Facial Motion Perception in Autism Spectrum Disorder and Neurotypical Controls

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DECLARATION

I hereby declare that this thesis has not been, and will not be submitted, in whole or in part to another University for the award of any other degree. Some of the studies (Chapters 2 and 4) or results (Chapters 3 and 5) presented in this thesis have been published in the following journals:

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CONTENTS

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ABSTRACT

Facial motion provides an abundance of information necessary for mediating social communication. Emotional expressions, head rotations and eye-gaze patterns allow us to extract categorical and qualitative information from others (Blake & Shiffrar, 2007). Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterised by a severe impairment in social cognition. One of the causes may be related to a fundamental deficit in perceiving human movement (Herrington *et al.,* (2007). This hypothesis was investigated more closely within the current thesis.

In neurotypical controls, the visual processing of facial motion was analysed via EEG alpha waves. Participants were tested on their ability to discriminate between successive animations (exhibiting rigid and nonrigid motion). The appearance of the stimuli remained constant over trials, meaning decisions were based solely on differential movement patterns. The parieto-occipital region was specifically selective to upright facial motion while the occipital cortex responded similarly to natural and manipulated faces. Over both regions, a distinct pattern of activity in response to upright faces was characterised by a transient decrease and subsequent increase in neural processing (Girges *et al.,* 2014). These results were further supported by an fMRI study which showed sensitivity of the superior temporal sulcus (STS) to perceived facial movements relative to inanimate and animate stimuli.

The ability to process information from dynamic faces was assessed in ASD. Participants were asked to recognise different sequences, unfamiliar identities and genders from facial motion captures. Stimuli were presented upright and inverted in order to assess configural processing. Relative to the controls, participants with ASD were significantly impaired on all three tasks and failed to show an inversion effect (O'Brien *et al.,* 2014). Functional neuroimaging revealed atypical activities in the visual cortex, STS and fronto-parietal regions thought to contain mirror neurons in participants with ASD. These results point to a deficit in the visual processing of facial motion, which in turn may partly cause social communicative impairments in ASD.

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ABBREVIATIONS

ASD	Autism Spectrum Disorders
AS	Asperger's Syndrome
CGI	Computer-generated imagery
fMRI	Functional magnetic resonance imaging
ROI	Regions of interest
RFX	Random effects analysis
EEG	Electroencephalography
ERP	Event-related potential
TMS	Transcranial magnetic stimulation
MEG	Magnetoencephalography
PET	Positron emission tomography
FG	Fusiform gyrus
FFA	Fusiform face area
OFA	Occipital face area
STS	Superior temporal sulcus
MNS	Mirror neuron system
IFG	Inferior frontal gyrus
IPL	Inferior parietal lobule
MTG	Middle temporal gyrus
mPFC	Medial prefrontal cortex
EBA	Extrastriate body area
PPA	Parahippocampal place area
OPA	Occipital place area
LOC	Lateral occipital complex
dPMC	Dorsal premotor cortex

CHAPTER 1 Literature Review

1.1 Overview

Human interaction is guided by the ability to correctly perceive and interpret the behaviours of others. Many, if not all social cues are dynamic in nature. Indeed, facial expressions, eye-gaze shifts, hand gestures and body language are all driven by distinct movement patterns. An inability to perceive these actions could lead to atypical social functioning (Pavlova, 2012). Autism Spectrum Disorder (ASD) is characterised by a profound impairment in social interaction, communication and imagination. Many theories have been developed regarding the aetiology of such symptoms. The most prominent accounts suggest a deficit in understanding mental states (Baron-Cohen *et al.,* 1985) or a cognitive discrepancy when integrating information at different levels (Frith, 1989). While these theories are well established, there is a possibility that poor social cognition might actually arise, in part, from a fundamental deficit in perceiving human movement (Herrington *et al.,* 2007). The broad aim of this thesis is to investigate biological motion perception in ASD, placing emphasis on dynamic faces.

The first part of this chapter will outline the symptoms associated with ASD. As this thesis is based on understanding social cognition in ASD, a review of the current literature discussing mentalising, emotion recognition and face processing from static representations will be presented. The last sections of this chapter will focus on the processing of dynamic stimuli (inanimate and biological). Each part will include neuroimaging and behavioural data collected from participants with ASD and neurotypical controls.

1.2 Autism Spectrum Disorder (ASD)

1.2.1 Symptoms

ASD is a pervasive condition characterised by abnormal brain development and profound behavioural manifestations (Sasson, 2006). Participants with ASD exhibit atypical reactivity to sensory inputs, ritualised behaviours, inflexible adherence to routines, highly restricted interests and repetitive motor gestures or speech (echolalia) (Bishop *et al.,* 2013; Lam & Aman, 2007; Stribling *et al.,* 2007). Deficits in social interaction and communication are also routinely present. Participants display difficulties in executing and understanding nonverbal communication, maintaining relationships and reciprocating socio-emotional interaction (Harrop *et al.,* 2015; Mehling & Tassé, 2014; Nadler, 2014).

Despite core symptomology, ASD is extremely heterogeneous in terms of severity and associated impairments (Bennett *et al.,* 2014). Participants with ASD are often co-morbid for other psychiatric disorders such as anxiety, depression, alexithymia and ADHD (Bird *et al.,* 2010; Matson & Williams, 2014; Mayes *et al.,* 2012; van Steensel *et al.,* 2013). Lower functioning individuals also present with

below average intelligence on tasks assessing verbal and nonverbal cognitive reasoning (Chiang *et al.,* 2014). Delayed language acquisition and poor verbal ability is further evident within this subgroup of participants (Bennett *et al*., 2014). Indeed, approximately 25-50% of diagnosed individuals never attain functional language (Klinger *et al.,* 2002). Difficulties in joint attention or atypical sensitivity to non-semantic information in speech may underlie these impairments (e.g., Heaton *et al.,* 2008). Furthermore, the deficit in receptive and expressive language appears to correlate with impoverished social development, particularly in reference to understanding the mental states of others (Anderson *et al.,* 2009) and orienting to socially relevant stimuli. For example, Stagg *et al.,* (2014) found that high-functioning children with ASD who exhibit language delays spend significantly less time observing interacting human figures, relative to neurotypical controls or verbally competent children with ASD. These findings therefore suggest that poor linguistic expertise is a secondary consequence of poor social attention and cognition. They also demonstrate the highly variable phenotypic quality of ASD.

1.2.2 Onset and Prevalence

In the majority of cases, these behavioural disturbances emerge early in development (Pellicano, 2007). Typical onset occurs by 3 years of age, although symptoms can be recognised much earlier in some individuals (Ozonoff *et al.,* 2008). For example, impairments in joint attention or communication have been noted in 12-month-old infants who later receive a diagnosis of ASD (Maestro *et al.,* 2005). Instances of regressive ASD have also been observed (Stefanatos *et al.,* 2002). This is where a child develops typically until 2 years of age, at which point they appear to lose previously acquired skills relating to language and/or social functioning (Ozonoff *et al.,* 2005; Ozonoff *et al.,* 2008). This unique pattern of onset could represent a distinct subtype of ASD (Goin-Kochel *et al.,* 2014).

ASD is more prevalent in males than females, with a ratio of 4:1 (Goldman, 2013). While the factors surrounding this bias remain unclear, it has been proposed that ASD reflects an extreme expression of male brain morphology (Baron-Cohen *et al.,* 2011). Supporting this prediction is the finding that neurotypical males have larger brains than neurotypical females (Ruigrok *et al.,* 2014), whilst participants with ASD have larger brains than both sexes (Courchesne *et al.,* 2010). A post-mortem study also reported that male children with ASD had 67% more neurons in the prefrontal cortex than neurotypical males (Courchesne *et al.,* 2011). Such hypermasculinisation has been explained in terms of excessive exposure to foetal androgens (Baron-Cohen *et al.,* 2011). Reports of increased testosterone levels in amniotic fluid and its correlation to poor empathising skills in childhood corroborate this hypothesis (Knickmeyer *et al.,* 2006). Androgen irregularities appear to persist into adulthood too. Bejerot *et al.,* (2012) observed higher total and bioactive testosterone levels in females with ASD relative to neurotypical females.

The 'Male Brain' theory of ASD may have clear implications regarding the aetiology of social communicative impairments (Baron-Cohen, 2009). It has been previously shown that female participants excel at emotion recognition and empathy judgments (Kret & De Gelder, 2012). Males, however, outperform on tasks involving mental rotation, systemizing and visual navigation (Baron-Cohen, 2002; Cook & Saucier, 2010). This sexual dimorphism is supported by specific neurofunctional variations. Empathic social cognition engages the inferior frontal gyrus and superior temporal sulcus more in females than males (Schulte-Rüther *et al.,* 2008). Female participants also show greater activation of the inferior frontal cortex when asked to infer the mental states of others from their eye movements (Derntl *et al.,* 2010). In participants with ASD however, evaluating the approachability or intelligence of another person evokes considerably less activity in these aforementioned social brain structures relative to neurotypical males, who in turn evoke less activity than females (Hall *et al.,* 2012). These data hint towards an exaggeration of normal male brain properties in ASD which may contribute to atypical social functioning.

1.2.3 Interim Summary 1

This section has provided a brief overview of the symptoms observed in ASD, including information regarding the development of expressive and receptive language, intellectual ability and related psychiatric challenges. We also discuss the onset and prevalence of this disorder. Further, the role of *in utero* exposure to androgens and its apparent hypermasculinising effect on the brain has been outlined, particularly in relation to socio-emotional perception. The next part of this chapter will now focus on what aspects of social cognition are actually impaired in participants with ASD.

1.3 The 'Social Network'

Humans are fundamentally social agents (Puce & Perrett, 2003). Imperative to survival is the ability to correctly infer what others are thinking or intending, recognising emotions and identifying faces (Stekelenburg & de Gelder, 2004). These functions are severely weakened in ASD (Nuske *et al.,* 2013). This section will discuss: (1) theory of mind; (2) emotion recognition; and (3) face processing.

1.3.1 Theory of Mind

Theory of mind refers to the phenomenon of being able to understand that other people have feelings and beliefs which may differ from one's own (Premack & Woodruff, 1978). It is traditionally tested using first-order false belief tasks. This is where a participant observes an object being relocated during the absence of an actor and are then asked where they will look for the object. The task rationale is that if a participant has a 'theory of mind', they will reason that the actor will look in the original location (Senju, 2012). Second-order false belief tasks are also used in theory of mind research and require inferences about someone's thoughts which are in turn about another person's mental state (e.g., X

believes that Y thinks). In typical development, children between 4 and 5 years of age perform well on these tests, indicating an intact theory of mind (Wimmer & Perner, 1983).

Extensive testing of theory of mind in ASD has shown poor mentalising skills to be a core deficit within this population (Heavey *et al.,* 2000; Leslie & Frith, 1988; Roeyers *et al.,* 2001; Zelazo *et al.,* 2002). Children with ASD perform significantly worse than neurotypical controls on both first and second order false belief tasks (Ozonoff *et al.,* 1991). The impairment is also evident when children with ASD are required to state their own prior false belief (Russell & Hill, 2001). In one study, children were shown a Smarties tube and asked what they thought was inside it (Williams & Happé 2009). While all participants responded "chocolate", opening the container actually revealed a pencil. They were then asked what they thought was inside the tube *before* the experimenter had opened it. Unlike neurotypical children, participants with ASD did not reason that they had initially assumed chocolates and instead reported that they thought a pencil was inside. These findings suggest that participants with ASD are unable to represent their own and other mental states. In turn, this would significantly impact their ability to interact with their peers (Colle *et al.,* 2007).

Hypoactivation of the medial prefrontal cortex (mPFC) and temporoparietal junction, as well as reduced connectivity between these regions, has been associated with poor mentalising skills in ASD (Bernhardt *et al*., 2013; Castelli *et al.,* 2002; Lombardo *et al.,* 2009; Lombardo *et al.,* 2011). O'Nions *et al.,* (2014) required participants to select the correct ending of a cartoon story by understanding behaviours based on intentions. The authors reported that children with ASD engaged the mPFC less than children with conduct problems and callous-unemotional traits. Other researchers have linked these regions with the mirror neuron system (MNS) - a network which maps both observed and executed goal-directed actions (Rizzolatti & Craighero, 2004). Kana *et al.,* (2014) documented a weakened connectivity between the ventral premotor cortex (i.e. MNS) and temporoparietal junction in participants with ASD relative to controls during intentional causal attribution. It is possible, therefore, that abnormalities within the MNS and its pathways to 'theory of mind regions' cause disruptions in deciphering intentions and understanding behaviours in ASD.

By contrast, there are studies which have failed to report theory of mind deficits in ASD. Begeer *et al.,* (2010) asked participants to retell a narrative about a social interaction and to participate in a communication game. The latter task required another person's perspective to be taken into account when interpreting what they said. While participants with ASD used fewer terms of belief, desire and emotion when retelling a story, they performed identically to neurotypical controls in the communication game. It is therefore possible that in some instances of social interaction, participants with ASD appear to demonstrate an intact theory of mind. This has been further verified in studies which utilise structured social interaction paradigms (Begeer *et al.,* 2007; Ponnet *et al.,* 2005).

It is possible that theory of mind impairments may correlate with intellectual aptitude. For example, children with ASD who exhibited verbal skills within the average range performed at ceiling on first-order false belief tasks (Fisher *et al.,* 2005; Happé, 1995). High-functioning children with ASD (IQ > 70) also showed similar abilities to controls when presented with social stories containing second-order false beliefs, display rules, double bluff, faux pas' and sarcasm (Scheeren *et al.,* 2013). Not all studies, however, reliably find verbal skill to be a predictor of such impairment. Senju *et al.,* (2009) reported that participants with Asperger's Syndrome, who experience no delays in language development, still do not pass spontaneous theory of mind tasks. Participants with ASD also show impairments on nonverbal tests such as mind-reading from eye information (Baron-Cohen *et al.,* 2001; Kaland *et al.,* 2008), indicating that language development cannot entirely explain these inconsistent findings.

Perhaps discrepant data is simply reflecting task complexity, although Sodian and Frith (1992) suggest impairments occur independently of this factor. Alternatively, mind-blindness could be caused by a poor recall of episodic autobiographical memories which require autonoetic consciousness (Kirsten *et al.,* 2014). Yet this claim only explains deficits in understanding own mental states. Instead, mind-blindness may be related to a problem in executive functioning and cognitive shifting - i.e. from one's own perspective to another's (Pellicano, 2007). In support of this hypothesis, Joseph and Tager-Flusberg (2004) reported significant correlations between mentalising, working memory and inhibitory control abilities in children with ASD. Pellicano (2013) further highlighted that such associations occur autonomously from age, language ability and nonverbal intelligence.

1.3.2 Emotion Processing

Neurotypical Controls

Appropriate social interaction is dependent on the ability to correctly identify and understand emotions (Frischen *et al.,* 2008). Neurotypical adults perform exceptionally well in emotion recognition tasks and are highly sensitive to contextual negative expressions. For example, detection of angry faces occurs much faster and accurately than happy faces when presented in a crowd (Hansen & Hansen, 1988). Higher attentional re-orienting or automatic processing is therefore associated with threatening stimuli. These findings are consistent with more recent data (Fox *et al.,* 2000; Öhman *et al.,* 2001; Maratos, 2011) and have been replicated with fearful expressions as well (Eastwood *et al.,* 2001). In opposition, other researchers report a non-existent pop out effect for angry/threatening faces, but instead a search bias for happy expressions (Becker *et al.,* 2011; Hunt *et al.,* 2007). This result is not entirely unanticipated as happy affects are recognised the fastest when presented in isolation (Kirita & Endo, 1995). Additionally, daily familiarity with positive expressions has been thought to aid cognitive priming

(Pinkham *et al.,* 2010). Despite these somewhat ambivalent results, it can be concluded that neurotypical controls are incredibly perceptive to many types of facial affects.

There are several substrates underlying emotion processing. The medial prefrontal cortex (mPFC) is involved in general emotion discrimination (Steele & Lawrie, 2004), developing negative emotions or impressions (Iidaka *et al.,* 2011) and regulating internal emotional states (Glotzbach *et al.,* 2011). It also contains different neural representations for different emotions. In one transcranial magnetic stimulation (TMS) study, participants discriminated angry and happy faces that were preceded either by a congruent or incongruent word prime. Temporarily disrupting the mPFC significantly affected the priming effect by selectively increasing response latencies during congruent trials (Mattavelli *et al.,* 2011).

The subcortical amygdala also mediates negative emotion processing (Corbett *et al.,* 2009; Lerner *et al.,* 2013). Patients with bilateral amygdala lesions are impaired on tasks recognising fearful expressions but are still able to discriminate other emotions (Adolphs *et al.,* 2005; Phelps & LeDoux, 2005). Such findings have led many authors to conclude that the amygdala is central to fear conditioning and threat detection (Feinstein *et al.,* 2011). There is evidence to challenge this view though. Gallagher and Chiba (1996) and Fossati (2012) report that its' function is in comprehending and evaluating the intensity of all emotions, in addition to regulating internal emotional states.

Two other substrates appear to contain emotion recognition properties. The insula cortex shows sensitivity to facial expressions of disgust (Krolak-Salmon *et al.,* 2003; Sambataro *et al.,* 2006). Viewing disgust-evoking images such as mutilation or recalling autobiographical events that were disgusting further engages this substrate (Fitzgerald *et al.,* 2004; Wright *et al.,* 2004). The anterior cingulate cortex is also relevant for emotion regulation and forming or expressing a negative impression from facial stimuli (Iidaka *et al.,* 2011). Collectively, the abovementioned findings indicate a network of fronto-limbic substrates involved in the recognition of emotional expressions. It is possible that any dysfunction to these regions (or insufficient connectivity between them) could cause deficits in emotion understanding and therefore social interaction.

ASD Populations

Emotion processing has been substantially investigated in ASD, but the current results are varied (Nuske *et al.,* 2013). Tracy *et al.,* (2011) did not observe any differences in the ability of children with ASD or neurotypical development to identify basic emotions, including those with socially complex elements. Tanaka *et al.,* (2012) also found that neurotypical controls and participants with ASD performed equally as well when matching a word label to the correct emotional expression. In addition, Cook *et al.,* (2014) failed to report any group differences when investigating the facial aftereffect

illusion (e.g. neutral faces taking on a happy expression after prolonged exposure to sad emotions). Similar findings have been reported elsewhere (Bernier *et al.,* 2005; Castelli, 2005; Song *et al.,* 2012).

On the other hand, many publications do report deficits in recognising facial and vocal emotional expressions in ASD (Ashwin *et al.,* 2006; Clark *et al.,* 2008; Philip *et al.,* 2010; Wallace *et al.,* 2008). Adults with ASD perform poorly when labelling both basic and socially complex emotions such as trustworthiness or jealousy (Bölte & Poustka, 2003; Rump *et al.,* 2009). They also misinterpret the intensity of facial expressions and consistently perceive ambiguous stimuli to be a negative affect (Kennedy & Adolphs, 2012; Kuusikko *et al.,* 2009). Other researchers suggest that the emotion recognition deficits in participants with ASD only occurs for low intensity presentations, indicating that it is the subtlety of facial expressions which cause profound impairments (Law Smith *et al.,* 2010).

Participants with ASD are also poor when matching a vocally expressed affect to static facial expressions or to emotion words (Boucher *et al.,* 1998; Stewart *et al.,* 2013). Heaton *et al.,* (2012) similarly reports that adults with ASD commit significantly more errors on tasks discriminating emotions from sounds or speech compared to controls. Interestingly, the authors observed strong associations between the severity of recognition deficits and co-morbidity for alexithymia. Alexithymia is a disorder characterised by difficulties in describing or distinguishing feelings from emotional arousal and is reported to occur in approximately 50% of participants with ASD (Lombardo *et al.,* 2007). Accordingly, it has been theorised that high levels of alexithymia, rather than ASD symptomology *per se*, predicts emotion recognition deficits in some individuals (Bird *et al.,* 2010). If this is the case, then it may explain why discrepant findings are often found within the ASD literature (Cook *et al.,* 2013).

Nonetheless, examining the timing of neural activity provides additional evidence for atypical emotion processing in ASD (Lartseva *et al.,* 2014). In neurotypical participants, the event-related N170 indexes early configural face processing (Bentin *et al.,* 1996) and emotional recognition (Blau *et al.,* 2007). Studies conducted with ASD adults reveal smaller N170 amplitudes in response to facial emotions (O'Connor *et al.,* 2005) and to fearful expressions relative to controls (Dawson *et al.,* 2004; Wagner *et al.,* 2013). Accordingly, these findings suggest that the deficits in emotion recognition stem from a dysfunction in the earliest stage of perception (Lerner *et al.,* 2013).

In addition, several volumetric studies have revealed structural abnormalities of the amygdala in participants with ASD (Aylward *et al.,* 1999; Schumann *et al.,* 2009). Children with ASD show bilateral amygdala enlargements (Schumann *et al.,* 2004) which appear to correlate with core symptom development and poorer clinical outcomes (Munson *et al.,* 2006; Sparks *et al.,* 2002). However, smaller volumes have been noted in adults, signifying that structural abnormalities vary with chronological age (Corbett, 2009). In support of this assumption, Courchesne (2004) reported that the

abnormal overgrowth of limbic and cerebral structures is followed by a premature arrest of neurodevelopment during the most critical stages of cerebral circuitry formation.

Further, fMRI studies have demonstrated atypical engagement of the amygdala during emotion recognition in ASD (Schultz, 2005; Weng *et al.,* 2011). In a matching and labelling paradigm, children with ASD recruited different neural networks despite being relatively unimpaired on the cognitive assessment of basic emotions (Wang *et al.,* 2004). Similar findings have also been observed in adult populations. A study with ASD males reported reduced activity within the amygdala and orbital frontal cortex during social perception tasks (Ashwin *et al,* 2006). In addition, adults with ASD do not activate differential patterns of activity when viewing fearful and neutral static bodies (Hadjikhani *et al.,* 2009). Controls, however, strongly activate the amygdala, nucleus accumbens and anterior insula during fear perception (Hadjikhani *et al.,* 2009). Less involvement of these key regions could lead to participants with ASD assigning inappropriate significance to emotions, which would then have a detrimental effect on social interaction.

1.3.3 Face Recognition

Background Information

Faces provide a wealth of visual information necessary for social communication. Consequently, face perception is a highly developed skill in humans (Haxby *et al.,* 2000). People can identify thousands of faces despite their similarity as visual objects. This is achieved by detecting and comparing the invariant aspects of facial features (Renzi *et al.,* 2014). Faces are typically processed holistically (unified gestalt) or configurally rather than in a part-based manner (Farah *et al.,* 1998). This effect has been demonstrated by inversion and composite paradigms which disrupt the discrimination of faces in neurotypical participants (Young *et al.,* 1987; Tanaka *et al.,* 2014).

The mechanisms which mediate face perception form a distributed network within the occipitotemporal extrastriate cortex. In a seminal PET study, Sergent *et al.,* (1992) first identified a region in the right fusiform gyrus (FG) which appeared active during face recognition. Haxby *et al.,* (1994) conducted a similar investigation, reporting greater FG activity for judgments of faces presented in different angles relative to judgments of objects. Functional MRI studies which compare the perception of faces with non-face stimuli also observe strong engagement of the FG, specifically in a region now called the fusiform face area (FFA; Kanwisher *et al.,* 1997). These data thus highlight the specificity of the FG/FFA in detecting and identifying faces.

The occipital face area (OFA) within the inferior occipital gyrus and the superior temporal sulcus (STS) also possess face-selective properties. Hierarchical feedforward models posit that the OFA is an early

module within the network, performing simple face detection tasks (Pitcher *et al.,* 2011). Information is then transmitted to the FFA or STS for more complex processing. Other researchers, however, implicate the OFA in analysing different face properties, including those related to facial identity and expression (Kadosh *et al.,* 2011). The OFA therefore receives both feedforward and re-entrant feedback from the FFA, meaning it has a role in the initial detection and integrative analysis of faces (DeGutis *et al.,* 2007; Rotshtein *et al.,* 2007; Solomon-Harris *et al.,* 2013). The STS on the other hand is not involved in the recognition of faces *per se*, but is attentive to changes in eye-gaze and variant facial aspects (Iidaka, 2014). It is also involved in predicting and detecting goal-directed social actions (Allison *et al.,* 2000). The STS has subsequently been heavily implicated in biological motion, which will be discussed in later sections.

ASD Populations

Several behavioural investigations have reported face processing deficits in ASD populations. The impairment is evident by approximately 18 months of age and affects most aspects of recognition (Dawson *et al.,* 2012). Indeed, children with ASD have trouble distinguishing familiar from unfamiliar faces (Boucher & Lewis, 1992). Studies conducted with adult samples further indicates that participants with ASD also experience difficulties with lip reading, detecting eye-gaze directions and discriminating genders from faces (Bradshaw *et al.,* 2011; Chawarska & Shic, 2009; Deruelle *et al.,* 2004). Deficits in facial emotion recognition have further been reported, with participants needing a more intense emotional expression for accurate perception (e.g., Poljac *et al.,* 2013).

Reduced interest most likely leads to less experience with faces, therefore causing face perception deficits (Schultz, 2005). Indeed, participants with ASD do not spontaneously attend to faces and spend less time looking at them compared to controls (Chawarska *et al.,* 2013; Ishii & Konno, 1987). Yet there is evidence of ASD samples actually looking significantly longer at facial stimuli relative to neurotypical participants (Elsabbagh *et al.,* 2013a; Webb *et al.,* 2010). This has also been correlated to a poorer perception of static faces (de Klerk *et al.,* 2014). It is possible that participants with ASD are attending to faces, but favour irrelevant or uninformative features (Speer *et al.,* 2007). For example, it has been shown that participants with ASD appear to focus more on non-feature areas such as the chin or hairline (Pelphrey *et al.,* 2002). Other researchers, however, report that these participants focus heavily on mouth movements and find this region to be the most helpful for facial identification (Joseph & Tanaka, 2003; Wolf *et al.,* 2008). Not only does this finding indicate a level of preservation in lip-reading abilities, but also that participants with ASD are seeking a more verbal way of understanding social cues (Sawyer *et al.,* 2012; Wilson *et al.,* 2012).

Face recognition deficits may also arise from an inability to process the spatial relations between face parts or perceive the unified gestalt (Gauthier *et al.,* 2009; Richler & Gauthier, 2014). Inversion

paradigms negatively affect face recognition in neurotypical participants by disrupting the configural percept (Itier & Taylor, 2002). As this disruption is less pronounced in participants with ASD, it has been taken as evidence for a featural or part-based processing style which is less effective for recognition (Langdell, 1978; Rose *et al.,* 2007; Tantam *et al.,* 1989). It is important to note though that some studies do observe an inversion effect to some degree in participants with ASD (Bar-Haim *et al.,* 2006; Nishimura *et al.,* 2008; Riby *et al.,* 2009). For example, Joseph and Tanaka (2003) reported that children with ASD were impaired when asked to identify faces from inverted mouths, but not from inverted eyes. This finding again indicates a deficit in processing eye information which may lead to atypical face processing in ASD (Weigelt *et al.,* 2012).

In support of these behavioural investigations, electrophysiological studies have reported abnormal occipitotemporal activity in participants with ASD. Dawson *et al.,* (2002) found that children with ASD do not show differential N170 responses to familiar and unfamiliar faces relative to controls. Similarly, McPartland, *et al.,* (2004) reported that adults with ASD had slower N170 latencies to faces than objects, compared to neurotypical controls. Khorrami *et al.,* (2013) also observed differences in the N170 latency between adults with ASD and neurotypical controls. These results have been interpreted as reflecting an impaired configural processing (O'Connor *et al.,* 2005).

Similarly, fMRI studies report hypoactivity in components of the face processing network, including the amygdala (Bookheimer *et al.,* 2008; Perlman *et al.,* 2011) and regions of the FG/FFA in participants with ASD (Corbett *et al.,* 2009; Hubl *et al.,* 2003; Nickl-Jockschat *et al.,* 2014; Piggot *et al.,* 2004; Wang *et al.,* 2004). Instead, the experimental group strongly activate regions involved in object processing (Schultz *et al.,* 2000). Interestingly, Kleinhans *et al.,* (2008) failed to observe any abnormality of the FG in participants with ASD. Perhaps it is irregular connectivity between regions of the social brain, rather than a deficit to a specific substrate, which causes face processing difficulties (Apicella *et al.,* 2012). Indeed, Bird *et al.,* (2006) suggests that the lack of attentional modulation to facial stimuli in ASD is caused by a poor connectivity between V1 and the extrastriate cortex. However, atypical eye-gaze patterns provide a crucial bias and may be a source of discrepancy within the literature. In this context, studies which instruct participants with ASD to focus on the eye region document normal FG activity (Hadjikhani *et al.,* 2004; Hadjikhani *et al.,* 2007).

Attention levels (Wojciulik *et al.,* 1998), emotional valence (Vuilleumier *et al.,* 2001) and the familiarity of facial stimuli could also contribute to these inconsistent findings. Typical FG activity is observed when participants with ASD view facial images of familiar family members relative to unfamiliar faces (Pierce *et al.,* 2004). Similar results have been found when participants with ASD view stimuli related to their special interest (Pierce & Courchesne, 2000). Indeed, more activation was observed in the middle FG and amygdala when an autistic child viewed their favourite cartoon compared to unfamiliar

faces (Grelotti *et al.,* 2005). These findings therefore indicate that face perception in ASD is preserved to some degree and depends on a number of factors relating to experimental design.

1.3.4 Interim Summary 2

Behavioural and neuroimaging studies have reported a weakened social network in ASD. Face perception, emotion recognition and understanding the beliefs of others are impaired in this disorder. Unfortunately, many of these investigations are limited by their common use of static stimuli. These do not depict real life, or how real people convey social information. Face-to-face interaction is dynamic in nature. For instance, understanding someone's emotional state is facilitated by perceiving how their face moves into a series of expressions. The next section of this review will focus on research which has utilised dynamic displays. There is strong evidence to suggest that difficulties in social cognition are related to difficulties in perceiving motion (Herrington *et al.,* 2007). Mechanical motion processing in ASD will be considered first, before discussing biological motion and its relation to social cognition.

1.4 Coherent Motion Perception

Within the last decade, there has been a substantial focus on how participants with ASD perceive moving stimuli (Kaiser & Shiffrar, 2009). The majority of these investigations utilise coherency paradigms such as the global dot motion task (e.g., Spencer *et al.,* 2000). This is where a portion of dots move across the computer screen in the same direction (i.e., coherently), while the rest move (pseudo) randomly. The participant is required to state the direction of the coherently moving dots.

Spencer *et al.,* (2000) investigated motion perception in ASD by observing the ability to integrate local motion signals over space. They found that motion coherence thresholds were on average 45.6% higher in children with ASD than neurotypical controls. This finding was later replicated by Pellicano *et al.,* (2005). Children with ASD needed 22.40% of the dots to move coherently in order to detect the direction of motion, compared to only 11.10% for controls. Similar results have been found when presenting the experimental group with random dot kinematograms for varied durations (Davis *et al.,* 2006) or with glass patterns (Spencer & O'Brien, 2006; Tsermentseli *et al.,* 2008). Language delays (Takarae *et al.,* 2008) or poor IQ levels (Koldewyn *et al.,* 2010) also correlate with elevated motion coherence thresholds in participants with ASD. The latter finding was not observed for performance on coherent form tasks, suggesting IQ may act as a proxy for temporal integration in ASD (Koldewyn *et al.,* 2010). To date, only one study has reported a superior performance on tasks involving the direction detection of luminance-defined motion gratings in participants with ASD (Foss-Feig *et al.,* 2013).

The deficit in motion processing appears to arise from a dorsal stream deficiency (Spencer *et al.,* 2000; Sutherland & Crewther, 2010). This pathway receives input from magnocellular portions of the lateral

geniculate nucleus, and then projects information to MT+/V5 and the posterior parietal cortex. In support of this assumption, one study observed increased activity in the left primary visual cortex and unmodulated engagement of MT+/V5 when adults with ASD viewed coherently moving dots (Brieber *et al.,* 2010). Abnormal activities in MT+/V5 have also been found when adults with ASD passively view movement or engage in a visual pursuit tracking task (Takarae *et al.,* 2014). Further, an ERP study found a deviant N200 component when participants with ASD discriminated the direction of coherently moving dots. Such ERPs reflects dorsal stream processing occurring over the occipitotemporal and parietal cortex (Greimel *et al.,* 2013). It is important to note, however, that participants with ASD sometimes perform poorly on form coherence tasks, raising the possibility of ventral stream deficits too (Fujita *et al.,* 2011; Koh *et al.,* 2010; Spencer & O'Brien, 2006; Tsermentseli *et al.,* 2008).

Many of the abovementioned studies implement first-order motion. Conversely, some investigations which use similar stimuli have reported an unimpaired perception of coherent motion in participants with ASD (De Jonge *et al.,* 2007; Del Viva *et al.,* 2006; Jones *et al.,* 2011; Price *et al.,* 2012). Instead, these authors have suggested that the deficit is specific to texture-defined (second-order) motion (Bertone *et al.,* 2003; Bertone & Faubert, 2006). This stimulus class is more complex, requiring further analysis and neural processing. It might, therefore, be possible to think of motion processing deficits in terms of abnormal visual integration rather than impairment to subcortical pathways. Indeed, there is evidence that participants with ASD fail to integrate coherent motion signals due to reduced attentional zoom out abilities (Ronconi *et al.,* 2012). A recent neuroimaging study reported that the early visual areas (V1 and MT+/V5) which process moment-to-moment motion signals were affected when stimuli duration was reduced (Robertson *et al.,* 2014). However, increasing the amount of time in which motion signals can be integrated significantly decreased the severity of global motion deficits in participants with ASD (Robertson *et al.,* 2012).

On the other hand, Vandenbroucke *et al.,* (2008) suggested that this is not the case at all. In their study, observers viewed plaid motion stimuli whereby two individual gratings were perceived as either sliding over each other or as a coherently moving pattern. The latter perception required the integration of motion features over space. The authors did not report any difference in the ability of ASD and control participants to perceive the coherent plaid. The current data is evidently mixed and further clarification is needed. Nonetheless, it is crucial to now extend investigations to dynamic stimuli which are biologically relevant.

1.5 Biological Motion in Neurotypical Controls

Biological motion is typically studied using point-light animations which track movement at the limb joints (e.g., knees, elbows and pelvis). It demonstrates the visual systems' ability to make sense of an

impoverished visual scene from motion cues alone (Johansson, 1973). As a result, the observer is able to create a percept of form-in-motion without any form cues actually being available. While the term 'biological motion' refers to all types of human movements (e.g., facial expressions, walking, hand gestures, eye-gaze patterns), this section will only focus on that evoked by the body.

1.5.1 Development

The mechanisms underlying biological motion perception develop during early infancy (Thompson & Parasuraman, 2012). It is unclear whether this function is an innate capability of the visual system or a by-product of experience (Bardi *et al.,* 2011). Preliminary studies indicated that 4-to-6 month old infants' preferred upright point-light motion compared to scrambled or inverted displays (Bertenthal *et al.,* 1984). Infants within this age range could also discriminate a canonical walker from stimuli with perturbed local rigidity between joints (Bertenthal *et al.,* 1987). More recent investigations have, however, reported that individuals can perceive biological motion before this time point (Bidet-Ildei *et al.,* 2013; Johnson *et al.,* 1991). For example, one study showed that infants as young as 2-days-old gazed longer at point-light displays of walking chickens relative to random or inverted movements (Simion *et al.,* 2008). Bardi *et al.,* (2013) reported similar findings in which 2-day-old newborns favoured upright (and therefore natural) depictions of a walking hen. Such preference for biological motion has also been described in newborn infants who are between 10 and 65 hours old (Bardi *et al.,* 2011). These findings provide evidence that the mechanisms responsible for biological motion perception are innate and are not specific to human motion.

However, it is plausible to suggest that such perceptual predisposition undergoes a maturity process during development. This would allow the discrimination of much more complex visual information. There is some evidence to support this view. Five-month-old infants were able to attend to biological motion while ignoring audio-visual synchronised events (Falck-Ytter *et al.,* 2011). Additionally, 6-month-old infants could differentiate the directionality (leftward or rightward) of upright point-light walkers (Kuhlmeier *et al.,* 2010). Lloyd-Fox *et al.,* (2011) suggested that the responsible neural substrates mature at approximately 5-months-old, while Hirai and Hiraki (2005) reported this occurs at 8 months of age. Recently, Furuhata and Shirai (2015) state the ability to process high-level information from biological motion (e.g., attentional direction of others) develops by 12-months of age. While the results are somewhat inconsistent between studies, they do highlight the importance of learning and experience when evaluating the developmental trajectory of biological motion perception.

1.5.2 Biological Motion as a Hallmark of Social Cognition

Body movements facilitate social perception and nonverbal communication (Bolling *et al.,* 2013; Miller & Saygin, 2013). Several studies have reported that qualitative discriminations can be derived from how a body is moving. For example, it has been shown that basic emotions (happiness, sadness,

surprise, fear, grief and disgust) can be recognised from dancing point-light figures (Clarke *et al.,* 2005; Dittrich *et al.,* 1996). Exaggerating these movements also evokes a more intense emotional expression to be perceived and therefore improves accuracy (Atkinson *et al.,* 2004). Other researchers demonstrate that point-light displays of simple everyday arm movements (e.g., knocking on a door) is sufficient in expressing internal mental states such as tiredness (Pollick *et al.,* 2001).

The way in which a body moves also conveys categorical information (Barclay *et al.,* 1978; Brooks *et al.,* 2008; Mather & Murdoch, 1994; Schouten *et al.,* 2013). Kozlowski and Cutting (1977) demonstrated that the gender of an individual could be recognised from point-light displays. Similar data has since been described with the aim of understanding what aspect of movement actually reveals gender (Pollick *et al.,* 2005). Cho *et al.,* (2004) suggested that the speed of step length differs between the sexes, with women walking significantly slower than men. Variations in lateral sway also indicate gender. Females exhibit increased translation of the hips whereas males show head and shoulder rotations (Murray *et al.,* 1970). It is these disparities which allow accurate perception of gender from sparse information displays.

As with face stimuli, the correct perception of biological motion is vulnerable to inversion (Blake & Shiffrar, 2007). In one study, all neurotypical participants were able to discriminate whether an upright point-light figure represented a body or not, but their performance severely decreased when the display was inverted (Shiffrar *et al.,* 1997). McGlothlin *et al.,* (2012) corroborated these findings, reporting that gender identification was substantially impaired when participants viewed inverted point-light human activities (walking, sitting and running). Accordingly, it has been suggested that biological motion relies on global configuration (Bertenthal & Pinto, 1994). Not all researchers agree with this hypothesis though, and instead claim that difficulties with inverted body motion might arise because the dot trajectories contradict gravity (Shipley, 2003; Bardi *et al.,* 2013; Maffei *et al.,* 2015). In this respect, Troje and Westhoff (2006) have shown that the perception of biological motion is possible with inverted stimuli when the dots representing the legs and feet remain upright. While these findings point to local motion cues driving human motion perception, we will see from the neuroimaging data that configural processing still has a significant role.

1.5.3 Neural Mechanisms

Functional Data (fMRI and TMS)

A constellation of substrates are central to biological motion processing (Grossman, 2005). The STS is at the forefront of this network, exhibiting increased activation during perception of body motion compared to non-biological motion (Allison *et al.,* 2000; Engell & McCarthy, 2013; Grézes *et al.,* 2001; Grossman & Blake, 2001; Pinto & Shiffrar, 2009). Pelphrey *et al.,* (2003) found strong activity occurring

in the right posterior STS (pSTS) to human and robot walking and very little activity to movements made by a clock or mechanical figure. Further, temporarily disrupting the STS in healthy patients via TMS caused impaired recognition of biological motion (Grossman *et al.,* 2005; Vangeneugden *et al.,* 2014). Van Kemenade *et al.,* (2012) similarly found that TMS applied to both the pSTS and premotor cortex significantly decreased the sensitivity to point-light animations. These effects were not observed for non-biological stimuli or when applied to a different substrate.

In addition, the activity of the STS increases when observers correctly identify the direction of movement (Herrington *et al.,* 2011) or the gender of point-light figures (Vaina *et al.,* 2001). It is also involved in analysing general social features (Lahnakoski *et al.,* 2012) and in social inclusion mechanisms (Bolling *et al.,* 2013). The posterior region is further engaged in action specific encoding (Grossman *et al.,* 2010) and perceiving the causality of movements (Morris *et al.,* 2008). Different regions of the STS are also activated by different biological movements. Mouth and eye motion elicit activity in the mid-posterior and right pSTS, respectively. Hand movements, however, activate the inferior and posterior portions of this structure (Pelphrey *et al.,* 2005).

The STS region does not just represent biological motion but also the intention related to it (Vander Wyk *et al.,* 2012). Vander Wyk *et al.,* (2009) presented video clips of simple grasping actions that were either congruent or incongruent with intentions established by a previous emotional (positive or negative) context. In an incongruent trial, participants were shown an actress frowning at a green cup and then proceeding to pick it up. The response would be unexpected given her prior emotional response. In a congruent trial, the actress would frown at the green cup and then proceed to pick up a different item. The authors discovered that the STS responded significantly more during incongruent trials, suggesting it is able to integrate information from biological motion and emotional contexts to produce an understanding of the current situation.

The STS has been considered as a convergence and communication point for various perceptual inputs. Grossman *et al.,* (2010) suggest it is an integration site for the dorsal and ventral visual streams, providing both form and motion information. High-level multimodal associations are also in place between the STS, amygdala and FG (Herrington *et al.,* 2011). For example, Sadeh *et al.,* (2010) observed a high correlation between the N170 component and activity of the FFA and STS when combining fMRI and ERP technologies. Other researchers note a concurrent increase in MT+/V5 and STS activity to biological motion (Herrington *et al.,* 2011). It may be that MT+/V5 responds to the complex motion pattern present in biological motion, while the STS activity reflects the action portrayed. However, these multimodal associations are not limited to cortico-cortical connections (Sokolov *et al.,* 2014). There is evidence that the cerebellum is responsive to displays of coherent motion presented in random noise (Jokisch *et al.,* 2005) and to body motion (Grossman *et al.,* 2000;

Vaina *et al.,* 2001). In support of this, patients with left lateral cerebellar lesions exhibit impairments in the perception of biological motion (Sokolov *et al.,* 2010). Recently, Sokolov *et al.,* (2012) observed engagement of the lobules Crus 1 and the VIIB of the left lateral cerebellum when healthy participants viewed point-light walkers. The strongest bi-directional communication also occurred between the left cerebellar lobule Crus I and the right pSTS. These associations collectively support biological motion processing by providing the pSTS with visuo-motor inputs.

Electrophysical Data (EEG and MEG)

Understanding the temporal dynamics behind biological motion perception has been achieved via ERP analysis (Buzzell *et al.,* 2013; Fraiman *et al.,* 2014; Saunier *et al.,* 2013). The current literature is consistent in observing two subsequent components, although the relative timing of each peak varies across studies. Hirai *et al.,* (2003) reported negative going ERPs over bilateral occipitotemporal scalp regions at 200ms and 240ms post motion onset. Other researchers have observed the N200 and N330 when participants viewed upright body motion (Hirai *et al.,* 2005). Similarly, Jokisch *et al.,* (2005) found that point-light walkers elicited the N170 and N300. As the first component showed greater sensitivity to upright figures than inverted, the authors suggest it is concerned with holistic processing and automatic feed-forward mechanisms. The later ERP, however, was not influenced by orientation, signifying it is sensitive to the analysis of biological motion exclusively.

While other ERP studies advocate this view (Hirai *et al.,* 2003; Hirai *et al.,* 2005), there is recent data to suggest otherwise. White *et al.,* (2014) required participants to view stick figures and point-light walkers (static and dynamic). Each category of stimuli was presented as upright, inverted and scrambled figures. Similar to Jokisch *et al.,* (2005), stronger negativities occurred for upright and inverted stimuli during late components (denoted here as N2). However, both static and dynamic point-light walkers evoked identical N2 peaks. The authors suggested that N2 is an index of human form recognition rather than being specific to biological motion. Notably, a subsequent component was identified and referred to as MPP/VAN. White *et al.,* conjecture that MPP/VAN reflects the recognition of human actions occurring within the pSTS.

Despite the aforementioned divergence, it is clear that the neurocognitive analysis of biological motion is a multiple-phase hierarchical process (Troje, 2008). These procedures occur via low-level visual mechanisms and are independent of top-down attentional control (Thornton & Vuong, 2004). Krakowski *et al.,* (2011) provides supporting data for these claims. Compared to scrambled motion, viewing intact point-light walkers produced a positive shift of the ERP between 100 and 200ms. This P1 component was sensitive to biological motion regardless of whether attention was directed toward the global configuration of the walker. Negativity then occurred from 200 to 350ms over posterior

middle temporal regions. Source analysis indicated that the first phase was generated by MT+/V5, whilst neuronal populations within the STS evoked the negative-going ERP.

The Mirror Neuron System (MNS)

While research has highlighted the STS in biological motion processing, there are more frontal-parietal networks to consider. The MNS is a neural organisation underlying stimulation processes within the motor and limbic system (Oberman *et al.,* 2008). Mirror neurons were first discovered in area F5 of the rhesus monkeys' premotor cortex (di Pellegrino *et al.,* 1992) and later in the inferior parietal cortex (Gallese *et al.,* 2002). These visuo-motor neurons would discharge when the monkey executed movements but also during the observation of similar actions. Such finding raised the question of whether comparable mechanisms were available in humans.

To investigate this, electrophysiological research has examined neural oscillations occurring over central motor cortices. Sharing a similar frequency to the alpha band (8-12Hz), mu rhythms index action planning and preparation within the somatosensory cortex (Keuken *et al.,* 2011; Mizuhara, 2012). Its' power is suppressed and attenuated when one performs an action, reflecting downstream sensorimotor and premotor activity (Cuellar *et al.,* 2012; Ulloa & Pineda, 2007). Indeed, Mizuhara (2012) found decreased mu rhythms over sensorimotor regions during visually guided motor execution (hand grasping) tasks. Further, the amount of mu suppression is influenced by whether the action is goal directed or meaningful (Cannon *et al.,* 2014). Hand grasping actions made towards an object, compared to simple hand extensions or non-object grasping movements, caused a stronger decrease in mu power (Muthukumaraswamy & Johnson, 2004b; Muthukumaraswamy *et al.,* 2004).

Significant mu suppression is also evident when one observes others' execute goal-directed actions (Aleksandrov & Tugin, 2012; Muthukumaraswamy *et al.,* 2004; Oberman *et al.,* 2005; Perry *et al.,* 2011). This is particularly evident when the observed action is within the participants own motor repertoire. Using fMRI, Calvo-Merino *et al.,* (2005) showed film clips of both ballet and capoeira movements to professional ballet dancers, capoeira dancers and novices. The dancers showed greater premotor-parietal activity for the genre in which they were trained in. Novices, however, elicited no differences in their neural response between dance types. It was later reported that such activity is also sensitive to gender effects. Ballet dancers evoked greater parietal-premotor activity to movements performed by their own gender (Calvo-Merino *et al.,* 2006). These putative mirror neurons are therefore sensitive to motor expertise and familiarity with the observed action (Cannon *et al.,* 2014).

Owning to its involvement in representing the actions of others, much research has suggested that mirror neurons play a role in social cognition (Oberman *et al.,* 2008). In corroboration with this hypothesis, significant mu suppression transpires when one perceives point-light actions (Cochin *et al.,*

2001; Ulloa & Pineda, 2007) or identifies the gender, intention and emotion of the figure (Perry *et al.,* 2010). Further, greater negative ERP components over central-parietal regions to point-light animations has been thought to reflect the involvement of sensorimotor cortices during the visualisation of biological motion (Saunier *et al.,* 2013; Virji-Babul *et al.,* 2007). The mechanisms behind motion perception and action understanding are associated, working together to produce a coherent social percept (Ulloa & Pineda, 2007).

The current findings suggest an observation-and-matching system is available in humans and facilitates social cognition (Puzzo *et al.,* 2010). The MNS is located primarily in portions of the precentral gyrus, posterior inferior frontal gyrus (IFG) and inferior parietal lobules (IPL) (Rizzolatti & Craighero, 2004). These form a complex network, receiving inputs from the visual cortex via the STS (Nishitani *et al.,* 2004; Lepage and Théoret, 2006). Together they facilitate imitation, action understanding and the use of movement to guide one's own behaviour (Molenberghs *et al.,* 2012).

1.5.4 Interim Summary 3

Biological motion perception utilises a network of cortical structures, including those sensitive to social, visual or motion information. The STS appears to be at the forefront of this system. Its role has been characterised as a convergence or communicational point for multiple visual pathways, but also as a redirecting system to other neural regions (e.g., FFA, MT+/V5, amygdala and cerebellum). Research has also highlighted the role of frontal-parietal networks thought to comprise the MNS. The projections from visual areas to more frontal substrates appear to support the analysis of human motion.

1.6 Biological Motion in ASD

Behavioural studies report impairments in perceiving biological motion (Koldewyn *et al.,* 2011; Moore *et al.,* 1997) in participants with ASD. Children with ASD commit more errors than controls when indicating whether point-light animations (depicting jumping, kicking, and walking) represent a body (Annaz *et al.,* 2010; Blake *et al.,* 2003; Nackaerts *et al.,* 2012). Similarly, participants with ASD are less accurate than controls when indicating whether a hand performing sinusoidal actions (minimum jerk) move in a natural or unnatural way (Cook *et al.,* 2013). Such biased perception of biological motion also appeared to correlate with abnormal kinematics (velocity, acceleration, and jerk) and symptom severity. Deficits are further evident when participants with ASD attempt to identify the emotion portrayed by point-light walkers (Atkinson, 2009; Hubert *et al.,* 2007; Parron *et al.,* 2008). Reduced accuracy on these tasks has been associated with increased reaction times and shorter or random looking patterns (Klin *et al.,* 2009). They also experience difficulty orienting to a point-light pointing gesture (Swettenham *et al.,* 2013). Pointing facilitates shared attention processes and is extremely

important for both social and linguistic functions (Brooks & Meltzoff, 2008). These findings indicate that the perception of emotional and non-emotional human actions is weakened in ASD.

Conversely, other researchers have reported intact biological motion mechanisms in ASD. Cusack *et al.,* (2015) designed a battery of experiments that sought to test the ability of adolescents with ASD to discriminate between: (1) animate motion and randomised or robotic-like sequences; (2) fighting or dancing gestures (action perception); and (3) stimuli whereby only the limbs were coherently intact or spatially scrambled. The data did not reveal any group differences between participants with ASD and neurotypical controls on any of these tasks. Similarly, Saygin *et al.,* (2010) presented participants with point-light displays (walking figures, translating triangles, or translating unfamiliar shapes) embedded in noise and asked them to determine the direction of movement. Participants with ASD performed identically to controls across all three tasks. Murphy *et al.,* (2009) also showed that participants with ASD could successfully identify the direction in which a point-light walker (embedded in noise) was moving. The authors speculate that the experimental group were able to integrate local motion cues to produce a coherent perception of biological motion.

Inconsistent findings may reflect an experimental bias caused by testing different age groups. Studies with children consistently report a deficit in biological motion perception whereas the adult data is less conclusive (Murphy *et al.,* 2009). Perhaps the perception of biological motion improves with chronological age (Hubert *et al.,* 2007). It is possible that older participants with ASD acquire compensatory mechanisms, thus performing similarly to controls. There is some evidence to support this view. Van Boxtel and Lu (2013) measured accuracy on a central counting paradigm while task-irrelevant biological motion was presented in the periphery of participants with low and high autistic traits. Stimuli were intact or spatially scrambled. Participants with fewer autistic traits were found to involuntarily process global aspects of biological motion even when it was detrimental to their central task performance. However, participants with high autistic traits did not show this pre-attentional distraction, performing identically on the central task in the scrambled and intact conditions. In a second experiment implicating an action adaptation paradigm, the authors demonstrated that deficits in global processing were compensated for by an increase in local processing. Alternatively, factors such as high symptom severity (Blake *et al.,* 2003) or low general intelligence (Jones *et al.,* 2011; Rutherford & Troje, 2012) could affect their ability to perceive biological motion.

1.6.1 Neuroimaging Data (fMRI and EEG)

The neural substrates responsible for biological motion have been found to be atypical in participants with ASD. At least two studies have observed a decreased volume (neuron density) of the pSTS (Boddaert *et al.,* 2004; von dem Hagen *et al.,* 2011). Using fMRI, other researchers have documented a reduced engagement of this region during biological motion recognition tasks in participants with

ASD relative to controls (Pelphrey *et al.,* 2005; Redcay, 2008; Zilbovicius *et al.,* 2006) or unaffected siblings (Kaiser *et al.,* 2010). This dysfunction has also been linked with an inability to integrate information from body motion with emotional contexts (Pelphrey *et al.*, 2011). In one study, high-functioning adolescents with ASD viewed reaching actions that were congruent or incongruent with an actor's prior emotional cue (negative or positive). Relative to controls, the ASD group did not show a strong effect of congruency within the right pSTS (Ahmed & Vander Wyk, 2013).

Irregular STS activity appears to coincide with an abnormal engagement of frontal-parietal structures. In one study, thresholds for biological motion perception were assessed via presenting participants with point-light walkers embedded in noise (Koldewyn *et al.,* 2011). Compared to participants with ASD, neurotypical controls evoked more activity in the dorsolateral prefrontal cortex, pSTS and intraparietal sulcus. Freitag *et al.,* (2008) also reported similar findings. Stronger activities were observed in the somatosensory cortex and IPL when controls perceived intact body motion relative to participants with ASD. These neural deficits could reflect under-connectivity between frontal and sensory areas, pinpointing a problem in coordinating neural networks during the redirection or integration of complex motion information (Just *et al.,* 2007; Minshew & Williams, 2007)

Additionally, there is less engagement of regions involved in early visuo-motor processing. Using EEG, Kröger *et al.,* (2014) presented children with ASD or neurotypical development with point-light walkers and scrambled stimuli. The P100 component, which reflects stimulus extraction and motion detection within the visual cortex, was reduced in participants with ASD across both conditions. In a second processing stage, intact and scrambled walkers evoked the N200 ERP. This was located over MT+/V5, and localised to the right hemisphere in neurotypical children only. Similar findings have been observed using fMRI. Herrington *et al.,* (2007) asked adults with neurotypical development or ASD to indicate the walking direction of a point-light figure. While both groups were identical in their ability to accurately perceive walking direction, participants with ASD showed hypoactivity in MT+/V5 relative to controls. Such alterations highlight the relevance of early visuo-sensory and motion processing on later stages of biological motion analysis.

These findings suggest that participants with ASD utilise the same basic network of areas to process biological motion as controls, but significantly under-activate them. On the other hand, it is conceivable that participants with ASD actually evoke a differential pattern of BOLD activity entirely. For example, an fMRI study found that neurotypical controls activated a pathway leading from the inferior temporal gyrus to the superior parietal lobule when viewing body motion stimuli. By contrast, participants with ASD utilised portions of the FG, inferior and middle occipital gyrus and middle temporal gyrus (McKay *et al.,* 2012). As no behavioural discrepancies were observed, the results would suggest that participants with ASD were able to use compensatory strategies to process biological motion. While

neurotypical participants integrate form and motion cues so that information can be transmitted to the parietal regions, participants with ASD process these data separately (McKay *et al.,* 2012).

1.6.2 The Mirror Neuron System (MNS)

A dysfunction of mirror neurons may underlie poor social cognition in ASD. Neuroimaging techniques ranging from EEG, fMRI and TMS have been implemented in an attempt to observe impairment in regions thought to comprise the classic MNS. However, the current results are mixed and present no clear deficit in ASD (Hamilton, 2013).

Many studies have investigated mu suppression in ASD via time-frequency analysis (Bernier *et al.,* 2007; Martineau *et al.,* 2008; Nishitani *et al.,* 2004). Oberman *et al.,* (2005) required high-functioning participants with ASD to watch videos of self-hand movements, other peoples hand movements, a bouncing ball or white noise. While controls exhibited significant mu suppression to both self and observed hand motion, participants with ASD only activated the MNS during own-hand perception. In a similar study, children with ASD observed hand grasping actions that were performed by an unfamiliar person, a familiar person (guardian) or themselves (Oberman *et al.,* 2008). Children with ASD did not evoke significant mu suppression when observing movements made by a stranger. However, both groups of children showed a greater amount of mu suppression (i.e., the MNS was more active) to their own movements, followed by those of a familiar person. Evidently, the MNS responds to observed actions in participants with ASD but only when they can identify with the stimuli.

While these data corroborate the 'broken mirror neuron' theory in ASD (Ramachandran & Oberman, 2006), other research has found no significant differences in the amount of mu-suppression occurring in participants with ASD and neurotypical controls (Fan *et al.,* 2010; Raymaekers *et al.,* 2009; Ruysschaert *et al.,* 2014). Discrepant results could be explained by developmental effects related to age, although this idea has been disproven by Oberman *et al.,* (2013). Instead, a recent study showed that dividing the mu rhythm into two sub-bands significantly impacts the resulting data (Dumas *et al.,* 2014). The analysis of lower mu (8-12Hz) revealed normal modulation of sensorimotor areas during action observation in participants with ASD. By contrast, the examination of upper mu rhythms (10-13Hz) indeed indicated irregular responses over occipito-parietal and frontal regions. It is important that future investigations continue to implement this segregation in order to produce a more unified account of mirror neurons in ASD.

In the context of TMS, a handful of studies have measured the excitability of mirror neurons during action observation in ASD by recording motor evoked potentials (MEPs) from dorsal intersseous muscles in the hand. Théoret *et al.,* (2005) presented videos of intransitive thumb and index finger movements that were from egocentric or allocentric perspectives. Compared to controls, participants

with ASD failed to show MEP enhancement over the primary motor cortex (M1) when observing egocentric hand movements. Similarly, Enticott *et al.,* (2012) required participants to view goal-directed hand movements or a static hand while TMS was applied to the left M1. Unlike controls, participants with ASD exhibited reduced MEPs during transitive action observation. Abnormal excitability of the MNS was also found to correlate with social symptom severity, suggesting that a dysfunction of mirror neurons contributes to the social deficits commonly seen in ASD. By contrast, Enticott *et al.,* (2013) failed to a find similar effect in their study examining the stimulation of M1 to perceived interactive hand actions. It is plausible that because the stimuli were representative of real interaction, participants were more motivated to attend to them, which in turn facilitated action processing. This is speculative though and more research is needed here.

Implementing fMRI allows direct observation regarding which MNS structures may be atypical in ASD. As mentioned previously, the classic MNS is composed of the IFG and IPL (Rizzolatti & Craighero, 2004). As it stands, the current data is mixed. One investigation had reported hyperactivity of the IFG in participants with ASD (Martineau *et al.,* 2010), whilst a similar study found no abnormality within this region (Dinstein *et al.,* 2010). In the context of emotional stimuli, the IFG is hypoactivated when participants with ASD view whole body actions depicting fearful behaviour (Grézes *et al.,* 2009). Other papers have reported that a dysfunction of mirror neurons is only present in younger participants with ASD. Bastiaansen *et al.,* (2011) utilised an emotional mirroring paradigm in which participants observed disgusting stimuli or tasted something disgusting. Data at the group level revealed no significant differences, but further analyses indicated that decreased activation of the right IFG occurred in young adults with ASD. It is plausible to suggest that participants with ASD may 'outgrow' deficits in the MNS as they learn to perform compensatory strategies (Enticott *et al.,* 2012).

1.6.3 Interim Summary 4

The current data would suggest a biological motion processing deficit in ASD (Blake *et al.,* 2003; Cook *et al.,* 2009). This impairment extends itself to both emotional and non-emotional contexts (Nackaerts *et al.,* 2012). In support of this, neuroimaging studies ranging from EEG to TMS have documented abnormalities within the pSTS, MT+/V5, FG and regions of the classic and extended MNS (Ahmed & Vander Wyk, 2013; Bastiaansen *et al.,* 2011; Enticott *et al.,* 2012; Kröger *et al.,* 2014; Oberman *et al.,* 2013; Pelphrey *et al.,* 2005). Not all data are uniform here though, with several studies failing to observe any behavioural or neurological differences between ASD and control groups (Fan *et al.*, 2010; Enticott *et al.,* 2012; Murphy *et al.,* 2009). Consequently, the age, clinical impairment and IQ level of the experimental group should be taken into account when assessing the current literature. It is also possible that the impairment in biological motion perception lessens over time as participants with ASD learn to perform compensatory mechanisms (McKay *et al.,* 2012).

1.7 Facial Motion in Neurotypical Controls

While biological motion refers to both body and facial movement (Pelphrey & Carter, 2008), there is substantially less research concerning the latter. It is crucial to extend our investigations to include this stimulus class, especially considering its prominent role in social communication (O'Brien *et al.,* 2014). Inclusion of facial motion stimuli would also benefit future investigations examining social cognition in ASD, given the deficits seen in this disorder. This section will review research which has implemented dynamic face stimuli in neurotypical populations.

1.7.1 General Perception of Dynamic Faces

The use of dynamic faces in perception research has begun to attract interest over the last few decades (Ichikawa *et al.,* 2010; Kamachi *et al.,* 2001; Puce *et al.,* 1998; Puce *et al.,* 2000; Rossi *et al.,* 2014). Lloyd-Fox *et al.,* (2009) measured the brain haemodynamic levels of typically developing infants during the perception of facial movements (eyes shifting or mouth deforming into vowel shapes). Greater neural activation was localised to regions of the posterior temporal cortex, namely over the STS. Similar findings have been reported in neurotypical adults. For example, dynamic faces elicited higher responses in the STS and MT+/V5 area relative to static or phase-scrambled controls (Schultz & Pilz, 2009). Further, Schultz, *et al.,* (2013) found that the perceived fluidity and meaning of facial stimuli increased with frame rate and correct frame order. This enhancement correlated with notably stronger BOLD responses in the STS. However, ventral temporal areas (FFA and OFA) were only influenced by frame rate, suggesting a higher sensitivity to static information contained within dynamic faces (Schultz *et al.,* 2013). There is some further evidence to support these claims. Both Pitcher *et al.,* (2011) and Polosecki *et al.,* (2013) revealed a clear functional dissociation between the STS and OFA/FFA, with the latter being insensitive to facial motion. These findings suggest that the STS region is most strongly associated with facial motion processing.

1.7.2 Emotion Recognition from Facial Motion

Two seminal studies using point-light faces found that emotional expressions were perceived correctly when in motion, but not when static (Bassili, 1978, 1979). These findings were later replicated by Pollick *et al.,* (2003) who also reported that the amplitude and velocity of motion affected the intensity of the perceived emotion. Further, using two-frame apparent motion and a smaller number of local motion vectors, expressions could be accurately recognised from point-light faces (Matsuzaki & Sato, 2008). These stimuli appear disorganised until set into motion, suggesting that the visual analysis of point-light faces relies on local motion cues (Garcia & Grossman, 2008).

Implementation of morphed or averaged dynamic faces has also shown motion signals to benefit emotion recognition by providing a more accurate mental representation of the expression (Ambadar *et*

al., 2005; Chiller-Glaus *et al.,* 2011; Harwood *et al.,* 1999). Bould and Morris (2008) reported that subtle facial expressions were better recognised in motion, although this effect was reduced for high intensity expressions. This may be because intense emotions already carry enough information for correct identification (Bould & Morris, 2008). Visual search of emotional expressions is also more efficient with dynamic than static stimuli, particularly for negative faces (Horstmann & Ansorge, 2009). The authors speculate that this has more to do with the amount of movement conveyed by angry faces, rather than the emotion itself. It appears, therefore, that perceptual differences in movement signals promotes the recognition of emotions.

The improvement in recognition correlates with notable increases in the functional activity of face-selective and emotion processing networks, including the FG and amygdala (Atkinson *et al.,* 2012; Kessler, 2011; Sato *et al.,* 2004; Trautmann-Lengsfeld *et al.,* 2013). LaBar *et al.,* (2003) displayed photographs and morphed videos of angry, fearful and neutral expressions. They observed stronger activity of the FG, ventromedial prefrontal cortex and STS for dynamic than static emotions. Similarly, Kilts *et al.,* (2003) compared dynamic and static faces depicting happy and angry expressions. Dynamic presentations increased activity in the MT+/V5, STS and periamygdaloid area for angry faces, and the cuneus, lingual gyrus, middle temporal and medial frontal gyrus for happy faces. Natural video recordings of facial expressions also evoke greater activity in the bilateral STS and middle temporal gyrus compared to static presentations (Foley *et al.,* 2012).

A comparison of dynamic and static emotional faces (happy and disgusted) revealed greater activation patterns in the parahippocampal regions, amygdala, FFA, STS and supplementary and premotor areas (Trautmann *et al.,* 2009). The latter two neural substrates demonstrate MNS involvement in recognising dynamic facial emotions. Similar activations during the passive viewing of dynamic emotions and point-light speech have been further observed (Hennenlotter *et al*., 2005; Santi *et al.,* 2003). Imitating and observing basic dynamic primary emotions also evokes greater activity in regions of the classic (IFG, posterior parietal cortex) and extended (STS, amygdala, pre/supplementary motor and somatosensory cortex) MNS (Van der Gaag *et al.,* 2007). Similarly, Sarkheil *et al.,* (2013) found task-dependent activation of the IPL during emotional expression analysis. This area has been thought to contain mirror neurons, given its role in sensorimotor integration (Clower *et al.,* 2001; Rizzolatti & Craighero, 2004). These findings demonstrate that the perception of dynamic facial emotions recruits a large network of neural substrates.

1.7.3 Categorical Discriminations From Facial Motion

Facial motion conveys information concerning the identity of an individual. Such influence appears to depend on a number of factors, including degree of familiarity and viewing conditions (Knappmeyer *et al.,* 2003). Studies which have implemented impoverished stimuli (threshold processed videos,

blurred/pixelated clips or limited frame sequences) report a beneficial effect of motion during familiar face recognition (Lander *et al.,* 1999; Lander & Bruce, 2000; Lander *et al.,* 2001). This effect was also found when using negated (contrast-reversed) images of famous and familiar faces (Knight & Johnston, 1997). This suggests that motion provides detailed 3D information concerning face structure, but also prompts recognition of idiosyncratic movements (O'Toole *et al.,* 2002).

Whether the motion is rigid (head translations and rotations) or nonrigid (expressional changes of the face) also influences identity recognition. Unfortunately, the data is rather divided with regards to which motion class actually benefits facial identification. Pike *et al.,* (1997) found an advantageous effect of rigid motion when participants viewed an unfamiliar face rotating. Using facial motion captures, Hill and Johnston (2001) also found rigid motion to be the most useful during identity discrimination tasks. A later study by Watson *et al.,* (2005) replicated this finding and extended it to recognising different individuals in varying view-points. However, there are a handful of studies which do not support the role of rigid motion in facial identification (Bonner *et al.,* 2003; Lander & Bruce, 2003; Lee *et al.,* 2010). Christie and Bruce, (1998) failed to show an improvement in the recognition of unfamiliar faces exhibiting rigid motion compared to multiple static views. A similar result was found when observers viewed degraded movies of familiar and famous faces moving rigidly (Lander & Chuang, 2005). Instead, other authors note that it is actually nonrigid motion that facilitates person identification (Thornton & Kourtzi, 2002). Indeed, Pilz *et al.,* (2006) found a benefit of nonrigid motion regardless of task type (sequential matching versus visual search) or viewpoint.

In addition to identification, facial motion facilitates the discrimination of male and female faces. Berry (1991) found that adults and children could correctly identify the gender of interacting point-light faces 69% and 58% of the time respectively. Using facial motion captures, Hill and Johnston (2001) report that correct gender discrimination was facilitated by nonrigid motion because such facial actions are functionally related to speech and expression which differ between the sexes. Information concerning age can also be derived from facial motion, although this effect has been substantially less explored. Berry (1990b) examined the ability of perceivers to detect age related social and physical qualities from facial motion. Participants observed point-light faces of children, middle aged adults and older adults, and were asked to rate the physical and social power of each display. As expected, higher power ratings were consistently given to adult stimuli.

1.7.4 Interim Summary 5

Facial motion conveys both categorical (age, identity, gender) and qualitative (emotional expressions) information, even in the absence of form cues. The addition of motion also facilitates recognition of specific facial information during suboptimal viewing conditions (e.g., blurred or pixelated videos). Further, the neuroimaging data suggests that compared to static stimuli, greater widespread activation

patterns are evident for dynamic presentations of faces in regions of the FG, amygdala, MT+/V5, and regions thought to comprise the MNS (e.g., IFG and IPL). The current findings also observe specific STS activity to perceived facial movements, suggesting it is sensitive to changeable face properties.

1.8 Facial Motion in ASD

Investigations of facial motion in ASD are sparse, with only a few research groups implementing dynamic stimuli (Enticott *et al.,* 2014; Miyahara *et al.,* 2007; Uono *et al.,* 2009). Pelphrey *et al.,* (2007) compared static images with emotion and identity morphed videos. Such contrast strongly activated the amygdala, right pSTS, bilateral FG and MT+/V5 in neurotypicals. In participants with ASD however, only motion sensitive MT+/V5 exhibited greater activation to dynamic versus static faces. The authors also revealed a lack of modulatory activity of the STS, in addition to hypoactivation of the FG and amygdala during fear and anger perception. Sato *et al.,* (2012) reported similar results and extended hypoactivation to the middle temporal gyrus (MTG), medial prefrontal cortex and IFG in participants with ASD. A weakened bi-directional connectivity within the V1-MTG-IFG network was also observed in this study. Abnormal functioning of the IFG has been previously found in studies investigating the MNS. Dapretto *et al.,* (2006) required neurotypical and autistic children to imitate and observe facial emotional expressions. While both groups exhibited no behavioural impairment, only control children showed enhanced activation in the pars opercularis of the IFG. This finding is in contrast to that reported by Rahko *et al.,* (2012) who observed increased IFG activity in participants with ASD compared to controls during valence scaling of dynamic facial expressions.

Nevertheless, these findings point to abnormalities within pathways leading from primary visual areas to specialised higher order cognitive regions. Specifically, it is possible that participants with ASD experience difficulties in the earliest visual component of dynamic face processing. Supporting this assumption, a recent study found that participants with ASD were impaired on tasks requiring them to rate the naturalness of facial expressions that differed in speed (Sato *et al.,* 2013). Such paradigms required participants to analyse dynamic properties of emotional expressions, a process that is meant to occur at the earliest stage of facial expression decoding (Haxby *et al.,* 2000). Indeed, investigations which have slowed down the speed of dynamic face presentations report a beneficial effect in aiding participants with ASD to recognise and imitate emotional expressions (Gepner *et al.,* 2001; Tardif *et al.,* 2007). Collectively, these studies indicate that participants with ASD have a weakened perception of facial motion. This function needs to be further explored for more concrete conclusions to be drawn.

1.9 Summarising the Previous Research - What has it Told Us?

Social cognition is central to survival. It is crucial to correctly perceive and interpret the actions of other people to ensure continual emotional and physical well-being (Pelphrey & Carter, 2008). Accordingly,

the brain has developed a specialised network of mechanisms supporting social perception. In general, the main components include the: (1) mPFC and temporoparietal junction for mental state attribution; (2) amygdala, insula and anterior cingulate cortex for emotion recognition; (3) OFA and FFA for face processing, including initial detection and identity analysis; and (4) STS for biological motion perception (Iidaka, 2014; Kana *et al.,* 2014; Kanwisher *et al.,* 1997; Krolak-Salmon *et al.,* 2003; Luo *et al.,* 2010; Pitcher *et al.,* 2011).

Participants with ASD are substantially impaired on tasks involving the recognition of emotions, identification of different faces and inference of mental states (Bradshaw *et al.,* 2011; Dawson *et al.,* 2002; Poljac *et al.,* 2012; Stewart *et al.,* 2013; Williams & Happé, 2009). These deficits are thought to be the result of abnormal gaze patterns or poor attention to socially relevant stimuli within the environment (Schultz, 2005). Indeed, participants with ASD tend to focus more on non-feature areas of the face and spend less time looking at the eye region compared to controls (Pelphrey *et al.,* 2002). Regardless of how subtle, eye movements convey an extraordinary amount of information necessary for mind reading (Baron-Cohen *et al.,* 1997). Observing the gaze direction of another person might inform you of their forthcoming intention or current emotional state.

There is an overwhelming amount of support for the 'poor eye gaze hypothesis' in ASD (Baron-Cohen *et al.,* 2001; Joseph & Tanaka, 2003; Klin *et al.,* 2002). For instance, participants with ASD engage the FFA less than controls during face processing tasks (Nickl-Jockschat *et al.,* 2014). Yet when instructed to focus on the eye region, they exhibit a normal neural response (Hadjikhani *et al.,* 2004; Hadjikhani *et al.,* 2007). Some investigations, however, fail to report a deficit in extracting eye information, indicating other factors must contribute to an impaired social cognition in ASD (Back *et al.,* 2007; Song *et al.,* 2012). This may include an inability to process the spatial relations between face parts or to perceive the unified gestalt (Richler & Gauthier, 2014). Instead, participants with ASD rely on feature-based processing which is much less effective for recognition (Rose *et al.,* 2007).

A limitation of past literature is that it implements static stimuli. These do not depict real life and could possibly contribute to the inconsistencies often found within the literature (O'Brien *et al.,* 2014). Much research is now being directed towards understanding how participants with and without ASD perceive dynamic, and therefore ecological valid, displays of social cues (Allison *et al.,* 2000). Most commonly reported is that neurotypical populations can recognise categorical and qualitative social information from just a dozen moving dots representing limb joints (Atkinson *et al.,* 2004; Blake & Shiffrar, 2007; Clarke *et al.,* 2005; Pollick *et al.,* 2005). The visual system is therefore able to reconstruct a perceptual scene from motion cues alone, indicating its role in social cognition (Johansson, 1973; Pavlova, 2012).

In ASD, it has been hypothesised that a weakened perception of biological motion underlies social impairments (Pavlova, 2012). Such an idea is diverging away from more traditional concepts (weak central coherence or atypical mentalising) which have been previously used to explain this deficit (Baron-Cohen *et al.,* 1985; Frith, 1989). The inability to infer intentions, recognise faces or understand emotions may instead be rooted in more low-level visual mechanisms (Herrington *et al.,* 2007). Indeed, several investigations have shown that participants with ASD are impaired when required to perceive emotional and non-emotional information from point-light walkers (Atkinson, 2009; Blake *et al.,* 2003; Hubert *et al.,* 2007; Parron *et al.,* 2008; Swettenham *et al.,* 2013). This impairment appears to be caused by a dysfunction to the STS and its supporting associations with V1, FFA, amygdala and MT+/V5 (Ahmed & Vander Wyk, 2013; Koldewyn *et al.,* 2011; Kröger *et al.,* 2014; Pelphrey *et al.,* 2005). Regions of the observation and matching system (IFG and IPL) are also atypical in ASD (Bastiaansen *et al.,* 2011; Dumas *et al.,* 2014; Freitag *et al.,* 2008; Martineau *et al.,* 2010; Oberman *et al.,* 2013). Less engagement of the early visual cortex may impact later stages of biological motion analysis in ASD. It is also evident that the under-connectivity between sensory and frontal areas reflects a problem in redirecting or integrating complex motion information (Just *et al.,* 2007).

In general, the current research supports the hypothesis that social impairments in ASD are driven by difficulties in the visual analysis of biological motion (Herrington *et al.,* 2007). Yet most of this data is derived from investigations with body motion. Considering our prior assumptions, it is important to examine motion perception in the most social aspect of humans - the face (Girges *et al.,* 2014). Many studies with neurotypical samples show that facial motion facilitates the recognition of emotions, identities and genders by providing more accurate mental representations and prompting the detection of idiosyncratic movements (Bassili, 1978; Bould & Morris, 2008; Hill & Johnston, 2001; McGuiness & Newell, 2014; O'Toole *et al.,* 2002). In terms of underlying neural mechanisms, facial motion appears to mainly engage the STS (Pitcher *et al.,* 2011; Polosecki *et al.,* 2013; Schultz *et al.,* 2013), highlighting its role in processing all types of biological movements. Other researchers demonstrate the involvement of the FFA, amygdala and regions of the MNS in facial motion perception (Atkinson *et al.,* 2012; Trautmann *et al.,* 2009; Van der Gaag *et al.,* 2007). These areas form a complex network and support processing by providing the STS with visuo-motor inputs.

Substantially less research has been directed towards understanding facial motion perception in ASD. The handful of studies available, however, demonstrate that participants with ASD are impaired on tasks requiring them to recognise eye-gaze direction, basic emotions, unfamiliar identities and genders from dynamic faces (Enticott *et al.,* 2014; O'Brien *et al.,* 2014; Uono *et al.,* 2009). The neuroimaging data supports these findings by observing atypical activation of the STS, IFG, FFA and amygdala (Dapretto *et al.,* 2006; Pelphrey *et al.,* 2007; Rahko *et al.,* 2012). Abnormalities within pathways

leading from early visual areas to specialised high-order regions has also been observed, again suggesting a dysfunction in the earliest visual component of facial motion analysis.

It is therefore possible that a problem in processing facial movement contributes to poor social cognition in ASD. If participants cannot perceive changes in eye-gaze or distortion of the facial muscles, then they will fail to pick up on certain cues needed for processes such as joint attention, inference of mental states, intention attribution and so forth. The previous literature has been highly informative and provided important theoretical groundwork. However, there is still so much of facial motion processing to explore in both participants with ASD and neurotypical development. The next section will outline the aims and justifications for the current studies presented in this thesis.

1.10 The Current Research - Study Outlines and Aims

While many researchers are now implementing dynamic face stimuli, much of this perception is still left relatively unexplored. Several investigations have highlighted the role of the STS in facial motion processing (Pelphrey *et al.,* 2007; Polosecki *et al.,* 2013), yet it is unclear what is occurring within the visual cortex before such data reaches 'specialised substrates'. It is important to gain a clear representation, especially if we are to assume that low-level visual deficits contribute to poor social communication in ASD (Herrington *et al.,* 2007). Accordingly, the processing of whole-face human motion was measured by observing changes within the posterior EEG alpha band (Chapter 2). This oscillation was chosen for analysis as it represents a neural signature of visual activity occurring in the occipital regions (Berger, 1929). It has also been previously shown that action observation evokes irregular responses over the parieto-occipital cortex in ASD (Dumas *et al.,* 2014). Because the study reported in Chapter 2 was novel, it was first conducted with neurotypical participants so that the methodology and resulting data could be evaluated.

Prior to collecting any neuroimaging data with ASD participants, it was necessary to conduct a behavioural study. There are currently only a handful of published papers exploring facial motion perception in ASD (Enticott *et al.,* 2014; Gepner *et al.,* 2001; Sato *et al.,* 2013; Tardif *et al.,* 2007; Uono *et al.,* 2009). The majority of these focus on emotion recognition. Faces however provide salient cues concerning identity, gender and age (Berry, 1990; Hill & Johnston, 2001). Chapter 3 describes the ability of participants with ASD to perceive facial motion and use such information when making specific categorical judgments (sequence, identity and gender). Configural and feature-based processing of facial motion was also measured via the use of inversion paradigms to facilitate our understanding of the perceptual mechanisms used in ASD.

While conducting this experiment, it was evident that new stimuli sets were needed. The facial motion animations used in Chapters 2 and 3 were taken from a database developed by Hill and Johnston (2001). Although these depicted motion-based information separately from other cues, some animations seemed inexpressive or limited in how much movement they actually conveyed. Chapter 4 outlines a new method in which marker-less technology generated fluid and accurate models of human facial deformation. Rigid and nonrigid motion (including eye and speech patterns) were recorded from real actors and applied to the same computer-generated face. The stimuli were validated in a behavioural study by asking neurotypical participants to discriminate categorical (video sequence and identity) information from these motion captures.

The study was then replicated in the MRI scanner (Chapter 5). The purpose of this was to evaluate the neural response to facial motion in neurotypical participants. Pre-existing studies often implement abstract or unnatural stimuli such as implied motion, morphed videos and cartoon avatars (e.g., Pelphrey *et al.,* 2007). But these representations do not fully capture the underlying mechanisms involved (Schultz & Pilz, 2009). It was therefore important to extend investigations to include ecologically valid dynamic faces. Other stimulus categories also were included in order to examine exclusive patterns of activity concerned with facial motion perception. Participants were thus presented with a range of static images (faces, bodies, objects and places) and moving videos (point-light walkers and coherent/random dot kinematograms). A second purpose of this study was to observe the mechanisms underlying identity recognition. While identity judgments with static faces evoke activity in the OFA and FFA, it is unclear if this response also occurs for dynamic faces or whether the STS is utilised instead. This was investigated by explicitly asking participants to discriminate identities based solely on differences in motion patterns.

The last experimental chapter of this thesis is concerned with the neural correlates of facial motion perception in ASD (Chapter 6) using fMRI. Similar to the experimental design implemented in Chapter 3, participants engaged in a sequence discrimination task using the new facial motion captures. At the time of writing, there are only a handful of published papers which have examined neural activity to facial motion in ASD (Dapretto *et al.,* 2006; Pelphrey *et al.,* 2007; Rahko *et al.,* 2012; Sato *et al.,* 2012). None of these studies implement highly controlled or natural depictions of facial movement. However, it is important that realistic stimuli are used in order to gain an accurate understanding of the neural mechanisms impaired in ASD. The experiments reported in this thesis will provide crucial and detailed information regarding the perception of facial motion in both neurotypical and ASD participants.

Chapter 2 Event-related Alpha Suppression in Response to Facial Motion

2.1 Introduction

The visual system can reconstruct an impoverished perceptual scene from motion cues alone. For example, a human walker can be detected from just a dozen moving dots representing the major limb joints (Johansson, 1973). This perception of biological motion has been thought to underlie many aspects of social cognition (Pavlova, 2012). Indeed, neurotypical populations can recognise basic emotional expressions (e.g., happiness, tiredness or fear) from point-light figures displaying dance steps or simple everyday actions (Dittrich *et al.,* 1996; Pollick *et al.,* 2001). This finding has recently been extended to stimuli which depict emotional movements in the upper body region only (Volkova *et al.,* 2014). Demographic characteristics such as gender can also be successfully conveyed via biological motion (Schouten *et al.,* 2013). Females appear to exhibit greater translations of the hips whereas males only show head and shoulder rotations (Cho *et al.,* 2004; Murray *et al.,* 1970).

The temporal processing of biological motion has been revealed via ERP analysis (Fraiman, *et al.,* 2014; Hirai *et al.,* 2003; Hirai & Hiraki, 2006a & 2006b; Krakowski *et al.,* 2011). Hirai *et al.,* (2003) found that the observation of upright point-light walkers evoked the N200 and N330 in neurotypical participants. Jokisch *et al.,* (2005) reported similar findings whereby point-light walkers elicited the N170 and N300. The first component showed greater sensitivity to upright than inverted figures, thus reflecting configural processing and automatic feedforward mechanisms. The N300, however, was not influenced by orientation, meaning that this stage was specific to biological motion analysis (Jokisch *et al.,* 2005). Yet not all data supports these conclusions. In one study, stick figures and point-light walkers (static and dynamic) were displayed as upright or inverted forms (White *et al.,* 2014). Although both orientations evoked stronger negativities in later components (denoted here as N2), this activity was identical for static and dynamic walkers. Such findings indicate that the N2 was primarily concerned with human form recognition and not biological motion *per se.* The authors then identified a subsequent component (MPP/VAN) occurring between 300 and 650ms which was thought to reflect action perception. Nonetheless, it is clear that the analysis of biological motion is a multiple-phase hierarchical process (Troje, 2008). These procedures occur via low-level visual mechanisms and are independent of top-down attentional control (Krakowski *et al.,* 2011).

A limitation of these ERP studies is that they only describe the neural response to bodily movements. Facial motion, however, facilitates social interaction by providing categorical and qualitative visual information (Bould & Morris, 2008; Hill & Johnston, 2001). Assuming that biological motion is a hallmark of social cognition (and impairment to such perception may underlie ASD symptomatology), it is important to extend our investigations to include faces. As it stands, very few EEG investigations have done this with neurotypical participants. Using apparent facial motion (i.e. static faces presented

to give the impression of movement), one study observed a larger N170 component in the posterior temporal cortex to mouth opening and averted eye gaze gestures (Puce *et al.,* 2000). Wheaton *et al.,* (2001) reported similar findings. Here, faces displaying real mouth opening (versus closing) movements evoked the largest ERP within 200ms post onset over temporoparietal locations. Puce *et al.,* (2003) and Rossi *et al.,* (2014) further corroborate this data. They show that mouth opening movements from natural and line drawn facial motion elicits a greater N170 response than mouth closing gestures. These temporal differences most likely reflect the underlying social significance of facial expressions (e.g., mouth opening to signal conversation) (Rossi *et al.,* 2014).

In the context of methodology, ERP components are identified by averaging across several trials so that any activity not phase-locked (evoked) to the stimulus is removed (Dawson, 1951). Yet this only represents part of the total neural response, meaning that any induced activity is disregarded (Rossi *et al.,* 2014). Observing the amplitude patterns of specific neural frequencies alleviates this issue. Accordingly, the current study examined facial motion processing by measuring changes occurring within the EEG alpha band (8-12Hz). These oscillations were chosen for study as they represent a neural signature of activity occurring within the occipital cortex (Berger, 1929). Indeed, alpha waves are suppressed during active visual perception (Toscani *et al.,* 2010). They appear to be synchronised with cyclic activity of the visual thalamic relay neurons, modulating signal transmission during early input stages (Lorincz *et al.,* 2009). Prestimulus alpha also fluctuates with the excitability of the visual cortex and is predictive of an imminent perception (Romei *et al.,* 2008; Van Dijk *et al.,* 2008).

In addition, occipital alpha indexes memory processes including those related to working memory loads and long-term stores (Jokisch & Jensen, 2007; Klimesch, 1997; Tuladhar *et al.,* 2007). Parieto-occipital alpha is further influenced by visual attention (Belyusar *et al.,* 2013; Capotosto *et al.,* 2009; Rihs *et al.,* 2007; Thut *et al.,* 2006). Alpha power is larger over visual cortices when attention is focused on the auditory part of an auditory-visual stimulus (Foxe *et al.,* 1998). In addition, participants show an interhemispheric difference in alpha amplitudes during the Posner cueing paradigm (Kelly *et al.,* 2006). The increase on the unattended side suggests alpha waves have a 'gating mechanism' (Pfurtscheller & Lopes da Silva, 1999). Such function may inhibit incoming sensory information in terms of its behavioral relevance (May *et al.,* 2012).

Several studies have observed alpha oscillations in response to static faces (Balconi & Pozzoli, 2008; Balconi & Mazza, 2009; Başar *et al.,* 2006). It has been shown that emotional faces increase alpha amplitudes at posterior occipital locations, whilst angry face stimulation specifically activates substrates over electrodes T5, P3 and O2 (Güntekin & Başar, 2007). Frontal alpha activity is also associated with previously formed concepts concerning negative emotional expressions, suggesting that the fronto-thalamic system is involved in the perception and evaluation of facial affects (Kostandov *et al.,* 2007;

Kostandov *et al.,* 2010). Regarding general static face perception, Hsiao *et al.,* (2006) found 4-25Hz activity in the middle occipital and occipitotemporal areas when participants viewed upright faces. Inverted faces, however, produced the most alpha enhancement in the right occipitotemporal area, indicating additional attentional requirements and increased synchrony between neuronal populations. Sakihara *et al.,* (2012) also found alpha, theta and beta suppression occurring over occipitotemporal areas during familiar, unfamiliar and own face perception. Such activity illustrates the structural and semantic encoding of facial information (Sakihara *et al.,* 2012).

At the time of writing, no published EEG study had directly examined posterior alpha suppression in response to whole-face natural human motion. Investigations here would significantly advance our understanding of how low-level visual mechanisms contribute to the processing of facial motion. It would also allow future comparisons to be made with populations who exhibit impairments in face perception and social cognition (e.g., ASD). In the current study, neurotypical participants were asked to discriminate between successive facial motion captures (one-back task) during EEG recordings. The stimuli were computer-generated averaged faces animated with motion sequences. These exhibited rigid (head rotations) and nonrigid (facial expressions) motion, including speech expressions and eye movements. The benefit of using such stimuli was that they depicted real human motion without the addition of confounding spatial cues.

The current study did not utilise inanimate (object) motion as a control as these comparisons involve many unrestrained differences in low-level stimulus properties (George *et al.,* 1999). Instead, orientation-inverted and luminance-inverted facial stimuli were used as these manipulations are known to affect face recognition. Inversion paradigms impair static face perception by disrupting configural processing (Valentine, 1998). The brain may also treat orientation-inverted faces in a similar manner to objects, considering the involvement of the lateral occipital area here (Pitcher *et al.,* 2011). Luminance-inverted faces also affect processing regardless of preserving normal face structure and spatial frequencies (Kemp *et al.,* 1996; Taubert & Alais, 2011). These negative images disrupt the N170 face-selective component and therefore early structural encoding (Itier & Taylor, 2002; Tomalski & Johnson, 2012). Together, these measures comprise an effective tool in evaluating facial motion perception.

2.2 Materials and Methods

Participants

Ethical approval was obtained from Brunel University. Participants were given a description of the study and written informed consent was obtained. A debriefing document was given to each participant after the experiment was terminated. Nineteen individuals (9 male, 10 female, age: *M* = 28.53 years, *SD* = 9.26, *Range* = 22 – 54 years) with normal or corrected-to-normal vision participated in this study. Sixteen participants were right handed, and three were left handed (or ambidextrous). None of the

sample had any previous history of neurological or psychological disorders. However, face-processing deficits have been observed in neurologically healthy adults (Le Grand *et al.,* 2004). Therefore, a static facial recognition test (Benton, 1983) was administered to participants to ensure intact face processing abilities. All the participants passed the Benton's test (Table 1).

Table 1. *Characteristics of the experimental group. Scores on the Benton's Test are out of 54 possible correct answers. A score over 41 indicates normal face recognition.*

	N	Mean Age (SD)	Benton Facial Recognition		
			% score	*Mean*	*Range*
Male	9	28.2 (6.89)	89.91	48.56	45-51
Female	10	28.8 (11.39)	90.93	49.10	45-51

Stimuli

The stimuli were taken from a video database developed by Hill and Johnston (2001) using motion capture technology. Using markers placed on major facial landmarks, motion was captured from 12 actors reciting simple question and answer jokes. These jokes allowed natural facial expressions (nonrigid motion), speech and head movements (rigid motion) to be captured. The motion sequences were then applied to a three dimensional computer-generated averaged head (taken from 100 men and 100 women) and outputted as 640 x 480 pixels, 25 frames-per-second movies. By using an average face on all sequences, facial motion could be measured independently from facial form. The appearances of all capture faces were therefore identical and only differed in the way they moved. An orientation-inverted and luminance-inverted version of each stimulus was generated in Matlab by manipulation and re-encoding of the original stimulus video file.

Procedure

Participants were seated in a Faraday cage in a dimly lit room. Observers viewed the dynamic stimuli on a computer screen. Viewing distance was 80cm, at which the distance of the 38cm x 30cm display subtended an angle of approximately 28º x 22º. The experiment consisted of 3 blocks, each with 50 trials; upright facial motion, orientation-inverted facial motion and luminance-inverted facial motion (Figure 1). Blocks were repeated 3 times in a counterbalanced order to avoid practice effects, fatigue or decreasing vigilance influencing the EEG waveform.

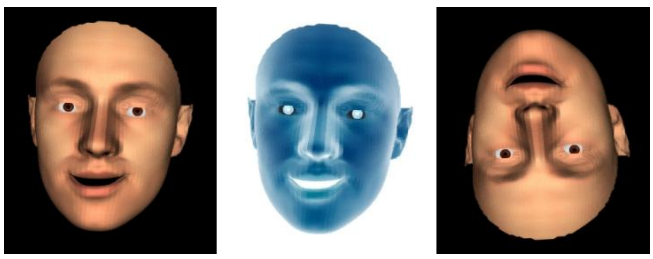

Figure 1. Upright, luminance-inverted and orientation inverted facial stimuli.

Participants completed a sequence discrimination task during the EEG recordings. This tested the participants' ability to differentiate between facial motion sequences that were presented in a continuous series. All animations were displayed for 3 seconds. A single animation was presented, and after an interstimulus interval (ISI) of 1 second, another animation appeared. During a second ISI, participants were required (according to pre-task instructions) to respond via the keypad, whether the 2 animations were the same (press 1) or different (press 2) from each other. This process continued throughout the testing period, such that they always judged whether the current animation was the same or different from the previous animation. The same format was used for all 3 conditions (upright, orientation-inverted and luminance-inverted).

EEG Recording and Analysis

EEGs were recorded with an average common reference from 64 Ag-AgCl electrodes (FP1, FPz, FP2, AF3, AF4, F7, F5, F3, F1, Fz, F2, F4, F6, F8, FT7, FC5, FC3, FC1, FCz, FC2, FC4, FC6, FT8, T7, C5, C3, C1, Cz, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPz, CP2, CP4, CP6, TP8, P7, P5, P3, P1, Pz, P2, P4, P6, P8, PO7, PO5, PO3, POz, PO4, PO6, PO8, CB1, O1, Oz, O2, CB2). The electrodes were filled with Quik Gel (Compumedics Neuromedical Supplies) and were placed according to the International 10/20 system. A horizontal electrooculogram (EOG) was recorded from electrodes placed on the outer canthi of both eyes. Vertical EOG electrodes were placed above and below the middle of the left eye. Impedances did not exceed 10KΩ. The EEG was amplified at a gain of 1000 and bandpass filtered at 0.1 – 100Hz. It was digitised at 1000Hz via a Synamps2 amplifier and Scan 4.4 acquisition and analysis software (Compumedics Neuroscan LTD).

Offline, a DC offset correction was applied to the raw waveform, and the time series was bandpass filtered at 0.1- 128Hz (24 dB/octave). A visual scan was conducted to mark 'bad' blocks and eye blink artifacts were removed by a principle components procedure. Using the cleaned EEG, an event file was created and used to epoch the data for each condition from -100 to 923ms (0ms = stimulus onset). Sweeps were baseline corrected (entire sweep) and amplitudes greater than ±75μV were rejected. An event-related band power analysis detected event-related frequencies within the alpha band. The data was bandpass filtered with a center frequency of 10Hz, and a half bandwidth of 2Hz (12dB/octave) within a moving 100ms window. The baseline, mid-point maximum and late-minimum amplitudes (and the latency in which they occurred), were detected and analysed.

Statistical Analysis

A repeated-measures ANOVA was used to test for differences between the alpha amplitudes elicited by each facial motion. *Time sample* (baseline, mid-point, late-minimum), *sequence type* (same, different) *face type* (upright, orientation-inverted, luminance-inverted), *hemisphere* (left, right) and

electrode site were the within-participant factors. A Bonferroni correction was applied to post-hoc contrasts. A repeated-measures ANOVA (*face type* x *hemisphere* x *electrode site*) was used to analyse differences in the latencies of alpha amplitudes produced by each face type. As *sequence type* did not yield any significant main effects, data was collapsed across these levels.

2.3 Results

The strongest alpha power was observed at parieto-occipital (PO7, PO5, PO3, PO8, PO6, PO4) and occipital (CB1, O1, O2, CB2) scalp locations. Amplitudes were observed at 3 time samples (*baseline* - 100 - 0ms, *mid-point* 300 - 500ms, and *late-minimum* 600 – 823ms). Observing data at 3 time-samples allowed a more detailed analysis to be made with regards to patterns of alpha activity post motion onset.

Grand Average Data

Data from one participant was excluded from the statistical analysis due to faults with the EEG recording system. At parieto-occipital (PO) and occipital (O) sites, upright facial motion increased alpha power before suppressing it. This pattern of activity did not occur for the other stimuli (Table 2).

Table 2. *Grand average amplitude and latency data for facial motion at PO and O sites.*

Site	Face type	Baseline*	Mid-point	Late minimum
PO	Upright	4.77μV at 0ms	4.94μV at 477ms	2.71μV at 733ms
	Orientation-inverted	4.52μV at 0ms	3.82μV at 466ms	2.80μV at 755ms
	Luminance-inverted	4.56μV at 0ms	4.22μV at 453ms	2.97μV at 734ms
O	Upright	5.24μV at 0ms	5.38μV at 443ms	3.48μV at 731ms
	Orientation-inverted	5.07μV at 0ms	4.27μV at 467ms	3.05μV at 754ms
	Luminance-inverted	5.17μV at 0ms	4.63μV at 460ms	3.40μV at 747ms

**Baseline amplitudes are considered the initial values of alpha.*

Amplitude Data

Facial motion (regardless of type) suppressed alpha power, as indicated by significant differences between the *time-sample* amplitudes (*O sites:* $F_{(2, 16)} = 32.45$, $p < .01$ and *PO sites:* $F_{(2, 16)} = 52.95$, $p < .01$). For PO data, simple contrasts indicated a significant difference between the late-minimum interval and baseline for all facial motion (*Upright:* $F_{(1, 17)} = 41.68$, $p < .005$; *Luminance-inverted:* $F_{(1, 17)} = 90.71$, $p < .005$; *Orientation-inverted:* $F_{(1, 17)} = 39.43$, $p < .005$). The difference between the recovery interval and baseline was significant for orientation-inverted faces only ($F_{(1, 17)} = 9.88$, $p < .05$). At PO and O electrodes, there was a significant main effect of *face type* on overall alpha power across the three time-samples ($F_{(2, 16)} = 3.97$, $p < .05$ and $F_{(2, 16)} = 4.67$, $p < .05$ respectively).

The amount of alpha suppression evoked by each facial motion only differed at PO sites, as revealed by a significant *time-sample* x *face type* interaction ($F_{(4, 14)} = 6.39$, $p < .01$). Simple contrasts showed

that this interaction was driven by a significant difference between upright and orientation-inverted faces in the mid-point time interval only ($F_{(1, 17)} = 11.64$, $p < .05$). See Table 3 for a summary of significant main effects and interactions.

Table 3. *Significant main effects and interactions at PO and O electrodes.*

Electrodes	Within-participant variables	*F*	*df*	*P*
PO	Time-sample*	52.95	2, 16	0.001
	Face type*	3.97	2, 16	0.040
	Time-sample x face type*	6.39	4, 14	0.001
	Electrode site	33.06	2, 34	0.001
	Time-sample x hemisphere	4.81	2, 34	0.014
	Sequence x face type x electrode*	4.89	4, 14	0.011
O	Time-sample*	32.45	2, 16	0.001
	Face type*	4.67	2, 16	0.025

**Taken from multivariate tests (Pillai's Trace) due to a significant Mauchly's test indicating that sphericity cannot be assumed.*

Latency data

Differences in the latencies of the peak alpha amplitudes were observed amongst the facial motion types (Table 4). *Face type* had a significant effect on the latency of the late-minimum amplitudes at PO sites ($F_{(2, 34)} = 3.44$, $p < .05$). Simple contrasts indicated that this was driven by a significant difference between upright and orientation-inverted facial motion ($F_{(1, 17)} = 6.27$, $p < .05$). Compared with other types, upright motion suppressed alpha at earlier latencies (733ms vs. 755ms for orientation-inverted stimuli). At O sites, the latencies of the mid-point amplitudes were significantly affected by *face type* ($F_{(2, 34)} = 4.57$, $p < .05$). Simple contrasts revealed a significant difference between the mid-point latencies for upright (443ms) and orientation-inverted (467ms) facial motion ($F_{(1, 17)} = 7.20$, $p < .05$).

Table 4. *Latency of mid-point peak and minimum amplitudes at PO and O electrodes.*

Latency of mid-point peak amplitudes				
Site	Within-participant variables	*F*	*df*	*P*
PO	Hemisphere x electrode	4.04	1.42, 24.07*	0.043
O	Face type	4.57	2, 34	0.018
	Upright vs. orientation-inverted	7.20	1, 17	0.016
Latency of late-minimum amplitudes				
PO	Face type	3.44	2, 34	0.044
	Upright vs. orientation-inverted	6.27	1, 17	0.023
	Electrode	10.15	2, 34	0.001
O	Hemisphere	8.58	1, 17	0.009

**Mauchly's test indicated that the assumption of sphericity had been violated. Degrees of freedom were therefore corrected using Greenhouse-Geisser estimates of sphericity.*

2.4 Discussion

Transient Alpha Increase

Unexpectedly, upright facial motion initially evoked an increase in alpha power over parieto-occipital and occipital scalp locations. The neural efficiency argument (Gauthier & Tarr, 1997) provides one interpretation for this result. Reflecting on our expertise, less information processing may be required for upright face perception (Diamond & Carey, 1986). This would certainly explain why the control faces evoked alpha suppression almost instantly; unfamiliar stimuli would require increased attentional effort and involvement of high-level cognitive resources (Cole & Ray, 1985; Kemp *et al.,* 1996). Yet this argument does not explain why upright facial motion subsequently suppressed alpha after this time point. Alternatively, the initial high alpha amplitude could reflect a 'gating' mechanism used to filter out task-irrelevant visual information (Okazaki *et al.,* 2014). In this case, form cues provided no additional information and were thus ignored. The subsequent suppression would therefore correlate with attention to motion cues when engaging in facial motion tasks. It is important to note, however, that this transient increase in alpha power following video onset could be due to the motion-onset ERP. As the data analysis was conducted using induced event-related bandpower measures, this point cannot be fully addressed. Future studies should utilise evoked synchronisation measures in order to observe a more distinct emergence of face-selective ERP components.

Posterior Activation to Facial Motion

With reference to the amount of suppression evoked by each facial motion, no difference emerged at occipital locations. This suggests that early visual processing occurs irrespective of orientation or luminance-reversal (Goffaux *et al.,* 2003; Kostandov *et al.,* 2010). This finding is in contrast to studies of static face perception. For example, Itier and Taylor (2002) reported that inverted and negative static faces affected early encoding, as demonstrated by a reduced N170 response. In the context of encoding and retrieval mechanisms, occipital alpha is suppressed when participants perceive famous (and thus familiar) faces compared to non-famous faces (Zion-Golumbic *et al.,* 2010). The authors suggested that face perception evokes interplay between semantic knowledge and episodic memory formation. In the case of biologically unfamiliar faces (orientation-inverted or luminance-inverted stimuli), we may expect less occipital alpha activity to occur. This effect was not found here. It is possible that early encoding processes remain unaffected by such visual manipulations, perhaps due to the detailed three-dimensional representation facial motion provides (O'Toole *et al.,* 2002). However, this view may be rejected as many studies do report a disruption in perceiving inverted point-light figures (Grossman & Blake, 2001; Hirai *et al.,* 2011; Jokisch *et al.,* 2005). To date, only one study has found a comparable response to upright and inverted walkers over the left occipital cortex (Pavlova *et al.,* 2004).

By contrast, upright facial motion reduced alpha more than control stimuli at parieto-occipital regions. A study comparing the ERP response to upright and scrambled point-light walkers also reported differences emerging over this region (Krakowski *et al.,* 2011). In addition, stronger alpha suppression following biological motion perception has been noted over the parieto-occipital cortex (Perry *et al.,* 2010). This enhanced activity may reflect a number of significant underlying processes. Firstly, the medial portion of the parieto-occipital cortex has been associated with attentional reorienting during cognitive-motor tasks (Ciavarro *et al.,* 2013). Other researchers extend this finding to the dorsal aspect of the parieto-occipital sulcus (Tosoni *et al.,* 2013). An increase in parieto-occipital activity may therefore indicate that higher attentional effort allocated to perceiving upright facial motion.

Secondly, the parieto-occipital cortex contains functional areas associated with the visual control of body effectors (Monaco *et al.,* 2011). Regions of the superior and medial portions play a critical role in proximal and distal aspects of reaching/grasping actions, pointing gestures, head movements and eye-gaze shifts (Fattori *et al.,* 2010; Rossit *et al.,* 2011; Tikhonov *et al.,* 2004). Motion selectivity has also been observed within this region (Stiers *et al.,* 2006), indicating the involvement of the dorsal visual stream (Blanke *et al.,* 2002). Perhaps observing upright facial motion, which included head and eye translations, activated a portion of these substrates. It is also possible that parieto-occipital electrodes are indirectly recording activity occurring within the posterior superior temporal sulcus (pSTS) - the neural substrate most commonly implicated in biological motion perception (Allison *et al.,* 2000). One study which found face-selective ERPs occurring over the parieto-occipital cortex to facial motion supports this idea (Puce *et al.,* 2000). There is also evidence that the STS may actually extend into the parieto-occipital and occipital regions (Matsumoto *et al.,* 2004).

The larger amount of suppression evoked by upright facial motion also occurred within the shortest latency at parieto-occipital sites. Such early processing could reflect a pop-out effect caused by familiar orientations (Jokisch *et al.,* 2005). If this was the case though, luminance-inverted faces would have also been processed just as quickly. Instead, automated feed forward systems may, in part, be responsible for the efficient processing of upright motion (Kawasaki *et al.,* 2012; Lehky, 2000). Yet, top-down computations should not be completely disregarded (Grinter *et al.,* 2010). For example, it has been shown that the perception of point-light body motion uses a feedforward and feedback functional loop between the right pSTS and left lateral cerebellum (Sokolov *et al.,* 2010).

Implications of the Current Study

The results reported here could have been influenced by sensorimotor alpha (mu rhythms) recorded over central electrodes. Mu rhythms index action planning and preparation within the somatosensory cortex (Mizuhara, 2012). They are suppressed and their power attenuated when one performs an action but also during the observation of biological movements (di Pellegrino *et al.,* 1992; Rizzolatti &

Craighero, 2004; Ulloa & Pineda, 2007). In the current study, participants responded via a button press after observing facial motion sequences. Such an experimental paradigm could have activated anterior systems. It should be noted, however, that central electrodes were analysed and no significant effects found. In addition, the current study did not use inanimate or scrambled motion as a control. Thus, it remains unknown whether differential activations would have occurred for any stimuli presented in unfamiliar contexts. However, the manipulations used here are known to disrupt configural and holistic processing, meaning that control stimuli may be processed in a manner similar to objects (Pitcher *et al.,* 2011). Accordingly, parieto-occipital activity is not reflecting familiarity but instead selectivity to upright facial motion.

2.5 Conclusion

The parieto-occipital cortex was more strongly activated by upright facial motion compared to other stimuli types. This reflects active visual perception, encoding/retrieval mechanisms and the involvement of areas responsible for visually controlling body effectors. In addition, upright facial motion activated these underlying structures within significantly shorter latencies relative to orientation and luminance inverted faces. Occipital activity, however, did not differ amongst the face types, suggesting early visual perception remains unaffected by manipulation. Unfortunately, it can only be speculated which parieto-occipital substrates are driving this selectivity to upright facial motion. Owning to the spatial limitedness of EEG, future experiments will be carried out using fMRI.

CHAPTER 3 Impaired Perception of Facial Motion in ASD

3.1 Introduction

The human body conveys an abundance of information necessary for mediating socio-emotional communication (Knappmeyer *et al.,* 2003). Bodily movements, facial expressions and eye gaze patterns allow the extraction of information from others. We can then use this to understand people's thoughts, intentions and moods (Koldewyn *et al.,* 2011). Without the ability to perceive this information, social interaction would be difficult. Autism Spectrum Disorder (ASD) is a pervasive developmental condition characterised by a severe impairment in social interaction. It is possible that this symptom arises, in part, from a fundamental deficit in perceiving human movement.

Several behavioural studies have reported impairments in perceiving biological (body) motion in participants with ASD. Children with ASD commit more errors than neurotypical controls when indicating whether a brief point-light animation represents a body or not (Blake *et al.,* 2003). Similarly, deficits are evident when participants with ASD attempt to identify the emotion portrayed by point-light walkers (Atkinson, 2009; Hubert *et al.,* 2007; Parron *et al.,* 2008). Reduced accuracy on these tasks has been associated with increased reaction times and shorter fixation times (Nackaerts *et al.,* 2012). Further, participants with ASD are less accurate than controls when indicating whether a hand performing sinusoidal actions (minimum jerk) moved in a natural or unnatural way (Cook *et al.,* 2013). They also experience difficulty orienting to a point-light pointing gesture (Swettenham *et al.,* 2013). Pointing, however, facilitates shared attention processes and is extremely important for both social and linguistic functions (Brooks & Meltzoff, 2008). These findings suggest that the perception of emotional and non-emotional biological motion is weakened in ASD.

Conversely, other researchers have reported intact biological motion mechanisms in ASD. Saygin *et al.,* (2010) presented participants with point-light displays (walking figures, translating triangles, or translating unfamiliar shapes) embedded in noise and asked them to determine the direction of movement. Participants with ASD performed similarly to controls across all three tasks. Murphy *et al.,* (2009) also showed that participants with ASD could successfully identify the direction in which a point-light walker (embedded in noise) was moving in. The authors suggested that participants with ASD were able to integrate local motion cues to produce a coherent perception of biological motion. On the other hand, a recent event-related potentials study actually observed atypical processing of local motion in ASD (Hirai *et al.,* 2014). Unlike the controls, participants with ASD evoked the same electrophysiological response to both scrambled and intact body motion, indicating over-sensitivity to local motion signals. It is therefore possible that while no behavioural differences emerge between the experimental and control group, significant abnormalities within the underlying neural circuitry could still exist.

Nevertheless, inconsistent data could reflect an experimental bias caused by testing different age groups. Studies conducted with children have consistently reported a deficit in biological motion processing (e.g., Blake *et al.,* 2003; Klin *et al.,* 2009), whereas the adult data is less conclusive. The perception might improve with chronological age, perhaps as older participants with ASD learn to perform compensatory mechanism (Hubert *et al.,* 2007; Van Boxtel & Lu, 2013). Yet Annaz *et al.,* (2010) reported data which contradicted this hypothesis. Taking a longitudinal approach, these authors tested children with typical development and ASD over a five year period (5-10 years old) on their ability to identify intact and scrambled point-light walkers. Unlike the controls, sensitivity to natural biological motion in ASD remained constant relative to chronological age and both verbal and nonverbal mental age. Alternatively, factors such as high symptom severity (Blake *et al.,* 2003) or poor general intelligence (Jones *et al.,* 2011; Rutherford & Troje, 2012) could affect biological motion perception in ASD and thus contribute to these discrepant findings.

While the existing data has been highly informative, there is a paucity of research exploring facial motion perception in ASD. The face facilitates social interaction by providing categorical (identity, gender, age) and qualitative (emotions, intentions, thoughts) visual information (Blake & Shiffrar, 2007). If we are to assume that biological motion deficits are accountable for impairments in social cognition, then it is essential we actually investigate this using moving faces. At the time of writing, there were only a few studies which had specifically focused on facial motion processing in ASD. Uono *et al.,* (2009) reported that the integration of dynamic emotion and gaze direction cues, needed for joint attention, was impaired in Asperger's Syndrome. Rating the naturalness of facial expressions which differed in speed was also problematic for participants with ASD (Sato *et al.,* 2013). The authors suggested that this reflects a dysfunction in the earliest visual component of processing. Recently, Enticott *et al.,* (2014) found that neurotypical participants were more accurate than participants with ASD when recognising anger and disgust from dynamic facial displays. Interestingly, decreasing the speed of video presentations has been shown to facilitate the individual's with ASD performance on facial recognition and imitation tasks (Gepner *et al.,* 2001; Tardif *et al.,* 2007).

These data therefore suggest that participants with ASD cannot process dynamic facial information relating to eye gaze or emotion. However, one study has failed to report a poor perception of facial motion in a sample of participants with Asperger's Syndrome (Miyahara *et al.,* 2007). Here, the experimental and control group exhibited comparable reaction times and accuracy rates when recognising facial affects (happiness and disgust) from dynamic videos. Yet at the individual level, statistical analyses revealed that participants with Asperger's were not as sensitive to the happy face advantage (i.e. recognising this emotion more effortlessly than others) as were the controls. On the

basis of this result, it appears that participants with Asperger's do have an impaired perception of facial motion, at least to some degree.

The perception of facial motion in ASD is beginning to attract the attention of researchers. Much of this cognition, however, is still relatively unexplored. For example, very little is known about how participants with ASD perceive categorical information from facial motion. Studies with neurotypical participants have shown that facial motion facilitates discriminations based on gender, identity and age (Berry, 1990b; Hill & Johnston, 2001). These studies demonstrated that dynamic information provides a better structural 3D depiction of the face by increasing view-points and/or conveying idiosyncratic movements (O'Toole *et al.,* 2002). Facial motion captures were therefore implemented in the current study. These stimuli depicted real human motion (rigid and nonrigid) in the absence of confounding spatial cues. Participants were asked to discriminate between sequences, identify different unfamiliar individuals and categorise genders from these stimuli. As the appearances of animations were identical to each other, judgments were based solely on differences in motion patterns.

In order to observe differences between the ability of participants with ASD and neurotypical controls to perceive facial motion, presentations varied between upright and inverted stimuli. Studies using static faces have demonstrated that inversion affects face recognition by disrupting configural processing and early structural encoding (Valentine, 1998). As a result, accuracy on such face perception tasks is significantly reduced. Previous research has not found this effect in ASD (Langdell, 1978; Webb *et al.,* 2012), suggesting a failure to utilise configural strategies and a reliance on feature-based processing (Spezio *et al.,* 2007). Recent reviews, however, have highlighted inconsistencies surrounding this manipulation in ASD (Weigelt *et al.,* 2012). For example, Hedley *et al.,* (2014) found that although participants with ASD performed worse than controls on the recognition task, they were similarly sensitive to face inversion effects.

In the current study two questions were addressed: (1) are participants with ASD able to perceive facial motion, and use such information when making judgments about sequence, identity or gender; and (2) is the performance of participants with ASD unaffected by inversion paradigms, therefore confirming a feature-based processing of faces. Answering such questions might shed light on whether an impaired perception of facial motion contributes to the social cognitive impairments seen in ASD.

3.2 Methods and Materials

Participants

Ethical approval was obtained from Brunel University. All participants gave written informed consent prior to the study and received a debriefing document following their participation. Two groups of adults participated in the present study (see Table 5): 14 individuals with ASD (11 male, 3 female, age: *M* =

33. 85, *SD* = 11.23) and 14 neurotypical controls (7 male, 7 female, age: *M* = 31.14, *SD* = 13.01). The participants with ASD had received a clinical diagnosis of an Autism Spectrum Disorder (subtype = Asperger's Syndrome, DSM-IV-TR code: 299.80) from a clinical psychiatrist. Exclusionary criteria for participants with ASD and neurotypical controls included schizophrenia, genetic disorders, seizures, birth defects and significant visual impairments. For control participants, these criteria also included developmental abnormalities and having first degree relatives with ASD.

Groups were matched on age and scales of non-verbal analytic intelligence, as measured by the Ravens Standard Progressive Matrices (Raven *et al.,* 2003). Such measures of IQ were used as the participants with ASD had already received standard IQ testing within a year prior to the current study. It was therefore necessary to use other measures to avoid practise effects. Both groups of participants were also tested on their ability to perceive static faces from the Benton's Facial Recognition battery (Benton *et al.,* 1983). The scores for both groups were within the normal range, suggesting that any difficulties experienced during experimental testing would indicate a specific problem in facial motion perception, rather than a generalised impairment in face processing *per se* (see Table 5).

Table 5. *Characteristics of adults with ASD and the neurotypical control group.*

		Controls	ASD	*P*-value
		n = 14	*n* = 14	
Age	*Mean*	31.14	33.85	0.570 ns
	Range	21 - 56	22 - 51	
Standard Progressive Matrices*	*Mean*	49.00	42.31	0.070 ns
	Range	31 - 56	19 - 52	
	% score	82	71	
Benton Facial Recognition*	*Mean*	47.79	45.79	0.287 ns
	Range	45 - 54	36 - 52	
	% score	89	85	
Autistic Quotient*	*Mean*	14.45	26.40	0.001
	Range	12 - 20	21-36	

** Maximum possible scores for the Standard Progressive Matrices = 60; for the Benton Facial Recognition test = 54; scores between 11 and 22 on the Autistic Quotient were considered average.*

Stimuli

The stimuli were taken from a video database developed by Hill and Johnston (2001) using motion capture technology. Using markers placed on major facial landmarks, motion was captured from 12 actors reciting simple question and answer jokes. These jokes allowed natural facial expressions (nonrigid motion), speech and head movements (rigid motion) to be captured. The motion sequences were then applied to a 3D computer-generated averaged head (taken from 100 men and 100 women) and outputted as 640 x 480 pixels, 25 frames-per-second movies. By using an average face on all sequences, facial motion could be measured independently from facial form. The appearances of all motion capture faces were therefore identical and only differed in the way they moved. An orientation-

inverted version of each stimulus was generated in Matlab by manipulation and re-encoding of the original stimulus video file.

Procedure

The dynamic face stimuli were presented using an LCD display with a resolution of 1024 x 768 and 60Hz refresh rate. Viewing distance was approximately 60cm, at which distance the 30cm x 22.5cm display subtended an angle of 26.6° x 20.6°. The height of the average face was approximately 10.5°. Instructions were given verbally and the experimenter recorded participants' verbal responses manually. Each participant took part in all of the experimental conditions.

There were 3 experimental tasks (sequence, identity and gender discrimination), each with 2 manipulations (upright and inverted). Each condition had 21 trials, plus 8 attention control trials. The first condition was the *sequence discrimination task.* Participants viewed a single facial animation displayed in the centre of the screen. They then viewed the same video again, plus a completely different animation, shown side-by-side on the screen. All animations were presented for 5 seconds. Using a two-alternative forced choice procedure, participants had to indicate which stimuli (left or right on the screen) were present in both trials. A similar format was used for the *identity discrimination task.* A single facial animation was presented, followed by another 2 animations. One of the test animations was from the same actor telling a different joke (the correct response), and the other was of a second actor telling another joke. The *gender discrimination task* required participants to view a single animation, and respond whether it was male or female. Conditions were randomised to avoid familiarity effects.

To ensure maximal attention to the stimuli throughout the testing period, attention-control trials were included. This provided a conservative criterion for rejecting any data where there was a possibility of non-perceptual factors (fatigue, intermittent confusion) influencing the performances (Spencer & O'Brien, 2006). On every fourth trial, the correct responses were indicated with a blue arrow placed above the animation. The arrow was present at the beginning of the trial, and remained on the screen until the participant made their response. Participants were aware that the arrow indicated the correct answer. The responses to these trials were not included in any subsequent analysis. All participants completed the attention control trials without error and no data was discarded.

3.3 Results

The proportion of correct responses made by participants with ASD and neurotypical controls for each task is presented in Figure 2. One-sampled t-tests were used to compare the performance in each condition with the chance response rate of 10.5 (50%). For the ASD group, performance was not significantly above the chance level of .05 (Bonferroni corrected) in the (1) inverted identity

discrimination; (2) upright gender discrimination; and (3) inverted gender discrimination tasks. The control group did not perform above chance on the inverted gender discrimination task. A below chance performance might indicate random or guessing responses made by participants and therefore bogus results.

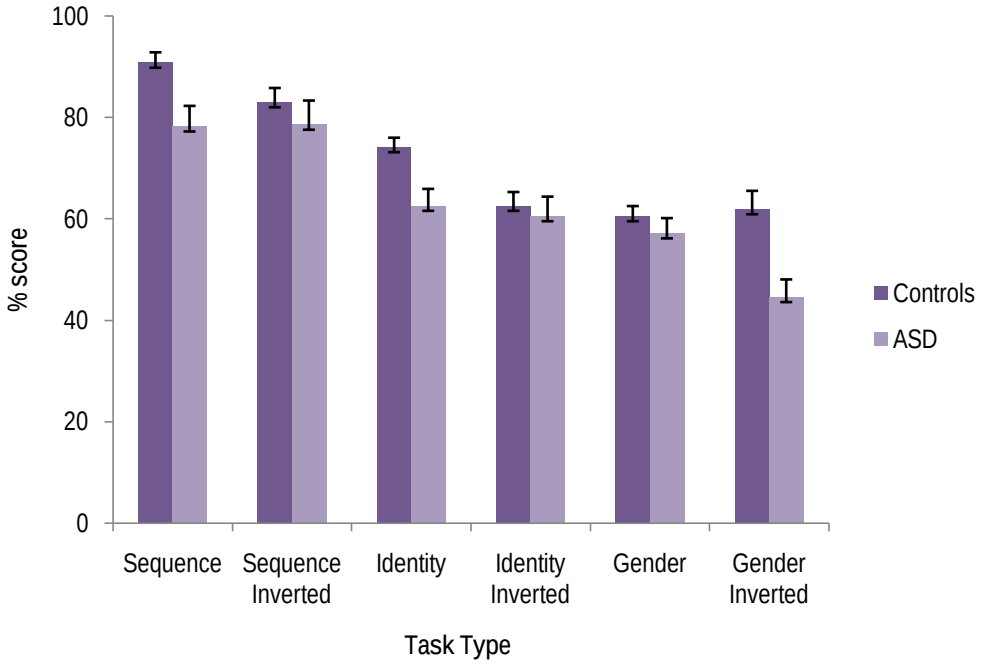

Figure 2. Proportion of correct responses (and SE) on each task for the control and ASD participants.

A mixed design ANOVA indicated a significant three-way interaction between *task type* (sequence, identity, gender) *orientation* (upright, inverted) and *group* (ASD, neurotypical controls) ($F_{(2, 52)} = 9.97$, $p < .001$). A further significant interaction between *orientation* and *group* on the facial motion sequence ($F_{(1, 26)} = 5.24$, $p = .030$), identity ($F_{(1, 26)} = 4.83$, $p = .037$) and gender ($F_{(1, 26)} = 9.07$, $p = .006$) discrimination tasks was also observed.

A follow up one-way ANOVA revealed a significant difference between the performance of the ASD and control group on the upright sequence ($F_{(1, 26)} = 7.73$, $p = .01$) and upright identity ($F_{(1, 26)} = 9.16$, $p < .01$) discrimination tasks. Compared to control participants, the ASD sample made more errors during these tests. There were no significant differences between ASD and control participants on the upright gender discrimination task ($p > .05$). However, the difficulty of this task was such that performance was above chance only for the control group in the upright condition.

The same analysis was applied to data from inverted conditions (Table 6). There were no significant differences between the ASD and control group for inverted sequence ($F_{(1, 26)} = 0.90$, $p > .05$) and identity discrimination tasks ($F_{(1, 26)} = 0.19$, $p > .05$). Inverted facial motion affected the controls,

decreasing their correct response rate. For participants with ASD, there was no difference in performance on upright and inverted conditions. Inverting the stimuli during gender discrimination tasks did however produce a significant difference between the control and ASD group ($F_{(1, 27)} = 11.89$, $p = .002$). This finding may be discounted though by the below chance performance evident in both groups.

Table 6. *Mean scores (standard deviations) and results from a one-way ANOVA.*

Variable	Mean (SD)		Differences between groups (One-way ANOVA)		
	ASD	Controls	*F*	*df*	*P*-value
Sequence	16.43 (3.18)	19.07 (1.59)	7.73	1, 26	0.010
Sequence Inverted	16.50 (2.93)	17.43 (2.21)	0.90	1, 26	0.352
Identity	13.14 (2.63)	15.57 (1.45)	9.16	1, 26	0.006
Identity Inverted	12.71*(3.05)	13.14 (2.14)	0.19	1, 26	0.671
Gender	12.00*(2.35)	12.71 (1.59)	0.89	1, 26	0.355
Gender Inverted	9.36* (2.74)	13.00*(2.68)	11.89	1, 26	0.002

**Indicates any result not above chance.*

3.4 Discussion

Impairments in perceiving biological motion has been suggested to underlie the social cognitive deficits in ASD (Herrington *et al.,* 2007). In the current study, these investigations were extended to facial motion perception, examining whether participants with ASD could use these cues to make specific categorisations. Participants therefore engaged in sequence, identity and gender discrimination tasks.

The current findings indicate that although participants with ASD were able to recognise static faces from the Benton's test, they were significantly less accurate than the controls when discriminating between upright sequences of facial motion. They were also significantly impaired on tasks requiring them to use upright facial motion as a cue for identity. An inability to recognise a number of different individuals from basic motion patterns may significantly impact social cognition in ASD. Moreover, unlike the control group, participants with ASD did not show an inversion effect in either task. Studies with neurotypical participants, however, show that motion information is processed configurally by a system tuned to upright faces, rather than by extraction of low-level cues (Hill & Johnston, 2001; Watson *et al.,* 2005). It would appear then that the neural mechanisms responsible for facial motion perception are weakened in ASD. This finding is comparable to other investigations which have utilised point-light body motion stimuli (Koldewyn *et al.,* 2011; Swettenham *et al.,* 2013).

Poor attentional abilities do not appear to be at the root of the problem as the participants with ASD scored correctly on the attention-control trials in each experimental condition. Incompetent cognitive skills can also be dismissed; all participants passed the Standard Progressive Matrices test within the typical range and understood the tasks well. Perhaps the impairment in facial motion perception arises

from problems in configural processing (Blake *et al.,* 2003). Participants with ASD may focus heavily on a particular and perhaps trivial feature, at the expense of global motion (Behrman *et al.,* 2006). There is some evidence to support this view. Van Boxtel and Lu (2013) measured accuracy on a central counting paradigm while task-irrelevant biological movements were presented in the periphery view of participants with low and high autistic traits. Stimuli were either intact or spatially scrambled. Participants with fewer autistic traits were found to involuntarily process global aspects of biological motion even when it negatively affected their central task performance. However, participants with high autistic traits did not show this attentional distraction, performing identically on the central task in the scrambled and intact conditions. An absence in configural (or global) processing would certainly support the indifference to orientation present in the current sample of participants with ASD. Engaging more in featural or local processing would by-pass the disruption caused by inverted facial motion (Webb *et al.,* 2012).

The impairment in perceiving facial motion could also be caused by dysfunctions in the earliest stage of visual processing. For example, Robertson *et al.,* (2014) found that the global motion processing deficits in ASD was attributed to a dysfunction of V1 and MT+/V5. Sato *et al.,* (2012) also reported a weakened bi-directional connectivity between V1, the middle temporal gyrus and inferior frontal gyrus when participants with ASD viewed dynamic displays of facial emotion. It is therefore possible that the transmission or integration of information from early visual areas to substrates involved in social cognition is weakened in ASD (Volkmar & Juraska, 2011; Zilbovicius *et al.,* 2006). This would explain why the superior temporal sulcus - a structure known for its involvement in biological motion processing - is often hypoactivated in participants with ASD (Pelphrey *et al.,* 2005; Zilbovicius *et al.,* 2006; Redcay, 2008). This neural deficit could underlie the impairment in facial motion perception seen in the current study.

The experimental and control group did not differ in their performance on *upright gender* discrimination tasks. This result is discussed in reference to the stimuli set. Some of the facial motion captures appeared to be impassive or expressionless. Female faces, however, are typically more animated during interaction than are male faces (Berry, 1991). For the control group, a higher percentage of animations may have therefore been incorrectly judged as male. This larger proportion of incorrect answers would then be more comparable with the experimental group, who seemed to completely guess answers as indicated by a below chance performance. The participants with ASD also showed an inversion effect during this task. This does not appear to be a genuine effect due to their below chance performance.

It is possible that the data is running into a floor effect on gender discrimination tasks. However, a similar study which looked at discriminating genders from facial motion found that healthy controls

could only do this for 68% of the trials (Berry, 1991). Such result is comparable to the 61% found in the current study. Hill and Johnston (2001) also reported a just above chance performance on gender discrimination tasks in neurotypical participants. More recently, one study found that while observers were able to correctly recognise genders 80% of the time, accuracy was higher for trials with static faces relative to facial videos (Thornton *et al.,* 2011). These findings demonstrate that accurate gender identification relies on the presence of both facial motion and characteristic structural form cues.

3.5 Conclusion

The current data indicates that participants with ASD are significantly less accurate than neurotypical controls on tasks discriminating facial motion sequences and different identities based on characteristic facial motion. The impairment may be caused by faulty configural mechanisms, which in turn would explain why the current ASD group were less sensitive to facial inversion. A weakened perception of facial motion could also be due to a dysfunction within the dorsal visual pathways leading to key biological motion substrates (e.g., the STS) or in the actual substrates themselves. These speculative points will be addressed in subsequent chapters which utilise fMRI in order to observe the neural basis of facial motion perception in ASD. Before doing so however, new facial motion stimuli need to be created. The animations currently implemented sometimes appeared inexpressive or unnatural, and could have contributed to the insignificant gender discrimination result. The next chapter describes a new stimuli method and provides data which validates its use in studies of human face perception.

CHAPTER 4 Categorising Identities from Facial Motion

4.1 Introduction

The mechanisms involved in facial identity recognition have been widely studied in both psychology (Bindemann *et al.,* 2013) and neuropsychology (Pitcher *et al.,* 2007; Rhodes *et al.,* 2009*;* Solomon-Harris *et al.,* 2013). While these investigations have been highly informative, many of them utilise static stimuli such as photographs, line drawings or CCTV images. Human faces, however, are intrinsically *dynamic* (Calder *et al.,* 2009). Verbal communication and emotional expressions occur via spatially distorting specific facial muscles. It is this continuous series of facial movement that provides an abundance of information necessary for social cognition (Knappmeyer *et al.,* 2003).

Knight and Johnston (1997) were among the first to consider how movement influences the identity recognition of contrasted-reversed famous faces. They found accuracy to improve only when faces were displayed dynamically relative to a single static image. Later studies, implementing other types of impoverished stimuli (threshold processed videos, blurred/pixelated clips or limited frame sequences), also reported a beneficial effect of motion during familiar face recognition (Lander *et al.,* 1999; Lander & Bruce, 2000; Lander *et al.,* 2001). This suggests that motion provides 3D information concerning face structure, but also prompts the recognition of idiosyncratic movements during sub-optimal viewing conditions (O'Toole *et al.,* 2002). Other researchers argue that this does not necessarily reflect a true dynamic effect though, and recognition might actually improve because the number of static frames contained within a moving sequence increases (Lander & Chuang, 2005). Lander *et al.,* (1999) have, however, shown that when the same frames were displayed either as a static array or animated sequence, identity recognition was still significantly higher for the moving sequence.

Several investigations have sought to examine which features of facial movement drive this increase in perception. Faces move in rigid (transient changes in head orientation) and nonrigid (expressional changes) manners. Both these categories improve identity recognition (Bruce & Valentine, 1988). Pike *et al.,* (1997) required participants to learn unfamiliar faces from static pictures (single and multiple sequences) or dynamic clips exhibiting rigid movement. At test, a single static image was shown and the task was to decide if the face was present in the previous learning phase. The authors found that identity recognition was significantly more accurate for faces initially presented as rigid motion sequences. Similarly, Thornton and Kourtzi (2002) observed a matching advantage for prime images of nonrigid motion (short video sequences) relative to a single static image. The benefit of nonrigid motion appears to exist regardless of task type (sequential matching vs. visual search) or viewpoint (Pilz *et al.,* 2006).

Other researchers have failed to observe advantages for faces viewed in motion over static pictures (Bonner *et al.,* 2003; Lander & Bruce, 2003; Lee *et al.,* 2010). Christie and Bruce (1998) found no improvement in the recognition of unfamiliar faces exhibiting rigid motion (shaking and nodding) compared to multiple static views. Lander and Chuang (2005) later replicated this finding using degraded movies of familiar and famous faces moving rigidly. Discrepant data could reflect an experimental bias caused by testing different age groups. For example, younger adults performed better than older adults when matching a learned dynamic (rigid or nonrigid) face to a static test image (Maguinness & Newell, 2014). Otsuka *et al.,* (2009) suggests that adults benefit less from motion as their perceptual abilities are already optimal. In younger participants however, face processing systems are less developed and need the additional data that facial motion provides.

Alternatively, the type of stimuli implemented across studies could contribute to inconsistent findings. Many use unnatural displays such as edited videos of image sequences (e.g., Lander & Bruce, 2003) or synthetic faces depicting computer-stimulated motion (e.g., Lee *et al.,* 2010). These representations of facial movement may not fully capture the mechanisms underlying its perception (Schultz & Pilz, 2009). Those who do utilise naturalistic videos often do not control for irrelevant non-motion data or residual spatial cues. For example, Lander and Bruce (2000) displayed video clips of people sometimes shown from either the shoulders or waist upwards. The addition of such information could, however, confound perception. To address this, Hill and Johnston (2001) first described a method to explore motion-based information independently of other cues. Facial animations were generated by applying the motion captured from 12 actors to the same 3D computer-generated (CGI) face. The technique also allowed the authors to separate rigid and nonrigid motion, thus evaluating their contributions to the categorisation of identity and gender respectively. These stimuli have since been successfully implemented in other studies examining the discrimination of individual faces (O'Brien *et al.,* 2014; Spencer *et al.,* 2006), viewpoint dependence (Watson *et al.,* 2005) and neural correlates of facial motion perception within the visual cortex (Girges *et al.,* 2014).

Yet it was highlighted in Chapter 3 that these representations of facial motion sometimes appeared impassive or expressionless. In turn, this could have potentially reduced the amount of socio-emotional information available for successful recognition (O'Brien *et al.,* 2014). It was therefore important to develop facial motion captures which resolved this issue. Building upon the work of Hill and Johnston (2001), recent developments in marker-less technology were exploited to generate accurate and realistic models of facial movement. Because this method did not require markers, a range of motion varying in magnitude (i.e. subtle to explicit) was captured from all facial regions. This facilitated in keeping the stimuli as authentic and ecologically valid as possible. Thus, simultaneous sequences of rigid and nonrigid motion (including speech expressions and eye blinks or gaze shifts) were recorded

from real actors reciting novelty poems. This was then applied to a standard CGI face so that motion perception could be measured separately from spatial cues.

To assess the stimuli and their ability to convey socio-emotional information, participants completed two tasks similar to those employed by Hill and Johnston (2001). In a video discrimination paradigm, a target video had to be matched to two subsequently presented animations. One was of the same target, the other a completely different foil animation. In the identity discrimination condition, the same experimental format was used. However, the two options were either of the original actor reciting another poem or a completely different actor. The task was to choose the same actor. As the appearances of animations were identical to each other, judgments were based solely on differences in motion patterns. If facial motion is indeed a cue for identity, these tasks should be completed with minimum error rates.

Facial motion captures were also presented upside down. Inversion paradigms affect static face recognition by disrupting configural processing and early structural encoding of facial features (Itier & Taylor, 2002; Valentine, 1988). A similar effect has been found for moving faces, in which inversion impairs the ability to accurately discriminate gender and identity (Hill & Johnston, 2001). While such data suggests facial motion is perceived configurally, other researchers argue it utilises part-based processing and therefore by-passes the inversion effect (Knappmeyer *et al.,* 2003; Xiao *et al.,* 2012). The current study aimed to investigate the inversion effect further by using accurate and realistic depictions of facial motion.

4.2 Methods and Materials

Participants

Twenty individuals (6 male, 14 female, age: *M* = 33.45 years, *SD* = 12.15, *Range* = 23 - 58 years) with normal or corrected-to-normal vision took part. Eighteen participants were right handed, and 2 participants were left handed (or ambidextrous). None of the sample had any history of neurological or psychological disorders. Participants were given a description of the study and written informed consent was obtained. They were debriefed after the experiment was terminated.

Stimuli Creation

Fifteen non-professional human actors (7 male, 8 female) were required to recite extracts from 6 short novelty poems (total of 90 different performances). Each poem contained similar number of words and took approximately the same time to read. The extracts ranged in emotional content, therefore eliciting a variety of different natural facial expressions (nonrigid motion), mannerisms, head movements (rigid motion) and speech. Before recording, actors had a practise trial run to ensure they were familiar with the content and spoke clearly, fluently and at an even pace.

A Kinect for Windows v2 sensor and Software Development Kit (SDK) captured the facial motion in 3D without the use of facial markers. The device featured an RGB camera (8-bit VGA resolution, 640 x 480 pixels) with a Bayer colour filter and both infrared and monochrome CMOS depth sensors (11-bit depth VGA resolution, 640 x 480 pixels, 2,048 levels of sensitivity). As the sensor captured the 3D motion, images were reconstructed (via *Light Coding* scanner systems) and directly live streamed into a motion tracking software (FaceShift Studio 1.1 - www.faceshift.com) at 30 fps (Figure 3). Motion was tracked in real-time ensuring high accuracy.

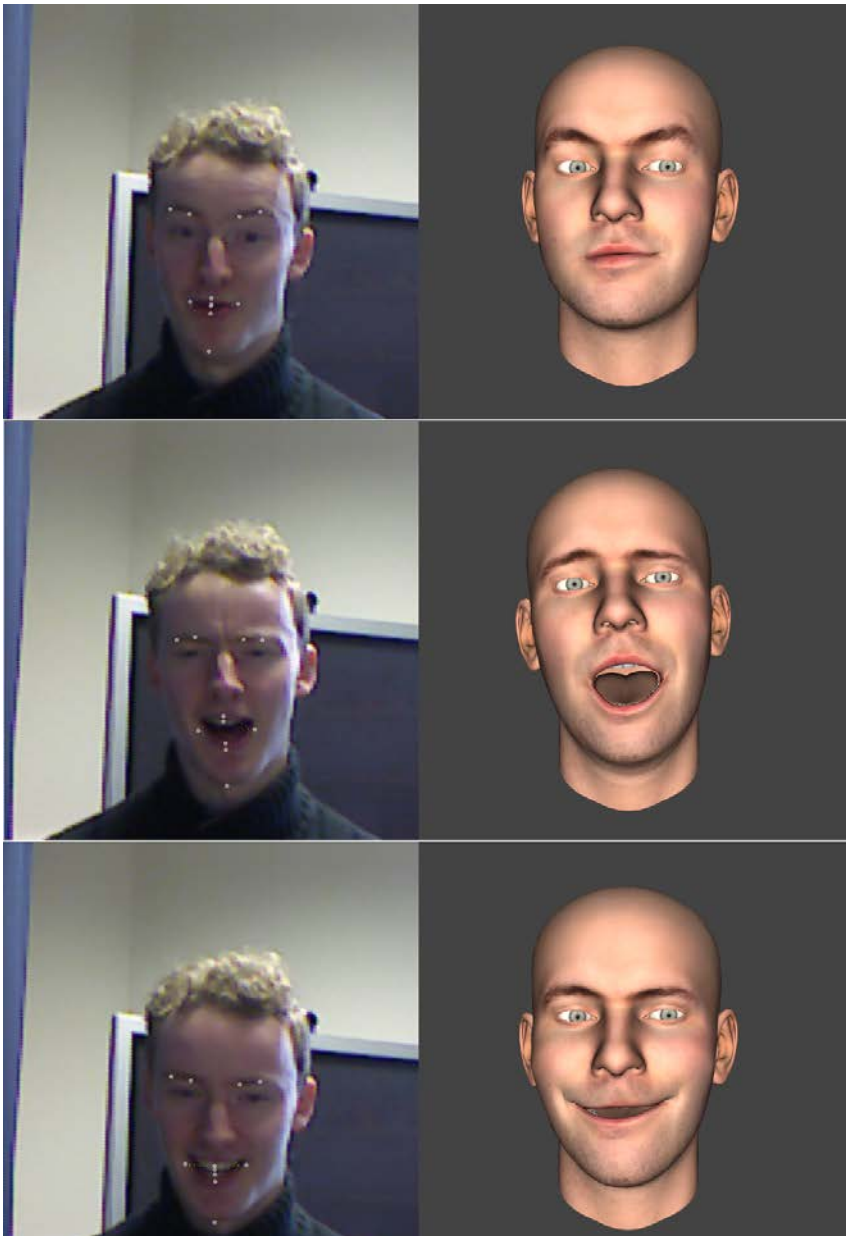

Figure 3. Example of how the motion was tracked using the Kinect Sensor and FaceShift studio. The left panel of screenshots show the real actor communicating. The right panel shows how the real motion is mapped onto an avatar in FaceShift. Note that this avatar was not the final model used in the experiment.

Using FaceShift, actors were first asked to elicit 23 training facial expressions prior to real motion recordings (neutral, open mouth, smile, brows down, brows up, sneer, jaw left, jaw right, jaw front, mouth left, mouth right, dimple, chin raise, pout, funnel, frown, m phoneme, grin, cheek puff, chew, lip down, eye blink left and eye blink right). Scanning these set of expressions enabled the program to calibrate each actors' motion and create a personalised avatar used for accurate motion tracking. Forty-eight blendshape parameters were tracked in total, meaning that emotions of all magnitude, eye-gaze (including eye blinks) and head pose were captured. Optimal recordings were best achieved by actors being seated 60cm away from the sensor (sensors angular field of view = 57^{o} horizontally and 43^{o} vertically). Actors were allowed to adjust their seating position and move in their chair during recordings.

Offline, each complete performance was imported into a 3D CGI rendering and animation application (Blender 2.66 - www.blender.org) as a .bvh file. These files contained 35 motion data points representing major facial regions (eyes, nose, chin, mouth, forehead, cheeks and ears). These points were all connected to a common reference point (neck bone) which controlled any rigid motion present in the motion sequence. The reference point also preserved the relative spatial structure between each point so that they all moved correctly in relation to one another. The motion data was then 'parented' to a greyscale computer-generated 3D face model[1] to begin the rigging process (Figure 4). Before this could happen, each individual motion point had to be readjusted to fit the computer-generated face. This was done by visual realignment and using a technique called ‘snapping’ which placed each point on the surface of the models skin. Once attached, the points essentially pulled and distorted the face into the specified motion pattern originally recorded from the Kinect Sensor (Figure 5). Any performances which did not map correctly onto the CGI faces or contained many artefacts were discarded from the database.

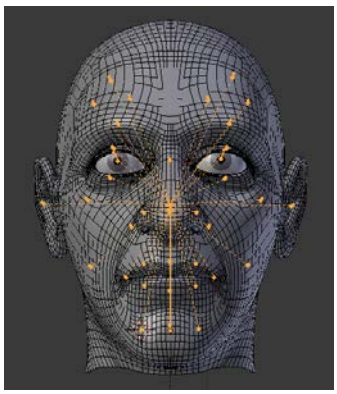

Figure 4. Computer-generated face model with the motion data points attached to the major landmarks.

[1]The computer generated model was created by Kent Trammel, and available online to download from http://cgcookie.com/blender/author/theluthier/. The model was edited to appear androgynous.

The greyscale face model was used for all 90 performances, allowing motion-based information to be measured independently from spatial cues. The appearances of all motion capture faces were identical to each other and only differed in the way they expressed motion. Each animation was encoded in h.264 format as an MP4 file. An orientation-inverted version of each animation was produced by rotating the stimuli along an 180º axis. None of the stimuli contained audio information.

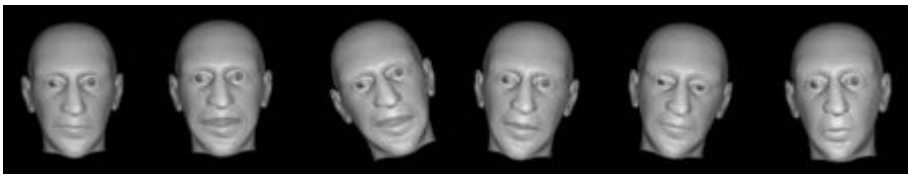

Figure 5. Screenshots of final stimuli.

To ensure the stimuli still represented the actual motion recorded from the original actors, a small preliminary experiment with a different set of participants (*N* = 15, 7 male, 8 female, age: *M* = 33.20, *SD* = 12.04) was performed. Participants observed a real video recording, followed by 2 facial motion animations presented side-by-side. Using a two-alternative-forced-choice procedure, the task was to indicate which animation represented the real video. This carried on for 20 trials. On average, participants scored 18.40 out of 20 possible correct answers (*SD* = 1.30, *Range* = 16-20, *Percentile score* = 92%). A one-sampled t-test confirmed an above chance performance (50%), $t_{(14)} = 25.06$, $p < .001$, Cohen's $d = 13.36$. This indicated that the participants were not making random responses and could identify which animation represented the real motion recordings, thus validating the stimuli.

Procedure

The dynamic stimuli were presented using an LCD display with a resolution of 1024 x 768 and a 60Hz refresh rate. Viewing distance was 60cm, at which the distance of the 30cm x 22.5cm display subtended an angle of 26.6º x 20.6º. The height of the average face was approximately 10.5º and the frame rate of the animation was 25fps. Participants engaged in 2 experimental conditions, each with 2 manipulations (upright versus orientation-inverted facial motion). Conditions comprised 21 experimental trials, plus 4 attention-control trials. Videos were edited so that only the first 5 seconds were shown to ensure equal viewing durations. The same experimenter always sat behind the participants to manually record their verbal responses. No feedback was given. The average duration of the experiment was approximately 25 minutes.

The first condition consisted of discriminating between different *videos* of facial motion. A single facial animation was displayed in the centre of a black screen. Immediately after, the same animation was presented again plus a completely different animation (shown side-by-side). The foil animation was chosen at random and could have been from the same actor reciting a different poem. Using a two-

alternative forced choice procedure, participants had to indicate which stimuli (left or right) were present in both trials. A similar format was used for the second condition, in which participants were required to discriminate between different *identities* of facial motion. A single facial animation was selected at random and its presentation was followed by another 2 animations. One was of the original actor reciting a different poem (target), and the other was of a completely different actor reciting any poem (foil). Using characteristic mannerisms and individuality of movements, participants had to discriminate which animation (left or right) represented the same individual from the first presentation. All observers viewed the same combination of videos across trials. Male and female performances were not intermixed within the same trial to avoid indirect judgments based on gender. Each task (video or identity discrimination) was carried out using upright and orientation-inverted stimuli. The order of conditions was counterbalanced across participants to avoid familiarity effects, boredom or fatigue influencing the data.

To ensure maximal attention to the stimuli throughout the testing period, attention-control trials were also included. This provided a conservative criterion for rejecting any data where there was a possibility of non-perceptual factors (fatigue, intermittent confusion) influencing the responses (Spencer & O'Brien, 2006). Attention-controls occurred on every eighth trial in all conditions, and were presented in a similar format as experimental trials. A single facial motion was displayed. Immediately after, the same video was shown again plus an orientation-inverted animation. Participants had to state which video (left or right) was present in both trials. As the orientation of one test stimuli was inverted, it could be excluded as a correct answer. Participants were aware which manipulations would indicate the correct answer. The responses to these trials were not included in any subsequent analysis. All participants completed these trials without error, therefore no data were discarded.

4.3 Results

A one-sampled t-test (test value = 10.5) was applied to the data to ensure all participants performed above chance level. A repeated measures ANOVA was then used to observe any main effects of *task type* (video, identity) and *orientation* (upright, orientation-inverted). Post hoc (paired samples t-test) tests were applied where appropriate. Table 7 presents the means (SD) from each condition. Participants significantly performed above chance level on all tasks: *upright video discrimination* ($t_{(19)} = 67.67$, $p < .001$, Cohen's $d = 31.05$), *orientation-inverted video discrimination* ($t_{(19)} = 13.24$, $p < .001$, Cohen's $d = 6.07$), *upright identity discrimination* ($t_{(19)} = 18.01$, $p < .001$, Cohen's $d = 8.26$), and *orientation-inverted identity discrimination* ($t_{(19)} = 5.07$, $p < .001$, Cohen's $d = 2.33$).

There was a significant main effect of *task type* ($F_{(1, 19)} = 48.01$, $p < .001$, $np^2 = .72$) with participants scoring higher on video discrimination tasks. Orientation also produced a significant main effect ($F_{(1, 19)} = 194.46$, $p < .001$, $np^2 = .91$). T-tests revealed a significant difference between the *upright* and

orientation-inverted video scores ($t_{(19)}$ = 7.71, p < .001, Cohen's d = 1.72) and between the *upright* and *orientation-inverted* identity scores ($t_{(19)}$ = 12.46, p < .001, Cohen's d = 2.79). Such results indicate an inversion effect present in this sample. There was also a significant interaction between *task type* and *orientation* ($F_{(1, 19)}$ = 32.51, p = .001, np^2 = .44). A larger inversion effect occurred for identity discrimination tasks compared to video discrimination tasks.

Table 7. *Mean correct scores (out of 21) for each task.*

Task	Orientation	Mean	SD	% score	Range
Video discrimination	Upright	20.65	0.67	98.33	19 - 21
	Inverted	17.05	2.21	81.19	14 - 21
Identity discrimination	Upright	19.25	2.17	91.67	15 - 21
	Inverted	13.10	2.29	62.38	9 - 17

4.4 Discussion

Building upon the pioneering work of Hill and Johnston (2001), recent developments in marker-less technology were exploited to generate models of facial movement. Simultaneous sequences of rigid and nonrigid motion (including eye-gaze and blinks) were recorded from real people and applied to a CGI display. These animations were used to evaluate motion-specific contributions in the categorisation of identity.

Recognition from Facial Motion

Consistent with the hypotheses, participants were able to distinguish between different facial motion videos and discriminate the faces of unfamiliar individuals. Other studies of face learning and recognition from dynamic stimuli have reported parallel findings (e.g., Hill & Johnston, 2001; Knight & Johnston, 1997; Pilz *et al.,* 2006). Similar results have also been documented in infant populations (Otsuka *et al.,* 2009). Spencer *et al.,* (2006) reported that infants between 4 and 8 months could discriminate sequences of facial motion and the identity of a speaker. Layton and Rochat (2007) observed an effect of motion cues at 8 months of age when infants viewed familiar faces (their mothers face). Bulf and Turati (2010) extended these findings, demonstrating that newborns were able to recognise the profile pose of unfamiliar faces moving rigidly. Evidently, the ability to perceive and utilise facial motion is acquired very early on in visual development. This in turn is thought to facilitate more efficient face recognition abilities as infants learn to identify characteristic patterns of movement (Xiao *et al.,* 2014).

There are two prominent hypotheses regarding how facial motion influences identification processes (O'Toole *et al.,* 2002). First, the *'supplemental information hypothesis'* states that idiosyncratic facial movements aids identification. This cue may be particularly useful when recognising already familiar faces. For example, you might identify a close friend by the way they smile, or characteristically nod

their head during conversations. By contrast, the '*representation enhancement hypothesis'* suggests that facial motion provides a better structural depiction of a 3D face. Learning new faces benefits from such enhancement. The number of view-points increases with learning, therefore refining mental representations of less familiar faces (O'Toole *et al.,* 2002).

While these hypotheses describe two different ways in which facial motion contributes to identity judgments, it does not mean that they are strictly exclusive for a specific type of recognition. Rather, they are interlinked. There is some neuroimaging evidence to support this conclusion. Encoding new views of an individual has been thought to operate within the fusiform gyrus (Longmore *et al.,* 2008), while identifying characteristic motion takes place within a portion of the superior temporal sulcus (Longmore & Tree, 2013). Past studies have shown both regions to be collectively active during facial motion perception (Furl *et al.,* 2010; Schultz & Pilz, 2009). It is unclear though whether ventral-temporal areas are showing a true dynamic response, or are simply sensitive to the static information contained within a motion sequence (Schultz *et al.,* 2013).

As with static face perception, inverting the stimuli significantly reduced participants' ability to discriminate video sequences or recognise the faces of different individuals. This was particularly true for judgments concerning facial identity, perhaps reflecting task complexity. To successfully discriminate different identities, participants had to perceive characteristic mannerisms, which would have been more difficult to do when the animations were inverted. It seems that the inversion effect is sensitive to task type and what information needs to be extracted. Many pre-existing studies report similar inversion effects with dynamic stimuli (Longmore & Tree, 2013; Watson *et al.,* 2005). Observers were poor on tasks requiring them to judge the gender of an inverted dynamic face (Thornton *et al.,* 2011). This suggests that motion information is processed configurally by a system tuned to upright faces, rather than by extraction of low-level cues (Hill & Johnston, 2001; Watson *et al.,* 2005).

In contrast, facial motion might utilise part-based processing and bypass the disruption caused by inversion. Indeed, a less pronounced inversion effect has been observed when faces are shown dynamically (Hill & Johnston, 2001; Knappmeyer *et al.,* 2003). More recent investigations using composite faces also support the featural influence hypothesis of facial motion perception. Xiao *et al.,* (2012) found that the upper and lower portions of composite faces were processed in a part-based manner, allowing participants to identify the test faces more accurately. Xiao *et al.,* (2013) later replicated and extended these findings, reporting a smaller composite effect for elastic (nonrigid) facial motion. The current data is evidently mixed and further clarification is needed. It may be that dynamic faces are still subjected to the inversion effect, but the addition of motion helps to minimise the disruption.

Comparison of Methodology with Other Approaches

A handful of face perception studies have implemented dynamic stimuli inspired by the Facial Action Coding System (FACS; Ekman & Friesen, 1978). FACS quantifies all possible facial muscle expressions and decomposes them into action units. Each unit is then plotted as a time course so that the spatiotemporal properties of local movements can be represented. This technique has been applied to motion-capture data to create highly controlled and meaningful facial animations (e.g., Curio *et al.,* 2006; Dobs *et al.,* 2014). The advantage here is that facial motion is annotated accurately and precisely with reference to underlying muscle activations. It is also easy to retarget motion onto any face model that uses the same semantic structure (Curio *et al.,* 2006). Yet, these FACS derived animations typically present only nonrigid motion - that is, facial expressions without changes in viewpoint. Head position and orientation, however, represent a powerful cue, especially with reference to identity recognition (Hill & Johnston, 2001). The stimuli presented here may therefore be more suitable than previous techniques when studying face perception.

Further, marker-based motion capture records data from a pre-defined set of facial points. Because of this, subtle or extremely implicit facial movements occurring in other 'un-marked' areas are disregarded. The method described here minimises such issues. Motion in all face regions was recorded resulting in extremely detailed and naturally fluid animations. As the stimuli closely resembled real human facial movement in the absence of spatial cues, it is possible to generalise the current data to faces in real life. Indeed, there is evidence that natural and synthetic faces are processed by similar neural mechanisms. Moser *et al.,* (2007) demonstrated that avatars elicit similar patterns of activation to human faces, particularly in the emotion-sensitive amygdala. It is important to note, however, that not all studies report comparable findings. Han *et al.,* (2005) found that although the neural response to real and virtual displays of human movement were similar at posterior brain regions, it did differ in the medial prefrontal cortex. The authors suggest that this could reflect the processing of low-level visual features versus high-level mental state attribution. Yet as Dobs *et al.,* (2014) points out, discrepant results are most likely caused by differences in how natural the synthetic face is, further highlighting the need for authentic stimuli.

As a side note, viewing such motion-rich stimuli could explain why some observers performed at ceiling in all but one condition (orientation-inverted identity discrimination). Stimuli high in detail would provide much information concerning identity, which in turn would facilitate perception. On the other hand, this could reflect aspects of the task design. Stimuli were presented consecutively without delays and participants were asked to make their decision immediately after each trial. This decrease in working memory could have evoked superior levels in face recognition (Weigelt *et al.,* 2013). Other experiments should perhaps allow a delay before recall in order to assess whether similar ceiling effects occur.

Limitations and Future Directions

While the current data indicates a significant ability in categorising identity from facial motion, it is possible that observers could do this just as easily with multiple static frames or snapshots of different head positions (Lander & Chuang, 2005). However, other researchers have shown it is the dynamic quality of motion, rather than the amount of static information, that is crucial for recognition (Lander & Bruce, 2000). For example, Lander *et al.,* (1999) have reported that identity recognition was better with moving sequences relative to a static array even though both stimuli contained the same amount of frames.

Moreover, several papers have attempted to discover which aspect of facial motion contributes to recognition. Unfortunately they provide mixed results. Hill and Johnston (2001) suggest it is head rotations and translations which are useful when categorising identity. However, at least three research groups have shown no advantage for rigid motion compared to static images (e.g., Christie & Bruce, 1998; Lander & Bruce, 2005; Lee *et al.,* 2010). As rigid and nonrigid motion cues were not separated within the current study, it cannot be said what aspect is driving the performance here. Of course, it may be that perception is facilitated by a combination of both cues. In real life, changeable facial expressions and head movements are encountered simultaneously rather than in isolation. Supporting this assumption, Maguinness and Newell (2014) reported that motion facilitates face learning across changes in both viewpoint (rigid) and expression (nonrigid).

Future studies are encouraged to extend this experiment by implementing conditions which compare performances based on rigid motion, nonrigid motion and combined motion cues. This would allow clear conclusions to be drawn regarding which type of facial motion is facilitating its perception. In addition, the stimuli method could be applied to the study of emotion processing. It has been previously shown that dynamic presentations aids the recognition of emotional expressions, but these conclusions are derived from implied motion or morphed videos (e.g., Bould & Morris, 2008; Puce *et al.,* 1998). Implementing such facial motion captures would significantly help in fully understanding the underlying mechanisms.

4.5 Conclusion

This chapter describes a new method to create facial motion stimuli that is free from surfaced-based visual cues but still accurate and authentic to real life interaction. While similar to those used by Hill and Johnston (2001), the current marker-less animations contain much more detail and move more naturally. From the use of such advanced stimuli, the data demonstrates that adult observers are able to perceive facial motion and can use it to make sensible categorisations concerning unfamiliar facial identities. This ability is very much central to appropriate social interaction. By recognising whether a

person is a friend or stranger, we can adjust our behaviour to suit the situation and therefore ensure both emotional and physical well-being. Further, the current data also supports the configural view of human face perception whereby observers are sensitive to inversion effects. This indicates that the visual system is very much attuned to biologically natural and familiar displays of facial motion.

CHAPTER 5 Neural Correlates of Facial Motion Perception in Neurotypical Controls

5.1 Introduction

Facial motion provides an essential source of social information. It conveys cues necessary for lip reading (Campbell, 1992) and judging the emotional state or intention of others (Bassili, 1979; Matsuzaki & Sato, 2008; Pollick *et al.,* 2003). In addition, it facilitates the recognition of facial identity (Knappmeyer *et al.,* 2003; Lander & Bruce, 2000), gender (Berry, 1991; Hill & Johnston, 2001; Thornton *et al.,* 2011) and age (Berry, 1990b). Human interaction is dependent on the ability to correctly perceive and deduce these dynamic cues (Blake & Shiffar, 2007; also see Chapter 1 –1.7).

The human visual system has developed highly specialised mechanisms which facilitate the detection and interpretation of facial motion (Atkinson *et al.,* 2011). Puce *et al.,* (1998) first described functional activity in the posterior superior temporal sulcus (pSTS) to perceived eye and mouth movements. Similarly, Lloyd-Fox *et al.,* (2009) found a greater haemodynamic response in the STS when 5-month-old infants viewed video clips of female actors moving specific face parts. Natural video sequences of facial motion also significantly activate the pSTS even when stimulus motion is controlled for (Schultz & Pilz, 2009). More recent studies report parallel results. Polosecki *et al.,* (2013) found only the STS region to show specific sensitivity to videos of actors vocalising and generating expressions. Increasing the frame rate and correct frame order of facial motion also evokes a greater BOLD response in the STS, indicating its involvement in assessing the fluidity and meaning of facial movements (Schultz *et al.,* 2013). Chewing gestures or fearful expressions further evokes a consistent lateralisation in the right pSTS relative to phase-scrambled stimuli (De Winter *et al.,* 2015). These findings suggest that the processing of variant and changeable facial aspects occurs primarily within the STS region.

Despite being informative, the current data only describes the neural response to dynamic faces exhibiting emotional expressions or everyday gestures. Yet facial motion also conveys information about the identity of conspecifics by providing 3D structural cues and prompting the recognition of idiosyncrasies during sub-optimal viewing conditions (O'Toole *et al.,* 2002; also see Chapter 4). It is important to examine which brain structures underlie this perception, especially if one considers its pertinent role in survival (Pavlova, 2012). As it stands, very few fMRI investigations have done this. Harris *et al.,* (2014) observed significant engagement of the pSTS, occipital face area (OFA) and fusiform face area (FFA) during the identity recognition of emotion-morphed videos. The latter two ventral temporal substrates are commonly implicated in the perception of identity from static faces (Slotnick & White, 2013). Hierarchical feedforward models posit that the OFA is an early module within the network, performing simple face detection tasks (Pitcher *et al.,* 2011). Information is then transmitted to the FFA or STS so that individual features can be encoded for successful recognition

(Kadosh *et al.,* 2011; Rhodes *et al.,* 2009). Such bottom-up processing may therefore underlie the perception of identity from facial motion.

While this data supports the role of the STS in identity detection, other researchers fail to report similar findings. In one study, the STS region was more active to changes in eye-gaze and emotional expression when the image sequences were of the same person relative to multiple identities (Baseler *et al.,* 2014). This effect appeared to be primarily driven by an increased functional connectivity to the FFA, suggesting that it is this region which processes specific facial aspects relating to identity. It is important to note, however, that the studies by Harris *et al.,* (2014) and Baseler *et al.,* (2014) did not utilise real dynamic face stimuli. This means that any neural activity evoked in the FFA or OFA could simply be reflecting static information processing rather than a true dynamic response (Schultz *et al.,* 2013). More research is needed here to gain clearer results.

Similarly, many of the abovementioned studies have implemented abstract or unnatural depictions of facial movement (implied motion images, morphed videos from static images or cartoon avatars). Yet these representations may not fully engage the underlying mechanisms (Schultz & Pilz, 2009). To address this issue, the present study implemented realistic facial animations derived from humans reciting poems. These exhibited simultaneous sequences of rigid (head rotations and translations) and nonrigid motion, as well as speech expressions and eye-gaze shifts. Neurotypical observers were asked to discriminate between different videos and to recognise unfamiliar facial identities. As the appearances of animations were identical to each other, judgments were based solely on differences in motion patterns. Facial motion captures were additionally presented upside down to form a control or baseline condition. This experimental paradigm appears to affect face recognition by disrupting configural processing and early structural encoding (Girges *et al.,* 2014; Girges *et al.,* 2015).

It was also necessary to include other stimulus categories so that the neural response to facial motion could be quantitatively compared to that evoked by various other biological and inanimate stimuli presented either dynamically or statically. Acquiring such data would allow unequivocal conclusions to be drawn with regards to which neural substrates are specialised for dynamic face processing. Static bodies, faces, objects and place images were presented in order to localise activity of the extrastriate body area, FFA, lateral occipital complex and parahippocampal place area respectively. An MT+/V5 localiser task was also administered via presentation of coherent and random dot kinematograms. Lastly, point-light walkers were used to examine whether similar mechanisms underlie all types of biological motion processing. The remainder of this review will briefly outline each localiser region[2].

[2] The purpose of conducting localiser tasks was not to investigate the functionality of each region *per se*, but to provide a conservative criterion in which to compare facial motion processing. To this end, the reader is directed to other papers for more detailed accounts (e.g., Iidaka, 2014; Taylor & Downing, 2011).

Fusiform Face Area (FFA)

The occipitotemporal extrastriate cortex represents static facial information. Positron Emission Tomography (PET) studies identified a region within the right fusiform gyrus (FG) which appeared significantly more active to face stimuli relative to objects (Haxby *et al.,* 1994; Sergent *et al.,* 1992). MRI investigations which compared the perception of faces with objects or phase-scrambled stimuli also observe strong engagement of the FG, specifically in a portion now referred to as the FFA (Kanwisher *et al.,* 1997; Puce *et al.,* 1995; Rangarajan *et al.,* 2014). This activity depends on a number of factors though, including attention levels, emotional valence and the familiarity of facial stimuli to the observer (Pierce *et al.,* 2004; Vuilleumier *et al.,* 2001; Wojciulik *et al.,* 1998).

Parahippocampal Place Area (PPA)

The visual processing of spatial layouts or place images occurs within a sub region of the posterior parahippocampal cortex known as the PPA. This neural substrate is highly sensitive to scenes relative to face or object images (Epstein *et al.,* 1999; Epstein *et al.,* 2003). Damage to the PPA causes an impaired visual identification of well-known landmarks and difficulty in identifying one's own location (Aguirre & D'Espositio, 1999; Habib & Sirigu, 1987), suggesting it represents local scene geometry and spatial expanse (Epstein *et al.,* 2003; Park *et al.,* 2011). The PPA is also connected to visual areas, particularly the retrosplenial cortex (BA 30) located in the transverse occipital sulcus (Dilks *et al.,* 2013; Rushworth *et al.,* 2006). Formally known as the occipital place area, research has found this region to preferentially respond to real world scenes (Bar & Aminoff, 2003; Epstein & Higgins, 2006), as well as rooms and city streets (Henderson *et al.,* 2011).

Lateral Occipital Complex (LOC)

Patients with bilateral damage to the LOC suffer profound object agnosia while still retaining the ability to recognise scenes (Steeves *et al.,* 2004). Similar findings have been found using repetitive TMS. For example, temporarily disrupting the functionality of the LOC significantly impairs the categorisation of objects in healthy adults (Mullin & Steeves, 2011). This finding has led to the hypothesis that such a discrete cortical area is selectively responsible for inanimate object recognition (Malach *et al.,* 1995; Pitcher *et al.,* 2009).

Extrastriate Body Area (EBA)

The perception of body form is primarily processed within a region of the posterior inferior temporal sulcus commonly referred to as the EBA (Grosbras *et al.,* 2012). Downing *et al.,* (2001) first observed that this neuronal population was selectively responsive to images of human bodies and body parts relative to objects or scenes. This was later extended to photorealistic depictions of bodies, line drawings and silhouettes (e.g., Peelen & Downing, 2005a). Other researchers note its involvement in mapping the morphological features of bodies and in the visual analysis of body appearance (Peelen &

Downing, 2007; Urgesi *et al.,* 2007). The response pattern in the EBA also appears to carry information concerning the body posture of point-light walkers but not of its motion (Vangeneugden *et al.,* 2014). This suggests that information about body configuration and kinematics are processed by separate neural mechanisms (Jastorff *et al.,* 2012).

Posterior STS for Body Motion Perception

The right pSTS has been highly implicated in body motion perception (Allison *et al.,* 2001; Vangenugden *et al.,* 2014). Pelphrey *et al.,* (2003) found such regions to respond more strongly to human and robot walking figures compared to movements made by a clock or mechanical object. Vander Wyk *et al.,* (2009) note that the pSTS is engaged in representing the intention behind specific actions as well as being involved in basic perception. Herrington *et al.,* (2011) postulate that this area functions by matching perceptual inputs against familiar internal depictions of human movement. The visuo-cognitive analysis of facial and body motion thus appears to occur within the pSTS.

MT+/V5

Neuronal populations within area MT+/V5 of the extrastriate visual cortex are preferentially activated by motion cues (Dumoulin *et al.,* 2000). Increasing the global coherence of random dot kinematograms correlates with higher observable BOLD signals in MT+/V5 (Braddick *et al.,* 2001). This relationship, however, appears to be lost when unilateral damage occurs to V1, suggesting that primary visual areas project information to MT+/V5 in a feedforward manner (Ajina *et al.,* 2015).

In conclusion, the purpose of the current study was threefold: (1) identify which neural substrate(s) process facial motion when representations are realistic but contain no confounding spatial cues; (2) investigate the neural correlates of perceiving identity from facial motion when interferences from variant form properties are limited; and (3) compare the mechanisms underlying facial motion perception with that of other biological and inanimate stimuli (presented statically and dynamically). Such experimental paradigm will hopefully facilitate our understanding of how facial motion is processed by the brain and whether this is different to that of other dynamic entities.

5.2 Methods and Materials

Participants

Ethical approval was acquired from Brunel University. Eight neurotypical adults (2 males, 6 females, age: *M* = 24.75, *SD* = 3.92, *Range* = 21-32) with normal or corrected-to-normal vision participated in the current study. Five participants were right handed, and three were left handed (or ambidextrous). None of the sample reported any previous history of neurological or psychological disorders. Informed consent was obtained and participants were debriefed at the end of the study.

Stimuli

FFA, PPA AND LOC LOCALISER

Greyscale photographs (~300 x 300 pixels) of unfamiliar faces (male and female), assorted objects and landscape scenes were supplied by the Kanwisher Lab (http://web.mit.edu/bcs/nklab/). The object photographs (or photo like images) were in a canonical viewpoint and were familiar to participants (e.g., spoon, camera and French horn). Scrambled photographs were included as a control (Figure 6).

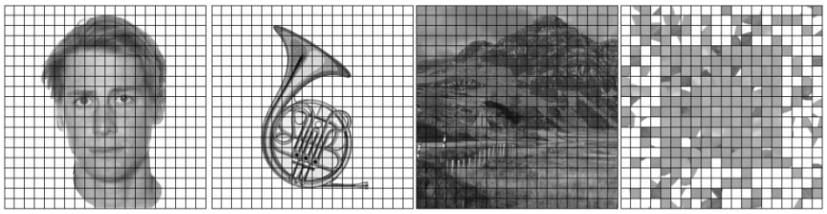

Figure 6. From left to right: Unfamiliar faces, objects, landscapes and scrambled images.

EBA LOCALISER

Greyscale photographs (~400 x 400 pixels) of headless bodies and chairs were taken from an online database (http://pages.bangor.ac.uk/~pss811/page7/page7.html) created by Downing *et al.,* (2001). Body images were male and female, and presented in a range of poses and clothing types (Figure 7).

Figure 7. Examples of the body and chair images.

BODY MOTION LOCALISER

Human and cat point-light walkers were taken from an online database (http://www.biomotionlab.ca/). The human sequences represented the average walker computed from the motion data of 50 women and 50 men. Eleven markers depicted movement tracked from major limb joints (head, shoulders, hips, elbows, knees). The gait frequency of the walker was 0.86Hz. The cat motion figures were derived from a high speed video sequence of a cat walking on a treadmill. Fourteen feature points were manually sampled from single frames and approximated with a third-order Fourier series (smoothed and looped) to produce a general walking cycle (gait frequency = 1.5Hz). All point-light figures presented stationary walking and were shown in a saggital view. Figures faced the left or right

direction. Scrambled stimuli were achieved by placing each dot trajectory at a random position within the centre of the display area (Figure 8).

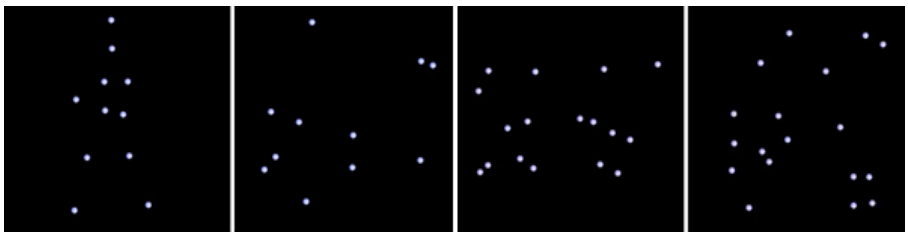

Figure 8. From left to right: intact human point-light walker (PLW), scrambled human PLW, intact cat PLW and scrambled cat PLW.

MT+/V5 LOCALISER

Stimuli were modelled on the description given by Smith *et al.,* (2006). High-contrast moving dot patterns were used. Dots moved in a straight path at a speed of 15 deg/s for a lifetime of 133ms (10 frames) before disappearing and reappearing at a new random location. Dots leaving the edge of the stimulus disappeared for the remainder of their lifetime before being replotted. Different dots were repositioned at different times, with 10% of the dots being repositioned on each frame update. A 70-deg circular image was filled with white dots that were made to move inwards or outwards along the radii of the image to produce an impression of expansion (Figure 9). The baseline condition was a circular image filled with dots that moved in a random direction with no global motion structure.

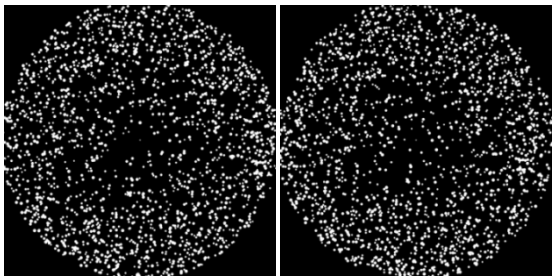

Figure 9. Example of the motion kinematogram.

FACIAL MOTION

The dynamic face stimuli implemented in the current study were identical to those described in Chapter 4. Refer to Section 4.2 (Stimuli Creation) for full details.

Functional MRI Tasks and Design

Conditions were configured in MATLAB (MATLAB 7.10.0. Natick, Massachusetts: The MathWorks Inc., 2010) and presented using an LCD display with a resolution of 1024 x 768 and a 60Hz refresh rate. All tasks shared the same design in that participants viewed 2 sequences of static images or dynamic

videos and judged whether they were identical to each other (2 interval forced-choice procedure; Appendix 1). Responses were made via a button press (left = yes, right = no). Sequences were separated by a 1 second interstimulus interval (ISI) and response timeout was set to 5 seconds.

For the EBA localiser, blocks began with 5 consecutive images of bodies or chairs. After a 1-second ISI, another 5 consecutive images from the same stimulus category were shown. These were either identical or different to the first sequence. There were 20 blocks in total, and each sequence was presented for 6 seconds. The same experimental design was used for the FFA, PPA and LOC localiser, in which participants viewed 40 blocks containing face, place, object or scrambled image sequences. Stimuli duration was 6 seconds and the task took 14 minutes to complete. Similarly, the body motion task contained 40 blocks of intact and scrambled human or cat point-light walkers. Each block contained 2 successive point-light displays (from a single category), in which the animation faced the right or left direction. Stimulus duration was 7 seconds and the task took 15 minutes to complete. Further, the MT+/V5 localiser included 20 interleaved blocks of coherent or random dot motion. Each block contained 2 successive videos (duration = 7 seconds) separated by a 1-second ISI.

The facial motion tasks comprised participants viewing upright or inverted videos. Within each block, 2 videos were shown successively and the task was to decide whether they were the same or different from each other (video discrimination). This procedure carried on for 40 blocks. Participants also completed an identity discrimination task. All the parameters were identical to the previous task, but the participants had to discriminate different identities from facial motion. To do this, characteristic movement patterns had to be correctly perceived in order to state whether the 2 videos were depicting movement from the same person. Stimuli were presented for 8 seconds and the task took 16 minutes to complete.

Image Acquisition and Analysis

Images were acquired on a 3.0 Tesla Siemens Magnetom Trio scanner with a 32 channel array head coil. Functional MRI was performed using a standard gradient echoplanar imaging (EPI) sequence (TR 3000ms, TE 30ms, flip angle 90 degrees, 3mm slice thickness, 64 x 64 matrix, 160 contiguous axial slices, bandwidth 1396 Hz/pixel). Functional scanning runs comprised up to 320 volumes lasting 16 minutes. Anatomical T1-weighted MPRAGE 3D MRI sequence images were also acquired during the scanning session (TR 1830ms, TE 4.43ms, flip angle 11^{O}, 160 axial slices, 1mm isotropic voxels, 256 x 256 matrix, bandwidth = 130 Hz/pixel). Visual stimuli were projected onto a screen (via a Sanyo LCD projector, PLC-XP1000L, native resolution = 1024 x 768) in the bore of the magnet and viewed through an angled mirror above the head coil. All stimuli were shown within a window measuring 420 x 420 pixels on the screen. At the effective total viewing distance (from projection screen to the coil mirror to the participants' eyes) of approximately 82cm, this corresponded to 12.0^{o} x 12.0^{o}.

The data were pre-processed and analysed by using Statistical Parametric Mapping (SPM8, Wellcome Institute of Cognitive Neuroscience, London, UK) in Matlab. For individual data sets, the images were corrected for head movement by realigning each EPI volume to match the volume in the first scan. The resulting images were then normalised to sterotaxic Montreal Neurological Institute (MNI) coordinates using trilinear interpolation. As a final pre-processing step, the normalised images were smoothed using a Gaussian filter with a full-width at half maximum parameter set to 8mm.

The analysis was based on a regular whole-brain SPM approach. Statistics were performed separately at each voxel and modelled using a box-car function convolved with a canonical hemodynamic response function. Contrasts were defined to compare the neural difference between two conditions by subtracting one beta from another (outlined in Table 8). Group averages of these first-level analyses are reported here. Plots of contrast estimates (90% confidence interval) were also produced by taking the mean value from all individual datasets. The purpose of this was to observe differences in the activity: (1) evoked by facial motion or specific localiser stimuli in regions of interest (ROI) defined by the previous results; and (2) evoked by facial motion or static faces, objects, places, point-light walkers, coherent motion and static bodies in the STS. Lastly, for the facial motion video discrimination task, a random effect analysis (RFX) was conducted across the group based on individual statistical parameter maps to allow for population inference. ROI were limited to the occipital and temporal lobes (conducted via the WFU PickAtlas toolbox in SPM8).

Table 8. *Contrasts performed for each experimental task.*

Discrimination task	Contrast
Facial motion - Video	Upright >* Inverted
Facial motion - Identity	Upright > Inverted
Static faces	Faces > Scrambled images
Static places	Places > Scrambled images
Static objects	Objects > Scrambled images
	Objects > Place images
Static bodies	Bodies > Chair images
Coherent motion	Coherent > Random motion
Body Motion	Intact human point-light walker > All other stimuli
	Intact human point-light walker > Baseline

**Greater than sign (">") refers to a t-contrast where one beta has been subtracted from another.*

5.3 Results

Group Average Data (First-level)

A summary of the main neural activations for each experimental task at the group level is presented in Figure 10. This demonstrates differences in the BOLD response of specific substrates during the perception of biological and inanimate stimuli (dynamic and static).

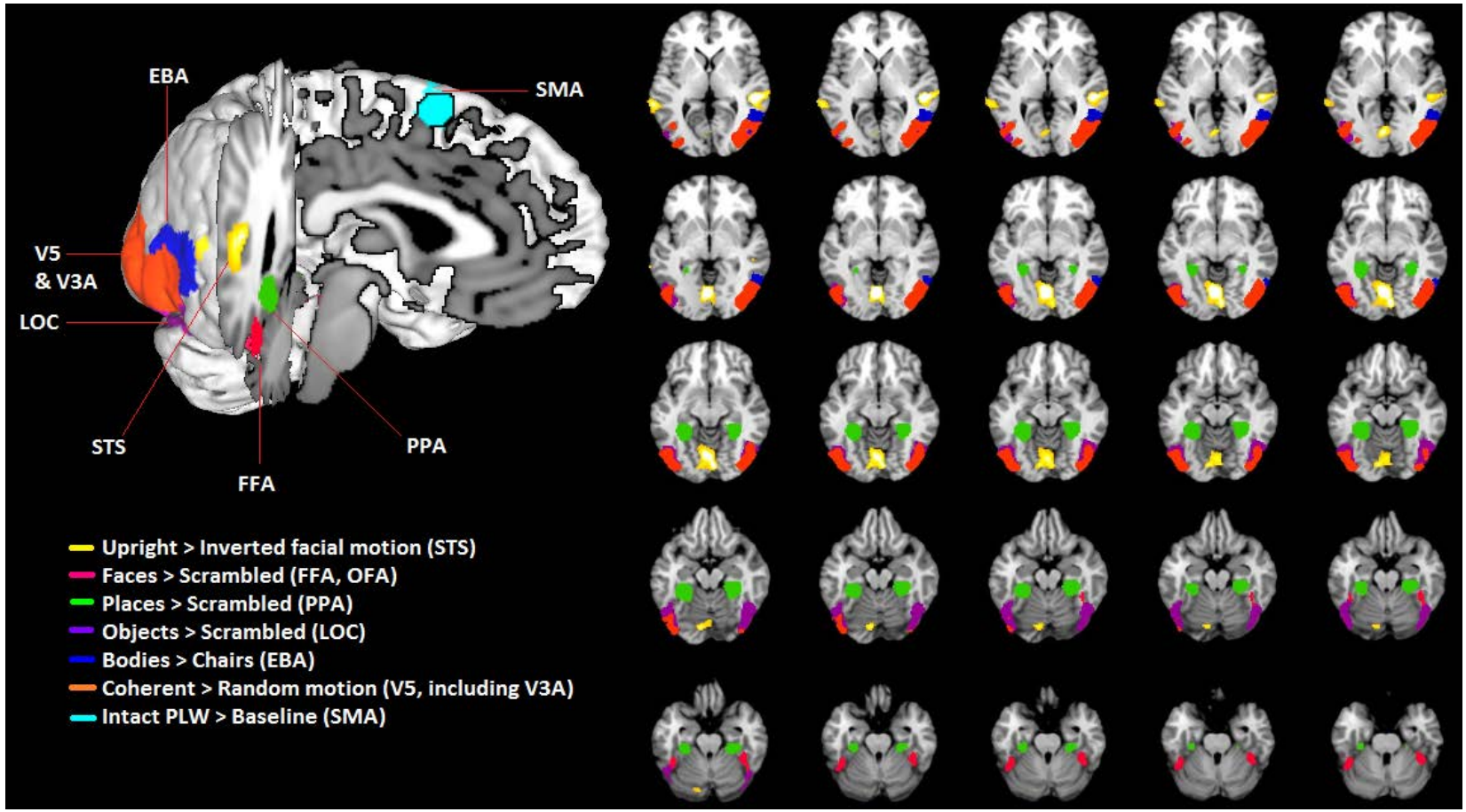

Figure 10. Group activity for each task. Superior temporal sulcus (STS) activated by the contrast of upright > inverted Facial Motion; Extrastriate body area (EBA) - Bodies > Chairs; Fusiform face area (FFA) - Faces > Scrambled; Lateral occipital complex (LOC) - Objects > Places; Parahippocampal place area (PPA) - Places > Scrambled; V5 complex including V3A - Coherent > Random Motion ($p < .001$, uncorrected); Supplementary motor area (SMA) - Intact human PLW > Baseline. All results are $p < .05$ FWE corrected unless stated otherwise.

FACIAL MOTION

When discriminating between different *videos* of facial motion, the upright > inverted contrast revealed significant neural activations in the bilateral posterior STS, extending into the middle temporal cortex. Activity was also observed in the bilateral lingual gyrus, right FG, left cerebellum, right inferior frontal gyrus (IFG), and a region of the right precentral gyrus known to contain the dorsal premotor cortex (dPMC). Similarly, for discriminations concerning *identity* from facial motion, the upright > inverted contrast revealed a significant cluster of activity in the middle temporal region, extending into the STS complex. However, while bilateral, the response was greater on the right hemisphere. The precentral gyrus (dPMC), right IFG (pars triangularis) and right inferior and middle frontal gyri also show some response here (Table 9; Figure 11).

Table 9. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size* are shown (k = 10 voxels, height threshold = p < .05, FWE corrected).*

Anatomy	BA	Coordinates			T-value	Size (mm^3)
		x	*y*	*z*		
Facial Motion Video: Upright > Inverted						
R. Superior temporal	22	52	-36	8	7.61	858
R. Superior temporal	48	68	-36	22	5.72	
R. Superior temporal	42	60	-36	20	5.57	
L. Lingual gyrus	17	-2	-72	-4	7.25	899
R. Lingual gyrus	18	6	-86	-6	6.49	
L. Cerebellum	18	-8	-82	-14	6.08	
L. Middle temporal	37	-62	-56	10	6.36	402
L. Middle temporal	21	-62	-48	8	6.18	
R. Fusiform gyrus	37	42	-44	-24	5.65	38
R. Inferior frontal (pars triangularis)	45	50	38	8	5.57	63
L. Cerebellum	37	-42	-54	-24	5.53	50
L. Cerebellum	37	-40	-44	-26	5.07	
R. Precentral gyrus (dPMC)	6	56	4	42	5.20	57
Facial Motion Identity: Upright > Inverted						
R. Inferior frontal (pars triangularis)	45	60	26	20	6.18	117
R. Middle temporal	22	62	-34	6	6.14	189
R. Middle temporal	21	50	-38	8	5.26	
R. Precentral gyrus	6	58	6	38	5.54	56
R. Middle frontal gyrus	6	48	0	56	5.36	39
L. Middle temporal	21	-58	-48	6	5.20	28
L. Precentral gyrus	6	-48	4	50	5.13	32

** Missing values in the size column indicate an activation peak that is part of the cluster listed above.*

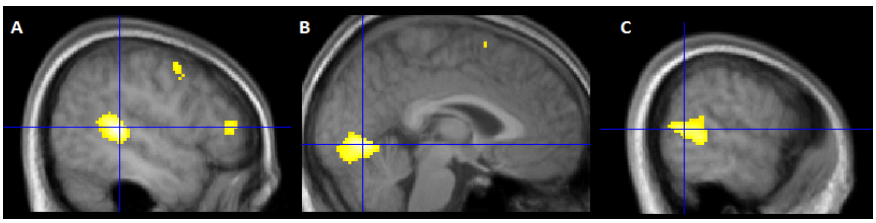

Figure 11. Saggital views of activity in the (A) right pSTS, (B) right lingual gyrus and (C) left middle temporal cortex (extending into the STS) for the contrast of *upright > inverted* facial motion video discrimination. The image on which activity is overlaid is the mean of the structural images from all participants. All results are reported at the *p* < .05 FWE corrected threshold level.

STATIC STIMULI

FACES, PLACES AND OBJECTS. As expected, the *face* > *scrambled images* contrast showed active voxels in the bilateral inferior occipital gyri (corresponding to the OFA) and the lateral portions of the fusiform gyrus (FFA). Other responsive regions included the middle temporal and middle occipital gyri. The *place* > *scrambled images* contrast revealed activation in the middle portion of the fusiform gyrus (PPA) bilaterally. Regions within and around the primary visual and middle occipital cortex were also significantly more active to images of place scenes. To examine neural correlates of object recognition, two contrasts were performed. The first (*objects* > *scrambled images*) revealed a number of responsive substrates, yet nothing that strongly corresponded to the LOC (Table 10). However, the *objects* > *place images* contrast showed activity in the bilateral inferior occipital region, which will now be referred to as the LOC. See Figures 12 and 13.

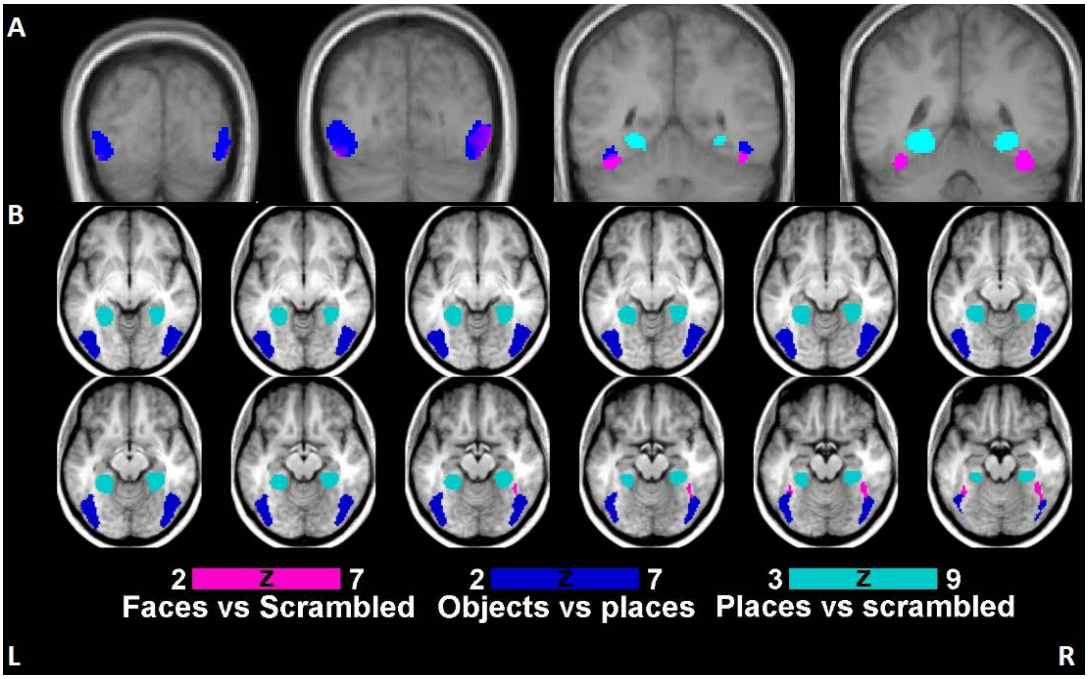

Figure 12. Activity evoked by faces > scrambled images (FFA), objects > scrambled images (LOC) and places > scrambled images (PPA). Coronal (Panel A) and axial (Panel B) slices are presented. The image on which activity is overlaid is the mean of the structural images from all participants. All results are reported at the $p < .05$ FWE corrected threshold level.

BODIES. Relative to chair stimuli, significantly greater activity to body images was found in the right middle temporal region, overlapping into the inferior temporal sulcus (Figure 13). Such area corresponds to the EBA (Vangeneugden *et al.,* 2014). A significant cluster was also observed in the left middle occipital cortex (Table 10).

Table 10. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size* are shown (k = 10 voxels, height threshold = p < .05, FWE corrected).*

Anatomy	BA	Coordinates			T-value	Size (mm^3)
		x	*y*	*z*		
Faces > Scrambled Images						
R. Inferior occipital	19	48	-76	-10	9.18	692
R. Fusiform	37	40	-46	-22	8.89	
R. Fusiform	37	46	-62	-16	7.42	
L. Fusiform	37	-40	-52	-22	7.96	342
L. Inferior occipital	19	-46	-76	-14	6.79	
L. Rectus	11	-8	48	-14	5.98	62
R. Middle temporal		62	-60	14	5.67	26
L. Middle occipital	19	-36	-80	40	5.55	34
L. Inferior orbitofrontal	47	-44	34	-10	5.44	115
L. Inferior orbitofrontal		-42	36	-18	5.21	
L. Middle occipital	39	-38	-66	30	5.32	75
L. Middle temporal	39	-48	-66	24	5.21	
Places > Scrambled Images						
L. Fusiform	37	-28	-42	-12	12.01	742
R. Fusiform	37	32	-42	-10	10.99	694
L. Middle occipital	19	-38	-82	28	9.37	630
L. Calcarine	17	-18	-58	16	8.68	627
R. Precuneus	17	16	-54	16	8.41	545
R. Middle occipital	39	46	-78	26	7.70	314
Objects > Scrambled Images						
L. Fusiform	37	-30	-40	-12	6.68	309
L. Fusiform	37	-28	-34	-22	5.79	
L. Fusiform	37	-40	-52	-20	5.56	
L. Middle occipital	19	-38	-68	32	6.39	518
L. Middle occipital	19	-38	-84	30	6.28	
L. Middle occipital	19	-36	-80	38	5.85	
L. Middle temporal	21	-64	-44	2	6.34	157
L. Middle cingulum		-4	-34	46	6.13	294
R. Middle cingulum	23	2	-42	34	5.22	
L. Precuneus	30	-4	-52	12	6.02	312
R. Calcarine	30	8	-54	12	5.24	
R. Fusiform	37	32	-40	-12	5.88	66
L. Middle occipital	19	-46	-80	0	5.74	152
L. Inferior occipital	19	-46	-76	-10	5.54	
L. Medial superior frontal	10	-14	54	0	5.73	231
L. Anterior cingulum	10	0	54	4	5.55	
L. Middle orbitofrontal	10	-6	50	-8	5.10	
R. Inferior occipital	19	46	-74	-8	5.28	37
Objects > Place Images						
R. Inferior occipital	19	44	-80	-12	9.86	1158
L. Inferior occipital	19	-42	-82	-10	9.37	1212
L. Inferior occipital	19	-44	-74	-12	9.16	
Bodies > Chairs						
R. Middle temporal	37	52	-69	6	9.09	961
L. Middle occipital	19	-48	-78	4	6.47	102

**Missing values in the size column indicate an activation peak that is part of the cluster listed immediately above.*

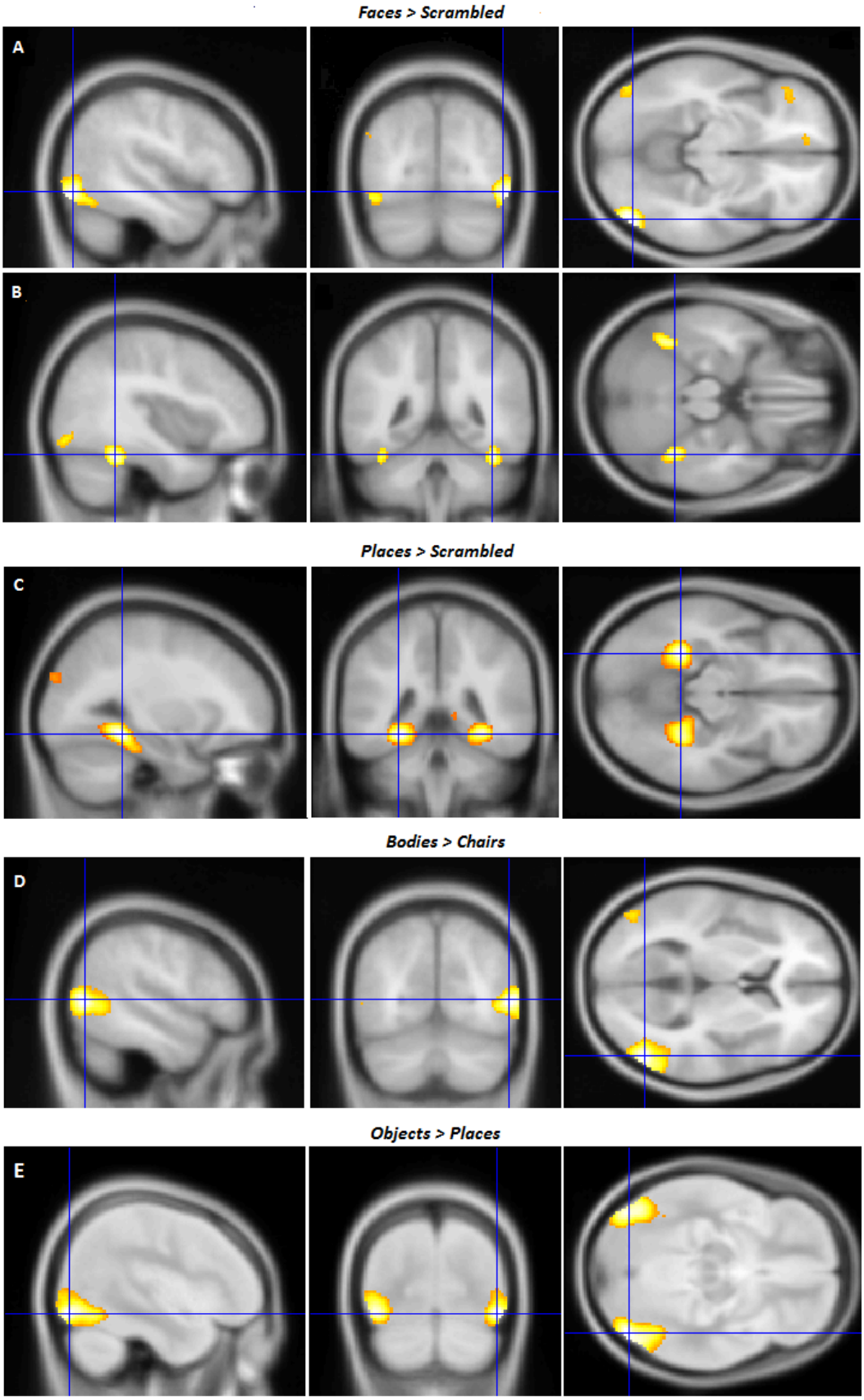

Figure 13. Location of the (A) OFA, (B) FFA, (C) PPA, (D) EBA and (E) LOC in standard MNI space. Saggital, coronal and axial views are presented. All results are reported at the $p < .05$ FWE corrected threshold level.

BODY MOTION

Compared to non-human motion (cat and scrambled stimuli), the perception of human point-light walkers (PLW) activated a single region of the right middle temporal cortex (MNI coordinates 54, -66, 4; t = 5.19, p < .05 FWE corrected, extent = 33 mm^3) corresponding to the EBA (Figure 14). As cluster size was small, activity was also considered at the p < .001 uncorrected threshold. The contrast revealed significant active clusters in the EBA and middle occipital cortex (Table 11). These findings are unanticipated as activity was expected to emerge in portions of the STS.

Table 11. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size* are shown (k = 10 voxels).*

Anatomy	BA	Coordinates			T-value	Size (mm^3)
		x	*y*	*z*		
Intact human PLW > All other stimuli (p < .001, uncorrected)						
R. Middle temporal	37	54	-66	4	5.19	427
R. Middle occipital	18	36	-92	10	3.92	117
R. Inferior occipital	18	34	-90	0	3.64	
L. Middle occipital	19	-44	-72	2	3.84	44
Intact human PLW > Baseline (p < .05, FWE corrected)						
L. Calcarine sulcus	18	-2	-92	10	6.52	1525
L. Cuneus	18	-4	-86	24	6.43	
R. Cuneus	18	8	-80	22	6.18	
L. Supplementary motor area	6	-8	2	56	5.98	403
R. Supplementary motor area	6	6	6	54	5.90	

**Missing values in the size column indicate an activation peak that is part of the cluster listed immediately above.*

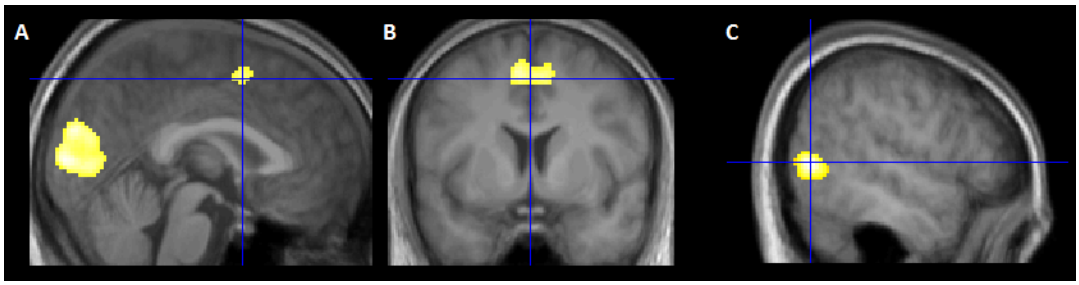

Figure 14. Panel A and B - activity occurring in the supplementary motor area (*intact human PLW > Baseline)*. Panel C - activity occurring in the EBA (*intact human PLW > All other stimuli).* The image on which activity is overlaid is the mean of the structural images from all participants.

The neural response to *intact human PLW > baseline* (no stimulus presentation) was also examined. At the p < .05 FWE corrected threshold, a large portion of the primary visual cortex was active in addition to the supplementary motor area (SMA) bilaterally (Figure 13). The latter sensitivity could possibly reflect the modulation of mirror neurons to the observation of human movement. Unfortunately, no other contrasts revealed any statistically significant results at the p < .05 (FWE, corrected) or p < .001 (uncorrected) thresholds.

MT+/V5 LOCALISER

At the $p < .05$ corrected threshold level, the *coherent > random motion* contrast showed significant activity in the right inferior occipital (MNI coordinates 44 -86 -2, $t = 5.31$, extent = 59 mm^3) and middle occipital (MNI coordinates 38 -86 6, $t = 4.87$) regions. As cluster size was small, activity was also considered at the $p < .001$ uncorrected level (Table 12). In the left hemisphere, a column of activation extended from the cerebellum through the fusiform, inferior and middle occipital gyri and middle temporal gyrus. A second column of activity in the right hemisphere extended from the inferior occipital, middle occipital and middle temporal regions. This cluster of activity appears to overlap with the human analogue of MT+/V5, as measured by Dumoulin *et al.,* (2000).

Table 12. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size are shown (k = 10 voxels, height threshold = p < .001, uncorrected).*

Anatomy	BA	Coordinates			T-value	Size (mm^3)
		x	*y*	*z*		
R. Inferior occipital	19	44	-86	-2	5.31	1266
R. Middle occipital	18	38	-86	6	4.87	
R. Middle temporal	37	54	-72	0	4.53	
L. Inferior occipital	19	-40	-84	-12	4.92	746
L. Middle occipital		-34	-94	10	4.18	
L. Inferior occipital	19	-42	-70	-6	3.96	
R. Postcentral	3	58	-22	46	4.53	233
L. Paracentral lobule		-2	-32	52	4.21	932
L. Middle cingulum		-6	-22	46	4.15	
R. Middle cingulum		8	-22	46	3.94	
L. Middle temporal	39	-40	-54	16	3.76	255
L. Middle temporal		-58	-66	18	3.72	
L. Precuneus	23	-8	-52	22	4.08	250
L. Posterior cingulum	30	-2	-44	18	3.80	
R. Postcentral	4	32	-34	72	4.07	203
L. Middle temporal	20	-48	-24	-12	4.02	92
L. Medial superior frontal	10	-6	60	10	4.01	670
L. Anterior cingulum	10	-6	48	-2	3.63	
R. Middle orbitofrontal	11	2	40	-10	3.60	
R. Middle temporal	22	54	-12	-12	3.98	91
R. Middle temporal	21	60	-6	-14	3.38	
L. Fusiform	37	-38	-50	-20	3.28	26
L. Cerebellum	37	-28	-46	-20	3.28	

**Missing values in the size column indicate an activation peak that is part of the cluster listed immediately above.*

RFX Analysis - Facial Motion (Video)

At the $p < .001$ uncorrected level, greater neural activity for observing upright versus inverted facial motion was seen in the bilateral STS. Activity extended to the middle temporal cortex, but only on the left hemisphere. Regions within the left calcarine sulcus, FG and precuneus also appeared responsive to upright facial motion (Table 13). These results support those reported in the group average analysis.

Table 13. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size* are shown (k = 10 voxels, height threshold = p < .001, uncorrected).*

Anatomy	BA	Coordinates			T-value	Size (mm^3)
		x	*y*	*Z*		
L. Calcarine sulcus	17	-6	-78	6	13.80	101
L. Calcarine sulcus	17	-10	-86	0	11.88	
L. Middle temporal	20	-52	-14	-12	11.37	62
L. Middle temporal	22	-60	-38	8	11.04	193
L. Middle temporal	22	-56	-46	12	9.03	
L. Middle temporal	22	-68	-34	6	6.91	
R. Superior temporal	22	60	-34	14	8.96	47
L. Calcarine sulcus	17	0	-70	16	7.53	31
L. Precuneus	18	2	-78	18	7.42	
L. Fusiform gyrus	37	-36	-38	-24	7.29	22

**Missing values in the size column indicate an activation peak that is part of the cluster listed immediately above.*

Contrast Estimates

A paired-samples t-test detected differences between the contrast estimates for activity evoked by facial motion videos or specific localiser stimuli in ROI defined by the previous results. The FFA, PPA and LOC responded more when participants viewed static faces, places and objects, respectively, than when they observed facial motion ($t_{(7)}$ = -4.36, p = .003, $t_{(7)}$ = -6.82, p = .001 and $t_{(7)}$ = -5.019, p = .002). The EBA also showed greater sensitivity to static bodies and intact point-light walkers than to facial motion ($t_{(7)}$ = -6.69, p = .001 and $t_{(7)}$ = -4.89, p = .002). Greater modulation by coherent motion over facial motion was seen in MT+/V5 ($t_{(7)}$ = -5.78, p = .001). There was no difference in the SMA activity evoked by dynamic faces or point-light walkers (p > .05). See Figure 15.

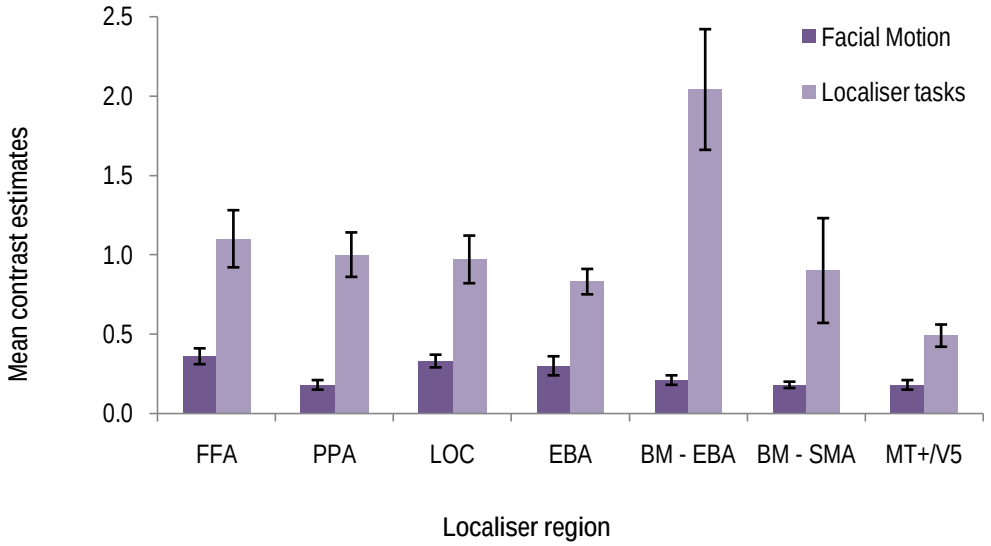

Figure 15. Contrast estimates (averaged across participants and hemispheres) for activity evoked by facial motion videos or localiser stimuli in ROI (FFA: faces > scrambled; PPA; places > scrambled; LOC: objects > scrambled; EBA: bodies > chairs, Body Motion (BM) - EBA: intact PLW > all other stimuli; BM - SMA: intact PLW > baseline and MT+/V5: coherent > random motion.

Contrast estimates were also analysed for activity occurring within the STS in response to participants observing facial motion, static faces, objects, places, point-light walkers, coherent motion and static bodies. The STS ROI was defined by visually locating the substrate in each individual dataset. To facilitate this process, the MNI coordinates (taken from the group average reported in Table 9) corresponding to this region were also used. The mean coordinates for the STS are reported in Figure 16. A one-way ANOVA revealed a significant main effect of stimulus type ($F_{(7)} = 8.18$, $p < .001$), whereby facial motion evoked the greatest activity within the STS complex ($M = 0.43$, $SD = 0.14$) compared to all other stimuli.

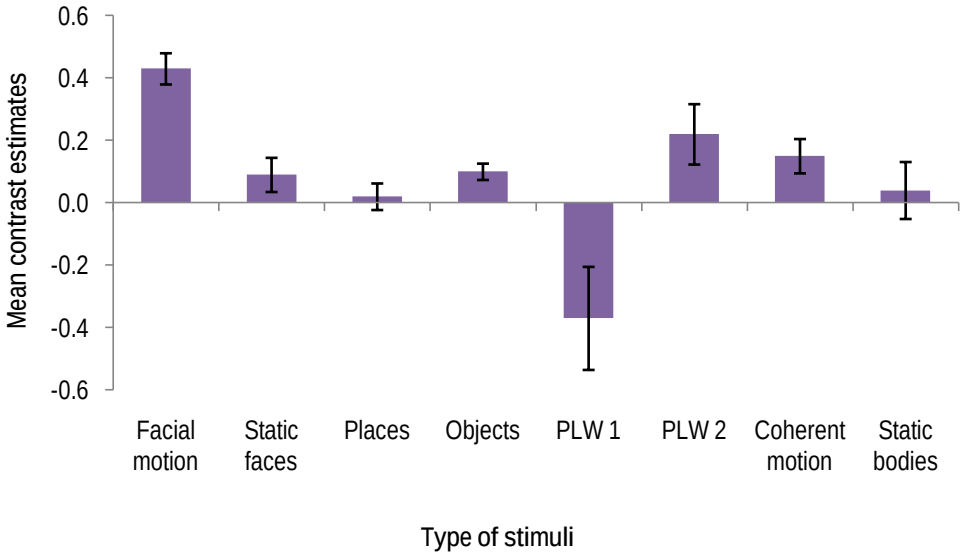

Figure 16. Contrast estimates (averaged across participants and hemispheres) for STS activity evoked by facial motion (upright > inverted), static faces (> scrambled), places (> scrambled), objects (> places), point-light walkers (intact PLW > baseline and intact PLW > all other stimuli), coherent motion (> random) and static bodies (> chairs). Right STS coordinates: $M = 51, -38, 9$; $SD = 2.45, 3.72, 4.46$. Left STS coordinates: $M = -55, -42, 9$; $SD = 5.36, 9.48, 5.06$.

5.4 Discussion

Neural Processing of Facial Motion Videos

Discriminating upright videos of facial motion evoked the greatest activity in the STS compared to discriminations with inverted types. Specifically, a large cluster was observed in the right posterior limb (pSTS). These findings corroborate previous studies which report the STS to be the region most strongly associated with the analysis of variant facial aspects (Puce *et al.,* 1998; Lloyd fox *et al.,* 2009; Schultz & Pilz 2009; Polosecki *et al.,* 2013; Schultz *et al.,* 2013). For example, Pelphrey *et al.,* (2005) found that the mid-posterior and right pSTS were sensitive to mouth and eye movements respectively. Similarly, there is evidence that the event-related N170 response to averted eye-gaze and mouth opening movements reflects the engagement of the STS (Puce *et al.,* 2000; Rossi *et al.,* 2014).

The perception of facial motion videos also evoked a response in part of the medial visual occipital cortex known as the lingual gyrus. Those who have examined the neural correlates of dynamic emotion recognition have reported activities occurring within portions of this substrate (Kilts *et al.,* 2003; Trautmann *et al.,* 2009; Trautmann-Lengsfeld *et al.,* 2013). By contrast, studies which implement computer-generated displays of nonrigid motion suggest that the lingual gyrus may only process categorical information. Indeed, Sarkheil *et al.,* (2013) observed a greater engagement of the lingual gyrus when participants were instructed to indicate the gender of a face compared to emotional ratings. However, it is possible that discrepant data is related to the subtype of motion viewed. For example, nonrigid motion appears to facilitate gender discriminations specifically because such facial aspects are functionally related to speech and expression which differ significantly between the sexes (Hill & Johnston, 2001). Nonetheless, the neural encoding of facial motion seems to be supported by early visual mechanisms.

There is also evidence to suggest that the lingual gyrus is influenced by the face selective fusiform gyrus in a feedforward and re-entrant manner (McKay *et al.,* 2012). While this cannot be fully commented on in the current study, small significant activities in regions corresponding to the FFA did emerge from the analysis. Yet such engagement could actually reflect the processing of facial form rather than of the motion contained within in it. In support of this claim, Schultz *et al.,* (2013) reported that both the FFA and OFA were sensitive to manipulations which distorted the frame rate but not frame order of dynamic facial stimuli. The authors explain such finding by suggesting that these ventral temporal regions are receptive to the increase in static information available from moving faces, rather than to the motion *per se*.

The analysis and decoding of facial movement also involves regions outside of the cerebral cortex. Large active voxels were seen in the left cerebellum during the discrimination of upright facial motion. It is widely acknowledged that the cerebellum has a role in motor functions, but there is evidence of this extending to sensory processing as well (Baumann & Mattingley, 2014). Lesions to this region can cause impairments on tasks detecting visual motion signals in noise, suggesting that it interacts with the dorsal stream (Jokisch *et al.,* 2005). At the time of writing, no study investigating facial motion processing has found this response. However, previous investigations which implement point-light walker stimuli have reported cerebellar activity (Grossman *et al.,* 2000; Sokolov *et al.,* 2014; Vaina *et al.,* 2001). For example, lobules Crus I and VIIB of the left lateral cerebellum exhibit an increased BOLD response during body motion perception (Sokolov *et al.,* 2012). The authors of this study further noted a bi-directional communication between the left lobule Crus 1 and right pSTS. Evidently, the STS region acquires many multimodal associations which are not limited to cortico-cortical connections.

Processing of Identity from Facial Motion

Facial motion is a cue to identity (Longmore & Tree, 2013; O'Toole *et al.,* 2002). Indeed, you might recognise a close friend by the way they smile or characteristically nod their head during conversations. While several behavioural investigations have attempted to uncover the mechanisms involved (reviewed in Chapter 4), there is hardly any neuroimaging data contributing to this open topic. Such issue was addressed in the current study.

The analysis revealed significantly greater activity occurring within the middle temporal and STS cortex during identity judgments from upright facial motion. Unlike Harris *et al.,* (2014) and Baseler *et al.,* (2014), no response was observed in the ventral temporal regions (OFA and FFA) typically responsible for individuating static faces (Rhodes *et al.,* 2009). This may be explained in reference to the stimuli set. Because all animations shared the same physical appearance, participants did not need to recruit these structures as there was no variant form features to encode. Instead, the STS complex was sufficient in processing facial identity when characteristic structural cues were absent. The inactivity of the OFA and FFA may also provide further support that these structures are only sensitive to static information contained with motion sequences (Schultz *et al.,* 2013). Since this did not vary between stimuli, there was no need to engage the ventral temporal cortex. Studies of patients suffering from prosopagnosia (caused by damage to the FFA) support these assumptions. Steede *et al.,* (2007) showed that while patient C.S. was impaired on static facial identity recognition tasks, he could use idiosyncratic facial motion to discriminate between different people. It further demonstrates that static and dynamic stimuli activate dissociated cognitive pathways.

Portions of the precentral gyrus also emerged as responsive to facial identity. This finding is not entirely unanticipated; there are many reports which show its association to general dynamic face processing (Sarkheil *et al.,* 2013; Schultz & Pilz, 2009; Trautmann-Lengsfeld *et al.,* 2013), perceiving socially neutral gestures (Saggar *et al.,* 2014), understanding societal inclusion or exclusion from bodily movements (Bolling *et al.,* 2013) and recognising or performing goal-directed actions (Grézes *et al.,* 2001). These findings have accordingly been interpreted as reflecting the putative involvement of mirror neurons (Rizzolatti & Craighero, 2004). Visual information is transmitted from V1 to the STS, and then to the polysensory neurons within the precentral gyrus (Cook *et al.,* 2003; Lepage & Théoret, 2006). In the context of the current data, it is possible that observing facial movements relating to identity engaged these mirror mechanisms. To gain clearer insights, future investigations are encouraged to replicate this study by implementing an execution phase with facial actions relating to self and other identities.

Comparison with Other Stimuli

A number of localiser tasks were conducted so that the neural correlates of facial motion perception could be fully evaluated. This served as a conservative criterion for establishing whether distinct or similar mechanisms are involved in processing different types of static and dynamic stimuli. Contrast estimates revealed that the FFA, PPA and LOC were significantly more active to static faces, places and object images respectively, than to facial motion videos. A similar effect was observed in the EBA, whereby static bodies evoked the greatest activation compared to dynamic faces. Greater modulation by coherent motion kinematograms was also seen in MT+/V5. These findings substantiate past literature (Ajina *et al.,* 2015; Amoruso *et al.,* 2011; Epstein *et al.,* 2003; Mullin & Steeves, 2011; Slotnick & White, 2013) and demonstrate that these neural substrates are not functionally specialised to process facial motion. Instead, the STS complex was found to be the most responsive to simultaneous sequences of rigid and nonrigid facial motion relative to these stimulus categories. This further indicates that the STS is highly specialised for processing human facial expressions (Schultz *et al.,* 2013).

In the context of body motion, the STS complex has also been consistently implicated in the processing of information from point-light animations (Grossman *et al.,* 2000; Grossman, 2005; Puce & Perrett, 2003). Conversely, the current study did not find any STS activity to point-light walkers (compared to all non-biological motion), but instead sensitivity of the EBA (neighbour to the STS). As previously discussed, this region only encodes visual information relating to body form (Downing *et al.,* 2001). This finding is unexpected and could possibly be due to the stimuli set moving too slowly to cause a significant response in motion-sensitive regions. By contrast, the observation of intact point-light walkers versus baseline (i.e. no stimuli presentation) evoked a significant response in the primary visual cortex and SMA. The latter substrate indicates involvement of frontal regions in the visual analysis of body motion. Indeed, several other neuroimaging investigations have also noted increased SMA activity to observed familiar biological motions (Hars *et al.,* 2011), repetitive finger actions (Holz *et al.,* 2008), point-light jumping and kicking (Ulloa & Pineda, 2007) and whole body gymnastics (Zentgraf *et al.,* 2005). Further, while this contrast did not reveal any response in the STS region, there is evidence to suggest that the SMA is involved in mediating communications between the STS and posterior parietal cortex (Koski *et al.,* 2003). Taken together, the SMA may be implicated in the accurate processing of biological motion by recovering lost object information (Ulloa & Pineda, 2007).

5.5 Conclusion

The current study found clear localised regions responding to specific types of visual stimuli. Static faces evoked a response in the FFA and OFA, while static bodies, places and objects activated the EBA, PPA and LOC respectively. Similarly, the motion-sensitive MT+/V5 region was selectively active

to coherent dot motion kinematograms. The results from the body motion localiser were however unexpected, with contrasts revealing active voxels in the EBA and SMA. As discussed, this could be due to slow frame rates. Regardless, such activations differed to those produced by discriminating videos or identities of facial motion. A greater neural response in the STS was observed here, suggesting that this region is the main neural substrate involved in processing all types of human movements. Now that this has been localised using highly realistic and ecologically valid stimuli, Chapter 6 will investigate whether the neural mechanisms behind facial motion perception are atypical in participants with ASD. Based on the results from Chapter 3, it is expected that participants with ASD will show significant abnormalities in regions of the STS and visual cortex.

CHAPTER 6 Neural Correlates of Facial Motion Perception in ASD

6.1 Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterised by a severe deficit in social communication and interaction (DSM-5). The ability to recognise socially relevant cues such as emotional states, intentions and thoughts from faces is a fundamental aspect of normal reciprocal social interaction (Sato *et al.,* 2012). It is likely that these impairments relate to a dysfunction of the brain system underlying face perception (Schultz *et al.,* 2000).

Behavioural investigations report domain-specific deficits in participants with ASD. Face perception is impaired relative to the recognition of inanimate objects, visual patterns and face-like stimuli (Davies *et al.*, 1994; McPartland *et al.,* 2011; Pallett *et al.,* 2014). For example, it has been shown that children with ASD are significantly less accurate than controls on tasks requiring them to discriminate a target from foil faces. However, when presented with car stimuli, they perform identically to participants with typical development (Rhodes *et al.,* 2014). Adults with ASD further experience difficulties when lip reading, detecting eye-gaze directions and discriminating the gender of faces (Deruelle *et al.,* 2004). More recently, the impaired recognition of facial identity and emotional expressions has also been observed (Sachse *et al.,* 2014). Greimel *et al.,* (2014) reported significantly higher error rates and reaction times when adults with ASD completed these tasks compared to controls. Interestingly, the ASD group's performance was comparable to that of the neurotypical children, indicating that poor face perception may be the result of stunted neural development (Greimel *et al.,* 2014). These findings indicate that ASD is associated with a failure to perceive facial stimuli and the information contained within them.

Eye-tracking data demonstrates that participants with ASD do not spontaneously attend to faces and spend less time looking at them compared to controls (Chawarska *et al.,* 2013). Indeed, children with ASD display reduced fixation patterns when presented with drawn faces of different emotional expressions (Ishii & Konno, 1987) or photographs of neutral female faces (Yi *et al.,* 2013). It has instead been shown that these participants gazed significantly longer at the socially irrelevant aspects of their surroundings (Papagiannopoulou *et al.,* 2014). It is probable that a reduced interest may lead to less experience with faces, thus resulting in face perception impairments. However, a lack of social orienting has not been consistently found throughout the literature. For example, although children with ASD were significantly impaired on tasks recognising facial expressions from dynamic movies, they did appropriately attend to the stimuli (Parish-Morris *et al.,* 2013). Other researchers report that participants with ASD actually spend more time observing faces compared to neurotypical controls (Elsabbagh *et al.,* 2013a; Webb *et al.,* 2010). In this context, increased fixations have been correlated with a poorer perception of

static faces (de Klerk *et al.,* 2014). These authors explain such finding by suggesting that participants with ASD focus more on individual face features, which would then increase the time needed to process the whole face as a unified gestalt.

It is possible that participants with ASD do attend to faces but favour irrelevant or uninformative aspects (Speer *et al.,* 2007). A study which shows that participants with ASD focus primarily on a person's chin, hairline or nose supports this hypothesis (Pelphrey *et al.,* 2002). A preferential reliance on the mouth region has also been observed in ASD (Guillon *et al.,* 2014; Joseph & Tanaka, 2003). Norbury *et al.,* (2009) reported that typically developing adolescents directed their gaze to the eyes of actors engaging in social interaction and speech whereas verbally competent ASD adolescents oriented to the mouth region only. Such behavioural discrepancy has also been correlated with the severity of social symptoms, highlighting the importance of eye information in social cognition (Jones *et al.,* 2008). This alternative perceptual strategy therefore suggests that participants with ASD are seeking a more verbal way of understanding socio-emotional cues (Grelotti *et al.,* 2002).

By contrast, not all studies observe face processing deficits. Wilson *et al.,* (2007) found that children with ASD and neurotypical controls performed identically to one another on tasks requiring them to discriminate familiar faces (e.g., school teacher). This could reflect methodological differences between investigations, particularly in the type of experimental paradigm implemented and whether it evoked high memory demands. For example, both Gepner *et al.,* (1996) and Weigelt *et al.,* (2013) found that recognition in ASD was only impaired when faces had to be remembered over a delay relative to immediate recall. From such data, face processing deficits in ASD may be partially process specific - that is, a problem only in remembering faces. Alternatively, discrepant data could be caused by factors relating to the heterogeneous quality of ASD symptomology, such as co-morbidity with other psychiatric disorders and severity of impairments (Nuske *et al.,* 2013).

Nonetheless, neuroimaging studies with ASD participants have reported abnormal activity in components of the face processing network, including the amygdala (Bookheimer *et al.,* 2008) and regions of the fusiform gyrus (FG; Corbett *et al.,* 2009). A recent meta-analysis consistently found hypoactivity of the left fusiform face area (FFA) in participants with ASD during face perception (Nickl-Jockschat *et al.,* 2014). Instead, these participants strongly activate regions involved in object recognition (Schultz *et al.,* 2000). Equally, there are studies which fail to report any abnormality of the FG in ASD (Perlman *et al.,* 2011). Perhaps it is irregular connectivity between regions of the social brain, rather than a deficit to a specific substrate, which cause face processing difficulties (Tardif *et al.,* 2007). However, eye-gaze patterns may be a source of discrepancy within the literature. For example,

instructing participants with ASD to focus on the eye region evokes normal FG activity (Hadjikhani *et al.,* 2004; Hadjikhani *et al.,* 2007; Perlman *et al.,* 2011).

While the existing literature has been highly informative regarding face processing in ASD, it is limited by its common use of static stimuli. Human faces are intrinsically dynamic whereby verbal communication and emotional expressions occur via the movement of specific facial muscles. Static representations, therefore, do not fully capture the mechanisms underlying face perception. Several studies with neurotypical participants have demonstrated that the addition of motion facilitates facial emotion (Bould & Morris, 2008), identity (Girges *et al.,* 2015; Spencer *et al.,* 2006), and gender (Hill & Johnston, 2001) recognition. This improvement also correlates with notable increases in the functional activity of face selective and emotion processing networks (Sato *et al.,* 2004; Trautmann *et al.,* 2009).

As discussed in Chapter 3, only a handful of research groups have specifically examined facial motion perception in ASD. Miyahara *et al.,* (2007) observed that at the individual level, participants with Asperger's Syndrome were not as sensitive to the happy face advantage (i.e. recognising this emotion more effortlessly than others) as were the controls when viewing dynamic facial affects. Enticott *et al.,* (2014) similarly found that neurotypical participants were more accurate than the experimental group when recognising anger and disgust from dynamic facial displays. Uono *et al.,* (2009) further reported that the integration of dynamic emotion and gaze direction cues, needed for joint attention, is impaired in Asperger's. Rating the naturalness of facial expressions which differ in speed is also problematic for participants with ASD (Sato *et al.,* 2013). The authors suggest this reflects a dysfunction in the earliest visual component of face processing. Interestingly, decreasing the speed of video presentations facilitates the ability of participants with ASD to recognise and imitate dynamic facial emotions (Gepner *et al.,* 2001; Tardif *et al.,* 2007). Collectively, these findings demonstrate that the impairment in facial motion perception may be related to a rapid visuo-motion integration deficit (Gepner & Mestre, 2002b).

In support of these behavioural data, neuroimaging studies have observed atypical functional activity in the substrates thought to underlie facial motion processing. Dapretto *et al.,* (2006) required children with ASD and neurotypical controls to imitate and observe facial emotional expressions. While both groups exhibited no behavioural impairment, only the typically developing children showed an enhanced activation in the pars opercularis of the inferior frontal gyrus (IFG). The authors suggest that such diminished activity reflects abnormal mirror neurons, meaning that participants with ASD cannot match observed facial actions with representations in their own motor repertoire. In addition, other researchers have reported less engagement of the amygdala, FFA and superior temporal sulcus (STS) when participants with ASD have viewed emotion and identity morphed videos (Pelphrey *et al.,* 2007). Rahko *et al.,* (2012) similarly reported hypoactivity of the STS in participants with ASD who were asked to scale the valence of dynamic emotions. As this temporal substrate has been highly implicated in the

processing of biological movements, a dysfunction here would significantly impact the perception of facial motion (Zilbovicius *et al.,* 2006). Furthermore, Sato *et al.,* (2012) also revealed hypoactivation of the STS, in addition to the amygdala, FG, middle temporal gyrus (MTG), medial prefrontal cortex (MPFC) and IFG when ASD participants discriminated the gender of dynamic emotional faces. A weakened bi-directional connectivity within the V1-MTG-IFG network in the ASD group was observed as well. These data accordingly point to abnormalities within pathways leading from primary visual areas to specialised higher-order regions in participants with ASD.

The present study aimed to further investigate dynamic face processing in adults with ASD. Rather than implementing videos of morphed static images or computer-generated synthetic motion, participants viewed facial motion captures. These were natural depictions of real human movements in the absence of residual spatial cues. The animations thus evoked both rigid and nonrigid motion, including speech expressions and eye-gaze patterns. The stimuli also shared the same appearance in order to limit interferences from individual differences in surface based visual cues. The participants with ASD and neurotypical controls were asked to discriminate between different facial motion sequences in a simple perception task. This experimental paradigm was similar to that implemented in Chapter 3. On the basis of those behavioural results, we would expect atypical neural activity to contribute to the impaired perception of facial motion in ASD (O'Brien *et al.,* 2014).

Further, presentations varied between upright and orientation-inverted stimuli. Studies using both static and dynamic faces have demonstrated that inversion affects face recognition by disrupting configural processing (Girges *et al.,* 2015; Valentine, 1998). As a result, accuracy on such face perception tasks is significantly reduced. Previous research has not found this effect in ASD (O'Brien *et al.,* 2014; Webb *et al.,* 2012), suggesting a failure to utilise configural strategies and a reliance on feature-based processing (Spezio *et al.,* 2007). Indeed, children with ASD show a superior perception of individual facial features and are better at recognising partially obscured faces than controls (Tantam *et al.,* 1989). Recent reviews, however, have highlighted inconsistencies surrounding this manipulation (Weigelt *et al.,* 2012) and instead report that participants with ASD are similarly sensitive to face inversion effects (Hedley *et al.,* 2014). This could be a consequence of studies implicating the often unrealistic static facial displays (O'Brien *et al.,* 2014).

6.2 Methods and Materials

Participants

Ethical approval was obtained from Brunel University. Two groups of adults participated in the current study: 6 individuals with ASD (2 male, 4 female, age: *M* = 35.17 years, *SD* = 8.38 years) and 8 neurotypical controls (2 male, 6 female, age: *M* = 24.75 years, *SD* = 3.92 years). Participants with ASD had a current clinical diagnosis of an Autism Spectrum Disorder based on the DSM-IV-TR criteria. In

addition, all participants with ASD met research diagnostic standards for ASD based on the Autism Diagnostic Observation Schedule (ADOS; Lord *et al.,* 1999) or the Autism Diagnostic Interview (ADI-R; Lord *et al.,* 1994). Exclusionary criteria for neurotypical and ASD participants included schizophrenia, epilepsy, genetic disorders, birth defects and significant visual impairments. Exclusionary criteria for neurotypical participants also included developmental abnormalities or first degree relatives with ASD.

For both groups, the Autism Quotient (Baron-Cohen *et al.,* 2001) was administered. This measures the number of autistic traits in participants with normal intelligence on a range of qualities such as social skills, attention switching, imagination and communication. The Ravens Standard Progressive Matrices (Raven *et al.,* 2001) and the Benton Facial Recognition Test (Benton *et al.,* 1983) were also used to assess non-verbal IQ and recognition of unknown static faces respectively (Table 14).

Table 14. *Characteristics of adults with ASD and the neurotypical group.*

		Neurotypical	ASD	*P*-value
		n = 8	*n* = 6	
Age	*Mean*	24.75	35.17	0.009
	Range	21 - 32	22 - 44	-
Standard Progressive Matrices*	*Mean*	54.75	50.40	0.155 ns
	Range	53 - 60	41 - 57	-
	% score	91.25%	84%	-
Benton Facial Recognition*	*Mean*	50.38	46.60	0.061 ns
	Range	45 - 54	46 - 49	-
	% score	93.30%	86.30%	-
Autistic Quotient*	*Mean*	14.86	30.80	0.008
	Range	10 - 26	15 - 45	-

**Maximum possible scores for the Standard Progressive Matrices = 60; for the Benton Facial Recognition test = 54; scores between 11 and 22 on the Autistic Quotient scale were considered average.*

Stimuli

Marker-less technology was implemented in order to create facial motion stimuli. The movement of 15 non-professional human actors, as they recited a range of poems, was captured and retargeted onto a standard avatar. Final animations exhibited both rigid (head rotations and translations) and nonrigid (expressional changes) motion patterns, as well as natural speech and eye-gaze movements. Individual differences in facial form were limited by all animations sharing the same appearance. Orientation-inverted versions were also presented. Chapter 4 discusses the stimuli method in detail.

Functional MRI task and design

Stimuli conditions were configured in MATLAB (MATLAB 7.10.0. Natick, Massachusetts: The MathWorks Inc., 2010) and presented using an LCD display with a resolution of 1024 x 768 and a 60Hz refresh rate. Participants viewed either upright or orientation-inverted facial motion. Within each block, 2 videos were shown successively and the task was to decide whether they were identical or

different from each other (two forced choice interval procedure). Responses were made via a button press in the scanner (left = yes they are identical, right = no they are not identical). Stimuli sequences were separated by a 1-second interstimulus interval (ISI) and participants had 5 seconds to make their choice. This procedure carried on for 40 blocks in total. All stimuli were presented for 8 seconds and the task took approximately 16 minutes to complete.

Image acquisition and analysis

Images were acquired on 3.0 Tesla Siemens Magnetom Trio scanner with a 32 channel array head coil. Functional MRI was performed using a standard gradient echoplanar imaging sequence (EPI) (TR 3000ms, TE 30ms, flip angle 90 degrees, 3mm slice thickness, 64 x 64 matrix, 160 contiguous axial slices, bandwidth 1396Hz/pixel). Functional scanning runs comprised up to 320 volumes lasting 16 minutes. Anatomical T1-weighted MPRAGE 3D MRI sequence images were also acquired during the scanning session (TR 1830ms, TE 4.43ms, flip angle 11 degrees, 160 axial slices, 1 mm isotropic voxels, 256 x 256 matrix, bandwidth = 130 Hz/pixel). Visual stimuli were projected onto a screen (via a Sanyo LCD projector, PLC-XP1000L, native resolution = 1024 x 768) in the bore of the magnet and viewed through an angled mirror above the head coil. Stimuli were shown within a window measuring 420 x 420 pixels on the screen. At the effective total viewing distance (from projection screen to the coil mirror to the participants' eyes) of approximately 82cm, this corresponded to 12.0^{o} x 12.0^{o}.

The data were pre-processed and analysed using Statistical Parametric Mapping (SPM8, Wellcome Institute of Cognitive Neuroscience, London, UK) in Matlab. In each individual data set, the images were corrected for head movement by realigning each EPI volume to match the volume in the first scan. The resulting images were then normalised to sterotaxic Montreal Neurological Institute (MNI) coordinates using trilinear interpolation. As a final pre-processing step, the normalised images were smoothed using a Gaussian filter with a full-width at half maximum parameter set to 8mm.

The analysis was based on a regular whole-brain SPM approach. Statistics were performed separately at each voxel and modelled using a box-car function convolved with a canonical hemodynamic response function. This was carried out for each participant in each group (although a separate group average for ASD and control data is reported here). Contrasts were defined to compare the neural difference between upright and inverted facial motion by subtracting one beta from another (as indicated by the use of the ">" sign). A second-level random effect analysis (RFX) was also conducted based on individual statistical parameter maps for the contrast upright > inverted facial motion. Here, a two-sampled t-test was used to detect significant neural differences between the neurotypical and ASD participants (contrast = neurotypical > ASD and ASD > neurotypical).

6.3 Results

Data from one ASD participant was excluded from the analysis due to incompletion of the experiment. At the individual level, the ASD data was highly variable between participants and specific neural substrates were not consistently activated across the whole group (see Table 15 for an example). Factors such as small sample size and the level of heterogeneity amongst participants with ASD (e.g., symptom severity or associated disorders) most likely contribute to these inconsistent effects. It was therefore appropriate to focus on the group average data.

Table 15. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size (mm^3) are shown (k = 10 voxels, height threshold = p < .05, FWE corrected)*

Participant Number	Anatomy	Coordinates			T- value	Size (mm^3)
		x	*y*	*z*		
1	L. Middle temporal	-66	-38	-2	5.78	37
2	*					
3	*					
4	L. Middle occipital	-18	-108	6	5.12	76
5	R. Cerebellum Crus 1	46	-54	-28	6.54	43
	R. Superior temporal	60	-12	-6	5.71	65
	R. Middle temporal	52	-48	12	5.63	89
	R. Superior temporal	52	-34	8	5.02	
	L. Superior temporal	-58	-44	14	5.43	15

**Individual data sets which did not survive thresholding at the corrected p < .05, FWE corrected level.*

First-level Data (Group average)

The effect of upright facial motion relative to inverted facial motion (UP > IN) was evaluated across both groups (Table 16, Figure 17). For the experimental group, it is important to note that an uncorrected threshold value ($p < .005$) was used as no active voxels survived the $p < .05$ (FWE, corrected) threshold. This means that the data is not directly comparable between groups. However, it does indicate that the regions involved in facial motion processing differ between participants with ASD and neurotypical controls.

A whole brain analysis with neurotypical control data revealed significant neural activations in the bilateral pSTS, extending into the middle temporal gyrus (MTG). Activity was also observed in the bilateral lingual gyrus, right FG, left cerebellum, IFG (pars triangularis) and a region of the right precentral gyrus known to contain parts of the dorsal premotor cortex (dPMC). By contrast, the ASD group showed engagement of the right anterior STS, extending into inferior and middle portions of the temporal lobe. In addition, the FG and dPMC was active on the right hemisphere. The left inferior and middle frontal gyri were also responsive to facial motion stimuli in participants with ASD (Table 16, Figure 17).

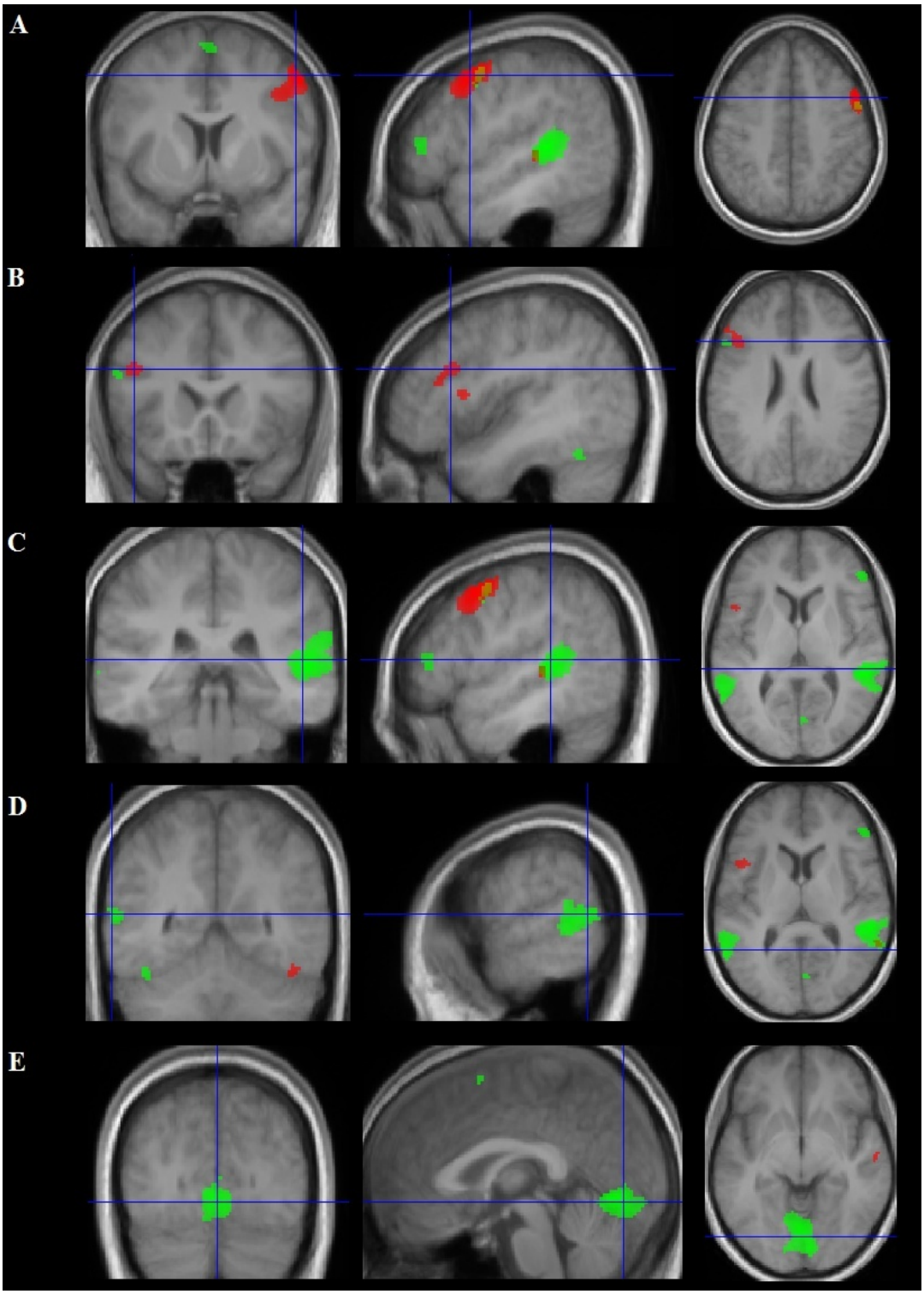

Figure 17. Red activity represents ASD group data ($p < .005$, uncorrected) whereas green activity demonstrates that evoked by neurotypical controls ($p < .05$, FWE corrected). The mustard yellow foci are where the BOLD responses from both groups overlap. The main foci are indicated by the crosshairs on panels (A) left frontal cortex; (B) precentral gyrus; (C) right STS cortex; (D) left middle temporal cortex; and (E) bilateral lingual gyrus. The image on which activity is overlaid is the mean of all the structural images from participants in both groups. Coronal, saggital and axial views are presented.

Table 16. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size (mm^3) are shown (k = 10 voxels).*

Upright > Inverted Facial Motion												
Anatomy	Neurotypical Controls (p < .05, FWE)						ASD (p < .005, uncorrected)					
	BA	Coordinates			*T*-value	Size	BA	Coordinates			*T*-value	Size
		x	*y*	*z*				*x*	*y*	*z*		
R. STS	22	52	-36	8	7.61	858	22	60	-14	-4	3.15	30
R. STS	48	68	-36	22	5.72		21	50	-32	4	2.94	22
R. STS	42	60	-36	20	5.57							
L. Lingual gyrus	17	-2	-72	-4	7.25	899						
R. Lingual gyrus	18	6	-86	-6	6.49							
L. Cerebellum	18	-8	-82	-14	6.08							
L. Middle temporal	37	-62	-56	10	6.36	402						
L. Middle temporal	21	-62	-48	8	6.18							
R. FG	37	42	-44	-24	5.65	38	20	36	-6	-42	3.66	47
L. Cerebellum	37	-42	-54	-24	5.53	50						
L. Cerebellum	37	-40	-44	-26	5.07							
R. Precentral	6	56	4	42	5.20	57	6	52	8	48	4.35	393
L. Inferior frontal	45	50	38	8	5.57	63	48	-52	12	8	2.90	59
L. Middle frontal							48	-44	20	26	3.03	153
L. Middle frontal							45	-48	28	20	2.93	
R. Inferior temporal							37	46	-54	-20	2.96	38
R. Middle temporal							21	62	-50	10	2.95	24

Contrast Estimates

The contrast estimates for activity evoked in the STS in response to facial motion was analysed across individual participants (and then averaged within groups). The STS was defined by visually locating the substrate in each individual dataset. To facilitate this process, the MNI coordinates (taken from the group average reported in Table 16) corresponding to this region were also used. A paired-samples t-test revealed that neurotypical participants engaged the right ($t_{(4)}$ = -3.07, p < .05) and left ($t_{(4)}$ = -7.24, p = .002) STS more strongly compared to participants with ASD (Table 17, Figure 18).

Table 17. *Mean (and standard deviation) contrast estimate values for activity evoked in the STS in response to facial motion in the neurotypical controls and ASD group.*

	Mean	SD
Right Hemisphere		
ASD	0.10	0.80
Neurotypical Controls	0.48	0.25
Left Hemisphere		
ASD	-0.02	0.13
Neurotypical Controls	0.38	0.11

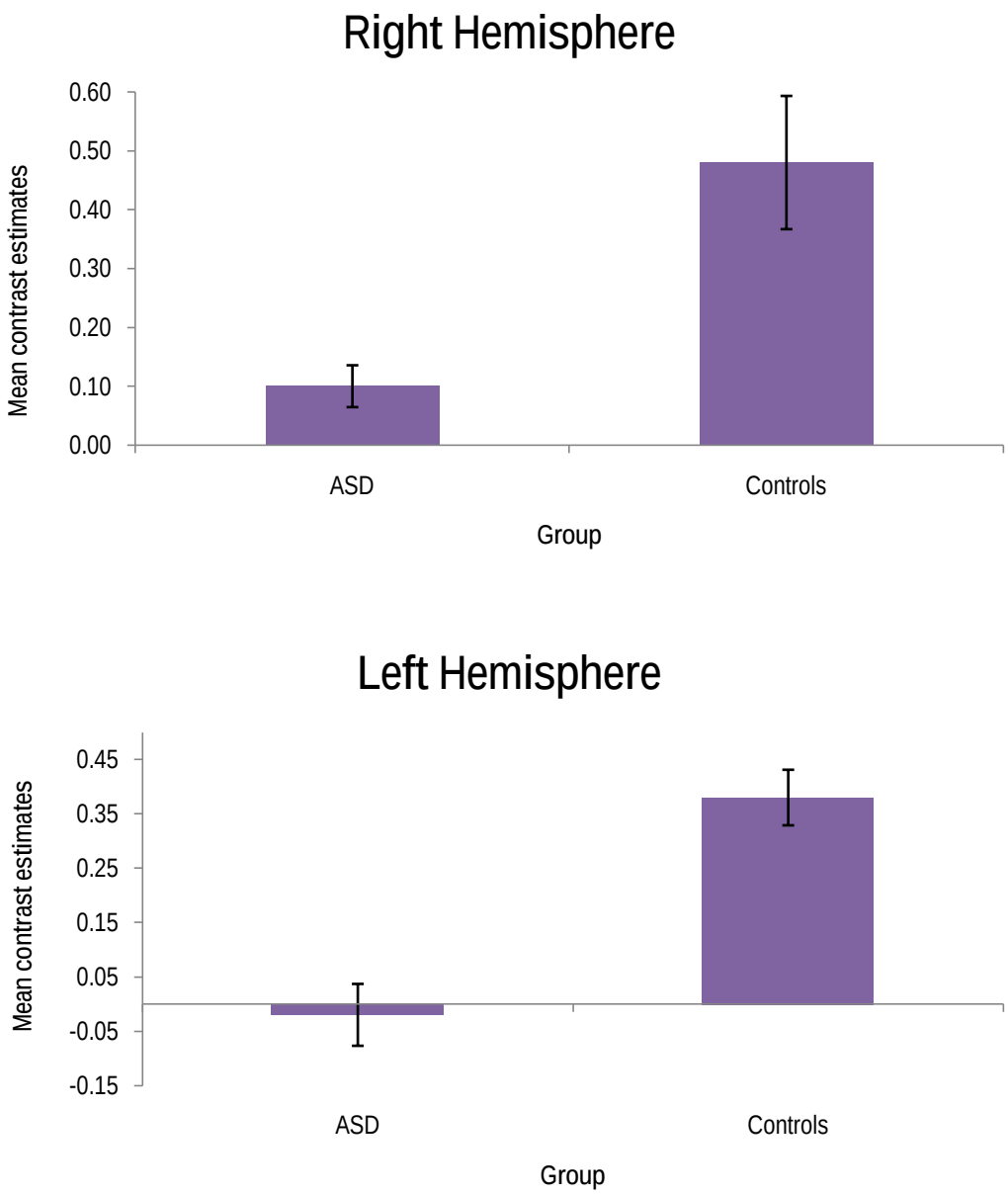

Figure 18. Mean contrast estimates (with standard error bars) for activity evoked in the left and right STS in response to facial motion across neurotypical controls and ASD groups.

RFX Analysis

While informative, group average data can sometimes appear misleading because the responses from a small number of participants might give the impression of a universal result that is not actually present within the entire group. It is therefore important to conduct a second-level RFX analysis. By doing so, we can observe which foci of activation can be generalised to the population being tested. The following results are reported at the $p < .001$ uncorrected level (Table 18).

For the UP > IN contrast, neurotypical controls elicited greater activity in several occipito-temporal, parietal and subcortical substrates compared to adults with ASD (Figure 19). This included a right hemisphere column extending across the cerebellum (CRUS I), V1, lingual gyrus, FG and inferior occipital gyrus. On the left hemisphere, greater activity emerged within the precuneus, inferior parietal lobule (IPL) and temporal pole. Engagement of the bilateral thalamus was also observed in neurotypical controls. The ASD group, however, showed greater significant activation within the right inferior frontal operculum and the superior and middle occipital gyri relative to neurotypical controls.

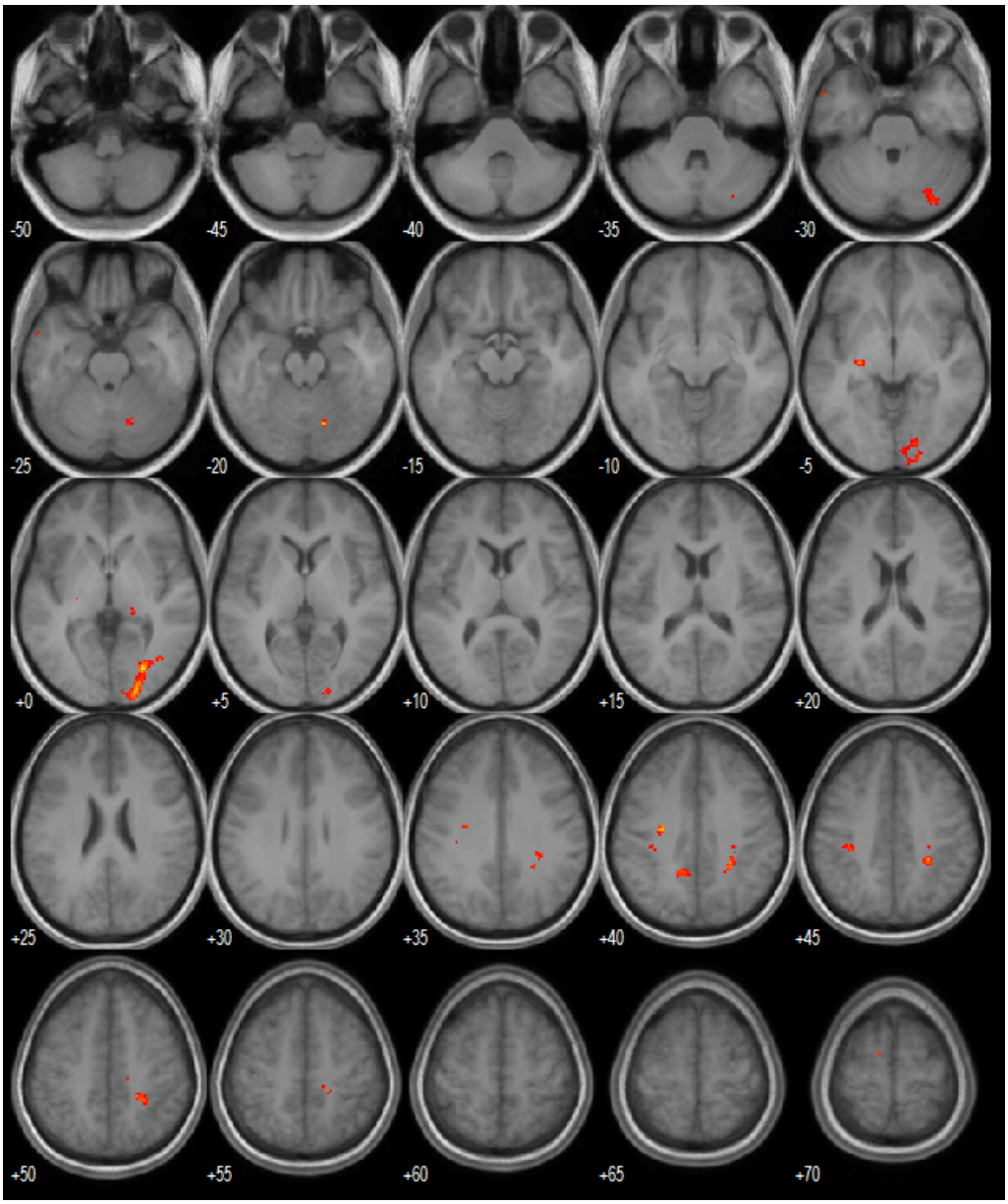

Figure 19. Montage of the results from the RFX analysis ($p < .001$, uncorrected) demonstrating the neural regions where neurotypical controls evoked more activity in response to perceived facial movements compared to participants with ASD.

Table 18. *The coordinates of the foci of activation in MNI space, their T-values and the cluster size (mm³)* are shown (k = 10 voxels).*

Anatomy	Neurotypical Controls > ASD (p < .001, uncorrected)					
	BA	Coordinates			*T*-value	Size
		x	*y*	*z*		
R. FG	18	26	-76	0	10.99	228
R. Calcarine sulcus (V1)	18	22	-92	2	7.36	
R. Lingual gyrus	18	16	-84	-6	5.25	
R. Cerebellum 6	18	14	-68	-20	6.99	23
L. Postcentral (IPL)	3	-34	-30	44	5.72	40
L. Thalamus		-14	-22	14	5.29	18
L. Thalamus		-4	-20	12	4.66	
R. Inferior occipital	37	40	-68	0	5.27	14
L. Temporal pole	21	-56	6	-28	5.12	19
L. Precuneus		-12	-50	40	5.07	33
R. Cerebellum CRUS I		28	-76	-32	4.92	45
R. Cerebellum CRUS I		36	-78	-28	4.49	
R. Thalamus		18	-26	2	4.34	11
	ASD > Neurotypical Controls (p < .01, uncorrected)					
R. Inferior frontal operculum	48	-42	12	10	3.72	14
L. Superior occipital		-14	-96	34	3.60	22
L. Middle occipital		-40	-86	22	3.26	16

**Missing values in the size column indicate an activation peak that is part of the cluster listed immediately above.*

6.4 Discussion

During observation of facial motion, neurotypical participants activated the pSTS, MTG, lingual gyrus, FG, cerebellum, IFG and dPMC. This pattern of activity was not consistently found in the participants with ASD reported in the current study. While the group data revealed some similar engagement of the STS, MTG, FG, IFG and dPMC to the neurotypical controls, participants with ASD activated a number of additional regions including portions of the inferior temporal gyrus and middle frontal gyrus. In addition, analyses with contrast estimates indicated that the STS region was less active in adults with ASD relative to neurotypical controls during the viewing of facial motion.

Previous neuroimaging studies with neurotypical participants have demonstrated the crucial involvement of the STS in the visual processing of changeable and dynamic face components (e.g., Atkinson *et al.,* 2011; Polosecki *et al.,* 2013; Puce *et al.,* 2000; Puce & Perrett, 2003; Schultz *et al.,* 2013). For example, De Winter *et al.,* (2015) reported consistent lateralisation in the right pSTS to videos of chewing actions or fearful expressions relative to scrambled faces. Harris *et al.,* (2014) also found this region to be most responsive to dynamic emotion and identity morphed animations. In participants with ASD, however, a number of existing investigations have shown a clear dysfunction of the STS during various facial motion processing tasks (e.g., Pelphrey *et al.,* 2007; Rahko *et al.,* 2012; Sato *et al.,* 2012). Indeed, Redcay *et al.,* (2013) found that participants with ASD evoked significantly less activity in the pSTS during interactive joint attention tasks conducted with dynamic faces. In the context of eye movements, Pelphrey *et al.,* (2005) asked participants to observe a virtual actor looking towards either a checkerboard that appeared in their field of view (congruent trial) or an empty space

(incongruent trial). While control participants evoked more activity in the right STS during incongruent gaze shifts, the adults with ASD exhibited undifferentiated responses between trials. The lack of incongruency effects within the STS has also been observed when adolescents with ASD view biological actions that are compatible or mismatched with an actor's prior emotional response towards an object (Ahmed & Vander Wyk, 2013). This suggests that participants with ASD may experience difficulty integrating information from biological motion with social contexts (Pelphrey *et al.,* 2011). These findings, combined with the results of the current study, indicate that an abnormality of the STS contributes to the facial motion perception deficits seen in ASD.

Lateralisation in the left inferior and middle frontal gyri to perceived facial movements was observed in the ASD group. The neurotypical participants, however, did not show strong engagement of such frontal regions and specifically lacked activity in the middle frontal cortex. The RFX analysis also confirmed that the inferior frontal gyrus was more active in participants with ASD compared to neurotypical controls. To date, only two other studies have reported a somewhat similar finding. Rahko *et al.,* (2012) observed increased IFG activity in participants with ASD compared to controls during the valence scaling of dynamic facial expressions. Martineau *et al.,* (2010) also found greater bilateral activation of the IFG (pars opercularis) during observation of hand movements in participants with ASD relative to controls. The authors take this as evidence of an atypical mirror neuron system (MNS). As discussed in previous chapters, the MNS is a fronto-parietal organisation underlying stimulation processes involved in observing and executing biological actions (di Pellegrino *et al.,* 1992). Abnormalities within this system may be at the core of social deficits in ASD (Vanvuchelen *et al.,* 2013).

Alternatively, it may be possible that participants with ASD are evoking a differential pattern of BOLD activity in order to process facial motion. McKay *et al.,* (2012) reported that while no behavioural discrepancies were observed between participants with ASD and neurotypical controls, each group activated significantly different substrates when perceiving biological (body) motion. Unfortunately, as task accuracy data was not collected in the current study, it remains unknown whether atypical frontal activity manifests behaviourally or indeed reflects compensatory strategies taken in ASD. However, the role of alternative perceptual mechanisms may be rejected, especially if we consider the results from O'Brien *et al.,* (2014). Using a similar paradigm, these authors demonstrated that participants with ASD were impaired on tasks involving the discrimination of sequences from averaged facial motion captures. It would seem that any additional neural activity echoes the deficit individuals have in perceiving facial motion from these displays.

An RFX analysis was also conducted in order to allow for population inference. Consistent with the group average data, the analysis highlighted many regions that were statistically less responsive in

participants with ASD during facial motion perception compared to neurotypical controls. This included portions of the cerebellum, primary visual cortex (V1), lingual gyrus, FG, inferior occipital gyrus, temporal pole, precuneus, IPL and thalamus. These findings are broadly consistent with previous data. For example, Jack and Morris (2014) found a reduced interaction between the cerebellum (CRUS I) and pSTS when participants with ASD were engaging in mentalising tasks. Further, both Sato *et al.,* (2013) and Rahko *et al.,* (2012) have reported hypoactivation of V1 in participants with ASD who viewed dynamic videos of specific facial expressions. As stimulus feature extraction (including motion detection) is thought to occur within this area, a deficit within V1 would indicate problems in early visual processing (Kröger *et al.,* 2014). This idea is further supported by participants with ASD evoking less activity in the thalamus. Here, cyclic activity of visual relay neurons modulates signal transmission during early input stages (Lorincz *et al.,* 2009). This in turn may have a negative impact on later or higher-order stages of facial motion processing.

The RFX analysis also revealed decreased sensitivity of the IPL and precuneus in participants with ASD. Both regions have been suggested to form part of a larger putative MNS (Grossman *et al.,* 2010) due to their involvement in action perception (Rizzolatti & Craighero, 2004). At the time of writing, no other study investigating facial motion perception in ASD has reported comparable results. However, reduced functional connectivity between portions of the STS and IPL has been found in participants with ASD who viewed point-light bodies expressing emotional states (Alaerts *et al.,* 2014). Kana *et al.,* (2014) also linked the IPL with theory of mind abilities, showing that hypoactivity within this region occurs when participants with ASD take part in intentional causal attribution task.

6.5 Conclusion

In the current study, participants were required to view displays of human facial motion in the absence of residual spatial cues which may confound perception. The aim was to extend, and build upon, previous research that had implemented dynamic videos derived from morphed static faces (e.g., Pelphrey *et al.,* 2007). As the facial motion captures closely depicted real human motion independently of other cues, a better experimental model of face processing in ASD could be produced. The findings revealed that the key component of human facial motion processing - the STS - was atypical in participants with ASD during the processing of facial motion. In addition, the participants with ASD evoked less activity in regions of the visual cortex and fronto-parietal structures (e.g., IPL and precuneus) assumed to comprise the MNS. These deficits could potentially contribute to the social cognitive deficits seen in ASD.

CHAPTER 7 General Discussion and Conclusion

7.1 Overview

Body language, facial expressions and eye movements provide an abundance of information necessary for mediating social communication (Knappmeyer *et al.,* 2003). From such visual input, one can infer the identity, gender, intentions, thoughts and moods of others (Blake & Shiffrar, 2007). From an evolutionary perspective, the correct perception and interpretation of another's actions is fundamental for survival (Atkinson *et al.,* 2004). For example, humans need to understand that a smiling expression signals approachability or mating opportunities, whereas frowning suggests antagonism. Incorrect analysis or a failure to perceive these biological movements in the first place, could potentially cause physical and emotional harm (Leopold & Rhodes, 2010). Accordingly, the human visual system has developed a unique ability to perceive biological movements even when the displays are impoverished or lack a clear form (Johansson, 1973).

Not all individuals, however, are proficient at understanding social information. Indeed, decades of research has shown that ASD is associated with an impaired recognition of faces, emotional expressions and intentions (Camargo *et al.,* 2014; Dawson *et al.,* 2012; Heaton *et al.,* 2012; Williams & Happé, 2009). Early accounts proposed that a deficit in comprehending high-level mental states or integrating information at different cognitive levels was at the root of these symptoms (Baron-Cohen *et al.,* 1985; Frith, 1989). Alternatively, it is possible that poor social cognition actually arises, in part, from a fundamental deficit in discriminating the movement of others (Moore *et al.,* 1997). In support of this hypothesis, several studies have reported that a weakened perception of body motion correlates with social symptom severity in ASD (Blake *et al.,* 2003; Cook *et al.,* 2013; Hubert *et al.,* 2007).

While the existing data has been highly informative, there is a paucity of research exploring the identification of dynamic faces in ASD. Although, if we are to assume that biological motion impairments are accountable for a reduced social cognition, then it is essential we actually pay attention to the facial regions. The purpose of this thesis was to thus rectify this issue (Chapters 3 and 6). In addition, there was also a need for control data. Facial motion perception in the neurotypical population had been scarcely investigated, especially with regards to the implementation of ecologically valid stimuli and understanding how low-level visual mechanisms contribute to the successful processing of facial motion. Chapters 2, 4 and 5 were therefore concerned with these research questions.

7.2 The Stimuli Dilemma

The perception of facial motion has primarily been investigated using unrealistic or abstract stimuli such as implied motion pictures, morphed videos from static images, cartoon avatars or synthetic faces

depicting computer-generated motion (e.g., Harris *et al.,* 2014; Lander & Bruce, 2003; Lee *et al.,* 2010; Pelphrey *et al.,* 2007). It is likely, however, that these representations do not fully engage the underlying mechanisms (Schultz & Pilz, 2009). On the other hand, investigations which do implement naturalistic videos often fail to control for irrelevant non-motion data even though such information could confound perception (e.g., Lander & Bruce, 2000). To address these problems, Hill and Johnston (2001) described a method to explore motion-based information independently of other cues. They recorded the facial movements of twelve actors reciting jokes and then applied it to the same three-dimensional face model. The benefit of such stimuli was that although they exhibited changes in head orientation and expression free of spatial cues, they still resembled a real human moving face. Accordingly, the empirical studies outlined in Chapters 2 and 3 employed these facial motion captures. It became evident, however, that the Hill and Johnston stimuli were not very detailed and sometimes appeared impassive or expressionless. In turn, this could have potentially reduced the amount of socio-emotional information available for successful recognition (O'Brien *et al.,* 2014).

As a result, it was important to build upon the pioneering work of Hill and Johnston by creating new stimuli (Chapter 4). Marker-less technology recorded a full range of facial motion from non-professional actors narrating poems, which was then applied to a realistic computer-generated face (Girges *et al.,* 2015). Their validity was assessed via a behavioural study with neurotypical participants. The purpose of this was to ensure that: (1) each video still represented the initially recorded motion; and (2) socio-emotional information could be recognised from these displays. Participants were therefore tested on their ability to discriminate between different video sequences or different unfamiliar identities in a two-alternative forced choice procedure.

In corroboration with previous data, the results indicated that neurotypical participants could accurately use idiosyncratic motion patterns in order to perceive the sequence or identity of actors. Further, orientation-inverted stimuli were also presented so that the role of configural processing could be assessed. As expected, participants committed more errors with manipulated stimuli, therefore indicating that facial motion is not processed via the analysis of low-level visual cues (Watson *et al.,* 2005). This was particularly true for judgments concerning identity, meaning that the inversion effect is sensitive to task type and what information needs to be extracted (Girges *et al.,* 2015).

The significance of this study was that it highlighted several advantages regarding the stimuli. Unlike those inspired by the Facial Action Coding System (Ekman & Friesen, 1978), the current dynamic faces included both rigid and nonrigid components. This facilitated in keeping the stimuli as close as possible to real life, especially if we consider that changes in head pose are normally accompanied by changes in facial expression. In addition, the use of marker-less technology allowed motion in all facial regions – not just those identified by facial markers – to be captured. The advantage here is that subtle

or extremely implicit movements were included in the final animation. Accordingly, the use of such stimuli may give rise to a more accurate and improved model of face perception in both neurotypical and ASD populations. These new facial motion captures were implemented in Chapters 5 and 6.

7.3 Facial Motion in Neurotypical Controls

Alpha Suppression

ERP analysis identifies components by averaging across several trials so that any activity not phased-locked to the stimulus is removed (Dawson, 1951). This method, however, only represents part of the total neural response, meaning that any induced activity is disregarded (Rossi *et al.,* 2014). To alleviate this issue, electrophysiological investigations have instead observed the power changes occurring within neural frequency bands. The study presented in Chapter 2 therefore measured alpha wave suppression in response to whole-face human motion. These oscillations were examined because they index visual activity (Berger *et al.,* 1926). No prior published EEG study had done this, even though such investigations would significantly advance our understanding of how low-level visual mechanisms contribute to the processing of facial motion. Neurotypical participants thus viewed the Hill and Johnston facial motion captures and discriminated between stimuli sequences in a simple one-back task. This was repeated for orientation and polarity-inverted facial motion. Activity was analysed across occipital and parieto-occipital scalp locations.

A number of significant and unexpected findings emerged from the data (Girges *et al.,* 2014). Upright facial motion actually evoked a transient increase in alpha power during the first 300ms post motion onset over both electrode sites. Essentially, this can be interpreted as less activity occurring within the visual cortex. This response was not observed for orientation or polarity-inverted faces, indicating that a higher level of information processing transpires when the brain is responding to unfamiliar stimuli (Gauthier & Tarr, 1997). However, this argument does not explain why there was subsequent alpha suppression to upright facial motion from 300ms onwards. In Chapter 2, this delayed processing is discussed in terms of gating mechanisms and filtering out irrelevant visual inputs, such as form cues, because these provided no additional information in this study (May *et al.,* 2012).

Another major finding of this study was that upright facial motion yielded the most amount of alpha suppression compared to the other face types only at parieto-occipital sites. This selectivity could reflect the activation of specialised structures which have a role in processing biologically relevant and familiar visual stimuli. This difference did not emerge over occipital locations though, suggesting that early visual processing occurs irrespective of orientation or luminance reversal (Kostandov *et al.,* 2010). Indeed, stimulus extraction and simple motion detection (Kröger *et al.,* 2014), two functions that are thought to operate in such regions, would have to occur regardless of novel viewing conditions. On

the other hand, it is important to highlight that studies which have implemented inverted point-light walkers or static faces have not observed a similar effect (e.g., Hirai *et al.,* 2011; Itier & Taylor, 2002). For example, Jokisch *et al.,* (2005) reported that the amplitude of the early N170 component (sourced over the middle occipital gyrus) was modulated by upright biological motion in comparison to inverted types. Further investigation is evidently needed here.

To conclude, the data discussed in Chapter 2 provided some new insights concerning the visual analysis of facial motion. Unfortunately, the neuroimaging techniques used were limited in identifying which specific neural substrates were driving the temporal differences between upright and manipulated stimuli. While it could be speculated that overlapping activity from the STS or dorsal stream was behind the parieto-occipital preference for upright facial motion (Blanke *et al.,* 2002; Matsumoto *et al.,* 2004; Puce *et al.,* 2000), nothing could be determined with confidence. For that reason, it was more appropriate to utilise fMRI in subsequent Chapters.

Neural Correlates of Facial Motion Processing

The purpose of the study outlined in Chapter 5 was threefold:

1. Identify which neural substrates processed facial motion when representations were realistic but contained no confounding spatial cues (i.e. testing the new stimuli).
2. Investigate the neural correlates of identity recognition from these representations.
3. Compare the mechanisms underlying dynamic face processing with that of other biological and inanimate stimuli (presented statically and dynamically).

Acquiring such data would allow clear conclusions to be drawn with regards to which brain regions show a specific sensitivity to facial motion. Group average data thus revealed that the pSTS responded preferentially to upright facial motion videos. Such activity did not occur for static images (faces, places, objects and bodies), random dot kinematograms or point-light walkers. This finding is consistent with previous studies and builds upon any existing literature which had used less ecologically valid stimuli (e.g., morphed videos). The animations implemented here were highly reminiscent of real life facial motion, hopefully giving rise to a more accurate neural representation.

Discriminating different unfamiliar identities from facial motion also engaged portions of the middle temporal and STS cortex. Interestingly, no activity was observed in the ventral temporal regions (e.g., FFA or OFA) typically responsible for individuating static faces (Rhodes *et al.,* 2009). Because all stimuli shared the same physical appearance, it is possible that participants did not need to recruit these structures. Instead, the STS complex sufficiently processed facial identity in the absence of characteristic form cues. Not all lines of evidence however support this view. Baseler *et al.,* (2014)

reported that the STS region was more active to changes in eye-gaze and emotional expression when the stimuli sequences were of the same person relative to multiple identities. This effect appeared to be driven by an increased functional connectivity to the FFA, suggesting that it is this region which processes facial identity. However, this study utilised sequences of static images. The FFA response could therefore be reflecting static information processing, rather than a true dynamic response (Schultz *et al.,* 2013).

Portions of the precentral gyrus also emerged as responsive to facial identity. This finding is not entirely unanticipated as there are many reports which show its association to general dynamic face processing and recognising goal-directed social actions (Sarkheil *et al.,* 2013; Grezes *et al.,* 2001). These previous findings have accordingly been interpreted as reflecting the putative involvement of mirror neurons whereby visual information is transmitted along the V1-STS-precentral gyrus pathway (Cook *et al.,* 2003; Lepage & Théoret, 2006). In the context of the current data, it is not clear why recognising unfamiliar facial identities from facial motion would engage these mirror mechanisms. To gain clearer insights, future investigations are encouraged to replicate this study by implementing an execution phase with facial actions relating to self and other identities.

7.4 Perceiving Facial Motion in ASD

An impaired perception of body motion has been thought to underlie the social cognitive deficits associated with ASD (Herrington *et al.,* 2007). In the current thesis, this hypothesis was extended to include facial motion stimuli (Chapter 3). Participants completed increasingly difficult tasks involving the discrimination of: (1) facial motion sequences; (2) male from female faces; and (3) unfamiliar identities. As the appearance of animations was identical to each other, judgements were based solely on differences in motion patterns. The analysis indicated that adults with ASD were significantly less accurate than neurotypical participants on all three experimental tasks. Interestingly however, the experimental and control group shared a similar recognition of static faces from the Benton's Test. Comparable results have been found elsewhere. For example, Weisberg *et al.,* (2014) reported that adolescents with ASD showed a selective deficit for dynamic stimuli depicting social interactions relative to photographs. This suggests a specific problem in perceiving facial motion, rather than a generalised deficit in face processing (O'Brien *et al.,* 2014).

Further in this context, Haxby *et al.,* (2000) proposed a distributed neural model whereby two distinct networks support the processing of facial stimuli. A core system mediates the visuo-perceptual analysis, whilst an extended system extracts information related to semantics. These authors further subdivided the function of the core system by advocating that invariant and changeable facial aspects engage the FG and STS, respectively. Because face structure did not vary across trials, a dysfunction of the STS could explain why the ASD group performed poorly in the current study. Indeed, the fMRI

technologies implemented in Chapter 6 did reveal hypoactivation of the STS complex during facial motion perception in participants with ASD. This finding parallels those previously reported (Pelphrey *et al.,* 2007; Rahko *et al.,* 2012; Sato *et al.,* 2012).

Strong activities in the inferior and middle frontal gyri to perceived facial movements were also observed in the ASD group. It is possible that these participants were prompting a compensatory neural recalibration in order to process facial motion. Instead of utilising occipitotemporal substrates, they decoded movements through more effortful and cognitively based mechanisms than neurotypical participants (Harms *et al.,* 2010). This idea is not new, and has been used to explain why some participants with ASD perform similarly to controls on tasks assessing body motion recognition (Rutherford & Troje, 2012). Such strategies develop over many years and probably mask the underlying visusoperceptual abnormalities in ASD (Scherf *et al.,* 2008). For instance, it has been shown that children with ASD atypically engage the primary visual cortex during empathy judgments, whereas adult participants utilise attentional top-down mechanisms via the dorsal medial prefrontal cortex (Schultz-Ruther *et al.,* 2014). Consequently, the transition from disordered visual processing to a more controlled method of analysis facilitated successful empathy judgments.

By contrast, this additional frontal activity could indicate a dysfunction of mirror neurons. Previous research which similarly found hyperactivity of the inferior frontal gyrus (IFG) when participants with ASD viewed hand gesture motion corroborates this hypothesis (Martineau *et al.,* 2010). Rahko *et al.,* (2012) also observed increased anterior IFG activity in participants with ASD who were asked to scale the valence of dynamic facial emotional expressions. The role of mirror neurons cannot be entirely evaluated though, especially as none of the current studies implemented execution or imitation phases. Regardless, it is likely that any additional activity echoes the behavioural deficit observed when participants with ASD perceived facial motion.

Returning to the hypothesis that social cognitive impairments are rooted in low-level visual deficits, the MRI data revealed decreased engagement of the primary visual cortex, lingual gyrus, inferior occipital gyrus and thalamus in the experimental group. Atypical neural responses at the earliest stages of visual processing have been previously reported. Robertson *et al.,* (2014) found that global motion processing deficits in ASD was attributed to a dysfunction of V1 and MT+/V5. Sato *et al.,* (2012) also found a weakened bi-directional connectivity between V1, the middle temporal gyrus and IFG when participants with ASD viewed dynamic displays of facial emotion. It is thus possible that the transmission or integration of information from visual areas to substrates involved in social cognition is weakened (Volkmar & Juraska, 2011; Zilbovicius *et al.,* 2006). If this is indeed the case, then it would explain why the STS region is often hypoactivated in participants with ASD.

Together, these findings demonstrate that the recognition of facial motion is impaired in ASD. It is plausible to speculate that a dysfunction in early visual processing is what drives this deficit. Abnormal connectivity between structures, as opposed to specific neural regions, may also cause such impairment. Accordingly, it is conceivable that a weakened perception of facial motion does contribute to the ASD phenotype regarding social cognition. Conceptualising ASD in this way could inform future therapies. For instance, Tardif *et al.,* (2007) showed that decreasing the speed of video presentations significantly helped ASD adults to recognise and imitate dynamic facial emotions. Because this finding demonstrates a failure to integrate rapid visuo-motion information, participants may benefit in real life if they were trained how to interpret and decode fast paced facial information.

Further in the context of therapeutic interventions, there is evidence that participants with ASD fixate less on dynamic faces compared to controls (Klin *et al.,* 2009; Nakano *et al.,* 2010). However, learning communicative behaviour through individual coaching and rehearsal has been shown to increase attention to faces in children with ASD (Sakuma *et al.,* 2012). Participants with ASD also exhibit atypical gaze patterns, choosing to focus on the mouth area rather than the eye region when viewing static faces (Jones *et al.,* 2008; Norbury *et al.,* 2009). As this is in contrast to the gaze patterns observed in controls, it appears that participants with ASD are seeking a more verbal way of understanding social cues (Grelotti *et al.,* 2002). Therapeutic interventions can therefore be designed to teach these participants how to attend to eye movements. In turn, this may facilitate face processing by increasing signals in face selective substrates. Indeed, studies which instruct participants to focus on the eyes report normal activity in the FG during face recognition tasks (Hadjikhani *et al.,* 2007). Targeting this behavioural discrepancy in early development could potentially alleviate some of the symptoms associated with poor social cognition in ASD.

7.5 Limitations and Future Directions

Experimental Design and Tasks

Although the purpose of this thesis was to investigate whether a deficit in biological motion perception contributed to poor social cognition in ASD, none of the current studies had examined the relationship between symptom severity, ability to perceive facial movements and activity in responsible neural structures. Other investigations have, however, shown that the degree of neuro-dysfunction in the right STS, during congruent and incongruent gaze shifts, correlates negatively with social perception impairments in ASD (Pelphrey & Carter, 2008). Similarly, atypical engagement of the right temporoparietal junction is associated with both mindblindness and poor social skills in adult males with ASD (Lombardo *et al.,* 2011). Robertson *et al.,* (2012) also reported a strong relationship between atypical visual integration of coherently moving dots and symptom severity in their sample of ASD

participants. Future investigations should collect comparable information so that a more detailed understanding of ASD symptomology can be acquired with regards to facial motion perception.

Furthermore, the studies outlined here have chiefly focused on the discrimination of categorical information (sequence, identity or gender). Research should now examine the recognition of both basic (e.g., happy, sad, fearful) and secondary (e.g., jealousy, irritable, embarrassment) emotions from facial motion captures. It would also be interesting to measure at which point in perception are participants with ASD able to recognise a facial affect, if at all. This could be achieved by presenting time-course videos which depict a neutral expression gradually transforming into the desired emotion at its peak. Similar work has been done with static morphed images and neurotypical populations (e.g., Sato *et al.,* 2004), but no research has examined this using the facial motion captures described here. It would be highly beneficial to the study of both emotion and facial motion recognition if investigations were extended to include such representations.

Participant Sample

The current investigations have mainly used samples of high-functioning participants with Asperger's Syndrome (characterised by the DSM-IV-TR). While the new diagnostic criterion now groups people with Asperger's, Autism and Pervasive Developmental Disability - Not Otherwise Specified under the term 'ASD', there are still important distinctions between these individuals in terms of symptom severity, language development and cognitive ability (Bennett *et al.,* 2014). It is thus crucial to investigate facial motion perception in all other subtypes of this disorder. It may be that impairments are more profound in participants who are towards the lower functioning end of the spectrum.

Similarly, the fMRI results reported in Chapter 6 are derived from a very small sample size of adults with ASD (n = 5). Because of this, the data was extremely inconsistent between participants and it was thus more appropriate to report a group average. However, such method of analysis may have yielded an effect that was actually present in only a few datasets (Button *et al.,* 2013). Population inference is obviously very difficult to achieve with such a limited sample, but these results do point to neurological discrepancies in ASD that should be further investigated on a much larger scale.

Social Attention and the 2D:4D Ratio

Chapter 6 indicated that an impaired perception of facial motion in ASD was caused by an atypical engagement of the STS, visual cortex and fronto-parietal substrates (corresponding to portions of the MNS). However, it is not clear *why* these brain regions are dysfunctional. It is possible that a lack of social orienting during the critical periods of development may cause specific neural structures to remain in a rudimentary state (Schultz, 2005). Indeed, models of associative learning propose that the maturity of mirror neurons is based on sensorimotor experience, which is largely built through social

interaction and attention to human actions (Cook *et al.,* 2014). To investigate this hypothesis more empirically, longitudinal investigations with high-risk infants should utilise eye-tracking technology in order to observe distinct gaze patterns towards facial motion (de Klerk *et al.,* 2014). This can then be correlated with performance on recognition tasks during specific points of development (e.g., infancy, adolescence and adulthood).

The 'Male Brain' theory of ASD may also explain why participants with ASD exhibit impairments in facial motion processing. This model proposes that an excessive exposure to foetal androgens evokes an extreme expression of male brain morphology (Baron-Cohen, 2009). In neurotypical populations, it is commonly reported that males excel at systemizing or spatial navigation whereas females outperform on tasks involving social cognition (Cook & Saucier, 2010; Gret & De Gelder, 2012). This sexual dimorphism is mediated by neurological variations, such as heighted STS activity in only female participants during empathic processing (Schulte-Ruther *et al.,* 2008). Researchers could therefore examine whether similar gender differences occur in facial motion perception abilities and how this might compare to participants with ASD. Similarly, it would be interesting to correlate ability on facial motion tasks with the ratio of the fourth to second finger length (2D:4D), as this is thought to indicate high prenatal testosterone levels (Milne *et al.,* 2006). Leow and Davis (2012) have conducted a similar study with non-clinical participants, showing that the 2D:4D ratio did predict their ability to perceive static faces and recruit specialised mechanisms. These points are extremely speculative, but they may provide novel insights with regards to why participants with ASD experience facial motion processing deficits (and therefore social cognition).

7.6 Concluding Remarks

The purpose of this thesis was to provide a more accurate description of facial motion perception in ASD and neurotypical participants by implementing ecologically valid stimuli. Through the use of EEG and MRI, the temporal and functional components of such perception were observed in non-ASD samples. The role of the STS was highlighted, in addition to the importance of early visual structures in processing dynamic and changeable aspects of faces. The involvement of the STS was also observed when participants were required to discriminate facial identity solely from differential movement patterns. Such finding implicates that ventral temporal substrates are not recruited when viewing stimuli which are invariant in appearance.

In participants with ASD, a clear impairment in perceiving categorical information from facial motion was found. The experimental group was less accurate than controls when asked to discriminate the sequence, identity and gender of averaged facial animations. The neuroimaging data supported these findings by demonstrating atypical widespread activities in the STS, visual cortex and fronto-parietal substrates thought to contain mirror neurons. Collectively, these neural dysfunctions most likely

contribute to the social cognitive deficits seen in ASD. Thus, the results presented here should be considered as a platform for future studies to build upon. The continued investigation regarding how participants with ASD perceive facial movements is fundamental to moving closer to a more unified conceptualisation of social cognitive deficits. Hopefully this would then allow the discovery of treatments which help to alleviate the social deficits associated with ASD.

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APPENDICES

Appendix 1.

Example trial from the FFA localiser. The same format was used for static bodies, objects and places images. (B) Example trial from the biological motion localiser. A similar format was used for the MT+/V5 and facial motion tasks.