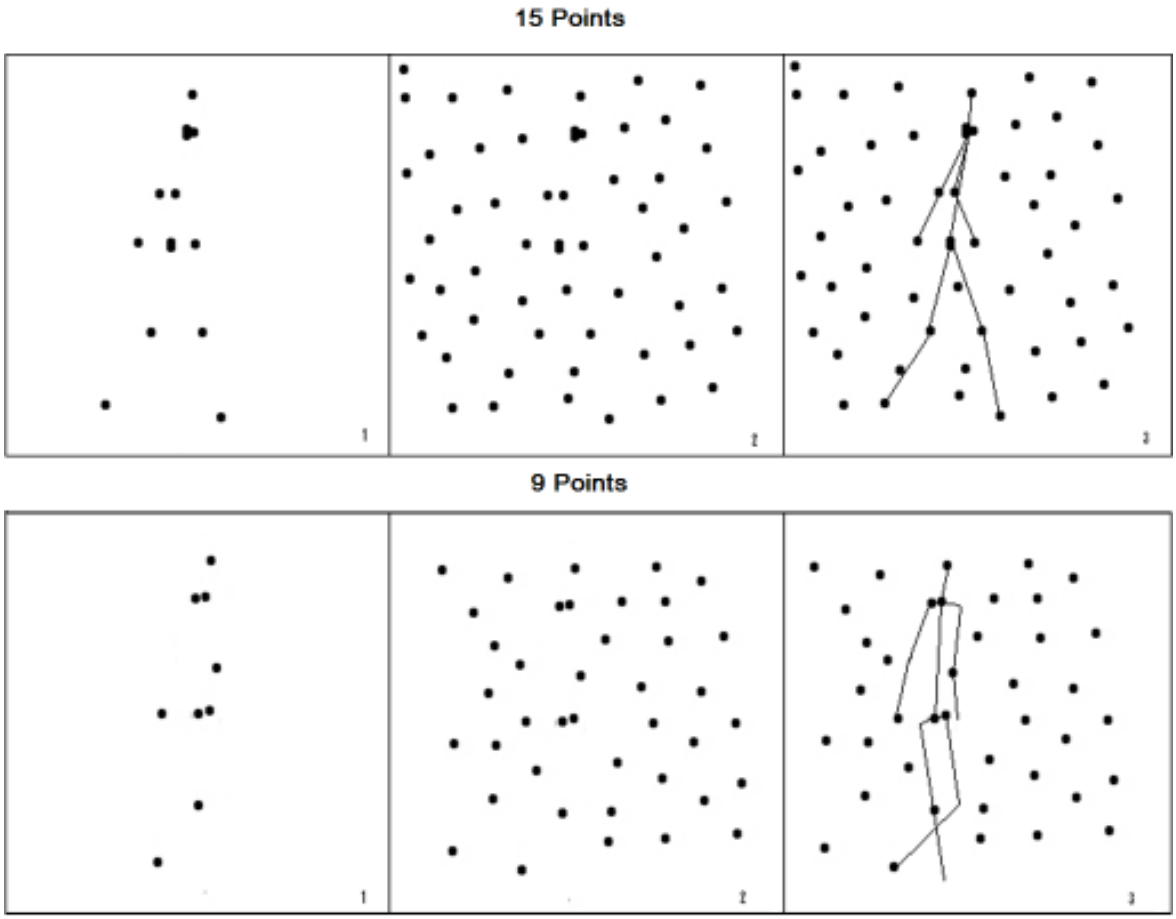


Figure 2.1: Three panels showing the noise masking technique for target simuli of 15 and 9 signal points. Panel 1 - Point light walker consisting of 9 points. Panel 2 - Actual Stimuli: Same walker embedded in noise. Panel 3 - Same walker in noise with lines connecting joints and showing where missing points are in space.

### 2.2.1.4 Procedure:

The procedures were identical for Experiments 1 and 2 and as such shall only be covered once. Participants were positioned across from a monitor with a viewing distance of approximately 1 metre. They were told that they would see a display of moving dots and that on either the left or right of that display a proportion of the dots would show the movement of a human walker and that the contralateral side would contain only randomly moving dots. Participants were asked to say whether the walker was on the left or right of the display. Before the experimental trials the participants were shown three practice

trials, which had varying numbers of signal points and noise points to ensure they were comfortable with the task. If participants had difficulty, the practice trials were repeated until the experimenter was satisfied that they were able to perform the task. No participants required more than 2 repetitions of the practise trials. The task took around 40 minutes to complete and afterwards participants were fully debriefed.

## 2.2.2 Task 2 – Discrimination between two instrumental blended actions

### 2.2.2.1 Participants

The participants in both experiments in Task 2 were the same as those in Task 1.

### 2.2.2.2 Design:

The same design was used for both Experiment 1 and 2 and as such shall only be covered once. A fixed interval 2 alternate forced choice paradigm was used to test people with ASDs' ability to use biological information given a strong, coherent and unmasked signal. Two distinct actions, a knock and a lift, were blended together in a systematic fashion in increments of 10% giving 11 stimuli levels. A method-of-constant-stimuli design was used for Task 2, with 20 repetitions of each stimulus level presented in a randomised order giving a total of 220 trials. Participants were asked to decide for each level of action blends which action it was that they saw. There was no correct answer and as such, the dependent variable was simply the proportion of responses categorised as a "knock".

### 2.2.2.3 Stimuli:

*Experiment 1* Stimuli were presented on a CRT monitor with a resolution of 1280 x 1024 pixels, on a display size subtending 22.62° of visual arc by 17.06° at a distance of 1m from the participant, with a refresh rate of 60 Hz.

The stimuli were generated using knocking and lifting actions from the motion capture library of Ma, Patterson and Pollick, (2006). Exemplars of each action were chosen in such a way as to match the durations of the actions without affecting their natural timing.

To do this, the velocity of the wrist joint for each movement was calculated, then the movements with the minimum, median and maximum speed for each action were chosen. Exemplars from the median speed knock and maximum speed lift (durations 2.15, 3.0 seconds respectively) were found to be the closest matches. To create blends of these two actions the algorithm of Kovar and Gleicher (2003) was used, which allowed the two actions to be blended together whilst maintaining the basic biomechanical properties of the human actor, ensuring that the resulting point-light displays still looked human in origin. After conversion from 3DS coordinate data to Biovision Hierarchical Data (BVH) format, the blending algorithm generated new BVH files that weighted the contribution of each of the original actions to the new blended actions in steps of 0, 10, 20, 30, 40, 50, 60, 70, 80, 90 and 100%. For instance, when the knocking component was 100% the lifting component would be 0%, but when the knocking component was only 30% the lifting component would be 70%. These new actions were then converted into point-light files containing 3D coordinates for the 15 points used, and lines between the major joints were added in such a way as to follow the skeleton of the body. These point-light files were then converted into Audio Video Interleave (AVI) format movies of dimensions 256 x 256 pixels, with the figure subtending a visual angle of approximately 3.44° x 1.26°.

*Experiment 2:* The stimuli were identical to those in Experiment 1 except that the lines forming the skeleton on the walker were removed in order to make the stimuli more compatible with the stimuli in Tasks 1 and 3.

#### 2.2.2.4 Procedure:

The procedure was identical for both Experiment 1 and 2 and as such shall only be covered once. Participants were seated with a viewing distance of approximately 1m. They were informed that they would see a display depicting an action that was either a knock or a lift, and that their task would simply be to record whether the action they saw was a knock or a lift using the predefined keys on the keyboard. The experiment took 25 minutes and once the experiment was over participants were fully debriefed and any difficulties or comments they had with the task were noted.

## 2.2.3 Task Three – Categorisation of Affect

### 2.2.3.1 Participants

Not all of the participants used in Task 1 and 2 were used in either of the experiments in Task 3. As such, the details will be given here in full.

*Experiment 1:* The participants in the ASD group consisted of 5 adult males aged between 18 and 25 who had been diagnosed independently as having autism or aspergers syndrome, and who had also met the required criteria to be classed as having ASD on the SRS (Constantino et. al., 2003) and the AQ (Baron-Cohen, et. al., 2001). In addition, 2 of the 5 participants had undergone clinical assessment specifically for the purpose of the study and had been confirmed using the ADI (Le Couteur, et. al., 1989; Lord et. al. 1994) as having an ASD. All participants can be considered to be of average or above average intelligence, as four of the five have attended university and the fifth attended a school for the academically gifted. Control participants consisted of 5 age matched males between the ages of 18 and 25 and all were university students.

*Experiment 2:* The participants in the ASD group consisted of 10 adult males aged between 18 and 36 who had been diagnosed independently as having autism or aspergers syndrome. Furthermore, 3 of the 12 participants had undergone further clinical assessment in the form of the ADI-R (LeCouteur, et. al. 1989; Lord, et. al., 1994) and the SRS (Constantino, et. al., 2003) and had met diagnostic criterion on these measures as well. The remaining 7 of the 10 were unable to have such diagnoses due to a lack of available developmental history. Each participant also underwent the Weschler Abbreviated Scale of Intelligence (WASI) test as an estimate of verbal, performance and full-scale IQ. This is an abbreviated version of the Wechsler Adult Scale of Intelligence (WAIS) and the scores correlate well between the two.

Table 2.2: Means and SDs of the age in years and IQ for each group

| | AGE | | IQ | |
|---|---|---|---|---|
| **Group** | **MEAN** | **STDEV** | **MEAN** | **STDEV** |
| ASD | 27.70 | 6.09 | 126.10 | 5.43 |
| Control | 27.40 | 6.11 | 125.50 | 8.78 |

Control participants were 10 age- and IQ-matched adults who were confirmed as not being on the Autism Spectrum as determined by assessment using the AQ. Paired samples t-tests confirmed that there were no significant difference between the two groups for either age ($t(9) = 0.474$, $p = .647$) or IQ ($t(9) = 0.502$, $p = .627$).

#### 2.2.3.2 Design:

The design for Experiment 1 & 2 was identical and as such shall only be covered once. In this task we used a four-alternative forced-choice paradigm, in which participants were asked to determine whether a point-light display of a throwing action was representative of either an angry, happy, neutral or sad affect. Participants saw 2 examples of each of the four emotions from 29 actors in a fully randomised order, giving a total number of trials of 232, which is 58 per affect.

#### 2.2.3.3 Stimuli:

*Experiment 1:* The stimuli were generated from throwing actions taken from the motion-capture library of Ma, Patterson and Pollick (2006). The library contains point-light displays of 29 actors performing throwing motions in 4 affect states. In order to induce the correct affect in each actor they first read a paragraph designed to induce affect (angry, happy, neutral, and sad). Once induced to feel the emotion, the actor threw a ball into a bucket on the floor 5 times in 2 separate recording sessions. As such, there were ten possible displays for each of the four emotions for each of the 29 actors giving a possible 1160 point-light displays to be sampled. Participants saw two randomly selected examples of each emotion for every actor, giving a total number of displays seen by the participant of 232.

The point light stimuli consisted of a display of just the thrower's arm: the shoulder, elbow, wrist and two points for the ends of the hands, seen from the side view to maximise the information available to the participants. Each point-light actor was scaled to the same height and the displays subtended an approximate maximum viewing angle of 5.71° x 4.48°, though the horizontal extent of each action varied by emotion. In order to reduce the possibility of using a simple velocity heuristic to judge the affect of the thrower, the mean wrist velocity of all the throws was normalised to the median velocity of the wrist of every throwing action.

*Experiment 2:* The point light stimuli consisted of the full 15 points of the actor, seen from the side view to maximise the information available to the participants. All displays were size normalized and, depending on the emotion and individual thrower, subtended an approximate maximum of 4.6° x 1.71°. As the full height of the thrower was included in the stimuli, the ratio of horizontal viewing angle to vertical viewing angle was considerably smaller in these stimuli than in those used in Experiment 1. In order to preserve all the natural signals that may be salient to a viewer, there were no manipulations of the velocities in the stimuli for experiment 2.

#### 2.2.3.4 Procedure:

The procedure for Experiment 1 & 2 was identical and as such shall only be covered once. Participants were seated with a viewing distance of approximately 1m from the screen and informed that they were going to see point light displays of people throwing. They were told that it was their task to determine which of the four emotions the throwing movement was being performed under, angry, happy, neutral or sad. A set of practice trials preceded the testing phase to ensure that participants were comfortable with the task. There were a total of 232 trials, which took approximately 40 minutes to complete. Once the experiment was finished participants were fully debriefed and any questions or comments they had regarding the experiment were noted and answered where possible.

## 2.3 Results

### 2.3.1 Task 1: Detection of Coherent Biological motion in Noise

The aim of Task 1 was to determine whether people with ASDs were able to detect a coherent point-light walker in a noise mask of biological motion. To do this we tested two groups, one with ASD and a control group, at 8 levels of signal points (points that were part of a coherent walker) and used a QUEST algorithm to determine the noise tolerance thresholds at each level of signal points. In Experiment 1 there were only 5 participants in each group and there had been no specific age or IQ matching whereas in Experiment 2 the age and IQ of the control group was pair-wise matched to that of the group with ASDs.

#### 2.3.1.1 Task 1 Experiment 1

Figure 2.2 shows the resulting noise-tolerance thresholds from the QUEST algorithm for each level of signal points in Log space, which are the units used by the algorithm. As can be seen from Figure 2.2, at the lowest level of signal points neither group were able to tolerate any noise while still being able to correctly detect the point light walker. For the control group the number of noise points needed to mask the walker increased as the number of signal points increased, indicating lower motion coherence thresholds. In the ASD group however, it is apparent that for low numbers of signal points, between 3 and 7, they were completely unable to detect the walker in the presence of any noise. Between 7 and 15 signal points they were able to distinguish the walker from noise, but at a far lower number of noise points than the controls, indicating much higher biological motion coherence thresholds.

These results suggest that the participants with ASDs were unable to detect biological motion when the signal strength (number of signal points) was low, but that once the signal was strong enough they were able to detect the signal, but only with low levels of noise.

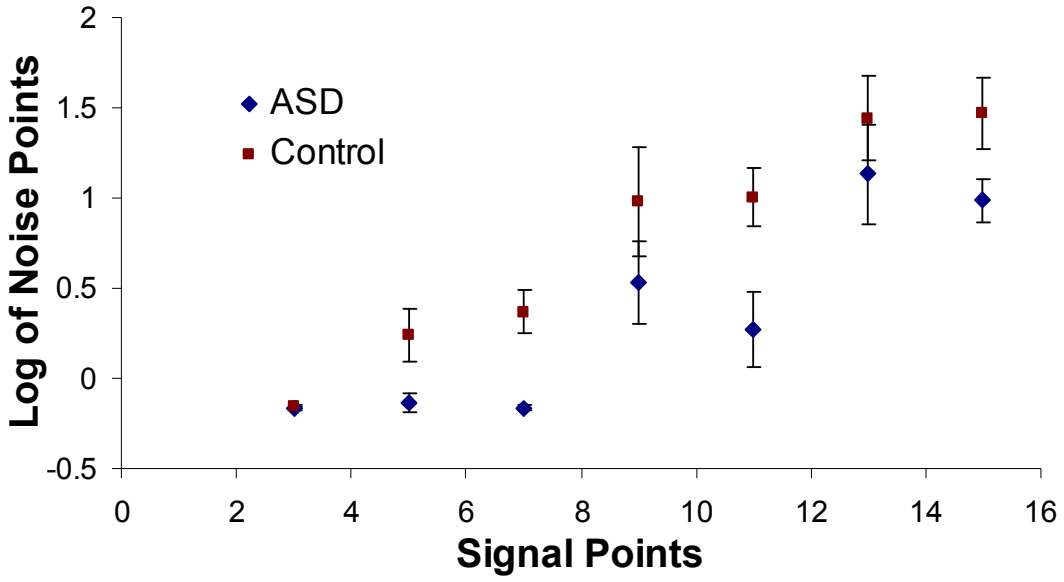


Figure 2.2 Mean 75% correct thresholds and standard errors derived from QUEST procedure for each level of signal points in pilot experiment. Thresholds are reported in the units used by the QUEST algorithm(Log)

The normality of the distributions of thresholds for each stimulus level in each group was tested using a Kolmogorov-Smirnoff test and all met normality assumptions. Furthermore,

Mauchleys test of Sphericity confirmed that there was equality of variance amongst all groups of means. As all the assumptions were met a two-factor mixed design ANOVA was used to determine whether the groups were significantly different across the stimulus levels. This revealed that there was a significant main effect of group $F(1,8) = 9.435$, $p = 0.015$, a significant main effect of Signal Strength $F(6,48) = 25.967$, $p < 0.001$, but no significant interaction $F(6,48) = 1.139$, $p = 0.355$. A summary of the post-hoc tests can be found in Table 2.3, and show that there was a significant increase in noise tolerance thresholds as the signal strengths increased.

Table 2.3: Differences between means ($\mu_1$–$\mu_{2)}$ and p-values from post-hoc tests

| | 5 | 7 | 9 | 11 | 13 | 15 | |
|---|---|---|---|---|---|---|---|
| 3 | -0.217<br>0.396 | -0.273<br>0.056 | -0.926<br>0.027 | -0.757<br>0.009 | -1.1469<br>0.001 | -1.422<br>0.001 | $\mu_1$–$\mu_2$<br>P |
| 5 | | -0.055<br>1.000 | -0.705<br>0.225 | -0.540<br>0.221 | -1.251<br>0.001 | -1.205<br>0.001 | $\mu_1$–$\mu_2$<br>P |
| 7 | | | -0.653<br>0.244 | -0.484<br>0.068 | -1.196<br>0.010 | -1.150<br>0.001 | $\mu_1$–$\mu_2$<br>P |
| 9 | | | | 0.169<br>1.000 | -0.543<br>0.309 | -0.497<br>0.604 | $\mu_1$–$\mu_2$<br>P |
| 11 | | | | | -0.712<br>0.190 | -0.665<br>0.016 | $\mu_1$–$\mu_2$<br>P |
| 13 | | | | | | -0.460<br>1.000 | $\mu_1$–$\mu_2$<br>P |

#### 2.3.1.2 Task 1 Experiment 2

Figure 2.3 shows the mean of the 75% correct threshold as measure in Log units. As can be seen there is little difference in performance between the groups, though the noise-tolerance thresholds of the ASD group are consistently below that of the control group. Given that the SEs are of the Log values, these are substantial, indicating a large amount of variance within both groups. This is further illustrated in Figure 2.4

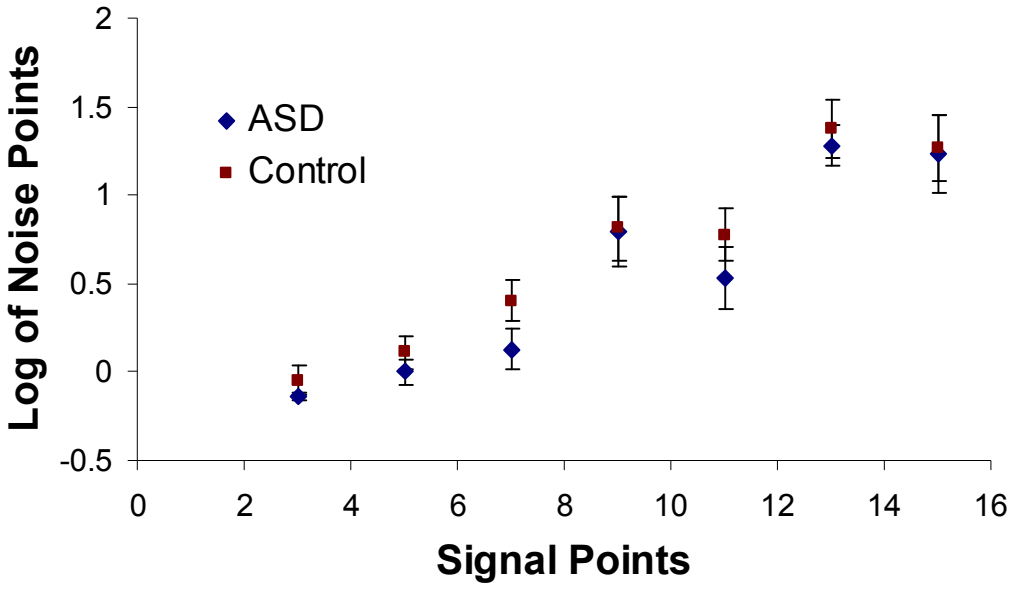


Figure 2.3 Mean 75% correct thresholds and standard errors derived from QUEST procedure for each level of signal points. Thresholds are reported in the units used by the QUEST algorithm(Log).

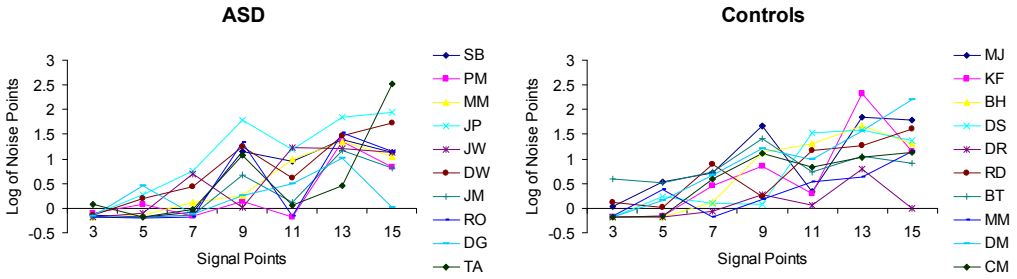


Figure 2.4: Individual 75% correct thresholds for both groups. Thresholds are reported in the units used by the QUEST algorithm(Log)

Box plots were generated in order to check if there were any outliers that were skewing the distributions. Outliers were categorised as being more than 1.5 the inter-quartile-range (IQR) above the 75$^{th}$ percentile or below the 25$^{th}$ percentile. Several outliers were found as can be seen in Figure 2.5.

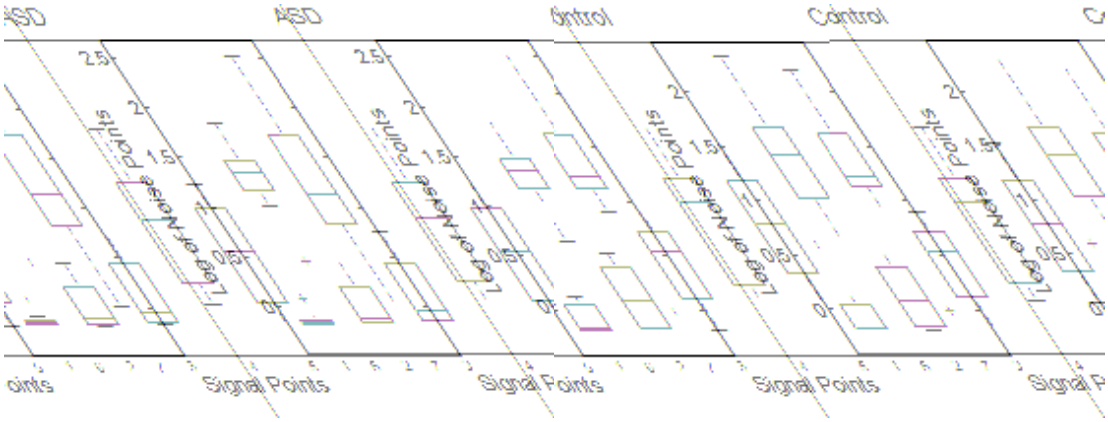

Figure 2.5: Box plots of group thresholds in Log units. Red lines indicate the median value, blue boxes represent the 25% and 75% inter-quartile range. Dotted lines represent the maximum and minimum scores still within the 1.5 x inter-quartile range. Red crosses represent outlying values.

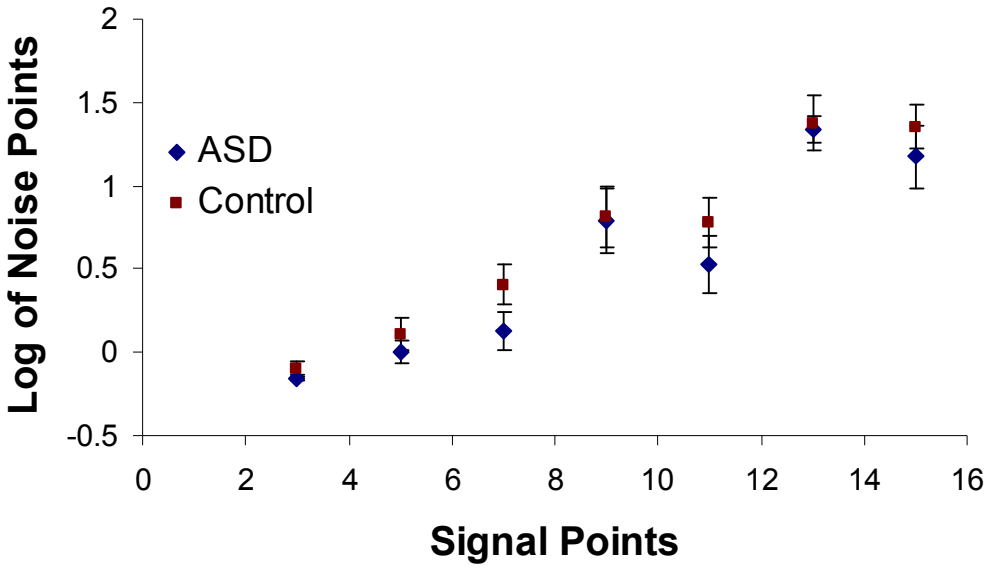


Figure 2.6 Mean 75% correct thresholds and standard errors derived from QUEST procedure for each level of signal points after removal of outliers. Thresholds are reported in the units used by the QUEST algorithm(Log).

These means were replaced with the value of the next nearest score. Re-categorization of the outliers led to some increases in the differences between the groups, as can be seen in Figure 2.6. However, a mixed ANOVA revealed that these were non-significant $f(1,18) = 1.506$, $p = 0.236$. There was a significant main effect of Signal Strength $f(6,108) = 47.174$, $p < 0.001$ and no significant interaction effect $f(6,108) = 0.381$, $p = 0.381$, indicating that the main effect of Signal Strength did not differ between the two groups. Table 2.4 shows the summary of the post hoc tests that showed that noise tolerance thresholds were higher in the conditions with larger numbers of Signal Points.

Table 2.4: Differences between means ($\mu_1 - \mu_2$) and p-values from post-hoc tests

| | 5 | 7 | 9 | 11 | 13 | 15 | |
|---|---|---|---|---|---|---|---|
| 3 | -0.181<br>0.067 | -0.389<br>0.001 | -0.927<br>0.001 | -0.778<br>0.001 | -1.1481<br>0.001 | -1.388<br>0.001 | $\mu_1-\mu_2$<br>P |
| 5 | | -0.208<br>0.687 | -0.747<br>0.001 | -0.597<br>0.003 | -1.300<br>0.001 | -1.207<br>0.001 | $\mu_1-\mu_2$<br>P |
| 7 | | | -0.538<br>0.010 | -0.389<br>0.041 | -1.092<br>0.001 | -0.999<br>0.001 | $\mu_1-\mu_2$<br>P |
| 9 | | | | 0.150<br>1.000 | -0.554<br>0.006 | -0.461<br>0.031 | $\mu_1-\mu_2$<br>P |
| 11 | | | | | -0.703<br>0.001 | -0.661<br>0.009 | $\mu_1-\mu_2$<br>P |
| 13 | | | | | | -0.093<br>1.000 | $\mu_1-\mu_2$<br>P |

#### 2.3.1.3 Summary of Results

In Experiment 1 we found that the ASD group were unable to perform the task with any noise points at low numbers of signal points, whilst the control group were able to perform the task with relatively few signal points. In addition, it appeared that the ASD group, although able to perform the task at higher signal strengths, had lower noise tolerance thresholds than did the control group. Overall, this suggested that the ASD group were impaired at detecting a coherent biological motion signal in a mask of randomly distributed biological motion. However, once these two groups were matched for age and IQ, despite a trend suggesting that the ASD group were slightly poorer than the controls, there were no significant differences between the two groups.

### 2.3.2 Task 2: Discrimination of Knocking and Lifting motion from Action Blends

In Task 2 participants were asked to report whether the action shown to them was a knock or a lift from a set of action blends containing different amounts of each of the two actions. This was to determine whether the ASD group were as sensitive to subtle kinematic differences in the stimuli as were the control group. In Experiment 1 there were only 5 male participants in each group and there was no explicit matching criterion. Furthermore, the stimuli in Experiment 1 had lines that joined the joints on the point-light actors, forming a skeleton that enhanced the human form. In Experiment 2 there were 10 participants in the ASD group and 10 age and IQ matched controls. The stimuli in Experiment 2 did not contain the form-enhancing skeleton of the stimuli in Experiment 1.

#### 2.3.2.1 Task 2 Experiment 1

In Task 2 we set out to determine whether the two groups were able to process low-level subtle kinematic details from noise-free biological motion signals using stimuli that had been blended together from knocking and lifting actions. As can be seen from Figure 2.7, although the performance of both groups on this task is similar, the slope of the curve of the ASD group is much shallower than the control group. To determine whether there were significant differences between the two groups, each individual's data were fitted with a cumulative guassian using the psychophysics toolbox in Matlab (Brainard, 1997;

Pelli, 1997). From this we derived the 50% knock thresholds, otherwise known as the Point of Subjective Equality (PSE), the 75% knock thresholds and slope of the curve. Given the small sample sizes, we then compared these for each group using a Mann Whitney-U test. This showed that although the slopes looked dissimilar, there were no significant differences between the two groups on any of the three measures (PSE $U = 12$, $n_1 = n_2 = 5$ $p = 0.917$; 75% knock threshold $U = 11$, $n_1 = n_2 = 5$, p = 0.0754; Slope $U = 10$, $n_1 = n_2 = 5$, $p = 0.602$).

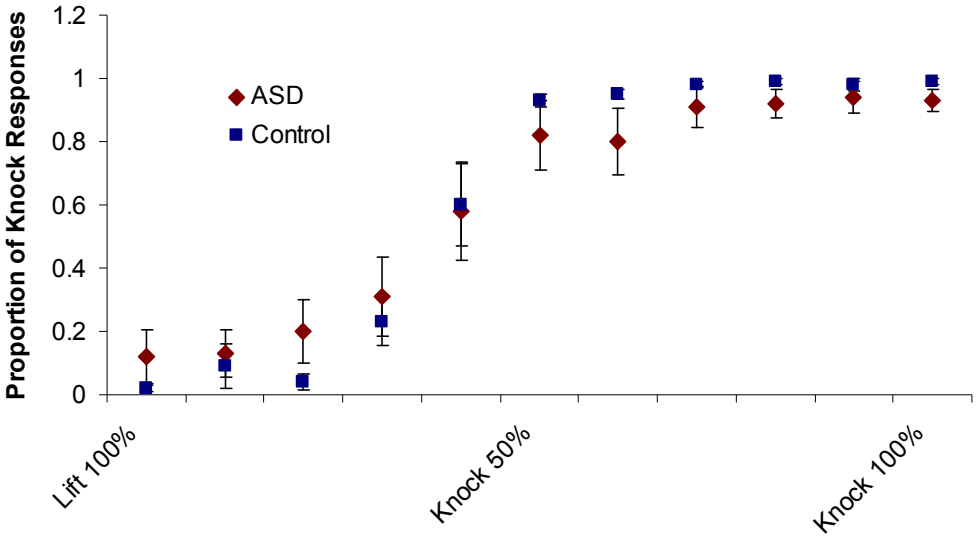


Figure 2.7: Mean proportion correct and SEs for each level of stimuli for pilot experiment. ASD group means are represented by red diamonds and control groups means are represented by blue squares.

Of interest however, is the much higher variance observed in the ASD group compared to the control group. Figure 2.8 shows the covariance matrices for the ASD group and the Control group. In both groups, the variance is maximal around the 40% knock stimuli, but is distributed much more widely in the ASD group than in the Control group. Furthermore, as can be seen from Figure 2.8, the variance across conditions is larger in the ASD group than in the Control group.

In order to test whether the variances were significantly different, an independent t-test was used, treating the variance at each stimulus level as an observation for each of the groups. A 1-sample Kolmogorov-Smirnoff test was used to ensure that the distribution of variances across the conditions did not differ significantly from the normal distribution (ASD, Z = 0.472, P = 0.979; CON, Z = 1.277, p = 0.077). Furthermore, a Levene's test for Homogeneity of Variance confirmed that the variance was equal in each group

(F(1,20) = 1.296, p = 0.268, and as such the basic assumptions required for a t-test were met. The results confirmed that the mean variance in the ASD group was significantly higher than in the Control group t(20) = 2.329, p = 0.030.

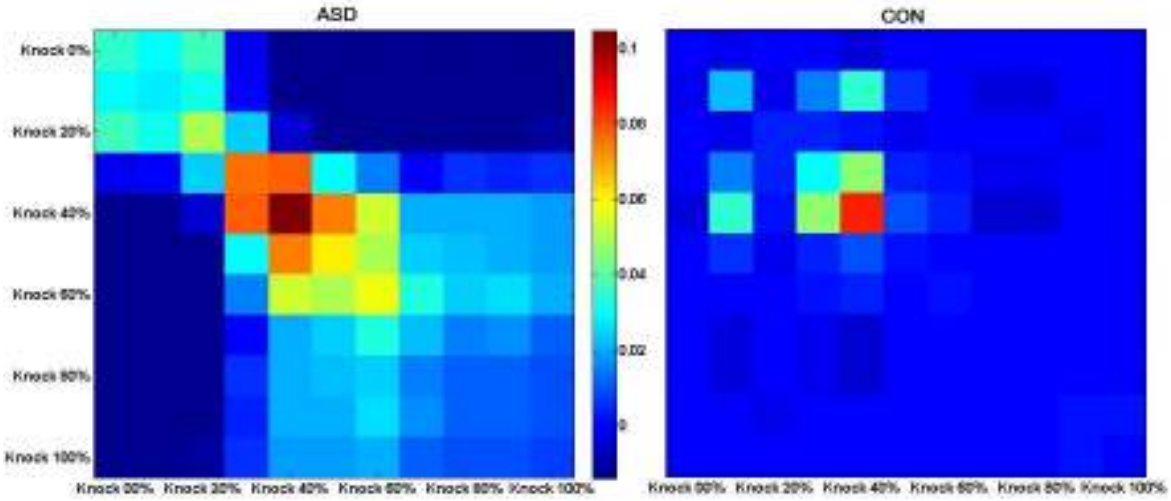


Figure 2.8: Colour chart visualisation of covariance matrices for each group by condition in the pilot experiment. Bluer colours represent low covariance between conditions whilst redder colours represent high covariance between conditions.

### 2.3.2.2 Task 2 Experiment 2

Figure 2.9 shows the mean proportion of knock categorisations at each stimulus level. As before, the performance of both groups is very similar, but the ASD group seem to show a shallower slope than the Control group. As in experiment 1, each participant's data were fitted with a cumulative Gaussian and the PSE, 75% correct threshold and the Slope were derived from these fits. Independent t-tests were used to test for significant differences between the groups for each of the three measures. A Levene's test for equality of variance found that the variance was not equal between the two groups on the measures of PSE and 75% knock thresholds. Correcting for this by adjusting the degrees of freedom for these measures, the t-tests revealed that despite the trend seen in the group data, there were no significant differences between the PSE, the 75% knock correct threshold or the slopes of the two groups (PSE, t(12.87) = 0.51, p = 0.620; 75% knock threshold, t(11.12) = 1.18, p = 0.262 ; Slope, t(18) = -0.268, p = 0.791.

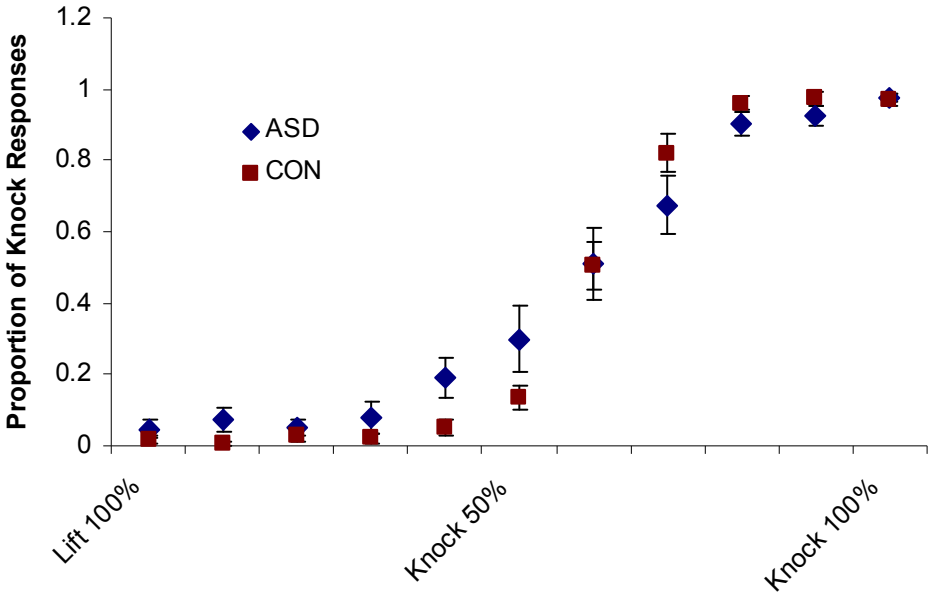


Figure 2.9: Mean proportion correct and SEs for each level of stimuli. ASD group means are represented by red diamonds and control groups means are represented by blue squares

In order to examine the variance in the raw data, as opposed to that obtained after psychometric curve fitting, we again looked at the variance across stimulus levels. Figure 2.10 shows the covariance matrices for each group and reveals that not only are the variances across the conditions larger for the ASD group than the Control group, but that these larger variances are distributed more widely in the ASD group than the Control group.

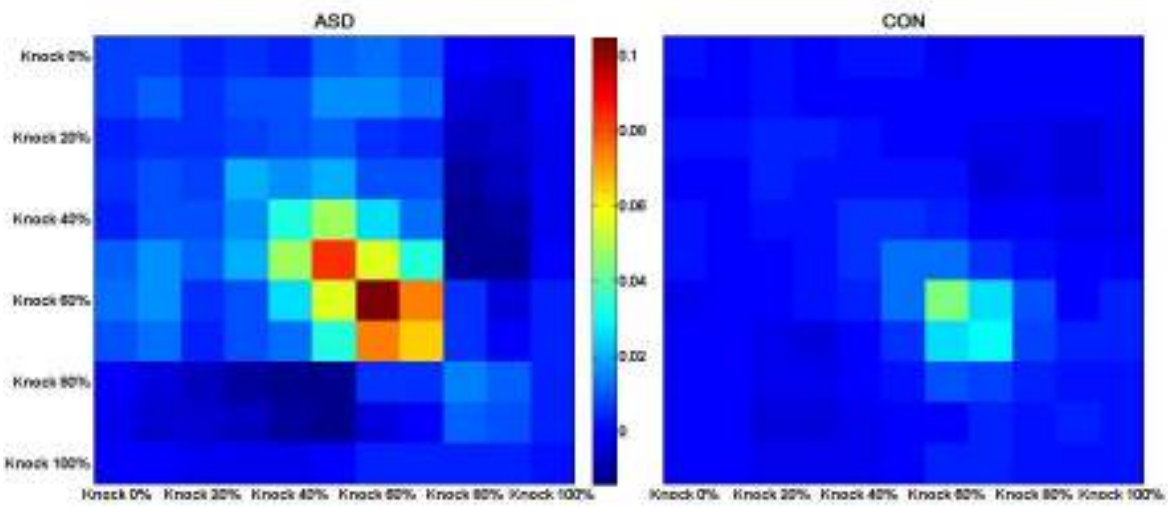


Figure 2.10 Colour chart visualisation of covariance matrices for each group by condition. Bluer colours represent low covariance between conditions whilst redder colours represent hi covariance between conditions.

To test this, an independent t-test was used using each groups variance for each condition as an observation. A Kolmogorov-Smirnov one-sample test was run, to ensure that the

variances did not violate the normality assumption of parametric tests, and confirmed that the variances across the conditions did not significantly differ from a normal distribution (ASD, $Z = 0.659$, $p = 0.778$; CON, $Z = 0.933$, $p = 0.349$). Furthermore, a Levene's test for Homogeneity of Variance was not significant $F(1,20) = 3.333$, $p = 0.083$, and as such, the basic assumptions for a t-test were met. The results showed that the variance within the ASD group was indeed significantly larger than the variance within the Control group $t(20) = 2.123$, $p = 0.046$.

#### 2.3.2.3 Summary of Results

In both Experiment 1 and Experiment 2 we found no significant differences in either the slope of the curves, the PSE or the 75% knock thresholds. However, it was found that the variance in the ASD group was significantly larger than in the control group, indicating more variability in the scores. Furthermore, in the control group the variance was predominantly found at stimulus levels close to the PSE, whereas in the ASD group it was dispersed across a wider range of stimulus levels.

Between Experiment 1 and Experiment 2, there was a shift in the PSE for both groups from around the point where knocking action contributed 40% to the action in Experiment 1, to Experiment 2 where the PSE was around the point where the knocking action contributed 60% to the motion. As the only difference in the two conditions was the removal of the skeleton, it is likely that the lines in the Experiment 2 enhance the salience of the knocking component of the actions. It is also important to note that for both groups in both Experiments, the majority of the variance, and hence differences in responses between participants, was focal around the PSE, and that at either end of the scale there was much more agreement. This was especially so in the Control group, but in the ASD group variances were both larger, as mentioned, and also more widely distributed across the conditions, indicating less agreement between participants on a broader range of stimulus levels.

### 2.3.3 Task 3: Categorisation of Affect

In Task 3 participants were asked to say whether the actions they were viewing were from one of four affect categories: angry, happy, neutral or sad. In Experiment 1 there were

only five male participants in each group and there was no explicit matching criterion and the stimuli consisted of only the arm of a point-light actor. In Experiment 2 there were 10 participants with ASD and 10 pair-wise age- and IQ-matched controls. The stimuli in Experiment 2 were point-light displays of the full body of the actor performing the throwing action.

#### 2.3.3.1 Task 3 Experiment 1

The means and standard deviations are summarised by affect and group in Table 2.5. As can be seen from figure 2.11, overall the proportion correct was low for both groups, but more so for the ASD group than for the control group.

Table 2.5: Means and SDs in for each stimulus type separated by group.

| | | Angry | Happy | Neutral | Sad | |
|---|---|---|---|---|---|---|
| Controls | μ | 0.775862 | 0.4 | 0.393103 | 0.324138 | 0.350125 |
| | σ | 0.105581 | 0.076525 | 0.158677 | 0.117823 | 0.188937 |
| ASD | μ | 0.557471 | 0.252874 | 0.448276 | 0.241379 | 0.371347 |
| | σ | 0.163869 | 0.196077 | 0.27097 | 0.215345 | 0.175009 |
| | μ | 0.693966 | 0.344828 | 0.413793 | 0.293103 | |
| | σ | 0.163761 | 0.141877 | 0.190214 | 0.151713 | |

An ANOVA revealed that there was a significant effect of group, $F(1,6) = 7.64$, $p = 0.033$, a significant effect of Affect ($F(1,6) = 29.581$, $p = 0.002$) but no significant interaction $F(3,18) = 0.85$, $p = 0.485$. This suggests that participants in the ASD group were less accurate than the control group in categorising the affect. Figure 2.11 shows the overall accuracy of the two groups and Figure 2.12 shows the proportions correct by emotion.

Given the small sample sizes, non parametric tests were used to confirm the results of the ANOVA. A Mann-Whitney-U test confirmed that the control groups were significantly better at judging the affect from the displays ($U = 0.5$, $n_1 = 5$ $n_2 = 3$, $P = 0.0036$) and a Friedman test revealed that there was a significant main effect of affect on overall proportion correct across affects ($\chi = 10.038$, $n_1 = 5$ $n_2 = 3$, $P = 0.018$.

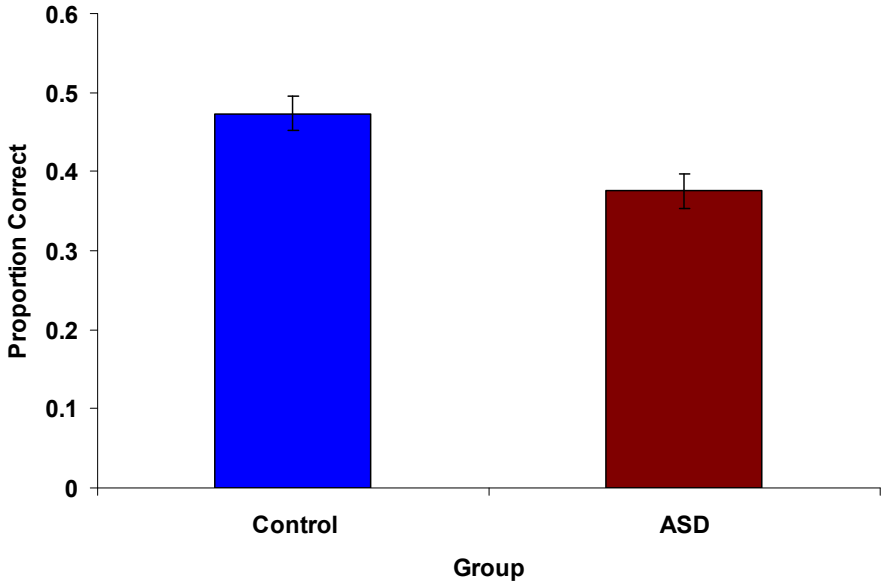


Figure 2.11: Overall proportions correct in identifying emotion for each group in the pilot experiment. Error bars represent the standard error.

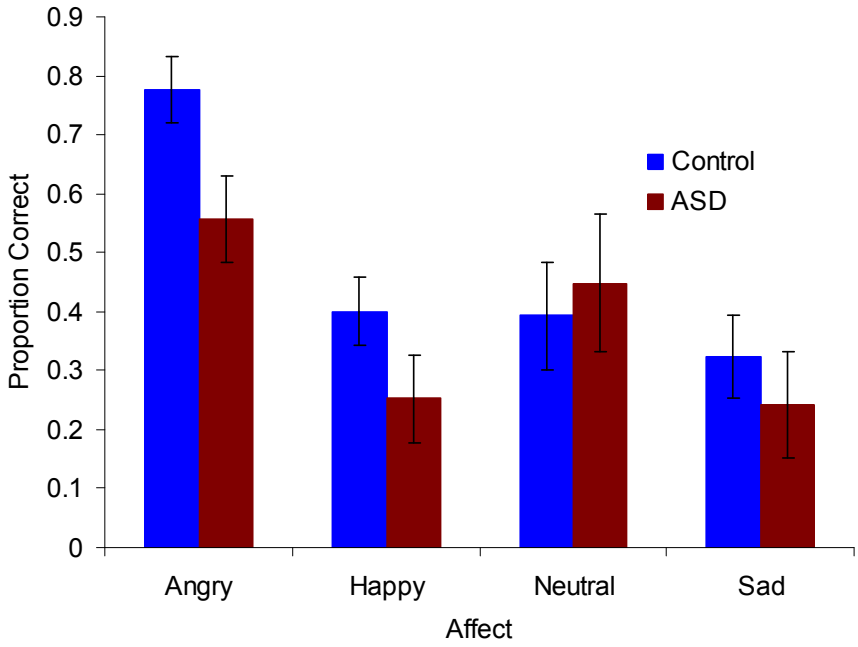


Figure 2.12: Proportions correct for affect judgements separated by stimulus type and group in the pilot experiment. Error bars represent Standard Errors.

To examine the main effect of affect we again used both parametric and non-parametric tests to ensure that no small effect was missed. Bonferroni t-tests found that the mean proportions correct in the Angry condition were significantly larger than in the Happy and Sad conditions, but was not significantly different from the mean proportion correct in the Neutral conditions, and that the mean proportion corrects in Happy, Neutral and Sad conditions did not significantly differ from each other. A Wilcoxon Signed Ranks Test,

showed that the proportion correct in the angry condition were significantly higher than the other 3 conditions, and that there were no significant differences between the proportions correct in the Happy, Neutral and Sad conditions. The results of the Bonferroni t-test and the Wilcoxon signed Ranks test are summarised in Table 2.6.

Table 2.6: Upper half of table are the results of the Bonferroni t-test, lower half are the results of the Wilcoxon signed ranks test.

| | | Angry | Happy | Neutral | Sad | |
|---|---|---|---|---|---|---|
| Angry | | | 0.340 | 0.246 | 0.384 | $\mu_1-\mu_2$ |
| | | | 0.032 | 0.114 | 0.023 | P |
| Happy | Z | -2.380 | | -0.094 | 0.044 | $\mu_1-\mu_2$ |
| | p | 0.017 | | 1.000 | 1.000 | P |
| Neutral | Z | -2.197 | -0.071 | | 0.138 | $\mu_1-\mu_2$ |
| | p | 0.028 | 0.943 | | 1.000 | P |
| Sad | Z | -2.527 | -1.192 | -0.762 | | |
| | p | 0.012 | 0.233 | 0.446 | | |

### 2.3.3.2 Task 3 Experiment 2

The means and standard deviations are summarised by affect and group in Table 2.7. As can be seen from Table 2.7 and Figure 2.12, there appear to be little differences between the 2 groups on any measure.

A mixed design ANOVA revealed that there was no significant main effect of group ($F(1,18) = 0.002$, $p = 0.968$) but that there was a main effect of Affect($F(3,54) = 22.033$, $p < 0.001$). Furthermore, there was no significant interaction between Group and Affect ($F(3,54) = 0.434$, $p = 0.729$).

Table 2.7: Means and SDs in for each stimulus type separated by group.

| | | Angry | Happy | Neutral | Sad | |
|---|---|---|---|---|---|---|
| Controls | μ | 0.656897 | 0.337341 | 0.527586 | 0.372414 | 0.473559 |
| | σ | 0.181822 | 0.091759 | 0.175113 | 0.112385 | 0.190555 |
| ASD | μ | 0.725862 | 0.313793 | 0.494828 | 0.363793 | 0.474569 |
| | σ | 0.158114 | 0.168696 | 0.151194 | 0.11187 | 0.215804 |
| | μ | 0.691379 | 0.325567 | 0.511207 | 0.368103 | |
| | σ | 0.169568 | 0.132719 | 0.160112 | 0.109227 | |

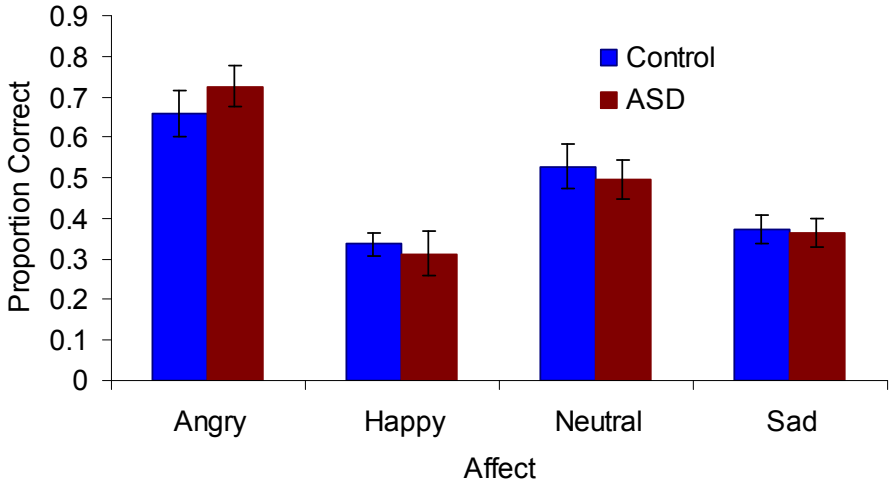


Figure 2.13: Proportions correct for affect judgements separated by stimulus type and group. Error bars represent Standard Errors.

Bonferroni t-tests were used to test the main effect of Affect and found that the mean proportion correct in the Angry condition was significantly higher than in the Happy and Neutral conditions, and that the mean proportion correct in the Happy condition was significantly lower than in the Neutral condition. The results are summarised in Table 2.8.

Table 2.8: Results from Bonferroni t-tests

| | | Angry | Happy | Neutral | Sad |
|---|---|---|---|---|---|
| Angry | $\mu_1-\mu_2$ | | 0.366 | 0.180 | 0.323 |
| | p | | 0.001 | 0.056 | 0.001 |
| Happy | $\mu_1-\mu_2$ | | | -0.186 | -0.043 |
| | p | | | 0.016 | 1.000 |
| Neutral | $\mu_1-\mu_2$ | | | | 0.143 |
| | p | | | | 0.086 |

## Analysis of False Positives

### Analysis of Overall Number of False Positives

In order to more fully understand the data, it was necessary to look at the errors made by each group and to determine if there were any systematic differences between them. Figure 2.13 shows the mean number of false positives, or incorrect responses, made by each group for each affect. For example, the number of times that happy, neutral and sad affects are mistakenly attributed to the angry condition. As can be seen, for angry, neutral and sad affects, there was little difference between the groups. However, for happy affect

throws it seems that there were a larger number of false positives, which would be expected from the poor overall performance in correctly identifying actions from this affect. Furthermore, it seems that this was primarily driven by the participants in the control group. A mixed ANOVA confirmed that there were main effects for Affect ($f(3,54) = 36.194$, $p < 0.001$) and group ($f(1,18) = 9.282$, $p = 0.007$), but these were confounded by an interaction effect ($f(3,54) = 6.559$, $p = 0.001$).

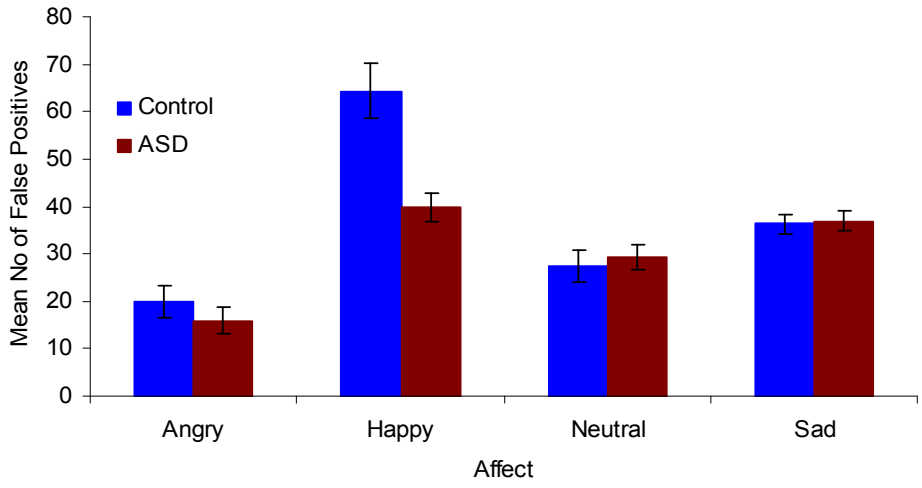


Figure 2.14 Mean number of false positives made by each group for each affect. Error bars represent Standard Errors

From Figure 2.13 it appears that the interaction effect is driven by the difference between the two groups in the Happy condition, and a lack of differences in the remaining conditions. As such, 4 pairwise t-tests were run comparing the two groups in each affect separately and corrected for multiple comparisons using a Bonferroni correction. These revealed that the mean number of false positives was indeed higher in the control group than in the ASD group $t(18) = -3.813$, $p = 0.004$, and that there were no significant differences between the two groups for the remaining affects (Angry $t(18) = -0.905$, $p = 1$; Neutral $t(18) = 0.448$, $p = 1$; Sad $t(18) = 0.172$, $p = 1$).

**Analysis of Distribution of False Positives**

Figure 2.14 presents the break down of each total from Figure 2.13 into which incorrect affect the error is attributed to. The distribution of false positives will be dealt with separately for each of the affects in the following sections.

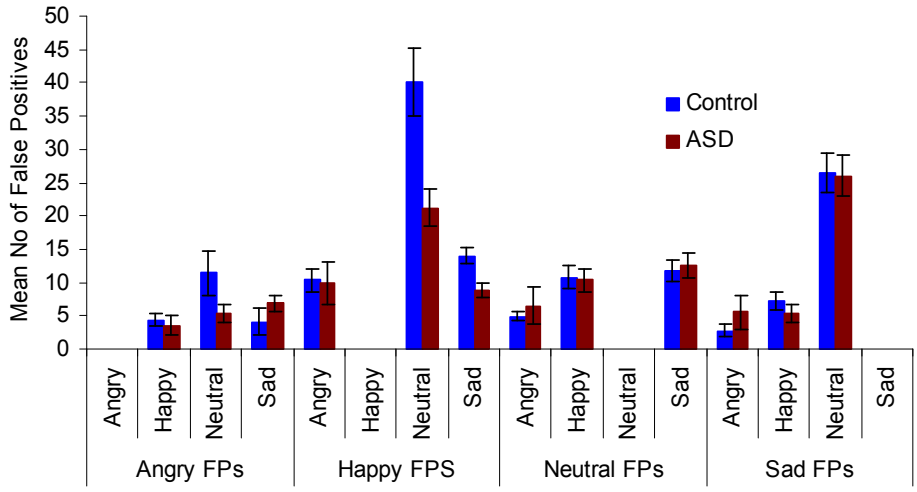


Figure 2.15: Distribution of responses amongst the incorrect affects for each stimulus type; angry, happy neutral and sad. Error bars represent the Standard Error.

Angry False Positives: In order to examine how each group differed in terms of where they made the most number of errors, the number of False Positives incorrectly attributed to each of the incorrect affects was calculated for each condition. Although there seems to be small differences between the groups in the distribution of false positives across the incorrect affects, a 3 x 2 mixed ANOVA revealed no significant main effect of Group $f(1,18) = 0.819$, $p = 0.377$, Affect $f(2,36) = 2.662$, $p = 0.084$ and no significant interaction between the two factors $f(2,36) = 2.603$, $p = 0.088$).

Happy False Positives: Incorrect responses to happy stimuli seemed to differ more in terms of mean number of false positives across the 3 incorrect affects, Angry, Neutral and Sad and between the two groups. Although the number of false positives attributed to the angry category are comparable between the two groups, it seems that the control group attributed more happy stimuli, and to a lesser extent sad stimuli, to the neutral category and that overall more happy stimuli were attributed to the neutral category than to the angry or sad categories. A 3 x 2 mixed ANOVA revealed a significant main effect of group $f(1,18) = 14.536$, $p = 0.001$ and Affect $f(1.233,22.197) = 29.323$, $p < 0.001$ (corrected for violation of the assumption of sphericity using the Greenhouse-Geisser correction). These were confounded however by a significant interaction effect between Group and Affect $f(1.233,22.197) = 5.074$, $p = 0.028$ (also corrected using Greenhouse-Geisser). From Figure 2.14 it appears that the interaction is being driven by the difference between the 2 groups in attributing stimuli in the Happy condition to the Neutral and Sad categories. To test this Mann-Whitney U tests were used, corrected for using a Bonferroni adjustment, as the data cannot be assumed to be of equal variance. These revealed that the proportion of

Happy stimuli attributed to the sad category did not significantly differ between the two groups $U = 40.5$, $n_1 = n_2 = 10$, $p = 1$. However, the number of Happy stimuli attributed to the Neutral and Sad categories was significantly higher in the control group than the ASD group, $U = 14.00$, $n_1 = n_2 = 10$, $p = 0.021$ & $U = 14.00$, $n_1 = n_2 = 10$, $p = 0.018$ respectively.

Neutral False Positives: As can be seen in Figure 2.14, it seems that neutral affect stimuli are incorrectly attributed to Happy and Sad categories slightly more than the Angry category and that the groups do not seem to differ in respect to the pattern of attribution. A 3 x 2 mixed ANOVA revealed that there was a significant effect of affect $f(2,36) = 6.332$, $p = 0.004$, but no significant main effect of group $f(1,18) = 0.200$, $p = 0.660$, and that there was no significant interaction between the two factors $f(2,36) = 0.160$, $p = 0.853$). T-tests, corrected for multiple comparisons, revealed that the mean number of false positives for Neutral throws attributed to the Sad category, was significantly higher than those attributed to the Angry category $t(19) = -3.438$, $p = 0.009$. After applying the Bonferroni correction, there were no differences in the number of Neutral stimuli attributed to the Angry category compared with the number attributed to the Happy category $t(19) = -2.413$, $p = 0.078$ and no difference between the number attributed to the Happy and Sad categories $t(19) = -0.963$, $p = 1$.

Sad False Positives: From Figure 2.14 it can be seen that, as for Neutral throws, there is little difference between the two groups for Sad throws. There does seem to be a strong tendency to attribute Sad stimuli to the neutral category. An ANOVA revealed that there was a main effect of Affect $f(1.487,26.767) = 46.579$, $p < 0.001$(corrected for violations of sphericity assumption using Greenhouse-Geisser), but no main effect of Group $f(1,18) = 0.030$, $p = 0.865$ and no significant interaction $f(1.487,26.767) = 0.418$, $p = 0.603$. As sphericity could not be assumed, Wilcoxon signed ranks tests were used to test the main effect of Affect, and were corrected for multiple comparisons using a Bonferroni adjustment. These confirmed that the number of Sad stimuli incorrectly categorised as Neutral was significantly higher than the number incorrectly categorised as Angry $Z = -3.623$, $n_1 = n_2 = 10$, $P < 0.003$ and the number incorrectly categorised as Happy $Z = -3.926$, $n_1 = n_2 = 10$, $P < 0.003$ and that there was no difference in the number incorrectly categorised as Angry and Neutral $Z = -1.724$, $n_1 = n_2 = 10$, $P < 0.225$.

**Analysis of Relative distribution of False Positives among Incorrect Affects**

Although the total number of false positives and their distribution amongst the incorrect affects provides information about the overall errors, it was necessary to examine how the relative proportions of the false positives are distributed within the groups and to look at whether this pattern differs between the groups. Figure 2.15 shows the proportion of false positives allocated to the incorrect affects.

Since for each group the proportions of false positives allocated to each incorrect Affect sum to 1, they cannot be assumed to be independent. As such, for each group and condition a non-parametric one-way Friedman ANOVA was calculated to determine if there were any significant differences in the distribution of False Positives. Post-hoc tests were repeated measure non-parametric Wilcoxon Signed Ranks tests, and all were corrected using the Bonferroni adjustment for multiple comparisons.

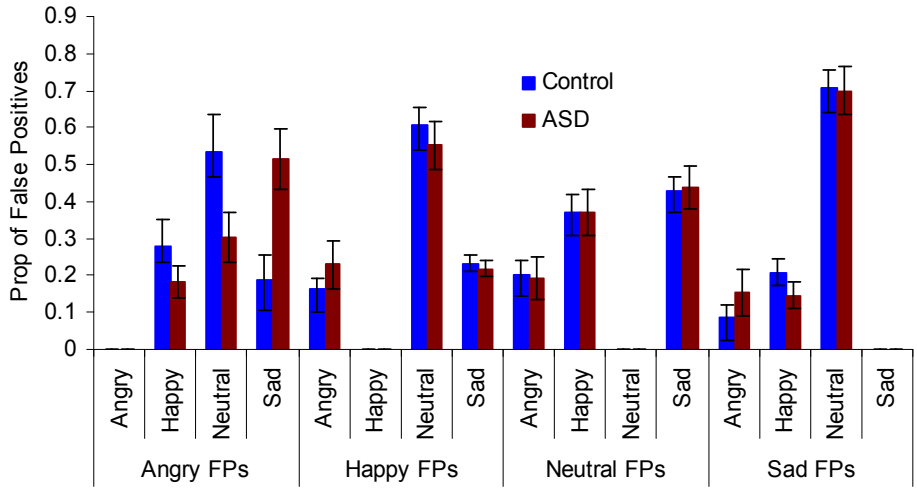


Figure 2.16: Proportion of errors broken down into the incorrect affect. For each of the stimulus affects the totals across incorrect affects within each groups sum to 1. Error bars represent the Standard Error.

Angry False Positives: As can be seen in Figure 2.15, the false positives seem to be distributed differently between the two groups, with stimuli in the Angry condition being largely attributed to the neutral category by controls, whilst the ASD group tend to attribute angry throws more to the Sad category. This effect was non significant for the Control group $\chi(2) = 2.389, P = 0.303,$ but was significant for the ASD group $\chi(2) = 7.515, P = 0.023$. Post hoc follow up tests found that the proportion of False positives categorised as Sad was significantly higher than those categorised as Happy $Z = -2.429, p = 0.045$, but

not Neutral $Z = -1.245$, $p = 0.639$, and that there was no significant difference in the proportion of false positives attributed to the neutral and happy categories $Z = -1.483$, $p = 0.414$. This suggests that the ASD group tend to misattribute Angry stimuli to the sad category more so than to the happy category, whilst the control group, although seeming to misattribute Angry stimuli to the neutral category, do not significantly differ overall in the pattern of their incorrect responses.

Happy False Positives: As can be seen from Figure 2.15, the pattern of false positives is very similar in each group, with a tendency to attribute more False Positives to the Neutral category. A non-parametric one-way ANOVA revealed that there was a significant effect in both the Control group $\chi(2) = 14.600$, $P = 0.001$ and the ASD group $\chi(2) = 9.800$, $P = 0.007$. Post hoc tests showed that these effects were driven by different patterns of incorrect responses in each of the groups. In the control group, the proportion of Happy stimuli attributed to the Neutral category was significantly larger than the number attributed to the Angry category $Z = -2.701$, $P = 0.0.021$ and the Sad category $Z = -2.805$, $p = 0.015$. Furthermore, the proportion of Happy stimuli incorrectly attributed to the Sad category was significantly higher than the proportion attributed to the Angry category $Z = -2.599$, $p = 0.027$. In the ASD group however, the proportion of Happy stimuli attributed to the Neutral category was only significantly larger than those attributed to the Sad category $Z = -2.599$, $p = 0.027$. There were no differences between the proportion of false positives assigned to the Neutral or Angry categories $Z = -1.886$, $p = 0.177$, or between the proportion assigned to the Angry and Sad categories, $Z = -0.204$, $p = 0.838$. These results suggest that both groups showed a strong tendency to misattribute Happy stimuli to the Neutral category, but that the effect is perhaps smaller in the ASD group.

Neutral False Positives: As can be seen from Figure 2.15, there seems to be a slight tendency for both groups to attribute a larger proportion of false positives to the Happy and Sad categories more so than to the Angry category. However, analysis revealed that there was only a significant effect in the Control group $\chi(2) = 7.744$, $P = 0.021$ and not in the ASD group $\chi(2) = 3.211$, $P = 0.201$. Post hoc tests revealed that this effect in the control group was driven by the proportion of False positives being attributed to the Sad category being significantly larger than the number attributed to the Angry category, $Z = -2.550$, $p = 0.033$. There were no significant differences in the proportion of false positives attributed to the Neutral and Angry categories $Z = -1.886$, $p = 0.177$, or the Neutral and Sad categories $Z = -0.770$, $p = 1$. This suggests that, although the trends seem very similar

in each group, the tendency to attribute Neutral affect stimuli to the Angry category was only significant in the Control group. However, it is possible that the lack of significant differences between the proportions of false positives may be driven by larger standard deviations among the ASD group.

Sad False Positives: From Figure 2.15 it is clear that there is a strong tendency by both groups to incorrectly attribute a larger proportion of their responses to the Sad stimuli to the Neutral category. Analysis revealed that there was a significant effect for both groups (Control $\chi(2) = 15.846, P < 0.001$; ASD $\chi(2) = 12.667, P = 0.002$). Post hoc tests, revealed that in the control group there was a significantly larger proportion of false positives attributed to the Neutral category than both the Angry category $Z = -2.805, p = 0.015$ and the Happy category $Z = -2.805, p = 0.015$, and that there was no difference between the proportions attributed to the angry or happy category, $Z = -1.956, p = 0.15$. The same pattern of difference were found in the ASD group with the proportion of false positives attributed to the Neutral category being significantly larger than the proportion attributed to the Angry category $Z = -2.599, p = 0.027$, and the Happy category $Z = -2.803, p = 0.015$, and also that there was no significant difference between the proportions attributed to the happy and angry categories $Z = -296, p = 1$. This suggests that both groups errors were mostly in misattributing Sad affect stimuli to the Neutral condition.

#### 2.3.3.3 Summary of Results

In the pilot experiment we found that the Control group was significantly better at correctly judging the affect of emotional throwing actions than the ASD group, and that both groups were most accurate for throws with an Angry affect. However, using the same stimuli but showing the whole body instead of just the arm, and age- and IQ-matching a larger sample of control participants, we found that the main effect of Group was no longer significantly different, though Angry stimuli were still categorised correctly more often than the other emotions.

Although the two groups did not differ in terms of proportion correct, either overall or for any individual affect, there were distinct differences in the pattern of errors they made in terms of which actions were confused with which. Looking at the overall total number of False Positives, it seems that the control group made more false positives for Happy stimuli than did the ASD group. Looking in more detail about how these were distributed

amongst the remaining incorrect affects revealed that the control group mis-categorised Happy stimuli as Neutral and Sad to a greater extent than did the ASD group. Though there were no further group differences in terms of the overall number of false positives, looking at how these false positives were distributed among the incorrect affects revealed differences in the pattern of error between the two groups. The ASD group showed a bias to misattribute Angry stimuli as Sad, which was not present in the Control group. Furthermore, both the ASD group and the Control group tended to mis-categorise Happy stimuli as Neutral more so than other affects, though this effect was much more pronounced in the Control group than in the ASD group. For Neutral stimuli both groups appeared to be performing comparably, with less Neutral stimuli being categorised as Angry, than as Happy or Sad. However, the Control group had a significantly higher proportion of incorrect responses in the Sad category than in the Angry category, which was not true of the ASD group, though it may be that this is due to a larger amount of variance in the ASD group. Lastly, it seemed that both groups showed a stronger bias to attribute Sad stimuli to the Neutral category, than to either the Angry or Happy category.

As such, it would appear that the ASD group use a different criterion for categorising Angry and Sad stimuli than do the control group, whilst happy and neutral stimuli seem to be processed comparably between the groups. These differences will be discussed further in the Discussion.

## 2.4 Discussion

A group of participants with ASDs and a group of matched controls performed a battery of experiments aimed at testing biological motion detection (Task 1), action categorisation (Task 2) and affect categorisation (Tast 3). In the pilot version of Task 1 we found that the ASD group required more 'signal' than the control group to detect a point light walker in noise and had lower noise-tolerance thresholds at each level of signal strength. In the main experiment of Task 1 we found no significant differences between the two groups in terms of the number of signal points required to detect the point-light walker, or in terms of the noise tolerance thresholds. In both the pilot experiment and the main experiment in Task 2 we found no significant differences between the two groups on measures of the PSE or the 75% knock thresholds. We did however find that the variance within the ASD group for both experiments in Task 2 was significantly higher than in the control group, more widely spread across the stimulus levels and was centred about the PSE for both groups. In the

pilot experiment for Task 3 we found that the ASD group were significantly poorer at categorising the affect of emotional throwing actions than a control group, and that this difference was driven by the control group more reliably categorising the angry affect throws than the ASD group. These differences were not significant in the main experiment for Task 3. The ASD and control groups were comparable both on overall proportion correct and in proportion correct for each of the different affects. However, there were significant differences between the groups in the patterns of errors made, with the ASD group typically mistaking angry actions for sad actions, whilst the control group typically mistook angry actions for neutral and to a lesser extent happy actions.

The pattern of results from the pilot experiments support the hypotheses based on the suggestions from Atkinson (2009) that tasks requiring global motion and form processing would be more difficult for the ASD group than the control group (Task 1 &3), whilst tasks that require only local motion processing (Task 2) should show no group differences. However, the main experiments in Task 1 and 3 do not support these hypotheses or the pattern of impairments suggested by Atkinson (2009). Each study will be examined in turn with relation to the previous literature, study hypotheses and methodological issues.

### 2.4.1 Detection of Biological Motion

The results of the pilot experiment supported the hypothesis that people with ASD would require more signal points than controls, and would be able to tolerate less noise than controls. However, in the main experiment these differences, although present, were small and non-significant. The most obvious reason for this discrepancy would be the rigorous matching of both age and IQ in Experiment 2. In Experiment 2 each control participant was pair wise matched to a participant in the ASD group, whereas in Experiment 1 only the approximate ages were matched. However, in Experiment 2 both groups FSIQs could be considered to be average or above as both were educated to university level, with the exception of one of the ASD group who had attended a school for the intellectually gifted. Another possibility is that the ASD group in the pilot experiment experienced more severe symptoms than the group used for the main experiment. This is partially borne out by self reports from the participants in Experiment 1 who reported more problems with social situations than the group in the Experiment 2. However, it is not possible to confirm this as there was no common diagnostic measure used for both groups.

The results of the main experiment are more in line with the findings of Moore et. al. (1997), Hubert et. al. (2007), Parron et. al. (2008) and Murphy et. al. (2009), who reported no significant differences in biological motion processing between the two groups and seem to suggest the ASD group used in the current experiment experienced no difficulties in integrating the local motion cues of the PLD into a coherent global whole. However, like Moore et. al. (1997) and Murphy et. al. (2009) the ASD group were slightly poorer at the task than the control group, though the difference was non-significant. Taken together with the findings of Blake et. al. (2003), Annaz, et. al. (2009) and Klin et. al. (2003, 2008, 2009), it seems that there may be some underlying low-level motion or biological motion processing impairment in ASD that manifests in some studies and not others. This may be related to the findings of Walter et. al. (2009), who found that within the autism spectrum, and the more general population, there are a subgroup of people who show a more pronounced local over global processing bias and that this group tend to score more highly on the Systemizing Quotient (SQ). It may be that this sub-group also have more difficulty integrating local motion cues into a global percept and may therefore experience more difficulty in processing biological motion from PLDs. The SQ is not routinely used in research or diagnosis, so as yet it is not possible to confirm or dismiss this idea. However, it does seem to be a plausible explanation for the conflicting results in the literature and in future it should perhaps be included in research, along with other measures such as the Empathising Quotient (EQ), to more fully define the populations being tested.

### 2.4.2 Categorisation of Action

The results of both the pilot experiment and the main experiment suggest that action categorisation is spared in people with ASDs. As Atkinson (2009) pointed out, often actions can be categorised based on some low level local motion cues, as was the case with the stimuli used in these experiments. Both groups were equally affected by the changes in stimuli between Experiment 1 and 2, showing a shift in PSE towards the stimuli with a higher knocking component when the form enhancing skeleton was removed. This suggests that the skeleton increased the salience of the knocking component for both groups, confirming that the ASD group were just as sensitive to the low-level kinematics of the wrist point as the control group.

However, the two groups did differ in terms of within group variance, with the ASD group being significantly more variable than the control group. Furthermore, the variance was

more widely distributed around the PSE in the ASD group than in the control group, suggesting that the consensus within the ASD group was lower across a larger range of stimulus levels. Given that the participants in Experiment 2 were pair-wise matched on age and FSIQ, it would seem that the increased variance in the ASD group is related to the disorder. This is, as mentioned in the previous chapter, a key issue in research in ASD; that there is little homogeneity within those diagnosed with ASD and even within sub-groups such as Aspergers. It may be that the participants within the ASD group are using different subjective criterion or strategies to categorise the action as a knock or a lift, though no evidence of this came out in debriefing. Alternatively, it may be that the participants in the ASD group were variably sensitive to the low level kinematics of the most informative points. Previous research has indicated a potential bias towards low-level features at the expense of global processing in people with ASDs (e.g.Shah & Frith, 1983, 1993; Plaisted et. al. 1998b). If there was such a low-level bias in processing the kinematics of the stimuli used in the current experiment, it would be expected that the ASD group should show a greater sensitivity to the knock component evidenced by a shift in PSE to stimuli with a lower amount of the knocking action in the blended movement. However, as the PSEs were equivalent for both groups and the variance evenly distributed around the PSE, it suggests that within the ASD group there was variability in how sensitive each individual was to the knocking component, or that the participants in the ASD group were perhaps less able to keep a stable criterion for the action category boundaries, suggesting that the internal representation of the movement may have been noisy. What can account for such heterogeneity within the group? It seems likely that within the ASD group there are key differences, perhaps on sub-scales such as SQ or EQ, that can account for these differences. However, as these were not measured in the current experiment, it is beyond the scope of the current thesis to draw any firm conclusions.

### 2.4.3 Categorisation of Affect from PLDs

In Task 3 we tested the ability of 2 groups to categorise affect from PLDs. In the pilot experiment (Experiment 1) only the arm points were visible and the velocities of the wrist points were normalised to the median velocity of all the actions, eliminating velocity as a potential source of information. In Experiment 2 the PLDs showed all fifteen points of the unaltered throwing actions of the actors. The results of Experiment 1 support previous findings that people with ASDs are poorer at categorising affect from PLDs of human actions (Moore et. al. 1997; Hubert et. al. 2007; Parron, et. al. 2008) and are particularly

poor for angry actions (Atkinson, 2009). However, in Experiment 2 there were no significant differences between the two groups in overall proportion correct or for any specific affect. There were however differences in the distribution of errors between the two groups. In terms of the overall number of errors made, the control group attributed significantly more happy actions to the neutral and sad categories than the ASD group. In terms of the proportion of errors made, a measure independent of overall error rate, suggest that the ASD group attributed angry actions more to the sad category than did the control group, and that the bias observed in the control group to attribute neutral actions to the Sad category was not shared by the ASD groups. This suggests that although performance overall was comparable between the two groups, the ASD group may have been using a different strategy to obtain the correct response than the control group. Some evidence from this may come from a comparison of the stimuli used in each of the experiments.

The two main differences between the stimuli of the two experiments were that, whereas Experiment 2 showed the non-manipulated whole body of the point-light actor, Experiment 1 showed only the arm of the actor with velocity removed as a cue to affect. According to Atkinson (2009), participants with ASD should be more impaired when global processing of a complete point-light actor is required. As such, it seems that the results of Study 3 contradict the idea that global processing deficit is responsible for poor affect categorisation in ASDs.

It may be that the additional information presented in the whole body PLDs, such as the postural cues, was sufficient to bring performance of the ASD group up to the same level as ASDs. However, this would not explain why the results of Experiment 2 are different from those found in the literature. There is a key difference between the stimuli used in the current Experiments to those used in previous studies. In previous studies PLDs are generated from actors asked to perform emotional gestures, such as shaking fists for angry. Although, typically developed people are particularly good at these tasks, these actions are less common than those used in the present study. Usually, it is the manner in which people carry out everyday actions that provides insight into the emotional states of others. The stimuli used in the present study require affect to be categorised from stimuli more comparable to these latter, more natural situations, in that the affect must be determined based on subtle differences in the kinematics of a throwing action than from overt emotional gestures. It may be that, as these stimuli are more closely akin to those experienced more regularly by people with ASDs in interactions with family members,

caregivers and friends, that this group have developed strategies to identify the emotions of those around them. This seems quite plausible in the group tested for this study, as they are all high-functioning and most are able enough to have attended institutions such as universities that require interactions with others. Being high-functioning and having above average IQs may mean that this group have developed strategies to identify the emotions of others, though these strategies may be quite different from the automatic affect recognition that occurs in typically developed people. A key point that came out in the debriefing of the ASD participants after this task was that many had intricate strategies based on the velocity of the wrist, the total movement of the wrist and the "jerkiness" of the motion, whereas the control participants experienced far more difficulty in specifying how they made their decisions. These strategies were more complex than a simple velocity heuristic, as the confusion of the ASD group between angry actions, which are by far the fastest, and sad actions, which are the slowest, excludes this option. More generally, the different pattern of errors between the two groups lends support to the notion that the two groups used different strategies, as it would be expected that if two groups were using the same processes and strategies that the pattern of errors would be the same.

In light of the findings from the current study and those of previous studies, it seems plausible that given sufficient information and experience with affective gestures, people with ASDs can attribute affect comparably with controls, potentially utilising different learned strategies to overcome impairments in affect categorisation.

### 2.4.4 General Summary

In this chapter we used three experiments to test biological motion detection, action categorisation and affect categorisation in groups with and without ASDs. Despite significant differences between the groups in two of the pilot tasks, in the main experiments that utilised a larger sample and an age- and FSIQ-matched control group, people with ASDs performed comparably to the control group in all three tasks. Considering the results of the pilot tasks and the main experiments together, it seems that the ASD group tested were more variable in terms of maintaining stable criterion when processing low-level kinematics of actions and may have been utilising different processes, strategies and criteria to achieve the same level of performance as the controls. Furthermore, the underlying variability in the ASD group tested may reflect differences in

processing abilities, particularly in processing local kinematics of actions and the integration of local elements into coherent wholes.

Overall, the experiments in this chapter support the findings of Moore et. al. (1997), Hubert et. al. (2007) and Parron et. al. (2008) that people with ASDs are comparable at processing biological motion to controls, but not the findings of impaired affect categorisation. This may be due to the group being high-functioning adults with above average intelligence, which could potentially have given them the time and the mental resources to develop strategies to offset any processing difficulties that were present in earlier life. This is partially supported by recent fMRI studies that have revealed that, despite equivalent behavioural performance in low-level feature processing of biological motion and action categorisation in adult groups with and without ASDs, the brain regions that are active during these tasks are different for each group (Herrington et. al. 2007; Freitag et. al. 2008). In particular, regions of the temporal lobe seem to respond to biological motion stimuli in controls, but not in people with ASDs. This suggests that the underlying neural architecture used when performing these tasks may be different for each group whilst still facilitates comparable performance.

Given the difficulty in studying people with more severe ASDs and in particular children with ASDs, it is quite often necessary to work with high-functioning adults in order to understand the processing impairments in the more difficult patient groups. Given that performance on behavioural tasks seems to more closely resemble that of controls as people with ASD get older, it is increasingly important to determine which processes recruit typical and atypical neural resources. For this, it is essential to utilise extreme rigour in methodological issues as well as advanced imaging techniques and analysis. In future, it may become more important to study similarities in cognitive social abilities in conjunction with differences in how these abilities are accomplished in order to understand the difficulties of those who show more severe impairments.

# Chapter 3: Novel Stimuli for Quantifying the Contribution of Configural Cues in PLDs

## 3.1 Introduction

In the previous chapter we found that people with ASDs did not seem to show any impairment in integrating local motion signals from PLDs into the percept of a human actor. However, there were some indications that the strategies by the ASD group to achieve this were different from the control group. If this was the case, then it is likely that the underlying neural systems used to process biological motion may show significant differences. In order to test this, it was necessary to develop stimuli that could be manipulated to quantify the amount of configural information available in PLDs, and also a method to determine the effect that the addition of structure has on both behavioural measures and measures of brain activity. This current chapter sets out to describe the development of such stimuli, taking into account the literature to date on biological motion processing in typically developed individuals. Furthermore, the new stimuli will be used to investigate the role that configural cues play in direction discrimination from PLDs in a typically developed group.

In humans, the ability to perceive the motions of other animate creatures has been extensively studied and shown to be surprisingly robust. Typically, the study of biological motion has involved using point-light displays that reduce human movement to a small number of moving points placed on the joints. Using this technique Johansson (1973; 1976) showed that when these point-light displays were presented as static frames, observers were unable to identify the stimuli. However, when presented dynamically, observers immediately recognised the stimuli as representing a human being and could readily identify complex actions. Later research has demonstrated that an extensive number of person properties can be gleaned from these stimuli, including the identity of the actor (Cutting & Kozlowski, 1977; Hill & Pollick, 2000; Jokisch, Daum, & Troje, 2006; Loula, Prasad, Harber, & Shiffrar, 2005; Troje, Westhoff, & Lavrov, 2005) the gender of the actors (Barclay, Cutting, & Kozlowski, 1978; Cutting, 1977; Jordan, Fallah, & Stoner, 2006; Kozlowski & Cutting, 1977, 1978; Pollick, Kay, Heim, & Stringer, 2002; Troje & Geyer, 2002; Troje, Sadr, Geyer, & Nakayama, 2006), the emotion of the actors

(Clarke, Bradshaw, Field, Hampson, & Rose, 2005; Dittrich, Troscianko, Lea, & Morgan, 1996; Pollick, Paterson, Bruderlin, & Sanford, 2001) and also the nature of the actions being carried out (Dittrich, 1993).

Although the information available in point-light displays supports the recognition of a variety of person properties, the perception of these displays seems particularly affected by temporal manipulations in the local motions of the individual joints that result in disrupted phase relations between the points on the walker (Bertenthal & Pinto, 1994; Grossman & Blake, 1999), and by playing the motion abnormally slowly (Beintema, Oleksiak, & van Wezel, 2006). However, provided the phase relations between the points are preserved and played at a biologically plausible speed, the perceptual system's ability to detect biological motion is surprisingly resistant to distortions of the walker or the embedding of the walker in noise. For instance, limiting the lifetime of the points on the walker or displacing them to points on the skeleton, as opposed to joint locations, barely diminished observers' recognition of point-light walkers (Beintema & Lappe, 2002; Mather, Radford, & West, 1992; Neri, Morrone, & Burr, 1998; Pinto & Shiffrar, 1999). Furthermore, masking the motion of the points using random dynamic noise dots, which is one of the most widely used forms of masking, still does not greatly reduce the impression of the stimulus as a human walker (Bertenthal & Pinto, 1994; Cutting, Moore, & Morrison, 1988; Ikeda, Blake, & Watanabe, 2005), unless this masking is used in combination with disruptions in the phase relations between the joints (Hiris, Humphrey, & Stout, 2005). Masking the walker in this way renders local motion cues ineffective for detecting biological motion or discriminating the direction of motion, forcing the perceiver to rely on global or configural cues. Even when the masking dots contain local motion signals identical to those of the walker, large numbers are required to diminish the impression of a human walker (Thornton, Pinto, & Shiffrar, 1998).

As would be expected, spatially scrambling the local motion of the joints to new locations produces a decrease in performance on tasks involving the discrimination of direction of locomotion of point-light walkers (Troje & Westhoff, 2006). These disruptions in configural information result in the loss of the perception of global structure from motion and reduce the walker to its unordered local motion components. It is argued that this breakdown in configural information is also responsible for the apparent inversion effect seen in biological motion processing (Bertenthal & Pinto, 1994; Pavlova & Sokolov, 2003; Sumi, 1984). Perception of inverted point-light walkers is impaired relative to upright

walkers and is independent of the location of the source of gravity (Shipley, 2003; Troje, 2003). However, despite this inversion effect, preservation of certain elements in the display enhances performance in the task. Troje and Westhoff (2006) demonstrated that, even when all points but the feet are inverted, observers were still able to accurately discriminate the direction of motion of a point-light walker embedded in a static limited lifetime mask. Furthermore, this effect persists even when the walker is scrambled, although to a lesser extent, arguing for a diminished role of configural cues and a more specialised mechanism that is tuned to the motion of the feet and their relation to gravity.

Although it has been demonstrated that local motion cues alone seem to be able to provide a large amount of information about the direction of locomotion of point-light walkers (Troje & Westhoff, 2006), there have been a number of studies that, using a variety of techniques, have shown the importance of configural cues in biological motion perception. For instance, detection and direction discrimination of point-light walkers seems to be preserved in the absence of local motion cues, such as in the case of the limited lifetime displays used by Bientema and Lappe (2002). In these displays points are randomly selected and made visible on the walker for between two to eight frames, before disappearance and replacement by another point. As such, in displays where the lifetime of points is short, such as two or three frames, there is little or no consistent local "image motion". However, observers are still able to recognise the signal as a human moving figure, with performance comparable to that with classical point-light stimuli (Beintema & Lappe, 2002). Bertenthal and Pinto (1994) demonstrated that even when masking noise was made up of arm and leg components, participants were able to detect whether a walker was present or absent in a display, suggesting that global form information may be processed prior to local configural elements. In support of this, Pinto and Shiffrar (1999) showed that detection of a walker was impaired when the four limbs were randomly positioned in the same walker space as an intact walker, though performance was still above chance as found with other scrambled walkers (Troje & Westhoff, 2006). Pinto and Shiffrar also compared detection of point-light walkers in visual noise when certain subsets of points were omitted with intact upright and inverted walkers. They found that omission of the central elements of the display (shoulders & hips) resulted in drastic drops in detection of the walkers in dynamic noise masks. Furthermore, detection was not impaired relative to the intact upright walker when the extremities (wrists & ankles) were omitted. This suggests that the central trunk of the walker is more important than the extremities in detecting walkers in noise. Mather & Murdoch (1992) however, found that in a direction

discrimination task, the omission of the extremities proved to be the more detrimental condition. These findings together suggest that different tasks call upon different configural properties of the point-light walkers, rather than individual local elements. Most studies on configural processing in biological motion have however, used large numbers of masking points to disguise the walker's structure.

In addition to the behavioural evidence on the role of configural cues, a number of recent models have had considerable success in modelling processes involved in biological motion using a combination of configural cues and global motion without a need to resort to local motion. For instance Lange and Lappe (2006) have developed a model of biological motion processing based on existing knowledge of template cells that respond to static postures. The model assumes two stages, one in which the static posture information is sequentially captured by the template cells, though with no knowledge of the temporal order, and a second stage in which the global motion is analyzed by explicitly analyzing the temporal order for a set of selected frames. Not only is the model neurally plausible, but Lange and Lappe also claim that the model can explain how biological motion processing is achieved in the presence of interfering noise. Furthermore, it seems feasible that such a system would be able to deal with the limited lifetime displays of Bientema and Lappe (2002), and also explain why some patients with severe bilateral lesions of MT are capable of seeing biological motion but are unable to integrate low level motion (McLeod, Dittrich, Driver, Perrett, & Zihl, 1996; Vaina, Cowey, LeMay, Bienfang, & Kikinis, 2002). If these models are correct, then it would be expected that tasks such as direction discrimination should be greatly enhanced by the inclusion of configural information, and would not rely on sub-configurations or local motion cues to perform tasks such as detection and discrimination of point-light walkers. In support of these models, single cell data from monkeys has shown that there are two distinct groups of neurons in the temporal lobe that are selective for static form and motion (Vangeneugden, Pollick, & Vogels, 2009). They found motion selective neurons in the upper fundus of the STS and snapshot neurons (Giese and Poggio 2003) which were selective for static postures in the lower fundus of the STS and the inferior temporal convexivity. Furthermore, fMRI studies in humans have revealed that the human extrastriate body area and fusiform body areas seem to activate to static images of human postures, while areas such as the STS are selective for moving biological displays (Peelen, Wiggett & Dowling, 2006; Jastorff & Orban, 2009; Grossman & Blake, 2002).

The present study set out to use novel point-light stimuli to examine the specific contribution that configural cues made to the perception of biological motion in a direction discrimination task. These novel stimuli used only the 15 points of a point-light walker, and varied the amount of configural information present by dividing them into two subsets. The target subset maintained their original joint locations but were accompanied by dynamic noise that was made up of the remaining points. These non-target points were scrambled to new locations and had their trajectories flipped across the horizontal axis. This ensured all displays had equivalent local motion information and varied only in the amount of configural information present. A second condition was used in which both sets of points were scrambled in space and had opposing trajectories; essentially both signal and noise lacked any configural cues. Although the technique does not exclude the option of manipulating the temporal phase relations between the joints, we chose not to do this in order to examine the specific effect of manipulating the configural information. Participants were asked to judge the direction of motion of the walker and we expected that, when configural cues were present, accuracy would be higher and performance would improve more sharply. In addition, we modelled the probabilities of certain sub-configurations being present in the signal point group, in the configural cues present condition to determine whether these could predict the pattern of results we found.

## 3.2 Methods

### 3.2.1 Participants

Eleven female and 5 male undergraduate students (Mean age = 22.4, SD = 2.1) consented to take part in the experiment. All had normal or corrected to normal vision.

### 3.2.2 Design

The task took the form of a two alternative forced-choice direction discrimination paradigm with 2 conditions: configural cues present and configural cues absent. There were eight stimulus levels in each condition, which were the number of signal points shown (1, 3, 5, 7, 9, 11, 13, and 15). The number of noise points always equalled 15 minus the number of signal points, ensuring that all stimuli comprised a total of 15 points. There were 20 trials per stimulus level in both conditions giving a total of 320 trials per person.

### 3.2.3 Stimuli Generation

Stimuli were presented on a CRT monitor with a resolution of 1280 X 1024 pixels, on a display size subtending 20.4° of visual arc by 15.36° with a refresh rate of 60 Hz at approximately 1m from the participant. Point-light stimuli were generated from an existing motion capture library (Ma, Paterson, & Pollick, 2006) and displayed and manipulated within the experiment using the Psychophysics Toolbox for MATLAB (Brainard, 1997; Pelli, 1997). The translational motion of the walker was subtracted from the displays so that the walker appeared as if it was walking on a treadmill. Each stimulus was presented for 60 frames (1 second) and showed a full gait cycle. The point-light walkers were scaled to a height of 200 pixels with the resulting figure subtending a visual angle of approximately 3.02° by 1.32° and consisting of white dots on a black background. The point-light walkers were randomly jittered horizontally and vertically within a window subtending a visual angle of 5.43° by 3.70°. The starting frames of the displays were at the points of minimal distance between the wrist and ankles. This was done because, in experiments without large additional noise masks, this point in a display contains the least amount of configural information, making it more difficult to discriminate scrambled displays from intact displays (Thirkettle, Benton & Scott-Samuel, 2009), which was important in this study in order to minimize any perceptual difference between the conditions other than those of experimental interest.

#### 3.2.3.1 Configural cues present

Walkers consisted of fifteen points and were split at random into two subsets; signal points and noise points. Those in the signal category were selected randomly from the 15 walker points and maintained their existing joint locations and trajectories. The direction of motion of the signal points was randomly varied between leftward and rightward motion. Those points not selected as signal points were spatially scrambled at random to new available joint locations. Furthermore, the trajectories of the noise points were flipped along the horizontal axis. The resulting displays therefore, had two opposing motion signals, one that preserved configural information (signal points) and another that contained no configural information (noise points). As all points came from the same walker the local motion signal contained in every display was the same, only the quantity of configural information and direction of motion was varied between trials. See Figure 3.1 for an illustration of the technique.

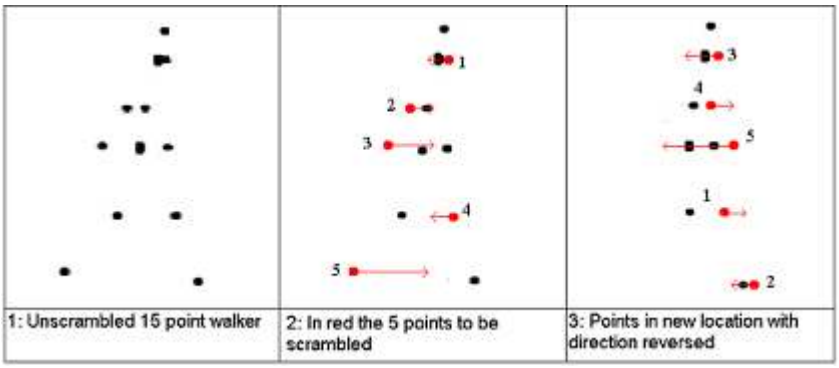


Figure 3.1: The first panel depicts the 15 points in a static first frame of an unscrambled point-light walker. The second panel shows the points that are to be scrambled and the original starting trajectories in red. The last panel shows where each of these points is scrambled to and the resulting direction of motion after the trajectories have been flipped. (Gait and stance have been exaggerated for clarity)

In the specific case in which there was only 1 noise point, and hence no other available location to be moved to, the point had its horizontal coordinate inverted and its trajectory flipped, as with all other noise points in other conditions. As such, although not scrambled to a new joint location it was still scrambled in space and provided an incorrect motion signal relative to the signal points. Beyond 1 noise point the number of possible configurations increases exponentially. For instance, in a condition in which there are 3 noise points, given that these noise points can be any of the 15 points at random, there are 2184 possible configurations of these points and by 5 noise points, there are 240240 possible configurations.

#### 3.2.3.2 Configural cues absent

In the configural cues absent condition the generation of the noise points was exactly the same. However, the signal points were also scrambled to new, free locations but maintained their original trajectories. This produced stimuli with two opposing motion signals with no configural information present in either subset. Thus the only difference in the signal subset between conditions was whether the original joint locations had been preserved or not.

### 3.2.4 Procedure

Each participant was instructed to view point-light walkers on the screen. Upon the completion of each stimulus presentation the participant was required to decide whether

the walker on the screen was moving from left to right or from right to left, using the arrow keys on the keyboard. A correct answer in the configural cues present condition was considered to be when the participant chose the direction of motion of the points with preserved trajectories and joint locations. In the configural cues absent condition a correct answer was operationally defined as when the participant chose the direction of motion of those points that had preserved their original trajectories, in essence the direction of locomotion of the original point-light display. This preserved a scale of proportion correct, allowing a direct comparison of performance in both conditions on a scale of 0 to 1, which would not have been possible with other definitions of "correct" such as the direction with the most overall energy.

Each participant completed a set of practice trials displaying several levels of stimuli from both conditions. Once a participant had completed these and were comfortable in the task, the main experimental blocks commenced. In both conditions, each of the eight stimulus levels were presented once in each of the twenty blocks, giving 160 trials per condition. Which condition each participant saw first was counterbalanced to remove any possible order effects. The experiment took just over 30 minutes to complete.

## 3.3 Results

The proportion of correct responses was calculated for each level of stimuli in both conditions for all participants and the data were fitted with a cumulative Gaussian using the psignifit toolbox for MATLAB (Wichmann & Hill, 2001a, 2001b). Slopes and 75% correct thresholds were calculated for each participant and for the group as a whole. Two participants were excluded from the analyses, as they failed to reach the minimum performance criterion of 75% correct in the configural cues absent condition. Note however, that had the two participants been included the difference between the two conditions would have been larger.

Figure 3.2 shows the fits for the data averaged across the remaining 14 participants. As can be seen, slopes in the configural cues present condition (m = 0.088, 95% CI = ±0.006) are steeper than in the configural cues absent condition (m = 0.056, 95% CI = ±0.004). Furthermore the 75% correct thresholds in the configural cues absent condition (m = 12.369, 95% CI = ± 0.463) are higher than those in the configural cues present conditions (m = 7.748, 95% CI = ± 0.272). T-tests on the combined data reveal that these

differences were significant (slope $t(13) = 7.155$, $p<0.001$; 75% threshold $t(13) = 8.732$, $p<0.001$).

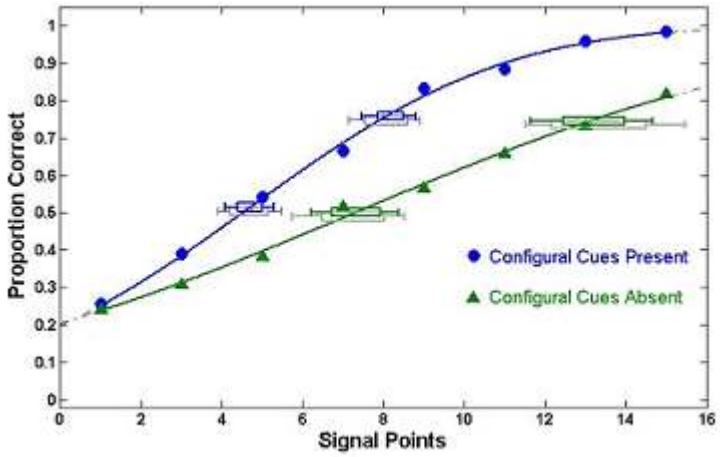


Figure 3.2. Circles represent the data points from the configural cues present condition, whilst the solid line represents the best fit cumulative Gaussian. Similarly, the triangles represent the data points from the configural cues absent condition, whilst the broken line represents the best-fit cumulative Gaussian. Error bars are placed at the 50% and 75% thresholds. Dark boxes and lines represent 95% confidence limits and light boxes and bars represent the worst case confidence limits encountered during boot-strapping sensitivity analysis (Wichmann & Hill, 2001a, 2001b)

Note also that in the configural cues absent condition, performance varies only between 20% and 80% correct. This is to be expected from the results of Troje and Westhoff (2006), as they found that accuracy in a direction discrimination of a scrambled point-light walker was approximately 80%. So in this experiment when all points were moving in the target direction participants responded correctly on 80% of trials, but when almost all the motion was in the opposite direction, participants responded correctly only 20% of the time. This is also the reason that in the configural cues present condition, performance does not drop below 20% as these displays are essentially equivalent between the two conditions at 1 signal point.

*Possible Confounding Sub-Configurations*

It is possible that a rule-based decision-making process, based on a sub-configuration of points on the walker, could account for the pattern of performance seen in the configural cues present condition. For instance, if a shoulder, elbow and wrist in the correct locations were sufficient to correctly judge the direction of motion, then it is likely that the proportion of trials on which these configurations would occur could predict the proportion correct results. Other potential sub-configurations that could influence direction discrimination would be both feet being in the signal group or all three leg points, or arm

points, or indeed both arms and legs being in the signal group. Such a strategy would be reliable only when the sub-configuration was present in the signal point category. As such, it is possible to calculate the likelihood that a sub-configuration of k points occurs within the signal point group of size n as defined by equation 1.

$$(1)\quad \frac{C_k^n}{C_k^{15}} = \frac{(15-k)!\,n!}{15!(n-k)!}$$

The critical issue here is the likelihood that these sub-configurations are present in the signal subset of points. As the number of signal points increases, so does the likelihood that any sub-configuration is present, and also the likelihood that larger sub-configurations are present. Figure 3 shows the probability distributions for each potential number of points in a sub-configuration for each level of signal points.

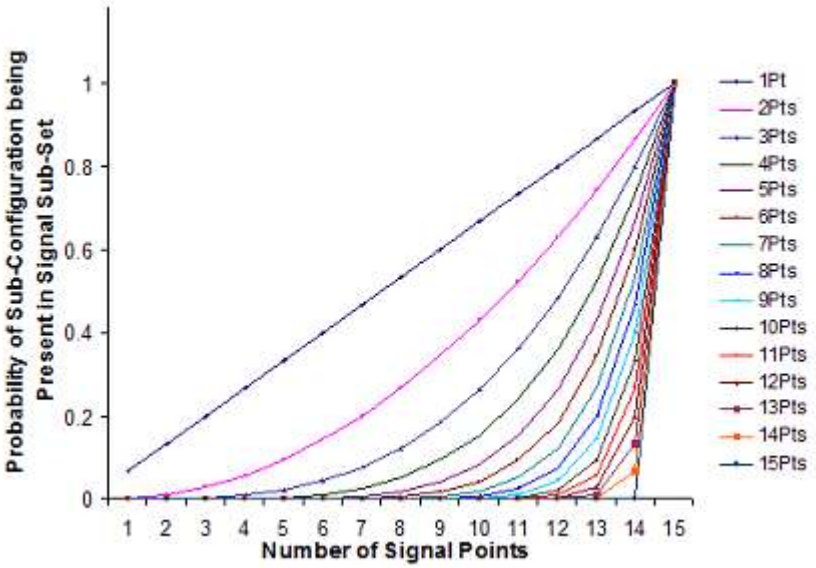


Figure 3.3: Probability of a sub-configuration being present given x signal points. Different coloured lines represent sub-configurations of different numbers of points.

Had participants been consistently using a strategy based on a specific number of points then the pattern of responses would follow the patterns seen in Figure 3.3. It is clear however that this is not the case ruling out the use of any specific sub-configuration.

## 3.4 Discussion

The results presented above suggest that configural cues play a crucial role in biological motion perception. The 75% correct threshold was significantly higher in the configural cues absent condition than in the configural cues present conditions, indicating that significantly more points were needed to reliably judge the direction of motion in the absence of configural information. In fact, the 75% correct threshold is reached using approximately five fewer points with configural cues than without. Furthermore, the mean slope of the curve in the configural cues present condition was steeper than in the configural cues absent condition, indicating that it was not merely the case that it was the number of points that was important, but that each additional point placed in the correct joint location improved performance far more than an additional point placed in a random joint location.

In support of this, looking at the probabilities that any combination of points within the signal point sub-group form any specific sub-configurations that could account for the results, it is clear that the probability distributions shown in Figure 3.3 do not match the pattern of results observed in the behavioural data. Had participants been consistently using a specific sub-configuration, or set of sub-configurations of points rather than the global configural cues, it would be expected that the behavioural results would be more like the probability distributions than the pattern observed. Some participants did report trying to use a strategy based on tracking the feet or wrist but said that it became too difficult given the brief displays and the lack of predictability in the position of the chosen target motion. This makes it unlikely that simple local configurations, such as the two feet being in the correct locations or all arm points being in the correct location, were sufficient to offset the opposing local motion signals without the other signal points that were outside the local configuration. It may be therefore, that, as proposed in recent models (Giese & Lappe, 2002; Lange & Lappe, 2006), there is some sort of template matching combined with temporal analysis, that is independent of any simple local motion analysis or analysis of sub-configurations. Indeed, this would seem to be borne out by some of the subjective reports of participants who claimed that in some of the conditions, those in which there was little or no true configural information, they had very occasional impressions of such things as “hula dancers” or “upside down rollerblading”, indicating what could be incorrect temporal integration of biologically plausible templates.

This is not to say though, that there are not certain sub-configurations, or indeed local motions, that convey more information than others. It seems likely that the feet and other extremities will convey more information about locomotion than the shoulders or head (Pinto & Shiffrar, 1999; Thurman & Grossman, 2008; Troje & Westhoff, 2006). Moreover, it has been demonstrated that emotions can be partially categorised using just the kinematics of the arm movements (Pollick et al., 2001), and that these kinematic cues can be sufficient to determine emotion in the absence of form information and temporal information, though poorly in comparison to when these cues are present (Atkinson, Tunstall, & Dittrich, 2007). There are also several studies that demonstrate the importance of hip and shoulder swing in gender categorisation (Johnson, Gill, Reichman, & Tassinary, 2007; Mather & Murdoch, 1994). The novel techniques devised for the current experiment may provide a convenient way to examine these relations. It would be possible to specifically manipulate which sub-configurations are present within displays, and examine the importance of each in different tasks without the need to use large numbers of masking dots that can differentially engage active and passive attentional mechanisms (Thornton, Rensink, & Shiffrar, 2002; Thornton & Vuong, 2004) or alter phase relations between joints or limbs (Neri, 2009), which essentially disrupts or degrades the coherence of the walkers.

These considerations are particularly important in fMRI studies investigating biological motion processing. For instance, Grossman, Blake and Kim (2004) found that there was a large amount of variance in the number of noise points it took to mask a point-light walker even with training, meaning that to test people at different performance thresholds required displays with differing levels of noise points, and hence motion energy, in each display. The stimuli used in this paper provide a means for testing participants at individual thresholds without having to increase the overall motion signal in the stimuli. This would also make them ideal stimuli for examining sensitivity to biological motion in patients with MT lesions (McLeod et al., 1996; Vaina et al., 2002) and autism spectrum disorders (Freitag, Konrad, Haberlen, Kleser, von Gontard, Reith, Troje, & Krick, 2008; Herrington, Baron-Cohen, Wheelwright, Singh, Bullmore, Brammer, Williams, 2007; Simmons, Robertson, McKay, Toal, McAleer, & Pollick, 2009).

In summary, the current experiment has shown that configural information contributes significantly to direction discrimination of point-light walkers. Furthermore, this novel technique allows for the masking of point-light walkers without adding large numbers of

noise dots or disrupting the coherence of the point-light figure. In the next chapter this technique will be used to explore the brain regions responsible for processing structure from biological motion in typically developed adults.

# Chapter 4: Neural Basis of Configural Processing of Biological Motion

## 4.1 Introduction

The perception of human movement is a fundamental ability for everyday life and appears to be innate and automatic. The human visual system seems able to process this information and derive a wide variety of information from even impoverished stimuli, such as point-light displays (see Blake and Shiffrar, 2007 for a review). In order to accomplish this, the brain must process information from local and global motion cues contained in the dots of the PLDs, and integrate them with form cues into a coherent percept of a human. Once integrated, the salient features must be extracted for the task at hand, whether this is action categorisation, intention recognition, goal perception or emotional understanding. As such, the brain must utilise a range of different regions in order to accomplish such tasks, with each one requiring different neural systems.

The most fundamental process, that of integrating local motion cues into a coherent percept of a human, is the least well understood. Early attempts to localise the brain regions that process biological motion typically contrasted intact and scrambled PLDs to find regions that were selective to human form. Howard, Brammer, Wright, Woodruff, Bullmore and Zeki (1996) compared coherent motion, optic flow and PLDs to static displays, random dot motion and scrambled biological motion. They identified areas of V5 and the Superior Temporal Gyrus (STG) that were anatomically distinct based on the type of motion. Howard et. al. (1996) attributed the activity outside the visual cortex (STG) to information being passed to the auditory cortex and suggested that these areas were responsible for the association of visual information with action representations. This was partly based on an earlier experiment that localised auditory areas in the STG, and also on work showing that a similar area in the monkey brain, the superior temporal polysensory area (STPa), contains neurons that are responsive to visual, auditory and somaesthetic stimuli.

Subsequent studies have revealed the importance of this temporal region in processing biological motion. Grossman, Donnelly, Price, Pickens, Morgan, Neighbor and Blake (2000) compared brain activity when viewing intact point light displays with random dot motion, and found that a posterior portion of the STS was more active for biological

motion, predominantly in the right hemisphere. They also reported increased activation in the cerebellum in response to biological motion stimuli. Vaina, Solomon, Chowdury, Sinha and Belliveau (2001) also found increased activation to intact PLDs over scrambled PLDs in the STSp and cerebullum, but also reported differential activation in MT+, the cuneus and precuneus, the parieto-occipital junction, the lingual gyrus, fusiform gyrus and parahippocampal gyrus, the ventral extrastriate body area(EBA) and 2 regions in the STSp corresponding to BA22 and BA38.

In a study comparing brain activity when viewing either videos of human actions, videos of moving tools, PLDs of human actions or PLDs of moving tools, Beauchamp, Lee, Haxby and Martin (2003) found that STSp and lateral fusiform gyrus both preferred human motion over tool use. This was true only for videos of human motion in the lateral fusiform areas, which showed minimal response to PLDs. However, the STSp was strongly activated for PLDs and more strongly activated for videos of human motion. Beauchamp et. al. (2003) claim that this suggests that the main contributors to the lateral fusiform gyrus are form, motion and colour whereas the STSp is responsible for the integration of these sources of information. Using a blocked design of intact PLDs of human action, scrambled PLDs and static dot displays, Saygin, Wilson, Hagler, Bates and Sereno (2004) found that the STSp also preferentially activates to scrambled PLDs of biological motion, when compared with static displays, but responds more to intact PLDs than scrambled stimuli. Furthermore, Saygin et. al. (2004) found that along with the STSp and the lateral temporal cortex reported by Beauchamp et. al. (2003), the inferotemporal cortex preferred intact over scrambled PLDs, as did the Inferior Frontal Sulcus (IFS) and the Inferior Pre-central Sulcus. These latter two areas have been implicated in action observation and the Inferior Pre-central Sulcus, due to its inclusion of the premotor area, has also been associated with the Mirror Neuron System (MNS) (e.g. Rizzolatti, Fadiga, Matelli, Bettinardi, Paulesu, Perani & Fazio, 1996b; Grézes, Costes & Decety, 1998; Grézes & Decety, 2001; Grézes, Armony, Rowe & Passingham, 2003; Grafton, Arbib, Fadiga, & Rizzolatti, 1996; Iacaboni, Koski, Brass, Bekkering, Woods, Dubeau, Mazziotta & Rizzolatti, 1999; Casile, Dayan, Caggiano, Hendler, Flash & Giese, 2009). It must be noted however, that these regions are typically only found when tasks involve imitating or mentally imitating goal-directed actions. The IFG and ventral pre-motor areas have been reported in response to non-imitative processing of PLDs, but they are preferentially active when the human form is disrupted, such as in inverted PLDs (Grézes, Fonlupt, Bertenthal,

Delon-Martin, Segebarth & Decety, 2001), or to non-human movements such as animate tool movements (Beauchamps, et. al. 2003).

It seems that the most robust finding in investigations comparing intact PLDs with scrambled PLDs is the STS, the inferotemporal cortex and the FBA. Peuskins, Vanrie, Verfaillie & Orban (2005) recently used a very elaborate design to determine the precise role of the STSp, MT+ and the ITG in biological motion processing. In a series of 5 experiments, each using precise manipulations of PLDs to selectively test processing of the motion signal, form signal and the action itself, Peuskins et. al. (2005) showed that the STSp was selective for the action portrayed in the PLD, the ITG was selective for form information, which would be expected given that it is part of the ventral processing stream, and that MT+ was selective for the processing of the complex motion signals contained in the PLDs. This finding supports the suggestion of Beauchamp et. al. (2003) that the STSp seems to integrate the various components of biological motion and processes the action as a whole. Peuskens et. al. (2005) argue that in the case of implied biological motion from static figures, the ITG can feed back the implied motion signal to MT+ to be sent to the STS. They also postulate a link from the dorsal stream (ITG) to the ventral stream (STSp) in which the motion signals, which are initially sent separately to the ITG and STS, are integrated to represent the action in the STSp.

Recently, increased attention has been paid to the roles of the fusiform body area (FBA) and the EBA. The EBA has been shown to respond strongly to human form (Downing, Jiang, Shuman & Kanwisher, 2001) and more recently body-selective responses have been found in regions of the posterior fusiform gyrus (pFG), close to the fusiform face area (Downing, Chan, Peelen, Dodds & Kanwisher, 2005). Some of the aforementioned studies have also found that these areas are more selective for intact biological motion over scrambled biological motion (Vaina et. al. 2001; Beauchamp et. al. 2003). Peelen, Wiggett and Downing (2006) recently investigated the functional and anatomical distinctiveness of these regions in the processing of biological motion. After localising MT+ and the EBA in the inferior temporal cortex, and the FFA and FBA in the pFG, Peelen, et. al. (2006) measured activity in each of the regions using a standard design of contrasting intact PLDs of human actions with scrambled versions of the same actions. They found that all the regions defined responded more strongly to intact biological motion than to scrambled biological motion, but that these effects were far more pronounced in the EBA and FBA. They then analysed the voxel by voxel correlations between biological motion selectivity

and selectivity for the localiser stimuli within each of the defined regions of interest (ROIs). They found that biological motion selectivity was significantly correlated with body selectivity in all ROIs, but not with motion selectivity or face selectivity. The finding that areas of the posterior fusiform gyrus showed correlations between body selectivity and biological motion confirm the dorsal stream's role in form processing. However, the finding that inferior temporal regions showed increased selectivity for form and biological motion than for non-biological motion is somewhat unexpected, given that the MT complex is strongly involved in motion processing (Downing, et. al. 2001). Peelen et. al. (2006) offer two potential explanations of this. The first is that within these regions there are two types of neurons distributed within the regions, one that is responsive to motion and another that is responsive to the visual features of the body. The second explanation is that each and every neuron is responsive to both form and motion, and that selectivity is due more to the size of the response to either of the two stimulus types. Both explanations require body and motion selectivity to be interleaved on a very fine scale within these regions, though the results support findings that there is consistent variation across voxels in relative proportions of neural selectivity of body and motion selectivity, without an absolute distinction between the two populations (e.g. Haynes & Rees, 2005; Kamitani & Tong, 2005). Peelen et. al. (2006) suggest that differences between the EBA and FBA areas and the STS are that the former two areas are selective for form information, whereas the STS is activated by specific patterns of motion through which form may be derived, which is similar to the idea proposed by Beauchamp et. al. (2002) that the STS integrates the motion signals into a global percept.

Downing, Peelen, Allison, Wiggett and Tew (2006) recently investigated the role of the EBA in action perception. Using movies of either continuous actions (coherent) or videos generated by splicing together individual frames from different actions (incoherent), they showed that STS and MT+ were more active during observation of coherent actions. Furthermore, they found that parts of the inferior-parietal sulcus/post-central gyrus and inferior frontal gyrus also showed a preference for coherent actions. These regions, as mentioned above, are linked with action observation and motor control. However, they also found that the EBA was actually more active for incoherent actions. Downing et. al. (2006) argue that, as the EBA was selective for videos of unrelated posture and not continuous actions, this region is selective for static form information and neural activity decreases, or adapts, when presented with images that depict relatively similar postures.

The picture that can be built up of the neural basis of biological motion processing seems to be that from motion sensitive areas, such as MT+, the information is passed on either as static form information through the ventral stream to areas like the EBA and FBA (Peelen et. al. 2006; Downing et. al. 2006; or as motion information to the dorsal stream to STS where it is processed as articulated body movements (Vaina, et. al. 2001; Beauchamp et. al. 2002; Grossman & Blake, 2002), perhaps integrating the local motions into a global percept using the form information that has been processed in the ventral stream (Peuskins, et. al. 2005). Then, if a specific action is to be processed, the information is passed further into the dorsal stream through parietal regions to inferior frontal regions and pre-motor areas associated with the MNS (Saygin et. al. 2004, Downing et. al. 2006).

Recently however, Jastorff and Orban (2009) have posed, and attempted to address, three main issues with the literature to date. The first is that the STSp, although implicated by some as being responsible for the processing of articulated body movements as described above, has also been implicated in social perception and the perception of intentional actions (Castelli, Happé & Frith, 2000; Grézes, Frith & Passingham, 2004; Pelphrey, Morris & McArthy (2004); Schultz, Imamizu, Kawato & Frith, 2004, Saxe, 2004). The second issue is that noted earlier, that areas that are supposed to be selective for form information such as the EBA, actually overlap with the motion sensitive area MT+ (Peelen, et. al. 2006). The final issue raised is that it is still unknown which cues are used to extract biological motion from point light displays. The two most prominent models are those of Giese and Poggio (2003) and Lange and Lappe (2006). Giese and Poggio (2003) propose that biological motion is based on the integration of motion and shape cues that are processed separately in the dorsal and ventral streams respectively, whereas the Lange and Lappe (2006) model proposes that the integration of static shape cues over time can produce the coherent percept of biological motion. To address these issues, the authors implemented 3 experiments in order to separate the processing of human form from the processing of biological kinematics, to separate the effect of action complexity from task demands, and to investigate the role of the congruence of the movement of the points belonging to a single limb and local opponent motion. The results showed a preference for form information in the ventral stream, extending from the lateral occipital sulcus to the posterior inferior temporal gyrus (ITG) and to the fusiform gyrus. Dorsal regions, including the STS and inferior temporal sulcus (ITS), showed a strong preference for the kinematics of the actions. There were also interactions between the dorsal and ventral areas. The EBA and FBA of the ventral stream activated regardless of stimulus

complexity or task instructions, whereas posterior superior temporal regions were active only during complex stimuli, such as boxing or aerobics, or when a one-back task was used instead of passive viewing. Frontal regions, comprising the posterior IFG, IFS and the ventral Pre-central sulcus, were also only selectively activated for biological motion when participants were required to perform a task. Jastorff & Orban (2000) argue that activation of the STSp is selective for animate or biological motion only when the stimuli are either behaviourally relevant, or when the movements are more complex and require more in-depth processing. Furthermore they argue that it is dorsal areas, such as the EBA and FBA, that are the first step in automatic processing of biological motion and that signal articulated human action to higher areas.

However, it is still poorly understood how the brain can utilise and perceive a coherent moving human form in amongst noise, and how the brain would match the salient configural information across time. It has been shown, using direction discrimination of scrambled point-light walkers, that participants can still gauge the direction of locomotion correctly around 80% of the time (Troje & Westhoff, 2006), suggesting that the local motions of the individual points are sufficient. It has also been shown however, that the configural information contained in a small number of correctly placed points on a walker, can substantially outweigh the influence of a large number of scrambled points (Chapter 3 & McKay, Simmons, McAleer & Pollick, 2009), suggesting that configural information is more informative than local motion alone. However, how the brain can utilise the configural cues in amongst noise is still unknown. It seems unlikely that the starting point for biological motion processing would be static images, as in the presence of noise the form of the point-light actor would be imperceptible in each individual frame. It is more likely that there is some type of template matching across successive frames (Lange and Lappe, 2006) that would utilise form areas together with global motion processing, or that there is a global matching of dynamic representations (Beauchamp et. al. 2003) perhaps using areas such as the ITS and STSp.

In the current chapter we aimed to investigate this issue using the technique described in Chapter 3 to identify individualised performance thresholds, which will give a measure of sensitivity to configural information for each participant. Experiment 1 was the same experiment as developed in Chapter 3 and was used to derive the performance thresholds required for Experiment 3. Experiment 2 utilized a well established fMRI design that

compared intact, scrambled and static PLDs in order to determine the regions sensitive to processing both configural cues from biological motion and also local motion cues. The regions derived from this experiment were used as a mask to restrict analysis in Experiment 3. Experiment 3 tested participants in three conditions, each using stimulus levels determined from each participant's individual results. Each participant viewed stimuli at which they performed at 50%, 75% and 95% correct in the behavioural experiment. This allowed us to compare performance across the group when the local motion cues were as influential as the configural cues (50% correct), when the configural cues were dominant but local cues still influenced the percept of the coherent walker (75% correct) and when the configural cues were sufficient to always perceive a coherent human walker (95%). In this way, it was hoped that the regions responsible for integrating local motions into a coherent form would be elucidated. Furthermore, using Granger Causality Mapping (GCM) to examine the directional influences to and from each of the regions derived from Experiment 3, it was hoped that the patterns of effective connectivity would help clarify the roles of the dorsal and ventral stream and any connections that exist between the two.

## 4.2 Methods

### 4.2.1 Participants

Participants were 12 typically developed males all aged between 18 and 29 years old (Mean age 22.3, SD 3.5) with normal or corrected to normal vision. Participants were paid £12 for participation and were fully debriefed at the end of the fMRI session.

### 4.2.2 Design

*Experiment 1:* The task took the form of a behavioral two alternative forced-choice direction discrimination paradigm with 2 conditions: configural cues present and configural cues absent. The participants' task was to say whether the point light walker was moving from left to right or right to left. There were eight stimulus levels in each condition, which were the number of signal points shown (1, 3, 5, 7, 9, 11, 13, and 15). The number of noise points always equaled 15 minus the number of signal points, ensuring

that all stimuli comprised a total of 15 points. There were 20 trials per stimulus level in both conditions giving a total of 320 trials per person.

*Experiment 2:* Experiment 2 was a blocked fMRI task similar to that used by Saygin et. al. (2004), in which 12-second blocks of intact biological motion, scrambled biological motion and static frames from a point-light actor were presented in a 1-back counterbalanced order. There were six repetitions of each stimulus condition with an ISI of 2 seconds between each block corresponding to 1 TR. There were 8 seconds of fixation at the start and end of the run, giving a total run length of 278 seconds, which equates to 139 volumes. Three separate runs were used with different stimulus ordering, and all were 1-back counterbalanced and contained the same number of stimulus presentations of each stimulus type.

*Experiment 3:* Experiment 3 was an event-related fMRI design using the same task as in Experiment 1. Participants were tested at 3 stimulus levels that were derived from the configural cues present condition in Experiment 1 and corresponded to their individual 95%, 75% and 50% (chance) correct thresholds. This ensured that all participants were tested at their own specific performance thresholds. A rapid event-related design was used in order to minimise stimulus predictability and adaptation effects to the stimulus levels. Participants were asked to perform the same task as they did in the behavioural experiment, that is, to determine the direction of locomotion of the 15 dot point light walkers. Participants saw 18 repetitions of each stimulus level per run. Each stimulus presentation was 1 second (1TR) long, followed by an ISI of 3 seconds (3 TRs) in order to give participants enough time to make a decision and push the button.

Stimulus ordering was optimised using the Genetic Algorithm(GA) developed by Wager and Nichols (2003), with the highest weight being given to optimising contrast detection, and reduced weight being given to HRF estimation and counterbalancing. In addition, using the same algorithm, the designs for each of the 3 runs were optimised for the contrasts of 95% correct > 75% correct, 75% correct > 50% correct and 95% correct > 50% in order to maximise detection for these specific contrasts. As shown by Wager and Nichols and subsequently by Kao, Mandal, Lazar & Stufken (2009), the efficiency of designs optimised using the GA far outweigh those of randomised, fully counterbalanced and m-sequence designs for detecting differences in signal between event types, and is far less laborious than searching the space containing all possible permutations of a design for

ones that maximise efficiency. Temporal jitter was added to the design by having the GA position fixation trials in a way that further enhanced the efficiency of the design. As such, there were 18 trials for each of the 3 stimulus levels plus 14 jitter trials, giving a total of 68 trials of 4 seconds long giving a run length of 272s.

### 4.2.3 Stimuli

*Experiment 1:* Stimuli were presented on a CRT monitor with a resolution of 1280 X 1024 pixels, on a display size subtending 20.4° of visual arc by 15.36° with a refresh rate of 60 Hz at approximately 1m from the participant. Point-light stimuli were generated from an existing motion capture library (Ma, et. al. 2006) and displayed and manipulated within the experiment using the Psychophysics Toolbox for MATLAB (Brainard, 1997; Pelli, 1997). The translational motion of the walker was subtracted from the displays so that the walker appeared as if it was walking on a treadmill. Each stimulus was presented for 60 frames (1 second) and showed a full gait cycle. The point-light walkers were scaled to a height of 200 pixels with the resulting figure subtending a visual angle of approximately 3.02° by 1.32° and consisting of white dots on a black background. The point-light walkers were randomly spatially jittered horizontally and vertically within a window subtending a visual angle of 5.43° by 3.70°. The starting frames of the displays were at the points of minimal distance between the wrists and ankles. This was done since experiments without large additional noise masks show that this point in a display contains the least amount of configural information, making it more difficult to discriminate scrambled displays from intact displays (Thirkettle, et. al. 2009). This was important in this study in order to minimize any perceptual difference between the conditions other than those of experimental interest.

In both the configural cues present and configural cues absent conditions, the method for creating and manipulating the signal and noise points was identical to that used by McKay et. al. (2009), and fully described in Chapter 3, and will not be repeated here.

*Experiment 2:* The stimuli were generated from real motion capture data from the library of Ma et. al. (2006). All displays consisted of centrally presented point-light displays of 15 white dots on a black background. Each moving display was generated by repeating a 1-second (1 gait cycle) point light walker. As in previous experiments, the horizontal translational movement was removed by holding the head point constant in the horizontal

axis, thereby creating the impression of walking on a treadmill, ensuring that the figure remained in the centre of the screen, whilst preserving the relationship between the head and other body points. To ensure there were no selective effects of locomotion direction each block was split into two, with the figure walking towards the left in one half of the block and to the right in the second half of the block.

Scrambled displays were generated by randomly scrambling the points of the walker to other joint locations such that no point maintained its original location. Each block was split into two halves in which the direction of motion was reversed halfway through the block. This was done to eliminate any direction-selective effects of the local motion signals of each point.

Static displays were generated by taking a random frame from the middle of a scrambled display, giving a display that had neither structural nor local motion information.

Each type of stimulus was converted into a 256 x 256 pixel .avi file using the psychophysics toolbox for Matlab. Each point-light figure was normalised to a height of approximately 171 pixels presented centrally within the movie. These movies were presented on Nordic Neurolabs Visual System goggles with a field of view of 30° x 22.5°. The point-light walkers subtended a visual angle of approximately 6.41° x 2.805°. Stimulus presentation and response collection was controlled using the software package Presentation from Neurobehavioural Systems Inc.

*Experiment 3:* Point-light files were generated using the same technique as in the configural cues present condition from Experiment 1, which is more fully described in chapter 3 and by McKay et. al. (2009). In order to cover the full possible range of signal points that may be required to match to participants' thresholds, point-light files were generated for each possible level of signal points i.e. 1-15. The point-light displays were converted into AVI format movies with a resolution of 256 x 256 pixels, so the walkers subtended approximately equivalent visual angles to the displays in Experiment 2. As in Experiment 2, these movie files were played using Presentation software and presented through Nordic Neurolabs Visualsystem goggles. The stimuli shown to each participant had a number of signal points that corresponded to their individual 95%, 75% and 50% correct thresholds derived from the configural cues present condition of the behavioural experiment.

### 4.2.4 fMRI Acquisition parameters

*Structural:* A Siemens 3T Tim Trio MRI scanner was used to acquire sagittal T1 weighted anatomical images and T2 weighted functional images. The anatomical scans were T1 weighted MPRAGE sequences with a TR of 1900ms, a TE of 2.52, a TI of 900ms with a flip angle of 9°. We collected 192 slices with an isovoxel resolution of 1mm x 1mm x 1mm and dimensions 256 x 256 with a FOV of 256 and a run time of 8 minutes and 8 seconds.

*Experiment 2:* Functional T2 weighted images were acquired with a TR of 2000ms, a TE of 30ms and a flip angle of 77°. We collected 35 slices for each of 139 volumes at a resolution of 2.5mm x 2.5mm x 3mm slice thickness and dimensions 84 x 84 per image with Integrated Parallel Acquisition Techniques (IPAT) and online motion correction giving a run time of 278 seconds. The data sets used were the motion corrected (moco) series output by the Siemens system.

*Experiment 3:* Functional T2 weighted images were acquired with a TR of 1000ms, a TE of 30ms and a flip angle of 62°. We collected 18 slices for each of 272 volumes at a resolution of 3mm x 3mm x 4.5mm slice thickness and dimensions 70 x 70 per image with IPAT and online motion correction giving a run time of 272 seconds. The data sets used were the motion corrected (moco) series output by the Siemens system.

### 4.2.5 Data Pre-Processing

*Experiment 2:* Brainvoyager QX 1.10 was used for processing all stages of the data. Structural scans were homogeneity corrected and transformed into talairach space using BVQX 1.10. Functional runs were slice scan time corrected, motion corrected and temporally filtered at 2 cycles across the run. The functional runs were aligned to the anatomical scans, transformed into talairach space and converted into 4D volumes. A Gaussian 5mm spatial filter was applied to the 4D volumes in order to improve the signal to noise ratio for group analysis.

*Experiment 3:* Functional runs were slice scan time corrected, motion corrected and temporally filtered at 5 cycles across the run. Higher frequency temporal filtering was possible in Experiment 3 due to the increased frequency of stimulus presentations, higher

TR and longer duration when compared to Experiment 2. The functional runs were aligned to the anatomical scans, transformed into talairach space and converted into 4D volumes. A Gaussian 5mm spatial filter was applied to the 4D volumes in order to improve the signal-to-noise ratio for group analysis.

### 4.2.6 Procedure

*Experiment 1:* Each participant was instructed to view point-light walkers on the screen. Upon the completion of each stimulus presentation the participant was required to decide whether the walker on the screen was moving from left to right or from right to left, using the arrow keys on the keyboard. A correct answer in the configural cues present condition was considered to be when the participant chose the direction of motion of the points with preserved trajectories and joint locations. In the configural cues absent condition a correct answer was operationally defined as when the participant chose the direction of motion of those points that had preserved their original trajectories, in essence the direction of locomotion of the original point light display. This preserved a scale of proportion correct, allowing a direct comparison of performance in both conditions on a scale of 0 to 1, which would not have been possible with other definitions of "correct" such as the direction with the most overall energy.

Each participant completed a set of practice trials displaying several levels of stimuli from both conditions. Once a participant had completed these and were comfortable in the task, the main experimental blocks commenced. In both conditions, each of the eight stimulus levels were presented once in each of the twenty blocks, giving 160 trials per condition. Which condition each participant saw first was counterbalanced to remove any possible order effects. The experiment took just under 30 minutes to complete.

*Experiment 2 & 3:* Participants underwent the scanner safety checklist to ensure there were no contra-indications that would make it unsafe for them to be scanned. Before entering the scanner each participant was shown the tasks that they would be performing to ensure that they were familiar with the task (in experiment 3 it was the same as the behavioural task) and to minimize the time they would be required to spend inside the scanner. All safety aspects of the scanning were explained to the participants and they were assured that at any point they could use the emergency buzzer to stop the experiment. Once this had been explained, participants were taken to the scanner and made

comfortable. The button box was shown to them as were the buttons pertaining to each task and they were given the emergency button. Vision was corrected using the Nordic Neurolabs Visualsystem goggles until participants were able to clearly see stimuli and instructions. Once participants were comfortable and confident they understood the instructions, they were moved into the scanner bore and all further communication took place via an fMRI safe intercom system. The instructions were repeated and participants comfort and desire to continue was checked before each scan.

## 4.3 Results

### 4.3.1 Experiment 1: Quantification of the contribution of configural cues

For each participant the proportion of correct responses was calculated for each level of stimulus in both conditions, and the data were fitted with a cumulative Gaussian using the psignifit toolbox for MATLAB (Wichmann & Hill, 2001a, 2001b). For each individual the 50%, 75% and 95% thresholds and the mean slope of the curve were extracted for the configural cues present and the configural cues absent condition separately. Figure 4.1 shows the mean slope for each condition. A repeated measures t-test showed that the mean slope in the configural cues present condition was significantly higher than the mean slope in the configural cues absent condition $t(11) = 7.892$, $p < 0.001$.

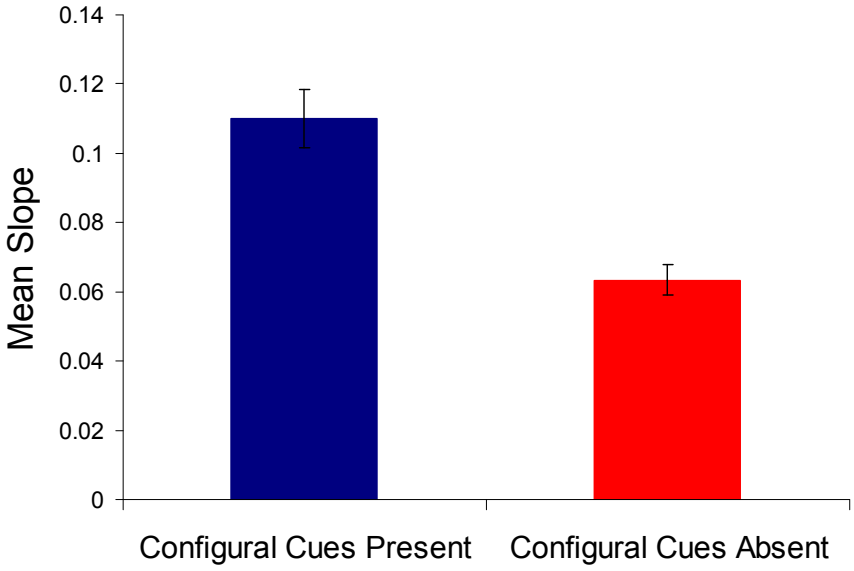


Figure 4.1: Mean Slope of psychometric curves in the configural cues present and configural cues absent conditions. Error bars represent Standard Error.

A repeated measures ANOVA was used to examine the main effect of Condition, Threshold and the Condition *x* Threshold interaction. The ANOVA revealed that there was a main effect of Condition ($F(1,11) = 142.351$, $p < 0.001$), a main effect of Thresholds ($F(2,22) = 167.173$, $p < 0.001$) and also a significant interaction effect between Condition and Threshold ($F(2,22) = 37.189$, $p < 0.001$). Figure 4.2 shows the mean number of signal points required to reach each of the three thresholds.

Post-hoc Tukey tests confirmed that the mean 50%, 75% and 95% thresholds differed significantly from each other within each condition and that across conditions, the 50%, 75% and 95% thresholds also differed significantly from each other. Table 4.1 shows the results of the post-hoc tests.

Table 4.1: Results of the post-hoc Tukey tests comparing each mean in the configural cues present condition (CP) and configural cues absent condition (CA)

| | CP 50% | CP 75% | CP 95% | CA 50% | CA 75% | CA 95% | |
|---|---|---|---|---|---|---|---|
| Mean | 4.62 | 7.21 | 10.93 | 7.25 | 11.81 | 18.37 | |
| 4.62 | 0 | 8.4** | 20.48** | 8.54** | 8.25** | 44.67** | q |
| 7.21 | | 0 | 12.08** | 0.14 | 6.39** | 36.28** | q |
| 10.93 | | | 0 | 11.94** | 0.24 | 24.20** | q |
| 7.25 | | | | 0 | 7.54** | 36.14** | q |
| 11.81 | | | | | 0 | 44.68** | q |

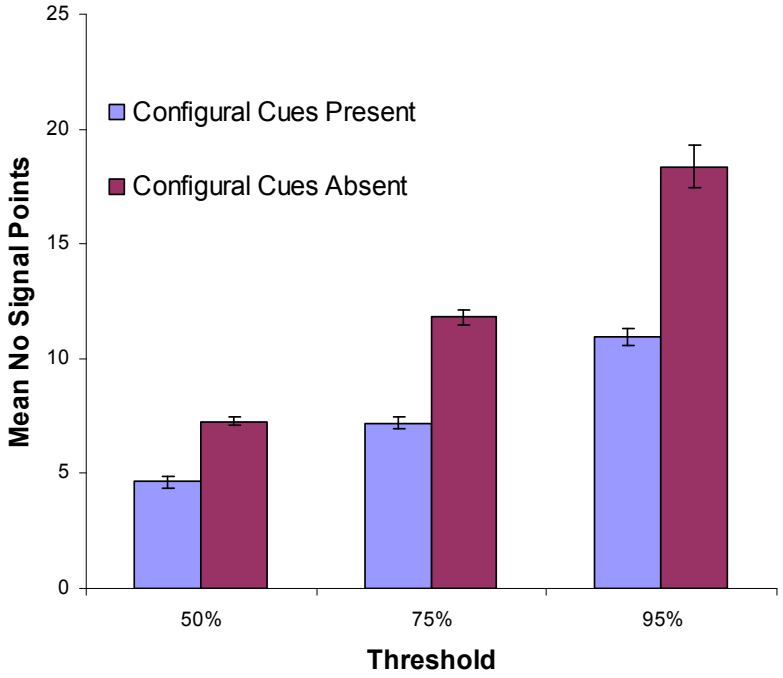


Figure 4.2: Mean number of signal points required to meet the 50%, 75% and 95% correct thresholds. Error bars represent the Standard Error.

The results show that the number of signal points required to perform at the 50%, 75% and 95% correct thresholds is lower in the configural cues present condition than in the configural cues absent condition. The interaction effect is driven by the differences between the number of signal points required to meet each of the thresholds being lower in the configural cues present condition than in the configural cues absent condition, reflecting the increased slope in the configural cues present condition. These results support the findings reported in Chapter 3, that configural information greatly improved the ability of participants to determine the direction of locomotion of a point-light walker when compared with stimuli in which no configural information is present.

## 4.3.2 Experiment 2: Investigation of the brain regions responsible for processing biological motion

Experiment 2 set out to determine which brain areas respond selectively to intact PLDs containing configural information and scrambled PLDs that did not contain configural information. A whole-brain fixed-effects GLM was calculated and the following contrasts were specified: intact point-light stimuli minus scrambled point-light stimuli and scrambled point-light stimuli minus static point-light displays. The results of each contrast are presented individually in the following sections.

### 4.3.2.1 Scrambled Point-Light Displays – Static Point-Light Displays

Volume maps were generated for the contrast scrambled point-light displays - static point-light displays. These were then thresholded with an FDR value of $q < 0.05$. Regions were defined based on these volume maps and a minimum cluster size of 5 voxels ($3mm^3$) was set, giving a minimum volume for a cluster of $45mm^3$. Where regions covered excessively large areas, such as those around the middle temporal and occipital regions, effort was made to ensure that the regions conformed to anatomical boundaries. Table 4.2 lists the talairach coordinates of the peak voxel for each region and the spatial extent in terms of the number of voxels ($1mm^3$) in each region.

We found a large number of regions that were significantly more active for scrambled point-light displays than for static-point-light displays. These include regions with peak voxels bilaterally in the STSp, ITG(MT+), the Pre-central Gyrus, the Cuneus and the Insula. Unilateral activations were found in the left post-central gyrus, IPL, fusiform

gyrus, inferior occipital gyrus, the medial frontal gyrus and the right middle frontal gyrus, middle occipital gyrus, and the parahippocampal gyrus.

We also found a number of regions that showed significantly higher activation for static point-light displays than for scrambled point-light displays. These include bilateral regions with peak voxels in the middle frontal gyrus, Cuneus and Culmen. Regions were found unilaterally with peak voxels in the anterior portion of the left STS, cingulate gyrus and lingual gyrus, and in the right STG, MTG and the parahippocampal gyrus.

Table 4.2: Peak Talairach coordinates, t-values, p-values and number of voxels for each region defined from the contrast of Scrambled Biological motion minus stationary PLDs

**Brain areas that prefer scrambled biological motion to static frames**

| | | x | y | Z | T | p < | No. Voxels |
|---|---|---|---|---|---|---|---|
| Superior Temporal Sulcus | L | -48 | -37 | 4 | 6.01 | 0.000001 | 1050 |
| | R | 45 | -37 | 7 | 8.40 | 0.000001 | 2620 |
| Cuneus | L | -21 | -82 | 22 | 9.08 | 0.000001 | 2694 |
| | R | 24 | -76 | 22 | 9.12 | 0.000001 | 2694 |
| Insula | L | -42 | -34 | 19 | 7.88 | 0.000001 | 1654 |
| | R | 51 | -31 | 19 | 9.79 | 0.000001 | 3433 |
| Inferior Temporal Gyrus | L | -45 | -70 | -2 | 21.52 | 0.000001 | 10509 |
| | R | 45 | -64 | -2 | 23.97 | 0.000001 | 10968 |
| Precentral Gyrus | L | -42 | -10 | 46 | 6.07 | 0.000001 | 4553 |
| | R | 36 | -10 | 52 | 3.62 | 0.000300 | 269 |
| Postcentral Gyrus | L | -48 | -28 | 40 | 4.15 | 0.000040 | 692 |
| Inferior Parietal Lobule | L | -33 | -49 | 43 | 6.85 | 0.000001 | 9912 |
| Fusiform Gyrus | L | -39 | -37 | -14 | 5.31 | 0.000001 | 289 |
| Inferior Occipital Gyrus | L | -36 | -80 | -2 | 7.46 | 0.000001 | 3236 |
| Medial Frontal Gyrus | L | -6 | 11 | 46 | 4.58 | 0.000006 | 365 |
| Middle Frontal Gyrus | R | 42 | -1 | 53 | 4.36 | 0.000020 | 467 |
| Middle Occipital Gyrus | R | 24 | -88 | 4 | 9.59 | 0.000001 | 5783 |
| Parahippocampal Gyrus | R | 39 | -34 | -11 | 4.60 | 0.000005 | 261 |

**Brain areas that prefer static frames to scrambled biological motion**

| | | x | y | Z | T | p < | No. Voxels |
|---|---|---|---|---|---|---|---|
| Superior Temporal Gyrus | L | -51 | -13 | 1 | -4.67 | 0.000004 | 1050 |
| | L | -51 | 8 | -14 | -4.08 | 0.000050 | 575 |
| Middle Frontal Gyrus | L | -30 | 29 | 37 | -3.87 | 0.000200 | 266 |
| | R | 18 | 5 | 52 | -3.85 | 0.000200 | 320 |
| Cuneus | L | -15 | -70 | 13 | -4.50 | 0.000008 | 2736 |
| | R | 3 | -85 | 13 | -9.70 | 0.000001 | 10020 |
| Culmen (Anterior Lobe) | L | -24 | -43 | -14 | -5.50 | 0.000001 | 2632 |
| (Cerebellum) | R | 6 | -70 | -5 | -8.47 | 0.000001 | 2473 |
| Superior Temporal Gyrus | R | 57 | -16 | 7 | -4.74 | 0.000002 | 2428 |
| Middle Temporal Gyrus | R | 39 | -67 | 25 | -3.48 | 0.000600 | 220 |
| Cingulate Gyrus | L | 0 | -52 | 28 | -3.84 | 0.000200 | 812 |
| Lingual Gyrus | L | -6 | -85 | -2 | -6.80 | 0.000001 | 2099 |
| Parahippocampal Gyrus | R | 24 | -40 | -7 | -5.53 | 0.000001 | 4577 |

### 4.3.2.2 Intact Point-Light Displays -Scrambled Point-Light Displays

The same criterion was applied to the volume map for the contrast of intact point-light displays to scrambled point light displays. Table 4.3 lists the Talairach coordinates of the peak voxel for each region and the spatial extent in terms of the number of voxels ($1mm^3$) in each region.

A number of regions were found that were significantly more active during displays of intact biological motion than scrambled biological motion. These regions have peak voxels in the middle frontal gyrus, IPL, fusiform gyrus, SOG and the cuneus of the right hemisphere and the SFG, cingulate gyrus, IOG, precuneus and the Supramarginal gyrus of the left hemisphere. Regions that were more active for scrambled point-light displays than intact point-light displays had peak voxels bilaterally in the lingual gyrus and insula and unilaterally in the left SPL, MOG, pre-central gyrus and cuneus, and in the right IPL, MTG and precuneus.

Table 4.3: Peak Talairach coordinates, t-values, p-values and number of voxels for each region defined from the contrast of intact biological motion minus scrambled biological motion.

**Brain areas that prefer intact biological motion to scrambled biological motion**

| | | X | Y | Z | T | p < | No. Voxels |
|---|---|---|---|---|---|---|---|
| Middle Frontal Gyrus | R | 8 | 30 | 29 | 4.80 | 0.000003 | 1338 |
| | R | 9 | 27 | 44 | 4.11 | 0.000050 | 352 |
| Superior Frontal Gyrus | L | -30 | 20 | 52 | 4.06 | 0.000050 | 137 |
| Cuneus | R | 18 | 6 | -88 | 4.98 | 0.000002 | 888 |
| Inferior Parietal Lobule | R | 40 | 48 | -43 | 4.27 | 0.000030 | 413 |
| Fusiform Gyrus | R | 37 | 33 | -46 | 4.79 | 0.000003 | 246 |
| Cingulate Gyrus | L | -9 | -37 | 34 | 4.68 | 0.000004 | 325 |
| Inferior Occipital Gyrus | L | -30 | -92 | -11 | 3.91 | 0.000100 | 241 |
| Precuneus | L | 7 | 0 | -61 | 5.15 | 0.000001 | 2096 |
| Superior Occipital Gyrus | R | 19 | 40 | -73 | 4.09 | 0.000050 | 269 |
| Supramarginal Gyrus | L | 40 | -57 | -52 | 3.76 | 0.000200 | 132 |
| | | | | | | | 6437 |

**Brain areas that prefer scrambled biological motion to intact biological motion**

| | | X | Y | Z | T | p < | No. Voxels |
|---|---|---|---|---|---|---|---|
| Insula | L | -45 | -31 | 19 | -5.14 | 0.000001 | 649 |
| | R | 48 | -31 | 22 | -5.00 | 0.000002 | 758 |
| Lingual Gyrus | L | -15 | -91 | 1 | -4.19 | 0.000030 | 151 |
| | R | 15 | -82 | -11 | -7.10 | 0.000001 | 1251 |
| Superior Pariental Lobule | L | 7 | -33 | -49 | -5.03 | 0.000002 | 1259 |
| Inferior Parietal Lobule | R | 33 | -37 | 49 | -5.17 | 0.000001 | 1708 |
| Middle Temporal Gyrus | R | 45 | -61 | 1 | -8.52 | 0.000001 | 3602 |
| Precentral gyrus | L | -42 | -10 | 46 | -4.37 | 0.000020 | 297 |
| Cuneus | L | 18 | -21 | -82 | -5.20 | 0.000001 | 1547 |
| Middle Occipital Gyrus | L | -39 | -67 | -2 | -7.12 | 0.000001 | 2572 |
| Precuneus | R | 24 | -79 | 25 | -5.01 | 0.000002 | 350 |

### 4.3.3 Experiment 3: Investigating brain regions selective for configural processing

In Experiment 3 we tested participants at 3 stimulus levels, which were the 50%, 75% and 95% correct thresholds derived from the psychometric curves obtained for each individual in Experiment 1. We anticipated that each participant would be performing at the same level inside the scanner as they did in Experiment 1. Figure 4.3 shows actual scanner performance levels of the group for each of the thresholds tested. It can be seen that the participants are performing very close to the thresholds derived from Experiment 1. To confirm this proportions correct were calculated for each stimulus level and each participant from the scanner task. One-sample t-tests were calculated for each stimulus level comparing the actual percent correct inside the scanner to their corresponding threshold values. These confirmed that there were no significant differences between the actual percentages correctly identified in the scanner, and the thresholds that the stimulus level corresponded to (50% - $t(11) = .433$, $p = 0.67$; 75% - $t(11) = 0.287$, $p = 0.78$; 95% - $t(11) = -1.163$, $p = 0.272$).

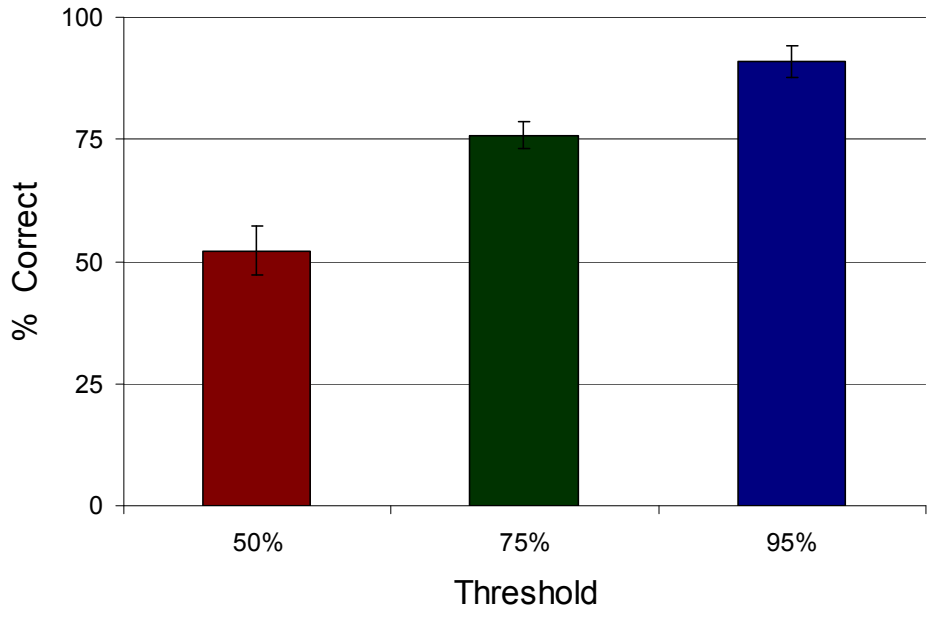


Figure 4.3: Mean proportion correct from task inside scanner by expected proportion correct based on Experiment 1 results. Error bars represent Standard Error.

A random-effect (RFX) GLM was carried out on the group data. As RFX GLMs tend to require large numbers of participants, we used a mask generated from the regions derived from Experiment 2 to restrict the analysis to only those regions we knew responded

differentially to either intact or scrambled biological motion. This reduced the number of voxels entered into the GLM to around 14% of the number of voxels entered into an unconstrained whole brain analysis, with a view to boosting power sufficiently to run the RFX GLM and be able to apply the results to the wider population. Figure 4.4 shows the mask used illustrated in a glass brain.

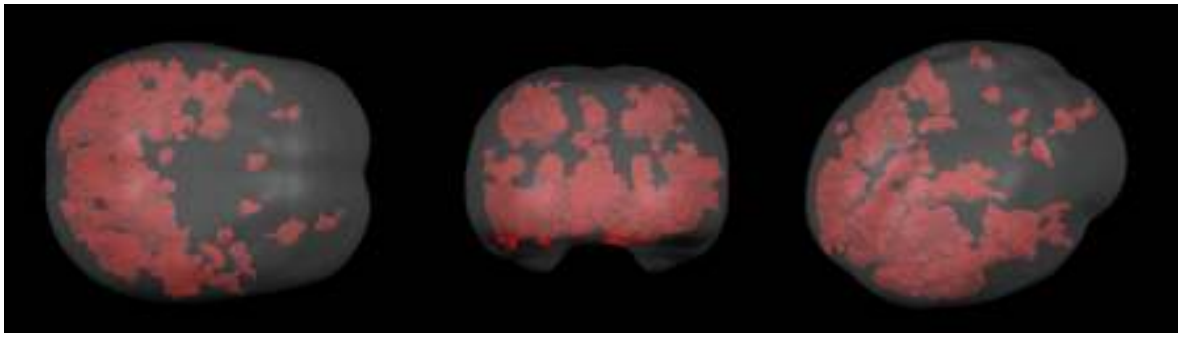

Figure 4.4: Mask generated from the regions found to be preferentially active either to intact over scrambled PLDs or scrambled PLDs over static frames in Experiment 2.

Volume maps were thresholded at $p < 0.01$ and cluster size threshold estimation (Worsley, Evans, Marrett & Neelin, 1992) was used to determine minimum cluster sizes for each contrast, based on a significance of $p<0.05$. Regions were defined based on those that survived cluster-size threshold estimation, and where regions covered excessively large areas effort was made to ensure regions conformed to anatomical boundaries.

The design had been optimised using the Wager and Nichols genetic algorithm for the contrasts 95>75, 95>50 and 75 > 50. Each contrast will be discussed in the following sections.

#### 4.3.3.1 95% correct - 75% correct

Table 4.4 shows the list of Talairach coordinates for the peak voxels of the regions derived from this contrast.

Table 4.4: Peak Talairach coordinates, t-values, p-values and number of voxels for each region derived from the contrast of 95% correct stimuli – 75% correct stimuli.

Brain areas that preferred 95% correct stimuli over 75% correct stimuli

| | | X | Y | Z | T | p > | No. Voxels |
|---|---|---|---|---|---|---|---|
| Superior Parietal Lobule | R | 22 | -64 | 43 | 3.52 | 0.005 | 611 |
| Lingual Gyrus | L | -10 | -76 | 7 | 4.14 | 0.002 | 688 |
| Posterior Cingulate | L | -18 | -58 | 4 | 5.32 | 0.0003 | 445 |
| Lingual Gyrus | L | -21 | -78 | 1 | 4.18 | 0.002 | 743 |
| Posterior Cingulate | L | -24 | -66 | 16 | 3.67 | 0.004 | 1138 |
| Middle Occipital Gyrus | L | -36 | -70 | 7 | 5.95 | 0.0001 | 830 |

We found regions that were preferentially active for stimuli presented at individuals' 95% correct threshold over the 75% correct thresholds in the right IPL and the left MOG, lingual gyrus and the posterior cingulate. There were no regions found that were preferentially active for 75% correct stimuli.

#### 4.3.3.2 75% Correct - 50% Correct

Only 1 region survived cluster-size threshold estimation and this was found to have its peak voxel in the post-central gyrus of the right hemisphere (Talairach coordinates x = 24 y = -40 z =39, t = 3.71, p<0.004, 487 voxels).

#### 4.3.3.3 95% correct - 50% correct

Table 4.5 shows the list of Talairach coordinates for the peak voxels of the regions derived from this contrast. We found regions that were more active for stimuli presented at individuals' 95% correct threshold than at 50% correct thresholds in the left SPL and ITG, and in the right pre-central gyrus and the cuneus. As in the previous contrasts no regions were found that were selectively active for 50% correct stimuli over 95% correct stimuli.

Table 4.5 Peak Talairach coordinates, t-values, p-values and number of voxels for each region derived from the contrast of 95% correct stimuli – 50% correct stimuli.

| Brain areas that preferred 95% correct stimuli to 50% correct stimuli | | X | Y | Y | T | p > | No. Voxels |
|---|---|---|---|---|---|---|---|
| Precentral Gyrus | R | 33 | -27 | 46 | 3.37 | 0.007 | 514 |
| Cuneus | R | 21 | -79 | 25 | 3.99 | 0.003 | 430 |
| Superior Parietal Lobule | L | -27 | -61 | 52 | 5.26 | 0.0003 | 1237 |
| Inferior Temporal Gyrus | L | -48 | -73 | 1 | 3.89 | 0.003 | 508 |

#### 4.3.3.4 Granger Causality Mapping

Each of the regions from the 3 aforementioned contrasts that showed greater activation to stimuli with increased amounts of configural information, were used as seeds for Granger Causality Mapping(GCM). From each of these seeds the GCM returns two volume maps. The first shows regions that correlate instantaneously with the activity in the seed region (iGCM). The second, more importantly, shows the directional influences of the seed on other regions and of other regions upon the seed (dGCM). Granger causality mapping ideally required seed regions of no more than 300 voxels in order to avoid spurious correlations with regions not truly correlated with the one of interest. To meet this

requirement, for any region of more than 300 voxels, we took the 299 voxels within the region that had the smallest Euclidian distance from the peak voxel. This typically gave spherical regions for larger clusters, but for smaller clusters that were perhaps narrower in one direction than others, ensured that the resulting regions were constrained by the contours of the original region. Figure 4.5 shows the seed regions derived from each of the 3 contrasts carried out in Experiment 3 after size adjustments for GCM analysis in a glass brain.

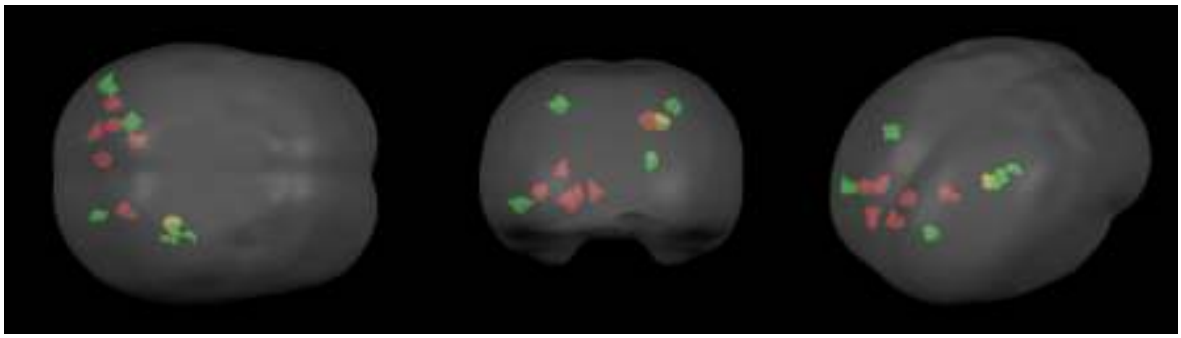

Figure 4.5: Regions derived from each of the 3 contrasts carried out in Experiment 3 separated by colour. In red the seed regions derived from the contrast of 95% correct stimuli - 75% correct stimuli, in yellow the seed regions derived from the contrast of 75% correct stimuli - 50% stimuli and in green the seed regions derived from the contrast of 95% correct stimuli - 50% correct stimuli.

A further constraint of GCM analysis is that it requires a stimulus event to be displayed, or repeated, over the course of at least 20 volumes/time-points. As Experiment 3 utilised a rapid event-related design with events of different stimulus types interleaved with each other rather than a blocked design, it was not possible to look at individual conditions using GCM. As such, we ran the GCM analysis over the whole time course.

The GCM analysis was run separately for each participant using the time course from each of the three runs. This produced a dGCM volume map for each individual for each of the seed regions. These were thresholded with a FDR of $q < 0.05$. These maps were combined and a t-test was carried out across the whole brain to identify voxels that were significantly different from zero.

Maps were generated from the t-tests where positive t-values represented areas that were influenced by the seed region, and negative values represented regions that influences the seed region. Table 4.6 summarises the results of the dGCM for each seed region, showing the peak talairach coordinates, t-values, p-values and the size of the regions as number of voxels($1mm^3$). Any seed region that did not show significant direction influence was omitted from the table. Figure 4.6 illustrates the patterns of directional effective

connectivity for the seed regions derived from each of the Experiment 3 contrasts separately.

Table 4.6: Peak talairach coordinates, t-values, p-values and number of voxels for regions derived from GCM analysis for each seed region from Experiment 3.

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas influenced by activity in the left Superior Parietal Lobule** | | | | | | | |
| Precuneus | L | -9 | -76 | 40 | 2.94 | 0.013528 | 31 |
| Superior Parietal Lobule | L | -24 | -58 | 58 | 3.00 | 0.012173 | 60 |
| Superior Parietal Lobule | R | 15 | -67 | 52 | 2.66 | 0.022299 | 129 |
| Precuneus | R | 6 | -61 | 55 | 2.66 | 0.022235 | 62 |
| **Brain areas influenced by activity in the right Superior Parietal Lobule** | | | | | | | |
| Precuneus | L | -12 | -79 | 43 | 3.15 | 0.009192 | 189 |
| Cuneus | L | -12 | -85 | 34 | 3.17 | 0.008943 | 75 |
| Precuneus | L | -12 | -58 | 43 | 4.46 | 0.000966 | 389 |
| Precuneus | R | 21 | -61 | 43 | 6.10 | 0.000078 | 3993 |
| **Brain areas that influence activity in the right Superior Parietal Lobule** | | | | | | | |
| Cuneus | L | -15 | -76 | 28 | -2.45 | 0.032084 | 31 |
| **Brain areas influenced by activity in the right Precentral Gyrus** | | | | | | | |
| Medial Frontal Gyrus | L | 0 | -16 | 49 | 3.02 | 0.015 | 251 |
| Pre-central Gyrus | L | -27 | -13 | 55 | 3.30 | 0.0075 | 142 |
| Inferior Parietal Lobule | L | -39 | -40 | 46 | 3.25 | 0.008 | 1096 |
| Post-Central Gyrus | L | -36 | -22 | 43 | 2.83 | 0.02 | 69 |
| Post-Central Gyrus | R | 27 | -34 | 49 | 6.46 | 0.00005 | 2648 |
| Precuneus | R | 15 | -46 | 49 | 3.47 | 0.0055 | 678 |
| **Brain areas influenced by activity in the left Posterior Cingulate** | | | | | | | |
| Precuneus | L | 0 | -52 | 46 | 2.72 | 0.02 | 162 |
| Posterior Cingulate | L | -18 | -61 | 7 | 4.32 | 0.0015 | 1512 |
| Lingual Gyrus | R | 12 | -64 | -2 | 2.95 | 0.015 | 128 |
| Posterior Cingulate | R | 9 | -55 | 7 | 4.52 | 0.0009 | 381 |
| **Brain areas influenced by activity in the right Post-Central Gyrus** | | | | | | | |
| Cingulate Gyrus | R | 24 | -43 | 37 | 3.02 | 0.015 | 47 |
| **Brain areas influenced by activity in the left Middle Occipital Gyrus** | | | | | | | |
| Middle Occipital Gyrus | L | -33 | -67 | 10 | 5.25 | 0.0003 | 932 |
| **Brain areas that influence activity in the left Middle Occipital Gyrus** | | | | | | | |
| Cuneus | R | 3 | -73 | 7 | -3.05 | 0.015 | 161 |
| Cuneus | R | 3 | -70 | 13 | -3.10 | 0.015 | 159 |
| **Brain areas influenced by activity in the left Lingual Gyrus** | | | | | | | |
| LingualGyrus | L | -6 | -76 | 7 | 5.73 | 0.00015 | 2482 |
| Cuneus | R | 27 | -76 | 7 | 3.18 | 0.009 | 115 |
| Cuneus | R | 12 | -73 | 10 | 3.47 | 0.0055 | 168 |
| Lingual Gyrus | R | 12 | -64 | 1 | 3.10 | 0.015 | 67 |
| Cuneus | R | 9 | -88 | 13 | 3.43 | 0.006 | 43 |
| Precuneus | R | 3 | -58 | 43 | 2.62 | 0.025 | 38 |
| **Brain areas influenced by activity in the left Inferior Temporal Gyrus** | | | | | | | |
| Inferior Temporal Gyrus | L | -48 | -76 | -2 | 4.16 | 0.002 | 988 |
| **Brain areas that influence activity in the left Inferior Temporal Gyrus** | | | | | | | |
| Middle Occipital Gyrus | L | -39 | -82 | 7 | -2.81 | 0.02 | 74 |
| **Brain areas influenced by activity in the right Cuneus** | | | | | | | |
| Cuneus | R | 21 | -73 | 19 | 3.14 | 0.009 | 520 |
| Cuneus | R | 21 | -79 | 25 | 4.93 | 0.0004 | 574 |
| Cuneus | R | 9 | -85 | 34 | 2.80 | 0.02 | 108 |

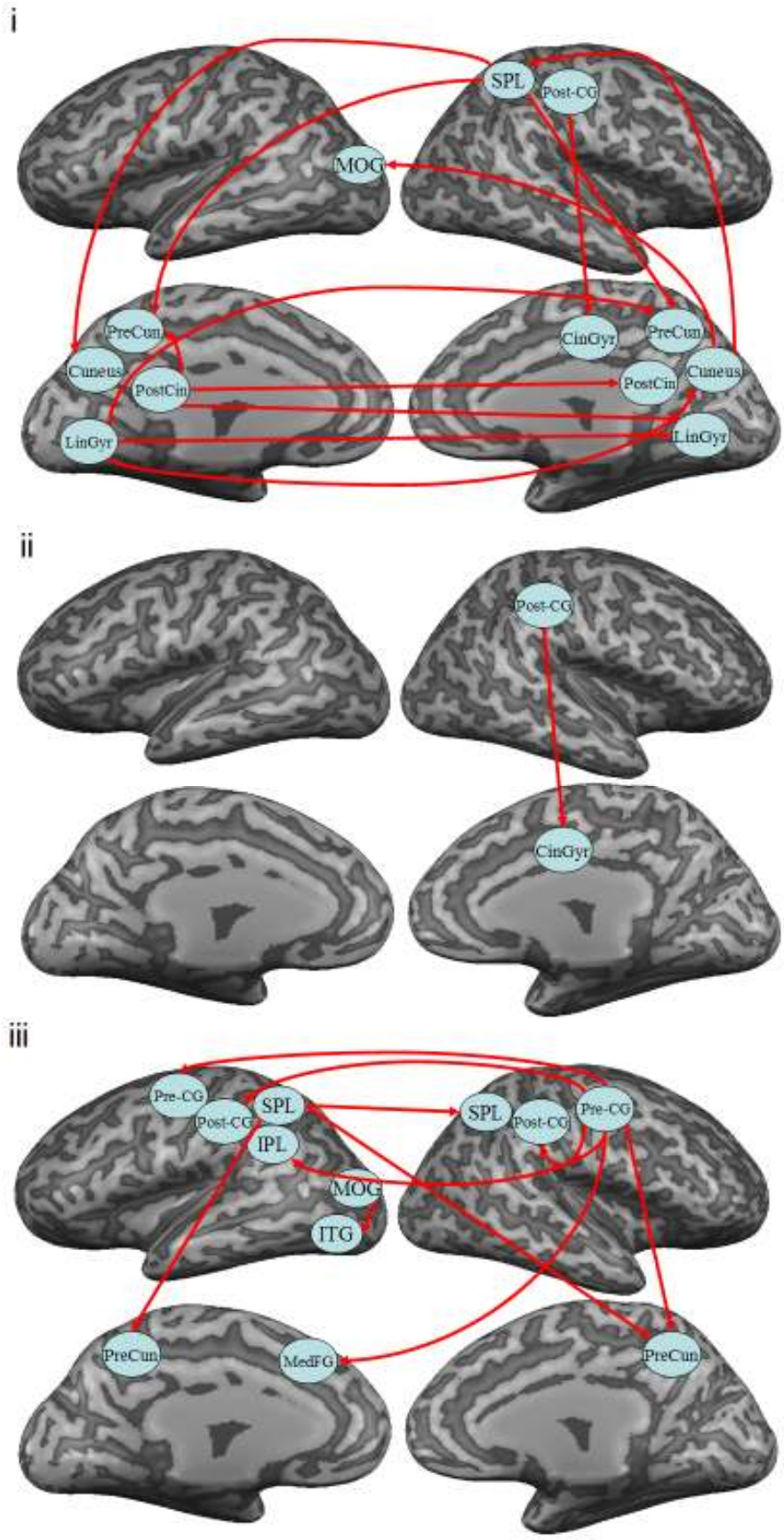


Figure 4.6: Patterns of directional connectivity from the seed regions defined from the contrasts. i. 95% correct-75% correct, ii. 75% correct-50% correct, iii. 95% correct-50% correct.

As can be seen from Table 4.6 and Figure 4.6, the pattern of directional influence is quite complex. The results suggest that for the seed regions derived from the contrast of 95% correct stimuli minus the 50% correct stimuli, the medial regions, the cuneus, precuneus, posterior cingulate, cingulate gyrus and lingual gyrus tend to have directional influence to a large number of other medial areas. The lingual gyrus and posterior cingulate of the left hemisphere seems to have the most widespread influence of these medial areas, in that the lingual gyrus influences the right lingual gyrus, cuneus and precuneus and the posterior cingulate influences the left precuneus and the right lingual gyrus and posterior cingulate. The lingual gyrus of the left hemisphere has no directional influence over other regions. Of these medial structures, the cuneus alone seems to influence more lateral regions of the cortex, showing directional influence over the left MOG and the right SPL. Influence from the lateral cortical areas to the medial areas seem to be mostly through the right SPL, which influences activity in the left precuneus and bilateral regions in the precuneus. The only other directional influence from the lateral cortex is from the right Post-Central Gyrus (abbreviated Post-CG in figure 4.6) to the Cingulate Gyrus.

For the seed region derived from the contrast 75% correct stimuli-50% correct stimuli, the only influence the right Post-Central Gyrus has is over the right cingulate gyrus. From the seed regions derived from the contrast 95% correct-50% correct there is a more widespread directional influence between lateral cortical areas than from the seed regions derived from the 95% correct-75% correct contrast. The right Pre-Central Gyrus (abbreviated Post-CG in Figure 4.6) influences the Post-Central Gyrus bilaterally and the left SPL and IPL, as well as medial regions of the right Precuneus and left Medial Frontal Gyrus (abbreviated MedFG in figure 4.6). The left SPL influences the right SPL and the precuneus bilaterally. Lastly, there is also directional influence of the left MOG over the left ITG.

## 4.4 Discussion

The experiments in this chapter set out to elucidate the nature of networks involved in processing configural information from PLDs of biological motion. Experiment 1 confirmed the results of Chapter 3, that configural cues greatly improve direction discrimination of point-light walkers. Experiment 2 used a standard paradigm to determine the regions involved in processing intact, scrambled and static PLDs and Experiment 3 examined how each of the regions derived from Experiment 2 responded to different levels

of configural information in PLDs and used GCM to examine the effective connectivity between these regions.

In Experiment 2 we contrasted intact PLDs of human walkers with scrambled PLDs, and scrambled PLDs with static frames of structureless dots. In the first contrast we found regions that were preferentially active for intact PLDs in the right IPL, fusiform gyrus, middle frontal gyrus (MFG), superior occipital gyrus (SOG) and the cuneus, and in the left SFG, cingulate gyrus, IOG, precuneus and the supramarginal gyrus. We also found regions that were more responsive to scrambled PLDs than static frames in the bilateral lingual gyrus and insula, the left SPL, MOG, pre-central gyrus and cuneus and the right IPL, MTG and precuneus.

Consistent with previous research, we found that a region in the fusiform gyrus slightly posterior in relation to the reported location of the FBA was more responsive to intact PLDs than scrambled PLDs. Interestingly, in addition to the FBA activity, we found that a region in the inferior portion of the right IPL also showed increased activation to the intact over scrambled stimuli. This is in line with the findings of Downing et. al. (2006), who found preferential activation of this inferior parietal regions for coherent, continuous displays of actions, which is close to the occipito-temporal junction and has been reported as showing a similar preference for intact biological motion over scrambled displays (Sinha & Belliveau, 2001). This region is thought to contain neurons with mirror properties (Fogassi, Gallese, Fadiga & Rizzolatti, 1998; Gallese, Fogassi, Fadiga & Rizzolatti, 2002) and has been implicated in the imitation of observed actions (Jeannerod, 1994) and action understanding (Rizzolatti et, al. 2001). Recently, the supramarginal gyrus has also been demonstrated to be selectively active to meaningful actions (Newman-Norlund, van Schie, van Hoek, Cuijpers, Bekkering, 2009). Why these parietal regions are activated in the present study is unclear given their proposed role in processing goal directed actions, though it may be that directional locomotion is perceived as a meaningful action. The regions in the occipital cortex, namely the cuneus, precuneus, and the inferior occipital gyrus, found in both contrasts more likely pertain to low-level visual processes, given the occipital lobes role in visual processing, including form and motion processing, and are likely to be non-biological motion selective, though the IOG is part of the extrastriate cortex and its proximity to the ITG may mean that it has some role in form processing.

In contrast to the IOG, the MTG was found to be more activated for scrambled PLDs motion than intact PLDs, suggesting that, in contrast to Peelen et. al. (2006) and Downing et. al. (2006), the MT+ region is more sensitive to local motion cues and may be less active during viewing of intact PLDs. Moreover, there were a number of frontal and parietal regions that were also more responsive to scrambled biological motion. Some of these are in line with previous research, such as the pre-central gyrus and dorsal regions of the parietal cortex (Beauchamp et. al. 2003; Grézes et. al. 2001) and it may be that the scrambled PLDs are on some level still being processed as belonging to a human.

Some of the regions that would be expected, based on previous research, to show a preference for intact over scrambled PLDs were actually found to be selective for scrambled biological motion over static structureless frames. These include the bilateral ITG, STSp and the left fusiform gyrus. The activity of the STSp is partly unexpected, given that the stimuli are of simple walking motions (Jastorff and Orban, 2009), though it has been reported before for the same contrast, though with complex actions (Saygin et. al. 2004). Given the reported involvement of the ITG and fusiform areas in form processing, particularly the EBA and FBA, it is unusual that these should be more activated for scrambled point light displays, though as mentioned, the fusiform activation was in the left hemisphere which has not been reported as showing the form selectivity found in the right FBA (Peelen et. al. 2007; Downing et. al. 2006; Jastorff & Orban, 2009). The peak voxels of the bilateral ITG regions were consistent with those reported by Peelen et. al. (2006), Downing et. al. (2006) and Jastorff and Orban (2009). Furthermore, regions of the somatosensory cortex and precentral gyrus also showed increased activity for scrambled PLDs over static frames.

This poses an interesting question; why do the ITG and fusiform regions seem more selective for biological motion, regardless of the structural information present in the displays? One reason may be the scrambling technique used. In previous studies using scrambled PLDs, typically the points are randomly positioned within the walker space (e.g. Jastorff & Orban, 2009; Saygin et. al. 2004; Vaina et. al. 2001; Grossman et. al. 2000). This means that the dispersion of the points can be wider than the original PLD and is independent of the original joints of the walker, creating a more diffuse field of motion. In the present experiment, the scrambled displays were generated by reallocating each point of the walker to a new joint location. Furthermore, the initial frame of the display was at the point in the motion in which the distance between the two hands and the distance

between the two feet was shortest, minimising static configural cues in the first frame. However, it may be that by constraining the motions of the points to be centred on the joints, even if the motions were not correct for their position, that the regions found may have tried to integrate the incoherent motions into a global percept. The activations seen in these areas could represent increased effort in an attempt to recover some semblance of coherence.

Alternatively, the ITG may be picking up on certain static frames within the stimuli in which a plausible human form is generated. These occurrences would be more common in the stimuli used in the present study, given that the motions are tied to the joint locations. This seems plausible in light of the findings of Downing et. al. (2006) that the part of the ITG known as the EBA is more sensitive to static postures, and that when viewing continuous displays of biological motion the activity in these regions decrease. They propose that the EBA is not specifically involved in biological motion processing, but only in the recognition of biologically plausible static postures. This information may then be passed to other regions, such as the Fusiform Body Area for temporal matching of postures or the STS for integration with local motion cues. Both these possibilities seem plausible, given that the STS is more responsive to the scrambled stimuli than static stimuli and the right FBA is not differentially activated by scrambled and static PLDs. As the fusiform gyrus is preferentially active for biologically plausible continuous actions, which is also in line with the findings of Dowling et. al. (2006), then perhaps this region matches these static postures across time, only responding if the subsequent postures are congruent with biologically plausible models. Furthermore, the fusiform gyrus has functional connections to parietal regions (e.g. Bokde, Lopez-Bayo, Meindl, Pechler, Born, Faltraco, Teipel, Möller & Hampel, 2006), which may explain why the STS, unlike parietal regions, did not respond differentially to intact and scrambled PLDs, as it may not receive input from fusiform areas. If the STS integrates local motions with static posture information from the EBA, then it would not differentiate between the coherent and scrambled biological motion, whereas the FBA may relay information about the temporally contingent postures directly to parietal or even pre-motor regions.

The results from Experiment 3 tentatively support the proposed roles of the ITG in form processing and the lack of discriminability of the STSp to varying degrees of configural information. In Experiment 3 we tested participants across 3 stimulus levels that corresponded to participants' individual 50%, 75% and 95% correct thresholds from a

direction discrimination task. Behavioural results from inside the scanner confirmed that participants were performing at the same levels during the fMRI task. The analysis was restricted using the regions from both contrasts from Experiment 2, confining the RFX-GLM to only those regions that responded to either configural cues or local biological motion signals. We found regions that were more responsive to increases in configural information, and hence corresponded with behavioural increases in accuracy, in the SPL, the ITG, the MOG and the pre-and post-central gyrus, as well as more medial areas such as the posterior cingulate, lingual gyrus and the cuneus. The activation of premotor regions is consistent with the findings of Jastorff and Orban who found that these regions respond to biological motion stimuli only if they are sufficiently complex or require the performance of a task. The ITG region defined in the contrast of 95% correct minus 75% correct, overlapped with the MOG region defined in the 95% correct minus 50% correct contrast and were only dissociable based on their peak voxels. Given that in both these contrasts there was also SPL activity, though it was unilateral in the right and left respectively, and that these were the only areas common to both contrasts, it seems that these may be the key regions used in processing configural information. As the ITG and MOG were both selectively activated by stimuli containing increased structural information, it seems plausible that the task is achieved by matching static postures from within the continuous displays. As there is noise present in all the stimuli, then subsequent frames in the displays would not present truly plausible human actions, which is potentially why the FBA was not active in any of the contrasts in Experiment 3. As such, the EBA may have been used to detect biologically plausible static postures across the frames of the stimuli, and its increased activity to stimuli containing more configural information could be a result of it processing a larger number of biologically plausible static postures. These could then be passed to the STS which, as noted earlier, may not selectively activate to the structural cues, but integrate this information with local motion cues before passing it to parietal or frontal regions. Here it important to note that the STS, as would be predicted, did not differentially activate to displays of varying degrees of configural information. It may be that the information processed by the FBA, EBA and STS is combined in the parietal regions. This is supported by findings that patients with parietal lobe lesions, whose general form-from-motion abilities were spared, showed deficits in identifying actions in point light displays and detection of coherent walkers in noise (Battelli, Cavanagh & Thornton, 2003).

The results of the GCM analysis do not help to clarify the roles of these regions. They suggest that the left ITG is actually influenced by activity from the left MOG. The left SPL seems to influence the right SPL and bilateral regions of the precuneus, but it does not seem to be influenced by any other region, which seems unlikely. Furthermore, activity in the right SPL appear to be influenced by activity in the right cuneus. These findings, taken together with numerous other directional influences between medial visual areas, such as the posterior cingulate, lingual gyrus, cuneus and precuneus, further complicate this matter. One strong pattern that does come out of the GCM analysis is that the direction of influence seems to predominantly go from the left hemisphere to the right hemisphere, particularly in more medial areas.

One reason for the complicated pattern of results is that the present study conducted GCM analysis across the entire time series, and did not differentiate between stimuli in different conditions. The reason for this is that there are a number of restrictions that are placed on designs in order to run GCM within conditions that reduce design efficiency for contrast detection. As the present study was comparing three stimulus levels, contrast detection efficiency was prioritised over GCM power. This meant that the GCM analysis would have picked up on directional effective connectivity only between regions that were permanently exchanging information across the whole time series, or alternatively picking up on directional connectivity between some regions at some time points and different regions at other points. GCM analysis does not allow for the separation of these different possibilities when analysis is performed across a whole time series. Another limitation of GCM in general, is that it can only discriminate effective connectivity, which means that it can only determine patterns of influence between regions and not direct functional or anatomical connectivity. As an illustration, the GCM analysis may show that region A influences region B. However there may be a region C mediating the influence that GCM analysis cannot detect.

Another limiting factor, for both the GLM-RFX analysis and the GCM analysis is that the design used was a rapid event-related design with 3 conditions and jitter trials tightly packed into short runs. This resulted in a relatively small number of stimulus repetitions, only 18 per stimulus level. It was hoped that this would be offset across the 3 runs, but the results may have been more robust had more signal been included. The problem could have been more severe however, had a randomised or counterbalanced design been implemented rather than the GA optimised design (see methods).

Despite the limitations of the current study, we found evidence to suggest that the processing of biological motion seems to recruit multiple pathways for different tasks. It seems that when the actions depicted in PLDS are continuous and unambiguous the FBA seems to process the information, potentially using a temporal analysis of static form cues from successive frames (Lange and Lappe, 2006). When the successive frames do not present biologically plausible continuous actions, the EBA seems to identify biologically plausible static form cues that are not necessarily contiguous and conceivably could be from different actions (Downing et. al. 2006). These are then potentially integrated with local motion cues, perhaps in the STSp, before being passed on to higher regions in the parietal or frontal cortices (Giese & Poggio, 2003; Downing et. al. 2006). The findings are in line with those of Peelen et. al. (2006) who proposed that the ventral processing stream, the EBA and FBA in particular, process static form cues and with those of Jastorff and Orban (2009) who propose that these regions form the starting point for the initial stages of processing of biological motion.

In the next chapter the contribution of configural information and the neural pathways involved in processing biological motion will be assessed in typically developed people and a group of high functioning adults with ASDs. The experiments used will address the limitations of the current study, in an effort to determine which brain regions process configural information from PLDs and how these regions communicate when participants can and cannot correctly gauge the direction of locomotion of a point-light walker. The main aim however, is to determine whether the brains of people with ASDs process biological motion in a similar way to typically developed people and if not, to define what these differences are.

# Chapter 5: Identifying differences in neural processing of configural processing from PLDs in people with ASDs and typically developed controls

## 5.1 Introduction

In the previous chapters, it has been shown that in tasks involving the detection of biological motion, and the categorisation of actions and affect from PLDs of human motion, people with ASDs did not differ from well-matched typically developed controls. However, there were some indications that the ASD group were slightly poorer than the control groups, and that performance was more variable within this population. Furthermore, it seemed that the ASD group employed slightly different strategies to differentiate between PLDs depicting different affects. In Chapter 3 we devised a novel technique for quantifying the contribution of configural information to a direction discrimination task that maintains a constant level of local motion, and in Chapter 4 used this technique to examine the neural correlates of configural processing in a group of typically developed adult males. The current chapter will bring together these elements to determine whether configural cues are as salient to people with ASDs as typically developed controls, and whether the underlying neural mechanisms for processing this information are the same for both populations.

The status of biological motion processing in ASDs is a matter of much debate. Initial studies suggested that people with ASDs had intact biological motion processing mechanisms and were only impaired at attributing affect to PLDs (Moore et. al. 1997). Subsequent studies suggested that in addition to difficulties in attributing affect to actions, people with ASDs showed reduced abilities in integrating local motion signals into a global percept of a human (Blake et. al. 2003), and that young children with autism do not preferentially attend to canonical displays of human motion (Klin et. al. 2003).

In recent years a number of studies have revisited the topic, providing equally mixed results. Klin and Jones (2008) and Klin et. al. (2009) have found that children with ASDs as young as 15 months show no preference for canonical human movement, and Kaiser, Fermano & Shiffrar (2008) have shown that adults with ASDs, unlike typically developed controls, do not show an increased sensitivity for human motion over object motion.

However, there are also a number of studies that have replicated the findings of Moore et. al. (1997) that there were no significant differences between people with ASDs and controls in tasks involving action perception, but that the ASD group were significantly worse than controls on affect recognition (Hubert, et. al. 2007; Parron et. al. 2008)

The two most recent papers on the topic have produced conflicting reports on the matter. Atkinson (2009) found that the ASD group tested were impaired relative to controls in recognition of happy and angry affects, but not fear or sadness. However, as performance on this task was correlated with motion coherence thresholds, Atkinson (2009) attributes this not to a top-down processing impairment, but to dysfunctional integration of low level motion cues into a global coherent percept. He argues that action categorisation can be achieved using low level motion cues, whereas affect recognition requires more global processing and that impaired global processing of overall motion signals can explain the pattern of deficits seen in people with ASDs in terms of biological motion processing. In contrast, Murphy et. al. (2009), using an experiment similar in methods to that used in Task 1 chapter 1 of this thesis, found no significant differences in detection of biological motion in noise between people with ASDs and controls. Murphy et. al. (2009) concluded that people with ASDs do not have difficulty in integrating local visual information into a global percept of the human form. It is worth noting however, that Murphy et. al. (2009) did find that the ASD group showed poorer performance in the task, though the difference was not significant. This is a common feature of those studies that have not shown significant differences between the two populations. Moore et. al. (1997), Hubert et. al. (2007) and Parron et. al. (2008), like Murphy et. al. (2009) all reported slightly, but not significantly, poorer performance in biological motion detection and action categorisation tasks.

Murphy et. al. (2009) acknowledge this and suggest that a non-specific motion integration problem may have resulted in the slightly poorer performance of the ASD group, though they also suggest that although not explicitly controlled, the ASD group may have been of slightly lower intellectual ability than the control group, as indicated by lower scores on the Ravens's Standard Progressive Matrices (Raven, Raven & Court, 1998), and that this may have caused the differences. In an attempt to explain the discrepancy between their own findings and those of Blake et. al. (2003) and other studies showing impaired biological motion processing, Murphy et. al. (2009) highlight age as a potential factor. In the original Blake et. al. (2009) study the participants were aged between 8 and 10 years

old, whereas in the Murphy et. al. study participants had a mean age of 25.6 years old. They suggest that perhaps the children used in the Blake et. al. (2003) study were perhaps developmentally delayed in processing biological motion, and that by adulthood people with ASDs have reached typical levels. A developmental delay is supported by the findings of Annaz et. al. (2009) that children with ASD show a flat developmental trajectory in biological motion processing between the ages of 5 and 12, and that by age 12 the ASD group are substantially poorer at discriminating intact from scrambled PLDs. Furthermore, an examination of the literature of the ages of participants used reveals a distinction. Those studies that have found no difference in biological motion processing have all tested participant groups that have extended into adulthood (Moore et. al. 1997 – 11-19 yrs old; Hubert et. al. 2007 – 15-34 yrs old; Parron et. al. 2008 – 7-18 yrs old; Murphy et. al. 2009 – mean CA 25.56 yrs old SD = 7.67 yrs), with only Kaisers et. al. (2008) finding a significant difference between a group of adults with ASDs and a mean age of 20 years old and a CA matched control group. Those studies that have found significant differences, with the exception of Kaiser et. al. (2008), have all tested children with ages far younger that those mentioned above (Blake et. al. 2003 – 8-10 yrs old; Klin et. al. 2003 – mean CA 2 years old; Klin & Jones, 2008 – CA 15 months old; Klin et. al. 2009 – mean CA 2 yrs old). Further support for this comes from the non-significant differences between people with ASDs and controls in biological motion detection and action categorisation from Tasks 1 & 2 of Chapter 2 of this thesis.

If it is the case that people with ASDs do show a developmental delay in processing biological motion, then there may be differences in underlying neural processing that are still evident in adulthood. Two recent studies have found some evidence for this. Freitag et. al. (2008) found that, despite comparable behavioural performances, adult participants with ASDs showed markedly different patterns of brain activation than controls when viewing intact and scrambled PLDs of human walking motions. Most notably, they found that the control group utilised the expected brain regions when processing the intact stimuli. These included the MTG, the STSp, the fusiform gyrus, the IPL, the intraparietal sulcus, the post-central gyrus and the superior frontal gyrus. The ASD group on the other hand showed a markedly sparse activation of regions, and no activation in the STSp, fusiform gyrus, IPL or intraparietal sulcus, which are all regions that have been implicated in biological motion processing (Howard et. al. 1996; Grossman et. al. 2000; Vaina et. al. 2001; Beauchamp et. al. 2003; Saygin et. al. 2004; Grézes et a. 2001; Grafton, Fagg Woods & Arbib, 1996; Puce, Truett, Bentin, Gore & McCarthy, 1998). Freitag et. al.

(2008) claim that there are two possible explanations of their finding; the first being that people with ASDs have difficulty in higher-order motion processing and the second being that people with ASDs have difficulties in integrating complex motion information in the associative cortex. Herrington et. al. (2007) used a direction discrimination task to investigate processing of biological motion in people with ASDs. Participants were asked to say whether they thought PLDs of intact or scrambled PLDs were moving to the left or the right whilst in an fMRI scanner. Like Freitag et. al. (2008), they found no significant differences in behavioural performance between the two groups, but significantly different patterns of neural activation to the intact PLDs versus fixation. In addition to those regions found to be activated differently in people with ASDs by Freitag et. al. (2008), such as the STS, inferior parietal regions, the precentral gyrus and the fusiform gyrus, Herrington et. al. (2007) also found that the ITG, MOG and the angular gyrus were less activated to these stimuli in the ASD group. They also reported that a region, extending from the STG to the angular gyrus was preferentially active for scrambled PLDs over fixation in the control group, but not in the ASD group. The finding that superior temporal regions were the only common activation in the 2 contrasts in controls, lends support to the notion that it is an integration-for-local-motions area, rather than a specific biological-motion-processing area (Beauchamp et. al. 2003; Peuskins et. al. 2005).

More interesting however, is that Freitag et. al. (2008) and Herrington (2007) found comparable behavioural performance between the two adult groups, which fits the aforementioned trend based on age, but that both studies reported different regions involved in the processing of intact PLDs. Of all the regions, the fusiform areas, the ITG and parietal regions are most likely the key points of interest. In particular the fusiform area and the ITG have been proposed as a potential starting point for automatic processing of biological motion (Jastorff and Orban, 2009). These regions seem to process static form cues, though in different ways. The fusiform area, often referred to as the fusiform body area (FBA) seems to process static frames and responds more if they are in a biologically plausible temporal order, whereas the ITG, which contains the extrastriate body area (EBA), is more responsive to static non-continuous displays of different body postures and exhibits adaptation if the series of postures form a continuous action (Downing et. al. 2006). It is thought that these regions process the initial static cues from displays of biological motion, and that the information may be passed separately to parietal or frontal regions either directly from the FBA, or from the EBA via intermediary regions, such as the STS, where information may be integrated with local motion signals. It may be that the

global percept of a human walker is not actually generated until the information is integrated in the associative cortex in parietal regions (Battelli et. al. 2003).

It is plausible then, that dysfunction in these regions at early stages of processing, may result in the pattern of reduced parietal and frontal activations found in people with ASDs in response to coherent biological motion. The question remains however, if these regions are dysfunctional in people with ASDs how, as adults, do they match typically developed controls in behavioural tasks involving processing of configural information from PLDs?

The present chapter aims to address this question directly. In order to determine whether behavioural performance is comparable between a group of adults with ASDs and controls, the direction discrimination task developed in Chapter 3 was used to quantify the effect that configural cues had in a direction discrimination task for each group. From this task, stimulus levels producing individual performance thresholds of 50% and 84% correct were derived and used to determine each individuals' stimulus levels in an fMRI experiment that addressed some of the shortcomings of the fMRI experiment in Chapter 4. Based on the previous literature, it was expected that performance thresholds in the behavioural task would be comparable for the two groups, but that we would find significant differences in the brain regions used to accomplish the task, including the fusiform gyrus, ITG, IPL and possibly the STSp. The revised design of the fMRI experiment in this chapter also allowed for GCM analysis to be conducted separately for each of the stimulus levels used, the 50% and 84% correct threshold. The aim was to elucidate the differences in the network that were specifically sensitive to configural information in each group.

## 5.2 Methods

### 5.2.1 Participants

The participants in the ASD group consisted of 10 adult males aged between 18 and 36 who had been diagnosed independently as having autism or aspergers syndrome. Further diagnosis in the form of the ADI-R ( Le Couteur, et. al., 1989; Lord, et. al., 1994) and the SRS (Constantino, et. al., 2003) was given to 3 of the 10 participants and confirmed the diagnosis of the independent clinicians. It was not possible to carry out the ADI-R or SRS for the remaining participants due to a lack of available developmental history. Each

participant also underwent the Weschler Abbreviated Scale of Intelligence (WASI) as an estimate of verbal, performance and full-scale IQ. There were originally 12 participants recruited for the study. However, 2 had to be excluded due to different symptomology and non-typical FSIQs.

Control participants were 10 age- and FSIQ-matched adults who were confirmed as not being on the Autism Spectrum as determined by assessment using the AQ with a cut-off of 18, which ensured all controls were either in the "average" or "below average" categories in terms of autistic traits measured by the AQ.

Table 5.1: Means and SDs of the age and FSIQ for each group.

| | AGE | | FSIQ | |
|---|---|---|---|---|
| **Group** | **MEAN** | **STDEV** | **MEAN** | **STDEV** |
| ASD | 28.60 | 6.92 | 125.00 | 7.01 |
| Control | 27.90 | 7.37 | 124.80 | 6.75 |

Paired samples t-tests confirmed that there were no significant difference between the two groups for either age ($t(9) = 0.89$, $p = .40$) or IQ ($t(9) = 0.20$, $p = .84$).

### 5.2.2 Design

*Experiment 1:* A similar behavioural experiment to that performed in Chapter 3 and 4 was used. This was a 2 alternative forced-choice direction discrimination paradigm with 2 conditions: configural cues present and configural cues absent. There were eight stimulus levels in each condition, which were the number of signal points shown (1, 3, 5, 7, 9, 11, 13, and 15). The number of noise points always equaled 15 minus the number of signal points, ensuring that all stimuli comprised a total of 15 points. There were 30 trials per stimulus level in both conditions giving a total of 480 trials per person.

*Experiment 2:* Experiment 2 was a blocked fMRI task similar to that used by Saygin et. al. (2004) in which 12-second blocks of intact biological motion, scrambled biological motion and static frames from a point-light actor were presented in a 1-back counterbalanced order. There were six repetitions of each stimulus condition with an ISI of 2 seconds between each block corresponding to 1 TR. There were 8 seconds of fixation at the start and end of the run giving a total run length of 278 seconds, which equates to 139 volumes.

Three separate runs were used with different stimulus ordering and all were 1-back counterbalanced and contained the same number of stimulus presentations of each stimulus type.

*Experiment 3:* Experiment 3 was an fMRI experiment using the same task as in Experiment 1. In order to maximise the detection power of the design only two stimulus levels were presented. These were derived from the configural cues present condition in Experiment 1, and corresponded to their individual 84% and 50% (chance) correct thresholds, instead of the 50%, 75% and 95% thresholds used in Chapter 4. The 84% correct threshold was chosen as, not only was it an intermediate level between the two above chance threshold levels in Chapter 4, but it is approximately equal to 1 standard deviation above the mean in two-alternative forced choice task, provided that the psychometric function is a cumulative Gaussian. As before, testing at threshold ensured that all participants were tested at their own specific performance thresholds. A rapid event-related design was used in order to minimise stimulus predictability. Participants were asked to perform the same task as they did in the behavioural experiment, that is, to determine the direction of locomotion of the 15-dot point-light walkers. Participants saw 45 repetitions at each stimulus level per run. In Chapter 3 it was found that 3s was more than ample time to respond, so instead of interleaving blank trials into the experiment, a variable ISI of between 2 and 4s was used in increments of the TR (1s).

In order to optimise the design of Experiment 3 for GCM, each stimulus type had to cover a large enough number of continuous volumes for the analysis to compare over. To accomplish this, stimulus lists were generated in which each stimulus level was repeated in pseudo-blocks of between 4 & 6 repetitions, with ISIs varying between 2 & 4 seconds (mean = 3 seconds), producing runs of a total duration of 360 seconds with 45 presentations of each stimulus type. Forty designs were generated and the efficiency of each design calculated. Of the 40 designs, the 3 with the highest efficiencies were chosen. To confirm that these designs were still more efficient than randomised or counterbalanced designs, 40 2-back counterbalanced designs were generated and their efficiencies were calculated. The 2-back counterbalanced designs were found to be only 56% as efficient as the mean efficiencies of those chosen for Experiment 3. This confirmed that our pseudo-block designs were still more efficient in terms of detecting differences between the two-stimulus types.

### 5.2.3 Stimuli

*Experiment 1:* Stimuli were presented on a CRT monitor with a resolution of 1280 X 1024 pixels, on a display size subtending 20.4° of visual arc by 15.36° with a refresh rate of 60 Hz at approximately 1m from the participant. Point-light stimuli were generated from an existing motion capture library (Ma et. al., 2006) and displayed and manipulated within the experiment using the Psychophysics Toolbox for MATLAB (Brainard, 1997; Pelli, 1997). The translational motion of the walker was subtracted from the walker motion so that the walker appeared as if it was walking on a treadmill. Each stimulus was presented for 60 frames (1 second) and showed a full gait cycle. The point-light walkers were scaled to a height of 200 pixels, with the resulting figure subtending a visual angle of approximately 3.02° by 1.32° and consisting of white dots on a black background. The point-light walkers were randomly jittered horizontally and vertically within a window subtending a visual angle of 5.43° by 3.70°. The starting frames of the displays were at the points of minimal distance between the wrist and ankles. This was done as in experiments without large additional noise masks this point in a display contains the least amount of configural information, making it more difficult to discriminate scrambled displays from intact displays (Thirkettle, Benton & Scott-Samuel, 2009). This was important in the current study in order to minimize any perceptual difference between the conditions other than those of experimental interest.

In both the configural cues present and configural cues absent conditions the method for creating and manipulating the signal and noise points was identical to those used by McKay et. al. (2009) and as described in Chapter 3, and therefore will not be repeated here.

*Experiment 2:* The stimuli were generated from real motion capture data from the library of Ma et. al. (2006). All displays consisted of centrally presented point-light displays of 15 white dots on a black background. Each moving display was generated by repeating a 1 second (1 gait cycle) point light walker. The coordinates of each display had been spatially smoothed by resampling the first ten and last ten frames, and re-calculating their coordinate positions using linear interpolation from the 49$^{th}$ frame to the 11th frame. This ensured that the start and end points of each gait cycle were congruent, creating the impression of a continuous walk with no gaps or impossible translations of limb movements. As in previous experiments the horizontal translational movement was removed by holding the head point constant in the horizontal axis, thereby creating the

impression of walking on a treadmill, ensuring that the figure remained in the centre of the screen whilst preserving the relationship between the head and other body points. To ensure there were no selective effect of locomotion direction each block was split into two, with the figure walking towards the left in one half of the block and to the right in the second half of the block.

Scrambled displays were generated by randomly scrambling the points of the walker to other joint locations such that no point maintained its original location. Each block was split into two halves in which the direction of motion was reversed halfway through the block. This was done to eliminate any direction-selective effects of the local motion signals of each point. Static displays were generated by taking a random frame from the middle of a scrambled display, giving a display that had neither structural nor local motion information.

Each type of stimulus was converted into a 250 x 250 pixel .avi file using the psychophysics toolbox for Matlab. Each point-light figure was normalised to a height of approximately 171 pixels presented centrally within the movie. These movies were presented on Nordic Neurolabs Visual System goggles with a field of view of 30° x 22.5° field of view. The point-light walkers subtended a visual angle of approximately 6.41° x 2.81°. Stimulus presentation and response collection was controlled using the software package Presentation from Neurobehavioural Systems Inc.

*Experiment 3:* Point-light files were generated using the same technique as in the configural cues present condition from Experiment 1, which is more fully described in chapter 3 and by McKay et. al. (2009). In order to cover the full possible range of signal points that may be required to match to participants thresholds, point-light files were generated for each possible level of signal points i.e. 1-15. The point-light displays were converted into AVI format movies with a resolution of 256 x 256 pixels and as such, the walkers subtended approximately equivalent visual angles to the displays in Experiment 2. As in Experiment 2, these movie files were played using Presentation software and presented through Nordic Neurolabs Visualsystem goggles. The stimuli shown to each participant had the equivalent number of signal points as those that corresponded to the number of signal points determined from the individuals' 84% and 50% correct thresholds, as derived from the configural cues present condition of the behavioural experiment.

### 5.2.4 fMRI Acquisition parameters

*Structural:* We used a Siemens 3T Tim Trio MRI scanner to acquire sagittal T1-weighted anatomical images. The anatomical scans were T1 weighted MPRAGE sequences with a TR of 1900ms, a TE of 2.52, and a TI of 900ms with a flip angle of 9°. We collected 192 slices with an isovoxel resolution of 1mm x 1mm x 1mm and dimensions 256 x 256 with a FOV of 256 and a run time of 8 minutes and 8 seconds.

*Experiment 2:* Functional T2-weighted images were acquired with a TR of 2000ms, a TE of 30ms and a flip angle of 77°. We collected 35 slices for each of 139 volumes at a resolution of 2.5mm x 2.5mm x 3mm slice thickness and dimensions 84 x 84 per image with IPAT and online motion correction giving a run time of 278 seconds. The data sets used were the motion corrected (moco) series output by the Siemens system.

*Experiment 3:* Functional T2 weighted images were acquired with a TR of 1000ms, a TE of 30ms and a flip angle of 62°. We collected 18 slices for each of 272 volumes at a resolution of 3mm x 3mm x 4.5mm slice thickness and dimensions 70 x 70 per image with IPAT and online motion correction giving a total run time of 272 seconds. The data sets used were the motion corrected (moco) series output by the Siemens system.

### 5.2.5 Data Pre-Processing

*Experiment 2:* Brainvoyager QX 1.10 was used for processing all stages of the data. Structural scans were homogeneity corrected and transformed into talairach space using BVQX 1.10. Functional runs were slice scan time corrected, motion corrected and temporally filtered at 2 cycles across the time-course. The functional runs were aligned to the anatomical scans, transformed into Talairach space and converted into 4D volumes. A Gaussian 5mm spatial filter was applied to the 4D volumes in order to improve the signal to noise ratio for group analysis.

*Experiment 3:* Functional runs were slice scan time corrected, motion corrected and temporally filtered at 2 cycles across the time-course. The functional runs were aligned to the anatomical scans, transformed into Talairach space and converted into 4D volumes. A Gaussian 5mm spatial filter was applied to the 4D volumes in order to improve the signal to noise ratio for group analysis.

### 5.2.6 Procedure

*Experiment 1:* Each participant was instructed to view point-light walkers on the screen. Upon the completion of each stimulus presentation, the participant was required to decide whether the walker on the screen was moving from left to right or from right to left using the arrow keys on the keyboard. A correct answer in the configural cues present condition was considered to be when the participant chose the direction of motion of the points with preserved trajectories and joint locations. In the configural cues absent condition a correct answer was operationally defined as when the participant chose the direction of motion of those points that had preserved their original trajectories, in essence the direction of locomotion of the original point-light display. This preserved a scale of proportion correct allowing a direct comparison of performance in both conditions on a scale of 0 to 1, which would not have been possible with other definitions of "correct" such as the direction with the most overall energy.

Each participant completed a set of practice trials displaying several levels of stimuli from both conditions. Once a participant had completed these and was comfortable in the task, the main experimental blocks commenced. In both conditions, each of the eight stimulus levels was presented once in each of the thirty blocks giving 240 trials per condition. Which condition each participant saw first was counterbalanced to remove any possible order effects. The experiment took just over 30 minutes to complete.

*Experiment 2 & 3:* Participants underwent the scanner safety checklist to ensure there were no contra-indications that would make it unsafe for them to be scanned. Participants were shown outside the scanner the task that they were going to be asked to perform until they were comfortable with what they were being asked to do. All safety aspects of the scanning process were explained, including the fact that at any point they could use the emergency scanner buzzer to stop the experiment. Once this had been explained, participants were taken to the scanner and made comfortable. The button box was shown to them, as were the buttons pertaining to each task, and they were given the emergency button. Vision was corrected using the Nordic Neurolabs Visualsystem goggles until participants were able to clearly see the stimuli and instructions. Once participants were comfortable and confident they understood the instructions, which were the same for Experiment 3 as for Experiment 1, they were slid into the scanner bore and all further

communication took place via an fMRI safe intercom system. The instructions were repeated and participants comfort and desire to continue was checked before each scan.

## 5.3 Results

### 5.3.1 Experiment 1: Quantifying the contribution of configural cues in biological motion processing in an ASD and control group

In Experiment 1 we set out to determine, using the same behavioural paradigm as used in Chapters 3 & 4, whether the contribution of structural information was as great for a group with ASDs compared with an age and IQ matched control group. As previously mentioned, the thresholds of interest in this task were the 50% and 84% correct thresholds. For each participant the proportion of correct responses was calculated for each of the 8 levels of stimuli in both conditions and the data were fitted with a cumulative Gaussian using the psignifit toolbox for MATLAB (Wichmann & Hill, 2001a, 2001b). From these we derived the 50% and 84% correct threshold for each individual. Figure 5.1 shows the mean number of signal points required to reach the 50% and 84% correct threshold for each condition and for both groups.

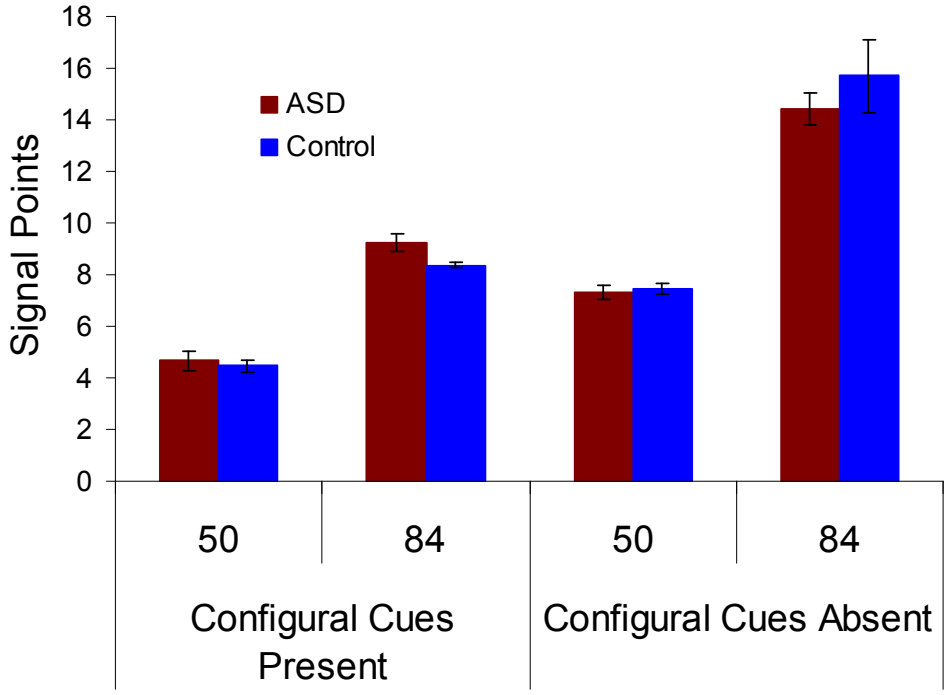


Figure 5.1: Mean number of signal points required to reach the 50% and 84% correct threshold in each condition for each group. Error bars represent the Standard Error

As can be seen from Figure 5.1, at the 50% correct thresholds both groups seem to perform comparably and both groups require more signal points to reach this threshold when configural information is absent from the stimuli. At the 84% correct threshold the two groups seem to show a slightly different pattern, with the control group requiring slightly fewer signal points than the ASD group to reach threshold when the stimuli contain configural information, whilst the ASD group seem to reach threshold with fewer signal points than the control group when the stimuli are devoid of configural information. Furthermore, the control group seems to be more variable in the number of signal points required to reach the 84% correct threshold in the absence of configural information. We tested the significance of these effects using the aforementioned planned comparisons and a 2 x 2 repeated measured ANOVA.

Four specific between-groups comparisons had been planned prior to the experiment. We used independent t-test to compare the 84% correct threshold of the two groups in each condition. The results found that, as hypothesized, in the configural cues presents condition the ASD group required significantly more signal points to reach 84% correct threshold than the control group $t(11.221) = 2.539$, $p = 0.027$ (adjusted for equality of variance assumption violation), though this did not survive a bonferroni correction for multiple comparisons ($p = 0.084$). The other prediction, that the ASD group would require fewer signal points in the configural cues absent condition, was not supported by the results $t(12.290) = -0.843$, $p = 0.159$l (adjusted equality of variance assumption violation). As predicted, the two groups did not differ significantly at the 50% correct threshold in either the configural cues present condition $t(18) = 0.493$, $p = 0.628$, or in the configural cues absent condition $t(18) = -0.316$, $p = 0.755$.

As the planned comparisons had addressed the differences between groups a 2 x 2 repeated measures ANOVA was carried out combining responses from the two groups to examine within group effects of "Condition" and "Threshold". This revealed a significant main effect of "Condition" $f(19) = 77.664$, $p < 0.001$ and "Threshold" $f(1,19) = 203.144$, $p < 0.001$, but also a significant interaction between the two $f(1,19) = 59.313$, $p < 0.001$. As can be seen from Table 5.1, post-hoc Tukey tests revealed that all means significantly differed from each other.

The results of the Tukey tests show that the number of signal points required for participants in both groups to reach the 50% and 84% correct thresholds in the configural

cues present condition was significantly lower than the numbers required to meet the same thresholds in the configural cues absent condition. These results support the findings reported in Chapters 3 and 4 that configural information greatly improved the ability of participants to determine the direction of locomotion of a point light walker, when compared with stimuli in which no configural information is present, and extends the finding to a group with ASDs.

Table 5.2: Results from post-hoc Tukey tests comparing all means from the configural cues present condition (CP) and the configural cues absent condition (CA).

| | CP 50% | CP 84% | CA 50% | CA 84% | |
|---|---|---|---|---|---|
| Mean | 4.55 | 8.81 | 7.37 | 15.07 | q |
| 4.55 | 0.00 | 12.78** | 8.46** | 31.59** | q |
| 8.81 | | 0.00 | 4.32* | 18.81** | q |
| 7.37 | | | 0.00 | 23.13** | q |

## 5.3.2: Experiment 2: Investigation of the brain regions responsible for processing biological motion in ASD and control group

Experiment 2 set out to determine whether the brain areas responsible for processing biological motion with and without configural information, were the same in groups with and without ASDs. A whole-brain fixed-effects GLM was calculated for both groups and the following contrasts were specified: intact point-light stimuli minus scrambled point-light stimuli and scrambled point-light stimuli minus static point-light displays. The results of each contrast are presented individually in the following sections.

### 5.3.2.1 Scrambled Point-Lights Displays - Static Point-Light Displays

Volume maps were generated for the contrast scrambled point-light displays - static point light displays. These were then thresholded at $p < 0.001$ and then cluster size threshold estimation was used to control for multiple comparisons with a criterion of $p < 0.05$. Regions were defined based on those clusters that survived the correction. Where regions covered excessively large areas, such as those around the middle temporal and occipital regions, effort was made to ensure that the regions conformed to anatomical boundaries. Tables 5.3 and 5.4 show the Talairach coordinates for the peak voxels from all the regions defined for the ASD group and the control group respectively.

Table 5.3: Coordinates, t-values, p-values and size of cluster in 1mm$^3$ voxels for the contrast of Scrambled point-light displays > Static point-light displays for the ASD group.

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that prefer scrambled biological motion to stationary PLDs** | | | | | | | |
| Cuneus | L | -18 | -85 | 22 | 8.03 | 0.000001 | 2058 |
| | R | 24 | -79 | 13 | 9.11 | 0.000001 | 4057 |
| | R | 24 | -89 | -1 | 8.66 | 0.000001 | 1059 |
| Fusiform Gyrus | L | -44 | -64 | -12 | 6.31 | 0.000001 | 864 |
| | L | -39 | -40 | -14 | 4.97 | 0.000002 | 255 |
| | R | 39 | -43 | -14 | 8.40 | 0.000001 | 3480 |
| Inferior Frontal Gyrus | L | -27 | 26 | -14 | 4.39 | 0.000015 | 148 |
| | R | 42 | 11 | 28 | 4.63 | 0.000005 | 933 |
| | R | 24 | 23 | -14 | 4.45 | 0.00001 | 137 |
| Insula | L | -48 | -40 | 22 | 6.70 | 0.000001 | 504 |
| | L | -30 | 23 | 19 | 4.03 | 0.00006 | 148 |
| | R | 39 | -19 | -8 | 4.20 | 0.00003 | 141 |
| Medial Frontal Gyrus | L | -12 | 26 | 40 | 4.61 | 0.000005 | 224 |
| | L | -6 | 8 | 46 | 4.19 | 0.00003 | 492 |
| | R | 6 | -22 | 67 | 4.17 | 0.00004 | 209 |
| Middle Frontal Gyrus | L | -42 | 14 | 43 | 4.16 | 0.000035 | 169 |
| | L | -30 | 50 | 1 | 4.15 | 0.000035 | 208 |
| Middle Occipital Gyrus | L | -24 | -85 | 4 | 10.40 | 0.000001 | 3482 |
| | L | -24 | -93 | 4 | 7.64 | 0.000001 | 1785 |
| | R | 27 | -85 | 1 | 15.48 | 0.000001 | 3747 |
| Middle Temporal Gyrus | L | -48 | -70 | 1 | 20.30 | 0.000001 | 11836 |
| | R | 45 | -61 | 1 | 22.14 | 0.000001 | 9726 |
| | R | 39 | -49 | 4 | 8.43 | 0.000001 | 1588 |
| | R | 57 | -22 | -11 | 4.16 | 0.000035 | 616 |
| Post-central Gyrus | R | 42 | -25 | 40 | 4.23 | 0.000025 | 157 |
| | R | 30 | -25 | 40 | 3.99 | 0.00007 | 196 |
| Pre-central Sulcus | L | -45 | -1 | 34 | 4.78 | 0.000003 | 1236 |
| | L | -21 | -13 | 46 | 4.54 | 0.000007 | 644 |
| Inferior Occipital Gyrus | L | -24 | -88 | -3 | 6.55 | 0.000001 | 994 |
| Inferior Temporal Gyrus | L | -45 | -52 | 1 | 7.76 | 0.000001 | 907 |
| Lingual Gyrus | R | 18 | -79 | -11 | 9.02 | 0.000001 | 1962 |
| Precuneus | L | -30 | -43 | 49 | 7.25 | 0.000001 | 4645 |
| Superior Frontal Gyrus | R | 21 | 56 | -2 | 4.39 | 0.000015 | 728 |
| Superior Parietal Lobule | R | 33 | -46 | 52 | 5.58 | 0.000001 | 3318 |
| Superior Temporal Gyrus | R | 57 | -31 | 16 | 7.35 | 0.000001 | 2254 |
| **Brain areas that prefer stationary PLDs to scrambled biological motion** | | | | | | | |
| Culmen | R | 6 | -55 | -2 | -4.33 | 0.00002 | 365 |
| Cuneus | R | 6 | -82 | 4 | -6.22 | 0.000001 | 11530 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Precentral Gyrus | R | 48 | -10 | 13 | -4.14 | 0.00004 | 291 |

Table 5.4: Coordinates, t-values, p-values and size of cluster in 1mm$^3$ voxels for the contrast of Scrambled point-light displays > Static point-light displays for the control group.

**Brain areas that prefer scrambled biological motion to stationary PLDs**

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| Cuneus | L | -24 | -82 | 28 | 4.42 | 0.00005 | 260 |
| | L | -15 | -97 | 1 | 6.47 | 0.000001 | 1947 |
| | R | 21 | -76 | 13 | 4.83 | 0.000005 | 1148 |
| Declive | L | -15 | -64 | -20 | 4.75 | 0.000005 | 124 |
| | R | 3 | -79 | -20 | 5.86 | 0.000001 | 833 |
| Fusiform Gyrus | L | -36 | -37 | -11 | 6.16 | 0.000001 | 1034 |
| | R | 21 | -82 | -11 | 6.68 | 0.000001 | 1866 |
| Insula | L | -45 | -37 | 25 | 6.64 | 0.000001 | 1148 |
| | R | 57 | -31 | 19 | 4.90 | 0.000002 | 178 |
| Middle Frontal Gyrus | L | -33 | 47 | -2 | 5.25 | 0.000001 | 705 |
| | L | -30 | 44 | 10 | 4.35 | 0.00002 | 222 |
| Middle Occipital Gyrus | L | -27 | -82 | -2 | 9.35 | 0.000001 | 1784 |
| | R | 39 | -61 | 4 | 16.76 | 0.000001 | 11040 |
| Culmen | L | -45 | -34 | -32 | 3.71 | 0.0003 | 113 |
| Inferior Frontal Gyrus | R | 42 | 5 | 31 | 4.60 | 0.000005 | 1090 |
| Inferior Occipital Gyrus | L | -45 | -76 | -2 | 15.68 | 0.000001 | 11776 |
| Lingual Gyrus | R | 18 | -91 | 1 | 6.65 | 0.000001 | 2392 |
| Middle Temporal Gyrus | L | -52 | -61 | 2 | 8.65 | 0.000001 | 2741 |
| Paracentral Lobule | L | -6 | -34 | 58 | 4.58 | 0.00001 | 112 |
| Parahippocampal Gyrus | R | 39 | -22 | -11 | 5.95 | 0.000001 | 1535 |
| Precentral Sulcus | L | -36 | -4 | 40 | 4.49 | 0.000008 | 1132 |
| Precuneus | L | -21 | -58 | 46 | 5.32 | 0.000001 | 4011 |
| Superior Parietal Lobule | R | 31 | -52 | 55 | 5.53 | 0.000001 | 4585 |
| Superior Temporal Sulcus | R | 39 | -43 | 10 | 5.77 | 0.000001 | 2208 |

**Brain areas that prefer stationary PLDs to scrambled biological motion**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Cuneus | L | -3 | -73 | 10 | -5.42 | 0.000001 | 1300 |
| Lingual Gyrus | L | -12 | -70 | -5 | -4.15 | 0.00004 | 140 |
| | R | 12 | -64 | 7 | -4.49 | 0.000008 | 227 |

<u>Regions common to both groups</u>

From Tables 5.3 and 5.4 it appears that the activation patterns for both groups were very similar regions. Those regions that were more active for scrambled point-light displays

than static point-light displays across both groups were the cuneus, the insula, the MOG and the fusiform gyrus bilaterally, the left MFG, pre-central sulcus, IOG, MTG and the precuneus, and the right IFG, SPL and the lingual gyrus. Only the left cuneus showed increased activation for static point-light displays over scrambled point-light displays across both groups.

Regions that differed across groups

Despite the very similar pattern of activity across the groups, there were a number of regions that were activated only in one of the two groups. In the control group the regions that were more active for scrambled point-light displays than for static point-light displays were the medial frontal gyrus bilaterally, the left IFG and ITG, and the right MTG, Post-Central Gyrus, SFG and the STG. In the ASD group the regions that were more active for scrambled point-light displays than for static point-light displays were the Declive Bilaterally, the right STS and parahippocampal gyrus and the left paracentral lobule and Culmen.

Regions that were more active for static point-light displays than for scrambled point-light displays in the controls only were the right Cuneus, Culmen and Precentral Gyrus, whilst in the ASD group only regions were found in the left cuneus and the lingual gyrus bilaterally.

#### 5.3.2.2 Intact Point-Light Displays-Scrambled Point-Light Displays

The same criterion was applied to the volume map for the contrast of intact point-light displays to scrambled point light displays. Tables 5.5 and 5.6 show the Talairach coordinates for each of the regions found from the contrast for the control group and the ASD group respectively.

Regions common to both groups

Unlike the previous contrast, only two regions was commonly activated for both groups. In both groups the right cingulate gyrus was more active for intact point-light displays over scrambled point light displays, and the right IPL was more active for scrambled point-light displays over intact point-light displays.

Table 5.5: Coordinates, t-values, p-values and size of cluster in 1mm$^3$ voxels for the contrast of Intact point-light displays > Scrambled point-light displays for the control group.

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that prefer intact biological motion to scrambled biological motion** | | | | | | | |
| Inferior Parietal Lobule | L | -54 | -37 | 43 | 4.55 | 0.00001 | 852 |
| | R | 54 | -37 | 43 | 3.66 | 0.0003 | 231 |
| Middle Temporal Gyrus | L | -42 | -80 | 19 | 3.99 | 0.00007 | 158 |
| Superior Frontal Gyrus | L | -39 | 44 | 32 | 3.75 | 0.0002 | 580 |
| Superior Occipital Gyrus | R | 36 | -76 | 28 | 3.90 | 0.0001 | 199 |
| Cingulate Gyrus | R | 9 | -46 | 40 | 4.35 | 0.000015 | 4129 |
| Culmen | L | -6 | -70 | -5 | 3.53 | 0.00045 | 161 |
| Lentiform Nucleus | L | -18 | 8 | -2 | 4.00 | 0.000065 | 412 |
| Lingual Gyrus | R | 12 | -88 | 4 | 6.71 | 0.000001 | 4452 |
| **Brain areas that prefer scrambled biological motion to intact biological motion** | | | | | | | |
| Middle Temporal Gyrus | R | 63 | -1 | -5 | -3.97 | 0.000075 | 220 |
| | R | 45 | -58 | 1 | -5.25 | 0.000001 | 2038 |
| Precuneus | L | -27 | -43 | 49 | -5.81 | 0.000001 | 469 |
| | R | 21 | -58 | 55 | -4.34 | 0.00002 | 484 |
| Cuneus | R | 21 | -79 | 28 | -5.45 | 0.000001 | 1450 |
| Declive | R | 18 | -76 | -11 | -5.84 | 0.000001 | 1109 |
| Inferior Parietal Lobule | R | 30 | -40 | 52 | -4.19 | 0.00003 | 341 |
| Inferior Temporal Gyrus | L | -48 | -64 | 1 | -6.26 | 0.000001 | 2362 |
| Parahippocampal Gyrus | L | -27 | -31 | -2 | -3.65 | 0.0003 | 189 |

Regions that differed across groups

The regions that were more active for intact point-light displays than scrambled point-light displays in the control group were the bilateral IPL, the left MTG, SFG, culmen and lentiform nucleus, and the right SOG, cingulate gyrus and lingual gyrus. In the ASD group the regions that were more active for intact point-light displays than scrambled point-light displays were the bilateral posterior cingulate and cuneus, and the left cingulate gyrus and parahippocampal gyrus.

For both groups, although there were a number of regions that were more active for scrambled point-light displays than for intact point-light displays, there was very little overlap. In the control group these regions were the precuneus bilaterally, the left ITG and parahippocampal gyrus, and the right MTG, Cuneus and Declive. In the ASD group the regions were the left MTG and Declive, and the right lingual gyrus.

Table 5.6: Coordinates, t-values, p-values and size of cluster in $1mm^3$ voxels for the contrast of Intact point-light displays - Scrambled point-light displays for the ASD group

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that prefer intact biological motion to scrambled biological motion** | | | | | | | |
| Cingulate Gyrus | L | -9 | -40 | 31 | 4.30 | 0.00002 | 90 |
| | R | 3 | -40 | 34 | 3.91 | 0.0001 | 148 |
| Precuneus | L | 0 | -52 | 37 | 4.77 | 0.000002 | 295 |
| | L | -6 | -58 | 31 | 3.93 | 0.00009 | 272 |
| | R | 3 | -61 | 19 | 3.92 | 0.0001 | 108 |
| Posterior Cingulate | L | -18 | -58 | 19 | 4.46 | 0.00001 | 210 |
| | R | 12 | -49 | 7 | 4.15 | 0.000035 | 307 |
| Parahippocampal Gyrus | L | -12 | -40 | 7 | 4.58 | 0.00001 | 697 |
| **Brain areas that prefer scrambled biological motion to intact biological motion** | | | | | | | |
| Inferior Parietal Lobule | R | 39 | -31 | 37 | -4.01 | 0.000065 | 100 |
| | R | 36 | -43 | 52 | -4.44 | 0.00001 | 314 |
| Inferior Parietal Lobe | R | 21 | -49 | 55 | -4.42 | 0.00002 | 249 |
| Middle Temporal Gyrus | L | -45 | -64 | 10 | -5.97 | 0.000001 | 1055 |
| Declive | L | -15 | -79 | -11 | -6.74 | 0.000001 | 2197 |
| Lingual Gyrus | R | 12 | -79 | -8 | -6.09 | 0.000001 | 1226 |

## 5.3.3 Experiment 3: Investigating brain regions selective for configural processing in an ASD and control group

### 5.3.3.1 Behavioural Data

In Experiment 3 we tested the participants from both groups at 2 stimulus levels. These were the 50% and 84% correct thresholds derived from the psychometric curves plotted for each individual in Experiment 1. As such, it was hoped that each participant would be

performing at the same level inside the scanner as they did in Experiment 1. Figure 5.2 shows actual scanner performance levels of the group for each of the thresholds tested.

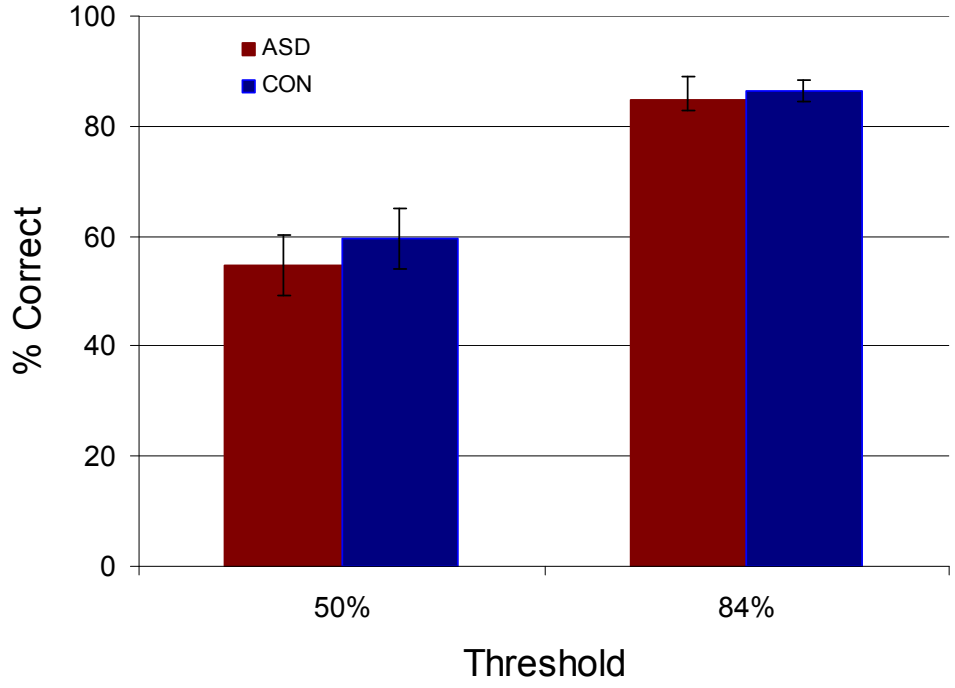


Figure 5.2: Mean proportion correct plotted against predicted proportion correct based on the results of Experiment 1 for each group. Error bars represent Standard Errors.

One-sample t-tests were used to determine whether either group was performing significantly above or below the level expected based on the thresholds predicted in Experiment 1. These confirmed that neither group were performing significantly differently for either the 50 % thresholds (ASD - $t(9) = 0.863$, $p = 0.411$; Control - $t(9) = 2.041$, $p = 0.072$) or the 84% correct threshold (ASD - $t(9) = 0.172$, $p = 0.867$, Control - $t(9) = 1.300$, $p = 0.226$).

In order to confirm that there were still significant differences between performance in each condition, and that there were no differences between the groups in each condition, a 2 x 2 repeated measured ANOVA was run with "Threshold" as a within-group factor and "Group" as a between-group factor. The ANOVA confirmed that the mean percentage correct in the 84% correct condition was significantly higher than the mean percentage correct in the 50% condition $f(1,18) = 67.692$, $p < 0.001$. Furthermore, there was no main effect of "Group" $f(1,18) = 0.441$, $p = 0.515$ or a "Group" x "Threshold" interaction $f(1,18) = 0.212$, $p = 0.651$, confirming that there were no difference in performance levels between the groups overall or within either of the conditions.

#### 5.3.3.2 fMRI Data

A random-effect (RFX) GLM was carried out on the group data. As in Chapter 4, we applied a mask generated from all the regions found to be more active for intact and scrambled PLDS derived from both groups in Experiment 2 to restrict the analysis. Figure 5.3 shows the masked regions derived from Experiment 2 in a glass brain. This reduced the number of voxels entered into the GLM to around 14% of the number of voxels usually entered into an unconstrained whole brain analysis, with a view to boosting power sufficiently to run the RFX GLM and be able to apply the results to the wider population.

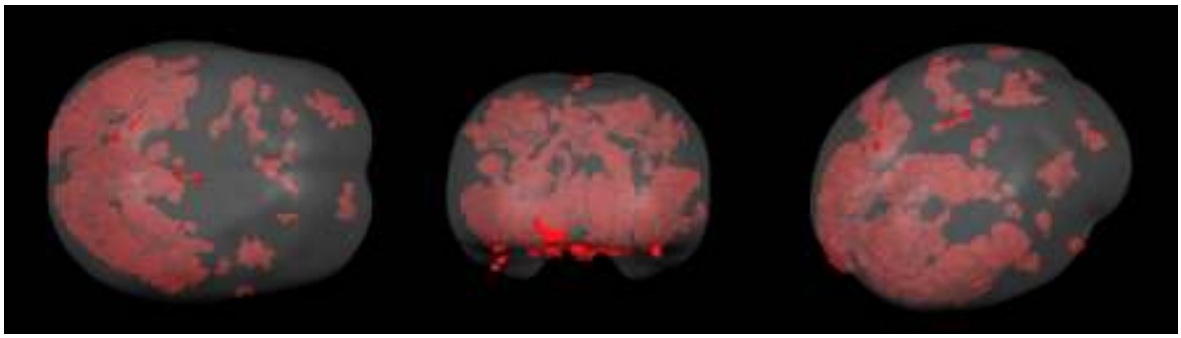

Figure 5.3: Mask generated from the regions found to be more active either to intact over scrambled PLDs or scrambled PLDs over static frames from both groups in Experiment 2.

We generated volume maps for the contrast *84% condition greater than the 50% condition* for both groups. These were thresholded at $p < 0.05$ and a multiple-comparisons correction was carried out using cluster size threshold estimation, with a threshold of $p<0.05$ to determine minimum cluster sizes for each contrast. Regions were defined based on those that survived cluster-size threshold estimation. Where regions covered excessively large areas, such as those around the middle temporal and occipital regions, effort was made to ensure that the regions conformed to anatomical boundaries. Tables 5.7 and 5.8 show the talairach of the peak voxels for each of the regions derived from this contrast for the control and ASD group respectively.

As can be seen from Tables 5.7 and 5.8 the patterns of activation appear to be different between groups. The two groups shared only one region that was more active in the 84% correct condition than in the 50% correct condition, which was the left Middle Frontal Gyrus. The left IPL, ITG and pre-central sulcus were found to be more active in the 84% correct condition in the control group only, whilst the regions found in only the ASD group were the left MOG and the right MTG and fusiform gyrus.

Table 5.7: Coordinates, t-values, p-values and size of cluster in 1mm$^3$ voxels for the contrast of 84% correct stimuli – 50% correct stimuli for the control group

**Brain area that preferred 84% correct stimuli to 50% correct stimuli**

| | | X | Y | Z | t | p> | Nr. Voxels |
|---|---|---|---|---|---|---|---|
| Inferior Parietal Lobule | L | -36 | -46 | 46 | 4.05 | 0.003 | 302 |
| Precentral Sulcus | L | -42 | -4 | 37 | 3.87 | 0.004 | 136 |
| Inferior Temporal Gyrus | L | -45 | -64 | 1 | 3.65 | 0.0055 | 489 |
| Middle Frontal Gyrus | L | -24 | -7 | 52 | 3.19 | 0.02 | 141 |

**Brain areas that preferred 50% correct stimuli to 84% correct stimuli**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Middle Frontal Gyrus | L | -39 | 5 | 52 | -3.69 | 0.006 | 251 |
| | R | 6 | 56 | 1 | -4.76 | 0.0015 | 167 |
| Middle Temporal Gyrus | L | -48 | -70 | 17 | -3.88 | 0.004 | 713 |
| | L | -43 | -76 | 25 | -4.50 | 0.0015 | 199 |
| | R | 51 | -64 | 13 | -4.51 | 0.0015 | 327 |
| Posterior Cingulate | L | -9 | -58 | 16 | -8.84 | 0.00002 | 1410 |
| | R | 13 | -49 | 7 | -5.15 | 0.00065 | 414 |
| Precuneus | L | -6 | -52 | 39 | -4.55 | 0.0015 | 1978 |
| | L | 0 | -37 | 44 | -5.09 | 0.0007 | 396 |
| | R | 6 | -58 | 33 | -3.60 | 0.006 | 209 |
| | R | 3 | -58 | 19 | -6.23 | 0.0002 | 231 |
| Angular Gyrus | R | 37 | -76 | 31 | -7.83 | 0.00003 | 496 |
| Cingulate Gyrus | R | 3 | -36 | 34 | -3.76 | 0.005 | 172 |
| Cuneus | L | -5 | -97 | 7 | -3.65 | 0.0055 | 242 |
| Lentiform Nucleus | L | -22 | 5 | -5 | -3.94 | 0.0035 | 311 |

Table 5.8: Coordinates, t-values, p-values and size of cluster in 1mm$^3$ voxels for the contrast of 84% correct stimuli – 50% correct stimuli for the ASD group

**Brain area that preferred 85% correct stimuli to 50% correct stimuli**

| | | X | Y | Z | t | p< | Nr. Voxels |
|---|---|---|---|---|---|---|---|
| Middle Frontal Gyrus | L | -35 | -1 | 43 | 5.07 | 0.0007 | 1569 |
| Middle Temporal Gyrus | R | 42 | -55 | 4 | 4.94 | 0.00085 | 398 |
| Fusiform Gyrus | R | 36 | -46 | -5 | 3.84 | 0.004 | 839 |
| Middle Occipital Gyrus | L | -36 | -61 | 4 | 3.77 | 0.0045 | 2421 |

**Brain areas that preferred 50% correct stimuli to 84% correct stimuli**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Posterior Cingulate | L | -6 | -43 | 7 | -3.51 | 0.007 | 616 |
| Middle Temporal Gyrus | R | 57 | -22 | -11 | -4.95 | 0.0008 | 730 |
| Cingulate Gyrus | R | 2 | -43 | 31 | -6.52 | 0.00015 | 1036 |

Of the regions that were more active in the 50% correct condition than in the 84% correct condition only two were shared by both groups, the right MTG and cingulate gyrus. Regions that were more active in the 50% correct condition than in the 84% correct condition in the control group only were the MTG, MFG, posterior cingulate and precuneus bilaterally, the left cuneus and lentiform nucleus, and the right angular gyrus and the lingual gyrus. In the ASD group the only the regions found were the left posterior cingulate and the right MTG and cingulate gyrus.

#### 5.3.3.3 Granger Causality Mapping

Each of the regions derived from Experiment 3 were used as seeds for GCM. From each of these seeds the analysis returns two volume maps, one showing regions that correlate instantaneously with the activity in the seed (iGCM) and another that, more importantly, shows the directional influences of the seed on other regions and of other regions upon the seed (dGCM). Granger Causality Mapping ideally required seed regions of no more than 300 voxels in order to avoid spurious correlations with regions not truly correlated with the one of interest. To meet this requirement, for any region of more than 300 voxels, we took the 299 voxels within the region that had the smallest Euclidian distance from the peak voxel. This typically gave spherical regions for larger clusters, but for smaller clusters that were perhaps narrower in one direction than others, ensured that the resulting regions were constrained by the contours of the original region.

As Experiment 3 had been designed with GCM in mind the stimuli in each condition had been blocked into groups providing data across enough volumes to look at the dGCM maps for each separately. In other words, the GCM analysis for the 84% correct condition used the regions that were more active in the 84% correct condition than in the 50% condition, and the GCM analysis for the 50% correct condition used those regions that were more active in the 50% condition than in the 84% correct condition. For all analyses the GCM analysis was run separately for each participant using the time course from each of the three runs. This produced a dGCM volume map for each individual and each condition for the specified seed regions. These were thresholded with a FDR of $q < 0.05$. These maps were combined and a t-test was run across the whole brain to identify voxels that were significantly different from zero. Maps were generated from the t-test where positive t-values represented areas that were influenced by the seed region and negative values represented regions that influenced the seed region. The results of the dGCM analysis will

be discussed separately below for the 50% correct stimuli and the 84% correct stimuli. For all analyses, any seed region that did not show significant direction influence was omitted from tables and figures.

***50% Correct Condition***

Figure 5.4 shows the seed regions for both groups in a glass brain and Tables 5.9 and 5.10 show the regions of influence to and from each of the seed regions in the 50% correct condition for the control group and the ASD group respectively. Figures 5.5 and 5.6 illustrate the directional influences to and from the seed regions for the control group and ASD group respectively.

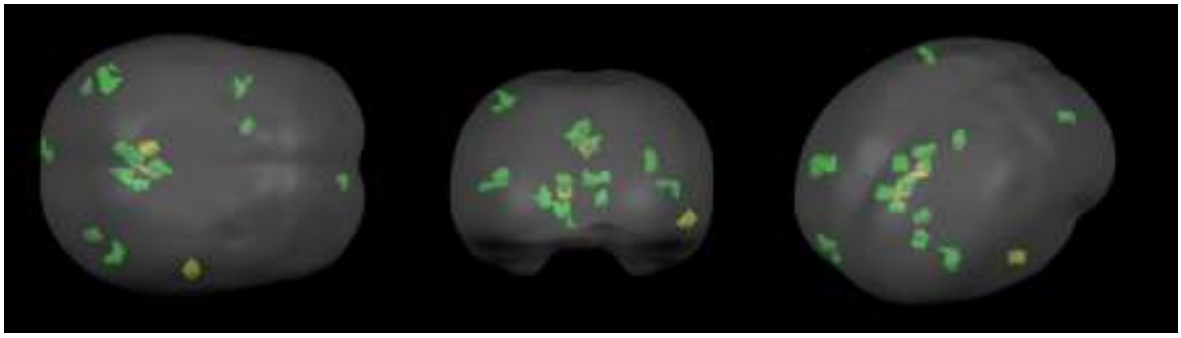

Figure 5.4: Seed regions that showed preferential activity for 50% correct stimuli over 84% correct stimuli separated by colour. In green the seed regions for the control group and in yellow the seed regions for the ASD group.

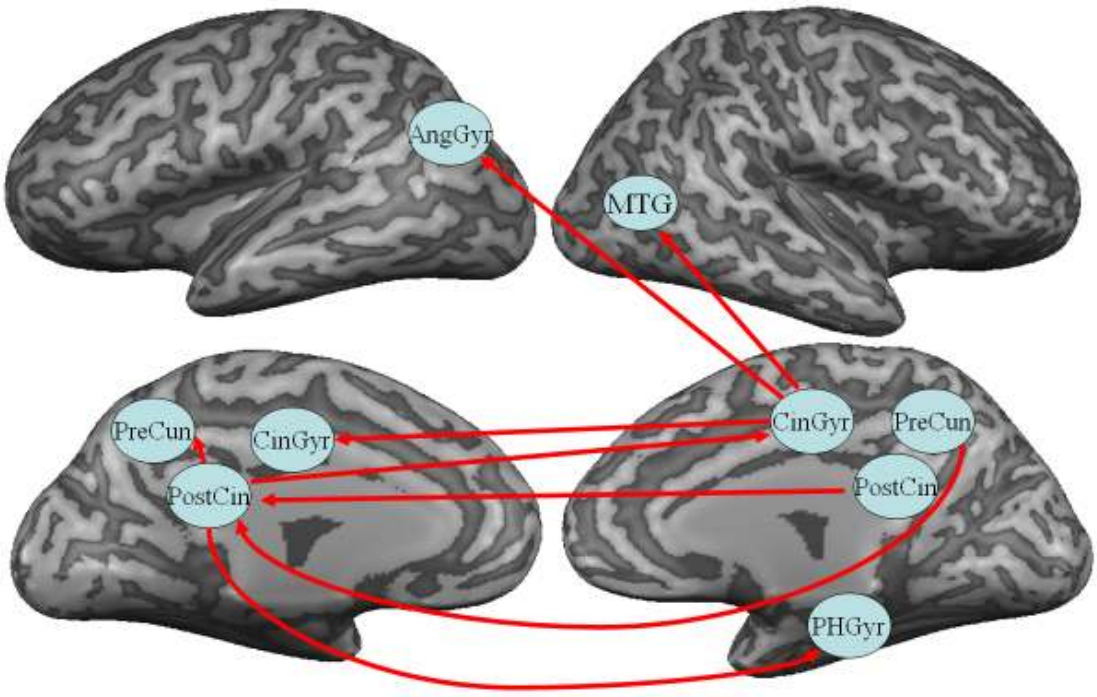


Figure 5.5: Directional Influences determined from dGCM analysis in the Control group for the 50% condition.

Table 5.9: Peak talairach coordinates, t-values, p-values and number of voxels for regions derived from GCM analysis for each seed region from Experiment 3 in the control group

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that are influenced by activity in the right Angular Gyrus** | | | | | | | |
| Superior Occipital Gyrus | R | 36 | -73 | 25 | 4.06 | 0.004 | 46 |
| **Brain areas that are influenced by activity in the right Cingulate Gyrus** | | | | | | | |
| Posterior Cingulate | R | 3 | -37 | 25 | 4.38 | 0.0025 | 841 |
| **Brain areas that influence the activity in the left Cuneus** | | | | | | | |
| Cuneus | L | -6 | -94 | 4 | -3.43 | 0.009 | 95 |
| Middle Occipital Gyrus | L | -18 | -97 | 4 | -3.26 | 0.015 | 46 |
| **Brain areas that are influenced by activity in the left Posterior Cingulate** | | | | | | | |
| Posterior Cingulate | L | -3 | -61 | 13 | 3.80 | 0.006 | 735 |
| Precuneus | L | -3 | -49 | 31 | 4.84 | 0.0015 | 1050 |
| Cingulate Gyrus | L | 0 | -34 | 37 | 3.55 | 0.008 | 202 |
| Superior Temporal Sulcus | L | -39 | -55 | 16 | 2.94 | 0.02 | 32 |
| Middle Temporal Gyrus | L | -39 | -70 | 25 | 4.18 | 0.003 | 166 |
| **Brain areas that are influenced by activity in the right Posterior Cingulate** | | | | | | | |
| Lingual Gyrus | L | 0 | -73 | 7 | 3.01 | 0.02 | 41 |
| Posterior Cingulate | L | -6 | -43 | 7 | 3.64 | 0.007 | 91 |
| Lingual Gyrus | L | -12 | -55 | 1 | 3.67 | 0.007 | 46 |
| Posterior Cingulate | R | 6 | -49 | 16 | 4.05 | 0.004 | 1295 |
| **Brain areas that are influenced by activity in the left Precuneus** | | | | | | | |
| Precuneus | L | -3 | -55 | 40 | 4.30 | 0.003 | 867 |
| **Brain areas that influence activity in the left Precuneus** | | | | | | p< | No. Voxels |
| Posterior Cingulate | L | -3 | -55 | 19 | -3.05 | 0.02 | 105 |
| Lingual Gyrus | L | -15 | -52 | 4 | -3.11 | 0.02 | 30 |
| Posterior Cingulate | R | 3 | -49 | 25 | -3.06 | 0.02 | 64 |
| **Brain areas that are influenced by activity in the right Precuneus** | | | | | | | |
| Precuneus | L | 0 | -55 | 43 | 3.00 | 0.02 | 63 |
| Posterior Cingulate | R | 9 | -55 | 19 | 4.59 | 0.002 | 640 |

As can be seen from Figures 5.5 and 5.6, there appear to be some similarities between the two groups, but also some rather substantial differences. In both groups the only shared influence is from the right posterior cingulate to the left posterior cingulate, and from the left posterior cingulate to the left precuneus.

In addition to the shared regions between the two groups, the control group shows directional influence in medial areas from the right precuneus to the left precueus, the right posterior cingulate to the left posterior cingulate and lingual gyrus, the left posterior cingulate to the left cingulate gyrus, precuneus, STS and MTG. There is also directional

influence from two regions on the lateral surfaces of the two hemispheres, these being from the right angular gyrus to the right SOG and the left MOG to the left cuneus.

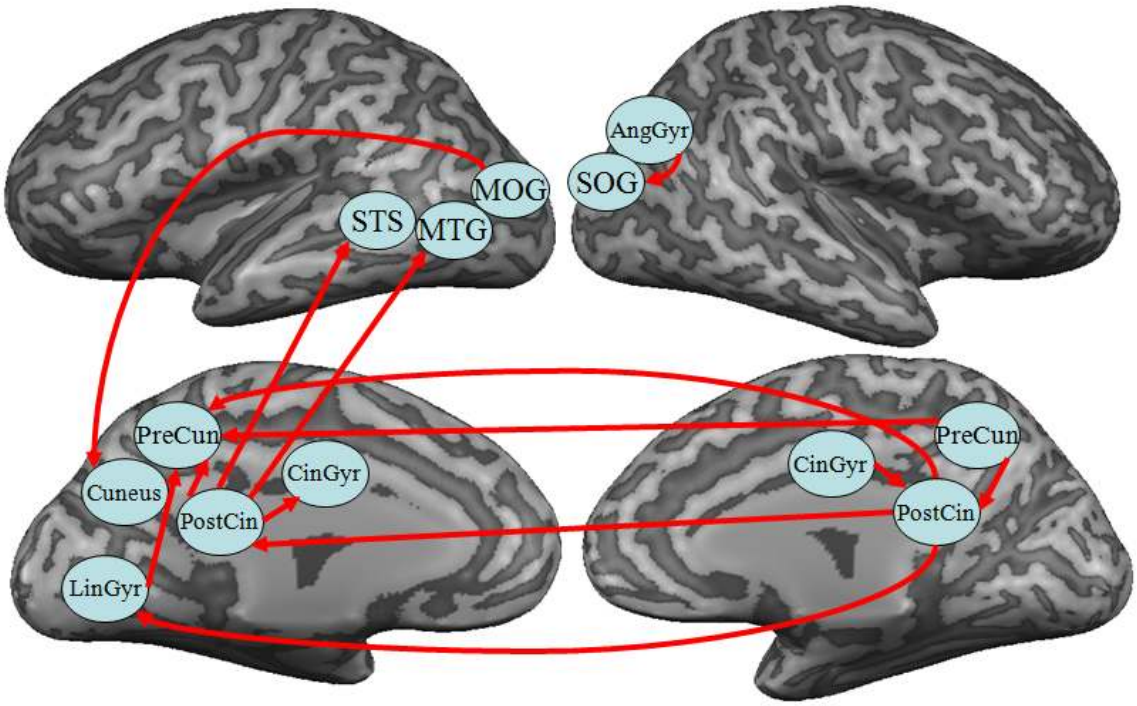


Figure 5.6: Directional Influences determined from dGCM analysis in the ASD group for the 50% condition.

Table 5.10: Peak talairach coordinates, t-values, p-values and number of voxels for regions derived from GCM analysis for each seed region from Experiment 3 in the ASD group

| | | X | Y | Z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that are influenced by activity in the right Cingulate Gyrus** | | | | | | | |
| Angular Gyrus | L | -36 | -58 | 40 | 2.93 | 0.02 | 73 |
| Cingulate Gyrus | L | 0 | -40 | 34 | 5.16 | 0.0006 | 2865 |
| Middle Temporal Gyrus | R | 39 | -55 | 25 | 3.06 | 0.015 | 36 |
| **Brain areas that influence activity in the right Cingulate Gyrus** | | | | | | | |
| Posterior Cingulate | R | 9 | -55 | 19 | -2.77 | 0.025 | 52 |
| **Brain areas that are influenced by activity in the left Posterior Cingulate** | | | | | | | |
| Posterior Cingulate | L | -9 | -43 | 7 | 4.16 | 0.0025 | 214 |
| Precuneus | L | 0 | -52 | 40 | 3.19 | 0.015 | 44 |
| Precuneus | L | -3 | -61 | 37 | 3.09 | 0.015 | 175 |
| Parahippocampal Gyrus | R | 9 | -37 | 4 | 3.42 | 0.008 | 176 |
| **Brain areas that influence activity in the left Posterior Cingulate** | | | | | | | |
| Posterior Cingulate | L | -15 | -61 | 13 | -2.75 | 0.025 | 135 |
| Posterior Cingulate | L | -6 | -52 | 19 | -3.62 | 0.006 | 43 |
| Precuneus | R | 3 | -64 | 22 | -3.91 | 0.004 | 30 |
| Posterior Cingulate | R | 6 | -55 | 16 | -4.35 | 0.002 | 550 |

In the ASD group the pattern of influence outside the shared regions are from the right cingulate gyrus to the right MTG, left angular gyrus and the left cingulate gyrus, from the right posterior cingulate to the left posterior cingulate, and from the left posterior cingulate to the to the right cingulate gyrus and parahippocampal gyrus.

### *84% Correct Condition*

Figure 5.7 shows the seed regions that showed preferential activity to 84% correct stimuli over 50% correct stimuli for both groups, and Table 5.11 and 5.12 show the regions of influence to and from each of the seed regions for the control group and ASD group respectively. Figures 5.8 and 5.9 illustrate the directional influences to and from the seed regions for the control group and ASD group respectively.

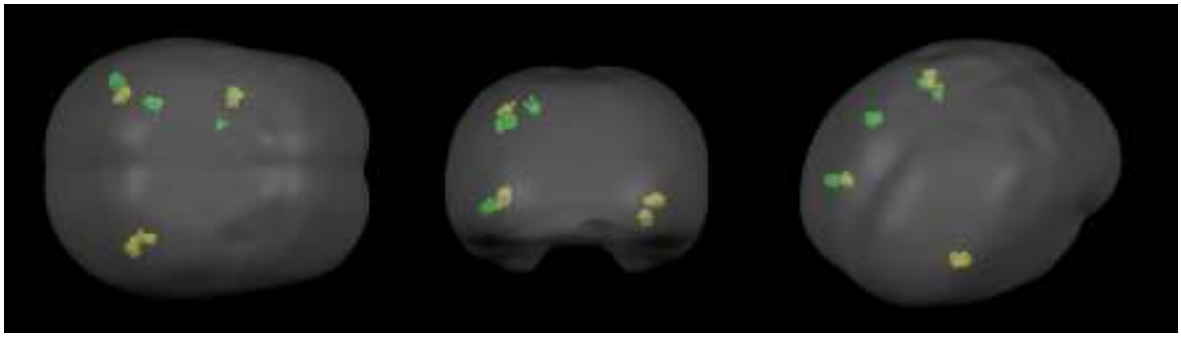

Figure 5.7: Seed regions that showed preferential activity for 84% correct stimuli over 50% correct stimuli separated by colour. In green the seed regions for the control group and in yellow the seed regions for the ASD group.

Table 5.11: Peak talairach coordinates, t-values, p-values and number of voxels for regions derived from GCM analysis for each seed region from Experiment 3 in the Control group

| | | x | Y | z | T | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that are influenced by activity in the left Inferior Parietal Lobule** | | | | | | | |
| Precuneus | L | -30 | -46 | 49 | 4.49 | 0.003 | 309 |
| Superior Parietal Lobule | L | -30 | -46 | 55 | 3.22 | 0.02 | 36 |
| Inferior Parietal Lobule | L | -36 | -37 | 40 | 4.00 | 0.004 | 264 |
| Inferior Parietal Lobule | L | -42 | -49 | 52 | 4.14 | 0.004 | 125 |
| **Brain areas that are influenced by activity in the left Inferior Temporal Gyrus** | | | | | | | |
| Superior Parietal Lobule | L | -33 | -52 | 49 | 2.74 | 0.03 | 40 |
| Middle Occipital Gyrus | L | -39 | -73 | 10 | 3.44 | 0.009 | 86 |
| Inferior Temporal Gyrus | L | -48 | -64 | 1 | 6.09 | 0.0003 | 1956 |
| Inferior Occipital Gyrus | R | 42 | -70 | -2 | 3.21 | 0.015 | 49 |
| Middle Occipital Gyrus | R | 33 | -79 | 1 | 2.80 | 0.025 | 40 |

Table 5.12: Peak talairach coordinates, t-values, p-values and number of voxels for regions derived from GCM analysis for each seed region from Experiment 3 in the ASD group

| | | X | Y | Z | t | p< | No. Voxels |
|---|---|---|---|---|---|---|---|
| **Brain areas that are influenced by activity in the right Middle Temporal Gyrus** | | | | | | | |
| Precuneus | L | 0 | -58 | 46 | 2.42 | 0.04 | 28 |
| Precuneus | L | 0 | -61 | 34 | 2.91 | 0.02 | 68 |
| Inferior Temporal Gyrus | R | 39 | -52 | 1 | 2.78 | 0.025 | 49 |
| Middle Occipital Gyrus | R | 39 | -67 | 7 | 2.79 | 0.025 | 75 |
| **Brain areas that are influenced by activity in the right Fusiform Gyrus** | | | | | | | |
| | | Peak X | Peak Y | Peak Z | t | p< | No. Voxels |
| Culmen | L | -6 | -67 | -8 | 6.98 | 0.000065 | 300 |
| Parahippocampal Gyrus | R | 36 | -49 | -5 | 20.84 | 0.000001 | 1535 |
| Inferior Occipital Gyrus | R | 33 | -82 | -2 | 9.46 | 0.000007 | 189 |
| Declive | R | 15 | -55 | -11 | 6.59 | 0.0002 | 538 |
| Lingual Gyrus | R | 12 | -52 | 1 | 6.70 | 0.00009 | 507 |
| **Brain areas that are influenced by activity in the left Middle Occipital Gyrus** | | | | | | | |
| Inferior Temporal Gyrus | L | -51 | -49 | -5 | 2.85 | 0.02 | 28 |
| **Brain areas that influence activity in the right Middle Occipital Gyrus** | | | | | | | |
| Lingual Gyrus | L | -12 | -61 | 7 | -2.99 | 0.02 | 40 |
| Middle Occipital Gyrus | L | -39 | -73 | 4 | -3.03 | 0.015 | 30 |
| Lingual Gyrus | L | -9 | -67 | 1 | -3.54 | 0.0065 | 93 |

As can be seen from Figures 5.8 and 5.9, unlike in the 50% correct condition, the pattern of influence seen in the two groups shares no commonality. In the control group the regions that influence each other are primarily on the lateral surface of the two hemispheres with directional influence from the left ITG to the left SPL, the right IOG and to the MOG bilaterally. The right IPL influences activity in the left SPL and also in the left precuneus.

In the ASD group however, there are many more medial areas than in the control group, both influencing and being influenced by more lateral regions. There seem to be two distinct networks of influence in the ASD group. The first involved influence from the right MTG to the left precuneus, right ITG and MOG. The right MOG is also influenced by the left lingual gyrus and the right MOG, which also influences the left ITG. Neither the left nor the right ITG show directional influence over any other region. The second network shows a pattern of influence from the right fusiform gyrus to the right IOG and lingual gyrus.

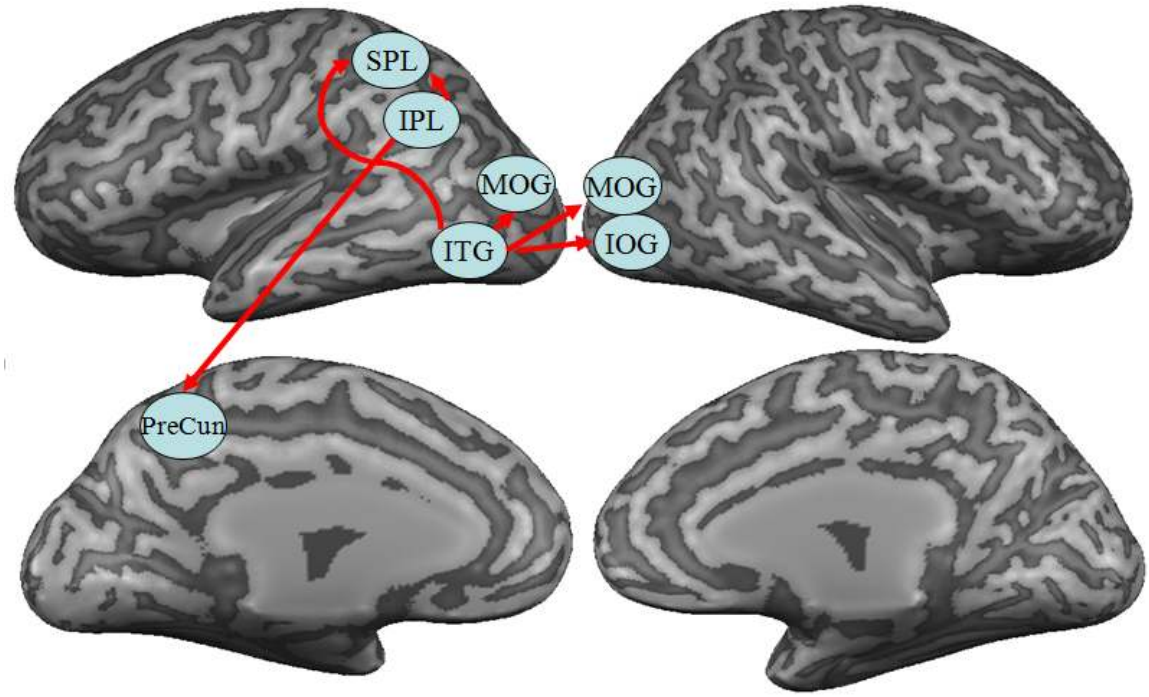


Figure 5.8: Directional Influences determined from dGCM analysis in the control group for the 84% condition.

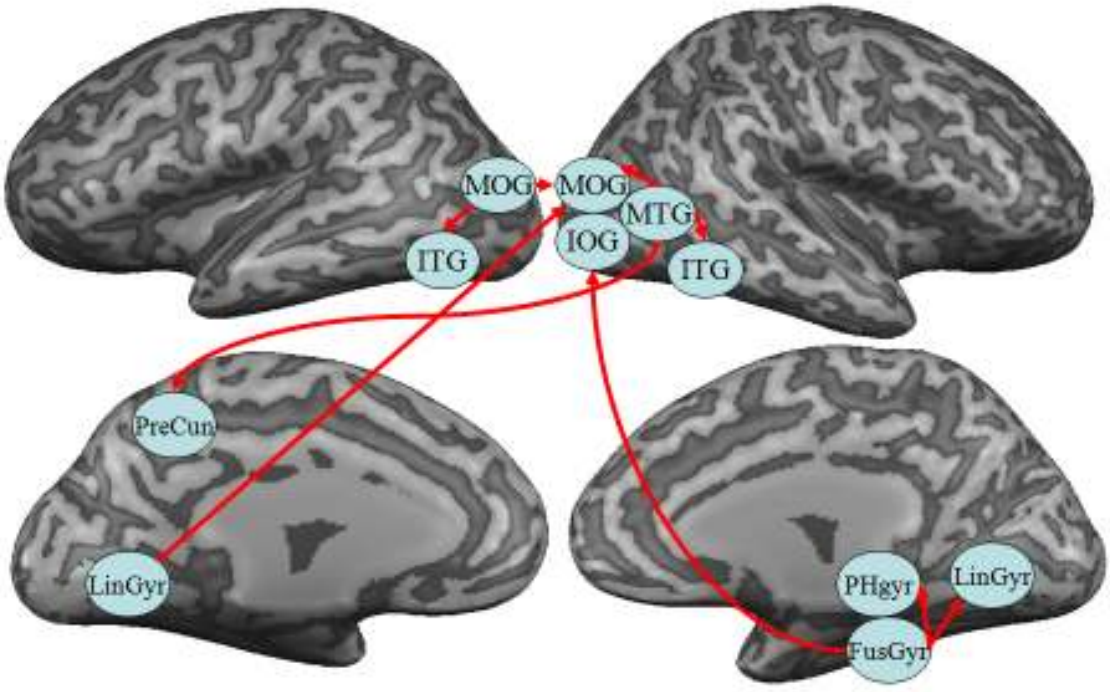


Figure 5.9: Directional Influences determined from dGCM analysis in the ASD group for the 84% condition.

## 5.4 Discussion

In this chapter, it was hypothesised that the behavioural performance on a direction discrimination task using PLDs would be comparable between an adult group with ASDs and an age- and IQ-matched control group, but that the underlying neural processes would be different, particularly in the fusiform gyrus, ITG, parietal regions and potentially the STSp. The hypothesis that performance in the behavioural task would be comparable between the two groups was confirmed. There was no significant difference between the 50% correct thresholds in the configural cues present condition and although the 84% correct thresholds in the ASD group were slightly higher than in the control group, this difference was not significant. This is in line with previous studies that have found small differences in performance between adult groups with ASDs and controls, with the ASD group tending to perform more poorly (Moore et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008; Murphy et. al. 2009). Furthermore, both groups were equally affected by the removal of configural cues from the stimuli.

In Experiment 2 we measured brain activity of the participants whilst viewing either PLDs of intact walking motions, PLDs of the same motion scrambled or static structureless frames containing the same number of dots as the moving PLDs. As hypothesised, the pattern of activation found was different for the two groups in response to both intact stimuli and scrambled stimuli. Of particular interest is that in response to scrambled PLDs, the control group showed bilateral activation of inferior temporal regions, middle, inferior and superior frontal regions that were not found in the ASD group. Furthermore, the IPL and SFG were found to be selective for intact over scrambled PLDs, whilst the ITG showed preferential activation for scrambled over intact stimuli in controls, but not the ASD group. The finding lends support to the suggestion that in typically developed people the percept of a coherent moving human figure is represented in inferior parietal regions, rather than in earlier temporal regions (Battelli, et. al. 2003), though this may be restricted to the more posterior portions of the IPL, as a more anterior region in the right hemisphere showed a preference for scrambled stimuli in both groups. Furthermore, as also found in the same experiment in Chapter 4, it seems that the ITG processes information from consecutive static frames that do not depict smooth continuous movements (Downing et. al. 2006) as it was more active for scrambled over intact stimuli and static frames. That these regions (IPL & ITG) were not found in the ASD, group lends support to the idea that

there may be dysfunction in these areas in this population, or that connections between these two areas may be disrupted.

Of further interest is that the fusiform gyrus was selectively active for scrambled stimuli over static stimuli, and did not differentiate between intact and scrambled stimuli in both groups. The fact that it did not differentiate between the scrambled and intact PLDs is curious, in that it is thought to process static form cues that are temporally congruent (Downing et. al. 2006). This may be due to the slightly different stimuli used in the present chapter. In Chapter 4 the scrambled and intact stimuli were generated by repeating 1s clips of walking actions, which resulted in a small jerking motion between the last frame of one clip and the first frame of the next clip due to the different start and end points of the joints in the gait cycle. For the present experiment, the start and end points were smoothed between repetitions of the clips, such that the local motions of the points looked smooth and continuous throughout the stimulus duration. This would mean that consecutive frames would appear as a continuous movement, either with structural cues in the intact stimuli, or without in the case of the scrambled stimuli. This does not however explain why these regions did not differentially activate for intact and scrambled PLDs. It is possible that the activity in these regions is actually just processing smooth continuous movements, or that by constraining the local motions to the joints creates latent structural cues across consecutive frames, as if processing a complex object in motion.

The regions found in experiment 2 that were selective for scrambled and intact PLDs, were combined for both groups and used as a mask to restrict the RFX analysis for Experiment 3. In this experiment stimuli were presented to each participant that corresponded to each individuals' 50% and 84% correct thresholds from the behavioural experiment, and participants were asked to perform the same task inside the scanner. Behavioural results from inside the scanner confirmed that the proportions correct matched the thresholds set. At the 84% correct threshold, participants were correctly able to discriminate the direction of locomotion using configural cues from the walker, whereas at the 50% threshold the local motion cues and form cues were equally salient, such that participants were at chance levels. It is thought that the global percept of a walker would only be distinguishable from the noise in the 84% correct stimuli.

Regions were found in the right MTG and cingulate gyrus and the left posterior cingulate that were common to both groups, and were preferentially active for 50% correct stimuli,

whereas only the left MFG showed a preference for 84% correct stimuli in both groups. In addition to those regions found that were commonly selective for 50% correct stimuli across both groups, several additional areas showed the same preference in the control group only, including the bilateral MFG and precuneus, the right posterior cingulate and angular gyri and the left MTG, cingulate gyrus and cuneus. There were no regions that were preferential for 50% correct stimuli in the ASD group that were not also found in the control group. Cortical regions that were preferentially active for 84% correct stimuli in the control group only were the left IPL, precentral sulcus and the ITG. Regions that were selective for 84% correct stimuli in the ASD group were the left MOG and the right MTG and fusiform gyrus.

This pattern of results for increased activations to the 50% correct stimuli over 84% correct stimuli suggest that both groups seem to use similar brain areas when processing stimuli in which configural cues are balanced by local motion cues and the percept of a coherent walker is not present, though the control group utilise a number of predominantly medial regions not used by the ASD group. When processing the stimuli in which the percept of a walker is apparent from amongst a small number of noise points, the 84% correct stimuli, the control group seem to recruit a network of regions comprising ITG, IPL and premotor areas. This is in line with the results of Chapter 4, and suggests that the static postural information processed in the ITG may be integrated with motion information, potentially in the STS (Peuskins et al, 2005) before being combined to generate a global percept of a walker in motion in the parietal cortex (Battelli et al, 2003). This would be in line with the model of biological motion proposed by Giese and Poggio (2003), in which snapshot neurons in the ventral stream are integrated with complex motion pattern neurons in the STS. In contrast however, the ASD group seemed to recruit the fusiform gyrus, rather than the inferior temporal regions, when processing these stimuli. This is rather surprising as it suggests that the stimuli are processed as a series of static postures rather, than being integrated with local motion cues and that the task is performed using occipital and temporal regions, rather than parietal and frontal regions. This would be more in line with the theory proposed by Lange and Lappe (2006), which postulates that biological motion processing can be accomplished without global or local motion processing by the matching of static frames containing human postural information across time.

The results of the GCM analysis appear to confirm that the two groups are using different networks when processing PLDs containing a coherent percept of a walker. The analysis

revealed that in the control participants, activity in the ITG influences activity in parietal regions and also in a number of occipital regions. Although the directional influence from ITG seems to be to superior parietal regions, the SPL is also influenced by the IPL. As mentioned, one restriction of GCM is that it does not distinguish intermediate regions that mediate the influences. It seems likely that the influence to SPL, may be through the STS, IPL or both. In contrast to the control group, the GCM analysis suggests that instead of the ITG being the key region, that the right fusiform gyrus and MTG are the key areas in processing the biological motion in the ASD group. The fusiform gyrus influences regions in the IOG, parahippocampal gyrus and lingual gyrus, whilst the MTG influences the right ITG, the left MOG and the precuneus. In the ASD group the left and right ITG do not influence any other regions, suggesting that this may be the key difference between the groups. If inferior temporal regions were either dysfunctional, or had disrupted connections with regions such as the STS, then this would potentially exclude the integration of static postures and local motions in regions such as the STS or even parietal regions.

The results of both Experiments 2 and 3 suggest that the ITG bilaterally may be dysfunctional in people with ASDs, or may have disrupted connections with parietal regions, which is in line with the findings of Herrington et. al. (2007). This region seems to play a key role in processing static cues for integration with motion cues in the dorsal stream (Peuskins et al, 2005; Downing et al, 2006). If these regions were dysfunctional in people with ASDs it would be expected that they would show impairments in biological motion processing. However, this is not the case in the adult groups tested in the present study, or indeed in previous studies (Moore et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008; Murphy et. al. 2009), though studies on children suggest that there is potentially a developmental delay (Annaz, et. al. 2009; Blake et. al. 2003; Klin et. al. 2003; Klin & Jones, 2008; Klin et. al. 2009). The results of experiment 3 may suggest a possible explanation for this. If ITG functioning is disrupted in people with ASD from an early age then perhaps the brains of this population are able, in some cases, to utilise another pathway that typically subserves different functioning. Both the models of Giese and Poggio (2003) and Lange and Lappe (2006) propose neurologically plausible theories of how direction discrimination can be achieved, either through integration of local or global motion cues with form cues, or through temporal template matching of static cues. The former theory, in light of recent evidence (e.g. Downing et. al. 2006; Peuskins et. al. 2005), would most likely utilise the links between the ITG and STS to integrate form information

with motion cues, whilst the latter theory could accomplish the task using only static congruent frames of a motion. It may be that in typically developed people both these systems are used depending on the task demands, but that in people with ASDs only fusiform areas, are available. As such, the developmental delay in biological motion processing seen in people with ASDs may be due to neural rewiring in order to accommodate the increased task demands placed on the fusiform gyrus. It would also appear that, although the fusiform gyrus is known to have connections with parietal regions Bokde et al. 2006, there is no directional influence between these areas in the task performed, further supporting the idea that the ASD group are using simple template matching and not efficiently integrating form and motion information.

It may be that, if the dysfunction is specific to the ITG rather than between connections between inferior temporal and parietal regions, the problem is with a more general complex motion processing deficit as found by Atkinson (2009) and others (e.g. Milne, et. al. 2006, 2002). Furthermore, the severity of symptoms may affect the extent to which the recruitment of other brain regions is possible within the ASD population. It is beyond the scope of the present paper to address these issues, but future studies may be able to shed light on them, and to directly test the effect that age and symptom severity has on motion and biological motion processing.

In conclusion, the present chapter has demonstrated that adults with ASDs perform comparably with typically developed controls in a task involving direction discrimination from PLDs, which is thought to require the integration of form and motion cues. However, the neural systems used by the two groups to perform the task appear to be substantially different, with the control group potentially using a form and motion integration circuit involving influence from the ITG to parietal regions, and the ASD group potentially utilising a temporal template matching approach that can operate independently of local motion cues. These findings suggest that ITG may be dysfunctional in people with ASDs, or may have dysfunctional connections with parietal regions, but that by adulthood regions in the fusiform gyrus have adapted to allow this group to utilise different strategies to perform the task using different information.

# Chapter 6: General Discussion

The aim of this thesis was to attempt to clarify the nature of biological motion processing in people with ASDs, and to elucidate the neural systems that may underlie any differences in processing between this clinical group and typically developed individuals. In a series of chapters we have investigated biological motion detection, action categorisation, affect categorisation and direction discrimination from PLDs in people with ASDs, and devised novel stimuli to examine biological motion processing in an fMRI environment. The results of each section will be discussed in turn, and then put into a more global context and discussed in terms of the implications for future research.

## 6.1 Detection of Biological Motion, Action categorisation and Affect categorisation in people with ASDs

In Chapter 2 we investigated the ability of people with ASDs to detect biological motion in noise, to categorise actions from action blends of knocking and lifting actions, and to categorise affect from throwing actions. Pilot experiments suggested that people with ASDs had difficulties with detecting biological motion in noise and categorising affect, particularly from angry stimuli. Furthermore, although performance did not significantly differ in the action categorisation task, the ASDs group was significantly more variable than the control group and this variance was distributed among a wider range of stimulus levels around the PSE.

However, in the main experiments, in which the control group was pairwise matched on CA and FSIQ, the differences in biological motion detection and affect categorisation became smaller and non-significant, though the ASD group were still significantly more variable on the action categorisation task. The findings that the ASD group did not differ significantly from the control group in the detection of biological motion and action categorisation, is in line with the findings from a number of studies that have suggested that people with ASD do not suffer from biological motion processing impairments (Moore, et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008), though the performance of the ASD groups were consistently below that of the controls. Although both results showed non-significant differences between the groups, the results of the pilot study on detection of biological motion taken together with the generally poorer performance in the

main experiment and the consistent finding of increased variability in the action categorisation tasks, suggests that there may still be issues in processing biological motion and a large amount of variability in processing local motion cues. Previous studies have indicated that children with ASDs do show impairments in biological motion processing (e.g. Blake et. al. 2003), that these impairments may be present at a very early age (Klin et. al. 2003, Klin & Kones 2008; Klin et. al. 2009) and that there may be a developmental delay associated with biological motion processing in ASDs (Annaz et. al. 2009). If it is the case that there is a developmental delay, then the participants in the present study, who were all adults, may have caught up with typically developed people. Another factor, that is not perhaps distinct from that of age, is the severity of symptoms. Although based mainly on observation and self report during debriefing, it seemed that the participants from the pilot study had slightly more severe symptoms, and were, incidentally, younger, on average than those used in the main experiments. It may be, therefore, that severity of symptoms is an indicator of difficulties with biological motion processing. Furthermore, there may be differences within the ASD group as to the type of symptoms they exhibit that may account for the increase in variability within the ASD group (Walter et. al. 2009), such as the "systemizing trait" (Baron-Cohen, 2008). However, as the participants used in the present experiments were diagnosed using a range of different scales, in particular the DISCO and ADI, it was not possible to test this directly.

More curious, is that the ASD group were comparable to controls on measures of affect categorisation. Difficulties with processing affect from PLDs of human actions are a consistent finding of the previous research (Moore et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008; Atkinson 2009). We did find significant differences between ASD and control participants in the pilot version of the task and the inconsistent findings could be due to a number of factors. Firstly, in the pilot task only the arm of the point-light actor was displayed, whereas in the main experiment all the points were shown. It could be that the increased informational content of the complete figure in the stimuli for the main experiment, facilitated better performance for the ASD group. Furthermore, in the pilot task the velocity of the wrist points in every action were normalised to the median velocity of all the actions, removing the velocity profile of the point as a potential source of information about the affect. Lastly, the same factors that may have influenced performance on the other two tasks, namely age and the severity of symptoms, could have played a role. It may have been for instance, that the ASD group have over time, developed strategies that allow them to correctly categorise affect that are different from

the strategies used by the control group. An analysis of the pattern of errors made by each group does suggest that there were differences. Unlike the control group, the ASD group tended to misattribute angry throws to the sad category, which is consistent with a difficulty in processing negative affects (Teunisse & de Gelder, 2001; Atkinson, 2009). This also suggests that velocity cues do not play a particularly important role in the strategy adopted by the ASD group, as the velocity profiles of the actions of each affect are markedly different, with angry actions tending to be much faster on average than sad actions (Patterson, Pollick and Sanford, 2001).

A potential reason for the different results in the affect categorisation task from the previous literature is the stimuli used. Typically, studies investigating emotion categorisation from PLDs use overt emotional gestures, such as a person shaking their fists for angry, that are quite rarely seen in an everyday environment. For the present thesis we used an everyday action, throwing, performed in different affects (Ma et al. 2006). This meant that instead of the overt kinematics of the action cueing the affect, the distinction between the different affects had to be derived from subtly different kinematic properties and postures of the same action, making the task more difficult, but also more natural. It may be that by using a task that required the categorisation of affect from more subtle kinematics that are more common to everyday life, the participants in the ASD group had developed their own, perhaps cognitive, strategies to discriminate emotions from whole body movements. This was partly substantiated by self reports from participants in the ASD group that they had learned, or "taught themselves", to recognise when other people, usually family, were, for instance, angry. Similar strategies have been reported by Grandin (1999).

Overall, the results of the biological motion detection task and the categorisation of action task seem to support the findings of Moore et al. (1997), Hubert et al. (2007) and Parron et al. (2008) that people with ASDs do not have difficulty in processing biological motion. However, the slightly poorer performance in the detection task and the significantly higher variability in the ASD group on the action categorisation task, hint at a potentially subtle difference between the two groups, particularly in processing the local motions as evidenced by the variability in the action categorisation task. The results of the affect categorisation task suggest that the ASD group were not significantly different from the control group in their ability to assign affect to throwing movements. However, close inspection of the patterns of errors made by each group suggested that the ASD group may

have been using slightly different strategies to achieve the same level of performance, and that the strategy used was not down to a general velocity heuristic.

## 6.2 Understanding human form processing and the underlying neural processes in typically developed adults

In Chapter 3 a novel technique was designed that allowed for the masking of the direction of locomotion of a point-light walker, without using large numbers of noise points or temporal manipulations of the points of the walker. This technique allowed for the systematic manipulation of the amount of configural information present in a point light display, allowing for the quantification of the contribution that configural cues play in direction discrimination of a point-light walker by contrasting these stimuli with displays that contained no configural cues. The results showed that when configural cues are present, participants were substantially better at determining the direction of locomotion, being able to achieve 75% correct with five fewer points than when configural cues were absent. Furthermore, the difference in the slopes of the curves in each condition suggested that the advantage gained from configural cues was not merely a linear increase, and that each additional point placed in its biologically correct position was far more informative than a point placed in a non-biologically correct location.

As individual performance can be systematically manipulated without the need to increase or decrease the amount of local motion information in the displays, as would be the case with previous masking techniques (e.g. Thornton, Pinto, & Shiffrar 1998; Bertenthal & Pinto, 1994; Cutting, Moore & Morrison, 1988; Ikeda, Blake & Watanabe, 2005), this new technique is ideally suited for assessing the contribution of configural cues in fMRI tasks. In chapter 4 we attempted to demonstrate the technique's suitability for use in fMRI experiments and we also replicated the findings of Chapter 3. We conducted 2 fMRI experiments, the first of which was used to determine which brain regions were selectively active for processing both intact point-light displays and local biological motion signals from scrambled point-light displays. In line with previous studies using similar techniques, we found regions that were preferential for intact over scrambled stimuli in the fusiform gyrus and IPL (Downing et. al. 2006; Peelen et. al. 2006) as well as frontal and occipital regions, but found that the ITG, which is thought to be selective for form cues, was more active for biological motion irrespective of whether form cues were present in the displays (Downing et. al. 2006; Saygin et. al. 2004; Peelen et. al. 2006; Peuskin et. al. 2005). We

attributed this to the scrambling technique used, which constrained the local motions of the points to starting points consistent with a human walker, though the motion did not match the joint locations. As such, it may have been that the ITG was detecting frames from within the stimuli that were consistent with human static postures, though not the initial frames as these were specifically set to be at the point in the gait cycle that contains the least form information (Thirkettle et. al. 2009). This is plausible as Downing et al. (2006) found that, unlike the FBA, the ITG is more active when it is presented with static frames that do not depict a continuous action.

We used the regions derived from Experiment 2 to restrict the analysis in Experiment 3 to only those regions that responded to either intact PLDs or the local biological motion signals of the scrambled PLDS. Furthermore, Experiment 3 used a genetic algorithm (Wager & Nichols, 2003) to optimise the design for the contrasts of the 95% correct stimuli minus the 75% correct stimuli, the 95% correct stimuli minus the 50% correct stimuli and the 75% correct stimuli minus the 50% correct stimuli. The results showed that premotor regions, parietal regions and inferior temporal regions all showed some degree of selectivity for configural information, though only a region in the SPL seemed to be consistently activated. The premotor activations are thought to be related more to the task and complexity of the stimuli rather than the structural information present in the displays (Jastorff and Orban, 2009), though premotor areas have been implicated in the MNS and so may contain a higher level representation of the walker (e.g. di Pelligreno et. al. 1992; Gallese et. al. 1996; Rizzolatti et. al. 1996; Buccino et. al. 2004). Curiously, unlike in Experiment 2, the fusiform gyrus showed no selectivity to the amount of configural information present in the displays, which would have been predicted as it is thought to process form information (Peelen et. al. 1996; Downing et. al. 2006). Furthermore, in neither Experiment 2 nor 3, did the STS show any selectivity for configural cues, as would have been expected based on the previous literature (e.g. Grossman et. al. 2000, 2004, 2005; Saygin et. al. 2004; Beauchamp et. al. 2004; Peuskins et. al. 2003; Howard et. al. 1996; Vaina et. al. 2001; Grezes et. al. 2001).

It has been proposed that both the ITG, containing the EBA, and the fusiform gyrus, containing the FBA, process static human form (Downing et. al. 2001; Grossman & Blake 2002; Downing et. al. 2006; Peelen et. al. 2006; Peuskins et. al. 2005; Grossman et. al. 2004; Jastorff & Orban 2009) and that, whereas the FBA responds more to static frames that show a continuous human action, the EBA responds more to static frames showing

human postures from different actions and shows adaptation to consecutive frames depicting a plausibly continuous action (Downing et. al. 2006). It has been suggested however, that the STS plays more of a role in integrating features, such as form and motion (Beauchamps et. al. 2003; Peuskins et. al. 2005) and that it is predominantly active when the stimuli are complex or a task is required that makes the stimuli behaviourally relevant (Jastorff & Orban, 2009). Peuskins et. al. (2005) postulated that form information processed in the ITG may be temporally integrated with motion information in the STSp. It is possible that, as the stimuli used in the present experiments always contain some noise, then the fusiform region has difficulty matching human form across successive frames, but that the ITG is more active as it can process static postures, independent of whether they represent a biologically plausible human action. These static form cues from the ITG may then be integrated with local or global motion cues from the MTG, potentially in the STS. From here, the information may be sent to parietal regions that have been suggested to process the global percept of point-light actors (Battelli, et al. 2003), which is partly supported by the finding in the present study that parietal regions seemed to respond more to PLDs containing structural information. This potential processing strategy would be in line with a model proposed by Giese & Poggio (2003), which postulated that information from snapshot neurons would integrate with optic flow patterns in the STS from which the action could be specified. If STS is more of an integration area, it may not necessarily activate differentially to biological motion displays containing different amounts of configural information.

In order to further understand if the local network proposed above was possible, we conducted GCM analysis, which reveals patterns of effective connectivity to and from seed regions, which were derived from the contrasts in Experiment 3. However, one of the limitations of the design used, which resulted from the optimisation for contrast efficiency, was that there were not enough consecutive volumes recorded for each of the 3 stimulus types to carry out GCM separately for each condition. Therefore the GCM was carried out across the whole time course, which resulted in a complex pattern of effective connectivity that could not discriminate between stimuli with different amounts of configural information. The suggested links between the aforementioned regions were not apparent from the results, except through rather lengthy and unlikely intermediate areas.

Despite the limitations of the designs of Experiment 3, the results of both fMRI experiments seem to implicate the ITG in processing PLDs in which there is some

disruption in the human form, such as the PLDs in the scrambled condition in Experiment 2 and all the stimuli in Experiment 3. Furthermore, parietal regions seem to activate in a similar pattern to the ITG suggesting a link between the two, possibly mediated by the STSp.

## 6.3 The neural substrates of biological motion processing in ASDs

In Chapter 5 we addressed the limitations of Experiment 3 from Chapter 4, by increasing the number of trials per stimulus level and using a design compatible with conducting GCM analysis at different stimuli levels. This was done in order to more clearly understand the processing of configural cues in typically developed people and also to determine if there were any differences in the brain regions recruited to process biological motion in people with ASDs. We tested both groups on the direction discrimination task designed in Chapter 3 to determine whether people with ASDs were comparable to controls in utilising configural information in PLDs. We hypothesised, based on the literature, that there would be no significant differences between the groups (Moore et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008), but that there may be differences in the neural processes used by the two populations (Herrington et. al. 2007; Freitag et. al. 2008).

In line with the hypothesis, we found comparable performance in the behavioural tasks between the two groups, though the ASD group were slightly poorer in the configural cues present condition than the controls. From this task we derived 84% correct and 50% correct thresholds and used these in a new fMRI design for Experiment 3 that allowed GCM analysis to be carried out separately for each stimulus level. As in Chapter 4, the analysis was constrained to regions found to respond selectively to either intact PLDs or the local motions of scrambled PLDS from Experiment 2. The results of Experiment 2 showed that similar regions to those found in Chapter 4 were selective for biological motion processing in the control group, but that in the ASD group the ITG did not show any differential activity to stimuli of different types. This tentatively suggested that there may be dysfunction of the ITG in the ASD group as also found by Herrington et al. (2007).

This was confirmed in Experiment 3 of Chapter 5, as in the ASD group the ITG showed no differences in activity to the 84% correct and 50% correct stimuli. In the control group, the left IPL, precentral sulcus and the ITG all showed increased activation when viewing the

84% correct stimuli, whereas in the ASD group the right fusiform gyrus and MTG, and the left MOG showed increased activation for the same contrast. Only the left MFG was common to both groups. This suggested that the ASD group, instead of using the ITG as the initial stage of processing, used the right fusiform gyrus. Furthermore, whereas the control group utilised parietal and premotor regions, the ASD group mostly utilised temporal and occipital regions.

The results of the GCM analysis supported these findings and suggested that when processing stimuli in the 84% correct condition the initial processing stage in controls is the ITG, which influences superior parietal areas, and parts of the occipital cortex. In the ASD group however, there appear to be two processing streams, one starting in the fusiform gyrus that influences activity in the IOG, as well as the parahippocampal and lingual gyri, and another that starts in the right MTG and influences the right ITG and MOG, and the left precuneus. The activity in the ITG bilaterally show no directional influence over any other area, lending further evidence to the theory that the ITG may be dysfunctional, or may have disrupted connections with other regions, in the ASD group. Furthermore, the ITG may have preserved motion processing neurons, but disrupted functioning in form processing neurons. These two distinct streams suggest that form and motion information may be processed independently in this group. This leads to an interesting question: How could the same levels of performance be achieved by both groups when the regions used were so substantially different?

One answer may be an alternate theory to that proposed by Giese and Poggio (2003). Lange and Lappe (2006) proposed a neurally plausible theory of biological motion processing that did not necessarily require the integration of motion. They proposed that a low-level analysis of static postures, that matched learned templates of human forms, could provide information on the detection of a human PLD in noise and also the direction of locomotion through temporal matching of the static postures contained in consecutive frames. Given the results of Downing et al. (2006) that the fusiform area, in contrast to the ITG, only responds to consecutive static frames that are consistent with a continuous action, the fusiform gyrus would be the prime candidate for this type of processing. As mentioned, activity in the ITG reduces when it is presented with consecutive frames representing a continuous action (Downing et al. 2006), so it would not necessarily be directly involved in the processing system proposed by Lange and Lappe (2006). As such, it seems that the ASD group may be using a processing strategy similar to that proposed by

Lange and Lappe (2006), whereas the control participants may be using a processing style more akin to that proposed by Giese and Poggio (2003), though both systems may be available to the latter group. This may be how the ASD group can reliably discriminate the direction of locomotion of PLDs and detect biological motion in noise, as in Experiment 1 of chapter 2, without recruiting superior temporal or parietal regions, as there is no need to integrate the motion signals with the form cues.

## 6.4 Different biological motion processing systems in ASDs

It appears that adults with ASDs extract relevant information from PLDs using different brain regions than do typically developed people. A major question would be "how do they come to use these regions?". One clue may be the findings of Annaz et. al. (2009) that there is a developmental delay in processing biological motion in people with ASDs. Klin et. al. (2003), Klin and Jones (2008) and Klin et. al. (2009) have repeatedly shown that very young children with ASDs show no preference for canonical displays of human movements, suggesting that biological motion processing is impaired from an early age in people with ASDs. Furthermore, Annaz et. al. (2009) found that between the ages of 5 & 12, children with ASDs show a flat developmental trajectory in differentiating intact from scrambled PLDs, unlike controls who showed continued improvement between the same ages. Studies involving participants into adulthood have, however, found no differences between people with ASDs and controls in tasks involving detection of biological motion and action categorisation (Moore et. al. 1997; Hubert et. al. 2007; Parron et. al. 2008), as has the present thesis. It may be that by adulthood the ASD group has recruited regions in the fusiform gyrus to derive relevant information from biological motion, that would normally be processed through the ITG, and that the developmental delay is due to the brain rewiring to accommodate this shift in processing and the additional demands placed on these regions.

It should be possible to explicitly test this idea. One way would be to test children in a range of biological motion tasks in a longitudinal fashion, testing their sensitivities to both biological motion and form cues. Alternatively, studies of functional and anatomical connectivity from the ITG and fusiform gyrus may reveal weaker connections between the ITG and temporal or parietal regions. Furthermore, clever manipulations of experimental stimuli may be able to devise tasks that separate form from motion that can confirm or disprove whether people with ASDs do use different processing strategies. For instance,

limiting the lifetime of the points in a PLD, though not particularly damaging in performance terms for controls (Beintema & Lappe, 2002; Mather, Radford, & West, 1992; Neri, Morrone, & Burr, 1998; Pinto & Shiffrar, 1999), should render any template matching technique useless and as such, this specific type of stimulus may substantially reduce the ability of people with ASDs to detect the direction of locomotion of a point light walker, particularly at short dot lifetimes. Furthermore, temporal manipulations of the joints, such as used by Blake et al. (2003), would also limit how well the ASD group could match the concurrent frames with template actions. Grossman et al. (2005) showed that repetitive TMS over STSp disrupted biological motion processing. If STSp is an integration region for form and motion, then this may be why disruption of its function disrupts biological motion perception in typically developed individual. However, if people with ASDs utilise a temporal template matching technique that does not require integration of these two cues, then disruption of the STSp may not affect this population to the same extent. Using these techniques combined with finer temporal sampling rates in an fMRI environment, future studies will be able to look at how these regions process form and motion cues in much more detail, both in people with ASD and in typically developed controls.

## 6.5 Limitations of the thesis

It must be acknowledged that there are some limitations to the conclusions of the current thesis. The first, and most salient, is that despite the best efforts of the researchers involved, there was no single measure of severity of the symptoms across participants and as such, symptom severity could not be correlated with measures of performance. This is a common feature of studies in which the population is recruited from different clinical practices and geographical locations, as the diagnostic tests administered can vary considerably. It may be that as the severity of symptoms in childhood increases, the potential for neural rewiring reduces. Typically, studies rely on relatively high-functioning participants for experiments, particularly those using fMRI. Determining to what extent there is dysfunction in people with more severe symptoms, and even younger children, is a far more difficult challenge but may be necessary to examine the extent to which low-level dysfunctions are typical of the disorders.

Another factor may be the types of symptoms that people with ASDs exhibit. Those with high systematizing traits seem to show a different pattern of sensitivities to visual illusions,

as do those with block design peaks (Caron et. al. 2006), and perhaps exhibit a more local processing bias (Walter et al. 2009). This has not yet been investigated in motion processing or biological motion processing, but it may be a factor and as such, future studies should perhaps characterise their population more carefully than has been done in the present thesis, on a wider range of measures than symptom severity and IQ.

Secondly, as there was no form-from-motion control stimuli used, it is not possible to exclude the possibility that there may simply be a motion coherence processing deficit (e.g. Pellicano et. al. 2005; Herrington et. al. 2007; Atkinson 2009). A motion processing deficit could account for the differences seen between the groups, particularly if the STS region integrated form cues with motion cues. If motion coherence processing was impaired, then it may not be possible to integrate form cues with motion cues, which may force a reliance on the temporal analyses of static form cues. This would however, still not explain why the ITG did not respond to different levels of configural information in the stimuli.

## 6.6 Conclusions

The present thesis has demonstrated that adults with ASDs are comparable with controls in a range of biological motion tasks, including the detection of a coherent point-light walker in masking noise, direction discrimination of point-light walkers in which configural cues are diminished and categorising actions and affect from PLDs. Although previous research has shown people with ASDs to have difficulty in categorising affect from PLDs, we speculate that the people in the present study were able to use learned top down strategies to achieve comparable performance to controls. We also found that despite equivalent behavioural performance, adults with ASDs utilise a different network of brain regions to age and FSIQ matched controls, and propose that the network used by people with ASDs involves a template matching of static form cues that is processed independently of motion cues, whereas TD adults used a process that integrates form cues with local or global motion information. We speculate that these differences arise due to an early dysfunction of inferotemporal regions, or disrupted connections between these regions and more parietal areas, that place additional demands on form processing areas in the fusiform gyrus, and that the developmental delay in processing biological motion processing seen in people with ASDs, is a result of neural rewiring to accommodate the additional demands placed on these regions.

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## Appendix 1: Relevant publications and abstracts from this thesis

McKay L. S., Simmons D., McAleer P. & Pollick F, E. (2009) Contribution of Configuration information in a direction discrimination task: Evidence using a novel masking paradigm. *Vision Research,* **49(20)**, 2503-2508.

Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P. & Pollick, F. E. (2009) Vision in Autism Spectrum Disorders. *Vision Research.***49(22)**, 2705-2739.

McKay L. S., McAleer P., Simmons D. & Pollick, F. (2009) Investigating the Action Understanding Circuit using Novel Point-Light Stimuli. [Abstract]. *Human Brain Mapping – Annual Meeting. San Francisco, June 17th – June 23rd.*

McKay L. S. , McAleer P., Simmons D., Brennan D., Piggot J. & Pollick F. E. (2009) ). Adults with autism spectrum diagnoses show different patterns of BOLD activity in response to displays of point-light biological motion. [Abstract]. *Scottish Imaging Network – A Platform for Scientific Excellence - Annual Meeting. Edinburgh, 17th June.*

McKay, L., McAleer, P., Simmons, D. & Pollick, F. E. (2008) A Novel Technique for Quantifying the Contribution of form Information in Point Light Displays. [Abstract]. *7th Annual International Meeting for Cognitive Science, Seoul, July 26th-28th.*

McKay, L., McAleer, P., Simmons, D., & Pollick, F. (2008). Evidence of atypical processing of biological motion in Autistic Spectrum Disorders. [Abstract]. *International Meeting for Autism Research, London, May 15-17th.*

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